Effect of posture on heart rate variability in school children

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ABSTRACT

Heart rate variability (HRV) refers to the beat-to-beat alteration in cardiac cycle length. The objective is to study effect of posture on HRV. Five minutes ECG of children (12 females and 20 males) with mean height 138.88±11.88 cm, weight 27.66±5.87 kg and BMI 16.11±1.38 kg/m^2 were recorded in supine, sitting and standing position. HRV parameters were compared by one-way ANOVA and Bonferroni test. Standing decreases Mean interval between successive RR waves (622.69±83.24 ms Vs 721.66± 89.30 ms Vs 750.28±107.917, $p<0.001$), the square root of the mean squared differences of successive interval (25.37±17.52 ms Vs 50.16± 25.03 ms, $p <0.001$),numbered of RR interval differences 50ms (40.59±61.58 Vs 115.72±75.408 Vs 134.16± 76.57, $p<0.001$), percentage NN50 (9.65±15.32 Vs 29.85±20.52, Vs 36.18±22.33, $p<0.001$), High frequency (HF) peak (152.63±254.41 Vs 427.84±369.62 540.84±452.12, $p<0.05$), HF power %(34.91±17.67 Vs 51.99±17.57 Vs 56.94± 17.06, $p<0.001$), HF normalised unit (45.05±17.82 Vs 61.11± 17.06 Vs. 65.73± 15.14, $p<0.001$) as compared to sitting and supine. Standing increases low frequency (LF) power % (39.78± 11.76 Vs 31.64± 12.33 Vs 28.53± 11.65, $p<0.05$), LF normalized unit (54.95± 17.82 Vs 38.89± 17.05 Vs 34.27± 15.14, $p<0.001$), LF: HF ratio (1.61± 1.26 Vs 0.808± 0.69 Vs 0.6335± 0.538, $p<0.001$) compared to sitting and supine. This study showed significant decreased in HRV parameters reflecting vagal activity and reciprocal increase in sympathetic activity in standing as compared to sitting and supine. There was no significant change in HRV in sitting as compared to supine.

Keywords: Heart rate variability, vagal, sympathetic.

INTRODUCTION

Heart rate variability is essentially a measure of the time between successive R-R intervals in the QRS waves of an ECG. Heart rate variability (HRV) analysis provides a quantitative marker of the autonomic nervous system (ANS) because the regulation mechanisms of HRV originate from the sympathetic and parasympathetic nervous systems. Increased efferent vagal activity is characterized by reduced heart rate, HRV whereas sympathetic stimulation increases heart rate, and decreased HRV.[1] HRV analysis results can be applied to both clinical use and research. HRV analysis can be used in hospitals to help diagnose various cardiac diseases. Researchers are able to find interesting relationships between HRV and diseases as well as drug pharmacodynamics. These articles observe relationships between HRV and blood pressure; myocardial infarction, nervous system, cardiac arrhythmia, diabetes, respiration, renal failure, gender, age, fatigue, drugs, smoking, alcohols, and so on. Heart rate variability measurements are easy to perform, are non-invasive and have good reproducibility if used under standardized conditions.[2, 3]

Various physiological manoeuvres have been used to gain a deeper insight into the functioning of the autonomic nervous system. Investigators often use a change in posture by an either passive head-up tilt or active standing to impose perturbation on the steady state functioning of the ANS. Cacioppo et al. have suggested that vagal activity is highest and sympathetic activity is lowest in the supine posture. The reverse occurs in the standing posture and combination is characteristic for sitting posture.[4]

On moving from supine to erect position, there is large gravitational shift of the blood away from chest to the distensible venous capacitance. As a consequence venous return to heart is reduced resulting in reduced stroke volume. Despite decreased cardiac output, fall in mean arterial pressure is prevented by vasoconstriction and increase in heart rate. These rapid short terms adjustment to orthostatic stress is mediated by the autonomic nervous system.

