tomedullary junction. Furthermore, lesions are often not confined to vascular territories and presence of lesions at other sites may cloud brainstem symptomatology. The manifestations of traumatic brainstem lesions are best considered under the headings of level of consciousness, respiratory pattern, pupillary reactions, ocular movements, cranial nerve palsies and the descending motor- and sympathetic pathways.

Patients with primary brainstem damage who reach the hospital alive usually have diffuse axonal injury and are in deep coma, with extensor posturing, anisocoria and impaired oculovestibular reflexes. Primary brainstem injury is rarely associated with cervico-medullary damage with respiratory arrest, hypotension, tetraplegia and perioral sensory loss and anaesthesia extending from C1–C4. The development of worsening clinical signs in secondary brainstem damage ranges from alteration of alertness and respiration with normal reactive pupils to disconjugate eye movements, absent pupillary reactions, bilateral extensor motor responses and finally flaccid motor responses, cardiorespiratory disturbances and death.

The importance of the clinical symptomatology of primary and secondary traumatic brainstem injury is worth mentioning because different treatment options can be assigned to the immediate treatment in the acute stages.

Morphology and pathogenesis of traumatic Brainstem lesions

K. A. Jellinger
Vienna, Austria

Brainstem lesions are important factors for the outcome of craniocerebral injuries, their incidence in fatal head trauma ranging from 15 to 60%. Primary traumatic lesions including ruptures, tears, hemorrhages, and axonal lesions in midbrain and pontine tegmentum and cerebral peduncles, often associated with multiple craniospinal injuries, result from shearing strains, rotational tension and angular acceleration. They usually lead to rapid death, although small lesions may be survived longer and are then to be distinguished from secondary brainstem lesions. These result from increased intracranial pressure with distorsion and herniation of the brain. They occur in 53% of all fatal head injuries, most frequently in fatal outcome within 18 and 48 hours, and in 90% of long survivors with persistent vegetative state. They show a characteristic pattern: superficial necrosis of cerebral peduncles and dorsolateral tectum/tegmentum resulting from compression at the tentorial edge, vascular lesions due to arterial or venous disruption caused by antero-posterior elongation, angulation, caudal distortion or vascular compression. Their pattern often corresponds to vascular supply areas. In acute fatal injuries, brainstem lesions are often located in central or paramedian areas of upper brainstem; in long survivors they prefer the lateral or dorsolateral tegmentum and small paramedian areas, while infarcts in pontine basis and medulla are rare. Although the distinction between primary and secondary traumatic brainstem lesions may be difficult, they usually show a constant pattern with strong correlation to clinical signs and prognosis. Experimental reproduction of traumatic brainstem lesions has contributed to the elucidation of the deleterious cascade of events causing direct and indirect sequelae of mechanical injuries.

Neuroimaging of traumatic-brainstem lesions

G. Birbamer, F. Gerstenbrand
Staffelstein, Germany; Vienna, Austria

Deep brain lesions produced in closed head injury of acceleration/deceleration type, are characteristically localized in the "centro-axial" regions of the brain. The extent of small and non-hemorhagic lesions of the brainstem are often underestimated by CT.

Magnetic resonance imaging (MRI) has proven superior to CT in the evaluation of traumatic brain damage. The pattern of lesions detected by MRI correlated well with our and earlier neuropathological studies, suggesting a multifocal pathogenesis of severe traumatic brain injury (inner cerebral trauma).

The improved information about the degree and extent of traumatic brain damage may aid in therapeutic decision making and in predicting the prognosis.

Electrophysiological examinations in posttraumatic coma

E. Rumpl
Neurological Department of the LKH Klagenfurt, Klagenfurt, Austria

Electrophysiological methods used in comatose patients after head-brain injury are presented according to their clinical relevance. Methods, strongly impaired by sedative drugs are separated from methods which are not or little impaired by these drugs.

The most important method influenced by sedative drugs is the EEG, most frequently used in intensive care units. The EEG is of high diagnostic and prognostic value and useful information can be obtained from a single record. Continuous EEG-monitoring using compressed spectral array (CSA) assesses the dominant frequencies, their distribution and their amplitude better than convential EEG. In contrast CSA has difficulties in detecting paroxysmal events, such as spikes, brief seizures, burst suppression and PLEDs. During anesthesia the EEG shows an increase of fast activity at low, an increase of slow waves at high dosis, even reaching burst suppression pattern in the case of barbiturate coma.

Less interest was paid to study the brainstem reflexes, such as the blink or Masseter reflex, although both methods are of high prognostic value. However, especially the R2 response of the blink reflex is strongly impaired by sedative drugs.

In sedated patients in posttraumatic coma most frequently early somatosensory-evoked potentials and brainstem acoustic-evoked potentials are used. Both methods are of high prognostic significance. The diagnostic value is poor except in cases with primary brainstem injury and isolated brainstem death.

European Federation
of Neurological Societies

European Journal of Neurology

Official Journal
of the
European Federation
of Neurological
Societies

Volume 6 Supplement 3

September 1999

Published by
Lippincott Williams & Wilkins
Third Floor, 241 Borough High Street,
London SE1 1GB
Tel: (+44) 0171 940 7500, *Fax*: (+44) 0171 940 7575

Abstracts of the

4th Congress of the European Federation of Neurological Societies

Lisbon, Portugal
September 7–11, 1999

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