

Noise-induced duodenal lesions

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SUMMARY

Vibroacoustic disease (VAD) is a systemic disease that results from long-term exposure to low-frequency noise (LFN). VAD can cause lesions in several organs. Noise-exposed individuals frequently present digestive symptoms, but only a few studies have attempted to evaluate gastrointestinal lesions. The aim of this study was to investigate the duodenal alterations in VAD, using an animal model of the disease. Adult Wistar rats were exposed to continuous LFN. After five, nine and thirteen weeks they were sacrificed. The duodenum was studied by light microscopy and scanning electron microscopy, and compared with those of animals kept in silence. Superficial erosions and widespread cell death with microvilli coalescence and fusion were observed, by light and electron microscopy. Erosion, cellular degeneration and death, and microvilli destruction, reflect noise-induced duodenal alterations in rats which may be equivalent to the ulcers and dyspeptic symptoms reported in human VAD patients.

Key words: Vibroacoustic disease – Low-frequency noise – Duodenal lesions

INTRODUCTION

Long-term exposure to low-frequency noise (LFN), ≤ 500 Hz, is known to cause vibro-

acoustic disease (VAD), a systemic disease associated with exposure to LFN in the workplace (Castelo Branco and Rodriguez, 1999). VAD lesions have been described in several organs. Neurological lesions were among the first to be described (Martinho Pimenta et al., 1991; Maurício et al., 1991; Martinho Pimenta and Castelo Branco, 1999), while cardiac (Castelo Branco et al., 1999; Marciniak et al., 1999), vascular (Albuquerque e Sousa et al., 1991; Martins dos Santos et al., 2002), and lymphatic alterations (Martins dos Santos et al., 2004) have been studied in depth. Respiratory lesions have also been reported (Reis Ferreira et al., 1999; Grande et al., 1999; Sousa Pereira et al., 1999; Oliveira et al., 2002; Castelo Branco et al., 2003). Additionally, the potential genotoxic effects (Silva et al., 1999) and immunological changes induced by LFN (Águas et al., 1999) have been documented.

Although digestive symptoms were present since the first reported cases of VAD (Alves-Pereira, 1999), the paucity of studies on noise-induced digestive lesions is striking. Clinically, gastrointestinal (GI) complaints usually begin after 3 years of professional activity involving exposure to LFN and the presence of duodenal ulcers is a known feature of Stage III disease (Castelo Branco, 1999). Some reports suggest that GI complaints are common among noise-exposed individuals. Cohen reported the frequent occurrence of stomach cramps, nausea, diarrhoea, and heartburn among boiler-plant workers (Cohen, 1979). Kim et al. exposed

healthy volunteers, dogs and rats to the noise of a jet engine. They studied gastric acid output in humans and dogs, and found important variations of secretion. They also exposed laboratory rats to this kind of noise and found a large number of ulcerative gastric lesions (Kim et al., 1968). Anthony et al. identified stomach constrictions in noise-exposed guinea pigs (Anthony et al., 1959).

LFN and whole-body vibration (WBV) are distinct but similar phenomena. Studies on the GI alterations occurring in WBV patients and animal models may be useful to understand the digestive manifestations of VAD. The alterations to gastric motility caused by WBV have been well documented (Kjellberg and Wikstrom, 1987; Ishitake et al., 1998; Ishitake et al., 1999). Bosco et al. (2000) studied the alterations of the neural and hormonal responses in WBV. Nakamura et al. compared the effects of vibration with those of forced water immersion (FWI) in the rat experimental model. They found a large number of gastric erosions in WBV-exposed animals and showed that the vibration-induced gastric lesions were not entirely mediated by the nervous or endocrine systems, indicating a specific role for a direct mechanical effect of vibration (Nakamura et al., 1992). To the best of our knowledge, there are no experimental studies addressing the evaluation of the duodenum exposed to LFN or WBV. The goal of the present study was to investigate the duodenal alterations in VAD, using an experimental animal model of the disease.

MATERIAL AND METHODS

Animals

Adult Wistar rats were divided in four groups: Ten animals of Group 1a were exposed to LFN over 5 weeks (850 hours). Ten animals of Group 1b were exposed to LFN over 9 weeks (1500 hours). Ten animals of Group 1c were exposed to LFN over 13 weeks (2200 hours). Fifteen animals of Group 2 (control) were kept in similar laboratory conditions, but in silence. All animals were fed standard rat chow, had unrestrained access to water, and were treated according to the EU directive on Animal Protection for Experimental and Scientific Purposes.

