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Duration of acute exposures to vibration and finger circulation

by Massimo Bovenzi, MD,¹ Christopher J Lindsell, BEng,² Michael J Griffin, PhD²

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Objectives This study investigated changes in finger circulation after different durations of exposure to hand-transmitted vibration.

Methods Finger skin temperature (FST), finger blood flow (FBF), and finger systolic blood pressure (FSBP) were measured in the middle fingers of both hands of 10 healthy men. Finger vascular resistance was also estimated. The right hand was exposed for 7.5, 15, and 30 minutes (static load 10 N) to 125-Hz vibration (root-mean-square acceleration 87 m/s²). Static load only was used as a control. Finger circulation was measured before the vibration and static load exposure and at fixed intervals during exposure and a 45-minute recovery period.

Results No significant changes were found with the static load. The FST and FSBP did not change significantly during vibration exposure, whereas vibration produced significant reductions in FBF and increases in vascular resistance at each duration when compared with preexposure and contralateral (non-vibrated) finger values. Temporary vasodilation occurred in the vibrated finger immediately after each vibration exposure. Recovery was complete for FBF and vascular resistance after the 7.5-minute vibration, whereas a progressive FBF reduction occurred in both the vibrated and the nonvibrated fingers after 15- and 30-minute exposure. The longer the duration of vibration exposure, the stronger the vasoconstriction in the vibrated finger during recovery.

Conclusions Vasoregulatory mechanisms mediated by both intrinsic (local) and extrinsic (neural or endocrine) control systems seem to be related to digital circulatory changes during 125-Hz vibration. It is concluded that, not only the frequency and magnitude of vibration, but also its duration contributes to the reaction of the digital vessels to acute vibration.

Key terms exposure duration, finger blood flow, hand-transmitted vibration, vasoregulatory mechanisms, vibration-induced white finger.

Current standards for hand-transmitted vibration assume that vibration magnitude, frequency, and duration represent the major explanatory variables for predicting the occurrence of adverse health effects from vibration in occupational groups using hand-held tools (1, 2). Annexes to the standards offer some information on the possible relationship between vibration exposure and the prevalence of vascular disorders in the fingers of vibration-exposed workers. These vascular disorders are characterized by symptoms of finger blanching and represent a secondary form of Raynaud's phenomenon, commonly called vibration-induced white finger (VWF) (3).

In the current standards, daily vibration exposure is expressed as 8-hour energy-equivalent frequency-weighted root-mean-square (rms) acceleration, a measure of vibration dose which assumes an inverse relation between daily exposure

duration and the square of the frequency-weighted magnitude of the vibration (2). A "second power" time dependency is convenient for instrumentation and measurement procedures and is commonly assumed in rms averaging methods. However, there is a shortage of both epidemiologic and experimental data to establish that such an "energy-equivalent" time dependency reflects human response to vibration of differing daily exposure durations (4).

Several studies have investigated the circulatory changes that occur in either experimental animals or in human hands during exposure to vibration with different frequencies and acceleration magnitudes (5—9). Surprisingly, there is limited knowledge on the influence of exposure duration on vibration-induced acute changes in peripheral circulation (10). The objective of our study was to investigate the changes in fin-

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ger blood flow, finger systolic blood pressure, and vascular resistance caused by varying the duration of exposure to hand-transmitted vibration. The circulatory effects of vibration were monitored in both vibrated and nonvibrated fingers.

Subjects and methods

Subjects

Ten healthy male volunteers, aged 21 to 45 (mean 29) years, participated in the study. None of the subjects were known to have any significant (regular or prolonged) exposure to hand-transmitted vibration in occupational or leisure activities. They filled out a health questionnaire, read a list of medical contraindications, and gave their written informed consent to the study. None of them reported neurological or cardiovascular disorders, connective-tissue diseases, injuries to the upper extremities, or a family history of Raynaud's phenomenon. All were nonsmokers. The mean height of the subjects was 180 (SD 6.0) cm and their mean weight was 76.1 (SD 10.0) kg. The finger length and the finger breadth and depth at the intermediate phalanx of the middle fingers were measured with vernier callipers to an accuracy of 0.5 mm. The volumes and surface areas of the fingers were then calculated (11). Each subject was requested not to consume coffee or alcohol for at least 2 hours before being tested. The study was approved by the Human Experimentation Safety and Ethics Committee of the Institute of Sound and Vibration Research.

Measurement of the finger circulation

Finger blood flow (FBF) and finger systolic blood pressure (FSBP) were measured in the middle finger of both hands by a strain gauge plethysmographic technique. Measurement methods for FBF and FSBP have been reported in more detail in previous papers (12, 13). Briefly, mercury-in-silastic strain gauges were placed at the base of the fingernails, and cuffs made of polyvinyl chloride plastic (2.4×9 cm) were fixed around the proximal phalanges and secured by a Velcro® strip. The soft plastic tubes of the digital cuffs were connected to the pneumatic system of the plethysmograph (Digitmatic 2000, Medimatic A/S, Copenhagen, Denmark).

