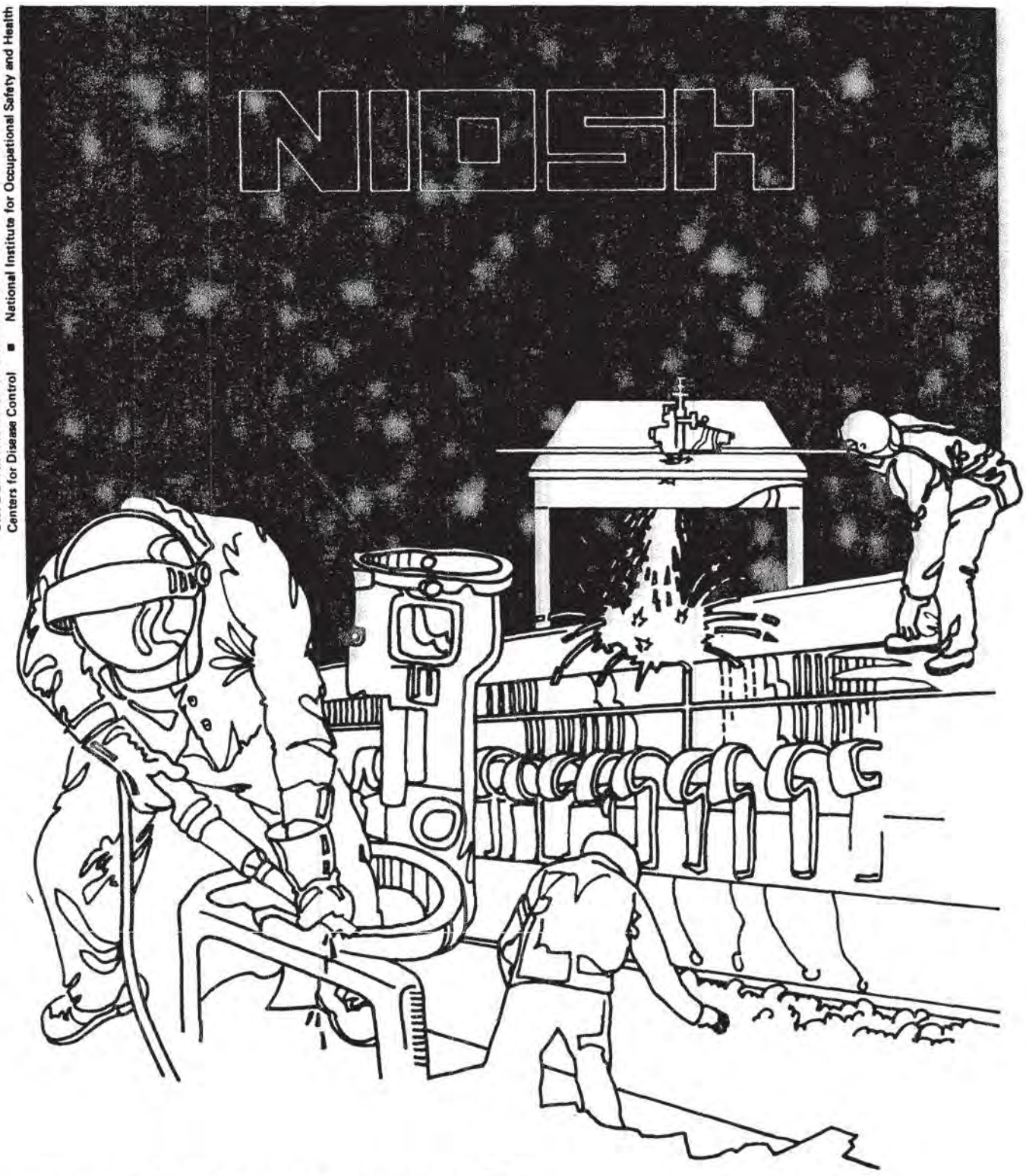
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES ■ Public Health Service
Centers for Disease Control ■ National Institute for Occupational Safety and Health



Health Hazard Evaluation Report

HETA 84-339-2052
WESTINGHOUSE ELECTRIC COMPANY
BLOOMINGTON, INDIANA

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

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WESTINGHOUSE ELECTRIC COMPANY
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I. SUMMARY

In May of 1984, the Indiana State Board of Health requested assistance from the National Institute for Occupational Safety and Health (NIOSH) to follow up workers occupationally exposed to polychlorinated biphenyls. These workers had participated in a cross-sectional medical study conducted by NIOSH in 1977[1]. Serum log PCB concentrations were quantified as lower chlorinated biphenyls, L-PCB, and higher chlorinated biphenyls, H-PCBs. By 1985, serum L-PCB concentrations in the Low exposure PCB group decreased by an average of 85% of the 1977 values. Serum L-PCB concentrations in the High exposure group decreased by an average of 90%. By 1985, the serum H-PCB level in the Low exposure and the High exposure groups had decreased by 39% and 58% respectively, of the 1977 values. No clinical abnormalities attributable to exposure to PCBs were observed. Serum PCB concentrations were positively and significantly correlated with triglycerides, cholesterol, total bilirubin, conjugated bilirubin, beta glucuronidase, 5-prime nucleotidase, serum apolipoprotein A1, serum apolipoprotein B, urinary creatinine, and urinary alanine aminopeptidase. When the prevalence of symptoms and overt clinical disease were investigated by exposure group, no differences between the groups could be ascertained except for a positive association with GGTP and a negative association with urinary creatinine.

This study documents the decrease in serum PCB concentration following cessation of exposure. The biochemical findings are indicative of PCBs physiological effect on lipidmetabolism, liver function and kidney function. The clinical significance of elevations in these parameters, eight years after the cessation of occupational exposures, remains unclear.

KEYWORDS: SIC 3629 (Electrical industrial apparatus, not elsewhere classified) polychlorinated biphenyls, PCB, Aroclor.

II. INTRODUCTION

On May 11, 1984, the Indiana State Board of Health (ISBH) requested assistance from the National Institute for Occupational Safety and Health (NIOSH) to follow up workers occupationally exposed to polychlorinated biphenyls (PCBs) at the Westinghouse Electric Corporation's Transmission and Distribution Components Division in Bloomington, Indiana. These workers had participated in a cross-sectional medical study conducted by NIOSH in 1977.[1] The ISBH had recently studied exposures and body burdens of PCBs among residents of Bloomington, Indiana. Concerns of potential adverse health outcomes resulting from occupational exposures to PCBs also had been expressed by past and present Westinghouse workers. In August 1985, investigators from the ISBH and NIOSH conducted a follow-up study of selected participants from the 1977 survey. In November 1985, all participants were notified of their serum PCB levels. In March 1986, all participants were notified of the results of all their medical tests.

III. BACKGROUND

The Westinghouse Electric Corporation's Transmission and Distribution Components Division in Bloomington, Indiana, began operation in the Fall of 1957. Although there are many different production operations at this facility, the capacitor manufacturing operation was the only one that used PCBs.

Large power capacitors were made at this facility. Bales of foil, paper, and plastic film were wound in a dust-free room where there was minimal exposure to PCBs. The size of the bale depended upon the size of the capacitor being made. A predetermined number of bales was then placed into a metal capacitor box, and the top of the capacitor, complete with connection bushings, was then put in place. A group of capacitors were then banded together and were placed in a vacuum chamber. The chamber was heated and placed under vacuum to remove all moisture from the capacitor. Impregnation of the capacitor consisted of admitting warm dielectric fluid (Aroclors 1242 and 1016, 42% and 41% chlorine by weight, respectively) under vacuum to the vacuum dried capacitor. This enabled complete filling of the entire capacitor. Impregnation procedures varied with the size of the capacitor. Large capacitors, those requiring several gallons of dielectric fluid, were filled manually through ports in the top of the capacitor. This operation resulted in a great deal of spillage and extensive dermal contact with the dielectric fluid by the workers. Following impregnation, the racks of warm, wet capacitors were transported to a

sealing station where the filling ports were soldered shut. They were then taken to a washing station for the removal of the fluid from the outer surfaces. An absorbent material was placed under the conveyor system to absorb the excess PCB fluid which commonly spilled onto the floor.

As a result of volatilization, condensation, dripping, and spillage, the capacitor impregnation and sealing operations created environments where significant portions of all exposed surfaces were wet with PCBs and where air levels in the immediate vicinity of these surfaces would become saturated with PCB vapors.[1] There were considerable opportunities for both dermal and respiratory uptake among individuals performing jobs related to these manufacturing operations.

Plant maintenance workers, through the nature of their work, spent considerable time in the impregnation, sealing, and washing areas of this plant. Although they were not directly involved in these operations, there was a great potential for exposure to flooring, absorbent materials on the floor, equipment, and other surfaces contaminated with PCBs.

Once the capacitors had been washed and painted, they then underwent quality control testing procedures. Numerous anecdotal reports have been provided, by past and present workers, of capacitors exploding when electrical test charges were applied, resulting in releases of PCB vapor. Capacitors which failed quality control tests were disposed of at three landfills in the city of Bloomington. Due to the labor-intensive operation of draining the dielectric fluid from the capacitor, rejected capacitors were disposed of while still containing their original quantity of dielectric fluid.

In 1976, PCBs were identified in Bloomington, Indiana, in sewage sludge which had been used by community residents as a garden fertilizer.[2] The source of the PCBs was traced to the Westinghouse Electric Corporation's Transmission and Distribution Components facility, which had been discharging its waste effluent to the municipal sewage system. In 1977, an epidemiological study of community residents with potential exposure to PCBs through use of the contaminated sludge[3], and of community residents without known unusual exposure to PCBs, was undertaken by the ISBH and the Centers for Disease Control.[3] A cross-sectional medical survey of the Westinghouse plant employees was undertaken by NIOSH in April 1977.[1] (Cross-sectional studies were also conducted by NIOSH on two smaller groups of workers exposed to PCBs in the maintenance and repair of electrical transformers at utility companies.)

In the 1977 cross-sectional study of Westinghouse workers, serum PCBs were measured on all participants, and quantitated as lower chlorinated biphenyls (L-PCBs) and higher chlorinated biphenyls (H-PCBs), which were, respectively, predominantly biphenyl molecules with four or fewer chlorine atoms per molecule (L-PCBs), and predominantly biphenyl molecules with five or more chlorine atoms per molecule (H-PCBs).[6] Serum L-PCB levels ranged from 1 to 3300 ug/l, and serum H-PCB levels ranged from 1 to 250 ug/l. Mean L-PCB and H-PCB levels among Bloomington residents, without occupational or known unusual exposure to PCBs, were 11.6 and 12.8 ug/l, respectively.[3] As summarized in the abstract of the 1977 report of the Westinghouse workers, "...statistically significant positive correlations of symptoms suggestive of mucous membrane and skin irritation, of systemic malaise, and altered peripheral sensation, were noted with increasing concentrations of serum PCB. No clinical abnormalities attributable to exposure to PCBs were observed. Serum PCB concentrations were positively and significantly correlated with serum glutamic-oxalacetic transaminase (SGOT), serum gamma-glutamyl transpeptidase (GGTP), and plasma triglyceride, and inversely correlated with plasma high density lipoprotein cholesterol. These findings are indicative of PCBs' physiological effect on the liver, whose long-range health significance is unknown..."[1]

In 1983, the ISBH requested the Centers for Disease Control (CDC) to assist them in studying exposure to and health effects from PCBs among residents near three waste sites in Bloomington, Indiana.[4] A survey was undertaken by the ISBH and CDC in 1984. Five participants in that survey had also participated in the 1977 NIOSH study of Westinghouse workers. Serum PCB levels in 1977 and 1984 were compared for those five individuals. From 1977 to 1984, serum L-PCB decreased between 89% and 94%, while serum H-PCB decreased between 14% and 53%.[5] Mean L-PCB and H-PCB levels among Bloomington residents without occupational or known unusual exposure to PCBs were 1.1 and 8.9 ug/l, respectively.[4]

In response to the ISBH's request to follow up workers studied by NIOSH in 1977, ISBH and NIOSH resurveyed participants from the 1977 study who had the highest and the lowest serum PCB levels measured in 1977. The study was conducted in August 1985.

IV. METHODS

The study was a follow-up of those participants whose serum PCB determinations in 1977 placed them in either the highest or lowest percentiles of the serum PCB distribution. Arbitrarily, the top and bottom ten percentiles were targeted for follow-up. To allow for

refusals to participate and problems in locating participants in the 1977 study who might have left employment, the top and bottom fifteen percentiles were solicited to participate in the study.

As noted above, in the study of community residents undertaken by the ISBH and CDC in 1984, five participants from the 1977 NIOSH study also participated. In the interval from 1977 to 1984, serum L-PCB levels had fallen between 89% and 94% of their 1977 level, while serum H-PCB levels had fallen between 15% and 53%.[5] It was felt that it would be preferable to sample persons based upon the serum H-PCB distribution, which had showed less of a decline from 1977 to 1984, than to sample on the L-PCB distribution. Fifteen percent of the participants in the 1977 study had H-PCB levels greater than 45 ug/l.

These participants were, exclusively, white males. An approximately equal number of demographically similar (i.e., white male) participants who had H-PCB levels less than 15 ug/l were also determined. Accordingly, any male participant in the 1977 study whose serum H-PCB level was greater than or equal to 45 ug/l, or less than or equal to 15 ug/l, was invited to participate in the follow-up study. These are referred to as the "High PCB Group" and the "Low PCB Group", respectively. Any participant in the 1977 study who had worked in the capacitor impregnation area, regardless of his serum H-PCB level, was also invited to participate. Sixty-six persons were identified for solicitation to participate in the study. Of these 66 persons, five refused to participate, and one was on vacation at the time of the study.

A. Questionnaire: Each participant responded to an interviewer-administered questionnaire, which was based upon the questionnaire used in the 1977 study (see Appendix I). The questionnaire elicited demographic data; job history since 1977; history of medical problems, symptoms, and reproductive outcomes; and alcohol, tobacco, and medication use. In addition, Westinghouse provided job history cards, from which department and job assignments could be determined, for each participant.

B. Blood Pressure Determinations: Three blood pressure determinations were made using a Hawksley Random Zero Sphygmomanometer. This instrument has a zero point that is varied randomly between 0 and 20 mm Hg. The true zero cannot be determined until after the blood pressure is taken, at which time it is subtracted from the measurement.

The deflated blood pressure cuff was applied snugly with the lower edge one inch above the antecubital space and the center of the blood pressure cuff bladder resting over the brachial artery. The observer recorded the onset of sound (Korotkoff-1), the muffling of sound (Korotkoff-4), and the disappearance of sound (Korotkoff-5). The zero point was subtracted from each measurement, and the true measurements were recorded.

C. Blood Tests: Fasting blood specimens were drawn for determination of:

polychlorinated biphenyl, quantitated as Aroclor 1242, Aroclor 1254, and Aroclor 1260;[6]
total and conjugated bilirubin;[7]
alkaline phosphatase;[7]
total cholesterol;[7]
HDL-cholesterol; [7]
triglycerides;[7]
creatinine;[7]
beta-glucuronidase;[8]
5'-nucleotidase;[9]
gamma glutamyl transferase;[10]
sorbitol dehydrogenase;[11,12]
alanine aminopeptidase;[13]
aspartate aminotransferase;[17]
alanine aminotransferase;[14]
bile acids;[15]
apolipoprotein A-1;[16]
apolipoprotein B;[16]
phospholipid fatty acids;[17,18,19] and
cholesterol ester fatty acids.[17,18,19]

Blood specimens were allowed to clot for 30-45 minutes, centrifuged, and the serum drawn-off and aliquoted into bottles. Specimens were frozen on dry ice, and shipped express mail to the Center for Environmental Health and Injury Control (CEHIC) laboratories in Atlanta, Georgia, for analysis.

D. Urine Tests: A first morning voided urine was obtained at home, the container put on ice in a Zip-lock storage bag, and transported to the study site. The urine was aliquoted into plastic containers with the appropriate preservatives, frozen on dry ice, and shipped express mail to the CEHIC laboratories for analysis of:

creatinine;[7]
gamma glutamyl transferase;[20]
alanine aminopeptidase;[13]
N-Acetyl glucosaminadase;[21] and
D-glucaric acid.[22]

V. RESULTS

A. Serum PCB Levels: Sixty of 66 white male subjects from the 1977 study solicited for the follow-up study, and participated in the examinations. There were 28 in the Low PCB group, and 32 in the High PCB group. Based on circumstantial evidence, it was determined that the serum PCB level measured in 1977 for one individual in the Low PCB group was in error (see Appendix II). His data were therefore eliminated from further consideration in the analyses.

Based upon the results of the 1977 study, the laboratory attempted to quantitate those PCB residues found by gas chromatography (GC) to elute prior to p,p'-DDE as Aroclor 1242, and those found by GC to elute after p,p'-DDE as Aroclor 1254. A review of the analytical runs indicated that these two Aroclors did not account for all of the responses observed in the GC tracings. It was therefore decided that, in addition, Aroclor 1260 would be used to account for those peaks observed with GC retention times beyond Aroclor 1254. In no instance was an apparent Aroclor response quantitated more than once.[26]

Summary statistics for age, L-PCB, and H-PCB, are listed in Table 1. The L-PCB and H-PCB levels include those serum PCB residues eluting from the gas chromatograph before and after p,p'-DDE, respectively. The H-PCB level drawn in 1985, as listed in Table 1, is the sum of the PCB residues quantitated as Aroclor 1254 and Aroclor 1260.

From Table 1, we note that the High PCB group participants were on average 5.6 years older than the Low PCB group. The mean age for the Low PCB group was 46.4 years with a range from 35.8 to 64.1 years. The mean age for the High PCB group was 52.0 years with a range from 32.2 to 67.9 years.

By 1985, the serum L-PCB concentrations in the Low PCB group had decreased by an average of 85% of the 1977 value. Serum L-PCB concentrations in the High PCB group decreased by an average of 90%. These decreases are shown graphically in histograms in Figures 1 and 2. There was only one individual in whom the serum L-PCB

concentration increased between 1977 and 1985. This was from 2 ug/l in 1977 to 2.38 ug/l in 1985, which accounts for the entry of +19% in the column headed "Maximum" for the "Low PCB Group" in Table 1. Except for this trivial apparent increase in serum L-PCB level, which is statistically indistinguishable from zero, all serum L-PCB levels decreased between 1977 and 1985.

By 1985, the serum H-PCB level in the Low PCB group had decreased by an average of 39% of the 1977 value. These decreases are shown in histograms in Figures 3 and 4. The serum H-PCB level in the High PCB group decreased by an average of 58%. There were three individuals in the Low PCB group and three individuals in the High PCB group in whom serum PCB levels increased between 1977 and 1985. In the Low PCB group, increases were from 14 to 15, 11 to 14, and 13 to 20 ug/l (107.2%, 127.3%, and 153.8% of the 1977 values, respectively). In the High PCB group, increases were from 65 to 68, 49 to 72, and 74 to 129 ug/l (104.6%, 146.9%, and 174.3% of the 1977 values, respectively).

B. Occurrence Of New Symptoms And Changes In Symptom Reporting Since 1977: During the 1977 study of occupational exposures, data regarding symptoms of disease were evaluated using two different methods. The first was a comparison of the prevalence of a symptom by exposure group (based on the individual's serum PCB level), and the second method utilized the outcome variable as a dependent variable in a regression model. In comparing the prevalence of the symptoms by the exposure group, only headaches, Odds Ratio (OR) 10.29, 95% confidence Interval (2.069 - 51.22) was significant in 1977 (see Table 2). Similar analyses of the 1985 data revealed no significant differences between the Low PCB group and the High PCB group based on the prevalence of the symptoms studied.

During the 1977 study, when the symptom was used as a dependent variable in a multiple linear regression model, the following symptoms were found to be significantly associated with serum PCB concentration: coughing on the job, or soon after work; irritated or burning eyes; unexplained loss of appetite; unexplained tingling in the hands; and rash or dermatitis. Regression models used in analyzing the 1985 data, controlling for known or possible confounders such as age, alcohol intake, smoking, etc., revealed no significant associations between either serum L-PCB or the serum H-PCB and the symptom modelled as the dependent variable.

C. Biochemical Parameters: If PCB exposure has a long-term, adverse effect on health, then we would expect to observe in 1985 the distributions of one or more of a battery of clinical tests to be different in the High PCB group, compared to the Low PCB group. For any particular test, one of the following would be expected:

(1) The test result for all persons within the High PCB group might be affected more or less equally. A shift upward in the test's mean and median would be observed for the High PCB group, compared to the Low PCB group. The dispersion of the distribution within either group would, however, be approximately equal. A side-by-side comparison of the distribution in either group would reveal that they appear visually to be similar, but the distribution in the High PCB group would be shifted upward. Mathematically, the variances of either group would be approximately equal, while the mean and median of the High PCB group would be greater than the mean and median of the Low PCB group. If enough observations were available, a statistical test comparing the group means (such as a t-test) or medians (such as Wilcoxon's test) would show a "statistically significant" difference.

(2) Only a few persons within the High PCB group might be affected. A shift upward in the test's values would be observed for one or more individuals within the High PCB group, compared to the Low PCB group, thus resulting in a few outlying observations within the distribution of the High PCB group. A side-by-side comparison of the distribution in either group would demonstrate that the High PCB group appears visually to have a greater dispersion than the Low PCB group. The medians of the two groups would be the same. The mean of the High PCB group would be somewhat greater than the mean of the Low PCB group. Mathematically, the variance of the distribution in the High PCB group would be greater than the variance in the Low PCB group. The medians would be approximately equal, while the mean of the High PCB group would be greater than the mean of the Low PCB group, although the difference would not necessarily be "statistically significant".

(3) A combination of (1) and (2) above. Here, there would be some shift upward in all test values, plus one or more outlying observations, in the High PCB group. Mathematically, the variance of the High PCB group would be greater than the variance of the Low PCB group, and the means and medians both would be greater in the High than the Low group.

Alternatively:

(4) If PCB has no chronic adverse effect, then the distributions in either group would appear to be the same. Mathematically, the variances, means, and medians of both exposure groups would be approximately equal. Sampling variation would be expected to result in some of the summary statistics being different between groups.

1. Assessment Of Liver Function: Simple correlations between serum log L-PCB and serum log H-PCB concentrations and the results of a battery of tests measuring liver function were performed. Analyses for some variables were performed on the log-transformed variate (to render the distribution more closely to normal in appearance). Statistically significant correlations are presented in Table 3. To control for possible confounding by age, alcohol intake, and consumption of drugs that might be expected to affect the liver, multiple linear regression equations were computed for each correlation with serum log PCB concentration, age, alcohol and drug usage as independent variables. Because of the collinearity of serum log L-PCB and serum log H-PCB concentrations, separate regressions with serum log L-PCB and the confounder(s) as predictors, and serum log H-PCB and the confounder(s) as the predictors were performed.

   As a result of the regression analyses, beta-glucuronidase, 5-prime nucleotidase, total bilirubin, and conjugated bilirubin, remained significantly associated with log L-PCB, controlling for confounding factors, while beta-glucuronidase was also significantly associated with log H-PCB. In the case of log SGOT, although significantly correlated with L-PCB, this association was found to be confounded by alcohol consumption.

   Figures 5 through 16 display side-by-side box plots of the distributions of each liver function test for both PCB exposure groups. Clinically significant medical history and alcohol consumption are given for outlying data values (indicated by asterisks). Table 4 summarizes each test's median and variance within either group, and provides a summary of statistical calculations performed to determine if the variances and medians are different. For those tests in which either the variance or the median were significantly different, multiple linear regressions were computed for either the variate or the log-transformed variate, regressed on exposure group (High vs. Low) and possible confounding

variables (alcohol consumption, and the consumption of therapeutic drugs that might be expected to affect the liver). In selected instances (as described below), regression diagnostics were examined to attempt to identify individual influential observations.

In accordance with the possibilities described above:

(1) Liver function tests in which the variances are equal between groups, but the medians are significantly different (case 1 above): Sorbitol dehydrogenase (SDH) appears to have homogeneous variance between groups, with an increased median in the High PCB group. However, regression analysis failed to retain any of the possible confounders, and the effect of PCB group (Low vs. High) to explain SDH variation was nonsignificant. It is not possible to give a meaningful interpretation to this contradictory pattern.

(2) Liver function tests in which the variance was significantly different between groups, but medians were not significantly different (case 2 above): Serum alanine aminopeptidase (SAAP), aspartate aminotransferase (SGOT), alanine aminotransferase (SGPT), and bile acids (BILA) appear visually to be different. Statistical comparisons demonstrate that the differences are due to increased variance in the High PCB group compared to the Low PCB group, without meaningful change in the median. Multiple linear regressions demonstrate that alcohol consumption (for SAAP, SGOT, and BILA) or consumption of drugs that are reported to have some cholestatic effect on the liver (for SGPT) are statistically significant explanatory variables for the observed distributions. PCB group (High vs. Low) contributes insignificantly to explaining the variation of the distributions. Regression diagnostics confirm the impression derived from visual inspection of the box plots: that one individual, who reported consuming the equivalent of 8 beers per day, was the most extreme outlier for three of these tests. The difference in these tests between groups appears to be due to confounding by alcohol or (therapeutic) drug consumption, and is not attributable to classification by PCB group.

(3) Liver function tests in which the variances and medians are significantly different between groups (case 3 above): conjugated bilirubin (CBIL), total bilirubin (TBIL), beta-glucuronidase (BGLU), 5'-nucleotidase (5NUC), and gamma

glutamyl transpeptidase (GGTP) appear visually to be different. Statistical comparisons demonstrate that the differences are due to increased variances and medians in the High PCB group compared to the Low PCB group.

Multiple linear regressions demonstrated that no potential confounding variable contributed significantly to explaining the variation of the distributions of CBIL, TBIL, BGLU, or 5NUC. Alcohol contributed significantly to explaining the variation of GGTP, but classification by PCB group also remained significant. Regression diagnostics revealed the same individual whose data influenced the distributions for SAAP, SGOT, and BILA, also was most influential in the regression of GGTP.

(4) Liver function tests with no difference between groups in variance or median (case 4 above): The distributions of alkaline phosphatase and urinary d-glucaric acid excretion appeared not to be dissimilar between High and Low PCB groups, based upon a visual examination of the box-plots and statistical comparisons of the distribution's variances and medians.

