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Body Fluid Distribution: Implications For Zero Gravity

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MANNED SPACE flight is now a reality and data is available regarding the physiologic effects of short periods of weightlessness.[1] The results of our initial manned orbital space flights indicate that although certain physiological changes do occur during relatively short exposures to weightlessness, such changes have not been of operational significance and have been of a rather minor nature. This lack of gross physiologic alteration was not unexpected as such pioneer experiments as the rocket studies of J. P. Henry in the late 1940's showed that in sedated monkeys exposure to weightlessness for periods of approximately 2 minutes produced no significant changes in arterial or venous blood pressure or heart rate.[2] However, the minor changes which have been observed in recent flights pose some intriguing questions concerning etiology and indicate some rather fertile areas for future research. It seems reasonable to consider that in a zero gravity environment significant alteration of certain gravity conditioned biologic systems will occur as predicted by Gauer and Haber in 1948[3] and inferred by Graveline, et al, from studies of human deconditioning.[4] The present discussion is an attempt to formulate an approach to one aspect of this large unknown.

RECUMBENCY AND IMMERSION AS ANALOGUES OF WEIGHTLESSNESS

Prolonged weightlessness as will be experienced by the space-flying astronaut cannot be produced on earth and must, for the present, be studied by analogy or indirection. Recumbency and water immersion are presently being reinvestigated as it is felt that with regard to certain physiologic systems these situations can be realistically called analogues of weightlessness. In particular in these situations, the hydrostatic pressure effects of body fluids due to gravity are minimized, in recumbency by reducing the height of the fluid column and in water immersion by the counter pressure of the immersion fluid. A decreased demand for musculoskeletal support to counteract the force of gravity is also characteristic of both situations.

In both recumbency and immersion significant redistribution and mobilization of body fluids occur. The hydrostatic pressure of body fluids due to gravity exerts considerable effect during a change in posture especially on the vascular compartment. Compensatory readjustments are required to maintain optimum balance between

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the circulatory system and other body fluids. On assuming the upright posture, systolic and diastolic blood pressure rise, the systolic somewhat less, the pulse pressure decreases, heart rate increases and the cardiac output falls.[5] In subjects changing from recumbency to standing, Thompson, et al, noted a loss of protein free fluid from the blood due to increased capillary pressure.[6] This amounted to 11 per cent of the total plasma volume. Using the carbon monoxide method of blood volume determination, Waterfield observed a 15 per cent decrease in total plasma volume after 40 minutes of quiet standing.[7] Epstein, et al, also reported a decrease in plasma volume during standing and described, in addition, a fall in renal plasma flow and filtration and in the excretion of water and sodium.[8]

Conversely, in the recumbent position, the hydrostatic column of blood due to gravity is diminished to approximately one-seventh its value erect and compensatory fluid shifts occur. Sjöstrand studied five male subjects and found that an average of 643 ml of blood or approximately 11 per cent of the average blood volume was shifted from the lower extremities to the rest of the body after lying down. Of this blood 78 per cent was taken up by the thorax.[9] The diuresis associated with recumbency has been long known and is discussed in detail in a recent review of salt and water volume receptor mechanisms.[10] Bazett, et al, in 1924 confirmed the then well recognized fact that a dilute urine may be excreted on assuming the recumbent posture.[11] Hulet and Smith state that the recumbent diuresis in hydropenic subjects is principally an osmotic diuresis related to an increased excretion of sodium.[12] Thomas has suggested that recumbency may affect an extracellular fluid volume receptor mechanism which, by decreasing aldosterone secretion by the adrenal, would decrease sodium reabsorption by the renal tubules.[13] Such volume receptor mechanisms have recently been identified for the reflex regulation of aldosterone secretion.[14] Citing evidence for cardiac atrial volume receptor mechanisms, Henry, et al, note that in general all procedures associated with increased filling of the intrathoracic circulation, including recumbency and immersion, are accompanied by an increased rate of urine flow. They present evidence for reflex inhibition of the antidiuretic hormone (ADH) by increased filling of the left atrium with a resulting diuresis.[15] This mechanism is now accepted as the Henry-Gauer reflex. Smith has stated that this mechanism is a reasonable explanation of the free water diuresis of recumbency.[10]

