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The clinical consequences and challenges of hypertension in urban-dwelling black Africans: Insights from the Heart of Soweto Study

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ABSTRACT

Background: There is a paucity of data to describe advanced forms of cardiovascular disease (CVD) in urban black Africans with hypertension (HT).
Methods: Chris Hani Baragwanath Hospital services the black African community of 1.1 million people in Soweto, South Africa. We prospectively collected detailed demographic and clinical data from all *de novo* presentations to the hospital's Cardiology Unit in 2006.
Results: Overall, 761 black African patients (56% of *de novo* cases) presented with a diagnosis of HT with more women (63%, aged 58.5 ± 14.9 years) than men (aged 58.0 ± 15.6 years). On presentation, 396 women (82%) versus 187 men (67%) had dizziness, palpitations and/or chest pain (OR 1.23, 95% 1.12–1.34: $p<0.0001$). HT was the primary diagnosis in 266 cases (35%). In the rest ($n=495$), non-ischaemic forms of heart failure were common (54% of total) while only 6.2% had coronary artery disease. Concurrent left ventricular hypertrophy, renal dysfunction and anaemia were present in 39%, 24% and 11% of cases, respectively, with a similar age-adjusted pattern of co-morbidity according to sex. However, men were more likely to present with impaired systolic function (OR 2.13, 95% CI 1.50 to 3.00; $p<0.0001$).
Conclusions: In the absence of effective primary and secondary prevention strategies, these unique data highlight the potentially devastating impact of advanced forms of hypertensive heart disease in urban black African communities with more women than men affected.
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1. Background

A key contributor to a global epidemic of cardiovascular disease (CVD), high blood pressure (BP) otherwise known as hypertension (HT), is a readily detectable and modifiable target for primary and secondary prevention. In 2001, HT was estimated to contribute to 7.6 million premature deaths (13.5% of total) and 92 million disability-adjusted life years on a global basis [1]. In low-to-middle income countries, HT has particular relevance to the phenomenon of epidemiological transition [2]. Due to altered socio-economic conditions in many vulnerable communities globally, a transition from traditional to more affluent lifestyles leads to a subsequent rise in the prevalence of HT and metabolic disorders. This in turn is likely to fuel a parallel increase in non-communicable forms of CVD [2]. In sub-Saharan African, CVD-related morbidity and mortality rates are already increasing, with one third of deaths in those aged >65 years already attributable to CVD [3,4]. The likely contribution of HT is underlined by a systematic review of 25 studies (>400 subjects in each) from 10 sub-Saharan countries during 1987–2004 showing a reported prevalence of HT ranging from 13% to 48% and rural versus urban gradients (more HT in the latter) linked to epidemiologic transition [5]. Consistent with these data, we recently found that 33% of more than 1600 black Africans screened in the urban townships comprising Soweto, South Africa, were hypertensive [6].

Despite a wealth of data to describe the pattern of HT in low-to-middle income countries, there is a paucity of high quality clinical and diagnostic data to describe its clinical consequences. Given the size of the populations at risk, understanding the consequences of largely undetected and untreated HT in vulnerable individuals living in urban communities in Sub-Saharan Africa is critical to efforts to minimise the emergence of non-communicable forms of CVD; both from a primary and secondary prevention perspective.

Abbreviations: CVD, cardiovascular disease; HT, hypertension; BP, blood pressure (SBP — systolic and DBP — diastolic); BMI, body mass index; NYHA, New York Heart Association; ECG, electrocardiograph; HF, heart failure; LVH, left ventricular hypertrophy; LVEF, left ventricular ejection fraction; eGFR, estimated glomerular filtration rate.

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2. Study aims

As part of the Heart of Soweto Study, [7] we recently reported the clinical spectrum of CVD and its risk factors in a large series of adults attending a tertiary referral hospital in Soweto during 2006 [8]. Significantly, HT was the most common diagnosis by far. In black Africans patients, HT was diagnosed in 52% (2923 of 4162) of cases and was often associated with advanced forms of heart disease [9]. We postulated that a more detailed analysis of HT-related heart disease would identify important primary prevention and treatment targets to limit the expected rise of non-communicable forms of heart disease in urban communities in Sub-Saharan Africa.

