Hand-Arm Vibration Syndrome

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The hand-arm vibration syndrome affects workers who perform tasks that generate vibration. Raynaud's phenomenon and sensory impairment of the fingers are the predominant effects. A history of hand-arm vibration (HAV) exposure in a patient with these symptoms should alert the physician to the diagnosis. Referral to a special clinic or hospital department for multiple clinical tests is required to confirm the diagnosis and, using the Stockholm classification, to grade the severity in each hand. The assessment permits the patient to be monitored either for progression of or recovery from the syndrome. Avoidance of further vibration exposure is recommended, together with the prescription of a slow-release calcium channel blocker to improve peripheral circulation.

Hand-arm vibration syndrome should be distinguished from carpal tunnel syndrome (CTS), which may have similar symptomatology but requires different treatments. Surgery is contraindicated in the former and should be the last resort for carpal tunnel syndrome in a worker requiring good grip-strength in future employment.

Key words. Vibration; carpal tunnel syndrome; arm injuries; hand injuries; treatment outcome; primary prevention; work. (*J Fam Pract 1994; 38:180-185*)

Adverse health effects can result from contact with almost any vibrating source if the vibration is sufficiently intense and within the frequency range of 4 to 5000 Hz for a significant period. The exposure time necessary may range from 1 month to 30 years, depending on the intensity of the vibration source, the transmissibility and absorption of vibration into the hand, and individual susceptibility. Workers with large, warm hands seem to be less at risk. The most common tools causing hand-arm vibration syndrome (HAVS) are pneumatic tools, such as grinders, drills, fettling tools, jackhammers, riveting guns, impact wrenches, and chainsaws. In high-risk work situations, the incidence and prevalence of HAVS can be as high as 90%, or possibly higher.[1] The symptoms are more commonly reported in cool climates and during winter rather than summer because of cold stress on the circulatory system. In some work situations, an increase in work intensity, introduction of new tools, or extended work hours may cause a HAVS epidemic. Exposure to domestic sources of vibration, such as lawn mowers, hedge cutters, chainsaws, house tools, or motorcycles, is usually too short to produce ill effects, but may enhance the risk from a work hazard.

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The Clinical Picture

Hand-arm vibration syndrome[2] is a disease with the following separate peripheral components: *circulatory disturbances*: cold-induced vasospasm with local finger blanching "white finger"; *sensory and motor disturbances*: numbness, loss of finger coordination and dexterity, clumsiness, and inability to perform intricate tasks; and *musculoskeletal disturbances*: muscle, bone, and joint disorders.

Vasospasm, also known as Raynaud's phenomenon, is precipitated by exposure to cold or damp conditions, and sometimes by vibration exposure itself. The period between first exposure to HAV and the onset of fingertip blanching is termed the *latent interval*. A short latent interval is indicative of a high-risk work situation, unless the worker is highly susceptible. Blanching is restricted initially to the tip of one or more fingers (Table 1) but

Table 1. The Stockholm Workshop Scale for the Classification of Cold-Induced Raynaud's Phenomenon in the Hand-Arm Vibration Syndrome

Stage	Grade	Description
0		No attacks
1	Mild	Occasional attacks affecting only the tips of one or more fingers
2	Moderate	Occasional attacks affecting the distal and middle fingers (rarely also proximal) phalanges of one or more fingers
3	Severe	Frequent attacks affecting all phalanges of most fingers
4	Very severe	As in stage 3, with trophic skin changes in the fingertips

NOTE: *Staging is determined separately for each hand. In the evaluation, the grade of the disorder is indicated by the stages of both hands and the number of affected fingers on each hand; for example, "2L(2)/1R(1)."*

progresses to the base of the finger as the vibration exposure time increases. Thumbs are usually the last to be affected.

Finger blanching is accompanied by numbness. As the circulation is restored to the digits, hyperemia, tingling, and pain usually occur. Tingling and paresthesia usually precede the onset of blanching in many subjects. These sensory symptoms and signs may be the only complaint voiced by some patients. Recognition of these as a distinct entity led to the revision of the Taylor-Pelmear classification for assessment of HAVS devised in 1968.[3] It was replaced in 1985 by the Stockholm classification,[4,5] in which history, supported by the results of clinical tests, is used to categorize by stage the severity of the vascular and sensorineural symptoms and signs, separately for both hands (Tables 1 and 2). This staging has proved useful for compensation assessment and for monitoring deterioration and recovery.

