Effects of Diuretics on + Gz Tolerance and Biochemical Parameters

Wg Cdr Kuldip Rai* Sqn Ldr S K Adaval** Sqn Ldr M Akhtar*** Sqn Ldr Randhir Singh****
and Shri N K Maruthi Ram*****

Abstract :

Pilots suffering from mild essential hypertension controlled on diuretics alone are permitted to fly in the IAF. A study conducted on four cases of Essential Hypertension and twelve Normotensive subjects, after administration of Polythiazide 1 mg OD for six days is reported. There are significant changes in some of the important biochemical parameters tested in these cases, in that there is an increase in haemoglobin, PCV, bicarbonates, uric acid, fasting and post prandial blood glucose values. The tolerance to +Gz shows a significant reduction of 0.6 G in the PLL value from 4.1 ± 0.7 g to 3.5 ± 0.8 g in the Normotensive subjects. There was a reduction in tolerance to + Gz in the Hypertensives also. All the subjects experienced marked symptoms of headache, weakness and malaise during the trials. The likely hazards of use of diuretics in pilots have been discussed.

Introduction :

Drugs and flying do not go together. The reasons for this rule are not far to seek. A pilot in control of an aircraft is expected to be in peak efficiency – both physically and mentally. In military aircraft the requirements of combat training impose a much higher load on the pilot. In a single crew member aircraft, all the tasks of flying the aircraft are to be carried out by the pilot alone. In view of all these requirements, the physical fitness of a pilot has been emphasised.

A pilot requiring drugs to normalise his physical state is not fully fit[15]. Secondly, all drugs however nontoxic and specific to a system, produce unwanted symptoms and alterations in physiology of man.

Carter et al[3] opined that commonly used antihypertensive drugs *eg:* Thiazides are a bar for fitness to fly. However, the USAF[18], RAF, Canadian Air Force and Indian Air Force have, over the years, adopted a policy which allows pilots with uncomplicated essential Hypertension, controlled with diuretics alone, to continue flying. This policy has been accepted even though there is no data available on the effects of these drugs on the performance of a flier. The diuretics most commonly used are the Hydrochlorothiazide or of the Polythiazide group.

Materials and Method :

The present study was initiated to elucidate the changes in + Gz tolerance and bio-chemical parameters, initially among Normotensives and subsequently in Hypertensives, after administration of diuretics. The study has been completed on 12 Normotensive subjects and in four Hypertensive cases in all respects. To reduce the number of variables, patients with mild Essential Hypertension, controlled on diuretics alone, were selected for the study. Due to lack of suitable cases among aircrew adequate number of trials with hypertensives could not be completed. Non aircrew hypertensive patients

* Classified Specialist in Aviation Medicine, Institute of Aviation Medicine, Indian Air Force, Bangalore - 560 017.
** Classified Specialist in Pathology, Institute of Aviation Medicine, Indian Air Force, Bangalore - 560 017.
*** Classified Specialist in Medicine, Institute of Aviation Medicine, Indian Air Force, Bangalore - 560 017.
**** Graded Specialist in Aviation Medicine, Institute of Aviation Medicine, Indian Air Force, Bangalore - 560 017.
***** Senior Scientific Assistant, Institute of Aviation Medicine, Indian Air Force, Bangalore - 560 017.

were found to be unsuitable for this study on the human centrifuge.

Twelve healthy, fully fit, male, volunteer subjects in age group, 23–40 years, formed the Normotensive group for the study. All the subjects were Medical Officers posted at IAM, who were quite experienced in riding the human centrifuge. All the subjects were fit Med Cat A4G1 and went through a thorough medical examination to exclude any disability.

A detailed clinical history of the subjects, weight and other parameters were recorded for all subjects. Blood pressure was recorded in three positions – lying, sitting and standing.

A bio-chemical assessment was made on each subject. The parameters measured and the techniques followed are given below :

Haemoglobin :	Acid Haematin method by using Spectronic 20 (Baush & Lomb).
PCV :	Wintrobes method.
Serum Na & K :	Flame photometric method by using EEL Flame Photometer.
Plasma Chlorides :	Titration method (Van Slykes).
Plasma Bicarbonates :	–do–
Blood Urea :	Urease : Nesslerisation Method.
Blood Uric Acid :	Brown's Method.
Blood Glucose :	Folin & Wu's Method.
Blood Cholestrol :	Ferric Chloride Method.

