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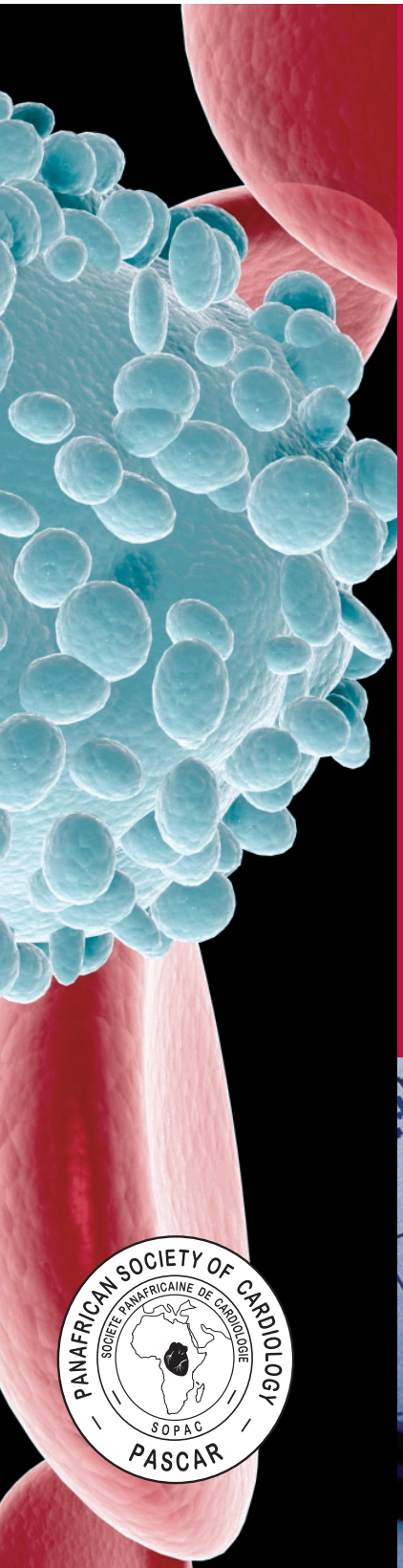

17th Congress of the Southern African Hypertension Society

3–5 March 2012

ACADEMIC PROGRAMME AND ABSTRACTS

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Academic programme and abstracts

Southern Sun Cape Sun, Cape Town, 3–5 March 2012

PROGRAMME
Theme: Hypertension, cardiovascular and stroke prevention

FRIDAY, 2 MARCH 2012		
TIME	**DESCRIPTION**	**VENUE**
18:30–20:30	Director's meeting	**Paarl Room, 2nd floor, Cape Sun**

SATURDAY, 3 MARCH 2012		
TIME	**DESCRIPTION**	**PRESENTER**
07:30–16:30	Registration open	**VOC Foyer, 3rd floor, Cape Sun**
07:30–20:00	Exhibition open	**VOC Foyer and VOC North, 3rd floor, Cape Sun**
	SESSION 1 **Congress opening**	**VOC South, 3rd floor, Cape Sun** Chairs: Prof Krisela Steyn, Prof Alta Schutte
08:30–08:35	Welcome and opening	Prof Gavin Norton, President of the Southern African Hypertension Society
08:35–09:20	The new South African hypertension guidelines	Prof YK Seedat
09:20–10:20	BP measurement – why, how, when and where?	Prof Eoin O'Brien
10:20–10:50	TEA BREAK	
	SESSION 2 **What the hypertension practitioner needs to know about:**	**VOC South, 3rd floor, Cape Sun** Chairs: Prof YK Seedat, Prof Gavin Norton
10:50–11:10	Practical lifestyle changes	Dr Vash Mungal-Singh
11:10–11:30	eGFR and microlbuminuria	Prof Yosuf Veriava
11:30–12:00	Blood pressure in pregnancy	Prof John Anthony
12:00–12:30	Management of type 2 diabetes	Prof Willie Mollentze
12:30–12:45	BREAK	
12:45–13:45	**LUNCH TIME SYMPOSIUM:** Optimal combination therapy for hypertension	Prof Neil Poulter (sponsored by Servier)
13:45–14:00	Break	
	SESSION 3 **What the hypertension practitioner needs to know about:**	**VOC South, 3rd floor, Cape Sun** Chairs: Prof Angela Woodiwiss, Prof Yusuf Veriava
14:00–14:30	Difficult-to-control BP	Prof Henry Krum (sponsored by Medtronic)
14:30–14:55	Important drug interactions	Prof Lionel Opie
14:55–15:20	Detection of LVH and diastolic dysfunction	Prof Gavin Norton
15:20–15:45	TEA BREAK	
	SESSION 4 **What the hypertension practitioner needs to know about:**	**VOC South, 3rd floor, Cape Sun** Chairs: Brian Rayner, Yvonne Trinder
15:45–16:15	Practical management of severe hypertension	Prof John Milne
16:15–16:35	The evaluation of the young hypertensive	Prof Brian Rayner
16:35–16:55	Common and less-common adverse effects of antihypertensives – A GP's perspective	Dr Yvonne Trinder
17:00–17:45	**NOVO NORDISK SPONSORED SYMPOSIUM:** Getting to the root of diabetes and the effect of GLP-1 on the cardio-vascular system	Prof Wolfgang Schmidt (sponsored by Novo Nordisk)
17:45–20:00	COCKTAIL PARTY IN THE EXHIBITION AREA	Sponsored by Novo Nordisk
18:30–20:30	SAHS Executive Committee meeting	**Paarl Room, 2nd floor, Cape Sun**

SUNDAY, 4 MARCH 2012		
07:30–16:00	Registration open	**VOC Foyer, 3rd floor, Cape Sun**
	SESSION 1	
08:30–09:15	Overview of the NICE guidelines	Neil Poulter (sponsored by Servier)
09:15–09:30	Comparisons of NICE and SA guidelines	Prof Gavin Norton
09:30–09:40	Discussion	
09:40–10:10	Prescribing drugs is not enough; BP control is what matters	Prof Eoin O'Brien
10:10–10:20	OP1: Is there an independent relationship between a high-normal blood pressure and target organ changes in a community sample of African ancestry?	Dr Hendrik L Booysen, University of the Witwatersrand (CPGRU)
10:20–10:30	OP2: From optimal blood pressure to hypertensive status over five years: what are the predictors in an African population?	Prof Alta Schutte, Hypertension in Africa Research Team (HART), North-West University
10:30–11:00	TEA BREAK	
	SESSION 2	
11:00–11:30	Drugs for the heart 2012	Prof Lionel Opie
11:30–12:00	The heart in hypertension	Prof Angela Woodiwiss
12:00–12:10	OP3: Potential mechanisms that account for obesity-related decreases in left ventricular diastolic function	Dr Carlos David Libhaber, Cardiovascular Pathophysiology and Genomics Research Unit, School of Physiology
12:10–12:20	OP4: The most reliable electrocardiographic criteria for left ventricular hypertrophy in a predominantly obese African population	Dr Fabian Maunganidze, Cardiovascular Pathophysiology and Genomics Research Unit, University of the Witwatersrand

12:20–12:30	OP5: Homocystine inhibits y^+L-arginine transport in human umbilical vein endothelial cells without affecting y^+ transport	Dr Marieta Nel, Surgery, Health Sciences, University of the Witwatersrand
12:30–13:10	Poster viewing	**Exhibition Hall, 3rd floor, Cape Sun** Poster presenters to be available at their poster
13:10–14:10	**LUNCH SYMPOSIUM:** New oral anticoagulation in atrial fibrillation: efficacy and time in therapeutic range (TTR)	Dr JL van Zyl (sponsored by Bayer)
14:10–14:15	Break	
	SESSION 3	
14:15–14:45	BP variability: are we faced with a new target for treatment?	Prof Eoin O'Brien
14:45–15:15	Beyond brachial BP – what is the role of large artery stiffness and central BP in clinical practice?	Prof John Cockcroft (sponsored by AtCor Medical and Shalom Laboratories)
15:15–15:45	Diuretics – what is the best choice?	Prof Neil Poulter (sponsored by Servier)
15:45–16:15	TEA BREAK	
	SESSION 4	
16:15–16:25	OP6: A comparison of the hypertension prevalence and quality of care in the urban black population of Cape Town between 1990 and 2008/09	Dr Nasheeta Peer, Medical Research Council
16:25–16:35	OP7: Ambulatory central aortic systolic pressure in South African CKD-5D patients	Dr Robert Freercks, Division of Nephrology and Hypertension, University of Cape Town and Groote Schuur Hospital
16:35–16:45	OP8: Intra-familial aggregation and heritability of aortic pulse pressure and pressure augmentation in a community with a high prevalence of uncontrolled hypertension	Dr Michelle Redelinghuys, Cardiovascular Pathophysiology and Genomics Research Unit, School of Physiology, Faculty of Health Sciences, University of the Witwatersrand
16:45–16:55	OP9: Contribution of circulating angiotensinogen concentrations to variations in aldosterone and blood pressure in a group of African ancestry depends on salt intake	Dr Frederic S Michel, University of the Witwatersrand
16:55–17:05	OP10: The five-year cardiometabolic changes in treated versus never-treated HIV-infected black South Africans: the PURE study	Dr Carla Fourie, HART, North-West University
17:15–18:30	SAHS AGM	**VOC South, 3rd floor, Cape Sun**
19:30–late	Congress dinner	**The Villa, 2nd Floor, Cape Sun**

