Echocardiographic Evaluation in 485 Aeronautical Workers Exposed to Different Noise Environments

W. MARCINIAK, M.D., PH.D., E. RODRIGUEZ, M.D., PH.D.,
K. OLSZOWSKA, M.D., O. ATKOV, M.D., PH.D.,
I. BOTVIN, M.D., A. ARAUJO, M.D., F. PAIS, M.D.,
C. SOARES RIBEIRO, M.D., PH.D., A. BORDALO, M.D.,
J. LOUREIRO, M.D., E. PRAZERES DE SÁ, M.D.,
D. FERREIRA, M.D., M. S. N. CASTELO BRANCO, M.Sc.,
AND N. A. A. CASTELO BRANCO, M.D.

MARCINIAK W, RODRIGUEZ E, OLSZOWSKA K, ATKOV O, BOTVIN I, ARAUJO A, PAIS F, SOARES RIBEIRO C, BORDALO A, PRAZERES DE SA, FERREIRA D, LOUREIRO J, CASTELO BRANCO MSN, CASTELO BRANCO NAA. *Echocardiographic evaluation in 485 aeronautical workers exposed to different noise environments.* Aviat Space Environ Med 1999; 70(3, Suppl.):A46–53.

Introduction: Vibroacoustic disease (VAD) is a heterogeneous and systemic entity, caused by long term (≥10 yr) exposure to noise environments characterized by large pressure amplitude and low frequency (LPALF) (≥90 dB SPL, ≤ 500 Hz), and not explained by other possible etiologic agents. The goal of this study was to identify possible structural changes in hearts of men with suspected VAD. ***Methods:*** A total of 485 men were divided into 3 noise groups: no noise exposure (≤70 dB), n = 48 (Group I); moderate noise exposure, (>70dB and < 90 dB), n = 113 (Group II); and intense noise exposure (≥90 dB), n = 324 (Group III). Echo-Doppler studies were performed (HP SONOS 1500) and recorded on coded videotapes. Three observers performed blinded evaluations of 26 echo-Doppler parameters. For the purpose of the present study only 12 morphological parameters were compared among the groups: thickening of the mitral, aortic, tricuspid, and pulmonary valves, pericardium and endocardium; mitral valve regurgitation, prolapse and ruptured chordae tendinae; and inflow velocities. Thickness and severity of the applicable parameters were scored in seven-grade scale (0,0.5,1,. . .,3). ***Results:*** All evaluated parameters were statistically significantly different in Group I vs. Group III, except flow velocity E. Comparison of Group I vs. Group II revealed statistically significant differences in mitral, aortic, tricuspid and pericardial thickening, with the strongest evidence for mitral and pericardial structures. ***Conclusions:*** This confirms the results of previous studies. Occupational exposure to noise environments characterized by LPALF noise causes structural changes in the heart. Mitral valve and pericardial thickening constitute the first signs of VAD.

Keywords: noise, vibration, occupational, thickening, pericardium, mitral, tricuspid valve, aortic valve, endocardium.

VIBROACOUSTIC DISEASE (VAD) is a heterogeneous and systemic entity, caused by long term (≥10 yr) exposure to noise environments characterized by large pressure amplitude and low frequency (LPALF) (≥90 dB SPL, ≤ 500 Hz), and not explained by other possible etiologic agents (4,5,9,14). Since 1987, attention has been paid to the echo-images in VAD patients. In previous echocardiography studies performed on VAD patients, 100% had thickening of some cardiac structure, with the vast majority presenting thickened pericardia and mitral valves (1,2,17).

In 1983, Matoba (15) first described pericardial thickening in noise exposed workers. His population consisted of chainsaw workers where, in spite of the predominant high frequency noise, there is also an important acoustic component within the lower frequency bands. These pericardial changes were not identified in the entire population, probably because of differences in total exposure time and individual susceptibility.

