# Casual blood pressure among Tanzanian undergraduate students: need for redefining population specific operational threshold between normotension and hypertension 

\author{


#### Abstract

<br> Background: Despite of the recommendations to use population specific blood pressure (BP) references which consider time, ethnicity and environmental factors, there is limited information regarding BP profile among Tanzanians. This cross sectional study was done to determine casual BP profile among healthy volunteer students of the Catholic University of Health and Allied Sciences in Mwanza, Tanzania. <br> Methods: Socio-demographic and lifestyle data were collected using questionnaires. Systolic BP (SBP) and diastolic BP (DBP) were measured using aneroid sphygmomanometer. <br> Results: A total of 299 students (males=204; females=95) were involved in the study. Their mean age was $23.4 \pm$ 0.2 years. SBP ranged from $82-150 \mathrm{mmHg}$ (mean $=115.7 \pm 0.7 \mathrm{mmHg}$ ) and DBP ranged from $44-100 \mathrm{mmHg}$ (mean= $71.9 \pm 0.6 \mathrm{mmHg}$ ). Mean arterial pressure (MAP) was $86.5 \pm 0.5 \mathrm{mmHg}$. Males had significantly higher BP than females; and BP was noticed to increase with increased age and body weight. Upper limits of the normal SBP and DBP calculated as mean +2 SDs and $95^{\text {th }}$ percentiles were 140.5 mmHg and 138 mmHg , respectively and for DBP were 91.8 mmHg and 90 mmHg , respectively. <br> Conclusion: The observed upper limits of the distribution of normal BP for the age of the participants are higher than the World Health Organization recommended values. We recommend a larger study to determine BP among healthy Tanzanians to establish the normal values.


}

Keywords: casual blood pressure, operational thresholds, hypertension, Tanzania

## Introduction

Blood pressure (BP) is the force of blood pushing against the walls of blood vessels. Arterial blood pressure (ABP) is usually expressed as systolic blood pressure (SBP) over diastolic blood pressure (DBP). Significant reduction of BP (hypotension) leads to hypoperfusion of organs. On the other hand, high BP (hypertension) increases the work load to the heart and blood vessels, the effect which can lead to cardiac failure, stroke, heart attack, renal failure and retinal damage (Luft et al., 1999; Cuspidi et al., 2001). In adults, the BP normally ranges between $90 / 60 \mathrm{mmHg}$ and $120 / 80 \mathrm{~mm}$ Hg . Therefore, the upper limits of BP which is considered normal for adults is $120 / 80 \mathrm{mmHg}$ (Robinson \& Brucer, 1939; Chobanian et al., 2003; Cifkova et al., 2003), although in clinical practice, hypertension is considered when a seated BP of 140/90 or higher is recorded with well-maintained equipment three or more consecutive times (Chobanian et al., 2003; Cifkova et al., 2003). Individuals with intervening levels ( $121 / 81-139 / 89 \mathrm{mmHg}$ ) are considered pre-hypertensive. This is the group that has increased health risks and from which definite hypertension is more likely to develop in near future (Chobanian et al., 2003).

Current recommendations from World Health Organization (WHO), International Society of Hypertension and European Society of Hypertension/European Society of Cardiology refer 120/80 mmHg as optimal, $120 / 80-129 / 84$ as normal, $130 / 85-139 / 89$ as high normal BP and $\geq 140 / 90 \mathrm{mmHg}$ as

[^0]hypertension (Cifkova et al., 2003). These reference values were set more than seven decades ago based on statistical and clinical as well as mortality rate studies which were done in developed countries (Robinson \& Brucer, 1939).

While it is customary to apply the same BP reference ranges to patients with diverse ancestral origins or who are exposed to different environmental conditions, it is known that there are differences, particularly between "normal" values obtained from different ethnic populations (Williams, 1969; Liebman et al. 1986; Alpert \& Fox, 1993; Manatunga et al., 1993; Muntner et al., 2004; Brummett et al., 2012) or the same ethnic group under different environment (Elliott \& Marmot, 1983; Poulter et al., 1984, 1990; Pauletto et al., 1996). These differences suggest that different reference ranges need to be considered for each population. The use of population specific reference values should be considered because choosing unrelated "normal ranges" may lead to either overestimation of hypertension and unnecessary use of antihypertensive drugs or delayed diagnosis of hypertension. Indeed, some countries have started establishing their own population specific BP reference ranges based on the distribution of BP among their populations (Chiolero, 2014). Additionally, age specific blood pressure has been reported to vary over decades (Kesteloot et al., 1980; de Gaudemaris et al., 1994; Muntner et al., 2004). For instance, studies in the United States in 1999-2000 showed that, age specific BP were significantly higher compared to 1988-1994 levels (Muntner et al., 2004).

