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CARDIOVASCULAR RESPONSES TO HEAD-DOWN-BODY-UP POSTURAL EXERCISE (*SARVANGASANA*)

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Abstract : Sarvangasana (SVGN) is a head-down-body-up postural exercise in a 'negative g' condition. Though highly recommended as one of the three best of all the asanas it has not yet been studied for its very obvious effects on the cardiovascular (CV) functions. This paper reports the results of the first systematic investigation on SVGN employing echocardiographic analysis in eight healthy male subjects before and after a practice of this asana twice daily for two weeks. The resting heart rate (HR) and left ventricular end-diastolic volume (LVEDV) were significantly reduced ($P<0.02$, $P<0.01$ respectively) after practising this asana. A tendency toward a mild regression of the left ventricular mass was noticed, though it was not statistically significant. The CV responses to acute 45° head-down tilt (HDT) in a tilt table was not altered after practising this asana. Also there was no orthostatic intolerance during the 3–5 min period of 70° head-up tilt (HUT). These results strongly indicate that further studies of this asana performed for a longer period is most likely to yield very significant observations of applied value.

Key words : negative g, head-down tilt, head-uptilt, echocardiography, yoga, *Sarvangasana*

INTRODUCTION

Asanas are various kinds of postural exercises mentioned in Sanskrit literature and being practised in India since ancient times. Pranayams are various types of respiratory exercises in particular postures. Regular practice of asanas and pranayams are believed to exert a sound physical and mental well being leading to a long, disease-free healthy life essential for persuing material and spiritual goals. There has been a revival of interest in the preventive and curative aspects of these yogic practices (1–5). But very few scientific studies have been conducted on any individual asana to investigate its effects on the body function.

Sarvangasana (SVGN) is a head-down-body-up (HDBU) posture in which the subject after lying supine raises his trunk and leg straight above his head with his neck, shoulder and arm muscles supporting the body weight. This asana is claimed to be one of the three best of the numerous asanas described and has been

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prescribed along with other asanas, as a therapeutic aid for various ailments including hypertension (6).

Recently, Selvamurthy et al (7) reported a significant reduction of blood pressure in hypertensive patients after practising a set of eight asanas for three weeks; SVGN was one of these. Considering the very obvious haemodynamic and other perturbations caused by the upside-down posture assumed in SVGN, one is not certain about the advisability of practising this asana even by a normal individual. There is no data available as to the effect of practising this asana on the arterial blood pressure and other cardiovascular variables. Therefore, we decided to start investigating the effects of this asana on cardiovascular functions.

METHODS

Subjects

Eight healthy male medical students aged 17–18 yrs were chosen after clinical examination. All of them were non-smokers and non-athletes in their habit. The protocol of the study was approved by the ethical committee of the Institution and informed consent was obtained from the subjects after explaining the procedures to be adopted.

Protocol

Measurement of arterial blood pressure and echocardiographic analysis of the following parameters were performed before and after a two weeks practice of SVGN.

1. Heart rate (HR).
2. Left ventricular End-Diastolic Volume (LVEDV).
3. Left Ventricular End-Systolic Volume (LVESV).
4. Stroke volume (SV).
5. Cardiac Output (CO).
6. Left Ventricular Posterior wall thickness (LVPW).
7. Ratio between interventricular septum and left ventricular posterior wall thickness (IVS/LVPW).
8. Left Ventricular (LV) Mass.
9. Ejection fraction (EF).

All these measurements were taken in (a) resting supine position, (b) during passive head-up tilt and (c) during passive head-down tilt using a tilt table. The purpose of tilting was to study the CV responses to two different types of acute postural stress both before and after the asana practice.

Arterial blood pressure was measured by a standard sphygmomanometer, the cuff being maintained at the heart level during all experiments. Mean arterial blood pressure (MAP) was calculated as the sum of the diastolic blood pressure (DBP) plus one third of pulse pressure.

Echocardiography was performed by the same operator by using a 3.5 Mhz transducer (Hwelett-Packard Co., Sonos 1000). Two dimensional M-mode echocardiographic imaging of the left ventricle was done with the subject on a tilt table at different degrees of tilt.

Tilting

For head-down tilt (HDT) the table was tilted gradually to 45° and for head-up tilt (HUT) to 70°. About 3–5 minutes were allowed for stabilization before taking the readings. A period of rest was given between head-down tilt experiment and head-up tilt experiment.

