WORLD JOURNAL OF PHARMACY AND PHARMACEUTICAL SCIENCES

SJIF Impact Factor 7.421

Volume 7, Issue 11, 110-130 Research Article ISSN 2278 – 4357

CORRELATION BETWEEN BLOOD PRESSURE AND BODY MASS INDEX AMONG MALE AND FEMALE (12-18 YEARS) BAGDAD

***Dr. Thaaban Khlbas and Dr. Razak Ali Hassan Shamaal**

AL-Timemi.

Article Received on
30 August 2018,

Revised on 20 Sept. 2018,
Accepted on 11 Oct. 2018,

DOI: 10.20959/wjpps201811-12551

***Corresponding Author**

Dr. Thaaban Khlbas

AL-Timemi.

SUMMARY

Background: In this study 1000 adolescents (12- 18) year were enrolled in different secondary schools of bagdad the period between April 26th till June 2nd 2013, the data received in a way so that to include 500 males and 500 females, 100 of them were 12 years with 150 for each other ages from 13 – 18 years. We did measure both the systolic and diastolic blood pressure in addition to calculation of their body mass index through the equation (weight /Height meter square). As a result we found that 939 (93.9%) of the cases were normotensive, 56 (5.6%) were prehypertensive and 5 (0.5%) were hypertensive. On making a correlation between body mass index and blood pressure we found that 60 (100%) of underweight student were normotensive, 765 (95.6%) of normal weight and 103 (93.6%) of the overweight were normotensive while only 11 (36.7 %) of the obese Students were normotensive. Regarding prehypertensive 35 (4.4 %) of normal weight were prehypertensive, 7 (6.4%) of overweight were prehypertensive while 14 (46.75) of obese patient were prehypertensive. Significantly all the five students (16.7%) who were in the range of hypertension were obese with body mass index of more than 95th percentile from this we concluded that there is a strong relation between high body mass index and high blood pressure (prehypertension and Hypertension). We recommend control of body weight through dietary regimen and regular physical exercise during adolescent period, as a prophylaxis against future hypertension and its complication.

INTRODUCTION

Blood pressure (BP) refers to the pressure that blood applies to the inner walls of blood vessels; high blood pressure is also called hypertension.

An individual's blood pressure is defined by two measurements:

Systolic pressure is the pressure in the arteries produced when the heart contracts (at the time of a heart beat).

Diastolic pressure refers to the pressure in the arteries during relaxation of the heart between heart beats.[1]

Definition

In children, the following definitions based upon the 2004 National High Blood Pressure Education Program Working Group (NHBPEP) are used to classify BP measurements in the United States.[1]

The definition of childhood hypertension (HTN) is statistically defined and based upon the normative distribution of BP in healthy children. This is in contrast to adult HTN, which is primarily defined by clinical outcome data i.e., risk of cardiovascular diseases and mortality, from large trials of antihypertensive therapy.[2]

This clinical definition cannot be applied to children because cardiovascular events other than left ventricular hypertrophy do not typically occur until adulthood.

Normative BP percentiles are based upon data on gender, age, height, and blood pressure measurements from the National Health and Nutrition Examination Survey (NHANES) and other population-based studies.

In children, definitions based upon BP percentiles were developed by the NHBPEP Working Group to categorize BP measurements based upon the normative data.[1]

The systolic and diastolic BP is of equal importance; if there is a disparity between the two, the higher value determines the BP category.

- Normal BP — both systolic and diastolic BP <90th percentile.
- Prehypertension — Systolic and/or diastolic BP ≥ 90th percentile but <95th percentile, or if BP exceeds 120/80 mmHg even if <90th percentile.

Prehypertension is predictive of hypertension. This was illustrated in one study based upon the National Childhood Blood Pressure database that demonstrated 14 percent of male and 12 percent female adolescents were hypertensive two years later.[3]

- Hypertension — HTN is defined as either systolic and/or diastolic BP ≥ 95th percentile measured upon three or more occasions.[3]

The degree of HTN is further delineated by the following two stages

Stage 1 hypertension — Systolic and/or diastolic BP between the 95th percentile and 5 mmHg above the 99th percentile.
Stage 2 hypertension — Systolic and/or diastolic BP ≥ 99th percentile plus 5mmHg.

Blood pressure varies with the age of the child and is closely related to height and weight. Significant increases occur during adolescence, and many temporary variations take place before the more stable levels of adult life are attained.

Exercise, excitement, coughing, crying, and struggling may raise the systolic pressure of infants and children as much as 40–50 mm Hg greater than their usual levels. Variability in blood pressure in children of approximately the same age and body build should be expected, and serial measurements should always be obtained when evaluating a patient with hypertension.