In advanced cases, peripheral circulation becomes sluggish, giving a cyanotic tinge to the skin of the digits, whereas in very severe cases, trophic skin changes (gangrene) will occur at the fingertips. The toes may be affected if directly subjected to vibration from a local source, such as vibrating platforms, or they may be affected by reflex spasm in subjects with severe hand symptoms. Reflex sympathetic vasoconstriction also may account for increased severity of noise-induced hearing loss in HAVS subjects.[6,7]

Table 2. The Stockholm Workshop Scale for the Classification of Sensorineural Effects of the Hand-Arm Vibration Syndrome

Stages	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 or manipulative dexterity or both

NOTE: *The sensorineural stage is to be established for each hand.*

In addition to tactile, vibrotactile, and thermal threshold impairment, which may vary among subjects, impairment of grip strength is a common symptom in workers exposed for a long time.[8,9] Discomfort and pain in the upper limbs is also a common complaint. Bone cysts and vacuoles, although often reported,[10,11] are more likely to be associated with forceful manual activity of the hands in work processes requiring heavy tool manipulation.

Carpal tunnel syndrome (CTS), an entrapment neuropathy affecting the median nerve at the wrist, is often associated with HAVS.[12–14] Usually it is caused by ergonomic stress factors, including the constant, repetitive nature of the work, force, or mechanical stresses such as torque and posture. The history and symptoms of HAVS, HAVS with CTS, and CTS alone are compared in Table 3. The pathophysiology, etiology, clinical picture, and treatment of CTS have been well reviewed by Carragee and Hentz,[15] who conclude that median nerve entrapment is likely to involve both mechanical and ischemic factors.

When vibration is the primary cause of median nerve neuropathy, the edematous reaction in the adjacent tissues and the nerve sheath compresses the central axon.[16] In two thirds of the cases, the median nerve is affected along with the ulnar. Sometimes the ulnar nerve is affected alone.[17] When the median nerve myelinated fibers are involved at the wrist level, HAVS can be confused with CTS nerve entrapment because the symptoms and signs are similar.

Smoking has been shown to increase the risk of HAVS.[18,19]

Pathophysiology

The pathophysiology of HAVS was recently well reviewed by Gemne.[20] Although the basic mechanism is not yet fully understood, mechanical stimulus is thought to cause specific anatomical changes in the digital vessels (ie, vessel wall hypertrophy and endothelial cell damage). In the initial stages, there is extrusion of fluid into the tissues. Combined with the subsequent spasmodic ischemia from cold-induced vasospasm, this edema damages the mechanoreceptor nerve endings and nonmedul-

Table 3. History and Symptoms of Hand-Arm Vibration Syndrome and Carpal Tunnel Syndrome

History/Symptoms	HAVS Only	CTS Only	HAVS and CTS
Exposure to vibration	Yes	No	Yes
Exposure to repetitive strain	No	Yes	Yes
Numbness in fingers	Yes	Yes	Yes
Tingling			
Median	Yes	Yes	Yes
Ulnar	Yes	Rarely	Yes
Sleep disturbance	Unusual	Common	Common
Muscle cramps	Common	Unusual	Common
Aches and pains in arms	Common	Unusual	Common
Reduced grip strength	Common	Unusual	Common
Raynaud's phenomenon	Yes*	No	Yes*

**May not be apparent to patient in early stage.*
HAVS denotes hand-arm vibration syndrome; CTS, carpal tunnel syndrome.

lated fibers. Subsequently, a demyelinating neuropathy of the peripheral nerve trunks develops.