Low-frequency Noise Exposure – Experimental Protocol

A noise generator produced an amplified and frequency-filtered signal, creating an acoustic environment rich in low-frequency components (Figure 1). Levels were above 90 dB in the frequencies ranging from 50 to 500 Hz.

Microscopy

Rats were sacrificed with a lethal intraperitoneal injection of sodium pentobarbital. Duodenum were removed. After gross observation, they were fixed in 10% buffered formalin, for light microscopy (LM), and in glutaraldehyde solution, for scanning electron microscopy (SEM). Sections were stained with haematoxylin-eosin for LM observation.

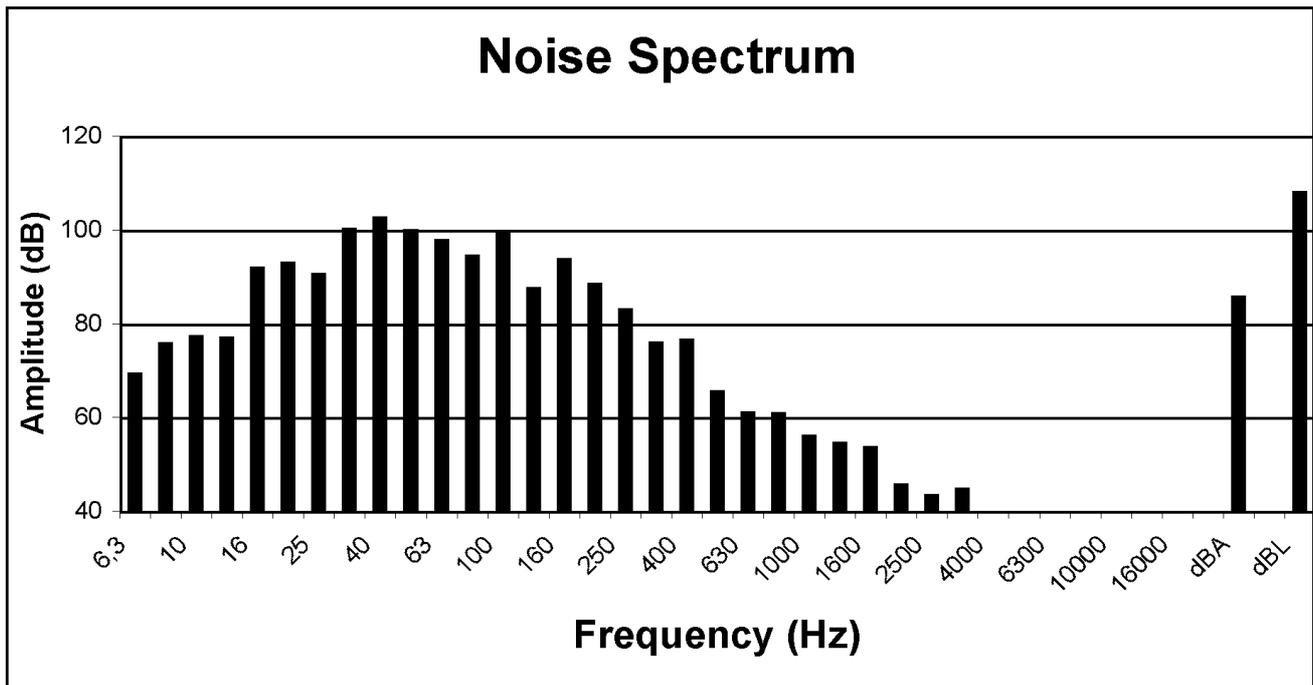


Figure 1.- Frequency distribution of the acoustic environment to which the rats were exposed.

RESULTS

The duodenum of control rats (Group 2) had a normal macroscopic appearance with circular folds. Observed with LM, they had the usual villi, covered with the absorptive epithelial layer. With SEM, the normal duodenal epithelium showed the microvillus tips lining a regular surface (Figure 3a).

Noise-exposed rats (Group 1) showed morphological alterations that were not present in any of the control rats. No macroscopic ulcerations were found upon examination of the duodenal mucosa. LM alterations included superficial erosions seen in the epithelial layer, and evidence of degeneration, pyknosis and cell death (Figure 2). There was evidence of a slight inflammatory infiltrate. No differences between the sexes were identified. These alterations did not change with increasing time of exposure to noise and there were no differences between the three groups 1a, 1b and 1c.

