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J. ERNSTING

AGARDOGRAPH 106

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THE ADVISORY GROUP
FOR AEROSPACE RESEARCH AND DEVELOPMENT OF
THE NORTH ATLANTIC TREATY ORGANIZATION

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SUMMARY

Positive pressure breathing with oxygen is a means whereby an acceptable arterial oxygen tension may be maintained at altitudes in excess of 40000 ft. The nature of the disturbances induced by raising the intrapulmonary pressure by between 30 and 140 mmHg has been investigated. It has been shown that these disturbances may be reduced to within acceptable limits by applying counterpressure to certain specific regions of the body. Pressure breathing at these pressures distends the lungs and induces a marked alveolar hyperventilation. The application of counterpressure to the trunk reduces these effects and is essential at positive pressures greater than 40 mmHg. The use of an oronasal mask for pressure breathing allows distension of the mouth and pharynx, increased activity of the carotid baroreceptors and haemorrhages in the conjunctivae and tympanic membrane. Counterpressure to the head and neck is required at positive pressures above 65 mmHg. Raising the intrapulmonary pressure reduces the effective blood volume and collapse occurs when the reduction exceeds 700 to 800 ml. These collapses, which have all the features of vasovagal syncope, may also be precipitated during pressure breathing by hypoxia, hypocapnia, discomfort or pain. The magnitude of the reduction of effective blood volume may be decreased by applying counterpressure to the limbs but the cardiovascular disturbances induced by pressure breathing limit the time for which this manoeuvre may be used at high altitude. It has been shown, however, that provided the duration of an exposure is less than four minutes, pressure breathing with limited counterpressure will provide protection against hypoxia at altitudes of up to 70000 ft.

EDITORS NOTE: Since the publication of this volume Air Commodore W. K. Stewart has been promoted to Air Vice Marshal and Group Captain H. L. Roxburgh to Air Commodore.

INTRODUCTION

Although altitude sickness was familiar to the mountaineers and balloonists of the early nineteenth century the cause of the condition was not settled until Bert 1878 (34) carried out his experimental studies of the effects of low and high pressures upon living organisms. He demonstrated in beautifully designed experiments that the principal symptoms of altitude sickness were the result of the lowering of the partial pressure of oxygen and not due to the reduction of total pressure per se. He showed in experiments on himself that the symptoms induced by reduction of environmental pressure could be prevented by the administration of oxygen. In spite, however, of his clear demonstration that serious impairment of consciousness developed rapidly at a pressure of 250 mmHg absolute and of the need for an adequate oxygen supply at this pressure, two balloonists, Croce-Spinelli and Sivel, perished in the tragic flight of the Zenith in 1875. These balloonists were the first of many aviators to die as a result of oxygen lack occurring in flight. Bert also demonstrated that if the environmental pressure was reduced to a sufficiently low level, even 100% oxygen would not maintain consciousness in a variety of animals. Further, he found that when the total pressure was reduced to about 80 mmHg, death became imminent.

The introduction of a simple and direct method for obtaining in man samples of alveolar air by Haldane and Priestly 1905 (137) opened the way to a quantitative study of the effects of reduced environmental pressure upon respiratory gas exchange. Using the normal values for the tensions of carbon dioxide and water vapour in the alveolar gas, Haldane 1920 (135) calculated that at an altitude of 35000 ft (barometric pressure — 179 mmHg, the relationship between altitude and barometric pressure is that defined by the International Civil Aviation Organisation and the United States Standard Atmosphere) (Fig. 1-6) the alveolar oxygen tension would still be at least 53 mmHg. He concluded that marked symptoms of oxygen lack when oxygen was breathed would only begin to appear at pressures below 140 mmHg absolute and that these symptoms would become urgent in an unacclimatized person at barometric pressures of less than 100 mmHg. Having made these calculations Haldane 1920 (135) continued:

> If it were required to go much above 40000 ft and to a barometric pressure below 130 mm (mercury) it would be necessary to enclose the airman in an air tight dress, somewhat similar to a diving dress but capable of resisting an internal pressure of, say, 130 mmHg. This dress would be so arranged that even in a complete vacuum the contained oxygen would still have a pressure of 130 mm. There would then be no limit to the physiological height obtainable.

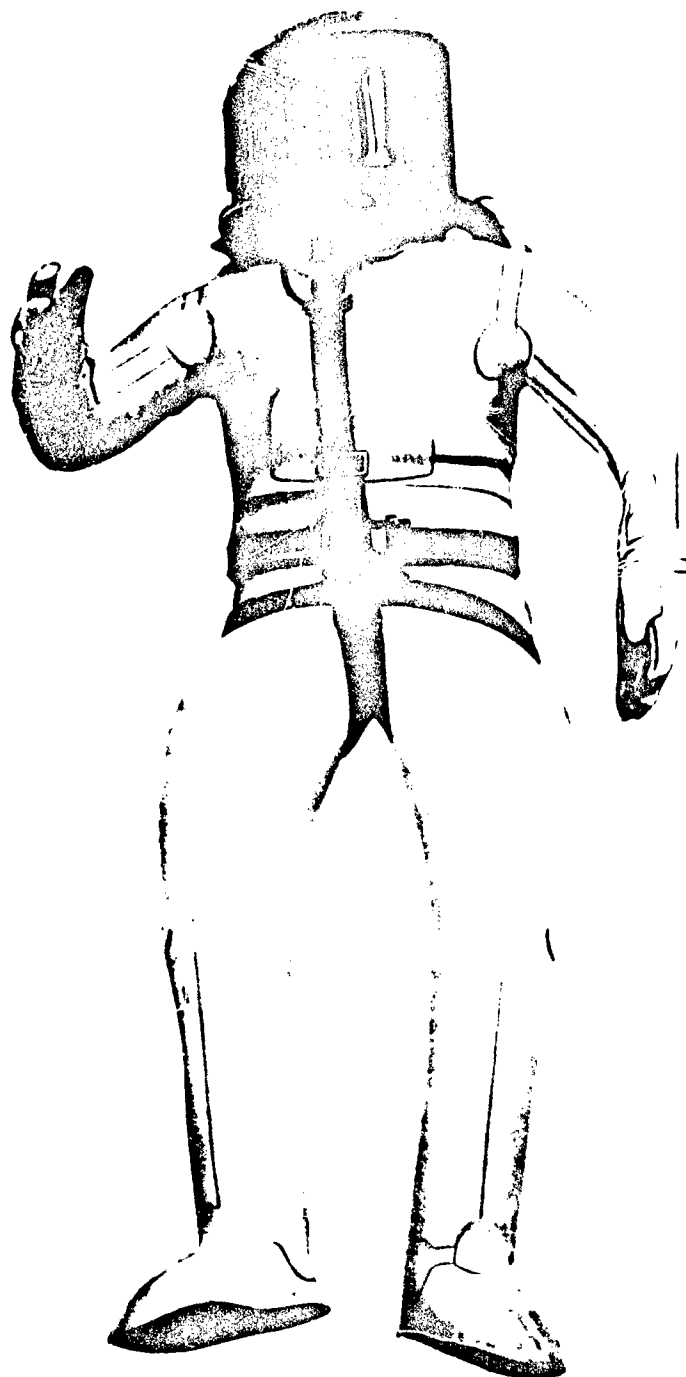It would appear that with this statement Haldane was the first investigator to

FIG. 1-1 Suit worn by Squadron Leader Swain in 1936

suggest the use of a pressure suit to protect man against hypoxia at altitudes above 40000 ft.

No attempt was made to follow up the concept of a high-altitude suit until 1933 when Ridge, an American balloonist, approached Haldane to seek assistance in the development and testing of such a suit. With the help of Davis of Messrs. Siebe Gorman and Co. a self-contained diving dress was modified so that it could be inflated with oxygen to a pressure of 150 mmHg. Whilst wearing this suit Ridge was exposed in a decompression chamber to an absolute pressure of 17 mmHg (equivalent to an altitude of 84000 ft) without any untoward effects (138) (72). In his account of these experiments Haldane also considered the possibility of the occurrence of decompression sickness and the protective value of breathing 100", oxygen before an exposure to the low environmental pressure. A modified version of this suit (Fig. 1–1) was used in 1936 by Squadron Leader Swain, R.A.F., who flew to an altitude of 49967 ft, and by Flight Lieutenant Adams, R.A.F., who reached an altitude of 53936 ft in 1937 (202).

An alternative method of protecting an individual against the effects of exposure to high altitude, the sealed gondola, was successfully put into practice in 1931 by Piccard 1933 (236). The gondola, which contained the crew, was sealed and the pressure within it was maintained during flight at one atmosphere by the vaporization of liquid oxygen. The concept of protecting the occupants of the cabin of an aircraft from the effects of high altitude by increasing the pressure of the air within the cabin was formulated at the end of the First World War. The first aircraft successfully fitted with such a pressurized cabin did not fly, however, until 1937 (7) and the majority of aircraft were not fitted with pressurized cabins until after 1945.

By 1939 it was generally accepted that in practice the maximum altitude to which an individual breathing 100", oxygen could be exposed without serious impairment of consciousness was 40000 ft (Armstrong, 1939). During the Second World War, however, it became necessary for certain military aircraft to operate at altitudes above 40000 ft. The cabins of these aircraft were not pressurized and the full pressure suits available at the time were too cumbersome to be of any operational use. In December 1941 Gagge, Allen and Marbarger 1945 (119) showed that it was possible to raise the altitude at which useful consciousness was maintained above 40000 ft by positive pressure breathing using a mouthpiece. In his initial experiments, which were performed at a minimum environmental pressure of 116 mmHg, the mask pressure was raised above that of the environment by 8 mmHg and he found that this procedure at a pressure-altitude of 43000 ft increased the arterial oxygen saturation as measured by an ear oximeter from 71% to 82%. In later experiments performed in 1942, Gagge showed that it was possible to breathe oxygen at a positive pressure of 20 mmHg and that pressure breathing at this level would maintain an arterial oxygen saturation of 82% at a simulated altitude of 50000 ft. In the same year, Bazett independently suggested the use of pressure breathing with oxygen as a means of increasing the altitude at which consciousness was unimpaired.

Although Gagge and Bazett's experiments were the first in which positive pressure breathing (the difference between the pressure in the respiratory tract and the pressure of the individual's immediate environment) was

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employed at a low environmental pressure, the manoeuvre had been studied in clinical medicine for many years previously. According to Barach, Martin and Eckman 1938 (19) the first report of the use of pressure breathing was that by Oertel who, in 1878, applied intermittent positive pressure breathing at a maximum positive breathing pressure of 8–15 mmHg in the treatment of severe asthma. Later, in 1897, Norton used the manoeuvre successfully in the treatment of a case of pulmonary oedema due to carbolic acid poisoning. Breathing at a positive breathing pressure of 5–10 mmHg was shown by both Poulton 1936 (233) and Barach, Martin and Eckman 1938 (19) to provide some relief in cases of acute pulmonary oedema due to left sided heart failure. Barach, Martin and Eckman 1938 (19) also studied the effects of continuous breathing at a positive breathing pressure of 2–6 mmHg upon normal subjects. In clinical medicine positive pressure breathing at these pressures was used in the treatment of acute pulmonary oedema, asthma and upper respiratory obstruction (15).

With the recognition of the value of positive pressure breathing as a means of decreasing hypoxia at altitudes above 40000 ft the physiological disturbances induced by this manoeuvre were subjected to intensive study (18). It was shown that the altitude gained by the use of pressure breathing was that to be expected from the increase of the partial pressure of oxygen produced by the manoeuvre. It was found that the maximum positive breathing pressure which healthy young men could tolerate for periods of several hours, using an oronasal mask alone, was 15 mmHg. The limit to the use of a mask was a positive breathing pressure of 30 mmHg when circulatory collapse occurred in twenty to thirty minutes at ground level and in a shorter period when pressure breathing was combined with hypoxia at reduced environmental pressure.

In addition to continuous pressure breathing other methods of increasing intrapulmonary pressure were investigated at this time. Intermittent positive pressure breathing in which the mask pressure was raised considerably during inspiration and allowed to fall to a low value during expiration was demonstrated to reduce the subjective difficulty of expiration which was experienced with continuous pressure breathing. The gain in oxygenation using intermittent pressure breathing at altitude was shown to be closely related to the mean mask pressure measured over the whole respiratory cycle and not to the maximum mask pressure (59). A more serious disadvantage of this manoeuvre, however, was the marked hyperventilation and hypocapnia which it induced (88). The value of voluntarily raising the interthoracic pressure during expiration was studied by Lilienthal and Riley 1943 (188). They showed that this manoeuvre, when correctly performed, would increase the maximum altitude at which useful consciousness was maintained when oxygen was breathed to about 44500 ft. They found, however, that the improved oxygenation was related to the mean intrathoracic pressure throughout the respiratory cycle and that in a large proportion of subjects the manoeuvre produced gross hypocapnia. As a result of these studies only continuous positive pressure breathing was used in high altitude flight.

During the Second World War continuous pressure breathing with a mask alone at a maximum positive breathing pressure of 15 mmHg was used for periods of several hours by the American Air Forces to maintain adequate

oxygenation at altitudes of up to 45000 ft (119). An important use of pressure breathing at a positive breathing pressure of 2 to 4 mmHg was the prevention of leakage of air into the mask at altitudes above 33000 ft where admixture of air with the oxygen delivered by the breathing equipment would result in serious hypoxia.

In his original study of pressure breathing Bazett proposed that counter-pressure should be applied to the trunk by means of a bag inflated to the same pressure as that delivered to the respiratory tract (30). He showed that the use of counterpressure removed the labour of breathing produced by pressure breathing with a mask alone. This proposal was rapidly exploited by the various groups working in this field and it became the basis of the pressure breathing equipment subsequently introduced to the Royal Air Force and the Royal Canadian Air Force (115). It was shown that the use of counterpressure to the trunk raised the positive breathing pressure which could be used for long periods without collapse to about 30 mmHg. Various investigators increased the area of the trunk covered by the counterpressure vest by extending the bladder to the lower abdomen and preventing it moving up by a pair of straps passed through the crutch (274). Drury, Henry and Goodman 1947 (82) showed that with adequate counterpressure to the whole trunk positive breathing pressures of up to 45 mmHg could be tolerated for ten to twenty minutes.

An important limitation to the time for which individuals could be exposed to altitudes above 30000 ft in addition to that imposed by hypoxia was known to be decompression sickness. Thus, although pressure breathing with or without counterpressure applied to the chest had been shown to be a practical method of preventing hypoxia at altitudes of up to 45000 ft, an individual using the equipment might well be forced to descend owing to the develop-ment of serious decompression sickness. Aircrew flying to these altitudes in unpressurized aircraft had to be, in fact, a specially selected group of indivi-duals who had a low susceptibility to decompression sickness. In certain circumstances denitrogenation by breathing oxygen before flight was also employed to reduce the incidence of decompression sickness at high altitude.

As, after the Second World War, aircraft were developed with pressurized cabins the circumstances in which pressure breathing might be used during flight underwent a marked change. Instead of being a method which allowed an aircraft without a pressure cabin to be flown routinely above 40000 ft, pressure breathing became an emergency procedure which was used to main-tain consciousness following failure of the pressure cabin at high altitude. As the heights to which aircraft could fly increased so the need for emergency protection against the effects of exposure to very high altitudes also increased, but the time for which protection was required diminished.

Bazett suggested in his early proposals that the application of counter-pressure to the lower limbs would be advantageous during pressure breathing (30). Counterpressure to this region would reduce the amount of blood displaced by the raising of intrapulmonary pressure and so decrease the cardiovascular disturbance produced by the manoeuvre. A further disadvan-tage to raising the positive breathing pressure to higher than about 30 mmHg was the severe discomfort which occurred in the face and neck when an oronasal mask was used to deliver the pressure to the respiratory tract.

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Pressure headpieces, by which the increase of pressure was applied to more or less of the head, were developed and used in later experimental procedures. Using a facepiece in place of a mask together with counterpressure to the trunk and lower limbs, Bazett (unpublished report, 1943) showed that a positive breathing pressure of 40 mmHg could be tolerated for long periods and that it would provide protection against severe hypoxia for an hour at a simulated altitude of 52 000 ft.

Later, in 1944, Henry and his colleagues, working at the University of Southern California, demonstrated that when adequate counterpressure was applied to the trunk and lower limbs subjects could tolerate positive breathing pressures of up to 60 mmHg for as long as thirty minutes. They exposed subjects using this equipment to simulated altitudes of up to 55 000 ft in a decompression chamber. By attaching inflatable sleeves to the trunk garment so that counterpressure was applied to both the upper and lower limbs as well as to the trunk, Henry, Greeley, Meehan and Drury 1944 (148) successfully exposed subjects to an absolute pressure of 60 mmHg (equivalent to an altitude of 58 000 ft) in a decompression chamber. The technique of applying counterpressure to the surface of the body by means of inflatable bladders was found by Henry to result in a bulky garment which greatly restricted mobility. An external system of bladders was adopted to apply counterpressure to the limbs. This employed the capstan principle which had been developed by Lamport, Hoff and Herington 1944 (180) as a method of applying counterpressure to the lower limbs in order to provide protection against accelerative forces. The limbs were covered by a close fitting layer of nylon fabric which was tensioned by the inflation of an external bladder which ran along the length of each limb and was connected by "figure of eight" tapes to the fabric.

The prototype suit employing the capstan principle was delivered to the United States Air Force Aero-Medical Laboratory in 1946 when Henry also joined the staff of this organization. The helmet and suit were progressively improved and it was shown that it would provide protection against hypoxia at simulated altitudes of up to 100 000 ft. The breathing system was designed so that at altitudes above 42 000 ft oxygen was delivered to the respiratory tract at an absolute pressure of 141 mmHg. The absence of pneumatic bladders encircling the limbs allowed sweat to pass through the nylon fabric and thus the heat load imposed by this garment was considerably less than that associated with the earlier forms of full pressure suit. In the first operational version of this suit (the capstan partial pressure suit) the trunk bladder was omitted and counterpressure was applied to the trunk as well as to the limbs by means of external capstans (162). The counterpressure provided by the capstan system was not uniform, however, and the suit in this form did not give full protection against the respiratory and circulatory stresses associated with positive breathing pressures above 50 mmHg (157) (53).

Further improvements were made to the suit in the United Kingdom, particularly in the pressure headpiece (Fig. 1-2) and it was adopted as an emergency garment for use in the test flying of aircraft at high altitudes (194). The experience gained thereby and from a series of sizing trials showed that the suit imposed serious limitations upon movement even in the uninflated state. Aircrew also complained of severe discomfort at positive breathing
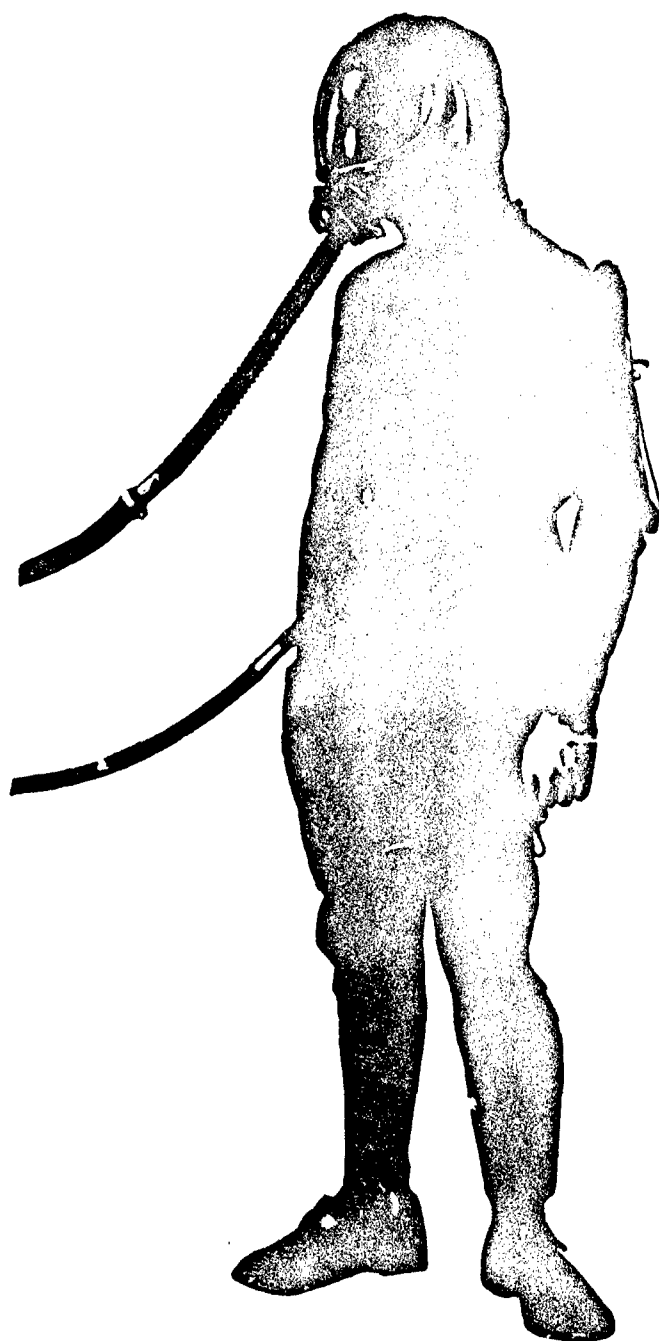
FIG. 1-2 The capstan partial pressure suit

pressures of the order of 80 mmHg (Ernsting and Holmes, unpublished observations, 1954). The difficulty of breathing experienced in the inflated suit was markedly reduced by the use of an inflated waistcoat beneath the trunk portion of the garment. In the later versions of the suit used operationally by the United States Air Force the original trunk bladder was refitted (288).

Although a full pressure suit (a garment which completely encloses the whole body and by means of which a pressure is applied evenly over the whole surface) had been used in unpressurized aircraft before 1939 this form of protective device was not used operationally during the Second World War. Further development of this concept continued, however, and with the improvements in fabric technology directly after the war, more satisfactory suits became available. The comfort of the uninflated suit was greatly improved by the use of lightweight fabrics and the minimum of metal parts and by the provision of an efficient system of ventilation beneath the impermeable layer of the suit. Ventilation was provided by passing dry, cool air over the surface of the limbs and trunk whilst oxygen was delivered to the respiratory tract by means of a face mask. A reasonable degree of mobility was attained in suits of this type when inflated at pressures of up to 150 mmHg gauge. The absolute pressure within these suits was maintained at between 226 and 180 mmHg when the pressure of the immediate environment fell below this level.

The improvements in comfort and mobility attained in modern full pressure suits (Fig. 1-3) led to the adoption of this type of suit by the United States Navy as an emergency protective device to be worn by the aircrew of aircraft flying to altitudes above 45 000 ft (122). A decade ago there was, therefore, a wide variety of devices either already available or under development for the protection of aircrew in the event of failure of the pressure cabin at high altitude. Many of these devices, were however, unsatisfactory in several respects and at that time an attempt was made to develop a rational philosophy with regard to the use of pressure suits in the Royal Air Force (255).

It is possible to divide the conditions in which personal pressure equipment may be employed in the event of the failure of the pressure cabin of an aircraft flying at high altitude into two broad categories: It may be used to provide short term protection to the wearer enabling him to descend to an altitude where protection is no longer required or to protect him for a long period and thereby allow the aircraft to remain at high altitude. The time for which protection is required may vary therefore from less than four minutes, the time taken to descend in a high performance interceptor aircraft from an altitude of 100 000 ft to below 40 000 ft, to about six hours, the time taken for a bomber or photographic reconnaissance aircraft to complete its mission. The nature and intensity of the physiological effects induced by an exposure to high altitude varies in an important manner with the duration of the exposure. The variability of these effects must be considered, therefore, in some detail before a satisfactory philosophy concerning the use of pressure clothing can be developed.

The physiological effects of failure of a pressure cabin may be divided into those related to the sudden change of cabin pressure and those produced by
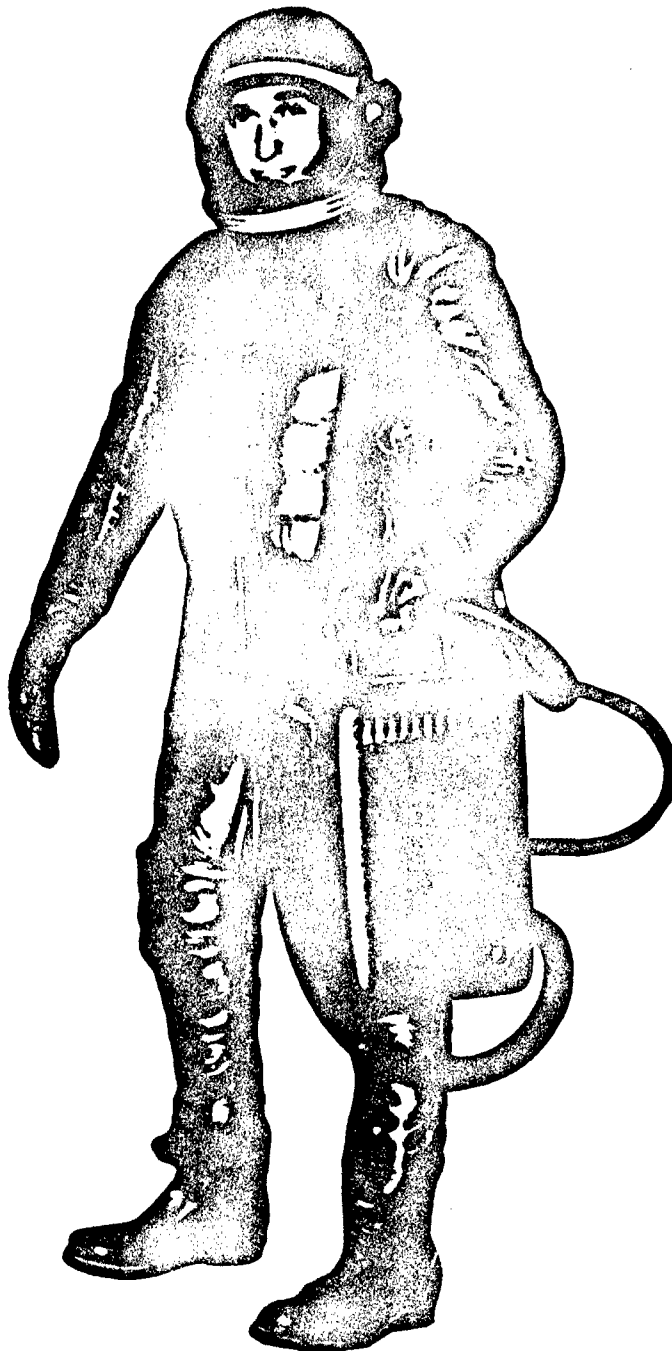
Fig. 1-3 A U.S. Navy full pressure suit
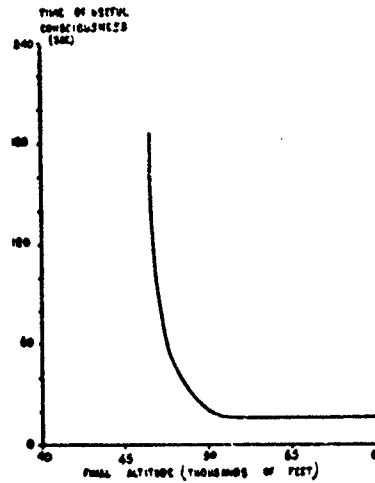
FIG. 1–4 The time of useful consciousness following rapid decompression to various altitudes whilst breathing 100% oxygen (Benzinger, 1943)
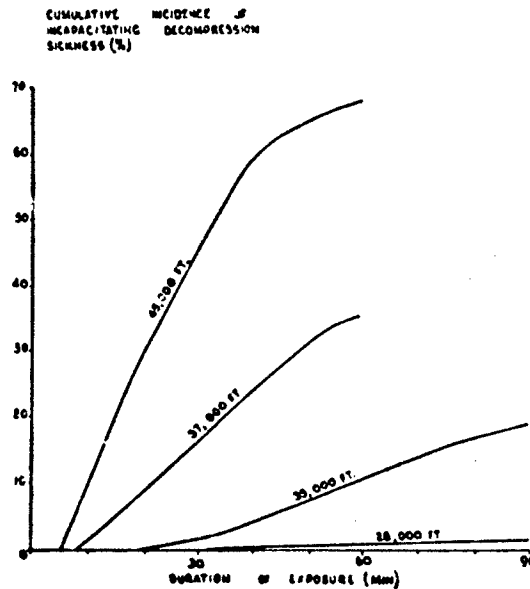
FIG. 1–5 The incidence of incapacitating decompression sickness during exposure to simulated altitudes of 28000 ft (2600 subjects, Fryer, 1962), 35000 ft (ninety subjects, Ferris *et al*, 1943), 37000 ft (1600 subjects, Colley, 1959) and 45000 ft (twenty-nine subjects, Ferris *et al*, 1943). In the last series of experiments the subjects were pressure breathing with oxygen at 20 mmHg

the low environmental pressure and temperature to which the crew are exposed following the failure. The effects of the reduction of pressure itself are due to the expansion of gas contained within the gastro-intestinal tract, the middle ear, the nasal sinuses and the lungs. Symptoms arise when the gas contained in these regions cannot escape freely to the environment. The commonest site for symptoms is the gastro-intestinal tract and although the the incidence of symptoms is extremely variable, they almost always arise immediately the pressure is reduced and are seldom seen at altitudes below 25 000 ft. Above 25 000 ft the incidence of abdominal symptoms increases as the altitude to which the subject is exposed increases. The symptoms vary from mild abdominal discomfort to severe colic which may be associated with vasovagal syncope (250) (Ernsting, personal observation).

The overall incidence of incapacitating symptoms due to the expansion of abdominal gas following decompression from 25 000 ft to above 40 000 ft in a group of 300 aircrew was 3% (Ernsting, unpublished observation). The expansion of the gases contained within the cavities of the skull does not give rise to any disturbance in normal subjects since the volumes of gas concerned are small in relation to the size of the passages connecting the cavities to the external environment. The respiratory tract differs from the other gas containing cavities of the body in that it normally contains a large volume of gas in relation to the size of the airways. Further, the lungs are relatively more susceptible to damage by over-distension than are other organs (144). Over a very wide range of rates of decompression, provided that the glottis is open, the alveolar gas can escape as it expands and no lung damage will occur. If, however, the rate and range of the decompression exceed certain limits the alveolar pressure will exceed the pressure of the environment to such an extent that the lungs are damaged by over-distension (279, 8, 195). Several cases of lung damage produced by rapid decompression have occurred in decompression chambers. In practical aviation however such decompressions have not occurred, although certain forms of breathing equipment do impose a high resistance to the outflow of gas from the respiratory tract during a decompression and could, in certain circumstances, produce dangerous overdistension of the lungs (99).

Exposures to the low pressure which characterizes the environment at high altitude can give rise to three distinct physiological effects, viz. hypoxia, decompression sickness and vaporization of tissue fluid. When the inspired gas is 100% oxygen the alveolar oxygen tension falls below the normal sea level value on exposure to altitudes above 33 700 ft, although very little impairment of performance due to hypoxia ensues until the altitude of the exposure exceeds 40 000 ft. The intensity of the hypoxia increases markedly as the altitude exceeds 40 000 ft and above 50 000 ft unconsciousness ensues fifteen to sixteen seconds after the beginning of the exposure (Fig. 1–4). When the altitude exceeds 50 000 ft consciousness is impaired if the duration of the exposure exceeds five seconds (197) (Ernsting, unpublished observation). Thus in practical aviation even when the duration of an exposure to an altitude above 40 000 ft is short, severe hypoxia will occur unless the absolute pressure within the respiratory tract is maintained in excess of 130 mmHg.

A prolonged exposure to an altitude in excess of 25 000 ft will, in the majority of subjects, give rise to one or more of the manifestations of decompression

sickness. Although the existence of this condition following decompression from pressures above one atmosphere was clearly recognized by the middle of the nineteenth century and although Bert 1878 (34), Hill (1912) (156) and Haldane 1920 (135) described clearly the mechanism of caisson disease the existence of a similar condition at high altitude was not generally recognized until 1939 (7). Both Henderson 1917 (143) and Haldane (138), however, recognized that decompression sickness can occur at low pressure and in fact Jongbloed 1931 (166) and Barcroft, Douglas, Kendal and Margaria 1931 (35) gave clear descriptions of the "bends" occurring at low environmental pressures. It was not until 1938 that a case of paraplegia occurring at reduced barometric pressure and cured by recompression to ground level was described (41).

- With the beginning of the Second World War and the ascent of aircraft routinely to altitudes above 30000 ft decompression sickness was studied intensely by many groups of investigators (118). The incidence of incapacitating decompression sickness rises with increase of the altitude and of the duration of the exposure (Fig. 1-5). Thus in a series of two-hour exposures to various simulated altitudes, the incidence of serious decompression sickness increased from 2% at 30000 ft to 24% at 38000 ft (104). Even moderate exercise greatly increases the incidence of decompression sickness (106). It is very rare, however, for decompression sickness to arise immediately on reduction of the environmental pressure. In practice the incidence of incapacitating decompression sickness in the initial five minutes of an exposure to reduced pressure is negligible ((256), (105), (127), Ernsting, personal observation). When the duration of the exposure to an altitude greater than 30000 ft exceeds ten minutes, incapacitating decompression sickness may occur and when the duration of an exposure is measured in hours, the incidence of serious symptoms due to this condition is very high.

Apart from maintaining the immediate environmental pressure at a value greater than 280 mmHg absolute, the incidence of decompression sickness may be reduced by the selection of relatively unsusceptible subjects or by removing nitrogen from the tissues by breathing 100% oxygen before exposure to reduced environmental pressure. Although both these procedures were used in the Second World War and more recently in test flying, neither of them is acceptable under modern operational conditions. Thus, if decompression sickness is to be avoided following failure of the pressure cabin at high altitude either the duration of the exposure to altitudes greater than 30000 ft must be short, less than five minutes, or the pressure of the aircrew's immediate environment must be at least 225 mmHg absolute.

When the total pressure in a tissue is less than the vapour pressure of the tissue fluid at the local temperature, the fluid will vaporize. Thus animals exposed to a pressure of 30 mmHg absolute develop vapour-thorax and gas bubbles within the circulation (50). Such extreme effects do not occur in practical aviation since protection against hypoxia demands that the absolute pressure within the respiratory tract and hence the abdomen and circulation shall be maintained at a value greater than 130 mmHg. Exposure of peripheral parts of the body, for example the hands, to an environmental pressure of the order of 40 mmHg absolute or less results in vaporization of tissue fluid in regions such as the dorsal tendon sheaths Ernsting (97). In these experi-

BAROMETRIC PRESSURE (mm Hg)

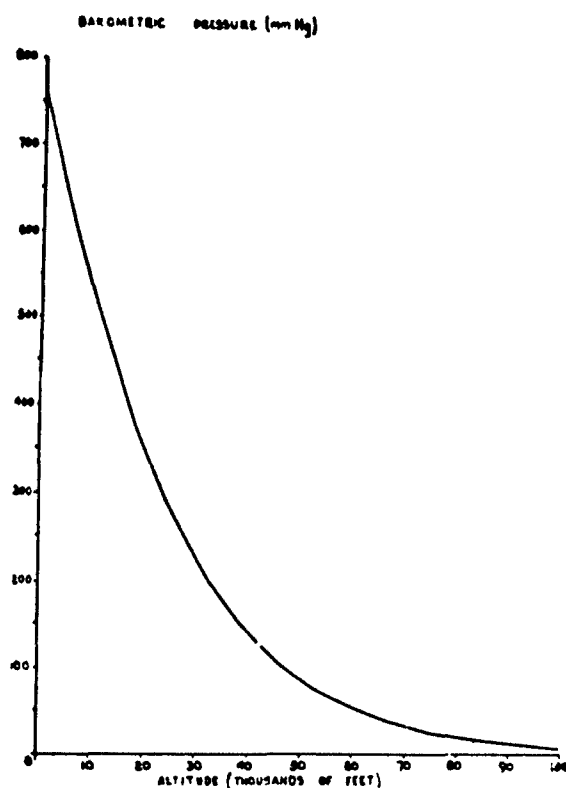FIG. 1-6 The relationship between barometric pressure and altitude as defined by the International Civil Aviation Organisation and the U.S. Standard Atmosphere

ENVIRONMENTAL TEMP (°C)

MAXIMUM
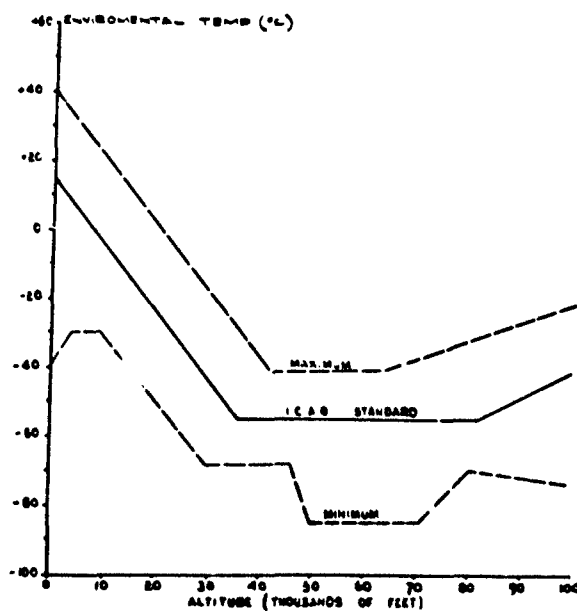
ICAO STANDARD

MINIMUM

ALTITUDE (THOUSANDS OF FEET)

FIG. 1-7 The variation of the temperature of the atmosphere with altitude. The standard curve is that defined by the International Civil Aviation Organisation

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mental exposures there was no impairment of function during and subsequent to pressures of the order of 35 mmHg absolute lasting two to three minutes. Thus although it is undesirable that any portion of the body should be exposed to environmental pressures lower than 47 mmHg absolute, no serious impairment of performance or damage to a peripheral region such as a hand or foot has been observed during an exposure limited to a few minutes.

Failure of the pressure cabin of an aircraft is frequently associated with a reduction of the environmental temperature as well as a fall of pressure. Further, following escape at high altitude a pilot is directly exposed to the low temperature of the atmosphere. The temperature of the earth's atmosphere falls progressively with increase of altitude until at a height of about 40000 ft it is of the order of $-55°C$ (Fig. 1–7). From 40000 ft to 80000 ft the temperature is fairly uniform and, depending upon the season of the year and the latitude, it varies between $-45°$ and $-90°C$. The degree of cooling of a body which results on exposure to low temperature depends not only on the temperature difference between the body and the surrounding air but also on the degree of air movement. There may be considerable air movement around the pilot following the failure of a hatch or window in the wall of the pressure cabin of his aircraft. Exposure to such low temperatures has both local and general physiological effects. The local effects which arise primarily in exposed regions such as the face and hands consist of cooling with impairment of function followed by tissue damage, frostbite. Frostbite of exposed skin occurs within a few minutes at temperatures of the order of $-40°C$ and below. The general effects of exposure to low temperature consist of progressive reduction of mental and physical efficiency followed by unconsciousness and death: These general effects only arise when the exposure exceeds several minutes.

The time course of the physiological effects is influenced markedly by the clothing which the individual is wearing. Thus an aircrew member wearing normal flying clothing including an oxygen mask and gloves will not suffer any serious damage or show any gross loss of efficiency during an exposure to the lowest temperature conditions which may be encountered at high altitude provided that the duration of the exposure is limited to about ten minutes. Exposure to such conditions beyond this time will, however, result in gross peripheral cold injuries and a progressive impairment of the ability to perform any useful task. Protection against the effects of exposure to a low temperature environment involves the provision of insulating material between the skin and the surrounding atmosphere and the supply of heat to the body from an external source. The heat can be supplied either by means of electrically heated clothing or by distributing hot air over the surface of the skin beneath the insulation layer of the clothing. It must be distributed in such a manner that thermal comfort is maintained and that the face, hands and feet are adequately protected.

It is apparent from these considerations that the nature of the effects of an exposure to low barometric pressure and temperature depend not only upon the absolute pressure and temperature but also upon the duration of the exposure. However extreme the altitude and temperature conditions, provided that the duration of the exposure is relatively short the only serious physiological disturbance will be hypoxia. If, however, the exposure is

prolonged, then both decompression sickness and cold injuries will occur in addition to hypoxia. The exposure may be considered to be of short duration if the time spent above 40000 ft does not exceed five minutes and if descent to lower altitudes is carried out as rapidly as possible. Even if the exposure to altitudes above 30000 ft is as long as ten minutes the incidence of decompression sickness and serious tissue damage due to cold will be relatively low. Thus the physiological requirements for protection following loss of cabin pressurization vary markedly with the duration of the exposure. When immediate descent can be carried out following depressurization and the exposure to altitudes above 30000 ft does not exceed ten minutes, it is only necessary to prevent hypoxia. If, however, the duration of the exposure to high altitude is longer than ten minutes, protection must be provided against decompression sickness and the effects of extreme cold as well as hypoxia.

Protection against all the physiological hazards which arise following loss of cabin pressurization at high altitude requires the use of a full pressure suit. It is only with such a garment that the absolute pressure of the immediate environment may be kept at such a level that decompression sickness will not occur. Further, it is simpler to provide and distribute the heat which is required to maintain thermal equilibrium in these conditions when the whole body is enclosed within a gas tight bag. It is very desirable, however, that the restriction imposed upon an aircrew member by the equipment which he wears in order to obtain protection against certain emergency conditions which are unlikely to occur should be minimal. A full pressure suit which meets all the physiological requirements for protection against the effects of exposure to high altitude even if well designed does, however, impose a considerable reduction in mobility and comfort upon the wearer. In practice, therefore, there is a certain degree of conflict between the need for comfort and full mobility during routine flight and the restrictions imposed by the equipment used to provide protection against the effects of failure of the pressure cabin at high altitude. When the duration of the exposure to high altitude is long then adequate protection can only be provided with a full pressure suit. Although a full pressure suit will obviously also provide adequate protection against a short exposure to high altitude, the protection which it gives is actually greater than the situation demands.

Hypoxia, which is the only serious hazard when the duration of the exposure to altitudes above 40000 ft does not exceed five to ten minutes may be prevented by positive pressure breathing with oxygen. When protection is required at an altitude at which the positive breathing pressure will exceed 30 mmHg, some form of body counterpressure is also required. These considerations suggest a rational philosophy for the use of pressure clothing. Thus when the duration of an exposure to high altitude is prolonged adequate protection can only be provided by a full pressure suit. If, however, immediate descent to low altitude can be undertaken following loss of cabin pressure, then pressure breathing with a limited degree of counterpressure applied to the body will prevent hypoxia. Further, the restrictions associated with wearing partial pressure clothing in normal flight are considerably less than those imposed by a full pressure suit.

Roxburgh, Howard, Dainty and Holmes 1953 (255) advanced the argument that since the time for which pressure breathing could be used at

high altitude was severely limited by the hazards of decompression sickness and cold injury, the degree of counterpressure which was given by, for example, the capstan partial pressure suit was considerably greater than was essential during the short period for which the suit could be used. They suggested that since this time was severely restricted the degree of counterpressure afforded by the pressure clothing could be reduced to the minimum which would give adequate protection against the respiratory and circulatory effects of the breathing pressures required to prevent hypoxia. Roxburgh. Howard, Dainty and Holmes (255) (1953) suggested that the counterpressure applied by the standard R.A.F. pressure breathing waistcoat and an anti-g suit which covered the lower abdomen and most of the lower limbs, used in conjunction with a pressure helmet, would suffice at positive breathing pressures of up to 80 mmHg for several minutes (Fig. 1–8). Later experimental studies by Badger, Ernsting and Roxburgh, (1956) (13) showed, however, that the counterpressure given to the abdomen by this combination was inadequate and that seven out of ten subjects were unable to complete a seven and a half minute exposure to a positive breathing pressure of 78 mmHg. Each of the seven subjects who failed to complete the exposure exhibited a circulatory collapse which had the clinical features of vasovagal syncope (187). It was found that, when the effectiveness of the counterpressure applied to the trunk by this combination was improved by encircling the whole trunk by an inflatable bladder, none of the experimental subjects collapsed when exposed to a positive breathing pressure of 78 mmHg for seven and a half minutes. This bladder garment which encircled the whole trunk became the basic garment of a series of partial pressure assemblies.

In 1954 positive pressure breathing with oxygen was adopted by the Royal Air Force as the method of providing short duration protection against hypoxia following either loss of cabin pressurization or escape at altitudes between 40000 ft and 100000 ft. In order to safeguard against hypoxia the maintenance of a certain minimum absolute intrapulmonary pressure is essential and previous studies suggested that this minimum lay between 120 and 141 mmHg absolute. To maintain this intrapulmonary pressure above an altitude of 40000 ft demands a degree of positive pressure breathing, the extent of which is a function of the altitude concerned (Fig. 1–9). Thus the maximum positive breathing pressure required in order to afford protection to an altitude of 100000 ft lay between 112 and 133 mmHg.

The work described in this thesis was undertaken in order to determine the physiological disturbances induced by the high positive breathing pressures required at altitudes greater than 50000 ft and to find the degree of counterpressure necessary to reduce the effect of each of these disturbances to a level which was thought to be acceptable during a short duration exposure. For this purpose the problems associated with high pressure breathing were divided into four groups:

Disturbances in the head and neck – The actual application of gas under pressure to the mouth and nose induced various changes in the head and neck. The physiological limitations to the use of an oronasal mask for this purpose were studied in detail and the parts of the head and neck to which counterpressure should be applied were determined.

Disturbances of respiration – Pressure breathing, particularly at positive

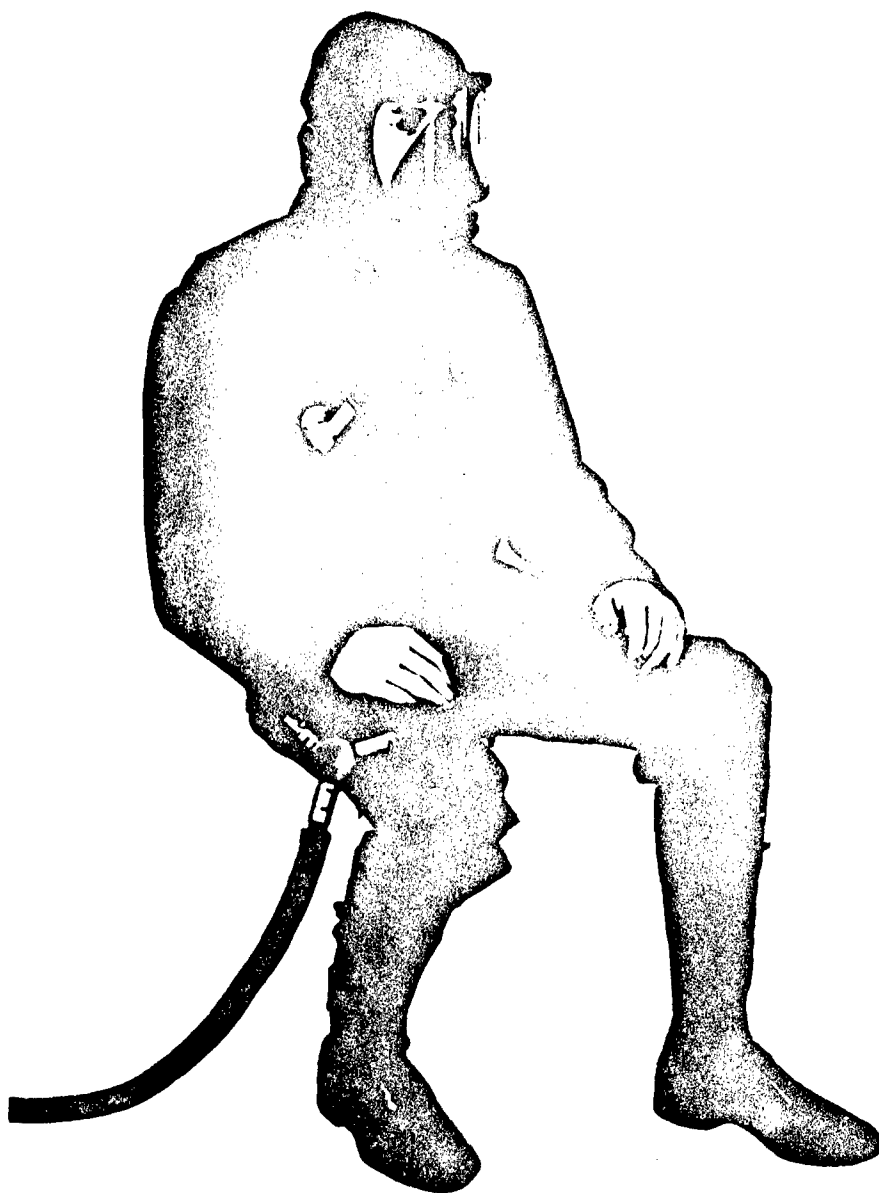FIG. 1-8 A standard R.A.F. pressure breathing waistcoat and anti-g suit
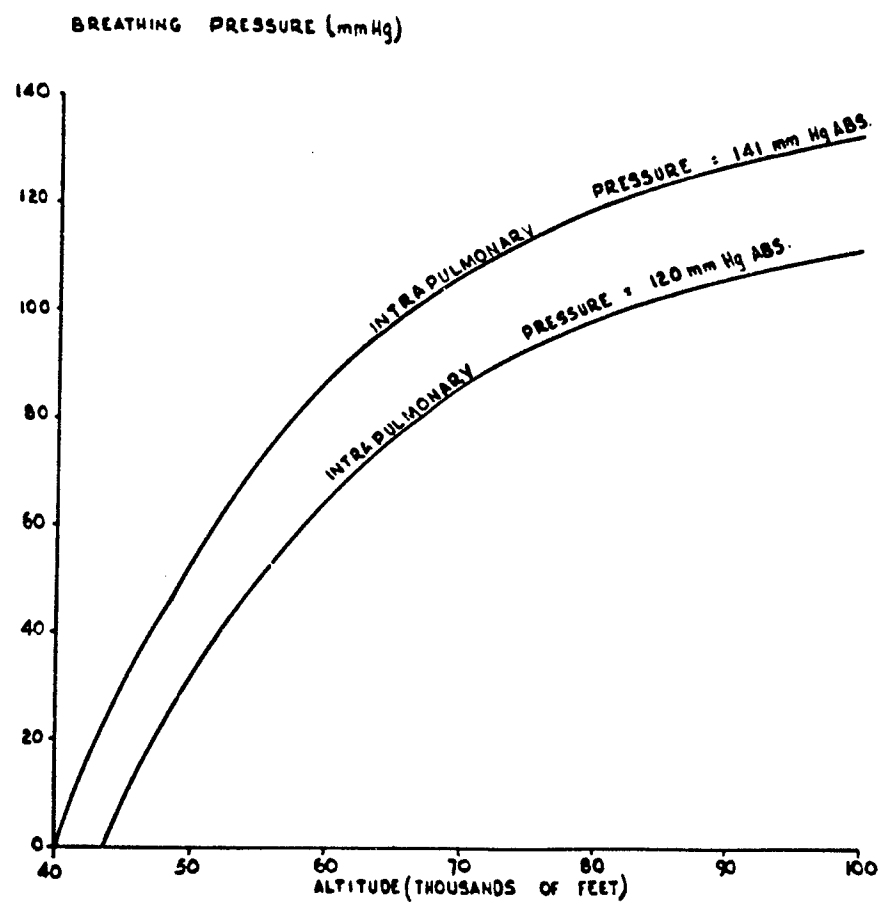
BREATHING PRESSURE (mmHg)

FIG. 1-9 The relationship between positive breathing pressure and altitude required to maintain intrapulmonary pressures of 120 and 141 mmHg absolute

18

breathing pressures above 30 mmHg, produced gross disturbances of the mechanics of respiration and of respiratory gas exchange. The efficiency of various degrees of counterpressure applied to the trunk in overcoming these disturbances was determined and an efficient trunk counterpressure garment, the pressure jerkin, was evolved.

Disturbances of circulation – The rise of intrathoracic pressure associated with pressure breathing produced cardiovascular changes of which the most important was the displacement of blood from the central part of the circulation into the limbs. The time for which pressure breathing could be performed at a given positive breathing pressure was limited primarily by these cardiovascular disturbances. It was shown that these effects could be reduced by the application of counterpressure to the lower limbs by means of an anti-g suit and virtually eliminated by the application of counterpressure to both the upper and lower limbs.

Hypoxia and pressure breathing – The influence of hypoxia upon the circulatory responses to pressure breathing was investigated since this determined in part the minimum absolute intrapulmonary pressure which could be used during pressure breathing at high altitude. The interaction between hypoxia and the circulatory load imposed by pressure breathing was studied in order to determine the most satisfactory compromise between the positive breathing pressure and absolute intrapulmonary pressure for adequate protection at a given altitude above 40000 ft.

These physiological studies which were performed in conjunction with a parallel programme of development of personal equipment resulted in the formulation of three assemblies, the purpose of which was to provide short duration protection against hypoxia at reduced environmental pressure. The protection afforded by each of these assemblies was assessed by exposing a series of subjects wearing the equipment to reduced environmental pressure in a decompression chamber. The assemblies, which were based upon the pressure jerkin, were:

The pressure breathing mask, pressure jerkin and anti-g suit assembly – Pressure breathing with this assembly was limited to a maximum positive breathing pressure of 60 mmHg and provided protection against hypoxia at altitudes of up to 56000 ft.

The partial pressure helmet, pressure jerkin and anti-g suit assembly – This combination was used at positive breathing pressures of up to 100 mmHg and provided protection at altitudes of up to 70000 ft.

The partial pressure helmet, pressure jerkin with sleeves (the "arm jerkin") and anti-g suit assembly – This assembly afforded protection against the effects of pressure breathing at positive breathing pressures of up to 140 mmHg and was used at altitudes of up to 100000 ft.

Each of these partial pressure assemblies has been introduced into the Royal Air Force as a means of providing short duration protection against the effects of loss of cabin pressurization at altitudes above 40000 ft.
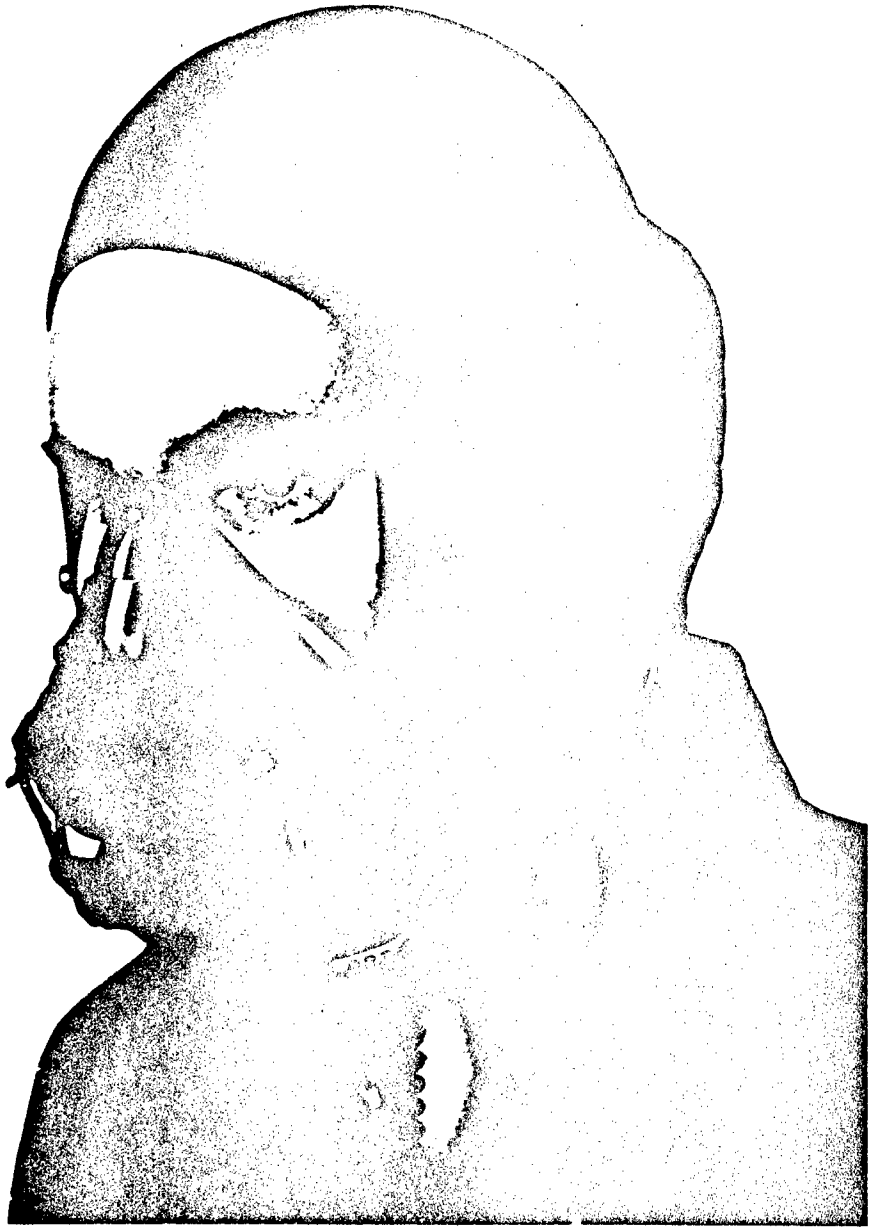
FIG. 2-1 An R.A.F. type Poronasal mask

EXPERIMENTAL METHODS

PRESSURE BREATHING EQUIPMENT

In the course of many of the experiments carried out in this investigation special equipment was used to deliver gas to the respiratory tract at a pressure greater than that of the environment and to apply counterpressure to various parts of the trunk and lower limbs. Some of these items were standard R.A.F. equipment; other items were, however, developed during the investigation and have subsequently become standard service equipment.

Pressure Breathing Mask – The R.A.F. type P oronasal mask (Fig. 2-1) has a reflected edge seal of thin rubber. The line of reflection lies over the bridge of the nose, in the nasolabial sulci and the mentolabial sulcus. The body of the mask, which carries the inlet and outlet valves and a microphone is supported by a rigid exoskeleton to which the harness is attached. The mask is held against the face by a pair of chains, each of which passes from a toggle bar attached to the exoskeleton to the side of a flying helmet which is worn on the head. The length of either chain of the harness may be adjusted by means of a turn-buckle incorporated in the attachment point to the helmet. A special feature of the harness is that the toggle bar has two positions. Rotating the toggle downwards through approximately $135°$ decreases the horizontal distance between the mask exoskeleton and the attachment points on the helmet by 2 cm. Thus by downward rotation of the toggle a mask adjusted for comfort may be forced on to the face so that no gross leak will occur when the gas pressure within the mask cavity is increased. The mask harness was normally adjusted with the toggle in the "low" pressure position so that no leakage occurred when the pressure in the mask was 5 cm of water greater than that of the environment. After rotation of the toggle harness to the "high" pressure position, the leakage of gas between the face and the mask at a positive breathing pressure of 60 mmHg did not usually exceed 10 litre/min.

Pressure Headpiece – The pressure headpieces used in the present study did not apply pressure to the whole head since, when gas pressure is applied evenly over the head, some form of downward restraint is required in order to prevent the helmet rising when it is inflated. In order to avoid this lift the helmets used in this study are designed so that no pressure is applied to an area of the crown of the head which is approximately equal to the cross sectional area of the neck.

Several forms of partial pressure helmet were used in this investigation. The basic construction of these helmets is very similar (Fig. 2-2). Each consists essentially of a double layer rubber bladder which envelops the head and upper part of the neck. The inner layer of the bladder lies closely against the skin of the head and neck. This layer has a free edge which is positioned around the periphery of the face, passing across the forehead about 1 cm

OUTER   FABRIC   RESTRAINER

FACE
SEAL

GAS BLADDER
OVER BACK
OF HEAD

VISOR

MASK

FACE
SEAL

FREE EDGE OF
FACE SEAL

FIG. 2-2 A diagrammatic sagittal section of a partial pressure headpiece showing the
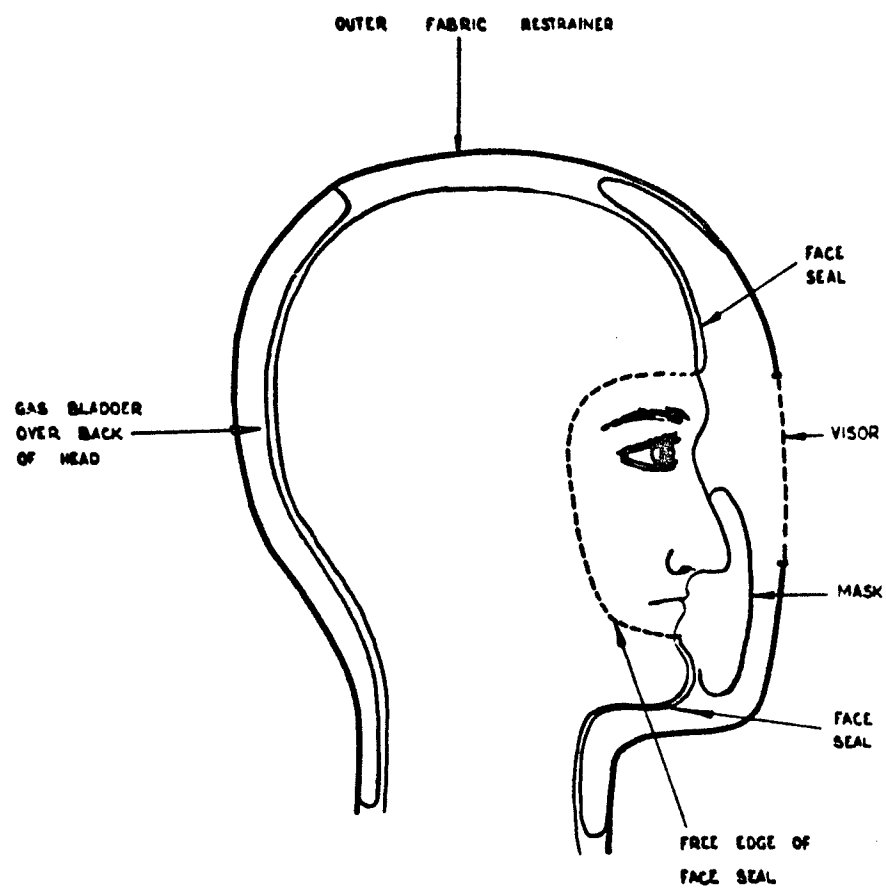gas bladders

22
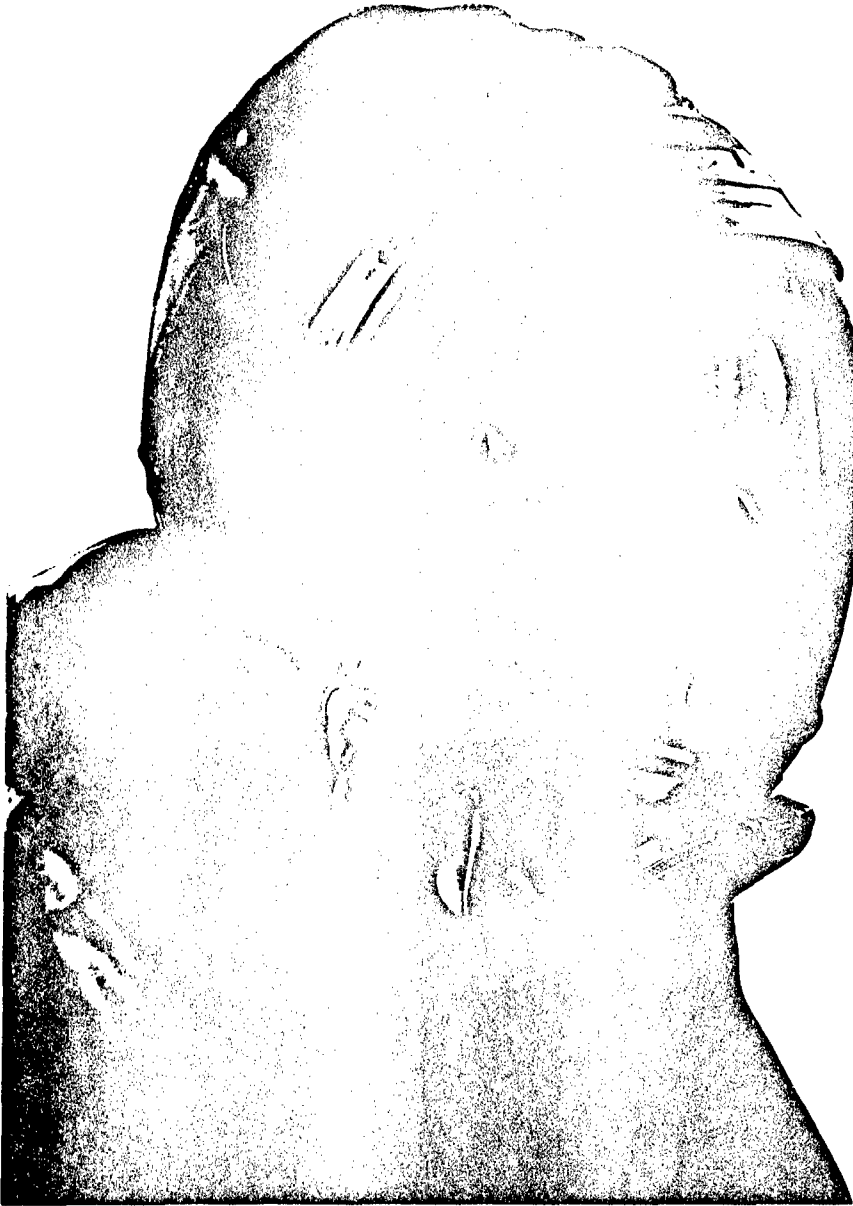
FIG. 2-3 Pressure helmet with fixed visor

FIG. 2-4 Pressure helmet with movable visor

above the supraorbital ridges and then down either side of the face just posterior to the lateral margin of the orbit and across the chin below the lower lip. An important feature of this face seal is that when the helmet is fitted to the head a certain degree of tension must exist in the free edge of the seal forcing it against the skin. From the free edge of the face seal the inner layer of the bladder passes back over the sides of the head to the entry slit where it is reflected off the head to become the outer layer. Upwards the inner layer is reflected off the crown of the head to become the outer layer so that an area of the crown remains uncovered. Similarly the inner layer covering the upper part of the neck is reflected off the neck and becomes the outer layer. The outer layer of the bladder is continuous over the head and neck except for an area in front of the face. Here it is attached to the margins of the visor aperture in the outer shell. In one type of helmet which was used in this investigation (the Type D, Fig. 2-3) the visor is permanently attached to the margins of this aperture. In the other form of helmet used the visor aperture is normally open and the visor drops automatically into place when decompression occurs (Fig. 2-4). In the partial type of headpiece pressure is applied to the mouth and nose and to that area of the face delineated by the free edge of the face seal directly by the gas in the helmet. Pressure is applied to the remainder of the head and neck through the inner layer of the rubber bladder. There is no direct communication between the gas within the bladder of the helmet and that in the auditory meati.

