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LOWER-BODY SUCTION AND CARDIOVASCULAR REFLEXES : PHYSIOLOGICAL AND APPLIED CONSIDERATIONS

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Abstract : Lower body subatmospheric pressure (LBSP) can be applied in a graded manner to a supine subject enclosed in a box upto the level of the iliac crest in order to elicit cardiovascular reflexes without a change of posture, and without a gravity induced shift of the central blood volume into the periphery. The procedure effectively produces a controlled, non-haemorrhagic hypovolaemia. The method may be used to differentiate the reflex cardiovascular effects induced by the deactivation of the low-pressure cardiovascular receptors (LBSP < 30mmHg), and those produced by the deactivation of the arterial baroreceptors (LBSP >30 mmHg). The former results in an increase in the limb vascular resistance without a change in heart rate and blood pressure, while the latter produces a tachycardia, an increase in the diastolic BP with a fall in the sytolic BP. The CVP and the cardiac output reduce with increasing suction. Cardiovascular reflex effects of -40 mmHg are similar to those produced by a change of posture from supine to standing. Vaso-vagal syncope appears with increasing frequency when LBSP exceeds -60 mmHg even in the normal subjects. The test is useful in the aeromedical assessement of apparently normal individuals with low orthostatic tolerance, in the evaluation of the effects of physiological and pharmacological interventions on cardiovascular reflexes, and in the evaluation of patients of autonomic neuropathies. LBSP is also a means of inducing safe, well controlled syncope in order to study the genesis of this phenonmenon.

Key Words : baroreceptors central blood volume heart rate postural stress

INTRODUCTION

A change of posture from the supine to the erect moves about 600 ml of blood from the thorax into the lower limbs (1) to form a blood pool there. This lowers the central blood volume (CBV) and the pre-load of the heart, threatening the maintenance of the cardiac output and the blood pressure. Simultaneously this also reduces the degree of stretch on the low-pressure cardiovascular receptors and the arterial baroreceptors (2,3) and subsequently the afferent input to the medullary cardiovascular areas, mainly the nucleus of the tractus solitarius (NTS) (4). The information is then processed by the paramedian reticular nuclei (4), the hypothalamus and the cortex (5) to direct an autonomically mediated efferent output to the heart and the peripheral vasculature to maintain the blood pressure (BP). During certain physiological stresses viz. prolonged standing, exposure to positive accelerations of aviation, and exposure to heat stress which produce more than the usual peripheral blood pooling, the integrity of these cardiovascular reflexes help to maintain the BP and the cardiac output as close to normal as possible. If these reflexes malfunction, a vaso-vagal syncope is likely to occur with an accompanying episodic loss of consciousness: a situation that is unacceptable in certain specialised work environments such as Aviation. In certain clinical disorders viz. Diabetis mellitus with autonomic neuropathy, a patient may present with orthostatic intolerance (5). It may therefore become necessary to evaluate the functioning of cardiovascular reflexes in order to ascertain the fitness of individuals required to undertake special tasks, or for the clinical assessment of patients (6).

Various tests may be employed to investigate this stability viz: quiet standing, exposure to the head-up tilt (HUT) table test, and the application of lower-body subatmospheric (negative) pressure: henceforth referred to as LBSP. Various cvs variables such as the heart rate, BP, cardiac output, forearm vascular resistance and the central venous pressure can be monitored during the test (6,7,8.9).

The physiological basis of LBSP application: The application of suction to the lower parts of the body decreases the tissue pressure in these parts to produce a fairly uniform increase in the transmural pressure of the vasculature particularly so of the capacitance vessels. This also creates a pressure gradient between the unexposed and the exposed parts, resulting in a transfer of blood to the peripheries, and the formation of a peripheral blood pool which is initially rapid. Greater the extent of this pool, more is the blood lost to circulation. It would be physiologically favourable therefore to limit the collection of blood in the periphery.

