In conclusion, a morphometric method for the identification and possible classification of myocardial abnormalities in suspected diseased hearts is described. Myofibre abnormalities are detected by comparing suspect data with a GS compiled from data obtained in the morphometric analysis of myofibres in the LVs of 24 normal hearts. It is hoped that the publication of the methodology for data collection and the formulas used to construct this standard will offer other investigators an objective means for establishing LV myofibre normality and detecting myofibre disease in hearts in which disease is suspected.

We would like to express our gratitude to Mrs S. Bux for her patience in making the morphometric measurements, Mr S. M. Ayer of the Department of Forensic Medicine for his assistance in the collection of the normal tissue and Drs I. D. Whitton and V. Chrystal for the selection and supply of the pathological material. This work was supported by a grant from the South African Medical Research Council.

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Hypertension, alcohol and cations in urban black and coloured South Africans

R. M. TOUYZ, S. RATHAKRISHNAN, S. G. REINACH, F. J. MILNE

Summary

Epidemiological evidence shows a positive relationship between alcohol consumption and blood pressure. High alcohol intake and hypertension are common in urban South African men. Relationships between mean arterial pressure (MAP), serum γ-glutamyltransferase (GGT) as an index of alcohol intake, age, mass and levels of cations in the serum and erythrocytes were investigated in normotensive and hypertensive black and coloured men. Serum levels of potassium, magnesium and calcium and the red blood cell magnesium level were found to be significantly decreased in the black hypertensives. Serum GGT was equally raised in normotensive and hypertensive blacks and was positively correlated with systolic blood pressure in the hypertensive subjects. The coloured hypertensives were heavier, older and had significantly higher serum GGT levels than their normotensive counterparts. Serum GGT was positively correlated with MAP in the coloured subjects. No consistent relationships were found between GGT and blood cations. These data suggest that in younger black subjects alcohol is associated with systolic blood pressure only once hypertension has developed. Other factors, such as cations, may be more important than alcohol in the pathogenesis of hypertension in this group. Alcohol consumption is an important risk factor in coloured hypertensives.

Department of Medicine, University of the Witwatersrand, Johannesburg
R. M. TOUYZ, M.B. B.CH., M.SC. (MED.)
S. RATHAKRISHNAN, M.B. B.CH.
F. J. MILNE, M.D. F.C.P. (S.A.)
Institute for Biostatistics of the South African Medical Research Council, Johannesburg
S. G. REINACH, M.SC., D.SC. (AGRIC)

Reprint requests to: Dr R. M. Touyz, Dept of Medicine, University of the Witwatersrand Medical School, York Road, Parktown, 2193 RSA.
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and these changes are associated with elevated blood pressure.\textsuperscript{11} This study investigates the relationships between blood pressure, serum $\gamma$-glutamyltransferase and certain blood cations in normotensive and hypertensive urban men from two racial groups.

**Subjects and methods**

Urban black labourers (296 subjects) and coloured (mixed race) men (73 subjects) were studied. The labourers were chosen from black men reporting to the municipal medical centre at the labour bureau in the city of Johannesburg. The coloured group was chosen from subjects presenting to hospital outpatient clinics in Johannesburg and included those with minor medical problems such as colds, headaches or sore throats. Also included in this group were healthy industrial workers and school teachers. All the subjects gave signed informed consent. Only males aged between 20 and 60 years, who had lived in Johannesburg for at least 5 years and had no history of diuretic or scheduled drug use and no evidence of renal impairment (indicated by an abnormal L-Combur-5 test result and a raised serum creatinine level) were selected. All the subjects were studied under the same standardised conditions. Approval and permission for this study was obtained from the Human Ethics Committee of the University of the Witwatersrand.

Data on age, length of residence in the city and daily alcohol consumption and a medical history were obtained by questionnaire. Adiposity was assessed using the Quetelet index, i.e. weight (kg)/height (m)$^2$. The same techniques for measuring blood pressure and pulse rate were used for all subjects. Readings were taken in the right arm in the seated position after 15 minutes of rest. Blood pressure was recorded three times, one reading every 5 minutes on each individual. The mean of the three readings was taken for analysis. Systolic and diastolic (phase V of the Korotkoff sounds) pressures were measured using a standard mercury sphygmomanometer with regular cuff size. Hypertension was defined as follows: diastolic blood pressure (DBP) $\geq 95$ mmHg, or systolic blood pressure (SBP) $\geq 160$ mmHg, or both; or mean arterial pressure (MAP) ($\text{MAP} = \text{SBP} + 1/3 (\text{SBP} - \text{DBP})$) $\geq 117$ mmHg.

