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by [Arneklo-Nobin B](#), [Johansen K](#), [Sjoberg T](#)

Affiliation: Department of Surgery, Lund University, Sweden.

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The objective diagnosis of vibration-induced vascular injury

by Birgitta Arneklo-Nobin, MD, PhD,¹ Kaj Johansen MD, PhD,² Trygve Sjöberg, BA¹

ARNEKLO-NOBIN B, JOHANSEN K, SJÖBERG T. The objective diagnosis of vibration-induced vascular injury. *Scand J Work Environ Health* 13 (1987) 337–342. For 44 patients with vibration-induced white finger and a reference group of 25 healthy men, finger systolic blood pressure (FSBP) before and after local cooling, skin temperature, and rewarming rates were determined before and after vasodilatation (body warming and alcohol). An estimation of the proportions of vasospasm and organic changes was possible, and cutaneous changes could be separated from changes in the main digital vessels. The arm blood pressure was higher for the patients and the fingertip temperatures were lower, but both normalized after vasodilatation. The FSBP values were equal in the two groups before local cooling. Afterward the patients had lower FSBP values which remained unchanged after the postvasodilatation cooling. Ten patients, all smokers, reacted with complete arterial closure after local cooling. When this group was separated from the other smoking patients, there was no significant difference between the smoking and nonsmoking patients, and the FSBP differences between the patients and referents was almost eliminated. It was concluded that, in vibration-exposed patients, injuries to skin circulation seem to be more frequent than injuries to the main digital vessels, except for some smokers, who have severe vasospasm combined with organic changes.

Key terms: cold-induced vasospasm, Raynaud's phenomenon, smoking, vibration.

The symptom of white cold fingers, as an indication of decreased digital circulation, is a common complaint which may arise from more than 40 different diseases or external agents (3, 5, 21), as shown in the following list:

A. PRIMARY RAYNAUD'S PHENOMENON (for minimal requisites see reference 1)

B. SECONDARY RAYNAUD'S PHENOMENON

Autoimmune diseases

Scleroderma-Crest syndrome
Systemic lupus erythematosus
Mixed connective tissue disorder
Thyroiditis
Primary biliary cirrhosis

Drugs

Beta-blockers
Ergotamine
Clonidine
Methysergide
Bleomycin
Vinblastine
Lithium
Methylamphetamine

Vasculitis

Polyarteritis nodosa
Polymyositis
Dermatomyositis
Thrombangiitis obliterans (Buerger's disease)
Temporal arteritis/polymyalgia rheumatica

Abnormalities of blood constituents

Macroglobulinemia
Polycythemia
Cryoglobulinemia
Cryofibrinogenemia
Cold agglutinins

Miscellaneous

Thrombosis or emboli
Atherosclerosis
Smoking
Status after B1 resection or bilateral ephorectomy

Environmental exposure or trauma

Lead
Polyvinyl chloride
Arsenic
Vibration (traumatic vasospastic disease, vibration-induced white finger)
Percussion: digital (pianists, typists), palmar (hypothener hammer syndrome)
Cold injuries, frostbite
Heat injuries
Local finger injuries

Nerve lesions or compression

Stroke
Poliomyelitis
Compression of the median nerve (carpal tunnel syndrome)
Compression of the ulnar nerve in Gyan's loge
Compression of the brachial plexus — thoracic outlet syndrome

The pathophysiology of decreased digital circulation may be exaggerated vasoconstriction, organic changes in the digital vessels themselves, or a combination of both. These changes can be induced by chronic exposure to vibration, usually on an occupational basis (4, 10, 11). The patients are often significantly hindered and occasionally incapacitated by these symptoms in their jobs or their daily lives. The clinical evaluation of these patients has been difficult because of the lack of objective means for identifying and quantitating the alterations in their digital circulation.

In this paper we present a noninvasive, reproducible, inexpensive, and accurate method for measuring finger circulation before and after vasodilating procedures in patients complaining of vibration-induced white finger. The method consists of digital plethysmography, skin temperature measurements, and calculation of rewarming rates, allowing a differentiation between vasospasm and organic vascular changes. Thus the dif-

¹ Department of Surgery, Lund University, Lund, Sweden.

² Dr Johansen was Loyal Davis Traveling Scholar of the American College of Surgeons at the time of this work.