A few years later in 1987, we performed an autopsy on a patient from our population (7), and found marked thickening of the pericardium and mitral valve. In the clinical history of this man there was no reference to any symptoms that could be linked to diastolic problems. Unfortunately, there was no echocardiogram for this patient. Since then we have been performing echocardiograms on our entire population and presented our first results in 25 patients in 1989 (1); thickening of the pericardium or mitral valve was found in 100%, aortic valve (70%), endocardium (90%) and tricuspid valve (60%). At this time, only the morphological study of the cardiac structures was performed. Later, Doppler studies were carried out (2); the pericardium was again the most common thickened structure, found in 100% population (n = 56). No statistical differences in the E/A ratio, related to either age or exposure time were found with Pulsed Wave Doppler. In 1993, in a population of 134 VAD patients employed in LPALF noise environments, we found that all subjects had thickening of at least one cardiac structure (17). Pericardial thick-

From the Cardiology Department, Central Hospital of Military School, Warsaw, Poland (W. Marciniak, K. Olszowska); International Institute for Human Performance, Los Angeles, CA (E. Rodriguez); Cardiology Research Center, Russian Academy of Medical Sciences, Moscow, Russia (O. Atkov, I. Botvin); Coronary Unit, Santa Maria University Hospital, Lisbon, Portugal (A. Araujo, F. Pais, C.Soares Ribeiro, A. Bordalo, E. Prazeres De Sa, D. Ferreira, J. Loureiro); Espírito Santo Banking Group, Lisbon, Portugal (M. S. N. Castelo Branco); Occupational Medicine Research Center, Lisbon, Portugal (N. A. A. Castelo Branco); and the Center for Human Performance, Alverca, Portugal.

Address reprint requests to: N. A. A. Castelo Branco, M.D., Estrada Nacional No.10, Edificio Cinema, sala 109, 2615 Alverca, Portugal. Prof. Marciniak is Associate Professor at the Central Hospital of Military Medical School, Warsaw, Poland.

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ening was found in 130 individuals. No changes in mitral inflow character were observed in any of the patients.

The goal of this study was to identify possible structural changes in hearts of men with suspected VAD.

METHODS

At OGMA, an aeronautical plant of 1500 workers, a random selection of 486 healthy male Caucasian employees with no known vascular risk factors were chosen as our study population. The exclusion criteria used is described elsewhere in this Supplement (Castelo Branco and E. Rodriguez, Table I, page A2). The average age of the population was 37.9 yr (range 19–63).

Employee workstations were classified into three categories depending on noise characterization, and study groups were divided accordingly: Group I (control group), n = 48, no noise exposure (≤70 dB), e.g., administrative personnel; Group II, n = 113, moderate LPALF noise (>70 dB and < 90 dB), e.g., ancillary workstation technicians; and Group III, n = 324, intense LPALF noise (≥90 dB), e.g., aeronautical technicians.

An echocardiogram was performed on the entire population using HP 1500 SONOS, 2-D, M mode, color Doppler analysis and spectral Doppler. All 486 echocardiograms were recorded on VHS video tape. They were later blindly evaluated by three independent observers (Poland, Portugal and Russia) who focused on the following parameters: 1) thickening of mitral valve; 2) tricuspid valve; 3) pulmonary valve; 4) aortic valve; 5) endocardium; 6) pericardium; 7) mitral valve regurgitation; 8) prolapse; 9) ruptured chordae tendinae; 10) velocity flow A; 11) velocity flow E; and 12) 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) and 3 points for maximum thickening (or severe regurgitation or prolapse). The results were compared among all groups. Statistical analysis was performed using the SPSS package (16). Statistical significance was established as follows: not significant if $p < 0.01$, significant if $p < 0.001$, and highly significant if $p < 0.0001$.

RESULTS

Please see **Tables I-IV** for summary of results, and **Figs. 1–4.**

Mitral valve thickening: Mitral thickness was identified as a more intensely lit screen image, less motion and an obvious thickened area. (See **Fig. 1.**) In some cases the leaflets had similarities to myxoma. Mitral leaflet thickness was normal in Group I (control group). There were statistically significant (s.s.) differences between the control group and both other groups as well as between Group II and Group III ($p < 0.0001$).

Aortic valve thickening: All groups presented s.s. differences regarding aortic valve thickening, being highly significant in Groups I vs. III and II vs. III.

Tricuspid valve thickening: Tricuspid valve thickening presented s.s. differences when comparing all groups, and was highly significant in Groups I vs. III and II vs. III.

TABLE I. MEAN SCORES FOR EACH NOISE GROUP.

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*
III	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

† Group I: ≤70 dB; Group II: >70 dB and <90 dB; Group III: ≥90 dB.
* The number of cases for this parameter is different than that for other parameters due to the lack of visibility in some of the videotaped echocardiograms.

Pulmonary valve thickening: Differences in pulmonary valve thickening were highly s.s. between Groups I and III, s.s. between Groups II and III, and not significant in Groups I vs. II.