Factors that increased the likelihood of a documented diagnosis of HTN included the following:

- Obesity
- More than three readings of elevated blood pressure measurements
- Stage2 HTN
- Increasing age
- Increased height for age.

Epidemiology

Since the late 1980s, the prevalence of childhood HTN has increased in the United States as demonstrated by several large population-based surveys from the United States.[4]

- BP values decreased from 1963 to 1988; however, this trend was reversed between 1988 and 1999.

- The mean systolic BP increased by 1.3 mmHg and the mean diastolic BP increased by 8.4 between the two study periods of 1988 to 1994 and 1999 to 2002, respectively.
- The rise in BP between the study periods of 1988 to 1994 and 1999 to 2002 is concurrent to an increase in the prevalence of obesity.

Risk Factors

The incidence of hypertension in children may vary based upon the patient's body mass index (BMI), gender, ethnicity, and family history

In a retrospective review of primary care visits of 18,618 children between 2 and 19 years of age, systolic and diastolic BP increased with increasing BMI in all age groups including children between two and five years of age.[5]

Gender

In the United States and Canada, the prevalence of HTN and prehypertension is greater in boys than girls.[6]

Genetics

A family history of HTN is present in as many as 70 to 80 percent of all patients with primary HTN (also referred to as essential HTN), which has no identifiable underlying etiology, and in approximately 50 percent of hypertensive children.[7]

In patients with essential hypertension, elevated BP is thought to result from the interaction of multiple genes and environmental factors. It has been estimated that genetic factors account for approximately 30 percent of the variation in blood pressure in various populations.[8]

- The BP correlation is stronger between parents and children than between spouses.[9]
- There is no significant BP correlation between parents and adopted children.[10]
- Most studies in twins have shown BP correlation is stronger between identical (monozygotic) twins than between fraternal (dizygotic) twins or siblings.[9] However, the observation that the BP correlation is stronger among dizygotic twins than other first-degree relatives indicates a nongenetic environmental effect.

It is possible that white coat HTN in children represents two populations: one that is destined to develop essential HTN (prehypertensive) and one that remains normotensive outside the clinical setting.

In adults, white coat hypertension appears to be a prehypertensive condition with increased left ventricular mass and progression to sustained hypertension. Similar findings were noted in a retrospective study of children with white coat hypertension from a tertiary center. About two-thirds of children with white coat hypertension had either left ventricular hypertrophy detected by echocardiography or systolic BP greater than the 95th percentile for age, height and gender during treadmill exercise.[13]

Low birth weight

There are limited data suggesting a role for low birth weight in the development of essential hypertension.

Etiology

Causes of childhood hypertension are separated into two classes:

- Essential or primary HTN — No underlying cause is identified.
- Secondary HTN — an identifiable cause is determined. In children with secondary

HTN, the underlying disorder may be curable with complete resolution of HTN.[12]

Essential hypertension — Essential hypertension is the most common cause of HTN in older children and is a diagnosis of exclusion. It is more likely in children who are post pubertal, have a family history of HTN, are overweight or obese, or have only mild hypertension (BP at or just above the 95th percentile). It is also more common in African Americans.

Secondary hypertension — The following sections review different etiologies of chronic (persistent) secondary hypertension.[13]

Renal diseases

Renal parenchymal disease — varieties of intrinsic renal disorders are associated with HTN and include the following:[14]

1. Glomerulonephritis
2. Henoch-Schönlein purpura
3. Renal parenchymal scarring
4. Polycystic kidney disease
5. Chronic renal failure

Renovascular disease

Hypertension due to renovascular disease is due to a decrease in renal blood flow resulting in increased plasma levels of renin, angiotensin, and aldosterone. Children with renovascular disease generally have stage 2 HTN.[15]

1- Fibromuscular dysplasia: Fibromuscular dysplasia is the most common etiology of renovascular disease. It is characterized by arterial stenosis due to a non-inflammatory, non-atherosclerotic process.[16]
2- Umbilical arterial catheterization: During the newborn period, catheterization of the umbilical artery may lead to a clot in the renal artery resulting in renal arterial injury and stenosis.
3- Other causes of renovascular disease include neurofibromatosis, arteritis, renal artery hypoplasia, and midaortic syndrome (segmental narrowing of the proximal abdominal aorta)[16]

Renal tubular disease

Monogenic diseases in which tubular reabsorption of sodium or chloride is increased are associated with increased vascular volume and hypertension, such as Liddle's syndrome,

Type 1 pseudohypoaldosteronism, Type 2 pseudohypoaldosteronism or Gordon's syndrome.