Practice of Sarvangasana

Duration of SVGN

Each subject was trained to assume the SVGN posture in training sessions. In each attempt he remained for a minute or more in the asana posture, brought his legs and trunk down to the supine posture, rest in shabasana and assume the asana posture again. The duration of staying in the asana posture was gradually increased in subsequent attempts. In this manner 3–5 times the asana posture was repeated. Subjects were instructed not to exert too much, and to take rest in shabasana any time they felt inconvenience and to note the duration and number of asana posture they performed each day. All subjects performed the asana twice a day for two weeks.

In any subject, the total duration of staying in SVGN posture per day was different in different days. Also, the average duration (averaged for the whole two weeks period) spent in SVGN posture per day showed subject variation. Approximately, a duration of 8 min (range 3 to 10 min) was spent in SVGN posture by each subject daily. A period of ½ to 1 min rest in shabasana was taken between each attempt in the 3 to 5 attempts per session.

Trial session

All the subjects were made to experience the head-up and head-down postures in the tilt tables, by way of trial, to familiarize them with the procedures to be adopted during the actual experiment. No echocardiographic readings were taken during these trial sessions; the BP and HR, however, were monitored. As a part of physiology teaching programme of this institute, all the MBBS students including these subjects visited the echocardiography laboratory previously. On the days of the experiment (before and after SVGN) the subject took rest in supine posture, on the tilt table for 10 to 15 min till the BP and pulse rate was stable. The stable BP and HR was noted and the echocardiography was taken more than once till one of us (JSB) was satisfied about the stability of the echo analysis, and this stable data only were noted. Next the subject was tilted and within 3 to 5 min the BP, HR and echo analysis was done in the same manner.

RESULTS

Effects of SVGN on cardiovascular functions at resting (supine) state

Table I shows the changes in CV variables before and after SVGN practice. LVEDV, SV, CO and LV mass are shown both in absolute values as well as in terms of body surface area or indices, by dividing each value with their body surface area in square meter (LVEDVI, SVI, etc.,) There was a small but significant decrease in resting HR after the two weeks period of SVGN practice. The LVEDV was significantly lower, though the reductions

TABLE I : Resting supine Cardiovascular variables before and after Sarvangasana.

	Parameters	*Before SVGN*	*After SVGN*	*P*
1.	HR (beats/min)	76.71±2.37	72.22±2.97	P<0.02
2.	i) LVEDV (mL)	99.06±3.79	87.78±5.53	P<0.01
	ii) LVEDVI (mL/m^2)	54.51±2.12	48.35±3.20	P<0.01
3.	i) SV (mL)	66.03±4.32	59.00±4.75	NS
	ii) SVI (mL/m^2)	36.37±2.49	32.50±2.69	NS
4.	i) CO (L/min)	5.06±0.37	4.32±0.47	NS
	ii) COI (L/min/m^2)	2.78±0.21	2.38±0.26	NS
5.	EF (percentage)	66.46±3.03	66.75±2.73	NS
6.	SBP (mm Hg)	105.50±4.03	105.00±5.66	NS
7.	DBP (mm Hg)	63.00±2.67	63.75±4.60	NS
8.	MAP (mm Hg)	77.13±2.93	77.46±4.78	NS
9.	Calculated PR (R unit)	0.94±0.08	1.16±0.13	NS
10.	i) LV mass (g)	181.12±11.87	152.62±7.55	NS (P>0.05)
	ii) LVMI (g/m^2)	99.82±6.86	84.07±4.25	NS (P>0.05)
11.	LVPW thickness	1.05±0.04	1.01±0.05	NS
12.	IVS/LVPW	0.85±0.05	0.83±0.03	NS

All values are mean ± SEM. Significance was calculated by paired t test.

in SV and CO were not statistically significant. There were no changes in the systolic (SBP), diastolic (DBP) and mean arterial blood pressure (MAP). The 15.7% reduction in the LV mass was just short of reaching statistically significant level.

Effects of SVGN on CV responses to postural stress by tilt table

Responses to head-down tilt

Before SVGN :

As shown in Table II a 45° HDT increased the EDV and CO though these changes did not reach statistically significant level. The changes in HR, SV and EF were mild and insignificant. There was a tendency for the MAP to rise in response to 45° HDT, which was also not statistically significant. Thus acute 45° HDT did not produce any significant changes in the CV parameters measured within 3–5 min of the tilt.

