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

(1962-1963 YEAR BOOK *Series)*

NEUROLOGY

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INCORPORATED

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THE PRACTICAL MEDICINE YEAR BOOKS

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INTRODUCTION

To make a relatively small but representative selection from the vast amount of each year's neurologic publications is an exercise of some daring; to summarize this selection in a few paragraphs designed to present the current visage of neurology is indeed presumptuous. To escape from one's personal myopia and bias, to judge between the significant and the trivial and to assess relative importance requires a quixotic self-assurance that is quite artificial. Neurology, like all living things, grows from year to year, not by major leaps but by a combination of minute progressions and withdrawals, so that each year closely resembles the last, as it forecasts the next.

The electron microscope continues to dominate neurohistology; the glia seems firmly established as a metabolic agent of great importance in the life of the neuron, its projections and their myelin sheaths. Microchemical studies reveal a high degree of activity of the glia, especially in pathologic states. The recent electron microscopic denial of extracellular space and restriction of edema to intracellular and especially intraglial water and electrolytes is now all but established, though the light microscopists (*vide* Feigin and Popoff) find some evidence for extracellular edema. An interesting recent discovery is the toxic effect on glia of blood serum from patients with certain neurologic diseases, as illustrated in the article by Berg and Källén in this YEAR BOOK.

In matters neurophysiologic, our understanding of the mechanisms of motor integration becomes clearer, though more complex, in the light of the marvelous reticular formation, no doubt the phylogenetically oldest part of the central motor apparatus. Denny-Brown's article is of much value here. At the distal level, the alpha and gamma systems, discussed in this YEAR BOOK by Struppler and associates, by Hofmann and by Stern and Ward, have assumed crucial importance as the servomechanism modulating motor activity, disruption of which may produce not only spasticity but parkinsonian rigidity and tremor as well.

Virologists, who are constantly identifying neurotropic

strains, continue to enlarge our image of the encephalomyelitides. (See reports on diphasic tick-borne viral encephalitis by Kääriäinen *et al.* and by Fraser and Schiff, mentioned in the comment to the Kääriäinen article.) Meanwhile, the immunologists, following their great successes in the field of vaccines against poliomyelitis, work to contrive similar methods of vaccination against other neurotropic viral diseases. In this YEAR BOOK will be found several articles on a number of viral infections producing inclusion body encephalitis, detailing their clinical features and describing efforts to produce oral attenuated vaccines, e.g., against Japanese B encephalitis (Rohitayodhin and Hammon). An attenuated virus of Venezuelan equine encephalitis has apparently exerted an antineoplastic effect on malignant lymphoma (Tigertt *et al.*)—a development which may be of tremendous significance. In the field of poliomyelitis, mass vaccination with oral attenuated virus, as noted in recent years, continues to prove its effectiveness and safety and to justify the editor's prediction 2 years ago (editorial comment, 1960-61 YEAR BOOK, p. 65) that "the future of vaccination against poliomyelitis lies in the use of attenuated live oral vaccines." That problems still exist in this field is emphasized by Koprowski, especially regarding the stability of the type 3 virus.

Inclusion body disease emerges continually in many different contexts—in the fetus (Elliott and Elliott), as a result of endemic herpes simplex (Drachman and Adams), in subacute encephalitides (Chao; Bornstein *et al.*) and in familial forms (Willi and Bischoff).

Vascular disorders of course still constitute a great bulk of the clinician's problems. The angiogram, with increasing safety, has become an almost routine diagnostic tool, though strange hazards, such as dissecting carotid aneurysm (Boyd-Wilson), still exist. In fact, despite his grateful appreciation of the value of the newer diagnostic gadgetry, and especially of cerebral angiography, which has become indispensable, the critical clinician observes with distress the growing tendency of residents and younger clinicians to resort to these technics with indiscriminate frequency and often without careful anamnestic and clinical study. The unfortunate patient is too often given over routinely to angiography,

electroencephalography, electromyography, pneumography or that newest wonder, the "radioactive brain scan," in default of prior careful personal examination and study and without due regard to their appropriateness. Thus, diagnosis becomes a problem in engineering rather than one in disturbed physiology.

The neurologic literature presents increasing evidence of the serious hazards of anticoagulation therapy. The editor has sounded this alarm often enough in the past; the dangers are ever more apparent. The article by Wiener and Nathanson is but a single, though striking report, emphasizing as it does, that careful control by attention to the coagulation time is not an adequate safeguard.

Puerperal and antepartum cerebral thrombophlebitis has appeared more frequently in the literature, as illustrated by the reports by Lorincz and Moore and by Bacallao and Martin.

Among the so-called degenerative diseases, myasthenia gravis continues to be frequent and to defy our search for basic cause and effective therapy. A recent concept would relate it to an autoimmune reaction in the thymus, against proteins of the motor end-plate or other muscle antigens. Histologic evidence for this possibility has been presented by White and Marshall in this YEAR BOOK. Also, electron microscopic study begins to show structural alterations (Zacks *et al.*), as well as histochemical data as to the distribution of acetylcholinesterase (Barrnett), in the neuromuscular end-plate in myasthenia gravis.

