RESEARCH INTO HAND-ARM VIBRATION SYNDROME AND ITS PREVENTION IN JAPAN

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ABSTRACT

Research on vibration syndrome in Japan began in the 1930s with studies of the disorder among railway, mining and shipyard workers. In 1947, the Ministry of Labor decided vibration syndrome among operators of rock drills and riveters etc. was an occupational disease. Industrial developments in the 1950s and 1960s promoted the survey of vibration syndrome in mining, stone quarrying and forestry. The Ministry of Labor (1965) and the National Personnel Agency (1966) legally recognized vibration syndrome among chain saw operators as an occupational disease. Guidelines for prevention and early therapy were issued in the 1970s and 80s. From the late 1970s into the 1980s, research focused on the clinical picture, diagnostic methods and therapy. In pathophysiology, advances were made in research into the autonomic nervous system during the 1980s. The 1970s and 80s saw a steady reduction in risks from technological change and working conditions, and advances in medical care, education and meteorological forecasting. A comprehensive prevention system established in the 1980s in the Japanese forest industry involved: 1) work restrictions, 2) an improved health care system, 3) advances in the design of vibrating tools, handle-warming devices, and 4) improved worker education. This comprehensive preventive system was legally introduced into other industries, resulting in a rapid decrease in the incidence of vibration syndrome in Japan.

Key Words: Vibration, Prevention, Clinical picture, Pathophysiology, Autonomic nervous system

VIBRATION SYNDROME IN JAPAN

Early cases of vibration syndrome in a railway factory were described by Murakoshi (1933), and, among miners, by Ishinishi (1939). European research results were reported in a book by Koinuma (1934). Next came more detailed papers by Matsufuji on railway factory workers (1942), and Kimura on shipyard workers (1943). Matsufuji, who worked in the Ministry of Railways, may be credited with some of the most valuable epidemiological studies and clinical observations made prior to World War II in Japan. However, vibration syndrome was still not included in the list of occupational disease, at that time.

1. Legal recognition of vibration syndrome as an occupational disease

In 1947, a new Labor Standard Law listed twenty-eight occupational diseases in Article 35 of “Detailed Regulations for Enforcement of the Labor Standard Law.” Item 11 was “disease by work which gives severe vibration to a worker’s body.” Symptoms of this disease were described as: disturbances of the vasomotor and sensory nerves; disorders of joints, tendons and muscles; and subjective symptoms such as fatigue, sleep and digestive disturbances. Work causing this disease included such occupations as operating pneumatic rock drills, air riveters, etc. Descriptions were based on Japanese and European reports on vibration syndrome prior to World War II.

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The late 1950s witnessed the revitalization of Japanese industry, including major changes in the production system. Many portable power tools were introduced into manual operations, resulting in a rise in vibration syndrome among many workers in shipyards, steel mills and automobile factories. Miura reported these cases in detail (1955) and Matsuoka reported mining cases. Miura tentatively proposed a preventive guideline for vibration exposure on the basis of his extensive research (1959). Many reviews by Miura laid the groundwork for the next generation's research. In 1961, Matsufuji reviewed Japanese surveys and introduced a book by the famous Soviet researcher Andreeva-Galanina, published in 1956.

In the late 1950s, chain saws were introduced into forest industry and severe cases of vibration syndrome occurred in the national forests of central Japan. Chain saw operators in this area called this disorder “white waxy disease” due to its white color, coldness and a loss of dexterity in the fingers. Our long-term research began in this area.

In May 1965, based on our research and recommendations, the Ministry of Labor decided that vibration syndrome among chain saw operators in private forests would become Item 11 in the list of occupational diseases. In the following year, the National Personnel Agency reached the same decision for government workers. These decisions sparked the development of research, and administrative management for the prevention of vibration syndrome also began at this time.

2. Legal guidelines for physical examinations, clinical inspections and interviews for vibration syndrome

Physicians in Japan use guidelines for the diagnosis of vibration syndrome and other occupational diseases. This diagnosis involves a physical examination, clinical inspection and interview by questionnaire.