Observed with SEM, the duodenum of the noise-exposed rats displayed epithelial cells dislocated, or even uprooted, from the epithelial lining. The villi had an irregular distribution, with coalescence and fusion (Figure 3b).

DISCUSSION

Since early reported human cases of VAD, clinical GI complaints have been well documented in this disease. The results obtained with models of animals exposed to WBV may be a useful approach to understand lesions in the VAD animal model. Nakamura et al., studied the relationship between the alterations in neural and hormonal responses in WBV. They compared the effects of vibration with those of forced water immersion (FWI) of the animals, a known cause of stress in rats. Both mechanisms produced alterations in gastric blood flow, and in plasma corticosterone and catecholamine levels. Truncal vagotomy did not have any effect on the alterations caused by WBV, indicating a specific role for a direct mechanical effect of vibration on the stomach. Although they did not study the duodenum, they found a large number of gastric lesions in WBV-exposed animals (Nakamura et al., 1992).

In our study, the duodenum of LFN-exposed rats presented significant alterations: namely, superficial erosions and widespread cell death with microvilli destruction, seen both in LM and SEM. The presence of similar aspects in the three noise-exposed groups was not expected, but is

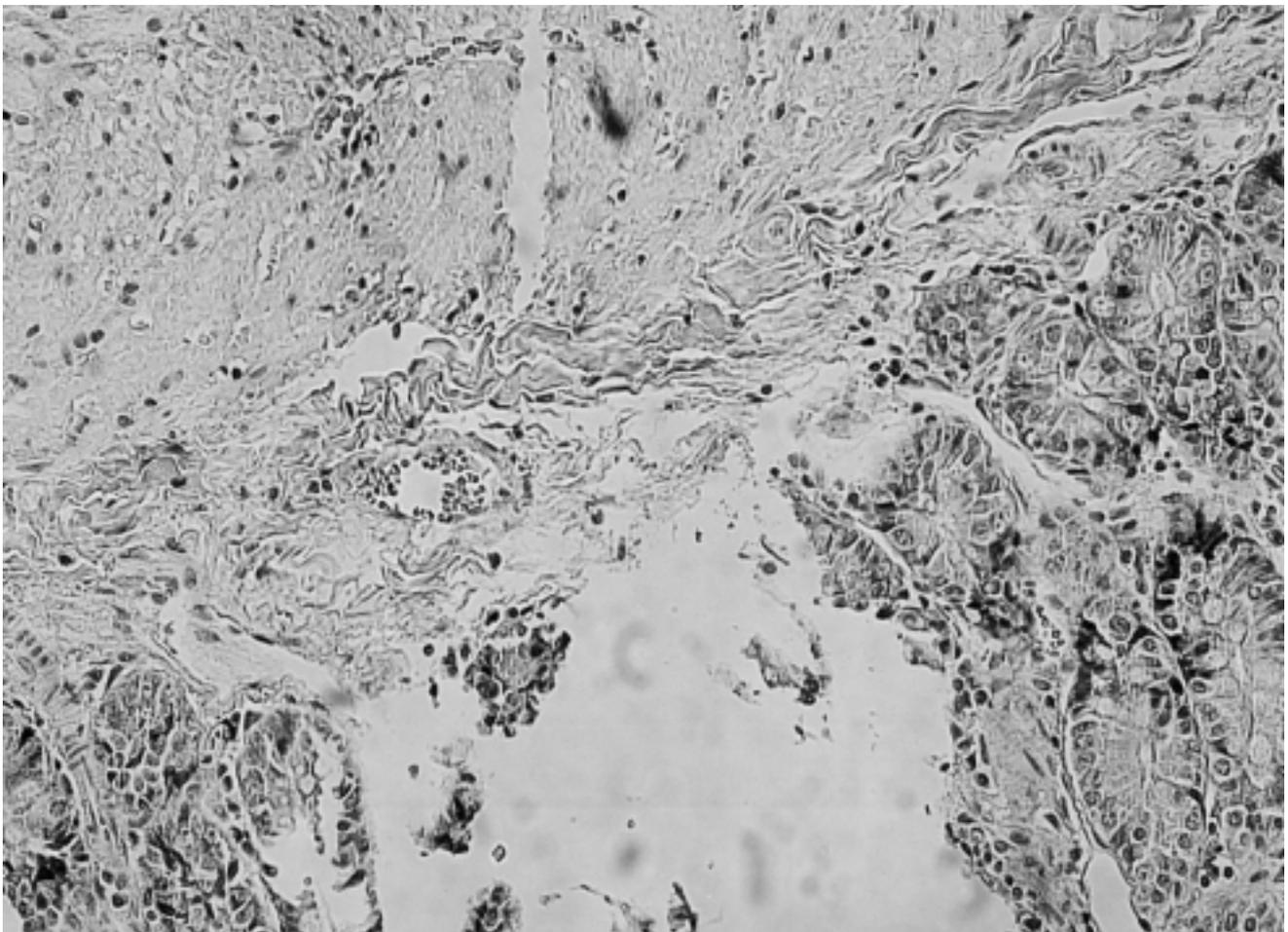


Figure 2.- Light micrograph of superficial erosions of the epithelial layer of noise-exposed duodenum (Haematoxylin-eosin, x 200).

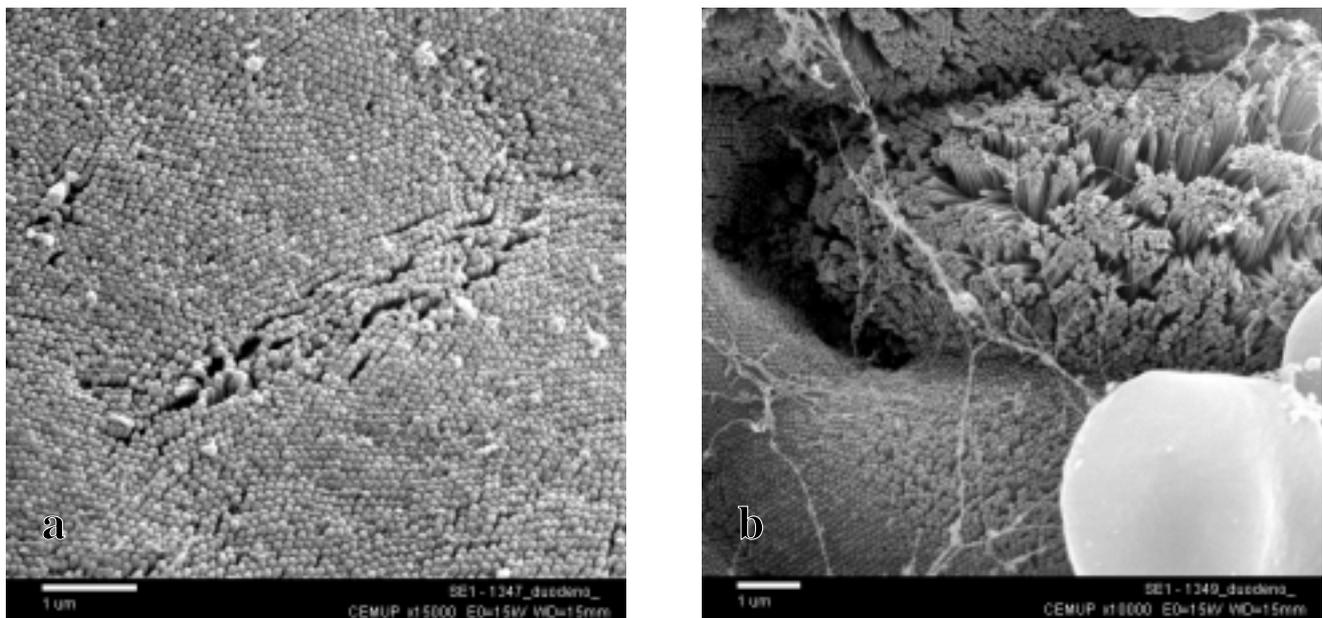


Figure 3.- SEM micrographs of epithelial layer of rat duodenum. Normal aspects from a control rat (figure 3a) and cell death and coalescence of villi observed in a noise-exposed rat (figure 3b).

an understandable feature. Since the erosions are confined to the epithelial layer, and do not penetrate into the submucosa, they are able to heal without any scar. They are certainly induced with less than 850 hours of exposure, are probably formed early in noise-exposed rats, and heal completely, leaving no signs of their previous presence. With continuous exposure to LFN, while some erosions are formed, others heal with no scar, producing a similar appearance in the three exposed groups. These findings encourage further investigations to define the minimum exposure time needed for the appearance of such alterations.

These noise-induced duodenal lesions may be the laboratory rat equivalent to the ulcers and dyspeptic symptoms reported in VAD patients.

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