The FBF measures were obtained with a venous occlusion technique. After calibration of the strain gauge, the pneumatic cuffs were instantaneously inflated to a pressure of 40–60 mm Hg (5.3–8.0 kPa), and the rise in fingertip volume was detected by the strain gauge. The FBF was obtained from the plethysmographic tracings according to the criteria of Greenfield et al (14). Three to 5 plethysmographic recordings of arterial inflow were made for each measurement, and the mean value was calculated.

The FSBP was measured according to the technique described by Hirai et al (15). The tips of the middle fingers were squeezed and then the pneumatic cuffs were inflated to a suprasystolic pressure of 250 mm Hg (33.3 kPa). The pressure in the digital cuffs was then automatically reduced

(3 mm Hg/s at 100 mm Hg or 0.4 kPa/s at 13.3 kPa). The FSBP was defined as the cuff pressure at which the first increase in fingertip volume was detected by the strain gauge.

In agreement with the method used by other researchers, the resistance of the finger circulation was estimated by dividing digital blood pressure by digital blood flow (16).

The systolic and diastolic pressures (millimeters of mercury) in the upper arm were measured at the beginning of each experimental session by an auscultatory technique using a standard rubber cuff (12×23 cm).

Finger skin temperature (FST) was measured using k-type thermocouples attached by adhesive tape to the dorsal surface of the midphalanges of the same fingers in which FBF and FSBP were measured. The thermocouples were connected to a HVLab® thermal esthesiometer so as to measure temperature to an accuracy of $\pm 0.2^\circ\text{C}$. The room temperature was measured by a mercury in glass thermometer to an accuracy of $\pm 0.1^\circ\text{C}$.

Experimental protocol

The investigation was conducted in a laboratory with a mean air temperature of 25.8 (SD 0.2) $^\circ\text{C}$. The subjects wore light clothing and lay supine on an examination couch, their hands positioned over their chest, just above the level of the heart. When the FST was stable and above 32°C , after about 20 minutes of acclimatization, the FBF and FSBP were measured in the middle fingers of both hands. After the base-line measurements were obtained, and while the subjects remained supine, the subjects were asked to place their right (exposed) hand palm downward on a wooden surface (100 mm \times 100 mm) secured to the table of a Derritron VP4® electrodynamic vibrator. All 5 fingers of the exposed hand were in contact with the wooden surface, but the left (unexposed) hand was positioned palm downward on a wooden table at a similar height and just above the level of the heart. Visual feedback through an analogue meter allowed the subjects to maintain a constant downward force of 10 N with the right hand. The arrangement for generating vibration and controlling contact force has been described in more detail in a previous paper (12). Sinusoidal vibration was produced in the vertical direction at a frequency of 125 Hz and an rms acceleration of 87 m/s^2 (unweighted). This value corresponds to a frequency-weighted rms acceleration (a_{hw}) of 11 m/s^2 according to the international standard ISO 5349 and the British standard BS 6842 (1, 2). The subjects were exposed to this magnitude of vibration for 7.5, 15, and 30 minutes on different days. With the use of the daily time dependency for hand-transmitted vibration assumed in current standards, such durations of exposure to a weighted rms acceleration of 11 m/s^2 give 8-hour equivalent exposures [A(8)] of 1.4, 1.9, and 2.8 m/s^2 , respectively. The dose-effect relationship included in an annex to BS 6842 predicts a 10% prevalence of VWF after about 15, 11, and 8 years, respectively, of daily exposure to the equivalent weighted acceleration magnitudes used in this study (2).

The FBF, FSBP, and FST were measured before, during, and after vibration exposure in both the vibrated (right) and the nonvibrated (left) middle fingers. The measures of finger circulation were commenced at regular time intervals: at 0.5, 3.25, and 7.5 minutes and then at every 7.5 minutes during exposure to vibration. The flow measurements took about 1 minute. Measures were taken at similar intervals during a recovery period of 45 minutes.

A control condition consisted of the same procedure, with the vibration magnitude reduced to zero but the contact force on the wooden surface maintained at 10 N for 30 minutes.

The exposure (vibration) and control (static load) conditions were presented randomly in 4 separate experimental sessions with 1 to 3 days between the exposures. Each measurement session lasted between 1.5 and 2 hours.

