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On the biological plausibility of Wind Turbine Syndrome

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An emerging environmental health issue relates to potential ill-effects of wind turbine noise. There have been numerous suggestions that the low-frequency acoustic components in wind turbine signals can cause symptoms associated with vestibular system disorders, namely vertigo, nausea, and nystagmus. This constellation of symptoms has been labeled as Wind Turbine Syndrome, and has been identified in case studies of individuals living close to wind farms. This review discusses whether it is biologically plausible for the turbine noise to stimulate the vestibular parts of the inner ear and, by extension, cause Wind Turbine Syndrome. We consider the sound levels that can activate the semicircular canals or otolith end organs in normal subjects, as well as in those with preexisting conditions known to lower vestibular threshold to sound stimulation.

Keywords: Wind Turbine Syndrome; vestibular function; Tullio phenomenon; superior semi-circular canal dehiscence; infrasound; vestibular evoked myogenic potentials (VEMP)

Introduction

Wind Turbine Syndrome (Pierpont 2009) is a term used to describe a range of vestibular and hearing symptoms that may affect the health of people living near to wind farms. This review examines biological mechanisms that might account for this syndrome.

Worldwide there is a need (perhaps an urgency) to promote wind power electricity generation. For example, wind farms in the USA currently generate over 60,000 MW annually, in China more than 90,000 MW, and in Canada about 8000 MW. Commercial-scale wind turbine facilities are ideally established in low-population areas, but more and more they are being built closer to the urban areas that they serve. In North America, Europe, Australia, and many other countries, a noise-level regulation is typically set such that turbine noise near homes does not exceed 40–50 dBA. Nevertheless, some people local to wind farms complain and there is concern about potential health issues.

There is clear evidence of an annoyance or irritability caused by the acoustic signal from wind turbines (e.g. Persson Waye & Öhrström 2002; Pedersen & Persson Waye 2004) that appears to be greater compared to other equivalent-level environmental noise such as airport or road traffic noise (Janssen et al. 2011). In this regard, wind turbine noise is unique in having low-frequency signal components including infrasound (below 20 Hz). The sounds that are audible have a distinct amplitude modulation component, generally described as a “swish” or “thump”. This rhythmic characteristic makes the

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noise difficult to ignore or to adapt to, and its enhanced perception compared to un-modulated noise appears to contribute to its increased annoyance factor. The general annoyance with wind turbine noise is not under discussion here. Many social, financial, and psychological factors play into the reactions of individuals who are unhappy to live near to wind farms. Biological health issues can arise when the irritability and annoyance leads to sleep disturbance and stress (Persson Waye et al. 2003; Persson Waye 2004; Bakker et al. 2012). These are complex and important issues and but are not dealt with in this discussion

Here the focus is on a more direct influence of wind turbine acoustic signals on inner ear function. There are the audible signals, but much of the energy in wind turbine noise is at very low frequency, outside of the human frequency range and below hearing thresholds, i.e. inaudible. There have been suggestions that both perceived and subliminal signals can activate cochlear and vestibular receptors and lead to hearing or vestibular disorders. One illness has been described by Pierpont (2009) as Wind Turbine Syndrome. Here, symptoms typically associated with vestibular dysfunction (vertigo, nausea, and nystagmus) are reported as well as auditory problems (aural fullness, hyperacusis, and tinnitus). In numerous review studies and expert panel assessments of wind turbine noise, the topic of Wind Turbine Syndrome almost always arises, but has rarely been discussed and assessed in a logical fashion. There are many who will not give credence to this syndrome because the sparseness of evidence. The primary description was presented as a series of case reports, with some peer review but no critical analysis of mechanisms. At the present time, there are many knowledge gaps, and we clearly await further evidence that it is constitutes a serious health issue. This short review will examine evidence for, and knowledge gaps that exist, regarding the biological plausibility of this syndrome.

