PREDISPOSING FACTORS OF HYPERTENSION IN BLACK MALES IN THE PROCESS OF URBANISATION

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DECLARATION BY AUTHORS (*)

It is declared that all mentioned authors gave written consent to be involved in this study.

- Supervisor

Prof NT Malan: Sampling, processing and the concluding of analysed data.

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Mr R Schutte: Initiated this part of the study, and was responsible for most stages of this dissertation, including literature searches, interpretation of results and writing the dissertation.

(*): See pages 18 and 38.

AFRIKAANSE TITEL

Predisponerende faktore van hipertensie in swart mans in die proses van verstedeliking.

OPSOMMING

Sosiale en kulturele ontwrigting as gevolg van verstedeliking is 'n groot oorsaak van chroniese vlakke van stres wêreldwyd, veral in ontwikkelende lande soos Suid-Afrika. Suid-Afrika se swart populasie (77.2 persent van die totale populasie) is in 'n groot proses van verstedeliking. Daar word geskat dat stedelike groei en die toename in populasie die stedelike inwonertal sal verdubbel of selfs verdrievoudig in die volgende tien jaar. Verstedeliking word gekoppel aan 'n afname in die gesondheidstoestand van verskeie populasiegroepe, waaronder veral swartmense.

Om die moontlike afname in kardiovaskulêre gesondheid, as gevolg van die blootstelling aan faktore wat geassosieer word met verstedeliking te evalueer, is 'n groep van 348 swart Setswana-sprekende mans in hierdie studie gebruik. Die groep het bestaan uit 127 mans van monokulturele plattelandse omgewings en 221 mans van stedelike multikulturele omgewings. Hierdie groepe is onderverdeel volgens ouderdom sowel as in hipertensiewe en normotensiewe proefgroepe. Die kardiovaskulêre parameters tydens rus en tydens die uitdaging van die kardiovaskulêre stelsel is geregistreer. Die kardiovaskulêre stelsel is uitgedaag deur 'n isometriese handdinamometer teen 50% van die maksimale kapasiteit van die proefpersoon te trek. Die totale bloeddrukgolf is met die Finapres apparaat geregistreer en die "Modelflow" sagteware program is gebruik om die kardiovaskulêre parameters te bereken.

'n Verstedelikingseffek was sigbaar in die kardiovaskulêre parameters tydens rus en tydens die uitdaging van die kardiovaskulêre stelsel, alhoewel die sistoliese en diastoliese reaktiwiteitswaardes gemengde resultate opgelewer het. Die afname in kardiovaskulêre gesondheid in die verstedelikte groepe het gewys na 'n moontlike vaskulêre perifere effek. Die verandering in die kardiovaskulêre parameters tydens verstedeliking toon dieselfde patroon as die verandering wat waargeneem word tussen die normotensiewes en

hipertensiewes. Die verstedelikte groepe het 'n toename in sistoliese bloeddruk getoon as gevolg van 'n afname in arteriële meegewendheid, 'n toename in diastoliese bloeddruk as gevolg van 'n toename in totale perifere weerstand, wat 'n afname in slagvolume en kardiale omset tot gevolg gehad het wanneer hulle met die plattelandse groepe en normotensiewe groepe vergelyk was. Met die afname in arteriële meegewendheid in die ouer verstedelikte groep is daar 'n moontlike interaksie gevind tussen ouderdom en die faktore geassosieer met verstedeliking.

Bo en behalwe die bogenoemde bevindinge, word die gevolgtrekking dus gemaak dat die faktore wat geasossieer word met verstedeliking 'n nadelige effek op die kardiovaskulêre gesondheid van swart Setswana-sprekende mans in die Noordwes provinsie het. Dit is die eerste studie wat 'n interaksie tussen verstedeliking en ouderdom op arteriële meegewendheid gevind het.

ABSTRACT

Social and cultural disruption resulting from urbanisation is a major cause of chronic levels of stress all over the world, especially in developing countries like South Africa. South Africa's black population (77.2 percent of the total population) is in a major process of ubanisation. It is estimated that urban growth and population increases will double or triple the number of urban residents in the next ten years. Urbanisation is linked to a decrease in the health status of various population groups, especially regarding black people.

In order to evaluate the possible decrease in cardiovascular health due to the exposure to factors associated with urbanisation, a group of 348 black Setswana speaking men was used in this study. The group consisted of 127 men from monocultural rural environments and 221 men from multicultural urban environments. These groups were further subdivided according to their age as well as their hypertensive and normotensive state. The cardiovascular parameters during rest and while challenging the cardiovascular system were registered. Applying 50% of the maximal tractive power of the subject on an isometric hand dynamometer challenged the subject's cardiovascular system. The whole pulse wave was recorded by the Finapres apparatus and the cardiovascular parameters were calculated by making use of the "Modelflow" software program.

An urbanisation effect was apparent with the cardiovascular parameters during rest and while challenging the cardiovascular system, although the systolic and diastolic reactivity values showed mixed results. The decrease in cardiovascular health amongst the urbanised groups pointed to the possibility of an underlying peripheral vascular mechanism. The changes in the cardiovascular parameters during urbanisation show the same pattern as the changes observed between the hypertensives and normotensives. The urbanised groups showed an increased systolic blood pressure due to a decreased arterial compliance, and an increased diastolic blood pressure due to an increased total peripheral resistance, resulting in a decreased stroke volume and cardiac output when compared to the rural and normotensive groups. A possible interaction between age

and the factors associated with urbanisation was found leading to the decreased arterial compliance observed in the older urbanised group.

The conclusion, apart from the above mentioned findings, is that the factors associated with urbanisation has a deleterious effect on the cardiovascular health of black Setswana speaking males in the North West province. This is the first study to determine an interaction between urbanisation and age on arterial compliance.

ABBREVIATIONS

CO	Cardiac output
C_W	Arterial compliance (Windkessel)
DBP	Diastolic blood pressure
Finapres	Finger arterial pressure
HR	Heart rate
HT	Hypertensives
MAP	Mean arterial pressure
NT	Normotensives
SBP	Systolic blood pressure
SV	Stroke volume
THUSA	Transition and health during urbanisation in South Africa
TPR	Total peripheral resistance
WHO	World Health Organisation
Z_O	Aortic characteristic impedance

CHAPTER 1

INTRODUCTION AND LITERATURE STUDY

1. GENERAL INTRODUCTION

The present population in South Africa, currently estimated at forty three million, consists of 77.2 percent blacks[1]. As late as 1970, black people that reached fifty years of age, had a longer life expectancy than white people due to the low prevalence of the chronic diseases of lifestyle. This is no longer the case due to the recent rise in non-communicable disorders/diseases, principally hypertension, obesity, diabetes, stroke and the cancers of prosperity[1]. Hypertension is a major disease in the black population of Sub-Saharan Africa[2]. There is a higher prevalence of hypertension in the urban black population of South Africa than in whites living in the same geographical areas[3]. Several factors including age, urbanisation, socio-economic status and acculturation seem to contribute to the differences in the prevalence of hypertension between black and white people[3].

The human body always strives towards maintaining homeostasis. Chronic stress places a strain on the physiological systems that maintain homeostasis, which leads to chronic wear and tear as well as a change in the operating range of physiological systems[4]. One of the major factors that could alter homeostasis is the stress associated with altered environmental conditions such as urbanisation.

Urbanisation has been linked to a decrease in the health status of various population groups, especially blacks[5]. Fray and Douglas[5] stated that at every level of society, black people are exposed to a larger number of chronic psychosocial stresses than their white counterparts.

All racial groups are exposed to stress on a daily basis. Stein *et al.*,[6] determined in their study that the autonomic reactivity in blacks and whites were similar when exposed to a laboratory stressor. However, the way the body reacted to the stressor was dissimilar. Whites normally show an increased cardiac reactivity, while blacks normally respond with an exaggerated peripheral effect[7].

Therefore, in line with what Fray and Douglas[5] proposed above, one could hypothesise that environmental factors instead of a genetic predisposition could lie at the roots of the high prevalence of hypertension in blacks.

Black South Africans are in a massive process of urbanisation[3]. Rural people moving to towns and cities will experience cultural shock leading to a large degree of acculturation[8], and thus high levels of stress.

1. AIM

To determine if there is a relation between age, urbanisation and hypertension with a resultant vascular effect in black Setswana speaking males.

2. HYPOTHESIS

- Environmental and social factors cause a decrease in cardiovascular health among urbanised populations.
- The decrease in the cardiovascular health of Setswana speaking males is based in the peripheral vascular system with an increased total peripheral resistance and a decreased arterial compliance.

4. STRUCTURE OF THIS DISSERTATION

Due to the magnitude of variables in this dissertation, it was decided to compile two separate papers (part 1 and 2) for publication on the predisposing factors of hypertension in black males in the process of urbanisation. The present chapter gives a general overview of the literature pertaining to the two papers. Chapter 2 (part 1) and chapter 3 (part 2) investigate the influence of urbanisation on the cardiovascular system of black Setswana speaking males. Systolic blood pressure, diastolic blood pressure and heart rate baseline, stress and reactivity values were included in this paper (part 1) to establish an association between urbanisation and a decrease in cardiovascular health amongst the black Setswana speaking males. Baseline values refer to the values obtained during resting conditions while the stress values refer to the plateau values obtained during the application of the stressor. The reactivity values describe the percentage increase from

the resting to the plateau values. In chapter 3 (part 2) the influence of urbanisation is investigated in more detail by including stroke volume, cardiac output, arterial compliance and total peripheral resistance. No published papers, to the best of our knowledge, are currently available on the influence of urbanisation on cardiovascular health that included the above mentioned variables. In chapter 4 a summary of all the results are provided, recommendations are made and conclusions are drawn. The relevant references are provided at the end of each chapter according to the authors' instructions provided by the journal, *Ethnicity and Disease*. For the purpose of uniformity, a similar style of reference was used throughout this dissertation.

5. LITERATURE STUDY

5.1 Urbanisation and health

Throughout Sub-Saharan Africa there is a difference in the prevalence of hypertension between the urban and rural black populations[9]. McDade and Adair[10] stated that public health researchers have been documenting the links between urban environments and various aspects of human health for decades. It is also well established that there is no simple association between urbanisation and health. They emphasised the need for a finer level of investigation in future studies.

The rate of urbanisation is three to four times higher in developing countries, where urban growth and population increase will double or triple the number of urban residents in the next ten years[10]. The transition from a rural area, which involves a system of cultural uniformity, to an unfamiliar disrupted environment during urbanisation, may result in a stressful experience by abandoning a traditional way of life and cultural beliefs[8]. Black people in an urban environment seem to be more vulnerable to chronic diseases of lifestyle and more vulnerable to excessive increases in blood pressure during stressful daily life events[11].

Stressful life events can be defined as situational occurrences that most individuals would perceive as threatening, challenging, or requiring effort at adaptation without regard to personal coping resources[12]. When individuals are exposed to frequent or ongoing life events like these, they may exhibit persistent

psychological and physiological changes that may adversely affect their health, including the development of cardiovascular diseases[12].

Roy *et al.*,[13] stated that social support may play a very important role in buffering the stress responses of individuals to daily life events and found that this buffering role was stronger when the stressful life-event frequency was high, and disappeared when this frequency was low. He stated further that low social support was associated with an enhanced sympathetic drive. According to Fray and Douglas[5], social support may be defined as the perception or belief that there are persons to whom an individual can turn for help or assistance in times of felt need. Epidemiological evidence strongly indicates that social support is associated with better health[14]. Joseph *et al.*,[15] determined in their study in men, which migrated from the Tokelau islands to New Zealand, that the change from their traditional ways to urban living conditions involved high levels of stress associated with a loss of social support.