The role of capacitance vessels in the formation and limitation of this peripheral blood pool has been controversial. Shepherd (10) has opined that there is no sustained increase in the tone of the capacity vessels during LBSP, and hence the veins do not have a significant role to play in limiting the peripheral blood pool. This opinion was supported by the observations made on a patient of idiopathic orthostatic hypotension whose limb capacitance vessels continued to show variations in tone during the normal respiratory cycle. Samueloff et al (11) reported that at -60 mmHg suction there was only a transient (for about 80 sec) increase in the forearm venous pressure, and that this increase was dependent on factors other than sympathetic vasoconstriction because it persisted even after adrenergic blockade. On the other hand Gilbert and Stevens (12) who also exposed their subjects to -60 mmHg LBSP, found a consistent increase in venomotor tone during the stress. They concluded that this venoconstriction played a major part in limiting the pool formation. Substantiating this are the more recent observations of Tripathi et al (13) that reflexes originating in the low-pressure cardiovascular receptors control sympathetically mediated venomotor tone. Therefore the response of the capacitance vessels to LBSP will infact determine the extent of the peripheral pool formed, and this in turn is likely to reflect upon the overall outcome of the stress and the cvs response to this stress.

At high grades of LBSP, filtration of the plasma may occur at the capillary level (14). Musgrave et al (15) have estimated that LBSP imposes upto 95% of the applied pressure on to the subcutaneous tissues, and about 80-90% to the muscles of the lower limbs. On the other hand not more than about 64% of the suction pressure applied to the necks of human subjects was transmitted to the tissues adjacent to the carotid sinus (16). The difference in these observations could be attributable to the variation in the compliance properties of the subcutaneous tissue in the two regions involved. Ludbrook et al (16) measured the intra carotid pressure along with the tissue pressure outside the carotid artery at the time of application of the neck suction in order to derive the extent of the transmitted pressure. As against this, Lundvall and Lanne (17) have reported that a defined reduction of atmospheric pressure around the upper arm led to a similar alteration of vascular transmural pressure. However at this point of time it may difficult to apply these findings for the interpretation of LBSP induced pressure transmission as the suction was applied only to discrete circumscribed anatomical portions of the limbs. These authors also contend that the occasionally recorded low degree of pressure transmission during suction application is attributable to inadequate pressure recording, a possibility which may have to be considered while taking into account Ludbrook et al's data.

The most important advantage of LBSP is that it can be applied in a graded fashion to produce controlled sequestration of the central blood pool in order to differentially deactivate the low-pressure cardiovascular receptors (LBSP from -10 to -30 mmHg), and later the arterial (high pressure) baroreceptors when the suction pressure exceeds -30 mmHg (2,18). As the subject is supine during LBSP, a gravity induced hydrostatic pressure column is not created in the dependent parts of the body. The supine posture does not interfere with the recording of various variables. Also the support to circulation from the anti-gravity muscles during standing is eliminated, while the vestibular stimulation which is known to modify cardiovascular responses during the application of HUT (2) does not occur. Hyatt et al (19) had opined that individuals undergoing the suction test manifested marked day to day variations in their heart rate response, but on further evaluation of the method conceded that this test was indeed more

advantageous as compared with the other tests available for the evaluation of orthostatic tolerance because of the reasons mentioned earlier.

The extent of LBSP to be applied depends upon the experimental protocol. When a deactivation of the low-pressure cardiovascular receptors is desired, suction pressures of -10 to -30 mmHg may be used, while at higher pressure, the arterial baroreceptor induced reflex effects on the cvs can be investigated (2). A controlled, safe vaso-vagal syncope may be precipitated at LBSP of -60 to -80 mmHg (8,20). The rate of suction application can be controlled as desired. Brown et al (21) reached their target level (-70 mmHg) at a rate which gave them 90% of the desired level in 3 seconds. A high rate of suction application may be useful while investigating cardiovascular responses of aircrew.

