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Experimental studies on the effects of vibration and noise on sympathetic nerve activity in skin

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Summary. Multi-unit sympathetic activity was recorded at elbow level from median nerve fascicles supplying glabrous skin of the left hand in five healthy subjects. The resultant vasomotor responses accompanying the neural activity were monitored by simultaneous recordings of skin blood flow using the laser doppler method and skin temperature in the innervation zones. No significant change in sympathetic activity was observed during handgrip exercise of the right hand under a constant gripping force of 2 kg. Subjects maintained the same gripping force of the right hand during exposure in random order to local vibration and/or noise, each type of exposure lasting 5 min with intervals of 20 min. A two-peaked significant increase in outflow from sympathetic nerves was observed during local exposure of the right hand to vibration with a frequency of 60 Hz and an acceleration of 50 $m \cdot s^{-2}$, followed by a postexposure period which revealed a relative suppression of sympathetic nerve activity and a significant increase in blood flow. Noise at 100 dB(A) showed only an initial effect on skin sympathetic nerve activity (SSA), whereas when combined with local vibration at 60 Hz, a pronounced increase in neural activity was noticed, indicating a combined effect of vibration and noise. These results from direct recordings of SSA suggest a sympathetic vasomotor reflex mechanism triggered by local vibration stimuli to the hand.

Key words: Vibration - Noise - Skin sympathetic activity

Introduction

There have been many studies on the pathogenesis of Raynaud's phenomenon occurring in workers using hand-held vibrating tools, although the mechanism still remains unclear. Some recent studies have focused on the effect of vibration and noise arising from vibrating tools on skin sympathetic nerve activity (SSA). The monitoring of SSA of the fingers has been performed mostly by methods such as skin temperature measurements or plethysmography and more recently by using direct blood flow measurements. The results of such studies have indicated that vibration on the fingers may affect the SSA in the finger (Pyykkö and Hyvärinen 1973; Hyvärinen et al. 1973; Okada et al. 1988; Kondo et al. 1987; Nasu 1977; Färkkilä and Pyykkö 1979; Sakurai 1977; Welsh 1980).

Skin temperature measurements, plethysmography and blood flow measurements are noninvasive and easy to perform, but it is possible that influences apart from nerve activity may influence the overall result. Mechanical effects accompanying the handgrip of tools, hormonal adjustments and further external influences, such as environmental temperature, can modify the values. We think direct recording of nerve activity will enable elucidation of characteristic patterns of response to different stimuli. In 1972, a microneurographic technique using metal micro-electrodes made possible the direct recording of SSA from conscious humans (Hagbarth et al. 1972). However, because of the difficulty of the technique there have been few reports on the relationship between vibration or noise and direct recordings of SSA.

It has been suggested that vibration transmitted to the hand-arm system may affect fingers of both hands through the somato-sympathetic reflex mechanism (Kondo et al. 1987; Nasu 1977; Färkkilä and Pyykkö 1979). In the present study, the microneurographic technique was employed to investigate changes in SSA on the hand contralateral to the vibration-exposed hand, so as to investigate the effect of vibration on the somato-sympathetic reflex. Further, the effect of noise on SSA was also examined. The purpose of this study was to clarify the role of a sympathetic vasomotor reflex in the pathogenesis of occupational Raynaud's phenomenon.

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Methods

Subjects. The subjects were five healthy men aged 20–24 years, average age 22.5 years. None of them had previous experience of using hand-held vibrating tools and all were right-handed. The subjects were fully informed of the purpose and procedures of the experiments. None of the subjects experienced pain during the experiment.

SSA recording. For 30 min before the start of the experiment, the subjects were kept in a room at a temperature of 23 ± 1° C, humidity 40%, in a supine position on a bed. The background noise level of the room was about 40 dB(A). A tungsten micro-electrode with a tip diameter of 1 μm axial diameter of 0.1 mm and impedance of 2–4 MΩ was inserted percutaneously without anaesthesia into the median nerve at elbow level. The sympathetic nerve impulses to the glabrous skin of the index and middle finger were recorded and identified using the procedure previously reported (Mano 1983a, b). The impulse activity was passed through a differential amplifier (DAM50; World Precision Instrument Inc, Florida, USA), a bandpass filter (bandwidth 600–3000 Hz) and monitored on an oscilloscope, loudspeaker and omnicorder (Fig. 1).