Reprint requests to: Dr B Arneklo-Nobin, Department of Surgery, Lund University, S-221 85 Lund, Sweden.

ferential diagnosis between several forms of Raynaud's phenomenon is facilitated. The paper also deals with the possibilities of distinguishing the vascular injury induced by smoking from that induced by vibration.

Subjects and methods

A group of 44 men aged 27 to 65 (mean 44) years with a history of chronic exposure to vibrating tools (patient group) was included in the study. The entire group had been referred to the hospital because of complaints of white cold fingers, especially in a cold environment. A group of 25 healthy men, aged 18 to 70 (mean 43) years, without symptoms or signs of white cold fingers, constituted the reference population (reference group). They all met the following inclusion criteria: all agreed to participate in the study; none were receiving any cardiovascular medication; none had a history or other indications of any disease or exposure (except for vibration in the patient group) related to primary or secondary Raynaud's phenomenon. The possibility of not fulfilling the criteria was ruled out by history, by adequate blood tests (sedimentation

rate, hemoglobin, antinuclear antibodies, immunoglobulins, etc) and by a thorough physical examination.

The patients wore conventional indoor clothing and were examined in the supine position at room temperature (21–22°C) after a 20-min rest. The fingertip temperatures of all the fingers were measured, as was the arm blood pressure (ABP) and finger systolic blood pressure (FSBP) before and after local cooling of the most severely symptomatic finger. FSBP was also measured in a control finger with the use of a normal air-filled cuff. Among the referents the middle finger was used for cooling and the index finger as the control. The FSBP/ABP ratio before and after the vasodilating procedures was calculated. (See the following text.)

Cooling was accomplished by water of 15°C and of 10°C perfusing through a specially-constructed digital blood pressure cuff (15). After local cooling to 10°C, the finger cuffs were removed, and the rate of rewarming (in centigrades per minute) was calculated from the temperature curves recorded from fingertip thermocouples. All the blood pressure and temperature measurements were repeated after body warming with a heating pad placed on the abdomen for 15 min, followed by oral intake of 30 ml of 40 % ethanol during continued body warming for another 15 min. The total investigation time was 90 min.

A cold reaction value (CRV) was computed and defined as the ratio between FSBP after local cooling and FSBP before cooling corrected for any change in FSBP in the control finger during the cooling period, thus:

$$FSBP_{11}$$

$$FSBP_{10} - [FSBP_{10} (\text{control finger}) - FSBP_{11} (\text{control finger})]$$

CRV = 1 signified no change in the FSBP after local cooling; CRV = 0 meant that cooling to some temperature at or above 10°C (the critical closing temperature) results in complete digital artery occlusion.

The data were analyzed by parametric statistics with Student's t-test. Values are presented in the text as mean values and standard errors of the means.

Table 1. Fingertip temperatures and arm blood pressure values from patients with vibration-induced white finger (patient group, N = 44) and a reference group (N = 25). The measurements were performed before and after vasodilating procedures.

	Fingertip temperature (°C)				Arm blood pressure (mmHg ^a)			
	At rest		After vasodilatation		Systolic		Diastolic	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Patient group	29.4 ± 0.8		34.1 ± 0.3		132 ± 2.3		83 ± 2.2	
	↓		↓		↓		↓	
	↓		NS		↓		↓	
Reference group	32.1 ± 0.7		34.5 ± 0.3		127 ± 3.1		75 ± 2.0	

^a 1 mmHg = 133.3 Pa.

* P < 0.05, ** P < 0.01, *** P < 0.001, NS = not significant.

Results

The initial fingertip temperatures of the patients were significantly lower at the beginning of the testing

Figure 1. Ratio between finger systolic blood pressure (FSBP) and arm blood pressure (ABP) for patients with vibration-induced white finger (2) in comparison with a reference group of healthy persons without complaints (1) and the following groups of patients with other forms of cold-induced white finger (Raynaud's phenomenon): scleroderma (3), primary Raynaud's phenomenon (Raynaud's disease) (4), and carpal tunnel syndrome (5) (** P < 0.01, *** P < 0.001). For further details see reference 2.

Figure 2. Ratio between the finger systolic blood pressure before and after local cooling to 10°C corrected for changes in the blood pressure of the control finger (CRV = cold reaction value) for patients with vibration-induced white finger [divided into nonsmokers (A) and smokers (B)] and a reference, ie, control, group of healthy nonsmokers (C1) and smokers (C2) without complaints and the following groups of patients with Raynaud's phenomenon: scleroderma (D), primary Raynaud's phenomenon (Raynaud's disease) (E), and carpal tunnel syndrome (F). (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$). For further details see reference 2.

procedure (table 1). This difference disappeared after the vasodilating procedures.