2. Assessment Of Serum Cholesterol, Triglycerides, and Apolipoproteins: Simple correlations between serum log L-PCB and log H-PCB concentrations and the results of serum cholesterol, triglycerides, and apolipoproteins were performed. Statistically significant correlations are presented in Table 5. To control for possible confounding by age and alcohol intake, multiple linear regression equations were computed for each correlation with serum log PCB concentration, age, and alcohol as independent variables. Because of the collinearity of serum log L-PCB and serum log H-PCB concentrations, separate regressions with serum log L-PCB and the confounder(s) as predictors, and serum log H-PCB and the confounder(s) as predictors were performed.

Significant associations with log serum L-PCB, controlling for possible confounding factors, were noted for cholesterol, serum apolipoprotein B, triglycerides, and log serum apolipoprotein B. In addition, serum log H-PCB was significantly associated with triglycerides controlling for possible confounding factors.

Neither serum log L-PCB nor log H-PCB were predictors of HDL, log HDL, serum apolipoprotein A1, or serum creatinine. Previous studies[1,4] have shown an inverse effect of serum PCB concentrations on log HDL-cholesterol. Although the correlation coefficients were negative, they were not statistically significant.

Figures 17 through 22 display side-by-side box plots of the distributions of each serum lipid and apolipoprotein test for both PCB groups. Clinically significant medical history and alcohol consumption are given for outlying data values (indicated by asterisks). Table 6 summarizes each test's median and variance within each group to determine if the variances and medians are different. For those tests in which either the variance or the median are significantly different, multiple linear regressions were computed, regressed on exposure group, log H-PCB, log L-PCB, and possible confounding variables (age and alcohol).

In accordance with the possibilities described previously:

(1) Lipid tests in which the variance was significantly different between groups, but the median was not significantly different (case 2 above): Triglycerides appeared to be visually different. Statistical comparisons demonstrate that the difference is due to increased variance in the H-PCB group compared to the L-PCB group without meaningful change in the median. Regression diagnostics confirm the impression derived from visual inspection of the box plots: that one individual who has a history of hypertension and diabetes was the most extreme outlier in this test. The difference in these tests between groups appears to be due to confounding and is not attributable to classification by PCB group.

(2) Lipid tests with no differences between exposure groups in variance or medians (case 4 above): The distributions of cholesterol, HDL-cholesterol, apolipoprotein A-1, apolipoprotein B, and serum creatinine appear not to be dissimilar between High and Low PCB groups, based on a visual examination of the box plots.

3. Assessment Of Phospholipid Fatty Acids And Cholesterol Ester Fatty Acids: Fatty acid profiles in both the phospholipid and cholesterol ester fractions of serum were determined by a combination thin-layer chromatographic and gas-liquid chromatographic procedure. The mean patterns for all 59 samples are summarized in Table 7. Although some differences between these values and those of a CDC reference group were apparent, the differences probably reflected dietary influences. For example, the lower linoleate (18:2 n-6) and relatively higher percentage oleate (18:1 n-9) in both the phospholipid and cholesterol ester fractions in the study population were consistent with a decreased intake in lower polyunsaturated

fatty acids. In general, the apparent differences between this study population and the CDC reference group were not consistent with differences predicted from the analysis of serum lipid profiles in rats dosed with high levels of polybrominated biphenyls.[19]

The results for both the phospholipids and the cholesterol esters were categorized by exposure group and analyzed further. As indicated in Table 8, both the phospholipid and cholesterol ester profiles were nearly identical in these two groups.

Histograms of all variables were plotted and examined for non-normality. Some of the fatty acid variables appeared to be normally distributed, others were not. Since the data were based on compositional biochemical information, none of the variables was transformed. Correlation matrices for phospholipid and cholesterol ester patterns, serum log PCBs, and age were then obtained. The highest correlations with serum log PCB in the phospholipid profiles were age (0.466, p-value=0.0002) and 20:3 n-6 (0.441, p-value=0.0005). Other variables were either not significant or represented very minor components in the profiles. Only one (minor) cholesterol ester was correlated with serum log PCBs at the 0.05 level of significance, 14:0.

Regressions of serum log PCBs with age and phospholipid fatty acids considered as independent variables yielded low R-squared values. The best single variable model included age (R-square=0.217), whereas the best two variable models included age and 20:3 n-6 (R-square=0.38). The R-square did not improve substantially with the addition of any more phospholipid fatty acid variables, and the R-square with all variables was only 0.52. Because the correlations for cholesterol ester fatty acids were lower as noted above, the analogous regressions yielded even lower R-square values, with a maximum of 0.41 when all variables including age were included in the model. The use of regression diagnostics did not reveal any influential points on the regression.

(1) In accordance with the possibilities described previously, (case 4 above), there was no difference between group variances or means. The distributions of phospholipids and cholesterol esters appeared not to be dissimilar between High and Low PCB groups based on a statistical comparison of the distributions of the variances and the means.

4. Assessment Of Kidney Function And Excretion Of Protein: Simple correlations between serum log L-PCB and log H-PCB concentrations and the results of a battery of tests measuring kidney function were performed. Statistically significant correlations are presented in Table 9. To control for possible confounding, multiple linear regression equations were computed for each correlation with serum log PCB concentration, age, and alcohol as independent variables. Because of the collinearity of serum log L-PCB and serum log H-PCB concentrations, separate regressions with serum log L-PCB and the confounder(s) as predictors, and serum log H-PCB and the confounder(s) as the predictors were performed.

   Only two parameters, urinary creatinine and urinary alanine aminopeptidase, were significantly correlated with either log L-PCB or log H-PCB. In the case of urinary creatinine, regression analysis failed to retain any of the possible confounders, and the effect of log H-PCB was nonsignificant. However, log L-PCB was statistically significant, controlling for possible confounders, in the regression model using urinary alanine aminopeptidase as the dependent variable.

   Figures 23 through 29 display side-by-side box plots of the distributions of each kidney function test for both PCB groups. Clinically significant medical history and alcohol consumption are given for outlying data values (indicated by asterisks). Table 10 summarizes each test's mean and variance within either group, and provides a summary of statistical calculations performed to determine if the variances and medians are different. For those tests in which either the variance or the median are significantly different, multiple linear regressions were computed for the variate, regressed on exposure group (High vs. Low) and possible confounding variables. In selected instances as described below, regression diagnostics were examined to attempt to identify individual influential observations. All of the variables were corrected for urinary creatinine excretion.

   In accordance with the possibilities previously described above:

   (1) Kidney function tests in which the variances are equal between groups, but the medians are significantly different (case 1 above): Urinary creatinine appears to have homogenous variance between groups with an increased mean in the Low PCB group. Multiple linear regressions demonstrate that exposure group is a statistically significant explanatory variable for the observed distribution. PCB exposure group is inversely statistically associated with urine creatinine.

(2) Kidney function tests in which the variance was significantly different between groups, but medians were not significantly different (Case 2 above): Urinary N-acetyl glucosaminadase appears visually to be different. Statistical comparisons demonstrate that the difference is due to increased variance in the High PCB group compared to the Low PCB group, without meaningful change in the median. Multiple linear regressions demonstrate that there are no statistically significant explanatory variables for the observed distribution. PCB group (High vs. Low) and all other confounding factors modelled do not contribute significantly to explaining the variation of the distribution. Regression diagnostics confirm the impression derived from visual inspection of the box plots: that one individual who reported consuming the equivalent of 8 beers a day was the most extreme outlier for this test. The difference in this test between groups appears to be confounded by alcohol. Although the T statistic in the regression model was not statistically significant at the 0.05 level, p-value 0.08, the coefficient was positive, whereas the coefficient for the exposure group was negative. The difference in this test is not attributable to classification by PCB group.

(3) Kidney function tests in which the variances and the medians are significantly different between groups (case 3 above): Urinary alanine aminopeptidase appears visually to be different. Statistical comparisons demonstrate that the difference is due to increases in both the median and the variance in the High PCB group compared to the Low PCB group. For this variate, multiple linear regressions were computed for the log transformed variate (to render the distribution more closely to normal in appearance).

Multiple linear regressions demonstrate that alcohol consumption, age, and L-PCBs are statistically significant explanatory variables for the observed distribution, with each having approximately equal effect. PCB group (High vs. Low), as well as H-PCB, contributes insignificantly to explaining the variation of the distribution.

(4) Kidney function tests with no difference between groups in variance or median (case 4 above): The distributions of serum creatinine and urinary gamma glutamyl transpeptidase appeared not to be dissimilar between High and Low PCB groups, based upon a visual examination of the box plots and statistical comparisons of the distribution's variances and medians.

D. Blood Pressure: Several studies[4,23] have reported possible associations between exposure to PCBs and resulting elevations in blood pressure. During the 1977 study, a significant correlation was noted between serum log H-PCB concentrations and diastolic blood pressure. Multiple linear regression, including possible confounding variables such as age and sex, showed the apparent association between diastolic blood pressure with increasing serum log H-PCB concentration to have been attributable to an association primarily with age and sex.

If PCBs are associated with increased blood pressure, we would expect to see differences in the distribution in either or both the systolic and diastolic blood pressures between the High and Low PCB groups. In an attempt to control for possible spurious elevated measurements, an average of the three systolic and three diastolic blood pressures was calculated. All analyses were based on the mean systolic and mean diastolic blood pressures. The mean systolic and diastolic blood pressures by exposure group are presented in Table 11. Neither the means nor the variances of the systolic or diastolic blood pressures by exposure group were significantly different. Increased diastolic blood pressure was defined as a diastolic blood pressure greater than or equal to 90 mm Hg. An increased systolic blood pressure was defined as a systolic blood pressure greater than or equal to 140 mm Hg.

Mantel-Haenszel odds ratios were calculated comparing the prevalence of elevated diastolic blood pressure by exposure group. The odds ratio was 1.2 with a 95 percent confidence interval of 0.1 to 26.4. The odds ratio comparing the mean systolic pressure by exposure group was 1.3 with the 95 percent confidence interval of 0.2 to 8.4. Neither were statistically significant.

In addition, both the mean systolic and mean diastolic values were treated as continuous variables and analyzed via multiple linear regression, controlling for known or possible confounding variables such as weight, family history, smoking, alcohol, and age. Multiple linear regression models controlling for the previously mentioned confounding variables demonstrated that, as found in 1977, any apparent association between either diastolic blood pressure or systolic blood pressure and increasing serum PCB concentrations was attributable to an association primarily with age. For the model using mean diastolic blood pressure as the dependent variable, the only significant variable was age ($F=0.708$, P-value=0.0014, r-squared=0.34); and for the model using mean systolic blood pressure as the dependent variable, again the only significant variable was age ($F=2.655$, p-value=0.011, r-squared=0.32).

It is possible that PCBs could be associated with blood pressure, and individuals could have been subsequently diagnosed and treated for their hypertension, resulting in no statistically significant differences between the High and Low groups based on actual blood pressure measurements. To rule out this possibility, the participant's response to a question, "Have you ever been diagnosed as having high blood pressure?", was stratified as to his exposure category. Although the Mantel-Haenszel odds ratio was elevated, 1.6, the 95 percent confidence interval (0.5 - 4.9) did contain the null value.

VI. DISCUSSION

During the 1977 study, serum log PCB correlated significantly with symptoms suggestive of mucous membrane irritation, of systemic malaise, and of altered peripheral sensation; and with serum log SGOT, log GGTP, plasma log triglyceride, and log HDL-cholesterol. Of these parameters, only triglycerides were significantly correlated with serum log PCB concentrations in 1985. Only total bilirubin and conjugated bilirubin were not significant in 1977 and were subsequently elevated in 1985. Other parameters significantly elevated in 1985 included beta glucuronidase, 5-prime nucleotidase, total bilirubin, conjugated bilirubin, serum apolipoprotein A1, serum Apolipoprotein B, and urinary alanine aminopeptidase. However, it should be noted that these tests were not performed in the 1977 study. Although log HDL-cholesterol was not statistically associated with serum log PCB, the trend of decreasing levels of HDL-cholesterol with increasing concentrations of serum log PCB was evident, though not significantly. The changes in serum lipids and liver enzymes are evidence of an effect on lipid metabolism and liver function resulting from exposure to PCBs. The biological significance of this relationship is not totally clear. Other studies have reported similar elevations in these parameters. Although they are reflective of microsomal enzyme induction, whether these specific enzymes are precursors to chronic liver dysfunction is not clear. It is important to note that formerly, serum log SGOT was significantly correlated with serum log PCB. This is no longer true. It appears that as serum log PCB concentrations, especially serum log L-PCB, continue to decline, the capacity of the PCBs to induce specific microsomal enzymes declines as well. During the 1977 study, concerns were noted due to the increased cardiovascular risks associated with exposure to PCBs. These concerns centered around the effect of serum log PCBs on triglycerides, and the inverse association with HDL-cholesterol. Although triglycerides were significantly associated with serum log PCBs, the findings in this study (no statistically significant association between HDL-cholesterol, systolic and diastolic blood pressures, and serum log PCBs) should tend to minimize those concerns regarding increased cardiovascular risk.

In this study new parameters were also evaluated. Urinary creatinine and urinary alanine aminopeptidase were found to be significantly associated with serum log PCB concentrations. These findings do suggest that PCBs have an effect on kidney function in individuals exposed. The clinical significance of these findings, as to the possibility of chronic kidney disease, remains unclear.

In general, it was noted during the 1977 study[1] that there was a lack of clinically apparent illness among the workers with the high levels of exposure and resulting high serum log PCB concentrations. This trend persisted during the 1985 study and has been documented in other studies[3,4] reported in the literature. When the prevalence of symptoms and overt clinical disease were investigated by exposure group, no differences between the groups could be ascertained except for a positive association with GGTP and a negative association with urinary creatinine. It is important to note that in the regression model using GGTP as the dependent variable, serum log PCB was not a significant explanatory variable, while in the model using exposure category as an independent variable, exposure category was a significant explanatory variable.

The formation of the exposure categories was based on the study participant's serum PCB concentration. Although other possible confounding factors such as age, alcohol and drugs, which might have had an effect on this enzyme, were controlled for, all of these variables were rejected by the regression model. In addition to elevated concentrations of serum PCB, the "High" exposure group was, on average, approximately 5 years older. It is not possible at this time to give a meaningful explanation to this contradictory pattern.

If exposure to PCBs was associated with overt clinical disease, one would expect that the greatest effects would be seen in those individuals with the greatest exposures. The concentrations of serum log PCBs documented in the workers participating in both the 1977 and 1985 studies, have been some of the highest levels recorded in this country.[27] The absence of either symptoms or overt clinical disease in these individuals would tend to refute concerns of adverse health outcomes associated with exposure to this chemical. None of the published occupational or epidemiological morbidity studies[1,3,4] have shown that occupational exposure to PCBs is associated with any adverse health outcome, with the exception of chloracne.[28] However, other occupational mortality studies[24,25,29] have shown excess mortality from cancer of the rectum, and a significant excess in mortality from leukemia, cancer of the liver, gallbladder, and biliary tract. This

study's apparent inability to significantly show a human adverse health effect resulting from exposure to PCBs, may be partially attributable to several factors. One is the confusion generated from exposure to multiple chemicals encountered in the workplace and in the general environment. These additional chemical(s), by themselves or in combination with other substances, may affect the health of those exposed, masking any effect attributable to exposure to PCBs. Secondly, the power of this study (the ability of a study to detect a difference between the exposure groups given a difference exists) may not have been large enough to detect a difference in symptoms and overt clinical disease between the two exposure groups. We have shown that for even those individuals in the Low exposure group, their serum PCB levels were significantly greater than those seen in individuals in the general community reporting no known exposure to PCBs. It is possible then, that the reason no differences were noted was the two exposure groups were too similar in makeup; therefore, not allowing us to detect any subtle differences. It is also possible, and is important to recognize, that the tests and methods utilized in this study are not sufficiently specific or sensitive to provide complete assurance that no adverse health outcome was overlooked. Finally, although the use of PCBs in this plant ceased in 1977, the latency period (the time of onset of exposure to the time when signs or symptoms of disease are first noted) may be greater than expected, resulting in our largely negative findings. It is possible that not enough time has passed for the adverse outcome to develop, thus precluding detection or distinguishing any differences between the two exposure groups.

Nevertheless, it is important to emphasize the following observations:

(1) Exposure to PCBs can result in elevations in certain serum lipids which can remain significant for years after exposures have ceased. The clinical significance of these continued elevations remains unclear.

(2) The distributions of a majority of the microsomal liver enzymes, formerly positively statistically associated with serum log L-PCB and exposure group, are becoming nonsignificant as serum log L-PCB levels continue to decline.

(3) Exposure to PCBs can result in elevations shown in tests of kidney function years after exposures have ceased. The clinical significance of these elevations remains unclear.

(4) Even though no symptoms or overt clinical disease could be correlated with serum log PCB concentrations and occupational exposure, the need to minimize exposure to PCBs still remains due to the documented effects of this chemical on serum lipids, kidney function, and liver function assays, and to their potential carcinogenicity.

VII. RECOMMENDATIONS

With the continued reduction in both serum log L-PCB and log H-PCB body burdens over time, it becomes more difficult to study possible adverse effects on either the signs or symptoms of diseases potentially associated with exposure to PCBs. If PCBs do have an effect, the strength of this association is small, or we would have been able to document the outcome in this or previous work. With serum log PCB levels approaching equal levels in all workers, categorical differences and possibly correlational differences between the serum log PCB congeners and the outcome under study will increasingly become nonsignificant, making inferences regarding exposure and adverse health outcomes more tenuous.

Of concern is the fact that specific indicators of lipid metabolism, liver function, and kidney function remain statistically significant, years after occupational exposures have ceased. Future investigations should address the possible development of chronic liver and/or kidney disease in occupationally exposed populations. If additional studies are performed, they should compare the morbidity/mortality experience of the exposed workers to non-exposed workers, categorized by historical serum PCB concentrations, or other pertinent historical environmental data.

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3) Association of Bloomington Westinghouse Salaried Employees
4) International Brotherhood of Electrical Workers, Local 2031
5) Indiana Department of Labor
6) Indiana State Board of Health
7) Occupational Safety and Health Administration, Region V

TABLE 1

AGE, SERUM PCB LEVEL, AND PERCENT CHANGE IN PCB CONCENTRATION BY EXPOSURE GROUP

	Year Measured	Low PCB Group* (Bottom 15 Percentile) N***	Mean	Minimum	Maximum	High PCB Group** (Top 15 Percentile) N	Mean	Minimum	Maximum
Age (yrs.)	1985	28	46.4	35.8	64.1	32	52.0	32.2	67.9
L-PCB ug/l	1977	27	58.3	2	130	32	583.4	85	3300
H-PCB ug/l	1977	27	12.4	5	16	32	76.5	30	250
L-PCB ug/l	1985	27	7.1	1.6	12.3	32	33.9	3.5	124.5
H-PCB ug/l	1985	27	7.5	2.5	19.7	32	27.7	5.2	129.1
Percent change in L-PCB (1977 to 1985)		27	-85%	-97%	+19%	32	-90%	-99%	-9%
Percent change in H-PCB (1977 to 1985)		27	-39%	-70%	+52%	32	-58%	-96%	+76%

* "Low PCB Group" = those participants in the 1977 study, whose serum H-PCB level was 15 ug/l or less. These individuals constitute the bottom 15th percentile of the serum PCB distribution in the 1977 study.

** "High PCB Group" = those participants in the 1977 study, whose serum H-PCB level was 45 ug/l or greater. These individuals constitute the top 15th percentile of the serum PCB distribution in the 1977 study.

*** N = number of participants in the analysis

TABLE 2

ODDS RATIOS AND 95 PERCENT CONFIDENCE INTERVALS FOR
SIGNS AND SYMPTOMS OF DISEASE BY EXPOSURE GROUP AND STUDY YEAR

Signs/Symptoms	OR 1977*	95% CI**	OR 1985***	95% CI**
Coughing	1.0	0.3 - 3.5	1.3	0.4 - 4.1
Wheezing	0.9	0.1 - 6.6	0.8	0.1 - 6.3
Tightness in Chest	1.9	0.3 - 11.0	0.6	0.1 - 2.6
Shortness of Breath	1.8	0.2 - 20.9	0.8	0.2 - 4.5
Nausea	5.0	0.5 - 45.8	0.8	0.2 - 3.2
Vomiting	2.7	0.1 - 69.1	1.7	0.1 - 20.2
Light Headed	3.0	0.6 - 16.3	2.1	0.7 - 6.7
Headaches	10.3	2.1 - 51.2	0.8	0.3 - 2.3
Eye Irritation	2.1	0.7 - 6.2	0.6	0.2 - 1.8
Poor Apetite	0.3	0.0 - 2.7	0.3	0.0 - 7.0
Diarrhea	0.0	0.0	0.8	0.2 - 3.7
Constipation	6.8	0.3 -136.9	0.8	0.1 - 14.1
Tired	1.4	0.4 - 4.7	0.0	0.0
Trouble Concentrating	1.9	0.3 - 11.0	1.8	0.6 - 5.4
Tense	1.4	0.4 - 4.7	0.7	0.2 - 2.2
Swollen Eyelids	6.8	0.3 -136.9	1.5	0.3 - 6.9
Swollen Feet	0.3	0.0 - 2.7	1.2	0.3 - 4.4
Dark Fingernails	2.7	0.1 - 69.2	0.0	0.0
No Sense of Smell	1.8	0.2 - 20.9	0.8	0.2 - 4.5
No Sex Drive	4.7	0.2 -101.5	2.7	0.6 - 11.3
Chest Pain	0.0	0.0	2.2	0.5 - 9.7
Arthritis	1.0	0.3 - 3.0	2.6	0.9 - 7.4
Muscle Weakness	0.3	0.0 - 7.2	2.7	0.3 - 27.5

(continued)

(Table 2 Continued)

Signs/Symptoms	OR 1977	95% CI	OR 1985	95% CI
Dark Skin	9.0	0.5 -175.3	1.8	0.3 - 10.6
Tingling in Hands	2.4	0.4 - 13.6	3.5	1.0 - 12.4
Pain Shooting Down Leg	2.8	0.3 - 28.5	2.2	0.5 - 9.7
Sores	2.5	0.4 - 14.1	1.6	0.4 - 6.2
Acne	0.9	0.1 - 6.6	0.1	0.0 - 1.0
Rash	2.3	0.7 - 7.8	1.0	0.3 - 3.8
Itching	1.5	0.3 - 7.1	0.0	0.0
Abdominal Pain	2.0	0.1 - 78.3	0.5	0.1 - 3.5
Significant Weight Loss	2.6	0.1 - 67.0	0.2	0.0 - 3.4
Ulcers	0.8	0.1 - 5.0	0.2	0.0 - 1.3
Diabetes	7.3	0.8 - 63.5	6.0	0.7 - 53.4
Anemia	0.0	0.0	0.3	0.0 - 7.0
Hypertension	1.5	0.4 - 5.2	2.6	0.7 - 9.6
Bronchitis	0.2	0.0 - 3.4	1.8	0.3 - 10.6
Emphysema	4.5	0.2 - 98.1.	0.0	0.0
TB	0.3	0.0 - 7.0	0.0	0.0
Pneumonia	4.5	0.2 - 98.1	6.0	0.7 - 53.4
Pleurisy	0.8	0.1 - 6.3	6.5	0.3 -132.2
Other Chest	1.7	0.1 - 20.2	0.8	0.1 - 14.1
Stroke	0.0	0.0	2.6	0.1 - 67.0
Nerve Injury	0.3	0.0 - 7.0	0.2	0.0 - 1.8
Heart Attack	2.4	0.1 - 62.2	1.7	0.1 - 20.2
Angina	0.0	0.0	0.8	0.1 - 14.1
Other Heart Problems	0.0	0.0	0.2	0.0 - 3.4
Hepatitis	0.0	0.0	4.5	0.2 - 98.1

(continued)

(Table 2 Continued)

Signs/Symptoms	OR 1977	95% CI	OR 1985	95% CI
Urinary Infection	0.0	0.0	2.3	0.4 - 13.0
Kidney Stones	0.0	0.0	0.6	0.1 - 2.9
Other Kidney	0.0	0.0	0.2	0.0 - 3.4
Cancer	0.0	0.0	0.8	0.1 - 14.1
Other Medical	0.0	0.0	2.3	0.6 - 8.4
Hospitalization	0.0	0.0	1.3	0.4 - 3.7

* Odds ratio comparing the prevalence of the sign/symptom between the exposure groups in the 1977 study

** 95 percent confidence interval for the Odds Ratio

*** Odds ratio comparing the prevalence of the sign/symptom between the exposure groups in the 1985 study

TABLE 3

CORRELATION OF LIVER FUNCTION TESTS WITH
SERUM LOG (L-PCB) AND SERUM LOG (H-PCB)

	Correlation Coefficient with Serum Log (L-PCB)	Correlation Coefficient with Serum Log (H-PCB)
Conjugated Bilirubin (CBIL)	0.38243*	0.24167
Total Bilirubin (TBIL)	0.28404*	0.26612*
Beta-Glucoronidase (BGLU)	0.40065*	0.44077*
Sorbitol dehydrogenase (SDH)	0.14464	0.15632
5'-nucleotidase (5-NUC)	0.32953*	0.27106*
Alkaline phosphatase (AK)	-0.07521	-0.14084
Log gamma glutamyl transpeptidase (GGTP)	0.17850	0.18486
Alanine aminopeptidase (SAAP)	0.23731	0.14244
Aspartate aminotransferase (SGOT)	0.24474	0.20942
log SGOT	0.26231*	0.21846
Alanine aminotransferase (SGPT)	0.28150*	0.25338*
log SGPT	0.25054*	0.25148*
Bile acids (BILA)	0.13536	0.11173
Urinary d-glucaric acid (UDGL)	-0.00892	0.18810

* Statistically significant ($P < 0.05$) correlation

Table 4

ANALYSIS OF MICROSOMAL LIVER FUNCTION
BY EXPOSURE GROUP

	GROUP		TESTS OF SIGNIFICANCE THAT		
	Median	Variance	Variances Are Equal (F-test p-value)	Medians Are Equal (Wilcoxon p-value)	Group effect Is Still Significant Given Conf'drs
Conjugated bilirubin (CBIL):					
Low PCB group	0.22	0.06	<0.0001	0.022	0.024
High PCB group	0.44	0.34			
Total bilirubin (TBIL):					
Low PCB group	0.68	0.04	<0.0001	0.034	0.028
High PCB group	0.81	0.21			
Beta-glucuronidase (BGLU):					
Low PCB group	2.67	1.36	0.062	0.014	0.012
High PCB group	3.31	2.82			
Sorbitol dehydrogenase (SDH):					
Low PCB group	1.05	0.80	0.152	0.034	N.S.
High PCB group	1.55	0.47			
5'-nucleotidase (5-NUC):					
Low PCB group	7.89	2.65	0.001	0.042	0.074
High PCB group	8.46	9.89			
Alkaline phosphatase (AK):					
Low PCB group	102	1203	0.211	0.803	N.S.
High PCB group	105	749			
Log10(gamma glutamyl transpeptidase) (GGTP):					
Low PCB group	2.82	0.03	0.002	0.069	0.052
High PCB group	2.98	0.10			
Alanine aminopeptidase (SAAP):					
Low PCB group	92.2	246	<0.0001	0.883	N.S.
High PCB group	93.5	2438			
Aspartate aminotransferase (SGOT):					
Low PCB group	19.7	32.4	<0.0001	0.334	N.S.
High PCB group	21.2	231.7			
Alanine aminotransferase (SGPT):					
Low PCB group	15.7	41.2	0.025	0.263	N.S.
High PCB group	17.5	99.1			

