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The *scientific* arguments against vibroacoustic disease

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ABSTRACT

For the past 27 years, the multi-disciplinary scientific team, led by author NCB, has been investigating the effects of Infrasound and Low Frequency Noise (ILFN, <500 Hz) on biological tissue, in human and animal models. The clinical pathology caused by excessive exposure to ILFN is called vibroacoustic disease (VAD). Despite the considerable amount of data collected to date, the debilitating effects of VAD are not yet acknowledged by the vast majority of the medical and scientific communities. Consequently, individuals exposed to ILFN are not afforded any protection against this physical agent of disease. This report will deconstruct the most common scientific arguments employed by mainstream scientists against the acknowledgement of VAD as a unique clinical entity. Both the physical understanding of acoustics and the mechanical understanding of biological systems are *sine qua non* conditions for comprehending the interaction of acoustical phenomena with biological tissue. Among several others, common arguments include 1) “what you can’t hear won’t hurt you”; 2) it is not biologically possible to have collagen growth without a concomitant inflammatory process; and 3) no abnormalities are detected with the majority of routine medical diagnostic tests. These issues involve key concepts that are required for understanding VAD as a pathological entity.

1 INTRODUCTION

When scientific experimentation confronts established dogmas, usually the dogmas are able to maintain themselves for a while, until they are no longer borne out through valid, scientific experimentation. Once this occurs, it becomes necessary to meet the challenge head on, and dissect, dogma by dogma, all the erroneous concepts that no longer stand up to scientific scrutiny. Such a juncture has been reached with the results obtained over the past quarter of a century, focusing on the biological response of infrasound and low frequency noise (ILFN, <500 Hz) exposure.

The goal of this report is to deconstruct the arguments used to deny the existence of a whole-body pathology caused by excessive exposure to ILFN, otherwise known as vibroacoustic disease (VAD) (1,2). The ultimate objective is to provide healthcare personnel with the necessary knowledge to better aid VAD patients; to provide researchers with new insight into biological behaviors in the presence of a physical agent such as ILFN; and to provide the general public, and VAD patients in particular, with some explanation of why it has been so difficult for the scientific community to acknowledge VAD.

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2 THE SCIENTIFIC ARGUMENTS - ACOUSTICAL

2.1 “The large wavelength of ILFN makes it impossible to interact with the cell, which is of a much smaller order of magnitude.”

Theoretically, waves interact with objects that are of the same order of magnitude as the wavelength of the wave phenomenon. When a pressure wave impacts on biological tissue, sheets of cells react, analogous to a tent canvas being perturbed by a breeze. Cells act in unison, and react as if they were aerodynamic sheets of viscoelastic material, distributing an external force throughout the extent of the structure. Being within a ILFN environment can be like being in a constant acoustic field.

2.2 “What you can’t hear won’t hurt you”.

There is something unique about acoustical phenomena that distinguishes them from many other physical agents: they must be perceived by the human senses in order to have a deleterious effect on the body. In other words, unless one hears the acoustical event, it will not have any effect upon the individual. Surely this must be a singular case among the History of Medicine. Why it is necessary for the host to perceive the agent of disease in order for it to have a deleterious effect on the host? Translating this curious concept to other agents of disease, this would be the same as stating that if one does not perceive x-rays, then there will be no harm done to the body. The validity of this assertion is nil.

2.3 “The physical agent causing damage is not acoustical, but vibrational.”

In terms of occupational safety and health, when one speaks of the health effects due to exposure to vibration, one is generally referring to the direct contact between the human and the vibrating structure. It is implied that there is a solid-to-solid transmission of vibrational energy. With ILFN, there is no such solid-to-solid transmission of acoustical energy. An airborne pressure wave (pressure waves across fluid mediums are beyond the scope of this report) impacting on viscoelastic and aerodynamic sheets of cells, originates an external perturbation upon the biological structure. The effects of interfacing with vibratory equipment and interfacing with airborne ILFN cannot be expected to be the same. Indeed, chainsaw and pneumatic hammer operators take much longer to develop VAD because the associated acoustical environment is not predominantly rich in ILFN.

On the other hand, there are environments that have large amplitudes of both vibrational and acoustical components, such as helicopters. Solid-to-solid vibration and ILFN have a synergistic effect on biological tissue. Previous studies have shown that rodents exposed to ILFN+vibration demonstrated a larger degree of genotoxicity than rodents exposed to ILFN or vibration alone (3). Moreover, VAD-associated pathology, such as squamous cell carcinoma of the lung (particularly of the right lung) is manifested earlier in helicopter pilots (average age 50 years) than in fixed-wing aircraft pilots (average age 60 years) (4), corroborating the results obtained with ILFN-exposed animal models.

3 THE SCIENTIFIC ARGUMENTS - BIOLOGICAL

3.1 “Pericarditis is not specific to VAD.”