After SVGN :

The HR, EDV, SV, and CO were all increased in response to 45° HDT but the elevation of none of these parameters were statistically significant. The arterial blood pressure was remarkably stable. The fall in calculated PR seen in response to HDT, were insignificant both before and after SVGN. Thus SVGN practice did not affect the CV response to acute 45° HDT in these subjects.

TABLE II: Changes in Cardiovascular variables during acute head-down tilt before and after Sarvangasana.

	Parameters	Before SVGN			After SVGN		
		Supine	45° HDT	P	Suprine	45° HDT	P
1.	HR (beats/min)	76.71±.37	79.23±3.73	NS	72.22±2.97	75.90±4.46	NS
2.	i) LVEDV (mL)	99.06±3.79	105.28±6.82	NS	87.78±5.53	97.25±3.73	NS
	ii) LVEDVI (mL/m^2)	54.51±2.12	58.03±3.90	NS	48.35±3.20	53.65±2.50	NS
3.	i) SV (mL)	66.03±4.32	72.50±5.53	NS	59.00±4.75	66.70±3.68	NS
	ii) SVI (mL/m^2)	36.37±2.49	39.96±3.17	NS	32.50±2.69	36.78±2.19	NS
4.	i) CO (L/min)	5.06±0.37	5.66±0.42	NS	4.32±0.47	5.15±0.52	NS
	ii) COI (L/min/m^2)	2.78±0.21	3.11±0.23	NS	2.38±0.26	2.83±0.28	NS
5.	EF (percentage)	66.46±3.03	68.43±2.31	NS	66.75±2.73	68.37±2.33	NS
6.	SBP (mm Hg)	105.50±4.03	108.75±3.98	NS	105.00±5.66	104.50±4.32	NS
7.	DBP (mm Hg)	63.00±2.67	69.00±4.20	NS	63.75±4.60	65.00±4.15	NS
8.	MAP (mm Hg)	77.13±2.93	82.13±3.92	NS	77.46±4.78	78.15±4.12	NS
9.	Calculated PR (R unit)	0.94±0.08	0.92±0.11	NS	1.16±0.13	0.99±0.13	NS

All values are mean ± SEM. Significance was calculated by paired t test.

Responses to head-up tilt

Before SVGN:

Within 3–5 min of 70° HUT (Table III) there was a significant (19.5%) reduction of LVEDV, but the 15.7% fall in SV was not statistically significant. The mild increase in CO also was not significant. The HR, SBP, DBP and MAP were all significantly increased within 3–5 min of 70° HUT.

TABLE III: Changes in Cardiovascular variables during acute head-up tilt before and after Sarvangasana.

	Parameters	Before SVGN			After SVGN		
		Supine	70° HUT	P	Suprine	70° HUT	P
1.	HR (beats/min)	72.51±2.96	91.35±5.41	P<0.01	72.55±2.66	88.52±3.73	P<0.01
2.	i) LVEDV (mL)	97.17±7.20	78.20±5.42	P<0.05	81.41±3.19	78.55±6.28	NS
	ii) LVEDVI (mL/m^2)	53.36±3.74	42.98±2.88	P<0.05	44.81±1.82	43.18±3.47	NS
3.	i) SV (mL)	64.93±5.20	54.68±4.65	NS	53.26±2.27	52.70±3.68	NS
	ii) SVI (mL/m^2)	35.62±2.66	30.05±2.51	NS	29.28±1.25	28.95±2.01	NS
4.	i) CO (L/min)	4.72±0.43	4.94±0.39	NS	3.85±0.17	4.61±0.23	P<0.05
	ii) COI (L/min/m^2)	2.58±0.22	2.70±0.20	NS	2.11±0.09	2.53±0.12	P<0.05
5.	EF (percentage)	66.93±2.21	69.58±1.87	NS	65.12±1.44	67.62±1.41	NS
6.	SBP (mm Hg)	91.52±7.32	103.90±6.75	P<0.01	105.00±4.81	111.25±4.40	NS
7.	DBP (mm Hg)	64.25±2.63	84.75±1.99	P<0.01	72.00±3.74	83.75±3.75	P<0.05
8.	MAP (mm Hg)	77.72±2.61	92.76±2.28	P<0.01	82.97±3.98	92.88±80	P<0.05
9.	Calculated PR (R unit)	1.05±0.12	1.17±0.10	NS	1.30±0.06	1.21±0.03	NS

All values are mean ± SEM. Significance was calculated by paired 't' test.