Statistical methods

Data analysis was performed using the software packages BMDP/Dynamic (release 7.0) and Stata (version 5.0). The data were summarized with the mean as a measure of central tendency and the standard deviation or standard error of the mean as the measures of dispersion. The difference between 2 means was tested by a paired or unpaired Student *t*-test when appropriate. The relation between 2 continuous variables was assessed by the method of the least squares. The Pearson product moment correlation coefficient was also calculated. Methods for repeated measures data analysis were used to test the hypothesis of no difference in the vascular responses under different exposure conditions ("treatments"). To handle repeated measures data that were, by design, unbalanced, the generalized estimating equation (GEE) approach to longitudinal data sets (17) was applied to estimate the treatment effect and the treatment-by-time interaction (Stata program *xtgee*). When the treatment-by-time interaction term was found to be significant ($P < 0.05$), a separate analysis of the results within treatments was made, and traditional analysis of variance (ANOVA) techniques for repeated measures data were applied. To control for the effect of covariates on the response variables, repeated measures analysis of covariance (ANCOVA) was also used. When the assumption of compound symmetry for the orthogonal polynomial components of repeated measures was violated, a conservative test of the repeated measures factor was used by reducing the degrees of freedom of the *F* ratio (Greenhouse-Geisser method). The 95% Bonferroni confidence intervals for pairwise mean comparisons of the response by time were used when the probability value for the *F* test of repeated measures ANOVA was $P < 0.05$ (two-sided).

Results

Vascular measurements before exposure

The air temperature in the laboratory did not vary significantly across the 4 experimental sessions (range 25.2—26.8°C).

The brachial systolic and diastolic arterial pressures measured before the exposure did not change significantly for the subjects across sessions (range of mean values 119/70—124/75 mm Hg or 15.9/9.3—16.5/10.0 kPa). There was no significant difference in the FBF, FSBP, and FST of either the exposed or the unexposed finger of the individual subjects when measured before exposure to the static load and before exposure to vibration. No differences in the preexposure measures of digital circulation were found between the middle right and left fingers. The FST values of both fingers correlated positively with room temperature ($P < 0.01$). For both fingers, the FBF was marginally related to room temperature ($P = 0.10$) and FST ($P = 0.15$). No significant association was found between FBF and FSBP. Neither age nor the dimensions of the middle fingers correlated with the base-line measurements of digital circulation.

Finger circulation during vibration exposure

After control for age, finger dimensions, and room temperature, no significant differences were found between the exposure to static load only and the various vibration stimuli for either the FST or the systolic blood pressure measured in either the exposed or the unexposed finger (results not shown).

The GEE approach to repeated measures data revealed significant treatment-by-time interactions for both the measures of FBF and the estimates of vascular resistance ($P < 0.001$). Therefore, the analysis of the results was performed for each exposure condition separately (tables 1 and 2). Figure 1 shows the mean FBF values measured for both the exposed and unexposed fingers before, during, and after the exposure to static load and vibration with different durations.

Exposure to static load caused no significant changes in FBF or vascular resistance with respect to the base-line measures in either the exposed or the unexposed finger. Exposures to 7.5, 15, and 30 minutes of vibration with a frequency of 125 Hz and an acceleration of 87 m/s² rms provoked significant changes in FBF and vascular resistance in the vibrated finger ($P < 0.0001$). Significant changes were also observed for the nonvibrated left finger during exposure of the right hand to 15- and 30-minute vibration ($0.001 < P < 0.02$). It is notable that, in the exposed finger, the decrease in FBF and the increase in vascular resistance during the first 15 minutes of vibration stimulation were very similar when compared with the measurements obtained in the different vibration exposures.

Over the different periods of vibration stimulation, the exposed finger showed reductions in blood flow and increases in vascular resistance which were significant when compared with the measurements taken before exposure (Bonferroni method, $P < 0.05$). Furthermore, the percentage changes in FBF and vascular resistance in the vibrated finger were greater than those observed in the unexposed finger ($0.001 < P < 0.05$). During the 30-min vibration exposure an

Table 1. Finger blood flow [ml (100 ml/min)] measured for 10 healthy subjects before, during, and after 30-minute exposure to static load (contact force 10 N) and 7.5-, 15-, and 30-minute exposure to vibration with a frequency of 125 Hz and an acceleration of 87 m/s² rms. The P-values of the repeated measures analysis of covariance (ANCOVA) have been adjusted by age, finger volume, and room temperature. (SEM = standard error of the mean, % = percentages of values before exposure)

