

CHAPTER II

LITERATURE REVIEW

A. Literature Review

1. Adolescent

a. Definition of adolescent

According to (Hockenbery and Wilson, 2007), the word adolescent is Latin in Origin, derived from verb *adolescere*, which means “to grow into adulthood”. In all societies, adolescent is a time of growing up, of moving from immaturity of childhood. Adolescent is a period of biological, psychological, social, and economic transition.

WHO (2005) defined adolescent as several definition based on physical, mental, and social and economic aspects. Based on physical aspect, adolescent was defined as changes in the reproductive system occur until it reaches its full development. Mentally, adolescent was defined as the child develops into an adult. Social and economically, children transition from being financially dependent into being financially self-supporting were defined as adolescent.

b. Classification of adolescent

WHO (2005) classifies adolescent into early, middle, and late adolescent. Early adolescent (aged 10-15 years) is a period of a spurt of physical growth. Middle adolescent (aged 16-19 years) is a stage of

experiencing wide mood swings, being influenced by peers, interest in the opposite sex, complete physical changes, and sexual experimentation. Late adolescent (aged 20-24 years) is the age of perceived gender role, independent living and working, increase interest in sexuality, and acceptance of advice regarding.

Age is the criterion for assigning the level of study. According to Departemen Pendidikan Nasional Republik Indonesia (Depdiknas), the education system in Indonesia (2010) for is divided into three levels, include primary level, secondary level, and higher level of education. Primary level is 9 years early education, which had 6-14 years old students or early adolescent students. Secondary level of education had 15-17 years old students or middle adolescent students, while higher level of education had more than 18 years old students or late adolescent students or older.

c. Development of adolescent

(1) Physical development

Guyton and Hall (2006) explained that the early years of adolescent are important because it is the transitional period from childhood into adulthood. Hormones influence physical development. Adolescent in this age often feel hungry and usually eat a large quantity of food. They usually do not pay much attention to the nutritional value of their diet. During this stage, oil glands and sweat

glands are functioning at their peak, so their faces are usually oily, and they may develop pimples. Many adolescents start to grow chubbier, taller, and heavier. Hair begins to grow on different parts of their body, such as in the armpits and the pubic area, menarche begins in girls and wet dreams happen in boys.

(2) Emotional development

American Academy of Child and Adolescent Psychiatry (2006) explained that adolescents are easily moved often obsessed with being socially included, and like to have many friends. They start to feel affection towards the opposite sex, and think about their careers and freedom.

(3) Intellectual development

Adolescents are the age when there is advanced intellectual development. When the adolescent period ends, children have a similar level of competence as adults. However, it would still differ from the adult's level in the dimension of reflection and experience.

2. Overweight

a. Definition of Overweight

Generally, overweight refers to the state of weighing more than average for height and body built (Hockenberry and Wilson, 2007).

Definition of overweight in adult was described as different state with the

definition in children and adolescents. Overweight for adult was determined by BMI. BMI for adult was described by WHO in following table:

Table 2.1. WHO Classification of BMI for Adult

BMI (kg/m ²)	Who Classification	Other Description
<18.5	Underweight	Thin
18.5-24.9	Normal range	Healthy, normal, or acceptable weight
25-25.9	Grade 1 overweight	Overweight
30-30.9	Grade 2 overweight	Obesity
≥40	Grade 3 overweight	Morbidly overweight

Source: World Health Organization (2010)

According to Robertson (2006), serial BMI was used as a representative measure of overweight. It, however, could not distinguish between excess weight produced by adiposity, muscularity or edema. In children with nutritional deficiency, and in the setting of overweight and obesity, it is useful measure of fatness. It is calculated from the formula BMI.

Ganong (2005) explained a convenient and reliable indicators of body fat is the body mass index, which is the body fat (in kilograms) divided by the square of the height (in meters).

$$\frac{\text{Body weight (kg)}}{\text{Body height (m)}^2}$$

Furthermore, Robertson (2006) explained calculated BMI values need to be compared with age-and sex-specific reference standards. In Australia, the recommended BMI charts are the BMI-for-age percentiles charts developed in the United States of America by the Centers for Disease Control

and Prevention (CDC). A BMI greater than or equal to 95th percentile refers to overweight in children and adolescents.

Differences of BMI classification in adult, and children and adolescents according to CDC were described in following table:

Table 2.2. Criteria for Defining Weight in Adults, and Children and Adolescents

Weight Category	Adults (21 year and older)	Children and Adolescents (2-21 year)
Underweight	BMI <18.5	<5 th percentile BMI for age
Normal weight	BMI >18.5-24.9	BMI for age > 5 th -85 percentile
At risk of overweight	Not used in adults	BMI >85 th -95 th percentile
Overweight	BMI > 25-29.9	BMI for age ≥95 th percentile
Obesity	BMI >30-39.9	Not used
Extreme obesity	BMI ≥ 40	Not used

Source: Modified from National Institute for Health Care Management Foundaton 2003 (Hockenberry and Wilson,2007)

b. Etiology of Overweight

Factors that lead to overweight in adolescents had been studied by many researchers. They included genetic factors, environmental factors, nutritional factors, molecular and metabolic factors, diseases, and psychological factors, (Birnkrant and Alarid, 2007; Hockenberry and Wilson, 2007; Porth and Matfin, 2009). Each of them will be discussed below.