In the water immersion situation the hydrostatic pressure effects of the circulatory system are largely neutralized by ambient water pressure and circulatory compensation for postural change is no longer required. Epstein noted that the diuresis of recumbency tends to persist when assuming the erect posture in water.[8] Gowenlock, et al, report that while sodium excretion decreases considerably standing in air after recumbency, there is no change while standing in water after recumbency. Aldosterone excretion decreases during recumbency and during standing in water but increases while standing in air.[16]

A pronounced diuresis has been observed consistently during immersion of human subjects in water. This was first described in detail by Bazett, et al, in 1924[11] and was noted during water immersion experiments done by Graveline, et al, at the USAF School of Aviation Medicine.[4] In the latter experiments the subject wore a dry type rubber suit of conventional SCUBA design and was immersed with his head out for seven days with the exception of brief daily periods of emersion for electrode change and skin hygiene. Diuresis began soon after immersion, and after six to 12 hours was accompanied by a demanding polydipsia. This phase lasted for about 72 hours at which time hematocrit values of 57 and 58 and clinical evidence of plethora were noted (Fig. 1). The diuresis was associated with a simultaneous increase in urinary nitrogen excretion.

Following this 72-hour period, the diuresis abruptly subsided to near control levels of urine flow, and hematocrit and urinary nitrogen excretion values returned to normal. A diuresis is known to be associated with negative pressure breathing.[15] Initially, this was believed to be the mechanism for the observed immersion diuresis for certainly an element of negative pressure breathing does exist in head-out water immersion.

Further studies were designed to investigate

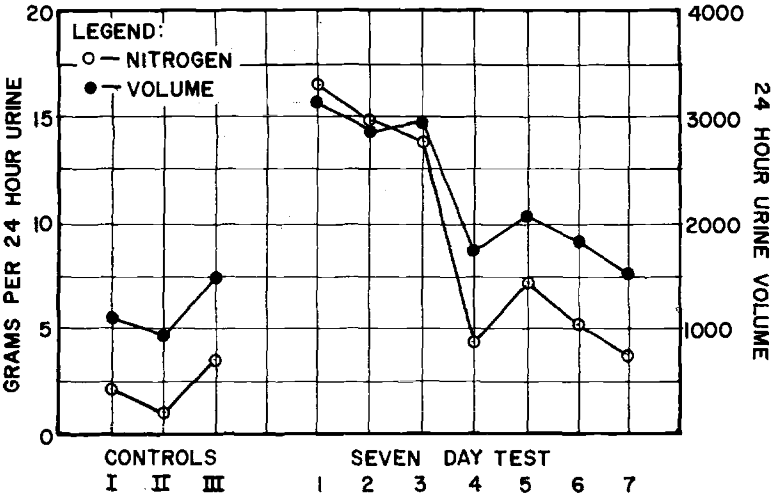

Fig. 1. Twenty-four-hour urine volume and urinary nitrogen excretion during seven days of immersion demonstrating the polyuria which was most marked during the first three days.

