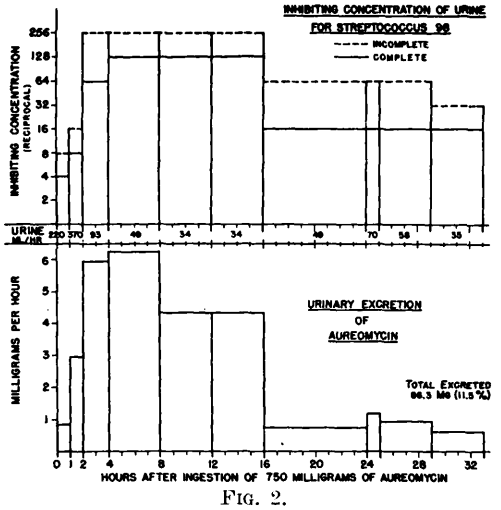

FIG. 2.

creted in both subjects at 55 and 33 hours, respectively, when the studies were terminated. The total amount recovered was 13.4% of the 500 mg dose in 55 hours and 11.5% of the 750 mg dose in 33 hours.

Blood levels carried out with the same organism were unsatisfactory since the maximum amount of antibiotic in the serum inhibited the test organism only in a final dilution of 1:2 or 1:4.

Total urinary excretion studies were made in 2 patients who were receiving 2 doses of aureomycin by mouth each day for several days. The urines were collected at 12-hour intervals and all voidings were stored at 5 to 10°C during the intervals. The tests were otherwise carried out in the same manner as before. In one of these patients who received 0.25 g twice a day for 10 days the concentrations of antibiotic in the urine were sufficient completely to inhibit the test streptococcus in dilutions of 1:4 to 1:16, and partial inhibition occurred in dilutions of 1:8 to 1:32. In the patient who received 0.5 g twice a day for 7 days the concentrations of aureomycin in the urine ranged from 10 to 32 μg per ml. In these 2 patients the total amount recovered was 5% and 2%, respectively of the amount administered. Antibiotic activity was present in the urine up to 72 hours and longer after the last dose. Control urines from these patients, obtained before and several days after the antibiotic was given failed to inhibit the test strain.

Both of the patients were acutely ill and the differences in the percentage recoveries may have been related to poor absorption of the drug associated with the illness. Technical difficulties may also have accounted, in part, for the discrepancies.

Summary. After a single oral dose of 0.5 or 0.75 g of aureomycin given to fasting normal subjects antibiotic activity was recovered in the urine for more than 33 to 55 hours. The antibiotic was excreted in high concentrations between 2 and 16 hours and the largest rate of excretion occurred between 2 and 8 hours. The findings suggest that the optimum intervals between oral doses of aureomycin should be about 8 hours.

16658 P

Stereoencephalotomy.*

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Using the previously described stereotaxic apparatus (stereoencephalotome) for the production of subcortical lesions in the human brain,[1] the following procedures have been developed.

I. *Dorsomedial thalamotomy* in mental dis-

* Aided by a grant from the Am. Med. Assn. Committee on Scientific Research.

[1] Spiegel, E. A., Wycis, H. T., Marks, M., and Lee, A. J., *Science*, 1947, **106**, 349.

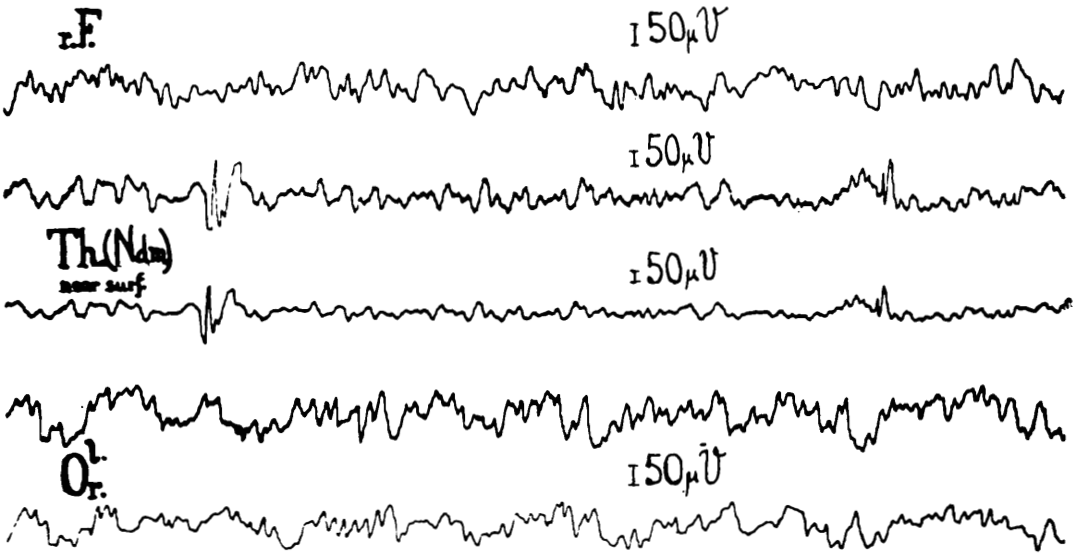

FIG. 1.
Electrothalamogram (Th) recorded from the dorsomedial nucleus near its surface simultaneously with the electroencephalogram recorded from the scalp in the frontal (F) and occipital (O) areas in a patient suffering from grand and petit mal attacks (patient under pentothal-ether-nitrous oxide anesthesia).

orders. Lesions have been placed bilaterally in the dorsomedial nucleus of the thalamus in mental disorders with a prevalent emotional component, in order to interrupt the cortico-diencephalic connections probably related to the mechanism of emotions (16 cases). Lesions covering about one seventh of the volume of this nucleus on each side have been able to produce relief of nervous tension, anxiety, depression, irritability, agitation, hallucinations or compulsions; this has been obtained[2] without at least some of the undesirable by-effects of prefrontal lobotomy such as epileptiform convulsions, childishness, facetiousness, distractibility, lack of tact or discipline.