Pericarditis is a condition where the sac that surrounds the heart acquires an inflammation and, consequently, its walls become swollen. Since it surrounds the heart, it must accompany the cardiac rhythm associated with the systolic and diastolic movements. In pericarditis, the increased wall thickness of this sac restricts the expansion (diastole) of the heart, i.e., pericarditis is usually accompanied by diastolic dysfunction. This condition can be diagnosed through echocardiography, where both cardiac structure and function are evaluated.

The issue of pericarditis is raised when speaking of VAD because echocardiography of VAD patients also reveals thickened pericardia (5-7). Indeed, pericardial thickening is the

hallmark of VAD (8). However, in VAD, no diastolic dysfunction exists *despite* the thickened sac. In VAD, pericardial thickening occurs in the *absence* of an inflammatory process. According to medical dogma, it is not possible to have pericardial thickening without a) an inflammatory process and b) diastolic dysfunction.

These arguments were put forth in the early 1990's. In 1996, pericardial fragments of VAD patients' hearts were studied with electron microscopy (9). The mystery of how pericardial thickening could occur in the absence of an inflammatory process, and with no diastolic dysfunction was solved (10-16). In normal pericardia, the wall of the sac is composed of three different layers of tissue, the middle one called fibrosa. In VAD, the fibrosa layer thickens due to the growth of collagen and elastin; it splits into two and, in between, there is the neo-formation of a (previously inexistent) layer of loose tissue. This new structure, formed by 2 collagen- and elastin-thickened fibrosa layers, sandwiching a layer of loose tissue (containing blood and lymphatic vessels and adipose tissue), provide the means for new types of pericardial dynamics. Through sliding movements, this pneumatic-like structure allows the pericardium to follow the cardiac rhythm, supplying sufficient compliance (increase of elastic fibers) and rigidity (increase in collagen) in order to accommodate a normal diastole.

3.2 “Collagen proliferation without an associated inflammatory process is not possible.”

This statement comes directly out of medical school dogma, and can no longer be upheld by modern scientific knowledge. Collagen proliferation in VAD features a unique trait: fibers are not arranged in anarchical manner, as in wound-healing. Instead, they are well organized and, together with elastin fibers, form functional units that are vital for maintaining the integrity of the cardiac rhythm, particularly of the diastolic function (10-16).

The astounding fact is that organized collagen proliferation, interlaced with elastin fibers, can be seen in numerous biological structures exposed to ILFN, such as human lung and pleura (17), trachea (18), and blood vessels (14,15,19), as well as rodent trachea (20,21), lung and pleura (20,22), blood vessel walls (20,23) lymphatic vessel walls (24), **and parotid (REF)**. In all these studies, an associated inflammatory process was never identified.

3.3 “There are no large-scale epidemiological studies demonstrating the existence of VAD.”

This *cannot* be surprising. ILFN is not a “high-priority topic” for most institutions that fund research studies. Useful large-scale epidemiological studies can not be conducted without a significant amount of funding to cover, at least, acoustical measurements and VAD diagnostic tests. Moreover, detailed interviews (or extensive questionnaires) must be carried out to determine personal ILFN exposure histories.

3.3.1 Control Populations

Selection of control populations can be tricky and, if incorrectly executed, can lead to a wasteful use of resources, as has happen in the past (25). The effects of ILFN on biological tissue are cumulative, and depend simultaneously on the frequency and amplitude of the acoustical events. Biological tissue does not discriminate between occupational, environmental or recreational ILFN exposures. Moreover, since there is no legislation effectively curbing the production of ILFN, it is allowed to proliferate throughout many activities of human society. The complexity of this situation has dire implications for selection control populations in ILFN-related studies.

An Example: The goal is to establish a difference between workers occupationally-exposed to ILFN (e.g. flight attendants) and workers who are not occupationally-exposed to ILFN (e.g. office workers). Other ILFN-exposures of elements in either group must be taken into account otherwise the following possibilities can occur: a second job with ILFN-rich environments (e.g. dance-club disk-jockeys, security or bartending); ILFN in the homes (e.g.

large volume highways, neighboring ventilation systems); ILFN exposure during fetal gestation or childhood (e.g. ILFN-exposed working mother or in-home ILFN); or even, significant ILFN exposure of office workers (e.g. in dense urban centers). These are just some of the numerous situations that can skew, and effectively invalidate, the data of any ILFN-related epidemiological study, if they are not taken into account beforehand.

3.3.2 Survivorship Bias

Another concern is the possibility of comparing older individuals, who have already survived extensive ILFN exposure, with members of the general population. Metaphorically, it would be like comparing trained, experienced and skilled soldiers with the average John Doe. Such is the case when one compares a group of retired ILFN-exposed professionals with a similar group from the general population. This group of retired ILFN-exposed individuals consists of ones who *survived* until retirement. Drawing conclusions from studies with this type of design could lead to misleading inferences.

3.4 “Some people become more annoyed with noise because they are more sensitive.”

