IV. BIOLOGIC EFFECTS

A. HAND-ARM VIBRATION SYNDROME (HAVS)

HAVS comprises a composite of pathophysiologic signs and symptoms that develop over time in workers who use hand-held vibrating tools. Many of the signs and symptoms of HAVS are also seen in other clinical entities such as primary Raynaud's disease, occlusive vascular disease, traumatic injury of hands, proximal vasculature compression, peripheral neuropathies, carpal tunnel syndrome, etc. [Taylor 1989; Taylor and Pelmear 1975; Taylor and Brammer 1982; Pyykko and Starck 1986; Wasserman 1987]. The factors listed in Table IV-1 [NIOSH 1983a] must be considered in differential diagnoses for HAVS.

B. PATHOPHYSIOLOGY OF HAVS

The development of HAVS is a gradual progressive process that may involve years of exposure to hand-arm vibration [NIOSH 1983a]. Some of the etiologic aspects of HAVS have been reviewed by Pyykko and Starck [1986], and Taylor [1988]. A requisite for diagnosis of occupational HAVS is a history of occupational use of vibrating tools such as drills, chipping hammers, grinders, concrete vibrators and levelers, polishers, swagging tools, shoe pound-up tools, caulking tools, fettling tools, clinching and flanging tools, burring tools, rock drills, chain saws, jackhammers, riveting hammers, bucking bars, and jackleg hammers.

In primary Raynaud's disease, the signs, symptoms, and involvements are usually symmetrical (same areas of both hands involved), whereas in secondary Raynaud's disorders, including HAVS, the involvement is usually asymmetrical [NIOSH 1983a]. Presently, however, no single test is available that will reliably distinguish HAVS from other secondary Raynaud's disorders [NIOSH 1983a; NIOSH 1984; Gemne 1982; Brammer et al. 1986; Pyykko 1986].

The classification of the clinical stages of signs and symptoms of HAVS most widely used in the past is the one suggested by Taylor and Pelmear [1975]. This classification assumes two major pathophysiologic consequences of using vibrating tools: peripheral neural and peripheral vascular involvements. This classification does not specifically distinguish the peripheral neural and peripheral vascular progressive changes as separate entities (Table IV-2).

A revision of the Taylor-Pelmear classification of the stages of HAVS has been proposed by Taylor [Brammer et al. 1986]. This revised classification takes into account the concept

Table IV-1.—Relationships considered in differential diagnoses for HAVS*

Medical condition	Signs or symptoms
Primary Raynaud's phenomenon	Constitutional white finger
Trauma direct to extremities	Injuries or fractures; vibration of occupational origin (HAVS); frostbite and immersion syndrome
Nerve compression	Carpal tunnel syndrome
Trauma to proximal vessels by compression	Thoracic outlet syndrome (cervical rib, scalenus anterior muscle), costoclavicular and hyperabduction syndromes
Occlusive vascular disease	Thromboangiitis obliterans, arteriosclerosis, embolism, thrombosis, Burger's disease
Dysglobulinemia	Cold hemagglutination syndrome, cryoglobulinemia, macroglobulinemia
Intoxication	Acroosteolysis; reactions to ergot, nicotine, and vinyl chloride
Neurogenic dysfunction	Poliomyelitis, syringomyelia, hemiplegia, polyneuropathy
Secondary connective tissue disease	Scleroderma, systemic lupus erythematosus, rheumatoid arthritis, dermatomyositis, polyarteritis nodosa, mixed connective tissue disease

*Adapted from NIOSH [1983a].

Table IV-2.—Taylor-Pelmear classification of vibration-induced white finger by stages*

Stage	Signs and symptoms	Interference with activities
0	None	None
0T	Intermittent tingling	None
0N	Intermittent numbness	None
0TN	Tingling and numbness	None
1	Blanching of one or more fingertips with or without tingling and numbness	None
2	Blanching of one or more fingers with numbness, usually during winter only	Slight interference with home and social activities; no interference with work
3	Extensive blanching with frequent episodes during both summer and winter	Definite interference with work, home, and social activities; restricted hobbies
4	Extensive blanching of most fingers; frequent episodes during summer and winter; finger ulceration	Occupation change required to avoid further vibration exposure

*Adapted from Taylor and Pelmear [1975].

that the peripheral neural and the peripheral vascular involvements in HAVS may be distinct entities and that the disabilities related to each may progress independently of the other. The revision also recognizes that the tactile sensory deficits can and should be measured and considered independently of the vasospastic episodes (Table IV-3).

At the 1986 Stockholm Workshop, a new staging classification of vibration-induced signs and symptoms was introduced [Brammer et al. 1987; Gemne et al. 1987; Taylor 1987; Taylor 1989]. The Stockholm Workshop staging classification of the peripheral-neural and the peripheral-vascular pathophysiologic effects of hand-arm vibration exposure are considered

Table IV-3.—Brammer et al. revisions of the Taylor-Pelmear clinical stages of vibration-induced white finger*

Stage	Signs and symptoms	Interference with activities
0N†, 0V†	No signs or symptoms	None
1N	Intermittent tingling and/or numbness	None
1V	Episodic blanching of one or more finger tips	None
2N	Intermittent numbness; reduced tactile perception	Possible interference with activities involving fine manipulative tasks
2V	Episodic blanching of one or more fingers, usually during winter only	Some interference with work and/or social activities
3N	Degraded tactile resolution; intermittent numbness	Interference with activities involving fine tasks at work and at home
3V	Extensive finger blanching, frequent episodes during both summer and winter; tissue changes (finger ulceration)	Restricted hobbies and social activities to avoid vasospasms; interference with work

*Adapted from Brammer et al. [1986].
†N = neural; V = vascular.

separately, thus reflecting the concept that they are two clinical entities. In addition, a system is provided for a semiquantitative expression of the extent of the involvement of each finger on each hand. This system provides a mechanism for a quantitative clinical estimate and description of the involvement. The stagings as presented at the Stockholm Workshop are shown in Tables IV-4 and IV-5. It was suggested at the Workshop that this staging classification be used in all future hand-arm vibration studies. A standard staging classification will enhance the comparability of the data.

The signs and symptoms and their time sequence in appearance indicate that vibration affects several components of hand and arm function [Taylor and Brammer 1982; Farkkila et al. 1982; Pyykko et al. 1982a, 1982b; Futatsuka and Ueno 1985; Farkkila 1986]. HAVS may involve, separately or in combination, the (1) peripheral neural system, (2) peripheral vascular system, (3) muscles of the hands and arms, (4) bones and joints of the hands and arms, and (5) central nervous system.

The Russian and Japanese [Griffin 1980; Habu 1984] classifications of the relative degree of the disorder in patients with HAVS include subjective symptoms, objective responses to tests, and clinical evaluations. The degree of impairment ranges from Stage 1 with minimal impairment to Stage 4 with extensive impairment. The systems impaired are considered separately as (1) vascular, (2) sensory, (3) musculoskeletal, and (4) brain stem and neuropsychiatric. The degree of impairment ranges from no change (–), to minimal change (+), to extensive involvement (++++). The possible involvement of the central autonomic nervous system in HAVS was the subject for an international symposium in 1983 [Gemne and Taylor 1983].

A classification for staging the severity of vibration-induced HAVS, including some functional changes, has been proposed in Japan [Okada 1983; Okada and Suzuki 1982]. The major features of the classification are summarized in Table IV-6.

The biological effects of vibration exposure may be influenced by many nonvibration factors [ACTU-VTHC 1982; see also Chapter III], including the following:

- Exposure pattern
- Length and frequency of work and rest periods
- Magnitude and direction of forces applied to the workpiece by the operator
- Body posture and orientation of the wrists, elbows, and shoulders
- Area of hand exposed to vibration
- Climatic conditions

Table IV-4.—The Stockholm Workshop classification scale for cold-induced peripheral vascular symptoms in the hand-arm vibration syndrome*,†

Stage	Description
0	No attacks
1	Occasional attacks that affect only the tips of one or more fingers
2	Occasional attacks that affect the distal and middle (rarely also proximal) phalanges of one or more fingers
3	Frequent attacks affecting all phalanges of most fingers
4	As in stage 3, with trophic skin changes in the finger tips

*Adapted from Gemne et al. [1987].
†The Stage is determined separately for each hand.

Table IV-5.—The Stockholm Workshop classification scale for sensorineural stages of the hand-arm vibration syndrome*,†

Stage†	Symptoms
0SN	Exposed to vibration but no symptoms
1SN	Intermittent numbness, with or without tingling
2SN	Intermittent or persistent numbness, reduced sensory perception
3SN	Intermittent or persistent numbness, reduced tactile discrimination and/or manipulative dexterity

*Adapted from Brammer et al. [1987].
†The sensorineural stage is determined separately for each hand.

Table IV-6.—Japanese staging classification for hand-arm vibration syndrome*

Classification	Signs and symptoms
Stage 1	Episodic blanching of distal phalanges Borderline decrease in motor and sensory conduction velocities Minimal changes in hand radiographs Periodic numbness and pain in fingers Paresthesia may be present
Stage 2	Extended episodic blanching Further decrease in motor and sensory conduction velocities Slight EMG abnormalities Moderate changes in hand and arm radiographs Pain and numbness lasting longer at rest and at night More pronounced hyperesthesia
Stage 3	Blanching extended to all fingers but not the thumbs Greater decreases in motor and sensory conduction velocities Pronounced EMG changes Pronounced changes in hand and arm radiographs Some restriction of hand and arm movement Atrophy of hand/arm muscles Exaggerated subjective symptoms
Stage 4	Frequent blanching of all fingers but not thumbs Pronounced decrease in motor and sensory nerve conduction velocities Very pronounced EMG changes Pronounced changes in radiograph Increased motility restriction and muscle atrophy Further exaggerated subjective symptoms

*Adapted from Okada [1983].

- Worker's skill and work practices
- Hand covering
- Maintenance of equipment
- Noise (possible synergistic effect)
- Use of tobacco, some drugs, and some chemicals

Taylor and Brammer [1982] and Brammer [1984] have categorized the factors as physical, biodynamic, and individual (Table IV-7). These factors may vary extensively in amount and combinations from one exposure situation to another and from day to day when using the same tool [Brammer 1984].

Knowledge of HAVS is based mainly on retrospective epidemiologic studies or clinical examinations and comparisons between workers who use and do not use vibrating tools, and who do or do not have symptoms of HAVS. Lack of objective data from controlled laboratory investigations limits the accuracy of any dose-response or risk factor predictions.

Taylor [1988, 1989], in reviewing the biological effects of hand-arm vibration, pointed out some of the information gaps in the understanding of the mechanisms involved in the neurological, vascular, and musculoskeletal damage.

1. Peripheral Neural Effects

The early symptoms frequently experienced by workers exposed to hand-arm vibration include intermittent attacks of tingling and/or numbness of the fingers with or without pain [Brammer 1984; Taylor and Brammer 1982; Brammer and Taylor 1982; Taylor 1982a; Taylor 1982b; Vines 1984; Pyykko 1986]. On continued exposure, the attacks may become more frequent and the symptoms more severe with decreased tactile sensitivity, decreased temperature sensitivity, and decreased manual dexterity and grip strength [Taylor and Brammer 1982; Brammer et al. 1986; NIOSH 1983a; Farkkila et al. 1982; Brammer 1984].

