Desynchronized Electroencephalogram in the Deeply Sleeping Cat

Abstract. Electroencephalographic patterns of the different stages of sleep in the cat are shown, with special reference to a desynchronized deep sleep. This sleep stage is characterized by an increase in reticular stimulation arousal threshold and by the reappearance of 5 to 6 spikes per second on the electroencephalogram immediately after threshold behavioral arousal.

After Dement and Kleitman (*1*) first described a low-voltage, fast electroencephalographic sleep cycle in humans, the same type of activity was described in cats by Dement (*2*) and later by Jouvet *et al.* (*3*). We have duplicated this desynchronized sleep electroencephalogram in five of our cats that have been implanted with bipolar electrodes in various deep and surface structures of the brain. These animals have been trained to go to sleep in a sound-proofed room. Behavioral and electroencephalographic arousal thresholds in response to stimulation of the reticular formation are then recorded.

The cats exhibited the normal behavioral and electroencephalographic patterns associated with going to sleep, and after 60 or more minutes of complete isolation they drifted into a very high frequency (40 to 50 per second), low amplitude, desynchronized activity (Fig. 1*B*). As described by Jouvet *et al.* (*3*), the animals were completely relaxed and deeply asleep. Especially noticeable are the occasional convulsive limb twitches. One of the cats slept with the eyes partially open during this phase. She showed marked nystagmic movements of the eyeball under relaxed nictitating membranes.

Although Dement (*4*) states that he cannot detect changes in arousal threshold between the fast- or slow-wave sleep stages, we have found increases in the reticular formation behavioral arousal thresholds of from 1 to 2.5 volts in all of our cats (Fig. 1). This finding confirms Jouvet's report (*3*) of increased auditory and reticular arousal thresholds during this sleep period.

One aspect of this desynchronized sleep stage not yet reported is shown in Fig. 1*C*. Recticular stimulation that was just enough to produce a minimal behavioral arousal (eyes open, head moves briefly) produced an almost immediate 5 to 6 per second activity which drifted into a slow-wave sleep activity and then once again into the 40 to 50 per second deep-sleep pattern.

Since the above-mentioned arousal threshold is higher than that of the slow-wave sleep, and since even minimal behavioral arousal from slow-wave sleep produces a considerable length of aroused electroencephalographic activity (instead of the 5 to 6 per second activity we find in the arousal from fast-wave sleep), we feel that the desynchronized sleep pattern is definitely indicative of a deeper sleep stage than the conventionally described deep sleep during a synchronized electroencephalogram.

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References and Notes

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3. M. Jouvet, F. Michel, J. Courjon, *Compt. rend. soc. biol.* **153**, 1024 (1959).
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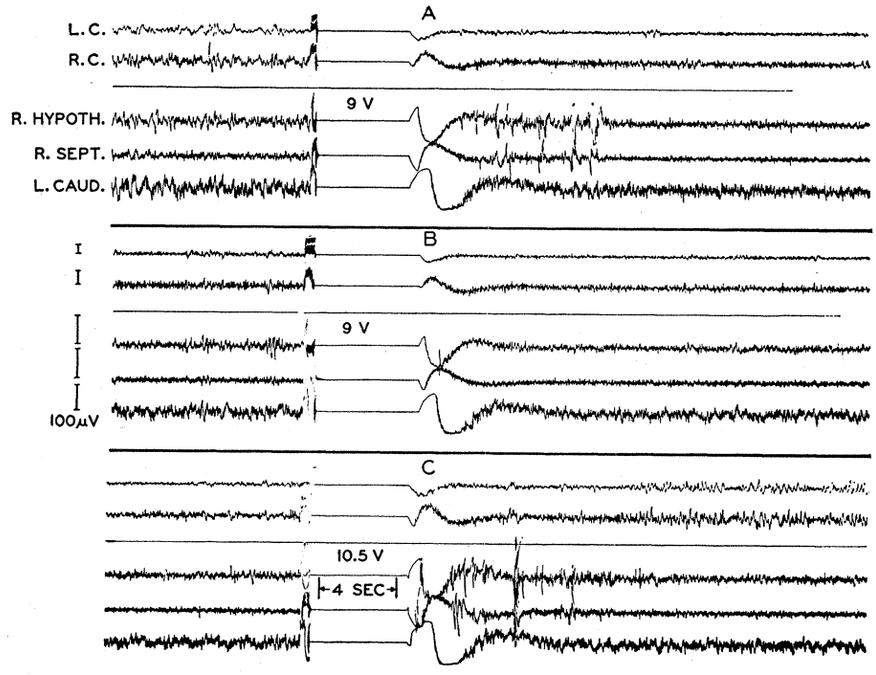

