REVIEW OF:

Schwartz Memorandum concerning:

The Relationship Between Blood Lead Levels and Its Cardiovascular Risk Implications

by

James L. Pirkle, M.D.,Ph.D.
Joel Schwartz, Ph.D.
J. Richard Landis, Ph.D.
William R. Harlan, M.D.

SUBMITTED BY:

Lewis H. Kuller, M.D., Dr.P.H.
Center for Environmental Epidemiology
University of Pittsburgh
Pittsburgh, Pa. 15261

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REVIEW OF: Joel Schwartz Memorandum

BY: Lewis Kuller, M.D., Dr. P.H.

This report is based on an apparent association between the blood lead levels and blood pressure, both systolic and diastolic, found by the merging of the HANES blood pressure data and the measurement of blood lead in this same population. The data is restricted to white men ages 40-59. Apparently the positive relationship between blood lead levels and blood pressure was not noted for women. No data or discussion is provided of the relationship between blood lead levels and blood pressure in blacks nor in other age groups but apparently the relationship was consistent for men. This point however, must be clarified.

The authors presume that this relationship between blood lead and blood pressure is causal and then attempt to estimate the economic impact of reducing the lead in gasoline and its effect on blood pressure. There are three essentially critical questions in this particular problem. 1) Is the relationship between the blood lead levels and blood pressure causal or a confounder that is related to some other variable which is correlated with both the blood lead and the blood pressure? 2) Given the fact that higher blood lead levels are associated with higher blood pressures and that this may be causal, what is the attributable risk of increased blood pressure due to elevated blood lead levels as compared to other risk factors for blood pressure especially obesity, alcohol intake, sodium potassium and calcium intake, and perhaps certain measures of environmental stress; and 3) What is the impact of reducing the blood lead levels on the blood pressure; what will this do to the risks of heart disease and stroke and other hypertensive related diseases; and what is the economic and health related impacts of such a change?

The authors suggest that prior studies are consistent with the observation that blood lead levels are related to blood pressure. On the contrary, however, the literature is quite confusing. The two papers on the studies in rats that they provide suggest that there was an increase in blood pressure when the rats were fed 100 ppm of lead in the water but no change when the dose of lead was increased to 500 ppm, nor was there any relationship between increased lead in the water and blood pressure among female rats. The experimental study in rats comparing the effects of various alpha and beta agonists on blood pressure in lead-fed and non-lead-fed rats is somewhat confounded by the fact that the rats fed the lead had higher blood pressures than the control rats. A better study certainly would have compared rats with similar blood pressures, that is increased blood pressure due to lead as composed to increasing in the blood pressure due to some other factor such as sodium and then compared the effects of alpha and beta agonists.

The study presented - of the relationship between hard and soft water, lead leached from the lead pipes in the soft water areas, blood lead levels and blood pressure in England is well-known. However, it is unclear in that study whether the relationship is with the soft water, which is correlated with the lead and obviously, with other factors, especially calcium and magnesium or other trace metals, or to the effects of lead itself. It would be important to evaluate the data relating soft and hard water in the U.S. from the same HANES data set to blood pressure. Studies of blood pressure levels in lead workers are very inconsistent.

The data from the HANES study relating blood pressure and blood lead levels must be viewed very cautiously. First, only two regression analyses are provided. In both of these regression analyses, it is clear that there are many factors which are apparently independently related to blood pressures. These include such factors as vitamin C, potassium, obesity, riboflavin, albumin in the blood, level of hemoglobin,

calcium as reported in other studies, amount of oleic acid which is probably a measure of either total fat intake or mono-unsaturates vs. poly-unsaturate intake of fat. Other investigators have apparently demonstrated that high intake of polyunsaturated fats are associated with lower levels of blood pressure. The complex inter-relationship of these environmental and dietary factors to blood pressures as well as the socio-economic correlates of blood pressure such as race, education level, and geographic area of the country, certain other markers of social class, the possible relationship to environmental stress and the known genetic correlates, all make one suspicious of any single relationship with blood pressure.

Before this analysis can be accepted as even showing a potential causal relationship, further information is required. First, a tabulation showing the relationship between blood lead levels and blood pressure is missing. Clearly this analysis must have been done. It is important to look at this independent of the other variables to get some idea about the relationship of blood lead levels to blood pressure within each age, race, and sex group. This should be the primary available analysis and without such documentation, the further data set is of little value. This could be easily done using a simple analysis showing various levels of blood lead on one axis and levels of blood pressure on the other. It needs to be done, however, specifically for each age, race and sex group. Second, it would be extremely important to show similar types of both univariate and perhaps bivariate relationships between the blood lead levels and a variety of other risk factors. The fact that lead is an independent predictor of blood pressure in a regression analysis does not exclude the possibility of a relationship between lead and other environmental or dietary variables which may be related to blood pressure. It may well be that lead is serving as a surrogate for some marker related to dietary intake such as total calories, amount of sodium in the diet, fat, etc. All of these variables are interrelated with lower socio-economic class in which there