The following characteristics identified the SSA:

1. Multi-unit grouped discharges
2. Independence from the heartbeat
3. Activity observable as a result of light touch sensation of the innervated skin, but not from tapping or squeezing of muscle
4. Activity increasing by electrical or arousal stimuli (Mano 1983b; Hagbarth et al. 1972; Vallbo 1979; Fig. 2).

The neurogram was stored on magnetic tape (RTP-550A; Kyowa Electric Inc, Tokyo, Japan). For analysis, the neurogram was fed through a full-wave rectifying and integrating circuit [time constant 0.1 s; Fig. 3, SSA(A)], from which the impulse frequency was counted and displayed [Fig. 3, SSA(B)].

Recordings of skin blood flow, skin temperature and surface electromyogram. Skin blood flow in the fingertip of the left middle finger was measured using a laser doppler method and skin temperatures on the fingertip of both index fingers were recorded every 30 s from thermistors. The electromyogram was monitored from muscles of the right forearm. After the experiments, the blood flow was calculated for 10-s periods and the mean for every minute for both blood flow and skin temperature.

Vibration exposure. The apparatus for vibration exposure consisted of an accelerator (VS-20-03; IMV Inc, Osaka, Japan), an amplifier (VA-ST-03; IMV Inc, Osaka, Japan), a function oscillator (AG202A; Trio Inc, Tokyo, Japan), and a function generator (VM-1960; IMV Inc, Osaka, Japan). A cylindrical handle made of acrylic fibre with a diameter of 40 mm was fixed on top of the accelerator, and a sensor for monitoring the vibration level was placed on the handle. All subjects gripped the handle with their right hands and local vibration occurred in the direction of the X-axis (International Standards Organisation 1982). During the

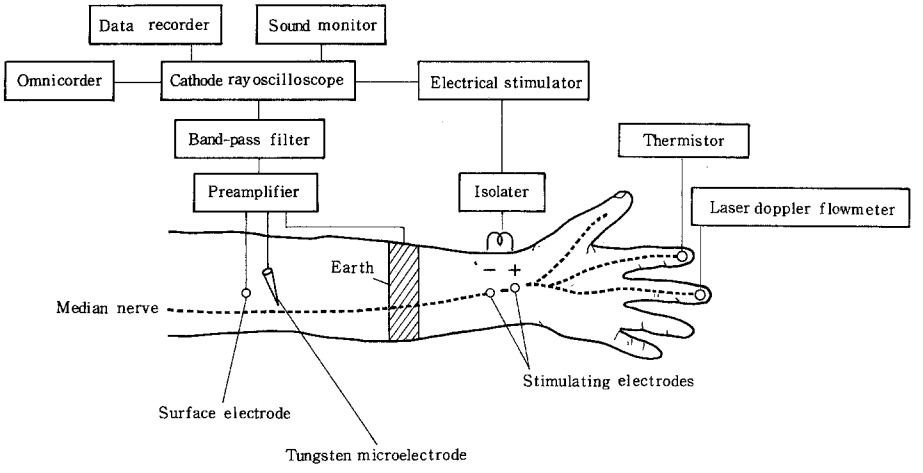

Fig. 1. Experimental set-up for recording skin sympathetic nerve activity, finger blood flow and skin temperature

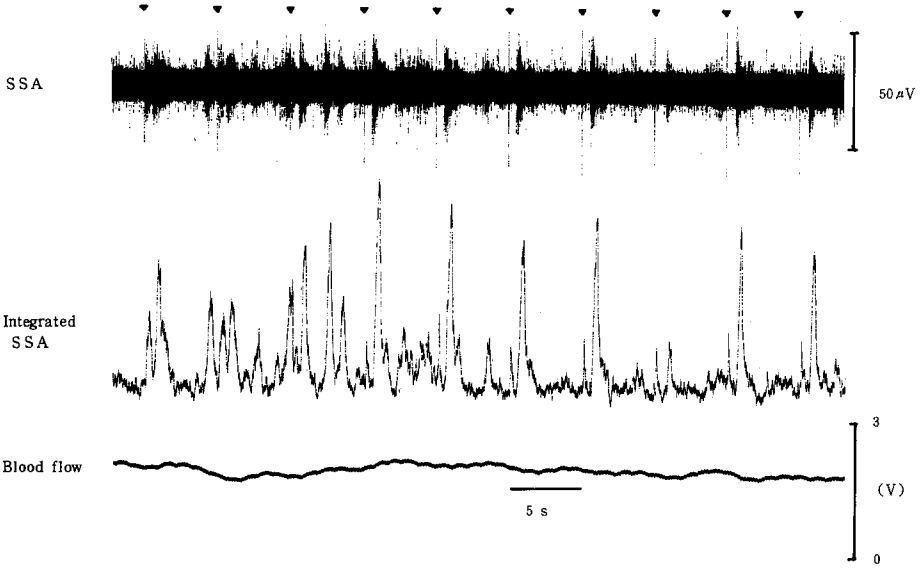

Fig. 2. Typical recordings of skin sympathetic nerve activity (*SSA*), full-wave-rectified and integrated skin sympathetic nerve activity (Integrated *SSA*) and finger blood flow. Bursts of skin sympathetic activity are induced by electrical stimuli to wrist (shown by ▼) and followed by decreases in blood flow

