

## Comparison of Some Physiological Parameters With LBNP and +Gz Stress

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*Lower Body Negative Pressure and +Gz acceleration stress are both potent stressors of the cardiovascular and respiratory systems. Though both stresses exert their effects by causing pooling of blood in the lower extremities and triggering of complex humoral and cardiovascular reflexes, the modality of blood pooling is different. 17 healthy subjects were subjected to graded presyncopal limited LBNP stress. 15 out of the 17 reached the endpoint of presyncope determined either objectively or subjectively. 11 of the 15 were exposed to graded +Gz stress as per the standard protocol then followed at IAM. 8 out of the 11 reached the endpoint of Peripheral Light Loss (PLL). A strong correlation was found between the tolerances to presyncopal limited LBNP and PLL - limited +Gz acceleration stress.*

**Keywords:** *Presyncope, Tolerance Index, PLL*

Lower Body Negative Pressure and +Gz acceleration are both potent stressors of the cardiovascular and respiratory systems. They exert their effects by causing pooling of blood in the lower extremities. While with LBNP, the pooling is due to increased transmural pressure as a result of decreased tissue pressure, that due to +Gz acceleration is because of the increased hydrostatic pressure<sup>1,2</sup>. This pooling results in transudation of fluid to the extravascular compartment, which is more with LBNP than with +Gz. There is thus sequestration of a large part of the circulating blood volume in the lower extremities which sets off complex circulatory and humoral reflexes aimed at maintaining perfusion to the vital organs<sup>1,2</sup>. The sequence of deactivation of the baroreceptors is slightly different with LBNP and +Gz stress. LBNP causes decreased venous return due to pooling, and since it is applied in the supine posture, the low pressure baroreceptors are deactivated first. It is only at higher levels of LBNP (>40 mm Hg) when the Cardiac output decreases significantly that the high pressure baroreceptors are deactivated<sup>3,4</sup>.

However with +Gz, the hydrostatic pressure gradient above the level of the heart decreases causing deactivation of the high pressure baroreceptors<sup>5,6</sup>.

The volume of blood which pools after LBNP application is about 0.9 l at - 40 mmHg and 1.2 l at - 60 mmHg<sup>1,3,7</sup>, while with +4 Gz the volume of blood pooled is about 60 - 100 ml in addition to 500 - 700 ml already pooled during the change of posture from supine to erect<sup>5,6</sup>. There is an increase in Heart Rate (HR), fall in Systolic Blood Pressure (SBP), little or no change in Diastolic Blood Pressure (DBP), fall in Pulse Pressure (PP) and little or no change in the Mean Arterial Pressure (MAP)<sup>1,3,5</sup>. There is an increase in Total Lung Capacity, Functional Residual Capacity, Residual Volume and Forced Vital Capacity. The number of unperfused alveoli increased from 11.4% at rest to 19.4% at - 50 mm Hg LBNP, while with +3 Gz the value was 18%<sup>8,9</sup>. Assessment of orthostatic tolerance is a must in cases of low +Gz tolerance. The assessment of orthostatic tolerance would be of greater value if this tolerance could be correlated to +Gz stress. Earlier attempts to use techniques like CVS response to 70° HUT have not been successful<sup>17</sup>. It is possible that controlled application of LBNP would be more useful as the stress imposed by LBNP is greater than conventional tilting. The present study is therefore aimed at investigating such a possibility.

### Material and Methods

The experimental protocol was divided into two phases. In Phase I, subjects were exposed to a graded presyncopal limited LBNP protocol, and in Phase II they were exposed to graded +Gz

stress limited by the occurrence of Peripheral Light Loss (PLL).

### Phase I

17 healthy adult male subjects were exposed to a graded LBNP protocol. Their mean age was 28.2 yrs and the mean height 168.5 cm. The LBNP box consists of a hemispherical perspex dome with a perspex base plate. There is an entry port on one side through which the subject enters and also where the waist seal is applied. The latter is made of 6 mm Neoprene rubber. Negative pressure was generated with a house hold vacuum pump (Eureka Forbes). The level of negative pressure could be varied by means of valves on the LBNP box and on the vacuum tubing. The maximum negative pressure that could be generated varied from -70 mmHg to -75 mmHg.

