The Physiology of Pressure Suits

By Wing Commander H. L. Roxburgh and
Squadron Leader J. Ernsting, RAF

DESPITE the recent advances in the field of pressure suit development no suit exists which, while giving adequate protection against the effects of exposure to low barometric pressure, does not seriously interfere with the functional efficiency of the wearer. Thus in high altitude military aircraft the cabin is generally pressurized, and pressure suits, if worn at all, are used for emergency purposes only. It is possible to envisage two different sets of conditions under which a pressure suit may be employed in the event of failure of the pressure cabin. The suit may be employed to give short term protection to the wearer, in order that he may descend to an altitude where protection is not required, either in his aircraft or following escape from it. Alternatively, the suit may be used to give protection for a long period to enable the airman to remain within his aircraft at a high altitude when the pressure cabin has failed.

The two physiologic effects of reduced pressure, which are of vital importance to this report, are anoxia and decompression sickness. Experience has shown that freedom from serious anoxia may be obtained in pressure suits by the breathing of pure oxygen and the maintenance of an absolute intrapulmonary pressure of at least 141 mm. Hg. (barometric pressure at 40,000 feet). Freedom from decompression sickness, however, requires either that the exposure to the reduced pressure be brief, or that the body is not exposed to a pressure less than 225 mm. Hg. absolute (barometric pressure at 30,000 feet), or that some special measures are taken to reduce the susceptibility of aircrew to this disturbance. These relatively simple facts lead to the conclusion that prolonged protection against very high altitudes can only be afforded by means of a suit which maintains the pressure around the body at a value equal to or greater than 225 mm. Hg. when the cabin pressure falls below this value. The only form of garment which can fulfill these requirements is the full pressure suit. On the other hand, if descent can be made rapidly the occurrence of decompression sickness is unlikely to be a serious factor in determining the efficiency of the aircrew member and under these circumstances the suit is only required to give protection against anoxia.

Partial pressure suits, which give protection against anoxia at altitudes greater than 40,000 feet, aim therefore at the maintenance of an absolute pressure of 141 mm. Hg. in the respiratory tract. The equipment which will perform this task ranges from the simple pressure oronasal mask alone

From the Royal Air Force Institute of Aviation Medicine, Farnborough, Hants, England.

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to the complete full pressure suit. The proportion of the body surface which must be pressurized in addition to the nose and mouth is determined by the magnitude of the breathing pressure employed, i.e. the maximum altitude at which protection is required and the length of time for which it is operative. The degree of body coverage required for a given breathing pressure and length of exposure is determined by the physiologic disturbances, primarily respiratory and circulatory, which are induced by the increased intrapulmonary pressure. While here too the full cover pressure suit represents the physiologic ideal the probable decrease in the efficiency of the wearer which it induces, even in the unpressurized state, dictates a compromise. This compromise must strike a balance between complete physiologic protection, which entails whole body coverage, and full flying and fighting efficiency which demands minimum body coverage. For the short term protection of high flying aircrew against anoxia in the event of loss of cabin pressure such a compromise is the correct and the best practical solution of a very complex problem.

THE TRUNK

The primary effect of a rise in intrapulmonary pressure is distension of the lungs. The pressure volume diagram of the lungs and thorax are now familiar features in respiratory physiology.[12] The changes in the vital capacity and its subdivision during pressure breathing in the seated position at various pressures with no trunk counterpressure are depicted in Figure 1. At a breathing pressure of 30 mm. Hg. the inspiratory reserve is reduced from 3.0 liters to 1.0 liter (B.T.P.S.*), and at higher pressure the chest is even more markedly distended; at very high pressures there is danger of rupture of the lung parenchyma owing to failure of the chest wall to protect the lungs. The work of breathing under such conditions is much increased as the relaxed lung volume is never reached during the respiratory cycle, the tension in the expiratory muscles being maintained above normal for as long as the intrapulmonary pressure is raised. Owing to this pulmonary distension and the circulatory effects produced by breathing pressures much in excess of 30 mm. Hg. this pressure represents the practical limits of the pressure breathing mask or helmet alone.