The basal +Gz tolerance of the subjects was determined on the Human Centrifuge after ensuring that the subjects were fully rested, had taken their normal meals and were feeling fit to undergo the test.

The subjects were given Tab. Polythiazide (Nephril) 1 mg OD for six days. The subjects were allowed to take potassium supplement in the form of Pot Chloride 1 gm/TDS. The centrifuge test was repeated on the 7th day and the PLL threshold determined. All the other physical and bio-chemical parameters were estimated on the 7th/8th day for comparison with the basal data.

In the hypertensive group, there were [illegible] patients comprising two pilots, one flight [illegible] and one technician. Their ages ranged between [illegible] and 46 years. The hypertension of these [illegible] was controlled by diuretics only. The dosage [illegible] the drug in use was ascertained and the [illegible] condition of each case was assessed. The [illegible] of hypertension was also confirmed after observ[illegible] for a few days. The bio-chemical investigations [illegible] the PLL threshold were determined, while [illegible] subjects was on diuretics. The patients were told [illegible] abstain from the drug for 10 days and the [illegible] were repeated. The difference in various parameters of these four cases has been presented.

The subjects were tested on the IAM Human Centrifuge, which has a 5 metre radius with a standard aircraft seat with a 13° tilt from the vertical (Photo 1). The Gondola is free to align with the resultant acceleration. The acceleration profiles are controlled remotely from the control room. The subjects were monitored on television and were in constant touch on the intercom. They wore normal working clothes without use of anti-G suits. They were instructed to stay relaxed throughout the test run and not to use any voluntary protective methods against +Gz in any of the test runs.

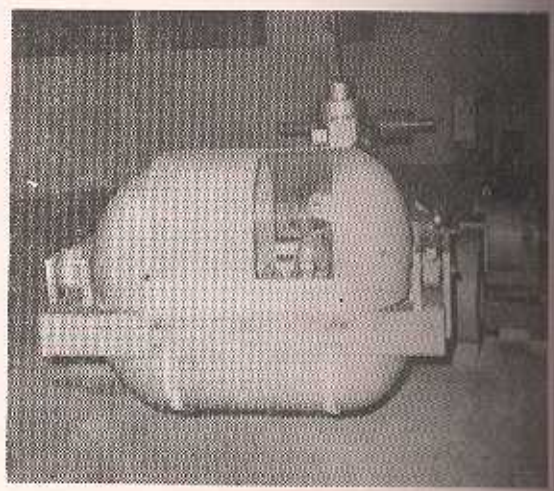

Photo 1
Human centrifuge gondola with a subject

The peripheral lights are mounted 28" apart and at a distance of 30" from the subject at [illegible] head level. These lights subtend an angle of 5° on the subjects eyes. He was asked to concentrate on a central red light and switch off the peripheral

lights as soon as they became visible, by pressing a micro switch on the mock control column. The resting reaction time was obtained by presenting the peripheral lights in a random sequence.

During exposure to +Gz the peripheral lights were switched on at varying intervals. An increase in the subject's reaction time, beyond twice his normal was considered as 'grey out' (PLL). The run was repeated till the 'grey out' level was established. Subjective confirmation of 'grey out' was also obtained. 'Black out' was ruled out by confirmation from the subject in that the central light was visible throughout the test. The Hypertensive and Normotensive subjects were instrumented to record one lead ECG and indirect BP at the Brachial Artery. These parameters were recorded on the multichannel recorder (Photo 2). The ECG was used to study changes during +Gz exposure and to know the heart rate.

Photo 2

Multichannel recorder and the television monitor of the of the Human Centrifuge.

All the subjects were exposed to 0.5G/Sec rate of onset and 0.1g/sec rate of deceleration with peak G maintained for 15–20 secs (Fig. 1). Each case was first tested at a low peak value, which was increased in steps till a firm PLL value was established.

