JOHANN SCHUTTE
CLINICAL PSYCHOLOGIST
www.js.net.nz
johann@js.net.nz

Age Related Cognitive Decline

In comparison to young, healthy adults, older people (above the age of 50) generally show a decline in memory and other cognitive abilities as a consequence of the ageing process, rather than as a result of a specific neurological disorder. Like every other organ of the body, the ageing brain loses some of its efficiency. The advent of decline is often signalled by complaints from individuals (or their family members) that they have difficulties remembering names and words.

Ageing impacts the brain from the cellular and sub-cellular level (the cell and its contents) to the structural level, i.e. larger neurological elements such as nerve bundles. Additionally, the physical structure of the brain as a whole deteriorates with age. This is related to on-going neuronal atrophy, reductions in the number of synaptic spines and functional synapses which contribute to increasing reductions in cortical thickness and sub-cortical volume in some regions of the brain. For example, the ageing process may account for 37% of volume variances in the thalamus, which is involved in sight, hearing, and the sleep-wake cycle. In summary, age related neuroanatomical changes account for an estimated 25% to 100% of the variance in cognitive ability between young and aged individuals.

Various diagnostic categorizations and terms have been developed over the years to describe age correlated decline in cognitive function. The National Institute of Mental Health (NIMH) introduced criteria for 'Age Associated Memory Impairment' (AAMI), whilst the American Psychiatric Association offers 'Age Related Cognitive Decline' (ARCD) and 'Mild Neurocognitive Disorder' (MNCD) in the current Diagnostic and Statistical Manual of Mental Disorders (4th edition), and the International Classification of Diseases includes Mild Cognitive Disorder (MCD) in their categorization. Various other diagnostic definitions have also been proposed by researchers and clinicians, with each of these endeavours aimed at improving clarity and provision of robust criteria.

Importantly, and irrespective of the definitional tensions that exist, it is clear that there is wide recognition that not all memory problems or forms of cognitive decline are due to serious neurodegenerative conditions, such as Alzheimer's disease. However, it is also recognized that cognitive decline could be the first signal of incipient dementia.

Cognitive decline is associated with several neurological markers. These include the loss of myelin integrity, cortical thinning, impaired serotonin, acetylcholine, and dopamine receptor binding and signalling, an accumulation of neurofibrillary tangles, and altered concentrations of various brain metabolites. Additionally, clear associations exist between the rate and severity of decline and a variety of factors. These include chronic low-level inflammation, declining hormone levels, excess body weight, suboptimal nutrition, cognitive inactivity and the absence of a gratifying social network. Many of these factors can be modified and therapeutically operationalized towards decreasing the rate of decline.

On examination of suspected decline, neuropsychological testing typically renders a cognitive performance profile within the normal range for the examinee's age, or one that is slightly lowered in terms of the age-group average. This is usually complemented by self- and/or informant -reports of a mild, yet noticeable decrease in functionality with regard to the domestic and occupational environments. Onset is gradual, and progression typically slow.

Last review of this document, March 2012

An accurate and comprehensive neuropsychological assessment is always desirable. The symptoms of decline may cause the unwarranted fear or anxiety that they may be early signs of Alzheimer's disease or another serious impairment. An evaluation addresses these fears, and offers a basis for differential diagnosis and management planning, where necessary. It may be necessary to repeat assessments over time (usually once every year or two) for monitoring purposes.

Neuropsychological assessment should supplement psychiatric, neurological and physical examinations (which include laboratory tests and neuroimaging), with a view to informing diagnosis, particularly as it pertains to differentiating between cognitive decline as:

- a direct and normal consequence of ageing,
- a primary consequence of an injury, disease or infection of the brain,
- a secondary consequence of another medical condition and/or its pharmacological treatment (e.g. sedatives, anticholinergic medications) and as
- the earliest manifestation of incipient neurodegenerative disease.

Neuropsychological assessment of age related memory impairment thus seeks to (a) quantify and characterize memory impairment, and (b) establish whether other cognitive systems are involved. Poor performance on facets of memory (e.g. immediate recall and consolidation) may be secondary to attentional problems such as distractibility and failure of concentration. Similarly, language disorders and frontotemporal (executive function) disorders may impair performance on memory tests.

At the very least, demonstration of a stable attentive capacity is essential before conclusions about memory function can be reached. The neuropsychological assessment process thus not only quantifies, but also assists in the localization and description of the mechanism of memory dysfunction.

If attentional integrity is neuropsychometrically demonstrated to be intact, the next step is to determine whether there is primary memory failure. Primary memory failure is usually indicated when short-term recall (measured by digit-span) is good, but consolidation is bad, i.e. there is a rapid rate of forgetting. With impaired consolidation there are usually high levels of susceptibility to interference effects and progressive worsening in recall. Novel associations (word pairs) are typically difficult to consolidate. Primary memory failure involves the memory circuits and more specifically, the hippocampi and thalami, and their connections. Impairment in attention by contrast, usually involves a greater spread of both cortical and subcortical areas. These areas include cortical areas of the frontal (where encoding of new material occurs), parietal and temporal lobes and subcortical structures such as the basal ganglia, caudate and white matter.

In the interpretation of neuropsychometric results that suggest mild cognitive decline in an elderly person, emotional and motivational influences, and sensory or motor impairment as well as several other potentially modulating influences need to be carefully considered, as the slightly lowered performance on assessment instruments that typically reflect ARCD/AAMI may simply be the result of fatigue, depression, trauma, grief, prescription and non-prescription drugs, alcohol use, performance anxiety or deterioration in visual or auditory perception.

It should be evident from the preceding argument that neuropsychological assessment of any specific aspect, must occur in the context of a more comprehensive examination. This would include the utilisation of EEG and Neuroimaging technologies such as MRI and CT scans, and from a neuropsychometric point of view, a comprehensive battery of tests that facilitates differential diagnosis.

Although there are no treatments of proven value currently available for age related cognitive decline, memory management strategies may be used to help an individual overcome some of the symptoms. Life-style changes may mitigate the effects of ageing. Additionally, some consideration with regard to support-needs may be necessary.

Last review of this document, March 2012