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Pathophysiology and multifactorial etiology of acquired vasospastic disease (Raynaud syndrome) in vibration-exposed workers

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GEMNE G. Pathophysiology and multifactorial etiology of acquired vasospastic disease (Raynaud syndrome) in vibration-exposed workers. *Scand j work environ health* 8 (1982) 243–249. The article reviews available pathophysiological evidence for a multifactorial etiology of the Raynaud type of peripheral circulation disorder in persons exposed to vibration from handheld tools and discusses the consequences this viewpoint may have for diagnostics, preventive work, and research.

Key terms: cold, cold provocation test, ergonomic factors, noise, stressors, sympathetic nervous system, traumatic vasospastic disease, white fingers.

Vibrations from handheld tools are considered to be one of the causes of the development, in certain individuals, of a tendency to react to cold with attacks of finger blanching and decreased sensibility of the skin (Raynaud phenomenon or *Raynaud syndrome*). The physiological condition behind the symptoms is an abnormally strong and lasting local constriction of skin vessels within the fingers, a "vasospasm." Since vibration is believed to cause traumatization, which leads to illness, the Raynaud syndrome *(secondary Raynaud)* in vibration-exposed individuals has been given the name of *traumatic vasospastic disease* (TVD). Popularly it is often called *white fingers*.

In advanced cases, the attacks cause difficulties in carrying out the job because of unpleasant feelings and the clumsiness resulting from the decrease in sensibility and stiffness of the fingers and hands. The disorder also negatively influences life quality outside the job by, eg, imposing restrictions on certain outdoor activities.

At present, there is no therapy with lasting effect. The disorder progresses until the causative agents are eliminated. If that occurs, the symptoms disappear in most cases within months or a year. If the exposure continues, the disturbances lead, in some cases, to a handicap which necessitates transfer to another job. The prevalence of the Raynaud syndrome in industry seems to be considerable among certain repair shop mechanics who use grinding and chipping tools. In other professions, improvements in the work environment have resulted in decreased prevalence.

In some individuals, the activity level of the sympathetic nervous system is more or less constantly elevated. This elevation results in an abnormally strong vasoconstrictor tone in the skin arterioles (stronger than motivated for reasons of conservation of body heat). Even in a moderately cold environment, or with emotional triggering, this may lead to intermittent, mostly generalized pallor, often symmetrical, of fingers and toes. This symptom is the so-called *constitutional Raynaud*. Such a condition was said to occur in about 6 % of a group of British miners without vibration exposure (4) and in about 2 % of French, nonexposed agricultural workers (2). A strong reduction in local finger blood flow, lasting for more than 20 min – with a decrease in sensibility and generalized pallor but without marked *local*

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finger blanching – has however been shown (10) to occur in an unexpectedly large proportion of subjects after cold provocation (hands for 2 min in water 7–10°C, without pain). Out of 170 healthy, job-active aviation repair shop mechanics, aged 25–69 a, without exposure to vibration and cold and with only low levels of noise in their environment and no heavy manual work, 37 subjects (22 %) showed the tendency described.

Individuals with constitutional Raynaud can more easily than others pass the threshold where blood flow is so severely reduced that finger pallor occurs (fig 1). Constitutional (and transient, periodic) differences in the level of sympathetic activity is the most likely explanation for the well-known observation that the sensitivity to injurious influence from vibrations seems to vary widely within one and the same professional group.

The width of finger skin vessels is regulated chiefly by changes in the *sympathetic efferent activity,* already discussed, but it is also influenced by *local factors* (12, 18) and by *purinergic mechanisms* (3).

Pathogenic theories

The pathogenic mechanism is not clear. There are chiefly three plausible theories (fig 2; the mechanisms presented in the illustrations are oversimplified). The physiological processes involved in the peripheral circulation are complex, and the reactions of the organism in response to such environmental factors as noise, cold, and vibration are insufficiently known.

