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Research Article

PULMONARY AND CIRCULATORY EFFECTS OF ACUTE PULMONARY VASCULAR ENGORGEMENT IN NORMAL SUBJECTS [1]

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Central and pulmonary vascular engorgement is a characteristic feature of congestive heart failure. It has been suggested many times that this vascular engorgement might be responsible for certain phenomena associated with congestive failure, such as stiffening of the lungs, dyspnea, and orthopnea. However, the simultaneous occurrence of pulmonary edema and other changes has made it difficult to single out the effects of simple vascular engorgement.

The present study was undertaken to investigate the pulmonary and circulatory effects of acute, reversible central and pulmonary vascular engorgement in normal man.

Central and pulmonary vascular engorgement was produced by rapid application of pressure over the surface of the body. Two methods were used to accomplish this: inflation of an aviator's "G suit," and submersion in water while breathing against atmospheric pressure.

The results indicate that vascular engorgement of the degree found in congestive heart failure can markedly stiffen the lungs. In addition, it was found that the production of acute central and pulmonary vascular engorgement in normal subjects by the present experimental means is quickly followed by changes which reduce the engorgement and which may represent an adaptive circulatory response. These changes could be modified by pre-treatment with drugs which alter vascular tone or by applying a painful stimulus.

METHODS

Subjects. All subjects studied in G suit experiments were male students 20 to 30 years of age. Three of the subjects studied during submersion were females, in the same age group. The subjects were indoctrinated by several dummy runs before measurements were made to minimize the effects of anxiety.

G Suit. The G suit [3] was a single chamber, balloon type garment which covered the feet, legs, and lower abdomen. The suit could be inflated to the desired pressure within 5 seconds by a standard Air Force G-valve. Suit pressure of 2 PSI (103 mm. Hg) was customarily used. The subjects were studied in the seated position before, during and after a 2-minute period of suit inflation.

Submersion. Seated subjects were studied before, during and after submersion in water to the neck and with the head just below the surface. At this depth, the mean pressure at hip level during total submersion was approximately 60 mm. Hg or 1.2 PSI.

Ventilatory measurements. Ventilation was measured with a wire screen flowmeter using a Statham differential strain gage. The amplified flow signal was either recorded directly or electrically integrated [4] to record respiratory volume change. During the G suit experiment, subjects breathed through a mouth piece almost directly into the flowmeter. Before and during submersion the mouth piece led to a 2.5 foot length of 1 inch I.D. corrugated tubing which in turn was attached to the flowmeter. Although this provided an undesirably large dead space, the same arrangement was used during both control and submersion studies, and the respiratory rates remained at all times within the usual normal limits. Tidal volumes with this arrangement were greater than normal and amounted to about 1000 ml. during both control and submersion periods.

Esophageal pressure was measured using an esophageal balloon as described by Mead and Whittenberger (1). The difference between esophageal pressure and mouth pressure was measured by means of a differential strain gage. This pressure, the "transpulmonary pressure," was recorded simultaneously with the flow and volume measurements.

From the recordings of flow, volume, and pressure, pulmonary compliance and resistance were measured as described by Mead and Whittenberger (1). Compliance is expressed as L. per cm. H_2O and resistance as cm. H_2O per L. per sec. at a flow of 0.5 L. per sec. In each

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[3] Made by the David Clark Company, Inc., Worchester, Mass.

[4] Modified Technitrol Integrator.

instance, compliance was measured on five successive breaths and the mean value used. Thus, for example, the pulmonary compliance at 30 seconds after G suit inflation or submersion in water, listed below, actually represents the mean value of the five breaths nearest the 30-second mark. In like manner, the respiratory resistance values represent a mean of five determinations in each instance.

Inflation of the G suit caused a decrease in functional residual capacity amounting to about 500 ml. Consequently, it was necessary to determine whether this change alone would affect the measurement of compliance, although volume changes of this amount do not significantly alter compliance under normal conditions (2). In 3 subjects, the end-expiratory lung volume was voluntarily held at the control level during G suit inflation. This was done by maintaining a constant end-expiratory position on the tidal volume record. Compliance changes during G suit inflation were found on repeated trials to be unaffected by controlling the lung volume in this way.