Despite of documented variations in BP by ethnicity and environmental factors, as well as by time, there is limited information regarding the BP profile among Tanzanians. In diagnosing hypertension or hypotension, physicians use reference values which were established more than seven decades ago based on studies done in developed countries. Applicability of these reference values to Africans particularly Tanzanians is not clearly known. Therefore, this study aimed to determine the casual BP pressure profile of undergraduate students of Catholic University of Health and Allied Sciences (CUHAS) in Tanzania.

## Materials and Methods

## Study subjects

The cross-sectional descriptive and analytical study was carried out at CUHAS in Mwanza, northwestern Tanzania. C Tanzanian healthy volunteers from among undergraduate students of CUHAS were enrolled in this study. Non-Tanzanian students and/or those who were known to be suffering from any cardiovascular disease such as hypertension and heart failure were excluded. Additionally, a checklist was used to exclude students with symptoms and/or signs of cardiovascular diseases. Female students who declared to be pregnant were also excluded. This population was selected because it has representation of various Tanzanians ethnic groups, as the University enrols students from different parts of the country.

## Data collection

Structured self-administered questionnaires were used to collect socio-demographic and lifestyle data as well as providing their recently measured body weight and height. Self-reported body weight and height were used to estimate body mass index (BMI). Casual ABP was measured using adult sized cuff aneroid sphygmomanometer (Wuxi Medical Instrument Factory, China) and a Littmann Classic II S.E. stethoscope (3M Health Care, USA). The accuracy of the aneroid sphygmomanometer was verified by comparing its readings with table mercury sphygmomanometer's readings for ten subjects who were recruited during their routine learning activities. BP was measured by auscultatory technique following the procedures described by

Chobian et al. (2003). In brief, subjects were required to seat quietly for at least 5 minutes in a chair with the arm flexed and rested on a table. Subjects who reported to have smoked or used caffeine or doing exercise were given at least 30 minutes resting before taking their BP. SBP was estimated as the point in the gauge at which the first Korotkoff sound was heard, and the disappearance of Korotkoff sounds was used to define DBP. SBP and DBP measurements were repeated twice for each subject and the average recorded.

## Data analysis

SBP and DBP were used to derive mean arterial blood pressure (MAP), which was calculated as DBP $+1 / 3$ (SBP - DBP). Statistical analyses were done using SPSS software version 17. Categorical data were cleaned, edited, coded and entered into Microsoft Excel together with continuous data. Data were then exported to SPSS for analysis. To determine the BP profile, the SBP, DBP and MAP are expressed in ranges and mean $\pm$ standard error of the mean (SEM). Means of various intervals of SBP, DBP and MAP calculated using SPSS were used to draw frequency and percentage cumulative curves for general description. Upper limits of normal BP distribution, which may be used as operational threshold between normotension and hypertension, were determined as mean +2 standard deviations (SDs) and the $95^{\text {th }}$ percentile. Comparison of mean SBP, DBP and MAP by various independent variables was done by repeated measures analysis of variance (ANOVA). When significant ANOVAs were obtained, post-hoc analysis was performed using Tukey's Multiple Comparison Testing. Statistical significance levels were fixed at two-tailed $p$-value of 0.05 .

## Ethical considerations

Ethical clearance was sought and provided by the joint CUHAS and Bugando Medical Centre Research Ethics and Review Committee. Subjects voluntarily participated in the study and they signed written informed consents.

