

EXPERIMENTAL AND CLINICAL OBSERVATIONS ON THE
SYNDROME OF PINEAL GLAND DESTRUCTIONJohn Martin, M.D.
Chicago, Ill.

THOUGH A LARGE VOLUME of experimental and clinical data is at hand concerning the physiologic action of the pineal gland, there is no common agreement as to the actual rôle this organ plays in the mammalian organism. The present investigation, which has been in progress for more than five years, was prompted by three factors: (1) The existing disagreement among comparative anatomists concerning the evolutionary implications of the pineal region of the brain; (2) the lack of agreement as to the exact histologic structure of the pineal gland, and the potentialities of function dependent upon its structure; (3) the possible endocrinic significance of pineal tumors in young human subjects so far as sexual and somatic development are concerned.

The results here reported were obtained following extirpation of the pineal gland from the brains of immature rats, cats, dogs, and monkeys. Litter-mate controls were used for each sex, operations and laboratory conditions were standardized, weight, length, roentgenographic and photographic data were secured at regular intervals, behavior and reproductive characteristics were recorded, and all autopsy material has been subjected to histologic study. The gland was removed from kittens six weeks old by means of the Horsley-Clarke stereotaxic instrument. In all the other animals the gland was removed manually by "open" operation—in the rats at 18 days, in the dogs at five weeks, and in the monkeys at two and one-half months. To date 57 rats, 125 cats, ten dogs and five monkeys have been used in this study.

There was apparently some hastening of maturity in the pinealectomized rats com-

pared to their controls, but our data of this animal have not led to positive conclusions. The results in the cats have been striking in that the pinealectomized males showed a maturity, both in sexual and somatic development, for to five months in advance of that seen in their control litter-mates. No somatic differences were noted in the female cats, but the pinealectomized females were late in their first estrus, litters were small and frequently dropped before term or born dead, and second generation cats were reared with great difficulty. Somatic and sexual maturity in the pinealectomized male dogs also preceded that of the control animals, and, again, as in the cats, the first estrus occurred later in the pinealectomized female dogs than it did in their normal controls. Complete data concerning the growth, sexual maturation, and histologic findings in the monkeys are not yet available. No degeneration has been found in the fiber tracts leading from the region of the pineal gland in our lesion animals, and no significant histologic changes have been noted in the endocrine organs.

Briefly, these results obtained in the experimental laboratory coincide in what must be more than an accidental manner with an analysis of a large series of patients with tumors of the pineal gland, reported elsewhere by the author. A tendency to somatic and sexual precocity in prepuberal boys with pineal tumors, and amenorrhea in postpuberal girls of otherwise normal development indicate, when considered together with these results of simple *apinealism* in animals, that the pineal gland may be postulated to bear significance as an endocrine organ, related to the processes of sexual and somatic maturation in mammals.

DIAGNOSIS AND TREATMENT OF
GLOSSOPHARYNGEAL NEURALGIAJ. Grafton Love, M.D.
Rochester, Minn.

From the Section on Neurosurgery, Mayo Clinic, Rochester, Minn.

GLOSSOPHARYNGEAL NEURALGIA is a distribution of the ninth cranial nerve. The major type of neuralgia occurring in the curative treatment has been well-established

since publication of Adson's paper on "The Surgical Treatment of Glossopharyngeal Neuralgia," in 1924.

The symptoms of glossopharyngeal neuralgia are fully as typical as those of trigeminal neuralgia. The severe pain occurs in paroxysms, and between attacks the patient is comfortable and able to go about his normal activities without discomfort. The paroxysms are sudden in onset, and the pain, which usually extends from the region of the tonsillar fossa of the affected side to the homolateral ear, is exquisite. During an attack, the patient usually becomes immobile and is unable to talk, chew, or swallow until the paroxysm has disappeared. Immediately thereafter, he may appear to be perfectly normal and in good health unless considerable loss of weight has resulted from his inability to eat. The paroxysms usually are brought on by chewing, swallowing, and yawning. The examiner may produce an attack by irritating the "trigger zone" in the tonsillar fossa of the side affected. Results of neurologic examination are negative and the pharynx and larynx are free from objective evidence of disease.

Glossopharyngeal neuralgia must be distinguished from trigeminal neuralgia, superior laryngeal neuralgia, and the so-called "atypical neuralgias" of the face and neck. In doubtful cases, the differential diagnosis can be established by cocainization of the

throat of the patient on the side involved. This anesthetizes the "trigger zone" of glossopharyngeal neuralgia, and during the period of anesthesia, the patient will be free of paroxysms of pain, so that the examiner will be unable to initiate an attack by irritation of the tonsillar fossa.

There is no specific medical treatment for glossopharyngeal neuralgia. The treatment of choice, and the one which produces excellent results, is intracranial section of the involved ninth cranial nerve in advance of the point at which it enters the jugular foramen. This operation can be performed with little risk through unilateral suboccipital craniotomy. The operation has no distressing sequelae.

DISCUSSION.—DR. HENRY J. VANDENBERG (Grand Rapids, Mich.): I should like to ask Doctor Love if he had used vitamin B at all, vitamin B compound, and vitamin B₁, for these glossopharyngeal neuralgias.

DR. J. G. LOVE (Rochester, Minn., closing): I have had no experience whatsoever with the use of the vitamin compound in glossopharyngeal neuralgia. I have not even tried it for trigeminal neuralgia, but I have had the pleasure of seeing quite a number of patients who had taken such medication without any benefit to trigeminal neuralgia, despite the reports in the literature that it is of distinct benefit.

THE USE OF CELLULOID PLATE TO COVER SKULL DEFECTS: CASE REPORTS

Albert S. Crawford, M.D.
Detroit, Mich.

From the Division of Neurologic Surgery, Henry Ford Hospital, Detroit, Mich.

According to Grekov, Jacob von Mackren, in 1670, successfully repaired a skull defect with a piece of dog's bone. Since then, from time to time, reports have appeared advocating the use of many and various substances to cover skull defects of all types.

All seem generally agreed upon the main indications for plastic repair of skull defects—as follows: (1) to remove the irritation in focal epilepsy; (2) to prevent injury to the unprotected brain; (3) to correct unsightly depressions; and (4) to

relieve pain or discomfort resulting from the defects.

The following have been the commonest substances used: (1) Metals—aluminum, gold, silver, and platinum; (2) celluloid and vulcanite; (3) dead bones—obtained from cadavers, dogs, sheep, geese, and oxen; and (4) human bone transplants—usually autogenous from skull, tibia, ribs, ilium, sternum, scapula, cartilage and bone chips.

The important requisites for a satisfactory defect-closing substance are: (1) That it be sufficiently thin and yet possess enough