Endocrinologic disease: Endocrinologic conditions associated with hypertension include the following

- Catecholamine excess — Catecholamine excess that results in hypertension.
- Corticosteroid excess — Corticosteroid excess is more commonly due to exogenous administration of glucocorticoids and rarely due to endogenous production of either glucocorticoids or mineralocorticoids. In both settings, corticosteroid excess results in hypertension.[17]
- Other endocrinologic disorders — other endocrinologic abnormalities associated with HTN include thyroid disorders (hypothyroidism and hyperthyroidism), and hypercalcemia (e.g., hyperparathyroidism).

Cardiac disease: Coarctation of the aorta is the primary cardiac cause of hypertension. The classic findings are hypertension in the upper extremities, diminished or delayed femoral pulses, and low or unobtainable arterial blood pressure in the lower extremities. The diagnosis is confirmed by echocardiogram.

Drugs and toxins: A variety of drugs and toxins can cause chronic hypertension including the following:

- Glucocorticoids
- Oral contraceptives.
- Arsenic
- Cyclosporine and tacrolimus.

Non pharmacological

- ✓ Moderate to vigorous physical aerobic activity 40 min, 3–5 days/week and avoid more than 2 h daily of sedentary activities.
- ✓ Avoid intake of excess sugar, excess soft drinks, saturated fat and salt and recommend fruits, vegetables and grain products.
- ✓ Implement the behavioural changes (physical activity and diet) tailored to individual and family characteristics.
- ✓ Involve the parents/family as partners in the behavioural change process
- ✓ Provide educational support and materials.
- ✓ Establish realistic goals.
- ✓ Develop health-promoting reward system.
- ✓ Competitive sports participation should be limited only in the presence of uncontrolled stage 2 hypertension.[18]

Therapeutic management of hypertension

Evidence for Therapeutic Management

- ➢ Therapeutic management of hypertension.
- ➢ Reduce mortality and sequelae in life-threatening conditions.
- ➢ Reduce left ventricular hypertrophy.
- ➢ Reduce urinary albumin excretion.
- ➢ Reduce rate of progression to end-stage renal disease.

When to Initiate Antihypertensive Treatment

Non-pharmacological therapy

Should be initiated in all children with high normal BP or hypertension; non-pharmacological therapy should be continued after starting pharmacological therapy.

Pharmacological therapy

Should be initiated when patients have symptomatic hypertension, hypertensive target organ damage, secondary hypertension or diabetes mellitus type 1 or 2 at the time of presentation.[19]

Therapeutic strategies

Several antihypertensive drugs are commercially available in liquid form or can be extemporaneously compounded for flexible dosing and ease of administration. Recent clinical trials have expanded the number of drugs that have pediatric dosing information based on dose finding studies.

Monotherapy

It is reasonable that in children, treatment should be started with a single drug administered at a low dose in order to avoid rapid fall in BP. If BP does not decrease sufficiently after a few weeks, usually 4–8, an increase to the full dose should be initiated. When BP does not respond adequately or significant side effects occur, the switching to another antihypertensive drug of a different class is recommended.[19]

Angiotensin-converting enzyme inhibitors

The oldest ACEI, Captopril, has been extensively studied in children. Its efficacy and safety appear to be well established, but the drug has a short duration of action. As it must be administered two or three times a day, it has been replaced by the longer-acting ACEIs. Some of these have been studied recently in children.[19]

Angiotensin receptor blockers

Data on the effects of ARBs in hypertensive children have accumulated recently. Short-term treatment with losartan in children with estimated GFRs 30 ml/min per 1.73m2 or more produced significant dose-dependent reductions in DBP. The effective starting dose was 0.75 mg/kg per day, but doses as high as 1.44 mg/kg per day were well tolerated. Recently, valsartan has effectively lowered SBP and DBP compared with placebo in children 1–5 years old.

Other antihypertensive agents

No pediatric studies have been conducted for diuretics, except for a very small old study on chlorthalidone, direct vasodilators, centrally acting agents, or alpha-1 receptor antagonists, despite their having a long history of clinical use in the pharmacological management of hypertension in children.

Loop diuretics such as furosemide are essential in children with advanced chronic renal failure or with heart failure.

Combination therapy

In children with renal disease, monotherapy is often not sufficient to achieve adequate BP control. Therefore, early combination therapy is required. Early dose combination of antihypertensive agents is more efficient and has a lower rate of adverse drug reaction compared with that of high-dose monotherapy.[19]

Obesity

Obesity has become one of the most important public health problems in the United States. As the prevalence of obesity increases so does the prevalence of the comorbidities associated with obesity. For this reason it is imperative that health care providers identify overweight and obese children so that counseling and treatment can be provided.[20]

“Overweight" technically refers to an excess of body weight, whereas "obesity" refers to an excess of fat. However, the methods used to directly measure body fat are not available in daily practice. For this reason, obesity is often assessed by means of indirect estimates of body fat (i.e., anthropometrics).[21]

The body mass index (BMI) is the accepted standard measure of overweight and obesity for children two years of age. Body mass index provides a guideline for weight in relation to height and is equal to the body weight divided by the height squared.[22]

Adults with a BMI between 25 and 30 are considered overweight; those with a BMI ≥ 30 are considered to be obese. Unlike adults, children grow in height as well as weight. Thus, the norms for BMI in children vary with age and sex. In 2000, the National Center for Health

Care Statistics and the Centers for Disease Control (CDC) published BMI reference standards for children between the ages of 2 and 20 years.

