MYOCARDIAL INFARCTION IN SOUTHERN RHODESIA

BY

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Myocardial infarction in Southern Rhodesia affects almost exclusively people of European descent, and to a much lesser extent the small Asian and Coloured (predominantly Eurafrican) communities. Thus out of a total of 608 carefully studied cases of cardiac disease admitted during three years to the Mpilo Hospital (for Africans) in Bulawayo, there was not a single unequivocal case of myocardial infarction (Baldachin, 1961). Working at Harare Hospital in Salisbury, Gelfand (1961) found no case of myocardial infarction in 70 consecutive African patients, aged 60 years and upwards, suffering from heart disease.

There are no country-wide statistics available for the incidence of the disease among Coloured and Asian subjects, but analysis of the reports of the City Medical Officer of Health for Bulawayo over the past ten years indicates an annual crude death rate from arteriosclerotic and degenerative heart disease among the two communities combined of 0·25 per 1000 out of a current population at risk of 3200. This represents a frequency of less than one-quarter that of the Europeans.

SUBJECTS AND METHODS

This study is based on 370 personal cases, all white, whose records are considered adequate enough for profitable evaluation. These patients were seen either in hospital or private consulting practice. All known to have had a previous myocardial infarction were excluded from the series, since this consisted of primary infarctions only. All patients had at least one routine 12-lead electrocardiogram taken, and where indicated additional leads were inscribed. For the last three years of the investigation serum glutamic oxalacetic transaminase (S.G.O.T.) estimations were available and freely used. They were found helpful in early diagnosis, especially in patients with dubious posterior infarction or with major arrhythmias, and in those where the electrocardiograms showed bundle-branch block or where a hypertensive pattern complicated an anterolateral infarct.

This is essentially a clinical study, as opportunities for autopsy do not readily occur in the Rhodesias, except in such cases as may have a medico-legal aspect.

Population at Risk. The estimated European (White) population of Southern Rhodesia on June 30, 1948 was 101,000 (male 54,000, female 47,000), and on June 30, 1960, 223,000 (male 115,000, female 108,000). A majority of the Europeans live in the two relatively big urban centres of Salisbury and Bulawayo. Patients in this series were drawn mainly from Bulawayo (European population 1950, 24,000 and 1960, 50,000) and surrounding districts.

Of the 370 patients in the study, a note was made of the birth place of 244. Of these, 49 were born in Southern Rhodesia, 67 in South Africa, 77 in the British Isles, 44 in Continental Europe, and 7 elsewhere. Table I shows the proportion of the total European population born in Southern Rhodesia and elsewhere at the last three censuses.

If it is assumed that those born in Southern Rhodesia are as liable to cardiac infarctions as those
TABLE I

<table>
<thead>
<tr>
<th>Place of birth</th>
<th>1946</th>
<th>1951</th>
<th>1956</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Southern Rhodesia</td>
<td>Elsewhere</td>
<td>Southern Rhodesia</td>
</tr>
<tr>
<td>Men</td>
<td>35.7</td>
<td>64.3</td>
<td>30.5</td>
</tr>
<tr>
<td>Women</td>
<td>40.1</td>
<td>59.9</td>
<td>32.5</td>
</tr>
<tr>
<td>Total</td>
<td>37.7</td>
<td>62.3</td>
<td>31.4</td>
</tr>
</tbody>
</table>

born elsewhere, then the 49 patients out of 244, which is 20 per cent, is lower than the figure of approximately 35 per cent that might be expected from Table I. However, the total numbers are small and weighted, in that the Rhodesian-born population is a young one, containing a high proportion of children and adolescents born to recent immigrants.

Of the 370 cases in the series, 278 were men (75%) and 92 were women (25%). This sex difference is roughly of the same order as that for a much larger series of 2724 cases of myocardial infarction (Mitchell and Parish, 1960) who were admitted to three hospitals in the English Midlands over a 20-year period: here 72 per cent were men and 28 per cent were women. Table II shows by decades the sex difference of incidence. It will be noticed that with advancing years the sex difference lessens, but at best, in the older age-groups, men were twice as commonly afflicted as women.

TABLE II

<table>
<thead>
<tr>
<th>Age of onset</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
<th>Acute mortality</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>20–29</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>30–39</td>
<td>16</td>
<td>1</td>
<td>17</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>40–49</td>
<td>55</td>
<td>9</td>
<td>64</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>50–59</td>
<td>73</td>
<td>23</td>
<td>96</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>60–69</td>
<td>81</td>
<td>34</td>
<td>115</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>70–79</td>
<td>38</td>
<td>19</td>
<td>57</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>80–89</td>
<td>12</td>
<td>6</td>
<td>18</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>90+</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>278</td>
<td>92</td>
<td>370</td>
<td>38</td>
<td>20</td>
</tr>
</tbody>
</table>

An effort was made in each case to place the position of the infarct accurately. This was possible in 329 patients (252 men, 77 women) and of the men 153 were placed as predominantly anterior, and 99 as predominantly posterior; of the women 51 were anterior and 26 posterior. In the total of the 329 cases of both sexes, anterior infarcts outnumbered posterior by 204 to 125.