3. Methods

3.1. Participants

We focused on black African patients who presented to the Cardiology Unit of the Chris Hani Baragwanath Hospital in Soweto for the first time for advanced diagnostic assessment and treatment during 2006 and were subsequently diagnosed with HT. Black Africans represented 85% (761 of 897 presentations) of *de novo* cases of HT [8]. The case presentation in this patient group can be categorized in the following ways:

- Emergency presentation direct to the Cardiology Unit with established CVD (11% of total case-load).
- External referral from local primary care clinics for advanced assessment and definitive treatment (14%).
- Internal referral of a patient as a current hospital inpatient (28%).
- Referral from another outpatient department (e.g., diabetic clinic — 47%).

The spectrum of patients described in this report, therefore, ranged from those with uncomplicated HT controlled with mono or combination antihypertensive therapy to newly diagnosed patients with advanced disease yet to be prescribed definitive treatment.

3.2. Clinical setting

The Heart of Soweto Study is one of Africa's largest and most detailed studies of emergent heart disease and its antecedents in the geographically compact townships that comprise Soweto (population 1.1 million) within the broader conurbation of Johannesburg, South Africa. [7] As with many other urban regions within Sub-Saharan Africa, [10] Soweto is in epidemiologic transition due to a rapidly changing socio-economic environment.

The unit is staffed by internal medicine specialists in cardiology training and supported by experienced cardiologists. Applying gold-standard cardiologic expertise and advanced diagnostic technical capacity (e.g., coronary angiography and nuclear imaging) it provides definitive health care to the local population.

3.3. Ethics

Ethical approval for the study was sought from the local Ethical Committee and permission confirmed through the relevant administrative bodies. The study conformed to the principles outlined in the Declaration of Helsinki.

3.4. Study design

Study methods for the Heart of Soweto Clinical Registry have been reported in greater detail previously [8,9]. Key data collected as part of comprehensive clinical and demographic profiling of the 761 black African patients included in this report include:

- A 12-lead electrocardiogram (ECG) performed by a trained ECG technician with subsequent independent coding according to Minnesota criteria [11]. ECG data were available in 671 (88%) of patients.
- Echocardiographic assessment with detailed assessment of ventricular function, valvular integrity and function and regional wall abnormalities (specific measurements were available in 707 cases [93%] and complete data in 676 [89%] of cases) using a previously described, gold-standard protocol [12].

Other data were available according to the clinical profile of the patient at the time of data collection. For example, haemoglobin, creatinine and fasting cholesterol levels were available in 632 (83%), 637 (84%) and 339 (45%) of cases, respectively. Complete BMI data were available for 361 (49%) of the cohort overall. Each patient in the study was assigned a unique identifying code and all documents labelled accordingly to maintain anonymity.

3.5. Data analyses

A diagnosis of HT was based on the Joint National Committee on Prevention, Detection, Evaluation and Treatment's Report [13] via a documented BP ≥ 140/90 mmHg and/or prescribed antihypertensive treatment. BP was assessed following 5 min rest; seated systolic (SBP) and diastolic (DBP) BP (in mmHg) and heart rate (beats per minute) measurements were performed using an appropriately sized arm cuff via a calibrated Dynamap (Critikon, Johannesburg, South Africa) monitor. The mean of three successive readings each separated by two minutes rest was taken. As reported previously [8,9], hypertensive heart failure (HF) was diagnosed on the basis of a documented BP of >180/100 mmHg and symptoms of HF (dyspnoea and tachycardia), increased left ventricular septal thickness (>13 mm) indicative of left ventricular hypertrophy (LVH), diastolic dysfunction and/or systolic dysfunction (left ventricular ejection fraction [LVEF] ≤ 45%) by echocardiography [14]. As such, these criteria are both consistent with our continued classification of cases within the Heart of Soweto cohort and with those applied by the Euro Heart Survey investigators [14]. Renal function was assessed with estimated glomerular filtration rate (eGFR) calculated via the Modification of Diet in Renal Disease abbreviated formula (eGFR mL/min/1.73 m^2 = 186.3 × (serum creatinine mg/dL)$^{-1.154}$ × (age)$^{-1.154}$ × (0.742 female sex) × (1.21 for black Africans)) using serum creatinine concentrations (μmol/L) converted to mg/dL. Mild renal dysfunction was defined as an eGFR of 60 to 90 mL/min/1.73 m^2 and moderate to severe renal impairment <60 mL/min/1.73 m^2.