The standard forms of partial pressure helmet used in this investigation were fitted with oronasal masks. In the fixed visor helmet (Type D) this mask does not fit the face closely and its purpose is to deflect the moist expired gas away from the inner surface of the visor. A well fitted oronasal mask is, however, an essential component of a helmet with an opening visor in order that the wearer may breathe oxygen enriched gas mixtures whilst the visor is open. When an open visor helmet is used any leakage of air into the mask from the visor compartment will give rise to a reduction of the inspired oxygen tension. Thus it is essential that the mask of the helmet should seal against the face. The presence of a significant inboard leak was detected in this study prior to an exposure to reduced barometric pressure by continuously monitoring the nitrogen concentration in the gas within the mask compartment whilst oxygen was breathed. The fit of the mask was considered adequate when the nitrogen concentration in the mask cavity remained at less than 5"₀ after breathing oxygen for several minutes.

With correct fitting the leakage of gas from a partial pressure helmet when it was pressurized was generally low. Experience showed that it was relatively easy to achieve a leak of less than 10 litre/min. when the pressure within the helmet was between 50 and 100 mmHg (gauge). Whilst such a leak was perfectly acceptable for the normal use of a helmet the leakage amounted to 50–100% of resting respiratory minute volume. Thus a modified helmet was developed specifically for measurement of respiratory function. The oronasal mask was removed from a Type D helmet and replaced by a mouthpiece which was connected by a short length of flexible hose to a 2.5 cm diameter brass pipe sealed into the outer layer of the helmet bladder below the helmet visor. The appropriate valve system was mounted on this tube outside the

helmet. The bladder of the helmet was inflated by a system separate from that which supplied the mouthpiece. In this manner, with the subject's nostrils occluded by a spring clip, it was possible to obtain a leak-free connection with the respiratory tract, whilst the appropriate counterpressure was applied to the face, head and neck.

The Mask and Helmet Valve System – Essentially the same valve system was fitted to all the standard masks and pressure headpieces used in this study. This consisted of a simple inlet non-return valve (Fig. 2–5) through which gas entered the mask cavity during inspiration, and a compensated outlet valve. The latter maintained in the mask cavity a pressure equal to that existing in the breathing system upstream of the inlet non-return valve when the latter pressure was greater than that of the environment. A compensated outlet valve consists of a valve plate which is held in the closed position by a light spring. A thin rubber diaphragm lies beneath the valve plate and forms one boundary of the compensating chamber. This chamber is in communication with the inlet tube of the mask by way of the compensating tube. In this manner the pressure existing in the inlet tube of the mask is applied to the external surface of the outlet valve, acting upon an area equal to that of the port of the valve. Thus the outlet valve cannot open until the pressure within the mask cavity exceeds that in the inlet tube. The presence of an inlet non-return valve is essential to the operation of a compensated outlet valve since, when the mask is connected to an oxygen regulator, an expiratory effort can only raise the mask cavity pressure above the inlet tube pressure when such a valve is present. Thus when gas at a pressure greater than that of the environment is delivered to the inlet tube of the mask or headpiece this pressure is applied to the gas in the respiratory tract. During inspiration the pressure in the mask cavity is reduced and gas flows through the inlet valve. The pressure upstream of the inlet valve is, however, greater than that in the mask cavity so that the outlet valve remains closed. During expiration the mask cavity pressure is increased above that in the inlet tube to the mask so that the inlet valve closes and the outlet valve is opened.

The Pressure Breathing Waistcoat and the Pressure Jerkin – In the course of the development of a system which would allow the use of breathing pressures of up to 130 mmHg several forms of respiratory counterpressure were used. A standard R.A.F. garment, the pressure breathing waistcoat, was used in certain preliminary experiments. The waistcoat (Fig. 1–8) consists of a rubberized fabric bag which covers the thorax. The lower border of this garment extends to about the lower edge of the rib cage, and when inflated it applies pressure to most of the thorax. It rapidly became obvious, however, that more complete respiratory counterpressure than that given by the waistcoat was necessary. A bladder garment, the pressure jerkin, which provided counterpressure to the whole trunk, was therefore developed.

It was decided that a gas-containing bladder encircling the trunk was the most effective method of applying counterpressure to this region. The garment consisted of a loose rubberized fabric bag enclosed within a non-extensible terylene outer layer. This form of construction ensured that the pressure applied to the surface covered by the inner layer of the garment was equal to the pressure of the gas within the bladder. A simple garment incorporating such a bladder was made and the efficiency and comfort of it

INLET NON-RETURN VALVE

MASK CAVITY

EXPIRATORY PORT

OUTLET VALVE

COMPENSATING CHAMBER

FLEXIBLE DIAPHRAGM

COMPENSATING TUBE

INLET TUBE

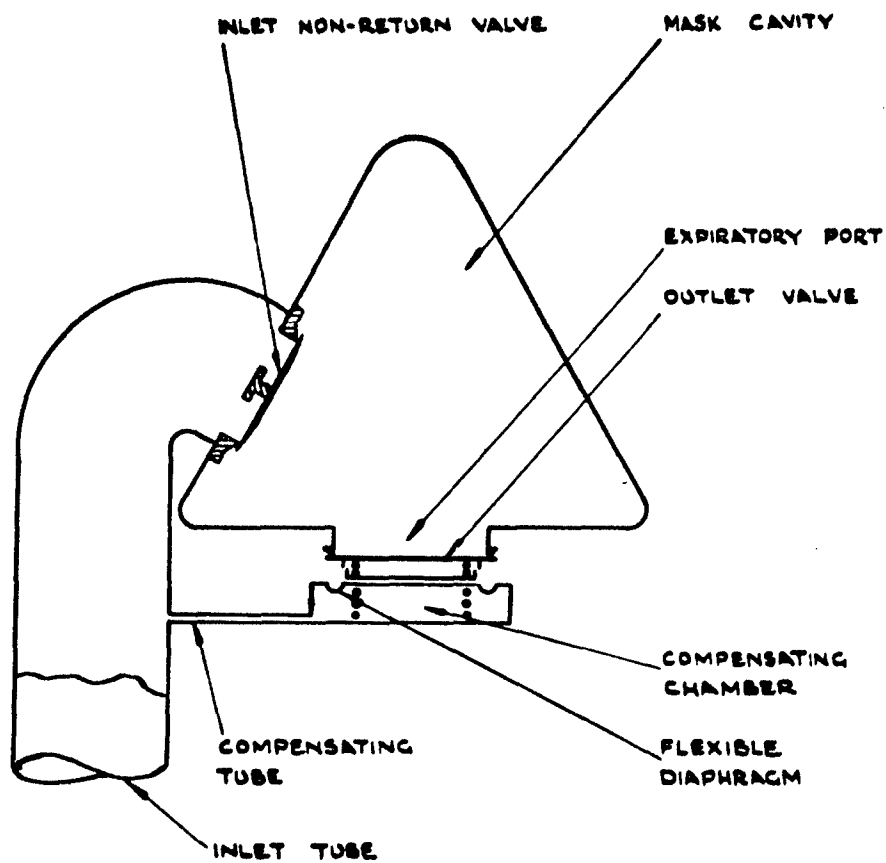FIG. 2-5 A diagram of the standard mask valve system which consists of an inlet non-return valve and a compensated outlet valve

was assessed by exposing several experienced subjects to pressure breathing at ground level whilst wearing the garment and a pressure helmet. As a result of this assessment several modifications to the basic garment were suggested and a new sample was manufactured. In this manner a series of garments were designed and made, each incorporating features which tests on previous garments had suggested would improve the comfort and efficiency of the clothing. Whilst some of those features were dictated primarily by the environment in which the garment was to be used, an important group of them was related directly to the function of the garment as a means of applying counterpressure to the trunk. The problems associated with the pressurization provided by a garment can be considered on a regional basis:

The Neck – In early prototype garments the bladder was brought close to the root of the neck in order to ensure that counterpressure was applied to as much of the surface of the trunk as was possible. During pressure breathing, however, compression of the neck occurred and gave rise to discomfort or even frank pain. In later versions of the pressure jerkin the neck line was lowered and the ballooning of the outer restraining layer of the garment reduced by using less extensible material in its construction.

The Armhole – The design of the armhole of the pressure jerkin was dictated by a desire to maintain upper limb mobility when the garment was pressurized. The front margin of the armhole was cut away until there was no significant restriction of abduction at the shoulder joint. In order to ensure that the hands could be elevated above the head the outer border of the garment over the top of the shoulder was taken medially to cross the clavicle at the junction between the outer and middle thirds. Only when the outer part of the shoulder was uncovered could the upper limbs be elevated freely whilst the garment was inflated.

The Scrotum – Early versions of the trunk bladder garment were fitted with a tail piece which, when the garment had been donned was brought forward between the thighs and attached to the lower end of the front of the trunk bladder. This tail piece was not inflated and severe testicular pain was common during pressure breathing with this arrangement. Even inflation of the crutch piece did little to decrease the incidence of testicular pain. When this version of the garment was inflated it tended to move backwards in relation to the pelvis and thighs so that the scrotum was forced against the front part of the crutch piece. This situation arose because, whilst the pressure in the bladder was distributed evenly over the sacral region and the buttocks, the holes for the passage of the lower limbs greatly reduced the front area of the pelvis to which pressure was applied. This unequal distribution of pressure over the pelvic region was eliminated by reducing the area of the posterior surface of the pelvis to which pressure was applied. The bladder of the garment was obliterated over a saddle shaped region which lay over the sacrum centrally and the medial third of each buttock laterally (Fig. 2–6). With this modification there was no movement of the garment on the pelvic region when it was inflated and testicular discomfort did not occur during pressure breathing.

Inguinal and Femoral Canals – In early forms of the trunk garment the bladder ended at the level of the groin so that when it was inflated the inguinal and femoral canals were unsupported. On several occasions subjects
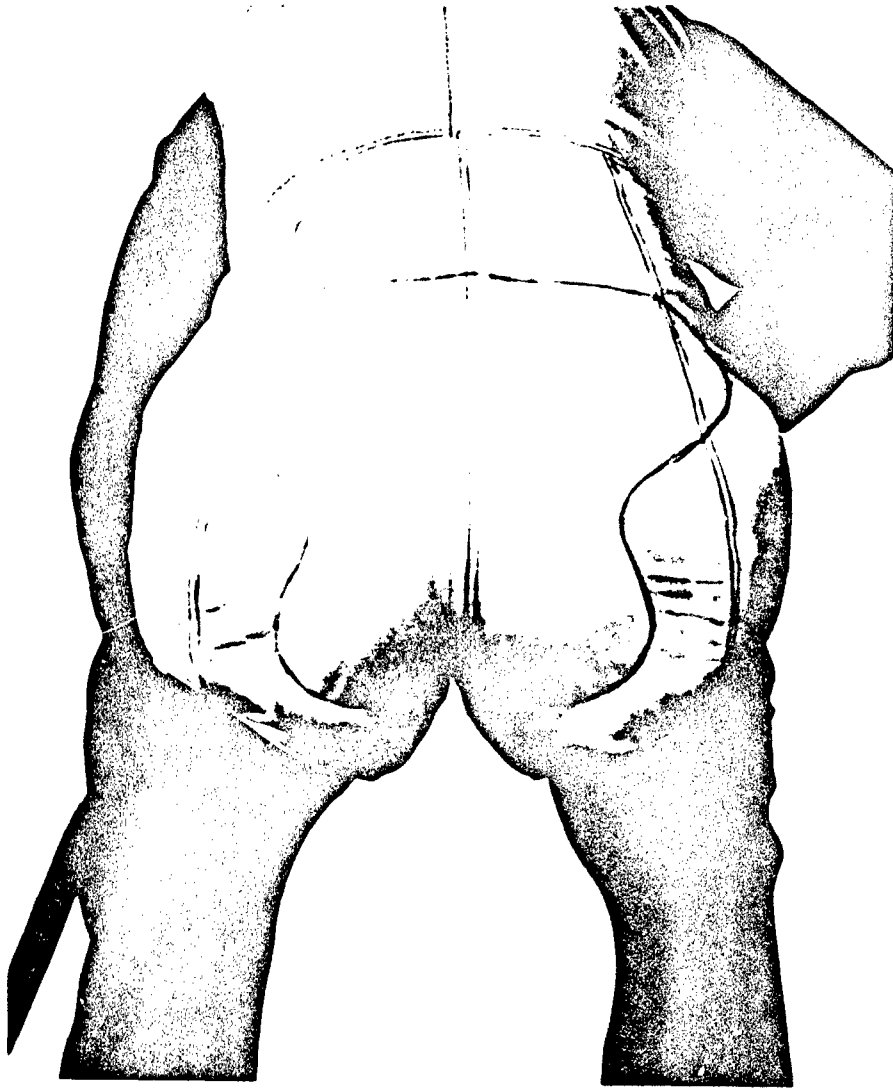
Fig. 2-6 Pressure jerkin showing uninflated saddle shaped region

complained of discomfort in the inguinal region during pressure breathing at 80 mmHg with this type of garment. The high intra-abdominal pressure produced by pressure breathing might induce a herniation of the abdominal contents through either the inguinal or the femoral canal. Although in the normal subject the risk of herniation was considered negligible, this procedure might well induce a hernia in a subject who had a congenital defect such as a patent processus vaginalis. In order to ensure adequate pressurization of the inguinal and femoral canals it was found that the lower edge of the bladder of the garment had to be carried completely around the upper thighs.

These considerations resulted in the final form of the pressure jerkin which was used to apply counterpressure to the whole trunk during pressure breathing. The bladder of the garment extended from the neck and armholes above to the upper thighs below (Fig. 2–7). In most experiments the bladder of the waistcoat or jerkin was inflated from the same source as that which supplied the respiratory tract. The bladder of the garment covering the trunk was connected by a simple T-piece into the hose passing from the breathing regulator to the helmet or oronasal mask. Thus the pressure applied to the surface of the trunk through the bladder equalled that delivered to the respiratory tract.

In the course of experiments in which the positive breathing pressure exceeded 100 mmHg, it became apparent that it was necessary to apply counterpressure to the upper limbs as well as to the trunk and lower limbs. A garment which consisted of the standard pressure jerkin, to which sleeves containing bladders were attached, was developed for this purpose (Fig. 2–8). In order to retain sufficient mobility of the upper limb when the garment was inflated the bladder coverage was omitted from certain regions of the upper limb. The axilla and the anterior and posterior aspects of the shoulder were not covered by the bladder so as to prevent serious limitation of movement at the shoulder. A small area over the external aspect of the elbow was not covered by bladder in order to allow flexion at this joint. The arm bladder ended at the level of the wrist. The bladder was connected to the main jerkin bladder by a pipe which passed over the top of the shoulder.

The Antigravity Suit – When required, counterpressure was applied to part of the surface of the lower limbs by means of bladders of a standard Royal Air Force antigravity suit (Mark 4) (Fig. 2–9). This garment consists of a series of rubber bladders which lie over the lower anterior part of the abdomen, the thighs and the calves; the bladders are contained within nylon bags. Donning is by way of appropriate entry slits which are closed by sliding fasteners. The tension in the outer layer of the lower limb portion of the suit can be adjusted for individual fit by means of external lacing. Inflation of the bladders applies pressure to the skin of the lower limb both directly through the inner walls of the bladders and indirectly through the encircling outer layer of nylon. This antigravity suit applies pressure to a limited area of the lower limbs only: the middle three-quarters of each thigh and to each calf. The uppermost part of each thigh, the knee, ankle and foot are not covered by the suit. The abdominal bladder of the suit is normally worn beneath the pressure jerkin. The anti-gravity suit was inflated from the same source and to the same pressure as the pressure jerkin and the helmet or mask.

Breathing and Inflation Equipment – Several methods of supplying gas
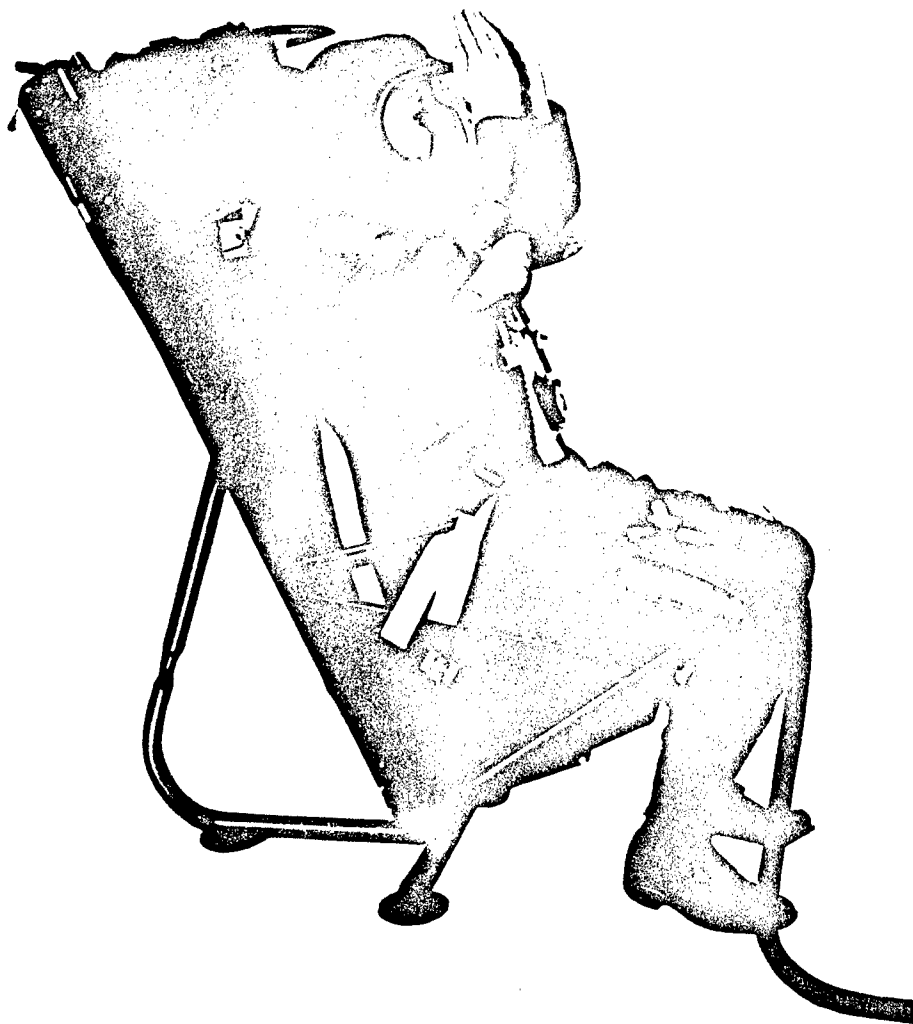
FIG. 2-7 Final form of pressure jerkin

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FIG. 2-8 Pressure jerkin showing bladder coverage of upper limbs
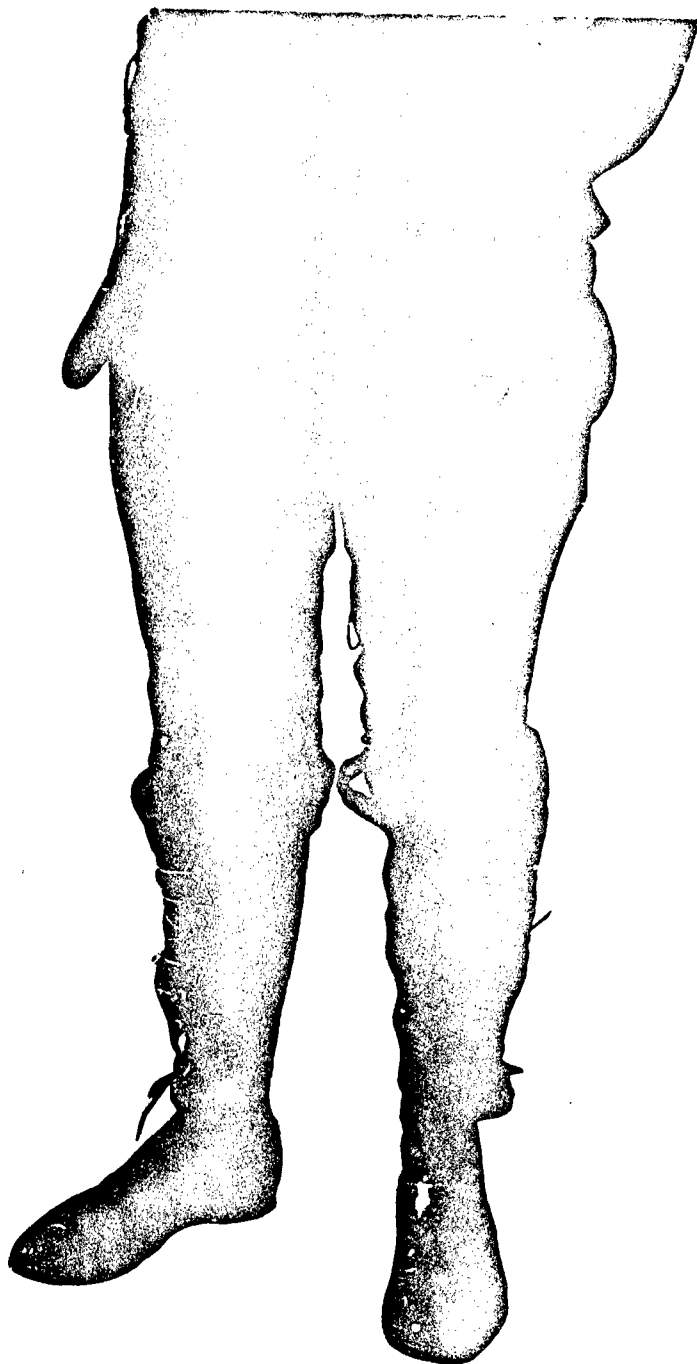
FIG. 2 9 Antigravity suit

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at a pressure greater than that of the environment to the respiratory tract and to the bladders of the pressure clothing were used in the course of this investigation. The method employed in any given experiment depended upon whether the experiments were carried out at ground level or at reduced environmental pressure and upon the nature of the measurements to be made in the course of the experiment.

Pressure demand oxygen regulators – In the majority of the experiments carried out at reduced environmental pressure the standard equipment which had been developed for use in conjunction with pressure clothing in aircraft was used. This standard equipment consisted of a high-pressure gaseous supply of oxygen and a variety of types of pressure demand regulator with the appropriate hose and connector assemblies. When used with these regulators a mask or helmet was fitted with the standard compensated outlet valve system. The delivery of gas by a demand regulator depends primarily upon the pressure at its outlet which is connected by wide bore hose to the cavity of the mask. Gas flows from the regulator when the pressure at the outlet is reduced at the beginning of inspiration and ceases when the regulator outlet pressure rises at the end of inspiration. The greater the inspiratory demand the greater is the reduction of the pressure at the outlet of the regulator and hence the greater the flow given by the regulator. Since with a simple demand regulator a reduction of the pressure in the mask below that of the environment is necessary before gas flows from the regulator, an ill-fitting mask, by allowing air to be drawn in, may well give rise to hypoxia at reduced barometric pressure. At pressure altitudes in excess of 12000 ft the regulators used in this investigation deliver gas until the pressure at the outlet of the regulator exceeds that of the environment by 2 to 3 cm of water. By this manoeuvre the pressure within the cavity of the mask during inspiration is maintained at a value slightly greater than that of the environment ("safety pressure") at least during quiet breathing. These regulators also mix air with oxygen, the proportion of the two gases depending upon altitude, so that the alveolar oxygen tension does not fall below that existing at ground level when air is breathed (Fig. 2-10). At altitudes above 33000 ft the regulators deliver 100% oxygen. Such a regulator can be set to deliver 100% oxygen under all altitude conditions.

At altitudes above 40000 ft the regulators employed in this investigation delivered oxygen at a pressure greater than that of the environment. Three types of oxygen regulator (Mark 17, 20 and 21), each providing a different relationship between delivery pressure and environmental pressure, were used. The nominal relationship between these two variables given by each of these regulators together with the specification tolerances are depicted in Fig. 2-11. The Mark 20 and 21 regulators are constructed so that when the environmental pressure suddenly falls to a value of less than 141 mmHg absolute oxygen is delivered at a very high flow (greater than 150 litres N.T.P. per minute). This high flow ensures that the required absolute pressure is attained in the pressure clothing and respiratory tract within three seconds of a sudden decompression.

The low-pressure oxygen system from the regulator to the oxygen mask or pressure helmet was designed so that it had the minimum resistance compatible with an acceptable installation. The regulator outlet was connected
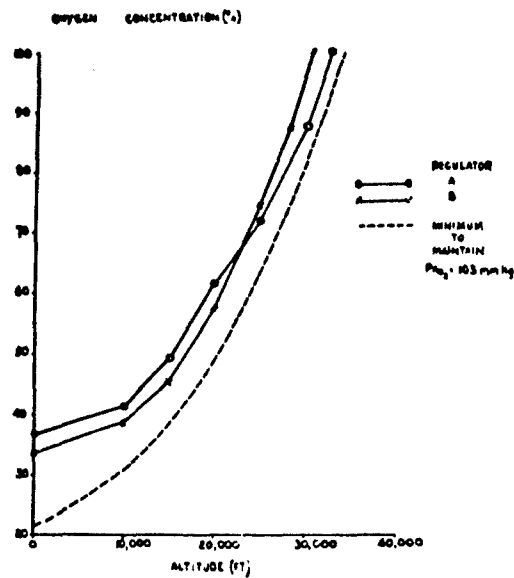
FIG. 2-10 The concentration of oxygen delivered at various altitudes by two typical pressure demand regulators. The lowest curve (interrupted line) defines the relationship between inspired oxygen concentration and altitude required to maintain an alveolar oxygen tension of 103 mmHg
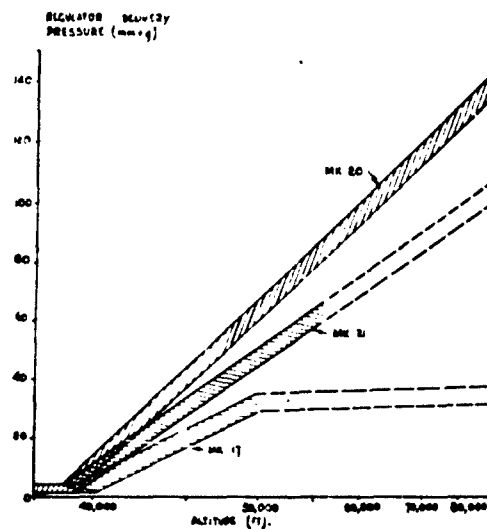
FIG. 2-11 The delivery pressures (gauge) of the Mark 17, 20 and 21 regulators at altitudes above 40000 ft. The shaded band for each regulator represents the tolerance in the delivery pressure allowed at a given altitude by the relevant specification

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by 1 to 2 m. of smooth bore hose (internal diameter 2.2 cm), a locking connector and about 1 m. of 1.9 cm internal diameter smooth bore hose to the oronasal mask or helmet, the pressure jerkin and the antigravity suit (Fig. 2-12). The resistance to flow of gas from the regulator outlet to the mask cavity of a typical experimental installation is shown in Fig. 2-13. The total resistance to breathing imposed by such an assembly depends not only on that of the low-pressure system but also upon the pressure delivery characteristics of the regulator and the resistance of the outlet valve of the mask or helmet. Performance data for a typical breathing system used in this study are given in Fig. 2-14.

Production of pressure breathing by means of a decompression chamber – When a breathing system which imposed very little resistance to respiration was required or gaseo s exchange studies were being made, an alternative technique was used to obtain pressure breathing. Although generally used at ground level this method was also employed on occasions at reduced barometric pressure. The technique made use of a decompression chamber in which the subject, wearing the pressure breathing equipment, was seated. The breathing compartment of the helmet or mask was connected by way of two pieces of smooth-bore hose (internal diameter 3.1 cm) to the external surface of the chamber. A pair of low-resistance non-return valves were fitted into the breathing system to ensure the uni-directional flow of respired gases through the hoses. The pressure clothing and the compartment of the helmet outside the breathing portion were connected by another hose to the external surface of the decompression chamber. Pressure breathing was produced by reducing the pressure in the decompression chamber by the desired amount. An advantage of this technique was that during pressure breathing the absolute pressure within the respiratory tract was equal to that at rest.

TECHNIQUE OF EXPERIMENTS AT REDUCED PRESSURE

The experimental exposures of subjects to reduced barometric pressure were made in one of two decompression chambers (a standard and a modified R.A.F. mobile decompression chamber Mark III). The standard decompression chamber consists of a cylinder 8 ft in diameter divided by a pair of doors into two compartments 7 ft and 13 ft long. The pressure within each compartment can be reduced independently by two rotary vacuum pumps. The performance of the decompression chamber in terms of the maximum rate at which air can be removed from and added to each compartment is shown in Fig. 2-15. In addition the smaller compartment is connected by a 12 in. diameter pipe to a large reservoir cylinder which can also be evacuated. The pipe between the smaller compartment and the cylinder contains a large butterfly valve. The pressure within the small compartment of the decompression chamber can be reduced suddenly by opening the butterfly valve after the air in the reservoir cylinder has been removed. The volumes of the smaller compartment and of the reservoir cylinder are such that when the latter is evacuated to a pressure of less than 8 mmHg absolute the small compartment can be decompressed from a pressure altitude of 27000 ft (258 mmHg) to 56000 ft (66 mmHg) in approximately 0.8 sec. The pipe between the small compartment of the reservoir also contains a valve plate, the position of which can be adjusted by an external handle. By means of this

MASK

MASK HOSE

MASK PLUG

PRESSURE JERKIN

JERKIN CONNECTOR

DEMAND REGULATOR

ANTI-G SUIT CONNECTOR

ANTI-G SUIT
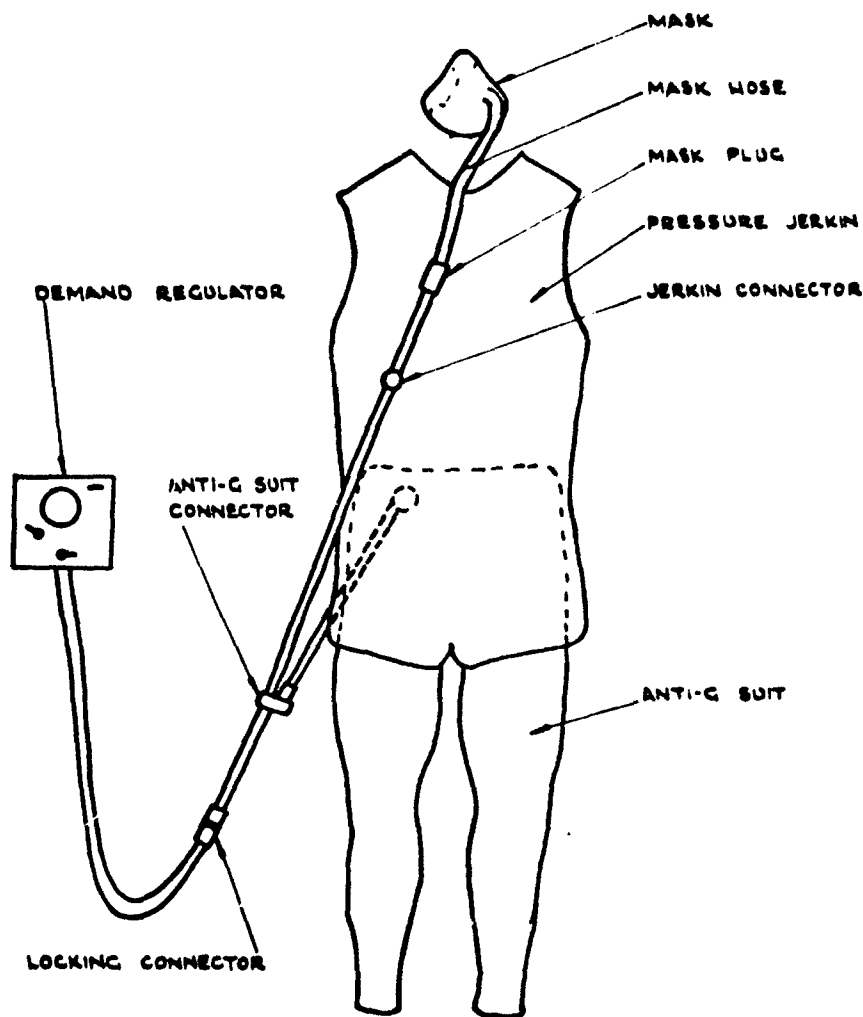
LOCKING CONNECTOR

FIG. 2-12 A typical low pressure oxygen system used with partial pressure clothing. The hose assembly from the locking connector below to the socket for the mask plug above is attached permanently to the jerkin by the jerkin connector

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PRESSURE DROP
(CM. WATER)
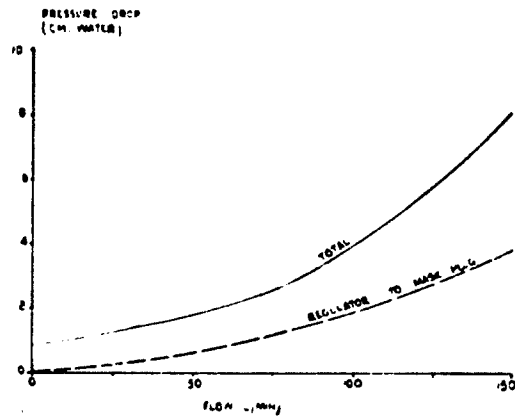
FIG. 2-13 The relationship in a typical system between flow and the pressure drop from the outlet of the regulator to the mask cavity measured under steady flow conditions at ground level. The contribution of the portion of the system from the outlet of the regulator to the mask plug is depicted by the interrupted line

MASK PRESSURE
(CM WATER)

PEAK INSPIRATORY OR EXPIRATORY FLOW (L/MIN)
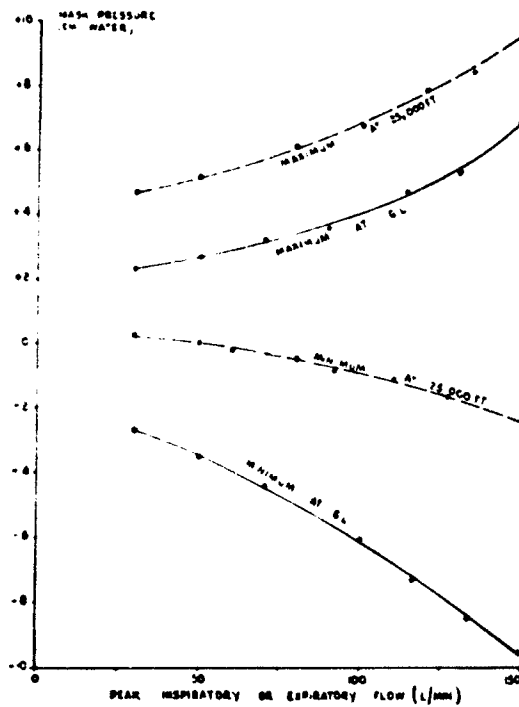
FIG. 2-14 The relationship between minimum and maximum mask pressures and peak inspiratory and expiratory flows in a typical experimental installation at ground level (GL) and at a simulated altitude of 25000 ft

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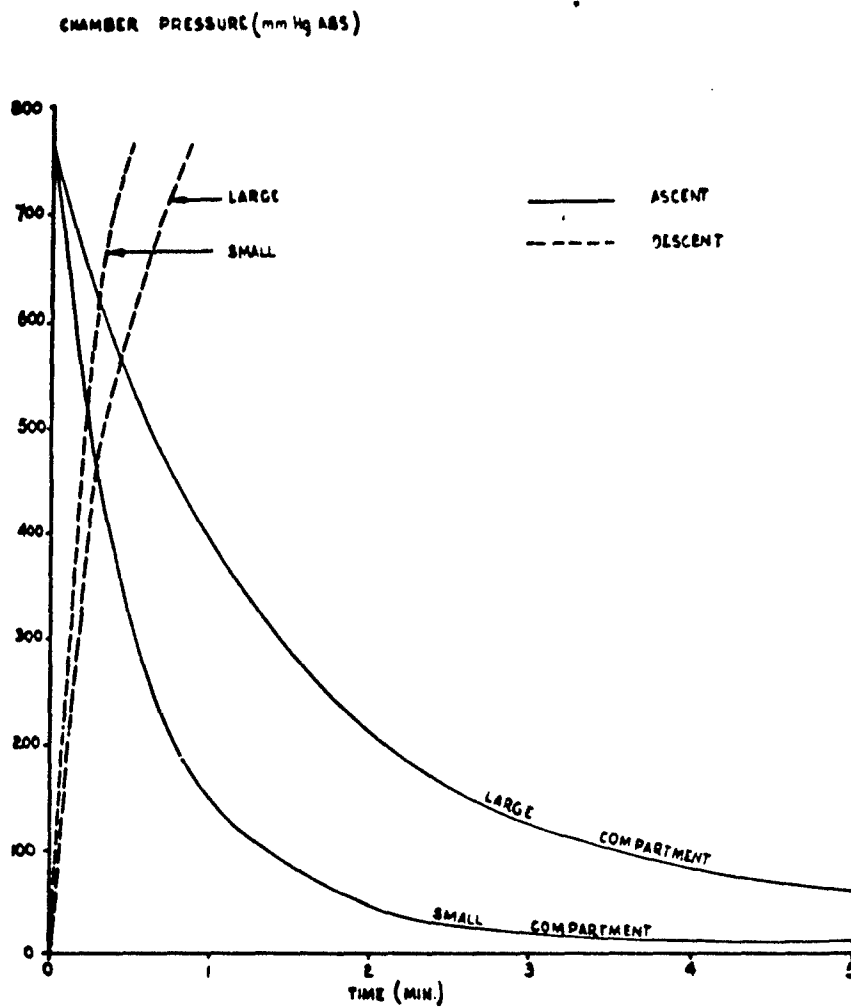
FIG. 2-15 The maximum rates of decrease (ascent) and increase (descent) of pressure which can be achieved in the small and large compartments of a mobile decompression chamber (Mark 111)

plate the time of decompression in the small compartment may be increased. In many of the exposures to simulated high altitude a decompression time of approximately three seconds was employed.

The other decompression chamber used in this investigation was modified by fitting a metal box ("capsule") with an internal volume of 22 cu. ft in the small compartment. This capsule was designed so that it would accommodate a man in the seated position (Fig. 2-16). It is attached to the doorway between the two compartments of the decompression chamber and is entered from the smaller of the two compartments. The capsule communicates with the large compartment of the decompression chamber by way of an orifice, the diameter of which can be varied by fitting a plate in which the desired size hole has been cut. The capsule was fitted primarily so that very rapid decompressions could be performed over wide pressure ranges. Before a rapid decompression was performed the orifice between the interior of the capsule and the large compartment of the decompression chamber was occluded by a sheet of radiographic film. As a safety precaution this orifice was also covered by a plate of metal which was removed just before the diaphragm was ruptured. The large compartment of the decompression chamber was evacuated to the desired final pressure whilst the subject was seated in the capsule, the door of which was closed. The small compartment and the interior of the capsule were evacuated together to the absolute pressure from which it was desired to rapidly decompress the subject. After the communication between the capsule and the small compartment was closed and the safety plate lying over the diaphragm was removed, the radiographic film was punctured. The air within the capsule flowed rapidly into the large compartment until the pressure of the two parts of the chamber was equal. In many of the experiments carried out in this investigation rapid decompressions were carried out from a simulated altitude of 25 000 ft to 56 000 ft or 60 000 ft in about one second.

In all the experiments carried out at reduced barometric pressure in the decompression chamber a team of experimenters was employed. At a minimum this team consisted of a medical officer and a decompression chamber operator in addition to the subject. The medical officer who was in charge of the team was also responsible for the medical care of the subject. The majority of the experiments performed at low barometric pressure conformed to the same pattern. The subject was decompressed from ground level to a pressure altitude of 25 000 ft at a rate simulating a rate of climb between 4 000 ft and 10 000 ft per minute. After a period at a simulated altitude of 25 000 ft to allow the subject's respiratory gas exchange to attain a steady state the decompression chamber operator carried out the final checks of the pressure in the chamber and in the decompression reservoir. The subject was then decompressed rapidly to the required final pressure. The subject was always warned that decompression was about to occur and a standard "count down" procedure was used so that the subject could regulate his respiration and was breathing out when the rapid decompression occurred. Following the rapid decompression the subject was kept at the final altitude for the desired time and then recompressed to ground level at a rate consistent with his ability to ventilate his paranasal sinuses and middle ear cavities.

Since the primary purpose of the experimental exposures to reduced barometric pressure performed in this investigation was to study hypoxia and its
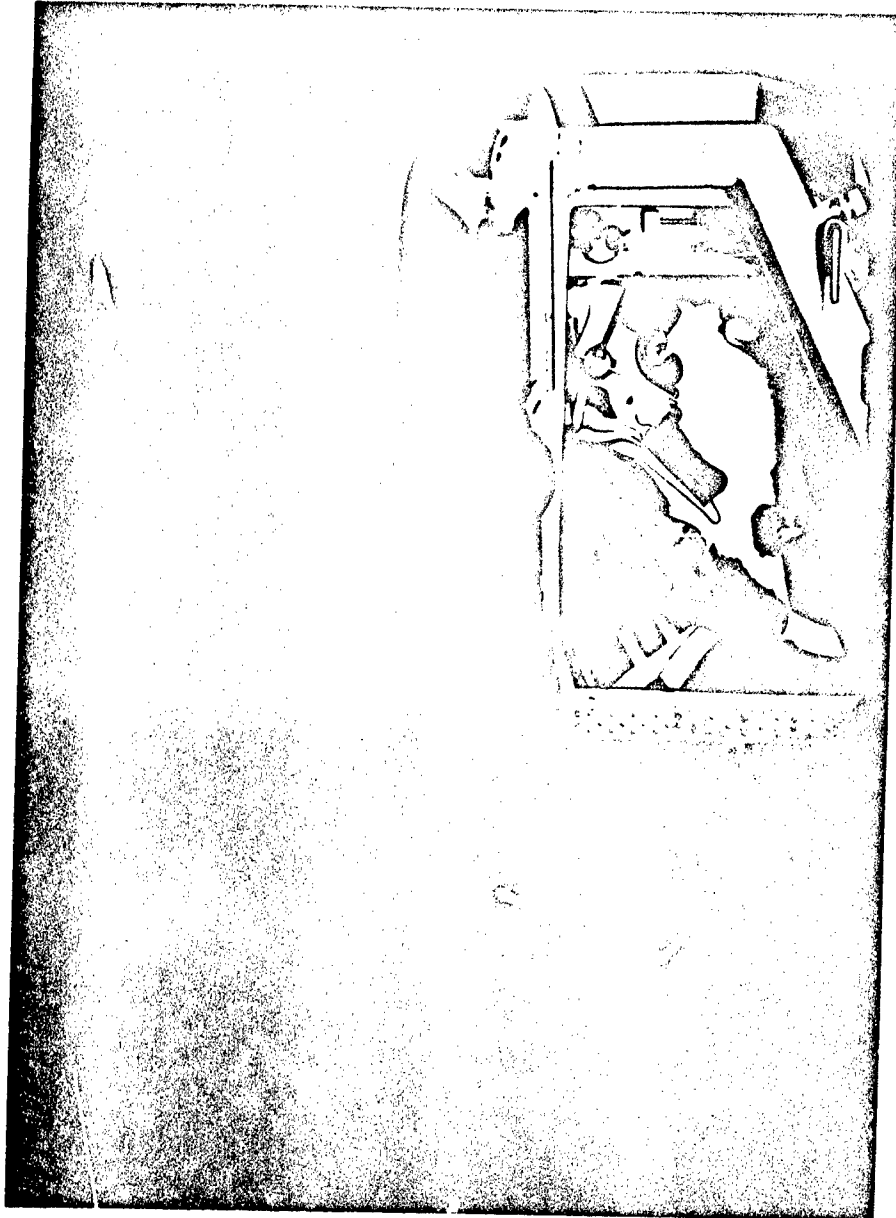
FIG. 2-16 Capsule in decompression chamber

prevention the subject was usually afforded protection against decompression sickness. A further reason for affording protection against this condition was that it can lead to serious circulatory and neurological disturbances which are occasionally fatal. This protection was given by removing a large proportion of the nitrogen normally held in the blood and tissue fluids before an exposure to low environmental pressure. Breathing 100% oxygen in an open circuit removes nitrogen from the alveolar gas and thus from the blood and tissue fluids. Experience with subjects with varying susceptibilities to decompression sickness has shown that breathing 100% oxygen for one hour provides adequate protection against the effects of exposure to a simulated altitude in excess of 30000 ft provided that the time of exposure does not exceed ten minutes. As a general rule all subjects exposed to pressure altitudes in excess of 30000 ft breathed 100% oxygen for at least one hour at ground level before decompression was performed. Special precautions were taken to ensure that no air was drawn into the breathing system during this period. The subject's oxygen mask was firmly secured and the pressure demand oxygen regulator set to deliver oxygen at a pressure between 2 and 4 mmHg greater than that of the environment. Since, however, the strict adherence to this procedure was very time consuming, denitrogenation before decompression was omitted in certain circumstances. These circumstances were that the susceptibility of the subject to decompression sickness was known from previous exposures to reduced barometric pressure without prior denitrogenation and that the exposure to pressures less than that equivalent to an altitude of 30000 ft did not exceed five minutes. In practice only the members of the High Altitude Research Unit of the Institute were exposed without denitrogenation.

Certain safety precautions were routinely practised in these experiments. The medical officer in charge of the decompression always ensured that the subject was medically fit to undergo the proposed experiment. One medical officer checked the subject's oxygen equipment before the decompression commenced. As an additional safety precaution the medical officer normally occupied the lock of the decompression chamber (the large compartment of the standard Mark III decompression chamber and the smaller compartment of the modified chamber outside the capsule), and was decompressed to a pressure equivalent to an altitude of 25000 ft. This medical officer kept the subject under continuous observation. If, following a rapid decompression, the subject indicated that he was in difficulties or if the medical officer decided that the subject's reactions were abnormal the subject was very rapidly re-compressed to a pressure altitude of 25000 ft (this normally took between one and two seconds). The medical officer could then pass into the compartment containing the subject and render him such aid as might be necessary. The pressure-altitude at which the medical officer remained when the subject was exposed to a lower pressure was chosen as 25000 ft since the subject could be recompressed at a very high rate to this pressure-altitude with very little risk of otitic or sinus barotrauma. Further, if the medical officer was decompressed to a pressure-altitude greater than 25000 ft he was likely to develop decompression sickness.

Facilities were always available for the treatment of any of the emergency conditions which might occur during the experimental programme. The serious conditions which it was considered might arise were:

(a) Unconsciousness
(b) Airway obstruction by inhalation of vomit
(c) Circulatory failure due to decompression sickness
(d) Pneumothorax and arterial air embolism
(e) Respiratory arrest
(f) Cardiac arrest.

Every instance of circulatory collapse was treated as decompression sickness until the latter diagnosis was excluded by an observation period of a minimum of four hours following the incident.

In all those experiments in which it was considered that syncope might arise and in many of the more routine exposures to reduced barometric pressure in the decompression chamber, the electrocardiogram and arterial blood pressure were recorded throughout the experiment. A second medical officer observed these recordings and in the event of any suggestion of impending collapse warned the medical officer in charge of the experiment who generally ordered the immediate cessation of the exposure to reduced environmental pressure.

RESPIRATORY TECHNIQUES

Measurement of pulmonary ventilation and collection of expired gas – Both open and closed circuit techniques were employed in the course of this investigation. Measurements of respiratory minute volume were made by means of a recording Tissot spirometer (capacity 150 litres). The circuit was usually arranged so that the subject inspired from the spirometer, particularly when collections of expired gas were made. These were generally taken by passing the expired gas into a series of Douglas bags of suitable capacity over timed intervals. Subsequently the contents of each Douglas bag were thoroughly mixed and the volume of gas collected measured by passing the contents at a steady rate (of less than 15 litre/min.) through a water sealed gas meter. As the contents of the bag were emptied a sample of the gas was taken through the side arm of the Douglas bag into a 100 ml capacity gas sampling tube by allowing the mercury with which it had been previously filled to flow out. Before the sample was taken the side arm of the Douglas bag was flushed by drawing through it and the side arm of the sampling tube 50 ml of the bag contents by means of a suitable syringe. By this technique the volume of gas drawn from the Douglas bag in the process of taking the sample was known accurately so that the total volume of the bag contents could be calculated. In most of the closed circuit experiments a standard 6 litre recording bell type spirometer was used. The circuit contained a carbon dioxide absorber (soda lime) and a respiratory pump which circulated the contents at approximately 50 litre/min.

Flow measurement – Various techniques were used to measure the velocity of flow of gases. When the flow was steady it was measured by means of a suitable rotameter. Care was taken to maintain the pressure and temperature conditions within the rotameter tube equivalent to those under which the instrument had been calibrated. The majority of flow measurements, however, were made during respiration. Two types of pneumotachygraph were used When only dry gas was to pass through the flowmeter a gauze screen (400 per in.) instrument based upon that developed by Lilly (1950) (190) was

used. Steady flow calibrations showed that there was a linear relationship between the flow of gas and the pressure drop across the screen at flows of up to 250 litre/min. When moist expired gas was to pass through the flowmeter a Fleisch capillary tube meter (1960) fitted with a heater was employed. Calibration of this instrument demonstrated that there was a linear relationship between the flow of gas and the pressure difference between the two tappings of the transducer. The pressure difference created between the pressure tappings of each of the flowmeters by gas flow was recorded by means of a differential capacitance manometer of suitable sensitivity and an appropriate amplifier. The output of the amplifier was fed on to the galvanometer of a photographic recorder. The tubes from the pressure tappings on the flowmeter to the differential manometer were made as short as possible (generally about 10 cm) and the lengths of the two tubes were kept equal. Before and after each series of measurements the experimental record was calibrated by passing known steady flows through the flowmeter using a rotameter and recording the corresponding galvanometer deflections.

The adequacy of the overall dynamic response of the flow measuring equipment was tested using an electrically driven pump which produced sinusoidal variations in flow. The stroke volume of the pump could be varied between 0.5 litre and 3.0 litre and its frequency from 8 to 40 strokes/min. The flowmeter was connected directly to the outlet of the pump and the flow patterns produced by various settings of the pump were recorded. The frequency and stroke volume of the pump were measured independently of the flowmeter with a stopwatch and a recording spirometer for each of the settings used. The peak flow was then calculated for each setting by means of the equation:

$$V = \frac{\dot{V}}{\pi f}$$

where

V = stroke volume
$\dot{V}$ = peak flow
f = frequency

The relationship between the calibrated peak flow and that recorded by the Lilly pneumotachygraph is shown in Fig. 2–17, for stroke volumes between 0.5 and 3.0 litre and frequencies between 10 and 40 per minute. It may be seen that there was excellent agreement between the calibrated and peak flows. Similar results were obtained with the Fleisch flowmeter. These results confirmed that the pneumotachygraphs and associated amplifiers and recording equipment used in this investigation faithfully reproduced the actual changes in the flow which were occurring through the flowmeter.

On some occasions the volume of each breath was obtained by planimetric integration of the area between the flow curve and the line representing zero flow. The overall accuracy of this technique was assessed by simultaneously recording the subject's tidal volume by means of a bell spirometer and the flow of gas by means of a pneumotachygraph. The areas contained by the flow curve for the two phases of each breath were measured three times by means of a suitable planimeter. The volume represented by a unit area of
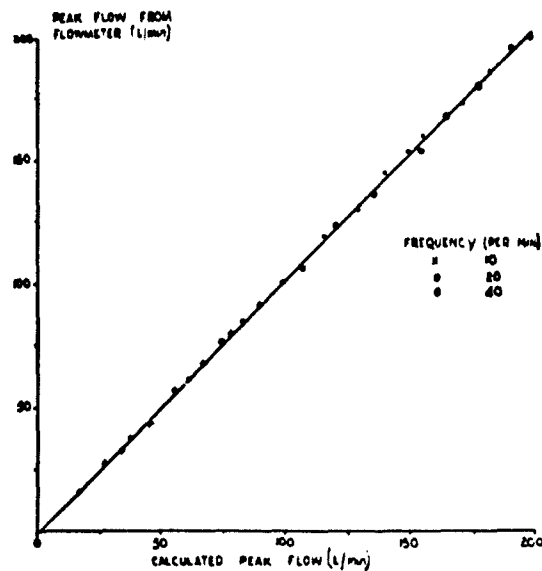
Fig. 2-17 The results of the dynamic calibration of the Lilly pneumotachygraph by means of a sine wave pump. The peak flow indicated by the Lilly flowmeter is plotted against the corresponding peak flow calculated from the stroke volume and frequency of the pump
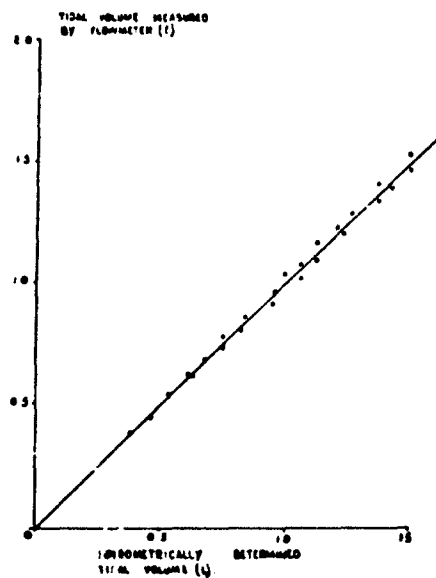
Fig. 2-18 The relationship between the value of the tidal volume obtained by planimetric integration of the output of the Lilly pneumotachygraph and the actual tidal volume measured directly with a recording spirometer

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record was determined by measuring the area contained between the calibration line produced by a known flow and the zero flow line for a length of record representing a known time. The tidal volumes calculated in this manner for twenty-five breaths are compared with the actual spirometrically determined volumes in Fig. 2–18. The tidal volumes calculated from the planimetric integration of the flow curve agreed with the corresponding spirometric tidal volumes within $\pm 3\%$ of the tidal volume.

Measurement of pressure in breathing equipment – In many circumstances it was necessary to measure the difference between the pressure within a part of the breathing equipment and that of the environment. When the velocity of gas flow at the point at which the pressure measurement was to be made was considerable, care was taken to ensure that only the lateral component of the total gas pressure was measured. This requirement was met when pressure measurements were made in a tube by inserting a piezometer ring with the same internal diameter as the pipe. Where the actual velocity of gas flow was relatively low, such as within the cavity of an oronasal mask, the pressure was measured by means of a simple tapping through the wall and an alcohol or mercury manometer depending upon the magnitude of the pressure. A rapidly changing pressure was measured by means of an unbonded strain gauge pressure transducer of the appropriate sensitivity. The change in the resistance of the strain gauge of the transducer was measured by means of a Wheatstone bridge circuit and a carrier amplifier, the output of which was fed on to the galvanometer of a bromide paper photographic recorder. Before and after each experimental period the output of the transducer was calibrated using an appropriate manometer. All the pressure transducers used in this study gave a linear relationship between pressure change and galvanometer deflection.

Intraoesophageal pressure measurement – The changes in pleural pressure during respiration were measured by means of an oesophageal balloon. As pointed out by Mead, McIlroy, Selverstone and Kriete (1955) (209) the length of the balloon used to record intraoesophageal pressure is important, the value of the pulmonary compliance given by a short balloon depending upon its position in the oesophagus. The thin latex balloons used in this investigation had the dimensions suggested by Mead, McIlroy, Selverstone and Kriete (1955) (209), i.e. they were 16 cm long and 0.8 cm in diameter. The pressure within the balloon was measured by way of a 75 cm length of polyethylene catheter (1.4 mm internal diameter) the distal end of which was sealed within the balloon. The wall of the part of the catheter within the balloon contained small holes approximately 1 cm apart. The pressure distension characteristics of each balloon used in this study were determined by introducing known volumes of air into the balloon and recording the corresponding pressure within the balloon by means of an alcohol manometer. A typical pressure-distension curve is presented in Fig. 2–19. The pressure within the balloon equalled that of the environment when the volume of gas within it was between 1 ml and 5 ml. The pressure within the balloon was recorded by means of a capacitance manometer of the Hansen (1949) (141) type with a chamber volume of less than 0.2 ml. Changes in the capacity of the transducer were amplified and fed on to the galvanometer of a photographic recorder.
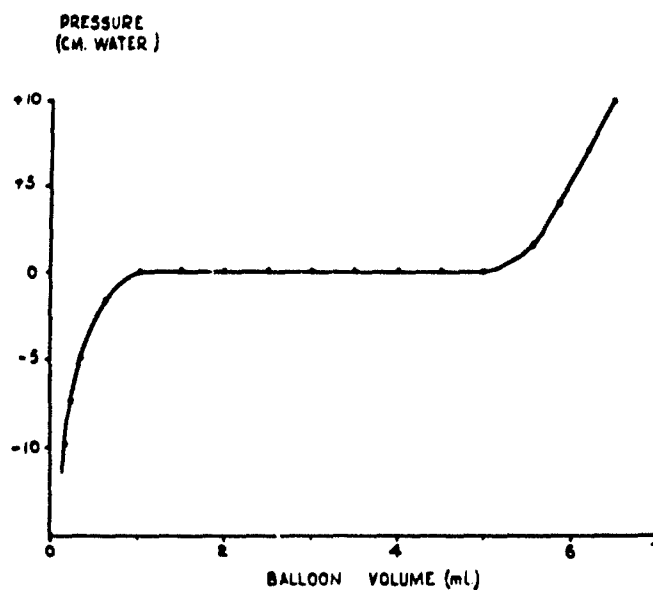
FIG. 2-19 The pressure-distension curve of a balloon used for the measurement of intraoesophageal pressure
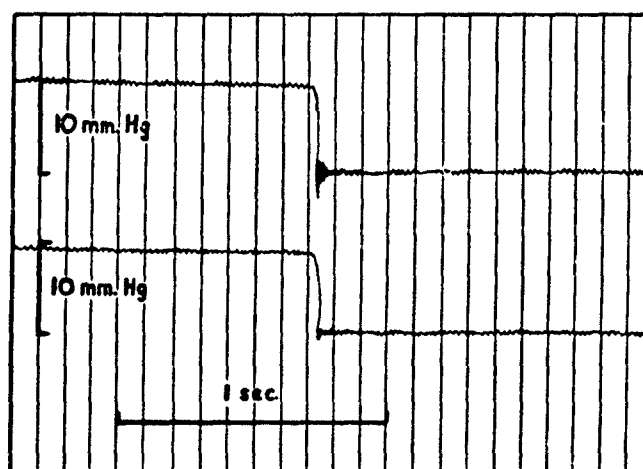
FIG. 2-20 The response of the intraoesophageal pressure recording system to a sudden reduction of the pressure around the balloon. The upper tracing is a record of the pressure around the balloon whilst the lower is the output of the balloon recording system

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The accuracy with which the balloon-catheter-transducer-amplifier-recorder system followed the changes in pressure immediately around the balloon was examined by placing the balloon within a flask. The pressure in the flask was varied by means of a syringe attached to a side arm of the flask. Sudden changes in pressure were produced by covering the opening of the flask with a rubber membrane, increasing the pressure in the flask and then suddenly rupturing the membrane. The changes in pressure within the flask were measured directly by means of a second capacitance transducer which was connected by a short length of rigid pipe to the cavity of the flask. A typical recording of the response of the balloon recording system to a sudden change in flask pressure is shown in Fig. 2-20. Further tests demonstrated that the balloon system recorded the actual pressure changes, both static and dynamic, up to pressures of ± 50 cm of water.

In order to measure intraoesophageal pressure the balloon was first swallowed through the mouth into the stomach. The minimum volume of air necessary to ensure that the recording system faithfully reproduced the pressure outside the balloon was introduced by means of a syringe. The balloon was then withdrawn until the pressure recorded by it fell during inspiration. In experiments in which the pressure within the stomach was measured a similar balloon was swallowed until it lay within the stomach. Frequently the oesophageal and gastric pressures were recorded simultaneously. In this case a double balloon was used. The polyethylene catheter of the distal balloon was carried through the lumen of the proximal one without communicating with it. The distal ends of the two balloons were separated by 20 cm

GAS ANALYSIS
Discrete samples – The concentrations of carbon dioxide and oxygen in discrete gas samples were determined by analysis by the standard Haldane technique (136) using an apparatus with a 21 ml burette. On every occasion duplicate analyses were performed and if the pair of analyses differed by more than 0.02 volumes per cent a third analysis was carried out. On many occasions the total concentration of absorbable gas exceeded 30% of the sample and in these circumstances a nitrogen dilution technique was used. The carbon dioxide and oxygen content of a 20 ml sample of room air was absorbed and a portion of the residual nitrogen in the apparatus expelled until a volume of between 15 and 15.5 ml remained. The volume of this nitrogen was accurately measured and then the gas was transferred into the oxygen absorbent. Approximately 5 ml of the sample to be analyzed was then drawn into the measuring burette taking the usual precautions to prevent contamination with laboratory air. The exact volume of nitrogen which had been stored in the oxygen absorbent was drawn back into the measuring burette and the total volume of nitrogen together with the sample was accurately measured. The volumes of carbon dioxide and oxygen content in the mixture were then determined in the usual manner. All the analyses performed by this method were carried out in duplicate. The accuracy of the nitrogen dilution technique was less than that of the standard Haldane technique. The variation between duplicate analyses was found to be less than ± 0.06 volumes per cent.

Continuous nitrogen analysis – The concentration of nitrogen in the respired gases was measured continuously by emission spectroscopy using the technique developed by Lundin and Akesson (1954) (198) from the original form of nitrogen meter designed by Lilly and Anderson (1944) (191). A small sample (100 ml/min.) of the gas to be analyzed was drawn continuously through the electrical discharge tube of the instrument by means of a rotary vacuum pump. The specially designed needle valve (198) was inserted in the hose through which the gas to be sampled flowed. The size of the sampling orifice could be varied so as to maintain the pressure within the emission tube at the desired level. The output of the nitrogen meter was displayed on a milliammeter and also fed on to the galvanometer of a photographic recorder.

Preliminary studies were made in order to determine the effects of variations in operating conditions upon the sensitivity of the instrument. The effects of variation of the pressure within the emission tube and of the current flowing through the emission tube (ionizing current) were investigated. The pressure within the vacuum system was measured with a McLeod gauge which was connected by means of a "T" piece between the emission tube and the vacuum pump 10 cm from the outlet of the emission tube. The relationship between the vacuum pressure and the output current when the instrument was sampling laboratory air is depicted in Fig. 2–21. Increasing the ionization current was found to increase the output of the instrument at a given nitrogen concentration and operating pressure. Water vapour was found not to have a specific effect upon the output of the instrument. With a given nitrogen concentration a sample saturated with water vapour gave a slightly smaller output than a dry sample. The decrease in output was found to be proportional to the dilution of the dry sample by water vapour. Carbon dioxide, however, did have a specific effect, although quantitatively this was relatively small (Table 2–1).

TABLE 2–1

THE EFFECT OF 100″₀ CARBON DIOXIDE
UPON THE OUTPUT OF THE NITROGEN METER

Ionization current = 3.5 mA

Operating pressure (mmHg abs.)	Meter reading with 100″₀ carbon dioxide (arbitrary units)	Equivalent nitrogen concentration (″₀)
0.8	13.0	2.4
1.2	10.6	2.6
1.8	6.0	3.1
2.5	5.0	1.5
2.8	3.5	1.1
4.3	2.5	0.8
6.0	1.5	0.5
7.0	1.0	0.4

METER READING

OPERATING PRESSURE (mm Hg ABS)
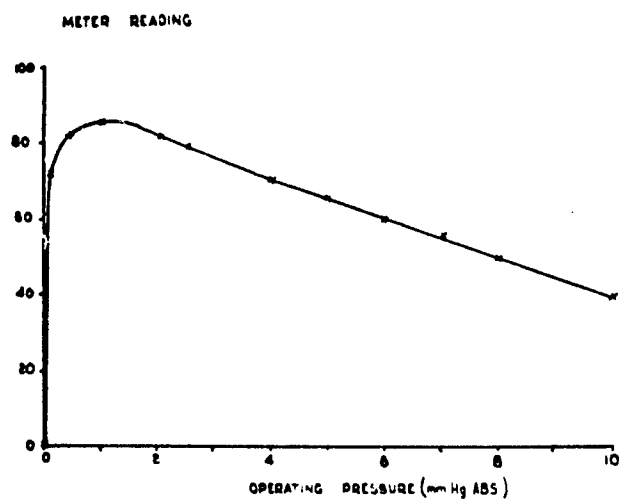
FIG. 2-21 The effect of varying the tube operating pressure upon the output of the
Lundin nitrogen meter whilst sampling air (discharge current   3.5mA)

GALVANOMETER DEFLECTION (mm)

NITROGEN CONCENTRATION (%)
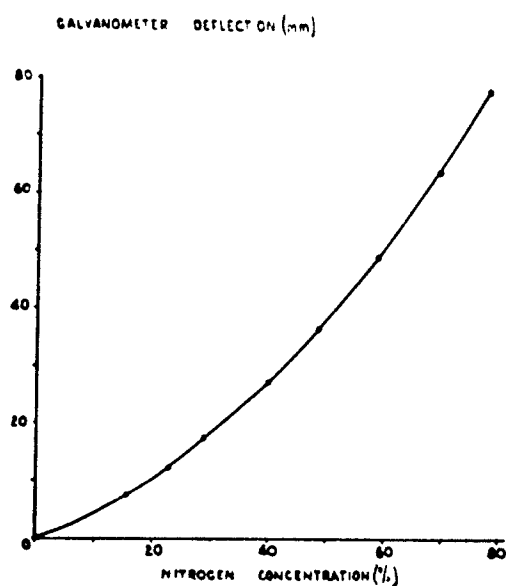
FIG. 2-22 A typical calibration curve for the output of the Lundin nitrogen meter

Since this effect was greatest at the lower operating pressures and since the total output of the instrument for a given nitrogen concentration fell with increasing operating pressure, an operating pressure of 2.5 mmHg absolute was adopted as a compromise throughout this experimental study.

