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Mania Induced by Stimulation following DBS of the Bed Nucleus of Stria Terminalis for Obsessive-Compulsive Disorder

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Anterior capsulotomy and stimulation of the anterior limb of the capsule have been used to treat intractable obsessive-compulsive disorder (OCD) [1]. On longer-term follow-up of patients undergoing anterior limb of internal capsule stimulation it was found that the responders had the electrode closure to the bed nucleus of the stria terminalis (BST). This prompted Nuttin and colleagues [2] to propose the BST as a target for refractory OCD.

We recently implanted a patient with refractory OCD using the protocols suggested by Nuttin et al. [3]. The patient had OCD for 15 years. The OCD was in the form of religious thoughts and moral responsibility. The patient would have to perform several religious tasks in order to ensure that no harm would come to his family. He had severe anxiety and moderate depression. He had a Y-BOCS score of 38, a HAM-A score of 21, and a HAM-D score of 19.

He underwent bilateral deep brain stimulation (DBS) of the BST with a Medtronic 3389 lead and rechargeable device. Postoperatively, he was started on stimulation with channel 1 (left brain) at 1–, 2+, 90 μ s/130 pps/0.5 V (pulse width, frequency, and voltage, respectively) and channel 2 (right brain) at 9–, 10+, 90/130/0.5. He showed an immediate improvement. Over the next few days he reported a reduction in his anxiety and OCD symptoms proportionate to the increase in the stimulation voltage. By day 3 of stimulation he was on 1–, 2+, 90/130/3.0 in channel 1 and 9–, 10+, 90/130/3.0 in channel 2. During this time he became very ‘active’. He struck a business deal whilst in hospital and reported to us that he was very happy to conclude this deal, which had been pending for a long time. Over the next 2 or 3 days his behaviour became even more ‘energetic’. He would stay awake through the night and go out of the ward and smoke cigarettes. The psychiatrist attending interpreted this as a mania, but the family told us that this was something that he had expressed in the past and hence not to overreact to it. The drugs were appropriately adjusted to calm him down. Following discharge, he went to his home town which was 2,000 km away. During the following days he developed a full-blown mania. He became very aggressive and had to be virtually restrained. Once he had to be taken to a police station in order to ensure the safety of other family members. When brought back he

burnt down a section of the house in retribution. We asked that he be taken immediately to one of the psychiatric centres in Delhi where he was hospitalized. On our instruction the pacemaker was turned off. This brought an immediate resolution of his mania symptoms, but his OCD came back as before. The window for titrating the OCD and the development of mania was too small, and every time we switched on the implantable pulse generator the mania would return.

We convinced the patient and his relatives to return to Mumbai so that we could try alternative programming. On readmission, we switched off his implantable pulse generator and started slowly stimulating one side at a time, titrating the symptoms. We found that channel 1 was the side responsible for his mania symptoms, as when we stimulated to 2.5 V and above, though the OCD symptoms improved, the mania returned. On stimulating channel 2 we got good response to his OCD symptoms at around 4 V, keeping all other parameters the same. We therefore finally set him up on 1–, 2+, 90/130/0.5 in channel 1 and 9–, 10+, 90/130/3.5 in channel 2, with good control of his OCD without mania.

On analysis of the postoperative imaging it was found that the centre of the active contacts (of channel 2) was located at a laterality of 7 mm, at the level of the posterior border of the AC and the level of the AC-PC, corresponding to the location of the BST. The channel 1 contact point (that provoked mania) was 10 mm lateral, at the level of the posterior border of the AC and at the AC-PC plane and corresponded to the location of the internal capsule.

Mania has been reported following DBS for OCD in an animal model. Hypomania has been reported as a stimulation-induced side effect in patients undergoing DBS for OCD [2, 4]. However, this is the first report of mania induced by stimulation of the BST. This underscores the effectivity of DBS as a unique treatment option offering titrability and reversibility along with possibilities of stimulation-related newer side effects.

References

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