

Circulatory reaction to heat and cold in vibration-induced white finger with and without sympathetic blockade — An experimental study

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GEMNE G, PYYKKÖ I, STARCK J, ILMARINEN R. Circulatory reaction to heat and cold in vibration-induced white finger with and without sympathetic blockade — An experimental study. *Scand J Work Environ Health* 12 (1986) 371–377. Central nervous, sympathetic mechanisms and local factors influencing the vascular response to cold in the finger skin were studied with plethysmographic and laser-Doppler methods in a chamber with controlled temperatures from 45 to about 5°C, with and without sympathetic finger blockade. Blood flow and pressure were assessed after finger occlusion, together with finger heating and cooling, in workers with vibration-induced white finger (VWF) and in healthy persons not exposed to cold or vibration. Finger blood flow at vasodilatation, as measured by finger arterial inflow (FAI) after occlusion was smaller in the VWF group than in the reference subjects both with and without anesthesia, and capillary flux in the anesthetized finger tended to be smaller. At vasoconstriction, with or without anesthesia, the VWF subjects had lower FAI values than the referents. The studied flow and pressure variables differed considerably between the groups. The peripheral resistance of the VWF subjects was higher than that of the referents. This difference was the most pronounced after finger anesthesia. A possible pathogenetic mechanism in VWF is an increase in peripheral resistance due to a local defect in the vessels, with subsequent reduction in flow and intramural pressure.

Key terms: capillary flux, cold exposure, finger blood flow, finger blood pressure, finger plethysmography, finger vasodilation, finger vasoconstriction, hand-arm vibration, laser-Doppler method, peripheral vascular resistance.

The peripheral circulatory disturbance known as vibration-induced white finger (VWF) — a strong and lasting, well-demarcated decoloration of the finger skin together with a decrease in sensibility — gradually develops in some workers exposed long term to hand-arm vibration. Cold is, typically, the triggering stimulus for the attacks, producing a physiologically adequate rise in sympathetic vasomotor tone. This phenomenon results in an arrest of blood flow through finger arteries and arterioles and a shunting of blood through the arteriovenous anastomoses, which abound in the finger skin. The pathogenetic mechanism is not known. Local factors, acting on the smooth muscles of the vessel walls, may be changed gradually during exposure to vibration in such a way that they contribute to the abnormal reaction to cold (6). Such “myogenic” factors may potentiate the sympathetic vasoconstrictor mechanisms (9, 10). Nevertheless, an insufficiency in vasodilator response to cold in the vascular bed — a protective mechanism which serves to uphold nutritive flow — may also contribute to the VWF

attack. The factors mentioned may be involved — alone or in combination — in the pathogenesis of VWF.

The present work was designed to study the relative importance of central nervous and sympathetic mechanisms and local factors for the vascular response to cold in workers with VWF. Healthy subjects not exposed to cold or vibration were used as the reference group.

Subjects

The following subjects participated in the experiments: 14 healthy men (laboratory personnel), aged 23–56 years, without occupational exposure to cold or vibration and without any indication of peripheral circulatory disease (reference group) and 13 men with VWF (nine grinders and four forestry workers), aged 37–53 years, who had been working with hand-held vibrating tools for 12 to 24 years (VWF group).

Methods

Blood flow and pressure assessment

Finger arterial inflow (FAI) after occlusion and finger venous occlusion inflow (FVOI) of blood were recorded (figure 1) with strain-gauge plethysmography (Medimatic®), and finger capillary flux (FCF) in the skin of the finger bulb was measured with the laser-