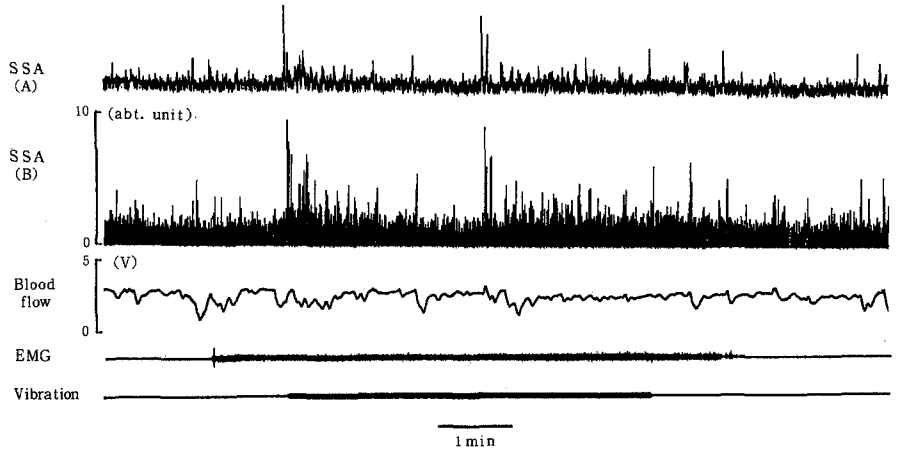

Fig. 3. An example of original recordings of full-wave-rectified and integrated skin sympathetic nerve activity (*SSA(A)*), instantaneous rate of *SSA* impulses (*SSA(B)*), blood flow and electromyogramme (*EMG*) in a male subject aged 20 years; abt. unit, arbitrary unit

exposure, the acceleration of the vibration was kept constant. A vibration frequency of 60 Hz was used in this experiment. The gripping force during handgrip alone as well as during vibration was kept at 2 kg.

Noise exposure. Noise at 57 dB(A), and at 100 dB(A), were played back from a tape recorder in the experiments.

Experimental procedure. After positioning the sensors for blood flow and skin temperature along with the electrodes for the electromyogram, a tungsten micro-electrode was inserted in the left median nerve and activity in the sympathetic nerve to the skin of the index and middle fingers was identified. Subsequently, the subject practised holding the handle at a constant gripping force of 2 kg, including the period of vibration. In such cases, attention was paid to ensure that no excessive tension was exerted on either arm, especially at the beginning and end of the handgrip. After 20-min rest, the experiment commenced when the SSA, blood flow and skin temperature had all stabilized. Each experiment was performed with the subjects in a supine position with their arms extended and relaxed. Firstly, the effect of handgrip was observed by asking the subject to grasp the handle for 5 min at a constant gripping force of 2 kg, with a 5-min rest before and after the handgrip.

Secondly, the following stimuli, (1) noise at 57 dB(A), (2) noise at 100 dB(A), (3) vibration at 60 Hz, 50 $m \cdot s^{-2}$, (4) noise at 100 dB(A) and vibration at 60 Hz, 50 $m \cdot s^{-2}$, were applied in random order for 5 min each with an interval of 20 min. For each stimulus, recordings at rest were made, and when the SSA stabilized, the subject gripped the handle with his right hand for 1 min. Continuing the handgrip, vibration or noise was applied, and on termination of the stimulus, handgrip was continued for 1 min before the subject rested his arms.

Results

Changes in skin temperature

The changes in skin temperature before, during and after vibration and/or noise are shown in Table 1. The values for each individual were normalized to the value at rest. No significant change was observed during and after handgrip alone compared to the value at rest. Similar changes in right and left hands were observed for exposure to noise at 100 dB(A), to local vibration, and to combined exposure to vibration and noise.

SSA and skin blood flow during and after handgrip

No significant change was observed in SSA or blood flow during and after handgrip compared to the value at rest (Fig. 4).

