

8 CARDIOVASCULAR DISORDERS

Concerns about chronic cardiovascular changes resulting from exposure to ELF fields originated from descriptions in the 1960s and early 1970s of the symptoms among Russian high voltage switchyard operators and workers (Asanova & Rakov, 1966; 1972). Further studies carried out in the Russian Federation in the 1980's and 90's reported various functional changes in the cardiovascular system, such as hypertension in workers in 500 kV, 750 kV and 1150 kV power installations (Rubtsova, Tikhonova & Gurvich, 1999). More recent investigations have focused mainly on direct cardiac effects of EMF exposure, mostly related to heart rate variability and subsequent acute cardiovascular events.

8.1 Acute effects

Current flow through the human body appears to be necessary in order to result in major cardiovascular effects from EMF exposure, such as the effects due to electric shock (Hocking, 1994). Normally electric shock requires direct electrical contact of a conductor with the body. It may, however, also occur if the body is exposed to very strong electric or magnetic fields (Foster, 1992). Minor effects have also been reported in other situations of low-level EMF exposure. Most human studies on EMF effects on the cardiovascular system have focused on acute rather than long-term effects.

8.1.1 *Electrocardiogram changes, heart rate, and heart rate variability*

Silny (1981) found no effects on the electrocardiogram (ECG) in 100 persons exposed to time-varying magnetic fields (5 Hz to 1 kHz, less than 100 mT). Hauf (1989) has performed human tests exposing the subjects to 50-Hz fields (20 kV m⁻¹ and 0.3 mT) with a current of 500 µA passing through the body. The experiments did not indicate any significant effects on the heart rate of the subjects. In another study, there were no significant changes in heart rate in persons exposed locally to pulsed magnetic fields up to 2.2 µT by transcranial magnetic stimulation (TMS) (see 5.2.2) (Chokroverty et al., 1995).

In a series of studies carried out by the Midwest Research Institute in the US, effects of ELF fields on the heart rate in humans have been investigated. In a set of studies by Graham et al. (1994), subjects were exposed to different levels of combined electric and magnetic fields (low: 6 kV m⁻¹ and 10 µT; medium: 9 kV m⁻¹ and 20 µT; high: 12 kV m⁻¹ and 30 µT). In the medium group a significantly decreased heart rate was observed, while in the other groups no change was found. In another study by the same group, six physiological parameters were examined at five sampling points with and without exercise (Maresh et al., 1988). During no-exercise sessions the cardiac interbeat interval was increased at two sampling points when subjects were exposed to 60-Hz fields. No other difference between the sham and exposed groups was found. A similar effect was found by another study of the same group (Cook et al., 1992).

In a replication study by Whittington, Podd & Rapley (1996), no effect of a higher magnetic field (100 μT , 50 Hz) on heart rate or blood pressure was found, however. Humans exposed for 1 hour to EMFs under a 400-kV power line exhibited no difference in pulse rate during autonomic function tests (Korpinen & Partanen, 1994a). The same researchers reported that exposure to 50-Hz fields (up to 10 kV m^{-1} and 15 μT for several hours) did not affect the incidence of extrasystoles or arrhythmia (Korpinen & Partanen, 1994b; Korpinen, Partanen & Uusitalo, 1993). A 2% decrease of heart rate was also observed by Sait et al. (1999) after a 100 to 150-s exposure to a 50 Hz, 28 μT magnetic field. From these studies, it can be noted that the positive but inconsistent results from the US Midwest Research Institute of reduced heart rates after exposure to EMF have not been confirmed by other studies. The heart rate effects, where such have been found, are generally of small magnitude, and can currently not serve as an indicator of an acute health effect (Hauf, 1982).

