

# Transitory postural vasomotor dysfunction in the finger after short term hand vibration

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**ABSTRACT** The transitory effects of hand vibration ( $a_{h,w} = 3.16 \text{ m/s}^2$  during three minutes) on postural vasomotor functions of skin capillary blood flow rate in the finger were studied by the local  $^{133}\text{xenon}$  washout technique in ten men with vibration induced white finger (VWF), nine men professionally exposed to hand-arm vibration but without finger symptoms (HAV), and eight male controls (MC). The following postural vasomotor functions were measured: (a) the vasomuscular, non-neurogenic autoregulation, tested by raising the finger 20 cm; (b) the local venoarteriolar vasoconstrictor axon reflex, tested by lowering the finger 40 cm; and (c) the central sympathetic vasoconstrictor reflex elicited by central baroreceptors and tested by changing the body posture from supine to sitting upright. Before short term vibration the vasoconstriction elicited by central baroreceptors was increased in VWF ( $p < 0.01$ ) and normal in HAV ( $p > 0.10$ ). The local axon reflex and the autoregulation functioned normally in VWF and HAV ( $p > 0.30$ ). Three minutes after vibration, autoregulation was abolished and the functions of the central and local sympathetic vasoconstrictor reflexes were equally impaired in all three groups ( $p \leq 0.01$ ). All three vasomotor functions were completely restored 60 minutes after vibration in MC ( $p > 0.10$ ) and also 30 minutes after vibration in one male control ( $p > 0.20$ ). The results indicate a hyperreactivity of the central sympathetic nervous system in VWF, and a transitory, impaired function of digital arterioles after short term vibration in all groups.

Raynaud's phenomenon, peripheral neuropathy, muscular fatigue, and enlarged fingers are all known complications of long term exposure to hand-arm vibration but the immediate effects of vibration are poorly recognised.<sup>1-4</sup> The haemodynamic effect in the finger during short term vibration has been shown to be a vasoconstriction mediated through a vibration elicited central sympathetic reflex.<sup>5</sup> Effects in the finger after cessation of vibration have not been reported.

The aim of the present study was to assess the immediate results of hand vibration on postural vasomotor functions of the finger. An impact drill of low vibration level widely used in private homes was used as the source of vibration and the following postural vasomotor functions were measured before and after a short term exposure to hand vibration in men with vibration induced white finger (VWF), men professionally exposed to hand-arm vibration but without finger symptoms (HAV), and age matched male controls (MC): (a) the vasomuscular, non-neurogenic autoregulation, tested by raising the finger

20 cm<sup>6</sup>; (b) the local venoarteriolar vasoconstrictor axon reflex, tested by lowering the finger 40 cm<sup>7,8</sup>; and (c) the central sympathetic vasoconstrictor reflex elicited by central baroreceptors<sup>9</sup> and tested by changing the body posture from supine to sitting upright.<sup>10</sup> The vasomotor responses to all three postural manoeuvres were judged by the relative change in skin capillary blood flow rate of the finger determined by local  $^{133}\text{xenon}$  washout technique.<sup>11</sup>

## Subjects and methods

The study groups included ten men with a history of VWF, nine with HAV, and eight MC. All were otherwise healthy. They gave informed consent to the study, which was approved by the local ethical committee. The three groups were matched according to age and smoking habits (table 1). The VWF and HAV groups were also matched according to the exposure period of hand-arm vibration. VWF was diagnosed and staged according to Taylor and Pelmear<sup>1</sup>: stage 1, blanching of one or more fingertips; stage 2, blanching of one or more fingers; stage 3, extensive blanching of

Table 1 Data for the three investigated groups of men

	MC (n = 8)	HAV (n = 9)	VWF (n = 10)
Age (y)	41 (32-45)	37 (29-43)	41 (30-49)
No of smokers	6	6	5
Tobacco/day (g)	16 (5-25)	16 (5-40)	20 (10-25)
Duration of exposure to vibration (y)		16 (9-19)	16 (12-19)
Stage of VWF			2 (1-3)
Duration of VWF (y)			10 (5-21)
Latent period of VWF (y)			8 (2-10)
Mean arterial blood pressure (mm Hg)	94 (82-107)	90 (83-108)	99 (84-107)
Systolic pressure gradient from upper arm to finger (mm Hg)	10 (-10 to 25)	5 (0-20)	5 (0-20)
Finger systolic pressure at 30°C (mm Hg)	120 (110-140)	110 (100-135)	110 (100-140)
FSP% (15°C)	90 (81-100)	91 (60-100)	67 (0-87)*†
FSP% (10°C)	85 (72-88)	78 (20-96)	30 (0-75)*†

