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Effect GPi Stimulation on Human Thalamic Neuronal Activity: A Decade Later

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It is now more than 14 years that we first published observations of the effect of motor internal pallidum (mGPi) stimulation on the neuronal activity in the thalamic nucleus ventralis oralis anterior (VOA) [1]. One of the main conclusions of this study was that DBS of mGPi decreased firing activity of a subpopulation of VOA neurones. This fact was against the admitted theory of dystonia as a hyperkinetic movement disorder resulting from disinhibition of the motor thalamus [2]. Three years later, Montgomery [3] published similar observations of decreased neuronal activity in 48% of recorded thalamic neurones in the nucleus ventralis oralis posterior during mGPi stimulation this again invalidated motor thalamus disinhibition as the main pathophysiological mechanism for dystonia. Since, GPi DBS or even pallidotomy have imposed themselves as validated techniques for functional treatment for isolated, generalized or focal dystonias and related disorders [4] such as Lesch-Nyhan syndrome [5].

New insights on the effect of GPi stimulation have arisen from modelling the volume of activation in the vicinity of the DBS electrode [6]. Interestingly, these studies pointed that depending on the stimulation intensity and location of the neurones to the current source, axo-somatic decoupling could happen with local somatic inhibition and axonal excitation, resulting in local inhibition with increased axonal transmission. Applied to our observation, local GPi inhibition could coincide to increase inhibitory GABAergic output to the motor thalamus and therefore thalamic neurons inhibition. Today new softwares are developed that combine advance imagery technics such as tractography and volume of activation models to facilitate patient-specific targeting for DBS surgery [7].

In another area, local field potentials (LFP) that measure mainly the synaptic activity around the DBS electrode contacts can be used to perform frequency and coherence analysis of the signal during DBS stimulation in the basal ganglia. This technic offers the advantage to correlate the clinical effect of DBS with change in LFP frequencies. In the GPi LFP as well as studies of neurons coupling during micro recording [8], have pointed to network hyper synchronization as one possible cause of dystonia. For instance, alpha (4-12 Hz) frequencies are associated with phasic dystonia. Interestingly phasic dystonia as well as alpha frequencies are reduced during GPi DBS [9]. In the same way beta (20-30 Hz) frequencies in the subthalamic nucleus are associated to rigidity and akinesia that are both reduced during DBS [10].

In conclusion, recent models and observation on the neuronal activity in the basal ganglia suggest that movement disorders do not result solely from hyper- or hypo-activity in the various nuclei. Today movement disorders are viewed as resulting from abnormal hyper synchronisation in the complex cortico-striato-thalamo-cortical motor loop first described by Alexander et al. [11]. Following this view, GPi DBS by perturbing abnormal hyper synchronisation can alleviate dystonic symptoms as well as Parkinsonian symptoms by "switching off" the abnormal motor loop. Future developments in electrical stimulation modelling as well as the use of closed-loop stimulation based on LFP analysis could help to improve the clinical effect of functional neurosurgery in movement disorders.

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