is a higher prevalence of obesity, greater caloric intake, apparently greater sodium intake, and less potassium, and probably less calcium intake. Lead intake or the association with environmental exposures to lead in the low socio-economic class may be serving as a surrogate marker for this constellation of risk factors. Many of these factors are poorly measured in the HANES analysis and it is possible that the blood lead levels are serving as a better surrogate marker than, let us say, sodium intake, some marker of obesity, some index of education, etc. On face value, the data is interesting but is far from causal and should not be used in its present format for any inference that the current blood lead levels are an important contributor to the distribution of blood pressure in the U.S.

The second question of importance is, given the possibility that there is some relationship to blood lead levels and blood pressure, what is the specific contribution of blood lead levels to the distribution of blood pressure in the population? As noted, there are numerous risk factors related to blood pressure. The presumption that changing the blood lead levels in any way will have a major impact on blood pressure is unacceptable.

The statistical analysis as presented to date is essentially a fairy tale unsupported or undocumented by any biologic or meaningful clinical observations. The absence of a relationship between the lead levels and blood pressure in the women is very worrisome. The authors pawn this off on the basis of the results in the rat experiments. There is, as far as I can tell, little or no evidence for any other environmental variable or dietary factor which preferentially causes an increase in blood pressure in men as compared to women, nor is there any evidence that the level of the blood pressure is a predictor of disease in men but not in women. Similarly, most of the risk factors that have been studied related to blood pressure appear to act in both blacks as well as whites. The strength of the association may be related to sex,

as well as genetic factors and race but certainly, absence of an association in women is quite worrisome. There is no documentation of the actual change in blood pressure with the change in units of blood lead levels and how these might be related to the distribution of blood pressure in the general population. There seems to be an inherent assumption that the blood pressures in the U.S. are declining, that is, the percentage with blood pressures greater than 90, and that this may have something to do with the changes in lead in gasoline. Quite on the contrary, the changes of blood pressure distribution in the U.S. appears to be primarily due to the treatment of hypertension, and the decline in blood pressures greater than 90 are because hypertensives are currently being treated with a variety of different drugs to lower their blood pressure. It is not completely clear from their analysis that those individuals on drug therapy or prior drug therapy for hypertension have been excluded, although I presume this is the case. No data is further provided on the consistency of the relationship in different geographic areas or in relationship to certain other social class markers which exist in the HANES set. They do, however, suggest that they have looked at multiple factors and that these do not account for the association between lead and blood pressure. A further question, of course, is whether the apparent elevated blood lead levels in people with increasing blood pressure as suggested, in the previous British study in Lancet are due to soft water and the leaching of lead from pipes or from the contribution of lead from gasoline. It is also important to consider the contribution of the lead from the food chain especially because of the possible association with caloric intake, obesity, and increasing blood pressure. Finally, I think it is important to look at the relationship between alcohol intake and cigarette smoking and blood lead levels independent of blood pressure to see whether these factors are in any way related to the blood lead levels.

There was also suggested evidence that the elevated blood lead levels are not due to changes in renal function in relationship to the blood pressure levels. The papers that have suggested this have looked at the serum creatinine levels and shown that the levels are not related to the blood lead levels. This of course, is a very crude estimate of small changes in renal function associated with the relatively modest increases in blood lead levels. This again, should be looked at carefully by some measurements of the lead excretion in the urine in relationship to the blood lead levels among the hypertensive and normotensive individuals.

Finally, the question of whether the relationship between the blood lead levels and blood pressure levels have an impact on disease is, of course, of considerable importance as well as the economic impact. The authors have made exaggerated claims about the efficacy of reducing lead in gasoline in relationship to reduction of blood pressure and subsequent incidence and mortality from stroke and coronary artery disease. There is certainly strong evidence of a relationship between the level of blood pressure and the risk of myocardial infarction and sudden death, and other manifestations of coronary artery disease. The evidence, however, that lowering of the blood pressure even by reducing lead in the blood would also have a direct effect on reduction in risk of heart attack is not substantiated. This will depend, of course, on two factors: 1) The actual contribution to the blood pressure distribution by the increasing lead in the blood as compared to the other risk factors and, 2) the impact of reduction of blood pressure on the risk of disease. The answer to the first question, as far as I am concerned, is unknown. The answer to the second question is equivocal. Clinical trials generally have not demonstrated a substantial reduction in CHD mortality or incidence in association with reduction of blood pressure through drug therapy, at least among hypertensives. On the other hand, a downward shift of the

entire distribution of the blood pressure curves in the U.S. might have an effect and in fact, may have been a major factor in the reduction in CHD mortality in the U.S.

