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Original Article

Relationship between Adiposity, Blood Pressure, Cardiac Autonomic Function and Arterial Stiffness in Young Healthy Individuals

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ABSTRACT

Background: The precise mechanism linking overweight/obesity with hypertension remains unclear. The present study was conducted (1) to evaluate the impact of adiposity on cardiac autonomic nervous system (ANS) activity and arterial health and (2) to determine the relationship between adiposity, cardiac ANS, arterial stiffness and blood pressure (BP). **Methods:** A cross-sectional study was conducted on 48 young healthy subjects with overweight/obesity (OW group, n=24) and normal weight (NW group, n=24) with age ranging between 18-24 years. Blood pressure, cardiac ANS and arterial health were evaluated. Heart rate variability (LF: low frequency; HF: high frequency; and LF/HF ratio) was assessed as an index of cardiac ANS function. Pulse wave velocity (brachial-ankle, carotid-femoral, heart-ankle, heart-brachial), aortic augmentation index, arterial stiffness index were measured as an indices of arterial stiffness. **Results:** LF and LF/HF ratio was significantly elevated ($p<0.001$) while HF was lowered ($p<0.001$) in obese/overweight subjects when compared to normal subjects. Though not significant but a mean increase in arterial stiffness was found in OW group when compared to NW group participants. Relationship between covariates in all the participants (n=48) is as follows: BMI was significantly correlated with MAP ($p=0.034$), HR ($p=0.048$) and LF/HF ratio ($p=0.02$). While, there was no significant correlation between (1) BMI and arterial stiffness; and (2) cardiac ANS function indices and arterial stiffness. BMI ($\beta=0.288$, $p=0.023$) and arterial stiffness ($\beta=0.516$, $p<0.001$) were the significant determinants of rising BP. MAP ($\beta=0.591$, $p<0.001$) was the significant determinant of the arterial stiffness. **Conclusion:** A complex relation exists between adiposity, BP, ANS and arterial stiffness. Excess adiposity may modulate sympathovagal balance by increasing sympathetic activity which can enhance the vascular tone and elevate BP. Inturn, increased BP induces stiffening of arteries that may further augment BP.

Key words: Arterial stiffness, Autonomic nervous system, Blood pressure, Overweight, Obesity.

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INTRODUCTION

The prevalence of obesity in young individuals is increasing rapidly worldwide. Obesity is associated with hypertension and cardiovascular (CV) diseases.[1] Studies have demonstrated that overweight and obesity predicts the development of hypertension.[2-3] Hypertension is considered as one of the primary mediator of CV diseases. However, the excess weight-induced physiological and molecular mechanisms that modulate blood pressure (BP) are not clearly understood.

Various mechanisms such as activation of renin-angiotensin-aldosterone system (RAAS), endothelial dysfunction, oxidative stress and inflammation have been identified as the causes of obesity-induced hypertension.[3-5] Few studies have shown that obesity may modulate sympathovagal balance towards sympathetic dominance which may be responsible for elevation of BP by increasing vascular tone,[6] while other study have not found any change in autonomic nervous system (ANS) function.[7] Hence, the influence of body mass index (BMI) on ANS function remains unclear. Endothelial dysfunction is shown to be associated with hypertension in individuals with overweight and obesity, and is predicted as the potential cause that may contribute to the hypertension.[8] An experimental study on animal model has demonstrated an increased BP and impairment in relaxation of the aorta in response to acetylcholine (an endothelium-dependent vasodilator) in mice with high-fat diet induced obesity.[9] Studies have also shown an association between arterial stiffness and obesity.[10-11] However, the precise mechanism linking overweight/obesity with hypertension remains unclear. The inter-relation between autonomic nervous system and arterial health and BP influenced by adiposity may help in understanding the pathophysiology of obesity induced hypertension. Therefore, the present study was conducted with two objectives (1) to evaluate the impact of adiposity on cardiac autonomic activity and arterial health and (2) to determine the relationship between adiposity, cardiac autonomic nervous system (ANS), arterial stiffness and BP.