Many untrained subjects when pressure breathing with no trunk counterpressure over-ventilate to such an extent that the alveolar carbon dioxide tension is lowered to the level at which clinical symptoms and signs of hypocapnia occur. This is probably the most serious disturbance induced by pressure breathing apart from the circulatory changes associated with the raised intrapulmonary pressure. Considerable training is required with many members of aircrew in order that they may overcome this tendency to hyperventilate. Our experiments and those of other investigators suggest strongly that in untrained subjects the tendency to overventilation during pressure breathing is markedly re-

*B.T.P.S.=Volume of gas measured at body temperature, intrapulmonary pressure and saturated with water vapour at body temperature.

duced by complete trunk counterpressure.

Some form of counterpressure must be applied to the chest at breathing pressures greater than 30 mm. Hg. in order to prevent the disturbances enumerated above, i.e., to prevent lung damage due to excessive distension, to decrease overventilation and to reduce the work of breathing to near its normal value. The efficiency of various degrees of chest and trunk counterpressure has been investigated by determining the values of the vital capacity and its subdivisions, at various breathing pressure up to 80 mm. Hg. In the ideal full cover suit there will be, by definition, no change in the values of the vital capacity and its subdivisions during pressure breathing as compared with their resting values. The less effective the counterpressure the larger will be the changes in the subdivisions of the vital capacity and the nearer these changes will approach the values seen in pressure breathing without counterpressure. The results are shown in Figure 1, the values being obtained in the seated position.

Counterpressure applied to the chest alone by means of an RAF pressure-breathing waistcoat in direct communication with the mask tube was found to restrict the lung distension with positive pressure breathing. A breathing pressure of 80 mm. Hg. with chest counterpressure produces a similar degree of lung distension as occurs during pressure breathing at 35 mm. Hg. (Fig. 1). The amount by which chest counterpressure alone fails to limit lung distension indicates the part played by the descent of the diaphragm in allowing lung distension during pressure breathing. The cap stan partial pressure suit with neither chest nor trunk bladder is no more efficient in applying counterpressure to the trunk than the pressure breathing waistcoat.

Finally, Figure 1 demonstrates the very moderate increase in the expiratory reserve which occurs when counterpressure is applied by a bladder covering the trunk and inflated to breathing pressure. At a pressure of 80 mm. Hg. the expiratory reserve is only increased by about 500 ml. (B.T.P.S.). The failure of the particular garment investigated to prevent any change in the resting chest position is due to the existence of unsupported areas and some elevation of the thoracic cage. In addition compression of abdominal gas and displacement of blood from within the thorax and abdomen into the extremities will increase the vital capacity and may be responsible for some of the increment in expiratory reserve. There is subjective comfort in this garment and much experience has been gained with unselected aircrew untrained in pressure breathing. Only one subject in a series of fifty men exhibited gross over-ventilation with clinical features of hypocapnia.

Thus from all physiologic aspects the counterpressure afforded by a bladder encompassing as much of the trunk as is compatible with efficiency of the wearer, is most desirable. The gross respiratory disturbances induced by pressure breathing are minimized by counterpressure of this type and distribution. In addition such a garment is simple, easy to don, comfortable and need not restrict activity in

