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Severe Head Injury: What Can Neuropsychology Do?

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Introduction

Head injury (HI) is a major public health problem in most developed countries, causing brain damage, enormous medical efforts and high economic costs (for review see 1). Advances in emergency evacuation and neurosurgical management of HI have reduced mortality, resulting in a large population of chronically disabled survivors with multiple neurobehavioral deficits (2).This review briefly elucidates the present role of neuropsychology in subjects who have suffered severe craniocerebral trauma.

The neuropsychological approach

Neuropsychology evaluates the effects of brain damage on cognitive skills and intellectual functions, such as language, memory, visuospatial abilities, recognition processes, and executive functions, to name a few. Neuropsychological methods are functional, specific, and problem oriented: they investigate behavioral functions over time, not morphology like brain imaging or pathology; they attempt an individual exploration of behavior on the basis of personal background variables like age, premorbid intelligence, professional skills and personality; and they take each patient's disease variables specifically into account: the type of brain lesion, the character of the resulting behavioral deficit, its prognosis and approach to recovery. Although group studies have played a major role in evaluating behavioral syndromes and in developing standardized assessment procedures, neuropsychology has traditionally been engaged in in single case studies of brain damaged subjects. Several methodological and practical issues suggest that a single-case approach in neuropsychological diagnosis and treatment is preferable to the use of fixed test batteries and rehabilitation programs for a heterogeneous group like HI patients (3). The following survey presents two main involvements of neuropsychology in HI, namely the assessment and remediation procedures of cognitive impairment.

Behavioral deficits and their assessment in head injured patients

Contusions, penetrating injuries, hematomas, diffuse white matter injuries, brain edema and ischemic necrosis are the most frequent causes of a large range of cognitive impairments in HI. Especially in blunt HI, these lesions are most marked in the dorsolateral and orbitofrontal, anterior and mesial temporal, brainstem and callosal areas; however, many other lesions sites leading to behavioral deficits have also been described (1, 4). Different from stroke, a well investigated paradigm of single, "focal" brain injury, lesions in HI often tend to be less well localized, and are often multiple. Consequently, most HI patients have clusters of behavioral deficits resulting from multifocal frontal, temporal and diffuse brain injury. In the early period after HI cognitive evaluation is a complex, time consuming and arduous procedure due to lack of attention, cooperation and the

severity of impairment; it often results in the tentative diagnosis of multimodal, overlapping and partly interdependent deficits. However, during chronic stages of HI specific

and relatively well defined neuropsychological syndromes can be found in many patients. Table 1 summarizes the most frequent cognitive syndromes in head injured patients.

Table 1. Neuropsychological syndromes in severe head injury

Deficits of attention

Impairment of learning and memory

Aphasia

Impairments of motor speech

Apraxia

Frontal lobe syndromes

Visuoperceptual, visuospatial and constructive impairments

Other cognitive deficits (disorders of perception and recognition, disconnection syndromes etc.)

Affective, personality and psychosocial disturbances

Attentional deficits are basic, universal, long lasting sequelae of HI even in patients with mild brain damage; in many cases they present severe problems for assessment and therapy attempts (5). Inattention may become variably manifest like poor concentration or vigilance, mental fatigability, inability to focus on a selective target, or loss of sustained or supervisory attentional control. In addition, hemi-inattention or neglect, a syndrome where patients fail to explore or to respond to one half of space or of their body can also be found in HI subjects. Clinical measurements of attention include various psychometric tasks such as mental tracking or tests of serial addition (PASAT) requiring supervisory control, mental speed and information processing capacities (for review see 6). Screening for neglect includes bilateral simultaneous visual or tactile stimulation, cancellation and bisection tasks (7).