SSA and skin blood flow during and after exposure to handgrip and noise at 57 dB(A)

No significant change was observed in SSA or blood flow during and after exposure to noise at 57 dB(A) compared to the value during handgrip prior to noise (Fig. 5).

SSA and skin blood flow during and after exposure to handgrip and noise at 100 dB(A)

The SSA significantly increased during the 1st min of noise at 100 dB(A), while blood flow significantly decreased, both compared to the value during handgrip prior to the noise stimulus. No significant changes were observed in either the SSA or blood flow after the 2nd min of exposure to noise at 100 dB(A) (Fig. 6).

SSA and skin blood flow during and after exposure to handgrip and local vibration at 60 Hz and 50 $m \cdot s^{-2}$

The SSA significantly increased during the 1st as well as the 5th min of vibration exposure compared to the value during handgrip prior to vibration. Blood flow significantly decreased during the 1st min of vibration compared to the value during handgrip prior to vibration. Blood flow significantly increased during the 3rd min after the end of vibration (Fig. 7).

Combined effect of handgrip, vibration and noise

The SSA increased significantly and blood flow decreased significantly during the 1st, 2nd, 4th and 5th min of simultaneous exposure to local vibration and

Table 1. Changes in skin temperature before, during and after various loads. Normalized value (%)

Time after handgrip	Handgrip only		Noise at 57 dB(A)		Noise at 100 dB(A)	
	RT	LT	RT	LT	RT	LT
1 min	100.3±0.13	99.7±0.17	100.2±0.14	99.5±0.11	100.7±0.23	99.7±0.13
2 min	99.2±0.35	100.0±0.22	99.5±0.20	99.6±0.23	100.2±0.28	99.5±0.20
3 min	98.9±0.62	100.6±0.39	99.3±0.19	99.7±0.35	99.4±0.31	99.2±0.30
4 min	99.0±0.94	101.3±0.56	99.0±0.22	100.1±0.37	98.9±0.32	98.8±0.28
5 min	98.5±0.94	101.8±0.76	99.0±0.22	100.7±0.42	98.5±0.32	98.9±0.27
6 min	100.0±0.77	101.9±0.81	98.8±0.31	101.0±0.54	98.3±0.40*	99.2±0.31
7 min	101.8±0.71	102.5±0.78	99.8±0.41	101.1±0.56	98.1±0.36*	99.0±0.41
8 min			99.8±0.43	101.3±0.67	99.0±0.48	99.8±0.60
9 min			100.5±0.34	101.6±0.76	101.7±1.20	100.4±0.72

Time after handgrip	Vibration at 60 Hz		Vibration at 60 Hz and noise at 100 dB(A)	
	RT	LT	RT	LT
1 min	100.6±0.20	99.5±0.13	100.4±0.18	99.7±0.16
2 min	99.5±0.18	98.9±0.23	99.9±0.17	99.3±0.20
3 min	98.6±0.32	98.4±0.35	99.2±0.21	98.8±0.25
4 min	97.9±0.47*	98.1±0.44	98.6±0.24	98.2±0.29*
5 min	97.9±0.70	98.1±0.48	98.0±0.24*	97.7±0.35**
6 min	97.4±0.71*	97.9±0.60	97.8±0.34*	97.4±0.40**
7 min	97.5±0.72*	97.8±0.52	98.1±0.29*	97.2±0.37**
8 min	99.4±0.62	98.0±0.69	98.3±0.26	97.3±0.40**
9 min	102.1±0.80	98.8±0.61	99.5±0.51	97.2±0.47**

RT, Right index finger; LT, left index finger; * $P<0.05$, ** $P<0.01$, significance of difference from the value at rest using one-way ANOVA followed by Tukey's multiple comparison