The electrodes and the leads for the ECG were first attached. The subject then wore the rubber skirt and entered the box, feet first. The remaining instrumentation for biomedical parameters was done, after which the waist seal was tightened around the waist at the level of the iliac crests with the help of a broad canvas belt.

All the parameters were recorded on a Grass model 7D polygraph. ECG was recorded using the 7P4 preamplifier using standard Lead II. Blood Pressure was recorded using a 7F8 preamplifier. The TR 1010 microphone was placed over the Brachial artery and a standard inflatable BP cuff was tied over it. Extra long tubing was used to connect the cuff to the polygraph. The calibration was done so that a 2cm deflection = 100 mmHg. Respiratory Rate (RR) was recorded using a Grass TCT-IR thermocouple clipped onto a nostril and attached to a 7P1G preamplifier.

The parameters were recorded every 5 min over a 15 min resting period and the average of the readings was taken as the base line value. Following the recording of resting parameters, the subjects were exposed to a graded negative pressure, starting with -30mmHg for 5 min, followed by -40 mmHg for 5 min. Thereafter the

decrements of negative pressure were -5 mmHg every 5 min till the subjects reached their endpoint (Defined later). In case the subjects did not reach the endpoint even after 5 min at maximum negative pressure, the run was carried on till the endpoint was reached. The parameters were recorded continuously at a paper speed of 2.5 mm/sec and every minute at a speed of 25 mm/sec. Post LBNP parameters were recorded at 0,1,2,4 and 6 minutes.

The endpoint of the run was the appearance of presyncope, with symptoms of nausea, light headedness, sweating etc. Objective criteria used were SBP <90 mmHg, fall in SBP of > 15 mm Hg over 2 successive readings, PP < 15 mmHg and a sudden fall in HR of > 20 bpm over two successive readings. A "tolerance index" was calculated as the "Sum of the products of pressure differential from control and the time (in min) at each level of LBNP". This was done in order to cater for the variations of the level of LBNP tolerated and the duration of time for which it was tolerated by different subjects<sup>1</sup>.

### Phase II

11 healthy subjects who had successfully completed Phase I were subjected to +Gz stress in the Human Centrifuge at IAM.

The protocol followed was the same as that which is being used in IAM<sup>10</sup>. The subject entered the gondola and was instrumented for ECG, RR and Superficial Temporal Artery Blood Velocity. The first two parameters were recorded on a Grass Model 7D polygraph and the blood velocity was measured using a Doppler Sonicaid Blood Velocimeter.

The acceleration profile followed was as follows : onset rate 0.5 G/s, followed by a 10 s period at peak G and an offset at 0.2 G/s. The runs were started at 2.5 G peak, and built up at increments of 0.5 G till the subject neared his endpoint of PLL. After this the increments in peak G were reduced to 0.1 to 0.2 G till PLL was reached. The endpoint of the run was appearance of PLL of 56/52 degrees using the GRADEPS

system of determining the PLL, as prevalent in this laboratory.

Statistical analysis of both sets of data was done. In phase I the stabilised readings at each level of LBNP, at maximum level of negative pressure, and at presyncope were used. Mean and SD were calculated and compared to the resting values using a Students test. Similar analysis was done with the data from Phase II also. The correlation coefficient was worked out between the Tolerance Index and tolerance to +Gz (PLL).

## Results

**Phase I** :- 15 out of 17 subjects reached the endpoint of presyncope. The runs had to be aborted prematurely in the case of one subject due to the appearance of Ventricular Premature Beats (VPB), and in another due to excessive subjective discomfort.

Of the subjects who developed presyncope, 11 had both objective as well as subjective criteria, 3 had only subjective criteria and 1 had only objective criteria. The average tolerance index for the group works out to be 1710.24 mmHg.min (LBNP of -65 mm Hg for 4.76 minutes). However the average tolerance index for the 8 subjects completing phase II works out to be 2088.75 mmHg.min. The response of Cardiovascular parameters and Respiratory Rate during LBNP exposure to presyncope and recovery are shown in Fig.1.