The mechanisms involved in the observed peripheral neural changes have not been fully described. In workers who have been exposed to hand-arm vibration on the job and who have developed intermittent peripheral neural symptoms (numbness, tingling, pain, loss of sensitivity), the increased vibration perception threshold may reflect the functional disturbance of the peripheral nerves, of the sensory nerve endings, or of the mechanoreceptors, including the Pacinian corpuscles [Lundstrom 1986]. Harada and Matsumota [1982] suggested that the peripheral neural effects are a pathophysiologic entity separate from and independent of the circulatory disturbances. This suggestion was based on neural and

Table IV-7.—Categories of factors that may modify the biologic effects of hand-arm vibration exposure*

Factor categories	Modifying factors
Physical	Dominant vibration amplitudes entering the hand Dominant vibration axis relative to the hand Years of employment involving vibration exposure Total duration of exposure each workday Pattern of work/rest exposure each workday Nonoccupational exposure to vibration
Biodynamic	Hand grip forces (compressive and push or pull forces) Surface area, location, and mass of parts of the hand in contact with the source of vibration Posture (position of the hand and arm relative to the body) Other factors influencing the coupling of vibration source to the hand (e.g., texture of handle—soft, complaint vs. rigid material)
Individual	Factors influencing source, intensity, and exposure duration (e.g., state of tool maintenance, operator control of tool, work rate, skill, and productivity) Biological susceptibility to vibration Vasoconstrictive agents affecting the peripheral circulation (e.g., tobacco, drugs, etc.) Predisposing disease or prior injury to the fingers or hands (e.g., trauma, lacerations, diabetes, connective tissue disorders)

*Adapted from Taylor and Brammer [1982] and Brammer [1984].

vascular tests of workers not exposed to vibration, workers who used vibrating tools but had no symptoms, and workers who used vibrating tools and had symptoms of HAVS. Workers with neurophysiologic symptoms compatible with hand-arm vibration syndrome have a decreased sensory nerve conduction velocity from the fingers to the wrist [Alaranta and Seppalainen 1977; Sakurai and Matoba 1986]. These findings are consistent with the concept of a direct pathophysiologic effect of vibration on the peripheral nerves and nerve endings.

On the basis of a study of 245 vibration-exposed subjects with a history of nerve symptoms or evidence of neural injury, Lukas [1982] and Lukas and Kuzel [1971] concluded that the peripheral neural lesions may result from causes other than the direct effect of vibration. Nerve conduction velocity measurements of the fast motor and sensory fibers of the median and ulnar nerves frequently indicated that only one of the nerves tested was involved. In individuals using hand-held vibrating tools, both nerves would be expected to be affected. The authors also observed that conduction velocities in the distal portion of the sensory and motor fibers of the median and ulnar nerves were significantly reduced in the patients with symptoms of vasoneurosis as compared with the patients without vasospastic symptoms. The author concluded that the peripheral neural damage was secondary to the complex damage to the vascular, joint, and muscular systems of the arms and hands (multifactorial). Juntunen et al. [1983] found that nerve conduction velocity was slower and motor nerve latency longer in the patients with vibration syndrome who had neurologic signs of polyneuropathy. The authors stated that these findings provided evidence of a wider neural involvement than just the peripheral nerves.

On the basis of an analysis of data from forest workers, Farkkila et al. [1988] suggested that a large part of the previously diagnosed vibration neuropathies belong to the category of carpal tunnel syndrome (CTS), which is the most common entrapment neuropathy among forest workers.

Brammer et al. [1986] evaluated the degradation of vibrotactile and spatial-tactile perception in workers who used hand-held vibrating tools. The vibrotactile perception threshold values in workers with HAVS Stage 1, 2, or 3 of the Taylor and Pelmear classification exceeded the values of workers with no vibration exposure by more than 2 standard deviations. On the spatial-tactile resolution test [Carlson et al. 1984], the workers with vibration syndrome had values exceeding the controls by 2 to 4 standard deviations. Brammer et al. [1986] concluded that the neurological signs and symptoms in workers with chronic hand-arm vibration exposure involve both the peripheral nerve fibers and the mechanoreceptors.

Several types of neurophysiologic structures in the skin of the fingers are involved in the sense of touch [Vallbo and Johansson 1984]. The different structures function (fire) in response to different mechanical stimuli. The Pacinian corpuscles, the quick-adapting receptors, and the two types of slow-adapting receptors are the most important mechanoreceptors in the skin that are involved in the sense of touch [Lundstrom 1986]. For the sense of touch as measured by the sharp edges of the aesthesiometer (tactospatial), the slow-acting Type I receptors were more sensitive than the Pacinian corpuscles, the quick-acting receptors, or the slow-acting Type II receptors. Brammer et al. [1986] reported a decrease in the vibrotactile sensitivity that reflected the threshold of the Pacinian corpuscles.

In a biopsy study of 60 fingers with HAVS, Takeuchi et al. [1986] reported a decreased number of axon cylinders and a destruction of the myelin sheath in 90% of the cases. Such histopathologic deterioration could provide the basis for the sensory changes.

Researchers in this field agree that exposure to hand-arm vibration will eventually result in peripheral neural impairment with sensory loss, numbness, and decreased sensory and motor nerve conduction velocities but that the cause-effect mechanisms are not clear [Brammer 1986; Taylor and Brammer 1982; Brammer and Taylor 1982; Pyykko 1986; Ekenvall et al. 1986; Lundstrom 1986; Saraki et al. 1988; Radwin et al. 1987]. Taylor [1982a] summarized the state of knowledge as follows: "It is not known whether vibration directly injures the peripheral nerves thereby causing numbness and subsequently sensory loss, or whether the para-anesthesia of the hands is secondary to the vascular constriction of the blood vessels causing ischemia . . . in the nerve-end organs."

Lundborg et al. [1987] investigated the value of the vibrotactile sense test in assessing vibration-related neuropathies. The vibration sensation threshold for individuals who had not used vibrating tools was constant at vibration frequencies from 8 to 250 Hz. At 500 Hz, the sensation threshold was higher. For workers who used vibrating tools and had symptoms ranging from intermittent numbness to constant numbness and pain, the vibration sensation threshold increased progressively with higher vibration frequencies. The abnormalities in the vibrogram (sensation threshold) correlated not only with numbness and pain, but also with the occurrence of white finger. The authors concluded that the numbness resulted from a change in the intraneural vascular function. The finger blanching and pain and the numbness all reflect a common peripheral vascular disorder.

2. Peripheral Vascular Effects

The earliest signs of peripheral vascular changes in HAVS are the episodic attacks of fingertip blanching. These initial attacks of ischemia usually occur during cold exposure [Taylor and Brammer 1982; Brammer 1984; Pyykko 1986; Wasserman 1987]. With the continued exposure to vibration, the frequency and severity of the episodes of white finger increase until the blanching extends to the base of the fingers and may occur even in warm weather. The time between the first use of vibrating tools and the first appearance of episodic finger blanching (vasospasms) is designated as the latent interval. The latent interval appears to vary with the vibration intensity, as well as with other factors such as the type, model, unique individual characteristics of the tool, material on which the tool is used, tool maintenance, operating speed, operating technique, user characteristics, hand-grip force, push force, hand and arm posture, work/rest regimen, air temperature and moisture, protective hand gear, and clothing [Taylor and Brammer 1982; Brammer 1984].

In one of the early reports, the peripheral vascular symptoms of HAVS were considered to be only a nuisance [Pecora et al. 1960]. However, for the individual worker, the effects may seriously interfere with work [Taylor et al. 1977; Pyykko 1986]. Laroche [1976] reported that 10 out of 13 patients with HAVS had to change jobs because of the disabling effects of the disorder.

Although the phenomenon of the vasospastic episodes (sequence of events) has been described and accepted, basic underlying pathophysiologic mechanisms are not understood.

Nonetheless several have been proposed. The present concepts are built on data derived mainly from epidemiologic and clinical studies.

The sequence of events in the progression of the episodes of blanching appear to involve (1) bouts of digital arterial spasms that become progressively more frequent and prolonged, (2) arterial hypertrophy with an increase in the medial muscular layer of the arterial wall, (3) perivascular fibrosis with increased collagen formation, and (4) increased vasoconstrictor sensitivity. The hypertrophy of the arterial smooth muscle may progress until the arterial lumen is partially or completely obliterated [Ashe et al. 1962, 1964; Pyykko et al. 1982a; Brammer and Taylor 1982; Brammer 1984; Wegelius 1972; Takeuchi et al. 1986]. Biopsies on 60 fingers from 30 patients with HAVS revealed increased thickening of the muscular layer that was severe in 82% and moderate in 15% when compared with 7 biopsies on 3 control subjects. Perivascular fibrosis was found frequently and was severe in 55% and moderate in 37%. Intimal thickening was minimal in 35% and moderate in 7% of the HAVS workers [Takeuchi et al. 1986]. Okada et al. [1987a] in animal experiments observed that vibration resulted in intimal changes in the small arterioles with minimal medial changes. Similar findings were reported by Inaba et al. [1988].

Several etiologic theories have been postulated to account for the observed pathophysiologic changes in the digital arteries, but no single theory can account for all the changes. The mechanisms that could help explain the events seen in HAVS include (1) direct effects of vibration on the digital arteries, (2) effects on the peripheral sensory aspects of the sympathetic nervous control of the peripheral circulation, (3) central neural control, (4) hypersensitivity to chemical mediators (prostaglandins, serotonin, and noradrenalin), and (5) some combination of the above [Brammer 1984; Taylor and Brammer 1982; Azuma and Ohhashi 1982; Gemne et al. 1986]. The use of tobacco has been reported to be an additional risk factor in the development of HAVS in workers who operate noise-producing, hand-held vibrating tools [Miyakita et al. 1987; Bovenzi 1986; Ekenvall and Lindblad 1989].

Ekenvall and Lindblad [1989] measured the levels of nicotine and cotinine in the blood of 111 tobacco users and nontobacco users who did or did not have HAVS as determined by the digital systolic blood pressure after finger cooling. Blood nicotine concentrations in ng/ml were 0.8 in nontobacco users, 14.5 in cigarette smokers, 10.8 in snuff users, and 16.3 in users of other tobacco products; the cotinine levels were 0.7, 196, 252, and 210 ng/ml, respectively. When the 111 workers were grouped according to the Taylor-Pelmear stages of HAVS, the blood levels of cotinine in ng/ml were 5.7 for stages 0T and 0V, 8.7 for Stages 1 and 2, and 11.8 for Stages 3 and 4; the cotinine levels were 124, 117, and 171, respectively. The digital systolic blood pressure (as a percent of the arm systolic pressure during finger cooling at 15°C) was significantly lower in the tobacco users: 52% for nonusers, 29% for smokers, and 45% for snuff users. On the basis of their study results and a survey of the relevant literature, the authors concluded that "habitual use of tobacco aggravates the symptoms of VWF disease and has a direct effect on the results of a cold provocation test in this disease" [Ekenvall and Lindblad 1989].

Obliterative histopathologic changes reported in biopsies and arteriograms of the peripheral arterioles in some workers with HAVS appear convincing. But whether these changes are relevant histopathologic factors concerned with episodic finger blanching has been questioned on the basis of physiologic blood flow studies [Ekenvall et al. 1987]. Using venous occlusion plethysmography before and after induced vasodilation, these authors observed that the maximum blood flow after vasodilation was similar in the control subjects who were not exposed to hand-arm vibration and in vibration-exposed workers with advanced symptoms of HAVS. They reasoned that if the vessel lumen were narrowed by occlusive muscle hypertrophy, then blood flow would not be normal when maximum vasodilation was induced. Arteriograms were not taken; consequently, evidence of whether these subjects actually had arterial occlusion was lacking.

