

Vasomotor oscillation in vibration-induced white finger

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PYYKKÖ I, GEMNE G, KOLARI P, STARCK J, ILMARINEN R, AALTO H. Vasomotor oscillation in vibration-induced white finger. *Scand J Work Environ Health* 12 (1986) 389—394. Vasomotor oscillation at different ambient temperatures, with and without anesthesia of the finger nerves, was studied in six persons with vibration-induced white finger (VWF) and in 10 referents. The power spectral density (PSD) of the Fourier transform of the laser-Doppler flowmeter signal from the finger pad vessels was analyzed. In both groups, the PSD of the vasomotor oscillation in the frequency range of 0.05—0.25 Hz was more pronounced in a cold than in a hot environment. Without finger nerve anesthesia, the oscillations of the referents were significantly greater ($p < 0.01$) than those of the VWF subjects (PSD -34.4 dB and -41.6 dB, respectively). With anesthesia of the finger vasomotor nerves, the PSD was reduced in both groups. In the anesthetized finger there was no statistically significant group difference. Thus the myogenic component of the vasomotor activity was the same in the referents as in the VWF subjects. The results show that the autonomic neural influence on vasomotor oscillation in skin of the finger pad is weaker in persons with VWF. This weakness may be a sign of peripheral neuropathy, which may lead to a denervation syndrome causing hypersensitivity of the adrenoceptors to cold.

Key terms: myogenic vasomotor activity, neurogenic vasomotor activity, peripheral neuropathy, Raynaud's phenomenon, smooth muscle tone.

Under certain physiological conditions, an intact and normally innervated blood vessel undergoes spontaneous intermittent contractions, which lead to oscillatory changes in blood flow. Only a few vessels, namely, rat portal vein (5), umbilical vessels (26), and vessels of bat wing (18), have been shown to exhibit spontaneous oscillations in vitro when the neural connections have been severed. Conceivably, these oscillations are regulated by the myogenic pacemaker activity (10). In the majority of vessels, the oscillations are believed to be "neurogenic," ie, controlled by the autonomic nervous system (16). However, Salerud et al (25) could not abolish the oscillation of isolated subcutaneous vessels by anesthetizing the finger nerves. This result shows that spontaneous oscillations can occasionally be of myogenic origin (10), as earlier described by Folkow (4) for muscular vessels.

The frequency and amplitude of the spontaneous oscillations are temperature dependent (18) and are related to wall tension (2, 12). Myogenic oscillations tend to increase when the temperature rises (29), but oscillations driven by the autonomic nervous system are negligible at a high environmental temperature and prominent at moderate or low temperatures (28). In the cutaneous circulation, high amplitudes of oscillations have been observed at skin temperatures of 20—25°C (27). The spontaneous oscillations in the skin show remarkable spatial independence; in one skin area

there can be vasoconstriction and in an adjacent area, vasodilatation (14).

The aim of the present study was to investigate the effect of environmental temperature and finger nerve anesthesia on spontaneous oscillations in the skin vessels of the finger pad of subjects with and without vibration-induced white finger (VWF).

Subjects and methods

Subjects

Six subjects with VWF (five grinders and one stone-worker) with a mean age of 44.3 (range 35—63) years formed the vibration-exposed (VWF) group. Ten healthy volunteers with a mean age of 40.2 (range 28—46) years made up the reference group. Six of the volunteers had been occupationally exposed to cold while working as meatcutters. Four of the referents were laboratory staff personnel.

