SUDDEN DEATH AND ISCHEMIC HEART DISEASE*

Correlation with Hardness of Local Water Supply

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Abstract Investigations conducted in several parts of the world have shown that residents of soft-water areas have a higher death rate from ischemic heart disease than residents of hard-water areas. The same pattern appears to exist in Ontario, but when deaths are divided into sudden and nonsudden (on the basis of whether or not the death certificate was signed by a coroner), the higher death rate in the soft-water areas is found to be due entirely to an excess of sudden deaths. This finding suggests that the correlation between cardiac mortality and water hardness may be the result of an increased susceptibility to lethal arrhythmias among residents of soft-water areas.

REGIONAL death rates from ischemic heart disease (IHD) in several countries have been found to be inversely related to the hardness of the local water supply.[1-3] British studies have demonstrated that this relation is not secondary to other variables such as climate and socioeconomic factors, and that calcium is the water component most closely correlated with the variation in death rate.[4,5]

These findings, together with the fact that neuromuscular excitability may be affected by changes in the serum level of ionized calcium (for example, tetany in hypoparathyroidism) have prompted us to explore the possibility that the higher death rate in soft-water areas might be the result of an increased susceptibility to fatal cardiac arrhythmias. Continuous monitoring of patients in coronary-care units has revealed that such arrhythmias are particularly likely to occur in the first few hours after myocardial infarction and that death due to "mechanical" failure of the heart is usually a less rapid process.[6] It seems likely, therefore, that a large proportion of the deaths in which the patient succumbs before he can be admitted to hospital are also the result of a fatal arrhythmia. Such deaths are typically those that may be handled by a coroner, and any regional variation in the frequency of fatal cardiac arrhythmias should be reflected in a comparable variation in the proportion of deaths certified by coroners rather than by private physicians.

We have therefore examined the variation in death rate from ischemic heart disease in three regions of Ontario, according to the hardness of the local water supply and whether or not the death was certified by a coroner.

MATERIALS AND METHODS

A substantial amount of information from death certificates is routinely coded and transferred to punch cards at the office of the Registrar General of Ontario. After the completion of the annual vital-statistics report, the tabulating department of the Registrar General's office kindly re-sorted the 55,000 cards for 1967 according to the following: status of person signing certificate (coroner or noncoroner); cause of death by ICD (International Classification of Diseases [WHO], seventh revision) rubrics 420 (arteriosclerotic and degenerative heart disease) or 001-795, excluding 420 (all other "natural causes"); residence – by counties and major urban areas; sex; and age – by 10-year age groups between 35 and 74.

Figures on the "hardness" of municipal water supplies were obtained from the Ontario Water Resources Commission, and were used to estimate the mean water hardness in each of the 55 counties of the province. Hardness was expressed in terms of parts per million (ppm) of calcium carbonate. A weighted mean hardness was calculated for each county, on the basis of the population served by the municipal water supply within the county. Counties were then ranked according to water hardness and divided into three regions: less than 100 ppm; 100 to 200 ppm; and greater than 200 ppm. The weighted mean hardness in each region was 53, 136 and 347 ppm respectively.

Death rates for each region were calculated with the use of the population figures from the 1966 census. Rates were calculated separately for males and females by 10-year age groups, and then combined in the proportions present in the total Ontario population to give a standardized death rate for both sexes combined over the age range of 35 to 74. To allow for possible distortion in the regional comparisons due to urban-rural differences, rates were also calculated for each region after cities with a population greater than 100,000 had been excluded.

RESULTS

The standardized death rate from ischemic heart disease declined from 416 per 100,000 in the area with water hardness of less than 100 ppm to 390 per 100,000 in the intermediate area, to 365 per 100,000 in the area with over 200 ppm. Excluding cities with over 100,000 population from the calculations, the comparable rates were 413, 388 and 353 per 100,000 (Fig. 1).