MONDAY, 5 MARCH 2012		
07:30–16:00	Registration open	**VOC Foyer, 3rd floor, Cape Sun**
	SESSION 1	**VOC South, 3rd floor, Cape Sun**
08:30–09:00	Sympathetic nervous system activation in the pathogenesis of hypertension and therapeutic impact of its blockade	Prof Henry Krum (sponsored by Medtronic)
09:00–09:45	Vascular aging: causes and consequences	Prof John Cockcroft (sponsored by AtCor Medical and Shalom Laboratories)
09:45–10:40	Industry-sponsored symposium: The role of renal sympathectomy in the management of resistant hypertension	Prof Henry Krum (sponsored by Medtronic)
10:40–11:10	TEA BREAK	
11:10–11:40	New era for non-communicable disease (NCD) policy – the UN Summit on NCDs	Prof Krisela Steyn
11:40–12:10	NCD policy for the Western Cape	Prof Craig Household
12:10–12:40	Why are we losing the battle against obesity?	Prof Vicki Lambert
12:40–13:10	The role of the nurse practitioner in control of NCDs in the primary care setting	Dr Lara Fairral
13:10–14:10	LUNCH	**Exhibition Hall, VOC Foyer and North, 3rd floor, Cape Sun**
	SESSION 3	
14:10–14:40	Why do black patients respond better to CCBs and diuretics?	Prof YK Seedat
14:40–15:10	Stroke in Africa	Prof Albertino Damasceno
15:10–15:40	The importance of mineralocorticoid-related hypertension in South Africa	Prof Brian Rayner
15:40–16:00	**CLOSING SESSION:** Awarding of prizes for best oral and best poster presentation Announcements Future directions of SAHS	Prizes sponsored by Discovery Health

ORAL PRESENTATIONS

OP1: IS THERE AN INDEPENDENT RELATIONSHIP BETWEEN A HIGH-NORMAL BLOOD PRESSURE AND TARGET-ORGAN CHANGES IN A COMMUNITY SAMPLE OF AFRICAN ANCESTRY?

Hendrik L Booysen[1], Angela J Woodiwiss, Olebogeng HI Majane, Muzi J Mase, Pinhas Sareli, Gavin R Norton
Cardiovascular Pathophysiology and Genomics Research Unit, School of Physiology, Faculty of Health Sciences, University of the Witwatersrand Medical School, Johannesburg

Introduction: The role of pre-hypertension [normal and high-normal blood pressure (BP): BP = 120–139/80–90 mmHg] in the development of cardiovascular disease is controversial. We have previously demonstrated in an urban, developing community of African ancestry that pre-hypertension is not related to target-organ changes independent of alternative risk factors. However, in that previous study, we were not statistically powered to evaluate relationships between a high-normal BP (130–139/85–90 mmHg) and target-organ changes.
Aim: To evaluate the relationship between a high-normal BP and target-organ changes independent of alternative cardiovascular risk factors in an urban, developing community of African ancestry.
Methods: In 1 140 randomly recruited participants of African ancestry from the South-West Township (SOWETO) of Gauteng, nurse-derived conventional BP, left ventricular mass indexed for height$^{1.7}$ (LVMI) (echocardiography), left ventricular early-to-atrial transmitral velocity (E/A) (echocardiography), carotid–femoral (aortic) pulse-wave velocity (PWV) (applanation tonometry), and the square-root of urinary micro-albumin/creatinine ratio (ACR) were determined.
Results: Independent of age, waist circumference, gender, regular tobacco smoking, regular alcohol intake, diabetes mellitus and/or HbA_{1c} level > 6.1%, heart rate or mean arterial pressure (in the case of PWV) compared to participants with an optimal BP (< 120/80 mmHg) (n = 135–280), participants with a high-normal BP (n = 95–140) had a similar LVMI ($p = 0.40$), aortic PWV ($p = 0.45$), and square root of ACR ($p = 0.47$), and a marginally lower E/A ($p < 0.05$).
Conclusions: Compared to an optimal BP, in a community of African ancestry, a high-normal BP is not independently associated with or only marginally associated with target-organ changes. These data suggest that people of African descent with a high-normal BP may not benefit from BP reduction.

OP 2: FROM OPTIMAL BLOOD PRESSURE TO HYPERTENSIVE STATUS OVER FIVE YEARS: WHAT ARE THE PREDICTORS IN AN AFRICAN POPULATION?

Schutte AE, van Rooyen JM, Schutte R, Fourie CMT, Huisman HW, Mala
Hypertension in Africa Research Team (HART), North-West University, Potchefstroom

Background: Epidemiological data on hypertension prevalence in sub-Saharan Africa is available, but there is an urgent need for longitudinal cohorts. We explored health behaviours and conventional risk factors of Africans with optimal blood pressure (BP) (≤ 120/80 mmHg) and their five-year prediction for the development of hypertension.
Methods: The South African leg in the North West Province of the PURE study (Prospective Urban Rural Epidemiology) included 1 994 Africans (aged > 30 years). At baseline and follow up we measured BP and conventional risk factors. At follow up we additionally measured central systolic BP, carotid cross-sectional wall area (CSWA) and augmentation index.
Results: At baseline 47.7% were hypertensive (≥ 140/90 mmHg). Seventy per cent of participants with optimal BP at baseline (n = 478) were followed successfully (213 normotensive, 68 hypertensive, 57 died). Africans who became hypertensive had similar ages, gender distribution, rural/urban location, levels of employment, education, self-reported stress, physical activity, macronutrient intake and levels of cholesterol, glucose, glycated haemoglobin, C-reactive protein, and uric acid to those who became normotensive ($p > 0.05$), but they smoked more ($p = 0.009$), were abdominally more obese ($p = 0.013$), and had higher γ-glutamyl transferase ($p < 0.001$). The five-year change in SBP, DBP and pulse pressure were consistently and independently explained by baseline γ-glutamyl transferase ($p < 0.05$). Alcohol intake also predicted central SBP and CSWA at follow up. Waist circumference at baseline was a strong, significant predictor of BP changes and end CSWA ($p < 0.006$). HIV infection was inversely associated with increased BP ($p < 0.001$).
Conclusions: Over five years, 24% of Africans with optimal BP developed hypertension. Our results confirm that the surge in hypertension in Africa is largely caused by modifiable risk factors, such as alcohol intake and obesity. Public health strategies should focus aggressively on lifestyle to prevent a catastrophic burden on the national health system.

OP 3: POTENTIAL MECHANISMS THAT ACCOUNT FOR OBESITY-RELATED DECREASES IN LEFT VENTRICULAR DIASTOLIC FUNCTION

Dr Carlos David Libhaber[1], Gavin R Norton[2], Olebogeng HI Majane[2], Muzi J Maseko[2], Pinhas Sareli[2], Angela J Woodiwiss[2]
[1]Cardiovascular Pathophysiology and Genomics Research Unit, School of Physiology and Division of Nuclear Medicine, Johannesburg,
[2]Cardiovascular Pathophysiology and Genomics Research Unit, School of Physiology, Faculty of Health Sciences, Johannesburg

Introduction: Obesity predicts the development of heart failure independent of conventional risk factors. We recently demonstrated in a community sample with a high prevalence of obesity that waist circumference has a strong and inverse relationship with early-to-atrial transmitral velocity (E/A), an index of diastolic function of the left ventricle (LV). The mechanisms that account for this relationship are unclear. Metabolic, neurohumoral, inflammatory (adipokines) or haemodynamic (central aortic blood pressure) factors may play a role.
Aims: To determine the relationship between transmitral E/A and indices of insulin resistance, measures of the circulating renin–angiotensin–aldosterone system (RAAS), the inflammatory marker C-reactive protein (CRP), or central aortic BP in a community sample with a high prevalence of obesity.
Methods: Transmitral E/A and left ventricular mass indexed for height$^{1.7}$ were determined using echocardiography in 672 randomly selected participants of a community sample of African ancestry, 63% of whom were overweight or obese. Circulating RAAS and CRP concentrations were determined using immunoradiometric techniques, an ^{125}I radioimmunoassay, and a solid-phase sandwich ELISA. Central aortic BP was assessed using radial tonometry and SphygmoCor software. Insulin resistance was evaluated from the homeostasis model (HOMA-IR) as determined from fasting insulin and glucose concentrations.
Results: Independent of a number of confounders including age, gender, pulse rate, conventional diastolic (or systolic) BP, antihypertensive treatment, left ventricular mass index (LVMI) and the presence of diabetes mellitus or an HbA_{1c} level > 6.1%, waist circumference was independently and inversely associated with E/A ($p < 0.0005$). Similarly, independent of confounders, HOMA-IR was independently associated with E/A ($p < 0.01$). However, despite CRP being strongly associated with indices of excess adiposity, neither measures of the RAAS, nor CRP were independently associated with E/A ($p = 0.376$–0.73) and central aortic BP was not associated with E/A independent of conventional BP.
Conclusions: Insulin resistance may, but not the RAAS, obesity-associated inflammatory changes, or central aortic BP could explain the relationship between obesity and a reduced LV diastolic function, beyond conventional cardiovascular risk factors or LVMI.