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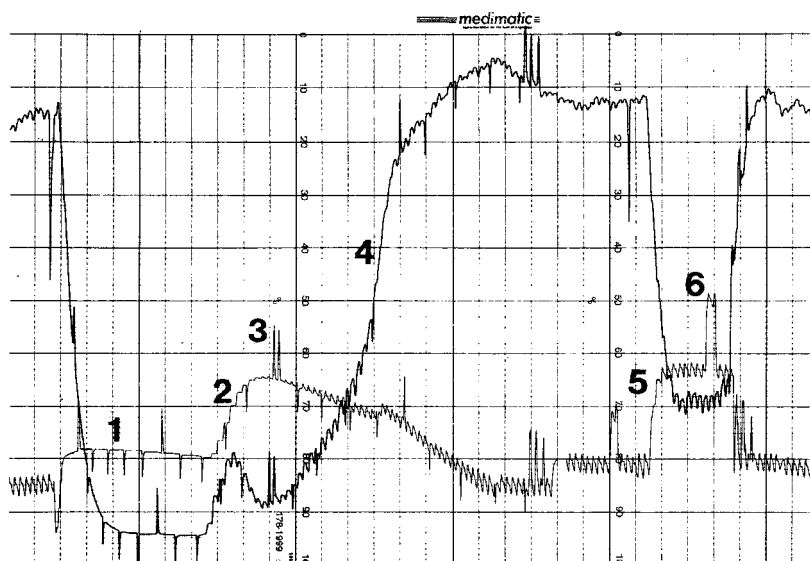


Figure 1. Strain-gauge plethysmography and laser-Doppler signal during the recording of resumed blood flow after arterial occlusion. (1 = strain-gauge plethysmography signal during arterial occlusion, 2 = resumed blood flow after arterial occlusion, 3 = 100 mm Hg (13.3 kPa) blood pressure indication, 4 = laser-Doppler recording of capillary blood flow, 5 = venous occlusion, 6 = calibration).

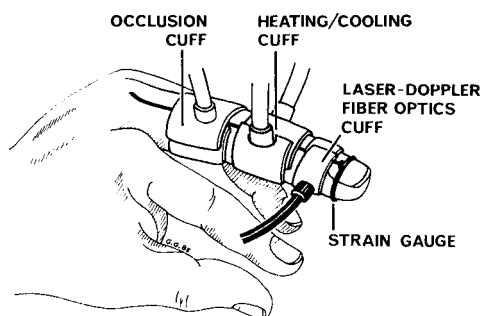


Figure 2. Position of the finger cuffs and detectors.

Doppler technique (Periflux®). The FVOP (finger blood pressure at the peak of FAI), an estimate of venous tone, and finger systolic blood pressure (FSBP) at the start of the FAI were read from the recordings. The finger dynamic pressure range (FDPR), defined as the difference between FSBP and FVOP, was also calculated, together with finger peripheral resistance (FPR), defined as FDPR divided by FAI.

The measurements were performed on one of the fingers afflicted by VWF in the subjects with occupational exposure to vibration and on the third finger of all the other subjects. The experiment was carried out with the subjects in the supine position and wearing standard 0.1-clo clothing in a climate chamber in which the computer-controlled temperature gradually decreased during 30 min from 45°C (maintained for an initial period of 15 min) to about 5°C. The first recording was ideally made immediately after the sub-

ject had entered the chamber and then repeated at 5, 15, 25, 35, 45, and 50 min.

The different phases of the study were as follows: the experiment was begun with local heating of the finger with running water at 45°C for 5 min ("vasodilatation stage") together with blood flow occlusion insured by the inflation of a finger cuff (figure 2) to 250 mm Hg (33.3 kPa). The cuff was then gradually deflated, and the plethysmographic recordings were made. After the end of the postarterial occlusion recording, when the flow had stabilized at its preocclusion level, the cuff was again inflated, now to supra-venous pressure (50–70 mm Hg or 6.7–9.3 kPa), and the venous occlusion plethysmographic recording was made. In an identical way recordings were made at the other points of time mentioned. At 50 min, after a 5-min period of local cooling with running water at 5°C together with occlusion of the blood flow ("vasoconstriction stage"), the finger circulation was reevaluated as described.