MATERIAL AND METHODS

A cross-sectional study was conducted on young healthy subjects (n=48) with overweight/obesity (n=24) and normal weight (n=24) with age ranging between 18-24 years. Healthy subjects with BMI ≥ 25 (obese/overweight) were included in the study group (OW/Obese group) and BMI from 18.5-24.9 (normal weight) in the control group (NW group). Subjects with history of acute or chronic diseases; habit of smoking, chewing tobacco and consumption of beverages; subjects taking any medications, herbal drugs and vitamin supplements and subject refusal were excluded from the study. Informed consent was obtained for participation in the study. Approval for the study was taken from the institutional ethical committee of Sri B. M. Patil Medical College, Hospital and Research Centre, BLDE (Deemed to be University), India, as per the guidelines (2017) of Indian Council of Medical Research.

Data collection

The data was collected in the morning between 9:00 am to 12:00 pm at room temperature following supine rest for 10 min.

(i) **Body mass index (BMI):** Body mass index, a measure of adiposity was calculated using body mass divided by square of the body height and was expressed as kg/m^2.

(ii) **Blood pressure measurement:** Blood pressure (mmHg) was measured by oscillometric method using digital blood pressure monitor (OMRON HEM-7111) in the sitting posture. Pulse pressure (difference between systolic and diastolic BP) and mean arterial pressure (MAP: diastolic BP + 1/3rd pulse pressure) were calculated from the obtained BP values.

(iii) **Measurement of arterial stiffness:** Arterial stiffness was measured by oscillometric method using a non-invasive automatic device: Periscope (Genesis Medical Systems, India), which is a validated 8-channel real time PC based simultaneous acquisition and analysis system.[12] The acquisition rate was 200 samples per second. This system uses BP cuffs (containing pressure sensors) and two channel ECG leads to record arterial pressure waveforms and ECG simultaneously. The recordings were made in supine position. ECG electrodes were placed on ventral surface of side of the chest and abdomen, and BP cuffs were wrapped on both upper arm and above ankles. The data obtained in 10 seconds was stored in the computer for further analysis. The device is fully automated and does not require any operator for handling any probe to record the waveforms; hence it is devoid of any operator bias. The method of calculation of pulse wave velocity (PWV), augmentation index (AIx) and arterial stiffness index (ASI) has been explained elsewhere.[13] The following arterial stiffness indices were included for the study.

   (a) Brachial-ankle PWV (baPWV): Pulse wave velocity is a measure of regional arterial stiffness. Higher the PWV more is the arterial stiffness. It is the velocity at which the arterial pulse propagates between brachial and ankle (tibial artery). It reflects the arterial stiffness of aorta and peripheral artery. Right baPWV (m/s) values were taken for the study.

   (b) Carotid-femoral PWV (cfPWV): It reflects the arterial stiffness of aorta. It was calculated by the composite baPWV found out by averaging left and right baPWV using a regression equation (0.8333* Avg. baPWV-233.33).

   (c) Heart-brachial PWV (hbPWV): It is the velocity at which the arterial pulse propagates from heart to brachial artery. It reflects the arterial stiffness of part of the aorta and peripheral artery.

   (d) Heart-ankle PWV (haPWV): It is the velocity at which the arterial pulse propagates from heart to ankle (tibial artery). It reflects the arterial stiffness of aorta and peripheral artery.

   (e) Arterial stiffness index (ASI): It reflects local arterial stiffness. Arterial stiffness index estimated using oscillometric method by quantifying the oscillometric envelopes derived from the oscillations in the respective artery.

   (f) Aortic augmentation index (AIx): It is a measure of combined arterial stiffness and wave reflection. The rise in the systolic pressure is called an augmentation pressure. Augmentation index is the ratio of augmentation pressure to the aortic PP and is expressed in percentage. An augmentation index normalized for a heart rate at 75 bpm (AIx@75) was used in this study.[14] Higher the AIx more is the arterial stiffness. Augmentation index also reflects vascular tone.

