**CORRESPONDENCE**

**Predictive value of nerve conduction studies**

**EDITOR,—**We read with interest the study of Werner et al (1) on the value of nerve conduction studies (NCS) for identifying future carpal tunnel syndrome (CTS) and we think that it deserves comment. Schottland et al (2) and Bingham et al (3) have shown that in a pool of job applicants, screening with NCS was able to identify the existing median nerve abnormalities among a considerable portion of asymptomatic people. These findings were independent of NCS technique or critical value used. The work of Werner et al also identified the group of asymptomatic workers; these findings were independent of critical value used.1 Werner et al studied 108 initially asymptomatic workers with a specific but insensitive measurement of median nerve latency (>14 cm sensory median-ulnar difference). After a mean follow up of 17 months, they found that seven cases and six controls (n=13 workers in total) developed specific, recurring, or persistent symptoms as assessed by them. There was no significant difference in the development of symptoms between the cases and controls, suggesting that pre-existing nerve abnormalities do not predict the development of CTS. As discussed by the authors, the few subjects and positive outcomes (n=13) limit the statistical power of this study.

Werner and his co-investigators have contributed to the case of CTS. They have discussed that the workers who had found that they had used a more sensitive NCS measurement such as the 8 cm sensory latency or the maximum latency difference,1 or if they had used an objectively confirmed case of CTS.

In their discussion, the authors state that "to date, there are no studies which define or characterise the natural history of median nerve function and hand symptoms among active workers." Over the past 10 years, we have published a series of articles that have considered this subject. One conclusion of our published five year and as yet unpublished 11 year follow up studies of active industrial workers is that NCS are the most reliable predictor of future persistent hand or wrist symptoms and CTS.

People who develop CNS abnormalities do not inevitably develop characteristic hand or wrist symptoms and clinical CTS, but we have found that asymptomatic workers with NCS abnormalities are much more likely to develop CTS than asymptomatic workers without NCS abnormalities. For a dichotomous condition as assessed by Werner et al (initial NCS normal v abnormal), we found an odds ratio of 4.3 (OR (361+15)/(70x18); P = 0.000) for 464 initially asymptomatic hands after 11 years. Interestingly, for a comparison of continuous variables (direct linear correlation between probability of future de novo CTS and initial maximum latency difference value), we found a highly significant direct, linear relation (R=0.275, P=0.000) and an odds ratio of 20.1 (OR (129x17)/(3x15); P = 0.000) comparing hands with maximum latency difference of >0.52 with <0.28 ms.

We encourage investigators to expand their studies to include more subjects, more sensitive NCS techniques, an objective case definition of CTS, and a longer follow up period. The findings from such more comprehensive studies may be helpful in helping to determine whether there is a role for electrodiagnostic screening tests in the workplace.

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6 Nathan PA, Meadows KD, Doyle LS. Relationship of age and sex to sensory conduction of the median nerve at the carpal tunnel and association of slowed conduction with symptoms. Muscle Nerve 1988;11:1149–53.


Author’s reply—We appreciate the comments made by Nathan et al regarding our recent article on the use of nerve conduction to predict future symptoms of carpal tunnel syndrome (CTS). Their group has done similar work but with some distinct differences. Their study population had a very low average participation rate (26% compared with 81% in our study) and is subject to potential selection bias. The main focus of their longitudinal studies was to evaluate the predictive value of abnormal median nerve conduction in determining future signs and symptoms of CTS regardless of initial symptoms. Considering their entire population of workers, an abnormal median nerve conduction study was predictive of symptoms consistent with CTS five years later. Many of the workers with an abnormal median nerve conduction were diagnosed as having CTS in the first evaluation (41%) and not surprisingly still had symptoms five years later. This is very different from our study of asymptomatic workers with an abnormal median nerve conduction compared with matched asymptomatic workers with normal median nerve conduction. They briefly considered the issue of workers with abnormal median nerve conduction who initially were not thought to meet the clinical definition of CTS but who were found to be classified as having clinical CTS five years later. These workers were not necessarily asymptomatic; they could have had hand or finger symptoms but did not initially meet their clinical definition for CTS.

They reported that 10% (10/114) of these workers went on to develop signs or symptoms consistent with their clinical diagnosis of CTS. This is almost identical with the incidence we found in our study. Unfortunately, they did not report or evaluate an age or sex matched control group of workers with normal median nerve conduction for comparison. In our matched control group with normal median nerve conduction, we found an almost identical incidence of symptoms consistent with CTS.