Regardless of the mechanisms involved in episodic finger blanching, the primary response concerns the digital arteries. During ischemic attacks, arterial blood flow to the affected segments of the fingers is reduced or completely shut off by contraction of the smooth muscles in the medial arterial wall. The episodic arterial muscle contractions could reflect an increased sympathetic nervous activity or an increased sensitivity of the arterial musculature (vasomotor tone) to local factors (chemical mediators including prostaglandins, serotonin, and noradrenalin) [Brammer 1984; Azuma and Ohhashi 1982; Bovenzi 1986]. Repeated contractions of the arterial muscles could lead to muscle hypertrophy. Gradual occlusion of the arterial lumen could result from intimal thickening. Stimulation of the vascular smooth muscle by adrenergic nerves constricts the digital artery lumen. Vasodilation occurs passively in the absence of vasoconstrictive action [Taylor and Brammer 1982].

The episodic ischemic attacks could be inappropriate pathophysiologic vascular responses to the vibration forces transmitted to the hands of the workers using vibrating tools. The medial muscle hypertrophy could be a histopathologic response to the sustained contraction of the vascular muscles. Any intimal changes could be a direct response to the vibration or a response to some changes in the blood chemistry [Taylor and Brammer 1982; Brammer 1984]. If the sympathetic nerve supply to the digital arteries is cut at the stellate ganglion, episodic blanching of the fingers induced by cold will return after an initial dilation of the digital arteries, which could last 3 to 4 months. This would imply that local chemical mediators may be at least partially involved.

A series of studies on workers with or without exposure to vibration and vibration-exposed workers with or without HAVS indicated that the finger vasospasm in the vibration-exposed group is the result of chronic stimulation of the mechanoreceptors in the fingers by the vibration. The receptors then serve as the sensory receptor link to the sympathetic nervous system with the effectors being the smooth muscle of the medial layer of the finger arterioles [Hyvarinen et al. 1973; Pyykko 1974b; Farkkila et al. 1985; Farkkila and Pyykko 1979; Pyykko et al. 1982a; Koradecka 1977].

Although the findings in these studies support the concept that the vibration sensory receptors contribute to HAVS by initiating excessive afferent impulses in the sympathetic

reflex involving the peripheral vascular musculature, the exact etiologic (pathophysiologic) mechanisms are not fully known [Pyykko et al. 1982a]. Generally, in experimental studies when the hand is stimulated by vibration, a vasoconstriction occurs in the fingers. In workers who have not used vibrating tools, the vasoconstriction response to vibration stimuli was mild (about 10% have a strong response). Among workers who have used vibrating tools occupationally, about 40% exhibited a strong vasoconstrictive response, and 40% had a mild response similar to that of the controls [Pyykko et al. 1982a]. This suggests that chronic vibration stimulation increases the sensitivity of the mechanoreceptors to the vibration stimuli and increases the level of afferent impulses to the central nervous system with resulting increased reflex-sympathetic vasoconstriction. The prolonged contraction of the vascular muscles could result in the hypertrophy of the medial musculature and increased narrowing of the vessel lumen [Ashe et al. 1962; Ashe and Williams 1964; Takeuchi et al. 1986].

3. Skeletal Muscle Force and Muscle Fatigue

The subjective complaints of deterioration of hand grip force and muscle strength in workers using vibrating tools have been mentioned in many of the studies of HAVS. This aspect of HAVS has received specific emphasis in studies in Japan [Miyashita et al. 1983] and in Finland [Farkkila et al. 1982; Farkkila et al. 1980; Farkkila et al. 1979; Farkkila 1978]. Among Japanese chain saw operators with more than 2,000 hr of chain saw use, about 28% reported symptoms of decreased grip force and muscle strength. These symptoms were reported by 20% of Finnish chain saw operators with more than 5,000 hr of chain saw use.

The Finnish group measured maximum grip strength and rate of development of muscle fatigue with or without acute vibration exposure under controlled laboratory conditions. Lumberjacks with or without symptoms of diminished grip force and with or without symptoms of HAVS were compared with a control group who had no occupational vibration exposure. In the group of lumberjacks with HAVS, some experienced only episodes of white finger without ever experiencing decreased grip force; some reported finger numbness without decreased grip force; and some experienced decreased grip force and white finger and/or finger numbness.

The data indicated the following relationships:

- Grip force was reduced in lumberjacks throughout the entire muscle fatigue curve when the hand was exposed to vibration.
- Grip force was reduced more in workers who reported numbness, reduced muscle strength, or muscle pains.
- Acute vibration exposures reduced grip force more in those with symptoms than in those without symptoms of white finger.

- Reduced muscle strength was present in lumberjacks with 5,000 or more hours of total chain saw operating time.
- Muscle force was reduced and the prevalence of symptoms increased when total exposure times exceeded 5,000 hr.
- Lumberjacks with white finger used a stronger grip force when using the chain saws than did those who did not have white finger.
- A dose-response relationship seemed to exist between diminished muscle force and vibration exposure.
- Grip strength decreased as a function of age in vibration-exposed and unexposed groups.
- Muscle strength fatigue curve for the chain saw operators was similar to that of unexposed workers.
- Muscle strength for controls and for chain saw operators was the same when the hand was not exposed to vibration.
- Muscle strength of the controls was similar to that of the lumberjacks who had no symptoms of white finger.

The pathophysiologic mechanisms involved in the reduced muscle strength aspect of HAVS are not clear. Some of the possibilities include neurogenic muscle dysfunctions, direct mechanical effect on the muscle fibers, biochemical alterations in the muscle intracellular substances, and neuropathies. Whatever the mechanism(s) involved, diminished grip force can progress to the point of significant occupational disability [Farkkila et al. 1982].

4. Bone Cysts

Degenerative changes in the bones of the fingers and wrists of workers using vibrating hand-held tools have been reported [McLaren and Camb 1937; Wilson et al. 1967]. The changes observed were mainly cysts, vacuoles, and areas of decalcification. It is clear, however, that bone cysts were not observed as being specific to HAVS [Casciu et al. 1968; Kumlin et al. 1971]. Kumlin et al. [1973] found that 7 of 35 lumberjacks (20%) studied showed radiographic presence of cysts and vacuoles in the metacarpal bones or the phalanges or both. These seven lumberjacks had used chain saws for 10 years or more and had one or more subjective symptoms typical of HAVS.

Radiographs of the hands were specifically included in a British health survey of occupational chain saw operators [James et al. 1975]. The objective of this investigation was the association, if any, between the presence of wrist and hand bone cysts and the occupational exposure to hand-arm vibration. X-rays of the hands were taken of 165 lumberjacks and

162 controls (manual laborers in the same environment as the lumberjacks). Each X-ray was read independently by three radiologists. Based on positive findings by two or more of the three readers, the incidence of vacuoles was 44% in the lumberjacks versus 33% in the controls. The difference between the incidence of vacuoles in lumberjacks and in other workers who did not use vibrating tools was not statistically significant.

In a 1977-78 study of Italian shipyard workers, 169 caulkers who worked with vibrating tools were compared with 60 welders and electricians who were not exposed to hand-arm vibration [Bovenzi et al. 1980]. Only the workers who used hand-held vibrating tools were X-rayed. In the caulkers, 51% exhibited HAVS in Stages 1 and 2 of the Taylor and Pelmear classification. Only 7% of the control group showed similar signs and symptoms. Bone cysts, vacuoles, or both were reported in the hand/wrist bones of 31% of the caulkers. No X-ray data from controls were available for comparison. This is similar to the incidence of bone cysts-vacuoles that James et al. [1975] reported in lumberjacks and controls not exposed to vibration.

A NIOSH study compared 205 foundry and shipyard chipping and grinding workers with 63 manual workers in the same industries who did not use vibrating tools [NIOSH 1984]. The frequency, location, and size of cysts and vacuoles in the hand and wrist bones as inicated on X-rays were compared. The films were read independently by two radiologists. The vibration-exposed and the control workers showed no statistical differences in frequency, location, or size of cysts and vacuoles. This study, with its adequate control group, supports the concept of James et al. [1975] that cysts and vacuoles occur in the hand and wrist bones of workers performing manual work but that these changes are not necessarily vibration related. The presence of bone cysts in workers exposed to vibration is therefore not a useful, objective diagnostic criterion for HAVS.

Gemne and Saraste [1987] surveyed the literature (125 published articles) to evaluate the evidence for and against radiological demonstrable effects of vibration on the bones and joints of the arm and hand. The authors concluded that the evidence does not support a "causal relationship between vibration exposure and the formation of bone cysts and vacuoles."

5. Central Nervous System

The inclusion of the central nervous system in the hand-arm vibration syndrome has been postulated by USSR and Japanese researchers [Griffin 1980; Matoba et al. 1975a, 1975b; Habu 1984]. The etiology of the "systemic effects" involves the concept that hand-arm vibration can impair central nervous system function through damage to the autonomic centers in the brain [Matoba et al. 1975a, 1975b].

The symptomatology alleged to be associated with vibration-induced central nervous system disturbances includes anxiety, depression, insomnia, headache, palmar sweating, vertigo, irritability, emotional instability, etc. [Habu 1984]. These signs and symptoms, derived from

statements made by the subjects being examined, usually have not been objectively assessed and are not specific to a single stressor such as vibration. Gemne and Taylor [1983], in summarizing the conclusions from an international symposium on hand-arm vibration and the central nervous system, stated that the present data do not support the hypothesis that exposure to hand-arm vibration may cause damage to the autonomic centers in the brain. In a study of 78 HAVS patients, Taylor et al. [1986] found no evidence to support an involvement of the central nervous system in HAVS.

6. Other Responses

Several other responses, whose significance in the identification and description of HAVS is unclear at present, may have some diagnostic value. These include the following:

a. In comparing the grip strength of nonvibration- and vibration-exposed workers, Miyashita et al. [1983] observed that grip strength progressively decreased as the total vibration exposure time increased: 52.5 kg grip strength in controls; 46.5 kg (–11.5%) in workers with up to 2,500 hr of total vibration exposure time; 40.1 kg (–24%) in wokers with more than 7,500 hr. Because of the wide differences in grip strengths among people, individual previbration-exposure values would be needed for grip strength to be a reliable diagnostic tool.

b. In the same control and exposure groups, sarcoplasmic enzyme levels (aldolase [ALD], creatinine phosphokinase [CPK], and lactic dehydrogenase [LDH]) in the exposure group increased over controls: 6% and 30% for ALD, 23% and 20% for CPK, 15% and 13% for LDH for <2,500 and >7,500 hr of vibrating tool use, respectively [Miyashita et al. 1983]. Adrenaline and noradrenalin in the urine of the exposed group were also measured and, when expressed as ng/mg creatinine, increased over control values by 260% and 269% for adrenaline, and 12% and 11% for noradrenalin for vibration exposures up to 4,000 hr and longer than 12,000 hr, respectively. Whether these changes are a general stress response or are specific to vibration stress remains to be proved.

c. It has been suggested that whole blood viscosity may play a role in HAVS and might be a useful tool in diagnosing HAVS between attacks. Whole blood viscosity in workers with HAVS was reported to be statistically significantly higher than in workers without HAVS ($p<0.01$) [Okada et al. 1982; Okada et al. 1987b]. However, Inaba et al. [1988] found no change in blood viscosity, hematocrit, cholesterol, and high density lipoproteins in vibration-exposed animals.

d. Okada et al. [1983] postulated a possible role of enhanced peripheral vasoconstriction caused by the exaggerated response of alpha adrenergic receptors in the arterial smooth muscles during attacks of white finger in workers with HAVS. These authors reported that during the cold provocation test (CPT), the plasma level of cyclic guanosine 3′, 5′-monophosphate (cyclic GMP) was increased to 170% over pretest levels in workers with

HAVS. The cyclic GMP levels did not increase in control subjects during the CPT. Without the CPT, the control and HAVS subjects had similar levels of cyclic GMP.