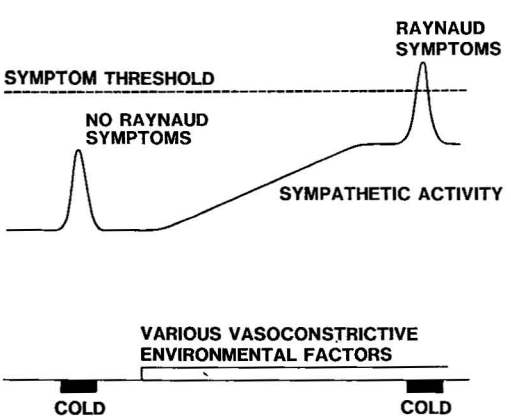

Fig 1. Schematic illustration of the triggering of cold-induced Raynaud symptoms at different levels of sympathetic nervous activity.

One theory (20, 21) postulates a *growth of the smooth muscle layer in the vessel wall.* This growth is caused by often repeated contraction – mediated by Pacinian corpuscles – during a long time, and it is induced by vibration from hand-held tools. The resulting *hypertrophy* reduces the lumen of the vessel (fig 2, B1). Cold stimulation of a certain magnitude therefore leads to a proportionately stronger reduction of the lumen in such a vessel (B2) than in a normal vessel (A2), whereby the blood flow is decreased to a degree where the skin no longer retains its normal, pink hue (finger blanching).

The hypertrophy theory is also part of a second model, which, physiologically, contrasts with the first-mentioned one (25). Local vibrations have been shown to cause relaxation of the smooth muscle in the blood vessel wall (16). During the vasodilatation which, according to this hypothesis, is induced by vibration from the tool the muscle metabolism of the vessel wall remains however high. If this condition occurs repeatedly for a long time, the muscular tissue will become hypertrophic, which leads to lumen reduction.

A *functional change* in the vasoregulatory system, manifesting itself as *sympathetic, reflex-mediated hyperactivity* (at least partly believed, as it seems, to be based upon anatomic alterations in nervous tissue) is another possibility, discussed by Soviet researchers [see, eg, the report of Drogitchina & Metlina (7)]. According to these theoretical considerations, sympathetic hyperactivity, released from the central nervous system under the influence of hand and arm vibration, would result in a lumen reduction that is abnormally great in relation to physiologically motivated needs. This condition, in connection with cold stimulation, is illustrated in fig 2 (C2).

There is no strong evidence for any one of the described theories, which however seem at least possible from a physiological point of view. They may all contribute to the development of the Raynaud syndrome in vibration-exposed workers.

Etiologic factors other than vibration

The proposed pathogenic theories have something in common which constitutes

the basis for the approach followed in the present review, ie, efferent sympathetic activity resulting in abnormally strong and longlasting vasoconstriction. It follows that also stimuli other than vibration, which can elicit sympathetically mediated peripheral vasoconstriction, must be taken into account as possible etiologic factors.

At intensities above 70 dB, *noise* has been shown to be an effective vasoconstrictor (15) to which there is no or only slight adaptation (5). Strong noise, in the form of recorded sound from chain saws in combination with vibration and cold, has been shown to elicit stronger vasoconstriction in the fingers than vibration and cold only (21).

Jansen (14) showed a significantly higher prevalence of "vessel disturbances" and skin pallor among workers exposed to strong noise than among a reference group with low noise exposure. In the first-mentioned group 28 % (184 out of 669 workers) complained of finger blanching ("Absterben der Finger") or paresthesia in the extremities, while the corresponding figure for the group with low noise exposure was 16 % (54 out of 336). The prevalence of circulatory disturbances was particularly high among persons working with vibrating tools (pneumatic chisels). The difference between the two exposure groups, however, proved to be "significant" also after these workers were excluded from the high noise exposure group. The author comments upon this result as follows: "Thus the question arises whether the circulatory disturbances among finishers (Putzer) – hitherto considered to be induced by vibrations – may not primarily be an effect of noise exposure, or that the two influences in combination may have resulted in a high incidence of these symptoms [p 256]."

