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Effects of DBS electrode design on the volume of activated tissue

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Since the 1990s deep brain stimulation is a widely used treatment for neurological disorders like Parkinson's disease and dystonia. Stimulation electrodes are implanted stereotactically in the target areas within the human brain. The main target areas used for the therapy of Parkinson patients are the subthalamic nucleus (STN) and the globus pallidus externus (GPe). The electric field generated by the electrode activates nerve fibers in the vicinity of the electrode.

Even though the different target nuclei offer considerable differences in their anatomical structure, only two types of electrodes are currently available. These consist of four cylindrical contacts with a height of 1.5 mm and a diameter of 1.27 mm. The distance between the contacts is either 0.5 mm or 1.5 mm. The contacts can be separately activated and the commonly used approaches are monopolar and bipolar stimulation. Monopolar stimulation causes a wider distribution of the current in all directions in contrast to bipolar stimulation where the current flows between the activated contacts and stimulates a more restricted area.

It is desirable to adjust the electric field and in particular the volume of activated tissue around the electrode with respect to the corresponding target nucleus such that side effects can be reduced. Furthermore, a more selective and partly activation of the target structure is desirable with respect to novel stimulation strategies, e.g. the coordinated reset of neuronal sub-population [1]. Hence we designed and analyzed a DBS electrode with a novel design, allowing more selective activation of the target structure. As the second derivative of the field potential generated by the implanted electrode is correlated to the response of the neural system, we have created a FEM model of the electrode and the surrounding tissue and analyzed the activating function and the volume of activated tissue for the newly developed electrode design.

References

1. Tass PA: **A model of desynchronizing deep brain stimulation with a demand-controlled coordinated reset of neural subpopulations.** *Biol Cybern* 2003, **89:**81-88.