The first Ministry of Labor guideline for vibration syndrome was issued in 1971 on the advice of the Research Committee of the Japan Association of Industrial Hygiene (Chairman, T. Miura). It stipulated examination methods for peripheral circulatory, nervous, and musculoskeletal functions as well as directives on clinical inspections and questionnaire interviews regarding subjective symptoms. The Committee on Special Medical Examination for Diagnosis of Vibration Syndrome in the Private Forest Industry (Chairman, T. Miura) was organized in 1972. S. Yamada headed a subcommittee on physical examinations to be conducted in six regions of Japan. The Committee recommended guidelines on health care and work regulations to the Ministry of Labor which issued a second version of the guidelines in 1976 on advice of the Committee. Therapy guidelines were issued in 1986, covering vasculatory and sensory neural disorders and subjective symptoms.

3. Problems in clinical picture, pathophysiology and pathogenesis

Initial research from the late 1960s to the early 1970s concentrated on early diagnosis of disorders arising from vibration exposure. The clinical picture for diagnosis was dysfunction in the peripheral circulatory, nervous and musculoskeletal systems. Investigation into severe cases of vibration syndrome encouraged studies in clinical therapy and pathophysiology from the late 1970s onward. This research centered on such problems as whether nerve impulse issued from hand by vibration exposure mediated the central nervous system. If such were the case, did this give rise to dysfunctions in other tissues, or even in the central nervous system itself? Attention then began to focus on the autonomic nervous system.

In the 1980s, many clinical observations were recorded, and neurochemical and neurophysiological methods were introduced in vibration syndrome research. Study of the role of autonomic dysfunction in vibration syndrome made steady progress. As the research evolved, discussions on the clinical picture, pathophysiology and pathogenesis focused on the following problems:
VIBRATION SYNDROME IN JAPAN

[Clinical picture]
1) What are the symptoms of vibration syndrome? Which body systems are involved?
2) How does vibration syndrome develop? What index reasonably explains the development process?
3) How is the correct stage of vibration syndrome determined?
4) How does recovery take place after vibration exposure? What factors are involved in recovery?
5) What symptoms are important in therapy?

[Pathophysiology and pathogenesis]
1) What factors are fundamental to the pathogenesis of vibration syndrome? What factors modify the clinical picture?
2) Does a dose-response relationship exist between cumulative vibration exposure and abnormal findings in examinations and symptoms?
3) Does the effect of vibration appear on the non-exposed areas of the body? What mechanism is involved?
4) Does the combined effect of vibration and noise reinforce vasoconstriction? Does the central nervous function have a reinforcing role?
5) Does the combined effect of vibration and cold increase susceptibility to cold in the hypothalamus? Does the decrease in peripheral skin temperature strengthen the nerve impulse to the cold receptor of the hypothalamus?
6) Do vibration, noise and cold act as stressors to cause functional change in the brain limbic system?
7) Does the pathogenesis of VWF involve peripheral and central factors? Do chills and a decrease in finger skin temperature necessarily precede the occurrence of Raynaud's phenomenon?
8) Why do patients with both VWF and numbness show more severe syndromatic changes than patients without them?
9) Are subjective symptoms, such as heavy headedness and irritability involved in vibration syndrome?
10) Are there individual differences in susceptibility to vibration, noise and cold in vibration syndrome?
11) Some researchers in Europe suggest that Soviet theory has influenced some Japanese research. Is this true?

AUTONOMIC NERVOUS SYSTEM IN VIBRATION SYNDROME

1. Theories concerning the autonomic nervous system
For a long time, two theories concerning the actual site of vibration syndrome were discussed in Japan. The local theory posits that disturbances in circulatory, nervous and musculoskeletal function are limited to the hand and arm. The whole-body theory holds that such symptoms of vibration syndrome first appear in the hand and arm, and then circulatory disturbances propagated to other body sites via autonomic nervous system mediated by vibration, cold and noise exposure. These circulatory disturbances then cause nervous disturbances in other body sites. Guidelines for therapy from the Ministry of Labor (1986) were based on the local theory, and the Supreme Court also decided a 1990 case of VWF workers on the basis of that theory. Nevertheless, research into the whole-body theory (which had continued from the 1980s into
the 1990s) produced the following findings.

