

Neurological Impairment from Hand–Arm Vibration Exposure [†]

Oscar Lundberg ¹, Ing-Liss Bryngelsson ¹ and Per Vihlborg ^{2,*} 

¹ Department of Occupational and Environmental Medicine, Faculty of Medicine and Health, Örebro University, SE 70182 Örebro, Sweden; oscar.lundberg71@gmail.com (O.L.); ing-liss.bryngelsson@regionorebrolan.se (I.-L.B.)

² Department of Geriatrics, Faculty of Medicine and Health, Örebro University, SE 70182 Örebro, Sweden

* Correspondence: per.vihlborg@regionorebrolan.se

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Abstract: The aim of this study is to investigate symptom of neurological impairment from occupational hand-arm vibration using a job exposure matrix. The result shows that paresthesia are significantly higher amongst individuals with a cumulative occupational vibration exposure over 9.08 m/s².

Keywords: hand-arm vibration; neuropathy; cumulative exposure; job exposure matrix

1. Introduction

For more than 100 years, hand–arm vibration (HAV), from vibrating tools, has been reported to cause vibration white fingers (VWF), neurosensory injury and carpal tunnel syndrome (CTS) [1]. The prevalence of vibration-caused complications is difficult to estimate. Approximately 440,000 individuals in Sweden spend at least 25% of their working day using a handheld power tool [2]. This study used a job exposure matrix (JEM), a tool which provides the quantification of exposure through proxy variables of the exposure, such as exposure for specific occupations or length of employment. At a population level, this provides an accurate measurement of the exposure, but is limited by not taking the specific exposure of every individual into account more than the variables provided [3].

The aim of this study is to investigate neurological impairment in relation to HAV exposure, using the JEM.

2. Materials and Methods

The study population consisted of 1623 cases with paresthesia and an equal number of controls for each group. The controls were selected to match by sex, age, and county of residence at the case diagnosis, and were assigned by Statistics Sweden (SCB) and the National Board of Health and Welfare (SoS).

To investigate neurological impairment, we chose the diagnosis paresthesia of skin (R20.2), according to the International Classification of Diseases (ICD 10). The cases in this study were obtained from the National Board of Health and Welfare (SoS). One control for each case was then assigned by Statistics Sweden (SCB), which was selected to match by sex, age, and county of residence at the case diagnosis.

To obtain the amount of HAV exposure for every specific individual, the JEM was used. The JEM uses the employment time and occupation to estimate the individual's HAV exposure. These are through occupational codes which are specific for each occupation. The JEM could also be used to calculate the HAV exposure before the diagnosis for each individual.

Conditional logistic regression was used to calculate the odds ratio (OR) for HAV exposure, for all cases and controls.



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3. Results

There were no significant findings regarding the OR for the prevalence of paresthesia stratified after yearly mean exposure. The OR for paresthesia stratified after cumulative exposure showed significance amongst total participants and men exposed to 9.08+ (m/s²), with an OR of 1.37 (CI 1.04–1.81) and 1.4 (CI 1.02–1.9), respectively (Table 1). Regarding women, there was a low number of those who were exposed.

Table 1. Conditional logistic regression presenting the odds ratio (OR) results for the prevalence of paresthesia of the skin, stratified by the mean yearly daily exposure (A8) and cumulative exposure (m/s²).

