

CHAPTER II

LITERATURE REVIEW

Body mass index classification

Obesity has been identified using the body mass index (BMI). Body mass index is a measure of body fat based on height and weight that applies to both adult men and women 20 to 65 years of age. Body mass index is calculated by dividing a person's weight in kilograms by the square of his or her height in meters (12). The body mass index classification for Asian descents is different from a classification for European population. For example, for Asian people to be classified as obese, the body mass index cutoff is at 25 kg/m^2 , whereas as for the Europeans the body mass index of 30 kg/m^2 is defined as obese(13). Comparisons of body mass index categories for Asian and Europids are shown in Table 1.

Table 1 Body mass index classification for Asian and Europids.

BMI categories for Asian		BMI categories for Europids	
Classification	Body mass index (kg/m ²)	Classification	Body mass index (kg/m ²)
Underweight	< 18.5	Underweight	< 18.5
Normal weight	18.5 to 22.9	Normal weight	18.5 to 24.9
At-risk of obesity	23.0 to 24.9	Pre-obesity	25.0 to 29.9
Obesity class I	25.0 to 29.9	Obesity class I	30.0 to 34.9
Obesity class II	≥ 30.0	Obesity class II	35.0 to 39.9
		Obesity class III	≥ 40.0

BMI is widely used a criterion for assessing obesity for two main reasons. First, BMI correlates significantly with body fat, morbidity, and mortality. Second, it can be calculated quickly and easily in clinical setting. Furthermore, recommendations for treatment of obesity are based on BMI. BMI of 25 kg/m² is the generally accepted for identifying a patient at higher risk for obesity-related diseases. Risk of death begins to increase at a BMI of 23 kg/m² when compared with the lowest risk group (BMI, 19.0 to 21.9 kg/m²). Medical risk rises with increasing degrees of obesity. More than 80% of deaths estimated to be caused by morbidities related with obesity occur in patients with a BMI of at least 30 kg/m² (12).

Health conditions associated with overweight and obesity

Excess body fat is associated with an increased risk of health problem, including hypertension, coronary heart disease (CHD), stroke, and osteoarthritis. Diseases associated with obesity maybe arise from two mechanisms: from the metabolic changes associated with excess fat, as type 2 diabetes mellitus and cardiovascular disease, or from the increased fat mass itself, as it is clearly the case for joint diseases. Obesity can affect almost all organs and tissues of the body, from the brain to the lower extremities, causing a multitude of clinical problems (14, 15).

Type 2 diabetes mellitus

Individuals who are overweight or obese increase in risk for type 2 diabetes mellitus. Women with a BMI in the average range (24 to 24.9 kg/m²) had up to a 5-fold elevated risk when compared with women with a BMI < 22 kg/m². In patients with a BMI of 31 kg/m², the risk of type 2 diabetes increased by more than 40-fold. High body weight is a strong predictor of risk for diabetes (14).

The obesity is believed to play a primary role in the development of type 2 diabetes. Although alterations of muscle-fuel metabolism are central in insulin resistance related to obesity, all other major glucose-regulatory tissues including liver and fat cells are affected. It is believed that obesity induced insulin resistance is mediated in part by high levels of free fatty acid and tumor necrosis factor released by excess adipocytes. The elevation of free fatty acid levels can inhibit muscle glucose utilization, increase hepatic glucose output, and stimulate insulin secretion from pancreatic β -cells, causing hyperinsulinemia (15).

Hypertension

Obesity and hypertension are comorbid risk factors for the development of cardiovascular disease. In adults with a BMI $> 30 \text{ kg/m}^2$, the prevalence of high blood pressure (BP) was 38% in men and 32% in women, as compared with 18% in men and 16% in women with a BMI $< 25 \text{ kg/m}^2$ (14). It was estimated that a 1 kg increase in weight is associated with a 5% increase in risk. An additional 10 kg in body weight was associated with higher BP, with increases of 3.0 mmHg insystolic and 2.3 mmHg in diastolic BP (1).