Genetic studies had found genetic contribution to overweight. Studies of twin and adopted children have proved evidence that heredity contributes to overweight (Porth and Matfin, 2009). When both parents are overweight,

is a 60% to 80% increase in the likelihood of the child becoming obese (Koeppen et al., 2001 cit Hockenberry and Wilson, 2007).

Although genetic factors may explain some of individual variations in terms of excess weight, environmental influences are the major contributors (Porth and Matfin, 2009). Environmental contributions included family eating pattern, physical inactivity, oversized food portion, lack of access to healthy foods, and food advertising.

The increasing rates of overweight and obesity within genetically stable populations suggest that environmental and some perinatal factors (e.g., bottle feeding) are contributors to the current increases in childhood obesity (Hockenberry and Wilson, 2007). In Indonesia, it has been studied that breastfeeding duration more than 12 months until less than or equal to 24 months could significantly decrease the risk of 3-5 years of age children's obesity compared to breastfeeding duration less than 6 months (Handayani, 2007).

Hockenberry and Wilson (2007) explained family and cultural eating patterns also play an important role in overweight. Many families and cultures consider fat to be an indication of good health. In some cultures obesity is a status symbol or an indication of affluence. It is not uncommon for obese children to have families that emphasize large meals or admonish children for leaving food on their plates. Parents may have a tendency to

concept of the amount of food children require and expect them to eat more than they need.

Adolescents are surrounded by ads from food companies. Often they are the targets of advertising for high-calorie, high-fat snacks and sugary drinks. Some of these meals and snacks can feed two or more people. Eating large portions means too much energy intake. Over time, this will cause weight gain if it isn't balanced with physical activity. The goal of these ads is to sway people to buy these high-calorie foods, and often they do.

Physical inactivity has also been identified as an important contributing factor in the development and maintenance of childhood overweight. The growing attraction and availability of many sedentary activities, including television, video games, computers, and the internet, have greatly influenced the amount of time that children spend participating in sedentary behaviors (Hockenberry and Wilson, 2007). Another physical inactivity contributing to overweight is reliance on the automobile for transportation (Porth and Matfin, 2009).

Molecular, metabolic, and endocrine factors also contributed to overweight development. Hockenberry and Wilson (2007) explained that a number of hormones and proteins that regulate appetite and weight have been identified in animal models. It is likely that these same mechanisms would apply to humans. However, the role of hormones and neurotransmitters in

determining overweight in humans remains unknown. Only a small number of enzyme abnormalities and metabolic defects have been identified, and these cannot account for the rapid increase in childhood obesity over the past 3 decades. There is a little evidence to support a relationship between obesity and low metabolism. There may be small differences in regulation of dietary intake or metabolic rate between obese and nonobese children that could lead to an energy imbalance and inappropriate weight gain, but these small differences are difficult to accurately quantify.

Kumar et al., (2005) explained fewer than 5% of the cases of childhood obesity can be attributed to an underlying disease. Such diseases include hyperthyroidism, adrenal hypercorticism, hyperinsulinism, and dysfunction or damage to the central nervous system as a result of tumor, injury, infection, or vascular accident. Obesity is a frequent syndrome complication of muscular dystrophy, paraplegia, Down syndrome, spina bifida, and other chronic illness that limit mobility.

Saladin (2010) explained psychologic factors also affect eating patterns. In infancy, children experience relief from discomfort through feeding and learn to associate eating with a sense of wellbeing, security, and the comforting presence of nurturing person. Eating is soon associated with the feeling of being loved. In addition, the pleasurable oral sensation of sucking provides a connection between emotions and early eating behaviors

Moreover, Guyton and Hall (2006) explained the eating patterns may become a habit, and the child may continue to use food as a reward, a comfort, and a means to deal with feelings of depression or hostility. Many individuals eat when they are not hungry or in response to boredom, loneliness, sadness, depression, or tiredness. Difficulty determining feelings of safety can lead to weight problems and may compound the factors of eating in response to emotional rather than physical hunger cues.

c. Pathophysiology of overweight

Pathophysiology of overweight or obesity in adolescents is complex. However, simply put, Kumar et al., (2005) explained that obesity is a disorder of energy balance. When food-derived energy chronically exceeds energy expenditure, the excess calories are stored as triglycerides in adipose tissue. The two sides of the energy equation, intake and expenditure, are finely regulated by hormonal and neural mechanism.

According to Salidin (2010), since the early 1990s, physiologists have discovered a still-growing list of peptide hormones and regulatory pathways that control short and long term appetite and body weight. Some of the hormones have been called *gut-brain peptides* because they act as chemical signals from the gastrointestinal tract to the brain.