The calibration curve of the instrument was determined by passing gas mixtures containing various proportions of oxygen and nitrogen through the needle valve and noting the corresponding output current. The mixtures were prepared by filling aircraft high pressure storage cylinders (which contained 750 litre N.T.P. of gas when filled to a pressure of 1 800 lb/sq. in.) with various proportions of oxygen and nitrogen. The approximate composition of the cylinder contents was determined as it was filled by means of an accurate high pressure gauge. In practice it was found that provided time was allowed to elapse for temperature equilibration to occur this method was accurate to within ± 0.5 volumes per cent. The accurate composition of the cylinder contents was determined by the Haldane analysis. A typical calibration curve for the nitrogen meter is shown in Fig. 2-22. The relationship between nitrogen concentration and meter output was linear at nitrogen concentrations below 10%, but at higher concentration it was curvilinear. The shape of the upper part of the curve was affected by variations in the operating conditions. In practice the operating conditions were kept constant and the output of the instrument was calibrated before and after each experimental period.

In experiments in which it was desired to follow the concentration of nitrogen when this was changing rapidly, the length of the tube from the needle valve to the emission tube of the nitrogen meter was kept to a minimum (generally less than 10 cm). This was in order to reduce the time delay between the change in the concentration of nitrogen at the sampling point and the start of the change in the output of the meter. In every experiment both the delay time and the response time of the measuring system was determined. The composition of the gas passing through the sampling needle valve was suddenly changed by means of a two-way tap, one arm of which was connected to a bag of oxygen, the other of which was open to the atmosphere. Gas was drawn from the tap and through the sampling valve at a high flow (greater than 200 litre/min.) by means of a suitable high capacity pump. The instant at which the composition of the gas was changed was recorded by means of a pneumotachygraph incorporated in one of the limbs of the tap. Whilst the delay time was found to vary between 0.1 and 0.3 sec. with various experimental arrangements, the time taken for completion of 95% of the total response was always less than 0.1 sec.

Continuous carbon dioxide analysis – The instantaneous concentration of carbon dioxide in the respired gases was measured continuously by infrared absorption spectroscopy using a Liston-Becker Model 16 analyzer. The measuring head of the instrument was mounted as close as possible to the subject who breathed to and fro through the sampling cell. The output of the instrument was fed on to either a single channel direct writing recorder or a galvanometer of a photographic recorder. When in use the instrument was calibrated every thirty minutes using three gas mixtures containing various concentrations of carbon dioxide (between 0 and 7%) in air. These gas mixtures were made in the same manner as the oxygen-nitrogen mixtures and their composition was determined by analysis in the Haldane apparatus. A

typical calibration curve is presented in Fig. 2–23. The speed of response of the instrument to a sudden change of carbon dioxide concentration was determined by filling the sample cell with 5% carbon dioxide in air and then suddenly removing it with a stream of air flowing at 200 litre/min. The air flow was also recorded. It was found that the output began to change within 0.01 sec. of the beginning of the air flow and that it was complete within a total time of 0.1 sec.

Measurement of concentrations of carbon monoxide and helium – In order to determine the diffusing capacity of the lung the concentrations of carbon monoxide and helium in various gas mixtures were measured. The carbon monoxide concentration was measured by an infra-red absorption technique using an analyzer constructed upon the principle developed by Luft. Although the detector of the instrument was filled with carbon monoxide preliminary studies showed that the instrument was slightly sensitive to carbon dioxide and water vapour. When these gases were present in the gas mixture to be analyzed the mixture was passed through tubes containing soda lime and silica gel in order to remove them. The concentration of carbon dioxide when this gas was present in the mixture was determined by Haldane analysis so that the carbon monoxide concentration in the original mixture could be corrected for the removal of this gas. The gain of the instrument was set up at the beginning of each series of experiments using a standard carbon monoxide mixture stored at high pressure in a suitably lined cylinder.

The linearity of the calibration curve of the carbon monoxide meter was determined by the progressive dilution of a gas mixture containing carbon monoxide with known amounts of oxygen. A closed circuit consisting of a 6 litre capacity bell spirometer and a centrifugal pump was set up. A fraction of the gas circulating around the main circuit was passed through a silica gel drying tube, the carbon monoxide meter and thence back into the main circuit. The gain of the carbon monoxide meter was set up in the standard manner and then the whole system flushed with oxygen and the bell of the spirometer emptied. Sufficient carbon monoxide was added to the circuit to give an initial carbon monoxide concentration of approximately 0.25%. Oxygen was then added to the circuit in increments of approximately 500 ml and the corresponding readings of the carbon monoxide meter taken.

The reciprocals of the carbon monoxide meter readings were then plotted against the corresponding increments of the volume of gas in the spirometer circuit. In each experiment a linear relationship was found between the reciprocal of the CO meter reading and the volume of oxygen added. Thus the carbon monoxide meter had a linear calibration curve. A linear regression line was calculated for each group of measurements and the average standard deviation about the regression line was found to be $\pm 0.0006\%$ CO. In two experiments the effect of varying oxygen-nitrogen concentrations upon the linearity of the calibration curve of the meter was determined by washing out the dead space of the circuit with oxygen and adding carbon monoxide, carrying out part of the dilution with oxygen and then completing it with nitrogen. It was found that varying oxygen-nitrogen concentrations in the diluting gas had no significant effect upon the linearity of the calibration curve of the carbon monoxide meter.

The concentration of helium was estimated by measuring the thermal con-
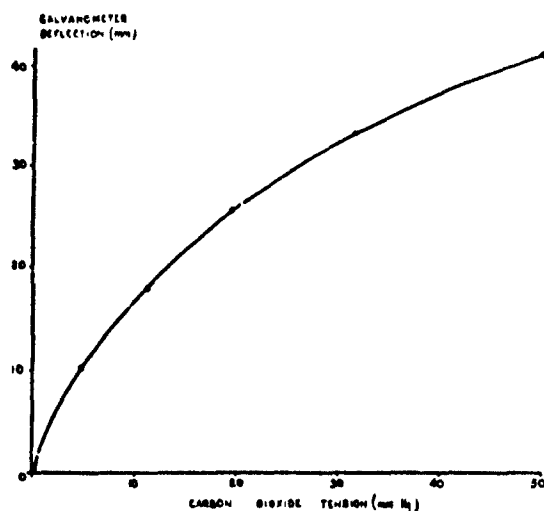
FIG. 2.23 A typical calibration curve for the output of the Liston-Becker carbon dioxide meter
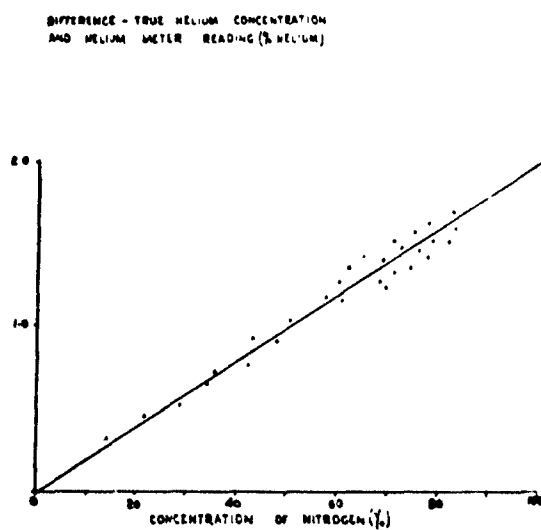
FIG. 2.24 The effect of variations in the concentration of nitrogen in a mixture consisting of helium, oxygen and nitrogen upon the output of the helium katherometer. The difference between the apparent helium concentration as read from the meter scale and the true helium concentration is plotted against the concentration of nitrogen in the mixture

ductivity of the gas mixture using a Cambridge katharometer. Since this technique is not specific and since the thermal conductivities of water vapour and carbon dioxide differ from that of oxygen, these gases were removed from the samples before passing them through the katharometer by means of silica gel and soda lime. Since there is also a small difference in the thermal conductivities of oxygen and nitrogen a standard procedure was developed and a calibration curve was constructed for the instrument to allow for changes in nitrogen concentration. The instrument was adjusted to read zero when oxygen was passed through it. The accuracy of the calibration of the instrument for helium in oxygen was determined by the dilution of the helium and oxygen mixture with oxygen. The actual experimental technique used to determine the linearity of the calibration curve of the helium katharometer was similar to .hat employed to check the linearity of the carbon monoxide infra-red analyzer. A closed spirometer circuit was used with a fraction of the gas passing around the main circuit passing through a katharometer. The circuit was washed out with oxygen and approximately 1 litre of this gas was left in the spirometer. The helium katharometer was then set up to read zero. Sufficient cylinder helium was then added to give a final concentration of the order of 14%, oxygen was added in approximately 500 ml aliquots and the corresponding helium meter readings noted. A linear relationship was found between the reciprocal of the helium meter reading and the corresponding increment in the volume of the gas in the circuit. The standard deviation about the linear regression line calculated for the data was found to be $\pm 0.034\%$. The dead space of the closed circuit was given by the value of the intercept of the regression line on the volume axis.

The effect of nitrogen upon the response of the helium katharometer to a given concentration of helium was determined by using nitrogen to dilute the initial helium in oxygen mixture in the closed circuit. The actual helium concentration after a given volume of nitrogen had been added was calculated from the dilution of the initial helium in oxygen mixture (using the volume of the dead space of the circuit and the amount of gas added to it). Thus it was possible to obtain by subtraction the difference between the true helium concentration in the circuit and the reading of the helium meter. It was also possible to calculate the nitrogen concentration in the circuit at any given point from the total volume of nitrogen added to the circuit and the initial amount of helium and oxygen added. Thus it was possible to relate the difference between the actual helium concentration and the reading given by the meter to the concentration of nitrogen in the circuit. The relationship between these two variables was found to be a straight line (Fig. 2-24) with a slope of 0.0198% per 1% nitrogen.

Arterial Blood – When required, samples of arterial blood were withdrawn from the brachial artery through a Riley needle. The needle was inserted after local analgesia had been produced by the infiltration of 2% lignocaine hydrochloride around the artery. The needle was connected by way of a three-way tap to a bottle of saline to which heparin had been added (10000 units of heparin to 500 ml of saline). Throughout the experimental period, except when samples of arterial blood were being withdrawn, a slow stream of the heparinized saline was infused to maintain the patency of the intra-arterial needle. Prior to sampling the infusion was stopped and the needle and

tap flushed with arterial blood. Each blood sample was taken into a 20 ml syringe, the dead space of which had been previously filled with a solution of heparin (500 units per ml) to which sodium fluoride had been added (7.6 mg per ml) together with a small amount of mercury. Between 16 and 18 ml of blood was taken on each occasion and the syringe capped with a sealed hypo-dermic needle hub. The syringe was then rotated for two minutes to ensure adequate mixing of its contents and placed in a beaker containing iced water. Analyses were carried out as soon as possible, the longest time elapsing between sampling and analysis being two hours.

The oxygen content of each sample of blood was determined in duplicate by the manometric technique of Van Slyke and Neill using 2 ml samples. The difference between duplicate analyses did not exceed 0.04 volumes per cent. About 8 ml of blood was saturated with air by rotating it in a tonometer for fifteen minutes at room temperature, air being flushed through the tonometer every five minutes. Where several arterial blood samples were taken in the course of a single experiment blood for the determination of the oxygen capacity was usually obtained by pooling the blood remaining after the analysis of oxygen content had been performed on the separate samples. When this procedure was followed haematocrit determinations were made on each sample and on the pooled sample used for the estimation of oxygen capacity in order that the effect of any change in red cell concentration could be corrected. Duplicate analyses of the oxygen content of the saturated blood were performed using the manometric technique. The oxygen capacity was calculated from the oxygen content of the saturated blood by subtracting the concentration of oxygen in physical solution using the partial pressure of oxygen in the air with which it was equilibrated and the data obtained by Sendroy, Dillon and Van Slyke (1934) (261).

The concentration of physically dissolved oxygen in each arterial sample was obtained by calculating the approximate percentage saturation from the oxygen content of the sample and the oxygen capacity of the blood. The corre-sponding oxygen tension was determined from a standard oxygen dissociation curve (74) and the concentration of the physically dissolved oxygen calcu-lated. The true oxygen saturation of the sample was then calculated from its oxygen content, corrected for the physically dissolved oxygen and the oxygen capacity of the blood. The concentration of hydrogen ions in a blood sample was measured by means of a glass electrode-calomel half-cell system at a temperature of 38°C. The measurements were made anaerobically in the apparatus designed by Astrup (1957) (11), the temperature of which was held constant at 38 ± 0.1°C. Preliminary experiments showed that when 2 ml of blood at room temperature was placed in the measuring chamber the tem-perature of the blood and the water in the jacket were equal after four minutes. In all the subsequent measurements four minutes was allowed to elapse between the introduction of a sample into the measuring chamber and the reading of its pH.

The pH meter (Radiometer Type 4) was used to measure the potential difference between the glass electrode and the calomel cell, all readings being made in millivolts. The relationship between pH and the output of the cell was determined daily using three standard buffers, viz. 0.05 M potassium hydrogen phthallate, the pH of which was taken to be 4.025 at 38°C; 0.025 M

disodium hydrogen phosphate and 0.025 M potassium dihydrogen phosphate, the pH of which was taken to be 6.839 at 38 C and 0.05 M tetraborate decahydrate, the pH of which was taken to be 9.085 at 38 C. A linear relationship was obtained between the output of the cell (millivolts) and the pH of these buffers. A fourth buffer of the Sorensen type was prepared (200 ml $\frac{M}{15}$ potassium dihydrogen phosphate and 800 ml $\frac{M}{15}$ disodium hydrogen phosphate) and the pH of this was determined daily using the three standard buffers. This buffer, which had a mean pH of 7.360 at 38 C, was used to standardize the pH meter during measurements of the pH of the blood samples.

After the output of the pH meter had been standardized using the pH 7.360 buffer the measuring chamber was washed thoroughly with distilled water. The syringe containing the blood sample was removed from the iced water and allowed to remain at room temperature for five minutes, during which time it was rotated (fifteen times per minute) by an electric motor so as to ensure that its contents were thoroughly mixed. The sealing cap was then removed from the syringe and replaced by a short piece of rubber tubing which covered the tip of the syringe. About 2 ml of blood was then introduced anaerobically into the measuring chamber and after four minutes its pH was measured. After washing out the blood a further volume of the pH 7.360 buffer was placed in the measuring chamber and the output of the cell was measured. Thus the measurement of the pH of each sample of blood was bracketed by a pair of measurements of the pH of the buffer. It was possible, therefore, to deduce whether any change had occurred in the output/pH ratio of the glass electrode-calomel cell. The reproducibility of the pH measurements was determined by carrying out a series of six consecutive measurements of the pH of a single blood sample. A variation of ± 0.002 unit was found.

When required the carbon dioxide tension of an arterial blood sample was measured by the interpolation technique developed by Astrup (1957) (11). This consists of measuring the pH of the blood sample and determining the relationship between carbon dioxide tension and pH for the plasma separated from the blood sample. The arterial sample was divided into two portions and the pH of one portion determined anaerobically as described above. The remainder of the sample was introduced anaerobically into a small centrifuge tube (capacity 8 ml) beneath a layer of liquid paraffin. Sufficient of the blood sample was delivered into this tube to displace all but a thin layer (0.5 cm) of the liquid paraffin. A rubber bung was then fitted to the centrifuge tube. In order to allow any trapped air and excess liquid paraffin to escape a hypodermic needle was placed through the bung before it was inserted into the tube. The needle was removed after the bung was in position. The tube and its contents were then centrifuged at 2000 r.p.m. for fifteen minutes at room temperature, when complete separation of the plasma occurred.

The plasma was removed and placed in the measuring chamber of the pH meter together with one drop of octanol to avoid foaming. The plasma was then equilibrated with a known tension of carbon dioxide by bubbling a humidified gas mixture containing approximately 5.5% carbon dioxide in

oxygen through the plasma in the measuring chamber for six minutes. The carbon dioxide-oxygen mixture was stored at high pressure in a cylinder and its composition was determined once a week by analysis in the Haldane apparatus. The pH of the plasma was then measured. The plasma was further equilibrated with a gas mixture containing approximately 2.5% carbon dioxide in oxygen and the corresponding pH value determined. The two pairs of values relating carbon dioxide tension and pH for each sample of plasma were plotted on semi-logarithmic paper ($\log_{10} PCO_2$ against pH) and the two points joined by a straight line (44). The tension of carbon dioxide in the original blood sample was then obtained by reading the carbon dioxide tension corresponding to the pH value of the blood sample from this straight line.

The accuracy of this method of measuring the tension of carbon dioxide in blood was determined by equilibrating samples of venous blood with various gas mixtures containing known tensions of carbon dioxide and then measuring the tension of the carbon dioxide of the blood in the manner described in the previous paragraphs. Venous blood to which heparin had been added was placed in a tonometer containing a mixture of carbon dioxide and oxygen and the gas and blood equilibrated by rotation of the tonometer in a water bath at 38 ± 0.1 C. After thirty minutes the blood was transferred into a syringe, the dead space of which had been filled with mercury and heparin solution. The carbon dioxide tension of the blood was then measured by the modified Astrup technique. The concentration of carbon dioxide in the gas phase in the tonometer was determined by analysis by the Haldane technique. Care was taken during the equilibration period to ensure that the total gas pressure within the tonometer equalled atmospheric pressure. The relationship between the carbon dioxide tension of the gas phase and the measured tension of carbon dioxide in the corresponding blood sample is shown in Fig. 2-25. The values lie closely along the line of perfect correlation. The mean error of 10 determinations was a blood carbon dioxide tension 0.17 mmHg greater than that of the gas. The standard error around this mean error was ± 0.95 mmHg. Thus there was no significant bias in the difference between the measured carbon dioxide tension and the carbon dioxide tension in the gas in equilibrium with the blood.

CARDIOVASCULAR TECHNIQUES

Intravascular pressure – Arterial and venous pressures were obtained by direct measurement using a strain gauge pressure transducer (Statham Type P.23Gb), an appropriate amplifier and a galvanometer photographic recorder. Arterial puncture was performed with a Riley needle as described previously for the collection of samples of blood. Venipuncture was performed after the local infiltration of 2% lignocaine using a 20 S.W.G. hypodermic needle. The lumen of the needle was then connected by means of a polyethylene cannula (internal diameter 0.5 mm) to one arm of a two-way tap which was also connected to the pressure transducer. The other arm of the fluid-containing head of the pressure transducer was connected through a second two-way tap to a fine adjustable needle valve. A bottle of sterile normal saline containing 2000 units of heparin per 100 ml was attached to the inlet of the needle valve. The pressure in the saline reservoir was raised to

at least three times the expected maximum vascular pressure by means of an air pump.

Before the pressure transducer was connected to the intravascular needle the needle valve was adjusted so that a very slow stream of saline (1–2 ml per minute) flowed through the transducer. This flow of heparin and saline was maintained whenever the vascular pressure was not recorded in order to prevent clotting of blood in the lumen of the needle. Whilst the pressure was actually recorded the flow of saline was diverted by turning the tap between the needle valve and the transducer so that the arm leading to the transducer was closed. At intervals whilst the intravascular pressure was being recorded the tap between the transducer and the intravascular needle was turned so that the transducer was connected to the environment through the side arm of this tap. This manoeuvre provided a zero reference pressure on the photographic record. Before and after each experimental procedure the output of the pressure transducer was calibrated against a mercury manometer. The deflections of the galvanometer were always found to be related linearly to the pressure applied to the transducer.

The ability of the measuring system to follow faithfully the rapid changes of pressure which occur in an artery was determined before each group of experiments. The behaviour of the whole recording system from the tip of the intra-arterial needle to the recording galvanometer was investigated by applying at the needle tip a constant amplitude pressure change, which was varied in a sinusoidal manner at frequencies between 1 and 30 c/s. These pressure changes were produced by a small piston driven by an electric motor acting upon air enclosed in a cylinder which had a capacity of 10 ml. The stroke volume of the pump was constant but the frequency could be varied at will. Preliminary experiments in which the Statham pressure transducer was connected directly into the cylinder and in which the whole recording system was filled with air showed that the amplitude of the pressure change produced by the pump was constant between frequencies of 1 and 50 c/s. When the saline-filled recording system described in the previous paragraph was connected, it was usually found that at frequencies between 10 c/s and 20 c/s the amplitude of the galvanometer was increased over that at 1 c/s. The frequency at which the amplitude was unchanged was increased by introducing a greater degree of damping of the fluid system by decreasing the diameter of the needle by which the polyethylene cannula was attached to the two-way tap fitted on the head of the transducer. Fourier wave analysis of records of arterial pressure waves suggests that a reasonable level of accuracy may be achieved by a recording system in which there is no distortion at frequencies of less than 5 c/s and in which the distortion does not exceed 5%, of the amplitude at a frequency of 15 c/s (141) (216). The degree of damping of the system was varied by changing the diameter of the connecting needle and of the polyethylene cannula until the distortion did not exceed 5% at a frequency of 15 c/s.

Indirect arterial blood pressure -- During most of the exposures to reduced environmental pressure performed in this study the arterial blood pressure was measured intermittently by a modified sphygmomanometric technique so that an indication of the cardiovascular response of the subject to his environment was immediately available. A standard arterial occlusion cuff 14 cm
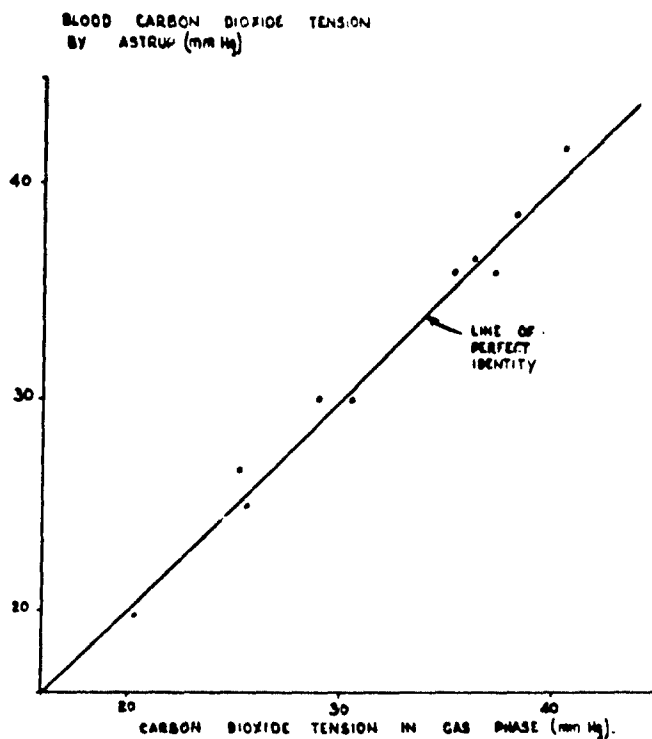
FIG. 2-25 The accuracy of the Astrup technique for measuring the carbon dioxide tension of blood. The result of each measurement by the Astrup technique has been plotted against the corresponding carbon dioxide tension in the gas with which the blood was equilibrated
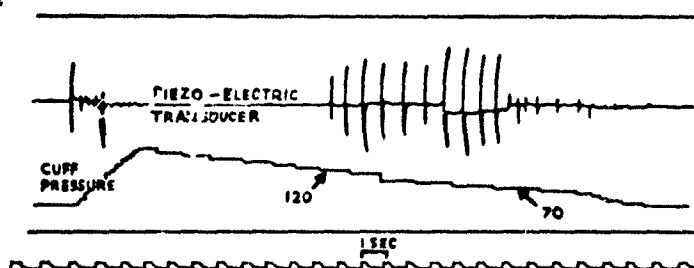
FIG. 2-26 A record of the output of the piezoelectric transducer and of the sphyg-momanometer cuff pressure during the determination of the arterial blood pressure of a subject at rest

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wide was placed around the upper arm. The movements of the wall of the brachial artery beneath the cuff were detected by means of a piezoelectric transducer attached to the inner surface of the cuff 3 cm from its lower border. The transducer consisted of a flexible diaphragm, the movement of which deformed a piezoelectric crystal (1 cm $\times$ 1 cm $\times$ 0.2 cm) mounted within a small housing, one side of which was the flexible diaphragm. The position of the brachial artery was detected by palpation and the cuff was positioned so that the piezoelectric transducer lay directly over the artery. The output of the piezoelectric crystal was amplified and fed on to one pen of a four-channel direct writing paper recorder.

The pressure in the cuff was recorded by means of a mercury manometer into which a series of platinum wire contacts had been introduced at 10 mm intervals. Each contact was connected by a resistor to its neighbour and a potential of 90 volts was placed across the whole length of the column. As the cuff pressure increased the mercury in the manometer rose and reduced the total resistance of the series of resistances. The current which passed through the circuit was recorded on a second pen of the direct ink writer. The record produced by this system consisted of a series of steps each of which represented a change of pressure of 10 mmHg. The pressure in the cuff was raised every thirty seconds to a level about 50 mmHg greater than the expected systolic pressure. The air in the cuff was then allowed to escape so that the pressure in it fell at a constant rate of about 5 mmHg/sec. until the pressure was less than the diastolic value. As the pressure in the cuff fell the piezoelectric transducer generated a signal with each cardiac cycle, the amplitude of which increased progressively until suddenly the signal virtually disappeared (Fig. 2–26).

Simultaneous records of intra-arterial pressure and of the output of the piezoelectric transducer showed that the cuff pressure was equal to the systolic pressure at the point at which the first signal was generated by the transducer. The pressure in the cuff equalled the diastolic pressure at the point at which the amplitude of the signal suddenly decreased. Direct comparisons of the values of the arterial blood pressure given by this indirect technique with those obtained by intravascular recording at rest and during pressure breathing showed that the indirect method gave values for both systolic and diastolic pressures which were within ± 5 mmHg of the directly determined values.

Limb Volume – The volumes of various segments of the limbs were measured during pressure breathing with water-filled plethysmographs. These experiments were performed in a draught free room, the temperature of which lay between 20° and 22°C. The subject, wearing the appropriate pressure clothing, was seated in a standard ejection seat. The straps of the seat harness were tightened firmly so as to reduce to a minimum the movement of the subject's limbs when his pressure clothing was inflated and deflated. Hand and forearm volumes were measured with simple metal plethysmographs. The hand was enclosed in a loose-fitting surgical glove, the wrist portion of which had been cemented to a thick rubber diaphragm, which fitted snugly around the wrist (168). This rubber diaphragm was secured to the end of the plethysmograph together with a metal supporting plate by means of wing nuts. When the forearm volume was to be measured two rubber diaphragms

were cut so that they fitted snugly around the upper and lower forearm respectively. The edges of these diaphragms were joined by a sleeve of very thin rubber (125). This sleeve loosely covered the forearm within the plethys-mograph. The diaphragms were supported in position by means of metal plates which also conformed to the shape of the forearm.

Care was taken to ensure that there was no actual constriction of the limb by the diaphragms or supporting plates. Each upper limb plethysmograph was placed so that the mid-plane of the segment lay 15 cm vertically below the sternal angle. Changes of thigh and calf volume were recorded by means of similar plethysmographs in which thin rubber sleeves separated the limb segment from the water in the plethysmograph. The lower limb in which measurements were made was supported in the horizontal position by means of wide webbing straps which passed behind the knee and ankle. The plethys-mograph was suspended by adjustable cords from the ceiling of the laboratory so that it was free to move with the limb when the pressure clothing was inflated and deflated (20).

When the plethysmograph had been fitted to the limb it was filled with water until the air-water interface lay in the lower part of the cylinder (3 cm in diameter) which was attached to the top of the plethysmograph. The upper end of the cylinder was open to the atmosphere. The temperature of the water within the hand plethysmograph was held at $32 \pm 0.5^\circ$C whilst that of the water in the forearm and lower limb plethysmographs was held at $34 \pm 0.5^\circ$C by intermittent heating (21). The change in the water level produced by a change of limb volume was recorded by means of a pair of vertical concentric electrodes inserted in the cylinder at the top of the plethysmograph (67). Changes in the impedance between the electrodes produced by alterations of the water level were recorded by means of a carrier-wave bridge amplifier, the output of which was fed on to a galvanometer of a photographic recorder. The volume recorded was calibrated by adding known volumes of water to the plethysmograph. The volume of the segment enclosed within the plethys-mograph was measured after each experiment either by water displacement into the case of the upper limb or from the physical dimensions of the part in the case of the lower limb segments.

LIMITATIONS OF VARIOUS TECHNIQUES
OF DELIVERING GAS UNDER PRESSURE
TO THE RESPIRATORY TRACT

INTRODUCTION

Gas may be delivered to the respiratory tract at a pressure greater than that of the immediate environment by means of a mouthpiece alone provided that the nose is closed with a suitable clip. Whilst this is the simplest method, it is generally impractical since severe discomfort rapidly occurs in the cheeks even at pressures as low as 20 mmHg. Further, at higher pressures the lips cannot be held against the mouthpiece and gas is lost through gaps between the mouthpiece and the lips. In practice the minimum acceptable standard for delivering gas under pressure is provided by a mask which covers the anterior part of the cheeks in addition to the mouth and nose (119). With a suitably designed oronasal mask (for example, the R.A.F. Type P mask) it is possible to deliver gas at positive breathing pressures of up to 100 mmHg without leakage of gas. With this equipment, however, no external support is applied to the eyes, to the external ear, to the floor of the mouth or to the neck during pressure breathing and disturbances of function arise in these regions.

The majority of these disturbances may be overcome by the use of a pressure headpiece designed to apply external support to the head and neck. All the pressure headpieces so far developed, however, have certain disadvantages when considered broadly as pieces of flying clothing. As compared with the simplicity of a pressure-sealing oronasal mask the weight, bulk and visual restriction of a pressure headpiece cannot but reduce the efficiency of the wearer during flight. The local functional disturbances induced by pressure breathing with an oronasal mask were studied, therefore, in order to assess the limitations in terms of breathing pressure and duration of exposure of this method of supplying gas under pressure to the respiratory tract.

GENERAL INVESTIGATION OF DISTURBANCES INDUCED IN THE HEAD AND NECK BY PRESSURE BREATHING WITH AN ORONASAL MASK

The type and incidence of the disturbances induced in the head and neck by pressure breathing with an oronasal mask were determined by exposing a group comprising four medical officers and sixteen aircrew to various breathing pressures. All the subjects had previous experience of pressure breathing at pressures of up to 80 mmHg using a pressure headpiece and a pressure jerkin. The experiments were performed at ground level. Each subject was clothed in a pressure jerkin and anti-g suit and fitted with a Type P oronasal mask. He was then exposed to pressure breathing for two minutes on four occasions, separated one from another by rest periods of five minutes duration. The positive breathing pressures employed were 40, 50, 60 and 70 mmHg and the order of the exposures was randomized. The amount of leakage of air

past the seal of the mask was determined during each exposure and if this exceeded 5 litre/min. or if gas flowed between the edge of the mask and the nose into the eyes, the exposure was discontinued and repeated after adjustment of the fit of the mask. Before and after each period of pressure breathing the conjunctivae and the tympanic membranes were inspected. Any changes in the head and neck induced by the procedure were recorded and after each exposure the subject was asked to describe his symptoms. In addition, the aircrew subjects were asked for their opinion as to the acceptability of the equipment as an emergency pressure breathing system under the conditions likely to be experienced following failure of the pressure cabin of an aeroplane flying at high altitude.

Results – All the twenty subjects accepted pressure breathing for two minutes at positive breathing pressures of up to 60 mmHg. The majority of the subjects, however, reported some discomfort during pressure breathing, especially at a pressure of 60 mmHg. At a positive breathing pressure of 70 mmHg severe discomfort and pain occurred in some of the subjects. The incidence of the various disturbances reported by the subjects is shown in Table 3–1.

TABLE 3–1

INCIDENCE OF SUBJECTIVE DISTURBANCES
INDUCED BY PRESSURE BREATHING
WITH AN ORONASAL MASK FOR 2 MINUTES

	Number of subjects affected from a group of 20			
	40 mmHg	50 mmHg	60 mmHg	70 mmHg
Eyes				
(i) Open nasolacrimal ducts	0	2	4	7
(ii) Blepharospasm	0	2	3	5
(iii) Impaired vision	0	1	1	2
(iv) Suffusion of the conjunctivae	0	1	2	7
Neck				
(i) Swelling	5	18	19	19
(ii) Discomfort	1	1	6	155
Ears				
(i) Discomfort	0	0	1	1

The commonest disturbances were related to the eyes, to the neck and to the floor of the mouth. At the higher positive breathing pressures the nasolacrimal ducts opened in some subjects and gas passed up the ducts through the lacrimal canaliculi into the conjunctival sacs. In these subjects the lacrimal secretions rapidly overflowed the lower lids and rolled down the cheeks. The stream of gas passing up the lacrimal ducts was associated with spasm of the eyelids which in one subject interferred with vision.
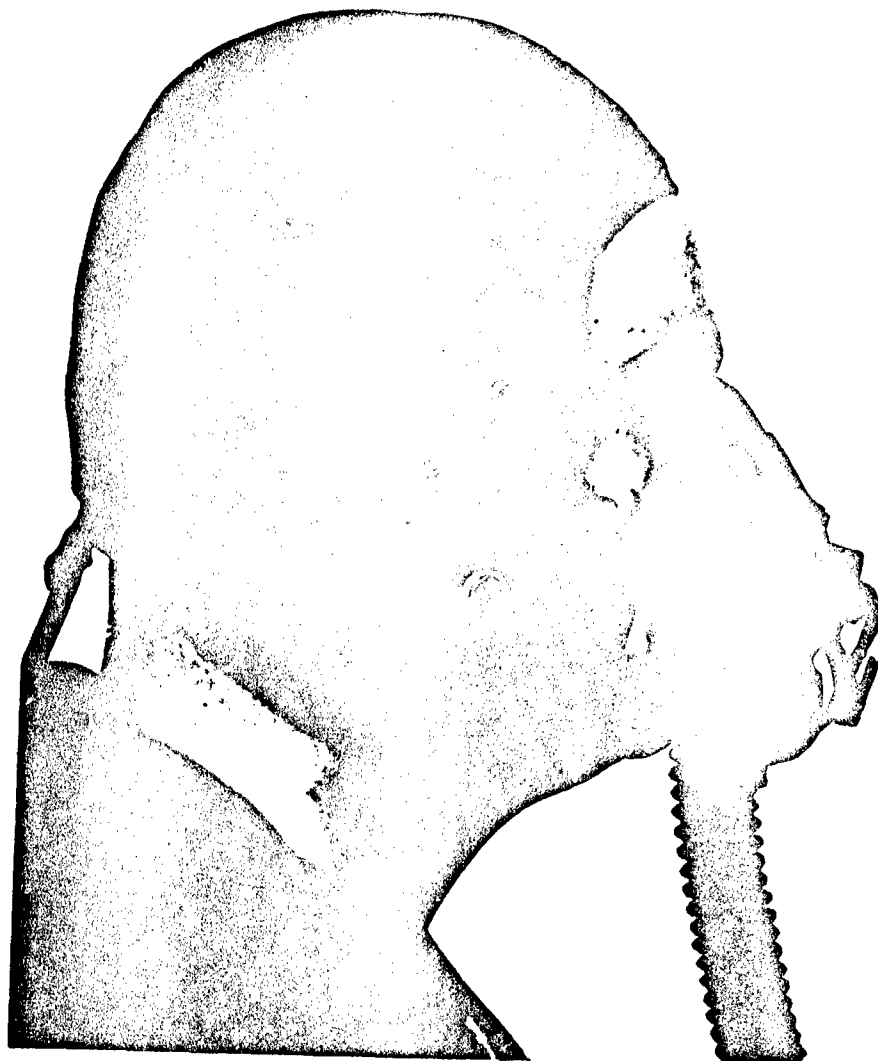
FIG. 3-1 Subject prior to pressure breathing
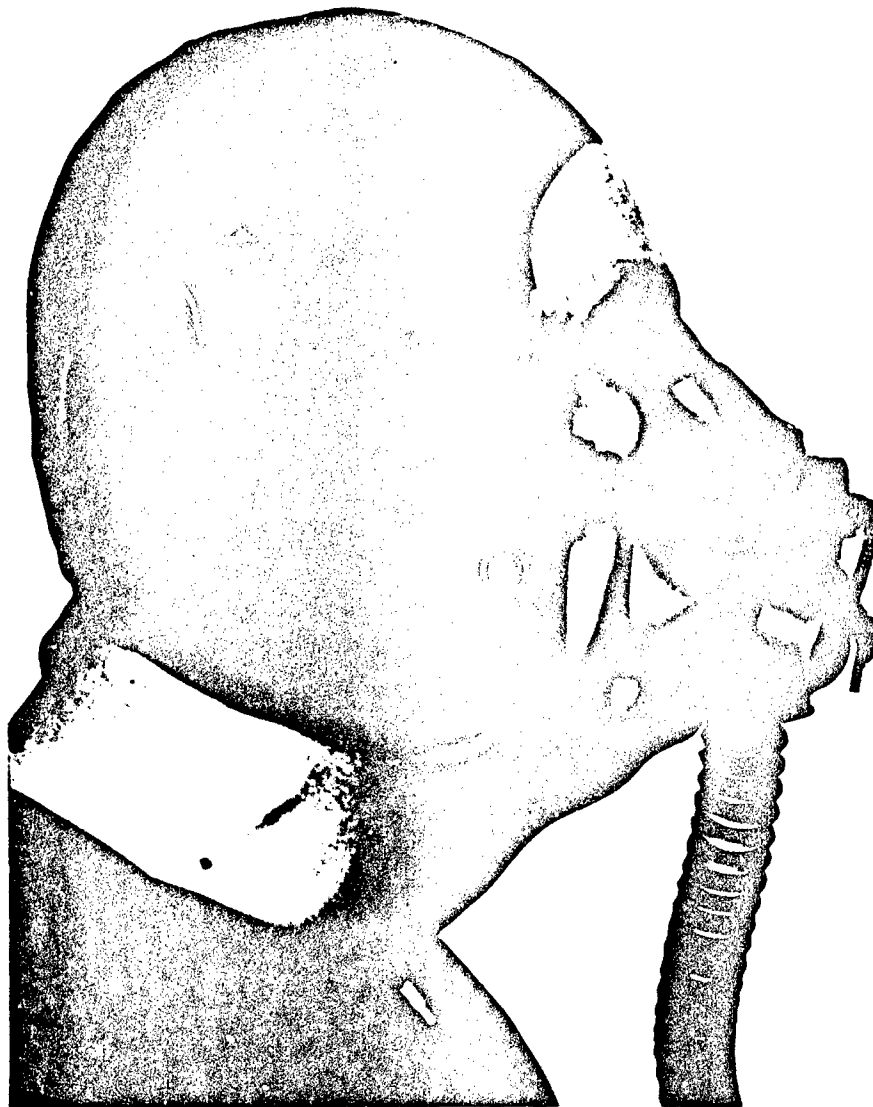
FIG. 3-2 Neck distension during pressure breathing at 70 mmHg pressure

Even at a positive breathing pressure of 40 mmHg there was very obvious distension of the neck and depression of the floor of the mouth in the majority of subjects. At the higher breathing pressures the distension of the neck increased further (Figs. 3-1 and 3-2) and at 60 and 70 mmHg it was associated with discomfort. This discomfort occurred in the floor of the mouth and pharynx. Occasionally the discomfort was severe but in the majority of subjects it was only moderate at a breathing pressure of 60 mmHg. At the highest positive breathing pressure studied, 70 mmHg, pain was reported by half the subjects and they considered that this symptom made this pressure unacceptable for use in an emergency pressure breathing system. Only one subject in this series of exposures experienced ear discomfort. This symptom arose when he swallowed during pressure breathing.

During exposure to positive breathing pressures in excess of 50 mmHg there was suffusion of the conjunctivae in a few of the subjects. The intensity of the conjunctival vascular dilatation was greater at the higher breathing pressures. In one subject whilst pressure breathing at 70 mmHg a small conjunctival haemorrhage occurred on the inner surface of the lower eyelid. There was marked distension of the superficial veins of the neck, the external jugular veins being especially prominent in most of the subjects. No changes were seen in the condition of the tympanic membrane when it was examined after an exposure to pressure breathing.

VISION AND THE APPEARANCE OF THE RETINA DURING PRESSURE BREATHING

A study of the effects of pressure breathing with an oronasal mask upon the visual acuity and the appearance of the retina was made in six subjects. Exposure to positive breathing pressures between 30 and 70 mmHg was carried out as in the previous group of experiments. Visual acuity was measured before, during and after each two-minute period of pressure breathing using Snellen's test types. The subject was placed 6 m. from the test card, and instructed to read the card from above (largest size letters) downwards. Since speech was difficult whilst pressure breathing at the higher pressures, the subject wrote down the letters which he could distinguish. Four Snellen text cards were used and they were presented to the subject in a random order. The retina was observed continuously by means of an electric ophthalmoscope before, during and after pressure breathing. Several drops of 2% homatropine were instilled into the conjunctival sac of one eye of each of two of the subjects in order to dilate the pupil.

Results – No change in visual acuity with pressure breathing as measured by the Snellen test card technique was detected in any of the six subjects, even at a positive breathing pressure of 70 mmHg. There were no changes in the appearance of the optic disc and the retina itself during pressure breathing. The calibre of the retinal veins was, however, slightly reduced during pressure breathing at 50 mmHg and 70 mmHg. No change in the retinal arteries was detected.

DISTENSION OF THE UPPER RESPIRATORY TRACT DURING PRESSURE BREATHING

The changes underlying the distension of the neck produced by pressure breathing with an oronasal mask were investigated by radiography and by measurement of the respiratory dead space.

Radiographic Studies – Lateral and antero-posterior radiographs were taken of the head and neck of two subjects. The radiographs were taken with the subjects sitting at rest and whilst pressure breathing at positive breathing pressures between 30 mmHg and 100 mmHg using an oronasal mask and a pressure jerkin. The distance between the X-ray tube and the film cassette was held constant throughout this study. In order to calibrate the radiographs so that various dimensions could be measured, a radiograph was taken of a lead bar notched at 2 cm intervals held in the same plane as that in which the subject's head was placed for the radiographic studies.

Results – The most striking feature of the radiographs taken during pressure breathing was the gross distension of the upper respiratory passages (Fig. 3-3). The mouth cavity and the lower pharynx shared in this distension. The cervical portion of the oesophagus which contained no air in the control radiographs was markedly distended with air in the radiographs taken during pressure breathing. The antero-posterior radiographs show that there was also gross lateral distension of the pharynx. There was virtually no dilatation of the trachea. The anteroposterior dimensions of the nasopharynx, the hypopharynx, the cervical oesophagus and the trachea have been measured at the levels depicted in Fig. 3-4, from the radiographs taken with the subject at rest and during pressure breathing. The relationships between these dimensions and the positive breathing pressure for one subject are shown in Fig. 3-5. The shape of the curves in this figure show that the distensibility of the pharynx and oesophagus is greatest at the lower breathing pressures and that there is little further increase in the antero-posterior dimensions of these cavities at positive breathing pressures in excess of 50 mmHg.

Respiratory Dead Space – The volume of the dead space was measured by the technique developed by Fowler (1948) (111) in which the instantaneous concentration of nitrogen in the expired gas is measured following a breath of oxygen. The sampling needle valve of the Lundin-Akesson nitrogen meter was fitted directly into the mouthpiece through which the subject breathed, whilst the nose was closed with a clip. A Fleisch flowmeter was attached directly beyond the sampling tube and a two-way tap was attached to the distal end of the flowmeter. One arm of this tap was open to the atmosphere and the other was connected to a bell spirometer. Before the apparatus was attached to the subject the spirometer and connecting tubing were filled with oxygen. The subject breathed atmospheric air to and fro through the mouthpiece, the attached sampling valve and the flowmeter. At the end of a normal exspiration he turned the tap and inspired one litre of oxygen from the spirometer. At the end of inspiration he held his breath for one second, during which the tap was turned so that the mouthpiece was opened to the atmosphere. The subject then breathed out in the normal manner. The two subjects used in this experiment had had previous experience of respiratory manoeuvres and were instructed to maintain the same

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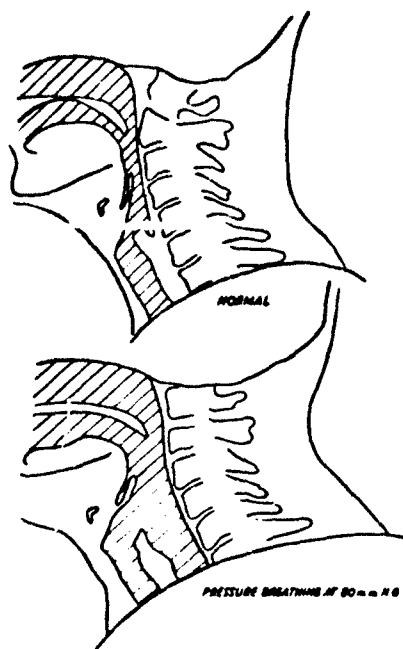
FIG. 3-3 Tracings of lateral radiographs of the head and neck taken with the subject at rest and whilst pressure breathing at a positive breathing pressure of 80 mmHg. The air-containing regions have been cross hatched
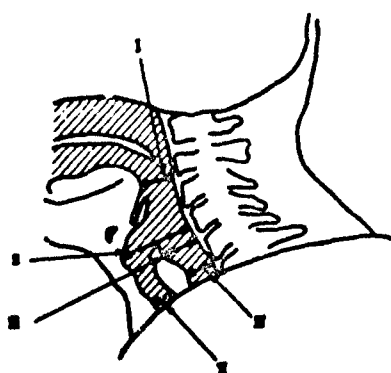
FIG. 3-4 A tracing of a lateral radiograph of the head and neck taken during pressure breathing showing the levels at which the antero-posterior diameters of the upper respiratory passages and oesophagus were measured

ANTERO - POSTERIOR DIAMETER
OF RESPIRATORY TRACT (mm)
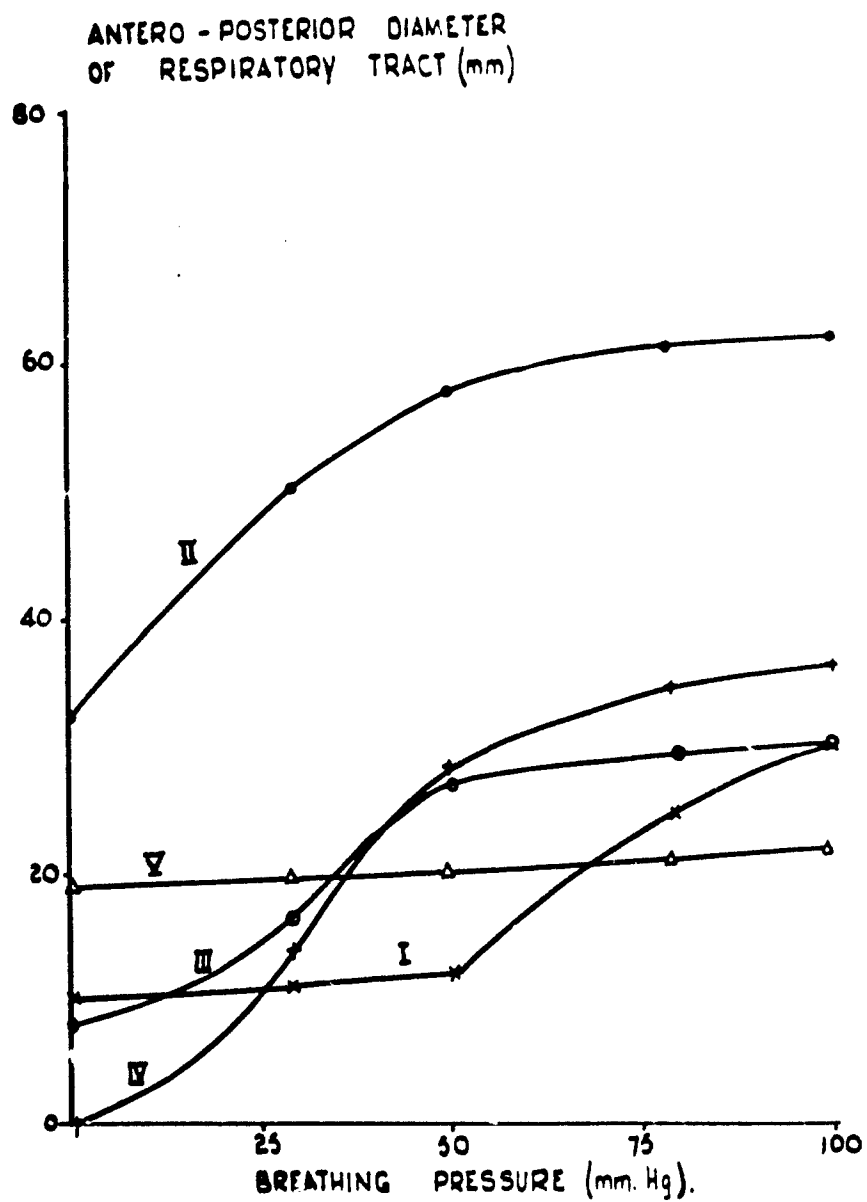
FIG. 3-5 The effect of pressure breathing with an oronasal mask upon the antero-
posterior dimensions of the upper respiratory tract

breathing pattern during each expiration. At least two minutes were allowed to elapse between each measurement of the respiratory dead space.

In order to assess the accuracy with which an increase in the respiratory dead space could be measured with this technique, a preliminary investigation was performed. Various lengths of 2.0 cm bore hose with a smooth internal wall were interposed between the subject's mouth and the needle valve of the nitrogen meter. Measurements of the overall dead space were made in exactly the manner described in the previous paragraph. The volume of each length of hose interposed between the subject's mouth and the needle valve was measured by water displacement.

The volume of the dead space was measured during pressure breathing with an oronasal mask and a pressure jerkin at positive breathing pressures of up to 60 mmHg. The oronasal mask was modified by incorporating the needle valve of the nitrogen meter in a short tube fitted to the entrance to the expiratory valve within the mask cavity. A Fleisch flowmeter was fitted in the outlet of the mask beyond the expiratory compensated valve. In order that a spirometer could be used to supply a known volume of oxygen, these experiments were performed with the subject seated in the decompression chamber. The spirometer filled with oxygen was mounted outside the chamber and the hose from it was passed through the chamber wall to one arm of a two-way tap fixed in the inspiratory port of the oronasal mask. The third arm of the tap was connected through the wall of the chamber to the external atmosphere. Pressure breathing was induced by reducing the pressure within the decompression chamber by the desired value. The respiratory dead space was measured in the same general manner as in the previous experiments. After pressure in the mask had stabilized the tap was turned and the subject inspired 1 litre of oxygen from the spirometer. The concentration of nitrogen and expired gas flow were recorded during the subsequent expiration.

The effect of a distending pressure within the upper respiratory tract upon the respiratory dead space was also studied by reducing the pressure in a perspex box enclosing the neck. This box (Fig. 3–6) encircled the neck and was sealed at its upper end against the skin along the mento-suboccipital diameter. The lower edge of the box was sealed against the skin lying over the upper part of the chest anteriorly and posteriorly and just medial to the shoulder joints laterally. The box was connected via a two-way tap to a large capacity reservoir, in which the pressure was maintained at known subatmospheric levels. The pressure within the perspex box was measured by means of a "U" mercury manometer. The capacity of the exhaust pump acting on the reservoir and the volume of the reservoir were such that the pressure within the perspex box was maintained at the required level below atmospheric despite small leakages of air inwards past the rubber seals. Subatmospheric pressures of up to 80 mmHg were applied to the neck. After a given subatmospheric pressure had been applied for thirty seconds the respiratory dead space was measured and then the pressure within the box was returned to atmospheric.

Results – A typical experimental record is reproduced in Fig. 3–7, showing the instantaneous concentration of nitrogen and the respiratory flow pattern during and following a single inspiration of oxygen. The volume of the dead space was estimated from such experimental records by the technique
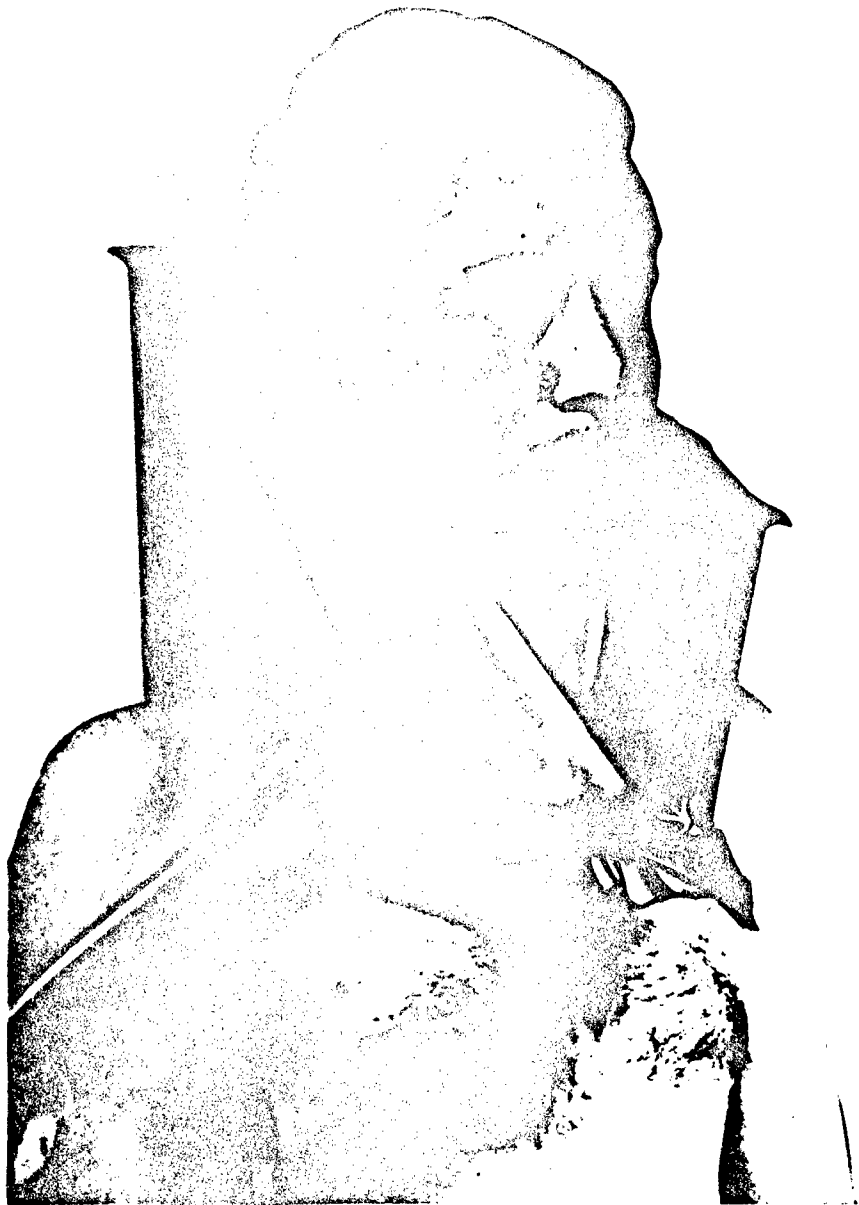
FIG. 3-6 Perspex box for maintaining subatmospheric pressure around neck
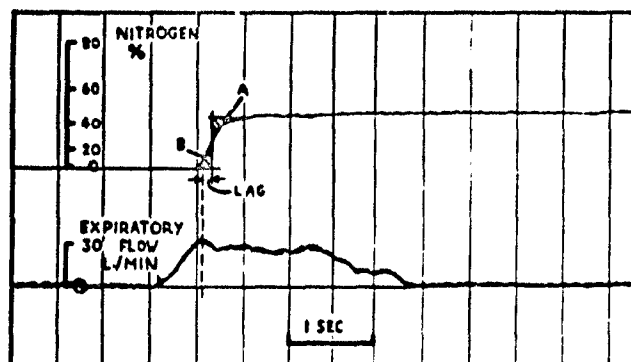
FIG. 3-7 The determination of the respiratory dead space. Records of the respiratory flow and of the concentration of nitrogen in the expired gas following a single inspiration of oxygen
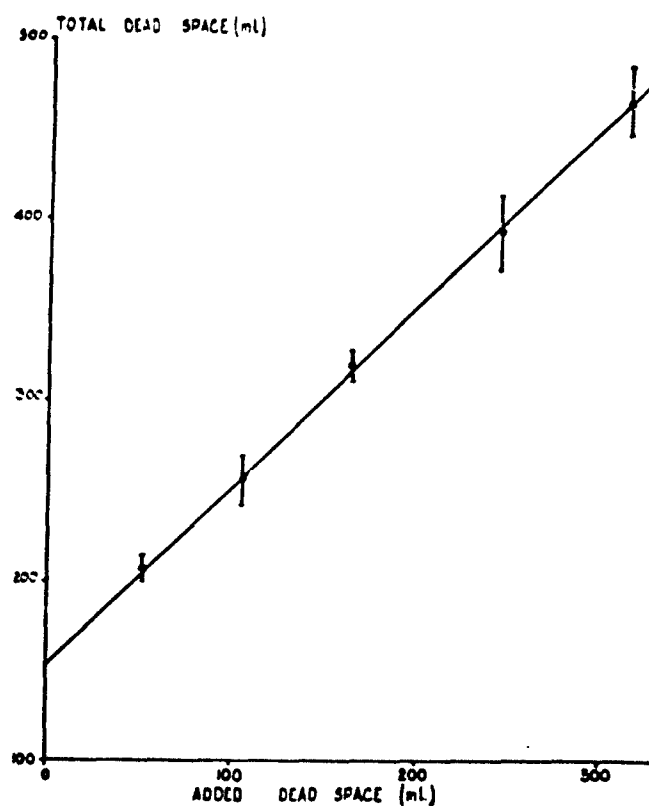
FIG. 3-8 The effect of introducing various lengths of smooth bore hose of known volume between the lips and the sampling valve of the nitrogen meter upon the measured total respiratory dead space

73
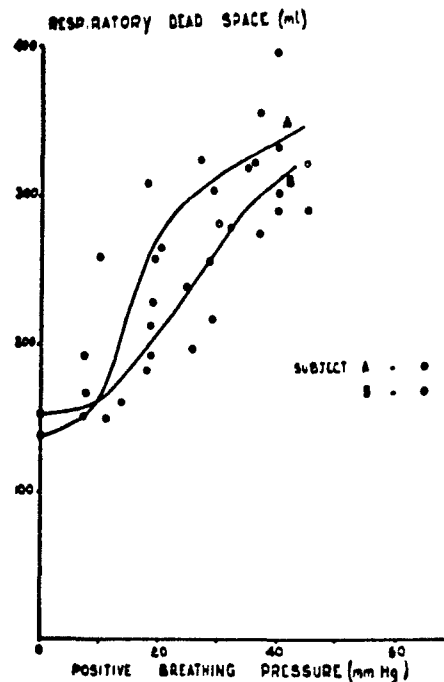
Fig. 3–9 The effect of pressure breathing with an oronasal mask and pressure jerkin upon the respiratory dead space in two subjects
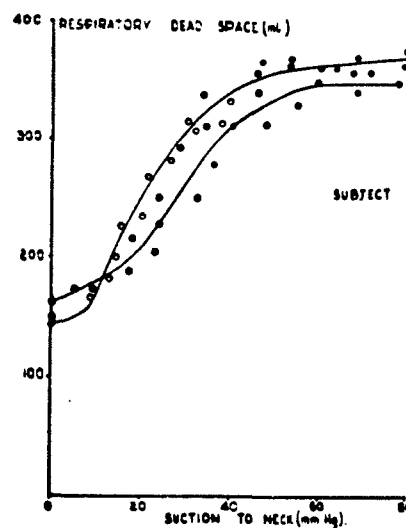
Fig. 3–10 The effect of the application of various sub-atmospheric pressures to the surface of the neck upon the volume of the respiratory dead space in two subjects

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described by Fowler (111). A vertical line was drawn on the recorded nitrogen concentration curve at the point at which it was rising rapidly so that the areas A and B (Fig. 3-7) were equal. A squared transparent ruler was used for this estimation. The lag of the nitrogen meter response behind that of the flow-meter which was determined at the end of each series of measurements (mean 0.08 sec.) was subtracted from the instant described by the vertical line through the nitrogen meter record and the resulting line extended across the flow record. The volume of gas expired up to this instant was determined by measuring the area enclosed by the expiratory flow curve to the vertical line already described by means of a planimeter. The true dead space was then obtained by subtracting from this value the volume of the instrumental dead space (35 ml). Ten measurements of the dead space in one subject at rest gave a mean value of 154 ml B.T.P.S. with a standard deviation of $\pm$ 10 ml. The relationship between the volume of the various lengths of smooth-bore tubing added as external dead space (ranging from 52 ml to 315 ml) to the corresponding total dead space volumes is shown in Fig. 3-8. Each point represents the mean of at least five measurements and the length of the bar through each point depicts the magnitude of twice the standard deviation of a single determination. The correlation coefficient between added dead space volume and the total dead space volume was 0.98 (N = 28). The slope of the linear regression line fitted to this data with the added external dead space volume as the independent variable was 1.04.

When the respiratory dead space was measured during pressure breathing with an oronasal mask several difficulties were encountered. It was extremely difficult to ensure a complete seal between the edge of the mask and the subject's face at positive breathing pressures greater than 30 mmHg. During these measurements even a leak which was usually quite acceptable (e.g. 1 litre/min.) resulted in the loss of a significant volume of gas from the mask during the period in which the dead space gas was expelled from the respiratory tract. The pattern of the expiratory flow of gas following the breath of oxygen varied considerably from one determination to another during pressure breathing. The dead space of the mask (85 ml) was subtracted from each measured value to give the true respiratory dead space. The results of the technically satisfactory measurements of the dead space volume during pressure breathing of the two subjects who were studied are presented in Fig. 3-9. There was a considerable variation between individual measurements in these experiments. The general trend, however, was that there was little change in dead space volume at breathing pressures of up to 10 mmHg and that at higher breathing pressures the volume of the dead space increased markedly. No technically satisfactory measurements were obtained at positive breathing pressures in excess of 45 mmHg owing to the presence of mask leaks at the higher pressures.

The results of the measurements of the respiratory dead space when various subatmospheric pressures were applied to the surface of the neck by means of the perspex box for the two subjects studied are presented in Figs. 3-10. The shapes of the pressure distension curves obtained in the two subjects were very similar. When the pressure difference applied across the walls of the upper respiratory passages was small (less than 10 mmHg), there was little change in the volume of these passages. A further increase in this pressure difference,

however, caused a large increase in respiratory dead space. A pressure difference of about 50 mmHg produced nearly maximal distension over the range of pressures studied. The increase in the volume of the dead space produced by a reduction of the pressure in the box of 50 mmHg was approximately 200 ml B.T.P.S. in both the subjects.

AUDITORY ACUITY AND THE APPEARANCE OF THE TYMPANIC MEMBRANE DURING PRESSURE BREATHING

Auditory Acuity – The auditory acuity of six subjects was measured at rest and whilst pressure breathing with an oronasal mask and a pressure jerkin at a positive breathing pressure of 60 mmHg at ground level. The subject, who wore a standard flying helmet (R.A.F. Type G), fitted with standard inductive type telephones, was seated in a decompression chamber. Pure tones at frequencies of 250, 500, 1000, 2000 and 4000 c/s were produced in the subject's telephones by means of an oscillator, the power output of which could be varied in known steps. The subject was provided with a signal key which he operated when he perceived a sound. The output of the oscillator was fed to the telephones by way of a second key. After the frequency and power output of the oscillator had been adjusted to the desired values this key was closed for a period of two seconds. At each of the chosen frequencies the threshold of auditory acuity was determined approximately by progressively decreasing the power of the signal until the subject failed to perceive it. The subject's threshold was then determined accurately by applying the signal at a known suprathreshold level for two seconds and then decreasing the power in 3 db steps until he did not hear it.

There were two sources of noise within the decompression chamber which interfered with the determination of the auditory threshold. One source was the vacuum pumps by means of which the pressure in the chamber was reduced. The noise from this source was eliminated by pumping the air from a large cylindrical reservoir before the measurements commenced and maintaining a constant pressure within the decompression chamber by allowing air to pass from it into the evacuated reservoir. The valves within the oronasal mask worn by the subject also generated considerable noise. This source of interference was eliminated by removing the valves from the mask and connecting the mask cavity to a canister containing soda lime by a short length of hose and a two-way tap. The distal end of the canister was connected by a short length of wide-bore hose to a Douglas bag filled with oxygen which was placed outside the decompression chamber. The subject breathed to and fro through the canister to the Douglas bag.

In these experiments the pressure within the decompression chamber was reduced to 60 mmHg less than the prevailing barometric pressure, whilst the subject breathed air from within the decompression chamber. The subject was instructed to maintain the patency of his pharyngo-tympanic tubes and he remained at the slightly reduced pressure for five minutes before the control measurements of auditory acuity were made. Pressure breathing was then instituted by connecting the pressure jerkin and oronasal mask to the exterior of the chamber by turning the appropriate taps. The auditory acuity of the subject was determined during the second and subsequent half minutes of the exposure to a breathing pressure of 60 mmHg. The acuity measure-

ments were repeated after the subject had swallowed several times. Measurements of auditory acuity were also made at simulated altitudes of 38000 ft and 50000 ft in the decompression chamber. The subject wore the standard type P mask, pressure jerkin and anti-g suit assembly which was connected to an automatic pressure demand regulator Mark 20.

The subject was decompressed to 38000 ft at 5000 ft per minute and then the auditory threshold was measured by the technique used in the ground level experiments whilst the subject breathed oxygen at a pressure of 1 to 2 mmHg greater than that within the decompression chamber. This was followed by recompression to a pressure altitude of 25000 ft during which the subject maintained the patency of his pharyngo-tympanic tubes. After three minutes had elapsed the subject was rapidly decompressed to 50000 ft in one second. Auditory acuity was measured as rapidly as was practicable whilst the subject breathed oxygen at a positive breathing pressure of 60 mmHg at 50000 ft and then the subject was rapidly recompressed to 38000 ft. After a further three minutes at this pressure altitude, a final determination of auditory acuity was made and the subject was then recompressed to ground level. The pressure within the decompression chamber was maintained at the desired value whilst acuity measurements were made by the technique used in the initial experiments at ground level. The intensity of the noise generated by the valves of the oronasal mask at 38000 ft and 50000 ft was negligible.

Appearance of the tympanic membrane – The tympanic membrane of one ear was continuously observed in twenty subjects whilst they were exposed to positive breathing pressures of up to 70 mmHg with an oronasal mask and a pressure jerkin. Each exposure lasted for two minutes. The tympanic membrane was observed by means of an electric auroscope through a hole made in the ear bun of a standard flying helmet.

