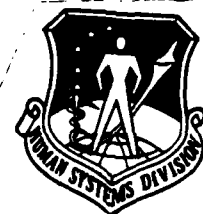


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FEASIBILITY OF EPIDEMIOLOGIC RESEARCH ON NONAUDITORY HEALTH EFFECTS OF RESIDENTIAL AIRCRAFT NOISE EXPOSURE

Summary of Literature on Cardiovascular Effects on Noise
Exposure

Volume III of III Volumes

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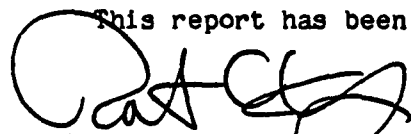
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19. ABSTRACT (Continue on reverse if necessary and identify by block number) This report examines the feasibility of conducting epidemiologic studies that would support inferences about effects of residential exposure to aircraft noise on nonauditory health. The type of aircraft noise of particular interest is that associated with supersonic and low altitude, high speed flight in Military Operating Areas (MOAs) and Military Training Routes (MTRs): both sonic booms and high peak level, rapid onset time subsonic noise. Potential studies considered are those with observational designs that are community-based or derived from audiometric databases. Since the primary goal of such studies is to improve the Air Force's ability to predict the effects on nonauditory health of noise exposure near MOAs and MTRs, such studies must provide: a demonstration of a causal chain from aircraft noise exposure to nonauditory adverse health consequences; and a reliable quantitative relationship between amount of noise exposure (dose) and degree of specific health consequences (effect).					
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1. Annotated Summary of Literature on Cardiovascular Effects of Noise Exposure

The tables in Volume III update the findings of EPA's review (Thompson, 1981) of the effects of noise on the cardiovascular system. They are divided into four sections containing summaries of laboratory and epidemiologic studies of aircraft, traffic, and industrial noise effects. The nine columns of each table are printed on left and right hand facing pages so that they may be read across the opened report.

This update includes, in addition to the studies in the EPA report, 6 studies on traffic noise, 6 on aircraft noise, 15 on industrial noise, and 6 frequently quoted laboratory studies which have been published in the English literature since 1981. Two earlier studies on aircraft noise have also been included. Although only research published in English was reviewed for this update, current literature searches indicate that most of the major epidemiologic studies of noise effects are now being published in English language journals. No previously unidentified studies were reported at the Fifth International Congress on Noise as a Public Health Problem in Stockholm, Sweden in August 1988.

Readers of this annotated review should note that publications were critiqued using criteria for judging causality in epidemiologic studies which included consideration of the reliability and validity of both noise and health outcome measurements, validity of the study design, temporal sequence of variables, potential biases, strength of observed associations, and evidence of data for dose response analysis.

Although a few researchers have reported that changes in fetal development (i.e., birth defects and low birthweight) and mental health problems may be related to high noise exposures, the weight of the evidence suggests that the cardiovascular system is the most likely target. Therefore, this summary considers only the noise effects research which focuses on the cardiovascular system. In addition to 17 population-based aircraft noise studies, the summary includes 75 epidemiologic studies of industrial noise, 12 of traffic noise, and 14 laboratory studies which are relevant for judging the effect of noise on nonauditory health.

No consistent patterns of response to noise emerge from studies of heart rate, cholesterol, beta-lipoproteins, cardiovascular disease rates or electrocardiographic changes. The cardiovascular effect of noise exposure that is best documented appears to be elevated blood pressure, as shown in this review. Major studies showing that blood pressures of workers with long-term exposure to continuous high level noise are higher than blood pressures of individuals working under less noisy conditions include those of Andriukin (1961), Shatalov and Murov (1970), Cieslewicz (1971), Parvizpoor (1976), Pilawska (1977), Jonsson and Hansson (1977), Britanov (1979), Friedlander et al. (undated), Belli et al. (1984), Verbeek et al. (1987), Wu et al. (1987), and Idzior-Walus (1987). Prevalence ratios for hypertension between high and low noise groups ranged from 1.3 to 2.8, and to 9.7 for special groups. It is evident in this literature review that the more recent studies which control for potential confounding factors, such as age, report lower prevalence ratios. Studies which refute the hypothesis of an association between industrial noise and elevated blood pressure include those of Malchaire and Mullier (1979), Lees and Roberts (1979), Demeter et al.

(1979), A. Cohen et al. (1980), Lees et al. (1980), Brini et al. (1983), Delin (1984), Talbott et al. (1985), and van Dijk, Souman and de Vries (1987). Many of these studies must be interpreted cautiously because of selection and temporal biases associated with cross-sectional designs, lack of standardization of blood pressure measurements, inadequate documentation of noise exposures, wide variability in noise exposures, and insufficient control of potential confounding variables.

Traffic noise studies are more difficult to assess due to the limited data on exposure and the many personal/social/living conditions that influence noise exposure. Although Von Eiff and Neus (1980) reported a significant prevalence ratio for hypertension of 1.6 for high traffic areas as opposed to low, the literature presented indicates that most studies showed no adverse effects of community noise on blood pressure (Drettner, 1975; Hedstrand et al., 1977; Takala et al., 1977; Knipschild and Salle, 1979; Neus, Ruddel and Schulte, 1983; Pulles et al., 1988). Neus, Ruddel, Schulte and Von Eiff (1983) and Otten et al., (1988) indicate small blood pressure differences--about 4 to 6 mm Hg--in residents of high traffic noise compared to lower noise areas.

The number of studies of health effects of aircraft noise has increased substantially since 1980. Two retrospective cohort studies of aircrew personnel (Brown et al., 1975; and Kent et al., 1986) failed to show an association between noise exposure and blood pressure, although Singh et al. (1982) reported elevations in both systolic and diastolic pressures in a cross-sectional study of armed forces personnel. Weak evidence of a positive association between aircraft noise and adverse health effects is presented by Karagodina et al. (1969), Koszarny et al. (1976), Knipschild (1977a and b), Knipschild and Oudshoorn (1977), Meecham and Shaw (1988), and Ising and Michalak (1988). The Ising and Michalak study is the only research to date which specifically addresses low altitude, high speed flight aircraft noise.

Overall, studies since 1980 show better control for confounding factors, more detailed description of noise parameters, and better quantification of effects with appropriate statistical analysis. Several research groups have begun to explore the link between factors such as annoyance, sensitivity, loss of sense of control, family history of hypertension, and noise exposure in the pathway to elevated blood pressure. Most of the studies continue to be cross-sectional in design although several ongoing prospective studies (the Caerphilly and Speedwell studies of Babisch et al., 1988) hold promise for definitive results within the next decade.

2. Summary of Epidemiological Studies of the Effects of Aircraft Noise on the Cardiovascular System

Table 2-1: Anton-Guirgis et al. (1986).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Anton-Guirgis, H.; Culver, B. D.; Wang, S.; Taylor, T. H; 1986, Vol II.; and Kamerman, C.; Sutherland, L.; Plotkin, K.; 1986, Vol. I.	Ecologic; Vital records; Mortality and hospital discharge morbidity data; Cross-sectional data. State of Nevada, including Tactical Fighter Weapons Center Range Complex of U.S. Air Force.	1970 and 1980 Census data for Nevada with estimates for 1968-1983. Large sample, data by exposure area not given but calculations show only 8% of population in low exposure townships.	Each county and township classified by yearly day-night average C-weighted sound level due to sonic boom, space averaged for each town. Average sound level equals sum of CLDN for 1969-83 divided by 15 (yrs.). Exposure area: high = CLDN >36 dB; med = CLDN 31-36 dB; low = CLDN <31 dB. Estimates reliable since derived from semi-automated entry procedure and fairly complete data sets.	Age, race, sex and cause specific mortality rates: ICD codes revision 7 and 8 for: cardiovascular diseases, hypertension, cerebrovascular accidents, cancer, all causes. Hospital discharge data for 1969-85 for same diseases collected from participating hospitals only. Response poor: 39% of the 33 hospitals refused: 42% in high noise, 29% in medium noise and 50% in low noise area.

Table 2-1: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential ecologic fallacy; Since townships could not be partitioned on age, race, sex, data could not be related to smaller exposure units; mortality data age/race/sex adjusted, but subject to errors in reporting; state sparsely settled resulting in small population at risk for various noise exposures.	No association observed for all cause, cardiovascular, cerebrovascular, or hypertension mortality and noise exposure when data sex/age adjusted. Multiple bivariate linear regressions between sonic boom and mortality revealed no consistent patterns.	"A preliminary exam of crude mortality rates showed that mean crude mortality increased systematically from low risk to high risk areas. However, this effect vanished when rates were age-adjusted and breaking the adjusted rates into cause-specific categories failed to resurrect it."	Noise exposure measure CLDN due to sonic boom relevant for Air Force, but poor quality of morbidity data and difficulties in relating age/race/sex specific mortality data to populations exposed makes this study poor for testing causal hypotheses.

Table 2-2: Brown et al. (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Brown, J. E.; Thompson, R. N.; Folk, E. D.; 1975.	Historical prospective/ Cross-sectional. Airline industry, U.S.A.	29 pilots; 29 non-flying executives.	8 h DRC (90 dBA) contour exceeded or equaled in all aircraft cockpits except jet transports. Duration - 6000 h of flying time or more; No noise levels given for executives.	Heart rate, SBP, DBP and Cholesterol level from annual health records.

Table 2-2: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Hearing thresholds monitored annually. Comparability of pilots and executives not given. No statistical controlling. Selection bias not addressed.	No effect parameters given. T-test used to detect differences between pilot and control group; F-test used to evaluate degree of change over time.	"No changes due to noise exposure were observed in BP, heart rate, cholesterol, or glucose levels."	Historical nature of data and type of noise make this study relevant for examining health effects of aircraft noise. Unfortunately, negative results may be related to small sample size.

Table 2-3: Cohen et al. (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cohen, S.; Evans, G. W.; Krantz, D. S.; Stokols, D.; 1980.	Cross-sectional. Community Survey - Schools; U.S.	262 children in 3rd and 4th grades; 142 from noisy schools; 120 from quiet schools.	Aircraft overflight noise; Mean peak in noisy school = 74 dBA; quiet school = 56 dBA; Highest read- ing in noisy school = 95 dBA; quiet school = 68 dBA; airport noise contours to approx. levels outside homes of noisy-school children; levels monitored 1 h in AM and PM.	Mean of 2nd and 3rd BP readings taken on consecutive days with automatic BP recorder in quiet room; coders "blinded" to test conditions.

Table 2-3: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Ecologic fallacy; self-selection; suspicion bias not controlled -- home noise contours not available for quiet homes; group matching and analysis controlled for hearing, grade, race, SES, number of children, but not age of child.	Regression analysis but no coefficients reported. SBP increased with years exposed but age not controlled ($p < .07$).	"Children from noisy schools have higher BP and are more likely to give up on a task than children from quiet schools... The greatest differences between the noisy and quiet groups occurred during the first two years of exposure." BP effects significant at $p < .05$.	Noise measured for 1 h in AM and 1 h in PM may not accurately reflect exposure. Duration of exposure not reported. Age effects ignored. Results must be interpreted with caution due to cross-sectional nature of study and potential bias.

Table 2-4: Cohen et al. (1981).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cohen, S.; Evans, G. W.; Krantz, D. S.; Stokols, D.; Kelly, S.; 1981.	Cross-sectional / longitudinal design. Community survey - schools U.S.	262 children in 3rd and 4th grades; 142 from noisy schools; 120 from quiet schools; At 1 yr. follow-up: 80 from quiet; 83 from noisy schools with 44 in noise at both testings and 39 in noise abated rooms at second testing.	Aircraft overflight noise levels in classrooms at T1: Noisy = 79.06 dBA; Abated = 63.17 dBA; Quiet = 56.60 dBA. T2: Noisy = 70.29 Leq; 55.82 L33; 91.50 Peak dBA Abated = 72.92 Leq; 49.27 L33; 71.27 Peak dBA. 300 overflights/day; 1 flight every 2 1/2 min. Noise contours from LA Airport used to approximate levels outside of homes of noisy-school children. Home noise levels not given.	Mean of 2nd and 3rd BP readings taken on consecutive days with automatic BP recorder in quiet room; coders "blinded" to test conditions.

Table 2-4: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Attrition bias: Noisy-school children who were not retested had higher BP than those retested whereas being retested was unrelated to BP for quiet-school children. Self-selection. Ecologic fallacy. Group matching and regression analysis controlled for hearing, grade, race, SES, number of children, but not age of child.	No association observed between noise exposure and elevated BP in school children. Regression analyses, but no coefficients given.	Cross-sectional sample at T1 "indicated inflated SBP and DBP for noisy-school children." There were no effects of noise in the longitudinal data which was as expected since a relatively high proportion of noisy-school children with high BP were lost to attribution... "Noisy-school children with the highest blood pressures move out of the noise area soon (within 2 yrs.) after the initial testing: $p < .001$ for SBP and $p < .03$ for DBP... Thus it appears that selective attrition, not adaptation, is responsible for the decrease of the difference between the noisy-school and quiet-school children."	Findings contribute little due to design problems as described.

Table 2-5: Frerichs et al. (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Frerichs, R. R.; Beeman, B. L.; Coulson, A. H.; 1980.	Cross-sectional; Vital Records Mortality Study; Neighborhood airport noise; mortality data; U.S.	1970 Census: Population of 89,019 in test area; 77,611 in control area.	Test area within the 90 dBA or more noise contour of airport; control area with noise level of 45-50 dBA.	Age, race, sex and cause of death -- specific-mortality rates. CVD: ICDA 390-448 and ICDA 430-438 as coded by the State of CA; limited to specific study areas.

Table 2-5: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Ecologic fallacy; errors in mortality data such as diagnosis, reporting; Self-selection into test and control areas. Data were adjusted for age, race, sex.	No associations observed. Standardized death rates and standardized mortality ratios given. The age/race/sex adjusted rates were nearly identical in the airport and control tract groups. SMR's for the two areas were very similar with values close to 1 indicating the observed deaths differed little from the expected. Other statistical testing apparently not done.	"We were unable to validate the findings of Meecham and Shaw..." Once the confounding effects of age, race, and sex were taken into account by direct and indirect methods of standardization, there was little difference in the mortality experience of the airport and control areas. Adjusted mortality rates due to all causes, cardiovascular diseases, or cerebrovascular disease did not differ appreciably between the two areas. Clearly any link between airport noise and mortality must be based on sounder evidence than has been presented to date if causality is to be inferred.	Because of ecologic nature of study, positive findings would only suggest hypotheses to be tested. These findings of no increase in mortality with noise correct a faulty analysis by Meecham and Shaw.

Table 2-6: Graeven (1974).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Graeven, D. B.; 1974.	Cross-sectional. Community survey; U.S.	Quota sample of 20% of females; 552 with 169 from Area I (highest noise). Area II = 96. Area III = 98. Area IV = 88. Control Area = 101.	Airplane noise measured by Noise Exposure Forecast (NEF): Area I: NEF > 40 Area II: NEF = 35-40 Area III: NEF = 30-35 Area IV: NEF = < 30. Average of 30 planes per hour during day and 8 per hour during night. Control Area = not within the flight path of a major airport.	Self-administered Symptom Check List; Poor measure of health problem.

Table 2-6: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Age, family income, education, length of residency in community, length of time in the home were considered. Control city respondents were older than noise exposed subjects.	Stepwise Regression for total sample and 5 separate regressions for the different levels of exposure showed no significant relationship between exposure to airplane noise and number of health problems. $R = -.03$ (zero-order) In multivariate analysis noise was 3rd variable to enter with $R = .198$, $p < .01$.	"Exposure to airplane noise was the 3rd most important factor in determining health problems." (Awareness and annoyance reactions were most important.)	Contributes little to judging causal relations due to design and unvalidated health measure.

Table 2-7: Ising and Michalak (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Ising, H.; Michalak, R.; 1988.	Quasi-experimental and cross-sectional; Haifa, Israel and Federal Republic of Germany.	24 volunteers, 70-88 yrs. old for exposure to subsonic low-level flight noise; excluded persons with conductive hearing loss greater than 10 dB at one or more frequencies between 0.25 and 4 kHz, or acute disease, or unstable BP. 430 children, aged 9-13 yrs. -- no details given.	Recorded flight noise with level increases of 30 dB rise time in 0.4 s with L max above 110-115 dBA via earphones to elderly. Children from 75 m low-level flight area and from 150 m flight area -- details not given.	BP and heart frequency with semi-automatic devices every 5 min while exposed to noise via earphones -- details not given.

Table 2-7: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Data not available for evaluation; confounders apparently not considered.	Means, S.D., distributions of differences graphed. BP for girls 9 mm Hg higher in 150 m area than in 75 m area. No blood pressure difference noted for boys.	Flight noise with fast level increase causes significant stronger blood pressure increases than flight noise with the same peak level but slower level increase... Field measurements of 430 school children in areas with 75 m and 150 m flights indicate that chronic exposure to low-level flight noise with peaks up to 125 dBA may lead to chronic blood pressure increases.	Too few details are given in the report to allow assessment of study validity. Inferences that chronic exposure to subsonic noise may lead to permanent BP changes on the basis of these short-term exposures and cross-sectional responses may not be appropriate.

Table 2-8: Karagodina et al. (1969).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Karagodina, I. L.; Soldatkina, S. A.; Vinokur, I. L.; and Klimukhin, A. A.; 1969.	Cross-sectional; Community Survey and analysis of diagnostic cards. USSR.	22,000 persons residing in 22 urban and rural areas within 40 km of airports. Breakdown by noise area not given. 145 diagnostic cards examined.	Noise patterns for the TU-104 plane given in dBA by climb pattern. No other details given.	Cardiovascular, nervous and gastro-intestinal diseases as defined on "diagnostic cards." Clinical exams of 9-13 yr. old children by clinical staff of Erisman Research Institute of Hygiene. Data inadequate for judging quality.

Table 2-8: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential for selection and misclassification bias. No data to indicate control for age or other confounders.	Incidence of cardiovascular (hypertension, hypotension) nervous and gastrointestinal diseases reported to be 2-4 times higher near airports than in control areas. No statistical analysis described.	"...analyzing 145,000 diagnostic cards revealed differences between populated areas located near the airport (1-6 km) and control areas located at fairly large distances (40 km) from the airport or air routes; there was a higher (by 2-4 times) incidence of otorhinolaryngological diseases (otitis and auricular neuritis), cardiovascular diseases (hypertension, hypotension, etc.), nervous diseases (neuritis, asthenic states), and gastrointestinal diseases (gastric and duodenal ulcers, gastritis). Clinical exams showed BP abnormalities, higher lability of pulse, cardiac insufficiency in children living near airports."	Probably prevalence data at best. Inadequate data provided to judge study design and quality. Does not contribute to understanding of effects of aircraft noise on health.

Table 2-9: Kent et al. (1986).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kent, S. J.; Von Gierke, H. E.; Tolan, G. D.; 1986.	Retrospective cohort of Air Force Aircrew Members. School of Aerospace Medicine (SAM); U.S.	2250 U.S. aircrew classified by hearing loss: 669 SNHL; 1339 Non SNHL; 20-57 yrs. of age.	Aircraft cockpit noise levels ranging from 89 dBA to 102 dBA; estimated 8 h exposures approximate: 90 dBA for 30-40 yrs. olds; 90-95 dBA for 41-50 yrs. olds; 95 dBA for 51-60 yrs. olds; Sensorineural hearing loss (SNHL) diagnosed by otolaryngologist using pure tone hearing thresholds at 3, 4, and 6 kHz. Noise exposures associated with SNHL calculated using tables based on Passchier-Vermeer data. Individual noise exposures not available.	BP taken pre-treadmill, in supine position, by trained technician using standard clinical sphygmomanometer. Hypertension: BP >140/90 mm Hg. on two readings/day for 3-5 days and no identifiable cause. Resting EKG, treadmill tests, serum lipids, glucose tolerance, selected angiography & physical exam to define coronary artery disease & other CVD diseases, ICD 390-430, obtained during first examination.

Table 2-9: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Healthy worker bias probably present. Other selection factors may exist since only borderline conditions referred to SAM. No data on smoking. Age controlled by stratification but other variables may not have been adequately controlled by categorizing into high risk and low risk on basis of BP, % body fat and relative weight.	Although the unadjusted RR of SNHL and CAD was 2.4, when adjusted for age there was no association between SNHL and CAD ($p < .05$). For essential hypertension, crude RR of 1.7 reduced to approx. unity when adjusted for age. Two-way ANOVA showed no significant difference at .05 level for each of 5 CV measures.	...After adjusting for age, no significant associations were found between sensorineural hearing loss and blood pressure or cardiovascular disease diagnoses. The incidence of hypertension with age is not significantly different from the incidence found in representative studies of the "average" U.S. population.	Major value of this study for assessing the effect of aircraft noise on CVD is that it used comprehensive clinical evaluations to determine disease status and it documented the exposure to aircraft noise. The fact that noise-induced hearing loss was used as a surrogate for exposure, that all subjects were exposed to relatively high levels of noise and that the cohort was not representative of the military or the general population, detracts from its usefulness.

