THE YEAR BOOK *of* NEUROLOGY, PSYCHIATRY *and* NEUROSURGERY

(1955-1956 YEAR BOOK *Series)*

NEUROLOGY

EDITED BY

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PSYCHIATRY

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INCORPORATED

200 EAST ILLINOIS STREET CHICAGO 11

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The articles abstracted herein are taken from journals received between November 1954 and October 1955.

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INTRODUCTION

The outstanding neurologic event of 1955, beyond all doubt, was the development of an effective vaccine against acute anterior poliomyelitis. The field trials in the United States in 1954 were carried out with scrupulous controls, and the great number of patients in the study made it the most extensive immunologic test ever conducted. The Francis report (this YEAR BOOK, p. 69) demonstrated the safety and effectiveness of the vaccine used in the field trials. After public release of the vaccine, 79 cases of poliomyelitis developed among children given injections of material from one laboratory, and of these, three-fourths were paralyzed and 11 died. An additional 105 cases occurred among family contacts and 20 among community contacts. Prompt action by the U.S. Public Health Service led to more careful methods of testing the vaccine and to new safety procedures, which, as explained by Salk[1], consisted chiefly of filtration of the virus within 72 hours of beginning inactivation, so that precipitates sheltering virus from the Formalin were removed. Under the new regulations, widespread vaccination proceeded, and by November 15 about 21,000,000 doses had been released, though the exact number of persons vaccinated is not yet known.[2] Use of the vaccine in the United States in 1955 was about 75% effective in preventing paralytic poliomyelitis among those vaccinated, although many children received only one injection. No further complications occurred. Salk recommends (loc. cit.) that vaccination consist of two intramuscular or subcutaneous injections of 1.0 cc., spaced four to six weeks apart, with a third some seven months later.

The World Health Organization, at a meeting of experts on poliomyelitis vaccination from many countries of the world, held at Stockholm Nov. 21-25, 1955, reported that in Canada some 860,000 children were given two injections each with only one possible case of paralytic poliomyelitis associated with the vaccination. In Denmark, 425,000, or 98% of all children aged 7-12 years, were each given 0.2-0.3

(1) Salk, J. E.: Poliomyelitis vaccine in the fall of 1955, Am. J. Pub. Health 46:1-14, January, 1956.

(2) Langmuir, A. D.; Nathanson, N., and Hall, W. J.: The surveillance of poliomyelitis in the U. S. in 1955, Am. J. Pub. Health (to be published).

ml. of vaccine, the dose being repeated four to six weeks later, with the result that no cases of paralysis occurred in any vaccinated child, and no serious reactions. In France no mass vaccinations were carried out, but in Germany 100,000 children aged 1-15 years received two injections each, among whom there were no serious reactions and no case of paralytic poliomyelitis. In South Africa a modified Salk vaccine, made from the Brunhilde strain and two local South African strains (Collans and Templeton, resp.), was given to about 15,000 children without any cases of paralytic poliomyelitis among those vaccinated. In Sweden, a Formalin-treated vaccine from virus grown on human embryonic tissue was tested on 2,000 school children with encouraging results and no serious reactions.

These evidences of safety and efficacy are most convincing, but the World Health Organization experts recommend extreme care and the highest technical skill in the production and testing of the vaccine in order to avoid accidents. This caution would seem warranted. The same group of experts pointed out that successful vaccination may depend on knowledge of the characteristics of the prevalent viruses in various parts of the world. Attention should also be given to other viruses which may cause diseases resembling poliomyelitis and thus mistakenly discredit the vaccine.

The year has seen continuing intensive work on the functions of the temporal lobe and the role played by the "visceral brain" in the behavior of the organism. Several articles in this YEAR BOOK describe contributions to this work. It is evident that the temporal lobe receives sensory impulses of many modalities, and these evoke visceral circuits which are determinative in the organism's affective responses. These visceral responses not only prepare the organism for its reactions to the environmental circumstances; they would seem also to offer the dynamic for extrinsic behavior. It seems probable, both from experimental work and from the study of patients with temporal lobe epilepsy, that these lobes are the chief areas in which affective states and responses are elaborated in the rich detail of emotional life (see Macrae, this YEAR BOOK, p. 161). That the temporal lobes are not the "seat of emotion" is evident, however, from the extensive work now indicating the importance of

the cingulate gyrus (Amyes and Nielsen, p. 21), the thalamus, the hypothalamus and many other areas. It is probable that the unique role played by the temporal lobe in memory is just this affective element, which, through a *feeling* of familiarity, establishes the "recognition" of incoming constellations of sensory items, and so sets the extrinsic response in the patterns already "learned" for those familiar patterns. Thus, the temporal lobes, and in particular their inferior and medial portions, become indispensable to memory, but not its sole repository.