Acute vibration exposure by one hand is accompanied by 1) an increase in skin nerve sympathetic activity (SSA) in the opposite hand,\(^1\) and 2) by increased perspiration, a drop in skin temperature and increased SSA in the leg.\(^2\)

In chronic vibration exposure, dysfunction in non-exposed site far from the hand was investigated in terms of autonomic dysfunction. Such dysfunctions included 1) polyneuropathy in the upper and lower limbs,\(^3\) 2) decreased hearing in workers with VWF,\(^4\) 3) coldness and decreased skin blood flow in hands and feet with no other disease,\(^5,6\) 4) pathological change in the blood vessels at the fingers and toes in workers with VWF,\(^7\) and 5) Raynaud's phenomenon in the fingers and toes without other diseases.\(^8\) From the neurological viewpoint, research was conducted into: 6) change of R-R interval as proof of hypofunction of the parasympathetic autonomic nervous system;\(^9,10\) 7) increase of neurotransmitter (NA: noradrenalin) in whole-body cold exposure in workers with VWF as proof of sympathetic autonomic hyperfunction in cold;\(^10\) 8) hypersensitivity to cold and chill in VWF workers; 9) the role of hypothalamus in mediating autonomic nervous system in long term vibration and cold exposure;\(^12\) 10) prolonged P300 as proof of dysfunction of the central nervous system;\(^11\) and 11) psychosomatic responses on the effects of three stressors, i.e., vibration, noise and cold.\(^13\)

These studies, including 1) to 7) and 11) are described in other papers in this volume. Research works 8) and 9) are described herewith.

2. Hypersensitivity to cold

VWF workers frequently complain of hypersensitivity to cold which takes such forms as ease in catching colds, susceptibility to chills after bathing, etc., need to wear many layers of clothing, keep their feet warm at night, etc. Table 1 shows the prevalence of hypersensitivity to cold as a subjective symptom by age and the years of vibration exposure among chain saw operators. As operator age, and duration of vibration exposure increase, so do the subjective symptoms of hypersensitivity. Long-term vibration exposure increases the rate of hypersensitivity to cold among operators in their 40s and 50s. But among those in their 30s, this tendency is not observed. Statistical differences were found between the 30s and 40s, 30s and 50s (age), below 5 years and over 10 years of chain saw operation. These results mean that hypersensitivity to cold has a close relation to age and vibration exposure.

Table 2 shows a significant relation between hypersensitivity to cold and VWF (p < 0.001). The prevalence of VWF among workers with hypersensitivity to cold was higher (74.40%) than in those without such hypersensitivity (44.1%). This result suggests that hypersensitivity to cold has a close relation to VWF.

<table>
<thead>
<tr>
<th>Table 1. Prevalence of hypersensitivity to cold by years of chain saw operation and age (among private forest workers, 1977) (* p&lt;0.05)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chain saw operation</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>below 5 years</td>
</tr>
<tr>
<td>5~10 years</td>
</tr>
<tr>
<td>over 10 years</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

\( p<0.05 \)
Table 2. Hypersensitivity to cold and VWF
(among chain saw operators in private forests, 1976)
($ \chi^2 $ 33.9 (1): p < 0.001)

<table>
<thead>
<tr>
<th>Hypersensitivity to cold</th>
<th>VWF (+)</th>
<th>VWF (-)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypersensitivity to cold (+)</td>
<td>99 (74.4%)</td>
<td>34 (25.6%)</td>
<td>133 (100.0%)</td>
</tr>
<tr>
<td>Hypersensitivity to cold (-)</td>
<td>132 (44.1%)</td>
<td>167 (55.9%)</td>
<td>299 (100.0%)</td>
</tr>
<tr>
<td>Total</td>
<td>231 (100.0%)</td>
<td>201 (100.0%)</td>
<td>432 (100.0%)</td>
</tr>
</tbody>
</table>

Worker skin temperature is altered during chain saw operation. Handle gripping constricts blood vessel, decreasing blood flow to the fingers. Exposure to cold and vibration causes vasoconstriction. Combined effect of cold, vibration and handle gripping causes a significant drop in blood flow and skin temperature, both of which rapidly revert to normal when the handle is released (Fig. 1). In healthy workers, these changes clearly appear during chain saw operation in both cold and hot weather. But in VWF workers, skin temperature remained at a lower level than that of the healthy control group, especially in cold weather. In hot weather, changes were the same in both groups. It seems that the VWF workers have an abnormal sensitivity of small arteries to cold and vibration as well as hypersensitivity to cold.

