Can Alzheimer's disease steal one's consciousness?

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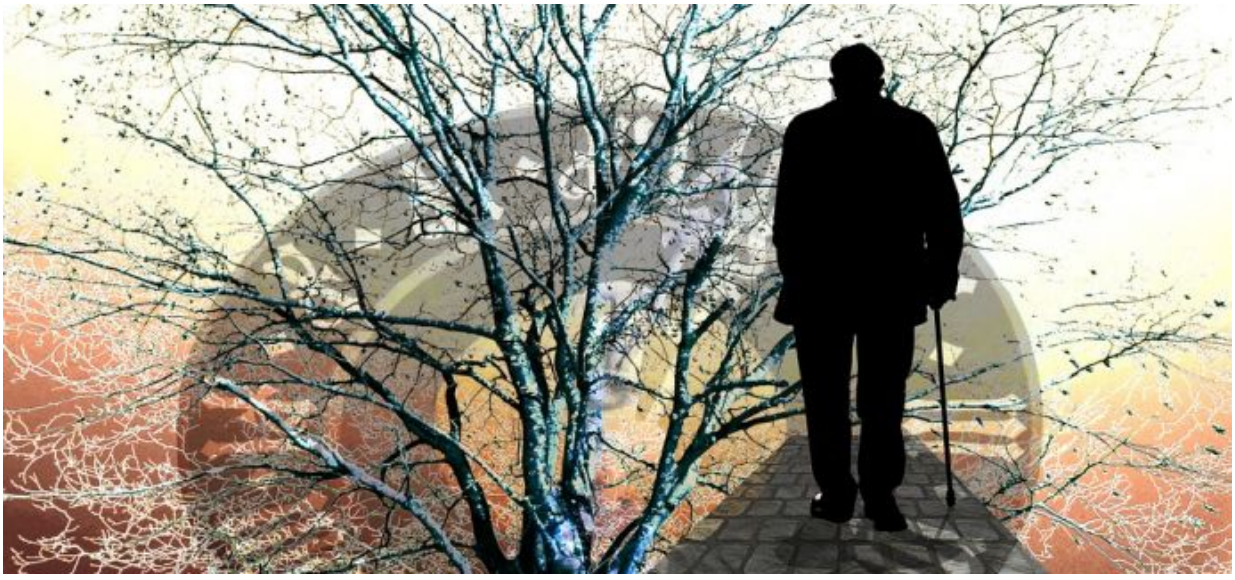

Alzheimer's disease (AD) has been characterized as a "complete loss of self." Early on when memory begins to fade, the victim has difficulty recalling names, their grocery list or where they put their keys. As the disease progresses, they have trouble staying focused, planning and performing basic daily activities. From the exterior, dementia appears to ravage one's intellect and personality; yet as mere observers, it's impossible to ascertain how consciousness of the self and environment is transformed by dementia. The celebrated late neurologist Oliver Sacks

once suggested that, "Style, neurologically, is the deepest part of one's being and may be preserved, almost to the last, in dementia." Is this remaining neurological "style" sufficient to preserve consciousness? Is the AD patient aware of their deteriorating cognition, retaining a sense of identity or morality, or can they still connect with friends and loved ones? Emerging advances in neuroscience have enabled researchers to more precisely probe the AD brain, suggesting that although some aspects of consciousness are compromised by dementia, others are remarkably spared.

Manifestations of consciousness

Scientists are beginning to piece together how the selective loss of some functions, but the preservation of others, alters consciousness in AD. A recent study found that the severity of cognitive impairment strongly relates to "meta-cognition" (reflecting on one's own condition), moral judgments and thinking about the future, whereas basic personal identity and body awareness remain. Perhaps the most widely observed deficit in consciousness is "anosognosia," impaired awareness of one's own illness; whereas individuals with mild cognitive impairment (MCI; considered a precursor to full AD) are aware of their declining memory, AD patients may be unaware of their impairments. These behavioral signs suggest that only some aspects of consciousness and self-awareness are truly lost in AD.

Although basic perceptual, sensory, and communication skills are relatively spared, the defining feature of AD is memory loss. The unique nature of these memory deficits provides clues to why sub-components of consciousness deteriorate in dementia. Memory for experiences (episodic memory; e.g., I went for a walk in the park yesterday) and recollection of their associated details are impaired, while memory for facts and meaning (semantic memory; e.g., Paris is the capital of France) is preserved. Recollection of episodic memories demands that the

rememberer engage with the memory, searching for context and reflecting upon the memory after it's recalled. In contrast, semantic memory can be automatic and doesn't require this level of attention or personal involvement. Impairment in personal meta-cognition may lie at the heart of these memory deficits. For instance, AD patients can accurately predict how well they'll later recall semantic information, but cannot predict how they'll recall episodic memory, pointing to a specific impairment in awareness of their personal memories. Perhaps then, loss of consciousness in AD stems from impaired control over attention to memory. Indeed, AD has been considered a disorder of "impaired attention to life." This is supported by reports that the feelings surrounding a memory and re-experiencing an episode during recollection are absent in AD. Unable to engage with a memory, the individual loses their conscious awareness of the experience.