The effect of pressure breathing at positive breathing pressures greater than 70 mmHg upon auditory acuity and the appearance of the tympanic membrane was determined by using a partial pressure headpiece to apply air under pressure to the respiratory tract. In the R.A.F. types of partial pressure headpiece there is no direct pressurization of the external auditory meatus. Measurement of the pressure within the external auditory meatus was made at positive breathing pressures of up to 140 mmHg by means of a fine polythene catheter (O.D. 0.7 mm) placed in the meatus and brought out through the bladder of the headpiece overlying the ear. The meatal pressure measurements which were made with a capacitance manometer showed that the rise in meatal pressure was less than 10% of the pressure applied by means of the headpiece to the respiratory tract.

Fifty subjects wearing a pressure headpiece, jerkin and anti-g suit were exposed to pressure breathing at 80 mmHg for four minutes on two occasions and at 100 mmHg for two minutes on two occasions. Further a group of ten subjects were exposed to a positive breathing pressure of 80 mmHg for twenty minutes and one of 115 mmHg for fifteen minutes whilst wearing a partial pressure headpiece, arm jerkin and anti-g suit.

RESULTS

Auditory Acuity – The changes of auditory acuity produced by pressure breathing under the various conditions studied were consistent from one subject to another. The mean values of the reduction of acuity relative to the level obtained at rest at ground level from the group of six subjects are presented in Fig. 3–11. Increasing the pressure in the respiratory tract by 60 mmHg at ground level did not produce any overall change of acuity until the subject swallowed. This manoeuvre when performed during pressure breathing resulted in a considerable loss of acuity, particularly at the lower frequencies. Breathing oxygen at the pressure of the environment at a simulated altitude of 38000 ft resulted in some loss of auditory acuity which was most marked at the lower frequencies. There was a further loss of acuity when the subject was exposed to a positive breathing pressure of 60 mmHg at a simulated altitude of 50000 ft. The absolute pressure within the respiratory tract was virtually the same during breathing oxygen at 38000 ft and during pressure breathing at 50000 ft.

Appearance of the tympanic membrane – Direct observation of the tympanic membrane during pressure breathing with an oronasal mask showed that no changes occurred in this organ in nineteen of the twenty subjects examined. In one subject there was a general bulging of the drumhead outwards at the commencement of pressure breathing. A similar change arose during pressure breathing in the other subjects when the subject swallowed. This movement affected the whole membrane but was greatest in the pars flaccida. The bulging of the membrane following a swallow was associated with discomfort in the ear and a subjective reduction of auditory acuity. No changes were seen in the appearance of the tympanic membrane in the fifty subjects who were examined before and after exposure to pressure breathing for short periods at positive breathing pressures of 80 to 100 mmHg whilst wearing partial pressure headpieces.

In the group of experiments in which the duration of pressure breathing was prolonged, marked changes were seen in the appearance of the tympanic

TABLE 3–2

INCIDENCE OF EAR CHANGES AFTER PRESSURE BREATHING FOR
LONG PERIODS

Condition	Total No. of Subjects	Injection of External Auditory Meatus	No. of subjects showing Petechial Haemorrhages in the[1] Tympanic Membrane			Fluid in Middle Ear
			†	††	†††	
80 mmHg for 20 mins	10	7	2	3	2	5
115 mmHg for 15 mins	10	8	2	3	3	6

[1] † → ††† depict increasing intensity of change

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HEARING LOSS (db)

FREQUENCY (c/s)

PRESSURE   BREATHING   AT G L.   ▲
PRESSURE   BREATHING   AT G.L (AFTER   SWALLOWING)   ●
BREATHING   OXYGEN   AT   38,000 FT.   ✗
PRESSURE   BREATHING   AT 50,000 FT.   ○
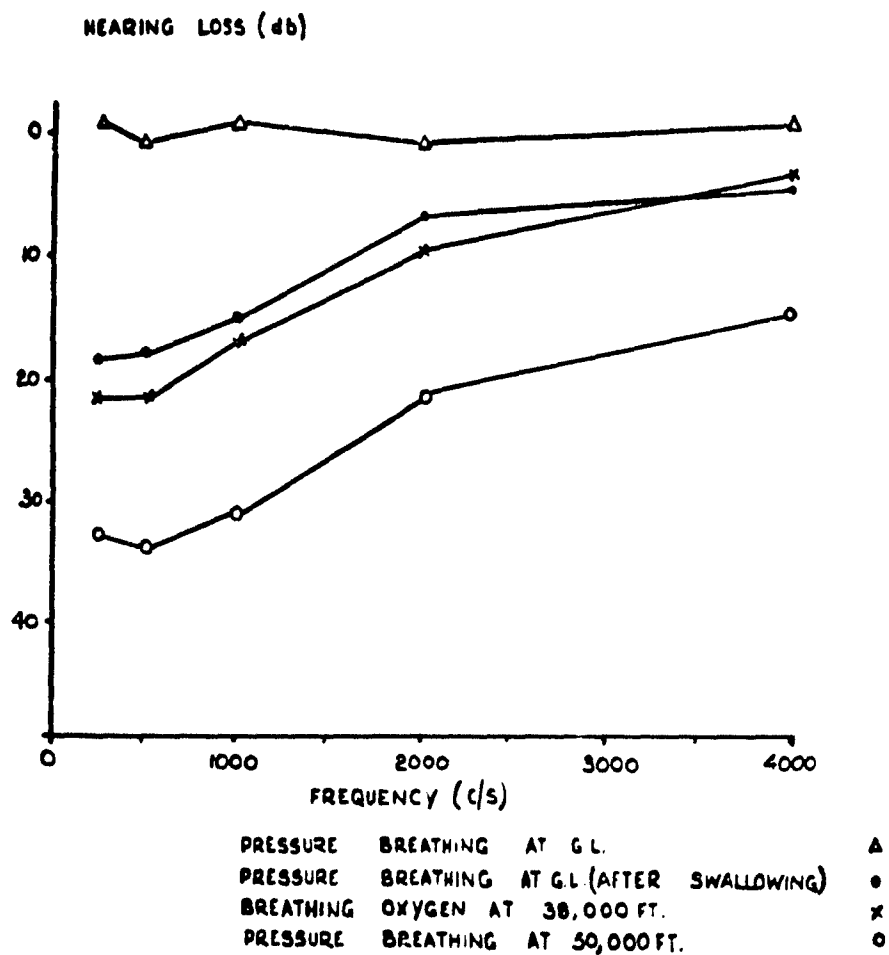
FIG. 3-11 The effect of pressure breathing and reduction of environmental pressure
upon the auditory threshold measured relative to the threshold at rest at ground
level. Each point represents the mean of the values obtained from six subjects

membranes after pressure breathing. Typically the inner part of the external auditory meatus and the tympanic membrane were injected; the membrane was covered with petechial haemorrhages and there was a fluid level in the middle ear. Not all the subjects showed all the changes. The incidence of the various signs is presented in Table 3-2. It may be seen that the incidence and intensity of the changes were slightly greater in the exposure to a positive breathing pressure of 115 mmHg for fifteen minutes than in the twenty-minute period of pressure breathing at 80 mmHg. One subject, two minutes after the start of an exposure to a positive breathing pressure of 115 mmHg experienced discomfort in his right ear and the exposure was ended. Examination of the ear revealed blood in the external auditory meatus and this was found to be coming from a ruptured haemorrhagic bulla in the antero-inferior sector of the membrane. The membrane itself was not perforated.

THE CARDIO-VASCULAR EFFECTS OF NECK COUNTERPRESSURE DURING PRESSURE BREATHING

The response of the cardiovascular system to the application and removal of counterpressure to the neck was studied during pressure breathing with an oronasal mask and a pressure jerkin. Pressure was applied to the neck by means of an inflatable rubber bladder made in the form of a collar. The bladder was shaped so that its upper border fitted up beneath the ears and it was secured in this position by means of adhesive tape. The lower border of the bladder reached the root of the neck. The bladder was covered by an inextensible layer of fabric which adjusted to fit the wearer's neck by means of lacing. The size of the bladder and the closeness of the restraining layer were selected to ensure that when the bladder was inflated the walls of the bladder were not overdistended. The bladder was inflated with compressed air by means of a 2 cm I.D. hose and a two-way tap. The pressure within the bladder was recorded by means of a capacitance manometer. The neck bladder was usually inflated at the commencement of a period of pressure breathing to the same pressure as that which was applied to the respiratory tract. The pressure within the neck bladder was suddenly reduced one minute after the beginning of pressure breathing. The effects of suddenly increasing the neck bladder pressure to its original value were also studied. All the experiments were performed with the subject in the seated position.

In the majority of experiments lead II of the electrocardiogram was recorded continuously. The arterial pressure responses to deflation and inflation of the neck bladder were measured by electromanometry by way of a Riley needle inserted in the brachial artery. The blood flow through the forearm was measured by means of a Whitney strain gauge (286) with an occlusion cuff placed around the wrist and a collection cuff around the upper arm just above the elbow (Fig. 3-12). The gauge was placed on the forearm 6 cm from the tip of the olecranon and held in position against the skin by two pieces of adhesive plaster. The upper limb was placed within a perspex box with a reflected rubber seal around the upper arm just proximal to the congestion cuff. The seal consisted of a thin cuff of rubber which was attached to the edge of a hole just large enough to contain the arm without constriction. The seal was reflected along the upper limb towards the elbow and its free

REFLECTED EDGE SEAL

COLLECTING CUFF

AMPLIFIER

WHITNEY GAUGE

WRIST OCCLUSION CUFF

PIPE CONNECTED TO BREATHING LINE

M

R

M

R

RESERVOIR

RESERVOIR
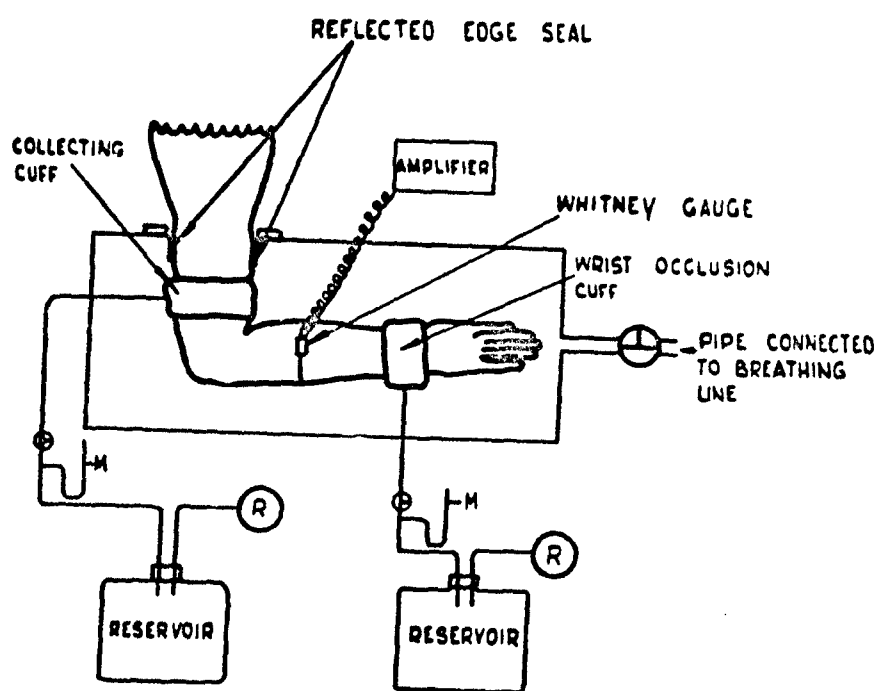
R - VARIABLE PRESSURE REGULATOR
M - MERCURY MANOMETER.

FIG. 3-12 The apparatus used for the measurement of forearm blood flow during pressure breathing

edge was fixed to the skin by means of adhesive plaster. The congestion cuff was applied over the seal.

The outer surface of the rubber sheet was supported by means of a perspex plate, the inner edge of which was shaped to fit closely against the skin of the upper arm without producing actual constriction. The limb was supported comfortably within the box with the elbow flexed by means of a padded block beneath the elbow and with the hand resting on a second block. The box was positioned so that the lower border of the forearm was at the horizontal level of the suprasternal notch. The wrist occlusion cuff was connected to a mercury manometer and an inflation pump outside the box. The collection cuff was connected by way of a wide bore two-way tap and hose to a large reservoir (100 litre), the pressure in which was maintained at the desired value by means of a demand regulator with a variable output pressure. During pressure breathing the box enclosing the upper limb was pressurized to the same pressure as that which was applied to the respiratory tract. In order to reduce to a minimum the displacement of the upper limb from the box when the latter was pressurized the subject was firmly secured in the seat and the box fixed relative to the seat.

When the apparatus was correctly positioned the subject rested in the quiet for ten minutes. Following the rest period measurements were made of the forearm blood flow. The wrist occlusion cuff was inflated to 300 mmHg one minute before the measurement of blood flow. The collecting cuff was then inflated for five seconds every twenty seconds and the consequent increase in forearm volume recorded. As a preliminary investigation at the beginning of each experiment the pressure within the collecting cuff was varied between 30 and 60 mmHg until the collecting pressure which yielded a constant rate of increase of forearm volume was found. When the subject was pressure breathing and the pressure within the box enclosing the upper limb had been raised, the pressure to which the collecting cuff was inflated was increased by an amount equal to the pressure applied within the box. In this manner the relationship between the pressure within the collecting cuff and the venous pressure in the forearm was unchanged throughout the experiment. During pressure breathing the neck bladder was deflated and inflated and the corresponding forearm blood flows were recorded.

Results – A reduction of the pressure within the neck bladder whilst the subject was pressure breathing generally caused a transient slowing of the heart rate (Fig. 3–13). There was a marked variation between the response of the heart rate to the same reduction of neck bladder pressure between one individual and another. In four out of the six subjects studied there was bradycardia following deflation of the neck cuff (Fig. 3-14) whilst the remaining two subjects exhibited no change of heart rate in this situation. The minimum pulse rate occurred within one or two beats of the reduction of cuff pressure. The pulse rate then increased to regain the prestimulus value five to fifteen seconds later. At a given positive breathing pressure the magnitude and duration of the slowing were related to the amount by which the pressure within the neck bladder was reduced (Fig. 3–15). A fall of bladder pressure of between 15 and 25 mmHg was found to be the smallest stimulus which produced an effect on the heart rate. The greater the fall in neck bladder pressure the greater was the degree and duration of the subsequent brady-
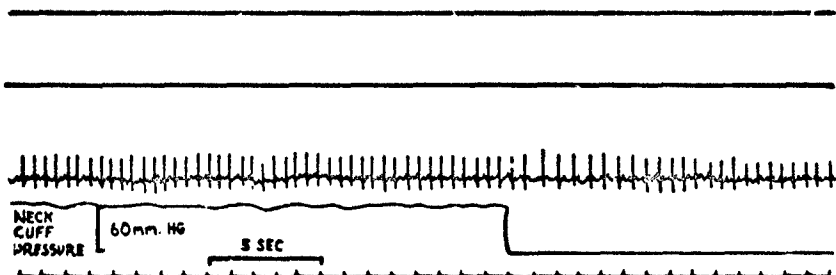
Fig. 3-13 The effect of sudden deflation of the neck bladder in a subject pressure breathing at 60 mmHg upon the electrocardiogram (Lead 11)
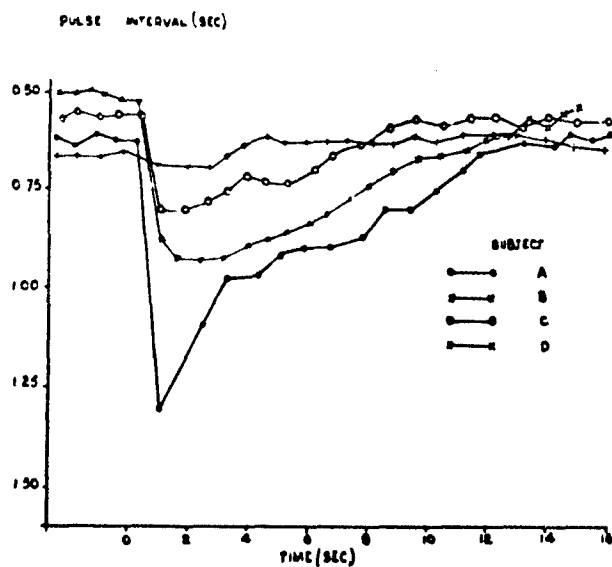
Fig. 3-14 The effect of sudden deflation of the neck bladder during pressure breathing at 80 mmHg upon the heart rate (expressed as the pulse interval) in four subjects

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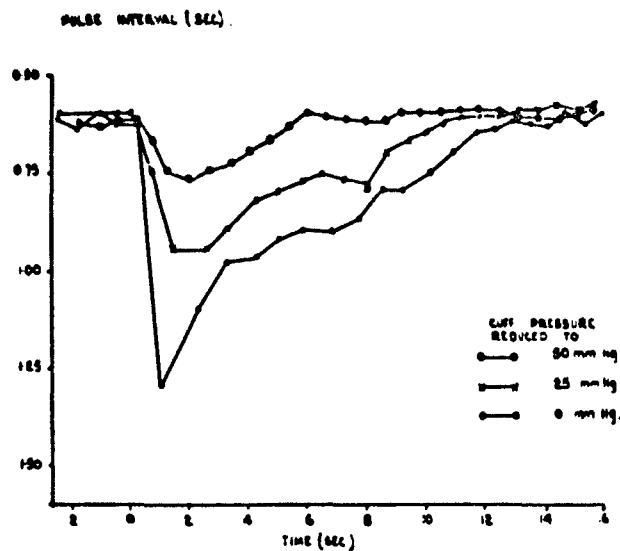
Fig. 3-15 The effect upon the heart rate (expressed as pulse interval) in one subject of suddenly reducing the pressure in the neck bladder from 80 mmHg to 50, 20 or 0 mmHg whilst the subject was pressure breathing at a positive breathing pressure of 80 mmHg
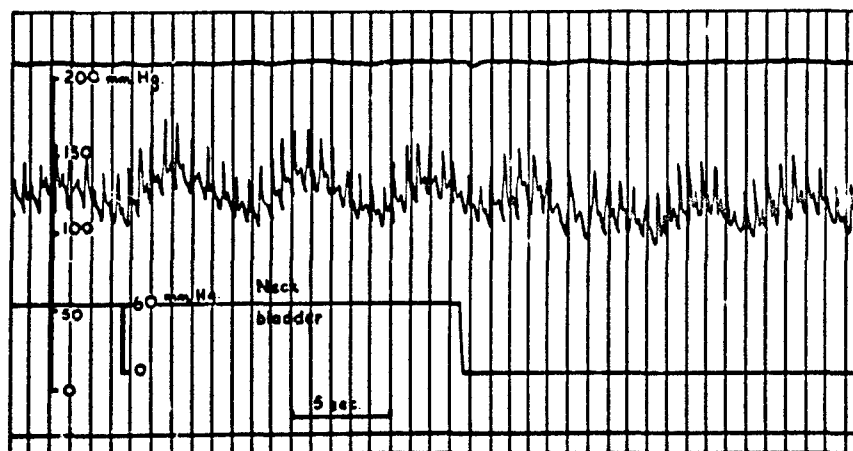
Fig. 3-16 The effect of deflation of the neck bladder in a subject pressure breathing at a positive pressure of 60 mmHg upon the arterial blood pressure

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FALL OF MEAN ARTERIAL
PRESSURE (.mm Hg)
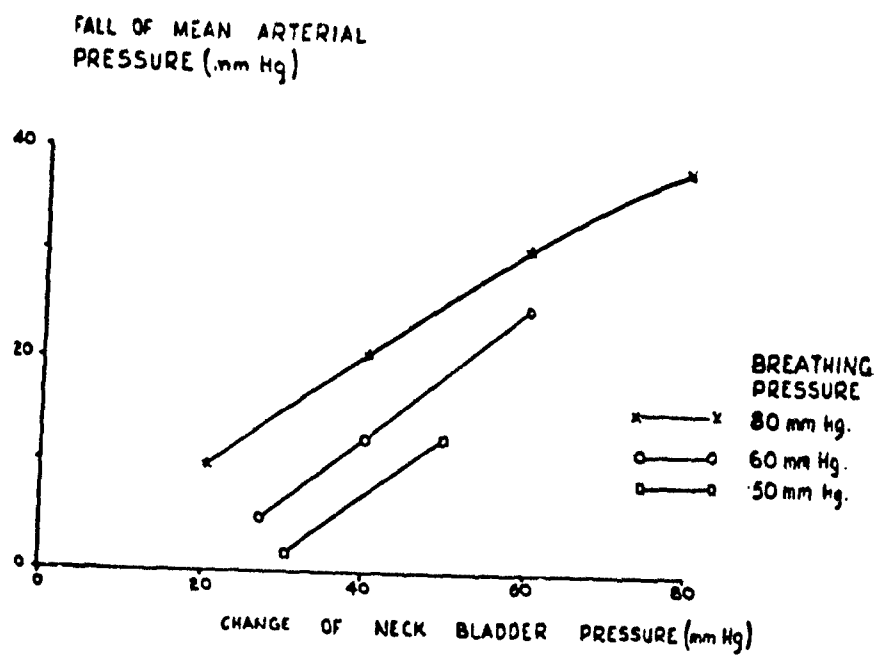
FIG. 3-17 The effects of various reductions of neck bladder pressure upon the mean
arterial blood pressure, whilst pressure breathing at positive breathing pressures of
50, 60 and 80 mmHg

cardia. The first one or two beats following the reduction of the pressure in the neck bladder showed a lengthening of the P–R interval of the electro-cardiogram. In two out of the six subjects pressurization of the neck bladder during pressure breathing produced a transient acceleration of the heart rate. In general, however, the cardiac effects of a given increase in neck bladder pressure were less conspicuous than an equal decrease of bladder pressure.

A reduction in the pressure in the neck bladder was followed by a fall of the arterial blood pressure (Fig. 3–16). The arterial pressure fell progressively for five to ten seconds, to a new steady value. All the subjects investigated showed this response although there was some variation in the magnitude of the reduction of arterial blood pressure caused by a given fall of neck bladder pressure. In a given subject pressure breathing at a constant value, the magnitude of the change of arterial blood pressure varied directly with the amount by which the pressure in the neck bladder was reduced (Fig. 3–17). The intra-venous administration of atropine (2 mg) ten minutes before an exposure to pressure breathing abolished the effects of a reduction of the neck cuff pressure upon the heart rate whilst the response of the arterial blood pressure to this stimulus remained unchanged.

The initiation of pressure breathing produced a gross disturbance of limb volume which, however, subsided after twenty seconds. Records of forearm blood flow were judged to be technically satisfactory when the increase of limb volume following inflation of the collecting cuff was linear with respect to time for the five second period during which the cuff was inflated. Such satisfactory results were obtained twenty seconds after the beginning of pressure breathing. The rate of blood flow through the forearm was calcu-lated from the slope of the volume curve whilst the collecting cuff was inflated, the calibrations of the gauge encircling the limb and the resting circumference of the limb being as described by Whitney (286). The results of a typical experiment are presented in Fig. 3–18. It may be seen that the blood flow following the initiation of pressure breathing at a positive breathing pressure of 60 mmHg was about one third of the value obtained in the resting state. Deflation of the neck cuff bladder which had been inflated to 60 mmHg at the beginning of the pressure breathing period caused a further reduction of the blood flow through the forearm. This further reduction of the forearm blood flow was maintained for as long as the neck cuff was deflated. Re-inflation of the cuff was associated with a rise of forearm blood flow. The results of all the experiments in which forearm blood flow were measured are given in Table 3–3. Each value is the mean of at least five consecutive measurements of blood flow for each of the three periods, viz. resting before the beginning of pressure breathing, during pressure breathing with the neck cuff inflated and during pressure breathing with the neck cuff deflated. The percentage reduction of forearm blood flow caused by deflation of the neck cuff has been plotted against the corresponding reduction in neck bladder pressure for all the experiments in Fig. 3–19. It may be seen that although there is considerable scatter of the individual points there is a direct linear relationship between these two variables.
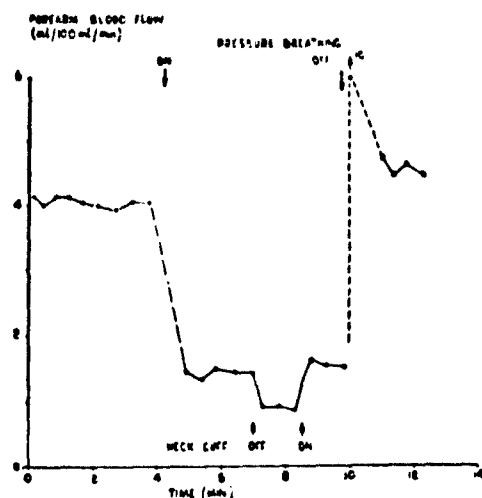
Fig. 3-18 The effect of pressure breathing upon the forearm blood flow. The neck cuff was deflated for a period of 1½ minutes during pressure breathing at a positive breathing pressure of 60 mmHg
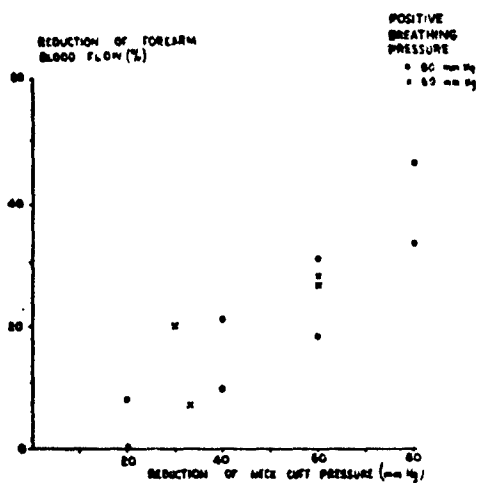
Fig. 3-19 The relationship between the reduction of neck cuff pressure whilst pressure breathing at 60 and 80 mmHg and the forearm blood flow. The forearm blood flow following the reduction of neck cuff pressure has been expressed as a percentage of the blood flow measured when the cuff was inflated to a pressure equal to breathing pressure

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TABLE 3-3

THE EFFECT OF PRESSURE BREATHING AND THE APPLICATION OF VARIOUS DEGREES OF COUNTERPRESSURE TO THE NECK UPON FOREARM BLOOD FLOW

Subject	Positive Breathing Pressure (mmHg)	Reduction of Neck Cuff Pressure (mmHg)	Forearm blood flow (ml/100 ml/min.)		
			Rest	Pressure Breathing with neck cuff inflated	Pressure Breathing with neck cuff pressure reduced
A	60	30	4.5	1.5	1.2
		60	4.C	1.4	1.0
A	80	20	4.3	1.2	1.1
		40	4.1	1.4	1.1
		60	3.9	1.3	0.9
		80	4.5	1.3	0.7
B	60	30	4.6	1.6	1.5
		60	4.4	1.4	1.1
B	80	20	4.2	1.1	1.1
		40	4.1	1.2	1.1
		60	4.3	1.3	1.1
		80	4.'	1.2	0.9

DISCUSSION

These investigations have shown the regions of the head and neck in which disturbances are induced by pressure breathing when an oronasal mask is used to deliver the increase in gas pressure to the respiratory tract. In most regions two mechanisms are responsible for these disturbances: the rise of pressure within the upper respiratory tract is responsible for some of the changes in that it produces a significant pressure gradient between the air-containing cavities and the surface of the skin; the second mechanism concerned is vascular. When trunk counterpressure is employed the arterial pressure is increased by 80–120% of the pressure applied to the respiratory tract whilst the venous pressure is increased by an amount which virtually equals the applied pressure. Thus the transmural pressure of all the vessels of the unsupported regions of the head and neck is increased during pressure breathing by an amount which is virtually equal to the breathing pressure.

Eye – The direct effect of the rise of the pressure within the respiratory tract upon the eye is the opening of the nasolacrimal duct. Each duct normally carries the lacrimal secretion from one conjunctival sac. The secretion flows to the inner canthus of the eye and then passes through the lacrimal cannaliculi which are situated at the inner end of the upper and lower lids, into the nasolacrimal sac and thence into the middle nasal passage. The edges of the lids containing the lacrimal cannaliculi are normally opposed to the surface of the scleral conjunctiva, and the walls of the nasolacrimal duct lie in contact

with one another. In the majority of subjects studied (65%) the nasolacrimal ducts remained closed even when gas at a positive pressure of 70 mmHg was applied to the respiratory tract. In a few subjects, however, gas flowed from the lacrimal cannaliculi at a pressure of 50 mmHg and the proportion of subjects affected was increased by a rise of breathing pressure. In these instances the increase of the pressure of the gas within the nose must have opened the nasolacrimal duct. Gas then passed up through the lacrimal cannaliculi lifting their conjunctival openings off the surface of the scleral conjunctiva.

Since the stream of gas passed up the nasolacrimal duct the tear secretions did not drain down the duct in the normal manner and the secretion rapidly flowed over the edge of the lower lid. The flow of gas through the lacrimal cannaliculi on to the surface of the scleral conjunctiva was associated in most cases with spasm of the eyelids. This blepharospasm was probably a reflex initiated by the irritation of the conjunctiva produced by the incident stream of gas from the cannaliculi. In only two of the subjects studied was this spasm severe enough to interfere with vision and at a positive breathing pressure of 60 mmHg impairment of vision arose in only one subject. This disturbance may obviously limit the pressure that can be applied by means of an oronasal mask. The results of the exposure of twenty subjects suggest, however, that the incidence of serious blepharospasm and consequent interference with vision is very low with positive breathing pressures of up to 60 mmHg. Subsequent experience in the training of one hundred aircrew in pressure breathing with an oronasal mask at pressures of up to 65 mmHg has confirmed these conclusions. No incident of impairment of vision was encountered in this extended training programme, although in several subjects there was a considerable degree of blepharospasm at the highest breathing pressure.

Since the conjunctival vessels lie directly beneath a thin epithelial layer, an increase of the pressure within them will probably not affect the pressure in the tissues surrounding them. Thus in pressure breathing the transmural pressures of these vessels are probably increased by an amount which corresponds to the rise of intravascular pressure. The conjunctival suffusion seen in some of the subjects at breathing pressures of greater than 50 mmHg was due to the vascular dilatation produced by this increase of vascular transmural pressure. The present series of experiments suggests that in the vast majority of individuals the conjunctival vessels can withstand an increase of transmural pressure of up to 70 mmHg without rupture since only one instance of subconjunctival haemorrhage occurred. Another circumstance in which the vascular pressure in the vessels of the eyes is raised is the application of longitudinal accelerations to the body acting from foot to head ("negative" G). Both in the goat and man, exposure to longitudinal acceleration acting from foot to head of 2.5×981 cm sec.$^{-2}$ for 15 sec. invariably produced conjunctival haemorrhages (120). During such an exposure the effective weight of the column of blood between the eye veins and the thorax is markedly increased so that at an acceleration of 2.5×981 cm sec.$^{-2}$ the venous pressure at eye level was 70 to 80 mmHg in the human experiment conducted by Gamble, Shaw, Henry and Gauer 1950 (120). It is apparent, therefore, that if the increase in vascular pressure at eye level produced by pressure breathing exceeds 70 mmHg conjunctival capillaries will be ruptured.

Rupture of an intraocular vessel may have much more serious consequences than the small subconjunctival haemorrhages which follow rupture of one of the vessels lying on the external surface of the eye. An intraocular vascular rupture may result in bleeding into either the anterior or posterior chamber or in direct damage to the retina, and any of these events can cause permanent loss of vision. Intraocular haemorrhage due to increased venous pressure in the head occurs in several circumstances, thus Henry (1950) (145) found that exposure to high longitudinal accelerative forces applied from foot to head of the order of 10×981 cm sec.$^{-2}$ produced occasional haemorrhages in the anterior chamber of the eye in goats. At this level of acceleration the venous pressure at eye level must have been of the order of 300 mmHg. Accidental exposure of aircrew during flight to negative accelerations of the order of 3×981 cm sec.$^{-2}$ has resulted in intraocular haemorrhages (Howard, personal communication). Similar haemorrhages have also been produced by high decelerative forces being applied to a man-seat system in which the man was secured by a seat harness passing across the abdomen (271). The mechanism of this injury was probably that the man was suddenly flung into the restraining harness and this caused a rapid and marked rise of intra-abdominal pressure, which was transmitted through the venous system to the intraocular vessels (158). In all these instances, however, intraocular haemorrhage was associated with venous pressures in excess of 100 mmHg at eye level and in most instances with a very rapid rise of venous pressure.

The mechanics underlying the behaviour of the intraocular vessels when a rise of venous pressure occurs differ considerably from those involved in the case of the conjunctival vessels. The intraocular vessels lie in a fluid-filled thick-walled sphere and are supported to a certain extent by the intraocular fluids. The magnitude of the increase of the transmural pressure of the intraocular vessels when the pressure within them is raised will be determined by the relative distensibilities of the ocular globe and the intraocular vascular bed. Thus at one extreme, if the sclera and cornea were indistensible a rise of intravascular pressure would be transmitted throughout the extravascular fluids of the eye without an increase of the transmural pressure and hence with no significant distension of the intraocular vessels. If the distensibility of the ocular globe were significant but considerably less than that of the vascular bed within the eye a small fraction of the total increase of intravascular pressure would appear as an increase of vascular transmural pressure and a slight distension of the capacity vessels of the intraocular bed would occur. In both these situations the likelihood of vascular damage occurring as a result of an increase of venous pressure would be very remote. If, however, the distensibility of the cornea and sclera exceeds that of the vascular bed within the eye a large proportion of any increase of intravascular pressure would be borne by the vessels themselves and the situation would approach that which exists in the conjunctiva. The intraocular haemorrhages produced by high levels of foot-to-head acceleration and by sudden blows to the abdomen could have arisen because either a very large increase of vascular pressure occurred and the support afforded by the ocular globe became inadequate or there was a temporal lag in the rise of extravascular pressure within the eye.

The rise of vascular pressure produced by pressure breathing at positive

breathing pressures of up to 70 mmHg is such that rupture of intraocular vessels due to inadequate support by the sclera and cornea would not be expected. No instance of intraocular haemorrhage has been reported as a result of pressure breathing with an oronasal mask and no evidence of retinal vascular damage was seen in the present experiments. Further, the direct observations of the large retinal vessels gave no evidence of vascular distension during pressure breathing. There was indeed a slight reduction of the diameter of the retinal veins during this manoeuvre. These ophthalmoscopic findings have been confirmed by Green (1961) (129) who took serial photographs of the retina before and approximately five and forty seconds after the onset of pressure breathing at 60 mmHg. He found that whilst there was no significant change of the retinal venous diameter in the first ten seconds of pressure breathing, after thirty seconds there was a significant reduction in the diameter of these vessels.

These results suggest that the ocular globe is considerably less distensible than the vasculature of the eye and that no significant temporal lag occurs between the ocular vascular pressure and the pressure within the extravascular fluids of the eye at the onset of pressure breathing, at least at positive breathing pressures of up to 70 mmHg. The mechanism of the reduction of the diameter of the retinal veins observed during pressure breathing is uncertain. Pressure breathing generally induces hyperventilation and hypocapnia causes constriction of the retinal veins. The onset of the constriction during pressure breathing is relatively rapid and the degree of hypocapnia produced by thirty seconds of pressure breathing is small. It is possible on the other hand that the retinal veins participate in the general peripheral venoconstriction which is induced by pressure breathing (Chapter 6). The measurements of visual acuity confirm that pressure breathing at pressures of up to 70 mmHg causes no disturbance of the peripheral processes which underlie vision.

Neck Distension – The distension of the neck is one of the most striking effects of the delivery of gas at positive pressures above 30 mmHg by means of an oronasal mask. The radiographic studies demonstrate that an increase in the volume of the upper respiratory tract is the principal cause of this distension although direct observations suggest that vascular congestion also plays a part. The two methods used to study the distension of the air-containing cavities of the head and neck, radiography and measurement of the respiratory dead space, are complementary. The former gives a qualitative indication of the parts involved in this distension whilst the latter gives a measure of the volume increase. Care was taken in the radiographic study to avoid as far as possible the distortion of the apparent dimensions of the air-containing cavities by employing a long tube-to-subject distance and calibrating the experimental radiographs under exactly the same conditions as were used with the human subjects.

The value of the dead space volume of a given individual as measured by monitoring the egress of nitrogen from the lungs during a prolonged expiration following an inspiration of oxygen is affected by a number of variables. The most important of these factors are the volume of oxygen inspired immediately before the measurement, the duration of the pause between the end of inspiration and the beginning of expiration and the pattern of this expiration

(111) (36). Thus in any experiment designed to measure a change of dead space volume produced by a given situation, the value of these variables must be held constant. In the present study the subjects had had previous experience of respiratory experiments and were capable of controlling their respiratory patterns. The volume of oxygen inspired and the duration of this inspiration were controlled by the subject watching the pen of the spirometer from which this gas was inspired as it moved over the recording drum.

After a little practice each subject was able to inspire 1 litre of oxygen over a period of three seconds. The subject was also able to control the duration of the inspiratory/expiratory pause by watching the movement of the recording drum of the spirometer and commencing expiration at a fixed interval (two seconds) after the end of inspiration. Although the subject was given no visual indication of his expiratory flow pattern the instruction to breathe out at a steady rate resulted in a reproducible expiratory flow pattern, particularly when the subject was at rest. The relatively small standard deviation obtained in the series of ten consecutive determinations of the dead space volume in a resting subject demonstrates the adequacy of control of these variables at least in the resting state. The reliability of this method of measuring added external dead space as demonstrated by the experiments in which smooth bore hose of known volume were added to the breathing circuit was satisfactory in view of the magnitude of the change found during pressure breathing.

The subjects found it very difficult to maintain a fixed expiratory flow pattern during pressure breathing and the influence of this factor is shown in the increased variability found in the measurement of dead space volume during pressure breathing. This difficulty together with the impossibility of obtaining a perfect mask seal at positive breathing pressures above 45 mmHg led to the adoption of the neck box as providing a second method of studying the effects of an increased pressure difference across the walls of the upper respiratory tract. The reduction of pressure around the neck did not interfere with normal respiratory mechanics so that the subjects were able to control their breathing satisfactorily. The proportion of the upper respiratory tract exposed to the distending pressure was slightly greater during pressure breathing than when suction was applied by means of the neck box. In the neck box experiments the cheeks were not exposed to the increased pressure differential and the intrathoracic airways were not distended by the increase of functional residual capacity normally induced by pressure breathing.

The increase in volume of the oral cavity produced by distension of the cheeks in pressure breathing has not been measured. The actual outward movement of the cheeks induced by pressure breathing is limited by the edge of the oronasal mask. The increase of the coronal diameter of the oral cavity cannot exceed 2 cm at a positive pressure of 60 mmHg. It may be estimated that the maximum increase of the volume of the oral cavity at this pressure will not exceed 15 ml. The increase of the anatomical dead space associated with a 0.5 l increase of the functional residual capacity, which is the order of the increase produced by pressure breathing, is about 15 ml (36). Thus the maximum difference at a distending pressure of 60 mmHg between the increment of the dead space volume given by the neck box method and by pressure breathing will not exceed 30 ml which amounts to only 12% of the total volume increase. The results of the neck box experiments may be applied

therefore to the situation where distension is produced by pressure breathing without introducing a serious error.

The pressure distension curve of the air spaces of the head and neck is sigmoid in shape and thus it is possible to distinguish three phases in the distension process. When the positive breathing pressure is less than 10 mmHg no significant distension occurs. Between a pressure of 10 mmHg and one of 50 mmHg the distensibility of the airways is very high, whilst above 50 mmHg the passages are virtually indistensible (within the range of pressures studied). It is possible to account for this curve by the following mechanism: at rest most of the passages are slit-like in cross section with the superio-inferior (oral cavity) or antero-posterior (oropharynx, hypopharynx and cervical oesophagus) walls virtually touching. When the pressure difference between the gas in the lumen of this tube and the surface of the skin exceeds about 10 mmHg the cross-sectional shape changes from a slit to an oval and finally to a virtual circle.

During this phase a very large increase of cross-sectional area and hence of the volume of these passages occurs with very little increase of circumference and therefore little increase of wall area. The increase of pressure associated with this phase of rapid increase of volume overcomes the resistance to deformation of the tissues lying between the lumen of this tube and the surface of the head and neck. Thus in this phase the floor of the mouth is depressed, the oropharynx and hypopharynx becoming widely dilated so that their lateral recesses, which are normally closed, open out to give a very wide tube. The cervical oesophagus is opened out into a tube which becomes virtually circular in cross section. A similar change occurs in the shape of the lower pharynx and oesophagus during deglutition when a large bolus is swallowed, except that only one part of the tube is opened widely at any instant during swallowing.

The principal causes of tissue resistance at this phase of distension are the tone of the striated muscles which form the floor of the mouth and which surround the pharynx and upper oesophagus, and the elastic fibrous tissue which envelops these regions. When the distending pressure is of the order of 50 mmHg the inelastic fibrous tissue which surrounds the muscle of the floor of the mouth and neck becomes tense and tends to prevent any further increase of the volume of the pharynx and oesophagus. Thus during pressure breathing at pressures greater than 50 mmHg the tissues of the walls of the mouth, the pharynx and the upper oesophagus are widely stretched.

The cervical oesophagus is distended throughout its length during pressure breathing at positive pressures greater than 10 mmHg. The portion of the oesophagus which lies within the thorax, however, is not involved in this distension. The pressure within the thorax is raised during pressure breathing by an amount which very nearly equals the positive breathing pressure, so that the pressure difference across the wall of the thoracic oesophagus is not increased significantly by pressure breathing. Thus in contrast to the pressure conditions existing in the cervical oesophagus pressure breathing does not produce any tendency to distension of the intrathoracic oesophagus.

Since the cervical oesophagus is not ventilated directly by the tidal volume, a considerable fraction of the air contained within the distended tube will not be included in the increase in dead space volume as measured by the Fowler technique. A certain degree of mixing will occur between the inspired

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air and the air held in the upper part of the oseophagus just below the opening of the larynx, especially if any turbulence occurs in the flow of gas into and out of the larynx. The volume of air contained within the distended oesophagus may be calculated approximately from the dimension of the shadow of this region in the lateral and antero-posterior radiographs taken during pressure breathing. This volume amounted to 60 ml at a positive breathing pressure of 60 mmHg.

The diameter of that part of the trachea which lies within the neck is slightly increased during pressure breathing. The increase of the antero-posterior diameter is due to bulging outwards of the posterior wall of the trachea where the cartilaginous rings are incomplete. The intrathoracic trachea does not undergo any significant increase in size since there is no increase in the pressure difference between the tracheal lumen and the pleural pressure. The larynx and trachea are moved forward relative to the cervical spine by distension of the pharynx and oesophagus. The opening into the larynx is widened by pressure breathing, particularly at positive breathing pressures in excess of 30 mmHg.

The measurements carried out with the neck box suggest that pressure breathing at 60 mmHg increases the dead space of the upper respiratory passages to more than double the resting value. Nunn, Campbell and Peckett 1959 (227) have also carried out experiments which illustrate the large variability of the dead space volume. They found that the respiratory dead space increased from a value of 70 ml when the neck was acutely flexed by as much as a further 70 ml when the head was fully extended. The increase of dead space in pressure breathing will lead to a diminution of the alveolar ventilation at a given level of pulmonary ventilation. Thus if no change of pulmonary ventilation occurred during the pressure breathing a breathing pressure of 60 mmHg would reduce the alveolar ventilation by 2 litres per minute at a respiratory frequency of 10 per minute. This reduction is a very considerable fraction of the alveolar ventilation of the resting subject. Thus if no hypernea occurred during pressure breathing with an oronasal mask a significant degree of hypercapnia would arise. In fact pressure breathing normally induces such a degree of hypernea that the arterial carbon dioxide tension actually falls. In these circumstances the increase of respiratory dead space due to the distension of the upper respiratory passages reduces the degree of hypocapnia induced by the increase of pulmonary ventilation.

At a certain level, which varies from one subject to another, and from time to time in the same subject, pressure breathing with an oronasal mask induces discomfort in the neck and the floor of the mouth. At the highest breathing pressures studied some of the subjects experienced frank pain in these regions. The existence of discomfort also depends upon the duration of the exposure. Thus when the duration of exposure is relatively short (less than four minutes) the incidence of discomfort is insignificant at positive breathing pressures of less than 50 mmHg. Above this pressure, however, discomfort and pain are common even if the exposure is short. The discomfort is of the dull, illocalized, nauseating type usually associated with the stimulation of simple sensory endings (282). The lowest breathing pressure at which this subjective disturbance arises is very close to the pressure at which the upper respiratory airways are fully distended and presumably the fascial layers become stretched.

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It is probable, therefore, that the discomfort and pain produced by pressure breathing with an oronasal mask at positive pressures greater than 50 mmHg arise from the stimulation of receptors lying in the fibrous tissue of the fascial layers of the neck. This discomfort and pain is the most important single factor limiting the pressure at which gas may be delivered to the respiratory tract by means of an oronasal mask. Apart from the conscious appreciation of this form of sensory stimulation afferent impulses of this type can have important cardiovascular effects. As will be discussed later, afferent impulses associated with the sensation of pain of the deep illocalized type may precipitate vasovagal syncope during pressure breathing. When the duration of pressure breathing with an oronasal mask is extended beyond five minutes discomfort may occur in the neck and floor of the mouth at breathing pressures of less than 50 mmHg. Pressure breathing at a positive pressure of 20 mmHg has been performed for at least one hour without any discomfort (161). Pressure breathing at a positive pressure of 30 mmHg gave rise to discomfort when the duration was extended beyond thirty minutes (161).

The distension of the mouth and pharynx induced by pressure breathing with an oronasal mask at positive pressures of greater than 30 mmHg interferes with the processes underlying speech. Pressure breathing of itself does not interfere with speech since normal intelligible speech can be produced when a pressure headpiece is used in conjunction with trunk counterpressure. Distension of the mouth and pharynx interferes with the process by which the vibrating air column produced by the lungs and larynx are transformed into speech. There is distortion of the sounds which depend upon the fine movements of the walls of the pharynx, the soft palate and the tongue. The movements of the lips are also restricted somewhat during high pressure breathing with a mask as the cheeks are forced out against the edge of the mask.

Disturbances in the Ear – The absence of any movement of the tympanic membrane in most individuals at the beginning of pressure breathing suggests that there is no significant increase of the pressure within the middle ear cavity during this manoeuvre. This conclusion is supported by the results of the measurements of auditory acuity made before and during pressure breathing at ground level. Van Dishoek 1941 (278) investigated the effects of increasing or decreasing the pressure within the exernal auditory canal upon hearing. He showed that the creation of a pressure difference across the tympanic membrane produced a loss of hearing and that the magnitude of this loss grew as the pressure difference was increased. The hearing loss was greatest at the lower frequencies. More recently Jones 1958 (164) demonstrated that a meatal pressure of 10 cm of water either greater than or less than atmospheric pressure produced a hearing loss of 4 db at a frequency of 50 c/s. Thus the measurement of auditory acuity is a sensitive method of deducing the presence of a pressure difference across the tympanic membrane. In the vast majority of subjects, since there was no change of acuity during pressure breathing, the pharyngo-tympanic tube did not transmit the increase of naso-pharyngeal pressure to the gas within the middle ear cavity. Swallowing, however, was followed by a rise of this pressure. The absence of any significant rise of middle ear pressure during pressure breathing at ground level was due to the rapid rise of naso-pharyngeal pressure to a level at which

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the pharyngeal ostium of the pharyngo-tympanic tube was firmly closed by the pressure within the pharynx itself.

Although no direct observations of the behaviour of the tympanic membrane were made following the induction of pressure breathing by rapid decompression to a simulated altitude in excess of 40000 ft, the measurements of auditory acuity performed at 50000 ft suggest that this situation differs from that which exists when pressure breathing is induced at ground level. Thus on every occasion there was a hearing loss of between 15 and 30 db during pressure breathing at 60 mmHg at 50000 ft. This is the magnitude of hearing loss which would be expected if the pressure differential across the tympanic membrane equalled the positive breathing pressure. In these measurements at simulated high altitude the subject underwent a rapid decompression from 25000 ft. Before such a rapid decompression the pressure of the gas in the middle ear cavity is approximately equal to the pressure within the decompression chamber (282 mmHg absolute).

As the pressure in the chamber falls on decompression the absolute pressure within the naso-pharynx falls from that of the environment at 25000 ft to the value delivered by the oxygen regulator (140–150 mmHg absolute). Thus the gas expanding within the middle ear cavity flows along the pharyngo-tympanic tube into the naso-pharynx until the absolute pressure within the middle ear approximately equals that within the naso-pharynx, i.e. 140–150 mmHg. A fraction of the increase of the volume of the gas in the middle ear cavity is taken up by the increase in the dimensions of this part produced by the bulging of the tympanic membrane into the external auditory meatus. Thus since the pressure within the external meatus follows that of the environment the tympanic membrane is subjected to the full pressure differential delivered by the oxygen regulator. The tympanic membrane appears to be sufficiently strong to withstand a pressure in the middle ear exceeding that in the meatus by up to at least 100 mmHg for short periods without any permanent damage. Some 400 subjects have been exposed to breathing pressures of between 60 mmHg and 100 mmHg at simulated altitudes above 40000 ft with only one incidence of ear damage which was probably vascular in origin.

A very considerable proportion of the blood vessels of the tympanic membrane are distributed in the thin layer of skin which forms its external surface. Since the pressures of the gas within the middle ear and that in the external auditory meatus are not normally raised by pressure breathing at ground level, the difference of pressure between the blood within the vessels of the membrane and the surface of the membrane is increased by an amount equal to the positive breathing pressure. It is not known how the extra-vascular pressure within the tympanic membrane behaves with a rise of intravascular pressure. In the superficial tissues of the limbs, however, there is no significant rise of tissue pressure during pressure breathing (91). It is probable, therefore, that the greater fraction of the increase of the pressure difference between the blood in the vessels of the membrane and the gas within the middle ear and external meatus occurs across the walls of the vessels. A rise of pressure in the middle ear cavity during pressure breathing will have an effect upon the extravascular pressure within the tympanic membrane. A gradient of pressure will be created between the inner and outer surfaces of

the membrane. It would appear likely that most of this fall of tissue pressure will occur within the collagen and elastic fibre layers of the membrane. Thus it is probable that the tissue pressure in the epithelial lining of the outer surface will not be increased significantly by a rise of pressure within the middle ear cavity.

The absence of any signs of vascular damage in the ear after a short exposure to positive breathing pressures of up to 100 mmHg suggests that the vessels of the tympanic membrane and of the epithelium lining the auditory canal are capable of withstanding an increase of transmural pressure of up to 100 mmHg. The appearance, however, of injection and petechial haemorrhages in the lining of the canal and on the surface of the membrane after prolonged exposure to pressure breathing at positive pressures of 80 and 100 mmHg, demonstrates that the capillary vessels of this region cannot withstand such a rise of transmural pressure over a long period. The similarity of the vascular changes seen in the wall of the deeper part of the auditory canal to those seen in the tympanic membrane itself demonstrate that these changes are vascular in origin and that they cannot be due to a rise of pressure in the middle ear. On one occasion in the experimental series a frank haemorrhage occurred from the rupture of a blood filled bulla. Similar vascular damage is seen when a frogman suit is used in diving (163). The design of the hood of this type of suit is such that the pressure within the external auditory meatus does not increase to as great an extent as does the hydrostatic and hence vascular pressures during descent in water. Frank bullae are frequently seen on the surface of the tympanic membrane in these circumstances.

The appearance of fluid within the middle ear cleft in many of the subjects exposed to prolonged pressure breathing adds further weight to the concept that the pressure within this cavity is not usually raised. The fluid presumably appears because the increase of the pressure in the vessels of the lining of the cleft disturbs the normal equilibrium between capillary pressure, tissue pressure and the osmotic pressures of the blood and the tissue fluids (272). This disturbance leads to the passage of fluid at an excessive rate from the blood. A high rate of fluid formation cannot arise if the pressure in the middle ear cavity increases *pari passu* with the pressure in the respiratory tract and hence the circulation. Thus failure to increase the pressure within the external auditory canal when the pressure within the respiratory tract is raised by pressure breathing can lead to damage of the vessels of the tympanic membrane and the skin lining the canal.

When the duration of any exposure is limited to less than four minutes, and the positive breathing pressure does not exceed 100 mmHg the incidence of overt vascular lesions in the ear is very low. In a series of 400 subjects exposed to pressure breathing under these conditions the total number of overt lesions was four (personal observation). Although the application of adequate pressurization to the external auditory meatus is the obvious method of overcoming the vascular distension induced by pressure breathing, this procedure has a certain disadvantage. Since in the majority of subjects the increase of naso-pharyngeal pressure is not communicated to the middle ear cleft, pressurization of the auditory canal will subject the tympanic membrane to the same stresses as arise when the barometric pressure is increased and thus introduce the risk of otitic barotrauma (73). With

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adequate instruction and training, however, the incidence of ear damage due to this mechanism can be reduced to an insignificant level.

Carotid Baroreceptors – The rise of the transmural pressure of the vessels of the neck normally induced by pressure breathing is prevented by inflation of a bladder around the neck to the same pressure as that applied to the respiratory tract. Thus deflation of the neck cuff increases the vascular transmural pressures in this region. The present study showed that a sudden reduction of neck bladder pressure during pressure breathing produces a transient bradycardia and a maintained reduction of the arterial pressure. These cardiovascular effects constitute the characteristic response to stimulation of the carotid artery stretch receptors which was first described by Hering 1924 (151). It is probable, therefore, that the effects of removal of counterpressure to the neck during pressure breathing arise reflexly from stimulation of the stretch receptors in the walls of the carotid arteries.

The exact relationship between the decrease of pressure in the cuff around the neck and the change of the transmural pressure of the carotid arteries is uncertain. When a bladder restrained by an outer non-distensible layer is used to exert pressure on the skin, the pressure applied will only equal that of the gas within the bladder if there is no tension in the inner layer of the bladder. Whilst this situation existed over much of the neck covered by the bladder there was tension in the wall of the bladder where it was reflected off the skin at its upper and lower borders. Thus the regions of the neck covered by the borders of the bladder were not subjected to the total pressure of the gas in the bladder. The upper border of the bladder was fixed as high as possible in an attempt to ensure that full counterpressure was applied to the skin overlying the carotid sinus region. It has been shown (Ernsting, 1955) (91) that a rise of the pressure within the fore-arm vessels induced by pressure breathing caused no significant increase of the local tissue pressure so that the entire increase of the pressure difference between the tissues and the blood within the lumen of the vessel occurred at the vessel wall. The presence in the neck of the strong superficial and deep layers of cervical fascia together with the dense carotid fascial sheath may modify this relationship between the tissue pressure and the intravascular pressure. No direct measurements of the tissue pressure within the carotid sheath have been made during pressure breathing.

The magnitude of the cardiovascular changes produced by removal of counterpressure from the neck during pressure breathing suggests, however, that a considerable fraction, if not all, of the pressure difference between the lumen of the vessels and the skin of the neck occurs across the vessel walls. The electrocardiographic changes seen following a large reduction of neck cuff pressure showed that the afferent discharge evoked by stimulation of the carotid artery stretch receptors affected both the heart rate and the rate of conduction by the atrio-ventricular bundle. The lengthening of the conduction time of the atrio-ventricular bundle was, however, of much shorter duration than the effect upon the sinuatrial node. This pattern of changes is typical of those mediated by the vagal efferent fibres to the heart. In one experiment in which the cuff pressure was reduced by 80 mmHg atrioventricular dissociation occurred for several beats immediately following the application of the stimulus owing to severe depression of atrio-ventricular

conduction. The abolition of these effects by atropine demonstrates conclusively that they were mediated by vagal efferent fibres. The transient nature of the cardiac slowing produced by deflation of the neck bladder is the typical reflex response to maintained stimulation of the carotid artery baroreceptors.

Thus, Hering 1927 (152) demonstrated that in animals the bradycardia induced by carotid sinus stimulation was of sudden onset, that its maximum effect was attained within a few seconds and that it was poorly maintained. Winder 1937 (287) concluded that the rapid recovery of the heart rate following stimulation of the carotid baroreceptors was due to afferent impulses arising from the aortic baroreceptors which were stimulated by the fall of arterial pressure induced by the carotid baroreceptor discharge. There was a close relationship between the intensity of the stimulation, reduction of neck bladder pressure and the cardiac response, bradycardia, in each experiment. A similar quantitative relationship was found by Ernsting and Parry 1957 (98) who stimulated the carotid artery stretch receptors by applying various subatmospheric pressures to the surface of the neck. Bronk and Stella 1932 (49) demonstrated that the intensity of the afferent discharge in the carotid sinus nerve bears a direct relation to the height of the arterial blood pressure. Thus in the present experiment, deflation of the neck bladder would be associated with an increase in the intensity of the afferent activity from the carotid sinus receptors and this increase in activity would be related directly to the magnitude of the reduction of neck bladder pressure.

The reduction of systemic arterial pressure produced by removal of counterpressure to the neck was maintained after the initial phase of cardiac slowing when the heart rate had returned to the prestimulation level. It also remained following abolition of the cardiac slowing by the administration of atropine. Thus the continuing arterial pressure response was independent of the change of heart rate normally induced by this stimulation. As with the heart rate response, the linear relationship between the change in neck bladder pressure and the response of the mean arterial pressure was demonstrated in each of the subjects used in the investigation. A similar relationship was demonstrated by Ernsting and Parry 1957 (98) when the carotid stretch receptors were stimulated in resting subjects. After atropinization the systemic arterial pressure fell relatively slowly when counterpressure was removed from the neck. Hering 1927 (152) demonstrated that the vasomotor response to carotid sinus stimulation was slower to develop than the cardiac slowing.

In order to elucidate the cardiovascular mechanism by which neck bladder reduced the arterial pressure, an attempt was made to determine the effects of this manoeuvre upon the blood flow through the forearm. Pressure breathing of the magnitude used in the present investigation causes a considerable rise of venous pressure and this is associated with distension of the capacity vessels of the limbs. Thus, conventional venous occlusion plethysmography (21) could not be used to measure the blood flow through a limb segment since this technique requires that the capacity vessels should be capable of receiving blood during the period that the venous cuff is inflated without the pressure within them rising significantly. The distension of the capacity vessels in pressure breathing can be prevented or reduced, however, by

applying external counterpressure. In the present study counterpressure was applied to the upper limb by air by means of a box enclosing the limb, so that the circumferential gauge would function satisfactorily. A pressure equal to that which was applied to the respiratory tract was used within the box encircling the limb.

Since frequently the rise of peripheral venous pressure induced by pressure breathing is not quite as great as the pressure applied to the respiratory tract, the transmural pressures of the capacity vessels of the limb exposed to gas counterpressure during pressure breathing may have differed from those which existed at rest. However, this difference could not be great and probably amounted to only 2 to 5 mmHg at a positive breathing pressure of 60 mmHg. Further, once the initial vascular disturbances produced by the beginning of pressure breathing subsided the transmural pressures of the vessels of the limb within the box remained constant. Thus whilst considerable difficulties arise if this technique is used to measure the change of blood flow induced by pressure breathing as compared with the resting state, it will give a satisfactory measure of changes of blood flow arising during pressure breathing once the initial disturbances produced by this manoeuvre have subsided. Further evidence in support of this contention may be obtained from the shape of the record of forearm volume following inflation of the venous collection cuff.

During the five seconds that the collecting cuff was inflated the limb volume increased at a constant rate. There was no evidence, therefore, of any reduction of the arterial inflow during the collection period. Although it is probable that the measurements of forearm blood flow obtained during pressure breathing did not reflect the arterial flow into the forearm which would have occurred in the absence of counterpressure to the surface of the limb, they suggest that pressure breathing reduces the blood flow into this region. There is considerable evidence that such a reduction of blood flow is produced by pressure breathing at much lower pressures. Thus Fenn and Chadwick 1947 (102) found a reduction of finger blood flow during pressure breathing at 30 mmHg, whilst Blair, Glover and Kidd 1959 (37) demonstrated a reduction of forearm blood flow which varied from 15 to 60% of the resting value in subjects exposed to pressure breathing at 15 mmHg. Personal measurements of peripheral vascular resistances in the forearm using the technique developed by Hayter and Sharpey-Schafer 1958 (142) have shown that pressure breathing at 60 mmHg with trunk counterpressure produces a 200 to 300% increase of vascular resistance.

The removal of the counterpressure to the neck during pressure breathing caused a reduction of the blood flow through the forearm (Fig. 3–18). Such a reduction of flow could have been the result of either a fall of the effective driving pressure, i.e. the difference between mean arterial and venous pressures, or an active constriction of the resistance vessels in the forearm. When the neck cuff was deflated there was a fall of arterial pressure (Fig. 3–17) and since the venous pressure was unchanged there was a reduction of driving pressure. It is possible to calculate the approximate value of the driving pressure associated with a given pressure in the neck bladder and a given breathing pressure from the aterial pressure measurements (Fig. 3–17). Thus at a positive breathing pressure of 80 mmHg reduction of the neck cuff

pressure by 40, 60 and 80 mmHg reduced the driving pressure by approximately 20, 30 and 40% respectively. Thus the reduction of blood flow through the forearm produced by deflation of the neck bladder was directly proportional to the corresponding decrease of driving pressure.

It follows, therefore, that the fall of the blood flow was produced by the reduction of the arterial pressure and that there was no significant change of arteriolar resistance in the forearm when the carotid artery stretch receptors were stimulated by removal of counterpressure to the neck. A similar relationship between blood flow through the forearm, hand and calf and the systemic arterial pressure was found by Ernsting and Parry 1957 (98) when the carotid baroreceptors were stimulated in resting subjects by reducing the pressure around the neck. Roddie and Shepherd 1957 (248) studied the cardiovascular responses to a fall of pressure in the carotid sinus produced by compression of the common carotid arteries. They found that this procedure produced no significant change of the resistance offered by the vessels of the forearm, calf and hand. These results are in conflict with measurements of limb blood flow made in animals. Thus Heymans, Bouckaert and Dautrebande 1931 (155), Grimson and Shen 1939 (132) and Lindgren and Uvnäs 1954 (192) found that stimulation of the carotid sinus baroreceptors produced by a rise of the transmural pressure caused an active vasodilatation in the limbs. The conditions of these animal experiments differed considerably, however, from those of the human studies, both in the form of the preparation and the size and nature of the stimulus applied to the carotid baroreceptors.

The maintained reduction of systemic arterial pressure produced by stimulation of the carotid baroreceptors during pressure breathing is not due to an arteriolar dilatation in the limbs. No further analysis of the cardiovascular changes induced by the removal of counterpressure to the neck was made during pressure breathing. Ernsting and Parry 1957 (98), however, studied the effects of increasing the transmural pressure of the carotid arteries in resting subjects upon the cardiac output. They measured the cardiac output by the direct Fick method following right heart catheterization in two subjects at rest and whilst various subatmospheric pressures were applied to the neck. In their experiments a reduction of the pressure around the neck of 40 mmHg caused no significant change of the cardiac output. In view of this finding it was suggested that the fall in arterial blood pressure produced by stimulation of the carotid baroreceptors was caused by a reduction of vascular resistance and that the site of this arteriolar dilatation was not in the limbs. The probable site of the arteriolar dilatation is the splanchnic circulation. This conclusion is supported by the results of many investigations of the effects of a rise of carotid sinus pressure in animals. Heymans, Bouckaert and Dautrebande 1931 (155) demonstrated mesenteric vasoconstriction following a drop of carotid sinus perfusion pressure and emphasized the important role played by the mesenteric vessels in the reflex response to carotid baroreceptor stimulation. Similar active vasoconstriction in response to a fall of carotid sinus pressure has been demonstrated by Heymans 1929 (154) in the kidney. Although no detailed analysis of the cardiovascular changes underlying the reduction of systemic arterial pressure produced by carotid artery baroreceptor stimulation has been undertaken during pressure

breathing there would appear to be no reason why the same mechanism should not be operative under these conditions.

Thus the absence of counterpressure to the neck during pressure breathing modifies considerably the cardiovascular changes induced by this procedure. The use of an oronasal mask reflexly reduces the normal increase of systemic arterial pressure. The order of this reduction is 10 to 25 mmHg at a positive breathing pressure of 60 mmHg. This relative hypotension is probably the result of dilatation of the resistance vessels in the splanchnic circulation and it will lead to an altered distribution of the cardiac output. Thus the difference between the carotid artery and jugular venous pressures will be reduced by some 15 to 30% by the use of an oronasal mask in place of a pressure headpiece. Such a reduction of the cerebral arterio-venous pressure gradient might be expected to produce a significant fall of blood flow through the brain. It is likely, however, that this effect is of less importance than the cerebral vaso-constriction produced by hypocapnia (169) which arises frequently during pressure breathing. There is no evidence that these changes of regional blood flow produce any significant effect upon overall performance during short duration exposures to high pressure breathing.

A further possibility to be considered, however, is that the specific cardio-vascular changes induced by stimulation of carotid artery stretch receptors may lead to syncope during pressure breathing. In certain susceptible subjects mechanical stimulation of the carotid sinus region produces syncope with a marked bradycardia and hypotension (283). The primary mechanism underlying these effects of carotid sinus stimulation is reflex cardiac slowing. Frequently the heart ceases to beat for five to ten seconds and unconsciousness supervenes. This form of syncope always follows immediately upon the application of the stimulus and is not accompanied by the facial pallor, nausea and sweating which typify vasovagal syncope (187). All the incidents of syncope which have been observed during pressure breathing have been of the vasovagal type. It has been seen that the absence of counterpressure to the neck during high pressure breathing gives rise to severe discomfort in certain subjects in addition to stimulation of the carotid artery stretch receptors. Discomfort and pain alone are potent causes of vasovagal syncope during pressure breathing. It is probable, therefore, that the discomfort produced in the neck by the use of an oronasal mask is the cause of syncope during pressure breathing rather than the concomitant stimulation of the carotid baroreceptors.

CONCLUSIONS

The experiments described in this chapter have shown that whilst there are limitations to the use of an oronasal mask to deliver gas under pressure to the respiratory tract, this technique is highly effective in certain circumstances. Whilst various types of oronasal mask have been employed in aviation for continuous positive pressure breathing at positive pressures of up to 30 mmHg since 1943 (119) (Roxburgh, personal communication) the value of this method at greater breathing pressures had not been studied. The scope of the present investigation was limited in that the exposures to pressure breathing were of relatively short duration, a single exposure to a positive pressure in excess of 50 mmHg lasting only two minutes. When the duration of the exposure to a positive breathing pressure of 60 mmHg is extended

to six minutes (94) the disturbances which are produced are similar to those which were found in the present study. The intensity and incidence of discomfort around the neck rises, however, with the increase of the duration of the exposure. The present study has demonstrated that high pressure breathing with a mask induces disturbances in the eye, the ear and the neck. Two of these disturbances set the limit to the maximum pressure which normal subjects will tolerate with this method of pressure breathing. These are discomfort owing to stretch of the soft tissues of the neck and spasm of the eyelids. The influence of the pressure at which the gas is delivered upon the incidence of these effects is such that the practical limit to the use of this technique is a positive breathing pressure of 65 mmHg. It may be concluded from the experiments described above that, provided the duration of the pressure breathing period is limited to two minutes, this pressure will be accepted by normal subjects and that no significant damage will be produced in the head and neck.