Results:

The results of trials on 12 Normotensive subjects and 4 patients of Essential Hypertension controlled only on diuretics are presented. Table I gives the

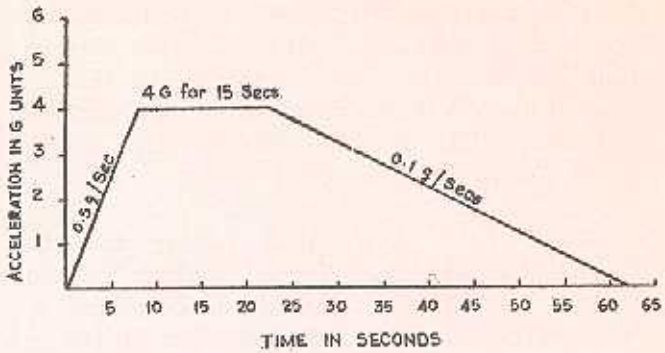

Fig. 1

heart rate and lying BP of the Normotensive group in pre and post diuretic phase. The results show that resting heart rate went up, after diuretic therapy in all subjects except two. The increase in HR has a mean value of 7/mt, which is found to be significant. BP, both systolic and diastolic, registered a significant fall in the post diuretic phase in all cases, more so in the systolic pressure. The sitting and standing BP of these subjects was also recorded, which showed a similar trend.

There was no significant change in HR in hypertension cases and BP showed either a rise or no change after withdrawal of diuretics, in sitting, standing and lying positions. However, the number of cases being very small, statistical significance of these findings could not be established.

The +Gz tolerance, as determined by PLL values for the Normotensive and Hypertensive subjects, under pre and post diuretic phases is given in Table II. In the Normotensive group the mean PLL value in pre-diuretic phase was 4.1 ± 0.7g and in post diuretic phase it was 3.5 ± 0.8g, thus showing a reduction of 0.6g. This has been found to be highly significant by the "t" test. In the Hypertensive group also there was a reduction in tolerance in three cases.

Hb and PCV values determined in all the subjects for pre and post diuretic phases are tabulated in Table III. There is a significant increase in both Hb and PCV in Normotensive group.

Table IV gives the values of serum sodium and potassium, plasma Bicarbonates and Chlorides for the two groups of subjects.

Serum sodium in post diuretic phase has registered an insignificant fall in both the groups. Serum potassium does not show any significant

change in either of the groups. Plasma Bicarbonates show a very significant increase in Normotensives, while the increase is not significant in the IInd group. Chlorides have shown a fall in Normotensives and a slight increase in Hypertensive, which is not significant in either group.

Blood Urea, Uric Acid, fasting and post prandial Glucose values for all subjects are tabulated as Table V (Post prandial blood gulcose was estimated 2 hours after administration of 100 gms of glucose). Uric Acid values are found to be higher in all the Normotensive cases and are statistically significant. The fasting and post prandial glucose levels also showed an increase, which is highly significant in fasting glucose values and significant in post prandial values. Serum cholestrol was estimated but showed no significant change.

Specific Gravity of Urine was determined random samples, voided in the morning subjects reported for biochemical investigation. There is a significant fall in the specific gravity urine in the post diuretic phase.

All Normotensive subjects reported a feeling general weakness, lassitude and mild headache the end of diuretic therapy. These symptoms were worsened by the exposure to +Gz on the centrifuge. Some cases experienced nausea, soon after the run. One subject reported gingivitis which subsided in a few days.

TABLE I

Values of Heart Rate and Blood pressure (Lying Position) for Normotensive Subjects in Pre and Post Diuretic Phase

$n = 12$

Sl. No.	Pulse Rate Pre	Pulse Rate Post	Pulse Rate Diff	BP lying systolic mm of Hg Pre	BP lying systolic mm of Hg Post	BP lying systolic mm of Hg Change	BP lying Diastolic mm of Hg Pre	BP lying Diastolic mm of Hg Post	BP lying Diastolic mm of Hg Chng
1.	60	72	12	144	110	−34	76	74	−2
2.	84	92	8	126	120	− 6	76	70	−6
3.	82	106	24	110	104	− 6	70	68	−2
4.	96	92	−4	110	106	− 4	84	80	−4
5.	80	86	6	119	110	− 9	94	87	−7
6.	78	76	−2	122	118	− 4	72	70	−2
7.	76	82	6	120	118	− 2	82	70	−12
8.	90	96	6	130	124	− 6	82	80	−2
9.	80	84	4	140	130	−10	82	78	−4
10.	72	84	12	130	126	− 4	86	82	−4
11.	76	84	8	116	110	− 6	74	70	−4
12.	72	76	4	130	110	−20	90	72	−18
Mean	78.8	85.8	7	124.7	115.5	−9.2	80.7	75.1	−5.6
± Sd	± 9.1	± 9.5		± 10.7	± 8.4		± 7.3	± 6.1	
t			3.39			3.51			1.1
Sig			**			**			**