Ghrelin, peptide YY (PYY), cholecystokinin (CCK) are three of the *gut-brain peptides* which act as short term regulators of appetite. Ghrelin is

produced by the stomach in response to hunger and stimulates food intake and induces metabolic changes leading to an increase in body weight and body fat mass (McCance et al., 2010). Ghrelin is one of the signals that begins a meal, whereas PYY is one of the signals that ends meal and elevated well after a meal. CCK which is secreted by enteroendocrine cells in the duodenum and jejunum, joins PYY as a signal to stop eating (Saladin, 2010).

Other peptides regulate appetite, metabolic rate, and body weight, over the longer term are leptin and insulin. They work as adiposity signals which is informing the brain of how much adipose tissue the body has and activating mechanisms for adding or reducing fat (Kumar et al., 2005).

An important brain center for appetite regulation is the arcuate nucleus (ARC) of the hypothalamus (Saladin, 2010). It has two sets of neurons with opposing effects that interact to regulate balance food intake and energy metabolism. One set of neurons produces neuropeptide Y (NPY) and agouti-related protein (AGRP), which stimulates eating and decreases metabolism. Another set of neurons synthesizes pro-opiomelanocortin (POMC)-producing peptide and cocaine-and-amphetamine-regulated transcript (CART), collectively as eating inhibitor and metabolism stimulator (McCance et al., 2010).

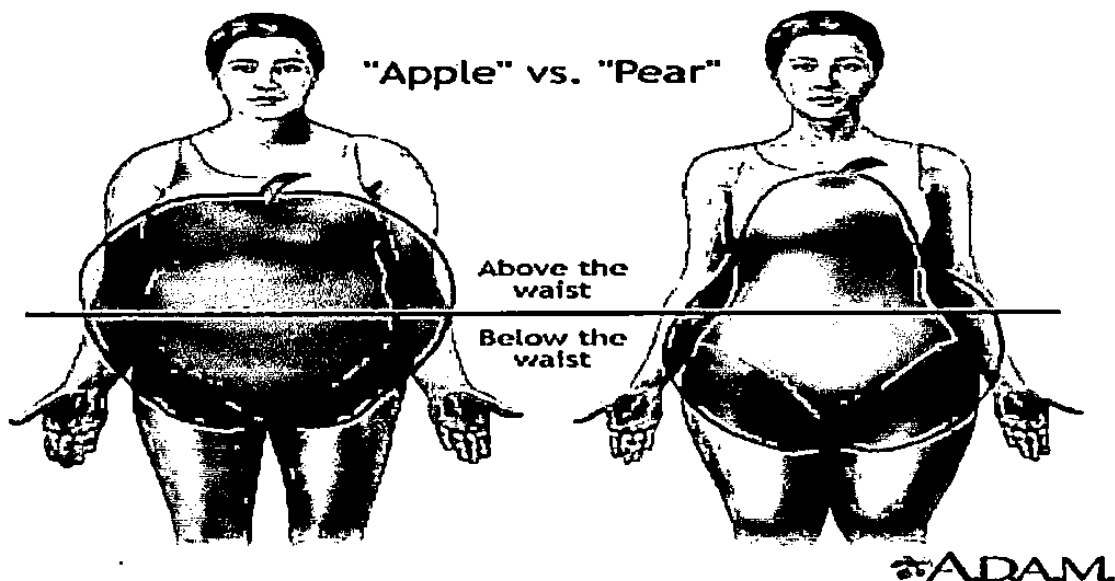
McCance et al., (2010) also explained molecules that stimulate eating are called orexins, while molecules that inhibit eating are called anorexins.

Peripheral effects of these signaling pathways are transmitted through the autonomic nervous and endocrine systems to regulate appetite, food intake, and energy metabolism.

Kumar et al., (2005) explained that obesity or overweight is associated with increased circulating plasma levels of leptin, insulin, ghrelin, and PYY. Interaction among these hormones at the level of the hypothalamus may be an important determinant of excessive fat mass. The high levels of leptin in obesity are ineffective at decreasing appetite and increasing energy expenditure. This is known as leptin resistance. Leptin resistance disrupts hypothalamic satiety signaling and promotes overeating and excessive weight gain.

According to McCance et al., (2010), the cause of leptin resistance is unknown. It may be related to a defect in leptin transport, an inability of leptin to cross the blood-brain barrier, an alteration in the permissive effect of leptin on urocortin, or a defect in the leptin receptors. Hyperleptinemia also stimulates the sympathetic nervous system, chronic inflammation, oxidative stress, and ventricular hypertrophy and many contributes to the pathogenesis of hypertension, atherosclerosis, and cardiovascular disease associated with

with lower body obesity, which is more pear shaped. Following picture describe the differences between apple and pear-shaped of obesity:



Picture 1. Apple and Pear-shaped of obesity
(Simon, et al., 2009)

WHO (2000) explained that in general, men have twice the amount of abdominal fat than is generally found in premenopausal women. As men age, the proportion of intra-abdominal fat to subcutaneous fat increases. After menopause, women tend to acquire more abdominal fat distribution.

