Clinical Protocol for Evaluating Pathology Induced by Low Frequency Noise Exposure

Nuno A. A. Castelo Branco, M.D.
Senior Surgical Pathologist, R. Prof. Dias Amado, No. 5-1B, 1600-612 Lisbon, Portugal

Mariana Alves-Pereira, Ph.D.
Biomedical Engineer, Lusófona University, Campo Grande 376, 1749-024 Lisbon, Portugal

Augusto Martinho Pimenta, M.D.
Senior Neurologist, Julio de Matos Hospital, Av. do Brasil, 53, 1700-063, Lisbon, Portugal

José Reis Ferreira, M.D.
Senior Pneumologist, Clínica Doentes Pulmonares, Campo Grande, 4, 1700-092, Lisbon, Portugal

Summary
Segments of the general population who complain about infrasound & low frequency noise (ILFN) in their homes or in their workplaces continue to increase. These individuals often complain about similar sets of concurrent symptoms, and frequently attribute their ailments directly to ILFN exposure. Oftentimes, however, routine clinical evaluations of these individuals reveal no apparent dysfunction, and patients with persistent complaints are subsequently referred to psychology or psychiatry health professionals. The goal herein is to present an objective clinical protocol that scientifically evaluates these complaints, leading to the elimination of malingerers, and to the proper medical assistance of those developing ILFN-induced lesions.

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Introduction

Since 1980, this team of scientists has studied the biological response to Infrasound & Low Frequency Noise (ILFN) exposure in both human populations [1-3] and laboratory animal models [4,5]. Over the decades, a valid clinical protocol has been established in order to ascertain a differential diagnosis for ILFN-exposed persons. The goal of this report is to list the medical diagnostic complementary tests relevant for evaluating ILFN-induced pathology. The rationale justifying each diagnostic test included in this ILFN clinical protocol will be but briefly described; extensive details can be found in the references provided.

2. Vibroacoustic Disease

The nosological entity triggered by excessive exposure to ILFN has been termed vibroacoustic disease (VAD) [1-3]. Despite the overwhelming antagonism with this nomenclature [6, for example], this time these authors have opted to use this specific name rather than "ILFN-induced pathology." VAD is a whole-body pathology characterized by proliferation of extra-cellular matrices (collagen and elastin) in the absence of an inflammatory process. VAD is a consequence of the disruption and restructuring of tissue and cellular components in order to maintain functional structural integrity in the presence of a mechanical agent of disease [further refs in 1]. The onset of VAD is insidious and frequently misdiagnosed. Individual ILFN exposure patterns dictate the time-evolution and ultimate severity of lesions, i.e., exposure times vs. recovery periods is of crucial importance, both for a prognostic view and for an appropriately detailed clinical history of ILFN exposure. Generally, ILFN exposure can be occupational or residential. With occupational exposures, recovery periods are usually certain, given the scheduled end of the workshift and the existence of weekends. Residential exposures, however, do not enjoy this type of recovery periods. The onset of VAD among individuals who are exposed to ILFN in their homes has been observed to be more rapid than those who are exposed to occupational ILFN. Over the past decades, there has been much controversy regarding several issues related to human health

and noise exposure, such as, a) the role and range of the human auditory system in ILFN-rich environments; b) the physiological pathways responsible for inadequate quality of sleep, for hypersensibility and intolerance to sound, and for cardiovascular disease; and c) the parameters used for measuring ILFN, and for assessing health impacts on ILFN-exposed populations. These issues have been extensively addressed by this team of researchers, and are currently beyond the scope of this report. The clinical protocol herein defined will provide family physicians and epidemiologists with objective, useful and clinically valid data that could ensure appropriate medical assistance to potential VAD patients.

3. Clinical Protocol for ILFN Exposures

3.1. Fundamentals

Auditory effects of ILFN exposure differ from the classical picture of hearing loss due to acoustic trauma. In VAD, patients report of *hearing too much*, and commonly lower the volume of audio devices (as opposed to increasing the volume to hear better). Whether in occupational or environmental ILFN exposures, early symptoms frequently include mood disorders and sleep disturbances. As overall exposure time accumulates, physicians are often confronted with a myriad of complaints referring to a wide variety of organs and systems. If unfamiliar with VAD, extensive complementary diagnostic tests will be prescribed to confirm or exclude a clinical suspicion. Most of these medical examinations will disclose negative or borderline values in ILFN-exposed patients, i.e. useless for a diagnosis. Generally, at this point, the clinician refers the patient to psychiatric care, with the likely suspicion of malingering or hypochondria. If, however, the physician suspects that ILFN may be playing a role in the patient's condition, the following complementary diagnostic tests are recommended for confirmation or exclusion of that hypothesis.

