had CT evidence of cerebral parenchymal damage and fully 63% had a 6-month outcome of severe disability, vegetative state or death.[3]

Some partial epileptic disorders may indeed be related to peripheral vestibular or cochlear insults, presumably through aberrant cortical reorganization, but would need to develop over a much longer period than the early seizures under discussion, and would (like their associated EEG abnormalities) represent, at that time, direct cortical involvement. This idea remains largely hypothetical. In contrast, focal cortical epileptic discharges causing symptomatic vertigo (or auditory hallucinations) have been demonstrated numerous times by direct brain recording and/or stimulation.[4-6]

The labyrinth may well be implicated in some curious neurologic phenomena. There is, however, no reason to invoke a labyrinthine etiology for the majority of early post-traumatic seizures.

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To the Editor:

Musical Hallucinosis With Brainstem Lesions

In 1994[1] I stated that there was no known cause of musical hallucinations (MHs) due to a neurological as opposed to otological lesion, and challenged neurologists to find such a case. Douen & Bourque[2] report a man with MHs of allegedly brainstem origin. Far from being a pure example of this, their case has 3 of the 4 features disqualifying a non-otological neurological case, i.e.: Drugs were not excluded – the man was alcoholic. Alcohol withdrawal triggers MHs from normal ears, even more so if abnormal. He presented with Meniere symptoms (nausea, vomiting, imbalance).

Deafness of unproven brainstem origin occurred. A more peripheral lesion was far more likely, since: 1) There was vestibular involvement, with nystagmus to the right; 2) Four adjacent cranial nerves were also involved; 3) Latent ear infection was a more plausible cause for the homolateral cerebellar abscess and circumscribed meningitis than bloodborne listeria; 4) The limited audiological testing showed speech discrimination apparently poorer than expected from the audiogram. This indicates a neural deafness, the usual cause for which is eighth nerve disorder; 5) As noted,[2] all ten prior cases of "brainstem" auditory hallucinations (AHs) had hearing loss ipsilateral to the AH. In none had sufficient audiological testing excluded a peripheral cause.

Douen & Bourque stated:[2] "Clearly, AHs which include verbal content or recognition of a specific melodic line must involve activation of cerebral cortical areas." If it is implied that AHs must be generated in those areas, then this needs critical discussion. Just because AHs end up in the brain, it doesn't mean they started there.

Douen & Bourque wrongly state[2] there is no evidence for a peripheral generator of AHs. Supposedly spontaneous or random cochlear nucleus activity is of carotid origin[3] – the noise of blood flow being transduced into cochlear electrical activity. Artistic creativity in composers[4] and in some poets seems to depend on MHs from hypersensitive ears. Even if one rejects this specific mechanism, it is clear that they really transcribed rather than composed, as music, even whole pieces, rose unbidden from the unconscious, consistent with a subcortical generator. A Heartsongs CD is commercially available on which real electrocardiograms generated melodies;[5] musical composition may involve "the recreation by the mind of the body's own naturally complex rhythms and frequencies". Even cochleas may be superfluous, as a temporal code in the auditory nerve alone is capable of defining musical pitch.[6] Variation in pulse rate on single bipolar electrodes can in totally deaf subjects result in pitch changes sufficiently salient to support musical interval perception.

The topic of insight shows how muddle and illogical current formulations of AHs are. Douen & Bourque[2] say their patient's "preserved insight and clear sensorium establish that these are hallucinations, not... tinnitus". Some definitions of AH use insight for the exact opposite purpose: if patients say they hear noises in their ears they are said to have tinnitus, whereas if they hear similar noises from non-existent environmental objects, they have AHs. Also, given that people with normal minds and brains but defective ears can have AHs that they falsely believe are real, it is not odd that in their case of alleged brainstem origin, insight was retained? This is not an isolated occurrence. Berrios[7] found that insight into MHs correlated with brain disease, not ear disease. In other words, the more MHs were attributed to brain disease, the more insight the patients had. Incidentally, Berrios was the main source for Douen & Bourque's assertion of a direct cerebral role for AHs. However, Berrios, despite his historical expertise, only found 46 cases of MH to review, 31 of whom were definitely deaf, strongly indicating otherwise. If brain lesions cause MHs, where are these cases? Douen & Bourque suspect psychiatric disorder for AHs with impaired insight. All the evidence points to insightlessness, in psychotics or others, being of otological not neurological origin.[4]

