Ischaemic heart disease and the water factor
A variable relationship

DEREK MEYERS
Brisbane, Australia

Mortality from ischaemic heart disease is found to be higher in Brisbane where drinking water is harder, than in Melbourne. Moreover, mortality from all causes is higher in Brisbane than in Melbourne.

A possible relationship between mortality from cardiovascular disease and the mineral content of drinking water has been studied for the last 15 years. Most, but not all, reports have recorded a statistical association between cardiovascular mortality and the mineral content. The literature has been reviewed by Crawford, Gardner, and Morris (1971a).

The cities of Brisbane and Melbourne offer a favourable opportunity for studying the question. Brisbane, capital of Queensland, lies on latitude 27° south, has a sub-tropical climate, an annual rainfall of 114·3 cm, and a hard water supply. The population is 850 000. Melbourne, the capital of Victoria, is a conurbation of 2½ million people, on latitude 38° south, has a mediterranean climate, an average rainfall of 66·1 cm, and a very soft water supply. (In contrast to the statement of Crawford, Gardner, and Morris (1968), higher rainfall is here associated with harder water.)

The population of the two cities is almost entirely white, predominantly of British origin, with a post-war addition of immigrants mainly from Europe. According to the 1966 census, 85% of the population of Brisbane was born in Australia, 8·6% in the United Kingdom and Ireland, and 0·9% in Italy. Corresponding figures for Victoria (figures for Melbourne were not available) were 79%, 7·4%, and 3·4%. As the Australian population is fairly mobile, and staff from various organizations and the public service are transferred from state to state, it is difficult to say how many people from the two cities have lived the whole, or even most, of their lives in a particular centre.

Medical education in the two cities is similar to the British system. Regular meetings of many learned societies, and appointments of graduates of one university to teaching posts in another, ensure that medical standards are fairly uniform throughout the Commonwealth, so that there is no likelihood of major differences in diagnostic fashion between the two centres.

MORTALITY FROM ISCHAEMIC HEART DISEASE
In an endeavour to compare mortality rates in the two cities, it was decided to restrict the study to four of the International Classification of Diseases categories of ischaemic heart disease:

410 acute myocardial infarction;
411 other acute and subacute forms of ischaemic heart disease;
412 chronic ischaemic heart disease;
413 angina pectoris.

The Commonwealth Bureau of Statistics provided figures for the population by 10-year age groups from 25 onwards, for both sexes, for the years 1968, 1969, and 1970, for Brisbane and Melbourne.
Ischaemic heart disease and the water factor

TABLE I
WOMEN
ICD 410-413
ANNUAL DEATH RATE PER 100 000
1968-70

<table>
<thead>
<tr>
<th>Place</th>
<th>Age Group</th>
<th>35-44</th>
<th>45-54</th>
<th>55-64</th>
<th>65+</th>
<th>All Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melbourne</td>
<td>35-44</td>
<td>11.6</td>
<td>61.9</td>
<td>279.9</td>
<td>1777.0</td>
<td>223.3</td>
</tr>
<tr>
<td>Brisbane</td>
<td>14.2</td>
<td>80.7</td>
<td>339.9</td>
<td>1890.3</td>
<td>253.7</td>
<td></td>
</tr>
<tr>
<td>( \chi^2 )</td>
<td>0.667</td>
<td>2.112</td>
<td>10.165**</td>
<td>7.333**</td>
<td>40.264***</td>
<td></td>
</tr>
</tbody>
</table>

* P<0.05 ** P<0.01 *** P<0.001

TABLE II
WOMEN
ICD 410
ANNUAL DEATH RATE PER 100 000
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<th>Place</th>
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<tbody>
<tr>
<td>Melbourne</td>
<td>8.0</td>
<td>44.5</td>
<td>185.8</td>
<td>1189.0</td>
<td>148.0</td>
<td></td>
</tr>
<tr>
<td>Brisbane</td>
<td>10.6</td>
<td>60.5</td>
<td>283.4</td>
<td>1273.0</td>
<td>158.8</td>
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</tr>
<tr>
<td>( \chi^2 )</td>
<td>0.632</td>
<td>3.868*</td>
<td>25.474***</td>
<td>4.018*</td>
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<td></td>
</tr>
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TABLE III
MEN
ICD 410-413
ANNUAL DEATH RATE PER 100 000
1968-70

<table>
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<tr>
<th>Place</th>
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</tbody>
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TABLE V
MORTALITY FROM ALL CAUSES (RATE PER 100 000)

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>1968</th>
<th>1969</th>
<th>1970</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>Men</td>
<td>Men</td>
<td>Men</td>
</tr>
<tr>
<td>Women</td>
<td>Women</td>
<td>Women</td>
<td>Women</td>
</tr>
</tbody>
</table>

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<th>Age Groups</th>
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<tbody>
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<td>Women</td>
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<td>Women</td>
<td>Women</td>
</tr>
</tbody>
</table>

Numbers of deaths by sex and age group in each of the above disease categories were also given. As the number of deaths in age group 25 to 34 was small, this group was not studied further. Death rates per 100 000 of population at risk per annum were calculated for the other four age groups, and for all ages together, and are set out in Tables I to IV.

