

## STRUCTURAL CHANGES OF THE HEART IN PERSONS WITH SUSPECTED VIBROACOUSTIC DISEASE(VAD) - ECHOCARDIOGRAPHIC FINDINGS.

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### SUMMARY

Occupational exposure to high intensity and low frequency noise leads to proliferation of extra-cellular matrix, evident in the thickening of cardiovascular structures. A blind study evaluating echocardiographic changes was performed on an industrial population. An echocardiogram was performed on 500 healthy employees, randomly selected from 1500 workers in an aeronautical plant, and workstations were classified into three categories depending on noise levels. There are morphological and functional changes in all the noisy environment subjects. There is a statistically significant relationship (s.s.r.) between age-groups and noise levels regarding thickening of the aortic, tricuspid and mitral valves, endocardium, and pericardium. There is a s.s.r. between age and aortic root diastolic diameter, left atrium and intraventricular septum in diastole, independent of noise levels. There is a s.s.r. between noise levels and dimensions of the left ventricle in diastole, and pulmonary valve thickness, independent of age. Mitral regurgitation, prolapse and chordae rupture, and tricuspid regurgitation are frequent findings in subjects of the higher noise environments. There is a s.s.r. between age and aortic regurgitation, independent of noise levels. There is a s.s.r. between pericardium effusion and pericardium and tricuspid thickening. There is a significant (sig.) positive correlation (corr.) between noise environments and pericardium thickening. There is a sig. negative corr. between noise levels and pericardium effusion, also between pericardium thickening and pericardium effusion, and again between age and pericardium thickening. This study confirms previous statements that the echocardiogram is the diagnostic tool of choice for evaluation of the effects of occupational exposure to noise. The frequency of mitral prolapse and chordae rupture warrants a new approach to this study.

### INTRODUCTION

The Vibroacoustic Disease (VAD) is a heterogeneous and systemic entity, caused by occupational exposure to noise environments characterized by high sound pressure and low frequency, and not explained by other possible etiologic agents (European Commission on Health and Safety, 1995).

Since 1987, attention has been paid to the echocardiography appearances in patients occupationally exposed to high intensity/low frequency noise, suffering from the Vibroacoustic DISEASE (VAD). In a previous study performed on 25 workers with the same disease, thickening of the pericardium and the mitral valve was found in 100%. Thickening was also found in the aortic valve (70%), endocardium (90%) and tricuspid valve (60%) (1).

The first paper that described pericardium thickening in noise exposed workers was by Prof. Matoba, in 1983 (3). His population consisted of chainsaw workers where, in spite of the predominant high frequency noise, there is also an important component of low frequency bands. These pericardial changes were not identified in the entire population, only in a few. This is probably because of differences in total exposure time and individual susceptibility. A few years later in 1987, we performed an autopsy on a deceased patient from our population (2), and we found important thickening of the pericardium and mitral valve. In the clinical history of this patient there was no reference to any kind of symptom that could be tied to diastolic problems. Unfortunately there was no echocardiogram for this patient. Since then we performed echocardiograms on our entire population and we presented the first results in 1989 (4). All subjects (100%) presented some degree of abnormal pericardial and mitral valve thickening. In 65% there was abnormal thickening of the aortic valve. At this time, only the morphological study of the cardiac structures was performed. Later, Doppler studies were carried out (6). The pericardium was again thickened in 100% of this population. No statistical differences in the E/A ration, related to either age or exposure time were found with Pulsed Wave Doppler.

Last year, in a population of 134 workers we found that all subjects had thickening of at least one cardiac structure. Pericardial thickening was found in 130 (1). Here also no changes in mitral inflow character was observed in any of the patients.

The aim of this study was to find possible structural changes in hearts of persons with suspected VAD.