The possibility arises that compliance measurements might be affected by a reflex change in esophageal tone induced by inflation of the G suit or submersion in water. These procedures caused a small immediate increase in the absolute level of intraesophageal pressure which was commensurate with the attendant decrease in resting lung volume and was ascribed to this cause. The new pressure level was stable. There was no increased tendency for intraesophageal pressure to fluctuate widely. On release of G suit pressure or emersion of the subject from water, the mean level of intraesophageal pressure decreased promptly again. The small pressure change which did occur was consistent, stable, and reversible. For this reason it appears unlikely that G suit inflation or immersion in water caused reflex changes in esophageal tone which could account for the measured changes in compliance.

Circulatory measurements. Brachial artery pressure was measured by way of an indwelling arterial needle. Central venous pressure was measured through a catheter (0.065 inch I.D.) in the superior vena cava. The fourth sterno-costal junction was taken as the reference point. Statham strain gages served as transducers for both measurements.

As a rough index of changes in intrathoracic blood volume during G suit inflation, radiographic density of the lungs was followed fluoroscopically and by roentgenograms, using standard exposure and development techniques. A series of control films was made at different depths of inspiration. Films made during G suit inflation were compared with control films showing the diaphragm at the same level.

As a further index of changes in intrathoracic blood volume, thoracic radioactivity was followed continuously during G suit inflation after an intravenous injection of 10 μc. of I^{131} albumin. Monitoring was accomplished by a sodium iodide, thallium activated scintillation crystal detector with the amplified signal recorded on a direct writing apparatus. The detector was shielded to monitor an area 5 inches in diameter at a distance of 6 inches. It was positioned over the right mid-lung field to exclude the heart and liver from the area monitored. Studies were performed within five minutes after injection when essentially all of the radioactive material remained in the blood. In order to determine whether this arrangement could reflect directional changes in intrathoracic blood volume, a trial was made in each subject of the effect of reducing intrathoracic blood volume by performing a Valsalva maneuver. In each case, this caused a definite

TABLE I

Pulmonary compliance, central venous pressure and arterial pressure during G suit inflation (2 psi)

Time after G suit inflation (*seconds*)		Pulmonary compliance *L./cm. H_2O*				Central venous pressure change *cm. H_2O*			Arterial pressure *mm. Hg*	
		Control	30	60	120	30	60	120	Control	30–120
Subject										
RS	1	.245	.167		.242					
TH	2	.182	.068	.136	.174	17.0	12.0			
CT	3	.328		.148	.202	39.5	32.2	25.4		
SB	4	.229	.092	.118	.145					
RN	5	.219	.066	.117	.127	23.0	16.0	13.0	122/70	150/100
CM	6	.212	.098	.111	.171	40.7	38.6	35.2		
KW	7	.281	.124	.130	.146	30.0	25.0	22.5		
ED	8	.214	.096	.122	.147	22.0	20.0	15.5	118/63	139/86
LB	9	.168	.076	.095	.103	21.7	17.7	13.0	96/70	120/80
ST	10	.220	.106	.133	.143	20.0	15.7	12.0	100/64	125/75
MM	11	.171	.121	.146	.150	27.0	23.0	18.0	110/70	120/75
WC	12	.128	.055	.075	.078	30.3	29.3	24.2	105/50	130/70
RS	13	.200	.125	.135	.145	18.5	14.1	10.4	95/56	100/68
CH	14	.209	.066	.079	.081	35.0	27.0	22.0	95/50	130/100
RK	15	.249	.044	.058	.064	23.4	18.9	15.5		
Average		.217	.093	.114	.141	26.8	22.3	18.9		
Std. Dev.		.048	.036	.027	.046	7.8	7.8	7.2		

TABLE II

Pulmonary compliance and respiratory resistance during submersion while breathing against atmospheric pressure

Depth of submersion	Pulmonary compliance L./cm. H_2O					Inspiratory resistance cm. H_2O/L./sec.	
	Control	Neck level		Head under		Control	Head under
Time after submersion seconds	Control	30	120	30	120	Control	120
Subject							
WD	.150			.093	.090		
GH	.185	.104	.118	.047	.093	0.9	0.9
FM	.138	.084	.095	.080	.102	1.4	1.8
JN	.168	.062	.080	.049	.104		
SB	.177	.120	.132	.051	.095	1.2	2.0
RF	.224	.122	.128	.076	.094	1.2	1.6
LB	.179	.133	.186	.061	.079		
LH	.125	.072	.072	.081	.144		
Average	.168	.100	.116	.077	.100		
Std. Dev.	.031	.042	.039	.027	.021		

decrease in radioactivity over the test area while intrathoracic pressure was increased. This result was taken to indicate the probability that changes in thoracic radioactivity which occurred during G suit inflation at least partially reflected changes in intrathoracic blood volume.