Table 2-10: Knipschild (1977a).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Knipschild, P.; 1977a.	Cross-sectional Community survey; The Netherlands.	2233 individuals in high noise area; 3595 in the lesser noise area; Ages 35-64.	Aircraft noise measured at center of village; Much noise: B = 40-60 where NNI > 37; Less noise: B = 20-40 where NNI = 20-37.	No raw data provided. Hypertension defined as SBP > 175 and/or DBP > 100; angina; pathological heart shape; pathological ECG. Data standardized by using same staff.

Table 2-10: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Less than 50% of adults responded: 39% in high noise area and 43% in lesser noise area. No evidence of controlling for confounding even though more smoked, were over-weight and of lower socio-economic status in the high noise area than in the low noise area.	Used Cochran's test for 2 X 2 contingency tables. Prevalence ratios calculated: Treatment for heart trouble = 1.4 ($p < .05$); Treatment for hypertension = 1.5 ($p < .001$); Taking CVD drugs = 1.4 ($p < .05$); Pathological heart shape = 1.6 ($p < .05$); High BP = 1.7 ($p < .001$). Regression line: % hypertension = $0.34 B - 0.57$ (for each 3 unit increase in B, there is a 1% increase in hypertensives.)	"In areas with much aircraft noise ($B > 40$) the prevalence of CVD appears to be higher."	Screening completed by same staff. Low response rate and confounding on weight, smoking, social class could account for the higher rates in the high noise area. Prevalence data must be interpreted with caution.

Table 2-11: Knipschild (1977b).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Knipschild, P.; 1977b.	Cross-sectional. General practice survey. The Netherlands.	PAR = 12,000 in high noise area. PAR = 17,500 in low noise area.	Aircraft flying over area. 3 areas exposed: E1: B = 45-55; E2: B = 35-45; NNI = 33-50. EC: B = 20-35; NNI = 20-33; Control area: B = < 20 or NNI < 20. Village exposed from 1968-1974 in a direct line of runway.	Dx. of CVD from general practitioners. Patients seen by different GP's in the areas. No definition of hypertension.

Table 2-11: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Age and sex controlled; no other variables considered; villages differed in socio-economic class; probable self-selection into services.	Significant associations and a dose response relation between noise and contact for: cardiovascular disease, hypertension, psychological and psycho-somatic problems. Prevalence ratios for CVD and hypertension 1.7 (high noise to low noise).	For persons aged 15-64 it was found that the contact rate in the exposed area was almost twice that in the non-exposed area. For 15-64 yr. olds, the contact rate for CVD was 9% in E1, 6% in E2 and 5% in area C. Complaints continued in weeks with no airplane flyovers, suggesting a long term effect of noise.	Study suffers from problems inherent in all cross-sectional surveys and by use of surrogate measure (contact rates) for disease prevalence.

Table 2-12: Knipschild and Oudshoorn (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Knipschild, P.; Oudshoorn, N.; 1977.	6 year (historical) longitudinal study. Community Drug survey. The Netherlands.	Same as General Practice Survey.	Same as General Practice Survey.	Purchase of drugs from pharmacies by inhabitants of each noise area: sleeping pills, sedatives, antacids, cardiovascular medications including anti-hypertensives. Drug index = total purchases divided by number of inhabitants per year.

Table 2-12: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential ecologic fallacy since unable to link purchases and demographic characteristics to individuals. Drug index may not be good indicator of true prescription of drugs nor of disease incidence/prevalence. No correction for age or other differences between the noise areas.	No statistical tests; anti-hypertensive drug index increased from 13 in 1968 to 28 in 1974 in high noise area but remained constant in low noise area.	"In a period of six years the purchase of anti-hypertensive agents had gradually increased to twice the initial amount; this result suggests that the prevalence of hypertension has increased accordingly."	Unfortunately, no incidence of disease data were collected in this time-dependent study of drug use. Findings difficult to interpret because of surrogate nature of health outcomes although noise exposure measures are probably adequate.

Table 2-13: Koszarny et al. (1976).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Koszarny, Z.; Maziarka, S.; Szata, W.; 1976.	Cross-sectional Neighborhood survey (airport & residential noise); Poland.	256 in high noise zone; 255 in low noise zone; men and women age 20-70 yrs.	High noise zone = > 100 dBA; Low noise zone = 80-90 dBA; No other information provided.	Frequency of symptoms including cardiac pains and taking of cardiac medicines.

Table 2-13: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Groups similar on type of education, occupation, working conditions, age and sex but differed on housing and living conditions -- poorer housing in low noise area. Data stratified by age and sex. No other evidence of control.	Strength of association not given but prevalence ratios can be calculated as follows for women only: nervousness 2.5; taking cardiac medications 1.5; heart pains 1.1; stomach complaints 1.6. All differences significant at $p < .05$.	"The relationship between some symptoms of aggravation and ill health and the acoustic conditions in the place of residence indicates the probability of the negative influence of airport noise on the residents' state of health." The high percentage of persons complaining of symptoms and the frequent occurrence of illness among women, but not men, from the worst acoustic conditions seems to support the relationship between airport noise and the state of health. "On the other hand, one cannot exclude the assumption that the above mentioned indexes result from other unfavorable effects of the environment, especially the work environment."	Findings contribute little to the understanding of noise and health effects because of poor specification of both noise and health parameters.

Table 2-14: Meecham and Shaw (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Meecham, W. C.; Shaw, N.; 1979.	Cross-sectional Neighborhood; airport noise; mortality data; U.S.	86,200 in test area; 77,968 in control area.	Aircraft, jet engine noise. Intensity: within 90 dBA+ noise contour for test area; 45-50 dBA for control area. No other information provided.	Stroke deaths from county mortality files. Other cause-specific mortality considered.

Table 2-14: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Two areas matched on age, income, racial breakdown; data presented to show areas actually differed on these variables. No controlling evident. Reporting errors differed in the 2 areas. Ecologic fallacy possible.	Chi-square test applied to diseases showing greater than 20% increase in mortality rates in the two areas: stroke and cirrhosis of liver.	"The main result is that for these two diseases, often associated with increased daily nervous tension, there has been an increase in mortality rates for the area of heavy noise radiation under landing jet aircraft as compared with an area removed from such noise effects."	Major problem of this mortality analysis is lack of adjustment for age and race differences. Reanalysis of data by Frerichs found no differences.

Table 2-15: Meecham and Shaw (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Meecham, W.C.; Shaw, N.A.; 1988	Cross-sectional. Ecologic analysis of mortality data; LAX airport, USA	103,000 people in exposed area; 91,000 in control area.	Exposed area: 90 dBA noise contour defined by technical reports and on-site measurements. Non-exposed area: nearby areas in which jet noise was not dominant.	Mortality data tapes for 1970-77 obtained from Los Angeles City Dept. of Health Services

Table 2-15: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Exposed and non-exposed areas similar on race and monthly income; unclear as to specific race-income adjustments for given age groups. No other confounders such as family history, smoking, cholesterol adjusted.	SMR using average of 1970-1980 census. SMR for deaths of people 75+ is 1.18.	"It is found that there is an eighteen percent increase in cardiovascular deaths, for people over 75, at a 97% confidence level, in areas around the airport". (No difference found for CVD deaths in ages 35-74).	Study suffers from potential ecologic fallacy. Authors state there is a "rapid turn over of residence in these areas...it appears that the average time of residence may be but a few years". Selective migration may possibly account for excess deaths.

Table 2-16: Singh et al. (1982).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Singh, A. P.; Rai, R. M.; Bhatia, M. R.; Nayar, H. S.; 1982.	Cross-sectional (quasi-experimental). Armed Forces Personnel; India.	75 healthy individuals exposed 10-15 years to occupational noise; 36 non-exposed; selected from large population of armed forces personnel.	Group of exposed and non-exposed workers tested in 90 dBA continuous white noise for 26 min in room at 20 degrees $\pm 1^{\circ}$ c. Noise level in work situation determined with B & K impulse precision sound-level meter in its linear as well as impact mode. History of total noise exposure taken. Pure tone audiometry made in sound-proof room using ISO calibrated audiometer.	BP, heart rate, oral temperature, respiration rate carried out at rest in supine position. EKG recorded using BPL model DR-108 electrocardiograph. Heart rate calculated by recording single lead EKG at regular intervals.

Table 2-16: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Possible selection bias -- no information as to how subjects were selected. No evidence of control for potential confounders such as obesity, family history of hypertension.	Mean blood pressure: Exposed: 121.95 SBP 79.20 DBP Non-exposed: 116.85 SBP 73.56 DBP Heart rate 66 in exposed and 62.76 in non-exposed group. Statistical tests not given.	"Blood pressure, both systolic ($p < 0.01$) and diastolic ($p < 0.05$) were found to be significantly higher in the exposed subjects. Irregularity in cardiac rhythm, both in amplitude and duration, was found in 18% of the exposed subjects as against 6% in the non-exposed group."	Lack of sampling information and small control group limits the usefulness of this study.

Table 2-17: Stansfeld et al. (1985).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Stansfeld, S. A.; Clark, C. R.; Turpin, G.; Jenkins, L. M.; Tarnopolsky, A.; 1985.	Cross-sectional Community survey. Neighborhood.	77 women, 18-50 yrs. of age interviewed in 1977 in West London survey; selected from both high and low noise zones, but distribution not given from high, medium, low noise sensitive groups.	Aircraft noise: High zone > 45 NNI; Low zone < 45 NNI Noise sensitivity defined by McKennell's list of annoying noises and by self report with many discrepancies in classification between the 2 measures.	Two BP readings while seated, averaged; taken by trained observers in home using random zero sphygmomanometer. Heart rate by Medilog portable recorder: mean interbeat interval and standard deviation, mean squared successive difference of interbeat interval and number of reversals. ECG electrodes of medicotest; observers trained to use equipment in home.

Table 2-17: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Noise sensitive (NS) group classification by two measures varied greatly suggesting possibility for misclassification bias; 11-29% of data considered unreliable and excluded; representativeness of sample not shown.	Differences between groups not shown. Multivariate analysis of variance used to test significance.	"SBP and DBP in women not related to sensitivity to noise in this sample... Heart rate was slower for high NS women which may reflect an absolute difference in tonic heart rate between high NS women and low NS women. The standard deviation of the heart rate, the mean squared successive differences, and the number of reversals were not affected either by noise sensitivity or by noise zone."	Sample size was small and it is not clear that noise sensitivity measure is independent of noise annoyance, but study may be helpful in explaining differences noted in general population studies.

3. Summary of Epidemiological Studies of the Effects of Industrial Noise on the Cardiovascular System

Table 3-1: Andriukin (1961).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Andriukin, A. A.; 1961.	Cross-sectional. Industry; Russia.	676 males; 556 females a. 307 in tool making b. 200 in sorting c. 464 in automatic lathes d. 263 in ball-bearings.	Unsteady workshop noises: a. = 93 dB b. = 103 dB c. = 103 dB d. = 120 dB Exposure: 7-8 h daily in 3 shifts, alternating weekly; 50% workers in same plant for more than 10 years.	BP recorded during work after 10 min break, on right arm using Riva-Rocci apparatus and Korotkov's method; repeated until constant values; 130/90 taken as upper limit of normal for < 40 yrs. of age and 140/90 for > 40 yrs. of age.

Table 3-1: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection criteria into study, non-response and attrition not stated; specific individual exposures not given; partial control for age; no control for sex, variation in exposure time, medications, comorbidities, etc.	No statistical test or evidence of such tests given. Although morbidity was said to be 2X as high in persons under 50 as in workers in quiet factories, study too poorly controlled to accept as significant.	"In workshops with intense noise (sorting room, automatic lathes, ball bearings) hypertension is found more frequently than in the less noisy workshop (toolmaking)... In the noisier workshops the morbidity is almost twice as high in persons under 50 years of age...the percentage of patients increase with the duration of employment; particularly marked is the increase after five years of work in the noisiest workshops."	Results of questionable value since control group was unspecified and there was no evidence of adjusting for confounding.

Table 3-2: Andrukovich (1965).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Andrukovich, A. I.; 1965.	Cross-sectional. Industry, (Bendery silk combine & textile weaving factory); Russia	846 female spinners & weavers, 16-49 yrs. of age, 1-5 yrs. employment compared to 8972 women in the population of the geographic area.	High frequency noise with intensity of 99-102 dB in weaving and 87-88 dB in spinning.	BP measured 4-8 times by Korotkov-Ianovskii method, with Riva-Rocci mercury manometer, right arm, 15-20 min after 1st shift. Taken over yrs. 1959-1963. Hypertension defined as 140/85 mm Hg; Hypotension $\leq$ 90/50 for 16-19 yr. olds and $\leq$ 100/60 mm Hg for 20-49 yr. olds.

Table 3-2: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No data as to selection of control group; possible selection bias and bias due to attrition over the 5 years; stratified into 5 year age intervals.	Conclusions not stated quantitatively; method of analysis is unclear.	"Arterial pressure was high in female weavers compared to the mean age-specific indexes for arterial pressure in women of the control...The processing of the data obtained demonstrated the statistical significance of the higher levels of the systolic pressure within the age groups 16-19, 30-39 and 40-49 years."	Characteristics of general population controls unclear. Poor study methodology.

Table 3-3: Antonova (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Antonova, K. P.; 1979.	Cross-sectional; Pre-/during-/post-shift measures. Industry; Russia	33 male workers; no controls.	Ore-dressing plant noise 92-112 dB mills; 97-104 dB separators; no details provided; employment 3 months to 11 yrs. Vibration measurement presented in detail.	Pulse rate, brachial and temporal arterial pressure. No data or measurement procedure provided.

Table 3-3: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Individuals measured at several points in time -- no mention of controls.	High noise workers said to show rise in BP but inadequate data to judge the association.	Brachial and temporal arterial pressure rose in mill operators, mill operator assistants had 2-6 mm increase in brachial arterial pressure whereas separator operators showed no significant change in arterial pressure.	Poor documentation in study makes this study useless for judging causality.

Table 3-4: Barhad et al. (1969).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Barhad, B.; Gradina, C.; Mihaila, I.; Deculescu, F.; Marinescu, V.; Cristecu, I.; Miculescu, S.; 1969.	Cross-sectional; Pre-/post-shift measures. Industry (Smelter); Rumania.	132 workers exposed to noise.	Short-term noise effects assessed; levels between 98-127 dB with spectrum extending over several octaves; 100 workers employed for 5 years.	SBP, DBP, pulse rate; digital plethysmography, coordinator-rhythm strength test; audi- tory analyzer; new cases of disease; no diagnostic criteria, defini- tions or measure- ment details reported.

Table 3-4: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No evidence of control for age, sex, weight, comorbidities, hearing levels or intra-individual variation of BP.	No statistical tests stated but said to show a higher rate of circulatory disease (ratio 1.26) among workers in higher noise.	"Under the effect of work carried out in noise...most of the workers showed toward the end of the work day a decrease of the systolic and diastolic pressure which varied between 7 and 14 mm Hg. compared to the values found at the beginning of work...The study of the clinical and statistical morbidity of smelting division workers shows, in relation to workers in the motor section, a significantly higher disease rate." (1.26 for circulatory disease) "Our results show that, under the influence of noise and vibrations, changes appear which are shown by disturbances of the functional condition of the entire organism, and not only of the auditory analyzer."	May be useful in judging short-term effects, but inadequate statistical analysis and limited information about health measures.

Table 3-5: Belli et al. (1984).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Belli, S.; Sani, L.; Scarficcia, G.; Sorrentino, R.; 1984.	Cross-sectional. Industry (Polypropilenic fiber plant); Italy.	490 exposed to noise; 450 not exposed; 89% of the textile workers and 90.9% of office workers included; Age Groups: < 35 yrs.; 36-45; 46-55; ≥ 56 yrs. old.	Exposed were all workers in area with average noise levels ≥ 85 dBA as measured by a Bruel and Kjaer 2204 phonometer with values averaged on 60 s time span; Ranges from 86-108 dBA. < 85 dBA represented non-exposure. Duration of employment not given.	Medical examination with highest several DBP and SBP, taken before work shift, in recumbent position with Riva-Rocci apparatus used to define hypertension as DBP > 95 mm Hg. Medical exam included age, smoking, blood values for cholesterol, glucose.

Table 3-5: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Data stratified by age, smoking habits, total cholesterol, triglycerides and glucose, with confounders examined one at a time except for triglycerides and glucose; possible selection bias since duration of employment not given; selective recall of smoking habits possible.	Stratified analysis using Mantel-Haenszel procedures. Crude RR = 1.24 N.S.; Controlling for triglycerides and glucose RR = 1.34 (1.14-1.57); for age RR = 1.25 (1.01-1.54); for smoking = 1.27 (1.05-1.54); for triglycerides = 1.31 (1.09-1.57); for glucose = 1.24 (1.00-1.54); for cholesterol = 1.21 (0.96-1.53 C.I.).	"...a statistically significant association of chronic arterial hypertension with professional noise exposure is obtained only when the data are stratified according to the blood concentration of both triglycerides and glucose: RR = 1.34 (1.14-1.57, 95% C.I.)."	Prevalence data suggestive of a weak association between high noise and hypertension but variables controlled singly; sample size relatively small given number of variables included; results would be more convincing if length of exposure were known.

Table 3-6: Brini et al. (1983).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Brini, D.; Ratti, R.; Torricelli, P. A.; Cirila, A. M.; 1983.	Cross-sectional. Industry; Italy.	1488 male workers; 365 exposed to continuous noise; 436 exposed to impulse noise; 667 not exposed to industrial noise.	Continuous noise of > 80 dBA = exposed. Impulse noise of > 90 dBA = exposed. Controls = no industrial exposure. Bruel-Kjaer Model 2209 phonometer used to classify noise level.	BP was measured by WHO criteria; Hypertension defined as SBP $\geq$ 160 and/or DBP $\geq$ 95 mm Hg. Subjects said to be similar on other characteristics such as length of service and pleasure giving habits.

Table 3-6: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No evidence of control for confounding variables, unable to judge possible selection biases due to scanty data.	Several chi-square values given, apparently for crude risk estimates. 12.46% workers exposed to continuous noise had hypertension compared to 13.94% of nonexposed (NS). 23.85% of subjects exposed to impulse noise had hypertension compared to 13.94% of controls for ratio of 1.71, chi-square = 17.65.	"Our findings indicate that hypertension is distinctly more prevalent in persons exposed to impulse as exposed to continuous noise."	Poor reporting of study makes evaluation difficult.

Table 3-7: Britanov (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Britanov, N. G.; 1979.	Cross-sectional. Industry (Chemical fiber); Russia.	113 women ages 23-49; 3 groups: I. Spinners; II. Warpers; III. Twisters.	Not specified for each group; 50-100 dB ranges with 63-8000 Hz frequencies. Group I: 90 dB with 125-2000 Hz frequencies. Group III: 98 dB. Workers had 6-10 yrs. of employment.	Blood pressure with hypertension defined as $\leq 160/95$ mm Hg.; borderline as 140/90 - 150/95 mm Hg. No information provided on measurements.

Table 3-7: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Possible selection bias; small sample size; no evidence of controlling for age, obesity or social class in BP analysis.	Reports prevalence ratio of 2.8 for hypertension but no evidence of statistical testing.	"In workers exposed to the combined effects of noise at the level of MPL or 5-11 decibels higher and to acetone within the limits of MPC, and also in those exposed to the isolated effect of similar noise, functional alterations of the nervous system following the pattern of neurovascular dysfunction and neurotic reactions, and borderline and arterial hypertension, were more frequent. Alterations of physiological functions (visual-motor reaction time, endurance of static work) were more pronounced in workers exposed to the simultaneous effects of noise and acetone than in those exposed mainly to either noise or to acetone."	Although suggestive of an association, it is not clear that age was adequately standardized.

Table 3-8: Capellini and Maroni (1974).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Capellini, A.; Maroni, M.; 1974.	Cross-sectional. Industry (Chemical); Italy.	1286 men; 60 women; 98.6% of employees.	No information provided. Implied periodic instrument samplings.	Hypertension: SBP > 160 mm Hg. and DBP > 90 mm Hg. Coronary disease: Myocardial infarction diagnosed by specialist on history and/or EKG and VCG; angina pectoris, abnormal S-T response to exercise.