Posttraumatic amnesia and residual disturbances of learning are characteristic features of HI resulting from both diffuse and focal (temporal, diencephalic and frontal) brain lesions and leading to a state with disorientation, impaired memory for episodic events before (retrograde) and after (anterograde) injury (for review *see* 8; 9). Amnesia in HI patients is usually multimodal including verbal and nonverbal materials; it affects recall, recognition and temporal ordering of autobiographical, historical and ongoing facts, commonly abolishing younger more than old or very long term memories. In most HI patients, amnesia represents one of the main obstacles for a return to normal everyday life and employment. Assessment of amnesia includes learning, recall and recognition of working memory with different materials such as word lists (e.g. the California Verbal Learning Test), prose, associate word pairs, pictures and geometrical designs (for review see 6), but also everyday memory capacities like picture, face or name learning, remembering appointments and hidden belongings, or skill learning (the Rivermead Behavioral Memory Test, 10).

Aphasia has been demonstrated in the majority of severely head injured patients (for review see 11; 12; 2); it occurs predominantly in subjects with penetrating wounds or contusions of the dominant hemisphere and is often associated with focal neurological signs and posttraumatic seizures. Frequent features of traumatic aphasia are anomia or Wernicke's aphasia with fluent paraphasic speech, poor comprehension for oral and written language and impaired repetition. Broca's aphasia with non-fluent, agrammatic language seems to occur rather rarely in blunt HI, but may be found after left frontotemporal missile or stabbing injuries (13). Other, more subtle impairments of linguistic knowledge include word list generation (14), and discourse with tangential, confused or inappropriate language, lack of cohesion and informational content (11; 15). Aphasia is tested by use of various standardized examinations, e.g. the Aachener Aphasietest (16), comprising the assessment of spontaneous speech, comprehension, repetition, naming, reading and writing.

Dysarthria is a common articulatory disorder in HI patients; it often accompanies aphasia during the early stages and may persist after restoration of other language capacities (17; 18). Most traumatic dysarthria are mixed pseudobulbar-bulbar speech syndromes with spastic, rigid and ataxic components clinically presenting as poorly articulated, slow speech with impaired control of voice qualities, loudness, stress and timing (19); traumatic dysarthria usually results from multiple coexisting lesions in the cortex, white matter, basal ganglia, brainstem and cerebellum. Less frequent non-aphasic sequelae of HI are apraxia of speech, an articulatory disorder resulting from defective speech programming (20), traumatic mutism, a central impairment of phonation found frequently in early stages of recovery from HI (21), echolalia and stuttering. Evaluation of dysarthria includes the assessment of rate, loudness, voice, articulation, intelligibility, prosody, and respiration in spontaneous speech, repetition and reading aloud (17; 22).

Apraxia, by definition a deficit in planning and performing motor action is difficult to assess because of frequent concomitant lower level motor and object recognition deficits in HI patients; also, goal directed action is often disturbed by a coexisting dysexecutive or frontal lobe syndrome prevailing the patient to organize and perform multistep actions through appropriate action strategies and feedback control. Characteristics of apraxia from HI include general action disorganization, loss of action sequence, misuse of objects, recognition and execution deficits of symbolic movements, and poor imitation of single and multiple limb and orofacial movements (23). Apraxic patients display a number of typical movement errors, among them substitutions, omissions, perseverations and "body-part-as-object" errors. Apraxia is tested separately for limb, orofacial and axial movements using verbal commands or imitation procedures; during assessment of ideational praxis, patients are required to perform ecologically valid action sequences like preparing meals, mailing letters, etc. (24). Many head injured patients suffer a severe and long lasting *frontal lobe syndrome* with loss of spontaneity, disinhibited social and sexual behavior, perseveration, distractibility, impaired temporal orientation and Korsakow-like amnesia (25). Other, higher-order frontocognitive impairments are