The plethysmographic and laser-Doppler signals were constantly recorded on tape (Racal Thermionic®) for later analysis of the blood flow fluctuations (10). Room temperature was read from both wet and dry bulb thermometers. The skin temperature of the finger bulb was measured throughout the experiment with a manually operated infrared sensor device (Thermophil Infra®, models T203 and TT1063). The average skin temperature of the body was assessed with thermistors in eight sites on the body. The esophageal temperature was recorded with a thermistor (Yellow Spring Instrument, model 423) introduced to the level of the right atrium by way of a catheter through one nostril.

This sensor was also used for continuous electrocardiographic recording.

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

Statistical methods

The statistical significance of the differences between the groups and between various phases of the experiment (within groups) was analyzed with the two-sample Wilcoxon rank test. For group mean comparisons of normally distributed variables the t-test was used.

Results

Effects of general and local heating and cooling on flow variables

Vasodilatation stage. At the beginning of the experiment (before any prolonged general warming of the body had taken place), there were no significant group differences in any flow variable (table 1). After local heating and occlusion of the *unanesthetized* finger (values at 5 min), the referents tended to have larger FAI and FVOI means than the VWF group, but the differences were not statistically significant. The FCF was virtually the same in both groups. With *anesthesia*, the flow variables showed statistically significantly lower median values for FAI and, particularly, for

Table 1. Blood flow and pressure variables of the subjects with vibration-induced white finger (VWF) after general and finger heating (vasodilatation stage) and cooling (vasoconstriction), without and with anesthesia of the finger nerve.

Variable	Vasodilatation			Vasoconstriction		
	N	Mean	SD	N	Mean	SD
Finger systolic blood pressure (FSBP) (mm Hg ^a)						
VWF group						
Without anesthesia	13	113	14	13	112	51
With anesthesia	13	89	17	10	138	25
Reference group						
Without anesthesia	14	116	15	14	134	24
With anesthesia	14	95	20	14	135	19
Finger venous opening pressure (FVOP) (mm Hg ^a)						
VWF group						
Without anesthesia	13	73	9	8	61	23
With anesthesia	12	56	13	11	87	26
Reference group						
Without anesthesia	14	79	13	14	64	21
With anesthesia	14	56	20	14	90	22
Finger arterial inflow (FAI) (ml·min ⁻¹ ·100 ml ⁻¹)						
VWF group						
Without anesthesia	13	23	11	13	2	2
With anesthesia	13	9	7	10	5	5
Reference group						
Without anesthesia	14	30	19	14	6	4
With anesthesia	14	24	22	14	17	11
Finger venous occlusion inflow (FVOI) (ml·min ⁻¹ ·100 ml tissue ⁻¹)						
VWF group						
Without anesthesia	13	33	15	12	7	6
With anesthesia	11	22	11	13	18	16
Reference group						
Without anesthesia	11	41	22	13	10	6
With anesthesia	13	42	25	13	25	17
Finger peripheral resistance (FPR) (units)						
VWF group						
Without anesthesia	13	2.1	1.3	8	36.9	49.0
With anesthesia	12	9.0	12.4	8	20.0	24.5
Reference group						
Without anesthesia	14	1.8	1.3	14	27.1	35.0
With anesthesia	14	3.3	3.0	14	5.9	9.4

^a 1 mm Hg = 133.3 Pa.

