NEUROPROTECTION IN ACUTE ISCHEMIC STROKE

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Focal Ischemic injury in the brain is related to both the intensity and the duration of the decrement in cerebral flow. The ischemic penumbra, an area characterized by levels of blood flow slightly greater than the ischemic core itself, is a zone exhibiting preserved or even accentuated metabolic rate, apparently driven by recurrent ischemic depolarisations. Excitatory amino acid neurotransmitter release and raised levels of oxygen radical activity occur within it. The penumbra represents that region of the focal ischemic lesion, which is potentially amenable to metabolic neuroprotection, and several classes of neuroprotective agents are currently under clinical evaluation for stroke. As the untreated penumbra deteriorates over time, animal studies indicate that therapy should be administered within a therapeutic window of no more than 3-6 h from stroke onset if it is to be successful. Neuroprotective drugs interfere with various stages of the ischemic cascade leading to irreversible tissue damage, and are not thought to affect bleeding. Since the early 90s many neuroprotective agents were investigated in numerous clinical trials on patients with acute ischemic stroke. However, the results were very disappointing and until now all of them failed to show beneficial effect on stroke outcome. There are several potential explanations for the negative results of the human clinical trials compared to the excellent results in animal models, which will be further discussed. The saga of neuroprotection continues and currently several studies are being conducted and we hope to finally find a “magic drug” which may salvage the ischemic neurons and improve patient outcome.