	Static load			7.5-min vibration			15-min vibration			30-min vibration														
	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger												
	Mean	SEM	%	Mean	SEM	%	Mean	SEM	%	Mean	SEM	%												
Before exposure	17.4	1.5	100	17.7	2.0	100	17.3	2.1	100	19.8	1.8	100	17.7	1.9	100	17.9	1.9	100	19.3	2.2	100	19.8	1.8	100
Exposure																								
0.5 min	16.1	1.6	91.1	19.0	1.5	109	6.8	1.4	40.1	20.9	1.7	106***	8.5	2.1	44.3	17.8	2.1	98.4***	8.4	1.8	40.6	18.4	1.9	95.4***
3.25 min	15.7	1.0	92.7	18.4	2.1	105	7.2	1.4	44.7	19.6	2.0	99.3***	7.1	1.5	38.2	14.6	1.1	86.5**	6.0	1.1	31.5	17.2	1.9	91.5**
7.5 min	15.7	1.0	92.6	18.2	1.7	105	7.6	1.1	47.2	17.5	2.0	88.8***	6.4	1.7	34.9	15.6	1.4	94.5**	7.2	0.9	39.5	15.8	1.7	81.3**
15 min	16.0	1.4	93.3	18.7	1.9	106	6.4	1.1	36.6	14.1	1.3	83.7**	6.0	0.7	37.4	17.7	1.6	92.3***
22.5 min	15.2	1.4	90.2	18.9	1.9	108	9.7	1.5	56.0	17.3	2.1	89.0***
30 min	17.8	1.3	104	18.3	1.7	105	12.1	1.4	70.4	18.5	1.9	94.7***
Recovery																								
0.5 min	18.7	1.7	107	19.1	1.8	109	24.0	1.5	135	21.2	2.2	107	20.3	2.8	118	17.7	1.3	102	28.0	3.7	151	20.5	2.7	103*
3.25 min	18.9	1.4	109	17.8	1.4	102	17.3	1.6	105	21.1	2.2	107	16.5	2.3	99.2	17.5	1.4	104	22.7	3.3	120	21.5	2.7	110
7.5 min	18.4	1.4	107	19.5	1.8	111	17.0	1.3	100	21.0	1.9	106	14.4	1.6	85.7	16.1	1.5	93.6	16.9	1.4	101	18.4	1.8	95.7
15 min	18.2	1.5	105	17.7	1.8	103	17.5	1.9	105	20.5	2.2	102	12.5	1.6	75.1	14.1	1.6	87.4	9.9	1.0	59.4	15.2	1.8	82.6*
22.5 min	18.0	1.9	103	19.6	2.1	113	16.1	1.8	95.9	19.7	2.1	98.1	9.2	1.1	53.8	11.1	1.2	65.5	7.0	1.0	39.8	12.5	1.4	66.4***
30 min	17.4	1.6	101	18.7	1.9	108	15.7	1.7	93.9	21.4	2.4	106*	8.3	1.0	48.3	10.3	1.0	63.0	5.9	0.9	34.7	9.8	1.6	51.7**
37.5 min	18.1	1.5	104	18.7	1.9	104	15.7	1.5	97.4	21.0	3.1	104	10.4	1.9	61.2	10.7	1.4	65.2	4.7	0.8	25.6	9.1	1.7	47.4**
45 min	17.6	1.6	101	16.5	2.1	92.0	16.2	1.4	99.5	20.6	2.7	101	9.9	1.6	59.3	12.4	1.6	71.9*	4.5	0.7	24.8	8.8	1.6	46.3**
ANCOVA (P-value)	0.25			0.20			< 0.0001			0.12			< 0.0001			< 0.001			< 0.0001			< 0.001		

* P < 0.05; ** P < 0.01; *** P < 0.001; paired t-test (exposed versus unexposed finger).

Table 2. Finger vascular resistance [mmHg (min/100 ml)] estimated for 10 healthy subjects before, during, and after 30-minute exposure to static load (contact force 10 N) and 7.5-, 15-, and 30-minute exposure to vibration with a frequency of 125 Hz and an acceleration of 87 m/s² rms. The P-values of the repeated measures analysis of covariance (ANCOVA) have been adjusted by age, finger volume, and room temperature. (SEM = standard error of the mean, % = percentages of values before exposure)