The situation with stroke is quite different. Clearly blood pressure is the major risk factor for stroke and any factor which will reduce the blood pressure levels would probably have an impact on stroke morbidity and mortality. Thus, even a modest change in the blood pressure in relationship to a reduction in blood lead levels could have an important effect on stroke mortality and morbidity. The stroke mortality and morbidity rates in the U.S. have been declining substantially, over 50% since 1960-68. Much of this decline is related to the treatment of hypertension but a substantial part of it occurred prior to the introduction of anti-hypertensive drug therapy and the impact of various environmental factors including blood lead should be considered. It would be quite perplexing however, to note that the decline in stroke mortality in the U.S. was probably occurring at a time when lead in gasoline was increasing because of the apparent greater use of gasoline and greater numbers of cars. The decline in stroke mortality being very substantial even from the end of the second world war through the 1968. Thus, any attempt to correlate changes in blood lead levels now and stroke mortality would clearly demonstrate a rather peculiar relationship, that is, a declining mortality in the fact of increasing lead in the U.S. and then a subsequent decrease in stroke mortality and apparent decrease in lead in the blood. The bottom line would be that there is no relationship between the amount of lead being used, probably the blood lead levels and either the changes in blood pressure in the U.S. or the stroke mortality.

Finally, it should be noted that this observation should not be dismissed as an interesting statistical artifact. Rather, the observation may have important implications. The problem is that the data as provided is both weak and far too premature for the exaggerated claims provided in the subsequent economic and health related analysis. Thus, the major recommendation at this time should be for a series of

carefully coordinated studies and evaluations to really look at the relationship between lead, blood pressure, and perhaps, cardiovascular disease and stroke, in order to either accept or disprove the causal associations suggested by this paper. Use of this data alone for policy decisions could be fraught with a substantial scientific incredulity if not shear folly. Studies that should be done include a much more detailed analysis of the data provided as suggested in the earlier discussion. Careful analysis of other data sets which include measurements of both blood pressure or blood lead or the addition of blood lead measurements to several on-going studies of blood pressure. Measurements of the effects of the blood lead levels and other determinants of hypertension especially obesity, of sodium, potassium and calcium intake should be done. Further experimental studies, perhaps using a different animal model than the rat to determine the relationship between blood lead and/or lead exposure and blood pressure, and more careful analysis of the HANES water data in relationship to hard and soft water and blood pressure levels are also needed. If the hypothesis is correct, then clearly individuals who have had a recent stroke, heart attack, etc., especially with hypertension, should have higher blood lead levels than controls or individuals with a heart attack without a prior history of hypertension. It should be reasonably simple to do these types of case-control studies and they would be of considerable interest and importance.

The economic analysis has some flaws in it but in general is quite interesting, although, again, based on some very specious biological and clinical associations.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
POLICY, PLANNING AND EVALUATION

MEMORANDUM

SUBJECT: Blood Lead and Blood Pressure

FROM: Joel Schwartz
Office of Policy Analysis

TO: Docket EN-84-05

Thus far, all of EPA's analysis of the health benefits related to the proposed gasoline-lead rulemaking has been for children. Recently, staff members from the Centers for Disease Control, EPA, and the University of Michigan completed and submitted for publication a paper discussing the relationship between blood pressure and blood lead for adults, derived from an analysis of the Second National Health and Nutrition Evaluation Survey (NHANES II).

We found that blood lead levels were a statistically significant predictor of blood pressure in adult males. This relationship held not merely when blood lead was evaluated in a regression with all known factors[1] that have previously been established as correlated with blood pressure; it also held when tested against 89 additional variables representing linear and non-linear functions of every dietary and serologic variable on the NHANES II survey. Recreational exercise, work-related exercise, blood pressure medication, and recent weight loss were

also considered, and did not effect the size or strength of the relationship. To ensure that the relationship was robust, we included all the variables that, while not significant at the $p = .05$ level, were significant at the $p = .15$ level, and considered every possible combination of those variables. We added all 255 of those combinations to the variables that were statistically significant and performed a regression on each one. The range of variation of the coefficient of the log of blood lead varied by only $\pm$ 10% from the value we obtained when we included only significant variables, and the highest p-value for lead was still less than .01. We also forced age and age-squared into our regressions, although in the age group we were analyzing (40-59 year-old men) blood pressure is independent of age. Since blood lead levels are correlated with age, this approach reduced both the coefficient and significance level of lead. I have attached to this memo several tables showing the regression coefficients we obtained.