(iv) **Assessment of autonomic function:** Autonomic nervous system function was assessed by measuring heart rate variability (HRV). A short-term ECG for 5 min was recorded in the standard limb lead II configuration using a four channel digital polygraph (Medicaid systems Pvt. Ltd, Chandigarh, India). Noise free data was included for HRV analysis. Offline scrutiny showed no ectopic beats. Subjects were advised to breath normally during recording of ECG. The data acquired (R-R interval) was analyzed by frequency domain method using HRV analysis program developed by the Biomedical Signal Analysis Group, University Kuopio, Finland.[15] Power spectral density of the RR series was obtained by a non-parametric Fast Furrier Transform (FFT) technique. Total power in the frequency range (0-0.40Hz) was divided into very low frequency (VLF: 0-0.04), low frequency (LF: 0.04-0.15Hz) and high frequency (HF: 0.15-0.40Hz). LF (nu) measure reflects mainly sympathetic activity while HF (nu) reflects parasympathetic activity. LF/HF ratio reflects sympathovagal balance.[16]

Statistical analysis

Data obtained was expressed as mean and SD. Data distribution was assessed prior to the applications of tests for significance. To find the significant difference in the outcome variable between the two groups, unpaired-t test was applied. Pearson correlation coefficient was applied to find a correlation between the variables. Multiple regression analysis was done to determine the predictors of increased BP in obese individuals. Statistical significance was established at P≤0.05.

RESULTS

Baseline characteristics of participants

A total of 48 participants with normal weight (n=24) and overweight/obese (n=24) were included in the study. Among these 12 were males and 12 were female in normal weight (NW) group and; 7 were males and 17 were female in overweight/obese (OW) group. A significant elevation was seen in SBP, DBP and MAP in individuals with overweight when compared to normal weight. PP and HR, although also raised in obese/overweight subjects, the increase was not significant (Table 1).

Arterial stiffness

Table 2 shows vascular stiffness indices in study participants. There was no significant difference in arterial stiffness between individuals with overweight/obese and normal weight. Though not significant but a mean increase in baPWV, hbPWV, AI@75, and ASI was found in OW group when compared to NW group participants.

Heart rate variability

Table 3 shows a significant variation was observed in LF, HF and LF/HF ratio between NW and OW groups. LF and LF/LF ratio was significantly elevated (p<0.001) while HF was significantly lowered (p<0.001) in obese/overweight subjects when compared to normal subjects, suggesting that increase in weight above normal can induce sympathovagal imbalance.

Correlations between BMI, BP, arterial stiffness and cardiac autonomic function

Table 4 shows correlation between BP and covariates. Mean arterial pressure (MAP) was significantly correlated with BMI (r=0.307, p=0.034), LF/HF ratio (r=0.325, p=0.024) and baPWV (r=522, p<0.001). Body mass index was significantly and positively correlated with MAP (r=0.307, p=0.034), HR (r=0.287, p=0.048) and LF/HF ratio (r=0.336, p=0.02) (Figure 1). While, there was no significant relationship between BMI and arterial stiffness. (Figure 2a and b).

Table 5 shows correlations between cardiac autonomic function and arterial stiffness. There was no significant correlation between cardiac autonomic function indices and arterial stiffness.

Further, multiple regression analysis was done to identify the significant predictors of BP in obese/overweight individuals. BMI (β=0.288, p=0.023)

Table 1: Characteristics of the participants.

Characters	Normal weight Group (n=24) (Mean ± SD)	Over weight/Obese Group (n=24) (Mean ± SD)	p-Values
Sex (M/F)	12:12	7:17	-
Age (years)	19.04±0.86	18.88±1.47	-
BMI (Kg/m^2)	19.72±2.54	28.80±5.9	<0.01***
SBP (mmHg)	117±12.34	124±20.15	0.044*
DBP (mmHg)	67.04±7.07	72.21±11.96	0.009**
PP (mmHg)	50.13±9.45	51.92±16.98	0.495
MAP (mmHg)	83.75±8.16	89.78±12.55	0.006**
HR (bpm)	70.73±11.78	77.09±10.93	0.059

Note: SBP: systolic BP; DBP- Diastolic BP; PP-Pulse pressure; MAP- Mean arterial pressure; HR-Heart rate; *p<0.05, **p<0.01, ***p<0.001.