Endocardial thickening: Differences in endocardial thickening were only s.s. between Groups I and III, and were not significant in Groups I vs. II and II vs. III (see **Table II**).

Pericardial thickening: There were highly s.s. differences among all groups regarding pericardium thickening (see **Figs. 1–4** and Table II).

Mitral valve regurgitation, prolapse and ruptured chordae tendinae: For all these parameters, s.s. differences were found between Groups I and III (see **Table III**). The severity of mitral regurgitation and prolapse in the Group III (<90 dB) was significantly higher than those of the control Group I (>70 dB).

Flow velocity: Only flow velocity A, and E/A ratio registered s.s. differences between Groups I and III (see **Table IV**).

DISCUSSION

Valves

Morphological changes of cardiac valves include thickening, calcification, degeneration and/or restriction of leaflet movement (3). In general, some of the more common reasons for morphological changes of the tricuspid and mitral valves are: rheumatic fever, endocarditis, myxomatous proliferation or connective tissue diseases (3). None of these conditions existed in our population.

Morphological changes of the aortic valve are most frequently due to overloading which may lead to ste-

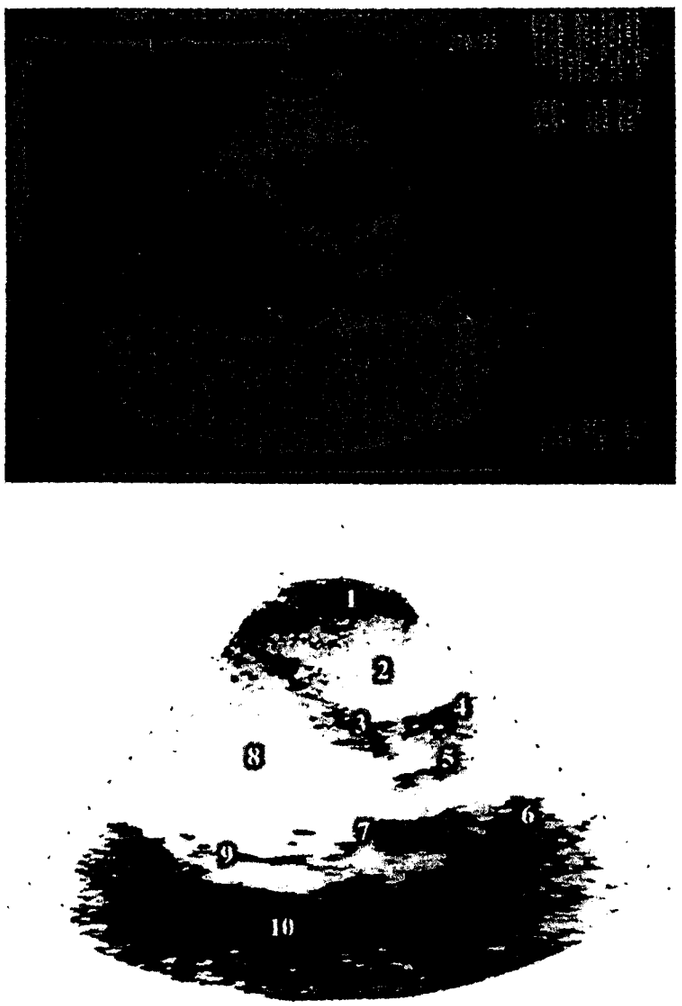

Fig. 1. Parasternal long axis view of a subject with thickened cardiac structures: right ventricular wall (1); right ventricle (2); ventricular septum (3); aorta anterior wall (4); aorta cusps (5); aorta posterior wall (6); mitral posterior leaflet (7); left ventricle (8); chordae tendinae (9); and the pericardium (10).

nosis. In young adults, stenosis of the aortic valve is largely due to congenital defects of the mitral valve. In our population we did not find anyone with such congenital defects. The normal aging process can also produce stenotic valves in the elderly (3). The average age of our population was 37.9 yr. For the pulmonary valve, the vast majority of morphological changes are due to congenital stenosis (3). This condition was non-existent in our population.

Highly statistically significant differences ($p < 0.0001$) between Groups I (≤70 dB) and III (≤90 dB) were found for thickening in all valves.

TABLE II. DEGREE OF SIGNIFICANCE WHEN COMPARING THE VARIOUS NOISE GROUPS.