3.6. Data and statistical analyses

All study data were entered into a dedicated data-base (Microsoft Access) by an experienced cardiac nurse, verified and then transferred to SAS version 9.1 (SAS Institute Inc., Cary, NC, USA) for all analyses. Normally distributed continuous data are presented as the mean ± standard deviation and non-Gaussian distributed variables as the median plus interquartile range (IQR). Categorical data are presented as percentages with 95% confidence intervals (CI) shown where appropriate. To compare patient groups according to demographic and clinical profile, we used Chi Square (χ^2) analysis with calculation of odds ratios (OR) and 95% CI (where appropriate) for discrete variables, and for normally distributed continuous variables we used Student's *t*-test and analysis of variance. Multiple logistic regression analyses (entry model) were performed on age, sex and baseline risk factors to derive adjusted ORs for the risk of presenting with various forms of advanced heart disease and/or renal dysfunction. Significance was accepted at the two-sided level of 0.05.

4. Results

4.1. Clinical and demographic profile

Table 1 summarises the clinical and demographic profile of this cohort. There were more women (482 cases — 63%) who were of a similar age as men (mean age of 58.5 ± 14.9 years vs. 58.0 ± 15.6 years, respectively: $p = 0.63$). Overall, 168 patients (22%) reported a family history of CVD, with proportionately more women reporting this risk factor than men (OR 1.63, 95% CI 1.20–2.23: $p = 0.001$). Women were also more likely to be obese (OR 2.66, 95% CI 1.83–3.86: $p < 0.001$). A total of 575 patients (76%) had multiple, modifiable risk factors, with men more likely to smoke (OR 4.72, 95% CI 3.44–6.45: $p < 0.0001$) and have a risk factor other than HT (OR 1.86, 95% CI 1.29–2.69: $p = 0.001$).

HT was the primary diagnosis in only 266 patients (35%) with the majority of patients presenting with advanced forms of CVD — (see Table 2). Many patients presented with symptoms indicative of underlying heart disease with 524 patients (69%) reporting dyspnoea equivalent to NYHA Class II–IV, 446 (59%) dizziness, 420 (55%) palpitations and 226 (30%) chest pain. The symptomatic profile of women was markedly worse than men. Overall, 396 women (82%) compared to 187 men (67%) reported a combination of dizziness, palpitations and chest pain (OR 1.23, 95% 1.12–1.34: $p < 0.0001$).

4.2. Blood pressure profile

The BP profile of this group of patients varied markedly according to stage of presentation and treatment status. Overall, 350 (46% for both sexes) patients were hypertensive (BP ≥ 140/90 mmHg) on presentation to the unit: 20% and 25% of both sexes, respectively, presenting with a BP indicative of Stage 2 HT (SBP < 160 mmHg and/or DBP < 100 mmHg) or pre-hypertension (SBP 120–139 mmHg). [13] There was a weak, positive relationship between age and SBP with a slightly stronger association in men (R^2 0.24, $p < 0.001$) than women (R^2 0.15, $p = 0.042$). Fig. (1) shows the distribution of SBP and DBP according the presence and absence of advanced CVD (see also Table 2). Patients without advanced forms of CVD presented with a

Table 1
Clinical and demographic characteristics of study cohort.