G suit inflation after pretreatment with hexamethonium. Intravenous injections of hexamethonium bromide were given to seated subjects to lower systolic pressure to approximately 70 mm. Hg. There was a wide variation in the dosage required to accomplish this. The mean dose (four subjects) was 225 mgm. Central venous pressure, arterial pressure and pulmonary compliance were measured during G suit inflation before and after administration of hexamethonium.

G suit inflation during norepinephrine infusion. The central venous catheter was used to allow alternate 5-second intervals of central venous pressure measurement and infusion of norepinephrine at a constant rate. The rate of infusion was adjusted to maintain the systolic blood pressure at approximately 170 mm. Hg. Central venous pressure, arterial pressure, pulmonary compliance and thoracic radioactivity were evaluated before and during G suit inflation both before and with norepinephrine infusion.

RESULTS

Changes during the first 30 seconds of G suit inflation (Table I)

The central venous pressure of each subject rose markedly upon inflation of the G suit. At 30 seconds, the mean increase in 13 subjects was 26.8 cm. H_2O. Pulmonary compliance decreased in every instance following G suit inflation (14 subjects). The mean control compliance was .217 L. per cm. H_2O. At 30 seconds after G suit inflation the group mean was .093 L. per cm. H_2O. Arterial pressure increased (8 subjects) by a group mean of 22 mm. systolic and 20 mm. diastolic at the 30-second point. The peak pressure, which occurred immediately after suit inflation, was slightly higher than this, but after a few seconds the pressure stabilized at a nearly constant level. There was a marked increase in radiographic pulmonary vascular density and in transverse cardiac diameter (4 subjects) as compared with control films showing the diaphragm at approximately the same level. All these changes were maximal within 15 seconds after G suit inflation. Thoracic radioactivity began a sharp increase with G suit inflation and reached a peak 15 to 30 seconds thereafter (5 subjects). There was no change in respiratory rate.

Changes during the first 30 seconds of submersion (Table II)

The effect of submersion was similar to that of G suit inflation. Pulmonary compliance decreased from a mean control value of .168 L. per cm. H_2O to .100 L. per cm. H_2O with 30 seconds' submersion to the level of the neck (7 subjects) and to .077 L. per cm. H_2O with 30 seconds' total submersion (8 subjects). Respiratory resistance was not greatly changed by submersion (4 subjects). Respiratory rate did not change with submersion.

Changes during prolonged G suit inflation

Further changes during the 2-minute period of G suit inflation are detailed in Table I. As G suit

pressure was maintained, the high central venous pressure began to decrease gradually. The mean increment above control level was 26.8 cm. H_2O at 30 seconds (13 subjects), 22.3 cm. H_2O at 60 seconds (13 subjects) and 18.9 cm. H_2O at 120 seconds (12 subjects). Correspondingly, pulmonary compliance, after the initial fall, began to increase and continued increasing throughout the last 1½ minutes of constant suit pressure. The mean compliance was .093 L. per cm. H_2O at 30 seconds (14 subjects), .114 L. per cm. H_2O at 60 seconds (14 subjects) and .141 L. per cm. H_2O at 120 seconds after G suit inflation (15 subjects). These progressive changes in central venous pressure and compliance were observed in each subject.

Following the initial increase in radiographic pulmonary density with G suit inflation, definite but incomplete clearing of the increment in density was observed by fluoroscopy and by films during the 2 minutes of continued G suit pressure (4 subjects).

Thoracic radioactivity, after the initial increase with G suit inflation, decreased throughout the remainder of the 2 minutes of continued suit pressure in 4 of 5 subjects. In the remaining subject, thoracic radioactivity showed the usual initial increase with suit inflation, but did not decrease during the remainder of the 2-minute period.

Arterial pressure and respiratory rate did not change during this prolonged interval.

The finding that radiographic pulmonary density and thoracic radioactivity began to decrease while G suit pressure was maintained indicates that the intrathoracic blood volume was also decreasing. This was associated with decreasing central venous pressure and increasing compliance of the lungs. These changes all began within 30 seconds after G suit inflation and the return toward control values continued during the remaining period of constant G suit pressure.