Table 3-8: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Age controlled by stratification. Blood pressure and sex not controlled in myocardial infarction data.	Chi-square, weighted regression after logistical transformation of disease data. Regression coefficients not given.	"The data indicate that the risk quota for coronary disease due to exposure to intense noise is equal to that in the non-exposed population by an increase in age of 10 years. No significant correlations were observed for hypertension."	Thorough statistical analysis but description of design is scanty making evaluation difficult.

Table 3-9: Cavatorta et al. (1987).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cavatorta, A.; Falzio, M.; Romanelli, A.; Cigala, F.; Ricco, M.; Bruschi, C.; Franchini, I.; Borghetti, A. 1987	Cross-sectional. Glass industry; Italy.	60 shift workers exposed to machinery noise; 52 controls from machine shop and sorting; matched for age, exposed to noise <78 dBA. Subjects with histories of heart disease and high BP excluded.	Noise measured by B&K phonometer with octave band analyser and General Rodo personal dosimeters. Ranged from 92-96 dBA per 8 h shift (continuous noise). Mean duration of exposure 8.4 yrs + 1.5 yrs.	Serum epi-nephrine, nor-epinephrine, dopamine, cortisol measured from blood drawn after worker lay supine 10 min before a shift, and at midshift (about 4 h). No smoking, alcohol, coffee for 2 h before each sample. BP measured with mercury sphygmomanometer before, half-way and at end of each shift with subject resting supine for 5 min before 1st reading; 2nd reading 2 min later. Laboratory methods described in detail. Urine samples collected on same schedule as blood to measure homovanillic acid, vanilmandelic acid and creatinine.

Table 3-9: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects reported to be healthy and no variables controlled in analysis. Not clear as to how sample was drawn.	Student's t-tests of means applied.	"Norepinephrine, epinephrine and vanilmandelic acid were significantly increased ($P < 0.01$) during work-shifts in the group exposed to 90dBA, compared with baseline levels and also with catecholamine levels in the group exposed to 78 dBA...Average values of systolic, diastolic and mean arterial pressure showed no significant variations between the different times."	Study may be useful in understanding physiological mechanisms of hypertension.

Table 3-10: Cieslewicz (1971).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cieslewicz, J.; 1971.	Cross-sectional. Industry (Spinning and Weaving); Poland.	Weavers: 490 women and 212 men; Spinners: 353 women and 252 men; Age groups: 18-29; 30-39; 40-49; & > 50.	Weavers exposed to noise ranging from N = 96-116 for octaves 250-8000 Hz; Spinners exposed at 84-90 dB. Weavers used own ear protectors. Frequency composition of noise for weavers shown; no data for spinners. Most weavers had worked 15 yrs. or more in the mill; no data on spinners.	Hypertension defined as BP > 150/95 mm Hg. Data from periodic examinations of workers. No information on measurement, etc.

Table 3-10: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection, selective survival; measurement bias. Data stratified by age; no evidence of controlling for other variables - smoking, shifts, housing, etc.	From tabled data, for ages over 50 yrs., prevalence ratios for hypertension for weavers compared to spinners: women = 2.2; men = 2.7. No statistical tests provided.	"The analysis of the frequency of illness among the workers of the weaving and spinning (hard waste) mill has shown clearly that hypertension, neurotic syndromes, and gastric and duodenal ulcers are more frequent among the workers at the weaving department. This difference must be connected with the sharply different working conditions of the two groups and, especially, with the extent of exposure to noise pollution."	Strength of study is its consideration of multiple variables in a large group of workers. It suffers from failure to control adequately for extraneous variables.

Table 3-11: Cohen et al. (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cohen, A.; Taylor, W.; Tubbs, R.; 1980a.	Cross-sectional. Industry (Paper-making Plant); U.S.	51 workers with hearing loss. 51 workers without hearing problems.	Proxy measure-high frequency noise induced hearing loss of 65 dB or more at 3k, 4k, and 6k Hz. Noise intensity levels varied: paper machines at 98-102 dBA and cutting- sorting-machines at 85-92 dBA. Duration of work exposure: 22.3 yrs. for hearing loss group and 12.5 yrs. for controls.	BP -- based on last 2 of 3 readings, taken in recumbent position after 30 min rest, by trained technicians unaware of hearing status, included first systolic phase and phase IV and V diastolic. Hypertension defined by WHO criteria of > 160/95. Pre-existing history and medications recorded.

Table 3-11: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Analysis controlled for age and body size. No controls noted for race, sex, comorbidity, temperature, etc.	Covariate analysis; differences between groups not specified.	"Statistical evaluation of the blood pressure data found no reliable difference between the worker groups with high-frequency loss vs normal hearing even with covariate analysis designed to adjust for differences in age and body size (weight/height ratio)...Clearly, it is too early to draw any conclusions about noise as a causal factor in cardiovascular disease."	Study suffers from a small sample and possibly Type II error. Hearing loss may not be a good surrogate of noise exposure.

Table 3-12: Cuesdean (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cuesdean, L.; 1977.	Cross-sectional. Industry (Chemical).	160 exposed to noise; 160 not exposed. (149 males and 11 females in each group.)	Intensity: air compressor operators = 100-106 dB; stokers = 100-106 dB; mechanics = 95-100 dB; electricians = 90-95 dB; lab assts = 85-95 dB; Exposure duration: 6 yrs. in 21-40 yr. old group; 10 yrs. in 41-60 yr. old group. Measurement 2 h after an 8 h morning shift; in winter.	ECG on 12 leads; Alterations classified by criteria of the Minnesota Code. BP measured during first h of work and at end of shift. Hypertension defined as $\geq 140/90$ mm Hg. Hearing loss.

Table 3-12: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No data provided as to total noise exposure, how subjects were selected or completeness of sample. Relative humidity of exposed groups varied, no data given for controls. Groups compared on smoking, overweight and diet excess in animal fat but no analysis. Sex, age, SES and physical activity not controlled.	None specifically stated.	"Data indicates an increased frequency of ECG alterations at rest. ...Indices 3-1, 3-3, 9-2 were more frequent among the men working in a medium of permanently intense noise. The highest incidence was that of upward deflection of segment ST, of the benign type. Intense permanent industrial noise causes...a high incidence of cardiovascular ECG alterations that can be statistically interpreted."	Study not described well. Although ECG changes are statistically significant, it is not clear that these are of any clinical significance as suggested by author.

Table 3-13: Dega and Klajman (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Dega, K.; Klajman, S.; 1977.	Quasi-experimental; Pre/post shift measures on exposed and nonexposed. Industry (Shipyard propeller grinders); Poland.	36 male propeller grinders; 20 workers in similar jobs as controls.	Source: pneumatic hammers and high speed grinders; variable frequency; intensity at all stations exceeded N-85 curve. Plotted by dB per octave for 31.5 through 8000 Hz. Duration: 1-14 yrs for 8 h daily; instrumentation with B&K set.	BP and heart rate; heart minute volume according to Starr's formula; no additional data presented.

Table 3-13: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Exposed and nonexposed similar as to mean age and average years employed in ship-building. No additional data on control of potential confounders given.	Means and t-tests used to compare exposed groups. Mean differences in heart minute volume before and after noise for: exposed = 1680 ml. non-exposed = 830 ml.	"In workers operating grinders, higher oscillations in the heart minute volume were observed than in workers not exposed to noise. Exposed workers wearing anti-noise ear protectors experienced a decrease in heart minute volume similar to the level in controls.	An attractive design in that it makes pre- and post measurements on an exposed and comparison group. However, the relationship of heart minute volume to disease is not known.

Table 3-14: Delin (1984).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Delin, C.-O.; 1984.	Cross-sectional; with 8 yr. followup. Industry (Engine rooms of Scandinavian Ferry Lanes); Sweden.	112 workers in engine and control rooms.	Engine noise = 100-115 dB; Control room = 75-80 dB. Severe hearing loss = 65 dB loss at 3k, 4k or 6k Hz. Normal hearing = less than 20 dB loss at all frequencies. Exposure duration: time in work for normal hearing 10-28 yrs.; for impaired hearing 23-49 yrs.; workers followed 8 yrs. and exposure said to be constant.	BP -- hypertension defined as ≥ 160 mm Hg. systolic or 100 mm Hg. diastolic; BP measured every second yr.; No measurement information given.

Table 3-14: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Author states age and weight taken into account, but appears to be examined separately. No data as to attrition.	Data not provided in form to calculate prevalence ratios. T-tests and Chi-square tests used.	"In 1983 (and earlier years) there was no significant difference between those with impaired hearing and those with normal hearing with regard to systolic and diastolic pressure or frequency of hypertension...Between 1976 and 1983 no new cases of hypertension were recorded. Two men acquired severe hearing impairment."	Failed to show data for the 112 original group by high and low noise. Conclusions clearly stated but impossible to determine temporal relationship from data presented. Sample size small.

Table 3-15: Demeter et al. (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Demeter, I.; Drasoveanu, C.; Cherestes, I.; Kertesz, I.; Demeter, E.; 1979.	Cross-sectional. Industry (Coal mining); Rumania.	100 male coal miners, mean age 45 yrs; divided into groups by hearing loss. 40 workers had extensive examinations for CV risk profile.	Uncontrolled coal mining noise; 90-105 dB; no additional information given; exposure duration by employment from 1-13 years. Audiometric exam performed, then grouped into normal hearing, incipient sonic trauma and advanced hearing loss.	Sublingual vessel morphology for determining arteriosclerosis; cholesterol levels and blood pressure.

Table 3-15: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias possible; controlled on age by stratification into < 40 or > 40 yrs. of age; unclear as to how family hx., smoking hx., ponderal index, hypertension were handled in analysis for vessel morphology.	No data presented as to prevalence of disease in the 2 groups. Feinstein's set theory and Venn diagrams used.	"The noise plays an inducing role in arteriosclerosis. The arteriosclerosis favors the occurrence of sonic trauma. The occurrence of sonic trauma is earlier in subjects over 40 years of age exposed to noise."	Study contains elements for a good analysis examining effects of noise while controlling for confounders. However, noise is poorly documented and data not examined extensively. These correlational data do not warrant the strong inference reflected by the authors' conclusions.

Table 3-16: van Dijk et al. (1987a).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Dijk, F. J. H. van; Verbeek J.H.A.M.; Fries [sic] F.F. de; 1987.	Cross-sectional. Industry; ship building and machine shops of the Dutch Royal Navy; The Netherlands.	257 workers; 162 in high noise of ship building; 95 in lower noise of machine shops.	Ship building and machine noise. In ship building, med. LA eq. = 98.0 dBA; in machine shop, 85.5 dBA. Dosimetry on a representative sample of 54 during a shift of 8 h with type Genrad 1954 (specif. ANSI SL 4-1971 dosimeters). Area monitored with calibrated B & K sound level meters, type 2204 and 2219. Otoscopic and audiometric exam (Peekel D77) in sound isolated cabin after 15 min removal from noise. Hearing: loss at 3, 4, 6 K Hz for either ear after presbycusis -- correction (Spoor). Workers with ear pathology excluded.	Casual blood pressure measured once in right arm with (blind) London School of Hygiene manometer while sitting, after 10 min rest.

Table 3-16: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
6% non-response equally distributed over groups; 74% shipbuilding workers used hearing protection compared to 14% of machine workers; selection into departments possible; BP corrected for age and weight (Quetlet). Although not controlled, groups were similar in use of medications, alcohol, and tobacco and in number reporting nervousness.	No differences in corrected mean BP; Student's t-tests used. Prevalence of hearing loss over 40 dB was 39% in shipbuilding compared to 9% in machine shop for a ratio of 4.3. Chi-square tests employed.	"This field study did not show any relation between exposure to noise levels above 80 dBA and blood pressure. The possibility cannot be excluded that the cross-sectional design obscured such a relationship." "In the machine shop, however, a positive correlation was found between noise-induced hearing loss and diastolic blood pressure after correction for age and relative weight ($r = 0.22$, $p < .05$, $n = 88$). "Prevalence of noise annoyance was 1.5 to 2 times higher in shipbuilding than in machine shop, but there was only a weak positive relation between noise annoyance and the duration of exposure in S; no relation was observed in M. This suggests the absence of substantial habituation to noise."	Fairly well-designed cross-sectional study. Inferences limited by lack of information on temporal relationship of the variables.

Table 3-17: van Dijk et al. (1987b).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Dijk, F.J.H. van; Souman, A. M.; de Vries, F. F.; (vi) 1987.	Cross-sectional. Industry; Production departments of 7 industries, 3 metal plants, 1 cattle feed, 1 household articles, 1 plastics, and 1 chemical plant. The Netherlands.	539 males: 21% < 25 yrs. old; 31% 25-34 yrs.; 33% 35-39 yrs.; 9% 40-54 yrs.; 6% 55-65 yrs. of age. Personal noise exposure levels (L_{eq}): 13% < 86 dBA; 28% = 86-90 dBA; 33% = 91-95 dBA; 25% > 95 dBA.	L_{eq} per 8 h shift measured by calibrated personal dosimeters with a short time constant. Three measures of total noise exposure based on L_{eq} and duration: $EE = (L_{eq}, 8 h) + 3.7 + 10 \log T/T_o$ for 274 workers without previous exposure; $ND =$ measured present noise level score X number of yrs. exposed for 274 workers where L_{eq} : < 80 dBA = 1; 81-85 dBA = 2; 86-90 dBA = 3; 91-95 dBA = 4; 95 dBA = 5. $ED =$ estimated noise level scores X number of yrs. exposed where no or little noise = 1; moderate = 2; rather much = 3; much = 4; and very much noise = 5. Previous and recent area noise levels available for all industries; noise levels had not changed over last decade; median duration of present jobs = 6.2 yrs. Air conduction, pure tone audiometry in sound-isolated cabins at least 15 min without noise and Otoscopic exams performed.	Measurement of casual BP with semi-automatic Waterpik Professional sphygmomanometer (automatically converts 1st and 5th Korotkoff tones) on the right arm in a sitting position after 20 min rest. BP measured 3 times; average of last 2 readings used. Questionnaire of subjective working conditions and symptoms and measurement of height and weight obtained.

Table 3-17: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
27% non-response due to absenteeism and language problems; hearing protectors used by 33%, more so by those exposed to high noise levels; annoyed workers used protectors more frequently than the less annoyed. Data apparently corrected "as much as possible" for age, use of hearing protection and other working conditions. Degree of selection bias into jobs unknown.	Multiple regression, Pearson Product-Moment Correlations were used in analysis. BP and noise exposure graphed with no r given. Hearing loss was significantly correlated with total noise exposure ($p < .05$) EE $r = 0.26$ ND $r = 0.30$; ED $r = 0.36$.	"In this study no relation was observed between total exposure to noise (EE, ND, ED) and blood pressure. A positive relationship between total exposure and hearing loss was firmly established." "Not expected and difficult to explain was the significant but weak, negative association between stress-index and noise annoyance with the corrected systolic blood pressure ($r = -0.15$ and $r = -0.19$, $p = 0.5$, $n = 405$)."	Data explored rather extensively for potential confounders but apparently did not lend itself to simultaneous control of all potential confounders nor to examinations of interactions as indicated by authors. Although sample size was relatively large, the populations were heterogeneous. Amount of reduction of noise to the ear by hearing protectors is not known.

Table 3-18: Folprechtova-Stenzlova and Janicek (1966).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Folprechtova-Stenzlova, A.; Janicek, M.; 1966.	Cross-sectional. Industry, (Foundry); Czechoslovakia.	94 1 male workers classified by noise area.	Foundry noise: Group A: 74.8 to 95.1 dB; average = 92.3 dB (Lin); Group B: 103-124 dB; average = 107.9 dB (Lin). Subjects are classified by exposure: < 1 yr.; 1-5 yrs.; 5-10 yrs.; > 10 yrs. employed. Noise (acoustic stress) measured with SLM, type B&K 2203 and B&K Octave Filter type 1613.	Blood pressure; lowest value of 3 readings used; taken in sitting position, on right arm, toward end of workshift, by Korotkov method; one observer.

Table 3-18: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential bias of non-response, selective survival, selective recall. Multiple variables available for analysis: age < and >40 yrs.; number of yrs. employed; work shift; smoking; number of children; time commuting to work; weight; sleep; salt intake; diet (fat, veg., fruits); amount of fluids consumed daily. Apparently only age was taken into account when single indicators were evaluated.	Effect parameters not stated. Analysis based on student's t-tests.	"Different levels of acoustic stress do not affect the level of BP if exposure (duration) and schedule are kept constant. If different exposure lengths and schedules are introduced, workers with a longer exposure do have a higher BP, and workers with a 2 or more shifts schedule do have a lower average BP." (Findings not consistent across ages.) "Our results seem to suggest that it may be next to impossible to evaluate the particular factors in isolation, and that results should always be seen as the outcome of the working and living environment as a whole. The difference between the two age groups can be explained by the effects of age itself or by the different time factors, combined with other particular causes."	Strength of the study is in its potential for considering multiple confounders such as weight and smoking. Unfortunately, only age was considered and it was poorly controlled. Further multivariate analysis of the data would be informative.

Table 3-19: Friedlander et al. (undated).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Friedlander, B.; Grebermann, M.; Wathen, G.; Zeidler, W.; (undated).	Historical-prospective; (incomplete cohort-five year exposure). Industry; Curtis Bay shipyard; U.S.	441 males categorized by age and race and by high, medium, and low noise exposure; no distributions given. Group 1 defined as men having BP reading within one year of initial employment; Group 2: all others.	Shipyard machine shop noise measured by Safety Dept. Noise exposure levels defined as: Low = < 70 dB Intermed. = 70-79 dB; High = > 80 dB. Duration of exposure: all men had been employed at least 5 years.	Blood pressure defined as: elevated systolic 140 mm Hg and elevated diastolic 90 mm Hg. Data from clinic medical records; no additional information given.

Table 3-19: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Incomplete identification and follow-up of a cohort; small and highly selective sample; comparability of low and high noise groups questionable; measurement bias from using extant medical record data.	Sample stratified on race and age. No evidence of controlling for any other variables. Relative risks for race/age groups: For Group 1, RR = 2.76 for DBP and 6.36 for SBP -- high noise compared to low noise exposure. Chi-square with Yates correction applied to data.	"The data presented indicates that with prolonged exposure to loud noise of an intermittent nature there is a trend towards both elevated systolic and diastolic pressure when compared to a control population exposed to relatively low-noise intensity; that the relative risk for developing hypertension is greater in those exposed to prolonged loud noise than in controls matched for age and race."	This ostensibly historical prospective study suffers from severe methodological problems -- cohorts incompletely defined and followed; sample size small and highly selective; variables inadequately measured and analysis incomplete." Senior author indicates this was a pilot project conducted by a group of summer students and should be considered as just that.

Table 3-20: Geller et al. (1963).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Geller, L. I.; Sakaeva, S. A.; Musina, S. S.; Kogan, I. A. D.; Belomytseva, L.; Ostrovskaya, R.; Volokhov, I. A. P.; Luk'ianova, E.; Popova, R. M.; Moskatel'nikova, E.; 1963.	Cross-sectional. Industry (Oil); Russia.	1482 workers in oil gases; 366 in oil gases and high noise compared to 263 administrative; 456 physical work.	High frequency 2400-6000 Hz; levels 115-125 dB.	Hypotension defined as < 99 SBP or <69 DBP; hypertension and cardiovascular neurosis not defined. No other data given.

Table 3-20: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection, selective survival. Stratified by sex and age (age < 40 or > 40).	Not specified in paper.	"In persons exposed to the effect of persistent high noise, arterial hypotension is less common and hypertension much more common than in workers whose work conditions are not connected with the influence of noise. Cardiovascular neurosis is found in a higher number of cases than in employees performing other physical or administrative duties."	A weak study due to inadequate information on both noise exposure and health outcomes.

Table 3-21: Gel'tishcheva (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Gel'tishcheva, E. A.; 1980.	Cross-sectional; measure during one day and one week and at end of one year. Industry (Watch assembly); Russia.	36 adolescents: 16-18 yrs. old.; 11 trainees: 16-17 yrs. old. Healthy.	Exceeded by 1-4 dB the maximum spectrum (MS) 65, but was not higher than P.S. 70; employed 1-3 yrs. Workers and trainees exposed to similar noise, differed on production stress.	SBP, DBP, pulse rate, EKG, no measurement procedures given.