Phentolamine administered before and during the CPT and atropine injected subcutaneously immediately before the CPT inhibited the plasma rise in cyclic GMP during the CPT. The authors suggested that in individuals with HAVS, the increase in endogenous noradrenalin was enough to cause a significant increase in cyclic GMP in response to cold exposure (the CPT). This increased cyclic GMP response could result in an enhanced alpha-adrenergic response and a peripheral vasoconstriction.

e. An increased digital blood vessel reactivity to cold was reported by Bovenzi [1986]. Brown et al. [1988] measured radial digital artery blood flow in workers with and without HAVS before and after 20 min of chipping hammer use. Blood flow was measured by a 20-MHz, pulsed, ultrasonic Doppler velocimeter. Digital artery blood flow rate following the 20-min use of the chipping hammer increased substantially in the HAVS group, but there was little change in the control group. Pre-exposure flow rates were approximately equal in the control group and those with Stage 2 HAVS. The authors suggest that the 20-MHz Doppler velocimeter might be a valuable indicator tool for studying HAVS pathology.

C. EPIDEMIOLOGIC STUDIES

Epidemiologic studies of workers using hand-held vibrating tools have been conducted in Europe, North America, and Asia. Workers using gasoline-powered chain saws in forestry have been studied most frequently, followed by studies of workers using pneumatic chipping hammers in foundries, shipyards, and quarries, and pneumatic jack-leg drills in mining. These have been cross-sectional studies except for a few longitudinal studies of chain sawyers in the United Kingdom, Finland, and Japan. Cross-sectional studies examine a group of workers using hand-held vibrating tools in an industry at one particular time to determine the proportion of workers with HAVS (i.e., the prevalence of HAVS). Longitudinal studies of HAVS examine a group of workers at more than one point in time. The prevalence of HAVS is usually expressed as the prevalence of specific symptoms of HAVS such as vascular or neurologic symptoms. Sometimes studies report the latency of HAVS symptoms, the years of exposure to hand-arm vibration from the tool, and the hand-arm vibration acceleration level of the tool. The latency of a HAVS symptom is defined as the time from first use of a tool to the first appearance of the symptom.

1. Cross-Sectional Studies of HAVS

Table IV-8 summarizes pertinent information from cross-sectional studies of HAVS. The prevalence of vascular symptoms of HAVS is shown in the table because these symptoms were reported more consistently among epidemiologic studies of HAVS than the prevalence of other symptoms. If reported, the mean latency and mean years of exposure for the group of exposed workers is presented in Table IV-8; otherwise, the median is presented. If the mean or median latency or years of exposure are not reported, these variables are presented

Table IV-8.—Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level† (m/sec^2)	Author and date of publication	Country where study was conducted
Chain saw	Forestry	76	39	1-10 (range)	6.3 (mean) for workers with symptoms	N.A.§	Barnes et al. 1969	Australia
Chain saw	Forestry	82	47	4.5 (median)	7.5 (median)	N.A.	Allingham and Firth 1972	New Zealand
Chain saw	Forestry	296	47	8 (mean)	N.A.	N.A.	Hellstrom and Andersen 1972	Norway
Chain saw	Forestry	550	41	3-5 for 42% of workers with symptoms	≥5 for 84% of exposed workers	N.A.	Laitinen et al. 1974	Finland
Chain saw	Forestry	728	25	1-13 (range)	5 (mean)	N.A.	Wakisaka et al. 1975	Japan
Chain saw	Forestry	24	54	5 (mean)	N.A.	N.A.	Miura 1975	Japan
Chain saw	Forestry	87	38	4 (median)	7.8 (mean)	75	Matsumoto et al.1979	Japan
Chain saw	Forestry	402	36	13 (median)	12 (median)	N.A.	Suzuki 1979	Japan
Chain saw	Forestry	52	8	N.A.	< for 75% of exposed workers	N.A.	Iwata et al. 1980	Japan

(continued)

*Stages 1V, 2V, and 3V as defined by the revised Taylor-Pelmear staging system (Table IV-3).
†Frequency unweighted unless otherwise noted.
§Denotes data not available from articles cited.

Table IV-8 (Continued).–Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level† (m/sec^2)	Author and date of publication	Country where study was conducted
Chain saw	Forestry	365	29	5.6 (mean)	8.5 (mean)	N.A.	Olsen et al. 1981	Denmark
Chain saw	Forestry	107	62	7.9 (mean)	17.5 (mean)	N.A.	Patri et al. 1982	France
Chain saw	Forestry	323	28	7 (mean)	N.A.	68	Pelnar et al. 1982	Canada
Chain saw	Forestry	1,055	30	7.8 (mean)	N.A.	N.A.	Theriault et al. 1982	Canada
Chain saw	Forestry	89	54	7.3 (mean)	≥6 for 90% of exposed workers	N.A.	Brubaker et al. 1983	Canada
Chain saw	Forestry	279	18	N.A.	10.4 (mean)	N.A.	Harkonen et al. 1984	Finland
Brush saw	Forestry	506	6	6.4 (mean)	7 (mean)	59	Futatsuka 1984	Japan
Riveter	Boiler repair	78	61	≤10 for 87% of workers with symptoms	>10 for 58% of exposed workers	N.A.	Hunter et al. 1945	United Kingdom
Riveter, drill, shaver, bar	Aircraft manufacturer	340	25	≥20 for 50% of workers with symptoms	≤5 for 50% of exposed workers	10 (weighted)	Engstrom and Dandanell 1986	Sweden

(continued)

Table IV-8 (Continued).--Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level† (m/sec^2)	Author and date of publication	Country where study was conducted
Riveter, caulker, chipping hammer	Shipyard	195	75	N.A.	≥20 for >80% of exposed workers	1,183 (caulker and riveter only)	Oliver et al. 1979	United Kingdom
Chipping hammer	Pressed steel	31	93	1.7 (mean)	N.A.	N.A.	Marshall et al. 1954	United Kingdom
Chipping hammer	N.A.	49	41	8 (mean)	N.A.	N.A.	Lidstrom 1977	Sweden
Chipping hammer	Steel foundry	21	24	3.8 (mean)	>5 for 52% of exposed workers	N.A.	Suzuki 1978	Japan
Chipping hammer	Granite quarry	18	72	13 (mean)	20 (mean)	N.A.	Olsen and Nielsen 1979	Denmark
Chipping hammer, grinder	Shipyard	169	31	N.A.	7.3 (mean)	205	Bovenzi et al. 1980	Italy
Chipping hammer, grinder, scaler	Foundry	49	45	2.2 (mean)	3.8 (mean)	424	Taylor et al. 1981	United States
Chipping hammer	Iron foundry	25	64	≤10 for 96% of workers with symptoms	>10 for 52% of exposed workers	378	Matsumoto et al. 1979, 1981	Japan

(continued)

Table IV-8 (Continued).--Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level† (m/sec^2)	Author and date of publication	Country where study was conducted
Chipping hammer, grinder	Foundry	147	47	2 (mean)	2-3 (mean)	251	Behrens et al. 1982, 1984, Wasserman et al. 1984	United States
Chipping hammer, grinder	Shipyard	58	19	17 (mean)	12 (mean)	29	Behrens et al. 1982, 1984, Wasserman et al. 1984	United States
Chipping hammer	Quarry	69	36	N.A.	>15 for 72% of exposed workers	N.A.	Sakakibara et al. 1984	Japan
Chipping hammer	Limestone quarry	15	80	7.7 (mean)	37 (mean)	2,014	Taylor et al. 1984	United States
Hand grinder	Aircraft manufacture	112	21	0.7 (mean)	N.A.	N.A.	Dart 1946	United States
Hand grinder	Foundry	233	70	2 (mean)	N.A.	N.A.	Agate 1949	United Kingdom
Hand grinder	Foundry	54	35	15.3 (mean)	19 (mean)	20	Pelmear et al. 1975, Taylor et al. 1975c	United Kingdom
Hand grinder	N.A.	44	20	9 (mean)	N.A.	N.A.	Lidstrom 1977	Sweden

(continued)

Table IV-8 (Continued).--Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level† (m/sec^2)	Author and date of publication	Country where study was conducted
Hand grinder	Steel foundry	30	23	2 (mean)	>5 for 34% of exposed workers	N.A.	Suzuki 1978	Japan
Pedestal grinder	Foundry	37	86	1.8 (mean)	N.A.	382	Agate et al. 1946, Agate 1949	United Kingdom
Pedestal grinder	Foundry	34	94	4.5 (mean)	12.5 (mean)	N.A.	Pelmear et al. 1975	United Kingdom
Pedestal grinder	Foundry	26	96	1.8 (mean)	4.1 (mean)	125	Pelmear et al. 1975, Taylor et al. 1975c	United Kingdom
Pedestal grinder	Foundry	74	11	14 (mean)	N.A.	60	Taylor et al. 1975a, Taylor et al. 1975c	United Kingdom
Pedestal grinder	Foundry	12	100	0.9 (mean)	1.1 (mean)	122	Starck et al. 1983	Finland
Jack-leg drill	Metal mine	185	72	N.A.	N.A.	121	Iwata 1968	Japan
Jack-leg drill	Copper mine	68	22	5 (mean)	N.A.	N.A.	Miura 1975	Japan

(continued)

Table IV-8 (Continued).—Summary of epidemiologic studies of hand-arm vibration syndrome (HAVS)

Tool types	Industry	Number of workers exposed to H-A vibration	Prevalence of vascular symptoms* of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A vibration	H-A vibration acceleration level+ (m/sec^2)	Author and date of publication	Country where study was conducted
Jack-leg drill	Zinc mine	45	80	N.A.	>10 for 64% of exposed workers	335	Matsumoto et al. 1977, 1979	Japan
Jack-leg drill	Uranium mine	96	70	>10 for 91% of workers with symptoms	>10 for 78% of exposed workers	339	Robert et al. 1977	France
Jack-leg drill	Fluorspar mine	42	50	5.7 (mean)	9.9 (mean)	362	Chatterjee et al. 1978	United Kingdom
Jack-leg drill	Hard rock mine	58	45	7.2 (mean)	N.A.	20 (weighted)	Brubaker et al. 1986	Canada
Rock drill	Stone quarry	70	37	4 (mean)	N.A.	N.A.	Miura 1975	Japan
Rock drill	Construction	40	55	8 (mean)	N.A.	N.A.	Lidstrom 1977	Sweden
Rock drill	Anthracite coal mine	208	13	N.A.	>8 for 28% of exposed workers	N.A.	Moon et al. 1982	Korea
Pavement breaker	Gas supply	851	10	N.A.	N.A.	195	Walker et al. 1985	United Kingdom
Motorcycle	Speedway racing	32	94	5 (mean)	N.A.	416	Bentley et al. 1982	United Kingdom
Riveter, chipping hammer, grinder	Railroad	1,028	13	9.2 (mean)	>10 for 32% of exposed workers	N.A.	Zeng-Shun et al. 1986	China

in the form given in the cited article. A method of standardization (presented in Appendix A) was applied to the acceleration measurements of hand-arm vibration if they were reported in a study. This method of standardization makes the measurements comparable with each other.