Changes during prolonged submersion (*Table II*)

The changes during submersion for 2 minutes were similar to those during prolonged G suit inflation. Following the initial decrease in pulmonary compliance with submersion, increasing compliance was observed. During submersion to the neck, compliance increased in 7 subjects from a mean of .100 L. per cm. H_2O after 30 seconds to a mean of .116 L. per cm. H_2O after 120 seconds. This change is statistically significant ($P < .05$). In 8 subjects during total submersion compliance increased from a mean of .077 L. per cm. H_2O after 30 seconds to a mean of .100 L. per cm. H_2O after 120 seconds ($P < .01$).

The effect on pulmonary compliance of submersion, like that of inflation of the G suit, is an

TABLE III

Pulmonary compliance, central venous pressure and arterial pressure during G suit inflation before and after hexamethonium bromide

Time after G suit inflation (*seconds*)	Pulmonary compliance *L./cm. H_2O*				Central venous pressure change *cm. H_2O*			Arterial pressure *mm. Hg*	
	Control	30	60	120	30	60	120	Control	30–120
Before hexamethonium									
Subject									
ST	.164	.050	.054	.072	29	27	23	100/64	125/75
WC	.128	.055	.075	.078	30	29	24	105/50	130/70
MM	.190	.121	.146	.152	27	23	18	110/70	120/75
LB	.234	.079	.076	.094	28	26	22	96/70	120/80
Average	.179	.076	.088	.098	28.6	26.3	21.8	103/63	124/75
After hexamethonium									
ST	.229	.084	.096	.102	17	14	13.5	60/40	
WC	.137	.088	.072	.087	19	20	17	90/50	120/80
MM	.176	.105	.092	.108	14.5	14.5	13	68/40	138/80
LB	.236	.058	.065	.074	20	17	15	76/50	110/80
Average	.194	.084	.081	.098	17.5	16.4	14.6	73/45	123/80

TABLE IV

Pulmonary compliance, central venous pressure and arterial pressure during G suit inflation before and during norepinephrine infusion

Time after G suit inflation (*seconds*)	Pulmonary compliance *L./cm. H_2O*				Central venous pressure *cm. H_2O*				Arterial pressure *mm. Hg*	
	Control	30	60	120	Control	30	60	120	Control	30–120
Before norepinephrine infusion										
Subject										
RS	.200	.125	.135	.145	2.0	20.5	16.1	12.4	95/56	100/68
RN	.184	.083	.090	.096	1.5	29.5	25.5	22.5	122/70	150/100
CH	.209	.066	.079	.081	1.5	36.5	28.5	26.5	95/50	130/100
ED	.214	.096	.122	.147	0.5	22.5	20.5	16.0	118/63	139/86
LB	.216	.076	.095	.103	1.0	23.2	19.0	14.5	88/50	126/73
Average	.204	.089	.104	.114	1.3	25.9	21.9	18.3	103/58	129/86
Std. Dev.	.013	.022	.023	.028	0.1	6.4	5.0	6.1		
During norepinephrine infusion										
RS	.111	.075	.080	.081	8.5	35.2	34.8	34.8	165/82	177/105
RN	.147	.064	.063	.061	4.0	34.0	32.5	32.0	183/78	191/99
CH	.167	.043	.046	.050	5.0	44.4	44.6	43.5	150/84	157/100
ED	.176	.082	.082	.086	6.5	46.5	48.5	48.5	192/82	205/110
LB	.209	.056	.060	.060	8.5	40.5	42.0	45.0	160/85	180/100
Average	.162	.064	.066	.068	6.5	40.1	40.5	40.8	170/82	182/103
Std. Dev.	.036	.015	.015	.015	2.0	5.5	6.7	7.0		

initial sharp decrease with subsequent incomplete return toward control values during continued submersion.

Changes during G suit inflation after hexamethonium injection (Table III)

G suit inflation after hexamethonium injection produced in 4 subjects a less marked increase in central venous pressure (mean 17.5 cm. H_2O) than it produced during control observations in the same subjects prior to hexamethonium injection (28.6 cm. H_2O). During continued G suit inflation a progressive fall in central venous pressure was again apparent, but it was less marked than that which occurred during the control observations.

Pre-treatment with hexamethonium did not alter the pulmonary compliance changes associated with G suit pressure.

Changes during norepinephrine infusion (Table IV)

In 5 seated subjects norepinephrine infusion consistently produced an increase in central venous pressure and a decrease in compliance. There was no detectable change in thoracic radioactivity during norepinephrine infusion.

Changes during G suit inflation with norepinephrine infusion (Table IV)

G suit inflation during norepinephrine infusion resulted in a marked immediate increase in central venous pressure and a simultaneous decrease in pulmonary compliance. During the 2-minute period of constant G suit pressure, there was no significant further change in either of these values.