Sastre, Cook & Graham (1998) performed studies on heart rate variability (HRV) of 77 healthy men exposed to 60-Hz magnetic fields of 14.1 μT or 28.3 μT . Statistically significant alterations in HRV were observed during intermittent exposure to the higher field strength, while no effects occurred at the lower field strengths or when the exposure was continuous. A reduction in the power ratio in the low band of the HRV spectra (0.02–0.15 Hz) to the high band (0.16–1.0 Hz) was also observed by Sait, Wood & Sadafi (1999) after a 100 to 150-s exposure to a 50 Hz, 28 μT magnetic field with the exposure conditions already mentioned. In two studies on the same issue, HRV was evaluated during intermittent exposure to 28.3 μT (Graham, Cook & Riffle, 1997; 1998). In the latter study, three different frequencies were used (16, 40, 60 Hz). Exposure to 16 Hz was associated with significant alterations of the HRV spectrum. However, in a later pooled analysis of several studies conducted at the same institute, Graham et al. (1999) reported that this effect occurred only in studies where hourly blood sampling was performed. The authors hypothesised that blood sampling altered the level of subject arousal, allowing EMF interaction to affect HRV. A multi-study analysis indicates that the effect on HRV happens when EMF exposure is accompanied by increases in physiologic arousal, stress, or a disturbance in sleep, such as blood collection, but not otherwise (Graham et al. 2000a).

Recently, Graham et al. (2000e) performed studies using a much higher magnetic field (127.3 μT) and both continuous and intermittent exposure. No alterations in HRV were observed by either exposure condition, and the researchers concluded that, taking into account earlier reports, direct excitation of the human heart is extremely unlikely under exposure to magnetic fields lower than 127.3 μT .

A summary of the studies into the effects of ELF fields on ECG and heart rate is given in Table 57.

Table 57. Studies of ECG and heart rates after ELF exposure

Test	Exposure	Response	Comments	Authors
ECG	5 Hz–1 kHz < 100 mT	No change.		Silny, 1981
Cardiac inter-beat interval	60 Hz 9 kV m ⁻¹ , 16 A m ⁻¹	Longer cardiac interbeat interval.		Maresh et al., 1988
Pulse rate	50 Hz 20 kV m ⁻¹ , 300 μT or combined + 200 and 500 μA- currents at 50 Hz	No change.	Protocol looks confused, mixing haematological and physiological parameters.	Hauf, 1989
Pain, ECG, heart rate	Not defined: magneto-stimulation	No pain, no change in ECG and heart rate.	No dosimetry	Nagano et al., 1991
Interbeat interval before, during and after exposure 30 male subjects	60 Hz 9 kV m ⁻¹ , 20 μT 2x3 h day ⁻¹ , 4 days	Interbeat interval longer during and immediately after exposure.	Double-blind, counterbalanced study.	Cook et al., 1992
Extrasystoles, pulse rate	50 Hz 0.14–10 kV m ⁻¹ , 1.0–5.4 μT 0.5 h – few hours	No more extrasystoles in the field than out the field. Small decrease in pulse rate can be due to changes in work load.		Korpinen, Partanen & Uusitalo, 1993 Korpinen & Partanen, 1994b
Interbeat interval before, during and after exposure 54 male subjects	60 Hz 6 kV m ⁻¹ , 10 μT 9 kV m ⁻¹ , 20 μT 12 kV m ⁻¹ , 30 μT	Interbeat interval longer during and immediately after exposure only at the intermediate level of exposure.	Double-blind, counterbalanced study.	Graham et al., 1994
Pulse rate	50 Hz 3.5–4.3 kV m ⁻¹ , 1.4–6.6 μT 1 h	No change in pulse rate.		Korpinen & Partanen, 1994a
ECG, systolic and diastolic blood pressure, orthostatic test, valsalva maneuver, deep breathing test	50 Hz 3.5–4.3 kV m ⁻¹ , 1.4–6.6 μT 1 h	No change.	CV autonomic tests were performed 0.5 h before and after the 1 h-exposure.	Korpinen & Partanen, 1995
Heart rate	pulsed magnetic fields of up to 2.2 μT used in TMS	No change		Chokroverty et al., 1995

Table 57. Continued

Heart rate and blood pressure 100 (male and female) subjects	50 Hz 100 μ T, intermittent 9 min	No effect.	Double-blind, counterbalanced study.	Whittington, Podd & Rapley, 1996
Heart rate variability 33 male subjects (exp 1) 40 male subjects (exps 2 and 3)	60 Hz 1 or 20 μ T, intermittent Overnight (23.00–07.00)	Altered heart rate variability at 20 μ T, but not at 1 μ T.	Double-blind (all studies) and counterbalanced (exps 2 and 3).	Graham, Cook & Riffle, 1997 Sastre, Cook & Graham, 1998
Heart rate and heart rate variability 18 (pilot study) and 35 subjects (follow-up study)	50 Hz 28 μ T, sinusoidal continuous or intermittent (15 s on-off), or square-wave for 100–150 s	Altered heart rate and heart rate variability by continuous sinusoidal fields, but not by intermittent or square-wave fields.	Blind and counterbalanced.	Sait, Wood & Sadafi, 1999
Heart rate variability 172 male subjects (pooled from 7 studies)	60 Hz 28.3 or 127.3 μ T, intermittent or continuous Overnight	Altered heart rate variability in some conditions.	Blood sampling (for another study) at night was critical for this effect. Double-blind studies.	Graham et al., 1999; 2000e; 2000d