Whether primarily cochlear or vestibular in origin, attempts to establish feasible mechanisms for wind turbine syndrome highlight a lack of knowledge about the impact of low-frequency acoustic signals on the inner ear. Because of a lack of good scientific evidence (peer-reviewed studies), there is some doubt on the validity of Wind Turbine Syndrome as a disease entity. On the other hand, there are a number of indirectly related experimental studies or clinical reports that hint at biological plausibility. Given the range of distinctly vestibular symptoms associated with wind turbine syndrome, this discussion will focus on the acoustic activation of the vestibular system.

The main question

Is it possible that the unique nature of wind turbine noise can directly stimulate vestibular receptors, and under some circumstances and in some individuals, lead to the reported symptoms of Wind Turbine Syndrome?

We know that high-intensity sound can activate the vestibular system; objective testing of vestibular function using acoustic stimulation is a well-established technique (e.g. Colebatch & Halmagyi 1992; Colebatch et al. 1994; Robertson & Ireland 1995; Todd et al. 2008; Zhang et al. 2012). Vestibular-evoked myogenic potentials (VEMP) are from neck or ocular muscle activity resulting from a vestibular reflex initiated by acoustic stimulation of the ear. Typically, the VEMP test stimulus is an acoustic click, or a low-frequency (500 Hz) tone pulse presented at a level of 110–120 dB SPL.

Animal model research in vestibular science typically uses physical head or body manipulations (angular acceleration; linear displacements) to stimulate the system. Vestibular research does not typically involve acoustic stimulation. However, there are

reports in which acoustic stimulation via bone or air conduction pathways has been employed, and these reports clearly demonstrate that vestibular end organs are activated by sound. For bone-conducted sound, the stimuli required equate to very high-intensity air-conducted sounds. In recent experiments, Curthoys and his team (Curthoys et al. 2006, 2014) recorded from vestibular neurons in Scarpa's ganglion of guinea pig, and reported otolith afferents responding to 500 Hz bone and air-conducted signals. The threshold levels for air-conducted sounds were high – around 120 dB SPL.

Evidence of increased vestibular sensitivity to sound stimuli

From both clinical and animal model studies, we can note that for normal subjects, acoustic signals below 100 dB SPL are unlikely to activate even the most sensitive end organs of the vestibule, the otoliths. However, there are many lines of clinical evidence to show that acoustic signals can activate the vestibular system at lower intensity levels in some pathological conditions. Directly at the heart of this matter is the Tullio phenomenon (1929) i.e. sound induced vestibular effects. This was described 1929 by Pietro Tullio after observing that a fistula in the inner ear will allow fluid vibrations caused by sound to activate the vestibular end organs, and can result in vertigo or abnormal eye movements elicited via the vestibulo-ocular reflex (VOR). This condition can result from lesions or anatomical abnormalities that lower the resistance of fluid movements between the cochlear and the vestibular inner ear compartments (e.g. barotrauma, enlarged vestibular aqueduct, perilymphatic fistula, and oto-syphilis). Perhaps the most important pathology in this regard is superior (semicircular) canal dehiscence syndrome (SCDS) as described initially by Lloyd Minor (Minor et al. 1998; Minor 2000). Here, an observable (by tomography or directly during surgery) fenestration of a semicircular canal is correlated with sound- or pressure-induced vestibular activity. It is assumed that the opening of the inner ear labyrinth (into the cranial space) creates a pressure release-site that, by analogy to the cochlear round window, allows sound-induced cochlear fluid movements to transfer to vestibular parts of the inner ear. To be more explicit, acoustic signals transfer mechanical energy at the stapes footplate to the cochlear fluids. If the inner ear labyrinth were a closed system here would be no movement of (incompressible) cochlear fluids. However, because the cochlear round window acts as a pressure-release valve, fluid movement within the cochlea and haircell activation is possible. In the pathological conditions listed above, particularly SCDS we see how acoustic signals can, under certain circumstances, be more effective in activating vestibular end organs. With a pressure release-site (canal dehiscence) in the vestibular part of the inner ear, mechanical signals originating at the stapes footplate can effectively reach and stimulate vestibular haircells. Is this perhaps how the symptoms of Wind Turbine Syndrome can be realized?