(continued)

Table 4 (continued)

	GROUP		TESTS OF SIGNIFICANCE THAT		
	Median	Variance	Variances Are Equal (F-test p-value)	Medians Are Equal (Wilcoxon p-value)	Group effect Is Still Significant Given Conf'drs
Bile acids (BILA):					
Low PCB group	1.7	0.80	<0.0001	0.791	N.S.
High PCB group	1.6	4.18			
Urinary d-glucaric acid (UDGL):					
Low PCB group	3.1	1.04	0.326	0.703	N.S.
High PCB group	3.4	1.51			

TABLE 5

CORRELATION OF SERUM LIPIDS WITH SERUM LOG (L-PCB)
AND SERUM LOG (H-PCB)

	# Observations	Correlation Coefficient with serum Log (L-PCB)	Correlation Coefficient with serum Log (H-PCB)
Cholesterol	59	0.25917*	0.24032
Triglyceride	59	0.39743*	0.24723*
HDL	59	-0.21819	-0.00704
Log (HDL)	59	-0.21675	-0.00149
Serum Apolipoprotein A1	59	-0.04326	0.05947
Log Serum Apolipoprotein A1	59	-0.06176	0.04845
Serum Apolipoprotein B	59	0.28893*	0.22111
Log Serum Apolipoprotein B	59	0.30015*	0.24204
Serum Creatinine	59	-0.20482	0.05410

* Statistically significant ($P<0.05$) correlation

Table 6

ANALYSIS OF SERUM CHOLESTEROL, TRIGLYCERIDES, BLOOD LIPIDS, AND APOLIPOPROTEINS BY EXPOSURE GROUP

	GROUP		TESTS OF SIGNIFICANCE THAT		
	Median	Variance	Variances Are Equal (F-test p-value)	Medians Are Equal (Wilcoxon p-value)	Group effect Is Still Significant Given Conf'drs
Cholesterol (CHOL):					
Low PCB group	221	1071	0.143	0.153	0.
High PCB group	235	1889			
Triglycerides (TGLX):					
Low PCB group	128	5139	<0.010	0.086	0.
High PCB group	155	14255			
HDL-cholesterol (HDLX):					
Low PCB group	40	54	0.929	0.404	0.
High PCB group	38	52			
Apolipoprotein A-1 (APA1):					
Low PCB group	139	254	0.601	0.843	
High PCB group	138	312			
Apolipoprotein B (APBX):					
Low PCB group	62.6	86	0.182	0.183	0.
High PCB group	63.2	144			
Serum creatinine (CREA):					
Low PCB group	1.0	0.030	0.727	0.720	
High PCB group	1.0	0.026			

TABLE 7

ANALYSIS OF PHOSPHOLIPID FATTY ACIDS
AND CHOLESTEROL ESTER FATTY ACIDS
Weight Percent (Mean ± SD)

FAME	PHOSPHOLIPIDS (N=59)	CHOLESTEROL ESTERS (N=59)
14:0	0.2 ± 0.09	0.5 ± 0.17
16:0	25.8 ± 1.83	10.9 ± 1.00
16:1	0.9 ± 0.42	3.3 ± 1.66
18:0	15.0 ± 1.31	1.3 ± 0.42
18:1	11.9 ± 3.66	20.0 ± 3.95
18:2	23.5 ± 3.21	54.1 ± 5.32
18:3	0.1 ± 0.06	0.4 ± 0.11
20:2	0.3 ± 0.07	-
20:3 (N-9)	0.2 ± 0.13	-
20:3 (N-6)	3.9 ± 0.76	1.0 ± 0.34
20:4	13.0 ± 2.33	8.6 ± 1.85
20:5	0.5 ± 0.17	-
22:4	0.6 ± 0.12	-
22:5 (N-6)	0.5 ± 0.14	-
22:5 (N-3)	1.0 ± 0.20	-
22:6	2.7 ± 0.86	-

TABLE 8

ANALYSIS OF PHOSPHOLIPID FATTY ACIDS
AND CHOLESTEROL ESTER FATTY ACIDS
BY EXPOSURE GROUP
Weight % (Mean ± SD)

	PHOSPHOLIPIDS		CHOLESTEROL ESTERS	
FAME	High PCB Group N=32	Low PCB Group N=27	High PCB Group N=32	Low PCB Group N=27
14:0	0.3 ± 0.10	0.2 ± 0.08	0.5 ± 0.18	0.4 ± 0.17
16:0	25.6 ± 1.78	25.9 ± 1.88	10.9 ± 0.94	10.9 ± 1.04
16:1	0.9 ± 0.43	0.8 ± 0.41	3.4 ± 1.84	3.3 ± 1.53
18:0	15.2 ± 1.29	14.9 ± 1.34	1.3 ± 0.43	1.2 ± 0.41
18:1	12.0 ± 4.52	11.8 ± 2.81	20.3 ± 4.72	19.8 ± 3.22
18:2	23.2 ± 3.06	23.7 ± 3.36	53.9 ± 5.58	54.4 ± 5.16
18:3	0.1 ± 0.06	0.1 ± 0.05	0.4 ± 0.13	0.4 ± 0.11
20:2	0.4 ± 0.07	0.3 ± 0.08	-	-
20:3 (N-9)	0.2 ± 0.14	0.2 ± 0.11	-	-
20:3 (N-6)	4.3 ± 0.77	3.7 ± 0.64	1.1 ± 0.39	0.9 ± 0.25
20:4	12.7 ± 1.77	13.3 ± 2.72	8.4 ± 1.79	8.7 ± 1.91
20:5	0.5 ± 0.18	0.5 ± 0.17	-	-
22:4	0.6 ± 0.12	0.6 ± 0.12	-	-
22:5 (N-6)	0.5 ± 0.14	0.5 ± 0.13	-	-
22:5 (N-3)	1.0 ± 0.18	0.9 ± 0.22	-	-
22:6	2.7 ± 0.85	2.7 ± 0.88	-	-

TABLE 9

CORRELATION OF URINARY ENZYMES WITH SERUM LOG (L-PCB) AND SERUM LOG (H-PCB)

	Correlation Coefficient with Serum Log (L-PCB)	Correlation Coefficient with Serum Log (H-PCB)
Urinary creatinine (UCRE)	-0.11557	-0.28536*
Urinary N-acetylglucosaminadase (NAGA)	0.14351	0.03939
Urinary gamma glutamyl transpeptidase (UGTN)	0.03343	-0.17379
Urinary alanine aminopeptidase (UAAP)	0.29657*	0.13561

* Statistically significant ($P < 0.05$) correlation

Table 10

ANALYSIS OF KIDNEY FUNCTION AND PROTEIN EXCRETION
BY EXPOSURE GROUP

	GROUP		TESTS OF SIGNIFICANCE THAT		
	Mean	Variance	Variances Are Equal (F-test p-value)	Medians Are Equal (Wilcoxon p-value)	Group effect Is Still Significant Given Conf'drs
Urinary creatinine (UCRE):					
Low PCB group	134	3048	0.584	0.025	
High PCB group	101	2482			
Urinary N-Acetyl glucosaminadase (NAGA): (uncorrected for creatinine excretion)					
Low PCB group	1.24	0.91		0.142	
High PCB group	1.30	3.54			
Urinary N-Acetyl glucosaminadase (NAGA_COR): (corrected for creatinine excretion)					
Low PCB group	0.0109	1E-04	0.02	0.988	
High PCB group	0.0128	2E-04			
Urinary gamma glutamyl transpeptidase (UGTN): (uncorrected for creatinine excretion)					
Low PCB group	26.8	188		0.041	
High PCB group	20.8	185			
Urinary gamma glutamyl transpeptidase (UGTN_COR): (corrected for creatinine excretion)					
Low PCB group	0.198	0.002	0.063	0.674	
High PCB group	0.198	0.004			
Urinary alanine aminopeptidase (UAAP): (uncorrected for creatinine excretion)					
Low PCB group	6.15	4.58		0.451	
High PCB group	7.00	34.4			
Urinary alanine aminopeptidase (UAAP_COR): (corrected for creatinine excretion)					
Low PCB group	0.050	3E-04	0.0001	0.014	
High PCB group	0.070	1.2E-03			

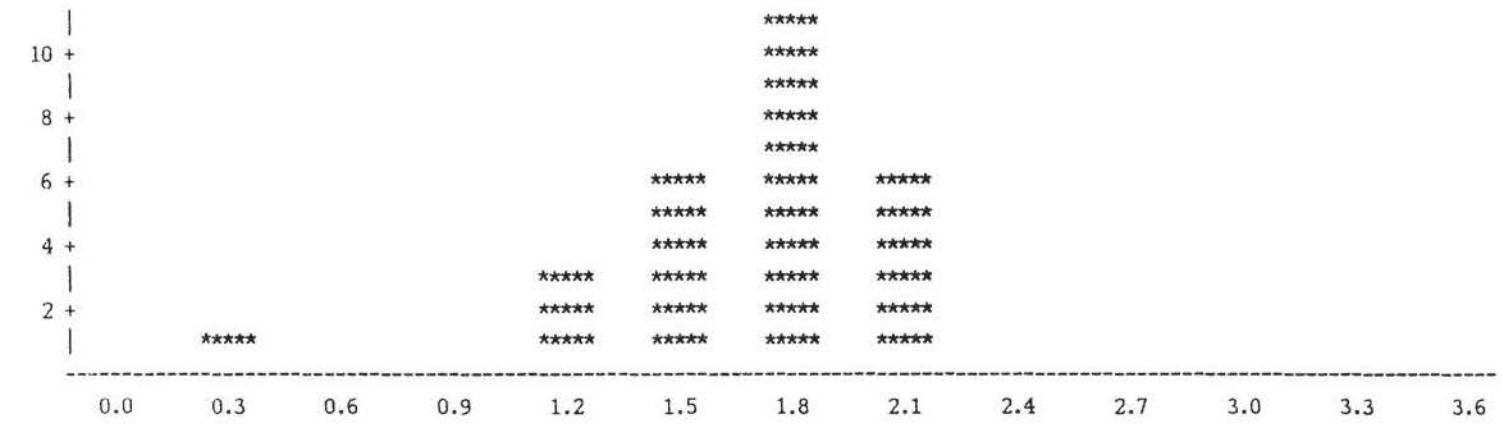
TABLE 11

COMPARISON OF MEAN DIASTOLIC AND SYSTOLIC BLOOD PRESSURE BY EXPOSURE GROUP

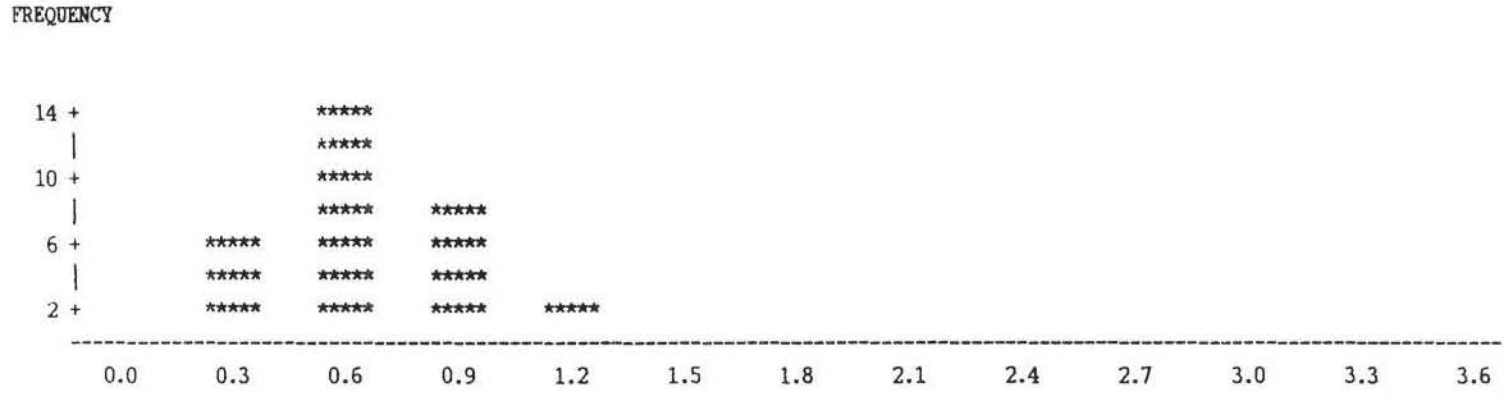
	Mean Systolic Blood Pressure	Mean Diastolic Blood Pressure
Low PCB Group	118.5 mm Hg	69.3 mm Hg
High PCB Group	123.8 mm Hg	70.9 mm Hg

Figure 1

CHANGES IN SERUM L-PCB CONCENTRATIONS
FROM 1977 TO 1985
IN THE LOW PCB GROUP



SERUM LOWER CHLORINATED BIPHENYLS, 1977: LOGARITHMIC SCALE

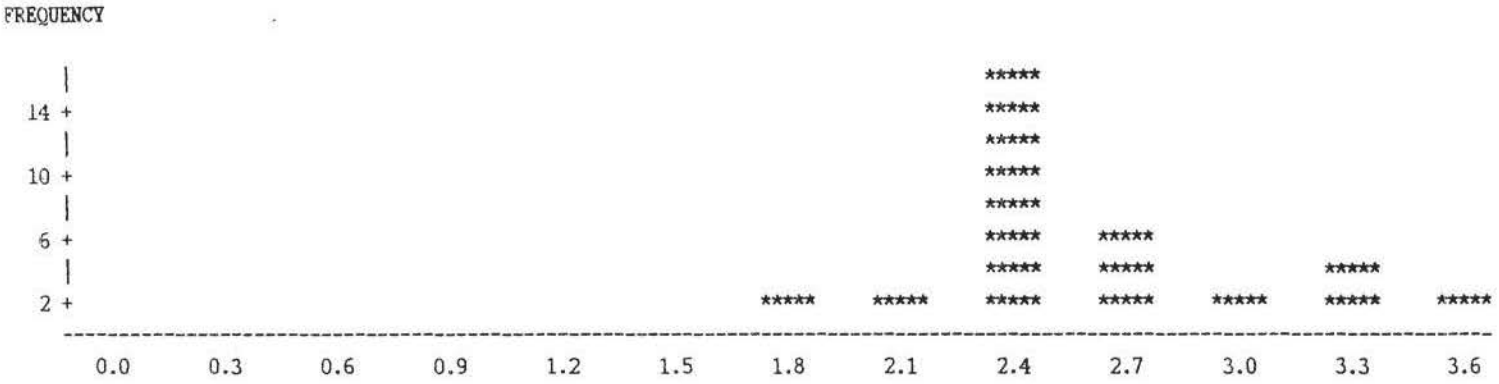


SERUM LOWER CHLORINATED BIPHENYLS, 1985: LOGARITHMIC SCALE

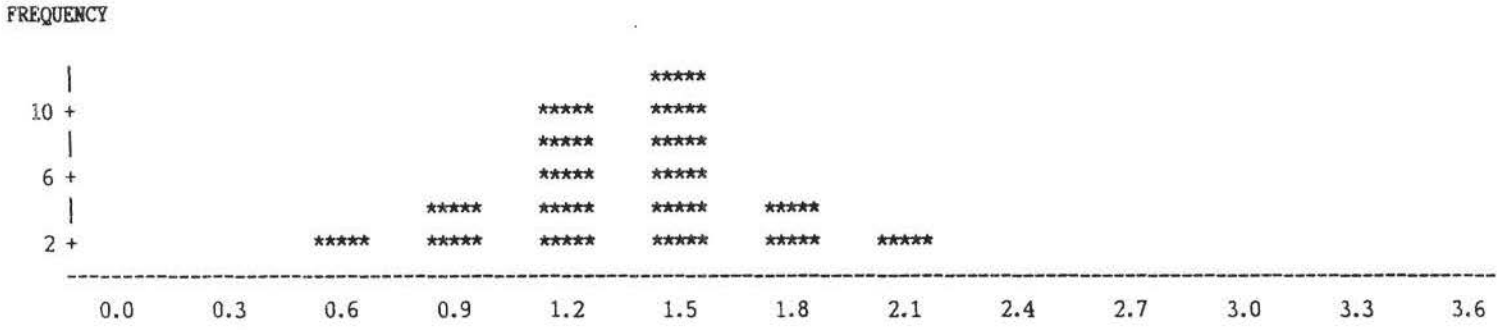
From 1977 (upper figure) to 1985 (lower figure), serum L-PCB decreased an average of 85% of the 1977 value in participants who, in 1977, had the lowest serum PCB values (i.e., the participants in the "Low PCB Group"). This is seen by a shift to the left of the serum L-PCB distribution in 1985 (lower figure), compared to the 1977 distribution (upper figure).

Figure 2

CHANGES IN SERUM L-PCB CONCENTRATIONS
FROM 1977 TO 1985
IN THE HIGH PCB GROUP



SERUM LOWER CHLORINATED BIPHENYLS, 1977: LOGARITHMIC SCALE

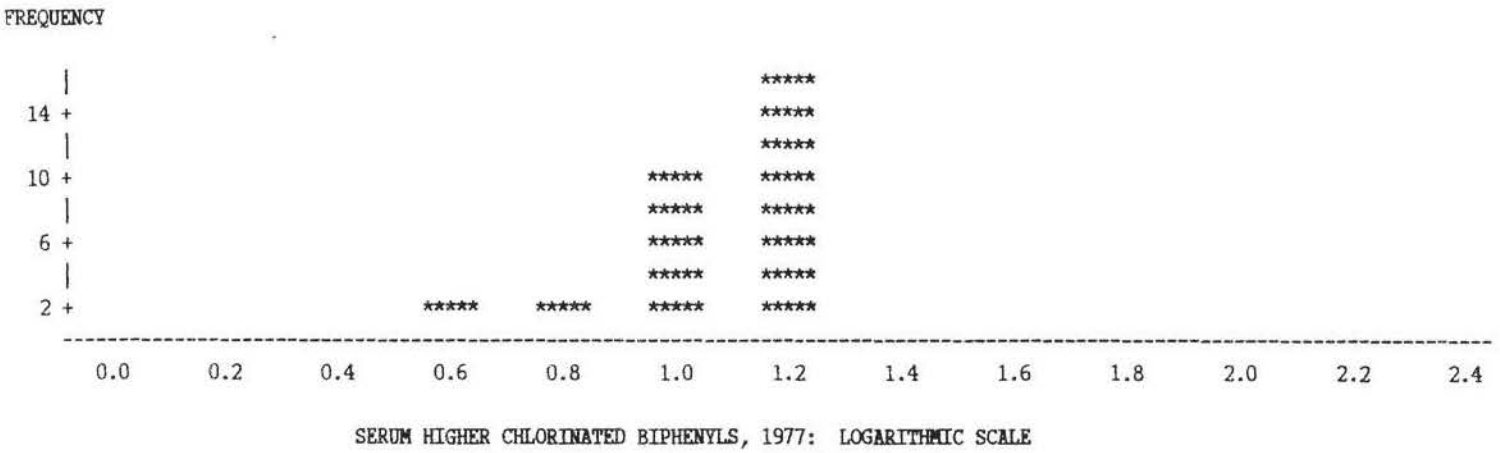


SERUM LOWER CHLORINATED BIPHENYLS, 1985: LOGARITHMIC SCALE

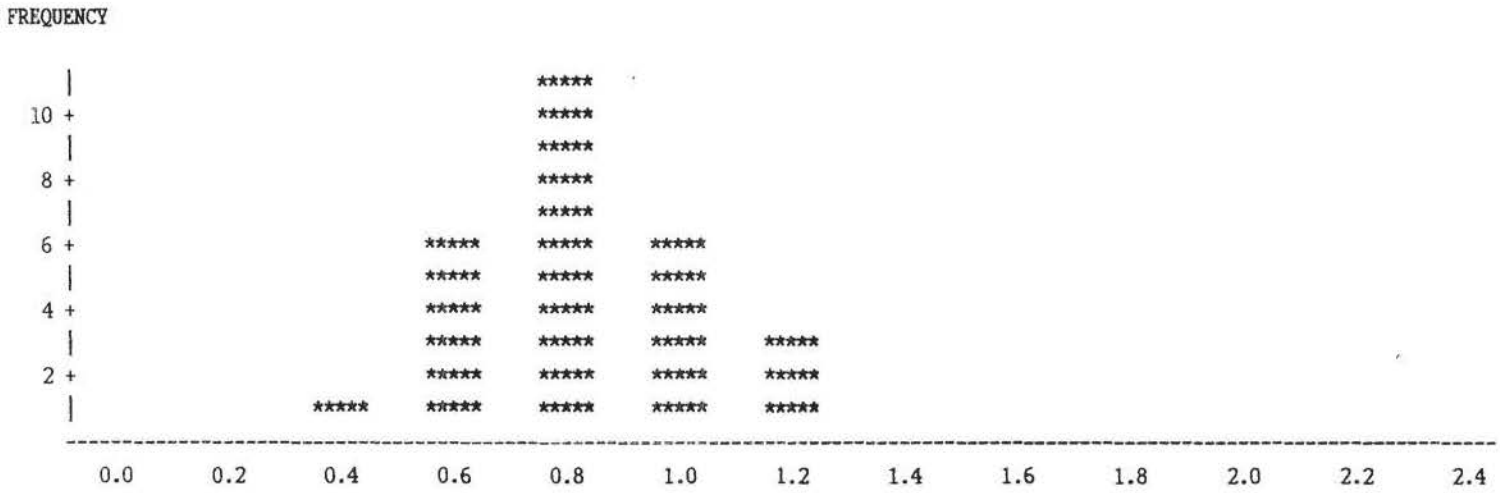
From 1977 (upper figure) to 1985 (lower figure), serum L-PCB decreased an average of 90% of the 1977 value in participants who, in 1977, had the highest serum PCB values (i.e., the participants in the "High PCB Group"). This is seen by a shift to the left of the serum L-PCB distribution in 1985 (lower figure), compared to the 1977 distribution (upper figure).

Figure 3

CHANGES IN SERUM H-PCB CONCENTRATIONS
FROM 1977 TO 1985
IN THE LOW PCB GROUP



SERUM HIGHER CHLORINATED BIPHENYLS, 1977: LOGARITHMIC SCALE

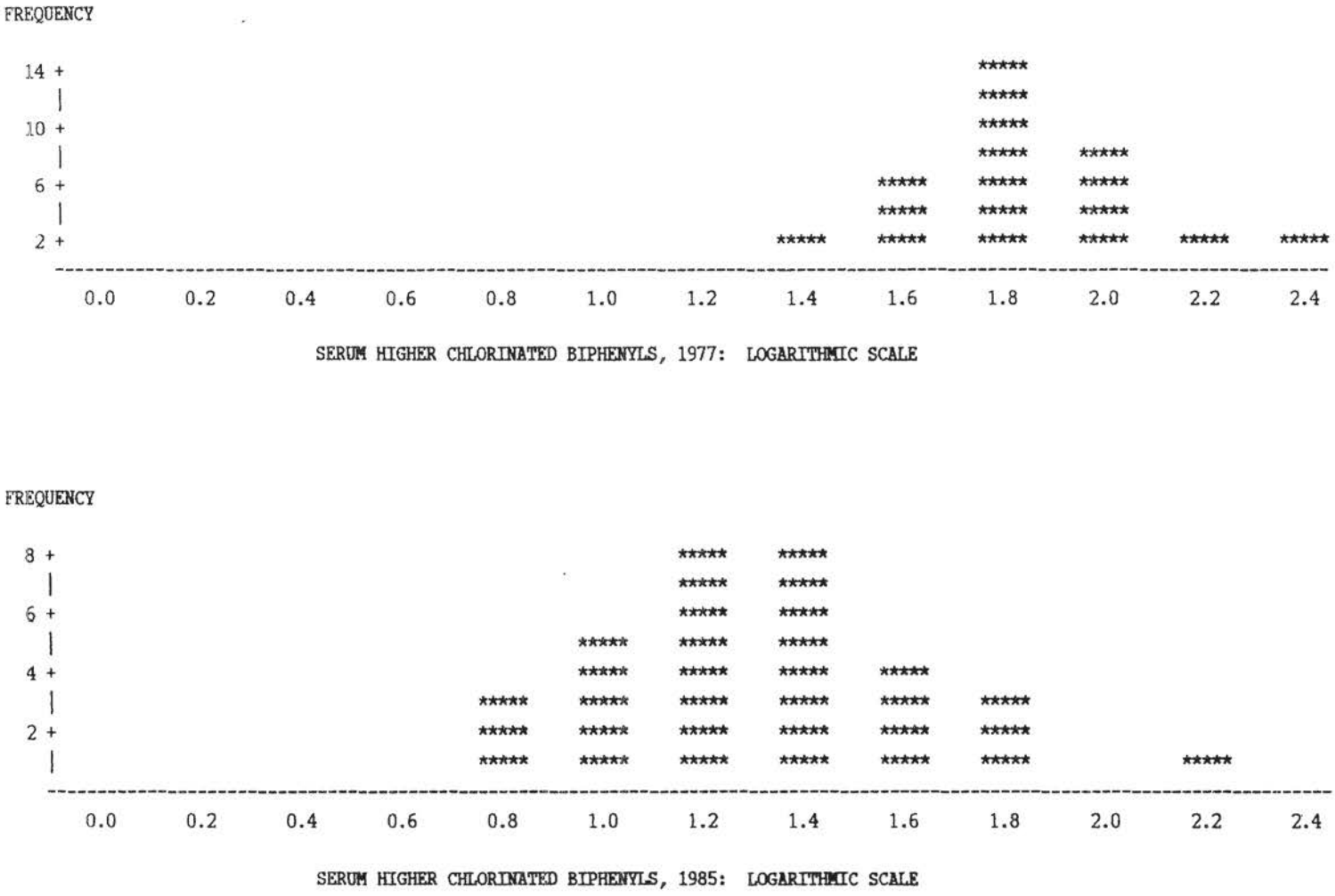


SERUM HIGHER CHLORINATED BIPHENYLS, 1985: LOGARITHMIC SCALE

From 1977 (upper figure) to 1985 (lower figure), serum H-PCB decreased an average of 39% of the 1977 value in participants who, in 1977, had the lowest serum PCB values (i.e., the participants in the "Low PCB Group"). This is seen by a shift to the left of the serum H-PCB distribution in 1985 (lower figure), compared to the 1977 distribution (upper figure).