Values are numbers or medians (ranges in parentheses). VWF = Vibration induced white finger, HAV = men professionally exposed to hand-arm vibration but without finger symptoms, MC = male controls, FSP% (15/10°C) = finger systolic blood pressure at finger temperature of 15° or 10°C in percentage of finger systolic pressure at 30°C. Stage of VWF refers to the Taylor-Pelmar scale evaluating severity of the disorder. Significant difference from corresponding value of MC and of HAV, respectively: \*p < 0.01; †p < 0.02.

most fingers with frequent episodes in summer and winter; stage 4, extensive blanching of all fingers in summer and winter leading to change of occupation.

All subjects abstained from tobacco, alcohol, and work with vibrating tools for at least three hours before the study. They were studied at a room temperature of  $24.0 \pm 0.5^\circ\text{C}$  after at least 45 minutes rest and when feeling comfortably warm without sweating. None had ischaemic or atrophic fingers. The most severely affected fingers were tested in VWF. The corresponding fingers (second to fourth) were tested in MC and HAV. Blood pressure was measured on the upper arm by auscultation method, using a 12 cm broad cuff. Mean arterial blood pressure was calculated as diastolic pressure plus one third of the pulse pressure (table 1). Finger systolic blood pressure was measured using strain gauge plethysmography.<sup>12</sup> The systolic pressure gradient from upper arm to finger of VWF and HAV was within the corresponding range of MC (table 1). It was therefore assumed that none of the vibration exposed subjects had significant obstructions in the arteries leading to and through the fingers<sup>13</sup> and that the disorder of the VWF group was a clear vasospastic disease. On a different day the vasoconstrictor response to cold was measured in the finger using a cuff and strain gauge technique.<sup>14</sup> The finger systolic blood pressure was measured during combined body and finger cooling to 30°, 15°, and 10°C. An attack of Raynaud's phenomenon was detected as an indirectly recorded zero pressure at 15°, or 10°C. Findings of the cold provocation test confirmed that VWF had an exaggerated vasoconstrictor response to cold compared with those of HAV and MC, and that no subjects of the HAV group showed subclinical Raynaud's phenomenon (table 1).

Postural vasomotor functions of the finger were estimated from the relative change in skin capillary blood flow rate, using the difference in washout rate of

a cutaneous <sup>133</sup>xenon depot before, during, and after each postural manoeuvre. The site of radioactive skin labelling was the dorsum of the distal interphalangeal joint, where there are no arteriovenous anastomoses.<sup>15</sup> A skin area of 12 mm diameter was labelled over two minutes by means of a modified atraumatic labelling technique,<sup>16</sup> using a bowl containing 0.35 ml <sup>133</sup>xenon in isotonic saline solution (80 MBq). The disappearance of radioactivity from the labelled area from one minute after labelling was monitored externally with a NaI (Tl) scintillation detector with the crystal surface 9 cm above the skin and wide collimation. The disappearance curve, corrected for background activity, was plotted against time in a semilogarithmic scale.

The reference body posture in all three postural tests was with the subject lying supine with the finger at the midaxillary line. Autoregulation was investigated by raising the finger 20 cm.<sup>6</sup> The washout rate of one depot was measured (a) during reference body posture, (b) when the finger was raised 20 cm, and (c) in the reference body posture. Each period of measurement lasted for about two minutes. The local venoarteriolar axon reflex was investigated by lowering the finger 40 cm.<sup>6,8</sup> The central sympathetic vasoconstrictor reflex, elicited by the central baroreceptors,<sup>9</sup> was investigated with the subject sitting on a chair with a straight back and with the finger kept at the level of the jugular notch.<sup>10</sup> Pulse rate and arm blood pressure was measured on the contralateral arm during each period of the sitting test. Each postural test was performed after repeated labelling.