TABLE II

Tolerance of both groups (normotensive and hypertensives) ie, pre and post diuretic phases

	PLL Value		
	Pre	Post	Change
NORMOTENSIVES (n = 12)			
	3.5	3.1	− .4
	5.8	5.2	− .6
	3.8	3.1	− .7
	3.5	2.5	−1.0
	4.2	2.8	−1.4
	4.0	3.0	−1.0
	5.3	4.6	− .7
	4.6	4.5	− .1
	3.9	3.5	− .4
	3.7	3.5	− .2
	3.5	3.1	− .4
	3.5	2.8	− .7
Mean	4.1	3.5	
±Sd	±0.7	±0.8	− .6
t			5.89
Sig			***
HYPERTENSIVE (n = 4)			
	3.8	3.5	−0.3
	4.0	4.7	0.7
	3.8	3.7	−0.1
	6.0	5.8	−0.2
Mean	4.4	4.4	0
±Sd	±1.1	±1.1	
t			0
Sig			Ns

TABLE III

Haemoglobin and PCV values in both groups in pre and post diuretic phases

Sl. No.	Hb gms %			PCV %		
	Pre	Post	Change	Pre	Post	Change
NORMOTENSIVES (n = 12)						
1.	15.5	15.5	0	43	43.5	.5
2.	16.5	17.0	.5	49	49.5	.5
3.	17.2	17.0	−.2	51	48	− 3
4.	16.0	16.6	.6	50.5	53	2.5
5.	15.5	16.0	.5	48	54	6
6.	15.0	15.5	.5	51	56	5
7.	15.5	16.0	.5	46	48	2
8.	17.0	17.6	.6	51	56	5
9.	12.7	13.0	.3	43	44	1
10.	16.6	16.5	−.1	48	47	− 1
11.	15.3	16.0	.7	46	49	3
12.	16.0	16.5	.5	48	49	1
Mean	15.7	16.1		47.9	49.7	
±Sd	±1.2	±1.1	.4	±2.9	±4.2	1.87
t			4.25			2.47
Sig			**			*
HYPERTENSIVE (n = 4)						
1.	15.5	15.5	0	52	55	3
2.	15.0	15.5	0.5	51	52	1
3.	15.0	14.8	−0.2	44	46	2
4.	14.5	15.1	0.6	50	52	2
Mean	15.0	15.2	0.2	49.2	51.2	2.0
±Sd	±0.4	±0.3		±3.6	±3.8	
t			1.08			4.90
Sig			Ns			*

TABLE IV

Values of Serum Sodium, Potassium, Plasma Bicarbonates and Chlorides in Both Groups in Pre and Post Diuretic Phases

Sl. No.	Na^+ mEq/Ltr			K^+ mEq/Ltr			HCO_3^- mEq/Ltr			Cl^- mEq/Ltr		
	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
					NORMOTENSIVE (n = 12)							
1.	141	127	- 14	4.1	4.1	0	26	26	0	96	90	- 6
2.	143	145	2	4.3	5.0	0.7	20	24	4	99	96	- 3
3.	148	150	2	4.4	5.2	0.8	21	23	2	97	94	- 3
4.	133	155	12	4.1	3.6	- 0.5	23.2	24	.8	102	100	- 2
5.	142	144	2	4.3	4.2	- 0.1	21.8	22	.2	98.2	98	- .2
6.	131	135	4	3.6	4.6	1.0	23.4	24	.6	94	94	0
7.	131	137	6	4.0	5.0	1.0	23.2	24.4	1.2	96	95	- 1
8.	140	124	- 16	4.7	3.5	- 1.2	25	26	1.0	99	98	- 1
9.	145	134	- 11	4.6	5.3	0.7	23	25	2	98	96	- 2
10.	136	125	- 11	4.7	6.0	1.3	22	28	6	98	101	3
11.	139	143	4	5.0	5.0	0	24	25.2	1.2	96	104	8
12.	143	131	- 12	4.2	4.0	- 0.2	20	24	4	106	102	- 4
Mean	139.3	136.7		4.3	4.6	.29	22.7	24.6		98.3	97.3	
±Sd	± 5.5	8.7	- 2.67	±0.4	±0.8	±0.21	± 1.8	1.6	1.92	± 3.2	± 4.0	0.93
t			0.98			1.36			3.63			0.89
Sig			Ns			Ns			**			Ns
					HYPERTENSIVES (n = 4)							
1.	148	140	- 3	4.1	4.0	- 0.1	22	24	2	96.0	98.0	2.0
2.	141	144	13	4.7	4.6	- 0.1	20	26	6	96.3	99.0	2.7
3.	145	135	- 10	4.6	5.0	0.4	25	25	0	90.4	99.9	8.6
4.	155	130	- 25	4.5	4.3	- 0.2	25	24	- 1	99.6	99.6	0
Mean	146.0	139.7	- 6.3	4.5	4.5	0	23.0	24.8	1.8	95.6	98.3	3.3
±Sd	± 6.2	± 10.3		±0.3	±0.4		± 2.4	± 1.0		± 3.8	± 0.7	
t			0.80			0			1.16			1.78
Sig			Ns			Ns			Ns			Ns