### POPULATION AND METHODS

In an aeronautical plant of 1500 workers, a random selection of 486 male, Caucasian, healthy employees with no known vascular risk factors were chosen as our study population. The average age of the population was 37.9 years (range 19-63). Exclusion criteria was as follows:

Pre-existing cardiovascular disease, but not labile hypertensives; Smokers of more than 20 cigarettes per day; Drinkers of more than a liter of wine or equivalent alcohol consumption per day; Users of any recreational or psychotropic drug; Diabetes mellitus; Streptococcal Infections, and inflammatory disease.

Employee workstations were classified into three categories depending on noise characterization: 1 - no noise exposure, e.g., office workers; 2 - moderate noise exposure, e.g. ancillary workstation technicians; and 3 - intense noise exposure, e.g., aeronautical technicians. The control group, Group I, consisted of 48 subjects who were in noise group 1. Group II consisted of 113 subjects who belonged to noise group 2. Finally, Group III consisted of 324 subjects from noise group 3.

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 and were later evaluated by three independent blind observers focusing on thickening of the mitral, tricuspid, pulmonary and aortic valves, endocardium and pericardium.

These six parameters were evaluated using a score system from 0 to 3 points: 0 points for no thickening and 3 points is maximum thickening. The results were compared among all three groups.

Statistical analysis was performed using SPSS package (5).

## RESULTS

Group I - Control (noise group 1)

Group II - subjects from noise group 2

Group III - subjects from noise group 3

### Mitral Valve Thickening

Mitral thickness was identified as a more intensely lit screen image, less motion and an obvious visual thickened area. In some cases the leaflets had similarities to myxoma.

Mitral leaflet thickness was normal in the Control Group (Noise Group 1). There were statistically significant (s.s.) differences between the control group and both other groups, as well as, between Group II and Group III ( $p < 0.0001$ ). (Table I and II)

Table I

| Groups                 | Deg. Signif. |
|------------------------|--------------|
| Group I vs. Group III  | $p < 0.0001$ |
| Group I vs. Group II   | $p < 0.0001$ |
| Group II vs. Group III | $p < 0.0001$ |

Table II

| Noise Group | Mean Score (+ S.D.) | CASES |
|-------------|---------------------|-------|
| 1           | 0.4375 (0.5013)     | 48    |
| 2           | 0.8850 (0.3472)     | 113   |
| 3           | 1.4969 (0.5308)     | 324   |

### Aortic Valve Thickening

All groups presented s. s. differences regarding aortic valve thickening. See Table III and IV.

Table III

| Noise Group | Mean Score (+ S.D.) | Cases |
|-------------|---------------------|-------|
| 1           | 0.250 (0.43)        | 48    |
| 2           | 0.4956 (0.51)       | 113   |
| 3           | 1.029 (0.53)        | 324   |

Table IV

| Groups                 | Deg. Signif.    |
|------------------------|-----------------|
| Group I vs. Group III  | $p < 0.0000001$ |
| Group I vs. Group II   | $p < 0.0046$    |
| Group II vs. Group III | $p < 0.0000001$ |

### Tricuspid Valve Thickening

Regarding tricuspid valve thickening, all groups presented s. s. differences. See Table V and VI.

Table V

| Noise Group | Mean Score (+ S.D.) | Cases* |
|-------------|---------------------|--------|
| 1           | 0.210 (0.418)       | 19     |
| 2           | 0.584 (0.497)       | 53     |
| 3           | 1.146 (0.435)       | 215    |

\* the number of cases for this parameter is different than that for other parameters due to the lack of visibility of the tricuspid valve in some of the videotaped echocardiograms.

Table VI

| Groups                 | Deg. Signif. |
|------------------------|--------------|
| Group I vs. Group III  | p< 0.000001  |
| Group I vs. Group II   | p<0.0046     |
| Group II vs. Group III | p<0.000001   |

*Pulmonary Valve Thickening*

Differences in pulmonary valve thickening was only s. s. between groups I and III and Groups II and III. (See Table VII and VIII.)

Table VII

| Noise Group | Mean Score (+ S.D.) | Cases* |
|-------------|---------------------|--------|
| 1           | 0.750 (0.50)        | 4      |
| 2           | 0.833 (0.38)        | 18     |
| 3           | 1.196 (0.41)        | 127    |

\* the number of cases for this parameter is different than that for other parameters due to the lack of visibility of the tricuspid valve in some of the videotaped echocardiograms.

