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HEART RATE AND BLOOD PRESSURE RESPONSES OF LEFT-HANDERS AND RIGHT-HANDERS TO AUTONOMIC STRESSORS

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Abstract : We hypothesized that cerebral dominance may contribute to differences in cardio-vascular responses of right-handers (RH) and left-handers (LH) to autonomic stressors. We tested this hypothesis by exposing 14 RH, and 14 LH males to *category I tests* in which the hand and cerebral cortex were involved in performing the test viz.- i) Cold pressor test (CPT), ii) Handgrip dynamometry (HGD) and; *category II* (no use of hand)- i) Orthostatic Tolerance Test (OTT), ii) Valsalva Manuever (VM), iii) Controlled Breathing Test for sinus arrhythmia (SA) in a random sequence, and measured their heart rate (HR/min) and blood pressure (MAP mmHg).

All subjects had similar resting HR and MAP values, and responded to the category I interventions with increased HR and BP. The absolute HR values of LH and RH did not differ significantly during the interventions. However, the increase in HR from control induced by the CPT, and the HGD was greater for LH ($P<0.05$). Also, LH showed a greater decrease in HR and MAP in the recovery phase ($P<0.05$). The VAS scores for degree of discomfort during the CPT were similar for both the groups. During the OTT, the increase in HR was more in RH ($P<0.05$). The Valsalva ratios for LH and RH were similar.

Our findings suggest that the autonomic control over the cardio-vascular system may be different in LH and RH, and that this imbalance could be attributable to a variation in cerebral dominance.

Key words : autonomic responses, cerebral asymmetry, cold pressor test, handedness, handgrip dynamometry, orthostatic tolerance test

INTRODUCTION

The physical expression of brain asymmetry is translated as right-handedness and left-handedness in humans (1). Brain asymmetry has also been seen as a difference in EEG responses of left-handers (LH) and right-handers (RH) to neuroleptic drugs, antihistamines and analgesics - drugs, which act centrally (2).

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This led us to use iv lobeline to stimulate J receptors and induce respiratory sensations and reflexes in left handed and right handed subjects (3). Contrary to expectations, the sensations generated did not differ, but a prominent reflex bradycardia occurred in the left handers. This could have represented a difference in overall autonomic control of the heart between the two groups.

Each hemisphere exhibits differential control over dynamics of the heart involving heart rate, blood pressure and myocardial contractility (4). The right hemisphere exerts greater control over heart rate while the left hemisphere affects myocardial contractility (5, 6). These studies suggest that cerebral hemispheres have differential control over the heart which must happen via the autonomic nervous system. There is evidence that sympathetic activity is controlled by the right cerebral hemisphere (4), which is dominant in left handers (7). Therefore, handedness may also modulate autonomic control of the heart. But none of the above studies take handedness into consideration. Following this argument, and our observation of reflex bradycardia in left-handers (3), we examined the hypothesis that responses of heart rate and blood pressure to autonomic stressors is likely to be different in left-handers and right-handers.

METHODS

Healthy young male volunteers (non-smokers) were recruited for the study, which was approved by the Ethical Committee of this Medical College. Of these, 14 were right-handers (mean age 19.5 yr ± SD 1.1; weight 57.7 kg ± 9.2, height 165.9 cm ± 6.7); while 14 were left-handers (20.1 yr ± 1.1, 56.7 Kg ± 6.1; 168.9 cm ± 6.1). Their physical attributes were not statistically different ($P>0.05$). They were free of any clinical disorder, and were not taking any medications. Their resting heart rate range was within 60–100 beats/mn. Their handedness was determined using the Lateral Preference Inventory (8). All the experiments were carried out in the Physiology laboratory between 9–11 am at a room temperature of 28°–29°C. The details of the experiment protocol were explained to all the subjects, and their written consent was obtained.

