Editorial
Vibration induced injury

Injuries caused by vibrating hand held tools have been increasingly described in association with technical development but we do not yet know the mechanisms by which vibration causes many of the symptoms reported. Despite the many norms and standards proposed, nobody knows which are the medically—not only subjectively—hazardous qualities of vibration (amplitude, acceleration, frequency, or impulsiveness, for example). The diagnosis of vibration injuries is controversial also because of the lack of objective tests to demonstrate the pathology behind the symptoms. Nevertheless, vibration induced white finger—with or without associated symptoms—has long been compensated as an occupational disease in many countries, and was recently prescribed in the United Kingdom.1

The intermittent blanching of the fingers (Raynaud's phenomenon) is the most prominent symptom to result from occupational exposure to vibration,2 and it is still the most frequent sign of the vibration syndrome. Early reports of the prevalence of vibration induced white finger (VWF) varied with different tools and working habits—for instance, in miners, rock drillers, and pneumatic hammer workers the prevalence was 30–73% and in chain saw operators 40–60%. The prevalence of VWF is higher when a rock drill is used with the arms in an upward position compared with using it with the arms pointing down. In many countries there have been improvements in vibration damping techniques and some methodological improvements (the tools are not hung on the hands now), and these prevalences are largely of historical interest. In Canada, the United States, and the United Kingdom, however, high prevalences of white finger have recently been reported,3 and sudden and unexpected new outbreaks of VWF have been described.4 Despite this, the prevalence and severity of Raynaud's phenomenon have decreased remarkably in many occupations.2 The vibration induced Raynaud's phenomenon has been shown to be reversible, except perhaps for the advanced cases (Taylor-Pelmear classification stage 4). An attack of white finger is provoked by cold, mediated as a central sympathetic reflex, and facilitated by local disturbances in the control of finger blood flow as described by Lewis.

The work of Marshall and his collaborators indicated that occupational exposure to vibration may cause peripheral neuropathy.5 Tingling and numbness in the hands are also associated with exposure to vibration and usually interpreted as the first stage symptoms in developing Raynaud's phenomenon.6 Numbness occurs among those exposed to vibration to a significantly greater extent (80%) than in non-exposed referents (30%).2 The paresthesiae and numbness among vibration exposed workers have also been associated with carpal tunnel syndrome.7 Nevertheless, numbness and neuropathic findings have not been related to the severity of VWF, so the numbness may not be vascular in origin. In a field study among forestry workers the prevalence of generalised polyneuropathy was only 4%. In a clinical examination of forestry workers neuropathic findings were seen in the hands in 7–5% of subjects. Electromyographic signs of previous or present median nerve entrapment in the carpal tunnel were found in 29 (36%) of the 80 lumberjacks exposed. Thus exposure to vibration does not cause polyneuropathy,8 and at least some of the local neuropathic findings are due to the entrapment of the median or ulnar nerves in the hand and arm. Vibration may cause the neurological signs by compression due to tissue swelling and excess collagen formation both intraneurally and extra-neurally.

Workers exposed to vibration often complain of decreased muscle force. Workers who take a firm grip around the handle of a tool are more prone to develop VWF and vibration work causes a decrease in the hand muscle force in excess of the age dependent decrease.9 An increase in the sarcoplasmic enzymes and hydroxyproline excretion in the urine has also been reported after exposure to vibration, indicating that some damage to the musculoskeletal system is induced by vibration. These findings suggest that direct mechanical trauma or a nerve lesion may cause the decreased muscle force.

The cysts and decalcification described in the bones of the wrist have no association with exposure to vibration.

Various non-specific symptoms referable to the central nervous system have been reported, including excessive sweating of the hands, headache, vertigo, insomnia, anxiety, and impotence. The effects of vibration are certain to be transmitted through the higher autonomic centres, but so far there is no convincing, hard data for permanent damage to the
hypothalamus or other autonomic centres. The symptoms relating to secondary stress effects may be shown in other workers who have no exposure to vibration. In our unpublished studies of 88 forestry workers with a mean of 16,500 hours occupational exposure to vibration no clinically significant signs of autonomic neuropathy were found in peak to peak variation measurements of ECG during orthostatic, controlled breathing and Valsalva tests. Neither headache nor vertigo were more common among vibration exposed than among non-exposed referents.

Histological studies have shown thickening of the muscular layer of the arteries with hypertrophy of the muscle cells without intimal layer fibrosis. There is also a demyelinating neuropathy in the peripheral nerves and a loss of nerve fibres with an increase in the number of the Schwann cells with much collagen formation. Thickening of the perineurium and epineurium due to an increase of collagen was often seen.

Attempts to develop objective, diagnostic tests have met with little success. In the cold provocation test Raynaud’s phenomenon can often not be induced, and the most promising test so far is the measurement of finger systolic blood pressure. Electromyography may show non-specific findings but cannot differentiate between vibration syndrome and other diseases. Muscle force measurements are not useful in clinical work because of the large variety in normal hand muscle force. There is no specific laboratory test for vibration injury, and biopsy specimens from hand tissues show changes that could also be induced by agents other than vibration. Thus the diagnosis of the vibration syndrome is based on the patient’s history of symptoms and the exclusion of causes other than vibration as possible aetiological factors.

The most important treatment is to diagnose the injury and plan protective changes at work. It seems that both Raynaud’s phenomenon and carpal tunnel syndrome are reversible. If medical treatment is considered necessary for VWF ketanserine and PGE,-infusion have proved useful. Calcium channel blockers such as nifedipine, or peripheral alpha-blockers such as prazosine are also worth trying.

Whole body vibration has been reported to cause different symptoms without any known pathology. Vibration resonances in the internal organs have been measured, but so far no disease has been linked to whole body vibration.

Vibration induces injury to different tissues by different mechanisms. VWF develops by its own mechanism, perhaps a combination of local changes and a central vasoconstrictive reflex, and entrapment neuropathies may be due to tissue swelling caused by vibration. This would explain the association of the carpal tunnel syndrome with exposure to vibration. Muscle weakness may be due to direct mechanical damage. Thus all the symptoms have a separate cause which makes it so difficult to diagnose and classify the cases. Nowadays, it is best to study all the symptoms separately and combine neurological and medical knowledge when diagnosing the vibration syndrome. Also the classification scales for vascular and neurological symptoms should be separated, since they are probably not at all linked to each other. There is also the so far overlooked possibility of a vibration induced carpal tunnel syndrome with muscle weakness without VWF, which might also be included in the vibration syndrome complex. At present autonomic and systemic symptoms should not be considered to result from vibration injuries.

M FÄRKKILÄ

Department of Neurology, University Hospital of Helsinki, Helsinki, Finland.

References