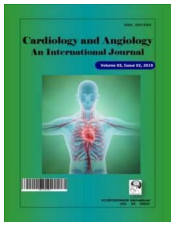

Cardiology and Angiology: An International Journal
6(2): 1-21, 2017; Article no.CA.32180
ISSN: 2347-520X, NLM ID: 101658392

SCIENCEDOMAIN *international*
www.sciencedomain.org

Prevalence and Correlates of Pre-hypertension and Hypertension Results-II: A Screening Plan in a Selected Military Community in Central Saudi Arabia

Ashraf E. Saad[1], Ahmed AL Shehri[2], Raouf M. Afifi[3,4*] and Mohamed A. Tashkandi[5,6]

[1]Department of Preventive Medicine, Armed Forces Hospital at Wadi Al-Dawasir, Kingdom of Saudi Arabia.
[2]Department of Family Medicine, Armed Forces Hospital at Wadi Al-Dawasir, Kingdom of Saudi Arabia.
[3]Community Health Research Institute, International Management-Health Services, Indianapolis, Indiana, USA.
[4] Board of Community Medicine Program, Armed Forces Hospitals, Taif, Kingdom of Saudi Arabia.
[5]Department of Preventive Medicine, Directorate of Health Affairs, Makkah, MOH, Kingdom of Saudi Arabia.
[6]Saudi Board of Preventive Medicine Program, Taif, Kingdom of Saudi Arabia.

Authors' contributions

This work was carried out in collaboration between all authors. Author AES led field data collection, interviews, data entry, participated in study instruments designing and preliminary data analysis. Author AAS participated in study protocol, furnished study approvals, shared in final report review, and supervised participants' screening and follow up. Author RMA designed study plan, wrote protocol, literature research, methodology plan, conducted statistical analyses, result display and discussion, and wrote final report draft. Author MAT shared in literature review, referencing, data entry, and shared in final report draft reviews. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/CA/2017/32180
Editor(s):
(1) Gen-Min Lin, Division of Cardiology, Hualien-Armed Forces General Hospital, National Defense Medical Center, Taiwan.
Reviewers:
(1) Miluda Rajab Elhamadi, University of Tripoli, Libya.
(2) Simona Lattanzi, Marche Polytechnic University, Italy.
(3) Pratima Tripathi, Sri Sathya Sai Institute of Higher Learning, India.
Complete Peer review History: http://www.sciencedomain.org/review-history/18683

Original Research Article

Received 12th February 2017
Accepted 8th April 2017
Published 18th April 2017

ABSTRACT

Background: Follow-up of patients destined to develop primary hypertension (HTN) demonstrates that blood pressure (BP) readings gradually increase over time.
Aim: Determine and analyze the prevalence and correlates of both pre-HTN and HTN among

**Corresponding author: E-mail: raoufafifi@hotmail.com;*

recruits serving in Wadi Al-Dawasir (WD) military district, central Saudi Arabia.
Methodology: Part of a "community diagnosis" plan led by the preventive medicine and family departments of the Armed Forces Hospital- WD (AFHWD), recruits were screened. A predesigned questionnaire and clinical interview were used to achieve study aim.
Results: The median age of participates was 34y (IQR 11.75); median systolic blood pressure (SBP) 120 mmHg (IQR 20), and diastolic BP (DBP) 80 mmHg (IQR 15). In screening, 531 recruits, all male, were surveyed. Forty-nine percent (253/516) were found with SBP 120-139 mmHg, compatible with "prehypertension;" 208/ (82.2%) of them were unaware they have prehypertension. Also, 12.2% (63/516) were found with SBP≥140 mmHg, meeting HTN diagnosis, 42 (66.7%) of whom were newly diagnosed. The participants' body mass index (BMI) averaged 27.6±5.4 kg/m^2, and 66.4% participants were overweight-obese. Abnormally high BMI levels were significant risk for high BP [Fisher's exact 64.6, p<0.0001]. "Now-smokers accounted 17.0% (n= 81); smoking impacted their hypertension (42.9%) and pre-hypertensive (25.4%) states [χ^2(df 2)=6.5, p=0.039]. Age significantly impacted BP level [χ^2(df 2)=14.3 p=0.001]; same as education [Fisher's exact 17.8, p=0.03]. Importantly, the recruits' SBP level differed between random plasma glucose (RPG) groups [U=4745, p=0.002]. Among chronic-disease comorbidities, having diabetes mellitus (DM) was significantly associated with hypertension (OR 2.93, 95% CI 134.6-637.6). Dyslipidemia also impacted high BP reporting [Fisher's exact =10.6, p=0.004]. The presence of family history of coronary heart disease (CHD) was significantly related to HTN among participants [χ^2(df 2)= 14.9, p=0.001].
Conclusions: Prehypertension, virtually the undiagnosed, is alarmingly prevalent in this study's population; hypertension, too, is less likely present. Most hypothesized risks were significant high BP correlates. With current insight, the main focus should be directed first to high BP and comorbid risks control; and continued screening to evaluate the effectiveness of intervention approaches on the recruits' lifestyle modifications and the impact of treatment policy on minimizing the risk of subsequent cardiovascular, stroke, and other systemic complications.

Keywords: Pre-hypertension; hypertension; Wadi Al Dawasir; Saudi Arabia.

1. INTRODUCTION

Hypertension (HTN), also known as high blood pressure (BP) is a chronic disease overwhelming healthcare systems worldwide. The condition is present when blood flows through the blood vessels with a force greater than normal. Knowingly, high BP can strain the heart, damage blood vessels, and increase the risk of heart attack, stroke, kidney problems, and death [1]. In many countries, 50% of the population over 60 has high BP. Clinically, HTN diagnosis is met with as repeatedly elevated BP exceeding 140/90 mmHg. The prevalence of hypertension is steadily increasing, even with the expanded use of antihypertensive medications [2]. It is widely recognized that hypertension is associated with increased cardiovascular disease (CVD) and all-cause mortality independently of other risk factors [3]. Studies have established association of HTN with "target organ damage" (TOD), such as increased left ventricular mass [4] and arterial wall changes [5] and screening for TOD has become an established practice in CVD preventive care [6]. Recent data suggest that TOD may begin at pre-hypertensive levels of BP [7].

1.1 Etiology and Pathophysiology of High BP

There is still uncertainty about the pathophysiology of HTN. The pathophysiology and etiology of hypertension are the two aspects of the group of vascular changes and response mechanisms incriminated in the development of hypertension [8]. Whereas some high HTN presentations have an underlying renal, neurologic, endocrinal or adrenal background for associated BP elevation, no clear single identifiable cause is found in the remaining hypertension states, in which case the term "essential hypertension" was traditionally given [9]. The multitude of factors contributing to high BP in hypertensive patients includes salt intake, obesity-insulin resistance, renin-angiotensin system (RAS), and the sympathetic nervous system. Locally, the endothelial layer of blood vessels produces a range of compounds, such as nitric oxide (NO) and "endothelin" both influence blood flow through regulating vascular tone and BP [10]. Evidence suggests that oxidant stress and "reactive oxygen species" (ROS) alter the endothelial modulation of vasomotor tone through inactivation of NO in high BP

conditions [11]. In addition, coagulation and fibrinolytic pathways may also be affected as a result of endothelial damage and subsequent vascular reactivity [12]. Angiotensin II is another factor thought to enhance formation of oxidant superoxide at concentrations that affect BP. (Altered endothelial functions are a reliable indicator of TO damage and atherosclerotic disease and prognoses). Diabetic "macrovascular" complications (involving medium-size- and also - large blood vessels) are triggered and get worsened by chronic hyperglycemia through many metabolic and structural derangements, including the production of advanced glycation end products (AGE),and abnormal activation of signaling cascades, such as protein kinase C (PKC) [13]. Because of impaired "baroreflexes" and stiffer arteries, patients with DM have higher BP variability than non-diabetic people [14]. Further, people with diabetes mellitus (DM) frequently have many traditional risk factors for CVD and HTN, including central obesity, dyslipidemia. The combination of metabolic risk factors including central adiposity, dyslipidemia (high triglycerides [TG] level and low fasting high density lipoprotein [HDL] level),inappropriately high plasma glucose [IHPG], and HTN in the general population is termed "metabolic syndrome" [15]. These factors, along with the independent risk factor of diabetes, can act both independently and cumulatively over time to significantly increase risk for CVD. Genetics may also play a role, whereas mutation involving a number of genes causing some Mendelian forms of high BP, initiated by altered renal salt handling, has been identified [16]. More recently, association results for BP could identify 31 new loci, in addition to 39 previously reported loci [17]. The identified variants implicate biological pathways related to cardio-metabolic traits, vascular function, and development. Genetic risk scores constructed from the identified variants were strongly associated with coronary heart disease (CHD) and myocardial infarction (MI). This large collection of BP-associated loci suggests new therapeutic strategies for HTN, emphasizing a link with cardio-metabolic risk [18]. Amidst such complex mechanisms respective to high BP, life style stands as a common denominator of hypertension pandemic. In essence, chronic diseases of the time (referred to as non-communicable diseases [NCDs]), including CHD, DM, dyslipidemias, metabolic syndrome, start to emerge after long exposure to unhealthful lifestyle, e.g., high-calorie diet, high salt intake, lack of regular physical activity (PA), and tobacco use [19]. This brings about risks that can act independently and synergistically, such as hypertension, obesity, and diabetes [20,21]. Among all, HTN is an increasingly important medical and public health problem. The worldwide estimates for the prevalence of HTN reaches upto 1 billion individuals, and approximately 9 million deaths per year may be attributable to HTN [22].

In 2003, the seventh "Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure" (JNC7), issued guidelines for the prevention and treatment of hypertension in hopes that earlier detection and better control of high BP will reduce rates of myocardial infarction (MI)and stroke and save lives [23,24]. However, JNC7also acknowledges that patient motivation to comply with effective treatment is crucial for optimal management, and that public health measures are also vital. According to the JNC7, 30% of adults may be unaware of their hypertension, 40% of individuals with HTN are not on treatment, and over 65% of hypertensive patients are not being controlled to BP levels less than 140/90 mmHg. Further, the decline in CHD and stroke-associated deaths in the western world since the 1970s and 1980s has plateaued, or even reversed in the past decade [25]. In addition, the prevalence and hospitalization rates of heart failure (HF) where in the majority of patients, who have HTN prior to developing HF have continued to increase. Likewise, hypertension is second only to diabetes as the most common antecedent for this condition [26]. Zhang and colleagues showed that patients with not-at-goal HTN bear a heavy burden of CV disease [27]. These patients had an estimated 10-year risk of CV events of 25.3% and a 10-year risk of CV death of 6.8%. Achieving adequate BP control can reduce morbidity and mortality from CV diseases. A 5-mmHg reduction in systolic BP (SBP) is estimated to result in a 14% overall reduction in mortality due to stroke, a 9% reduction in mortality due to coronary heart disease, and a 7% decrease in all-cause mortality.