Table 3-21: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; bias due to changes in the work environment during the year.	Statistical significance stated, but no tests given.	"The intensive delicate visual work performed under exposure to occupational noise not exceeding MS 70 (75 dB) shows an adverse effect on the functional state of the cardiovascular system in adolescents. The changes of the functional state of the cardiovascular system in the process of occupational activity are more pronounced in the adolescent workers than in the students of VTS."	No apparent comparison group, small sample size and poor specification of noise and CV parameters.

Table 3-22: Gibbons et al. (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Gibbons, S. L.; Lewis, A. B.; Lord, P.; 1975.	Quasi-experimental; subjects as own controls. Oil tankers; England.	Officers on 4 tankers.	NVR values (dB) ranged from 45 to 96; ships ranked by NVR level.	24 h. urinary 17 ketosteroid levels and urinary volume.

Table 3-22: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No data other than annoyance as possible confounding variables; volunteer bias; no data on comparability of subjects; small sample.	Non-parametric, Wilcoxon matched-pairs test.	Decrease in level of 17- ketosteroids and urinary volume while men were serving at sea compared to mean values obtained when men were on leave. Control values were in range of reported values of workers not exposed to noise suggesting officers recover "while on leave."	Since randomization of subjects to test and control situations is unlikely, findings are of limited value.

Table 3-23: Graff et al. (1968).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Graff, V.; Bockmuhl, F.; Tietze, V.; 1968.	Cross-sectional. Industry (Boiler Plant); Germany.	117 exposed to noise; 50 workers in same plant without noise males without history of hypertension; both groups of same age distribution.	Group of locksmiths, blacksmiths, welders, tube-benders, transportation workers and crane drivers exposed to 90-110 dBA noise of medium to high frequencies. No noise exposure data given for controls of transportation workers without noise strain.	Increase in BP classified into 4 grades according to WHO standards beginning with SBP 150 mm Hg and DBP of 95 mm Hg. Numerous other parameters such as EKG, EEG, clinical examination assessed but not defined. Men with history of hypertension excluded.

Table 3-23: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and selective survival bias; failure to use controls in comparisons for most of analyses; no evidence of control for confounding factors.	Data presented in graph format; no statistical tests described.	A larger proportion of hypertensive patients and patients with other heart and blood circulation sickness were found among the noise exposed group than among the workers without noise strain. Within the noise exposed group, 25-35 year olds, locksmiths and blacksmiths and individuals exposed at least 8 years were most likely to present with hypertension and other circulatory problems. "In the patients with other heart and blood circulation disorders and in the group without pathological findings, low grade, medium and high grade hard-of-hearing persons were found, but the number of normal hearing persons was lowest in the group of persons with high blood pressure."	Major methodological problems prohibit the use of this study in judging causality.

Table 3-24: Grusha (1974).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Grusha, A. N.; 1974.	Cross-sectional; patient series. Industry (Unknown, neurological unit, Chernikhov Regional Hospital); Russia.	134 noise-exposed patients with neurosis-like states; 100 patients with closed craniocerebral injuries; 110 patients with infectious arachnoencephalitis.	134 patients with neurosis like states "as a result of a long-term effect of high frequency industrial noise". No further details given.	Brachial arterial pressure measured by Korotkov-Ianovskii method; temporal pressure by Markelov method; central retinal arterial pressure (CRA) by Baiiar method. No details given.

Table 3-24: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Apparently no control group for comparison; no evidence of controlling for unequal sex distributions of groups; no age data provided; potential selection bias.	None stated in paper.	Of patients (134) with neurosis-like states from long-term effects of industrial noise, a greater proportion suffered from temporal arterial hypertension and central retinal arterial hypertension than from brachial arterial hypertension.	Study is of questionable etiologic relevance because of poor specification of the variables and the apparent failure to use a control group.

Table 3-25: Hannankari et al. (1978).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Hannankari, I.; Jaarvinen, E.; Partanen, T.; 1978.	Cross-sectional and historical/prospective with sub-groups. Industry (Railway workers); Finland.	437 locomotive engineers exposed to railroad noise; 1575 trainmen; 1224 railroad clerks as reference group.	Intensity: 45% of the measured noise levels exceeded 85 dBA during 0.5-2 h. measuring time period. Exposure from Dec. 1955 to Dec. 1973. In followup analysis all engineers, every 2nd trainman and all railroad clerks employed on Dec. 1, 1955 were included.	Reported on questionnaire any diagnosed hypertension, M.I.; mortality data from circulatory diseases.

Table 3-25: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential confounders such as age, previous exposure, hearing loss, etc. not statistically controlled in the analysis.	None stated in paper.	Cross-sectional data showed no significant findings on CVD. "The evidence on the relatively high risk of disease of the circulatory system and tumors of the engineers during the follow-up period was, as summarized, well established...According to the results of the study, technical improvements which lower noise and vibration (are recommended)."	Although this mortality analysis is suggestive of an association between noise and CVD, further analysis controlling for confounders is required for its acceptance as a valid study.

Table 3-26: Idzior-Walus (1987).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Idzior-Walus, B.; 1987	Cross-sectional Industrial plant, Poland.	481 men (8 refused) exposed to vibration and noise; 303 controls from same factory, similar on age, sex, place of residence, socio-economic class and occupational physical activity.	Exposure from pneumatic hammers used in riveting, for minimum of 4 h daily. Vibration in frequency band 32-64-125 Hz with total range of 32-2000 Hz. Noise reached 105-116 dB. Exposure period not shorter than 2 yrs with mean of 15.1 years.	Exam included standardized questionnaire, wt., ht., 12 lead resting ECG coded according to Minnesota code, fasting serum cholesterol, BP taken by staff trained from London School of Hygiene tapes, diagnoses of effort angina and intermittent claudication based on Rose questionnaire. Lab methods given; coders trained. Systolic hypertension defined as SBP > 159 mm Hg and diastolic hypertension as DBP > 94 mm Hg.

Table 3-26: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Health exam for exposed conducted by author, controls examined by author and a group of trained students; observer-detection bias may exist. Major risk factors for CHD controlled.	Z-tests, analysis of covariance and Dunnet test applied. Prevalence of systolic hypertension in exposed 7.3% compared to 2.0% in controls (PR= 3.65); Prevalence ratio for DBP was 1.82, sign. at .01 level.	Incomplete right bundle branch block was observed in about 10% of exposed workers (nearly 4 times as commonly as in the controls). "Mean blood pressure values and the percentage with hypertension were significantly higher in the exposed than in the reference group (P=0.01)." ... "Clustering of two of the main coronary risk factors (i.e. hypercholesterolemia, hypertension and cigarette smoking) is significantly higher in the exposed than in the control group (P<0.05)	This is one of only a few studies which have reported vibration exposure levels as well as noise. Cross sectional data would be more helpful if noise without vibration group had also been included.

Table 3-27: Ising et al. (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Ising, H.; Gunther, T.; Havestadt, C.; Krause, Ch.; Markert, B.; Melcher, H. U.; Schoknecht, G.; Theseld, W.; Tietz, K. W.; 1979.	Cross-sectional and intervention (with and without hearing protectors). Industry, (Brewery); Germany.	90 volunteers; 36 working in noisy areas & 54 in areas of less noise; controls studied for 1 day; 18 noise-exposed workers examined for 2 days and 12 workers examined for 2 weeks while wearing hearing protectors half the time.	Noise levels given for each job in 6 work environments. All workers wore individual dosimeters: bottling cellar workers = mean of 95 dBA ± 0.7 dB; Control group = mean of 82 dBA ± 1.2 dB.	Blood pressure taken with semi-automatic device; subject sitting, at end of shift, mean of 4 BP readings taken as measured value. Blood and urine tests; epinephrine, norepinephrine, cholesterol, magnesium, etc.

Table 3-27: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection; possible Type II error due to small sample size; no statistical controlling on age, sex, weight, smoking.	No data given for comparison of noise workers and control workers. T-test used.	"When working without ear defenders at a mean exposure to noise of 95 dBA, the systolic blood pressure was higher by almost 7 mm Hg ($p = 0.001$)... ...The actual daily average noise level reduction of this device was 13 dB...The evaluation of the parameters of 26 test persons showed a negative correlation of $c = -0.52$ ($* = 0.003$) between the magnesium content of blood sediment and the increase in blood pressure when exposed to noise."... "The comparison of noise workers and a control worker group, however, indicated no (statistically) significant difference"... "Only the examination of the same test subject under two or more different noise stress conditions is suitable for proving the effects of noise with statistical significance."	A useful pilot study since noise and health parameters were specified in detail. Not definitive for drawing inferences due to small sample size and inadequate control group.

Table 3-28: Jansen (1959).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Jansen, G.; 1959.	Cross-sectional. Industry, (Smelting); Germany.	669 workers from high noise areas; 336 workers from low noise areas; average age 42 yrs.; response rate high.	Actual noise levels not reported; 34 occupations listed. Workers divided into 4 groups: Groups I & II: Noise < 90 dB; Groups III & IV: Noise > 90 dB. Subjects had worked at least 3 yrs. under same conditions.	Symptoms of vascular and cardiac problems. By medical exam: tachycardia, extrasystoles and blood pressure readings. No information on diagnostic criteria or measurement procedures.

Table 3-28: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; measurement bias; groups described on personal, economic, living, working conditions and family life, but no evidence of control in analysis.	Measure of association not stated; Chi-square tests employed.	"Vascular disturbances in the extremities, skin findings (pale-ness) and cardiac findings (rhythm disturbances, tachy-cardia, extrasystoles) occur statistically significantly more frequently in subjects exposed to high noise levels." "Thus, all this would prove is that vascular disturbances, skin findings, and cardiac findings as indicated in workers exposed to noise are caused by noise and thus could be designated as 'noise determined' symptoms or findings. The proof can be based on previous results of experimental noise research."	Although a large sample study, inferences are difficult because noise and CVD were poorly documented by the data and bias including confounding likely.

Table 3-29: Jansen (1961).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Jansen, G.; 1961.	Cross-sectional. Industry, (Smelting); Germany.	669 workers from high noise areas; 339 workers from low noise areas; average age of 42 yrs.; no chronic diseases.	High noise group exposed to more than 90 dB(B); low noise group exposed in noise not less than 65 dB(B); average length of employment 11 years.	Symptoms of vascular and cardiac problems; tachycardia; extra-systoles.

Table 3-29: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and measurement bias; no variables controlled in analysis.	Magnitude of association not stated; statistical tests not specified.	"The total result of the examination showed that no defined noise disease could be determined, with the exception of hearing damage."..."The vegetative disturbance is stronger than had been suspected; it occurs especially with wideband noises and can be detected in persons at rest and engaged in physical activity. A comparative examination of 1005 persons employed in the smelting industry confirmed these findings."	Contributes little to understanding of noise effects.

Table 3-30: Jirkova and Kremarova (1965).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Jirkova, H.; Kremarova, B.; 1965.	Cross-sectional. Industry, (Large engineering factories); Czechoslovakia.	34 noisy workplaces -- 766 men and 203 women. 6 not-noisy workplaces 371 men and 318 women.	Noisy areas ranged from 85-115 dB for greater part of the day with noise considered disturbing; not-noisy areas had dB levels of < 70 and noise not disturbing.	Medical record data indicating hypertension, subjective complaints, absences from work due to illness. No diagnostic criteria or definitions reported. Data collected by plant physicians with no standard procedures.

Table 3-30: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias probable; stratified by sex. Age partially controlled. No other confounders considered in the analysis.	Not stated; Chi-square tests employed.	"The results of the comparison show a higher occurrence of hearing damage, subjective complaints, hypertension, and peptic ulceration in persons exposed to noise." On the other hand, the authors did not show that the frequency of the diseases investigated, with the exception of hearing damage, depended on the length of employment. Findings were not consistent for men and women.	Adds no new information. Inadequate study description and poor control for confounding.

Table 3-31: Jonsson and Hansson (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Jonsson, A.; Hansson, L.; 1977.	Cross-sectional. Industry; Sweden.	196 males; 44 with noise-induced hearing loss; 74 with normal hearing; 78 dropped.	Surrogate measure: noise induced loss of acuity to 65 dB or more at 3000, 4000, 6000 Hz; assumed repeated and prolonged exposure.	1 measure of BP, supine, after 15 min rest; hypertension -- 160/100. No quality control of observers.

Table 3-31: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Groups compared as to mean age; no controlling for age, anti-hypertensive medications, history of disease, etc.	Prevalence ratio for hypertension = 2.80 (impaired to normal hearing acuity. Student t-tests for paired observations.	BP and hypertension was "significantly higher in workers with noise-induced hearing loss" than those with normal hearing.	Frequently quoted study supporting an association between noise and hypertension but is based on cross-sectional data and a surrogate measure of noise exposure.

Table 3-32: Kachnyi (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kachnyi, G. G.; 1977.	Cross-sectional. Industry, (Cloth combine plant); Russia.	591 female weavers from 15-27 years of age divided into 2 groups by noise levels and 20 healthy controls working in administration.	High frequency noise from looms. Group 1: 106-108 dB; Group 2: 102 dB; Duration of employment: Group 1: 17 for 3-6 mos.; 172 for 1-5 yrs.; 111 for 6-10 yrs.; Group 2: 18 for 3-6 mos.; 164 for 1-5 yrs.; 109 for 6-10 yrs.	Arterial and temporal BP. Arterial taken with Mercury manometer, on both arms, middle of 1st shift of last of a 5 day work week; hypertension $\geq 140/90$ mm Hg; hypotension $\leq 100/55$ mm Hg. No information regarding quality control of measurement.

Table 3-32: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Controlled for other industrial noise exposures by selecting women with 1-2 years work on collective farms or in quiet offices.	Proportions compared but no inferential statistics used.	"Hypotension was more frequently observed with a shorter length of employment." 64.7% hypotensives were noted among weavers in Group 1 and 50% among Group 2 with 3-6 months employment. "Our data confirm the opinion of M. M. Pokrovskii (1968) that a lowering of the arterial pressure occurs frequently in young persons." The number of hypertensives increased with the length of employment. Deviations of the value of the temporal-brachial coefficient from the norm were not observed.	Study of limited usefulness due to lack of adequate control group and failure to control for other factors. Meaning of hypotension as a health effect is unclear.

Table 3-33: Kalicinski et al. (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kalicinski, A.; Straczkowski, W.; Nowak, W.; Proniewska, W.; Ro'zan'ska, T.; 1975.	Cross-sectional. Industry, (Spinning and weaving); Poland.	140 women 45-51 years of age; mean age = 49; compared by years of work in noise: Group I = 31 workers; Group II = 48 workers; Group III = 61 workers.	Steady noise from spinning and weaving industry at frequencies between 32 and 16,000 Hz with intensities 95-105 dB; women grouped by time worked in noise: Group I = 1-6 years; Group II = 7-12 years; Group III = ≥ 13 years worked.	BP taken after 30 min rest; hypertension defined as SBP > 140 mm Hg and DBP > 90 mm Hg. Cardiac ischemia defined by S-T depression > 0.1 mV on a 12 lead EKG taken at rest.

Table 3-33: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Authors state some evidence of selection in the degree of appearance of coronary complaints, some women changed or quit their jobs and so escaped the study.	Hypertension prevalence ratio for Group III (high duration) to Group I (low duration) = 1.8; for Group II to Group I = 1.5; for Group III to Group II = 1.3. Chi-squared tests used.	The frequency of hypertension is significantly greater, the longer the occupational exposure to noise. There were no significant differences between subject groups as to "tendency toward inadequate blood supply" in the EKG... The frequency of coronary disease in women with hypertension was 6 times greater than in persons with normal blood pressure. "The studies show that many years of work under noise significantly affect the frequency of the occurrence of arterial hypertension and symptoms of inadequate blood supply of the heart muscles in the EKG. The frequency of these disturbances is greater the longer the occupational exposure to noise."	Study of limited usefulness because so little information is provided about sampling, measurement and statistical analysis.

Table 3-34: Kanevskaia et al. (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kanevskaia, Zh. S.; Maksimova, L. I.; Kublanova, P. S.; Shevyreva, N. A.; Sineva, E. L.; Markova, T. S.; 1977.	Cross-sectional. Industry, Russia.	Males and females (35-39%) aged 25-45 years. Group 1 = 256 workers exposed to stable noise; Group 2 = 284 exposed to pulsed noise; Group 3 = controls not exposed to noise exceeding the Maximum Permissible Level. No vibration in work area.	Group 1 = 90-100 dBA steady noise; Group 2 = 107-117 dBA pulsed noise; Group 3 = levels not given; 60% of workers employed $\geq$ 10 yrs. No information on sources, instrumentation measurement or subjects.	Blood pressure; urine adrenaline and noradrenalin. No information provided on diagnostic criteria or measurement. (Other measures were symptoms, EEG responses, hearing loss, skin vibrational sensitivity.)

Table 3-34: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and measurement bias possible. No evidence of statistically controlling for any variable.	No data are provided.	"Therefore, the continuous exposure of the organism of the worker to industrial noise can be compared to chronic acoustic stress capable of causing various functional cerebral-visceral disorders." ... "It can be assumed that pulsed noise as well as stable noise provokes the stimulation of the adrenaline ring."	Study provides essentially no data for scientific scrutiny.

Table 3-35: Kangelari et al. (1966).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kangelari, S.; Abramovich-Poliakov, D.; Rudenko, V.; 1966.	Cross-sectional. Industry (Motor-testing shop & foundry); Russia.	135 motor mechanics; 152 cleaners; males, aged 20-39 yrs. 30.8% employed ≥ 3 yrs.; 17.3% up to 5 yrs.; and 51.9% employed ≥ 5 yrs. Unstated number in control group of fitters.	Motor mechanics = 116-120 dB; Fitters = 88-90 dB; Cleaners = 100-120 dB.	Sickness rate = cases per 100 based on temporary work disability for 3 yrs. Angina stated as a specific form of illness, but no diagnostic criteria stated. Diseases of nasopharynx, gastritis, ulcers, and myositis also studied.

Table 3-35: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and measurement bias; no evidence of statistically adjusting for confounding variables.	Not stated in paper.	Angina sick rate 8.9 in motor mechanics and 7.8 (cases per 100) in fitters. "The indexes of the sick rate for the cleaners suffering from vibration illness are higher for every disease than for the cleaners not suffering from vibration illness."	Study is weak in design and totally lacking in control of potential confounders. CVD diagnostic entity, angina, is very subjectively defined.

Table 3-36: Kavoussi (1973).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kavoussi, N.; 1973.	Cross-sectional. Industry, Silo; Iran.	465 males.	Not measured, but "workers could not ever understand each other when speaking." Employment: 10 years; 11-25 years; over 25 years.	Average of 3 BP readings using a portable Vaquez apparatus. BP taken in a.m. before work, after being seated 1 h and resting supine for 5 min. Hypertension defined as 140/90 or greater. No information about data collectors or reliability of measurements.

Table 3-36: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No controls, no data as to previous or concurrent noise exposure, health conditions, hearing thresholds, ear disease, etc. 235 or 34% of workers not included because they "were in administrative positions or absent." Workers stratified by age (<39, 40-54, 55-64). Labor turnover during the 34 year operation of the plant was "very low."	Calculated prevalence ratio for hypertension = 2.3 (exposure > 25 years to exposure 11-25 years). No statistical tests given.	1) There is no case of abnormally high BP in men up to age 39. "This means that the length of exposure to a noisy environment does not induce high BP in men under 40 years of age." 2) "After 40 years of age when there is a greater tendency to develop high BP...the length of exposure to a noisy environment can be an additional factor in inducing this condition." 3) In older age group "there is greater incidence of high BP because age and length of exposure are greater than in the previous categories."	Study contributes little to assessing long-term CV effects of noise since noise per se is not measured, there is no control group of low noise exposure and potentially confounding variables are not controlled.

Table 3-37: Khomulo et al. (1967).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Khomulo, P.; Rodionova, L.; Rusinova, A.; 1967.	Cross-sectional. Industry, (Type not given); Russia.	69 men and 34 women working under high noise; 38 men and 13 women served as controls (low noise) observed over the course of 7 years.	Noise exposed group: 117 dB, high frequency; Control groups: 60-95 dB low to medium frequency noise. Subjects grouped by yrs. employed: 1-2; 3-4; 5-9; 10-14; 15-30.	Blood serum cholesterol; B-lipoprotein; neurocirculatory asthenia; hypertension; arteriosclerosis. No diagnostic criteria given.

