Chapter 5

Discussion
DISCUSSION

A. Meal Frequency and Cardiovascular Determinants of Blood Pressure:

The current study does not show any significant effect of meal frequency on the physiological determinants of blood pressure as indicated by the insignificant differences in the pulse rate, pulse pressure, mean arterial pressure, cardiovascular reactivity and CVDP. It was hypothesized that meal frequency may be influencing the cardiovascular determinants of blood pressure by affecting the body composition as indicated by previous studies conducted in adults (Fabry et al. 1968 and Stote et al., 2007). But, since meal frequency is not found to show any significant impact on the body composition in this adolescent population, it probably therefore does not show any significant effect on blood pressure.

B. Sleep Duration And Cardiovascular Determinants of Blood Pressure

The current study shows that sleep duration does not affect the arterial blood pressure in the Gujarati adolescents. This is probably because of the finding that there is no significant effect of sleep duration on the resting Sympathetic activity, which is considered to be one of the potential mechanism through which sleep deprivation predisposes individuals to hypertension. These results are contradictory to the findings in other studies conducted in adolescents as reported by Sampei et al and Javaheri et al. A major reason for this discrepancy could probably be the fact that the subjects in the current study have been grouped into ISDN and ASDN groups based on the sleep duration reported by the subjects themselves and was not actually measured. Therefore the
amount of time reported for sleep may not be the actual time, as the subjects reported sleep duration to be the duration from the time the subjects went to bed and woke up in the morning which does not necessarily involve sleep.

However, an important finding which has been surfaced is that, in both genders inadequate sleep duration is significantly associated with higher pressor response to sympathetic stress, lower vascular distensibility and higher total as well as visceral adiposity. Studies conducted in other parts have given concrete evidences regarding the role of sleep deprivation in the causation of obesity (Gangswich et al 2005). Sleep deprivation has been shown to produce neurohumoral consequences like sympathovagal imbalance, excess Cortisol levels, changes in Leptin and Ghrelin level which are probably responsible for sleep deprivation induced weight gain and obesity (Spiegel et al 2004).

Regarding the effect of sleep deprivation on cardiovascular reactivity, some reports indicate that sleep deprivation leads to increase pressor response to stressful stimuli (Bonnet and Arrand) while others report that there is no effect of sleep deprivation on cardiovascular reactivity (Kato et al). But the studies which report the association of sleep deprivation and cardiovascular reactivity have been conducted on adults and therefore the results of the current study need not be similar to them.

Decrease vascular distensibility observed in adolescents with inadequate sleep probably indicates the prevalence of endothelial dysfunction amongst these adolescents due to excess visceral adiposity. The findings of the current study
are important because high visceral adiposity and higher vascular reactivity to sympathetic stress are risk factors for hypertension in children and adolescents as reported by Kaur et al, Thakor et al, Matthews et al, Newman et al and Falkner et al. \(^{(2, 4, 146, 147, 148)}\)

These observations thus indicate that inadequate sleep duration in this population should raise the blood pressure in these adolescents due to the associated increase in vascular reactivity and adiposity. However, this is probably not observed because the effect of inadequate sleep on vascular reactivity, vascular distensibility and adiposity is not large enough so as to cause a significant rise in blood pressure. Nevertheless it is possible that a longer exposure to inadequate sleep may cause hypertension in these adolescents in the future by its effects on adiposity, vascular distensibility and sympathetic vascular reactivity.

**C. Physical Activity Status and Cardiovascular Determinants of Blood Pressure:**

The current study does not show any significant effect of Physical activity status on the cardiovascular determinants of blood pressure in both genders. The study was conducted based on the hypothesis that physical activity would tend to lower the blood pressure by 1. Lowering cardiovascular sympathetic activity, 2. Lowering cardiovascular reactivity 3. Enhancing vascular distensibility and 4. Reduction in Adiposity. However, no significant differences have been found in pulse rate, pulse pressure, diastolic blood pressure, mean arterial pressure,
pressor response to acute isometric exercise and total adiposity (BMI, FMI) between physically active and physically inactive groups. Similar findings have also been reported earlier regarding the role of physical activity in lowering the blood pressure where no relationship was found between physical activity and blood pressure (Brage et al, Rizzo et al, Kvaavik et al and Soudarssanane et al). (69, 70, 73, 82) However, reports are also available that indicate a marginal blood pressure lowering effect of physical activity in adolescents (Leary et al, Gidding et and Fasting et al). (75, 76, 77) The major limitation regarding the reports of most of these studies including the result of current study is that the physical activity has been evaluated based on subjective data. Therefore, studies need to be conducted where in physical activity is objectively assessed using accelerometers (pedometers) over a period of days and than related to other physiological variables.

However, although physical activity does not seem to affect the blood pressure in the Gujarati adolescents, the current study shows that a significant association exists between physical fitness and blood pressure across both genders in this population. This is similar to the earlier reports where physical fitness rather than physical activity has been found to have a beneficial effect on the blood pressure in adolescents (Brage et al, Rizzo et al, Raitakari et al and McMurray et al). (69,70, 71, 79) The probable mechanism through which physical fitness affects the blood pressure in this adolescent population however seems to be gender dependent. In boys, physical fitness seems to act predominantly on the vascular distensibility as indicated by the significant negative correlation of physical fitness with pulse pressure and in girls physical fitness seem to predominantly act on the
sympathetic vascular tone as indicated by the significant negative correlation of physical fitness with mean arterial pressure. The probable reason for these gender differences is the fact that the female sex hormone estrogen is believed to protect the post-pubertal female vasculature from atherosclerosis and make the blood vessels more distensible while the male sex hormone testosterone predisposes the male vasculature to endothelial dysfunction and atherosclerosis.