- Underweight — BMI <5th percentile for age and sex.
- Normal weight — BMI between the 5th and 85th percentile for age and sex.
- Overweight — BMI between the 85th and 95th percentile for age and sex. The CDC uses the term "at risk for overweight" for this category.
- Obese — BMI ≥ 95th percentile for age and sex. The CDC uses the term "overweight" for this category.

Waist circumference reflects visceral obesity better than BMI and it is measured in the level midway between the lowest rib margin and the iliac crest.

Abdominal obesity defined as the ratio of waist circumference and height > 0.5

Epidemiology

Currently, almost one third of children and adolescents in the United States are either overweight or obese, childhood obesity is more common among Native Americans, non-Hispanic blacks, and Mexican Americans than in white. Having an obese parent increases the risk of obesity by two-to three folds.

Etiology

Environmental factors

Almost all obesity in children is strongly influenced by environmental factors, caused by either a sedentary lifestyle or a caloric intake that is greater than needs.

The contributions of specific environmental influences are the subject of considerable discussion and research. Increasing trends in glycemic index of foods, sugar-containing beverages, and portion sizes for prepared foods, fast food service, and decreasing structured physical activity have all been considered as causal influences on the rise in obesity, but the evidence base remains weak.

Television: Television viewing is perhaps the best established environmental influence on the development of obesity during childhood. The period spent in watching television is directly related to the prevalence of obesity in children and adolescents. In two longitudinal cohort studies, television viewing at ≥ 5 years was independently associated with increased

BMI at age 26 to 30 years. Other studies suggest that the association between television viewing and obesity is considerably weak.[20]

Sleep: Cross-sectional studies suggest an association between shortened sleep duration and obesity or insulin resistance. Two longitudinal studies also showed associations after adjustment for confounders, suggesting that the association may be causal. The mechanism for the association has not been established, but may include alterations in serum leptin and ghrelin levels, both of which have been implicated in the regulation of appetite, or perhaps a longer opportunity to ingest food.[23]

Genetic factors

Genetic factors play a permissive role and interact with environmental factors to produce obesity. Studies suggest that heritable factors are responsible for 30 to 50 percent of the variation in adiposity.[24]

Endocrine disease

Endocrine causes of obesity are identified in less than 1 percent of children and adolescents with obesity. The disorders include hypothyroidism, cortisol excess (e.g., the use of corticosteroid medication, Cushing syndrome), growth hormone deficiency, and acquired hypothalamic lesions (e.g., infection, vascular malformation, neoplasm, trauma). Most children with these problems have short stature and/or hypogonadism.[25]

Psychological and Social Consequences of Obesity

From an early age, society stigmatizes obese persons as lazy, stupidslow, and self-indulgent. Children express negative attitudes toward their obese peers as early as kindergarten and even prefer a playmate that uses a wheelchair or has a major physical disability to one who is obese.

Initiation of weight management at 6 to 9 years of age can improve the chance of overall success. Socioeconomic complications of obesity among teenage girls include persistent and severe disturbances in body image, lower likelihood of marriage, fewer years of education, low income, and a high incidence of poverty. Overweight men are less affected in these ways.[26]

Endocrine Changes and Obesity

Serum values of GH-binding protein are directly proportional to BMI and are high in obesity. Increased binding sites for GH may explain the normal serum level of IGF-I and excellent growth among obese children despite low GH secretion. IGFBP1 is suppressed by insulin, and serum values are low in obesity because of increased insulin secretion. Hypothyroidism often is thought to be a cause of obesity. However, even among untreated children with hypothyroidism, weight gain is modest, and massive obesity rarely is explained by hypothyroidism. Treatment does produce modest weight loss.[27]

Evaluation and Management of Obesity

A history of headaches or neurologic symptoms, evidence of polyuria and polydipsia, or evidence of a psychopathologic condition may indicate a central nervous system tumor. Constipation, cold intolerance, and dry skin may suggest hypothyroidism. Linear growth failure is uncommon among obese children, who usually are tall for age. Cushing syndrome, pseudohypoparathyroidism, and hypothalamic lesions must be considered if the child is short and obese.[28]

The physical examination is focused on blood pressure, BMI, distribution of fat, optic disc contour, and the presence of acanthosis nigricans.