Suction could be applied at the level of the xiphisternum instead of at the iliac crests (22). However, this produces a more severe disturbance of circulation as the splanchnic and the renal beds are involved. Also the movements of the diaphragm tend to be restricted making inhalation difficult (23). Tolerance to -60 mmHg LBSP applied at the xiphisternum was reached within 1 minute (22). These disadvantages are not there with the more usual level of suction application viz. the iliac crests (24).

The duration of application of LBSP varies. Most cardiovascular reflex adjustments to suction reach a steady state within the first 5 minutes (8), and if incremental suction has to be applied, it is most suitable to do this at 5 min intervals (25, 26). Others have used a duration of 10 min (8), 20 min (27), while Brown et al (21) and Bennett et al (28) have found 1 min applications useful. In evaluating the effects of prolonged bed rest used for simulation of subgravity state, Stevens et al (29) exposed their subjects to LBSP for 8 hours a day, applying -50 mmHg for 4 min followed by -25 mmHg for 2 min. Depending upon the experimental protocol, incremental suction may be applied in a continuous manner, or at the end of each suction level, a few minutes recovery time could be given.

A variety of factors are likely to influence the physiological response to LBSP. These include environmental temperature, invasive instrumentation for monitoring various cardiovascular pressures, pre-exposure physical activity and athletic training (30). In our series of 108 LBSP exposures, at least two episodes of pre-syncope/syncope could have resulted because of a rapid change in the room temperature from 25° C to 28°. These experiments were carried out on Caucasian male volunteers. Perhaps such small change in the environmental temperature may not affect responses of Indian subjects significantly because of their ability to tolerate severe heat stress well (31). Apprehension and pain produced by intra-arterial/intra-venous catheterisation during head-up tilt tests increased the incidence of vaso-vagal syncope in a study by Stevens (32). This is likely to be true also during LBSP exposures.

The technique of applying LBSP : Lower body suction is best applied at the level of the iliac crest (21,30,33) of a subject enclosed in a rigid container box (25, 34). An adjustable foot-board prevents the subject's sliding down the box when the suction is applied (25, 30). This also gives a sensation similar to that of standing without affecting the degree of stimulus being applied (30). A saddle or a harness may also be used to achieve this objective, but the subject generally finds these inconvenient. A simpler version of the method consists of enclosing the subject in a thick pvc sheet and applying suction (30).

In order to achieve adequate sealing around the waist while applying suction the subject wears a plastic skirt which is taped firmly around the waist. The free end of the skirt is then draped around the box opening (Fig. 1). Suction is applied by using a domestic vacuum cleaner with a perforated sleeve mounted on its connection with the box. This sleeve can be rotated to adjust the rate of leak required to maintain the LBSP at the desired level. Recently we have been using a rheostat to control the degree of suction generated by a standard Euroclean 200 room vacuum cleaner. The degree of suction can be monitored using a manometer, or a pressure transducer (25). An increase in the negative pressure recorded means greater suction. Electrical controls must be provided in the circuit to enable the observer to terminate the suction instantantly in case of an emergency (7).

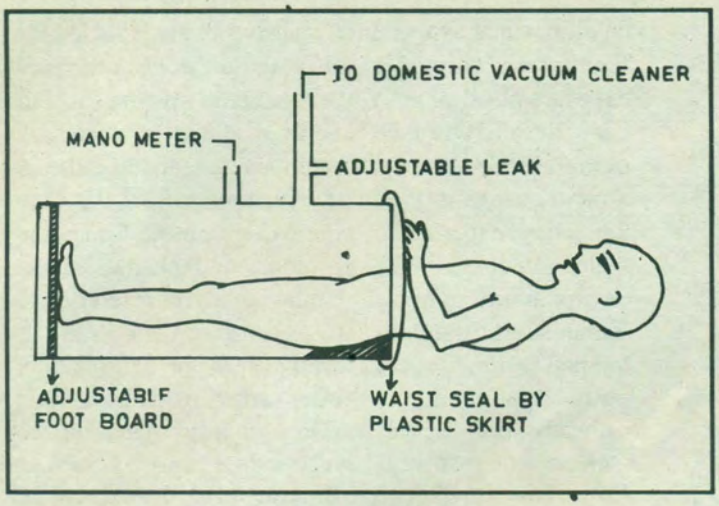

Fig. 1: Line diagram of the LBSP box with a subject positioned in it.