	All $n=761$	Men $n=279$	Women $n=482$	p value
Socio-demographic profile				
Mean age (years) ± SD	58.0 ± 15.2	58.0 ± 15.6	58.5 ± 14.9	[0.632]
No/<5 years education	248 (33%)	84 (30%)	164 (34%)	[0.246]
Live in Soweto	481 (63%)	173 (62%)	308 (64%)	[0.671]
Mean years living in Soweto	43 ± 16	40 ± 16	45 ± 17	0.043
Risk factor profile				
Family history of CVD	168 (22%)	44 (16%)	124 (26%)	0.001
History of smoking	291 (38%)	170 (61%)	121 (25%)	<0.0001
Dyslipidemia*	81 (24%)	29 (20%)	52 (27%)	[0.119]
Low:high density lipid (mmol/L)	2.4 ± 1.00 : 1.2 ± 0.5	2.5 ± 1.00 : 1.2 ± 0.5	2.4 ± 1.00 : 1.2 ± 0.6	[0.269 : 0.762]
Total serum cholesterol (mmol/L)	4.2 ± 1.2	4.1 ± 1.2	4.3 ± 1.3	[0.265]
Obese (BMI>30 kg/m^2) *	162 (46%)	29 (25%)	133 (57%)	<0.001
BMI kg/m^2*	29.8 ± 6.8	26.3 ± 6.3	31.6 ± 8.1	<0.001
Multiple risk factors	575 (76%)	230 (82%)	345 (72%)	<0.001
Clinical presentation				
NYHA class II:III/IV	309 (41%):215 (28%)	98 (35%):60 (22%)	211 (44%):155 (39%)	<0.001
Mean heart rate/minute	86 ± 19	84 ± 18	87 ± 20	0.032
Mean Systolic BP (mmHg)	137 ± 28.1	135 ± 28.4	139 ± 27.9	[0.148]
Mean diastolic BP (mmHg)	77 ± 17.1	78 ± 18.5	77 ± 16.4	[0.347]
Dizziness	446 (59%)	128 (46%)	318 (66%)	<0.001
Palpitations	420 (55%)	126 (45%)	294 (61%)	<0.001
Angina pectoris/chest pain	226 (30%)	67 (24%)	159 (33%)	0.014
Edema (pulmonary/peripheral)	250 (33%)	81 (29%)	169 (35%)	[0.261]
Estimated GFR (ml/min/1.73 m^2)*	83 ± 34.4	83 ± 39.5	80 ± 31.8	[0.214]
Median glucose mmol/L*	6.2 (5.1–8.1)	6.1 (5.1–7.7)	6.3 (5.2–8.4)	[0.239]
Co-morbidity				
HT as primary diagnosis	266 (35%)	87 (31%)	179 (37%)	NS [0.097]
Heart failure	410 (54%)	166 (59%)	244 (51%)	0.018
Structural valve disease	58 (7.6%)	19 (6.8%)	39 (8.1%)	NS [0.773]
Coronary artery disease	47 (6.2%)	22 (7.9%)	25 (5.2%)	NS [0.136]
Stroke	25 (3.3%)	8 (2.9%)	17 (3.5%)	NS [0.623]
Renal disease	69 (9.1%)	30 (11%)	39 (8.1%)	0.001
Anemia*	63 (11%)	17 (7.2%)	46 (12%)	NS [0.069]
Diabetes	101 (12%)	32 (12%)	69 (14%)	NS [0.265]
HIV positive¶	18 (2.4%)	5 (1.8%)	13 (2.7%)	NS [0.429]

Legend: * denotes clinical data were not available on all patients (see Methods). ¶ HIV status data are only available in the limited numbers of patients who consented to voluntary testing.

significantly lower SBP (135 ± 27 vs. 144 ± 28 mmHg; $p<0.0001$) but not DBP. Moreover there was no correlation between DBP and age.

On presentation, 473 patients (62%) were on active anti-hypertensive treatment (including HF-specific therapy). The most commonly prescribed anti-hypertensive agents were loop-diuretics (49%), angiotensin converting enzyme inhibitors (39%), beta-blockers (25%), aldosterone inhibitor (20%) and a calcium channel antagonist (20%). Combination therapy (of any kind) was prescribed in 384 patients (81% of those actively treated), of whom 211 (55%) were prescribed triple therapy. Overall, combination therapy was associated with lower SBP (133 ± 29 vs. 140 ± 28 mmHg: $p=0.034$) and DBP (74 ± 17 vs. 78 ± 16 mmHg; $p=0.052$) than monotherapy. Those prescribed an angiotensin converting enzyme inhibitor (SBP 134 ± 27 vs. 141 ± 29 and DBP 74 ± 16 vs. 79 ± 16 mmHg: $p<0.001$) or an aldosterone inhibitor (SBP 133 ± 24 vs. 142 ± 30 mmHg) had significantly lower BP ($p<0.01$ for all comparisons). Those 120 patients with advanced heart disease prescribed combination anti-hypertensive therapy including a thiazide diuretic also had a significantly lower SBP (133 ± 24 vs. 139 ± 28 mmHg; $p=0.028$) but not DBP (76 ± 14 vs. 77 ± 16 mmHg; $p=0.592$) profile.

The pattern of prescribed treatment varied according to gender with women less likely to be prescribed a loop-diuretic (198 [41%] vs. 174 [62%], OR 0.42 [95% CI 0.31 to 0.57]; $p<0.0001$), an ACE inhibitor (160 [33%] vs. 129 [46%], OR 0.58 [95% CI 0.43 to 0.78]; $p<0.0001$) and a calcium channel antagonist (57 [12%] vs. 58 [21%], OR 0.51 [95% CI 0.34 to 0.76]; $p=0.001$). Overall, women were less likely to receive any (261 [54%] vs. 212 [76%], OR 0.37 [95% CI 0.27 to 0.52]; $p<0.0001$) or, indeed, combination anti-hypertensive therapy (217 [45%] vs. 67 [60%], OR 0.55 [95% CI 0.41 to 0.74]; $p<0.0001$) compared to men; who were more likely to have concomitant left ventricular systolic dysfunction.