MEASUREMENT AND PARAMETERS OF HRV

Heart rate variability can be assessed by calculation of indices based on statistical operations on R-R intervals (times domain) or by spectral (frequency domain) analysis of an array of R-R intervals. The parameters of time domain are NN interval (interval between successive RR waves), RMSSD (the square root of the mean squared differences of successive interval), NN50 count(numbered of RR interval differences 50ms), pNN50(percentage NN50 count). The parameters of frequency domain are : peak, power, power%, nu of HF that are sensitive to vagal activity, of LF that are sensitive to sympathetic activity and LF:HF ratio represents sympathovagal balance.

Heart rate variability can also be assessed by Geometrical methods which uses the sequence of RR intervals to construct certain geometrical forms and extract the assessment of HRV from this, e.g. SD 1 which represents the short term HRV and SD 2 which represents the long term HRV.

The analysis can be performed on short electrocardiogram (ECG) segments (lasting from 0.5 to 5 minutes) or on 24-hour ECG recordings.[5]

Most of the studies showed that HRV decreases linearly with age especially after adolescence and the parameters regarding vagal tone decrease on active standing. But the review regarding effect of sitting position on HRV is found to be lacking. So in this context it would be appropriate to study HRV on sitting position in combination to supine and standing. We attain these positions: supine, standing, sitting in our daily life and it is seen that in supine to standing autonomic nervous shift from parasympathetic to sympathetic predominance. But we don't know exactly what happens to the vagal tone from supine to sitting position in children. If vagal tone isn't significantly different from supine to sitting, we can perform HRV in sitting position in patient which would be comfortable to them using the same normative data as for supine position.

We also know that HRV depends on age which is one of the major factors. One value of the HRV parameters cannot serve the purpose for all the age group. Therefore age dependent values of HRV should be formulated to assess cardiovascular function in patients. The data obtained from this study might show a direction rather that provide normative data.

MATERIALS AND METHODS

Subjects were randomly selected by simple random sampling method. Thirty two subjects (12 females and 20 males) were taken having following inclusion criteria and exclusion criteria:

Inclusion criteria

Healthy children who had no signs and symptoms regarding autonomic function.

Age 6 to 12 years, BMI 5 to 85 percentile.

Exclusion criteria

I Children with hypertensive, cardiovascular disorders, drug dependence and other disease, those are likely to affect autonomic nervous system

II. BMI> 85 percentile.

Recording procedure

Those children who fulfill above inclusion criteria were brought to human physiology lab of BPKIHS between 8:00 to 10:00 and their height and weight were recorded. After supine rest for 15 minutes, cardiopulmonary parameters were recorded followed by recording of baseline ECG for 5 minutes at spontaneous respiration in supine position. After recording for 5 minutes the child was asked to sit on the bed. They were given 5 minutes of rest in this position. Blood pressure was recorded at 4 minutes. Then 5 minutes long sitting ECG was recorded. Then the child was asked to stand up and BP at standing position was measured at 4 minutes. Then again one more 5- minute long ECG segment was recorded.

The ECG of all the children was stored in the computer of Hewellet Packard. The ECG signal was captured using Coulbourn Instrument by using software WINDAQ Pro/ Pro+, model number DI-400 series, USA. The respiratory rate was calculated from the respiratory wave which was recorded at channel 1 along with ECG wave.

The entire 5-mins ECG epoch was edited if any ectopic beat or artifact was present and if any R wave was missing, it was inserted appropriately. The data were saved as lotus 2 files, opened by MS excel and cumulative value of R-R interval was converted into individual R-R interval. These intervals were saved in ASCII format which was readable and analyse by software HRV analysis.

Statistical analysis

The HRV parameters among three postures were compared with each other by one-way ANOVA and post hoc Bonferroni comparison using software version 10.2.

RESULTS

General characteristics of subjects

The mean age of the subjects was 9.5±1.97 years. Their mean height was 130.88±11.89 cm and weight was 27.66±5.87 Kg with body mass index (BMI) of 16.113±1.39 kg/m^2. The BMI was within the range of 5 to 85 percentile.

