

DIAGNOSIS OF VIBROACOUSTIC DISEASE – PRELIMINARY REPORT

J. Reis Ferreira¹, C. I. Mendes¹, M. Antunes¹, A. J. Martinho Pimenta²,
E. Monteiro^{3,5}, M. Alves-Pereira^{4,5}, N. A. A. Castelo Branco⁵

¹ Portuguese Air Force Hospital, Lisbon, Portugal; ² Department of Neurophysiology, St. António dos Capuchos Hospital, Lisbon, Portugal; ³ Abel Salazar Institute for Biomedical Sciences, University of Porto, Portugal; ⁴ Dept. of Environmental Sci. & Eng., New University of Lisbon, Caparica, Portugal; ⁵ Center for Human Performance, Alverca, Portugal

Introduction In 1980, this team initiated research into pathology induced by low frequency noise (LFN) (≤ 500 Hz, including infrasound). In 1996, the clinical stages of vibroacoustic disease (VAD) were established [1]. VAD is a whole-body pathology, compromising several organs and systems, and which develops over years of repetitive exposure to LFN. Despite the diversity of ailments associated with VAD, an inexpensive and unique diagnostic tool has yet to be identified for VAD. This report describes the types of reliable diagnostic tools that have been used, and their drawbacks. The latest results concerning a new, and probably definitive, non-invasive, reliable, objective and inexpensive diagnostic tool for VAD will also be discussed.

Chronology In 1980, late-onset epilepsy was found to have been diagnosed among 10% of aircraft technicians, when the expected value was 0.2% [2]. This fact initiated a neuropsychological evaluation of all these workers. Statistically significant (s.s.) memory and attention deficits were identified [3], however the dispersion of values, low accuracy, and individual variance doomed these tests as routine for VAD monitoring. Brainstem evoked potentials demonstrated s.s. delays in nerve conduction for waves III, IV and V [4]. Altered topology and asymmetries in brain potentials were identified using endogenous potentials and brain-mapping [4]. Since all these data suggested that organic brain lesions could be at play, workers were voluntarily submitted to brain MRI which disclosed the existence of lesions in the subcortical and periventricular white substance, basal ganglia and brainstem [4]. These features are common to aging processes, as well as pathological conditions, and are considered a risk factor for cardio-cerebro-vascular disease. Cerebral atrophy and dilation of the Virchow-Robin perivascular spaces were also identified among these workers although these, too, are situations are common to other medical conditions. A possible VAD-specific correlation was identified between the delay in N2 endogenous potentials and the existence of brain lesions. Despite the magnificence of this neurological data, no consistent, inexpensive and readily available diagnostic tool had yet been identified. In 1987, autopsy findings of a deceased VAD patient disclosed that LFN-pathology was not restricted to the neurological realm. Thickening of cardiovascular structures, as well as lung fibrosis and two malignant tumors (brain and kidney) were observed [5]. Consequently, an echocardiography protocol was initiated among the technicians. Pericardial thickening proved to be the most consistent feature in the echocardiograms of these workers although mitral and aortic valve thickening, as well as mitral valve prolapse, were very frequent findings [6]. The anatomical correspondence of the pericardial thickening observed through echo-imaging was subsequently obtained [7]; today, pericardial thickening in the absence of an inflammatory process, and with no diastolic dysfunction is the hallmark of VAD [8]. Finally, it seemed that a possible diagnostic technique was becoming available. In commercial aircraft pilots [9] and in a civilian population exposed

to environmental LFN [10], echocardiography results were consistent: all revealed pericardial thickening in the absence of an inflammatory process and with no diastolic dysfunction. It was the most frequent finding in LFN-exposed individuals, independent of age, and directly related to the amount of cumulative LFN exposure. New problems arose when technician subjectivity began to interfere with the consistency of results. All our echocardiography studies had always been performed by the same medical doctors in cardiology whose specialty was echo-Doppler techniques. Since no standardized method exists in echo-imaging for enhancing the view of the pericardium in order to evaluate its thickness, technician subjectivity is easily introduced. Thus, echocardiography became a weak parameter for reliably monitoring VAD.

