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## Alcohol (Ethanol) Related Neuropathy

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Article Last Updated: Jul 10, 2006

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**Synonyms and related keywords:** alcoholic neuritis, neuritic beriberi, neuropathic beriberi, alcohol-related neuropathy, ethanol neuropathy, alcohol neuropathy, alcoholic peripheral neuropathy, ethanol-related neuropathy, peripheral nerve disease

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### Background

The clinical symptoms of alcoholic peripheral neuropathy were described more than 200 years ago. The descriptions by Lettsom (1787) and Jackson (1822) have led to the recognition and association of peripheral nerve disease with excessive ethanol use. Several terms connote alcohol neuropathy, including neuritic beriberi, neuropathic beriberi, and alcoholic neuritis. In patients with alcoholic neuropathy, nutritional deficiency goes hand in hand with alcohol abuse.

The similarity between beriberi and alcoholic neuropathy had long been noted, but Shattuck in 1928 was the first to seriously discuss the relationship. He suggested that "polyneuritis of chronic alcoholism" was caused chiefly by failure to take or assimilate food containing a sufficient quantity of vitamin B<sub>12</sub> and might properly be regarded as true beriberi. However, this theory may be only partly true.

### Pathophysiology

The precise pathogenesis of alcohol neuropathy remains unclear. Separating ethanol use from nutritional and vitamin deficiencies, especially thiamine, has always been difficult and a source of long-standing debate. Nutritional deficiency (frequently associated with alcohol neuropathy) and/or the direct toxic effect of alcohol or both have been implicated and studied. In Wernicke-Korsakoff syndrome, a clear association between reduction of thiamine levels or thiamine-mediated enzyme activity (transketolase) has been established, though this has not been conclusively established in the case of peripheral neuropathy.

In their comparison of alcoholics and nonalcoholic control subjects, Behse and Buchthal concluded that nutritional deficiencies alone did not produce the neuropathy.

Monforte et al concluded that alcohol appears to be toxic to autonomic and peripheral nerves in a dose-dependent manner, on the basis of heart rate, blood pressure, and electrophysiologic examination.

In a study of macaque monkeys, Hallet et al failed to produce clinical and electrophysiologic signs of neuropathy in monkeys that were given a certain amount of alcohol for 3-5 years.

Studies in rats also failed to demonstrate a direct toxic effect of alcohol on the peripheral nerves.

Most studies of peripheral neuropathy in humans and animals implicate nutritional deficiency as an etiology as opposed to the direct toxic effect of alcohol.

## Frequency

### International

Depending on criteria and patient selection, incidence of peripheral neuropathy ranging from 10-50% has been reported. These studies included alcoholics hospitalized for other reasons or for detoxification. Neuropathy is more prevalent in frequent, heavy, and continuous drinkers compared to more episodic drinkers (Monforte, 1995). When electrodiagnostic criteria are added, neuropathy detection increases to 25-90% (Vittadini, 2001).

## Mortality/Morbidity

Johnson and Robinson studied the mortality rate of alcoholics with autonomic neuropathy.

- Their findings suggested that evidence of vagal neuropathy in long-term alcoholics is associated with a significantly higher mortality rate than in the general population (a reported 88% survival rate at 7 years in alcoholics with autonomic neuropathy as compared to 94% in the general population).
- Deaths due to cardiovascular disease are a major factor.
- Many deaths were attributed to strokes, since heavy alcohol consumption is a significant risk factor for stroke.

## Sex

- A high incidence of alcoholic polyneuropathy has been observed in women.

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## History

Clinical manifestations of alcoholic neuropathy can be summarized as slowly progressive (over months) abnormalities in sensory, motor, autonomic, and gait function. Patients might ignore early symptoms, and seek help only when significant complications develop. Symptoms are often indistinguishable from other forms of sensory motor axonal neuropathy.

- Sensory symptoms include early numbness of the soles, followed by dysesthesias of feet and legs, especially at night. "Pins and needles" sensation, which is reported commonly, progresses to severe pain that is described as burning or lancinating. Symptoms start typically distally, to progress slowly to involve proximally (dying-back neuropathy). When symptoms extend to involve above the ankle level, the fingertips often get similarly involved, giving rise to the well-known stocking and glove pattern of sensory involvement. Paresthesia might become unpleasant, even painful.
- Motor manifestations include distal weakness and muscle wasting.

- When proprioception becomes involved, sensory ataxia will occur giving rise to gait difficulty, independent of alcoholic cerebellar degeneration.
- Autonomic disturbances are seen less commonly than other neuropathic conditions (eg, diabetes).
  - Dysphagia and dysphonia are prominent secondary to degeneration of the vagus nerve. Other parasympathetic abnormalities include depressed reflex heart rate responses, abnormal pupillary function, sexual impotence, and sleep apnea.
  - Sympathetic dysfunction is rare but if present can produce orthostatic hypotension and hypothermia.
- Frequent falls and accidents are common. These are secondary to gait unsteadiness and ataxia that are caused by cerebellar degeneration, sensory ataxia, or distal weakness.