Effect of posture on cardiopulmonary parameters

No significant difference in supine systolic and diastolic BP in response to sitting or standing. The standing heart rate was significantly higher as compared to supine and sitting (98.33±12.98 bpm vs. 81.97±13.48 and 84.97±11.89 bpm, $p<0.001$)

Table-1: Effect of posture on heart rate and blood pressure of children (n=32)

Variables	Supine	Sitting	Standing	P value
SBP (mmHg)	105.19±7.18	105.75±6.79	106.38±6.49	NS
DBP (mmHg)	71.25±5.32	71.88±5.08	71.94±4.69	NS
HR (bpm)	81.97±13.48	84.97±11.89	98.33±12.98* #	* # P<0.001 standing vs. sitting and standing vs. supine
RR (per min)	19.5±3.73	21.19±4.62	23.13±5.43	P<0.01 standing vs. supine

SBP= systolic blood pressure, DBP= diastolic blood pressure, HR= heart rate, RR= respiratory rate

The standing respiratory rate was significantly higher as compared to supine (23.13± 5.43 per minute vs. 19.5± 3.73 per minute, p<0. 01) (Table-1).

Effects of posture on HRV

The standing MeanRR, SDNN, RMSSD, NN50, pNN50, decreases significantly as compared to supine and sitting (Table-2).

The standing HF power, HF power percent, HFnu, LF peak, LF power decreases significantly as compared to sitting and supine whereas LF power percent, LFnu, LF/HF ratio, VLF power percent increased in standing position as compared to sitting and supine (Table-3).

DISCUSSION

Heart Rate Variability is widely accepted as a valuable tool to investigate the sympathetic and parasympathetic contribution to regulation of heart rate rhythm. HRV has especial value in assessing cardiovascular status in diabetic and post infarction patients.[5]

The time and frequency domain HRV variables that are highly correlated with vagal tone were changed significantly from supine to standing and sitting to standing whereas none of the time and frequency variables showed significant change from supine to sitting position.[5]

This study attempts to explore the HRV response to standing and sitting from supine position. The study showed significant increase in heart rate in standing as compared to supine and sitting (Table-2). It is known from various studies that immediate increase in heart rate from lying to standing position occurs within 15 seconds.[6] However, in a study done by Yamaguchi there was no significant difference in HR changes in the later stage during 7 min of standing.[7]

Effect of posture change on time and frequency domain variables of HRV

The study showed significant decrease in time domain HRV measures (SDNN, RMSSD, NN50, pNN50) reflecting vagal component in response to standing as compared to sitting or supine posture. These findings are supported by decrease in high frequency HRV measures (HF power and its percent and normalized unit) in response to standing. Further, there was decrease in NN interval and increase in respiratory rate in standing posture.

Table-2: Time domain comparison among three positions in children (n=32)

Variables	comparison among variables with mean ±SD		P value
Mean NN (ms)	Supine=750.28± 107.92	Sitting=721.66± 89.30	NS
		Standing=622.69±83.24	P<0.001
SD of NN (ms)	Supine=49.53± 18.64	Sitting=45.97± 17.49	NS
		Standing=30.13 ±14.23	P<0.001
RMSSD (ms)	Supine=58.18 ±27.95	Sitting=50.16± 25.03	NS
		Standing=25.37± 17.52	P<0.001
NN50 (count)	Supine=134.16± 76.57	Sitting=115.72± 75.41	NS
		Standing=40.59 ±61.58	P<0.001
pNN50 (%)	Supine=36.18± 22.33	Sitting=29.85± 20.52	NS
		Standing=9.65± 15.32	P<0.001
SD 1 (ms)	Supine=42.07± 19.98	Sitting=36.42± 18.00	NS
		Standing=18.59± 12.61	P<0.001
SD 2 (ms)	Supine=62.84±20.02	Sitting=60.52± 20.08	NS
		Standing=44.22±19.65	P<0.001

MeanNN= mean of successive R-R intervals; SDNN= standard deviation of R-R intervals; RMSSD= root mean of squared successive R-R interval differences; NN50= successive R-R interval differences greater than/equal to 50 ms; pNN50= Percentage of NN50; SD 1= SD 1; SD 2= SD 2.