3.2. Echocardiogram

Thickening of the parietal pericardium was first observed in autopsy [7], then studied through echocardiography [8-10], and then confirmed through surgical pathology [11,12]. Pericardial thickening in the absence of diastolic dysfunction, and in the absence of an inflammatory process, is a hallmark response to ILFN exposure in humans. In VAD, pericardial echogenicity is visible with a GAIN setting of <45. See Fig. 1.

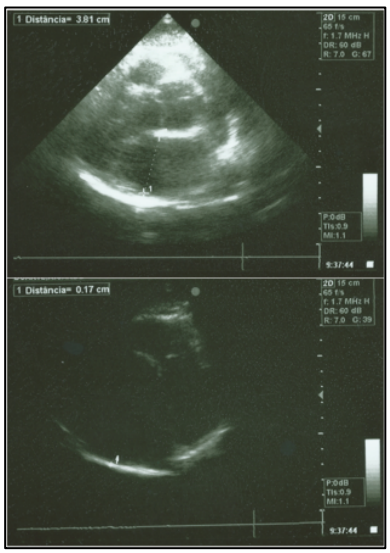

Figure 1 shows echocardiograms of Mr. R [13-15]. *Top image*: Gain set at 67. *Bottom image*: Gain set at 39.

Valve pathology may also be present, particularly in the more severe cases [8-10]. When thickened pericardia are observed through echocardiography, and no accompanying diastolic dysfunction exists, this indicates that significant ILFN exposure has taken place. Echo-imaging alone, however, is insufficient to establish a full VAD diagnosis. Firstly, because this organic manifestation is a *sign* of ILFN exposure, and not a nosological entity in itself. Secondly, no standardized methodology exists to numerically evaluate pericardial thickness. Hence, an undesirable element of subjectivity is introduced with this diagnostic test, making it susceptible to erroneous interpretations [16, for example] and/or deliberate manipulations. With the introduction of GAIN considerations, however, the degree of subjectivity of this imaging technique can be drastically reduced. Echocardiography is, therefore, the diagnostic method of choice for exposure confirmation and patient selection.

3.3. Brain MRI

This imaging test includes the evaluation of structural changes in brain tissues. Albeit non-specific to VAD, brain tissue changes in ILFN-exposed patients include: a) hyperintense foci in T2 of the subcortical and periventricular white

matter, basal ganglia and brain stem, and b) cerebral atrophy and dilation of the perivascular Virchow-Robin spaces. The most frequent locations for these appearances are the sublenticular and periatrial areas, and semioval centers [17]. While these situations may be expected in older populations, they are not desirable among working populations within the 35-55 year age range. VAD patients often exhibit low tolerance to this examination, and frequently remain bed-ridden for the rest of the day.

3.4. P300 Event Related Evoked Potentials

This non-invasive and objective neurological test evaluates nerve conduction times of processes that occur in the cerebral cortex. The P3 and N2 components of Event Related Potentials (ERP) are related to decision processing and stimuli classification, respectively, and increased latencies in these components have been associated with cognitive deterioration [18]. Longer latencies and lower amplitudes of ERP are observed in ILFN-exposed individuals [17]. VAD patients who present with the above described brain MRI lesions, also exhibit significant latencies of the endogenous N2 component, and significantly decreased amplitudes of the P3 component [17]. The presence of cortical lesions, confirmed through brain MRI, is associated with the changes seen in the P300 ERP values. Frontal topography and multipeaked appearances, as observed in these ILFN-exposed individuals, are similar to those found in the elderly and in patients with degenerative processes of the brain.

3.5. Brainstem Auditory Evoked Potentials

This is another non-invasive and objective neurological test. Here, nerve conduction times of processes occurring within the brainstem are evaluated. These can be altered due to demyelinization foci or expansive lesions. In ILFN-exposed individuals, brainstem auditory evoked potentials (BAEP) exhibit increased latencies in wave intervals III, IV and V [19]. Increased BAEP wave V interval latencies were correlated with hyperintense foci in T2 observed in the brainstem through MRI. VAD patients often develop balance disturbances, and these were associated with the existence of asymmetric values for the BAEP wave V interval latency, in both ears [20]. BAEP results suggest that some central dysfunction is occurring at the level of the brainstem. This is further corroborated by the results obtained in ILFN-exposed individuals through the next diagnostic test in this discussion.