Fig. 1. *A*, Behavioral and electroencephalographic arousal with 9.0 volts at synchronized sleep stage. Notice movement artifact after stimulation. *B*, No behavioral arousal with 9.0 volts at desynchronized sleep stage. *C*, Behavioral and electroencephalographic arousal with 10.5 volts at desynchronized sleep stage. Recording pens off during stimulation (5 sec, 100 cy/sec, 0.1 msec).

An Unfortunate Event

We have had our attention drawn to several discrepancies in a paper by Pande *et al.* (*1*) which was recently published in *Science* under the title "Toxoplasma from the eggs of the domestic fowl (*Gallus gallus*)."

The discrepancies concern the figures used to illustrate the paper. Figures 1 and 2 represent the same object, although they are described as picturing different forms of *Toxoplasma gondii* from different sources. A point-by-point comparison of the two figures shows beyond doubt that Fig. 1 is merely an enlargement of a part of Fig. 2 and that it is tilted at a slight angle.

The relevant part of the caption for Fig. 1 reads: "Cyst stages of *Toxoplasma gondii* in the impression smear of chorioallantoic membrane"; that for Fig. 2 reads: "Pseudocyst of *Toxoplasma* in the ovary of white Leghorn hen." In addition, the "ghosts" of red blood cells in the background of these figures, professedly of avian origin, appear nonnucleated, and therefore mammalian.

Figure 3 in the same paper, which purports to be an original photograph, is a slightly enlarged copy of Fig. 1 in an earlier paper by Frenkel (*2*), which was entitled, "Pathogenesis of toxoplasmosis and of infections with organisms resembling *Toxoplasma*." Frenkel's caption to his Fig. 1 reads: "*Toxoplasma*, fresh preparation from peritoneal exudate of mouse, showing organism

free and within macrophage. Red blood corpuscles, some of which are crenated, provide a comparison for size. Phase-contrast microscope. Times 1000." The caption for Fig. 3 in the paper by Pande *et al.* reads: "Extracellular forms in the impression smear of peritoneal exudate of mice. Note the crenated erythrocytes (phase-contrast × 1500). [May-Grunwald-Giemsa]."

We apologize to our readers for this unfortunate event, thus following the precedent set by the Editorial Board of the *Journal of Infectious Diseases* (*3*) in a similar case in which at least five of the six figures used to document an article (*4*) "were taken from the previously published work of other authors."

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Phagocytized Platelets: A Source of Lipids in Human Thrombi and Atherosclerotic Plaques

Abstract. Phagocytosis of lipid-rich platelets by monocytes and the transformation of such cells into lipophages containing fat was observed in human thrombi. The lipophages are similar to lipophages in atherosclerotic plaques. This observation supports the idea that some atherosclerotic plaques are organized mural thrombi.

Recently, in this laboratory, the conversion of autologous pulmonary arterial thromboemboli to typical fibrofatty atherosclerotic plaques containing foam cells (lipophages) was observed in the rabbit (*1*). The lipophages were derived from monocytes that had phagocytosed and digested lipid-rich platelets within the thromboemboli. After this experiment with the rabbit, a study was made of human in vivo and in vitro thrombi to determine whether or not phagocytosis of platelets could be found.