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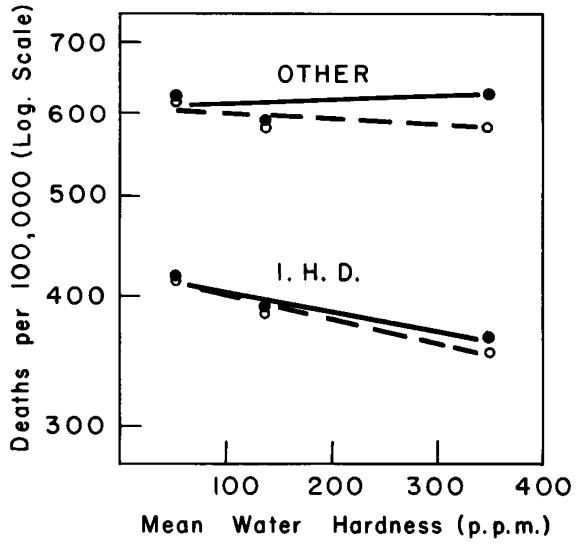

FIGURE 1. *Standardized Death Rates from Ischemic Heart Disease (IHD) and Other Natural Causes for Males and Females 35 to 74 Years of Age in Three Regions of Ontario.*

Regression lines have been calculated by the method of least squares, and a semilogarithmic (proportional) scale has been used to facilitate the comparison of slopes. Rates based on total population of regions are indicated by solid lines, and those based on population of regions excluding cities with over 100,000 population by broken lines.

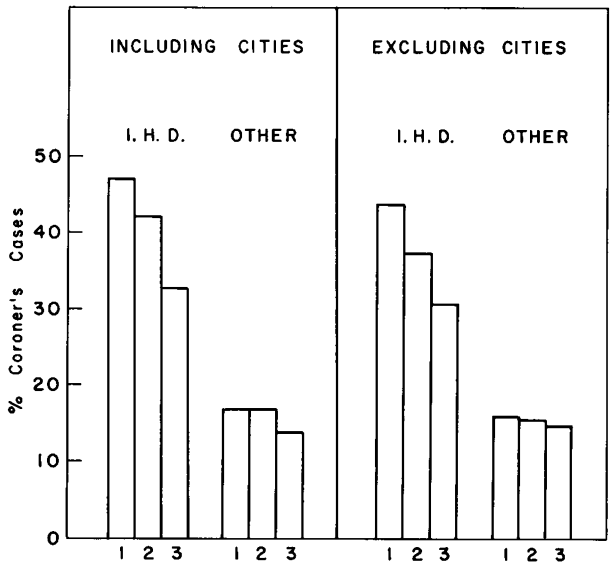

FIGURE 2. *Percentage of Death Certificates Signed by a Coroner for Deaths Ascribed to Ischemic Heart Disease and Other Natural Causes in Three Regions of Ontario.*

Figures are based on an age and sex standardized population of males and females between the ages of 35 and 74. The first column indicates water hardness of less than 100 ppm, the second column water hardness 100 to 200 ppm, and the third column water hardness over 200 ppm.

For "other" diseases there was no obvious trend when the death rates were based on total population (622, 589 and 629 per 100,000), but a slight gradient existed when cities were excluded (613, 582 and 582 per 100,000). Previous studies[2] have found that death rates from some diseases other than ischemic heart disease (such as cerebrovascular disease) may be related to water hardness, and the existence of a slight gradient among the "other" group is therefore not entirely unexpected. No attempt has been made in the present study to determine the other diseases responsible for this gradient because of the relatively small population and short period of observation on which our data are based.

In all three regions the proportion of death certificates signed by a coroner was higher for deaths ascribed to ischemic heart disease than for deaths ascribed to other diseases. Forty-seven per cent of certificates of death from ischemic heart disease were signed by the coroner in the region with the softest water, 42 per cent in the intermediate area and 33 per cent in the hard-water area. Comparable figures for other fatal diseases were 17 per cent, 17 per cent and 14 per cent. Excluding cities, the proportions were 44 per cent, 37 per cent and 31 per cent for deaths from ischemic heart disease, and 16 per cent, 15 per cent and 15 per cent for other diseases (Fig. 2).

If the rate at which other diseases were reported to the coroner can be taken as an index of reporting policy in the three regions, there seems to have been little variation in reporting policy across the province. The marked gradient that existed in the reporting of deaths from ischemic heart disease may therefore represent a true difference between the regions in the frequency with which death from that cause took the form of sudden rather than nonsudden death.