8.1.2 Blood pressure

No changes were observed in blood pressure, neither in subjects exposed to a 50 Hz field (20 kV m⁻¹ and 0.3 mT) with a current of 500 μ A passing through the body (Hauf, 1989), neither in humans exposed for 1 hour to EMFs under a 400-kV power line (Korpinen & Partanen, 1996), nor in persons exposed locally to pulsed magnetic fields up to 2.2 μ T by TMS (Chokroverty et al., 1995). These studies are summarized in Table 58.

Table 58. Studies of blood pressure after ELF exposure

Test	Exposure	Response	Comments	Authors
Blood pressure	50 Hz 20 kV m ⁻¹ , 300 μ T or combined + 200 and 500 μ A-currents at 50 Hz	No change.		Hauf, 1989
Blood pressure	pulsed magnetic fields of up to 2.2 μ T used in TMS	No change.		Chokroverty et al., 1995
Blood pressure	50 Hz 3.5–4.3 kV m ⁻¹ , 1.4– 6.6 μ T 1 h	No change.		Korpinen & Partanen, 1996

8.2 Long-term effects

Knave et al. (1979) and Stopps, Janischewskyj & Alcock (1979) found no significant effects on cardiovascular function in male workers who were exposed occupationally for more than 5 years to electric fields from 400 kV power lines. Checcucci (1985) found no effect on the cardiovascular system in 1200 workers at high-voltage railway substations (1–4.6 kV m⁻¹ and 4–15 μT). In a health survey of 627 railway high-voltage substation workers, Baroncelli et al. (1986) found no difference in the ECG between exposed and control groups. Table 59 gives a summary of these studies.

Table 59. Studies of cardiovascular effects after long-term ELF exposure

Test	Exposure	Response	Comments	Authors
CV parameters and diseases	50 Hz 400 kV power lines > 5 years	No effect.	Better psychologic performance linked to higher education level, and lower fertility predominant on boys, anterior to the exposure period.	Knave et al., 1979
CV parameters and diseases	50 Hz 400 kV power lines > 5 years	No effect.		Stopps, Janischewskyj & Alcock, 1979
CV parameters and diseases	1–4.6 kV m ⁻¹ , 4–15 μT	No effect.		Checcucci, 1985
Haematology electro-cardiogram	50 Hz HV railway substation < 5 kV m ⁻¹ , 15 μT 0, 1, 10 and 20 h / week	No effect.		Baroncelli et al., 1986

Based on the idea put forth by Sastre, Savitz (1999) hypothesized an association between exposure to EMF and cardiovascular disease. This hypothesis was based on two independent lines of evidence. The first was experimental data on heart rate variability described above in which intermittent 60-Hz magnetic fields were found to reduce the normal HRV (Sastre, Cook & Graham, 1998). The second came from several prospective cohort studies which indicated that reductions in some components of the HRV increase the risk for: (1) heart disease (Dekker et al., 1997; Liao et al., 1997; Martin et al., 1987; Tsuji et al., 1996); (2) overall mortality rate in survivors of myocardial infarction (Kleiger et al., 1987; Lombardi et al., 1987; Vaishnav et al., 1994); and (3) risk for sudden cardiovascular death (Malik, Farrell

& Camm, 1990). Thus, they postulated that occupational exposure to EMF will increase the risk for cardiac arrhythmia-related conditions and acute myocardial infarction, but not for chronic cardiovascular disease.