Subsequently, the subjects were exposed to a unilateral hand vibration of 165 seconds duration by using an impact drill (Bosch SB 350E) kept with minimal pressure on a flint stone embedded in a concrete block. Typical root-mean-square acceleration spectra of the handle of the used impact drill measured in the direction of the Z axis<sup>17</sup> are shown in

fig 1. The acceleration level and its reproducibility were calculated from repeated measurements of three operators according to the guidelines of the International Organisation for Standardisation.<sup>17</sup> Ten measurements were performed for each operator. The median acceleration level and its total ranges were  $L_{h,w} = 130.0$  dB (129.8–130.4) with no significant difference between the three operators ( $p > 0.95$ ). Expressed in  $m/s^2$  the median acceleration was 3.16 ( $a_{h,w}$ ). This vibration should induce VWF after four hours daily exposure during ten years in about 11% of such exposed subjects.<sup>17</sup>

Fifteen seconds after vibration exposure ended, the finger was labelled, and three minutes later, measurement of the first reference period of the sitting test was made. The contralateral hand was then exposed to vibration followed by measurements of the relative blood flow rate when the finger was raised 20 cm. The hand first vibrated was then re-exposed after at least one hour followed by measurements on the ipsilateral finger when the finger was lowered 40 cm. Thus all three postural tests were repeated 195 seconds after the end of vibration in all subjects. The MC group was further tested 60 minutes after the end of vibration; one male control was also tested 30 minutes after vibration ceased.

The five months variation coefficient of relative blood flow rate in the sitting test was calculated from double determinations of the HAV group. The haemodynamic response to sitting and to lowering the finger 40 cm was also measured before vibration during conventional nerve block of the finger in eight of the HAV group and in eight of the VWF group. Blockade was performed by applying 4 ml 2% lido-

caine at the base of the finger and was considered to be effective when perception was lost to pinprick and temperature. These <sup>133</sup>xenon measurements started once the finger systolic pressure had returned to normal. The measurements of the postural test, where the finger was raised 20 cm, were repeated before vibration in ten men (4 MC, 2 HAV, and 4 VWF subjects) three minutes after a firm hand grip during 165 seconds around the handle of the non-running impact drill to assess the possible effect of hand grip on the function of autoregulation.

CALCULATIONS AND STATISTICS

The washout rate constant ( $\text{min}^{-1}$ ) in all three postural tests was defined as (a) the slope of the radioactivity disappearance curve ( $k_1$ ) in the first reference period before the postural stimulus, (b) the slope ( $k_2$ ) in the test period, and (c) the steepest slope ( $k_3$ ) in the second reference period after the postural stimulus.<sup>10</sup> The rate constants were computed from the regression line calculated from the least square method. The relative blood flow rate (F) of each postural test was defined as the numerical value of  $k_2/(0.5 \cdot (k_1 + k_3))$ .<sup>8,10</sup> Examples of <sup>133</sup>xenon washout curves have been published.<sup>10</sup> The degree of impairment of the autoregulation three minutes after vibration was calculated as follows:  $100 \cdot (F_1 - F_2)/(1 - F_2)$  (%);  $F_c = (MABP - 15.7)/MABP$ , where  $F_1$  denotes relative blood flow rate (F) before vibration,  $F_2$  denotes F after vibration,  $F_c$  denotes the estimated F at completely abolished autoregulation, MABP denotes mean arterial blood pressure when lying supine with the finger at the midaxillary line, and 15.7 is the estimated hydrostatic pressure in mm Hg of 20 cm whole blood. With the finger lowered 40 cm and with the finger at the jugular notch when sitting the degree of impairment of these two vasomotor functions was calculated as:  $100 (F_2 - F_1)/(1 - F_1)$  (%), where  $F_1$  and  $F_2$  denotes relative blood flow rate of the respective tests before and three minutes after vibration. Change in mean arterial blood pressure and pulse rate from lying to sitting upright was calculated as the difference between the value of sitting and the average value of lying measured before and after the period of sitting. The numerical mean value of the washout rate constants of the first reference period was calculated from the three postural tests before and three minutes after vibration, respectively.