Generally, the prevalence of HTN increases with advancing age; More than half of people 60–69 years of age are affected [28,29]. On the other hand, the long-term risk of HTN is best summarized by the lifetime risk statistic, which is the probability of developing HTN during the remaining years of life. In the Framingham Heart Study, investigators reported that the lifetime risk

of HTN was approximately 90% for men and women who were non-hypertensive at 55 or 65 years and survived to age 80–85 [30]. Even after adjusting for competing mortality, the remaining lifetime risks of HTN were 86% - 90% in women and 81% - 83% in men. Specifically, age-related rise in SBP [31], coupled with the time-bound nature of the hypertensive phenomenon urged for adopting an early prevention strategy to mitigate the progression of BP in high risk individuals to hypertensive level. The term "pre-hypertension" was first introduced by JNC7, as for SBP level 120-139 mmHg and diastolic BP level of 80-89 mmHg in its 2003 report [24].These cutoffs were further reaffirmed by the American and International Societies of Hypertension (ASH/ISH) [32].

1.2 Lessons from JNC7 Guidelines

Prior to the time of JNC7 work, alongside with the improved awareness of HTN from a level of 51% in 1976 - 1980 to 70% in 1999–2000 in USA, an increase in anti-HTN treatment rate from 31% to 59%had been reported, associated with a similar increase in the control rate of HTN to below 140/90 mmHg from 10% up to 34%, paralleled by an improvement in age-adjusted death rates from stroke and CHD by approximately 60% and 50%, respectively. These gains have occurred independent of gender, age, race, or socioeconomic status. Committed to evidence-based practice in providing basis for the prevention and management of hypertension, JNC7 reported that a better treatment of HTN was associated with a considerable reduction in hospital case-fatality rate of HF within the last three decades [23]. Although JNC7's report largely serves as a guide, the committee emphasizes that responsible physician's judgment remains paramount; that is positive experience, trust in the clinician and empathy improve patient motivation and satisfaction. key messages of the JNC7 report are: a) in those older than 50, SBP of >140 mmHg is a more important CVD risk factor than diastolic BP (DBP); *b)* pre-hypertensive individuals require health promoting lifestyle modification to prevent the progressive rise in BP and CVD; and *c)*for uncomplicated HTN, the report delineates specific high-risk conditions which are compelling indications for the use of other antihypertensive drug classes (angiotensin-converting enzyme inhibitors [ACEIs], angiotensin-receptor blockers[ARBs], calcium channel blockers [CCBs]); Two or more medications will be required to achieve goal BP,<140/90 mmHg SBP or <130/80 mmHg DBP in diabetics and chronic kidney disease [CKD]. The report stresses that meticulous clinical trials to scrutinize the impact of non-pharmacological therapies upon BP and CVD events in patients with HTN, e.g., over periods up to 1 to 2 years showed that interventions, such as weight reduction in overweight patients [33], increased PA [34], sodium reduction [35] and potassium supplementation [30], decreased alcohol intake, and stress management [36], all can lead to variable reductions in BP ranging from 2 to 15 mmHg [23].

In the Kingdom of Saudi Arabia (KSA), the local situation regarding the patternsand risks of hypertensive diseases is no departure from that described elsewhere. In an epidemiological study to investigate the prevalence and risks of elevated BP, 25.2% - and 43.0% - of Jeddah school teachers were found with HTN and pre-HTN, respectively; only 30.4% of those with HTN were aware of their condition. Hypertensive individuals also had either abnormally high BMI and or were older than normotensive peers [37]. More recently in Abha, KSA, one year after attending a newly established health promotion program clinic to prevent and control hypertension stressing on diet and PA counseling; 90% (n=429) of participants of the program failed to abandon imbalanced diet or to perform the recommended type of PA [38]. Further, 25% of the study subjects were overweight. Pre-HTN and HTN were therefore detected in 44% and 12% of the study population, respectively, and 21% of individuals had pre-diabetes. There is limited data regarding pre-hypertension among the Saudi military populations, those stationed in remote and non-metropolitan areas, such as that in Wadi Al-Dawasir, central Riyadh district, KSA. As we gather more evidence of high BP readings in asymptomatic adults, it might be imperative to screen military recruits with pre-hypertension to prevent CVD and future TOD. There are a multitude of reasons why hypertension should be particularly less prevalent in people in uniform. Hypertension per se often disqualifies for joining active duty jobs, where candidates are required to exhibit physical fitness; Which in turn expectedly minimizes the probability for morbid BP elevation. In view of the accentuated relationship and overlapping between hypertension and a number of risk factors related to demographic, body weight, diet, and comorbid conditions, and given the alarming reports on raised BP conditions among the Saudi adult

populations, a national task, such as WD military community needs to be surveyed and protected from high BP and its complications, to help keeping it away from CV risk during the course of the service and in the future.

2. METHODOLOGY

The authority of Wadi Al-Dawasir (WD) military district in collaboration with the preventive medicine and family medicine departments at the Armed Force Hospital- WD (AFHWD) has approved and launched a "community diagnosis" project to evaluate and promote health among WD recruits community. Wadi Al-Dawasir town is the largest populous gathering (68,201populations) which lies in WD valley in Najd desert, central Saudi Arabia, some 613 km south of Capital Riyadh [19]. The project adopts screening and prevention as most cost-effective approach for maintaining and improving the health status of WD enlisted recruits. In the first phase of the project in 2014, the prevalence of some NCDs of interest to WD populations, including CVD, bronchial asthma (BA), renal disease, hypothyroidism, liver disease, sickle cell disease (SCD), and malignancy were screened. In the current phase, the prevalence of pre-HTN and HTN, together with prediabetes/diabetes and associated health and behavioral trends were assessed. The dataset in this report is focused on BP status in association with hypothesized demographic and lifestyle factors, as well as some selected comorbidities. All military corps and affiliated camps and barracks in WD military district were covered by the study.

2.1 Data Collection, Population and Study Variables

A cross sectional approach was embraced to achieve study aim. A validated questionnaire modified from "Behavioral Risk Factor Survey" (BRFS) instrument [39] was used to screen the study population. All recruits enlisted in WD district were invited to the study; none was excluded because of demographic, health status or lifestyle variations. The questionnaire consists of 44 "items" (variables) under five scales: *a)* demographic criteria, e.g., age, income, education, *b)* risk factors, e.g., smoking and tobacco use, *c)* lifestyle- related factors, e.g., BMI, PA, *d)*health status and comorbidity, such as DM, dyslipidemia, and *e)* screening attitude, e.g., lipid profile and colonoscopy screening (for population at risk). The questionnaire takes 20-25 minutes to complete. Official approval from WD Headquarters and clearance from AFHWD management were granted. Returned questionnaires reporting valid answers on ≥80% of the items would be included in the analysis. A prior pilot administration was conducted and test-retest reliability score of the utilized questionnaire ranging between 0.72 and 0.93 was recorded [21]. On the field, recruits were addressed of the screening plan and study aim, and furthered with a health education session about hypertension and related risks, and the role of prevention in alleviating its burden upon individuals and the community. Participants were informed that enrolment in the screening plan was voluntary, and any participant could opt out of the study without negative implications on their health benefits or professional appraisal. Acceptance to take the questionnaire and submission to clinical exam (see next) was considered as an informed consent to participate in the study. The participants were reassured of the confidentiality of their collected information and only group results would be displayed in appropriate scientific settings. Upon completion of the questionnaire, each surveyee was invited to a clinical interview. Important socio-demographic data of the questionnaire include: age, as an interval ratio scale (IRS),marital status (binary: married single), monthly income in Saudi Riyal (SR =$0.27) (ordinal: ≤10,000- 20,000; >20,000-<30,000, ≥30,000), education (ordinal): ≤preparatory, secondary (high school equivalent), intermediate (postsecondary -less than baccalaureate, e.g., associate degree, professional or military training degree), and college degree or above (postgraduate education). Risk factors, lifestyle and health status criteria included: *a)*smoking daily (dichotomous: Yes/no),where a "smoker" is "one who is currently smoking daily; number of smoked cigarettes/day (IRS),and quit is one who "gave up smoking ≤5 years" prior to the survey [20], *b)*participation in any PA other than the regular job at least once during the last month (dichotomous); *c)* ever diagnosed with HTN (dichotomous), and ever diagnosed with any of the chronic diseases listed (dichotomous), such as DM, dyslipidemia and BA (all to be diagnosed and under treatment at a professional medical setting), and whether any of these conditions were present in the family. Most quantitative items, e.g., age, number of daily smoked cigarettes and duration of smoking, were reported first hand as in their original IRS form in the data collection instruments. These variables may often be converted to categorical forms to

conform statistical tests of interest, as need arises, for instance number of smoked cigarettes/day as an IRS variable could be stratified as: light (1- 5), moderate-1 (6-10), moderate-2 (11-15), heavy-1 (16-20), and heavy-2 (>20). (Defining cutoffs for smoking was modified from smoking behavior research data [40]and enlightened by previous researches on smoking risk and habits among Saudis [19,21,41]). (See later for clinical IRS variables stratification).

