Introduction

Ischemic Brain Infarction

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Stroke is the third most common cause of mortality and the leading cause of disability in the United States. There are about 700,000 new cases each year with ischemic brain infarction accounting for almost 85% of all strokes. In this issue of CNS Spectrums, new and potential treatments for acute ischemic stroke and the cognitive changes that often result are discussed.

There are approximately 3 million survivors of stroke in the United States, most of whom are disabled. In addition to motor, sensory, and speech deficits, there is significant morbidity due to emotional and cognitive dysfunction, including behavioral changes and depression. These behavioral changes that follow stroke contribute significantly to the overall disability. Dr. Erhlan and colleagues review the emotional changes that occur following right sided cerebral infarction, while Drs. Starkstein and Manes provide an in-depth analysis of depression and apathy following stroke. Both articles discuss various treatment options for these behavioral changes, including medications and psychotherapy.

Ischemic stroke results following an occlusion of a cerebral vessel usually caused by in situ thrombosis, embolism from the heart, or a proximal vessel (such as the cervical carotid artery). A fall in cerebral blood flow occurs in the territory supplied by the blocked cerebral artery, resulting in a densely ischemic region of tissue surrounded by a marginally perfused area frequently referred to as the ischemic penumbra. While the most ischemic tissue may become irreversibly damaged, subsequent damage continues to occur in the surrounding brain tissue in the region of the ischemic penumbra for some time after the initial insult. If reperfusion or exposure to neuroprotective agents can be modified to reduce the incidence of stroke.

Neuroprotective agents would protect brain tissue in the ischemic zone from permanent ischemic damage. Various neuroprotective drugs that act at different points in the cytotoxic cascade triggered by cerebral ischemia are being tested. Similar to thrombolytic agents, the neuroprotective drugs are more likely to be effective when given early after stroke onset, usually within 6 hours. A combination therapy of neuroprotective drugs and thrombolytics may be the most effective way to prevent extensive ischemic brain injury.

As discussed in the paper by Dr. Felberg and colleagues, many studies of the various neuroprotective agents have been disappointing to date, but each study contributes to our understanding of the cytotoxic effects that take place following brain ischemia. This will undoubtedly lead to the discovery of effective neuroprotective agents in the near future. Dr. Felberg and colleagues discuss thrombolysis and the ischemic cascade leading to acute and delayed neuronal cell death, the steps in the process that might be affected by the various agents, and the rationale for neuroprotection.

Once stroke has occurred, many patients receive rehabilitation in the form of physical therapy. Physical rehabilitation may be less effective if the patient has depression, apathy, or cognitive deficits. The use of neurostimulants has recently been found to help some patients recover faster during the rehabilitation process. Dr. Flanagan provides an in-depth review of these agents and discusses the various neurochemical pathways that can be manipulated by these drugs to improve overall outcome.

Since prevention of stroke is the overall primary goal, Dr. Tuhrim reviews established as well as recently discovered risk factors for stroke and discusses how these risk factors can be modified to reduce the incidence of stroke.

I would like to thank CNS Spectrums and the contributing authors to this volume on stroke. The future is promising as there continues to be advances in stroke prevention, diagnosis, and treatment. CNS

REFERENCES