Table-3: Effect of posture on frequency domain parameters of HRV in children (n=32)

Variables	comparison among variables with mean ±SD		P value
HF peak Hz	Supine=0.292± 0.06	Sitting=0.2773± 0.072	NS
		Standing =0.271± 0.073	NS
HF power, ms^2	Supine=540.84± 452.12	Sitting=427.84±369.62	NS
		Standing=152.63± 254.41	P<0.001
HF power %	Supine= 56.94± 17.06	Sitting=51.99±17.57	NS
		Standing=34.91±17.67	P<0.001
HF nu	Supine=65.73± 15.14	Sitting= 61.11± 17.06	NS
		Standing= 45.05±17.82	P<0.001
LF peak, Hz	Supine (0.0849± 0.035)	Sitting= 0.0774± .029	NS
		Standing= 0.0668± 0.021	P<0.001
LF power, ms^2	Supine= 226.88± 160.8	Sitting= 202.56± 139.12	NS
		Standing= 135.34± 143.84	P<0.05
LF power %	Supine=28.53± 11.65	Sitting= 31.64± 12.33	NS
		Standing= 39.78± 11.76	P<0.001
LFnu	Supine 34.27± 15.14	Sitting= 38.89± 17.05	NS
		Standing= 54.95± 17.82	P<0.001
LF/HF ratio	Supine =0.6335± 0.538	Sitting= 0.808± 0.69	NS
		Standing= 1.61± 1.26	P<0.001
VLF peak, Hz	Supine =0.2086± 0.0073	Sitting= 0.0228± .0069	NS
		Standing= 0.03108± 0.035	NS
VLF power ,ms^2	Supine=89.63 ±59.29	Sitting= 104.81± 72.78	NS
		Standing= 77.25± 69.12	NS
VLF power %	Supine (14.52±11.40)	Sitting= 16.36± 8.98	NS
		Standing= 14.52±11.40	P<0.001

VLF: very low frequency, LF: low frequency, HF: high frequency, nu: normalized unit

There was reciprocal increase in low frequency components of HRV, namely, low frequency power percentage and LF normalized unit. However, the LF power was decreased and LF peak shifted to further lower peak in response to standing. There was no difference in these two variables between sitting and standing, in contrast to all other HRV measures, which showed difference between sitting and standing.

The increase in LF power percent and normalized unit is supported by change in LF/HF ratio suggesting shift of sympathovagal balance toward sympathetic predominance in standing position as compared to other two postures.

Although for Poincare plot SD2, (an indicator of long term HRV) a 20-minutes ECG

epoch is recommended, it showed decrement in standing as compared to supine or sitting.[1] This finding is also supported by increase in VLF power percent suggesting some long-term (slow) frequency components affected HRV even in 5-minutes long ECG-based HRV.

In our study HF component decreased while active standing from supine or sitting posture. However LF power percentage, LFnu and LF/HF ratio increased significantly on changing posture from supine to standing and from sitting to standing. In upright posture, there is clear release from baroreceptor restrain of sympathetic activity with its LF rhythmicity, together with vagal withdrawal.[8] So there is increase in variables reflecting sympathetic activity (LF component, LF/HF ratio) and corresponding decrease in HF components or variables reflecting parasympathetic activity. Finely found that in the age 5-10 years LF and LF/HF ratio significantly decreased in supine position than in standing position.[9] Similar result was seen in adult but the ratio of LF/HF upon standing from supine position was significantly lower in children as compared to adult.[10,11] As with our study result, HFnu and HF power decreased significantly while active standing.[10] Even in diseased subject HF decreased during active standing but in 30% of all subject (control+diseased) HF power increased during active standing.[12] Supine and sitting HF were higher than standing HF.[13]

Vagal blockade in supine, sitting and standing position showed decreased in HF power LF power. Age related decline in LF power was greater in standing than in supine position.[14]

In our study, the value of standing LFnu was statistically increased as compared to sitting LFnu and supine LFnu. Similar response was seen in a study done by Weise in which LF power and LF nu raised significantly on standing position as compared to supine position.[15] There was no change in LF power on standing but LF nu was significantly raised. The LF/HF ratio was significantly increased in response to standing as compared to sitting and supine. Interestingly the changes in LF were not accompanied by change in blood pressure.[10,16] However, a study done on heart rate response and blood pressure from supine to standing position in teenage boys (aged 10-15 years), after 1-2 min standing, young subjects showed a pronounced increase in HR and diastolic pressure with little change in systolic pressure.[17] Kalisnik showed the lower LF/HF ratio in supine position even after treatment with certain drugs like imipramine.[18] In a study done in asthmatic children rise in LF/HF ratio was more pronounced in asthmatic children than in control group.