After a further 15 minutes' rest, 15 - 20 ml venous blood was drawn from an antecubital vein (without cuff compression) using the Vacutainer technique. Serum $\gamma$-glutamyltransferase (GGT), albumin, creatinine, sodium (Na$^+$), potassium (K$^+$), calcium (Ca$^{2+}$) and magnesium (Mg$^{2+}$) and red blood cell (RBC) Na$^+$, K$^+$ and Mg$^{2+}$ were measured in all the subjects. The samples were analysed blind, i.e. the technicians performing the biochemical tests were unaware of the blood pressure readings. Serum GGT was determined by an automated enzymatic method, and albumin and creatinine by colorimetric methods. Serum Na$^+$ and K$^+$ were measured by standard flame photometry\textsuperscript{12} and Mg$^{2+}$ and Ca$^{2+}$ by atomic absorption spectroscopy.\textsuperscript{13} Ca$^{2+}$ values were corrected according to the following formula: total serum Ca concentration + (40 - (albumin x 0,02)). RBC measurements were based on the method of Fortes Mayer and Starkey.\textsuperscript{12} RBCs were prepared according to previously described techniques.\textsuperscript{14}

Accuracy of techniques was established by performance of duplicate cation estimations by two different laboratories. The Wilcoxon test showed no significant difference in the results obtained between the two laboratories ($P > 0.05$).

Significance was assessed using Student's $t$-test for unpaired observations, and a correlation coefficient and regression analysis was done to determine associations between the variables.

**Results**

Of the 296 black subjects, 214 were found to be normotensive and 82 hypertensive, while of the 73 coloureds 63 were normotensive and 10 hypertensive. According to World Health Organization criteria the mean blood pressure (for all subjects) in both groups was rated 'moderate' (Table I). The black normotensive and hypertensive subjects were of similar ages, whereas the coloured hypertensives were significantly older than their normotensive counterparts. There was no significant difference in Quetelet index between normotensive and hypertensive subjects in either group (Table I).

In the black group 64% of normotensive and 36% of hypertensive subjects admitted to consuming alcohol on a daily basis. Of the coloureds, 11% of normotensives and 65% of hypertensive subjects consumed alcohol regularly. Because of discrepancies between the history of alcohol intake and the value of serum GGT value (i.e. denial of alcohol consumption coupled with a high serum GGT value), serum GGT was used as the determining factor for alcohol consumption. In the black group, although there was no significant difference in GGT values between the normotensive and hypertensive subjects, over a third of the total population sample analysed had raised levels, and in both groups the mean value was higher than normal. Serum GGT values were significantly higher than normal in the hypertensive coloured subjects (Fig. I).

Extracellular and RBC intracellular concentrations of the cations in the different groups are set out in Table II. Correlations between the variables and GGT were determined within each group and in the groups combined (Table III). In the groups combined, there was a significant positive relationship between GGT and age and also between GGT and MAP (Figs 2 and 3). In the black subjects age was positively related to GGT in both the normotensive and the hypertensive subjects. Serum Na$^+$ was negatively and serum K$^+$ positively correlated with GGT in the black hypertensives, in whom GGT was also positively correlated with systolic blood pressure ($r = 0.485; P < 0.05$). It must be kept in mind, however, that in all the above inter-relationships less than 20% of the variation in any one variable can be explained by any other variable. Consequently no one variable can be used as a reliable predictor for any of the others.

**TABLE I. DEMOGRAPHIC DATA AND SERUM GGT LEVELS (MEAN ± SD)**

<table>
<thead>
<tr>
<th></th>
<th>Blacks</th>
<th></th>
<th>Coloureds</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NT</td>
<td>HT</td>
<td>NT</td>
<td>HT</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>92.2 ± 8.4</td>
<td>119.5 ± 11.3</td>
<td>96.1 ± 12.4</td>
<td>125.8 ± 6.5</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>33.4 ± 6.3</td>
<td>35.1 ± 7.5</td>
<td>37.4 ± 10.5</td>
<td>48.1 ± 11.6</td>
</tr>
<tr>
<td>QI (kg/m$^2$)</td>
<td>22.0 ± 3.0</td>
<td>24.0 ± 3.0</td>
<td>23.0 ± 5.0</td>
<td>25.0 ± 7.0</td>
</tr>
<tr>
<td>GGT (U/l)*</td>
<td>66.8 ± 74.6</td>
<td>71.8 ± 62.4</td>
<td>44.8 ± 51.5</td>
<td>117.6 ± 146.4</td>
</tr>
<tr>
<td></td>
<td>$&lt; 0.001$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.001$</td>
<td>$P &lt; 0.001$</td>
</tr>
</tbody>
</table>

*Normal < 50 U/l.

NT = normotensive; HT = hypertensive; QI = Quetelet index.
subjects studied were generally healthy. It should, however, be borne in mind that other subclinical factors, e.g. hepatitis and cirrhosis, may have contributed to the elevated serum GGT levels in some subjects. Many studies demonstrate a direct correlation between alcohol intake and blood pressure, independent of age and weight. 16,17 The exact effects of these factors on hypertension genesis is unclear, and the precise mechanisms by which alcohol causes hypertension remain obscure. There have been few studies of associations between blood pressure, alcohol and cations in Johannesburg residents. 14 From the histories given by the subjects and the raised GGT levels, it is evident that a high percentage of the black population in the present study consumed alcohol. Discrepancies between enzyme levels and a history of alcohol consumption may be due to the fact that it is very difficult to obtain an

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**Discussion**

In this study a random sample of subjects was investigated, and the results should not be regarded as prevalence or incidence rates. Although the liver enzyme GGT is not an absolute indicator of alcohol consumption, it has been widely accepted as a good marker for high alcohol intake. 8,9 The