VWF chain saw operators frequently had VWF attacks while riding motorcycles in cold weather. We observed the process of attacks as follows:

Color changes in the finger nails as well as in the finger skin proceeded as follows when
riding a motorcycle: at first nails turned white. Small white spots then appeared on the skin. The radius of these spots broadened, abruptly fusing into a single large spot resulting in white finger skin bordering the other skin. When I touched the nail of an unaffected finger, it suddenly turned white. Mechanical stimuli seemed to provoke vasoconstriction in the nail bed. Based on this observation, I devised the nail-press test.\textsuperscript{15)}

In this test, the subject's finger is submitted to 10 seconds of pressure between the tester's thumb and second finger. Using a stopwatch to insure that 10 seconds have elapsed, pressure on the nail is released. The nail will immediately begin to recover its normal color. When normal coloration is restored to the entire nail, the stopwatch is turned off. The time from the release of pressure to the complete disappearance of any whiteness is referred to as the "nail-press test value." That value depends on the degree of constriction in the small arterioles and on the flow rate of arterial blood into the capillaries. A high value indicates severe constriction of the small arterioles.

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Fig. 2. Change of color in finger skin and nail bed before onset of white finger.

1 \textup{↓} \textup{↑} Nail color turns white
2 \textup{↓} Small white spots appear on the finger skin
3 \textup{↓} Finger skin color turns white with boundary

Four chain saw operators with VWF and control workers remained outdoors in 5°C weather. After 20 minutes, 3 of them complained of a chill in their backs. The fourth felt a severe chill and "white finger" appeared in his hand. In this process, drops in skin temperature were greater than control. Their nail-press test values were over 10 seconds, whereas values in the control group range from 3 to 6 seconds after 40 minutes (Fig. 3). High nail press test levels suggested chill as well as vasoconstriction in cold.

One cold morning, a VWF worker mentioned that because his body felt warm, he did not expect to see "white finger" when riding his motorcycle. This suggested that the degree of experienced body warmth or chills is closely related to the appearance of VWF. Tests with riders wearing thick and thin clothes were then devised.

After beginning to run at a speed of 30 km/hour, their finger skin temperature rapidly decreased by severe cold exposure. Difference of the fall in skin temperature while riding between thin and thick clothes was greater in control than in VWF workers. This result due to hypersensitivity to cold in VWF workers, caused intense vasoconstriction even in the thick clothes.
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In VWF riders wearing thin clothes, VWF appeared in two workers after 5 minutes, another after 10 minutes and in yet another after 20 minutes. All of them complained of chills prior to the onset of VWF. In thick clothes, however, VWF did not appear at all.

Group differences regarding changes in nail-press test values, although significant among thickly clad riders, were even more marked among riders in thin clothes. After riding they stood still outdoors for twenty minutes. Recovery in skin temperature and nail-press test value occurred much later in the VWF than in the control group.16)

Table 3. Change of skin temperature and nail press test value by motorcycle riding (at 5°C)

<table>
<thead>
<tr>
<th>Examination item</th>
<th>Wearing</th>
<th>Subject</th>
<th>Number</th>
<th>before riding</th>
<th>5 minutes riding</th>
<th>10 minutes riding</th>
<th>20 minutes riding</th>
<th>10 minutes standing</th>
<th>20 minutes standing</th>
</tr>
</thead>
<tbody>
<tr>
<td>VWF</td>
<td>thin clothes</td>
<td>VWF</td>
<td>4</td>
<td>onset: 2 none</td>
<td>onset: 1 continues: 2 none</td>
<td>onset: 1 continues: 2 none</td>
<td>onset: 1 continues: 2 none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td></td>
<td>thick clothes</td>
<td>VWF</td>
<td>4</td>
<td>20.0 ± 2.3 23.8 ± 1.9</td>
<td>13.4 ± 1.8 13.5 ± 1.1</td>
<td>11.3 ± 1.5 13.5 ± 1.1</td>
<td>9.5 ± 1.1 12.8 ± 0.8</td>
<td>*11.8 ± 3.6 17.4 ± 1.5</td>
<td>*11.4 ± 3.1 18.5 ± 1.5</td>
</tr>
<tr>
<td>Skin temperature (°C)</td>
<td>thin clothes</td>
<td>VWF</td>
<td>4</td>
<td>20.0 ± 2.3 23.8 ± 1.9</td>
<td>13.4 ± 1.8 13.5 ± 1.1</td>
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</tr>
<tr>
<td></td>
<td>control</td>
<td>VWF</td>
<td>4</td>
<td>20.0 ± 2.3 23.8 ± 1.9</td>
<td>13.4 ± 1.8 13.5 ± 1.1</td>
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<td>*11.8 ± 3.6 17.4 ± 1.5</td>
<td>*11.4 ± 3.1 18.5 ± 1.5</td>
</tr>
<tr>
<td>Nail press test (seconds)</td>
<td>thin clothes</td>
<td>VWF</td>
<td>4</td>
<td>5.5 ± 1.1 2.3 ± 0.3</td>
<td>15.5 ± 4.6 5.6 ± 1.2</td>
<td>18.5 ± 2.6 7.6 ± 1.1</td>
<td>20.0 ± 0.0 8.1 ± 1.4</td>
<td>*15.5 ± 4.6 6.6 ± 1.3</td>
<td>**14.8 ± 5.3 4.4 ± 1.6</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>VWF</td>
<td>4</td>
<td>5.5 ± 1.1 2.3 ± 0.3</td>
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</tr>
</tbody>
</table>