This study has shown, however, that the use of an oronasal mask to deliver gas under pressure to the respiratory tract has disadvantages even when the pressure and the length of the exposure are within the limits given in the previous paragraph. Distension of the neck causes some discomfort at positive breathing pressures of greater than about 30 mmHg. The pressure of the edge of the mask against the face is also a source of discomfort. All the specific disturbances caused in the head and neck by pressure breathing with an oronasal mask may be prevented by the use of a suitably designed headpiece which ensures a pressure equal to that applied to the respiratory tract is applied to the surface of the head and neck. In many situations in aviation, however, where the maximum positive breathing pressure required in an emergency will not exceed 65 mmHg and the total duration of the exposure to pressure breathing will not exceed two minutes, the advantages of wearing an oronasal mask instead of a pressure headpiece during routine flight out-weigh the disadvantages of using this method for emergency protection against exposure to high altitude.

When either the positive breathing pressure or the length of the exposure to pressure breathing exceed the limits which are imposed by the use of an oronasal mask, some form of pressure headpiece must be used. In its simplest form a pressure headpiece consists of a spherical globe which encircles the head and which has a circular seal through which the head is inserted and which abuts against the skin at the root of the neck. There are, however, several practical difficulties in the construction of such a headpiece. Further, a headpiece of this type may prove bulky and may significantly reduce the efficiency of the user. Thus there is room for a further compromise. The results of the experiments described in this chapter suggest that, at a mini-mum, a pressure headpiece should apply counterpressure to the face, the floor of the mouth, the external auditory meati and the neck. Practical pressure headpieces by means of which counterpressure may be applied to these regions, with the exception of the external ear, have been developed and used in flight (R.A.F. partial pressure headpiece, Chapter 2). The absence of counterpressure to the auditory meati limits the use of this type of pressure helmet to a maximum positive breathing pressure of approximately 100 mmHg, with an exposure time not exceeding four minutes. When the

magnitude of the breathing pressure is reduced the duration of an exposure may be extended. Thus a partial pressure headpiece may be used to deliver a positive breathing pressure of 50 mmHg for at least thirty minutes without any significant disturbance arising in the head and neck.

There are, therefore, three practical methods by which gas may be delivered to the respiratory tract at a pressure above that of the immediate environment. When this positive breathing pressure does not exceed 65 mmHg and the duration of an exposure is limited to less than four minutes a pressure sealing oronasal mask will suffice. A partial pressure helmet by which counterpressure is applied to the face and neck is suitable for short duration exposures to positive breathing pressures of up to about 100 mmHg. When these time limits are exceeded a pressure headpiece which encloses the entire head and neck must be used.

THE MECHANICS OF RESPIRATION DURING PRESSURE BREATHING AND THE EFFECTS OF CHEST AND TRUNK COUNTERPRESSURE

INTRODUCTION

One of the most striking effects produced by the delivery of gas to the respiratory tract at a pressure greater than that of the environment is the increase in the volume of gas in the lungs. In a subject who is instructed to relax his respiratory muscles the lungs are fully distended when the pressure delivered to the mouth is of the order of 20 to 25 mmHg and if this pressure exceeds 80 to 100 mmHg, tearing of the lung parenchyma may occur (144). In a normal subject, however, the expiratory muscles are contracted throughout the respiratory cycle during positive pressure breathing at pressures of greater than about 10 mmHg. Experience has shown that pressure breathing can be performed continuously for ten to twenty minutes at a breathing pressure of 30 mmHg (161). If the pressure is raised above this level expiration becomes very difficult and extreme fatigue sets in very rapidly. In practice the maximum positive pressure at which this form of continuous pressure breathing can be used is 30 mmHg. The distension of the lungs and the difficulty of expiration associated with pressure breathing may be reduced or prevented by applying pressure to the outer surface of the trunk (30). In this study of the respiratory disturbances induced by breathing at pressures of up to 130 mmHg two forms of respiratory counterpressure were used. A standard R.A.F. garment, the pressure breathing waistcoat, which applies counterpressure only to the chest, was used in certain preliminary experiments. It rapidly became obvious, however, that more complete respiratory counterpressure was necessary at the higher breathing pressures. The pressure jerkin which provides counterpressure to the whole trunk was developed. It was found that this garment would allow pressure breathing at pressures of up to 130 mmHg without any gross subjective disturbance of breathing.

The disturbance of the mechanics of respiration induced by pressure breathing and the influence of various degrees of respiratory counterpressure upon these disturbances have been investigated in detail at ground level. The maximum positive breathing pressures used in these experiments were limited by the cardiovascular effects of raised intrapulmonary pressure. Thus when counterpressure was applied to the chest alone the maximum positive pressure which could be used was 80 mmHg. When a pressure jerkin was worn the effects of positive breathing pressures of up to 100 mmHg were studied. The effects of positive breathing pressures above 100 mmHg were not studied in detail because of the absence of a pressure headpiece which applied adequate counterpressure to the external auditory meati. The experiments performed in this part of the investigation were carried out using four subjects, each of whom had had considerable experience of pressure breathing, both with and

without respiratory counterpressure. The physical characteristics of the subjects are shown in Table 4-1. The mechanical behaviour of each subject's respiratory apparatus was determined in a preliminary investigation by measuring the relaxation pressure-volume curve of the lungs and thoracic cage. The effects of pressure breathing and of varying degrees of respiratory counterpressure were investigated by measuring the total lung volume and its sub-divisions, the respiratory flow, the intraoesophageal and intragastric pressures and by radiographic studies.

EXPERIMENTAL INVESTIGATIONS

Relaxation Pressure Volume Curve – The relaxation pressure volume curve of the lungs and thoracic cage was determined for each of the four subjects used in this investigation. The seated subject was connected by way of a mouthpiece and a wide bore tube to a closed circuit consisting of a recording spirometer, carbon dioxide absorber and a circulating pump filled with oxygen. A water manometer was attached to the mouthpiece. At approximately one minute intervals the subject either inhaled or exhaled to change his lung volume and the tap between the mouthpiece and the spirometer was then closed. The subject was instructed to relax his respiratory muscles. The reading of the water manometer was taken and the mouth tap was open so that the subject was reconnected to the spirometer circuit. Each subject repeated this manoeuvre at various volumes above and below his normal resting functional residual capacity. An average of 20 points were obtained in this manner for each subject. Whilst connected to the spirometer circuit the subject also performed several maximum inspirations and expirations. The volume of gas within the respiratory tract in excess of the subject's residual volume corresponding to the relaxation pressures recorded at the mouthpiece was obtained from the spirometer record.

Results – The values of the mouthpiece pressures obtained during relaxation were plotted against the corresponding lung volumes expressed as the volume in excess of the residual volume for each subject. A typical curve is presented in Fig. 4-1. The shape of the curve obtained from each subject was very similar, being slightly sigmoid. A composite relaxation pressure volume curve was calculated from the experimental results obtained with the four subjects. In order to facilitate this calculation, lung volumes for each subject were expressed as a proportion of the subject's resting vital capacity. The mean composite relaxation pressure volume curve is presented in Fig. 4-2.

Sub-Divisions of the Total Lung Volume – Measurements of the vital capacity, its sub-divisions and of the residual volume were made with the four subjects seated at rest and whilst pressure breathing. Pressure breathing was performed with no respiratory counterpressure or whilst using either a pressure waistcoat or a pressure jerkin. In all the experiments the subject wore the modified pressure helmet fitted with a mouthpiece. For the determination of the vital capacity and of the expiratory and inspiratory reserve volumes this mouthpiece was connected to a wide bore "T" piece. The subject, wearing the appropriate pressure garment, sat within the decompression chamber. A pair of hoses (3.1 cm I.D.) connected the "T" piece attached to the mouthpiece to a 7.5 litre recording spirometer placed outside the decompression chamber The closed circuit so formed also contained a gas circulating pump

TABLE 4-1

The physical characteristics of the subjects used in the study of the mechanics of respiration

Subject	Age (yr)	Height (cm)	Weight (kg)
A	30	179	76.3
B	29	177	67.5
C	22	175	71.5
D	23	168	66.1

TABLE 4-2

The mean values of vital capacity and expiratory reserve volume obtained in duplicate experiments with four subjects

Positive breathing pressure (mmHg)	Vital capacity (% resting value)[1]		Expiratory reserve volume (% resting vital capacity)[1]	
	mean	S.E.	mean	S.E.
Rest	100.0	± 1.1	30.7	± 3.5
Pressure breathing				
(a) No counterpressure				
10	102.1^{2}	± 0.9	50.5^{2}	± 3.5
20	104.3^{3}	± 1.2	70.9^{3}	± 4.1
30	106.4^{3}	± 1.6	80.8^{3}	± 3.6
35	106.7^{3}	± 1.7	85.5^{3}	± 2.5
(b) Chest counterpressure				
20	101.8	± 1.7	56.5^{3}	± 4.1
40	106.2^{3}	± 1.6	67.5^{3}	± 3.5
60	109.2^{3}	± 1.5	79.3^{3}	± 4.9
80	108.9^{3}	± 1.6	81.5^{3}	± 4.1
(c) Trunk counterpressure				
20	99.1	± 1.5	37.6^{2}	± 2.5
40	102.0	± 1.6	35.1^{2}	± 3.5
60	104.5^{3}	± 1.4	39.5^{3}	± 3.0
80	105.2^{3}	± 1.5	41.6^{3}	± 2.5

[1] All values expressed as a percentage of the mean resting vital capacity (5.26 litre B.T.P.S.).

Significance of difference from resting value.

[2] $0.001 < P < 0.01$

[3] $P < 0.001$
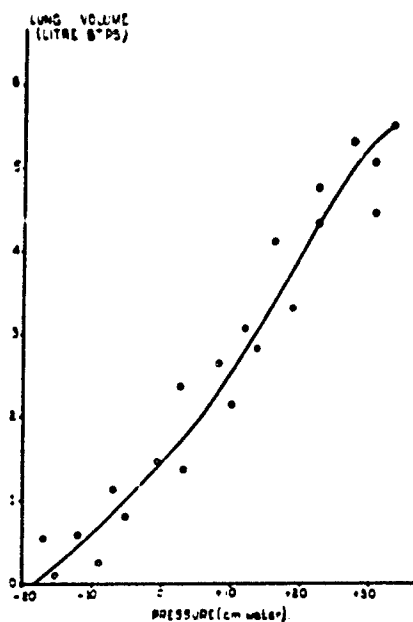
FIG. 4-1 The relaxation pressure volume curve of the lungs and thoracic cage of subject B. The mouth pressure generated with the respiratory muscles relaxed has been plotted against the corresponding lung volume
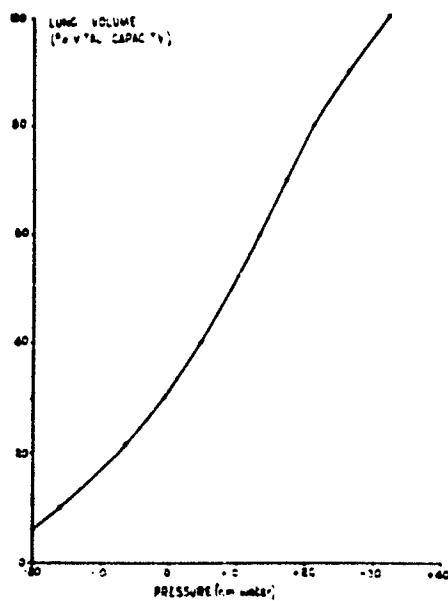
FIG. 4-2 The mean relaxation pressure-volume curve of the lungs and thoracic cage for the four subjects. The lung volumes have been expressed as a proportion of the corresponding resting vital capacity

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and a carbon dioxide absorber. Care was taken to ensure that no leakage occurred from the portion of the closed circuit situated within the decompression chamber when the pressure within the chamber was reduced to 150 mmHg less than atmospheric pressure. The face compartment of the helmet and the bladder of the pressure garment, when one was worn, were connected through wide bore tubing to the exterior of the decompression chamber. The closed circuit was filled with 100% oxygen and the helmet placed on the subject's head, after his nostrils had been occluded with a clip. Following an initial rest period of three to five minutes the subject was asked to perform three slow maximal inspirations and expirations, each separated by a period of quiet breathing. The pressure in the decompression chamber was then reduced by the desired amount in order to induce pressure breathing. Pressure breathing was continued for one to two minutes and then the subject was instructed to repeat the vital capacity manoeuvres. Two to three minutes after the cessation of pressure breathing the subject performed a maximal inspiration and expiration a further three times.

The residual volume of the lungs was measured by the nitrogen dilution technique developed by Rahn, Fenn and Otis 1949 (241). In this group of experiments a two-way wide bore tap was connected to the mouthpiece of the modified pressure helmet. A rubber bag with a capacity of 3 litres was attached to one limb of the tap. The other limb was connected by means of a "T" piece to a pair of wide bore pipes which passed to the exterior of the decompression chamber. A non-return valve was placed in each of these pipes to ensure the unidirectional flow of gas within them. The rubber bag was enclosed within a 10 litre aspirator bottle which was also connected to the exterior of the decompression chamber by a wide bore pipe. A side tapping was placed in the limb of the tap to which the rubber bag was attached. Before a measurement was made the rubber bag and the connecting limb of the two-way tap were flushed with oxygen and the bag was sucked empty. Then with the tap turned so that the bag was isolated 2 litres of oxygen were carefully measured into it. After the remainder of the circuit had been flushed with air the subject donned the appropriate pressure clothing and the helmet. At the desired time the subject was instructed to expire fully and hold his breath. The tap was then turned so that the mouthpiece was in direct communication with the bag. The subject breathed rapidly into and out of the bag taking three seconds to complete each respiratory cycle. He was instructed to completely empty the bag during inspiration and to breathe out as far as he could during expiration. At the end of the third expiration the subject again held his breath and a sample of the gas in the mouth of the bag was taken through the side tapping into a previously evacuated sampling tube. The tap was then returned to its original position and the subject allowed to breathe in. The concentrations of carbon dioxide and oxygen in each of the samples of gas were determined using the Haldane gas analysis apparatus. In preliminary experiments in which air was breathed throughout end-expiratory Haldane-Priestley samples of alveolar gas were obtained.

The values of the positive breathing pressures used were varied with the degree of respiratory counterpressure. When no counterpressure was employed the subject was exposed on two separate occasions to positive pressure breathing at 10, 20, 30 and 35 mmHg arranged in a random order. With respiratory

counterpressure, however, positive breathing pressures of 20, 40, 60 and 80 mmHg were used. Again each subject experienced each pressure twice and the order of the exposure was randomized.

RESULTS

The vital capacity and its subdivisions – A typical spirometer record obtained in an experiment in which the subject was exposed to pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure is presented in Fig. 4-3. When pressure breathing was induced there was a sudden reduction of the volume of gas within the spirometer. After several breaths, however, a new end-expiratory level was attained. This level was maintained for the remainder of the pressure breathing period. Cessation of pressure breathing was accompanied by an equally rapid increase of the spirometer volume. The magnitude of the change of spirometer volume varied with the breathing pressure, the degree of respiratory counterpressure and the subject. The vital capacity and expiratory reserve volume were determined from each experimental record for the control, pressure breathing and recovery periods. Each volume was corrected to the conditions present in the respiratory tract, i.e. saturated with water vapour at body temperature. The values of vital capacity and expiratory reserve volume obtained for each subject during each recovery period did not differ significantly from the corresponding control values. The control and recovery values were combined together, therefore, to give the resting values of these volumes.

In order to facilitate comparisons between one experimental condition and another the values of vital capacity and expiratory reserve volume obtained during the exposures to pressure breathing have been expressed as a proportion (percentage) of the resting vital capacity. The means of the percentage values of vital capacity and expiratory reserve volume obtained in the duplicate experiments on the four subjects are presented in Table 4-2 and Fig. 4-4 with their respective standard errors. There was a small increase of the vital capacity during pressure breathing, although the increase produced by a given pressure was reduced when counterpressure was applied to either the chest or the trunk. Pressure breathing without respiratory counterpressure caused a marked increase of the expiratory reserve volume, the value at a positive breathing pressure of 35 mmHg being two and a half times the control value. The counterpressure given to the chest by the pressure waist-coat reduced the increase of the expiratory reserve volume caused by pressure breathing. At a positive breathing pressure of 80 mmHg, however, the expiratory reserve volume was some two and a half times the control value. When counterpressure was applied to the whole trunk by the pressure jerkin there was only a small increase of the expiratory reserve volume.

The analysis of spirometer records obtained in these experiments was extended to the measurements of the tidal volume. The mean tidal volumes have been measured from each spirometer record over the last minute of the control period and the last minute of the pressure breathing period. The mean tidal volumes for the resting state and for each of the experimental conditions have been calculated from the eight results obtained from the four subjects (Table 4-3). Pressure breathing caused an increase of the tidal volume, although there was a considerable variation of response between the four

Fig. 4-3 A typical spirometric record obtained during the determination of the vital capacity and its sub-divisions during rest and pressure breathing at 30 mmHg without respiratory counterpressure

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LUNG VOLUME
(% RESTING VITAL CAPACITY)
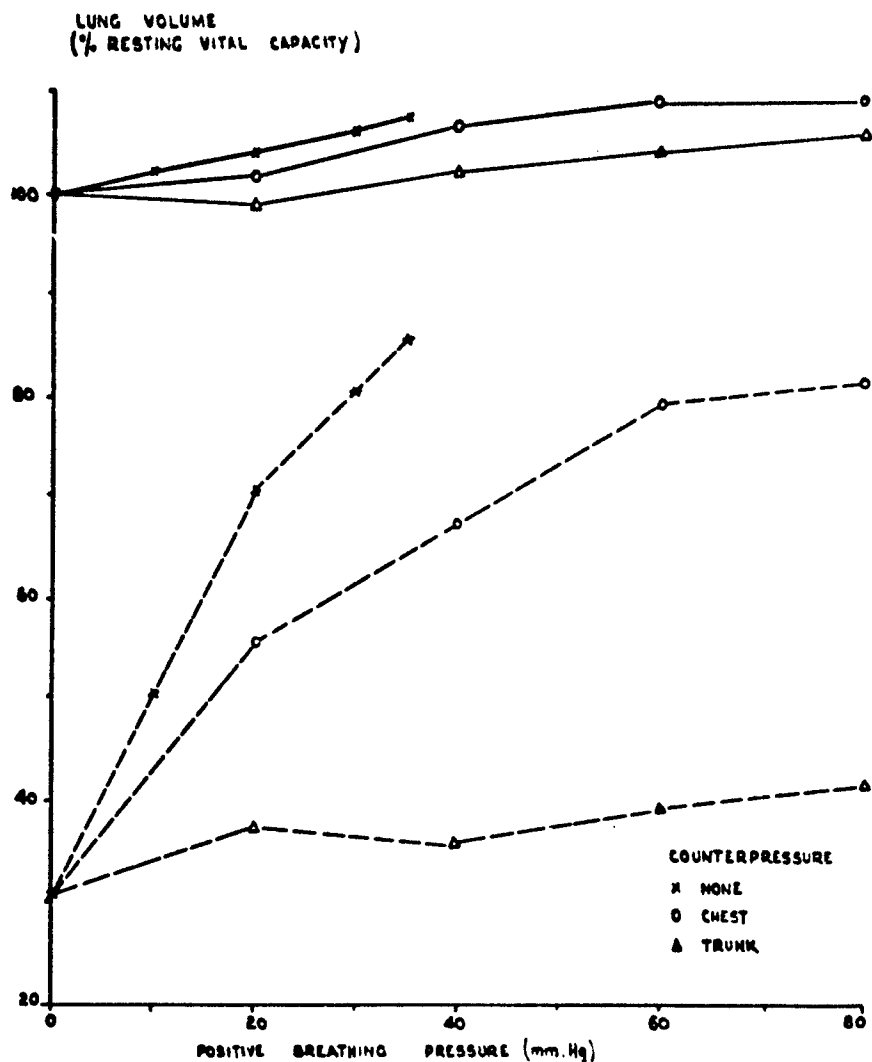
FIG. 4-4 The mean values for four subjects of the vital capacity (solid line) and expiratory reserve volume (interrupted line) during pressure breathing with no respiratory counterpressure (X), with chest counterpressure (O) and trunk counterpressure (△). The lung volumes have been expressed as a percentage of the resting vital capacity

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TABLE 4-3

THE MEAN VALUES OF THE TIDAL VOLUME
OBTAINED IN DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Positive breathing pressure (mmHg)	Tidal volume (litre B.T.P.S.)	
	Mean	S.E.
Rest	0.69	±0.04
Pressure breathing		
(a) No counterpressure		
10	0.68	±0.05
20	0.71	±0.07
30	0.90[2]	±0.06
35	0.94[2]	±0.09
(b) Chest counterpressure		
20	0.71	±0.06
40	0.78[1]	±0.05
60	0.84[2]	±0.07
80	0.95[2]	±0.09
(c) Trunk counterpressure		
20	0.67	±0.05
40	0.71	±0.04
60	0.75	±0.06
80	0.81	±0.05

Significance of difference from resting value
[1] 0.001 < P < 0.01
[2]   P < 0.001

TABLE 4-4

THE MEAN VALUES OF THE RESIDUAL VOLUME
OBTAINED IN DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Positive breathing pressure (mmHg)	Residual Volume (litre B.T.P.S.)	
	Mean	S.E.
Rest	1.61	±0.04
Pressure breathing		
(a) No counterpressure		
10	1.73[1]	±0.05
20	1.82[2]	±0.04
30	1.88[2]	±0.05
35	0.90[2]	±0.06
(b) Chest counterpressure		
20	1.72[1]	±0.03
40	1.86[2]	±0.05
60	1.91[2]	±0.06
80	1.89[2]	±0.09
(c) Trunk counterpressure		
20	1.75[1]	±0.0.
40	1.89[2]	±0.06
60	1.86[2]	±0.05
80	1.92[2]	±0.04

Significance of difference from resting value
[1] 0.001 < P < 0.01
[2]   P < 0.001

subjects. In the absence of respiratory counterpressure all the subjects showed a considerable increase of tidal volume. In contrast there was only a small increase of tidal volume when trunk counterpressure was applied by the pressure jerkin.

The residual volume – The residual volume was calculated from the dilution of the alveolar nitrogen produced by re-breathing oxygen in the following manner:

If

F_{AN2} = concentration of nitrogen in the alveolar gas before re-breathing

$F_{AN2'}$ = concentration of nitrogen in gas from neck of re-breathing bag at the end of the third expiration

F_{N2} = concentration of nitrogen in oxygen placed in the re-breathing bag

V_B = volume of oxygen in bag and the attached limb of the tap; corrected to the pressure and temperature conditions existing in the respiratory tract

V_D = volume of apparatus from mouthpiece to tap

then the residual volume, V_R is given by:

$$V_R = \frac{V_B(F_{AN2'} - F_{N2})}{(F_{AN2'} - F_{AN2'})} - V_D$$

The initial studies of the alveolar gas composition when air was breathed during the control and pressure breathing periods were performed in order to determine the alveolar nitrogen concentration immediately prior to re-breathing of oxygen. These studies showed that pressure breathing caused no significant change of the concentration of nitrogen in end-expiratory Haldane-Priestley samples of alveolar gas. The mean concentration was 80.00% (S.E. ± 0.05) and this value was employed in the calculation of the residual volume. The oxygen used in these experiments contained 0.3% nitrogen. Finally the dead space of the apparatus, which was measured by water displacement, was 70 ml.

The residual volume of each subject was measured in duplicate at each breathing pressure with varying degrees of respiratory counterpressure. The means of the residual volumes obtained with the four subjects under various experimental conditions are presented together with their respective standard errors in Table 4–4. Pressure breathing produced a small but significant increase of the residual volume. The degree of increase varied with the type of counterpressure applied to the trunk.

Chest radiographic studies – Limited radiographic studies were made of the chest during pressure breathing with either chest or trunk counterpressure. Antero-posterior and lateral radiographs were taken of the chest with the subject seated 6 ft from the X-ray tube. One pair of radiographs were taken at rest and a second pair were taken during pressure breathing with the pressure waistcoat or pressure jerkin at a positive pressure of 80 mmHg. Care was taken to reduce to a minimum movement of the subject when pressure breathing was induced. All the radiographs were taken at the end of a quiet expiration. These studies were conducted on the four subjects who were experienced in pressure breathing.

Results – Very Similar changes were induced by pressure breathing in the four subjects. Typical antero-posterior chest radiographs are presented in Fig. 4-5. During pressure breathing the sternum was slightly elevated in relation to the vertebral column. The pressure breathing also caused some straightening of the thoracic portion of the spine. With trunk counterpressure there was no significant change in the level of either of the domes of the diaphragm in relation to the vertebral bodies, and the costophrenic angles were slightly increased by a positive pressure of 80 mmHg. Pressure breathing with chest counterpressure resulted in descent of the diaphragm and widening of the costophrenic angles. The most striking effect of pressure breathing was upon the size of the heart shadow. The maximum transverse diameter of the cardiac shadow was reduced by 10 to 20%. The cardio-thoracic ratio was calculated for each subject at rest and during pressure breathing and the values are presented in Table 4-5. The vascular markings of the lungs both at the hila and in the periphery were smaller and less dense during pressure breathing than at rest.

Respiratory Flow Pattern – Total respiratory flow was recorded in the four subjects at rest and during pressure breathing at various levels with and without respiratory counterpressure. In all the experiments the subject, who was seated within the decompression chamber, wore the modified pressure helmet fitted with a mouthpiece. The mouthpiece was connected to the exterior of the decompression chamber by 20 cm of smooth bore hose (2.5 cm internal diameter). A heated flowmeter (Fleisch) was inserted in the hose. This simple breathing assembly was employed in order to avoid the use of non-return valves in the system. The total volume of the breathing system from the mouthpiece to the exterior of the decompression chamber was 200 ml. The bladder of the pressure garment when one was used and the face compartment of the pressure helmet were connected to the exterior of the decompression chamber by a second wide bore pipe. The pressure created across the flowmeter by flow through it was measured by means of a capacitance manometer and amplified. The output of the amplifier was fed on to the galvanometer of a bromide paper recorder. During the third minute of each rest and pressure breathing period the respiratory flow was recorded for twelve complete breathing cycles. Each of the subjects was exposed to pressure breathing at positive pressures of 15 and 30 mmHg without respiratory counterpressure and to positive breathing pressures of 30, 50 and 80 mmHg on two occasions, once wearing a pressure waistcoat and once wearing a pressure jerkin.

Results – The general shape of the respiratory flow patterns recorded under the same experimental conditions was similar in the four subjects studied but between subjects there was a considerable quantitative variation. Some of the recordings obtained from subject A are presented in Fig. 4-6. During the control period the inspiratory flow pattern was smooth and rounded, whilst during expiration the maximum flow value was attained rapidly. The peak inspiratory flow was greater than the maximum expiratory flow whilst the duration of inspiration was less than that of expiration. Pressure breathing without respiratory counterpressure markedly increased the maximum inspiratory flow and the rate at which the flow increased and decreased during inspiration. The maximum expiratory flow was slightly increased and
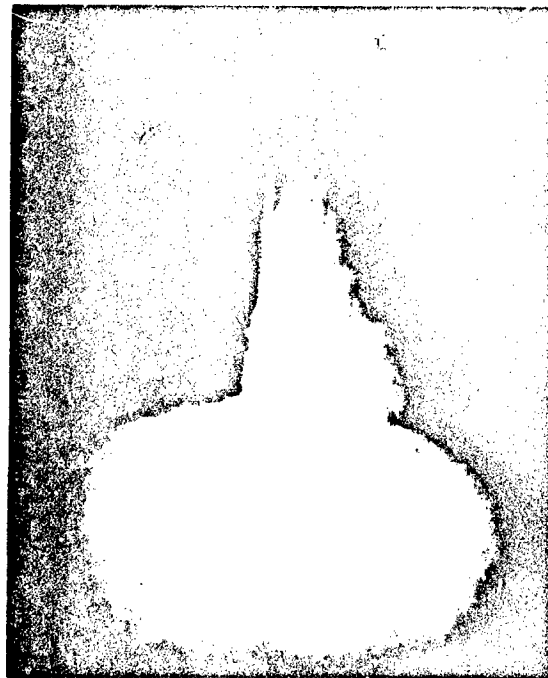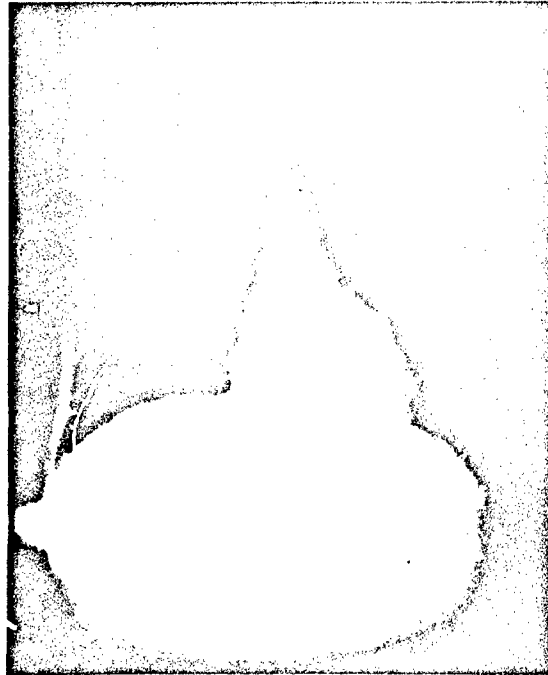
FIG. 4-5 Typical antero-posterior chest radiographs
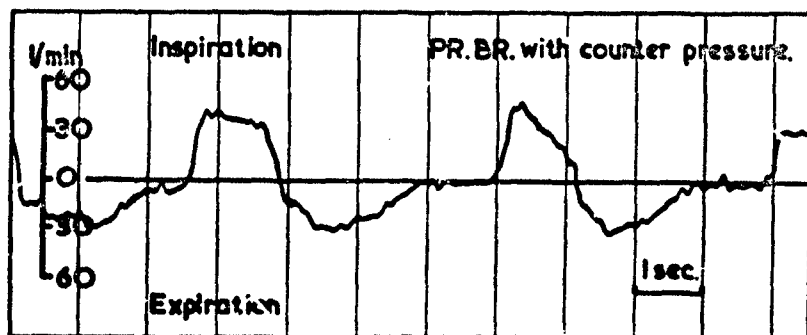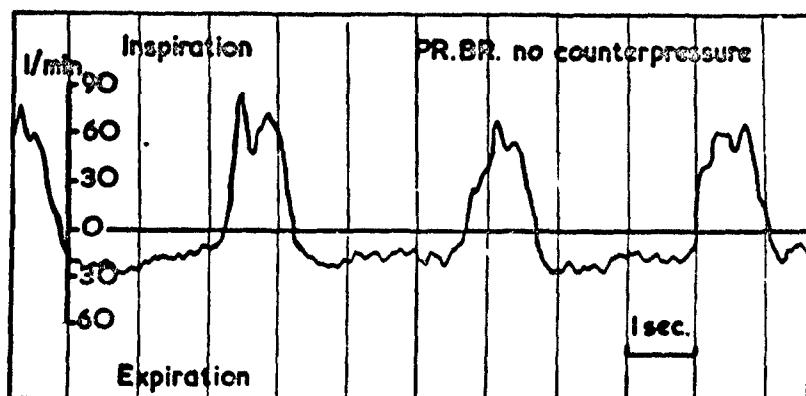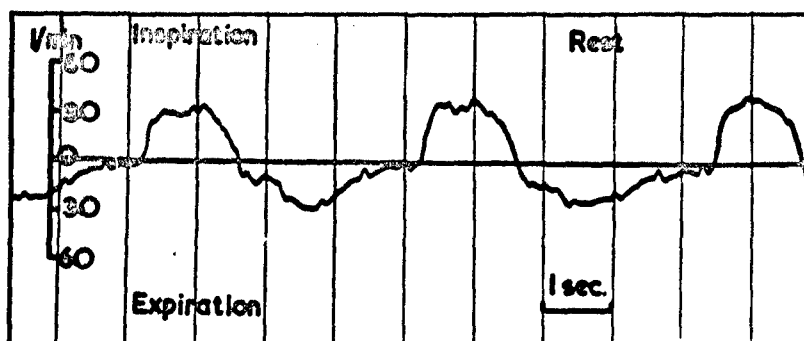
FIG. 4-6 Typical respiratory flow patterns obtained with subject A at rest and during pressure breathing without counterpressure at a positive breathing pressure of 30 mmHg and with trunk counterpressure at 80 mmHg

TABLE 4-5

THE CARDIO-THORACIC RATIO MEASURED
FROM ANTERO-POSTERIOR RADIOGRAPHS OF THE CHEST

Subject	Rest	Cardi thoracic ratio Pressure breathing[1] with chest counterpressure	Pressure breathing[1] with trunk counterpressure
A	0.52	0.42	0.41
B	0.50	0.41	0.43
C	0.51	0.39	0.40
D	0.49	0.38	0.41

[1] At a positive breathing pressure of 80 mmHg.

TABLE 4-6

THE CHARACTERISTICS OF THE RESPIRATORY FLOW PATTERNS
OBTAINED IN SINGLE EXPERIMENTS ON EACH OF FOUR SUBJECTS

(A)

Positive breathing pressure (mmHg)	Maximum flow (1 min.) Inspiration Mean	±S.E.	Expiration Mean	±S.E.	Duration (sec.) Inspiration Mean	Expiration Mean
Rest	39.4	±3.5	31.1	±3.2	1.94	2.69
Pressure breathing						
(a) No counterpressure						
15	40.8	±4.0	34.6	±4.2	1.61	2.52
30	61.1	±6.1	38.1	±5.1	1.32	2.50
(b) Chest counterpressure						
30	50.4	±3.9	30.5	±5.2	1.72	2.65
50	65.1	±7.2	39.1	±4.1	1.61	2.71
80	84.9	±6.9	42.6	±3.9	1.24	2.50
(c) Trunk counterpressure						
30	38.5	±3.2	34.1	±3.1	1.95	2.75
50	40.5	±3.9	30.2	±3.5	2.06	2.50
80	44.5	±4.1	31.7	±3.2	2.01	2.49

(B)

Rate of change of flow (1 sec.[2])

Positive breathing pressure (mmHg)	Inspiration Increase	Decrease	Expiration Increase	Decrease
Rest	1.81	1.25	1.38	0.51
Pressure breathing				
(a) No counterpressure				
15	2.52	2.10	2.56	0.79
30	6.43	4.31	5.21	1.09
(b) Chest counterpressure				
30	2.16	3.05	2.97	0.91
50	3.51	3.21	3.91	0.61
80	5.01	4.57	5.06	2.15
(c) Trunk counterpressure				
30	1.92	1.51	1.61	0.71
50	2.07	1.62	1.53	0.83
80	1.85	1.59	1.63	0.75

tended to be maintained through the greater part of this phase of the respiratory cycle. Similar changes were seen when counterpressure was applied to the thorax during pressure breathing by means of a pressure waistcoat, although at the same breathing pressure the changes were less profound when counterpressure was used. There were only small changes in the pneumotachygram when pressure breathing was performed with trunk counterpressure even at a positive pressure of 80 mmHg.

Each experimental record was analyzed by measuring the maximum inspiratory and expiratory flows, the duration of the inspiratory and expiratory phases and the rate of increase and decrease of respiratory flow at the beginning and end of each phase of the respiratory cycle. Since in many breaths the rate of change of flow varied continuously throughout the respiratory cycle, an arbitrary definition of this measurement was required. The mean rate of increase of flow was measured over the flow range from one tenth to one half the peak flow value. The mean rate of decrease of flow was similarly defined as the rate of decrease from a flow of half to one tenth of the peak value. Each of these measurements was determined for the twelve breaths recorded under each experimental condition and the mean values, and in certain instances the standard errors of the mean values, were calculated. The results of these calculations are presented in Table 4-6. The values for the maximum inspiratory and expiratory flows obtained in the four subjects have been averaged and these mean values are presented in Fig. 4-7.

Transpulmonary pressure – The behaviour of the transpulmonary pressure during pressure breathing was investigated by measuring the difference between the pressure in the oesophagus and that at the mouthpiece. The simple breathing system used in the study of respiratory flow patterns was also used in this investigation. In a proportion of these experiments a wide bore "T" piece was attached outside the decompression chamber to the open end of the hose attached to the mouthpiece. The two arms of this "T" piece completed a closed circuit which consisted of a recording spirometer, a carbon dioxide absorber and a circulating pump. Before each experiment the circuit was flushed with oxygen. The balloon employed to transmit the pressure in the oesophagus was introduced through the mouth or nose and the polyethylene catheter attached to it was attached to one side of a capacitance pressure transducer. A lateral tapping in the mouthpiece was connected to the reverse side of the capacitance transducer. Respiratory flow was measured by means of a heated Fleisch flowmeter placed in the pipe between the mouthpiece and the chamber wall. The pressure drop created across the flowmeter by flow was recorded by a capacitance manometer and an appropriate amplifier. The amplified outputs of the two capacitance transducers were fed on to the galvanometers of a bromide paper recorder. The respiratory flow and mouth-intraoesophageal pressure difference were continuously recorded for six to twelve complete respiratory cycles with the subject at rest and whilst pressure breathing with various degrees of respiratory counterpressure. The recordings were made during the second and subsequent minutes of exposure to pressure breathing. The spirometer record was carefully marked when the recording camera was started and stopped.

Results – A typical experimental record obtained from a subject at rest and whilst pressure breathing at a positive pressure of 30 mmHg with trunk
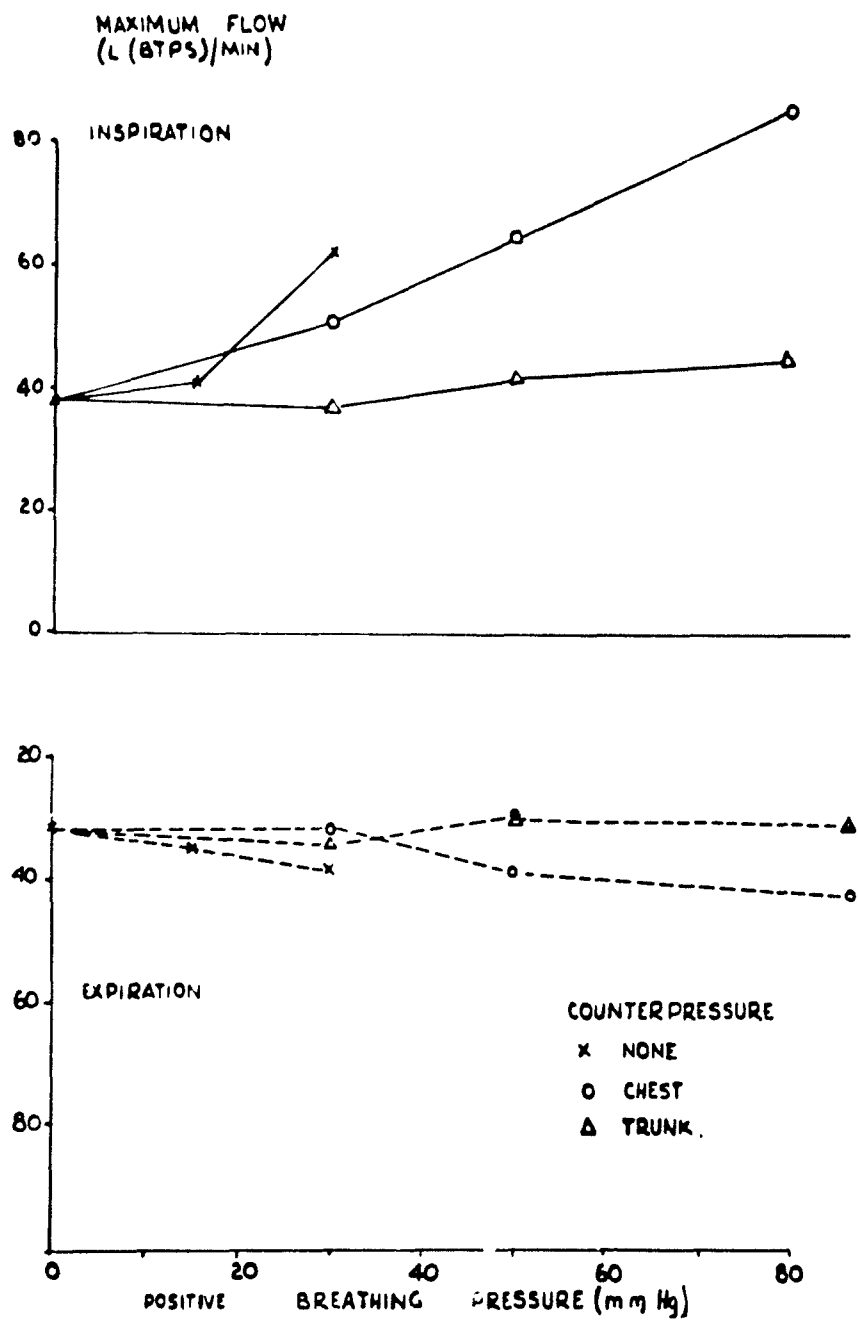
Fig. 4-7 The effect of pressure breathing upon the maximum inspiratory flow (solid line) and maximum expiratory flow (interrupted lines) with no respiratory counterpressure (X), with chest counterpressure (O) and trunk counterpressure (`). Each point represents the mean of the values obtained in four subjects

counterpressure is presented in Fig. 4-8. The oesophageal pressure was less than that at the mouthpiece throughout the respiratory cycle. The mouth-intraoesophageal pressure difference rose during inspiration and fell during expiration. The instant during the respiratory cycle at which the mouth-intraoesophageal pressure difference reached its minimum and maximum values varied with the pattern of respiratory flow. During pressure breathing there was no gross change in the relationship between respiratory flow and this pressure difference. The difference between mouth and intraoesophageal pressures at the end of expiration was, however, increased in certain of the pressure breathing experiments. The simultaneous records of respiratory flow and of the difference between mouth pressure and that in the oesophagus obtained in these experiments were subjected to three distinct analyses:

End Expiratory Mouth-Intraoesophageal Pressure Difference – The mouth-intraoesophageal pressure difference was measured at the end of each expiration and a mean value calculated for each experimental condition. The mean values of this pressure differential are plotted for the four subjects in relation to the breathing pressure in Fig. 4-9. The difference between mouth and intraoesophageal pressures increased markedly with breathing pressure when no respiratory counterpressure was applied. The use of a pressure jerkin reduced the mouth-intraoesophageal pressure differences to values which were only slightly greater than those of the control period.

Pulmonary Compliance – The change of the mouth-intraoesophageal pressure difference during each respiratory cycle was measured by noting the values of this pressure difference at the instant at which the respiratory flow was zero. The change of intraoesophageal pressure was then obtained by subtracting the difference at the end of expiration from that at the end of inspiration. The volume of the expiration was determined either by plani-metric integration of the area under the expiratory flow curve or from the spirometer record when this was available. Lung compliance (C_L) was calculated from the relationship:

$$C_L = \frac{V_T}{\Delta P_{oes}}$$

where

    V_T = expiratory tidal volume (litre B.T.P.S.)

    ΔP_{oes} = change of intraoesophageal pressure during expiration (cm water)

The lung compliance was calculated for each of the six to twelve expirations recorded in each experimental situation. The mean and standard error of these values for the four subjects were then determined and the results are presented in Fig. 4-10. Pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure caused a slight decrease of pulmonary compliance whilst pressure breathing at 80 mmHg with trunk counterpressure did not cause a significant change of compliance.

Non-Elastic Pulmonary Resistance – The record of respiratory gas flow and mouth-intraoesophageal pressure difference for each complete respiratory cycle was divided into a series of intervals each of 0.2 sec. duration. The volume of gas respired in each 0.2 sec. period was calculated by integrating
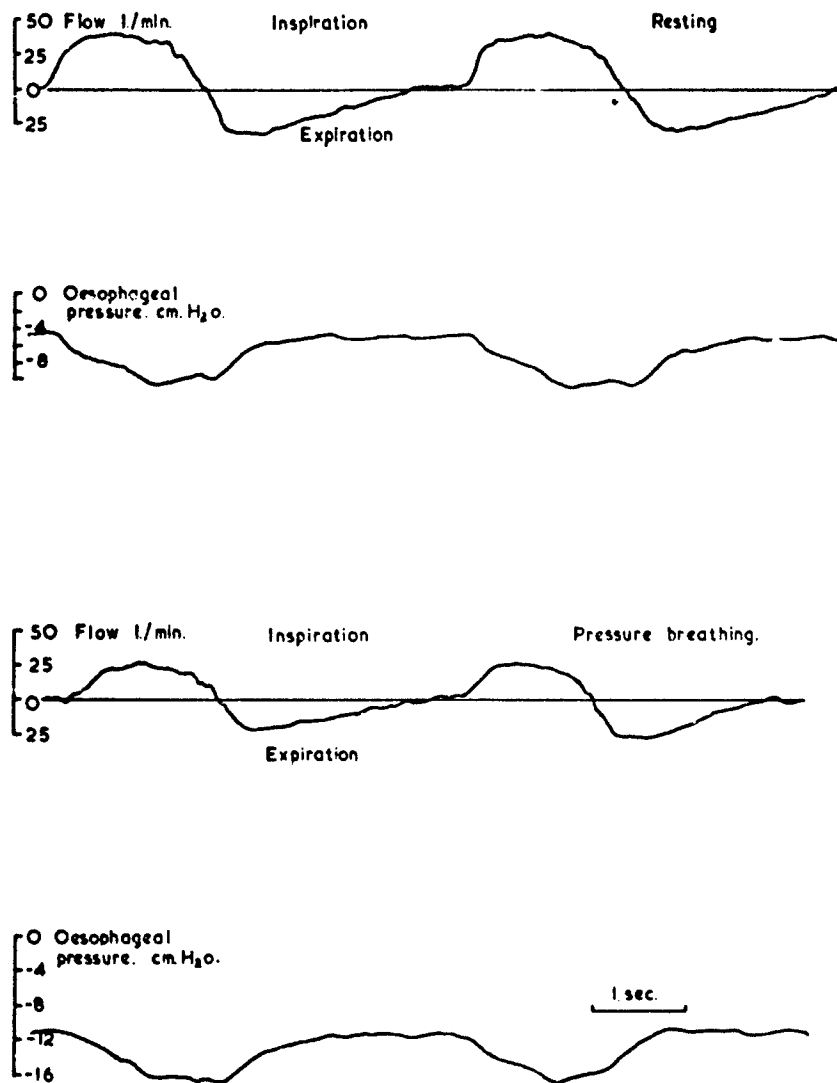
FIG. 4 8 A record of mouth-intraoesophageal pressure and respiratory flow obtained from subject C at rest and during pressure breathing at a positive breathing pressure of 300 mmHg with trunk counterpressure

ENO-EXPIRATORY MOUTH- INTRAOESOPHAGEAL
PRESSURE DIFFERENCE (CM. WATER)
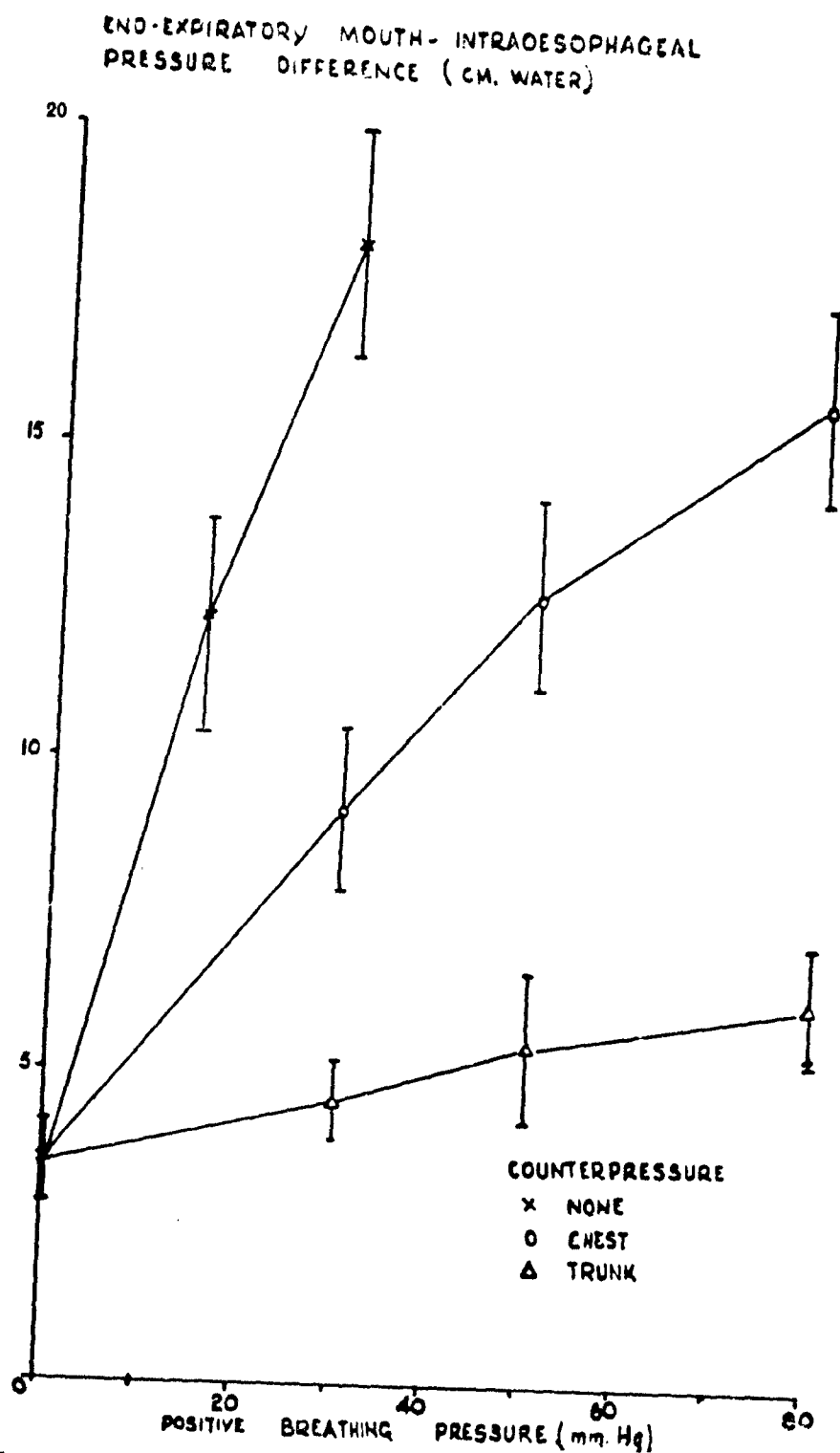
FIG. 4-9 The effect of pressure breathing upon the mouth-intraoesophageal pressure difference at the end of expiration using either no respiratory counterpressure (X), or chest counterpressure (O), or trunk counterpressure (△). Each point is the mean of twelve values for each of the four subjects and the vertical bar represents ±1 S.E.

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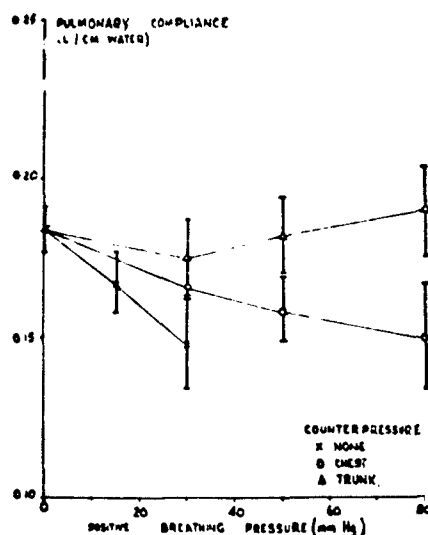
Fig. 4-10 Pulmonary compliance during pressure breathing with no respiratory counterpressure (X). Chest counterpressure (O), or trunk counterpressure (Δ). Each point is the mean of twelve values for each of three subjects. The vertical bar depicts ±1 S.E.
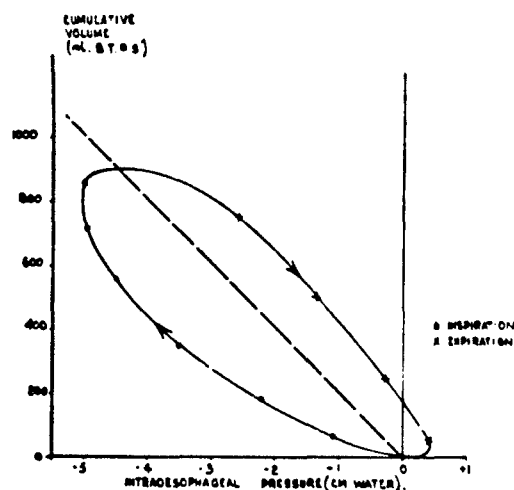
Fig. 4-11 A typical pressure volume loop for the lung and the gas within it for a single respiratory cycle plotted from the measurements made during pressure breathing at a positive breathing pressure of 30 mmHg without respiratory counterpressure. The intraoesophageal pressure has been plotted as the difference from the value which existed at the end of the previous expiration. The elastic resistance of the lungs is depicted by the interrupted line

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the area between the respiratory flow curve and the zero flow line for the period. The value of the mouth-intraoesophageal pressure difference at the end of each 0.2 sec. period was then plotted against the cumulative inspiratory or expiratory volume to give the pressure volume diagram of the lung and the gas within it for that particular breath (Fig. 4-11). The values of the mouth-intraoesophageal pressure difference were expressed relative to the value of the pressure difference which existed at the beginning of the inspiration. The proportion of the change of the mouth-intraoesophageal pressure difference which was expended in overcoming the elastic resistance of the lungs was assumed to vary directly with the change of lung volume. It was depicted on the pressure-volume diagram as a straight line joining the values of mouth-intraoesophageal pressure difference at the beginning and the end of inspiration. The difference at any point between total change of the mouth-intraoesophageal pressure difference and that depicted by this straight line gave the non-elastic component of the pressure change. The magnitude of the non-elastic component was measured at various respiratory gas flows. Mean curves relating the non-elastic component of the mouth-intraoesophageal pressure difference to the respiratory gas flow were constructed from the six to twelve breaths recorded in each experimental situation. Typical curves are presented in Fig. 4-12. All the curves relating the non-elastic transpulmonary pressure to respiratory flow were virtually linear. Departure from linearity only occurred at flows greater than 50 litre min. The non-elastic resistance of the lungs (tissues and air) is given by the slope of such a curve. The slope of each of the experimental curves was measured at a flow of 30 litre/min. and expressed as a change of transpulmonary pressure (cm water) per unit of respiratory flow (litre/sec.). The mean values of the non-elastic respiratory resistance obtained in each experimental condition are presented together with their standard errors in Table 4-7. Pressure breathing without counter-pressure caused a marked reduction of the non-elastic resistance of the lungs. A similar reduction was caused by pressure breathing with chest counter-pressure at a higher pressure. When full trunk counterpressure was employed, however, no change of non-elastic resistance was produced by pressure breathing.

Intragastric-mouth pressure difference – The behaviour of the abdominal pressure during pressure breathing was investigated by recording the pressure within the stomach. In order that the pressure difference across the diaphragm could also be assessed the pressure in the lower part of the oesophagus was also recorded in many of these experiments. The intragastric and intraoesophageal pressures were recorded by means of a double balloon system which was swallowed until the distal balloon was in the stomach and the proximal balloon in the oesophagus. The pressures transmitted from the balloons were measured by means of a pair of capacitance pressure transducers. The reverse side of each of these transducers was connected to a lateral tapping in the mouthpiece. Respiratory flow was recorded by means of a heated flowmeter and a capacitance pressure transducer. Pressure breathing was induced by the technique employed in the previous group of experiments. Records of mouth-intraoesophageal and intragastric-mouth pressure differences and respiratory flow were obtained at rest and during pressure breathing with various degrees of respiratory counterpressure. Recordings of
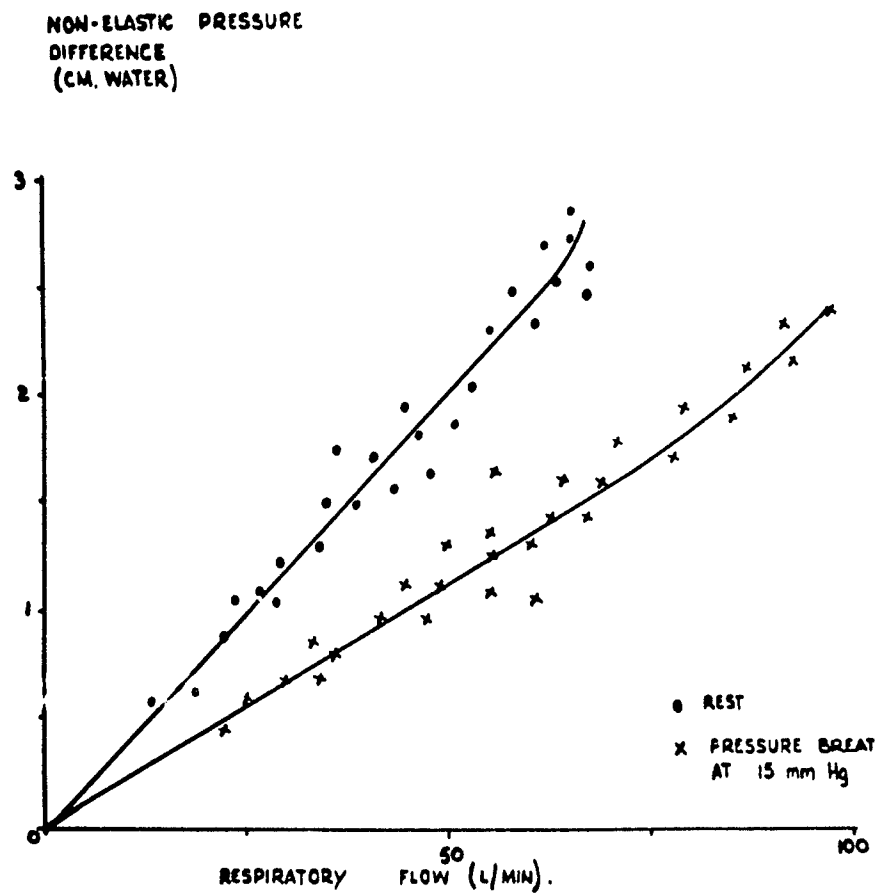
FIG. 4-12 The relationship between the non-elastic component of the change of intraoesophageal pressure and the corresponding respiratory flow in subject A at rest (●) and during pressure breathing without respiratory counterpressure at a positive breathing pressure of 15 mmHg (X)

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TABLE 4-7

THE MEAN VALUES OF THE NON-ELASTIC PULMONARY RESISTANCE
OBTAINED IN DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Positive breathing pressure (mmHg)	Non-elastic pulmonary resistance (cm of water per 1 sec.)	
	Mean	S.E.
Rest	2.36	±0.15
Pressure breathing		
(a) No counterpressure		
15	1.61[1]	±0.20
30	1.14[1]	±0.22
(b) Chest counterpressure		
30	1.63[1]	±0.17
50	1.31[1]	±0.20
80	1.22[1]	±0.27
(c) Trunk counterpressure		
30	2.51	±0.17
50	2.34	±0.14
80	2.27	±0.19

Significance of difference from resting value
[1] P < 0.001

TABLE 4-8

THE MEAN VALUES OF THE MOUTH-INTRAGASTRIC
PRESSURE DIFFERENCE OBTAINED FROM 6–12 RESPIRATORY CYCLES
IN EACH OF FOUR SUBJECTS

Positive breathing pressure (mmHg)	Mouth-intragastric pressure difference (cm water)			
	End-expiratory value		Maximum change during the respiratory cycle	
	Mean	S.E.	Mean	S.E.
Rest	+6.7	±0.8	6.5[1]	±1.8
Pressure breathing				
(a) No counterpressure				
15	−1.3	±1.3	5.5[1]	±2.0
30	−5.4	±1.4	+.3[2]	±2.3
(b) Chest counterpressure				
50	−2.3	±1.6	4.2[2]	±2.1
80	−3.6	±1.4	5.4[2]	±1.9
(c) Trunk counterpressure				
50	+6.3	±1.6	6.1[1]	±1.7
80	+5.6	±1.4	6.4[1]	±1.5

[1] Pressure greater than that at the end of expiration
[2] Pressure less than that at the end of expiration

TABLE 4-9

MEAN VALUES FOR AIRWAY RESISTANCE MEASURED
BY THE INTERRUPTER TECHNIQUE
IN SINGLE EXPERIMENTS ON EACH OF FOUR SUBJECTS

Positive breathing pressure (mmHg)	Airway resistance (cm of water per 1/sec.)	
	Mean	S.E.
Rest	2.55	±0.23
Pressure breathing		
(a) No counterpressure		
30	1.10[1]	±0.29
(b) Chest counterpressure		
80	1.26[1]	±0.27
(c) Trunk counterpressure		
80	2.61	±0.26

Significance of difference from resting value
[1] P < 0.001

six to twelve complete respiratory cycles were taken during the second minute of the exposure to pressure breathing.

RESULTS

Intragastric-mouth pressure difference – Similar records of the behaviour of the intragastric-mouth pressure difference during respiration were obtained from the four subjects and typical records are presented in Fig. 4-13. At the end of expiration at rest the mean intragastric pressure measured relative to mouth pressure was + 6.7 cm water. During inspiration the intragastric pressure rose progressively to reach a maximum towards the end of this phase of the respiratory cycle. The intragastric-mouth pressure difference fell in the early part of expiration to reach a steady value which was sustained until the beginning of the next inspiration. The mean increase of intragastric-mouth pressure difference during inspiration at rest (mean tidal volume 0.68 litre) amounted to 6.5 cm water. Pressure breathing at a positive pressure of 15 mmHg without respiratory counterpressure gave a mean end-expiratory intragastric-mouth pressure difference of 1.3 cm water. In this situation the intragastric-mouth pressure difference increased progressively during inspiration as at rest but the mean increase of pressure during inspiration (tidal volume 0.75 litre) was slightly less, 5.5 cm water. The pressure fell in the early part of expiration only to increase and fall again towards the end of this phase of the respiratory cycle. Pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure considerably changed the shape of the intragastric pressure record. The intragastric-mouth pressure difference actually fell rapidly at the beginning of inspiration and then increased progressively during the remainder of the respiratory cycle to reach a peak towards the end of expiration. The mean intragastric-mouth pressure difference at the end of expiration under these circumstances was 5.4 cm water. Similar changes in the shape of the intragastric pressure record were produced by pressure breathing with chest counterpressure alone. When, however, counterpressure was applied to the whole trunk during pressure breathing, the shape of the intragastric pressure record was virtually unchanged from that at rest. The mean values of the measurements made from the intragastric pressure records obtained during rest and pressure breathing are presented in Table 4-8.

Intragastric-intraoesophageal pressure difference. – The difference between the intragastric and the intraoesophageal pressures was measured at intervals during several respiratory cycles from the experimental records. The mean values of the pressure difference for a given experimental condition were plotted against time from the start of inspiration. The mean curves of the intragastric-intraoesophageal pressure difference throughout the respiratory cycle obtained at rest and during pressure breathing are presented in Fig. 4-14. In all the conditions examined the intragastric pressure was about 10 cm of water greater than the intraoesophageal pressure at the end of expiration. As inspiration occurred the difference increased to reach a maximum just before this phase of the respiratory cycle ceased. The differential pressure decreased progressively during expiration. The general behaviour of the pressure difference during the respiratory cycle was unaffected by pressure breathing. The maximum differential pressure was, how-

Inspiration. Expiration. Resting.