Ciokan (2010) explained that generally, physical condition and physical activity has a genetic and hormonal influence, fat distribution is influenced by age, genetic inheritance, race, but to a greater extent by gender-specific hormones. They are responsible for the distribution of fat on certain areas of our bodies. Estrogens, for example, which are responsible for the typical female sexual characteristics, will influence fat deposit in the pear

format. They deposit on the hips, thighs, and belly, while testosterone will "lead" fat mostly towards the stomach and upper body.

Abdominal fat distribution is more danger than subcutaneous fat distribution. The presence of excess fat in the abdomen is an independent predictor of risk factors and mortality (Porth and Matfin, 2009). Of particular importance is the effect excess abdominal fat has on cardiometabolic risk. Cardiometabolic risk represents the overall risk of developing diabetes and/or cardiovascular disease, such as coronary artery disease, stroke, peripheral vascular disease, which is due ti a cluster traditional risk factors such as obesity, hypertension, and dyslipidemia (Underwood, 2004).

3. Weight Circumference

a. Definition of Waist Circumference

Garnett at al., (2007) explained the long-term health outcomes of children and adolescents with different amounts of total body fat are unknown because most large-scale studies of the effects of childhood obesity have not used measures of body fat, but rather proxies such as body mass index (BMI). The use of BMI to classify children and adolescents as overweight or obese is well established. However, BMI may not indicate the level of central adiposity, which is also associated with the clustering of cardiovascular disease (CVD) risk factors, including dyslipidemia, hypertension, and insulin resistance. Clusters of risk factors are fairly stable characteristics that tend to

track from adolescence to adulthood; hence the early identification of children who are likely to develop an elevated risk profile is of interest.

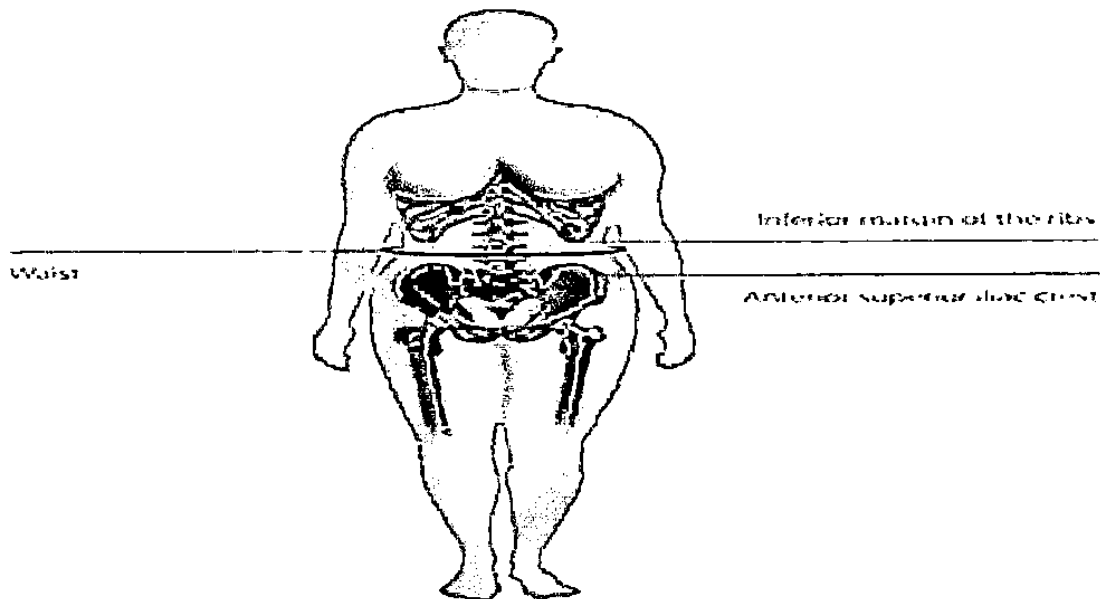
According to WHO (2000), over past 10 years or so, it has become accepted that a high waist-hip ratio (WHR) greater than 1.0 in men and greater than .85 in women, indicates abdominal fat accumulation. However, recent evidence suggests that waist circumference alone may provide a more practical correlate of abdominal fat distribution and associated ill health. European adult men and women with a waist circumference of >102 cm and >88 cm, respectively, are considered to have a higher risk of obesity-related disorders than do those with smaller measurements. While among adult Asian population, waist circumference greater or equal to 90 cm for men and greater or equal to 80 for women referred to be abdominal obesity.