The difference in nominal values between independent groups of subjects was tested with the chi-squared test for k independent samples. The difference in numerical values between independent groups was tested with the Kruskal-Wallis one way analysis of variance, followed by the Mann-Whitney rank sum test if the Kruskal-Wallis test showed significant differences. The difference in numerical values between related samples was tested with the Friedman

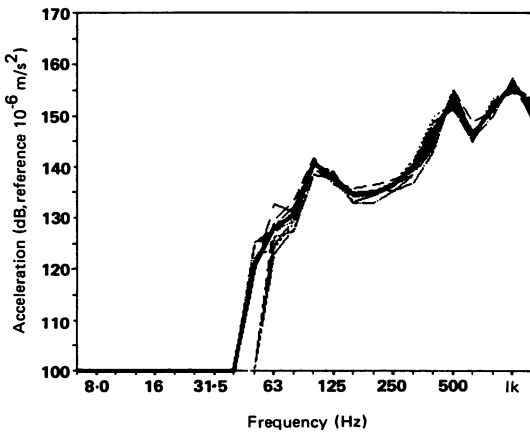


Fig 1 Reproducibility of vibration spectrum for handle of used impact drill. Thin curve lines represent ten repeated recordings obtained from one operator. Thick curve line represents their mean value.

two way analysis of variance if more than two samples followed by the Wilcoxon matched pairs signed ranks test if the Friedman test showed significant differences. In the single male control, who was also tested 30 minutes after vibration, the standard deviation (SD) of a F value was calculated from the standard deviations of the washout rate constants—that is,  $SD(k_1)$ ,  $SD(k_2)$ ,  $SD(k_3)$ —using the formula:

$$SD(F) = F \cdot \sqrt{\frac{SD^2(k_2)/k_2^2 + [(SD^2(k_1) + SD^2(k_3))/(k_1 + k_3)^2]}{2n}}$$

The difference between two F values of this subject was then tested against the null hypothesis. A significance limit of 0.05 (two tailed) was used in all statistical tests. Results are given as medians and ranges or as F values and 1 SD unless stated otherwise.

The variation coefficient of the sitting test was calculated as  $SD_{diff}$  in percentage of mean F value of the first determination where  $SD_{diff} = \sqrt{d^2/2n}$ ; d is the difference between two repeated measurements and n is number of duplicates.

## Results

There was no change in mean arterial blood pressure from lying supine to sitting upright in the total group of subjects ( $p > 0.10$ ,  $n = 27$ ) before vibration and there were no significant differences between the three groups ( $p > 0.10$ ). There was, however, a significant increase in pulse rate of  $5 \text{ min}^{-1}$  (0–14) ( $p < 0.01$ ,  $n = 27$ ) with no significant difference between the three groups ( $p < 0.10$ ). This was unchanged three minutes after vibration. Table 2 gives the five months variation coefficient of the sitting test and other methodological results.

Table 3 shows the relative blood flow rates of the finger during postural manoeuvres before and after hand vibration in the three groups. Before vibration

the vasoconstriction to sitting was increased in VWF when compared with those of the HAV and MC groups ( $p < 0.01$ ) whereas the HAV and MC groups showed the same degree of vasoconstriction ( $p > 0.10$ ). The relative blood flow rates before vibration did not differ significantly between the three groups when the finger was lowered 40 cm and when the finger was raised 20 cm ( $p > 0.30$ ). Three minutes after vibration the vasoconstriction to sitting and to lowering the finger 40 cm decreased in all three groups ( $p \leq 0.01$ ). The F value three minutes after vibration of raising the finger 20 cm was equally decreased in all groups ( $p \leq 0.01$ ). Sixty minutes after vibration the F values of all three postural tests of the MC group did not differ from those measured before vibration ( $p > 0.10$ ). The degree of impairment of the three postural vasomotor functions three minutes after vibration did not differ between the three groups ( $p > 0.10$ ). In total ( $n = 27$ ) its median was 107% with the finger raised 20 cm, 39% with the finger lowered 40 cm, and 54% when sitting upright, without significant difference between the two last mentioned values ( $p > 0.10$ ).