## Physical

Examination shows distal sensory loss in lower extremities. In severe cases, the hands may be involved. In addition to distal atrophy and weakness, deep tendon reflexes usually are decreased or absent. Stasis dermatitis, glossiness, and thinness of skin of the lower legs are common findings. Hyperesthesia and hyperalgesia may be seen along with hyperpathia. Excessive sweating of the soles and dorsal aspects of the feet and of the palms and fingers is a common manifestation of alcoholic neuropathy and is indicative of involvement of the peripheral (postganglionic) sympathetic nerve fibers. Occurrence of trophic ulcers is rare.

## Causes

- Variants
  - Rare cases of acute or subacute alcoholic peripheral neuropathy have been described. They mimic Guillain-Barré syndrome, although biopsy and electrodiagnostic studies had revealed an axonal neuropathy, with normal CSF parameters. A causal but an unproven association with ethanol is present.
  - Pressure palsies: Alcoholics with generalized axonal peripheral neuropathy are prone for pressure palsies at multiple sites. Associated nutritional deficiency and weight loss might potentiate the same. Neurapraxia is more common than axonotmesis, and recovery is often the rule, although elderly patients do poorly.

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## Other Problems to be Considered

Amyloidosis  
 Cryoglobulinemia  
 Selected nutritional deficiencies  
 Porphyrin  
 Lumbosacral disk syndromes  
 Neuromuscular diseases  
 Paraneoplastic neuropathy

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## Lab Studies

- The diagnosis is based on accurate history of prolonged and excessive alcohol intake, clinical signs and symptoms, and electrophysiologic testing. Behse and Buchtal suggested that a minimum of 100 mL of ethyl alcohol (3 L of beer or 300 mL of spirits) per day for 3 years will precipitate the neuropathy.

## Other Tests

- Electrophysiologic findings primarily reveal evidence of primary axonal sensory motor polyneuropathy.
  - Sensory conduction studies may be abnormal even before the advent of clinical symptoms.
    - Sural nerve sensory action potentials (SNAP) are reduced slightly to moderately in conduction velocity and SNAP amplitudes also are reduced.
    - As the condition worsens, the sensory potentials may become unobtainable. The median, radial, and ulnar nerves show the same response as the disease progresses.
  - Motor conduction studies of the lower extremities (tibial and peroneal nerves) may reveal a slight reduction in conduction velocity (not to exceed 70-80% of the lower limit of normal), with diminution of the compound muscle action potential (CMAP) amplitude with a slight prolongation in distal latency. The upper extremity nerves follow the same pattern as time progresses.
  - The tibial H reflex latency is prolonged and becomes unobtainable if the condition continues to progress. The F waves are obtained more easily but reveal slight to moderate prolongation of latency.
- Needle electromyography (EMG) examination of the distal muscles of the lower extremities shows active denervation as well as chronic changes in the form of re-innervation patterns.
  - Spontaneous activity (positive sharp waves and fibrillation) is seen in the tibialis anterior and gastrocnemius.
  - The motor unit action potentials are reduced in recruitment pattern, with high-amplitude, long-duration, and polyphasic motor units.

- Avaria Mde et al (2004) have demonstrated that prenatal alcohol exposure is associated with abnormalities in nerve electrical properties and that the pattern is different from that seen in adults, showing conduction slowing and decrease in proximal and distal amplitude. Inference can be made by demonstrating other abnormalities of alcohol abuse, particularly abnormal liver function test results and red cell macrocytosis. Thiamine levels are not consistently reduced, but the thiamine-mediated enzyme transketolase estimation is often abnormal.
- Cerebrospinal fluid (CSF) is typically normal or might show a mildly elevated total protein level.
- Patients have an increased risk of compression neuropathy, and electrodiagnostic findings can be complicated by superimposed mononeuropathies that are present. Recent methods of demonstrating small-diameter fiber neuropathy, such as quantitative sensory testing and intraepidermal nerve fiber density, have been applied but need to be applied in large scale.
- Sural nerve biopsy often shows evidence of generalized distal axonal loss affecting both large and small fibers but without distinctive pathologic features.
- Autonomic testing of parasympathetic and sympathetic reflexes is often abnormal (25% in one study), including analysis of heart rate variability, Valsalva maneuver, handgrip, tilt table, and standing maneuvers (Agelink, 1998). The pattern of abnormalities often resembles the changes in diabetes and other causes of autonomic failure.

## Histologic Findings

Pathologic findings of the peripheral nerve in alcoholic neuropathy generally are agreed to consist of axonal degeneration with secondary segmental demyelination.

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## Medical Care

Treatment is directed toward stopping further damage to the peripheral nerves and returning to normal functioning. These can be achieved by alcohol abstinence, a nutritionally balanced diet supplemented by all B vitamins, and rehabilitation. However, in the setting of ongoing ethanol use, vitamin supplementation alone has not been convincingly shown to be sufficient for improvement in most patients.

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## Prognosis

- The prognosis of alcoholic neuropathy generally is good, as reported by Hillbom and Wennberg in their series of 10 patients.
  - Provided that alcohol intake is discontinued and other causes of neuropathy (eg, malignancy, diabetes, nerve trauma) are carefully excluded, clinical and electrophysiologic examinations returned to normal or near normal. This is independent of age.
  - Prognosis is generally better in patients who are healthy and well nourished. Recovery is presumed to be due to regeneration and collateral sprouting of damaged axons.
- Studies have shown that patients with mild-to-moderate neuropathy can significantly improve, but the improvement is usually incomplete in those with severe findings.

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