Vascular lesions and vibroacoustic disease

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SUMMARY

Low frequency noise (LFN) (≥ 90 dB SPL, ≤ 500 Hz) is an agent of disease that regularly goes unchecked during standard noise assessment procedures. Vibroacoustic disease (VAD) is an extra-aural noise-induced systemic pathology, caused by long-term exposure to LFN and characterised by a proliferation of extra-cellular matrices. The present study attempts to elucidate the behaviour of medium-and large-calibre blood vessels in the presence of insult due to noise and vibration. Thirty-five adult Wistar rats were studied. The animals were divided into 3 groups. One group of 20 rats was exposed to large pressure amplitude and low frequency (LPALF) noise in an occupationally simulated schedule: 8 h/day, 5 days/week, and weekends in silence, for 968 to 1984 (median 1576 h) cumulative hours of noise exposure. Another group of 5 rats was exposed to 24 h of continuous noise. The last group of 10 rats (control) was kept under identical conditions but in a silent environment. Overall noise levels were recorded above 109 dB, the A-weighted levels being around 98 dB (A). The rats were sacrificed and fragments of aorta, inferior vena cava and femoral artery and vein from both hindlimbs, were collected; these were prepared for histological examination. With long-term exposure, the aorta and the femoral artery showed a focal thickening of the intima, disruption of the internal elastic lamina and a proliferation of smooth muscle cells in the intima in 70% of the cases. With acute exposure, the lesions appeared in 60% of the cases observed. Most of the lesions involved the appearance of clefts in the media, pulling the cells and the elastic membranes apart. The media thickness-to-inner caliber ratio of the femoral artery was increased ($p<0.05$). Our results point to a remodelling of the vessels that can be attributed to vibration and flow disturbances. The observed remodelling was not observed in small vessels.

Key Words: Vibroacoustic disease – Low frequency – Noise – Vibration – Vessels

INTRODUCTION

Low frequency noise (LFN) (≥ 90 dB SPL, ≤ 500 Hz) is an agent of disease that is regularly overlooked during standard noise assessment procedures. The historical reason for this is the mistaken assumption that acoustic phenomena only affect the auditory system, i.e., the ear (Alves-Pereira, 1999). Since the human ear is less attuned to acoustic phenomena within the lower frequencies (≤ 500 Hz), and is incapable of perceiving infrasound (<20 Hz), LFN is not taken into account when noise protection procedures are assessed and implemented.

Low frequency noise – induced pathology has been studied in Portugal since the early 80's (GIMOGMA, 1984).

Vibroacoustic disease (VAD) is an extra-aural noise-induced systemic pathology caused by long-term exposure to LFN and characterised by a proliferation of extra-cellular matrices (Castelo Branco, 1999a, 1999b). Its features have been studied in most of organ systems. In the vascular system, the effects of vibration and low frequency noise are well known, particularly as regards the small blood vessels (Takeuchi et al., 1984; Okada et al., 1987; Inaba et al., 1988). A proliferation of the extra-cellular matrix occurs that causes hyperplasia of the intima, together with a migration of the smooth muscle cells of the media.

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One of the common complaints in VAD patients is the "white finger" phenomenon, which is brought by a reduction in blood flow due to a combination of vasospasm and a permanent narrowing of the vessel lumen (Littleford et al., 1997).

The aim of the present study was to attempt to elucidate the behaviour of medium-and large-calibre blood vessels in the presence of noise and vibration insult.

Material and Methods

Animals

Thirty-five adult Wistar rats were studied. All were housed individually, fed standard rat food, and had unrestrained access to water. The animals were treated in accordance with the EU Commission on Animal Protection for Experimental and Scientific Purposes (86/609/EEC) and with the Portuguese legislation for the same purpose.

LPALF Noise Exposure

The animals were divided into 3 groups: One group of 20 rats (Group 1) was exposed to LPALF noise in an occupationally simulated schedule: 8 h/day, 5 days/week, and weekends in silence. Another group of 5 rats (Group 2) was exposed to 24 h continuous noise. The other group of 10 rats (control) was kept under identical conditions but in a silent environment.

A sound signal was generated by an analog noise generator, amplified and frequency-filtered. Fig. 1 shows the overall linear and A-weighted noise levels, as well as the spectral analysis of the excitation signal collected at a position near the test rat group inside the chamber. This noise was analysed by a B & K 2144 digital real time analyser (Denmark). In this experiment, the sound energy was highly concentrated in the lower frequency bands due to the influence of the low-pass filter. In the frequency bands ranging from 50-500 Hz, noise levels were higher than 90 dB. Overall noise levels were recorded above 109 dB, the A-weighted levels being around 98 dB (A).