The ratio of LF/HF upon standing from supine position was significantly lower as compared to adult.[11]

The present study has shown that HF power, HF power %, HF nu decreased in response to standing as compared to supine and sitting and LF power %, LF nu increased in response to standing as compared to supine and sitting in school children. However, LF peak decreased in response to standing as compared to supine and sitting. Time domain variables also decreased significantly on standing from supine and sitting postures. Since variables of HRV that highly correlates the vagal tone decreased on standing from supine and sitting, it is concluded that HRV decreased from supine to standing position.

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REFERENCES

1. Juha-Pekka N, Tarvainen MP, Ranta-aho PO, Karjalainen PA. Software for advanced HRV analysis, *Computer Methods and Programs in Biomedicine*, 2004; 76: 73-81.
2. Acharya UR, Paul Joseph K, Kannathal N, Lim Choo Min, Jasjit Suri S, Heart Rate Variability, Advances in Cardiac Signal Processing, *Springer-Verlag Berlin Heidelberg* 2007: 121-65
3. Grossman P, Karemaker J, Wieling W. Prediction of tonic parasympathetic cardiac control using respiratory sinus arrhythmia: the need for respiratory control. *Psychophysiology* 1991; 28: 201-16.
4. Cacioppo JT, Bernton GG, Binkley PF, Quigley KS, Uchino BN, Fieldstone A. Autonomic cardiac control. II. Noninvasive indices and basal response as revealed by autonomic blockades. *Physchophysiology* 1994; 31: 586-9.
5. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. Special Report. *Circulation* 1996; 17: 354-81.
6. Pal GK, Pal P. Autonomic function test. In Textbook of practical physiology. Orient Longman Private Limited 2003: 350-9.
7. Yamaguchi H, Tanaka H, Adachi K, Mino M. Beat-to-beat blood pressure and heart rate responses to active standing in Japanese children. *Acta Paediatr* 1996; 85: 577-83.
8. Malliani A, Pagani M, Furlan R *et al*. Individual recognition by heart rate variability of two different autonomic profiles related to posture. *Circulation* 1997; 96: 4143-5.
9. Finley JP, Nugent ST, Hellenbrand W. Heart-rate variability in children. Spectral analysis of developmental changes between 5 and 24 years. *Canadian J Physiol Pharmacol* 1987; 65: 2048-52.
10. Srinivasan K, Sucharita S, Vaz M. Effect of standing on short term heart rate variability across age. *Clin Physiol Funct Imaging* 2002; 22: 404-8.
11. Yeragani VK, Sobolewski E, Kay J, Jampala VC, Igel G. Effect of age on long-term heart rate variability. *Cardiovasc Res* 1997; 35: 35-42.
12. Vuksanovic V, Gal V, Kalanj J, Simeunovic S. Effect of posture on heart rate variability spectral measures in children and young adults with heart disease. *Int'l J Cardiol* 2005; 101: 273-8.
13. Chan HL, Lin MA, Chao PK, Lin CH. Correlates of the shift in heart rate variability with postures and walking by time-frequency analysis. *Compute Methods Programs Biomed* 2007; 86: 124-30.
14. Shannon DC, Carley DW, Benson H. Aging of modulation of heart rate. *Amer J Physiol* 1987; 253: H874-7.
15. Weise F, Heydenreich F, Runge U. Contributions of sympathetic and vagal mechanisms to the genesis of heart rate fluctuations during orthostatic load: a spectral analysis. *J Auton Nerv Syst* 1987; 21: 127-34.
16. Triedman JK, Saul JP. Blood pressure modulation by central venous pressure and respiration. Buffering effects of the heart rate reflexes. *Circulation*, Vol 89, 169-179.
17. Dambrink JH, Wieling W. Circulatory response to postural change in healthy male subjects in relation to age. *Clin Sci (Lond)* 1987; 72: 335-41.
18. Jurij-Matija Kalisnik, Viktor Avbelj, Roman Trobec, Borut Gersak. Imaging of power spectral heart rate variability regarding subject position. *Eur J Physiol* 2001.