TABLE IV: Percentage changes in cardiovascular variables in response to HUT.

	Parameters	*Before SVGN*	*After SVGN*	*P (before vs after SVGN)*
1.	HR (beats/min)	+25.98*	+22.01*	NS
2.	LVEDVI (mL/m^2)	−19.45*	−3.63	P<0.05
3.	SVI (mL/m^2)	−15.63	−1.12	P<0.01
4.	COI (L/min/m^2)	+4.65	+19.90*	P<0.01
5.	SBP (mmHg)	+13.52*	+5.95	NS
6.	DBP (mmHg)	+31.90*	+16.31*	P<0.05
7.	MAP (mmHg)	+19.35*	+11.94*	NS

*Shows significant changes
+/−Indicate rise/fall in comparison to supine value.

After SVGN:

The fall in LVEDV and SV seen in response to HUT was absent after SVGN practice. The HR rise was significant but similar in magnitude (22%) to that seen before SVGN. There were no symptoms of syncope in any subject. There was a significant rise in CO (19.75%) in response to HUT after SVGN practice. The DBP and MAP were significantly increased (16% and 11.9% respectively). The increase in arterial blood pressure in response to HUT was somewhat lesser in magnitude than recorded before the asana practice, though this difference was not statistically significant. The calculated PR was increased by 10.5% in response to 70° HUT before asana but decreased by 6.9% after asana. However, these changes were also not statistically significant.

DISCUSSION

Mild head-down-tilt (HDT) bed rest has been used as a method of simulating microgravitational environment (8–10). In this posture the hydrostatic forces on the vascular tree is minimum, blood shifts toward the thorax stimulating cardiopulmonary baroreceptors, and the skeletal muscles remain in a state of inactivity. The frequent activation of arterial baroreceptor mechanisms influencing the heart and vascular smooth muscles as occurs in everyday life is absent. Which of these factors play the dominant role in producing the deconditioning syndrome is not clear (11).

Being a nearly total head-down-body-up posture, the SVGN, on the other hand, simulates a 'negative g' condition rather than microgravity. Though both passive HDT bed rest and SVGN are head-down postures, the SVGN is uniquely different in that in this posture there is a constant stimulation of the arterial high-pressure baroreceptros in addition to the stimulation of low-pressure cardiopulmonary receptors. The resulting sympathetic inhibition, however, is also accompanied by some amount of sympathetic stimulation caused by the isometric contraction of the upper limb and neck muscles to support the body, in SVGN. The interaction of these two factors must be playing a role in determining the net effect of SVGN on the body function.

Effect on resting CV function

Significant reductions of the HR and LVEDV and a tendency to a reduction in LV mass at resting supine state are the main changes found after 2 weeks of SVGN practice.

Inspite of a mild but significant reduction in HR, the resting arterial blood pressure was maintained at pre-asana level. There was a tendency for the SV and CO to fall (10.6% and 14.6% respectively) though these were not statistically significant. The calculated peripheral resistance (PR) was 23% higher than the pre-SVGN value which again was not statistically significant.

The significant reduction of the resting LVEDV is difficult to explain. A decrease in total blood volume which is known to occur in prolonged microgravity condition (12–15) may account for this. This possibility can be ruled out only by measuring blood volume before and after SVGN practice. Alternatively, a decreased sympathetic venomotor tone in the extra-thoracic veins resulting from repeated twice daily stimulation of both the low-pressure and high-pressure baroreceptors may cause a redistribution of blood volume thereby reducing the cardiopulmonary fraction of the total blood volume.

LV mass-The 15.7% reduction of LV mass (also the LV mass index) though found to be just below the statistically significant level, may not be ignored. The LV mass in this experiment was calculated according to the following formula :–

LV mass at end diastole = $0.8 \times 1.04 ([LVIDd + LVPWd + IVSd]^3 - LVIDd^3) + 0.6$. (LVIDd = Left ventricular internal diameter at end diastole, LVPWd = Left ventricular posterior wall dimension at end-diastole, IVSd = Interventricular septal dimension at end-diastole).