Laboratory evaluation may be needed to exclude the causes of obesity discussed earlier. If none of these causes is suspected, the appropriate laboratory evaluation is directed at screening for complications of obesity. For a child with a BMI in the 85th to 94th percentile, only a fasting lipid profile is recommended unless the risk factors include cigarette smoking, the presence of diabetes mellitus, low physical activity, high blood pressure, a family history of early cardiovascular disease, or a family history that suggests diabetes or hyperlipidemia. In such cases, and for all children with a BMI greater than 95th percentile.[19]

Treatment

Mild, uncomplicated cases of obesity usually can be approached in the primary physician's office.

Severe or complicated cases necessitate a comprehensive approach, which is available in relatively few centers. The goals of a weight management program are improvement of medical and psychological health and prevention of comorbidities of obesity.

A change in diet that maintains weight stability or promotes slow weight loss is the most reasonable approach for most children.

Two kilograms to 4 kg is a reasonable first goal; if preferred, a rate of 0.5 to 2 kg a month can be established. Lessons on healthful eating, portion control, choosing appropriate foods, label reading, and modification of recipes are essential to help the family and child achieve successful weight management.

Limitation of fat intake is beneficial above and beyond the effect on calories. Keeping a dietary log helps the person concentrate on desired changes.

It is necessary to provide parents with a specific calorie-per-day recommendation that follows guidelines for percentages of fat, protein, and carbohydrates.

Tobacco use must be discouraged; some teenagers smoke in a misguided attempt to control weight.[30]

Physical Activity

A child cannot exercise enough to burn off a high-calorie diet. A children's meal of cheeseburger, French fries, and a milk shake in a fast-food restaurants exceeds 750 calories; large-size children's meals that exceed 1000 calories have appeared.

It would take more than 1.5 to 2 hours on a ski machine or stationary bicycle to work off such a single meal.

The goal is to avoid such high-calorie loads and to reduce sedentary activity rather than to encourage a regimen of exercise to which no one can adhere. Fostering activities that can be carried through life is best. Some centers suggest the use of hip-hop or other popular dances as exercise.[31]

Surgery

Gastric bypass or banding has benefits among morbidly obese adults with sustained weight loss. Monitoring for nutritional complications of protein deficiency, malnutrition, vitamins A, D, and B12, and folic acid is important.

Other complications include dumping syndrome and surgical adhesion. There is a little experience with this therapy in childhood and young adolescence.[32]

Medications

No medications have been approved as therapy for obesity among persons younger than 16 years. They are being tested on children 12 to 16 years of age.

Sibutramine, a serotonin reuptake inhibitor, can be used by older adolescents to increase the feeling of satiety and decrease intake.
A side effect is a slight increase in blood pressure.

Orlistat is an intestinal lipase inhibitor that decreases absorption of fat. Side effects such as bloating, flatulence, and diarrhea occur if fat intake is not reduced. As the biological mechanism of appetite control and obesity is elucidated, it is expected that better targeted, safer agents will become available.[33]

AIMS OF THE STUDY

- To assess the normal range of blood pressure among adolescents (12-18y).
- To know the prevalence of over weight and the obesity in these ages.
- To detect the relation between obesity and high blood pressure.

Patients and method

In this study 1006 adolescents were enrolled in different secondary schools of bagdad in the period between April -26th- till June -2nd – 2013 , the age of the students age were between 12-18 years of both sexes and the data were received in a way so that to include nearly equal number for both ages and sexes.

We classified the students to three social classes (Low, Medium and High) according to life style, income of parents, number of family member and own house with number of rooms.

Six cases had been excluded because their ages were out of the range (above 18 years).

At first we took short history about their life style, active or passive smoking and family history of hypertension.

Then we measured height and weight of them by using special gauge for the weight (electronic gauge) and through plotting the wall we had got the height of them, then from these two measures we obtained body mass index via the equation (weight in kg / height in meter square).

Lastly we did measure both systolic and diastolic blood pressure by using different size of cuff of mercury sphygmomanometer (auscultatory method) in the left arm (sitting position at the level of the heart) as a standard of all.

The posture and psychological aspect of view were taken in a consideration.

Those cases that were suspected to have borderline or high blood pressure repeated measurement (two times) had done for them for the final decision.

Data entry and analysis

All Data entered and analyzed by using the SPSS (statistical package for social science) – version16.

Chi-square test used for calculating of p value and p value <0.05 regards as significant level.

RESULTS

The frequencies of the variables studied are shown in table (1)

Age of child 12years (10%), 13year(15%),14year (15%), 15year (15%), 16year (15%) 17year (15%), 18year (15%).age of child: male (50%) &female (50%).

