demonstrated that when the latter was rigidly supported the ears could be gently held and injected without mishap. After progressing through several models, a rabbit holder was designed according to the arrangement shown in Fig. 1, which aimed primarily at immobilization of the rabbit's head.

The head holder is made by molding a liquid thermo setting plastic* around a plaster of Paris model which has been shaped to conform to that of the head of a rabbit weighing about 2.5 kg. Provisions are made for leaving a hole from the nose to the outside. Within the 2 horizontal, tubular supports which are included in the plastic casting run two 3/16″ ball chains 12 inches long. The anterior three-fourths of the rabbit's head is held in the cast by a horse-collar-shaped neckpiece to which are attached the ball chains. Once the rabbit's head is placed in the cast, the operator exerts traction on the chains, and under moderate tension the latter are secured by fitting them into 2 narrow vertical slots on the ends of the horizontal support tubes.

The body of the rabbit is confined in a 5″ x 5″ x 12″ metal box with a hinged lid and clasp. Two flanges fastened to the front of the box prevent the rabbit's legs from reaching the area about the ears. All parts are mounted on a piece of 12″ x 27″ x ¾″ plywood, the top and edges of which are covered with sheet metal 0.050″ thick. For maximum rigidity, durability, and ease in cleaning, stainless steel, chrome-plated brass, and plastic were used. Wood and plaster of Paris are less durable but are otherwise adequate.

It has been found that by transilluminating the rabbit's ear from below, locating and visualizing particularly the smaller veins is greatly facilitated. With this procedure it is rarely necessary to clip or shave the hair over the vein in preparation for injection. A flexible arm with a small socket and bulb attached may be brought into any desired position beneath either ear.

The apparatus has been used several thousand times and has proved very satisfactory for rabbits weighing from 1.5 to 4 kg. Confining the rabbit in the holder causes no apparent discomfort.

Summary. A device is described which has been found to be of great advantage in effectively holding rabbits and immobilizing their ears for bleeding or injection procedures. The significant feature of the apparatus is a mask, shaped to accommodate the anterior three-fourths of the rabbit's head. An inverted, U-shaped collar holds the head firmly in the mask so that the position of the ears is correspondingly stabilized.

* "Catavar 101" (cream colored) or "Catavar 1001" (clear), Catalin Corporation of America, New York, N.Y. Plaster of Paris may also be used.

Acknowledgment is made to Mr. Clifford Wilson and to Mr. Roland Parker for suggestions in design.

15500

Retromammillary Inhibition of Cortically Induced Movement.

R. RHINES AND H. W. MAGOUN.

*From the Department of Anatomy, Northwestern University Medical School.**

Recent investigation has revealed 3 regions of the brain whose stimulation inhibits the response evoked from the motor cortex: cortical area 4-S,[1] the caudate nucleus,[2] and the bulbar reticular formation.[3]

* Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

[1] Dusser de Barrenne, J. G., and McCulloch, W. S., *J. Neurophysiol.*, 1941, **4**, 311.

[2] Mettler, F. A., Ades, H. W., Lipman, E., and Culler, E. A., *Arch. Neurol. Psychiat.*, 1939, **41**, 984.

[3] Magoun, H. W., and Rhines, R., *J. Neurophysiol.*, 1946, **9**, 165.

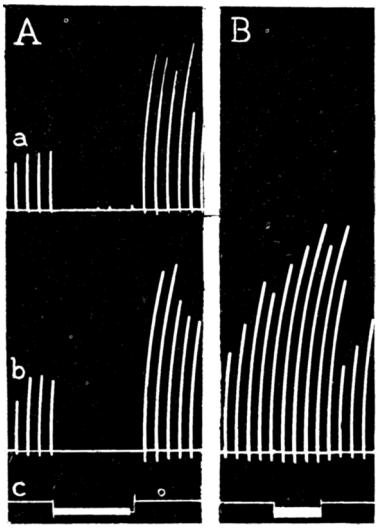

FIG. 1.
A. Cortically induced response of foreleg (a) and hindleg (b) inhibited during stimulation (c) of midbrain tegmentum. B. Patellar reflex (b) slightly augmented during stimulation (c) of same tegmental point.

During an experimental study of brain stem facilitation of cortically-induced movement,[4] it was observed that stimulation of a limited area of the rostral midbrain resulted in complete inhibition of the cortical motor response, while activation of a somewhat more extensive adjacent region produced depression of that response.

Methods. The left motor cortex of cats under light chlorolosane anesthesia was stimulated every 2 seconds by induction shocks and the responses of the right fore and hind legs were recorded with a kymograph. Using the Horsley-Clarke technic, the brain stem was stimulated in an exploratory fashion with 60 cycle sine wave current at intensities which did not by themselves evoke movement.

Stimulation of some of the points yielding inhibition was tested against the patellar reflex, elicited mechanically at 2-second intervals, and in one animal the midbrain tegmentum was stimulated against the flexor reflex of the foreleg, evoked every 2 seconds by exciting a cutaneous nerve.

[4] Rhines, R., and Magoun, H. W., *J. Neurophysiol.*, 1946, **9**, 219.