TABLE V

Values of Blood Urea, Uric Acid, Fasting Sugar and Post Prandial Sugar

Mean	146.0	139.7	−6.3	4.5	4.5	0	29.0	24.6	1.6	95.6	98.9	3.3
±Sd	± 6.2	± 10 3		±0.3	±0.4		± 2.4	± 1.0		± 3.8	± 0.7	
t			0.80			0			1.16			1.78
Sig			Ns			Ns			Ns			Ns

TABLE V

Values of Blood Urea, Uric Acid, Fasting Sugar and Post Prandial Sugar in both Groups in Pre and Post Diuretic Phases

Sl. No.	Urea mgm%			Uric acid mgm%			Fasting Glucose mgm%			PP Glucose mgm%		
	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
						NORMOTENSIVES (n = 12)						
1.	22	20	−2	3.2	3.3	.1	80	—	—	93	—	—
2.	27	26	−1	2.7	3.0	.3	68	76	8	72	92	20
3.	24	25	1	4.0	4.0	0	72	76	4	76	72	−4
4.	32	32	0	2.3	3.6	1.3	74	80	6	80	88	8
5.	31	24	−7	2.6	3.7	1.1	84	102	18	108	126	18
6.	24	25	1	3.3	3.0	−.3	72	92	20	78	104	26
7.	26	25	−1	3.8	3.6	−.2	80	92	12	84	104	20
8.	28	26	−2	3.2	3.0	−.2	72	80	8	84	85	1
9.	23	25	2	2.8	3.0	.2	78	75	−3	71	75	4
10.	24	32	8	3.0	4.0	1.0	72	76	4	76	78	2
11.	27	28	1	3.4	3.6	.2	76	—	—	80	74	−6
12.	27	32	5	2.5	3.5	1.0	84	96	12	80	107	27
Mean	26.2	26.7		3.1	3.6		75.6	84.5		75.6	80.8	91.4
±Sd	±3.1	±3.7	0.4	±.5	±.5	.38	±5.6	±10.0	8.9	±5.6	±9.9	±17.1
Sig			Ns						**			*
						HYPERTENSIVES (n = 4)						
1.	27	22	−5	4.4	3.4	−1.0	80	94	14	94	107	13
2.	33	26	−7	4.3	4.0	−0.3	80	80	0	94	100	6
3.	25	25	0	3.0	3.2	0.2	65	75	10	68	85	17
4.	26	22	−4	3.0	3.0	0	80	92	12	86	94	8
Mean	27.7	23.7	−4.0	3.7	3.4	−0.3	76.2	85.2	9.0	85.5	96.5	11.0
±Sd	±3.6	±2.1		±0.8	±3.4		±7.5	±9.2		±12.3	±9.3	
Sig			Ns			Ns			Ns			*

Discussion :

The diuretic and antihypertensive effects of thiazide drugs are extensively reported.[9, 11, 20, 21] The increased rejection of filtered sodium and chloride appears to take place in both the proximal and distal portions of the tubule and the ascending limb of Henle's loop.[11 & 21] The anti-hypertensive action which persists with the prolonged administration of the thiazide drugs is due to a decrease in peripheral resitance.[9 & 21] It does not appear to be secondary to their natriuretic action. The exact mechanisms producing these diuretic and anti-hypertensive effects have not been fully identified.