Several other studies have suggested a relationship between blood pressure and blood lead levels in humans,[2] and experiments on rats confirmed that moderate doses of lead can increase blood pressure and that the effect is restricted to males.[3] The rat experiments also suggest a pathway: lead interfering with nerve signals to the muscles around the arteries that control blood pressure.[4] Some of these studies are attached.

All of this suggests that the relationship is causal. Moreover, specific analysis to determine whether there is a lower threshold below which lead has no effect on blood pressure showed that the data was best fit with a threshold of zero.

We have examined the public health implications of this relationship using several established correlations between blood pressure and the risk of heart attacks, strokes, and deaths, based upon long-term cardiovascular epidemiological studies. The classic study, which was important in establishing cholesterol as a major factor in the risk of heart disease, was the Framingham study.[5] Extensive analyses of these data have indicated the probabilities of such coronary events as a function of several variables, including blood pressure. In the 1970's, the National Institutes of Health funded the Pooling Project,[6] which combined the Framingham data with data from five other long-term studies to improve the accuracy of the risk coefficients. The Pooling Project analyzed the occurrence of serious heart attacks (myocardial infarctions) in white men who entered the study at ages 40-59 and who were followed for at least 10 years. The stroke regressions, also based on a 10-year follow-up, were taken from the Framingham study, as were the estimates of deaths. Because we wanted to predict health outcomes, we also restricted our blood pressure regressions to data on white men aged 40-59. We have used our regression of the relationship between blood lead and blood pressure to predict changes in blood pressure due to the proposed EPA rule, and we have used the Framingham

and Pooling Project coefficients of the risks of heart attacks, strokes, and deaths as a function of blood pressure to predict the health outcomes.

To produce estimates for all 40-59 year old white males, the individual risk of each person sampled in the NHANES was summed and then averaged. Since the sampled individuals represent the U.S. population for their specific age-race-sex category, their average risk represents the average risk for all 40-59 year old white men. Because blood lead levels have dropped from the NHANES period until now, we corrected for that change and then evaluated the effects of EPA's proposed rule. Only white men were examined because there were too few blacks in the Framingham study, and their risk might be different from whites. However, we did predict the change in the number of males with high blood pressure for both races because the based data we used (the NHANES) has adequate information on both blacks and whites.

The fact that gasoline lead levels would slowly decline even without new EPA actions created a slight complication. Because gasoline lead levels fall over time in both our base case and the low-lead case, the difference in blood lead levels resulting from the proposed rule will change over time. Therefore, we recalculated the risk estimates for each year, assessing the change in blood lead levels for that year due to the proposed rule.

The heart attack regressions predict the occurrence of myocardial infarctions (MI's) in the next 10 years, given current blood pressure and age. Lacking contrary information, we have

assumed MI's are uniformly distributed over time, i.e., one-tenth of the heart attacks would be avoided each year of the 10-year predicted period. We applied the same technique to strokes and deaths. The population at risk was adjusted for the increases in the U.S. population of white males aged 40-59, and the regression predicting deaths for ages 40-54 was extended to 40-59 for data comparability and uniformity. Because the death rate actually increases with age, this is a conservative estimate. Predictions of the change in the number of males aged 40-59 with hypertension include both races, since again in this case we used the NHANES data.

Based on this approach, the estimates for each year were computed and are shown on Table 1, for EPA's proposed rule* (0.10 grams per leaded gallon (gplg) in 1986), and for the alternative rule** discussed in the preamble to the proposed rule (0.50 gplg on July 1, 1985; 0.30 gplg on January 1, 1986; 0.20 gplg on January 1, 1987; and 0.10 gplg on January 1, 1988).

Table 1

Estimated Reduction in the Number of Heart Attacks, Strokes, and Deaths Due to Lead Phase down
(While Males Aged 40-59)

Heart Attacks

Year	1985	1986	1987	1988	1989	1990	1991	1992
*PROPOSED RULE	0	5348	4821	4584	4241	3899	3928	3821
**ALTERNATIVE	2719	3758	4095	4584	4241	3899	3928	3821

Strokes

*PROPOSED RULE	0	1737	1564	1487	1375	1263	1271	1236
**ALTERNATIVE	887	1256	1331	1487	1375	1263	1271	1236

Deaths

*PROPOSED RULE	0	5904	5322	4963	4680	4303	4302	4333
**ALTERNATIVE	2353	3708	3933	4963	4680	4303	4302	4333

Estimated Reduction in the Number of Males Aged 40-59 with High Blood Pressure

Year	1985	1986	1987	1988
*PROPOSED RULE	0	1,681,000	1,499,000	1,100,000
**ALTERNATIVE	896,000	1,244,000	1,290,000	1,100,000

Year	1989	1990	1991	1992
*PROPOSED RULE	1,300,000	1,190,000	1,185,000	1,150,000
**ALTERNATIVE	1,300,000	1,190,000	1,185,000	1,150,000

Table 2 presents our estimates of the monetized benefits of avoiding these heart attacks, strokes, deaths, and cases of hypertension. The methodology used to derive the estimates follows the table. For convenience, deaths from heart attacks and strokes are valued under the heart attacks and stroke categories. The remaining deaths are valued separately.