In this study the IVS and LVPW did not show any significant change. The reduction in LV mass could be due to the nature of calculation (16).

On the other hand, rapid alterations in LV mass has been demonstrated during initiation or cessation of training (17). Removal of adaptive stimulus is also known to produce regression of LV mass (18–20). Though it appears unlikely that such a brief duration of twice daily exposure to negative g condition produced by SVGN practice for 2 weeks can induce atrophy of myocardium, it need not be ruled out altogether before further study. Cardiac atrophy has been suggested to cause orthostatic intolerance after bed rest deconditioning (21).

Effect on head-down tilt

Head-down tilt at an angle more than 30° is known to stimulate both the low pressure cardiopulmonary and high pressure arterial baroreceptors (22). Cardiovascular responses to HDT show wide variations. The reports available on this are not in accord with one another. Terai (23) found increased LVEDV within 1 min of HDT which disappeared after 10 min but no changes in BP. Weise et al (22) got a significant increase in SBP and a fall in HR without any change in MAP, but Nagaya (24) found no changes in HR and BP though the central venous pressure, SV and CO were increased.

The rise in mean HR in this study, though not found to be statistically significant, could be a manifestation of the Bainbridge effect. The tendency for the LVEDV, SV and CO to rise in response to HDT is expected and is in accord with other's observation. The immediate MAP also showed a tendency to rise before SVGN practice; but this tendency was absent after SVGN. Otherwise, the practice of SVGN did not influence the cardiovascular response to 45° HDT, as measured within 3–5 min of the tilt.

Effect on head-up tilt

Cardiovascular responses to the orthostatic stress produced by the 70° HUT were observed in this study before and after SVGN. A higher incidence of orthostatic hypotention is known to occur in healthy individual following a prolonged HDT bed rest method of microgravity simulation (25). Repetition of an "active head-down-body-up postural exercise" in the form of SVGN for two weeks in our study did not produce any hypotension or syncope-like symptoms in any of the subjects during the 3–5 min of HUT.

When expressed as percentage changes and compared between before and after SVGN practice (Table IV) the changes in HR responses to HUT were insignificant i.e., HR responses to HUT were not significantly modified by SVGN. Changes in LVEDVI, however, were significantly reduced by SVGN, suggesting that practising Sarvangasana significantly minimised the fall in LVEDVI in response to HUT. In a similar way, SVGN significantly reduced the fall in SVI in response to HUT. The COI was thus remarkably raised in response to HUT, after practising Sarvangasana ($P<0.01$). It is difficult to explain how the decrease in LVEDVI seen in response to HUT before the asana practice were absent after asana practice. It appears that the sympathetic venomotor response to HUT was more prompt after SVGN practice.

The rise in SBP in response to HUT was not much altered by SVGN, but the DBP rise in response to HUT was somewhat attenuated after SVGN practice. However, the MAP response to HUT was not significantly altered by practising SVGN for two weeks. This suggests that the SVGN practice modified the baroreflex in these subjects in such a way that there was a greater increase in CO than peripheral resistance (the percentage rise in DBP was somewhat less after SVGN) to maintain a normal rise in MAP in response to the head-up tilt.

Selvamurthy et al. (7) concluded the improvement in baroreflex sensitivity in their hypertensive patients after yoga practice, on the basis of (i) decrease in resting HR, (ii) sharper initial HR response ("rate and magnitude of cardioacceleration") to orthostatic tilt, and (iii) a fall in resting BP. Orthostatic responses in our normal young adults after SVGN resulted in (i) decrease in resting HR, (ii) no significant change in initial HR response, and (iii) no change in resting BP. The differences in the subjects used in these two studies (hypertensive Vs normotensive young adults) are most likely to account for these differences in the results. In addition, as already mentioned, the functioning of the baroreflex in our subjects appears to be altered in such way by SVGN that the orthostatic response was sharper in improving the CO more than by increasing the peripheral resistance in order to maintain the BP.

In conclusion, this first systematic study of sarvangasana shows that a practice of this postural exercise for two weeks produces resting bradycardia and a decreased LVEDVI. It does not alter the CV responses to acute 45° HDT; nor does it impair the normal CV responses to orthostatic challenge of 70° HUT. A further study with a greater number of subject practising this asana for a longer period is necessary to evaluate the potential role of SVGN as a non-pharmacological therapy of hypertension, or as a model exercise in space science research.

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