ECRONICON OPEN ACCESS

EC NEUROLOGY
Editorial

Cognitive Recovery after Acquired Brain Injury - Neurocognitive Mechanisms and their Clinical Implications

Jesper Mogensen*

The Unit for Cognitive Neuroscience (UCN), Department of Psychology, University of Copenhagen, Denmark

***Corresponding Author**: Jesper Mogensen, The Unit for Cognitive Neuroscience (UCN), Department of Psychology, University of Copenhagen, Oester Farimagsgade 2A, DK-1353 Copenhagen K, Denmark.

Received: September 21, 2016; **Published:** September 27, 2016

Acquired brain injury (ABI) (of vascular origin or in the form of traumatic brain injury) almost always results in a certain level of sensory, motor or cognitive impairments. All such posttraumatic symptoms represent a significant burden on the patient as well as the family and friends of the patient. Additionally, the consequences of ABI are a major societal burden regarding healthcare, in the form of potential necessity of long-term nursing home housing etc. All types of ABI-associated impairments may represent severe burdens on the future life of the patient. Cognitive impairments, however, appear to have the greatest impact regarding such factors as quality of life, ability to return to independent living and potentially even return to work [1]. Fortunately, the posttraumatic cognitive symptoms will in most instances (especially if adequate cognitive rehabilitative training is administered) diminish significantly and the patient may even reach a level of "full recovery" - defined as a cognitive proficiency similar to what was seen pretraumatically [2-11].

Clinically, the potential of a pronounced cognitive recovery after ABI is encouraging and the administration of cognitive rehabilitative training should never be neglected. The relatively successful clinical results are, however, mostly achieved on a backdrop of missing conceptual frameworks and a lack of theoretical models. The theoretical as well as clinical need for improved conceptual clarifications and theoretical frameworks has been emphasized regarding recovery processes within both the motor [12] and cognitive [13,14] domains.

Frequently, a distinction is made between on the one hand the posttraumatic cognitive recovery associated with rehabilitative training and on the other hand what is termed "spontaneous recovery" [15]. Implicitly (and often explicitly) the spontaneous recovery is taken to be the result of experience-independent processes. A number of such "automatic" processes after ABI do exist. One example is the disappearance of an injury-associated "penumbra". Penumbras normally disappear spontaneously and such a disappearance can be associated with a return to normal functional performance within the part of the brain initially affected by the penumbra [16]. It should, however, be remembered that in parallel to such experience-independent processes, the initial posttraumatic period (when most of the "spontaneous" recovery is seen) is likely also to be a period of various kinds of "informal training". Formal cognitive training may not occur during this period but the patient (dependent on her/his general condition) is likely to engage in for instance attempts to communicate, orientation towards the environment, attempts to recognise individuals etc. All such (more or less successful) cognitive activities and the associated informal feedback regarding the success or failure of such attempts represent an informal training that will contribute to the recovery process (see below regarding the importance of all aspects of post-ABI experiences).

Another distinction often made regarding both ABI and other neurological pathologies [17] is the one between recovery and compensation. The term recovery is sometimes (e.g. by the present author) used to indicate any reduction or disappearance of symptoms. But when contrasted with compensation, recovery is mainly taken to indicate a re-creation of what was originally lost to injury [12,18]. Levin., *et al*. [12] have emphasized the importance of clarifying at a number of levels of analysis the relative degree of compensation and recovery during motor rehabilitation after stroke. And a similar analysis is essential regarding cognitive rehabilitation after any type of ABI [19].