poor adaptation to changing situations and a problem solving deficit; this so called "dysexecutive syndrome" (26) becomes especially apparent in novel situations which cannot be overcome by strong habitual responses but involve planning, decision making, strategy generation and execution, feedback-mechanisms and error correction. Some authors favor a behavioral and anatomical differentiation of frontal brain syndromes into an orbitofrontal syndrome with "pseudopsychopathic" features (i.e. lack of impulse control, irritability and/or hyperkinesia) and a fronto-convex, "pseudo-depressed" syndrome (slowness, apathy, diminished initiative, and indifference). The clinical evaluation of frontal brain syndromes is manifold and comprises e.g. word and design fluency, the Stroop test, conceptual thinking paradigms (e.g. proverb interpretation, Category Test), various sorting (e.g. Wisconsin Card Sorting Test), reasoning (e.g. Raven's Progressive Matrices) and problem solving tasks (for further reading see 6; 27). Finally, an analysis of psychopathological symptoms is crucial in frontal lobe syndromes. It is important to recognize that frontal injury can influence psychometric measurement of other, "non-frontal" functions as well because of these patients' inability to use acquired knowledge, their distractibility, perseverations, inflexibility and lack of mental effort.

Visuoperceptual, visuospatial and constructive abilities are frequently reduced in HI patients with right or left posterior, mostly occipital brain damage, among them visual acuity and visual fields, perception of color and angulation, stereopsis, or oculo-motor functions. An outline of measurements includes basic neuro-ophthalmologic functions, acuity thresholds, color vision, perimetry, and contrast sensitivity; useful clinical tests are line orientation (28), maze tasks, tests for geographical and personal orientation, figure copying and free drawing, and various assembling procedures for measuring three-dimensional constructive abilities (e.g. the Block Design subtest of the WAIS, 6).

Other cognitive deficits after HI include perceptual disorders of hearing and olfaction as late sequelae of skull fractures, impairments of visual recognition (prosopagnosia, visual agnosia, simultanagnosia) from occipital, temporal and parietal lesions, hemispheric (mostly subclinical) disconnection syndromes due to axonal injury in the corpus callosum (29), and rare disorders stemming from frontal injury such as the alien hand syndrome, or imitation and utilization behavior syndromes (30).

In addition to intellectual loss HI generally induces ***affective, personality and psychosocial disturbances,*** most of them associated with brain damage, but some possibly having a psychological and sociological basis (31; 32; 33). After regaining consciousness and in early remission periods HI patients often display variable transient psychoorganic syndromes including disorientation, disturbances of sleep-wakefulness cycle, hallucinations, increased psychomotor activity, marked aggressive behavior, mood disturbances including manic or depressive symptoms and general emotional instability. Patients in the chronic stage of HI often display personality changes, mostly subsumed under the term "frontal syndrome" with lack of concern, coarseness, euphoria, disinhibition and increased sexuality, emotional lability and loss of insight. Other frequent chronic psychic changes after severe HI are depression, loss of interest and motivation, increased somatic concern, apathy, indifference or emotional withdrawal; and the postconcussional syndrome including anxiety, insomnia,

inability to concentrate and cope with stress, and repeated complaints of headache, chronic fatigue, vertigo, drowsiness or blurred vision. In a high percentage of HI patients cognitive and personality changes and chronic disability induce psychosocial disturbances with negative impact on familial relationships, employment and general social life (34). Psychological changes in chronic HI require a carefully balanced analysis of the patient's neurological and cognitive findings, his psychological status, and his preexisting social and emotional situation. Guidelines to a psychological diagnosis which is hard to objectivize but sometimes of forensic interest, are "hard" clinical symptoms, behavioral observation and rating procedures (35), and a skillful psychiatric interview.

Cognitive remediation

A large and growing body of literature and the appearance of new facilities specially designed for cognitive rehabilitation indicate that the traditional view of neuropsychology has changed from a purely diagnostic to both, an assessment and an interventional tool. Cognitive therapy is impeded by a number of novel methodological difficulties, among them the selection of patients with promising outcome variables, the design of specific therapy programs with ecological validity, the evaluation of program efficacy, and many practical problems such as the planning, content, level and frequency of training sessions, the didactic approach, type of material and eventual employment of automated programs to be used. Absolute prerequisites of cognitive therapy are an analysis of patient and disease variables (36), initial and longitudinal monitoring of major cognitive deficits, planning of therapy goals, selection of training procedures and materials and documentation of recovery. To cope with the multidisciplinary aspects of cognitive remediation most therapy centers employ neurologists, psychologists, speech and language pathologists, ergo- and cognitotherapists for a cooperative therapy program. A small selection of remediation approaches with promising results in HI patients is listed below.