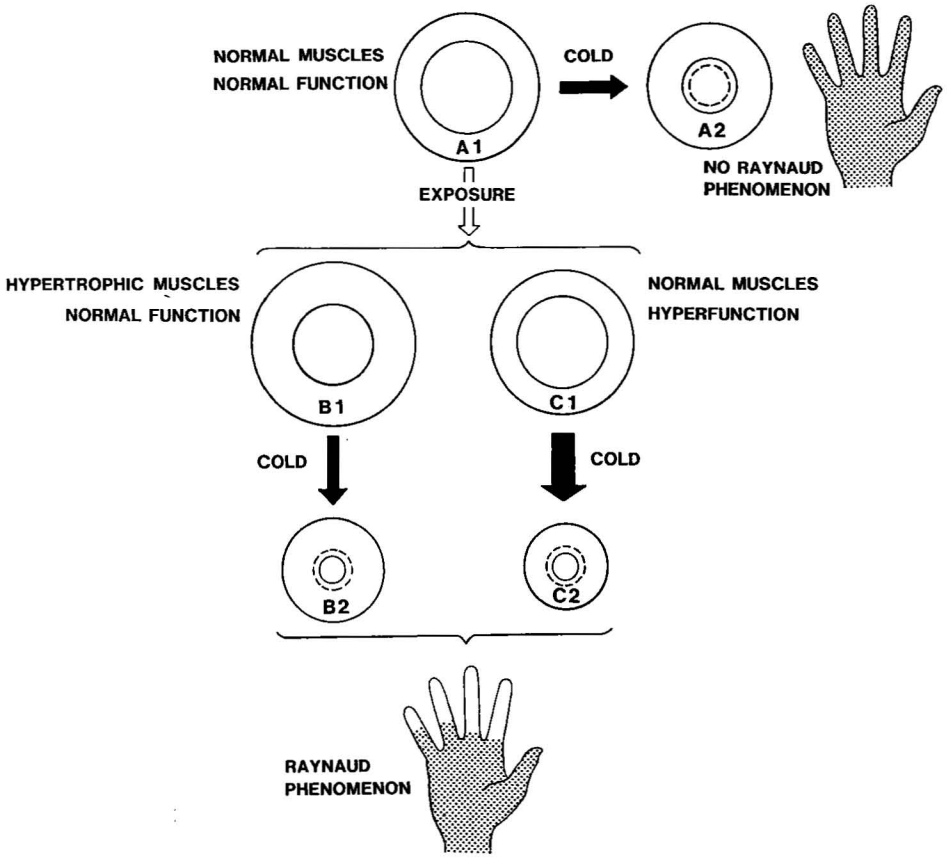

Fig. 2. Schematic illustration of proposed pathogenic mechanisms in the development of the Raynaud syndrome. The width of the solid arrows symbolizes the strength of vasoconstriction in response to cold exposure of a certain degree. Broken circles represent the mininum lumen size above which there is no Raynaud phenomenon.

The physiological data presented, together with the experimental and epidemiologic findings, seem to constitute a strong case for the etiologic importance of noise in the development of the Raynaud syndrome.

Cold is the natural symptom-eliciting environmental factor in cases of vasospastic disturbance in vibration-exposed persons. Against the background of the pathogenic models described, cold must therefore be included in the list of possible etiologic factors. As such, it occurs to a varying degree in many of the workplaces where vibrating handheld tools are used. In particular, vasoconstriction in the hands cannot only result when the hands are cooled directly, but also when certain other parts of the body are exposed to cold, especially the face and forehead (13), and when a person experiences a sense of general chilliness.

Many job tasks involving vibration exposure require *hard muscular work.* The great demand for adequate blood supply to the skeletal muscles is partly met through a redistribution of blood from the skin of the area (1, 23). During this process there is initial vasoconstriction in some parts of the vascular bed of the finger skin, repeated each time a new grip is taken around the tool after a period of muscle relaxation. An overall increase in sympathetic efferent activity (24) and a decrease in finger blood flow (22) are caused by *strong physical exertion in the erect position.*

Vasoconstriction in some parts of the vascular bed of the finger skin is also elicited by the blood flow arrest caused by a *hard, maintained grip around a tool* (17). A positive correlation exists between the occurrence of white fingers and a great strength of hand grip (8).

