Congenital Amusia: Myths and Realities of Tone Deafness

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Although music, and music making is a universal human trait, the ability to perceive and perform music varies according to societal practice and cultural norms. In Western music, the scientific concept of perceptual and performance difficulties associated with pitch has been established since the first models of language and cognition were developed in the early 19th century. An early case study from this period describes a severe handicap in pitch perception and performance in a normal 30-year-old-man (Allen, 1878). Researcher Grant Allen coined the term “note-deafness” to describe his subject’s condition. He based this idea upon the notion that note-deafness could be a congenital condition much like the visual abnormality known as color-blindness. From 1888 through 1890, German researcher August Knoblauch described a cognitive model of music processing hypothesizing nine music disorders of production and perception that he termed amusia (Johnson & Graziano, 2003). The idea that otherwise healthy individuals with intact hearing sensitivity and language skills exhibit impaired pitch perceptual skills is commonly known as tone deafness. It is more accurately described as congenital amusia—an innate neurological deficit involving pitch perception and performance.

Since the early models from the late 19th century, time has served to perpetuate the romantic myth of music as talent, reinforcing the idea that tone deafness is a rather ordinary phenomenon. It is not unusual for individuals to claim they possess the inability to enjoy and practice music due to lack of talent, or more specifically tone deafness. A questionnaire distributed to 1,105 university students in 2005 found that 59% claimed they could not imitate melodies by singing (Pfordresher & Brown, 2007). Scientific studies suggest otherwise, estimating that somewhere between 4-5% of the population are afflicted with some form of amusia (Fry, 1948; Kalmus & Fry, 1980). Even these findings are not without controversy.
because the early study by Fry (1948) lacked data analysis and the later study by Kalmus & Fry (1980) used a single measure of musical ability suggesting questionable validity and reliability.

Pfordresher and Brown conducted two experiments in 2005; the first studying individuals with normal hearing, no vocal pathology, and no musical training. The second study sampled randomly regardless of training. Both experiments pre-screened for self-proclaimed poor-pitch singers. Participants received a battery of perceptual and performance-based tests. Although the self-proclaimed poor-pitch singers did not differ from good singers in pitch discrimination accuracy, they appeared to be “hindered rather than helped by singing with correct accompaniment” (Pfordresher & Brown, 2007). Based on a detailed quantitative analysis, Pfordresher & Brown rejected their initial hypothesis that poor pitch singing results from deficits in perception. Instead they surmise that poor-pitch singing results from sensorimotor mistranslation during imitation. In other words, poor-pitch singing may result when auditory representations of pitch are incorrectly mapped onto incorrect motor representations for phonation. Findings also suggest that memory retrieval of more familiar melodies as well as an identifiable comfort pitch prevalent in poor-pitch singers may cause interference in production of the test melody in a new tonal center. Pfordresher and Brown claim that the actual proportion of poor-pitch singers in the population is closer to 15%, diverging from the self-report data of 59% cited earlier. The reality is that even the majority of these poor-pitch singers can perceive music normally. Pfordresher and Brown acknowledge that even the previously cited 4-5% of perceptual-based congenital amusia may be an overestimate.

Until recently, the colloquial notion of tone-deafness has been based primarily upon anecdotal reports. One modern method of studying amusia involves recruiting otherwise normal individuals who describe lifelong music perception difficulties and subjecting them to a battery
of tests to verify their condition. Once confirmed, amusic individuals are paired with a random population sample and given pitch perception tasks. One such study found significant differences \((p<0.01)\) in pitch-difference tasks between the pre-selected participants and control subjects (Foxton, Dean, Gee, Peretz & Griffiths, 2004). This study also suggests that the perception of pitch and the manipulation of pitch are separate entities in which training can improve both perceptual skills and performance skills. Another study involving high school wind instrumentalists found that receiving private instruction significantly \((p<0.01)\) affected their tuning accuracy and ability to perform on the pitch perception tasks (Yarbrough, Morrison & Karrick, 1997). Studies like these confirm the idea of the interrelationship of pitch-perception and performance deficits in congenital amusia, but do little to explain the cause of the condition.

