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Deep brain stimulation for psychiatric versus neurological disorders: A call for nuance

Abstract: In *Neuroethics: Agency in the Age of Brain Science* (2023), Joshua May arrives at a cautiously optimistic appraisal of deep brain stimulation (DBS) for brain-based disorders. May does not, however, distinguish between disorders that are properly considered neurological and those that are properly considered psychiatric (or psychopathological). After motivating this distinction, I argue that May's discussion of DBS fails to account for the added complexities and potential ethical harms of DBS for psychiatric conditions.

0. Introduction

Joshua May's insightful and uniquely accessible *Neuroethics: Agency in the Age of Brain Science* (2023) presents a picture of the human mind that prioritizes nuance as well as optimism about the prospects of novel neurotechnologies for our collective wellbeing. May's appraisal of the prospects of direct brain interventions falls squarely within this theme of "cautious optimism" that runs throughout *Neuroethics* (May (this volume), p. 1, May (2023), p. 200, 229). In his "Manipulating Brains" chapter, which focuses primarily on the prospects of deep brain stimulation (DBS) for treating brain-based disorders, May argues that the purported risks to patient autonomy, personality and personal identity are "easily overinflated" (p. 67). He repeats this sentiment in his "Précis," in which he states that "…these concerns are often overblown, given that agency and autonomy are dynamic and flexible" (p. 3). Much of this optimism flows from May's adaptation of Neil Levy's (2007) *parity principle* to the case of direct brain intervention, which he refers to as the *Brain Intervention Parity Principle* (henceforth BIPP):

> *Brain Intervention Parity Principle*: A neurobiological treatment does not raise special ethical issues just because it intervenes directly on the *brain* (as opposed to one's body or environment) (May 2023, p. 67, emphasis in original).

In this reply, I will argue that May's deployment of the BIPP applies only to neurological disorders, which he fails to distinguish from psychiatric disorders. Once this distinction

is made, however, I will argue that the situation vis-à-vis DBS for psychiatric conditions is far more complicated and ethically fraught. Situating this critique within May's broader project, this reply should be viewed as a call for more nuance[1], more caution, and a bit less optimism.

1. Neurological versus psychiatric disorders

A foundational issue with May's chapter on brain manipulation procedures such as DBS for brain-based disorders is that he does not distinguish between disorders that are properly considered neurological, on the one hand, and psychiatric (or psychopathological[2]) on the other. Indeed, there are times that May moves from referring to the same set of disorders as psychiatric and then as neurological (p. 66), or as psychopathological and then as neurodegenerative (p. 86). Within philosophy of psychiatry, however, it is common to distinguish the neurological from the psychopathological, given the different levels at which the disorder concepts associated with these two categories operate. Graham (2010), for instance, takes pains to distinguish neurological from psychopathological disorders due to the fact that distinctly mental phenomena (e.g., intentionality, rationality) do *not* factor into our best explanations of neurological conditions but *do* enter into our best understandings of psychopathological conditions. This holds true even for disorders such as Alzheimer's, which may have psychological symptoms and side effects but whose mechanisms can be explicated in wholly neurobiological terms. Arpaly (2005) similarly argues that some mental disorder symptoms are categorically unlike non-mental disorder symptoms, given that the former category can bear content, exhibit content efficaciousness, and can be responsive to reasons. Furthermore, Pickard (2009) argues that even our conception of schizophrenia, which is often thought to be the mental disorder with the best chance of having a distinctly neurobiological origin, cannot do without personal-level psychological properties, thereby distinguishing it (and all other mental disorder categories) from non-mental bodily illnesses.

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[1] Hence the title, which references King and May's (2018) paper.
[2] Note that I will be using "psychopathological" and "psychiatric" interchangeably, both of which should be interpreted as "whichever symptoms, behaviors, and disorders are currently viewed as worthy of psychiatric intervention and treatment".