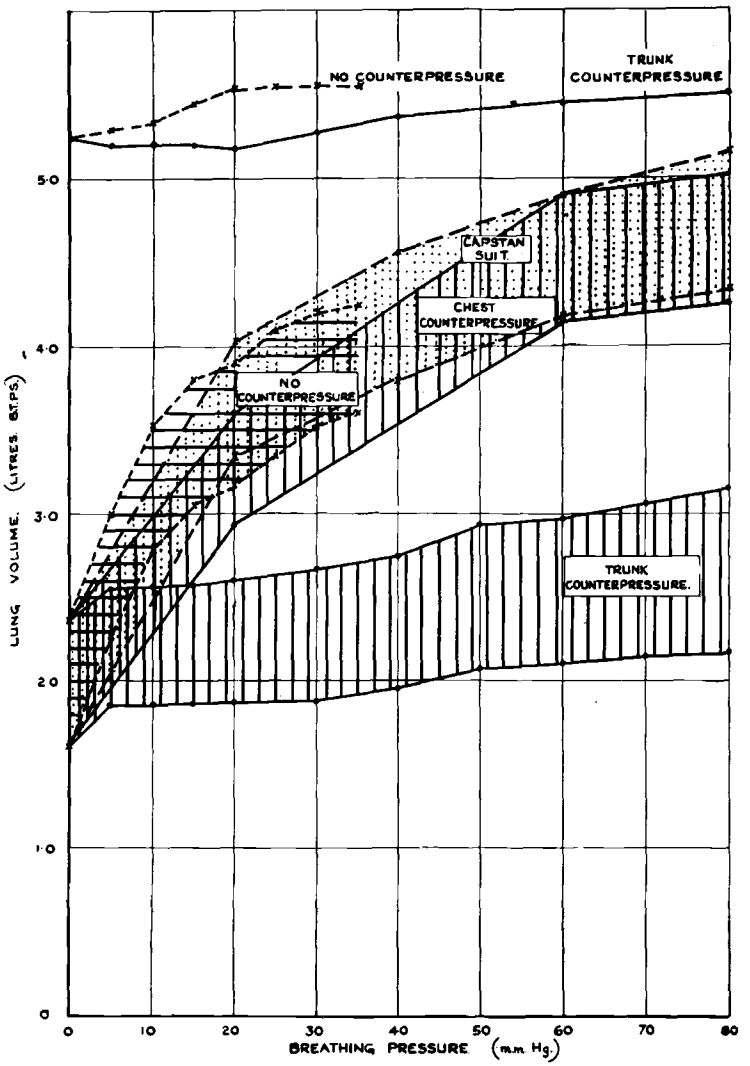

Fig. 1. Pressure-volume diagram of chest with varying degrees of trunk counterpressure. Upper lines represent vital capacity. Shaded areas represent positions of tidal air bands in the pressure-volume diagram, during pressure breathing with (1) no counterpressure, (2) chest counterpressure, (3) counterpressure given by capstan suit and (4) trunk counterpressure by gas filled bladder. Lung volumes are expressed as liters of gas measured at body temperature, intrapulmonary pressure and saturated with water vapour at body temperature (B.T.P.S.).

the cockpit during flight nor is the fitting problem critical. In the pressurized state upper limb and head mobility are good.

THE HEAD AND NECK

The oronasal mask represents the minimum cover which can be applied to this region. By means of such a

mask positive pressure may be applied to the gases in the respiratory tract. In addition to the general respiratory and circulatory disturbances produced

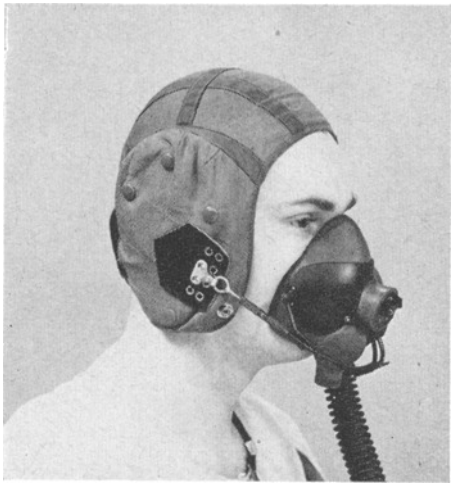

Fig. 2. Subject wearing RAF Type "N" pressure breathing mask with toggle harness.

in all the uncovered areas of the body special problems arise associated with the eyes, the ears and the neck. While there is extensive literature with regard to pressure breathing, the pressure differentials at which serious disturbances occur quickly in these regions with an oronasal mask have only been attained of recent years, the limit in the past being the difficulty of obtaining an adequate pressure seal between oronasal mask and the face at a pressure much in excess of 30 mm. Hg. We have conducted experiments using equipment which has enabled us to obtain satisfactory seals at much higher pressures.

The failure to obtain a high pressure seal using an oronasal mask with a reflected rubber edge is generally due to either weakness of the mask itself or an inadequate mask harness. The first requirement for a satisfactory high pressure mask is a firm exoskeleton which fits snugly over the external surface of the mask and affords adequate securing points for the harness. The other requirement is a satisfactory system of connection between the mask exoskeleton and the helmet. The nature of such a system is determined by the tension it has to exert upon the mask, relative to the pressure to be sealed by the mask.