Roberton and South (2006) explained in children and young people, just as in adults, waist circumference is correlated with abdominal fat, as well as with cardiovascular risk factors. While waist circumference charts are available for some individual countries, there are no internationally accepted criteria for high-or low-risk waist circumference in this age group. Of course, as with BMI-for-age percentiles chart, nationally developed waist circumference-for-age charts can be used to monitor the clinical progress of an individual patient. Waist circumference more than or equal to 90th percentile in children and adolescents referred to be as abdominal obesity

(McCarthy et al. 2001; Fernandes et al. 2004)

b. Weight Circumference Measurement

Waist circumference is measured to the nearest 0.1 cm at the mid-point between the lower costal border and the top of the iliac crest with the measurement taken at the end of a normal expiration (WHO, 2000; Liu et al., 2010). The subject was standing up with feet distance as long as 25-30 centimeters without shoes (Han et al, 2001 cit Perdana, 2008). Following picture describe waist circumference measurement:



Picture 2. Guide for measuring waist circumference
(International Diabetic Federation, 2006)

4. Blood Pressure

a. Definition and Regulation of Blood Pressure

Blood pressure means the force exerted by the blood against any unit area of the vessel wall and it almost always is measured in millimeters of

mercury (mmHg) (Guyton and Hall, 2006). In other hand, blood pressure defines as proportional to cardiac output and peripheral vascular resistance (Kumar at al., 2005). It is determined by cardiac output, blood volume, and vascular resistance (Tortora and Derrickson, 2009).

Cardiac output is the amount of blood pumped by the heart each minute and equals heart rate multiple by stroke volume (Copstead and Banasik, 2005). Assuming no other changes in the system, an increase in either stroke volume or hearth rate can increase blood pressure by increasing cardiac output. Factors that influence stroke volume include preload, contractility, and afterload (Tortora and Derrickson, 2009).

Another determinant of blood pressure is systemic vascular resistance. Salidin (2010) explained systemic vascular resistance can be calculated by using a derivation of the Poiseuille law, which states that resistance in a rigid tube with laminar flow is determined by the radius of the tube, the length of the tube, the viscosity of the fluid in the tube, and several constants.

In human body, the viscosity of the blood flowing through the system and length of the system are relatively constants (Copstead and Banasik, 2005). Therefore, the radius of the arteriolar diameter is the major determinant of resistance. The high resistance in the arteriols dampens the pulsatile flow of blood initiated by ventricular contraction, and thus flow is constant rather than pulsatile in the capillaries (Saladin, 2010)

Tortora and Derrickson (2009) explained blood pressure is also depends on the total volume of blood in the cardiovascular system. The normal volume of blood in an adult is about 5 liters. Any decrease in this volume, as from hemorrhage, decreases the amount of blood that is circulated through the arteries each minute. A modest decrease can be compensated for by homeostatic mechanisms that help maintain blood pressure, but if the decrease in blood volume is greater than 10% of the total, blood pressure drops. Conversely, anything that increases blood volume such as water retention in the body tends to increase blood pressure.

According to Waugh and Grant (2006), the systemic arterial blood pressure, usually called simply arterial blood pressure, is the result of discharge of blood from the left ventricle into the already full aorta. When the left ventricle contracts and pushes blood into the aorta the pressure produced within the arterial system is called the systolic blood pressure. When complete cardiac diastole occurs and the heart is resting following the ejection of the blood, the pressure within the arteries is called diastolic blood pressure.

Tortora and Derrickson (2009) systolic blood pressure is the highest pressure attained in arteries during systole, and diastolic blood pressure is the lowest arterial pressure during diastole. As blood leaves the aorta and flows through the systemic circulation, its pressure falls progressively as the distances to about 35 mmHg as blood passes from systemic arteries through systemic arterioles and into capillaries where the pressure fluctuations

disappear. At the venous end of capillaries, blood pressure has dropped to about 16 mmHg. Blood pressure continues to drop as blood enters systemic venules and then veins because these vessels are farthest from the left ventricle. Finally, blood pressure reaches 0 mmHg as blood flows into the right ventricle.

b. Control of Blood Pressure

According to Salidin (2010), vasomotion is a quick and powerful way of altering blood pressure and flow. There are three ways of controlling vasomotion included local, neural, and hormonal mechanisms. Local control included autoregulation, neural control included cardiovascular center, while hormonal control included angiotensin II, aldosterone, atrial natriuretic peptide, antidiuretic hormone, and epinephrine and norepinephrine hormone.

Autoregulation means the ability of tissues to regulate their own blood supply. According to the metabolic theory of autoregulation, if a tissue inadequately perfused, it becomes hypoxic and its metabolites accumulate, lactic acid, and adenosine, for example. These factors stimulate vasodilatation, which is increases perfusion. As the bloodstream delivers oxygen and carries away the metabolites, the vessels reconstrict.

The cardiovascular centre (CVC) is a collection of interconnected neurons in the brain and is situated within the medulla and pons. The CVC receives, integrates, and coordinates inputs from baroreceptors (pressure

receptors), chemoreceptors, and higher centers in the brain or medullary ischemic reflex (Waugh and Grant, 2006).