For the purposes of this reply, it will suffice to note from the above examples that the distinction between neurological disorder and psychiatric disorder is a useful and well-trodden one, and that often this distinction is made in order to effectively highlight the ways in which mental disorder symptoms are deeply unlike (non-mental) bodily disorder symptoms. Bringing our focus back to DBS, this means that the symptoms to be targeted in the case of neurological disorders such as Parkinson's disease (which May primarily focuses on) are going to be very unlike the symptoms to be targeted in the cases of the psychiatric disorders May discusses, such as, e.g., anorexia nervosa (AN). That is, in the former case, providers are seeking to reduce the patient's tremors, rigidity, and dyskinesia (Hariz and Blomstedt 2022). In the latter, providers are seeking to reduce patterns of restrictive dieting behaviors that are often incorporated into the patient's identity and values (Maslen et al. 2015, Tan et al. 2010), which renders the direct targeting of such symptoms a far more complicated proposal.

2. Evaluating the BIPP: Does DBS present "special ethical issues" for psychiatric disorders?

With the distinction between neurological and psychiatric disorders now in hand, let us see how May's BIPP stacks up against the prospects of DBS for psychiatric conditions in particular. In order to illustrate the extent to which May's optimistic portrayal of the status of DBS does not apply to psychiatric conditions in the same way it might for neurological disorders, I will now briefly outline some of the ways in which the situation surrounding DBS for AN differs from the situation surrounding DBS for Parkinson's disorder, given that the two disorders serve as useful markers for each of their respective categories (i.e., neurological disorders and psychiatric disorders, respectively). With respect to the former, Parkinson's is currently the primary disorder for which DBS is used, as well as the disorder that May's chapter opens with and that he dedicates the most time to. It is also a neurological disorder that is progressive (i.e., neurodegenerative) and that tends to be diagnosed in patients later in life (typically after age 60) (Razzak et al. 2020). AN, on the other hand, is the psychiatric condition that May devotes the most space to in his chapter, and it is also arguably one of the most

ethically fraught and complex psychiatric disorders to be included in the discussion of potential DBS therapy. In stark contrast to Parkinson's disease, the average age of onset is quite young (~15 years), and although it is currently difficult to predict which patients will go on to achieve full remission, AN is not considered to be an irreversible or progressive condition (van Eeden et al. 2021, Giordano 2010).

As noted above, the symptoms which comprise a given mental disorder include bona fide mental states with content, which means these symptoms can be incorporated into the subject's broader psychological ecosystem in ways that the symptoms of neurological conditions cannot. Once recognized, this observation quickly leads to a far more complicated picture when assessing the relative merits of DBS for psychiatric disorders such as AN. Firstly, as Maslen et al. (2015, also cited in May 2023) note, the three potential target sites identified in their paper for DBS treatment of AN would each involve their own distinct mechanisms, which would in turn affect markedly different mental processes. This would then lead to the need for (at least) three different bioethical analyses of the relative pros and cons of the proposed site interventions for DBS therapy of AN. At the time of publication, the three DBS mechanisms for AN treatment that had been suggested in the neurological literature were:

1. Modification of aberrant reward processing
2. Increased control over (or reduction of) the drive towards compulsive behavior, and
3. Regulation of aversive mood and affect (p. 17-18).

Maslen et al. go on to highlight the distinct philosophically-relevant consequences for each of these three mechanisms, which are, respectively:

1. The imposition or amplification of a desire for food,
2. The promotion of comparative cognitive control over behavior, and
3. The modification of emotional symptoms or traits (p. 18).

Although a full explication of these mechanisms (and their associated philosophical consequences) is beyond the scope of this reply, it is useful to reproduce them here in order to demonstrate the extent to which these DBS mechanisms look nothing like the DBS mechanisms that are in play for neurological conditions such as Parkinson's. When trying to assess the risks and benefits of employing DBS in treating AN, it is not just a question of whether DBS is justified—it is of whether a *particular mechanism* for DBS is

justified. With respect to Mechanism 1, for instance, Maslen et al. note that the results of the amplification of a desire for food could in some cases cause intense psychological distress (even a "psychological hell," as Wu et al. (2013) suggest) for anorexic patients, who might experience such amplified desires as alienating urges that they may try to "fight against" (Maslen et al. 2015, p. 227).

