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Pharmaceuticals for Poststroke and Brain Injury Rehabilitation

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In the June and July issues, we introduced the first half of a four-issue special series of the *American Journal of Physical Medicine & Rehabilitation*, focused on innovative, physiologic treatments for stroke and traumatic brain injury.^{1,2} These disorders are leading causes of adult disability in the United States today, accounting for tremendous personal, social, and financial costs for survivors, caregivers, and society.³ In the June issue, Hillis⁴ has provided an up-to-date review on how physiologic treatments may optimize poststroke aphasia recovery. Choi et al.⁵ and Buxbaum et al.⁶ have presented data in the July issue on treatment of poststroke spatial neglect.

In this issue, we consider pharmaceutical interventions. Minimal profit incentive to study patent-expired medications for cognition exists—thus, older, familiar medications with proven safety are not usually eligible for industry support, which is available to research newer, relatively unproven agents. The burden of developing a pharmaceutical treatment science for older medications, balancing patient safety with innovation, may have fallen on the resources of individual researchers and public and private grant funding. Clinicians wishing to provide best care for patients despite these limits have established the current practice standard for brain injury rehabilitation, including off-label use of stimulants, dopaminergic agents, cholinesterase inhibitors, and other agents with possible neurotropic or neuroprotective effects. Last year, Hokenson et al.⁷ reported that more than 30% of acute rehabilitation patients with poststroke spatial neglect received off-label dopaminergic, stimulant, and alerting medications.

In this issue, we present two papers on pharmaceuticals in neurorehabilitation. Barrett and Eslinger⁸ present a preliminary report suggesting that amantadine improved speech fluency in an inhomogenous group of brain-injured subjects, some of whom had linguistic problems, and some of whom may have had attentional or cognitive deficits. Barrett and Eslinger propose studying amantadine for abnormal speech output in acquired aphasia associated with perisylvian cortical injury. Martin et al.⁹ performed a meta-analysis of a series of “*n* = 1” trials of methylphenidate in minimally conscious patients and those in vegetative states following TBI. Surprisingly, they report no definite evidence of a methylphenidate treatment effect.

Group studies with parallel designs may better address whether persistent improvement is associated with methylphenidate treatment of TBI, because single-subject designs with on–off periods are less sensitive to detect these changes.

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