Smoking: passive Smoking (22.5%) & non smoking (77.5%).

Family history of hypertension: first degree (22%), Second degree (19.5%), no family history (58.5%).

Socio economic class: low (14%), medium (75.5%), high class (10.5%).

According to this study which shown in figure (1) we found that 800 (80%) of the students had normal weight (Body mass index) between 5th to 85th percentiles, whereas 110 (11%) were over weight (Body mass index) between 85th to 95th percentiles, 60 (6%) of them were underweight (Body mass index) below 5th percentile while 30 (3%) were Obese (Body mass index) was above 95th percentile.

We also measure both the systolic and diastolic blood pressure of all the selected samples by auscultatory method with a suitable gauge according to the age and arm circumference.

We found that (939) 93.9% of the cases were normal and (56) 5.6% were pre hypertensive Whereas only (5) 0.5% was hypertensive according to the chart of center of disease control (CDC), figure 2 shows the descriptive measures of the blood pressure ranges.

Lastly we made a correlation between the body mass indices and both the systolic and diastolic blood pressure via the analysis of the sample variables then we reached the following results.

60(100%) of the under weight students were normotensive, 765(95.6%) of the normal weight and103 (93.6%) of the over weight were normotensive, while only 11(36.7%) of the obese students were normotensive.

About prehypertensive only 35(4.4%) of normal weight were prehypertensive and 7(6.4%) of overweight were prehypertensive whereas 14 (46.7%) of obese students were prehypertensive.

And finally the five students (16.7%) who were regarded as hypertensive range, all were obese with a body mass index of more than 95th percentile.

So the heaviest the weight the grater the possibility of having hypertension.

Table 2 shows the correlation between the range of blood pressure and body mass index.

Table 1: Shows descriptive analysis of the sample variables.

Socio-demographic characteristics	Frequency	Percent
Age of child (Years)		
12	100	10.0
13	150	15.0
14	150	15.0
15	150	15.0
16	150	15.0
17	150	15.0
18	150	15.0
Gender of child		
Male	500	50.0
Female	500	50.0
Smoking		
Passive	225	22.5
No smoking	775	77.5
Family history of hypertension	220	22.0
1st degree relative	195	19.5

2nd degree relative No family history	585	58.5
Socio economic class Low Medium High	 140 755 105	 14.0 75.5 10.5

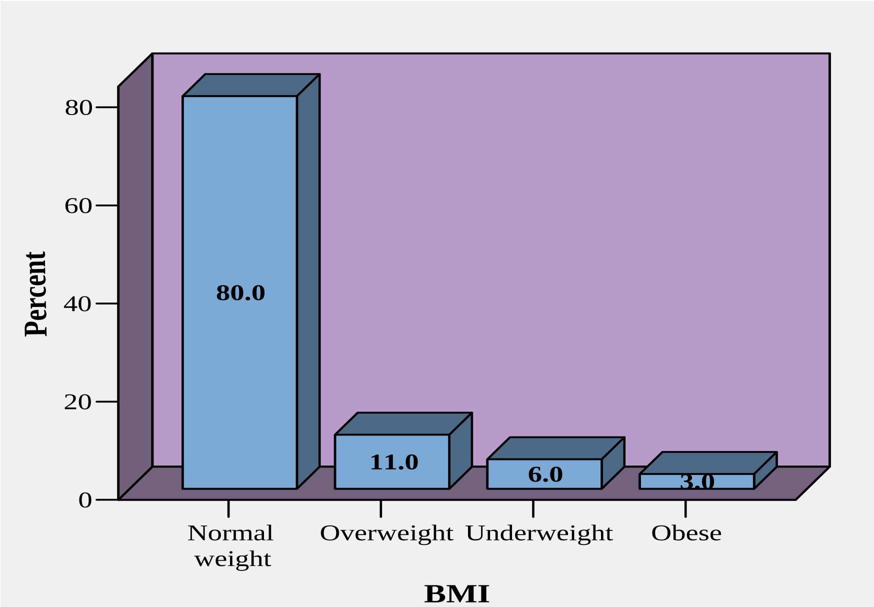

Figure 1: Shows the distributions of percentage of Body mass index.