According to Du Preez[16], the present day urban black South African originates from the traditional black person in the tribe. There are various values and needs that are important to this traditional black person. One of these is that the social aspect or group awareness is inextricably interwoven with his world of belief. His whole understanding of values and behaviour is determined by the social group and its predominant religion. Their value system can, therefore, be termed a socio-religious system of values where far more value is attached to social form than inner values.

With the transition of the traditional black to an urban environment, he is exposed to change and foreign circumstances. The way of life, social structure, and general environment in the city differ radically from that of a tribe. This means the changeover from a regimented, cultural uniformity (high social support) to a strange, disrupted westernised environment with low social support, which is not conductive to a healthy adjustment. He now has to face a strange, confusing environment in which he has to find his way as an

individual. Social adaptation demands of him a reconciliation between the tribal, urban and Western societies[16].

Thus, it may be sufficient to assume that rural and urban settings are generally considered to represent environments exhibiting high and low social support respectively, due to the absence of accurate data currently available in South Africa.

5.2 Cardiovascular/autonomic reactivity

Cardiovascular reactivity may be defined as the change in blood pressure, heart rate, or other hemodynamic parameters in response to physical or mental stimuli[7]. Matthews *et al.*,[17] stated that there is little understanding regarding whether reactivity represents the result of a disease, a mechanism for its creation, or a marker for a common process. Yet, it may be the hyperresponsive individual who is at greatest risk for hypertensive diseases that may follow from heightened cardiovascular reactivity to stress. It is well documented that the physiological response to various laboratory stressors is normally peripherally based in blacks with an increased total peripheral resistance, and centrally (cardiac) based in whites with an increased heart rate and cardiac output[18].

Chronic social stresses augment sympathetic tone, producing norepinephrine-induced vasoconstriction in blacks, which might lead to hypertension[19]. This does not necessarily mean that environmental or psychic stress could cause the hypertension, but rather that an altered neurogenic response to mental stimuli could induce or propagate the process leading to hypertension[7].

An informative study by Stein *et al.*,[20] determined that there was no evidence that sympathetic responses were greater in blacks than in whites, yet the blacks exhibited higher vascular resistance. Based on their results, they came to three conclusions: firstly, that vascular α-adrenergic vasoconstrictor sensitivity is increased in normotensive black men; secondly, that an exogenous sympathetic stimulus, the cold pressor test, despite a

similar increase in norepinephrine spillover in blacks and whites, resulted in a greater increase in peripheral vascular resistance in blacks; and thirdly, that attenuated β-adrenoceptor-mediated vasodilation and enhanced α-adrenergic vasoconstrictor sensitivity in blacks were independent of each other and hence would produce additive effects. Walker *et al.*,[21] also found in their study on cardiovascular and plasma catecholamine responses to exercise in blacks and whites, that blacks had higher levels of epinephrine and lower levels of norepinephrine during maximal exercise when compared to whites. No racial differences were apparent for heart rate and cardiac output, but differences did exist for the total peripheral resistance with the blacks showing a greater increase, despite a decrease in plasma norepinephrine levels, which supports the finding of Stein *et al.*[20].

Another proposed mechanism is that of salt sensitivity in blacks. Although salt sensitivity was not determined in this study, the interrelationship of sodium retention and the sympathetic nervous system[22,23] cannot be ignored.

Genetic differences in salt sensitivity are evident in humans. It is well established that blacks have an increased ability to conserve salt, and that the prevalence of salt sensitivity is higher in blacks when compared to whites[23]. Deter *et al.*,[24] found an increased cardiovascular responsiveness to mental stress in salt-sensitive subjects when compared to normotensive controls. Volume loading due to altered sodium metabolism resulting from an increased activity of renal sympathetic nerves was the proposed mechanism. Mental stress is known to elicit a defense reaction, characterised by sympathetically mediated rises in blood pressure[25]. Fray and Douglas[5], suggested that exposure to acute and chronic stress may be linked to increased resting sympathetic nervous system activity, increased sodium retention, increased blood volume and increased sympathetic reactivity in blacks. The increased salt retention could in turn potentiate the sympathetic nervous system-induced vascular reactivity, completing a vicious cycle[23].

5.3 Arterial compliance (C_W)

Compliance is the total quantity of blood that can be stored in a given portion of the circulation for each millimeter of mercury pressure rise[25]. The arteries are able to store part of the stroke volume during each systole and draining this volume during diastole ('Windkessel' effect). It is important to distinguish between compliance and distensibility (elasticity) of an artery. Compliance is equal to distensibility times volume. Thus, a highly distensible vessel with a small volume will have less compliance than a less distensible vessel with a large volume[25].

It is well known that distensibility and the resulting compliance of large arteries decrease with age[26]. This normally leads to an increased pulse wave velocity, and an increase in the velocity of reflective waves leading to an increased left vetricular afterload resulting in an increase in systolic blood pressure. Ageing may not be the only factor that decreases arterial compliance. Breithaupt and Belz[27] suggested three major interacting groups of parameters that could influence the distensibility of the aorta and the large arteries, which include: physiological properties like age, gender, body height, blood pressure, hormonal state and genetic factors; environmental factors like psychosocial stress, nutrition, smoking, performance of sports and aerobic capacity; and diseases like hypertension, hypercholesterolemia, diabetes, coronary heart disease, cerebrovascular disease, renal failure, and growth hormone deficiency. Injury to the vasculature may ensue from these interacting factors. Vascular injury includes vessel wall thickening with lipoprotein deposition and endothelial denudation and dysfunction that culminate in the formation of arteriosclerotic plague. Other factors include humoral factors such as angiotensin II and catecholamines that increase the contractile state of the large arteries[28].

Arterial compliance is not only a characteristic of the large conduit arteries. The arterial tree has a capacitive component, generally referred to as C_1, and an oscillatory component, C_2[26]. The capacitive component assesses the arterial storage capacity, which is a predominant function of the larger conduit arteries. The oscillatory or reflective component is related to the cushioning effect of compliance at the arterial reflective

sites that are thought to reside primarily in small arteries and arterioles, and at branching sites of small arteries[26]. Cohn[29], stated that endothelial dysfunction results in reduced compliance or increased arterial stiffness, particularly in the smaller arteries. Beltran *et al.*,[26] discovered that when they compared isolated systolic hypertensives and essential hypertensives, the isolated systolic hypertensives showed a higher total peripheral resistance, indicating that the vascular abnormality is not confined to the large artery, but also to the oscillatory component (C_2) of the arterial tree.

5.4 Total peripheral resistance (TPR)

Total peripheral resistance is determined by resistance vessels, which are predominantly small arteries, arterioles and capillaries[30]. The hemodynamic hallmark of hypertension is a reduction in diameter of the small resistance vessels[28]. An increase in arterial pressure distends blood vessels and decreases vascular resistance. However, under sympathetic stimulation, constriction of the arterioles takes place which greatly increases vascular resistance[25]. Therefore an increased reactivity to stress leading to increased blood pressure due to an increased vascular resistance mediated by the sympathetic nervous system could be the cause or consequential mechanism underlying cardiovascular pathologies in blacks.

Lipp & Anderson[31], confirmed that social stress elevated blood pressure acutely via a vascular mechanism. As noted above, blacks at every level of society are thought to be exposed to greater levels of social stress than whites. It is well documented that blacks are vascular responders which leads to an increased peripheral resistance, with little or no significant increases in heart rate and cardiac output[23, 32]. Stein *et al.*,[20] proposed an increased adrenergic vasoconstriction and decreased vasodilation as a possible mechanism to clarify the greater vascular responsiveness found in blacks, as discussed above.

5.5 Hemodynamic variables determining blood pressure in blacks

Arterial pressure is influenced by various hemodynamic variables that can be described by the following formula[30]:

$$MAP = CO \times TPR$$

and

$$CO = SV \times HR$$

where MAP = Mean arterial pressure

CO = Cardiac output

TPR = Total peripheral resistance

SV = Stroke volume

HR = Heart rate

For the human body to maintain a constant blood pressure during rest, there needs to be a balance between cardiac output and total peripheral resistance. When exposed to a laboratory stressor, racial differences in the way the body elevates blood pressure have been observed between blacks and whites. Daniels *et al.*,[33] found that their white subjects had a higher stroke volume, cardiac output and contractility responsiveness while the black subjects had a higher total peripheral resistance responsiveness in the absence of greater heart rate reactivity due to a decreased sympathetic drive on the myocardium. The decreased cardiac output though, was largely a function of stroke volume. Grossman *et al.*,[34] conducted a study on the left ventricular filling to a stress response in essential hypertensives. They found that in essential hypertensive patients with preserved left ventricular filling had an increase in arterial pressure predominantly through an increase in cardiac output associated with a small increase in plasma norepinephrine levels. In contrast, the increased arterial pressure in patients with impaired left ventricular filling was mainly through an increase in total peripheral resistance that was associated with a marked elevation in plasma norepinephrine levels. To the extent that blacks are at considerably greater risk than whites for developing hypertension, and elevated total peripheral resistance is usually associated with hypertension, increases in total peripheral resistance rather than cardiac output in response to behavioral stressors may be significant for the development of hypertension in blacks[18]. If this is true, it suggests that the progression of hypertension development that has previously been formulated based upon observations made primarily on whites (i.e., elevated cardiac output with normal total peripheral

resistance in the early stages vs. normal cardiac output and elevated total peripheral resistance in the later stages[25]) occurs earlier for blacks before the onset of hypertension[18].

Differences in reactivity between racial groups for the hemodynamic parameters mentioned above might help clarify factors contributing to the greater risk of hypertension observed in blacks[18].

5.6 The FINger Arterial PRESsure (FINAPRES) apparatus

The method is based on the development of the dynamic (pulsatile) unloading of the finger arterial walls using an inflatable finger cuff with a built-in photoelectric plethysmograph. From the finger pressure waveform, heartbeats are detected and systolic, diastolic and mean pressure and pulse rate is obtained in a beat-to-beat mode[35].

The Finapress consists of four main components, viz:

- Rigid segmented cuff in which a light-plethysmograph is mounted.
- Proportional valve, controlling fluid pressure in the cuff.
- Manometer, reading cuff pressure.
- Servo system, which keeps the plethysmographic output from the finger constant by comparing it to a set value.

According to Wesseling[36] a potentiometer is set at a value equivalent to the amount of light detected by the plethysmograph when the venous compartment is collapsed and the arterial compartment under the cuff is compressed to its unstretched size from the outside. When intra-arterial pressure tends to increase, more blood is pressed into the arteries, thus increasing light attenuation and therefore decreasing the plethysmographic output. The difference, which now develops, is amplified and controls the proportional valve to increase its output pressure and thereby cuff pressure. Thus cuff pressure follows intra-arterial pressure in a continuous fashion, provided the servo system acts fast enough. The correct setting of the

potentiometer so that the transmural pressure is kept at zero, is of paramount importance and determines the accuracy achieved by this method. Otherwise, a positive or negative transmural pressure would remain giving systemic errors.

Zero transmural pressure occurs at a volume equal to about one-half to one-third of the open volume. When intra-arterial pressure pulsates and the cuff pressure is kept constant and in value near mean arterial pressure, the arterial volume changes can be observed dynamically from the plethysmogram, from which the open and the collapsed arterial volumes can be derived. The set point of the servo loop is then adjusted to a proper value about halfway between those extremes.

The whole pulse wave is recorded on a magnetic tape of a Kyowa, RTP-50A tape recorder. Blood pressure measured in this way is normally 6 mmHg below the actual value. The possible reason for this is the pressure gradient that exists in the arterial system of the arm between the brachial- and finger arteries. To compensate for this deviation, the finger must be held approximately 10 cm below the level of the tricuspid valve during recordings.

By using the Modelflow software program, the recorded data on the Kyowa tape is converted to digital data through which the systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), cardiac output (CO), stroke volume (SV), heart rate (HR), arterial compliance (C_W) and total peripheral resistance (TPR) can be obtained[36].

