A 44 year old woman was admitted to our clinic because of progressive multiple sclerosis. One year before admission she developed paresis of the legs, and subsequently of the arms. She became incontinent for urine and feces. She also complained of numb feelings and muscle cramps in her legs. The medical history mentioned hypertension for which she used propranolol and hydrochlorothiazide. The family history was negative for multiple sclerosis. On examination there was vertical nystagmus, slight paresis of the arms and legs, incordination of the arms, and loss of sensation from a mid-thoracic level. The tendon reflexes of the legs were very brisk, and both plantar responses were extensor. Examination of CSF showed eight white cells/mm³ (all lymphocytes), and an intrathecal production of IgG and IgM. Brain MRI and the cervical part of the spinal cord showed multiple white matter lesions. Additional investigations excluded other diseases—for example, borreliosis and lupus erythematosis. A 10 day treatment with daily administration of 1000 mg intravenous methylprednisolone was started. Methylprednisolone was given in its injectable form, methylprednisolone sodium succinate, which hydrolyses to methylprednisolone in the body. The infusion period was one hour. Because of cystitis she also received trimethoprim. One day after the intravenous methylprednisolone course had ended, the patient developed generalised urticaria which disappeared after a few days, and which could have been induced by either drug. After informed consent of the patient it was decided to give another course, as the intravenous methylprednisolone course improved her multiple sclerosis. To guarantee minimal risk, we gave 1000 mg intravenous methylprednisolone under close monitoring. After the first infusion there was a reactivation of the skin rash, and difficulty with swallowing and breathing, suspicious of anaphylaxis. This was given intravenously, after which the symptoms immediately resolved. Because of clinical improvement, therapy was continued with a 1000 mg course of intravenous methylprednisolone divided into two, again under close monitoring. No symptoms developed. The next day we gave the full 1000 mg dose of intravenous methylprednisolone leading to high plasma concentrations. However, raised concentrations were not found. The histamine release reaction for methylprednisolone sodium succinate was not indicative of an IgE mediated reaction. Skin tests have been used to investigate the nature of side effects to intravenous methylprednisolone. We showed that skin tests are unreliable as they also gave positive reactions in the healthy volunteers. The "allergic" reactions are probably not basophil mediated. Treatment with methylprednisolone, and a radio allergosorbent test (RAST) for IgE antibodies was positive. No information regarding the RAST procedure was mentioned. Methylprednisolone was administered corticosteroids in patients have been found but occur infrequently (0.3% of the patients). Risk factors for developing allergic reactions after receiving intravenous methylprednisolone are asthma and aspirin intolerance. Our patient had no history of asthma or other allergies. Skin tests have been used to investigate the nature of side effects to intravenous methylprednisolone. We showed that skin tests are unreliable as they also gave positive reactions in the healthy volunteers. The "allergic" reactions are probably not basophil mediated. Treatment with methylprednisolone, and a radio allergosorbent test (RAST) for IgE antibodies was positive. No information regarding the RAST procedure was mentioned. Methylprednisolone was administered corticosteroids in patients have been found but occur infrequently (0.3% of the patients). Risk factors for developing allergic reactions after receiving intravenous methylprednisolone are asthma and aspirin intolerance. Our patient had no history of asthma or other allergies. Skin tests have been used to investigate the nature of side effects to intravenous methylprednisolone. We showed that skin tests are unreliable as they also gave positive reactions in the healthy volunteers.
Unilateral auditory hallucinations: ear or brain?

Brasic and Perry's convincingly describe a boy with unilateral otopathic auditory hallucinations. However, their literature review is very much biased towards conclusions. For four CNS causes for auditory hallucinations other than from a hyperactive ear, they also do not cite a relevant prior case. Their paper starts: "Unilateral auditory hallucinations...are associated with contralateral CNS lesions". Their only supporting reference is Toulouse (1892), who reviewed four cases of unilateral auditory hallucinations from homolateral ear disease. Four of these patients had, there were peripheral lesions sufficient to trigger tinnitus and auditory hallucinations, although in alcoholic patients these can probably be triggered from ears with relatively normal hearing. It is noteworthy that the EEG changed from an awake pattern to seizure activity. In fact, it is consistent with peripheral impulses funnelling up via the thalamus.