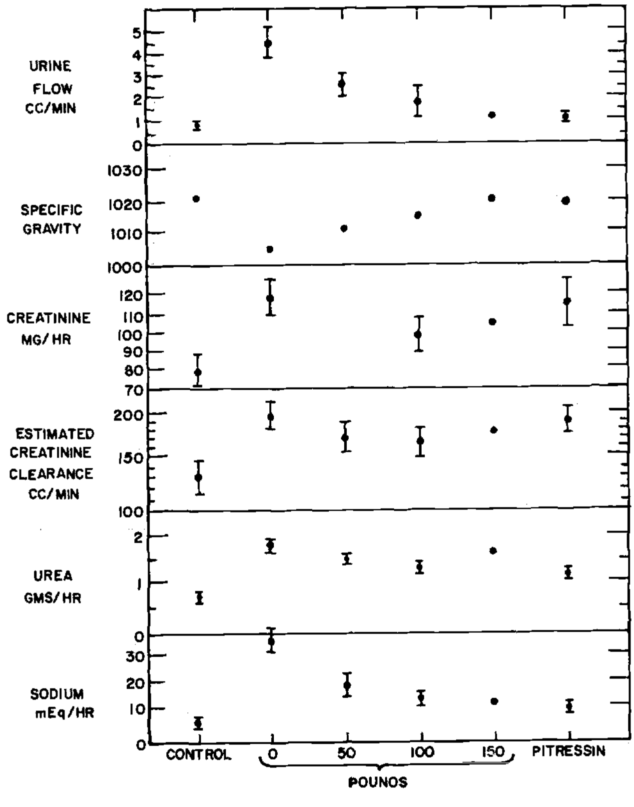

Fig. 2. Effect of weight-bearing and pitressin administration on urine flow, urine solute concentration and estimated creatinine clearance during one hour of water immersion. The mean and range of the values obtained from the ten subjects are shown. Only two subjects were given the 150-pound weighted test.

TABLE I. EFFECT OF WEIGHT-BEARING AND PITRESSIN ON URINE FLOW, URINE SOLUTE AND ESTIMATED CREATININE CLEARANCE DURING ONE-HOUR OF WATER IMMERSION

Subject	Urine Flow cc/min						Urine Specific Gravity						Creatinine mg/hr					
	*C	0	50	100	150	P	C	0	50	100	150	P	C	0	50	100	150	P
1	.68	5.5	5.3	1.3	.68	.92	20	01	02	18	22	20	56	116	111	115	88	179
2	.81	8.5	2.0	7.5	1.6	1.6	24	01	09	01	19	20	78	179	110	135	123	114
3	.71	2.0	1.7	1.9		.97	23	08	11	17		19	146	135	104	128		128
4	.92	4.1	5.3	2.2		1.3	20	05	05	14		23	86	102	105	110		188
5	.45	5.5	4.2	1.7		1.5	18	01	03	15		20	42	105	113	119		79
6	.63	.97	.90	.72		1.0	25	22	21	22		22	90	116	119	124		102
7	.60	2.7	.62	.97		.83	27	11	28	22		19	87	99	51	80		84
8	.81	2.5	1.6	1.7		1.1	21	08	18	12		18	87	96	171	45		83
9	.71	8.2	3.8	.90		1.2	26	01	05	20		23	50	98	46	65		91
10	1.0	4.5	1.7	1.33		1.0	16	07	16	20		20	74	130	101	56		90
Mean	.73	4.4	2.7	2.0	1.2	1.1	22	06	12	16	21	20	80	118	103	98	105	114
SE±	.05	.80	.56	.62		.07							9.2	8.1	11.14	10.4		12.5

Subject	†Creatinine Clearance cc/min						Urea GMS/hr						Sodium mEq/hr					
	C	0	50	100	150	P	C	0	50	100	150	P	C	0	50	100	150	P
1	93	193	185	191	146	299	.55	3.53	1.69	1.38	1.41	.84	3.5	13.2	25.3	12.8	5.8	7.4
2	130	299	184	225	203	189	1.26	1.65	1.80	2.41	1.89	1.24	7.2	72.7	16.8	20.7	15.2	14.5
3	241	226	173	212		213	1.01	1.66	2.59	1.74		1.81	4.3	15.5	11.4	16.2		8.5
4	144	169	174	183		312	.54	1.33	1.53	1.52		.68	9.5	25.1	36.6	16.8		11.9
5	71	174	189	198		132	.47	1.75	1.48	1.43		1.32	3.5	53.3	39.6	14.8		13.9
6	150	194	198	209		170	.81	1.21	1.04	.84		.89	6.5	9.8	9.2	5.9		8.6
7	145	165	85	134		140	1.09	1.89	1.13	.34		.86	4.1	24.2	5.3	8.1		6.8
8	144	161	284	75		138	.90	1.33	1.49	.59		.70	6.3	19.7	14.2	10.0		6.2
9	81	163	77	108		152	.77	1.31	1.35	1.16		1.56	6.2	69.1	9.6	5.6		7.8
10	124	216	168	93		149	.66	1.79	.76	1.18		.73	6.6	42.7	15.5	12.7		6.4
Mean	132	196	172	163	175	190	.81	1.74	1.49	1.26	1.65	1.06	5.8	34.5	18.2	12.4	10.5	9.2
SE±	15.2	13.5	18.4	17.3		20.6	.08	.21	.16	.19		.13	.6	7.4	3.7	1.5		1.0