4.3. Advanced cardiovascular disease

Overall, 494 patients (65%) presented with advanced forms of CVD (predominantly chronic forms of heart disease) with a combination of HF (410 cases — 54%), coronary artery disease (47 cases — 6.2%) and stroke (25 cases — 3.3%) — see Table 2. An additional 98 patients (13%) had a valvular abnormality detected by echocardiography. The majority of these (58 cases — 59%) were due to underlying structural valve disease, mainly comprising 47 cases (81%) of concomitant rheumatic valve disease and 9 cases (9.2%) of degenerative valve disease. A further 69 patients (9.1%) were diagnosed with established renal disease and 63 patients (11%) were anaemic on presentation. Based on eGFR levels, 150 patients (24%) had moderate to severe renal dysfunction and a further 258 (41%) had evidence of mildly impaired renal dysfunction. On an adjusted basis, only age (adjusted OR 1.03 per year, 95% CI 1.02 to 1.04; $p<0.0001$) predicted underlying moderate-to-severe renal dysfunction.

Although women presented with more clinical symptoms, proportionately, they were less likely to present in HF (OR 0.85, 95% CI 0.75–0.97: $p=0.018$). They were also less likely to be diagnosed with renal disease (OR 0.47, 95% CI 0.30–0.73: $p=0.001$), but more likely to present with a valvular abnormality (OR 1.42, 95% CI 1.00–2.03: $p=0.046$). While the pattern of concurrent CVD was different between men and women, overall, when adjusting for the baseline profile of cases (see Table 2 for uncomplicated versus complicated

Table 2
Comparison of uncomplicated cases versus those with advanced forms of heart disease.

	Uncomplicated HT $n=266$	HT + CVD $n=495$	p value
Socio-demographic profile			
Mean age (years) ± SD	58.1 ± 15.2	58.3 ± 15.6	[0.827]
Women	179 (67%)	303 (61%)	[0.098]
No/<5 years education			
Live in Soweto	166 (62%)	316 (64%)	[0.694]
Risk factor profile			
Family history of CVD	55 (21%)	117 (24%)	[0.170]
History of smoking	90 (34%)	202 (41%)	[0.061]
Dyslipidemia *	40 (35%)	42 (19%)	0.002
Obese (BMI>30 kg/m^2) *	61 (48%)	101 (44%)	[0.458]
Type II diabetes	64 (23%)	101 (20%)	[0.268]
Multiple risk factors	199 (75%)	376 (76%)	[0.724]
Clinical presentation			
NYHA class II:III/IV	97 (36%):73 (27%)	212 (43%):142 (29%)	[0.169]
Mean heart rate/minute	83 ± 19	87 ± 19	0.006
Mean systolic BP (mmHg)	144 ± 28	135 ± 27	<0.0001
Mean diastolic BP (mmHg)	78 ± 16	76 ± 16	[0.194]
Dizziness	146 (55%)	309 (62%)	0.055
Palpitations	135 (51%)	284 (57%)	[0.133]
Angina pectoris/chest pain	75 (28%)	152 (31%)	[0.503]
Edema (pulmonary/peripheral)	69 (26%)	179 (36%)	0.004
Estimated GFR (ml/min/1.73 m^2)*	84 ± 40	81 ± 31	[0.474]

Legend: * denotes clinical data were not available on all patients (see Methods).