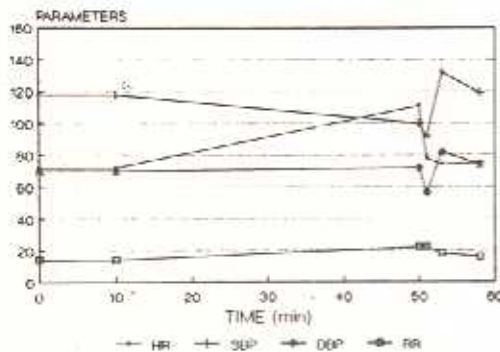


Fig 1 : Cardiovascular and Respiratory Rate Response of 11 subjects at control, during LBNP, at presyncope and during recovery.

**Phase II** :- 8 out of 11 subjects reached their endpoint of PLL. The runs had to be aborted in three cases due to appearance of VPB, and in one case each due to excessive nausea and subjective discomfort.

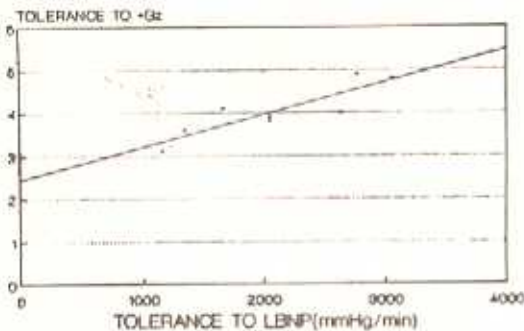
The comparison between the two stresses, that is the results of both Phase I and II in the eight subjects who reached the endpoints both under LBNP and +Gz are given in Tables I and Ia. Fig.2 shows the trend line when the tolerances to both stresses are plotted against each other.

Table I : Comparative Values for Baseline Heart Rate (BHR), Max Heart Rate (MHR) and Increase in Heart Rate (HR) for + Gz and LBNP Experiments. G Values at PLL and Tolerance Index (TI) for LBNP are also Tabulated

S No	+G				LBNP			
	BHR	MHR	HR	G	BHR	MHR	HR	TI
1	90	138	48	3.1	72	96	24	1180
2	84	126	42	3.6	60	90	30	1340
3	84	134	50	4.1	72	110	38	1660
4	84	132	48	3.8	76	96	20	2050
5	90	132	42	3.9	90	138	48	2050
6	84	126	42	4.0	52	102	50	2635
7	90	144	54	4.9	70	114	44	2765
8	90	144	54	4.8	84	120	36	3050
Mean	87	134.5	47.5	4.02	72	108.3	36.3	2088.75
±SD	3.21	7.07	5.10	0.59	12.14	15.7	10.9	685.54

Table Ia

	BHRG - BHR LBNP	MHRG - MHR LBNP	HRG - HR LBNP
MD	15	26	11
p	<0.001	<0.001	<0.01
r	0.62	0.37	-0.17



n = 0.87

Fig 2 : Eye Fit Datum Line for LBNP Tolerance Index and +Gz tolerance in eight subjects.

## Discussion

LBNP decreases the tissue pressure, and hence increases the transmural pressure. This causes dilatation of the capacitance vessels, and pooling of blood in the lower limbs<sup>4,11</sup>. Classically LBNP has been applied in the supine position with the waist seal positioned around the iliac crests<sup>1,2</sup>. We followed this convention in the present study. There are various protocols used for applying LBNP ranging from the "jump" to the stepwise decrement either with an abrupt release or a stepwise release of negative pressure. Since a definitive endpoint was required for this study, presyncope was chosen as it is well defined<sup>4</sup>. There were two factors used in selecting the protocol for this study. Firstly, the presyncope is rarely encountered below -50 mmHg, and the frequency of subjects reaching presyncope increase exponentially after -50 mmHg<sup>2</sup>. Secondly, the cardiovascular variables usually stabilise by 3 to 4 minutes at each level of LBNP<sup>1,2,6,12</sup>. Thus the protocol used increments of suction of 5 mmHg after -40 mmHg, so that an exact level of negative pressure, at which presyncope occurs, could be identified for each subject. The cardiorespiratory parameters were recorded at the 4th and 5th minutes respectively so that only the stabilised readings at each level of negative pressure were recorded.