OP 4: THE MOST RELIABLE ELECTROCARDIOGRAPHIC CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY IN A PREDOMINANTLY OBESE AFRICAN POPULATION

Fabian Maunganidze, Gavin Norton, Angela Woodiwiss, Carlos Libhaber, Olebogeng HI Majane, Muzi Maseko
Cardiovascular Pathophysiology and Genomics Research Unit, University of the Witwatersrand, Johannesburg

Introduction: Left ventricular hypertrophy (LVH) determined from electrocardiographic (ECG) recordings independently predicts cardiovascular outcomes. However, ethnicity, obesity and gender significantly affect the ability to detect LVH using ECG criteria. The use of novel ECG criteria (Composite time–voltage and Gubner-Ungerleider product) has been proposed in preference to classic ECG criteria (Cornell voltage, Sokolow-Lyon voltage, 12-lead voltage, 12-lead QRS sum) in East Africans. However, the validity of these novel ECG criteria has only been assessed in a predominantly lean (mean BMI ~ 25 kg/m^2) population.
Aims: To determine the accuracy of both classic and novel ECG criteria in a population of African ancestry with a high prevalence of obesity.
Methods: LVH determined by ECG was compared to LVH determined by echocardiography (LV mass index > 51 g/m$^{2.7}$) in 479 participants in an urban, developing community of African ancestry in South Africa.
Results. Sixty-seven per cent of participants were overweight or obese (24.8% overweight, 42.2% obese), 65.3% were female and 44.0% had hypertension. Using echocardiographic criteria for LVH, 23.8% had LVH (24.7% men, 23.3% women). The Cornell voltage emerged as the most useful ECG criterion for the detection of LVH [14.8% LVH, sensitivity = 29.0%, specificity = 89.6%, accuracy = 75.16%, negative predictive power (NPP) = 80.15%, area under receiver operating curve (AUC) = 0.646] followed by the Lewis voltage (10.23% LVH, sensitivity = 18.42%, specificity = 92.33%, accuracy = 74.74%, NPP = 78.37%, AUC = 0.554) and the Gubner-Ungerleider product (7.72% LVH, sensitivity = 17.54%, specificity = 95.34%, accuracy = 76.43%, NPP = 78.73%, AUC = 0.564). Combining these three criteria improved their ability to detect LVH (20.88% LVH, sensitivity = 37.72%, specificity = 84.38%, accuracy = 73.28%, NPP = 81.27%, AUC = 0.611). Sensitivity for the other seven ECG criteria assessed was generally poor (< 10%) in this population, although the specificity was high (> 90%).
Conclusion: In a population of African ancestry with a high prevalence of obesity and hypertension, the most reliable ECG criterion for LVH was the Cornell voltage.

OP 5: HOMOCYSTEINE INHIBITS Y$^+$L-ARGININE TRANSPORT IN HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS WITHOUT AFFECTING Y$^+$ TRANSPORT

MJ Nel[1], AJ Woodiwiss[2], GP Candy[1]
[1]Department of Surgery, Health Sciences, University of the Witwatersrand, Johannesburg
[2]Department of Physiology, University of the Witwatersrand, Johannesburg

Studies have shown an elevation in routinely measured total plasma homocysteine (tHcy) concentrations to be an independent risk factor for cardiovascular disease. Large, prospective supplementation trials conducted in patients with existing cardiovascular disease, although decreasing tHcy, showed no reduction in subsequent cardiovascular events. As only a fraction of tHcy is actually present as the free reduced sulphydryl, tHcy may be simply a marker of cardiovascular risk. As the disulphide, homocystine (HcySS) shares common transport with arginine, the nitric oxide (NO) precursor, we determined whether HcySS reduced transport of arginine and/or affected NO production using non-linear modelling of uptake.
Methods: HUVEC and ECV$_{304}$ cells were grown to confluence and depleted of arginine. Uptake of labelled [^{3}H]L-arginine was measured with unlabelled arginine, with or without HcySS or modifier. NO-specific fluorescent DAF was used to detect NO production by the cells. Kinetic constants were determined in Graphpad.
Results: HcySS significantly inhibited arginine uptake by y$^+$L transport in both HUVEC ($p < 0.0005$) and ECV$_{304}$ cells ($p < 0.05$). HcySS reduced the K_{ma} of y$^+$L transport in HUVEC cells (< 0.0001), affecting uptake in a competitive manner. Pre-incubation of the ECV$_{304}$ cells with arginine was able to reverse this inhibition by HcySS. By contrast, HcySS increased uptake by y$^+$ transport in HUVEC cells ($p < 0.01$). However, HcySS did not affect the rate of NO production.
Discussion: HcySS is almost undetectable in plasma of subjects without disease, and has been determined to be 1–2 μM in chronic renal-failure patients, and as high as 7 μM in homocysteinuric children. The study shows physiological concentrations of HcySS inhibited arginine uptake by HUVEC cells by y$^+$L transport. Previous studies have demonstrated y$^+$L transport plays a role in NO synthesis in HUVEC cells although under the experimental conditions used, effects on NO production were not shown.
Conclusion: By demonstrating that HcySS directly inhibits arginine uptake by HUVEC cells, these data may suggest HcySS to be the risk factor for occlusive cardiovascular disease, not the routinely measured tHcy. Studies to support these data are required to determine associations between elevated HcySS and occlusive cardiovascular disease.

OP 6: A COMPARISON OF THE HYPERTENSION PREVALENCE AND QUALITY OF CARE IN THE URBAN BLACK POPULATION OF CAPE TOWN BETWEEN 1990 AND 2008/09

Nasheeta Peer[1], Krisela Steyn[2], Carl Lombard[3], Nomonde Gwebushe[3], Naomi Levitt[2,4]
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[2]Chronic Disease Initiative in Africa, Department of Medicine, University of Cape Town, Cape Town
[3]Biostatistics Unit, Medical Research Council, Cape Town
[4]Division of Endocrinology, Department of Medicine, University of Cape Town, Cape Town

Objective: To determine the current prevalence, determinants and quality of care of hypertension in the 25–74-year-old urban black population of Cape Town and examine the trend between 1990 and 2008/09.
Methods: In 2008/09, a cross-sectional sample, stratified for age and gender, was randomly selected from the same townships sampled in 1990. The prevalence of hypertension and quality of care were determined by administered questionnaires and clinical measurements. Logistic regression analysis assessed the independent effects of determinants on hypertension.
Results: There were 1 099 participants (response rate 86%) in 2008/09. The age-standardised prevalence of hypertension was 39.4% (95% confidence interval (CI): 36.0–42.7) with similar rates in men and women. In 25–64 year olds, the crude hypertension prevalence increased from 25.0% (95% CI: 21.8–28.4) in 1990 to 35.6% (95% CI: 32.3–39.0) in 2008/09, reaching significance in 25–34 year olds and 35–44 year olds by age categories. BMI ≥ 30 kg/m^2 increased from 29.8% (95% CI: 26.4–33.4) in 1990 to 36.8% (95% CI: 33.1–40.8) in 2008/09. In 2008/09, increasing age [odds ratio (OR): 1.08, 95% CI: 1.07–1.10], family history of hypertension (OR: 1.43, 95% CI: 1.05–1.96), BMI ≥ 25 kg/m^2 (OR: 1.89, 95% CI: 1.28–2.74) and urbanisation (OR: 1.01, 95% CI: 1.00–1.01) were significantly associated with hypertension. Hypertension awareness, treatment and control at 48.3, 35.4 and 19.3%, respectively, among hypertensive participants in 2008/09 remained low with minimal change since 1990 (48.2, 34.9 and 13.3%, respectively), except for improved hypertension control in women (1990: 14.1%, 95% CI: 8.5–22.5 vs 2008/09: 31.5%, 95% CI: 25.9–37.7).
Conclusions: The rising prevalence of hypertension is of grave concern and requires population-based preventive strategies. In addition, the low levels of control, especially in men, need innovative interventions.