It has been established that the level of sound required to activate the normal human vestibular system is approximately 110 dB SPL (as used in VEMP testing) and is reported at around 120 dB SPL in animal models (Curthoys et al. 2014). The next question is how much lower is the activation threshold in pathological conditions such as those described above, in particular in SCDS? There are a number of studies that have used VEMP testing and compared the sensitivity of the vestibular system to sound in patients with various degrees of canal dehiscence (Pfammatter et al. 2010) or in patients before and after surgery for plugging superior canal dehiscence (Welgampola et al. 2008). These authors report VEMP thresholds in SCDS patients at about 85 dB SPL, a reduction of 20–30 dB compared with normal thresholds. Using a different method of vestibular function monitoring, the click evoked vestibulo-ocular reflex (VOR), Aw and colleagues (2006) reported

on SCDS patients with click-evoked VOR responses 10–40 dB below a normal baseline value of 145 dB SPL. Thus, the sound activation of the vestibular system is significantly enhanced in subjects with SCDS.

The prevalence of individuals with vestibular sensitivity to sound

When SCDS was first described (Minor et al. 1998), it was observed in a very small population of patients with significant sound-induced vestibular symptoms. Because canal dehiscence could be detected on CT images, there were subsequent studies of CT images in normal subjects or patients with unrelated diseases. These studies were important because they showed that canal dehiscence is not so rare. In normal subjects, CT images of the semi-circular canals reveal a surprisingly high prevalence of dehiscence. In adults, Russo and colleagues report 5 % (Russo et al. 2014). Cho et al. (2014) find that 1.2 % of normal adults have definite or suspicious dehiscence. Erdogan and co-workers (2011) also report that 1.2 % of adults have canal dehiscence. Hagiwara and colleagues (2012) report a 3 % occurrence in adults and 27 % in children under 2 years; the latter indicating that perhaps bony development of canals is still incomplete at this age. Furthermore, otologic symptoms of SCDS have been found in many patients with only a thinning of canal bone – described as a "near-dehiscence" (Ward et al. 2013). All of these data indicate that perhaps a relatively large number of adults (and many more infants) may be more susceptible to acoustic activation of the vestibular system than the general population. While most of these individuals may not have vestibular problems, perhaps specific low-frequency components in wind turbine noise can induce vestibular symptoms.

Conclusions

Back to the question: can the acoustic signals from wind turbines activate vestibular end organs and cause symptoms associated with vestibular disease? The normal threshold for acoustic activation of the vestibular system as judged from VEMP testing is around 110 dB SPL. With click-evoked VOR it is higher. Basic animal research indicates that otolith organ stimulation can be achieved with sounds at 120 dB SPL (Curthoys et al. 2014). At the standard 40–50 dBA noise-level regulation commonly in place it is clear that no component of wind turbine noise approaches levels high enough to activate the vestibular system. It has been argued, quite correctly, that "standard" reporting of wind turbine noise levels with an A weighting filter (dBA) underestimates the levels of very low-frequency components. However, the typical spectrum of 45 dBA wind turbine noise does not have low-frequency components that exceed about 60 dB SPL. So, vestibular stimulation in normal adults is very unlikely. Is it possible that in the 1–2 % of "otherwise normal subjects" who have some inner ear pathology such as SCDS and who have a reduced threshold of vestibular system activation can be stimulated by wind turbine noise? Here the gap between low-frequency acoustic signal at 60 dB SPL and the vestibular thresholds of 85 dB SPL (e.g. Welgampola et al. 2008) is closed *but not completely.*

As wind turbine power generation becomes more common, and as more communities are subjected to wind-farm noise, so complaints and reports of adverse health effects will increase. There are many different causes of irritation and annoyance, and in some cases vestibular symptoms are reported. It would be of some interest to look for canal dehiscence in CT images from subjects with Wind Turbine Syndrome. If a correlation

was found it could give some credence to the possibility that wind turbine noise can activate the vestibular organs in some individuals. Not least it could help to separate biological vs. psychological etiology.

In summary, we still have significant knowledge gaps regarding the activation of vestibular system with acoustic signals, particularly low-frequency and infrasound (below 20 Hz) components. If we attempt to explain the vestibular symptoms of Wind Turbine Syndrome within the framework of our present knowledge, we have to conclude that there is no evidence for biological plausibility.

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