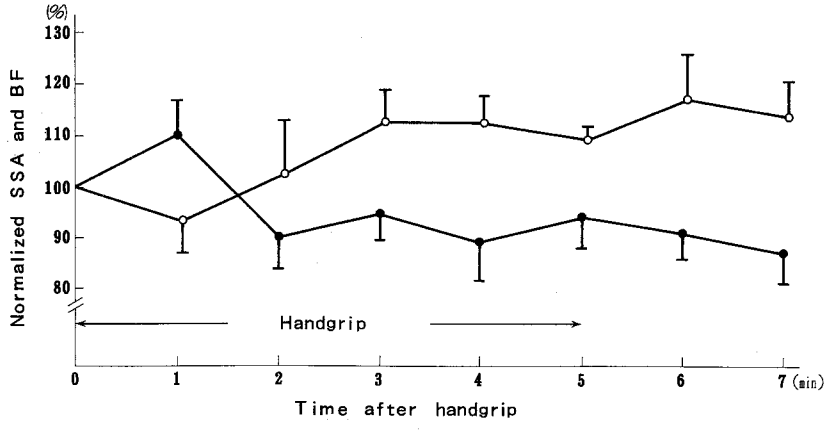

Fig. 4. Changes in skin sympathetic nerve activity (*SSA*) and blood flow (*BF*) during and after handgrip with a constant force of 2 kg. ●, *SSA*; ○, blood flow. Individual values of both sympathetic nerve discharge frequency (as shown in Fig. 3) and blood flow calculated every 10 s were averaged for 1 min and normalized to the value at rest, which was the average value during the minute directly preceding handgrip. Values are means and SEM

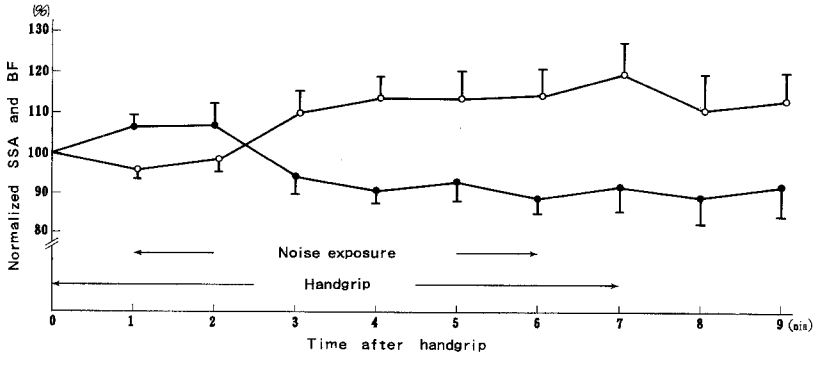

Fig. 5. Effect of noise [57 dB(A)] on skin sympathetic nerve activity (*SSA*) and blood flow (*BF*). Values are means and SEM. For definitions of symbols and method of calculation see Fig. 4

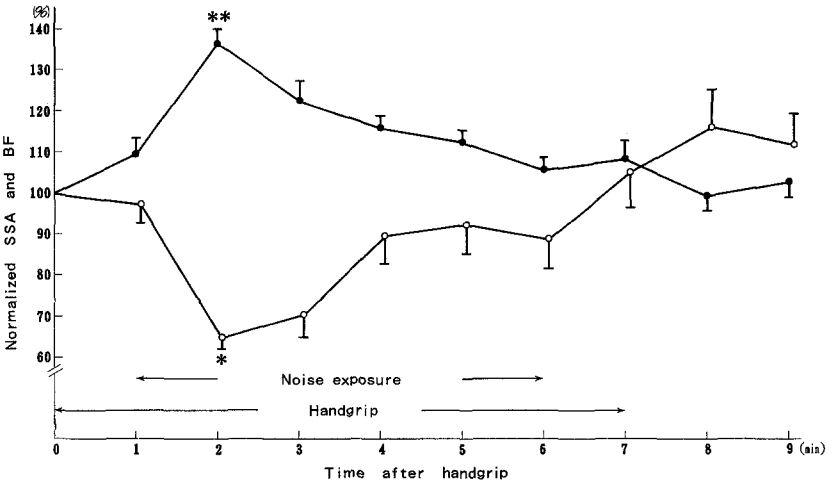

Fig. 6. Effect of noise [100 dB(A)] on skin sympathetic nerve activity (*SSA*) and blood flow (*BF*). Values are means and SEM. * $P<0.05$, ** $P<0.01$, significance of difference from the value during handgrip prior to noise using one way ANOVA followed by Tukey's multiple comparison. For definition of symbols and method of calculation see Fig. 4