OP 7: AMBULATORY CENTRAL AORTIC SYSTOLIC PRESSURE IN SOUTH AFRICAN CKD-5D PATIENTS

Robert Freercks[1], Charles Swanepoel[1], Henri Carrara[2], Sulaiman Moosa[3], Anthony Lachman[4], Brian Rayner[1]
[1]Division of Nephrology and Hypertension, University of Cape Town and Groote Schuur Hospital, Cape Town
[2]School of Public Health and Family Medicine, University of Cape Town, Cape Town
[3]Radiologist, 2 Military Hospital, Cape Town
[4]Cardiologist, 2 Military Hospital, Cape Town

Background: Vascular calcification (VC) has emerged as a strong predictor of death in CKD-5D patients. VC is associated with stiffening of the aorta and increased aortic pulse-wave velocity (PWV). Increased PWV is associated with augmentation of central aortic systolic pressure (CASP), but whether VC is directly associated with CASP is not known. Recently, the FDA called for the inclusion of central pressures in studies of hypertension. Since ambulatory blood pressure monitoring (ABPM) may best correlate with outcomes, we aimed to determine ambulatory central blood pressure (BP) in a young South African dialysis cohort and whether coronary artery calcification (CAC) had any effect on this and the prevalence of left ventricular hypertrophy (LVH). We also sought to determine the utility of interdialytic office BP in predicting ambulatory parameters.
Methods: Seventy prevalent CKD-5D patients from one centre were studied prospectively from June 2010 to October 2011. All subjects underwent chest CT for CAC scoring (CCS), echocardiography for left ventricular mass index (LVMI) and ABPM. The ABPM was performed using the BPro® Radial Pulse Wave Acquisition Device and CASP was determined by the A-PULSE CASP® software (HealthStats) system. Conversion of BPro® data to ambulatory CASP was done by the manufacturer.
Results: Baseline characteristics by coronary calcification are presented; 27 subjects had CCS ≥ 1 (CA+) with a median CCS of 140.8 (IQR: 564.6); 43 had a CCS = 0 (CA–). Significant predictors of CAC were: age, non-African ethnicity, time on dialysis and presence of diabetes (all $p < 0.05$). Mean ambulatory systolic BP (SBP), diastolic BP, dipping, LVMI and CASP were not different between the groups. Furthermore, there was no difference in CASP after controlling for brachial systolic pressure by comparing CASP: SBP ratios and absolute differences. Interdialytic blood pressure and CASP correlated very well with ABPM ($r = 0.9$).
Conclusion: In this young South African CKD-5D cohort, VC is not associated with changes in ambulatory CASP. Age, non-black race, time on dialysis and the presence of diabetes are significant predictors of VC while interdialytic office BP and CASP are good predictors of ambulatory measurements.

OP 8: INTRA-FAMILIAL AGGREGATION AND HERITABILITY OF AORTIC PULSE PRESSURE AND PRESSURE AUGMENTATION IN A COMMUNITY WITH A HIGH PREVALENCE OF UNCONTROLLED HYPERTENSION

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Introduction: A number of studies have demonstrated significant heritability of the functional changes in large arteries that may contribute to central aortic pulse pressure (PPc). However, only a few studies have reported on the heritability of PPc *per se* and the results are conflicting.
Aim: To determine the intra-familial aggregation and heritability of central (aortic) pulse pressure and indices of pressure augmentation independent of steady-state pressure and other confounders in a community sample with a high prevalence of uncontrolled hypertension.
Methods: Central PP, central systolic blood pressure (SBPc), the forward pressure wave (P1), and augmented (AP) pressure waves and central (AIx) and radial (AIr) augmentation indices were determined with the use of applanation tonometry at the radial artery and a validated transfer function (SphygmoCor software) in 946 participants from 258 families with 23 families including three generations from an urban developing community of black Africans. Heritability estimates were determined from SAGE software. Echocardiography was evaluated in 480 participants in order to determine stroke volume.
Results: Parents (58.4%) and offspring (19.8%) had uncontrolled hypertension. Age and MAP were independently associated with all radial and aortic values ($p < 0.0001$). Heart rate was independently associated with AP, PPc, P2, AIx and AIr ($p < 005–0.0001$); and height was independently associated with AP, PPc, P2, AIx and AIr ($p < 0.01–0.0001$). However, stroke volume was not independently associated with PPc or P1 ($p > 0.05$). With adjustments for confounders; parent–child ($p < 0.05$), and sibling–sibling ($p < 0.005$ for all) correlations were noted for PPc, SBPc, P1, AP, P2, AIx and Air, while no independent correlations between fathers and mothers were observed. Independent of MAP and additional confounders, significant heritability was identified for PPc ($h^2 = 0.33 \pm 0.07$, $p < 0.0005$), SBPc ($h^2 = 0.30 \pm 0.07$, $p < 0.005$), P1 ($p < 0.0001$), AP ($p < 0.005$), AIx ($p < 0.01$), and AIr ($p < 0.01$).
Conclusion: In a community sample with a high prevalence of uncontrolled hypertension, independent of steady-state pressures and other confounders, aortic SBP and PPc as well as the component forward and augmented pressure waves and other indices of pressure augmentation were significantly inherited.

OP 9: CONTRIBUTION OF CIRCULATING ANGIOTENSINOGEN CONCENTRATIONS TO VARIATIONS IN ALDOSTERONE AND BLOOD PRESSURE IN A GROUP OF AFRICAN ANCESTRY DEPENDS ON SALT INTAKE

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Introduction: High-Na^+, low-K^+ diets in salt-sensitive individuals suppress renin release. Therefore the role of the renin–angiotensin–aldosterone system (RAAS) in salt-sensitive groups is uncertain. However, in salt-sensitive hypertension, aldosterone concentrations may not be suppressed. The mechanisms responsible for RAAS activation downstream from renin in salt-sensitive hypertension and whether these effects contribute to increases in blood pressure (BP), are uncertain.
Aims: To determine whether circulating angiotensinogen concentration ([AGT]) or its determinants may contribute to maintaining serum aldosterone concentrations ([aldosterone]) and increases in BP on high-Na^+, low-K^+ diets in a salt-sensitive community.
Methods: In 579 participants of a community sample of African ancestry, Na^+ and K^+ intake were determined from 24-hour urine samples according to pre-specified quality-control criteria. Plasma renin, and AGT and serum aldosterone concentrations were determined using immunoradiometric techniques (renin), an ^{125}I radioimmunoassay (aldosterone), and a solid-phase sandwich ELISA (human AGT). Blood pressures were determined from high-quality, nurse-derived assessments using a mercury sphygmomanometer.
Results: Plasma renin concentrations were inversely related to BP ($p < 0.0001$) and an index of salt intake (24-hour urinary Na^+/K^+, $p < 0.0001$). An interaction between [AGT] and urinary Na^+/K^+ was independently associated with [aldosterone] ($p < 0.001$) and systolic BP (SBP, $p < 0.05$). Independent of confounders, in participants with urinary $Na^+/K^+ \geq$ the median for the sample, [AGT] was positively associated with [aldosterone] ($p < 0.0001$) and SBP ($p < 0.005$). No independent [AGT]–[aldosterone] or [AGT]–SBP relationships were noted in participants with urinary $Na^+/K^+ <$ the median for the

sample. Standardised β-coefficients (slopes) of [AGT]–[aldosterone] and [AGT]–SBP relationships were greater in participants with urinary $Na^+/K^+ \geq$ the median ([AGT]–[aldosterone] = 0.30 ± 0.06, [AGT]–SBP = 0.16 ± 0.05) compared to those with urinary Na^+/K^+ < the median ([AGT]–[aldosterone] = –0.04 ± 0.06, [AGT]–SBP= –0.03 ± 0.05, $p < 0.01$–0.0001 for comparison of slopes). The [AGT]–SBP relationship in participants with urinary $Na^+/K^+ \geq$ the median for the sample was equivalent to the relationship between body mass index and BP.

Conclusions: In participants of African ancestry, in the presence of high-Na^+, low-K^+ diets, which suppress renin release, RAAS activation and its impact on BP was maintained in part by circulating angiotensinogen concentrations. These data suggest that even on a high-Na^+, low-K^+ diet in salt-sensitive populations, the RAAS contributes to increases in BP.

OP 10: THE FIVE-YEAR CARDIOMETABOLIC CHANGES IN TREATED VERSUS NEVER-TREATED HIV-INFECTED BLACK SOUTH AFRICANS: THE PURE STUDY

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Background: The prevention and treatment of hypertension is marginalised in South Africa by the overwhelming prevalence of human immunodeficiency virus (HIV) infection. HIV-1 infection and the treatment thereof paradoxically affect cardiovascular risk factors, and antiretroviral therapy is associated with an increase in dyslipidaemia, lipodystrophy, endothelial dysfunction and accelerated atherosclerosis. Whether ethnic and subtype differences may influence the latter is uncertain. We therefore aimed to compare the five-year cardiometabolic changes of black South Africans who received antiretroviral treatment to those who had never received treatment.