A proforma was designed to verify the health data collected from the clinical interview, including measurements such as *a)* weight, measured to the nearest of 0.1 kg on physician balance beam scales, with the subject wearing light clothes and with no shoes; all scales were identical and calibrated with a standard Inter ASIA protocol [42], *b)* height, measured without shoes to the nearest of 0.1 cm using a stadiometer, *c)* BMI in kg/m² was calculated as follows: as underweight <18.5, normal weight 18.5-24.9, overweight 25-29.9, stage-1 obesity 30-34.9, stage-2 obesity 35-39.9, morbid obesity ≥40) [43] (often underweight and normal weight may be collated in one BMI category (<20 - <25 kg/m²), *d)* waist circumference (WC) in cm, measured mid-way between the lateral lower ribs and the iliac crest, using tape measure (cutoff ≥102 cm used for central obesity), *e)* random plasma glucose (RPG) (mg%) by fingertip pinprick method (using Accu-Check® glucometer system from Roche, manufacturer SKU 04528280001, ADM ID: 3231, (cutoff for "prediabetes" was a RPG level≥200 mg% [11.1 mmol/l] with or without symptoms of diabetes, and without regard to time of last meal) [44],and *f)* BP (mmHg), measured at the right arm in sitting position, using mercurial sphygmomanometer (Sphygmomanometer MDS9410-Premier Aneroid-1999 Medline Industries, Inc., Mundelein, IL 60060, USA. www.medline.com 1-847-949-3150 RJ99NSS) and stethoscope [45]. Each subject was asked to take rest for at least 10 minutes in the supine position before recording BP. Another measurement of BP was taken after the interview, using the same sphygmomanometer and by the same health-worker [46]; the average of both measurement would be considered as the BP reading of each participant. Blood pressure was measured using cuff of a size appropriate to the arm circumference in accordance with the recommendations of the British Hypertension Society. [47] (The scales and sphygmomanometers were checked regularly). Hypertension in this screening was defined as "a SPB of 140 mmHg or higher or a DBP of 90mmHg or higher" [23]. Pre-HTN was defined as "a SPB of 120-139 mmHg or a DBP of 80-89 mmHg" [23].Eventually, each participant could be classified as either high - or low- risk for each HTN risk category, based on JCN7 staging of HTN. For instance, stage-1 HTN means BP in mmHg ranging from 140 to 159 systolic (or 90 to 99 diastolic); stage-2 means BP ranging from 160 to 179 systolic (or 100 to 109 diastolic), and stage-3 means BP equals to or greater than 180 systolic (or 110 diastolic) [48]. On our part, those with SBP 120-139 mmHg (or DBP 80-98 mmHg) would be labeled as "pre-hypertensive." We also tended to advocate SBP, not the DBP, measurement in the most part of the analysis, since SBP reportedly predicts CVD risks better than diastolic BP [36], particularly in those older than 50 [3]. Further, the presence of history of (H/O) HTN reported in the questionnaire would be confirmed from the patients' medical records available to AFHWD (see next). Subjects who attain high BP levels as per JNC7, could then be labeled as either "diagnosed" (known H/O pre-HTN or HTN, where applicable) or "undiagnosed" (unknown or "unaware" they have HTN or pre-HTN, where applicable). In this report, the latter group could also be called "newly diagnosed" [20].(Likewise, the term "pre-diabetes" would be given to those who had random blood glucose (RBG)≥200 mg% and were unaware of their PG condition) [19,21]. In parallel with receiving responses and the clinical interview, the participants medical records were accessed, upon permission, in order to match all reported health status data and clinical findings with the medical information on the records. Subjects with abnormal BP readings and those with any other health conditions, such as improper RPG levels or abnormal clinical findings of were referred to concerned family medicine clinics of AFHWD for confirmatory diagnoses and follow up.

2.2 Statistical Analysis Plan

Returned questionnaires reporting valid answers on ≥80% of the items were included. Data were entered to a Microsoft program with adequate back up until analyzed. Open-ended questions were coded, and observations were made ready for statistical analysis. Categorical data were described as count (%), while IRS data were summarized as range (minimum - maximum) and mean ± standard deviation (SD), or median and interquartile range (IQR), where appropriate, based on normality distribution. The latter could

be calculated using normality distribution techniques, such as one-sample Kolmogorov-Smirnov (K-S) test. Given the SBP variable nature, the analytical statistical plan was set forward so that SBP could first be tackled while in its initial its IRS form, as an intermediary outcome of interest. In which case, the effect of hypothesized risks on the variability in SBP level, not specifically the development of HTN or pre-HTN, could be calculated using statistical parametric techniques (PMTs); or the non-PMT alternatives, where appropriate. For example, *t*-test could be used to compare the mean difference among binary chronic disease groups, assuming normality distribution; otherwise Mann-Whitney *U* test would be more appropriate. Likewise, the mean differences in SBP among risk variables of three or more levels, e.g., income or BMI could be calculated using one-way analysis of variance (ANOVA) test, or Kruskal-Wallis (K-W) test based on normality assumption requirement. Second, SBP could be categorized as SBP<140 mmHg and SBP≥140 mmHg, where the relationship between the presence or absence of frank HTN (as a binary variable) and other risk categories, e.g., comorbidity or education could be calculated using chi-square (χ^2) test of independence (or Fisher's exact test, where appropriate). (*NB.* If suspected, the effect of a "confounding", e.g., age≥40y on any risk-outcome χ^2 calculation, adjusting for that confounding can be attempted through calculating both the crude odds ratio [OR] with the 95% confidence interval [CI] and adjusted OR [95% CI], e.g., age-adjusted OR, or smoking-adjusted OR, and then apply Mantel Haenszel summary OR statistic to estimate the difference and decide on confounding (summary OR_{MH}: OR-crude – OR-adjusted / OR-crude; if ≥10% so the adjustment variable can be confounding). Regarding smoking, if an initial χ^2 analysis based on dichotomous smoking exposure (yes/no) revealed significant results, no further analyses of ordinals smoking data would be needed. Handing SBP as a biphasic outcome as in the above provides us with the opportunity of early detection of risks related with the tendency for BP elevation in the study group. In which case, those risks could be obviated before time enough before the development of frank HTN or progressing to TOD. The SPSS software for Microsoft- version-20 was used for statistical analyses. Our level for tolerating alpha error was α=0.05, and results with p-values less than 0.05 were considered "statistically significant."

3. RESULTS

In this work, 531 participants turned in valid questionnaire responses. The median age of the studied group was 33.7 y (IQR 11.9) (Table 1a). Most (84.2%, n=431/512) participants were married. Intermediate education accounted 60.5% (305/504) of the recruits. Economically, most (66.3%, n=352) recruits were from the lowest income class. Table 1a also shows that most recruits were non-smokers, compared to smokers (395/476 =83.0; vs. 81/476= 17.0%, respectively). Among the latter, moderate smoking was dominant (40/81=49.3% of smokers), (and 8.4% of 476 valid observations). The participants' body mass profile was toward normal- to- overweight MBI (33.6% normal and 35.4% overweight). Obesity rate accounted total 31.0% (23.5% stage-1 obesity; 5.4% stage-2 obesity; 2.1% morbid obesity. We can conclude from Table 1a that 478/507 (94.3%) recruits had lowest RPG (<200 mg%) level, among whom 26 (5.1%) were known diabetic and under anti-diabetes therapy. Recruits with RPG ≥200mg% accounted 29/507 (5.7%): 23/507 (4.3%) known diabetic (uncontrolled) and 6 (1.2%) were newly diagnosed (Table 1a). Further description of BP distribution (Table 2) reveals that 253 out of 516 (49.0%) of the recruits had a SBP value within the pre-HTN range (120-139 mmHg), most of whom (208/253, 82.2%) did not have H/O HTN, so, newly diagnosed with pre-HTN. This group constitutes almost half (47.5%) of those with no H/O HTN. The remaining 45 (out of 253 pre-HTN subjects=17.8%) were known hypertensive, accounting the majority (57.7%) of all HTN group. Similar trend is observed in the hypertension level group, too, where most subjects (42/63, 66.7%) did not have H/O HTN (newly diagnosed HTN) (Table 2).

In Table 3a, most recruits 40 ys of age or above (59/128=46.1%) were within the pre-HTN zone; the rest were either normotensive (41/129=32%) or hypertensive (28/128=21.9%). The trend in BP levelsis fashioned that among the higher age category (≥40) the contribution to BP level elevation generally increases from the normal – to the high BP range; the opposite occurs in the younger age category (<40). Obviously, 21.9% (28/64) of hypertension observations is contributed by participants 40 years old or above vs. 9.2% (36/64) hypertension observations by younger peers. On the other hand, 41.2% of normal BP observations are contributed by <40 ys subjects vs. 32.0% normal BP observations by older peers.

Table 1a. Participants' demographic, risk and physiological criteria (n = 531)

Category	Characteristic/measurement	n (%)	% total valid	Total valid/missing n (%)
Age(y)	Range:33 (20 min.; 53 max.)			
	Mean ±SD: 33.7 ±7.5	--------	531/331 (100.0)	531(100.0)
	Median(IQR): 33.7(11.9)			
	K-S: Z=2.1, p=0.01			
	<40y	391 (75.7)	391 (75.2)	520 (97.9)
	≥40y	129 (24.3)	129 (24.8)	11 (2.1) missing
Marital status	Single	81 (15.2)	81/512 (15.8)	512(96.4)
	Married	431 (81.1)	431/512 (84.2)	19(3.6%) missing
Education	≤Preparatory	26 (4.9)	26/504 (5.1)	
	Secondary	82 (15.3)	82/504 (16.3)	504(94.9)
	Intermediate	305 (57.6)	305/504 (60.5)	27(5.1) missing
	College/postgraduate	91 (17.2)	91/504 (18.1)	
Income	<10,000 - <20,000	352 (66.3)	352/531 (66.3)	
level(SR)	>20,000 - <30,000	167 (16.7)	167/531 (16.7)	531(100.0)
	≥30,000	12 (2.3)	12/531 (2.3)	
Smoking*	Light smoker-$_{1,2}$	15 (2.8)	15/476 (3.1)	
	Moderate smoker-$_{1,2}$	40 (7.5)	40/476 (7.4)	476(89.6)
	Heavy smoker-$_{1,2}$	26 (4.9)	26/476 (5.5)	55(10.4) missing
	Nonsmoker	395 (74.4)	395/476(83.0)	
BMI (Kg/m^2)	20 - <25(normal)*	176 (33.1)	176/523 (33.6)	
	25 - <30 (overweight)	185 (34.8)	185/523 (35.4)	523(98.5)
	30 - <35 (obesity-1)	123 (23.1)	123/523 (23.5)	8(1.53) missing
	35 - <40(obesity-2)	28 (5.3)	28/523 (5.4)	
	≥40 (morbid obesity)	11 (2.0)	11/523 (2.1)	
RPG (mg/dl)	<200 No H/O DM	452 (85.1)	452/507 (89.2)	
	<200 - H/O DM	26 (4.9)	26/507 (5.1)	507(95.5)
	≥200 - No H/O DM	6 (1.3)	6/507 (1.2)	24(4.5) missing
	≥200 - H/O DM	23 (4.3)	23/507 (4.5)	

** Light-$_{1,2}$: 15/81 = 18.2%; moderate-$_{1,2}$: 40/81=49.3%; heavy-$_{1,2}$: 26/81 = 32.1%; all =81/476(17.0%); Non-smoker: never smoked 363/395+quit 32/395. ** BMI <20kg/m^2 (underweight)=33(5.3%)*

Table 1b. Participants' risk and physiological criteria: continuous data distribution (n = 531)