Several studies, published before the specific hypothesis of an effect on HRV was suggested, examined general cardiovascular mortality in relation to EMF (Table 60). In a Canadian retrospective cohort study of 21744 men employed in an electrical utility company in the province of Quebec between 1 January 1970 and 31 December 1988, the standardised mortality ratio (SMR) for circulatory diseases was below 1 in all job categories and with all exposure levels to magnetic fields, electric fields and pulsed electromagnetic fields (Baris et al., 1996b). Exposure information was obtained from a job-exposure matrix (JEM) constructed on the basis of the last job held in the industry. The JEM was constructed for a larger study of employees in the utility industry in Canada and France (Theriault et al., 1994) and included a measurement protocol of magnetic fields, electric fields and pulsed electromagnetic fields among 466 employees for one week. Among employees exposed to magnetic fields $> 0.16 \mu\text{T}$, 137 persons died from circulatory diseases (adjusted RR = 0.91; 95% CI 0.73–1.14). The SMRs, when using electric fields and pulsed electromagnetic fields as the exposure variables, were close to those observed for exposure to magnetic fields. [It must be noted that no definition of diagnoses included as “circulatory diseases” are given. Likewise, there is no reference to the quality of the Canadian mortality statistics in this paper, or to what extent it is the underlying cause of death, the contributory cause of death or a combination of the two, which is used as the outcome measurement.]

A retrospective cohort study from the US (Kelsh & Sahl, 1997) of 40335 men and women employed between 1960 and 1991 in a Californian utility company observed a significantly reduced SMR of 0.62 (95% CI 0.59–0.65) for both sexes combined. This was based on a comparison with the general population of the geographical area of the utility company and included 1561 cases of cardiovascular death (ICD-9, 3900–4489). The risk estimates in different occupational categories were very close to each other and all but the category “Meter readers/Field service” had significantly decreased mortality. Exposure information was primarily based on job title and work environment and included no measurement protocol. Each employee was assigned to one of seven categories based on the occupation held for the longest time. Information on mortality was obtained from three public sources and also from company records, indicating some underreporting to the primary sources. In internal analyses conducted across employment categories and using administrative employees as a reference group, mortality from “Major Cardiovascular” (category not defined) was significantly increased in all categories, the highest RR being 1.71 (95% CI 1.13–2.58) in the “Meter Reader/Field service” category. When stratifying the internal analyses by work employment period (before or after 1960) no clear pattern emerged. [A clear healthy worker effect seems to explain the results of the external analyses. However, no clear explanation can be given for the increased risk of death from “Major Cardiovascular” as no precise and

detailed information was available for exposure to known risk factors for these disorders (tobacco smoking, alcohol consumption or physical activity).] A re-analysis of this study cohort (Sahl et al., 2002) is described below.

In a nationwide retrospective cohort study in Denmark of 21236 men employed in utility companies between 1900 and 1993 the causes of death were ascertained for 1 January 1974 through 31 December 1993, and cause-specific mortality was analysed by latency and estimated levels of exposure to 50 Hz electromagnetic fields (Johansen & Olsen, 1998b). A dedicated job-exposure matrix was designed that distinguished between 25 different job titles held by utility company employees and 19 work areas within this industry. Each of the 475 combinations of job title and work area was assigned an average level of exposure to 50 Hz EMF during a working day, which in turn was grouped into five categories of exposure to ELF fields. The conversion program was constructed by four engineers from the utility companies experienced in the planning and operation of electric utilities in Denmark. The construction of the matrix was based partly on a series of 196 24-hour measurements of 50 Hz EMF among 129 employees in six Danish utility companies and partly on judgements. The individual exposure assignment was based on the characteristics of the first employment held. Overall, 3540 deaths were observed as compared with 3709 expected from national mortality rates, yielding a standardized mortality ratio of 0.96 (95% CI 0.93–0.99). Overall mortality caused by acute myocardial infarction (ICD-8, 410) yielded an SMR of 0.95 (95% CI 0.9–1.0) based on 713 cases. SMR for cardiac sclerosis (ICD-8, 412) was 0.9 (95% CI 0.8–1.0) based on 300 cases and for mortality caused by other heart disorders (ICD-8, 394–402; 413; 420–429; 450 and 782) the SMR was 0.9 (95% CI 0.8–1.0). When analysing the cause-specific mortality by time since first employment or categories of estimated EMF exposure no increased mortality for these disorders appeared. [No information was available about known risk factors for cardiovascular disease. The exposure assessment in this study is based on few measurements and historical records and one cannot exclude that misclassification has taken place. In addition the use of mortality records as the measurement of the outcome may have caused some additional misclassification as the autopsy rates in Denmark has been decreasing in the study period. This study only use external comparisons as the method of analyses and this does not take into account a possible healthy worker effect.] This study cohort was later followed up for risk of pacemaker implantation – summarized below (Johansen et al., 2002).