Occlusive changes in the arteries of the hand have been shown (26) to occur with higher frequency in vibration-exposed workers and in persons doing hard manual work than in nonmanual workers. There was no difference in prevalence between the two first-mentioned groups.

Thus, there are different kinds of arguments pointing to the possible etiologic role played, in particular, by a strong grip around a (vibrating) tool handle. The hemodynamic changes previously mentioned, known to take place when a hand grip is applied, and the quoted finding of a positive correlation between a strong hand grip and Raynaud prevalence join forces with the functional-anatomic data just presented. Together they emphasize the etiologic importance that a strong hand grip may have for the development of white fingers in vibration-exposed workers.

Declivity (ie, the fact that a part of the body – in this discussion the hand – is held considerably lower than heart level) is sometimes an essential ergonomic component in vibration-exposed work; furthermore, it often occurs together with hard muscular work, a hard maintained grip around a tool, and strong physical exertion in the erect position. Declivity leads to a temporary vasoconstriction in precapillary resistance vessels and precapillary sphincters, effects mainly myogenic but possibly also adrenergic in nature (19).

The large group of *stressors* comprises not only emotional stimuli, but also such physical factors experienced as a strong load upon the organism. The influence of stressors (including the experience of frustration and dissatisfaction with conditions within the work environment, as well as certain psychosocial factors outside the job) results in a contraction, mediated by sympathetic efferent activity, of the smooth muscles in the blood vessel walls of the finger skin (6, 17).

Mechanisms of *conditioning* actually find their strongest expression in cardiovascular reactions to physical stimuli alone or together with emotional stimuli (9). In a work environment where vibration-exposure takes place, machine noise may serve as a conditioned stimulus and elicit peripheral skin vasoconstriction even in the absence of vibration (as unconditioned stimulus) or together with relatively weak vibration. These mechanisms may operate for instance in workplaces where good vibration damping has been achieved and other vasoconstrictive environmental factors have been eliminated.

The importance of the *potentiation* mechanisms involved in an interaction between two or more environmental factors – possibly coupled with conditioning – is practically unknown. Nevertheless it must necessarily be listed as

a very likely mechanism in this connection. If a clear understanding of the role of conditioning and potentiation in vibration work could be obtained, it would of course be of great interest for preventive work.

Differential etiologic diagnostics

In all likelihood there are individuals with long periods of *elevated sympathetic activity* caused by influence from one or more vasoconstrictors in their total environment (fig 1). It may be that these persons, together with individuals with a constitutional Raynaud tendency, comprise the majority of a vibration-exposed group showing the Raynaud syndrome. This is an attractive approach; the reverse situation, a *low* level of sympathetic activity (constitutional or existing for a limited period), could be the explanation for the noteworthy fact that certain individuals do not develop the Raynaud syndrome at all, despite a strong and longlasting exposure to many of the environmental factors discussed.

There are *other individual factors* which can modify the influence of vasoconstrictive environmental factors. In cold provocation experiments (10) on subjects without vibration exposure, the following factors were found to correlate with finger temperature: age, body surface, and certain factors directly related to circulation.

Age and *body surface* showed a complex relationship to some phases of the temperature reaction to cold. There was also a correlation between skin temperature after cold provocation and *certain factors directly related to circulation*, ie, strong smoking habits, constitutional Raynaud tendency (frequent sense of cold in fingers and toes), arteriosclerosis, and hypertension. Among the aviation repair shop mechanics mentioned previously there was, furthermore, a correlation between a disturbance in peripheral nerve function – measured as an elevated vibration threshold on the dorsum of the hand (os metacarpale II) – and a slowing of the restitution of blood flow after cold provocation.