In order to address such issues in a meaningful way and in order to allow clinical efforts to be based on a more solid foundation - improved neurocognitive models of the processes mediating cognitive improvements after ABI are essential [19]. Traditional "modular" theories [20-22] are unable to account for the reduction and potentially even elimination of cognitive symptoms. Most connectionist models [23-26] are better equipped to explain such posttraumatic processes but fail to account for the well-documented functional localization in the uninjured brain. In contrast, the REF (Reorganization of Elementary Functions) Model [18,19,27-31] is able to account for both the functional localization and the rehabilitation-related processes. It does so by being based on connectionist networks but also having a "modular" aspect in the form of strictly localized (and never re-created) Elementary Functions (EFs). EFs are not the traditional cognitive functions but relatively low level information processing elements. Each EF typically contributes to several or even many cognitive domains. EFs are via experience mediated reorganization of the connectivity between the neural substrate of the EFs organized (and potentially re-organized) into the actual mechanism of the traditionally defined cognitive functions: The Algorithmic Strategies (ASs). During rehabilitative training after ABI, the EFs of the spared parts of the brain are re-organized into new ASs - ASs potentially able to allow cognitive processes of similar proficiency to what was seen pre-traumatically. As argued elsewhere [18] such mechanisms do not represent a re-creation of what was lost. But the clinical outcome may be a patient with different but as proficient mechanisms of cognition (compared to the pretraumatic situation). Thus, rehabilitation may be claimed to be "compensational". But then compensation has to be seen as something different rather than something inferior. Conceptualizing the rehabilitative process in this way has a number of important clinical implications. Space only allows a couple of examples to be presented here.

Since cognitive rehabilitation after ABI is mediated by a reorganization of the spared regions of the brain rather than a re-creation of what has been lost, the most successful clinical procedures may not be the ones directly attempting to train what has been lost. A striking example can be found in the treatment of ABI associated hemispatial neglect. While little or no success is achieved directly training the impaired attentional processes the prism adaptation therapy (PAT) may have positive and lasting effects [32] in spite of not addressing attentional processes directly.

It is also important to realize that these reorganizational processes may sometimes to a surprising degree depend upon what may appear to be unimportant aspects of a training procedure. For instance, a computerized version of the PAT [33] only provided therapeutic results if the feedback to the patient was provided in the form of a virtual finger shown on the screen but not if similar feedback was given in the form of an "X" in the same screen-position. Such results emphasize the need for proper development and evaluation of computer-based rehabilitative methods [34] and an openness regarding what constitutes the crucial aspects of any rehabilitative procedure.

Further development of neurocognitive models of both the normal brain and the mechanisms of cognitive recovery after ABI is needed. And clinical practice also needs to implement the insights growing out of such theoretical and conceptual developments.

Bibliography

1. Moore AD and Stambrook M. "Cognitive moderators of outcome following traumatic brain injury: a conceptual model and implications for rehabilitation". *Brain Injury* 9.2 (1995): 109-130.

2. Ansaldo AI and Arguin M. "The recovery from aphasia depends on both the left and right hemispheres: three longitudinal case studies on the dynamics of language function after aphasia". *Brain and Language* 87.1 (2003): 177-178.

3. Ansaldo AI., *et al.* "The contribution of the right cerebral hemisphere to the recovery from aphasia: a single longitudinal case study". *Brain and Language* 82.2 (2002): 206-222.

4. Baumgaertner., *et al.* "Differential recovery of aphasia and apraxia of speech in an adolescent after infarction of the left frontal lobe: longitudinal behavioral and fMRI data". Brain and Language 95 (2005): 211-212.

5. Crosson B., *et al*. "Regional changes in word-production laterality after a naming treatment designed to produce a rightward shift in frontal activity". *Brain and Language* 111.2 (2009): 73-85.

6. Meinzer M., *et al.* "Recovery from aphasia as a function of language therapy in an early bilingual patient demonstrated by fMRI". *Neuropsychologia* 45.6 (2007): 1247-1256.

7. Meinzer M., *et al.* "Functional re-recruitment of dysfunctional brain areas predicts language recovery in chronic aphasia". *NeuroImage* 39.4 (2008): 2038-2046.

8. Perani D., *et al.* "A fMRI study of word retrieval in aphasia". *Brain and Language* 85.3 (2003): 357-368.

9. Specht K., *et al.* "Joint independent component analysis of structural and functional images reveals complex patterns of functional reorganisation in stroke aphasia". *NeuroImage* 47.4 (2009): 2057-2063.

10. Thomas C., *et al*. "Language processing in aphasia: changes in lateralization patterns during recovery reflect cerebral plasticity in adults". *Electroencephalography and Clinical Neurophysiology* 102.2 (1997): 86-97.

11. Thulborn KR., *et al.* "Plasticity of language-related brain function during recovery from stroke". *Stroke* 30.4 (1999): 749-754.

12. Levin MF., *et al*. "What do motor "recovery" and "compensation" mean in patients following stroke?" *Neurorehabilitation and Neural Repair* 23.4 (2009): 313-319.