ABP, in particular the diastolic pressure, was significantly higher in the patient group (table 1).

For both fingers, there was no difference in FSBP between the patients and the referents before and after the vasodilating procedures. Because of their higher ABP, the vibration-exposed group had a significantly lower FSBP/ABP ratio (figure 1).

The CRV was significantly reduced in the patients, with only a slight and insignificant increase after body warming and alcohol consumption (figure 2). Ten patients had a CRV of zero (B1 in figure 3), signifying severe vasospasm with complete arterial closure at 10°C or above. None of the referents had zero CRV values. For the patients with complete arterial closure after local cooling, the CRV values increased after the body warming and alcohol consumption. This phenomenon was not seen in the other patients or in the referents (figure 2).

All the ten patients that reacted to local cold provocation by complete occlusion of their digital arteries (CRV 0) were smokers. Further evaluation of this aspect of the patient and control groups' histories showed that, among the patients, 15 (32 %) were nonsmokers (group A) and 29 (68 %) were smokers (group B). This ratio was almost reversed in the reference group, which had 17 (70 %) nonsmokers and 8 (30 %) smokers. The smokers in the patient group with a CRV equal to zero have been denoted as group B1 and the smokers with a CRV not equal to zero are referred to as group B2 (figure 3).

Figure 3. Cold reaction values (CRV) for patients with vibration-induced white finger divided into nonsmokers (A) and smokers (B1 = smokers with a CRV equal to zero, B2 = smokers with a CRV not equal to zero) and for a reference, ie, control, group, also divided into nonsmokers (C1) and smokers (C2) (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$).

Figure 4. Rewarming rate after local cooling to 10°C for patients with vibration-induced white finger (A = nonsmokers, B1 = smokers with a cold reaction value equal to zero, B2 smokers with a cold reaction value not equal to zero) and a reference, ie, control, group of healthy persons without symptoms [divided into nonsmokers (C1) and smokers (C2)], as well as for the following groups of patients with other forms of Raynaud's phenomenon: scleroderma (D), primary Raynaud's phenomenon (Raynaud's disease) (E), and carpal tunnel syndrome (F) (***) $P < 0.001$. For further details see reference 2.

The CRV was lower in group B than in group A, but this difference was significant only after the reduction of normal vasoconstriction by vasodilating procedures (figure 2). When, however, group B2 only was used for the comparison (figure 3), no significant difference remained between the values of the vibration-exposed smokers and nonsmokers. Stated another way, the objective circulatory changes, concerning FSBP, were about the same for 75 % of the vibration-exposed group whether or not they smoked, but the remaining 25 % — all smokers — manifested severe vasospasm with arterial closure in response to cooling. That this local cold-induced ischemia could only partly be relieved by vasodilating procedures indicates the presence of both reversible vasospasm and irreversible organic vessel wall changes in the digital vessels of these patients.

The rewarming rate was slower for the patients than for the referents, and the decrease in this rate was the most pronounced for the smokers, especially for the smokers in group B1, who had the slowest recovery rate (figure 4).

Discussion

White fingers provoked by exposure to cold is a well recognized symptom of a number of disease states, as well as a sign of occupational and environmental exposures. One of the most interesting and frustrating syndromes is the episodic digital ischemia induced by chronic exposure to vibration. A number of studies have attempted to categorize this disease on the basis of age, period of exposure, type of vibrating tool, and

the adjunctive effect of cold, smoking, and other risk factors (2, 4, 6, 8, 10, 14, 20, 23).

Some investigations have failed to demonstrate any derangement in the digital circulation at all, possibly because a number of patients in these surveys have volunteered their symptoms only on request [eg, equivalent to Taylor-Pelmeur scale group 1 and sometimes 2] (6, 18, 22), which indicates that the disturbances had been mild and perhaps transient. This problem of selection bias was partly avoided in the present study, because all the patients enrolled had themselves sought medical attention because of digital ischemic symptoms. Each patient underwent a thorough clinical examination to rule out organic causes other than chronic vibration exposure for their symptoms (most commonly, in our experience, connective tissue disorders, thoracic outlet syndrome, and beta-blocker medication).