* **: p < 0.05, p < 0.01 between VWF and control group
† † †: p < 0.05, p < 0.01 between thin and thick clothes

Fig. 3. Change of finger skin temperature and nail press test, when nail-press standing outdoors for 40 minutes at 5°C.
The results in Table 1 to 3 and Fig. 1 to 3 suggest that 1) hypersensitivity to cold in VWF workers has a close relation to VWF, 2) small arteries in VWF workers may have hypersensitivity to cold, 3) hypersensitivity to cold in blood vessel walls and central nervous system may concern VWF of finger, 4) chills preceded the occurrence of Raynaud’s phenomenon, and 5) the high nail-press test value reflects the severity of chill and the intense constriction of capillaries, which may indicate the onset of Raynaud’s phenomenon.

3. Hypothalamus and autonomic nervous function in VWF

Skin temperatures in fingers and toes of VWF workers are lower than in control workers. This means that VWF workers issue cold signals to the central nervous system, hypothalamus, much more often than controls.

Extensive research from the 1960s to the 1980s clarified the existence of warm and cold thermosensitive neurons in the hypothalamus. Experimental studies on the response of rat thalamic neurons to skin cooling established the existence of neurons activated by cold in the thalamus. The appearance of a FOS protein-like immunoreactivity in the hypothalamus of developing rats in response to cold ambient temperature was reported. From such reports, it became clear that cold-signal receptors are located in the hypothalamus.

Lin reported that changes in environmental temperature affect both hypothalamic somatostatin (SS) levels and thermoregulatory responses in rats. Activation of cold receptors in the periphery, in addition to elevating hypothalamic SS-levels, led to increased metabolism and cutaneous vasoconstriction in cold weather.

Gotoh indicated a clear relationship in rats between hypothalamic NA activity and NA activity in IBAT (interscapular brown adipose tissue), which is strongly controlled by sympathetic nerves, following stress induced by a cold swim. As a result, Gotoh suggested a possible role for hypothalamic NA activity neurons in modulating the sympathetic outflow, although the exact mechanisms and pathways between these neurons and outflows remain unclear.

Harada and Nakamoto reported higher serum levels of NA during whole-body cooling in VWF workers than in controls. They suggested hyperactivity in the autonomic nervous system of VWF workers exposed to cold.

These results suggest that the role of the hypothalamus in relaying cold signals from the hand and arm in VWF may have a connection with hypersensitivity to cold and to autonomic sympathetic hyperactivity in vibration syndrome.

4. Pathophysiology and pathology in progress and recovery of vibration syndrome

Attacks of VWF involve hypersensitivity of the vascular walls of the finger to cold and autonomic hyperactivity in cold. In the progress of vibration syndrome, peripheral hypersensitivity and central hyperactivity are reinforced by vibration, noise and exposure to cold. Low skin temperature causes many cold signals to be relayed to the central nervous system where hyperactivity to cold may result. Subjects with severe VWF complained intensely of many subjective symptoms related to the central nervous system, i.e., heavy headedness, irritability etc. If temperature at home and in the workplace were higher, warm signals might increase and this reinforcing activity might diminish.