The development of hypertension associated with obesity is related to a combination of increased sodium retention, increased sympathetic nervous system activity, alterations of the rennin-angiotensin-aldosterone system and insulin resistance. In obesity-related hypertension, the cardiovascular abnormalities produced by sodium retention and intravascular volume expansion, which induces an increase in venous return and cardiac output, as well as an increase in peripheral vascular resistance, are well described. The maintenance of hypervolemia with hypertension implies a resetting of pressure toward higher BP. These changes in the cardiovascular system and the kidney may be related to insulin resistance, the enhancement in sympathetic nervous activity, and the activation of the renin-angiotensin-aldosterone system. In addition, the renal medulla of obese persons demonstrates histologic changes including interstitial cell proliferation and deposition of noncellular matrix, which can lead to compression of tubules and vasa recta, increased sodium reabsorption (14, 15).

Coronary heart disease (CHD)

The obesity and excess abdominal fat are directly related to cardiovascular risk factors. Obesity and abdominal fat are associated with increased morbidity and mortality from CHD. Relative risks for CHD are twice as high at BMI of 25 to 28.9 and more than three times as high at BMI of 29 or greater, compared with BMI of less than 21 (1).

Obesity, particularly of the abdominal type, and sedentary life style may induce excessive CHD morbidity and mortality through several mechanisms. First, obesity promotes the development of multiple morbid conditions that are independent risk factors for CHD, including type 2 diabetes, hypertension, and dyslipidemia. Second, obesity promotes the clustering of these risk factors, thus increasing the risk exponentially. Third, obesity has an effect on the cardiovascular system that appears to be independent of diabetes, hypertension and dyslipidemia. In addition, obstructive sleep apnea, hyperinsulinemia, and hemorheologic abnormalities including elevated plasminogen activator inhibitor-1 (PAI- 1) levels and high blood viscosity may contribute to the pathogenesis of coronary atherosclerosis in obese persons. High levels of PAI-1, produced by visceral fat, cause impaired fibrinolytic activity and an increase in the extension of thrombosis (1, 14).

Recent clinical studies have shown a correlation between BMI and coronary events, including nonfatal myocardial infarction, angina pectoris, and death from CHD. The greater requirement in blood flow to supply expanding adipose tissue increases cardiac workload, total body oxygen consumption, and cardiac output, which lead to

ventricular dilatation and hypertrophy. The combination of the high cardiac workload and hypertension predisposes obese patients to develop CHD (1).

Stroke

Obesity and overweight person might contribute to risk of stroke, independent of the known association of hypertension and diabetes with stroke. However, the risk of stroke shows a graded increase as BMI rises (1).

The association between obesity and stroke remains controversial, although most studies have shown a positive relation. Increasing BMI is associated with a rise in the risk of total, ischemic and hemorrhagic stroke independently of the presence of hypertension, diabetes and dyslipemia. Other studies have underlined abdominal obesity as an independent risk factor for ischaemic stroke in all race-ethnic groups, being a stronger risk factor than BMI and with a greater effect among young subjects. Plasma leptin has been considered a risk factor for first-ever haemorrhagic but not ischaemic stroke, in an independent manner of other risk markers for CVD. If we consider that most obese people have leptin resistance with high levels of plasma leptin, we could speculate that leptin might be the link between obesity and haemorrhagic stroke (15).

Osteoarthritis

Individuals who are overweight or obese increase their risk for the development of osteoarthritis. Survey data show that adults in the United States with a BMI > 30 kg/m² have over a 4-fold higher prevalence of radiographic knee osteoarthritis than those with a BMI < 30 kg/m². In middle-aged women, it was estimated that for every kilogram increase of weight, the risk of developing osteoarthritis increases by 9 to 13 percent. An

increase in weight is significantly associated with increased pain in weight-bearing joints (1, 16). Mechanical trauma associated with excess body weight contributes to osteoarthritis, whereas arthritis of joints that do not bear weight is probably due to some systemic factors secreted in obese persons that cause abnormalities of bone and cartilage metabolism (14).

Normal gait cycle (17-19)

The gait cycle is the rhythmic alternating movements of the two lower extremities which result in the forward movement of the body. Cycle begins when heel contacts the ground and ends when it contacts the ground again. The gait cycle is subdivided into a stance phase and a swing phase. Figure 1 shows phase of gait cycle (20). The stance phase takes about 60% of the gait cycle and the swing phase about 40%.