	Range(Min.-Max)	Mean ±SD	Median (IQR)	Normality (K-S test: z, p-value)
BMI (Kg/m²)[a]	38.4 (15 - 53.4)	27.6 ±5.4	27.3(7.1)	1.0, p=0.27
WC (cm)[a]	82 (62 - 144)	94.7±13.3	94.0 (16.7)	1.04, p=0.23
RBG (mg/dl)[b]	359 (70 – 429)	118.5 ±45.2	106 (21)	4.6, p<0.0001
Smoking (y)[b]	29 (1 - 30)	11.6 ±7.6	10 (9)	1.1, p<0.0001
SBP (mmHg)[b]	98 (90-188)	120.7 ±13.7	120 (20)	3.6, p<0.0001
DBP (mmHg)[b]	75 (50 - 125	77.7 ±9.5	80 (15)	3.1, p<0.0001

[a]Normally distributed; [s]kewed

Table 2. Participants' SBP distribution by H/O HTN

SBP category(mmHg)	Descriptive	No H/O HTN	H/O HTN	Total	Notes
<120	n	188	12	200	Normal
	% in raw	94.0	6.0	100.0	
	% in column	42.9	15.4	38.8	
	% of total valid	36.4	2.3	38.8	
120 -139 *(Pre-HTN level)*	n	208	45	253	208 undiagnosed; 42 diagnosed
	% in raw	82.2	17.8	100.0	
	% in column	47.5	57.7	49.0	
	% of total valid	40.3	8.7	49.0	
≥140 *(HTN level)*	n	42	21	63	42undiagnosed; 42 diagnosed
	% in raw	66.7	33.3	100.0	
	% in column	9.6	26.9	12.2	
	% of total valid	8.1	4.1	12.2	
Total (n, % all study)		438 (82.5)	78 (14.7)	516 (97.2)	Missing 15(2.8%)

In the pre-HTN group, the opposite happens, where the frequency of SBP 120-139 mmHg observations is higher (49.6%) in <40 subjects, compared to 46.1% of pre-HTN observations among older peers *[χ^2(df 2)=14.3, p=0.001]* (Table 3a).In parallel, SBP levels significantly differed between the study's two age groups (Table 3a footnote) *[U=19540, p<0.001].* Education protects against elevated BP; that involvement in higher BP level decreases in ≥college recruits; the opposite occurs in <college-educated peers.

For example, 21.3% normotensives were ≥college-level educated v. 2.0% had preparatory-level education. Conversely, 16.2% normotensives were secondary education graduates compared to 15.1% pre-HTN and 20.6% HTN recruits holding intermediate degrees *[Fisher's exact 17.8, p=0.03]*(Table 3 a).

In Table 3b, the significantly stressing effect of risk factors on BP status is vividly clear. The gradual involvement into elevated SBP states by higher risk exposure states, compared with a milder involvement among lower risk exposure states is paramount. For instance, 42.9% of hypertensive individuals were current smokers, compared to 25.4% being pre-hypertensive and smoker. In contracts, 74.6% of normotensive were non-smokers and 57.1% BP peers who also do not smoke *[χ^2(df 2)=6.5, p=0.039].*

The frequency of being placed in the range of high SBP among the obese increases, in comparison to that among the non-obese, e.g., 3.4% normotensive individuals were level-2 obese, and up to 15.4% of hypertensives were also level-2 obese. Conversely, 49.3% of normotensive subjects had a normal BMI and only 9.2% of hypertensives had a similar BMI range (Table 3b) *[Fisher's exact 64.6, p<0.0001].* Within the HTN-group, 15.6% subjects had RPG≥200 mg%, compared to 5.4% pre-hypertensive- and only 2.5% normotensive individuals who had a similar RPG range.

Table 3a. Influence of hypothesized correlates on BP level: Age, income and education

Category	Descriptive	SBP category (mmHg)			Total	Statistic, p-value
		<120	120 - 139	≥140		
Age(y)*	n (% in raw)	161 (41.2)	194 (49.6)	36 (9.2)	391 (100)	
<40	% in column	79.7	76.7	56.3	75.3	χ^2(df 2)
	% of total valid	31.0	37.4	6.9%	75.3	=14.3, p=0.001
≥40	n (% in raw)	41 (32.0)	59 (46.1)	28 (21.9)	128 (100)	
	% in column	20.3	23.3	43.8	24.7	
	% of total valid	7.9	11.4%	5.4	24.7	
Total	n (%total valid)	202 (38.9)	253 (48.7)	64 (12.3)	519 (100)	
Monthly	n (% in raw)	129 (39.3)	162 (49.4)	37 (11.3)	328 (100)	
income(SR)	% in column	65.2	65.3	60.7	64.7	
1000-<2000	% of total valid	25.4	32.0	7.3	64.7	
20-<30	n (% in raw)	64 (38.3)	79 (47.3)	24 (14.4)	167 (100)	Fisher's exact
	% in column	32.3	31.9	39.3	32.9	2.3, p=0.68
	% of total valid	12.6	15.6	4.7	32.9	
≥30	n (% in raw)	5 (41.7)	7 (58.3)	0 (0)	12 (100)	
	% in column	2.5	2.8	0.0	2.4	
	% of total valid	1.0	1.4	0.0	2.4	
Total	n (%total valid)	198 (39.1)	248 (48.9)	61 (12.0)	507 (100)	
Education	n (% in raw)	4 (15.4)	16 (61.5)	6 (23.1)	26 (100)	
Preparatory	% in column	2.0	6.5	9.5	5.1	
	% of total valid	0.8	3.2	1.2	5.1	
	n (% in raw)	32 (39.0)	37 (45.1)	13 (15.9)	82 (100)	
Secondary	% in column	16.2	15.1	20.6	16.2	Fisher's exact
	% of total valid	6.3	7.3	2.6	16.2	17.8, p=0.03
	n (% in raw)	119 (38.9)	149 (48.7)	38 (12.4)	306 (100)	
Intermediate	% in column	60.4	60.8	60.3	60.6	
	% of total valid	23.6	29.5	7.5	60.6	
College	n (% in raw)	42 (46.1)	43 (47.3)	6 (6.6)	91 (100)	
and above	% in column	21.3	21.8	9.5	18.2	
	% of total valid	8.3	8.5	1.2	18.2	
Total	n (% total valid)	197 (39.0)	245 (48.5)	63 (12.5)	505 (100)	

** Mann-Whitney test: SBP in age groups: U=19540, p<0.0001*

Table 3b. Influence of hypothesized correlates on BP level and the presence of hypertension in studied recruits: Smoking, BMI, RPG

Category		SBP category (mmHg)			Total	Test statistic, p-value
	Descriptive	<120	120 - 139	≥140		
Smoking	n	60	97	16	173	
Non-smoker	% in column	60.6	74.6	57.1	67.3	χ^2(df 2)
	% of total valid	23.3	37.7	6.3	67.3	=6.5,
	n	39	33	12	84	p=0.039
Smoker	% in column	39.4	25.4	42.9	32.7	
	% of total valid	15.2	12.8	4.7	32.7	
Total	n	99	130	28	257	
	% of total valid	38.5	50.6	10.9	100	
BMI[a]	n	100	73	6	179	
20-<25*(normal)*	% in column	49.3	28.0	9.2	33.8	
	% of total valid	18.9	13.8	1.1	33.8	
	n	66	98	24	188	Fisher's
25<30*(overweight)*	% in column	32.5	37.5	36.9	35.5	exact 64.6,
	% of total valid	12.5	18.5	4.5	35.5	p<0.0001
30<35	n	29	71	23	123	
(obesity-1)	% in column	14.3	27.2	35.4	23.3	
	% of total valid	5.5	13.4	4.3	23.3	
35-<40	n	7	11	10	28	
(obesity-2)	% in column	3.4	4.2	15.4	5.3	
	% of total valid	1.3	2.1	1.9	5.3	
≥40	n	1	8	2	11	
(morbid obesity)	% in column	0.5	3.1	3.1	2.1	
	% of total valid	0.2	1.5	0.4	2.1	
Total	n (%total valid)	203(38.4)	261(49.3)	65(12.3)	529	
RPG[b,c]	n	198	247	54	499	
<200mg%	% in column	97.5	94.6	84.4	94.5	Fisher's
	% of total valid	37.5	46.8	10.2	94.5	exact 13.4,
	n	5	14	10	29	P=0.001
≥200mg%	% in column	2.5	5.4	15.6	5.5	
	% of total valid	0.9	2.7	1.9	5.5	
Total	n (%total valid)	203(38.4)	261(49.4)	64(12.1)	528(100)	

[a]*K-W test: SBP/BMI: H(4)=74.9, p<0.0001.* [b]*SBP/RPG: U= 4745, p= 0.002.*[c]*SBP/WC: U=17693, p<0.0001*

In contracts, 84.4% of the hypertensives had RPG<200 mg%, while 94.6% pre-hypertensive- and as many as 97.5% normotensive individuals, all had RBP<200mg% (Table 3b) *[Fisher's exact 13.4 P=0.001.* In parallel, SBP levels significantly differed in the study population's RPG groups *[U=4745, p=0.002], BMI groups [H(4)=74.9, p<0.0001], and WC groups[U=17693, p<0.0001]* (Table 3b footnote).

As in Table 4, the frequency of having HTN together with self-identified dyslipidemia is significantly greater than in the pre-HTN group, and both are greater than having normal SBP (10.6%, 9.5%, and 1.5%, respectively). Calculating to determine whether age was confounding revealed the following: ***a)*** $OR_{\text{-crude}}$ 7.0, 95% CI 28.1-532.6; ***b)*** $OR_{\geq 40y}$ 6.1, 95% CI 1.4 – 27.9 (significant); ***c)*** summary OR_{MH} calculation ($OR_{\text{-crude}}$ – $OR_{\geq 40y}$ / $OR_{\text{-crude}}$): (7.0 - 6.1)/7.0 = 0.12 = 12%, so confounding (Table 4 footnote). The opposite trend was found among the non-dyslipidemia group (89.4% HTN, 90.5% pre-HTN, 98.5% normal SBP, respectively) *[Fisher's exact =10.6, p=0.004].* The relationship between H/O DM and the subject's SBP status sustains the same pattern between RPG and SBP (Table 4). Hypertensive patients constitute a greater proportion of diabetes groups (HTN/DM 16.7%, pre-HTN/DM 11.6%, normal BP/DM 4.5%, vs. HTN/no DM 83.3%, pre-HTN/no DM 88.4$, normal BP/no DM 95.5%) *[χ^2(df 2)= 10.7, p=0.005]* ($OR_{\text{-crude}}$ 2.93, 95% CI 134.6-637.6; age adjusted $OR_{\geq 40y}$2.7, 95% CI 0.95 – 7.7, so age was not confounding; and therefore Mantel Haenszel summary OR_{MH} calculation was not needed) (Table 4 footnote). Unexpectedly, the relationship between the presence of CHD among individual subjects and HTN was not significant. However, the presence of CHD among the subject's family was significantly related to SBP level (20.0% of those

with HTN, 10.7% of those with pre-HTN, and 4.1% of those with normal SBP), all had family history (F/H) of CHD. At the same time, only 80.0% of those with HTN; 89.3% of those with pre-HTN, and as many as 95.9% of those with normal SBP did not have F/H of CHD *[χ^2(df 2)= 14.9, p=0.001].*

Table 5 shows that among all 78 hypertensive patients screened, 21 (26.9%) were not controlled (SBP≥140 mmHg). Among those who do not have H/O hypertensive, 42/438 (9.6%) were newly diagnosed (Table 5; also Table 2).