Figure 2 shows the results of the single male control, who was also tested 30 minutes after vibration. The vasomotor functions of the three postural tests were significantly impaired three minutes after vibration ( $p \leq 0.0004$ ) and completely restored 30 and 60 minutes after vibration ( $p > 0.20$ ).

The median value of the calculated washout rate constant of the first reference period was in total ( $n = 27$ )  $0.365 \text{ min}^{-1}$  before vibration and  $0.309 \text{ min}^{-1}$  three minutes after vibration ( $p < 0.03$ ) with no significant difference between the three groups ( $p > 0.10$ ).

## Discussion

### METHODOLOGICAL ASPECTS

Using the present techniques, the vasomuscular autoregulation and the local venoarteriolar reflex were

Table 2 Methodological results of relative blood flow rate in the finger during postural manoeuvres

Postural manoeuvre	Situation	Relative blood flow rate	Group of men
Sitting with finger at jugular notch	First determination	0.78 (0.71–0.91)	HAV (n = 9)
	Second determination	0.80 (0.75–0.92)†	
	Variation coefficient (%)	5.6 (five months)	
Finger lowered 40 cm	Without proximal nerve block	0.57 (0.42–0.71)	VWF (n = 8)
	During proximal nerve block	0.60 (0.49–0.75)†	
Sitting, finger at jugular notch	Without proximal nerve block	0.81 (0.71–0.91)	HAV (n = 8)
	During proximal nerve block	1.01 (0.90–1.09)*	
Finger raised 20 cm	Before hand grip	0.98 (0.92–0.99)	Ten men: MC(n = 4) HAV (n = 2), VWF (n = 4)
	Three min after hand grip	0.99 (0.92–1.08)†	

Values are medians (ranges in parentheses). Reference body posture was lying supine with finger at midaxillary line. Nerve block was performed at the base of the finger. Hand grip refers to a firm grip during 165 s around the handle of the non-running impact drill.

\*Significantly different from corresponding value of same postural manoeuvre in other situation of same group of men,  $p = 0.01$ .

†Not significantly different from corresponding value of same postural manoeuvre in other situation of same group of men,  $p \geq 0.10$ .

Table 3 Relative blood flow rates of the finger during postural manoeuvres before and after short term hand vibration

Postural manoeuvre	Tested vasomotor regulatory mechanism	Group of men	Relative blood flow rate		
			Before vibration	Three min after vibration	Sixty min after vibration
Finger raised 20 cm	Arteriolar muscular autoregulation	MC (n = 8)	0.99 (0.97-1.11)§	0.80 (0.71-0.94)*	1.00 (0.96-1.06)§
		HAV (n = 9)	0.96 (0.92-1.09)§	0.80 (0.67-0.90)*	
		VWF (n = 10)	0.99 (0.96-1.01)§	0.82 (0.73-1.00)*	
Finger lowered 40 cm	Local venoarteriolar axon reflex	MC (n = 8)	0.54 (0.44-0.75)	0.69 (0.59-0.99)*	0.58 (0.42-0.73)
		HAV (n = 9)	0.59 (0.40-0.69)	0.73 (0.57-1.10)*	
		VWF (n = 10)	0.57 (0.42-0.71)	0.66 (0.49-1.04)*	
Sitting with finger at jugular notch	Central sympathetic baroreceptor reflex	MC (n = 8)	0.83 (0.71-0.92)	0.95 (0.85-1.10)*§	0.81 (0.73-0.88)
		HAV (n = 9)	0.80 (0.75-0.92)	0.89 (0.75-1.03)*	
		VWF (n = 10)	0.56 (0.47-0.66)†‡	0.76 (0.58-0.97)*†‡	

Values are medians (ranges in parentheses). Reference body posture was lying supine with finger at midaxillary line. § Significantly different from corresponding value of same postural manoeuvre before vibration in same group of men,  $p \leq 0.01$ . \* Significant difference from corresponding value of same postural manoeuvre at same situation in MC and HAV, respectively: †  $p < 0.02$ ; ‡  $p < 0.05$ . †‡ Not significantly different from unity,  $p > 0.10$ .