Thrombi made in vitro (*2*) from human plasma were incubated at 34° to 37°C for 1 to 6 days. The thrombi were composed of clumps and columns of aggregated platelets surrounded by monocytes and by granulocytes, and bound together by strands of fibrin. Phagocytosis of platelets by monocytes within the thrombi occurred on the second day of incubation (Fig. 1*a*), and the transformation of such cells to lipophages containing fat occurred by the fifth day (Fig. 1*b*). The cytoplasm of the phagocytic monocytes was filled with numerous platelets, and the nucleus was displaced to an eccentric position. Swollen platelets could be distinguished in the monocytes in the early stages of fatty change. However, by the sixth day many of the phagocytized platelets had become lysed and were replaced by fatty vacuoles characteristic of foam cells.

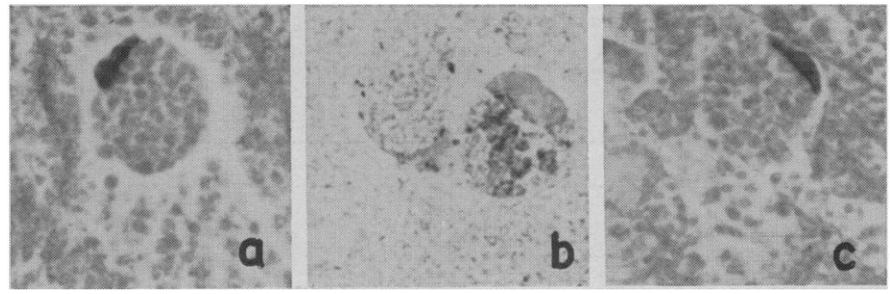

Fig. 1. Phagocytized platelets (× 400) in monocytes of in vitro thrombi (*a*, *b*) and an in vivo thrombus (*c*). Note the similarity of the phagocytic monocytes in *a* and *c* (hematoxylin and eosin stains). Two monocytes in *b* contain platelets. The platelets in the cell on the right are undergoing fatty change (Fettrot fat stain; fat is black in photograph).

The degree of phagocytosis and fatty change varied from cell to cell. Some monocytes that had not phagocytosed platelets accumulated fine droplets of fat. These cells retained their normal size and could be easily distinguished from the large monocytes that contained phagocytized platelets and fatty vacuoles. In some thrombi a few unphagocytized platelets underwent fatty change.

In each experiment 10 ml of venous blood was collected in a plastic tube (*3*), and plasma was obtained by centrifugation of the blood in the collection tube at 650 to 700 rev/min for 5 to 10 minutes. The plasma was transferred in 1-ml portions to each of three polyvinyl chloride tubes 20 cm long by 0.140 inch in internal diameter. The ends of the tubes were joined with outside plastic collars to form a circle. Then the tubes, filled approximately half with plasma and half with air, were rotated at 17 rev/min on inclined turntables (Fig. 2) until thrombi formed (15 to 20 min).

The contents of one circular tube—the thrombus in its own plasma—were incubated in each experiment. Before incubation, the tube was further sealed with paraffin at the junction, and the air in the tube was replaced with a mixture of 95 percent oxygen and 5 percent carbon dioxide by means of inlet and outlet needles. The remaining two tubes were stored at 5°C, and the plasma was used for replacement of the autologous media in the incubated tube on the second and fourth day of incubation. After each plasma exchange, the gas mixture was also replaced.

The thrombus was incubated at 34°C for 2 days and at 37°C for the next 1 to 4 days. A temperature of 34°C was used for the first 2 days because in earlier experiments initial incubation at 37°C caused much multiplication of monocytes and minimal phagocytosis of platelets by the monocytes. The circular tube was rotated on a vertical turntable at 1 rev/min during incubation so that the thrombus was carried alternately through the plasma and the

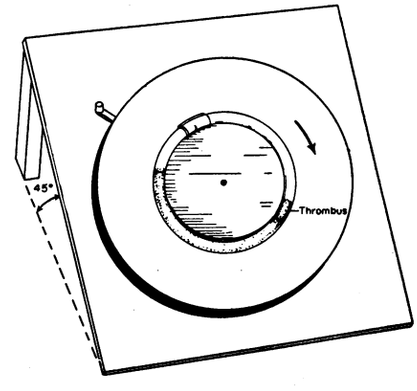

Fig. 2. Inclined turntable. The thrombus forms in the plasma as the column of plasma flows through a circular tube that rotates on the turntable.