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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.<sup>1</sup> However, careful examination of reported cases<sup>2-5</sup> 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.<sup>3</sup> 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,<sup>2-4</sup> 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.<sup>6</sup> 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<sup>5</sup> and others involving acute brainstem injury<sup>2-4</sup> likely involve a deaf-ferentation "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,<sup>3</sup> while our patient heard English,<sup>5</sup> suggesting that higher cortical centres are involved since a peripheral generation of audible sound should be non-specific.

The Heartsongs CD<sup>7</sup> 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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