CT Examination of the Pericardium and Lungs in Helicopter Pilots Exposed to Vibration and Noise

Jan Ivar Kåsin, Tor Ole Kjellevand, John Kjekshus, Geir B. Nesheim, and Anthony Wagstaff

Introduction: Helicopter pilots are exposed to whole body vibration and noise in their working environment. Some researchers have found that kinetic energy from both noise and vibration is believed to affect pericardial thickness and lead to pulmonary fibrosis, known as vibroacoustic disease. The aim of this project was to determine whether we could discover similar findings in a selection of helicopter pilots. Methods: A case control study where 27 helicopter pilots were compared to an age-matched control group of typical office workers was conducted. High resolution CT scanning of the thorax was used as the diagnostic method. Two medical radiologists interpreted the images independently, blinded to whether the subjects were pilots or from the control group. Results: There were no signs of pericardial thickening or significant lung fibrosis formations in either of the groups. The average pericardium thickness for the helicopter group was 1.38 mm, SD = 0.54 mm, and for the control group: 1.37 mm, SD = 0.33 mm. There was no significant correlation between pericardium thickness and flight hours or age. Discussion: The average pericardium thickness values for the helicopter and the age-matched control groups were almost identical. The results are within normal limits and comparable to an American study where 21 normal individuals were measured to 1.2 mm ± 0.8 mm in an average of 26 different points by using trans-esophageal echocardiography. Conclusion: On the basis of the CT scans, our findings do not support the existence of vibroacoustic disease, where pericardial thickening is the most prominent sign. Keywords: vibroacoustic disease, pulmonary fibrosis, whole body vibration.

HELICOPTERS ARE complex machines with many parts in rotational movement used in all kind of weather conditions with changing temperature, air pressure, and wind. The main rotor, tail rotor gearbox, and turbine engine interact with the different environmental factors, resulting in a degree of vibration and noise inside the aircraft. Helicopter pilots are exposed to whole body vibration in their working environment. The vibration is transmitted to the aircrew at all contact surfaces, including floor, control devices, and seating. The aircrew is also exposed to high noise levels in the cockpit environment. Kinetic energy from both noise and vibration will impact the human body. The way this energy is transmitted to different tissues and organs depends on intensity, frequency, and resonance phenomena within the body. The law of conservation of energy states that energy can neither be created nor destroyed: it can only be transformed from one state to another (31). The kinetic energy from noise and vibration that enter the human body affects the various tissues (10,30); this energy could gradually transform from kinetic movement of human cells to deformation and heating. Effects of vibration energy have been seen on the immune system with an increase in CD4+ and CD8+ T cells in humans during acute hand vibration exposure at 63 Hz (23) and has been associated with low back pain (24,27). Vibration energy has also been used in the treatment of acute coronary thrombosis (13) and is also used as a therapy for various neurologic diseases (16).

Researchers in Portugal have for more than three decades reported that kinetic energy from noise and vibration affects the human body and could cause vibroacoustic disease. Vibroacoustic disease is thought to produce psychological (11), physical (25), physiological (7), and cellular changes to the human body (28). One of the major reported effects is alteration in connective tissue, with a characteristic thickening of the pericardium (22). Fibrosis in lung tissue has also been detected by computed tomography (CT) in subjects believed to have vibroacoustic disease (26). Formation of focal lung fibrosis has been demonstrated in Wistar rats that were exposed to cyclical sound energy concentrated in the 50-500 Hz region, with noise levels higher than 90 dB for 4000 to 5000 h (12). It is suggested that long-term (>10 yr) exposure of low-frequency noise and vibration (>90 dB SPL, >500 Hz) can cause vibroacoustic disease, with thickening of the pericardium as the most prominent sign (4). However; as Von Gierke stated in 2002: “Vibroacoustic disease remains an unproven theory belonging to a small group of authors, and has not found acceptance in the medical literature” (32). The aim of the study was to investigate whether helicopter pilots exposed to low-frequency noise and vibration for a cumulative total of more than 4000 flight hours have evidence of pathological changes in the pericardium or lung tissue, such as thickening of the pericardium or focal...
lunb fibrosis, compared to an age-matched control group of typical office workers.

