tion of the posterior limb is the true correlate of the rolandic lesion. Thus, with the possible exception of Cas Schweigoffer, Dejerine's data is quite consistent with recent anatomical observations concerning the location of the pyramidal tract in the internal capsule which, in turn, is consistent with my findings using whole brain dissection.

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Arteriographic findings and neurologic symptoms

To the Editor: Thiele et al[1] demonstrated that most patients with either TIA or completed stroke have ulcerated or irregular atheroma in the ipsilateral carotid artery. This supports the widespread opinion that artery-to-artery embolism is the most common cause of ischemic disease in the anterior circulation. We should not, however, conclude that carotid endarterectomy is the best therapy for all types of ischemic events in this area. We need to look at the natural history and therapeutic result for each clinical syndrome to utilize angiographic data for therapeutic decisions.

The patients who did best after surgical therapy in a large controlled study[2] were those who had TIA and unilateral carotid stenosis. The data are not as clear for the substantial group of patients with less than 50% stenosis and an ulcerated plaque; in this group, surgery has not been proven superior to medical therapy.

Finally, I believe that we should be especially cautious before assuming that surgical therapy is the best bet for patients with completed infarctions (fixed neurologic deficits). Thiele et al make a statement that is premature on the basis of present evidence: "Therefore, aggressive investigation is justified in these patients to identify those with a potential symptom-producing lesion in the carotid bifurcation, which may be amenable to surgical correction."[1] They did not state the severity of the fixed deficits in their series. I assume that they were small, because angiography is not ordinarily considered for patients with devastating hemispheric damage. Even so, the yield of lesions potentially amenable to surgery was lower in their group with fixed deficits, because 34% of this group had either occluded carotids or normal vessels. Surgically treatable lesions may be even less common in patients with fixed deficits who have never had a TIA.[3]

Furthermore, the benefit of surgery may be less after completed stroke than after TIA. Ninety percent of patients with fixed deficits in the series of Thiele et al had intracerebral abnormalities; merely performing a carotid endarterectomy might have had a less favorable impact on their prognosis than in patients with TIAs, among whom only 39% had intracerebral abnormalities. The natural history of carotid syndromes may differ as well: In two series,[4,5] TIA recurred more often than did RIND (reversible ischemic neurologic deficit). In some patients TIAs cease after a fixed deficit finally occurs.

Eventually, I hope that we can allocate patients to appropriate therapy groups—carotid surgery, intracranial bypass, drug treatment—based on knowledge of outcome for each clinical syndrome and each arteriographic configuration. Lacking this, we must not equate visible angiographic findings with probable therapeutic results. The authors of this paper have done a valuable service in pointing out some differences in the arterial structure of patients who suffer fixed deficits from those with TIAs. Data of this type may provide a foundation for studies to evaluate therapy.

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Reply from the Authors: Our study was not undertaken to determine the relative roles of surgical or medical therapy in the treatment of patients with carotid bifurcation atherosclerosis but rather to question whether lesions producing greater than 50% diameter

reduction were more likely to be associated with neurologic symptoms. This study clearly demonstrated that all degrees of stenosis are capable of producing symptoms, especially when there is evidence of irregularity or ulceration.

As noted by Dr. Faught, the degree of stenosis (determined arteriographically) is often used to select patients for surgical or medical therapy. This is, in part, related to the concern that strokes occur more commonly in patients with high- grade lesions which may progress to complete internal occlusion. Our study suggests that the converse (that patients with lesions less than 50% diameter reduction are not likely to develop strokes) is certainly not true and one of the determinant factors is the status of the cerebral circulation. It is therefore important, when studies of the natural history of carotid bifurcation atherosclerosis are being designed, to include patients with all degrees of disease at the bifurcation.

Because similar neurologic syndromes may occur in patients with markedly different pathology at the appropriate carotid bifurcation, the extent of disease at the carotid bifurcation is only one of the variables involved in the production of cerebral ischemia. As noted by us, the presence of disease in the siphon or absence of communicating vessels in the circle of Willis is probably also a significant variable and information regarding this factor should also be included in studies of the natural history. Lack of this information may be responsible for the conflicting reports of the natural history of carotid bifurcation disease.

With regard to patients with neurologic deficits, we included only those who demonstrated significant neurologic recovery in days or weeks. All the patients had neurologic signs for at least 24 hours. Since the pathogenesis of recurrent stroke is obscure, our study suggests that persistence of potential embolic lesions at the carotid bifurcation may be one mechanism. In our institution, the finding of such a lesion has served as an indication for carotid endarterectomy, although here again, as noted by Dr. Faught, data for or against such an approach are lacking. We suggest that patients with complete neurologic deficits and good recovery should have further study to determine the internal carotid artery on the appropriate side is patent or occluded. A study of patients classified purely on the basis of the clinical syndrome would be of little value because of the different degrees of pathology at the bifurcation.

