Upon impact, glutamate and other excitatory neurotranmitters attach to N-methyl-D-aspartate (NMDA) receptors leading to a rapid ion shift across the cell membrane. Rapid loss of intercellular potas- sium and influx of calcium forces up-regulation of the sodium-potassium pumps in an attempt to restore nor- mal resting membrane potential. As sodium-potassium pumps deplete cerebral stores of adenosine triphos- phate (ATP) compensatory hyperglycolysis occurs