Table 2: Arterial stiffness in normal and overweight/obese participants.

Parameter	Normal weight Group (n=24)	Overweight/ obese Group (n=24)	95% CI	p-value
	(Mean±SD)	(Mean±SD)		
baPWV (cm/s)	990.68±128.67	996.76±91.08	-70.85, 58.7	0.851
cfPWV (cm/s)	609.17±139.6	582.41±78.83	-39.13, 99.66	0.418
AI@75	5.54±3.96	6.04±3.78	-2.75, 1.75	0.657
hbPWV (cm/s)	290.71±37.65	300.67±35.15	-31.12, 11.2	0.348
haPWV (cm/s)	424.29±45.89	419.58±47.62	-22.47, 31.89	0.728
bASI	21.83±5.62	22.37±5.32	-3.72, 2.64	0.735
aASI	32.39±8.59	34.48±6.87	-6.61, 2.43	0.357
ABI	1.06±0.07	1.01±0.10	-0.006, 0.094	0.08

*p<0.05, **p<0.01, ***p<0.001.

Table 3: Heart Rate Variability in normal and overweight/obese participants

Parameter	Normal weight Group (n=24) (n=24) (Mean ± SD)	Overweight/ obese Group (n=24) (Mean ± SD)	95% CI	p-value
LF(nu)	83.57±1.67	70.54±3.78	-2.97, -0.96	<0.001***
HF(nu)	16.43 ±1.68	14.46 ±1.78	0.96, 2.97	<0.001***
LF/HF ratio	5.43 ± 0.65	6.01 ± 0.81	-1.31, -0.46	<0.001***

*p<0.05, **p<0.01, ***p<0.001.

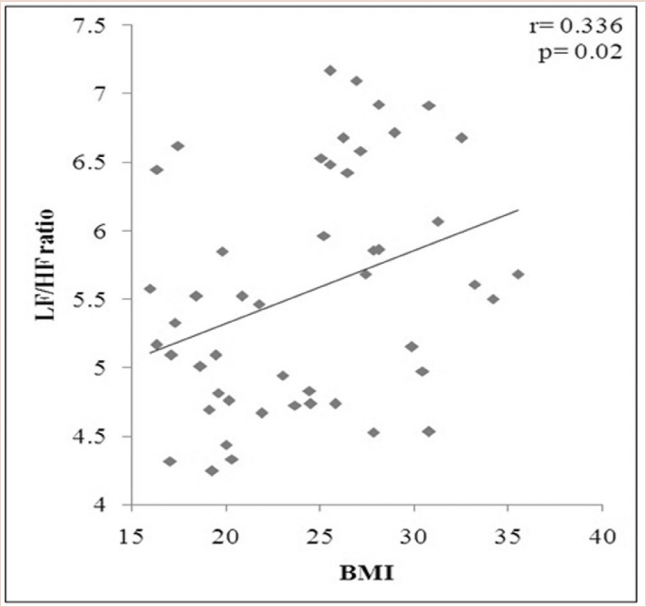

Figure 1: Correlation between body mass index and sympathovagal balance.

