ELECTROCONVULSIVE THERAPY

This article was written as a consequence of reading the book "Electroshock" written by Max Fink M.D.

As you may know, electroconvulsive therapy (ECT) grew from the observation that schizophrenic persons "Who developed epileptic seizures after a head injury or after encephalitis were occasionally cured of their mental disorder". In an effort to induce a controlled epileptic fit, intramuscular injections of camphor in oil or intravenous injections of Metrazol (Pentylenetetrazol) were used. Because of the sometime traumatic effects of these chemical convulsives, an alternative method of inducing seizures was sought and electric shock was eventually tried; first on animals (Not stated what animal(s) but I imagine monkeys) and then humans.

Without the muscle seizures, the treatment fails and that is what led me to hypothesize that the electric signals from the muscle stretch receptors were the key to understanding how ECT works. Consider a patient with severe depression who does not respond to simple questions i.e. what is your name? etc. I assume the auditory neurons are still working so that the words "What is your name" still generate f.m. electric signals that are sent to the brain stem via the auditory nerve. The answer to the question is stored in what we call human memory and my hypothesis is that the memory in the depressed individual is not fully functioning and not stimulating the nerve cells in the motor cortex (they are the cells that when caused to fire, cause the voluntary muscles to contract) to fire and cause the muscles of the lips, tongue, vocal chords etc to contract in the correct way to pronounce the correct answer to the question.

To proceed with the analysis one needs to know the neurophysiological correlate of human memory i.e. the engram. My view is that memory is stored as very small voltage (~1/100 volt) f.m. signals in circuits made up of oligodendrocytes (The glial cells that wrap around the neuron axon. The other glial cells in the C.N.S. are the astrocytes.) Observationally, each O (Short for oligodendrocyte) has multiple microtubules running through it and I hypothesize that the tubules continuously run through multiple O's in a closed loop and that each tubule has its own electric current. Some where on each loop there is a connection with a nerve cell and the current from the O, modulates the current in the nerve cell. That is how memory controls voluntary movement.

In the depressed state, the voltage in the O's falls below that which is necessary to modulate the signal in the nerve cells (Due to reduced oxidation of glucose) and the patient does not respond to the usual stimuli and is deemed depressed. ECT stimulates the oxidation of glucose and brings the voltage of the O's back up to the voltage necessary to modulate the current in the nerve cells.

The current view is that the electric shock from the electrodes directly stimulate the neuroendocrine system to secrete hormones that control the depression. It is not known what hormone(s) actually are effective in controlling the depression nor how the hormone(s) actually control the depression. This interpretation does not explain why the muscles have to convulse in order to control the depression.

In my view the electric shock from the electrodes directly stimulate the voluntary muscles to contract and that causes the stretch receptors to send a voltage pulse to the brain and it is that pulse that directly stimulates the oxidation of glucose and brings the voltage of the O's back up to the voltage necessary to modulate the current in the nerve cells.

An alternative possibility, is that the pulse from the stretch receptors directly cause the neuroendocrine system to secrete hormones that directly stimulate the oxidation of glucose and bring the voltage of the O back up to the voltage necessary to modulate the current in the nerve cells.

It is true that ECT causes a memory loss of events immediately proceeding ECT (Short term memory loss). Presumably, sort term and long term memory loss are stored in different sets of oligodendrocytes. Due to voltage differences between the two sets, ECT destroys the stored electric signals in the short term memory O's and stimulates the voltage of the long term memory O's to stimulate the oxidation of glucose and brings the voltage of the O's back up to the voltage necessary to modulate the current in the nerve cells.

The idea that it is a voltage pulse from the stretch receptors that is the controlling element in controlling depression can be tested (As suggested by Christopher Kingsley Ph.D.) by conducting ECT on quadriplegic patients in depression. Incidentally, ECT requires the consent of the patient. (In the case of the criminally insane, I believe the use of ECT rests with the prison psychiatrist and not with the prisoner.)

A further development of a neuronal model for memory and learning can be found by clicking on the tiles found on the upper left hand side of the title page of my website www.jmkingsleyiii.info I trust you will find them of interest.

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