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TECHNICAL REPORT

**THE EFFECTS of
INORGANIC LEAD on
BEHAVIORAL and
NEUROLOGIC FUNCTION**

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THE EFFECTS OF INORGANIC LEAD ON
BEHAVIORAL AND NEUROLOGIC FUNCTION

Final Report

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ABSTRACT

REPKO, J. D., CORUM, C. R., JONES, P. D., and GARCIA, L. S., JR. *The effects of inorganic lead on behavioral and neurologic function. Final Report of HEW (NIOSH) Contract No. 210-75-0054* -- Fifty-three behavioral measures of sensory and motor functioning, six measures of nerve conduction velocity, five indices of inorganic lead absorption, a clinical electromyogram, a clinical neurological examination, and demographic data were obtained from 85 experimental and 55 comparison subjects. The experimental subjects were paid volunteers from among workers exposed to inorganic lead in the storage battery manufacturing industry. The comparison group consisted of paid volunteers from a light manufacturing industry (manufacture of battery cases), service industries, or were unemployed. The comparison group participants had no known occupational exposure to inorganic lead or other neurotoxic chemical. The data were collected at the authors' laboratory or at a portable field laboratory. Analysis of the data showed that the two study groups were statistically identical in terms of age, height, and weight; a slight, inconsequential difference in educational level was noted, however. The results of the blood and urine biomedical determinations showed that the experimental group had a mean blood lead of 46ug/100ml; the mean blood lead for the comparison group was 18ug/100ml. The results further indicate that for the experimental subjects, PbB, ALA-D, FEP, PbU, and δ -ALA were inter-correlated and each measure could be predicted from each of the other measures. The greatest intercorrelations were between PbB and the remaining four measures. However, except for a positive relationship between FEP and certain of the pure-tone threshold measures, none of these biomedical indicators bore a significant relationship to NCV or behavioral measures. Results from the clinical electromyogram and neurological examinations indicated that the experimental group was asymptomatic with respect to lead and did not differ clinically from the comparison group. Differences between the two groups were evident in the NCV and behavioral measures. The lead-exposed workers showed a statistically significant lower conduction velocity in the magnitude of 5 to 9m/sec for the MCV of the median, ulnar, posterior tibial, and deep peroneal nerves. Also, the SCV of the ulnar nerve was significantly slower for the lead workers; no significant differences in the CVSF of the ulnar nerve were noted. The results of the behavioral measures showed that deficits in visual reaction time, under response control of the ulnar nerve, as well as deficits in auditory functioning, in terms of both pure-tone thresholds and tone-decay, were all adversely affected by low-level lead absorption. No differences were noted in the grip strength, eye-hand coordination, or other psychological/social measures.

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THE EFFECTS OF INORGANIC LEAD ON BEHAVIORAL AND NEUROLOGIC FUNCTION

INTRODUCTION

Workers in certain industries and occupations have been exposed for many years to varying levels of such neurotoxic materials as pesticides, heavy metals, and solvents. Health effects of this on-the-job exposure have been detected in some instances only after workers had developed frank clinical signs and symptoms of poisoning or had suffered permanent functional impairment and were unable to work. Of the heavy metals, inorganic lead is widely used. Potential health hazards from industrial exposures are created in the United States by the use of over one million tons of lead each year, the release of hundreds of thousands of tons of lead into the atmosphere each year from automobile exhaust emissions, and the occupational exposure to lead of workers in at least 110 occupations.

The adverse effects on the nervous system of acute and chronic exposure to neurotoxic materials are well documented. Based on reports by Repko, Morgan, and Nicholson (1975), Seppalainen and Hernberg (1972), and Seppalainen, Tola, Hernberg, and Kock (1975) several deleterious effects of lead upon the nervous system and sensory and neuromuscular functioning have been demonstrated. There are, however, several additional basic research areas which must be investigated to assess the extent and nature of specific functional disorders and the relationship between these disorders and occupational safety and health.

Recent research by Catton, *et al.* (1970), Fullerton (1966) and by Seppalainen, *et al.* (1972, 1975) has emphasized the early detection of sub-clinical lead neuropathy by measurements of the maximal conduction velocity and the conduction velocity of slow fibers of peripheral nerves through the use of standard electromyographic techniques. Conduction velocity in the more peripheral portions of these nerves is decreased and fibrillations have been seen in electromyographic recordings. Repko, *et al.* (1975) have studied the effects of lead exposure upon a variety of behavioral measures which included sensory functions, vigilance, visual pattern discrimination, arithmetic computation, muscular strength and endurance, tremor, and eye-hand coordination. Their results indicate that there are significant relationships between body burden indices of lead and several measures of behavioral functioning. These behavioral findings are generally congruent with clinical reports and with recent electrophysiological findings. However, it is important that these behavioral and electrophysiological findings be replicated in a single study with individual workers providing both behavioral and electrophysiological data. Greater precision results in evaluating within the same population the relationships among specific peripheral neuropathies, overt behavioral dysfunction, and the body burden of lead.

GENERAL BACKGROUND OF PREVIOUS LEAD RESEARCH

The results and discussion of the analyses of the behavioral measures are adequately documented in the previously referenced *NIOSH Research Report* (Repko, *et al.*, 1975). As reported in that document, the relationships among the measures of task performance and the clinical indices of lead absorption were examined through the use of standard statistical procedures. The results of those analyses indicated that the intellectual functions measured were unaffected by increases in absorbed lead. On the other hand, sensory (hearing), neuromuscular or psychomotor (tremor and eye-hand coordination), and psychological (hostility, aggression, and general dysphoria) functions were all detrimentally influenced by increased lead absorption. Moreover, study data indicate that blood lead (PbB) may not be a sensitive measure of changes in functional capacity since red cell aminolevulinic acid dehydrase (ALA-D) was found to be the most sensitive predictor of task performance. These conclusions concerning changes in functional capacity were based entirely upon significant correlations between specific measures of task performance and one or more of the clinical indicators.

Repko, *et al.* (1975) reported that the clinical indicators bore no statistically reliable relationship to the types of behavioral functioning assessed through the use of a multiple task performance battery, a test of visual acuity, and a digit span test. This is not to say that the functional areas assessed by these tests were unaltered by lead; it can only be stated that the specific tests employed previously did not detect differences in functioning. In regard to the performance battery, the task situation was sufficiently complex that asymptotic performance was not obtained in the time provided. Moreover, both age and level of education were the primary contributing factors accounting for the observed performances on this task battery. At the other extreme, the tests of visual acuity and digit span failed to detect functional differences because of simplicity and lack of sensitivity. Finally, the measures of urine lead (PbU), urine coproporphyrin (CPU), and urine δ -aminolevulinic acid (δ -ALA) were not consistently related to functional capacity.

As reported by Repko (1977), critical evaluation of the Repko, *et al.* (1975) study data indicates that a paradoxical relationship exists between the control and experimental groups. On the one hand, many measures of performance obtained from the lead-exposed workers indicate significant decreases in functional capacity with increases in PbB or decreases in ALA-D. On the other hand, the study failed to demonstrate differences as compared to the control group; in fact, the control group presented an even poorer performance level than did the lead-exposed group in several instances. The differences in the outcome of the two groups is not paradoxical, however, when motivation to participate and other factors are taken into account. Specifically, two important factors were identified (Repko, 1977): First, while the two groups were demographically similar, their motivation for participation, and therefore their probable level of effort during the testing process, may have been quite dissimilar. Second, the variability in performance exhibited by the controls suggests not only inconsistent test behavior but also an overall group shift in a criterion of what constitutes appropriate test behavior.

Recognizing that the utilization of the control data in the earlier research has severe limitations were motivation is a factor influencing performance,

or where differences in test behavior are suggested, or where antecedent noise exposure may affect hearing levels, the correlative changes in functional capacity exhibited by the lead-exposed workers *must be regarded as more conclusive* since the changes are related to biomedical indicators of exposure and effect. The tests involving visual acuity and auditory acuity are motivationally independent tests, whereas, all other tests utilized in that study required some optimum efficiency in performance, especially in the absence of individual baseline data.

Additional analyses were later performed on the lead-exposed workers' data through the use of a univariate analysis of variance of the difference between the group of workers possessing PbB values of 69ug/100ml or below at the time of testing and the group of workers with PbB of 69ug/100ml or above. In general, the Repko (1977) report of these additional analyses did not change the basic conclusions summarized in the earlier, published report. Quite the contrary, these additional analyses enhanced the conclusion that functional capacity is decreased in workers whose ALA-D activity is approximately 90 percent inhibited or in workers exhibiting a PbB of 70ug/100ml greater.

The Repko (1977) report of analyses of the measures of auditory functioning demonstrated that the mean hearing thresholds were significantly higher in all cases for the high-lead group, irrespective of whether a PbB exposure criteria was used or an ALA-D effect criterion was used. Despite the fact that the auditory tests were conducted in the open, without a sound attenuation chamber, the data showed true effects with respect to the *relative* hearing thresholds of the workers. Workers whose PbB levels exceeded 70ug/100ml or whose ALA-D activity was substantially inhibited showed statistically significant changes representing progressive hearing loss. The significant correlations between PbB and hearing as well as between decreases in ALA-D and hearing threshold substantiated this conclusion. The test of tone decay also provided evidence of auditory dysfunction. Workers with the higher PbB levels showed a statistically greater amount of decay than those with lower levels; moreover, the correlation between these two measures was also significant.

The results of the analyses of the data obtained from the tests of tremor and eye-hand coordination reported by Repko (1977) are consistent with the earlier published conclusions; the analyses revealed that significantly greater amounts of tremor and significantly increased latency and response variability of eye-hand coordination were evidenced. Those data suggest that ALA-D activity is in some way related to functional changes involving fine motor coordination. Except for only one measure, decreases in ALA-D significantly correlated with increases in tremor and loss of eye-hand coordination. Increases in PbB were also related to correlative increases in tremor and loss of eye-hand coordination but not in the same manner nor as extensively as was the relationship with ALA-D. On the other hand, both ALA-D and PbB were associated with changes in gross motor control. Analyses of the three measures associated with strength indicated that strength increased with increases in the level of PbB and with decreases in ALA-D activity. Essentially the same conclusion was discussed in the Repko, *et al.* (1975) report.

While it was stated by Repko, *et al.* (1975) that the psychological impact of working in a leaded environment was one of increased hostility, depression and general dysphoria, subsequent analyses did not support this conclusion.

Since the control group did not provide consistent and reliable data as a baseline for comparison, it is inappropriate to draw conclusions concerning psychological processes. Moreover, the additional analyses reported by Repko (1977) provided an adequate indication of the lack of a relationship between these measures of mood or affect and either the effects of exposure or the presence of lead in the blood.

SPECIFIC OBJECTIVES

It is known from research done in the domain of work psychology and human performance that sensory-perceptual and psychomotor abilities are the most important psychological functions utilized in factory work containing manual tasks. Thus a decline of these abilities means that the individual's resources or capacity to meet the demands of the job are reduced. The implication then is that psychological health and the ability of the individual to work safely are detrimentally affected by neurotoxins such as lead.

Whether described as changes in behavioral or psychological functioning, as Grandjean (1977) or Repko (1977) have reported, or as changes in nerve conduction velocity as Seppäläinen (1977) has reported, it is clear that changes in functional capacity represent a material impairment of health. It is important not to disregard or discount such effects or to classify them as some lesser effect. Subclinical effects, irrespective of whether they are evidenced as metabolic, neurological, or behavioral changes, are clinical once the effect has been delineated. There should be no dichotomy between clinical or subclinical effects; it is an artificial, academic exercise in classification.

The central purpose of the current study was to assess the effects of inorganic lead found in the work place on worker behavior and neurologic function. A recent review of the neurological and behavioral sequelae of inorganic lead absorption describes the deleterious effects of lead on the nervous system and sensory and neuromuscular functioning (Repko & Corum, 1976). Moreover, the studies by Repko, *et al.* (1975) and Seppäläinen (Seppäläinen & Herberg, 1972; Seppäläinen, 1977; Seppäläinen, *et al.*, 1975) and by Grandjean (1977) clearly demonstrate specific effects of lead. However, several additional basic research areas are investigated herein to assess the extent and nature of specific functional disorders and the relationship between these disorders to occupational safety and health. As a secondary purpose of the study, the information generated aids in detecting early reversible changes in workers experiencing long-term, low-level exposure to inorganic lead. These effects of lead are significant in terms of evaluating the adequacy of recommended safety exposure levels and for rating risk of safety-health problems at the job site. As an adjunct to the study, it is important that a non-invasive behavioral test or set of tests is developed which can be employed as early warning indicators of potential adverse effects of lead. The results of the study, therefore, are adequate and appropriate for diagnosing a sub-clinical peripheral neuropathy and concomitant diminished behavioral performance.

METHOD

The research reported herein was conducted during a 28-month period from 26 June 1975 through 15 November 1977. The first ten months of the research project were devoted to the development and pilot-testing of a multifactor behavioral and neurological test battery for the assessment of functional changes that result from exposure to inorganic lead. The data collection phase of the research project was completed during the period from April 1976 through April 1977. Performance and behavioral test measurements, neurological and electroneuromyographic examinations, as well as biologic samples, were obtained individually from a group of 140 workers. The final months of the project were devoted to the analysis of the data and preparation of this report.

DESCRIPTION OF STUDY POPULATION

The subjects employed in this study were 140 physically normal male and female volunteers from two regions of the United States. Of this total sample, 85 workers constituted the *experimental* (lead-exposed group), while the remaining 55 workers constituted the *comparison* (or control) group having no known occupational exposure to lead. The individuals in the experimental group were all engaged in various aspects of the storage battery manufacturing industry. In addition, the experimental subjects were accepted for inclusion in the study according to the following criteria: (a) a current and continuous history during the past five years of a mean blood lead (PbB) level below 80ug/100ml, (b) a history of not more than one excursion about 80ug/100ml, and (c) no previous history of PbB in excess of 90ug/100ml. The workers in the control group, on the other hand, were not currently exposed to, nor had they ever knowingly worked with inorganic lead. Controls were drawn from light manufacturing, service industries, and the unemployed. Insofar as possible and practical, the two groups of subjects were matched for race and sex and in a ratio of two experimental for each control within five-year age blocks.

Protection of Subjects

In order to ensure that the subjects were properly informed, each volunteer was given a complete and comprehensive explanation of the study (describing the objectives and experimental procedures) by the Project Director. It was explained that certain minimal elements of psychological and physical risk might be involved, and that no one was under any obligation to participate in the research. After at least a 24hr period, volunteers were given the opportunity to ask questions concerning the study and to express their desires concerning participation in the study. Volunteers were allowed to withdraw from the study at any time. During the neurological and electroneuromyographic examinations a physician (neurologist) was always present.

Each worker who volunteered for the study was asked to read and sign a statement regarding the purposes, risks, and confidentiality of the results of the study (the specific forms each worker was required to sign are attached to this report in Appendix B). In addition, each volunteer was asked to designate a physician, other than the company physician, to whom any significant behavioral or medical test results should be sent if they so desired.

TEST PROTOCOL

The cooperation of workers at each of the two locations was obtained through correspondence and meetings with representatives of the company and subsequent meetings with the workers as well as with officers of local unions representing the workers. The performance and medical testing was conducted in a temporary laboratory set up in the field or in the laboratory facilities of the authors. A general description of the events at each location is given in the paragraphs which follow.

The initial activity at each location involved setting up the apparatus used in the behavioral test battery and the equipment used by the neurologist and electromyographer; particular attention was given to the testing of equipment and calibration of the electromyograph and audiometer. The next step involved coordination of the scheduling of volunteers to be tested at the completion of each daily work shift. During these initial steps in the procedure, the Project Director was available to the workers for the purpose of answering questions regarding the study.

Two or three subjects were scheduled to report for behavioral testing at the same time. Approximately 2 1/2 to 3hr were allotted for the testing of each group of subjects; between one and three groups were tested during each day of testing. Testing was scheduled at the end of each work shift so as not to interfere with the workers' normal work schedule. At the beginning of the testing period, each subject was required to complete the necessary consent and release forms. Immediately upon completion of the necessary forms, each worker was required to complete certain psychological tests. The workers were then evaluated through the use of the behavioral tests. When the workers finished, each received a personal-data questionnaire and a 150ml polypropylene urine-collection container which were to be returned at the time of the neurological examination.

The neurological and electroneuromyographic (ENMG) examinations were conducted on a subsequent day in order to reduce the amount of continuous time involved in the testing procedures and to facilitate the work schedule of the neurologist and electromyographer. The neurological examination required approximately 1/2 to 3/4hr for completion; the time devoted to the ENMG examinations was also 1/2 to 3/4hr. These medical examinations were conducted individually after the regular work day. At the time of examination, a blood sample was collected in a 10ml, heparinized vacutainer. Finally, the completed personal-data questionnaire and the urine sample were collected from each worker; each worker was then paid \$15.00 (plus expenses for transportation, where necessary) for participation in the study.

General descriptions of the neurological examination, electroneuromyographic examination, the behavioral test battery, and the procedures involved in the blood and urine analyses, are given in the following sections. Comprehensive and detailed descriptions, however, are provided elsewhere as noted.

NEUROLOGICAL EVALUATION

The neurological examination consisted of a battery of tests which included an examination of (a) each of the twelve cranial nerves, (b) the motor system, (c) the sensory system (including visual, auditory, cutaneous, and proprioceptive systems), and (d) coordination and reflexes. The examination also included inspection, palpation, and auscultation of the head. The symmetry and deformities of the head, spine, and extremities were also examined. The specific examination sequence and the evaluative criteria are described adequately elsewhere (see Repko, *et al.*, 1976, Appendix D).

ELECTRONEUROMYOGRAPHIC EXAMINATION

The electroneuromyographic examination involved both measures of the nerve conduction velocity (NCV) of the peripheral nerves of the extremities and a myographic evaluation (EMG) of the muscles innervated by these nerves. Measures of the motor conduction velocity (MCV) of the median, ulnar, deep peroneal (anterior tibial nerve), and posterior tibial nerves as well as the maximum sensory conduction velocity (SCV) and the conduction velocity of slow fibers (CVSF) of the ulnar nerve were obtained from the right extremities of the workers tested. An evaluation of both the right and left extremities was conducted on workers who had a left-side dominance. If the worker was willing to undergo additional tests, the EMG was conducted to investigate evidence of denervation potentials in the muscles (i.e., positive sharp waves, etc.) and ulnar nerve threshold excitability.

The equipment used for the electroneuromyographic examinations was implemented by the authors from commercially available components (see Figure 1). Briefly, it involved the use of a Tektronix (Model R-7313) oscilloscope with a Tektronix (Model 7A22) differential amplifier and dual time-base (Model 7B53A) module, a Grass stimulator (Model S-88) with accessory footswitch, and two Grass stimulus isolation units (Model SIU 5-300). The pre-amplifier was constructed, with minor modifications in input circuitry, based on the instrumentation amplifier described by Huntsman and Nichols (1971). A detailed description of the electroneuromyographic system is provided in Appendix A. Limb temperatures, both core and surface, were obtained through the use of a Bailey Instrument Thermalert (Model TH-2) with digital readout. All electroneuromyographic (ENMG) tests were conducted in a portable, shielded room in order to isolate the equipment from interfering electrical signals.

Before each data collection period the ENMG equipment was calibrated for the proper timebase and voltage. Prior to the examination the procedure was explained to each subject. The areas where the electrodes were to be placed were abraded with alcohol to provide good skin contact. The electrodes were then moistened with electrode gel (Teca Electrode Conductivity Gel) and secured to the proper area with surgical tape.

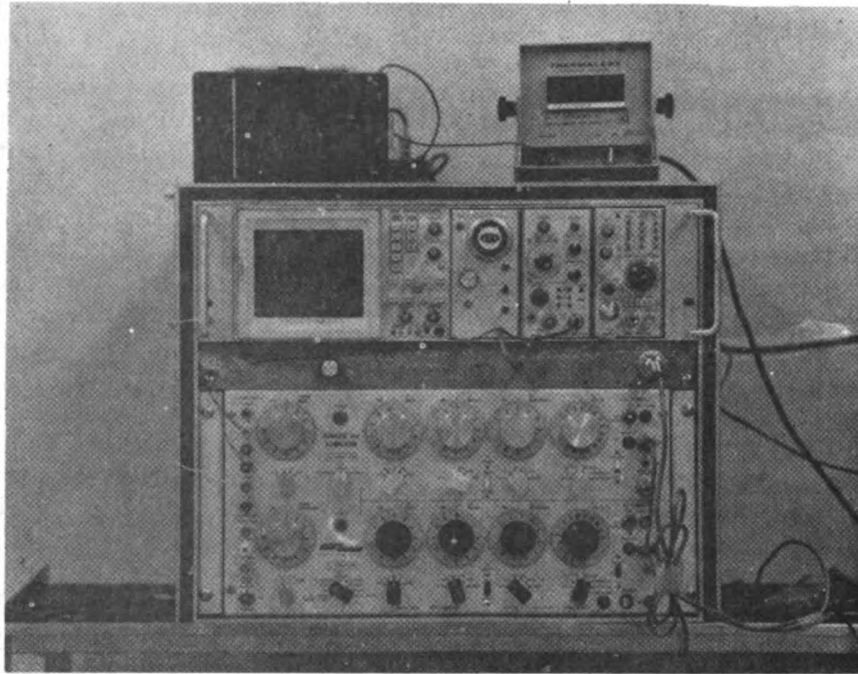


Figure 1. Front view of the electroneuromyographic system showing oscilloscope, differential amplifier, dual-time base module (upper unit), and stimulator (lower unit).

The surface temperature of the upper extremities was maintained at between 30° and 33°C at both the proximal and distal stimulation sites. If the temperature did not fall within this range, the extremities were either cooled with the aid of a water bath or warmed by wrapping with a turkish towel. A core (internal) temperature was obtained by inserting a 1.59cm long temperature probe into the belly of the brachioradialis muscle. No attempt was made to maintain surface temperature of the lower extremities within a specified range because of the difficulty involved in cooling or warming such a large body area. The core temperature of the leg was obtained by inserting the temperature probe into the belly of the gastrocnemius muscle.

The maximal motor conduction velocity (MCV) of the workers was measured with the standard method described by Hodes, Tarrabee, and German (1948). To obtain the MCV of the median nerve, stimulation was introduced distally by a hand-held stimulator at approximately 2cm above the wrist fold and either just above or just below the Ligament of Struthers of the dorsal aspect of the elbow. At each site, supramaximal square-wave stimuli of 0.3ms duration were delivered and the muscle response was recorded with skin electrodes on the belly and tendon of the extensor digitorum brevis. The MCV of the ulnar nerve was obtained in the same manner. The distal stimulation point was approximately 2cm above the wrist-fold and the approximate site was 2 to 5cm above

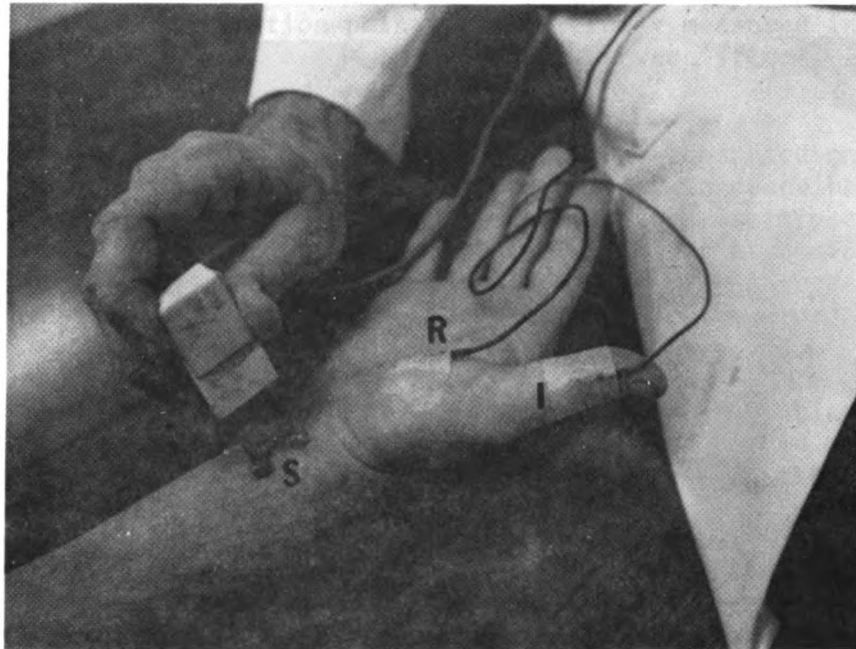


Figure 2. View of distal electrode placement for measures of MCV of the median nerve, showing stimulator (S), recording electrode (R), indifferent electrode (I), and ground electrode (not visible).

the sulcus nerve ulnaris. The muscle response was recorded with skin electrode on the belly and tendon of the abductor digiti minimi. Figures 2 and 3 demonstrate the placement of electrodes for obtaining the MCV of the median and ulnar nerve.

