Echocardiography in vibroacoustic disease

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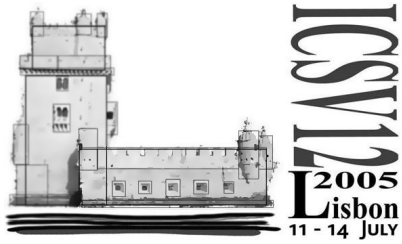

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ECHOCARDIOGRAPHY IN VIBROACOUSTIC DISEASE

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Abstract

Introduction. In 1980, research into the effects of low frequency noise (LFN, <500 Hz, including infrasound) -induced pathology began to be studied among a group of aircraft technicians. Vibroacoustic disease (VAD) has since been defined as the whole-body pathology caused by excessive exposure to LFN. In 1987, the first autopsy of a VAD patient was performed. Among the plethora of findings that laid the foundation of many concurrent avenues of research, was the enormous thickening of the pericardium (a thin sac, <0.5mm thickness, that surrounds the heart). Based on this finding, an echocardiography program was set up among an initial group of 10 technicians. This report exposes the chronological findings of echocardiography in VAD patients. Methods. The results of echocardiograms performed among aircraft technicians, commercial airline pilots and flight attendants, and populations exposed to environmental LFN are gathered and collectively discussed. Results. Echocardiograms of VAD patients disclose pericardial thickening that may appear concurrently with valve thickening, but with no consequences for diastolic mechanics. The degree of severity of VAD is related to the amount of thickening seen through echocardiography, which, in turn, is related to the cumulative amount of LFN-exposure the individual has had. Discussion. Echocardiography is the diagnostic tool of choice for VAD. The pericardial thickening seen through echo-imaging has been confirmed with histological and electron microscopy imaging and is the object of an independent study. Pericardial thickening due to excessive LFN exposure is not exclusive to LFN-exposed workers but is seen even in children who live in LFN environments. Children with *in utero* exposure to LFN noise have been the most severe cases. Despite the validity of pericardial echo-imaging for VAD diagnosis, a common protocol among mainstream science has yet to established.

INTRODUCTION

In 1987, the investigation developed by this team into the pathology developed by workers exposed to low frequency noise (LFN, ≤ 500 Hz, including infrasound) had already included neurophysiological and neuropsychological tests, brain MRI and several biochemical studies (1-3)[1]. The neurovascular nature of this pathology had already been identified, and the respiratory pathology developed by these individuals was still of unknown aetiology.

Five years before, in 1983, one of the workers exhibiting LFN-induced pathology died, and the family withheld authorization for an autopsy. Mr. A, aware of this case, became aggravated by the loss of opportunity to clarify the clinical situation through autopsy examination. To insure that this would not happen in the event of *his* death, he made a will requesting that our team perform an autopsy, no matter what the apparent cause of death. In September of 1987, early in the morning, author Castelo Branco received a telephone call from Mr. A, saying he was unwell and had already called an ambulance. He was certain he was going to die, and requested that Castelo Branco meet him at the hospital to perform the autopsy. Upon arrival at the hospital, 30 minutes later, Mr. A was already deceased. In accordance with his will, Castelo Branco personally performed the autopsy. The findings so graciously bequeathed to this team by Mr. A are, still today, a source of tremendous importance and have been decisive for determining the direction of the research studies into LFN-induced pathology (4).

One of the many autopsy findings was a grossly thickened pericardium (2.8 mm), which appeared globular, tense and pearl-coloured. The heart disclosed endocardial thickening, and thickened valves, especially the mitral valve, which seemed swollen. Coronary artery walls were very thickened causing decreased lumen. Instead of the classical atherosclerotic lesions, a continuous thickening of the intima lined the vessels along their length. Microscopic examinations revealed intimal thickening in the cerebral arteries, but not as intense as in the extra-cranial regions. Pericardium and cardiac valves demonstrated intense fibrosis, and so did the intima of blood vessel walls (4). Based on this case, echocardiography studies of workers exhibiting LFN-induced pathology seemed to be the logical next step.