In the recovery, following cessation of vibration exposure, VWF attacks gradually decrease in frequency and severity. If therapeutic heat is applied and the subject is kept warm at home and at work, recovery is hastened. It is a basic principle of therapy to allay bodily damage from cold stimuli by substituting warm stimuli. This is necessary in order to reverse thermosensitive dysfunction in the peripheral and central nervous systems. Thus, hypersensitivity as a subjective symptom decreases gradually.
However, in severe cases, a drop in skin temperature continues for a long time. This is related to decreased blood flow following pathological changes in the blood vessels, i.e., increase of adventitia combined with increase of connective tissue in the periarterial wall, and thickening of media by muscle hypertrophy. In some cases, the threshold of pain sensation may remain at high levels for a long period even while numbness and pain persist in the hand and arm. This is due to histological changes in nerve fiber endings, i.e., a decrease in the number of neurons, a loss of myelin, the disappearance of axons, increases in Schwann’s cells, collagen and perineural tissue, particularly in perineural connective tissue which forms an onion layer. Such changes are irreversible and cause abnormalities in sensation and an alteration in nerve-conduction velocity. If numbness and pain in hand and arm remain severe, they are accompanied with other symptoms such as feelings of heavy headedness, irritability and the like.

A decrease in muscle power, especially in grasping power, is observed only in severe cases. This is concomitant with abnormal EMG findings. Neurologically, motor-nerve fiber is more robust than that of sensory nerves, and is injured only in severe cases. But reduced dexterity of finger movement and of pinching power between fingers began earlier than a reduction in grasping power. A comprehensive study of this subject remains to be undertaken.

In summary, hypersensitivity to cold and autonomic hyperactivity in cold increase in the progressive stage and decrease in recovery, and these changes are accompanied by the change of frequency and severity of VWF attack, while severe neurological changes in the tissues of blood vessel and nerve fiber are irreversible and accompanied by numbness and pain.

5. Effect of aging

In the administration of long-term health care, new problems are uncovered in the medical examinations. One such problem is the effect of aging. Examination results are influenced by the age of workers. We often misjudged test results in those over 50 years because the same standard used for younger workers was used. In examining 30-, 40- and 50-year-old workers, we tested for changes in specificity and sensitivity. Significance of these two factors diminished as age advanced, especially in the 50-year-olds. This suggests that the influence of aging is very important in the evaluation of examination results.

6. Individual differences

Individual differences are observed in acute and chronic vibration exposure. In acute vibration exposure of the right hand, skin temperature of fingers in both exposure and the opposite non-exposed side falls in some but not in others, and even rises in a few cases. In one experiment, perspiration response in palm to vibration exposure appeared to be strong in one third of subjects, medium in another third, and weak in the remaining third.

In chronic vibration exposure, some vibration syndrome patients show severe VWF and numbness while no symptoms at all appear in others with the same age and years of chain saw operation. These results suggest individual differences.

Who will be affected by long-term vibration exposure? Why do workers with the same age and years of chain saw operation show such a disparity in test results? Such questions pose an interesting challenge to future researchers.

7. Soviet and Japanese theories of vibration syndrome

The final problem to be addressed here involves Japanese and Soviet conceptions of vibration syndrome. Some researchers in Europe suggest that Soviet diencephalic theory has influenced some Japanese research, since we reported subjective symptoms concerning the central nervous system.
In Japan, subjective symptoms including headaches or heavy headedness, sleep disturbance and irritability etc. were reported from the late 1960s onward.12,13,31–34) These subjective symptoms were diagnosed on the basis of inquiries by physicians, not on the basis of Soviet theory. Such symptoms were recently analysed again in new surveys. The prevalence of these symptoms is related to years of vibration exposure.33,34) A correlation existed between the severity of VWF and numbness and these subjective symptoms among groups of the same age and years of operation.12)

How did such a misunderstanding arise in Europe? It may have to do with the fact that Soviet theory was introduced to Japan via Andreeva-Galanina’s classification of stage which was briefly in vogue in Japan during the mid-70s. Her book, “Vibration and Its Significance in Labor Hygiene” (1956), was first introduced by Matsufuji in a review article in 1964. Introducing her data on the classification of vibration disease, Matsufuji remarked: “Andreeva-Galanina’s conception of vibration disease is very different from ours, but we do not have sufficient data in Japan to adequately critique her work. Further research will be needed.”