The tests selected for the assessment of the Autonomic Functions were: 1) Cold Pressor test (CPT); 2) Handgrip dynamometry (HGD); 3) Orthostatic Tolerance Test (OTT); 4) Controlled Breathing test for Sinus Arrhythmia (SA); and 5) Vasalva Manuever (VM). They were given as per the protocol described (9). The heart rate (HR) was recorded using three chest electrodes on one of the two channel Physiograph (Biodevices; Chandigarh) at a paper speed of 5 mm/sec (calibrated). Respiratory movements were recorded simultaneously on the other channel with a T 303 differential pressure transducer (Biodevices). The blood pressure was measured where applicable with a standard mercury sphygmomanometer. The recording of the BP and the HR coincided during each test at the designated time interval. The 5 tests were arbitrarily classified as a. *Category I Tests* - which involved use of the dominant hand in performing the experiment, and cortical influences to evoke autonomic responses viz. CPT and HGD (10, 11), and b. *Category II*

Tests - which excited the reflex autonomic responses at the sub-cortical level without the involvement of the hand viz OTT, SA and VM.

Every subject was familiarized with the test to be undergone. On the day of the experiment, the subject reported to the laboratory at 0900 hr. He was connected for the ecg and respiration recording. The baseline BP and HR were recorded after a rest of about 10 min. He was then given all five stress tests in a random sequence determined independently by one of us (VRP). It was ensured that after every test, the physiological variables returned to baseline before administering the subsequent test.

1. Cold pressor test (CPT)

The pre test HR and BP were measured in the sitting position. The dominant hand of the subject (as decided previously by LPI score) was immersed in ice-cold water at 4°C for 2 min. The HR and BP were recorded at every ½ min interval for 2 min during immersion and recovery. The subject then marked the degree of discomfort and/or pain felt during the test on a VAS scale (Visual Analogue Score; 0-no discomfort to 10-intolerable).

2. Handgrip dynamometry (HGD)

The test was done using a standard handgrip dynamometer (Anand Agency, Pune; range 0–100 kg). The pre test HR and BP were obtained in the sitting position. The subject generated maximum force with the dominant hand. He was then asked to maintain 30% of maximum force generated for 2 min as test. The HR and BP were recorded at 0, 1 and 2 min intervals during the grip, and after releasing the grip.

3. Orthostatic tolerance test (OTT)

Pre test HR and BP were obtained in the supine position. The subject then stood up rapidly and maintained this position without support for 5 min. BP and HR were recorded at 0 min (immediately on standing), and thereafter at the 1st, 2nd, and 5th min of standing. The subject then went back to the recumbent position with the same precautions, and the measurements were repeated as above. This simple method was used as orthostatic stress testing may be done and interpreted without sophisticated equipment (12).

The changes induced by the interventions in test nos. 1, 2 and 3 for HR and Mean Arterial Pressure (MAP = DBP + 1/3 PP) were calculated against the pretest values. The end of the intervention period value of the variables was taken as reference values for calculating the changes during recovery.

4. Controlled Breathing test (for sinus arrhythmia) (SA)

The subject was asked to inspire deeply over 5 sec, and breath out over 5 sec (Respiratory rate 6 per minute). HR and respiration were recorded simultaneously and continuously. (BP was not recorded). The ratio of six longest RR intervals during expiration to six shortest RR intervals during inspiration (E : I ratio), and the

differences between maximum inspiratory HR, and minimum expiratory HR ($HR_{max-min}$) were obtained.

5. Valsalva manuever (VM)

The subject was asked to breath out at the end of tidal inspiration into a mercury manometer to raise the mercury column to 40 mm Hg, and maintain it till break point, or for a maximum period of 1 min. A trace of respiratory movements was monitored continuously on the Physiograph to ensure proper breath-hold. HR was recorded continuously. The ratio of the longest RR interval after the VM to the shortest RR interval during the breath hold (Valsalva ratio-VR) was calculated (9).

Statistics

The sample means and standard deviations were tested for normality. The Fisher's 'F' test was applied to test the significance of SD; and Mann-Whitney test, and Student's unpaired 't' test were applied for testing the significance of means at $P<0.05$ (13).