Cardiac Parameters	Groups*		
	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

* Group I: ≤70 dB; Group II: >70 dB and <90 dB; Group III: ≥90 dB.
† n = not significant, $p < 0.01$; s = significant, $p < 0.001$; hs = highly significant, $p < 0.0001$.

Endocardia

Intensely lit images and thickening of the endocardium is a situation that can be observed in conditions such as ischemic heart disease, hypertension, endomyocardial fibrosis, and radiation exposure (3). Subjects with pre-existing cardiovascular disease were excluded from our population. Other conditions described above were not present in our population.

Intensely lit images and endocardium thickening appeared more evident in Group III than in Group I with a high degree of statistical significance ($p < 0.001$). Comparison among Groups I–II and II–III were not significant ($p < 0.01$).

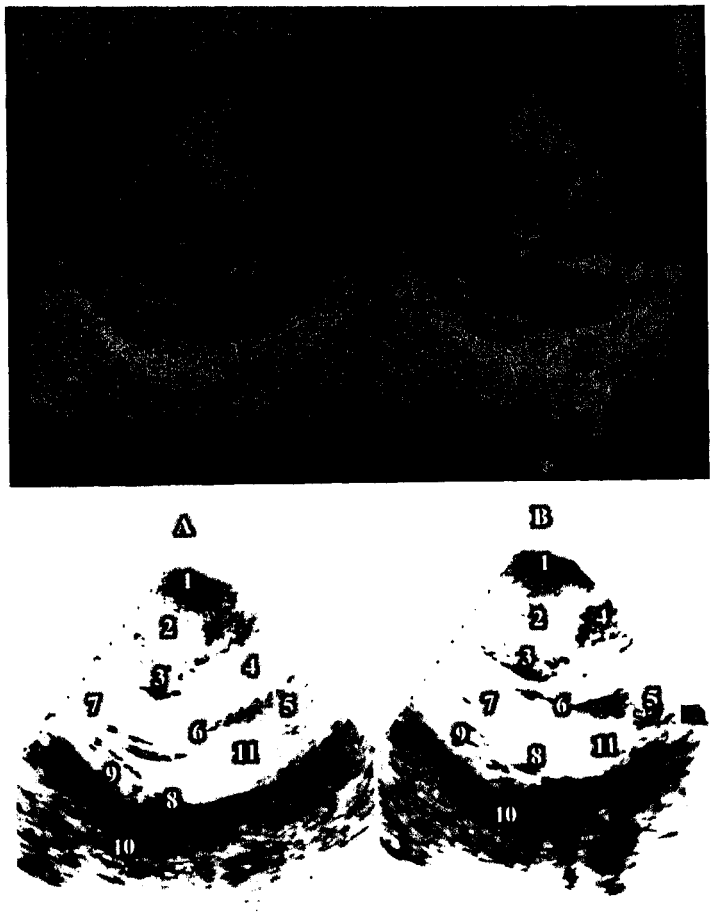

Fig. 2. Parasternal long axis view of a patient with thickened cardiac structures in systole (A) and in diastole (B): right ventricle anterior wall (1); right ventricle (2); ventricle septum (3); aorta anterior wall (4); aorta posterior wall (5); mitral valve anterior leaflet (6); left ventricle (7); mitral valve posterior leaflet (8); left ventricle posterior wall (9); pericardium (10); and left atrium (11).