Cardiovascular response to LBSP

Suction pressure less than -30 mmHg : Low level of LBSP (-5 to -30 mmHg) has been used to study cardiovascular and hormonal effects produced by the deactivation of the low-pressure cvs receptors (18). The CVP falls by about 3 to 4 mmHg in this range (8, 18). Bennett et al (28) did not observe a change in the heart rate and the blood pressure of their normal subjects exposed to -25 mmHg for mmHg for 1 min. On the other hand the forearm vascular resistance (FVR) increased markedly even at -5 mmHg with a concomittant decrease in the forearm blood flow (FBF). These effects are caused by a sympathetically mediated vasoconstriction. Goldsmith et al (3) measured plasma NE levels during the application of a low grade of suction of unspecified value. They reported an increase in the level of this catecholamine from 147 to 212 picogm/ml in their experiments in which there was no tachycardia, nor BP changes. An increase in the LBSP produced the tachycardia and BP changes with a further rise of the plasma NE level to 291 picogm/ml. Obviously the arterial baro-receptors had been deactivated.

The FVR and the FBF changes have been reported to reach near their peak levels with LBSP of -5 to -25 mmHg applied for about 2 min (2) 18, (28), and the further increment in the plasma NE levels (291 picogm/ml) as reported by Goldsmith et al probably did not increase the FVR further because the fore-arm vasculature was optimally vasoconstricted by the NE levels found during low-grade LBSP. Our study seems to support the above findings because, by -30 mmHg suction the FVR of our subjects increased by 40.9%, and with increasing suction (-50mmHg), increased further only to 56.4% while the HR and the BP altered incrementally with increasing suction (26). The fore-arm blood flow decreased to 26.5% and this further decreased only to 34% at a suction pressure of 50 mmHg. Tripathi and Nadel (1986) reported a decrease in the FBF by 39% by -20mmHg suction. This decreased further to 56% at -50mmHg LBSP, though at a much slower rate. Compared to the changes we measured in our subjects' FBF values at -50 mmHg, their reported values for the reduction in FBF are higher. Calculating from their data, about 61% of the 6.4 ml/100ml of the FBF of their subjects (measured at 28°C) can be attributed to the skin blood flow, and because the musle vasculature is maximally constricted by -20 mHg LBSP, the reduction in the FBF of their subjects at high grades of LBSP (>30mmHg) can be attributed mainly to the vasoconstriction of the skin vessels during suction. The higher room temperature during their study may have contributed to a degree of dilatation in the skin vessels, giving rise to a relatively high resting FBF value (of 6.4 ml/100ml) as against our resting value of 3.87 ml/100ml, and during suction >30 mmHg, it is the skin BF which reduced, contributing to the higher percentage of change observed by these workers. If Tripathi and Nadel were to repeat their experiments at a lower room temperature, they too may find a less degree of fore-arm vascular bed constriction at high grades of LBSP. Suction of a high order may in fact activate a different vascular bed viz. the splanchnic, and, or the renal.

The findings of Goldsmith et al (3) that high grade of LBSP increases the plasma NE level considerably are in variation with those of Mancia et al (35) who reported that a selective deactivation of the arterial baroreceptors by using neck pressure applied uniformly, failed to increase the plasma NE level though the HR and BP changes expected with the deactivation of these receptors did take place. Perhaps the difference in the methods used to deactivate the receptors accounts for this difference in their observations.