The MCV of the deep peroneal nerve was obtained by stimulating distally at the dorsal aspect of the ankle and proximally just below the head of the fibula. The muscle response was recorded with skin electrodes on the belly of the extensor digitorum brevis and the distal interphalangeal joint of the great toe. The MCV of the posterior tibial nerve was obtained by stimulating distally, just posterior to the medial malleolus and proximally at the posterior knee region. The muscle response was recorded with skin electrodes on the belly of the abductor hallucis and the distal interphalangeal joint of the great toe.

Before stimulating the nerve, the tips of the stimulator were moistened with electrode jelly. The nerve was then palpated at the point of stimulation with the stimulator placed over the nerve. The subject was notified as to the beginning with a small amount of current. Rapidly, the supramaximal level was reached and a clean trace of the distal nerve response was displayed and

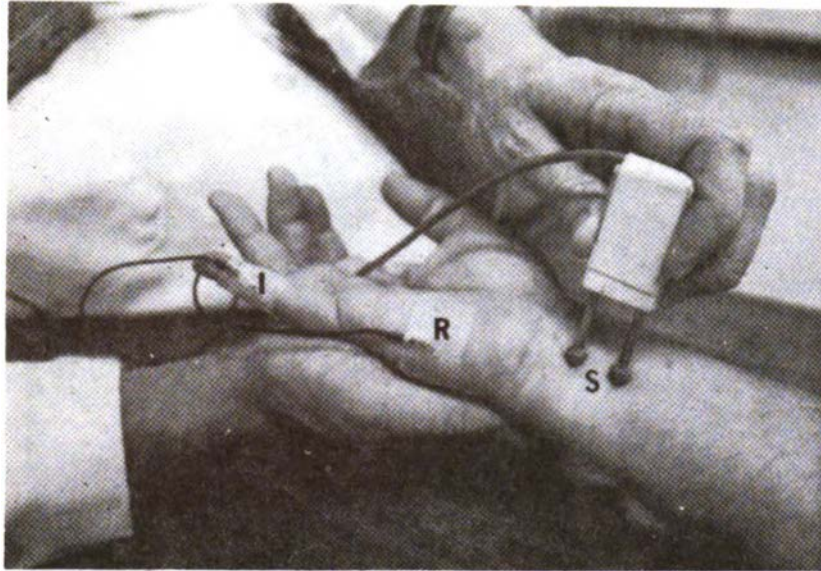


Figure 3. View of distal electrode placement for measures of MCV of the ulnar nerve, showing stimulator (S), recording electrode (R), indifferent electrode (I), and ground electrode (not visible).

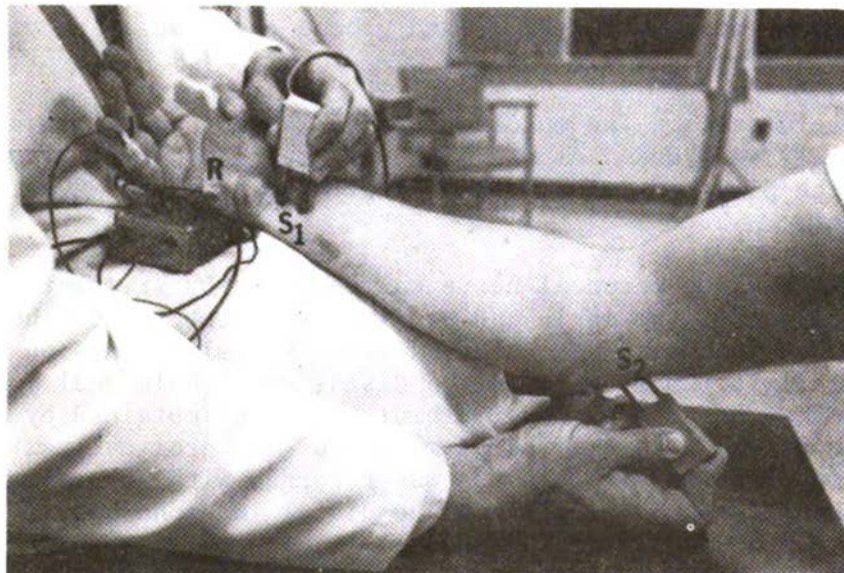


Figure 4. View of electrode placement for measures of the CVSF of the ulnar nerve, showing stimulators (S₁ and S₂), recording electrode (R), indifferent electrode (I), and ground electrode (not visible).

stored on the upper screen of the oscilloscope; the same procedure was followed at the proximal site and a clean trace was displayed and stored on the lower screen of the oscilloscope. This display was then recorded by taking a picture with a Polaroid camera. For both distal and proximal responses the latencies were measured and recorded with the aid of the potentiometer. The distance between stimulation points was carefully measured in centimeters with a tape measure and recorded. The distance between stimulation points was divided by the latency to determine the MCV.

The sensory conduction velocity examination was conducted on the ulnar nerve alone and was basically identical to the procedure described for determining MCV. For these measures, the sweep speed on the oscilloscope was decreased and the sensitivity level increased. Instead of standard surface electrodes for reference and pickup, flexible lead wire was formed into a semicircle which could be secured to the distal and proximal phalanges.

To determine the conduction velocity of the slower fibers (CVSF) of the ulnar nerve, the technique developed by Seppäläinen (1971), which is a modification of the method first used by Hofp (1962), was employed. This technique involves a partial antidromic block and thus required the simultaneous use of two hand-held stimulators. The skin electrodes remained placed as they were for determining the MCV and both stimulators were placed over the nerve distally and proximally at the same points used in determining the MCV (see Figure 4). Separately, the evoked muscle action potentials were stored on the upper screen of the oscilloscope to verify both proper stimulator placement and delivery of full response. Both distal and proximal sites were stimulated with the proximal stimuli being delayed sufficiently to allow two successive muscular responses corresponding to each of the stimuli. The interval between the two stimuli was then decreased until the proximal response began to decrease. The point of response just prior to this decrease was stored on the lower screen of the oscilloscope and a Polaroid picture made. The distance between stimulation points was measured and the time interval between the two full responses was recorded. The distance was then divided by the latency minus 1ms (refractory period of the nerve fibers) to determine the CVSF.

The electromyography (EMG) examination was conducted after the completion of the NCV examinations. As with the NCV examinations, the EMG procedure was explained to the worker at the outset. The skin on the posterior portion of the hand was abraded and a ground electrode secured with tape. The first dorsal interosseous muscle was the first site tested and was cleaned thoroughly (with alcohol) before needle insertion. A 3.8cm long coaxial needle was used. After muscle relaxation the needle was inserted with a swift, smooth movement. The insertional activity was viewed on the oscilloscope and monitored auditorily as it was simultaneously being recorded on the FM tape recorder. The needle was then moved in a quadrant format and then remained in a given position (for approximately 20sec) in order to determine any abnormal potentials.

The worker then was asked to contract the muscle maximally and the examiner described the interference pattern on the voice channel of the tape recorder. The subject was then requested to relax as the examiner observed this action and recorded any abnormal potential. Upon complete muscle relaxation the needle was removed from this site. The abductor digiti minimi was then examined and potentials recorded in the same manner.

CLINICAL DETERMINATIONS

Five biochemical measures of lead absorption were obtained for each participant. Three were obtained from blood samples: blood lead (PbB), free erythrocyte protoporphyrins (FEP), and aminolevulinic acid dehydrase (ALA-D). Two measures were based upon urine samples: urine lead (PbU) and delta-aminolevulinic acid (δ -ALA). Blood and urine samples were obtained individually from each worker at the time of performance testing. The subject's blood sample was collected in a 10ml, lead-free vacutainer (Becton-Dickinson Co.) from which approximately 1.0ml was immediately decanted into a 12 x 75mm polypropylene test tube. This small test tube of blood was immediately frozen to inhibit the breakdown of ALA-D, while the remaining sample of vacutainer blood was sealed and stored under normal refrigeration until delivery to the Laboratory.

The 150ml polypropylene container used for each subject's urine sample was prepared in the following manner: the container was washed, rinsed with distilled water, washed with 1:1 nitric acid, rinsed several times with glass-distilled/deionized water and air dried. To this container was added 1.0ml of glacial acetic acid which brought the urine pH to approximately 2.0, thereby inhibiting the breakdown of δ -ALA.

Upon receipt of the blood and urine samples by the Laboratory, all samples were refrigerated, with the exception of the small test tube of frozen blood which was placed in a freezer. A brief description of the methodology involved in each of these determinations is provided in the paragraphs which follow; detailed descriptions of the clinical methodologies are provided in Repko, *et al.* (1975, Appendix E). Each of the measures chosen is based on its respective sensitivity to long- or short-term exposure to inorganic lead.

Blood hematocrits were determined in duplicate using standard hematocrit capillaries. The average value of the duplicate analysis was reported for each sample. ALA-D was determined according to the method of Granick and Mauzerall (1958) as modified by Lichtman and Feldman (1963). Basically the method consisted of adding an excess of the ALA-D substrate, delta-aminolevulinic acid (ALA), incubating the mixture for one hr and then colorimetrically determining the amount of porphobilinogen which had been enzymatically synthesized. The extent of porphobilinogen formation then served as an indicator of the enzymatic activity of the blood ALA-D. The determination of urine δ -ALA was performed by the method of Mauzerall and Granick (1956) as modified by Davis and Andelman (1967). This consisted of double ion-change column chromatography to remove interfering substances, followed by a colorimetric analysis of the ALA. Free erythrocyte protoporphyrins (FEP) in the blood were determined by the conventional ethyl acetate-acetic acid and HCL method of Piomelli, Davidow, Guinee, Young, and Gay (1973).

Blood-lead determinations were performed according to the method of Hessel (1968). This consisted of extracting the blood lead, which had previously been chelated and thus rendered soluble, into an organic solvent. The organic solvent in turn was analyzed for its lead content by atomic absorption spectroscopy. The method of Yeager, Cholak, and Henderson (1971) was selected

for urine-lead determination because of its utilization of atomic absorption spectroscopy and because this particular method eliminated interferences from iron, copper, or zinc. As with the blood lead analysis, this consisted of extracting chelated lead into an organic solvent followed by atomic absorption determination of the resulting lead concentration.

BEHAVIORAL TEST BATTERY

A total of six behavioral tests and a comprehensive personal-data questionnaire were selected for use in this research. Each test was chosen on the basis of its potential usefulness in measuring a behavioral or neurological function that might be expected to change as a result of low-level absorption of inorganic lead. A review of the literature dealing with the behavioral and neurological effects of inorganic lead (see Repko & Corum, 1976), as well as information gained from previous behavioral studies of inorganic lead (see Repko, *et al.*, 1975), provided a set of tests which would potentially be sensitive to functional changes occurring during low level exposure and absorption of inorganic lead.

The core of the test battery consisted of four neuromuscular tasks, three of which have been used previously in studies of inorganic lead. These particular tasks involved measures of muscular strength, endurance, and recovery, tremor, and eye-hand coordination. The fourth task involving neuromuscular functioning consisted of a visual reaction time test. This test was chosen on the basis of previous data obtained in this laboratory (see Repko, *et al.*, 1975) and in the literature (see Boyadzhiev, Stoev, & Petkov, 1962) which indicate that neurological effects can be measured indirectly in tasks that require rapid motor responses. In addition, two tests of auditory functioning were administered; these included tests of auditory thresholds obtained via both air conduction and bone conduction, as well as a test of tone decay. The final test selected for inclusion in the test battery was the Clinical Analysis Questionnaire (CAQ). This test was supplemented with the Marlowe-Crowe Scale, which is typically used in conjunction with the CAQ. These tests were selected in order to assess the subjective states and personality characteristics of the workers involved in the study. Table 1 provides a summary of the aforementioned six tests in terms of the functional category evaluated by the test, name of the test, and the approximate time required for its administration.

Strength, Endurance, and Recovery

This task utilized a portable ergometric system which provided for a dynamic input from the worker and a dual output of information. To perform this task, the worker was seated in a chair so that a handgrip dynamometer was positioned on the preferred side. The handgrip was adjusted at approximately the height of the worker's knee such that his forearm and upper arm formed a 90° angle at the elbow (see Figure 5). Each worker's strength, endurance, and recovery (SER) was then recorded according to Caldwell's (1963) procedure: the subject was required to pull his maximum strength; then, exactly 1min later, he was required to pull, for as long as possible, a load equal to 50 percent of the original maximum strength; this was followed by a 1min rest and then a final maximum strength response. Detailed descriptions of the instructions are provided elsewhere (see Repko, *et al.*, 1976, Appendix E).

Table 1
Summary of Tests Included in the
Behavioral Test Battery

Test Area	Specific Instrument	Functional Category Tested	Time Required
Muscular strength, endurance, & recovery	SER apparatus	Neuromuscular	10min
Tremor	Vertical movement transducer	Neuromuscular	10min
Eye-hand coordination	Michigan eye-hand coordination test	Neuromuscular	5min
Visual reaction time	Visual RT apparatus	Neuromuscular	5min
Auditory acuity and tone decay	Bekésy Audiometer	Sensory	40min
Subjective feelings & personality	CAQ and Marlow-Crowne Scale	Psychological	60min (approx.)

Tremor

The test of resting tremor was interrelated with the SER tests. For this tremor task, the worker was seated in a chair and positioned with the preferred shoulder directly in front of the finger-tremor apparatus so that the worker had room to partially extend (in an approximate angle of 120° at the elbow) the preferred arm. The worker then placed his finger in a finger coupler, with the wrist resting on a platform in front of and slightly below the coupler. Once the worker appeared ready, the experimenter initiated a 1min trial by starting an FM recorder. At the end of 1min, the experimenter then turned off the recorder and told the worker to prepare to repeat the trial using the non-preferred hand. Once the worker was properly positioned, the recorder was started and the trial was repeated as before. At the end of these pre-test trials, the worker performed the SER tasks. Immediately upon cessation of the SER task, the tremor task was repeated in exactly the manner and order (preferred hand, non-preferred hand) in which it was performed before. A view of the worker performing the tremor task is shown in Figure 6; detailed descriptions of the task are given in Repko, *et al.* (1976), Appendix E.



Figure 5. View of arm position during the test of strength (SER); both the handgrip dynamometer and subject display meter can be seen in this illustration.

Eye-hand Coordination

The test of eye-hand coordination used in this study, developed by Pooch (1967), has been used in studies of occupational exposure to low levels of inorganic mercury (Chaffin, Dinman, Miller, Smith & Zontine, 1973), inorganic lead (Repko, *et al.*, 1975), and methyl chloride (Repko, *et al.*, 1976); the test is adequately described in these publications and will be briefly described here. The apparatus consisted of a hole plate (on which interconnect-lines from a maze pattern which the workers were required to follow in performing the task), a sounding board, a contact microphone, a tape recorder, and a stylus. To perform this task, the worker was seated facing the apparatus and was required to grasp the stylus in his preferred hand. He was then instructed to insert the stylus into each of the 119 holes in the plate in the order indicated by the black lines, proceeding as quickly as possible without missing any holes; this procedure was then repeated for the non-preferred hand. The electrical contact of the stylus striking the plate beneath the maze created a square-wave pulse which was recorded on an AM tape recorder; the latency between successive pulses was later scored by use of a digital computer. A worker performing the eye-hand coordination test is shown in Figure 7.

Visual Reaction Time

A simple visual reaction time task was specifically designed for use in this study; the response involved the direct motor control of the fifth

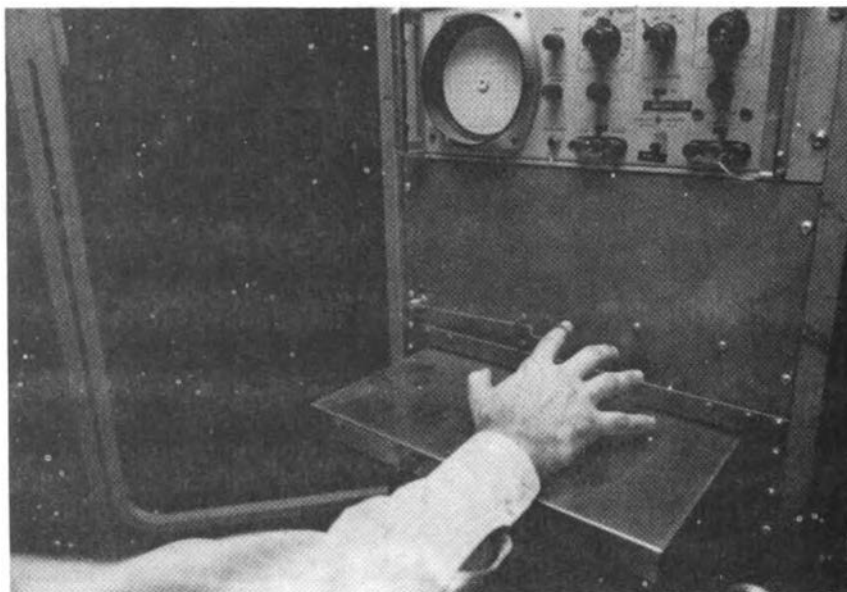


Figure 6. View of workers performing the test of tremor showing proper hand position, figure-cup-transducer assembly, and oscilloscope.

finger (fourth digit) by the ulnar nerve. The worker's task was to respond (from a sitting position) as quickly as possible to the onset of a red light-emitting diode (LED) by pushing a microswitch located on the table in front of him. A signal consisted of the illumination of the LED to a level of 10ftL (34.25cd/m^2) for a period of 500msec. Ten signals each were presented at random for trials with both the right and left hands (five practice signals for each hand were also administered). The response involved the movement of the fifth finger through an arc of 2.5cm with the remaining digits, hand, and arm in a stabilized or non-movable position. The response button consisted of a 2.5 x 5cm plexiglass plate attached to a microswitch. The proper position of a worker's arm and hand for performing this task is shown in Figure 8.

Auditory Acuity and Tone Decay

Two assessments of auditory functioning were made for each worker; these included pure-tone threshold tests via air conduction in both the right and left ears and via bone conduction and a test of tone decay. Threshold values were acquired by means of a Grason-Stadler Model 1702 Audiometer; this unit met all requirements specified by the American National Standards Association for a fixed-frequency, wide-range, pure-tone audiometer (see Figure 9). The matched Telephonic TDH-50 earphones, supplied by Grason-Stadler, were calibrated for this system. Individual audiometric tests were administered in a portable Industrial Acoustics Company single-wall sound booth (with 51bB

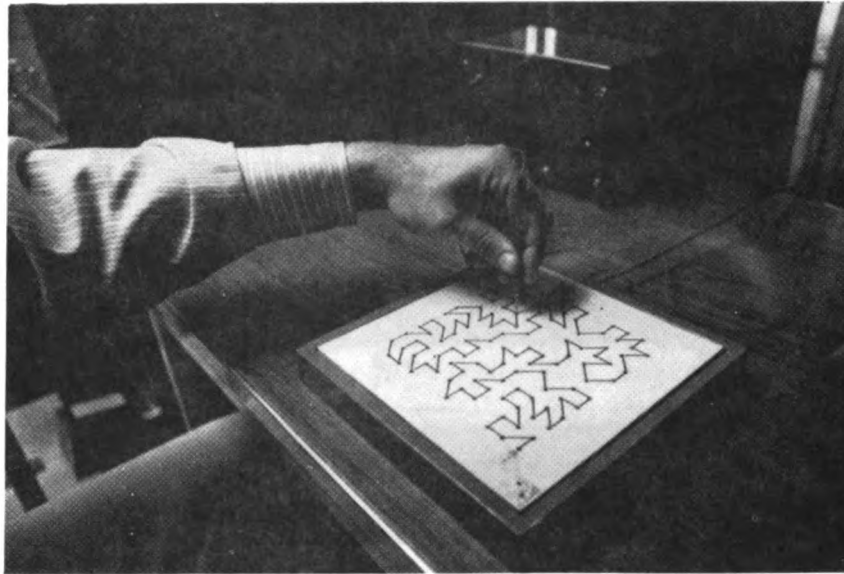


Figure 7. View of worker performing the eye-hand coordination task.

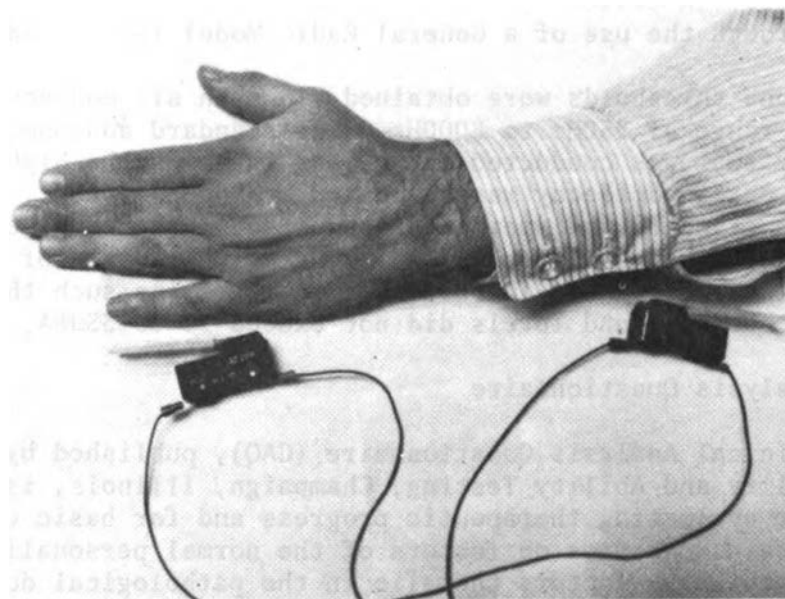


Figure 8. View of worker's arm and hand in proper position for performing the visual reaction time task.

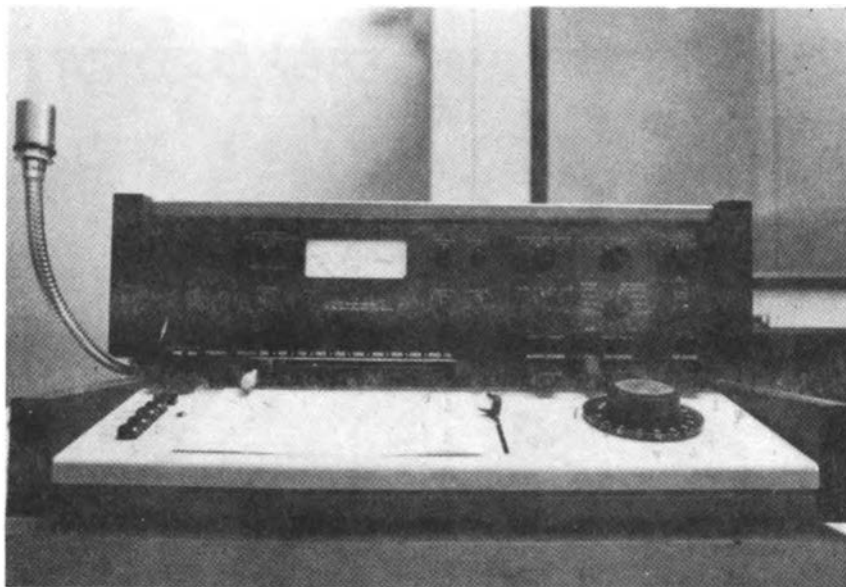


Figure 9. Front view of the Grason-Stadler Model 1702 Audiometer.

noise reduction at 2000Hz). The audiometric System was calibrated prior to each test session; the internal system calibration circuitry permitted the checking of the calibration of the entire pure-tone audio system, including the earphones. In addition, the calibration of the audiometric system was verified through the use of a General Radio Model 1933 Sound Analysis System.