Table 3-37: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; age, length of employment, specialization, and state of the nervous system were controlled in analysis. Nutrition controlled on day of blood testing.	Effect parameters not stated. P-values given, but statistical tests not described.	"Industrial noise with an intensity of 95-117 dB acting during the course of 5 years or longer leads to impairment of the metabolism of lipids in workers. The disorders are manifested as hypercholesterolemia and a tendency toward an increased quantity of total lipids and beta-lipoproteins in blood. The degree of increase in the cholesterol content in the blood depends on the length of employment in conditions of intense industrial noise. After 5-15 years of exposure to industrial noise, endogenous hypercholesterolemia, more pronounced in persons with functional disorders of the nervous system, developed in the majority of the workers."	Weak study methodologically. Appears to lack a control group and noise levels not measured.

Table 3-38: Kobets (1972).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Kobets, G. P.; 1972.	Cross-sectional. Industry, Russia.	Multiple groups: 446 women employed in warehouse & storage work; 390 practically healthy workers; 147 female concrete workers; 144 women subject to the effect of noise; 89 persons suffering from noise disease; similar on mean ages with average length of employment from 7 to 14 years.	Noise sources not specified; noise parameters presented for women subject to noise and suffering from noise diseases: 112-122 dB predominately in 1200-1800 Hz range.	Disorders of blood pressure; functional disorders of the heart; temporary work disability.

Table 3-38: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; measurement bias; no evidence of controlling for confounders.	No data provided to confirm statement that disorders in the regulation of the arterial BP, in the direction of higher as well as lower were 5-8 times more frequent among those subject to vibration and noise than in the control group. No statistical methods stated.	"...specific disease and disorders (arterial hypertension or hypotension, functional disorders of heart action, neurosis) are consequences to a certain degree of the specific effects of the named hazards..." The index of cases of the sick rate (total data) was lowest in the control group, rose 2.5 times in workers subject to the effect of noise, increased 3.9 times with development of noise illness, 3.2 times in healthy concrete workers and 3.9 times in individuals with vibration disease.	Study findings of little usefulness since direction of associations between BP and noise levels not clear and groups under investigation were not clearly defined by noise and health outcome level.

Table 3-39: Konarska (1983).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Konarska, M.; 1983.	Longitudinal (measures taken on same person 3 times a year). Industry; Poland.	93 workers: 66 operators of industrial washers, 36 women and 30 men; 21-61 yrs. of age; control group of 27 non-exposed persons age 23-59 years.	Noise exposure: intensity 95-111 dB in middle frequencies of 10 and 20 KHz. No measurement information provided. Audiometric results were the same for exposed and control groups.	Exam by physicians. EKG -- no data provided. Blood samples for biochemical exam (glucose, free fatty acids) taken 3 times a shift at 10 a.m., 1 p.m., and 3 p.m. after a 400 and 300 Kcal breakfast at 8 a.m. and 11 a.m. No quality control data provided.

Table 3-39: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Appears to be a convenient group of workers followed with no baseline data. Age considered in analysis. Insufficient data to judge confounding.	Data presented graphically. Statistical significance stated, but tests not described.	"It was found that the glucose level in exposed workers was the same as of the control group. Dynamics of changes during the working day were similar, too. The contents of total fatty acids in exposed group was slightly lower than 10% of the control group. A statistically significant decrease of the level of free fatty acids was stated in the exposed workers."	Laboratory and study design methods described insufficiently to evaluate validity of the study.

Table 3-40: Lanzetta et al. (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Lanzetta, T.; Giovanazzi, A.; Furlanello, F.; 1979.	Cross-sectional (Serial EKG measurements). Industry (Type not given); Italy.	12 workers exposed to noise in two work areas; 5 controls, not exposed to noise and of normal hearing. Sex and age of workers not stated. In exposed group, 8 had normal hearing and 4 were hearing impaired.	One work area = 91 dBA; another work area = 101 dBA; 4 1/2 h exposure to usual work noise on the day of testing with 2 successive hours of rest. No other specific noise data given.	Holter (dynamic) monitoring of heart rate; arrhythmias, and ST-T differences (repolarization).

Table 3-40: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Multiple environmental and personal variables such as smoking monitored. Unclear as to how these were used in analysis; selection bias potential.	Effect parameters not directly relevant to long-term noise exposure. Serial data presented in graphic and table format with p-values but no statistical tests stated.	"The increase in cardiac rate was indeed constant in the groups 'exposed' with normal auditory capacity, was maintained throughout the work shift, and re-entered the normal limits only after cessation of work. The results were statistically significant in comparison with the group of workers 'not exposed' to noise and with those with hearing impairments. It is interesting to observe the persistence of the response in the increase in cardiac rate (under exposure) to environmental noise, even after many years of exposure to this noise."	This study of short-term EKG responses to noise may be of some use in judging long-term effects. However, the unclear methodology and small sample size reduce its scientific value.

Table 3-41: Lees and Roberts (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Lees, R. E. M.; Roberts, H. J.; 1979.	Cross-sectional. Industry; Canada.	62 with noise-induced hearing loss; 62 controls.	Hearing loss used as surrogate for noise exposure -- cases with hearing loss > 30 dB at 4000 Hz; control group of 62 drawn at random from quiet areas. Noisy areas = 95-98 dBA. Duration of exposure not given. Audiograms after 12 h out of noisy area.	BP after 7 min rest, taken "blind". Hypertension defined as $\geq 140/90$ mm Hg.

Table 3-41: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
(Stratified by age and random sample drawn) -- matched by age and duration of employment. No other variables considered.	Was not stated in paper.	"No evidence of a relation between increased SBP or DBP and hearing loss."	Small sample size and negative results suggest concerns for Type II error.

Table 3-42: Lees et al. (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Lees, R. E. M.; Smith, C. S.; Wetherall, L. D.; 1980.	Historical prospective, paired cohort. Industry; Canada.	70 pairs of workers.	High noise = > 90 dBA for a minimum of 3 consecutive years: low level < 85 dBA for total work history in plant. Duration: 8 h shifts, 30 pairs exposed 3-6 years. 22 pairs exposed 7-20 years. 18 pairs exposed 11-15 years.	Counts of new events of IHD, hypertension, M.I. No diagnostic criteria or baseline data shown.

Table 3-42: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
70 pairs matched on age, exposure period, duration of employment; 36 persons could not be matched; no attrition noted. No data on hearing thresholds. Work shift controlled in analysis.	Risk ratio for any disease category was approximately 2. Pearson correlation coefficients, Chi-square tests and one-way ANOVA employed.	"There were no significant differences between the 2 groups for both the exposure periods and total study period in incidence of new medical conditions."	A strength of this study is its historical prospective approach. Unfortunately the sample size is prohibitively small and matched pair analysis was not used although design was matched.

Table 3-43: Liubashevskaja and Solonin (1976).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Liubashevskaja, Z.A.; Solonin, I.U. G.; 1976.	Before-after improvements in work area; apparently not the same workers. Industry (Forging and pressing shops); Russia.	28 workers 22-54 yrs. of age before improvements; 30 workers after improvements.	Before improvements general noise level: 95 dB average and 78-120 dB ranges; after 3 yrs. improvement: 90 dB average and 71-110 dB range.	Blood pressure; no description and no data provided.

Table 3-43: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Apparently no confounders considered; potential for many internal validity and selection problems; apparently different group of workers before and after improvements.	Was not stated in report text.	Arterial pressure (systolic and diastolic) had a decreasing tendency at the end of the shift...Repeated physiological examination in 15 forge workers and 15 apprentices showed a decrease in the functional strain upon the organism....after noise reduction measures.	Apparently a poorly designed study with multiple changes over the 3 year period. Before/after comparisons appear to be more cross-sectional than longitudinal.

Table 3-44: Malchaire and Mullier (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Malchaire, J. B.; Mullier, M.; 1979.	Cross-sectional. Industry; Belgium.	1030 car assembly line; 581 wire mill workers; 501 controls. Males.	Intensity = 92 to 100 dBA for assembly line; 93 to 97 dBA for wire mill with average equivalent noise level = 95+ dBA; no information on dosimetry, duration of exposure. Audiometric tests performed over 3-4 year period.	BP taken by occup. MD. Criteria for determining DBP reading not stated; pre- or post-work shift not stated.

Table 3-44: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Control of age and hearing deficit by stratification – controls lacked data on hearing; excluded males with known CVD; all of similar social class.	Prevalence ratios not given. 27 Chi-square tests with 1 statistically significant.	No relationship between noise and BP observed.	Study design is weak for evaluating etiologic effects.

Table 3-45: Manninen and Aro (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Manninen, O.; Aro, S.; 1979.	Cross-sectional. Industry (Engineering factories); Finland.	188 males; 92 females classified by hearing levels.	No noise parameter data of engineering production line; surrogate measure, using the frequencies of 3000, 4000, 6000 Hz subjects classified: Class I: mild hearing loss, no more than 40 dB at any of 3 frequencies; Class II: moderate loss of hearing 45-60 dB; Class III: severe loss of hearing 60 dB or more. Noise intensity levels and duration of exposure not reported.	Casual BP reading, right arm, in sitting position, at rest; SBP recorded at second sound, DBP at Phase V; all readings by a nurse, apparently "blind" to hearing levels.

Table 3-45: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Mean BP given by sex, and age groups 26-40 and 41-65 years. Relative weight for sex and age group similar in different hearing classes. Excluded those on anti-hypertensive drugs, with chronic urinary infection or hearing impairment not due to noise damage.	Not stated quantitatively. Student's t-tests employed.	"--Results ... indicate that there is a relationship between BP and noise-induced hearing disorder. Prolonged exposure to industrial types of noise first elevates the SBP and to some extent the DBP as well. If the noise exposure continues, with the resulting severe hearing loss, the SBP tends to return to normal. DBP seems to either rise or fall. Obviously, prospective studies are needed to verify these conclusions."	Study poor relative to reporting of noise parameters. Possible confounders were not utilized to the maximum as continuous variables in the analysis.

Table 3-46: Mosskov and Ettema (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Mosskov, J. I.; Ettema, J. H.; 1977b (III).	Lab experiment with industrial noise. The Netherlands.	12 healthy males; 19-26 yrs. old; served as own controls.	Intensity: Leq 98 dBA. Taped noise through headphones in sound-proof room; sessions in random sequence of adaptation, rest, exposure, rest. Total duration of noise 30-40 s; highest intensity for 6-10 s. Duration: three 15 min periods exposure with 2 rest periods of 5 min	BP by cuff method, heart rate, pulse pressure, respiratory rate, sinusarrhythmia.

Table 3-46: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Random assignment of noise type used; no consideration of BP lability in small sample.	Was not stated in report text. Wilcoxin test used.	"Increase of DBP and of respiratory rate and decrease pulse pressure and heart rate observed."	The lack of precision on reporting parameters in this simulation study invalidates the authors' conclusions of a causal relationship between noise and cardiovascular disease.

Table 3-47: Ohrstrom and Bjorkman (1978).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Ohrstrom, E.; Bjorkman, M.; 1978.	Cross-sectional. Industry; Sweden.	75 in machine industry; 99 in 3 textile mills, working in noise 8 h a day; 49 workers exposed 4 h a day.	Machine shop: 70-80 dBA with peak to background 10 dBA; Textile mills: 70-80 dBA with peak to background 10 dBA; 80-90 dBA with peak to background 5 dBA; 90-100 dBA with peak to background 5 dBA.	Fatigue, vertigo, headache and other symptoms obtained by questionnaire.

Table 3-47: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No data on participation rate. No evidence of controlling for age, sex, duration of exposure although data were collected.	Not stated in report text.	"Fatigue increases with a higher noise level and a longer period of exposure." Headaches were somewhat less related to noise.	Not a useful study for assessing cardiovascular effects of high noise exposure.

Table 3-48: Paranko et al. (1974).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Paranko, N. M. Vyschipan, V.; Naumenko, B.; 1974.	Cross-sectional. Industry (Mining); Russia.	103 miners; 61 drillers; 42 sinkers. 2 mines studied; 62 males aged 30-40 years and 41 males aged 41-50 years.	Mine noise of high frequency; workers wore anti-noise helmets VTSN- 11-T-2M with noise estimates of 85 dB SPL based on previous measurements.	Arterial blood pressure with increase defined as: 130/90 mm Hg at ages <40 yrs.; 140/90 mm Hg at ages >40 yrs.; heart rate quickening defined as >80/min, slackening to <70/min

Table 3-48: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No controls or control group used; no statistical control for potential confounders such as age; selection bias, selective survival and selective recall possible.	Proportion of workers with high BP by years of employment. No statistical methods given.	"Thus, the results presented show that in miners exposed to the effect of vibration and noise at levels allowable by hygienic norms, stable functional shifts develop. However, their development occurs more slowly than in those exposed to the effect of vibration and noise of high parameters."	Fails to employ an adequate control group and to specify noise and health parameters in detail.

Table 3-49: Parvizpoor (1976).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Parvizpoor, D.; 1976.	Cross-sectional. Industry, Iran.	821 male weavers; 412 controls.	96 dBA in weaver group; no documentation of noise levels among controls.	1 BP, sitting after 5-10 min rest; quiet room; pre-shift.

Table 3-49: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Stratified by age; similar social class; no data on length of employment, sex of controls, previous noise exposure, etc.	Crude prevalence ratio for hypertension 4.13. Statistical tests not given.	Weavers had "significantly greater risk of developing hypertension" than controls; difference appears at "young ages (30-39) and increases with length of employment."	Study quoted often, but provides poor noise exposure data and inadequate control for potential confounding.

Table 3-50: Pilawska et al. (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Pilawska, H.; Mikulski, T.; Rosin, J.; Soroko, M.; Wysocki, K.; 1977.	Cross-sectional. Industry (Large shipyard). Poland.	1826 exposed to noise; 5825 working in low noise area; 67.5% of employees included; men and women.	Multiple source shipyard noise of >85 dB Lqm for longer than 5 h a day for exposed workers and <75 dB Lqm for non-exposed workers. NPL method of measurement used; plant divided into 3 parts with each part measured separately using 9 day time and 12 night time points.	Hypertension; no diagnostic criteria, no definitions; from periodic health exams. Records of 13 doctors in the industrial clinic. Also studied were hearing damage, stomach ulcers, and psychic problems.

Table 3-50: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Potential non-respondent and selection bias; no evidence of controlling for age, sex, treatment, co-morbidities or length of employment with hypertension data.	Prevalence ratio for hypertension of 2 noted. No statistical tests given.	"Significantly higher numbers of differences in the state of health detected in workers during periodic examinations, such as hearing damage, ulceration of the stomach and duodenum, and psychic disturbances and neurosis in the group of workers who were in the zone of high noise level during work, were confirmed" ... "Smaller differences, but also statistically significant occur in the frequency of hypertension found in both groups (more than double in group A)."	Strength of the study was in its specification of noise exposure.

Table 3-51: Pokrovskii (1966).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Pokrovskii, N. N.; 1966.	Cross-sectional. Industry (Machine Building); Russia.	995 fitters & lathe operators, ages 17-55 years.	Group 1: 408 exposed to medium frequency stable noise at 80-85 dB; Group 2: 587 workers exposed to high frequency noise at 90-95 dB.	Blood pressure taken during first 2-3 h of AM shift, after 20 min rest, in plant clinics; classified by Rosin as hypotension, diastolic hypotension, systolic hypertension, decapitated hypertension and hypertonic condition. EKG readings on 71 workers exposed to 80-85 dB noise and 113 workers exposed at 90-95 dB.

Table 3-51: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias, non-response and measurement bias. Age partially controlled; no evidence of control for family history, kidney disease, obesity, exercise, medications, etc.	Prevalence Ratios: DBP 3.87 times higher among persons exposed to 90-95 dB than for those in 80-85 dB noise. Other ratios by age, hypertension, and SBP given. Fisher test employed.	"Under the influence of intense industrial noise, the arterial pressure of workers can change in relation to individual characteristics in both directions: toward its increase, and toward its decrease as well. In persons exposed to the systematic effect of intense noise, blood pressure is characterized by more pronounced oscillations."	Methodologically weak study.

Table 3-52: Proniewska (1972).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Proniewska, W.; 1972.	Cross-sectional. Industry; Poland.	121 females in noise areas; 9 controls in quiet areas.	Intensity: 110 dB, frequency ranged from 31.5 Hz to 16,000 Hz; temperature 24 C; relative humidity 78%; work experience from 1-10 years.	Total serum lipids; beta lipoprotein, total cholesterol, FFA.

Table 3-52: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects on optimal diet; ages ranged from 20-40 years; cross-sectional data treated as serial measurements.	Not stated. Student's paired test used for the serial data.	In the first 2 h interval the cholesterol, beta-lipoprotein and FFA were raised with a decrease in total lipids. In second 6 h interval, only an FFA increase was observed.	Study has no baseline measurement to support conclusions of change in lipid levels with change in duration of exposure to noise.

Table 3-53: Rai et al. (1981).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Rai, R. M.; Singh, A. P.; Upadhyay, T. N.; Patil, S. K. B.; Nayar, H. S.; 1981.	Cross-sectional. Industry (Machine noise); India.	75 healthy males in noisy shop, mean age 33 years. 35 men not exposed, mean age 31 years.	Noise measured with B & K Precision Sound Level Meter Type 2204 with levels ranging from 88-107 dBA and impulse sound levels of 95-99 dBA.	Total cholesterol, cholesterol free and ester and cholesterol free ester; uric acid, serum protein fractions; cortisol ug/100 mg; albumin/globulin ratio. Methods described for each.

Table 3-53: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects similar on age, service period, activity level, smoking, nutritional status, oil fumes and ventilation. Noise level of non-exposed not given. Unclear as to how the exposed and non-exposed groups were selected. No controlling of variables after determining groups comparable.	Mean values and Z statistics applied to determine significance of differences noted.	"...free cholesterol was significantly higher ($p < .001$) in the experimental group resulting in significant difference ($p < .001$) in the ratio of free to esterified cholesterol. Total cholesterol was slightly higher in the experimental group." Serum cortisol and γ -globulin protein levels were significantly higher ($p < .01$) in the noise exposed group.	May be useful in suggesting mechanism for noise effects since both continuous and impulse noise was included.

Table 3-54: Raytheon Report (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Raytheon Report. 1975.	Before/after comparison (Hearing Conservation Program). Industry 4 Plants: Boiler; Nuclear Comp.; Tube mill; Superheater. U.S.	434 workers in high noise group; 432 workers in low noise group; 44 workers lost to follow-up.	Noise in production of large pressure boilers; noise levels given for 75 jobs; Intensity: high noise = >95 dBA; low noise = <80 dBA; exposures variable; no ear protection in 1969-70, all workers in high noise area with ear protection in 1972-73. Ear protector usage based on self-report and 1 observation by project director.	Diagnosed medical problems and symptoms from extant records; coders "blind" to exposure conditions; diagnostic criteria not given.

Table 3-54: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Worker age, job type, length of service, work shift considered in analysis. Previous medical history and use of outside medical services not explored.	Not quantifiable as prevalence ratios. Wilcoxin Matched-Pairs Signed Rank Test and Median Test employed.	"Expected relationships between rated usage of ear protectors and incidence of extra-auditory problems were orderly in some cases, but not in others. Methodological shortcomings in determining ear protector usage may have been responsible for this result."	Methodological shortcomings and small sample size make interpretations of the data presented difficult.

Table 3-55: Rumiantsev et al. (1971).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Rumiantsev, G. T.; Mekhelson, D. A.; Sechenov, I. M.; 1971.	Cross-sectional. Industry (ships); Russia.	85 sailors; 25-35 yrs. of age with >3 yrs. employment, 8 ships.	Steady noise of high speed engines. Group 1: (engine squad): low & medium frequencies; Group 2: low & medium frequencies and diet supplement; Group 3: crewmen (controls).	Blood cholesterol, blood sugar, B-lipoprotein level; gamma-globulin level; blood pressure and pulse.

Table 3-55: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Self-selection; bias due to noncomparable comparison groups; no evidence of controlling for age, diet, exercise, diabetes, weight, etc.; non-response or missing data bias.	Strength of association and statistical tests not given. P-values stated.	"Thus, significant increase of the sugar concentration in the blood on the 15th day of the course and of B-lipoproteins in the blood serum on the 30th day of the course in sailors of the 1st group, with no analogous group, can probably be treated as the consequence of noise-vibration effect. The changes of the arterial pressure and pulse before and after the watch in the sailors of the 1st group can be explained in the same manner."	Study not informative for evaluating the association between noise exposure and CVD.