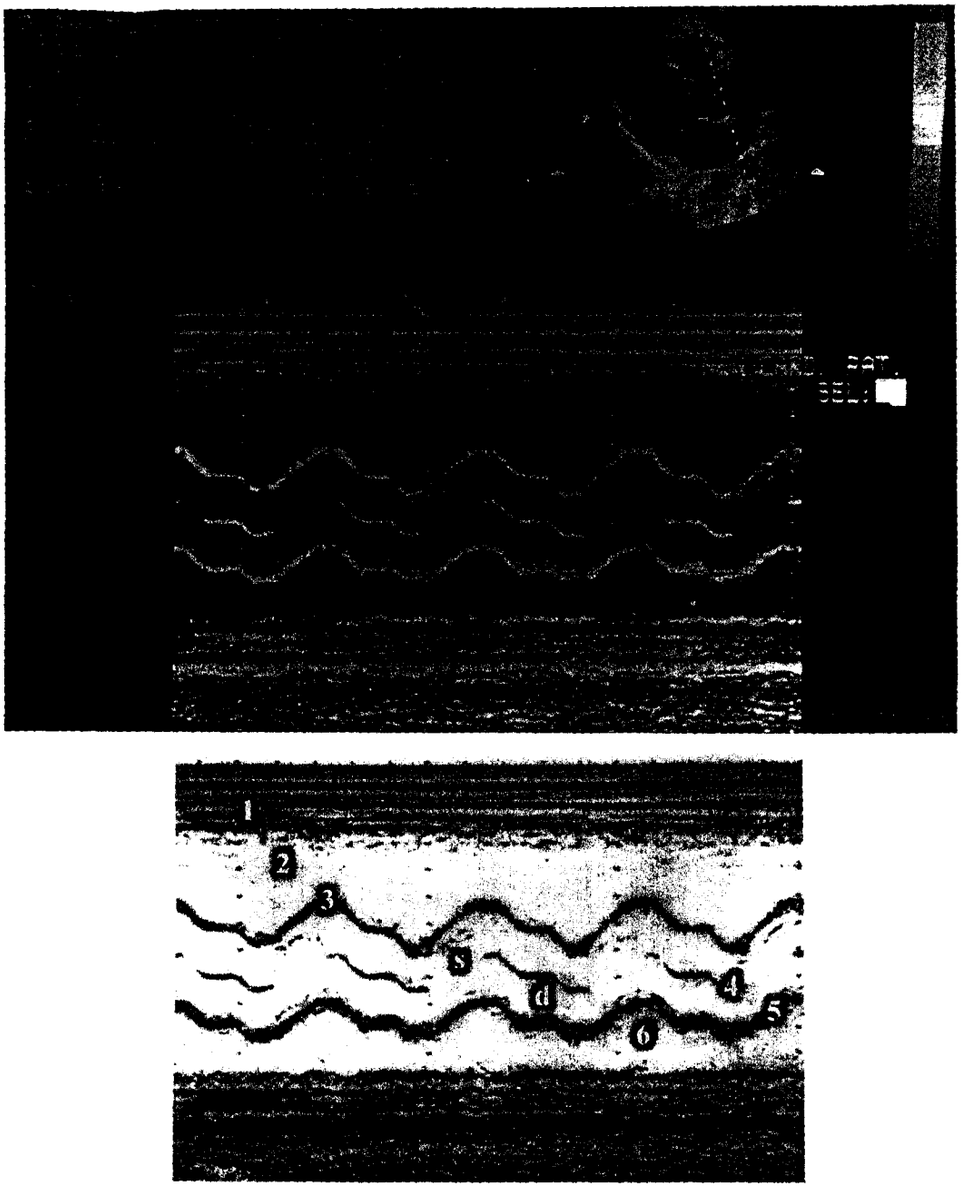

Fig. 3. M-mode recording through the aortic root in an adult with thickened cardiac structures: right ventricle anterior wall (1); right ventricle (2); aorta anterior wall (3); anterior and posterior aortic cusps in systole (s) and diastole (d) (4); aorta posterior wall (5); and left atrium (6).