Methods: At baseline (2005), 300 participants were newly identified as being HIV infected. After five years, 137 of these HIV-infected participants (71 never-treated and 66 treated) were followed up. Eighty-seven participants were lost to follow up, 69 died and seven discontinued their treatment. Using standard techniques, these participants' cardiovascular, biochemical and lifestyle variables were assessed in 2005 and 2010. Blood pressure was measured with the OMRON HEM-757 device, while central systolic blood pressure (cSBP) and augmentation index (AI) were measured with the OMRON 9000AI. Pulse-wave velocity (PWV) was measured with the Complior SP device and carotid intima–media thickness (IMT) using the SonoSite Micromaxx ultrasound system. Carotid pedalis PWV, cSBP, AI and IMT were measured only at follow up.

Results: The treated group showed a higher percentage change in pulse pressure ($p = 0.013$) and systolic blood pressure ($p = 0.046$) over five years. During follow up (2010), higher levels of total cholesterol ($p < 0.001$), low-density lipoprotein cholesterol ($p < 0.001$) and triglycerides ($p = 0.043$) were observed in treated participants. The waist circumference of the treated group showed only a borderline ($p = 0.06$) increase over the five-year period, while their body mass index remained unchanged.

Conclusions: Africans receiving treatment had a greater increase in pulse pressure and a worse lipid profile when compared to never-treated participants at follow up. No vascular functional (central systolic blood pressure, carotid–dorsalis pulse-wave velocity and augmentation index) or structural (intima–media thickness) differences were observed after five years. Whether antiretroviral treatment will lead to increased arterial stiffness, vascular aging or accelerated atherosclerosis in this HIV-infected African population remains to be seen.

POSTER PRESENTATIONS

PP1: RELATIVE ROLES OF AORTIC AUGMENTATION AND FORWARD-WAVE PRESSURES ACROSS THE ADULT LIFESPAN IN A COMMUNITY WITH A HIGH PREVALENCE OF UNCONTROLLED HYPERTENSION

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Introduction: Central aortic pulse pressure (PPc) predicts cardiovascular outcomes beyond brachial blood pressure (BP). Current antihypertensive therapy reduces the augmented pressure (AP) but not the forward (P1) pressure component of PPc independent of distending pressures. Although in largely healthy, normotensive population samples, AP may play a more important role in determining age-related increases in PPc, whether similar effects are noted in communities with a high prevalence of uncontrolled hypertension is uncertain.

Aims: To determine the relative contribution of P1 and AP to increases in PPc and left ventricular mass in a community sample with poor blood pressure control.

Methods: Applanation tonometry and SphymoCor software were used to determine aortic BP in 1 015 randomly recruited participants (age range = 16–88 years) from a randomly selected community sample of black African descent, 37.7% of whom had uncontrolled BP. Left ventricular mass (LVM) and stroke volume were determined using echocardiography.

Results: Across the adult lifespan and independent of distending pressures (mean arterial pressures), for every one SD increase in age (18.4 years), PPc increased by 6.2 ± 3.0 mmHg, AP by 3.6 ± 1.7 mmHg and P1 by 2.6 ± 2.1 mmHg. The age-related increase in PPc and P1 was not associated with similar age-related increases in stroke volume. With appropriate adjustments, the relationships between P1 and PPc (partial $r = 0.90$, $p < 0.0001$) and P1 and LVM index (LVMI) (partial $r = 0.25$, $p < 0.0001$) were equally as strong as the relationships between AP and PPc (partial $r = 0.87$, $p < 0.0001$) and AP and LVMI (partial $r = 0.23$, $p < 0.0001$).

Conclusions: In a community of African ancestry with a high prevalence of uncontrolled BP, P1 contributed as much as AP to age-related increases in PPc and to variations in PPc and LVMI. These data support the development of antihypertensive therapy that targets P1 to achieve optimal decreases in PPc and target-organ effects in communities with a high prevalence of hypertension.

PP 2: VALIDITY OF RADIAL LATE SYSTOLIC SHOULDER-DERIVED AORTIC PRESSURES AND RELATIONS WITH TARGET-ORGAN CHANGES INDEPENDENT OF BRACHIAL PRESSURE

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Introduction: Central aortic blood pressure (BPc) predicts cardiovascular outcomes beyond brachial BP. In this regard, the application of a generalised transfer function (GTF) to radial pulse waves for the derivation of BPc is an easy and reproducible measurement technique. However, the use of the GTF requires expensive software. As the peak of the second shoulder of the radial waveform (P2) is closely associated with BPc, BPc may be assessed without the need for a GTF. However, compared to GTF-derived BPc, a bias in P2-derived BPc may exist.

Aims: To determine whether the bias between GTF-derived and P2-derived BPc may be mathematically corrected and whether

P2-derived BPc is as closely associated with cardiovascular damage independent of brachial BP as GTF-derived BPc.
Methods: In 1 167 participants from a community sample, BPc was assessed using applanation tonometry and SphygmoCor software. Left ventricular mass was indexed for height$^{1.7}$ (LVMI) (n = 678) and carotid intima–media thickness (IMT) (n = 462) was determined using echocardiography and vascular ultrasound.
Results: P2-derived BPc values were in close agreement with GTF-derived values, but an error, which could be mathematically corrected, was noted. Independent of brachial conventional or 24-hour pulse pressure (PP), both before (partial r = 0.18-0.40, p < 0.0001) and after (partial r = 0.13–0.41, p < 0.005) correcting for the bias in P2 versus GTF-derived aortic PP (PPc) relations, P2-derived PPc was as strongly associated with LVMI and IMT as GTF-derived PPc (partial r = 0.17–0.37, p < 0.0001), while with adjustments for BPc, brachial PP target-organ relations were eliminated (partial r = –0.21 to 0.05). Brachial BP-independent relationships between P2- versus GTF-derived PPc and target-organ changes were similar (p > 0.46).
Conclusions: The bias in P2- and GTF-derived PPc relations can be corrected using a mathematical transformation. Independent of brachial PP, both transformed and non-transformed P2-derived PPc were as closely associated with target-organ changes as GTF-derived PPc. These data suggest that inexpensive approaches to the assessment of central aortic BP may be developed.

PP 3: IS THERE AN INDEPENDENT RELATIONSHIP BETWEEN MILD (GRADE 1) HYPERTENSION AND TARGET-ORGAN CHANGES IN URBAN, DEVELOPING COMMUNITIES OF AFRICAN ANCESTRY?

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Introduction: It is well established that in developed communities of largely European ancestry, benefits are derived from BP lowering in mild (grade 1) hypertension (BP = 140–159/90–100 mmHg). However, whether similar benefits accrue from managing mild hypertension in urban, developing communities of African ancestry, a significant proportion of whom have 'white coat' effects (alerting response), is uncertain.
Aim: To evaluate the relationship between grade 1 hypertension and target-organ changes independent of alternative cardiovascular risk factors in an urban, developing community of African ancestry.
Methods: In 1 140 randomly recruited participants of African ancestry from the South West Township (SOWETO) of Gauteng, nurse-derived conventional BP, left ventricular mass indexed for height$^{1.7}$ (LVMI) (echocardiography), left ventricular early-to-atrial transmitral velocity (E/A) (echocardiography), carotid–femoral (aortic) pulse-wave velocity (PWV) (applanation tonometry), and the square root urinary microalbumin/creatinine ratio (ACR) were determined.
Results: Independent of age, waist circumference, gender, regular tobacco use, regular alcohol intake, diabetes mellitus and/or HbA_{1c} level > 6.1%, heart rate, or mean arterial pressure (in the case of PWV), compared to participants with an optimal BP (BP < 120/80 mmHg) (n = 135–280), participants with grade 1 hypertension (n = 140–211) had a similar LVMI (p = 0.52), aortic PWV (p = 0.47) and ACR (p = 0.78), but a lower E/A (p < 0.01).
Conclusions: In an urban, developing community of African ancestry, compared to an optimal BP, mild (grade 1) hypertension, as diagnosed with conventional BP measurements, was not or was only modestly and independently associated with target-organ changes. These data suggest that the role of mild hypertension in the development of cardiovascular disease in Africa requires urgent re-evaluation.