3.6. PCO_2 respiratory drive evaluation

This evaluation is performed within the context of lung function tests. The neurological centers of the control of breathing are located in the brainstem. This control system modulates respiratory rate and respiratory (either inspiratory or expiratory) pressure depending on CO_2 concentration: as CO_2 concentration increases, so does the neurologically-controlled respiratory rate and respiratory pressure drive. In VAD patients, this increased respiratory rate and drive in the presence of increased CO_2 is only mildly observed [21]. The significance of this impaired partially autonomic reflex among ILFN-exposed individuals is not yet clearly understood, and raises many (more) questions regarding the pathophysiological mechanisms of this agent of disease. Nevertheless, it further corroborates the existence of brainstem lesions in ILFN-exposed humans. In respiratory functional tests, changes of metacholine sensitivity is common in VAD patients, probably related to the cellular changes in epithelial bronchial cells which present cellular cholinergic degranulation processes [1,4,22].

3.7. Bronchoscopy

This is a highly invasive examination, and is only recommended for forensic purposes within the context of legal proceedings. In VAD patients, vascular-like lesions are observed in both tracheal and bronchial trees, uniformly distributed bilaterally near the spurs [23]. Biopsies of these lesions were taken and studied with light and electron microscopy, revealing the same features observed before in ILFN-exposed human and animal samples, namely, organized proliferation of collagen and elastin in the absence of an inflammatory process [1,2]. Respiratory diseases (specifically, asthma-like conditions, and squamous cell carcinomas, particularly of the right lung) that develop among noise-exposed populations should be carefully considered in light of the morphological impact that ILFN has on respiratory tract structures [22].

3.8. Voice Acoustic Analysis

In a more recent area of study for VAD researchers, this non-invasive test evaluates changes in voice production as a consequence of

physiological changes of the laryngeal anatomical structure system. Morphological changes of the respiratory tract structures, such as those seen in ILFN-exposed human and animal models, can alter several parameters associated with voice acoustics. The fundamental frequency among three vowels significantly increases with increasing ILFN exposure time [24,25]. Changes in other voice acoustic parameters, such as jitter and shimmer perturbation measures, harmonic-to-noise ratio, and maximal phonational frequency range, also exhibited changes but these are not yet fully understood. Bronchoscopy imaging of the vocal folds revealed the same, above-mentioned, vascular-like lesions as observed in the tracheal and bronchial trees [23]. This non-invasive, voice acoustic evaluation that seems to reflect a dose-response pattern for VAD patients is poised to become an invaluable complementary diagnostic tool for ILFN-induced pathology

3.9. Hemostasis and Coagulation Parameters

In extreme stress environments, states of elevated hypercoagulability have been documented [26,27]. In ILFN-exposed individuals, spontaneous platelet aggregation was observed in the most severe cases while platelet aggregation values are abnormally high in all VAD patients. In ILFN-exposed persons the plasminogen activator inhibitor-1 (PAI-1) is significantly increased, even after several days post-ILFN exposure [27]. Increased PAI-1 is an indicator of fibronolysis inhibition and activation of coagulation, leading to situations of hypercoagulability.

3.10. Immunological Parameters

Autoimmune disorders, particularly collagenous diseases, are common among the more extensively ILFN-exposed individuals [1,9]. Lupus-prone animal models exposed to ILFN saw an earlier onset and higher mortality rate than the non-noise-exposed control group [28]. ILFN-exposed Wistar rats saw the pleural immune mechanisms highly impaired, when compared to non-exposed controls [5]. In ILFN-exposed animals, splenic $CD4^+$, $CD8^+$, and IgM^+B lymphocyte populations were decreased when compared to non-exposed controls [29]. In VAD patients, the number of circulating $CD8^+$ and $CD4^+$ T lymphocytes was significantly increased when compared to non-noise-exposed controls [30]. Pericardial fragments of VAD patients observed through electron microscopy revealed remarkable amounts of cellular debris due to non-programmed (non-apoptotic) cellular death [1,11,12]. It is, therefore, unsurprising that VAD patients often develop lupus, vitiligo, and other autoimmune disorders [1]. Hence, complementary examinations should include assays for antinuclear and anti-mitochondrial antibodies, C-reactive protein, and systemic lupus erythematous.