Experimental Protocol

Group 1: After 968 to 1984 (median 1576 h) cumulative hours of noise exposure, the animals were sacrificed.

Group 2: The animals were sacrificed after 24 h of continuous noise.

The rats were anesthesized with an intravenous injection of ketamine (Ketalar, Parke-Davis Co., Barcelona, Spain; 4.0-8.0 mg/Kg of weight). The abdominal aorta was cut to exsanguinate the animals, and the vascular system was washed with saline at 37ºC before perfusion fixation with 10% buffered formalin.

Fragments of aorta, inferior vena cava and femoral artery and vein from both hindlimbs were collected. Care was taken to include the supporting muscle in order to prevent vessel "shrinking", in accordance with the guidelines of the American Heart Association (Stary et al., 1992). The fragments were fixed in a 10% buffered formalin, sectioned, and prepared for histological observation using standard methods. The sections were stained with hematoxylin-eosin and Masson trichrome solution.

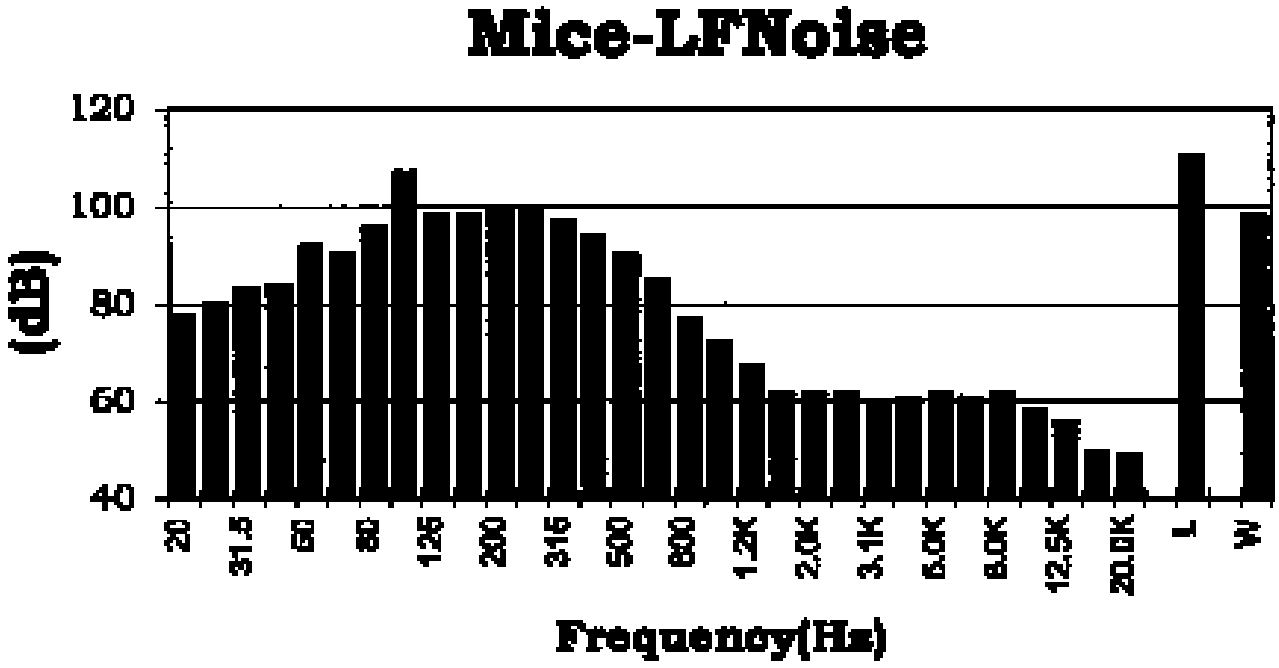

Figure 1 .- Linear (L) and A-weighted (W) noise levels to which the rat populations were exposed and spectral analysis.