Table VIII

| Groups                 | Deg. Signif. |
|------------------------|--------------|
| Group I vs. Group III  | p< 0.038     |
| Group I vs. Group II   | n. s. s.     |
| Group II vs. Group III | p<0.0006     |

*Pericardium Thickening*

There were strong s. s. differences among all groups regarding pericardium thickening. (See Table IX and X.)

Table IX

| Noise Group | Mean Score (+ S.D.) | Cases |
|-------------|---------------------|-------|
| 1           | 0.479 (0.50)        | 48    |
| 2           | 0.950 (0.26)        | 112*  |
| 3           | 1.819 (0.50)        | 324   |

\*In one case the pericardium thickness was not evaluated.

Table X

| GROUPS                 | DEG. SIGNIF |
|------------------------|-------------|
| Group I vs. Group III  | p< 0.000001 |
| Group I vs. Group II   | p<0.000001  |
| Group II vs. Group III | p<0.000001  |

*Endocardium Thickening*

There was a s. s. difference in the endocardium thickening between Groups I and III only. (See Table XI and XII.)

Table XI

| Noise Group | Mean Score (+ S.D.) | Cases |
|-------------|---------------------|-------|
| 1           | 0.333 (0.47)        | 48    |
| 2           | 0.741 (0.44)        | 112*  |
| 3           | 1.376 (0.57)        | 324   |

\*In one case the endocardium thickness was not evaluated.

Table XII

| GROUPS                 | DEG. SIGNIF |
|------------------------|-------------|
| Group I vs. Group III  | p< 0.000001 |
| Group I vs. Group II   | n. s. s.    |
| Group II vs. Group III | n. s. s.    |

All evaluated parameters were *s. s.* different in Group I vs. Group III; in Group II vs. Group III only the endocardium thickness did not differ. Comparison of Group I vs. Group II revealed *s. s.* differences in Mitral, Aortic, Tricuspid and Pericardium thickening with the strongest evidence for Mitral and Pericardial thickness.

## DISCUSSION

### VALVES

Morphological changes of cardiac valves include thickening, calcification, degeneration and/or restriction of leaflet movement. 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. None of these conditions existed in our population.

Morphological changes of the aortic valve are most frequently due to overloading which may lead stenosis. In young adults, stenosis of the aortic valve is largely due to congenital defects of the bicuspid valve. The normal aging process can also produce stenotic valves in the elderly.

In our population we did not find anyone with bicuspid valve. For the pulmonary valve, the vast majority of morphological changes are due to congenital stenosis. This condition is non-existent in our population.

Among all three groups *s. s.* differences were found for the aortic, tricuspid and mitral valve thickening. Differences in pulmonary valve thickening was observed to be *s. s.* between Group I and III, and Group II and III.

### PERICARDIUM THICKENING

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. Autopsy findings indicate that pericardial inflammation has an incidence of 2%-6%. Only 0.1% of the hospital-admitted population has symptoms of pericarditis. Although the subjects exposed to low-frequency noise appear to have a depression of the immune system, it could also be the case that the cause of reason is direct chest trauma by large amplitude sound pressure. In our population, Groups II and III had a larger degree of pericardial thickening than the control Group.

### ENDOCARDIUM THICKENING

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. In our population intensely lit images and endocardium thickening appeared more evident in Group III than in Group I. However, none of the subjects of Group III had a history of any of the medical condition mentioned above.

## CONCLUSION

The overall results of the echocardiographic evaluation regarding thickening of mitral, tricuspid, aortic and pulmonary valves, pericardium and endocardium suggests that occupational exposure to low frequency and high intensity noise may induce the morphological changes observed in these subjects. The group of subjects exposed to intense, low frequency noise presented a more obvious thickening of the cardiac structures, unusual for the population at large. The most considerable thickening was found in the pericardium and mitral valve.

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 worker disability.

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