Suction pressure in excess of -30 mmHg : The most impressive cardiovascular effect of high grade of suction is the tachycardia which increases almost linearly with increasing suction. This tachycardia has been shown to be dependent upon the deactivation of the arterial high pressure cardiovascular receptors of which the carotid baroreceptors have been fairly well investigated. In a series of elegant human experiments Abboud et al (36) induced a tachycardia by applying LBSP. When this procedure was combined with neck suction which has been known to activate carotid baroreceptors selectively (37), the tachycardia and the splanchnic vasoconstriction produced by LBSP alone almost disappeared while there was no appreciable change in the FVR and the FBF.

The systolic BP falls by about 10 mmHg at a suction level of 50 mmHg. There is some increase in the diastolic BP which limits any major changes in the mean arterial pressure (MAP). The heart rate (HR) and BP changes produced by -40 mmHg suction and by changing posture from supine to standing are similar (38). Our findings have corroborated this (25). Typical cvs response to graded LBSP in normal Indian subjects (mean age 34 yr) is given in Fig. 2a, while that for young Caucasian males is given in Fig. 2b.

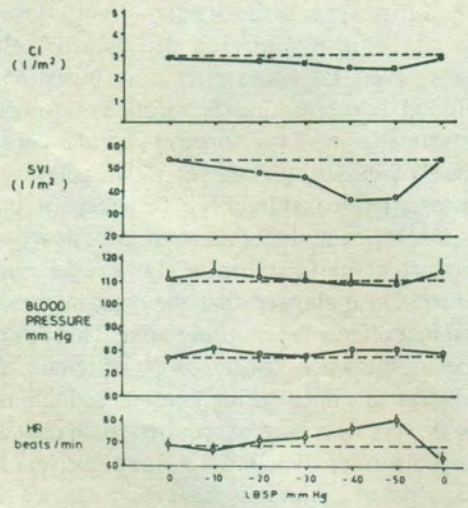

Fig. 2 a: Mean (SEM) values for heart rate (HR; beats per min) and blood pressure (mmHg) of 9 Indian male subjects (mean age 34 yr; range 22-46 yr) exposed to graded LBSP upto -50 mmHg. for 5 min at each suction level. For -10 mmHg, variables were recorded for only 3 subjects. The stroke volume (cardiac output) indices were calculated from the left ventricular ejection time measured from the carotid pulse tracing (Ref. 39) in 2 of the subjects at the end of 5 min of exposure to each LBSP level.

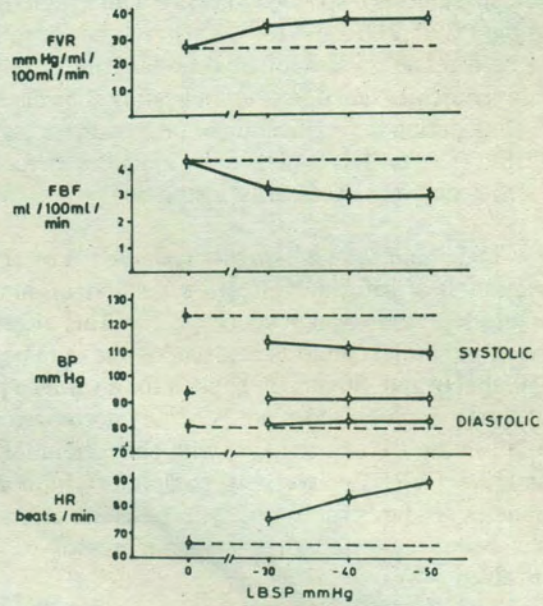

Fig. 2b: Mean (SEM) values for heart rate (HR; beats/min), blood pressure (BP; mmHg), forearm blood flow (FBF; ml/100ml/ min) and forearm vascular resistance (FVR; mmHg/ml/ 100ml/min) of 12 Caucasian males (mean age 22 yr; range 18-36 yr) subjected to graded 5 min exposures to LBSP from -30 to -50 mmHg. The values plotted between the systolic and diastolic BP are those for the mean arterial blood pressure. Data modified from Patrick et al 1987 (26).