## Results

## Characteristics of study participants

A total of 299 university students, aged $19-43$ years (mean $=23.4 \pm 0.2$ years) participated in this study. Out of 299 participants, 204 ( $68.2 \%$ ) were males and 95 ( $31.8 \%$ ) were females. The participants were from different parts of the country with majority of them ( $48.5 \%$ ) being from Lake Zone regions, namely Mwanza, Geita, Kagera, Shinyanga and Mara. Most of participants (58.9\%) did not have family history of hypertension. About three quarters ( $75.9 \%$ ) of the participants reported to be using moderate amount of salt in their diet while only $4(1.3 \%)$ reported to use large amount of salt and the rest using small amount. Most students (82.3\%) were using fat diets while only $17.7 \%$ were avoiding use of fat diets. About half participants were performing regular exercises on daily basis. More than $98 \%$ participants were non-smokers and more than $82 \%$ were non-alcohol users.

## Normal casual blood pressure

The SBP ranged from $82-150 \mathrm{mmHg}$ with a mean of $115.7 \pm 0.7 \mathrm{mmHg}$. The SBP values were normally distributed with males' curve being relatively higher compared to females' curve (Figure 1A). Most male and female participants had SBP of $110-120 \mathrm{mmHg}$. Upper limit of the SBP calculated as Mean + 2SDs was 140.5 mmHg ( 141.4 mmHg for males and 133.5 mmHg for females). The SBP covering $95^{\text {th }}$ percentile was 138 mmHg with the central $95^{\text {th }}$ percentile range of $90-140 \mathrm{mmHg}$ as read off the cumulative percent curve (Figure 1C). There was significant difference ( $p=0.000$ ) in mean SBP
between males and females with males having higher SBP ( $118.7 \pm 0.8 \mathrm{mmHg}$ ) than females ( $109.3 \pm 1.2$ mmHg ).


Figure 1: Distribution of casual SBP among undergraduate students. A: frequency distribution curves; B: cumulative frequency curves; C : cumulative percent curves

The DBP ranged from $44-100 \mathrm{mmHg}$ with a mean of $71.9 \pm 0.6 \mathrm{mmHg}$. Most frequently measured DBP was $61-70 \mathrm{mmHg}(71-80 \mathrm{mmHg}$ for males and $61-70 \mathrm{mmHg}$ for females) (Figure 2A). The DBP was also normally distributed with the males' graph being higher than females' graph (Figure 2A). Upper limit of DBP calculated as Mean+2SDs was 91.8 mmHg ( 92.5 mmHg for males and 89.5 mmHg for females). The DBP covering $95^{\text {th }}$ percentile was 90 mmHg with the central $95^{\text {th }}$ percentile range of $50-90 \mathrm{mmHg}$ as read off the cumulative curves (Figure $2 C$ ). DBP was significantly ( $p=0.002$ ) higher among males ( $73.2 \pm 0.7 \mathrm{mmHg}$ ) than among females ( $69.3 \pm 1.0 \mathrm{mmHg}$ ).


Figure 2: Distribution of casual DBP among undergraduate students. A: frequency distribution curves; B: cumulative frequency curves; $C$ : cumulative percent curves

MAP ranged from $63-113 \mathrm{mmHg}$ with a mean of $86.5 \pm 0.5 \mathrm{mmHg}$. Most participants (both males and females) had MAP of $81-90 \mathrm{mmHg}$ (Figure 3A). The MAP was also normally distributed with the males' graph being higher than females' graph (Figure 3A). Upper limit for MAP calculated as Mean + 2SDs was found to be 105.1 mmHg ( 106.1 mmHg for males and 100.5 mmHg for females). The $95^{\text {th }}$ percentile of the MAP was 102 mmHg with the central $95^{\text {th }}$ percentile range of $68-105$ mmHg as read off the cumulative curves (Figure 3C). MAP significantly ( $p=0.000$ ) differed between males and females, with males having higher MAP ( $88.3 \pm 0.6 \mathrm{mmHg}$ ) than females ( $82.6 \pm 0.9$ mmHg ).