Methods for ***amelioration of attention disorders*** train the patient to scan, detect and respond to environmental signals and cues, to improve concentration on internal sources such as time estimation exercises, or to synchronize with complex rhythms, and have shown to correlate with psychometric and clinical variables (37). Other training procedures try to improve processing speed and attention by use of computerized choice reaction time tasks or paradigms requiring divided attention (38). Neglect therapy has adopted several approaches, mostly forms of visual and tactile awareness and exploratory training towards the neglected body parts or space region, including practical applications like picture description, dressing, shaving, eating and reading, and representational exercises like drawing, copying or mental imagery; other attempts of neglect therapy feature auditory feedback training (39), or the correction of the displaced subjective midline via vestibular stimulation (40).

Rehabilitation of memory disorders has become a major issue in neuropsychology (41; 8) and includes several approaches. Patients

with a severe amnestic syndrome will profit from organizing their surroundings, e.g. by external memory aids like alarms, biographical cues for personal and landmarks for local orientation, timetables and activity lists for the day, but also by maintaining the local set and a small number of caregiving persons as long as possible (42). More specific memory training aims at the development of mnemonic strategies, e.g. learning of face-name associations by use of affective and phonological cues, or will try to induce semantic elaboration via search for striking characteristics among the material to be recalled. Other strategies for recall of single items or names try to evoke visual imagination, use "the method of loci", concentrate on phonological cues like the initial letter, or embed to-be-learned items in short stories (41; 43; 44). In amnestics, recall of prose and other textual information has been shown to profit from activation of related knowledge, the generation of questions about the text, a profound discussion or some other detailed analysis of the relevant information (41). Other mnemonic remediation techniques include operant reinforcement schedules, cognitive retraining by teaching patients necessary skills rather than mnemonic strategies, procedural learning as a memory aid, or compensatory techniques (41; 43; 45).

Speech and language therapy is probably the most developed intervention procedure and also has a well-based knowledge of prognostic factors of recovery (for review see 46). Classical aphasia therapy includes e.g. stimulation therapy, where access to language modalities is facilitated in response to particular stimuli (47); Luria's approach trying to achieve recovery through reorganization of functional systems and transfer of affected functions to new structures; or neurolinguistic approaches like melodic intonation therapy or deblocking techniques (48; 49). Modern aphasia rehabilitation concentrates on two efforts: to recover modality specific deficits in comprehension, lexicon and semantics, phonology and syntax (50; 51; 52), and to improve the communicative abilities of aphasic patients, e.g. by the PACE-technique (53), group or family therapy (54; 55), or by computer-based aphasia treatment (56). Remediation of reading and writing disorders, or word finding difficulties follows separate therapeutic principles (57; 58). Compensation for dysarthria is achieved by making the speaker continuously monitoring his performance; by a step-by-step articulation training which includes speech rate modification, deliberate syllable-by-syllable production, consonant exaggeration, and practicing of difficult phonemes; and by adjustment of respiration, pitch, loudness, vocal qualities and prosody to improve audibility, intelligibility and emphasis (17; 59). Special methods have been developed for mixed and spastic dysarthria as found in head injured patients (60).

