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read with great interest the recently published article by Arai et al¹ in the *American Journal of Neuroradiology*. In this article, the authors claimed that the asymmetric visualization of nigrosome 1 is affected by the magic angle and susceptibility. They also suggested that these challenges in visualization are caused by the anatomic slant structure of nigrosome 1. For proper assessment of nigrosome 1 on MR imaging, it is of the utmost importance that researchers should be familiar with its anatomy and obtain high-spatial-resolution imaging to reduce partial-volume effects. In this regard, I am concerned about the methods used by the authors of this article.

First, the authors claimed that the MR imaging visualization of nigrosome 1 is often poor because of the asymmetry of this cell cluster, regardless of whether nigrosome 1 is healthy. I tried to find any relevant studies with regard to the cell cluster asymmetry in nigrosome 1 but to no avail. It, therefore, would be better to add any reference to this description.

Second, it has been reported that the CNS does not show magic angle effects (T2 prolongation at certain angles) because it has no ordered collagen. It, therefore, is implausible to describe that visualization of nigrosome 1 is affected by magic angle effects. Because a multiecho gradient recalled-echo sequence was used in this study, there may be changes of T2* contrast in certain regions of the white matter. It has been shown that the relative orientation of white matter fibers to the B_0 field significantly affects T2* measurements, and the dominant source of this orientation dependency is susceptibility effects from myelin. Myelin, however, is not, or is scarcely, present in nigrosome 1. Thus, neither magic angle effects nor susceptibility effects may affect the image contrast in healthy nigrosome 1.

Third, even if nigrosome 1 visualization is affected by the magic angle effects due to its slant, nigrosome 1 should have higher signal intensity in nontilted head imaging than in tilted head imaging. The opposite results, however, were presented in this study.

Fourth, the authors indicated nigrosome 1 on the susceptibility map in Fig 5. The indicated structures with hypointensity are

connected to the slightly hypointense regions below the substantia nigra. Nigrosome 1, however, is located between the 2 hyperintense areas on quantitative susceptibility mapping because it is seen as a hyperintense region between the 2 hypointense regions on SWI (also known as the "swallow tail sign"). Nigrosome 1 is also located more anteriorly than that in Fig 5. The structure indicated by the authors, rather, would be the medial lemniscus (based on the *Duvernoy's Atlas of the Human Brain Stem and Cerebellum*), not nigrosome 1, which is supported by the fact that it shows orientation-dependent signal changes because of its myelin content. It would have been better to test if higher-spatial resolution imaging (eg, $0.5 \times 0.5 \times 1.0 \,\mathrm{mm}^3$) shows similar results because nigrosome 1 is very small and easily affected by a partial-volume effect.

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