The goal of this report is to give a chronological account of the findings obtained in 7 echocardiography studies, conducted from 1989 until 2004, in order to demonstrate the usefulness of echo-imaging in the diagnosis of vibroacoustic disease (VAD), the systemic, whole-body pathology caused by excessive exposure to LFN (5).

METHODS

The results obtained in echocardiography studies of workers exhibiting LFN-induced pathology is chronologically reported, based on previously published material (see below). All echocardiograms were performed with a HP 1500 SONOS, and analyses

[1] See “Haemostasis and coagulation changes in vibroacoustic disease,” and “Neurological disorders in vibroacoustic disease I, -III,” included in these Proceedings.

were made in 2-D, M mode, color and spectral Doppler, except for the 2001-1 Study, which used a Agilent Sonnos 1800.

Evaluated parameters varied in each study, in accordance to build-up of information. In the 1989 Study, mitral and pericardial valve thickening while in the 1999 Study the following parameters were assessed: thickening of mitral valve, tricuspid valve, pulmonary valve, aortic valve, endocardium, and pericardium; mitral valve regurgitation, prolapse, and ruptured chordae tendinae; flow velocity A and E, and the E/A ratio. Applicable parameters were evaluated using a seven-grade score system from 0 to 3 points (0, 0.5, 1, 1.5, 2, 2.5, 3): 0 points for no thickening (regurgitation or prolapse) 3 points for maximum thickening (or severe regurgitation or prolapse). Statistical analysis was performed using SPSS package, and statistical significance was established as follows: not significant if $p < 0.01$, significant if $p < 0.001$, and highly significant if $p < 0.0001$. Beginning with the 1999 Study (see below), all echocardiograms were recorded on VHS videotape, to be later evaluated by independent blind observers. All echo-images (from 1989-2004) were captured by the same team of cardiologists, except for the 2001-1 Study. At no time was thickening associated to pericarditis, bacterial endocarditis, or rheumatic fever.

RESULTS

1989 Study

The study group consisted of 25 aircraft technicians (average age: 43 years, range: 33-57) with an average exposure time of 24 years (range: 5-34) and exhibiting LFN-induced pathology. Table 1 summarizes the echo-imaging findings. In the 8 individuals with very thickened mitral valve, 5 disclosed a small anterior leaflet. Other features, such as, intrauricle septal thickening and myocardial echo-structure, were frequent but difficult to quantify. The hepatic Glissom capsule appeared thickened in all subjects, and liver echo-structure appeared altered. Pericardial thickness values were the most surprising, with 20 patients showing echo-dense pericardia > 6 mm thick (as measured through echo-imaging). There was no correlation with 11 cases of previously diagnosed arterial hypertension (clinical information). All correlations with other LFN-induced pathology were negative, e.g., MRI brain lesions, retinal vascular lesions, hemostasis and coagulation changes. Additionally, no correlation was found with overall exposure time or with age. Concluding remarks include the possibility of future studies that might explain why neither diastolic nor systolic dysfunction was observed (6).

Table 1 – Summary of first echo-imaging results by number of cases.

	Normal	Thickened	Very Thickened
Aortic Valve	8	15*	2**
Mitral Valve	0	8*	17**
Tricuspid Valve	10	15*	
Aortic Wall	5	17*	3**
Endocardium	1	14	10
Pericardium	0	1*	24**

** Indicates a statistically significant difference (p<0.001)*

*** Indicates a highly significant statistical difference (p<0.0001)*

1991 Study

This study focused on 56 men (average age: 39, range: 20-57), occupationally-exposed to LFN for 1 to 35 years (average: 20) and exhibiting LFN-induced pathology. Controls consisted of 10 men (average age 38 years, range: 20-60) who had never been employed within LFN-rich environments. In the LFN-exposed workers, cavity dimensions, wall thickness and left ventricular systolic function were normal. However, the aortic, mitral and tricuspid valves were statistically significantly thicker than in controls. Pericardial thickening was present in all LFN-exposed subjects, but not in controls. Pulsed wave Doppler analysis did not disclose any E/A ratio abnormalities. (7)