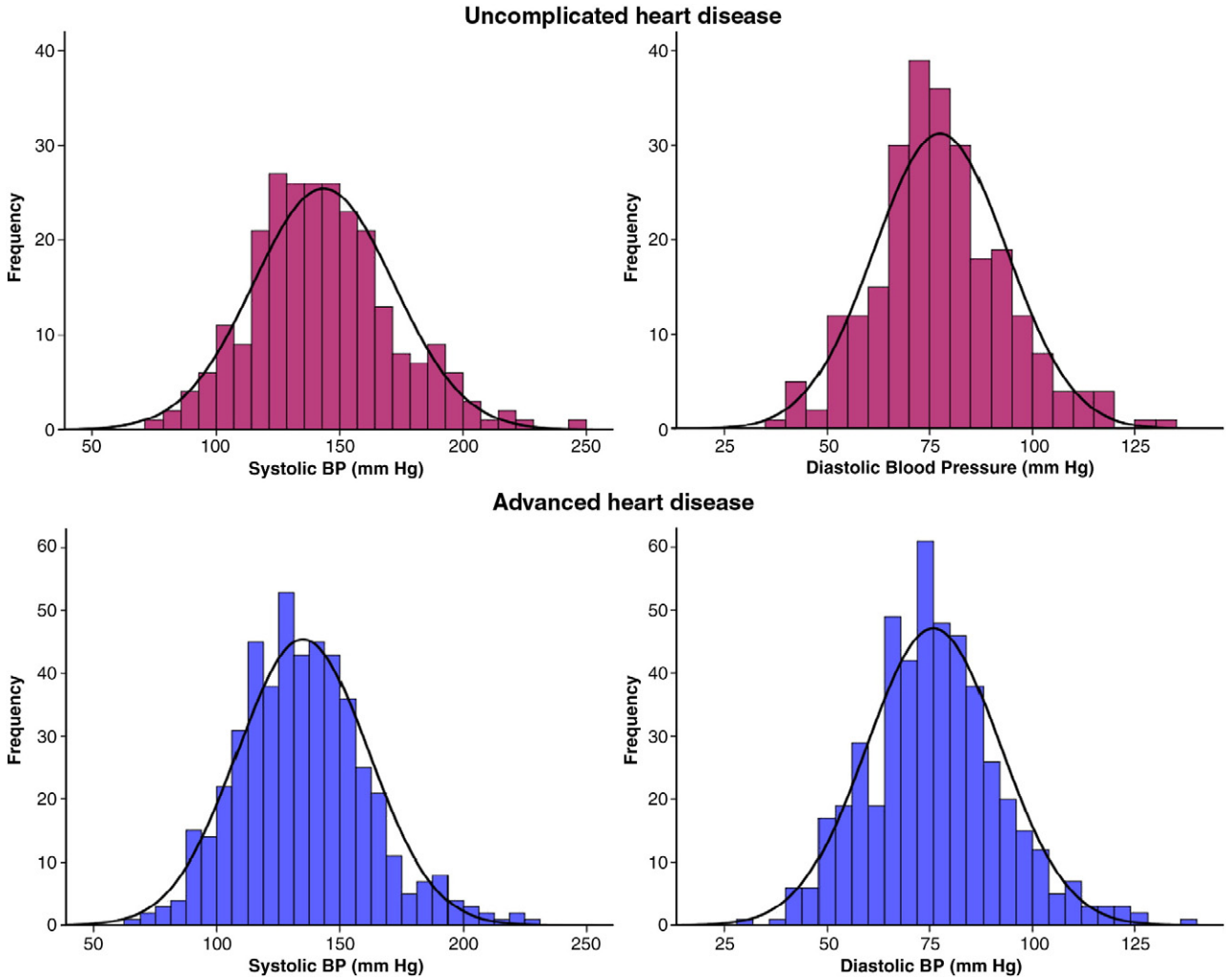

Fig. 1. Distribution of SBP and DBP in uncomplicated cases (top panel) versus those with advanced forms of heart disease (bottom panel).

cases), there was no significant gender difference in respect to the proportion of individuals presenting with established CVD (adjusted OR 1.28, 95% CI 0.77 to 2.11; $p=0.344$ for men versus women). On an adjusted basis, those with a primary diagnosis of HT were significantly less likely (adjusted OR 0.68, 95% CI 0.47 to 0.98: $p=0.037$) to have even mildly impaired renal dysfunction.

4.4. Underlying cardiac dysfunction

Table 3 shows the echocardiographic ($n=676$) and 12-lead ECG ($n=671$) profile of all those with complete data for both assessments. Overall, a total of 264 patients (39%) presented with LVH on echocardiography (14% patients with ECG evidence), 158 patients (24%) with moderate to severe systolic dysfunction, 163 patients (24%) with diastolic dysfunction and 103 patients (24%) with evidence of right HF.

Men had a significantly lower mean LVEF and significantly larger left ventricular dimensions being more likely to present with impaired systolic function (OR 2.13, 95% CI 1.50 to 3.00; $p<0.0001$) overall. Although there were no gender differences in LVH on echocardiography (septal thickness > 13 mm), women were less likely to have ECG changes of the same (OR 0.57, 95% CI 0.36 to 0.88); results not confounded by obesity. The odds of presenting with HT and concurrent systolic dysfunction ($n=279$) increased in older individuals, men, smokers, and in those with an elevated heart rate and DBP (see Table 4). There was a clear gradient of risk in the latter two variables. Conversely, the odds of concurrent systolic dysfunction decreased with increasing SBP (see text box below). There was a weak, positive relationship between SBP (on presentation) and LVEF (R^2 0.17, $p=0.028$). Alternatively, there was no correlation between LVEF and DBP. Diastolic as opposed to systolic dysfunction was associated with greater age (adjusted OR 1.02 per year, 95% CI 1.00 to 1.03: $p=0.021$) and a reported family history of CVD (adjusted OR 1.83, 95% CI 1.12 to 2.98: $p=0.015$).