For an extensive and eminently informed discussion of the major functions of the temporal lobe, the reader is referred to an epochal symposium, published under the direction of Prof. Th. Alajouanine and entitled *Les Grandes Activités du Lobe Temporal* (Paris: Masson & Cie, 1955). Unfortunately, economy of space forbids an abstract of this 300 page volume.

Among other important advances during the year one must mention the work on so-called "tranquilizing drugs," which suggests that many more pharmacodynamic approaches to therapy may be expected. Encouraging progress is evident in the field of degenerative diseases, some of which, like Wilson's disease, may prove to be of metabolic origin and perhaps eventually amenable to therapy. A valuable monograph on Huntington's chorea, entitled *Setesdalsrykka (Chorea Progressiva Hereditaria)* by Alf. Lorentz Orbeck and Thordar Quelprud of Oslo, appeared late in 1954. This book, in Norwegian, has good summaries in English and in French. Such surveys of the prevalence of multiple sclerosis as those of Handley in Scotland (p. 96) and of Allison and Millar in Northern Ireland (1954-55 YEAR BOOK, p. 118), as well as of Maretschek, Schaltenbrand and Siebert in Germany (this YEAR BOOK, p. 94), suggest a marked variation in the frequency of this disease in different localities even within relatively small regions like the British Isles or Germany, in addition to the well known increased prevalence in the high latitudes. What these local variations mean is not yet apparent; that they have a meaning for etiology can not be doubted, even though these studies seem to exclude climatic, geographic, dietetic, mineralogic, nutritional, genetic and infectious factors.

The eager study of the convulsive disorders yields data

which make it ever more clear that EEG patterns are not readily translatable into clinical terms and that this effective tool in the study of epilepsy needs much more study in correlation with clinical, physiologic and pathologic data before its effective use with patients can be fully achieved. The current lazy appeal to the EEG for the answer to all questions of diagnosis, prognosis and therapy is more than ever to be deprecated. It appears that any epileptic discharge may at times lead to grand mal; that seizures vary clinically only in their minor degrees, but that these clinical variations are highly suggestive as to the focus of discharge; that the 3/second spike and wave pattern may arise from various sources, and that the EEG is often strangly silent in precisely those patients whose clinical abnormalities are most striking.

A number of new anticonvulsant drugs continues to appear; only experience can serve to evaluate them. Likewise, many other therapeutic substances are made available each year—for myasthenia, myotonia, Huntington's chorea, for blocking the sympathetic pathways, for reducing agitation, and so on. For details, the reader is referred to the pages to follow.

—ROLAND P. MACKAY.

ANATOMY

Spinocortical Fibers in Man. P. W. Nathan and Marion C. Smith[1] (National Hosp., London) present evidence of a tract originating in the spinal cord, ascending in the medulla and pons with the corticospinal tract and running through the internal capsule toward the cerebral cortex.

Material was from 43 patients who had spinothalamic chordotomy for relief of intractable pain. The fibers of the spinothalamic, spinocerebellar, spinoreticular and spinotectal tracts were divided. In most patients the lesion involved the descending as well as the ascending fibers. In eight patients the operation was at the 2d or 3d cervical segment. Three patients died too soon and one too late to demonstrate degenerating fibers. The other 35 patients were operated on at various levels between the 5th cervical and 1st lumbar vertebrae.

In all patients with lesions in the upper cervical cord, the lesions extended posteriorly and degeneration descended in or adjacent to the lateral corticospinal tract. There were some ascending degenerating fibers among the intact fibers of the corticospinal tract. Cells of origin were not demonstrable.

Most, but not all, of the fibers crossed in the pyramidal decussation. They were diffusely scattered in the pyramids and in the corticospinal tract area of the pons. They concentrated into a small region of the crus and the posterior part of the internal capsule. In the highest levels of the internal capsule they were confined to the retrolenticular area. In the capsule the course was not the same as that of the descending corticospinal fibers. Many of the ascending fibers must go to the cortex, for there is no other gray matter at this level.