Pure-tone thresholds were obtained via both air and bone conduction through the range of 250Hz to 8000Hz using standard audiometric techniques. A tone-decay test was conducted on the ear in which the highest threshold values were obtained; decay was evaluated at 500Hz, 2000Hz, 4000Hz, and 8000Hz. The standard audiometric procedures and the tone-decay test are adequately described by Green (1972). During the course of the study the sound booth was positioned in relatively quiet areas such that the internal (within the booth) sound levels did not exceed 32 to 35dBA.

Clinical Analysis Questionnaire

The Clinical Analysis Questionnaire (CAQ), published by the Institute for Personality and Ability Testing, Champaign, Illinois, is an instrument designed for evaluating therapeutic progress and for basic clinical research. The CAQ contains measures on factors of the normal personality plus scales and items for twelve factors that lie in the pathological domain. Since the *Manual for the Clinical Analysis Questionnaire* (Dehees & Cattell, 1971) fully deals with these normal personality and pathological factors, it is not necessary to expand further on these here. Administered in conjunction with the

CAQ was the Marlow-Crowne Scale which contains a list of 33 statements concerning personal attitudes and traits. The purpose of this test is to determine the general veracity, or lack thereof, in responding to the items contained in the CAQ (as well as the test situation generally). The Scale specifically attempts to isolate individuals who describe themselves in favorable, socially desirable terms in order to achieve the approval of others. A full description of the Marlowe-Crowne Scale and the purposes for which it is intended can be found in Crowne and Marlowe (1964).

The workers were administered the Marlowe-Crowne Scale followed by the CAQ (Part I) during their first test session; when the workers returned for the neurological examination they completed the CAQ (Part II). A CAQ test booklet was furnished to each worker along with a standard Op-Scan answer sheet in which the responses were recorded; responses to the Marlowe-Crowne Scale were recorded directly in the test booklet.

RESULTS

STATISTICAL TECHNIQUES

In the analysis of the study data, four primary techniques were employed, namely, (a) Chi-square, (b) correlation, (c) independent groups *t*-test, and (d) the *Mann-Whitney U*. The Chi-square (χ^2) was used for analytical comparison of the results obtained from the neurological examination and the personal-data questionnaire. Since these data are discrete data (data merely enumerated or counted) and nominally classified, χ^2 is the appropriate statistic. In the analysis of the remaining behavioral and neurological measures, the first step was the computation of the coefficients of correlation (*Pearson r*) between the neurobehavioral measures and the five biomedical indicators of lead exposure and absorption. The *r* statistic was computed from the data of the experimental group and was considered to be significantly different from zero if the chance probability was less than or equal to five percent.

The experimental and control groups were compared on each of the neurobehavioral measures by means of an independent groups *t*-test. Although the *t* statistic does not require knowledge of the population variances, it does assume population homoscedasticity. For most real data, the variances for both sample groups are about equal ($\sigma_1^2 \approx \sigma_2^2$). Since the *t* statistic is quite robust with respect to dissimilar sample variances, there usually is no need to be concerned unless one group's variance is three or four times the size of the other's (Senter, 1969). A parametric solution to the unequal variance problem was proposed by Behrens and enlarged upon by Fisher (1935). The resulting test is known as the *Behrens-Fisher test*. Another solution to the same problem has been proposed by Cochran and Cox (1957). The *Cochran-Cox t'* test is somewhat conservative in that the value of *t'* required for significance is too high (see Bliss, 1967, for a general discussion of two-group comparisons with equal and unequal variances). Because both of these parametric *t'* tests are less powerful than the non-parametric alternative, the *Mann-Whitney U*, the *U* statistic was selected for analysis of those study data in which the sample variances were very divergent and the assumptions of homogeneity would not be met.

DESCRIPTION OF STUDY POPULATION

A total of 140 industrial workers served as subjects in one of two groups, namely, (a) the experimental group--85 workers who were exposed to inorganic lead in their jobs, and (b) the control group--55 workers who had no present or known prior exposure to lead or other toxic agents. This final sample involved 140 workers, although 157 workers volunteered for participation in the study. Three lead-exposed workers who did not want to participate in the medical aspects of the evaluation withdrew; the data of five control workers under age 20 and who completed all of the tests were randomly deleted from the sample population since there was an excess of volunteer workers in the age group of 16 to 20yrs; and nine workers were initially excused from participation because they did not meet criteria concerning prior exposure to other toxic agents, had a history of excessive blood lead, or their age was

Table 2
Statistical Summary of Sample Group
by Age and Sex

Measure	<u>Experimental Group</u>		<u>Control Group</u>	
	Frequency	Percent	Frequency	Percent
<hr/>				
<u>Age</u>				
16 to 20 years	1	1.18	2	3.64
21 to 25 years	7	8.23	5	9.09
26 to 30 years	15	17.65	9	16.36
31 to 35 years	17	20.00	9	16.36
36 to 40 years	18	21.18	9	16.36
41 to 45 years	10	11.76	8	14.55
46 to 50 years	7	8.23	6	10.91
51 to 55 years	4	4.71	4	7.27
56 to 60 years	3	3.53	3	5.46
61 to 65 years	3	3.53	0	0.00
Total	85	100.00	55	100.00
Mean Age	37.37 years		37.20 years	
Range	19 to 64 years		18 to 57 years	
Standard Error	1.097		1.410	
t (df = 138)	0.0929 (ns)			
<u>Sex</u>				
Males	81	95.28	46	83.64
Females	4	4.72	9	16.36
Total	85	100.00	55	100.00

inappropriate for their inclusion in the control group. For a variety of reasons, however, it was impossible to collect a complete set of data from each of these subjects. Some of the analyses described below include data from fewer than 85 experimental or 55 control subjects. In those cases where data are missing, the analyses were computed on the basis of all available data, and a notation has been made as to the number of observations (or degrees of freedom, df) included in each analysis.

At the outset of the study, an attempt was made to match the control group to the experimental group as closely as possible according to age, sex, height, weight, and educational level. Since subjects were selected strictly on a voluntary basis, perfect matching along all of these dimensions was not totally achieved. Statistical summaries of the experimental and control groups are provided in Tables 2, 3, and 4.

Table 3
Statistical Summary of Sample Group
by Height and Weight

Measure	Experimental Group ¹	Control Group ²
<u>Height</u>		
Mean	174.48cm	174.20cm
Range	150 to 188cm	150 to 191cm
Standard Error	0.876cm	1.280cm
<i>t</i> (df = 128)	0.1903 (ns)	
<u>Weight</u>		
Mean	79.44kg	76.68kg
Range	50 to 120kg	50 to 113kg
Standard Error	1.273kg	1.798kg
<i>t</i> (df = 138)	1.2867 (ns)	

¹N = 85; ²N = 55

Presented in Table 2 are the data involving the age and sex of the sample population. It can be seen that the number of controls is distributed in a similar manner to that of the experimentals across the ten 5yr age groups. The range of ages in the experimental group is 19 to 64yrs and in the control group it is 18 to 57yrs. Overall, the workers in the control group represented a slightly younger group, but the mean age difference was only 0.17yr, or approximately 2mos. This difference in age between the two groups is not statistically significant. It can also be seen in Table 2 that the number of women in the total sample was small ($N = 13$). Moreover, the proportion of women in the control group is considerably larger (16.36%) than in the experimental group (4.72%).

The statistical summaries of the sample population for height and weight are presented in Table 3. It can be seen in both cases that the differences between the two groups were not statistically significant. The mean heights of the two groups differed by less than 0.3cm and the difference in weight was approximately 2.8kg. The ranges in both height and weight were also very similar between the two groups.

The statistical summary of the educational background of the sample population is presented in Table 4. The distribution includes elementary, high school, and college education; no workers had less than a sixth grade education. In both groups the most frequently completed grade was twelve or

Table 4
Statistical Summary of Sample Group
by Level of Education and Duration of Employment

Measure	Experimental Group		Control Group	
	Frequency	Percent	Frequency	Percent

<u>Level of Education</u>				
6th grade	0	0.00	1	1.82
7th grade	4	4.71	4	7.27
8th grade	13	15.29	0	0.00
9th grade	6	7.06	6	10.91
10th grade	6	7.06	4	7.27
11th grade	4	4.71	2	3.64
12th grade	45	52.94	24	43.64
Freshman (college)	2	2.35	3	5.45
Sophomore (college)	4	4.71	3	5.45
Junior (college)	0	0.00	0	0.00
Senior (college)	1	1.17	8	14.55
Total	85	100.00	55	100.00
Mean Grade Level	Grade 10.92		Grade 11.76	
Mode	Grade 12		Grade 12	
Range	7th to College Graduate		6th to College Graduate	
Standard Error	0.213		0.347	
t (df = 138)	2.2467 ($p < .05$)			

<u>Duration of Employment</u>		
Mean Months	104.66 Months	57.15 Months
Range	3 to 408 Months	1 to 312 Months
Standard Error	9.071	9.520
t (df = 138)	3.4593 ($p < .05$)	

completion of high school (mode = 12). However, the range of educational levels attained was from seventh grade to completion of college in the experimental group and from the sixth grade in the control group. The mean difference in level of education between the two groups was less than one yr of education and this difference was significant ($p < .05$). This difference may be attributable to the larger proportion of college graduates in the control group (14.55%) as opposed to the experimental group (1.17%). Also given in Table 4 are the mean durations of employment for both groups. It is

Table 5

Coefficients of Correlation (r) between
Primary Demographic Measures and Biomedical
Indices of Lead

Measure	Correlations (r) ¹				
	PbB	ALA-D	FEP	PbU	δ -ALA
Age	-.0987	-.0748	.0576	-.0292	.0477
Height	.0698	-.1132	.0601	.0519	.0789
Weight	.0351	-.1933	.2398*	.1155	.0837
Education	-.0400	.0826	-.0532	-.1499	-.0715
Employment	.1130	-.1023	.0624	.0702	-.0201

¹76 \leq N \leq 80; * p < .05

evident that the lead workers varied in their length of employment from three to 408mos, with an average duration of 104.66mos; the control group had a mean duration of 57.15mos and ranged from one to 312mos of continuous employment. This difference in duration of employment was significant (p < .05).

The coefficients of correlation between the primary demographic measures described above and the biomedical indices of lead exposure and absorption for the experimental group are given in Table 5. Except for a significant correlation between weight and free erythrocyte protoporphyrin (FEP) (p < .05), which would be expected to occur by chance in a group of 25 independent correlations, none of the coefficients of correlation between the biomedical indices and the demographic measures were statistically significant. In particular, despite the fact that the duration of employment, and therefore exposure, ranged up to 408mos (34yrs), no relationship existed between these biomedical measures and the length of employment. Similarly, there was no significant relationship for either of the measures of education or age and these indices of absorption and exposure. Considering that the sample of lead-exposed workers represented low-level exposure as determined *a priori* by their previous blood-lead histories, these results are not unexpected.

The results of the biomedical determinations are summarized in Table 6 and, indeed, characterize a sample group exhibiting low-level lead exposure and absorption. The mean blood-lead (PbB) level for the lead-exposed workers was 46.0ug/100ml while the mean for the control group was 18.0ug/100ml; this latter value is slightly higher than would be expected in the normal population, although it is still well within the range considered normal (i.e., below 20 to 40ug/100ml, see Zielhuis, 1977). Mean aminolevulinic acid dehydratase (ALA-D) activity of 28.8units/liter rbc indicates that the formation of ALA-D was suppressed in the experimental group; in the control group, the

Table 6
Statistical Summary of Sample Group
for Biomedical Indices of Level

Measure	Experimental Group ¹	Control Group ²
<u>Hematocrit</u>		
Mean	44.0	44.0
Standard Error	0.4	0.6
<u>Blood Lead (PbB)</u>		
Mean	46.0ug/100ml	18.0ug/100ml
Standard Error	1.7	1.0
<u>Aminolevulinic Acid Dehydrase (ALA-D)</u>		
Mean	26.8units/liter rbc	62.7units/liter rbc
Standard Error	1.5	3.2
<u>Free Erythrocyte Protoporphyrin (FEP)</u>		
Mean	115.2ug/100ml	22.6ug/100ml
Standard Error	9.1	1.1
<u>Urine Lead (PbU)</u>		
Mean	100.1ug/liter	14.0ug/liter
Standard Error	8.7	2.0
<u>-Aminolevulinic Acid (-ALA)</u>		
Mean	0.7mg/100ml	0.3mg/100ml
Standard Deviation	0.8	0.2

¹_N = 85; ²_N = 55

ALA-D activity mean of 62.7 units/liter rbc does not indicate that the formation of this enzyme was inhibited for this group. The determinations for free erythrocyte protoporphyrin (FEP) produced a mean level of 115.2ug/100ml in the experimental group and a mean of 22.6ug/100ml in the control group. The mean urine-lead level (PbU) of 100.1ug/liter for the experimental group also indicates low-level lead absorption. In the control group, the mean was 14.0ug/liter. The final biomedical measure, urinary δ -aminolevulinic acid (δ -ALA) further demonstrates the early inhibition of ALA-D in the low-level range; in the experimental group mean δ -ALA was 0.7mg/100ml and in the control group the mean was 0.3mg/100ml. Although the hematocrit is not a biomedical indicator of low level lead exposure or absorption, it was determined for each

Table 7

Intercorrelations of Hematocrit, Blood Lead, Blood ALA-D,
FEP, Urine Lead, and Urine ALA

	Hematocrit	Blood Lead	Blood ALA-D	FEP	Urine Lead	Urine ALA
Hematocrit	1.0000	.1674	-.1616	-.2163*	-.0810	-.0114
Blood Lead		1.0000	-.2983***	.3623***	.5795***	.4632***
Blood ALA-D			1.0000	-.2679**	-.2008*	-.3315***
FEP				1.0000	.1825	.4497***
Urine Lead					1.0000	.4166***
Urine ALA						1.0000

¹76 < N ≤ 85; *p < .05; **p < .01; ***p < .001

worker. The values given in Table 6 show that the mean hematocrits for both groups are within a normal range and that there is little difference between the two sample groups.

The degree of relationship among these measures was determined by computing the intercorrelations; these results are presented in Table 7. It is evident from these data that most all of these measures intercorrelate well. Since PbB and PbU are both direct indicators of exposure, it is not surprising that the highest (positive) correlation, $r = .5795$ ($p < .001$), was obtained between these measures. Moreover, PbB correlated significantly with each of the three remaining biomedical indices ($p < .001$), although the correlation between FEP and PbB was lower than that reported by others. For example, Tomokumi (1975) reported a correlation of 0.63 between PbB and the logarithm of FEP. The correlations between δ -ALA and other biomedical indices were also significant ($p < .001$). Although significant, the negative correlation between FEP and ALA-D was rather low, $r = -.2679$ ($p < .01$), and the correlation between FEP and PbU was not significant. Similarly, the correlation between PbU and ALA-D was low, $r = -.2008$ ($p < .05$). With the exception of the significant negative correlation between the hematocrit and FEP ($p < .05$), the correlations between the hematocrit and each of the remaining biomedical indices are not significant.

NEUROLOGICAL EVALUATION RESULTS

Provided in Table 8 is a summary of the results of the neurological examination. To simplify the results of the neurological examination, the presence or absence of an abnormality was determined for (a) visual acuity, (b) tremor (eyes open and closed), (c) vibration sense, (d) position sense, (e) auditory air conduction, and (f) deep tendon reflexes. An overall impression of the neurological evaluation is also provided. Review of the data in Table 8 indicates that there was very little difference between the experimental and control groups in each of the categories evaluated. Moreover, the overall impression of the neurological examination of the workers was that 57.65 percent of exposed workers and 60.00 percent of the control workers exhibited some abnormal trait or combination of abnormal traits. A Chi-square $X^2 = .0762$, which for one degree of freedom was not significant. The experimental (and control) group was asymptomatic with respect to neurological signs and symptoms characteristic of lead poisoning. Those signs and symptoms tabulated in Table 8 do not differ from what would be expected to occur in a random sample of a population of industrial workers with a distribution of age from 18 to 64yrs, evaluated after 8hrs of work (see Repko, *et al.*, 1977).

In both groups, the predominant neurological deficit which occurred in the absence of all other symptoms was decreased visual acuity ($N = 46$). In the experimental group, 18 workers exhibited decreased visual acuity alone, while others exhibited this deficit in combination with tremors ($N = 2$), with tremors and decreased vibration sensation ($N = 1$), with decreased vibration sensation ($N = 2$), or with hearing difficulties ($N = 2$). Twelve of the lead-exposed workers exhibited fine terminal tremor (with eyes open and/or closed, finger-to-nose test), while the remaining workers exhibiting tremor did so in combination with symptoms of decreased visual acuity ($N = 2$), with decreased visual acuity and decreased vibration sensation ($N = 1$), with decreased vibration sensation ($N = 1$), or with impaired position sensation ($N = 1$). In this same group of workers, eight exhibited decreased vibration sensation alone

Table 8
Summary of Results of the Neurological Examination

Neurological Criteria	Experimental Group		Control Group	
	Frequency	Percent	Frequency	Percent
<u>Decreased Visual Acuity</u>				
Abnormal	25	29.41	21	38.18
Normal	60	70.59	34	61.82
<u>Tremor</u>				
Abnormal	17	20.00	9	16.36
Normal	68	80.00	46	83.64
<u>Decreased Vibration Sensation</u>				
Abnormal	13	15.29	12	21.82
Normal	72	84.71	43	78.18
<u>Impaired Position Sensation</u>				
Abnormal	2	2.35	2	3.64
Normal	83	97.65	53	96.36
<u>Hearing Difficulty</u>				
Abnormal	5	5.88	6	10.90
Normal	80	94.12	49	89.10
<u>Pathological Reflexes</u>				
Abnormal	1	1.76	0	0.00
Normal	84	98.24	55	100.00
<u>Neurological Impression</u>				
Abnormal	49	57.65	33	60.00
Normal	36	42.35	22	40.00

and two showed some hearing difficulties without other signs or symptoms. One worker showed decreased vibration sensation, impaired position sensation, and hearing difficulties; another had equivocal plantar extensor response (Babinski) and present suck and snout reflexes.

In the control group, 11 workers had decreased visual acuity alone. Other workers exhibited decreased visual acuity in combination with symptoms of tremor ($N = 3$), of tremor and decreased vibration sensation ($N = 1$), of decreased vibration sensation ($N = 3$), of hearing difficulties ($N = 2$), and of tremor, decreased vibration sensation, and impaired position sensation ($N = 1$). Two workers exhibited tremor alone, one worker exhibited tremor with decreased vibration sensation, and another exhibited tremor along with hearing difficulties. Four workers showed decreased vibration sensation alone, and one worker exhibited this symptom in conjunction with hearing

difficulties, and another exhibited decreased vibration sensation with impaired position sensation. Finally, two workers exhibited hearing difficulties without other neurological signs or symptoms.

ELECTRONEUROMYOGRAPHIC EVALUATION RESULTS

The electroneuromyographic (ENMG) examination consisted of an electromyographic evaluation (EMG) of two muscles innervated by the ulnar nerve, the first dorsal interosseous and the abductor digiti minimi, and an evaluation of the conduction velocities (NCV) of the median, ulnar, posterior tibial, and deep peroneal nerves. In only two of the workers evaluated were EMG abnormalities noted. One lead-exposed worker showed positive sharp waves and fibrillations in the EMG; and, one control worker also showed positive sharp waves. However, in each case, the abnormalities were borderline and not considered significant, since the deficits in these two workers were not confirmed by any related abnormalities developed from the neurological evaluation of the motor system. In all other cases, the EMG was normal; there was no evidence of a diminished number of motor units in maximal contraction, no evidence of abnormal insertional activity, and no evidence of fibrillations, fasciculations, or positive sharp waves.

Presented in Table 9 are the analyses of the temperature data obtained at the time of the examination of the conduction velocities of the right extremities. As noted earlier in this report, both core and surface temperatures were monitored throughout the course of the ENMG. It should be clear from the data presented in this table that there were no significant differences in mean temperature between the two groups overall. This is true not only in terms of core temperature but also in terms of the distal surface temperatures of both limbs and of the proximal surface temperatures of the arm. Only the proximal surface temperatures of the posterior tibial and deep peroneal nerves showed significant differences between the study groups ($p < .05$). Despite these specific differences, however, the core temperatures should be regarded as more significantly indicative of leg/nerve temperature because of the relatively large mass of body tissue in the legs as opposed to the arms, especially at the proximal location. Finally, the differences in temperature at the proximal location represent differences of less than 1°C.

Notwithstanding the fact that core and surface temperatures were adequately controlled, the conduction velocities obtained from each worker in both groups were corrected to a standard limb temperature of 31.5°C. DeJesus, Hausmanowa-Petrusewicz, and Barchi (1973) provide convincing evidence that nerve conduction velocities decrease with limb temperature. These authors derived a single temperature correction formula, expressed as a semilogarithmic function, which can be used in both sensory and motor conduction velocity studies; the correction formula applied to the conduction velocity data obtained in this study was:

$$Y_2 = Y_1 e^{(M_2 \Delta T)}$$

where Y_2 is the corrected velocity at the standard temperature, Y_1 is the measured velocity at the experimental temperature, e is the base of the natural log system, $M_2 = 0.0419$, and ΔT is the difference in degrees centigrade

Table 9

Group Means, Standard Errors, and
t-Values for Limb Temperatures (°C)

Measure	Means		df	t
	Exp.	Con.		
<hr/>				
<u>Right Arm¹</u>				
Core	34.85 (0.124)	34.86 (0.126)	119	0.0888
Distal Median	31.33 (0.107)	31.30 (0.121)	122	0.1889
Proximal Median	32.16 (0.080)	32.15 (0.107)	122	0.0414
Distal Ulnar	31.59 (0.115)	31.52 (0.139)	121	0.4017
Proximal Ulnar	31.84 (0.093)	31.76 (0.125)	121	0.4755
<u>Right Leg²</u>				
Core	32.95 (0.172)	33.44 (0.203)	65	1.8299
Distal Tibial	28.65 (0.304)	29.31 (0.319)	67	1.4956
Proximal Tibial	30.88 (0.186)	31.56 (0.177)	67	2.6039*
Distal Peroneal	29.62 (0.210)	29.49 (0.295)	66	0.3536
Proximal Peroneal	30.83 (0.160)	31.52 (0.152)	66	2.9661*

¹Exp: $77 \leq N \leq 78$, Con: $N = 46$; ²Exp: $36 \leq N \leq 37$, Con: $N = 32$; * $p < .05$

between the standard (i.e., 31.5°C) and experimental temperature (for a full discussion of the derivation and application of this formula, see deJesus, *et al.*, 1973).

