source of embolism is substantial. However, a potential cardioembolic source, large vessel disease, or even the absence of hypertension do not exclude the presence of small vessel disease as the cause of a lacunar infarct presenting with AH/DCHS. Patients could have more than one type of vascular disease, one of which becomes symptomatic first. The fact that most silent brain infarcts in patients with a cardioembolic territorial infarct are small lesions also point at this possibility.[3] I wonder whether Moulin *et al*[1] would recommend carotid endarterectomy in patients with AH/DCHS with a small deep hemispheric infarct on brain imaging and a > 70% ipsilateral internal carotid artery stenosis? I know neurologists who, considering a carotid lesion a coincidental feature, don't even perform carotid ultrasound in patients with lacunar stroke.

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1 Moulin Th, Bogousslavsky J, Chopard JL, Ghika J, Crépin-Leblond Th, Martin V, Maeder P. Vascular ataxic hemiparesis: a re-evaluation. *J Neurol Neurosurg Psychiatry* 1995;**58**:422–7.
2 Lodder J, Bamford J, Kappelle J, Boiten J. What causes false clinical prediction of small deep infarcts? *Stroke* 1994;**25**:86–91.
3 Boon A, Lodder J, Heuts-van Raak L, Kessels F. Silent brain infarcts in 755 consecutive patients with a first-ever supratentorial ischemic stroke. Relationship with index-stroke subtype, vascular risk factors, and mortality. *Stroke* 1994;**25**:23–84.

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 ataxic hemiparesis.

We also agree with the point that the exact aetiology 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 commonly compatible with underlying 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 source of embolism is indeed shown. The argument of the coexistence of cardioembolic territorial infarct with silent small deep infarcts[1] (Lodder's ref[3]), 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 deep 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 carotid surgery in symptomatic patients with >70% carotid stenosis.

1 Boon A, Lodder J, Heuts-van Raak L, Kessels F. Silent brain infarcts in 755 consecutive patients with a first-ever supratentorial ischemic stroke. Relationship with index-stroke subtype, vascular risk factors, and mortality. *Stroke* 1994;**25**:23–84.

Do musical hallucinations have a neurological cause?

Wodarz *et al*[1] 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 tinnitus. The patient had "cerebellar" ataxia, but no other cerebellar symptoms, with apparently no check if this ataxia was partly or wholly of vestibular origin. She had chronic hypoparathyroidism, yet no mention was made of any drugs she was taking.

These criteria[2] were set up on the general scientific principle that if most cases of a phenomenon are caused by a known factor (in this case ear disease), one 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*[1] 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 normal hearing.[3] It seems that the extra factor is an endolymphatic hydrops, as seen in incipient Meniere's disease. This can cause fluctuating or progressive hearing loss, hyperacusis, or no deafness but with audiosensitivity, or tinnitus, or vertigo. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects; deaf ears are even more susceptible to drugs.[2] In their own case Wodarz *et al*[1] attribute deafness to presbyacusis. This is an overused default diagnosis, which should be confined to patients with smooth high tone symmetric losses. Their patient had mild pancochlear perceptive hearing loss, worse on the left, far more consistent with at least some hydrops component. A third of patients with hypoparathyroidism[4] had inner ear hearing loss, not presbyacusis but suggestive of cochlear hydrops (slight or unilateral or asymmetric or low tone losses, pancochlear losses, etc). Other factors which could trigger an acute hydrops in a deaf or normal ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss,[3] or, as in this case,[1] electrolyte imbalance.

Wodarz *et al*[1] claim that musical hallucinations have not been reported before in postsurgical hypoparathyroidism. There is, however, a case[5] distinguished by the richness and intensity of hallucinations, which is very instructive as necropsy showed absolutely no brain pathology. (There are many other reports that I have not checked.) She[5] 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 the main hallucinations were a feeling of flying through the air and oscillations in the head. No mention was made of otological examinations. An autobiographical account, however, by a social worker[6] confirms that vestibular and auditory 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 and by opening her eyes. Her balance was very poor. She had brief Menieriform attacks comprising nausea, a whirring 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 theory[2 3] and so reduce the causal possibilities for musical hallucinations. My previous appeal[2] for such a case has been unsuccessful, so it is reasonable to assume that there is no case in the medical literature. Wodarz *et al*[1] 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 neurologists to publish new non-deaf cases of musical hallucinations.

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1 Wodarz N, Becker T, Deckert J. Musical hallucinations associated with post-thyroidectomy hypoparathyroidism and symmetric basal ganglia calcifications. *J Neurol Neurosurg Psychiatry* 1995;**58**:763–4.
2 Gordon AG. Musical hallucinations. *Neurology* 1994;**44**:986–7.
3 Gordon AG. Benzodiazepines and the ear—tinnitus, hallucinations and schizophrenia. *Can J Psychiatry* 1993;**38**:156–7.
4 Ikeda K, Kobiyashi T, Kusakari J, Takasaka T, Yumita S, Furukawa Y. Sensorineural hearing loss associated with hypoparathyroidism. *Laryngoscope* 1987;**97**:1075–9.
5 Schoelly ML, Heuscher JE. Contribution à l'étude des psychoses tétaniques. *Monatsschrift für Psychiatrie und Neurologie* 1950;**119**: 141–55.
6 Dda CF. Psychological states resulting from parathyroid deficiency. *Journal of Abnormal and Social Psychology* 1939;**34**:481–96.

Wodarz et al reply:
Gordon's interesting comments and the additional three patients with postsurgical hypoparathyroidism and associated psychosis are very much appreciated. The musical hallucinations in these patients, however, were associated with various other psychiatric symptoms such as delirious state, epileptic seizures, and paranoid ideation. On the contrary, our patient, in terms of psychopathological syndromes, presented 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 in our patient is, however, based on some misunderstandings.[2]

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