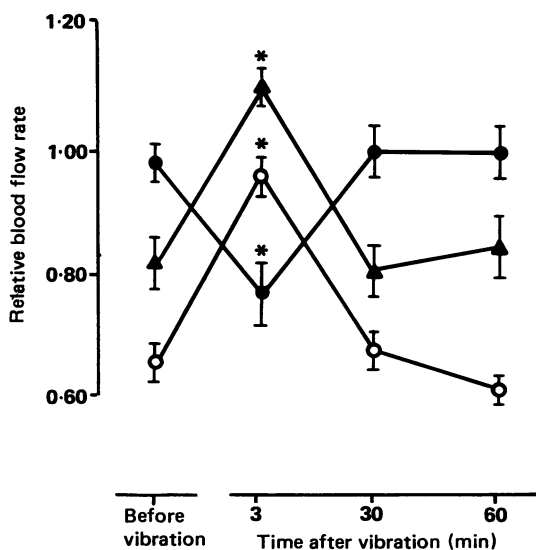


Fig 2 Relative blood flow rates of finger during postural manoeuvres before and after short term hand vibration in a male control. Reference body posture was lying supine with finger at midaxillary line. Values are relative rates  $\pm 1$  SD. ● Finger raised 20 cm, ○ finger lowered 40 cm, ▲ sitting upright with finger at jugular notch. \* Significantly different from corresponding value of same postural manoeuvre before vibration,  $p \leq 0.0004$ .

recognised in the normal finger before vibration as in other studies.<sup>6</sup>

The vasoconstrictor response to sitting was investigated using nervous blockade at the base of the finger placed 6-7 cm proximal to the area investigated. This distance exceeds the radius of action of the local axon reflex.<sup>7,8</sup> Thus vasoconstriction of the axon reflex was expected to remain unaffected if the volume of lidocaine did not significantly compress the veins draining the investigated anatomical region. In

agreement with these assumptions the vasoconstriction elicited by lowering the finger 40 cm was unchanged during nerve block. The applied blockade seems therefore suitable in distinguishing between centrally and locally mediated sympathetic vasoconstrictor reflexes. The vasoconstriction in sitting posture was significantly abolished by nervous blockade, indicating a central sympathetic vasoconstrictor reflex in agreement with our earlier suggestion.<sup>10</sup> This was further supported by our results of an unchanged mean arterial blood pressure in parallel with increased pulse rate during sitting upright. The results correspond with those from earlier passive head-up tilt studies.<sup>9</sup> The sitting test was found to have an acceptable reproducibility, the five months variation coefficient being about 6%.

The vasomuscular autoregulation appears to be sensitive to arterial carbon dioxide tension and oxygen saturation.<sup>18</sup> Autoregulation, however, was intact three minutes after a three minutes firm hand grip around the handle of the non-running impact drill. Thus an impairment of autoregulation three minutes after vibration was assumed to be exclusively caused by vibration and not by ischaemia.

PREVIBRATION SITUATION

No injury of the postural vasomotor functions was detected in the HAV group. The VWF group showed a normal function of the autoregulation and the local axon reflex. Consequently, no damage to the digital arterioles or the postganglionic sympathetic nerve fibres was detected in this group. The VWF group showed an exaggerated vasoconstriction to sitting upright, verifying earlier findings.<sup>10</sup> This result may be explained by a hyperreactivity of the central sympathetic nervous system in subjects with VWF as their postganglionic elements functioned normally. Our results seem to correspond with Raynaud's suggestion of hyperreactivity of the central sympathetic nervous

system in subjects with vasospastic types of the phenomenon<sup>19</sup> and contrast with the suggestion by Lewis of a "local digital vascular fault."<sup>20</sup>