Figure 4

CHANGES IN SERUM H-PCB CONCENTRATIONS
FROM 1977 TO 1985
IN THE HIGH PCB GROUP



From 1977 (upper figure) to 1985 (lower figure), serum H-PCB decreased an average of 58% of the 1977 value in participants who, in 1977, had the highest serum PCB values (i.e., the participants in the "High PCB Group"). This is seen by a shift to the left of the serum H-PCB distribution in 1985 (lower figure), compared to the 1977 distribution (upper figure).

Figure 5

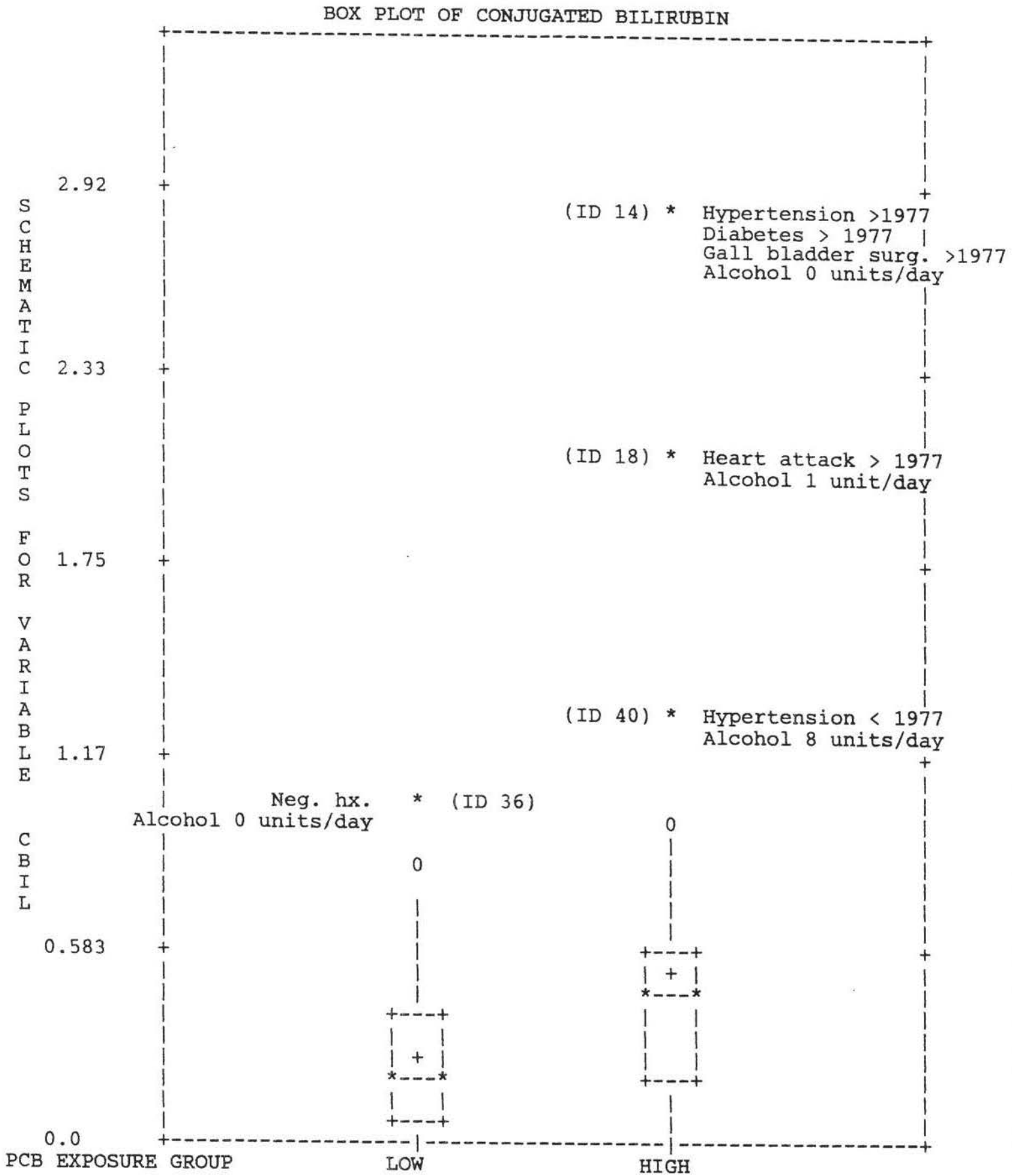

BOX PLOT OF CONJUGATED BILIRUBIN
SCHEMATIC PLOTS FOR VARIABLE CBIL
2.92
2.33
1.75
1.17
0.583
0.0
(ID 14) * Hypertension >1977
Diabetes > 1977
Gall bladder surg. >1977
Alcohol 0 units/day
(ID 18) * Heart attack > 1977
Alcohol 1 unit/day
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
Neg. hx. * (ID 36)
Alcohol 0 units/day
PCB EXPOSURE GROUP
LOW
HIGH

Figure 6

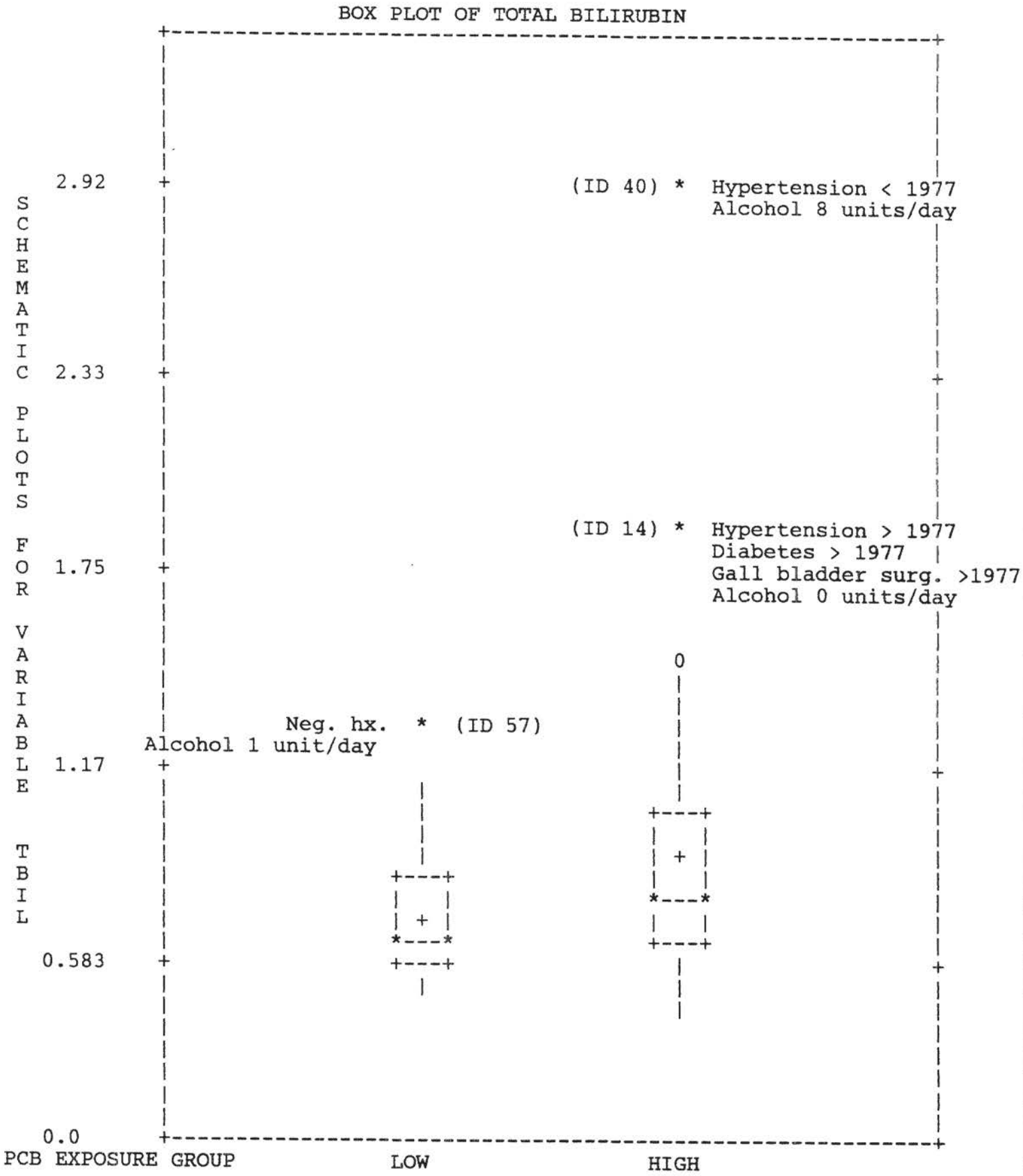

BOX PLOT OF TOTAL BILIRUBIN
SCHEMATIC PLOTS FOR VARIABLE TBIL
2.92
2.33
1.75
1.17
0.583
0.0
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
(ID 14) * Hypertension > 1977
Diabetes > 1977
Gall bladder surg. >1977
Alcohol 0 units/day
Neg. hx. * (ID 57)
Alcohol 1 unit/day
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 7

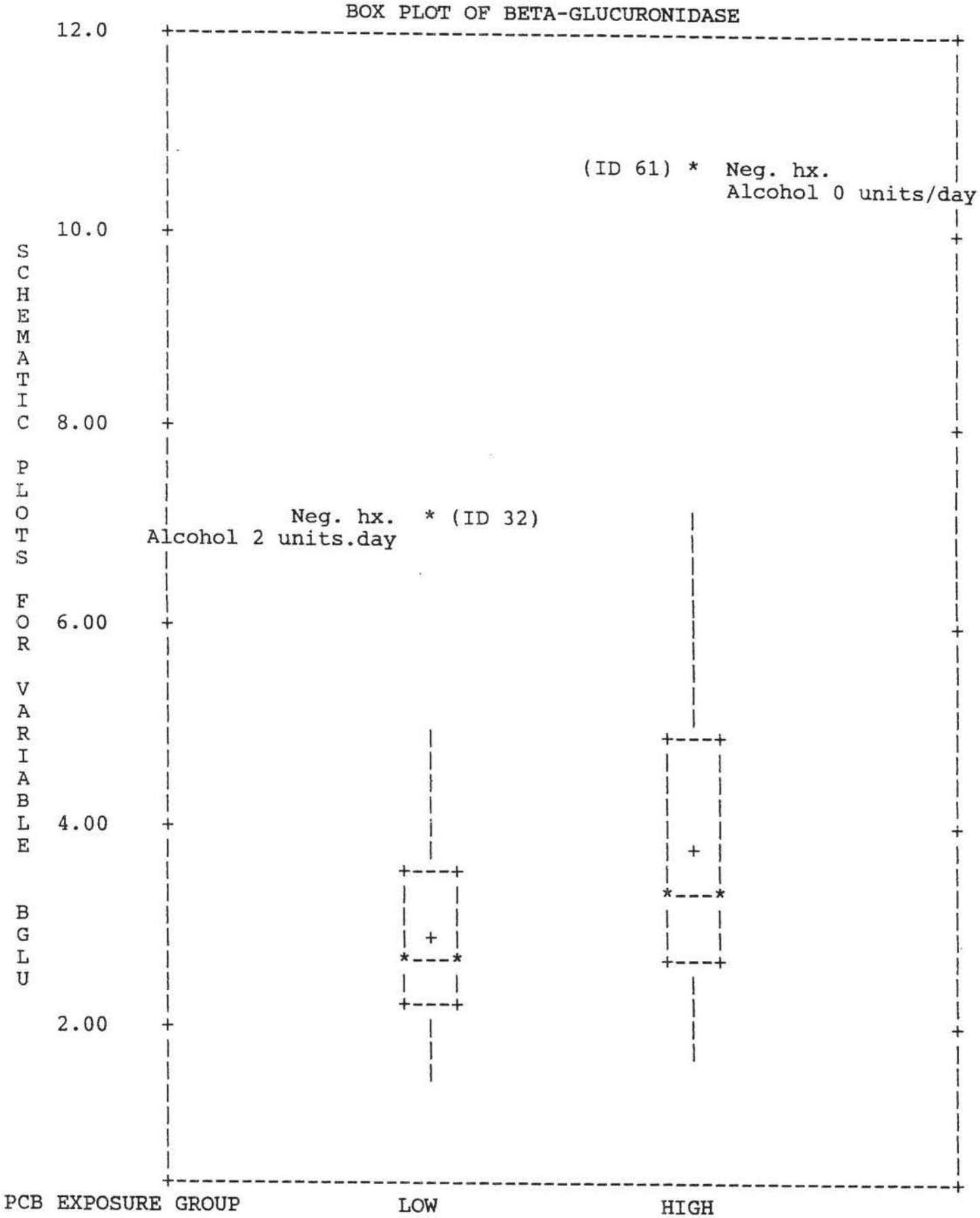

BOX PLOT OF BETA-GLUCURONIDASE
12.0
10.0
8.00
6.00
4.00
2.00
SCHEMATIC PLOTS FOR VARIABLE BGLU
(ID 61) * Neg. hx.
Alcohol 0 units/day
Neg. hx. * (ID 32)
Alcohol 2 units.day
PCB EXPOSURE GROUP
LOW
HIGH

Figure 8

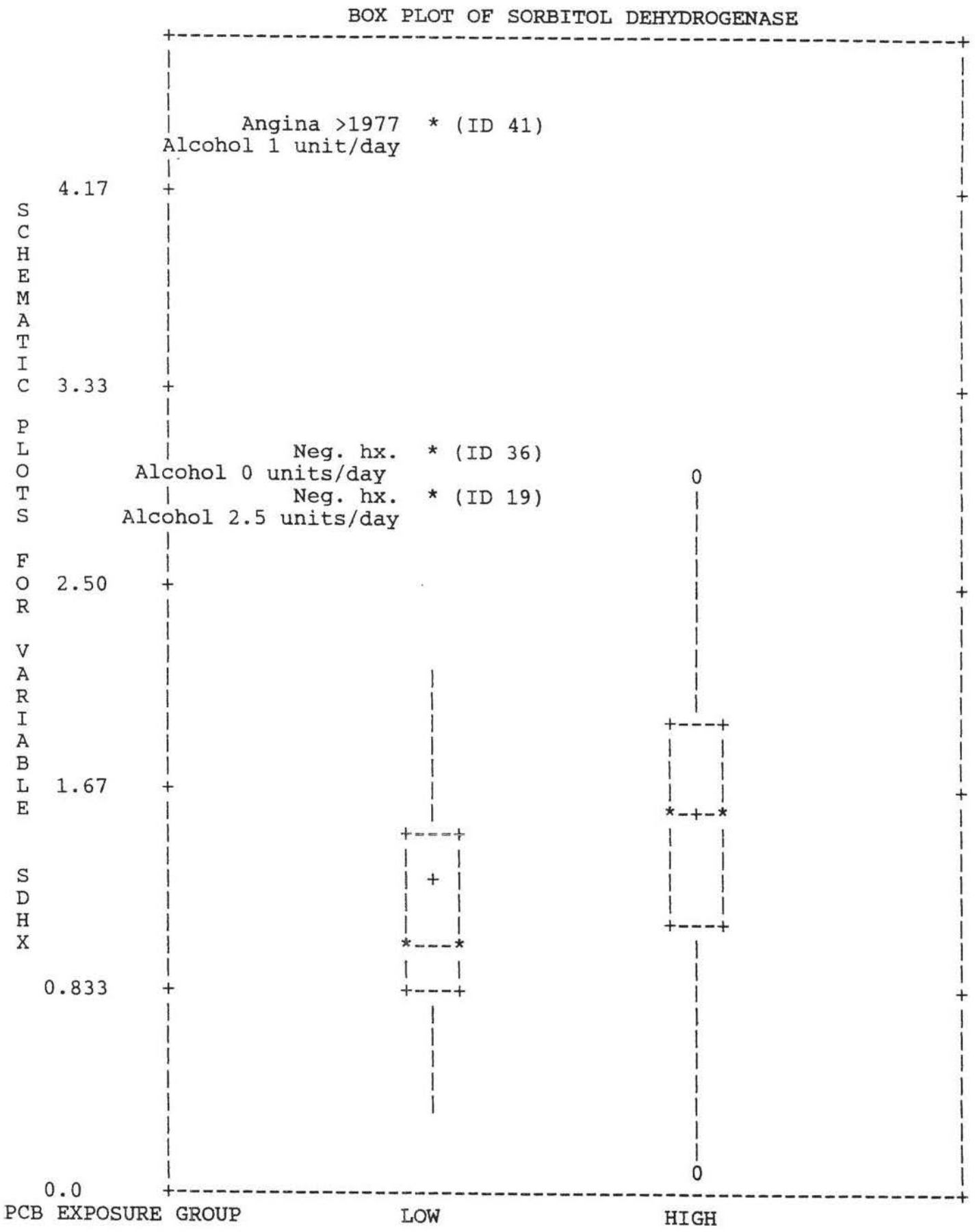

BOX PLOT OF SORBITOL DEHYDROGENASE
Angina >1977 * (ID 41)
Alcohol 1 unit/day
4.17
3.33
Neg. hx. * (ID 36)
Alcohol 0 units/day
Neg. hx. * (ID 19)
Alcohol 2.5 units/day
2.50
1.67
0.833
0.0
0
0
SCHEMATIC PLOTS FOR VARIABLE SDHX
PCB EXPOSURE GROUP
LOW
HIGH

Figure 9

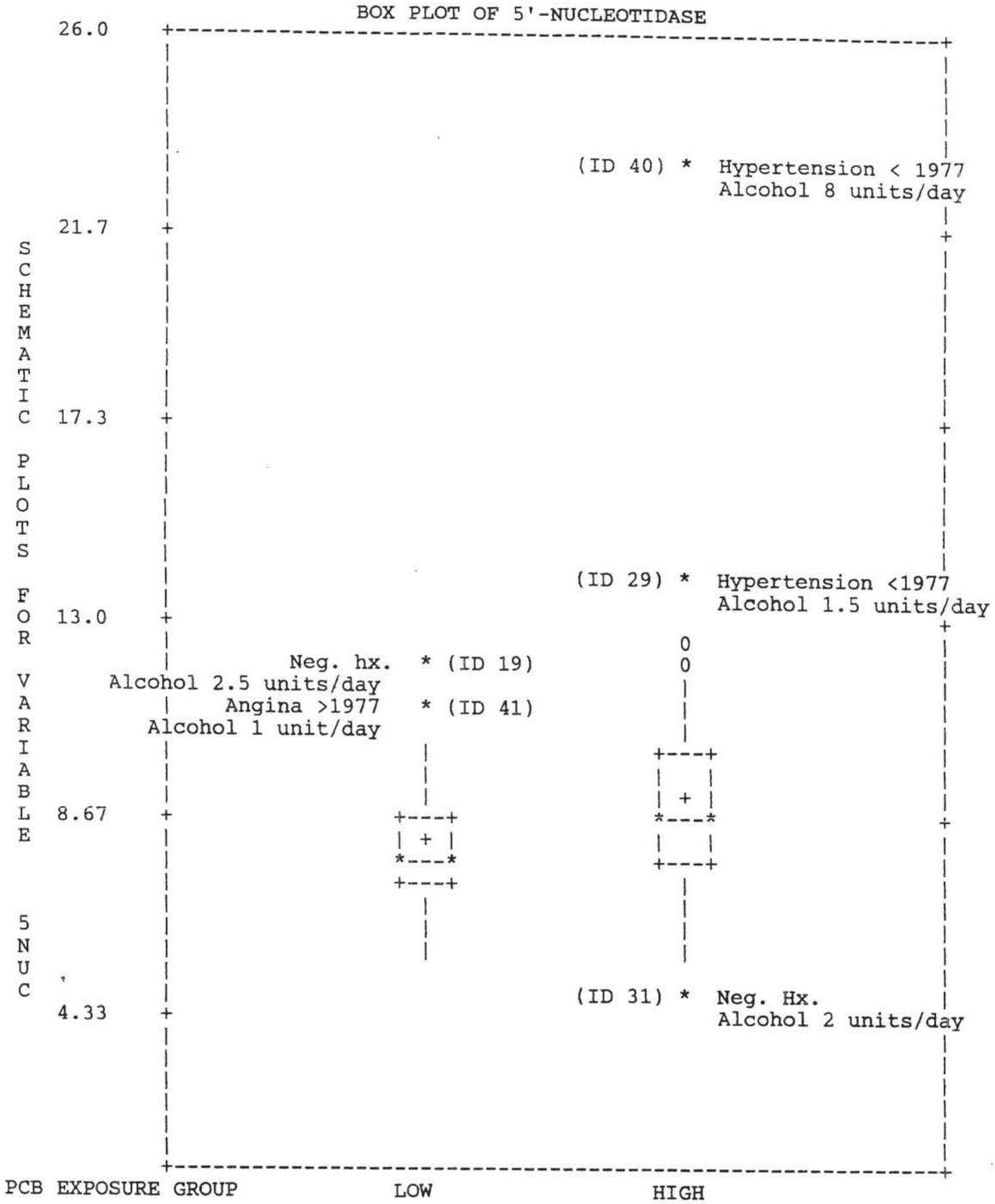

BOX PLOT OF 5'-NUCLEOTIDASE
26.0
21.7
17.3
13.0
8.67
4.33
SCHEMATIC PLOTS FOR VARIABLE 5NUC
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
(ID 29) * Hypertension <1977
Alcohol 1.5 units/day
(ID 31) * Neg. Hx.
Alcohol 2 units/day
Neg. hx. * (ID 19)
Alcohol 2.5 units/day
Angina >1977 * (ID 41)
Alcohol 1 unit/day
0
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 10

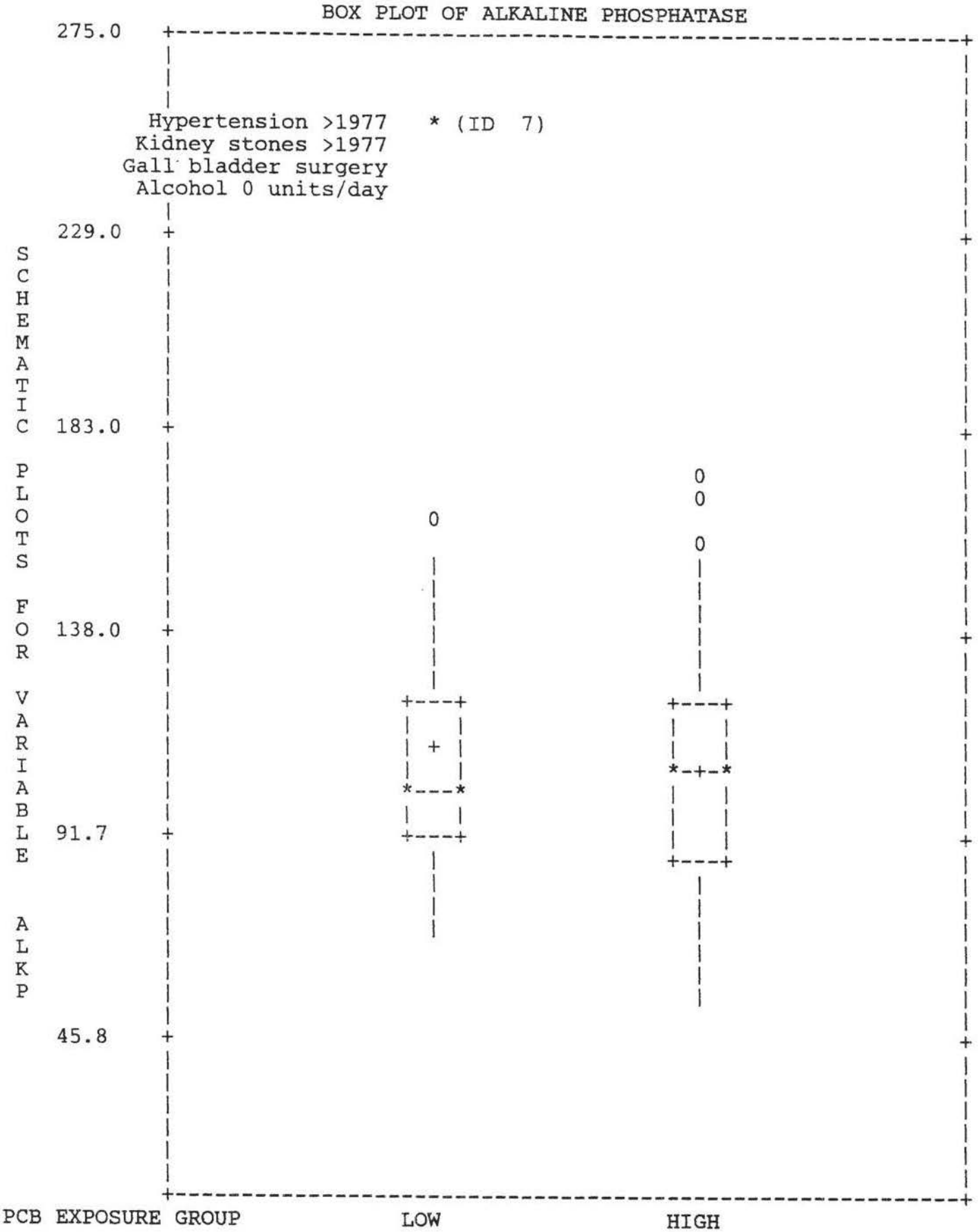

BOX PLOT OF ALKALINE PHOSPHATASE
275.0
Hypertension >1977
Kidney stones >1977
Gall bladder surgery
Alcohol 0 units/day
* (ID 7)
229.0
183.0
138.0
91.7
45.8
SCHEMATIC PLOTS FOR VARIABLE ALKP
PCB EXPOSURE GROUP
LOW
HIGH

