CATS WITH ROSTRAL TEGMENTAL AND MIDLINE
THALAMIC LESIONS*

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An earlier study (Adametz') emphasized the operational aspect of lesions placed in the tegmentum of the midbrain of the cat as it relates to the severity and kind of neurological deficit observed. Extensive (in rostrocaudal extent) bilateral electrolytic lesions of the tegmentum of the midbrain placed in two stages, or a transverse series of lesser lesions added one at a time until much of the cross-section of the tegmentum had been destroyed by the multi-stage procedure, did not inevitably cause lasting prostration and coma. In fact, such step-wise addition produced a significantly less severe deficit than did a single lesion of corresponding size made at one sitting. Furthermore it was noted that when lesser lesions, produced seriatim, coalesced over time to produce a single major one, close attention to inter-operative and postoperative care was essential to recovery. In animals in which lesions of small size were confined to, or significantly encroached upon, the periaqueductal gray matter, muteness could appear and persist despite the eventual disappearance of other neurological signs from the chronic preparation. In several animals with tegmental lesions produced by coagulation, generalized convulsions occurred, seeming to justify the term "centrencephalic seizure" used by the Montreal school.7

The current study was designed to accomplish a somewhat different objective—the observation of behavioral and electrocorticographic changes in cats with unilateral, bilateral and midline tegmental lesions of a type designed to expand and later to regress. Such a lesion can be prepared by depositing alumina gel in a cavity made in the tegmentum or other deeplying structure by electrocoagulation. Similar observations were made on a series of cats with medial thalamic lesions. The epileptogenic propensity of alumina gel led to other significant observations.

The data we present are based upon the serial neurological examinations of cats with tegmental and midline thalamic lesions, supplemented by still and motion-picture photography. Electrocorticography of the drowsy or sleeping cat, using implanted electrodes and applying arousal stimuli, was done in the search for evidence of remaining activation. Seizures were studied

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both by motion photography and electrical recording. All studies were
tcontrolled histologically.

METHOD

We report the results of 4 unilateral, 18 bilateral and 3 midline tegmental
lesions. Midline thalamic lesions were made in an additional 21 cats. Of
these 15 survived for a sufficient time to permit comparison with animals
having tegmental lesions. To provide controls, 5 animals received alumina-
gel injections of 0.05 ml. into the 3rd ventricle, 3 received injections of 0.05
ml. into the cisterna magna, and 3 were given intramuscular injections of
0.5 ml. All tegmental and midline thalamic lesions were placed with a Hors-
ley-Clarke stereotaxic apparatus. Electrolytic lesions were made first
(4.8 mA., 60 sec., cathode) and the alumina gel (0.05–0.1 ml.) was injected
into the cavity.

In the animals prepared for studies of activation cortical electrodes were
implanted over the frontal, posterior and posterolateral areas bilaterally. A
quiet, darkened room was indispensable for promotion of the drowsy or
sleeping state in an otherwise alert cat. Our observations do not indicate
that activation (the substitution of a low-voltage fast electrocorticogram
for the higher-voltage slow tracing of sleep) invariably follows the applica-
tion of an external stimulus calculated to produce arousal, even in the intact
drowsy or sleeping cat. The stimuli used were a loud snap, bang, whistle,
touch, or vigorous pinch. Some normal controls with very light sedation, and
many cats sick for whatever reason, showed no activation. Thus, if activa-
tion does not develop as a response to a suitable stimulus, its nonoccurrence
cannot be said to have any special significance since under changed condi-
tions the same cat might have been activated satisfactorily.

On the other hand, the proof that activation can be achieved has definite
value in assessing the effects of large tegmental and midline thalamic lesions
in terms of current theory. Other general factors besides sickness that
mitigate against obtaining evidence of activation are fatigue and wakeful-
ness. After prolonged wakefulness (48 hours) in the intact cat, the addition
of even a small amount of Nembutal (12 mg.) produces such a sound sleep
in our experience that all stimuli become ineffective as activators. The sub-
stitution of a novel stimulus (whistle) for one to which the animal had be-
come habituated13 aided in obtaining a trace of activation; and the best
circumstance of background found for producing activation was that of
normal postprandial sleep. Small doses of Nembutal would often block
activation of the electrocorticogram even though the animal might be
aroused in the behavioral sense.

At the termination of each experiment the animal was perfused through
the left ventricle with saline and formalin, and serial frozen transverse sec-
tions were made through each critical area of brain. Alternate frozen sec-
tions were stained with luxol fast blue (80μ) and thionine (160μ). Thin
paraffin-embedded sections from the same blocks were stained with hema-
toxylin and eosin, phosphotungstic acid hematoxylin, periodic acid-Schiff