Original Article

Cardiovascular Changes during Weightlessness as Simulated by Dry Flotation

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A variety of ground based studies using analogues of weightlessness have been developed and dry flotation is one such method. A body that floats can be considered to be apparently weightless as floating occurs only when the weight of the body is opposed by an equal force of buoyancy. Cardiovascular parameters were studied on 10 healthy men during 6 hours immersion in a Dry Flotation tank indigeneously developed at the Institute of Aerospace Medicine. It was found that there was a steady fall in resting heart rate (67 to 60 bpm), systolic and mean blood pressure (SBP 117 to 111 and MAP 85 to 75 mm Hg) throughout the period of flotation. Diastolic pressure did not show any significant variation during the exposure (72 to 73 mm Hg). Orthostatic tolerance as assessed by 70° head up tilt showed significant reduction after exposure to weightlessness. This was indicated by significant increase in heart rate rise (17 to 32 bpm) and in orthostatic index (21.3 to 25.9).

Key words : Orthostatic tolerance, water immersion.

Much before the dreams of a manned space programme were fulfilled, it was realised that the condition of weightlessness would be one of the primary environmental concerns of space (1,2). This was borne out by the findings of the various space programmes of both the U.S. and U.S.S.R.[2].

The physiological changes occurring in the weightless environment were seen to be an adaptive response of the body to the new environment of ' Zero g' (2). Similarly, the process of readaptation from the " Zero G" environment of space flight back to the "One G" environment on return to earth is also manifested with adverse and alarming physiological reactions affecting the well being of the astronauts[2-4]. Thus, it is very essential for the spacecrew to adapt to these changing environments with a minimum of physiological disturbances. Though a large number of countermeasures are available to ameliorate the ill effects of spaceflight, spacecrew still manifest signs of psychophysiological maladjustment, and the cardiovascular system is the one which is highly deconditioned in the process[5].

A large volume of data has been gathered over the past three decades on the various cardiovascular changes occurring during spaceflight. However, the mechanisms underlying these changes are still not fully understood. Since a comprehensive study of these underlying mechanisms during an actual spaceflight would be strewn with complexities and inherent constraints, a variety of ground based studies using analogue of weightlessness have been developed[6]. Among the first to be proposed was the Keplerian trajectory model to simulate short periods of weightlessness suggested by Haber et al in 1950[7]. Though this is the best simulation of "Zero G" available, its application is limited due to two shortcomings viz. (a) the period of weightlessness so achieved is of a very short duration of 30 to 40 secs, and (b) it is expensive, requiring a suitably modified transport aircraft to carry out the manoeuvre. This is followed by water immersion (WI) studies, horizontal bed rest, chair rest, and head down tilt[3]. A modification of the water immersion model was described by Shulzenko et al (1976) called Dry Flotation or Dry Immersion[8]. This is similar to the method of water immersion, but for the subject is protected from direct contact with water by a thin water proof plastic sheet. This probably is the ideal analogue of weightlessness as it neither has the problems of maintenance of hygiene and prevention of skin maceration seen in WI, nor the discomfort of head down tilt.

To study the effect of dry immersion (DI) on healthy adult Indian males, a DI facility has been established at IAM,IAF, Bangalore. This facility was designed at the Institute by the authors and

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fabricated locally. The technical details of this system have been reported earlier[9,10].

Material and methods

Ten healthy adult Indian male volunteers (age: 20.2 ± 5.5 yrs, height: 170 ± 6.4 cm, weight: 62.6 ± 10.9 Kg) were exposed to DI for 6 hours. Informed consent was obtained after the details of the study were explained to each one of them.

Heart rate (HR) and blood pressure (BP) responses were studied at pre-exposure to DI, and at hourly intervals during the exposure. The orthostatic tolerance was assessed by standard 70° HUT before and after the exposures. Orthostatic index (OI) for each individual was also calculated[11].

Results

During the immersion, there was a steady fall in heart rate (HR) throughout the exposure (67±7 to 60±5 bpm: ($p<0.01$)). Systolic blood pressure (SBP) fell from of 117±8 fell to 111±8 at the end of exposure ($p<0.01$), but diastolic pressure (DBP) exhibited a small and non significant increase (72±13 to 73±9). Pulse pressure (PP) and mean arterial pressure showed falls of 10 mm Hg and 1 mm Hg respectively (Fig 1 &2). The orthostatic tolerance as assessed by 70° head tilt up (HUT) indicated that the heart rate response after the DI was significantly higher than the pre-immersion values (Table-1). Increase in orthostatic index (21.3 to 25.9) also confirmed the deterioration in orthostatic tolerance after the exposure.