Table 2

Benefits of Avoiding Heart Attacks, Deaths, Strokes, and Hypertension Due to EPA's Proposed Gasoline Lead Regulations* (millions of 1983 dollars)

Benefits of Avoided Heart Attacks

Year	1985	1986	1987	1988	1989	1990	1991	1992
*Proposed Rule	0	2979	2685	2553	2362	2171	2188	2128
**Alternative	1514	2149	2281	2553	2362	2171	2188	2128

Benefits of Avoided Deaths

*Proposed Rule	0	2848	2568	2371	2283	2099	2060	2152
**Alternative	797	1502	1593	2371	2283	2099	2060	2152

Benefits of Avoided Strokes

*Proposed Rule	0	782	704	699	619	568	572	556
**Alternative	399	565	599	699	619	568	572	556

Benefits of Avoided Hypertension

*Proposed Rule	0	370	330	237	286	262	261	253
**Alternative	197	273	284	237	286	262	261	253

Total Benefits

Year	1985	1986	1987	1988	1989	1990	1991	1992
*Proposed Rule	0	6,979	6,287	5,860	5,550	5,100	5,081	5,089
**Alternative	2,907	4,489	4,757	5,860	5,550	5,100	5,081	5,089

*The proposed rule would reduce the lead content of gasoline to 0.10 grams per leaded gallon (gplg).

**The alternative rule would reduce lead to 0.5 gplg in July 1985, 0.30 in January 1986, 0.20 in January 1987, and 0.10 in January 1988.

COST OF CORONARY DISEASE

Coronary heart disease is not merely a major cause of mortality and morbidity in the United States; it accounts for a significant fraction of total U.S. medical expenditures (now approximately 10 percent of the Gross National Product) and results in substantial productivity loss due to reduced participation in the labor force of people in their peak productive years. We have estimated the benefits of avoiding the mortality, medical expenses, and reduced labor force participation that would result from the predicted decrease in coronary events due to reductions in blood lead levels resulting from EPA's proposed rule.

To estimate the value of avoiding such losses, we have generally followed Hartunian et al. (1981),[7] who recently completed a detailed analysis of the costs of various diseases.

Medical Costs

Briefly, Hartunian divided coronary heart disease into five subcategories: sudden death, fatal myocardial infarction (fatal MI), non-fatal myocardial infarction (non-fatal MI), coronary insufficiency, and angina pectoris uncomplicated.

For each category and each age group, Hartunian et al. obtained data on the type of medical services needed (e.g. ambulance, coronary intensive care unit, etc.), the fraction of cases using each service, and the costs in 1975 dollars. They also determined the annualized recurrence and follow-up costs, by

age, for each condition. These were then present-valued (using a 6 percent real discount rate) to the time of initial occurrence to estimate the cost, in current dollars, of each new case. Hartunian also obtained data indicating the probability distribution of cases among the four categories. We are concerned only with sudden death, fatal MI, and non-fatal MI. Approximately 77.5 percent of these events are MI's, of which 2.4 percent are fatal for males under 55, and 7.7 percent are fatal for males over 55. The rest of the events are sudden deaths.[8] The present value of the direct medical costs for males aged 40-59, in 1975 dollars, was $96 for sudden death and $7,075 for all MI's, including fatal MI's.

We have done three things to adjust these costs to 1983 dollars. First, we inflated them to 1983 costs. Since most of the costs were hospital-related, with the rest principally being physicians' fees, we inflated Hartunian's costs by a weighted average of 80 percent of the percent change in the Consumer Price Index for hospital rooms and 20 percent of the change in the Consumer Price Index for physicians' expenses. Since approximately 90 percent of Hartunian's MI costs were hospital-related, not physicians' fees, this approach is conservative.

Second, cost indices only account for the increase in cost of the same procedure, in this case principally the initial hospitalization for a heart attack; it does not provide the cost of new or different procedures. Since 1975, however, the fraction of people suffering coronary heart disease who subsequently undergo

coronary bypass operations has substantially increased. The number of bypass operations tripled in seven years, from 57,000 in 1975 to 170,000 in 1982,[9] while the number of cases of coronary heart disease has remained relatively constant. Based on Hartunian's data, in 1975, 7.1 percent of MI cases had subsequent bypass operations. Assuming that they shared proportionately in the tripling of the bypass-operation rate, we estimated that an additional 14 percent of MI's now result in a bypass operation. Hartunian estimated the cost of bypass operations at $6,700 in 1975 dollars, or $16,800 in 1983 dollars. Adding 14 percent of this cost to the other direct costs yields an estimate of the total direct costs in 1983 dollars: $20,100 for an MI and $240 for sudden death.