A new edition of the same investigator’s very interesting book “Vibration Disease” was published in 1965 in old Leningrad. During World War II, Soviet industrial production facilities were much destroyed. Postwar reconstruction began under appalling conditions which gave rise to many severe cases of vibration disease. This experience prompted extensive research in the epidemiological, clinical and pathophysiological fields. She described many cases of vibration disease based on a conception of the disorder as consisting of a peripheral and central nervous system dysfunction. Giving detailed descriptions of clinical cases at every stage as well as of diagnostic methods, she divided vibration disease into three types: local, with and without whole-body vibration, and whole-body vibration. But her book had few readers in Japan and was not translated into Japanese. In 1972, “Preventive Measurements and Research in the USSR” was published in Japanese by Watanabe and Yamada. In it Watanabe introduced Andreeva-Galanina’s classification as well Matsufuji.

A Special Medical Examination Committee for the Diagnosis of Vibration Syndrome, which was organized by the Ministry of Labor in 1971, proposed prevention guidelines in 1973. These guidelines classified the stages of vibration syndrome with reference to Andreeva-Galanina’s theory. But doubts were raised as to whether or not her third and fourth stages could be verified in actual cases. For this reason, her classification of stages was utilized by relatively few researchers.

Another book, “Vibration Disease in Industry,” was published in 1972, by a Moscow research group whose approach differed from that of the Leningrad group. They described local vibration disease in combination with whole-body vibration disease, involving diencephalic theory. This book was translated into Japanese in 1988 by Watanabe and Ito. It reported on research in a wide range of medical fields, presenting data that stimulated further Japanese research. However, the diencephalic disturbance theory was not accepted by Watanabe or other Japanese researchers.

In the 1980s, Japanese researchers investigated the clinical pictures, subjective symptoms and pathophysiology concerning autonomic nervous system on the basis of their data.

**COMPREHENSIVE PREVENTION SYSTEM**

1. Long history of comprehensive prevention system

Japan’s contribution to the field of prevention was the establishment of a comprehensive prevention system.
Research into and practical steps toward prevention made significant progress in the national forest industry. Our national forests grow on steep mountain slopes at about 1,000 m above sea level. It is cold in late autumn and winter, and very rainy in early summer, which easily causes chill. The average diameter of coniferous trees is about 0.5 to 1.5 m. Felling and cutting of trees is hard labor. Vibration acceleration of chain saws is over 100 m/s² with a sound level of about 110–115 dB. Chain saws of 80 to 100 cc displacement weigh about 12 to 15 kg. These facts posed special dangers to Japanese forest workers who were physically small and often poorly nourished. Furthermore, the forest industry was organized on a piece-work basis, and workers were only seasonally employed and accustomed to long working days. These conditions only served to contribute to a deterioration in health.

Based on research in the national forests of central Japan, we recommended a comprehensive prevention system in 1965. Our recommendations included restrictions on operating time, improvements in chain saw design, better health care and education, and measures against the cold. The Forestry Agency of Japan did not accept our recommendations, preferring instead the theory of Drs. Negishi and Katsunuma of Tokyo University who reported that complaints of chain saw operators were largely psychological in origin. As a consequence, many cases of vibration syndrome among forest workers went untreated.

Five years later, the Forestry Agency changed its policy under pressure from researcher recommendations, the forest worker's union and social criticism. At first, they accepted only restrictions on chain saw operating time. This delay in embracing comprehensive measures influenced other industries, especially the private forest industry where, as a result, many severe cases of vibration syndrome developed. After prolonged effects, a comprehensive prevention system for the national forests was adopted in 1977. The Ministry of Labor mandated this system in other industries in the 1980s, and the incidence of vibration syndrome was gradually reduced.

Over the years, discussion has focused on the following problems:
1. Why are there so many cases of vibration syndrome in Japan? Why is the disorder so severe in many cases?
2. What prevention methods are truly effective?
3. What data should be used as a basis for restricting operating time? Are such restrictions feasible under a piece-work system? How do restrictions on operating time affect the work process? What can we expect from time-restriction arrangements?
4. Which mechanical components of vibrating tools can be improved? How should the evaluation of improved tools be conducted before newly designed tools are used on the job?
5. What level of acceleration should we use as the benchmark for improvement? What data should serve as a basis for that?
6. How were the ISO prevention guidelines evaluated in Japan?
7. Can the remote-control chain saw really be considered a technical advance in view of the fact that it reduces productivity?
8. What constitutes a suitable health care system?
9. Why do we need measures to protect against the cold?
10. What kind of education is needed for prevention? Is some intervention in workers' lives necessary for prevention?
11. What system is needed to rehabilitate patients with vibration syndrome?
12. What lessons can we learn from the long history of preventive measures?