4. DISCUSSION

Globally, the magnitude and burden of CVD, including hypertension is terrifying. The British Heart Foundation called for an urgent improvement in high BP diagnosis frequencies, condemning the situation as "unacceptable." At least one in four adults has high BP, half of whom has not even received a diagnosis [49]. In the US, 30% of adults in the two last decades may be unaware of their HTN [24]; Where updated data indicate that about one in five adults was also unaware of having high BP and would not report having it [50]. Herein, we found 8.1% (42/516) of our recruits were unaware of their frank HTN. We also found that 26.9% (21/78) of the hypertensive were not controlled, compared to over 65% known hypertensives, and also 46% with uncontrolled HTN in updated US reports [50,51]. Although the hypertension picture in our population is generally less startling, prehypertension situation is. Moreover, about half (47.5%, n = 208/438) of the recruits with prehypertension are unaware they already have it, e.g., compared to 33.3% American adults who have prehypertension [52]. The relatively low range of BP elevation confining pre-hypertension stage (120-139 mmHg), e.g., compared to higher ranges as SBP 140-159 (stage-1 HTN)- or SBP≥160 (stage-2 HTN)-mmHg [48] often tempts to underestimate the true risk indigenous with prehypertension, probably this is why the JNC7 had created the term prehypertension among its risk priority analyses.

Table 4. Blood pressure and comorbidity status among studied recruits

Category	Descriptive	SBP category (mmHg)			Total	Statistic
		<120	120 - 139	≥140		p-value
DM[a]	n	190	220	50	460	
-ve	% in column	95.5	88.4	83.3	90.6	χ^2(df 2)
	% of total valid	37.4	43.3	9.8	90.6	= 10.7
+ve	n	9	29	10	48	p=0.005
	% in column	4.5	11.6	16.7	9.4	
	% of total valid	1.8	5.7	2.0	9.4	
Total	n (%total valid)	199 (39.2)	249 (49.0)	60 (11.8)	508	
CHD	n	193	241	56	490	
-ve	% in column	96.5	96.8	94.9	96.5	
	% of total valid	38.0	47.4	11.0	96.5	Fisher's
+ve	n	7	8	3	18	exact
	% in column	3.5	3.2	5.1	3.5	=0.78
	% of total valid	1.4	1.6%	.6%	3.5	p=0.74
Total	n (%total valid)	200 (39.4)	249 (49.0)	59 (11.6)	508	
CHD/Family	n	186	217	48	451	
-ve	% in column	95.9%	89.3%	80.0%	90.7%	
	% of total valid	37.4%	43.7%	9.7%	90.7%	χ^2(df 2)
+ve	n	8	26	12	46	= 14.9
	% in column	4.1%	10.7%	20.0%	9.3%	p=0.001
	% of total valid	1.6%	5.2%	2.4%	9.3%	
Total	n (% total valid)	194 (39.0)	243 (48.9)	60 (12.1)	497	
Dyslipidemia[b]	n	132	153	42	327	
-ve	% in column	98.5%	90.5%	89.4%	93.4%	Fisher's
	% of total valid	37.7%	43.7%	12.0%	93.4%	exact
+ve	n	2	16	5	23	=10.6
	% in column	1.5%	9.5%	10.6%	6.6%	p=0.004
	% of total valid	0.6%	4.6%	1.4%	6.6%	
Total	n (%total valid)	134 (38.3)	169 (48.3)	47 (13.4)	350	

[a]*Crude OR2.93, 95%CI134.6-637.6. Age-adjusted: $OR_{\geq 40\ y}$ 2.7, 95%CI 0.95 – 7.7; age not confounding.*
[b]*Crude OR7.0, 95%CI 28.1-532.6. Age-adjusted $OR_{\geq 40\ y}$ 6.1, 95%CI1.4 – 27.9; age is confounding*

Table 5. SBP level in relation to the awareness status of the study subjects of the presence or absence of HTN among them

Category	HTN awareness status*						Test statistic	p-value
	Diagnosed*		Undiagnosed		Total			
	n	%	n	%	n	%		
SBP≥140 mmHg	21	4.0	42	8.0	63	11.9	χ^2(df 1)	p<0.0001
SBP<140 mmHg	57	10.7	396	74.6	453	85.3	=17.6	
Total	78	14.7	438	82.6	516	97.2		

Mann Whitney test: SBP in HTN/non-HTN groups: U= 9483.5, p<0.0001

4.1 Updated BP Guidelines and the Study Findings

The new JNC8 guidelines are shaped around the view that a SBP goal <140 mmHg for most patients may have been unnecessarily low [53]. The rationale is to introduce new recommendations designed to promote safer use of ACEIs and ARBs. While the same definition of HTN did not change in the new JNC8 guidelines, the latter critically" raises target for people ages 60 and older from <140/90 limit to only below 150/90 mmHg. For people under 60, the cutoff remains at 140/90 mmHg. Prominently, JNC8 also opts that people with DM or CKD would rather be kept on BP readings below 140/90- not 130/80 mmHg. Notably, age 50 was given a special attention in JNC7 guidelines that SBP>140 mmHg could be more important CVD risk factor than diastolic BP, a view not fully supported by JNC8report. A shift to "more DBP-based goal" in young patients with a new diagnosis of HTN arguably leads to the use of fewer medications and may improve adherence and minimize adverse events (AEs) associated with low SBP, such as sexual dysfunction [54]. Tell present, critiquing JNC7 guidelines was barely focused on the reports' analytical background or stipulated BP cutoffs, but on the recognition that the reports were not being used to their maximum benefit. Comparatively, criticism of the new JNC8 guidelines, including voices from JNC8 panel members, involves core guidelines contents, especially the "more relaxed" systolic target for older people and chronic patients, except perhaps for those over 80 [55]. One more clear difference was the choice of initial drug in patients without compelling indications. For instance, where thiazides were recommended to be the initial choice in patients without compelling indications, no such recommendation has been made in JNC8 [53]. Unions of ranked professional institutions such as the American Heart Association (AHA) and American College of Cardiology (ACC) after thorough comparative review of JNC8 report have conducted comprehensive reviews and came up with their updated guidelines, emphasizing sticking with the 140/90 cutoff [56]. On our part, it would be hard to generalize any BP management approach or the same follow up standards for all patients. A treatment decision should be individualized; where the doctor-patient relationship comes into play, and based on each patient's presentation and clinical judgment, considering interacting risks, genetic and environmental factors, and health condition. Bottom line, more time is needed to observe and conclude reliable results from the JNC8 guidelines, and many healthcare systems, including this screening program would feel they are committed to JNC7 goals beside compiling their own HTN treatment policies, aided agreed-upon guidelines, best practice and own experiences. And, now that there is lack of consensus about raising the systolic threshold, for instance if a patient was between 60 and 80 years of age, the attending physicians would rather work with them to get SBP below 140 mmHg, first via lifestyle modifications and, if that was not sufficient, medication [23,24,41,55]. However, if SBP was only brought down to target and there was a low risk for CVD, or if there was an increased chance that intensifying drug therapy will cause AEs, SBP between 140 and 150 might be acceptable. For those over age 80, the higher cutoff really does place them in the safest health zone, since it usually takes more intensive treatment to get SBP to less than 140 at that age and they are more likely to experience AEs or interactions with other drugs [55], and so forth. The number of newly diagnosed hypertensive recruits among our study population (42/516=8.1%) represents 66.7% (42/63) of all hypertensive recruits, a ratio that still raises a concern about BP health awareness of WD community, and probably the investment in screening and education of risk groups on the implications of high BP on health and quality of life (QOL). Citing lack of improvement in HTN

awareness elsewhere, treatment rates in the USA, for instance, had increased during the past two decade by less than 10%, and control rates were only 34%, falling short of the Healthy People 2010 goal of 50% [24]. As of 2013, about 75 million American adults (32%) have high BP, (that's 1 in every 3 adults, same as pre-HTN estimate) [49]. With those disappointments in mind, the role of comprehensive programs promoting lifestyle modification, environmental and public health measures, and early clinical intervention is emphasized. In spite of the thoroughness and level of evidence of ranked research that stipulate robust treatment options for HTN of variable severity and risks [57], such treatments would control HTN mostly if patients were motivated to be compliant with treatment recommendations.

4.2 High BP Prevalence

Ultimately, the study group's BP profile (median: SBP120.7, IQR 20 - and diastolic 80 mmHg, IQR 15) was around high normal- to- prehypertension range. In the second phase of a chronic disease screening plan in Taif military district, west KSA, 117 servicemen were surveyed [41]. The median BP level of Taif recruits was SBP 125 (IQR 10) and DBP 80 (IQR 10) [41]. The difference in SBP level between the two studies may well be related to higher age range of Taif population (median: 45y, IQR 10 in Taif group vs. 33.7y, IRQ 11.9y in WD). Literally, SBP tends to be higher with progressing age, and that is why it is used as a preferred guideline for BP monitoring, than DBP in ≥50y old people [23]. Further, Taif population was rather riskier with respect to the recruits' BP make-up, where 20.5% of recruits were hypertensive and 59.8% were pre-hypertensive, compared to 12.2% (63/516) and 49.0% (253/516) of WD population, respectively. In Taif military district study, too, the vast majority (91.7%) of hypertensive patients were unaware they had HTN and in WD 66.7% were unaware of their high BP state. In the first phase of the screening plan of Taif district in 2008, 19.1% (237/1,238) recruits of all adult age range were found hypertensive [58], most of whom (91.6%=217/237; and also =17.5% / total) were newly diagnosed. The very apparently "low" pre-HTN rate in Taif 2008 study, compared to same district 2013 study (59.8%) and also compared to ours (49.0%) is attributed to BP cutoffs and definitions of hypertension used in the 2008 Taif study, which was ≥140/90 mmHg (in the 2008 Taif study, no 120-139/80-89 mmHg was included in BP classification policy). In Jeddah, Saudi Arabia, 25.2% of screened school teachers were found hypertensive; 69.6% of whom were unaware they had high BP, and also 43.0% were pre-hypertensive [37]. Internationally, statistics document a variety of high BP rates, comparable to the Saudi's. In the Department of Health's 2010 "Health Survey for England" the prevalence of HTN in people 16 years or older was 31.5% in men and 29.0% in women [59].