According to Jellema *et al.*,[37] the Modelflow method uses a nonlinear three-element model of the aortic input impedance and digitally computes an aortic-flow waveform from a peripheral arterial pressure signal.

The first of the three-element model is aortic characteristic impedance (Z_O) which describes the relation between pulsatile flow and pulsatile pressure at the entrance of the aorta. Left ventricular outflow increases

aortic pressure. This increase in aortic pressure depends on instantaneous flow, cross-sectional area of the aorta, and aortic compliance. Z_O represents the aortic opposition to pulsatile inflow from the contracting left ventricle. Z_O thus represents pressure divided by flow.

The second element is arterial compliance (C_W) which describes how much the pressure in the aorta increases for a certain volume of blood. A compliant aortic wall expands easily, leading to only small increases in pressure (Windkessel function). As compliance decreases, the aortic pressure increases due to a reduction in the elasticity of the aorta. The change in volume divided by the change in pressure in the aorta describes compliance.

The third element is peripheral vascular resistance that is defined as the ratio of mean pressure to mean flow. It is a measure for the ease of constant blood drainage from the compliant aorta into the vascular beds.

With the arterial pressure waveform, subject gender, and age as input, the Modelflow software computes C_W and Z_O for each new pressure sample taken at 100Hz. Instantaneous values of C_W and Z_O are used in the model simulation, resulting in the computation of an aortic-flow waveform. Integrating this waveform per beat provides left ventricular stroke volume. By multiplying stroke volume and heart rate, cardiac output is computed. Peripheral vascular resistance is calculated for each heartbeat as the quotient of mean arterial pressure and computed continuous model-simulated cardiac output.

5.7 The hand dynamometer (isometric exercise)

According to Matthews *et al.*,[17] static, or isometric exercise, normally produces a pressor response characterised by an increase in mean arterial pressure in an attempt to overcome the reduced effective perfusion pressures occurring in the region of intense muscle contraction. The pressor response increases in proportion to both intensity and amount of muscle mass recruited in the maximum voluntary contraction.

Matthews *et al.*,[17] stated further that the mechanisms involved include both central and peripheral mediation, hence active and passive coping properties. Active coping is associated with a beta-adrenergic influence on the heart that increases the systolic blood pressure and heart rate of subjects. In contrast, passive coping is associated with an alpha-adrenergic influence on the vasculature leading to an increase in total peripheral resistance. This was supported by Grossman *et al.*,[34], who found that isometric stress increased mean arterial pressure by thirty percent by an increase in cardiac output and total peripheral resistance associated with an increase in plasma catecholamine levels.

Isometric exercise is usually performed by having the subject execute a maximum voluntary contraction and then sustain a force at fifty percent of maximum voluntary contraction for one to three minutes[17].

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CHAPTER 2

THE INFLUENCE OF URBANISATION ON THE CARDIOVASCULAR SYSTEM OF BLACK TSWANA SPEAKING MALES: PART 1

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Key words: urbanisation, cardiovascular health, blacks, stress, hypertension, systolic blood pressure, diastolic blood pressure, urban, rural

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Running head: Urbanisation and cardiovascular health

Acronyms: THUSA = Transition and Health during Urbanisation in South Africa; Finapres = finger arterial pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate

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ABSTRACT

Objective: Urbanisation is a characteristic of developing countries worldwide. People undergoing such a transition from a homogenous cultural environment to a more westernised urban environment are exposed to various psychological and physiological stresses that are believed to adversely affect their health. Chronic diseases of lifestyle, like diabetes and hypertension, are more prominent amongst these people suggesting an increased susceptibility. Blacks in South Africa are in a massive process of urbanisation. This study was conducted to determine if there is a decrease in cardiovascular health amongst Setswana speaking males in transition to confirm or counter the general worldwide trend. **Design:** The study included 348 subjects where 127 of these subjects came from monocultural rural environments and 221 subjects from multicultural urban environments. **Methods:** The Finapres apparatus was used to obtain the baseline (resting) and stress systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR). Stress was induced in the subjects by means of the hand dynamometer (active and passive coping stressor). Cardiovascular reactivity was calculated as the percentage change from the resting to plateau stress values. **Results:** 3.94 percent of the rural subjects and 16.29 percent of the urbanised subjects were hypertensive. The SBP and DBP baseline and stress values generally increased from the rural to the urbanised groups. The HR was of little significance. **Conclusions:** A decrease in cardiovascular health was apparent amongst the urbanised Setswana speaking males as a result of a possible urbanisation effect. The underlying mechanisms could be peripherally based.

INTRODUCTION

The excess of hypertensive diseases among urban blacks has been recognized since early in the twentieth century[1], and the emphasis has been laid upon some vaguely defined aspect of their genetic make-up. Cooper *et al.*[2] suggested that a more fruitful approach to understanding the high levels of hypertension among blacks would begin by abandoning conventional hypotheses about race. To do this, one has to recognize that hypertension arises through many different pathways, involving complex interactions among external factors (the environment, stress, and diet), internal physiology, and the genes involved in controlling blood pressure. One approach to evaluate the influence of different environmental factors on cardiovascular health would be to keep the genetic background of people in distinct environments constant, and focus on the variations in their living conditions or behaviour[2]. By doing this, overwhelming evidence has suggested that one or more of the changes occurring during urbanisation could lie at the root of this problem[3-10].

With the migration of black people to urban areas, social and cultural disruption takes place and a more westernised lifestyle is adopted. Among the many changes associated with urbanisation, an increase in psychosocial stress is no exception[11]. Urbanisation is usually accompanied by a lack of social support, which causes stress. Social support is defined as the perception or belief that there are persons to whom an individual can turn for help or assistance in times of need[11]. When there is a lack of this support, a person that is incompatible with his or her environment will experience a continuous state of stress that will in turn result in chronic elevations in sympathetic outflow[11]. Observations on the adverse effect of chronic environmental stress on blood pressure, and the incidence of hypertension in populations have shown abnormal physiological responses to stressful stimuli. This may play a role in the development of human hypertension with augmented reactivity that is often observed in hypertensive patients[12]. Racial differences in reactivity have been shown, with enhanced vascular reactivity observed among blacks compared with greater cardiac reactivity in whites, although the role that reactivity plays in hypertension and the development thereof remains to be determined[13]. Reactivity is defined as the change in blood pressure, heart rate or other hemodynamic parameters in response to physical or mental stimuli[13].

The aim of this study was to determine if the effects of urbanisation, which is widely documented to cause an increase in the prevalence of hypertension, also occurred among the uniquely selected groups of black Setswana speaking males. This group was selected from distinct environments that would adequately represent people from rural and urban settings, so that a clear distinction could be made on the effects of urbanisation on cardiovascular health.

METHODS

The present study stems from the THUSA (Transition and Health during Urbanisation in South Africa) study, which included a cross-sectional, multi-disciplinary survey conducted from 1996 to 1998 in the North West province, South Africa[14]. Thirty-seven randomly selected sites, representing all the districts in the North West, were investigated in rural and urban areas.

A cardiovascular response was elicited by a laboratory stressor in the subjects to evaluate the groups' cardiovascular reactivity. Static or isometric exercise by means of the hand dynamometer was used to create the stress response in the subject. This type of stressor is regarded to have active as well as passive coping properties resulting in both central (cardiac) and peripheral (vascular) mediation[15].

Experimental groups

The subjects were divided into two main groups according to their level of urbanisation and further subdivided according to age. These age groups were also subdivided into normotensives and hypertensives (Fig. 1).

Group A (rural) consisted of men living in traditional African villages with a tribal head. Group B (urbanised) included men living in informal housing areas, also known as 'squatter camps', found adjacent to all major towns and cities as well as subjects from established urban townships (previously known as black locations),

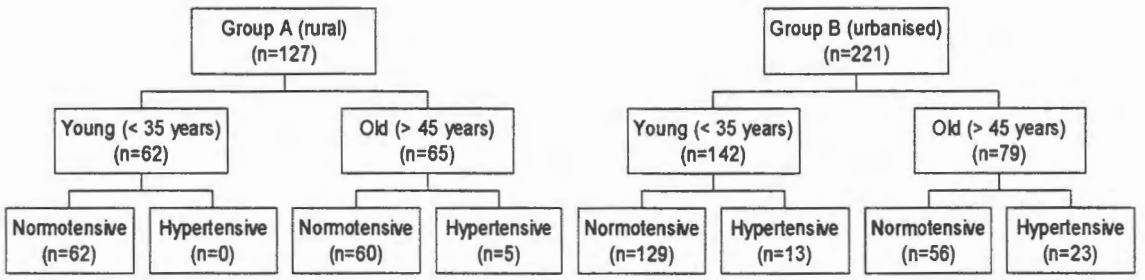

Fig. 1 Schematic representation of group subdivision according to level of urbanization, age, normo- and hypertensive state.

working as labourers in various industries and institutions[14]. After the division of the rural group (group A) and the urbanised group (group B) according to age, these groups were further subdivided into hypertensives and normotensives according to the guidelines of the WHO (≥140/ 90mmHg)[16].

The rationale for the division of groups A (rural) and B (urbanised) was to study the effect of urbanisation on the cardiovascular health of Setswana speaking men. To be able to look at the effect of urbanisation on young and old men separately, and to eliminate the effect of ageing, groups A and B were subdivided into young (< 35 years) and old (> 45 years). By further subdividing each age group into normo- and hypertensives, the differences in cardiovascular health between hypertensives, and hypertensive and normotensive black males at different levels of urbanisation could be studied.

Exclusion criteria included the use of antihypertensive medication; epilepsy; a history of, or current psychotherapy and serious metabolic, cardiovascular, or serebrovascular illnesses. People suffering from any illness that could have an influence on absorption through the gastrointestinal tract for example bulimia nervosa and anorexia nervosa were also excluded[17]. All research subjects gave written informed consent and minors participated with parental permission. The Ethics Committee of the University of Potchefstroom approved the study.

Experimental procedure

A multidisciplinary research team was responsible for the collection of the data relevant to the study. Fieldworkers were recruited before the start of the project and trained on how to recruit the subjects and what information to provide them with. Information regarding the date, time and place where recordings were to take place, was relayed via the fieldworkers to the subjects.

On the day of the recordings, the subjects were received and introduced to the experimental procedure and apparatus in their language medium, namely Setswana. Questionnaires, with the help of the fieldworkers, were completed through which information from the subjects pertaining to language, date of birth, family history of illness, use of medication, socio-economic status as well as their qualifications and occupations were obtained.

After completion of the questionnaire, the subject was seated in a separate room for the purpose of creating a calm, relaxed atmosphere and connected to a Finapres (finger–arterial pressure) apparatus[18]. The subject was allowed to relax for 10 minutes for the blood pressure to stabilize and the resting or baseline blood pressure was recorded continuously by the Finapres apparatus for one minute thereafter.

On completion of the baseline recording, the subject was exposed to physical stress (isometric exercise) by applying 50% of maximum tractive power for one minute while the Finapress apparatus recorded the blood pressure. Thereafter the apparatus was disconnected from the subject.

The pulse wave was recorded on a magnetic tape of a Kyowa, RTP-50A tape recorder. By using the Modelflow software program, the recorded data on the Kyowa tape was converted to digital data[19] to obtain the systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR). Cardiovascular reactivity was calculated as the percentage change from resting to plateau values obtained during the application of the stressor.

Statistical analysis

The data was transferred to a Microsoft Office spreadsheet, Microsoft Exell, where further processing of the data was performed. The computer software package Statistica w/5.0 was used for the statistical analysis of the data. The T-test for independent samples was used to show significant differences between groups. The Tukey HSD test (ANOVA) for unequal group sizes (Spjotvoll/Stoline test) was used to show significant differences between the different groups while compensating for unequal group sizes. The Fischer exact probability test[20] was used to determine if there was a relation between urbanisation and hypertension. The results were seen as statistically highly significant when the p-value was less or equal to 0.01 and statistically significant when less or equal to 0.05.

