Cochlear sound-movement and musical misperception

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Griffiths et al. (1997b) reported a 75-year-old man with a right hemisphere infarction which was supposedly causing misperception of sound-source movement and amusia. This contravenes Morgan’s Canon, that no behaviour should be explained in terms of higher psychological processes until simpler physiological or sensory mechanisms have been excluded.

Griffiths et al. (1997a) described another patient with sound movement detection deficit and a trapezoid body lesion, and wondered if other patients with lesions in the auditory pathways would show similar deficits. In fact, Sir Alexander Ogston in his Cavendish Lecture of 1890 has already done so, showing conclusively that an inflammatory hydrops (his words) of the labyrinth due to pressure changes could produce this and other subtle auditory defects.

Ogston’s main point (Ogston, 1890), as true now as it was then, was that subclinical Menière’s disease was very common but often missed by doctors. Thus, the patient of Griffith et al. (1997a) had problems hearing speech in noise, rotatory vertigo, and dextral headaches with tinnitus, yet there was no mention of any cochlear lesion. Also, from my experience, her pure tone audiogram appeared virtually diagnostic of bilateral hydrops. Despite bemoaning the absence of accurate audiotometry, Ogston nevertheless came up with the identical characteristic pattern of low-tone and high-tone loss with intact mid-tones. He also stressed that hydrops symptoms could vary from hour to hour, so a single audiogram is worse than useless. Both ears often fluctuate independently, so that the worst ear can differ.

The first case of Griffiths et al. (1997b) also had a mild hearing loss similar to hydrops since it extended to the lowest frequencies. This must be a more plausible cause of his unexpected sound-movement defect than any brain lesion. Did their patient have any of the hydrops symptoms listed by Ogston?

Ogston measured the external horizontal and vertical fields of hearing carefully, showing that in hydrops the points of most acute hearing shifted to different parts of the auditory field, giving rise to predictable distortions of sound localization, even in unilateral cases. In one of his patients ‘this difficulty was so decided that when crossing the street, if a carriage chanced to be approaching, he became confused, not knowing from what direction the noise of the wheels was advancing, or whether one or more vehicles were producing it. On entering on one occasion a room where a small clock was concealed, he detected its ticking, but failed to discover whence the sound proceeded.’ The other patient of Griffiths et al. (1997a) had remarkably similar complaints. When standing on a platform, she was unable to tell which way a train was travelling and she could not locate ringing office phones.

As for amusia from brain lesions, I am not aware of a single published case of pure amusia with normal ears, though there are cases of amusia with aphasia. However, there are descriptions of pure amusia in educated observers with Menière’s disease, mostly from the last century. Ogston briefly described two cases of cochlear hydrops with amusia which reversed as diplacusis recovered. A self-taught chorister who had inherited a marked taste for music lost his passionate love of music, and became quite indifferent to it when his attacks began; he played and sang out of tune, ceased to recognize false notes, and could not complete a familiar tune. A lady entirely lost the love of music and her previous skill in it; she disliked playing or hearing music on account of the positive discomfort it caused her, and lost the power of recognizing tunes. The case of Griffiths et al. (1997b) was strikingly similar—a self-taught chorister with a life-long interest in music who lost the ability to recognize tunes. A particular feature was that music no longer brought him any enjoyment, and actually sounded unpleasant. He sang in tune, though he did not think so.

Would Griffiths et al. not agree that Ogston’s explanations for the disordered perception of sound-movement and music are clearer, simpler and more logical and consistent with all that is known about ear and brain function than theirs?

References


Detection of acoustic temporal and spatial features in Menière's disease

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We thank Gordon for pointing out historical anecdotes about the effects of possible Menière's disease in the presumed absence of a central auditory lesion. We were only aware of modern, rigorous psychophysical studies of the detection of spatial features (Hausler et al., 1983) and studies of masking-level difference (Quaranta and Cervellera, 1974; Olsen et al., 1976) in Menière's disease. We entirely reject Gordon's suggestion that the patient (H.V.) we recently described in Brain (Griffiths et al., 1997b) had Menière's disease, and that this was responsible for his symptoms of loss of musical appreciation, or for his deficit in sound movement detection.

H.V. had no symptoms of aural fullness, deafness, tinnitus or vertigo, either fixed or intermittent, when he was assessed, and he had no previous history of such symptoms. The audiogram, showing symmetrical pure tone thresholds within 20 dB of national control data up to 4 kHz, does not support the diagnosis either. We accept that pure tone thresholds can fluctuate in Menière's disease, and would add that sensation levels were set at 500 Hz, the carrier frequency for most of our stimuli, for each of 8 days of psychophysical testing over a period of 9 months. There was no variation in his pure tone threshold at 500 Hz between these sessions. Further, the pattern of his symptoms would not fit with a fluctuating process such as Menière's disease; his symptoms of lack of musical appreciation were fixed, though improving very slowly over the course of his assessment. The two cited historical cases with amusia were symptomatically deaf, which H.V. was not, and improved when their deafness resolved. Finally, we would point out that H.V. had a clear-cut right hemisphere infarction just before the onset of the musical symptoms, which we have argued is the most parsimonious explanation for the deficits observed, and in accord with animal and human lesion data on tone sequencing, and with human functional imaging studies, including our own. The central point of our report was that deficits in processing of complex acoustic stimuli should be considered in terms of end organ dysfunction, central auditory processing and cognitive processing, in that order. The deficit in this case was due to a deficit in central auditory processing.

Gordon also mentions another case we have reported elsewhere with a symptomatic auditory spatial deficit due to a brainstem lesion (Griffiths et al., 1997a). That patient did not have Menière's disease, either, and had a lesion involving the trapezoid body; thus his symptoms and lesion were in accord with established animal physiology and lesion work.

In patients who do have Menière's disease, the presence of temporal and spatial processing deficits is not clearly established, though this has received limited systematic study. We are not aware of any studies of tone sequencing in Menière's disease. With respect to deficits in interaural processing, studies including a total of 29 patients with unilateral Menière's disease (Quaranta and Cervellera, 1974; Olsen et al., 1976) showed a decrease in masking-level difference, reflecting a poorer ability to use interaural phase cues to separate a 500-Hz auditory target from a noise background. That decrease was more marked in subjects showing asymmetries in threshold at 500 Hz of >15 dB. Hausler et al. (1983) describe tests of discrimination of spatial cues and interaural cues in nine subjects with Menière's disease with unilateral hearing losses between 40 and 80 dB. Subjects with Menière's disease showed normal or nearly normal tests of spatial discrimination in the free field using a broad band noise stimulus, and similar performance in discrimination of interaural amplitude or interaural onset time for noise bursts. Performance with 500-Hz tone bursts was poorer, though only a limited number of measurements were performed. Hausler et al. (1983) also investigated subjects with disorders at the central auditory pathway including acoustic neuroma and multiple sclerosis and found that many of them, in contrast to the patients with Menière's disease, showed deficits in free field and interaural discrimination tasks. Moreover, there was a marked difference in symptoms related to spatial processing between subjects with Menière's disease and subjects with more central lesions of the auditory pathway. Subjects with lesions central to the cochlea were more symptomatic, with examples being given of a mother with a Schwannoma who could not tell where her children were, and a nurse with multiple emboli who could not tell which alarm bell was going off.

We suggest that the presence of spatial and other deficits in Menière's disease can only be properly established by rigorous psychophysical testing in subjects in whom central lesions have been excluded by appropriate imaging. We hope that this discussion might stimulate such work.
References