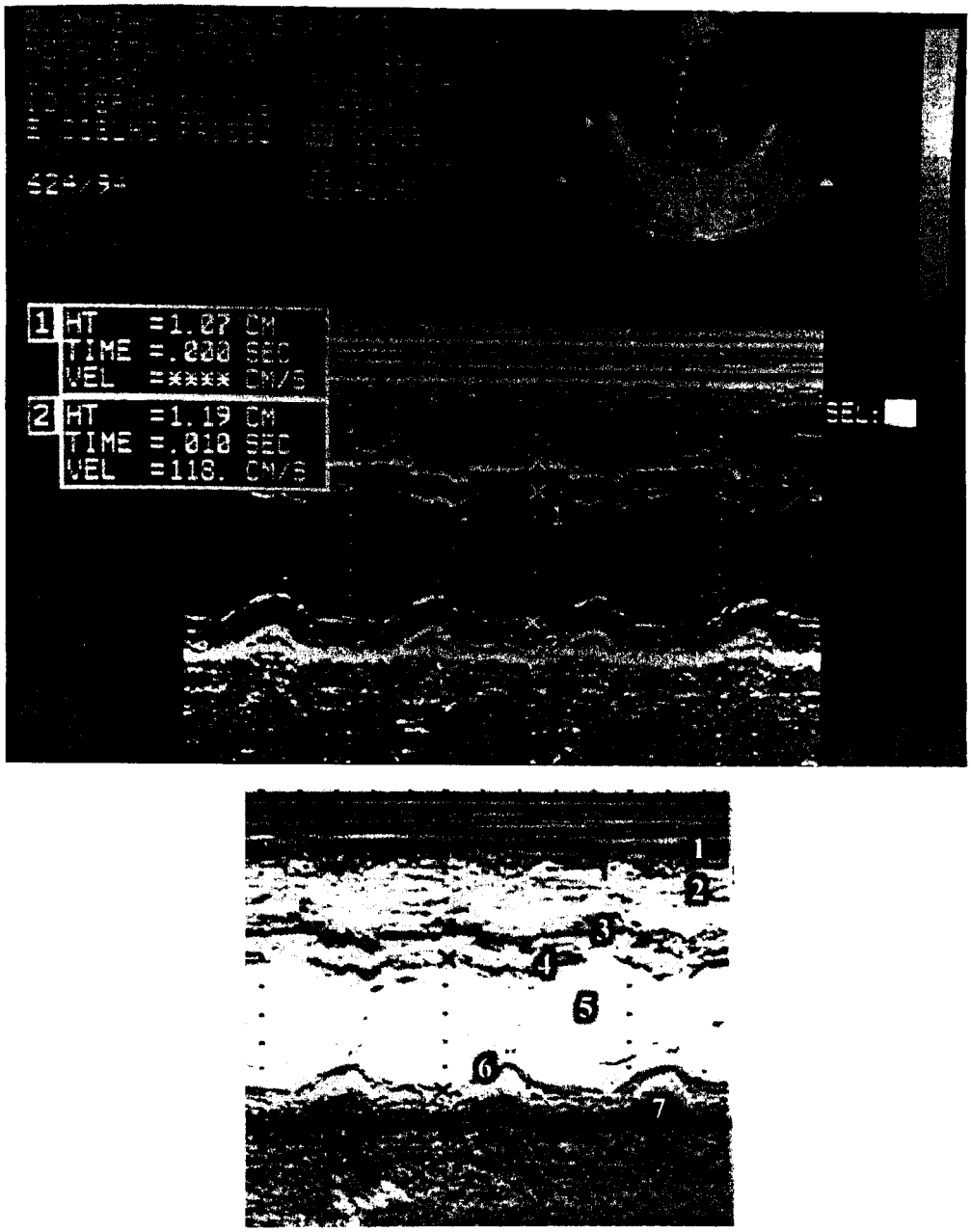

Fig. 4. M-mode recording in a VAD patient demonstrating thickened structures: right ventricle anterior wall (1); right ventricle (2); right side of the ventricular septum (3); left side of the ventricular septum (4); left ventricles (5); posterior wall of the left ventricle (6); and the pericardium (7).

TABLE III. INCIDENCE OF CASES WITHIN EACH NOISE GROUP.

Noise Groups*	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**

* Group I: ≤70 dB; Group II: >70 dB and <90 dB; Group III: ≥90 dB.
† Values are in percentages (%) of group.
** $p < 0.0001$ compared to Noise Group I.

Mitral Valve Regurgitation

Mitral regurgitation can be associated with a variety of conditions, e.g., rheumatic heart disease, connective tissue diseases, endocarditis, dilatation of valve annulus, congenital defects, mitral valve prolapse, and ruptured chordae tendinae. The latter two conditions were identified in many subjects of Group III, but it is unclear whether these conditions were directly related to the cases of regurgitation. All other conditions were nonexistent in our population.

Mitral Valve Prolapse

Echocardiography is an extremely reliable diagnostic tool for mitral valve prolapse. With this method, during systole one can clearly see one or both valve leaflets billowing into the left atrium. In this population, 4% of Group I vs. 19% in Group III had prolapsed mitral valve. In accordance with Braunwald (3), 3–5% of the population at large has mitral valve prolapse of varying degrees, mostly as a primary condition but also caused by hereditary connective tissue diseases, von Willebrand's disease, congenital thoracic deformities and others. In Marfan Syndrome, 90% of the patients have mitral valve prolapse (3). In our population none of these conditions existed.

Ruptured Chordae Tendinae

This condition may occur as a consequence of rheumatic fever, endocarditis, congenital abnormalities, ischemic heart disease, dilation of left ventricle, and direct trauma to the chest. None of these conditions existed in our population. In most cases, however, ruptured chordae tendinae is an idiopathic situation.

