Indian J Physiol Pharmacol 2002; 46 (3) : 307–312

MODULATION OF COLD PRESSOR-INDUCED STRESS BY SHAVASAN IN NORMAL ADULT VOLUNTEERS

MADANMOHAN, KAVIRAJA UDUPA, ANANDA BALAYOGI BHAVANANI, N. KRISHNAMURTHY AND G. K. PAL

Department of Physiology, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry – 605 006

(Received on March 5, 2002)

Abstract : Shavasan is known to enhance one's ability to combat stressful situations. The present study was planned to determine if shavasan could modulate the physiological response to stress induced by cold pressor test (CPT) and the possible mechanisms involved. Ten normal adults were taught shavasan and practised the same for a total duration of seven days. RR interval variation (RRIV), deep breathing difference (DBD), and heart rate, blood pressure & rate-pressure-product (RPP) response to CPT were measured before and immediately after shavasan. Shavasan produced a significant increase in DBD and an appreciable but statistically insignificant increase in RRIV suggesting an enhanced parasympathetic activity. Significant blunting of cold pressor-induced increase in heart rate, blood pressure and RPP by shavasan was seen during and even five minutes after CPT suggesting that shavasan reduces the load on the heart by blunting the sympathetic response. It is concluded that shavasan can enhance one's ability to withstand stress induced by CPT and this ability can be achieved even with seven days of shavasan training.

Key words : cold pressor test; deep breathing difference; RR interval; shavasan

INTRODUCTION

Modern age is the age of stress and stress-induced disorders are posing a great challenge to the present society. Yogic techniques in general and shavasan in particular are known to improve psychosomatic health and enhance one's ability to combat stressful situations. Datey et al (1), Patel and North (2) and Wilson et al (3) have reported the effectiveness of shavasan in the management of psychosomatic disorders such as hypertension and bronchial asthma. The effectiveness of shavasan in producing psychosomatic relaxation has been reported by earlier workers (4, 5, 6, 7). Agarwal et al (8) have studied the effect of shavasan on vascular responses in hyper-reactors by means of cold pressor test (CPT), which has been used as a means to assess the stress-induced sympathetic response since 1932 (9).

*Corresponding Author

However, to the best of our knowledge there is no scientific report on the effect of shavasan on the physiological response to CPT in normal adults. Moreover, no study has been done to determine the effect of a single session of shavasan on physiological functions. Hence, we planned to study if shavasan is effective in modifying the response to stress induced by CPT in normal adult volunteers. In order to determine the mechanism of anti-stress effect of shavasan, if any, we also included in the present study, deep breathing difference (DBD) and RR interval variation (RRIV), both indicators of parasympathetic activity (10, 11).

Hence the present study was designed with the following objectives :

1. Whether a single session of shavasan can modulate the physiological response to stress induced by CPT even of the duration of shavasan training is only 7 days.
2. To explore the possible mechanisms involved in achieving this ability.

METHODS

Healthy young adult volunteers (6 males and 4 females) were recruited for the present study. Their age was 22–30 (26.6 ± 0.78, S.E.) years, weight 60–84 (65.9 ± 2.48, S.E.) kg, and height 160–178 (166.9 ± 1.94, S.E.) cm. Prior approval was obtained from the Institute Ethical Committee and informed consent was obtained from the subjects. They were taught shavasan and practised the same for 15 minutes daily under our direct supervision for a total duration of seven days. The technique of shavasan is given elsewhere (12). After the 7-day training was over, recordings were taken as per the following protocol :

Supine rest	RRIV	DBD	CPT	Break	Shavasan	RRIV	DBD	CPT
10 min	3 min	1 min	2+5 min	10 min	10 min	3 min	1 min	2+5 min