On the other hand, the prospects of Mechanism 2 appear more promising, given that such an intervention would increase the patient's ability to act on her own considered deliberative goals. While Maslen et al. primarily focus on the positives of this (Mechanism 2) intervention, however, it is also important to note that it has been argued that an increased capacity for cognitive control is partially responsible for the maintenance of anorexic symptomology itself (Brooks 2016, Brooks et al. 2017). If this is in fact the case (indeed, if it is even the case for *some* AN patients who might be considered for DBS therapy), then a regimen of DBS therapy which targeted Mechanism 2 could have the unintended result of increasing the patient's ability to carry out eating disordered behaviors rather than reducing them. In other words, employing Mechanism 2 would potentially enable the patient to better enact whichever behaviors she presently deemed worth engaging in, which could mean either disorder-congruent or disorder-incongruent behaviors depending on the individual's relationship to her disordered behaviors at that point in time[3].

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[3] Since the time of Maslen et al.'s (2015) publication, we have further (though still quite limited) data on the efficacy of DBS for AN. Karaszewska et al. (2022), for instance, performed a meta-analysis of the available studies on DBS for AN as of November 2021, ultimately identifying four (non-randomized, non-controlled) clinical trials that were sufficiently suitable for analysis, with a collective sample size of 56 participants across the four studies. The authors found a statistically large beneficial effect on BMI as well as on secondary quality-of-life measures, though it was determined that the risk of bias for secondary outcome measures was serious while risk of bias for BMI outcome was moderate. This is in part due to the ethical barriers of implementing control groups in these treatment-refractory patient populations. It is interesting to note in the context of my concerns regarding Mechanism 2, however, the target site of Mechanism 2 was not included among the diverse stimulation sites across the four trials. Furthermore, given that the majority of participants (32 out of 56) received stimulation to the nucleus accubens (the target site of Mechanism 1), the concerns raised above regarding the possibility of psychological distress following DBS-induced weight gain absent an accompanying reduction of AN-related psychological symptoms remain relevant, particularly given the high likelihood of relapse among chronic and severe AN patients. In other words, higher quality data on secondary outcome measures (particularly in the long-term) would be needed in order to rule out the possibility that using DBS (particularly Mechanism 1) can have the effect of quickly increasing patient BMI without the accompanying changes in psychological symptoms, which could in turn lead to relapse or other adverse long-term outcomes.

When it comes to DBS treatment for Parkinson's, however, the bioethical and practical issues are comparatively far less severe. Indeed, as May notes, the medication commonly used to treat Parkinson's works by increasing dopamine production and has similar side effects to one of the primary DBS target sites for Parkinson's, which also targets dopamine production. There are, to be sure, still important practical and ethical questions regarding DBS treatment for Parkinson's—one current debate, for instance, concerns the stage of illness at which DBS treatment should first be implemented (Cf. Schupebach et al. 2013, Sperens et al. 2017). Despite this, it does appear that May's BIPP holds true for conditions such as Parkinson's—similar mechanisms appear to be in place for at least some of the target sites, and one mode of treatment does not seem to present truly novel problems over and above those of extant therapies. For the case of AN, however, it is not at all clear that the BIPP applies. Extant AN therapies, such as cognitive-behavioral therapy, general talk therapy, and SSRIs used to treat comorbid depression, work only indirectly on AN symptomology and are not at all analogous in terms of mechanism or associated risk when compared to the proposed DBS therapies for AN (Focquaert and Schermer 2015, Maslen et al. 2015).

Furthermore, the AN case also helps to illustrate the extent to which the concerns regarding authenticity in the context of DBS for psychiatric conditions are not assuaged by May's comparisons to L.A. Paul's (2014) conception of transformative experiences. In responding to the concern that prolonged stimulation from DBS may alter patients' qualitative identity (thereby potentially compromising authenticity), May follows Pugh (2019) in comparing the potentially transformative nature of DBS therapy to Paulian transformative experiences. The thrust of this comparison for May is that, given that virtually all individuals go through some small number of transformative experiences throughout the course of their lifetimes, the fact that DBS patients may undergo transformative experiences as a result of this procedure does not amount to posing any

special bioethical risk. After all, "[t]ranformative experiences are a natural part of neurotypical life" (May 2023, p. 79).