The results of the analyses for the nerve conduction velocities of the median, ulnar, posterior tibial, and deep peroneal nerves are given in Table 10. For each NCV measure are given the group means, the number of observations, the *U*-values, and the probabilities associated with values as extreme

Table 10

Group Means, the *U* Statistic, *z* Scores, and Associated Probabilities for Nerve Conduction Velocities of the Right Limbs

Conduction Velocity Measures	Means		<i>U</i>	<i>z</i>	<i>p</i> ¹
	Exp.	Con.			
Median MCV (m/sec) (<i>N</i>)	53.41 (80)	59.89 (43)	953.0	4.068	.00003
Ulnar MCV (m/sec) (<i>N</i>)	55.56 (79)	64.47 (44)	608.0	5.962	.00003
Ulnar SCV (m/sec) (<i>N</i>)	56.43 (21)	62.96 (29)	194.0	2.172	.0150
Ulnar CVSF (m/sec) (<i>N</i>)	47.98 (36)	45.72 (29)	445.5	1.010	.1562
Tibial MCV (m/sec) (<i>N</i>)	50.46 (37)	55.49 (28)	290.0	3.020	.0013
Peroneal MCV (m/sec) (<i>N</i>)	49.33 (36)	54.23 (30)	313.0	2.923	.0018

¹Probability values from Siegel (1956), p. 247.

as the observed values of *z*. The Mann-Whitney *U* was chosen as the preferred statistic because of differences in the variances between the two study groups. Since slight NCV differences do occur between males and females and there was a higher percentage of females in the control group, the NCV analyses were performed on the data both with and without the data from the female sample. The results of both analyses did not differ in outcome; therefore, in the results described below, the data of both males and females were pooled.

Since the conduction velocities were not determined for all the experimental and all the control workers, the total number of observations is less than the total sample of 140 workers. Insufficient time was available to obtain, from the entire subject population, measures of the SCV and CVSF of the ulnar nerve and to obtain the MCV of the tibial and peroneal nerves. Moreover, a great deal of time was devoted to verification of instrument calibration (including time-base) and to cooling or warming the extremities; both of these factors decreased the time available for measuring all six conduction velocities on all workers.

From the data presented in Table 10 and in Figure 10, it is clear that the MCV of the ulnar and median nerves was slower among lead-exposed workers than controls. The differences of about 6.5m/sec for the median nerve and of

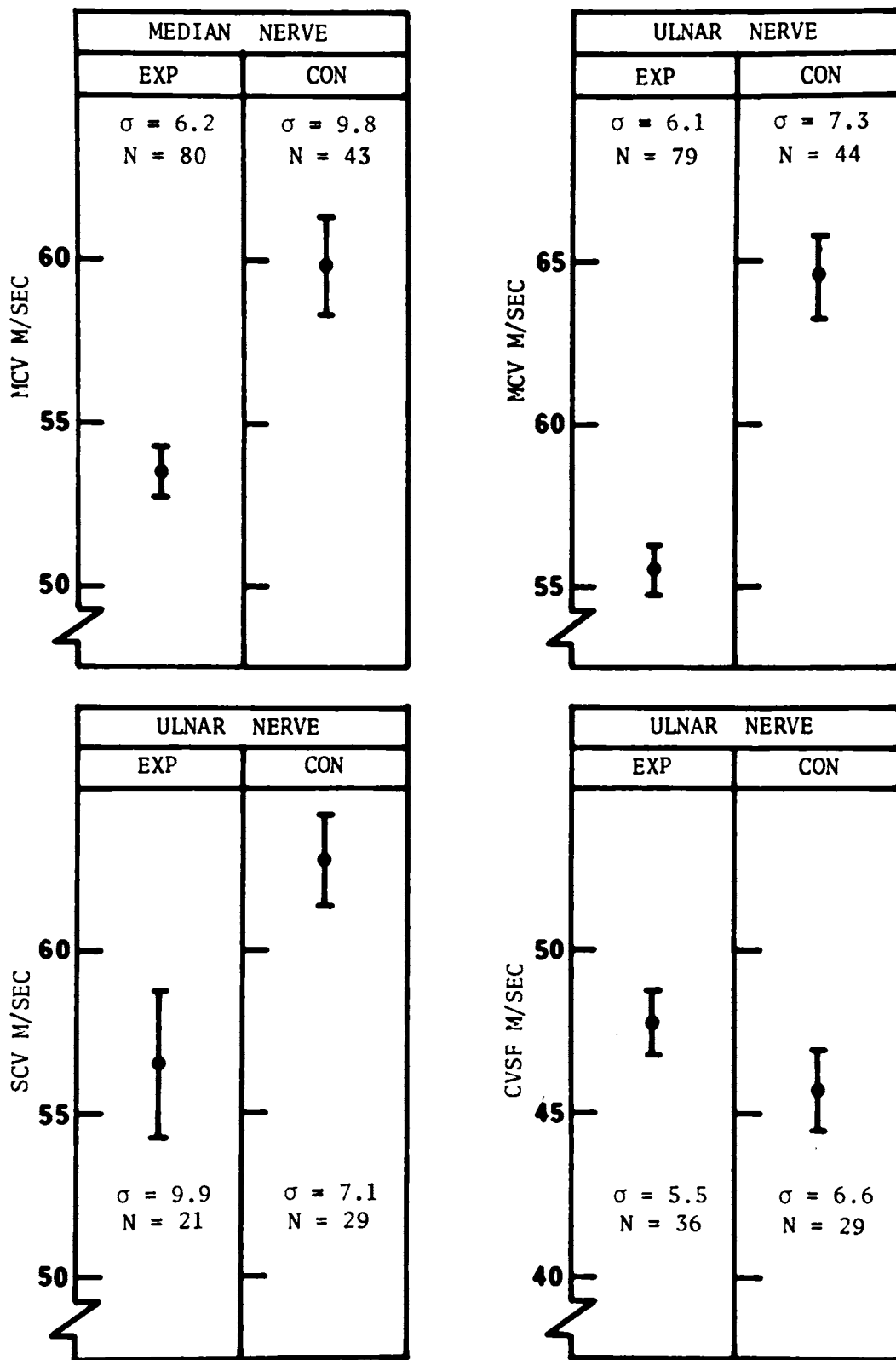


Figure 10. The means, standard errors (plotted), and standard deviations (σ) of the NCV measures of the right arm.

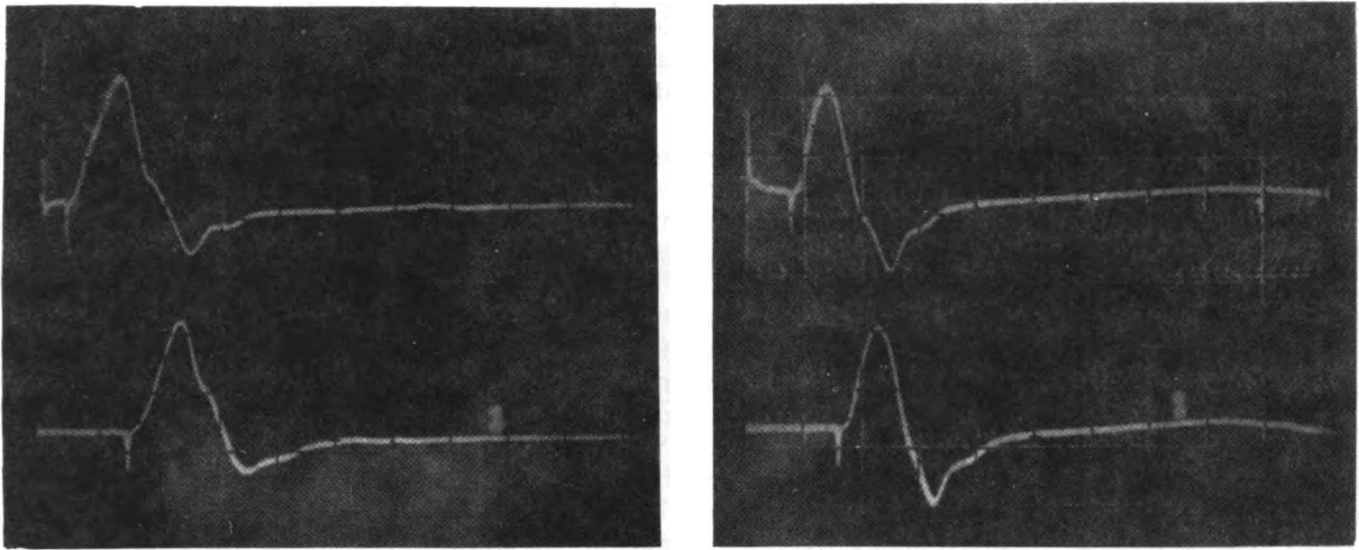


Figure 11. Distal action potential (upper trace) and proximal action potential (lower trace) for the median (left frame) and ulnar nerves (right frame).

about 8.9m/sec for the ulnar nerve are statistically significant ($p < .00003$). Examples of distal and proximal action potentials, well defined in amplitude and configuration, for both the median and ulnar nerves, respectively, are shown in Figure 11. In this figure, the distal latency of the median nerve action potential was 4.0msec and the proximal latency was 8.4msec; over a distance of 25.5cm, the MCV was 58.0m/sec. Action potentials for the ulnar nerve are also shown in Figure 11; a MCV of 52.5m/sec was calculated from a distal latency of 2.3msec, a proximal latency of 8.2msec, and a distance of 31.0cm.

Because the sensory distal and proximal action potentials are quite low in amplitude and of short duration, it is difficult to obtain a response; sensitivity must be set (on the amplifier) sufficiently high such that the response is often masked by noise. Data were recorded only from those workers who provided clearly defined sensory responses. The results of the analysis of the SCV data, shown in Table 10 and Figure 10, indicate that the SCV of the experimental group was slower than for the control group. This difference was statistically significant ($p < .015$) and represents a mean SCV difference of about 6.5m/sec.

Data of the CVSF were obtained from 65 workers. Between the experimental and control groups there was no statistically significant difference ($p < .15$). In fact, the data in Table 10 and Figure 10 indicate that the CVSF for the lead-exposed group was slightly higher than that of the control group. Technically the more difficult to obtain, the CVSF of the ulnar nerve is of particular interest because it involves the method of partial antidromic blocking.

In the application of this technique (see Appendix A), when the proximal stimulus is delayed, two separate action potentials are obtained. Beginning at a point at which the two responses appear, the interval in time between deliverance of the two stimuli is decreased continuously until the second response just begins to decrease. The measure of the CVSF is taken at the point of the last full response before a decrease in amplitude occurs.

Determining the point at which the second response (or third, etc.) begins to decrease is subject to differences in neuronal response. Figure 12 shows three characteristically different responses recorded from separate individuals. In the lower trace of Figure 12(A), the distal and proximal responses are displayed on an oscilloscope in the storage mode and the repeated tracings show the decrease in interval between distal and proximal stimulation. This tracing is particularly good because it shows consistency in waveform during repeated stimulation and clearly shows the point at which the response begins to decrease. The upper trace is a verification of the two independent distal and proximal waveforms just before the response begins to decrease. The lower tracings in Figure 12(B) show that closer intervals were required to determine where antidromic blocking occurs. It is very difficult to determine from this picture the point of the last full potential before blocking. Also, this tracing shows a slight change in configuration before a change in amplitude. It is only on rare occasions that a configuration change will occur before an amplitude decrease. However, the measure is still determined on the basis of the amplitude change and can be verified on the potentiometer (of the oscilloscope). The lower tracings in Figure 12(C) are similar to the tracings in Figure 12(A) and 12(B) except that the change in amplitude is much less and the interval between stimuli is also less. The waveform, however, does not change with repeated stimulation.

The MCV of the posterior tibial and deep peroneal nerves was also slower among lead workers when compared with the control group. Although the total number of observations was obtained on only about half of the total sample, the differences were statistically significant. A difference of about 5.0m/sec for the posterior tibial nerve was significant ($p < .0013$) and the difference of about 4.9m/sec for the peroneal nerve was also significant ($p < .0018$). These results are given in Table 10 and shown graphically in Figure 13. Well defined tracings of the distal and proximal action potentials for both the posterior tibial and deep peroneal nerves are shown in Figure 14. For the tibial nerve action potentials illustrated in Figure 14, a MCV of 47.9m/sec was calculated from a distal latency of 5.3msec, a proximal latency of 14.6msec, and a distance of 44.5cm. Similarly, the distal peroneal latency of 4.7msec, proximal latency of 11.0msec, and distance of 32.5cm yielded a MCV of 51.6m/sec.

Presented in Table 11 are the coefficients of correlation between each of the six NCVs of the right extremities and each of the five biomedical indicators of lead absorption and exposure. It can be seen from these data that the correlations are overall quite low and that only two relationships attained significance. The correlation between the MCV of the ulnar nerve was significantly related to PbB ($r = .2274$; $p < .05$) and to PbU ($r = .2988$; $p < .01$).

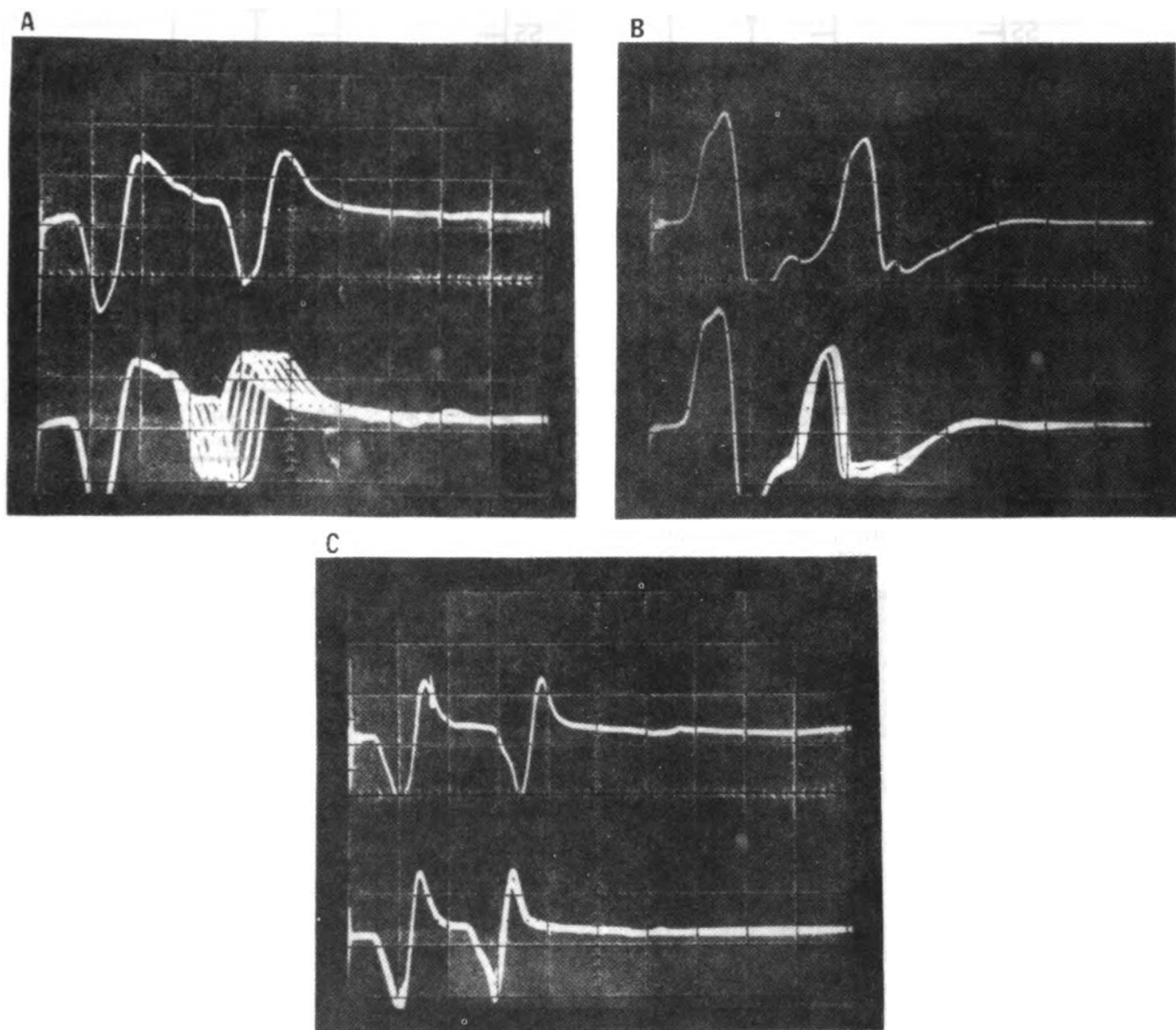


Figure 12. Three characteristically different CVSF responses. The lower trace in each frame shows the distal and proximal responses with repeated stimulation. The upper trace is a verification of both the configuration and amplitude of the waveforms.

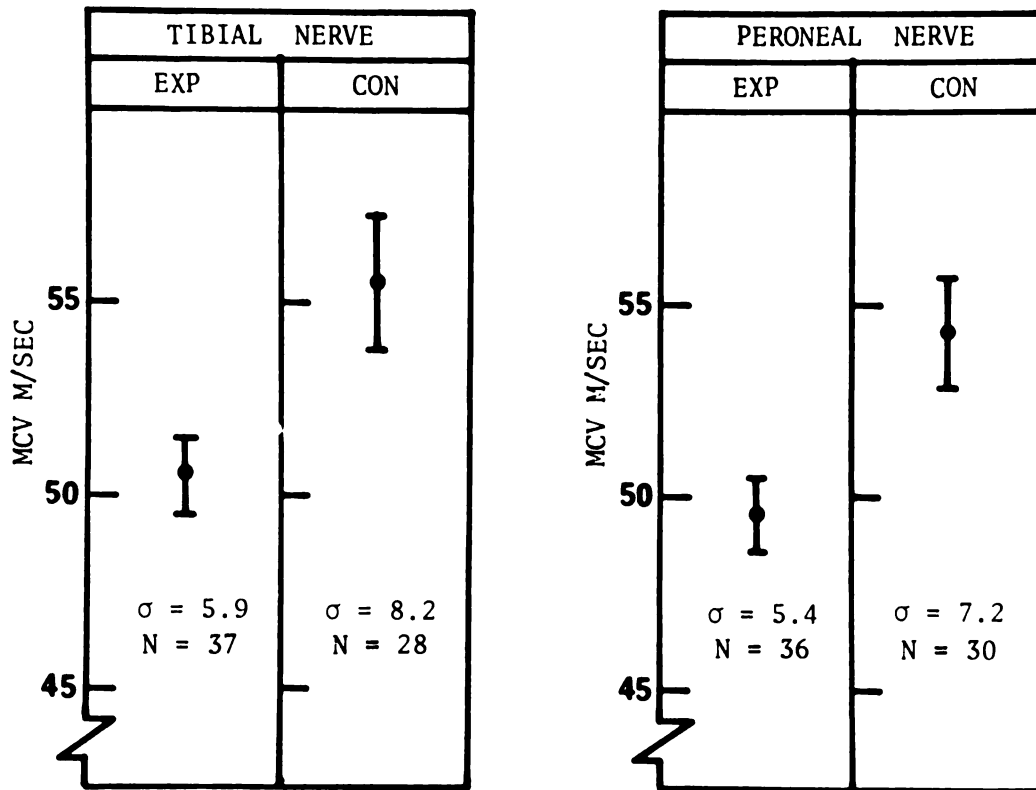


Figure 13. The means, standard errors (plotted), and standard deviations (σ) of the MCV measures of the right leg.

BEHAVIORAL TEST BATTERY RESULTS

A total of 53 primary performance measures was obtained from the six behavioral tests employed in this study. The results of the test of tremor are not included in the text because of difficulties encountered in retrieving these data from FM magnetic tape; in the recording process, a great deal of noise from the recording amplifier masked the recorded tremor signal. In all of the following tables pertaining to the behavioral measures, the number of observations approaches the total N of the study population. In each case, the N associated with the respective analysis is noted in the table. In addition to the computation of the correlation coefficients, values for the t statistic were computed to determine the significance of the difference between the experimental and control group means. In most cases, the variances were approximately equal for both groups, thus indicating that the t statistic was appropriate. The values of r and t were considered to be significant only if the chance probabilities were less than or equal to five percent.

Strength, Endurance, and Recovery

Each worker's gross muscular strength was analyzed in terms of the original maximum strength, length of endurance, and a secondary or recovery strength. As can be seen in Table 12, the lead-exposed workers did show a

Table 11
Coefficient of Correlation (r) for
Nerve Conduction Velocities of the Right Limbs

Conduction Velocity Measure	Correlations (r)				
	PbB	ALA-D	FEP	PbU	δ -ALA
Median MCV ¹	.0389	.0605	-.0613	.0542	-.1122
Ulnar MCV ²	.2274*	-.1849	.0658	.2988**	-.0228
Ulnar SCV ³	.0661	-.1426	.2236	.1265	-.1526
Ulnar CVSF ⁴	.1608	-.1323	.0256	.0404	-.0096
Tibial MCV ⁵	.0648	.0566	-.2893	-.0325	-.0418
Peroneal MCV ⁴	.0233	.1827	-.1759	-.0699	-.1933

¹76 < N < 79; ²75 < N < 78; ³19 < N < 20; ⁴32 < N < 35; ⁵33 < N < 36;
* $p < .05$; ** $p < .01$

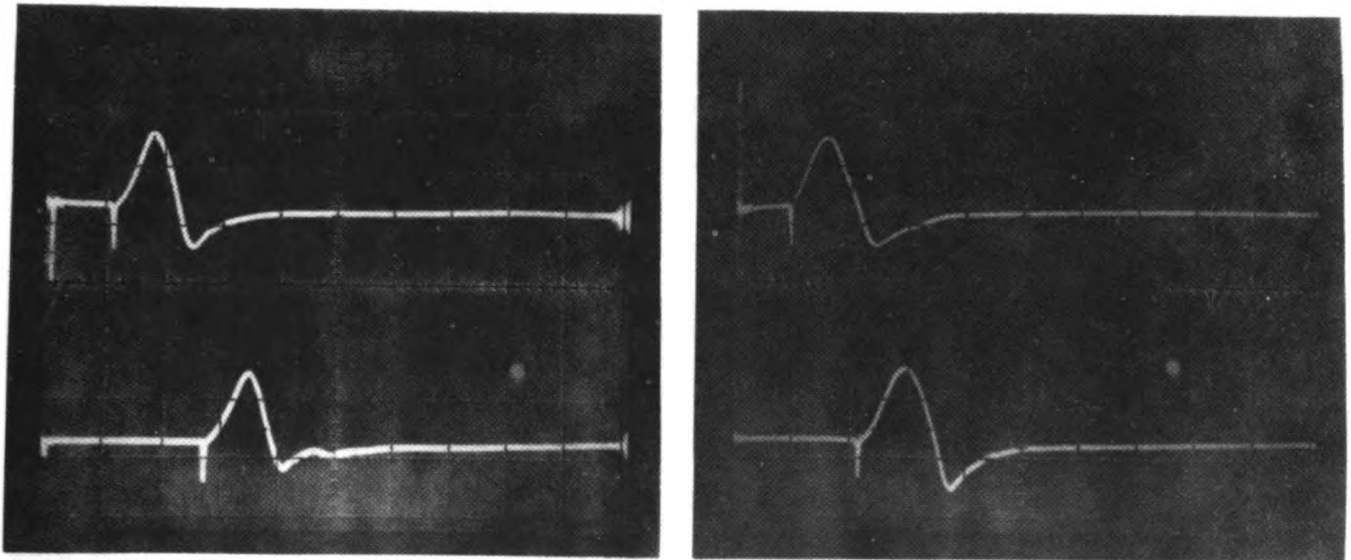


Figure 14. Distal action potential (upper trace) and proximal action potential (lower trace) for the posterior tibial (left frame) and deep peroneal nerves (right frame).

tendency to have a slightly greater strength and endurance than the control workers. These differences between the two groups, however, were not statistically significant. From the data presented in Table 13 it is also clear that the relationships between the strength measures and the biomedical indices were also not significant.

Since strength is a measure which is influenced by height and weight, the data presented earlier in Table 3 indicate that these two variables were adequately controlled with respect to the overall sample group populations. On the other hand, differences in strength do exist between males and females and the percentage of females in the control group was a great deal higher than the percentage in the experimental group. Therefore, the three measures of strength were also analyzed without the inclusion of the data obtained from the females. These results were substantially no different from those obtained with the females included. The t -values associated with the measures of strength, endurance, and recovery, respectively, were $t = 0.7432$, $t = 1.5126$, and $t = 0.4226$, all of which were not statistically significant ($df = 125$).