Fig 1 HEART RATE RESPONSE DURING DRY IMMERSION

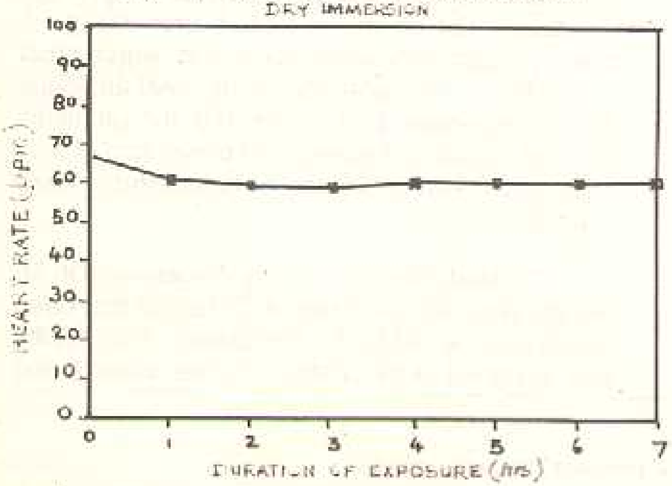

Fig 2 Blood Pressure Response during DRY IMMERSION

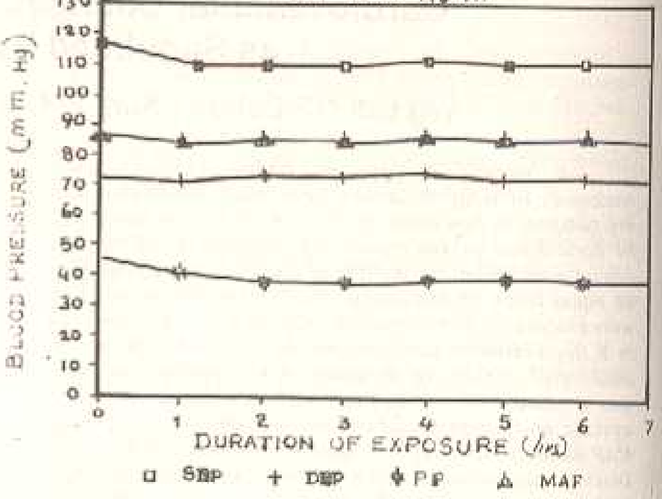

Table-I Heart rate and Blood pressure responses on 70 deg head up tilt (HUT) before and after Dry immersion (mean values)

	Heart Rate (bpm)			Blood Pressure (mmHG)				Ortho-
				Systolic		Diastolic		static
	Supine	HUT	Increase	Supine	HUT	Supine	HUT	index
Pre DI	65	81	16	117	112	75	82	21.3
Post DI	66	98	32	116	112	78	88	25.9

Discussion

During immersion, the hydrostatic pressure gradient generated within the vertical blood columns by gravity is eliminated[3]. This results in an immediate shift of intravascular fluid from the lower part of the body to the thorax and head, followed by a slower shift of interstitial fluid into the blood vessels of the lower limb on account of a decrease in the high hydrostatic pressure difference across the capillaries of the lower limbs[3].

This central shift of fluid causes a sustained increase in central blood volume and central venous pressure of short duration, which in turn activates various compensatory mechanisms to reduce extracellular volume[3,4]. This adaptation is achieved primarily by a diuresis and reduction in plasma volume associated with a decrease in ADH, aldosterone, renin-angiotensin activity and a reduction in sympathetic nervous activity[4]. The changes in cardiovascular function during this process of adaptation include an increase in cardiac output (CO) due to increased venous return, increased end diastolic volume, and stroke volume[4]. However, this increase in CO tends to raise the systolic blood pressure causing a reflex decrease in heart rate . Therefore, the increase in CO may be only marginal[4]. This explains the insignificant change in CO observed during actual spaceflight and many simulation studies[4]. However, the reduction in effective blood volume and changes in vasomotor tone alongwith capillary filtration mechanics causes cardiovascular deconditioning which manifests as reduction in orthostatic tolerance[3].

In this study, a sustained fall in heart rate was noticed throughout the period of immersion as it had been recorded in many other simulation studies[4]. The initial increase in central blood volume and central venous pressure and the consequent stretch of right atrial receptors would reflexly lower the HR through the baroreceptors. Though most of the shifted blood is sequestered in the pulmonary bed and the right side of the heart, a slight increase of cardiac output is seen which would also tend to lower the HR through the baroreptors mechanism. There was no significant change in DBP during 6 hours of immersion; however, the SBP and therefore the PP showed a significant fall.

There was a significant decrease in orthostatic tolerance following immersion. 4 out of the 10 subjects could not complete the 20 min HUT protocol after the DI, and the HUT had to be aborted due to subjective symptoms as well as objective signs of impending syncope. The average increase in HR on HUT following DI was greater than that during pre DI, and the difference in the increase in HR on the two occasions was highly significant ($p<0.01$). The orthostatic index was also found to be higher following DI indicating a loss of orthostasis adaptabilty.

Poor orthostatic tolerance caused by cardiovascular deconditioning has been reported ever since the first water immersion studies were carried out in early 60's. Subsequently, after every space flight, a loss of orthostatic tolerance was noticed in spacecrew in missions which lasted from a few days to a few weeks. Even with all the counter measures available today, viz. onboard exercise, wearing of special clothing during flight and on re-entry, LBNP in flight and ingestion of istonic saline a few hours prior to re-entry, orthostatic intolerance still manifests itself, though of a lesser magnitude than that reported earlier.

Inadequate extracardiac regulation of circulation precipitates orthostatic intolerance following DI. It is very likely that the reduction of circulating blood volume produced by diuresis plays a major role. However, diminution of plasma volume alone cannot fully explain this phenomenon. Enhanced compliance of capacitance vessels could be another factor. Venous compliance was found to be increased during spaceflight as well as in ground simulation studies, and appears to contribute to the reduced orthostatic tolerance. A third factor could be a resetting of the carotid barorecptors thereby effecting a marked reduction in the gain of the carotid baroreflex response during orthostasis, probably as a result of reduced vagal activity. Impaired vasomotor response from an adrenergic stimulated vasodilatation, which would allow greater pooling of blood in the dependent parts on orthostasis would be another factor.

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