METHODS

**Subjects**

The study was approved by the regional ethics committee. All subjects signed an informed consent form. Recruited for the study were 30 male helicopter pilots from civilian helicopter companies. During the course of routine medical checks, the pilots were informed about the project and invited to participate in the study. The first 30 subjects who fell within the inclusion criteria regarding exposure (>4000 h of flying helicopters) were selected for the study. The noise environments of the helicopters flown by these pilots are generally well above 90 dBA. Helicopter noise consists of a broad range of frequencies, with most of the energy below 100 Hz. Low-frequency noise is generated aerodynamically, with the rotor blade pass producing a high energy peak at 17 Hz and harmonically related repetitions at 34, 51, 68, and 85 Hz (S-70A-9 Black Hawk) (17, 18).

The vibration levels for helicopters are typically between 0.3–0.5 m·s⁻² during a calculated 8-h working day (20). The vibration frequencies are, similarly to noise, closely related to the blade pass frequency. Therefore, the highest vibration peaks are, for instance, approximately 17 Hz for both the Westland Sea King MK43B and Sikorsky S92A, and 21 Hz for the Bell 412.

Exclusion criteria were any medication use, tobacco use, hypertension (>140/80 mmHg), any history of heart or lung disease, and any ongoing infectious disease. Of the 30 subjects, 3 subjects did not complete the study. Two subjects could not complete the study for practical reasons and one was excluded due to the finding of pulmonary sarcoidosis. Since this diagnosis has not been suggested as connected to noise or vibration exposure, it was considered as an unrelated diagnosis and therefore falling within the exclusion criteria.

A control group of 27 volunteers was recruited from typical low noise and vibration office environments. An office environment, according to regulations in Norway, shall not exceed 55 dBA for more than 1 h during an 8-h working day (2). They were matched to the study participants, age matching within ±8 mo for each pair. The control group were subject to the same exclusion criteria as the study group. Additional reasons for exclusion were a previous working environment with noise and/or vibration and/or regular participation in a hobby or activity that involved high noise and/or vibration levels.

**Procedure**

A Philips Brilliance 64-slice CT scanner (Phillips Healthcare, Holland) was used both for evaluating pericardial thickness and lung fibrosis. Settings were 2-mm sections with a pitch setting of 1.1, kv 120, mas 400. The algorithm was set to Cardiac Sharp CC. An electrocardiography device was coupled to the CT scanner in order to time the measurement exactly at end-diastole (75% of the cardiac cycle). We selected seven well-defined anatomical locations for evaluation of pericardial thickness in order to optimize measurement reliability. In particular, measurement points were selected in areas with fat tissue in order to optimize the contrast around the pericardial tissue. The superior and inferior aspects of the heart were avoided due to the obliquity of the pericardium in relation to the axial plane at these points, which would give rise to erroneous measurements.

Measurement points at the level of the origin of the right coronary artery were located in the following areas (Fig. 1): M1, in the AV atrioventricular groove; M2, between right and left atrium lateral right side; and M3, left anterolateral pulmonary artery. At the level of the outlet of the coronary sinus, the measurement points were located at (Fig. 2): M4, atrioventricular groove; M5, between the coronary sinus and the right atrium; M6, lateral aspect of the left ventricle; and M7, anterior aspect of the left ventricle. The perpendicular thickness in each location was registered manually by the built-in measurement tool in the CT-scanner picture diagnostic software. Two medical radiologists carried out the measurements independently of each other at separate occasions. Neither was aware of whether the subjects were pilots or from the control group.

The lung tissue was evaluated by the CT scanner in order to detect lung fibrosis. Settings were 2-mm sections with pitch of 1.1, kv 120, mas 100. The algorithm was set to Standard B. Signs of pulmonary fibrosis were placed in one of four diagnostic categories:

- 0 – No signs of pulmonary fibrosis;
- 1 – Moderate signs of pulmonary fibrosis with some thickening of interlobar septum;
- 2 – Moderate fibrotic changes with thickened interlobar septum and reticular subpleural opacities; or
- 3 – Fulminant fibrosis with findings as in stage 2 in addition to honeycombing, subpleural bullae and possible traction bronchiectasis.

Two medical radiologists carried out the evaluation independently of each other on separate occasions. Neither was aware of whether the subjects were pilots or from the control group.

**Statistical Analysis**

PASW statistics 18 (IBM Corp., New York) was used in the statistical analysis. The normal distribution was tested by using a Q-Q plot. Each anatomical location between the pilot and control groups was compared by using an independent samples *t*-test assuming equal variances. Correlations were tested with the Pearson correlation test. The interobserver variability was calculated by using the Bland Altman method in Excel 2010 (Microsoft, Redmond, WA).