Recordings were taken in an air-conditioned laboratory (room temperature 27 ± 1°C) two hours after light breakfast. RRIV was measured from 150 successive RR intervals and expressed as the coefficient of variation (CV) about mean RR interval from RR interval sequence (CV = SD/mean X 100). This parameter has been reported to be a reliable and reproducible measure of cardiac parasympathetic activity (11). DBD was determined by asking the subject to breathe deeply and uniformly at a rate of six breaths per minute, taking 5 seconds for inspiration and 5 seconds for expiration. Lead II ECG was recorded on an 8-channel polygraph (RM 6000, Nihon Kohden Corporation, Japan). The signal thus obtained was converted into digital format using analog-digital converter (Mi², USA) and analysed with the help of a data processing software (Bio Windows, Modular Instruments Inc. USA). With this software instantaneous heart rate was obtained with an accuracy of 0.01 beats/min. Maximum and minimum heart rates during each cycle were identified from the heart rate plot and the difference between them was calculated. Mean of the difference during six such successive respiratory cycles was calculated and rounded to the nearest whole integer and expressed as DBD (10).

CPT was performed by asking the subject to immerse his/her hand in cold water maintained at 4 ± 0.5°C and the hand was kept immersed in the water up to the distal palmar crease for a duration of two minutes (13, 14). The subjects were instructed to relax, breathe quietly and avoid Valsalva-like maneuver during the immersion. After the immersion, the hand was dried on a towel (15). To avoid observer bias in the measurements, blood pressure and heart rate were recorded using automatic non-invasive blood pressure apparatus (Press-Mate BP 8800, Colin Corporation, Japan). Readings were taken before the immersion, at 30 s intervals during the immersion and at 1, 2, 3, 4 and 5 minutes during the post-immersion period. Rate-pressure-product (RPP), which is an index of myocardial oxygen consumption (16) was calculated as a product of heart rate (HR) and systolic blood pressure (SP) divided by 100 ($RPP = HR \times SP \times 10^{-2}$).

The data was analysed using Student's paired 't' test. P values of less than 0.05 were accepted as indicating significant difference between the compared values.

RESULTS

The results are given in Table I and Fig. 1. Basal RRIV was 5.82 ± 0.58 (SE). After shavasan, it increased to 6.47 ± 1.12, the increase being statistically insignificant (Fig. 1). DBD increased significantly from the control value of 21.53 ± 2.58 to 24.78 ± 2.72, the P value being less than 0.01. After the performance of shavasan, there was statistically significant (P<0.05) decrease in the basal heart rate (Table I). Immersion of hand in cold water produced a marked and statistically significant increase in the heart rate. However, it returned towards the pre-immersion value by the end of 2 minutes immersion period. This immersion-induced increase in the heart rate was significantly blunted following shavasan practice. During the post-immersion period, heart rate was lower

TABLE I: Effect of *shavasan* on cold pressor response. Heart rate (HR), systolic pressure (SP), diastolic pressure (DP) and rate-pressure-product (RPP) before (B) and after (A) *shavasan*.

	HR (beats/min)		*SP (mmHg)*		*DP (mmHg)*		*RPP*	
	B	*A*	*B*	*A*	*B*	*A*	*B*	*A*
Basal	72.6±3.17	68.2±2.71*	107.6±2.15	106.6±2.9	60.2±1.46	60.8±2.06	78.3±4.29	73.0±4.18*
During immersion								
30 s	86.2±4.63	78.9±3.67**	125.2±4.48	122.2±3.81	70.9±2.65	72.5±2.4	109.2±9.01	97.1±6.68**
60 s	81.4±4.09	79.0±4.65	134.3±5.35	130.8±4.09	78.0±3.15	76.7±3.29	109.8±7.51	103.7±7.32*
90 s	76.8±3.2	74.3±3.38	129.4±4.17	131.6±4.23	77.1±2.53	75.7±4.1	99.6±5.74	97.9±5.64
120 s	73.7±3.27	72.5±3.1	130.6±4.3	133.0±4.83	71.5±2.85	71.3±3.31	96.3±5.19	97.2±6.57
Post immersion								
180 s	68.8±4.15	62.4±2.22*	114.2±4.42	113.5±4.37	61.2±2.12	59.2±2.59	78.6±5.49	70.5±2.79*
240 s	69.1±2.88	64.8±2.55**	109.7±2.74	108.4±3.7	58.6±1.85	55.6±1.56	76.0±3.97	70.2±3.63**
300 s	68.1±3.14	64.1±2.44*	108.8±2.35	106.6±3.18	56.9±1.61	56.9±1.49	74.2±4.05	68.6±3.77*
360 s	67.5±3.12	65.3±2.32	108.8±2.37	105.2±2.56*	57.7±1.45	53.2±1.2**	73.7±4.33	68.9±3.34*
420 s	67.7±2.9	63.7±2.34*	105.3±2.99	106.1±2.84	56.4±1.87	55.0±1.44	71.4±3.85	67.8±3.66*