Inspiration Expiration Pressure breathing

2 sec
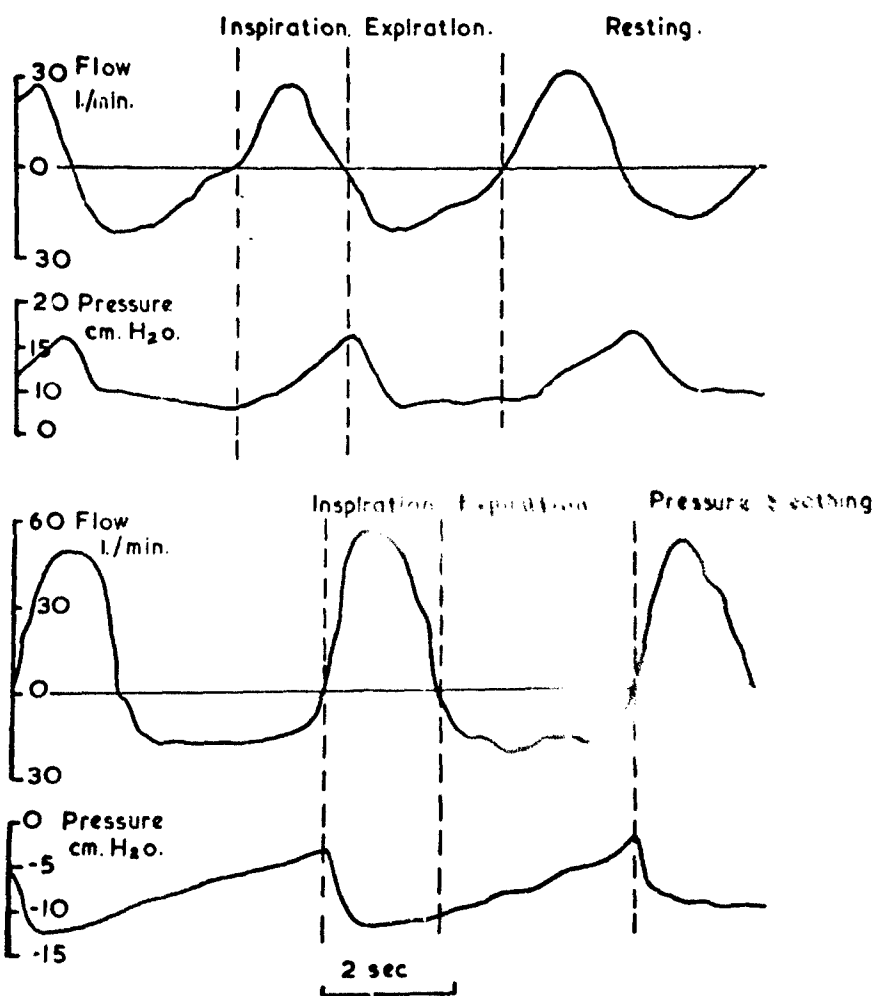
FIG. 4-13 Simultaneous records of respiratory flow (upper trace) and intragastric-mouth pressure difference (lower trace) in subject A at rest (upper record) and during pressure breathing at 30 mmHg without respiratory counterpressure (lower record)
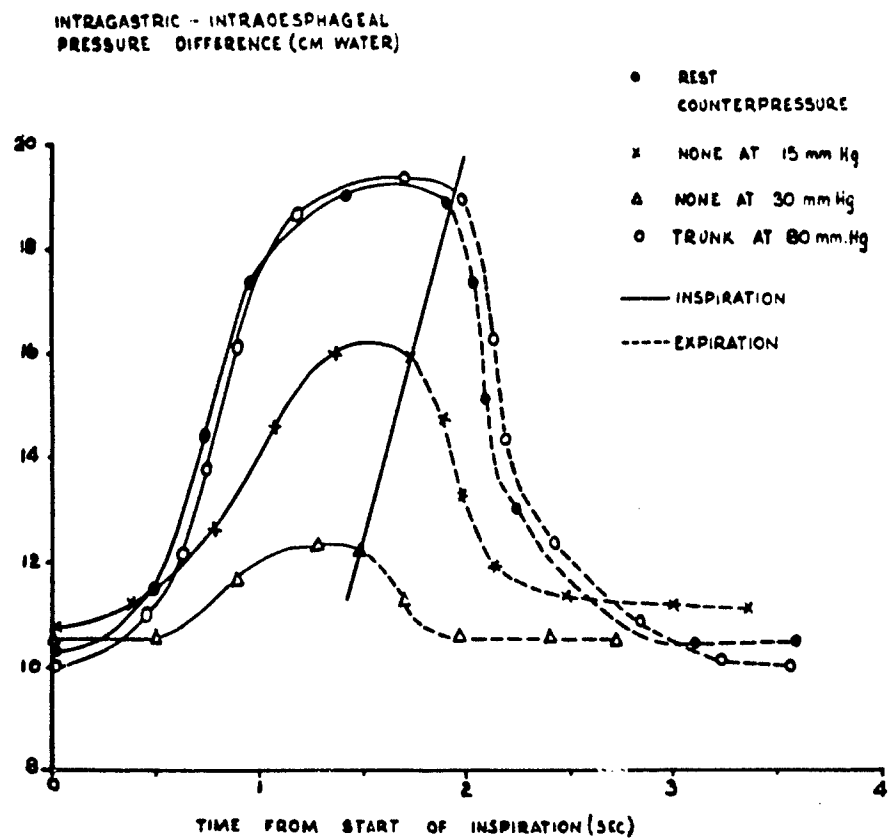
Fig. 4-14 The behaviour of the intragastric-intraoesophageal pressure difference during the respiratory cycle in subject B at rest, whilst pressure breathing at 15 and 30 mmHg without respiratory counterpressure and at 80 mmHg with trunk counterpressure. Each point is the mean value for eight to ten respiratory cycles. The oblique line represents the end of inspiration

ever, considerably reduced by pressure breathing without counterpressure. When counterpressure was applied to the whole trunk there was no decrease of the intragastric-intraoesophageal pressure difference during pressure breathing. **Airway Resistance** – The resistance offered by the respiratory tract to gas flow was measured during pressure breathing by the interrupter technique developed by Otis and Proctor (1948) (230). The subject was seated within the decompression chamber and wore the modified pressure helmet fitted with a mouthpiece. The mouthpiece was connected to the external surface of the chamber by a smooth bore tube (2 cm internal diameter). The flow through this tube was interrupted periodically by means of a pneumatically operated valve which was placed 8 cm from the mouthpiece. A pneumatic control circuit closed this valve for 200 msec every second. The time taken for complete interruption of flow through the valve was 10 to 15 msec. A heated Fleisch flowmeter was placed in the hose between the pneumatic valve and the wall of the decompression chamber. The pressure at the mouth was recorded by means of a differential pressure transducer which was connected to a lateral tapping in the breathing tube close to the mouthpiece. The reverse side of the differential pressure transducer was connected to the external surface of the decompression chamber. The bladder of the pressure garment, when one was worn, and the face compartment of the pressure helmet were connected to the exterior of the decompression chamber by a second wide bore pipe. The outputs of the pressure transducer attached to the flowmeter and the differential pressure transducer connected to the mouthpiece were fed on to the galvanometers of a bromide paper recorder. During the second minute of each rest and pressure breathing period the interrupter was brought into operation and a record taken for ten to twelve complete respiratory cycles. The subject was instructed to increase his respiratory movements towards the end of each recording period. Each of the subjects was exposed to pressure breathing at a positive pressure of 30 mmHg without respiratory counter-pressure and to pressure breathing at 80 mmHg once when wearing a pressure waistcoat and again wearing a pressure jerkin.

Results – In the majority of the experiments the subject reported no un-pleasant sensations whilst the respiratory flow was being interrupted. When interruption occurred during pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure two subjects reported a definite sensation of obstruction, particularly when interruption occurred during inspiration.

A typical experimental record is presented in Fig. 4–15. When the inter-rupter valve closed the respiratory gas flow fell directly to zero. At the moment at which occlusion occurred the mouth pressure changed abruptly and then performed a series of damped oscillations, the mean of which con-tinued to change until the valve was opened. The direction of the pressure change depended upon the phase of the respiratory cycle in which interrup-tion occurred. Occlusion during inspiration reduced mouth pressure whilst occlusion during expiration increased it. The pressure at the mouth at the instant after the interruption of flow was determined by drawing a straight line through the centre of the pressure oscillations and extrapolating it to cut the initial abrupt change in pressure. This intercept was taken as the intra-alveolar pressure at the instant before interruption. The mouth-alveolar
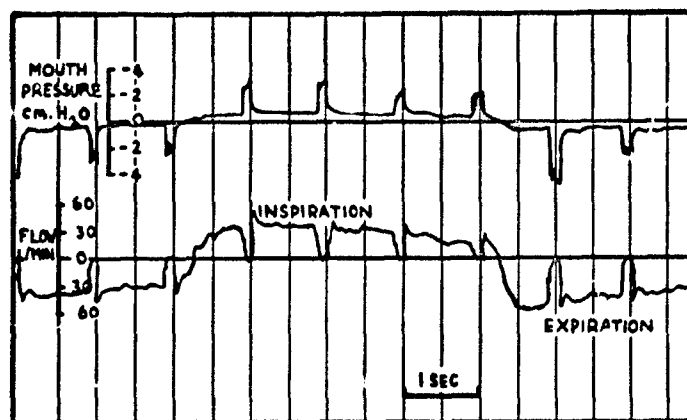
FIG. 4-15 A typical experimental record of the mouth pressure (upper trace) and respiratory flow (lower trace) obtained using the interrupter technique, in subject D at rest
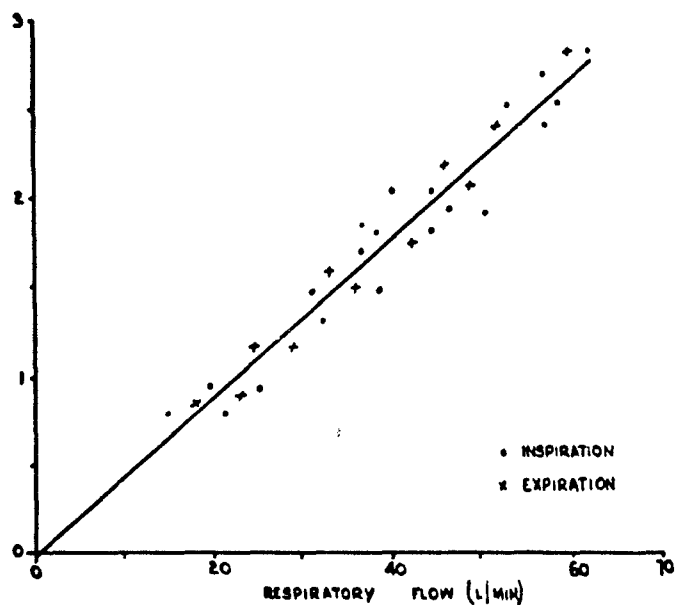
FIG. 4-16 The relationship between respiratory flow and mouth-alveolar pressure difference obtained by the interrupter technique in subject C whilst at rest

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pressure gradient was the difference between this alveolar pressure and the mouth pressure immediately before flow was interrupted. The respiratory gas flow at this instant was determined from the flow record. The corresponding values of the mouth-alveolar pressure gradient and the respiratory flow for each interruption were determined and plotted against each other for each experimental condition and subject (Fig. 4-16). No significant difference was found between the relationship of the mouth-alveolar pressure gradient to flow during inspiration and that obtained during expiration under the same experimental conditions. The relationship between the pressure gradient and flow was only slightly alinear. The value of the respiratory resistance given by this technique was measured from the slope of each of the plotted curves at a flow of 30 litre/min. The resistance was expressed as the pressure difference (cm of water) per unit of flow (litre/sec.). The mean values obtained from the respiratory resistance by the interruption technique in the various experimental situations are presented in Table 4-9. Whilst pressure breathing without counterpressure or with counterpressure to the chest alone caused a marked reduction of resistance, there was no change of resistance when pressure breathing was performed with counterpressure to the whole trunk.

DISCUSSION
Lung Volume – The observation that positive pressure breathing without respiratory counterpressure is generally accompanied by distension of the lung, was made in the early studies of the effects of this manoeuvre (243). The results obtained in the present investigation confirm this observation. In order to permit an assessment of the part played by the elastic forces of the lung and thorax in the increase of lung volume induced by pressure breathing, the pressure-volume relationships of the respiratory system were determined in the relaxed subject. When the respiratory muscles are relaxed voluntarily with the mouth and nose shut the position taken up by the lung and thoracic cage is that at which the net pressure created by the elastic recoil and the weight of these structures is equal and opposite to the pressure difference between the alveolar gas and the environment. Thus the pressure measured at the mouth when it is shut in the relaxed subject at a given degree of lung distension is a measure of the pressure exerted by the elastic recoil of the total respiratory apparatus.

The scatter of the individual experimental points obtained in the present study (Fig. 4-1) was due primarily to the difficulty which the subjects experienced in producing complete relaxation of their respiratory muscles. The mean relaxation pressure-volume curve (Fig. 4-2) constructed from the results obtained from the four subjects was virtually a straight line over a considerable range of lung volume but at both high and low lung volumes the mean values deviated from this straight line so that overall the curve was slightly sigmoid in shape. These results agree closely with those obtained by Knowles, Hong and Rahn 1959 (171) under similar experimental conditions. Detailed analyses of the factors contributing to the relaxation pressure-volume curve have been made by Rahn, Otis, Chadwick and Fenn 1946 (243) and Knowles, Hong and Rahn 1959 (171). These investigators have shown that throughout the vital capacity range the elastic recoil of the lung acts so as to reduce the lung volume. At lung volumes which are less than half the
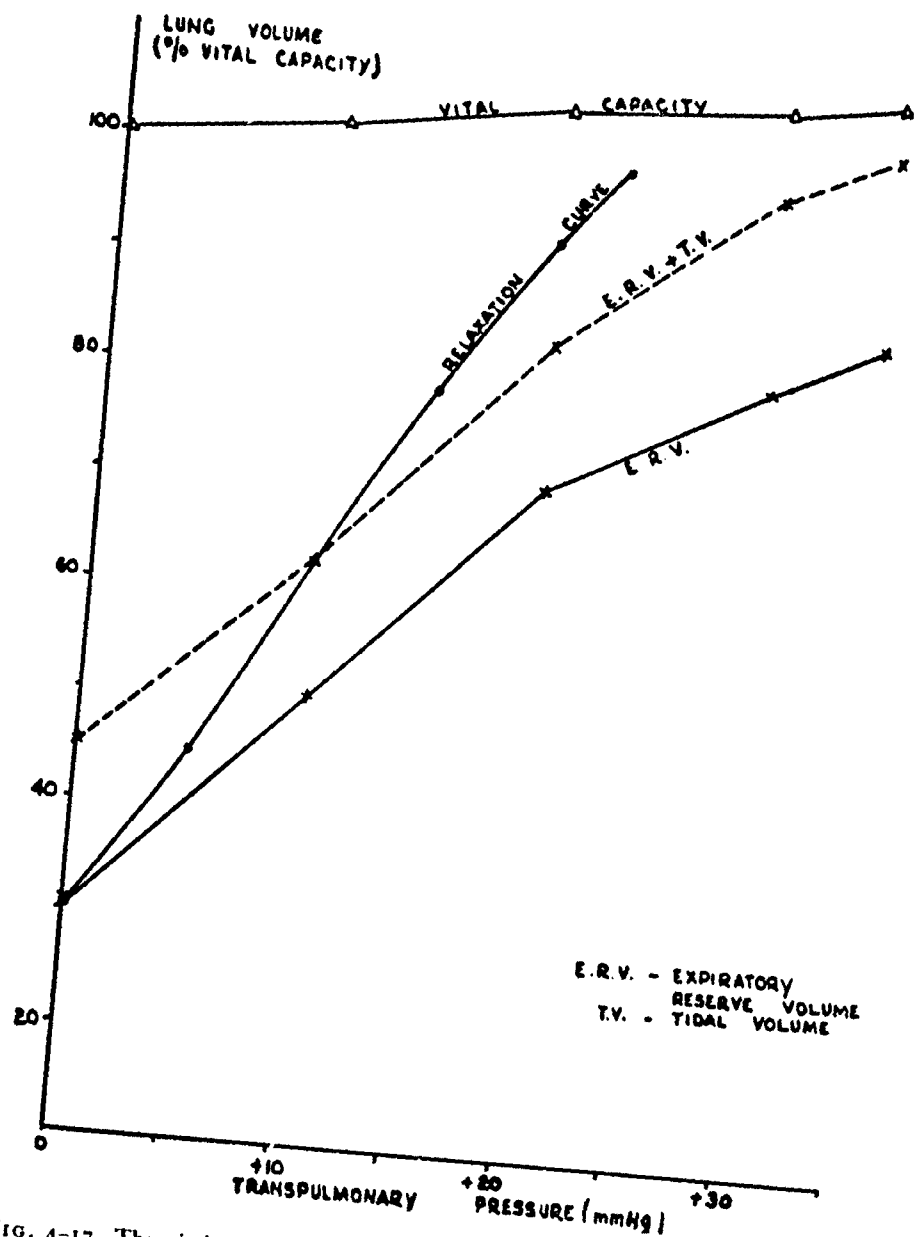
FIG. 4-17 The vital capacity and its sub-divisions in pressure breathing without respiratory counterpressure. Each point represents the mean value obtained from four subjects. The mean relaxation pressure volume curve of the respiratory apparatus for the same four subjects is also shown. All lung volumes have been expressed as a proportion of the mean resting vital capacity

vital capacity the elastic forces of the relaxed thoracic cage tend to increase the lung volume and hence oppose those of the lung itself. When, however, the lung volume exceeds 50% of the vital capacity the elastic forces of the chest wall and abdomen act in the same sense as those of the lung, both tending to reduce the volume of gas within the lung.

The portion of the relaxation pressure-volume curve at lung volumes greater than that which exists at the end of a normal expiration at rest is of direct interest in considerations of the mechanics of respiration during positive pressure breathing. This curve describes the degree of lung distension which would be produced by continuous pressure breathing in the relaxed subject. It may be seen from the curve obtained in the present study (Fig. 4-2) that the lungs of a relaxed subject would be fully distended by a breathing pressure of 24 mmHg. The results of the measurements of the expiratory reserve volume and tidal volume during pressure breathing without respiratory counterpressure have been combined and are presented in Fig. 4-17 together with the relaxation pressure-volume curve already described. It is apparent that at all the pressures investigated the lung volume during pressure breathing was significantly less than that which existed at the same pressure in the relaxed subject. Thus during pressure breathing the respiratory muscles were not relaxed at the end of expiration, in contrast to the condition which exists at the end of expiration during quiet breathing at rest. At positive breathing pressures of less than 10 mmHg the lung volume at the end of inspiration was, however, greater than the lung volume in the relaxed subject at the same pressure.

Under these conditions, therefore, muscular energy was expended during inspiration in overcoming the elastic forces of the respiratory system. When the breathing pressure exceeded 10 mmHg, however, the lung volume throughout the respiratory cycle was less than the relaxed lung volume at the same pressure. Thus during pressure breathing at pressures of greater than 10 mmHg the tone of the expiratory muscles was increased throughout the respiratory cycle as compared with their tone at rest. The discrepancy between the end-expiratory lung volume during pressure breathing and the corresponding relaxation lung volume became greater as the pressure was increased (Fig. 4-17). Thus the tension exerted by the expiratory muscles was increased as the breathing pressure was raised. Whilst in quiet breathing at rest the respiratory muscles are relaxed at the end of expiration and the active phase of the respiratory cycle is inspiration, during pressure breathing at positive pressures in excess of 10 mmHg expiration becomes the active phase and there is active muscular contraction throughout the respiratory cycle. In spite of the maintained contraction of the expiratory muscles throughout the breathing cycle the expiratory reserve volume was increased some two and a half times by pressure breathing at a positive pressure of 35 mmHg. Since the tidal volume was increased at the higher breathing pressures, the reduction of the inspiratory reserve volume caused by pressure breathing was even greater.

Pressure breathing without respiratory counterpressure induces an increase of the vital capacity (Table 4-2). The mean increase of the vital capacity produced by pressure breathing at a positive pressure of 35 mmHg was 350 ml B.T.P.S. The changes underlying this increase of the vital capacity are uncertain since the factors which determine this quantity are not clearly defined.

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Since pressure breathing reduces the intrathoracic blood volume at least two factors could be responsible for the increase of vital capacity induced by this manoeuvre. Firstly, the rise of pressure could increase the distension of the lungs at maximum inspiration. Secondly, the reduction of the volume of the blood within the thorax could lead to an increase in its capacity to hold gas. Although, as Campbell 1958 (54) has pointed out, many investigators have assumed that the important factor which limits the maximum inspiratory and expiratory volumes is the maximum force of contraction of the respiratory muscles, there is a considerable body of evidence which suggests that this is not so. Thus Campbell and Green 1953a (55) have shown that the electrical activity in the abdominal muscles during a maximum expiratory effort is much less than that recorded from these muscles during movements of the trunk. Further, Mills 1959 (214) and Campbell and Green 1953b (56) found that the abdominal muscles contract at the end of a maximal inspiration, thereby limiting the maximum volume of the lung. It would appear, therefore, that the maximum and minimum volumes are limited by reflex changes of the tone of the respiratory muscles. The sites of the receptors which initiate these reflexes are not known but they probably lie within the lung tissue.

Amongst the procedures which have been shown to cause alterations of the vital capacity in normal subjects are those which are associated with changes in the distribution of the circulating blood volume. Thus Hamilton and Morgan 1931 (140) demonstrated for the first time that the reduction of the vital capacity induced by changing from the erect to the supine position could be reduced or even abolished by placing occlusion cuffs around the upper thighs before the posture was changed. Glaser and McMichael 1940 (123) found that a venesection of 380 ml in blood donors caused a mean increase of 153 ml in the vital capacity and 181 ml in the total lung capacity. The observation that the vital capacity was significantly increased when the circulating blood volume was decreased has been confirmed repeatedly (80, 213, 270) although the change of vital capacity was always less than the change of blood volume. The mechanism whereby an alteration in the circulating blood volume induces a change in the vital capacity is obscure. Changes in the intrathoracic blood volume might be expected to alter the total lung capacity since the total capacity of the thoracic cavity is limited and an increase in the blood content of the lung must be compensated by a reduction in the total volume of gas which the lungs can contain. The experimental evidence available confirms that an increase in the circulating blood volume is associated with a reduction of the total lung capacity (269). Mills 1949 (213) concluded that the changes of vital capacity induced by alterations of the blood content of the lungs were probably due to reflex changes in the limitations of respiratory movements.

In view of the large variation of the expiratory reserve volume between one subject and another during pressure breathing, it was decided to measure the residual volume directly rather than to determine the functional residual capacity. The nitrogen dilution technique developed by Rahn, Fenn and Otis 1949 (241) was employed as it was simple and rapid. The standard errors of the values obtained in the present study were very similar to those given by Rahn, Fenn and Otis 1949 (241). The results of the present measure-

ments (Table 4-4) showed that pressure breathing without respiratory counterpressure caused an increase of the residual volume, the mean increase at a positive breathing pressure of 35 mmHg being 300 ml B.T.P.S. This amounted to 5.7% of the resting vital capacity. The observed increase of the residual volume in pressure breathing could have been caused by the raised intrapulmonary pressure acting directly or by the consequent displacement of blood from within the lung to the periphery. The mechanism of the increase of residual volume was investigated by repeating this measurement with counterpressure applied to the trunk by means of a pressure jerkin.

These measurements (Table 4-4) showed that the residual volume was increased by pressure breathing even when the external surface of the trunk was supported. The increase of the residual volume induced by a given breathing pressure was reduced by the application of counterpressure to the trunk. The displacement of blood from the chest caused by pressure breathing is increased by the application of counterpressure. Since trunk counterpressure prevented the lung distension normally induced by pressure breathing it would appear that the increase of the residual volume found in pressure breathing without trunk counterpressure was in part due to displacement of blood from the lungs and in part due to a limitation of full expiration.

The changes of the total lung capacity produced by pressure breathing without counterpressure have been calculated from the separately determined values of the vital capacity and the residual volume. The results of these calculations are presented in Fig. 4-18.

Pressure breathing at a positive pressure of 35 mmHg increased the total capacity of the lungs by 9%. Although part of this increase was due to the displacement of blood from within the thorax, it is probable that the lung was more distended at the end of a maximal inspiratory effort during pressure breathing at 35 mmHg than at the end of a full inspiration at rest. Pressure breathing without trunk counterpressure at positive pressures in excess of 35 mmHg introduces the possibility of lung damage. This aspect has been studied in some detail in animals by several investigators. Thus Polak and Adams 1932 (237) studied the effects of raising the intrapulmonary pressure upon the respiratory and cardiovascular systems in the anaesthetized dog. They also placed a bubble trap in the carotid artery. When the intrapulmonary pressure was raised to 80 to 100 mmHg for a period of ten seconds there was a profound fall of systematic arterial pressure and numerous gas bubbles appeared in the carotid artery trap. In an extensive series of experiments it was found that intrapulmonary pressures of 90 mmHg or more caused immediate air embolism, whilst pressures of less than 80 mmHg did not give rise to air emboli.

Examination of the lung following the application of intrapulmonary pressures above 90 mmHg revealed extensive interstitial emphysema along the vascular sheaths within the lung and in the mediastinum and neck. There was microscopic evidence of rupture of alveoli. Polak and Adams also demonstrated that these effects could be prevented by applying external support to the chest. They concluded, therefore, that the primary mechanism underlying the air embolism produced by high intrapulmonary pressures was overdistension of the lung. Henry 1945 (144) extended this type of study to a wide variety of mammals, including the mouse and the steer. In lightly

anaesthetized animals he introduced a gradually increasing volume of air into the lungs until leakage occurred into the tissues. He found that no leakage of air occurred until the intrapulmonary pressure was raised to between 60 and 100 mmHg. He also showed that widely opening the thorax so that the lungs were unsupported reduced the pressure at which leakage into the tissues occurred to 30 to 60 mmHg. Henry also confirmed the findings of Polak and Adams 1932 (237) that considerably higher intrapulmonary pressures did not cause damage when expansion of the thorax and abdomen was prevented.

Recently Malhotra and Wright 1960 (200) investigated the effects of increasing the tracheal pressure in fresh warm human cadavers. They found that leakage of air from the lungs with interstitial emphysema occurred at a pressure of 80 mmHg. The application of a tight binder to the thorax and abdomen increased the tracheal pressure at which air leakage occurred to about 190 mmHg. This study confirms the conclusion drawn from the animal experiments that when the thorax and abdomen are relaxed rupture of alveoli and air embolism will occur if the intrapulmonary pressure exceeds about 80 mmHg. During ascent through water the intrapulmonary pressure can be considerably greater than the pressure at the surface of the trunk, particularly if the subject fails to allow the gas expanding in the respiratory tract to vent freely through his mouth and nose (77). Over fifty cases of air embolism, the majority of which involved embolism of cerebral vessels have been reported in the literature of submarine medicine. The mechanism underlying this condition is considered to be the same as that observed in animals, i.e. overdistension of the lungs. Overenthusiastic inflation of the lungs in newborn babies suffering from asphyxia neonatorum has also led to a cerebral air embolism and death. The increase of intrapulmonary pressure per se cannot be the direct cause of lung damage since very high intra-pulmonary pressures of the order of 150 to 200 mmHg occur during straining and coughing (263) with no untoward effect upon the respiratory tract. In these situations, however, the lungs are supported since the increase of intra-pulmonary pressure is produced by contraction of the expiratory muscles. Thus there is considerable evidence both from animal experiments and clinical observation that the lung damage produced by a high intrapulmonary pressure is due to overdistension of the alveoli and not to the rise of pressure itself.

In the present investigation the inspiratory reserve was reduced to about 5% of the total lung capacity by pressure breathing at a positive pressure of 35 mmHg (Fig. 4–18), so that the lungs were almost fully expanded at the end of inspiration. It was considered unwise, therefore, to investigate in detail the effects of higher positive breathing pressures without applying external support to the respiratory system. On two occasions, however, experimental subjects were accidentally exposed to a positive breathing pressure of between 60 and 70 mmHg without external support. On both these occasions the pressure applied to the respiratory tract was very much greater than the subject had expected. When the rise occurred there was a marked increase in the circumference of the chest and the subject experienced retrosternal pain. The positive breathing pressure was reduced to zero within ten seconds. The pain which was dull and ill-localized, persisted, however, for about an hour. Immediate clinical and radiological examination of the chest revealed no

FIG. 4-18 The effect of pressure breathing without respiratory counterpressure upon the total lung capacity and its sub-divisions

abnormality on either occasion. These accidental exposures to high breathing pressures in the absence of respiratory counterpressure served to emphasize the potential dangers associated with positive intrapulmonary pressures in excess of 40 mmHg.

The effect of applying counterpressure to the thorax by means of the pressure waistcoat upon the response to pressure breathing was investigated by measuring the total lung capacity and its sub-divisions. The mean values of these measurements have been calculated and are presented in Fig. 4-19. The increases of residual volume and of vital capacity produced by a given breathing pressure when chest counterpressure was employed were not significantly different from those which were produced when no counterpressure was used. The application of counterpressure to the thorax resulted, however, in a much smaller increase in the functional residual capacity than that associated with simple pressure breathing at the same positive pressure (Fig. 4-18). The value of the functional residual capacity associated with pressure breathing without counterpressure at a positive pressure of 35 mmHg was not reached when thoracic counterpressure was used until the intrapulmonary pressure was raised to a positive pressure of 80 mmHg. Thus the application of counterpressure to the thorax reduced the degree of lung distension produced by a given breathing pressure. It did not, however, prevent a significant increase of the volume of gas within the respiratory tract, especially at the higher breathing pressures. Pressure breathing with thoracic counterpressure at positive pressures in excess of about 40 mmHg was found to be extremely tiring because of the need to maintain the muscles of the abdominal wall in a state of continuous contraction. The radiographic studies conducted during pressure breathing with this form of counterpressure showed that in spite of this voluntary contraction there was a marked descent of the diaphragm. The diaphragmatic descent was in fact the principal cause of the observed increase of the functional residual capacity.

The effect of the counterpressure applied to the trunk by the pressure jerkin was also assessed. The increase of the total lung capacity during pressure breathing under these conditions was slightly less than when counterpressure applied to the chest alone. The most striking effect was the relatively small increase of the functional residual capacity (Fig. 4-20). A positive breathing pressure of 80 mmHg caused a mean increase of 0.87 litre B.T.P.S. This increase of the functional residual capacity was probably due to several factors, but it was impossible to determine their relative importance from the measurements available. Direct measurements of limb volume have shown that pressure breathing at a positive pressure of 80 mmHg with trunk counterpressure increases the blood content of the limbs by 300 to 400 ml. A large proportion of the blood displaced from the trunk in this manner probably comes from within the thorax. Thus part at least of the observed increase of the functional residual capacity during pressure breathing with trunk counterpressure was due to the associated shift in the distribution of the circulating blood volume.

The increase of functional residual capacity induced by a positive breathing pressure of 20 mmHg amounted to 70% of that produced by a positive breathing pressure of 80 mmHg. This relationship cannot be explained fully on the basis of the displacement of the blood from the lungs since the total
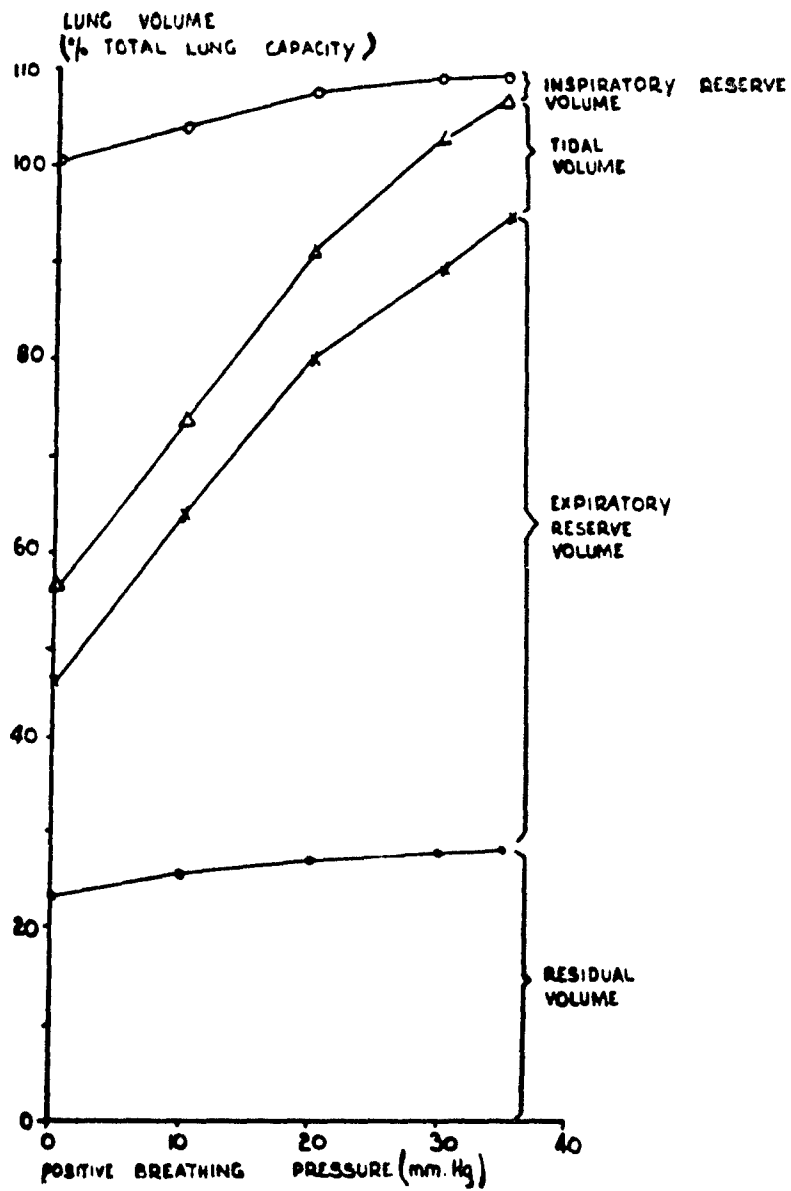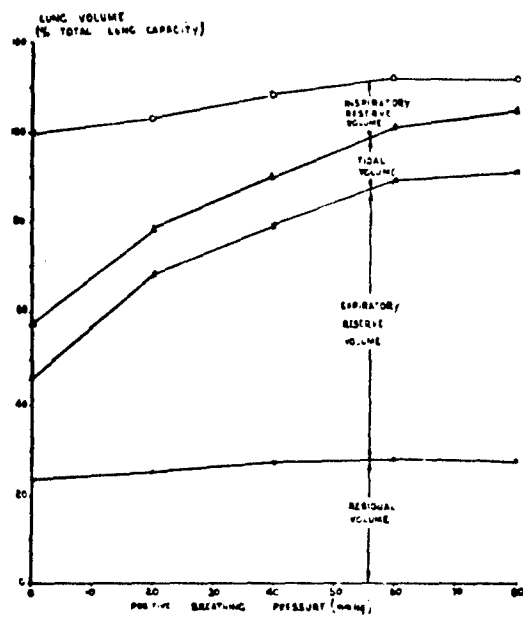
FIG. 4 19 The effect of pressure breathing with chest counterpressure upon the total lung capacity and its sub-divisions
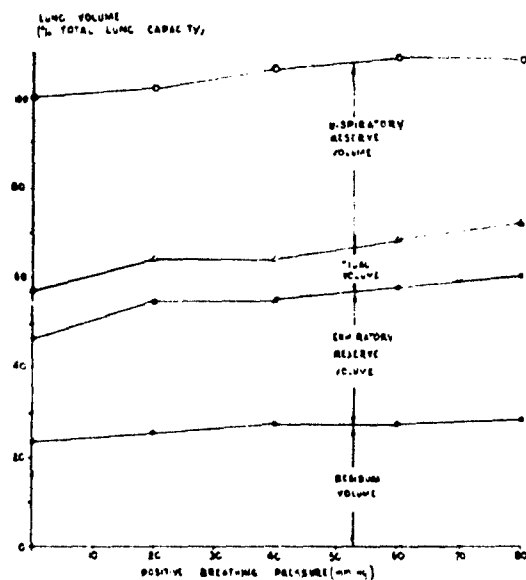
FIG. 4 20 The effect of pressure breathing with trunk counterpressure upon the total lung capacity and its sub-divisions

increment of limb volume caused by a positive pressure of 20 mmHg is only 30 to 40% of that produced by a positive breathing pressure of 80 mmHg. It might well, however, be produced by a failure to achieve, via the jerkin, a pressure to the surface of the trunk as great as that in the respiratory tract. The relaxation pressure volume curve of the total respiratory system obtained using the same group of subjects (Fig. 4-2) showed that the increase of the expiratory reserve volume obtained during pressure breathing at a positive pressure of 80 mmHg with the pressure jerkin was produced in the relaxed state by an intrapulmonary pressure 3 to 4 mmHg greater than the pressure at the surface of the trunk.

At very low positive breathing pressures the inner lining of the jerkin and the inner layer of the garment bladder do not take up the contours of the trunk completely owing to the resilience of the fabric. Further, there are certain areas of the surface of the trunk, namely the upper part of the front of the chest, the supraclavicular fossae and the axillae which are not completely covered by the inflated jerkin. The pressure exerted by a bladder restrained by an outer inextensible fabric layer also varies with the distance from the edge along which the garment is reflected off the surface to which it is applied. Thus at the line at which the reflection occurs the pressure applied to the underlying surface will be only a small fraction of the pressure within the bladder. However, at some distance from this line beneath the bladder the pressure exerted by the garment will equal that of the gas in the bladder. These considerations suggest that the mean pressure applied to the total surface of the trunk by the pressure jerkin is slightly less than the pressure of the gas within its bladder. At a positive breathing pressure of 80 mmHg this discrepancy may well amount to 3 to 4 mmHg, although it is not possible to quantitate this effect accurately.

Under the conditions of the present study the absolute pressure of the gas within the respiratory tract and hence of the gas within the abdomen, was unchanged by the induction of pressure breathing. Pressure breathing was frequently produced, however, by raising the absolute pressure of the gas delivered to the respiratory tract. Even in these circumstances the contribution of the compression of the gas within the abdominal cavity to the increase in the total lung capacity is insignificant. Recent estimates of the volume of gas within the abdominal portion of the alimentary tract have shown that in normal subjects this volume does not exceed 150 to 200 ml (31). Thus the decrease of the volume of the abdominal contents produced by pressure breathing at 80 mmHg amounts to only 20 ml B.T.P.S. at ground level. Further, Mills 1949 (213) has shown that removal of 1 litre of water from the stomach does not alter the vital capacity.

The chest radiographs taken during pressure breathing with trunk counterpressure at 80 mmHg confirmed that this procedure induced very little change in the volume of the thoracic cavity at the end of a quiet expiration. Detailed studies of the apices of the lungs were made in an attempt to discover whether the absence of counterpressure to the supraclavicular fossae and the posterior triangle of the neck resulted in a detectable change at the lung apex during pressure breathing. No change was seen in the shape and position of the lung at a positive breathing pressure of 80 mmHg. The absence of any effect was probably due to the low extensibility of the cervical

pleura. The amount of X-radiation absorbed by the normal lung is determined primarily by the quantity of blood within it. In order to infer changes in blood content from changes in radiotranslucency the conditions under which the various radiographs were taken including the lung volume should be strictly similar. These conditions were fulfilled in the experiments in which chest radiographs were taken during pressure breathing with the pressure jerkin. The obvious increase in the translucency of the lungs during pressure breathing was due, therefore, to a reduction of their blood content. The associated reduction in the transverse diameter of the heart shadow and the consequent increase in the cardiothoracic ratio produced by pressure breathing was also evidence that this procedure displaces blood from within the thorax.
Respiratory Gas Flow – The gross changes induced in the pneumotachygram by pressure breathing without counterpressure were first described by Otis, Sheldon and Rahn 1955 (233). They reported that at positive breathing pressures above 20 mmHg the expiratory pattern consistently showed an abrupt termination, whilst at the beginning of inspiration flow it increased very rapidly to reach a peak value considerably greater than that recorded during rest. The results obtained in the present investigation during pressure breathing without respiratory counterpressure confirmed these earlier observations. A striking feature was the extreme variability in the response of each of the four subjects to pressure breathing. The respiratory flow pattern in one subject at a positive pressure of 30 mmHg was very similar to that recorded at rest. At the other extreme another of the subjects exhibited a large increase of peak inspiratory flow and a flattening of the expiratory flow pattern during pressure breathing. The mean peak inspiratory flow for the four subjects was increased by 55"₀ and the mean rates of increase and of decrease of inspiratory flow were more than doubled by pressure breathing at a positive pressure of 30 mmHg. During expiration the flow remained relatively constant for most of the phase. This plateau value was generally slightly greater than the resting peak expiratory flow.

The rate of increase of expiratory flow to a value equal to about half the maximum value was virtually always equal to the rate of decrease of flow during the preceding inspiration. The flow declined very slowly towards the end of expiration. Thus on the average pressure breathing without counterpressure, particularly at the higher pressure (30 mmHg) used in this study markedly increased the maximum flow and the rate of change of flow during inspiration. These changes were associated in three of the four subjects with an increase in the tidal volume, a 50"₀ reduction of the duration of inspiration and a decrease of the ratio of inspiratory time to total cycle time from a mean control value of 0.37 to one of 0.29. During pressure breathing at positive pressures above 10 mmHg inspiration occurs by relaxation of the expiratory muscles. The increased peak flow and the increased rate of change of flow produced by pressure breathing at 30 mmHg in three of the four subjects used in this study suggested that nervous control of respiration under these conditions is less precise than during normal inspiration, when the lung volume is increased by contraction of the inspiratory muscles. The flattening of the expiratory flow pattern produced by pressure breathing was very similar to the change produced by the imposition of resistance to expiration (268).

The application of counterpressure to the chest by means of a pressure waistcoat reduced the disturbances of respiratory flow induced by breathing without counterpressure at a positive pressure of 30 mmHg (Fig. 4-7). At the higher positive breathing pressures, 50 and 80 mmHg, however, there was a marked increase in the maximum flow and the rate of change of flow during inspiration. At the highest positive breathing pressure studied (80 mmHg) the mean peak inspiratory flow was more than doubled. In contrast to the marked changes produced by a positive pressure of 80 mmHg with chest counterpressure, pressure breathing at this level with trunk counterpressure produced only minor changes in the respiratory flow pattern. Thus the profound changes produced by pressure breathing when chest counterpressure alone was used, was due to the absence of support to the abdomen. This observation suggests that the nervous co-ordination of the partial relaxation of the abdominal muscles during inspiration under these conditions was less precise than is the normal co-ordination of the inspiratory muscles.

Intraoesophageal Pressure – The value of the intraoesophageal pressure as an indirect measure of the mean intrapleural pressure has been studied by several investigators (210) (60) (208) (171). Comparisons of intraoesophageal pressure with the pressure in a small pneumothorax have shown that there is no consistent relationship between the absolute values of these pressures although in the same subject changes of intraoesophageal pressure do approximate to those of pleural pressure. The agreement between the change of intraoesophageal pressure and change of intrapleural pressure is best in the erect posture (208) and when the former is measured in the lower third of the oesophagus. The discrepancies introduced when the pressure is measured in the middle third of the oesophagus or when the subject is in the supine position are probably due to external compression of the oesophagus by the contents of the mediastinum. The marked increase of the difference between the intraoesophageal pressure and that at the mouth which occurs during a maximal expiration is probably due to the same mechanism (171).

Thus changes of intraoesophageal pressure reflect fairly closely the simultaneous changes of intrapleural pressure in the erect posture provided that the lung volume is not reduced below the normal end-expiratory level. Mead and Gaensler 1959 (208) also demonstrated that the flow resistance component of the intraoesophageal and intrapleural pressures corresponds more closely than do the elastic components of these pressures. Thus greater reliance may be placed upon the values of the non-elastic resistance to air flow calculated from the recorded changes of intraoesophageal pressure than on values of pulmonary compliance calculated from the same pressure changes. Care was taken in the present investigation to place the recording balloon in the lower third of the oesophagus and the lung volume was always increased by pressure breathing. Since also all the experiments were conducted with the subject in the seated position, it was concluded that this method of measuring changes of intrapleural pressure was suitable for the study of the mechanics of respiration during pressure breathing.

Rohrer 1915 (249) was the first to make a detailed analysis of the mechanical behaviour of the respiratory system. He considered three groups of forces, namely:

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(a) **elastic,** which depend upon the degree of deformation of the system and hence up   the volume of the lungs;

(b) **frictional,** generated by the resistance to gas flow of the respiratory tract and the resistance of the lung parenchyma and surrounding tissues to deformation, which depend upon the rate of change of volume of the lungs and

(c) **inertial,** generated by the acceleration of the system which depend upon the rate of change of the volume of the lung.

Rohrer considered that under normal conditions the inertial forces were negligible. Recently Mead 1956 (207) demonstrated experimentally that during normal breathing the pressure required to overcome the inertia of the lungs and the gas within them amounts to about 0.5% of the total pressure change in the pleural space during the respiratory cycle. Even during heavy exercise Mead calculated that the fraction of the total pleural pressure swing which is exerted against inertial forces is less than 5%. Thus when considering the lungs and the gas within them the forces which must be overcome during respiration may be separated into two fractions: an elastic component which is directly proportional to the change in volume and a non-elastic component which varies with the rate of gas flow (28) (210) (211). This is the form of analysis which was used in the present investigation.

Lung Compliance – At the end of expiration when there is no air flow through the airways, the difference in pressure between the mouth and the pleural space is a function of the elastic properties of the lungs. In all the experiments the intraoesophageal pressure was less than the pressure at the mouth at the end of expiration and this pressure difference was increased by pressure breathing. Pressure breathing without respiratory counterpressure caused a marked increase of this pressure difference, the increase being greater at the higher breathing pressures. The application of counterpressure to the chest reduced the increase of the mouth-oesophageal pressure difference induced by a positive breathing pressure of 30 mmHg in the absence of counterpressure, but even with chest counterpressure a positive breathing pressure of 80 mmHg induced a very large increase of this pressure difference. When trunk counterpressure was employed the increase of the mouth-intraoesophageal pressure was relatively small. Since in some of these experiments the change of lung volume induced by the pressure breathing was also recorded it is possible to compare the latter with the corresponding increase of the mouth-intraoesophageal pressure difference, Fig. 4–21. For a given subject there was, over a wide range of lung volumes, a linear relationship between the change of the expiratory reserve volume and the end-expiratory value of the mouth-intraoesophageal pressure difference. The linearity of this relationship suggests that the elastic properties of the lungs are unchanged over a wide range of lung volumes and further that these properties are not affected by pressure breathing. These conclusions were confirmed by the values of the lung compliance obtained in these experiments (Fig. 4–10). The increase in the end-expiratory mouth-intraoesophageal pressure difference produced by pressure breathing was due, therefore, to the concomitant increase of the end-expiratory lung volume which this manoeuvre induced.

The absence of any significant change of pulmonary compliance following the induction of pressure breathing at a positive pressure of 80 mmHg when
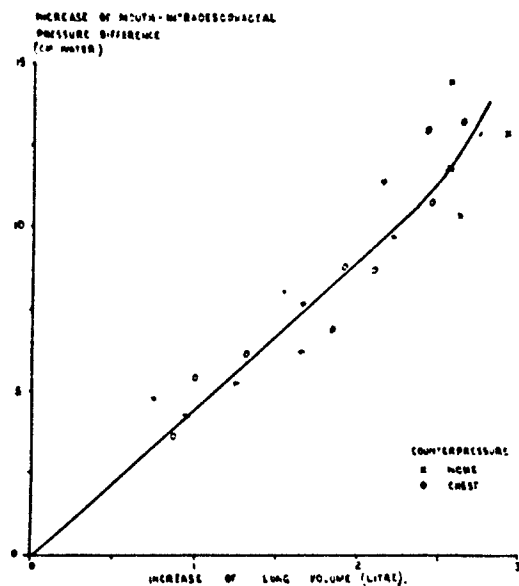
FIG. 4-21 The relationship between the increase of the expiratory reserve volume and the corresponding increase of the mouth-intraoesophageal pressure difference at the end of expiration in subject B during pressure breathing
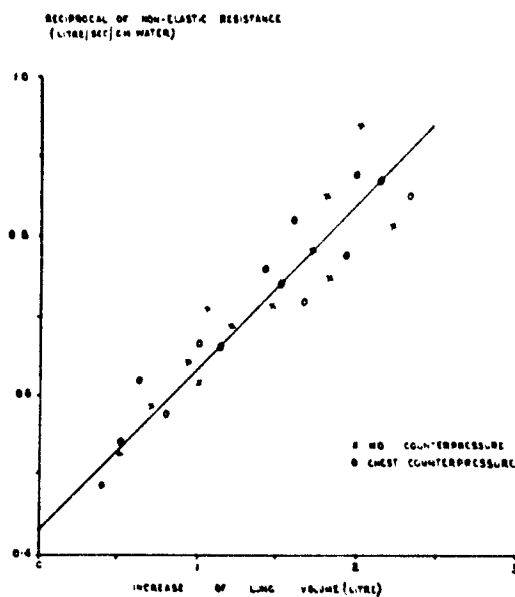
FIG. 4-22 The relationship between the increase of lung volume induced in subject C by pressure breathing with and without chest counter pressure and the reciprocal of the corresponding value of the non-elastic resistance

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trunk counterpressure was employed is of particular interest. In this condition the blood content of the lungs was considerably reduced. This observation suggests, therefore, that the elasticity of the lungs was not influenced by a reduction of their blood content. Several studies of the effect of pulmonary congestion upon lung compliance have been made in animals but there is some conflict between the results of various investigators. Thus Drinker, Peabody and Blumgart 1922 (81) showed that constriction of the pulmonary veins about halved the compliance of the lungs of cats with intact chests. In isolated dog's lungs, Mack Grossman and Katz 1947 (199) found that the injection of blood into the pulmonary vessels markedly reduced the distensibility of the lung. These and other studies (153) were, however, marred by the absence of any measurement of the pulmonary vascular pressures achieved during congestion. In 1955 Frank, Radford and Whittenberger (113) found that increasing the pulmonary capillary pressure from 0 to 12 mmHg in excised lungs did not change the compliance. Hughes, May and Widdicombe 1958 (160) investigated the effects of increasing the vascular pressure in perfused lungs in the rabbit and cat and in the intact cat. In isolated lungs they found that the mean reduction of compliance per mmHg of increase of pulmonary vascular pressure was 0.8%, whilst in cats with intact chests the mean decrease of compliance was 0.3% per mmHg increase of left atrial pressure.

These animal experiments suggest, therefore, that even moderate pulmonary congestion causes only a small decrease of pulmonary compliance. Studies in man have also given rise to conflicting results. Thus there is no doubt that in mitral stenosis and congestive heart disease the pulmonary compliance is reduced (62,) (260). Saxton et al found, however, that no correlation existed between compliance and pulmonary wedge pressure in patients with heart disease. Further following alleviation of the pulmonary congestion by mitral valvulotomy, the compliance did not increase. There is little doubt that in chronic pulmonary congestion the reduction of compliance is due to secondary vascular and parenchymal changes such as fibrosis. Further, pulmonary oedema is known to produce a marked reduction of compliance (160) and to some degree oedema generally exists in chronic pulmonary congestion. In normal subjects, Pryor and Page 1954 (239) showed that large intravenous infusions of saline produced a marked reduction of pulmonary compliance, but they did not measure pulmonary vascular pressures and further interstitial oedema may have occurred.

Bondurant, Hickam and Isley 1957 (39) induced pulmonary congestion by either compressing the lower limbs and abdomen with a pressure suit or by immersion up to the neck in water, whilst Ernsting 1958 (92) and Ting, Hong and Rahn 1960 (276) used negative pressure breathing as a means of increasing the blood content of the lungs. All these manoeuvres induced an approximately 50% reduction of the compliance of the lung. Since, however, these procedures also reduced the functional residual capacity below the resting value the intraoesophageal pressure probably bore little relation to the pleural pressure and no reliance can be placed upon the compliance values obtained in these circumstances (276). Thus the effect of pulmonary congestion upon compliance in man is uncertain. In all these studies, however, the blood content of the lung was increased above the normal resting value

whereas in positive pressure breathing the pulmonary blood volume is reduced below the resting level. The absence of a significant change of compliance in positive pressure breathing is in agreement with more recent animal studies. There is, however, a need for further experiments in man in order to determine the effects upon compliance of an increase of the pulmonary blood volume above the resting value.

Airway Resistance – The non-elastic component of the resistance to movement of the lungs is due partly to the resistance to the flow of gas through the airways and partly to frictional resistance in the lung tissues themselves. No direct method of measuring the viscous resistance of the lung tissue has yet been developed. Indirect measurements of tissue viscous resistance based upon the determination of airway resistance using the interruption technique (230) or by the use of gases of different viscosities and densities (222) have yielded widely varying values for the magnitude of this quantity. In 1956, however, Marshall and Du Bois (204) made simultaneous determinations of total non-elastic resistance and airway resistance using an intraoesophageal balloon to measure the former and a body plethysmograph (83) to measure the latter. They found that the tissue viscous resistance amounted to about one sixth of the total pulmonary non-elastic resistance. Thus by far the greater part of the non-elastic resistance to lung movement is due to the resistance to gas flow through the airways.

For a given experimental situation in the present investigation there was a consistent relationship between the non-elastic component of the change of intraoesophageal pressure and the simultaneous respiratory gas flow. This relationship was slightly alinear at the higher values of flow and other investigators (Mead and Whittenberger, 1953) (210) have treated the curve relating flow to pressure difference as a parabola. In view, however, of the relatively small degree of alinearity, it was considered that the non-elastic resistance at a single flow value could be used in order to simplify comparison between one experimental condition and another. Since the peak inspiratory flow in the majority of the conditions investigated was between 40 and 80 litre/min., the non-elastic resistance was calculated for a flow of 30 litre/min. The normal convention of expressing the non-elastic resistance as the non-elastic component of the mouth-intraoesophageal pressure change (cm H_2O) per unit of respiratory gas flow (1 litre per second) was followed. No measurable difference was found in any of the conditions studied between inspiratory and expiratory non-elastic resistance and the inspiratory and expiratory values have been used together in the calculation of the mean non-elastic resistance.

The mean value of 2.36 cm water per litre/sec. obtained for the non-elastic resistance in the four subjects at rest agrees closely with the mean value of 2.4 cm water per litre/sec. obtained by McIlroy, Eldridge and Stone 1956 (219) and the mean value of 2.29 cm water per litre/sec. obtained by Marshall 1957 (203). Marshall and Du Bois 1956 (204), however, obtained a considerably lower value (1.2 cm water per litre/sec.) for the total non-elastic resistance in the course of their measurements of the tissue viscous resistance, but their subjects were instructed to breathe rapidly and shallowly which reduced the airway resistance. Pressure breathing without respiratory counterpressure caused a marked reduction of the non-elastic resistance, the reduction being greater at a positive breathing pressure of 30 mmHg than at

15 mmHg (Table 4-7). When complete trunk counterpressure was used only a small and inconsistent decrease of non-elastic resistance occurred even at a pressure of 80 mmHg. Pressure breathing with counterpressure applied to the chest alone resulted in a marked decrease of non-elastic resistance, the reduction increasing as the breathing pressure was raised.

The change of non-elastic resistance produced by pressure breathing appeared to be related to the increase of lung volume induced by this manoeuvre rather than to the magnitude of the positive breathing pressure itself. Since in some of these experiments the increase of lung volume was measured simultaneously with the non-elastic resistance it was possible to determine the relationship between these two variables. In Fig. 4-22 the reciprocals of the values of the non-elastic resistance have been plotted against the corresponding changes of lung volume. For each subject there was an approximately linear relationship between these two quantities. The reciprocal of the non-elastic resistance was used for this purpose since Briscoe and Du Bois 1958 (48) demonstrated a linear relationship between airway conductance, the reciprocal of airway resistance, and lung volume in normal subjects. These investigators found that in man the airway conductance expressed as litre/sec. per cm of water increased 0.28 for each litre increase of lung volume over a wide range of lung volumes. In the present study the reciprocal of the total non-elastic resistance increased by a mean value of 0.20 (range 0.15 to 0.25) litre/sec. per cm of water for each litre increase of lung volume.

Although the reciprocal of the total non-elastic resistance is not exactly equivalent to airway conductance the results of Marshall and Du Bois' study of the relationship between total non-elastic and airway resistances already referred to showed that about five-sixths of the total non-elastic resistance is due to the resistance to gas flow through the airways. It is reasonable to infer, therefore, at least an approximately constant relationship between changes of total non-elastic resistance and of airway resistance. The fact that the tissue viscous resistance was not changed significantly when the airway resistance was increased twofold to threefold by the inhalation of a histamine aerosol (204) suggests, however, that this relationship is not always constant. The similarity of the relationship between the reciprocal of non-elastic resistance and the change of lung volume in pressure breathing on the one hand to the relationship between airway conductance and the change of lung volume in normal subjects breathing at various lung volumes on the other, lends strong support, however, to the contention that the reduction of non-elastic resistance induced by pressure breathing is due to an increase of airway conductance.

It is clearly very desirable that a more direct determination of airway resistance should be made in pressure breathing. The most satisfactory technique for the determination of airway resistance is the direct measurement of the mouth-alveolar pressure difference by means of the body plethysmograph (83). It is impossible to see, however, how this technique could be used in pressure breathing since it depends upon the free flow of gas between the lungs and the plethysmograph. Another method, which has been used in the past for the determination of airway resistance, is the recording of the change of pressure at the mouth when the air flow is suddenly interrupted

(230). As has been pointed out, however, by Marshall and Du Bois 1956 (204) the movement of the chest and lungs must be halted when interruption occurs and the energy which is being exerted against tissue resistance at the moment of interruption is transformed into pressure in the lung. Thus the interruption pressure measured is really the sum of the pressures exerted against both airway and tissue resistance. That this technique measures total non-elastic resistance has been shown experimentally by Mead and Whittenberger 1954 (211). A limited series of measurements using the interrupter technique were made in the present study. Results obtained for the pulmonary resistance both at rest and during pressure breathing were very similar to the values of the non-elastic resistance found in the corresponding experimental situation. The results given by the interrupter technique serve to confirm, therefore, the measurement of the total non-elastic resistance obtained with an intraoesophageal balloon.

Thus pressure breathing produces a reduction of airway resistance when there is a concomitant increase in the lung volume. There are several mechanisms by which the airway resistance could be reduced in these circumstances. The most obvious and the most important mechanism is an increase in airway diameter, caused by the distension of the lungs. The direct correlation between the change of non-elastic resistance and the increase of lung volume during pressure breathing supports this contention. The increase of airway diameter produced by distension of the lungs has been well documentated by anatomical, radiological and physiological studies. Thus Shepard, Campbell, Martin and Enns 1957 (267) and Birath 1959 (36) have demonstrated that the anatomical dead space increases directly with the functional residual capacity when the lung volume is voluntarily increased in normal subjects. The results of these investigations and of calculations by Briscoe and Du Bois 1958 (48) suggest that those airways which have the major part of the airway resistance of the lungs as a whole, are as distensible as the alveoli.

The measurements of the effect of pressure breathing upon the anatomical dead space presented in Chapter 5 add further support to the hypothesis that the reduction of airway resistance caused by this manoeuvre is due to an increase in the diameter and hence the volume of the resistance airways. The airway resistance can also be modified by reflex and humoral activity. The absence of any significant change of airway resistance when trunk counterpressure was employed suggests, however, that this mechanism is not important in pressure breathing. Finally, changes in airway calibre could be produced in pressure breathing by alteration in the vascularity of the bronchial mucosa. The blood content of the lungs and presumably the degree of filling of the vascular bed of the bronchial mucosa are reduced during pressure breathing. Again the absence of a significant reduction of airway resistance during pressure breathing with trunk counterpressure suggests that this mechanism does not contribute to the reduction of airway resistance found when lung distension is produced by pressure breathing.

Respiratory Work – The modern analysis of the mechanical work performed by the respiratory muscles was first proposed in detail by Otis, Fenn and Rahn 1950 (229). They measured the total mechanical work of breathing by passively ventilating the subject in a respirator. With the advent of the

indirect measurement of intrapleural pressure by recording the pressure in the lower oesophagus (51,) (78) it became possible to determine the work done during breathing upon the lungs and the gas within them under a wide variety of circumstances, both in health and disease (221,) (218). When the transpulmonary pressure (mouth-intraoesophageal pressure) is plotted against the corresponding change of lung volume during a complete respiratory cycle, a closed loop is produced (Fig. 4–23). Area on such a pressure-volume diagram is the product of pressure and volume change and this represents work. Inspiratory work is represented by the inspiratory part of the pressure volume loop (A.I.B., Fig. 4–23). The elastic component of the lung forces opposing inspiration is represented by the straight line AB which joins the points of zero flow on the pressure volume loop. Thus the work performed during inspiration against the elastic forces of the lungs is represented by the area ABC (Fig. 4–23).

The inspiratory work performed against the non-elastic resistance to respiration is represented by the area AIB. The work done on the lungs and the gas within them during inspiration is represented, therefore, by the area AIBC. The work expended against the non-elastic resistance during expiration is similarly represented by the area BEA. During pressure breathing the transpulmonary pressure at the end of expiration was greater than that which existed during normal breathing because of the lung distension induced by the pressure breathing. Thus in pressure breathing additional inspiratory work was done on the lungs in sustaining the initial distension whilst the lung volume was further increased during inspiration. This additional inspiratory work against the elastic recoil of the lungs is represented by the area of the rectangle ACDH where DH is the transpulmonary pressure at the end of a quiet expiration at rest. Using these definitions the work performed upon the lungs and in moving the gas within them during the respiratory cycle was calculated for the various conditions investigated. The total inspiratory trans-pulmonary work, its components and the total non-elastic work performed on the lungs were measured for each of the transpulmonary pressure-tidal volume diagrams constructed in the course of the measurement of the non-elastic pulmonary resistance. The 6–12 values obtained for each of these quantities for each subject in each experimental situation were averaged and the results are presented in Table 4–10.

In an analysis of the significance of the results of these calculations of the work done on the lungs during respiration the limitations of the trans-pulmonary pressure-respiratory volume diagram become apparent. The respiratory work calculated from these measurements refers only to the work done on the lungs and in moving the gas through the airways during the respiratory cycle. The values of inspiratory work calculated in this manner give no direct indication of the muscle forces expended in ventilating the lungs. The magnitude at any instant of the intrapleural pressure measured relative to that of the environment gives the net force exerted upon the lungs by the action of the respiratory muscles, the elastic and viscous forces of the chest wall and any counterpressure applied to the external surface of the trunk. Thus although pressure breathing without counterpressure at positive pressures of 15 and 30 mmHg markedly increased the inspiratory trans-pulmonary work, the work performed by the inspiratory muscles under these
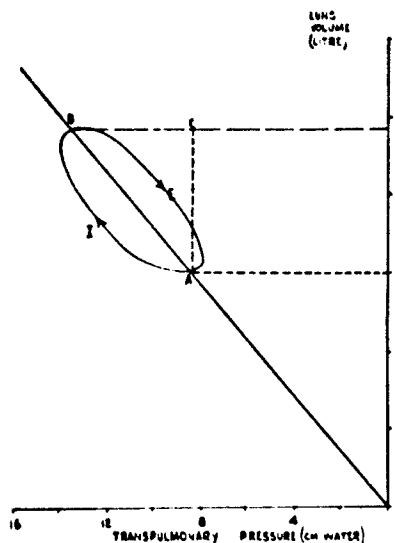
FIG. 4-23 The relationship between lung volume and transpulmonary pressure during a complete respiratory cycle in subject C whilst pressure breathing without respiratory counterpressure at 30 mmHg (closed loop). The transpulmonary pressure is shown as the change from the value which existed at the end of a quiet expiration at rest. The diagonal through the origin and points A and B represents the compliance of the lung
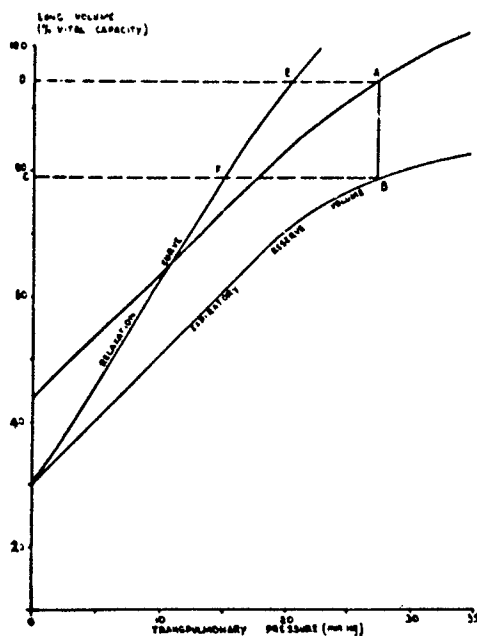
FIG. 4-24 The expiratory reserve volume and tidal volume during pressure breathing without respiratory counterpressure. The relaxation pressure volume curve of the respiratory apparatus is also shown

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conditions was nil (243). Provided, however, that these limitations are recognized, the calculation of the work done on the lungs and in moving gas through the airways during pressure breathing is of value.

Inspection of Table 4.10 reveals that the total work expended on the lungs and in moving gas through the airways during inspiration was greatly increased by pressure breathing in the absence of respiratory counterpressure. Although this increase was reduced by the application of counterpressure to the chest, pressure breathing at 80 mmHg with a pressure waistcoat increased the work sevenfold. The application of counterpressure to the whole of the trunk greatly reduced the increase in the inspiratory work induced by pressure breathing so that even at a positive breathing pressure of 80 mmHg the total work performed on the lungs and the gas within them during inspiration was only just over twice that expended at rest. The increase of inspiratory work induced by pressure breathing was due to increases in both the elastic and non-elastic components, but most of the increase was contributed by the elastic fraction. The work done against the elastic forces of the lungs has been subdivided further into that expended in increasing the lung volume during inspiration and that required to maintain the existing end-expiratory tension (Table 4.10).

The elastic work done in increasing the lung volume was increased by a third by pressure breathing without counterpressure at 30 mmHg. The application of counterpressure to the trunk prevented most of this increase whilst the greatest increase of this component of the elastic work occurred during pressure breathing at 80 mmHg with the pressure breathing waistcoat. Since pressure breathing only caused a small decrease of lung compliance these increases were primarily a function of the changes of tidal volume induced by this manoeuvre. In the resting state the lung and thorax returned to the relaxed position so that no work was expended in producing a maintained distension of the lung. In all the pressure breathing situations, however, there was a maintained distension of the lungs. The work done against this component of the elastic forces during inspiration was determined by the magnitude of the increase of the transpulmonary pressure induced by the pressure breathing and the tidal volume. Thus the greatest increase of this component was produced by pressure breathing with chest counterpressure alone at a positive pressure of 80 mmHg (Table 4.10).

The work expended in overcoming the non-elastic resistance of the lungs and in moving the gas in the airways was only moderately increased by pressure breathing. The decrease in non-elastic resistance which occurred during pressure breathing when there was distension of the lungs did not compensate completely therefore for the associated increase of the inspiratory flow. The greatest increase of non-elastic inspiratory work occurred at a positive breathing pressure of 80 mmHg with chest counterpressure. Pressure breathing caused a relatively small change of the expiratory non-elastic work (Table 4.10), because the expiratory flow was not greatly increased and the increase which did occur was partially compensated by the decrease of the non-elastic resistance of the lungs. In all the situations studied no active work was required to overcome the non-elastic pulmonary resistance to expiration since this was supplied by the elastic recoil of the lungs.

The relationship between the work performed by the respiratory muscles

TABLE 4-10

MEAN VALUES FOR THE WORK DONE ON THE LUNG
AND ITS CONTENTS CALCULATED FROM THE RESULTS
OF DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Positive breathing pressure (mmHg)	WORK PER BREATH (kg-cm) INSPIRATION				EXPIRATION
	Moving Lung	Elastic Sustaining Distension	Non-Elastic	Total	Non-Elastic
Rest	1.37	0.0	0.81	2.18	0.60
Pressure breathing					
(a) No counterpressure					
15	1.60	3.20	0.91	5.21	0.62
30	2.03	7.20	1.11	10.34	0.78
(b) Chest counterpressure					
50	2.09	5.21	0.95	8.25	0.71
80	3.30	10.00	1.76	15.06	0.77
(c) Trunk counterpressure					
50	1.51	1.10	0.85	3.46	0.65
80	1.60	2.00	0.93	4.53	0.71

TABLE 4-11

MEAN VALUES FOR THE WORK DONE ON THE WHOLE
RESPIRATORY APPARATUS DURING EXPIRATION CALCULATED
FROM THE RESULTS OF DUPLICATE EXPERIMENTS ON FOUR
SUBJECTS

Positive breathing pressure (mmHg)	Expiratory work per breath (kg-cm)		
	Elastic	Non-elastic	Total
Pressure breathing with no counterpressure			
15	5.9	1.2	7.1
20	8.8	.4	10.2
30	13.7	1.6	15.3

and the actual work done on the lungs and in moving the gas through the airways, is a complex one. The disturbances of lung mechanics induced by pressure breathing made a quantitative assessment of the muscular effort expended in breathing extremely difficult and the problem is made even more difficult by the application of counterpressure. At positive breathing pressures in excess of about 10 mmHg the pressure applied at the mouth was more than adequate to overcome both the elastic and non-elastic forces of the lungs and the thoracic cage opposing inspiration. Thus during pressure breathing without counterpressure at positive pressures greater than 10 mmHg no mechanical work was performed by the inspiratory muscles on the lungs and the gas within them. There was, however, under these conditions active contraction of the expiratory muscles throughout inspiration. Expiration in these circumstances involved active contraction of the expiratory muscles since the elastic recoil of the lungs and thoracic cage was inadequate to overcome the pressure applied at the mouth and the non-elastic resistance of the respiratory system to expiration. The expiratory muscles were assisted during expiration by the elastic recoil of the complete respiratory system.