RESULTS

From Fig. 2 it is evident that there was an increase in almost all the parameters from group A (rural) to group B (urbanised).

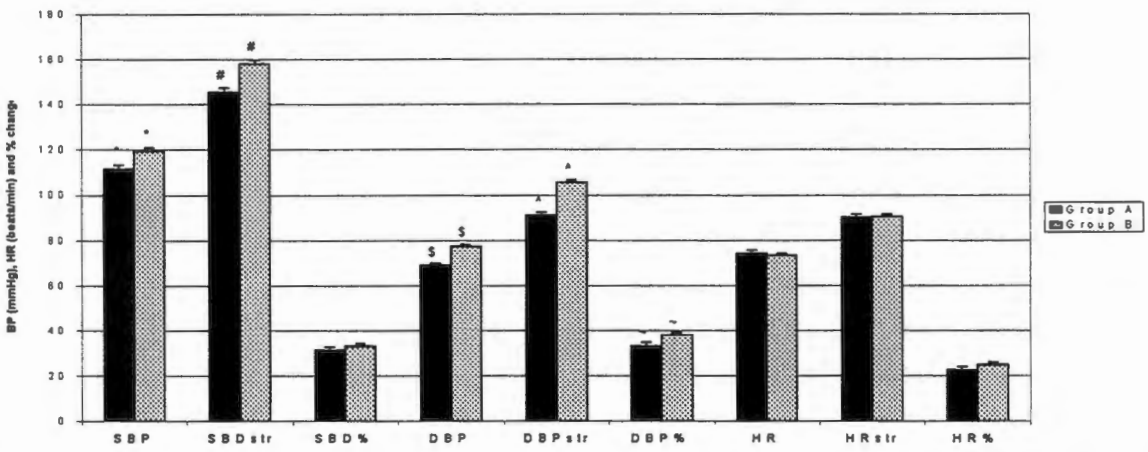

Figure 2. Comparison of all the men in group A with all the men in group B, irrespective of age. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $; ^ : $p \leq 0.01$ (Statistically highly significant)
~ : $p \leq 0.05$ (Statistically significant)

Statistically highly significant ($p \leq 0.01$) differences were apparent between groups A and B for the SBP baseline values (Group A: 111.6±17.3 mmHg; Group B: 119.6±19.6 mmHg) and DBP baseline values (Group A: 69.0±11.6 mmHg; Group B: 77.3±13.4 mmHg), as well as during the application of the hand dynamometer for the SBP (Group A: 145.5±22.1 mmHg; Group B: 158.1±25.6 mmHg) and DBP (Group A: 91.0±14.6 mmHg; Group B: 105.5±18.5 mmHg). The DBP reactivity values showed a statistically significant ($p \leq 0.05$) increase from group A (33.2±17.6%) to group B (37.9±19.9%). During the application of the stressor (stress values), the SBP and DBP and HR for both groups increased significantly ($p \leq 0.01$) from baseline to stress (significance not shown). The SBP for group B increased by 37.2±18.6 mmHg compared to a 33.7±16.2 mmHg increase for group A. Group B showed a 27.4±13.9 mmHg increase for the DBP and group A a 21.8±10.6 mmHg increase.

By subdividing groups A and B into young (< 35 years) and old (> 45 years) and comparing the same age groups from groups A and B (Fig. 3 and 4), the same trend was apparent.

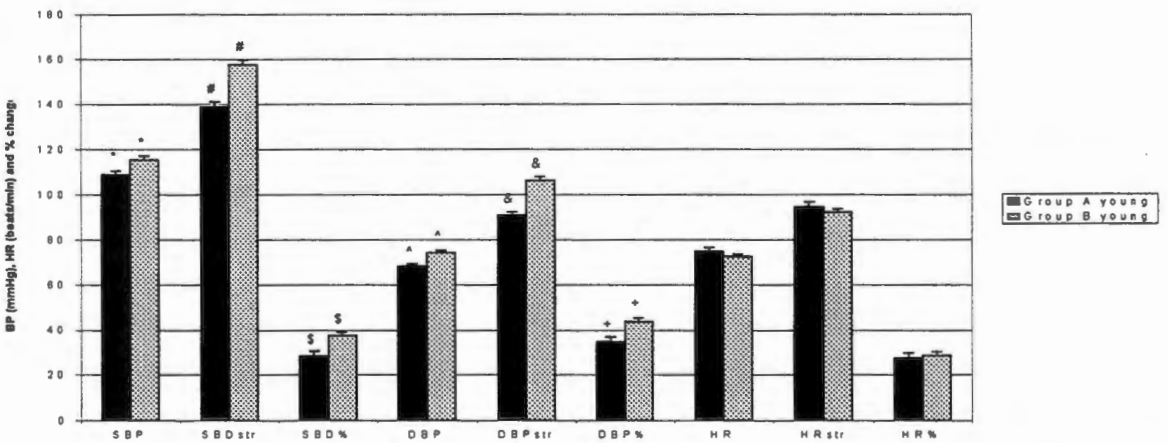

Figure 3. Comparison of young men (< 35 years) from group A with young men (< 35 years) from group B. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $; ^; &; + : $p \leq 0.01$ (Statistically highly significant)

The increase in the parameters (SBP and DBP) from baseline to stress for the younger groups (Fig. 3) were highly significant ($p \leq 0.01$). In group A the SBP (baseline: 108.8±13.9 mmHg; stress: 138.9±17.9 mmHg)

increased with 30.1±15.5 mmHg, the DBP (baseline: 68.3±9.9 mmHg; stress: 91.0±11.3 mmHg) with 22.7±9.8 mmHg and the HR (baseline: 74.9±14.6 beats/min; stress: 94.7±17.4 beats/min) by 19.8±11.6 beats/min compared to group B where the SBP (baseline: 115.5±17.6 mmHg; stress: 157.5±24.5 mmHg) increased 40.9±23.4 mmHg, DBP (baseline: 74.4±12.0 mmHg; stress: 106.3±18.6 mmHg) increased 31.1±16.7 mmHg and the HR (baseline: 72.6±13.9 beats/min; stress:92.4±17.5 beats/min) 19.2±14.4 beats/min. Highly significant differences ($p \leq 0.01$) between groups A and B for the younger subjects were evident for the SBP and DBP baseline, stress and reactivity (SBP=Group A:28.6±16.7%; Group B:37.5±18.4% and DBP= Group A:34.7±17.2%; Group B:43.9±19.8%) values.

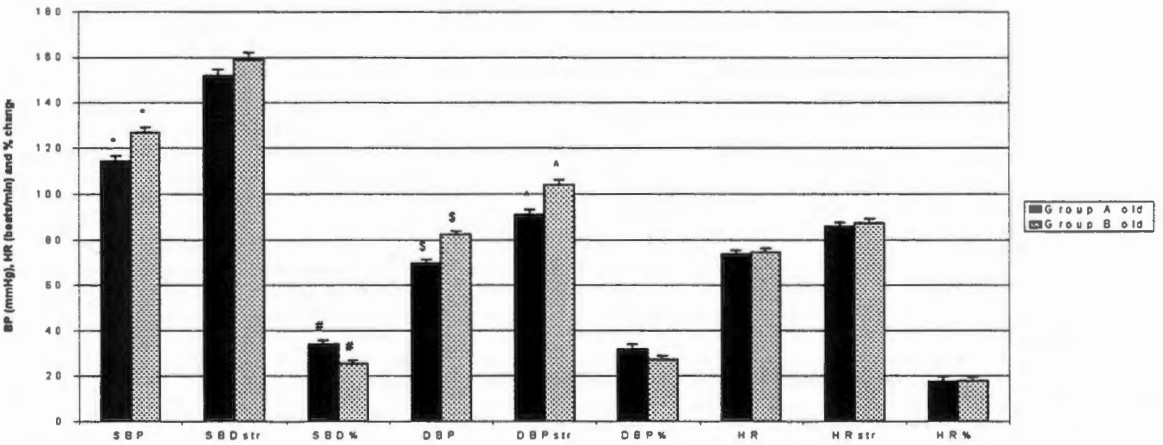

Figure 4. Comparison of old men (> 45 years) from group A with old men (> 45 years) from group B. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $; ^ : $p \leq 0.01$ (Statistically highly significant)

A slightly different pattern can be seen with the older groups (Fig. 4). Although there was still an increase for the SBP baseline (Group A: 114.4±19.8 mmHg; Group B: 127.0±21.0 mmHg) and stress (Group A: 151.9±24.0 mmHg; Group B: 159.0±27.6 mmHg) values and the DBP baseline (Group A: 69.7±13.0 mmHg; Group B: 82.3±14.3 mmHg) and stress (Group A: 91.0±17.2 mmHg; Group B: 104.2±18.5 mmHg) values from groups A to B, the percentage change from baseline to stress (SBP) was higher in group A (33.9±15.3%) leading to a higher reactivity value compared to group B (25.5±12.1%). Group A's SBP increased by 37.0±16.1 mmHg, compared to group B's 32.0±14.9 mmHg increase. There were highly

significant ($p \leq 0.01$) differences between groups A and B for the SBP baseline and reactivity values (percentage change) as well as for the DBP baseline and stress values.

By comparing the hypertensives (HT) and normotensives (NT) of the same age groups in groups A and B, a more detailed reflection is obtained.

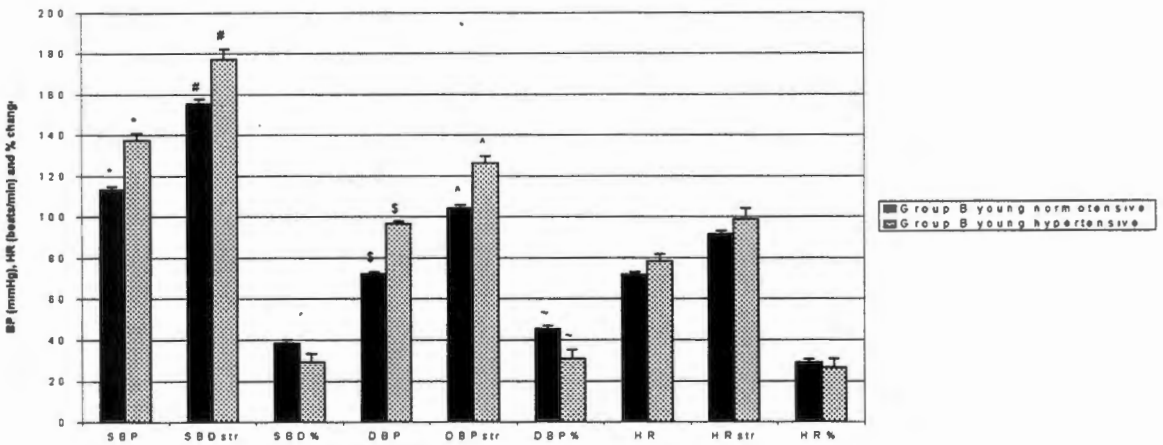

Figure 5. Comparisons of normotensive young men (< 35years) from group B with hypertensive young men (< 35 years) from group B. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $; ^ : $p \leq 0.01$ (Statistically highly significant)
~ : $p \leq 0.05$ (Statistically significant)

There were no young HT subjects in group A. By comparing the young HT with the young NT from group B (Fig. 5), it is apparent that the NT group showed a greater percentage change from baseline to stress for all the parameters. A finding which is similar to the older urbanised group (Fig. 4). Highly significant ($p \leq 0.01$) differences between the NT and HT (Fig. 5) for the SBP baseline (NT: 113.3±16.4 mmHg; HT: 137.6±12.0 mmHg) and stress (NT: 155.6±24.1 mmHg; HT: 177.2±19.2 mmHg) values and DBP baseline (NT: 72.2±10.0 mmHg; HT: 96.6±4.9 mmHg) and stress (NT: 104.3±17.9 mmHg; HT: 126.2±12.6 mmHg) values were apparent. A statistically significant ($p \leq 0.05$) difference between the HT and NT could be seen with the DBP reactivity values (NT: 45.2±19.8%; HT: 30.9±14.9%).