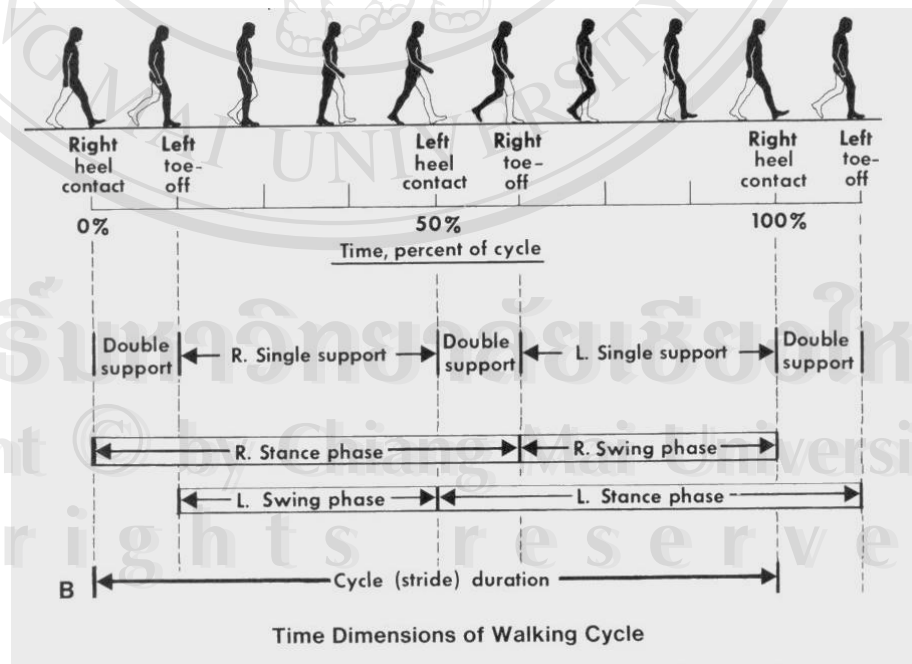


Figure 1 Phase of gait cycle.

Phase of gait cycle

1. Stance phase is the period of time when the foot is in contact with the ground. During the stance phase, the lower limb has to provide a semirigid support for the body weight to maintain balance and allow to forward propulsion. The stance limb has a role in compensating for uneven ground and when positioned correctly, it enables an accurate swing phase on the contralateral limb to take place. The stance phase can be divided into 5 stages.

1.1.Initial contact

At the moment of initial contact the following limb is also in contact with the floor, the limb is optimally positioned to initiate both progression and knee stability. This is moment when the whole body center of gravity is at its lowest and the walker is most stable. At initial contact, the ankle is a neutral position. Knee joint is slight flexion helps absorb the impact of the foot contacting the ground. Hip flexion helps provide for proper placement of foot so that the heel make contract with the ground.

1.2.Loading response

This phase begins with initial floor contact and continues until the other foot is lifted for swing. Loading response is the phase of greatest muscular activity, since demands in all three planes must be controlled. The whole foot comes into contact with the floor, allowing it to accept the weight of the body as the mid-stance phase take place. During initial contact and loading response there is rapid loading of the limb and absorb the sudden imposition of ground reaction force. During loading response, knee flexes 15 degrees while ankle plantarflexes 15 degrees, which is energy conserved mechanism.

This phase hamstrings and ankle dorsiflexors remain active. Then, quadriceps and gluteal muscles act during loading and throughout early mid-stance to maintain hip and knee stability

1.3. Mid-stance

It begins as the other foot is lifted and continues until body weight is aligned over the forefoot. This phase is the time when the body weight line changes anterior/posterior alignments at each joint. The center of gravity passes from behind to in front of the stance foot and rises to higher position in relation to the supporting surface. This is the position when the walker is least stable due to the small base and the relatively high center of gravity. In this phase, the knee is extended and ankle is neutral again. The gastrocnemius acts to control tibialis anterior for preventing the tendency for the ankle to dorsiflex due to body weight and inertia.