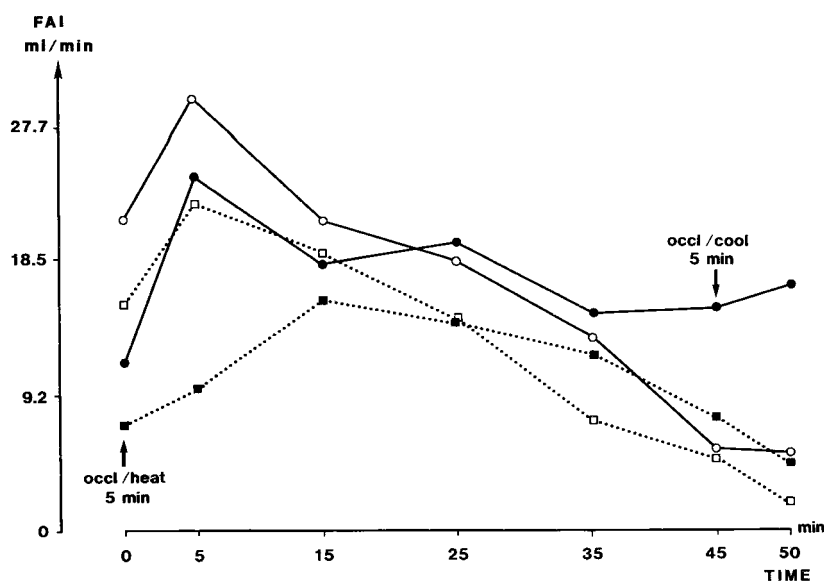


Figure 3. Mean of the finger arterial inflow (FAI) values of the referents without (○) and with (●) finger nerve anesthesia and of the subjects with VWF without (□) and with (■) finger nerve anesthesia during the course of the test. (occl = occlusion, heat = heating, cool = cooling)

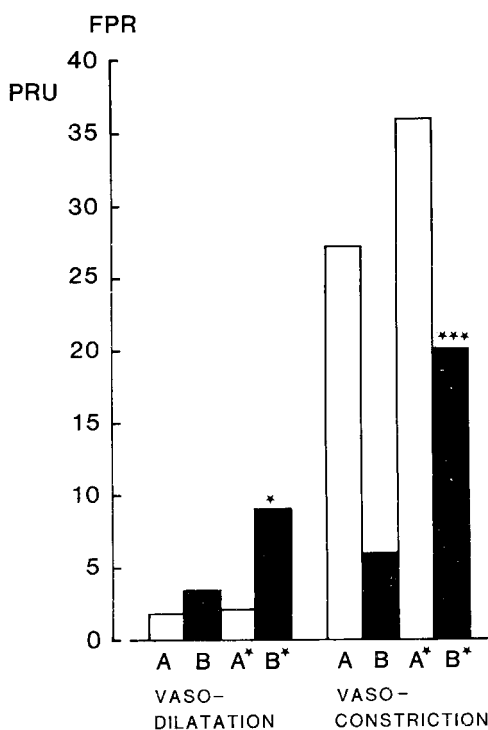


Figure 4. Finger peripheral resistance (FPR) in the referents without (A) and with (B) finger nerve anesthesia and in subjects with VWF without (A*) and with (B*) finger nerve anesthesia. The stars above the bars indicate significant differences from the referents under respective measurement conditions (* $p < 0.05$, *** $p < 0.001$). (PRU = arbitrary peripheral resistance unit.)

FVOI — but not for FCF — in the VWF subjects than in the referents (figure 3).

Vasoconstriction stage. At low room temperatures, prior to local cooling and occlusion, no (statistically significant) flow differences were observed between the groups (table 1). In the *unanesthetized* finger, local cooling and occlusion (values at 50 min) induced a large and statistically highly significant reduction in all flow variables in both groups in comparison with the values at vasodilation. In comparison with the referents the VWF subjects had a significantly lower FAI median after the cooling. The FVOI and FCF were the same in the VWF subjects as in the referents. Also in the *anesthetized* finger, cooling induced flow reduction in comparison with the means in the vasodilation stage. For the FAI and the FVOI, the referents had higher group median values at vasoconstriction than the VWF subjects. This difference attained statistical significance only for the FAI.