Methods

The experiments were conducted in a climatic chamber under controlled environmental conditions. Different air temperatures were combined with low water vapor pressure (< 10 mbar or 10^3 Pa) and constant air velocity (< 0.3 m/s). During the test the subjects were supine and dressed in 0.1-clo standard clothing. At the start of the test, the most affected finger was locally heated by running water at 45°C for a 5-min period, during which the finger circulation was arrested with a tourniquet. The vasomotor oscillation was then recorded at a room temperature of 45°C. The chamber

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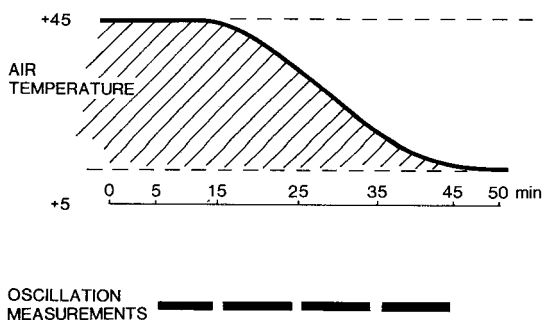


Figure 1. Schematic drawing of the experimental design and the temperature of the climate chamber.

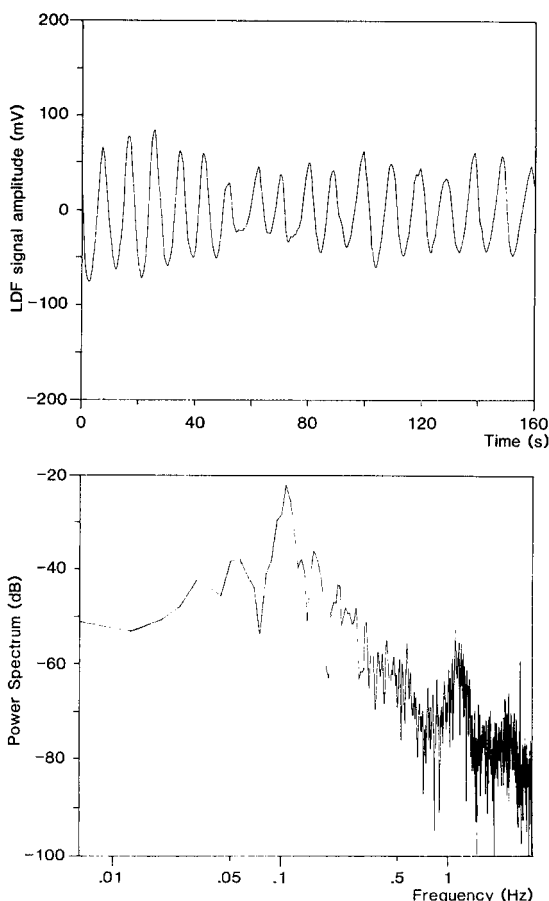


Figure 2. Vasomotor oscillation in a nonanesthetized vessel (upper figure) and the result of the corresponding power spectral density analysis (lower figure).

was subsequently cooled in a regulated manner down to 5°C during a period of 35 min (figure 1), and recordings were made during the intervals 15–25, 26–35, and 36–45 min. The recording was carried out for an average of 7 min during each period.

After this first experimental session, the air temperature was again elevated to 45°C within about 45 min,

the subject being covered with a blanket. Injections of 5 ml of 2 % lidocaine were made bilaterally at the finger base, and the experiment was repeated with complete finger anesthesia.

During the whole experiment, the esophagus temperature (T_e) and the skin temperature at eight body sites were continuously recorded by means of thermistors (Yellow Spring Instruments, models 423 and 427). The finger temperature (T_{fing}) was measured at the end of each recording period by means of an infrared thermometer (Thermophil Infra, T203 and T1063). A modified formula according to Olesen (20) was used for the calculation of the weighted mean skin temperature (T_{sk}). The mean body temperature (T_b) was calculated according to the following formula (17):

$$T_b = 0.67 \times T_{\text{fing}} + 0.33 \times T_{\text{sk}}.$$

For recording the vasomotor oscillation, a laser-Doppler flowmeter (Periflux®, Perimed, Sweden) was used. This system records the blood flux, ie, the product of blood flow velocity and number of red blood corpuscles present in those vessels from which signals are picked up by the probe (a circular area of about 2 × 2 mm, down to a depth of about 1 mm). The probe was placed on the finger pad of the most affected finger. A time constant of 1.5 s and an upper cut-off frequency of 4 Hz was used. The laser-Doppler flowmeter signal was recorded on paper and stored on an instrumental tape recorder (Brüel & Kjaer, Denmark, model 7003) for later analysis.