A monograph on multiple sclerosis in Iceland deserves mention (Studies in Multiple Sclerosis: V. Multiple Sclerosis in Iceland, Gudmundsson, K. R., and Gudmundsson, G.: Acta neurol. scandinav., supp. 2, vol. 38, 1962). These authors, in a meticulous survey in this island of only 158,000 population, found a prevalence of 44/100,000 and a yearly incidence of 2.26/100,000. The mean age at onset was 25.4 years, the ratio of female to male 5:3. Familial occurrence was found in 8.75%. These figures are in accord with those found in other northern European countries.

No great advances can be reported in the field of the myopathies, in which the chief recent develpment is the continued verification of the glycogen storage disease (Mc-

Ardle's syndrome) as a true enzyme-deficient disorder. Many, if not all, of the myopathies seem likely to be based on such metabolic deficiencies, attributable, of course, to genetic factors.

ROLAND P. MACKAY

ANATOMY

Anatomic Pathways Related to Pain in Face and Neck are described by James A. Taren and Edgar A. Kahn[1] (Univ. of Michigan). The existence of alternate paths for perception of facial pain was suggested by the ability of the nervous system to readjust to surgical lesions. Nerve fiber degeneration was studied by the Weil and Marchi technics after retrogasserian rhizotomy in 3 humans and 3 monkeys, medullary tractotomy in 2 humans and 2 monkeys and extirpation of the cervical part of the nucleus of the descending tract of the trigeminal nerve in 2 monkeys.

Parts of all divisions of the 5th cranial nerve were found to be represented as far caudally as the 4th cervical level in the descending tract, with a dorsal to ventral arrangement of the mandibular, maxillary and ophthalmic divisions; the most central areas of the face terminated highest on the pars caudalis. It was concluded that a level 6 mm. below the obex is best for medullary tractotomy. An incision 4 or 5 mm. deep, extending from the bulbar accessory rootlet to a line drawn from the posterior rootlets of the 2d cervical root is dorsal enough to insure inclusion of the mandibular fibers.

The ventral secondary ascending tract of the 5th cranial nerve subserves facial pain, as a secondary pathway; the lateral spinothalamic tract subserves cervical pain secondarily. Since the nucleus of the spinal tract of the 5th cranial nerve is overlapped by the dorsal horn gray, the cell bodies of origin of the second-order neurons of these two tracts are close to one another. It was found that the ventral ascending tract crosses chiefly at medullary levels, rather than at its origin. Thus, cross-synapses in the upper cervical cord are likely, with fibers carrying pain from the face crossing with fibers entering the lateral spinothalamic tract from the upper cervical area. Facial pain impulses can thereby get into the upper cervical region of the thalamus and the "upper cervical field" of the cortex. Conversely, pain impulses from the neck may cross into the ventral ascending tract of the 5th cranial nerve. Though the paths exist, it

(1) J. Neurosurg. 19:116-121, February, 1962.

remains to be proved whether cross-synapses actually occur. That they do is suggested by the facts that tic douloureux can be relieved by occipital nerve block and that high cervical cordotomy without bilateral posterior rhizotomy at the 2d, 3d and 4th cervical vertebrae may fail to relieve cervical area pain. Cortical sensory patterns are determined by the peripheral sensory input and must shift functionally whenever the peripheral pattern is shifted.

Topographic Projection of Fibers from Anterolateral Quadrant of Spinal Cord to Subdiencephalic Brain Stem in Man was investigated by David Bowsher[2] (Univ. of Liverpool). This study is the first to be made of the ascending afferent connections of the brain stem in which a modern silver technic was used for preterminal degeneration. The material consisted of brain stems from 7 patients on whom spinal cordotomy or medullary tractotomy was performed for intractable pain. Undegenerated portions of sections in which degeneration was seen served as controls. The method of Nauta was used for staining.

There was profuse degeneration in the superior colliculi, mostly ipsilateral to the cord lesion, and in the intercollicular commissure; changes were scanty in the inferior colliculi. Degeneration was noted in four cranial nerve nuclei: the lateral vestibular nucleus (ipsilateral, dorsal part only; also on the lateral edge of the descending vestibular nucleus), the nucleus of the solitary tract (bilateral, mostly caudal), the spinal nucleus of the trigeminal nerve (bilateral, caudal part) and the dorsal column nuclei of both sides. Among nuclei of cerebellar projection, the lateral reticular nucleus was most heavily involved (ipsilaterally); the paramedian and pontine nuclei bilaterally and (in 4 cases) the inferior olives of both sides showed degeneration.

Extensive bilateral degeneration was seen in the reticular formation, primarily medially and in the caudal half. Laterally, the nucleus parvocellularis and nucleus paragigantocellularis lateralis were sparsely involved; the nucleus subcoeuruleus had some fibers. Centrally, there was profuse degeneration in the rostral third of the central reticular nucleus of the medulla and caudal half of the nucleus gigantocellularis. There was less profuse degeneration in the caudal part of the nucleus pontis centralis caudalis and in the

(2) Psychiat. et neurol. 143:75-99, 1962.