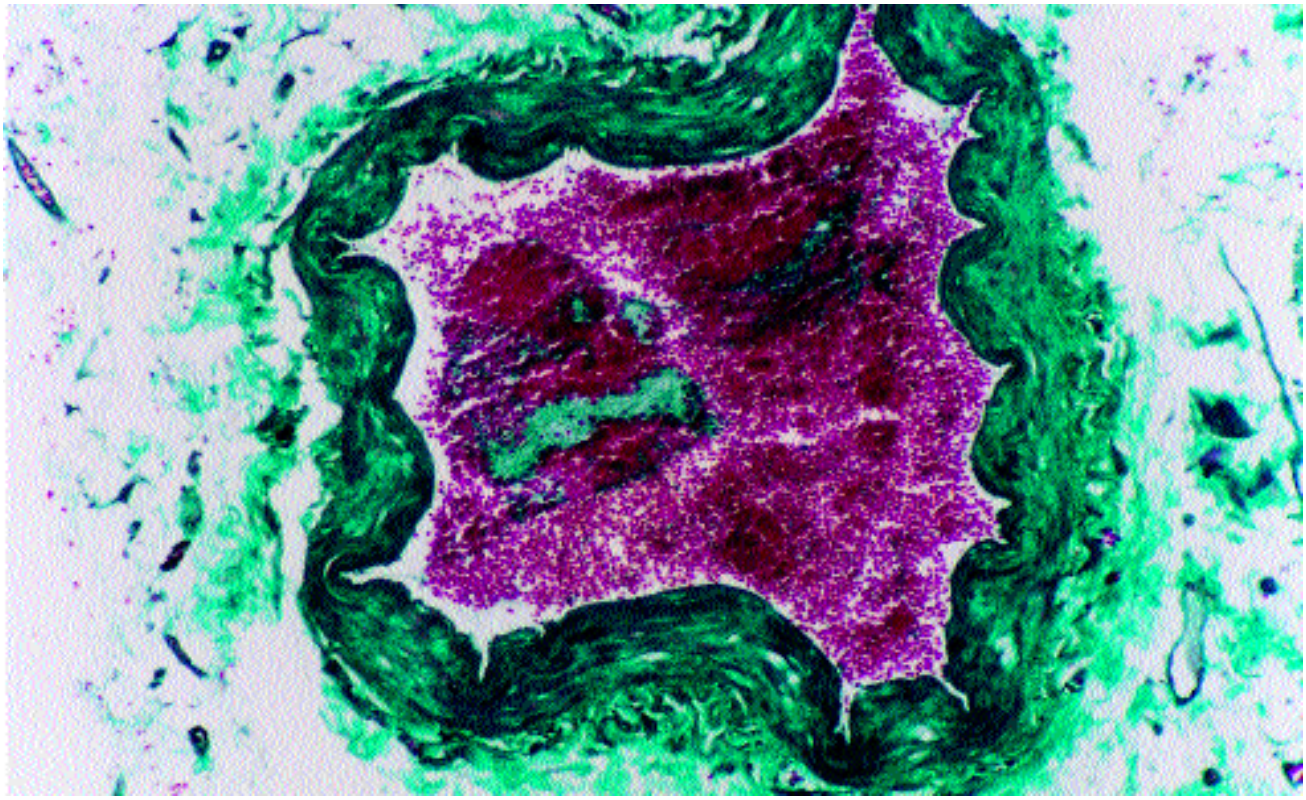

Figure 2. - Femoral artery (Masson trichrome solution). Focal thickening of the intimal layer (long-term exposure). Note the increase in the wall thickness-to-inner caliber ratio, which is significant ($p<0.05$). x 100.

Morphometric and statistical analysis

Data were acquired from the histologic images using a computer image analysis software (OPTIMAS). The calibres of the vessels and the thicknesses of the walls were measured. The results were tested with the non-parametric Mann-Whitney test.

Results

Microscopy

Group 1 (Long-term exposure)

The aorta and the femoral artery showed histologic lesions in 70% of the cases. These alterations consisted of a focal thickening of the intima layer (Fig. 2), disruption of the internal elastic lamina (Fig. 3), and a proliferation of smooth muscle cells in the intima (Fig. 4).

Group 2 (Acute exposure)

The lesions appeared in 60% of the cases observed. Most of the lesions involved the appearance of clefts in the media, pulling the cells and the elastic membranes apart (Fig. 5).

Morphometry

Group 1 (Long-term exposure)

The media thickness-to-inner calibre ratio of the femoral artery was increased in this group with respect to the control group; this increase was significant ($p<0.05$). All the other variables studied showed non-significant differences with the control group.