PP 4: ARE OBESITY-RELATED CONVENTIONAL CARDIOVASCULAR RISK FACTORS ASSOCIATED WITH ATHEROMA FORMATION IN URBAN, DEVELOPING COMMUNITIES OF AFRICAN DESCENT

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Background: Because of the high prevalence of obesity, it is alleged that urban, developing communities of African ancestry are in the early phase of a population health transition. Although obesity is associated with conventional cardiovascular risk factors, the extent to which obesity-related increases in conventional risk factors contribute toward atheroma-related cardiovascular disease in these communities is uncertain. Our aim was to evaluate whether in a community sample with a high prevalence of obesity, adiposity indices were associated with carotid intima–media thickness (IMT), an index of atheroma formation, and whether these relations were accounted for by conventional cardiovascular risk factors.
Methods: Carotid IMT was determined from carotid Doppler images obtained using a SonoSite, SonoCalc™ IMT 3.4 in 446 randomly recruited participants of African descent, ~63% of whom were either overweight or obese. An excess adiposity was determined from measures of waist circumference (WC), waist-to-hip ratio (WHR), body mass index (BMI) and skin-fold thickness.
Results: In bivariate analysis, all indices of adiposity were relatively strongly associated with carotid IMT [BMI (r = 0.51, p < 0.0001), WC (r = 0.54, p < 0.0001), WHR (r = 0.22, p < 0.05) and skin-fold thickness (r = 0.45, p < 0.001)]. However, age was the strongest determinant of carotid IMT in this community (r = 0.80, p < 0.0001) and although indices of obesity were associated with conventional cardiovascular risk factors, after adjustments for age alone, no index of adiposity was independently associated with carotid IMT.
Conclusion: In a community of African ancestry, independent of age, obesity was not associated with carotid IMT. These data suggest that obesity-related cardiovascular risk is presently still at an early phase in the population health transition in this community and that this may reflect a window of opportunity to successfully prevent an epidemic of cardiovascular disease by implementing community-wide weight-reduction programmes.

PP 5: INVESTIGATION OF AMBULATORY BLOOD PRESSURE PROFILE AND THE PREVALENCE OF CKD PARAMETERS IN HEALTHY HIV-POSITIVE PATIENTS, PRE AND POST HAART

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Introduction: Few studies have been done to establish the effects of HIV on the kidney in stable HIV-infected outpatients in South Africa. Our aims were (1) to document the prevalence of renal dysfunction and blood pressure at baseline in a HAART-naïve cohort and to document changes at six months; (2) to observe characteristics of ambulatory blood pressure in a subset of patients.
Methods: We conducted a longitudinal prospective study of HAART-naïve HIV-positive patients at Crossroads Community Health Centre as part of the MCHAART study group. We measured renal function parameters (microalbumin:creatinine ratio, creatinine, dipsticks and eGFR calculation) at baseline and at six months, after the initiation of HAART. A subset of patients underwent ambulatory blood pressure monitoring (ABPM). Ethics approval was obtained from the University of Cape Town ethics committee.
Results: No patient had an eGFR below 60 ml/min. Three patients had microalbuminuria and only 1% had overt albuminuria. No patient was hypertensive and there was a significant rise in office

systolic BP after six months of HAART, which was not confirmed on ABPM. The very high prevalence of non-dipping on ABPM was not improved by HAART.
Conclusion: The prevalence of CKD in HAART-naïve patients in a typical HIV outpatient clinic was much lower than previously described. This suggests that early introduction of HAART may have a major impact on the prevalence of HIVAN. The high prevalence of ambulatory non-dipping status, which is unexplained, suggests an underlying dyregulation of the cardiovascular system, and may be associated with future cardiovascular risk.

PP 6: ACUTE MODERATE-INTENSITY EXERCISE DECREASES CENTRAL PRESSURES IN MILD HYPERTENSIVES

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Background: Exercise is beneficial in the management of hypertension as post-exercise hypotension [PEH, a reduction in resting blood pressure (BP) after acute exercise] occurs. PEH has only been demonstrated using measurements of peripheral (brachial) BP; however, central (aortic) BP more accurately predicts myocardial workload and is a better prognostic marker of cardiovascular mortality and morbidity.
Methods: We determined the central and brachial BP response to acute aerobic exercise of moderate or high intensity in mild hypertensives. Thirty-nine men and women (age 30–57 years) with untreated pre-hypertension or stage 1 hypertension volunteered for the study. Twenty-two participants were randomly assigned to the moderate-intensity exercise group and 15 to the high-intensity exercise group. Brachial BP, central BP and pulse-wave analysis (radial applanation tonometry, SphygmoCor software), and carotid–femoral pulse-wave velocity (PWV, SpygmoCor) were determined at rest, 15 min and 24 hours after an acute exercise session.
Results: Compared to baseline, peripheral SBP, DBP and pulse pressure (PP), and central DBP and PP as well as PWV were no different at 15 min or 24 hours after either moderate- or high-intensity exercise ($p > 0.05$). However, decreases in central SBP (9.6 ± 9.1 mmHg; $p = 0.01$), augmentation pressure (6.5 ± 4.7 mmHg; $p = 0.0003$) and augmentation index (17.1 ± 14.5, $p < 0.0001$) occurred at 15 min after exercise in the moderate-intensity group. The central SBP remained decreased 24 hours after exercise (8.9 ± 13.0 mmHg; $p = 0.03$) but central augmentation pressure and augmentation index were no different from baseline. In the high-intensity exercise group there was no significant decrease in central SBP (6.0 ± 8.4.0 mmHg; $p = 0.44$) at 15 min after exercise. Central augmentation pressure (5.1 ± 3.3 mmHg, $p = 0.04$) and augmentation index (12.1 ± 10.3, $p= 0.04$) were reduced at 15 min after exercise, but returned to near baseline at 24 hours after exercise.
Conclusion: The results show that acute moderate-intensity exercise produced decreases in central systolic pressures that were not observed peripherally or with high-intensity interval exercise. The change in central SBP, which was sustained 24 hours after moderate-intensity exercise, is likely to be attributed to a change in the properties of medium-sized arteries, and not to an acute change in large-artery functioning.

PP 7: DIETARY INTERVENTION IN BLACK SUBJECTS WITH MILD-TO-MODERATE HYPERTENSION DOES NOT ALTER NOCTURNAL DIPPING STATUS

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Background: Non-dipping status has been associated with a poorer prognosis in patients with essential hypertension. The effect of dietary intervention on nocturnal dipping has not been studied in patients with essential hypertension. The aim of the study was to determine the effect of a dietary intervention of Na^+ restriction and increased K^+, Mg^{++} and Ca^{++} on nocturnal dipping.
Methods: Black subjects aged 50 to 75 years with drug-treated mild-to-moderate hypertension were eligible for the study. Non-dipping was defined in a < 10% fall in nocturnal BP. Subjects were randomised to an eight-week dietary intervention ($n = 40$) or standard diet ($n = 40$). Twenty-four-hour ambulatory BP was measured at baseline and eight weeks. Data were analysed using generalised linear models.
Results: Baseline characteristics were similar between the groups. Overall, 43.8 and 17.5% of patients were non-dippers for systolic and diastolic BP, respectively. Between-diet difference in change in office systolic BP was –6.19 mmHg ($p = 0.021$) and mean 24-hour ambulatory systolic BP between-diet change was –4.53 mmHg ($p = 0.05$). There was no difference in dipping status between the groups at the end of eight weeks' intervention.
Conclusion: Dietary intervention improved BP in black subjects with mild-to-moderate hypertension, but did not influence dipping status.

PP 8: GENDER-SPECIFIC EFFECTS OF ADRENERGIC-INDUCED ADVERSE CARDIAC REMODELLING IN SPONTANEOUSLY HYPERTENSIVE RATS

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Introduction: In response to a pressure overload, females develop more marked concentric cardiac hypertrophy, and systolic function remains higher than in males. However the mechanisms that explain these gender-related differences are uncertain. Whether female gender protects against adrenergic-induced adverse cardiac remodelling (cardiac dilatation), a mechanism thought to be in part responsible for the transition from concentric cardiac hypertrophy to cardiac dilatation in hypertension, is uncertain.
Aim: To determine whether gender influences the ability of chronic β-adrenergic receptor stimulation to promote the progression from left ventricular hypertrophy to cardiac dilatation in spontaneously hypertensive rats (SHR).
Methods: Chronic β-adrenergic receptor activation was induced by daily injections of isoproterenol (ISO) (0.04 mg/kg/day) for five months, starting at nine months of age in male and female SHR. At the end of the study, left ventricular (LV) chamber dimensions were determined *in vivo* by echocardiography and LV remodelling was assessed *ex vivo* in a load- and heart rate-independent manner in isolated, perfused, paced heart preparations by assessing LV diastolic pressure–volume relations.
Results: LV end-diastolic diameters (LVEDD), as well as the volume intercept (V_0) of the LV diastolic pressure–volume relationship were increased in male, but not female SHR.
Conclusion: Male rats were more susceptible than females to the ability of chronic adrenergic stimulation to promote the progression from left ventricular hypertrophy to cardiac dilatation in hypertension.

PP 9: A SIX-MONTH STUDY EVALUATING THE COMPLIANCE OF PATIENTS TREATED WITH A FIXED-DOSE COMBINATION ANTI-HYPERTENSIVE THERAPY, EXFORGE (AMLODIPINE/VALSARTAN) IN SOUTH AFRICA (THE EXCEED SOUTH AFRICAN SURVEY)

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Background: Compliance is of paramount importance in blood pressure control, particularly in patients at high cardiovascular risk. Single-pill combination therapy simplifies therapy and so

increases compliance. Exforge® (amlodipine/valsartan) is a fixed-dose combination-pill combining two antihypertensive compounds with complementary mechanisms of action. The objective of this study was to determine compliance rates in South African patients treated with amlodipine/valsartan and to evaluate compliance in different cardiovascular risk categories.