1.4. Terminal stance

It begins with heel rise and continues until the other foot strikes the ground. Throughout this phase body weight moves ahead of the forefoot.

1.5. Pre-swing

It begins with initial contact of the opposite limb and ends with ipsilateral toe off. At preswing, knee flexes 35 degrees and ankle plantarflexes 20 degrees. In the last phases of stance, the toes have been neutral and the metatarsophalangeal joints dorsiflex.

2. Swing phase is the period of time when the foot is not in contact with the ground. During swing phase, the swing limb moves in front of the stance limb so that forward progression may take place. The limb must be shortening sufficiently to enable

the foot to clear the ground and this is achieved by flexion of the hip and knee joints and dorsiflexion of the ankle. In normal adult gait the clearance of the foot from the ground is successful swing phase but in order to conserve energy it is important that the limb is not lifted further than is necessary. The swing phase can be divided into three stages.

2.1. Initial swing

It begins with lift of the foot from the floor and ends when the swinging foot is opposite the stance foot. This phase has contraction of knee flexion because due to foot clearance of the floor and advancement of the limb from its trailing position. And the force generated by the hip flexors and to lesser extent by the plantarflexors accelerates the non weight bearing limb forward.

2.2. Mid-swing

It begins as the swinging limb is opposite the stance limb and ends when the swinging limb is forward and the tibia is vertical. This corresponds with mid-stance and at the moment the swing phase limb passes the stance limb it is at its shortest.

2.3. Terminal swing

It begins with a vertical tibia and ends when the foot strikes the floor. Limb advancement is completed as the leg move ahead of the thigh. Hamstrings muscles become active to decelerate forward swing of the leg and thereby control the position of the foot at heel strike. The muscle action in this phase is usually eccentric and requires less energy than those times in the gait cycle when concentric activity is needed to accelerate a limb.

Other temporal and spatial components

1. Cadence is used to indicate the number of steps per a minute. The cadence mainly depends on the velocity of walking.

2. Stride length is distance between successive foot-floor contacts with the same foot. Figure 4 demonstrates stride length.

3. Step length is distance between successive foot-floor contacts with opposite feet. Figure 2 demonstrates step length.

Stride and step length are dependent on several factors including the length of the lower limb, the age of the subject and the velocity of walking. Short lower limb length, increasing age and decreasing velocity will reduce stride and step length.

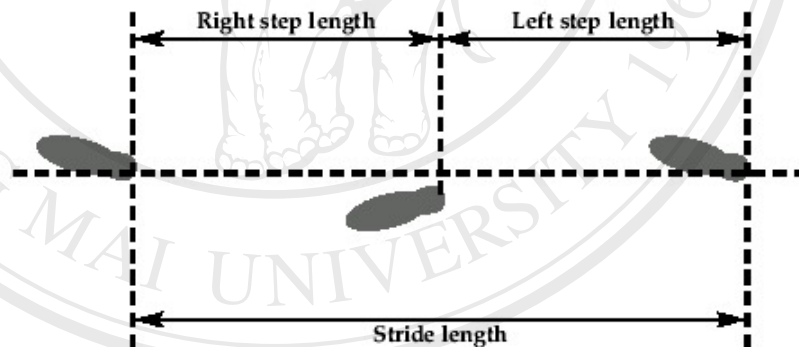


Figure 2 Step and stride lengths (21)

Gait characteristics of obese persons

Temporospatial variables

Obese persons walked at slower speed than the nonobese persons. The slower speed of the obese subjects was attributed to both their slower cadence and shorter stride length. A possible explanation for a slower gait pattern of the obese persons could be that

they take shorter stride in order to maintain balance, the obese subjects also had a larger step width than the nonobese subjects. The increased step widths of the obese group may be the result of excessive amounts of adipose tissue between the thighs in addition to providing a larger base of support for better body balance during walking (4-6).

Obese persons spend greater percentage of time in double support and stance time than nonobese persons. The greater support time is indicative of a safer and more tentative ambulation that reduces the nonsupport period and the possibility of instability and obese group might have been generating adequate push off force muscularly to overcome inertia due to the mass of the stance limb and the obese group decreased plantar flexion of the ankle (4-6).