Ahimastos et al reported the findings of their study which was conducted to determine the gender differences in large artery stiffness in pre and post puberty stages. The study showed that prepubertal males and females did not differ in body size, cardiac output, or heart rate. It also showed that the prepubertal females had stiffer large arteries and higher pulse pressure than age-matched males. However, on the other side, post pubertal males were taller and heavier and had a greater cardiac output and lower heart rate compared with similarly aged females. In relation to pubertal status, females developed more distensible large arteries post puberty whereas males developed stiffer large vessels.

Herman et al have demonstrated that the dilatation of brachial artery in response to increase blood flow (endothelial dependent dilatation) was higher amongst individuals who were having low levels of serum testosterone. This finding suggested that testosterone causes endothelial dysfunction. Ba et al have also reported similar results regarding the effect of testosterone on endothelial function.
Therefore after learning from the above reports it is believed that the dominant effects of physical fitness on the vascular distensibility in males and sympathetic tone in females is probably observed because the sympathetic over activity associated with low physical fitness is masked by the associated decrease in vascular distensibility associated with low physical fitness amongst boys.

D. Body Composition And Cardiovascular Determinants of Blood Pressure

Enough evidence has been found from the current study which indicates a significant effect of body composition on various cardiovascular determinants of blood pressure in the Gujarati adolescent population. Total as well as visceral adiposity as indicated by FMI and WC respectively show a significant positive correlational relationship with SBP and DBP in both genders. However, the cardiovascular determinants which are affected by adiposity seem to differ in both genders.

In Boys, adiposity shows a more stronger and significant positive correlational relationship with vascular distensibility (PP & %RSBP) than with sympathetic vascular tone (DBP and MAP) and does not seem to influence cardiac sympathetic activity (PR). In girls, adiposity shows a stronger and significant correlation with sympathetic vascular tone (DBP) than with vascular distensibility (PP & %RSBP). However, it seems that it is mainly the visceral fat that would be playing a major part in affecting vascular distensibility across both genders as indicated by the significant relationship of WC with %RSBP even in the absence of a significant effect of FMI with %RSBP in girls. The gender difference in the effect of adiposity on the sympathetic vascular tone and vascular distensibility is probably due to the
effects of sex hormones on the cardiovascular system as discussed earlier in relation to the influence of physical fitness on the cardiovascular profile of this population. The probable mechanisms through which obesity leads to sympathetic overactivation are insulin resistance and selective leptin resistance while the mechanisms by which increase adiposity is linked to decrease vascular distensibility are atherosclerosis and endothelial dysfunction. Results similar to the findings of the current study indicate that vascular distensibility is affected by obesity at an age as early as adolescence (Whincup et al 2005, Zebekakis et al 2005 Singhal et al 2002 and Urbina et al 2001). (152,153,154,155)

Another important finding of the study is that adiposity does not affect the sympathetic vascular reactivity (%RDBP) in boys while total as well as visceral adiposity shows a significant positive correlationship with vascular reactivity to stress (%RDBP) in girls. The probable reason for this difference is that girls have lower physical fitness then boys. Since physical fitness is found to have a strong and negative correlationship with sympathetic vascular reactivity, the lower physical fitness along with an increase in adiposity increases the vascular reactivity to stress in girls.

The current study also shows that it is not only the adiposity which tends to affect the blood pressure but lean body mass (FFM) also seems to have a strong influence on the cardiovascular determinants of blood pressure in both boys and girls. In both boys and girls, fat free mass has the predominant effect on the sympathetic vascular tone (DBP, MAP) reflecting the muscle sympathetic nerve activity. Apart from influencing the sympathetic vascular tone it also shows a significant positive correlationship with vascular reactivity which is ought to
happen due to increase muscle mass and therefore a higher activation of sympathetic system.

The study also found total body weight, total adiposity (FMI) and lean body mass (FFMI) to be significant determinants of left ventricle mass (CVDP). However these relationships were found only in girls and not boys. Such relationship of weight, adiposity and lean body mass with left ventricle mass in children and adolescents has been reported earlier. Muscatine study showed that, in boys 72\% variance in LVM was related to FFM, sum of skinfolds and peak SBP during isometric handgrip while in girls, FFM and Peak SBP were the major determinants of LVM.\(^{(156)}\) Trieber et al also found adiposity and peak SBP response to stress to be significant determinants of LVM.\(^{(157)}\) Daniel et al however found that it is mainly the lean body mass which has the major effect on the LVM and not adiposity or SBP.\(^{(158)}\) The probable reason for the presence of an association of adiposity and FFM with LVM in girls and not boys is the fact that in girls adiposity is found to be significantly associated with an increase in the sympathetic vascular reactivity to isometric exercise which is an independent predictor of LVM as reported by Murakami et al.\(^{(159)}\)