19

Posttraumatic Syndrome: Critique

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To facilitate the discussion of the papers relating to the posttraumatic syndrome, I shall attempt to group them by general subject rather than to discuss them in the order in which they appear.

By means of an isotopic scan technique, Dr. Taylor reported a persistent slowing of the cerebral blood flow as a late effect of head injury. In his excellent review of the literature, he discussed the possible cause of this phenomenon. Two types of brain lesions were thought to be related to the circulatory disturbance, mainly multiple discrete infarcts scattered throughout the brain and diffuse areas of torn axons as described by Dr. Strich in Part Five (Chapter 52) of this volume. In and about the infarcted areas, vascular abnormalities—both anatomical and functional—are present. In this regard, Pappenheimer's report[1] of carbon dioxide-sensitive receptors in the reticular formation, scattered throughout the brain and directly accessible to profusion, may be pertinent. Although this hypothesis may not be completely established it provides an explanation for the intracerebral autonomic regulation of the brain circulation. Dr. Taylor also referred to the use of artificial hyperventilation for the relief of cerebral edema, a simple technique with great therapeutic value.

Dr. Jacobson's report related to minor traumas, particularly of the neck, a field which has had very little systematic research. Because the number of patients with minor trauma is so great and has such great socioeconomic importance, the subject is of even greater interest than the major injuries. In acute traumatic states, Dr. Jacobson differentiates "neck trauma without sensorium defect" and the "brain lesions due to trauma." He relates the severity of brain injury to the duration of amnesia and notes that in this classification neck trauma is considered the least severe brain injury but that, if associated with major intracranial pathology, it may be the most severe.

In a long-term study of 347 patients, Dr. Jacobson noted four categories of symptoms: headache, vertigo, disturbances of psychic functions, and disturbances of mental function. Headache was found in 78 percent of the cases, less frequently in the most severe and in the least severe cases. Two months after trauma most patients had recovered from headache and all were free of symptoms within four years. An early neurotic background seemed particularly important in the persistence of the headaches.

Dr. Jacobson thought that vertigo was related to damage of the inner ear rather than to brain stem lesions, since there is no increase in the vertigo of the patients with amnesia of longer duration. This symptom was found in nearly 50 percent of his cases and lasted longer than headache. Patients with severe head injuries had a somewhat

lower percentage of vertigo, and patients with neck injury had a relatively higher percentage (28%).

Disturbances of psychic function are found in 42 percent of the group and were directly related to the severity of injury (from 11% to 50%).

The psychological disturbances consisted mainly of neurotic symptoms and were more commonly found in the severely injured individuals and in older people.

The clinical symptomatology and the course of severely brain-injured patients is described by Dr. van der Zwan. His observations relate to cases which are usually classified as the apallic syndrome. The course typically begins with acute midbrain, bulbar, or brain stem signs and symptoms and terminates in a chronic brain syndrome or occasionally without any defects. It should be emphasized that in these severe cases the rehabilitation treatment must be started immediately and must be continued in intensive care wards throughout the prolonged comatose stage. At times the results are surprisingly good.

Dr. Black reports on the clinical symptomatology and the course of brain-injured children. It seems to me that in his series the severity of the brain lesions varied greatly, some being very serious and some very slight. The percentage of hyperkinesis is remarkably high. Personally, I think that a definite diagnosis of the acute stage, which Dr. Black did not stress, would enable one to make a better prognosis.

Dr. Friedman emphasizes the common occurrence of headache as a sequelae of a head injury and notes its persistence even with mild trauma. He postulates that in most cases it is only one of many symptoms in the complex posttraumatic state which occurs in 40 to 60 percent of head injuries. He discusses the pathogenesis of posttraumatic headache based upon his own experience and the extensive literature on the subject. The headache is often accompanied by a neurotic reaction which tends to prolong the headache. Other psychological factors such as compensation may have similar affects. Dr. Friedman outlined his pharmacological and psychotherapeutic treatment for such cases.

In a brief communication, Dr. Velasco-Suarez presents a theory to explain the progressive ventricular dilatation which develops in the severe cases. He emphasizes that intracerebral hemorrhage and secondary lesions of the white matter due to diffuse cerebral edema and primary secondary hypoxic lesions of the cortex and basal ganglia give rise to a shrinkage of the damaged tissue, which produces an enlargement of the ventricle and atrophy of the cortex.

Dr. Toglia's discussion of dizziness associated with whiplash and closed head injuries points out the high incidence of this symptom after a brain injury. He suggests that it is due to transient vertebral artery compression with ischemia of the brain stem and cerebellum. He has systematically examined the vestibular function, using electronystagmography. In 65 percent of the patients he found abnormalities; 32 percent had unilateral impairment and 14 percent bilateral changes. He differentiated three types of labyrinthine damage: (1) labyrinthine concussion, (2) paroxysmal positional vertigo, and (3) labyrinthine contusion. On the other hand, he was not able to demonstrate a relationship between the intensity of the dizziness and the severity of the trauma and the degree of vestibular abnormality. He emphasized the necessity of utilizing electronystagmography in patients with minor head injuries and whiplashes, since its use may allow a differentiation of organic reactions from purely neurotic ones.