**RESULTS**

Included were 27 helicopter pilots from civilian helicopter companies (ages 34-57) with an average of 7795 helicopter flight hours (SD = 2432) who were compared to 27 office workers. When comparing the average pericardial thickness of all seven anatomical locations on the heart of all pilots with the same anatomical locations obtained from the office workers, there is a difference of 0.01 mm
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Fig. 1. Location of measurement points M1–M3 at the level of the origin of the right coronary artery.

(helicopter: 1.38 mm, SD = 0.54 mm; control: 1.37 mm, SD = 0.33 mm; Fig. 3). This minor difference is below the CT image resolution and can therefore be ignored as not clinically relevant. Furthermore, comparison of each anatomical location (M1-M7) on the heart between the helicopter pilot and control groups respectively did not show any significant differences for any of the locations (Table I). The thickest area of pericardium was 6.9 mm measured in M7 in one pilot and 4.0 mm in M4 in one individual in the control group (Table II and Table III). The pericardial thickness had a normal distribution in every anatomical location except in the M7 position in the pilot group. The distribution at this location was slightly skewed upwards. There was no significant correlation between pericardium thickening and flight hours or age.

One of the pilots had pulmonary fibrosis in category 1 according to the predefined diagnostic categories. Pulmonary fibrosis was not seen in any of the controls. The mean differences between the observations were 0.05 mm, (SD = 0.31). The interobserver variability was normally distributed and 96% of the differences were within ± 1.96 SD from the mean (3).

DISCUSSION

There were no significant differences in pericardial thickness for any anatomical location between the pilot and control groups. Furthermore, there was no correlation between flight hours or age and pericardial thickness. The average pericardial thickness values for the helicopter and the age matched control groups were almost identical (helicopter: 1.38 mm, SD = 0.54 mm; control: 1.37 mm, SD = 0.33 mm). The results are within normal limits and comparable to an American study where 21 normal individuals were measured to 1.2 ± 0.8 mm in an average of 26 different points by using transoesophageal echocardiography (21). In one pilot, the examiners found the pericardium thickness to be 6.8 and 6.9 mm at the point of M7, and in one control person 3.8 and 4.2 mm at M4. In another pilot the examiners did not agree about the thickness and the results were 4.8 and 1.6 mm at the point of M7. M7 was defined with a wider anatomical margin than the other points and this could explain the discrepancy found here. A local fluid accumulation is another possibility for the thickened pericardium in both the pilot and control subjects or that there is a focal pericardium thickening in M7 and M4. CT images of the normal heart frequently show focal areas of pericardial thickening. The etiology of these focal thickenings is not always clear (29). Measurement of the thinnest portion of the pericardium is subject to much lower intraobserver variation than measurement of the thickest portion. A study concluded that measurement of the thickest portion of the pericardium is not useful when determining
whether a subject has generalized pericardial thickening (5). The six other measurement points in all three subjects did not show any signs of pericardial thickening. Therefore, the findings are overall negative in relation to the hypothesis. Furthermore, we could not demonstrate any significant pulmonary fibrosis by CT scanning.

Our findings are in contrast to a Portuguese study where aeronautical technicians exposed to a long-term high noise occupational environment (≥90 dB, <500 Hz, >10 yr) was compared to a typical long-term low noise exposed administrative personnel group (≤70 dB, >10 yr). In the aeronautical technicians group, significant pericardial thickening was found using echocardiographic evaluation (22). At a later date, four individuals from the same aeronautical workers group were diagnosed with vibroacoustic disease and underwent a bypass surgery or aortic aneurysm correction. One part in the pericardium in each individual was histologically examined. The thicknesses were, respectively: 1.11 mm, 1.35 mm, 2.19 mm, and 2.33 mm. These findings are suggested as elevated compared to the normal population, where the pericardium was described to be ≤0.5 mm (6). There was no reference in this paper that supported the origin of this normal value. However, anatomical cadaver studies have reported a normal pericardial thickness of 0.4–1 mm (9,14). The values fit well with a British study of normal values, where 100 CT-scanned people were evaluated. Pericardial thickness of normal values was found to be from an average minimum of 0.5 mm (SD = 0.1) to an average maximum of 2.8 mm (SD = 1.0) (5). These results are comparable to our study, where the average pericardial thickness was found to be 1.38 mm (SD = 0.45) in both the helicopter and control groups. This indicates those Portuguese groups’ findings in the vibroacoustic disease patients do not seem to be elevated from normal values and that normal pericardial thickness may have been underestimated. The Portuguese group defines low frequency to be below 500 Hz; in the helicopter environment most of the energy is between 1–100 Hz. This is the frequency range where the human body, because of its acoustic properties, is affected. Frequencies above 250 Hz seem to be less relevant for humans, since they affect the tissue to a lesser extent (30). This fact is also reflected in the weighting curves used in the whole-body vibration measurement standard (15).