Third, Hartunian only used a 6 percent real discount rate to present value the future year costs, whereas OMB requires a 10 percent discount rate. Fortunately, Hartunian performed sensitivity calculations for other discount rates, including 10 percent. Using this adjustment, the costs per case are $18,100 for an MI and $216 for sudden death, with a weighted (by incidence rate) average cost per event of $14,076.

Foregone Earnings

Hartunian also calculated foregone earnings based on reduced labor force participation using data for all four categories of coronary heart disease, broken down by sex and 10-year age categories. We have again adopted his basic results, with one substantial difference.

Hartunian valued fatalities by the foregone income that person would have earned had he or she continued to a normal life expectancy. This "human capital" approach to valuing mortality has been critized as an estimate of the value of avoiding the risk of death, because it places no value on avoiding the death of non-participants in the labor force (e.g., retirees, the handicapped, housewives), nor does it correctly reflect the willingness of people to pay for reductions in the risk of death. For these reasons, we valued fatalities separately from foregone earnings.

We then adjusted Hartunian's foregone earnings estimate, by age category, to exclude fatalities. The revised estimates were also adjusted for the increase in average non-farm compensation from 1975 to 1983, using data from Data Resources, Inc. (DRI), and for the use of the OMB-required 10 percent discount rate, rather than Hartunian's 6 percent discount rate, to determine the present value of future earnings.

This yields $90,000 in foregone earnings for non-fatal heart attack victims under 45 years old; $47,000 in foregone earnings for victims between 45 and 54 years old; and $22,000 in foregone earnings for victims over 55 years old. Based on the data from the Pooling Project and the NHANES, 16.1 percent of the non-fatal heart attacks in this age group occur in men under 45, 50.9 percent occur in men 45-54, and 33 percent occur in men over 54. Therefore, the weighted average of foregone earnings is $45,600 per event.

Fatality Costs

The reason for not using lost wages as our measure of the value of avoiding the risk of death is that it does not accord with economic theory. We live in a market economy, and in general, the value of goods and services is determined by what people are willing to pay for them, rather than by some absolute sense of what they are "worth".

People are willing to pay an unlimited amount to save their own lives, and indeed when an identifiable individual's life is in jeopardy, society as a whole is willing to spend large sums to preserve it. However, that is not the situation with which we are dealing. There are no identifiable individuals who will die if their blood pressure rises a few millimeters; rather, their risk of death increases by a small amount. Risk, not life, is the object we are trying to value. People make decisions that trade off risk against other things, including money, all the time. For instance, when purchasing a car, consumers compare price, quality, styling, and how safe the car is. Or, construction workers who weld the steel skeletons of skyscrapers are paid higher wages than workers in less risky building construction. Economists have studied such situations to estimate the "risk premium" in wages. For convenience, in comparing the results of different studies, the risks and risk premiums involved are multiplied by the population exposed to obtain a risk premium per statistical life saved. While this is the most convenient way to express the result, we must remember that we are really measuring

the willingness of large numbers of people to pay to reduce their individual risk by an amount which, when summed over all of them, has an expected savings of one life, and not the value to save a specific individual's life.

This said, economic research to date has indicated that people, on average, are willing to pay between half a million dollars and seven million dollars to reduce the risk that produces one statistical life saved. For example, Smith (1976)[10] regressed the log of wage rates against educational level, work experience, union membership, firm size, occupational category, full or part time work, marital status, health status, size of urban area, region of the country, industry category, and the accidental death rate per 1 million human work-hours, using data from the 1973 Current Population Survey and the 1970 Bureau of Labor Statistics Injury Reports. The variable for the risk of death (death rate per million work-hours) was highly statistically significant and suggested that workers required compensation for increasing their risk of death at a level of $3.22 million (1983 dollars) per statistical life saved. We have used a value of $1 million per statistical life saved to value reductions in the risk of dying from a heart attack due to lowering blood pressure. Obviously, this is near the bottom end of the range and, therefore, conservative.

Mortality Rates

All of the sudden death cases and fatal MI's need to be valued by the method above. But also, in addition to the immediate mortality associated with myocardial infarctions, patients who survive their first heart attack are at substantially increased risk of dying subsequently. Goldberg et. al. (1979)[11] studied hospital discharges for myocardial infarctions in Baltimore to compare prognoses. Life-table analysis of the data yields the following rates for survival.