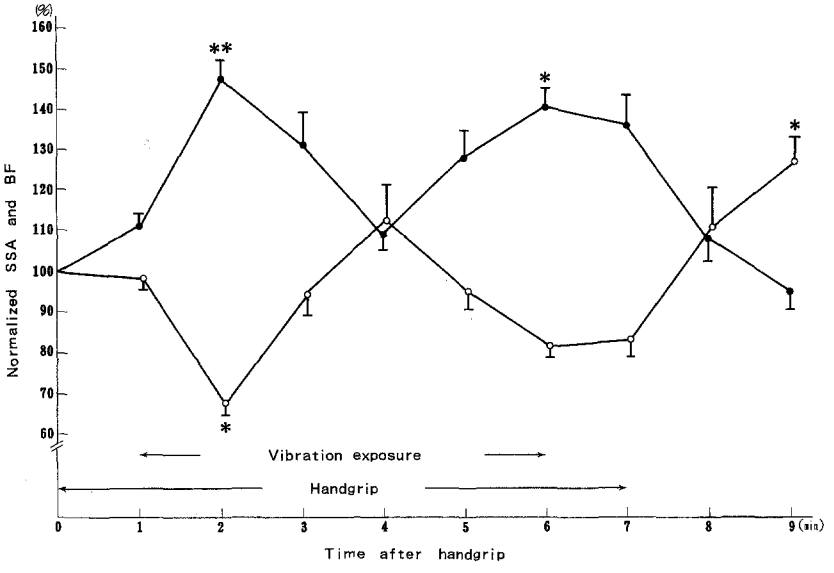

Fig. 7. Effect of local vibration (60 Hz, 50 $m \cdot s^{-2}$) on skin sympathetic nerve activity (*SSA*) and blood flow (*BF*). Values are means and SEM. * $P<0.05$, ** $P<0.01$, significance of difference from the value during handgrip prior to vibration using one-way ANOVA followed by Tukey's multiple comparison. For definition of symbols and method of calculation see Fig. 4

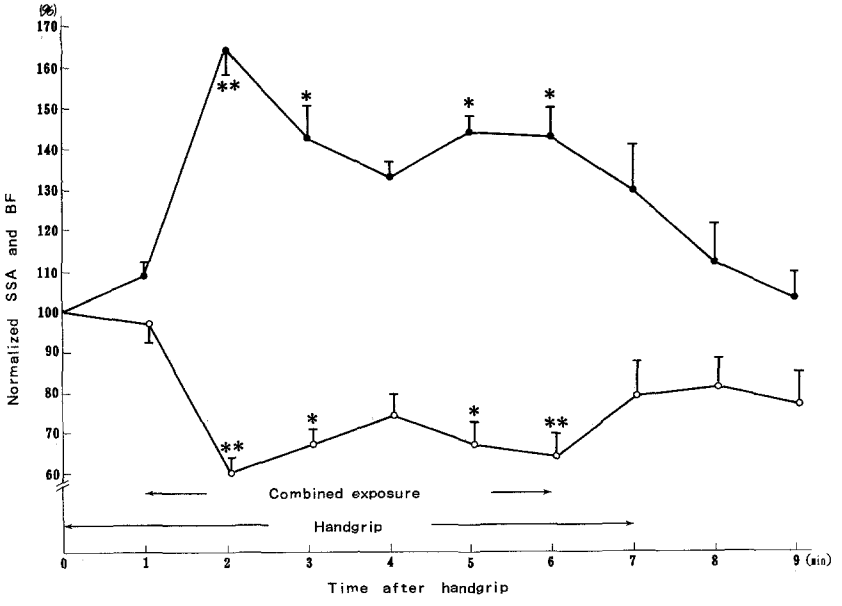

Fig. 8. Effect of combined local vibration (60 Hz, 50 $m \cdot s^{-2}$) and noise [100 dB(A)] on skin sympathetic nerve activity (*SSA*) and blood flow (*BF*). Values are means and SEM. * $P<0.05$, ** $P<0.01$, significance of difference from the value during handgrip prior to combined exposure using one-way ANOVA followed by Tukey's multiple comparison. For definition of symbols and method of calculation see Fig. 4