13. Robertson IH. "Cognitive rehabilitation: attention and neclect". *Trends in Cognitive Sciences* 3.10 (1999): 385-393.

14. Wilson BA. "Towards a comprehensive model of cognitive rehabilitation". *Neuropsychological Rehabilitation* 12.2 (2002): 97-110.

15. León-Carrión J and Machuca-Murga F. "Spontaneous recovery of cognitive functions after severe brain injury: When are neurocognitive sequelae established?" *Revista Española de Neuropsicologia* 3.3 (2001): 58-67.

16. Choi JY., *et al.* "Subcortical aphasia after striatocapsular infarction: Quantitative analysis of brain perfusion SPECT using statistical parametric mapping and a statistical probabilistic anatomic map". *Journal of Nuclear Medicine* 48.2 (2007): 194-200.

17. Tracy JI., *et al.* Cognitive plasticity in neurologic disorders. *New York: Oxford University Press* (2015).

18. Mogensen J. "Recovery, compensation and reorganization in neuropathology – levels of conceptual and methodological challenges". In: J.I. Tracy, B.M. Hampstead and K. Sathian (Eds.) Cognitive plasticity in neurologic disorders. New York: Oxford University Press (2015): 3-28.

19. Mogensen J. "Cognitive recovery and rehabilitation after brain injury: mechanisms, challenges and support". In: A. Agrawal (Ed.), Brain injury – Functional aspects, rehabilitation and prevention. Rijeka, Croatia: InTech (2012): 121-150.

20. Barrett HC. and Kurzban R. "Modularity in cognition: Framing the debate". *Psychological Review* 113.3 (2006): 628-647.

21. Fodor J. "The mind doesn't work that way: The scope and limits of computational psychology". Cambridge, MA: MIT Press (2000).

22. Pinker S. "How the mind works". London: Penguin Books (1999).

23. McClelland JL., *et al*. "Parallel distributed processing": Vol. 2. Psychological and biological models. Cambridge, MA: MIT Press (1986).

24. McLeod P., *et al.* "Introduction to connectionist modelling of cognitive processes". Oxford: Oxford University Press (1998).

25. Rogers TT and McClelland JL. "Parallel distributed processing at 25: Further explorations in the microstructure of cognition". *Cognitive Science* 38 (2014): 1024-1077.

26. Rumelhart D., *et al* Parallel distributed processing. Cambridge, MA: MIT Press (1986).

27. Mogensen J. "Almost unlimited potentials of a limited neural plasticity: levels of plasticity in development and reorganization of the injured brain". *Journal of Consciousness Studies* 18.7 (2011): 13-45.

28. Mogensen J. "Reorganization in the injured brain: implications for studies of the neural substrate of cognition". *Frontiers in Psychology* 2.7 (2011).

29. Mogensen J. "Reorganization of Elementary Functions (REF) after brain injury: Implications for the therapeutic interventions and prognosis of brain injured patients suffering cognitive impairments". In: A.J. Schäfer and J. Müller (Eds.) Brain damage: Causes, management and prognosis. Hauppauge, NY: Nova Science Publishers, Inc. (2012): 1-40.

30. Mogensen J. "Reorganization of Elementary Functions (REF) after brain injury and in the intact brain: A novel understanding of neurocognitive organization and reorganization". In: J. Costa and E. Villalba (Eds.) Horizons in neuroscience research. Vol. 15. New York: Nova Science Publishers, Inc. (2014): 99-140.

31. Mogensen J. and Malá H. "Post-traumatic functional recovery and reorganization in animal models. A theoretical and methodological challenge". *Scandinavian Journal of Psychology* 50.6 (2009): 561-573.

32. Newport R and Schenk T. "Prisms and neglect: what have we learned?" *Neuropsychologia* 50.6 (2012): 1080-1091.

33. Wilms I and Malá H. "Indirect versus direct feedback in computer-based Prism Adaptation Therapy". Neuropsychological Rehabilitation 20.6 (2010): 830-853.

34. Wilms I and Mogensen J. "Dissimilar outcomes of apparently similar procedures as a challenge to clinical neurorehabilitation and basic research – when the same is not the same". *NeuroRehabilitation* 29.3 (2011): 221-227.

Volume 3 Issue 4 September 2016