Table 3-56: Sanden and Axelsson (1981).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Sanden, A. J.; Axelsson, A.; 1981.	Cross-sectional (quasi-experimental). Industry (shipyard workers); Sweden.	40 males, 40-50 years of age, with at least 10 years noise exposure; 20 noise sensitive (NS) and 20 noise resistant (NR).	Noise measurements not given. Hearing loss used as surrogate: Noise sensitive: mean hearing level at 3000-8000 Hz of >40 dB HL; Noise resistant: mean hearing level at 3000-8000 Hz of <30 dB HL. Pure tone audiogram used.	Medical and physical exams with hemoglobin level, sedimentation rate, serum triglycerides & serum cholesterol, resting SBP and DBP, heart rate and electrocardiogram; heart rate and BP measured while on ergometer cycle of 18 min without any noise, with sound at 62 dBA and at 95 dBA shipyard noise.

Table 3-56: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Highly select groups, but noise sensitive and noise resistant groups similar on lifetime noise exposure, physical fitness, diet, alcohol consumption, smoking habits, hereditary hearing disorders, diseases and medications.	Mean BP for the two groups: NR: 136.2 mm Hg SBP 84.5 mm Hg DBP NS: 131.3 mm Hg SBP 83.4 mm Hg DBP Fisher's permutation test and Pitman's nonparametric permutation tests used.	"In spite of the similarity between the two groups in resting cardiovascular function, physical fitness and smoking habits, the NS-group showed a statistically significant more rapid heart rate increase during work load than the NR group ($p = 0.997$). This difference was found both with and without noise during the work load." ... "Within each group there was no statistically significant difference in systolic and diastolic blood pressure during the work load test dependent upon the presence or absence of noise." "...a tendency was noted for NS individuals to have slightly higher mean serum cholesterol values."	Sample size is very small. The suggestion by the authors that individuals most likely to develop noise induced hearing loss may be those with high mean cholesterol levels who experience high noise is worth noting.

Table 3-57: Sanova (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Sanova, A. G.; 1975.	Cross-sectional. Industry (Compressor shops); Russia.	144 male compressor operators, 20-50 yrs. of age; 30 controls from the same plant working under low noise.	Actual noise level not reported. In compressor shops 87-98 dBA and 90-110 dB infrasound levels. Duration of exposure in years employed ranged from <1 to >20 years.	Arterial blood pressure, EKG, contractile and minute blood volume, peripheral resistance. Definitions and measurement procedures not stated.

Table 3-57: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Small sample size bias; selection and measurement bias; groups stratified by age; no other confounding variables considered.	Associations observed not stated quantitatively. No statistical methods given.	"The analysis of the indexes of the functional state of the cardiovascular system indicated that the percentage of workers with normal blood pressure decreases the longer the length of employment in compressor shops." ... "In the basic group (noise), we observed the increase of systolic pressure to be more pronounced in relation to age (than in workers not exposed to noise)." ... "The reduction of the contractile function of the myocardium, increasing with the length of employment in the compressor shop, can be related to the disorder of the functional state of the myocardium under exposure to noise."	Study of limited usefulness due to inadequate controls and analysis.

Table 3-58: Semczuk and Goms (1971).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Semczuk, B.; Goms, H.; 1971.	Mixed cross-sectional experimental. Acoustic darkroom and worksites. Poland.	200 healthy males; (50 in test group; 50 in controls; 100 in worksite).	Intensity: 1: audiometer = 100-110 dB for 40 s intervals. 2: industry = 80-115 dB "on the average" for 8 h shifts.	CRIS - index of cardiorespiratory efficiency.

Table 3-58: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
50 controls and 100 test subjects worked in similar temperatures and humidity. No controlling for age, length of exposure, etc.	Was not stated in report text.	"In 73% of examined individuals after 8 h of work in noise of 90-115 dB intensity, the CRIS value decreased while in a control group of 50 workers in less noise, 65 dB, such changes were not observed."	The clinical significance of the health outcome -- CRIS index -- is very questionable especially for population based studies.

Table 3-59: Shatalov et al. (1962).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Shatalov, N. N.; Saitanov, A. O.; Glotova, K. V.; 1962.	Cross-sectional; before and after work. Industry (Ball bearing and twisting shop). Russia.	156 workers in twisting plant; 144 workers in ball-bearing plant.	Twisting shop: 85-95 dB; ball bearing shop: 114-120 dB; mixed medium and high frequencies in both shops. Duration of exposure -- 143 persons had worked 10 yrs. or more.	Blood pressure by Korotkov method; high BP >130/90 mm Hg for persons <40 yrs. of age and 140/90 for persons >40 yrs.; ECG, oscillography, ballistocardiography. No data on measurement procedures or conditions of testing.

Table 3-59: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; no evidence of controlling for any major potential confounders in the analysis. Age may have been partially controlled, sex not considered.	Was not stated in report text.	"Very often the workers examined had labile arterial pressure. According to the electrocardiographic data, bradycardia with tendency toward retardation of the intraventricular conductivity and the falling off of the T-wave were observed, which were more common after physical stress and at the end of the work day. In the group of workers exposed to the effect of noise of greater intensity, functional changes in the cardiovascular system were more common and more pronounced." The hypertensive effect of noise was not observed.	No low noise level control group severely limits the value of this study. The bradycardia, T-wave changes are non-specific and of questionable significance.

Table 3-60: Shatalov (1965).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Shatalov, N. N.; 1965.	Cross-sectional. Industry (Type not stated); Russia.	1019 exposed to continuous noise and 338 to intermittent noise; also for special studies: Group 1: 368 in continuous noise; Group 2: 221 in intermittent noise.	No information provided on noise exposure for group of 1357 studied for BP; For Group 1: 85-122 dB, wide band spectrum with high frequency domination; continuous noise. For Group 2: 85-111 dB, wide band spectrum with high frequency domination, intermittent noise.	Blood pressure taken at beginning of work day after 10 min rest. High blood pressure defined as > 130/90 mm Hg for persons < 40 yrs. old and > 140/90 mm Hg for those > 40 yrs. EKG, ballistocardiography, phonocardiography, peripheral resistance, venous pressure, heart size. No definitions or criteria given.

Table 3-60: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and measurement bias. No evidence of control of any confounders such as age and sex. Individuals with history of heart disease excluded from study.	Was not stated in report text.	"Very often the workers in 'noisy' occupations have complaints of a cardiac character; their arterial pressure is changed. In persons exposed to continuous noise, first of all the symptoms of vascular dysfunction are observed (lability of the arterial pressure, tendency toward the reduction of venous pressure and the reduction of peripheral resistance, bradycardia). Under exposure to intermittent noise in the course of a work day a clear tendency to hypertension is present..."	Study contributes little to understanding noise effects. Author's conclusions are not supported with data.

Table 3-61: Shatalov et al. (1969).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Shatalov, N.N.; Ostapkovich, V.E.; Panomareva, N.I.; 1969.	Cross-sectional. Industry (Ball-bearing plant); Russia.	806 persons exposed to noise; 210 men and women of respective ages with normal hearing, not exposed to noise.	General noise level 90-122 dB, high frequency.	Blood pressure readings taken in AM before work. No definitions or criteria given.

Table 3-61: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection; non-response; measurement bias. Stratified by age of <40 yrs. and >40 yrs. No adjustment for obesity, job stress, co-morbidities.	Strength of association not stated. Means, standard deviations and t-values given.	"The comparison of the state of hearing and arterial pressure in persons working in conditions of the effect of intense industrial noise showed that the changes of the arterial pressure precede hearing damage. Therefore, one cannot exclude the role of vascular disorders in the development of occupational hearing impairment. In those cases when hearing is already lowered there is no further progression of the processes of sharp dependence on the state of the arterial pressure."	Poor study with inadequate information on exposed and control groups, too few control subjects, little information on sound and BP measures and incomplete control of confounding.

Table 3-62: Shatalov and Murov (1970).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Shatalov, N.N.; Murov, M.A.; 1970.	Cross-sectional. Industry (Ball-bearing plant); Russia.	2034 men, 1896 women, 20-59 yrs. of age, grouped by noise exposure: 1: 1275 fitters, loaders, lathe operators; 2: 339 operator testers of cranes; 3: 1172 scientists; 4: 1144 lab technicians and skilled mechanics.	Group 1: broad band noise at 95-112 dB with high frequencies dominating; Group 2: noise analogous to Group 1 plus tension; Group 3: no noise (implied); Group 4: control; work not connected with noise. No additional information provided.	Systolic and diastolic blood pressure; hypertensive disease; no definitions or criteria stated.

Table 3-62: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias, measurement bias, age and sex controlled by stratification and standardization procedures. No controlling evident for other potential confounders such as obesity, exercise, medications taken. Hearing thresholds not obtained. Duration of employment unknown.	Calculated from table: Age-adjusted (male) prevalence ratio of hypertension for noise exposed plus tension group compared to control group 3.4. Multiple t-tests and means used to describe data.	"The studies conducted show that the exposure to industrial noise causes an increase of systolic pressure (in the age groups below 40 years) and a rise of incidence of hypertensive disease, compared to the control."	Study poor in design with incomplete analysis. Major problem is that prevalence data is treated as incidence data.

Table 3-63: Stasiow et al. (1974).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Stasiow, A.; Guzikowska, H.; Guzikowski, J.; 1974.	Cross-sectional data; pre/post shift measures. Industry (Mine); Poland.	31 workers in the washers, a mechanical coal processing section of mine, ages 35-62 years; employed >6 years. No controls.	Noise levels ranged from 86-102 dB; 7.5 h exposure daily; noise measurements in the work place, in a position near the head of the worker.	Arterial BP, in sitting position, with mercury sphygmomanometer, after a night's rest, before work, and repeated after 7.5 h of noise exposure; cold pressor test; EKG at rest with 12 standard leads and evaluated by Minnesota Code.

Table 3-63: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Self-selection; interaction of selection and exposure to noise; regression to the mean.	Degree of association not stated. Sign test employed.	"In the examined group of workers, under the influence of exposure to industrial noise, a significant increase in the diastolic pressure and a spastic state of the arterial vessels on the periphery were confirmed. It was shown that exposure to industrial noise causes an increased vessel reactivity. The observed changes in the EKG curve can speak for the domination of the parasympathetic system."	Study provides insufficient information for inferring an effect of noise on cardiovascular responses.

Table 3-64: Suvorov et al. (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Suvorov, G. A.; Denisov, E. I.; Ovakinov, V. C.; Tavtin, IU. K.; 1979.	Cross-sectional. Industry (Machine building); Russia.	740 workers exposed to high noise divided into 4 groups and 121 workers exposed to low noise of about 70 dBA. 587 males and 274 females; ages 34-38 years.	Group 1: 121 workers at 70 dBA noise level; Group 2: 139 at 84 dBA; Group 3: 168 at 93 dBA; Group 4: 267 at 100 dBA; Group 5: 166 at 115 dBA; average length of employment 11-16 years.	Hypertension defined as BP > 140/90 mm Hg; hypertensive disease as BP 159/94 mm Hg; hypotension as BP < 100/60 mm Hg; neurocirculatory asthenia by medical specialist Dx.

Table 3-64: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias and non-response bias; incomplete control of age. No controlling for sex, weight, history of disease.	Increase in neurovascular impairment of 0.5% with each 1 dBA increase in noise level. Regression analysis used.	"...At low noise levels, neurovascular disorders prevail, while hearing losses prevail at higher levels. Qualitative and quantitative dose-response relationships were established in respect to both the hearing function and mediated responses: the rate of increase in hearing and neurovascular impairments depended on the level of noise, and was 1.5% and 0.5% respectively, per 1 dBA of increase in the noise level; the increased noise level accelerates the symptoms of non-specific mediated impairments (on the average by 13 years with the noise level increased by 31 dBA), most of which belong to hypertonic states."	A fairly well designed cross-sectional study which would be more informative if confounding variables had been adequately controlled.

Table 3-65: Talbott et al. (1985).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Talbott, E.; Helmkamp, J.; Matthews, K.; Kuller, L.; Cottingham, E.; Redmond, G.; 1985.	Cross-sectional. 2 Industrial plants; noisy: fabrication & assembly of large metal parts; less noisy: manufacture of heating elements. U.S.A.	197 in noisy plant; 167 in less noisy plant; white males, 40-63 yrs. of age; had worked at least 10 years at either plant.	Current assessment of noise with Gen Rad 1565A Sound Meter and metrologger dB-301 dosimeter in noisy plant - average noise level 89 dBA; 39.6% men wore hearing protection almost always since 1978. No formal noise survey in less noisy plant -- but current assessment with metrologger dB-301 dosimeter gave average ambient noise level < 81 dBA. Continuous employment: Noisy plant: 28 yrs. Less noisy plant: 32.8 yrs. Pure tone hearing thresholds by certified audiologist in testing booths meeting Am. Nat. Standards criteria. Questionnaire on military, occupational history and noisy hobbies.	1st and 5th Korotkoff sounds for SBP and DBP respectively, taken on right arm in sitting position with standard mercury sphygmomanometer and random zero device. BP determined by a nurse 3 times within 5 min after a 10 min rest period prior to working day and repeated by second nurse 15 min later. Defined as overall mean of 4 zero muddler measurements. No significant intra- or inter-observer differences for the 2 nurses. Hearing impairment: greater than a 25 dB average threshold at 500, 1 k and 2 kHz in better ear. Noise induced hearing loss 65 dB or greater threshold at 3, 4, or 6 kHz.

Table 3-65: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Alcohol use, smoking, family history, body mass index, age controlled in analysis; Possible misclassification of noise exposure since current levels used; sample size smaller than that calculated (250/group) to detect a 5 mm Hg difference in mean BP; potential temporal bias since only cross-sectional data; only 66% response rate in noisy and 68% response rate in less noisy plant.	State of the art methods--analysis of variance, multiple regression and logistic modeling techniques were used to control for known risk factors. No significant associations found between noise and BP in total group. Prevalence ratio of hypertension of about 2.0 for those with noise induced hearing loss vs. those with no hearing loss among older workers.	No significant differences between workers in the two plants in either SBP or DBP. In the noisier plant, men who wore hearing protection all or most of the time had mean BP levels equal to those who indicated little or no use (128.2 mm Hg SBP and 81.1 DBP vs. 129.2 mm Hg SBP and 81.3 mm Hg DBP). The prevalence of severe noise induced hearing loss was significantly ($p < .01$) greater in men in the noisy plant (65%) compared with those in the less noisy plant (47%). For men over 56 yrs. of age, prevalence of hypertension was about 2 times as great in those with severe hearing loss as those without such hearing loss ($p < .05$). In both plants, prevalence of hypertension did not increase substantially in those without noise-induced hearing loss. After adjusting for risk factors, there was a strong relationship ($p < .02$) between noise induced hearing loss and high BP in the 56+ age group in both plants. In the noisier plant, body mass index, noisy hobbies and severe noise-induced hearing loss explained 18.6% of the DBP variability ($p < .05$).	Useful study since it applied state of the art statistical methods to data although it can be criticized on a few problems inherent in cross-sectional data. First study in U.S. in which noise induced hearing loss was found to be a significant predictor of DBP and hypertension when controlling for risk factors for high BP.

Table 3-66: Tavtin (1976).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Tavtin, IU. K.; 1976.	Cross-sectional. Industry (Machine building plant); Russia.	861 workers divided into 5 noise groups; 281 women to 580 men; employed <5 yrs. to >20 years.	5 noise exposure groups. Group 1: 121 workers, in 70 dB noise; Group 2: 139 workers, in 83 dB noise; Group 3: 168 workers, in 94 dB noise; Group 4: 267 workers, in 110 dB noise; Group 5: 166 workers, in 114 dB noise.	Clinical examinations with diagnoses of functional disorders of the cardiovascular and nervous system, hypertension and arteriosclerotic heart disease.

Table 3-66: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection and selective survival; impure noise exposure groups; no evidence of control for any variables including age and sex.	No high to low noise group comparison.	"Under exposure to continuous noise of 110 dB ...significant increase of the functional disorders of the nervous (12.5%) and cardiovascular (6.3%) systems was observed. In the exposure to a pulsed noise level of 110 dB ...functional disorders of the nervous system in 16.2%, functional disorders of the cardiovascular system in 8.4% of the cases."	Data in publication is inadequate to support author's conclusions. Apparently high noise group responses were not compared to low noise group responses.

Table 3-67: Terentiev et al. (1969).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Terentiev, B. G.; Sheludiakov, E. E.; Sviridova, E. S.; 1969.	Cross-sectional with no controls; before/after work measures; anecdotal; experimental. Industry and laboratory (aviation); Russia.	90 engineering and technical staff; 15 healthy men.	Separate conditions of 100-102, 110-112, 118-120, and 130-136 dB; subjects exposed either daily or 2-3 times a week for 1-6 h each time. Laboratory: exposure at 1 h, 3 h and 6 h.	Blood pressure; pulse rate; EKG changes. No diagnostic criteria or definitions given.

Table 3-67: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection; impure exposure group; no control group; no evidence of analysis of data in a before/after mode.	Was not stated in report text.	A one-hour per day exposure to noise in the 110-112 dB range can be successfully endured, but that daily exposures of 3 and 6 h will bring about harmful effects. ...During daily exposure of 1, 3, 6 h of noise at 120 dB level, an increase in systolic and diastolic blood pressures was observed.	These anecdotal, cross-sectional and experimental findings are of little value to the study of noise effects because of poor study design.

Table 3-68: Theriault et al. (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Theriault, G.P.; Tremblay, C.G.; Armstrong, B.G.; 1988	Matched case control; aluminum smelter, Canada	306 cases: 150 angina and 156 myocardial infarctions. 575 referents matched on birth date, hiring date, within 5 yrs. on length of service, alive at date case was diagnosed.	Detailed occupational histories used by industrial hygienist to classify each exposure benzene, fluoride, total dust, sulfur dioxide, carbon monoxide, thermal stress, noise, physical load, mental load. No specific exposure levels stated.	Cases confirmed by written opinion of cardiologist, EKG and/or angiographic evidence. Absence of disease in researchers reviewing medical records. 3 professional nosologists extracted medical files for wt., ht., BP, biomedical tests, EKG readings, personal medical history, smoking status, lung function tests, as blindly as possible. Self-admin questionnaire sent to each subject or his family for other data such as family history of IHD.

Table 3-68: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Multiple factors considered and hypertension, hyperglycemia, hypercholesterolemia, obesity and smoking controlled in analysis due to confounding identified. Selection bias possible with vulnerable workers leaving the contaminated area soon after employment. Relevant exposure measures may not have been made.	For noise, odds ratio for IHD was 0.93(0.56=1.54), using logistic regression analysis.	Results showed that white collar workers had a significantly lower risk of IHD (OR=0.47, C.I. 0.31-0.70). Among blue collar workers, a significant higher risk (1.72) was observed for workers in reduction division. "The search for associations (among blue collar workers) of risk with nine specific contaminants (...noise..) proved inconclusive, with no association reaching statistical significance."	Inaccuracies in measurement of specific exposures and fairly high levels of exposure among cases and controls may account for findings. Sample size small relative to number of variables controlled.

Table 3-69: Troianskii et al. (1971).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Troianskii, M. P.; Sidortsov, I. P.; Petrova-Golubenko, L. B.; 1971.	Before/during/after work comparisons on a cross-sectional group of workers. Industry (Diesel and blower specialists); Russia.	55 workers; Group 1: 16 working 12 h with 24 breaks; Group 2: 24 working with 12 h breaks; Group 3: 15 working under same conditions without noise (control group).	Noise range of 94-97 dBA in medium and high frequencies.	Blood serum cholinesterase by Hestrin's method; arterial blood pressure; no additional information provided.

Table 3-69: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection, no controlling for any variables such as age, sex, length of employment.	No data shown for complete comparison with control group. Means, standard deviations, and p-values stated, no specific tests noted.	"In specialists working under exposure to the effect of acoustic noise, during examinations immediately at the place of work, statistically significant lowering of the activeness of cholinesterase in blood, extension of the latent period of the dermatographic reaction, and a clearer retardation of the pulse during Oschner's test, with a maximum definiteness at the end of the shift, were observed. After the end of the effects of noise, the activeness of cholinesterase was restored. With 12 h shifts, full restoration occurred after 24 h.	Although study uses control group, no direct comparisons between exposed and controls are made. Small sample size, little data on exposure and health states limits its usefulness in inferring cause.

