disconnection from the anterior part of the brain) prevents or retards the appearance of the ipsilateral EEG patterns of desynchronized sleep, while the integrity of even a small part of them, belonging to either "specific" or "aspecific" systems (compare the two anatomical schemata of Fig. 1), permits their normal appearance and development. Unsatisfactory as it is, our hypothesis of a general participation of the rostropontine structures in the EEG desynchronization of the deep sleep phase seems the only one fitting the findings of the present results.

The next experiments constituted a search for the ascending pathways carrying the impulses desynchronizing the EEG during sleep, and was made by unilateral lesions at midbrain level. As with the preceding group of experiments, three types of lesions were made (19 animals): (i) lesions involving mainly "specific" structures; (ii) lesions involving mainly "aspecific" structures (Fig. 3A); (iii) lesions involving both "specific" and "aspecific" structures (Fig. 3, B and C). Electroencephalographic asymmetry during deep sleep, of the type observed following brain stem hemisection (namely, suppression or delayed appearance of the EEG desynchronization in the hemisphere ipsilateral to the lesions) was produced by the lesions of types ii and iii, provided that they were of large size (Fig. 2B).

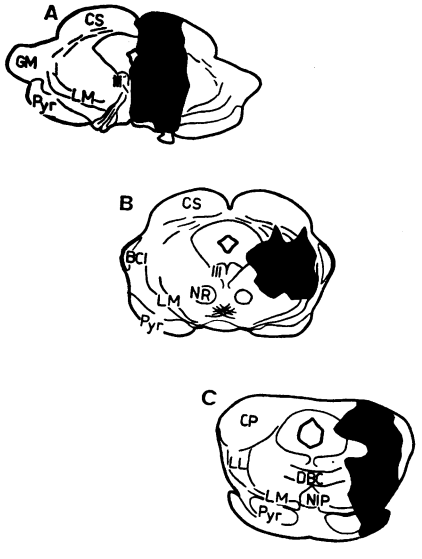

Fig. 3. Mesencephalic unilateral (right) lesions in three different cats. In the three animals the EEG patterns of desynchronized sleep were altered as in Fig. 2B. *BC*, brachium conjunctivum; *CP*, posterior collicullus; *CS*, superior colliculus; *DBC*, decussatio brachium conjunctivum; *GM*, medial geniculate body; *LL*, lateral lemniscus; *LM*, medial lemniscus; *NIP*, nucleus interpeduncularis; *NR*, red nucleus; *PYR*, pyramidal tract; *III*, nucleus of the third cranial nerve.

These findings confirm that the EEG desynchronization of deep sleep is due to or is facilitated by structures located in the low brain stem. If it is accepted, on the basis of previous work (*2, 3*), that the site of origin of the desynchronizing influence is at pontine level, our findings indicate that this influence is carried rostrally by paths running through the midbrain tegmentum. No evidence for a discrete path has been obtained, EEG changes having been observed following both medial and lateral tegmental lesions (compare A and B in Fig. 3), and no EEG effect having been produced by small lesions (that reproduced in Fig. 3B is one of the smallest effective lesions of our series). As is well known (see *4* for references), so-called "specific" fiber systems pass through the "aspecific" tegmental mesencephalic structures to ascend to the diencephalon. Therefore, our results do not bring crucial evidence of the nature of the pathways mediating the ascending influence desynchronizing the EEG during deep sleep. However, on the basis of analysis of the topographical details of the effective and noneffective lesions in our animals, it is unlikely that the EEG-desynchronizing pathways belong to anatomically known ascending "specific" fiber systems. The hypothesis of their "aspecific" nature indirectly supports the "aspecific" nature of the structures from which the desynchronizing influence would originate (*2, 3*).

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Genetics of Mitochondria

McDaniel and Sarkissian [*Science* **152**, 1640 (1966)] appear to have demonstrated complementation between two kinds of mitochondria in maize, but they did not ask a perhaps equally interesting question to which their data are also relevant. This is whether the mitochondria from F_1 heterozygotes are superior to a 1 : 1 mixture of mitochondria from the two parental lines. If such a superiority is found, an influence of the F_1 genotype (presumably the chromosomal part) on its own mitochondria would be indicated. The underlining in their Table 1 suggests that two comparisons of this type do not individually give a difference significant at the 5 per cent level, but the other relevant comparisons are not made and the matter is not discussed in the text.

This hypothesis cannot be adequately tested by the published results; but of the eight comparisons given, all independent under the null hypothesis, seven are in the expected direction, $p = .06$. This value should not be taken seriously because the hypothesis was suggested to me by some of the data. The appropriate test, which is of a powerful kind almost never used, is to compare each pair (F_1 and mixed) separately by a two-tailed t test and then to combine the resulting probabilities, most easily by R. A. Fisher's method [*Statistical Methods for Research Workers* (Hafner, New York, 1958)]. Use of one one-tailed test per cross, if randomly allocated, will not affect the assumption of independence and is permitted by the hypothesis. The null hypothesis tested is that all of the differences within pairs are due to chance, or, more broadly, that there is no real difference within pairs in the direction predicted.

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Genetics of Mitochondria

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