Group 2 (Acute exposure)

The measurements did not show any significant differences between this group of animals and the controls.

Discussion

In 1984, Takeuchi et al. reported the appearance of intimal thickening in skin biopsies of the fingers of workers who used vibrating tools. The presence of fibrocellular thickening in the small arteries of vibration-exposed rats was reported in 1987 (Okada et al., 1987) after 90 days of vibration exposure. In 1988, Inaba et al. reported alterations of the intima of small arteries with 30 d exposure. Our results revealed a similar pattern in the medium-and large-calibre vessels.

The action of LFN and vibrations on blood vessel walls is direct and, in small blood vessels, causes an increase in permeability and a proliferation of the intimal layer, accompanied by the appearance of fibrocellular thickening (Inaba et al., 1988). This increase in capillary permeability leads to intravascular hemoconcentration and a rise in whole-blood viscosity and microvascular damage (Greenstein and Kester, 1997). LFN causes fibrosis and a proliferation of the collagen fibres and extra-cellular matrices of several tissues, particularly in the lung (Grande et al., 1999a).

In 1999, Grande et al. (1999b) reported the appearance of intimal hyperplasia in the arteries

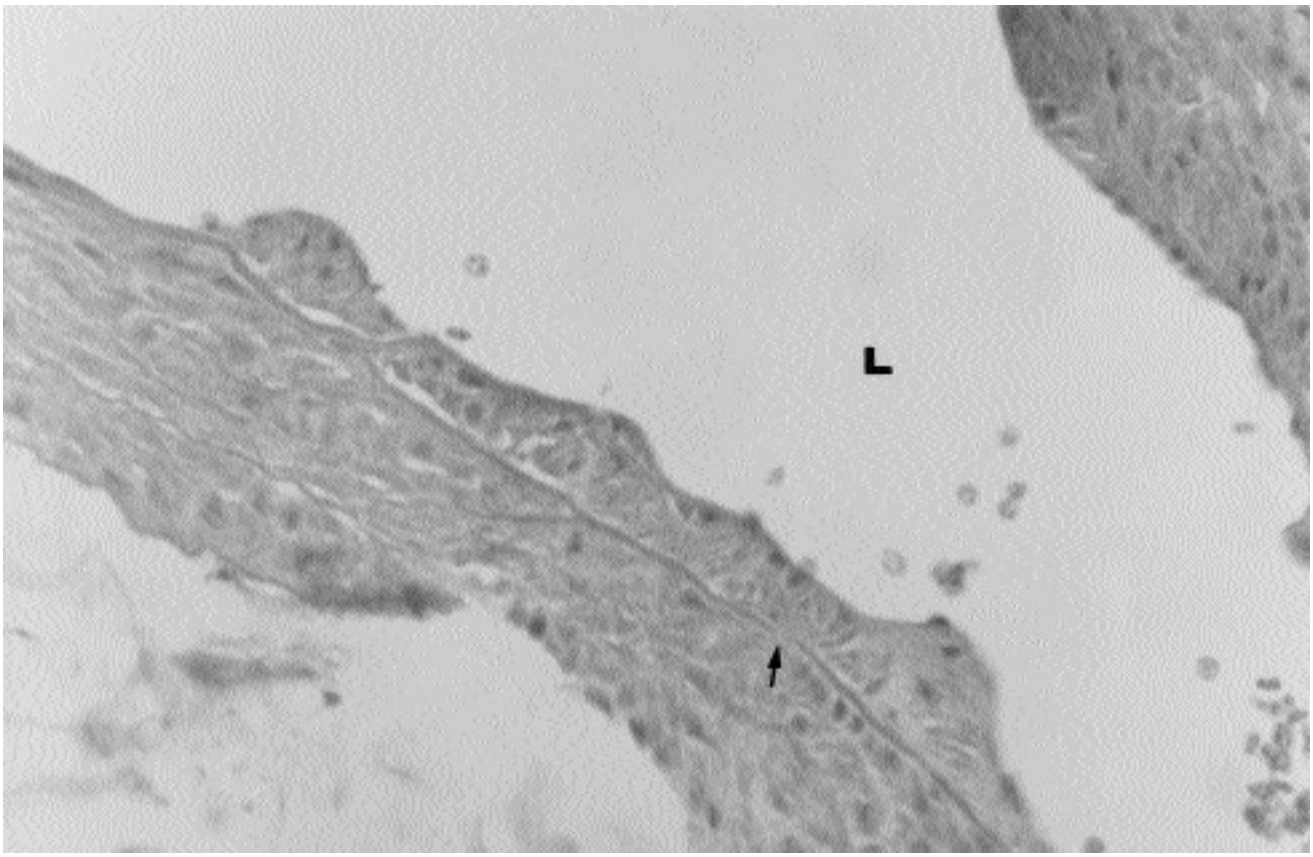

Figure 3.- Femoral artery (hematoxylin-eosin). Disruption of the internal elastic lamina (arrow). Intimal proliferation (long-term exposure). L = Lumen. x 400.