Tortona and Derrikson (2009) explained baroreceptors in the carotid sinus and in the arch of the aorta are sensitive changes in mean arterial pressure (MAP) and help regulate blood pressure by influencing both cardiac output and systemic vascular resistance. The baroreceptors send impulses to a cardiovascular control center in the medulla of the brainstem. The baroreceptors alter their rate of discharge in response to changes in MAP. An increase in pressure causes an increase in impulse discharge from the baroreceptors. An increased baroreceptors firing rate causes a decrease in sympathetic outflow, as well as an increase in parasympathetic outflow to the heart and vasulature, thus slowing the heart rate and causing vasodilatation. These changes result in a decrease in arterial blood pressure.

Copstead and Banasik (2005) explained circulating hormones can also affect blood pressure. For example, epinephrine and norepinephrine are released from the adrenal medulla in response to low MAP, stress, and hypoxia. The effects of these hormones are the same as those of direct sympathetic nervous system stimulation such as vasoconstriction, increased heart rate, and increased myocardial contractility. These mechanisms work to raise blood pressure.

According to McCance at al., (2010), the rennin-angiotensin-aldosterone system is also involved in blood pressure control. Renal

hypoperfusion, decreased sodium delivery and sympathetic activation stimulate the release of rennin from the juxtaglomerular cells in the kidney. Rennin acts on circulating angiotensinogen to produce angiotensin I. Angiotensin-converting enzyme (ACE), found primarily in the vasculature of the lungs, converts angiotensin I to angiotensin II, one of the most potent vasoconstrictors known being four to eight times as active as norepinephrine. In addition, angiotensin II promotes sodium and water retention by the kidney directly and by stimulating the release of aldosterone from the adrenal cortex. The resulting expansion of volume along with the vasoconstriction increases blood pressure.

According to Salidin (2010), atrial natriuretic peptide (ANP) is released from atrial myocytes in response to stimulation of stretch receptors by excess volume. Release of ANP results in an increased glomerular filtration rate, excretion of sodium and water, and vasodilation. In addition, ANP inhibits the secretion of rennin, aldosterone, and vasopressin. These actions result in reduction of blood pressure. A defect in ANP regulation may contribute to hypertension.

Lip and Hall (2007) explained vasopression, or antidiuretic hormone, which is released from the posterior pituitary gland, has a direct vasoconstricting effect. In addition, vasopressin helps regulate volume by

stimulating the release of rennin from the kidney. These actions result in an

According to Copstead and Banasik (2005), other vasoactive substances such as endothelin-1, which is found in many tissues, and thromboxane A₂, derived from platelets, contribute to vasoconstriction. Nitric oxide, produced by arterial and venous endothelium, kinins, prostacyclin, and histamine all contribute to vasodilatation. Angiotension II decreases nitric oxide production and enhances endothelin release, increasing blood pressure.

c. Classification of Blood Pressure

Copstrad and Banasik (2005) explained a classification scheme for blood pressure has been developed by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7). Blood pressure classification is listed in table below:

Table 2.3. Classification of Blood Pressure for Adults

Category	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)
Normal	<120	<80
Prehypertension	120-139	80-89
Hypertension, stage 1	140-159	90-99
Hypertension, stage 2	≥160	≥100

Source: *U.S.Department of Health and Human Services,2003 (Copstead and Banasik, 2005)*

In children and adolescent, classification of blood pressure is determined by percentile for age. It describes in table below:

Table 2.4. Classification of Blood Pressure for Children and Adolescents

Category	Blood Pressure (percentile)
Normal	Systolic and diastolic blood pressure <90 th
High-normal	Systolic and diastolic blood pressure ≥90 th and <95 th
Hypertension	Systolic and diastolic blood pressure ≥95 th

Source : *Update on the task force report (1987) on high blood pressure in children and adolescents: a working group report from the National High Blood Pressure Education, 1996(Copstead and Banasik, 2005)*

d. Blood Pressure Measurement

Tortona and Derrikson (2009) explained that blood pressure is usually measured in the brachial artery in the left arm. The device used to measure blood pressure is a sphygmomanometer. It consists of a rubber cuff connected to a rubber bulb that is used to inflate the cuff and meter that registers the pressure in the cuff.

If possible, the patient's arm should be at the level of the heart and supported. As the brachial or radial artery is palpated, the cuff is inflated while the observer notes at what level the pulse disappears (James and Ashwill, 2007). When pressure is measured in the arm, the patient should be resting and relaxed, in a quiet room, and either sitting or supine (Rudolph et al., 2002).

Copstead and Banasik (2005) explained the technique for obtaining an auscultated blood pressure is similar to that for palpating the pulse to determine blood pressure. After the cuff is applied and inflated to 30mmHg

above the patient's selected systolic blood pressure, the observer will

deflated while the observer listens through the stethoscope placed over the brachial artery. The return of blood flow through the artery is signaled by Korotkoff sounds, named after the Russian physician who first described them. The sound are to turbulent through the partially occluded artery.