For the year 1968, death rates were available only for all categories combined. Therefore, in Tables II and IV, illustrating category 410, only the years 1969 and 1970 appear.

As a further comparison between the two cities, total mortality was calculated for the same years and age groups, and is set out in Table V.

The tables show:

For all age groups and both sexes, the death rate for all categories of ischaemic heart disease, and for acute myocardial infarction alone, was higher in Brisbane than in Melbourne, the difference varying from significant to highly significant.

For males, the difference was highly significant for ages 45 to 64, and for all ages together.
For females, there was a highly significant difference in rates for acute myocardial infarction in the age group 55 to 64, and for all categories when all ages were considered together.

For both sexes, the death rate increased with age.

For females in the age groups up to 64 the death rate was similar to that of males ten years younger.

Total mortality for all years, both sexes and all age groups, with only one exception, was higher in Brisbane.

**Quality of Drinking Water**

Figures for water quality, obtained from the Department of Water Supply and Sewerage of the Brisbane City Council, show that Brisbane water contains more soluble solids, calcium, magnesium, sulphate, chloride, and fluoride than Melbourne’s, has a higher alkalinity expressed as calcium carbonate, a greater degree of total hardness expressed in the same way, and a higher pH (Table VI).

These figures show that the death rate from ischaemic heart disease is higher in Brisbane, where drinking water is hard and alkaline, and where the rainfall is fairly high, than in Melbourne, where the drinking water is soft, of lower alkalinity, and where the rainfall is lower.

**Discussion**

There are many difficulties in a population study of this type, such as the following:

**Diagnosis**

The figures given have been collected from death certificates. Those completed after necropsy are accurate, but others may be misleading. Thus Burry (1965) found that final clinical diagnoses corresponded with necropsy diagnoses in only about 70% of cases. Moreover, it may be difficult even at necropsy to distinguish between a major and a contributing cause of death. Possibly, with large figures such as these and thousands of deaths in the older age groups, differences between the two cities may even out.

Only in a small closed community could a comprehensive study of disease mortality be undertaken, and then only if the necropsy rate was a high percentage of the total deaths. In this circumstance, however, the actual number of deaths per age group for any one cause would be likely to be very small, so that meaningful figures may not be obtained.

**Domicile**

Even within the continent of Australia, many families change their addresses fairly frequently. While the usual death certificate records the place of death, there is no means of knowing how long a person has lived in any given place. With the large numbers involved in this series, one can again only hope that frequency of change of domicile is similar in the two cities.

**Effect of Climate**

Factors such as temperature, humidity, rainfall and its distribution throughout the year, type of winds, and sudden changes of weather may have an effect on disease incidence and mortality at least as important as any other environmental variable. A

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**Table VI**

**ANALYSIS OF WATER SUPPLIES FOR BRISBANE AND MELBOURNE (parts per million)**

<table>
<thead>
<tr>
<th>Water</th>
<th>Brisbane</th>
<th></th>
<th>Melbourne</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Max.</td>
<td>Av.</td>
<td>Min.</td>
<td>Max.</td>
</tr>
<tr>
<td>Total soluble solids</td>
<td>564</td>
<td>320</td>
<td>142</td>
<td>75</td>
</tr>
<tr>
<td>Calcium</td>
<td>45</td>
<td>29</td>
<td>10</td>
<td>2.2</td>
</tr>
<tr>
<td>Magnesium</td>
<td>35</td>
<td>15</td>
<td>5</td>
<td>1.9</td>
</tr>
<tr>
<td>SO4</td>
<td>66</td>
<td>30</td>
<td>9</td>
<td>2.5</td>
</tr>
<tr>
<td>Chloride</td>
<td>196</td>
<td>88</td>
<td>20</td>
<td>17</td>
</tr>
<tr>
<td>Fluoride</td>
<td>0.2</td>
<td>0.15</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Alkalinity (expressed as CaCO3)</td>
<td>152</td>
<td>80</td>
<td>24</td>
<td>12</td>
</tr>
<tr>
<td>Total hardness (expressed as CaCO3)</td>
<td>242</td>
<td>142</td>
<td>52</td>
<td>19</td>
</tr>
<tr>
<td>pH</td>
<td>7.7</td>
<td>7.1</td>
<td>6.4</td>
<td>7.0</td>
</tr>
</tbody>
</table>
latitude effect has thus been noted in Britain, the death rate being higher in the higher latitudes (Gardner, Crawford, and Morris, 1969). Here, however, it is higher in the lower latitudes.