This reply does seem to hold water when considering disorders such as Parkinson's, particularly given the progressive nature of the condition and the stage of life at which such patients would be considering DBS. For the case of AN, however, May's reply does not seem apt. For one thing, the level at which concerns over authenticity play out in the case of AN is far deeper than that of Parkinson's. As Pugh and colleagues (2017) are right to point out, concerns over authenticity in the context of DBS for Parkinson's tend to involve potentially inauthentic *traits* (e.g., excessive gambling or hypersexual behavior). In the context of DBS for AN, however, what we are dealing with is potentially inauthentic *values* (and thus a potentially inauthentic identity altogether) (Pugh et al. 2017). Although May does briefly acknowledge the issues surrounding authenticity in the context of DBS for AN, (2023, p. 76-77), he goes on to answer his own question of whether direct brain interventions pose special threats to personality and identity in the negative by way of his analogy to Paulian transformative experiences. The fact that May does not seem to properly address or circle back to the significance of the authenticity issue in the AN case, however, can be seen as a side effect of his failure to distinguish psychiatric from neurological disorders throughout.

Given that the risks to authenticity (in addition to the complexities involving the proposed mechanisms) are so great in the case of DBS for AN, one may be tempted to respond to the worries I have raised by pointing out that a given AN patient may simply refuse such treatment if it were to become available. This, however, brings us to a final disanalogy between DBS for neurodegenerative conditions such as Parkinson's and DBS for psychiatric conditions such as AN, which is the issue of obtaining valid consent in the latter case. As Maslen et al. stress, AN patients are often subject to both formal compulsion and informal coercion over the course of their treatment. If DBS were to be approved for treating AN, patients may opt for DBS over force-feeding because they may believe (perhaps falsely, in the case of Mechanism 1) that they would be able to stave off the influence of DBS treatment in a way that they would be unable to in the case of force feeding (Maslen et al. 2015). To add to this concern, a budding subfield within

philosophy of psychiatry and biomedical ethics has argued that the extent of informal coercion experienced by psychiatric patients is far more widespread than previously thought and is often unacknowledged or unrecognized by psychiatric practitioners themselves (Cf. Jaeger et al. 2014, Valenti et al. 2015, Silva et al. 2023). The potential influence of psychiatric coercion in the context of DBS is particularly alarming given the invasiveness of the DBS implantation procedure and the physiological fragility of chronic and severe AN patients. To add to these concerns, the youth and gender makeup of this patient population makes them particularly vulnerable to informal coercion and psychiatric paternalism (Radden 2021, Evans 2025), which again distinguishes this case from that of Parkinson's.

4. Conclusion

So, where does this leave us with respect to May's case for cautious optimism regarding DBS treatment? From what I have said, May's conclusions regarding DBS for neurological conditions such as Parkinson's disease remain intact. However, once the much-needed distinction between neurological and psychiatric conditions is introduced, we have seen that this cautious optimism no longer seems to hold for at least some psychiatric conditions such as AN. The BIPP, after all, merely claims that a treatment's intervening directly upon the brain is not sufficient for there being "special ethical issues" (p. 67) regarding said treatment. Where May and I diverge, then, is with respect to whether we believe that there are in fact further considerations that obtain in the case of DBS for psychiatric conditions to merit further ethical consideration.

All of this is not to say, however, that the possibility of DBS for AN should be entirely discarded, much less for other psychiatric conditions that may be less bioethically challenging. That being said, the foregoing discussion suggests that any discussion of DBS for psychiatric conditions will have to be carried out on a disorder-by-disorder, mechanism-by-mechanism, and, perhaps, even a patient-by-patient basis. The sheer practical limitations of applying this level of nuance to the DBS question in psychiatry may well tell against its prospects altogether, but I leave this issue aside for now. In terms of situating this reply within May's overarching project, my intention is

for it to be in keeping with the spirit of emphasizing nuance and complexity that can be found throughout May's work (e.g., Chapters 4 and 5, also King and May 2018). Given that the themes of cautious optimism and nuance may at times be in tension with one another, my hope is that this critique can serve as a constructive counterbalance.

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