The tension in the mask-helmet harness has been measured at various breathing pressures using an RAF oronasal mask (type 'N'). The relationship between the maximum mask pressure sealed and the corresponding mask harness tension is linear. In order to seal the mask under normal non-pressure breathing conditions the harness tension required is about 1.0 kg.wt., whereas the harness tension necessary to seal against a breathing pressure of 40 mm. Hg. is in the region of 6.0 kg.wt. A harness tension of 6.0 kg.wt. with normal respiratory mask pressure fluctuations gives very severe face discomfort when it is exerted for any but very short periods. It is evident that a practical answer to the problem of sealing a high pressure in an emergency is to employ a harness with a readily variable tension. Such a harness, the toggle harness (Fig. 2), has been developed and has undergone extensive trials. It is essentially a non-extensible system which allows two pressures to be applied to the mask exo-skeleton. The one insures mask seal under ordinary circumstances with very good comfort,

while the other tension gives a mask seal at high breathing pressures.

Considerable experience has been gained during recent years in high pressure breathing with trunk counterpressure employing an oronasal mask on the one hand and the RAF partial pressure suit headpiece which gives full pressurization to the face and some to the upper neck, on the other hand. The practical advantage of the mask as against any form of headpiece has encouraged close study of the special physiologic disturbances induced by failure to pressurize certain regions of the head and neck during high pressure breathing. These special features arise in connection with the eyes, with the extension of the respiratory passages in the form of the nasolacrimal duct to the cannaliculi of the eyelids and the pharyngotympanic tube to the middle ear, and with the special receptor regions of the vascular tree in the carotid arteries in the neck. Much previous experience demonstrated that breathing pressures up to 30 mm. Hg. could be tolerated for short periods using a mask only. Recent experimental work using oronasal masks with the toggle harness have suggested that this form of head "pressurization" may be employed for short periods at considerably higher pressures provided, of course, that the counterpressure on the trunk is adequate.

Eyes.—The unsupported vessels of the conjunctiva are subjected to a marked increase in transmural pressure, the increase amounting virtually to the applied breathing pressure. Studies in the effects of negative accelerations have suggested that when the increment in the venous pressure at the level of the eye exceeds 50 mm. Hg. rupture of conjunctival vessels and subsequent hemorrhage may occur.[7] In our experience with high pressure breathing no conjunctival hemorrhages have occurred but the highest pressures have only been applied for periods of from 30 to 60 seconds.

The vessels within the globe of the eye are loosely supported, but they are in very close proximity to the aqueous and vitreous humours. The evidence at present available[11] suggests that an increase in the venous pressure within or without the ocular globe is immediately followed by an equal increment in the pressure in the intraocular extravascular fluid. This consideration makes the occurrence of intraocular hemorrhages during high pressure breathing very improbable. This is parallel to the case within the skull and vertebral canal where even at very great negative accelerations no damage has been noted in the central nervous system.[8]

Nasolacrimal Ducts.—In many subjects it has been found that the nasolacrimal ducts are opened by a high pressure in the nose. Under these circumstances gas streams through the lacrimal cannaliculi into the conjunctival sacs, and incites reflex spasm of the eyelids with consequent reduction in vision. This feature may be a serious factor limiting the use of such a system for emergency short term high pressure breathing.

Ears.—In a large series of exposures to a breathing pressure of 50

mm. Hg. with a mask and trunk counterpressure no complaints of ear pain occurred. It is apparent that the pressure in the middle ear cavity was

Fig. 3. Subject pressure breathing at 52 mm. Hg. with RAF type "N" pressure breathing mask and toggle harness.

probably not raised in these subjects. If it had been the drumhead would have been exposed to a pressure differential of 50 mm. Hg., and ear pain may have resulted. This was confirmed by direct examination of the drumhead. These observations serve to confirm that the pharyngotympanic tube is normally closed and that a high oronasal pressure does not of its own necessarily cause an increase in middle ear pressure.

In our experience the occurrence of ear pain is not a serious factor limiting the use of an oronasal mask for high pressure breathing for short periods.

The Neck.—The distention which occurs in the neck with pressure breathing at 50 mm. Hg. with an oronasal mask and trunk counterpressure is shown in Figure 3. The floor of the mouth bulges downwards, the superficial veins are engorged with blood and the lower part of the neck is markedly distended. The bulging of the floor of the mouth is due to the inability of the muscles to maintain it in its normal position. This stretching of the floor of the mouth can give rise to some discomfort.

The marked distension of the neck induced by high pressure breathing is due to an increase in the blood content of the region and distension of the pharynx and upper respiratory passages. Radiographs of the neck demonstrate that the lung apices, although not subjected to counterpressure, do not expand into the neck.