Thoracic radioactivity increased markedly with G suit inflation during norepinephrine infusion (3 subjects). During continued G suit pressure, thoracic radioactivity either remained constant or continued to increase slightly.

Immersion of arm in ice water during G suit inflation

The effect of a strong autonomic stimulus was examined in 2 subjects by immersing one arm deeply in agitated ice water for 2 minutes. As indicated in Table V, this procedure alone caused a definite fall in compliance and an elevation in central venous pressure. Table VI shows the effect of immersing the arm in ice water coincident with G suit inflation. As with norepinephrine infusion, the initial changes in compliance and venous pressure tend to be maintained during the

TABLE V

Effect of immersion of the arm in ice water on pulmonary compliance and central venous pressure

Time after immersion (*seconds*)	Pulmonary compliance *L./cm. H_2O*		Central venous pressure *cm. H_2O*	
	Control	120	Control	120
Subject				
PK	.170	.098	1.0	3.0
MM	.200	.135	1.5	6.0

entire 2-minute period instead of returning toward control values.

Release of G suit pressure and emersion from water

All measured values returned to control range within 10 seconds after release of G suit pressure or emersion from water. Invariably arterial pressure and central venous pressure fell below control levels for 3 to 5 seconds after release of G suit pressure.

DISCUSSION

Inflation of the G suit caused marked central and pulmonary vascular engorgement, as evidenced by an increase in central venous pressure, in radiographic density of the lungs, and in thoracic radioactivity after injection of I^{131} labeled albumin. Pulmonary engorgement was always accompanied by a large decrease in compliance. Upon release of suit pressure, these changes all disappeared promptly. The compliance change produced by G suit inflation was quite comparable in magnitude to that which is found in congestive heart failure (3).

The present evidence indicates that simple pulmonary vascular engorgement can produce a marked decrease in compliance, but the manner in which engorgement acts to produce this effect is not clear. It has been suggested that the lungs may be stiffened in some way by an increase in pulmonary intravascular pressures (3–8).

This factor would certainly be operative under the present conditions. It has been reported that inflation of a G suit causes an elevation of intravascular pressure throughout the pulmonary circulation comparable to the increase produced in the central venous pressure (9). However, G suit inflation also increases intrathoracic blood volume, and the present study does not exclude this factor as a determinant of compliance changes.

Similar changes in compliance have been produced by increasing the pulmonary vascular pressure in experimental animals (4, 6, 7), and rapid intravenous infusions have been reported to produce a compliance fall in normal human subjects (10). These results also suggest that simple pulmonary vascular engorgement can produce a decrease in compliance.

The mechanism of the decreased compliance in congestive heart failure appears to be somewhat more complicated. Brown, Fry, and Ebert (3) found that pulmonary compliance was decreased in patients with congestive failure, and the extent

TABLE VI

Effect of immersion of the arm in ice water on the change in pulmonary compliance and central venous pressure during G suit inflation

Time after G suit inflation (*seconds*)	Pulmonary compliance *L./cm. H_2O*				Central venous pressure *cm. H_2O*			
	Control	30	60	120	Control	30	60	120
Before immersion of arm in ice water								
Subject								
PK	.178	.055	.071	.076	1.0	22	15	11
MM	.200	.107	.120	.126	1.5	29	27	24
Average	.189	.081	.095	.100	1.2	25.5	21	17.5
During immersion of arm in ice water								
PK	.168	.069	.079	.070	1.0	33	24	28
MM	.210	.079	.083	.085	1.5	22	30	26
Average	.189	.074	.081	.076	1.2	27	27	27

of the change appeared to be related to the severity of the failure. There was a good correlation between decrease in compliance and reduction in vital capacity. Examinations have been made of the effect on compliance of elevating pulmonary vascular pressures by exercise in patients with mitral stenosis (8, 11). This often causes a decrease in compliance, but it may fail to do so in cases where compliance is already much reduced at rest. In such cases, the reduction in compliance may depend on the fibrotic pulmonary changes which are known to occur in severe mitral stenosis. It is probable that pulmonary edema also contributes to the reduced compliance of patients with congestive failure. Pulmonary edema produced by ANTU will lower compliance in the dog (12). In general, it appears that simple pulmonary vascular engorgement can reduce compliance to the extent seen in congestive heart failure, but that the compliance reduction in this disorder may depend on other factors as well.