Values are mean ± SE from 10 subjects. *P<0.05; **P<0.01.

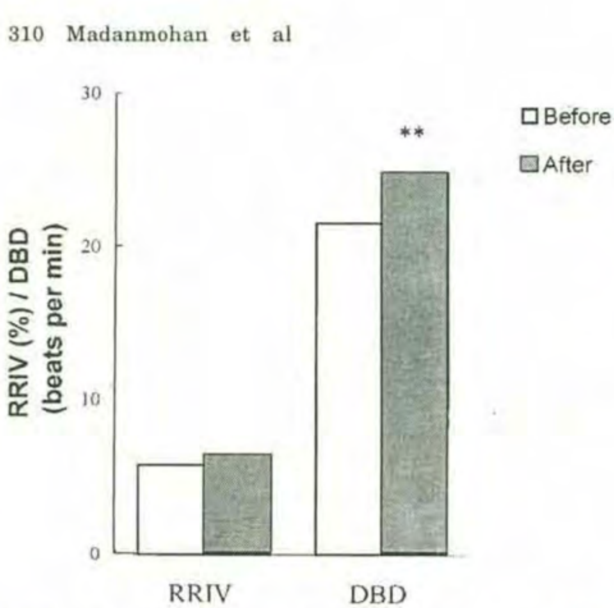

Fig. 1: RR interval variation (RRIV) and deep breathing difference (DBD) before and after shavasan practice. **P<0.01

than the basal value. The decrease in heart rate was more pronounced and statistically significant (P<0.01) after shavasan practice. There was a significant increase in systolic as well as diastolic pressures following cold immersion (Table I). Both these values returned towards the basal values during the post-immersion period. After shavasan, this immersion-induced increase in blood pressures was less marked. Following the performance of shavasan, there was decrease in systolic and diastolic pressures during the post-immersion period and this was statistically significant at the fourth minute of post-immersion period (P<0.05 and P<0.01 respectively). During the immersion as well as the post-immersion periods the trend of changes in RPP was similar to that of heart rate.

DISCUSSION

It has been reported that shavasan produces a significant decrease in heart rate and blood pressure (1, 4, 6, 7). These studies were based on shavasan training of long duration. The present study was planned to determine whether shavasan enhances one's ability to withstand stressful stimuli and whether this ability can be achieved with a shavasan training of short duration (7 days). Earlier workers (4) have used exercise as the stressful stimulus, but we used CPT, a known stressor (9), in the present study.

In our subjects performance of shavasan produced a significant decrease in heart rate. Earlier workers (1, 4, 6, 7) have reported that shavasan training produces a significant decrease in basal heart rate. It is interesting to note that the duration of shavasan training in our subjects was only seven days whereas the training period was longer in earlier studies. This decrease in heart rate after performance of shavasan can be explained on the basis of an increase in parasympathetic tone as evidenced by increased RRIV (by 11.18%) and DBD (by 15.11%, P<0.01) in our subjects. Increase in RRIV and DBD are known to indicate enhanced cardiac parasympathetic activity (10, 11).