The same can be seen when the older NT and HT from group A were compared (Fig. 6), and the older NT and HT from group B were compared (Fig 7).

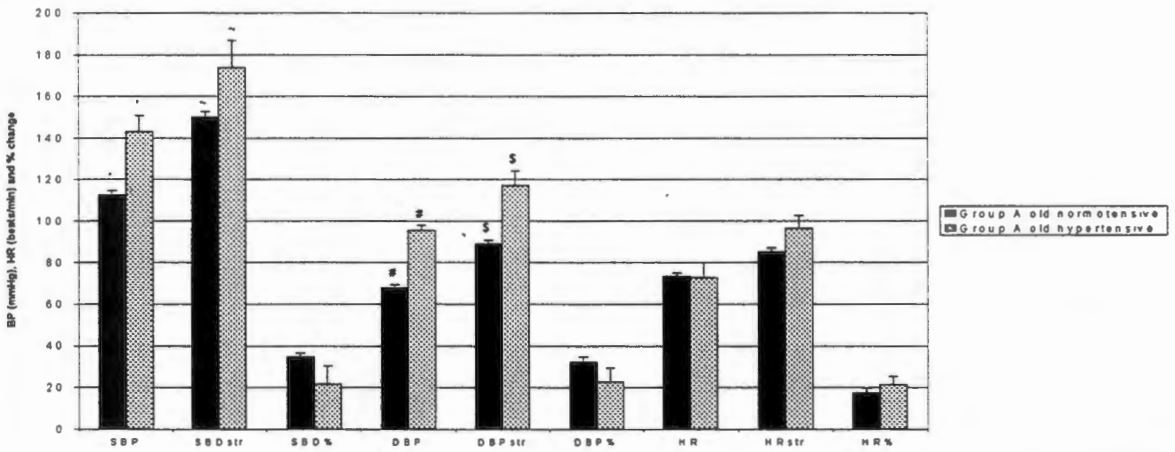

Figure 6. Comparison of normotensive old men (> 45 years) from group A with hypertensive old men (>45 years) from group A. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $: $p \leq 0.01$ (Statistically highly significant)
~ : $p \leq 0.05$ (Statistically highly significant)

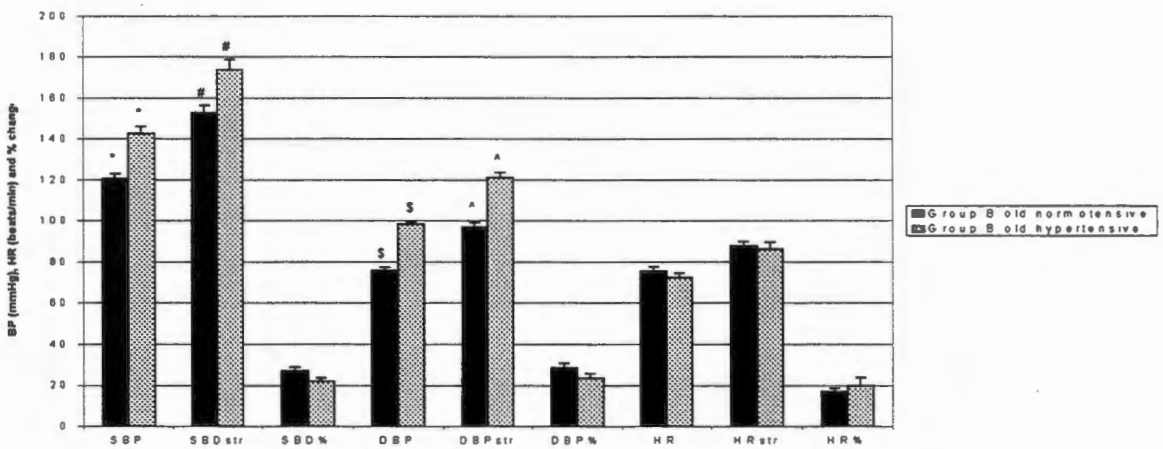

Figure 7. Comparison of normotensive old men (> 45 years) from group B with hypertensive old men (> 45 years) from group B. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

*; #; $; ^ : $p \leq 0.01$ (Statistically highly significant)

Statistically highly significant ($p \leq 0.01$) differences between the older NT and HT from group A were apparent for the SBP baseline (NT: 112.3±18.2 mmHg; HT: 143.2±17.2 mmHg) and DBP baseline (NT:

67.9±11.3 mmHg; HT: 95.3±5.5 mmHg) and stress (NT: 88.8±15.4 mmHg; HT: 116.9±16.3 mmHg) values, while a significant ($p \leq 0.05$) difference was reflected by the SBP stress (NT: 149.8±22.7 mmHg; HT: 173.6±29.2) mmHg values.

The older NT and HT from group B showed statistically highly significant ($p \leq 0.01$) differences for the SBP baseline (NT: 120.5±19.28 mmHg; HT: 142.6±16.3 mmHg) and stress (NT: 152.8±27.1 mmHg; HT: 174.0 mmHg) values and DBP baseline (NT: 75.8±11.1 mmHg; HT: 98.4±6.3 mmHg) and stress (NT: 97.1±16.2 mmHg; HT: 121.3±11.4 mmHg) values.

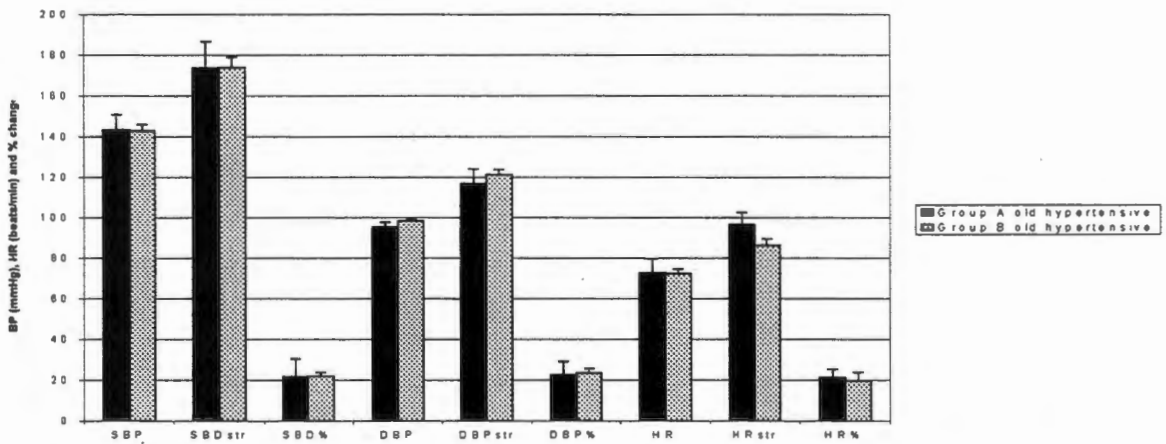

Figure 8. Comparison of hypertensive old men (> 45 years) from group A with hypertensive old men (> 45 years) from group B. Baseline systolic blood pressure (SBP), systolic blood pressure-stress (SBPstr), percentage change from baseline (SBP%), baseline diastolic blood pressure (DBP), diastolic blood pressure-stress (DBPstr), percentage change from baseline (DBP%), baseline heart rate (HR), heart rate-stress (HRstr), percentage change from baseline (HR%).

By comparing the HT of the same age group from group A and B (Fig. 8), no significant differences were obtained in the young or old groups.

As mentioned there were no young hypertensives in group A. It is worth noting that there was a slight increase in the DBP parameters when the older hypertensive groups from groups A and B (Fig. 8) were compared.

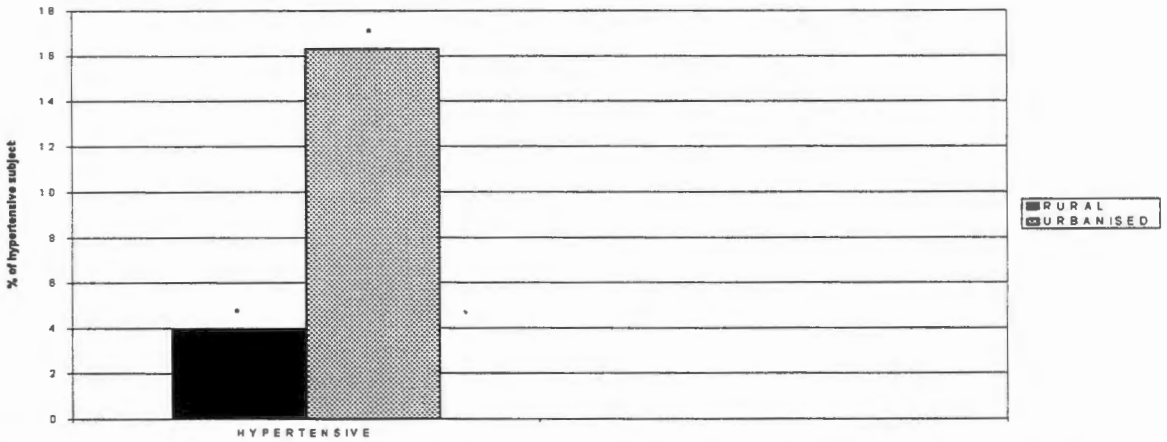

Figure 9. The comparison of the percentage of hypertensives from the rural group (Group A) with the percentage of hypertensives from the urbanised group (Group B).

* : $p \leq 0.01$ (Statistically highly significant)

Fig. 9 compares the percentage hypertensives in the rural group (3.94%) with the percentage hypertensives in the urbanised group (16.29%).

DISCUSSION

Hypertension accounts for approximately 7 percent of all deaths worldwide[2]. It is known that black South Africans, who constitute about 77.2 percent of the South African population, have been shown to have a significantly higher hypertensive mortality and morbidity rate than white South Africans[21].

Moving from a monocultural rural setting to a multicultural urban environment is a stressful experience[22], and major life events like these increases the vulnerability to chronic disease[23]. Urbanisation is expected to increase the level of cardiovascular disease risk factors as a result of the adoption of new dietary habits, sedentary lifestyle and the stress of working and living conditions in an urban area[24].

The results show a possible urbanisation effect on the cardiovascular health with 3.94 percent of the rural group being hypertensive compared to the 16.29 percent ($p \leq 0.01$) of the urbanised group (Fig. 9). There were

no hypertensives among the younger (< 35 years) subjects from the rural group (Group A) and only 5 hypertensives among the older (> 45 years) rural subjects. Thus 5 out of 122 subjects were hypertensive in the rural group. The urbanised group on the other hand, had 13 young hypertensive subjects and 23 older hypertensive subjects. In this case, 36 out of 221 subjects were hypertensive. This finding, that a decline in cardiovascular health results from the transition from a rural to an urbanised setting, was confirmed by several studies[4-8, 17, 21, 25, 26-28]. Malan[17] concluded that the underlying mechanism involved in the heightened blood pressure of the urbanised group could be peripherally based with an increased total peripheral resistance (TPR), which increases DBP. Another indicator of blood pressure that is peripherally based is arterial compliance (C_W) which normally increases systolic blood pressure when it decreases[29]. Although TPR and C_W were not included as variables in this study, the SBP and DBP parameters of the present study can possibly support a peripheral effect due to the HR which shows little change between the groups. This is illustrated when the rural (group A) and urbanised (group B) groups were compared showing greater DBP baseline ($p \leq 0.01$), stress ($p \leq 0.01$) and reactivity ($p \leq 0.05$) values for the urbanised group (Fig. 2). This is also true for the SBP baseline and stress values (Fig. 2). The increased cardiovascular reactivity in the urbanised group, compared to the rural group, was contradicted by Malan *et al.*,[30] but supported by Van Rooyen *et al.*[9].