Although most of the subjects were trained observers, only one complained spontaneously of dyspnea during G suit inflation. Two others described upon questioning a vague sense of shortness of breath during the experiments. The youth and vigor of the subjects as well as the acute nature of the experiments render difficult the interpretation of this subjective sensation. It is evident that the healthy young adult tolerates very low pulmonary compliance for short periods without experiencing dyspnea at rest.

On inflating the G suit and maintaining its pressure, there was at first an abrupt increase in central and pulmonary vascular engorgement, and then a gradual decrease, as indicated by a progressive decline in central venous pressure, in radiographic density of the lungs and in thoracic radioactivity, and by a simultaneous increase in compliance. Similar compliance changes occurred during submersion. These changes suggest that the initial abrupt increase in intrathoracic blood volume is being reduced by redistribution of blood to the periphery. The combination of a gain in peripheral blood volume occurring conjointly with a fall in central venous pressure might be produced by an overall fall in peripheral vascular tone, particularly venous tone. It might also be caused by pooling of blood in vascular regions which are under less than the mean externally applied pressure and which can accept additional blood from the arterial side until the local venous pressure rises sufficiently to open venous valves and re-establish a return flow into the general venous system. The first of these mechanisms would represent a vasodepressor response, the second a passive pooling. To investigate this subject further, trials were made of the effect of altering venomotor tone by appropriate drugs before G suit inflation.

Partial ganglionic blockade with hexamethonium greatly reduced the venous pressure rise after G suit inflation, possibly because of a decrease in "resting" venomotor tone, but the effect on compliance was unchanged. This response does not differentiate between the two mechanisms.

Pre-treatment with norepinephrine abolished the evidence of redistribution of blood from the central region back into the periphery. It is conceivable that this blocking effect might result either because the direct action of norepinephrine on vessels prevented the occurrence of a vasodepressor response or because overall venous tone was so increased that no significant passive pooling could occur. Central venous pressure was increased by norepinephrine alone and was very greatly increased by subsequent inflation of the G suit.

The existence of venomotor reflexes which are capable of producing a considerable increase in the central venous pressure is well established (13, 14). Whether the present redistribution phenomenon represents an actual venodepressor response or passive pooling, it is apparent that the extent of the initial rise in venous pressure and the subsequent course of events depend very closely on venous tone. It is still not clear whether a decrease in venous tone occurs during the period of G suit inflation and contributes to the redistribution of blood.

The ice water experiments indicate that the nervous system can mediate changes in venous tone in normal subjects which are associated not only with an increase in central venous pressure but also with a considerable fall in compliance. By analogy with the other experiments in which central venous pressure is elevated, it appears likely that this decrease in compliance reflects some degree of pulmonary vascular engorgement resulting from a transfer of blood from the systemic to the pulmonary circulation. When cen-

tral and pulmonary vascular engorgement are already present, as after G suit inflation, the nervous system can mediate an increase in venous tone which is able to sustain very high pressures and which apparently maintains pulmonary engorgement by resisting transfer of blood from the pulmonary to the systemic circulation. The results obtained with ice water closely simulate those obtained during norepinephrine infusion.

SUMMARY

1. Brief, acute central and pulmonary vascular engorgement was induced in normal subjects by application of pressure to the body surface. This was done either by inflation of an aviator's G suit to 2 PSI or by submersion in water while breathing against atmospheric pressure. G suit inflation produced in 15 subjects an increase in central venous pressure of 26.8 (S.D. 7.8) cm. H_2O and a fall in compliance from the control value of .217 ± .048 L. per cm. H_2O to .093 ± .036 L. per cm. H_2O. A similar compliance fall occurred with submersion.

2. On maintaining G suit pressure or submersion for a 2-minute period, central venous pressure and compliance showed a progressive, but incomplete, return toward control levels. Infusion of norepinephrine or immersion of an arm in ice water during this time prevented the return of venous pressure and compliance toward control levels.

3. Administration of norepinephrine or immersion of an arm in ice water without suit inflation caused a moderate increase in central venous pressure and a moderate decrease in compliance.

4. When the G suit was inflated during norepinephrine infusion, the resultant elevation of central venous pressure and the drop in compliance were greater than in untreated subjects. Pretreatment with hexamethonium diminished the rise in central venous pressure which occurred with suit inflation, but did not affect the compliance changes.

5. It is suggested that the compliance changes induced by these procedures depend, at least partially, on varying degrees of pulmonary vascular engorgement and that this, in turn, is partially dependent, under the present experimental conditions, on peripheral venous tone.

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