Table I

Survival Rates of Survivors of Initial Myocardial Infarctions*

Time From Discharge (years)	Cumulative Survival (percent)
1	84%
2	77%
3	71%
4	65%
5	60%
6	56%
7	52%
8	47%

It is clear that a heart attack is associated with a considerable risk of post-discharge mortality. Therefore, to value the benefit of avoiding deaths due to heart attacks, we must include this increased risk of short-term future mortality as well.

* Data from Goldberg et al. (1979)

We will first compute the total value of avoiding the subsequent mortality associated with the survivor of an initial heart attack who is then discharged from the hospital. If we assume a constant value per statistical life saved, we can discount the value of lives saved in future years back to the year of the initial heart attack at a 10 percent discount rate.

This gives PV = V [.16+(.07)(.9)+(.06)(.81)+(.06)(.729)+(.05)(.656)+(.04)(.59)+(.04)(.53)+(.05)(.478)] = V[.417]

We must then subtract from this the present value of the average mortality risk for this age group, which is the value of avoiding the background risk of death. This gives V [.07]. The difference is the value of avoiding the excess risk of mortality due to an additional heart attack. This is the expected value of subsequent mortality avoided by preventing the initial non-fatal MI.

Since the value for fatal MI's and sudden death cases is just V, the weighted average value of avoided mortality per coronary case avoided, using $1 million per life saved, is $489,000 for people under 55 years old and $513,000 for people over 55 years old.

Summary

Combining these calculations, the average value of avoiding a heart attack is $59,750 for medical costs and foregone earnings, and $497,000 for the value of avoiding the subsequent mortality risk, for a total of $556,750.

VALUE OF AVOIDING STROKES

Strokes are also a major factor in medical care expenses, reduced participation in the labor force, and increased mortality. Again, we have relied principally on Hartunian for many of our statistics. Hartunian estimated the direct cost of the medical care received by stroke victims in 1975. We have inflated these to 1983 dollars and adjusted them to reflect an OMB-required 10 percent real discount rate for calculating present values. The results for males aged 35-64 are shown on Table 3 for hemorrhagic and infarctive strokes, and for transient ischemic attacks (TIA).

Table 3

Medical Costs for Strokes, by Type
(in 1983 dollars)

Males, by Age	Hemorrhagic	Infarctive	TIA
35-44	12,600	17,600	3,184
45-54	13,300	18,100	3,184
55-64	17,200	23,600	3,184

Mortality is also a frequent outcome in stroke victims, as the following table taken from Hartunian's data indicates.

Table 4

Excess Probability of Dying After a Stroke, by Year and Type

Hemorrhagic Stroke (males)

Age	1st Year	2nd Year	3rd Year	4th Year	5th Year
35-44	.60	.01	.01	.01	.01
45-54	.60	.01	.01	.01	.01
55-64	.59	.01	.02	.01	.01

Infarctive Stroke (males)

Age					
35-44	.26	.04	.04	.04	.03
45-54	.26	.05	.04	.04	.04
55-64	.26	.05	.05	.04	.04

TIA (average for all ages of men)

1st year	2nd year	3rd year	4th year	5th year
.089	.003	.024	.011	-.002
6th year	**7th year**	**8th year**	**9th year**	**10th year**
.020	.015	.056	.003	.014

We valued a reduction in the risk of dying in each year at $1 million per statistical life saved, and took the present value of the resulting amounts at a 10 percent real interest rate to obtain the following estimate of the value of reducing the mortality risk associated with strokes.

Table 5

Value of Avoided Mortality Risk
for Men by Age and Type of Stroke
(1983 dollars)

Age	Hemorrhagic stroke	Infarctive Stroke	TIA
35-44	630,000	380,000	171,000
45-54	630,000	390,000	171,000
55-64	630,000	410,000	171,000

Finally, we have again used Hartunian's estimates for foregone earnings, after adjusting for the OMB-required 10 percent discount rate and excluding that fraction of Hartunian's foregone earnings estimate that was associated with mortality. These results are on Table 6.

Table 6

Foregone Earnings Per Male Stroke Victim
(1983 dollars)

Age	Hemorrhagic Stroke	Infarctive Stroke	TIA
35-44	41,000	71,000	1,114
45-54	26,000	43,000	3,076
55-64	11,000	14,000	8,280

The weighted average value of avoiding a stroke incident is shown below.

Table 7

Total Value for Men
(1983 dollars)

Age	Mortality	Medical Expenses and Foregone Earnings	Total
35-44	424,000	59,000	483,000
45-54	416,000	43,000	459,000
55-64	385,000	30,000	415,000

Other Factors Affecting Stroke Victims

We have been unable to estimate a value for avoiding the loss in quality of life that occurs in stroke victims. This is a significant omission. For example, of the people in the NHANES II who reported having had a stroke in the past, 45 percent suffered paralysis in the face and 13 percent still had at least partial facial paralysis, 54 percent suffered paralysis in at least one arm and 21 percent still had paralysis of the arm, 59 percent had numbness in arms or legs and 28 percent still had numbness, 30 percent had vision impairment and 13 percent still had vision impairment, and 50 percent had speech impairment with 22 percent still suffering from speech impairment. While we have no estimates of peoples willingness to pay to avoid the risk of these profound injuries, common sense suggest that it is high.