Subject	Potassium mEq/hr					
	C	0	50	100	150	P
1	1.5	3.6	5.7	6.2	2.4	8.1
2	4.6	8.2	10.2	10.8	8.0	11.5
3	3.7	7.1	4.0	12.3		6.5
4	1.7	4.0	10.2	8.3		3.3
5	1.9	47.0	16.1	7.5		11.1
6	2.6	5.0	4.6	4.9		9.4
7	4.0	9.1	2.9	3.5		5.8
8	3.5	7.2	5.8	4.8		7.5
9	3.1	6.8	6.2	2.8		6.3
10	4.1	10.6	15.8	6.1		6.6
Mean	3.1	10.9	8.2	6.7	5.2	7.6
SE±	.3	4.1	1.5	.9		.8

*C=unimmersed control, 0-150=amount of weight carried, and P=Pitressin.
†Estimated endogenous, creatinine clearance calculated as described in the text.

the mechanisms of the diuretic response to complete immersion with unrestricted activity. A modified partial pressure helmet with a regulator to compensate for ambient water pressure was used.[17] In such experiments the diuresis still occurs. In a series of experiments (Fig. 2), the effects of weight bearing and of the administration of pitressin on the diuresis of water immersion were studied in 10 subjects immersed for one hour.

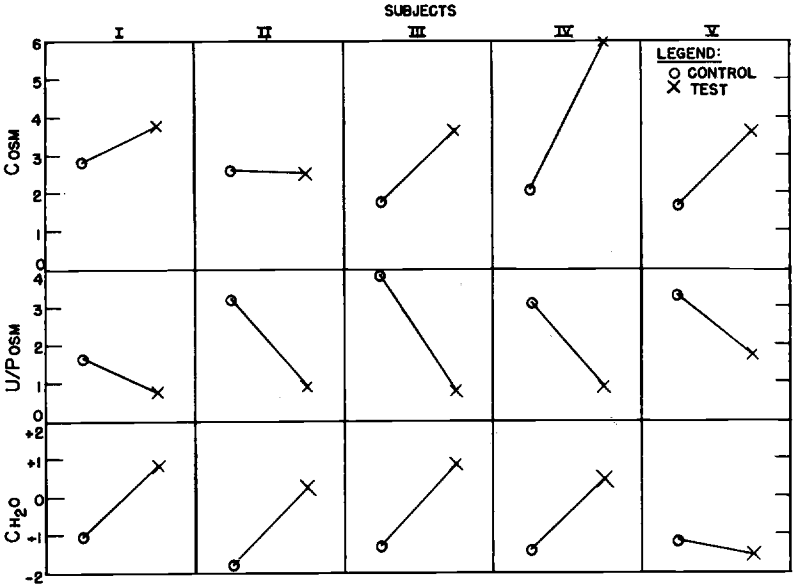

Fig. 3. Osmolar clearance, urine to plasma osmolar ratio and free water clearance comparing the six-hour test results of each subject with his unimmersed control.

In the free-floating (non-weight bearing) immersion situation, a four-fold increase in urine flow is seen with a two-fold increase in urea and a six-fold increase in sodium excretion. Solute concentration as reflected in the urine specific gravity is markedly reduced. The administration of pitressin returns urine flow and solute excretion to the control range. This suggests that the increased urine flow during immersion is the result of the inhibition of ADH release with a resulting water diuresis as ADH (pitressin in this case) has little effect on a solely solute diuresis. ADH similarly inhibits the diuresis of recumbency.[10] The release of ADH by the central nervous system is known to be extremely sensitive to a variety of cortical and sub-cortical influences; indeed, a free water diuresis can be elicited by hypnotic suggestion[18], but the consistency of the diuretic response to water immersion in large numbers of subjects in a variety of experimental conditions mitigates against any such non-specific response.[4,11,17,19] Simple weight-bearing with a camper's back pack during immersion with weight added in 50-pound increments produces a corresponding decrease in urine flow. The mechanism of this observation is obscure. Exercise is known to cause a variable and inconsistent anti-diuresis but the effect of weight-bearing during immersion on cardiovascular and renal function is unknown.