Methods: A multicentre observational study was conducted on 366 patients with essential hypertension prescribed amlodipine/valsartan for ≤ one month, three visits were scheduled: visit 1 – enrolment; visit 2 – month 2; visit 3 – month 6. Patients were stratified according to cardiovascular risk as per the ESH/ESC guidelines. Patients were either assigned to category 1 (average, low and moderate risk) (n = 246, 67.2%), or category 2 (high and very high risk) (n = 118, 32.2%) at visit 1. Demographics and data on antihypertensives and concomitant medication were collected. Compliance was evaluated as very good ≥ 80% (missed ≤ six tablets per month) or poor < 80% (missed > six tablets per month). Compliance was assessed in patients whose data were complete.

Results: A total of 366 patients were enrolled (51.6% males; 48.4% females), with 340 patients at visit 2 and 306 at visit 3.

At visit 2, 318 patients (94.9%) demonstrated very good compliance overall (95% CI: 92.6–97.3). In risk category 1, 210 patients (94.6%) demonstrated very good compliance (95% CI: 91.6–97.6). In risk category 2, 106 patients (95.5%) demonstrated very good compliance (95% CI: 91.6–99.4).

At visit 3, 281 patients (93.4%) demonstrated very good compliance overall (95% CI: 90.5–96.2). In risk category 1, 182 patients (95.3%) demonstrated very good compliance (95% CI: 92.3–98.3). In risk category 2, 99 patients (90%) demonstrated very good compliance (95% CI: 84.4–95.6).

Adverse events were responsible for discontinuation from the study in 7.1% patients. The most commonly observed adverse event was peripheral oedema in four (1.1%) patients, followed by insomnia in three (0.8%) patients.

Conclusions: Patients on single-pill combination therapy with amlodipine/valsartan demonstrated very good compliance regardless of cardiovascular risk. Amlodipine/valsartan was well tolerated.

PP10: NON-LINEAR MODELLING OF CATIONIC AMINO ACID UPTAKE INTO HUVEC AND ECV_{304} CELLS ALLOWS DISTINCTION BETWEEN TRANSPORTERS

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Arginine and cationic amino acid uptake into endothelial cells is mediated by the high-affinity/low-rate y^+L transporter and the low-affinity/high-rate y^+ transporter. Uptake in the absence/presence of leucine and sodium has been used to distinguish transport by subtracting arginine transport in the presence of the leucine-/sodium-free medium from the total uptake, respectively. This assumes uptake by the two transporters is additive at all concentrations of leucine, sodium and arginine. Methods are still needed to selectively determine the role of each transporter in arginine uptake and we compared results using a non-linear model of uptake with those reported in the literature.

Methods: Details of determining the kinetic constants of arginine uptake using the initial rate of uptake have been described previously. When arginine uptake was measured in the presence of inhibitors (NEM, BCH, leucine and removal of sodium), the type of inhibition was determined from the changes in V_{max} and/or $K_{m'}$, using the appropriate equation for each of the transporters.

Results and Discussion: Non-linear modelling suggested that leucine affected both transporters differently without either transporter being completely inhibited at any of the concentrations of leucine tested. Leucine affected y^+ transport competitively whereas inhibition of y^+L transport was complex (mixed model of inhibition). N-ethylmaleimide, as reported in the literature, completely inhibited y^+ transport and data suggested BCH may be a selective inhibitor of y^+L transport. The absence of sodium reduced arginine uptake by y^+L transport and reduced the affinity ($K_{m'}$), whereas reducing sodium decreased uptake by y^+ transport without affecting the $K_{m'}$.

Conclusions: Non-linear modelling allowed the kinetic constants of uptake in the presence of inhibitors and modifiers, allowing the type of inhibition to be determined. The results obtained mostly differed from those reported in the literature. Neither leucine nor cationic amino acid uptake in the absence of sodium can be used to distinguish between the two transporters, as has been extensively reported in the literature. The sulphydryl reagent NEM, inhibits y^+ transport and BCH, or structurally related analogues may be a selective inhibitor of y^+L transport.

PP 11: MODELLING OF CELLULAR ARGININE UPTAKE BY MORE THAN ONE TRANSPORTER

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Background: Cardiovascular disease remains a leading cause of death in all age groups in South Africa. Supplementation with arginine, the precursor of vasodilator nitric oxide (NO) has been shown to be beneficial in heart failure and other cardiovascular diseases. As arginine plasma concentrations are elevated in hypertension, it may be that membrane transport is impaired or NO availability is limited. Most studies determining the uptake of radiolabelled amino acids have calculated the kinetic constants from linearisation of dilution-corrected uptake counts using Eadie–Hofstee plots. We previously reported uptake of arginine by ECV_{304} and HUVEC cells using such methods. Such approaches may not be correct and other methods are needed to determine uptake using more than one transporter.

Methods: The initial rate of uptake of trace [3H]L-arginine by arginine-depleted HUVECs and ECV304 cells, in the presence of a range of unlabelled arginine was determined. The initial rate of uptake of the radio arginine was expressed as nM/4 × 10^5 cells/min. The kinetic constants of uptake were determined using the expression describing arginine uptake by two additive transporters using Michaelis–Menten kinetics with a diffusion component. The kinetic constants were determined by adjusting the constants to best-fit experimental data using non-linear regression analysis (GraphPad Prism® Version 5, GraphPad Software Inc, La Jolla, California, USA) with appropriate curve-fit and normality checks as described previously.

Results: The linearity of uptake with time and concentration of arginine was established, and it demonstrated that uptake of the tracer arginine in the presence of unlabelled arginine was not linear. Theoretical plots of uptake derived from constants determined from Eadie–Hofstee graphs overestimated uptake, whereas those from the non-linear modelling approach agreed with experimental data. Furthermore the contribution of uptake by individual transporters could be modelled.

Conclusion: The non-linear modelling approach using raw data avoided the errors inherent in methods deriving constants from the linearisation of the uptake processes following Michaelian kinetics. This study provides explanations for discrepancies in the literature and suggests that a non-linear modelling approach better characterises the kinetics of amino acid uptake into cells by more than one transporter.

PP 12: RESERPINE+AMILOZIDE STILL THE FIRST-LINE DRUG THERAPY OF COMMON HYPERTENSION

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Background: Mild-to-moderate hypertension (MHBP) (the usually associated insulin-resistance syndrome–adiposity lipidaemia) is the commonest chronic systemic disease of first-world adults, especially

in Africa. The only rational approach addresses all risk factors: diet, lifestyle, exercise, supplements (insulin sensitisers, antioxidants, nitric oxide, including fish oil, vitamins, minerals, metformin, appropriate hormones), and if high BP persists, low-dose daily reserpine, e.g. 0.0625 mg plus low-dose amilozide 13.75 mg/d (hydrochlorothiazide HCTZ + amiloride), even just three days a week. These old, proven, safe, effective drugs with flat dose–response curves do not have the common risks of more modern fashionable drugs, the angiotensin/alpha/beta-blockers.

Methods: Literature review.

Results: The pharmaceutical industry has never published a trial comparing the above old triple-drug regime with modern antihypertensives. At least 23 published trials (1977–2002) show that thiazide (10 trials) or reserpine (nine trials) was as good as or better than a single modern drug (tabulated at http://healthspanlife.files.wordpress.com/2009/04/reserpine_table.pdf).

In three trials (1981–1986), amilozide was better for high BP control than either component alone. Henry Black from New York (2011) observed that newer drug combinations add to cost. In USA, the recent average drug use per patient is two: ACEI > CCB > diuretic > ARB > centrally acting/ vasodilator/alpha-agonists. Reserpine (half-life two to seven days) is a very effective part of a two- or three-drug regime. Egan (Univ. Carolina) (August 2011) states that apparent treatment-resistant hypertension in USA increased from 16% in 1998 to 28% in 2008 of treated patients (therapeutic inertia). Is this any less in RSA?

Experience with reserpine–amilozide is that most MHBP patients have well-controlled BP, heart, circulation and metabolism. Few require the addition of amlodipine to reduce high BP below about 135/80 mmHg. A review of patient follow up will be presented.

Conclusions: Unlike alpha/beta-blockers or ACEI/ARB, low-dose reserpine + amilozide has no adverse effects on metabolism, circulation and brain function. It slows the heart rate and rarely causes wheeze, cough, rash or dizziness. Authorities must quote evidence to justify HCTZ or furosemide and other antihypertensives with their increased risks in the first-line drug therapy of hypertension, over low-dose reserpine–amilozide, costing ~US$1 a month retail in South Africa.