The central blood volume (CVB) reduces with increasing suction. This is reflected as a progressive reduction in the stroke volume and the cardiac output which begins at a pressure as low as -10 mmHg. (Fig. 2a). The tachycardia which sets in at higher level of suction is unable to compensate fully for the fall in the SV (8).

Left ventricular function is compromised in dogs exposed to LBSP between -30 and -90 mmHg (14). There is a decrement in the left ventricular filling pressure, the ejection fraction, and the developed force of the left ventricle. However these experiments were carried out in dogs who had developed a high initial heart rate (about 156 beats min) because of the chloralose anaesthesia used. This could have interfered with the ventricular response. These workers also reported that LBSP did not interfere with myocardial contractility as such because there was no appreciable

change in the velocity of circumferential fibre shortening, nor in the dp/dt ratio. On the other hand, human subjects responded to LBSP with a prolongation in the PEP/LVET ratio (39). This ratio has been found to be a reliable index of myocardial contractility and left ventricular function (40). In healthy subjects such a finding during the application of orthostatic stress using LBSP (39) or HUT (41) only indicates myocardial inefficiency, not necessarily insufficiency.

LBSP and the vaso-vagal syncope : Lower body suction is a safe and effective method of inducing controlled vaso-vagal syncope (8, 20). This effect may occur in normal subjects at a suction pressure between -50 mmHg and -80 mmHg applied for a variable period of time. Such episodes are generally accompanied by a fall in the BP and the HR, with changes in the FBR and the FVR. Recovery is complete within a few minutes. The mechanism of syncope is not clear, but it has been suggested that a vasodepressor reflex is involved (42).

The effect of atropine injection on the development of syncope has been studied (20). Atropine injected after the syncope had developed produced an enhanced tachycardia, but did not affect the already developed BP and FBF changes, nor the clinical syndrome. Similar observations were reported by others (43, 44). The increase in the HR would have been expected to alter the systolic BP response to some extent, and modify the clinical syndrome. Atropine injected prior to the application of the LBSP prevented the bradycardia usually associated with syncope, but yet again did not alter the clincal syndrome. An arbitrary "Tolerance Index" measured by the authors was slightly higher when pre-suction atropine was adminstered. (The tolerance Index was the product of the time (min) spent at each LBSP level and the pressure difference at that level, and was expressed as mmHg/min. A higher value meant a better tolerance). An increase in the venous tone and a blockade of the cholinergic vasodilator fibres could have been the contributory factors in giving this marginal benefit, the latter having been implicated in the precipitation of the vaso-vagal syncope in human subjects. The workers did not explain as to why these effects did not help when the drug was adminstered AFTER the syncope had set in . Perhaps the explaination can be found in Epstein et al's support of Sir Thomas Lewis's hypothesis that it is the vasodilatation of the resistance vessels that is the most important factor associated with causation of a vaso-vagal syncope (45).

A recent study has reported that repeated expsures to pre-syncopal levels of LBSP produces some adaptation of the cvs to hypovolaemia (46). The finding may be useful while considering methods of improving the orthostatic tolerance of people performing special tasks viz. fighter pilots who are exposed to high +gz (hyper-orthostatic) stress as a routine. This may be studied by exposing such subjects to pre-syncopal LBSP repeatedly, and then assessing their +gz tolerance on a Human Centrifuge. Also, apparently normal subjects who may have had unexplained vaso-vagal syncopes could be given LBSP training to improve their orthostatic tolerance.