RESULTS

There was no statistical difference between the mean ± SD baseline values for HR (LH: 82.1 ± 19.8; RH: 81.3 ± 13.8), and MAP (LH 89.0 ± 8.8, RH 90.9 ± 6.5; $P>0.05$) amongst LH and RH before commencement of the interventions.

Category I test: (CPT and HGD)

Heart rate responses

The mean HR in both the RH and LH increased during these tests (Table I) but there was no statistical difference between the two groups. However, LH showed a greater change in the HR from pre test values as compared with the RH at

TABLE I: Depicts mean ± SD of heart rates in 14 right-handers (RH) and 14 left-handers (LH) during a: Cold Pressor Test (CPT); and b: Handgrip Dynamometry (HGD), and recovery after the test. The values were not significantly different.

a. Heart rate during CPT

		Test					*Recovery*				
	C	*0 min*	*0.5 min*	*1 min*	*1.5 min*	*2 min*	*0 min*	*0.5 min*	*1 min*	*1.5 min*	*2 min*
RH	80.5±10.6	93.6±17.3	87.9±16.3	86.5±15.8	87.1±15.8	83.7±13.2	84.6±15.4	76.5±11.6	77.5±11.2	80.1±13.6	88.4±11.3
LH	82.6±16.9	91.4±14.1	88.9±15.7	90.3±16.1	90.3±16.3	89.2±16.5	88.3±17.3	78.1±18.1	77.9±15.4	78.6±13.0	79.2±17.2

b. Heart rate during HGD

		Test			*Recovery*		
	Control	*0 min*	*1 min*	*2 min*	*0 min*	*1 min*	*2 min*
RH	79.4±9.5	93.2±15.2	89.2±12.1	91.7±13.6	83.7±11.6	78.3±10.0	77.2±09.0
LH	78.9±14.6	91.3±17.2	90.4±14.9	96.7±14.2	85.2±13.4	79.8±14.2	79.5±15.4

TABLE II: Gives the change in heart rate (mean ± SD) from control value in 14 RH and 14 LH during a: CPT; and b: HGD. *=P<0.05. All others not significant. The – ve sign during the recovery phase denotes the fall in the variable from the end-test value.

a. Change in HR during and after CPT

	Test					*Recovery*				
	0 min	*0.5 min*	*1 min*	*1.5 min*	*2 min*	*0 min*	*0.5 min*	*1 min*	*1.5 min*	*2 min*
RH	13.0±11.0	4.9±11.8	5.9±10.1	7.1±11.9	3.4±7.8	0.1±10.4	–7.6±8.4	–6.6±6.3	–3.7±7.3	–3.5±6.8
LH	9.3±9.3	6.3±8.4	7.7±9.6	8.0±12.8	7.2±11.4	–1.4±7.9	–11.9±9.3	–12.5±9.7	–9.5±12.5	–10.7±10.6
Sig.								*	*	*

b. Change in HR during and after HGD

	Test			*Recovery*		
	0 min	*1 min*	*2 min*	*0 min*	*1 min*	*2 min*
RH	13.8±10.7	9.8±8.6	12.2±9.1	–7.9±7.7	–12.8±9.3	–14.4±10.3
LH	12.4±7.8	11.9±8.0	17.9±11.1	–11.5±7.8	–16.9±12.9	–17.3±12.9
Sig.			*			

the end of 2 min of the CPT (P<0.05). The degree of fall in HR that occurred in the post CPT period in the LH was also significantly greater (P<0.05) (Table II). The peak change in HR reached at the end of 2 minutes during handgrip was significantly greater in LH (17.9 ± 11.1 for LH; 12.2 ± 9.1 for RH; P<0.05) (Table II).