Though verified only in small groups, treatment of ***reasoning and problem solving disorders*** in brain injured patients can be fairly successful. Several recent studies used problem-solving training specifically designed to improve problem formulation, strategy specifically designed to improve problem formulation, strategy and alternatives generation, decision making and solution

verification (61). To enhance these capacities, patients were requested to produce goal-directed ideas, discriminate between relevant and irrelevant information, and to selectively combine problem related information in small group training sessions. Results indicated positive treatment effects as measured psychometrically by behavioral ratings and planning test scores. Other authors have tried similar programs, among them forms of motivational and awareness training, exercises to develop abstract reasoning, concept formation and productive thought for practical problems, and training to utilize cues and shift response sets (62). Alternative programs use forms of positive reinforcement, cognitive overlearning and comparable behavior modification techniques (63; 64).

Computer based retraining and sensory cueing methods have been elaborated for patients with visual field losses, other forms of visual imperceptions or lateral scanning disorders to regain basic visual perceptual functions, visual searching, but also more skill-demanding processes like reading, route finding or driving (for review see 65; 66). Finally, remediation procedures for visual recognition deficits like object and face recognition, visual closure, and spatial integration are now being developed, mostly in single-case studies (67).

Conclusions

As evident from this review, neuropsychology in patients suffering from HI is still strongly biased towards diagnosis and assessment procedures, compared to the rather short tradition and limited experience of neuropsychologial rehabilitation. Similarly, every attempt to improve the cognitive impairment of HI subjects shows that successful remediation is more difficult to initialize and perform, and also takes a lot more of endurance and patience than sole assessment of cognitively impaired patients. However, after decades of research in the fields of imaging, pathophysiology and diagnostic measurement, the time may now be ready to accept cognitive rehabilitation as one of the forthcoming issues, and to accept neuropsychology as a promising domain in the management of severe head injury.

References

1) Dacey RG and Jane JA. Craniocerebral trauma. In: Joynt RJ (ed.), Clinical Neurology, vol 3. Philadelphia: Lippincott. 1989.

2) Levin HS, Benton AL and Grossman RG. Neurobehavioral consequences of head injury. New York: Oxford Press. 1982.

3) Caramazza A. On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: the case for single-patient studies. Brain and Cognition 5, 41-66. 1986.

4) Levin HS, Amparo E, Eisenberg HM, et al. Magnetic resonance imaging and computerized tomography in relation to the neurobehavioral sequelae of mild to moderate head injuries. Journal of Neurosurgery 66, 706-713. 1987.

5) Gronwall D. Advances in the assessment of attention and information processing after head injury. In: Levin HS, Grafman J and Eisenberg HM (eds.), Neurobehavioral recovery from head injury. New York: Oxford Press. 1987.

6) Lezak MD. Neuropsychological assessment. New York: Oxford Press. 1983.

7) Halligan PW, Cockburn J and Wilson BA. The behavioral assessment of visual neglect. Neuropsychological Rehabilitation 1. 5-32. 1991.

8) Levin HS. Memory deficit after closed head injury. In: Boller F and Grafman J (eds.), Handbook of Neuropsychology vol 3. Amsterdam: Elsevier. 1989.

9) Baddeley A, Harris J, Sunderland A, Watts KP and Wilson BA. Closed head injury and memory. In: Levin HS, Grafman J and Eisenberg HM (eds.), Neurobehavioral recovery from head injury. New York: Oxford Press. 1987.

10) Wilson BA, Cockburn J and Baddeley A. The Rivermead Behavioral Memory Test. Reading: Thames Valley test Company. 1985.

11) Hartley LL and Levin HS. Linguistic deficits after closed head injury. Aphasiology 4, 353-370, 1990.

12) Levin HS, Grossman RG and Kelly PJ. Aphasic disorders in of patients with closed head injury. Journal Neurology, Neurosurgery and Psychiatry 39, 1062-1070. 1976.

13) Mohr JP, Weiss G, Caveness WF, et al. Language and motor deficits following penetrating head injury in Vietnam Neurology 30, 1273-1279. 1980.

14) Lohman T, Ziggas D and Pierce RS. Word fluency deficits on common categories by subjects with closed head injury. Aphasiology 3, 685-693. 1989.

15) Dennis M and Barnes MA. Knowing the meanin, getting the point, bridgingthe gap, and carrying the message: aspects of

discourse following closed head injury in childhood and adolescence. Brain and Language 39, 428-446. 1990.