	Static load			7.5-min vibration			15-min vibration			30-min vibration														
	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger	Exposed finger		Unexposed finger												
	Mean	SEM	%	Mean	SEM	%	Mean	SEM	%	Mean	SEM	%												
Before exposure	7.7	0.8	100	8.2	1.1	100	8.6	1.1	100	6.5	0.5	100	7.9	0.9	100	7.3	1.0	100	7.4	1.0	100	6.7	1.0	100
Exposure																								
0.5 min	8.9	1.6	112	7.2	0.5	93.2	26.3	4.4	329	6.4	0.5	99.8***	26.5	7.6	304	7.7	0.9	113*	24.8	6.6	333	7.6	1.0	116*
3.25 min	7.9	0.6	108	7.8	0.9	96.8	25.1	4.8	342	7.3	0.7	112**	25.4	4.4	322	9.0	0.8	138**	27.0	8.1	371	10.0	3.1	154*
7.5 min	8.4	0.7	114	7.7	0.8	96.9	20.5	3.0	266	7.9	0.8	124**	23.2	6.6	299	9.0	1.1	144**	19.4	1.9	285	9.3	1.4	139*
15 min	8.3	0.9	110	6.4	0.5	85.0	25.6	4.3	329	9.6	0.7	146**	23.2	2.9	352	7.6	0.8	117***
22.5 min	8.2	0.6	112	6.8	0.7	88.8	16.1	2.6	239	8.4	1.0	127***
30 min	7.1	0.5	95.1	7.6	0.6	99.4	11.7	1.0	176	7.7	1.0	116**
Recovery																								
0.5 min	6.5	0.5	88.3	6.9	0.5	89.7	6.4	0.5	83.5	6.1	0.4	90.3	7.2	1.1	93.4	7.5	0.7	111	5.2	0.6	73.3	6.9	0.6	104*
3.25 min	7.1	0.6	96.4	7.4	0.5	97.2	7.6	0.6	98.9	6.5	0.6	99.8	9.2	1.5	122	7.7	0.8	116	7.0	0.9	98.0	6.7	0.7	103
7.5 min	7.3	0.7	96.3	6.7	0.5	88.8	7.9	0.5	98.8	6.4	0.6	98.4	10.3	1.5	134	8.9	1.2	128	8.3	0.7	125	7.7	0.7	118
15 min	7.3	0.6	97.4	7.8	0.9	99.7	8.3	0.9	99.5	6.8	0.7	104	13.9	3.7	173	11.1	2.3	167	14.9	2.1	232	9.4	1.2	151*
22.5 min	8.0	0.9	105	7.1	0.2	93.2	9.2	1.0	111	7.3	0.7	110	17.1	3.4	214	14.7	3.6	201	23.0	3.7	352	11.9	1.8	190*
30 min	7.8	0.7	105	7.4	0.7	96.1	9.7	1.2	114	6.8	0.7	103*	17.4	2.3	226	13.4	1.6	203	28.4	5.4	451	17.5	4.0	283*
37.5 min	7.6	0.8	100	7.8	1.0	94.5	9.0	1.2	112	6.8	0.7	105	17.2	3.6	222	14.3	2.2	222	35.0	4.4	518	18.7	3.2	300**
45 min	8.1	1.0	106	8.8	1.0	101	8.7	1.0	107	6.9	0.6	106	16.4	2.4	218	11.7	1.4	176	33.9	3.9	484	20.6	4.0	330*
ANCOVA (P-value)	0.24			0.14			< 0.005			0.16			< 0.005			< 0.02			< 0.001			< 0.02		

* P < 0.05; ** P < 0.01; *** P < 0.001; paired t-test (exposed versus unexposed finger).

increase in FBF, even though not significant, was observed in the exposed finger during the second half of the exposure.

Finger circulation after vibration exposure

In both fingers, FST and FSBP did not change significantly during the recovery period after the exposure to either static load only or vibration with different durations (results not shown). Immediately after the vibration ended, the exposed finger showed an increase in blood flow which was greater after exposure to 30-minute vibration than after either 7.5- or

15-minute vibration. Compared with the preexposure measures, the immediate vasodilation after vibration ended was significant only in the finger exposed to 30-minute vibration (P<0.05). The immediate increase in FBF after vibration exposure was significant when compared with the measures of blood flow taken during the corresponding exposure period (Bonferroni method, P<0.05). The left (nonvibrated) finger also showed an immediate, although small, increase in FBF after vibration stopped, but this increase was not significant with respect to any of the blood flow measures obtained in

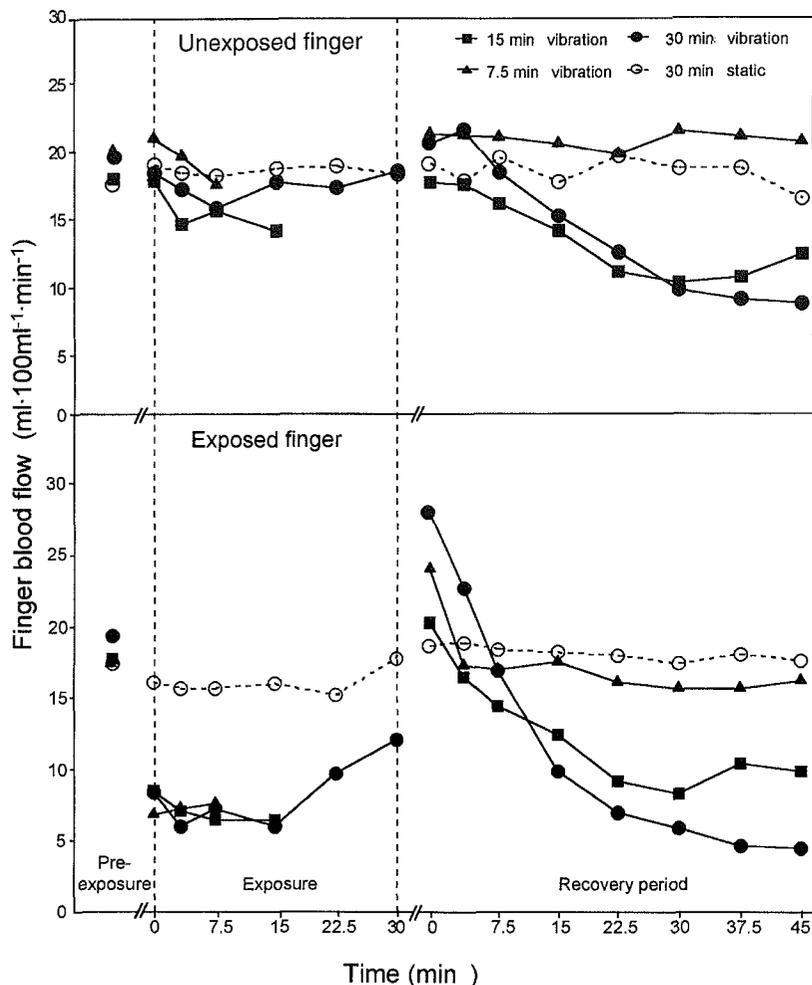


Figure 1. Mean values of the finger blood flow measurements of the exposed and unexposed fingers before, during, and after exposure to static load and vibration.