Steven Pinker defines consciousness as the sum of sentience, access to information, and self-knowledge. While the first two are relatively intact in AD, episodic memories are heavily dependent on the last. In addition to problems with episodic memory, some AD patients have difficulty remembering semantic information when it's personal, suggesting that hindered access to personal details of a memory may, to some extent, account for impairments in consciousness. As dementia often warps one's sense of time continuity, this temporal disconnect could further degrade one's ability to accurately place themself in the context of past and future.

Neural underpinnings of altered consciousness

The unique pattern of brain pathology in AD might explain the resulting partial loss of consciousness in its victims. Since consciousness requires the coordination of distributed brain networks that support sensation, memory, attention and emotion, dysfunction of this system could lead to changes in cognition and awareness. In AD, brain atrophy begins in the

medial temporal lobe, followed by lateral temporal, parietal and frontal areas. These regions may engender consciousness by supporting recall of the source of a memory, self-referential processing, and self-identification. Metabolism is reduced in temporal and parietal regions, and these changes are associated with the severity of cognitive impairment and changes in awareness. Furthermore, in line with the finding that AD patients have trouble understanding another's personality, stimulating the temporoparietal junction can induce an "out-of-body experience," where the person perceives themself as someone else.

Deficits in meta-cognitive ability could further stem from disconnect among key brain hubs. The pathways connecting the frontal, parietal and temporal cortices, which integrate information across the brain, become compromised in AD. Although these widespread brain networks aren't affected until late in the disease, the earliest target of AD-related neurodegeneration, the medial temporal lobe, lies at the core of this system. In the medial temporal lobe, the hippocampus binds contextual information about an experience into memory, but must communicate across the brain to store these details into long-term memory. Therefore, early damage to the medial temporal lobe might be a first step towards a disrupted sense of awareness.

Preserving consciousness

This research linking the select deficits in self-awareness and meta-cognition with the characteristic pattern of neurodegeneration in AD is invaluable for informing about the neurobiological origins of consciousness. Perhaps more importantly, understanding which aspects of consciousness go awry in AD could theoretically promote the development of more effective treatment strategies. If attention to one's inner mental landscape, and awareness of one's personal state, are targets of AD, there's hope that training these skills could confer resilience

against the neuropathology degrading these functions. Although in its infancy, studies testing this possibility have shown promise. Older adults who are experienced mediators were more resilient to the detrimental effects of age on cognitive function than non-meditators, and an intervention of eight weeks of meditation training increased brain blood flow and improved cognitive function in individuals with MCI and AD.

Although it's tempting to attribute changes in awareness to strictly biological aberrations, we cannot overlook the physical and social interactions that mold our mental state. Being labeled "demented", infantilized or treated as incapable, as can be done to AD victims, would be damaging to anyone's self-esteem and sense of dignity. Perhaps then, by nurturing the AD mind by reinforcing their human worth, we could dampen the pathological fire working to degrade their personhood.

More information: O. Blanke. Linking Out-of-Body Experience and Self Processing to Mental Own-Body Imagery at the Temporoparietal Junction, *Journal of Neuroscience* (2005). DOI: 10.1523/JNEUROSCI.2612-04.2005

Nora Breen et al. Mirrored-self Misidentification: Two Cases of Focal Onset Dementia, *Neurocase* (2001). DOI: 10.1093/neucas/7.3.239

Arnaud D'Argembeau et al. Self-referential reflective activity and its relationship with rest: a PET study, *NeuroImage* (2005). DOI: 10.1016/j.neuroimage.2004.11.048

Juergen Dukart et al. Generative FDG-PET and MRI Model of Aging and Disease Progression in Alzheimer's Disease, *PLoS Computational Biology* (2013). DOI: 10.1371/journal.pcbi.1002987

E. Kalbe et al. Anosognosia in Very Mild Alzheimer's Disease but Not in Mild Cognitive Impairment, *Dementia and Geriatric Cognitive Disorders*

(2005). DOI: 10.1159/000084704

Beata Lipinska et al. Feeling-of-knowing in fact retrieval: Further evidence for preservation in early Alzheimer's disease, *Journal of the International Neuropsychological Society* (2009). DOI: 10.1017/S1355617700001375

Giuseppe Pagnoni et al. Age effects on gray matter volume and attentional performance in Zen meditation, *Neurobiology of Aging* (2007). DOI: 10.1016/j.neurobiolaging.2007.06.008

CHARLES F. REYNOLDS. The Loss of Self: A Family Resource for the Care of Alzheimer's Disease and Related Disorders, *American Journal of Psychiatry* (1988). DOI: 10.1176/ajp.145.6.760

E. Salmon et al. Cerebral metabolic correlates of four dementia scales in Alzheimer's disease, *Journal of Neurology* (2005). DOI: 10.1007/s00415-005-0551-3