**PLACE OF BIRTH**

Stenhouse and McCall (1970) have shown that mortality rates for cardiovascular disease were appreciably higher in persons born in Australia than in immigrants from England, Wales, and Italy (Scottish immigrants had high rates). The difference was most marked for Italians, but this group forms only a small percentage of the population, greater in Melbourne than in Brisbane. Even if one-tenth of the population of Melbourne had double that city’s average mortality, the mortality rates for the two cities would then be roughly only equal. The difference in recorded mortality cannot, therefore, be explained by a difference in proportion of migrants.

**SUDDEN DEATH**

Anderson, Le Riche, and Mackay (1969) have suggested that the excess mortality from cardiovascular disease in areas with soft drinking water is due to an increased incidence of sudden death. This concept is extremely difficult to define. Thus in Queensland if a person dies suddenly and has not been under medical supervision for three months, the coroner must be notified. On the other hand if a person under regular supervision and known to have coronary disease should die suddenly, in all probability his usual doctor will issue a death certificate which may not make it clear whether death was sudden or not. Again, a patient who has been admitted to hospital with acute myocardial infarction may survive episodes of rhythm disturbance in a coronary care unit, only to die some days later. Is this death to be called sudden? Furthermore, causes of sudden death after myocardial infarction are multiple: rhythm disturbance, further myocardial infarction, rupture of the heart, and pulmonary embolism may all abruptly terminate the illness, but may not necessarily have any common cause.

**TRACE ELEMENTS**

Neri, Hewitt, and Mandel (1972) suggested that the value of water hardness as a predictor of coronary artery disease mortality may be related to trace metal contents and they proposed to study lithium, chromium, zinc, copper, cadmium, molybdenum, vanadium, and magnesium. Voors (1971) found that lithium levels correlated negatively with atherosclerotic heart disease in whites, and molybdenum in non-whites. He proposed research into the desirability of mineral enrichment of the drinking water in some communities. Comstock (1971) did not find that water hardness was a real risk for cardiovascular disease, but thought the role of trace elements should be investigated. Other writers, possibly in lighter vein, have suggested that boiling water to make tea may remove a protective hardness factor, and that nitrates may have a beneficial vasodilator action. The possibilities for further studies seem endless.

**OTHER CARDIOVASCULAR DISEASE**

The main causes of death from cardiovascular disease in Australia, apart from ischaemic heart disease, are essential hypertension and cerebral vascular disease; congenital, rheumatic, and pulmonary heart disease play a relatively minor role. Hypertension caused by chronic renal disease is also common, but in this case, death may be certified as due to any one of a number of causes, such as, uraemia, cerebral haemorrhage, cerebral thrombosis, left ventricular failure, or congestive heart failure, depending on the extent to which the doctor who issues the certificate is familiar with the case.

Renal disease in Queensland has in the past been due to lead poisoning, and more recently to analgesic nephropathy, as well as post-streptococcal and other forms of nephritis, and has been shown by Burry (1966) to have a higher incidence in Queensland than in other Australian states. The reason for this may be climatic. Essential hypertension, by definition, is of an unknown cause, but there is no reason to suspect that it and atheroma have any common aetiological factors.

In view of the number of possible aetiological factors in the different conditions, consideration of death from all forms of cardiovascular disease together may hamper the search for environmental agents contributing to specific disorders.

**CONCLUSION**

In a condition such as a coronary artery disease which may have a multiplicity of causes, many unrelated factors probably play a part, and field studies must be affected by the many variable characteristics of the population; it is difficult to confirm that a single environmental factor plays a dominant role in the aetiology.

Table VI shows a very marked difference in the quality of water supply in Brisbane and Melbourne; the former city has water that contains more soluble solids, is more alkaline, and harder. Mortality figures reveal a higher incidence of fatal ischaemic heart disease in Brisbane than in Melbourne, for both sexes and all ages. The suggestion that the
death rate from cardiovascular disease (or at least that part of it due to ischaemic heart disease) is lower in areas where the drinking water is hard, is not correct for all places. However, if there is a protective factor in hard water (or an adverse factor in soft water) the effect can be outweighed by some other unidentified factor capable of affecting the whole population.

The suggestion of Crawford, Gardner, and Morris, (1971b) that water supply authorities should consider the practicability of artificially hardening naturally soft drinking water should not yet be taken up outside England and Wales. On the other hand a search should continue for other adverse or protective environmental factors.

Generous help from Mr V. McLean, of the Commonwealth Bureau of Census and Statistics, the late Mr D. Madden, of the Brisbane Water Supply and Sewerage Department, and the late Dr H. Silverstone, former Reader in Medical Statistics at the University of Queensland, is gratefully acknowledged.

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REFERENCES


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