the same finger during the exposure of the right hand to the corresponding vibration stimuli. Significant differences in FBF and vascular resistance between the exposed and the unexposed finger immediately after vibration ceased were found only after the cessation of the 30-minute vibration ($P < 0.05$).

The exposed and the unexposed fingers showed an FBF and vascular resistance that was equal to the preexposure conditions after 45-minute of recovery after exposure to 7.5-minute vibration. In contrast, both fingers showed a decrease in FBF and an increase in vascular resistance during the recovery period, about 15 to 30 minutes after the end of exposure to either the 15- or 30-minute vibration exposure. The repeated measures ANCOVA revealed that the changes in FBF and vascular resistance during the recovery period differed significantly between the 4 experimental conditions. A multiple comparison test showed that the decrease in FBF and the increase in vascular resistance from 15 to 45 minutes during recovery were significantly greater after 15- and 30-minute vibration than after 7.5-minute vibration or static load only in both the vibrated and the nonvibrated fingers ($P < 0.05$). During recovery, significant differences in FBF

and vascular resistance were found after exposure to 15- and 30-minute vibration only for the vibrated finger ($P < 0.05$). Digital vasoconstriction in the vibrated finger 15 to 45 minutes after the 30-minute vibration exposure was stronger than that in the nonvibrated finger ($0.001 < P < 0.05$).

Possible relations between vibration-induced changes in the digital circulation and the characteristics of vibration exposure were also investigated. Figure 2 shows that the percentage reduction in FBF in the exposed finger at 45 minutes after vibration stopped was inversely related to the digital vasodilation that occurred immediately after the end of the different vibration exposures. The scattergram of data points suggests that the strongest changes in FBF occurred when the finger was exposed to 30-minute vibration.

Discussion

Circulatory effects during vibration exposure

In our study the effects of the duration of vibration exposure on digital circulatory function were assessed using a sinusoi-

dal vibration stimulus with a frequency of 125 Hz. Some previous investigations have led to the conclusion that the vascular response to acute vibration in the human finger is greater at 125 Hz than with the same acceleration magnitude at lower or higher vibration frequencies (5, 7). It was therefore assumed that the use of a vibration frequency of 125 Hz would assist the observation of circulatory changes associated with the duration of vibration exposure. The findings showed that acute exposures to 125-Hz vibration of any duration provoked a reduction in FBF and an increase in the vascular resistance of the vibrated finger, both of which were significant when compared with the preexposure measures. The short latency period for the earliest changes in FBF observed in the vibrated finger at the beginning of all 3 vibration exposures suggests that a neurogenic vasoconstrictor reflex was involved. The intervention of a centrally mediated reflex also seems to be suggested by the consistent trend towards the reduced FBF and increased vascular resistance observed in the unexposed finger during vibration exposure. It has been shown that digital nerve blockade can abolish vibration-induced vasoconstriction within the vibrated and nonvibrated fingers of healthy subjects and in patients affected with either VWF or primary Raynaud's phenomenon (10). Some studies have also reported a significant decrease in the blood flow to the toes during short periods of exposure (1–2 minutes) of the hands to vibration (18, 19). These findings suggest that a central vasomotor mechanism may be involved in the immediate response of finger circulation to vibration exposure.

Immediate postvibration circulatory effects

Our study showed that the response of finger circulation to vibration with a frequency of 125 Hz was influenced by the duration of the vibration exposure. All 3 vibration exposures caused consistent vasoconstriction in the exposed finger during the first 7.5 minutes of exposure. After about 15 minutes, the digital vasoconstriction showed a tendency to reverse, and an increase in FBF was observed during the second half of the exposure to 30-min vibration. It can be argued that the vasoconstrictor effect of prolonged exposure to vibration was locally counteracted by a vasodilation of the vibration-exposed digital vessels in order to prevent an excess reduction of blood flow to the tissues of the finger. The hint of vasodilation observed towards the end of the 30-minute vibration exposure was clearly evident immediately after the end of the exposure when a marked increase in FBF was observed in the exposed finger, significant only after exposure to 30-minute vibration when compared with the FBF in the unexposed finger. This finding is consistent with the results of our previous investigations in which immediate local vasodilation was revealed in the vibrated finger after exposure to vibration with the same frequency and magnitude as used in the present study (12, 13). The mechanism by which vibration can induce a local vasodilatory effect is not yet understood. Ljung & Sivertsson (6) first suggested that vibration could provoke relaxation in the vascular smooth muscle of