4.3 Obesity and Age in Relation with High BP

We found that higher age groups (≥40) significantly contribute to high BP (21.9% vs. 9.2% HTN in the <40y old group). However, young recruits tended to contribute to pre-HTN events more prominently (49.6%) than older counterparts (46.1%). The effect of age on raising the BP in high risk people is widely endorsed [28,41]. Chronologically, the progression of essential HTN begins with pre-HTN in persons between 10-30y (with cardiac output overload) and then progresses to early HTN at 20-40 (with remarkably increased peripheral resistance), then to established HTN by 30-50, and finally to complicated HTN by 40-60ys of age [60]. Considering SBP in this screening program helps track recruits through more progressing age ranges efficiently and reliably. That CVD risk doubles per each BP 20/10 mmHg increment (from 115/75 mmHg), individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing HTN [23].Several other studies acknowledged the impact of age on BP level, whether TTN or pre-HTN [61], as well as the liability for CVD and TOD complications [4-7,24,61]. Part of a major national "Coronary Artery Disease in Saudis Study (CADISS)" project, Al-Nozha and colleagues revealed that the prevalence of overweight in Saudi households was 36.9%. Also age-adjusted obesity was as 35.5% prevalent [62]. Knowingly, the Saudi BMI profile sustains tendency for both overweight and obesity, which affect 37% -60% and 28.7% - 35.5% of adult populations, respectively (with some variation by sex, age and PA) [34]; the older the age the greater the BMI[63]. In comparison, more than one-third (35.4%) of our recruits are overweight, and just less than one-third (30.7%) are obese (BMI≥30 kg/m^2). In a cross sectional study on the prevalence of obesity and HTN in a large sample of healthy Pakistani army recruits [64], Karman and his colleagues found that 34.4% (n=763) soldiers were overweight, 35.4% were obese,

and 9.8% were found with HTN. The study utilized the BMI scale for Asian populations (overweight 23-24.9 kg/m^2, and obesity >25 kg/m^2) [65]. In the Pakistani army experience, too, 8.9% of the overweight (69/763) were significantly hypertensive; compared to 12.8% (24/188)of recruits in our study. Also, 15.1% (119/785) obese Pakistani soldiers were hypertensive, compared to 21.6% (35/162) of WD recruits obese and hypertensive. Although our recruits seem to have a relatively low HTN risk (12.2%), Pakistani comrades look less risky. In USA, screening records of service members who completed health risk assessments at a western states garrison (Fort Lewis in Tacoma, WA), were examined [61]. Out of 15,391 screened, 13% servicemen met definition for HTN, and 62% met that for pre-HTN, compared to 12.2% HTN and 49.0% pre-HTN of WD servicemen, respectively. Both age and BMI were significant correlates for the US soldiers' high BP readings, a finding comparable to that reported in our study. The authors concluded that HTN were more prevalent in the US troops than had been previously reported, and among whom HTN may be more common than in the general population. In contrast, pre-HTN was more prevalent in our troops (49.0%) than has been estimated in the Saudi general population in (40.6%) [66], 43.0% [37]; 44% [38]. To the best interest of our screening program, the prevalence of pre-HTN found in the young American troops (mean age 28y±3.7) suggests a need to better define the risks and benefits associated with the diagnosis and treatment of prehypertension in low-risk populations. Nationally, hypertensive individuals [37] also had abnormally high BMI and were significantly older than normotensive counterparts. In the 2010 Abha southern KSA study, pre-HTN and HTN were detected in 44% and 12% of study population, respectively [38]. Frequently, clinical measurement findings get inflated by the "alarm reaction" which occurs in some patients during physician visits; manifested as a transient increase in BP and has been frequently termed "white coat effect" [67]. Although its magnitude increases with the severity of hypertension, the impact of the white coat effect on the measured prevalence of hypertension is reportedly most pronounced in stage-1 disease. Because no estimation of the magnitude of the white coat effect in pre-HTN has been reported, the value reported for stage-1 HTN was conservatively applied in a sensitivity analysis attempt in the US army study to all pre- and stage-1 hypertensive readings [61]. The prevalence of HTN and pre-HTN were both found to be sensitive to this adjustment (6% vs. 13% for HTN and 48% vs. 62% for pre-HTN), although pre-HTN remained significantly prevalent. Notably, the American study used single un-averaged BP measurement the time of their survey; in which case the effect of alarm reaction on having inflated BP readings could be anticipated. Now that an averaged BP reading of two BP measurements taken during screening were used, we can argue against a significant effect of such alarm reaction on invalidating our study findings. Nonetheless, and for the sake of discussion, we could look up for "adjusted BP" rates to write off any residual inflation, if any, of our reported BP figures. The 49.0% pre-HTN figure might only be reduced to 38.0%, assuming the same adjustment rate (48% / 62%=77.4%) of the US study [61]. Doing so, the prevalence of pre-HTN in our population could be 38.0% (=49% * 0.774) a rate that is slightly less than the national 40.6%-44% rate. To this end, 38% of pre-HTN risk in such active duty population is still not too little. Overall, the comparative analysis of high BP frequencies between this work and other HTN epidemiological studies shows that WD servicemen still have relatively least risky BP status in comparison to national and also international figures. Nonetheless, pre-hypertension remains a stressing challenge, especially for young recruits; and more work has to be done to contain this threat.

4.4 Education and Recruits' BP Status

Interestingly, more educated recruits in WD were less likely to record high BP observation than less educationally fortunate peers. A leading European study was conducted on a large sample of hypertensive outpatients to evaluate whether educational level and the awareness of stage I-II HTN with TOD was education-related [68]. Evidently, most hypertensive patients were highly educated; contrary to our study, and also contrary to the finding that diabetics with little education significantly accounted 95% (n=190) of the European study population. Notably, most European hypertensive-highly educated patients worked at sedentary jobs. Otherwise, only DBP was independently associated with low educational level, in general agreement with our study findings. Public health education has a role in understanding and preventing not only HTN but all NCDs dimensions. For instance, it is rather easy to explain to audience the role of high consumption of olive oil and a high intake of vegetables, fruits, whole grains, legumes, fish, and nuts and limiting unhealthy fats in preventing

and mitigate hypertension and overlapping diseases, such as hypercholesterolemia, atherosclerosis, and ischemic heart disease (IHD). Our screening plan should be supported and continued on a long-term basis in order to protect the young and less educated through early detection and intervention for pre-HTN and HTN should be among AFHWD chronic disease prevention and control priorities.

4.5 Diabetes and RPG Level with Associated Risks in Relation to Recruits' BP Status

There is a large body of evidence on the "coupling" between diabetes and high BP [19]; given an established overlap between the two conditions in etiology and mechanisms [69]. Having diabetes can be followed by "vasculopathy" and atherosclerosis, and subsequently high BP [13,14,41]. Among our BP groups, 15.6% hypertensives significantly had RPG≥200mg%; 5.4% pre-hypertensives were similarly hyperglycemic. Having diabetes also contributed more frequently to HTN level BP, and less likely to pre-HTN in our population. The burden of diabetes upon most Saudi communities is devastating [41,62,63]. The time people are diagnosed with diabetes they frequently have developed severe complications linked to the macroangiopathic changes, which are a strong predisposing factor to HTN [69]. The fact that lifestyle risks, and medical and environmental diabetogenic factors and that diabetes leads to consequences, some of which are underlying triggers, warrants early intervention to interrupt the diabetes- HTN circle. The latter can lead to and make worse many complications of diabetes. Diabetes makes high BP problems more likely, because of the cascade of vasculopathic changes, stated above, ending up with disturbed vasoconstriction-vasodilatation mechanism and loss of blood vessels integrity and elasticity [13,14,41]. High BP if not treated, in turn leads to further blood vessels damage and worsening angiopathic-related complications of DM, including, IHD, renal failure, retinopathy and stroke [41]. When we first hypothesized for this research, we did not expect to get a direct cause-effect relationship between BP as an independent exposure and hyperglycemia as an outcome. However we were willing to understand the presence of a close link between both conditions. To that end, we learn that the significantly tight link between SBP and RPG/diabetes supports the perspective that patients with prediabetes/diabetes are likely to develop pre-HTN/HTN [41].

Central obesity as a result of excessive deposition of body fat around the waist is consistent with large body size levels (BMI), and is known cause for insulin resistance [19,21,41,71]. Therefore constitutes potential risk for diabetes. As before, many factors incorporated in the provision of "metabolic syndrome" components are experienced with variable degrees and severity between subsets of this study. The large WC group tends to have high SBP, compared to the smaller WC group. The same finding was reported in similar research [6]. Largely, BMI and WC sustain a common health risk behavior, and this is pretty much evidenced in metabolic syndrome and encompassed individual chronic disease conditions. Likewise, diagnosed dyslipidemia patients were more prone to either HTN or pre-HTN. Further, age ≥40 was confounding for this relationship. This ascertains the need to consider optimizing future preventive program objectives to variable age- risk groups. Evidence suggests that hypertension may share a similar pathophysiology with CVD; thus, dyslipidemia, a strong predictor of CVD, may also predict incident hypertension. Atherogenic lipid abnormalities clearly induce endothelial dysfunction [10], where a dysfunctional endothelium, possibly through impaired NO, production as well as alterations in endothelin-1, cannot respond to changes in intravascular conditions to constrict and dilate as needed. Because atherosclerosis can be a diffuse process, it is possible that hypertension is a manifestation of a diffuse atherosclerotic process in large conduit arteries, as well as smaller resistance vessels [10,70]. Age did not confound the relations between DM and HTN among our recruits, making diabetes more of an independent determinant for HTN. Luckily, recruits with high BP were not significantly prone to CHD reporting; despite the presence of CHD among their family members. Now that the hypertensive- and often the pre-hypertensive-patients were more prone to the multitude of hypothesized risks, namely obesity, large waistline, IHPG, and high cholesterol level, they are at a greater risk of developing resistant hypertension, especially if their hypertensive conditions were not controlled to below target [71]. In these patients, weight reduction not only improves BP control but brings about a considerable reduction in HTN complications of IHD, stroke and KD.