In Figure 3 rates of death from ischemic heart disease have been plotted for the three regions as deaths reported and those not reported to the coroner. There is a marked gradient for coroner's deaths (195, 164 and 120 per 100,000), but little or no gradient for noncoroner's deaths (220, 226 and 246 per 100,000). Excluding cities from the calculations does not materially affect the pattern. The correlation between total deaths from ischemic heart disease and water hardness shown previously (Fig. 1) thus appears to be the result of a selective increase in sudden deaths, and the rate of nonsudden deaths from ischemic heart disease was apparently little affected by the hardness of the local water supply. (The slight reverse slope of the noncoroner death rates may be a statistical artifact, or may be due to the removal – by sudden death – of some persons who would otherwise have died later and whose deaths would then have increased the rate of noncoroner deaths in the soft-water areas).

DISCUSSION

Although it is now well established that mortality from ischemic heart disease may be related to water hardness, the mechanism remains obscure. By analogy with the "furring" of water pipes, one might

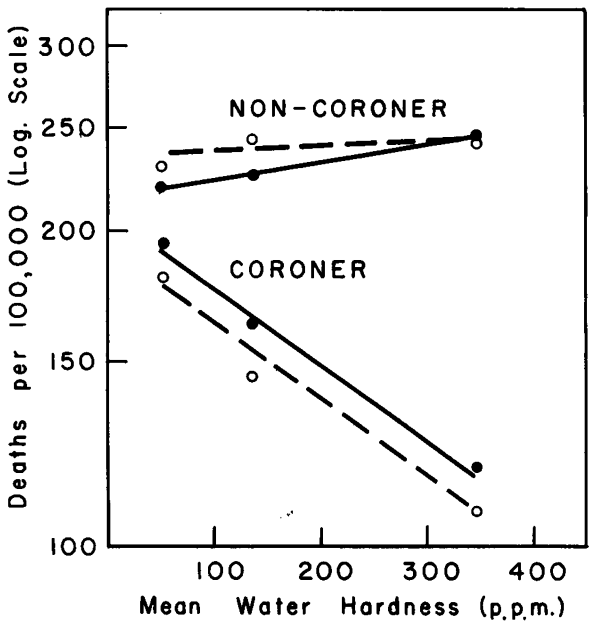

FIGURE 3. *Standardized Death Rates from Ischemic Heart Disease for Males and Females 35 to 74 Years of Age in Three Regions of Ontario, According to Whether or Not the Death Certificate Was Signed by a Coroner.*

Regression lines have been calculated by the method of least squares, and a semilogarithmic (proportional) scale has been used to facilitate the comparison of slopes. Rates based on total population of regions are indicated by solid lines, and those based on population of regions excluding cities with over 100,000 population by broken lines.

anticipate that arterial stenosis would be more prevalent in hard-water areas. However, not only would this presumably result in a trend in the opposite direction to that actually observed (rates of death from ischemic heart disease are lower in hard-water areas) but an autopsy study by Crawford and Crawford[7] showed no difference in the prevalence of coronary atherosclerosis between residents of hard-water and soft-water areas. These authors concluded that there might be a myocardial factor involved, resulting in an increased tendency to infarction in the soft-water area.

Although the present investigation provides no information on the relative frequency of myocardial infarction in the three regions studied, it is unlikely that the differences observed could be due solely to variation in the infarction rate. An increased tendency to myocardial infarction in soft-water areas would presumably lead to an increase in all manifestations of the disease, both nonfatal and fatal, sudden and nonsudden, and the ratio of sudden to nonsudden deaths should show little, if any, variation. Thus, our finding that the rate of deaths from ischemic heart disease reported to coroners in the region with the softest water supply is almost double that in the hard-water region, whereas noncoroner death rates are virtually the same, suggests that the main effect of water hardness may be on the mechanism causing death rather than on the underlying process of myocardial infarction.

Whether, as suggested above, this is the result of a simple relation between water hardness, serum levels of calcium or other ions and myocardial excitability is purely speculative. It is also possible that our present findings are entirely fortuitous, and that the differences that we found between the three regions are unrelated to "water hardness" but are the result of other factors such as climate, population density and socioeconomic levels. We are now accumulating mortality data for several past years to be able to determine the effect of some of these other variables. Meanwhile, we are presenting this preliminary report to alert other investigators to the possibility that the mode of dying is an important aspect of regional differences in mortality from ischemic heart disease and contributes to an understanding of the mechanisms involved.

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