2. Risk factors causing vibration syndrome

The fundamental approach to prevention of occupational disease has been to investigate risk
factors and, having done so, either to eliminate them or thwart their further development. Risk factors in vibration syndrome do not exist independently of one another. If the disorder is identified too late, patients may suffer severe and prolonged consequences. For this reason, a comprehensive prevention system should be established early. The risks addressed by such a system arise from technological, medical, working, meteorological and educational factors.

1. Risk from technological factors
   Technological risk in the forest industry appeared following mechanization in the cutting and transportation of trees. While mechanization served to reduced accidents and the expenditure of human energy, it also caused back pain due to poor working posture during operation, seat vibration and hearing loss. The introduction of chain saws is the most recent step in the mechanization of the forest industry sector. Development of chain saw improvements such as an anti-vibration apparatus in the late 1960s and better-balanced engines in the late 1970s diminished the risks of vibration syndrome and hearing loss. Remote-control and carrier-mounted chain saws introduced in the early 1970s distanced workers from the vibration source. As for other technological factors, reductions in the weight of chain saws are discussed below. In short, we may say that technological risks diminished gradually in the 1970s and rapidly in the 1980s.

2. Risks from working conditions
   Risks in the workplace increased with the introduction of piece-work in the 1960s by encouraging longer working days. These risks were dramatically reduced following the adoption of chain saw operating time restrictions in the 1970s. The risks further diminished with the substitution of piece-work by a fixed-wage system in the 1980s. In terms of human engineering, the excessive weight of chain saws and the resultant ill effects on posture while felling trees on steep slopes was a high-risk factor in the 1960s and 70s. Some of these risks were alleviated by the weight reduction in 60 cc-displacement engines and the introduction of the bantam chain saw with a 35 to 40 cc displacement and a weight of only 4 to 5 kg. Shifting operations to plantation-type forests promoted use of bantam chain saws. However, risky working conditions on steep slopes remain a problem.

3. Risks from inadequate health care
   Health care concerns escalated with the introduction of the chain saw in the 1960s without any provision for a system of medical checkups. Health care systems were established locally by various researchers in many forest areas, and these gradually spread to all forests. The Ministry of Labor recommended a system of medical checkups in 1971, and a comprehensive health care system in 1976. This gradually reduced the risk, and much data on the health effects of vibration exposure could be gathered.

4. Weather-related risks
   Continuous vibration exposure in cold weather strongly promotes peripheral vasoconstriction, and reinforces hypersensitivity to cold in the central nervous system. In the 1960s, this risk was aggravated by workers operating chain saws with no protection from the cold and by the practice of commuting to and from work by motorcycle. A significant risk reduction resulted from the introduction of commuting buses, warm lodges and chain saws with heated handles.

5. Risks from lack of education
   Technological and hygienic education in chain saw operation is very important for avoiding muscle strain and reducing the length of exposure to vibration. Lack of such education in the
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1960s increased the risk, although the situation slowly improved in the 1970s and 80s. Training in Scandinavian lumbering techniques as well as a training system developed in the Bohemian Rehabilitation Center in Czechoslovakia served to improve our education system.

Summarizing the risks traceable to faulty education, the problem was at its most serious from the late 1960s to the early 1970s, but things improved significantly in the following decade.

6. Five stages of progress in comprehensive prevention system

Our comprehensive prevention system achieved success among national forestry workers only after a 20-year effort. It became a model for prevention in other industries. The number of new cases of occupational vibration syndrome in Japan steadily decreased, and there are fewer new cases of a severe nature now. I divided this long process into five stages on which I have reported elsewhere.35,36,37)

CONCLUSION

The emergence of vibration syndrome among Japanese national forestry workers in the 1960s prompted clinical, pathophysiological and preventive research in many industries in Japan. In clinical research, the clinical picture and therapy were studied from the late 1970s into the 1980s. In the field of pathophysiology, research into the autonomic nervous system progressed in the 1980s and 90s. Risks associated with technological change, working conditions, health care, meteorological conditions and lack of education became fewer in the 1970s and 80s. In the national forestry sector, a comprehensive system was established in the 1980s. This system involved 1) work restrictions, 2) a responsive health care system, 3) technical improvements in vibration tools, 4) warming measurements against the cold, and 5) educational programs. This comprehensive prevention system was legally introduced into other industries, resulting in a rapid decrease in the incidence of vibration syndrome in Japan.

REFERENCES


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