BP responses

The mean BP values of the two groups during these tests are shown in Fig. 1. The mean pre-test values of BP in left-handers were lower than for the RH (P<0.05) for both the tests. During the stress, there were no significant differences, but in the recovery phase, the LH had a significantly lower BP. The changes in the mean MAP in LH during this test were significantly more (Table III).

VAS: The VAS for pain sensation/discomfort

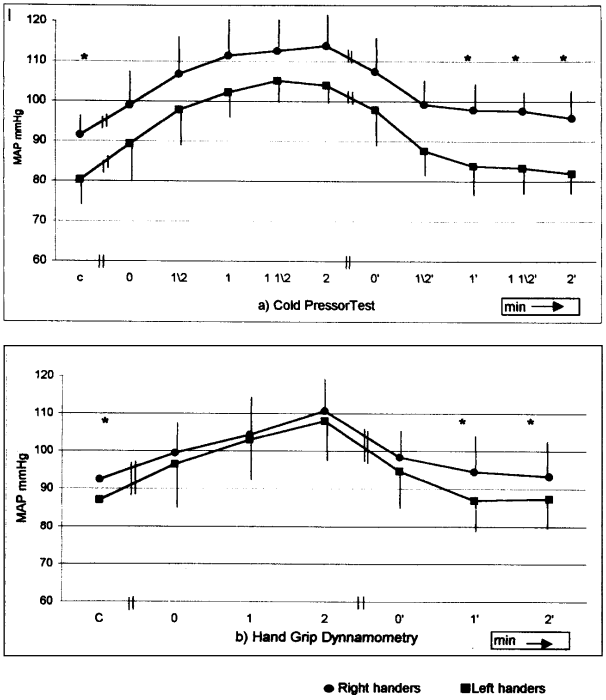

Fig. 1: Represents the mean values ± S.D. for mean arteral BP (MAP; mmHg) at control (C), during the tests - 0 to 2 min, and during the recovery period (0' onwards) during a) the Cold Pressor test (CPT) and b) Hand Grip Dynamometry (HGD). The * denotes (P<0.05). All other values are not significant. || Indicate break in the time axis.

TABLE III: Shows the change in mean arterial pressure (MAP) mmHg (mean ± SD) during a: CPT; and b: HGD., and recovery from the tests. *=P<0.05. All others not significant. The – ve sign during the recovery phase denotes the fall in the variable from the end-test value.

a. Change in MAP during and after CPT

	Test					*Recovery*				
	0 min	*0.5 min*	*1 min*	*1.5 min*	*2 min*	*0 min*	*0.5 min*	*1 min*	*1.5 min*	*2 min*
RH	7.5±5.4	15.3±5.1	19.9±7.7	21.1±7.7	22.4±9.1	–6.4±4.9	–14.6±8.0	–16.0±8.9	–16.1±8.9	–18.0±8.3
LH	9.6±5.3	18.8±5.3	23.5±4.6	26.6±5.9	25.3±8.5	–6.4±4.4	–17.5±6.5	–21.5±5.8	–21.9±5.8	–23.5±5.9
Sig.		*		*				*	*	*

b. Change in MAP during and after HGD

	Test			*Recovery*		
	0 min	*1 min*	*2 min*	*0 min*	*1 min*	*2 min*
RH	7.1±5.1	11.9±6.3	18.2±8.9	–12.3±4.7	–6.2±7.7	–17.3±7.1
LH	9.4±5.0	15.9±6.9	20.9±7.3	–13.3±3.9	–21.1±7.1	–20.6±7.3
Sig.		*			*	*

TABLE IV: Depicts a. change in heart rate (beats/min), and b. change in MAP (mmHg) before, during 5 min of quiet standing, and return to supine, for right handers (RH) left handers (LH). *=P<0.05; all others not significant. The – ve sign during the recovery phase denotes the fall in the variable from the end-test value.