Figure 11

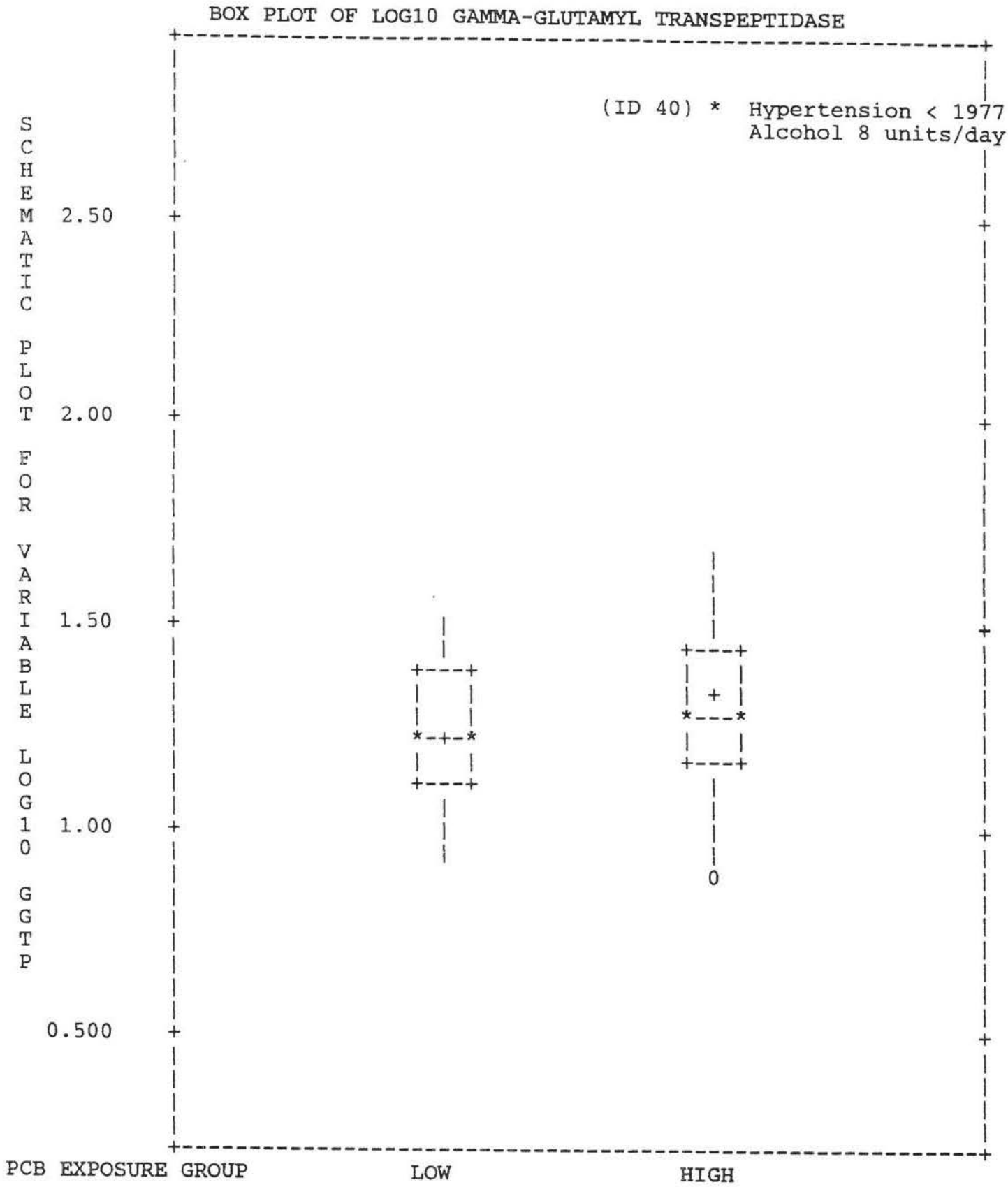

BOX PLOT OF LOG10 GAMMA-GLUTAMYL TRANSPEPTIDASE
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
SCHEMATIC PLOT FOR VARIABLE LOG10 GGTP
2.50
2.00
1.50
1.00
0.500
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 12

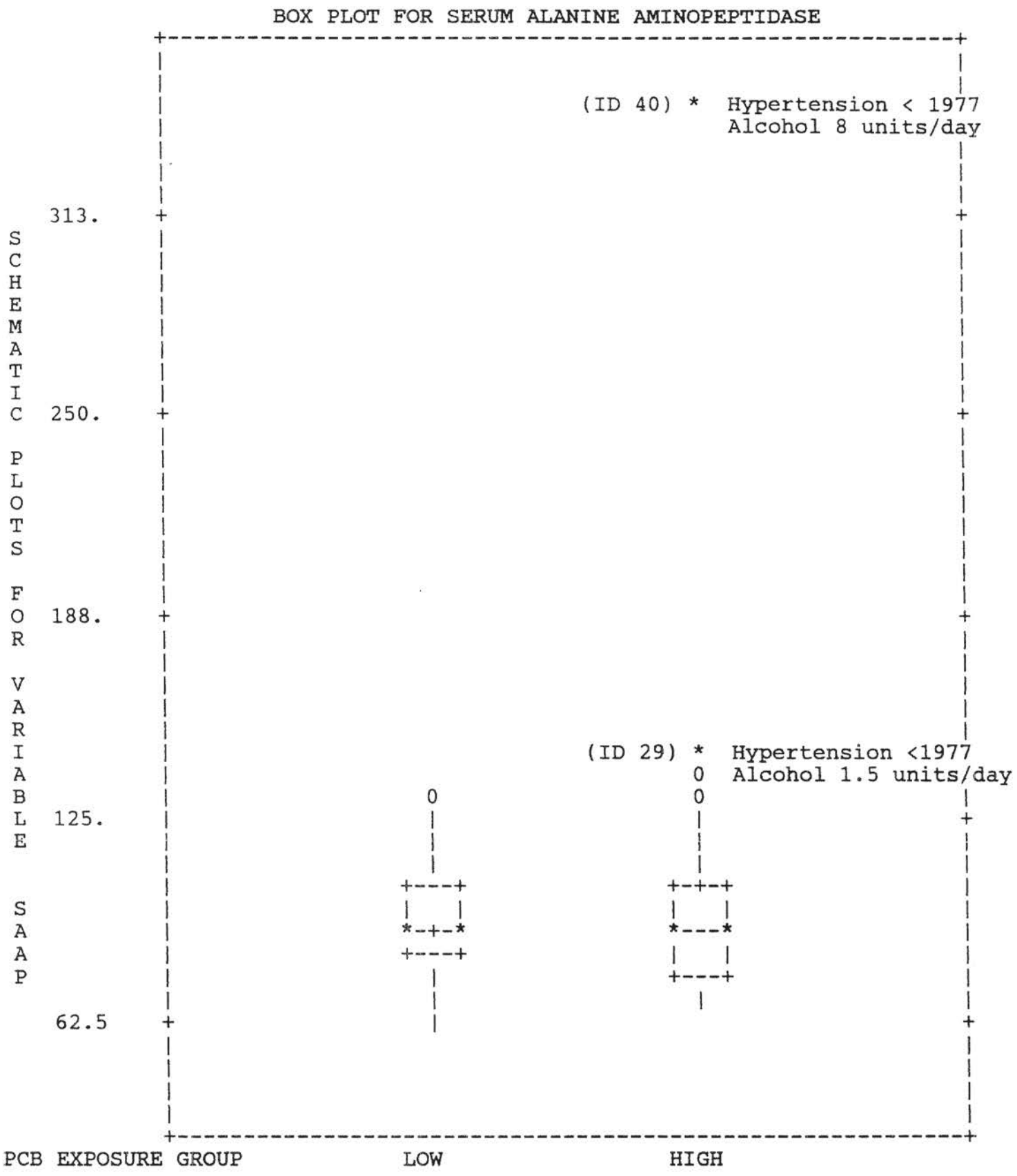

BOX PLOT FOR SERUM ALANINE AMINOPEPTIDASE
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
313.
250.
188.
125.
62.5
SCHEMATIC PLOTS FOR VARIABLE SAAP
(ID 29) * Hypertension <1977
0 Alcohol 1.5 units/day
0
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 13

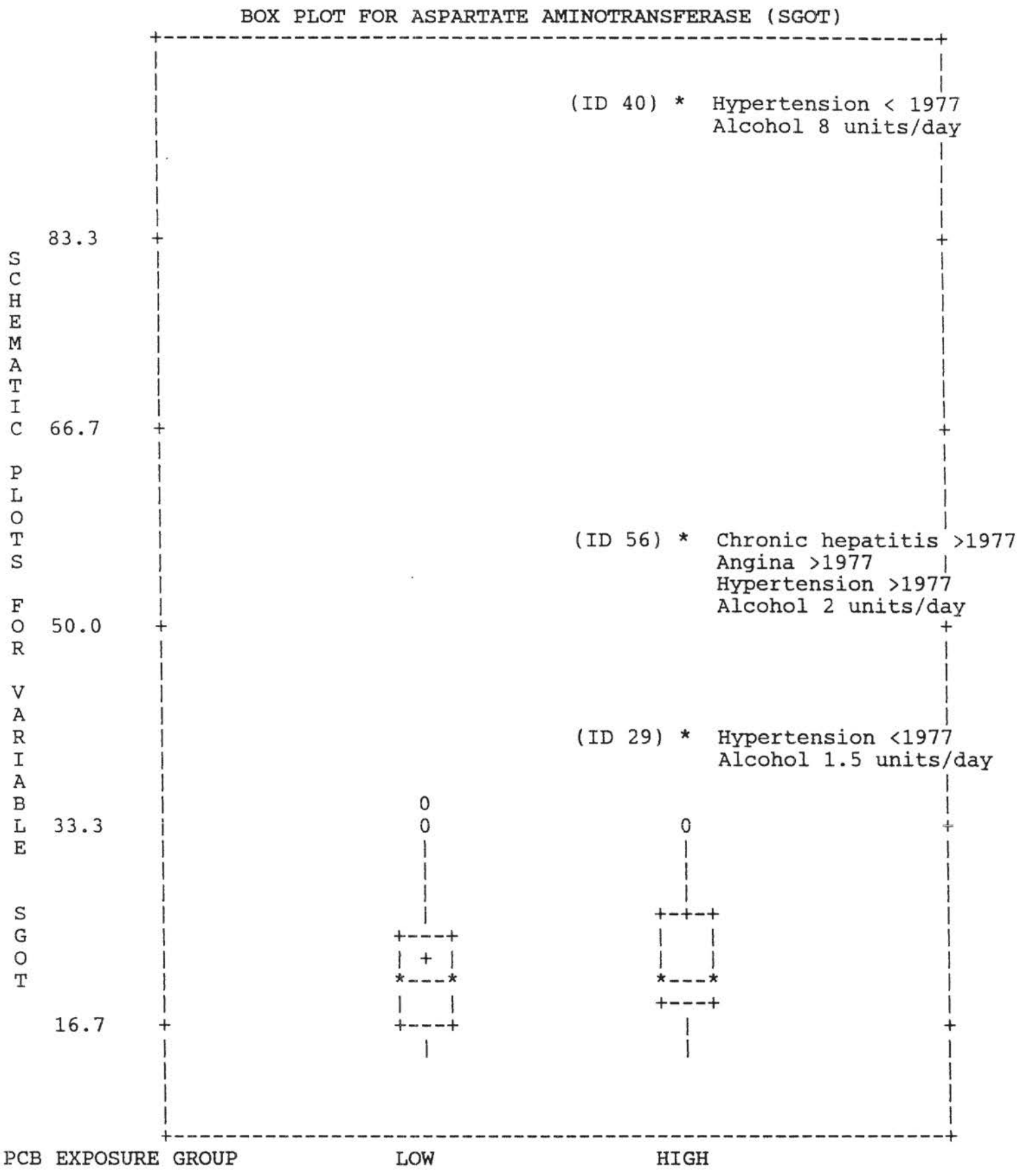

BOX PLOT FOR ASPARTATE AMINOTRANSFERASE (SGOT)
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
(ID 56) * Chronic hepatitis >1977
Angina >1977
Hypertension >1977
Alcohol 2 units/day
(ID 29) * Hypertension <1977
Alcohol 1.5 units/day
83.3
66.7
50.0
33.3
16.7
SCHEMATIC PLOTS FOR VARIABLE SGOT
PCB EXPOSURE GROUP
LOW
HIGH

Figure 14

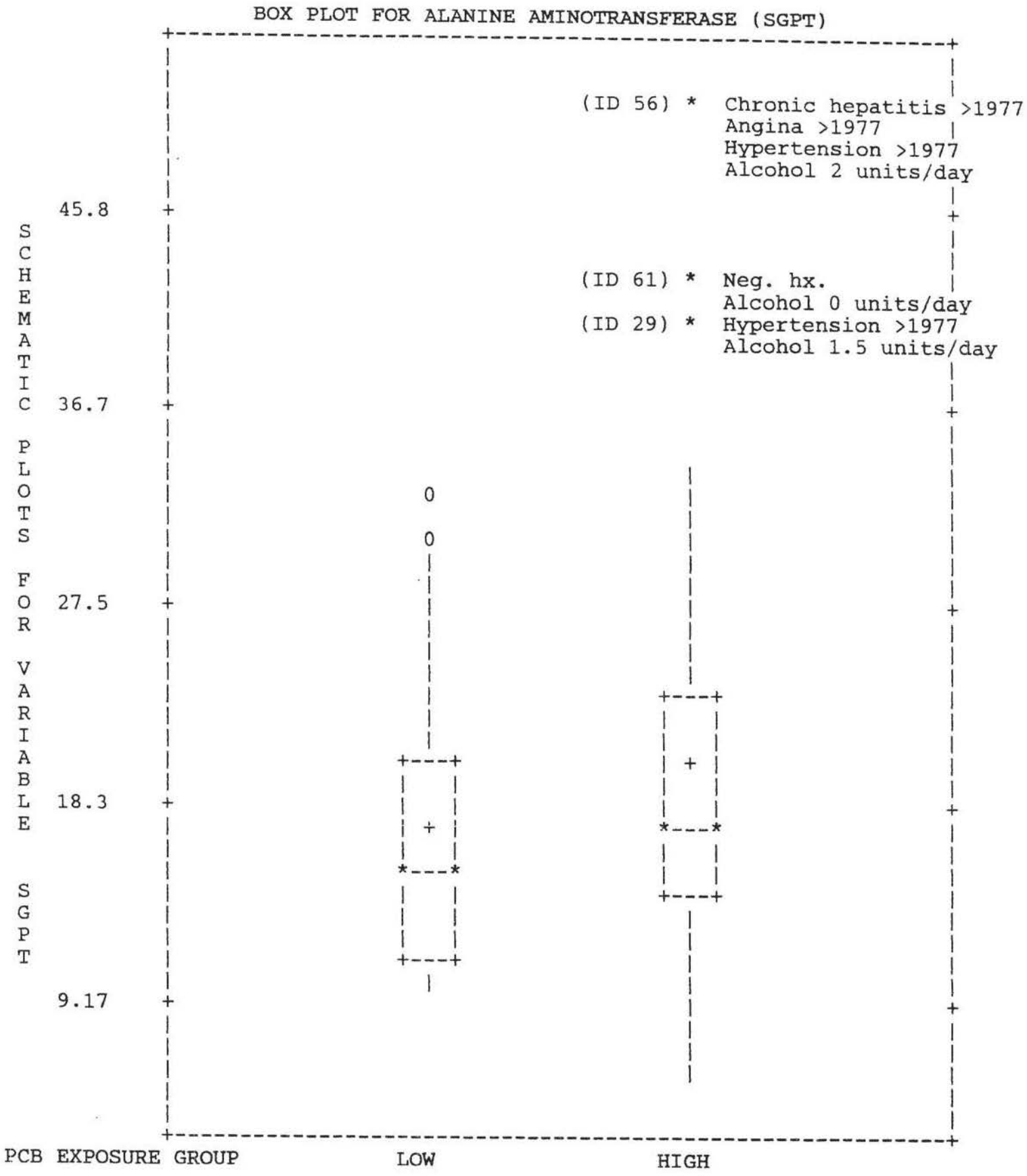

BOX PLOT FOR ALANINE AMINOTRANSFERASE (SGPT)
(ID 56) * Chronic hepatitis >1977
Angina >1977
Hypertension >1977
Alcohol 2 units/day
(ID 61) * Neg. hx.
Alcohol 0 units/day
(ID 29) * Hypertension >1977
Alcohol 1.5 units/day
SCHEMATIC PLOTS FOR VARIABLE SGPT
45.8
36.7
27.5
18.3
9.17
0
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 15

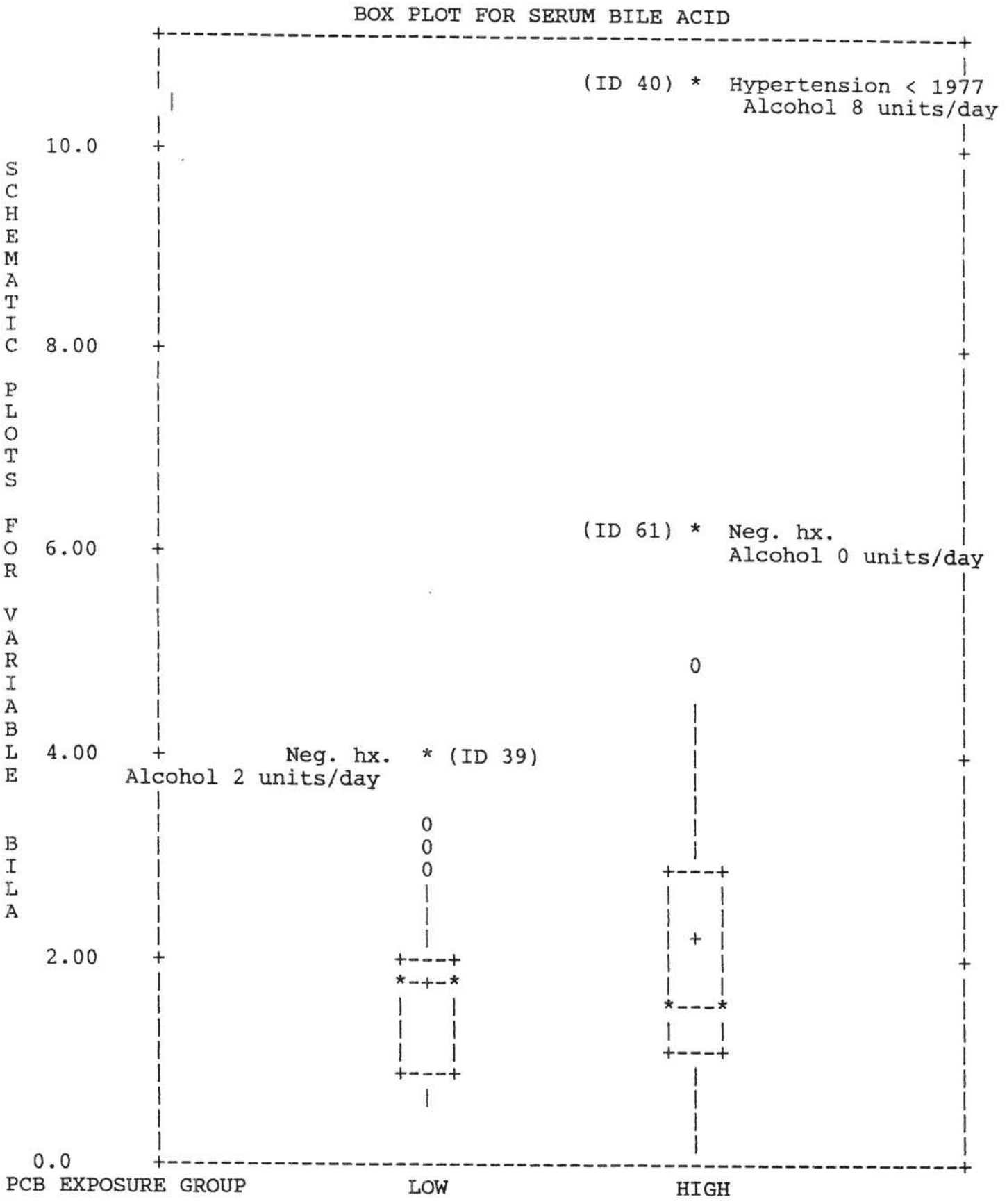

BOX PLOT FOR SERUM BILE ACID
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
10.0
8.00
6.00
4.00
2.00
0.0
SCHEMATIC PLOTS FOR VARIABLE BILA
(ID 61) * Neg. hx.
Alcohol 0 units/day
Neg. hx. * (ID 39)
Alcohol 2 units/day
PCB EXPOSURE GROUP
LOW
HIGH

Figure 16

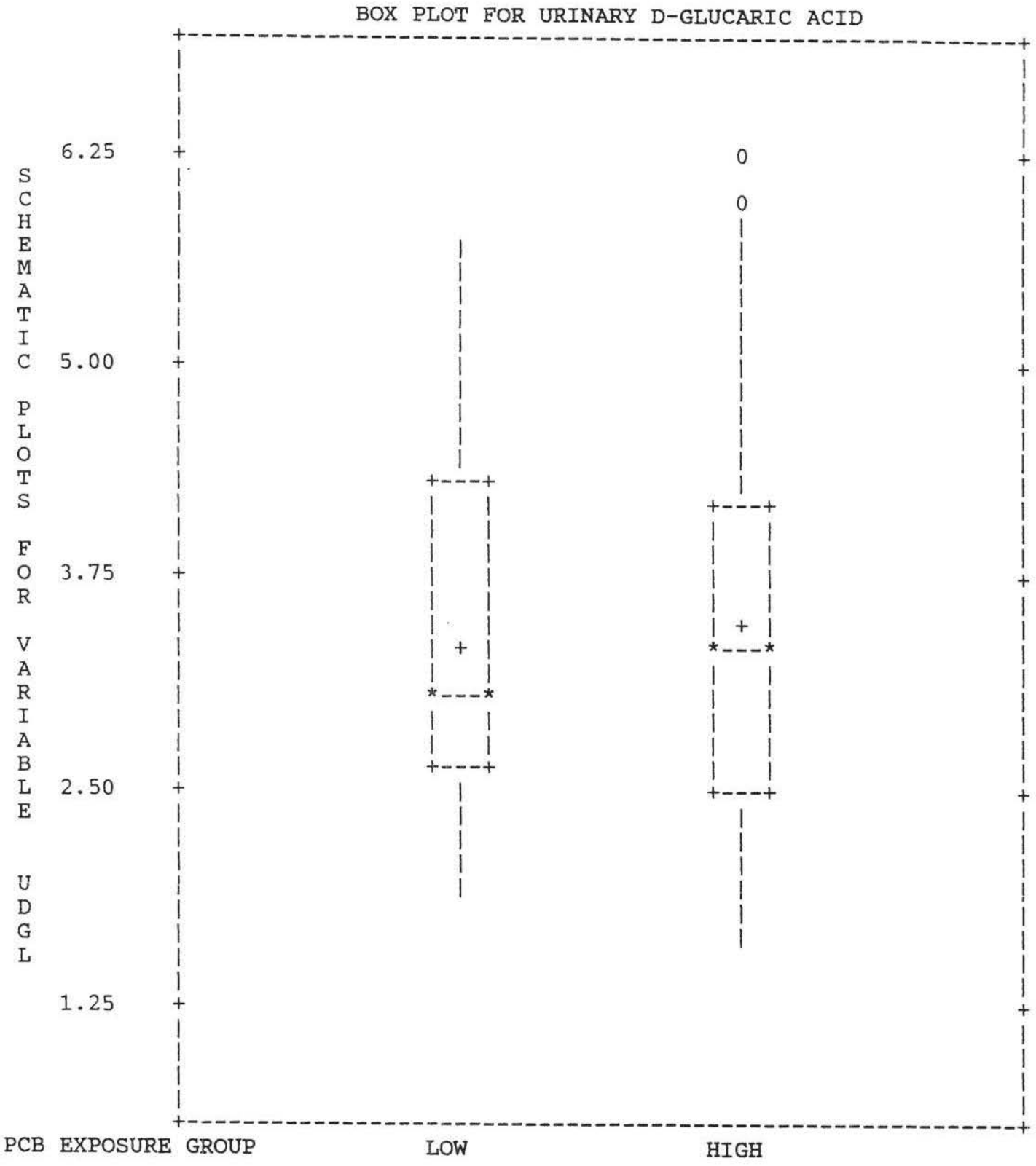

BOX PLOT FOR URINARY D-GLUCARIC ACID
SCHEMATIC PLOTS FOR VARIABLE UDGL
6.25
5.00
3.75
2.50
1.25
0
0
PCB EXPOSURE GROUP
LOW
HIGH

Figure 17

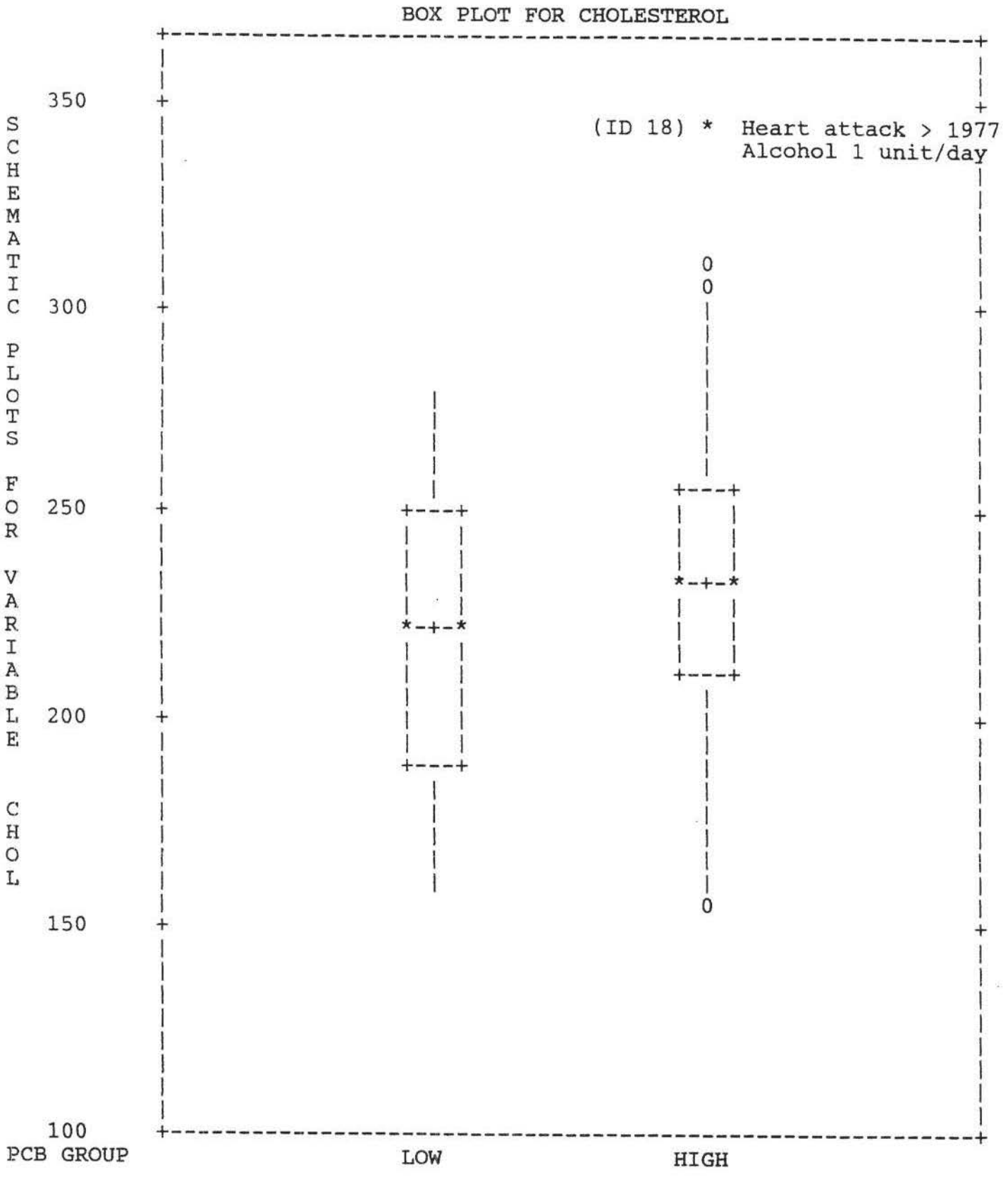

BOX PLOT FOR CHOLESTEROL
(ID 18) * Heart attack > 1977
Alcohol 1 unit/day
SCHEMATIC PLOTS FOR VARIABLE CHOL
350
300
250
200
150
100
PCB GROUP
LOW
HIGH