In a recent follow-up study of Thai employees of the Electricity Generating Authority of Thailand, changes in levels of vascular risk factors over 12 years, and the associations of baseline risk factors with mortality were examined (Sritara et al., 2003). Over the 12-year period, levels of all major vascular risk factors, apart from smoking, worsened in this occupational study population. Although the authors note that the increases appear to exceed those expected from ageing of the cohort alone, very little regarding the impact of exposure on disease and mortality can be inferred from this study.

In the first study conducted with the specific aim to test the hypothesis of an association between EMF and acute cardiovascular disease risk another US retrospective cohort study of utility workers (Savitz et al., 1999) was analysed. It included 138903 men employed for six months or more between 1950 and 1986. The authors report a significantly increased risk of mortality from arrhythmia-related conditions and acute myocardial infarction among workers with long duration of work (with rate ratios of 1.4–1.5 for the longest employment intervals) and with high exposure to magnetic fields (with rate ratios of 1.6–2.4 in the highest exposure category). As postulated a positive association was seen for acute myocardial infarction (AMI) and an inverse association for chronic cardiovascular disease (CVD). The EMF exposure categories were based on 2842 complete work shift time weighted average magnetic field exposure measurements and information on outcome (death certificate) was obtained from 97% of the deceased men. This cohort was reanalysed by Van Wijngaarden et al. (2001a), however, not providing further information on the hypothesis of an association between EMF exposure and mortality of arrhythmia-related cardiovascular diseases or AMI. Finkelstein (1999) questioned the use of death certificates as a source of information on a diagnosis of loss of autonomic cardiovascular control by Savitz et al. (1999) and pointed out that etiologic conclusions could not be drawn on the basis of death certificate codes (Finkelstein, 1999). Problems in using subtypes of CVD as coded on death certificates, which are of uncertain validity and reliability, are particularly evident in this study where the excess of deaths in acute cardiovascular categories coincides with a deficit of deaths in chronic categories for all exposure groups except the highest group. This either suggests specificity of effect or miscoding. In addition, they could not examine the temporal relation between exposure and outcome in any detail other than to look at jobs (with their estimated mean) and death, but not diagnosis. They also lacked information on other CVD risk factors.

The hypothesis of an association between exposure to EMF and the risk for arrhythmia-related cardiovascular disorders was further addressed in the Danish cohort of utility workers (Johansen et al., 2002). The incidence of severe cardiac arrhythmia as indicated by the need for a pacemaker was investigated by a linkage to the nationwide, population-based Danish Pacemaker Register. The study identified all cases of pacemaker implantation among 24056 male utility workers between 1982 and 2000 and compared this number with the corresponding numbers in the general population. In addition, the data on utility workers was fitted to a multiplicative Poisson regression model in relation to estimated levels of exposure to 50 Hz electromagnetic fields. Overall, the risk was not increased for severe cardiac arrhythmia among employees in the utility companies, based on 135 men with pacemakers with 140 expected, yielding a risk estimate of 0.96 (95% CI 0.8 –1.1). No clear dose-response pattern emerged with increasing level of exposure to EMF or duration of employment. [The study investigated the risk of a morbidity, which leads to the implantation of a pacemaker. One may also consider that other arrhythmias, which are not associated with a pacemaker implantation, and thus not included here, may be associated with the

exposure under study. Furthermore the files of the workers were established years before the events reported to the Danish Pacemaker Registry and were supported by personal data from the nationwide, compulsory pension fund and the public payroll system kept for administrative purposes. The completeness of these registries of employments and pacemaker implantation highly reduces the likelihood of selection and information bias. Comparisons with the general population might have been influenced by the healthy worker effect. This was not the case, however, in the internal comparisons within the cohort of different exposure groups. No control of confounding for other risk factors was made. As mentioned before, the exposure assessment in this study is based on few measurements and historical records and one cannot exclude that misclassification has taken place.]

