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 predicting future carpal tunnel syndrome (CTS) and we think that it deserves comment. Schottland et al2 and Bingham et al2 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 a subgroup 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 CTS (but 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 complaints as used by Werner et al. 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 scant and limited clinical CTS, but this subject, but we are concerned that the limited findings from their study will be generalised to imply that NCS are not valuable for predicting future hand or wrist symptoms and CTS. We wonder if the authors would have found had they used a more sensitive NCS measurement such as the 8 cm sensory latency or the maximum latency difference,3 or if they had used an objectively confirmed case definition.

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published a series of articles that have considered this subject.4 One conclusion of our published five year and as yet unpublished 11 year follow up studies of active industrial workers is that NCS are the most reliable predictor of future persistent specific hand or wrist symptoms and CTS. People who develop NCS abnormalities do not inevitably develop characteristic hand or wrist symptoms and CTS, but we have found that asymptomatic subjects with NCS abnormalities are much more likely to develop CTS than asymptomatic subjects without NCS abnormalities. For a dichotomous outcome 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 continuous parameter of various conditions (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 (129x+7)/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 would be helpful in helping to determine whether there is a role for electrodiagnostic screening tests in the workplace.


6 Werner 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.


Authors’ 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.1,4 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. We 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 went on 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% (4/44) 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, sex matched control group of asymptomatic 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 generic 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 used 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 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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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 Ampreg 20) and the associated SP Ampreg 20 standard hardener (3-aminoethyl-3, 5, 5-trimethylcyclohexamylamine and 4, 4'- isopropylidenediphenol) (Standard Polymer Systems). Six months previously he had developed severe contact dermatitis which resolved on use of protective 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 that might have caused this disease. He 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 due to contact with the symptoms had improved with the end of direct contact.

Epoxy resins are formed by the condensation of epichlorohydrin and a diphenol 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

Workers chronically exposed to volatile organic solvents have been shown to have significantly more protein in their urine than controls.2 Cases of acute interstitial nephritis have been reported associated with volatile hydrocarbons. Two were related to chronic exposure in girls aged one after a single episode of exposure to polymamide epoxy high gloss paint fumes.3

Interstitial nephritis is a pathological entity characterised by a mononuclear cell infiltrate 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.

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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 even when 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 has succeeded in maintaining ignorant of dust problems. (For that matter it is too nihilistic to think that where the infrastructure for dust control is absent, that it will be unlikely that the facilities recommended by WHO would be available, and if they were, whom would they be for?) Screening and surveillance is presented as an integral part of a disease prevention programme, intended to detect disease in its preclinical stage at a phase when its progression can be arrested. Surveys to monitor the study of trends and distribution of disease incidence through the systematic collection, analysis, and evaluation of morbidity and mortality reports and other data. To it the authors add the requirements for reporting findings, and for intervention to prevent disease.

As a test of the recommendations of the committee, consider the case of asbestos workers, where the hygiene standard set to protect workers remains to be evaluated. Further, as the author notes, there is uncertainty about the natural history of asbestosis so that we do not know whether it can be detected at a stage where further progression will not occur if exposure ceases.

This was recognised in the discussions leading up to the United Kingdom 1969 Asbestos Regulations. The inadequacy of statutory medical examinations as then practiced and understood was recognised. In this place, Lloyd Davies was able to obtain agreement for the need for a national longitudinal survey of asbestos workers, by a few dedicated physicians, that standardised the medical and radiological, and physiological investigations. As the examinations were primarily for research purposes and for the indirect measures of the effectiveness of the regulations or their implementation, and could not be shown to benefit individual workers at risk, it was agreed that participation should be voluntary for workers, but mandatory for employers for permit examination. In parallel, measures of environmental exposure began to be collected, and cancer morbidity and mortality registers were established.

Other times, other ways. Somewhat belatedly, the European Community woke up, and despite the fact that clinical benefit to individual workers had not been established and in the absence of advance in treatment of any of the asbestos diseases since 1969, directed that asbestos workers shall be compulsorily examined. According to this WHO report (annexe 2), United Kingdom asbestos workers currently undergo primarily a screening programme with some surveillance. The target of the programme it declares is to detect asbestos and pleural changes. The two year interval and the programme should include: medical and occupational histories; chest x ray films; and pulmonary function tests (forced expiratory volume in one second and forced vital capacity (FEV1, and FVC). In fact, Health and Safety Executive regulations implementing the European Directive do not prescribe the details of examination, they are left to the discretion and clinical judgement of the appointed doctor. The clockwork has been left to be eventually turned back to the appointed doctor's system, largely eradicated on 1 February 1973.

The WHO states that the effectiveness of the United Kingdom programme has not been assessed by published statistics. Annual audit of the mesothelioma register is certainly published, and there has been a mortality analysis of subjects on the asbestos register, but...
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