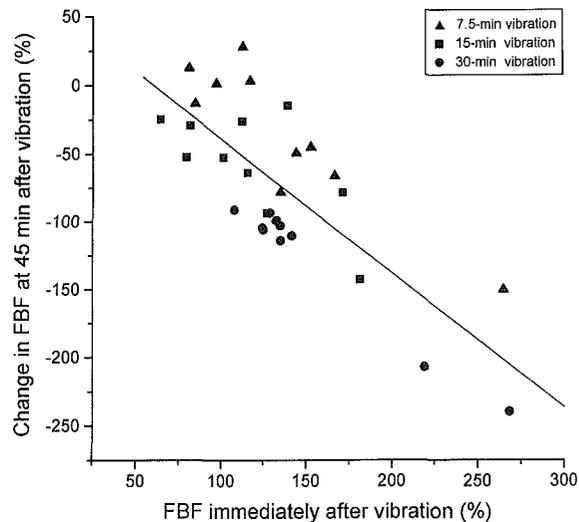


Figure 2. Relation between the change in finger blood flow (FBF) at 45 minutes after the end of the vibration and FBF immediately after 7.5-, 15-, and 30-minute exposure to vibration in the exposed finger. The change in FBF(y) = $[(C-B)/A] \times 100$, where A = FBF before vibration, B = FBF immediately after vibration, C = FBF at 45 minutes after vibration ended. The percentage FBF immediately after vibration (x) = $(B/A) \times 100$. [$y = 59.5 - 0.99(x)$; $R = -0.78$ ($P < 0.001$)] [separate coefficients of correlation: 7.5-min vibration: $R = -0.91$ ($P < 0.001$); 15-min vibration: $r = -0.68$ ($P = 0.03$); 30-min vibration: $r = -0.98$ ($P < 0.001$)]

isolated vein and artery preparations by a direct mechanical inhibition of contractile muscle proteins. This view was also supported by Lindblad et al (8), who found that exposure to vibration of various magnitudes and frequencies was associated with a depression of the contractile processes in canine cutaneous arteries. However, other mechanisms should also be considered to explain the temporary vasodilating effect of vibration. Experimental and clinical studies have shown that the endothelium actively participates in the local regulation of blood flow by producing both relaxing factors (nitric oxide, prostaglandins) and constricting factors (endothelin-1) in response to various chemical and physical stimuli (20). It has been reported that mechanical vibration can induce an excess increase in arterial wall shear stress and therefore cause endothelial damage (21). Since shear stress is considered one of the most powerful stimuli for the release of endothelium-derived relaxing factors, it is reasonable to speculate that local mechanisms of endothelial origin may have a role in the temporary digital vasodilation induced by vibration. Alternatively, it might be thought that the immediate increase in blood flow in the exposed finger after the end of vibration exposure is the result of diminished discharge from the sympathetic vasoconstrictor nerves due to a decreased number of afferent inputs from the cutaneous mechanoreceptors no longer activated by the vibration stimulus. The small, even though not significant, increase in FBF recorded in the unexposed finger immediately after vibration stopped may indicate that digital vasodilation was, to a less extent, determined by a reduction

of the sympathetic vasomotor tone. However, the greater increase in FBF observed in the exposed finger in comparison with that of the control finger suggests that local vasodilatory mechanisms are mainly involved in the immediate postvibration response of the digital vessels.

Vibration after-effects during recovery

In this investigation, we found different durations of vibration exposure to be associated with different patterns for the recovery of FBF and vascular resistance. There was complete recovery to the resting values of FBF and vascular resistance after exposure to 7.5-minute vibration. In contrast, a progressive reduction of FBF was observed in both the vibrated and nonvibrated fingers during the second half of the recovery periods after exposure to both 15- and 30-minute vibration. The hemodynamic changes in both fingers were related to the duration of vibration exposure in that the decrease in FBF and the increase in finger vascular resistance were greater after 30-min vibration than after 15-min vibration. This effect is evident in figure 3 (log-log scales), which shows that the reduction of FBF at the end of the recovery period after the vibration exposures was roughly inversely proportional (slope = -0.94) to the estimated vibration dose ($a_{hw}^2 \times \text{duration}$) received by the exposed hand. A similar, but directly proportional (slope = 0.96), relation was observed between the estimated resistance of the finger circulation and vibration dose ($r = 0.80$, $P < 0.001$).