Eye-hand Coordination

Performance on the eye-hand coordination task was analyzed in terms of the mean hole-to-hole movement time and the variability of performance for both the right and left hands. Examination of Table 14 reveals that the controls were slightly slower and somewhat more variable in their performance of this task than the lead-exposed workers. However, comparison of the two groups by the t statistic indicated that these differences were not significant. The coefficients of correlation between these measures and the biomedical indicators were, overall, quite low (Table 15). Only the correlation between the mean hole-to-hole time for the left hand and serum ALA-D was significant ($r = .2524$; $p < .05$). That only one correlation was significant in this set of 20 correlations is not unusual, since one or two significant correlations would be expected to occur by chance alone.

Visual Reaction Time

Only two measures were obtained from the test of visual reaction time, the mean response time (in hundredths of a sec) for both the right and left hands. These data are presented in Table 16. Inspection of this table reveals that for both the right and left hands, the lead-exposed workers were slower in response than the control workers. This difference between the means for the performance of the right hand was statistically significant ($p < .05$); the difference for the left hand was not significant.

Since the response required for the performance of this task requires the innervation of the ulnar nerve, the coefficients of correlation between the reaction times for the right hand and the four right arm NCV measures were computed. The results of these analyses are given in Table 17. It can be seen that there is a significant negative relationship between increases in visual reaction time and decreases in the MCV of the median ($r = -.2226$; $p < .05$) and ulnar nerves ($r = -.2699$; $p < .02$). The correlations between

Table 12
Group Means, Standard Errors, and
t-Values for Strength, Endurance, and Recovery

Performance Measure	Means		<i>df</i>	<i>t</i>
	Exp. ¹	Con. ²		
Maximum Strength (lbs)	61.54 (1.602)	57.35 (2.095)	138	1.6080
Endurance (sec)	56.79 (3.894)	48.80 (3.579)	138	1.4177
Recovery Strength (lbs)	56.25 (1.605)	52.73 (2.197)	138	1.3207

¹*N* = 85; ²*N* = 55

Table 13
Coefficients of Correlation (*r*) for
Strength, Endurance, and Recovery

Performance Measure	Correlations (<i>r</i>) ¹				
	PbB	ALA-D	FEP	PbU	δ-ALA
Maximum Strength	.0549	-.0264	.0354	.1365	.0644
Endurance	.0952	-.1710	.0892	-.0116	.0602
Recovery Strength	.0022	-.0231	-.1114	.0904	-.0169

¹76 ≤ *N* ≤ 80

visual reaction time and the SCV or the CVSF of the ulnar nerve were not significant. It should be noted that the numbers involved in these latter correlations were quite low, *N* = 21 and *N* = 36, respectively. Despite the fact that poorer reaction times were obtained for the lead-exposed workers, there were no significant relationships between the two visual reaction time measures and the biomedical indicators (see Table 18).

Auditory Acuity and Tone Decay

The results of the analyses of the pure-tone auditory thresholds and the tone-decay measures are presented in Tables 19 through 22. Pure-tone auditory

Table 14
Group Means, Standard Errors, and
t-Values for Eye-Hand Coordination

Performance Measure	Means		df	t
	Exp. ¹	Con. ²		
<hr/>				
<u>Right Hand</u>				
Mean RT (msec)	546.37 (8.326)	549.66 (11.683)	132	0.2350
Variability	162.88 (4.987)	173.25 (6.851)	132	1.2465
 <u>Left Hand</u>				
Mean RT (msec)	584.74 (8.285)	600.73 (11.702)	134	1.1457
Variability	170.19 (4.953)	181.09 (6.924)	133	1.3135

82 ≤ *N* ≤ 83; 51 ≤ *N* ≤ 53

Table 15
Coefficients of Correlation (*r*) for
Eye-Hand Coordination

Performance Measure	Correlations (<i>r</i>) ¹				
	PbB	ALA-D	FEP	PbU	δ-ALA
<u>Right Hand</u>					
Mean RT	-.0096	.1599	-.1025	-.0974	-.0674
Variability	.0041	-.0315	.0305	-.0844	.1437
<u>Left Hand</u>					
Mean RT	-.0056	.2524*	-.1361	-.0124	-.1547
Variability	.1153	.0366	-.1236	-.0239	-.1344

¹74 ≤ *N* ≤ 781 **p* < .05

Table 16

Group Means, Standard Errors, and
t-Values for Visual Reaction Time

Performance Measure	Means		df	t
	Exp. ¹	Con. ²		
Right Hand RT (hsec)	32.87 (0.729)	30.64 (0.710)	137	2.0863*
Left Hand RT (hsec)	31.52 (0.721)	29.62 (0.576)	137	1.8869

¹N = 84; ²N = 55; *p < .05

Table 17

Coefficients of Correlation (r) between
Arm NCV Measures and Right Visual Reaction Time

NCV Measure	N	Correlations (r)
Median MCV	79	-.2226*
Ulnar MCV	79	-.2699**
Ulnar SCV	21	.0958
Ulnar CVSF	36	.2846

*p < .05; **p < .02

Table 18

Coefficients of Correlation (r) for
Visual Reaction Time

Performance Measure	Correlations (r) ¹				
	PbU	ALA-D	FEP	PbU	δ-ALA
Right Hand RT	-.0929	.0493	-.0994	-.1409	-.0763
Left Hand RT	-.1543	.0188	.0489	-.0382	-.0152

¹76 ≤ N ≤ 79

thresholds were obtained at frequencies from 250Hz to 8000Hz, which provided 10 measures each for the right and left ear. It should be noted that the number of observations for each measure is smaller than the total population of workers tested. From either the neurological evaluation, a previous history of known auditory damage or known source of severe noise exposure, or a clear auditory problem (such as otosclerosis) revealed during the measurement of hearing via bone conduction, the data from 11 workers (experimental and control) were not included in the analyses. The measurement of pure-tone thresholds via bone conduction was primarily included in the test procedure for screening subjects with clear indications of otosclerosis. In nine other instances, the particular audiograms could not easily be read at certain specific frequencies; in these cases the data for that worker at that specific frequency was also not included in the analysis.

Examination of the data in Table 19 reveals that the pure-tone auditory thresholds were slightly higher for the lead-exposed workers across all frequencies in both the right and left ears except at 8000Hz (right ear only). Moreover, these differences for the right ear were statistically significant at 5000Hz, 750Hz, 1000Hz, and 4000Hz ($p < .05$); similarly, for the left ear at 3000Hz, 4000Hz, and 6000Hz the differences were also significant ($p < .05$). A plot of the threshold means across the test frequencies for the lead-exposed and control workers is provided in Figure 15. It can be seen from these data that there is a slight, parallel increase in threshold for both groups as the frequency increases to 8000Hz.

Presented in Table 20 are the coefficients of correlation between each of the auditory threshold measures and the five biomedical indicators. With the exception of a significant positive correlation between FEP and threshold at 250Hz for the left ear ($p < .05$), none of the indicators of exposure or absorption was significantly related to the left ear threshold measures. On the other hand, for the right ear there was clear evidence of a relationship between FEP and the threshold measures. It is unclear to us why FEP correlated significantly with right ear hearing data, but did not for the left ear. Except for the pure-tone thresholds at 250Hz and 4000Hz, the remaining coefficients of correlation were significant at chance probability levels of less than five percent or less than one percent. The other remaining biomedical indicators were not significantly related to the right ear threshold measures; although δ -ALA did show a significant correlation with threshold at 250Hz ($p < .01$).

Presented in Table 21 are the results of the t -test analyses of the 12 measures obtained from the tone-decay test. The tone-decay test was conducted in the poorer ear at frequencies of 500Hz, 2000Hz, 4000Hz, and 8000Hz. The three measures at each frequency represent the amount of tone decay--actually, the amount of time the tone remained audible--at threshold and at 5dB and 10dB above threshold. It can be seen from these data that tone decay occurred earlier in the lead-exposed workers than it did in the non-exposed controls, especially at threshold and at 5dB above threshold. At 500Hz, 2000Hz, and 8000Hz, these differences between the two groups were statistically significant ($p < .05$ and $p < .01$ in two cases). In addition, at 10dB above threshold at 8000Hz the difference between the two groups was also statistically significant ($p < .05$). None of the tone-decay differences at 4000Hz was statistically significant. The coefficients of correlation between each of the tone-decay measures and the biomedical measures are presented in Table 22. In this

Table 19
Group Means, Standard Errors, and
t-Values for Pure-Tone Auditory Thresholds

Performance Measure	Means		df	t
	Exp. ¹	Con. ²		
<u>Right Ear</u>				
250Hz	15.59 (1.206)	12.98 (1.119)	126	1.4630
500Hz	9.278 (1.040)	5.688 (1.007)	125	2.3194*
750Hz	7.432 (1.091)	3.083 (0.940)	127	2.7510*
1000Hz	7.358 (1.185)	3.479 (0.859)	127	2.3126*
1500Hz	6.259 (1.246)	3.458 (1.008)	127	1.5592
2000Hz	4.123 (1.419)	2.167 (1.308)	127	0.9310
3000Hz	13.469 (1.763)	9.250 (2.133)	127	1.4970
4000Hz	20.506 (2.181)	13.000 (2.366)	127	2.2276*
6000Hz	21.642 (1.843)	16.542 (2.807)	127	1.5819
8000Hz	17.025 (2.008)	17.625 (2.660)	127	0.1810
<u>Left Ear</u>				
250Hz	13.913 (0.995)	11.229 (1.500)	126	1.5496
500Hz	5.728 (1.011)	4.063 (1.558)	127	0.9372
750Hz	4.568 (1.012)	3.271 (1.654)	127	0.7090
1000Hz	3.975 (1.069)	2.125 (1.483)	127	1.0291

Table 19 (Continued)

Performance Measure	Means		<i>df</i>	<i>t</i>
	Exp. ¹	Con. ²		
1500Hz	4.395 (1.299)	3.146 (1.613)	127	0.5961
2000Hz	5.037 (1.514)	3.979 (1.920)	127	0.4300
3000Hz	18.864 (2.201)	11.255 (2.635)	126	2.1622*
4000Hz	26.123 (2.407)	16.787 (2.865)	126	2.4305*
6000Hz	27.160 (2.449)	19.106 (2.784)	126	2.0909*
8000Hz	22.063 (2.433)	15.213 (2.669)	125	1.8132

¹78 ≤ *N* ≤ 80; ²46 ≤ *N* ≤ 48; **p* < .05

group of 60 correlations only three significant relationships emerged. These were between δ -ALA and threshold at 2000Hz ($r = -.2134$; $p < .05$), between ALA-D and both threshold at 4000Hz ($r = .2346$; $p < .05$) and threshold plus 10dB at 4000Hz ($r = .2144$; $p < .05$). In view of the large number of correlations obtained, these three are probably spurious.

Psychological and Social Assessment

An analysis of the workers' single score obtained from the Marlowe-Crowne Scale indicated that there was no significant difference between the experimental and control groups in responding to the statements concerning personal attitudes and traits ($t = 1.7736$; $df = 137$; $p > .05$). Thus, it is apparent that the two groups were responding to the Clinical Analysis Questionnaire, in particular, and the test situation generally, in approximately the same manner. Tables 23 and 24 contain the results of the analyses of the data obtained from the CAQ. The results were scored and analyzed in terms of the 12 pathological factors contained in the CAQ. For only two of the pathological factors was there a significant difference between the two groups. These factors involved the dimensions of zestfulness-disgust ($p < .05$) and guilt and resentment ($p < .01$). However, in both cases, the scores indicate a relatively moderate response in which the workers are somewhat less than contented about life and surroundings and are slightly troubled by feelings of (inwardly-directed) guilt. The significant difference between the groups suggests that these personality characteristics are more intense in the

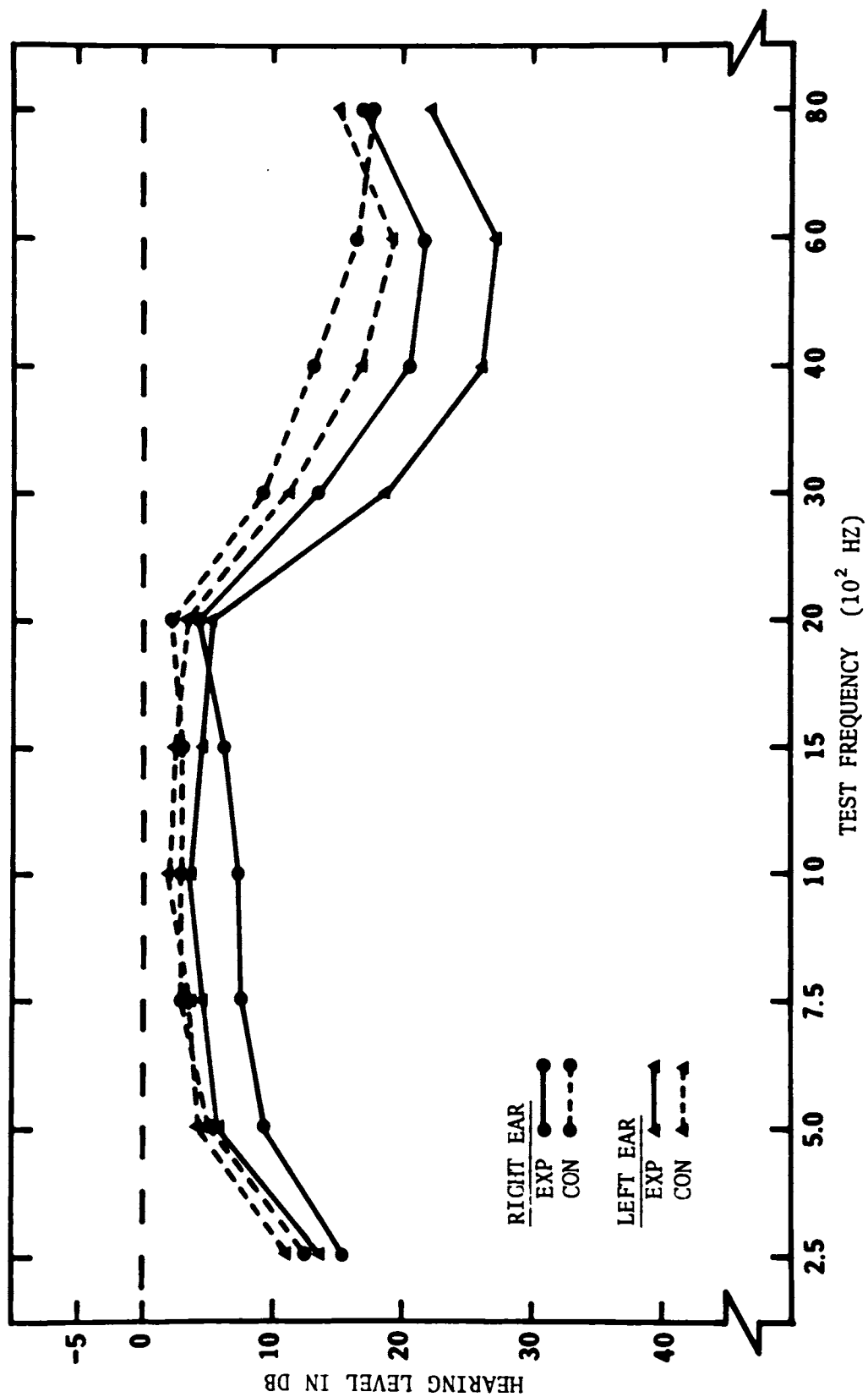


Figure 15. Mean hearing levels for right and left ear of lead exposed and control workers.

Table 20
Coefficients of Correlations (*r*) for
Pure-Tone Auditory Thresholds

Performance Measure	Correlations (<i>r</i>) ¹				
	PbB	ALA-D	FEP	PbU	δ-ALA
<u>Right Ear</u>					
250Hz	.2246	-.1387	.1550	.2474	.2859**
500Hz	.0336	-.1567	.3135**	.0132	.1403
750Hz	-.0010	-.0230	.2593*	-.0968	.0206
1000Hz	.0533	-.1079	.2982**	-.0938	.1068
1500Hz	-.0048	-.0565	.2926**	-.1229	.0512
2000Hz	-.0652	-.0168	.3057**	-.0149	.0610
3000Hz	-.0303	-.0040	.2480*	.0005	.0952
4000Hz	-.0181	.0259	.1408	-.0158	-.0168
6000Hz	-.0214	-.0457	.2333*	.1059	.0244
8000Hz	.0961	.0451	.2816**	.1232	.0463
<u>Left Ear</u>					
250Hz	.1376	-.0631	.2075*	.1258	.0717
500Hz	-.0902	.0668	.0929	-.0445	-.0764
750Hz	-.1274	.0381	.1113	-.1670	-.1289
1000Hz	-.0150	-.0232	.1059	-.1125	-.0442
1500Hz	.0525	-.0245	.0699	-.0036	-.0793
2000Hz	.0708	.0959	.0190	.0194	-.0410
3000Hz	-.0512	.0042	.0701	-.0736	-.0769
4000Hz	-.0129	-.0478	.0579	.0057	-.0489
6000Hz	-.0075	-.1014	.0837	.0098	-.0314
8000Hz	.0466	-.0651	.0551	.0105	-.0116

¹70 ≤ *N* ≤ 76; **p* < .05; ***p* < .01

Table 21
Group Means, Standard Errors, and
t-Values for Tone Decay

Performance Measures	Means		df	t
	Exp. ¹	Con. ²		
<hr/>				
500 Hertz				
Threshold	42.747 (2.655)	52.552 (2.397)	127	2.5051*
Threshold + 5dB	53.356 (1.752)	58.195 (1.257)	127	1.9550*
Threshold + 10dB	58.318 (0.856)	59.328 (0.672)	127	0.8226
2000 Hertz				
Threshold	41.977 (2.414)	52.788 (2.337)	127	2.9894**
Threshold + 5dB	52.501 (1.629)	57.370 (1.487)	127	2.0221*
Threshold + 10dB	58.203 (0.699)	57.686 (1.324)	127	0.3787
4000 Hertz				
Threshold	44.637 (2.243)	49.635 (2.836)	126	1.3685
Threshold + 5dB	52.463 (1.688)	53.144 (2.323)	126	0.2401
Threshold + 10dB	56.831 (0.943)	55.461 (1.876)	126	0.7257
8000 Hertz				
Threshold	38.387 (2.379)	48.110 (2.606)	125	2.6327**
Threshold + 5dB	47.477 (2.045)	54.834 (1.922)	125	2.4131*
Threshold + 10dB	51.861 (1.656)	56.892 (1.671)	125	1.9840*

¹81 ≤ *N* ≤ 83; ²47 ≤ *N* ≤ 48; **p* < .05; ***p* < .01

Table 22
Coefficients of Correlation (*r*) for
Tone Decay

Performance Measure	Correlations (<i>r</i>) ¹				
	PbB	ALA-D	FEP	PbU	δ-ALA
<u>500 Hertz Threshold</u>	.0602	.0060	-.0267	.0261	-.0423
Threshold + 5dB	-.0896	.0705	-.1273	-.1422	-.0124
Threshold + 10dB	-.1444	.1533	.0067	-.0380	.0167
<u>2000 Hertz Threshold</u>	-.0249	.1138	-.1287	.1326	-.2134*
Threshold + 5dB	-.0055	.1861	-.1039	.0518	-.1406
Threshold + 10dB	-.1665	.1814	-.1580	-.1201	-.1396
<u>4000 Hertz Threshold</u>	-.0074	.2346*	-.0974	.1913	.0471
Threshold + 5dB	.1232	.2144*	.1080	.1867	-.0001
Threshold + 10dB	-.0002	.1260	-.0396	.0212	-.0126
<u>8000 Hertz Threshold</u>	.0622	.1683	-.0431	.0604	-.0845
Threshold + 5dB	.0771	.1125	-.1390	.0124	-.1650
Threshold + 10dB	.0889	.0386	-.1876	.0913	-.1689

¹70 ≤ *N* ≤ 76; **p* < .05

control group. Overall, the scores for both groups (given as percentiles) range within what is regarded as average for an adult population. In terms of these group data, no pathologically negative extremes were noted.

SUMMARY OF THE PERSONAL-DATA QUESTIONNAIRE RESULTS

The results of the Chi-square analyses of the response to the primary items contained in the personal-data questionnaire are presented in Table 25. The responses are clustered into five categories, namely, eating and sleeping habits, cigarette smoking, drinking, and work habits. A summary of the percentage response to the questions within each of the categories is shown

graphically in Figures 16 through 20. It is evident from the results of the χ^2 analyses and the graphical representation of the data that the differences between groups are small and that the responses do not differ from what would be expected in the normal population.

Only with respect to the cluster of items involving work habits is there a significantly different distribution of responses between the two groups. Since the controls would not be expected to shower or use respirators, the significant χ^2 values associated with these responses is not unexpected. On the other hand, the significant χ^2 associated with the question of whether or not they have been involved in work accidents in their current jobs is of interest. In the experimental group, 35 workers, or 41.2 percent, indicated that they had been involved in a work-accident, whereas in the control group, only five, or 9.1 percent, responded positively to this question.

