

Transhuman Neurology

Clinical death involves a cascade of irreversible damage to cellular integrity due to the lack of oxygen and glucose for normal mitochondrial production of ATP. Cells die either by necrosis or apoptosis. This process begins at clinical death and ends when the last skin cell dies months later. In considering transhuman survival of clinical death, most of the body can be discarded for several reasons. First, limbs and organs that are sometimes amputated or otherwise removed while maintaining viability during life, as treatments for certain medical conditions, so are obviously discardable extra baggage during transhuman survival. Most of the body can be discarded at clinical death. The functioning of the parts of the brain that matter for transhuman survival cannot be discarded, but must be repaired from the state they take at clinical death. The ischemic damage to the nerve complex in the brain due to lack of oxygen is basically due to the permanent depolarization of the membrane so that the action potential cannot propagate, and the dumping of neurotransmitters from the synapse, so that synaptic transmission is blocked by lack of reuptake. These are separate problems, each of which must be solved in order to achieve transhuman survival. In nerve cells, when ATP is no longer available due to lack of oxygen, then the K^+/Na^+ pump has no energy and after depolarization cell permeability to Ca and water creates clinically irreversible conditions of cell swelling and no mechanism for removing the Ca from the cell. However, one crucial thing is still fairly intact, the topology of the brain connections. If the key to transhuman survival is the reuse of this topology, then a new polarization layer that can transduce action potentials must be rebuilt on the framework that remains. Fortunately, the steps of this

process can first be used to repair stroke damage and therefore has a clinical application path for development, and patient trials, before the problem of treating clinical death like a disease is even attempted. Once the molecular principles of post-ischemic repolarization are established treatments for non-fatal ischemic insults, then the solution can be expanded to solve the problem of survival after clinical death.

The assumption of topology survival in ischemic damage must be examined since it is the central principle that repolarization strategies will be built on. Electrical synapses do not use neurotransmitter release, and as such present one less hurdle to overcome. The inhibitory or excitatory synaptic clefts stand further from their targets, and may tend to separate further after cell death. Synaptic plasticity as a viable feature of the brain will also be absent in the sense that normally if a cleft detaches it can reattach forming new connections as in some learning models. Electrical synapses would probably be more likely to be preserved after clinical death or ischemic insult than the inhibitory and excitatory synapse, and synaptic plasticity and its role would cease. So, it is assumed that synaptic plasticity is not required for the new repolarization architecture, which means that though brain function would be restored, there might be clinical neurological manifestations of this residual deficit in learning and other cognitive functions that depend on synaptic plasticity.

Post-ischemic perfusion strategies can be used to restore the cell volume in order to maintain as close as possible pre-ischemic topology. Replacing the cell contents including the water, but preserving the critical outer membrane, would require a special material that could perfuse without causing further topological damage. Formaldehyde can be used at ambient temperatures to inhibit some apoptosis. Silicone can be used as a

possible material to replace the cytoplasm. Cryonic approaches should be avoided. The goal of cryonics is to preserve not resurrect tissue function, so perfusions that lower the freezing point of the water in the cell are used. The tissue is then subject to extremely low temperature for preservation in the hope that a future nanotechnology can overcome the warm up issues, or the mapping issues involved in restoration or cloning of new function. Passing through a freezing point is extremely disruptive to the critical topology of the nerve cell membrane. Shards of ice crystals form from the cytoplasmic water content and literally shred what must be preserved at all costs, the topology of the brain. The ideal perfusion would reverse cell swelling and preserve the topology of both the cell membrane and the synapses.

Without a Na^+/K^+ pump, the cell membrane must have a new electrical basis for the action potential. Also, the synapses must have their inhibitory and excitatory function restored. The latter may require another perfusion, which targets the post-ischemic state of the synaptic cleft. This synaptic perfusion would not reverse the damage, but build a new basis for inhibitory and excitatory function. One can imagine that the voltages involved in the new bases for the synapse and the membrane will be different, but still be able to sum the voltages for generation of an action potential, albeit at a transhuman voltage level. This potential as it travels down the preserved topology must be appropriately inhibitory or excitatory at the synaptic cleft, and match polarity with the neurotransmitter/receptor pair that was previously there,

As a stroke treatment, the energy from the surrounding tissue potentials will respect the new artificial replacement and properly conduct in the replacement area. The replacement area would feed live tissue downstream in the normal way restoring higher

purpose of the tissue, e.g. reversing paralysis. In clinical death, there is no feeding energy source, so artificial energy sources must be considered. In this case, the entire brain, or vital subset thereof, is perfused and ready at both the membrane level and the synaptic level. An external artificial electrical source would provide the waking voltage. At this point, EEG should show a modified but otherwise analogous waking pattern. The experience of the individual would be altered, but viable. One advantage of the transhuman brain in this state is that it does not depend on anything but electrical energy and brain topology, both membrane and synaptic, in order to function. The transhuman brain does not depend on oxygen or cell metabolism. There is no need for respiration or food intake. This new energy basis obsoletes the entire clinically dead body except the brain and any sensory organs that can be salvaged. Speech and each sensory organ, ear, eye, tactile, or olfactory would present yet another set of restoration problems. A reasonable first target would be the eye and ear, and possibly some tactile sensation. Because of its reliance on ATP, olfactory function is probably out of reach. Speech will become a particularly difficult if not impossible restoration area for the same reason, and may require an artificial replacement with a direct transhuman brain/computer interface. If the quality of the waking experience is likened to severe brain trauma where the patient is questionably awake but vegetative, and non-communicative, then the transhuman experience is worthless. On the contrary, the goal is restoration of full cognitive thought powered by an artificial electrical source, on the topology of a clinically dead brain. The new basis would extend the life of the individual and their brain function in years by orders of magnitude longer than the current human life span. Aside from transhuman

neurological disease, the other new limits to lifespan would include but not be limited to accident, homicide, war, adverse geophysical events, and adverse solar or galactic events.

In summary, the needed technologies would be the proper perfusions after clinical death to maintain topology and the polarity of the synaptic cleft, control deleterious cell swelling, and provide a new electrical basis, not dependent on ATP, for the action potential's propagation. Other needed technologies would be brain/computer interfaces and an artificial energy source. The problem of the restoration of key sensory organs, sight, hearing, and some tactile function would also be needed. The sensory issue to a large extent is part of current ongoing clinical research for aiding the blind, deaf, and paralyzed. Prototypes of the perfusion technologies could be tested as a therapy in stroke recovery research after passing animal studies.