Table 3-70: Verbeek et al. (1987).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Verbeek, J.H.A.M.; Dijk, F.J.H. van; de Vries, F. D.; 1987.	Cross-sectional. Industry (Production factories); Netherlands.	428 male workers in 6 factories; 131 excluded due to ear pathology or exposure to explosions; 10% non-response due to sickness or holiday leave. Group 1: 238 exposed to noise in present work; Group 2: 59 previously exposed.	Intensity range of 78-98 dBA in work site measured by Bruel & Kjaer SL meter, Type 2209 for area sampling. Assumed levels in past similar to those measured. Surrogate: noise induced hearing loss defined as largest loss at 3150, 4000, or 6300 Hz after correction for presbycusis. Peekel D77 audiometer.	1 measure of BP in sitting position with calibrated aneroid sphygmomanometer, Manuell Type Primus II. Hypertension: >160 mm Hg SBP; >95 mm Hg DBP or both. No additional information given.

Table 3-70: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects apparently not compared by noise levels but by number of years exposed by employment history. Selection bias possible. Age and ear pathology were only factors controlled. Not clear as to how hearing loss was used as a measure of exposure.	Weak association ($p < .05$) if accept duration of employment as exposure. Prevalence of hypertension rose from 9% with 0-9 yrs. work to 28% at 20 yrs. Mean SBP rose from 128 at 0-9 yrs. to 132 at 10-19 yrs. work to 145 by 20+ yrs. work after age correction. DBP increased from 79 to 81 to 87 for the same time periods.	"After correction for age, the SBP was on average 16 mm and DBP on average 7 mm higher in those exposed at least 20 years than in workers exposed fewer than 10 years. After standardization for age, percentage of workers with hypertension rose from 9% in those with fewer than 10 years exposure to 28% in those exposed for over 20 years. A significant positive relation between degree of hearing loss and BP for all 297 workers disappeared after correction for age."	Study of questionable value since all workers were exposed to noise levels greater than 78 dBA and years of employment were used as surrogate for noise exposure. Methodology represents state of the art for cross-sectional occupational studies.

Table 3-71: Volpikina (1959).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Volpikina, G.I.; 1959.	Cross-sectional; before/after work shifts. Industry (Textiles); Russia.	300 spinners, 300 weavers. Number of unspecified controls.	No measurement provided; subjects were spinners, weavers and controls were workers in packing and mechanical plant.	Blood pressure. No information provided on measurement.

Table 3-71: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection; non-response; measurement bias. No evidence of control of any variables other than stratification by age.	Was not stated in report text.	It is clear from our documentation that an unfavorable microclimate and work-related stress do provoke characteristic disturbances in the reactions of the cardiovascular system and the higher nervous activity among the women working in the textile industry.	Study poorly designed and/or details missing. It is unclear what group comparisons were actually made.

Table 3-72: Wu et al. (1987a).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Wu, T.N.; Ko, Y.C.; Chang, P.Y., 1987	Cross-sectional and Case-reference. Shipyard, Taiwan.	158 male workers from area with noise <85 dBA vs 158 random matched controls from 2572 workers in the <80 dBA noise area.	Workplaces with sound levels >85 dBA vs those <80 dBA as measured by RION NL-10 sound level meter. Noise measured from 6/83-8/83 under "blinded" conditions.	BP taken with sphygmomanometer by nurses according to method of Jonsson and Hansson, in the morning before work and before physical exam. Hypertension defined according to WHO, 1978 as SBP 160 mm Hg or more or DBP 95 mm Hg or more.

Table 3-72: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects were matched on age (#±# 6 months); employment duration (+6 months) and body mass index (#±# 0.5 Kg/m ²). Excluded were workers with family history of hypertension, high BP-related disease, documented hypertension before employment, exposure to the 80-85 dBA noise area and females.	Student's paired T-Test, Fisher's Exact Test, McNemar Chi-square test used. RR=2.38 (p<.05).	"The result shows that the exposed group had a higher prevalence rate of hypertension than did the reference group. Moreover, the result from case-reference study indicates that close to a doubling of risk of hypertension occurred among workers who were exposed to higher noise levels."	It is unclear how prevalence rates were calculated from matched cross-sectional data. Although a limited number of potential confounders were considered, the use of only incident cases of hypertension in the matched case-reference study increases the validity of the findings.

Table 3-73: Wu et al. (1987b).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Wu, T.N.; Chou, F.S.; Chang, P.Y. 1987	Cross sectional, steel mill, Taiwan	151 workers with highest hearing threshold at 4000 Hz and no family history or treatment for hyperten- sion.	Not stated; employment duration 39-100 months. Testing with MAICO audiometer in chamber with ambient noise 30-40 dBA. Both ears tested at frequencies of 500,1000,2000,4000 Hz with air conducted pure tones. Hearing loss calculated by sexenary method $(500 + 1000 \times 2 + 2000 \times 2 + 4000)/6$. Standardized procedures used and tests conducted prior to work day.	BP taken with mercury sphy- gmomanometer in afternoon by MD according to method of Jonsson and Hansson.

Table 3-73: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Excluded subjects with confounding conditions; used analyses of covariances to adjust for age, employment duration, and body mass index. Potential selection bias related to hearing loss and/or elevated BP.	Multiple regression and analyses of covariance. Statistical parameters with p-values given. No associations between hearing loss and BP noted.	"Results from analyses of covariances indicated that noise-induced hearing loss is unrelated to systolic and diastolic blood pressures after adjusting for these confounding factors"....It seems that hearing loss is not appropriate as a noise exposure index to measure the relationship between noise exposure and blood pressure.	Although cross-sectional in design and sample selection criteria unclear, several major confounding variables were controlled. Study is of interest since it explores the use of noise-induced hearing loss as a surrogate for noise exposure. Sexenary method for calculating hearing loss may not be appropriate in US populations.

Table 3-74: Zvereva et al. (1975a).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Zvereva, G. S.; Onopko, B. N.; Ratner, M. V.; 1975a.	Cross-sectional; (before/after work shifts). No controls. Industry (Dolomite-Flux plants); Russia.	334 workers in 8 professional groups, aged 30-40 years.	Quarries, excavation and drilling places: 95-100 dB, low and medium frequencies. Crushing-enriching plant; 86-106 dB, predominate high frequency components. Noise measured in 164 work places.	Arterial pressure; state of the capillaries; complaints such as heart pain and fatigue; asymmetry of arterial pressure, arisocoria and trembling of the hands.

Table 3-74: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; bias due to lack of controls and non-comparable subjects within the study groups.	Was not stated in report text.	"The data obtained show that noise and vibration in the plants of the flux-dolomite industry have quite high levels and cause changes in hearing and disorders in the function of nervous and vascular systems typical of vibration-noise pathology."	Design is inappropriate for inferring cause.

Table 3-75: Zvereva et al. (1975b).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Zvereva, G. S.; Ratner, M. V.; Kolganov, A. V.; 1975b.	Cross-sectional Industry (Pipe & sheet rolling); Russia.	340 workers; no age given; not categorized by exposure levels.	4 areas of the plant described; 1 = 122 dB; 2 = no data; 3 = 115 dB; 4 = up to 142 dB; no use of dosimeters; duration of service given as < 1 yr.; 3-5 yrs.; 6-9 yrs.; and 10 yrs. or more.	Arterial pressure; pulse rate; complaints. No quantitative data provided; no diagnostic criteria or measurement information.

Table 3-75: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection, non-response bias; no obvious control group.	Was not stated in report text.	"One-hundred sixty-eight persons (50%)...were found to have hearing impairment. ...Complaints in many cases were combined with objective symptoms: disorders of arterial pressure (more often elevated pressure), pulse lability, decrease of vibration and pain sensitivity (up to full anesthesia), thermal asymmetry, trembling of arms and eyelids, instability in the Romberg position."	Design described too poorly and failure to report BP data invalidates this study for the purpose of judging associations between noise and CVD.

4. Summary of Laboratory Studies of the Effect of Noise on the Cardiovascular System

Table 4-1: Burger and Klimes (1975).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Burger, F.; Klimes, J.; 1975.	Quasi-experimental; Simulated noise of tank motors. Laboratory; Czechoslovakia.	20 untrained conscripts, aged 19.3 $\pm$.09 yrs.; 10 exposed to 20 min noise while exercising; 10 exposed to 2 h of noise with exercise.	Steady noise of tank motors with a known spectrum recorded and conveyed to room by 2 loudspeakers; short-term noise at 90, 100, 110 dB; long-term noise at 80, 100 dB.	Pulse frequency; EKG curve evaluated with a 1-channel electrocardiograph.

Table 4-1: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; Latin square design used to avoid habituation; no other evidence of controlling for confounding.	Associations not stated quantitatively. Test of pairs used.	"Even though our experiments allow the conclusion that a decrease of physical ability does not occur, it is, in view of its effects upon the circulatory system, impossible to affirm unconditionally that exposure to noise is completely harmless to the organism...but that its negative effects will become manifest after an exposure lasting a number of years. The use of protectors against noise does not prevent its effects upon the circulatory system."	Study is weak in that it did not use sufficient subjects to allow random assignment of groups to treatments as a better means of control. Data insufficient to warrant inference about long term effects.

Table 4-2: Carter (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Carter, N.L. 1988	Cross-sectional. and quasi-experimental pilot project. Australian artillery range	6 males, aged 17-24 years, volunteers from Artillery unit.	Impulse noise from firing of two 105 mm howitzers placed 7 m apart. M1 ammunition used 1st day and M2(louder) ammunition used 2nd day. Total 40 rounds per gun per day. All subjects wore EAR ear-plugs. Specific noise levels not stated, but suggest that peak overpressures may be up to 181 dB.	Heart rate and arrhythmia obtained from medilog 400(Holter) monitor with medilog 4000 replay system(MP-40). Monitors synchronized with time generator of video-recording system of gun firing. BP measured within 10 min before and as soon as practicable (about 1 min) after firing with space labs ambulatory BP monitor (model 5200) and random access memory pack (model 5250).

Table 4-2: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
All subjects had normal spirometric and chest x-rays, no history of respiratory problems, no illness. Hearing levels no >25 dB in either ear up to and including 8000 Hz. Potential selection bias from volunteers. BP measured at other times to estimate typical BP and before/after simulated firings to estimate effect of physical activity without noise.	Means of BP and BP differences graphed for individual subjects. No statistical testing of data.	"The results suggest that an increase in heart rate of 10-30% occurs which is attributable to the noise and which does not habituate to successive firings, and that an acute rise in blood pressure may have been associated with the firing of the higher-powered charge." The SBP's before and after firing of the higher-powered charge showed a mean difference of 14.5 mm Hg compared with -2.9 mm Hg for SBPs not associated with gunfire.	Although a pilot project of only 6 men, experiencing extremely high noise from 2 guns placed close together and fired simultaneously, this is one of the few studies of intermittent noise under realistic conditions. Although noise exposure may be too high to be relevant for aircraft studies, only the higher powered charges and not the M1 firings showed changes in BP.

Table 4-3: Cesana et al. (1982).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Cesana, G. C.; Ferrario, M.; Curti, R.; Zanettini, R.; et al. I and II; 1982a and b.	Convenient group of volunteers compared but called experimental by authors; 10 days duration. Newspaper Industry; Egypt.	15 Pressmen exposed to noise and on rotating shifts; 15 maintenance workers on same rotating shift not exposed to noise; 15 clerical workers not exposed to noise nor shift work.	Noise levels described elsewhere but not available for this review.	Catecholamines, epinephrine and norepinephrine urine samples collected at specified times and stored and analyzed with HPL chromatographic method; Dopamine urinary excretions; Methods described.

Table 4-3: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subject selection not random from 3 groups; subjects asked to follow uniform diet, eat no food with amines, limit use of coffee, alcohol and tobacco for 20 days; no other controlling.	Not stated in reported text. No statistical tests given.	Norepinephrine excretion in those on shift work and exposed to noise was greater than those on shift work only and in turn greater than that of individuals not on shift work and not exposed to noise. Wide dopamine distribution and its renal production makes urinary excretion a reliable indicator of sympathetic activity.	Frequently referenced but appears to be of little use due to lack of control of multiple variables.

Table 4-4: di Cantogno et al. (1976).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
di Cantogno, L. V.; Dallerba, R.; Teagno, P. S.; Cocola, L.; 1976.	Quasi-experimental. Laboratory "silent" booth; Italy.	33 males exposed to noise: 11 normal; 11 diabetics (male); 11 coronaropathic; 11 normal males were controls.	Short-term: 10 one minute recordings of traffic noise. Intensity: LndB = 88.8 LeqdB = 89.4 LndB(A) = 71.6 LeqdB(A) = 73.1 Administered via headphones; Uher 4400 Stero Report 1C recorder fed noise through amplaid 500 audio meter; amplifon G5 Silent booth erected in a room lined with soundproof panels.	BP automatically taken every minute with Erka Diasist Apparatus; ECG and polygraphic data collected using standard techniques.

Table 4-4: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Control group younger than comparisons; Mean ages: Controls = 27 yrs. Normal subjects = 36.7 yrs. Coronaropathic = 52.0 yrs. Diabetics = 46.4 yrs.	Not stated in reported text.	Uric acid increased with noise exposure, especially in dysmetabolics; blood cholesterol values increased; product of cardiac frequency and systolic arterial pressure tended to fall during acoustic stimulation in normal subjects, but stayed high in dysmetabolic group.	Addresses short-term noise exposure only.

Table 4-5: Ising (1981).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Ising, H.; 1981.	Laboratory; animal and human studies. Germany.	For study of humans, about 45.	Tape recorded traffic noise; no details given; 95 dBA high noise vs. 82 dBA low noise.	E magnesium and S magnesium levels; no measurement data.

Table 4-5: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Unable to evaluate; description of methods not reported.	Statistical methods not stated.	"that prolonged stress together with a marginal dietary intake will lead to an intracellular Mg decrease and parallel to that to an increase of Ca."	May be useful in suggesting a potential mechanism by which noise affects the cardiovascular system.

Table 4-6: Ising et al. (1980).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Ising, H.; Dienel, D.; Gunther, T.; Markert, B.; 1980.	Laboratory experiment with traffic noise. Germany.	57 volunteer males, 18-34 years of age.	Simulated traffic noise at $Leq = 85$ dBA played through loud speakers into room. Subjects worked with noise 1 day and 1 day without noise alternatively. Low noise day: $Leq < 50$ dBA.	Blood pressure: 5 measures averaged over 8 h period; Epinephrine, cAMP, urine and serum magnesium, protein, cholesterol, Na, renin, erythrocytes. Blood and urine test methods not described. Questions about psychic state.

Table 4-6: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
7 volunteers not used; selection criteria not given; subjects served as own controls but health state unknown.	Wilcoxon test used. Correlation coefficients and change in mean values given.	Statistically significant reactions to noise were an increase of BP by 1 to 5 mm Hg and pulse frequency; increase of epinephrine, cAMP, urine and serum Mg, protein and cholesterol, decrease of erythrocyte Na and renin. "...The catecholamines released under stress are apparently capable of increasing the sMg level. The slightly decreased eMg level and the 15% increase of the Mg excretion in the urine show, that under noise the cellular Mg is decreased. Stress and a marginal Mg nutrition supply may cause a chronic negative Mg imbalance. This hypothetical process would lead to Mg decreases and Ca increases in the myocardium and thus increase the risk of a heart attack and the so-called sudden death ischemic heart disease."	Study of short-term effects of noise but suggest a potential mechanism by which noise may act as a stressor to impact the cardiovascular system.

Table 4-7: Klotzbuecher (1976).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Klotzbuecher, F.; 1976.	Quasi-experimental. Laboratory; Germany.	10 healthy male vocational students; 17-19 years of age.	3 test conditions randomly assigned to subjects: wide band (white) noise at: (1) 55 dB(AI); (2) 70 dB(AI); (3) 85 dB(AI); 3 test groupings: (1) rest, 10 min sitting; (2) 3 min rest with noise; (3) 90 min arithmetic tasks with noise; Tests conducted over a 12 day period.	Heart rate measured by EKG; respiratory rate by thermister; catecholamines determined by method of Euler and Floding.

Table 4-7: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Sequence of test series was random; subjects not randomly selected; no data as to comparability on work exposure, obesity, exercise, etc.	T-tests and correlations used. Associations not stated quantitatively.	"The number of significant correlations between performance and physiological reactions decreased a lot more from noise level 55 dB (AI) to noise level 70 dB (AI) than from noise level 70 dB (AI) to 85 dB (AI) "...The rise of adrenalin secretion under noise strain and mental work found by us is twice as high as 55 dB(AI) - as with noise strain." "...The recommended noise level at concentrated mental work should therefore be lower than 55 dB (AI)."	Physiological responses poorly defined and sample size small. Relationship between physiologic responses and overt cardiovascular disease in unclear.

Table 4-8: Mariniako and Lipovoi (1975).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Mariniako, A.; Lipovoi, V.; 1975.	Quasi-experimental. Laboratory; Russia.	20 healthy males; 20 observations in each of 4 conditions.	Series 1: 110 dB low frequency noise; steady. Series 2: 110 dB low frequency; intermittent. Series 3: 105 dB high frequency noise. Series 4: 105 dB high frequency; intermittent.	Tone of the vessels and pulse rate.

Table 4-8: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Selection bias; no randomization described.	Was not stated in reported text.	"A rather noticeable difference in the effects of stable and intermittent noise (especially with high frequency compositions) was noticed in the study of the tone of the vessels." ..."After one hour of effects of stable and intermittent noises, a tendency toward decline in pulse frequency, on the average of 2-3 per minute was observed."	Offers no new insight. Design as described is poor but findings consistent with other studies.

Table 4-9: Mosskov and Ettema (1977).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Mosskov, J. I.; Ettema, J. H.; (II) and (IV); 1977a and b.	Controlled Laboratory experiments with aircraft and traffic noise. The Netherlands.	12 healthy males (different subjects each noise source) 19-26 year olds; served as own controls.	Aircraft: Leq = 84-91 dBA; Traffic: Leq = 83.5 dBA; Total duration of noise 30-40 s; highest intensity for 6-10 s Taped noise presented through headphones in soundproof room. Three 15 min exposures with 2 rest periods of 5 min between.	BP, pulse rate, respiratory rate.

Table 4-9: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Random assignment of noise type not performed; random assignment of noise, mental load, combined load within each experiment.	Strength of association not stated. Wilcoxin test applied to data.	Exposure to aircraft and traffic noise caused an increase in DBP, and decrease of pulse pressure and heart rate/respiratory rate.	Findings of questionable value since difference in SBP, DBP, pulse pressure and sinoarrhythmias between the rest periods may mitigate some of the differences with exposure.

Table 4-10: Petiot et al. (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Petiot, J.C.; Parrot, J.; Lobreau, J.P.; Smolik, H.J., 1988.	Laboratory experiment. France	13 female subjects, 18-23 yrs. of age.	Each subject exposed to noise at 3 different sessions, at 1 week intervals, at same time of day. Each exposure was for 35 min to intermittent pink noise made of 3, 5 min noise steps at 105 dBA, alternating with 4, 5 min low level noise-steps at 40 dBA. Noise transmitted to subject through TDH-39 earphones mounted in MX-41 cushions; generated by feeding output of B&K 1405 generators into amplifier connected to earphones.	Heart rate in beats/min continuously recorded by a Telemetric Narco Biosystem, fed into a Physiograph MKIII and processed by a cardi tachometer. SBP and DBP measured in mm Hg. by auscultory method, using Vasquez-Lambry sphygmotensometer and spengler autotensometer. 14 BPs taken during 1st and last minute of each low or high level noise step.

Table 4-10: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
All subjects free of CV and ear pathology with normal hearing thresholds within +10 dB of audiometer zero point between 125 and 8000 Hz. 7 of 13 women took oral contraceptives which may be related to interaction between noise and hormonal factors. Subjects entered experiment in random order at different phases of ovarian cycle.	Ascendant clustering techniques and F-test analysis applied to data.	Application of a clustering algorithm to inter-correlation data on individual heart rates results in subsets of sensitive, moderately sensitive and poorly sensitive subjects. Sensitive always reacted progressively stronger to intermittent noise, moderately sensitive reacted moderately and poorly sensitive, poorly; heart rate did not habituate in any group with repeated exposures. Mean levels of SBP and DBP decreased sign. with repeated exposures in moderately and poorly sensitive subjects, but not in sensitive subjects.	This experiment is an attempt to develop a method to take into account individual differences in examining the effect of noise on the cardiovascular system.

Table 4-11: Quaas et al. (1970).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Quaas, M.; Geiller, W.; Platzbecker, L. U.; Zoellner, G.; 1970.	Quasi-experimental. Laboratory; Germany.	8 clinically and otologically healthy males; 22-35 yrs. of age.	3 Test Periods: Test 1: wore ear plugs; 10 min rest; 30 min continuous exercise; 10 min rest. Test 2: wore ear plugs; 10 min rest; 30 min continuous exercise under 90 dB wide band noise; Test 3: did not wear ear plugs; 10 min rest; 30 min continuous exercise under 75 dB wide band noise; 10 min rest.	Heart rate, oxygen consumption, carbon dioxide output. Instrumentation described.

