voltage-dependent blockade by magnesium and modulation by glycine and polyamines. For example, in the low magnesium model, spontaneous synchronous burst discharge in hippocampal pyramidal cell populations is sensitive to NMDA antagonists. That finding suggests that it is the opening of NMDA channels, by relieving the magnesium blockade, that facilitates epileptiform activity.

Significant attention in the literature is also being given to gamma-amino butyric acid (GABA) receptors for the potential role in control of excitability. Changes in GABA inhibitory efficacy can lead to important effects on the excitability of the system. GABAergic inhibitory post-synaptic potentials (IPSPs) have been shown to be quite labile in response to repetitive activation of cortical cell populations, as may occur during epileptiform discharge. Scientists have shown that even a small percentage change in GABA inhibition can have profound effects on neocortical epileptogenesis. These changes in GABAergic inhibition may be the key to an explanation of how repetitive discharge patterns give rise to ictal discharge. Further, there appears to be a significant increase in excitatory postsynaptic potential (EPSP) frequency prior to seizure initiation an observation that is consistent with loss of IPSP efficacy prior to ictal onset.

The above hypotheses describe different mechanisms of epileptogenesis, but it is quite possible that all of these mechanisms take place, and they reflect large variety of types of epileptic seizures. The common principle of the mechanisms proposed is the change of membrane properties (i.e., conductance, permeability etc.) of certain neurons which results in depolarization and burst discharging. Some factors (e.g., trauma) can affect these specific neurons and initiate synchrony for neurons that control internal communication and communication with various muscle systems not associated with vital functions (i.e., heart beating, breathing). High strength pulsed electric fields could also be such a factor.

Mechanism to Reproduce the Desired Effects

Application of electromagnetic pulses is also a conceptual nonlethal technology that uses electromagnetic energy to induce neural synchrony and disruption of voluntary muscle control. The effectiveness of this concept has not been demonstrated. However, from past work in evaluating the potential for electromagnetic pulse generators to affect humans, it is estimated that sufficiently strong internal fields can be generated within the brain to trigger neurons. Estimates are that 50 to 100 kV/m free field of very sharp pulses (~ 1 nS) are required to produce a cell membranic potential of approximately 2 V; this would probably be sufficient to trigger neurons or make them more susceptible to firing.

The electromagnetic pulse concept is one in which a very fast (nanosecond timeframe) high voltage (approximately 100 kV/m or greater) electromagnetic pulse is repeated at the alpha brain wave frequency (about 15 Hz). It is known that a similar frequency of pulsing light can trigger sensitive individuals (those with some degree of light-sensitivity epilepsy) into a seizure and it is thought that by using a method that could actually trigger nerve synapses directly with an electrical field, essentially 100% of individuals would be susceptible to seizure induction. The photic-induced seizure phenomenon was borne out