Other investigators have been interested in the behavior of the carotid sinus mechanism during pressure breathing,[2] but these observations have been made at relative low breathing pressures. The part played by the carotid sinus mechanism in the cardio-vascular response to high pressure breathing has been investigated by Ernsting and Parry[5] in experiments in which neck counterpressure has been either applied or released during pressure breathing with an oronasal mask and trunk counterpressure. The magnitude of the initial cardiovascular disturbance induced by a change in the state of the neck depends in part upon the direction of the change. Following the release of neck pressurization there is a marked bradycardia and a fall in blood pressure of between 25 and 30 mm. Hg. (with a breathing pressure of 50 mm. Hg.) after about eight seconds (Fig. 4). After about fifteen seconds, however, the heart rate and arterial pressure reach a new steady level. While the heart rate is not significantly altered, as compared with the rate before removal of pressurization, the mean arterial pressure is from 10 to 15 mm. Hg. lower. In contrast,

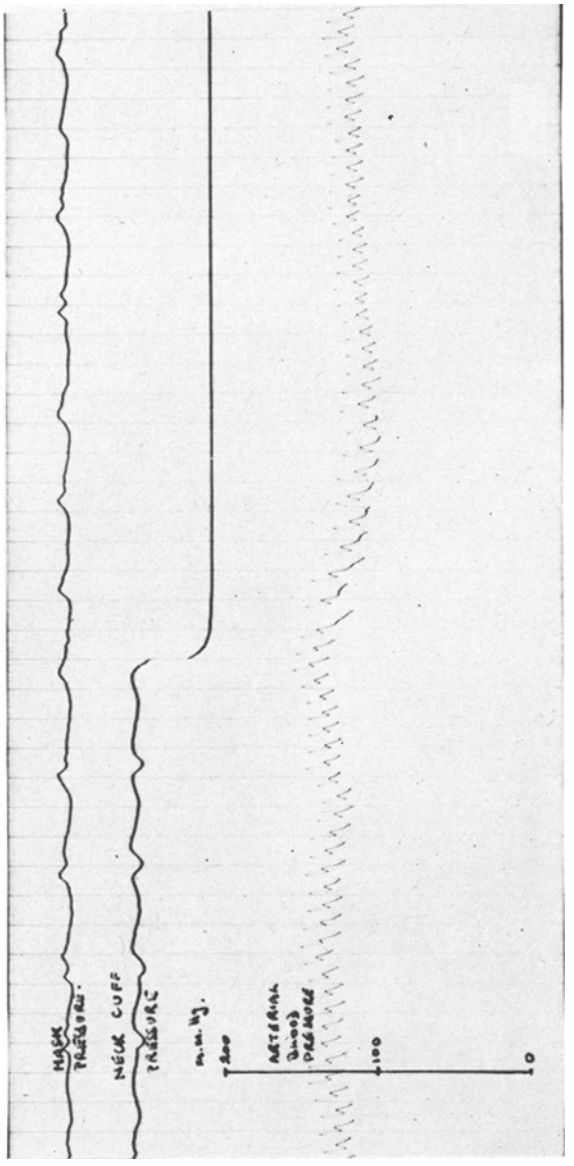

Fig. 4. Effect of sudden depressurization of the neck during pressure breathing with a mask and trunk counterpressure at 52 mm. Hg. upon arterial blood pressure. Records from above downwards are pressure in neck bladder, pressure in mask and brachial arterial pressure. Vertical lines are one second apart.

when counterpressure is applied to the neck during pressure breathing there is no significant change in pulse rate and the arterial pressure gradually rises to a new steady state in about six seconds. The increment in mean arterial pressure amounts to between 10 and 15 mm. Hg.

The most interesting feature of these experiments is the transient nature of the bradycardia which follows depressurization of the neck. The bradycardia might be due either to a transient stimulation of the receptors in the neck arteries or to a change in the response of the cardiovascular coordinating mechanism to the impulses arising from these receptors. Immediately following depressurization of the neck region there is a marked increase in the transmural pressure of the carotid artery, and hence stimulation of the receptors in its wall, giving the initial bradycardia. Following depressurization of the neck blood collects in the vessels, particularly the veins of the part, distending them until their intraluminal pressure once again exceeds the breathing pressure. It might be supposed that this distension of the large vessels, in particular the internal jugular vein, would raise the pressure without the carotid sinus and artery and hence reduce the transmural pressure. Anatomically, however, the fascia of the carotid sheath which encloses the carotid artery and jugular vein, is not inextensible and the maintained bradycardia of negative accelerations, which is abolished by counterpressure over the neck, discounts such a mechanism. In addition the maintained decrease in arterial pressure during pressure breathing following depressurization of the neck illustrates that there is maintained stimulation of the pressure receptors in the arteries of the neck.

