THE ROLE OF THE RETICULAR FORMATION IN THE COMA OF HEAD INJURY*

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HEAD injury has been a major problem of man for centuries. The first written account of the treatment of such appeared in the Edwin Smith Papyrus around the 17th century, B.C. Thirty-four centuries later in 1833, Jean Louis Petit was the first to recognize cerebral concussion as a separate entity and distinct from brain compression by hemorrhage. Cerebral concussion was first defined by Benjamin Bell in 1873 when he said, “Every affection of the head attended with stupefaction, when it appears as the immediate consequence of external violence, and when no mark of injury is discovered, is in general supposed to proceed from commotion or concussion of the brain, by which is meant such a derangement of this organ as obstructs its natural and useful functions, without producing such obvious effects on it as to render it capable of having its real nature ascertained by dissection.” Little actual refinement in this definition of cerebral concussion has occurred in the subsequent 80 years.12 The physiologic basis for the clinical phenomenon of cerebral concussion is not completely understood even though closed head injury is an increasingly common occurrence.

The mechanics of brain trauma were thoroughly studied by Denny-Brown and Russell14,15 who established the laboratory criteria for concussion and made a distinction between acceleration and compression concussion. Holbourn,12 studying the physical forces involved in head injury, showed that the rotational rather than linear acceleration forces are the main cause of brain damage in head injury, these forces being responsible for “contre-coup” damage, hemorrhage, and “probably” concussion. Gurdjian et al.11 analyzed physical factors in concussion and demonstrated that acceleration, deceleration and compression all result in measurable elevation of the intracranial pressure. They concluded that sudden increase in intracranial pressure caused concussion and that the shorter the time duration of the increased intracranial pressure, the higher the pressure necessary to cause a concussive effect.

Investigations of the physiological basis of head injury have proceeded slowly along two lines—the measurement of biochemical changes and meas-
measurement of cerebral electrical activity alterations. Bornstein\textsuperscript{2} as well as Tower and McEachern\textsuperscript{16} have shown that acetylcholine appears in the cerebrospinal fluid following concussion and that it depresses integrated neuronal activity. Its effects can be counteracted by the use of anticholinergic drugs. Ward\textsuperscript{18} showed that atropine is clinically helpful in severe closed head injuries, and Ruge\textsuperscript{14} substantiated experimentally that atropine prevents the muscarinic effects of the acetylcholine that appears following trauma of the central nervous system.

The changes in electrical activity of the brain following head injury were studied by Walker, Kollros, and Case\textsuperscript{17} who felt that the unconsciousness of concussion was the result of sudden intense neuronal discharge of the brain resembling a seizure discharge. Foltz, Jenkner, and Ward\textsuperscript{6} were unable to support this conclusion since their electroencephalographic studies during experimental concussion showed flattening of the cortical electrogram and changes that were otherwise surprisingly minimal. Their subcortical recordings, likewise, showed the same depression and flattening. The one dramatic exception to this was the activity recorded from the medial reticular formation which always showed far greater and longer electrical depression than other structures studied.

In view of this finding, certain major points concerning the function of this central brain stem area are pertinent: (i) electrical potentials evoked by peripheral stimulation may be corticopetally conducted not only through the classical lemniscal system and thalamic relays, but also are conducted through this central brain stem and medial thalamus;\textsuperscript{9} (ii) electrical stimulation in this area, from which potentials evoked by peripheral stimulation can be recorded, will cause the so-called “arousal” reaction in the dormant animal and is associated with changes in the electroencephalogram from a sleeping to a waking state;\textsuperscript{19} (iii) electrolytic lesions of this area produced in the experimental animal cause prolonged coma and unreactivity to environmental stimuli.\textsuperscript{8}

This central region in the cephalic tegmentum is thus intimately concerned with consciousness. Since this region receives impulses from the classical sensory pathways, it is presumed that this continuous sensory bombardment “drives” the reticular activating system and thus maintains the conscious state. Reduction in this sensory input results in spontaneous sleep from which the subject may be aroused by appropriate increase in the sensory input. Abolition of this sensory driving results in coma.

It was postulated that the unconsciousness produced in cerebral concussion could be based on changes that prevent the normal sensory “driving” of the reticular formation. This could occur by direct neuronal depression of the cells in the reticular core, or by loss of collateral afferent conduction into the central core from the main ascending sensory pathways. Since initial experimental work has shown that the total electrical activity in this area is reduced,\textsuperscript{6} the present effort was intended to determine the effect of concussion on the reactivity of the reticular core to certain of the sensory stimuli which ordinarily “drive” the reticular formation.