Douen & Bourque propose[2] that AHs of aural or brainstem origin result from deafferentation. This ignores tinnitus, of which many with MHs complain bitterly (e.g., Margery Kempe, Luther, Rousseau, Schumann).[4] They do not say if their patient heard tinnitus (e.g., buzzing or humming). Confusingly, Berrios[7] labeled all such sounds (as occur in ENT patients) hallucinations, consistent with most psychiatric definitions (perceptions without objects).

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Reply from the Authors:

Dr. Gordon purports that complex auditory hallucinations (AH) are invariably peripheral (otologic) in nature, never central (neurologic). In fact, Dr. Gordon has challenged neurologists to find a case of the latter.[1] However, careful examination of reported cases[2-5] lends considerable support to a neurologic basis for complex AH. In particular, we draw Dr. Gordon's attention to the article of Murata, et al., describing a 54-year-old Japanese man with left ear deafness since childhood.[3] This patient experienced *no* AH until he presented with right sided hearing loss attributable to pontine tegmental hemorrhage. This patient's bilateral deafness was supported by audiogram. Furthermore, right BAER during his deafness and AH showed preservation of wave I only suggesting normal right cochlear function. Importantly, his AH disappeared when his right sided hearing returned; BAER also normalized. In this case, the patient's longstanding left sided deafness secondary to otologic disease was a very unlikely factor in generating AHs.

We recently reported a case of a 43-year-old man with Listeria Rhombencephalitis who developed acute right sided hearing loss and complex AH.[5] MRI demonstrated hindbrain abscess and edema. This case was in accord with previous reports,[2-4] in that there were clear temporal relationships between the unilateral hearing loss and development of complex AH and also between the resolution of the hearing loss and disappearance of the AH. Although our patient had a history of ethanol abuse, there was no evidence of alcohol withdrawal. Furthermore, his nausea, vomiting, and imbalance ("Meniere's symptoms", as referred to by Dr. Gordon) were only part of a larger constellation of symptoms related to involvement of rhombencephalon, including long tract signs. The suggestion of a latent ear infection and subsequent meningitis as proposed by Dr. Gordon is unfounded. Listeria Rhombencephalitis often produces multiple brainstem abscesses and asymmetric CN involvement with long tract signs.[6] In addition, there was no meningeal enhancement on a gadolinium infused MKRI.

In our article, we discussed the complexity of the genesis of AH and acknowledged that some cases result from lesions of the peripheral auditory pathways. However, we feel that our case[5] and others involving acute brainstem injury[2-4] likely involve a deafferentation "release" phenomenon. Patients reporting voices as part of complex AH often hear these voices in a familiar language. The previously referred to Japanese patient heard oriental voices,[3] while our patient heard English,[5] suggesting that higher cortical centres are involved since a peripheral generation of audible sound should be non-specific.

The Heartsongs CD[7] simply demonstrated that melodies can be generated by digital tape recordings of heartbeats of fifteen people. We fail to understand how Dr. Gordon can imagine that our patient's peripheral auditory pathways could have exactly translated the "body's own naturally complex rhythms and frequencies" into a well known song, such as Summer Girl. We can accept that spontaneous activity in peripheral auditory pathways may be the generator of elementary sounds involving rhythmic sequences and pitch variation. To postulate that the cochlea or auditory nerve by themselves could store and "replay" complex sound sequences corresponding to previously learned songs would seem fanciful.

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