SBP, DBP and MAP were all higher among older students than younger ones (Figure 4). SBP for <21 years, 21-30 years and >30 years old participants were $113 \pm 1.9 \mathrm{mmHg}, 115.9 \pm 0.8 \mathrm{mmHg}$ and $127.6 \pm 4.5 \mathrm{mmHg}$ respectively ( $p=0.014$ ). DBP were $69.2 \pm 1.6 \mathrm{mmHg}, 72.2 \pm 0.6 \mathrm{mmHg}$ and $79.9 \pm 2.3$ mmHg for participants aged $<21$ years, $21-30$ years and $>30$ years, respectively $(p=0.018)$. MAP was $83.8 \pm 1.3 \mathrm{mmHg}$ for students aged <21 years, $86.7 \pm 0.6 \mathrm{mmHg}$ for students aged $21-30$ years and $95.9 \pm 3.0 \mathrm{mmHg}$ for $>30$ years old students $(p=0.004)$. However, a post-hoc analysis showed no significant difference in SBP, DBP and MAP between subject aged <21 years and those aged 21-30 years.

Mean differences in SBP, DBP and MAP between subject aged <21 years and those aged 21 30 years were $-2.84(p=0.331,95 \% \mathrm{Cl}-7.53,1.85),-3.01(p=0.148,95 \% \mathrm{Cl}-6.79,0.77)$ and $-2.89(p=$ $0.130,95 \% \mathrm{Cl}-6.40,0.62)$, respectively. SBP mean difference between subjects >30 years and <21 years old was $14.55(p=0.010,95 \% \mathrm{Cl} 2.87,26.22)$ and with those aged $21-30$ years was 11.71 ( $p=$ $0.034,95 \% \mathrm{Cl} 0.71,22.71$ ). DBP significantly differed (mean difference 10.68, $p=0.021,95 \% \mathrm{Cl} 1.28$, 20.07) between subjects aged <21 years and subjects aged >30 years but there was no significant difference (mean difference $7.66, p=0.105,95 \% \mathrm{Cl}-1.19,16.51$ ) between $21-30$ years old subjects and those aged $>30$ years. MAP like SBP significantly differed between subjects aged $<21$ years and subjects aged $>30$ years and between $21-30$ years' subjects and those aged $>30$ years. MAP mean difference between subjects >30 years and <21 years was $12.02(p=0.004,95 \% \mathrm{Cl} 3.29,20.74)$ and with those aged $21-30$ years was $9.12(p=0.025,95 \% \mathrm{Cl} 0.90,17.34)$.

A


B

c


Figure 3: Distribution of MAP among undergraduate students. A: frequency distribution curves; B: cumulative frequency curves; $C$ : cumulative percent curves

DBP and MAP increased with increase in BMI although no significant variation in SBP by BMI was observed. SBP among underweight, normal, overweight and obese subjects were $106.9 \pm 3.7 \mathrm{mmHg}$, $115.9 \pm 0.7 \mathrm{mmHg}, 115.4 \pm 2.3 \mathrm{mmHg}$ and $122.8 \pm 6.4 \mathrm{mmHg}$ respectively ( $p=0.064$ ). DBP among underweight, normal, overweight and obese subjects were $67.7 \pm 3.5 \mathrm{mmHg}, 71.7 \pm 0.6 \mathrm{mmHg}, 72.2 \pm$ 1.5 mmHg and $82.8 \pm 2.8 \mathrm{mmHg}$ respectively ( $p=0.010$ ). DBP mean difference between underweight and obese subjects was $-15.08(p=0.008,95 \% \mathrm{CI}-27.33,-2.84)$ and between subjects with normal BMI and obese ones was -11.07 ( $p=0.009,95 \% \mathrm{Cl}-20.13,-2.01$ ). DBP mean difference between overweight and obese subjects was -10.57 ( $p=0.026,95 \% \mathrm{Cl}-20.24,-0.90$ ). However, there was no significant difference in DBP between underweight and normal subjects ( $p=0.624$ ) as well as between underweight and overweight subjects ( $p=0.589$ ).


Figure 4: Increase in blood pressure with age
MAP among underweight, normal, overweight and obese subjects were $80.8 \pm 3.3 \mathrm{mmHg}$, $86.4 \pm 0.6 \mathrm{mmHg}, 86.6 \pm 1.5 \mathrm{mmHg}$ and $96.1 \pm 3.6 \mathrm{mmHg}$, respectively ( $p=0.007$ ). MAP mean difference between underweight and obese subjects was -15.35 ( $p=0.003,95 \% \mathrm{Cl}-26.77,-3.93$ ) while between normal BMI and obese subjects was -9.72 ( $p=0.016,95 \% \mathrm{CI}-18.17,-1.27$ ) and between overweight and obese subjects was $-9.55(p=0.033,95 \% \mathrm{Cl}-18.56,-0.53)$. There was no significant difference in MAP between other group categories.