Table 2.5. Korotkoff Sounds

Phase	Description
I	Initiation of clear tapping sounds (systolic blood pressure)
II	Murmuring or swishing sounds
III	Increase in intensity and crispness of sound
IV	Muffling of sounds
V	Disappearance of sounds (diastolic blood pressure)

Source: *Copstead and Banasik (2005)*

Phase I is associated with the onset of tapping sounds heard through the stethoscope and is recorded as the systolic blood pressure. In the past, phase IV was used as the diastolic blood pressure in the children. Recent studies have determined that phase V, the disappearance of Korotkoff sounds, should be used for diastolic blood pressure in both children and adults.

e. Factors that Alter Blood Pressure Level

Alteration on blood pressure level could be defined as either more than normal or less than normal. Blood pressure level which is more than normal (hypertension) or less than normal (hypotension) occurred due to several conditions.

Factors that increase blood pressure level according to Copstead and Banasik (2005); and Barth and Motfin (2000) included family history, age

factors, race, obesity, nutritional factors, smoking, and several diseases, such as renal disorder, hypovolemia and shock.

(1) Family History

Porth and Matfin (2009) explained that the inclusion of heredity as a contributing factor in the development of hypertension is supported by the fact that hypertension is seen most frequently among persons with a family history of hypertension. The strength of the prediction depends on the definition of positive family and the age of the person at risk.

(2) Age

Lip and Hall (2007) explained blood pressure rises consistently with age, beginning at levels as low as 50/40 mmHg in newborns and increasing to over 200 mmHg in some elderly subjects. Many vascular changes occur with aging process. Both systolic and diastolic blood pressure increase with age.

(3) Race

Copstead and Banasik (2005) explained that hypertension in African-Americans is among the highest in the world. They have higher rates of stage 2 hypertension than whites with respectively higher cardiovascular mortality rates than the general population. Furthermore, there is earlier evidence of target organ damage such as increased left ventricular mass than whites with similar blood pressure levels. Although the exact cause

for these differences is unclear, salt sensitivity, elevated endothelin levels, and decreased rennin levels have been proposed as possible factors.

(4) Nutritional factors, life style and disease contributions

High sodium levels and excess alcohol consumption also alter levels of blood pressure. According to Copstead and Banasick (2005), the mechanism of sodium-related hypertension is multifactorial including elements of the rennin-angiotensin-aldosterone system, nitric oxide, catecholamines, endothelin, and atrial natriuretic peptide. Salt restriction decreases the development of left ventricular hypertrophy and reduces aortic stiffness. This condition also occurs with several diseases, such as renal diseases that alter blood pressure.

According to Copstead and Banasick (2005), other factor that alters blood pressure is smoking. Chronic smoking habits had the responsible in altering blood pressure. This condition commonly happened in adult. In children and adolescent, there is no significant alteration of blood pressure caused by smoking. Smoking alters blood pressure in short periods, smoking will increases blood pressure in early 30 minutes. The alteration of blood pressure is not happened in 30 minutes after smoking.

(5) Hypovolemic and Shock

Lip and Hall (2007) explained that hypovolemic and shock altered blood pressure. Hypovolemic means body tissues require more blood flow

for their survival. When blood lost less than 1500 milliliters, blood

pressure tend to be normal. Moreover, when blood loss more than 1200 milliliters, blood pressure increases. The mechanisms included body's compensation.

(6) Obesity

Kumar et al., (2005) also explain factors that altered blood pressure level. One of them is obesity which is associated with elevated levels of blood pressure. Obesity in childhood is a predictor of high blood pressure in adulthood. Obese persons are three times more likely to develop hypertension, with young adults having 5.5-fold increased likelihood of developing hypertension.

McCance et al (2010) explained that obese individuals, especially those with visceral obesity, often have mild to moderate increases in plasma rennin activity, angiotensinogen, ACE activity, ANG II, and aldosterone levels. Activation of the RAS in obese subject occurs despite sodium retention, volume expansion, and hypertension. Visceral obesity increases blood pressure by activation of the sympathetic nervous system, the rennin-angiotensin-aldosterone system, and by physical compression of the kidney from the fat surrounding the kidneys.

f. Measurements errors of blood pressure

Lip and hall (2007); Copstead and Banasik (2005) explained a number of factors can affect blood pressure measurements. These factors are of two

major types. They are technical factors and factors involving the subjective influence of observer.