THE COST OF HIGH BLOOD PRESSURE

Whether or not it leads to coronary or cerebrovascular disease, high blood pressure is a significant chronic illness. It also results in economic costs, including drugs, physicians' visits, hospitalization, and work loss. We used data from the NHANES II and from the National Institutes of Health to estimate the value of avoiding a case of high blood pressure. The NHANES II ascertained how many times per year a person saw a physician because of high blood pressure. The weighted average, for males between 40-59 years old with diastolic blood pressure over 90mm, was 3.27 visits per year.

The same population averaged 0.41 days per year when they were forced to remain in bed because of their high blood pressure, and 29 percent of them were on medication. The National Hospital Discharge Survey (1977) found that, excluding those with heart disease or cerebrovascular disease, people with high blood pressure used 4.6 percent of the occupied hospital days that year; this translates to an average of 3.8 percent of those with high blood pressure being hospitalized for 1 day each year. We have assumed that these results apply to the 40-59 year old age group as well.

Using these data, we estimated the value of avoiding a case of high blood pressure for one year. We assumed that medication costs $200 per year, that hospitalization costs $400 per day, and that physicians' visits for high blood pressure cost $35 each. We have valued avoiding one day that a person is forced to remain in bed at the average daily wage - $80. The expected

value of avoiding a case of high blood pressure, exclusive of its impact on heart disease and strokes, is $220. It should be noted that only 29 percent of the people with blood pressure above 90mm were on medication in part because some of them had not previously been detected as having high blood pressure. Therefore, average cost for a detected case will be higher. For example, Weinstein et al.,[12] used an average cost of $200 in 1975 dollars or about $450 in 1983 dollars, for treatment costs for patients undergoing medical care for hypertension. Nevertheless, we have used that $220 figure to be conservative.

Deaths from Other Causes

The National Institute of Health also analyzed the Framingham data for deaths from all causes. Again, they found a significant relationship with blood pressure. These deaths include deaths other than from heart attacks and strokes, because high blood pressure is generally unhealthy condition. Blood pressure is a risk factor for other diseases, as well as, on its own. We used the logistic regression coefficients that were available for males 40-54, and subtracted the predicted deaths from heart attacks and strokes to avoid double counting. As with heart attacks and strokes, we did our simulations on the NHANES II sample because it represents the entire United States population. As before, these were valued at $1 million per statistical life saved. And again, we prorated the deaths evenly among the years for lack of any evidence suggesting an uneven distribution.

APPENDIX

Table 1

Regression of Diastolic Blood Pressure in White Males Aged 40-59

Variables	Coefficient	T-Statistic	Probability
Age	0.2768	0.17	0.8636
Age^2	-0.0014	0.10	0.9321
Body Mass Index	1.131	8.55	0.0001
Log(blood lead)	3.954	2.85	0.0080
Dietary Potassium	-0.0018	4.92	0.0001
Hemoglobin	1.548	3.90	0.0005
Albumin	3.587	2.50	0.0179
Log(dietary vitamin C)	1.838	4.65	0.0001

Table 2

Systolic Blood Pressure in 40-59 Year Old White Males

Variables	Coefficient	T-Statistic	Probability
Age	1.311	0.57	0.5720
Age^2	-0.0068	0.30	0.7706
Body Mass Index	1.736	9.42	0.0001
Log(blood lead)	8.436	3.24	0.0028
Albumin	7.088	2.50	0.0178
Log(dietary Vitamin C)	2.411	3.84	0.0005
Log (dietary riboflavin)	-5.509	3.07	0.0044
Log(dietary oleicacid)	3.992	2.49	0.0183
Log(serum vitamin C)	-3.472	2.47	0.0184

Table 3

Weighted Logistic Regression Probablity of Blood Pressure Greater Than or Equal to 90 mm Hg Men aged 40-59

Variable	Coefficient	T-Statistic	P-Valve
Constant	-16.41	10.13	.0000
Log(Blood Lead)	0.693	3.96	.0000
Albumin	0.0873	3.70	.0001
Body Mass Index	1.700	9.34	.0000
Hemoglobin	0.0329	5.25	.0000
Log(Vitamin C)	0.3585	5.98	.0000
Dietary Potassium	-0.00058	7.47	.0000
Total Carbohydrates	0.00246	3.09	.0010

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