Also, their analysis was done on a per hand instead of per person basis. This analysis is inappropriate as it contradicts the assumption of independent observations; a person’s hands are not independent of each other and are exposed to the same genetic foundation, body mass index, diet, and other health related factors.

Nathan et al comment that we did not use an electrodiagnostic technique as sensitive as theirs for diagnosing a median mononeuropathy. We maintain that that is precisely what is wrong with some forms of electrodiagnostic testing—namely, sensitivity is increased at the expense of specificity. We found a 15% false positive rate for carpal tunnel syndrome with standard electrodiagnostic techniques and yet Nathan et al argue that we should have been using a more sensitive technique; a suggestion that would only serve to increase the false positive rate. We also analysed the data to look at the more severe cases of median mononeuropathy and to see if these workers were more likely to develop symptoms of CTS. This subset of workers were slightly less likely to develop subsequent symptoms than matched controls.

In regard to their concern that we did not use a standardised definition of CTS in our follow up survey, we maintain that a worker with no complaints of numbness, tingling, pain, or burning in the hand or fingers would not be classified as having CTS if a Tinel’s or Phalen’s sign was present. We did not repeat nerve conduction studies or physical examinations on our follow up study but this would not have increased the incidence of CTS.

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Workers chronically exposed to volatile organic solvents have been shown to have significantly more protein in their urine than controls. Cases of acute interstitial nephritis have been reported associated with volatile hydrocarbons. Two were related to chronic exposure in girls, one after a single episode of exposure to polymide epoxy high gloss paint fumes. This is a pathological entity characterised by a monocellular infiltration of the renal tubular interstitium. Although lymphocytes predominate eosinophilia can occur particularly when drugs are identified as the cause. Clinically the picture is of an acute decline in renal function which can be associated with heavy proteinuria and peripheral oedema. Improvement often occurs after removal of the offending agent, but uncontrolled observations suggest that moderate doses of corticosteroids hasten recovery. Recovery should be complete although evidence of interstitial fibrosis at biopsy is associated with a poorer outcome.

**BOOK REVIEWS**


This is a short booklet on an important topic. It is based on contributions made by some two dozen experts participating in a World Health Organisation (WHO) meeting focusing on asbestos, crystalline silica, and coal mine dusts. It aims to be a step by step approach to the development of programmes particularly for developing countries, where "... effective measure are not taken because of a lack of awareness of the problem." The reader may well wonder whether there still exist countries in which workers in multinational giants are not involved either in financial or advisory roles, management is sophisticated enough to have mastered mining and fibre processing technology and yet

Prolonged exposure to an epoxy resin leading to interstitial nephritis

A 51 year old administrator was transferred with a four week history of malaise and intractable vomiting. His creatinine had deteriorated over 10 days from 343 μmol/l to 524 μmol/l. There was no notable personal or family history. He was not taking any medications or herbal remedies. Further questioning showed that over the preceding 18 months he had been building his own aeroplane in a large but enclosed aircraft hangar. This involved the use of a blended epoxy resin (SP Ampeg 20) and the associated SP Ampeg 20 standard hardener (3-aminoethyl-3, 5, 5-trimethylcyclohexylamine and 4, 4'- isopropylidenediphenol) (Standard Polymer Systems). Six months previously he had developed severe contact dermatitis which resolved on use of occlusive hand protection.

Physical examination was unremarkable. Urinary analysis showed 850 mg protein/ 24 hours and negative microscopy. Routine immunological tests and ultrasound of the renal tract were normal. Histological examination of a percutaneous renal biopsy showed an interstitial nephritis with lymphocytes, plasma cells, and a few eosinophils. There was mild interstitial fibrosis.

The patient was started on oral prednisolone at 0.5 mg/kg/day. This was slowly reduced over eight weeks to 10 mg daily and the creatinine improved to 155 μmol/l six months later.

We think that this patient developed interstitial nephritis secondary to inhalation of volatile substances associated with the use of an epoxy resin. He had no contact with other chemicals which could cause this disorder. He had experienced general malaise which cannot be accounted for by the degree of renal impairment, but is consistent with the systemic effects of resin exposure. Previous severe dermatitis may have contributed to the development of renal disease. Since that time he has been advised to cease occupational exposure.

Epoxy resins are formed by the condensation of epichlorhydrin and a dienol in the presence of an amine hardener. Allergic illnesses, particularly dermatitis, can be caused either by the resin or the hardening agents. Contact can be direct or by inhalation of volatile hydrocarbons.1