The recorded time-dependent signal of the laser-Doppler flowmeter was Fourier-transformed to the frequency domain in a signal analyzer (HP 5420A). To perform a constant direct current offset subtraction, the alternating current coupling was approximately 0.2 Hz. During replay, the tape speed was increased tenfold in order to reduce the cut-off frequency to 0.02, which is below the interesting frequency range. The results of the analysis are thus represented with tenfold frequencies. All results, however, are in their natural frequencies. The total average 7-min recording was reduced to 42 s by the increase of the replay speed. Prior to analysis, the signal was visually inspected (at tenfold speed). Recordings without artifacts and transients during < 20 s (ie, < 200 s at the recording speed) were rejected.

The Fourier transform of the autocorrelation function of the signal of the laser-Doppler flowmeter was performed with a frequency resolution of 0.125 Hz and a sample length of 16 s. Up to 10 overlapping samples were analyzed and then averaged, with equal weightings. The length of the overlap of consecutive samples was 15 s. Each sample was analyzed by a Hanning window, the sampling number depending on the length of the artifact- and transient-free recordings. The results of the measurements at four different temperatures were combined to represent the vasomotor oscillation of each subject with and without anesthesia.

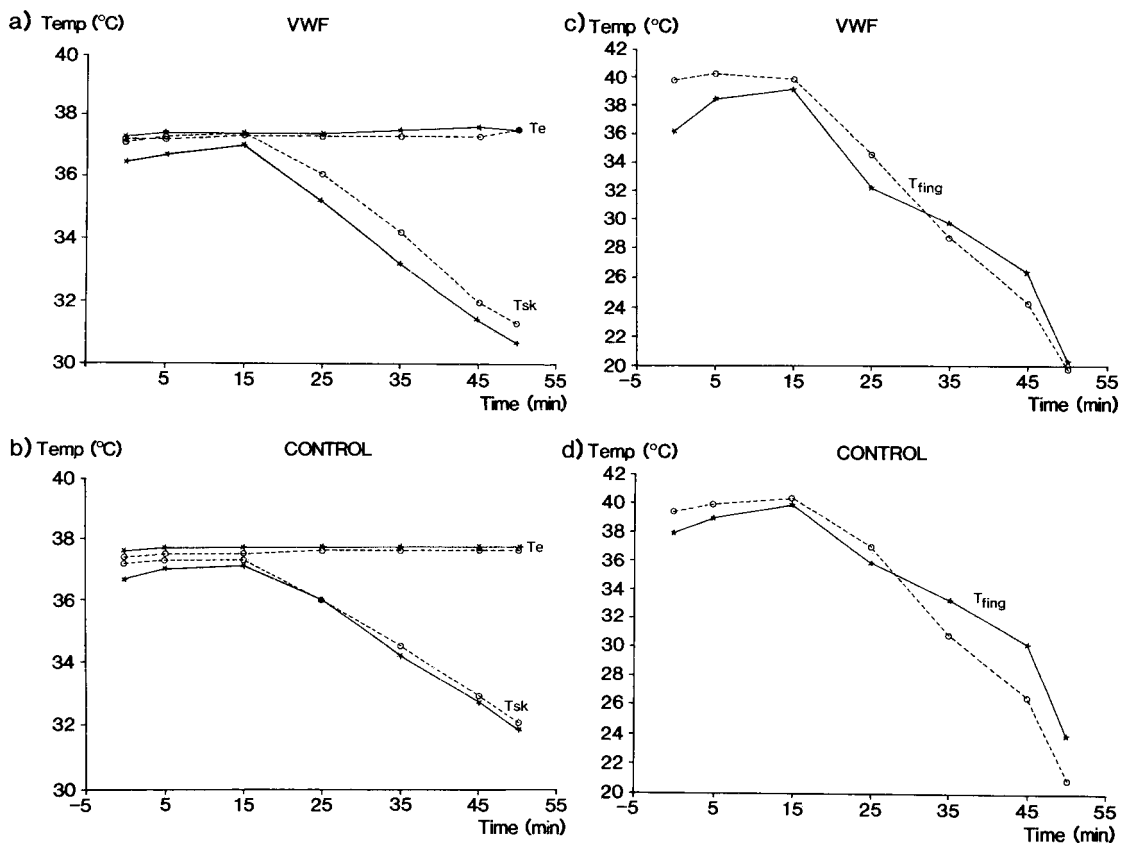


Figure 3. Mean body and esophageal temperature (temp) (a, b) and finger skin temperature (c, d) during the experiment with and without finger vasomotor nerve anesthesia of the referents (controls) and VWF subjects. (Solid lines = without anesthesia, broken lines = with anesthesia, T_e = esophageal temperature, T_{sk} = weighted mean skin temperature, T_{finger} = finger temperature).

