Abnormal nerve conduction studies as an isolated finding should not be treated or used to screen out workers but rather should be followed up to establish, if it exists, the natural history of median nerve function and hand symptoms. The sound practice of medicine dictates treatment of the patient, not the abnormal laboratory value. Nerve conduction studies must use norms based on a healthy population without known exposures. If a shift in median nerve function is found more often in workers involved in repetitive activities, this cannot be accepted as a worker norm in the absence of prospective studies to determine the long term consequences. An oft quoted saying by Carl Sagan fits the study’s conclusion of a median sensory nerve conduction not predicting hand symptoms, “Absence of evidence is not evidence of absence.”

**CORRESPONDENCE**

Use of screening nerve conduction studies for predicting future carpal tunnel syndrome

Editor—Werner et al consider a question critical to the interpretation of nerve conduction studies used as a screening tool to identify workers at risk of hand and finger disorders associated with repetitive motion. They demonstrate that an abnormal median sensory nerve conduction in asymptomatic active workers is not predictive of development of hand and finger symptoms in the future. The results do not discuss the possibility of selection bias in choosing asymptomatic workers employed for years in manufacturing jobs with a range of repetitive hand movements. Active workers with abnormal nerve conduction studies who perform repetitive tasks for years and remain asymptomatic may be the ones that have attributes, yet to be determined, that prevent the attainment of a symptom threshold. The lack of follow up of nerve conduction studies did not allow the authors to answer their question regarding the predictive value of abnormal median sensory nerve conduction. Without these data it is not known whether the active workers and controls who developed symptoms had a proponent (carpal tunnel syndrome) often found example, it is not known if the nerve conduction studies of the 77 asymptomatic workers would remain abnormal at follow up one to two years later.

Median mononeuropathy associated with carpal tunnel syndrome has numerous underlying pathological processes from ischemia secondary to altered blood flow, direct axonal compression, and chronic degenerative changes. Therefore it is not surprising that various combinations occur when screening workers with quantitative nerve studies and a symptom questionnaire. Those who commonly examine cases of median mononeuropathy (carpal tunnel syndrome) often find a low correlation between symptoms, physical findings, and nerve conduction studies. This is not unexpected as carpal tunnel syndrome is a multifactorial problem in which the time line for change and the underlying pathology may differ.

Before discarding nerve conduction studies as a screening tool, another approach that may yield more predictive risk of is the examination of median nerve function in workers at pre-employment with follow up studies to identify change and the association with ergonomic stressors. Also electodiagnostics measures known to better identify abnormalities in the carpal canal portion of the median nerve such as the midpalmar latency have increased sensitivity.¹

Abnormal nerve conduction studies as an isolated finding should not be treated or used to screen out workers but rather should be followed up to establish, if it exists, the natural history of median nerve function and hand symptoms. The sound practice of medicine dictates treatment of the patient, not the abnormal laboratory value. Nerve conduction studies must use norms based on a healthy population without known exposures. If a shift in median nerve function is found more often in workers involved in repetitive activities, this cannot be accepted as a worker norm in the absence of prospective studies to determine the long term consequences. An oft quoted saying by Carl Sagan fits the study’s conclusion of a median sensory nerve conduction not predicting hand symptoms, “Absence of evidence is not evidence of absence.”

Authors’ reply—We appreciate the comments from Bleecker regarding our recent article on the predictive value of nerve conduction studies in predicting future symptoms consistent with carpal tunnel syndrome (CTS). The issues raised are appropriate and will hopefully encourage more research in this area. The concerns about a possible selection bias are real as our study was based upon active workers and did not represent an inception cohort of workers. We agree that this type of studies need to be duplicated with an inception cohort to show the natural history of median nerve conduction across the wrist over time, with a close correlation of hand and finger symptoms. Our sample was randomly chosen from a high participation rate. The duration of employment at their present job did not influence the reporting of symptoms in our study; this argues against a selection bias of survivors but does not rule out the possibility.

The issue of whether or not the latency of the median nerve evoked response changes over time in active workers was not considered in our study and may provide some additional information regarding the natural history of median mononeuropathy at the wrist but it does not assess the risk for CTS. Our study supports the conclusion that an active asymptomatic worker, with a documented median mononeuropathy, is not at increased risk for developing CTS (even though this contradicts our original hypothesis). Knowing the change in latency at follow up would not change this conclusion.

We are in agreement with Bleecker that you treat the patient and not the test. Unfortunately, many clinicians think that a prolonged median latency is the equivalent of carpal tunnel syndrome. This is in fact true of the high sensitivity reported for nerve conduction studies in relation to CTS.¹³ We raise the issue that the high sensitivity noted in a clinical setting with symptomatic patients is not found in the cross sectional screening of the workplace.¹⁴ Also, the specificity is lower than previously thought.¹⁶

This study questions the value of a screening nerve conduction study but also raises issues with their value as a diagnostic test. We think that the current criteria for determining a threshold for a prolongation of the median nerve evoked response across the wrist in the active worker needs to be re-evaluated. Our normative data, from our asymptomatic workers, suggest that a relative difference of 0.8 ms be used instead of 0.5 ms that is used in many laboratories.¹⁷ The data from Stetson et al supports the assumption that active industrial workers represent a shift in the population with more prolongation of the median nerve present among active workers. A longitudinal study will be necessary to find whether the active worker with a prolonged median nerve evoked response across the wrist develops any significant problems in the future. Our study showed that within a mean of 17 months of follow up that asymptomatic workers with a median mononeuropathy were not at greater risk of developing symptoms consistent with CTS. This argues against using nerve conduction testing as a screening procedure but a definitive study remains to be done.

