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Perspectives on Recovery from Coma and Low Arousal

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Introduction

Early after acquired brain injury many people are in a semicomatose or low arousal state and some individuals are even less responsive and either in a vegetative or minimally conscious state. The individual with low arousal has diminished ability to respond and perform cognitive and physical tasks, to fully engage in rehabilitation and maximize their recovery. When individuals are in these states, their residual functioning cannot be accurately assessed. Therapists often have to use frequent auditory and sometimes tactile stimulation to keep someone aroused enough to participate in therapy tasks. Research suggests that dopamine dysfunction may be associated with these states. During the first hours after TBI, catecholamine levels in the cerebral spinal fluid are increased, but production consistently decreases thereafter. It has been hypothesized that dopamine dysfunction may be causally related to these altered states of arousal and responsiveness. Numerous pharmacological interventions have some empirical support to treat low arousal states. Many dopaminergic agonist medications have been trialed in off label use to enhance arousal. These include: L-dopa [1], Bromocriptine [2], Amantadine [3,4] and Pramipexol [4]. One of the more popular dopaminergic agonists is amantadine which enhances presynaptic dopamine release and inhibits dopamine reuptake, resulting in an increased amount of dopamine in the synaptic cleft. Amantadine may also increase the density of postsynaptic dopamine receptors and alter the conformation of these receptors. Some research suggests Amantadine at 200-400 mg daily safely and effectively improves arousal and cognition after brain injury [5]. For patients in minimally conscious states, zolpidem has been shown to have paradoxically stimulating effects and efficacy. It has been speculated that in patients with impaired consciousness, zolpidem reduces the activity of an area of the brain that would otherwise inhibit activity in other regions of the brain. However, greater specificity and understanding of the mechanisms of consciousness in these patients, is needed [6].

Findings such as that exemplify how complex these issues are. Some key issues for clinicians working with low arousal patients are the objective measurement and tracking of arousal and responsiveness level. In working with the hypoaroused patient this is a complex undertaking especially given the high level of daily variability and the unclear recovery trajectory. Some measures such as the Coma Recovery Scale Revised (CRS-R) are often necessary to distinguish between a vegetative state and the more purposeful minimally conscious state. The Coma/Near Coma Scale can also be helpful, but at different stages in the arousal continuum there does not appear to be a real gold standard measure. Clinicians monitoring arousal, duration of wakefulness, task persistence and other indices of alertness need to be consistent and have frequent communication to help track incremental improvements as related to the current medication regime. This process is confounded by frequently disrupted sleep-wake cycles and also the natural recovery process and often many other medical factors. Far more empirical research is needed to establish decisive decision trees and prescribing and treatment protocols. Hopefully readers of this journal will submit case studies and more systematic research to promote a further understanding of these clinical issues.

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