In five subjects the effects of a six-hour period of complete water immersion with compensated respiratory pressures on urine flow, solute excretion, hematocrit, serum solutes and renal function were studied.[17] Osmolar clearances, the urine to plasma osmolar ratio and free water clearances were determined for the five subjects during six-hour non-immersed control periods

TABLE II. EFFECT OF IMMERSION ON URINE FLOW AND SOLUTE EXCRETION*

Subject	†Surface Area m^2	Experiment	Flow ml/min	NA		K		Creatinine		‡Urea		Osmolarity mOsm/L
				mEq/L	gms/6 hr	mEq/L	gms/6 hr	mg %	gms/6 hr	mg %	gms/6 hr	
I	1.85	Control	1.80	86	1.24	37	0.92	78	0.48	1027	6.56	504
		Test	4.62	49	1.85	18	1.19	34	0.56	877	14.54	230
II	1.80	Control	0.83	130	0.89	49	0.55	120	0.36	1331	3.97	956
		Test	2.74	64	1.47	12	0.46	61	0.64	963	9.49	258
III	1.91	Control	0.45	120	0.45	68	0.43	157	0.26	1759	2.90	1118
		Test	4.47	65	2.40	13	0.80	37	0.60	803	12.92	228
IV	1.93	Control	0.69	111	0.63	53	0.51	137	0.34	1333	3.27	930
		Test	6.43	71	3.73	15	1.37	22	0.51	738	17.07	268
V	1.80	Control	0.49	119	0.49	62	0.42	148	0.26	1498	2.38	980
		Test	2.13	102	1.81	27	0.81	71	0.54	1284	9.84	462

*All control values are the average of three samples taken on three days.
†Data not corrected for surface area.
‡Determined as urea nitrogen x 2.14=urea.

TABLE III. EFFECT OF IMMERSION ON HEMOTOCRIT, SERUM SOLUTES, AND RENAL FUNCTION*

Subject	Experiment	Hematocrit		BUN mgm%		Na mEq/L		K mEq/L		Osmolality mOsm/L		C_{urea} ml/min	‡C_{creat} ml/min	C_{H_2O} ml/min	Cosm ml/min	C_{urea} / C_{creat}	Uosm / Posm
		†0800	1400	0800	1400	0800	1400	0800	1400	0800	1400						
I	Control	42	42	12.0	11.9	140	140	4.8	4.4	301	298	72	140	−1.03	2.84	.51	1.68
	Test	47	46	12.0	10.3	148	148	4.0	3.2	287	276	169	157	−0.84	3.78	1.08	0.82
II	Control	45	44	12.9	12.7	148	146	3.7	4.2	299	298	40	100	−1.79	2.62	.40	3.20
	Test	45	48	12.5	11.0	—	—	—	4.3	281	285	105	168	−0.24	2.50	.62	0.92
III	Control	44	43	13.3	14.1	137	143	4.8	4.0	295	294	27	71	−1.29	1.75	.38	3.80
	Test	45	47	10.3	8.0	141	136	4.0	3.8	286	273	182	167	−0.83	3.64	1.09	0.82
IV	Control	46	44	12.4	12.0	145	145	3.5	4.3	302	299	35	94	−1.40	2.10	.37	3.10
	Test	46	49	9.0	7.0	142	—	3.7	3.7	291	287	279	140	−0.47	5.96	1.99	0.94
V	Control	41	40	12.5	12.4	139	138	4.2	4.3	296	295	27	73	−1.15	1.64	.38	3.30
	Test	42	46	12.0	10.0	135	133	4.1	5.3	270	269	116	151	−1.51	3.64	.77	1.72

*All control values are the average of three samples taken on three days.
†0800 is immediately prior to and 1400 immediately following immersion.
‡Endogenous creatinine clearance calculated as described in the text.

and during immersion (Fig. 3 and Tables II and III). There is an increase in osmolar clearance during immersion reflecting increased solute excretion, particularly of urea and sodium. However, all subjects demonstrated a decrease in the urine to plasma osmolar ratio and the free water clearance tended to become positive during immersion. In this study plasma creatinine determinations were not made but a mean value of 1.0 mg/100 ml was used to estimate endogenous creatinine clearance as an indication of glomerular filtration (GFR). A mean increase in the estimated GFR of about 75 per cent occurred during immersion.