The prevalence of vascular symptoms of HAVS in Table IV-8 ranged from 6% to 100%. To evaluate the relative significance of these prevalence values, they must be compared with the background rate of vascular symptoms among worker populations that have not been exposed to hand-arm vibration. Nineteen of the studies reported the prevalence of vascular symptoms among a group of control workers who had not been exposed to hand-arm vibration and had worked at the same site as the exposed workers [Allingham and Firth 1972; Hellstrom and Andersen 1972; Taylor et al. 1974; Pelmear et al. 1975; Taylor et al. 1975a; Matsumoto et al. 1977; Chatterjee et al. 1978; Bovenzi et al. 1980; Matsumoto et al. 1981; Moon et al. 1982; Patri et al. 1982; Pelnar et al. 1982; Theriault et al. 1982; Brubaker et al. 1983; Behrens et al. 1984; Harkonen et al. 1984; Walker et al. 1985; Brubaker et al. 1986; Zeng-Shun et al. 1986]. The prevalence of vascular symptoms among these 19 groups of control workers ranged from 0% to 14% with a mean prevalence of 5.4% and a median of 4%.

Most of the studies listed in Table IV-8 had prevalence rates of vascular symptoms that were well above background rates. More than half of the studies had HAVS prevalence rates that were greater than 40%. Epidemiologic studies of HAVS clearly confirm an association between vascular symptoms and exposure to hand-arm vibration from hand-held vibrating tools and workpieces. These studies also provide clues to HAVS prevention. A study showing a relatively low prevalence of vascular symptoms or a relatively long latency of vascular symptoms may provide such clues. In addition, some of the studies in Table IV-8 had tool vibration measurements taken at the time of the cross-sectional medical evaluations, and these studies indicate the exposure-response relationship between HAVS and hand-arm vibration.

Nine of the studies in Table IV-8 [Taylor et al. 1975a; Iwata et al. 1980; Behrens et al. 1982, 1984; Moon et al. 1982; Futatsuka 1984; Harkonen et al. 1984; Walker et al. 1985; Zeng-Shun et al. 1986] reported prevalence rates of vascular symptoms among the exposed workers that were nearly within range of the prevalence rates for the control group (i.e., from 0% to 14%). The study by Iwata et al. [1980] found an 8% prevalence of vascular symptoms among chain saw operators. A control group was not included in this study. The low prevalence rate may have been due to the high proportion (75%) of chain saw operators in this study who had less than 10 years of exposure to hand-arm vibration from chain saws, or it may have been that the use of antivibration chain saws by 1980 reduced exposure. Although the authors did not report the latency of vascular symptoms for this group of chain saw operators, the average latency for other studies of chain saw operators in Table IV-8 ranged from 4 to 13 years. Thus the group of chain saw operators in the study by Iwata et al. [1980] probably had not experienced sufficient exposure for all cases of HAVS (with vascular symptoms) to manifest themselves.

Harkonen et al. [1984] observed the relatively low 18% prevalence of vascular symptoms of among a group of chain saw operators. A control or reference group of peat bog workers were examined and were found to have a 3% prevalence of vascular symptoms. The authors reported that the difference in prevalence between the exposed and control groups was statistically significant ($p<0.001$).

In the study of workers using gasoline-powered brush saws, Futatsuka [1984] reported a 6% prevalence of vascular symptoms, the lowest reported prevalence in Table IV-8. No control group was examined in this study. Among the exposed group, the mean duration of exposure to brush sawing was 7 years and the mean latency of vascular symptoms was 6.4 years. Because the mean exposure time was greater than the mean latency, the prevalence of vascular symptoms in this group of brush saw operators would not be likely to increase appreciably with further exposure. The author reported some factors that probably contributed to the low prevalence of vascular symptoms in these brush saw operators. Workers used the brush saws for 4 to 5 months a year, during the warmer season of May to September. Also, since 1970, the use of the brush saw was limited to 2 to 3 hr/day. Before that date, workers were using the brush saws up to 6 hr/day. The effect of limiting the number of hours of brush saw use per day was revealed in the difference between the prevalence of vascular symptoms in workers who started using the brush saws in 1961 and 1962 (12% peak prevalence from 1961 to 1980) and workers who started in 1969 and 1970 (0% prevalence from 1969 to 1980).

A relatively low prevalence of vascular symptoms among chipping and grinding workers was reported by Behrens et al. [1982, 1984]. In this study of workers using pneumatic chipping hammers and grinders at a shipyard, the prevalence of vascular symptoms was 19%. A control group working at the shipyard was included in this study, and the prevalence of vascular symptoms in the control group was 0%. The difference in prevalence rates between the exposed and control groups was statistically significant ($p<0.0001$) [Wasserman et al. 1982]. In this group of chipping hammer and grinder operators, the prevalence would probably increase over time because the mean exposure time (12 years) was less than the mean latency for vascular symptoms (17 years).

Four other studies in Table IV-8 showed relatively low prevalence rates of vascular symptoms [Taylor et al. 1975a; Moon et al. 1982; Walker et al. 1985; Zeng-Shun et al. 1986]. All of these studies reported the prevalence of vascular symptoms in a control group of workers. The study of pedestal grinders by Taylor et al. [1975a] found an 11% prevalence in the exposed group and 4% in the control group. The authors did not compare the prevalence statistically. Also, the authors did not report a mean exposure time for this group of pedestal grinders, although the mean latency of vascular symptoms was reported as 14 years. In the study by Moon et al. [1982], the prevalence of vascular symptoms among anthracite miners using pneumatic rock drills was 13%, whereas the prevalence in the control group was 0.9%. The statistical comparison of prevalence rates was significant ($p<0.05$). Similarly, Zeng-Shun et al. [1986] reported that the prevalence of vascular symptoms among

railroad workers using riveters, chipping hammers, and grinders was 13%, whereas the control group prevalence was 1.6% ($p<0.001$).

The Walker et al. [1985] study of workers in the gas industry who used pavement breakers is the only study in Table IV-8 in which the difference in prevalence between the exposed and control groups was not statistically significant. The prevalence rates of vascular symptoms in the workers using pavement breakers and the workers not exposed to hand-arm vibration were both 10%. After adjustment for differences in the ages of the exposed and control groups, the age-adjusted prevalence was 12% in the exposed group and 10% in the control group. This comparison was not statistically significant. The authors did not report the latency of vascular symptoms, but the prevalence of vascular symptoms among workers using the pavement breakers for 1 to 5 years was 9%, whereas the prevalence in workers using the pavement breakers more than 20 years was 18%. Two factors may have contributed to the nonsignificant and relatively low prevalence of vascular symptoms in this group of pavement breaker operators. First, the average latency of vascular symptoms may have been longer than latencies recorded for exposures to other tool types because the prevalence among the exposed workers did not increase much above that of the control group until the exposed workers had more than 20 years of exposure. Second, the prevalence of vascular symptoms in the control group was relatively high (10% prevalence in this control group compared with a mean prevalence of 5.4% in 19 control groups from Table IV-8).

A recent survey of workers who used impact power tools indicated a prevalence rate of 29% for numbness and tingling of the fingers and 17% for white finger. The workers used the impact tools for an average of 23% of the workday. Frequency-weighted acceleration levels exceeded 12 m/sec^2 for all measurements, and they averaged approximately 24 m/sec^2 for chipping hammers and rammers. The mean cumulative exposure for each worker over a working lifetime was nearly 4,000 hr. The 17%- to 29%-prevalence of signs and symptoms of HAVS occurred even though the daily tool use time was only about 2 hr [Musson et al. 1989].

Forty-four studies listed in Table IV-8 included some information about the latency of vascular symptoms among exposed workers. Thirty-three of these 44 studies reported the mean latency of vascular symptoms. These 33 mean latencies ranged from 0.7 to 17 years, with a mean value of 6.3 years. When compared with the range and mean values of the 33 mean latencies, four studies [Pelmear et al. 1975; Taylor et al. 1975a; Olsen and Nielsen 1979; Behrens et al. 1982, 1984] reported relatively long mean latencies. A relatively long latency of vascular symptoms suggests the possibility that some workers may never show vascular symptoms.

Olsen and Nielsen [1979] found a 13-year mean latency for vascular symptoms among 18 (out of 20 total) workers using pneumatic chipping hammers at a granite quarry. Despite the relatively long latency, the prevalence of vascular symptoms was 72% (13/18). The high

prevalence is expected because the mean duration of exposure (20 years) for the workers was greater than the mean latency. For the 18 quarry workers, the individual latencies varied from 0 to 27 years. A latency of 0 years may indicate that this quarry worker had vascular symptoms before starting to use the chipping hammer at this quarry. The next four lowest latencies for individual workers were 2, 4, 7, and 7 years. Thus the individual variation in onset of vascular symptoms shows that all workers in this group could not be protected by limiting the years of exposure.

In a study of shipyard workers using chipping and grinding tools [Behrens et al. 1982, 1984], the mean latency of vascular symptoms was 17 years, with a range of 4 to 35 years. Eleven shipyard workers had vascular symptoms, and 5 of these 11 workers had latencies of less than 10 years. At the high end of the range, 2 of the 11 workers had latencies greater than 30 years. Therefore, the mean latency was relatively long among these shipyard workers because of the wide variation in individual latencies, as was also shown in the study by Olsen and Nielsen [1979].

The Pelmear et al. [1975] study of hand grinders found a 13.7-year mean latency of vascular symptoms, and the Taylor et al. [1975a] study of pedestal grinders found a 14-year mean latency. Neither of these studies reported the range of latencies or individual latency values. The mean exposure time (19 years) for workers using hand grinders in a foundry [Pelmear et al. 1975] was greater than the mean latency (13.7 years), which resulted in a substantial 35% prevalence of vascular symptoms. Workers using pedestal grinders in a foundry [Taylor et al. 1975a] had an 11% prevalence (and a 14-year mean latency), but the authors did not report the average years of exposure. As a result, the relationship between the latency, the years of exposure, and prevalence in this group of workers is not known.

Vibration measurements were reported for 23 of the studies listed in Table IV-8. These 23 studies are ranked in Table IV-9, in descending order, from highest to lowest hand-arm vibration acceleration level. The prevalence of vascular symptoms, the tool type, and the publication reference are also repeated in Table IV-9. The 23 hand-arm vibration acceleration levels in Table IV-9 range from 10 to 2,014 m/sec^2, and the mean and median values were, respectively, 312 and 195 m/sec^2. The relationship between the hand-arm vibration acceleration level and the prevalence of vascular symptoms for these 23 studies was tested for linearity by calculating a correlation coefficient. The correlation coefficient was 0.67 and was statistically significant at the 1% level ($p<0.01$). Thus the prevalence of vascular symptoms tends to increase as the hand-arm vibration acceleration level increases for these 23 studies.

In general, the studies in Table IV-9 that showed hand-arm vibration acceleration levels greater than the median acceleration level for these 23 studies (195 m/sec^2) also showed prevalence rates of vascular symptoms greater than 40%. Only the Bovenzi et al. [1980] study deviated from this trend. Also, in general, the studies in Table IV-9 with acceleration levels less than or equal to the median acceleration level had prevalence rates of less than

Table IV-9.—Hand-arm vibration acceleration levels ranked from highest to lowest for studies listed in Table IV-8.