3.11. Exposure Histories

To assess the probability of a patient's symptomatology being associated with excessive ILFN, a comprehensive noise exposure history must be taken. Not only will this aid diagnosis, but it will also provide valuable prognostic information. Noise exposure histories begin with fetal exposure that will depend on the mother's profession and residential conditions. Residential exposures during childhood are of fundamental importance given the cellular processes that occur only during this time of human physical and emotional growth. The relative position of bedrooms in relation to noisy streets, potentially concurrent with occupational exposures, and a variety of different types of recreational noise exposures, must all be taken into account in order to obtain an accurate clinical picture of the patient. The development of ailments in the individual's history should be viewed in light of the chronological exposure to noise. Each type of individual noise source also provides key information for ascertaining the extent of the risk factors for VAD to which the individual has been exposed. Documentation of the time spent away from the ILFN-rich environments (i.e., recovery periods) is crucial for characterizing the evolution of VAD.

4. Health Impact of ILFN Exposure

Over the past decade, the health impact of ILFN exposure has taken on a new life due to the worldwide energy crisis and the urgent implementation of numerous industrial wind turbines (IWT), as decreed by many governments. Since little regard was initially given to the ILFN generated by these devices, it was not long after their installation that families living in their vicinity began having complaints. Recognizing the problematic situation, several agencies and institutions worldwide began conducting studies and publishing papers with titles suggesting that health impacts were being evaluated among these residential populations. However, upon a closer examination, significant design flaws rendered

most of these studies useless [31, for example]. While this may not have been a concerted effort, it is however a consequence of the perpetuation of an historical fact: acousticians, usually with no medical background, are generally greatly involved in noise-impact study designs. Therefore, information on the "health impacts," as rudimentarily obtained through questionnaires or interviews, is often deemed sufficient in order to establish whether or not ILFN is, *de facto*, having an impact on the health of human populations. Objective clinical evaluations of families and animals living in the vicinity of IWT are not commonly performed. ILFN is a physical agent of disease, and infrasound (<20 Hz) is internationally classified as *non-ionizing radiation*. The type of toxicological effect and the evolution and onset of disease caused by ILFN exposure is partially analogous to that caused by radiation. As with radiation exposure, a) the onset of ILFN-induced disease greatly depends on time-exposure patterns; b) different wavelengths of the physical agent affect different organs and tissues; c) the individual need not perceive the agent of disease for a pathological effect to occur within the body; and d) prior exposure histories are a determining factor of key importance. These facts *must* condition the design of studies purporting to evaluate the health impacts of residential ILFN exposure; but currently, they do not. It is recognized that there is an added cost to conducting real clinical studies among ILFN-exposed populations. The next obvious question, though, is: what is the added cost associated with continuing with the *status quo*. While this issue is way beyond the scope of this paper, it is nevertheless pertinent to recall episodes in humanity's recent history, having to do with asbestos, smoking and pesticides.

5. Looking Forward

In the interest of becoming a responsible and mature human society, activities that are deleterious to human health and wellbeing should not be hidden, obfuscated, or otherwise camouflaged. History unequivocally demonstrates the benefits of dealing outright with any potential human-health issue. It is recognized that the production of electrical energy has become the warp and woof of modern societies, and hence the urgency to procure new ways of harvesting energy that can be rapidly (and inexpensively) transformed into electrical energy. There need not be, however, such antagonistic, acrimonious and spiteful endeavors between ILFN disturbed families and *industry*, be it in of the energy, transportation, military, entertainment or manufacturing sectors of society. Having contextualized the issue, it is therefore of paramount importance that the clinical parameters used to evaluate the specific type of pathology caused by excessive ILFN exposure be the most appropriate for the job. Audiometry, electrocardiography, cortisol levels, and number of nighttime arousals are remarkably imprecise measures to clinically evaluate the adverse health impacts of noise exposure. Whether transverse or longitudinal epidemiological studies are planned, the clinical parameters required to ascertain the extent of ILFN-induced pathology have herein been outlined. It is hoped that this report will contribute to a symbiotic relationship between the ILFN-generating industries and world citizens.

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