5. Discussion

In one of Africa's largest and most comprehensive studies of heart disease to date, [7,9] we prospectively examined the characteristics and consequences of HT in 761 urban dwelling black Africans presenting to a tertiary care centre in one year [8]. Overall, almost 40% of patients presented with LVH confirmed by echocardiography. Moreover, two thirds of cases had previously undiagnosed heart and vascular disease (predominantly HF and renal failure) [5]. Significantly, these findings had been predicted because of worryingly low levels of detection, treatment and control of HT in urban Sub-Saharan Africa [5]. High mortality rates in those hospitalised with HT in the region have also been reported [15]. These unique data confirm, therefore, the critical importance of targeting HT as a major contributor to highly preventable, non-communicable forms of heart disease in vulnerable communities undergoing epidemiological transition.

Table 3
Results of echocardiography and 12-lead electrocardiography ($n=676$).

	All	Men	Women	p value
Systolic and diastolic function	($n=676$)	($n=250$)	($n=426$)	
Mean LVEF % (SD)	55 ± 17.0	51 ± 17.9	57 ± 16.1	<0.0001
Moderate to severe LV systolic dysfunction	185 (24%)	93 (37%)	92 (22%)	<0.0001
LV end diameter at diastole (mm)	48 ± 11.5	51 ± 10.9	47 ± 11.6	<0.0001
LV end diameter at systole (mm)	34 ± 12.6	37 ± 12.6	33 ± 12.3	<0.0001
Intra-ventricular septum diameter at diastole >13 mm	264 (39%)	97 (39%)	167 (39%)	[0.973]
Right ventricular systolic pressure >35 mmHg	103 (15%)	37 (15%)	67 (16%)	[0.967]
Diastolic dysfunction	163 (24%)	64 (26%)	99 (23%)	[0.437]
Valvular abnormality	($n=676$)	($n=250$)	($n=426$)	
Mitral regurgitation	30 (4.4%)	8 (3.2%)	22 (5.2%)	[0.232]
Tricuspid regurgitation	25 (3.7%)	7 (2.8%)	18 (4.2%)	[0.082]
Aortic stenosis	15 (2.2%)	4 (1.6%)	11 (2.6%)	[0.711]
Other abnormalities	($n=676$)	($n=250$)	($n=426$)	
Pericardial effusion	12 (1.8%)	6 (2.4%)	6 (1.4%)	[0.347]
Regional wall abnormality	11 (1.6%)	7 (2.8%)	4 (0.9%)	[0.061]
12-Lead ECG	($n=671$)	($n=245$)	($n=426$)	
Sinus rhythm	605 (90%)	222 (91%)	383 (90%)	[0.767]
Atrial fibrillation	54 (8.1%)	22 (8.9%)	32 (7.5%)	[0.501]
Left ventricular strain pattern	91 (14%)	44 (18%)	47 (11%)	0.012
Axis deviation (left or right)	158 (24%)	52 (21%)	106 (25%)	[0.282]
Bundle branch block)	40 (6.0%)	13 (5.3%)	27 (6.3%)	[0.587]

Legend: right ventricular systolic pressure — specific values recorded in 645 cases. Moderate to severe left ventricular systolic dysfunction = LVEF<45%.

While our data do not provide a complete picture of the natural history of HT leading to advanced heart disease in this urban African community, they do provide important insights for preventative strategies. Given that LVH is 2–3 fold more common in African Americans, [14] it is conceivable that we have uncovered the early signs of an epidemic of hypertensive heart disease and the worse is yet to come; particularly if adequate primary and secondary prevention is not applied. Fortunately, HT is an easily detectable condition that can be readily controlled with treatments that are not only cheap, but relevant to salt-sensitive black Africans (e.g., thiazides and dihyrdopiridine calcium antagonists) [16] in whom regression of LVH is achievable. [17] In this context, it is clear from our own [9] and previous African and African-American [18] studies that HT is an extremely important antecedent for HF. However, the progression from concentric LVH to HF has not well been defined in Africa. A report indicating that around one in five of predominantly hypertensive African-Americans with LVH developed newly impaired systolic function after a median follow up of 4 years is of some relevance [19]. Unfortunately, the confounding effects of an absence of underlying coronary artery disease in the black African context have yet to be elucidated. Moreover, the most cost-effective way to detect advanced heart disease in patients with HT (potentially using a combination of 12-lead ECG [20] and N-terminal brain natriuretic peptide [21]) in the primary care setting is yet to be determined.

