source of embolism is substantial. However, a potential cardioembolic source, large vessel disease, also large vessel disease and the absence of small vessel disease, one of which becomes symptomatic first.

The fact that most silent brain infarcts in patients with cardioembolic territorial infarcts are small lesions also point at this possibility. I wonder whether Moulin and Bogousslavsky would recommend carotid endarterectomy in patients with AH/DCS with a small deep hemispheric infarct on brain imaging and a >70% ipsilateral internal carotid artery stenosis. Relevant to their statement is that small cerebral infarcts, with apparently no check if this axioma was partly or wholly of vascular 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, then the 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 other symptoms. They find that the extra factor is an endolympathic hydrops, as seen in incipient Meniere’s disease. This case can fluctuate or progress hearing loss, hyperacusis, or no deafness but with auditory symptoms, or with features of Meniere’s disease, particularly vertigo. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects, deaf ears are even more sensitive to cochlear hydrops (slight or unilateral or asymmetric or low tone losses, cochlear losses, etc.). Other factors which could trigger an acute hydrops in the inner ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss, or, in this case, electrolyte imbalance.

Wodoraz et al.1 claim that musical hallucinations have not been reported before in postural hypoparathyroidism. There is, however, a case2 distinguished by the richness and intensity of hallucinations, which is very instructive. As neopoamyotonia showed absolutely no brain pathology. (There are many other reports that I have not checked.) She heard music and bells ringing and talked of composing a symphony! In other hallucinations she felt herself being thrown through the air or down a hole. Her sight was very poor and she had vivid visions thought to have been of retinal origin. In another patient she reported the experience of flying through the air and oscillations in the head. No mention was made of otoacoustic emissions. An autobiographical account, however, by a social worker3 confirms that sensory hallucinations are prominent features of psychosis. She felt herself suspended in mid-air or upside down, or suddenly being moved.

These hallucinations could be checked by having a light on after dark, but blinding her eyes. Her balance was very poor. She had brief Menieriform attacks comprising nausea, a whirling vibration in her head like an egg beater, deafness, and mental confusion. She could tolerate the cramps and other symptoms, but most of all she was in terror of these vibrations, later described as loud buzzing, roaring in the ears, noises in the head, or pulsations. The auditory sensation depended on the rapidity of the vibration. It only needs a couple of cases with consistent neurological lesions but no deafness to completely sink the otogenic theory4 and so remove the causal possibilities for musical hallucinations. My previous appeal5 for such a case has been unsuccessful, so it is reasonable to assume that there is no case in the medical literature. Wodoraz et al.6 state that musical hallucinations can occur with brainstem lesions, but give no reference. Please could they cite one which includes patients without cochlear or neural deafness? I appeal again to neurophysiologists with new norm-deaf cases of musical hallucinations.

Moulin and Bogousslavsky 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 axial 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 certainly compatible with 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 indeed shown by 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 supratentorial small infarct and ipsilateral >70% carotid stenosis seems to lie in the randomised trials of carotid endarterectomy, in which these patients were included and contributed to the global superiority of surgery over medical treatment alone. Thus the neurologists mentioned by Lodder are not following the scientific data, which showed the usefulness of surgery in symptomatic patients with >70% carotid stenosis.


Do musical hallucinations have a neurological cause?

Wodoraz et al.7 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.2 There was no evidence for epileptic activity, but there was deafness and unilaterality. The patient had a true ‘cerebellar ataxia’ but no other cerebellar symptoms, with apparently no check if this axioma was partly or wholly of vascular 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, then the 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 other symptoms. They find that the extra factor is an endolympathic hydrops, as seen in incipient Meniere’s disease. This case can fluctuate or progress hearing loss, hyperacusis, or no deafness but with auditory symptoms, or with features of Meniere’s disease, particularly vertigo. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects, deaf ears are even more sensitive to cochlear hydrops (slight or unilateral or asymmetric or low tone losses, cochlear losses, etc.). Other factors which could trigger an acute hydrops in the inner ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss, or, in this case, electrolyte imbalance.

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Wodoraz et al. reply: Gordon’s interesting comments and the additional three patients with postural hypoparathyroidism and associated psychosis are very much appreciated. The musical hallucinations in these patients, however, were associated with various other psychiatric symptoms such as delusional state, epileptic seizures, and paranoid ideation. On the contrary, our patient, in terms of psychopathological syndromes, presents with isolated musical hallucinosis. As in his previous comments to other papers Gordon attributes musical hallucinations to a peripheral otogenic mechanism.1 His support for this hypothesis, even interpreted by Gordon himself, is based on some misunderstandings.2

1. (1) We did, in fact, report the drugs given to our patient during inpatient treatment. In the six months preceding the hallucinations she received the equivalent of 2-3 mmol Ca2+ intravenously only when symptoms of tetany occurred (once or twice per month). This might well result in an acute ear hard of hearing induced by Gordon’s patient. As the musical hallucinosis disappeared, however, after addition of oral dihydrotachysterol plus oral and intravenous Ca2+, a drug induced