The results contained an average amplitude distribution of several samples in the frequency domain. A power spectral density function was therefore calculated in frequency bands between 0.5 and 14 Hz (ten-fold frequencies). For detailed group comparisons with and without anesthesia, the power spectral densities were analyzed in the following three frequency ranges: 0.05–0.25, 0.30–0.50, and 0.60–1.40 Hz. The power spectral density was determined for each frequency range. The difference between the oscillations in the anesthetized and the nonanesthetized fingers was analyzed through a comparison of the surface areas in each frequency range.

Statistical methods

The Mann-Whitney two-sample rank test was used for the statistical evaluation of group median differences. With $p < 0.05$ the difference was considered statistically significant.

Results

The power spectral density of the laser-Doppler flow-meter signal showed that the most pronounced vaso-

motor oscillation occurred in the frequency range of 0.10–0.25 Hz (figure 2). Only in some subjects was the oscillation fixed at a specific frequency, for instance, almost sinusoidal. In most subjects it contained irregular pauses between oscillations with different amplitudes, which resulted in a spreading of peak energy over a larger frequency range.

Effects of ambient temperature

At 45°C the power spectral density of the vasomotor oscillation was smaller than at lower temperatures. The most pronounced power spectral density was obtained in nonanesthetized referents at about 20°C room temperature, with $T_{sk} = 34.6$ (SD 1.2) °C and $T_f = 30.8$ (SD 3.8) °C. The corresponding values for the subjects with VWF at a room temperature of 20°C were 34.2 (SD 1.2) °C and 28.7 (SD 7.4) °C. For all the subjects the T_e changed a maximum of 0.2°C during the test (figure 3).

Effects of anesthesia

After the finger was anesthetized, the power spectral density was reduced in both groups as compared with the values of the nonanesthetized finger. In some sub-

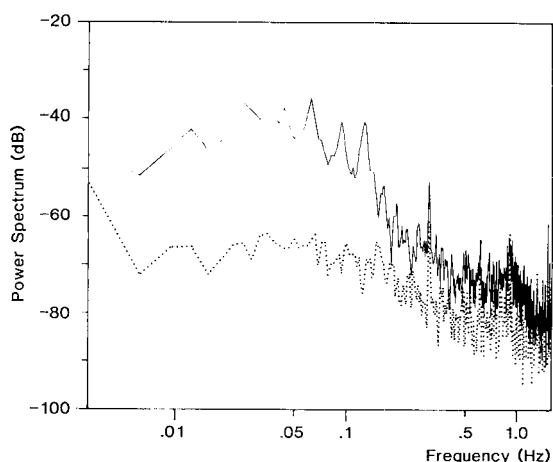


Figure 4. An example of the power spectral density of vasomotor oscillation in a referent with (·····) and without (—) finger anesthesia.

