Advances in the Treatment of Psychiatric Disrorders

Future Treatments for Non-Alzheimer Dementia

Most illnesses that involve dementia involve a primary process that damages neurons and secondary processes that extend the damage, such as release of excitatory amino acids (eg, glutamate). Excitatory amino acids act on postsynaptic receptors (eg, the NMDA receptor), causing them to open calcium channels that let calcium ions into postsynaptic neurons. In moderate amounts, this will promote long-term potentiation, a fundamental aspect of learning; however, in excessive amounts (which happens when the brain is damaged), high levels of calcium ions begin a cascade of events that result in oxidative stress and apoptosis.

"Current treatments aimed at dementing illnesses and other neurological conditions do not reverse the primary cause of the dementia," said Steven L. Dubovsky, MD, State University of New York, Buffalo, NY. "They are treating downstream effects, including progressive and more global neuron death, impairment of memory, behavioral disinhibition, and psychosis." One of these treatments is memantine, a version of the antiviral drug amantadine, which is used in the treatment of Parkinson disease. Memantine reduces excitotoxicity by blocking the NMDA receptor [Chen H-S V et al. *J Neurosci* 1992].

Experimental research for new treatments for dementia is underway using a family of proteins called neurotrophic factors, which are responsible for the growth and survival of neurons during development and for maintaining adult neurons. In test tubes and in animal models, they also have been shown to promote survival of damaged neurons. These large proteins, however, cannot cross the blood-brain barrier, so the focus of the research is to induce the genes for neurotrophic factors to make amino acids that form them. While there has been increasing work in this area, it remains very experimental.

A Framework for Developing Individualized Treatment Plans for Autistic Spectrum Disorders

As with many other psychiatric disorders, we now realize that autism is really a spectrum of disorders. Bryna Siegel, PhD, University of California, San Francisco, CA, provided insight into how clinicians might go about developing a functional model for treating patients with autistic spectrum disorder (ASD).

"Rather than spending a lot of time on the diagnosis," Dr. Siegel told the audience, "begin by cataloging the individual's deficits and strengths." Each individual with ASD is different, and an effective treatment plan will take into account how the individual perceives, processes, stores, and retrieves information. The plan should be based on the interrelationship between the individual's specific 'autistic learning disabilities' (eg, his social, verbal and non-verbal communication, and play and exploration deficits) and the 'autistic learning styles' that have automatically developed to compensate for these impairments. Dr. Siegel provided several examples. For patients who have a strong need for routine, rather than thinking of the routines as bad, make them functional and use them as a way to make the environment more predictable; for individuals who have verbal communication deficits, encourage affiliative contact by focusing conversations toward topics that are of interest to the patient.

In concluding, Dr. Siegel told the audience that each individual with ASD will be different and that it is necessary to go beyond diagnostic checklists. "Individualized interventions for the best outcomes are those that exploit relative strengths to compensate for relative weaknesses. Selection of tailored treatments should be based on this profile."