inhibition of presynaptic cholinergic signaling by stimulation of presynaptic muscarinic type 2 receptors and nonphysiologic, tonic stimulation of postsynaptic nicotinic and muscarinic receptors.[6] Despite these limitations, mobility benefits of AChEIs have been reported in PD.[7]

Preliminary data in animal studies suggest that drugs that selectively stimulate α4β2(*) nicotinic receptors not only have excellent entry into the brain but also may improve mobility functions in rats with dual dopaminergic and cholinergic lesions.[8] Such selective agonists are superior to the nonselective nicotine mother drug because nicotine interferes with the phasic cholinergic activity that is essential for cognitive function.[9] Further investigation of this class of drugs to treat mobility problems in PD is necessary.

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CORRECTION

Tumefactive MS lesions under fingolimod: A case report and literature review

In the article "Tumefactive MS lesions under fingolimod: A case report and literature review" by G. Pilz et al. (*Neurology*® 2013;81:1654–1658), there is an error in the description of figure B.c. The description should have read, "Follow-up MRI 6 months later: a new large T2 hyperintense lesion with perifocal edema is seen in the right frontal white matter which shows ring-like gadolinium enhancement (lower panel)." The authors regret the error.