After controlling for age (Fig. 3 and 4), a similar pattern was observed for the SBP and DBP baseline and stress values. Interestingly enough the older urbanised group showed a highly significant ($p \leq 0.01$) decrease in their SBP reactivity values when compared to the older rural group. According to Fig. 4, the DBP reactivity values showed a decrease from the old rural to the old urbanised group, although not significant. When expressed in absolute values (mmHg) from baseline to stress, the SBP and DBP for the urbanised group show a more extensive mmHg increase. The decrease in the SBP and DBP reactivity expressed as a percentage could be explained by the higher baseline values of the older urbanised group leading to a weakening of the percentage value. The same trend was observed when the normotensives and hypertensives were compared

(Fig. 5, 6 and 7). This might lead to the question of how useful reactivity is when expressed as a percentage change, compared to the absolute change of values from baseline to stress.

When normotensives and hypertensives were compared after controlling for age and the level of urbanisation, similar results were obtained for the SBP and DBP baseline and stress values as when the rural and urbanised groups were compared. Both the urbanised groups and hypertensives showed similar tendencies.

Falkner[13] stated that the risk for future hypertension is significantly increased in individuals exhibiting a 20 mmHg increase in SBP during the cold pressor test (passive coping stressor). Although the hand dynamometer was used in this study (active and passive coping stressor[15]), the rural and urbanised groups exhibited SBP increases in excess of 20 mmHg during the isometric exercise. Although the rural groups, according to Falkner[13], are also at risk, only 3.94 percent of the rural subjects were hypertensive (Fig. 9).

It has been determined that a monocultural rural environment represented a higher level of social support when compared to an urbanised environment[31]. No accurate data is currently available on the levels of social support in the rural and urban communities of South Africa.

According to Du Preez[32], the traditional South African black in transition from a rural to an urban environment is exposed to changing and strange circumstances. The way of life, social structure, and general environment in the city differ radically from that of tribal areas. This means the changeover from a regimented, cultural uniformity (high social support) to an unfamiliar, disrupted westernised environment with low social support, which is not conductive to health[32]. He now has to face a strange, confusing environment in which he has to find his way as an individual. Social adaptation demands of him a reconciliation between the tribal, urban and Western societies[32]. In light of this, it may be sufficient to assume that rural and urban settings are generally considered to represent environments exhibiting high and low social support respectively, due to the lack of accurate data currently available in South Africa.

Roy *et al.*[23] determined that changes in the SBP, DBP and TPR from baseline to stress in their study, was smaller in the high social support groups (rural) compared to the low social support groups (urbanised) suggesting a buffering function to cardiovascular stress responses in the high social support groups. This finding is supported by our results for the SBP and DBP (Fig. 2 and 3), but not in the old urbanised group which showed mixed results. Thus, higher levels of social support, which might persist in the rural setting, can possibly explain a smaller increase from baseline to stress shown by our results.

Falkner[13] further stated that under stress, there are indeed detectable alterations in the function of the peripheral vascular system among blacks, with heightened vasoconstriction, blunted vasodilation, or both. This is supported by the findings of Stein *et al.*[12] who compared a group of black and white males. They found that an exogenous sympathetic stimulus, the cold pressor test, despite a similar increase in norepinephrine spillover in blacks and whites, resulted in a greater increase in TPR in the black group. The explanation for this was that of an increased α-adrenergic sensitivity coupled with attenuated β-adrenergic and nitric oxide-mediated vasodilation (in conductance and resistance vessels) in blacks under conditions of stress, hence urbanisation. This finding can possibly explain the higher SBP and DBP baseline and stress values for the urbanised groups due to the possibility of a decreased C_W and an increased TPR respectively resulting from a decreased buffering capacity to sympathetic outflow.

We conclude by supporting the findings that urbanisation plays a major role in the decrease in cardiovascular health by reporting a similar finding amongst black Setswana speaking men. We further conclude that the apparent lack of social support experienced by urbanised people, leading to stress, can possibly be one of the reasons for the decrease in cardiovascular health due to increased sympathetic outflow and a reduced buffering capacity to this outflow. Altered adrenergic receptor sensitivity is speculated upon as a possible underlying mechanism in the periphery. We also note that the absolute values pertaining to the increase from baseline to stress can be a more accurate indicator of cardiovascular reactivity than when expressed as a percentage.

Future studies should include parameters such as TPR, C_W and related parameters, which could possibly lead to a more complete description of the cardiovascular health of people in transition. Adequate means to assess social support and perceived levels of stress should also be incorporated in future studies.

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CHAPTER 3

THE INFLUENCE OF URBANISATION ON THE CARDIOVASCULAR SYSTEM OF BLACK SETSWANA SPEAKING MALES

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Key words: urbanisation, cardiovascular health, blacks, stress, hypertension, systolic blood pressure, diastolic blood pressure, stroke volume, cardiac output, arterial compliance, total peripheral resistance, urban, rural

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Running head: Alterations in vascular health amongst blacks in transition

Acronyms: THUSA = Transition and Health during Urbanisation in South Africa; Finapres = finger arterial pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; CO= cardiac output; SV = stroke volume; C_W = arterial compliance; TPR = total peripheral resistance

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ABSTRACT

Objective: The prevalence of hypertension in developing countries, including South Africa, is a major problem and many factors associated with urbanisation is considered to play a major part in the etiology thereof. To our knowledge, this study is the first to include such a variety of parameters in attempting to evaluate the relationship between urbanisation and cardiovascular health in black Setswana speaking males, and to elucidate possible underlying mechanisms involved. This study forms part of a previous study. **Design:** The study included 348 black Setswana speaking males from thirty seven randomly selected sites in the North West province, South Africa. A distinction could be made between rural men, and men in transition. 127 black men came from monocultural rural environments and 221 black men came from multicultural urbanised environments. **Methods:** The Finapres apparatus was used to obtain the data relevant to the study. The systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) parameters were obtained by the previous study. Stroke volume (SV), cardiac output (CO), total peripheral resistance (TPR) and arterial compliance (C_W) were included in this study. Stress was induced in the subject with the use of a hand dynamometer, which has active and passive coping properties. Cardiovascular reactivity was calculated as the percentage change from the resting or baseline to the plateau stress values. **Results:** The decrease in cardiovascular health pertaining to the systolic and diastolic blood pressure parameters was confirmed by our results in the present study. We observed similarities between the normotensive groups and the rural groups as well as between the hypertensive and urbanised groups. The urbanised groups showed a decreased SV, CO and C_W coupled with an increased TPR when compared to the rural groups. The C_W showed a possible interaction between the factors associated with urbanisation and age. **Conclusions:** The findings of the present study complimented the findings of the previous which proposed an exaggerated peripheral effect due to a possible overactivity of the sympathetic nervous system. This overactivity of the sympathetic nervous system may be due to chronic levels of stress associated with the process of urbanisation.

INTRODUCTION

Hypertension is a major health challenge in South Africa[1]. Black South Africans, who constitute about 77.2 percent of the nation, have a significantly higher hypertensive mortality and morbidity incidence rate than white South Africans[2]. Various genetic determinants of hypertension have been proposed in an attempt to explain the increased prevalence of hypertension amongst blacks. Genetic differences between black, white and other races may indeed exist, but these differences cannot adequately explain why there is a higher prevalence of hypertension in, for example, black people. Instead an interaction between genetic and environmental factors may be required to initiate the onset of hypertension[3,4].

Urbanisation, or more accurately, the changes associated with urbanisation, has widely been reported to cause a decrease in cardiovascular health. This has been observed in Africans[5,9], African Americans[10] and the Chinese[11] to mention a few populations. Thus, populations from distinct environments all tend to show a decrease in their health status when in transition from a monocultural to a multicultural urbanised environment.

Fray and Douglas[12] suggested, based on a model by Anderson and Mc Neilly, that race should be viewed as a proxy for effect of differential exposure to chronic social and environmental stressors, rather than a proxy for the effect of genetic differences. He stated further that blacks at every level of society are exposed to a larger number of chronic psychosocial stresses than their white counterparts. Chronic activation of the sympathetic nervous system may then ensue with its accompanying deleterious effects. These deleterious effects have been observed to be peripherally based in blacks with heightened vasoconstriction, blunted vasodilation or both[13].

The increased vascular reactivity observed could possibly play a role in the etiology of the high rates of hypertension among blacks, although the role that reactivity plays in hypertension and the development

thereof remains to be determined[13]. Reactivity is defined as the change in blood pressure, heart rate or other hemodynamic parameters in response to physical or mental stimuli[13].

The present study attempted to evaluate the possibility that the process of urbanisation causes a decrease in cardiovascular health amongst blacks in transition. Although this was determined by previous studies, as mentioned above, systolic blood pressure, diastolic blood pressure and heart rate were predominantly the parameters involved. The present study added stroke volume, cardiac output, total peripheral resistance and arterial compliance as parameters and their baseline, stress and reactivity values were determined to obtain a more detailed reflection of the influence of the factors associated with urbanisation.

In order to study the decrease in cardiovascular health observed amongst people in transition from a monocultural rural environment to a multicultural urban environment, and to hold constant the genetic background, a distinct group of Setswana speaking males was selected. The subjects represented people from rural and urban environments recruited from thirty-seven randomly selected sites in the North West province, South Africa. This study forms part of a previous study[14] to evaluate the effects of urbanisation on cardiovascular health.

METHODS

Experimental groups

The division of the groups was as follow (Fig. 1):

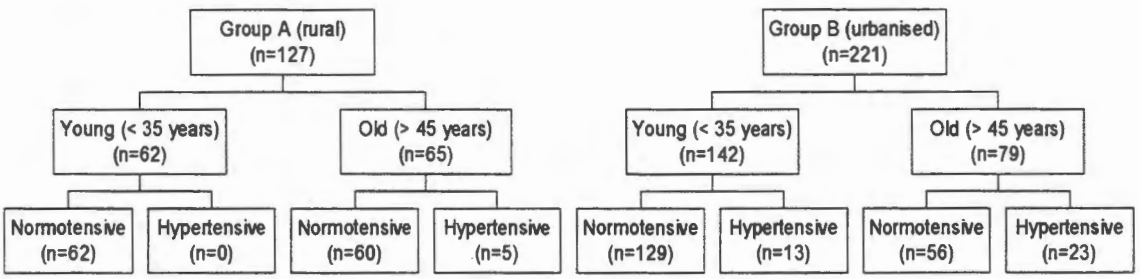

Fig. 1 Schematic representation of group subdivision according to level of urbanisation, age, normo- and hypertensive state.

Group A (rural) consisted of men living in traditional African villages with a tribal head. Group B (urbanised) consisted of subjects living in informal housing areas, also known as 'squatter camps', found adjacent to all major towns and cities, and subjects from established urban townships (previously known as black locations), working as labourers in various industries and institutions[15]. After the division of the rural group (group A) and the urbanised group (group B) according to age, these groups were further subdivided into hypertensives and normotensives according to the guidelines of the WHO (≥140/90mmHg)[16].

The rationale for the division of groups A (rural) and B (urbanised) was to study the effect of urbanisation on the cardiovascular health of Setswana speaking men. To be able to look at the effect of urbanisation on young and old men separately, and to eliminate the effect of ageing, groups A and B were subdivided into young (< 35 years) and old (> 45 years). By further subdividing each age group into normo- and hypertensives, the differences in cardiovascular health between hypertensives, and hypertensive and normotensive black males at different levels of urbanization, could be studied.

Exclusion criteria included the use of antihypertensive medication; epilepsy; a history of, or current psychotherapy and serious metabolic, cardiovascular, or serebrovascular illnesses. People suffering from any illness that could have an influence on absorption through the gastrointestinal tract for example bulimia nervosa and anorexia nervosa were also excluded[17]. All research subjects gave written informed consent and minors participated with parental permission. The Ethics Committee of the University of Potchefstroom approved the study.