In addition to the work involved in displacing the tidal volume from the respiratory tract against the applied breathing pressure, the expiratory muscles also expended energy in limiting the distension of the lungs and thoracic cage. A serious limitation in the present context to the use of the conventional definition of mechanical work as a force acting through a distance is that it takes no account of the energy expended during the isometric contraction of a muscle. During pressure breathing without counterpressure a large proportion of the total energy expended in breathing was consumed by the isometric contraction of the expiratory muscles which prevented the gross distension of the lungs which would otherwise occur. It is not possible, therefore, to estimate this fraction of the work done by the expiratory muscles from the pressure-volume loops obtained from records of mouth-intraoesophageal pressure difference and tidal volume.

The work done by the expiratory muscles in expelling the tidal volume against the pressure applied at the mouth during pressure breathing can, however, be assessed from the position of the tidal air band relative to the relaxation pressure volume curve for the total respiratory apparatus (243). The results of the measurements of these quantities obtained during pressure breathing without counterpressure are presented in Fig. 4.24. The elastic work performed in expelling the tidal volume AB whilst pressure breathing at a positive pressure of 28 mmHg is represented by the area ABCD. Part of this work, represented by the area CDEF, was done by the elastic potential energy stored in the thoracic wall and the lungs so that the net work done by the active contraction of the expiratory muscles is shown by the difference between these two values, i.e. the area ABFE. This work has been estimated for each level of pressure breathing without counterpressure studied and the results of these calculations are presented in Table 4.11. The work expended against the non-elastic resistance of the lungs during expiration at these various levels of pressure breathing has been estimated from the results presented in Table 4.10. Further, if it is assumed that the frictional resistance to respiration offered by the chest wall and abdomen is equal to the non-elastic resistance of the lungs (229) it is possible to estimate the total work performed

during expiration against the non-elastic resistance of the whole respiratory apparatus. The results of these calculations are also presented in Table 4.11. The work done by the expiratory muscles in reducing the volume of the lungs and chest and overcoming the non-elastic resistance of the respiratory apparatus was very considerable. These values may be compared with the total work expended during respiration at rest which amounted to 4.0 kg.cm/breath.

The work performed by the respiratory muscles when counterpressure was applied to the trunk during pressure breathing could not be assessed quantitatively. If the counterpressure applied to the surface of the trunk exactly equalled the pressure applied to the respiratory tract then the work of breathing would not be increased above that expended at the same level of ventilation at rest. The present investigation has demonstrated, however, that the counterpressure applied by the jerkin was not perfect. There was some distension of the lungs and thorax equivalent to pressure breathing without counterpressure at a positive pressure of 3–4 mmHg. There was also an increase of the tidal volume. The energy expended by the abdominal muscles during pressure breathing with chest counterpressure alone was certainly greatly increased, although the magnitude of this increase could not be assessed quantitatively.

It is of interest to consider briefly the effect of the increased respiratory work associated with pressure breathing upon the metabolic oxygen consumption. Several investigators have measured the additional oxygen consumption associated with an increase of ventilation produced either by voluntary hyperventilation or by adding carbon dioxide to the inspired gas 189) 226) (58). At rest the oxygen consumption of the respiratory muscles amounts to between 2 and 8 ml (S.T.P.) per minute. The efficiency of the respiratory muscles can be calculated by measuring the mechanical work performed and dividing it by the energy equivalent of the oxygen consumption of the respiratory muscles. Otis, Fenn and Rahn 1950 (229) estimated the efficiency to be 3 to 7% whilst Campbell, Westlake and Cherniack 1957 (58) obtained values of between 5 and 10% in three young normal subjects. Thus if the efficiency of the respiratory muscles was unchanged during pressure breathing, the oxygen cost of pressure breathing at a positive pressure of 30 mmH without counterpressure would be of the order of 12–25 ml per minute. The increase of oxygen consumption over that at rest induced by a positive breathing pressure of 30 mmHg would be expected therefore to be approximately 10–20 ml per minute. The mean value actually obtained in the present study (Chapter 5) amounted to 21 ml per minute. There was, therefore, good agreement between the measured and predicted values for the increase of metabolic oxygen consumption induced by pressure breathing. The predicted increase in the metabolic oxygen consumption occasioned by pressure breathing with trunk counterpressure at a positive pressure of 80 mmHg would be considerably less than 10–20 ml per minute. In fact pressure breathing at 80 mmHg with trunk counterpressure did not cause a significant change in the total oxygen consumption from that measured at rest.

Intragastric Pressure – The continuous measurement of the pressure within the stomach has been used by several investigators (84) (215) (55, 56) (3), in an attempt to analyze the behaviour of the diaphragm and abdominal muscles during normal breathing. The relationship between the pressure

recorded by an intragastric balloon and that at the abdominal surface of the diaphragm is determined by the tone of the gastric musculature and the hydrostatic pressure exerted by the abdominal contents lying between the diaphragm and the balloon in the stomach. Agostoni and Rahn 1960 (3) found that in the relaxed subject at all lung volumes greater than 20"„ of the vital capacity the pressure recorded by the intragastric balloon was about 11 cm water greater than the simultaneously determined intraoesophageal pressure. These authors assumed that the diaphragm was relaxed in this condition and that since therefore the pressures on the two surfaces of the diaphragm were equal, the intragastric pressure was some 11 cm water greater than that at the abdominal surface of the diaphragm. In the present study the intragastric pressure at the end of expiration was found to exceed the corresponding value of the intraoesophageal pressure by a mean value of 10.4 cm water (S.E. ± 0.8 cm water). These results also agree closely with those obtained by Duomarco and Rimini 1947 (84).

The general behaviour of the intragastric pressure found during breathing at rest agreed with the studies performed by Duomarco and Rimini 1947 (84) and Campbell and Green 1953 (55, 56). Campbell and Green 1955 (57) demonstrated that even in the erect posture there may be no electrical activity in the muscles of the anterior abdominal wall. When these muscles exhibited a respiratory rhythm it took the form of a decrease of activity during inspiration and an increase during expiration. Thus the rise of abdominal pressure during inspiration was not due to contraction of the abdominal muscles. It was the result of the contraction and the consequent descent of the diaphragm. The marked increase in the difference between the pressures in the oesophagus and stomach which occurred during inspiration was also evidence of (Fig. 4–14) active contraction of the diaphragm. The reduction of the intragastric and transdiaphragmatic pressures which occurred during expiration at rest reflected relaxation of the diaphragm. By recording the electrical activity of the diaphragm with intraoesophageal electrodes Agostini, Sant, Ambrogio and Del Portillo-Carrasco 1960 (4) have confirmed this pattern of activity of the diaphragm during quiet breathing.

It is possible to calculate the pressure at the abdominal surface of the dia-phragm relative to that of the environment (the abdominal pressure) from the recorded mouth-intragastric pressure difference, assuming that the intragastric pressure was 10.4 cm water greater than the abdominal pressure. During pressure breathing without respiratory counterpressure at a positive pressure of 15 mmHg this pressure at the end of expiration was 8 mmHg, whilst the corresponding value at a positive breathing pressure of 30 mmHg was 19 mmHg. These values of the abdominal pressure are a measure of the tension created by the muscles of the abdominal wall during pressure breath-ing. The increase of the transdiaphragmatic pressure during inspiration which occurred at rest was reduced by a positive breathing pressure of 15 mmHg and almost eliminated by pressure breathing at 30 mmHg (Fig. 4–14). Thus the contribution of the active contraction of the diaphragm to inspira-tion was progressively reduced as the breathing pressure was increased. At the higher pressure inspiration was almost solely due to partial relaxation of the intercostal and abdominal muscles.

It is of interest that active contraction of the diaphragm occurred during

pressure breathing at 15 mmHg since relaxation of the expiratory muscles of itself could produce inspiration at this breathing pressure. Similar results at positive breathing pressures of up to 22 mmHg have recently been obtained by Agostoni 1962 (2). He also recorded the electrical activity of the diaphragm during pressure breathing and found that there was activity during inspiration even at a positive breathing pressure of 22 mmHg. The appearance of an increase of the gastric pressure during expiration with the induction of pressure breathing is evidence of an increase in the tension of the abdominal muscles during this phase of the respiratory cycle. The transdiaphragmatic pressure was virtually zero throughout expiration during pressure breathing at 30 mmHg. At this pressure, therefore, the diaphragm was acting in a passive manner during most of the respiratory cycle. The function of the diaphragm during pressure breathing with chest counterpressure was similar to that seen when no counterpressure was employed. The tension created by the contraction of the abdominal muscles during pressure breathing at positive pressures of 50 and 80 mmHg was much greater, however, as is reflected by the end-expiratory abdominal pressure which amounted to 41 and 70 mmHg respectively.

The application of counterpressure to the whole trunk during pressure breathing raised the intra-abdominal pressure by virtually the pressure applied to the respiratory tract. Thus the end-expiratory abdominal pressure during pressure breathing at 80 mmHg was 76 mmHg. The pattern of the intragastric and transdiaphragmatic pressure changes during the respiratory cycle, when breathing with trunk counterpressure were almost indistinguishable from those recorded at rest (Fig. 4–14). Thus when full trunk counterpressure is used the diaphragm plays an important part in the production of inspiration even at a positive breathing pressure of 80 mmHg.

SUMMARY

This experimental investigation of the effects of pressure breathing upon the mechanics of respiration confirmed that the primary disturbance induced by this manoeuvre was pulmonary distension. The elastic recoil of the lungs and of the chest and abdominal walls opposed the distending force and, in addition, at positive breathing pressures in excess of 10 mmHg the expiratory muscles were active throughout the respiratory cycle. In spite of the increase of tension of the expiratory muscles the lungs were almost fully distended by a positive breathing pressure of 30 mmHg. The compliance of the lungs was virtually unaffected by pressure breathing, but the non-elastic pulmonary resistance was markedly reduced. The work done upon the lungs and in moving the gas within them was however increased during pressure breathing because of the concomitant increase of the tidal volume. Pressure breathing at positive pressures in excess of 8–10 mmHg reversed the active phase of respiration so that there was an increase of tone in the expiratory muscles during expiration and inspiration occurred by partial relaxation of these muscles. There was evidence, however, that even at a breathing pressure of 15 mmHg there was active contraction of the diaphragm during inspiration. It was concluded that the practical limit to pressure breathing without the use of external respiratory counterpressure was a positive breathing pressure of 30 mmHg.

The application of counterpressure to the thorax by means of a pressure waistcoat inflated to the same positive pressure as that applied to the respiratory tract reduced the disturbances induced by simple pressure breathing alone. A positive breathing pressure of about 80 mmHg was required to fully distend the lungs when chest counterpressure was employed. In these circumstances, however, the abdominal muscles were actively contracted in order to maintain a pressure of the order of 70 mmHg at the abdominal surface of the diaphragm throughout the respiratory cycle. Breathing in this state was extremely tiring and there was a large increase in the tidal volume. The practical limit to pressure with counterpressure to the chest alone, it was concluded, was a positive breathing pressure of the order of 30-40 mmHg.

A study of the mechanics of respiration during pressure breathing when counterpressure was applied to the whole of the trunk by means of a pressure jerkin revealed that with this garment even pressure breathing at a positive pressure of 80 mmHg induced only very minor disturbances. The counterpressure afforded by the pressure jerkin fell only slightly short of the ideal. Pressure breathing at 80 mmHg induced a small increase of lung volume and the increase of abdominal pressure was some 3-4 mmHg less than the pressure applied to the respiratory tract and in the bladder of the garment. Essentially, the behaviour of the respiratory muscles during pressure breathing with counterpressure applied by means of this garment was the same as at rest. The practical limit to pressure breathing with trunk counterpressure afforded by the pressure jerkin is not defined by respiratory factors since it effectively prevents any serious disturbance of the mechanics of respiration during pressure breathing.

PULMONARY GAS EXCHANGE IN PRESSURE BREATHING

INTRODUCTION

Since pressure breathing is used to maintain the oxygenation of the arterial blood at greatly reduced environmental pressures the effects of this manoeuvre upon the gaseous exchange between the inspired gas and the blood flowing through the lungs is of great interest. Pressure breathing generally induces an increase of the pulmonary ventilation even when full trunk counterpressure is employed. It is important to determine, however, the actual change of alveolar ventilation produced by pressure breathing, since this, amongst other factors, controls the actual gaseous exchange with the blood flowing through the pulmonary capillaries. Pressure breathing has very marked effects upon the circulation, thus the regional distribution of the pulmonary capillary blood flow and hence the oxygenation of the arterial blood could be affected by this manoeuvre. It is also conceivable that this procedure could affect the uptake of oxygen from the alveolar gas by the blood flowing through the pulmonary capillaries.

These various aspects of pulmonary gaseous exchange were studied during pressure breathing with the full trunk counterpressure afforded by the pressure jerkin. The maximum positive breathing pressure employed was 80 mmHg and in many experiments positive pressures of 30 and 60 mmHg were used. Although pressure breathing with trunk counterpressure was the primary interest, some of the investigations were repeated during pressure breathing without counterpressure. However, it was only possible to find two subjects who were able to maintain a regular breathing pattern when exposed to pressure breathing at a positive pressure of 30 mmHg and these were investigated in the limited study of pressure breathing without counterpressure. In most of the experiments the same four subjects were studied as were used in the experiments reported in the previous chapter.

EXPERIMENTAL INVESTIGATION
Pressure Breathing at Ground Level
Pulmonary Ventilation and Overall Gas Exchange – The pulmonary ventilation, oxygen consumption and carbon dioxide production were measured in the resting subject and during pressure breathing by the open circuit method at ground level. The subject, wearing a pressure jerkin, was seated in an ejection seat within the decompression chamber. The mouthpiece of the modified pressure helmet worn by the subject was connected directly to a wide bore T piece (2.5 cm I.D.) the other two arms of which were connected to the exterior of the chamber by means of smooth-bore hose. A low resistance non-return valve was fitted in each of these two hoses. A recording Tissot spirometer was placed outside the decompression chamber

TABLE 5-1

THE EFFECT OF PRESSURE BREATHING AT 30 mmHG WITHOUT TRUNK COUNTERPRESSURE UPON PULMONARY VENTILATION AND GAS EXCHANGE – INDIVIDUAL VALUES OBTAINED IN QUADRUPLICATED EXPERIMENTS ON TWO SUBJECTS

Subject	B				C			
Pulmonary Ventilation (l. min. B.T.P.S.)	1	2	3	4	1	2	3	4
Control[1]	7.50	7.42	7.31	7.62	7.08	6.98	6.83	7.12
Pressure breathing[2]	10.23	10.09	11.56	10.50	9.28	10.01	10.85	10.92
Recovery[3]	6.32	6.79	5.91	6.21	6.05	5.93	5.76	6.55
Oxygen uptake (ml. min. S.T.P.)								
Control	280	285	271	283	260	269	270	265
Pressure breathing	309	290	283	318	279	276	295	311
Recovery	273	284	281	278	263	255	280	267
Carbon dioxide output (ml min. S.T.P.)								
Control	226	236	214	229	213	223	218	212
Pressure breathing	294	264	280	296	259	273	280	278
Recovery	186	202	205	192	182	171	176	163
Respiratory exchange ratio								
Control	0.81	0.83	0.79	0.81	0.82	0.83	0.81	0.80
Pressure breathing	0.95	0.91	0.99	0.93	0.93	0.99	0.95	0.90
Recovery	0.68	0.71	0.73	0.69	0.69	0.67	0.63	0.61

[1] Expired gas collected for 5 min. before pressure breathing
[2] Expired gas collected over 4th to 6th min. of pressure breathing
[3] Expired gas collected for 5 min. starting 1 min. after cessation of pressure breathing

TABLE 5-2

THE EFFECT OF PRESSURE BREATHING AT 60 mmHG WITH TRUNK COUNTERPRESSURE UPON PULMONARY VENTILATION AND GAS EXCHANGE – INDIVIDUAL VALUES OBTAINED IN DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Subject	A		B		C		D	
Pulmonary Ventilation (l. min. B.T.P.S.)	1	2	1	2	1	2	1	2
Control[1]	7.15	7.86	6.85	7.31	6.90	7.10	6.75	7.06
Pressure breathing[2]	10.16	10.50	9.61	9.06	8.45	9.63	9.96	10.37
Recovery[3]	5.92	6.13	5.54	6.03	5.92	6.15	5.95	6.03
Oxygen uptake (ml min. S.T.P.)								
Control	279	285	290	275	275	290	261	258
Pressure breathing	284	278	301	272	269	286	263	255
Recovery	276	293	291	290	261	288	262	252
Carbon dioxide output (ml min. S.T.P.)								
Control	226	234	240	222	230	235	220	207
Pressure breathing	278	264	298	280	272	306	266	270
Recovery	193	211	218	157	170	199	191	191
Respiratory exchange ratio								
Control	0.81	0.82	0.83	0.81	0.84	0.81	0.82	0.81
Pressure breathing	0.98	0.95	0.99	1.03	1.01	1.07	1.03	1.07
Recovery	0.70	0.72	0.75	0.68	0.65	0.69	0.73	0.76

[1] Expired gas collected for 5 min. before pressure breathing
[2] Expired gas collected over 2nd to 4th min. of pressure breathing
[3] Expired gas collected for 5 min. starting 1 min. after cessation of pressure breathing

TABLE 5-3

THE EFFECT OF PRESSURE BREATHING AT 80 mmHG WITH TRUNK COUNTERPRESSURE UPON PULMONARY VENTILATION AND GAS EXCHANGE – INDIVIDUAL VALUES OBTAINED IN DUPLICATE EXPERIMENTS ON FOUR SUBJECTS

Subject	A		B		C		D	
Pulmonary Ventilation (1/min. B.T.P.S.)	1	2	1	2	1	2	1	2
Control[1]	8.01	7.55	7.52	7.40	7.30	7.55	7.01	7.32
Pressure breathing[2]	10.15	10.58	10.55	10.01	10.02	11.11	10.35	10.31
Recovery[3]	6.51	7.02	6.45	6.58	6.15	5.65	5.55	5.62
Oxygen uptake (ml/min. S.T.P.)								
Control	295	300	285	275	270	290	255	268
Pressure breathing	290	305	282	274	278	281	261	263
Recovery	293	290	278	280	280	283	260	265
Carbon dioxide output (ml min. S.T.P.)								
Control	246	245	232	228	226	238	212	224
Pressure breathing	278	299	290	274	295	306	248	263
Recovery	228	224	209	216	204	212	187	210
Respiratory exchange ratio								
Control	0.83	0.82	0.81	0.83	0.84	0.82	0.83	0.85
Pressure breathing	0.96	0.98	1.03	1.00	1.06	1.09	0.95	1.00
Recovery	0.78	0.77	0.75	0.77	0.73	0.75	0.72	0.79

[1] Expired gas collected for 5 min. before pressure breathing
[2] Expired gas collected over 3rd and 4th min. of pressure breathing
[3] Expired gas collected for 5 min. starting 1 min. after cessation of pressure breathing

TABLE 5-4

THE EFFECT OF PRESSURE BREATHING UPON PULMONARY VENTILATION AND GAS EXCHANGE – MEANS OF CHANGES OBTAINED EXPERIMENTALLY

Pressure breathing condition	Mean (± S.E.) of individual changes from corresponding control values					
	Without counterpressure 30 mmHg		With trunk counterpressure			
			60 mmHg		80 mmHg	
Pulmonary ventilation (1/min. B.T.P.S.)						
Pressure breathing	+3.20	±0.26[1]	+2.58	±0.23[1]	+2.80	±0.14[1]
Recovery	−1.04	±0.11[1]	−1.29	±0.17[1]	−1.27	±0.17[1]
Oxygen uptake (ml/min. S.T.P.)						
Pressure breathing	+21.0	±4.3[2]	0.0	±2.4	+0.3	±2.2
Recovery	−0.3	±2.9	−0.1	±3.1	−1.1	±2.5
Carbon dioxide output (ml/min. S.T.P.)						
Pressure breathing	+56.6	±5.0[1]	+53.8	±4.3[1]	+50.3	±5.1[1]
Recovery	−35.5	±4.7[1]	−30.5	±3.3[1]	−20.1	±1.8[1]
Respiratory exchange ratio						
Pressure breathing	+0.13	±0.01[1]	+0.18	±0.02[1]	+0.18	±0.02[1]
Recovery	−0.15	±0.01[1]	−0.10	±0.01[1]	−0.07	±0.01[1]

[1] $P < 0.001$
[2] $0.001 < P < 0.01$

and connected through a two-way tap to the hose through which the subject inspired gas. Before each experimental period the spirometer was filled with air and the two-way tap turned so that the subject inspired air from it. When necessary the spirometer was refilled with room air. The hose carrying the expired gas was fitted with a two-way tap outside the decompression chamber so that the expired gas could be led into a Douglas bag.

The subject wearing the helmet fitted with the mouthpiece, rested in the quiet for fifteen minutes before a timed five minute collection of expired gas was made. Pressure breathing at the desired level was induced after the completion of the resting collection and a further collection of expired gas was made during the second or third and subsequent minutes of pressure breathing. The duration of the collection period during pressure breathing was varied with the magnitude of the positive breathing pressure. The pressure breathing period was followed by a third collection of expired gas which was made for five minutes, starting one minute after the cessation of pressure breathing. In all the experiments at least two hours elapsed between the subject's last meal and the start of the experiment. The volume of gas collected in each bag was measured with a water gas meter and the composition of the mixed expired gas determined by duplicate analyses by the Haldane technique. Results – Duplicate experiments were performed with each of the four subjects whilst pressure breathing with trunk counterpressure at positive pressures of 60 and 80 mmHg. Four periods of pressure breathing at 30 mmHg without counterpressure were studied in two subjects. The record of the inspiratory minute volume obtained by means of the Tissot spirometer in each experiment was measured and the volume of air inspired in each minute together with the corresponding number of breaths calculated. The mean values of the pulmonary ventilation in the experiments performed on each subject are presented in Figs. 5–1, 5–2 and 5–3. In all the four subjects pressure breathing caused an increase of pulmonary ventilation which was greatest in the first minute of exposure. The respiratory frequency was only slightly increased during pressure breathing.

The pulmonary ventilation, oxygen consumption, carbon dioxide production and respiratory exchange ratio were calculated from the volume and composition of each collected sample of expired gas. The results of these calculations for each of the three conditions of pressure breathing investigated are presented in Tables 5–1 to 5–3. The change of each parameter observed during pressure breathing and the subsequent recovery period relative to the corresponding control value has been calculated and the mean changes and the corresponding standard errors for each of the environmental conditions are presented in Table 5–4. Pressure breathing without counterpressure at a positive pressure of 30 mmHg caused a consistent increase of the pulmonary ventilation, oxygen consumption, carbon dioxide output and respiratory exchange ratio (Table 5–4). The increase of oxygen uptake was statistically significant ($0.001 < P < 0.01$). Pressure breathing with trunk counterpressure also induced an increase of pulmonary ventilation in all the subjects. There was, however, no consistent change of oxygen consumption and in the group as a whole pressure breathing did not cause a significant alteration of oxygen uptake. The output of carbon dioxide was however significantly raised. The increase of the carbon dioxide output without a concomitant
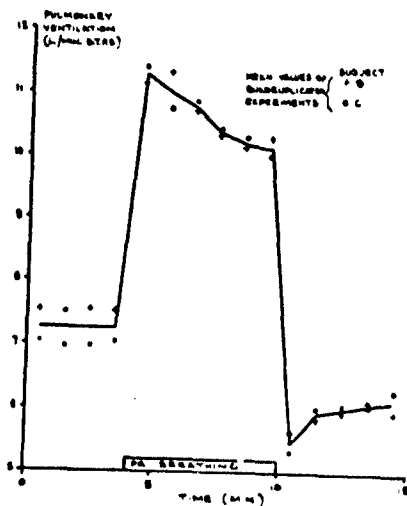
Fig. 5-1 The effect of pressure breathing at a positive breathing pressure of 30 mmHg without respiratory counterpressure upon the pulmonary ventilation
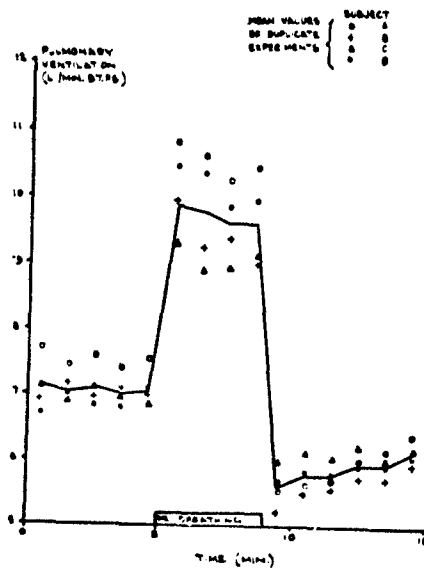
Fig. 5-2 The effect of pressure breathing at a positive breathing pressure of 60 mmHg with trunk counterpressure upon the pulmonary ventilation
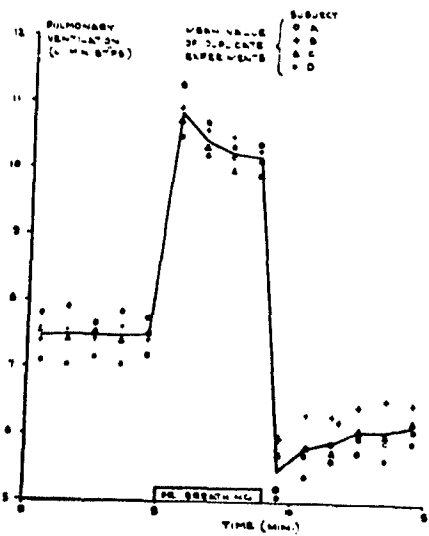
Fig. 5-3 The effect of pressure breathing at a positive breathing pressure of 80 mmHg with trunk counterpressure upon the pulmonary ventilation

change of oxygen consumption was reflected by the rise of the respiratory exchange ratio.

The Distribution of Inspired Gas – The uniformity of the distribution of the inspired gas within the lungs was investigated at ground level by following the concentration of nitrogen in the expired gas when the inspired gas was changed from air to oxygen. The subject was seated within the decompression chamber and wore a pressure jerkin and the modified pressure helmet fitted with a mouthpiece. The sampling needle valve of the Lundin Akesson nitrogen meter was attached to the mouthpiece so that it lay 3 cm from the lips. Beyond the sampling valve of the nitrogen meter the mouthpiece was attached to a small dead space valve box. The dead space of the apparatus to the sampling valve of the nitrogen meter amounted to 12 ml, whilst the total dead space rebreathed on inspiration was 35 ml. A two-way tap was attached directly to the inlet tube of the valve box. The remaining two arms of this tap were connected by separate smooth-bore hoses (2.5 cm I.D.) to the exterior of the decompression chamber. One tube allowed air to be inspired from outside the decompression chamber whilst the other hose was connected to a 100 litre Douglas bag placed outside the decompression chamber. The latter was filled with cylinder oxygen (99.5"₀ oxygen) and before each experiment the hose up to the two-way tap at the inlet of the valve box was thoroughly washed through with oxygen. The outlet pipe of the valve box was connected by smooth-bore hose to the Tissot spirometer which was placed outside the decompression chamber. A two-way tap was placed outside the decompression chamber upstream of the spirometer so that the subject's expired gas could be directed either to the exterior or into the spirometer. In some of the preliminary experiments a heated Fleisch flow meter was inserted in the expiratory gas stream at the outlet of the valve box. The pressure difference created by expiratory flow was recorded by means of a capacitance pressure transducer and an appropriate amplifier. The output of the nitrogen meter was recorded on a direct ink writer. The amplification of the instrument was increased when the nitrogen concentration fell below 20"₀ to give an increased sensitivity at the low nitrogen concentrations. The output of the nitrogen meter was calibrated against oxygen-nitrogen mixtures of known composition before and after each experiment.

After a preliminary rest period of at least five minutes during which the subject breathed air, the expired gas was directed into the spirometer and one minute later the inlet tap was turned during an expiration so that the inspired gas was changed to oxygen. The expired nitrogen concentration was recorded and the expired gas collected in the spirometer until the expired nitrogen concentration fell below 1.5"₀. The subject was then returned to breathing air and at least ten minutes elapsed before a further experiment was performed. Nitrogen washout curves were obtained in this manner with the subject at rest and during the second and subsequent minutes of pressure breathing. Positive breathing pressures of 30 and 60 mmHg were used when trunk counterpressure was worn whilst a positive breathing pressure of 20 mmHg was investigated when no counterpressure was used. In a short series of experiments the instantaneous nitrogen concentration and the expiratory flow were recorded simultaneously on a bromide paper recorder during oxygen breathing both at rest and during pressure breathing.

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Results – The experiments in which the nitrogen concentration and expiratory flow were recorded simultaneously demonstrated that there was a definite plateau of nitrogen concentration in the later part of expiration under all the conditions used in this investigation. The value of the nitrogen concentration at the end of expiration was used in the subsequent analysis of the nitrogen washout curve. Since the output of the nitrogen meter was calibrated with dry gas and the expired gas sampled by the meter had a water vapour pressure of 47 mmHg a correction was applied to the values of nitrogen concentration measured from the output of the nitrogen meter.

Inspection of the spirometer records showed that in the majority of the experiments the tidal volume varied only slightly during the nitrogen washout period. In some experiments, however, particularly during pressure breathing without counterpressure, there were fairly large fluctuations of tidal volume. The effect of these fluctuations of ventilation upon the end-expiratory nitrogen concentration was minimized by plotting the end-expiratory nitrogen concentration for each breath after the beginning of oxygen breathing against the cumulative expiratory volume. The mean tidal volume over the period of the nitrogen washout was then calculated and the values of end-expiratory nitrogen concentration corresponding to breaths of constant volume determined from the original curve. These corrected values of the end-expiratory nitrogen concentration were used in the detailed analysis of the nitrogen washout curve.

The results of each nitrogen washout were analyzed by plotting the logarithm of the corrected end-expiratory nitrogen concentration for a given breath against the number of breaths taken from the start of oxygen breathing using semi-logarithmic paper. A typical curve obtained by this procedure is presented in Fig. 5-4. Although the curve obtained was always alinear the latter part of each curve approximated closely to a straight line. It was possible, therefore, to analyze the nitrogen washout curve expressed in this manner into its components. The straight line drawn through the latter part of the original curve was extrapolated to breath zero (Fig. 5-4). The values of nitrogen concentration for each of the early breaths given by this extrapolated line were read from the graph and then subtracted from the corresponding values of the experimentally determined end-expiratory nitrogen concentration. The values so obtained were then plotted on the semi-logarithmic paper against the corresponding breath number. In all the present experiments this plot came very close to a straight line. Each of the experimentally determined nitrogen washout curves was expressed in terms of these two derived components representing a pair of compartments, one of which was washed out more rapidly than the other. From this graphical analysis of the nitrogen washout curve the fraction of the total ventilation passing to each compartment and the volume of the compartment were calculated using the methods developed by Fowler, Cornish and Kety (1952) (112) and Briscoe and Counand (1959) (47).

The fraction of the alveolar ventilation which was received by each of the compartments was calculated from the intercept of the corresponding semi-logarithmic plot on the nitrogen concentration axis at breath O according to the equation:
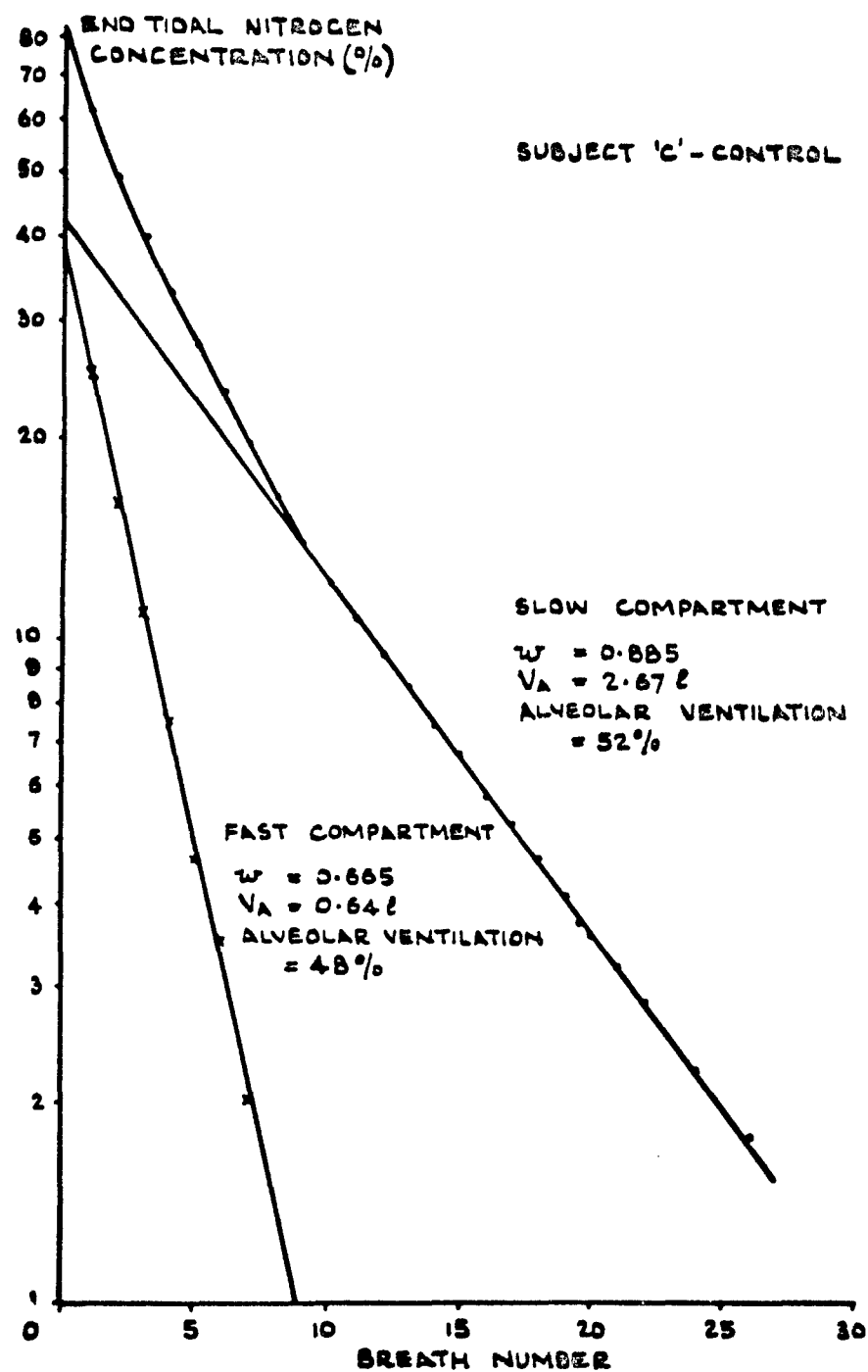
FIG. 5 4 A semi-logarithmic plot of the end-tidal nitrogen concentration during the breathing of 100% oxygen at rest. The curve has been analysed into two components

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$$\frac{F_{Ax}}{F_{Ao}} = \frac{V_{Ax}}{V_A}$$

Where:

F_{Ax} = Intercept of straight line representing compartment x

F_{Ao} = Intercept of original nitrogen washout curve (alveolar nitrogen concentration before breathing oxygen).

V_{Ax} = Alveolar ventilation per breath received by compartment x

V_A — Total alveolar ventilation per breath.

The total alveolar ventilation per breath was obtained by subtracting the anatomical dead space from the mean tidal volume. The appropriate values of the anatomical dead space for the subjects and conditions used in this investigation were obtained separately using Fowler's method (1948). It was possible with this information to calculate the actual ventilation per breath received by each compartment of the alveolar volume.

The alveolar dilution ratio for each compartment of the lung volume, which was an expression of the relation between the volume of the compartment and the ventilation it received per breath, was calculated from the slope of the straight line which represented the compartment in the graphical analysis of the nitrogen clearance curve. The alveolar dilution ratio which was defined as:

$$\omega_x = \frac{V_x}{V_x + V_{Ax}}$$

was related to the slope of the straight line by the expression:

$$m_x - \log_{10} \omega_x = \log_{10} \frac{V_x}{V_x + V_{Ax}}$$

Where:

ω_x = Alveolar dilution ratio of compartment x

V_x = Volume of compartment x

V_{Ax} = Ventilation per breath received by compartment x

m_x — Slope of semilogarithmic plot representing compartment x

From the value of the alveolar dilution ratio and the corresponding value of the actual ventilation received by the compartment the volume of the compartment was calculated. The sums of the volumes of the two separate compartments gave the functional residual capacity.

Two nitrogen washout curves were obtained during rest for each of the three subjects used in this part of the investigation. The values of the functional residual capacity, the alveolar dilution ratio, the volume and the fractional ventilation of the fast and slow compartments of the lungs obtained from the analyses of these curves are presented in Table 5-5. Duplicate experiments were performed during pressure breathing on each subject and the results of the analyses of these curves are also given in Table 5-5. Pressure breathing at 30 and 60 mmHg with trunk counterpressure produced a small increase in the functional residual capacity and the volumes of both compartments were also slightly increased. There was no change in the distribution of the alveolar ventilation between the two compartments. Pressure breathing at 20 mmHg without counterpressure induced a much greater increase in the functional residual capacity. This was accompanied by

TABLE 5-5

THE EFFECT OF PRESSURE BREATHING
UPON THE NITROGEN CLEARANCE CURVE

Subject	Tidal Volume (l/ B.T.P.S.)	Anatomical Dead Space (l/ B.T.P.S.)	F.R.C.[1] (l/ B.T.P.S.)	SLOW COMPARTMENT			FAST COMPARTMENT		
				Volume (l/ B.T.P.S.)	Alveolar Ventilation (%)	Alveolar Dilution Ratio	Volume (l/ B.T.P.S.)	Alveolar Ventilation (%)	Alveolar Dilution Ratio
Control									
B	0.751	152	3.07	2.23	61	0.863	0.84	39	0.780
B	0.733	154	2.85	2.04	52	0.871	0.81	48	0.745
C	0.806	161	2.97	2.24	45	0.883	0.73	55	0.668
C	0.822	165	3.32	2.67	52	0.885	0.64	48	0.665
D	0.790	152	2.98	2.23	48	0.879	0.75	52	0.691
D	0.798	154	3.23	2.52	56	0.874	0.71	44	0.713
Mean	0.783	156	3.07	2.32	52.3	0.876	0.75	47.7	0.710
Pressure breathing with trunk counterpressure at 30 mmHg									
B	0.920	167	3.46	2.75	55	0.870	0.71	45	0.676
B	0.852	170	3.49	2.66	49	0.889	0.83	51	0.707
C	0.813	175	3.13	2.13	45	0.881	1.02	55	0.745
C	0.827	178	3.52	2.44	48	0.885	1.08	52	0.760
D	0.798	168	3.15	2.20	49	0.879	0.95	51	0.680
D	0.802	165	3.36	2.34	51	0.876	1.02	49	0.750
Mean	0.837	171	3.35	2.42	49.5	0.880	0.94	50.5	0.720
Pressure breathing with trunk counterpressure at 60 mmHg									
B	0.824	175	3.33	2.53	48	0.889	0.80	52	0.703
B	0.863	171	3.59	2.80	52	0.882	0.79	48	0.710
C	0.901	168	3.57	2.92	53	0.881	0.65	47	0.657
C	0.873	170	3.62	3.02	54	0.880	0.60	46	0.655
D	0.852	173	3.52	2.42	47	0.882	1.10	53	0.753
D	0.831	170	3.53	2.53	52	0.878	1.00	48	0.770
Mean	0.837	173	3.52	2.70	51.0	0.882	0.82	49.0	0.708
Pressure breathing without trunk counterpressure at 20 mmHg									
B	0.956	195	4.94	4.10	49	0.920	0.84	51	0.691
B	0.800	190	5.07	4.32	55	0.930	0.71	45	0.734
C	0.970	183	5.14	4.36	53	0.905	0.78	47	0.683
C	1.050	175	5.15	3.92	52	0.897	1.23	48	0.751
D	0.880	195	4.64	3.72	45	0.933	0.92	55	0.708
D	1.050	190	5.51	4.43	51	0.910	1.08	49	0.721
Mean	0.951	188	5.07	4.14	50.8	0.914	0.93	49.2	0.712

[1] Functional Residual Capacity

proportional increases in both the fast and slow compartments of the lung volume.

Respiratory Dead Space – Anatomical and physiological dead spaces were measured virtually simultaneously during rest and pressure breathing at ground level. The anatomical dead space was measured by following the concentration of nitrogen in the inspired gas following a single breath of oxygen using the technique described in Chapter 3. The physiological dead space was determined from the carbon dioxide tensions of samples of the expired gas and the arterial blood collected simul'aneously.

The experiments were performed within the decompression chamber. The modified pressure helmet fitted with a mouthpiece was used. The sampling needle of the Lundin nitrogen meter was fitted directly into the mouthpiece, the distal end of which was connected to a valve box of minimal dead space. The dead space of the apparatus was equal to that of the apparatus used in the previous study. Two hoses led from the exterior of the decompression chamber to a two-way tap which was connected directly to the inlet of the valve box. Whilst the subject breathed air through one of the inlet hoses the other hose was connected to a 6 litre bell spirometer placed outside the decompression chamber. This spirometer was positioned so that the subject could clearly see the movements of the pen on the recording paper. Before each experiment the spirometer and the hose to the two-way tap were purged with oxygen and the spirometer was filled with this gas before each determination of the anatomical dead space. The outlet of the valve box was connected to the exterior of the decompression chamber where the expired gas could be collected in a Douglas bag. A heated Fleisch flowmeter was placed in the expiratory gas stream directly beyond the outlet of the valve box. The amplified output of the capacitance pressure transducer measuring the pressure difference created across the flow meter by expiratory flow and the output of the nitrogen meter were fed on to the galvanometers of a bromide paper recorder. The flow meter record was calibrated before and after each experiment using a standard rotameter. The output of the nitrogen meter was calibrated at the same time using mixtures of oxygen and nitrogen of known composition. The delay and the response times of the nitrogen meter to a square wave change of composition of gas at the needle valve were also determined before and after each experiment.

When the subject had donned the pressure jerkin and pressure helmet a Riley needle was introduced into a brachial artery under local analgesia. The patency of the needle was maintained by passing through it a slow infusion of physiological saline to which heparin had been added. After a preliminary rest period of at least ten minutes a timed three minute collection of expired gas was made with the subject breathing air. The number of expirations collected in the Douglas bag was counted. During the middle two minutes of this period a 20 ml sample of arterial blood was withdrawn at a steady rate. When the measurement was made during pressure breathing the duration of the expired gas collection, which was started during the second minute of the exposure, was reduced to two minutes. The sampling of arterial blood was performed over the middle ninety seconds of this two minute period. After the completion of each expired gas collection the anatomical dead space was measured immediately employing the technique described in

Chapter 3. The volume of expired gas collected was measured by means of a wet gas meter and the carbon dioxide concentration in the expired gas was determined in the Haldane apparatus. The carbon dioxide tension of the arterial blood was determined by the Astrup (1957 (11)) technique.

Results – Measurements of dead space were carried out at rest and during pressure breathing without counterpressure at a positive pressure of 20 mmHg and with counterpressure at positive pressures of 30 and 60 mmHg in three subjects. Duplicate determinations were made at rest and at each level of pressure breathing. The mean tidal volume was calculated from the volume of expired gas collected in the Douglas bag. The physiological dead space was determined using the Bohr equation, assuming that the arterial carbon dioxide tension represented the "effective" alveolar carbon dioxide tension:

$$V_D = \frac{F_{ACO2} - F_{ECO2}}{F_{ACO2}} V_T$$

Where:

V_D = Physiological dead space

V_T = Mean tidal volume

F_{ACO2} = Mean fractional concentration of carbon dioxide in alveolar gas.

F_{ECO2} = Mean fractional concentration of carbon dioxide in the expired gas.

The true physiological dead space was obtained by subtracting the instrumental dead space from the value obtained by this calculation.

The results of these calculations are presented in Table 5–6. The physiological dead space was consistently greater than the anatomical dead space both at rest and during pressure breathing. Both anatomical and physiological dead spaces were increased by pressure breathing. The increase of the physiological dead space was consistently greater than that of the anatomical dead space.

Alveolar Gas Tension – Two techniques were used to investigate the effect of pressure breathing upon the alveolar gas tensions. Intermittent sampling of the alveolar gas by the Haldane-Priestley technique was used extensively in this study. A limited series of experiments was carried out using a rapid response carbon dioxide analyser to measure the end-tidal carbon dioxide concentration and the effect upon it of pressure breathing.

Sampling of the alveolar gas by the Haldane-Priestley technique was performed in the decompression chamber. The subject wore the modified pressure helmet fitted with a mouthpiece. The mouthpiece was connected directly to a two-way tap which was attached to the external surface of the helmet. The arm of the tap which was in direct line with the lumen of the mouthpiece was connected by smooth-bore hose (I.D. 2.5 cm) to the exterior of the decompression chamber. A lateral tapping (3 mm I.D.) was fitted in this tube directly beyond the tap. A previously evacuated 100 ml gas sampling tube was attached to this tapping by a short length of rubber tubing. A valve box was connected to the second arm of the tap attached to the mouthpiece of the pressure helmet. The inlet and outlet of the valve box were connected to the exterior of the decompression chamber by smooth-bore hoses.

TABLE 5-6

THE VOLUMES OF THE ANATOMICAL AND PHYSIOLOGICAL DEAD SPACE

Subject	Tidal Volume (ml B.T.P.S.)	Art. CO_2 tension (mmHg)	Dead Space Volume (ml B.T.P.S.) Anatomical	Physiological
A. Control				
A	650	41.5	158	221
A	690	40.0	160	195
D	690	39.5	165	174
D	654	42.0	169	217
C	612	41.2	148	210
C	630	40.0	159	178
Mean	654	40.7	160	201
B. Pressure breathing at 20 mmHg without counterpressure				
A	674	39.5	199	305
A	747	38.0	201	350
D	846	39.5	189	280
D	928	37.5	188	250
D	796	38.0	176	290
C	685	40.5	190	230
Mean	779	38.8	191	284
C. Pressure breathing at 30 mmHg with trunk counterpressure				
A	720	39.2	170	280
A	658	40.2	173	333
D	697	39.5	161	236
D	645	38.0	175	283
C	778	38.0	178	348
C	870	37.0	161	248
Mean	728	38.7	170	288
D. Pressure breathing at 60 mmHg with trunk counterpressure				
A	747	38.0	167	397
A	740	37.5	180	290
D	860	38.5	176	339
D	894	38.6	164	356
C	849	37.6	175	276
C	788	39.0	178	334
Mean	813	38.2	173	332

The tap attached to the helmet was normally positioned so that the subject breathed through the valve box. When an alveolar gas sample was required the subject turned this tap at the end of a normal expiration and performed a rapid deep expiration. He then immediately returned the tap to its original position and resumed a normal breathing pattern. A sample of the alveolar gas trapped beyond the tap was taken rapidly into the evacuated sampling tube which had been previously connected to the side tapping. When the sampling tube had been replaced by another evacuated tube a further alveolar sample could be obtained. Normally at least one minute elapsed between the taking of samples. The concentrations of carbon dioxide and oxygen in the alveolar samples were determined in the Haldane apparatus.

The concentration of carbon dioxide in the respired gases was followed continuously by means of a rapid response infra-red analyzer. The measuring head of a Liston Becker Model 16 carbon dioxide analyzer fitted with a breathe-through cell was supported from the roof of the decompression chamber. It was attached directly to the mouthpiece of the modified pressure helmet. The volume of the system from the mouthpiece to the infra-red radiation pathway in the analyzer was 30 ml. The other end of the breathe-through cell of the analyzer was connected directly to a simple valve box, the inlet and outlet of which were connected to the exterior of the decompression chamber by smooth-bore hoses. The output of the carbon dioxide analyzer was fed on to a direct writing recorder. The carbon dioxide concentration in the respired gas was measured continuously, before, during and after pressure breathing at various levels. The output of the carbon dioxide analyzer was calibrated before and after each experiment with mixtures of carbon dioxide in air of known composition.

Discrete sampling of the alveolar gas and continuous recording of the respired carbon dioxide concentration were performed at rest, during pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure and during pressure breathing with trunk counterpressure at 50 and 80 mmHg. Four subjects, each of whom had had previous experience of pressure breathing, were studied.

Results – The individual values obtained for the carbon dioxide and oxygen tensions in the alveolar gas samples obtained in this study are presented in Figs. 5-5, 5-6 and 5-7. The alveolar carbon dioxide tension was markedly reduced by pressure breathing at a positive pressure of 30 mmHg without counterpressure and there was a corresponding increase of the alveolar oxygen tension. When trunk counterpressure was employed the changes were in the same direction but of lesser degree.

Continuous records of the respired carbon dioxide concentration were made in duplicated experiments in which each subject was exposed to the same conditions under which the Haldane-Priestley samples were obtained. The end tidal carbon dioxide tension was measured from each record. Comparison of the results of these measurements with the corresponding values of alveolar carbon dioxide obtained by Haldane-Priestley sampling show that there was close agreement between the two sets of results. Curves showing the mean time course of the alveolar tensions of carbon dioxide and oxygen have been constructed from the results obtained for all four subjects under the various conditions studied (Fig. 5-8).

FIG. 5-5 The effect of pressure breathing at a positive breathing pressure of 30 mmHg at ground level without respiratory counterpressure upon the alveolar oxygen and carbon dioxide tensions
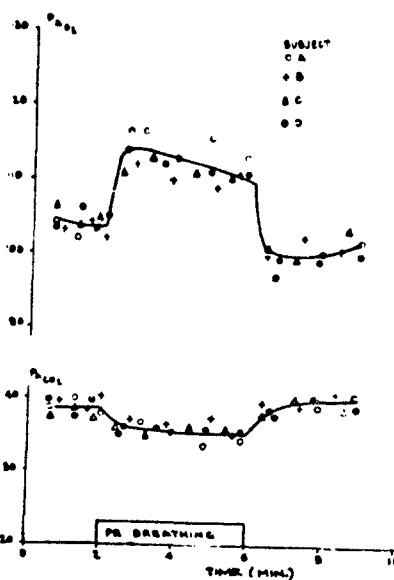
FIG. 5-6 The effect of pressure breathing at a positive breathing pressure of 50 mmHg with trunk counterpressure upon the alveolar oxygen and carbon dioxide tensions
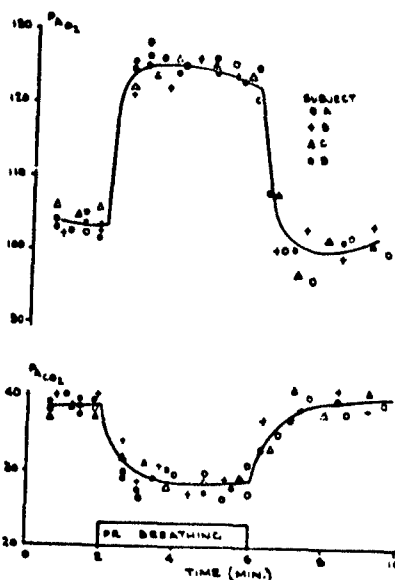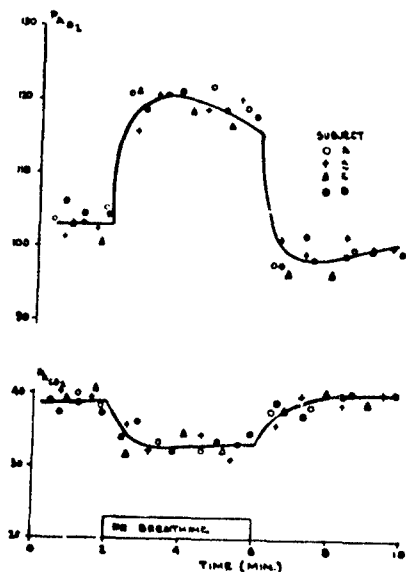

FIG. 5-7 The effect of pressure breathing at a positive breathing pressure of 80 mmHg at ground level with trunk counterpressure upon the alveolar oxygen and carbon dioxide tensions
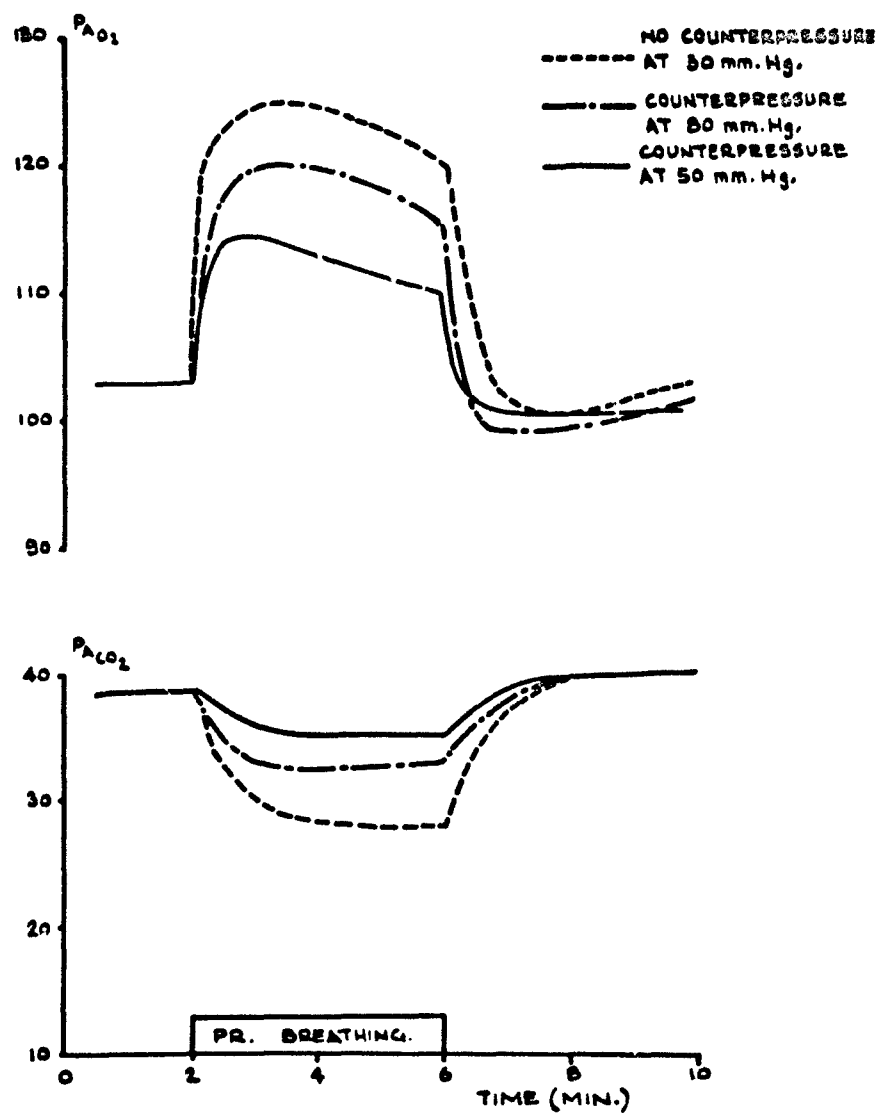
FIG. 5-8 The mean values of the alveolar gas tensions obtained before, during and after pressure breathing at 30 mmHg with no counterpressure and at 50 and 80 mmHg with trunk counterpressure

Diffusing Capacity of the Lungs – The effect of pressure breathing upon the exchange of gases between the alveolar space and the pulmonary capillary blood was investigated by measuring the diffusing capacity. This was estimated for carbon monoxide using the modified breath holding technique developed by Forster, Fowler, Bates and Van Lingen 1954 (109). The apparent diffusing capacity was measured at different levels of alveolar oxygen tension so that the diffusing capacity could be analyzed into its components (254).

The apparatus (Fig. 5–9) which was mounted in the decompression chamber consisted of two rubber bags, one of 8 litres and the other of 6 litres capacity, sealed within a metal box. Each bag was connected through the wall of the box to one arm of a two-way tap. The pair of two-way taps were connected together and to a third two-way tap in such a manner that the lumen of the third tap could be connected to either of the bags in turn (the smaller bag was closer to the third tap than the larger bag) or directly through the lumens of the two taps. One arm of the third tap ("helmet" tap) was connected to the mouthpiece of the modified pressure helmet. A simple valve box was attached to the third arm of the helmet tap. The inlet and outlet of the valve box were connected to the exterior of the decompression chamber by a pair of smooth-bore hoses. The other end of the taps attached to the bags within the box was connected by smooth-bore hose (2.5 cm I.D.) to a spirometer placed immediately outside the decompression chamber. This hose was also connected to the interior of the box containing the bags. The spirometer which had a lightweight bell, was fitted with a high speed kymograph which gave a recording paper speed of 1 cm/sec. An electric time clock and kymograph marker were used to mark one second intervals on the kymograph record.

In order to prepare the apparatus for the determination of the diffusing capacity, both the bags within the box were emptied. The larger bag was then filled with the gas mixture to be inspired. Two gas mixtures were used for this purpose, both mixtures containing about 0.25$\%$ carbon monoxide and 10$\%$ helium. In one mixture the only other gas was oxygen, whilst in the other oxygen and nitrogen were added to give an oxygen concentration of about 21$\%$. Each mixture was made up before the start of an experiment in a Tissot spirometer and stored in a Douglas bag. The composition of each of the inspired mixtures was determined immediately before the actual measurement of the diffusing capacity.

The subject, wearing a pressure jerkin, was seated before the apparatus in the decompression chamber and the helmet fitted with the mouthpiece was donned. Prior to the donning procedure the helmet tap was turned so that the mouthpiece was connected to the valve box. After a five-minute rest period during which the subject breathed either air or oxygen through the mouthpiece, the kymograph was started and the subject instructed to perform a maximum expiration and then hold his breath. Whilst the subject held his breath the helmet tap and the tap attached to the bag filled with the mixture to be inspired were opened so that the bag communicated with the mouthpiece. The subject made a maximum inspiration without excessive effort and then maintained the fully inflated position with his glottis open for ten seconds. During this period the tap attached to the inspirate bag was closed so
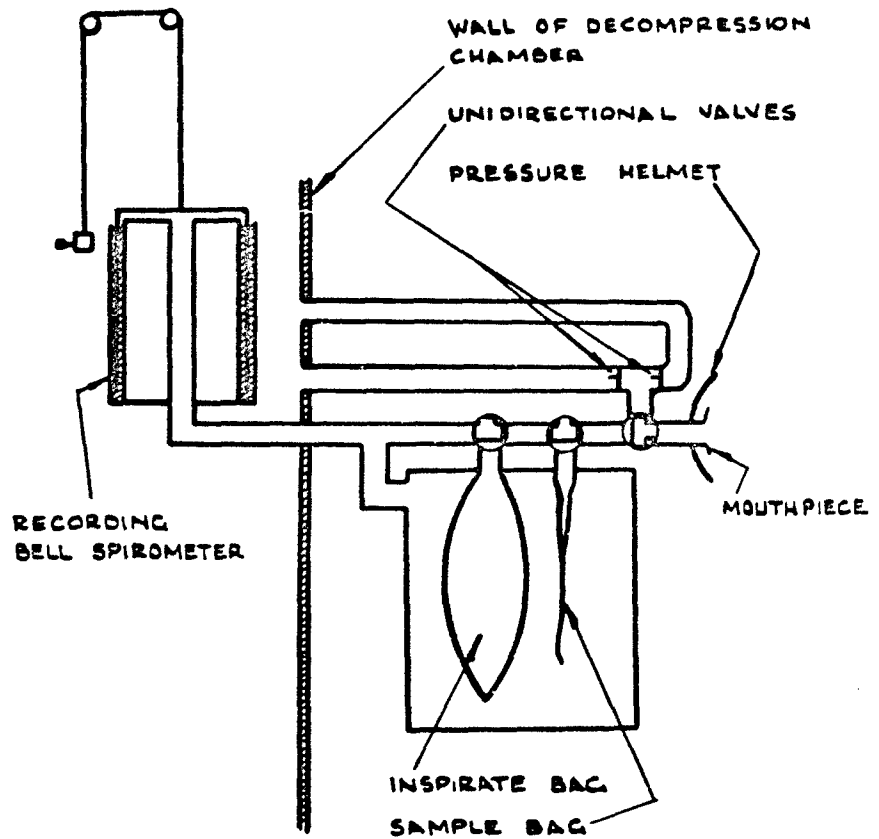
Fig. 5 9 The apparatus used for the determination of the diffusing capacity during pressure breathing

that the mouthpiece was connected to the spirometer. The subject performed
a rapid expiration at the end of the breath holding period. After about
1 litre of expired gas had passed into the spirometer, the tap of the smaller
bag (sample bag) was turned and the next litre or so of expired gas was
collected. After this fraction of the expired gas had been collected the tap into
the sample bag was closed and the remainder of the expiration passed into
the spirometer. The helmet tap was returned to its original position so that
the mouthpiece communicated with the valve box and the subject resumed a
normal breathing pattern. The expired gas sample was then transferred to a
rubber bag and the concentrations of carbon monoxide, helium, carbon
dioxide and oxygen in it were determined.

The diffusing capacity was measured at various alveolar oxygen tensions
by varying the oxygen concentration in the carbon monoxide-helium mixture
to be inspired and by the subject breathing air or oxygen for five minutes
before the measurement. Measurements made during pressure breathing were
performed during the second minute of the exposure. Only one measurement
was made during each exposure to pressure breathing. Generally four to six
consecutive measurements of diffusing capacity were made in any one experi-
ment, measurements during pressure breathing being interspersed by
measurements on the resting subject. Before and after each group of measure-
ments of diffusing capacity the equilibrated pulmonary capillary carbon
monoxide tension was determined. The subject hyperventilated whilst
breathing 100% oxygen for two minutes, held his breath for two minutes and
then delivered a sample of alveolar gas into an evacuated rubber bag. The
carbon monoxide and oxygen concentrations in the sample were then
measured. Repeated measurements of diffusing capacity at various alveolar
oxygen tensions were made in three subjects at rest and whilst breathing at
positive pressures of 40 and 80 mmHg with trunk counterpressure. The
residual volume for each subject was determined in duplicate in each of these
experimental conditions by the technique described in Chapter 4.

The Calculation of the Apparent Diffusing Capacity – The apparent
diffusing capacity (D_L) was calculated using the equation derived by Krogh
and Krogh 1910:

$$D_L = \frac{V_A}{(P_B - 47)t} \times \ln \frac{Initial\ F_{ACO}}{Final\ F_{ACO}}$$

D_L = apparent diffusing capacity (ml S.T.P./min./mmHg)
P_B = absolute pressure in the lungs (mmHg)
t = period of breath holding (minute)
V_A = alveolar volume (ml S.T.P.)
$Initial\ F_{ACO}$ = alveolar concentration of carbon monoxide at the
beginning of the breath holding period
$Final\ F_{ACO}$ = alveolar concentration of carbon monoxide at the end
of the breath holding period

The alveolar volume (V_A) was calculated by adding the residual capacity
obtained independently to the inspired volume read off the kymograph
record of the spirometer volume. The time of breath holding was measured
from the spirometer kymograph record. The interval from a point one third
of the duration of inspiration from the start of inspiration to the mid-point of

sampling was used (165). The initial alveolar concentration of carbon monoxide was calculated from dilution of the inspired helium by the residual gas according to the equation:

$$\text{Initial } F_{ACO} = \frac{F_{He} \text{ in expired alveolar sample}}{F_{He} \text{ in inspired gas}} \times F_{CO} \text{ in inspired gas}$$

The initial and final alveolar carbon monoxide tensions were corrected by subtracting the pulmonary capillary carbon monoxide tension to obtain the true alveolar-capillary tension gradient. The pulmonary capillary carbon monoxide tension existing in each experiment was calculated from the equilibrated pulmonary capillary carbon monoxide tension which was estimated before and after each group of measurements of diffusing capacity. The carboxyhaemoglobin concentration in the mixed venous blood was estimated from the carbon monoxide and oxygen concentrations in the alveolar gas sample obtained following breath holding after hyperventilation with oxygen by the Haldane relationship:

$$\frac{\%CO \text{ } Hb}{\%O_2 \text{ } Hb} = 210 \times \frac{F_{ACO}}{F_{AO2}}$$

which, since the concentration of haemoglobin was negligible, could be reduced to:

$$\%CO \text{ } Hb = \frac{100}{\dfrac{F_{AO2}}{210 \times F_{ACO}} + 1}$$

In this way the mixed venous carboxyhaemoglobin concentration was estimated before and after each group of measurements of diffusing capacity. The venous carboxyhaemoglobin concentration for a given measurement was calculated by interpolation. The equilibrated carbon monoxide tension for the measurement was then calculated using the Haldane relationship knowing the mean alveolar oxygen concentration which existed during the measurement. This calculation was simple when the alveolar oxygen tension exceeded 200 mmHg since the concentration of reduced haemoglobin in the pulmonary capillary blood could be assumed to be negligible and the following relationship could be used:

$$F_{ACO} = \frac{F_{AO2} \times \%CO \text{ } Hb}{210 \times (100 - \%CO \text{ } Hb)}$$

When however the alveolar tension was less than 200 mmHg it had to be assumed that the mean capillary oxygen tension was 10 mmHg less than the alveolar oxygen tension and that the mean oxyhaemoglobin concentration was the corresponding saturation (110). The following form of the Haldane relationship was then used to calculate the equilibrated carbonmonoxide concentration:

$$F_{ACO} = \frac{\%CO \text{ } Hb \times F_{AO2}}{\%O_2 \text{ } Hb \times 210}$$

The apparent diffusing capacity obtained in a given experiment was related to the mean alveolar oxygen tension which existed during the measurement. It was assumed that the mean alveolar oxygen tension was 5 mm

greater than the value measured at the end of the breath-holding period since in the resting subject the alveolar oxygen tension falls about 10 mmHg during a ten second breath hold.