Exposure	Gender	Cases	Controls	OR	p-Value	95% CI
Mean Yearly Exposure						
0	Total	1131	1163	1		
0.01–2.5		464	433	1.11	0.20	0.94–1.31
2.5+		28	27	1.08	0.77	0.63–1.84
0	Men	349	375	1		
0.01–2.5		257	234	1.19	0.15	0.94–1.51
2.5+		27	24	1.20	0.51	0.69–2.10
0	Women	782	788	1		
0.01–2.5		207	199	1.85	0.67	0.84–1.31
2.5+		1	3	0.34	0.35	0.04–3.24
Cumulative Exposure						
0	Total	1131	1163	1		
0.01–2.03		180	196	0.94	0.60	0.76–1.17
2.04–9.07		166	148	1.18	0.19	0.92–1.50
9.08+		146	116	1.37	0.03	1.04–1.81
0	Men	349	375	1		
0.01–2.03		58	64	0.96	0.82	0.65–1.41
2.04–9.07		99	94	1.13	0.45	0.82–1.58
9.08+		127	100	1.40	0.04	1.02–1.90
0	Women	782	788	1		
0.01–2.03		122	132	0.94	0.62	0.72–1.22
2.04–9.07		67	54	1.24	0.25	0.86–1.79
9.08+		19	16	1.21	0.59	0.61–2.40

4. Discussion

The main findings of this study are that there was a significantly higher prevalence of paresthesia of the skin amongst all individuals in the group with cumulative work-related HAV exposure of 9.08+ (OR 1.37, 95% CI 1.04–1.81) amongst men (OR 1.4, 95% CI 1.02–1.90). Amongst women, on the other hand, there was not a significant difference in the prevalence.

An explanation of the neurological symptoms developed from HAV is that the repetitive trauma of vibration leads to microscopical tears in the nerves of the hand, and a subsequent fibrosis of the nerve [4]. Our results shows that there was not a significant relation between occupational HAV exposure and yearly mean exposure, which further points toward that it is the cumulative exposure, which is of interest when developing HAV-caused paresthesia of the skin, not the intensity of the exposure, i.e., the yearly mean exposure.

In another study derived from the same study material as this study, but which studied CTS relationship to vibration exposure, it was found that CTS increased with increasing

mean yearly exposure amongst men [5]. The pathophysiology of CTS is described as repetitive traction and compression of the median nerve, leading to obstructed venous flow and edema [6]. An explanation of this is possibly that the pathophysiological events of CTS are more triggered by acute trauma than the demyelination and incomplete nerve generation, causing neuropathy.

In a Finnish study on a similar population, it was found that CTS was more prevalent with increasing cumulative HAV exposure. The study also found that amongst the patients with CTS, the average conduction velocity in the median nerve to the hand was significantly slower [7]. Our findings regarding paresthesia of the skin are very similar to that study, suggesting that this is an adequate theory explaining the neurological damage caused by cumulative occupational HAV.

The observations from the study derived from the same material as this study and the Finnish study regarding CTS and occupational HAV exposure stated different things. One concluded a relationship between CTS and the mean yearly HAV exposure, and one a relationship between CTS and cumulative HAV exposure. An explanation of this would be that CTSs have a multifactorial pathophysiology. The acute effects of vibration, quantified as the mean yearly HAV exposure cause damage to the structures surrounding the median nerve. However, biopsies have shown a connection between long-term HAV exposure and neural edema, suggesting a long-term genesis of CTS, which explains the findings in the Finnish study [7].

The conclusion of this study is that the risk of developing neuropathy increases with increasing cumulative HAV exposure rather than the intensity.

Author Contributions: P.V., I.-L.B. and O.L. researched the literature, conceived the study, and formulated the aims. I.-L.B. developed the protocol and collected the data. P.V. and I.-L.B. applied for ethical approval, funding acquisition and performed data analyses. O.L. wrote the first draft of the manuscript. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: This study is a retrospective register study, where no individual data can be distinguished from the study population. SoS and SCB had anonymized the data before our acquisition. The study was conducted in accordance with the Declaration of Helsinki, and approved by the Swedish Ethical Review Authority (Dnr 2021–03243).

Informed Consent Statement: Not applicable due to the study design with anonymized data before our acquisition.

Data Availability Statement: The data used in this study were derived from patient data access to data. Any researcher, granted that they have an ethical approval from a regional ethical board, can contact the Department of Environmental and Occupational Medicine at Örebro University Hospital (USÖ) for the study data. However, the Swedish National Board of Health and Welfare will also put restrictions on sharing sensitive information.

Conflicts of Interest: The authors declare no conflict of interest.

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