There were 39 cases of bundle-branch block in this series, or 10.5 per cent. This compares with an incidence of 12 per cent in 1058 cases collected from published reports by Master, Dack, and Jaffe (1938), and 15 per cent in 375 cases of their own. The smaller percentage in the present series could be due to the increased discovery of minor degrees of infarction, made possible by the more thorough electrical exploration by multiple leads.
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In localizing the infarcts in cases associated with bundle-branch block, the criteria laid down by Somerville and Wood (1949) were followed. Electrocardiographic signs of infarction were found in 8 of 23 cases with left bundle-branch block, or 35 per cent, and in 12 of 16 cases with right bundle-branch block, or 75 per cent. There were 9 cases of heart block in the series, which were observed during the acute phase, i.e. within three months of the infarction. Of these, two had Grade I heart block, two cases had two-to-one heart block, and five cases had complete atrio-ventricular block, one of whom suffered a fatal Stokes-Adams attack. Of these 9 cases, 8 had anterior infarctions and 1 a posterior infarction.

Direct trauma to the precordium was believed to have been a precipitating factor in five cases. Ten women and 23 men were known to have sustained two or more infarctions during the period of observation, but the total number so affected may have been greater, as the follow-up was incomplete. Of the 10 women who had had multiple infarctions, 7 died of a second or third infarct, and of the 23 men with multiple infarctions, 13 were known to have died of a second or subsequent cardiac infarct.

At the end of the 10 years and 9 months covered by the survey, 155 of the 370 cases were known to be dead. The mortality may have been greater, as all patients could not be traced. Many of these deaths were due to unrelated diseases, especially in the older age-groups, where carcinoma took a heavy toll. Any patient dying within three months of his or her first cardiac infarction was considered to have died as a result of that attack. The number so dying was 38 men and 20 women, total 58 (Table II). The precise site of the infarction did not appear to influence the immediate mortality. The 58 acute deaths out of 370 constitute 15-7 per cent or 1 death in every 6-4 of initial attacks. There is no real yardstick against which to measure these figures as mortality estimates vary so much from series to series (Evans, 1954).

Five patients in the series of 370 (1-4%) developed a perforated inter-ventricular septum. Harrison et al. (1961) reviewing earlier publications found the reported incidence of septal perforation in myocardial infarction to vary from 0-9 to 1-6 per cent of all myocardial infarctions.

Cardiac aneurysm affecting the left ventricle was discovered in 16 cases. Two of these were diagnosed clinically, but the rest were found, often rather unexpectedly, at routine radioscopy. It was not searched for specifically, and the true incidence is probably much higher: it may be found at autopsy in as many as 22 per cent of fatal cases (Wartman and Hellerstein, 1948).

In as much as it may modify the natural history and mortality of the disease, treatment must be discussed briefly. From the beginning of the series, cases were treated on broadly similar lines. Anticoagulants were used in the great majority, and were omitted only in special circumstances. At the time of diagnosis, bishydroxy-coumarin or one of its congeners was given. For the last four years of the series, warfarin sodium was the drug of choice. Heparin was given for the first 24 to 48 hours, for all but the mildest attacks. Long-term anticoagulant therapy was used in some patients, without any great conviction as to its usefulness. Its main advantage was that it could be a means of bringing back the carefree patient for periodical checks on his weight and blood pressure.

In this series, early rising was favoured, and patients were allowed to sit out of bed once the initial shock had passed, and adequate anticoagulation was established. Pressor amines were found useful in raising the blood pressure of very shocked patients: noradrenaline was used for this purpose in five, but none of them survived. A digitalis preparation was given in any case showing evidence of congestive failure, and serious arrhythmias were controlled with quinidine or procaine amide.

DISCUSSION

Clinical details of 370 white patients suffering from myocardial infarction in Southern Rhodesia are analysed. Those born in Central and Southern Africa appear to be almost as susceptible as those born in Europe.
The total white population of Southern Rhodesia in 1950 was 129,000, with a crude death rate of 6.7 per thousand; 149 deaths were registered as due to arteriosclerotic and degenerative heart disease, a crude death rate of 1.15 per thousand. In 1959 the population was 215,000, with a crude death rate of 5.9 per thousand: 231 deaths were registered as due to arteriosclerotic and degenerative heart disease, a crude rate of 1.07 per 1000. Thus the death rate from this disease is stationary or declining slightly at a rate lower than, but of the same order as, in England and Wales (Registrar General’s Statistical Review of England and Wales for 1958, 1960). The general incidence and pattern of attack is the same as in northern countries.

