source of embolism is substantial. However, a potential cardioembolic source, large vessel disease, the absence of carotid stenosis do not exclude the presence of small vessel disease as the cause of a lacunar infarct presenting with AH/DCHS. Patients could harbor other sites of small vessel disease, one of which becomes symptomatic first. The fact that most silent brain infarcts in patients with AH/DCHS with a small deep hemispheric infarct on brain imaging and a > 70% ipsilateral internal carotid artery stenosis is not uncommon. The neurologists who considering a carotid lesion a coincidental feature, don't even perform carotid ultrasound in patients with lacunar stroke.

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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 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 not necessarily 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 mechanism is indeed shown. 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 AH/DCHS and insular lesion 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 neuro- logical cause?

Wodarz et al1 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 otorlogical cause for musical hallucinations.2 There was no evidence for epileptic activity, but there was deafness and uninit. The patient had a history of "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 were set up on the general scientific principle that if most cases of a phenomenon are caused by a known factor (this case is an exception), it could 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 al2 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 history of deafness. 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 auditory-perceptive "ataxia", but no other physical signs. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects; deaf ears are even more 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 normal ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss,3 or, as in this case,4 electrolyte imbalance.

Wodarz et al2 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 necropsy showed absolutely no brain pathology. (There are many other reports that I have not checked.) She1 heard music and bells ringing and talked of composing a symphony! In other hallucinations she felt her self 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 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 worker4 who had auditory hallucinations (in a hospital for the deaf) 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 light on after dark and examining 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 theory1 and so re-evaluate the causal capacities for musical hallucinations. My previous appeal2 for such a case has been unsuccessful, so it is reasonable to assume that there is no case in the medical literature. Wodarz et al2 state that musical hallucinations can occur with brain-stem lesions, but give no reference. Please could they cite one which includes patients without cochlear or neural deafness? I appeal again to neuro-otology with "new non- deaf cases of musical hallucinations."

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Do musical hallucinations have a neurological cause?

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