Hand-arm vibration acceleration level (m/sec^2)*	Prevalence of vascular symptoms of HAVS (%)	Tool type	Author and date of publication
2,014	80	Chipping hammer	Taylor et al. 1984
1,183†	75	Riveter, caulker, chipping hammer	Oliver et al. 1979
424	45	Chipping hammer, grinder, scaler	Taylor et al. 1981
416	94	Motorcycle	Bentley et al. 1982
382	86	Pedestal grinder	Agate et al. 1946 Agate 1949
378	64	Chipping hammer, grinder	Matsumoto et al. 1979, 1981
362	50	Jack-leg drill	Chatterjee et al. 1978
339	70	Jack-leg drill	Robert et al. 1977
335	80	Jack-leg drill	Matsumoto et al. 1977, 1979
251	47	Chipping hammer, grinder	Behrens et al. 1984 Wasserman et al. 1984
205	31	Chipping hammer, grinder	Bovenzi et al. 1980
195	10	Pavement breaker	Walker et al. 1985
125	96	Pedestal grinder	Pelmear et al. 1975 Taylor et al. 1975c
122	100	Pedestal grinder	Starck et al. 1983
121	72	Jack-leg drill	Iwata 1968

(continued)

See footnotes at end of table.

Table IV-9 (Continued).—Hand-arm vibration acceleration levels ranked from highest to lowest for studies listed in Table IV-8.

Hand-arm vibration acceleration level (m/sec^2)*	Prevalence of vascular symptoms of HAVS (%)	Tool type	Author and date of publication
75	38	Chain saw	Matsumoto et al. 1979
68	28	Chain saw	Pelnar et al. 1982
60	11	Pedestal grinder	Taylor et al. 1975a Taylor et al. 1975c
59	6	Brush saw	Futatsuka 1984
29	19	Chipping hammer, grinder	Behrens et al. 1984 Wasserman et al. 1984
20§	45	Jack-leg drill	Brubaker et al. 1986
20	35	Hand grinder	Pelmear et al. 1975 Taylor et al. 1975c
10§	25	Riveter, drill, shaver, bar	Engstrom and Dandanell 1986

*Frequency unweighted except as noted.
†Measured riveter and caulker only.
§4-hr ISO weighted value.

40%. Four studies (Iwata [1968]; Pelmear et al. [1975]; Starck et al. [1983]; and Brubaker et al. [1986]) did not follow this trend.

In the Bovenzi et al. [1980] study of shipyard workers using chipping hammers and grinders, the hand-arm vibration acceleration level was 205 m/sec^2 and the prevalence of vascular symptoms was 31%. The authors did not report the latency of vascular symptoms for those shipyard workers with symptoms. They did report that the mean duration of exposure to hand-arm vibration was 7.3 years for the 169 shipyard workers studied, and that 46% had been exposed for 5 years or less and 44% for 6 to 10 years. Also, in this study, the mean duration years of exposure for shipyard workers with vascular symptoms was 10.2 years, whereas the mean duration of exposure for shipyard workers without vascular symptoms was 6.1 years. Thus at least 46% of the shipyard workers studied had been exposed for fewer years (i.e., <6) than the mean years of exposure for workers with vascular symptoms (approximately 10 years). These results indirectly indicate that the prevalence of vascular symptoms in the group of shipyard workers studied by Bovenzi et al. [1980] would probably increase over time and with further exposure.

Two other studies in Table IV-9 that deviate from the trend of a linear relationship between hand-arm vibration acceleration levels and prevalence of vascular symptoms are the pedestal grinder studies of Pelmear et al. [1975] and Starck et al. [1983]. For both of these studies, the prevalence of vascular symptoms was exceptionally high (96% for Pelmear et al. [1975] and 100% for Starck et al. [1983]). The authors for both of these studies attributed the high prevalence rates to the use of zirconium wheels on the pedestal grinding machines because the prevalence of vascular symptoms increased markedly in their study groups when wheels of "softer" material were replaced by "harder" zirconium wheels. The Taylor et al. [1975a] study of pedestal grinders in Table IV-9 reported a relatively low prevalence (11%), an average acceleration level of 100 m/sec^2, and the use of "soft" wheels on the pedestal grinding machines. The only other study of pedestal grinders in Table IV-9 (by Agate et al. [1946]) did not specify the type of wheels used.

Brubaker et al. [1986] found that 45% of hard rock miners using jack-leg drills had vascular symptoms but that the hand-arm vibration acceleration level for the jack-leg drills was frequency weighted at 20 m/sec^2, a relatively low acceleration level. Acceleration levels for jack-leg drills were reported for four other studies in Table IV-9; the levels were all greater than 100 m/sec^2, and for three of these studies, the levels were greater than 300 m/sec^2. Brubaker et al. [1986] measured the jack-leg drills "under actual drilling conditions at the mine sites according to guidelines specified in ISO 5349." The authors pointed out that ISO 5349 [ISO 1986] suggests that 50% of the workers exposed to a frequency-weighted vibration level of 20 m/sec^2 will develop vascular symptoms after a 3- to 5-year exposure. Therefore, a 45% prevalence of vascular symptoms may not be unexpected for this group of workers. Brubaker et al. [1986] did not compare their measurements of jack-leg drills to those of other investigators and thereby offered no explanation for the relatively low acceleration levels they reported.

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The study by Iwata [1968] shows a similar result to that of Brubaker et al. [1986]. For workers using jack-leg drills, Iwata [1968] found a relatively high prevalence of vascular symptoms (72%) but a below-average hand-arm acceleration level (121 m/sec^2). Iwata's study [1968] was reported earlier than the other studies of jack-leg drills in Table IV-9. He could not compare his hand-arm vibration measurements with other studies of jack-leg drills, and he did not perform the measurements according to international standards recommended during the late 1960s.

2. Longitudinal Studies of HAVS

Longitudinal studies of workers exposed to hand-arm vibration demonstrate the effect that lowering the acceleration level of a hand-held vibrating tool has on the prevalence of HAVS. Three longitudinal studies have been conducted; all of them concern forestry workers using gasoline-powered chain saws. Tables IV-10, IV-11, and IV-12 summarize the longitudinal studies of chain saw operators in the United Kingdom, Finland, and Japan, respectively. Neither a longitudinal nor a cross-sectional study of chain saw operators has been done in the United States.

Gasoline-powered chain saws in the 1950s were large and difficult to maneuver, and their use was limited to 1 to 2 hr per day. In the 1960s, technical improvements in the design of chain saws allowed their use to be extended to 4 to 6 hr per day. By the early 1970s, initial reports of HAVS among chain saw operators were made public. In the early 1970s, chain saws were redesigned to lower the vibration acceleration levels. These saws are called antivibration chain saws. By the 1980s, the prevalence of HAVS among chain saw operators had been reduced.

Table IV-10 presents a summary of the longitudinal studies of antivibration chain saw operators in the United Kingdom reported by Taylor and co-workers [Taylor et al. 1974, 1975a, 1975b, 1975c, 1977; Riddle and Taylor 1982]. An original group of 46 forestry workers using gasoline-engine-powered chain saws was followed from 1970 to at least 1981. In 1970, the prevalence of vascular symptoms in this group was 85%. Antivibration chain saws were introduced in 1973, and by 1975, 73% of the 44 remaining chain saw operators had vascular symptoms. In 1981, 28 of the original 46 chain saw operators were still working in forestry, and 46% of these workers had vascular symptoms. Taylor and co-workers [Riddle and Taylor 1982] noted that some workers had recovered and no longer had vascular symptoms after the introduction of antivibration chain saws. Also in 1981, 18 chain saw operators who had worked exclusively with antivibration chain saws

Table IV-10.—Summary of epidemiologic studies of forestry workers using chain saws in the United Kingdom*

Number of forestry workers	Prevalence of vascular symptoms of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A† vibration	H-A vibration acceleration level (m/sec^2)
46 in 1970 (non A-V§ chain saw users only)	85	N.A.**	6 (mean)	150-350 for non A-V chain saws
44 in 1975 (non A-V and A-V chain saw users)	73	3 (mean)	11 (mean)	150-350 for non A-V and 15-50 for A-V chain saws
18 in 1981 (A-V chain saw users only)	17	N.A.	5 (mean)	15-50 for A-V chain saws

*Sources: Taylor et al. [1974, 1975a, 1975b, 1975c, 1977], Riddle and Taylor [1982].
†Hand-arm.
§Antivibration.
**Denotes data not available from articles cited.

Table IV-11.—Summary of epidemiologic studies of forestry workers using chain saws in Finland*

Number of forestry workers	Prevalence of vascular symptoms of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A† vibration	H-A vibration acceleration level (m/sec^2)
66 in 1972	34	5 (mean)	N.A.§	162
66 in 1977	10	5 (mean)	N.A.	17
66 in 1983	5	5 (mean)	N.A.	16

*Sources: Pyykko [1974a] and Pyykko et al. [1978, 1982b, 1986b].
†Hand-arm.
§Denotes data not available from articles cited.

Table IV-12.—Summary of epidemiologic studies of forestry workers using chain saws in Japan*

Number of of forestry workers	Years workers began using chain saws	Prevalence of vascular symptoms of HAVS (%)	Latency of vascular symptoms of HAVS (years)	Years of exposure to H-A† vibration	H-A vibration acceleration level (m/sec^2)
123	1958 and 1959	63	N.A.§	14 (mean)	111-304 for chain saws made in 1966
114	1968 and 1969	22	N.A.	7 (mean)	49-105 for chain saws made in 1970
103	1974 and 1975	2	N.A.	5 (mean)	10-33 for chain saws made in 1975
1,330	1956 to 1979 (includes workers given above)	28	6.4 (mean)	10 (mean)	(same as above)

*Source: Futatsuka and Ueno [1985, 1986].
†Hand-arm.
§Denotes data not available from articles cited.

were examined and were found to have a 17% prevalence of vascular symptoms. Hand-arm vibration acceleration levels for chain saws measured before the introduction of antivibration designs ranged from 150 to 350 m/sec^2, whereas antivibration chain saws ranged from 15 to 50 m/sec^2.

Pyykko and co-workers [Pyykko 1974a and Pyykko et al. 1978, 1982b, 1986b] in Finland followed 66 forestry workers using gasoline-powered chain saws from 1972 until 1983 (see Table IV-11). The prevalence of vascular symptoms in this group was 34% in 1972 and reached a peak of 38% in 1975. Antivibration chain saws were introduced in the mid-1970s, and the prevalence of vascular symptoms in the study group of 66 chain saw operators had decreased to 5% by 1983. Hand-arm vibration acceleration levels on chain saws measured in 1972 were 162 m/sec^2. Antivibration chain saws were measured at 17 m/sec^2 in 1977 and at 16 m/sec^2 in 1983.

A longitudinal study of forestry workers using gasoline-engine-powered chain saws in Japan was conducted by Futatsuka and Ueno [1985, 1986]. Forestry workers employed by the Japanese government in national forests have been examined on a regular basis since 1965, and results of these examinations have been reported through 1980. Chain saw operators in this study were grouped according to the year when they first began using a chain saw (see Table IV-12). The peak prevalence of vascular symptoms for chain saw operators who began chain saw use in 1958 and 1959 was 63%. For chain saws measured in 1966 (the earliest year measurements were available), the vibration acceleration level ranged from 111 to 304 m/sec^2. The peak prevalence for chain saw operators who began chain saw use in 1968 and 1969 was 22%. For nonantivibration chain saws measured in 1970, acceleration levels ranged from 49 to 105 m/sec^2. The peak prevalence for chain saw operators who began working in 1974 and 1975, after the introduction of antivibration chain saws, was only 2%. For antivibration chain saws measured in 1975, acceleration levels ranged from 10 to 33 m/sec^2.

The Japanese government in the early 1970s began restricting the number of hours per day that chain saws could be operated. Between 1970 and 1975, chain saw use was limited to 2 hr/day for the national forestry operations. Futatsuka and Ueno [1985, 1986] reported that of the 185 chain saw operators who began using chain saws in 1972 and 1973, only 3 (2%) had vascular symptoms as of 1980 (not shown in Table IV-12). These chain saw operators had been exposed for at least 7 years, a period of time greater than the 6.4-year mean latency of vascular symptoms for the entire study population (see Table IV-12).