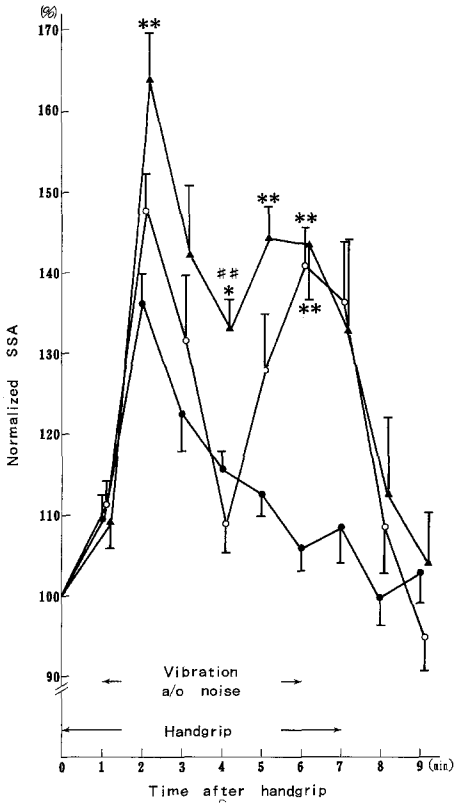

Fig. 9. Effect of combined local vibration and noise on skin sympathetic nerve activity (*SSA*). ○, local vibration (60 Hz, 50 $m \cdot s^{-2}$); ●, noise [100 dB(A)]; ▲, local vibration (60 Hz, 50 $m \cdot s^{-2}$) + noise [100 dB(A)]. Values are means and SEM. Statistical analysis was performed using one-way ANOVA followed by Tukey's multiple comparison. * $P<0.05$, ** $P<0.01$, significance of difference from the corresponding value for noise alone. ## $P<0.01$, significance of difference from the corresponding value of vibration exposure alone. For explanation for data calculation see Fig. 4; a/o, and/or

noise [100 dB(A)], compared to the value during handgrip prior to the combined exposure (Fig. 8). The increase in SSA was significantly greater during the 1st, 3rd, 4th and 5th min of combined exposure compared to the corresponding response to noise alone. The increase in SSA was significantly greater during the 5th min of vibration exposure compared to the corresponding response to noise alone. Furthermore, the SSA was significantly greater during the 3rd min of combined exposure compared to the corresponding value not only to noise alone but also to vibration alone (Fig. 9).

Discussion

When applying vibration locally to the hand, the effect of handgrip itself must be taken into consideration. In this study, we have performed a control experiment using the same gripping force as when vibration was applied. The gripping force was set at 2 kg, after a preliminary experiment to determine the force which could be maintained at a fixed level while keeping the subjects' fatigue at a minimum. No significant changes were observed in either the SSA, blood flow or skin temperature during the 5 min of handgrip.

Workers using vibrating tools are often exposed to noise levels as high as 80–120 dB(A) from the tools and to cold as well as to local vibration on the hand-arm system. In the present study, attention was paid to noise. Firstly, we observed the response to noise at 57 dB(A), which was the noise level associated with the vibration generator used in the present experiment. The levels of both SSA and blood flow were maintained during the 1st min after the noise stimulus. The level of SSA was quite low and the blood flow high after the 2nd min of noise with no marked changes observed after cessation of the noise, which was an altogether similar response pattern to handgrip alone. Changes in skin temperature to noise at 57 dB(A) were also similar to changes with handgrip alone. From such findings, the effect of noise at 57 dB(A) on SSA seemed to be small.

However, loud noise is known to affect the peripheral circulation. Nakamura et al. (1987) have reported that when exposed to noise at 100 dB(A) during handgrip, tissue blood flow in the finger on the side of the hand grip showed a tendency to decrease after the 1st min of exposure, followed by recovery. Pyykkö and Hyvärinen (1973) have observed a bilateral decrease in the amplitude of the finger plethysmogram after exposure to noise at 100 dB(A) and a significant decrease of skin blood flow in the 1st min after exposure, demonstrating that the change in blood flow was due to an increase of SSA.

We have reported that by exposing rats to a noise level of 100 dB(A), noradrenaline concentration decreased significantly in the midbrain and also tended to decrease in the hypothalamus (Okada et al. 1984). These findings suggested that loud noise stimulated activity of noradrenaline neurons, inducing vasomotor reflexes through the midbrain and hypothalamus, thus causing peripheral vascular responses. Furthermore, after the 2nd min of noise, SSA and blood flow gradually recovered, which implied that vasomotor activity adapted to this stimulus.

It is known that the vibration acceleration arising from vibrating tools occurs at 0.316–100 $m \cdot s^{-2}$. In our study, acceleration was kept constant at 50 $m \cdot s^{-2}$, and we selected a frequency of 60 Hz, because that had been the frequency observed in our laboratory as being the one which significantly decreased finger blood flow at this acceleration. The SSA increased significantly after hand-arm vibration at 60 Hz, when compared with the response to handgrip alone. Miyamoto and Alanis (1970) have reported a sympathetic nerve reflex response to vibration stimuli in the pad of the cat's paw. Our study demonstrated, for the first time in humans, the existence of a vibration-induced somato-sympathetic reflex. Moreover, compared to the response to noise, SSA increased significantly not only immediately following the stimulus, but also at the 5th min after the start of the stimulus, showing a distinct contrast with the response pattern to noise. It is suggested that such a difference in the response originated from the differ-

ence in the reflex pathway between vibration and noise stimuli.