The volunteers of Ketter et al. consistently (29 out of 32 subjects) reported procaine induced auditory hallucinations (unusual buzzing, ringing, or electronic sounds). They considered procaine a selective limbic activator, even though no change in cerebrum blood flow corresponded to the "auditory hallucinations". In an ear, nose, and throat clinic the above noises would definitely be labelled tinnitus, and an obvious peripheral cause usually found. In line with Grisar this is consistent with peripheral pathology, yet no evidence to explain the onset of the neural activity.

In their 1986 review of hallucinating schizophrenic patients, Silbersweig and colleagues (29 out of 32 subjects) reported procaine cochlear hyperactivation (increased wave I and II amplitude in dogs); in view of the link between epilepsy and the ear, it is noteworthy that the EEG changed from an awake pattern to seizure activity. The 1986 review of sensory deprivation by Petrof and colleagues was cited as an example of auditory hallucinations from sensory disinhibition. I reviewed some of literature, concluding that for musical hallucinations there is as much evidence for ear disease and labyrinthe hyperactivity as when they occur in all other conditions and diseases. Depivation is a much simpler psychological condition; cochlear hyperactivity caused by white noise is used to mask environmental sounds, whereas if all background noises are reduced, normal subjects will start to have tinnitus. I recently tested a 16 year old dyslectic patient who had never known silence ("I think I have a permanent noise running through it"); he had no symptoms of tinnitus. Sensory deprivation is more likely than deprivation, as in fact misprinted!

A similar case to that of Brasic and Perry throws considerable light on pathophysiologic processes involved. Both were tormented by voices of devils and demons, hearing symptoms of unilateral ear disease; saw the same doctor; had fever-interchanging; had fiery visions (fire surrounded by cockroaches, devil transforming into burning wisp of straw); possible fluid in one middle ear (blocked grommet plus air-bone gap; fiery discharge from one ear).

Conclusively omitted was any mention of tinnitus, the most likely generator of the auditory hallucinations. Fortunately, the other case was Martin Luther, who clearly described ringing in his ears, undendurable buzzing, thundering, cracks, thumps, etc. Once, he had a musical hallucination (bells of specific church towers) which woke him in bed because of noises in his head. Curiously, Luther did not have a simple demonic or religious explanation for his torments. Instead he blamed Satan for his Meniere's symptoms (headache, episodic vertigo, tinnitus), which in turn he recognised caused his hallucinations. In fact his symptoms were typical of temporal lobe epilepsy which is supported by good evidence that he had "French disease".

Brasic and Perry still assert a CNS origin for auditory hallucinations they need an original case report, not reviews or secondary sources. Repeated appeals for non-otological neurological musical hallucinations have failed (suggested cases with brainstem findings were also dejected). I would now like to broaden the challenge to cover auditory hallucinations as well. Unless someone can come up quickly with a case of auditory hallucination due to a clear neurological lesion in someone with normal ears and hearing, the only proved cause of auditory hallucinations is otological.

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Brasic and Perry reply: Gordon conjectures that otological pathology is the necessary and sufficient condition for auditory hallucinations. We disagree. We hypothesise that auditory hallucinations have many aetiological factors and that they are identified as otological, neurological, neuropsychiatric, and combined. Auditory hallucinations may result from the multiple effects of otopathological, such as altered signal transduction in hair cells. For example, in response to minimal environmental stimuli, disturbed auditory perception can occur when inner ear frequencies producing white noise perceived as tinnitus in some persons. Auditory hallucinations may also result from neurological illnesses, including after right temporal lobectomy for intracerebral hemorrhage without seizures. We are preparing a manuscript on auditory hallucinations in neurological disorders. Auditory hallucinations due to neuropsychiatric disorders are being studied, particularly in schizophrenia. On functional MRI, two patients with schizophrenia experiencing auditory hallucinations showed decreased responses of the temporal cortex to external auditory stimulation. Therefore, auditory hallucinations in some patients with schizophrenia may correspond with maximal activation of the auditory association cortex. The physiology of thinking in words was assessed utilizing PET in six persons with schizophrenia who experienced coherent hallucinations, six persons with schizophrenia...