II. *Mesencephalothalamotomy* (mty) for relief of intractable pain. Lesion of the long ascending pain-conducting pathways in the midbrain has been combined with a lesion of the dorsomedial nucleus. Since patients become unconcerned about thair pain following prefrontal lobotomy (Freeman and Watts[3]), and since the dorsomedial nucleus degenerates following this procedure, a lesion of this nucleus is added to that of the long ascending pain-conducting pathways in order to induce a relative indifference to pain perceived through remaining auxiliary pathways. In a woman suffering from unilateral facial pain for 6 years despite retrogasserian rhizotomy, mty on the opposite side abolished the pain completely except for occasional paresthesias in the maxillary region.[4] In a second patient suffering from diffuse burning pain and spasms in the right lower extremity following an injury to the lumbar spine and unrelieved by sympathectomy, rhizotomy and bilateral chordotomies, the bilateral mty reduced the painful area to the region around the right buttock, while the spasms of the leg and the sensation of the muscular contractions persisted.

III. *Paracommissural thalamotomy* in petit mal. In cases suffering from grand and petit mal attacks, lesions were placed bilaterally at the level of the commissura media

[2] Spiegel, E. A., Wycis, H. T., and Freed, H., Joint Meeting Philadelphia and New York Neurologic. Soc., April 23, 1948.

[3] Freeman, W., and Watts, J. W., *Lancet*, 1946, **1**, 953.

[4] Spiegel, E. A., and Wycis, H. T., *Wien. med. Wochenschr.*, in press; Wycis, H. T., Soloff, L. and Spiegel, E. A., Philadelphia Neurolog. Soc., Feb. 27, 1948.

around the medial part of the internal lamina medullaris.[5] Electric stimulation of this region in experimental animals yields a spike and dome pattern in the electroencephalogram (Jasper[6]). Preceding this lesion, the electrothalamogram was recorded simultaneously with the electroencephalogram;[7] spike discharges were found in the electrothalamogram.† These spike discharges were not limited to the immediate vicinity of the internal lamina medullaris but sometimes showed the maximum in more dorsal parts of the thalamus close to the ventricle (Fig. 1). In two cases, the operation did not influence the grand mal attacks; it diminished, at least transitorily, the frequency of the petit mal attacks and the severity of accompanying motor phenomena.

Summary. The stereotaxic technique has been applied to the human brain for relief of mental disorders with a prevalent emotional component and of intractable pain. In epileptics suffering from grand and petit mal, the electrothalamogram was recorded, revealing spike discharges; lesions placed in the vicinity of the commissura media diminished frequency and severity of the petit mal attacks.

5 Spiegel, E. A., Wycis, H. T., Freed, H., and Lee, A. J., Am. Neurol. Assn., June 16, 1948.

6 Jasper, H. H., and Droogleever-Fortuyn, J., *Res. Publ. Assn. Nerv. Ment. Dis.*, 1947, **26**, 272.

7 Wycis, H. T., Spiegel, E. A., and Lee, A. J., Intl. League against Epilepsy, Am. Branch, June 13, 1948.

† Gibbs, Hayne and Gibbs[8] also recorded spike discharges from various subcortical areas in petit mal. The authors inserted the recording electrode into the brain of the patients without the guidance of a stereotaxic apparatus, trying to establish the location by x-ray studies after air injections into the ventricles.

8 Gibbs, F. A., Hayne, R., and Gibbs, E. L., Int. League against Epilepsy, Am. Branch, June 13, 1948.

16659

Possible Role of Free Phenols in Renal Uremia.*

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The work of Becher and his associates,[1-6] has stressed the importance of free phenols in blood as a major factor in the development and intensification of the uremic syndrome. In an extensive review of the pathogenesis of this syndrome, Harrison and Mason[7] state that "whether in patients with nephritis phenol retention is sufficiently marked to produce further renal damage is an interesting but still unsettled problem." Dickes,[8] using the method of Theis and Benedict[9] for determining blood phenols, noted that 21 out of 23 uremic patients showed a direct correlation between the degree of cerebral depression and the height of the blood phenols. However, the correlation of total phenols was

* Data from thesis submitted in partial fulfillment of the requirements for the Degree Master of Science.

1 Becher, E., *Deut. Arch. f. Klin. Med.*, 1925, **145**, 333.

2 Becher, E., *Ergeb. d. ges. Med.*, 1933, **18**, 51.

3 Becher, E., and Koch, F., *Deut. Arch. f. Klin. Med.*, 1925, **148**, 78.

4 Becher, E., and Litzner, S., *Klin. Wochschr.*, 1926, **5**, 147.

5 Becher, E., and Litzner, S., *Klin. Wochschr.*, 1926, **5**, 1373. Cited *Chem. Ab.*, 1926, **20**, 3192.

6 Becher, E., Litzner, S., and Taglich, W., *Z. f. Klin. Med.*, 1926, **104**, 182.

7 Harrison, T. R., and Mason, M., *Medicine*, 1937, **16**, 1.

8 Dickes, R., *Arch. Int. Med.*, 1942, **21**, 662.

9 Theis, R. C., and Benedict, S. R., *J. Biol. Chem.*, 1924, **61**, 67.