Effects of anesthesia on flow variables

In the anesthetized finger of the *referents*, the FAI value in the vasodilation stage was lower than without anesthesia, but the difference was not statistically significant. The means of the other flow variables were the same as in the unanesthetized finger. In the *VWF subjects*, however, both FAI and FVOI were considerably lower with than without anesthesia ($p < 0.01$), and there also was a tendency towards lower FCF values that almost reached statistical significance.

There were some group differences for FAI (but not for FVOI and FCF) at vasodilatation in the anesthetized finger. Thus the referents had higher FAI and FVOI medians than the VWF subjects, the differences being statistically significant. In the anesthetized finger of the referents, as well as of the VWF subjects, the FAI, FVOI, and FCF values of the vasoconstriction stage were much higher than without anesthesia.

Effects of heating and cooling on pressure variables

Vasodilatation stage. The pressure variables (FSBP, finger blood pressure at the start, and FVOP, at the point where the increase of blood inflow after occlusion ended) showed virtually the same values at the beginning of the experiment in the two groups, both with and without anesthesia. After local heating and occlusion of the unanesthetized, as well as the anesthetized, finger, there were no FSBP or FVOP group differences.

Vasoconstriction stage. Immediately prior to local cooling there were no group differences between the pressure variables, either with or without anesthesia. There were no statistically significant group differences for either FSBP or FVOP at vasoconstriction, with or without anesthesia. After cooling of the *unanesthetized* finger of the referents, the FSBP had considerably higher and the FVOP considerably lower median values than at vasodilatation (with statistical significance). In the VWF subjects, both the FSBP and FVOP were the same after cooling as after heating. After cooling of the *anesthetized* finger in both groups, the FSBP and the FVOP were considerably higher than after heating. There were no group differences at either stage.

Dynamic pressure range

Effects of heating. For the referents the median FDPR value of the anesthetized finger at vasodilatation was the same as without anesthesia, whereas for the VWF subjects it was lower ($p < 0.02$). This difference in reaction to heating occurred due to a relatively larger FSBP decrease and a relatively smaller FVOP decrease in the VWF subjects than in the referents.

A comparison of the FDPR mean values after heating between the *unanesthetized* and the *anesthetized* fingers showed that the FDPR decreased 16 % in the VWF group but increased 1 % in the reference group. Further analysis revealed the following reason for this difference: the proportion of FVOP in relation to FSBP decreased only 3 % (from 65 to 62 %) in the VWF group but decreased 10 % (from 70 to 60 %) in the reference group.

Effects of cooling. After cooling of the *unanesthetized* finger, the FDPR was higher than after heating in both groups ($p < 0.01$), with no group mean difference at 50 min. Further analysis showed that the higher values at vasoconstriction were due to a higher level of FSBP than at vasodilatation, and a lower level of FVOP. The proportion of FVOP in relation to FSBP after the cooling was 47 % for the referents as compared with 68 % after the heating. The corresponding figures of the VWF subjects were 42 and 65 %.

Also in the *anesthetized* finger, the FDPR had essentially the same value in the two groups after the cooling. The reaction of the VWF subjects to the cooling differed, however, from that of the other group in one essential respect. The *change* in FDPR from the vasodilatation to the vasoconstriction stages in this group was larger (an increase of 42 % as compared with 17 % in the reference group). This group difference in reaction occurred because the increase in FSBP was larger and the increase in FVOP smaller in the VWF group.

Peripheral resistance

In both groups, the effect of general and local cooling of the unanesthetized finger was a pronounced increase in the mean FPR (table 1). In the *referents*, the FPR of the finger with sympathetic blocking was approximately the same after cooling as after heating, whereas for the *VWF subjects* the mean value at vasoconstriction was considerably higher than at vasodilatation (20.0 as compared with 9.0, $p < 0.01$).

Vasodilatation. After heating *without finger anesthesia*, the mean FPR was low in both groups (1.8 for the referents and 2.1 for the VWF subjects) (figure 4). In the anesthetized finger, the FPR was higher in the VWF group than in the reference group (mean value 9.0 and 3.3, respectively). This difference, however, did not attain statistical significance due to the large variance in the VWF group.