Although the diuretic response of immersion appears to be an extension of recumbency diuresis, it remains to be determined whether the naturesis and diuresis of water immersion may be ascribed to volume receptor mechanisms or to altered renal hemodynamics or both. The increased free water clearance during immersion suggests that the diuresis is due in part to ADH suppression as in recumbency. Urinary urea excretion is consistently and persistently elevated during the water immersion diuresis (Fig. 2; Tables I, II and III). Whether the urea originated from muscle tissue under relative disuse conditions or whether it was "flushed out" of renal medullary interstitium by the increased rate of urine flow also remains to be determined.

IMPLICATIONS FOR ZERO GRAVITY

The data obtained from the study of weightless analogues can be extended to describe weightlessness in space only with great caution. In a weightless environment hydrostatic pressure effects are eliminated. In the situation of recumbency in which hydrostatic pressure influences are minimized by the horizontal position, significant redistribution of body fluids occurs. In recumbency blood volume initially increases, and is redistributed cephalad with an increase in the filling of the intrathoracic circulation. Atrial volume receptors are presumably stimulated, reflexly inhibiting the release of ADH, causing a water diuresis. Renal blood flow is augmented and glomerular filtration is increased. The urine excreted in this circumstance is characterized by decreased osmolarity and by decreased concentration but increased output of sodium, potassium and urea. This response appears to be directly related to hydrostatic pressure influences and suggests the real possibility that in a weightless state significant redistribution of body fluids can be expected with a compensatory diuretic response having the above characteristics.

The diuresis seen during recumbency is transient and self-limiting as compensatory mechanisms come into play. However, in all reported studies of the diuresis of immersion, with head out or with compensated respiration, the diuresis is persistent or non-adapting. Graveline et al, in their seven-day study of head-out immersion, noted a persistent 2½ fold increase in urine flow for the first 72 hours of immersion.[4] Further studies with complete immersion and compensated respiratory pressures demonstrated an increased free water clearance throughout a six-hour immersion test.[17] In the case of immersion, the postulated reflex inhibition of ADH apparently persists for some hours unlike the diuresis of recumbency or continuous negative pressure breathing in which urine flow returns to normal after 60 to 90 minutes in spite of continuation of the stimulus.[20] Other short term studies confirm persistence as a characteristic of the water immersion diuresis.[11,19] A result of the persistent diuretic response in these cases has been a tendency to hemoconcentration and dehydration.

A number of mechanisms can be suggested to explain the phenomenon of non-adaptation or persistence of the diuresis of immersion. A relative decrease in osmotically active muscle tissue components due to decreased muscle activity may occur during immersion with loss of muscle tissue water into the extracellular fluid, including the vascular compartment. The effect of continued high levels of urinary nitrogen excretion (Fig. 1) as an osmotic diuretic must also be considered. Another possible contributing factor is contained in the relation of altered proprioceptive input to the central nervous system. On earth, in a one-gravity force field, much of the sensory input which originates in receptors in muscles, tendons and joints can be regarded as gravity dependent and relates to the anti-gravity component of musculoskeletal activity. Proprioceptive input from these receptors has been termed mechanoreceptive in order to differentiate it from that which originates in the labyrinth. In the free-floating condition of weightlessness and of water immersion, because there is no requirement for anti-gravity compensation, this mechanoreceptive feedback is theoretically considerably diminished. This feedback has been suggested as playing a significant part in the maintenance of a variety of reflex systems,[21,22,23] but its effect on specific reflex arcs such as the ADH regulation of blood volume is unknown. Gazenko,[24] in his discussion of the Russian bioastronautics results, noted a number of physiological findings which pointed towards a certain instability of the central apparatus regulating the vegetative functions. Mention was made of the possibility that the observed phenomena were caused by altered function of the receptor instruments of a number of afferent systems, including the vestibular apparatus, under the state of weightlessness.