Certain side-effects commonly occur with the administration of thiazide drugs. The kaliuresis appear to be secondary to the natriuresis and with some of the thiazide drugs, to their carbonic anhydrase inhibitory action.[9] Metabolic alkalosis is actually a more consistent finding than is hypokalemia, and the latter in part may be due to the former.[21] Interference with carbohydrate metabolism is brought about both by a reduction in circulating insulin and by a direct inhibitory effect on glucose utilization by tissues.[9, 16, 17] These side-effects occasionally may be troublesome clinically.

Diuretic therapy causes a reduction in plasma volume, at least transiently which is confirmed by the rise in haematocrit and serum protein concentrations. It also causes a reduction in peripheral resistance which lowers BP. Any reduction in plasma volume and BP below normal values, even in hypertensives, could be cause for reduced tolerance to +Gz. Postural hypotension if present is a serious condition and is not acceptable in a pilot.

There was a significant reduction in +Gz tolerance in all the subjects (except in one hypertensive patient). The mean PLI. value fell from 4.1 ± 0.7g to 3.5 ± 0.8 G in the Normotensive group, showing a change of 0.6 g (15%), which is highly significant. In the hypertensive group also there is reduction of 0.22 g, which could not be statistically confirmed due to the small number of cases. Pfaff and New-berry[18] in their trial with 6 Normorensive subjects on Hydrocholorothiazide 50 mgm BD, observed a reduction in tolerance to +Gz from 3.9 to 3.1 G after 2 weeks and to 3.0 G after 4 weeks of therapy. The results of our study are in conformity with these workers.

The observed increase in resting heart rate in all Normotensive subjects after diuretic administration was significant, and can be attributed to hypovolaemia.[9, 12, 20, 21] The systolic and diastolic BP in sitting, standing and lying positions, showed significant fall after diuretics. Weller and Miller attribute the hypotensive action of diuretics to decrease in peripheral resistance, reduction in plasma volume and extra cellular fluid volume, alteration in catecholamine metabolism and change in carbohydrate metabolism. Nickelson[9] enumerates the above factors and in addition attributes the fall in BP to decrease in total exchangeable sodium and potassium especially in the early phase of thiazide therapy. As our cases were exposed to diuretics for only 6 days, the most important mechanisms involved appear to be sodium and water depletion.

The increase in Haematocrit values are well documented[9, 11, 12, 21] especially in early phase of diuretic therapy due to fluid loss. The fall in serum sodium level is due to the expected action of Polythiazide on the proximal and distal renal tubule, by reducing the sodium reabsorption [10, 11, 12]. Potassium is also lost alongwith the sodium and normally shows a fall.[9, 11, 21] In this series it has not shown a decrease, probably due to the potassium supplements freely taken by all the subjects. However, potassium depletion may exist in the presence of a normal plasma concentration (most of the K^+ being intracellular) and this fact, renders accurate assessment of K^+ balance difficult.[21]

The marginal decrease in plasma chloride found in these cases is due to reduced reabsorption of chloride (fixed anion) which is documented.[9] It is thus accompanied by hypochloraemic alkalosis.[9] Black[1] has opined that chlorothiazide evokes the urinary excretion of chlorides rather than Bicarbonate as the major anion accompanying Na & K. The significant increase in plasma bicarbonate observed in this study is consistent with the current literature.[1, 9, 11]

In this study, a significant increase in blood uric acid levels has been observed. Mudge[9] has reported that the excretion of uric acid in man is decreased by thiazides, though the mechanism is not understood. This has also been reported by Turner et al[20] and Talso and Remenchik.[19]

There is a significant increase in fasting and 2 hr blood glucose values in post diuretic phase in this series. Hyperglycaemic effect of thiazides is well recorded and appears to be of multifactorial etiology.[4, 16, 17, 21] Chazan et al[1] attribute it to inhibition of release of insulin and blockage of peripheral glucose utilisation. Weller et al[21] have also opined that thiazides reduce the level of circulating plasma insulin like activity and also the activity of insulin dependent enzymes *eg:* liver glucokinase and dihydroxyacetonekinase.