TABLE IV. DEGREE OF SIGNIFICANCE WHEN COMPARING MITRAL VALVE FLOW PARAMETERS AMONG ALL NOISE GROUPS.

Noise Groups*	I vs. III (N = 48)	I vs. II (N = 111†)	II vs. III (N = 314†)
Velocity Flow E	n‡	n	n
Velocity Flow A	hs	n	n
E/A ratio§	s	n	n

* Group I: ≤70 dB; Group II: >70 dB and <90 dB; Group III: ≥90 dB.
† The number of cases for this parameter is different than that for other parameters due to the lack of visibility in some of the videotaped echocardiograms.
‡ n = not significant, $p < 0.01$; s = significant, $p < 0.001$; hs = highly significant, $p < 0.0001$.
§ All E/A values were within normal values.

Flow Velocity

Unexpectedly, it was Group III which had the lower value of E/A. When we divided the entire population into three age groups, (A <39, >39 B <45, C >45), the E/A parameter decreased as expected, It should be noted that even though there was a statistically significant difference between Groups I and III, all E/A values were within normal limits.

Pericardia

Evaluation of the pericardium using computed tomography (CT) and magnetic resonance imaging (MRI) is a frequent method used in cardiac diagnosis (10,13). Echocardiography and Doppler ultrasound are also very useful in evaluating cardiac abnormalities (3,8). Both M-mode and two dimensional echocardiography may be very useful in diagnosing thickened cardiac structures. These echocardiographic signs have a high degree of sensitivity and specificity (8).

Pericardial thickening is not a very common finding (3). Some of the unusual cases can be observed in collagenous diseases, infections, tumors and in asbestosis (10,11,13,18,19). None of these conditions were identified in our population.

Pericarditis is a condition that could lead to pericardium thickening. Pericarditis is mostly commonly caused by viral, bacterial (especially tuberculosis), fungi and parasite infections, uremia, acute myocardial infarction, neoplasm, and direct chest trauma (3,10–13,18,19). Autopsy findings indicate that pericardial inflammation has an incidence of 2–6% and only 0.1% of the hospital-admitted population has symptoms of pericarditis (3). No element of our population had been identified with pericarditis nor pericardial inflammation. Differences in pericardial thickening were found to be highly statistically significant in all Group comparisons (see Table II).

Pericardial Thickening and VAD

All the known reasons for pericardial thickening, (e.g., pericarditis, asbestosis, etc.) have been eliminated within our population. The degree of pericardial thickening seems to increase with the level of noise (compare mean scores in Table I with noise level in each of the Groups).

Pericardial thickening has been identified by this team in noise-exposed individuals *not* employed by the aeronautical industry (5). Moreover, anatomical correspondence to this echo-image of pericardial thickening has been obtained through ultrastructural studies of pericardial fragments of VAD patients[1] (6). In VAD, pericardial thickening is due to the formation of an extra layer of loose tissue, sandwiched in between two thickened layers of fibrosa which contains an overabundance of collagen fibers (6). We believe that this type of pericardial thickening is specific to VAD; i.e., it is induced by LPALF noise exposure.

[1] These patients underwent surgery for other reasons (6).

CONCLUSIONS

The overall results of the echocardiographic evaluation regarding thickening of mitral, tricuspid, aortic and pulmonary valves, pericardium and endocardium suggest that occupational exposure to LPALF noise may induce the morphological changes observed in these subjects. This confirms the results of previous studies (1,2,17). The group of subjects diagnosed with VAD had a more obvious thickening of the cardiac structures. These findings are unusual for the population at large of the same age group (average 37 yr, range 19–63). The degree of thickening increased with the level of noise.

The most considerable thickening was found in the pericardium and mitral valve. Considering that all known reasons for pericardial thickening were eliminated from our population, we believe that this form of thickening identified in these patients is VAD specific, and is caused by exposure to LPALF noise.

The incidence of mitral valve regurgitation, prolapse and ruptured chordae tendinae is also unusual for the population at large, and suspicion is warranted that these conditions may be directly related to individuals' exposure to noise.

Given these results, we strongly suggest that all workers in noise environments be evaluated not only with an audiogram, for the purpose of hearing protection, but also by echocardiography so as to avoid the evolution of VAD.

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