#### POSTVIBRATION SITUATION

The results obtained three minutes after vibration showed abolished autoregulation and equally impaired functions of the local and central sympathetic vasoconstrictor reflexes in all three groups. In total the impairment was about 100% for autoregulation and about 40–50% for the two neurogenic reflexes. Thus autoregulation was the most vulnerable vasomotor function injured by vibration. It has been suggested that the local axon reflex and the central sympathetic reflex might affect greater arterioles, which are richly innervated by adrenergic sympathetic nerve fibres, whereas non-neurogenic autoregulatory reactions of arteriolar smooth muscle cells take place preferentially in the terminal arterioles.<sup>7</sup> This postulate seems verified by the findings of many young insulin-dependent patients with bioptically verified terminal arteriolar hyalinosis in the skin having disturbed or impaired autoregulation,<sup>21</sup> but normal local and central postural reflexes.<sup>21,22</sup> Our results may therefore, indicate that short term low level vibration leads to an immediate abolition of intrinsic autoregulatory function in terminal arteriolar smooth muscle cells and a net impairment of the function of postganglionic sympathetic nerve fibres and smooth muscle cells of greater arterioles. The assumption of a changed activity of the central sympathetic nervous system three minutes after vibration is therefore not needed for further explanation. It is reasonable to suppose that the musculature of larger arterioles also shows an impaired function after vibration. This would agree with results of *in vitro* and *in vivo* studies in animals, showing a vibration induced inhibition of smooth muscle contraction in veins and arteries<sup>23–25</sup> probably caused by a direct mechanical effect on the actin myosin cross links of the vascular smooth muscle cell.<sup>25</sup> Our results cannot evaluate whether an affection of postganglionic sympathetic nerve fibres contributes to the net impairment of the neurogenic vasomotor functions.

The current impairment of vasomotor functions was associated with a small decrease in skin capillary blood flow rate at reference body posture. This may be explained as an after effect of the vasoconstriction induced during vibration.<sup>5</sup> Contrary to this outcome, the results obtained in anaesthetised, vibration exposed dogs show an immediate after effect of increased arterial blood flow rate in the test limb,<sup>26</sup> apparently because anaesthesia of dogs with sodium pentobarbital<sup>26</sup> blocks the vibration elicited central sympathetic vasoconstrictor reflex.<sup>5</sup>

Sixty minutes after vibration, all three postural

vasomotor functions were completely restored in the male controls, and even after 30 minutes in the one person tested. No hyperreactivity of the functions was measured at that time, contrary to earlier results obtained from *in vitro* and *in vivo* animal experiments showing hyperresponsiveness of arterial smooth muscle to adrenaline one hour after vibration<sup>23,26</sup> but in agreement with later *in vitro* studies.<sup>24</sup> This discrepancy of results may be explained by differences in the used frequencies, levels, and periods of vibration.

We may conclude that the present vibration of low level widely experienced in private homes induces an immediate impairment of postural vasomotor functions, completely restored within 30 minutes and without hyperreactivity 60 minutes after vibration.

#### PATHOPHYSIOLOGICAL ASPECTS

Our present and earlier results indicate that activation of the vibration elicited central sympathetic vasoconstrictor reflex during several years of work with vibrating hand tools may induce hyperreactivity of the central sympathetic nervous system in predisposed subjects.<sup>5,10</sup> This central neurogenic hyperreactivity probably causes exaggerated digital vasoconstriction to cold and exaggerated cochlear vasoconstriction to noise, leading to Raynaud's phenomenon<sup>27</sup> as well as to hearing loss.<sup>28</sup> Episodes of Raynaud's phenomenon never or only rarely occur immediately after exposure to vibration but are displayed typically in the morning before work and in leisure periods.<sup>1</sup> This characteristic pattern of attacks of VWF may be caused by the demonstrated impairment of postganglionic elements immediately after exposure to vibration.

Repeated transitory impairment of the postural central sympathetic vasoconstrictor reflex, which contributes to the maintenance of systemic blood pressure by increasing the peripheral resistance to blood flow during erected body posture,<sup>9</sup> may contribute to the development of autonomic disorders such as the dizziness and orthostatic hypotension reported in some operators of vibrating hand tools.<sup>29</sup>

The postvibration impairment of the local veno-arteriolar axon reflex, which serves as a local oedema protecting factor,<sup>7,8</sup> may cause transitory oedema in the finger similar to the vibration induced epineural oedema observed in the sciatic nerve of rats.<sup>30</sup> Repeated oedema during several years of exposure to vibration may lead to enlarged fingers,<sup>4</sup> peripheral muscular fatigue,<sup>3</sup> and peripheral neuropathy<sup>2</sup> in operators with and without VWF.

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