1996 Study

Larger populations and exposed to different types of occupational LFN-rich environments were studied. Group 1 consisted of 134 workers (average age: 42$\pm$7 years) exhibiting LFN-induced pathology, Group 2 consisted of 20 helicopter pilots (≥2500 hours of logged flight time), and Group 3 (controls) was composed of 30, age-matched male Caucasians, who had never been employed in LFN-rich environments. All subjects were subjected to exclusion criteria that included: streptococcal infections (by history), diabetes mellitus, pre-existing cardiovascular disease, tobacco abuse (>20 cig/day), alcohol abuse (>1 liter wine/day, 10-12% alcohol content), drug abuse (recreational or psychotropic). Echocardiography revealed thickening of some cardiac structure in 100% of the study population: thickening of the pericardium (N=130, 97%; 48 cases with score 2), mitral valve (N=96, 71%), and aortic wall (N=94, 70%). Diastolic dysfunction was identified in 41 patients (31%), 9% with A>E and 21% with A=E, all over the age of 50. Audiograms were also obtained in Groups 1 and 3. Within the control population, at the 4000 Hz notch, 4 had minor hearing losses, of which 3 exhibited discrete pericardial thickening. In Group 1, 43% (N=58) had a trivial auditory deficit (<15 dB) while 5 individuals (3%) exhibited significant losses at the 4000 Hz notch (>50 dB). The remaining Group 1 population exhibited significant losses (>15 dB) but not at the 4000 Hz notch. For the 134 LFN-exposed workers, the summation of the auditory deficit at all frequencies was independent of age (F=3.46) but dependent on exposure time, in years (F=4.47). (8)

1999 Study

Three double-blinded teams of cardiologists (Portuguese, Polish, Russian) examined 485 echocardiograms of workers (recorded on VHS) exposed to three different levels of occupational noise environments: Group I: ≤70 dB, administrative workers, N=48; Group II: >70 dB and <90 dB, ancillary workstation technicians, N=113; Group III: ≥90 dB, aircraft technicians, N=324. The average age of the entire population was 37.9 years (range 19-63), and the same exclusion criteria as described in the 1999 study were applied. Echo-imaging parameters were quantified with a 7-grade scoring system (see Methods). Tables 2 & 3 show that thickening of cardiac structures, namely the pericardium and mitral valve, can be directly related to the individual's

occupational noise exposure. Table 4 shows other cardiac pathology observed in the study groups. (9)

Table 2 – Mean Scores For Each Noise Group

[1]Groups	Mean score (SD)	No. Cases
Mitral Valve Thickening		
I	0.43 (0.50)	48
II	0. 88 (0.34)	113
III	1.49 (0.55)	324
Aortic Valve Thickening		
I	0.25 (0.43)	48
II	0.49 (0.51)	113
III	1.02 (0.53)	324
Tricuspid Valve Thickening		
I	0.21 (0.41)	19*
II	0.58 (0.49)	53*
III	1.14 (0.43)	215*
Pulmonary Valve Thickening		
I	0.75 (0.50)	4*
II	0.83 (0.38)	18*
III	1.19 (0.41)	127*
Endocardial Thickening		
I	0.33 (0.47)	48
II	0.74 (0.44)	112*
II	1.37 (0.57)	324
Pericardial Thickening		
I	0.47 (0.50)	48
II	0.95 (0.26)	112*
III	1.81 (0.50)	324

[1] Group I: £70 dB (N=48), Group II: >70 dB and < 90 dB (N=113), Group III: ³90 dB (N=324)
The number of cases varies due to the lack of visibility in some of the videotaped echocardiograms.