Clinico-physiological use of LBSP : The utility of LBSP in delineating the reflex cardiovascular effects produced by the the low and the high pressure cardiovascular receptors has been discussed. More recently we have used this technique to study the effects of Temazepam, a short acting tranquiliser, on the cardiovascular reflex status (26, 47). Our findings suggested that a single therapeutic (20mg) dose of this tranquiliser did not affect orthostatic tolerance adversely, even 1.5 hours after drug ingestion (when peak blood concentration is reached). However the sympathetically mediated forearm vascular constriction (mediated by the low-pressure cardiovascular receptors) was attenuated considerably. Temazepam probably affects this reflex at the brain stem level as reported by Bittencourt et al (48) for ocular movements. The experiments also showed that the drug does not leave any residual effects the morning after. This information may be useful when considering prescribing suitable tranquilisers to pilots during combat conditions, or to vehicle drivers who may be required to drive at unusual hours. The former hypothesis needs to be investigated in greater detail by human centrifuge studies. Elderly people who may be required to get out of their beds at night may also be prescribed this medicine without undue anxiety about their orthostatic tolerance.

Aschoff and Aschoff (49) have reported a circadian variation in cardiovascular reflex response to orthostatic

stress. During the LBSP series of experiments we have also been able to compare the cvs reflex response of normal subjects to suction at night (10:30 pm) with the same response the following morning (6.30am) (Fig. 3).

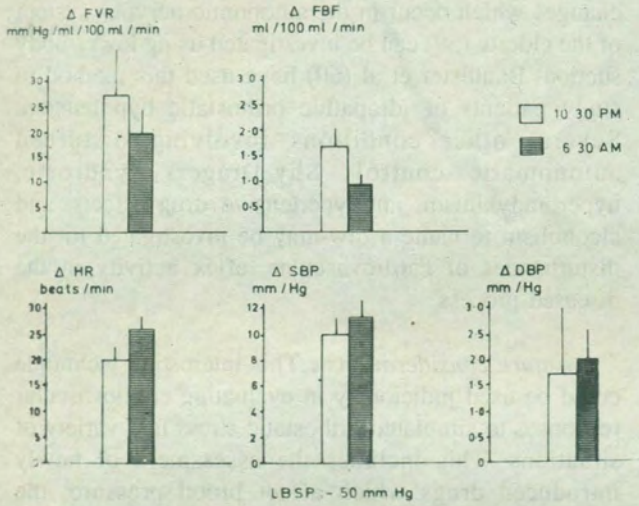

Fig. 3: The change from control values of heart rate (HR; beats/min), systolic blood pressure (SBP; mmHg), diastolic blood pressure (DBP; mmHg), forearm blood flow (FBF; ml/100ml/min) and the forearm vascular resistance (FVR; mmHg/ml/100ml/min) after application of LBSP of -50 mmHg for 5 min at 10:30 pm (blank) and at 06:30 am (strippled) in 12 Caucasian males. Data modified from ref. 26.

The pre-LBSP FBF was less in the morning, with a reciprocally greater FVR, while there were no differences in the resting heart rate and the BP values in the two experimental conditions. The morning vasoconstrictor tone of the forearm vasculature was therefore more as compared with the evening tone. During LBSP however, the tachycardia response was greater in the morning, while the suction induced reduction in the FBF was slightly less as compared with the late night FBF reduction. This could be explained by the fact that the need for the forearm vasculature to constrict was less because of a higher resting vasoconstrictor tone in the morning, while the deactivation of the high pressure baroreceptors resulted in a better homeostatic response. The results could mean a better overall stability of the cvs and its reflex status in the morning hours- an observation which may have applied implications in aviation.

Endurance exercise training produces beneficial cardiovascular adjustments, but orthostatic tolerance of athletes may be reduced because the tachycardia response of such subjects during LBSP is attenuated suggesting an altered responsiveness of the high-pressure cardiovadcular receptors to hypovolaemia (50). Recently, Mack et al (51) have showed that even the responsiveness of the low-pressure cvs receptors to graded non-hypotensive hypovolaemia is attenuated because the degree of forearm vasoconstriction during low-grade LBSP is less in athletes as compared with non-athletes. From the same laboratory it has been reported that the low-pressure cvs receptors are involved in initiating sympathetically mediated veno-constriction while the arterial baroreceptors have little if any role to play in this reflex response excited by the application of graded LBSP (13).