A re-analysis of the data reported by Kelsh & Sahl (1997) did not confirm the findings from Savitz' study (Sahl et al., 2002). In this cohort of 35391 male utility workers in southern California, USA, with follow-up from 1960 to 1992, 369 cases of chronic coronary heart disease and 407 cases of myocardial infarct were identified. For cumulative exposure, adjusting for socioeconomic factors, no association was observed with mortality from acute myocardial infarction (rate ratio per $\mu\text{T-year} = 1.01$, 95% CI 0.99–1.02) or chronic cardiovascular heart disease (rate ratio per $\mu\text{T-year} = 1.00$, 95% CI 0.99–1.02). In this study (Sahl et al., 2002) the analyses were performed by the same methods and analytical models as those used by Savitz et al. (1999) in an attempt to conduct as a close a replication as possible of the Savitz work. In the previous study (Kelsh & Sahl, 1997) men aged > 80 years were excluded, EMF exposure was defined on the basis of the worker's usual occupation as opposed to a detailed occupational history, and different reference groups were used in the internal analyses. One group with a significantly increased mortality from cardiovascular disease, but with low EMF exposure, was assigned to the reference group in the present study (Sahl et al., 2002). This might explain some of the observed changes in risk estimates. [Weaknesses include the inability to control for potentially important factors that may influence mortality due to cardiovascular disease, the use of death certificates to identify the cause of death, and the reliability of the distinction between AMI and chronic cardiovascular heart disease as recorded on the death certificate. Strengths include large number of exposed, improved exposure assessment and an attempt to indirectly examine smoking as a potential confounder.]

A population-based case-control study from Sweden (Ahlbom et al., 2004) investigating risk factors for acute myocardial infarction in the city of Stockholm included information on occupational EMF exposure based on job titles one, five, and ten years prior to diagnosis. The analysis was restricted to the 695 cases and 1133 controls with information on job titles. Of these, 595 cases and 949 controls had jobs that were common enough to have been classified according to a previously developed JEM. The study used two approaches to classify exposure. First, specific individual job titles with presumed elevated EMF exposure were investigated and secondly, the subjects were classified according to a JEM. Both analytical approaches

revealed risk estimates for acute myocardial infarction below or close to one. [The strengths of this study include the fact that it is population based, looks at morbidity rather than mortality, the high participation rates and finally the high validity of the AMI diagnoses. This study is the first to include information on potential confounders, in particular blood pressure, serum cholesterol, socio-economic status, and cigarette smoking. The limitations of this study include the use of the previously developed JEM. Although this has been utilized in several other studies and seemingly performed well, its sensitivity and specificity in relation to classification of EMF exposure are not assessed. Thus, it is not entirely inconceivable that non-differential recall bias plays a role. On the other hand, several specific job titles were also analyzed and gave consistent results.]

Another Swedish study of the association between EMF exposure and mortality from heart diseases utilised data from the Swedish twin registry including close to 28 000 twins from two different cohorts of twins in Sweden (Håkansson et al., 2003). These twins were interviewed in 1967 and 1973 and at that time their occupation was recorded. In addition the interview covered information on smoking, alcohol consumption, level of physical activity and body mass index. The analyses were based on the primary and contributory cause of death followed up until 1996 utilizing the previously described exposure matrix (Ahlbom et al., 2004) adjusted for the previously mentioned risk factors. The results did not show an overall increased risk for arrhythmia related death, ischemic heart disease other than AMI or atherosclerosis. A non-significantly increased risk for AMI was observed in the highest exposure group (RR = 1.3; 95% CI 0.9–1.9; exposure level > 0.3 μ T). Since this study was conducted within a twin cohort a sub-analysis that took into account the twin information was conducted. In this analysis the authors observed a larger increase in risk for AMI and magnetic fields in genetically susceptible subgroups (i.e. among the monozygotic twins, one of whom previously had an AMI) for which there is no obvious explanation. [Note that this study included subjects from the general population and its exposure assessment was based on a single question on the subjects' "main occupation" at one point in time in the past.]