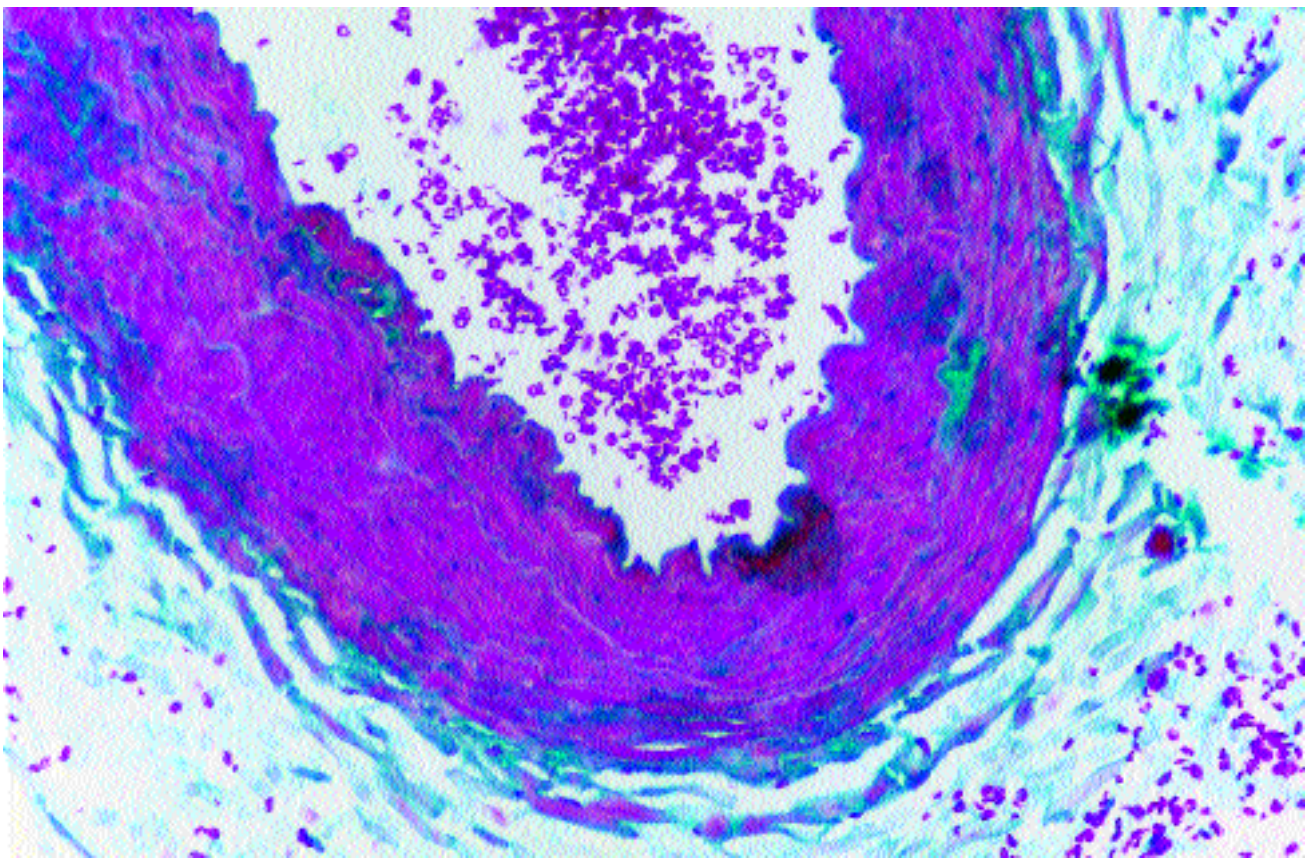

Figure 4.- Femoral artery (Masson trichrome solution). Intimal hyperplasia with proliferation of smooth muscle cells (long-term exposure). x 400.