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**The sex ratios of offspring of people exposed to non-ionizing radiation**

Editor—In recent years there has been increasing concern that exposures to non-ionizing electromagnetic fields have adverse reproductive effects.¹ It is well established that women, many forms of non-endocrine disease are associated with low testosterone, or high gonadotrophin concentrations, or both.¹ There is very substantial evidence that
such a hormone profile predisposes men to sire a disproportionate number of daughters. Moreover there are several forms of occupational exposure which are known or suspected to be associated with such a hormone profile in men with low sex ratios (proportion of daughters). For instance, such hormone profiles and sex ratios have been reported for professional divers, applicators of the nematocide DBCP, and men exposed to dioxin, vinclozolin, and borates. There are also cases reported in carbon setters and in professional divers and men engaged in the alcohol trade. I suggest that there is now enough evidence to warrant such concern about exposure to non-ionising radiation.

Knave et al. cited studies documenting fatigue, headaches, dizziness, impaired memory, nausea, loss of strength in limbs, respiratory difficulties, sleep disturbances, and reduced libido in workers exposed to electric fields. Exposure to microwaves is reportedly associated with a reduction in sperm count in humans, and exposure to electric fields (50 Hz, 5kV/m) was reported to produce such a reduction in sperm concentration in adult rats. Lastly, Free et al. reported significantly reduced plasma testosterone in rats exposed to strong (60 Hz) electric fields.

The table reproduces data on the sexes of offspring of people exposed to various forms of non-ionising radiation. (1) The data on men exposed to high voltages suggest a low offspring sex ratio. This in turn propels a low hormone profile in non-specific pathology. Men engaged in such work should have their hormone concentrations assayed, especially those complaining of such symptoms as are outlined above. (2) The two sets of data for female physiotherapists are in conflict. Larsen et al. reported a highly significant dose related excess of daughters born to exposed women but Gubérán et al. failed to replicate this finding. The discrepancy could be due to methodological error. In both studies the women were questioned about exposure after conception, but the relevant exposure may precede conception. Both authors were assuming that if radiation affects sex ratio, it does so by causing early sex related fetal mortality. However, radiation may affect sex ratio by altering maternal hormone concentrations preconceptionally.

(3) Little is known about the effects on the sex ratio of offspring of women exposed to potential pathogens. Some forms of maternal exposure are reportedly associated with an excess of daughters—for example, to high frequency electromagnetic radiation, strong static fields, or low frequency electromagnetic fields, and (possibly) dioxin. It is not known whether these excesses of daughters are hormonally caused. It would be interesting to know whether women in these categories who produce excess daughters complain of non-specific malaise (as described by Knave et al. for men) or whether they report a sense of well-being.

In order to determine whether the effects of non-ionising radiation are different from those of ionising radiation. If ionising radiation were to cause sex linked mutations, then exposed fathers could produce an excess of sons, and exposed mothers an excess of daughters. The data of Cox et al. and of Dickinson et al. suggest that this expectation is fulfilled.

(5) The low sex ratio of children in the vicinity of a radiolocation system is suggestive if only because the authors could think of no explanation for it. Attempts should be made to confirm this finding in eastern Europe and elsewhere.

I am grateful to Mr Roger Coghill, Coghill Research Laboratories, Wivenhoe, for helpful correspondence, and to Mr A Irgens, Department of Occupational Medicine, Haukeland Hospital, Bergen, Norway, for forwarding me the abstract of a paper in press of which he is a co-author.

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Table 1

<table>
<thead>
<tr>
<th>Reference</th>
<th>Exposed</th>
<th>Controls</th>
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<td>Parents exposed to strong static and low frequency electromagnetic fields:</td>
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<td>Irgens et al.</td>
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<tr>
<td>Exposed men sired offspring with a slightly reduced sex ratio. Exposed women produced offspring with a significantly and substantially reduced sex ratio</td>
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</table>

*The P values correspond to the results of two sided t 2 + y 2 tests.
†In the data of Nordström et al., the controls were pregnancies sired by men who were less exposed or unexposed in the same environment.
‡In the data of Nordström et al., the controls were the pregnancies sired by the men before exposure.
§Mubarak and Mubarak provided no control data, but if their sex ratio is contrasted with an expected* live birth white birth sex ratio of 0.514, the value of the resultant χ2 is well beyond tabulated values.
∥The data of Kolodynska and Kolodynska are not live births but of children living in schools. The exposed children lived within 20 km of the radioelectric system. The controls lived in a nearby region unexposed to the system. Both areas are reportedly without major point sources of pollution. The difference between the sex ratios of the exposed and the controls in these data is only suggestive (P = 0.08). But if the sex ratio of the exposed is contrasted with an expected* live birth sex ratio of 0.514 then χ2 is 0.000001. If high voltage electricity is not included, then χ2 is 0.00001. If we do not leave school earlier that girls in this region, then an explanation is lacking, and should provisionally be suspected in the radioelectric system.
| The raw data of the study of Irgens et al. will become available on publication of their paper. | | | | | | |
The sex ratios of offspring of people exposed to non-ionising radiation.

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