Figure 18

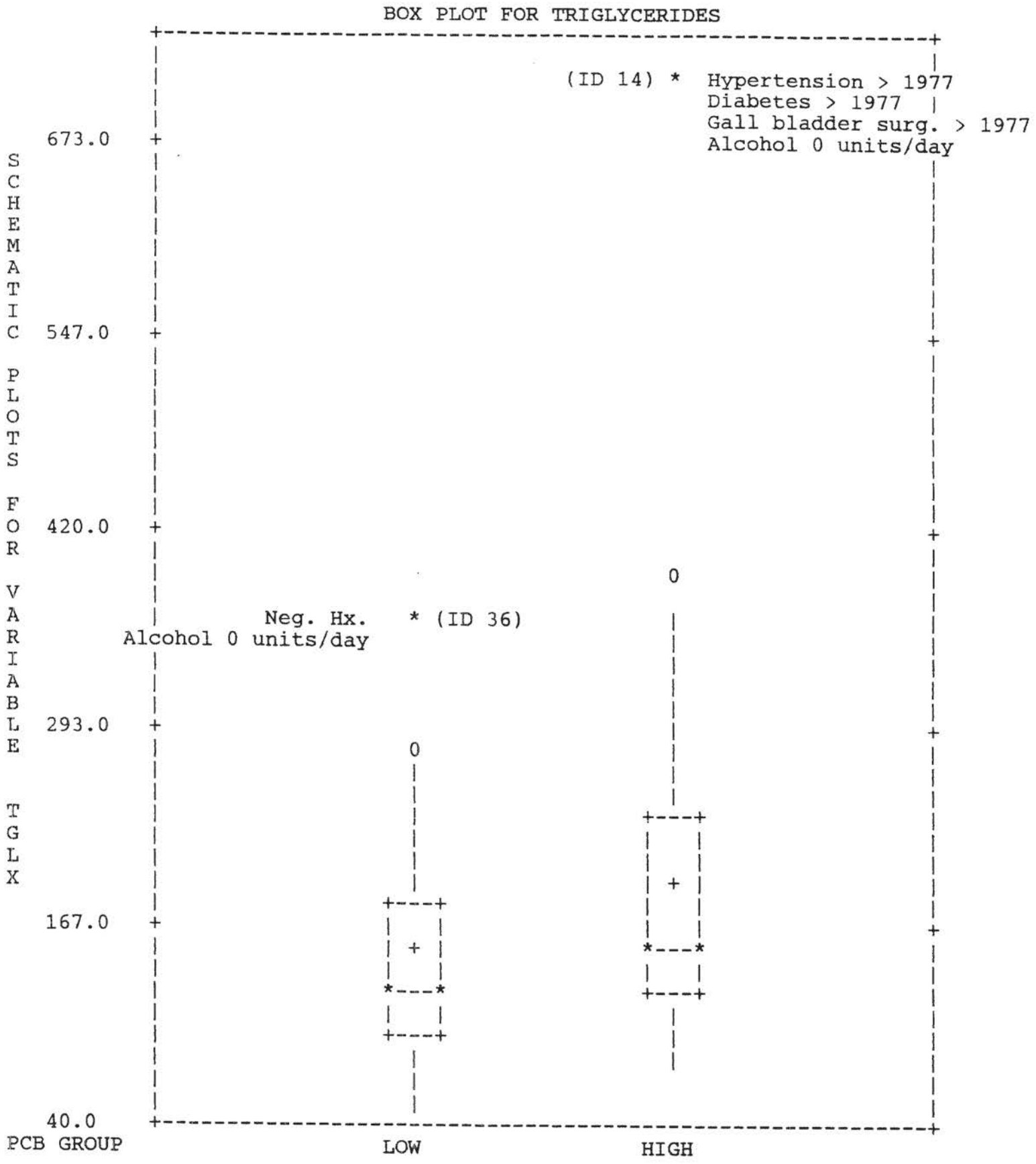

BOX PLOT FOR TRIGLYCERIDES
(ID 14) * Hypertension > 1977
Diabetes > 1977
Gall bladder surg. > 1977
Alcohol 0 units/day
673.0
547.0
420.0
293.0
167.0
40.0
SCHEMATIC PLOTS FOR VARIABLE TGLX
Neg. Hx. * (ID 36)
Alcohol 0 units/day
0
0
PCB GROUP
LOW
HIGH

Figure 19

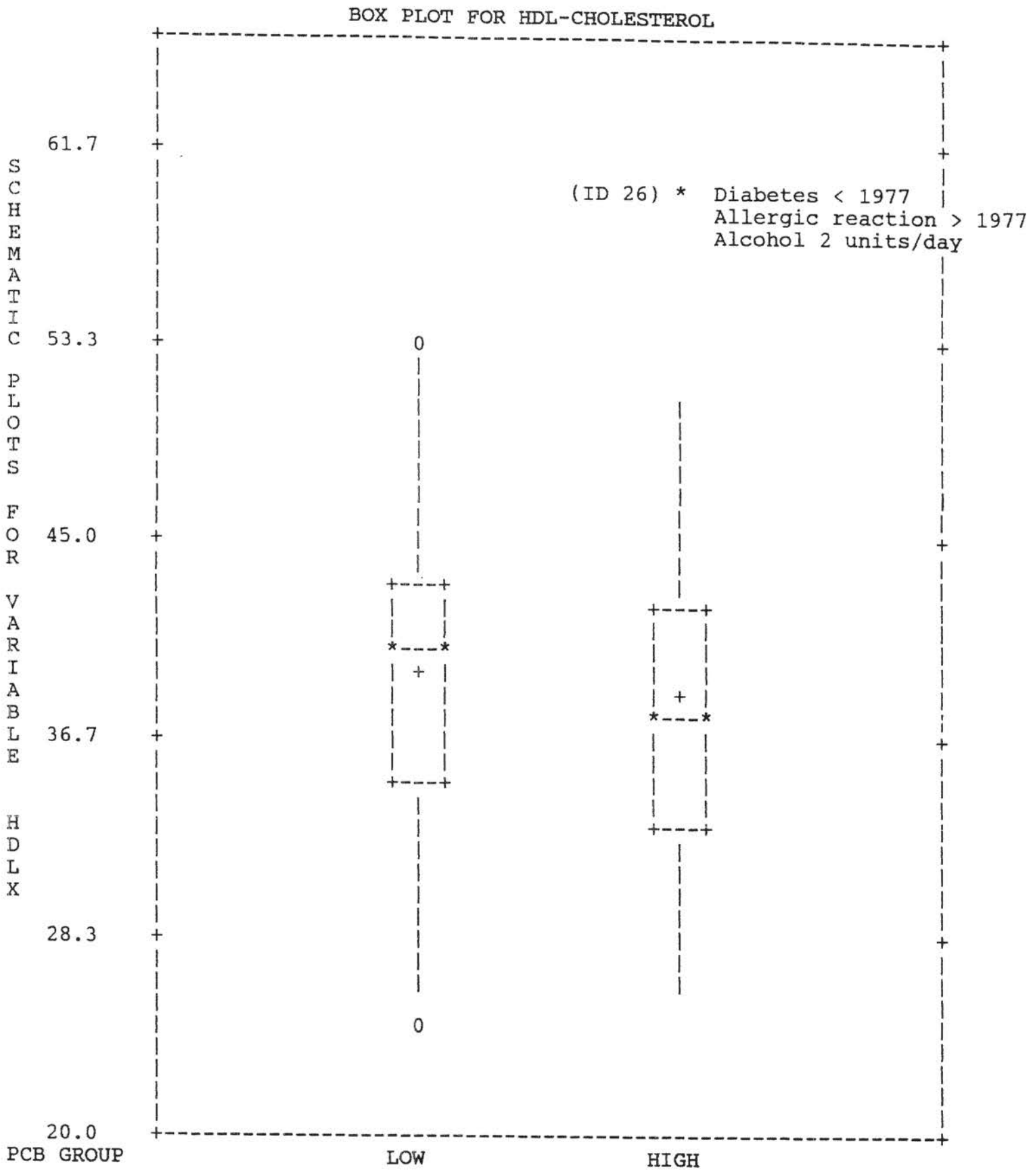

BOX PLOT FOR HDL-CHOLESTEROL
SCHEMATIC PLOTS FOR VARIABLE HDLX
61.7
53.3
45.0
36.7
28.3
20.0
(ID 26) * Diabetes < 1977
Allergic reaction > 1977
Alcohol 2 units/day
0
0
PCB GROUP
LOW
HIGH

Figure 20

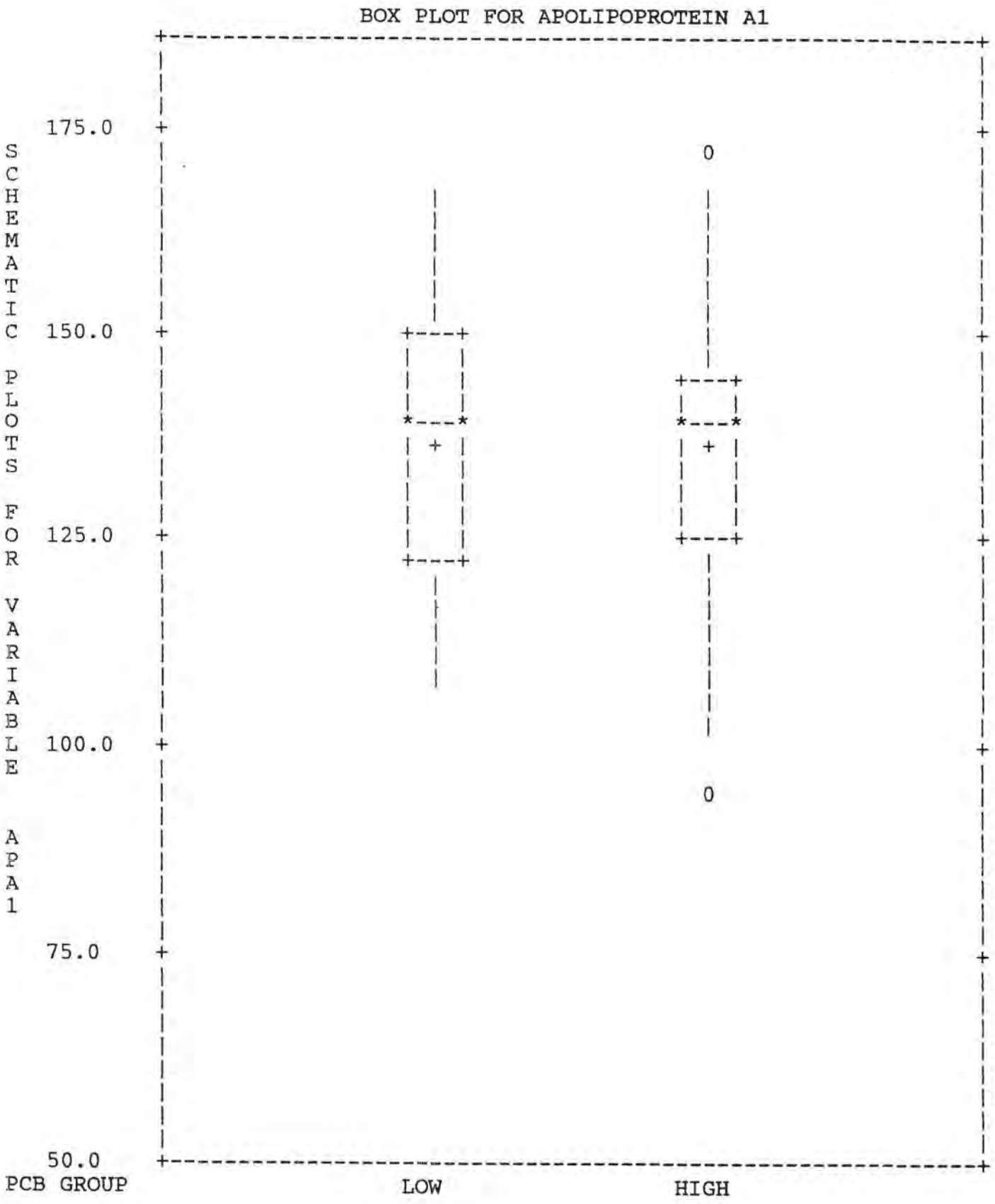

BOX PLOT FOR APOLIPOPROTEIN A1
SCHEMATIC PLOTS FOR VARIABLE APA1
175.0
150.0
125.0
100.0
75.0
50.0
0
0
PCB GROUP
LOW
HIGH

Figure 21

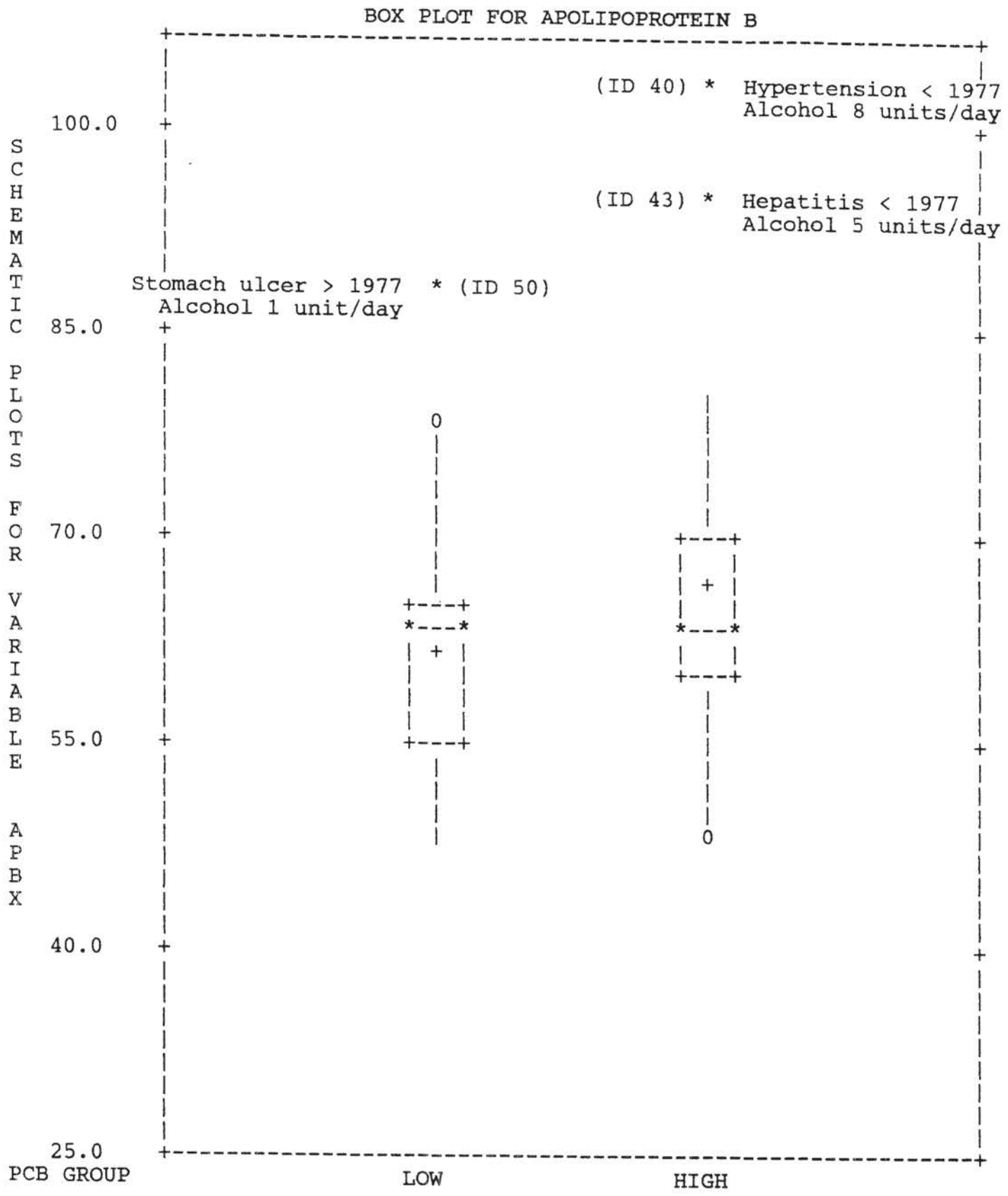

BOX PLOT FOR APOLIPOPROTEIN B
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
(ID 43) * Hepatitis < 1977
Alcohol 5 units/day
Stomach ulcer > 1977 * (ID 50)
Alcohol 1 unit/day
SCHEMATIC PLOTS FOR VARIABLE APBX
100.0
85.0
70.0
55.0
40.0
25.0
PCB GROUP
LOW
HIGH

Figure 22

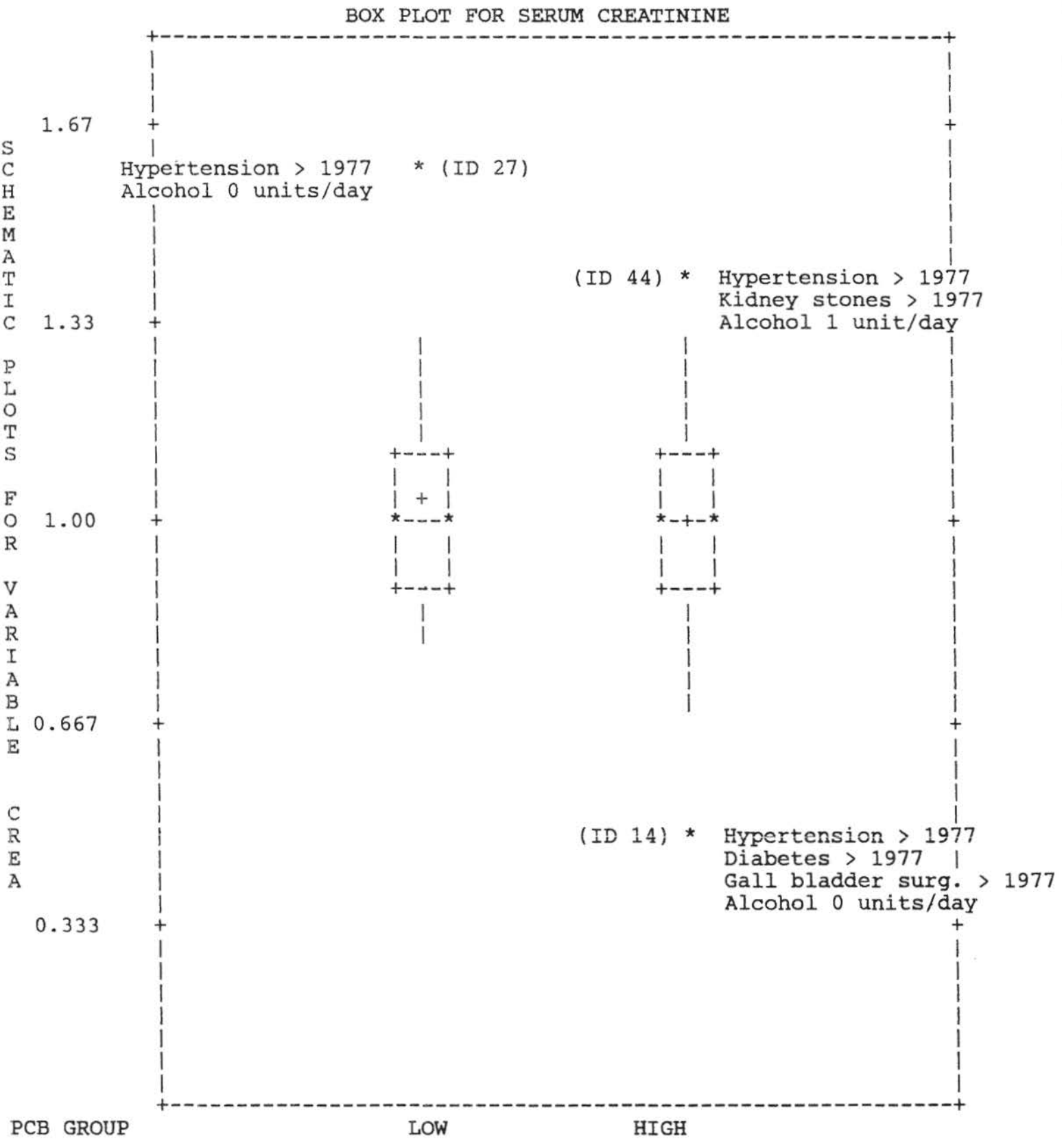

BOX PLOT FOR SERUM CREATININE
1.67
1.33
1.00
0.667
0.333
SCHEMATIC PLOTS FOR VARIABLE CREA
Hypertension > 1977 * (ID 27)
Alcohol 0 units/day
(ID 44) * Hypertension > 1977
Kidney stones > 1977
Alcohol 1 unit/day
(ID 14) * Hypertension > 1977
Diabetes > 1977
Gall bladder surg. > 1977
Alcohol 0 units/day
PCB GROUP
LOW
HIGH

Figure 23

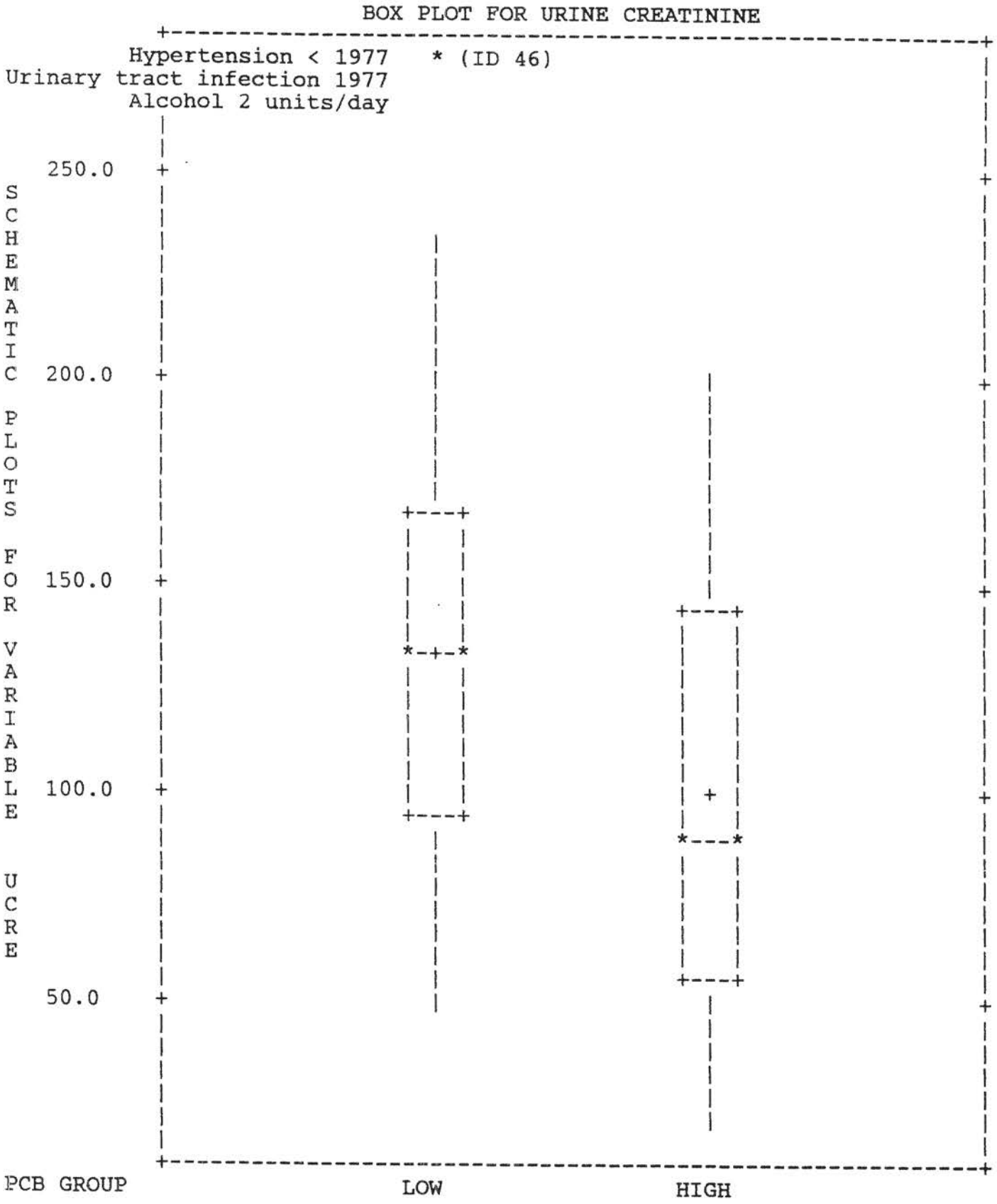

BOX PLOT FOR URINE CREATININE
Hypertension < 1977 * (ID 46)
Urinary tract infection 1977
Alcohol 2 units/day
SCHEMATIC PLOTS FOR VARIABLE URCRE
250.0
200.0
150.0
100.0
50.0
PCB GROUP
LOW
HIGH