Table 23
Group Means, Standard Errors, and
t-Values for the Clinical Analysis Questionnaire

Performance Measure	Means		<i>df</i>	<i>t</i>
	Exp. ¹	Con. ²		
Hypochondriasis	56.2 (3.24)	59.8 (4.00)	137	0.7056
Zestfulness-disgust	51.3 (3.71)	64.5 (4.25)	137	2.2798*
Brooding discontent	42.2 (3.14)	48.5 (4.39)	137	1.1996
Anxious depression	69.9 (2.72)	67.0 (3.30)	137	0.2241
Energy euphoria and depression	56.2 (3.14)	63.3 (3.19)	137	1.5220
Guilt and resentment	54.1 (3.44)	68.5 (3.72)	137	2.7433*
Bored depression	65.5 (2.70)	64.3 (3.94)	137	0.2530
Paranoia	68.0 (2.37)	66.2 (4.05)	137	0.4113
Psychopathic deviation	42.6 (3.00)	46.2 (4.09)	137	0.7157
Schizophrenia	64.3 (2.49)	65.6 (3.86)	137	0.2976
Psychasthenia	62.3 (2.74)	62.5 (3.80)	137	0.0330
General Psychosis	55.4 (2.99)	58.7 (4.28)	137	0.6481

¹*N* = 85; ²*N* = 54; **p* < .05

Table 24

Coefficients of Correlation (r) for
the Clinical Analysis Questionnaire

Performance Measure	Correlations (r) ¹				
	PbB	ALA-D	FEP	PbU	δ -ALA
Hypochondriasis	.1225	.0780	-.1634	-.0157	-.0726
Zestfulness-disgust	.2065	-.1656	-.0295	.0603	-.0576
Brooding discontent	-.0870	.0271	-.1475	.0590	-.0620
Anxious depression	.0567	-.1059	-.1613	-.1275	-.0598
Energy euphoria and depression	.0907	-.1320	-.1446	-.0135	-.0510
Guilt and resentment	.2144	-.0764	-.0443	.0137	.0829
Bored depression	.1979	.0624	-.1328	.0270	-.1537
Paranoia	.0579	-.0158	-.0537	.2088	.0767
Psychopathic deviation	-.0742	-.0974	.1451	.0443	.1223
Schizophrenia	.0884	-.1272	-.1021	.1060	-.0335
Psychasthenia	.1279	-.0660	-.0722	.0821	.0390
General Psychosis	.0733	-.2263*	-.2000	-.0519	-.0454

¹77 $\leq N \leq$ 80; * $p < .05$

Table 25

Summary of Chi Square (χ^2) Analyses
for Responses from the Personal Data Questionnaire

Question	df	χ^2
<u>Eating Habits</u>		
Attitude Toward Meals	4	4.863
Vitamin Supplements	1	0.146
Quality of Appetite	4	4.924
Special Diet	1	2.574
<u>Sleeping Habits</u>		
Time to go to sleep	3	4.291
Time Most Alert	4	5.774
Sleep Interruption	3	4.040
Restedness	3	5.947
<u>Cigarette Smoking</u>		
History of Smoking	1	3.138
Quantity per Day	5	7.862
<u>Drinking</u>		
Beer	4	5.303
Wine	4	7.636
Whiskey (or Other Liquor)	4	5.152
Normal Drink Time	4	5.924
Period of Time	3	3.095
<u>Work Habits</u>		
Showering	1	32.033***
Respirator Usage	5	15.716**
Work Accidents	1	16.771***

** $p < .01$; *** $p < .001$

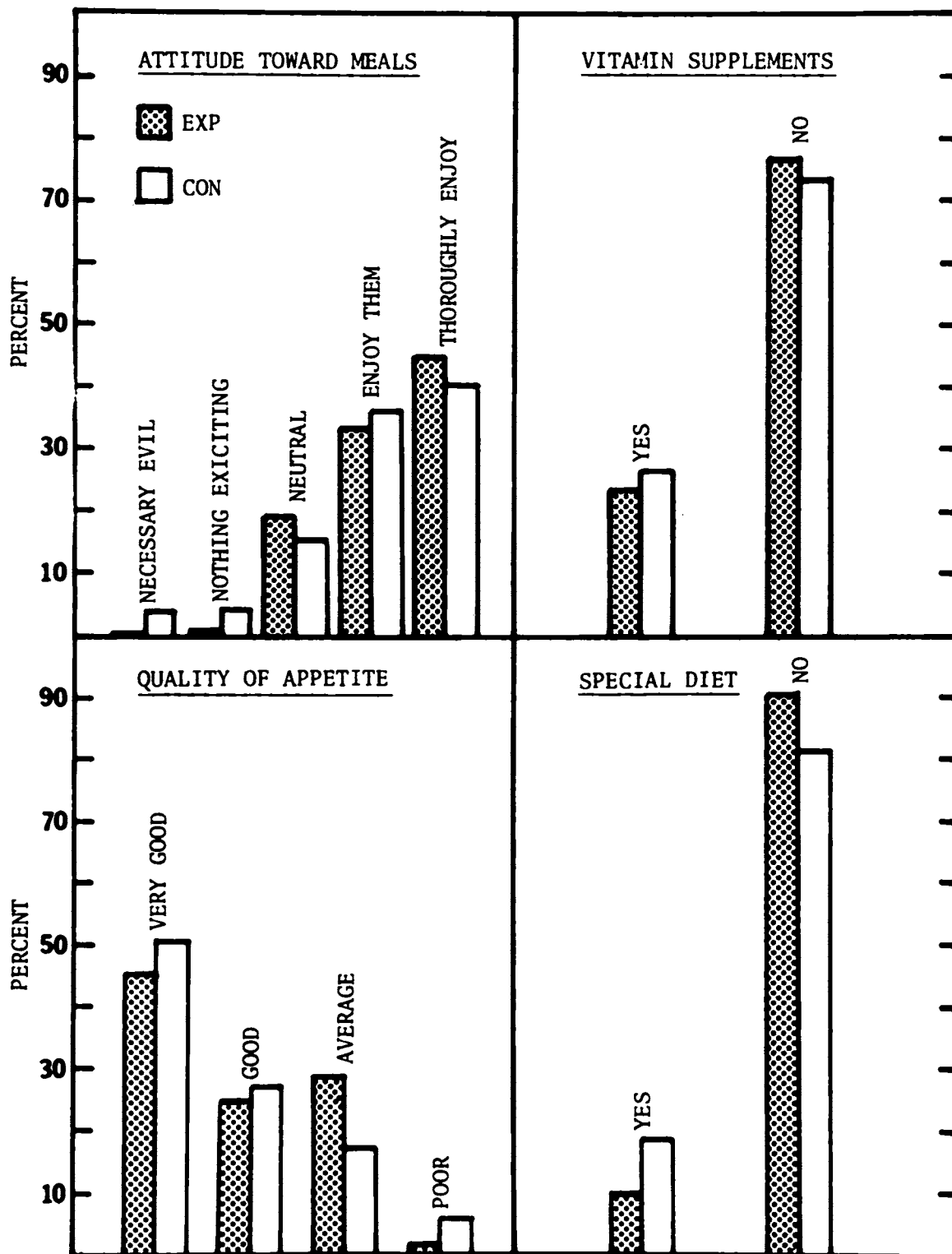


Figure 16. Percentage distribution of responses to the personal-data questionnaire regarding eating habits.

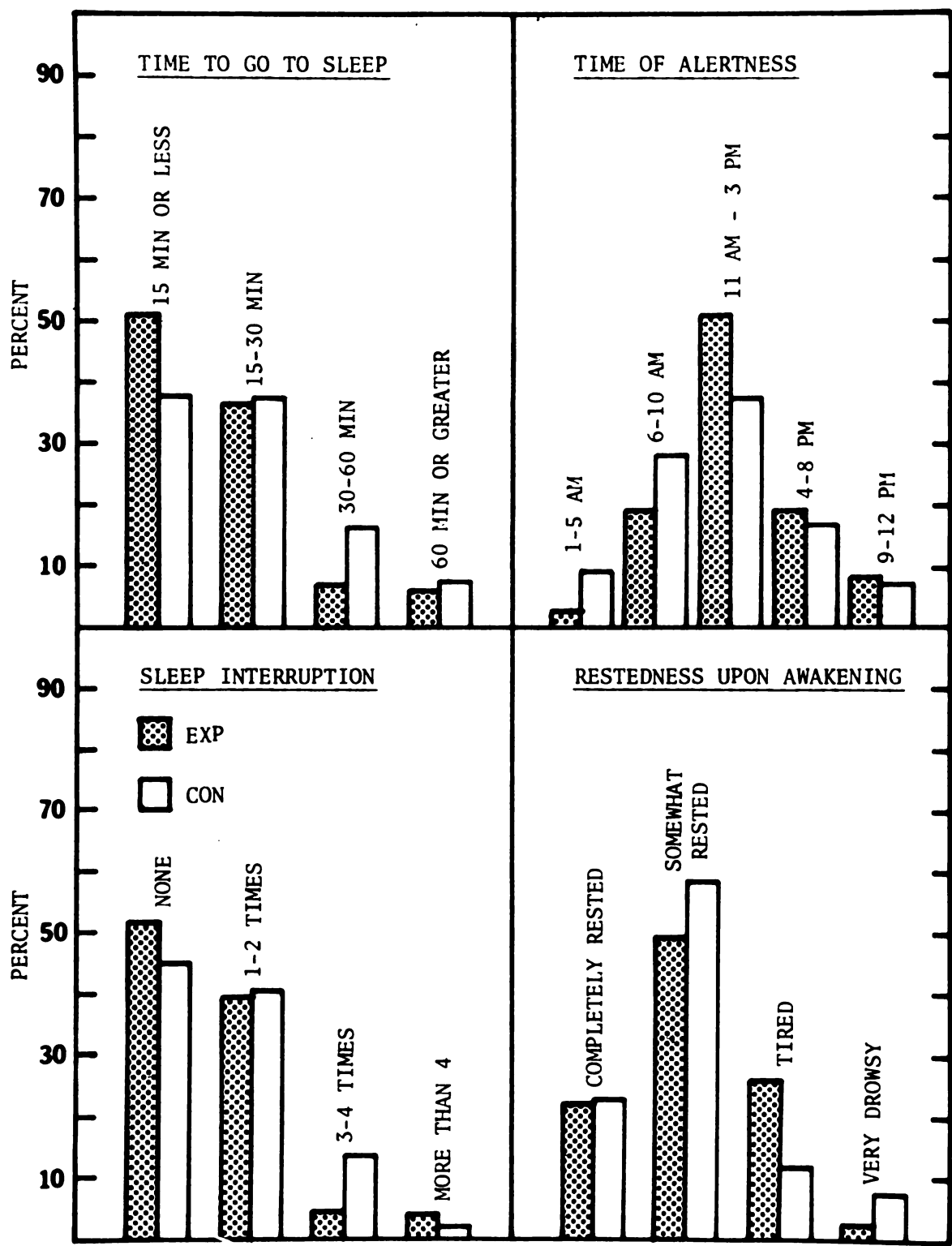


Figure 17. Percentage distribution of responses to the personal-data questionnaire regarding sleeping habits.

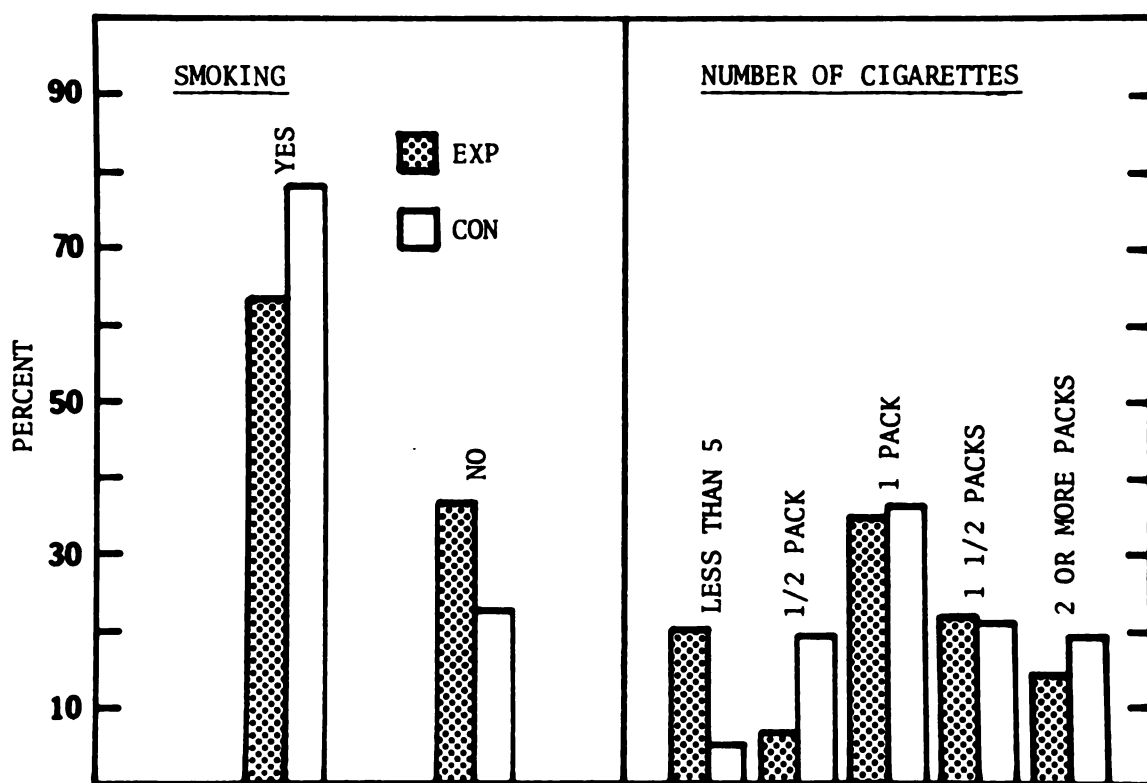


Figure 18. Percentage distribution of responses to the personal-data questionnaire regarding smoking habits.

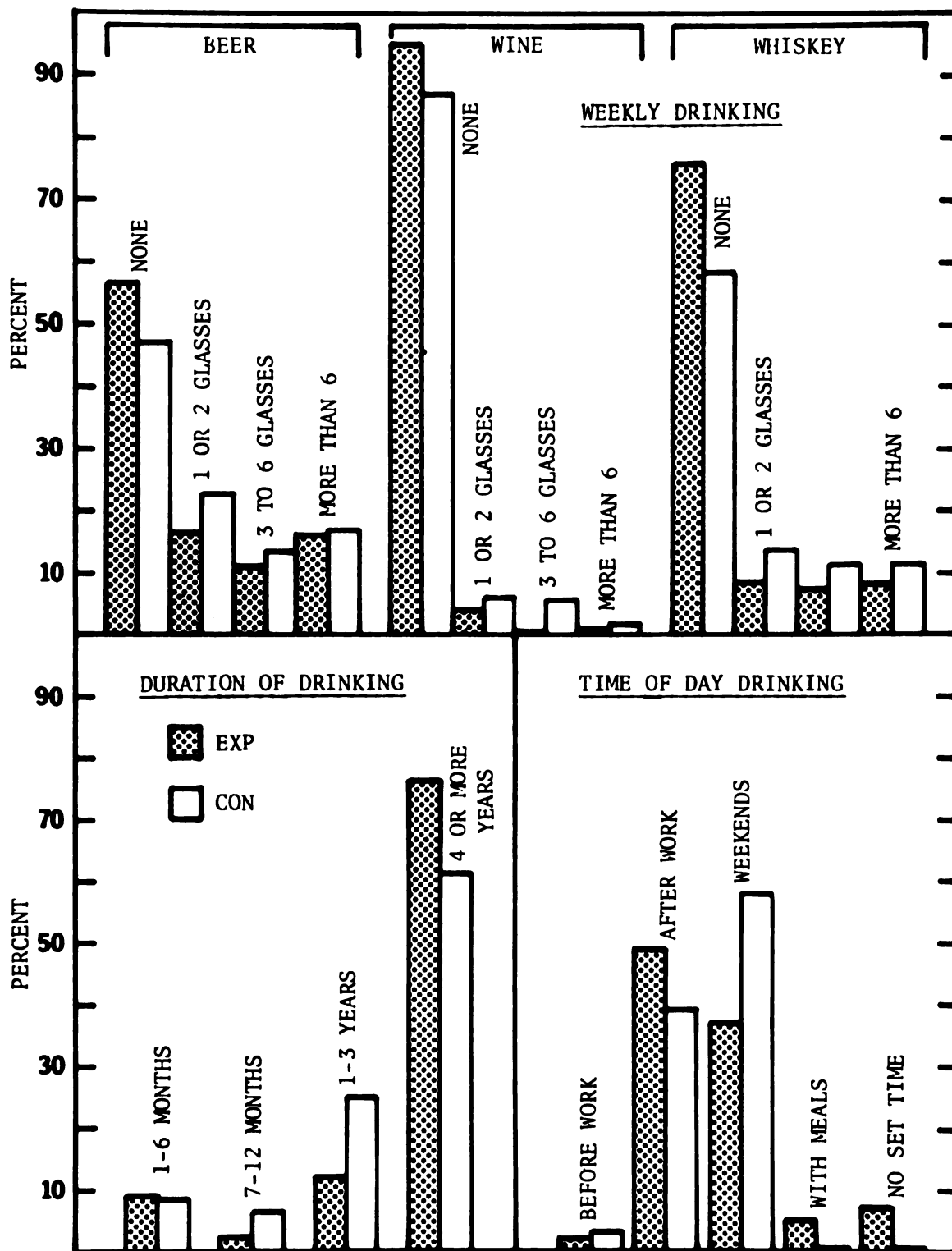


Figure 19. Percentage distribution of responses to the personal-data questionnaire regarding drinking habits.

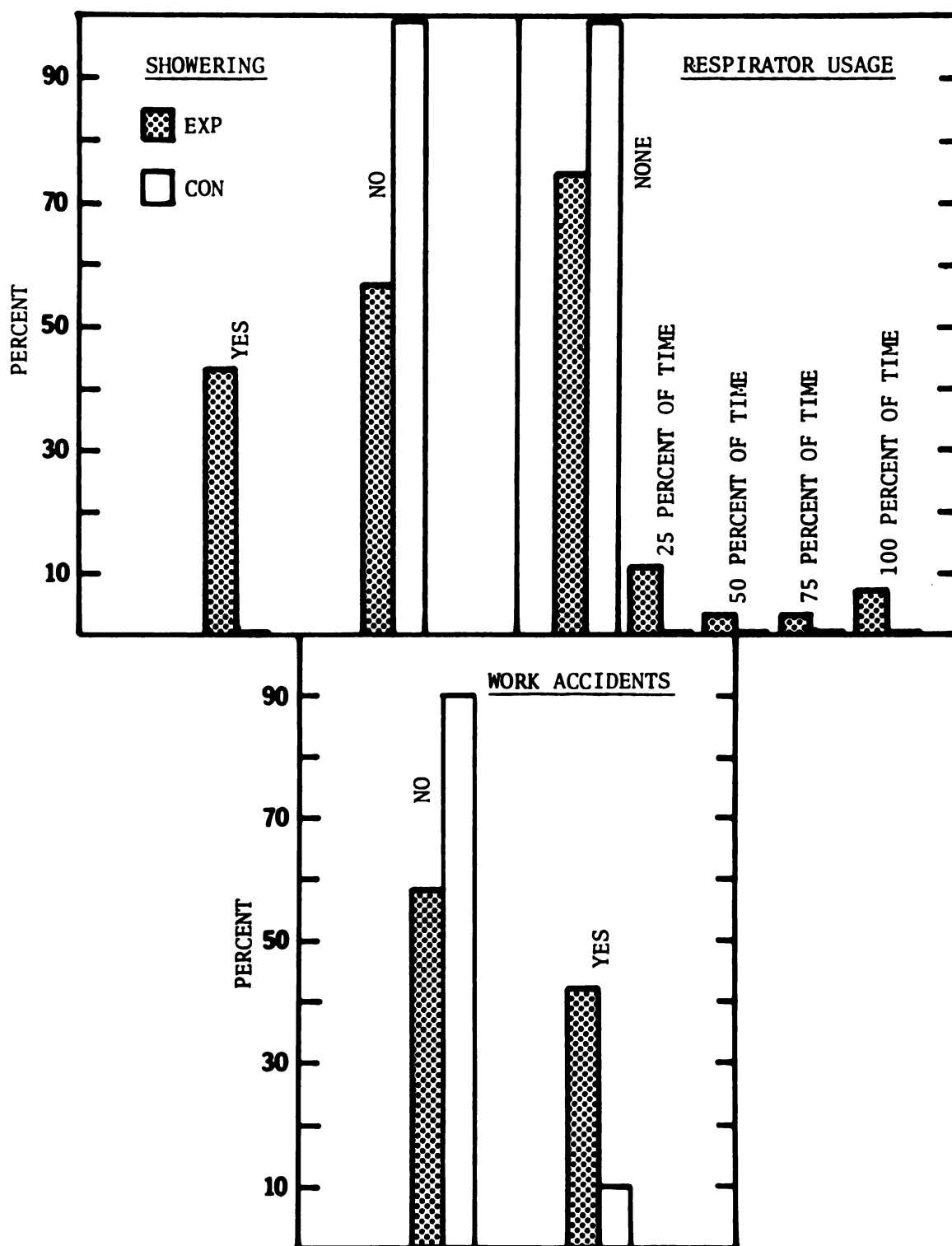


Figure 20. Percentage distribution of responses to the personal-data questionnaire regarding work habits.

DISCUSSION

The central purpose of the present study was to assess the effects of low-level inorganic lead absorption on worker behavior and neurologic function. A recent review of the behavioral and neurological sequelae of lead poisoning indicates that the behavioral correlates of the medical and clinical dysfunctions, and other neurotoxic effects, clearly suggest a gradual behavioral and neurological degradation which is associated with increased lead absorption (Repko & Corum, 1976). Several deleterious effects of lead at or below 80ug/100ml upon the nervous system and sensory and neuromuscular functioning have been reported (Repko, *et al.*, 1975; Repko, 1977; Seppalainen, *et al.*, 1975; Seppalainen, 1977). At a recent *International Workshop on Permissible Levels for Occupational Exposure to Inorganic Lead*, the participants concluded that changes in NCV occur in some lead workers at blood-lead levels about 50ug/100ml and who are otherwise asymptomatic; the conclusion regarding psychological functioning indicated that impairments have been reported below 100ug/100ml, but that further documentation was required (Zielhuis, 1977). This working group recommended that PbB should not exceed 60ug/100ml in *male* workers' individual blood lead. Since a great deal is currently known, therefore, about the effects of lead on behavior and neurologic function above 100ug/100ml, and even at 80ug/100ml and above, the present study specifically focused on workers whose lead exposure has resulted in blood-lead levels less than 80ug/100ml.

The present study measured and evaluated the changes in functional capacity which result from low-level absorption of inorganic lead in the workplace. Data from a total of 140 lead workers from within the storage battery manufacturing industry were compiled for analysis in this study. The two groups consisted of 85 experimental or lead-exposed workers and 55 control workers possessing no known occupational exposure to inorganic lead or other toxic industrial agent. Despite the fact that workers were selected on a voluntary basis from a fairly limited population group, the two study groups were approximately matched in terms of age, level of education, height, and weight. Few women and minority group members volunteered for participation in the study.

The results obtained from the blood and urine samples indicated that the experimental group represented a sample of workers with low-level absorption. The mean blood lead of approximately 46ug/100ml, independent of or in conjunction with the four other biomedical measures, adequately defines a low-level exposed group (no worker's individual blood lead exceeded 79ug/100ml). Similarly, the results of the biomedical determinations for the control group indicated that they were indeed a non-exposed group; the mean blood lead of approximately 18ug/100ml and mean urine lead of 14ug/liter are definitely within the normal range, even where the most conservative standards are used (see Repko & Corum, 1976; see also Zielhuis, 1977).

One of the central questions to which the study addressed itself was the extent to which functional capacity can be measured by non-invasive behavioral tests, and the extent to which exposure to and toxicity of inorganic lead is reflected in the obtained behavioral measures. It has been reported that any relationship between performance and blood lead may actually be

demonstrating a relationship between blood lead and stratification according to general overall ability and the desirability of the job (Crockford & Mitran, 1976). These authors imply that those who have high blood-lead levels are less able intellectually, do the dirtier and less desirable jobs, and are less able to maintain standards of hygiene necessary to reduce absorption. Their results, however, offer little to either reject or accept the null hypothesis. In the present study, there was no evidence to suggest that stratification of the experimental group was a factor influencing the outcome of the behavioral and neurological measures. To the contrary, it was obvious in terms of blood lead and the criteria used in the selection of workers that the experimental group represented a group exposed to low-level airborne lead levels. Workers who would normally be associated with very dirty jobs or high airborne lead areas and unable to maintain appropriate personal hygiene standards and who would usually show a blood lead above 80 μ g/100ml, or at least have had more than one excursion above that level during the previous five years of employment, were excluded from participation in the study.

Crockford and Mitran (1976) do, and rightly so, point up the problems involved in matching the exposed and control groups. In the present study, considerable effort was devoted to matching the two groups. In terms of age, there was no significant mean difference between the two groups. Moreover, in terms of height and weight, there was also no significant difference between the two groups. Although a significant difference did exist between the exposed and non-exposed groups in terms of the mean level of education, the difference is quite small and cannot account for all the differences between groups discussed later. The significant difference in education is not meaningful, however, since there is little difference between individuals who have almost completed the tenth grade (i.e., grade 10.92) and those who are completing the eleventh grade (i.e., 11.76). One year's difference in formal education at the high school level in a group of workers, currently out of school and with an average age of 37, may be significant statistically, but from a functional standpoint is inconsequential. Any importance attached to this difference is further reduced by the fact that the performances or outcome measures are not affected by such slight differences--and even much greater differences--in education on the specific tests chosen for use in this study. The tests employed in this study are specifically sensitive to differences in the integrity of the neurological and sensory and motor systems rather than to differences in normal intellectual ability.

While it is important from a demographic standpoint that the two groups be matched, it is equally important that motivation, level of effort, and test behavior during the evaluation are consistent for the two groups. Although it is difficult to control these factors, it was evident from the results of the psychological and social assessment that the two groups represented a homogeneous population. There were no significant differences between the lead-exposed and control groups on the score obtained from the Marlowe-Crowne Scale. This scale specifically attempts to isolate individuals who describe themselves in favorable, socially desirable terms in order to achieve the approval of others (Crowne & Marlowe, 1964). This test is typically employed in conjunction with other clinical psychological assessments as an index of a subject's attitude toward the test situation. From the group results there was no indication either that the attitude of the

workers differed between groups or that the workers were responding inappropriately to the test situation. Moreover, from the results of the CAQ it was evident that personality and pathological factors were similar for the two groups. No extremes of personality or clinical pathology were noted in either of the study groups.