4.6 Smoking and Risk of HTN

Smoking significantly impacted both on HTN (43%) and pre-HTN (25%), despite a predominant light-moderate smoking attitude among recruits. In literature, even light smoking (<5 cigarettes/day) was associated with a significantly higher risk of dying from IHD, and also significantly dying from lung cancer and dying from all other causes [72]. (*NB.* Smoking does interfere with the prognosis of NCDs; however, analyzing for these risks, we did not feel the need to adjust for smoking because the number of smokers was rather little for cumbersome OR_{MH} comparisons). An analysis of the dose-response relationship based on combined data of passive smoking, particulate matter from air pollution, and active light and heavy smoking indicates that low levels of tobacco exposure as in light smoking (4–7 cigarette/day) has about 70% of the effect of heavy smoking (≥ 23 cigarette/day) [72]. In addition, the risk of IHD in light smoking (1–4 cigarette/day) men and women ages 35–39 was nearly three times that of a nonsmoker; while adult men who consume 6–9 cigarette/day also had a relative risk of 2.1 for MI compared to nonsmokers [73]. Eventually, smoking control policymakers and health educators should emphasize more strongly that light smoking also endanger their health. It is worthwhile to mention here that other factors, such as BP swings and fluctuation can be a sign of a severe deterioration of the blood vessel integrity and susceptibility to expedited cardiovascular complications. A large scale prospective study [74] on BP and associated risks control and treatment found that visit-to-visit variability (VVV) in SBP (and DBP) has been significantly associated with CVD and mortality. Apart from the well-known role of HTN in cerebrovascular disease, VVV of BP is emerging as an independent risk factor for stroke. Although the underlying mechanism is not fully understood, artery remodeling is thought to be closely involved in this relationship. Silent cerebral injury is considered to serve as a common pathophysiology in the relationship of visit-to-visit BP variability with cognitive impairment and stroke [75]. Likewise, higher visit-to-visit variability in blood pressure independent of average blood pressure was associated with impaired cognitive function in old age [76]. Thereby not only should hypertensive patients have their BP adequately controlled but they should be aware that any remarkable variations in their BP could be a sign of irreversible life-threatening vascular damage. The prospective nature of the relationship between BP fluctuation and liability to HTN morbidity and mortality is out of the scope of this work; yet, it could be a matter of future research. Some limitations have been encountered in this work. We were unable to endorse some HTN-risk indicators in the analysis, such as the current lipid profile or other chronic disease markers, due to some technical and time constraints. Nonetheless, the study has several strengths, such as the use of averaged BP reading and incorporating all WD military members in the screening. Also utilizing standardized data collection instruments and including F/H of relevant NCD widen the scope of the analysis to the best interest of more validity and generalizability of the study findings.

5. CONCLUSION

A diagnosis of HTN can have serious occupational implications. The group of risks, including hypercholesterolemia and insulin resistance, together with uncontrolled BP poses an eminent threat on the TOs' health. Diabetic-HTN among our population is rather notable. However, the prevalence of pre-HTN is up to four-times notable. Understanding common causes of these pathways allows more proactive approaches in maintaining good cardiovascular health of our recruits. Today, high-income countries have begun to reduce HTN in their populations through strong public health policies such as reduction of salt in processed food and population-based screening that tackle HTN and associated risks. This approach is similarly quite feasible in the Saudi military population setting. The accumulated insights would be crystallized into an action plan where significant reductions in the numbers of high BP conditions would be achieved. Further, ongoing screening and health education to keep up with the modifications in the recruits' lifestyle and the impact of treatment policies of bringing BP to acceptable levels would be among our population's health priorities.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. National Institute of Health. National institute of diabetes and digestive and kidney diseases. PubMed Health Glossary. Available:https://www.ncbi.nlm.nih.gov/pubmedhealth/PMHT0024199/
2. Burt LE, Culter VL, Hughes JA, J, Roccella EJ, Sorlie P. The burden of adult hypertension in the United States 1999 to 2000: A rising tide. Hypertension. 2004;44:398–404. DOI:0.1161/01.HYP.0000142248.54761.56
3. Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks: US population data. Arch Intern Med.1993;153:598–615.
4. Levy D, Anderson KM, Savage DD, Kannel WB, Christiansen JC, Castelli WP. Echocardiographically detected left ventricular hypertrophy: Prevalence and risk factors. The Framingham Heart Study. Ann Intern Med. 1998;108:7-13.
5. Roman MJ, Saba PS, Pini R, Spitzer M, Pickering TG, Rosen S, et al. Parallel cardiac and vascular adaptation in hypertension. Circulation. 1992;86: 1909-1918.
6. Rosendorff C, Black HR, Cannon CP, Gersh BJ, Gore J, Izzo JL Jr, et al. Treatment of hypertension in the prevention and management of ischemic heart disease: A scientific statement from the American heart association council for high blood pressure research and the councils on clinical cardiology and epidemiology and prevention. Circulation. 2007;115:2761-88.
7. Markus MR, Stritzke J, Lieb W, Mayer B, Luchner A, Doring A, et al. Implications of persistent prehypertension for ageing-related changes in left ventricular geometry and function: The MONICA/KORA Augsburg study. J Hypertens. 2008;26:2040–49.
8. Beevers G, Lip GYH, O'Brien E. The pathophysiology of hypertension. Br Med J. 2001;322(7291):912–916.
9. Carretero OA, Oparil S. Essential hypertension. Part I: Definition and etiology. Circulation. 2000;101(3):329-35. DOI: 10.1161/01.CIR.101.3.329
10. Oparil S, Zaman MA, Calhoun DA (November 2003). Pathogenesis of hypertension. Ann Intern Med. 2003;139(9):761–76. DOI:10.7326/0003-4819-139-9-200311040-00011
11. Cai H, Harrison DG. Endothelial dysfunction in cardiovascular diseases: The role of oxidant stress. Circulation Research. 2000;87(10):840-44.
12. Beevers G, Lip GYH, O'Brien E. ABC of hypertension. London: BMJ Books; 2007. ISBN 1-4051-3061-X
13. Cade WT. Diabetes-related microvascular and macrovascular diseases in the physical therapy setting. Phys Ther. 2008;88(11):1322-1335. DOI: 10.2522/ptj.20080008
14. Torp-Pedersen, Jørgen Jeppesen. Diabetes and hypertension and atherosclerotic cardiovascular disease related or separate entities often found together christian. Hypertension. 2011;57:887-888. Available:https://doi.org/10.1161/HYPERTENSIONAHA.110.168583 (cited 2011 April 20)
15. executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel iii). J Am Med Ass. 2001;285:2486–2497.
16. Corvol P, Persu A, Gimenez-Roqueplo AP, Jeunemaitre X. Seven lessons from two candidate genes in human essential hypertension: Angiotensinogen and epithelial sodium channel. Hypertension. 1999;33(6):1324-31. DOI: 10.1161/01.hyp.33.6.1324
17. Liu C, Kraja AT, Smith JA, Brody JA, Franceschini N, Bis JC, et al. Meta-analysis identifies common and rare variants influencing blood pressure and overlapping with metabolic trait loci. Nat Genet. 2016;48:1162–1170. DOI: 10.1038/ng.3660
18. National institute of health. National institute of diabetes and digestive and kidney diseases. PubMed Health Glossary. Available:https://www.ncbi.nlm.nih.gov/pubmedhealth/PMHT0024199/
19. Afifi RM, Omar, SR, El Raggal A. A community screening plan for the prevalence of some chronic diseases in specified adult populations: Pre-diabetes and diabetes mellitus. Int J Diab Dev Ctries. 2015;35:149. DOI: 10.1007/s13410-013-0189-0

20. Steven K, Damasceno A. Lifestyle and related risk factors for chronic diseases. Jamison DT, Feachem RG, Makgoba MW, Eduard R Bos, Florence K Baingana, et al. editors. Disease and mortality in Sub-Saharan Africa. 2nd ed. Washington (DC): World Bank. 2006;374-375.
21. Afifi R, Saad AE, Al Shehri A. Prevalence and correlates of prediabetes and diabetes results-I: A screening plan in a selected military community in central Saudi Arabia. J Diab Mell. 2017;7(1):12-30.
DOI: 10.4236/jdm.2017.71002
22. World Health Organization (WHO). A global brief on Hypertension. Silent killer, global public health crisis. World Health Day; 2013.
Available:http://ish-world.com/downloads/pdf/global_brief_hypertension.pdf
23. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. U.S. department of health and human services, national institutes of health, national heart, lung, blood institute, national high blood pressure education program; 2004.
Available:http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.pdf
(NIH Publication No. 04-5230)
24. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC7 report. J Am Med Ass. 2003;289:2560.
25. Jones DS, Greene JA. The decline and rise of coronary heart disease: Understanding public health catastrophism. Am J Pub Hlth. 2013;103(7):1207–1218.
DOI: 10.2105/AJPH.2013.301226.
26. Centers for disease control and prevention (CDC). Racial differences in trends of end-stage renal disease, by primary diagnosis. United States, 1994-2004.MMWR Morb Mortal Wkly Rep. 2007;23;56(11):253-6.
27. Zhang Y, Lelong H, Kretz S, Agnoletti D, Mourad JJ, Safar ME, et al. Characteristics and future cardiovascular risk of patients with not-at-goal hypertension in general practice in France: The Avant'age study. J Clin Hypertens (Greenwich). 2013;15:291–295.
28. Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, et al. Prevalence of hypertension in the US adult population. Results from the third national health and nutrition examination survey, 1988-1991. Hypertension. 1995;25:305-13.
29. Vasan RS, Larson MG, Leip EP, Kannel WB, Levy D. Assessment of frequency of progression to hypertension in nonhypertensive participants in the framingham heart study: A cohort study. Lancet. 2001;358:1682-6.
30. Vasan RS, Beiser A, Seshadri S, Larson MG, Kannel WB, D'Agostino RB, et al. Residual lifetime risk for developing hypertension in middle-aged women and men: The framingham heart study. J Am Med Ass. 2002;287:1003-10.
31. Franklin SS, Gustin W, Wong ND, Larson MG, Weber MA, Kannel WB, et al. Hemodynamic patterns of age-related changes in blood pressure. The framingham heart study. Circulation 1997;96:308-15.
32. Weber MA, Schiffrin EL, White WB, Mann S, Lindholm LH, Kenerson JG, et al. Clinical practice guidelines for the management of hypertension in the community a statement by the American Society of hypertension and the international society of hypertension. 2014;16(1):14-26.
DOI: 10.1111/jch.12237
33. Blumenthal JA, Sherwood A, Gullette EC, Babyak M, Waugh R, Georgiades A, et al. Exercise and weight loss reduce blood pressure in men and women with mild hypertension: Effects on cardiovascular, metabolic, and hemodynamic functioning. Arch Intern Med 2000;160(13):1947-58.
34. Cleroux J, Feldman RD, Petrella RJ. Lifestyle modifications to prevent and control hypertension. Recommendations on physical exercise training. Canadian hypertension society, Canadian coalition for high blood pressure prevention and control, laboratory centre for disease control at health Canada, Heart and Stroke foundation of Canada. Can Med Ass J [Internet]. 1999;160(9 Suppl):S21-8.
Available:https://www.ncbi.nlm.nih.gov/pubmed/10333850
(cited 1999 May 4)
35. Hooper L, Bartlett C, Davey Smith G, Ebrahim S. Systematic review of long term effects of advice to reduce dietary salt in adults. Br Med J. 2002;325(7365):628.
36. Batey DM, Kaufmann PG, Raczynski JM, Hollis JF, Murphy JK, Rosner B, et al.