The results from this study show that the changes in physiological variables are very similar to that reported in the literature. The increase in HR is characteristic and is due to sympathetic discharge following baroreceptor deactivation<sup>2,13,14</sup>. The cardiac output is maintained at lower levels of LBNP (upto -45 mmHg) just by the increase in HR. At higher levels of LBNP the cardiac output falls as tachycardia alone is not sufficient to maintain the cardiac output. The SBP shows a fall which is proportional to the level of LBNP. This fall is due to the decrease in the cardiac output on which SBP is dependent. DBP on the other hand is dependent on peripheral resistance. The peripheral resistance in the forearm vasculature increases (19% to 56% at -40 mmHg) and hence there is not much change in DBP. The response of Mean Arterial Pressure (MAP) is dependent on the DBP response. Thus there is normally very little change in MAP as well. The fall in PP is mainly due to the decrease in SBP<sup>2,13-15</sup>.

At presyncope however the changes are dramatic. There is a sudden bradycardia and hypotension associated with symptoms of presyncope. This has been postulated to occur due to the firing of the ventricular wall tension receptors which are stimulated because the ventricles are contracting against a negligible blood volume. Post LBNP the classical Blood Pressure overshoot is seen as the sequestered blood now reenters the circulation as a bolus<sup>2,4,7,14</sup>.

With +Gz acceleration also there is pooling in the lower limbs, causing a decreased venous return which in turn results in the deactivation of the baroreceptors setting of the reflex changes seen. The classical increase in HR was seen in the subjects exposed to this stress and was similar to the results found by other workers<sup>5,6,10</sup>. The pre-acceleration HR was always greater than the pre-LBNP HR. This is attributed to the sitting posture and also the anticipatory increase in HR which has been found to be proportional to the G level to which the individual is to be subjected<sup>6</sup>.

The use of PLL as an endpoint has been the subject of controversy since it is purely a subjective endpoint, but it is accepted as a reliable indicator of a person's +Gz tolerance<sup>13</sup>. Cessation of blood flow in the superficial temporal artery has been used with success as an objective endpoint. In our study also this was used and seven of the eight subjects had cessation of flow at PLL as reported in literature<sup>10</sup>.

#### LBNP and +Gz

In the past, workers have tried to find a correlation between tolerance to +Gz acceleration and orthostatic tolerance (70° HUT) without success<sup>17</sup>. Conventional (70°) HUT and passive standing exposes man to 1.0 G. Equivalence of -50 mmHg LBNP to 70 deg HUT has been reported<sup>2</sup>. Since the LBNP exposure was to a level greater than -45 to -50 mmHg, the stress imposed was greater. Using HR criteria alone, it has been suggested that -50 mmHg in the sitting posture could be equivalent to +2 Gz. However this conjecture was based on a review of data from various studies<sup>18</sup>. It has also been suggested that it is possible to find an equivalence of LBNP with +Gz<sup>17,18</sup>.

In this study, two well defined endpoints viz presyncope and PLL were used. Furthermore the tolerance index lends itself well to statistical analysis. A very high degree of correlation of 0.87 was found between the tolerances to the two stresses. From Table 1 and Fig 2 it is evident that a subject showing a higher tolerance to LBNP also showed a higher +Gz tolerance. Subjects 3,4 and 5 all had average LBNP and +Gz tolerances. Similarly subject 1 had low LBNP and +Gz tolerances, and subjects 7 and 8 had high tolerances to both stresses. It is thus evident that cardiovascular responses to these two orthostatic stresses are comparable. However there were certain shortcomings in this study : (i) LBNP was applied in the supine position thus eliminating the hydrostatic pressure gradient produced by gravity, (ii) The time taken to reach peak LBNP and the time for peak G were very different and (iii) the number of the subjects used in the study was

small. If LBNP is applied in the sitting posture, the comparisons may be more appropriate. With this there will be a hydrostatic pressure gradient acting from head to foot, however the time course of events to the endpoints with LBNP will always be much more than with +Gz as the onset rates of 0.5 to 1.0 G/s means that the subjects reach the endpoint of PLL earlier.

That such a correlation exists excites the speculation that there may be a role for baroreceptor training<sup>15</sup>. It is known that repeated LBNP increases a person's tolerance to LBNP<sup>19</sup>. Also a long lay off from flying decreases a person's G tolerance. Could LBNP be used to improve a person's G tolerance, and to reestablish a decrement when he has been off flying? These are the possibilities which need to be investigated further.

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