We concur with Dr. Faught that the relative roles of surgical and medical therapy in the treatment of carotid bifurcation atherosclerosis remain extremely controversial. This is in part due to inadequate design of the studies which did not encompass variables that may have a significant impact on the natural history of cerebral ischemia.

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Corrections

"The neuropathy of abetalipoproteinemia" by Robert G. Miller, M.D., Christopher J. F. Davis, Ph.D., M.R.C.P., D. Roger Illingworth, M.D. Ph.D., and Walter Bradley, D.M., F.R.C.P., December 1980, p. 1286. The last line of the abstract should read, "On the basis of findings in three patients, we believe that large fibers in peripheral nerves are selectively affected and that weakness may be due to chronic partial denervation."

The affiliation for Drs. E. Kessler and D. Bergen, two of the authors of a letter to the editor, December 1980, p. 1338, should be changed to the Department of Neurological Sciences, Rush-Presbyterian-St. Luke's Medical Center.

book reviews

Muscles Alive: Their Functions Revealed by Electromyography, Ed. 4

by J.V. BASMAJIAN, 495 pp., ill., Baltimore, The Williams & Wilkins Company, 1978. $36.00

Textbooks of kinesiology generally present kinetic and structural aspects of movements with a heavy emphasis on biomechanics of bones, joints, and muscles and an emphasis on particular functions, such as walking, running, and jumping. Extensive reviews of anatomy and the forces acting at joints are usually included, with only limited reference to the electrophysiologic studies and their place in detailing the functions of individual muscles.

The fourth edition of *Muscles Alive,* a 495-page book by Dr. J.V. Basmajian, elegantly makes up for these deficits by describing the electrophysiology of individual muscles during specific movements, as defined in the many years of study by Dr. Basmajian and his coworkers. While the text does not provide the broad range of information found in many textbooks of kinesiology, the unique combination of 4 chapters of techniques and 12 chapters of detailed description of individual muscle function makes this book a valuable adjunct to standard texts. This edition has added new information that Dr. Basmajian and his co-workers have accumulated in recent years, including 400 new references. A new chapter on the electromyographic signal is an excellent basic summary of the electrical activity of motor unit potentials; other new chapters on the kinesiology of eye muscles and the larynx are valuable additions. The text limits itself to the normal electrophysiology of muscles and is not meant to be a textbook of diagnostic clinical electromyography. There is only limited information on nerve conduction studies and no discussion of disease entities.

Although the book's excellent index makes it a useful reference source, there are some surprising publication errors in a fourth edition, such as inverted illustrations and repetitions of entire paragraphs. Nonetheless, *Muscles Alive* is a useful repository of information on the electrical activity of individual muscles during the normal functions of rest, posture, and movement.

JASPER R. DAUBE, M.D.

Experimental and Clinical Neurotoxicology

edited by PETER S. SPENCER and HERBERT H. SCHAUMBURG, 929 pp., ill., Baltimore, The Williams & Wilkins Company, 1980. $110.00

Recognition that a newly published book is clearly a benchmark of excellence in its field and destined to become a classic reference is usually an easy and obvious determination. However, when a new book's publication has the further impact of synthesizing and consolidating a whole body of knowledge so as to literally create and legitimize a new field in clinical neurology, it is indeed an extraordinary, prescient contribution to the literature. *Experimental and Clinical Neurotoxicology,* edited by Spencer and Schaumburg, is such a brilliant new beacon on the horizon, and its light will shine bright, far, and well into the future in providing an important compendium of information for the newly forged disciplines of experimental and clinical neurotoxicology. The book is divided logically into five major sections: Targets and Classification of Neurotoxic Substances: Pathophysiological Aspects of Toxic-Metabolic Disease; Specific Environmental Neurotoxins; Applied Neurotoxicology; and Public Issues and Neurotoxicology. Scholars actively involved in each of these areas of endeavor have contributed richly factual and authoritative chapters.

Price and Griffin provide a clear analysis of the regional pathology of motor neurons and Schwann cells for a series of toxic agents, the mechanism of their action, and a graphic quantitative description of altered axoplasmic flow for several agents. Spencer and Schaumburg describe a lucid classification of the various diseases based on a morphologic model, citing specific cellular target sites that are characteristic for the different classes of toxins. Powell, Myers, and Lampert describe their unique experimental approach in measuring endoneural fluid pressures as an index of intraneural edema and its attendant pathologic consequences due to specific toxins. Mendell and Sahenk describe altered axoplasmic transport profiles in various toxic-induced polyneuropathies, and give the reader a real feeling for the altered dynamics of neuronal cell processing and/or axoplasmic flow. The N-hexane and methyl-M-butyl ketone story is graphically described in two fine chapters, one by Spencer, Couri, and Schaumburg and another by Normal Allen who originally described the endemic outbreak in 1973 of peripheral neuropathy in an industrial plant, caused by ketone exposure. Essential chapters, well written and sufficiently detailed, describe the neurotoxicity of selected metals, organophosphorus compounds, selected drugs, selected chemicals, and biological toxins. A glimpse into future research approaches giving a molecular explanation for the effects of these toxic agents is provided in two elegant chapters, by Trapp and Richelson and by Damstra and Bondy. Various specific parameters of the biochemical basis of neural differentiation are described, citing sensitive and neural specific enzyme systems for possible future neurotoxicologic investigations and development of useful diagnostic bioassays.