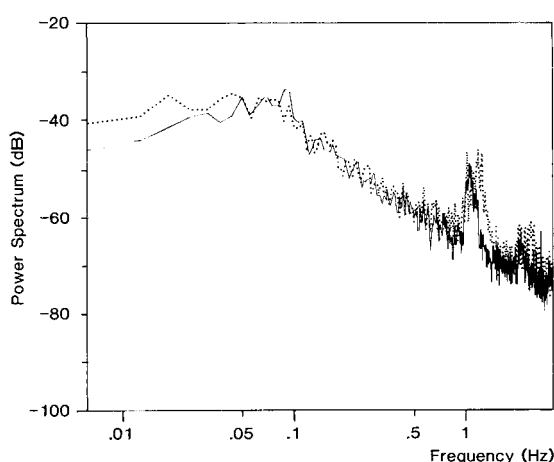


Figure 5. An example of the power spectral density of vasomotor oscillation in a VWF subject with (·····) and without (—) finger anesthesia.

jects the oscillation was totally abolished (figure 4), whereas in others no change was observed (figure 5). In the reference group only one subject did not react to anesthesia with a reduction in the power spectral density, whereas in the VWF group anesthesia had no effect on the power spectral density of three subjects.

In the frequency range of 0.05 to 0.25 Hz, the power spectral density had a mean value of -34.4 dB for the referents and -41.6 dB for the VWF subjects (p of group difference <0.01). Thus without anesthesia the referents had more pronounced vasomotor oscillations than the VWF subjects.

With anesthesia of the vasomotor nerves of the finger, the power spectral density was lower than in non-anesthetized fingers, the reduction being 9.5 dB for the referents and 6.7 dB for the VWF subjects. However, neither the reduction nor the power spectral density level after anesthesia showed a statistically significant group difference. Thus the myogenic component of vasomotor oscillation was the same in both groups. No statistically significant group differences were observed in either the 0.30 – 0.50 Hz or the 0.60 – 1.40 Hz frequency ranges.

Discussion

In the present study the vasomotor oscillation in the finger pad skin was examined in VWF subjects with and without anesthesia of the finger nerves and compared with the corresponding values of referents. The results showed that the power spectral density of vasomotor oscillation was more pronounced in the nonesthetized than in the anesthetized finger and more pronounced in the referents than in the subjects with VWF. Thus, in the finger skin circulation, a large part of the vasomotor tone and oscillation is derived from the outflow from the autonomic nervous system

(neurogenic component). Part of the oscillation, however, is derived from the pacemaker activity of the smooth muscles of the vessel wall, known as the myogenic tone of the vessel.

The neurogenic component of the vasomotor activity was found to be weaker in the VWF subjects; this finding indicates damage to the vasomotor nerves controlling the vessel tone. The results therefore suggest that there is a localized neuropathy in the autonomic nerves controlling the blood flow in the finger vessels of vibration-exposed workers with VWF. Such a local neuropathy may facilitate “spreading” of receptors on the smooth muscles in the same manner as surgical sympathectomy produces hypersensitivity of the vessels to adrenaline (19). Thus, under cold conditions, exaggerated responses may develop (9) and provoke cold hypersensitivity in vibration-exposed workers, the result possibly being Raynaud’s phenomenon (13, 15). Such a hypersensitivity of the vessel would lead to much the same effects as an enhanced sympathetic outflow (21). Different sensory stimuli would then be able to cause an excessive response of the finger circulation (22).