Table 1: Summary of the upper limits of the distribution of normal casual blood pressure

| Blood Pressure | Mean $\pm$ SEM | Upper Limits of the Distribution <br> Mean +2 SDs |  |
| :--- | :--- | :--- | :--- |
|  | $115.7 \pm 0.7$ | 140.5 | 138.0 |
| Diastolic Blood Pressure $(\mathrm{mmHg})$ | $71.9 \pm 0.6$ | 91.8 | 90.0 |
| Mean Arterial Pressure $(\mathrm{mmHg})$ | $86.5 \pm 0.5$ | 105.1 | 102.0 |

## Upper limits of the distribution of casual blood pressure

The upper limits of BP distribution which can be used as an operational threshold between normal blood pressure and hypertension among Tanzanian undergraduate students are summarized in table

1. The upper limits were determined by calculating the Mean +2 SDs and the $95^{\text {th }}$ percentile of the BP distributions.

## Discussion

The present study is the first to describe blood pressure profile among selectively and subjectively healthy Tanzanians. The study has determined reference values of casual BP among Tanzanian undergraduate students, a group of young adults. The study has also established the upper limits of BP distribution which can be used as the operational thresholds between normotension and hypertension by calculating the mean two standard deviation and the $95^{\text {th }}$ percentile of the BP distribution in subjectively healthy subjects. The approach based on the mean +2 SDs is a parametric method that assumes normally distributed data and the approach based on the $95^{\text {th }}$ percentile limit is a nonparametric and distribution-free method. The $95^{\text {th }}$ percentile application is therefore not limited by the characteristics of the underlying distribution.

Empirical ranges of SBP, DBP and MAP observed in the our study are not comparable with the ranges among undergraduate students in Uganda who were noted to have SBP, DBP and MAP ranging $100-179 \mathrm{mmHg}, 60-139 \mathrm{mmHg}$ and $80-159 \mathrm{mmHg}$, respectively (Bimenya et al., 2005). Likewise, the ranges covering the central $95^{\text {th }}$ percentile in our study differ from those observed in the Uganda study (Bimenya et al., 2005). In our study, both the empirical and central $95^{\text {th }}$ percentile ranges appear to be down displaced. The likely reason for these deviations is the differences in the characteristics of the subjects. We excluded subjects who were known to be hypertensive and those with symptoms of hypertension while the Uganda study, included the subjects indiscriminately. Genetic differences between subjects enrolled in Uganda and those in our studies as well as differences in environmental factors may also be contributing factors.

Subjects with diverse ancestral origin have varied BP (Williams, 1969; Liebman et al., 1986; Alpert \& Fox, 1993; Manatunga et al., 1993; Brummett et al., 2012; Muntner et al., 2004). Effects of environmental factors such as diet and altitude on PB have been widely described (Marticorena et al., 1969; Gleibermann, 1973; Oliver et al., 1975; Elliott \& Marmot, 1983; Carvalho et al., 1989; Poulter et al., 1984, 1990). A study examining mixed effects of ethnicity and environmental factors on BP among 52 populations across the world reported that Yanomamo and Xingu Indians of Brazil and rural populations in Kenya and Papua New Guinea had significantly lower BP than the rest of populations studied (Carvalho et al., 1989). Other studies have shown that when people from rural communities with low mean BP migrate to urban areas, their BP levels increase significantly, thereby emphasising the influence of environmental factors on BP (Cruz-Coke et al., 1964; Shaper et al., 1969; Sever et al., 1980; Poulter et al., 1990).

Observed mean SBP and DBP in the current study are comparable with the means calculated by Thijs and colleagues (1998). The means SBP and DBP in our study are closer to means obtained among adults of $25-34$ years and lower than values among subjects who were above 35 years as reported by a study in Dar es Salaam (Bovet et al., 2002). However, unlike in our study, Bovet et al. (1998) in Dar es Salaam included hypertensive patients who were on medications.