Results. The predominant alteration of cortically-induced movement during stimulation of the upper brain stem consisted of facilitation.[4] However, as shown in Fig. 1A, the response to cortical stimulation ceased or was diminished when the sites indicated in Fig. 2 were stimulated. Depression of response (small circles) was obtained from the hypothalamus and overlying thalamus, and complete inhibition (large circles) was elicited from the midline region of the posterior hypothalamus, dorsal to the mammillary bodies (Fig. 2A), from the retromammillary region (Fig. 2A and B) and from the tegmentum of the rostral midbrain (Fig. 2A and C). Stimulation of this region also inhibited responses evoked from the bulbar pyramid.

Stimulation of the same sites that caused inhibition of the cortical motor response, produced no change, or in a few instances slight facilitation of the patellar reflex (Fig. 1B). The flexor reflex was, however, inhibited by stimulating the same area of the midbrain tegmentum which inhibited cortically-induced movement, and inhibition of the flexor reflex was also obtained by exciting the periaqueductal grey.

Though these same areas were explored in the brain stem of the monkey, inhibitory responses were not encountered.

Discussion. If the sites whose stimulation depressed or inhibited cortically-induced movement in these experiments belong to a single neural system, it would appear to be distributed diffusely in the posterior diencephalon, to be concentrated ventromedially in the most anterior part of the midbrain and to shift dorsolaterally into the tegmentum at more caudal midbrain levels. The presence of intermixed facilitatory elements may, however, have prevented detection of a possible further distribution. The subsequent augmentation of cortical motor response after inhibition, seen in Fig. 1A, might, for example, have been produced by combined stimulation of excitatory and inhibitory components.

A rostral midbrain inhibitory influence is of interest from the point of view of 3 release phenomena which have been found to fol-

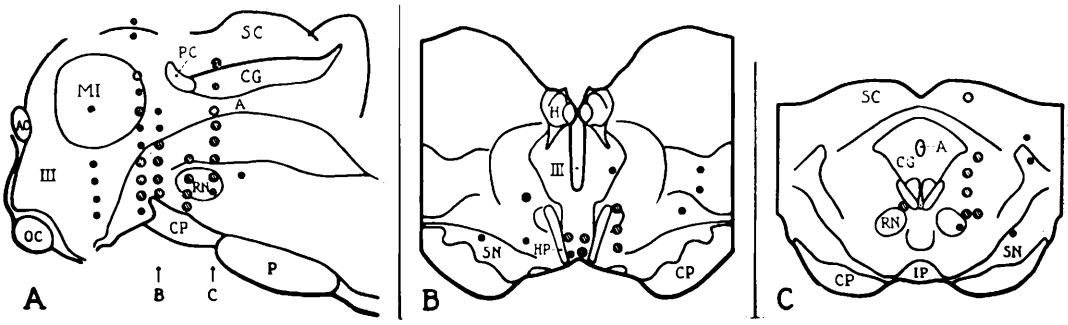

FIG. 2.

Distribution of sites whose stimulation completely inhibited (large circles) or depressed (small circles) cortically induced leg movement. A. Midsagittal reconstruction of cat's brain stem. B. Transverse section of retromammillary region. C. Transverse section of midbrain. Levels B and C are indicated by arrows in A.

A—Aqueduct
AC—Anterior commissure
CG—Central gray
CP—Cerebral peduncle
H—Habenula
HP—Habenulo-peduncular tract
IP—Interpeduncular nucleus
MI—Massa intermedia
OC—Optic chiasm
P—Pons
PC—Posterior Commissure
RN—Red nucleus
SN—Substantia nigra
III—Third ventricle

low injury to the mesencephalon. First, since Sherrington's discovery of decerebrate rigidity, it has been thought that one of the chief inhibitory centers from which lower brain stem levels are released in this condition is located in the midbrain. Ingram and his associates have made it clear that the red nucleus is not the exclusive midbrain inhibitory component eliminated by decerebration, as was once believed, though some rigidity does follow destruction of the red nuclei.[5] The sites yielding inhibition in the present study bear no obvious topographic relation to the red nucleus.

Second, a release phenomenon described as "obstinate progression" has been shown by Bailey and Davis[6] to follow destruction of the interpeduncular nucleus. Ventromedial midbrain sites whose stimulation yielded inhibition in the present experiments were located just rostral to the interpeduncular nucleus, but inhibitory responses were not obtained from this nucleus itself, nor from the habenulae or positions that were exclusively referable to the habenulopeduncular tract leading from it to the interpeduncular nucleus.

Third, ventromedial retromammillary lesions have been shown by Ingram, Barris and Ranson[7] to produce catalepsy in the cat. A large number of the inhibitory responses elicited in the present experiments were obtained from the area destroyed in such cataleptic animals, but to what extent injury to these inhibitory elements was involved in producing the cataleptic syndrome is at present uncertain.

Summary. A rostral midbrain area, the stimulation of which inhibits cortically-induced movement in the cat, has been described.

[5] Ingram, W. R., and Ranson, S. W., *Arch. Neurol. Psychiat.*, 1932, **28**, 483.

[6] Bailey, P., and Davis, E. W., PROC. SOC. EXP. BIOL. AND MED., 1942, **51**, 307.

[7] Ingram, W. R., Barris, R. W., and Ranson, S. W., *Arch. Neurol. Psychiat.*, 1936, **35**, 1175.