Hitherto, the recording of rewarming time and the measurement of finger blood flow or blood pressure with plethysmographic or Doppler methods have been considered the most accurate means for diagnosing and quantitating circulatory disturbances in patients with white fingers (6, 7, 9, 12, 17, 23). However, the failure of most of these and other commonly accepted vascular physiological techniques to provide clear delineation of the changes in all patients with vibration-induced white finger suggests that marked injury to the circulatory system does not result from vibration exposure in contemporary experience. The present study thus attempts to provide a more sensitive means of differentiating the objective circulatory changes that occur in this group of patients.

Digital ischemia after vibration exposure does not necessarily result in episodic complete closure of the main digital arteries. Since vibration is a form of trauma to the skin, it is logical to assume that many of these patients have injuries restricted to the skin and its blood vessels. Indeed, the majority of the patients in this study (75 %) had no significant change in FSBP after local cooling, a finding suggesting the presence of normal main digital arteries. However, the fact that they had decreased fingertip temperatures, normalizing after the vasodilating procedures, suggests hyperactivity in the sympathetic nerves to the skin blood vessels, as proposed by others (8, 16, 19).

Digital rewarming time has been reported to be prolonged even in asymptomatic persons exposed to vibration and thus has been proposed to be one of the most sensitive physiological tests for diagnosing vibration-induced vascular injuries (13, 24).

In the current study, the rewarming rates after local cooling were also markedly slower in the vibration-exposed patients than in the referents, even after vasodilating procedures. This finding suggests the possibility that organic skin changes have resulted from chronic vibration exposure. Since the simultaneously measured FSBP was normal in 75 % of the patients, their white fingers cannot be secondary to a hyper-reactivity of the main digital arteries but rather an isolated decrease in skin circulation. An interesting observation was that the patients, but not the referents, experienced pain during the cooling procedure, something not occurring in patients with other types of more severe vasospastic disorders. This result suggests the possibility that chronic vibration induces a cutaneous neuropathy. In fact, studies have suggested that chronic vibration exposure leads to substantial changes in vibration sensitivity in the digits (14).

Twenty-five percent of the patients in this study had severe vasospasm, resulting in complete occlusion of the blood flow (CRV 0) during local cold provocation. The laboratory values for this group, even on an individual basis, were clearly separable from those of the referents and from patients without arterial closure at cold provocation. We conclude from this finding that, if cold cannot provoke a zero CRV value in vibration-exposed patients, little will be gained from measuring blood flow in the main digital arteries, since the values will not differ from those of normal subjects. The reason for this lack of discriminatory power of the method is that the symptoms arise primarily from episodic vasoconstriction, which might be isolated to cutaneous vessels due to skin injuries, or neurological disturbances rather than being permanent changes in the arteries.

The objective measurements of the digital circulation presented allow an estimation of the contribution of vasospasm and permanent vessel-wall changes to the symptom complex of a patient. A CRV value of 0.42 after the vasodilating procedures would imply that the remaining 58 % of reduced digital perfusion is

caused by organic vascular changes. This calculation may be of interest for the evaluation of vasodilator therapy and for the prognosis of the reversibility of vasospastic disorders in general.

An additional, seemingly crucial, observation was that all patients with complete closure of their arteries after local cold provocation were smokers. In our measurements this group (B1) could be clearly distinguished from the smokers in group B2 and the reference group. Although smoking cannot alone explain the alterations in vascular function seen in some persons with chronic vibration exposure, it clearly plays a role in the disorder and must be taken into account when such patients are evaluated.

Vibration injury probably not only affects the cutaneous arteries, but also the superficial veins and the main deep digital arteries. An acceptable objective diagnostic method must assess the circulation in all these areas. The method currently presented not only appears to be accurate, reproducible, and atraumatic, it also separates organic from vasospastic phenomena, as well as isolated cutaneous changes from those induced in the main digital vessels. If no circulatory changes can be demonstrated with this method in a patient with a persuasive symptom complex, more extreme cold provocation, for example, with total body cooling, might be considered.

The method allows patients to be followed over time, and it may be useful not only for demonstrating the natural history of the disease, but also for showing the impact of various therapeutic maneuvers. Avoidance of further vibration exposure, of smoking, and of cold environments are hallmarks of current therapy for vibration-induced white finger. The further possibility of benefit from vasodilator treatment — as we have demonstrated in patients with other forms of vasospastic disease — could be quantitated by the serial measurements of digital perfusion according to the described method.

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