16) Huber W, Poeck K, Weniger D, Willmes K. Der Aachener Aphasie-Test. Gottingen: Hogrefe. 1983.

17) Darley FL, Aronson AE and Brown JR. Motor speech disorders. Philadelphia: Saunders. 1975.

18) Levin HS. Aphasia in closed head injury. In: Sarno MT (ed.), Acquired Aphasia. New York: Academic Press, 1981.

19) Ziegler W and von Cramon D. Differentialdiagnose der traumatisch bedingten Dysarthrophonie. In: Springer L, Kattenbeck G (eds.), Aktuelle Beitrager zur Dysarthrophonie und Dysprosodie. Munchen: Tuduv. 1987.

20) Square-Storer P. Acquired apraxia of speech in adults. London: Taylor & Francis. 1989.

21) von Cramon D. Traumatic mutism and the subsequent reorganization of speech functions. Neuropsychologia 19, 801-805. 1981.

22) Schaferskupper P and von Cramon D. Untersuchung und Beurteilung zentraler StOrungen der Stimme und des Sprechens Teil 1: Artikulation. Die Sprachheilarbeit 30, 68-74. 1985.

23) Poeck K. Apraxie. In: Poeck K (ed.), Klinische Neuropsychologie, New York: Thieme. 1989.

24) Schwartz MF, Reed ES, Montgomery M, Palmer C and Meyer NH. The quantitative description of action disorganization after brain damage. Cognitive Neuropsychology 8, 381-414. 1991.

25) Perecman E. The frontal lobes revisited. New York: IRBN Press. 1987.

26) Shallice T. A specific impairment of planning. Phil.Trans. R.Soc.Lond.B. 298, 199-209. 1982.

27) von Cramon D. Planen und Handeln. In: von Cramon D, Zihl J (eds.) Neuropsychologische Rehabilitation. New York: Springer. 1988.

28) Benton AL, Varney R and Hamsher K deS. Visuospatial judgement: a clinical test. Archives of Neurology 35, 364-367. 1978.

29) Bogen JE. The callosal syndromes. In: Heilman KM and ork: Oxford Press. 1985.

30) Lhermitte F, Pillon B and Serdaru M. Human autonomy and the frontal lobes. I-Imitation and utilization behavior: a neuropsychological study of 75 patients. Annals of Neurology 19, 326-334. 1986.

31) Lishman WA. The psychiatric sequelae of head injury: a review. Psychological Medicine 3, 304-318. 1973.

32) Bond MR. The psychiatry of closed head injury. In: Brooks N (ed.), Closed head injury:psychological, social and family consequences. New York: Oxford Press. 1985

33) Prigatano GP. Personality and psychosocial consequences after head injury. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

34) N Brooks. Head injury and the family. In: Brooks N (ed.), Closed head injury:psychological, social and family consequences. New York: Oxford Press. 1985

35) Grant I and Alves W. Psychiatric and psychosocial disturbances in head injury. In: Levin HS, Grafman J and Eisenberg HM (eds.), Neurobehavioral recovery from head injury. New York: Oxford Press. 1987.

36) Gerstenbrand F. Rehabilitation of the head jury. In: Walker AE, CAveness WF and Critchley M (eds.), The late effects of head injury. Illinois: Springfield. 1969.

37) Ben-Yishay Y, Piasetsky E and Rattock J. A systematic method for ameliorating disorders in basic attention. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

38) Saring W. Aufmerksamkeit. In: von Cramon D, Zihl J (eds.) Neuropsychologische Rehabilitation. New York: Springer. 1988.

39) Robertson I and Cashman E. Auditory feedback for walking difficulties in a case of unilateral neglect: a pilot study. Neuropsychological Rehabilitation 1, 175-183. 1991.

40) Rubens AB. Caloric stimulation and unilateral visual neglect. Neurology 35, 1019-1024. 1985.