Table 3 – Statistical Significance of Comparisons Among Different Noise Groups

Cardiac Parameters	Groups[1]		
	I vs. III	***I vs. II***	***II vs. III***
Mitral Valve Thickening	hs	hs	hs
Aortic Valve Thickening	hs	s	hs
Tricuspid Valve Thickening	hs	s	hs
Pulmonary Valve Thickening	hs	n	s
Endocardial Thickening	hs	n	n
Pericardial Thickening	hs	hs	hs

[1] Group I: £70 dB (N=48), Group II: >70 dB and < 90 dB (N=113), Group III: ³90 dB (N=324)
n, not significant ($p < 0.01$), *s*, significant ($p < 0.001$); *hs*, highly significant ($p < 0.0001$)

Table 4 – Percentage of cases disclosing the associated cardiac abnormality.

Cardiac Parameters	*Noise Groups*[1]		
	I ***(N=48)***	***II*** ***(N=113)***	***III*** ***(N=324)***
Mitral Regurgitation	33.3	44.2	66.0**
Mitral Prolapse	4	9	19**
Ruptured Chordae Tendinae	2.2	10	17.4**

[1] Group I: £70 dB (N=48), Group II: >70 dB and < 90 dB (N=113), Group III: ³90 dB (N=324)
*** Indicates a highly significant statistical difference (p<0.0001)*

2001 Study -1

This study refers to the first documented cases of environmental LFN exposure. Volunteer (exposed) population (N=50): 34 males and 16 females (ave. age 42.4, range 7-78), all permanent residents of Vieques, Puerto Rico[2] since birth. These individuals were chosen from the crowds of residents of Vieques that gathered at the school where the echocardiograms were been performed, demanding that they and their families be included in the study. Volunteer control population (N=50): 45 males and 5 females (ave. age 43.6, range 7-79), all permanent residents of Ponce (city located on a different island) since birth. The principal professional activity of the adult males of both groups was fishing. The exclusion criteria for both groups was intended to be the same as that used in previous studies, however given the crowds of parents demanding that researchers examine their children, time did not permit for an adequate selection of the adults. Echocardiograms were recorded on VHS in Vieques, evaluated by Puerto Rican cardiologists, and then blindly evaluated by Portuguese and Polish cardiologists. Pericardial thickening above 2mm (as measured through echo-imaging) was identified among 96% of the Vieques residents. At least 2 cardiac abnormalities were observed in 80% and at least 3 abnormalities in 36%. The distribution of valve abnormalities was distinct in both populations: Ponce control group valve abnormalities were related to age, but this did not hold for the exposed group of Vieques residents, indicating that their valve abnormalities were not conditioned by age. (10)

Table 5 –Cases disclosing the associated cardiac abnormality (N=50)

Cardiac Abnormality	No. Cases	Ave. age (years)	Age Range (years)
Thickening of			
-- Aortic Valve	27	52	16 – 78
-- Mitral Valve	15	49	7 – 78
Regurgitation of			
-- Aortic Valve	19	49	11 – 78
-- Mitral Valve	24	48	7 - 78

[2] Vieques, Puerto Rico, has been the site of naval military training exercises for the past 40 years. These activities produce large amount of LFN, extending to the populated areas via air and water.

2001 Study -2

The occupational environment within commercial airlines was the focus of this study. Study populations consisted of 30 male pilots, ave. age 48 (range 30-61), and 30 female flight attendants, ave. age 43 (range 27-57). Total exposure time of all individuals (i.e. years of professional activity) was >8 yr. Six of the female flight attendants were on sick leave, waiting for disability retirement. All 6 had been diagnosed with severe balance disorders, 3 with simultaneous late-onset epilepsy, and 1 of these 3 also suffered from systemic lupus erythematous. All pilots were on active duty. The same scoring system was used as in 2001 Study-1. No differences were found when the overall scores were compared. However, if both groups were divided by age (≤45 and >45 years of age), scores were significantly ($p<0.001$) larger for pilots than for flight attendants, within the ≤45-year age group. This has been associated with the fact that cockpit environments yield higher levels of infrasound than cabins, due to the impact of aerodynamic flow (11,12)