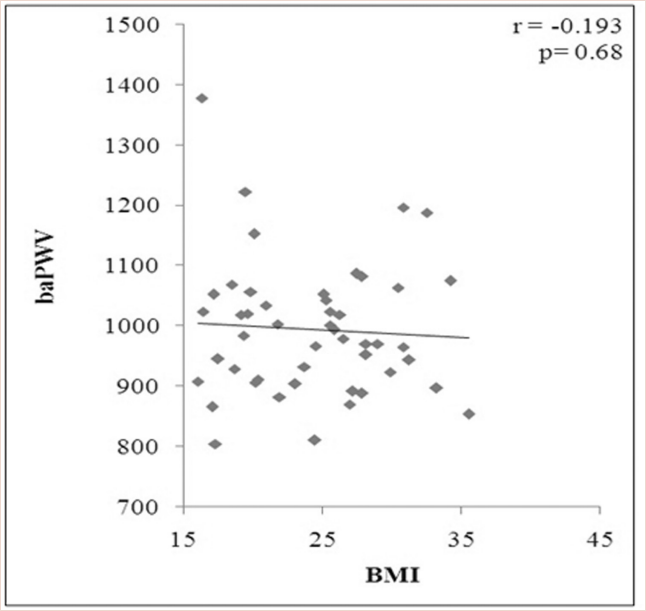

Figure 2: Correlation between body mass index and arterial stiffness indices (a) brachial-ankle pulse wave velocity

and arterial stiffness (baPWV) (β=0.516, p<0.001) were the significant predictors for increased BP. MAP (β=0.591, p<0.001) was the significant predictor for the arterial stiffness.

DISCUSSION

The present study has evaluated (1) the impact of adiposity on heart rate variability (as an index of cardiac autonomic function) and arterial stiffness; and (2) the relationship between adiposity, cardiac ANS activity, arterial stiffness and BP, to explore the possible mechanism of obesity induced elevation of BP. We observed a significant elevation in BP and sympathetic dominance in overweight/obese individuals as compared to normal weight subjects. Though not significant, there was a mean increase in arterial stiffness in overweight /obese individuals than subjects with normal weight. An increase in mean arterial pressure (MAP) was significantly correlated with rise in BMI, LF/HF ratio and baPWV. A significant

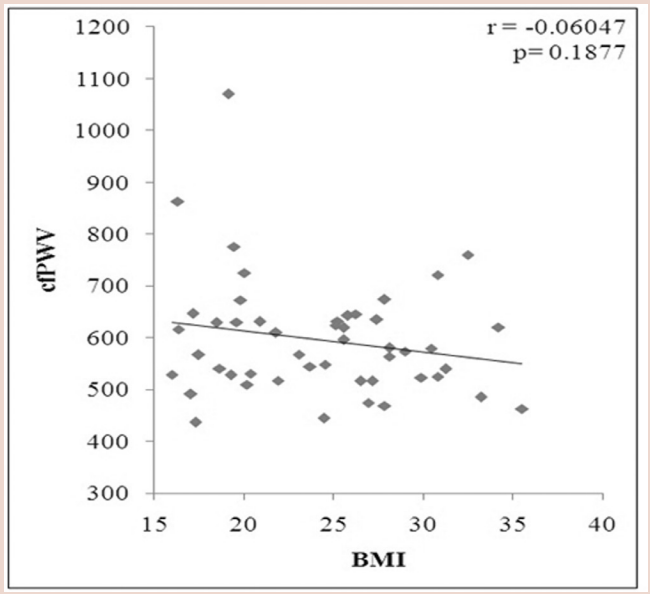

Figure 2b: Correlation between body mass index and arterial stiffness indices (b) carotid-femoral pulse wave velocity

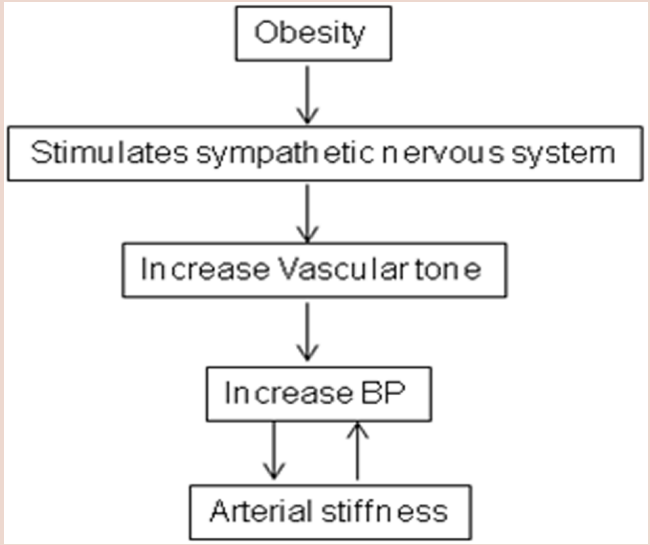

Figure 3: Interrelation between adiposity, autonomic nervous system, blood pressure and arterial stiffness (Missing in galley proof).