During the past thirty years there has been three important advances in the field of neuroscience and music: a) the application of techniques that demonstrate the functional organization of the normal musical brain including haemodynamic (PET and fMRI) and electrophysical (EEG and MEG) imaging techniques, b) evolution of constructs which have allowed for the systematic evaluation of musical disorders through aphasia batteries such as the Aachen Aphasia Test and the Montreal Battery of Evaluation of Amusia (MBEA), and c) the ability to examine changes in the brain that accompany musical disorders, such as lesions and subtle anatomical changes with greater precision (Stewart, von Kriegstein, Warren & Griffiths, 2006). What makes studying musical responses in the brain so complicated and yet so compelling for researchers is that music is not a localized phenomenon. Unlike speech and sight, music perception and performance relies on a network of various way-stations that sort, process and route information to other areas depending upon the type of activity being performed.
Stewart et al. acknowledge that the problem with assessment of musical disorders lies in the difficulty of defining a normal musical brain.

One difficulty in mapping pitch processing in the normal musical brain is due to its ability to change over time. Longitudinal studies have demonstrated functional brain reorganization after even short periods of musical training (Stewart et al., 2006), although the extent to which the changes endure is uncertain. This is partially due to the difficulty of conducting longitudinal studies in neurologically impaired subjects. Therefore, researchers have come to rely on examining acquired deficits in musical listening in musician brains through clinical case studies. In research settings, the MBEA can be used to document disorders of musical abilities that can occur after brain damage, where ordinary musical skills are sometimes spared. According to its authors, the measure is “sensitive, normally distributed, reliable on the test-retest, and correlates with Gordon’s Musical Aptitude Profile” (Peretz, Champod & Hyde, 2003). The relevance here is that the effect of lesions or similar forms of damage (largely vascular) depends upon premorbid levels of competence and awareness which varies widely within the population. In other words, the value of documenting neurological damage due to hemorrhages or strokes is only relevant to the extent of the individual’s ability to function musically prior to the impairment. Since musical skills vary greatly within the population, the key to mapping the pitch process in the musical brain is to understand how it functions prior to impairment. The fact that the MBEA correlates with Gordon’s Musical Aptitude Profile (MAP) is important because: a) this test (MAP) can be used on musicians and non-musicians alike, b) its use is fairly widespread and c) assuming a pre-trauma MAP score could be located, a regression analysis between MAP scores and the MBEA could be used to measure the correlation between musical aptitude, especially in regards to pitch perception, and neurological impairment.
A limitation involving measuring pitch perception and performance in the musical brain is that researchers rely upon a limited pool of subjects from which to draw; namely neurologically impaired individuals. As mentioned, this makes pre and post hoc testing problematic; no one plans for neurological impairment, thus longitudinal studies are difficult at best. A recent study offers exciting insight to discovering how pitch perception can be altered in otherwise normal individuals. The study involves a 14-year-old Japanese girl with normal growth and several years of piano study that was treated with carbamazepine, a first-line anticonvulsant used to treat epilepsy. Upon the oral administration of the drug she reported that sounds of instruments, the chime in her school, and the telephone ringing seemed to have a lower pitch. She reported feeling strange when playing the piano. Upon cessation of carbamazepine, she no longer complained of abnormal pitch perception (Yoshikawa & Abe, 2003). Other cases of carbamazepine-induced abnormal pitch perception have been documented with similar findings (Wakamoto, Kume & Nakano, 2004). Researchers speculate about the cause of the abnormality with explanations ranging from subtle changes in the organ of Corti to sensory amusia. Yoshikawa and Abe (2003) suggest that since “carbamazepine is a sodium canal blocker, [it] might be…affecting the tiny but sensitive stapedius muscle that determines the tension on the tympanic membrane that mechanically perceives sound waves, thus pitch.” If modern imaging techniques could be utilized before, during and after treatment, neurologists might have a better understanding of how the brain and peripheral auditory system responds to changes induced by carbamazepine. Until such tests are conducted, researchers will remain unsure as to whether the drug affects the peripheral or central auditory nervous system.

Due to musical brain plasticity, it is difficult to predict, track, and map congenital amusia in the brain. This combined with the differences between the musical and non-musical brain
serve to further confound the problem. As research indicates, it is less likely that individuals in the population are symptomatic of the scientific description of congenital amusia. Rather, they may be poor-pitch singers who have not received adequate or appropriate musical training. However, whether through training deficiencies, neurological impairment, or chemical induced abnormal pitch perception, finding and mapping where, when, and how the brain perceives and responds to pitch is important to extinguishing the colloquial understanding of tone-deafness.
References