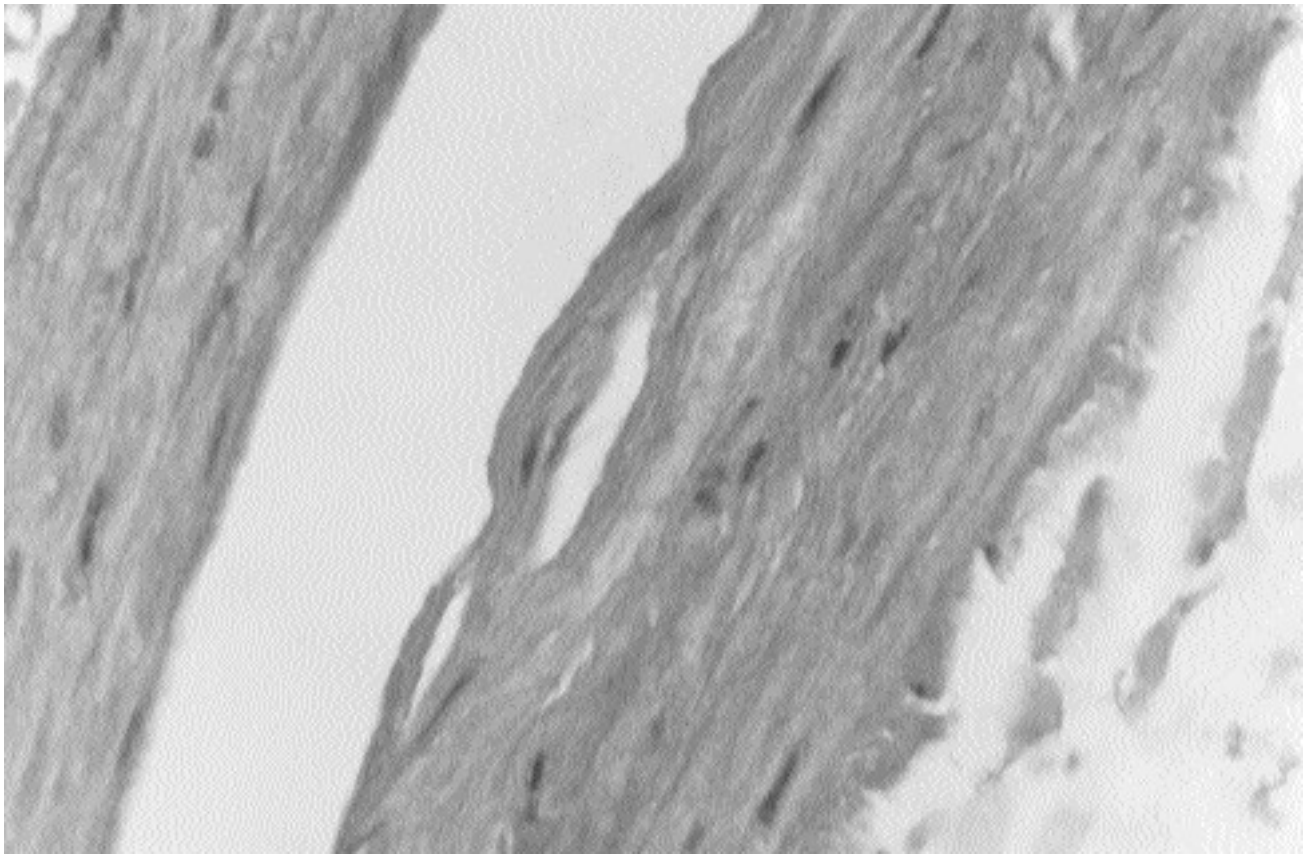

Figure 5. - Femoral artery (hematoxylin-eosin). Clefts in the media layer (short-term exposure). x 400.

of immobilised limbs. They stated that these lesions were caused by the biophysical flow conditions of the immobilised patients.

In the light of the results of the present study, we propose that vibration injures the endothelium, causing a proliferation of intimal smooth muscle cells. It also injures the elastic fibres and membranes, facilitating the migration of media smooth muscle cells to the intima. This intimal proliferation and hyperplasia also lead to flow disturbances that cause further intimal proliferation.

Our results point to a remodelling of the vessels that can be attributed to vibration and to flow disturbances. The observed remodelling was not reported in small vessels (Inaba et al., 1988).

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