Vasoconstriction. After cooling, the mean FPR of the *unanesthetized* finger was high in both groups, with no statistically significant group difference. In the *anesthetized* finger, the mean FPR of the VWF subjects was considerably higher than that of the referents (20.0 and 5.9, respectively), the difference showing strong statistical significance ($p < 0.01$).

Discussion

The hemodynamic changes leading to vasospastic attacks in persons with VWF are not known. Both local

and central mechanisms have been hypothesized to produce the attack. (For a review, see reference 8.) The role of the sympathetic nervous system in provoking the attacks seems to be established. The outflow in the sympathetic vasoconstrictor nerves is targeted to the resistance arterioles, precapillary sphincters, and arteriovenous anastomoses (1). A slight decrease in the radius of the vessel causes a pronounced increase in peripheral resistance and a reduction in the flow, provided that the pressure does not change. Thus, changes in radius provides an efficient way to control skin circulation.

Olsen et al (7) showed that, if the sympathetic nervous outflow to the fingers of VWF patients is blocked with local anesthetics, the response of the vessels to cold is normal, whereas in unanesthetized fingers local cooling leads to an exaggerated response. This increase in sensitivity to sympathetic outflow during cooling may, however, depend on defects in the function of adrenergic receptors (3), centrally mediated increased sympathetic outflow (4, 11), or structural changes in the vessel walls (2, 14). Cooling of the smooth muscles of the vessel walls does not in itself produce a white-finger attack.

A generally higher sympathetic tone in the VWF subjects than in the referents of the present study seems possible in view of the overall smaller values of some flow variables in the former group. However, the results also indicate that local factors manifesting themselves in the form of enhanced peripheral resistance are responsible for the more pronounced cold reaction of the VWF subjects.

In careful studies, a poor relationship was observed between the vasomotor oscillation in nailfold capillaries and that in finger arteries (12). The blood flow of the capillaries is presumably regulated by local mechanisms. In a healthy subject, local mechanisms operating on the vessel wall of the resistance arterioles and the capillary beds significantly contribute to vasoconstriction during cooling (15). In the present work, the FVOI after local cooling was about 2.5 times larger in the sympathetically blocked finger than in the unanesthetized finger.

By its dual action of increasing the central sympathetic outflow and affecting the peripheral receptors (5, 15), local cooling is a particularly effective vasoconstrictive stimulus. If the "local defect" produced in the vessel wall by vibration exposure also involves the receptor-mediated sensitivity to cold, then this normal physiological reaction is further potentiated in VWF subjects.

In the VWF subjects of the present study, significantly smaller flow values at vasodilatation were observed also in the anesthetized finger. It is unlikely that an otherwise successful blockade of the sympathetic nerves would leave enough vasomotor tone to explain this difference, which instead points to a mechanism distinct from the central nervous regulation of blood flow. It is highly conceivable that this mechanism is

local, operating in the periphery. The changes in FDPR in subjects with VWF were also different from those of the referents, with a prevailing increase in FDPR during finger anesthesia. The results indicate that vibration exposure may produce a local defect in the vessel wall, leading to the following sequence of events in reaction to local cooling: a more powerful constriction of the vessels, an increase in peripheral resistance, a reduction of flow, an initial increase in intramural pressure followed by a decrease in intramural pressure when the vasospastic attack is touched off.

In the subjects with VWF of the present study, the FDPR was augmented, and subsequent local cooling of the finger potentiated this response. In three VWF subjects, zero pressure was recorded in the vasoconstriction phase. These recordings represented a "laboratory attack" of Raynaud's phenomenon, presumably reflecting a collapse of intraluminal pressure occurring at the point of "critical opening pressure" described by Roddie & Shephard (13). In contrast to the "clinical" attacks experienced by VWF subjects, this condition lasted only some seconds before blood flow was restored.

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