Nucleus accumbens deep brain stimulation decreases ratings of depression and anxiety in treatment-resistant depression.

Background
While most patients with depression respond to combinations of pharmacotherapy, psychotherapy, and electroconvulsive therapy (ECT), there are patients requiring other treatments. Deep brain stimulation (DBS) allows modulation of brain regions that are dysfunctional in depression. Since anhedonia is a feature of depression and there is evidence of dysfunction of the reward system, DBS to the nucleus accumbens (NAcc) might be promising.

Methods
Ten patients suffering from very resistant forms of depression (treatment-resistant depression [TRD]), not responding to pharmacotherapy, psychotherapy, or ECT, were implanted with bilateral DBS electrodes in the NAcc. The mean (±SD) length of the
implanted with bilateral DBS electrodes in the NAcc. The mean (±SD) length of the
current episode was 10.8 (±7.5) years; the number of past treatment courses was 20.8
(±8.4); and the mean Hamilton Depression Rating Scale (HDRS) was 32.5 (±5.3).

Results

Twelve months following initiation of DBS treatment, five patients reached 50%
reduction of the HDRS (responders, HDRS = 15.4 [±2.8]). The number of hedonic
activities increased significantly. Interestingly, ratings of anxiety (Hamilton Anxiety Scale)
were reduced in the whole group but more pronounced in the responders. The [18F]-2-
fluoro-2-deoxy-D-glucose positron emission tomography data revealed that NAcc-DBS
decreased metabolism in the subgenual cingulate and in prefrontal regions including
orbital prefrontal cortex. A volume of interest analysis comparing responders and
nonresponders identified metabolic decreases in the amygdala.

Conclusions

We demonstrate antidepressant and antianhedonic effects of DBS to NAcc in patients
suffering from TRD. In contrast to other DBS depression studies, there was also an
antianxiety effect. These effects are correlated with localized metabolic changes.

Key Words

Deep brain stimulation; functional neuroimaging; major depression; neuromodulation;
nucleus accumbens; treatment resistance

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