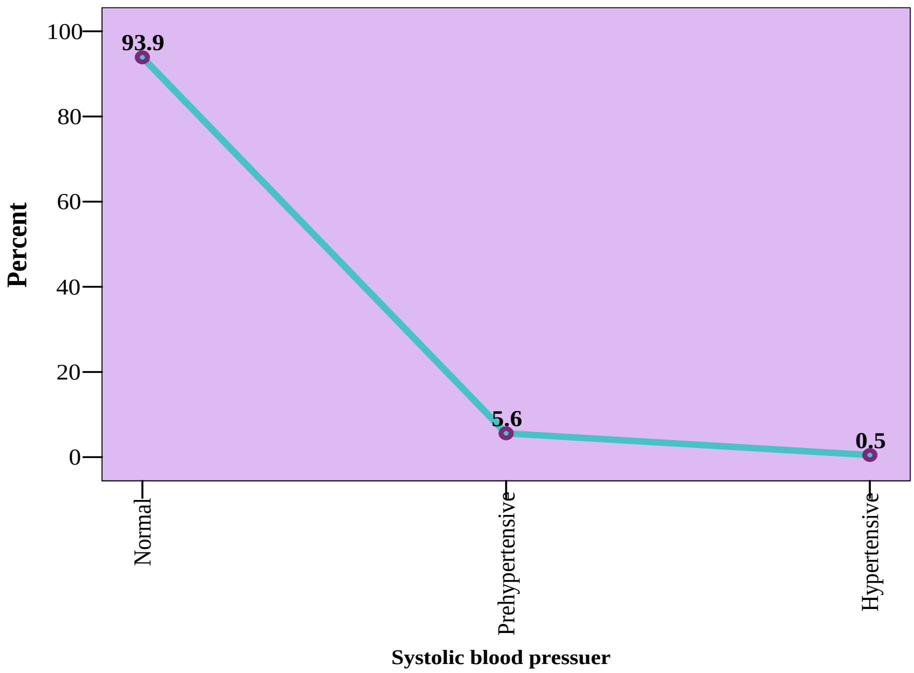

Figure 2: Shows the descriptive measures of the blood pressure ranges.

Table 2: Shows the correlation between the range of blood pressure and body mass index.

Blood pressure Status	BMI				P value
	Underweight N (%)	Normal weight N (%)	Overweight N (%)	Obese N (%)	
Systolic blood pressure					
Normal	60(100.0)	765(95.6)	103(93.6)	11(36.7)	**0.000**
Prehypertensive	0(0.0)	35(4.4)	7(6.4)	14(46.7)	
Hypertensive	0(0.0)	0(0.0)	0(0.0)	5(16.7)	
Diastolic blood pressure					
Normal	60(100.0)	770(96.3)	99(90.0)	13(43.3)	**0.000**
Prehypertensive	0(0.0)	30(3.8)	11(10.0)	12(40.0)	
Hypertensive	0(0.0)	0(0.0)	0(0.0)	5(16.7)	

DISCUSSION

According to preventive department in Sulaimanyia this is the first time for such study in Sulaimanyia governorate, so we have no local similar study to compare with.

But over the entire world many similar researches had been done like Cross-sectional assessment of blood pressure (BP) in 6790 adolescents (11–17years) in Houston schools was conducted from 2003–2005. Initial measurements included height, weight, and four oscillometric BP readings. Repeat measurements were obtained on two subsequent occasions in students with persistently elevated BP. Final prevalence was adjusted for loss to follow-up and logistic regression used to assess risk factors, then also they found that Hypertension and pre-hypertension increased with increasing BMI.[34]

Also Empar Lurbea,b, Renata et al.[35] did similar research but they compare the blood pressure with height percentile for age instead of body mass index.

Conclusion and Recommendation

We found that there is a strong relation between high body mass index (obesity) and high blood pressure (pre-hypertension and hypertension).

We recommend control of body weight via dietary regime or regular physical exercise during adolescent period, as a prophylaxis against future hypertension and its complication.