Table 4: Bivariate correlation between blood pressure and covariates (n=48).

	SBP(mmHg)		DBP(mmHg)		PP(mmHg)		MAP(mmHg)	
	R	P-value	R	P-value	R	P-value	R	P-value
BMI (Kg/sqm)	0.268	0.066	0.276	0.058	0.136	0.355	0.307	0.034*
HR (bpm)	0.114	0.44	0.339	0.019*	-0.123	0.404	0.267	0.066
LF/HF ratio	0.308	0.033*	0.229	0.117	0.234	0.109	0.325	0.024*
baPWV (cm/s)	0.569	<0.001***	0.424	0.003**	0.426	0.003**	0.522	<0.001***
cfPWV (cm/s)	0.405	0.004**	0.311	0.032*	0.293	0.043*	0.371	0.009**
AI@75	0.285	0.049*	0.326	0.024*	0.097	0.514	0.311	0.031*

*p<0.05, **p<0.01, ***p<0.001

Table 5: Correlation between arterial stiffness and autonomic function (n=48).

	LF		HF		LF/HF	
	R	P-value	R	P-value	R	P-value
baPWV (cm/s)	0.147	0.319	-0.147	0.319	0.139	0.347
cfPWV (cm/s)	0.026	0.860	-0.020	0.86	0.023	0.876
AIx	0.183	0.214	-0.183	0.214	0.18	0.221
hbPWV (cm/s)	0.073	0.622	-0.073	0.622	0.07	0.634
haPWV (cm/s)	0.125	0.397	-0.125	0.397	0.129	0.383
bASI	0.158	0.285	-0.158	0.285	0.156	0.29
aASI	0.178	0.227	-0.178	0.227	0.149	0.311

correlation exists between an increase in BMI and sympathovagal imbalance but not with arterial stiffness.

Autonomic nervous system plays an important role in regulatory mechanism of BP and CV homeostasis. Sympathetic nervous system (SNS), one of the two divisions of the ANS regulates vascular tone and BP.[17] Recent evidence suggests that SNS is not only involved in short-term regulation of BP, but also plays a key role in long-term BP regulation.[18-19] Thus, sympathetic over activity increases vascular tone (mainly vasoconstriction of small resistance muscular arteries) and BP. Studies have demonstrated that sympathetic over activity is one of the major mechanisms for elevation of BP through increasing vascular tone.[20-21] As stated earlier, HRV is one of the best tools to measure sympathetic and parasympathetic activity. Kaufman CL *et al.*[22] demonstrated a significant difference in HRV variables among normal, overweight and obese group. They showed significantly higher LF and LF/HF ratio and reduced HF in obese individuals as compared to normal weight subjects. While Krishna P *et al.*[7] in their study did not find any impact of overweight on sympathovagal balance. In the present study, we found a significant elevation in LF (nu), LF/HF ratio and decrease in HF (nu) in overweight/obese individuals when compared with normal weight subjects. These changes in HRV suggest a shift in the autonomic balance towards sympathetic dominance with sympathetic over activity and reduced parasympathetic tone in OW/obese individuals.

We also observed a significant correlation between increase in BMI and sympathovagal imbalance [Wide Figure 1]. Further, there was a significant correlation between sympathovagal imbalance and MAP [wide Table 4]. These data suggest that a significant interrelation exists between BMI, sympathovagal balance and MAP. Several studies have linked the sympathetic over activity with the mechanism of development of metabolic dysfunction and obesity, but yet it is a matter of debate whether SNS over activity is a cause or consequence of metabolic dysfunction.[23-24] We presume that sympathetic over activity in overweight/obesity is an essential modulation to fulfill the excess demand (oxygen and nutrition) of the overweight body, so it may be a consequence of overweight/obesity. This overweight/obesity induced essential sympathetic over activity might in turn have increased the heart rate, vascular tone and BP in the young obese individuals.