The differing incidence of arteriosclerotic heart disease between the European and Bantu populations is a matter of endless interest to physicians practising in Central and Southern Africa. “It very rarely happens that an effect is brought about by a single cause—rather do we find that a certain combination of circumstances is necessary, and that the absence of even one of them is enough to prevent the occurrence of the event,” (Moroney, 1956). In their masterly analysis of the natural history of coronary disease in England and Wales, Ryle and Russell (1949) anticipated this principle, and stated that “the effect of multiple cumulative causes has never, perhaps, been sufficiently emphasized in considering the genesis whether of symptoms or of pathological changes.”

The only controlled autopsy study published on the differing incidence of coronary atheroma between Europeans and Africans in the Federation is that of Hannah (1958) in Northern Rhodesia. Among his 22 European subjects dying sudden deaths from any cause, all showed coronary atheroma, and of his 42 corresponding African subjects, 7 showed coronary atheroma which was severe in only one. Nussbaum of Bulawayo has kindly allowed me access to his unpublished data on comparative serum cholesterol studies between Europeans and Africans (Lieberman-Burchardt method). He examined the sera of 100 African and 89 European healthy subjects, aged from 25 years upwards: the mean European figure was 185 mg./100 ml., and the mean African figure was 126 mg./100 ml. Since geographical and climatic factors are the same for both races, they can be excluded from consideration in explaining the different incidence of the disease. Factors that must be examined are genetics, social class, stress, and diet, the last three of which inevitably overlap.

The European in Central Africa cannot be placed conveniently in one of the five social classes of the Registrar General. Broadly speaking, and excluding the farmer, there are only two classes, the white-collar worker and the artisan, and these two classes often merge into each other. The European has a high standard of living which, as yet, only a minority of Africans has attained.

Brock and Gordon (1959) pointed out that in so far as stress is concerned, the difference between the Bantu and the White races is qualitative rather than quantitative. Southern Rhodesia has the highest percentage of literate autochthonous inhabitants in Africa. The transition from a tribal to an industrial urban life is proceeding apace, and with the emergence of business and professional men, trade union leaders, and politicians, this qualitative difference of stress between the urbanized African and the European is disappearing rapidly.

A genetic cause for the differing incidence must be seriously considered, but can not be assessed until the broad mass of an African population achieves a standard of living comparable with that of Western Europe or America. As yet no African state remotely approaches that standard.

Apart from the, at present, unassessable genetic factor, the main environmental difference between the African and the European in Central Africa lies in their respective diets. The Europeans eat a completely “Western” one, with heavy consumption of meat, eggs, and dairy produce. A small percentage of Africans eat a western-style diet, but the average urban African family lives at subsistence level on ground maize (mealie-meal), white bread, dried legumes, and a little lean meat when they can afford it. The adults supplement this with generous amounts of “Kaffir beer,” an inexpensive beverage with a low alcohol and high vitamin content. As an index of the different diets of the two races in Bulawayo, the Dairy Marketing Board which has a virtual monopoly of milk supply, sells an average of 17.5 oz. (495 g.) of fresh milk to each of the 50,000 Europeans in the city, and an average of 0.7 oz. (19 g.) daily to each of the 125,000 Africans: the consumption of butter, cheese, cream, and eggs is probably on an increased differential.
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CONCLUSION

The European in coming to an entirely new geographical and climatic environment in Central Africa has been unable to leave his genetic inheritance behind him, and he has deliberately brought with him his “western way of life” with its concomitant stresses and its eating habits, in particular its avid consumption of milk and its products, beyond infancy, through adult life, and into the decrescent years. He suffers a high incidence of arteriosclerotic heart disease, with myocardial infarction, which has a similar clinical pattern and age and sex incidence to that in northern countries. It is considered that the high incidence in Europeans and the very low incidence in Africans can only be ascribed to either genetic or dietary differences, or possibly a combination of both. The respective importance of these two factors will be assessable only when the broad mass of the African population reaches an economic standard comparable with that of the European.

SUMMARY

Among Europeans in Southern Rhodesia 370 cases of primary myocardial infarction are analysed and discussed. The extremely low incidence of the disease among Africans is described. The causes for this differing incidence are considered.

My thanks are due to the Director of the Central Statistical Office, Salisbury, Southern Rhodesia, for the information contained in Table I, and to Dr. Leon Nussbaum for the use of his unpublished data on serum cholesterol levels among Africans and Europeans in Bulawayo.

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