Pacinian corpuscles, which are receptors widely distributed in the hand, are highly sensitive to vibration at frequencies of 50–400 Hz and are also recognized anatomically as having connections with sympathetic fibres (Santini et al. 1971). With the acceleration used in this experiment, it is assumed that vibration stimuli at 60 Hz induced a sympathetic reflex through the Pacinian corpuscles. However, the involvement of other receptors such as the Meissner corpuscles, which respond to vibration frequencies of 5–60 Hz (Lundström 1986), cannot be completely discounted.

It is interesting that blood flow increased significantly after ceasing vibration at 60 Hz compared to the value before the stimulus. Schmidt and Schönfuss (1970), from an experiment recording somato-sympathetic reflex activity by applying electrical stimuli to a sensory nerve, showed that it was markedly suppressed after cessation of the stimuli.

In the present study, after the beginning of the stimulus, SSA was at its lowest level at the 2nd and 3rd min after the cessation of the stimulus. Therefore the increase in skin blood flow was thought to be due to a relative suppression of SSA after cessation of vibration. The change in finger skin temperature in the right and left hands during and after vibration was almost synchronous. Thus the changes in blood flow during and after vibration have been assumed to be induced bilaterally through vasomotor reflexes. The prompter recovery of the skin temperature in the right compared to the left hand was probably because the response on the stimulated side involved factors such as release of mechanical compression of the vessels and tissue metabolism, including recovery heat after muscle constriction (Nagasaka et al. 1990), in addition to the suppression of the sympathetic reflexes.

We combined noise at 100 dB(A) with vibration at 60 Hz and observed that SSA became higher than during exposure to 60 Hz vibration alone. The blood flow also responded markedly to the combined stimuli, corresponding to the change in SSA. It was noteworthy that the recovery of finger temperature of both hands was delayed compared to that of 60-Hz vibration alone. This finding suggested that marked vascular responses through vasomotor reflexes were still continuing after cessation of the stimulus. Dupuis (1987) has reported that there was no change in the amplitude of the finger plethysmogram of the contralateral hand after local vibration alone, whereas when there was exposure to noise at 95 dB(A) together with vibration, the amplitude decreased significantly at the 1st min after the stimulus.

On the other hand, Hyvärinen et al. (1973) have reported that the amplitude of the finger plethysmogram decreased even with vibration at 125 Hz alone, and the amplitude decreased further when noise was also applied. Nakamura et al. (1987) have observed the combined effect of vibration and noise by measuring blood flow, while the present authors were the first to record the SSA directly, together with blood flow, to investigate the combined effect of vibration and noise. We think that the sympathetic reflex response to noise which is transmitted through the central nervous system and the efferent nerve response to vibration stimuli transmitted in the spine are integrated in the spine and the sympathetic ganglia. In the stellate ganglion, which controls the sympathetic discharge to the skin of fingers, there is temporal and spatial summation or occlusion of excitation. These processes make it possible for the effectors to receive multiple instructions simultaneously. Such mechanisms would explain the interpretation made of the combined effect of vibration and noise.

Raynaud's phenomenon is often induced by cold exposure (Okada et al. 1971; Taylor 1974). Therefore, the response of SSA to cold is interesting. Ekenvall and Lindblad (1986) have presented a hypothesis that the hyperresponsiveness of the finger vessels to cold is due to damage of α-1 receptors of the finger vessels by long term vibration, resulting in the predominance of α-2 receptors which are thought to participate in the response to cold. It can be suggested that even if there is no adverse response of sympathetic nerves to cold, the finger vessels themselves can show a hyperresponsiveness, if long-term exposure to vibration had already physically damaged the vessel, e.g. by producing hypertrophy of the internal membrane of the vessel.

The present study suggests that in the early stages of exposure to vibration, the increase in SSA is accompanied by an ischaemia of the fingers, which can be compensated by a reflex increase in blood flow after the cessation of the stimulus. Thus the following series of changes to explain the mechanism of Raynaud's phenomenon observed in workers using vibrating tools are proposed. At first ischaemia in the fingers is compensated by a reflex increase in blood flow after the cessation of vibration. Later, in addition to the increase of basal SSA, there is an increase in the concentration of serum noradrenaline due to long-term and repeated exposure to vibration, hypersensitivity of the smooth muscle in the vessels exposed to vibration, and furthermore, hypertrophy of the vessel walls and damage to the adrenaline receptors, combing to bring about an excessive vasoconstriction and a disorder in the vasodilatation response to cold exposure.

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