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Identifying patterns of cognitive deficits: the path to better outcomes after stroke

Stephanie Clarke

Cognitive impairment after stroke: frequency, patterns, and relationship to functional abilities
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Today cognitive models are bound to impact on neurorehabilitation and define patient profiles, which respond to specific therapeutic interventions

The seminal paper by Tatemichi and colleagues[1] sets a cornerstone for the diagnosis of poststroke cognitive impairment. The authors documented the diversity of stroke-related cognitive deficits that were present 3 months after an ischaemic stroke. They used an extended neuropsychological battery, covering memory, orientation, language, visuospatial skills, abstract reasoning and attention and compared the performance of 227 patients with that of 240 control subjects. Cognitive impairment, defined as deficient performance in more than three domains, was found in 35.2% of stroke patients (vs in 3.8% of control subject) and involved mostly memory, orientation, language and attention. These deficits impacted on the ability to live independently.

Establishing the patterns of cognitive impairment after stroke was grounding for three lines of research. First, a series of studies documented the contribution of poststroke cognitive impairment to dementia.[2] Second, the introduction of endovascular treatment improved the functional outcome of stroke patients, including the severity of cognitive syndromes.[3] Third, innovative approaches in cognitive rehabilitation were shown to improve recovery during the postacute stage of stroke.[4] The challenge which we face today is how to prevent the decline in dementia and how to create a continuum of care in which thrombolysis and/or thrombectomy during the acute stage is combined with appropriate cognitive rehabilitation during the later stages.

POSTSTROKE DEMENTIA VERSUS NEUROPSYCHOLOGICAL SYNDROMES

A meta-analysis of patient cohorts that were investigated from the late 1980 to early 2000 highlighted the causal role of stroke in dementia. The authors estimated that 10% of patients develop new dementia after a first stroke and >30% after a recurring stroke.[5] The incidence of new-onset poststroke dementia continues to rise during the following years.[6] Dementia, which is diagnosed in the aftermath of stroke, is mostly associated with small-vessel occlusions and lacunar infarctions rather than large focal lesions.[7]

Large focal lesions tend to be associated with specific neuropsychological syndromes; the site of lesion tends to be typical of a syndrome, such as the infarction in the territory of the left middle cerebral artery in aphasia or of the right middle cerebral artery in unilateral neglect 4. Numerous studies investigated the neural basis of specific neuropsychological syndromes by testing subpopulations of patients who had a stroke with extended batteries of syndrome-specific tests. Concurring evidence from patient studies and from activation studies in normal subjects shows that a given neuropsychological syndrome comprises a range of functional features. These specific features can be impaired selectively, following lesions at critical locations, as demonstrated, for example, for different aspects of auditory verbal comprehension[8] or for different types of auditory neglect.[9]

THROMBOLYSIS AND THROMBECTOMY: DECREASING THE SEVERITY OF NEUROPSYCHOLOGICAL SYNDROMES

Thrombolysis and/or thrombectomy are part of the integral management of patients who had a acute ischaemic stroke.[10 11] Whereas early studies failed to show a positive effect of thrombolysis on the cognitive outcome, there is some evidence in later, more focused investigations, where intravenous thrombolysis was reported to be associated with less severe neuropsychological syndromes in patients who had a a first ischaemic stroke.[3] In particular, this has been demonstrated for aphasia during the acute and postacute (3 months) stages of left middle cerebral artery infarction[12] and for the severity of visuoperceptual dysfunction during the acute stage of right hemispheric infarction.[13]

IMPACT OF COGNITIVE MODELS ON STROKE REHABILITATION

Rehabilitation of cognitive functions has developed in parallel to motor rehabilitation. It comprises a wealth of specific approaches, many of which have used with some success in subpopulations of patients who had a stroke.[4] Despite these encouraging reports, systematic reviews of randomised controlled trials tend to take, with few exceptions, a negative view. The positive effect has been attested for speech and language therapy. The authors of a seminal Cochrane Collaboration review conclude that speech and language therapy, as compared with no therapy, leads to the improvement of functional communication, reading, writing and expressive language. Furthermore, the therapy administered at high intensity, high dose or over a longer period may confer additional benefits.[14] Other cognitive domains fare less well in systematic

Service de neuropsychologie et de neuroréhabilitation, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland

Correspondence to Professor Stephanie Clarke, Service de neuropsychologie et de neuroréhabilitation, Centre Hospitalier Universitaire Vaudois, Lausanne 1011, Switzerland; stephanie.clarke@chuv.ch

reviews. A Cochrane Collaboration reviewed evidence from randomised controlled trials for the effectiveness of cognitive rehabilitation in spatial neglect. The authors found that neglect rehabilitation interventions have a positive immediate effect on standardised neglect tests but do not influence the significantly long-term outcome as assessed in activities of daily living or standardised neglect tests. Furthermore, the authors deplored methodological weaknesses in over half of the studies, such as inadequate allocation concealment. They concluded that 'the effectiveness of cognitive rehabilitation intervention for reducing the disabling effects of neglect and increasing independence remains unproven'.[15]

Apart from methodological drawbacks, such as inadequate concealment, which were pointed out in the Cochrane Collaboration review on neglect,[15] cognitive rehabilitation suffers from conceptual problems, for which unilateral neglect offers a good illustration.[16] Unilateral neglect is clinically very heterogeneous, affecting to a varying degree, visual, auditory, somatosensory and motor modalities. Even within the most often affected, visuospatial modality there are surprising dissociations between symptoms. Conversely, a whole range of therapeutic interventions is used in neglect rehabilitation, from top–down approaches, which aim at increasing the attentional load to the left side by behavioural training or TMS (transcranial magnetic stimulation) inactivation of the contralesional hemisphere, to bottom-up approaches which modulate sensorimotor integration.[17] In clinical trials, the same therapeutic intervention is administered typically to all patients, independently of their neglect profiles. Evidence from recent studies indicates that a specific therapeutic intervention tends to be successful in a subgroup of neglect patients and that responders versus non-responders have different profiles, often with respect to the site of lesion. Neglect rehabilitation by means of prismatic adaptation is probably the most thoroughly investigated example of this. Unilateral neglect results from damage to the right-dominant ventral attentional system.[18] Brief exposure to prismatic adaptation alleviates neglect symptoms[19] by shifting the dominance of the ventral attentional system from the right to the left hemisphere.[20] This model of neglect recovery predicts that only patients with preserved dorsal attentional system and preserved posterior callosal pathway will benefit from the shift in hemispheric dominance and hence respond to the treatment. This prediction has been validated in three independent studies.[21–23]

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