The occurrence of the delayed recovery to the basal conditions found for both the vibrated and the nonvibrated finger suggests that vasoconstrictor mechanisms linked to either central sympathetic nervous activity or circulating vasopressor agents may be involved in the persisting after-effects

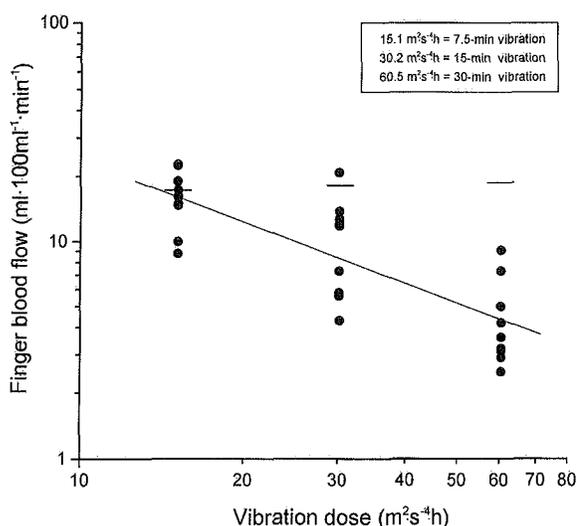


Figure 3. Relation between finger blood flow at 45 minutes after the vibration ended and the estimated vibration dose received in the exposed hand. Horizontal bars represent the mean values of the finger blood flow before the vibration exposure. [$y = 207/x^{0.94}$; $r = -0.80$ ($P < 0.001$)]

caused by vibration exposure. In a study of the cold-induced vasoconstrictor response in the digital arteries of 12 healthy subjects exposed to unilateral vibration with a frequency of 31.5 Hz and a weighted acceleration of 16 m/s² rms, Olsen (9) observed that 30-minute exposures to such vibration caused a significant increase in cold-induced arterial responsiveness in both vibrated and nonvibrated fingers 60 minutes after the end of the vibration exposure. In agreement with the findings of his experimental study, our investigation confirmed the existence of vibration-induced circulatory after-effects in the human finger, and it disclosed a significant relation between such after-effects and some characteristics of the vibration exposure, such as its duration and the derived estimate of vibration dose.

It has been suggested that the persisting vasoconstrictor after-effects of vibration may be due to excess digital arterial contractility mediated through vibration-induced hyperreactivity of the central drive to the sympathetic nervous system (9). This phenomenon may be a plausible explanation for the circulatory after-effects caused by vibration exposure because the after-effects were observed in both the ipsilateral and the contralateral finger. Nevertheless, these findings cannot exclude the possibility that local vasoconstrictor mechanisms may contribute, at least partially, to vibration after-effects. It has been reported that threshold concentrations of the vasoactive peptide endothelin-1 can sensitize the blood vessel wall to the vasoconstrictor effects of substances released from the adrenergic nerves (22). This finding suggests that the vibration after-effects observed by us may be interpreted as the result of a vibration-induced vasoconstrictor response of the digital vessels mediated by the sympathetic nervous system and locally amplified by endothelium-derived contracting factors.

Considerations of a possible exposure-effect relationship

In a previous investigation we found that the effects of acute exposure to vibration on finger circulation depended on the frequency and magnitude of the vibration stimuli (13). It was also observed that the frequency weighting curve of ISO 5349 did not reflect the pattern of the response of the digital vessels to acute vibration. Exposure to 125-Hz vibration was found to cause an increase in the digital vasomotor tone greater than that produced by 31.5 Hz vibration at the same acceleration magnitude. In contrast, the current ISO standard for hand-transmitted vibration suggests a greater effect at lower frequencies than at higher frequencies (1). Our present study extends our previous findings on vibration-induced changes in digital circulation and shows that such changes are also dependent on the duration of vibration exposure. Vibration-induced vasoconstrictor after-effects were found to increase as the duration of acute exposure to vibration increased. This finding seems to be consistent with those of some epidemiologic studies suggesting a positive association between the duration of daily vibration exposure and the risk

for VWF among occupational groups using hand-held vibrating tools (3).

The design of this experimental study did not allow the consideration of whether the time dependency associated with rms averaging ($a^2t = \text{constant}$), as proposed by ISO 5349 (1), is adequate to assess the risk for vibration-induced vascular disorders because, in the present study, the vibration exposures were represented by stimuli with different durations but with constant magnitude. Further experiments are required to study whether exposures to the same frequency-weighted energy-equivalent acceleration derived from different combinations of vibration magnitudes, durations, and frequencies produce the same adverse effects in digital vasculature.

Concluding remarks

The results of this study indicate that complex pathophysiological mechanisms are involved in the response of finger circulation to acute exposure to vibration. Both vasodilation and vasoconstriction are elicited by a vibration stimulus with a frequency of 125 Hz. The circulatory effects in the vibrated finger seem to be mediated by both intrinsic (local) and extrinsic (neural or endocrine) vasoregulatory factors, while those observed in the nonvibrated finger are likely to be due to a sympathetic reflex mechanism. This study may contribute to the understanding of the relation between some of the physical characteristics of hand-transmitted vibration and vibration-induced changes in digital circulatory function. The findings suggest that, in addition to the frequency and magnitude of the applied stimulus (13), the duration of vibration plays a role in the reaction of the digital vessels to acute vibration.

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