a. Change in heart rate during and after OTT

	Test				*Recovery*			
	0 min	*1 min*	*2 min*	*5 min*	*0 min*	*1 min*	*2 min*	*5 min*
RH	32.0±10.5	18.2±8.5	20.8±10.1	20.1±11.1	–7.5±13.3	–17.3±10.4	–19.1±10.6	–20.9±9.8
LH	29.9±8.1	10.9±7.0	13.9±7.2	13.9±8.7	6.9±15.4	–18.1±8.6	–20.6±9.5	–20.5±9.5
Sig.		*	*					*

b. Change in MAP during and after OTT

	Test				*Recovery*			
	0 min	*1 min*	*2 min*	*5 min*	*0 min*	*1 min*	*2 min*	*5 min*
RH	5.8±7.4	7.7±7.2	7.9±6.3	9.2±6.4	–9.1±6.1	–9.5±5.7	–9.3±5.9	–9.8±6.0
LH	4.5±5.7	5.8±6.0	6.8±5.6	5.4±5.2	–3.0±5.6	–4.6±5.8	–5.4±6.0	–6.9±5.9
Sig.								

during the CPT was scaled at 4.6 ± 2.2 for LH and 5.0 ± 2.6 for RH; (P>0.05).

In summary, category I tests brought out a significant difference in the HR and BP responses of LH and RH.

Category II tests:

Orthostatic tolerance test

HR response

All our subjects responded to Orthostatic stress with the expected increase in HR and the MAP (Fig. 2). The mean absolute HR while standing, and in the post stress period did not differ significantly between the two groups (P>0.05). However, the change in HR while upright was less for LH than for RH (P<0.05; Table IV). The two groups had similar HR during recovery.

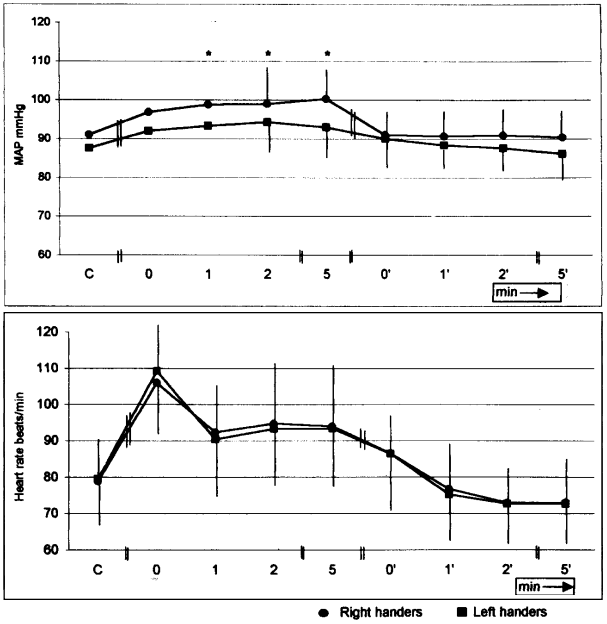

Fig. 2: Mean Arterial Pressure (MAP; mmHg) and Heart rate (HR/min) values (Mean ± SD) before (C), during 1, 2 and 5 min of quiet standing (OTT), and during 5 min of recovery in the supine posture in 14 RH and 14 LH. The * denotes P<0.05. All other values are not significant. || Indicate break in the time axis.

Mean arterial pressure (MAP) response

The pre OTT MAP was 87.5 ± 6.9 mmHg in LH and 91.0 ± 7.6 mmHg in RH (P>0.05). At 1, 2 and 5 min during standing, and at the fifth min of recovery, there was a significant difference between the two groups (P<0.05; Fig. 2). The change from pre test MAP between the two groups during this test was not significant (Table IV).

Controlled breathing test (for sinus arrhythmia) (SA)

$HR_{max-min}$ as an indicator of SINUS ARRHYTHMIA was 18.1 ± 7.0 in LH, and 15.7 ± 5.2 in RH (P>0.05). However the SD for this change was significantly different (P<0.05). Because of this finding, a ranking test (Mann Whitney) was applied to the medians of the two values (17.5 ± 3.3 LH and 16.7 ± 1.5 in RH), which were found to be statistically significant (P<0.05). This helped to conclude that the test induced a difference in response between the two groups. E/I ratios for LH and RH were 1.37 ± 0.24, and 1.47 ± 0.18 respectively (P>0.05).

