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Is Brain Death Reversal Possible in Near Future: Intrathecal Sodium Nitroprusside (SNP) Superfusion in Brain Death Patients=The 10,000 Fold Effect

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Abstract : Background: Primary or secondary brain death is also accompanied with vasospasm of the perforators other than tissue disruption & further exaggerates the anoxic damage, in the form of neuropraxia. In normal conditions the excitatory impulse propagates as anterograde neurotransmission (ANT) and at the level of synapse, glutamate activates NMDA receptors on postsynaptic membrane. Nitric oxide (NO) is produced by Nitric oxide Synthetase (NOS) in postsynaptic dendride or cell body and travels backwards across a chemical synapse to bind to the axon terminal of a presynaptic neuron for regulation of ANT this process is called as the retrograde neurotransmission (RNT). Thus the primary function of NO is RNT and the purpose of RNT is regulation of chemical neurotransmission at synapse. For this reason, RNT allows neural circuits to create feedback loops. The haem is the ligand binding site of NO receptor (sGC) at presynaptic membrane. The affinity of haem exhibits > 10,000-fold excess for NO than Oxygen (THE 10,000 FOLD EFFECT). In pathological conditions ANT, normal synaptic activity including RNT is absent. NO donors like sodium nitroprusside (SNP) releases NO by activating NOS at the level of postsynaptic area. NO now travels backwards across a chemical synapse to bind to the haem of NO receptor at axon terminal of a presynaptic neuron as in normal condition. NO now acts as impulse generator (at presynaptic membrane) thus bypasses the normal ANT. Also the arteriolar perforators are having Nitric Oxide Synthetase (NOS) at the adventitial side (outer border) on which sodium nitroprusside (SNP) acts; causing release of Nitric Oxide (NO) which vasodilates the perforators causing gush of blood in brain's tissue and reversal of brain death. Objective: In brain death cases we only think for various transplantations but this study being a pilot study reverses some criteria of brain death by vasodilating the arteriolar perforators. To study the effect of intrathecal sodium nitroprusside (IT SNP) in cases of brain death in which: 1. Retrograde transmission = assessed by the hyperacute timings of reversal 2. The arteriolar perforator vasodilatation caused by NO and the maintenance of reversal of brain death reversal. Methods: 35 year old male, who became brain death after head injury and has not shown any signs of improvement after every maneuver for 6 hours, a single superfusion done by SNP via transoptic canal route for quadrigeminal cistern and cisternal puncture for IV ventricular with SNP done. Results: He showed spontaneous respiration (7 bouts) with TCD studies showing start of pulsations of various branches of common carotid arteries. Conclusions: In future we can give this SNP via transoptic canal route and in IV ventricle before declaring the body to be utilized for transplantations or dead or in broader way we can say that in near future it is possible to revert back from brain death or we have to modify our criterion.

Keywords : brain death, intrathecal sodium nitroprusside, TCD studies, perforators, vasodilatations, retrograde transmission, 10,000 fold effect
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