The mean SBP determined in the current study is lower than SBP among fish consuming population near Lake Nyasa (Pauletto et al., 1996). The observed mean DBP in our study is comparable to the DBP of residents of the shores of Lake Nyasa but lower than that of individuals living in the hills around Lake Nyasa (Pauletto et al., 1996). The mean BP levels observed in our study are also lower compared to mean values obtained among healthy Caucasians in France (Rigat et al., 1990). This is contrary to previous knowledge that Caucasians have lower mean BP than Blacks (Liebman et al., 1986; Rigat et al., 1990; Manatunga et al., 1993; Alpert \& Fox, 1993; Muntner et al.,

2004; Brummett et al., 2012;). The observed mean SBP and DBP in the present study are slightly lower than values documented in various British, French and Germany literature which shows that the normotensive subjects (Staessen et al., 1994). The SBP and DBP in our current study are also lower than BP levels of Japanese as reported by studies done during the 1990s (Imai et al., 1993a,b; De Gaudemaris et al., 1994). Like in our findings, studies elsewhere in the world have reported higher casual PB among males than females (Kesteloot et al., 1980; Imai et al., 1993a, b; De Gaudemaris et al., 1994; Ohkubo et al., 1998). Increase in BP with increasing BMI noted in our study is in agreement with observations made in previous studies (Stamler et al., 1978; MacMahon et al., 1987; Dyer \& Elliott, 1989; Cassano et al., 1990; Tesfaye et al., 2007).

Upper limits of SBP and DBP by parametric method in our study were higher than the limits reported by Robinson \& Brucer (1939) using a similar approach. The smaller sample size in our study could explain this difference. Genetic differences and time factor between the participants in these two studies may also be another reason accounting for higher upper limits in our study. We studied mainly black Tanzanians, while analysis done by Robinson \& Brucer (1939) was based mainly on Caucasian subjects. Several previous studies have reported higher BP among black subjects than Caucasians (Liebman et al., 1986; Alpert \& Fox, 1993; Manatunga et al., 1993; Muntner et al., 2004). Age specific BP levels are known to increase over decades. For instance, comparison of adolescents' BP measured in 1988-1994 and 1999-2000 demonstrated that both systolic and diastolic blood pressure levels were significantly higher in 1999-2000 than in 1988-1994 (Muntner et al., 2004). The upper limits of normal SBP and DBP determined in this study were also higher than the reference values for the self-recorded blood pressure determined by Thijs et al (1998). Thijs and colleagues relied on self-measured pressures which are usually lower than clinic or hospital measured pressures (Møller et al., 2003).

In conclusion, compared to the current WHO recommended reference values for diagnosis and treatment of hypertension in adults, the upper limits of the distribution of BP observed in our study are higher for the mean age of the participants. This indirectly signifies that, higher upper limits of BP for healthy Tanzanians aging 30 years and above should be expected. Because the present reference values were derived from a population-based study in a University population, applicability of the findings to the whole Tanzanian population should be considered with caution. We recommend a larger study to determine BP among healthy Tanzanians to establish the normal values.

## Acknowledgements

The authors wish to thank the students who participated in this study. We also thank Dr. Humphrey Mazigo for technical assistance in data analysis.

## Declaration of Interest

There is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

## References

Alpert, B. \& Fox, M. (1993) Racial aspects of blood pressure in children and adolescents. Pediatric Clinics of North America, 40: 13-22.