Myogenic and nervous control of vasomotor oscillation

The vessels of the bat wing have been used as a model for studying vasomotor oscillation (18). These arteries exhibit active vasomotion in two distinct ways. First there is a slow caliber change that appears to depend on the tone of the muscular coat. This basic tone seems to regulate the pressure in the capillary network and probably contributes to the mechanism that keeps the vessels partially dilated (10, 11, 14). Second there is a rapid oscillation in the vessels that represents coordinated contraction of the muscle coat. Such a vasomotor oscillation is typically observed following a direct excitation of the nerves to the area (18), and

with injury of these nerves the oscillation is momentarily abolished (18). Thus the medium- and small-sized arteries of the bat wing are undoubtedly controlled by the autonomic nervous system. According to the present results the vasomotor oscillation in the cutaneous finger tissue of man is also mainly regulated by the autonomic nervous system.

The vasomotion in arterioles and precapillary arteries is free from autonomic nervous control (18). The smooth muscles of these vessels possess an inherent capacity to change their tone or contract suddenly in response to intramural pressure changes or to humoral factors (2, 8, 10, 18). Thus these vessels are controlled by myogenic mechanisms (1), although they are not entirely independent of autonomic control.

After finger sympathetic blocking, laser-Doppler flowmetry showed distinct oscillations, indicating that, in the finger vessels, too, there is a myogenic component. The VWF subjects also showed a prominent myogenic component in comparison with the neurogenic component. This phenomenon probably reflects only a decrease in sympathetic control of the vasomotor oscillation. In absolute values, the myogenic tone was the same in the VWF subjects as in the referents. Thus changes in myogenic tone could not explain the increased cold sensitivity of the VWF subjects.

Nature of vasomotor oscillation

In man, vasomotor oscillations in the finger arteries seem to be separate from capillary oscillations (24). The output signal from the laser-Doppler flowmeter corresponds to the integrated velocity and number of red cells in the tissue volume studied. Therefore, the power spectral density of vasomotor oscillation is compounded from movements in small arterioles, capillaries, and arteriovenous anastomoses. Thus the flow oscillation in the present study represents a combination of oscillations in vessels of different sizes and with different control mechanisms. Physiologically, these oscillations are considered important since they assist in the kinetics of capillary exchange (12) and redistribute the blood flow within various capillary networks (3, 7, 8, 23). In isolated vessels an increase in the tone or in the intramural pressure causes a rise in oscillation frequency (2, 8, 10). In general, as the frequency of the oscillations increases, the amplitude decreases (2). Thus the wall tension remains unchanged (12, 16). A consistent rise in finger systolic blood pressure during ambient cooling was observed also in the present study. It is possible that a pressure elevation in itself contributes to the extent of vasomotor oscillation in finger tissue.

Mahler et al (14) conducted pressure measurements during oscillation in the skin capillaries and observed that when the pressure increased the flow would also increase, and vice versa. The changes in flow are well correlated with changes in velocity, as measured by the laser-Doppler flowmeter (6). However, flow is equal

to velocity multiplied by the cross-sectional area through which the particle is moving. If the cross-sectional area of flow remains constant, the velocity measured will therefore be proportional to the flow. The area which is illuminated by the laser is large in comparison with the cross-sectional area of individual capillaries, and the total capillary cross-sectional area within it may not differ very much. Thus the mean velocity would maintain a proportional relationship not only to flow, but also to pressure variation (6). Consistently, the vasomotor oscillations seem to be an effective way of regulating nutritive blood flow in the skin and, by opening and closing arteriovenous shunts, to regulate skin temperature at various ambient temperatures. Allegedly, a loss of neurogenic control leads to disturbances in the flow-pressure regulation and results in improper circulatory changes in the skin, in severe cold as well as in hot ambient temperatures.

Conclusion

The findings in the present work indicate that subjects with VWF have reduced nervous control and relatively enhanced myogenic control of the smooth muscles of the blood vessel walls and therefore suggest damage to the nerves which control smooth muscle contraction. This phenomenon indicates an autonomic neuropathy, presumably accompanied by peripheral localization and limited to areas in contact with vibration.

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