One of the important findings of this study is that neurophysiological and behavioral dysfunction occurred in a group of lead workers whose blood lead was consistently below $80\mu\text{g}/100\text{ml}$ and who were otherwise asymptomatic. The results of the clinical neurological examination and the clinical electromyogram were normal with respect to signs or symptoms of lead poisoning. While the clinical neurological examination elicited several neurological deficits, the distribution of these deficits did not differ between the two groups and do not differ from what would be expected to occur in a random sample of industrial workers (see Repko, *et al.*, 1976).

CONDUCTION VELOCITY OF THE PERIPHERAL NERVES

The results demonstrating a slowing of the maximal nerve conduction velocities of the peripheral nerves are in agreement with the results of others investigating low-level lead absorption. Seppäläinen, *et al.* (1975), in a group of lead workers whose PbB never exceeded $70\mu\text{g}/100\text{ml}$, found significant differences between lead-exposed workers and controls in the MCVs of the median and ulnar nerves and the CVSF of the ulnar nerve. Although the SCV of the ulnar nerve and the MCVs of the peroneal and posterior tibial nerves were slower in lead workers, those differences were not significant. The differences in conduction velocity between groups in the Seppäläinen, *et al.* (1975) study ranged from $4.0\text{m}/\text{sec}$ for the MCV of the median nerve to $1.2\text{m}/\text{sec}$ for the MCV of the tibial nerve. Preliminary results of a recently completed epidemiologic study of lead smelter workers also demonstrates a slowing of the MCV of both the ulnar and peroneal nerves in male and female lead workers (Johnson, 1977, personal communication). Conduction velocities were slower by approximately $2\text{m}/\text{sec}$ in lead smelter males who had an average PbB of $56.1\mu\text{g}/100\text{ml}$ and also in women production workers with an average PbB of $29.5\mu\text{g}/100\text{ml}$.

The differences in NCVs found in this study, however, differ from both the Seppäläinen, *et al.* (1975) and Johnson (1977) studies in that both the magnitude of the NCV differences is greater between exposed and controls (i.e., 5 to $9\text{m}/\text{sec}$) and the overall NCVs for both groups are numerically higher in the present study. Comparison of NCV data among studies is difficult because of many factors which differ among laboratories. Although standard conduction velocity ranges have been published for the various nerves, each laboratory should establish individual standards (see Seppäläinen, 1975). Even within a given laboratory in which very precise measurements are conducted, some variations result when different individuals determine NCVs. Standards which have been published are quite variable. Some investigators consider NCVs between 45 and $75\text{m}/\text{sec}$ in individuals as within an acceptable range for the ulnar and median nerves and between 38 to $55\text{m}/\text{sec}$ in individuals for the deep peroneal and posterior tibial nerves (Goodgold & Eberstein, 1972). Krusen, Kottke, and Ellwood (1971) report

standards for the peroneal and tibial nerves as high as 65m/sec. Woodburn (1967) notes that the rate of conduction in human myelinated nerves may rise to 125m/sec.

Other factors also make comparison difficult; more critical are age, limb temperature and previous disease states. No evidence was developed during the clinical neurological examinations of previous disease states in either subject group. In general, conduction velocity decreases approximately 1.0m/sec per decade between age 20 and 60. Statistically both groups in this study were identical in terms of age. The most critical factor influencing NCV, however, is limb temperature. Various estimates of the effect of temperature have been offered. Goodgold and Eberstein (1972) report a decrease of 2 to 2.4m/sec per decrease of 1°C in temperature; Krusen, *et al.* (1971) reported that for a similar 1°C decrease, NCV may decrease 1/8 to 4.0m/sec. In a comprehensive investigation of the effect of limb temperature, de Jesus, *et al.* (1973) derived a single semilogarithmic correction formula appropriate for use with both sensory and motor conduction velocity measures. To take into account potential temperature differences, which might have decreased because of individual variations in limb temperature (due to location, climatic, or inherent individual differences), the de Jesus, *et al.* (1973) correction formula, referenced to a standard 31.5°C, was utilized with the NCV data obtained in this study.

Because of these many factors which influence conduction velocity to lesser or greater extents, numerical comparison of NCV data between laboratories must be done with caution. It is sufficient, and important, that the NCV results of this study are in qualitative agreement with other epidemiologic investigations of low-level lead absorption. Seppäläinen, *et al.* (1975) have pointed out that although these types of results demonstrate a toxic effect on the *group* level, the results cannot be used for diagnostic purposes in the *individual* case. The present results, along with those of Seppäläinen, *et al.* (1975) and Johnson (1977), are extremely important from the standpoint of worker health, since demonstrable NCV decrements do occur in workers where lead absorption results in PbB substantially below 80µg/100ml. From a clinical standpoint, clear deficits in nerve conduction velocity of two or more nerves is an early stage in the development of manifest neuropathy. It was the consensus of the *International Workshop* that such changes in conduction velocity are a critical effect in that cellular functioning of a critical organ is adversely affected (see Zielhuis, 1977).

BEHAVIORAL FUNCTIONS

Two important findings with respect to the evaluation of behavioral functions were elicited by the analysis of study data. First, auditory impairment in lead workers was indicated by the results of the pure-tone threshold and tone-decay tests; and, second, visual reaction time, under response control by the ulnar nerve (to the onset of a visual stimulus), was increased in lead workers. The results obtained from the analysis of data of the tests of strength and eye-hand coordination did not show any differences between lead-exposed workers and controls. Moreover, significant correlations between the behavioral measures and the biomedical indices were sparse.

Only one relationship of importance was noted--a significant positive relationship between certain pure-tone threshold measures and FEP.

In general, the results of the auditory tests are consistent with results cited in the literature. A hearing deficit of 30 to 40dB at the high frequencies has been reported in workers in European battery plants (Gammarota & Bartoli, 1964; Carducci & De Judicibus, 1961; Balzano, 1952). Valcie and Manojlovic (1969) studied the effects of carbon monoxide, lead, and carbon disulfide on the auditory threshold of workers from several different industries and found deficits in the 4000Hz to 8000Hz range. Of the 65 leaded workers tested, 9.1 percent had hearing losses although the amount of loss did not correlate with other symptoms of lead poisoning. With a few exceptions, ambient noise levels at the plants did not exceed 80dB. Similarly, Balzano (1952) studied 16 workers who had been exposed to lead fumes or dust for several years, or who showed clinical symptoms of lead poisoning, and found no deficit in hearing low tones (below 512Hz), a mild deficit in the middle tones (512Hz, 1024Hz, and 2044Hz), and large deficits in the high tones (above 2044Hz). Koch and Serra (1962) found impairment only above 2000Hz in 12 workers exposed to tetraethyl lead. However, their subjects showed normal audiograms below 2000Hz; the authors found the damage to be of cochlear origin, though how this determination was made is not clear. Since the workers were all between 26 and 45 years of age, presbycusis was not responsible for the hearing loss. Finally, Atchabarov, Moshkevich, and Pyataev (1967), in a study of 41 leaded workers, found that 15 percent of the workers tested had deficits; those showing such losses had good sound perception at the medium frequencies but poor hearing at the low and high frequency ranges.

There are no published, epidemiologic studies which investigated auditory functions in lead workers and which report no effect (see Repko & Corum, 1976). Moreover, Repko (1977) reported that the average hearing levels of workers with PbB above 70 μ g/100ml, *relative* to the hearing levels of workers below 70 μ g/100ml, were higher. The Repko, *et al.* (1975) study suggested that increases in hearing level were significantly correlated with decreases in ALA-D, thereby indicating that as lead absorption is increased there is a commensurate loss of auditory function. No definitive relationships were noted in the present study between ALA-D and hearing level; FEP did show a clear positive relationship to an increase in hearing level in the right ear. It is suggested that perhaps this significant relationship emerged because FEP varies over a broad numerical continuum even at low level exposure and because FEP is a good measure of the apparent early *effect* on the hemopoietic system of exposure rather than exposure to lead per se.

It is clear from the present study data that the pure-tone thresholds of exposed workers are consistently higher for both the right and left ears at the frequencies tested. Of these differences in pure-tone threshold, 35 percent were statistically significant. These data are further enhanced by the results from the tone-decay test which demonstrate that at threshold and at 5dB above threshold, the lead workers exhibited a greater amount of decay than non-lead workers. Normal functioning of the auditory system should not produce tone decay; Glorig (1965) has indicated that for low intensity tone levels, a loss in audibility may be indicative of retrocochlear pathology. Based upon the group data of the auditory tests as well as the clinical

neurological examination, both conductive and psychogenic hearing impairments do not appear to be causative factors. The observed hearing loss is most probably sensorineural, although a central hearing loss cannot be eliminated completely (see Balzano, 1952). Sensorineural hearing loss may be attributed to various factors, including drug toxicity (Anticaglia, 1973). Corroborative evidence of auditory pathology has been demonstrated in young guinea pigs who received repeated peritoneal injections of lead acetate. Gozdzik-Zolnierkiewicz and Mosznski (1969) examined the sensory cells of the inner ear; however, no histopathological changes of the spiral and vestibular ganglion cells were observed. On the other hand, examination of the VIII cranial nerve (auditory nerve) of the lead poisoned animals showed segmental demyelination and axonal degeneration. The demyelinating neuropathy noted in this study is identical to the demyelination in peripheral nerve fibers (Fullerton, 1966; Seppäläinen & Hernberg, 1972) and in nerve fibers of the brain and spinal cord (Sauer, Zook, & Garner, 1970; Krigman, Druse, Traylor, Wilson, Newell & Hogan, 1974) evidenced in chronic lead poisoning.

The second important behavioral finding relates to the visual reaction time test. Reaction times of lead intoxicated workers have been compared to those of non-exposed workers by various Soviet and Eastern European scientists (Jacobson & Seiter, 1972). Increased reaction times have been reported in leaded workers in response to spoken words or other auditory stimuli (Cupcea, Raucher, Derevenco, Deleanu, Pop, & Gross, 1954; Boyadzhiev, Stoev, & Petkov, 1962), to visual stimuli (Boyadzhiev, *et al.*, 1962), and to electrical stimuli (Cupcea, *et al.*, 1954; Timofeev, Spivak, Deinichenko, 1955). The results showing increased reaction times are consistent with findings noted in the literature. The particular motor response involved in the visual reaction time test requires control by the ulnar nerve. The ulnar nerve is the primary motor nerve responsible for lateral movement of the fifth finger. It is also quite interesting that a significant negative relationship was obtained between increases in reaction time and decreases in the maximal motor conduction velocity of the ulnar nerve.

Such findings are impressive, they provide important support to the notion that data derived from behavioral toxicology methods should be utilized in establishing the health status of groups of individuals regularly exposed to lead. The extent to which the neurobehavioral dysfunctions noted in this study and in other epidemiologic studies contribute to increased accidents must be investigated further. From the limited information gained in this study involving accidents, it can be said that the lead workers in the sample group did show significantly more accidents than the workers in the control group. It is clear, in summary, that lead exposure, even at PbB levels substantially below 80µg/100ml, may result in various interrelated neurobehavioral dysfunctions; the consequences of such dysfunctions are to detrimentally affect the performance of tasks or jobs involving motor responses.

RECOMMENDATIONS AND CONCLUSIONS

The use of a number of well-known neurotoxic materials is involved in current industrial practice. These materials range from organic solvents used in degreasing operations, for example, to the heavy metals used in foundries and smelters and in product manufacturing. The adverse effects on the nervous system of acute and chronic exposure to neurotoxic materials are well documented (Seppäläinen, 1977; Repko, 1977; Seppäläinen, *et al.*, 1975; Repko, *et al.*, 1975, 1976; Stewart, *et al.*, 1974; Chaffin, *et al.*, 1973). In the long run, research should be directed toward the development of common tests sensitive to behavioral impairments and associated neurophysiological disturbances caused by exposure to a variety of chemical and physical agents at the workplace. In the short run, however, development is based on the evaluation of functional capacity in select worker groups subjected to specific neurotoxic agents. It is from these specific data that general tests of functional disorder are constructed.

Based on the data of this study and others (see Repko & Corum, 1976; Repko, 1977; Repko, *et al.*, 1975; Seppäläinen, 1977; Seppäläinen, *et al.*, 1975), the adverse effects of occupational exposure to inorganic lead on certain functional capacities have been demonstrated. It is evident from the data that subtle, quantifiable behavioral and neurological effects are evidenced at levels below the accepted biologic guideline of 80 µg Pb per 100ml of blood. In the group of lead workers with a mean blood lead of 46 µg per 100ml, it was clear that deficits occur in the NCV of the peripheral nerves and that these deficits are in the magnitude of five to nine meters per second. Moreover, in this same group of workers, deficits in visual reaction time, under response control of the ulnar nerve, as well as deficits in auditory functioning, in terms of both pure-tone thresholds and tone-decay, were all adversely affected by low-level lead absorption.

In conclusion, it is believed by these authors that the data obtained in this study further confirm the previous findings reported in the literature concerning the behavioral and neurological effects of occupational exposure to inorganic lead. The task of evaluating the effects of low-level exposure to inorganic lead is by no means complete. Although the data of this study answered many questions concerning both behavioral and neurological functional impairment in *groups* of workers, questions concerning the progression of such effects and their reversibility in *individual* workers must be investigated. The long-range goals of these neurobehavioral toxicology methods are to assess the effects of toxic agents, such as inorganic lead, in the pre-clinical or clinical stage of disease, and to provide the occupational physician a more complete picture of the total health significance of specific occupational exposures.

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APPENDIX A

AN ELECTRONEUROMYOGRAPHIC EQUIPMENT DESIGN FOR USE WITH THE METHOD OF PARTIAL ANTIDROMIC BLOCKING IN PERIPHERAL NERVES

Recent attention in occupational hygiene in the area of industrial lead exposure has led to the development of a method of partial antidromic blocking to determine the conduction velocity of the slower nerve fibers of the ulnar nerve (Seppäläinen, 1971, Seppäläinen & Hernberg, 1972). This method is a modification of a technique described by Hopf (1962). Since the slower conducting fibers of peripheral motor nerves are first susceptible to damage in some neuropathies, namely lead poisoning, it appears appropriate to employ this technique to detect subclinical nerve damage in individuals without clinical neurological symptoms.

Basically this technique involves delivering paired supramaximal stimuli of 0.3-1.0msec duration almost simultaneously to separate points along the pathway of a nerve. To determine the conduction velocity of the slower fibers (CVSF) of the ulnar nerve, the distal stimulation point is approximately 2cm above the wrist fold and the proximal stimulation point is approximately 5-7cm above the sulcus ulnaris. When simultaneous stimuli are delivered, the antidromic volley originating from the distal stimulus stops or inhibits the orthodromic volley originating from the proximal stimulus, therefore only allowing one muscular response. However, when the proximal stimulus is delayed, two separate muscular responses may be obtained. Surface electrodes placed on the tendon and belly of the abductor digiti minimi muscle are used to pick up the responses which are then amplified by an electromyograph.

Beginning at a point in which two responses are recorded, the interval in time between deliverance of the two stimuli is continuously reduced until the second response just begins to decrease. At this point some fibers are beginning to be inhibited by the antidromic volley and the latency of the slow fibers between the points of stimulation may be obtained. The CVSF is determined by recording the shortest interval between *full* responses, subtracting 1ms, (representing the refractory period of the nervous fibers) and then dividing the distance between stimulation points by the latency time. Figure A-1 shows oscilloscope tracings obtained to determine the conduction velocity of the slower fibers of the ulnar nerve. On the upper half of the screen the first deflection represents the distal response and the second deflection represents the proximal response. These are obtained to verify register of both responses and to assure that both responses are matched as closely as possible in configuration and amplitude. On the lower half of the screen both responses are presented with the distal response being shown at the first reduction in amplitude after gradually decreasing the interval between delivery of the two stimuli. In Figure A-2 the proximal response in the lower trace is shown at the point which measurements are taken to determine CVSF. This represents the shortest interval between delivery of the two stimuli in which there is no change in the proximal response.

Seppäläinen (personal communication) in her studies uses a two-channel Disa Electromyograph with a built-in Ministim unit and a Disa Multistim

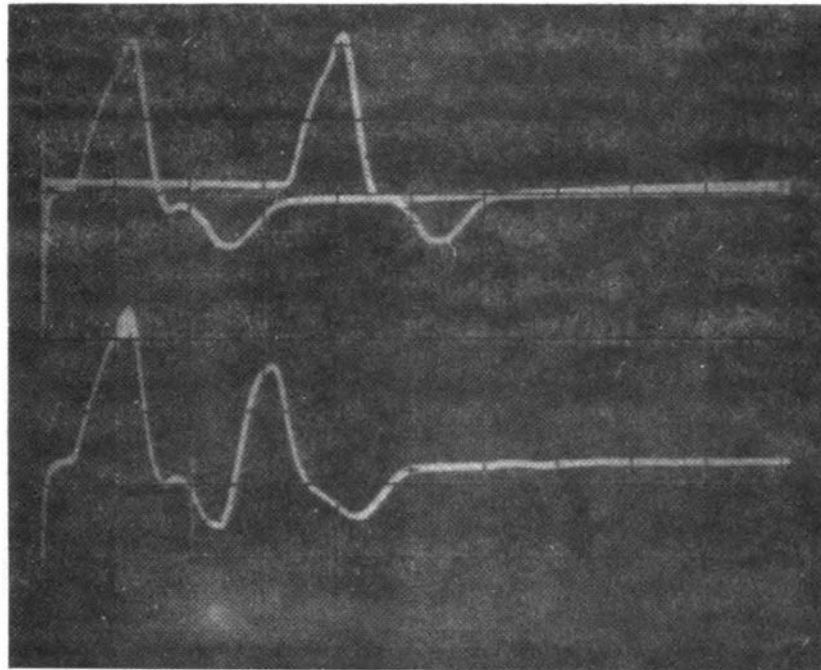


Figure A-1. Oscilloscope tracings for determining the conduction velocity of CVSF. On the upper half of the screen the first deflection represents the distal response and the second deflection represents the proximal response. Both responses are again presented with the distal responses being shown at the first reduction in amplitude after decreasing the interval between stimuli.

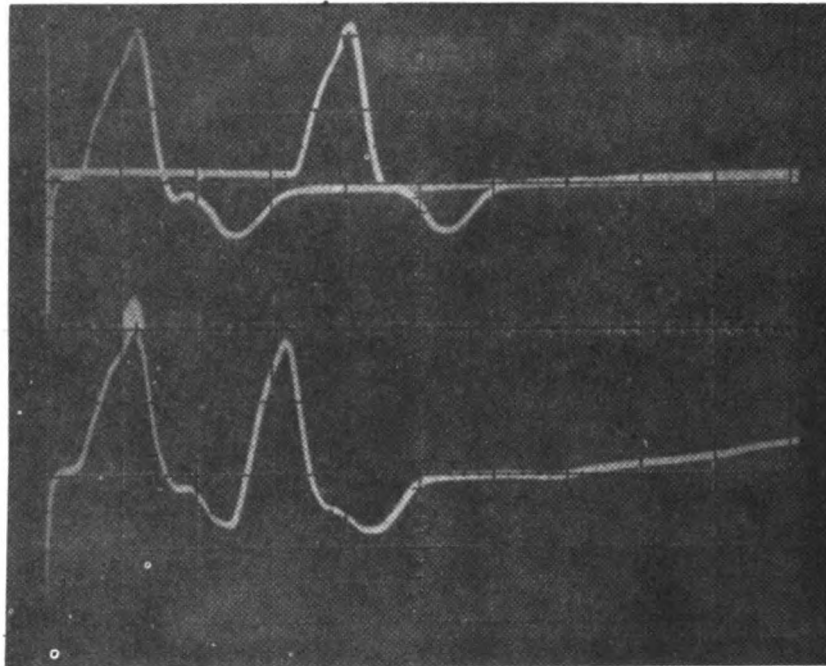


Figure A-2. The proximal response in the lower trace is shown at the point which measurements are taken to determine CVSF. This represents the shortest interval between delivery of the two stimuli in which there is no change in the proximal response.

capable of delivering two stimuli at two different points for determining CVSF. It is the opinion of these authors that a more versatile, less expensive, and in the case of field studies, a more easily portable piece of equipment may be employed. The following equipment described (Figure A-3) was implemented for this research project.

The oscilloscope employed in our equipment is a Tektronix Model R/7313 with a Model 7A22 differential amplifier and a Model 7B53A dual time base plug-in module. This oscilloscope is a solid-state instrument, with three plug-in compartments and a bi-stable cathode ray storage tube having an 8 x 10cm display area divided into two areas divided into two 4 x 10cm storage screens. The storage screens may be independently controlled, thus enabling split screen application. In addition, a modification to place the plus-gate signal from the output board to the card connectors enabling step-raster function was found to be advantageous when determining neural conduction velocities.

The plug-in differential amplifier, comprising one plug module, is DC coupled and has excellent low noise characteristics and high gain for low-level application. The DC offset capability permits the display of very small low-frequency signals containing a large DC component at deflection factors that would not be obtainable with AC coupling. The vertical deflection factor range of this amplifier is from 10 μ V to 10V. To set the bandwidth of the instrument, the high and low frequency -3db points may be easily selected, therefore in low frequency applications the signal-to-noise ratio can be improved by restricting the bandwidth.

The dual time base comprising the second plug-in module provides main intensified, delayed, and mixed sweep operations. Calibrated sweep rates from 5 seconds per division to 50 nanoseconds per division and triggering to 100 megahertz are available. A 0 to 10 continuous sweep delay, variable main and delayed sweep rates and variable main sweep holdoff are also provided in this unit. Separate triggering controls are installed for main and delayed sweep triggering and when operating in the automatic main triggering mode, a bright base line is displayed on the scope when a trigger signal is absent. Modified control circuits necessary for the correct implementation of this equipment are arranged in the third plug-in module.