Bimenya, G., Byarugaba, W., Kalungi, S., Mayito, J., Mugabe, K., Makabayi, R., Ayebare, E., Wanzira, H. \& Muhame, M. (2005) Blood pressure profiles among Makerere University undergraduate students. African Health Sciences 5: 99-106.
Bovet, P., Ross, A.G., Gervasoni, J.-P., Mkamba, M., Mtasiwa, D.M., Lengeler, C., Whiting, D. \& Paccaud, F. (2002) Distribution of blood pressure, body mass index and smoking habits in the urban population of Dar es Salaam, Tanzania, and associations with socioeconomic status. International Journal of Epidemiology 31: 240-247.
Brummett, B.H., Babyak, M.B., Siegler, I.C., Surwit, R., Georgiades, A., Boyle, S.H. \& Williams, R.B. (2012) Systolic blood pressure and adiposity: examination by race and gender in a nationally representative sample of young adults. American Journal of Hypertension 25: 140-144.
Carvalho, J.J., Baruzzi, R.G., Howard, P.F., Poulter, N., Alpers, M.P., Franco, L.J., Marcopito, L.F., Spooner, V.J., Dyer, A.R., Elliott, P. \& Et Al. (1989) Blood pressure in four remote populations in the INTERSALT Study. Hypertension 14: 238-246.
Cassano, P.A., Segal, M.R., Vokonas, P.S. \& Weiss, S.T. (1990) Body fat distribution, blood pressure, and hypertension: a prospective cohort study of men in the normative aging study. Annals of Epidemiology 1: 33-48.
Chiolero, A., (2014) The quest for blood pressure reference values in children. Journal of Hypertension 32: 477-479.
Chobanian, A.V., Bakris, G.L., Black, H.R., Cushman, W.C., Green, L.A., Izzo, J.L., Jones, D.W., Materson, B.J., Oparil, S. \& Wright, J.T. (2003) Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension 42: 1206-1252.
Cifkova, R., Erdine, S., Fagard, R., Farsang, C., Heagerty, A.M., Kiowski, W., Kjeldsen, S., Luscher, T., Mallion, J.M. \& Mancia, G. (2003) Practice guidelines for primary care physicians: 2003 ESH/ESC hypertension guidelines. Journal of Hypertension 21: 1779-1786.
Cruz-Coke, R., Etcheverry, R. \& Nagel, R. (1964) Influence of migration on blood-pressure of Easter Islanders. Lancet 283: 697-699.
Cuspidi, C., Macca, G., Salerno, M., Michev, L., Fusi, V., Severgnini, B., Corti, C., Meani, S., Magrini, F. \& Zanchetti, A. (2001) Evaluation of target organ damage in arterial hypertension: which role for qualitative funduscopic examination? Italian Heart Journal 2: 702-706.
De Gaudemaris, R., Chau, N.P., Mallion, J.-M. \& De la Mesure, G., (1994) Home blood pressure: variability, comparison with office readings and proposal for reference values. Journal of Hypertension 12: 831-838.
Dyer, A.R. \& Elliott, P. (1989) The INTERSALT study: relations of body mass index to blood pressure. INTERSALT Co-operative Research Group. Journal of Human Hypertension 3: 299-308.
Elliott, P. \& Marmot, M. (1983) International studies of salt and blood pressure. Annals of Clinical Research 16: 67-71.
Gleibermann, L. (1973) Blood pressure and dietary salt in human populations. Ecology of Food and Nutrition 2: 143-156.
Imai, Y., Nagai, K., Sakuma, M., Sakuma, H., Nakatsuka, H., Satoh, H., Minami, N., Munakata, M., Hashimoto, J. \& Yamagishi, T. (1993a) Ambulatory blood pressure of adults in Ohasama, Japan. Hypertension 22: 900-912.
Imai, Y., Satoh, H., Nagai, K., Sakuma, M., Sakuma, H., Minami, N., Munakata, M., Hashimoto, J., Yamagishi, T. \& Watanabe, N. (1993b) Characteristics of a community-based distribution of home blood pressure in Ohasama in northern Japan. Journal of Hypertension 11: 1441-1449.