Current situation The dramatic involvement of the respiratory system, seen in both LFN-exposed human [5,11] and animal models [12], as well as in the initial stages of VAD [1], taken together with the extensive involvement of the neurological system [3,4], has led this team to question the status of the involuntary reflex response in these patients [13]. In pulmonary functional tests, the $P_{0.1}$ index is a measure of the suction developed at the mouth 0.1 seconds after the start of inspiration. This initial respiratory drive originates in the autonomic (or involuntary) pathway of the neural control of the respiratory function. By rebreathing CO_2 , normal individuals would present a minimum seven-fold increase in the $P_{0.1}(CO_2)$ index when compared to normal $P_{0.1}$. If the neural control of respiration is compromised, then a less-than seven-fold increase would be expected in this index. Evaluations began in late 2002, and are still ongoing, as is the selection of an appropriate control population. Preliminary data on 11 male VAD patients, average age 50.6 years (range: 38-63) had a $P_{0.1}(CO_2)$ index average value of 22.9% (range: 11-38%). The normal value should be around $\geq 70\%$. These data indicate that the autonomic respiratory control in these patients is severely incapacitated. Pending further research, this could be the diagnostic tool for VAD that would not only be non-invasive, reliable, objective and inexpensive, but could also help monitor the progression and follow-up of the disease.

Keywords: MRI, echocardiography, P300, brain potentials, pericardium, tumors, pulmonary functional tests, respiratory drive, low frequency noise, occupational, pathology

References

- [1] Castelo Branco NAA. The clinical stages of vibroacoustic disease. *Aviat Space Environ Med* 1999; 70(3, Suppl): A32-9.
- [2] GIMOGMA. [Epilepsy of vascular etiology, a clinical picture of vibration disease?] *Rev Port Med Mil* 1984; 32: 5-9.
- [3] Gomes L, Martinho Pimenta AJF, et al. Effects of occupational exposure to low frequency noise on cognition. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A115-8.
- [4] Pimenta MG, Martinho Pimenta AJF, et al. ERP P300 and brain magnetic resonance imaging in patients with vibroacoustic disease. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A107-14.
- [5] Castelo Branco NAA. A unique case of vibroacoustic disease. A tribute to an extraordinary patient. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A27-31.
- [6] Marciniak W, Rodriguez E, et al. Echocardiography in 485 aeronautical workers exposed to different noise environments. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A46-53.
- [7] Castelo Branco NAA, Águas AP, et al. The human pericardium in vibroacoustic disease. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A54-62.
- [8] Holt BD. The pericardium. In: Furster V, Wayne Alexander R, Alexander F, eds. *Hurst's The Heart*. 10th ed. New York: McGraw-Hill Professional Publishing, 2000: 2061-82.

- [9] Araujo A, Pais F, et al. Echocardiography in noise-exposed flight crew. *Internoise 2001*, The Hague, Holland 2001: 1007-10.
- [10] Torres R, Tirado G, et al. Vibroacoustic disease induced by long-term exposure to sonic booms. *Internoise 2001*, August 2001, The Hague, Holland: 1095-98.
- [11] Reis Ferreira JM, Couto AR, Jalles-Tavares, et al. Airflow limitations in patients with vibroacoustic disease. *Aviat Space Environ Med* 1999; 70 (3, Suppl): A63-9.
- [12] Castelo Branco NAA, Alves-Pereira M, et al. SEM and TEM study of rat respiratory epithelia exposed to low frequency noise. In: A. Mendez-Vilas (ed). *Science and Technology Education in Microscopy: An Overview*. A. Mendez-Vilas (Ed.), Formatex: Badajoz, Spain, 2002, Vol. II: 505-33.
- [13] Castelo Branco NAA, Martinho Pimenta AJ, et al. Monitoring vibroacoustic disease. *Proc. SSGRR2003w*, January 2003, L'Aquila, Italy: No. 102.