Experimental procedure

A multidisciplinary research team was responsible for the collection of the data relevant to the study. Fieldworkers were recruited before the start of the project and trained on how to recruit the subjects and what information to provide them with. Information regarding the date, time and place where recordings were to take place, was relayed via the fieldworkers to the subjects.

On the day of the recordings, the subjects were received and introduced to the experimental procedure and apparatus in their language medium, namely Setswana. The subject was placed in a separate room for the purpose of creating a calm, relaxed atmosphere, and connected to a Finapres (finger–arterial pressure) apparatus[18]. The subject was allowed to relax for 10 minutes for the blood pressure to stabilize and the resting or baseline blood pressure was recorded continuously by the Finapres apparatus for one minute thereafter.

On completion of the baseline recording, the subject was exposed to physical stress (isometric exercise) by applying 50% of maximum tractive power for one minute while the Finapress apparatus recorded the blood pressure. The apparatus was disconnected from the subject on completion of the stress recording.

The pulse wave was recorded on a magnetic tape of a Kyowa, RTP-50A tape recorder. By using the Modelflow software program, the recorded data on the Kyowa tape was converted to digital data[19] to obtain the systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR) stroke volume (SV), cardiac output (CO), total peripheral resistance (TPR) and arterial compliance (C_W).

According to Wesseling *et al.*[19] and Jellema *et al.*,[20] the Modelflow method uses a nonlinear three-element model of the aortic input impedance to digitally compute an aortic-flow waveform from a peripheral arterial pressure signal. By integrating the computed aortic-flow waveform per beat provides left ventricular stroke volume. By multiplying stroke volume and instantaneous heart rate, cardiac output is obtained. Total peripheral resistance is calculated for each heartbeat as the quotient of measured arterial pressure and computed cardiac output. The three elements of this model are aortic characteristic impedance, arterial compliance and total peripheral resistance. The accuracy of this method is improved considerably as the size of the subject groups increase. Cardiovascular reactivity was calculated as the percentage change from resting to plateau stress values obtained during the application of the stressor.

Statistical analysis

The data was transferred to a Microsoft Office spreadsheet, Microsoft Exell, where it was further processed. The computer software package Statistica w/5.0 was also used for analysis of the data. The T-test for independent samples was used to show significant differences between groups. The Tukey HSD test (ANOVA) for unequal group sizes (Spjotvoll/Stoline test) was used to show significant differences between the different groups while compensating for unequal group sizes. The Fischer exact probability test[21] was used to determine if there was a relation between urbanisation and hypertension. The results were seen as statistically highly significant when the p-value was less or equal to 0.01 and statistically significant when less or equal to 0.05.

RESULTS

From table 1 and 2, a distinctive pattern emerges. Similarities can be observed by comparing the different rural and urbanised groups, and by comparing the hypertensives and normotensives within each group.

The comparison of the groups shown in Tabel 1 and 2 can be split into two parts. The first part looks at the differences between the rural and urbanised groups as a whole and within each age group. The second part focuses on the differences between normotensives and hypertensives within each age group. The hypertensives of the same age groups from the rural and urbanised groups were also compared, but not included in the data because of the absence of significant differences between these groups.

Table 1 was thoroughly discussed in the previous study[14]. To summarise, there was an apparent urbanisation effect for the systolic blood pressure (SBP) and diastolic blood pressure (DBP) parameters that generally increased from the rural to the urbanised groups. The urbanised groups and hypertensives showed similarities, while the rural groups and normotensives showed similarities.

Table 1. Summary of the different group comparisons. Previous study[14].

	SBP (mmHg)	SBP stress (mmHg)	SBP %	DBP (mm Hg)	DBP stress (mmHg)	DBP %	HR (bts/ min)	HR stress (bts/ min)	HR %
Group A	112±17 ✚	146±22 ✣	31±16	69±12 ✢	91±15 ✱	33±18 ▲	74±13	90±16	23±18
Group B	120±20 ✚	158±26 ✣	33±17	77±13 ✢	106±13 ✱	38±20 ▲	73±14	91±17	25±18
Group A young	109±14 ✚	139±18 ✣	29±17 ✢	68±10 ✱	91±11 ●	35±17 ◆	75±15	95±17	28±18
Group B young	116±18 ✚	158±25 ✣	38±18 ✢	74±12 ✱	106±18 ●	44±20 ◆	73±14	92±18	29±19
Group A old	114±20 ✚	152±24	34±15 ✣	70±13 ✢	91±17 ✱	32±18	74±11	86±13	18±17
Group B old	127±21 ✚	159±28	26±12 ✣	82±14 ✢	104±19 ✱	27±15	75±14	87±17	18±15
Group A young normotensive	109±14	139±18	29±17	68±10	91±11	35±17	75±15	95±17	28±18
Group A young hypertensive	–	–	–	–	–	–	–	–	–
Group B young normotensive	113±16 ✚	156±24 ✣	38±19	72±10 ✢	104±18 ✱	45±20 ▲	72±20	92±17	29±19
Group B young hypertensive	138±12 ✚	177±19 ✣	29±15	97±5 ✢	126±13 ✱	31±15 ▲	78±13	99±19	26±16
Group A old normotensive	112±18 ✚	150±23 ▲	34±15	68±11 ✣	89±15 ✢	32±18	73±12	85,2± 13	17±17
Group A old hypertensive	143±17 ✚	174±29 ▲	22±19	95±6 ✣	117±16 ✢	23±15	73±16	96±12	21±8
Group B old normotensive	121±19 ✚	153±27 ✣	27±13	76±11 ✢	97±16 ✱	29±16	76±16	88±17	17±12
Group B old hypertensive	143±16 ✚	174±23 ✣	22±8	98±6 ✢	121±11 ✱	24±11	72±10	86±17	20±20

✚; ✣; ✢; ✱; ●; ◆ : $p \leq 0.01$ (Statistically highly significant)
▲ : $p \leq 0.05$ (Statistically significant)

The first part of the table shows a decrease in SV baseline, stress (Table 2) and reactivity values (Fig. 2) from the rural to the urbanised groups in all three comparisons (Group A (rural) with Group B (urbanised), Group A young (< 35 years) with group B young (< 35 years), and Group A old (> 45 years) with group B old (> 45 years)).

Table 2. Summary of the different group comparisons.

	SV (ml)	SV stress (ml)	CO (l/min)	CO stress (l/min)	TPR (mmHg /ml/s)	TPR stress (mmHg /ml/s)	CW (ml/m mHg)	CW stress (ml/m mHg)
Group A	51,9±16,4	50,7±17,1 ✚	3,8±1,2	4,5±1,6 ✣	1,6±0,7 ✣	1,8±0,9 0 ✱	1,7±0,3	1,3±0,3 ●
Group B	48,9±19,0	44,1±16,5 ✚	3,5±1,2	3,9±1,6 ✣	2,0±1,0 ✣	2,3±1,2 ✱	1,6±0,3	1,1±0,4 ●
Group A young	50,8±16,5	50,7±17,8 ▲	3,7±1,2	4,7±1,7 ✚	1,6±0,6 ✣	1,7±0,8 ✣	1,7±0,3	1,4±0,3 ✱
Group B young	49,9±18,4	45,3±16,0 ▲	3,5±1,0	4,1±1,2 ✚	1,8±0,7 ✣	2,2±1,0 ✣	1,7±0,3	1,2±0,3 ✱
Group A old	53,0±16,3	50,8±16,5 ✚	3,8±1,3	4,3±1,5 ✣	1,6±0,9 ✣	1,9±1,0 ✱	1,7±0,4 ●	1,2±0,3 ◆
Group B old	47,3±20,1	42,1±17,3 ✚	3,5±1,4	3,7±1,5 ✣	2,2±1,3 ✣	2,6±1,5 ✱	1,4±0,4 ●	1,0±0,3 ◆
Group A young normotensive	50,8±16,5	50,7±17,8	3,7±1,2	4,7±1,7	1,6±0,6	1,7±0,8	1,7±0,3	1,4±0,3
Group A young hypertensive	–	–	–	–	–	–	–	–
Group B young normotensive	51,0±18,6 ▲	45,5±16,1 ✚	3,5±1,0 ■	4,1±1,2 ✣	1,8±0,6 ✣	2,1±1,0 ✱	1,7±0,2 ●	1,3±0,3 ◆
Group B young hypertensive	37,7±9,6 ▲	33,4±8,5 ✚	2,9±0,8 ■	3,2±0,7 ✣	2,7±0,8 ✣	2,9±1,0 ✱	1,4±0,2 ●	0,9±0,2 ◆
Group A old normotensive	54,2±16,1 ▲	52,3±16,3 ■	3,9±1,3 ♣	4,5±1,5 ♠	1,5±0,7 ✚	1,8±0,9 ✣	1,7±0,4 ✣	1,2±0,3 ●
Group A old hypertensive	37,7±9,9 ▲	33,2±6,1 ■	2,7±0,9 ♣	2,8±0,6 ♠	3,2±1,5 ✚	3,3±0,7 ✣	1,1±0,3 ✣	0,8±0,3 ●
Group B old hypertensive	51.4±20.8 ✚	45.9±17.6 ✣	3.8±1.4 ✣	4.0±1.5 ✱	1.8±0.9 ●	2.1±1.0 ◆	1.6±0.4 □	1.1±0.3 #
Group B old hypertensive	37,2±14,2 ✚	32,7±12,7 ✣	2,7±1,0 ✣	2,8±1,2 ✱	3,3±1,7 ●	3,8±1,9 ◆	1,1±0,2 □	0,7±0,1 #

✚; ✣; ✣; ✱; ●; ◆; □; # : $p \leq 0.01$ (Statistically highly significant)
▲; ■; ♣; ♠ : $p \leq 0.05$ (Statistically significant)

The SV baseline values (Table 2), although it decreased from the rural to the urbanised groups, did not show significant differences between groups A and B, groups A young and B young and groups A old and B old. Similar results were found for the CO. The CO stress values (Table 2) decreased from group A to group B ($p \leq 0.01$), group A young to group B young ($p \leq 0.01$) and group A old to group B old ($p \leq 0.01$). Significant

differences ($p \leq 0.05$) in CO reactivity (Fig. 2) were apparent with the comparison of group A and group B, and group A young and group B young, decreasing from the rural to the urbanised groups. Worth mentioning is that the decrease in CO reactivity (Fig. 2) from group A old to group B old had a ninety percent confidence interval ($p \leq 0.1$). The TPR baseline, stress (Table 2) and reactivity (Fig. 2) values increased from group A to group B, group A young to group B young and group A old to group B old with highly significant ($p \leq 0.01$) differences shown by the baseline and stress values. A highly significant ($p \leq 0.01$) difference from group A young to group B young was apparent for the TPR reactivity values (Fig. 2).

The C_W baseline, stress (Table 2) and reactivity (Fig. 2) values decreased from group A to group B, group A young to group B young and group A old to group B old. The comparison of groups A and B, and group A young and B young show statistically highly significant ($p \leq 0.01$) differences for the C_W stress (Table 2) and reactivity values (Fig. 2). Highly significant differences ($p \leq 0.01$) were apparent for the C_W baseline and stress (Table 2) values with the comparison of group A old and group B old.