3. Summary of Epidemiologic Studies of HAVS

Epidemiologic studies of workers using hand-held vibrating tools show a strong association between exposure to hand-arm vibration and vascular symptoms of HAVS or Raynaud's phenomenon. Only 9 of the cross-sectional studies presented in Table IV-8 had relatively

low prevalence rates of vascular symptoms (i.e., less than 20%) compared with prevalence rates of vascular symptoms in 19 control groups. In five of these nine studies, the prevalence of vascular symptoms in the exposed group of workers was compared with the prevalence in a control group. In four of these five studies, the prevalence was significantly higher in the exposed group than in the control group even though the prevalence of vascular symptoms in the exposed group was relatively low.

The study [Futatsuka 1984] with the lowest prevalence of vascular symptoms (6%) in Table IV-8 was the only one of the eight studies with relatively low prevalence rates that offered a plausible reason for the low prevalence. In this study of brush saw workers, the use of the brush saw was restricted to 4 to 5 months/year during the summer season and to 2 to 3 hr/day. The conditions of using the brush saw in this study [Futatsuka 1984] seemed to produce a very low prevalence despite a hand-arm vibration acceleration level of 59 m/sec^2. The study in Table IV-9 with the lowest frequency-weighted acceleration level (10 m/sec^2) [Engstrom and Dandanell 1986] reported a prevalence of vascular symptoms (25%) among workers using pneumatic drills, riveters, shavers, and bucking bars that was four times greater than that reported by Futatsuka [1984] although the frequency-weighted acceleration level (10 m/sec^2) was less. The vibration frequencies produced by the tools used in the Engstrom and Dandanell [1986] study were much higher (up to 10,000 Hz) than those reported by Futatsuka [1984].

Twenty-three studies from Table IV-8 included measurements of hand-arm vibration acceleration levels. These studies showed a statistically significant linear relationship between increasing exposure to hand-arm vibration and increasing prevalence of vascular symptoms of HAVS. Even though this relationship could be demonstrated statistically, only 3 of the 6 studies reporting the lowest acceleration levels (i.e., less than or equal to 60 m/sec^2) among these 23 studies (see Table IV-9) also reported relatively low prevalence of vascular symptoms (i.e., less than 20%). The relationship between hand-arm vibration exposure and the prevalence of vascular symptoms of HAVS among these 23 studies was more consistent for those with higher acceleration levels than for those with lower levels.

The longitudinal studies of chain saw operators were able to demonstrate an appreciable decrease in the prevalence of vascular symptoms after the introduction of the antivibration chain saw. The lower acceleration level of the antivibration chain saw apparently contributed to the decrease, although the amount of decrease varied from study to study. Riddle and Taylor [1982] reported a 17% prevalence among 18 workers exclusively using antivibration chain saws; Pyykko et al. [1986b] reported a 5% prevalence for 66 workers in 1983 who had used antivibration chain saws since the mid-1970s; and Futatsuka and Ueno [1985] reported a 2% prevalence in 185 workers who started using chain saws in the mid-1970s when antivibration chain saws were introduced. The exceptionally low prevalence in Japanese chain saw operators [Futatsuka and Ueno 1985] could reflect the requirement that they do not operate chain saws for more than 2 hr/day [Saito 1987].

4. Conclusions from Epidemiologic Studies of HAVS

HAVS was found among workers exposed to hand-arm vibration in all of the epidemiologic studies cited, regardless of the level of vibration exposure. These studies therefore provide no basis for determining an exposure level at which no cases of HAVS would occur. However, the studies do provide ample evidence that the use of vibration-producing, hand-held tools is associated with the development of HAVS.

D. SCREENING AND DIAGNOSTIC TESTS

The signs and symptoms characteristic of HAVS are also observed individually or in various combinations in some other disorders. No single sign or symptom is specific to HAVS alone. This introduces uncertainties and difficulties in the classification and diagnosis of HAVS. Regardless of the complaints and symptoms presented by an individual, a diagnosis of HAVS is not justified unless a history of using vibrating tools is present [Taylor 1982a, 1982b; Gemne 1982; NIOSH 1984; Pyykko and Starck 1986; Matoba and Sakurai 1987; Farkkila 1987]. Several tests can be used to help substantiate a clinical diagnosis of HAVS.

- **Screening Tests**

  Vascular Assessment

  Adson's test (neck rotation and deep inspiration)
  Allen's test (compression of vessels at wrist)
  Lewis-Prusik test (nailbed compression)
  Doppler test (segmental arm and digital blood flow and pressure)
  Cold provocation test (immersion of digits and hands)

  Neurologic Assessment

  Light touch (cotton wool)
  Pain (pin prick)
  Temperature (cold and heat appreciation)
  Aesthesiometry (two-point and depth-sense discrimination)
  Vibration perception threshold
  Phalen's test (wrist flexion)
  Tinel's test (carpal tunnel percussion)

  Musculoskeletal Assessment

  Grip strength (dynamometer)
  Pinch test (thumb and fingers)
  Manipulative dexterity

- **Laboratory/Hospital Tests**

  Vascular Assessment

  Doppler (peripheral segmental blood flow and pressure in arms)
  Digital plethysmography (at rest and following cold stress)
  Digital systolic pressure (plethysmography with local cooling, and whole body cooling as well if necessary)
  Cold provocation test (immersion of digits and hands with recording of digital temperatures)

  Neurologic Assessment

  EMG (to record median and ulnar sensory and motor nerve conductivity)

  Hematologic Assessment

  Total and differential, sedimentation rate, blood viscosity, uric acid, rheumatoid factor, antinuclear antibodies, cryoglobulins, serum protein electrophoresis

  Urinalysis

  Proteinuria, glycosuria

  X-rays

  Cervical spine and ribs (to exclude costoclavicular syndrome)

Olsen [1988] conducted a comparative study of diagnostic tests for vibration white finger. In the four tests used, various aspects of finger blood flow and finger blood pressure were measured. The study concluded that the finger color test may be as valuable as finger systolic blood pressure for diagnostic purposes. The diagnostic values of some of the tests are discussed below.

1. Cold Provocation Test (CPT)

Two generic variations of the CPT have been used in studying HAVS. The versions vary mainly in the length of exposure to the stimulus, the temperature of the cold stimulus, and the responses measured. The subject must be in thermal balance before the test. This is achieved by having the subject rest for about 30 minutes in a room with air temperatures from 21° to 24°C (70° to 75°F).

a. Short Exposure

In the short exposure version of the CPT, the hand is immersed up to the wrist in ice water for 1 to 3 minutes. Blood flow in the fingers of the opposite hand can be measured by plethysmography before, during, and after immersion. The most important measurements involve the reduction in blood flow during cold exposure and the recovery of blood flow following immersion. Finger rewarming time after a short (3-minute) immersion in water at 10°C (50°F) was 1.4 minutes for the control subjects and 2.9 minutes for workers with HAVS ($p = <0.01$). Excluded from the HAVS group were those whose finger temperature did not rise to 30°C during body heating at a room temperature of 40° to 45°C (104° to 113°F) for 15 minutes [Welsh 1986]. Niioka et al. [1986] found that the finger skin rewarming time after a CPT could distinguish between HAVS patients and controls with a false discrimination of 6%, a sensitivity of 80%, and a specificity of 100%.

b. Long Exposure

In the long exposure CPT, after the subject has achieved thermal equilibrium in a thermal neutral room, the arm up to the elbow or shoulder is immersed in water at 10° to 15°C (50° to 59°F) for a period of 10 to 15 minutes. After the hand and arm have been removed from the cold water and dried, the time is measured for the finger skin to regain its color and/or temperature (indicating vasodilation and resumption of blood flow). Skin temperature below control values, 5 and 10 minutes after a 10-minute cold exposure, gave a correct diagnosis in 80% to 90% of workers with HAVS and workers without HAVS who had more than 5,000 hr of chain saw use [Kurumatani et al. 1986; Welsh 1986; Niioka et al. 1986]. However, finger-skin temperature response did not distinguish between subjects who did not use vibrating tools, those with Raynaud's disease, and those with less than 5,000 hr of saw use [Welsh 1986; Kurumatani et al. 1986; Niioka et al. 1986].

The response of finger-skin temperature to cold exposure in nonsymptomatic, vibration-exposed and unexposed workers was reported by Scheffer and Dupuis [1989] for laboratory and field studies. The test conditions (5°C air temperature, vibration acceleration of 6.3 m/sec^2 [frequency-weighted], grip force of 15 N, and push force on the tool of 40 N) all contributed to the skin temperature response. The authors suggest that improved protection against cold (e.g., heated tool handles) could be an effective preventive technique.

The response of finger systolic blood pressure (FSBP) to finger cooling has been used to measure changes in peripheral vascular response to cooling in vibration-exposed workers. Olsen et al. [1981] and Olsen and Nielsen [1979] reported that FSBP changes predict 60% to 85% of workers with vibration white finger even when the history of HAVS is not known. Their subjects, however, had vibration white finger, Stage 2 to 3 [Taylor and Pelmear classification]. In less severe cases of HAVS (Stage 1), neither FSBP nor peripheral blood flow measurements were very sensitive. During field conditions, however, measuring FSBP is easier than measuring blood flow [Pyykko et al. 1986a]. The fingernail compression test may also be used. The occurrence of spastic vasospasm and pain in the finger is noted.

Hack et al. [1986] compared responses on the cold provocation test and reactive hyperemia with the history of vibration-induced white finger in workers with no symptoms, symptoms of tingling and numbness, and symptoms of HAVS. The tests distinguished between groups, but only about 60% of the subjects fell into the correct staging category.

Bovenzi [1986] reported that the skin temperature recovery time following finger cooling was significantly prolonged in workers with HAVS, indicating that the workers with HAVS had a more severe and prolonged vasoconstrictive response to the cold provocation test.

Gemne et al. [1986] found the finger blood flow at vasodilatation (finger arterial inflow) after occlusion was less in the group with HAVS than in a reference group without HAVS. Peripheral resistance was also higher in the HAVS group. The authors suggested that the increased peripheral resistance in the HAVS group may be due to a local defect in the vessels with a reduction in flow and intramural pressure.

Arneklon-Nobin et al. [1987] reported that the FSBP, the ratio of the finger systolic blood pressure to the arm systolic blood pressure (FSBP/ASBP), and finger skin temperature measured before and after vasodilatation by body warming are altered in vibration-exposed workers. Finger skin temperature and FSBP were lower in the workers with HAVS both before and after vasodilatation. Digital rewarming time was markedly slower in the vibration-exposed individuals. These studies suggest that measuring the finger skin temperature and FSBP before and after finger cooling and after finger rewarming could be useful diagnostic tests.

Bovenzi [1988] suggested that the finger systolic blood pressure response with finger cooling and ischemia may be a useful objective test for vascular hyperactivity in subjects with HAVS. In his study, the test had a sensitivity to increased arterial tone of 100% and a specificity (negative test without HAVS) of 87%.

2. Plethysmography

Finger plethysmography, a technique used for measuring finger blood flow, is based on the fact that with each heart contraction, the volume of the fingers increases. This change in finger volume can be measured with a photocell or strain gauge. In practice, the photocell plethysmograph is used more frequently than the strain gauge. The instrumentation for a photocell plethysmograph and the test conditions that can be used in field and laboratory studies have recently been published [Samueloff et al. 1984; Samueloff et al. 1981]. Finger plethysmography, after local cooling, has been recommended as an objective test for HAVS [Pelnar 1986]. Vibration instead of cold water may be used as the stimulus. Standardization of the test procedure and strict adherence to the test protocol are of utmost importance if the results from different studies are to be compared.