Valsalva manuever (VM)

The Valsalva ratio during the VM was 1.44 ± 0.41 for RH and 1.59 ± 0.43 for LH (P>0.05). The mean breath hold time (BHT) values of 28.3 ± 7.6 sec for RH, and 25.5 ± 8.4 sec for LH were also not significantly different.

DISCUSSION

We report here a new finding that the cardiovascular reflex responses to stress tests that invoke autonomic reactions are different in LH as compared with RH in some aspects.

All the five tests given during the study are known to exert autonomic influences over heart functions (9, 12) and thus their use by us in the present series of experiments is validated. We chose to use a number of tests because there is *no single best test*, to indicate autonomic responses to stress (12). The tests chosen were such that each employed a different sensory input, viz cutaneous cold and pain during CPT, stimulation of muscle spindles during the handgrip test, and deactivation of baroreceptors during quiet standing (OTT). Marked increase in sympathetic muscle nerve activity was shown to occur in normal human subjects exposed to CPT (14), and during sustained handgrip at 30% of maximal voluntary contraction (15, 16), under influence of a central command (15). The above were the basis of our arbitrary classification of tests as 'category I' and 'category II'. In category II tests the hand is not used while generating reflex autonomic response.

The right hemisphere has been associated with chronotropic effect on the heart as well as sympathetic activation (6). Left-handers are thought to have a right hemispheric dominance (7). Also, cortical influences have been linked to tachycardia response during isometric exercise viz handgrip test (10, 11, 15). Hence, it was expected that when left-handers use their dominant hand to perform the tests, they would generate more sympathetic drive probably through right hemispheric activation. Our findings in Cat I tests for the HR (Table II) and MAP (Table III) substantiate this hypothesis. The VAS scores for the degree of discomfort/pain during the CPT were similar for both the groups (4.5 ± 2.2 for LH; 5.03 ± 2.61 for RH; $P>0.05$). Therefore, the difference in the HR change between the LH and RH was not influenced by the pain/discomfort felt during this stress test.

Apart from reacting with powerful sympathetic activation during category I tests, they also seem to demonstrate a greater degree of withdrawal of the sympathetic activation on termination of the stress as seen from a greater fall in the HR (Table II), and the MAP during the recovery phase (Table III). No explanation can be offered at this stage for this observation.

Category II tests also bring out differences between the responses of the two groups. However, there was a variation from what happened in category I tests. During OTT (which deactivates arterial baroreceptors) the change in HR was more in the RH as compared with the LH.

The response during the Valsalva's Manuever did not differentiate between the two groups. This may have happened because in this test, the resultant bradycardia occurs because of an interaction of both the sympathetic and parasympathetic arms of the ANS (10). The sinus arrhythmia test on the other hand denotes predominantly variation in parasympathetic activity (17). Its results are difficult to

interpret as, one of the parameters ($HR_{max-min}$), was statistically significantly different for the two groups, while the E/I ratio, the other parameter, did not differ.

The cardiovascular status of both LH and RH was physiologically similar prior to beginning of the experimental protocol because their mean heart rate and mean arterial blood pressure values were similar. Therefore the difference observed in the responses of the two groups to the various stress tests would have to be attributed to a variation in the regulatory mechanisms of their autonomic nervous systems engineered by handedness, and associated cortical influences.

Conclusion

- The heart rate and blood pressure responses of LH and RH were different to CPT and HGD which involved the dominant hand.
- The findings reflect a difference in the degree of autonomic control of the cardiovascular system in the two groups. This could be attributed to a difference in handedness and associated cerebral dominance.
- The findings may be relevant to the clinical presentation of left-handed patients with cardiovascular conditions.

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