The latest study of utility workers examined a cohort of 83 997 workers in the UK employed for at least six months between 1973 and 1982 and followed up from 1973 to 1997 (Sorahan & Nichols, 2004). Estimates were obtained for lifetime exposure and exposures accumulated during the most recent 5 years using comprehensive occupational magnetic field exposure assessment. Causes of death (both underlying and contributing) from cardiovascular diseases were grouped into four categories: (1) arrhythmia related, (2) acute myocardial infarction, (3) atherosclerosis related, and (4) chronic/sub-chronic coronary heart disease. Poisson regression modeling with adjustments for age, sex, calendar time, beginning year of employment, and an indicator for socioeconomic status was used. Only for arrhythmia-related death, the relative risk estimates were greater than one for all exposure categories, however, the estimates were based on small numbers, showed no monotonic trend with increasing exposure, and were not statisti-

cally significant. (RR per 10 μ T-years = 1.1; 95% CI 0.8–1.6). [Of note in this study is the exposure assessment, which was based on elaborate methods that considered individual job histories, job environments, and local sources of magnetic fields in individual job locations, which is likely to have reduced misclassification.]

Table 60. Studies of general cardiovascular mortality in relation to EMF

Population	Design	Exposure	Outcome	Size	Results (95% CI)	Authors
Workers employed in electrical company between 1970–1988	Cohort SMR and internal comparisons	Member of cohort, job-exposure matrix	Circulatory disease mortality	Circulatory deaths: 137 Cohort: 21 744	Highest exposure category: SMR = 0.63 (0.53–0.74) RR = 0.91 (0.73–1.14)	Baris et al., 1996b
Utility workers, employed between 1960–1991, followed - 1992	Cohort SMR and internal comparisons	Member of cohort, certain occupational categories	CVD mortality	CVD deaths: 1561 Cohort: 40 335	Total cohort: SMR = 0.62 (0.59–0.65) Linemen: RR = 1.42 (1.18–1.71)	Kelsh & Sahl, 1997
Male utility workers, employed 3 months between 1990–1993, followed 1974–1993	Cohort SMR	Member of cohort, classification of workplaces based on measurements	CVD mortality	CVD deaths: 713 Cohort: 21 236	High exposure work-place, AMI: SMR = 0.095 (0.9–1.0)	Johansen & Olsen, 1998b
Male utility workers, employed 6 months between 1950–1986, followed - 1988	Cohort SMR and internal comparisons	Duration of work in jobs with elevated EMF	CVD mortality	CVD deaths: 6802 Cohort: 138 903	Highest μ T-year category, AMI: RR = 1.62 (1.45–1.82) Chronic CHD: RR = 1.0 (0.86–1.77)	Savitz et al., 1999
Male utility workers, employed 3 months between 1990–1993, followed 1974–1993	Cohort SMR	Member of cohort, classification of workplaces based on measurements	Pace-maker implantation	Implants: 135 Cohort: 24 056	Highest exposure category total SIR = 1.00 (0.6–1.5)	Johansen et al., 2002

Table 60. Continued

Male utility workers in Kelsh & Sahl, 1997	Cohort SMR and internal comparisons	Duration of work in jobs with elevated EMF	CVD mortality	AMI deaths: 407 CCHD deaths: 369 Cohort: 35 391	Highest μ T-year category, AMI: RR = 0.99 (0.65–1.51) Chronic CHD: RR = 1.19 (0.79–1.77)	Sahl et al., 2002
Swedish twins responding to job questionnaire in 1967 or 1973	Cohort Cox analysis	Job-exposure matrix	CVD mortality	Twin cohort: 27 790	Highest exposure group, AMI: RR = 1.3 (0.9–1.9)	Håkansson et al., 2003
Male population of Stockholm 1992–1993	Population-based case-control	Job titles, job-exposure matrix	AMI morbidity	695 and 1133 cases and controls	Highest exposure category: RR = 0.57 (0.36–0.89)	Ahlbom et al., 2004
Utility workers, employed 6 months between 1973–1982, followed – 1997	Cohort SMR and internal comparisons	Duration of work in jobs and locations with elevated EMF	CVD mortality	CVD deaths: 6802 Cohort: 79 972	Highest μ T-year category, AMI: RR = 1.03 (0.88–1.21) Chronic CHD: RR = 0.92 (0.73–1.16)	Sorahan & Nichols, 2004

8.3 Discussion

8.3.1 Heart rate variability hypothesis

Occupational exposure to electromagnetic fields has been suggested to increase the risk for cardiac arrhythmia-related conditions and acute myocardial infarction (Savitz et al., 1999). This hypothesized association between exposure to EMF and cardiovascular disorders was based on experimental data on HRV (Sastre, Cook & Graham, 1998). These experimental data were obtained in a double-blind laboratory investigation in which exposure to 20 μ T of intermittent 60 Hz magnetic fields was found to reduce the normal variation of the HRV (Sastre, Cook & Graham, 1998). However, these findings have not been reproduced, and subsequent studies with volunteers did not always produce consistent results regarding HRV and exposures to magnetic fields. After conducting a multi-study analysis, it was concluded that differences in study design factors related to physiologic arousal might explain the apparent inconsistency (Graham et al., 2000d).