To produce the stimulus, a Grass S-88 Stimulator with accessory foot-switch is used. It has independent four parameter control of two outputs which are necessary for measuring CVSF. It is capable of delivering DC, single, repetitive, twin pulses, pairs of unlike pulses, trains of pulses from one or both outputs plus pre-mid or post-train pulses. The design of this stimulator allows a single arrhythmic pulse, either within a train of pulses or during continuous stimulation and synchronous inputs and outputs allow it to drive or be driven by another stimulator, external source or to synchronize the cathode ray oscilloscope (CRO) trace. The outputs of this stimulator are switch controlled to allow either or both pulses to be delivered. At the point in which the first pulse is originated in the stimulator, a synchronizing signal is sent to the CRO external trigger input. However, it is important to disable the DC continuous output of this stimulator at both output switches to prevent accidental lethal shock delivery to a patient

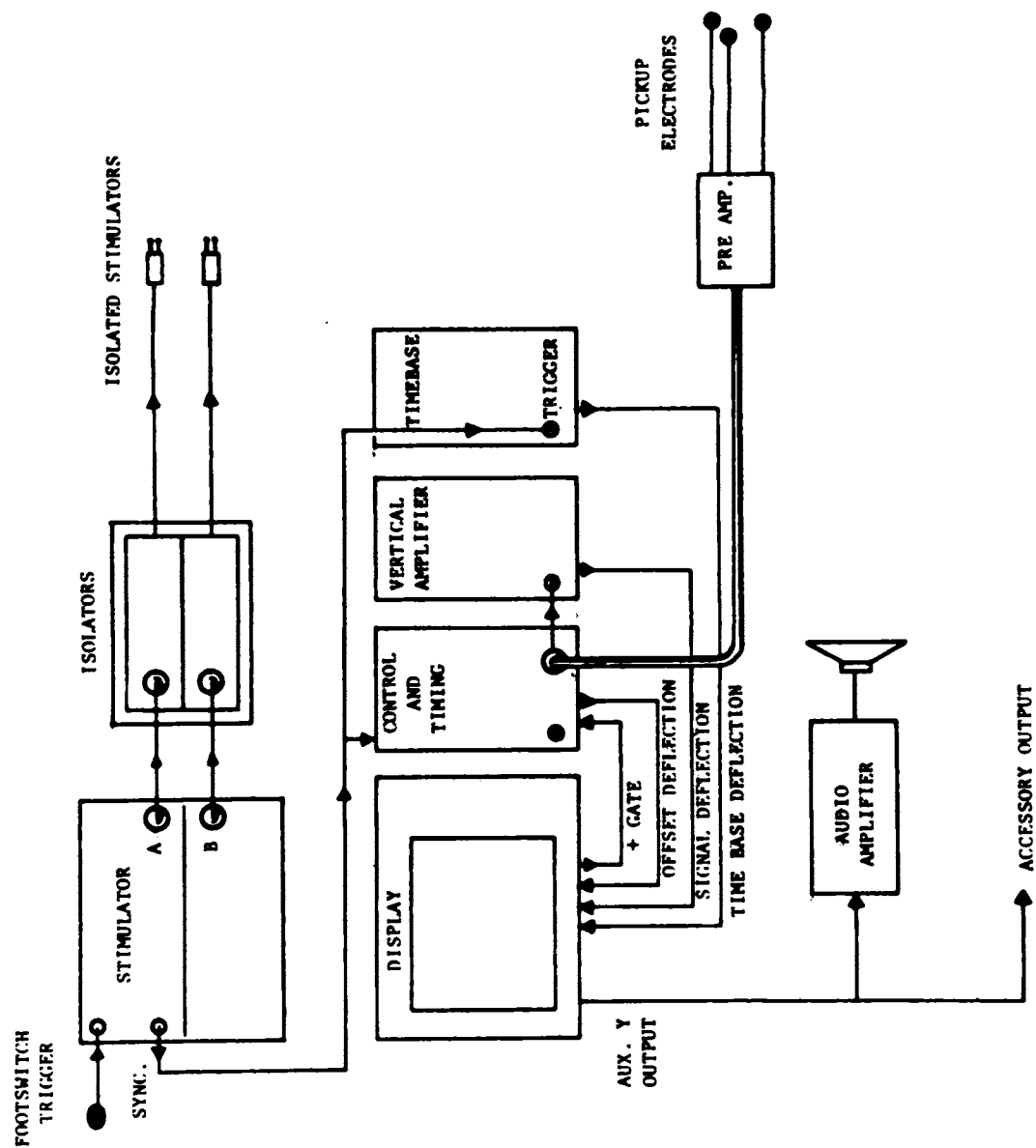


Figure A-3. Block diagram of the electromyographic equipment.

or subject. The overall parameter calibration of this instrument is better than 5 percent.

By using two Grass SIU 5-300 stimulus isolation units, true biphasic pulses are obtainable while an extra margin of patient safety preventing a shock-hazard risk and a reduction in stimulus artifact during the recording of an electrical response to stimulation are insured. These units have an upper delivery capacity of 300V, transistorized radio frequency circuits and are powered by stimulus pulses thus eliminating the need for any additional power source.

The trace offset circuits and marker generator circuit are enclosed in the control circuit plug-in module and are transistor-transistor logic (TTL) with the operational amplifier circuits being powered by the main-frame of the oscilloscope. The trace offset circuits allow a trace to be displayed in the top portion, center or lower portion of oscilloscope screen by switch selection. In the case of repetitive traces, they may be displayed on the top portion, the bottom portion, or only the middle portion by switch selection. Further offset circuitry provides trace marker deflection when a timing marker is desired. The switch selection greatly speeds up recording since the oscilloscope would otherwise repeatedly require tedious adjustment of control knobs to correctly position each trace. Included also are circuitry to accept the signal from a tape recorder and display the signal on the oscilloscope screen from sources other than the pre-amplifier. The marker generator is actuated by an on-off toggle switch and accepts the pulse from the pulse-one-trigger output of the stimulator to start a linear ramp generator which is compared with the output of a digital knob type, linear potentiometer graduated in milliseconds. This comparison provides a pulse when the ramp potential equals the potentiometer potential and thus supplies the calibrated marker signal to the offset circuits for purposes of timing the latency of neural responses.

The pre-amplifier is patterned after the instrumentation amplifier described by Huntsman and Nichols (1971). With modifications in the input circuitry, this amplifier allows large unbalanced source resistances with no degradation in common mode rejection. To obtain + 10B common mode input characteristics, two voltage followers buffer the input signal and drive a balanced differential amplifier. The resistors are matched to 0.1 of 1 percent to provide the maximum common mode rejection. The voltage gain of the pre-amplifier is approximately 1000 with input impedance of the amplifier as modified being greater than 10 megohm. High frequency response is set by the feedback configuration at 20 kilohertz for the -3dB point and if no unusual frequency compensation is necessary the roll-off is approximately six decibels per octave above this frequency. The low frequency cutoff is set by the input DC circuits at .3 hertz and may be increased by changing settings of the vertical amplifier to the oscilloscope. The harmonic distortion is less than 0.05 percent. Adequate by-pass of the components is essential to prevent any interaction of active components, although filter power is provided from the plug-in compartment of the oscilloscope. This pre-amplifier is physically located on a printed circuit board enclosed in a 5 x 7 x 2cm "Bud Box" attached to the EMG unit by a twin-paired, shielded cable. The input wires are connected to input plugs leading directly to the

circuit board. It is concluded that this circuit is excellent for the precise measurement required in electroneuromyographic research. It requires only an occasional rebalancing to set quiescent DC input to ground level.

For determining neural motor conduction velocities and CVSF, Teca 6030 surface electrodes are used for recording and a Teca 6008 electrode is used for ground. For recording sensory neural conduction velocities, lead wire electrodes, 1.6mm in diameter x 16cm in length and connected to an input lead are employed to wrap around the preferred digit. Both types of electrodes and the ground are easily connected to the pre-amplifier by jack plugs. To deliver the stimuli, two separate hand-held stimulators enclosed in 2.8cm x 5cm x 1.25cm plastic oases with 4.5mm diameter brass probes, 2cm apart extending 4.5cm are used. These are connected to the isolation units with twinpaired, shielded cables.

If this unit is used for electromyography, an audio-amplifier has been installed which receives the signal from a "Y" auxiliary output on the oscilloscope. It is a simple audio I C circuit with a potentiometer, on-off switch, and a capacitively coupled 10.2cm x 15.2cm, 8ohm speaker. This LM-380 circuit has a sound level that is adequate with minimum components. The "Y" auxiliary output of the oscilloscope is also the location for the signal input of a tape recorder which may be operated simultaneously with the audio-amplifier. The oscilloscope and stimulator are mounted in a 54cm wide x 54cm deep x 43cm tall metal cabinet and weighs 44.5kg. The isolation units are mounted in a separate, 23cm wide x 12.7cm deep x 15.2cm tall metal box which weighs 1.8kg. Therefore the size of the equipment and the solid-state circuitry provides easy two man portability.

APPENDIX B

REQUIRED FORMS

Each of the forms requesting information from the workers participating in this study is included in this appendix. Each worker was required to read and sign the consent form; this form describes the purpose of the study, confidentiality of information, extent of testing, release of test results and employee protection. Also, each worker was asked to read and sign (if they so desired) the form providing for the release of Company Medical Records and the release of Study Results to a designated physician; each subject was also asked to read the Assurance of Confidentiality statement. The Employee Questionnaire was completed by each subject volunteer on the day prior to his scheduled testing. Finally, the protocol which was followed for the Neurological Examination is also included.

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
Cincinnati, Ohio 45226

PART I. PROJECT DESCRIPTION

Project Title and Number: Effects of inorganic lead on
behavior and neurologic function;
DHEW Contract No. 210-75-0054

Project Director: Dr. John D. Repko
Performance Research Laboratory
Telephone: (502) 588-6571

Project Sponsor: Dr. Barry L. Johnson
National Institute for Occupational
Safety and Health
Telephone: (513) 684-8386

Purpose and Benefits: The major purpose of the proposed
contract is to identify behavioral
and neurological changes associated
with chronic exposure of workers to low levels of inorganic lead
and to examine the usefulness of these measures as early warning
indicators of potential adverse effects on worker safety and
health. Information generated from this contract study will aid
in detecting early reversible functional changes in workers ex-
periencing long-term exposure to inorganic lead. These effects
of inorganic lead may be significant in terms of human capability
to work safely and/or may be used as early indicators of clinical
disease. Findings will be meaningful in terms of evaluating
the adequacy of recommended safe exposure levels and for rating
risk of safety-health problems at the job site.

II. CONSENT TO PARTICIPATE

I, _____, age _____, hereby voluntarily
agree to cooperate in the above name study and to undergo the
tests listed in Attachment A. The study has been discussed with
me and I have been given a copy of this document. I understand
that:

1. The procedures and tests to be followed are as stated in
Attachment A with those procedures which are experimental
so identified.

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2. Attendant discomforts and risks are as noted in Attachment A and, except as noted, are minimal and provision has been made for any necessary medical care, and I have been told what to do if I have any reaction.
3. Benefits are as indicated in the Purpose and Benefits section in Part I.
4. If alternative procedures advantageous to me are available, they are specified in Attachment A; and if they become available during the project, the procedure most advantageous for me will be indicated and used or an explanation will be given to me as to use of any other procedure.
5. My inquiries will be answered by the project director or other personnel involved in the project or by Dr. John Repko, whose phone number is 502-588-6571.
6. I am free to terminate my consent and to discontinue participation in the project at any time without prejudice to myself.
7. My identity and my relationship to any information (1) disclosed by me in completing any project questionnaire and (2) reported by me or derived from me during my participation in the above named project shall be kept confidential and will not be disclosed to others without my written consent except as required by law and except that such information will be used for statistical and research purposes in such a manner that no individual can be identified. I understand that if any information is found out concerning me that can endanger the health and safety of others, this information will be given to the proper authority.
8. If any of my medical records are required for purposes of this project, a separate written consent for release of the records will be requested from me.
9. There may be questions that I must answer, and my inquiries concerning the questions will be answered by Dr. John Repko, whose phone number is 502-588-6571.

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SIGNATURE _____ DATE _____
Subject

SIGNATURE _____ DATE _____
Parent or Guardian

INVESTIGATOR _____
Name, Title and Signature

III. REQUEST AND AUTHORIZATION FOR RELEASE OF INFORMATION

I, _____, hereby request and authorize the Project Director to inform the following physicians whose names and addresses I have entered below of any significant findings from the above named study concerning me. (Do not leave blank. Write "No" where you do not wish to give a name and address.)

1. My Personal physician(s):

Dr. _____

Street: _____

City: _____

2. (Company), (Other) Physician:

Dr. _____

Street: _____

City: _____

Signature _____ Date _____

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- IV. If a project questionnaire is required, it will constitute this Part IV as a separate attachment to be retained by the Project Director. A copy of the questionnaire is not retained by the participant.

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ATTACHMENT A

- A. Project Title and Number: Effects of Inorganic Lead on Behavior and Neurologic Function.
No. 210-75-0054.
- B. Procedures and tests which involve human subjects in conduct of subject project are as follows:
1. Study tests:
 - (a) A work history questionnaire must be answered;
 - (b) samples of blood, and urine will be required for monitoring purposes and for special clinical tests;
 - (c) performance tests will be given to measure hearing acuity, tremor, eye-hand coordination, strength and endurance, reaction time, and visual functions.
 - (d) psychological tests will be given to measure my present mood and subjective feelings; and
 - (e) all performance tests will be conducted immediately after my regular work-shift and will require approximately 1 1/2 to 2 hours for completion.
 2. Medical Examination:
 - (a) A neurological examination of peripheral nerves conducted by a certified neurologist and which will involve electrical stimulation of nerves in an arm and leg;
 - (b) an electromyogram (muscle electrical activity) will be required of each participant and will be recorded using small needle electrodes inserted under the skin.
 3. Compensation:
 - (a) Each participant will be paid for participation in the study.
- C. In accordance with the Privacy Act the following information is brought to the attention of all participants in this study:

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1. The information in this survey is being collected under the authority granted by the Occupational Safety and Health Act of 1970. Your participation is completely voluntary.
2. The principal purpose for collecting this information is to investigate the toxicity of inorganic lead exposure.
3. The information collected during this study will be used only for composite data reporting. All information will be reported in summary statistical form so that no individual can be identified.
4. Your decision not to supply any or all of the requested information will have no effect on your job status, health, or well-being. You are free to terminate your consent and to discontinue participation in the project at any time without prejudice to yourself.

NOTE: INSTRUCTIONS TO PROJECT DIRECTOR OR CONTRACTOR OR GRANTEE.

- A. A fair explanation of the procedures and tests to be followed must be given, including:
 - (1) an identification of those which are experimental;
 - (2) a description of the attendant discomforts and risks;
 - (3) a disclosure of appropriate alternative procedures that would be advantageous for the subject and an explanation of why these are not used, if appropriate.
- B. Section II represents the minimum information a subject should receive and additional sheets to this attachment should be used to include any other relevant matter.

O.M.B. No. 68-S76014
(Expires Dec. 1977)

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Center for Disease Control
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Project Title: Effects of Inorganic Lead on Behavior and Neurologic
Function

Assurance of Confidentiality

Your identity and relationship to any information in our possession disclosed by you through this questionnaire will be kept confidential and will not be disclosed without your written consent in accordance with Public Health Service Regulation 42 Code of Federal Regulations Part 1 except that such information will be used for statistical and research purposes in such a manner that no individual can be identified.

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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EFFECTS OF INORGANIC LEAD
ON BEHAVIOR AND NEUROLOGIC FUNCTION

Plant Code: _____ Employee Code: _____

PART IV EMPLOYEE QUESTIONNAIRE

1. Name (please print): _____
(Last) (First) (Middle)

Your Mailing Address: _____
(Street) (City) (Zip)

2. Age: _____ (years) 3. Height: _____ (inches)
Weight _____ (pounds) 5. Sex: M F

6. Circle highest grade completed:

1 2 3 4 5 6 7 8 1 2 3 4 1 2 3 4
Pre-high school High school College

7. Name of plant where you work: _____

8. Job title: _____

9. How long have you worked for this company? _____ (years)

10. How long have you worked in your present job? _____ (years)

11. For what other companies have you worked during the last 20 years?

	<u>Name of Company</u>	<u>Job Title</u>	<u>How long (years)</u>	<u>Years Worked</u>
				From: _____ To: _____
1)	_____	_____	_____	From: _____ To: _____
2)	_____	_____	_____	From: _____ To: _____
3)	_____	_____	_____	From: _____ To: _____

(Use back side of this sheet if necessary)

12. Did you work with any of the following chemicals in the companies you listed under question 11?

	<u>NO</u>	<u>YES</u>	<u>DON'T KNOW</u>	<u>Name of Company</u>
a) Metallic mercury	()	()	()	_____
b) Methyl mercury	()	()	()	_____
c) Carbon disulfide	()	()	()	_____
d) Vinyl chloride	()	()	()	_____
e) Manganese	()	()	()	_____
f) Arsenic	()	()	()	_____
g) Thallium	()	()	()	_____
h) Acrylamide	()	()	()	_____
i) Methyl bromide	()	()	()	_____
j) Carbon monoxide	()	()	()	_____
k) Inorganic or organic lead	()	()	()	_____
l) Chloroform	()	()	()	_____
m) Carbon tetrachloride	()	()	()	_____
n) 1,1,1-Trichloroethane	()	()	()	_____
o) 1,1,2-Trichloroethane	()	()	()	_____
p) <i>sym</i> -Trichloroethane	()	()	()	_____
q) Trichloroethylene	()	()	()	_____
r) Tetrachloroethylene	()	()	()	_____
s) Tannic acid	()	()	()	_____
t) Other solvents Name _____	()	()	()	_____
u) Pesticides Name _____	()	()	()	_____

13. Were any of the chemicals listed in question 12 used at all in the companies you worked for in the last 10 years?

	<u>Chemical</u>	<u>Name of Company</u>
a)	_____	_____
b)	_____	_____
c)	_____	_____

14. Did you work with inorganic lead or were you exposed to inorganic lead in the companies you listed under question 11?
- | <u>NO</u> | <u>YES</u> |
|-----------|------------|
| () | () |

15. Are you exposed to inorganic lead in your present job?
- | | |
|-----|-----|
| () | () |
|-----|-----|

16. Describe the location within the plant in your present job _____

a) What is your department number (if any)? _____

17. Do you shower at the plant when your shift is over? () ()

18. About how much of your time on the job do you wear a respirator? (Circle closest answer)

0% 10% 25% 50% 75% 100%

19. Have you been involved in a work accident at this plant? () ()

a) If yes, when? _____(year)

b) If yes, how many? _____

c) If yes, causes _____

d) If yes, how many total days work did you lose? _____

_____ (days)

20. Have you been involved in work accidents at a previous employer's plant during the last 10 years? No YES
() ()
- a) If yes, name of company _____
- b) If yes, causes _____
- c) If yes, extent and type of injuries _____

- d) If yes, how many total days of work did you lose? _____
21. Have you ever smoked cigarettes, cigars or a pipe regularly? If your answer is 'no' go to question 25. () ()
22. Started smoking: _____(year)
23. Quit smoking: _____(year)
24. Circle how many cigarettes per day you smoked:
- a) less than 5
b) 1/2 pack
c) 1 pack
d) 1 1/2 packs
e) 2 or more packs
25. Do you sleep well? () ()
26. Which of the following reflects your attitude towards meals?
- a) They are a necessary evil _____
b) They are nothing exciting _____
c) No particular feeling toward them _____
d) Sort of enjoy them _____
e) Thoroughly enjoy them _____
27. Do you take vitamin supplements? () ()

28. Is your appetite NO YES
- a) Very good? _____
- b) Good? _____
- c) Average? _____
- d) Poor? _____
- e) Very poor? _____
29. Are you on a special diet? () ()
- Brief description: Weight Watchers _____
- Other _____
30. Approximately how long does it take you to go to sleep at night?
- a) 15 mins or less _____
- b) 15-30 mins _____
- c) 30-60 mins _____
- d) Longer than an hour _____
31. When do you feel you are most alert?
- a) 1-5 am _____
- b) 6-10 am _____
- c) 11 am-3 pm _____
- d) 4-8 pm _____
- e) 9-12 pm _____
32. How often do you wake up while sleeping?
- a) Don't wake up until time to get up _____
- b) 1-2 times _____
- c) 3-4 times _____
- d) More than 4 _____
33. Do you know why you wake up during your sleep? () ()
- If yes, why (for example, neighbor slams doors at 3 am every night)?
- _____
- _____
34. How do you feel when you get up?
- a) Completely rested _____
- b) Somewhat rested _____
- c) Tired _____
- d) Very drowsy _____

35. During a "typical" week, how much of the following do you drink? If your answer is 'none', skip questions 36 through 39.

a) Beer:

- 1) None _____
- 2) 1 or 2 glasses _____
- 3) 3-6 glasses _____
- 4) 7-12 glasses _____
- 5) More than 12 glasses _____

c) Whiskey or other liquor:

- 1) None _____
- 2) Less than 1 drink _____
- 3) 1-2 drinks _____
- 4) 3-5 drinks _____
- 5) 6 or more drinks _____

b) Wine:

- 1) None _____
- 2) 1 or 2 glasses _____
- 3) 3-6 glasses _____
- 4) 7-12 glasses _____
- 5) More than 12 glasses _____

36. When do you normally drink your favorite alcoholic beverage?

- a) Before work _____
- b) After work _____
- c) Weekends _____
- d) With meals _____
- e) Other (please indicate) _____

37. When was the last time you had an alcoholic beverage of any type?

- a) Within 6 hours _____
- b) 6-12 hours _____
- c) 12-24 hours _____
- d) 1-5 days _____
- e) More than 5 days _____

38. How long have you been drinking the amount indicated in question 35?

- a) Less than 1 month _____
- b) 2-6 months _____
- c) 7-12 months _____
- d) 1-3 years _____
- e) 4 or more years _____

39. Have you ever sampled or drunk moonshine
(white lite'ning, etc.)?

NO
()

YES
()

- If yes, a) how much? _____
b) when was the last time? _____

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EFFECTS OF INORGANIC LEAD
ON BEHAVIOR AND NEUROLOGIC FUNCTION

Employee Code: _____ Plant Code: _____

PART V. NEUROLOGICAL EXAMINATION

Patient _____
(Name of subject volunteer)

Examined by Dr. _____

Date _____

SYMBOLS: (✓) Normal; (*) Abnormal; (0) Absent;
(#) Decreased; (##) Increased; (-) Not Tested

MENTAL: Mood _____ Orientation _____ Memory _____ Judgment _____
Intelligence _____ Cooperation _____

CRANIUM: Symmetry _____ Size _____ Tender _____ Bruit _____

SPINE: Movement _____ Scoliosis _____ Kyphosis _____ Lordosis _____
Tender _____

CRANIAL NERVES:

- I. Smell _____
II. Visual Acuity: O.S. _____) .D. _____ Visual Fields _____
Fundus _____
III, IV. Oculomotor Apparatus: Movements _____ Convergence _____
Ptosis _____
VI. Diplopia _____ Nystagmus _____
Pupils: Size - Left _____ Right _____ Shape - Left _____
Right _____
Reaction to Light _____ To Convergency _____ Hippus _____
Ex- or Enophthalmos _____
V. Sensory: Touch _____ Pain _____
Motor: Jaw Movements _____ Nutrition _____

- VII. Facial Movements: Volitional _____ Emotional _____ Palpebral
Fissure _____ Nasolabial Fold _____ Tremor _____
- VIII. Hearing: Air conduction (watch or tuning fork) _____ Bone
Conduction _____ Weber _____ Tinnitus _____ Vertigo _____
Past Pointing _____
- IX, _____
- VII. Tast: Anterior 2/3 _____ Posterior 1/3 _____
- X. Swallowing _____ Palatal Movement _____ Voice _____
- XI. Trapezius _____ Atrophy _____ Fibrillation _____ Tremor _____
- XII. Tongue Movement _____ Atrophy _____ Fibrillation _____
Tremor _____

MOTOR SYSTEM:

General: Occupation _____
Right or Left Handed _____ Speech _____ Dysphasia _____
Posture _____ Gait _____ Sphincters _____ Involuntary
Movements _____ Associated Movements _____ Convulsions _____
Romberg _____

Arms: Posture _____ Muscle Strength _____ Consistency (flaccid,
spastic) _____ Nutrition (atrophy, hypertrophy) _____
Irritability _____
Finger-Nose Test: Eyes open _____ Eyes closed _____ Dysmetria
(rebound phenomenon) _____ Asynergia _____ Tremor _____
Involuntary Movements _____

Legs: Posture _____ Muscle strength _____ Consistency (Flaccid,
spastic) _____ Nutrition (atrophy, hypertrophy) _____
Irritability _____
Heel-Shin Test: Eyes open _____ Eyes closed _____ Toe-object
Test _____ Tremor _____ Involuntary Movements _____

Trunk Muscles: Shoulder Girdle _____ Chest _____ Abdomen _____
Pelvic Girdle _____ Lateral Postural Reactions _____ Forward
P.R. _____ Backward P.R. _____ Rising from Bed _____ Rising from
Chair _____ Combined Flexion Test _____

REFLEXES: _____

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SENSORY SYSTEM: Light Touch _____ Pin Prick _____ Heat and Cold _____
Two Point _____ Vibration _____ Position _____
Stereognosis _____ Topognosis _____ Skin Writing _____
Tenderness _____ Achilles _____ Pain _____

SYMPATHETIC: Temperature of Extremities _____ Nails _____ Sweating _____
Dermatographia _____ Gooseflesh _____ Flushing _____
Palpitation _____ Color of Extremities _____

IMPRESSION: _____

* U.S. GOVERNMENT PRINTING OFFICE: 1976— 757-141/6776