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**TABLE II. BIOCHEMICAL DATA (MEAN ± SD)**

<table>
<thead>
<tr>
<th>Serum</th>
<th>Blacks</th>
<th>Significance</th>
<th>Coloureds</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NT</td>
<td>HT</td>
<td>NT</td>
<td>HT</td>
</tr>
<tr>
<td>Na⁺ (mmol/l)</td>
<td>137.9 ± 2.3</td>
<td>139.9 ± 2.4</td>
<td>P = 0.465</td>
<td>140.4 ± 3.2</td>
</tr>
<tr>
<td>K⁺ (mmol/l)</td>
<td>3.9 ± 0.34</td>
<td>3.7 ± 0.38</td>
<td>P = 0.010</td>
<td>3.8 ± 0.45</td>
</tr>
<tr>
<td>Ca⁺⁺ (mmol/l)</td>
<td>2.42 ± 0.2</td>
<td>2.23 ± 0.2</td>
<td>P &lt; 0.001</td>
<td>2.36 ± 0.2</td>
</tr>
<tr>
<td>Mg⁺⁺ (mmol/l)</td>
<td>0.94 ± 0.07</td>
<td>0.81 ± 0.08</td>
<td>P &lt; 0.001</td>
<td>0.85 ± 0.08</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>40.9 ± 2.7</td>
<td>41.0 ± 3.0</td>
<td>P = 0.969</td>
<td>42.6 ± 9.3</td>
</tr>
<tr>
<td>RBC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na⁺ (mmol/l)</td>
<td>7.1 ± 2.0</td>
<td>8.5 ± 2.7</td>
<td>P &lt; 0.001</td>
<td>8.2 ± 2.6</td>
</tr>
<tr>
<td>K⁺ (mmol/l)</td>
<td>95.4 ± 10.2</td>
<td>93.6 ± 9.7</td>
<td>P = 0.027</td>
<td>99.9 ± 11.5</td>
</tr>
<tr>
<td>Mg⁺⁺ (mmol/l)</td>
<td>2.2 ± 0.5</td>
<td>1.9 ± 0.4</td>
<td>P &lt; 0.001</td>
<td>1.9 ± 0.4</td>
</tr>
</tbody>
</table>

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Fig. 1. Serum GGT levels in the black and coloured groups.

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Fig. 2. Correlation curve showing a relationship between serum GGT levels and MAP in the black and coloured groups combined.
accurate alcohol history. This study reflected no direct relationship between alcohol consumption (as serum GGT) and MAP in black migrant workers. However, in the hypertensive group, GGT values were significantly related to systolic pressure. Alcohol may have an indirect hypertensive action through its effect on sodium and potassium metabolism. In the coloured group a history of alcohol consumption was significantly more common among the hypertensive subjects, and furthermore serum GGT values were positively related to blood pressure independently of age, weight and cations; this suggests that in the coloured population alcohol consumption may directly influence blood pressure. This positive association was age-dependent, but the correlations were weak.

The differences in the results for the two groups may be related to the different types of alcohol consumed; spirits, for example, may have more pressor effects than wine and/or beer. It is generally believed that Johannesburg blacks consume mainly beer and wine whereas coloureds prefer wine or spirits. The sodium content of most beers is low, and the negative association found between serum GGT and serum sodium values in the black hypertensives may be due to the fact that these subjects were heavy beer drinkers. Alcohol-related hypertension appears to depend on the chronicity of alcohol intake, i.e. the pressor effect is a long-term one. The coloured subjects were older than the blacks and had probably been heavy drinkers for a longer time. (The blacks may have tended to be younger because men seeking employment or registering at a labour bureau are usually aged between 25 and 40 years.)

It is difficult to draw clear-cut conclusions about the relationships between alcohol and cations from this study, because the results are inconsistent. There were, however, significant associations between cations and blood pressure, especially in the black groups. The hypomagnesaemia in the hypertensive subjects (who had raised GGT values) may be due to the effect of alcohol. There was, however, no significant correlation between GGT and Mg++. Alcohol consumption is among the commonest causes of decreased body magnesium. The hypertensive blacks also had significantly lower levels of serum K and Ca++ and red blood cell Mg++, and raised levels of intracellular Na.

Many studies have shown a close correlation between alcohol consumption and blood pressure in alcoholics and hypertensives, as well as in the general population. This relationship may not be a direct causal one. Other compounding factors such as obesity, psychosocial stresses, and differences in salt intake may also be important. Mechanisms of alcohol-induced hypertension are numerous and include volume and sodium...
overload or increased sensitivity to renin, cortisol, angiotensin or catecholamines. Alcohol may also raise blood pressure by a direct effect on arteriolar vasculature. The precise mechanisms of alcohol in the causation of essential hypertension still need to be clarified.

We conclude that the relationship between alcohol and hypertension varies in different Johannesburg ethnic groups. Alcohol is deemed an important risk factor in coloured hypertensives, but it is not as important in young black labourers. Other factors such as cations could be more significant than alcohol in young hypertensive blacks.

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