The fall in urine specific gravity seen in our cases is expected due to the diuretics.

It is well known that circulation is more profoundly affected by positive G than any other system.[2, 5, 7, 8, 15, 22] The progression of symptoms from minimal visual impairment to "Grey out," "black out" and unconciousness are explained by the progressive decrease of blood flow to the upper part of the body. The blood flow above the heart increases due to the following factors:

(*i*) Fall of BP due to hydrostatic pressure effects.
(*ii*) Reduction in Venous return and peripheral pooling.
(*iii*) Reduction in cardiac output.
(*iv*) Reduction in output pressure of the heart.
(*v*) Loss of fluid to the extravascular compartment.

The compensatory mechanisms set in and attempt to correct the above and show their effect by tachycardia and peripheral vasoconstriction.[8]

With the use of diuretics in Noromotensives or Hypertensives there is a loss of fluid in the initial stages, thus reducing the circulating blood volume. This reduces BP and cardiac output. Later on, the blood volume may get restored to near normal values but the BP does not return to normal, because of the direct action of diuretics on the peripheral resistance. Thus the effects of diuretics is to lower BP by lowering cardiac output and by reducing peripheral resistance.

In such a state if +Gz stress is applied to the body, which is an exaggerated Orthostatic stress, the response of the body cannot be adequate. The head level BP will fall to low values much earlier than in a normal man. More over the reflex compensatory rise of BP which normally takes place, will not be seen in such cases. The poor or delayed compensatory rise of BP, will manifest itself in a lowered tolerance to +Gz, as seen in this series. This is also confirmed by Pfaff and Newberry.[18]

In Essential Hypertension, it has been reported that incidence of Orthostatic Hypotension is more common in comparison to normals, more so when the peripheral pooling has taken place. Fotino et al[6] report that 11 out of 33 hypertensives showed evidence of Orthostatic hypotension on standing up with occlusion of venous return, in comparison to none out of 23 Normotensives. They opine that this may be due to a functional defect at the adrenergic neuro effector junctions of the arterioles in hypertensives. It is also well known that the cardiac reserve and the capacity of the cardiovascular system to produce reflex increase in cardiac output and BP are limited among hypertensives. Thus such cases of Hypertension are a poor risk on exposure to +Gz, more so when diuretics have been instituted to reduce the BP.

Loss of Na & K, has an adverse effect on the arteriolar tone. Maintenance of this tone helps in reflex responses to fall in BP and upkeep of peripheral resistance. In a patient with loss of Na & K, there is a definite lowering of CVS response to +Gz.

The occurrence of subjective symptoms of malaise, weakness and headache, in a large majority of the subjects, indicates the general feelings of being unwell. Such symptoms are exaggerated if potassium supplements are not taken by the patient. In this series, there has been no significant fall in serum potassium since the subjects were taking supplemental potassium chloride. In hypertensive occurrence of such symptoms will not be conducive to a feeling of well being which is very necessary for a pilot to undertake effective part in combat flying.

The reduction in tolerance to +Gz seen in Normotensives is 0.6 g at "Grey out" levels, which itself is sufficiently large to be significant, but this trend does not augur well for a pilot flying at much

higher +Gz values during combat. The reduction in tolerance is likely to be much higher at these high +Gz values, causing "Black out" or unconsciousness earlier than desirable.

Conclusions :

Administration of diuretics in Normotensive and Hypertensive subjects, produces reduction in BP. Significant changes in biochemical parameters are seen with diuretic therapy. There is an increase in Hb, PCV, Plasma Biocarbonates and Uric acid values. The carboydrate metabolism is also affected by diuretic therapy.

There is a significant reduction in tolerance to +Gz in normotensive subjects after administration of the diuretic. There is reduction in tolerance to +Gz in Hypertensive cases also.

There are symptoms of general discomfort with diuretic therapy, which reduce the patients' sense of well being.

The reduction in +Gz tolerance in a fighter pilot can have disastrous consequences in operational flying. It is, therefore, recommended that pilots suffering from Essential Hypertension, if requiring diuretic therapy, be evaluated very thoroughly before being permitted even restricted flying. Tests on simulators (Centrifuge and Psychomotor tests) be carried out before reflighting such cases especially in cases of fighter pilots.

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