It is fitting and appropriate that the publication of this book edited by Spencer and Schaumburg coincides with the establishment of their new Institute of Neurotoxicology at the Albert Einstein College of Medicine and their prominent involvement in a new journal devoted to this field. They have set the keel for their institute, have launched definitively the field of neurotoxicology for both experimental and clinical neurologists with this publication, and have set a course which will alert modern civilization more readily to the perils of neurotoxins. It is hoped it will lead to improved public measures of containment and research into their mech-

anisms of action. This book is a comprehensive and scholarly statement of current knowledge of neurotoxicology and is an indispensible map for anyone planning to travel in these charted or uncharted waters. In my view, this book will soon join the esteemed rank of now classic books published previously by such persons as H. Houston Merritt, Goodman and Gilman, Cecil and Loeb, Lehninger, and even Rachel Carson.

ROGER N. ROSENBERG, M.D.

Other Releases

Radiation Damage to the Nervous System: A Delayed Therapeutic Hazard
edited by HARVEY A. GILBERT and A. ROBERT KAGEN, 225 pp., ill., New York, Raven Press, 1980. $25.00

Bromocriptine: A Clinical and Pharmacological Review
edited by MICHAEL O. THORNER, E. FLUCKIGER, and DONALD B. CALNE, 190 pp., ill., New York, Raven Press, 1980. $22.00

Progress in Clinical Neurophysiology: Spinal and Supraspinal Mechanisms of Voluntary Motor Control and Locomotion
edited by J. E. DESMEDT, 374 pp., ill., Basel, S. Karger Medical and Scientific Publications, 1980. $82.75

Phenytoin-Induced Teratology and Gingival Pathology
edited by THOMAS M. HASSELL, MALCOLM C. JOHNSTON, and KENNETH H. DUDLEY, 252 pp., ill., New York, Raven Press, 1980. $25.00

Neuromotor Examination of the Limbs
by MARIO P. SMORTO and JOHN V. BASMAJIAN, 113 pp., ill., Baltimore, The Williams & Wilkins Company. 1980

The Musculoskeletal System in Health and Disease
by CORNELIUS ROSSE and D. KAY CLAWSON, 425 pp., ill., Hagerstown, MD, Harper and Row, 1980. $35.00

Diagnostic Reference Index of Clinical Neurology
by PASQUALE F. FINELLI, M.D., 380 pp., Baltimore, The Williams &Wilkins Company, 1980. $33.00

Alcoholic Korsakoff's Syndrome
by NELSON BUTTERS and LAIRD S. CERMAK, 188 pp., ill., New York, Academic Press, 1980. $16.50

Management of Acute Head Injuries
by RICHARD HAYWARD, 106 pp., ill., Oxford, Blackwell Scientific Publications, 1980. $13.75

Essentials of Neurosurgery
by RICHARD HAYWARD, 275 pp., ill., Oxford, Blackwell Scientific Publications, 1980. $26.50

CNS Complications of Malignant Disease
edited by J. M. A. WHITEHOUSE and H. E. M. KAY, 419 pp., ill., Baltimore, University Park Press, 1980. $49.50

The following books represent the proceedings of conferences, congresses, symposia, and workshops:

Evoked Potentials
edited by COLIN BARBER, 614 pp., ill., Baltimore, University Park Press, 1980. $34.50

Biochemistry of Dementia
edited by P. J. ROBERTS, 272 pp., ill., New York, John Wiley & Sons, Inc., 1980. $46.00

The Inferior Olivary Nucleus: Anatomy and Physiology
edited by JACQUES COURVILLE, CLAUDE de MONTIGNY and YVES LAMARRE, 407 pp., ill., New York, Raven Press, 1980. $49.00

Cerebral Circulation and Neurotransmitters
edited by ANDRE BES and GILLES GÉRAUD, 268 pp., ill., Amsterdam, Excerpta Medica, 1980. $48.75

Search for the Cause of Multiple Sclerosis and Other Chronic Disease of the Central Nervous System
edited by A. BOESE, 502 pp., ill., Deerfield Beach, FL, Verlag Chemie International Inc., 1980. $55.00 (paper)

Limbic Epilepsy and the Dyscontrol Syndrome
edited by M. GIRGIS and L. G. KILOH, 282 pp., ill., Amsterdam, Elsevier/North Holland, 1980. $48.75.

Corrections

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