41) Wilson BA: Rehabilitation of memory. New York: Guilford Press. 1987.

42) Schuri U: Lernen and Gedachtnis. In: von Cramon D, Zihl J (eds.) Neuropsychologische Rehabilitation. New York: Springer. 1988.

43) O'Connor M and Cermak L. Rehabilitation of organic memory disorders. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

44) Gruneberg MM, Sykes RN and Hammond V.Face-name associations in learning-disabled adults:the use of a visual association strategy. Neuropsychological Rehabilitation 1, 113-116. 1991.

45) Glisky EL, Schacter DL and Tulving E. Computer learning by memory-impaired patients: acquisition and retention of complex knowledge. Neuropsychologia 24, 313-328. 1986.

46) Special issue: Recovery from aphasia. Aphasiology 6, 1992.

47) Sarno MT. Recovery and rehabilitation in aphasia. In: Sarno MT(ed.), Acquired Aphasia. New York: Academic Press, 1981.

48) Basso A. Approaches to neuropsychological rehabilitation: language disorders. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

49) von Stockert T. Theorie und Praxis der Aphasietherapie. Munchen: Fink. 1984.

50) Greitemann G. Sprache. In: von Cramon D, Zihl J (eds.) Neuropsychologische Rehabilitation. New York: Springer. 1988.

51) Weniger D and Springer **L**. Therapie der Aphasien. In: Poeck K (ed.), Klinische Neuropsychologie, New York: Thieme. 1989.

52) Peuser G. Aphasie. Munchen: Wilhelm Fink Verlag. 1978.

53) Davis GA and Wilcox MJ. Adult aphasia rehabilitation: applied pragmatics. San Diego: College-Hill. 1985.

54) Clinical forum: Family therapy for aphasics. Aphasiology 3/6. 1989.

55) Clinical forum: Group therapy for aphasics. Aphasiology 5/6. 1991.

56) Clinical forum: Computer-based aphasia treatment. Aphasiology 4/6. 1990.

57) De Langen EG. Lesen und Schreiben. In: von Cramon D, Zihl J (eds.) Neuropsychologische Rehabilitation. New York: Springer. 1988.

58) Davis A and Pring T. Therapy for word-finding deficits: more on the effects of semantic and phonological approaches to treatment with dysphasic patients. Neuropsychological Rehabilitation 1, 135-145. 1991.

59) Berry WR. Clinical dysarthria. San Diego: College-Hill. 1983.

60) Mitto H and Legart L. Die Behandlung der spastischen Dysarthrophonie. In: Springer **L**, Kattenbeck G (eds.), Aktuelle Beitrager zur Dysarthrophonie und Dysprosodie. Munchen:Tuduv. 1987.

61) von Cramon D, Matthes-von Cramon G and Mai N. Problem-solving deficits in brain-injured patients: a therapeutic approach. Neuropsychological Rehabilitation 1, 45-64. 1991.

62) Goldstein FC and Levin HS. Disorders of reasoning and problem-solving ability. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

64) Alderman N and Ward A. Behavioral treatment of the dysexecutive syndrome: reduction of the repetitive speech using response cost and cognitive overlearning. Neuropsychological Rehabilitation 1, 65-80.1991.

65) Wood RL. Brain injury rehabilitation: a neurobehavioral approach. London: Croom Helm. 1987.

66) Gianutsos R and Matheson P. The rehabilitation of visual perceptual disorders attributable to brain injury. In: Meier M, Benton A and Diller L (eds.), Neuropsychological Rehabilitation. New York: Guilford Press. 1987.

67) Zihl J. Zur Behandlung von Patienten mit homonymen Gesichtsfeldst8rungen. Zeitschrift fur Neuropsychologie 2, 95-101. 1990.

68) Humphreys GW and Riddoch J. To see but not to see. A case study of visual agnosia. London: Lawrence Eribaum. 1987.

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526. **Kenda Miran F; Fras Zlatko**

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