Immersion of hand in cold water produced a marked increase in heart rate (Table I). This increase in heart rate has been attributed to an increase in the sympathetic activity with release of norepinephrine and epinephrine (15, 17, 18, 19). This cold pressor-induced increase in heart rate was significantly blunted by shavasan. It is interesting to note that this effect continued even in the post immersion period, suggesting that the physiological response to shavasan continues for a longer time.

In the present study, basal systolic and diastolic pressures were not affected by performance of shavasan. Udupa et al (7) also have reported that shavasan training of two months does not produce any decrease in resting blood pressure. In an earlier study, we have found that shavasan produces a significant fall in basal blood pressure in subjects trained for one year (6). From the present study it is clear that after a short training of seven days, one can achieve a significant reduction in heart rate but not blood pressure by shavasan. In all our subjects immersion of hand in cold water produced a marked increase in systolic as well as diastolic blood pressures. Other workers have also observed the same response and attributed this to increased sympathetic activity (13, 15). This increase in systolic and diastolic pressures was reduced after the practice of shavasan although this reduction was statistically insignificant. Our findings are similar to those of Khanam et al (20) who found that seven days of yogasan training in asthmatics produces a significant reduction in heart rate but no significant change in blood pressure in response to CPT. During the post immersion period, systolic and diastolic blood pressures were reduced after shavasan practice and this reduction was statistically significant at fourth minute of the post immersion period ($P<0.05$ and <0.01 respectively, Table I).

Datey et al have attributed this modulation of heart rate and blood pressure response by shavasan to an altered proprioceptive and enteroceptive influences to the hypothalamus (1). Dikshit et al have reported that CPT-induced increase in the heart rate and blood pressure is significantly reduced by propranolol (13). This suggests that shavasan-induced decrease in heart rate and blood pressure is due to reduced sympathetic activity. From this, it can be inferred that shavasan-induced modulation of heart rate and blood pressure response to CPT can be explained on the basis of altered hypothalamo-sympathetic activity.

Immersion of hand in cold water produced a marked and significant increase in RPP (Table I). In our subjects, the maximum value of RPP during the immersion was 109.8 ± 7.51. Dikshit et al (13) have also reported that the highest value of RPP during cold pressor test is 109 units. RPP, which is an index of myocardial oxygen consumption (16) indicates the load on the myocardium. It has also been reported that myocardial ischemic pain during exercise in patients with coronary artery disease is associated with a rise in RPP to about 200 units (21). In the present study shavasan produced a significant blunting of RPP response to CPT (Table I). CPT-induced maximum rise of RPP (103.7 ± 7.32) was significantly less than pre-shavasan value ($P<0.05$). This shavasan-induced blunting of RPP response was maintained throughout the five-minute post immersion period. Thus, the present study shows that shavasan reduces the load on heart and this beneficial effect is maintained during the post shavasan period as well.

In conclusion, our study shows that a session of shavasan can enhance the parasympathetic activity, blunt the sympathetic activity and reduce the load on the heart. Further, shavasan can enhance one's ability to withstand stress induced by CPT. Shavasan can be learnt sufficiently well to achieve these results even with seven days of shavasan training. If might be argued that the effect observed in the study is not due to shavasan *per se* but due to the additional rest after the 10 minutes of supine rest. For this another study which has a control group incorporating supine rest instead of shavasana is required. But tentatively, it may be concluded that a session of shavasana has beneficial effects, and that physiologically effective shavasan can be learnt in as short a period as seven days.

ACKNOWLEDGEMENTS

The authors wish to express their gratitude to the Central Council for Research in Yoga and Naturopathy (CCRYN) for funding the Research Project.