There will be a variance in thickness between the measurement done directly during thoracotomy or indirectly by magnetic resonance imaging, echocardiography, or CT. There are individual variances and there are variances depending on the anatomical location of the measurements. The validity is higher in our study with measurement of seven different anatomical locations compared with a study where only one location was

Fig. 2. Location of measurement points M4–M7 at the level of the outlet of the coronary sinus.
looked at. The current study is based on CT, which has a higher resolution than echocardiography. Soft tissue contrast on CT scans is superior to echocardiogram contrast. CT imaging provides excellent anatomic delineation and enables precise localization of pericardial masses. In addition, CT is performed in standard imaging planes and does not require use of a transducer; it is, therefore, less operator dependent than conventional echocardiography (1,33). Use of high-resolution CT is undoubtedly a better methodology than conventional echocardiography to determine pericardial thickness. The results show consistency between the pilot and control group.

One limitation of the study is the limited number of subjects. The clear non-significant differences are, however, not indicative that a larger number of subjects (within practical reason) would have given us different results. Another limitation is the difficult nature of measuring pericardial thickness, as mentioned earlier, although we believe we have used the best current method. Notwithstanding the limitations of the study, some caution should be taken in generalizing the applicability of these results.

The helicopter pilots used in the study are a homogeneous and selected group since they are required to undergo regular medical examinations. They log every flight hour and helicopter noise and vibration exposure levels in general are well documented (20). The age-matched control group is recruited from typical office working environments without exposure to vibration and assumed to be representative for the average population of workers in low noise and vibration environments. The helicopter pilots have regularly been exposed to more noise and vibration energy than a typical office worker during their career and the study was unable to demonstrate a measurable increase in pericardial

![Fig. 3. Boxplot CT-measurements. K = control; P = pilot; * = Cases with values more than 1.5 box (interquartile) lengths from the upper or lower edge of the box; † = Cases with values more than 3 box (interquartile) lengths from the upper or lower edge of the box.](image)

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SD = standard deviation; P-value: independent samples t-test assuming equal variances. All measurement points in each location are included in the t-test.
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thickness or pulmonary fibrosis, suggesting that the level of noise and vibration experienced by the pilots does not have any effect on the tissue. This lack of long-term effect also fits well with a study where short-term effects in systemic biomarkers after a 3.5-h helicopter flight were not found (17), although very high energy vibration levels equal to 40 G may induce CK release (10) and high energy mechanical stress may cause long-term effects on connective tissue detected by changes in sensitive biochemical markers (8,19). We cannot exclude

### TABLE II. PERICARDIUM THICKNESS (mm) IN THE PILOT GROUP.

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Examiner 1: aM1, aM2, etc. Examiner 2: bM1, bM2, etc.

### TABLE III. PERICARDIUM THICKNESS (mm) IN THE CONTROL GROUP.

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Examiner 1: aM1, aM2, etc. Examiner 2: bM1, bM2, etc.
that the kinetic energy of noise and vibration may affect other organs in the body with different properties and resonance frequencies. Our intention was, however, to look at the presence of pericardial thickening and fibrosis as a part of vibroacoustic disease, and, fortunately for helicopter pilots, we did not find evidence to support such a diagnostic entity.

In conclusion: 1) 27 pilots with 4000 to 11,500 flight hours in helicopters exposed to low-frequency noise and vibration did not have any changes in the thickness of their pericardium or significant pulmonary fibrosis formation compared to an age-matched typical office worker control group; and 2) earlier findings made by other research groups may have been different due to lack of blinding, ultrasound assessment inconsistencies, and other definitions of the normal range for pericardial thickness. On the basis of CT scans, our findings do not support the existence of vibroacoustic disease, where pericardial thickening is the most prominent sign.

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