The transient nature of the bradycardia following the removal of neck counterpressure is due, therefore, to changes in the response of the cardiovascular coordinating mechanism to the impulses arising in the carotid sinus. For a short period the carotid sinus mechanism apparently overrides the afferent impulses from other regions of the cardiovascular system, but the heart rate rapidly returns to its previous level. However, the maintained reduction in the arterial pressure rise during pressure breathing without neck pressurization indicates that the arterial pressure receptor mechanism has a continued influence upon the cardiovascular centre.

Thus in addition to the removal of the physical discomfort associated with the gross distension of the neck vessels the application of counterpressure to this region has certain physiologic advantages. It prevents blood pooling, excessive loss of fluid into the tissues of the neck, and the stimulation of the pressure receptors in the carotid arteries.

THE EXTREMITIES

A large series of experiments have been conducted with the full trunk counterpressure described above and a pressure helmet to determine the limits of protection afforded by these garments against the circulatory effects of high pressure breathing. Under certain conditions of pressure and time unconsciousness occurs. These collapses are essentially an expression of the effect of absence of counterpressure over the extremities and lower neck in high pressure breathing.

The clinical form of these collapses in our experiments has been very consistent in the same and in different subjects. It is generally heralded by nausea, a feeling of uneasiness and sweating. These symptoms are rapidly followed by dimming of vision and in many experiments blackout, and finally unconsciousness. On two occasions convulsive movements of the limbs have occurred. The whole sequence takes place vey rapidly and at the stage of actual collapse there is extreme facial pallor and the skin of the extremities is cold, pale and damp. The pulse is extremely slow and has a very poor volume. The breathing pressure

has always been released at this point. Consciousness is rapidly recovered while the bradycardia, facial pallor, nausea and sense of "faintness" persist for an hour or so to varying degrees.

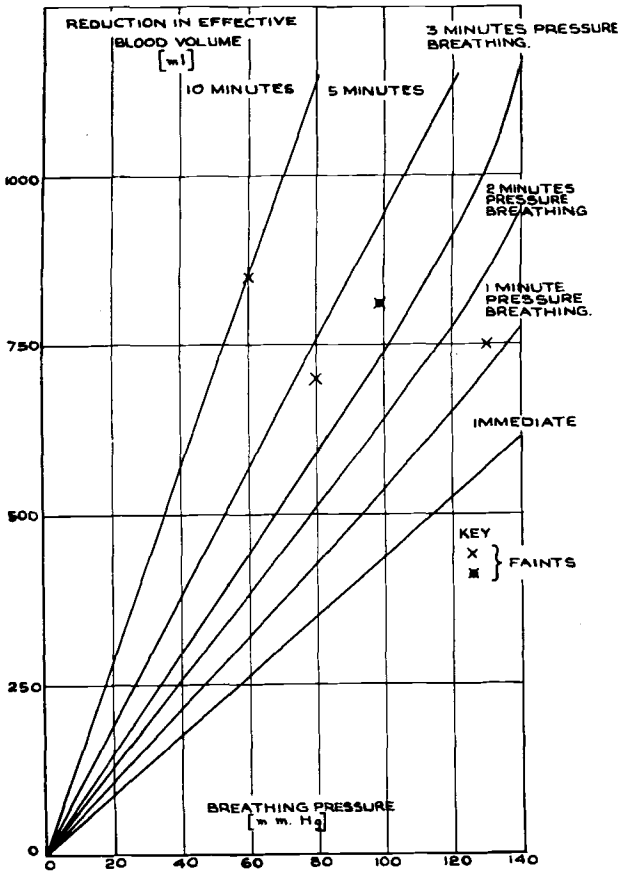

Fig. 5. Reduction in the effective blood volume, caused by pressure breathing with trunk counterpressure, as related to the breathing pressure and the length of exposure to it. The crosses depict the points in the pressure-time relationship at which fainting occurred.