LBSP has been used to compare the pre and post space flight orthostatic responses of astronauts and a cardiovascular deconditioning has been reported after the weightlessness exposure (52). That this deconditioning occurs during the actual space flight was confirmed by demonstrating an exaggerated tachycardia response to graded LBSP upto -50 mmHg with presyncopal episodes in crew members of the Skylab 2 mission while in space (53). In fact since then repeated applications of inflight LBSP has been rcommended as a measure for preventing, at least partially, cvs deconditioning in space.

The effect of hypoxic hypoxia on cardiovascular responsiveness and blood pressure regulation has been studied by Heisted and Wheeler (1970) (54) using LBSP. They demonstrated that forearm vascular constriction in response to hypoxia (12% Oxygen in nitrogen mixture-arterial blood Po_2 41 mmHg) was attenuated, and this effect was the result of poor responsiveness to norepinephrine. In a later study, Heisted et al (55) also showed that exposure to 36 hours of hypoxia continued to show the attenuated vasoconstrictor response during LBSP application. They however did not study subjects who had successfully undergone acclimatization to high altitude hypoxia. Such an investigation is likely to be helpful in tracking the process of acclimatization in sea-level subjects exposed to high a altitude for a prolonged soujourn.

Underfeeding in normal subjects is thought to reduce the rate of catecholamine release into the plasma. Bennett et al (56) investigated this hypothesis by subjecting normal males who had fasted for 12 and 48 hours respectively, to LBSP (-10 to -50 mmHg). They found that the longer period of fasting adversely affected cardiovascular response to graded LBSP. There was impairment of forearm vascular constriction, and an exaggerated tachycardia with a marked fall in the systolic BP. Fasting for 12 hours did not produce any adverse responses. Again these findings are likely to be of significance to people who routinely undergo exposure to severe postural stress.

We have demonstrated that small airway airflows (measured from the effort independent parts of the partial flow-volume curves) increase with increasing suction, but the extent of increase in the these variables produced by changing posture from supine to standing is much greater than that produced by the application of -40 mmHg suction which produces cardiovascular responses similar to that produced by changing posture from supine to standing (25). More recently, an attempt has been made to correlate the physiological responses produced by the application of pre-syncopal levels of LBSP and +Gz (acceleration) stress given to normal male subjects on a human centrifuge (57).

Cardiovascular reactions of patients of diabetic autonomic neuropathy have been examined using this technique (5, 28). The normal function of the low-pressure cardiovascular receptor is unaffected. However, in some diabetic patients the cvs response to the application of high level of suction is impaired because the efferent vasoconstrictor activity is impaired. This observation is supported by the finding in that autopsies of diabetic patients with a history of postural hypotension revealed degenerated greater splanchnic nerves while diabetic patients without postural hypotension did not exhibit this finding (58). Functional changes which occur in the autonomic nervous system of the elderly (59) can be investigated using lower-body suction. Bannister et al (60) have used this method to study patients of idiopathic orthostatic hypotension. Several other conditions involving disturbed autonomatic control- Shy-Dragers syndrome, hyperbradykinism, antihypertensive drug effects, and alcoholism to name a few-may be investigated for the disturbances of cardiovascular reflex activity in the diseased process.

Future considerations : This interesting technique could be used judiciously in evaluating cardiovascular responses to simulated orthostatic stress in a variety of situations. This includes the assessment of newly introduced drugs which affect blood pressure; the cardiovascular reflex adaptation to high altitude exposure as a part of the acclimatisation process; to assess the tolerance of subjects likely to be subjected to high levels of +Gz stress- this could be perhaps better done in subjects in whom LBSP is applied in a sitting posture as against the supine. The method is therefore a most useful tool in various investigations aimed at a better understanding of cardiovascular reflex adjustments.

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