In considering any preventative strategies it worth noting that in contrast to high income countries, [22] hypertensive heart disease occurred in relatively young individuals and, consistent with a review of HT in Sub-Saharan Africa, [5] predominantly women. As such, the potential pathways to HT appeared to reflect social trends in Soweto, with women more likely to be obese and men more likely to smoke with multiple modifiable risk factors a common factor in both sexes [6]. These data, therefore, highlight the need for rigorous anti-smoking campaigns among men in particular and the need to tackle poor dietary and exercise habits in women while combating the social stigma associated with weight loss and infectious status (particularly HIV infection). Reflecting the nature of the clinical registry, the majority of patients (two thirds) presented with a low to normal BP (54% overall) and advanced forms of heart disease. The most notable were HF and/or valvular dysfunction in over two-thirds of cases with associated "burn-out" of previously recorded high BP levels. For many, therefore, the opportunity to detect and prevent the consequences of HT had long passed. It is also worth noting, given the advanced forms of HF, traditional forms of secondary prevention would also be inappropriate. Consistent with a previous report, [23] we found comparatively low levels of concurrently diagnosed renal disease and anaemia. However, we did determine around two thirds of patients had a combination of moderate to severe (24%) or mild (41%) renal dysfunction most probably attributable to their underlying HT status. The challenge of adequately controlling BP and other risk factors in this setting, has been highlighted by the recently reported HiHi study of hypertensive black Africans in Cape Town townships [24].

Despite adhering to Strobe guidelines wherever possible [25] this study has a number of limitations that require comment. Firstly, not all patients with HT in Soweto are managed by the Cardiology Unit at Baragwanath Hospital. Moreover, we acknowledge that individuals with milder forms of HT would not be captured by the Heart of Soweto Clinical Registry [8] and this was a cross-sectional survey of more advanced forms of HT while potentially failing to capture hemorrhagic strokes diagnosed via CT scans with an obvious non-cardiac origin. It is important to note that use of private health facilities in black South Africans is very low [26] and we are unlikely to have missed many patients seeking specialist care from other facilities in the region. As a clinical registry, we did not capture identical clinical data for all patients and have relied on clinical diagnoses. However, our ability to provide comprehensive 12-lead ECG and detailed echocardiographic data for nearly all patients is a unique strength.

In conclusion, these data extend upon previous reports from urban regions in sub-Saharan Africa suggesting that the phenomenon of epidemiologic transition has led to a rising prevalence of HT [5,6]. Overall, these data demonstrate the potential for undetected and untreated HT to lead to advanced forms of heart disease (particular HF). As such the development and application of gender specific community-based screening and prevention programs adapted to the African context are urgently required to truncate an almost inevitable rise in the burden of HT heart disease in vulnerable communities in Sub-Saharan Africa.

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Table 4
Independent correlates of LV systolic dysfunction.

	Adjusted OR	95% CI	p value
Age	1.01	1.00 to 1.03	0.037
Women versus men	0.49	0.32 to 0.73	0.001
Smoker versus non-smoker	1.75	1.18 to 2.59	<0.001
Lowest systolic BP quartile versus			
Q2: 119 to 136 mmHg	0.49	0.27 to 0.90	0.22
Q3: 137 to 153 mmHg	0.37	0.19 to 0.73	0.004
Q4: 154 to 247 mmHg	0.21	0.10 to 0.47	<0.0001
Lowest diastolic BP quartile versus			
Q2: 67 to 76 mmHg	1.42	0.77 to 2.64	0.263
Q3: 77 to 86 mmHg	1.86	0.95 to 3.66	0.072
Q4: 87 to 142 mmHg	2.42	1.11 to 5.28	0.027
Lowest heart rate quartile versus			
Q2: 73 to 84 beats/min	2.53	1.36 to 4.69	0.003
Q3: 85 to 98 beats/min	2.79	1.53 to 5.11	0.001
Q4: 99 to 148 beats/min	4.02	2.21 to 7.30	<0.0001

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