Figure 24

BOX PLOT FOR N-ACETYL GLUCOSAMINIDASE
(UNADJUSTED FOR URINE CREATININE EXCRETION)

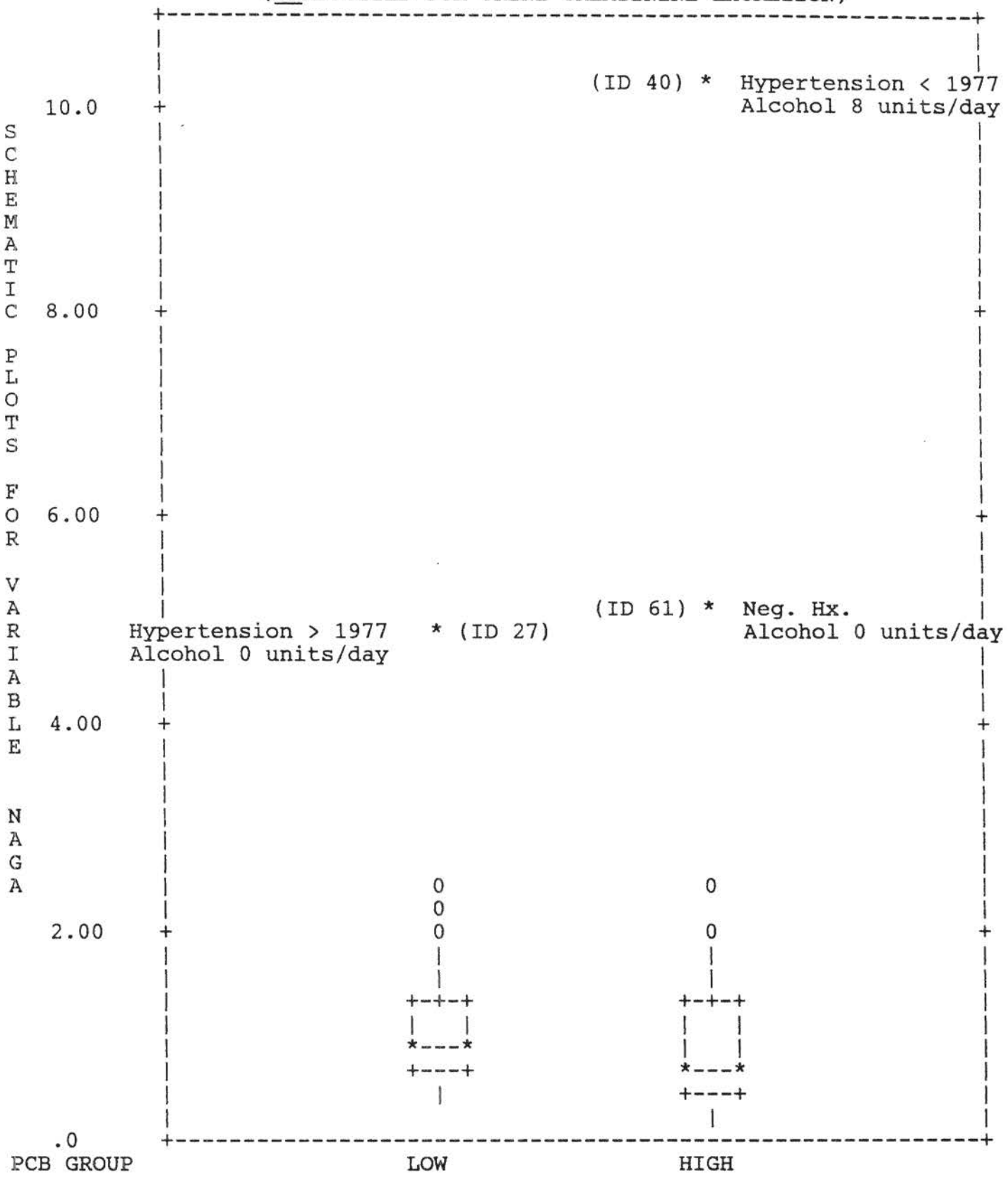


Figure 25

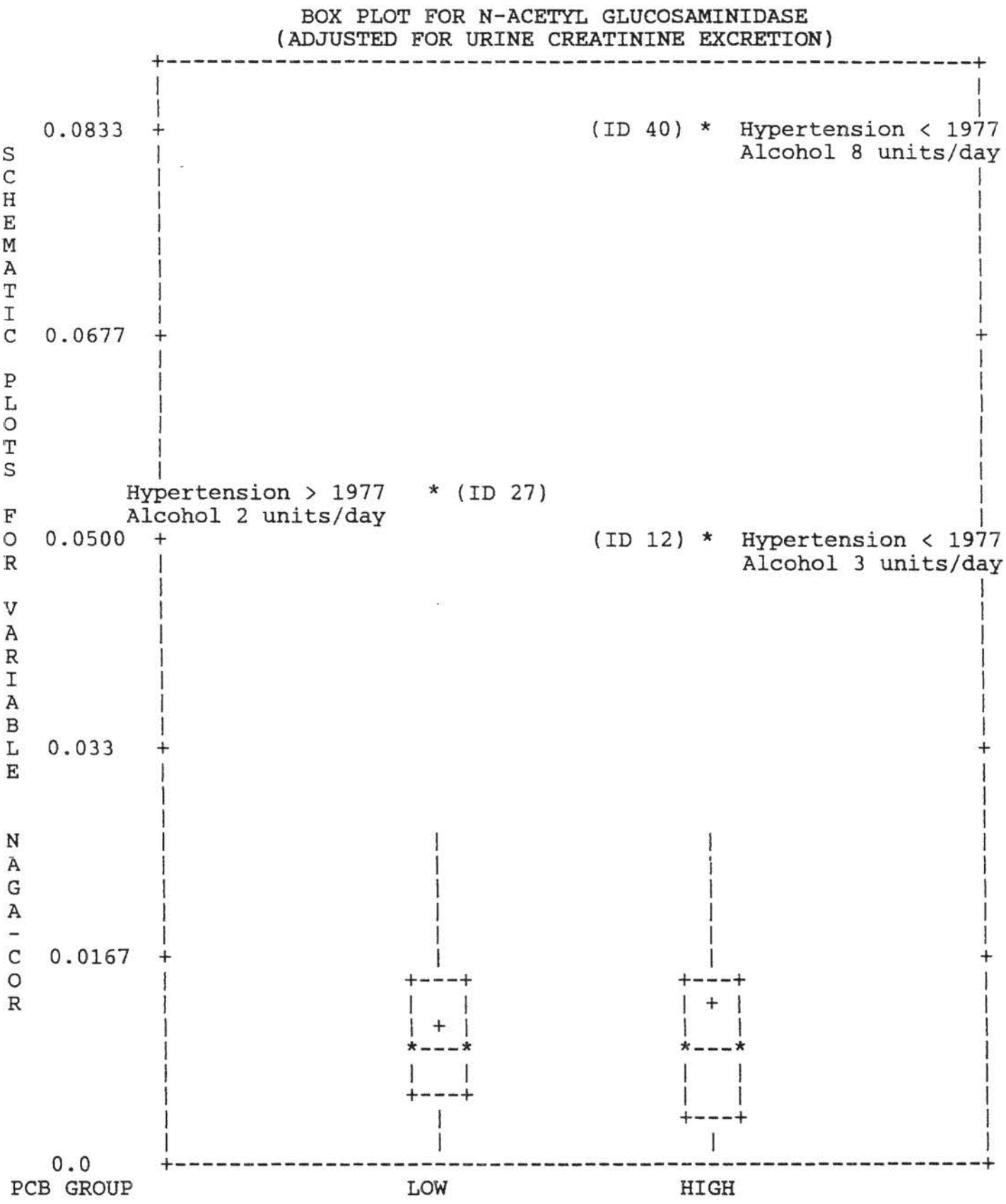

BOX PLOT FOR N-ACETYL GLUCOSAMINIDASE
(ADJUSTED FOR URINE CREATININE EXCRETION)
SCHEMATIC PLOTS FOR VARIABLE NAGA-COR
0.0833
0.0677
0.0500
0.033
0.0167
0.0
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
Hypertension > 1977 * (ID 27)
Alcohol 2 units/day
(ID 12) * Hypertension < 1977
Alcohol 3 units/day
PCB GROUP
LOW
HIGH

Figure 26

BOX PLOT FOR URINARY GAMMA GLUTAMYL TRANSPEPTIDASE
(UNADJUSTED FOR CREATININE EXCRETION)

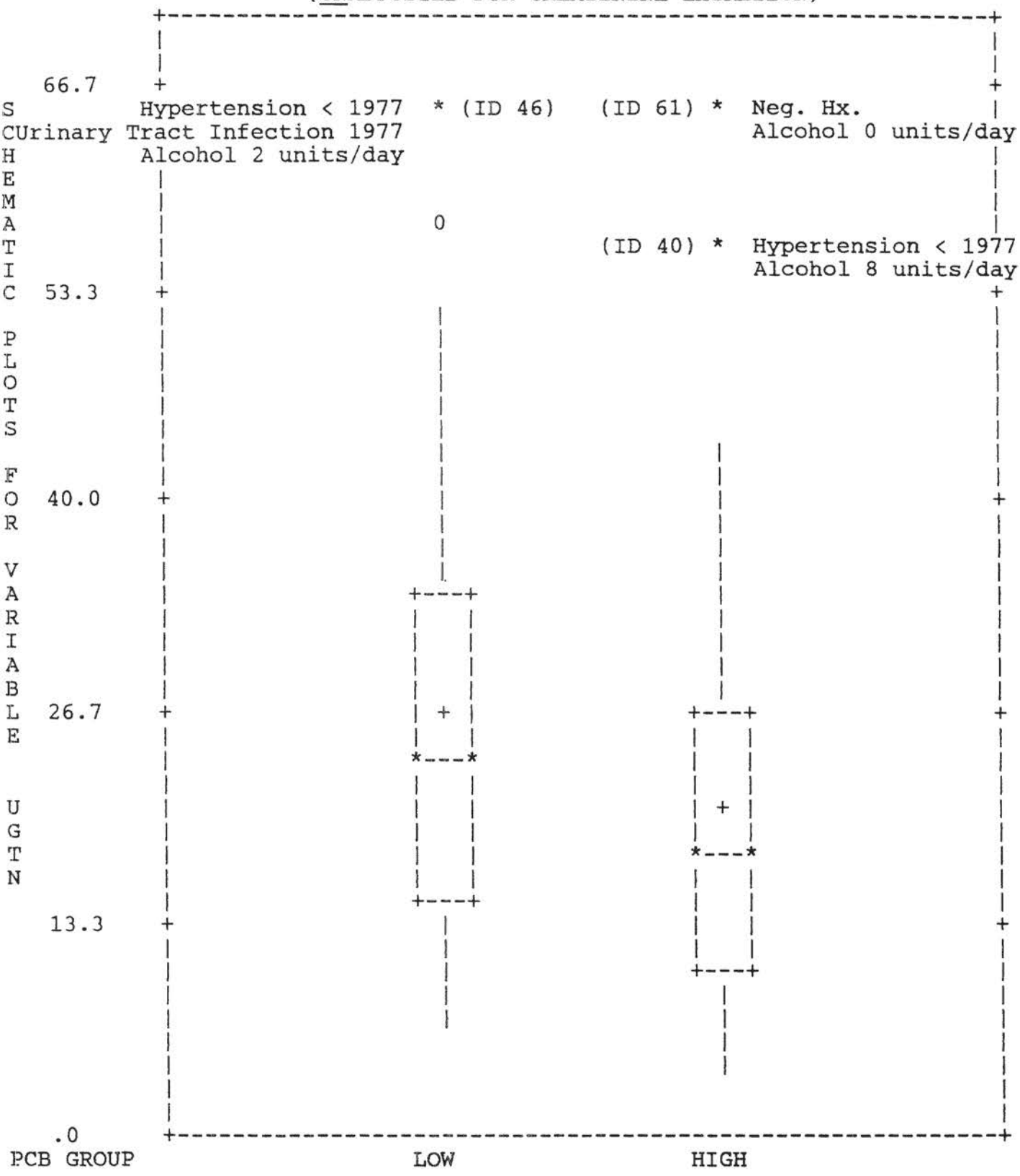


Figure 27

BOX PLOT FOR URINARY GAMMA GLUTAMYL TRANSPEPTIDASE
(ADJUSTED FOR CREATININE EXCRETION)

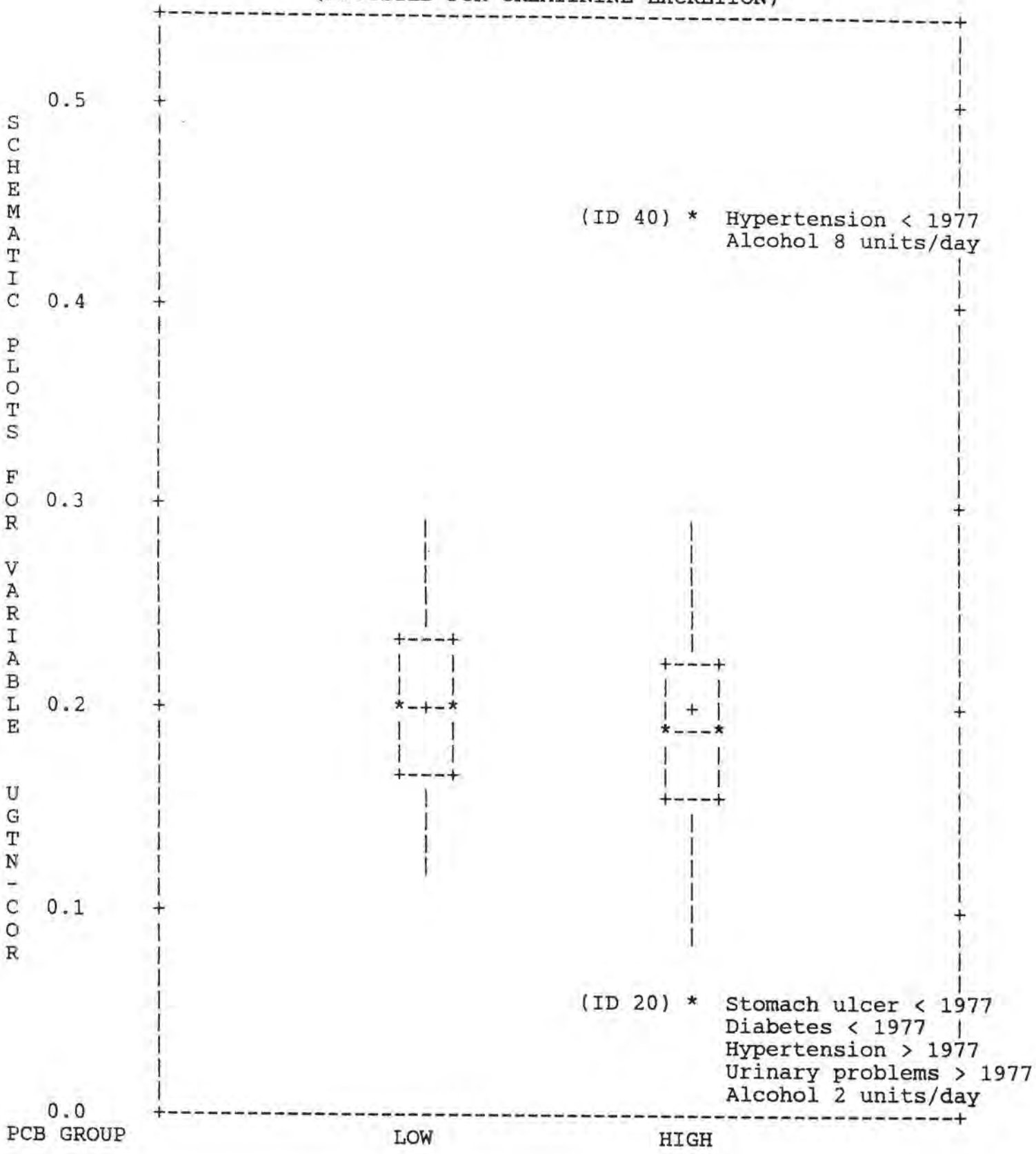


Figure 28

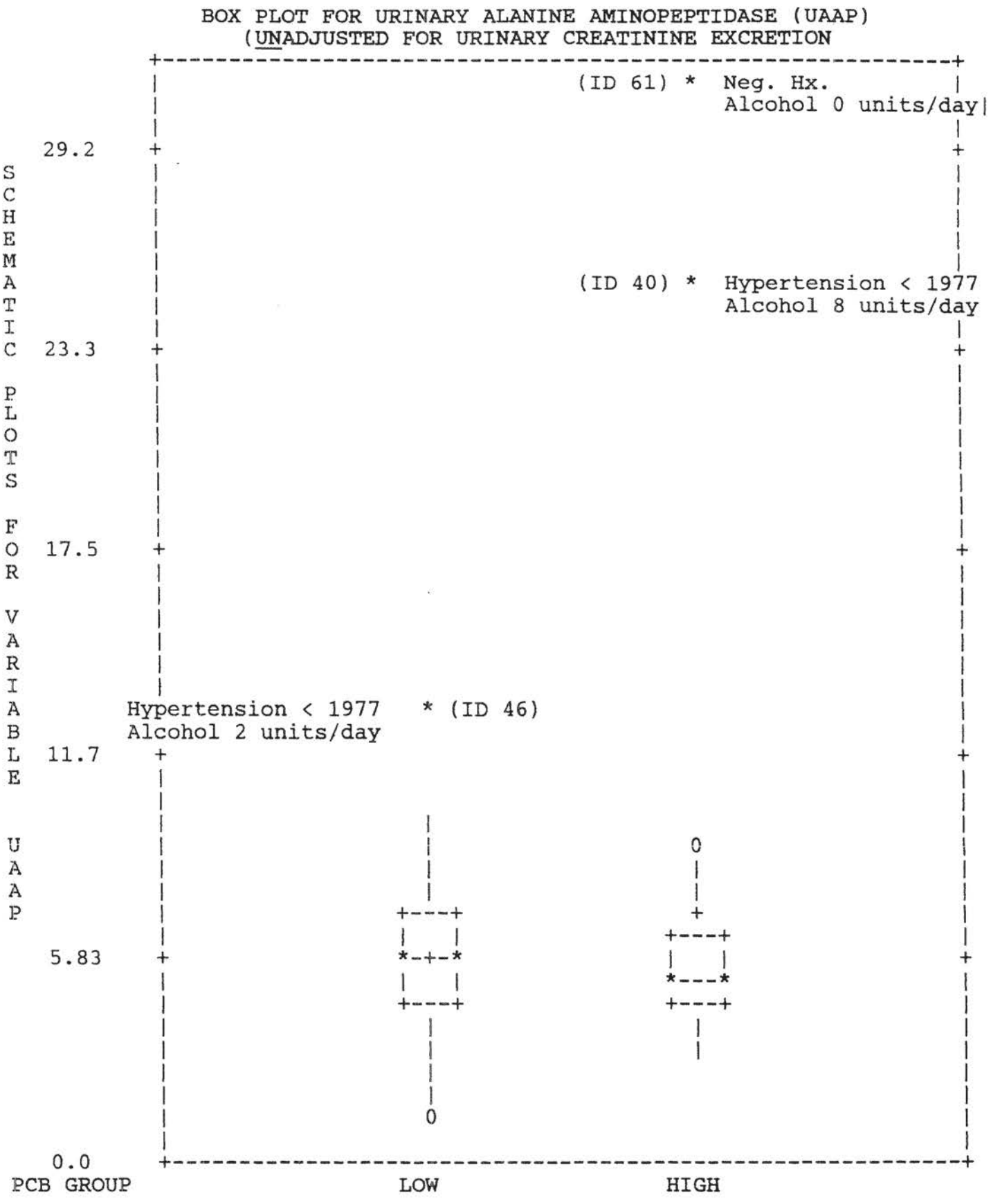
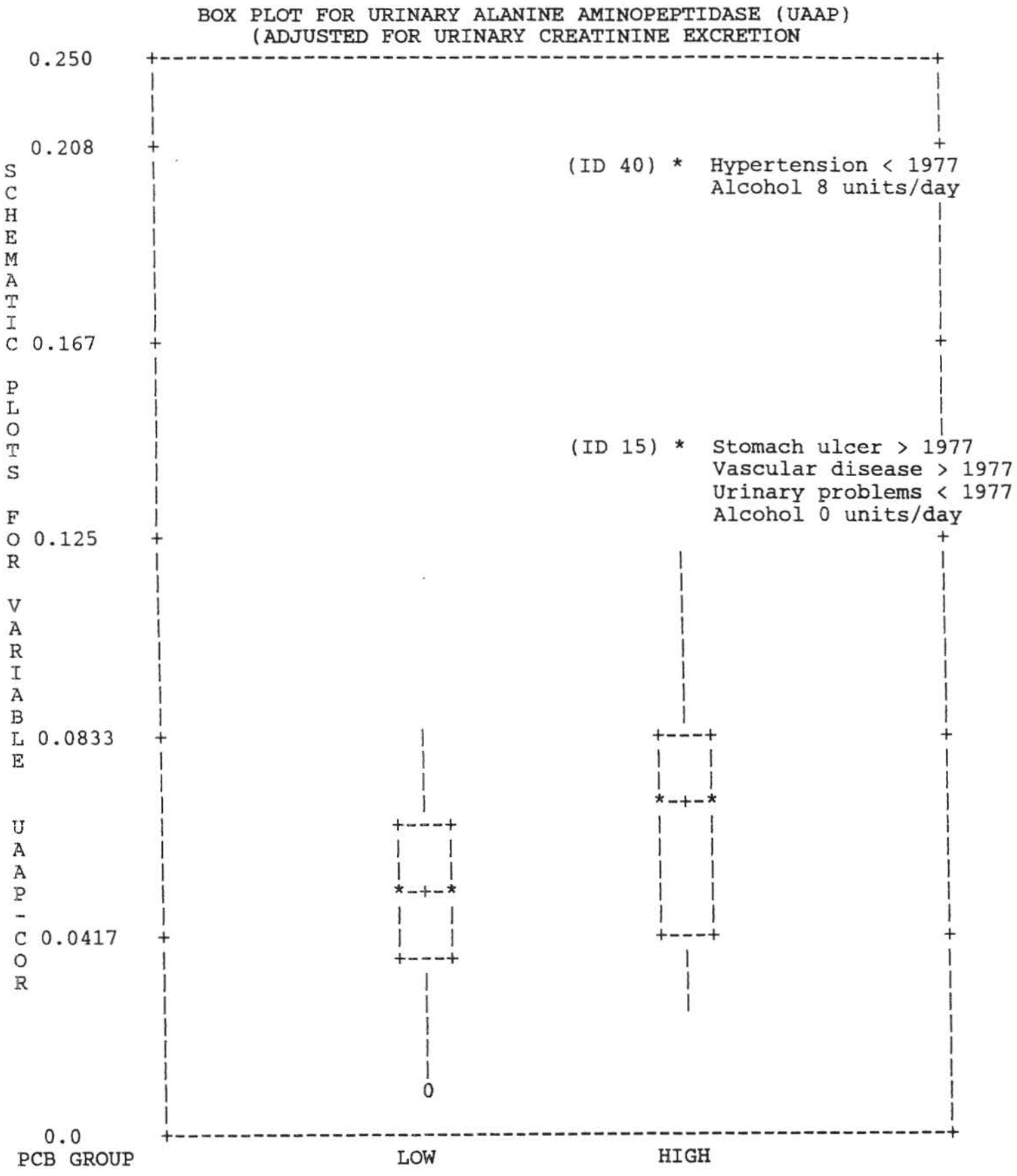

BOX PLOT FOR URINARY ALANINE AMINOPEPTIDASE (UAAP)
(UNADJUSTED FOR URINARY CREATININE EXCRETION
(ID 61) * Neg. Hx.
Alcohol 0 units/day
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
Hypertension < 1977 * (ID 46)
Alcohol 2 units/day
SCHEMATIC PLOTS FOR VARIABLE UAAP
29.2
23.3
17.5
11.7
5.83
0.0
PCB GROUP
LOW
HIGH

Figure 29


BOX PLOT FOR URINARY ALANINE AMINOPEPTIDASE (UAAP)
(ADJUSTED FOR URINARY CREATININE EXCRETION
SCHEMATIC PLOTS FOR VARIABLE UAAP-COR
0.250
0.208
0.167
0.125
0.0833
0.0417
0.0
(ID 40) * Hypertension < 1977
Alcohol 8 units/day
(ID 15) * Stomach ulcer > 1977
Vascular disease > 1977
Urinary problems < 1977
Alcohol 0 units/day
0
PCB GROUP
LOW
HIGH

APPENDIX I

WESTINGHOUSE
BLOOMINGTON, INDIANA
HETA 84-339

QUESTIONNAIRE

I.D. Number: |_|_|_|_|_|
(1-5)

Today's date: |_|_| |_|_| |_|_|
(6-11) Month Day Year

Name: |_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|
Last name (12-26)

|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_| |_|
First name (27-41) Middle initial (42)

Address: |_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|
Street (43-62)

|0|1|
(79-80)

|_|_|_|_|_|_|_|_|_|_|_|_|_|_|_|
City (6-20)

|_|_| |_|_|_|_|_|
State (21-22) Zip code (23-27)

Telephone number: Home (28-37) |_|_|_| - |_|_|_| - |_|_|_|_|
Area code

PERSONAL DATA:

Race (check one): (48)

White, not of Hispanic origin	____	1
Black, not of Hispanic origin	____	2
Hispanic	____	3
American Indian or Alaskan Native	____	4
Asian or Pacific Islander	____	5

Sex (check one): (49)

Male	____	1
Female	____	2

Date of birth: |_|_| - |_|_| - |_|_|
(50-55) Month Day Year

|0|2|
(79-80)

OCCUPATIONAL HISTORY:

We have copies of your job history as of April 1, 1977. Please tell us about all your jobs since April 1, 1977, either at Westinghouse or at any other employer.

	Westinghouse?	Department/Operation or description	Dates Started, Stopped	[LEAVE BLANK]
1.	YES 1 NO 2			(6-7)
2.	YES 1 NO 2			(8-9)
3.	YES 1 NO 2			(10-11)
4.	YES 1 NO 2			(12-13)
5.	YES 1 NO 2			(14-15)
6.	YES 1 NO 2			(16-17)
7.	YES 1 NO 2			(18-19)
8.	YES 1 NO 2			(20-21)
9.	YES 1 NO 2			(22-23)
10.	YES 1 NO 2			(24-25)

0 3
(79-80)

SYMPTOMS:

Now I'm going to read a list of health conditions. Please tell me if you have had in the past year any of the following while you were on the job, or soon after work. Answer YES or NO to each condition.