The vascular response to cold is complex because, in addition to the diversity of receptor systems (adrenergic, cholinergic, purinergic, and serotonergic), there are several subtypes of specific receptors.[21] Although the differential distribution and functional significance of the various receptor types is largely unknown, it is probable that the cold-induced pathological closure (noradrenaline-triggered vasoconstriction) of the digital arteries and end vessels is mainly mediated by α_1 adrenoceptors in the wall of the arterioles and veins. It has been demonstrated that the α_1 receptors are more sensitive to the cold stimulus. It is postulated that HAV results in selective damage of vessel wall α_1 receptors, which relax cold-stimulated vessels; hence, the cold stimulus is more effective.[22] While arterial spasm is necessary to stop blood flow, vasospasm in the skin arterioles is essential to produce blanching.

Cold, as well as vessel wall injury, causes platelet aggregation.[23] The subsequent release of serotonin (5-hydroxytryptamine [5-HT]) promotes further release of 5-HT from the platelets, and the increased concentration stimulates smooth muscle to contract. Besides promoting contraction, serotonin also may contribute to vasodilation by inducing the release of endothelium-derived relaxing factor (EDRF) and prostacyclin from the endothelial cells. Acetylcholine and its agonist metacholine, acting through the muscarine receptors, also release EDRF, whereas nitric oxide and its agonists, nitroprusside and nitroglycerine, release prostacyclin. Besides inhibiting platelet aggregation, the release of prostacyclin and EDRF stimulates the production of cyclic adenosine monophosphate (cAMP) and cyclic guanosine monophosphate (cGMP) in the smooth muscle cell. The latter substances inhibit calcium utilization by the smooth muscle cells to prevent contraction. A delicate balance between smooth-muscle contraction and relaxation is produced by simultaneous interaction of these mechanisms.[20]

Diagnosis

The diagnosis of HAVS is based initially on a history of vibration exposure and the exclusion of other causes of Raynaud's phenomenon, such as primary Raynaud's phenomenon (Raynaud's disease or constitutional white finger) and secondary Raynaud's phenomenon from local trauma to the digital vessels, thoracic outlet syndrome, drugs, peripheral vascular disease, and collagen diseases, including scleroderma.

The diagnosis of HAVS is confirmed and the severity assessed and categorized by stage based on laboratory tests.[17,24–26] A clinical assessment with laboratory tests is always necessary because, in our experience, when the diagnosis is based on history alone, there is less than a 40% agreement in severity for both vascular and sensorineural effects. The stage of severity will help determine the need for treatment and degree of impairment for compensation when appropriate.

The office-based vascular tests for HAVS include Adson's,[27] which detects thoracic outlet obstruction; Allen's,[28] which checks circulation in the superficial and

deep palmer arches; and immersion of the hands in cold water to stimulate blanching. The latter often is unsuccessful because blanching usually requires central body cooling in addition to hand cooling. Feasible sensory tests available to family physicians for detection of diminished skin sensitivity include pin prick, cotton wool, and von Frey or Semmes-Weinstein monofilament hairs, together with calipers for two-point discrimination.

More sophisticated tests are required to confirm the diagnosis and grade the severity. Vascular tests should include Doppler and Duplex studies, which check the patency and blood pressure ratios in the peripheral vessels; plethysmography to evaluate the pulse waves before and after cold stress; finger systolic pressure measurement to compare the blood pressure of an affected digit before and after cold stress with the blood pressure of the thumb, which is not usually affected; and cold water provocation tests, which involve immersion of the digits in water for 2 to 7 minutes with recording of skin temperature, to detect reactive hyperemia during immersion and delay in recovery afterward.

Of these vascular tests, the most valuable for diagnostic purposes are plethysmography, which has high sensitivity and specificity, and cold-water provocation, which is the most sensitive and specific for objectively diagnosing HAVS and grading its severity. However, the latter is available only in centers where HAVS cases are regularly assessed. Normal and very mild cases of HAVS will show a hyperemic reaction to cold-water immersion followed by rapid recovery. In moderate to severe cases, there is no hyperemic reaction, and recovery is moderately delayed or prolonged.

Sensorineural tests should include depth sense and two-point discrimination using von Frey or Semmes-Weinstein monofilament hairs, calipers, or plastic blocks with split levels and widening grooves,[29] and one or more of the following tests: fingertip vibration threshold measurement (8 to 500 Hz with vibrometer instrumentation[30]), thermal hot/cold perception using Minnesota thermal discs[31] or instrumentation,[32] and current perception threshold (detection of a 0 to 10 mA current at 5, 250, and 200 Hz[33]). Although subjective, these tests will detect damage to the mechanoreceptors in the skin and the distal digital branches of the nerve fibers.