In addition, several prospective cohort studies have indicated that reductions in some components of the variation in heart rate increase: (1) the risk for heart disease (Dekker et al., 1997; Liao et al., 1997; Martin et al., 1987; Tsuji et al., 1996), (2) overall mortality rate in survivors of myocardial infarction (Kleiger et al., 1987; Lombardi et al., 1987; Vaishnav et al., 1994), and (3) the risk for sudden cardiovascular death (Malik, Farrell & Camm, 1990). Changed HRV reflects changed cardiac autonomic control (Akselrod et al., 1981; Willich et al., 1993), suggesting this is a possible mechanism of action of EMF exposure on the heart

Thus, while reduced HRV seems to be predictive for the development and survival from heart disease, it is difficult to explain how the mechanism underlying the transient changes in heart rate variability seen in healthy young men after EMF exposure in controlled settings (Graham et al., 2000a; Sastre, Cook & Graham, 1998; Tabor, Michalski & Rokita, 2004) can also explain deaths from arrhythmia and infarction many years after long-term occupational exposure to ELF fields. Furthermore, the influence of EMF on HRV seems questionable.

8.3.2 Epidemiologic evidence

The biologically plausible model described above gave Savitz et al. (1999) the impetus to look at cardiovascular mortality in a cohort of utility workers. As postulated *a priori* Savitz observed an increased risk from AMI and arrhythmia related death, but not from chronic cardiovascular disease (Savitz et al., 1999). The only and limited support for the original observation comes from a study based on data from the Swedish twin registry (Håkansson et al., 2003), which observed a nonsignificantly increased risk for AMI. However, seven other studies failed to support to this hypothesis. The first three of these studies were done before the HRV hypothesis was introduced and were mainly descriptive and did not focus on cardiovascular disease. The other four studies were specifically designed to test this hypothesis from different point of views: two (Sahl et al., 2002; Sorahan & Nichols, 2004) were replications of the original study, and like Savitz et al. (1999), focused on cohorts of utility workers. One study focused specifically on arrhythmia (Johansen et al., 2002); one study investigated cardiovascular morbidity and was the first study to have detailed information on confounding factors and thus an ability to control for them (Ahlbom et al., 2004).

Thus only mortality studies of the association between occupational exposure to EMF and cardiovascular diseases have reported an association (Håkansson et al., 2003; Savitz et al., 1999). Studies of cardiovascular diseases which rely on mortality records as the measure of outcome are limited because the disease under study may not be mentioned on the death certificate, and if so, the accuracy of the diagnosis may not be correct. It is well known that death certificates do not provide the same quality of outcome measure as compared to incidence records which mainly can be obtained in disease registries or prospectively designed cohort or case-control studies. There are limitations to speculating about causal mechanisms of types of CVD as coded on death certificates of uncertain validity and reliability

(Finkelstein, 1999). A recent UK study identified inaccuracy in identifying underlying cause of death on the death certificates and difficulties in differentiating between acute and chronic cardiac causes (Mant et al., 2006). Thus, on balance, the evidence supporting an etiologic relation between occupational EMF exposures has been overturned by more focused and rigorous studies.

8.4 Conclusions

Experimental studies of both short- and long-term exposure indicate that, while electric shock is an obvious health hazard, other hazardous cardiovascular effects associated with ELF fields are unlikely to occur at exposure levels commonly encountered environmentally or occupationally. Although various cardiovascular changes have been reported in the literature, the majority of effects are small and the results have not been consistent within and between studies. With one exception, none of the studies of cardiovascular disease morbidity and mortality has shown an association with exposure. Whether a specific association exists between exposure and altered autonomic control of the heart remains speculative. Overall, the evidence does not support an association between ELF exposure and cardiovascular disease.