REFERENCES

1. Datey KK, Deshmukh SN, Dalvi CP, Vinekar SL. Shavasan : a yogic exercise in the management of hypertension. *Angiology* 1969; 20: 325–333.
2. Patel C, North WRS. Randomised controlled trial of yoga and biofeedback in management of hypertension. *The Lancet* 1975; 2: 93–95.
3. Wilson AF, Honsberger R, Chiu JT, Novey HS. Transcendental meditation and asthma. *Respiration* 1975; 32: 74–80.
4. Bera TK, Gore MM, Oak JP, Recovery from stress in two different postures and in shavasan - a yogic relaxation posture. *Indian J Physiol Pharmacol* 1988; 42: 473–478.
5. Gopal KS, Anantharaman V, Nishith SD, Bhatnagar OP. Effect of yogasan on muscular tone and cardiorespiratory adjustments. *Indian J Med Sci* 1974; 28: 438–443.
6. Madanmohan, Rai UC, Balvittal V, Thombre DP, Swami Gitananda. Cardiorespiratory changes during savitri pranayam and shavasan. *The Yoga Review* 1983; 3: 25–34.
7. Udupa KN, Singh RH, Settiwar RM. Studies on the physiological effects of the yogic posture shavasan. *J Res Indian Med Yoga Homeopathy* 1978; 12: 147–149.
8. Agarwal RC, Dikshit N, Maheshwari R, Bose S, Bisariya BN. Effect of shavasan on vascular response to cold pressor test, serum cholesterol level and platelet stickiness in hyperreactors. *Indian Heart J* 1977; 29: 182–185.
9. Hines EA, Brown GE. A standard stimulus for measuring vasomotor reactions : its application in the study of hypertension. *Mayo Clin Proc* 1932; 7: 332–335.
10. Mathias CJ, Bannister R. Investigation of autonomic disorders. In Bannister R, Mathias CJ eds. Autonomic failure : A textbook of clinical disorders of the autonomic nervous system. Oxford, Oxford University Press 1992: 268.
11. Ewing DJ. Analysis of heart rate variability and other non-invasive tests with special reference to diabetes mellitus. In Bannister R, Mathias CJ eds. Autonomic failure : A textbook of clinical disorders of the autonomic nervous system. Oxford, Oxford University Press, 1992: 312–314.
12. Swami Gitananda. Shavasana, the corpse posture. In Yoga : Step-by-Step Pondicherry, Satya press 1981: A-4.
13. Dikshit MB, Patrick JM. Beta-adrenoreceptor blockade and cardiovascular response to the cold pressor test. *Indian J Physiol Pharmacol* 1986; 30: 1–10.
14. Blanc LJ, Dulac S, Cote J, Girad B. Autonomic nervous system and adaptation to cold in man. *J Appl Physiol* 1975; 39: 181–186.
15. Fagius J, Karhuvaara, Sundlof G. The cold pressor test: effects on sympathetic nerve activity in human muscle and skin nerve fascicles. *Acta Physiol Scand* 1989; 137: 325–334.
16. Gobel FL, Nordstrom LA, Nelson RR, Jorgenson CR, Wang Y. The rate-pressure-product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. *Circulation* 1978; 57: 549–556.
17. Houben H, Thien Th, Wijnands G, Laar AVt. Effects of cold exposure on blood pressure, heart rate and forearm blood flow in normotensives during selective and non-selective β-adrenoreceptor blockdae. *Br J Clin Pharmacol* 1982; 14: 867–870.
18. Robertson D, Johnson GA, Robertson RM, Nies AS, Shand DG, Oates JA. Comparative assessment of stimuli that release neuronal and adrenomedullary catecholamines in man. *Circulation* 1979; 59: 637–643.
19. Sethi A, Vaney N, Tandon OP. Sensory nerve conduction during cold pressor response in humans. *Indian J Med Res* 1994; 99: 279–282.
20. Khanam AA, Sachdeva U, Guleria R, Deepak KK. Study of pulmonary and autonomic functions of asthma patients after yoga training. *Indian J Physiol Pharmacol* 1996; 40: 318–324.
21. Robinson BF. Relation of heart rate and systolic blood pressure to the onset of pain in angina pectoris. *Circulation* 1967; 35: 1073–1083.