Stress management intervention for primary prevention of hypertension: Detailed results from phase I of trials of hypertension prevention (TOHP-I). Ann Epidemiol. 2000;10(1):45-58.

37. Ibrahim NK, Hijazi NA, Al-Bar AA. Prevalence and determinants of prehypertension and hypertension among preparatory and secondary school teachers in jeddah. J Egypt Publ Hlth Assoc. 2008;83(3-4):183-203.
38. Al-Shahrani AM, Al-Khaldi YM. Experience of the health promotion clinics in Aseer region, Saudi Arabia. J Fam Community Med. 2011;18:130-4.
39. Behavioral Risk Factor Survey Module: Definitions of Survey Measures. Wisconsin BehavioralRisk Factors Survey. (Internet); 2014.
Available:http://www.dhs.wisconsin.gov/wish/main/BRFS/definitions.htm
40. Schane RE, Ling PM, Glantz SA. health effects of light and intermittent smoking: A review. Circulation. Published in final edited form as: Circulation. 2010;121(13): 1518–1522.
DOI: 10.1161/CIRCULATIONAHA.109.904235
41. Afifi R, Omar SR, Raggal A. A community screening plan for the prevalence of some chronic diseases in specified adult populations: pre-hypertension and hypertension. Intern J Biomed Res.2015;4(5).
DOI: http://dx.doi.org/10.7439/ijbr.v4i5.258
42. Rachel P Wildman, Dongfeng Gu, Kristi Reynolds, Xianfeng Duan, Jiang He. Appropriate body mass index and waist circumference cutoffs for categorization of overweight and central adiposity among Chinese adults. Original research communication. American Journal of Clinical Nutrition. 2004;i80(5):1129-36.
43. World Health Organization (WHO). Obesity: Preventing and managing the global epidemic. WHO Technical report series. Geneva. National center for chronic disease prevention and health promotion. Defining overweight and obesity. Overweight and obesity among adults. 2000;894.
Available:http://www.athealth.com/consumer/disorders/definingobesity.html
(Updated 2004 June 25).
44. American Diabetes Association (ADA). Screening for type 2 diabetes. Position statement. Diabetes Care 23. 2000;(Suppl. 1):S77S79.
Available:http://care.diabetesjournals.org/content/27/suppl_1/s11.full?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=screening+for+type+2+diabetes&searchid=1083098689392_7996&stored_search=&FIRSTINDEX=0&sortspec=relevance&journalcode=diacare
45. Williams JS, Brown SM, Conlin PR. Blood-pressure measurement. N Engl J Med. 2009;360(5):e6.
DOI: 10.1056/NEJMvcm0800157
46. Al Osaimi S, AL-Gelban KS. Diabetes Mellitus- prevalence and associated cardiovascular risk factors in a Saudi sub-urban community. Biomed Res. 2007;18(3):147-153.
Available:http://www.alliedacademies.org/articles/diabetes-mellitus--prevalence-and-associated-cardiovascular-risk-factors-in-a-saudi-suburban-community.pdf
47. British hypertension society. Technique of blood pressure measurement. J Hypertension. 1985;3:293-312.
48. The sixth report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med. 1997;157(21):2413-46.
49. British heart foundation. High blood pressure.
Available:https://www.bhf.org.uk/heart-health/risk-factors/high-blood-pressure
50. Farley TA, Dalal MA, Mostashari F, Frieden TR. Deaths preventable in the U.S. by improvements in the use of clinical preventive services. Am J Prev Med. 2010;38(6):600–9.
51. Centers for disease control and prevention (CDC). division for heart disease and stroke prevention. high blood pressure in the United States (Internet).
Available:https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_bloodpressure.htm
(Updated 2016 June 16).
52. Nwankwo T, Yoon SS, Burt V, Gu Q. Hypertension among adults in the US: National health and nutrition examination survey, 2011-2012. NCHS Data Brief. 2013;(133):1-8.
Available:https://www.ncbi.nlm.nih.gov/pubmed/24171916
53. James PA, Oparil S, Carter BL, Cushman WC, Dennison- Himmelfarb C, Handler J, Lackland DT, et al. Evidence-based guideline for the management of high

blood pressure in adults: Report from the panel members appointed to the Eighth Joint National Committee (JNC 8). J Am Med Ass. 2014;311:507–520.
DOI: 10.1001/jama.2013.284427

54. Page MR. The JNC 8 hypertension guidelines: An in-depth guide. Pharmacy times. Practical information for today's pharmacists (Internet). USA; 2014.
Available:http://www.pharmacytimes.com/news/the-jnc-8-hypertension-guidelines-an-in-depth-guide?p=1
(cited 2016 Nov 11)
55. Jones DS, Greene JA. The decline and rise of coronary heart disease: Understanding public health catastrophism. Am J Publ Hlth. 2013;103(7):1207–1218.
DOI: 10.2105/AJPH.2013.301226
56. Rosendorff C, Lackland DT, Allison M, Aronow WS, Black HR, Blumenthal RS, et al. American heart association (AHA), American college of cardiology (ACC), and American society of hypertension (ASH). Treatment of hypertension in patients with coronary artery disease. A scientific statement from AHA/ACC/ASH. hypertension. Hypertension. 2015;65:000-000.
DOI: 10.1161/HYP.0000000000000018
57. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: A report of the American college of cardiology/American heart association task force on practice guidelines. Circulation. 2014;129(25 Suppl 2):S76-99.
DOI: 10.1161/01.cir.0000437740.48606.d1
58. Al-Asmary SM, Al-Shehri AA, Farahat FM, Abdel-Fattah MM, Al-Shahrani MM, Al-Omari FK, Al-Otaibi FS, Al-Malki DM. Community-based screening for pre-hypertension among military active duty personnel. Saudi Med. J. 2008;29(12):1779-84.
59. Statistical Information on Cardiovascular Disease in the UK; 2014.
Available:http://bhsoc.org/resources/statistical-information-on-cardiovascular/
60. Dreisbach AW. Pathophysiology of Hypertension. Medscape; 2015.
Available:http://emedicine.medscape.com/article/1937383-overview
61. Smoley BA, Smith NL, Runkle GP. Hypertension in a population of active duty service members. J Am Board Fam Med. November-December. 2008;21(6):504-511.
DOI:10.3122/jabfm.2008.06.070182
Available:http://www.jabfm.org/content/21/6/504.full]
62. Al-Nozha MM, Al-Mazrou YY, Al-Maatouq MA, Arafah MR, Khalil MZ, Khan NB, et al. Obesity in Saudi Arabia. Saudi Med J. 2005;26(5):824-9.
63. Memish Z, El Bcheraoui C,Tuffaha M, Robinson M, Daoud F, Jaber S. Obesity and associated factors -Kingdom of Saudi Arabia, 2013. Prev Chronic Dis. 2014;11:E174.
DOI: 10.5888/pcd11.140236
64. Kamran SM, Ftikhar R, Roshan R. Frequency of obesity and hypertension in armed forces: it is time to face reality. Pak Arm Frc Med J. 2016;66(0):S36-S40.
65. Alberti, KG, Zimmet, P, Shaw, J. The metabolic syndrome--a new worldwide definition. Lancet. 2005;366:1059.
66. El Bcheraoui C, Memish ZA, Tuffaha M, Daoud F, Robinson M, Jaber S, et al. Hypertension and its associated risk factors in the Kingdom of Saudi Arabia, 2013: A national survey. Int J Hypertens. 2014;(2014):8.
(Article ID 564679).
DOI: 10.1155/2014/564679
67. Mancia G, Parati G, Pomidossi G, Grassi G, Casadei R, Zanchetti A. Alerting reaction and rise in blood pressure during measurement by physician and nurse. Hypertension 1987;9:209–15.
68. Tedesco MA, Di Salvo G, Caputo S, Natale F, Ratti G, Iarussi D, et al.Educational level and hypertension: How socioeconomic differences condition health care.J Hum Hypertens. 2001;15(10):727-31.
69. Cheung BMY and Li C. Diabetes and Hypertension: Is there a common Metabolic Pathway? Curr Atheroscler Rep. 2012;14(2):160–166.
DOI: 10.1007/s11883-012-0227-2
70. Nickenig G, Harrison DG. The AT(1)-type angiotensin receptor in oxidative stress and atherogenesis: Part II: AT(1) receptor regulation. Circulation. 2002;105:530–536.
71. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, et al. Resistant hypertension: Diagnosis, evaluation, and treatment. A scientific statement from the American heart association professional education committee of the council for high blood pressure research. Hypertension. 2008;51(6):1403-19.

DOI:10.1161/HYPERTENSIONAHA.108.189141

72. Pope CA, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ. Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: Shape of the exposure-response relationship. Circulation. 2009; 120(11):941-948.
73. Bjartveit K, Tverdal A. Health consequences of smoking 1–4 cigarettes per day. Tob Control 2005;14:315-320. DOI: 10.1136/tc.2005.011932
74. Muntner P, Whittle J, Lynch AI, Colantonio LD, Simpson LM, Einhorn PT. Visit-to-visit variability of blood pressure and coronary heart disease, stroke, heart failure, and mortality: A cohort study. Ann Intern Med. 2015;163(5):329-338. DOI: 10.7326/M14-2803
75. Nagai M, Kario K. Visit-to-visit blood pressure variability, silent cerebral injury, and risk of stroke. Am J Hypertens. 2013;26(12):1369-1376. DOI: 10.1093/ajh/hpt167
76. Sabayan B, Wijsman LW, Foster-Dingley JC, Stott DJ, Ford I, Buckley BM. Association of visit-to-visit variability in blood pressure with cognitive function in old age: Prospective cohort study. Br Med J. 2013;347:f4600. DOI:https://doi.org/10.1136/bmj.f4600

© 2017 Saad et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
http://sciencedomain.org/review-history/18683