Obese subjects showed a degree of asymmetry in comparisons of left and right limbs. The asymmetry displayed by obese children may be related to their body composition and affected by their speed of walking (4, 5).

Joint angle displacement

The obese group demonstrated a lesser magnitude of hip flexion at heel strike as well as a lesser mean magnitude of hip flexion during swing for the nonobese group for possible reasons: (a) the obese subjects' swing time was reduced, and (b) due to the excessive body weight, obese persons lack the muscular effort and neuromuscular control to bring about greater flexion that may have threatened body balance (4, 6).

The obese subjects presented a greater mean magnitude of hip abduction than the nonobese group throughout the walking cycle. This abducted walking pattern throughout the walking cycle for the obese group as well as their hip external rotation at

heel strike may be due in part to the excessive adipose tissue between their thighs, and may be an attempt to widen the base of support to facilitate proper balance (6).

The reduction in knee flexion during swing phase contributed to minimal toe clearance for the obese group (4). The obese persons demonstrated greater magnitudes of dorsiflexion and lesser magnitudes of plantar flexion than nonobese persons. This appears to be directly related to the reduced hip flexion angle at heel strike in the obese group. The diminished hip flexion angle coupled with the reduced stride length of the obese subjects allowed the obese persons to accept the weight of their body by moving their ankles a lesser magnitude of plantar flexion compared with nonobese group. This leg and foot position may also be a mechanism to bring the body weight over the flat foot as soon as possible to aid balance in the obese subjects. The push off period for the obese subjects was characterized by a smaller magnitude of plantar flexion than nonobese group. This diminished magnitude of average plantar flexion during the push off period contributes to a diminished push off force, a reduced swing period, and the smaller stride lengths observed in the obese subjects (6).

Kinematic comparison of treadmill and overground walking

Alton et al. (9) compared overground and treadmill ambulation for possible differences in gait temporal variables and leg joint kinematics. They found greater hip range of motion, greater hip flexion, greater cadence, and lesser stance time in treadmill ambulation than overground.

The authors pointed out that the reduction of stance time on the treadmill resulted from increase in cadence in order to maintain walking velocity. The higher cadence could be caused by place the foot of the swing limb onto the treadmill as the stance limb is carried backwards by the treadmill.

The greater hip ROM in treadmill walking resulted from greater hip flexion angle. Considering all of the changes in gait from overground to treadmill walking together, the subjects were using hip flexion more, and slightly less extension while reducing stance time, perhaps as a means of avoiding falling off the back of the treadmill and keeping up with the belt speed.

Effects of slope on gait characteristics

Leroux et al. (10) investigated the postural adaptation to uphill and downhill walking in healthy subjects (aged 25-52 year) on treadmill. Increasing the treadmill slope from 0 to 10% uphill led to increase in hip, knee and ankle flexion at initial foot contact as well as a progressive forward tilt of pelvis and trunk and a gradual increase in stride length.

The postural adaptation to inclined walking is observed (10). There are changes in trunk and pelvic orientations related to vertical in the sagittal plane in order to maintain the same patterns of movement across the different treadmill inclinations. Trunk and pelvic alignment showed a forward tilt during uphill walking as compared with level walking because the central nervous system assist in propelling the body forward. By inclining the trunk forward, subjects would move the center of mass slightly ahead of the

foot and the center of gravity would tend to accelerate the body forward relative to the base of support. Trunk tilt during walking can assist the lower limbs in generating forward momentum. The need for generating this greater momentum is reflected by a progressive increase in stride length as the treadmill slope becomes steeper in uphill walking. Changes in pelvic orientation in the sagittal plane were mainly performed to assist hip movements during swing phase and gravity affected to hip movements and contribute to smoothing the gait. The key mechanism when adapting to uphill walking is to lift up the swinging leg by performing a simultaneous increase in hip and knee flexion of the limb.