REFERENCES

1. Falkner, B, Gidding, SS, The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics, 2004; 114: 555.
2. Chobanian, AV, Bakris, GL, Black, HR, Cushman, WC. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report. JAMA, 2003; 289: 2560.
3. Falkner, B, Gidding, SS, Portman, R, Rosner, B. Blood pressure variability and classification of prehypertension and hypertension in adolescence. Pediatrics, 2008; 122: 238.
4. Sinaiko, AR, Gomez-Marin, O, Prineas, RJ. Prevalence of "significant" hypertension in junior high school-aged children: the Children and Adolescent Blood Pressure Program. J Pediatr, 1989; 114: 664.
5. Falkner, B, Gidding, SS, Ramirez-Garnica, G, et al. The relationship of body mass index and blood pressure in primary care pediatric patients. J Pediatr, 2006; 148: 195.
6. Din-Dzietham, R, Liu, Y, Bielo, MV, Shamsa, F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. Circulation, 2007; 116: 1488.
7. Jung, FF, Ingelfinger, JR. Hypertension in childhood and adolescenc}e. Pediatr Rev., 1993; 14: 169.
8. Hunt, SC, Ellison, RC, Atwood, LD, et al. Genome scans for blood pressure and hypertension: the National Heart, Lung, and Blood Institute Family Heart Study. Hypertension, 2002; 40: 1.
9. Havlik, RJ, Feinleib, M. Epidemiology and genetics of hypertension. Hypertension, 1982; 4: III121.
10. Biron, P, Mongeau, JG, Bertrand, D. Familial aggregation of blood pressure in 558 adopted children. Can Med Assoc J, 1976; 115: 773.
11. Martin, RM, Ness, AR, Gunnell, D, et al. Does breast-feeding in infancy lower blood pressure in childhood? The Avon Longitude Study of Parents and Children (ALSPAC). Circulation, 2004; 109: 1259.
12. Ocon-Pujadas, J, Mora-Macia, J. White coat hypertension and related phenomena. A clinical approach. Drugs, 1993; 46 Suppl 2: 95.
13. Kavey, RE, Kveselis, DA, Atallah, N, Smith, FC. White coat hypertension in childhood: evidence for end-organ effect. J Pediatr, 2007; 150: 491.
14. Wyszynska, T, Cichocka, E, Wieteska-Klimczak, A, et al. A single pediatric center experience with 1025 children with hypertension. Acta Paediatr, 1992; 81: 244.

15. Dillon, MJ. The diagnosis of renovascular disease. Pediatr Nephrol, 1997; 11: 366.
16. Deal, JE, Snell, MF, Barratt, TM, Dillon, MJ. Renovascular disease in childhood. J Pediatr 1992; 121: 378.
17. Piercy, KT, Hundley, JC, Stafford, JM, et al. Renovascular disease in children and adolescents. J Vasc Surg, 2005; 41: 973.
18. Lurbe E, Parati G. Out-of-office blood pressure measurement in children and adolescents. J Hypertens, 2008; 26: 1536–1539.
19. Lippincott Williams & Wilkins, Managing hypertension in children and adolescents Lurbe et al. 1731 Copyright © Lippincott Williams & Wilkins.
20. Dietz, WH, Robinson, TN. Clinical practice. Overweight children and adolescents. N Engl J Med, 2005; 352: 2100.
21. Flodmark, CE, Lissau, I, Moreno, LA, et al. New insights into the field of children and adolescents' obesity: the European perspective. Int J Obes Relat Metab Disord, 2004; 28: 1189.
22. Deurenberg, P, Weststrate, JA, Seidell, JC. Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. Br J Nutr., 1991; 65: 105.
23. Flint, J, Kothare, SV, Zihlif, M, et al. Association between inadequate sleep and insulin resistance in obese children. J Pediatr., 2007; 150: 364.
24. Bouchard, C. Genetic determinants of regional fat distribution. Hum Reprod, 1997; 12Suppl 1: 1.
25. Reinehr, T, Hinney, A, de Sousa, G, et al. Definable somatic disorders in overweight children and adolescents. J Pediatr, 2007; 150: 618.
26. Barlow SE, Dietz WH: Obesity evaluation and treatment: expert committee recommendations. Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. Pediatrics, 1998; 102: E29.
27. Barsh GS, Farooqi IS, O'Rahilly S: Genetics of body-weight regulation. Nature, 2000; 404: 644-651.
28. Bray GA, Tartaglia LA: Medicinal strategies in the treatment of obesity. Nature, 2000; 404: 672-677.
29. Chagnon YC, Chen WJ, Perusse L, et al: Linkage and association studies between the melanocortin receptors 4 and 5 genes and obesity-related phenotypes in the Quebec Family Study. Mol Med, 1997; 3: 663-673.

30. Epstein LH: Family-based behavioural intervention for obese children. Int J Obes Relat Metab Disord, 1996; 20: S14-S21.
31. Friedman JM: Obesity in the new millennium. Nature, 2000; 404: 632-634.
32. Hixson JE, Almasy L, Cole S, et al: Normal variation in leptin levels is associated with polymorphisms in the proopiomelanocortin gene, POMC. J Clin Endocrinol Metab, 1999; 84: 3187-3191.
33. Bray GA, Tartaglia LA: Medicinal strategies in the treatment of obesity. Nature, 2000; 404: 672-677. (Claement K, Vaisse C, Lahlou N, et al: A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction. Nature, 1998; 392: 398-401.
34. Antal M, Regoly-Merei A, Nagy K, Greiner E, Biro L, Domonkos A, et al. Representative Study for the Evaluation of Age-and Gender-Specific Anthropometric Parameters and Blood Pressure in an Adolescent Hungarian Population. Ann Nutr Metab, 2004; 48: 307–13. [PubMed: 15467280].
35. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R,Germano G, et al. Guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens, 2007; 25: 1105–1187.