Sensitivity levels are compared with normative reference data. Damage in more advanced cases also occurs in the medullated nerve fibers. Hence, objective nerve conduction tests,[34] readily available in all hospitals, should always be undertaken to confirm presence and severity of the neuropathy. In HAVS cases, as opposed to CTS, the ulnar nerves are often affected in addition to the median nerves, and sometimes alone. Based on sensorineural (SN) test results, subjects with mechanoreceptor or distal digital branch nerve fiber impairment should be graded as stage 2 SN, and when there is nerve conduction impairment, as stage 3 SN.[14]

Treatment and Management

To reduce the frequency of blanching attacks, central body temperature should be maintained and cold exposure must be avoided. Mitts rather than gloves should be worn, and smoking should be avoided because of the adverse vasoconstrictor action of nicotine on the digital arterial system.

To reverse the disorder and achieve recovery, further vibration exposure should be avoided. If avoidance of HAV is impossible, a modified work routine, eg, alternative work or frequent work breaks, should be introduced to reduce vibration exposure and slow the progression of this condition.

Recent advances in drug therapy have focused on three areas: (1) use of calcium channel antagonists to produce peripheral vasodilation; (2) use of drugs such as pentoxifylline to improve red blood cell flexibility and promote platelet deaggregation in combination with the above; and (3) drugs such as isosorbide dinitrate with prostaglandin E_1, to reduce platelet deposition and increase platelet survival. Nifedipine (20 to 40 mg daily) or felodipine (5 to 10 mg daily) are the preferred calcium channel antagonists because they are slow-release products and they have fewer side effects. Treatment should be continued indefinitely unless recovery occurs.

The necessity for drug therapy increases with the severity of the symptoms and the age of the subject. The results are encouraging for resolution of vascular symptoms, particularly if vibration exposure is avoided. Unfortunately, drug intolerance to the earlier calcium channel antagonists has caused some patients to abandon their use prematurely. When this occurs, newer products should be tried. Recovery from the vascular effects of HAVS on cessation of vibration exposure is not unusual in workers under the age of 40 who have not progressed beyond stage 2. Although recovery from the sensoineural effects following cessation of HAV exposure has been reported,[35] it is less common, and in severe cases, unlikely.

When patients are thought to be suffering from HAVS, their employers should be advised initially to assess the work situation and secondly to introduce preventive procedures. The employer and the physician responsible for treatment then should initiate a compensation claim following state-approved procedures to ensure that the patient is fully investigated, medical impairment

is properly assessed,[35] and compensation is provided as appropriate.

Prevention and Control

Because the development of HAVS is progressive, ie, related to dose (time plus intensity), effective control measures should first be directed to a reduction in the intensity of vibration at the source,[36] followed by the use of isolation and damping techniques to reduce transmission.

Only antivibration tools conforming to recommended safety limits should be used. They also should be ergonomically designed to place minimum strain on the user; have a high power-to-weight ratio; have low torque with a cutoff rather than slip-clutch mechanism; and the handles should have a nonslip surface to reduce the need for excessive grip force.

Wearing gloves at all times is recommended to maintain hand temperature, reduce the risk of cuts and abrasions, and decrease the transmission of high-frequency vibration. Leather gloves have proved to be very effective for this purpose. Newer materials, eg, Sorbothane and Viscolax now being used as inserts, should be evaluated carefully. The selection of material is important because vibration transmission can increase as a result of resonance depending on the material's vibration characteristics.[36]

Administrative controls should be introduced in high-risk situations to reduce exposure time by means of job rotation or rest periods. Alternative vibration-free work should be made available to workers who have stage 2 or 3 HAVS as assessed by means of the Stockholm classification.

All workers should be advised of a potential vibration hazard and receive training on the need to service their tools regularly; to grip the tools as lightly as possible within the bounds of safety; to use protective clothing; to have periodic medical checkups; and to report all signs and symptoms of HAVS as they develop.[37]

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