Table 4-11: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects used as own controls -- very small sample. Study conducted under well controlled conditions; noise attenuation with ear protectors not documented.	T and F tests used but association not quantified.	"The use of ear protectors does not prevent influence of 90 dB wide band noise upon the heart-circulatory system." Heart rate increased under noise conditions.	Study is of interest since few have examined cardiovascular effects with noise attenuation with ear protectors and this experiment suggests health effects even with use of ear plugs.

Table 4-12: Von Eiff et al. (1982).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Von Eiff, A. W.; Friedrich, G.; Neus, H.; 1982.	Quasi-experimental. Laboratory: Germany.	56 men chosen at random from 2 noise regions defined in city of Bonn traffic study.	Emotional noise of ca 90 dB administered with mental arithmetic task for 5 min; 30 min exposure to experimental traffic noise ca 72 dB. No details.	Systolic and diastolic BP; Measurements not described.

Table 4-12: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Difficult to evaluate — experiment not described.	Significance levels, but no statistical tests reported.	"During exposure to both types of stressors the individuals having a positive family history of hypertension exhibited higher increase in systolic as well as in diastolic blood pressure when compared with those without family history." ... "With regard to noise a constant increase of diastolic blood pressure without a tendency of adaptation was observed." SBP rose but then dropped below baseline.	Findings in short term exposure consistent with several long-term observations.

Table 4-13: Yamamura et al. (1982).

<i>Summary of Laboratory Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Yamamura, K.; Maehara, N.; Sadamoto, T.; Harabuchi, I.; 1982.	Quasi-experimental. Laboratory; Japan.	8 healthy male college students selected once a week; total number not given.	8 noise exposure periods with total time being 14 h. Control period, 5 intermittent noise exposures and 2 steady state exposures described in detail.	TTS; saliva cortisol; Urinary 17-OHCH levels; methods described in detail.

Table 4-13: continued.

<i>Summary of Laboratory Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Subjects used as own controls; experimental conditions apparently well controlled.	Not stated in reported text.	"Urinary 17-OHCS did not increase with exposure to intermittent noise at 75 dBA for 14 h, and to steady state noise at 71 dBA and 73 dBA for 14 h, but did increase under exposure to intermittent noise at 80 dBA for 14 h." No differences were noted post noise exposure.	May suggest noise exposure limits if urinary 17-OHCS proves to be directly related to overt disease.

Table 4-14: Yazburskis (1971).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Yazburskis, B. I.; 1971.	Quasi-experimental; ultra-sonic lab under work conditions. Industry; Russia.	36 healthy workers; 21 males, 15 females; a: 10 at ultra-sonic disperser of 20 kc; b: 14 at 18 kc emitter; c: 12 at 8 kc emitter.	Source: RUZ ultra-sonic disperser of 20 kc, emitter of 18 kc. Intensity: a: up to 160 dB; b: up to 100 dB; c: up to 132 dB; Exposure in work 4-5 h/day. Instrumentation and measurement not given. Subjects working in ultra-sound 2-5 years.	ECG and BP; no details given.

Table 4-14: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
No evidence of randomization or statistical control of variables.	Was not stated in report text.	Workers engaged in the operation of high-power acoustic units displayed reduced heart rate, enlarged T-wave, diminished P and R waves and reduced systole:diastole ratio. Exercise caused a downward shift of the RS-T segment of the ischemic type immediately after work with ultrasound. BP fell towards the end of the workday and did not return to its initial level 5 min after an exercise tolerance test in all groups.	Design offers little support of an etiologic hypothesis especially in population-based studies.

5. Summary of Epidemiological Studies of the Effect of Road Traffic Noise on the Cardiovascular System

Table 5-1: Babisch and Gallacher (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Babisch, W.; Gallacher, J.E.J.; 1988. also Babisch, W.; Gallacher, J.E.J.; Elwood, P.C.; and Ising, H., 1988.	Cross-sectional data from prospective study; United Kingdom.	Caerphilly: 2512, total sample of 45-59 yr. old men on electoral roles; Speedwell: 2030, random sample of 45-59 yr. old men from health centers; homes randomly distributed in noise areas.	In Caerphilly long-term (2 days) and short-term (30 min) measures of A-weighted SPL (Leq) traffic noise at distance of 10 meters from center of streets. Men grouped into 5 dB categories of traffic noise Leq, 6-22 h, workday, ranging from 51-70 dBA. In Speedwell, similar short-term noise measures taken.	Clinical exams, ECG recordings, BP readings, fasting blood samples, medical and socio-demographic questionnaires provided by ongoing Collaborative Heart Study. Core standardized protocol and methods used.

Table 5-1: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Response rate 90%; major confounders of age, social class, relative body weight, smoking, alcohol, physical activity and family history adjusted in analysis. Room orientation, residence time, noise annoyance and occupational noise (for total sample) not considered.	Analysis of variance and covariance used; chi-square statistics for group differences. Mean and S.D. presented and graphed.	No associations of noise with prevalent IHD were found. There were higher levels of serum total cholesterol, HDL, and LDL cholesterol, blood glucose and plasma viscosity in the highest noise category (66-70 dBA); the effect for HDL-cholesterol was against the hypothesis that noise exposed subjects run a higher risk for ischemic heart disease. No statistical noise effects were detected for diastolic blood pressure, VLDL-cholesterol, blood clotting tests and leucocyte count. The relations of platelet count and systolic blood pressure with noise level were found to be inconsistent in the two samples.	Detailed documentation of traffic noise levels for each house and estimates of work noise from a sub-sample should allow valid inferences to be drawn from the longitudinal data which are yet to come. Room orientation, window opening habits, residence time and self-reported occupational noise exposure have been added to the prospective study.

Table 5-2: Babisch et al. (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Babisch, W.; Gallacher, J.E.J.; Bainton, D.; Elwood, P.C.; Ising, H.; Sweetnam, P.M.; Yarnell, J.W.G.; 1988.	Cross-sectional data from prospective study; United Kingdom.	Caerphilly: 2512, total sample of 45-59 yr. old men on electrical roles; Speedwell: 2030, random sample of 45-59 yr. old men from health centers; homes randomly distributed in noise areas.	In Caerphilly long-term (2 days) and short-term (30 min) measures of A-weighted SPL (Leq) traffic noise at distance of 10 meters from center of streets. Men grouped into 5 dB categories of traffic noise Leq, 6-22h, workday, ranging from 51-70 dBA. In Speedwell, similar short-term noise measures taken. Occupational noise: from random sample of 500 Caerphilly men plus 84 men living on noisy streets, 255 selected to wear personal noise dosimeters at work for 2-3 consecutive days.	Clinical exams, ECG recordings, BP readings, fasting blood samples, medical and socio-demographic questionnaires provided by ongoing Collaborative Heart Study. Core standardized protocol and methods used.

Table 5-2: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Response rate 90%; major confounders of age, social class, relative body weight, smoking, alcohol, physical activity and family history adjusted in analysis. Room orientation, residence time, noise annoyance and occupational noise (for total sample) not considered.	Analysis of variance and covariance used; chi-square statistics for group differences. Mean and S.D. presented and graphed.	Men in the highest noise category had the highest readings on blood lipids, plasma viscosity and glucose levels. BP readings were inconsistent between the 2 samples. Effects on BP and blood lipids were more pronounced in men also exposed to high levels of work noise.	Detailed documentation of traffic noise levels for each house and estimates of work noise from a sub-sample should allow valid inferences to be drawn from the longitudinal data which are yet to come. Room orientation, window opening habits, residence time and self-reported occupational noise exposure have been added to the prospective study.

Table 5-3: Drettner et al. (1975).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Drettner, B.; Hedstrand, H.; Klockhoff, I.; Svedberg, A.; 1975.	Cross-sectional. Community survey; Sweden.	100 males; age 30 yrs.; 388 reported history of noise exposure, 374 no exposure.	Questionnaire used to determine exposure; amount of hearing loss expressed as the sum of dB hearing loss of both ears at 5 frequencies -- levels 1000-6000 Hz.	Heart rate, BP measured after 10 min rest in supine position; cholesterol, triglycerides.

Table 5-3: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Non-response bias -- only 83.9% participation rate; self selection and selective recall; potential for misrepresentation of data with the repeated multiple correlations of all variables.	K-S test. No significant correlations between hearing loss and 10 risk factors except for smoking habits -- factors included SBP, DBP, heart rate.	"Among 92 who had smoked more than 10 cigarettes daily, but had not been exposed to noise, the amount of R-sided hearing loss was greater than in 105 individuals who had never smoked and had not been exposed to noise."	Hearing loss may not be appropriate surrogate for noise exposure when examining CVD outcome.

Table 5-4: Hedstrand et al. (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Hedstrand, H.; Drettner, B.; Klockhoff, I.; Svedberg, A.; 1977.	Cross-sectional. Community survey; Sweden.	393 males with noise induced hearing loss; 376 males with normal audiograms.	Surrogate measure -- noise-induced hearing loss defined as >65 dB at 3, 4, or 6 kHz and normal tone audiograms as <20 dB at all frequencies.	BP in supine position; hypertension defined as >160/100 mm Hg.

Table 5-4: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Age and sex controlled. No controlling for years of noise exposure, comorbidities, etc.	No associations noted. Means, standard deviations, and test of differences employed.	No significant differences in mean BP between hearing loss group and controls.	Evaluation is difficult because of limited information. May be continuation of 1975 study.

Table 5-5: Knipschild and Salle (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Knipschild, P.; Salle, H.; 1979.	Cross-sectional. Community with noisy streets in Netherlands.	399 living in noisy areas. 1342 living in quiet areas. Housewives of age 40-49 yrs.	Automobile noise, Noisy streets: Leq = 65-70 dBA, thus Ldn > 62.5 (A). Quiet streets: Leq = 55-60 dBA, thus Ldn < 62.5 (A).	Consultation with cardiologist. Hypertension SBP >160 and DBP >105 mm Hg at rest in sitting position; Angina pectoris; Ischemia on ECG by Minnesota Code.

Table S-5: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Data from screening program; states that attention was paid to possibly confounding factors (age, civil status, financial situation, smoking habits, weight, physical activity); self-selection; response rate differed between noisy and quiet areas.	No significant differences at $p = .05$ by Fisher's test for differences between high and low noise area on cardiologist consultations (6.3% in high vs. 9.7% in low), hypertension (5.8% in high vs. 9.4% in low) and angina (1.5% in high vs. 2.3% in low noise).	"This study showed no indication for a relationship between traffic noise and cardiovascular disease."	Cross-sectional nature of study with self-selection into the areas plus the relatively small differences in noise exposure of the two groups may result in observation of no differences in health due to noise.

Table 5-6: Meinhart and Renker (1979).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Meinhart, P.; Renker, U.; 1979.	Cross-sectional. Industrial community; Germany.	807 males with noise impaired hearing compared to 3948 selected to be representative of men seen in health clinic in the same district.	Noise from industrial area of Halle District; no information as to noise parameters and no hearing-impairment criteria given.	All circulatory diseases, functional heart disease, myocardium injury, hypertension, hypotension, peripheral circulation problems. Survey and clinic record data. No diagnostic criteria and no definitions given.

Table 5-6: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Ecologic fallacy; selection bias; age partially controlled by stratification; no evidence of control for co-morbidities, treatment or other variables.	Data presented as proportions for comparison; statistical tests apparently applied, but not described. Prevalence ratios implied for noise injured vs. controls: Myocardial injury = 2.0; Hypertension: 12-40 yr. olds = 7.6 40-65 yr. olds = 9.7 65+ yr. olds = 8.2.	The prevalence of myocardial injuries was about twice as high for the noise-injured group as the controls; hypertension was higher among noise-impaired at all ages except the 65 and older; for all age classes, the frequency of heart circulatory diseases and especially hypertension and hypotension began rising after five years employment and rose precipitously after 20 years work; no differences between the groups were observed for functional heart-circulatory diseases or peripheral perfusion disturbances. "The steady noise influence is an important factor of the origin of heart-circulatory diseases."	Noise exposure measure poor. Probably represents a mixture of exposed and non-exposed industrial workers. Confounding variables uncontrolled which reduces value of study.

Table 5-7: Neus et al. (1983a).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Neus, H.; Ruddel, H.; Schulte, W.; 1983.	Cross-sectional survey. Community (traffic noise); Bonn, Germany. Random sample from complete list of inhabitants.	931 originally with 919 with complete data: 447 living in noisy area and 442 in the control area.	Residential area with road traffic noise Leq > 73 dBA an a control area with noise Leq < 51 dBA. Measures of subjective reaction (annoyance) and noise attitudes (health, sensitivity and adaptability) developed by factor analysis.	Medical history of treatment for hypertension and other problems. Standardized questionnaire.

Table 5-7: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Misclassification into treated or untreated groups potentially high with self report.	Association between low annoyance and low use of hypertensive treatment in control area only. Correlations and graphing of mean scores given.	"There was no relationship between the noise attitudes and the frequency of anti-hypertensive treatment, either in the noise area or the control area." However... "within the subjects not feeling annoyed by the traffic noise and living in the control area the incidence of antihypertensive treatment was much lower than in all other groups ($p = .007$). 50.2% of all subjects living in the control area were in this group whereas only 9.6% of the subjects in the noisy area had similar scores for noise tolerability."	Re-analysis of Bonn Traffic Study to examine relationship between subjective factors and blood pressure. Findings indicate that subjective factors do not modify risk of hypertension in noisy areas whereas they may act in less noisy conditions.

Table 5-8: Neus et al. (1983b).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Neus, H.; Ruddel, H.; Schulte, W.; Eiff, A.W. von; 1983.	Followup of N = 28 and N = 36 normotensives of Bonn Traffic Noise Study, for 16 & 27 months. Community traffic noise; Bonn, Germany.	N = 28 followed for 16 months; N = 36 followed for 27 months. Distri- bution: 14 from noisy area and 22 from control area.	High traffic area = 63-73 dBA noise level; Low traffic area = max of 50 dBA. Subjects lived in same residence throughout study period.	Clinical investigation. BP taken in afternoon after 10 min rest in recumbent position. Quality of data unknown.

Table 5-8: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Sample sizes small. No information given on subject selection, comparability or control for confounding.	Changes in diastolic BP greater in noisy area than in control area. BP values not given. Difference statistically significant.	In both groups, N = 28 and N = 36, "the residents of the noisy area exhibited greater increases of DBP (5.1 mm Hg and 10.7 mm Hg; $p = 0.0355$ and $p = 0.006$) as compared to the control group. This effect was about the same in both sexes. Therefore there is increasing evidence for a pathogenetic influence of environmental noise on blood pressure."	Sample selection and sample size makes study of questionable value; increases in BP over short time period unusually large compared to industrial noise data.

Table 5-9: Otten et al. (1988).

Summary of Epidemiologic Studies				
Citation Source	Study Type and Site	Sample Size	Noise Exposure Measure - Quality Long vs. Short Term	Cardiovascular Response Measure - Quality
Otten, H.; Schulte, W.; Eiff, A.W. von; 1988.	3 year longitudinal study; Bonn, Germany.	97 women and 95 men, 20-35 yrs. of age who moved to the respective noise areas as defined in the Bonn traffic noise study, at least 6 months prior to screening and who had no disorders of the cardiovascular system, no longer than 2 months absence from residence per yr. and no planned moving.	Average noise level Leq, 6-22h, front of buildings. Noisy area: noise level greater than 63 dBA, range 63-78 dBA. Control area: noise level less than 55 dBA.	BP in seated position by random-zero-sphyg. assessed twice a day at clinical exams; ECGs, family history, mental and physical stress testing, routine blood parameters, noise sensitivity, noise annoyance and health behaviors -- assessed at entry, 6, 12, 18, 24, and 36 months. Procedures not described.

Table 5-9: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
High migration rate -- only 21% had not moved after 3 years.	Means, S.D.s, and p values reported. For men 4 mm Hg increase in SPB and a 3 mm Hg increase in DBP.	For males who did not move, both systolic and diastolic BP increased throughout the followup period. In males, an increase in noise sensitivity and annoyance was significantly correlated with increase in BP. No differences were observed in BP changes for women in the noisy and control areas; females living in noisier area described noise as more intolerable and moved out of the area earlier than women living in the control area.	High attrition rate raises questions as to study validity and demonstrates a major difficulty in conducting longitudinal studies of traffic noise.

Table 5-10: Pulles et al. (1988).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Pulles, T.; Biesiot, W.; Stewart, R.; 1988.	Cross-sectional around Leeuwarden and Twenthe military airbases and in the cities of Amsterdam and Groningen; The Netherlands.	830 subjects, 20-55 yrs. of age.	Noise levels (LAeq) expressed as Kosten Eenheden (KE) for aircraft noise categorized as greater than 56 KE, 51-55, 46-50, 41-45, 36-40 and less than 36 KE; for traffic noise greater than 71 dBA, 66-70, 61-65, 56-60, 51-55 and less than 51 dBA.	ECG readings coded using Minnesota Code; weight, height, blood parameters (ureum, creatinine, glucose, hemoglobin, cholesterol and HDL-cholesterol), 15 min medical exam with BP measured at entrance and just before end of exam; 1 1/2 h interview on age, sex, annoyance, symptom checklist, sleep quality, disturbance and stress due to noise, coping strategies and perceived locus of control.

Table 5-10: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
27% refusal rate. Confounders apparently adjusted, known cases of disease excluded.	Stepwise multivariate linear regressions of BP measurements showed 80% of variance unexplained.	"..we concluded that no significant dependence of blood pressure upon environmental noise is present in this survey. Apparently noise induced blood pressure changes are too small to be detected in a medical field survey with (two times) 400 subjects." Individuals exhibiting a coping style based upon avoidance showed a higher noise sensitivity than those using other coping styles.	It is not clear that a comparison of aircraft and traffic noise exposure in a cross-sectional study addresses the question of interest to NSBIT.

Table 5-11: Takala et al. (1977).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Takala, J.; Varke, E.; Vaheri, E.; Sievers, K.; 1977.	Cross-sectional. Community survey; Finland.	32 men exposed to noise and with hearing loss; 67 men with normal hearing.	Noise exposure hearing loss defined as thresholds of hearing 65 dB or more at 4000 and 6000 Hz; normals at 20 dB.	Single BP reading, right arm, sitting for 3-5 min, 5th phase DBP recorded. Hypertension defined as > 160/100 mm Hg.

Table 5-11: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
93% participation rate. No variable, including age, controlled in the analysis. Volunteer and migration bias possible.	No association noted. Student's t-test.	No statistically significant differences in SBP nor DBP between the normal hearing and noise defect group.	Hearing loss may not be good indicator of noise exposure.

Table 5-12: Von Eiff and Neus (1980).

<i>Summary of Epidemiologic Studies</i>				
<i>Citation Source</i>	<i>Study Type and Site</i>	<i>Sample Size</i>	<i>Noise Exposure Measure - Quality Long vs. Short Term</i>	<i>Cardiovascular Response Measure - Quality</i>
Von Eiff, A. W.; Neus, H.; 1980.	Cross-sectional Community survey (Traffic noise); Bonn, Germany.	458 men; 437 women; ages 20- 59 selected by random household sur- vey.	High traffic area = 63-73 dBA noise level; Low traffic area = 50 dBA. Exposed for minimum of 3 yrs.	Hypertension and treat- ment determined by questionnaire.

Table 5-12: continued.

<i>Summary of Epidemiologic Studies - continued</i>			
<i>Bias and Potential Bias Due to Confounding</i>	<i>Strength of Association(s) and Statistical Tests</i>	<i>Summary of Findings as Reported by Author</i>	<i>Comments</i>
Ecologic fallacy, measurement bias.	22.8% people in noisy area treated for hypertension compared to 14.6% in control area; prevalence ratio 1.6 ($p < 0.05$).	"Noise is very troublesome on streets with high volume traffic. Specific high blood pressure as a treated disease was mentioned significantly more often in the high noise area than in the low noise area." ... "The data on men between the ages of 20 and 39 who lived in the loud-noise area revealed hypertensive treatment to be dependent upon length of residence. This was not the case in the low-noise area." ... "The results of the investigation justify a prospective, interdisciplinary epidemiological study, in which the physical measurements are correlated with the measured blood pressure data."	Study poor for judging effects of noise on CVD because of self-report of hypertension and general noise measure.

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