Kesteloot, H., Park, B., Lee, C., Brems-Heyns, E. \& Joossens, J. (1980) A comparative study of blood pressure and sodium intake in Belgium and in Korea. In: Epidemiology of Arterial Blood Pressure. Springer Netherlands.
Liebman, M., Chopin, L.F., Carter, E., Clark, A.J., Disney, G.W., Hegsted, M., Kenney, M.A., Kirmani, Z.A., Koonce, K.L. \& Korslund, M.K. (1986) Factors related to blood pressure in a biracial adolescent female population. Hypertension 8: 843-850.
Luft, F.C., Mervaala, E., Müller, D.N., Gross, V., Schmidt, F., Park, J.K., Schmitz, C., Lippoldt, A., Breu, V. \& Dechend, R. (1999) Hypertension-induced end-organ damage A new transgenic approach to an old problem. Hypertension 33: 212-218.
Møller, D.S., Dideriksen, A., Sørensen, S., Madsen, L.D. \& Pedersen, E.B. (2003) Accuracy of telemedical home blood pressure measurement in the diagnosis of hypertension. Journal of Human Hypertension 17: 549-554.
MacMahon, S., Cutler, J., Brittain, E. \& Higgins, M. (1987) Obesity and hypertension: epidemiological and clinical issues. European Heart Journal 8: 57-70.
Manatunga, A.K., Jones, J.J. \& Pratt, J.H. (1993) Longitudinal assessment of blood pressures in black and white children. Hypertension 22: 84-89.
Marticorena, E., Ruiz, L., Severino, J., Galvez, J. \& Peñaloza, D. (1969) Systemic blood pressure in white men born at sea level: changes after long residence at high altitudes. American Journal of Cardiology 23: 364-368.
Muntner, P., He, J., Cutler, J.A., Wildman, R.P. \& Whelton, P.K. (2004) Trends in blood pressure among children and adolescents. Journal of the American Medical Association 291: 2107-2113.
Oliver, W.J., Cohen, E.L. \& Neel, J.V. (1975) Blood pressure, sodium intake, and sodium related hormones in the Yanomamo Indians, a" no-salt" culture. Circulation 52: 146-151.
Pauletto, P., Puato, M., Caroli, M.G., Casiglia, E., Munhambo, A.E., Cazzolato, G., Bon, G.B., Angeli, M. T., Galli, C. \& Pessina, A.C. (1996) Blood pressure and atherogenic lipoprotein profiles of fishdiet and vegetarian villagers in Tanzania: the Lugalawa study. Lancet 348: 784-788.
Poulter, N., Khaw, K., Hopwood, B., Mugambi, M., Peart, W., Rose, G. \& Sever, P. (1990) The Kenyan Luo migration study: observations on the initiation of a rise in blood pressure. British Medical Journal 300: 967-972.
Poulter, N., Khaw, K.T., Hopwood, B., Mugambi, M., Peart, W.S., Rose, G. \& Sever, P.S. (1984) Blood pressure and associated factors in a rural Kenyan community. Hypertension 6: 810-813.
Rigat, B., Hubert, C., Alhenc-Gelas, F., Cambien, F., Corvol, P. \& Soubrier, F. (1990) An insertion/deletion polymorphism in the angiotensin I-converting enzyme gene accounting for half the variance of serum enzyme levels. Journal of Clinical Investigation 86: 1343.
Robinson, S.C. \& Brucer, M. (1939) Range of normal blood pressure: a statistical and clinical study of 11,383 persons. Archives of Internal Medicine 64: 409-444.
Sever, P., Gordon, D., Peart, W. \& Beighton, P. (1980) Blood-pressure and its correlates in urban and tribal Africa. Lancet 316: 60-64.
Shaper, A., Leonard, P., Jones, K. \& Jones, M. (1969) Environmental effects on the body build, blood pressure and blood chemistry of nomadic warriors serving in the army in Kenya. East African Medical Journal 46: 282.
Staessen, J.A., O'brien, E.T., Amery, A.K., Atkins, N., Baumgart, P., De Cort, P., Degaute, J.-P., Dolenc, P., De Gaudemaris, R. \& Enström, I. (1994) Ambulatory blood pressure in normotensive and hypertensive subjects: results from an international database. Journal of Hypertension 12: S13S22.

Stamler, R., Stamler, J., Riedlinger, W.F., Algera, G. \& Roberts, R.H. (1978) Weight and blood pressure: findings in hypertension screening of 1 million Americans. Journal of the American Medical Association 240: 1607-1610.
Tesfaye, F., Nawi, N., Van Minh, H., Byass, P., Berhane, Y., Bonita, R. \& Wall, S. (2007) Association between body mass index and blood pressure across three populations in Africa and Asia. Journal of Human Hypertension 21: 28-37.
Thijs, L., Staessen, J. A., Celis, H., De Gaudemaris, R., Imai, Y., Julius, S. \& Fagard, R. (1998) Reference values for self-recorded blood pressure: a meta-analysis of summary data. Archives of Internal Medicine 158: 481-488.
Williams, A.W. (1969) Blood pressure differences in Kikuyu and Samburu communities in Kenya. East African Medical Journal 46: 262.


[^0]:    * Corresponding E-mail: hdika2001@yahoo.co.uk