Dr. Ishii also emphasized the involvement of neck structures as a factor in the posttraumatic syndrome. As a result of systematic examinations and experimental studies with electroencephalography, nystagmography, local stimulation, and local infiltration with procaine and saline as well as centrophenoxine, he concluded that research into the reactions of the vascular structures of the neck may give a clue to the patho-

genesis of the cervical discomfort in whiplash and cerebral injuries.

Dr. Silfverskiöld emphasized the necessity of an aggressive physiotherapeutic program in patients with chronic neurotic symptoms as a result of head injuries.

Based upon his extensive experience with the psychiatric manifestations of brain injuries, Dr. Ota divides the symptoms into psychogenic complications, endogenous psychoses, and posttraumatic sequelae. He believes that the degree of injury and the localization of brain lesions are different in these states. The posttraumatic syndromes he differentiates into epilepsy, psychotic states, contusion, and neurotic complications. However, he admits that it is not easy to make a clear-cut distinction between the acute and chronic stages. The most significant psychiatric findings were psychic changes and neurotic complications. The psychic changes related to a frontal lobe syndrome and the neurotic complications were particularly hypochondriasis, conversion neurotic symptoms, and obsessions. The role of compensation in the psychodynamics of these complications seems to be very important. The psychotic states occurred rarely. Patients with neurological symptoms such as hemiplegia and hemianopsia were relatively few.

Dr. Hillbom discussed the posttraumatic Korsakoff syndrome on the basis of 29 patients. The main symptom related to disturbances in memory. It was interesting to note that in his cases the brain structures involved seem to relate to injuries to the sites where the memory processes are commonly thought to occur, namely bilateral injuries of limbic pathways, particularly their connections to the frontal lobe. He noted a diminution in the general intelligence, although he found it difficult to differentiate between dementia and the traumatic Korsakoff syndrome.

In another series of communications, precise psychological techniques were described for the localization of brain lesions. Dr. Milner showed that abnormalities, demonstrable by psychological testing, could have localizing value. Furthermore, Dr. Teuber showed that, by nonspecific and material specific tests, many different types of disturbances might be brought out: (1) impairment of overall brain function, (2) emotional disturbances, (3) the relationship of local lesions to total brain function, aphasia, et cetera, and (4) the differentiation of organic from neurotic manifestations.

Dr. Unterharnscheidt discussed the physical disturbances which occur when rotational, translational, and other forces are applied to the head, modifying the site and type of damage to the brain. These contributions give greater insight into the general pathology of head injuries.

In my general review of the topic of Part One, I wish to comment upon the use of the term "posttraumatic syndrome." This term should be confined to a clinical symptomatic unit resulting from brain or neck injury. However, the clinical term "syndrome" (a well-defined unit of various symptoms) seems to be too diffuse and might well be replaced by the term "posttraumatic state."

The second point that struck me as I reviewed the papers was related to the term "posttraumatic," which does not distinguish between the acute and subacute traumatic states. Thus, occasionally in these papers the acute period was not sharply enough separated from the subacute or even the chronic phases, with the result that the defects described were, in some instances, actually those of the acute phase. I believe that the posttraumatic syndrome should relate only to the chronic phase.

From the clinical point of view, I think that one might examine the posttraumatic syndrome from two angles: (1) from the standpoint of the minor head injuries such as so-called whiplash injuries and (2) from the standpoint of severe head injuries. In Dr. Strich's excellent demonstrations in Part Five of this volume, we will see the typical findings in severe head injuries; Dr. van der Zwan, for one, has elucidated the clinical

symptomatology of this state. This well-defined syndrome has been called by various names. I would suggest that this clinical condition be called the "apallic syndrome," the term originally introduced by Kretschmer[2] in 1940. This term has come into common usage in European countries and many other parts of the world. If it were universally accepted, perhaps some of the confusion that surrounds the clinical picture would be eliminated.

Difficulties still arise in the diagnostic classification of patients with head injuries of intermediate severity, in both the acute and chronic states. To establish the degree of neurologic involvement, one should carry out intensive clinical examinations and laboratory studies by electroencephalography, pneumoencephalography, electromyography, et cetera. By these diagnostic procedures, we may early establish the extent and severity of the lesions in various parts of the nervous system, so that adequate therapeutic measures may be instigated and continued throughout the course of the condition. Only by such a broad program can the brain-injured patient be adequately rehabilitated.

References

1. Fencl, V., Miller, T. B., and Pappenheimer, J. R.: The respiratory response to disturbances of acid-base balance. In Caveness, W. F. and Walker, A. E. (Eds.): *Head Injury Conference Proceedings.* Philadelphia, Lippincott, 1966.
2. Kretschmer, E.: Das apallische Syndrom. *Z Ges Neurol Psychiat, 169:* 576, 1940.