Body fluid distribution is only one of the biologic systems having known gravity orientation or dependence. Other areas of prime concern are the anti-gravity components of the musculoskeletal and cardiovascular systems. It has been amply demonstrated that serious deconditioning of these systems occurs with prolonged exposures to environments in which the necessity for gravity-compensation has been reduced.[4,22,25,26,27] The metabolic and circulatory changes associated with bed rest or immobilization are well known to physicians and include increased excretion of nitrogen and calcium, loss of muscle mass and weakness and impaired orthostatic tolerance.[25] Similar effects are observed during and following water immersion.[4] Blood volume is also known to decrease progressively for the first two to three weeks of prolonged bed rest in spite of its initial augmentation due to recumbency.[9,25,27] Although much of this decrease is undoubtedly due to decreased demands for hydrostatic pressure compensation, teleologically, it may also reflect the decreased needs for oxygen transport in the hypodynamic bed rest condition.

Only by extending our knowledge to the basic physiological mechanisms involved in gravity-compensation will we be in a position to devise the appropriate protective devices or techniques necessary to each new space operation. Studies are in process to devise the most effective approach to the maintenance of musculoskeletal tone, including consideration of friction devices, electrical motor point stimulation, exercise couches and joint resistive suits. Intermittent venous occlusion with extremity tourniquets during water immersion has been demonstrated as effective in maintaining cardiovascular reflex adaptability as measured by tilt table testing, presumably by simulating hydrostatic pressure effects and thereby "triggering" compensatory cardiovascular reflexes.[28] This is analogous to the use of the oscillating bed during prolonged recumbency to accomplish the same effect.[29] Other techniques including the use of pharmacologic agents must be thoroughly explored. Concerning body fluids, ADH is known to inhibit the diuresis of recumbency[10] and of immersion (Table I). The use of ADH to maintain blood volume in recumbency and immersion and to improve subsequent cardiovascular performance is under investigation in this laboratory. In any event, the first astronauts making prolonged space flights must certainly take careful note of their state of hydration and correct fluid deficits, if they exist, prior to re-entry.

OBSERVATIONS FROM SPACE FLIGHT DATA

A limited amount of information pertinent to the area of body fluid physiology is currently available from our two manned orbital space flights. This data is not accurately defined and must be interpreted with caution, yet certain trends were apparent, particularly from the second manned orbital flight. This astronaut experienced a substantial decrease in body weight during this mission. Regardless of fluid intake during this time, it is evident from the weight change that fluid output exceeded intake by a considerable amount and, by definition, a condition of relative dehydration resulted. Several known factors contributed to this response primary among which was a recurrent suit inlet temperature problem with overheating and sweating but the physiologically intriguing observation is that during this flight a large amount of very dilute urine was excreted.* This inappropriate urinary response is compatible with significant and protracted ADH suppression. A similar trend seems to be evident from the first manned orbital flight but is much less striking.[30]

Although it does appear that the urinary response and implied ADH suppression were inappropriate under the existing conditions, further definition of this phenomenon will require considerably more detailed physiologic studies during future orbital flights.

SUMMARY

In a weightless environment hydrostatic pressure effects are eliminated. In the situation of recumbency in which hydrostatic pressure influences are minimized by the horizontal position, significant redistribution of body fluids occurs. In recumbency blood volume initially increases and is redistributed cephalad with increased intrathoracic filling. Atrial volume receptors are presumably stimulated reflexly inhibiting the release of ADH, causing a water diuresis. Renal blood flow is augmented and glomerular filtration is increased. The urine excreted in this circumstance is characterized by decreased osmolarity and by decreased concentration but increased output of sodium, potassium and urea. This response appears to be directly related to hydrostatic pressure influences and suggests the possibility that in a weighless state significant redistribution of body fluids can be expected with a compensatory diuretic response having the above characteristics.

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*The urine collection bag contained 2360 cc with specific gravity 1.003.

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