PATHOLOGY OF COMMUNICATION IN CLOSED HEAD TRAUMA:
A REVIEW

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Traumatic closed head injuries may cause a period of coma of variable duration and severity; in many cases, after the acute phase, there is a progressive evolution of the picture toward a recovery of vigilance, attention and contact with the environment. At this stage we observe communicative and motor deficits of various type (Held 1975). The aim of the language rehabilitation effort is to program specific therapeutic interventions in order: 1) to establish contact with the patient in an appropriate way, considering the clinical evolution and the nature of the present communicative deficit, 2) to determine the type of the linguistic, cognitive and amnestic syndromes, 3) to create environmental conditions, motivation, therapeutic devices and exercises for the resolution or reduction of the pathology, 4) to guide and counsel relatives and nursing professionals on the most adequate modalities of interaction with the patient (Arné 1975).

In the initial stage, the treatment will be aimed at facilitation of awakening and at remediation of vigilance and attentional impairments (Eyssetett 1975). Later it will become more specific and it will focus on the type of emerging linguistic or communication deficit (Levin 1981). If we consider also the evolution of the patient with head trauma, the type of intervention will be modified according to the progressive changes of the clinical situation and of the impairment (Mandleberg 1975-1976).

We are convinced that treatment should start early in the post-acute phase, with appropriate environmental modification of stimuli, and continue without interruption up to the point of social and eventually professional reintegration of the subject.

We would like moreover to specify that speech therapy rehabilitation should include the aspects of remediation of incorrect respiratory patterns or of feeding problems (chewing and swallowing rehabilitation), which are often important obstacles in the initial recovery stage from a prolonged period of coma.

Specifically returning to the linguistic disturbances of the head trauma
Patient, we should notice that they are often complex and strictly correlated with cognitive and mnemonic deficits of various types and degrees (Ducarme 1976). In fact, the presence of amnesia or behavioral disruption, due for example to a frontal lobe syndrome, makes it more difficult to classify the specific linguistic impairment observed. Trying to establish a taxonomy of such pathological components, we find it useful to distinguish the possible speech or language disturbances in the following three categories: 1) Pneumo-phono articulatory deficits, as for example is the case of the dysarthric syndrome, 2) Aphasic deficits, which are usually different from the ones observed in vascular lesions (Heilman 1971), 3) Complex cognitive-linguistic disturbances (Geschwind 1964). Such a subdivision does not exclude, naturally, that deficits of the different categories may coexist.

Examining the first category, we should add that, in addition to the various types of dysarthria described (spastic, flaccid, ataxic, ipo-kinetic, mixed) (Boldrini 1986), we can have voice impairments, both central and peripheral (dysphonia), pneumo-phonic incoordination, or oversegmentary disorders such as dysprosodia. Often we may encounter complex, mixed clinical pictures that cannot be easily labelled. A common characteristic of these forms, however, is that the central cerebral structure for linguistic processing is not directly involved in the pathology. Therefore language is substantially unimpaired, while only speech and articulatory production are altered for disruption of control over motor coordination.

The second group of observable deficits is the aphasia one (Levin 1976). Such impairments are usually different from the ones encountered in vascular lesions of the dominant hemisphere. In fact severe and persistent aphasia is not usually the case, unless gross anatomical lesions of language brain areas are present.

More often traumatic aphasia is transitory and rapidly and spontaneously evolves toward anomia (Weinstein 1963). The only deficit left may be the difficulty to evoke an appropriate lexicon, and the use of frequent circumlocutions. Other times, in case of large frontal lobe lesions, we may have prefrontal dynamic aphasia, with reduced linguistic initiative and speech. In other occasions, it is possible to collect examples of paraphasias, egophasias or reduced verbal comprehension.

The third and last group of linguistic disturbances really collects a number of faulty behaviors caused also by attentional deficits, by difficulty in rapidly processing information, or by memory problems, interwoven with cognitive and relational impairments (Weinstein 1962) (Lackner 1974). Some patients tend to be overly talkative and unable to coherently organize the different parts of their linguistic production; occasionally their ideas are confused and disconnected, with a possibility for confabulation or paranoid ideation.
We may have, in addition, erroneous interpretations of other people's language, for reduced or slowed capability of acoustic stimuli elaboration. The speech production of these patients may be syntactically bizarre or inappropriate, with an incomplete or inaccurate lexicon. We may also encounter reading problems (wrong semantic interpretation of texts, visual confusion, perseverations, reduced memory capacity), or writing disturbances that reflect the characters of verbal production (orthographic errors, syntactical or semantic substitutions). The analysis of language and speech in head trauma patients is, for all these reasons, difficult and complex. In many instances it is not possible to assign every subject to any one particular category, due to the overlapping of the deficits observed in the three areas of analysis. As a conclusion, we would like to make some comments on the therapeutical implications of our taxonomy.

In the area of pneu-phon-articulatory disturbances, therapeutical procedures used by our speech therapists are equivalent to the ones used for similar vascular or degenerative pathologies. Other linguistic impairments, both aphasic and non-aphasic, should be treated initially with a particular focus on reorganizing attention, memory and sensory processing of information. In many instances, in fact, inattention, reduced short-term memory and difficulty in rapidly processing complex auditory and visual stimuli make it impossible for the patient to correctly elaborate the meanings of linguistic messages. It will be also necessary to program cognitive rehabilitation of conceptual associations, categorization, logical organization of thought and reasoning. The more specific linguistic sessions will be dedicated to syntactic and semantic reorganization of speech and to reading and writing exercises.

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