Gait symmetry and limb dominance

According to the results of the study by Hannah et al. (12) and Gundersen et al. (13), for 2-D motion analysis, movement can be viewed at either side of the body. Hannah et al. (22) examined the extent of kinematic symmetry of lower limbs when walking. Comparisons of right and left sagittal, transverse, and coronal plane motions were examined in the time and frequency domains, and indices were developed to express these symmetries in each domain. This study was found high levels of symmetry in all three planes of the hip and in the sagittal plane of the knee during natural walking. Reduced symmetry in the transverse and coronal planes was attributed to the lower signal to noise ratio for these planes. However, the able-bodied individual walks with reasonable kinematic symmetry at hips and knees.

Gundersen et al. (23) examined the relationship between lower extremity dominance and kinematic symmetry during gait. Fourteen healthy volunteers without any observable gait deviations participated in the study. The subjects (8 male, 6 female) ranged in age from 19 to 56 years. Lateral dominance is one factor that was thought to play an important role in understanding asymmetries in gait. This study demonstrated significant differences within-subject analysis; however, lateral dominance could not be related predictably to these variations. Limb dominance was more thorough in including both stability and mobility tasks; but these are not actual events occurring in the gait cycle. Although limb dominance may not influence noncortical automotive activities for which both legs are required to carry out the same sequence of tasks. A healthy person's typical gait pattern may not be stressful enough to emphasize lateral dominance in the lower extremity.

Physical activity questionnaire

The present study will include a short physical activity questionnaire used in the European Prospective Investigation into Cancer and Nutrition (EPIC) study. The validity and repeatability of the questionnaire has been found to be high (weighted kappa = 0.6, $P < 0.0001$). Wareham et al. (24) assess validity and repeatability of a simple index designed to rank participants according to their energy expenditure estimated by self-report, by comparison with objectively measured energy expenditure assessed by heart-rate monitoring with individual calibration subjects aged 40-65 years participated in the study. A simple four-level physical activity index was derived by combining occupational

physical activity together with time participating in cycling and other physical exercise. Energy expenditure was assessed over one year by four separate episodes of 4-day heart-rate monitoring, a method previously validated against whole-body calorimetry and doubly labeled water. Cardio-respiratory fitness was assessed by four repeated measures of submaximum oxygen uptake. At the end of the 12-month period, participants completed a physical activity questionnaire that assessed past-year activity.

The repeatability of the physical activity index was high. There were positive associations between the physical activity index from the questionnaire and the objective measures of the ratio of daytime energy expenditure to resting metabolic rate and cardio-respiratory fitness. Although it was designed for the EPIC–Europe study, the simplicity and ease of comprehension of the short EPIC physical activity index may make it suitable for other situations where a simple global index of activity is required. For the present study, the EPIC questionnaire will be administered in Thai. (Appendix A)

EPIC physical activity questions

1. We would like to know the type and amount of physical activity involved in your work. Please tick what best corresponds to your present activities from the following four possibilities:

- Sedentary occupation _____

You spend most of your time sitting (such as in an office)

- *or* Standing occupation _____

You spend most of your time standing or walking. However, your work does not require intense physical effort (e.g. shop assistant, hairdresser, guard, etc.)

- *or* Physical work _____

This involves some physical effort including handling of heavy objects and use of tools (e.g. plumber, cleaner, nurse, sports instructor, electrician, carpenter, etc.)

- *or* Heavy manual work _____

This involves very vigorous physical activity including handling of very heavy objects (e.g. docker, miner, bricklayer, construction worker, etc.)

2. In a typical week during the past 12 months, how many hours did you spend on each of the following activities? (Put '0' if none)

- Walking, including walking to work, shopping and leisure
_____ hours per week

- Cycling, including cycling to work and during leisure time
_____ hours per week

- Gardening
_____ hours per week

- Housework such as cleaning, washing, cooking, childcare
_____ hours per week

- Do-it-yourself
_____ hours per week

- Other physical exercise such as keep fit, aerobics, swimming, jogging

_____ hours per week

3. In a typical week during the past year did you practise any of these activities vigorously enough to cause sweating or a faster heartbeat?

Yes _____ No _____ Don't know _____

- If yes, for how many hours per week in total did you practise such vigorous physical activity? (Put '0' if none)

_____ hours per week

4. In a typical day during the past 12 months, how many floors of stairs did you climb up? (Put '0' if none)

_____ floors per day