2004 Study

This is another environmental case of excessive LFN exposure, this time in suburban – Lisbon[3]. Mr. F., a 39-year-old architect, lives with his family in Restelo, located on the hillside of the northern margin of the Tagus River, just outside of Lisbon. Directly in front, on the opposite side of the river, operates a grain cargo-ship docking site. Until 2003, this industrial complex could function at any hour of the day or night. In February 2003, Mr. F. contacted our team complaining about LFN in his home. Mr. and Mrs. F., a 42-year-old forestry engineer (employed in an office), had moved into their home in August 1992. Today they have two children, P. their 10-year-old son, and an 18-month-old baby. Mr. F., Mrs. F and P. disclosed a pericardial thickening of 2-points, 1-point and 3 points, respectively. Both Mrs. F. and P. had mitral valve thickening of 1- and 3- points respectively. The most severe cardiac pathology was disclosed by 10-year-old P. Based on studies carried out with LFN-exposed animal models (13,14), it is hypothesized that P.'s severity is related to the fact that Mrs. F. already spent her pregnancy in this home, while exposed to LFN. (15)

DISUCSSION

Today, echo-imaging has become one of the most important tools in diagnosing VAD. However, echo-imaging increases anatomical size by a maximum factor of 3, depending on equipment model and manufacturer. The natural question would then be: "what is causing this thickening?" Autopsy results showed it was increased amount of fibrous tissue, however that alone did not explain the lack of expected diastolic dysfunction, given the obvious enlargement of the pericardium. Moreover, the true anatomical thickness of these pericardia remained unknown.

In order to further explain the echo-images observed in VAD patients (in M-mode), it is necessary to understand the electron microscopy results obtained in the

[3] See "Environmental vibroacoustic disease – An example of environmental low frequency noise exposure", included in these proceedings.

study of pericardial fragments[4]. Some VAD patients are recommended for cardiac bypass surgery due to coronary insufficiency (caused by blood vessel wall intima thickening, and not atherosclerotic plaques). With patients' fully-informed consent, and Hospital Ethics Committee approval, fragments of the pericardial sac were removed during surgery (to reach the heart the pericardial sac must be opened) and studied under light and electron microscopy. Indeed, pericardial thickening, as seen through echo-imaging, had an anatomical correspondence. Normal pericardial thickness is <0.5 mm. In the 12 VAD cases observed to date, thickness ranged from 1.03 mm to 2.23mm. Thickening was due to the splitting of the pericardial fibrous layer into two halves, and the neo-formation of a loose tissue layer in between these halves (16,17)[2]. Thus, the VAD pericardium has an internal fibrous layer (closer to the pericardial sac) and an external fibrous layer (adjacent to the epipericardium). In severe cases of LFN-induced pericardial thickening, such as in the 10-year-old boy (2004 Study), the newly formed internal and external fibrous layers are visible in M-mode echocardiography: the internal layer follows the systolic wave while the external layer does not.

The relationship of echocardiograms and audiograms (1996 Study) shows that the audiogram is not a good indicator of LFN-induced pathology, or of the progression of VAD. This is especially the case if only the 4000 Hz notch is considered. Losses at the 250 Hz and 500 Hz notches are significant in VAD patients, but are irrelevant for the establishment of the only legally-recognized pathology associated with noise exposure – hearing loss.

Echocardiography would be an excellent, non-invasive and relatively inexpensive VAD diagnostic tool if technician subjectivity were not an issue, or if a standardized procedure to assess pericardial thickening were established. Pericardial thickening is seen as an echo-dense image. This density can be increased or decreased with the GAIN function, and thus assessment of pericardial thickness becomes tricky, and difficult to standardize. Hence, the search for a more objective diagnostic tool has never ceased, and new developments have been unfolding within this context in the field of VAD-associated respiratory pathology (18,19)[5].

SUMMARY

In vibroacoustic disease, echocardiography discloses thickened pericardia and mitral valves, among other pathology. Although the echocardiogram has a large degree of subjectivity, it is a valuable tool for diagnosis LFN-induced pathology.

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[4] See "The pericardium in vibroacoustic disease I, II and III", included in these proceedings, and which provide an extensive and detailed report of electron microscopy findings in VAD patients' pericardia.

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