A systematic review and meta-analysis of 1281 obese children between 5 and 24 years of age has shown that child/adolescent obesity is associated with greater arterial stiffness.[10] In another study conducted on 196 participants between the age group of 20-40 years, with a follow up after two years, a linear relationship was observed between the annual weight gain and increase in aortic pulse wave velocity (PWV); and a linear decline in aortic PWV was observed corresponding to annul weight lost.[11] Aortic pulse wave velocity is an index of aortic stiffness. As mentioned above, higher the pulse wave velocity more is the arterial stiffness. Though, our study showed a mean increase in arterial stiffness in overweight/obese subjects, there was no statistically significant difference between two groups of overweight and normal weight individuals. Further, there was no significant correlation between increasing BMI and arterial stiffness. In contrast, other studies have shown a positive correlation between increasing adiposity and arterial stiffness.[25-26] Another finding of our study is that arterial stiffness was significantly correlated with BP. Arterial stiffness was also the significant predictor of obesity-induced BP and in turn rising BP was the significant determinant of stiffening of arteries. This data indicates a bidirectional relationship between arterial stiffness and BP.[27] Further, there was no significant co-relation between arterial stiffness and increased sympathetic activity or decreased parasympathetic tone.

The interrelation between adiposity, cardiac ANS activity, arterial stiffness and BP is depicted in Figure 3. We presume (from our data) that excess adiposity induces sympathetic over activity which enhances the vascular tone and so BP. Increased BP may initiate vascular damage and induces stiffening of the arteries; and in-turn stiffened arteries may increase the BP, indicating a possible vicious cycle between BP and arterial stiffness.[13,27-28] This vicious cycle may be further aggravated by adiposity associated CV risk factors like glucose intolerance or insulin resistance.[29]

CONCLUSION

Increase in BMI induces a modulation in autonomic function through a shift in sympathovagal balance towards sympathetic dominance. Though not significant, mild increase in vascular stiffness in overweight/obese subjects indicate an initiation of vascular damage in them. Adiposity, cardiac ANS, arterial stiffness and BP are interrelated and the relation is complex. Excess adiposity may modulate sympathovagal balance by increasing sympathetic activity. Sympathetic over activity can enhance the vascular tone and elevate BP. Inturn, increased BP induces stiffening of arteries that may further augment BP. Further, long-term follow up studies may be required to understand precisely the inter-relationship between adiposity, BP, ANS and stiffening of arteries.

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CONFLICT OF INTEREST

The authors declare no conflcit of interest.

ABBREVIATIONS

BMI: Body mass index; **CV:** Cardiovascular; **ANS:** Autonomic nervous-system; **BP:** Blood pressure; **MAP:** Mean arterial pressure; **PP:** Pulse-Pressure; **HR:** Heart Rate; **LF:** Low Frequency; **HF:** High Frequency; **PWV:** Pulse Wave Velocity; **ASI:** Arterial Stiffness Index; **AI:** Augmented Index; **ABI:** Ankle Brachial Index.

SUMMARY

We have investigated (1) the impact of adiposity on cardiac autonomic function and arterial stiffness; and (2) the relationship between adiposity, cardiac ANS activity, arterial stiffness and BP with an aim to explore the possible mechanism of obesity induced elevation of BP. A complex relation exists between adiposity, BP, ANS and arterial stiffness. We observed a significant increase in BP and sympathetic activity in overweight/obese individuals as compared to normal weight subjects. Though not significant, there was a mean increase in arterial stiffness in overweight / obese individuals than subjects with normal weight. An increase in mean arterial pressure was significantly correlated with BMI, sympathovagal imbalance and arterial stiffness. A significant correlation exists between increasing BMI and sympathovagal imbalance but not with arterial stiffness.We presume that excess adiposity may modulate sympathovagal balance through sympathetic activation. Sympathetic overactivity may enhance the vascular tone and elevate BP. In turn, increased BP induces stiffening of arteries that may further augment BP.

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