PP 13: NT-proBNP IS ASSOCIATED WITH FIBULIN-1 IN AFRICANS: THE SAFREIC STUDY

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Background: The N-terminal pro-hormone B-type natriuretic peptide (NT-proBNP) is a cardiac hormone involved in the regulation of cardiac volume overload. Fibulin-1 on the other hand is a component of many extracellular matrix proteins, including those present in atherosclerotic lesions and those expressed in blood vessel walls by elastin-containing fibres. This study investigated the associations of NT-proBNP with fibulin-1 and markers of arterial function in African and Caucasian men and women.

Methods: We included 177 Africans and 215 Caucasians of similar age (35.6 ± 9.2 years) from South Africa. Serum NT-proBNP and fibulin-1 levels were determined with immunoassays. Arterial compliance and pulse-wave velocity were also measured.

Results: Africans had higher blood pressure and fibulin-1 levels than Caucasians, and African men had higher NT-proBNP levels than Caucasian men. In single-regression analysis, NT-proBNP was significantly associated with fibulin-1 in all groups, with African men showing borderline significance ($r = 0.19$; $p = 0.070$). NT-proBNP correlated negatively with arterial compliance only in Africans, but significance was lost after adjustment for confounders. Previous associations of NT-proBNP with arterial compliance disappeared in all groups. After full adjustment for confounders, the association of NT-proBNP with fibulin-1 was confirmed only in African men ($R^2 = 0.361$; $\beta = 0.394$; $p < 0.001$) and African women ($R^2 = 0.366$; $\beta = 0.220$; $p < 0.05$).

Conclusions: Only Africans indicated a significant independent association between NT-proBNP and fibulin-1, suggesting that cardiovascular alterations were already present in this relatively young African population. Africans may also be at higher risk to develop future cardiovascular damage as opposed to Caucasians.

PP 14: CAROTID CROSS-SECTIONAL WALL AREA IS SIGNIFICANTLY ASSOCIATED WITH LEPTIN LEVELS, INDEPENDENT OF BODY MASS INDEX: THE SABPA STUDY

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Background: Hypertension and obesity are serious health burdens in sub-Saharan Africa and urbanised Africans seem to be more susceptible to the development of these diseases than Caucasians. Current research suggests that leptin may be an important contributor to the development of hypertension and atherosclerosis. The aim of this study was to investigate leptin levels and associations with cardiovascular function in urbanised Africans ($n = 199$) and Caucasians ($n = 208$).

Methods: Serum leptin, ambulatory blood pressure and carotid intima–media thickness were measured, and the cross-sectional wall area calculated.

Results: Africans had higher leptin levels ($p < 0.001$), ambulatory blood pressure ($p < 0.001$), carotid intima–media thickness ($p < 0.01$) and cross-sectional wall area ($p < 0.01$) than Caucasians. Because we found no interaction with ethnicity and gender for the association between leptin and the cardiovascular variables, we focused mainly on the total group of Africans and Caucasians. In single, partial and multiple regression analyses, positive associations between ambulatory systolic blood pressure ($\beta = 0.261$; $p < 0.001$), diastolic blood pressure ($\beta = 0.144$; $p = 0.012$), pulse pressure ($\beta = 0.335$; $p < 0.001$) and cross-sectional wall area ($\beta = 0.121$; $p = 0.019$) with leptin were observed. Even after adjusting for body mass index, the association obtained between cross-sectional wall area ($\beta = 0.121$; $p = 0.019$) and leptin remained.

Conclusions: Our findings therefore suggest that leptin may contribute to the development of atherosclerosis, independent of body mass index.

PP 15: DETERMINANTS OF CIRCULATING ANGIOTENSINOGEN CONCENTRATIONS IN AN URBAN, DEVELOPING COMMUNITY OF AFRICAN ANCESTRY

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Introduction: We have recently demonstrated the importance of circulating angiotensinogen concentrations ([AGT]) as a determinant of variations in serum aldosterone concentrations and blood pressure (BP) in salt-sensitive populations of African ancestry when renin release is suppressed. However, the factors that influence circulating [AGT] in these populations are uncertain.

Aims: To determine the extent to which phenotypic and genetic factors determine variations in circulating AGT concentrations in an urban, developing community of African ancestry.

Methods: In 579 participants of a community sample of African ancestry plasma, [AGT] was determined using a solid-phase sandwich ELISA. C-reactive protein (CRP) concentrations were measured using an ultrasensitive immunoturbidimetric assay and the A→C transition at nucleotide –20 and the G→A substitution at position –217 of the 5′ upstream promoter region of the AGT gene was undertaken using PCR-restriction fragment-length polymorphism-

based techniques using the appropriate primer pairs and restriction enzymes.
Results: In multivariate analysis, age, gender, body mass index, diabetes mellitus or an HbA_{1c} level > 6.1%, regular tobacco use, regular alcohol use, treatment for hypertension, the A→C transition at nucleotide –20 of the AGT gene (partial $r = 0.13$, CI = 0.05–0.21, $p < 0.005$) and log CRP concentrations (partial $r = 0.13$, CI = 0.05–0.21, $p < 0.005$), but not the G→A substitution at position –217 of the AGT gene ($p = 0.28$), or other demographic or phenotypic factors were independently related to circulating [AGT].
Conclusions: In an urban, developing community of African ancestry, the AGT gene and inflammatory changes may be important determinants of variations in circulating [AGT] and hence in BP control.

PP 16: PILOT STUDY: TWO RESPONSE GROUPS AMONG YOUNG PRE-HYPERTENSION CASES TO AUTOHYPNOSIS

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Background: If psychological stress is a precursor to hypertension and pre-hypertension, then any relaxation techniques such as hypnosis and meditation must reduce the stress and blood pressure levels. There are no reports in the literature on the effect of autohypnosis on pre-hypertension in young patients.
Methods: After learning the technique, 12 young patients performed a daily 10-min autohypnosis session for about three months. They visited the centre every weekend for 13 weeks.
Results: Blood pressure measurements in three of the 12 patients showed a dramatic response to the treatment and returned to normal levels after one month, but the rest of the group did not show this response. Their changes in blood pressure levels were non-significant.
Conclusions: During this pilot study for assessment of the effect of autohypnosis on pre-hypertension, we found that there were two types of response among our young patients: a normal response and a dramatic response. The latter group we named the autohypnosis-sensitive group. We suggest a larger study is needed to reveal whether these early findings are representative or not. If daily short autohypnosis works as a treatment for hypertension in a sensitive group of patients, it has capacity to reduce the cost and side effects of drug therapy in pre-hypertension patients.

PP 17: HYPERTENSION IN WOMEN OF CHILDBEARING AGE

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The incidence of hypertension is likely to increase in young women due to the prevalence of the metabolic syndrome, dyslipidaemia and obesity. It is very likely that pregnant women on blood pressure-lowering agents will develop eclampsia and pre-eclampsia and that the drugs may well be teratogenic, with deleterious results on the offspring.

Pre-eclampsia is a specific problem that may affect the cardiovascular system and pre-conception requires specific counselling skills. Doctors need to have an in-depth knowledge of the physiological changes that occur during pregnancy and also the side and adverse effects of the medication used to combat hypertension. Regular blood pressure monitoring is required and management by health professionals experienced in this field will minimise sequelae associated with disorders in pregnancy. This may have a positive impact on women's cardiovascular events and outcome years after the affected pregnancies.

PP 18: LIRAGLUTIDE, A GLP-1 ANALOGUE AND CARDIOVASCULAR RISK FACTOR

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Background: Cardiovascular disease (CVD) is the major cause of mortality in type 2 diabetes. Hyperglycaemia and diabetes co-exist with other well-established CVD risk factors. Data from the LEAD TM programme were analysed to evaluate the effect of liraglutide, a glucagon-like peptide (GLP-1) analogue and other antihyperglycaemic agents on CVD risk factors.
Methods: The LEAD TM programme comprised six phase 3 clinical trials involving over 4 000 type 2 diabetes subjects treated with liraglutide and various other antihyperglycaemic agents, either as monotherapy or combination therapy.
Results: Subjects randomised to liraglutide showed a significant improvement in glycaemic control in all six trials. Control was superior compared to glimepiride, rosiglitazone, glargine and exenatide. Significant weight reduction was seen in the liraglutide and exenatide groups, but not in the other treatment arms, in some of which there was weight gain, namely those on insulin glargine, glimepiride and rosiglitazone. Blood pressure decreased significantly with liraglutide compared to glimepiride, combined therapy with metformin and glimepiride, and insulin glargine. Liraglutide showed a significant reduction in total cholesterol and LDL cholesterol compared to rosiglitazone, insulin glargine, glimiperide and placebo. Emergent CVD risk factors such as BNP, hsCRP and PAI-1 were significantly reduced compared to baseline in the liraglutide group. In addition, rates of hypoglycaemia, which predisposes to CVD events, were much lower with liraglutide than with glimepiride.
Conclusion: Data from the LEAD TM programme have shown significant improvement in CVD risk factors with liraglutide. The question as to whether these beneficial effects on surrogate markers will result in a reduction in CVD mortality and morbidity remains unanswered at present. Several long-term studies are being conducted to answer this question.

Notes:

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