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

CONDITION	RESPONSE	FREQUENCY	CURRENT
11. Coughing (6-8)	YES___1 NO___2 DK___8	1-TIME___1 1-4/MO___2 1-4/WK___3 5-7/WK___4 DK___8	YES___1 NO___2 DK___8
12. Wheezing (9-11)	YES___1 NO___2 DK___8	1-TIME___1 1-4/MO___2 1-4/WK___3 5-7/WK___4 DK___8	YES___1 NO___2 DK___8
13. Sudden, unexplained tightness in your chest (12-14)	YES___1 NO___2 DK___8	1-TIME___1 1-4/MO___2 1-4/WK___3 5-7/WK___4 DK___8	YES___1 NO___2 DK___8
14. Sudden, unexplained shortness of breath (15-17)	YES___1 NO___2 DK___8	1-TIME___1 1-4/MO___2 1-4/WK___3 5-7/WK___4 DK___8	YES___1 NO___2 DK___8
15. Nausea or feeling sick to your stomach (18-20)	YES___1 NO___2 DK___8	1-TIME___1 1-4/MO___2 1-4/WK___3 5-7/WK___4 DK___8	YES___1 NO___2 DK___8

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

CONDITION	RESPONSE	FREQUENCY	CURRENT
16. Vomiting (21-23)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8
17. Feeling light-headed (24-26)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8
18. Fainting (27-29)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8
19. Headaches (30-32)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8
20. Burning or irritated eyes (33-35)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8

0 4
(79-80)

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this?
(IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

ONSET: In about what year did you first notice this?

CONDITION	RESPONSE	FREQUENCY	CURRENT	ONSET
21. Unusually poor appetite (6-10)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
22. Lasting or repeated diarrhea (11-15)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
23. Lasting or repeated constipation (16-20)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
24. Feeling tired or rundown most of the time (21-25)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
25. Trouble concentrating or remembering things (26-30)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
26. Frequently feeling tense and irritable (31-35)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

ONSET: In about what year did you first notice this?

CONDITION	RESPONSE	FREQUENCY	CURRENT	ONSET
27. Feeling as though your eyelids were swollen (36-40)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
28. Feeling as though your feet were swollen (41-45)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
29. Unusual darkening of your fingernails (46-50)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
30. Bloody or red urine (51-55)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
31. Loss of your sense of smell (56-60)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
32. Decreased interest in sex (61-65)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
33. Chest pain, pressure or heaviness, when you hurry or walk up-hill (66-70)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|

\|0\|5\| (79-80)

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

ONSET: In about what year did you first notice this?

LOCATION: What part of the body is affected? (See location codes at end of questionnaire.)

CONDITION	RESPONSE	FREQUENCY	CURRENT	ONSET
34. Arthritis or pain in your joints (6-10)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (11-16)	|_|_| |_|_| |_|_|	
35. Weak muscles (17-21)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (22-27)	|_|_| |_|_| |_|_|	
36. Unusual dark patches of skin (28-32)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (33-38)	|_|_| |_|_| |_|_|	
37. Unexplained tingling in your hands or legs (39-43)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (44-49)	|_|_| |_|_| |_|_|	

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

ONSET: In about what year did you first notice this?

LOCATION: What part of the body is affected? (See location codes at end of questionnaire.)

CONDITION	RESPONSE	FREQUENCY	CURRENT	ONSET
38. Unexplained loss or diminished sense of touch (50-54)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (55-60)	|_|_|_| |_|_|_| |_|_|_|	
39. Pains shooting down your legs (61-65)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (66-71)	|_|_|_| |_|_|_| |_|_|_|	
				|0|6| (79-80)
40. Skin sores that do not heal well (6-10)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (11-16)	|_|_|_| |_|_|_| |_|_|_|	
41. Acne or pimples (17-21)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 |_|_|
		LOCATIONS (22-27)	|_|_|_| |_|_|_| |_|_|_|	

Secondary Questions

IF YES ask:

FREQUENCY: About how often do you have this? (IF only one occurrence, go to next condition.)

CURRENT: Have you had this in the past month?

ONSET: In about what year did you first notice this?

LOCATION: What part of the body is affected? (See location codes at end of questionnaire.)

CONDITION	RESPONSE	FREQUENCY	CURRENT	ONSET
42. Skin rash or dermatitis (28-32)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 ___
		LOCATIONS (33-38)	___ ___ ___	
43. Unexplained itching (39-43)	YES 1 NO 2 DK 8	1-TIME 1 1-4/MO 2 1-4/WK 3 5-7/WK 4 DK 8	YES 1 NO 2 DK 8	19 ___
		LOCATIONS (44-49)	___ ___ ___	

44. Frequent abdominal pain (50) — YES 1, NO 2, DK 8

IF YES to frequent abdominal pain:

45. Is the pain relieved by eating food? (51) — YES 1, NO 2, DK 8

46. Is the pain relieved by antacids? (52) — YES 1, NO 2, DK 8

47. Have you had an unexplained weight loss? (53)

YES ___ 1
NO ___ 2
DK ___ 8

IF YES to weight loss:

48. Was this in the past year? (54)

YES ___ 1
NO ___ 2
DK ___ 8

49. What is the most you ever weighed? (55-57)

|_|_|_| pounds

50. In what year? (58-59)

19 |_|_|

|0|7|
(79-80)

Have you ever been told by a doctor that you had any of the following conditions?:

IF YES, ASK: In what year were you first told about this?:

		YES	NO	If YES, enter year
51.	Stomach Ulcer..... (6)	___1	___2	19 |___| (7-8)
52.	Diabetes or sugar in the urine..... (9)	___1	___2	19 |___| (10-11)
53.	Anemia..... (12)	___1	___2	19 |___| (13-14)
54.	Thyroid disease..... (15)	___1	___2	19 |___| (16-17)
55.	High blood pressure..... (18)	___1	___2	19 |___| (19-20)
56.	Bronchitis..... (21)	___1	___2	19 |___| (22-23)
57.	Emphysema..... (24)	___1	___2	19 |___| (25-26)
58.	Bronchiectasis..... (27)	___1	___2	19 |___| (28-29)
59.	Bronchial asthma..... (30)	___1	___2	19 |___| (31-32)
60.	Pulmonary edema..... (33)	___1	___2	19 |___| (34-35)
61.	Tuberculosis..... (36)	___1	___2	19 |___| (37-38)
62.	Pneumonia..... (39)	___1	___2	19 |___| (40-41)
63.	Pleurisy..... (42)	___1	___2	19 |___| (43-44)
64.	Any other chest trouble..... (45)	___1	___2	19 |___| (46-47)

		YES	NO	If YES, enter year
65.	Stroke..... (48)	___1	___2	19 |___| (49-50)
66.	Nerve Injury..... (51)	___1	___2	19 |___| (52-53)
67.	Hepatitis..... (54)	___1	___2	19 |___| (55-56)
68.	Cirrhosis..... (57)	___1	___2	19 |___| (58-59)
69.	Enlarged liver..... (60)	___1	___2	19 |___| (61-62)
70.	Any other liver condition..... (63) If YES, specify: _____	___1	___2	19 |___| (64-65)
				|0|8| (79-80)
71.	Urinary/bladder infection..... (6)	___1	___2	19 |___| (7-8)
72.	Kidney stones..... (9)	___1	___2	19 |___| (10-11)
73.	Any other kidney condition..... (12) If YES, specify: _____	___1	___2	19 |___| (13-14)
74.	Heart attack..... (15)	___1	___2	19 |___| (16-17)
75.	Angina pectoris..... (18)	___1	___2	19 |___| (19-20)
76.	Any other heart condition..... (21) If YES, specify: _____	___1	___2	19 |___| (22-23)
77.	Cancer..... (24) If YES, specify: _____	___1	___2	19 |___| (25-26)

		YES	NO	If YES, enter year
78.	Any other medical condition that..... we have not already mentioned (27)	___1	___2	19 ___ (28-29)

If YES, describe: _____

_____

_____

0 9
(79-80)

79. Have you ever been hospitalized for any illness, injury, or surgery? (6)

Yes ___ 1
No ___ 2

IF YES, for each hospitalization give the reason and the date:

	REASON	MONTH	DAY	YEAR	(LEAVE BLANK)
80.	_____	___ (7-12)	- ___	- ___	___ (13-14)
81.	_____	___ (15-20)	- ___	- ___	___ (21-22)
82.	_____	___ (23-28)	- ___	- ___	___ (29-30)
83.	_____	___ (31-36)	- ___	- ___	___ (37-38)
84.	_____	___ (39-44)	- ___	- ___	___ (45-46)
85.	_____	___ (47-52)	- ___	- ___	___ (53-54)

1 0
(79-80)

Please list any medications you have taken within the past month:

	MEDICATION	REASON	AMOUNT (DOSE, HOW OFTEN)	
86.				(6-7)
87.				(8-9)
88.				(10-11)
89.				(12-13)
90.				(14-15)
91.				(16-17)
92.				(18-19)
93.				(20-21)
94.				(22-23)
95.				(24-25)
96.				(26-27)

1 1
(79-80)

CIGARETTE SMOKING:

97. DO you smoke cigarettes? (6) YES___1 NO___2

98. IF NO: Have you ever smoked cigarettes? (7) YES___1 NO___2

IF NO TO QUESTIONS 97 AND 98, SKIP TO QUESTION 103

99. IF YES: Have you smoked as many as five packs of cigarettes, that is, 100 cigarettes, during your entire life? (8) YES___1 NO___2

IF NO TO QUESTION 99, SKIP TO QUESTION 103

100. How old were you when you started smoking cigarettes? (9-10) |_|_| Years old

101. How many cigarettes a day do (did) you smoke? (Code less than 1 per day as "00") (11-12) |_|_| Per day

102. How old were you when you last gave up smoking? (13-14) (Enter "99" if current smoker.) |_|_| Years old

ALCOHOLIC BEVERAGES:

103. Have you ever drunk as many as 20 alcoholic beverages in your entire life? (15) (IF NO, skip to question 110.) YES___1 NO___2

104. Do you drink alcoholic beverages now? (16) YES___1 NO___2

105. IF NO: How old were you when you gave up drinking? (17-18) |_|_| Years old

106. About how many 12 oz. cans or bottles of beer do/did you usually drink a day? (19-20) |_|_| Beer/day

107. About how many quart bottles of wine do/did you usually drink a week? (21-22) |_|_| Wine/wk.

108. About how many 2 oz. drinks of liquor do/did you usually drink a day? (23-24) |_|_| Liq./day

109. How old were you when you first started drinking? (25-26) |_|_| Years old

REPRODUCTIVE HISTORY:

110. Are you..... (27)

Single ___ 1
Married ___ 2
Divorced ___ 3
Separated ___ 4
Widowed ___ 5

IF SINGLE, skip to question 144.

111. How many times have you been married? (28-29) | | | Times

| 1 | 2 |
(79-80)

If one marriage, fill out page 17 only. Skip pages 18-20.
If two marriages, fill out pages 17-18. Skip pages 19-20.
If three marriages, fill out pages 17-19. Skip page 20.
If four marriages, fill out pages 17-20.

Ask for all wives (husbands):

What are/were the dates of your present or most recent marriage (from, to)?

Did you have (have you had) trouble having a family with your wife (husband) despite a desire to have one?

How many children, born alive, did you have (have you had) with your (present, previous, last previous, etc.) spouse?

How many children, born alive, died within 4 weeks of birth?

How many miscarriages or spontaneous abortions did your wife (did you) have?

How many stillbirths did your wife (did you) have?

How many children, born alive, were born with a birth defect?

QUESTION	PRESENT OR MOST RECENT SPOUSE
112.Dates of Marriage	From ____ ____ Month (6-7) Year (8-9)
113.	To ____ ____ Month (10-11) Year (12-13)
	(Above, enter today's date for present marriage.)
114.Problems having a family.	YES__1 NO__2 (14)
115.Children born alive.	____ # of children (15-16)
116.Children dead within 4 wks.	____ # of children (17-18)
117.No. of miscarriages	____ # of miscarriages (19-20)
118.No. of stillbirths	____ # of stillbirths (21-22)
119.No. of children with birth defects	___ # of children (23-24)

1 3 (79-80)

Ask for all wives (husbands):

What were the dates of your previous marriage (from, to)?

Did you have trouble having a family with your wife (husband) despite a desire to have one?

How many children, born alive, did you have with your ex-spouse?

How many children, born alive, died within 4 weeks of birth?

How many miscarriages or spontaneous abortions did your ex-wife have?

How many stillbirths did your wife (did you) have?

How many children, born alive, were born with a birth defect?

QUESTION	PREVIOUS SPOUSE
120.Dates of Marriage	From |___| |___| Month (6-7) Year (8-9)
121.	To |___| |___| Month (10-11) Year (12-13)
122.Problems having a family.	YES___1 NO___2 (14)
123.Children born alive.	|___| # of children (15-16)
124.Children dead within 4 wks.	|___| # of children (17-18)
125.No. of miscarriages	|___| # of miscarriages (19-20)
126.No. of stillbirths	|___| # of stillbirths (21-22)
127.No. of children with birth defects	|___| # of children (23-24)

\|1\|4\| (79-80)

Ask for all wives (husbands):

What were the dates of your next previous marriage (from, to)?

Did you have trouble having a family with your ex-wife despite a desire to have one?

How many children, born alive, did you have with your ex-spouse?

How many children, born alive, died within 4 weeks of birth?

How many miscarriages or spontaneous abortions did your ex-wife have?

How many stillbirths did your wife have?

How many children, born alive, were born with a birth defect?

QUESTION	NEXT PREVIOUS SPOUSE
128. Dates of Marriage	From ____ Month (6-7) ____ Year (8-9)
129.	To ____ Month (10-11) ____ Year (12-13)
130. Problems having a family.	YES__1 NO__2 (14)
131. Children born alive.	____ # of children (15-16)
132. Children dead within 4 wks.	____ # of children (17-18)
133. No. of miscarriages	____ # of miscarriages (19-20)
134. No. of stillbirths	____ # of stillbirths (21-22)
135. No. of children with birth defects	___ # of children (23-24)

[1|5]
(79-80)

Ask for all wives (husbands):

What were the dates of your next previous (from, to)?

Did you have trouble having a family with this ex-wife despite a desire to have one?

How many children, born alive, did you have with your this ex-wife?

How many children, born alive, died within 4 weeks of birth?

How many miscarriages or spontaneous abortions did this ex-wife have?

How many stillbirths did this ex-wife have?

How many children, born alive, were born with a birth defect?

QUESTION	NEXT PREVIOUS SPOUSE
136. Dates of Marriage	From ____ ____ (Month (6-7), Year (8-9))
137.	To ____ ____ (Month (10-11), Year (12-13))
138. Problems having a family.	YES 1 / NO 2 (14)
139. Children born alive.	___ # of children (15-16)
140. Children dead within 4 wks.	___ # of children (17-18)
141. No. of miscarriages	___ # of miscarriages (19-20)
142. No. of stillbirths	___ # of stillbirths (21-22)
143. No. of children with birth defects	___ # of children (23-24)

16 (79-80)

OTHER:

144. Have you ever opened capacitors from Lemon Lane, Bennett's Quarry, or Neal's Landfill to remove copper from them?

YES 1
NO 2
(6)

145. IF YES: About how many transformers or capacitors have you opened up from those sites, in your lifetime?

| | | | |
(7-9)

Have you ever swum in the following areas:	IF YES:	How many times?
146. Bennett's (Packinghouse) quarries	YES 1 NO 2 (10)	(11-13)
147. Richland Creek	YES 1 NO 2 (14)	(15-17)
148. Salt Creek	YES 1 NO 2 (18)	(19-21)
149. Clear Creek	YES 1 NO 2 (22)	(23-25)
150. East Fork White River	YES 1 NO 2 (26)	(27-29)
151. Stout's Creek	YES 1 NO 2 (30)	(31-33)
152. Bean Blossom Creek	YES 1 NO 2 (34)	(35-37)

1 7
(79-80)

Within the past year, have you <u>eaten</u> fish caught in any of the following areas?

	IF YES:	How many times?
153. Bennett's (Packinghouse) quarries	YES 1 NO 2 (10)	(11-13)
154. Richland Creek	YES 1 NO 2 (14)	(15-17)
155. Salt Creek	YES 1 NO 2 (18)	(19-21)
156. Clear Creek	YES 1 NO 2 (22)	(23-25)
157. East Fork White River	YES 1 NO 2 (26)	(27-29)
158. Stout's Creek	YES 1 NO 2 (30)	(31-33)
159. Bean Blossom Creek	YES 1 NO 2 (34)	(35-37)

160. Within the past year, have you spread sewage sludge around your property? YES 1 NO 2 (38)

1 8
(79-80)

OTHER:

144. Have you ever opened capacitors from Lemon Lane, Bennett's Quarry, or Neal's Landfill to remove copper from them?

YES___1
NO___2
(6)

145. IF YES: About how many transformers or capacitors have you opened up from those sites, in your lifetime?

|___|___|___|
(7-9)

Have you ever swum in the following areas:	IF YES:	How many times?
146. Bennett's (Packinghouse) quarries	YES___1 NO___2 (10)	|___|___|___| (11-13)
147. Richland Creek	YES___1 NO___2 (14)	|___|___|___| (15-17)
148. Salt Creek	YES___1 NO___2 (18)	|___|___|___| (19-21)
149. Clear Creek	YES___1 NO___2 (22)	|___|___|___| (23-25)
150. East Fork White River	YES___1 NO___2 (26)	|___|___|___| (27-29)
151. Stout's Creek	YES___1 NO___2 (30)	|___|___|___| (31-33)
152. Bean Blossom Creek	YES___1 NO___2 (34)	|___|___|___| (35-37)

1	7
(79-80)	

LOCATION CODES

01 = Face

02 = Neck

03 = Right arm

04 = Left arm

05 = Both arms

06 = Right hand

07 = Left hand

08 = Both hands

09 = Chest

10 = Back

11 = Abdomen

12 = Right leg

13 = Left leg

14 = Both legs

WESTINGHOUSE
BLOOMINGTON, INDIANA
HETA 84-339

BLOOD PRESSURE RECORDING FORM

SUBJECT ID: |_|_|_|_|_|_| (1-5)

DATE (Month/Day/Year): |_|_| / |_|_| / 19 |_|_| (6-11)

TIME (Military Clock): |_|_|_|_| (12-15)

1ST BLOOD MEASUREMENT

1. ASK PARTICIPANT THE FOLLOWING QUESTIONS BEFORE THE FIRST BLOOD PRESSURE MEASUREMENT:

   a. IS YOUR BLADDER EMPTY? 1__Yes 2__No (16)

   b. HAVE AT LEAST 30 MINUTES PASSED SINCE YOU LAST ATE, EXERCISED OR SMOKED? 1__Yes 2__No (17)

   c. WHEN DID YOU LAST TAKE MEDICATION FOR HIGH BLOOD PRESSURE? (Code all 9's if never took)

      (1) Date (Month/Day/Year): |_|_| / |_|_| / 19 |_|_| (18-23)

      (2) Time (Military Clock): |_|_|_|_| (24-27)

2. FREE UP RIGHT SLEEVE. SIT DOWN AT PROPER CHAIR HEIGHT AND MEASURE THE ACROMION-OLECRANON LENGTH (centimeters): |_|_|.|_| (28-31)

3. MARK THE LATERAL MIDPOINT WITH THE ARM HANGING DOWN. MEASURE THE MID-UPPER-ARM CIRCUMFERENCE (centimeters): |_|_|.|_| (32-35)

4. INDICATE THE CUFF SIZE CHOSEN:

1___Adult (23" x 12") 2___Large adult (36" x 18") (36)

5. WRAP CUFF AND POSITION ARM.

6. COUNT PULSE BEATS FOR 30 SECONDS: |_|_|_| (37-38)

7. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_|_| (39-41)

8. DEFLATE THE CUFF.

9. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

10. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE FIRST MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (42-50)
(Sys) (Dia 4) (Dia 5)

11. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_|_| (51-52)

12. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (53-61)
(Sys) (Dia 4) (Dia 5)

CARD |2|0| (79-80)

NOTE: Repeat the same procedure (steps 7-12) for each of the 2 additional measurements and record the results on the following pages.

2ND BLOOD MEASUREMENT

13. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_| (6-8)

14. DEFLATE THE CUFF.

15. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

16. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE SECOND MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (9-17)
(Sys) (Dia 4) (Dia 5)

17. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_| (18-19)

18. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (20-28)
(Sys) (Dia 4) (Dia 5)

3RD BLOOD MEASUREMENT

19. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_| (29-31)

20. DEFLATE THE CUFF.

21. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

22. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE THIRD MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (32-40)

23. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_| (41-42)

24. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (43-51)

CARD |2|1| (79-80)

WESTINGHOUSE
BLOOMINGTON, INDIANA
HETA 84-339

BLOOD PRESSURE RECORDING FORM

SUBJECT ID: |_|_|_|_|_|_| (1-5)

DATE (Month/Day/Year): |_|_| / |_|_| / 19 |_|_| (6-11)

TIME (Military Clock): |_|_|_|_| (12-15)

1ST BLOOD MEASUREMENT

1. ASK PARTICIPANT THE FOLLOWING QUESTIONS BEFORE THE FIRST BLOOD PRESSURE MEASUREMENT:

   a. IS YOUR BLADDER EMPTY? 1__Yes 2__No (16)

   b. HAVE AT LEAST 30 MINUTES PASSED SINCE YOU LAST ATE, EXERCISED OR SMOKED? 1__Yes 2__No (17)

   c. WHEN DID YOU LAST TAKE MEDICATION FOR HIGH BLOOD PRESSURE? (Code all 9's if never took)

      (1) Date (Month/Day/Year): |_|_| / |_|_| / 19 |_|_| (18-23)

      (2) Time (Military Clock): |_|_|_|_| (24-27)

2. FREE UP RIGHT SLEEVE. SIT DOWN AT PROPER CHAIR HEIGHT AND MEASURE THE ACROMION-OLECRANON LENGTH (centimeters): |_|_|.|_| (28-31)

3. MARK THE LATERAL MIDPOINT WITH THE ARM HANGING DOWN. MEASURE THE MID-UPPER-ARM CIRCUMFERENCE (centimeters): |_|_|.|_| (32-35)

4. INDICATE THE CUFF SIZE CHOSEN:

1___Adult (23" x 12") 2___Large adult (36" x 18") (36)

5. WRAP CUFF AND POSITION ARM.

6. COUNT PULSE BEATS FOR 30 SECONDS: |_|_|_| (37-38)

7. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_|_| (39-41)

8. DEFLATE THE CUFF.

9. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

10. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE FIRST MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (42-50)
(Sys) (Dia 4) (Dia 5)

11. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_|_| (51-52)

12. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (53-61)
(Sys) (Dia 4) (Dia 5)

CARD |2|0| (79-80)

NOTE: Repeat the same procedure (steps 7-12) for each of the 2 additional measurements and record the results on the following pages.

2ND BLOOD MEASUREMENT

13. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_| (6-8)

14. DEFLATE THE CUFF.

15. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

16. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE SECOND MEASUREMENT:

|_|_|_| (Sys) / |_|_|_| (Dia 4) / |_|_|_| (Dia 5) (9-17)

17. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_| (18-19)

18. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| (Sys) / |_|_|_| (Dia 4) / |_|_|_| (Dia 5) (20-28)

3RD BLOOD MEASUREMENT

19. SET R-Z VALVE TO "OPEN", THEN TO "CLOSE" AND INFLATE THE CUFF SMOOTHLY. RECORD THE APPARENT PULSE OBLITERATION PRESSURE (mmHg): |_|_|_| (29-31)

20. DEFLATE THE CUFF.

21. SET THE R-Z VALVE TO "OPEN" AND ROTATE THE WHEEL RIM.

22. INFLATE TO 30 mm ABOVE THE APPARENT PULSE OBLITERATION PRESSURE OR AT LEAST TO 160 mm. HOLD THE INFLATION LEVEL FOR 5 SECONDS. THEN SET THE R-Z VALVE TO "CLOSE" AND BEGIN DEFLATING THE CUFF AT 2 mm PER SECOND. RECORD THE BLOOD PRESSURE FOR THE THIRD MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (32-40)

23. DISCONNECT THE CUFF. ENTER THE ZERO-CORRECTION: |_|_|_| (41-42)

24. ENTER THE CORRECTED BLOOD PRESSURE MEASUREMENT:

|_|_|_| / |_|_|_| / |_|_|_| (43-51)

CARD |2|1| (79-80)

APPENDIX II

In only one participant in the follow-up study did serum L-PCB and H-PCB levels appear to have increased in 1985 by more than 100% of the recorded 1977 value. This was an individual whose serum L-PCB went from 1 ug/l in 1977 to 51 ug/l in 1985, and whose serum H-PCB went from 1 ug/l in 1977 to 29 ug/l in 1985, representing apparent increases of 5004% and 2822%, respectively. The 1985 determination was reexamined, and it was concluded that it was not in error. The 1977 value was reexamined. This, it turned out, was a determination that had been reported as below the level of detection at the time, which was 1 ug/l. The 1977 value had therefore been reported as 1 ug/l. This was the only PCB level reported to us as undetectable.

In unpublished work performed in 1981, one of us (ABS) had attempted to model serum PCB levels measured in 1977 as simple mathematical functions of times spent assigned to various departments within the plant, based upon information extracted from the plant employment records. A number of mathematical models were explored, and the fit of the prediction model to the data examined by looking at the standardized residual values for each observation (or participant), that is, the difference between the observed and predicted values, divided by the root mean-squared error of the model. The observation with the most extreme standardized residual was for the individual whose serum L-PCB and H-PCB levels were 1 ug/l (and actually had been reported as non-detectable). The absolute residual value exceeded three root mean-squared-errors of the prediction equation.

For the sole individual whose serum L-PCB and H-PCB levels between 1977 and 1985 appeared to increase by more than 100%:

(1) no error in the 1985 determination could be identified by the laboratory,

(2) his were the only serum PCB data reported as non-detectable in 1977, and

(3) his serum PCB data in 1977 had the greatest outlying values relative to predicted when included in mathematical models with all other data;

strongly suggest that the 1977 serum PCB data for this individual were in error. Since we cannot know where on the serum PCB distribution his 1977 data lie, but suspect that it is likely to have been greater than the (lower) 15th percentile, we conclude that this individual's data from the 1985 study should be excluded from further analysis.