Do musical hallucinations have a neuro-logical cause?

Wodarz et al. present a case of musical hallucinations attributed to basal ganglia calcifications. The patient, however, satisfies only one of the four criteria for determining a neurological as opposed to epileptic or otological cause for musical hallucinations. There was no evidence for epileptic activity, but there was deafness and unfitness. The patient had a small aneurysm, but no other cerebellar symptoms, with apparently no check if this anaxia was partly or wholly of vestibular origin. She had chronic hypoparathyroidism, yet no mention was made of any drugs she was taking.

These criteria were set up on the general scientific principle that if most cases of a phenomenon are caused by a known factor (case 1), it should be very cautious before concluding that the remaining cases are due to a second, quite different factor (brain disease), rather than being variants of the first cause.

Wodarz et al. offer a simplistic version of the otogenic theory, which, not surprisingly, they then dismiss. It is clear that hearing loss itself is not a sufficient factor, and indeed drugs can induce musical hallucinations in people with no underlying neurologists who, considering a carotid lesion a coincidental feature, don’t even perform carotid ultrasound in patients with lacunar stroke.

Moulin and Bogoausljevsky reply:

We are delighted that the data from Maastricht confirm our study—that is, that potential sources of embolism to the brain are not uncommon in patients with acute stroke presenting as astatic hemiparesis.

We also agree with the point that the exact etiology of brain infarct is presumed rather than proven in most instances, but it may be presented also from another perspective: the presence of a small deep infarct is only compatible with undiagnosed small vessel disease, although there is no test which can show this in vivo; however, this assumption does not rule out the possibility that it may also be embolic, especially if a stroke is actually embolic. It is indeed shown as the argument of the coexistence of cardioembolic territorial infarct with silent small deep infarcts (‘Lodder’s ref’), could also be presented the other way around to support the concept that small deep infarcts may often be embolic in origin.

The answer to the question asked by Lodder about the performance of carotid endarterectomy in patients with a lacunar infarct ipsilateral to a carotid territory, in which these patients were included and contributed to the global improvement in surgery for medical treatment alone. Thus the neurologists mentioned by Lodder are not following the scientific data, which showed the uselessness of surgery in symptomatic patients with >70% carotid stenosis.

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