The Marchi bodies varied from 3 to 25 μ in diameter. As Marchi bodies represent disrupted myelin sheaths, they cannot indicate the exact caliber of the fibers from which they came. However, large Marchi bodies have been observed only in those tracts known to contain thick fibers. Therefore, the tract probably contains thick fibers. The degenerating fibers described apparently represent a true spinocerebral tract.

(1) J. Neurol., Neurosurg. & Psychiat. 18:181-190, August, 1955.

Ascending Fibers in Brain Stem Reticular Formation of Cat. Alf Brodal and Gian Franco Rossi[2] (Univ. of Oslo) inserted blunt spatulas into different levels of the upper brain stem of kittens 8 or 9 days old. Four to 13 days after the operation they were killed and the brains studied.

Characteristic retrograde changes had occurred in all types of cells—giant, large, medium-sized and small—in the reticular formation of the brain stem. These changes indicated that the affected cells had axons projecting rostrally.

Neurons were distributed along the entire length of the medullary, pontine and mesencephalic reticular formation. Those with ascending axons were restricted to the medial two-thirds. They were particularly abundant at the levels of the rostral third of the inferior olive and the abducens nerve; only in the latter are rostrally projecting giant cells present. Some cells of the ventral part of the raphe nucleus of the medulla and pons also were changed. The predominant forms were homolaterally ascending fibers. In the maximally affected areas about one third of all giant, large and medium-sized cells had an ascending axon.

A considerable proportion of all neurons projecting rostrally had long axons passing beyond the mesencephalon. Whether shorter ascending axons were present could not be determined, but seemed likely. Some giant cells projecting rostrally had axons dichotomizing in ascending and descending branches. Many reticular neurons with long ascending axons indicated that the ascending activating system is more than just a system of short neuronal relays. The aggregation of cells with long ascending axons at several levels suggested entry of afferents influencing the activating system.

Anatomy and Physiology of Pain is reviewed by F. Bremer[3] (Brussels). The dissociation of sensations proved essential to formulation of pain as a particular sensory modality rather than as an emotional state. Only for pain has it been possible to assign a particular sensory end organ, the diffuse fibrillary network of bare nerve endings present in the same form whenever pain arises. The fibers involved are in the delta and C groups of Erlanger and Gasser's classifica-

(2) A.M.A. Arch. Neurol. & Psychiat. 74:68-87, July, 1955.
(3) Acta neurol. et psychiat. belg. 54:771-785, October, 1954.

tion. They can be separated from the largest A fibers by ischemia and drugs. Mechanical stimulation of the finely myelinated or nonmyelinated fibers produces pain; similar stimulation of the bundles of myelinated fibers innervating the hairs does not. Many encapsulated receptors have double innervation, so that pain can appear when stimulation becomes dangerously excessive. However, there is some touch modality in the bare nerve endings, as in the cornea after weak local anesthesia or section of the trigeminal tract in the medulla.

Delta fibers are used for immediate, short "first pain," whereas the C fibers subserve the prolonged, delayed "second pain," less well localized spatially and having a burning component. These two modalities can be dissociated by anesthesia and other technics, especially when long pathways are involved (so the conduction velocities can make themselves evident). The delayed disagreeable pain in tabes has been attributed to the greater damage to A fibers than to smaller ones; however, a similar dissociation could be produced by changes in excitability in recently damaged or regrowing fibers. There is little doubt that sensory fibers (delta, B, C) accompany sympathetic trunks and enter dorsal or cranial nerve roots.

In summarizing the details of the anatomic course of pain fibers, Bremer recalls the metameric distribution of fibers in the tract. The sacral fibers lie posteriorly and near the surface; this position may be responsible for the retention of sensation of fulness of the rectum and bladder, or the latter may be due to proprioceptive fibers intact after the usual chordotomy for pain. There is good evidence of some uncrossed pain fibers, mostly of visceral origin. In contrast to the million nerve fibers in the pyramidal tracts, the spinothalamic tract in the brain stem contains only 1,500-2,000 fibers. This may be associated with the phylogenetic newness of this direct pain pathway; it is said not to occur below primates (lower forms have a multineuron chain passing in the reticular formation). The admixture of pain and lemniscal fibers in the posterior-ventral thalamus makes it impossible to trace pain pathways beyond this region. Convergence of metameric innervation is used to explain certain types of referred pain, e.g., subscapular pain with phrenic irritation. Facilitatory convergence raises the question of possible con-