It should be emphasized that a positive result in a *cold provocation test* only affirms the presence of a peripheral circulatory disorder, but it does not give any information about the *etiology* of this disturbance. In many investigations using cold provocation tests, there has also been a very weak correlation between the result of the test and the history of subjective symptoms, particularly often falsely negative results. It must be concluded from past experience, documented in the literature, that there is no scientific justification for not accepting anamnestic data on Raynaud attacks because a cold test failed to provoke such signs.

Nor do *other clinical methods for the study of peripheral circulation* give any information about what *environmental* occupational factor(s) may have resulted in the development of the Raynaud syndrome. Such examination methods as plethysmography and various thermometric techniques should, however, not be left out of the clinical investigation, since their results may reveal the presence of pathological conditions that might be treated (for instance, arterial obstruction). Anatomic-orthopedic conditions (for instance, "thoracic outlet syndrome" and carpal tunnel syndrome) and autoimmunologic diseases, as well as endocrinologic disorders, should be taken into consideration with the help of clinical laboratory methods.

Conclusions

Clinical investigators of the Raynaud syndrome in patients exposed to vibration have, traditionally, almost exclusively concerned themselves with vibration as the causative agent. There are, however, strong physiological reasons for the view that the peripheral circulation disorder ("white fingers"), which often occurs in persons working in a job where vibrating handheld tools are used, has a *multifactorial etiology*.

Noise, cold, ergonomic factors (hard muscular work, a hard maintained grip around a tool, declivity as a component of the work posture, etc) should be given as serious a consideration as vibration. Emotional stimuli and other stressors of the total psychosocial situation, as well as conditioning and potentiation, may also be of importance for the develop-

ment of the Raynaud syndrome. Constitutional Raynaud should also be considered, its prevalence having possibly been hitherto underestimated. Several other individual factors, eg, smoking habits, age, and body surface, together with certain factors directly related to circulation, are part of the picture, although less well understood.

Probability diagnosis and terminology

There is a long list of potential vasoconstrictive factors and mechanisms in the environment of persons who work with handheld vibrating tools. To make the Raynaud syndrome in such individuals equivalent with "vibration injury" is scientifically unsatisfactory. The situation seems to indicate a need for a change in terminology; "acquired vasospastic disease" would be a better diagnostic alternative. The *diagnosis,* in all cases, must be founded upon the subjective probability that an environmental factor, alone or in interaction with other factors, has caused the disorder. From the point of view of the patient it is essential that the diagnosis – and the measures taken in the patient's work environment – should not be exclusively dependent on the decision of whether or not vibration exposure has been great enough to cause the symptoms. In those cases in which the cause is judged to be (specified or nonspecified) factors present in the work environment, the connection can be indicated by a suitable choice of words, for instance, "occupationally acquired vasospastic disease." The diagnosis can best be made by industrial physicians or occupational medicine clinicians and should be founded upon knowledge of what vasoconstrictive factors have been present in both the patient's occupational and nonoccupational environment.

Other factors relevant for the total evaluation of an individual case have been discussed elsewhere (11).

Prevention and research

What has been presented in this review should be of consequence for *preventive work* and *research* and should lead to an altered basic approach and the allocation of resources. In the improvement of workplaces, more attention should be paid to exposures other than vibration, particularly noise, cold, certain work ergonomic conditions, and psychological factors.

In research, projects should be supported that are based upon the multifactorial viewpoint presented in this communication, and they should aim at identifying high risk individuals. For such a pursuit, prospective investigations would be of great value. Thus it would be of interest to study certain groups of workers with respect to exposure data (not only vibration), medical parameters and clinical laboratory tests, and psychological factors, from the time when they enter vibration-exposed work in a vocational training school. Of great importance, too, would be investigations on the prevalence of the Raynaud syndrome within professional groups with homogeneous vibration exposure but with different degrees of exposure to other factors, for instance, noise, cold, and/or static work load, as well as stressors of various kinds.

The evidence that noise, as well as a hard grip around a tool and other (correlated) ergonomic factors, may be important for the development of white fingers in vibration-exposed workers is strong enough to merit testing in exposure-controlled laboratory experiments and prospective field investigations.

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