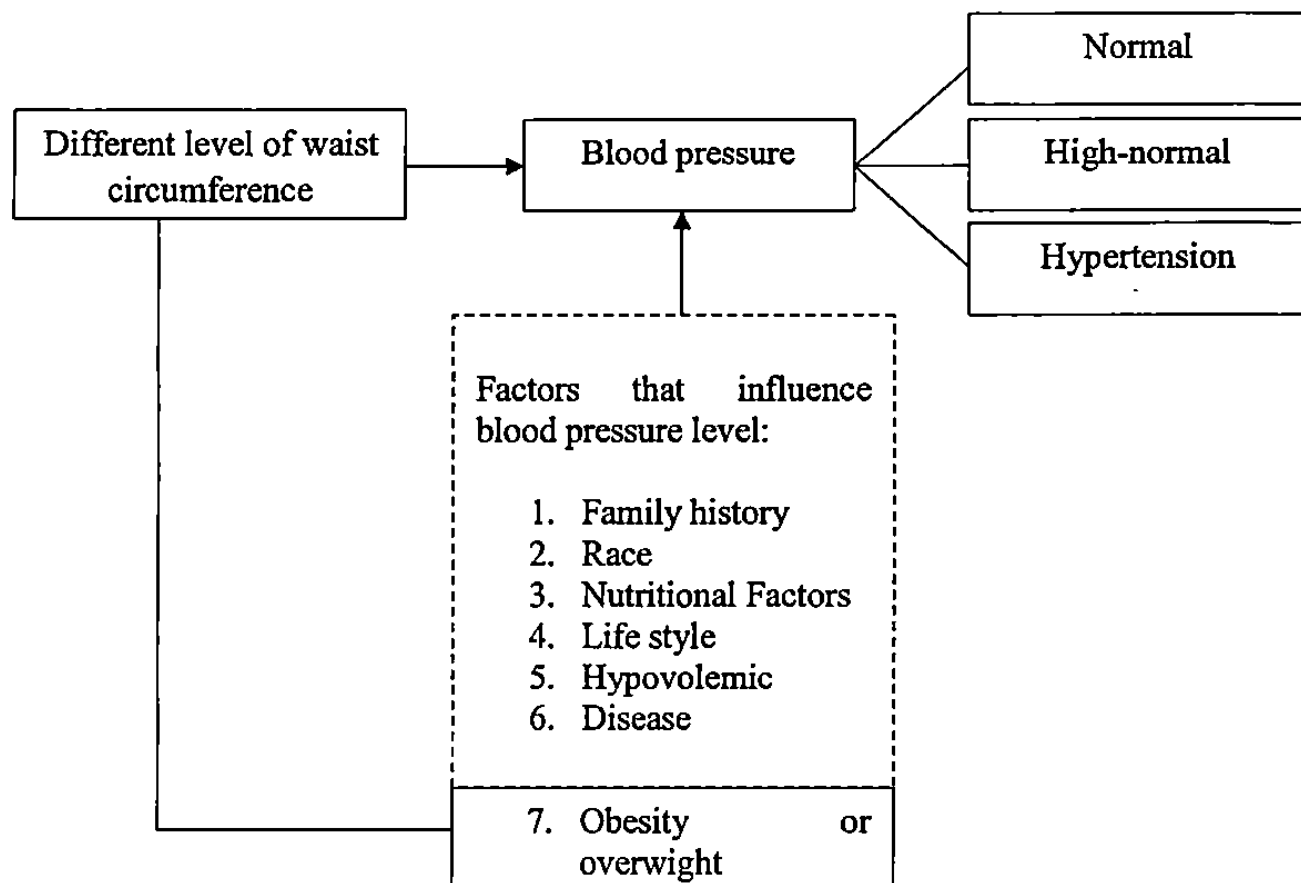
Copstead and Banasick (2005) explained technical aspects include such things as body positioning and size of the cuff used. If the arm used for blood pressure measurement is positioned above the level of the heart, the blood pressure may be artificially lowered. If the arm is positioned below the heart, blood pressure may be artificially raised. If the arm is unsupported, isometric activity by the patient in an effort to keep the arm elevated may result in falsely high readings.

According to Lip and hall (2007), The American Heart Association recommends the use of a cuff with a bladder width that is 40% of the circumference of the midpoint of the limb. Use of cuff with bladders that are too narrow results in falsely elevated blood pressure readings. Use of bladders that are too wide results in erroneously low readings.

Table 2.6. Technical, Subjective, and Other Factors that Influence Blood Pressure Readings

Factors	Effect on Blood Pressure
Technical Factors	
Cuff	
Too wide, too long	BP falsely low
Too narrow, too short	BP falsely high
Arm position	
Above heart	BP falsely low
Below heart	BP falsely high
Unsupported	BP falsely high
Excessive pressure on head of stethoscope	Diastolic progressively decreased with increased pressure
<1 min between readings	BP falsely high
Subjective Factors	
Terminal digit preference	Tendency to end BP in zero
Observer bias	Record BP higher or lower, depending on recorder's biases
Poor hearing	Altered reading
Other Factors	
"White Coat" phenomenon	Higher BP reading when measured by physician
Eating, smoking, taking caffeine, exercising within 30 min of BP	Increased BP
Sneaking while BP measured	Increased BP

B. Conceptual Framework



C. Hypothesis

Ho : There is no correlation between waist circumference and blood pressure among overweight students SMA Muhammadiyah 3 Yogyakarta.

Ha : There is a correlation between waist circumference and blood pressure among overweight students SMA Muhammadiyah 3 Yogyakarta.