Annoyance is an interesting parameter that should be given the import of any other subjective measure. It is a measure of how irritated individuals can get when exposed to noise. Annoyance levels are now part of most European legislations. They are established though the analysis of a 1/3 octave band frequency distribution, obtained in dBA. If any band exists that is 5 dB larger than the adjacent bands (simultaneously on either side) then annoyance levels are exceeded. The rationale is that larger levels of annoyance lead to higher levels of stress, and this leads to a higher risk for cardiovascular disease.

Within the context of VAD, patients complain of “hearing too much”, even though many have some degree of classical hearing loss (1,2). They claim they “any noise” bother them, “even music”. Indeed, while the classical hearing impaired individual will turn up the volume of the television set in order to hear better, the VAD patient turns it down because he/she “cannot stand noise”. For VAD researchers, these complaints constitute a clinical sign indicating a high probability that the individual has already been exposed to excessive ILFN.

In the cochlea of ILFN-exposed rodents, actin-based stereocilia were found fused amongst themselves and with the upper tectorial membrane (20,26-28). Unlike the age-matched controls, ILFN-exposed rodents did not lose stereocilia with the natural aging process. If fused among themselves and to the tectorial membrane, cilia cannot freely vibrate as is intended when the sound pressure wave is transduced within the cochlea. In fact, by becoming a rigid structure, any attempt at vibrating them might, understandably, produce discomfort. How closely related this phenomenon is to the concept of annoyance is still unclear, however a relationship is clearly suggested, especially since annoyance has already been specifically associated with the presence of LFN (29).

3.5 “There is no evidence linking ILFN exposure to respiratory cancers.”

The genotoxicity of ILFN has already been demonstrated. Among aircraft technicians (30) and military pilots (31), there was a statistically significant increase in the frequency of sister chromatid exchanges, after adjusting for age and other known carcinogens, such as tobacco smoking. ILFN-exposed rodents also revealed an increase of the frequency of sister chromatid exchanges (3). To date, 100% of the respiratory cancers in VAD patients have been squamous cell carcinomas: 8 in the lung and 2 in the glottis (4). In these 10 cases, 3 were non-smokers (2 lung and 1 glottis). All cases of lung tumors were located in the upper right lobe. In ILFN-exposed rodents, electron microscopy images of the tracheal epithelium revealed dysplasia and metaplasia (20,21). These are pre-tumoral lesions that, with time, lead to squamous cell carcinomas.

4 DISCUSSION

On March 8th, 2007, the Portuguese Ministry of Labor, through its National Center for Occupational Diseases, attributed 100% professional disability to a 40-year-old flight attendant for having acquired VAD on-the-job, diagnosed in 2001. Two other VAD patients were also attributed the same degree of disability. The International Federation of Airline Pilots Associations (IFLAPA) has included VAD in its Human Performance Committee 2007 Work Programme. It would seem to be a matter of time before ILFN becomes acknowledged as an agent of disease since VAD, its ensuing pathology, is already being recognized.

The arguments discussed in the above sections have been raised many times over the past quarter of a century. It is hoped that this report will put some of these questions to rest. New scientific data that go against established concepts are often slow to enter mainstream science. However, some of these arguments have been ongoing for the past 25 years. When the health and well-being of countless individuals, including children, depends on new paradigm shifts, perhaps these ought to be made.

Not all resistance against the acknowledgement of VAD is scientific. There are other sectors of society that oppose the establishment of ILFN as an agent of disease. While these are a bit beyond the scope of this report, they are important to mention, because ignoring them will merely perpetuate the attitude of “ostrich with its head buried in the sand”.

Economical considerations are, obviously, at the forefront of many concerns. The asbestos “problem” is still too recent, and insurance companies are still reeling from other worldwide events, such as increased frequency of natural disasters and acts of terrorism. Energy companies investing in wind energy are facing serious problems when deciding where to place the visually obtrusive windmills, which are also significant sources of ILFN (32). Industry has the double problem of reducing ILFN emissions, a technical feat all on its own, and simultaneously dealing with (past and present) worker exposure to ILFN.

“Who am I going to sue over this?” is probably one of the most feared questions. “The physician who misdiagnosed me? Or the employer who exposed me? Or the energy company that put ILFN in my home?” These are the entities to which people will look toward when ILFN is acknowledged as an agent of disease. On the other hand, several sectors may actually benefit financially from the acknowledgement of ILFN as an agent of disease... Perhaps an (utopian?) agreement could be reached whereby no one could sue over having contracted VAD but, “in return”, all healthcare costs would not be supported by the individual.

5 CONCLUSIONS

Shifting the way in which one has been thought to think is often a difficult task. In science, when old dogmas are proven incomplete or untrue by repeated and consistent, robust and valid scientific data, then true scientists *must* consider the new implications - especially if human health is the issue.

ILFN is an agent of disease and it is the etiological factor for VAD.

The authors invite fellow, intellectually honest scientists to design the necessary experiments to disprove this assertion.

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