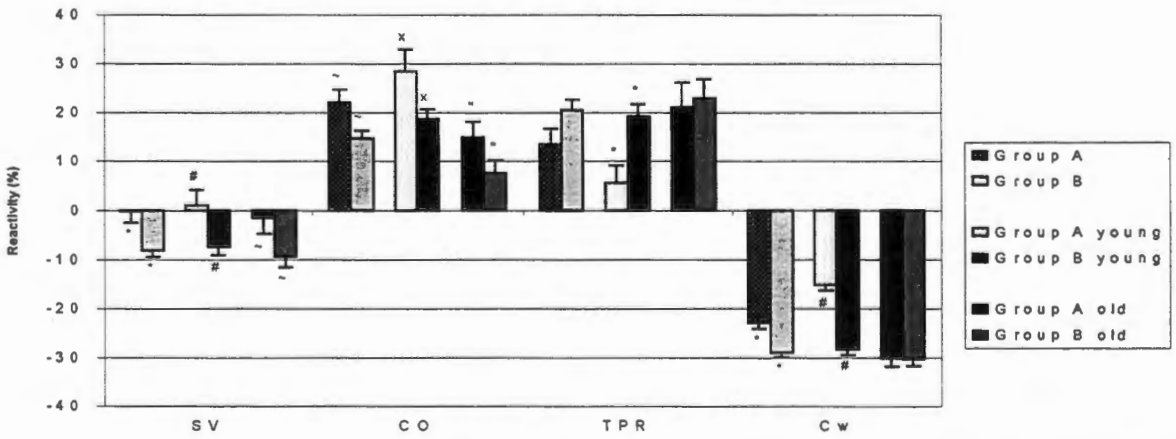

Figure 2. Comparison of reactivity (%) values between group A and group B; group A young and group B young; group A old and group B old. Stroke volume (SV), cardiac output (CO), total peripheral resistance (TPR), arterial compliance (C_W).

*; # : $p \leq 0.01$ (Statistically highly significant)
~; X : $p \leq 0.05$ (Statistically significant)
" : $p \leq 0.01$

The second part of the table, as mentioned, represents differences between normotensives and hypertensives within each age group in the rural and urbanised groups (Table 2). A similar and significant trend can be seen

with the comparison of these groups. All the comparisons (group A young normotensive with group A young hypertensive; group B young normotensive with group B young hypertensive; Group A old normotensive with group A old hypertensive and group B old normotensive with group B old hypertensive) show a statistically highly significant ($p \leq 0.01$) or a statistically significant ($p \leq 0.05$) increase or decrease for the different baseline and stress parameters from the normotensives to the hypertensives. The SV and CO parameters decreased, the TPR parameters increased and the C_W parameters decreased from the normotensive to the hypertensive groups (Table 2). None of the reactivity values showed significant differences between the normotensives and hypertensives with each comparison as mentioned above. For this reason the reactivity values of the hypertensives and normotensives are not included in Fig. 2.

DISCUSSION

It was previously determined[14] that urbanisation was indeed associated with a deterioration of cardiovascular health as far as the SBP and DBP parameters are concerned in black Setswana speaking males. The HR parameters were of no significance throughout the study. We concluded that the increase in the SBP and DBP parameters in the urbanised group was the result of a possible underlying peripheral effect. To elucidate on this conclusion SV, CO, TPR, and C_W were included in the present study.

The greater prevalence of hypertension in urban subjects indicates that the stress associated with urbanisation is important in its genesis, as ethnically both urban and rural groups are similar[22]. Chronic stress would be expected to augment sympathetic tone, producing norepinephrine-induced vasoconstriction[23].

Our results have shown that the differences between the rural and urbanised groups are similar to that of the normotensives vs. hypertensives. These similarities include a decreased SV, CO and C_W coupled with an increased TPR. An increase in TPR causes a decrease in venous return that would in turn decrease the SV and CO[24], which is a common finding in hypertensives[25]. Saab *et al.*,[25] suggested that normotensive blacks predominantly show similarities to the classic model of the progression of hypertension development that has

been determined mainly in whites i.e., elevated CO with normal TPR in the early stages vs. normal CO and elevated TPR in the later stages. Thus the effect of the factors associated with urbanisation could possibly potentiate the early onset of an elevated TPR and normal CO, explaining the similarities between the urbanised groups and the hypertensives. This is shown by our results (Table 2 and Fig. 2) for the baseline, stress and reactivity values. A possible explanation for the increased TPR and decreased C_W and hence, increased DBP and SBP respectively, from the rural to the urbanised groups, could be the result of a possible overactivity of the sympathetic nervous system due to chronic levels of stress[26]. What might further potentiate this effect is the observation made by Stein *et al.*[27] who suggested an increased α-adrenergic and decreased β-adrenergic vascular sensitivity in blacks compared to whites, without enhanced sympathetic spillover in blacks.

Although not determined in this study, salt sensitivity can briefly be speculated upon. Salt sensitivity is a heritable trait that is a hallmark of hypertension in blacks[28,29]. Fray *et al.*[12] suggested that exposure to acute and chronic stress may be linked to increased resting sympathetic nervous system activity, increased sodium retention, increased blood volume and increased sympathetic reactivity in blacks. The increased salt retention could in turn potentiate the sympathetic nervous system-induced vascular reactivity, thus completing a vicious cycle[30].

As mentioned, the results show a decrease in C_W from the rural to the urbanised groups for the baseline, stress and reactivity values. Interesting results were obtained when the young rural group was compared with the young urbanised group (Table 2), and the old rural group was compared with the old urbanised group (Table 2). Of particular interest were the baseline values. There were no significant differences between the young rural and urbanised groups, but an urbanisation effect ($p \leq 0.01$) was apparent between the old rural and urbanised groups. It is well documented that C_W decreases with age, with an increased pulse pressure, increased pulse wave velocity, and an increased velocity of reflected waves, leading to an increased ventricular afterload with a resultant increase in SBP[31,32]. When the young rural group was compared with the

old rural group, an age effect for C_W was absent. Conversely, the expected age effect was present ($p \leq 0.01$) with the comparison of the young and old urbanised groups. Although the age effect ($p \leq 0.00$; $t = 6.31$) was stronger than the urbanisation effect ($p \leq 0.00$; $t = 3.54$), it does however indicate a possible interaction between urbanisation and age because of a lack of an age effect between the young and old rural groups. This finding is supported by Breithaupt *et al.*[33] who suggested a complex interaction between physiological properties e.g. age and gender, environmental factors e.g. nutrition, and diseases e.g. hypertension that could influence arterial compliance. He further stated that physiological and environmental factors can modulate the effects of aging and that diseases generally seem to amplify the decrease in compliance.

A decrease in C_W normally results in an increased SBP and a decreased DBP[34]. The results from the previous study[14] mainly showed an increase in the SBP and DBP parameters. de Chaplain *et al.*[35] suggested that a decreased compliance cannot be due exclusively to structural changes, but may also reflect an exaggerated contribution of α-adrenoceptors in the presence of decreased sensitivity of β-adrenoceptors in the large conduit arteries.

It seems feasible to conclude that the increased levels of stress associated with urbanisation, can result in chronic sympathetic activation, possibly coupled with salt sensitivity, acting on more sensitive α-adrenergic and less sensitive β-adrenergic receptors. This will result in a decreased distensibility, and thus decreased compliance, of the conductance vessels coupled with increased arteriolar constriction (resistance vessels). The decreased compliance can possibly be the reason for the elevated SBP due to an increased ventricular afterload, and the increased DBP can be the result of the increased TPR in the urbanised groups. We conclude further that C_W decreases with age only in the urbanised group, suggesting that an interaction between age and one or more factors associated with urbanization could result in the decreased compliance, a finding that has not yet been reported.

Pulse pressure, sympathetic activity and salt sensitivity were not included in this study. Future studies should attempt to include these parameters in combination with the parameters presented in this study. By doing this, researchers may steer closer to the answer of why hypertensive diseases are so common among blacks in transition. Additionally, studies should focus more on an integrating model involving the interaction between genetic predispositions and environmental circumstances to which people are exposed. This would enable researchers to adequately counteract the problem of hypertensive diseases worldwide, especially in developing countries like South Africa.

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CHAPTER 4

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

4.1 SUMMARY OF MAIN FINDINGS

In the first paper a distinctive pattern was observed amongst the different groups. The urbanised group (Group B), young urbanised group (group B young) and older urbanised group (Group B old) showed a higher SBP and DBP baseline and stress values when compared to the rural group (Group A), young rural group (Group A young) and older rural group (Group B old) respectively, suggesting an urbanisation effect. This finding was supported by the fact that only 3.94 percent of the subjects from the rural group were hypertensive compared to the 16.29 percent in the urbanised group. The first hypothesis may thus be accepted. There were no young hypertensives in the rural group. The heart rate was of no significance and the reactivity values for the SBP and DBP showed mixed results. It was proposed that the absolute increase from baseline to plateau values in mmHg might be a more useful determinant of cardiovascular reactivity than the percentage change from baseline.

The second paper was compiled for the purpose to possibly complement the findings of the first. The urbanised groups had a decreased SV and resulting CO for the baseline, stress and reactivity values compared to the rural groups. The TPR baseline, stress and reactivity values increased while the C_W baseline, stress and reactivity values decreased from the rural to the urbanised groups. The urbanised groups showed similarities to the hypertensive groups, while the rural and normotensive groups showed similar trends.

The increased DBP could possibly be explained by the increased TPR in the urbanised groups[1]. The increased TPR could in turn explain the decrease in SV and CO due to an increased resistance to venous return to the heart[2]. The decreased C_W could possibly increase the SBP due to the possibility of an increased pulse pressure, increased pulse wave velocity, and an increased velocity of reflected waves, leading to an increased ventricular afterload with a resultant increase in SBP[3]. Our results thus indicate that the factors associated with urbanisation predominantly affected the vasculature of our subjects, resulting in the acceptance of the second hypothesis.

One of the main findings of this study pertained to the C_W baseline values when the young rural and urbanised groups and the older rural and urbanised groups were compared. An urbanisation effect was absent with the comparison of the young groups, but present with the older groups. When a possible age effect was determined, it was absent in the rural group, but present in the urbanised group. The age effect was statistically stronger than the urbanisation effect, but the fact that an age effect was absent in the rural group suggests an interaction between the effects of urbanisation and age in the urbanised group. This is a finding that has not been documented to date.

The fact that the urbanised groups showed similarities to the hypertensive groups and the rural group similarities to the normotensives was supported by the findings of Saab *et al.*,[4]. He suggested that normotensive blacks predominantly show similarities to the classic model of the progression of hypertension development that has been determined mainly in whites i.e., elevated CO with normal TPR in the early stages vs. normal CO and elevated TPR in the later stages. Thus the effect of the factors associated with urbanisation could possibly potentiate the early onset of an elevated TPR and normal CO, explaining the similarities between the urbanised groups and the hypertensives.

Due to the limitations coupled to this study, possible mechanisms could only be speculated upon. One of the main mechanisms was that of an increased alpha-adrenergic sensitivity coupled by a decreased beta-adrenergic sensitivity in the conductive and resistive blood vessels[5], possibly explaining the increased TPR and decreased C_W. Lack of social support and the general stress associated with urbanisation leading to chronic activation of the sympathetic nervous system was proposed to potentiate the above mentioned mechanism[6].

4.2 CONCLUSION

It seems feasible to conclude the following:

- The factors associated with urbanisation cause a decrease in the cardiovascular health of black Setswana speaking males, suggesting an urbanisation effect.
- The decrease in cardiovascular health is peripherally based.
- The increased SBP and DBP in the urbanised group can be ascribed to the decreased C_W and increased TPR respectively, possibly due to altered adrenergic receptor sensitivity in the vasculature.
- The cardiovascular changes associated with urbanisation correspond to the changes found in the hypertensive groups.
- There is an interaction between urbanisation and age relating to the C_W baseline values, with urbanisation possibly potentiating the age effect by decreasing compliance.

4.3 RECOMMENDATIONS

After the interpretation of the results, it became clear that the following recommendations need to be made to obtain a complete profile of the black Setswana speaking male in transition:

- Recovery time of the subject after activation need to be included.
- A well structured questionnaire adapted for the population group under study should be formulated to assess levels of social support and perceived stress.
- Sympathetic activity needs to be determined.
- Pulse pressure, pulse wave velocity and the velocity of reflective waves need to be included to adequately describe compliance.
- Salt sensitivity should be determined.

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