3. Aesthesiometry

One of the tests of peripheral neural changes is the finger tip two-point and depth discrimination tests. A version of the test instrumentation has been described [Carlson et al. 1979; Carlson et al. 1984]. As with other semiobjective tests, standardization of the instrumentation and strict adherence to the test protocol are very important (e.g., small differences in the pressure applied by the finger tip against the grooves will change the detection point sensitivity). The results of the tests can be used to support a diagnosis of advanced HAVS (Stages 2 or 3), but because of the number of false-positive and false-negative results, the test data should not be used to override other data and the examining physician's judgment [Carlson et al. 1984; Sivayoganathan et al. 1982]. Data obtained using the improved test correlated significantly with the clinical staging of HAVS in workers with Stage 2 and above [Taylor et al. 1986]. For individual diagnostic purposes, however, the test lacked sufficient discriminatory power.

Haines and Chong [1987] reviewed the literature in which peripheral neurological tests were used to assess the acute and chronic effects of exposure to hand-arm vibration. The peripheral neurological tests demonstrated their usefulness in epidemiologic studies.

4. Arteriography

Ashe et al. [1962], Ashe and Williams [1964], and Takeuchi et al. [1986], utilizing finger biopsy material, observed extensive damage to the digital arterial walls with narrowing of the lumen in the fingers of workers with HAVS. These findings led to the concept that hand arteriography might be a useful tool for the diagnosis of HAVS [Wegelius 1972; Zweifler 1977; James and Galloway 1975; James et al. 1975; Takeuchi et al. 1986]. The procedure does, however, require an intraarterial injection of a dye and, therefore, is an invasive procedure.

The data reported by James and Galloway [1975] indicate that almost all of the workers who had symptoms of HAVS showed digital artery occlusion that was partial or nearly complete. Their control data were limited to three nonvibration-exposed individuals (members of the observation team) who showed little evidence of digital artery narrowing or occlusion.

Takeuchi et al. [1986] reported medial muscular hypertrophy and intima fibrosis in the digital arteries of vibrating tool users.

Okada et al. [1987a] observed thickening of the intima of finger arteries in workers who used vibrating tools. Inaba et al. [1988] reported a thickening of the intima in animals subjected to vibration. Intimal thickening appears to be part of the arterial pathological changes.

5. Grip Force

Hand and finger grip force can be easily measured with a strain gauge or a simple spring resistance dynamometer. Several types of measurements can be made. These include (1) maximum grip force, (2) fatigue curve while producing maximum grip force, and (3) fatigue curve during rhythmic contraction-relaxations. These measurements can be made both before and after a work period, or before and after exposure to vibration [Farkkila et al. 1982].

Because maximum grip force appears to be reduced, and strength fatigue is faster in workers who have used vibrating tools for several years (and may or may not have symptoms of HAVS), a simple grip strength measurement (hand dynamometer) can have some diagnostic usefulness. However, as pointed out earlier, normal standard values for grip strength and grip fatigue indicate a wide inter- and intra-individual variability, which make comparisons difficult to interpret.

6. Nerve Conduction

A decrease in motor and sensory peripheral nerve maximum conduction velocity in the median and ulnar nerves has been reported in workers with histories of occupational hand-vibration-exposure [Lukas 1982; Seppalainen 1972; Sakurai and Matoba 1986]. Maximum conduction velocity of the ulnar or median nerves can be determined by electrical stimulation of the nerves at a designated point and by recording the time for a motor response to occur. The time required for the nerve impulse to travel the distance between the two points can also be calculated [Seppalainen 1972]. In a group of vibration-exposed workers who had complaints and symptoms of HAVS, about 50% showed conduction velocity reduction [Lukas 1982]. Sakurai and Matoba [1986] also reported a decreased motor nerve conduction velocity (MCV) and sensory nerve conduction velocity (SCV) in workers who used vibrating tools. Chatterjee et al. (1978) found that in rock drillers, the SCV in the median nerve was reduced but the MCV was not. Latency, duration, and amplitude of the sensory action potential were also significantly changed in the rock drillers.

Brammer and Pyykko [1987] analyzed the electroneurographic data from 23 studies of workers who used hand-held vibrating tools. After control of the data for polyneuropathy and the effects of hard manual work, a neuropathy remained that involved mainly the sensory nerves in the hands. This sensory neuropathy could be distinguished from compression neuropathies (carpal tunnel syndrome) by measuring the nerve conduction velocity.

Araki et al. [1988] reported that the distribution of sensory nerve conduction velocities (median nerve) was altered and the magnitude of the sensory nerve conduction velocities was significantly slowed in chain saw operators.

Farkkila et al. [1988], in a neurological study of 186 forestry chain saw operators (average usage time 16,600 hr) and 31 nonvibration-exposed workers, found that the disturbance of

the ulnar and median MCV and distal latency (DL) did not correlate significantly with the history of HAVS or numbness of the hands. However, a significant correlation was reported between vibrotactile detection thresholds and MCV and DL of the median and ulnar nerves.

7. Sensory Acuity

Some of the common tests of finger tip sensory acuity may assist the physician in diagnosing HAVS. These include (1) cotton wool test (light touch), (2) hot and cold probes (temperature), (3) pin prick (pain), and (4) tuning fork (vibrotactile). A decrease in sensitivity may indicate peripheral neural changes. Taylor et al. [1986] reported that stage assessment of HAVS, based on medical examination and history of exposure, did not correlate well with tests of sensory loss, loss of pain, and temperature discrimination. Sensory acuity tests cannot be used as positive indicators of HAVS [Harada and Matsumoto 1982]. Ekenvall et al. [1986] reported that the temperature neutral zone was increased from about 5°C (9°F) in controls to 10°C (18°F) in 17 vibration-exposed workers with neurological symptoms of HAVS. The vibration threshold was also nearly doubled in the vibration-exposed group as compared with controls.

E. TREATMENT

Several recent studies reported on hospitalized patients with different stages of HAVS who received various types of therapeutic treatment. The effectiveness of the various treatments was analyzed and evaluated. Because multiple treatments were used in all the studies, direct evaluation of the effectiveness of any single treatment is not possible.

Matoba and Sakurai [1986] described their experiences in treating 500 male workers with HAVS over a 10-year period. The workers had used vibrating tools for an average of 10.5 years, and all exhibited mild to severe symptoms and signs of HAVS. All treatments were given in a hospital, with an average hospital stay of 105 days. Treatment consisted of physiobalneotherapy (water bath), alone or with one or more drugs, nerve blocking, and/or surgical therapy, along with education and training.

Recognizing that an evaluation of the relative effectiveness of all the various combinations of treatment is not possible, the researchers nevertheless selected a group of 60 inpatients matched for age and vibration exposure. Some of the patients received only physiobalneotherapy (group P), whereas the others received physiobalneotherapy plus vasodilating drugs (group D) over a 6-week period. Subjective and laboratory signs improved about 30% to 40% in group P and 60% to 80% in group D [Matoba and Sakurai 1986]. The authors concluded that physiobalneotherapy is the key treatment but that drug therapy, vasodilators, and calcium channel blockers can accelerate improvement in the circulatory, neural, and motor problems present in HAVS. Improvement was not as good in patients with severe HAVS as in those with lesser stages of involvement.

Bielski [1988] reported the benefits derived from balneological treatment of 824 chain saw operators who had peripheral vascular symptoms of HAVS (Stage not specified). Thermography and plethysmography tests indicated statistically relevant improvement in 91% of the patients treated with a brine bath at 34°C (93.2°F) for 20 minutes a day for 24 days. The improvement persisted for at least 6 months after treatment even though the workers were again using chain saws. The author suggested that workers who use chain saws should undergo a 2 to 3 week annual treatment with balneotherapy to reverse and prevent the progression of HAVS. The vibration level produced by the chain saws used was not reported.

Nasu [1986] reported that the use of defibrinogenating drugs in the treatment of HAVS patients provided both subjective and objective improvement. With treatment, statistically significant improvement was observed in the finger-skin temperature, the amplitude of the finger plethysmogram, and the nail compression test before and after cold provocation. The subjective feeling of warmth after treatment was reported by all but 5 of the 118 patients studied. The beneficial effects of the treatment did not appear to be permanent but "reaggravation ranged usually from several months to more than a year" after treatment was stopped. The author also reported (without substantiating data) that the addition of alpha1 blocker (bunazosin hydrochloride) had a better effect than the defibrinogenating drug alone.

The results obtained from treatment trials must be interpreted with caution because (1) with the present state of knowledge, the extent of spontaneous (without treatment) reversal of HAVS when vibration stimulus is withdrawn is unknown, (2) in many studies control populations given hospital treatment and therapy are not included, and (3) there is doubt about the accuracy of assessment of subjects in the absence of proven objective tests. Subjective improvement without objective tests is not acceptable.

F. REVERSIBILITY

The British and Canadian groups have emphasized prevention over treatment as the better approach to the control of HAVS [Brammer 1984; Taylor and Brammer 1982; Taylor 1982a, 1982b]. If HAVS has not progressed beyond the Taylor-Pelmear Stage 2, the signs and symptoms tend to disappear with time if no further exposure to vibration is permitted or if the exposure level (acceleration and time) is sufficiently reduced. For workers with Stages 3 and 4 vibration syndrome, no tested regimen of treatment has resulted in a significant reversal of HAVS signs and symptoms. Therapy is essentially palliative [Brammer 1984; Taylor and Brammer 1982].

Olsen and Nielsen [1988] reported data from a 5-year study of three groups of forestry workers examined in 1978 and again in 1983. Group A (n=13) had no subjective symptoms in 1978 and continued sawing until 1983; group B (n=12) had no symptoms in

1978 and stopped sawing; group C (n=12) had symptoms of HAVS in 1978 but did not stop using chain saws. FSBP was measured with a CPT in 1978 and again in 1983. In 1978, all groups had increased response to the cold provocation when compared to 20 nonvibration-exposed controls. From 1978 to 1983, the vasoconstriction response to the CPT increased in group A ($p<0.05$), was unchanged in group B ($p>0.10$), and improved in group C ($p<0.05$). Antivibration saws were used between 1978 and 1983. In group A, the use of antivibration saws did not prevent further increase in hyperactivity; the improvement of workers in group C (who had HAVS) may have been due to a shift from regular to antivibration saws in 1978. The data suggest that while the use of antivibration saws will not entirely prevent the development of HAVS, it may reduce the occurrence and progress of the disorder.

A 3-year followup study of 55 forestry workers with HAVS was conducted by Ekenvall and Carlsson [1987] to determine the effect of cessation of working with vibrating tools on subjective symptoms and FSBP during finger cooling. The group on first examination included 14 with Stage 1 Taylor-Pelmear symptoms, 25 with Stage 2, and 16 with Stages 3 and 4. Of the 15 workers who continued outdoor work with vibrating tools during the 3-year followup period, none showed any improvement of symptoms (8 showed no change and 7 showed increased subjective impairment). Of the 32 who did not use vibrating tools during the 3-year followup study, 8 showed improvement, 19 showed no change, and 5 showed increased impairment. The subjective improvement reported in this study could not be confirmed by CPT. On the other hand, this study showed an increased reactivity to cold in the impaired group. Thus the CPT appears to provide objective confirmation of the deterioration.

Other studies have indicated that the signs and symptoms of HAVS may be reduced or reversed in some chain saw operators, chippers, and grinders when the worker is no longer exposed to vibration [Riddle and Taylor 1982; Hursh 1982].