These collapses had all the appearances clinically of the vasovagal syndrome so clearly described by Lewis.[10] The full mechanism of this syndrome was elicited during and after the last war by Barcroft, Sharpey-Schafer, Edholm and their colleagues. They induced faints by venous congestion of the lower limbs with venesection, acute anoxia, emotional disturbances and passive tilting. They demonstrated that the sudden extreme arterial hypotension which characterizes vasovagal syncope is due to a profound active vasodilatation of muscle and some visceral arterioles, the cardiac

output being maintained without reduction in spite of the bradycardia.[1]

Our investigation of high pressure breathing collapses is not incomplete but there is much evidence that the mechanism is that of the vasovagal attack. The arterial pressure which is grossly elevated during pressure breathing with trunk counterpressure is maintained at its high level until the commencement of a collapse when it falls precipitously to recover slowly with a marked bradycardia. Measurements of peripheral blood flow by a variety of methods have shown a great increase in limb, and particularly muscle, blood flow during collapses. Although we have not demonstrated the absence of a gross fall in cardiac output in the pressure breathing faints it is probable that their mechanism is very closely akin to that of vasovagal faints.

The reduction in effective blood volume caused by pressure breathing has been noted by other workers.[6,9] With the form of counterpressure employed in our experiments blood pooling and increase in tissue fluid volume can only occur to any significant degree in the limbs and lower neck. The changes in limb volumes due to both these factors have been studied in detail by Ernsting[3] in a series of subjects while pressure breathing seated in an ejection seat at an ambient temperature of from 18° to 22° C. The combined effects of these two factors upon the effective blood volume is shown in Figure 5, along with the time-pressure values at which fainting occurred in two experienced subjects. The reduction in effective blood volume, due to the unpressurized limbs, was between 650 and 850 ml. when these collapses occurred. Thus the magnitude of the reduction in effective blood volume associated with pressure breathing collapses is similar to that found by Wallace and Sharpey-Schafer,[13] to precipitate vasovagal faints due to hemorrhage alone allowing for the differences in the posture of the subjects in the two series.

These observations support the concept that the primary cause of fainting in high pressure breathing with trunk counterpressure is a reduction in the effective blood volume. In some experiments, especially with unexperienced subjects, collapses have occurred when the calculated total reduction in effective blood volume has amounted to only about 400 ml. In these cases however, other potent stimuli of vasovagal syncope have been present. Among these stimuli, emotional disturbance, testicular or arm pain, hyperventilation with hypocapnia, acute anoxia, relatively painless venepuncture and ill health have all been noted. Such disturbances are most unlikely in trained subjects and this conclusion has been supported by experiments involving fifty aircrew.

The vascular disturbances induced in the unpressurized limbs by high pressure breathing are due to the increase in transmural pressure in the limb vessels. The changes in transmural pressures equal the changes in intravascular pressures in most regions for only in the deep muscles of the calf and the anterior tibial group is the tissue pressure raised significantly during high pressure breathing.[4] By applying external counterpressure to the limbs so that the tissue pressure is

raised by an amount equal to the breathing pressure blood pooling and loss of fluid from the vessels may be prevented. Counterpressure has been applied to the lower limbs during high pressure breathing by means of a *g*-suit, and in certain circumstances to the upper limbs by a series of bladders similar to those of the *g*-suit.

SUMMARY

Equipment which will maintain an absolute pressure in the lungs of 141 mm. Hg. in the event of exposure of aircrew to altitudes above 40,000 ft. will afford short term protection against those altitudes and enable emergency descent to be made. A compromise is dictated between the physiologic ideal of full body pressurization and the operational ideal of the fully efficient man. The degree of regional counterpressure required is dependent upon the magnitude of the pressure necessary to maintain an intrapulmonary pressure of 141 mm. Hg. and the length of time for which it is operative.

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"No Smoking in This Space Ship"

Although the first space ship travelers may not want to smoke during their flight, it is likely that the effect of competition would eventually lead to space ships in which one could smoke. However, a cigarette lighted in an atmosphere of pure oxygen burns at approximately the rate and with the same appearance as a Fourth of July sparkler. Food in an all-oxygen atmosphere would discolor and become unappetizing very quickly.—FREDERICK H. GREEN: Airconditioning in Upper Space, *Western Aviation*, February, 1956.