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Editorial Comment

This manuscript tried to demonstrate an interesting animal model for deprivation of central dopamine. This animal model has the potential for advancing knowledge of the central mechanism of penile erection. However, this model may have a couple of questionable aspects.

First, unfortunately, the results did not clearly show the relationship between the deprivation of dopamine and the number of erections following apomorphine injection. The authors divided the animals in the groups with bilateral and unilateral 6-OH-DA injections into two subgroups based on the average number of erections in the control group. Eight animals and five animals demonstrated higher and lower numbers of apomorphine-induced erections, respectively. There seems to be no correlation between the number of erections and deprivation of central dopamine. In addition, I do not think this method of data analysis is reasonable. Instead of such analysis, the average numbers of erections of all animals in each treatment group (ie bilateral or unilateral 6-OH-DA injections) are appropriate parameters to prove the authors' hypothesis. The average values of the erections should then be compared with that of the control group statistically. If there is a significant difference, there is clear evidence that supports the authors' hypothesis.

Secondly, the animals injected with 6-OH-DA demonstrated a significantly lower ICP response following cavernous nerve stimulation. However, electrical stimulation of the cavernous nerve may not be a suitable stimulation method for the purpose

of the authors, because the ICP response due to the cavernous nerve stimulation provides information about erectile conditions in peripheral systems (ie the peripheral nervous system and corporal smooth muscle, and peripheral vascular system). In other words, cavernous nerve stimulation can induce a normal ICP response, if the peripheral conditions are normal, even if disorders in the CNS exist. Therefore, for the authors' purpose, measurement of ICP during electrical stimulation of the CNS (ie the MPOA,^{1,2} or apomorphine-induced erectile response³ are more suitable.

To this end, this animal model that mimics Parkinson's disease may have potential, but more work and better analyses are needed for it to become a reliable animal model.

Y Sato

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