Results – The values of the apparent diffusing capacity obtained under the various experimental conditions used in this study are present in Table 5–7 in relation to the corresponding mean alveolar oxygen tension. In each experimental condition the diffusing capacity fell as the alveolar oxygen tension was increased. Pressure breathing reduced the diffusing capacity at all alveolar oxygen tensions. For each subject in each experimental condition the relationship between the apparent diffusing capacity and the mean alveolar oxygen tension was used in order to analyse the apparent diffusing capacity into its two components, the diffusing capacity of the pulmonary membrane itself (D_M, ml S.T.P./min./mmHg) and the rate of uptake of carbon monoxide by the blood in the pulmonary capillaries exposed to the alveolar gas. This analysis was based upon the equation developed by Roughton and Forster 1957 (254):

$$\frac{1}{D_L} = \frac{1}{D_M} + \frac{1}{\theta . V_c}$$

D_L = the apparent diffusing capacity (ml S.T.P./min./mmHg)

D_M = diffusing capacity of the pulmonary membrane (ml S.T.P./min./mmHg)

θ = rate of uptake of carbon monoxide (ml S.T.P./min.) by unit volume of blood (ml) per unit of carbon monoxide tension (mmHg)

V_c = average volume of blood in pulmonary capillaries (ml)

The value of θ depends upon the pulmonary capillary oxygen tension; as the oxygen tension increases the value of θ decreases because of the competition of oxygen with carbon monoxide for haemoglobin. In order to obtain the correct value of θ for each measurement of the diffusing capacity the mean capillary oxygen tension was calculated. This quantity was obtained by subtracting the mean alveolar gas to capillary oxygen tension difference from the average alveolar oxygen tension during breath holding. The average difference was calculated by dividing an assumed oxygen consumption by the apparent diffusing capacity for oxygen using the relationship:

$$D_L \text{ for oxygen } = 1.23 \times D_L \text{ for carbon monoxide}$$

The value of θ corresponding to the calculated mean capillary oxygen tension was obtained from the data of Roughton and Forster (1957) (254) using a value of the ratio of membrane permeability to that of the interior of the corpuscle of 2.5. It was assumed that the blood of all the subjects had a carbon monoxide-carrying capacity of 20 ml per 100 ml of blood. The values of $\frac{1}{\theta}$ were calculated from the relationship:

$$\frac{1}{\theta} = 0.72 + 0.0057 \, P_{CO_2}$$

where P_{CO_2} is the mean capillary oxygen tension.

For each individual series of measurements of diffusing capacity the values

TABLE 5-7

THE EFFECT OF PRESSURE BREATHING
UPON THE APPARENT DIFFUSING CAPACITY

Condition	Control		Pressure breathing at 40 mmHg		Pressure breathing at 80 mmHg	
Subject	Mean alveolar oxygen tension (mmHg)	Diffusing capacity (ml/min./ mmHg)	Mean alveolar oxygen tension (mmHg)	Diffusing capacity (ml/min./ mmHg)	Mean alveolar oxygen tension (mmHg)	Diffusing capacity (ml/min./ mmHg)
	108	32.8	105	29.1	106	24.4
	113	33.1	130	26.0	125	21.7
B	228	26.8	256	21.5	232	16.7
	433	19.6	205	15.6	390	14.1
	522	18.4	252	14.0	490	11.2
	565	17.1	530	14.2	530	11.4
	109	33.3	106	26.5	104	23.4
	121	29.6	123	27.4	163	22.5
C	260	25.1	221	21.1	220	18.4
	275	23.8	335	19.1	410	15.4
	285	18.6	440	15.2	473	13.1
	510	19.2	512	14.9	503	13.7
	107	29.6	98	25.6	101	25.3
	155	28.6	113	22.2	162	20.7
D	186	24.7	215	20.2	233	19.6
	355	19.2	349	15.1	360	14.2
	472	18.1	426	14.8	435	13.4
	538	15.6	482	12.9	545	11.5

TABLE 5-8

THE EFFECT OF PRESSURE BREATHING
UPON THE DIFFUSING CAPACITY OF THE PULMONARY MEMBRANE
AND THE PULMONARY CAPILLARY BLOOD VOLUME

Subject	Diffusing capacity of pulmonary membrane (ml/min./mmHg)	Pulmonary capillary blood volume (ml)	Mean alveolar volume during breath hold (litre B.T.P.S.)
		Control	
B	60.6	94.2	6.05
C	52.0	104.6	5.75
D	52.6	89.5	5.83
Mean	55.0	96.1	5.88
		Pressure breathing at 40 mmHg	
B	66.0	65.0	6.47
C	55.1	70.3	5.93
D	48.2	62.3	6.23
Mean	56.4	65.9	6.21
		Pressure breathing at 80 mmHg	
B	58.0	30.5	6.61
C	50.3	62.2	6.22
D	56.1	53.5	6.33
Mean	54.8	55.4	6.3

of the reciprocals of the apparent diffusing capacity were plotted against the corresponding calculated values of $\frac{1}{\theta}$. In each of the various series of experimental results the points thus plotted lay close to a straight line (Fig. 5-10). A straight line was fitted to each group of points by the method of least squares with $\frac{1}{\theta}$ as the independent variable. The value of the intercept of this line on the $\frac{1}{D_L}$ axis and its slope were measured. The reciprocal of the $\frac{1}{D_L}$ intercept gave the diffusing capacity of the pulmonary membrane whilst the reciprocal of the slope of the line was the mean pulmonary capillary blood volume.

The results of this resolution of the apparent diffusing capacity into its components are presented in Table 5-8. Whilst pressure breathing caused no significant change in the diffusing capacity of the pulmonary membrane there was a progressive reduction in the pulmonary capillary blood volume as the positive breathing pressure was increased.

PRESSURE BREATHING AT REDUCED BAROMETRIC PRESSURE

Pulmonary Ventilation – A limited series of measurements of pulmonary ventilation were made during pressure breathing at reduced barometric pressure in the decompression chamber by recording the inspiratory flow. The subject wore a pressure jerkin and the modified pressure helmet fitted with a mouthpiece. A standard inlet non-return and compensated outlet valve system was connected directly to the mouthpiece. The outlet of a pressure demand regulator (Mark 20) was connected to the inlet tube of the mouthpiece, the face compartment of the pressure helmet and the bladder of the pressure jerkin. A Fleisch flowmeter was fitted directly upstream of the inlet valve of the mouthpiece and the pressure difference created across it by inspiratory flow was recorded on a galvanometer recorder by means of a capacitance transducer and an appropriate amplifier. The pressure in the mouthpiece was measured by means of a mercury manometer placed within the decompression chamber. After a preliminary ascent to a pressure altitude of 25 000 ft where a two minute record of the resting inspiratory flow was taken, the subject was decompressed in two seconds to a simulated altitude of 56 000 ft. The demand regulator automatically delivered a positive breathing pressure of 80 mmHg at 56 000 ft. This pressure-altitude was maintained for two minutes and followed by immediate descent. The flow record was calibrated with a standard rotameter at ground level before and after each experiment. Results – Each of the four subjects was decompressed to a pressure altitude of 56 000 ft on two separate occasions. The positive breathing pressure measured at the mouthpiece at the final altitude was 80 mmHg. The volume of each breath was determined by the planimetric integration of the inspiratory flow record The inspiratory minute volume was calculated for each minute of the rest and pressure breathing periods. These results are presented in Table 5-9. There was some variability in the response from one subject to another, although the ventilation was always increased following decompression. The mean increase of pulmonary ventilation during the two minute period of pressure breathing as compared with the corresponding control period was 3.89 litre (standard error $\pm$ 0.22).
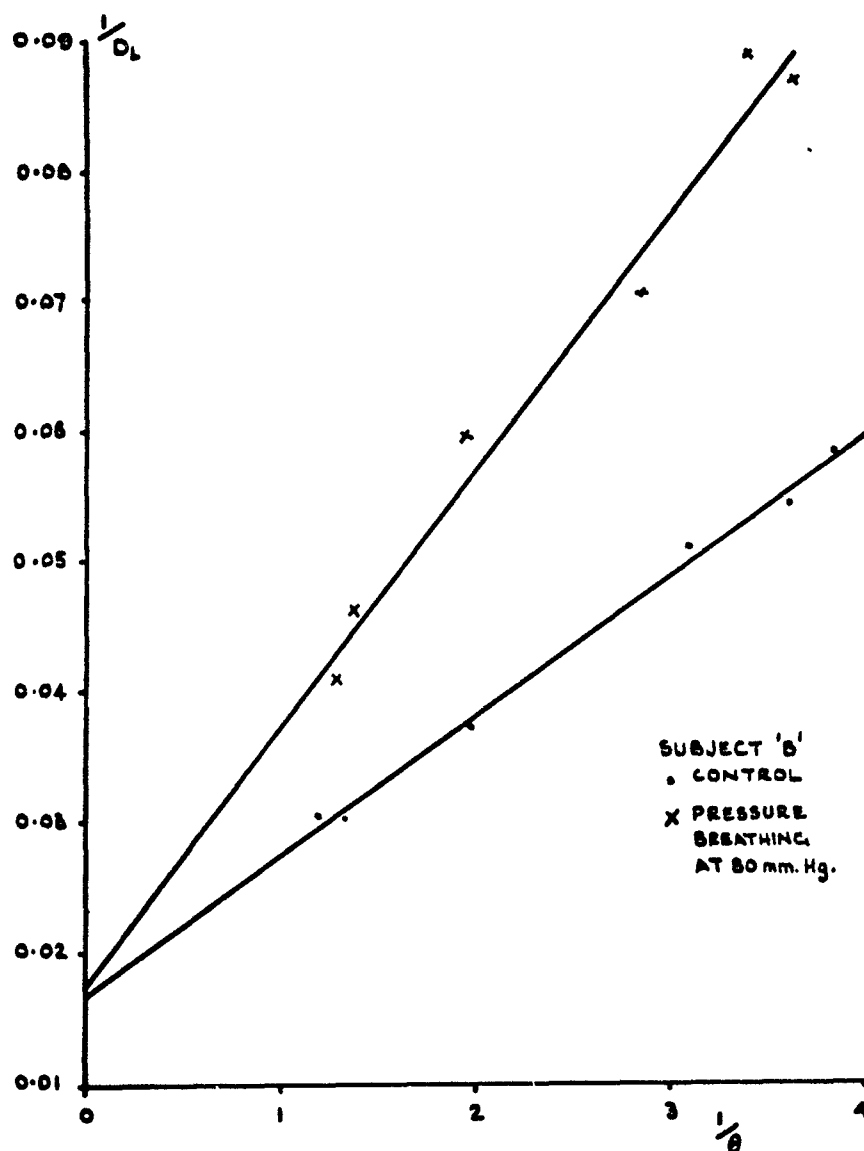
FIG. 5-10 The relationship between the reciprocal of the apparent diffusing capacity and $\frac{1}{\theta}$ in subject B at rest and whilst pressure breathing at 80 mmHg with trunk counterpressure

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Alveolar Gas Tensions – Intermittent sampling of alveolar gases was performed by the Haldane-Priestley technique during pressure breathing at reduced barometric pressure. The effects of various positive breathing pressures and various absolute intrapulmonary pressures were investigated.

Essentially the apparatus used for alveolar gas sampling at reduced barometric pressure was very similar to that used for Haldane-Priestley sampling at ground level. The subject wore the pressure helmet fitted with a mouthpiece which was connected through a two-way tap to a valve box. A standard inlet non-return and compensated outlet valve system was fitted to the valve box. The outlet of an appropriate pressure demand regulator was connected, to the inlet of the valve box, the face compartment of the helmet and the bladder of the pressure jerkin. The sampling hose, which was thick walled, led from the other arm of the two-way tap attached to the mouthpiece to the second compartment of the decompression chamber. The pressure at the mouthpiece was measured by means of a mercury manometer placed in the decompression chamber. Before the subject was decompressed to the final altitude the pressure in the second compartment of the decompression chamber was reduced to equal the absolute pressure which it was expected the oxygen regulator would deliver after the decompression. The latter was determined from a preliminary calibration of the pressure breathing characteristics of the regulator. In practice the absolute pressure in the respiratory tract following decompression never differed by more than 2 mmHg from the pressure held in the second compartment of the decompression chamber.

The actual sampling of alveolar gas at reduced barometric pressure was carried out in the same manner as that at ground level. In order, however, that two samples of alveolar gas could be obtained following a decompression to reduced pressure two 100 ml evacuated gas sampling tubes were attached to the lateral tapping in the sampling hose. In these circumstances the dead space between the lumen of the hose and the tap of each of the tubes was filled with mercury before the ascent to reduced barometric pressure. Following the completion of sampling the subject was brought to a pressure altitude of 38000 ft and at this level each sampling tube was fixed vertically, attached to a mercury reservoir and the lower tap opened. During this procedure care was taken to avoid the contamination of the sample with air. As the pressure in the decompression chamber was increased further the subject ensured that the pressure of each of the samples exceeded that in the decompression chamber by maintaining the level of the mercury in the reservoir higher than that in the sampling tube. The concentrations of carbon dioxide and oxygen in the alveolar samples were determined in the Haldane apparatus using the nitrogen dilution technique.

Alveolar gas samples were obtained before rapid decompression after the subject had breathed 100"₀ oxygen at a pressure altitude of 25000 ft for five minutes. The subject was decompressed to the final altitude and the exposure maintained for two minutes during which two Haldane-Priestley samples of alveolar gas were obtained. Several series of experiments were performed in which the final pressure altitude and the breathing pressure were varied. Conditions which were investigated were:

    (a) A final pressure altitude of 50000 ft at a positive breathing pressure of 30 mmHg without respiratory counterpressure.

TABLE 5-9

THE PULMONARY VENTILATION DURING PRESSURE BREATHING
AT REDUCED ENVIRONMENTAL PRESSURE

Subject	Pulmonary Ventilation (1/min. B.T.P.S.)				Positive Breathing Pressure
	Control		Pressure Breathing		
	(at simulated 25000 ft)		(at simulated 56000 ft)		(mmHg)
Time (min.)	1	2	1	2	
A	8.5	8.4	12.5	11.3	80.5
A	8.3	8.5	13.6	11.8	79.0
B	7.8	7.5	11.9	11.6	80.0
B	8.1	8.3	12.7	12.0	79.0
C	7.8	8.1	10.9	10.8	81.0
C	7.9	7.8	12.1	11.5	81.5
D	7.7	8.0	11.6	11.3	80.5
D	8.3	8.5	10.9	10.7	79.0

(b) A final pressure altitude of 54000 ft at a positive breathing pressure of 50 mmHg using a pressure jerkin.

(c) A final pressure altitude of 55000 ft at a positive breathing pressure of 60 mmHg using a pressure jerkin.

(d) A final pressure altitude of 57500 ft at a positive breathing pressure of 80 mmHg using a pressure jerkin.

A limited number of experiments were performed in which the tensions of the alveolar gases were determined when oxygen at the pressure of the environment was breathed at various absolute pressures between 117 and 141 mmHg (equivalent to altitudes of 44000 and 40000 ft respectively). The same apparatus was used as in the pressure breathing experiments except that a demand regulator which provided oxygen at the pressure of the environment at all altitudes was used. After breathing oxygen for five minutes at a simulated altitude of 25000 ft the subject was decompressed in two seconds to a final pressure altitude which was varied between 40000 and 44000 ft. The final pressure altitude was maintained for two minutes during which the subject delivered two Haldane-Priestley samples of alveolar gas.

Results – The samples of alveolar gas obtained whilst breathing 100"₀ oxygen at a simulated altitude of 25000 ft before decompression had a mean carbon dioxide tension of 40.3 mmHg (S.E. $\pm$1.5 mmHg) and a mean oxygen tension of 192 (S.E. $\pm$2.1 mmHg).

In certain of the experimental conditions used the subject became confused before the completion of a two minute exposure. This situation arose during breathing at a positive pressure of 30 mmHg at a simulated altitude of 50000 ft and whilst breathing oxygen at 118 mmHg absolute. When confusion occurred the subject was immediately recompressed to a higher pressure. In all the alveolar gas samples taken at reduced pressure the nitrogen concentration was less than 3"₀. The alveolar gas tensions were calculated from the results of the analyses of the Haldane-Priestley samples and the absolute intrapulmonary pressure. During pressure breathing the absolute intrapulmonary pressure was calculated by adding the pressure recorded by the mercury manometer connected to the mouthpiece to the absolute pressure within the decompression chamber (water vapour pressure was assumed to be 47 mmHg). The individual values obtained for the four subjects used in this study have been plotted in relation to the instant at which they were obtained for each of the four pressure breathing conditions studied. These results are presented in Figs. 5–11 and 5–12. In each condition the alveolar carbon dioxide tension was reduced immediately after the decompression and increased again during the subsequent two minutes of pressure breathing. The rate at which the carbon dioxide tension increased varied with the breathing pressure. The oxygen tension changed in the opposite manner to the alveolar carbon dioxide tension. The alveolar carbon dioxide tension obtained during the breathing of oxygen at absolute pressures between 117 and 141 mmHg are presented in Fig. 5–13 in relation to the instant at which decompression occurred. The patterns of change of alveolar carbon dioxide and oxygen tensions in these experiments were similar qualitatively to those obtained during pressure breathing.

Arterial Blood Gases – The arterial oxygen and carbon dioxide tensions achieved by pressure breathing at reduced environmental pressure were

Fig. 5-11 The alveolar gas tensions obtained during pressure breathing with oxygen at simulated altitudes of 50000 ft and 54000 ft (Pr.Br. — positive breathing pressure; PT — absolute intrapulmonary pressure)

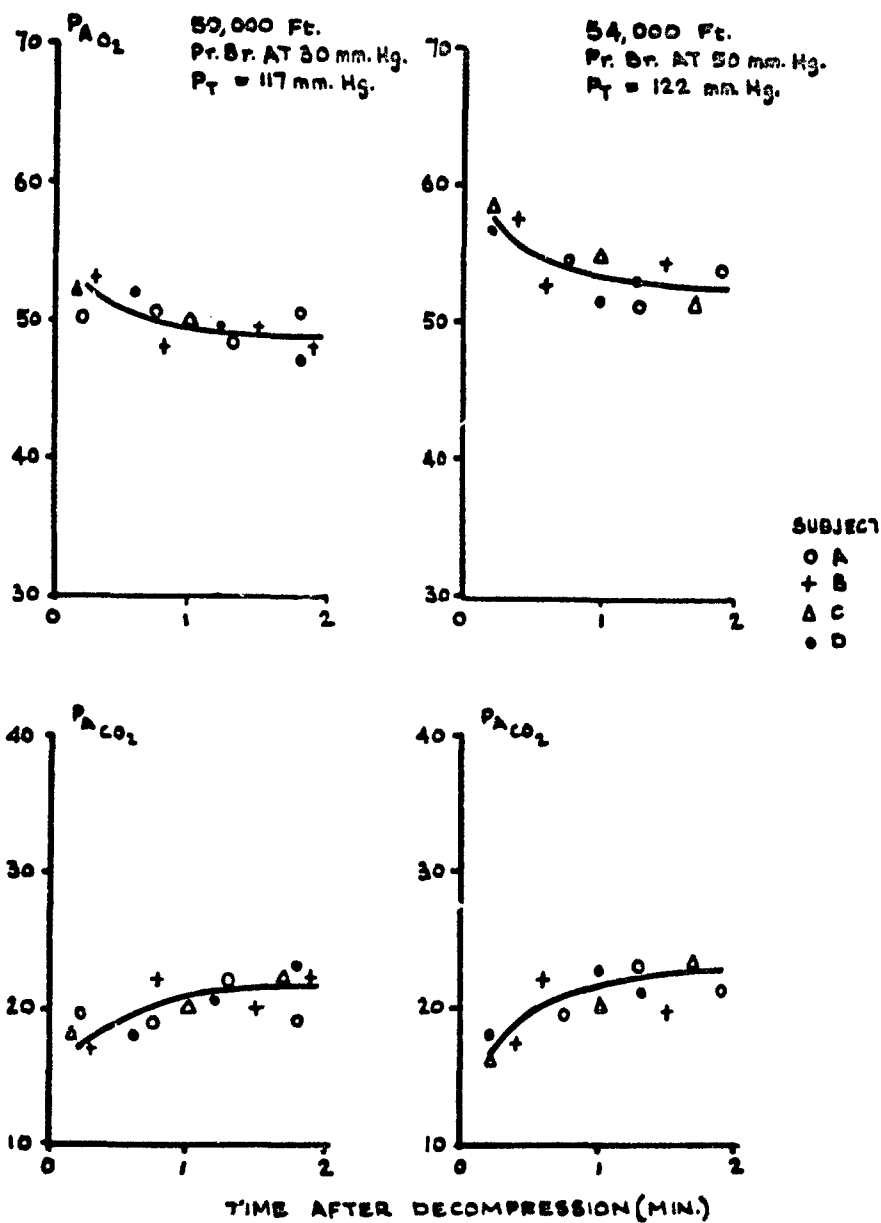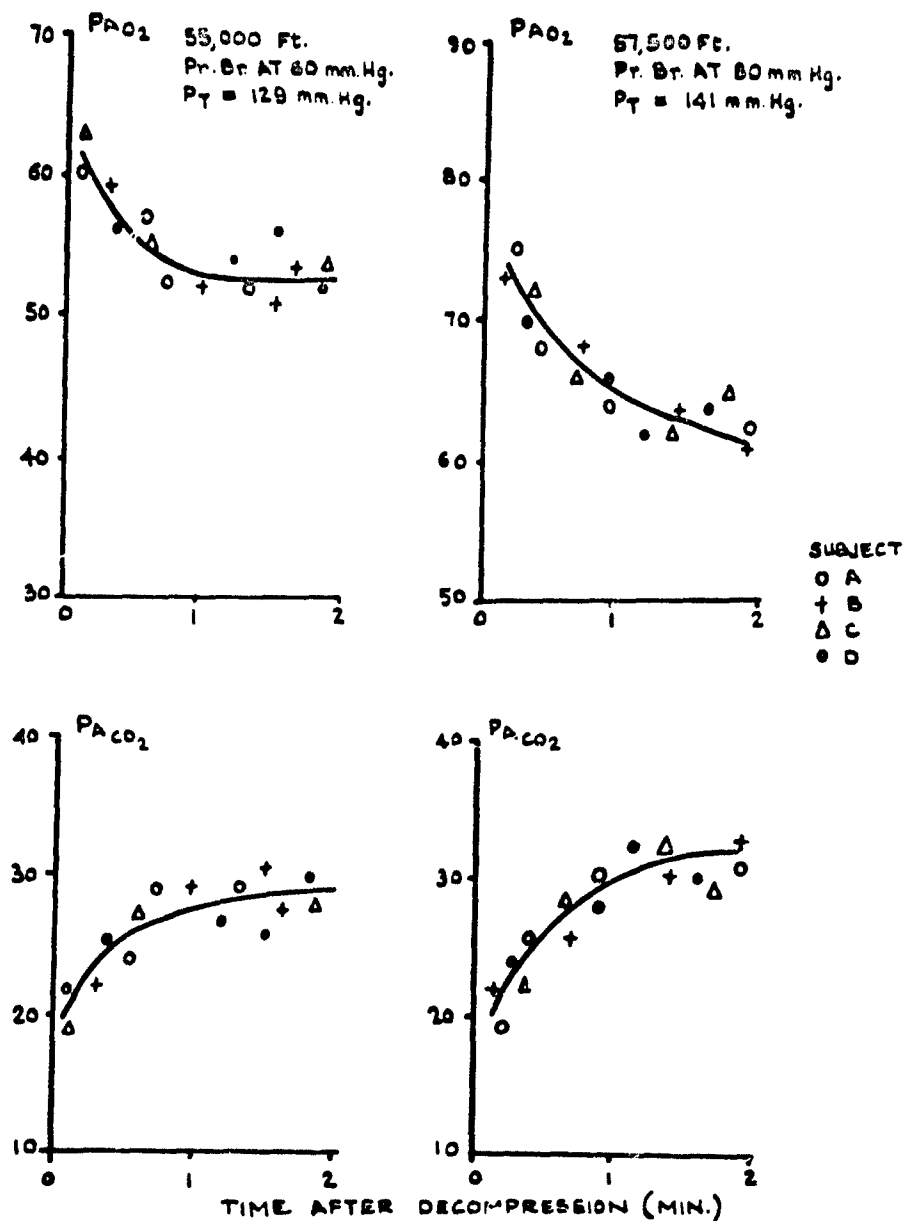FIG. 5-12 The alveolar gas tensions obtained during pressure breathing at simulated altitudes of 55000 ft and 57500 ft (Pr.Br. — positive breathing pressure: Pr — absolute intrapulmonary pressure)
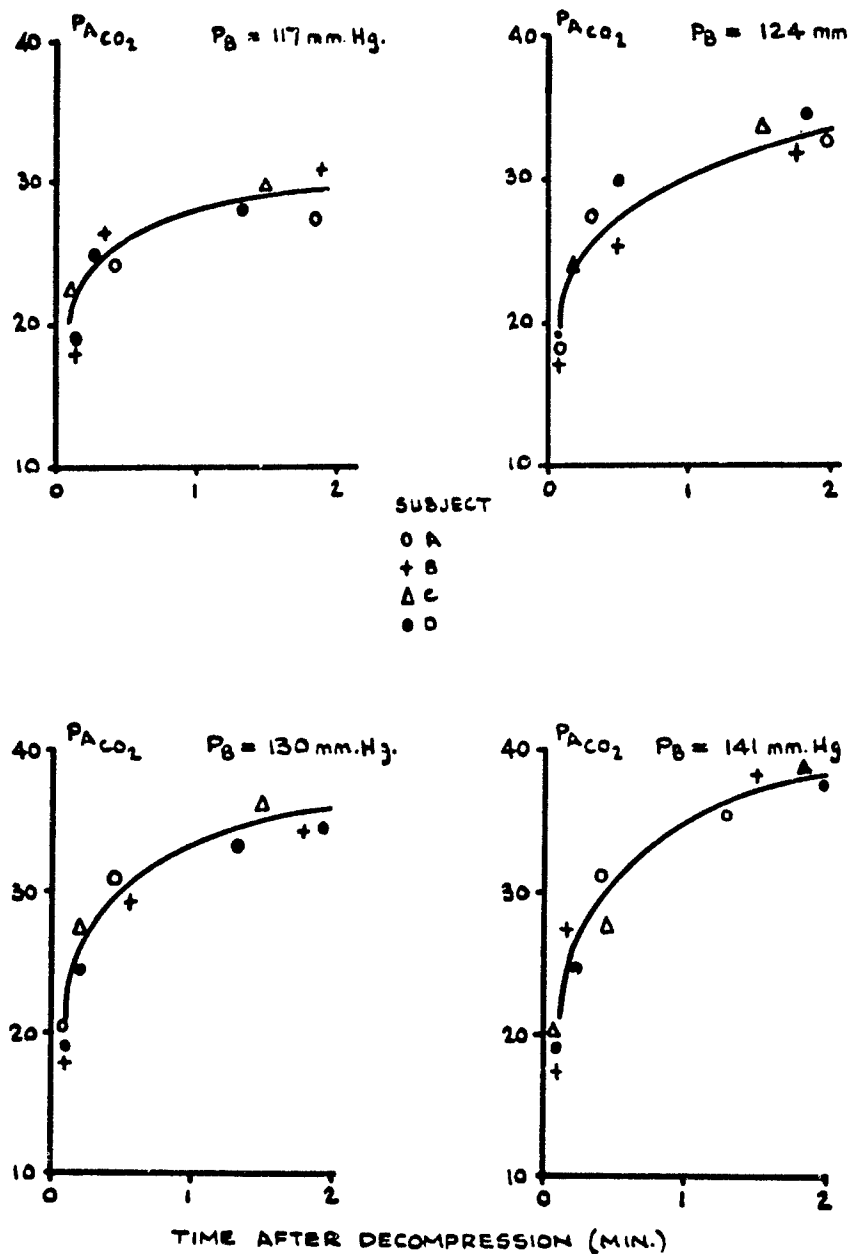
Fig. 5-13 The alveolar carbon dioxide tensions whilst breathing oxygen at various environmental pressures following a rapid decompression from a simulated altitude of 25000 ft (PB absolute environmental pressure)

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studied by sampling blood from the brachial artery following decompression in the decompression chamber. Two pressure breathing systems were investigated at a simulated altitude of 56000 ft, namely the combination of an oronasal mask, jerkin and anti-g suit with the Mark 21 pressure demand regulator, and the combination of a pressure helmet, jerkin and anti-g suit with the Mark 20 pressure demand regulator. Since the environmental pressure at a simulated altitude of 56000 ft was considerably lower than the sum of the partial pressures of the gases in the arterial blood, a special blood sampling technique was developed in order to avoid the formation of gas bubbles in the arterial sample.

The subject, wearing the appropriate standard pressure clothing assembly, was seated in the decompression chamber. The pressure at the mouth was measured by means of a mercury manometer which was mounted in the decompression chamber. A Riley intra-arterial needle was introduced into one brachial artery under local analgesia. The needle was then connected by a short (3 cm) length of polyethylene tubing to a pair of three-way taps connected in series (Fig. 5–14). The side arm of the first tap was attached through an adjustable needle valve to a bottle of sterile physiological saline to which heparin had been added. The air inlet to the space above the saline in the bottle was connected to a pressure demand regulator which maintained an outlet pressure 300 mmHg greater than that of the pressure within the decompression chamber. The side-arm of a "T" piece placed in the connection between the regulator and the bottle containing saline was closed with a clamp. This system could be vented to the decompression chamber by removing the clamp. A mercury manometer was also attached to the system so that the pressure within it could be determined. The second of the pair of taps connected to the intra-arterial needle was attached to a 20 ml syringe. The barrel of the syringe was passed through the wall of a perspex box and clamped so that the piston of the syringe was within the box. A metal rod which also passed through a wall of this box was attached to the piston of the syringe so that the piston could be pulled along the barrel of the syringe when the box was sealed. The interior of the box was connected to a pressure demand regulator which maintained an outlet pressure of 141 mmHg absolute at pressure-altitudes above 40000 ft. This pressurization system also contained a relief valve which operated when the pressure in the box exceeded that in the chamber by 100 mmHg. The side arm of the second tap was connected to a waste bottle which was also in communication with the outlet of the regulator controlling the pressure within the syringe box.

Before the intra-arterial needle was inserted the dead space of the syringe and of the taps attached to it were filled with mercury and heparin solution. The first tap was then turned so that saline flowed from the reservoir bottle, through the polyethylene tubing to the intra-arterial needle. The flow of saline was adjusted to a very slow rate by means of the needle valve. A medical officer wearing a partial pressure helmet, jerkin and anti-g suit connected to a Mark 20 regulator accompanied the subject in the decompression chamber. After the insertion of the intra-arterial needle and the setting up of the slow infusion of heparinized saline the pressure within the decompression chamber was reduced slowly until a pressure altitude of 25000 ft was attained. During the reduction of pressure the vent on the system pressurizing the saline bottle

STERILE SALINE
+ HEPARIN

MERCURY
MANOMETER

VENT

NEEDLE VALVE

SAMPLE SYRINGE

CONSTANT
PRESSURE
REGULATOR
(+300 mm. Hg.)

'RILEY' INTRA-ARTERIAL
NEEDLE

THREE WAY TAPS

RELIEF VA
(100 mm. H

WASTE BOTTLE

REGULATOR PROVIDING
141 mm. Hg. abs.
AT ALTITUDES GREATER
THAN 40,000 Ft.
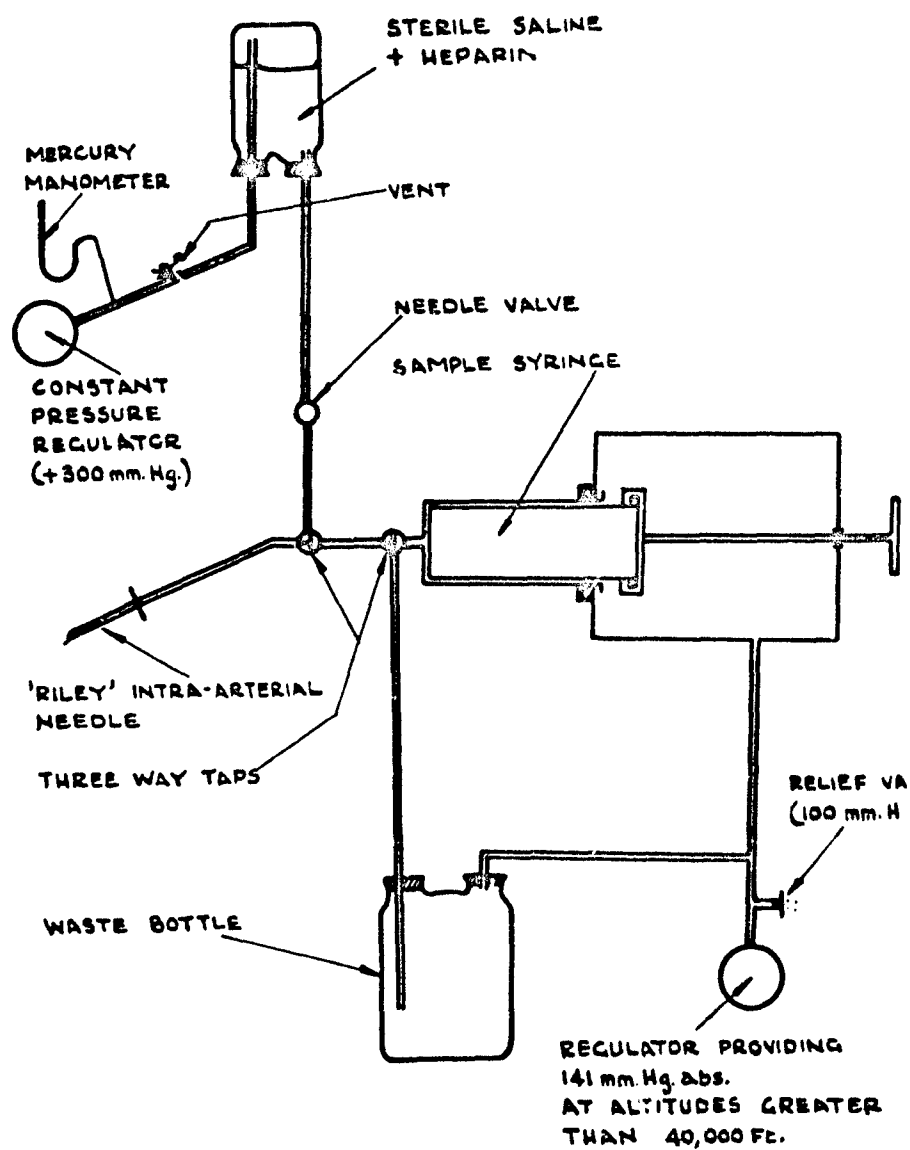
FIG. 5-14 The apparatus used for the intermittent sampling of arterial blood during
pressure breathing at simulated high altitude

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was opened periodically to prevent an excessive build-up of pressure in the system. After a rest period of three to five minutes at a pressure altitude of 25,000 ft, the subject and the accompanying medical officer were decompressed to a final pressure altitude of 56,000 ft in two seconds. Immediately before the decompression the vent of the pressurization system for the saline bottle was opened. It was closed again immediately after the decompression. The lumen of the intra-arterial needle, the polyethylene tubing between it and the taps and the lumen of the taps were flushed with arterial blood by turning the taps so that the needle was connected to the waste bottle. Actual sampling of the arterial blood was started one minute after decompression and continued for two minutes. Directly the sampling was finished the pressure within the decompression chamber was increased. During the descent the intra-arterial infusion of saline was recommenced and the taps removed from the syringe which was then capped and rotated slowly. The oxygen content of the arterial sample was determined by duplicate analyses in the manometric Van Slyke apparatus; the pH and carbon dioxide tension of the sample were determined by the interpolation technique of Astrup (1957). The oxygen-carrying capacity of the blood sample was also determined in duplicate analyses.

In this investigation each subject was usually decompressed twice in a single experiment. The exposures to pressure breathing were separated by at least twenty minutes. In order to reduce the risk of the development of decompression sickness, both the subject and the accompanying medical officer preoxygenated for one hour before the ascent to reduced pressure.

Results – Each of the four subjects was exposed on two separate occasions to each of the two breathing pressures at a pressure altitude of 56,000 ft. The oxygen saturation of each arterial sample was calculated from the oxygen content and capacity after making allowance for the physically dissolved oxygen. The oxygen saturation, pH and carbon dioxide tension of each sample of arterial blood, together with the corresponding positive breathing pressure, are presented in Table 5–10. There was some variation in the carbon dioxide tension and oxygen saturation of the arterial blood achieved between one subject and another even when the same pressure clothing assembly was used. The arterial carbon dioxide tension and arterial saturation were, however, always greater with a positive breathing pressure of 80 mmHg than with one of 60 mmHg.

DISCUSSION

Pulmonary Ventilation – A very striking feature of the response of subjects undergoing training in the use of pressure breathing equipment was the increase of the depth of breathing which was induced by pressure breathing, even when trunk counterpressure was employed (Ernsting, personal observation). Accurate measurement of the pulmonary ventilation during pressure breathing was virtually impossible, however, when standard pressure breathing equipment was used, since significant outboard leakages occurred from the breathing compartment of the mask or pressure helmet. It was necessary, therefore, to resort to the use of a modified pressure helmet fitted with a mouthpiece in order to ensure adequate collection of the expired gas. The use of a special breathing device incorporating a mouthpiece immediately

TABLE 5–10

THE EFFECT OF PRESSURE BREATHING AT A SIMULATED ALTITUDE
OF 56000 FT (PB 65.7 mmHg) UPON THE COMPOSITION OF THE
ARTERIAL BLOOD

	Positive Breathing Pressure (mmHg)	Arterial blood		
		Saturation ("o)	pH	Carbon dioxide tension (mmHg)
Control (breathing oxygen at simulated 25000 ft)				
A	---	100.0	7.405	40.0
A	---	99.8	7.410	38.0
B	--	99.6	7.395	39.5
B	----	100.0	7.399	39.0
E	--	99.2	7.405	42.5
E		99.5	7.415	41.0
F	----	100.0	7.425	38.0
F	---	100.0	7.420	38.5
Mean		99.8	7.409	39.6
Pressure breathing with mask, jerkin and anti-G suit				
A	63	83.5	7.476	30.2
A	61	83.5	7.495	28.5
B	63	85.5	7.510	26.6
B	60	82.5	7.502	28.3
E	63	91.0	7.514	26.5
E	61	83.2	7.490	29.4
F	60	82.0	7.449	31.0
F	60	83.2	7.495	29.2
Mean	61.4	84.3	7.491	28.7
Pressure breathing with helmet, jerkin and anti-G suit				
A	77	88.8	7.435	34.5
A	80	92.0	7.510	29.5
B	77	93.0	7.495	30.2
B	80	92.2	7.492	30.6
E	79	93.2	7.505	29.0
E	80	91.0	7.452	32.5
F	80	92.8	7.449	31.5
F	78	89.2	7.435	34.5
Mean	78.9	91.5	7.471	31.5

restricted the group of subjects which could be used for this study. Attempts to make measurements of pulmonary ventilation using this apparatus in aircrew who had completed their training in pressure breathing were totally unsuccessful. This group of potential subjects either refused to don the helmet fitted with the mouthpiece or had an abnormally high pulmonary ventilation when wearing the apparatus in the resting state. Shortage of time precluded a long period of familiarization with the equipment. Thus these studies of the effect of pressure breathing upon the pulmonary ventilation were performed upon a small group of subjects who had previous experience of respiratory experiments and who had been exposed to pressure breathing on numerous occasions.

In the selected group of trained individuals employed in the present study, pressure breathing with trunk counterpressure caused an increase in the pulmonary ventilation (Table 5-4). The greatest increase occurred at the beginning of pressure breathing and although the pulmonary ventilation was raised throughout the pressure breathing period, the ventilation declined slightly as the procedure was continued (Figs. 5-1, 5-2 and 5-3). The increase in pulmonary ventilation was due principally to an increase in the tidal volume, although the respiratory rate was also increased slightly. The magnitude of the increase of the pulmonary ventilation in a given subject was not related to his experience of pressure breathing. The increase in the carbon dioxide output in the absence of a concomitant rise of the oxygen uptake suggested that the increase of pulmonary ventilation was a true hyperventilation. This suggestion was confirmed by the increase in the respiratory exchange ratio which occurred during pressure breathing (Table 5-4). Further evidence in favour of the occurrence of hyperventilation was that the pulmonary ventilation during the early part of the recovery period was less than the control value and that the respiratory exchange ratio also fell below the control value during the recovery period. Similar evidence suggested that the increase of pulmonary ventilation produced by breathing at a positive pressure of 30 mmHg without respiratory counterpressure was also a true hyperventilation.

The pulmonary ventilation declined progressively during the exposure to pressure breathing. The duration of an exposure was limited, however, by the cardiovascular disturbances which are associated with pressure breathing at these relatively high pressures. It was not possible, therefore, to ascertain whether the pulmonary ventilation would attain a steady value during pressure breathing under these circumstances. The complete absence of a respiratory "steady state" during pressure breathing was evidenced not only by the changing pulmonary ventilation but also by the raised respiratory exchange ratio. The rate of change of pulmonary ventilation became less, however, as the time of exposure to pressure breathing lengthened. The collection of expired gas for the measurement of oxygen and carbon dioxide exchange was therefore performed as late as possible in the pressure breathing period. Thus, in the measurements performed during pressure breathing at positive pressures of 60 and 80 mmHg the expired gas collection was started at the beginning of the second or third minute of the exposure whilst during breathing at a positive pressure of 30 mmHg without counterpressure, the collection was started in the fourth minute.

The cause of the increase of pulmonary ventilation during pressure breathing is obscure. There was no significant increase in the oxygen uptake when counterpressure was employed so that the raised pulmonary ventilation was not due to an increase in the metabolic oxygen consumption. The small increase of oxygen uptake found during breathing at a positive pressure of 30 mmHg without counterpressure was probably due to the increased work performed by the respiratory muscles in this situation. Although pressure breathing with an oronasal mask produced a large increase in the volume of the upper respiratory airways (Chapter 3) the pressure helmet which was used during this measurement of pulmonary ventilation applied counterpressure to the face and neck so that this cause of an increase in the respiratory dead space was eliminated. This was confirmed by the direct measurements of the anatomical dead space made during pressure breathing with this helmet. Although an increase in the physiological dead space might have been one of the factors which gave rise to the increase of pulmonary ventilation, the observed increase in the respiratory exchange ratio during pressure breathing showed that in fact the increase in pulmonary ventilation more than compensated for any increase of dead space which might have occurred in this situation. This observation was confirmed by the finding that pressure breathing under these circumstances also reduced the alveolar and arterial carbon dioxide tensions. This latter finding in fact eliminated an increase of respiratory dead space as a cause of the increase of pulmonary ventilation since an increase in dead space increases the pulmonary ventilation by reducing the alveolar ventilation and raising the alveolar carbon dioxide tension. The increase in pulmonary ventilation produced by breathing at a positive pressure of 30 mmHg without counterpressure was probably associated with the gross alteration of the mode of operation of the respiratory muscles which occurred in this situation.

Evidence for the impaired nervous co-ordination of the respiratory muscles in pressure breathing without counterpressure was presented in the previous chapter. It was also shown that the mechanics of respiration were not restored completely to normal by the application of the counterpressure afforded by the pressure jerkin. The slight distension of the lungs which occurred during pressure breathing with trunk counterpressure might have been a stimulus to pulmonary ventilation. Furthermore, high pressure breathing caused marked changes in the pattern of the afferent nervous impulses passing to the central nervous system as evidenced by subjective sensation. The inflated helmet and pressure jerkin gave sensations of tightness around the head and trunk respectively. In addition, discomfort due to vascular congestion occurred in the upper limbs, particularly during breathing at a positive pressure of 80 mmHg. These forms of sensory stimulation commonly give rise to an increase in pulmonary ventilation in the absence of pressure breathing. It may be concluded, tentatively, therefore, that these sensory stimuli are also responsible for the hyperventilation produced by high pressure breathing with trunk counterpressure.

In the steady state the uptake of oxygen from the respired gases reflects the metabolic oxygen consumption. Temporary differences can arise, however, between these two rates when the size of the oxygen store of the body is changing, as during an alteration of pulmonary ventilation, which induces a

uncertain. The ability to analyze a nitrogen clearance curve into two components, each of which appears to be evenly ventilated, does not imply that in fact the lungs consist of two clearly defined volumes, each with its own ventilation. Indeed, the accepted concept of the ventilation of the lung is that there is a continuum of different degrees of ventilation amongst the various alveoli. Further, as Briscoe and Cournand 1959 (47) have pointed out, a washout curve which can be analyzed into two components, can, in fact, be the result of the summation of a considerably greater number of components. The accuracy of the technique is partly determined by the possibility of detecting departure from linearity when the analysis of the components is made by the semi-logarithmic plot of the experimental data. The method of analysis proposed by Fowler, Cornish and Kety 1952 (112) does, however, allow a quantitative assessment of the unevenness of the distribution of the inspired gas so that the evenness of ventilation may be compared in different experimental conditions and between one subject and another.

The results of the analysis of the nitrogen clearance curves obtained from the subjects at rest (Table 5-5) showed that the compartment with the slower clearance rate constituted about 75"ₙ of the functional residual capacity and that it received about half of the total alveolar ventilation. The other compartment, although it constituted only 25"ₙ of the functional residual capacity, received the other half of the alveolar ventilation. These results agree well with those obtained by Fowler, Cornish and Kety 1952 (112). The increases in functional residual capacity caused by pressure breathing were very similar to those presented in the previous chapter. The proportions of the functional residual capacity occupied by the two compartments remained virtually unchanged in pressure breathing, even when the functional residual capacity was markedly increased by pressure breathing without respiratory counterpressure. A similar constancy of the proportions of the end-expiratory lung volume occupied by each of the compartments was demonstrated by Bates, Fowler, Forster and Van Lingen 1954 (26). The subjects in this investigation voluntarily maintained various increases of the end-expiratory volume. Haab and Cimono 1960 (134) obtained a similar result in a study of the distribution of the inspired gas during pressure breathing at positive pressures of 11 and 18 mmHg without respiratory counterpressure.

The alveolar dilution ratios obtained in the present study (Table 5-5) showed that the ventilation of each of the two compartments of the lung volume was not significantly changed by pressure breathing. Further, the proportion of the alveolar ventilation distributed to each compartment remained unchanged during pressure breathing, whether or not trunk counterpressure was used. Bates, Fowler, Forster and Van Lingen 1954 (26), whose subjects varied their end-expiratory lung capacity by voluntary effort, found that the alveolar dilution ratio was slightly increased by an increase of the functional residual capacity. In their experiments, however, the tidal volume, and hence the alveolar ventilation per breath, was held constant in spite of the change of lung volume. In the present experiments the total alveolar ventilation per breath was increased at the larger lung volumes and this tended to reduce the increase of the alveolar dilution ratios which would otherwise have occurred. Haab and Cimono 1960 (134) also found no change in the alveolar dilution ratios during positive pressure breathing.

change in the alveolar oxygen tension. During the period over which expired gas was collected in the pressure breathing experiments, the pulmonary ventilation was changing relatively slowly. Further, the direct measurements of alveolar gas tensions confirmed that the alveolar oxygen tension altered very little over this period. When no respiratory counterpressure was used only two subjects could maintain the regular breathing pattern necessary if significant changes of the quantity of oxygen stored in the body were to be avoided. It appeared, therefore, that in the present experiments the oxygen uptake measured by analysis of the respired gases, reflected the metabolic oxygen consumption. Breathing at a positive pressure of 30 mmHg without respiratory counterpressure caused a mean increase of 21 ml S.T.P./min. in the oxygen uptake (Table 5-4). This increase, which was statistically significant, was probably associated with the large increase in the work performed by the respiratory muscles in these circumstances (Chapter 4). No significant change of oxygen uptake could be detected when trunk counterpressure was used in pressure breathing (Table 5-4). The actual increase in work performed by the respiratory muscles in these circumstances was small (Chapter 4) and calculations suggested that the associated increase in the metabolic oxygen consumption would be of the order of 2 to 5 ml per minute, a change which could not be detected.

Distribution of inspired gas – The manner in which the inspired gas is distributed within the lungs is one of the factors which determines the efficiency of the gaseous exchange between the gaseous and blood phases in the lungs. The presence of uneven ventilation of the lungs in normal subjects was demonstrated early in this century by Krogh and Lindhard 1917 (175), Darling, Cournand and Richards 1944 (71) found in several healthy subjects that pulmonary nitrogen clearance did not follow the course predicted for uniform ventilation. In 1952 Fowler, Cornish and Kety (112) published a method of analysis of nitrogen clearance curves which gave a quantitative expression for the degree of unevenness of alveolar ventilation. They showed that in most normal subjects there was some unevenness of ventilation.

The method used in the present study to determine the effect of pressure breathing upon the distribution of inspired gas within the lungs was essentially that developed by Fowler, Cornish and Kety 1952 (112). Their method of analysis of nitrogen clearance curves was, however, modified in certain respects. The original analysis was based upon the mean concentration of nitrogen for each expiration. The calculation of this quantity was very laborious. A preliminary study of the change of the concentration of nitrogen during expiration over the "plateau" of nitrogen concentration showed that this never exceeded 1.5%. It was concluded, therefore, that it was acceptable to use the end-tidal concentration of nitrogen in plotting the nitrogen clearance curves. The use of the end-tidal nitrogen concentration required a modification of the analysis propounded by Fowler, Cornish and Kety 1952 (112). Such a modification has been presented by Briscoe and Cournand 1959 (47) who followed the end-tidal gas concentration using a Rahn sampler. No correction was applied in the present analysis for the contribution of the tissue and blood nitrogen to the expired nitrogen since the quantity of nitrogen coming from this source was relatively small and

Both Bates, Fowler, Forster and Van Lingen 1954 (26) and Haab and Cimono 1960 (134) found that the distribution of the alveolar ventilation between the two compartments was not affected by an increase in the lung volume.

The present investigation of the washout of nitrogen from the lungs demonstrated, therefore, that pressure breathing produced no detectable change in the distribution of the inspired gas within the lungs. There are limitations to the sensitivity of this method of detecting unevenness of pulmonary ventilation. It is, however, the most sensitive method available for assessing the evenness of the distribution of the inspired gas per se apart from those methods employing the detection of the radiation from inspired radio-active gases by means of scintillation counters placed at various positions over the chest wall (85). The distribution of the inspired gas to the various groups of alveoli within the lungs is not of itself an important factor controlling the gaseous exchange between the gas and blood phases in the lungs. The important factor is the relation of the distribution of ventilation to the distribution of pulmonary capillary blood flow. The nitrogen clearance measurements, however, give no information with regard to this relationship unless some assumptions are made concerning the distribution of blood flow through the lungs. Briscoe 1959 (46) has presented a method whereby the contribution of the unevenness of the distribution of the inspired gas to the oxygen tension difference between the alveolar gas and the systemic arterial blood may be assessed. Such calculations have little application, however, to the present investigation. The absence of any change in the degree of unevenness of alveolar ventilation with the induction of pressure breathing would suggest that any impairment of the exchange between the gas and blood phases within the lungs demonstrated in pressure breathing must be due to changes in the distribution of pulmonary capillary blood flow or to changes in the diffusion characteristics of the alveolar capillary membrane.

Respiratory Dead Space -- Considerable controversy has centred around the interpretation of the various measures of the respiratory dead space. It is possible, however, to distinguish clearly two distinct forms of respiratory dead space, namely the anatomical and the physiological dead space. The anatomical dead space is the volume of the conducting airways down to the region in the lungs where the inspired gas is diluted by the alveolar gas. It is, therefore, that volume of the tidal air which does not contribute to the ventilation of the alveoli. It is measured by recording simultaneously the instantaneous concentration of either nitrogen after a single breath of oxygen or carbon dioxide at the lips and the expiratory flow (118). By an extrapolation of the alveolar concentration plateau the concentration of gas in the alveolar air which washed out the dead space is calculated and the volume of this dead space is estimated using the Bohr equation. In the present study the anatomical dead space was measured by the single breath of oxygen technique developed by Fowler 1948 (118). The physiological dead space, on the other hand, is a measure of the effectiveness with which the inspired gas removes carbon dioxide from (and adds oxygen to) the blood flowing through the lungs. It is measured by determining simultaneously the carbon dioxide tension of the expired gas and of the systemic arterial blood Riley and Cournand 1949 (244).

The rate of diffusion of carbon dioxide is so high that in a normal subject the tension of this gas in the blood leaving an alveolus virtually always equals that in the gas within the alveolus. Further, the carbon dioxide dissociation curve is almost linear over the physiological range so that the carbon dioxide tension of the blood leaving the lungs represents the mean of the carbon dioxide tensions in all the alveoli weighted in proportion to the capillary blood flow through each alveolus. Since the difference between the mixed venous and arterial carbon dioxide tensions is relatively small the normal right to left anatomical shunt of blood does not significantly change the carbon dioxide tension of the blood flowing from the pulmonary capillary bed. The systemic arterial carbon dioxide tension may be used, therefore, as a measure of the mean alveolar carbon dioxide tension. The dead space volume calculated using this measure of the alveolar carbon dioxide tension is the volume of the tidal gas which does not remove carbon dioxide from the blood flowing through the alveolar capillaries. The difference between the physiological dead space and the anatomical dead space, which is termed the alveolar dead space, is, therefore, an expression of the proportion of and the degree to which certain alveoli are under-perfused with blood in relation to the ventilation which they receive. If the ventilated alveoli are divided into two groups, one group of which are not perfused, the other group of which are evenly perfused in relation to their ventilation, then the proportion of alveoli not perfused is given by the ratio of the alveolar dead space volume to the alveolar tidal volume (262).

The values of the volume of the anatomical dead space obtained with the subjects in the resting state (Table 5–11) were very similar to the mean value of 156 ml (S.D. ±28 ml) reported by Fowler 1948 (111) for a group of forty-five normal male subjects. Pressure breathing at a positive pressure of 20 mmHg with no respiratory counterpressure caused a mean increase of the anatomical dead space of 31 ml B.T.P.S. which was highly significant. The magnitude of this increase of the anatomical dead space was greatly reduced by the application of counterpressure to the trunk. Thus when counterpressure was employed breathing at a positive pressure of 30 mmHg only increased the anatomical dead space by 10 ml B.T.P.S. Even pressure breathing at a positive pressure of 60 mmHg with trunk counterpressure only induced a mean rise of 13 ml B.T.P.S. in the volume of the anatomical dead space. In all these experiments the bladder of the pressure helmet applied counterpressure to the head and neck. This counterpressure was probably adequate to prevent any significant distension of the extrathoracic portion of the upper respiratory tract. The measurements of the anatomical dead space during pressure breathing with trunk counterpressure certainly did not show the very large increase of dead space which was produced by pressure breathing with an oronasal mask (Chapter 3). There was a small gap (2 to 4 cm in width) between the lower border of the neck bladder of the pressure helmet and the upper border of the pressure jerkin. The neck bladder did, however, apply counterpressure to the neck to within 2 cm of the sternal notch so that all the extrathoracic portion of the upper respiratory tract, with the exception perhaps of a 2–3 cm length of the trachea, was adequately supported during pressure breathing. Care was taken in these experiments to avoid any alteration in the posture of the head and the position of the lower jaw when pressure

TABLE 5-11

THE ALVEOLAR DEAD SPACE IN PRESSURE BREATHING — MEAN
VALUES FOR DUPLICATE EXPERIMENTS ON THREE SUBJECTS

Condition		A. Control	B. Pr. Br. (a 20 mmHg	C. Pr. Br. (a 30 mmHg[1]	D. Pr. Br. (a 60 mmHg[1]
Tidal volume (ml B.T.P.S.)		654	779	728	813
Anatomical dead space (mlB.T.P.S.)		160	191	170	173
Physiological dead space (ml B.T.P.S.)		201	284	28?	332
Alveolar dead space	Mean	40	94	118	159
(ml B.T.P.S.)	S.E.	± 10	± 16	± 16	± 16
Ratio of alveolar dead	Mean	0.08	0.16	0.21	0.25
space to alveolar tidal volume	S.E.	± 0.02	± 0.03	± 0.04	± 0.04

[1] Pressure breathing with trunk counterpressure

breathing was induced since Nunn, Campbell and Peckett (1959) (227) demonstrated that such alterations of posture produced considerable changes in the volume of the extrathoracic dead space. It may be concluded, therefore, that there was no, or at the very most, only a very small increase in the volume of the extrathoracic portion of the upper respiratory tract in the course of these experiments.

The observed differences in the increase of the anatomical dead space induced by pressure breathing with and without respiratory counterpressure were due to variations in the volume of the intrathoracic portion of the anatomical dead space. As has been discussed in Chapter 3 the volume of the anatomical dead space is determined by a number of factors, of which the volume of oxygen inspired, the duration of the delay before expiration and the expiratory flow pattern were carefully controlled in the present experiments. Fowler 1948 (111) demonstrated that the anatomical dead space was increased by an increase of the end-expiratory lung volume and the relationship between these two variables was investigated in detail in one subject by Shepard, Campbell, Martin and Enns 1957 (267). They found that there was an approximately linear relationship between the volume of the anatomical dead space and the end inspiratory lung volume, the increase of anatomical dead space being 12.5 ml B.T.P.S. per litre of increase of inspiratory lung volume. The experiments described in Chapter 4 showed that breathing at a positive pressure of 20 mmHg without respiratory counterpressure induced a mean increase of 3.0 litre B.T.P.S. in the end-inspiratory volume.

Thus on the basis of the results obtained by Shepard, Campbell, Martin and Enns 1957 (267) the lung distension induced by breathing at a positive pressure of 20 mmHg would have increased the anatomical dead space volume by 37.5 ml B.T.P.S. The magnitude of this predicted increase may be compared with the mean increase of 31 ml B.T.P.S. found in the present experiments (Table 5-11). Further, the increase of dead space volume induced by breathing at a positive pressure of 60 mmHg with trunk counterpressure predicted from the results of Shepard, Campbell, Martin and Enns 1957 (267) was 10 ml B.T.P.S. which may be compared with the mean increase of 13 ml B.T.P.S. obtained experimentally. The observed increase of the intrathoracic portion of the anatomical dead space induced by pressure breathing was due, therefore, to the concomitant increase in the volume of the lungs.

The variability of the values of the physiological dead space obtained in each of the subjects in the resting state (Table 5-6) was relatively small and the mean values obtained in this series are similar to that reported by Asmussen and Nielsen 1956 (10) and Gray, Grodins and Carter 1956 (126). The mean of the ratios of the physiological dead space to the tidal volume which amounted to 0.29 (S.D. ±0.05) was close to the mean value of 0.31 (S.D. ±0.06) reported by Larson and Severinghaus 1962. The volume of the physiological dead space in the resting subject was considerably greater than the corresponding value for the anatomical dead space, the mean alveolar dead space amounting to 40 ml (S.E. ±10 ml) B.T.P.S. Larson and Severinghaus 1962 (184) reported a mean value of 41 ml (S.D. ±40 ml) B.T.P.S. for the alveolar dead space in a group of eleven subjects under the same

conditions as those employed in the present study. The ratio of the alveolar dead space to the alveolar tidal volume which indicates the proportion of ventilated alveoli which are not perfused, was 0.08 (S.E. ±0.02).

Several investigators have provided evidence that gravitational forces may influence the distribution of the pulmonary blood flow within the lungs. Thus the bronchospirometric studies of Mattson and Carlens 1955 (205) showed that the oxygen uptake in the dependent lobes of the lungs was greater than in the upper and that this relationship could be reversed by reversing the gravitational relationship of the parts. They concluded that the hydrostatic forces acting upon the pulmonary circulation increased the blood flow through the lower parts at the expense of the upper. Riley, Permutt, Said, Godfrey, Cheng, Howell and Shepard 1959 (246) concluded from the increase in the physiological dead space which occurred on standing erect from the supine position that in the upright position approximately one seventh of the total alveoli were not perfused. More recently the elegant studies of West and Dollery 1960 (285) in which the clearance rate of radio-active carbon dioxide from various regions of the lungs was measured with externally placed counters, demonstrated that in the erect posture the pulmonary capillary blood flow decreases in a linear manner from the base to the apex of the lungs where it is virtually zero. Thus the alveolar dead space detected in the resting subjects in the present investigation was due to the unevenness of the distribution of the pulmonary blood flow in the lungs in the seated position.

Pressure breathing, both with and without trunk counterpressure, caused a marked increase in the volume of the physiological dead space (Table 5–11). The increase varied with the breathing pressure. A proportion of the increase of the physiological dead space was due to the concomitant enlargement of the anatomical dead space. The rise in the volume of the physiological dead space was, however, considerably greater than that of the anatomical dead space so that in the present investigation pressure breathing always induced an increase of the alveolar dead space (Table 5–11). The ratio of the alveolar dead space volume to the alveolar tidal volume was increased during pressure breathing, particularly at a positive pressure of 60 mmHg. Thus the proportion of the non-perfused ventilated alveoli was markedly increased by this manoeuvre. This change in the alveolar dead space could have been due to one of several different factors. The physiological dead space as defined in this context is increased by an increase in the tidal volume. Thus Asmussen and Nielsen 1956 (10) found that the physiological dead space varied directly with the tidal volume when the pulmonary ventilation was increased by exercise. Further, Gray, Grodins and Carter 1956 (126) showed that this relationship also held when the pulmonary ventilation was increased by adding carbon dioxide to the inspired gas. The increase in the volume of the physiological dead space to be expected from the observed increase in tidal volume induced by pressure breathing was, however, less than 10% of the measured increase.

Severinghaus and Stupfel 1957 (262) determined in the anaesthetized dog the effect of changes of the end-expiratory lung volume upon the physiological dead space. They found that when the tidal volume was kept constant the volume of the physiological dead space was independent of the

end-expiratory lung volume. Since the anatomical dead space increased as the lung volume was raised the alveolar dead space volume actually decreased as the lung volume was raised. In the present experiments the increase in physiological dead space persisted during pressure breathing when the lung distension normally induced by this procedure was greatly reduced by the application of respiratory counterpressure. Further, in the limited number of conditions studied the magnitude of the increase of physiological dead space, and more particularly of the alveolar dead space, appeared to be related more closely to the breathing pressure than to the degree of lung distension which pressure breathing produced. Thus pressure breathing induced a very significant increase of the alveolar dead space which appeared to be independent of any change in the mechanics of respiration which accompanied this manoeuvre. Folkow and Pappenheimer 1955 (108), using their iso-saturation technique, demonstrated that pressure breathing at 15 mmHg with an oronasal mask alone caused an increase of both the series dead space (equivalent to the anatomical dead space) and of the parallel dead space (equivalent to alveolar dead space).

These findings with regard to the increase of the volumes of the physiological dead space and of the alveolar dead space caused by pressure breathing could only be explained by postulating an increase of the proportion of the ventilated alveoli which were not perfused by blood. Since the analysis of the nitrogen clearance curves obtained during pressure breathing showed no evidence of any alteration in the distribution of inspired gas within the lungs this increase in the proportion of non-perfused alveoli was due primarily to changes in the pulmonary circulation. The rise of intrathoracic pressure associated with pressure breathing produces profound changes in the cardiovascular system. At the beginning of pressure breathing there is a displacement of the blood from the thoracic and abdominal viscera into the periphery. This is accompanied by a fall of the effective pressure in the right atrium (Chapter 6), and by a reduction of the cardiac output (53) (231).

It is very probable, therefore, that the pressure in the pulmonary artery measured relative to the intra-alveolar pressure is reduced by pressure breathing as is the systemic blood pressure in relation to the intra-alveolar pressure (Chapter 6). Thus the transmural pressures of the pulmonary capillaries throughout the lung are lowered by this manoeuvre. In the seated posture, therefore, the proportion of the apical alveoli which are not perfused during the cardiac cycle is increased, since the flow of blood through the capillaries of an alveolus depends upon the pressure within the capillaries exceeding that in the alveolus. The sensitivity of the distribution of the pulmonary capillary blood flow within the lungs to changes in cardiac output is illustrated by the very mild degree of exercise required to abolish the uneven distribution of pulmonary blood flow seen in the resting erect subject (West and Dollery, 1960) (285). Thus it is suggested that the reduction of cardiac output and fall of effective pulmonary artery pressure induced by pressure breathing reduce the perfusion of the apices of the lungs and this causes the observed increase in the volume of the alveolar dead space.

Since pressure breathing enlarges the physiological dead space the total pulmonary ventilation must be increased during this manoeuvre if the same alveolar ventilation is to be maintained during pressure breathing as at rest.

The metabolic oxygen uptake is virtually unchanged by pressure breathing so that the maintenance of an alveolar ventilation equivalent to that at rest would ensure that the alveolar gas tensions remain unchanged. With the observed increase of the physiological dead space during breathing without respiratory counterpressure at a positive pressure of 20 mmHg, an increase of about 1.3 litre/min. B.T.P.S. in the pulmonary ventilation would be necessary at a respiratory frequency of fourteen per minute to maintain a normal alveolar ventilation. The corresponding increase in pulmonary ventilation required during breathing at a positive pressure of 60 mmHg with trunk counterpressure would be approximately 1.9 litre/min. B.T.P.S. If an oronasal mask were used in place of a pressure helmet during pressure breathing with trunk counterpressure at 60 mmHg the total increase in physiological dead space would be about 0.3 litres B.T.P S. so that the pulmonary ventilation would have to be increased by over 4 litre/min. B.T.P.S. if the alveolar ventilation was not to be reduced below normal.

Alveolar Gas Tensions – The Haldane-Priestley 1905 (137) technique of direct sampling of the alveolar gas by the performance of a fast, deep expiration has been subjected to some criticism of recent years. It has been claimed (Rahn, 1949) (240) that it yields values for carbon dioxide tension which are slightly higher than the true mean alveolar tension of this gas. As Bannister, Cunningham, and Douglas 1954 (14) have pointed out, however, the sample must be delivered very rapidly and Bannister *et al* 1954 (14) quote experimental evidence in support of the conclusion that there is no significant difference between the carbon dioxide tension given by the end-expiratory Haldane-Priestley sample and that of the arterial blood sampled under the same conditions. In the present investigation, therefore, the end-expiratory Haldane-Priestley sample was used to follow the composition of the alveolar gas. The four subjects used for this part of the study were experienced in the performance of this technique. This method of sampling the alveolar gas has the disadvantage, however, that the breathing pattern is interrupted whenever a sample is given. The gas leaving the respiratory tract at the end of a normal expiration has been taken as being representative of the alveolar gas by numerous investigators. End-tidal sampling using the Rahn-Otis 1949 (242) apparatus has been used extensively and several comparisons have been made between the end-expiratory carbon dioxide tension given by this method and the carbon dioxide tension of the arterial blood determined simultaneously (273) (259).

These comparisons have shown that there is no significant difference between simultaneously measured end-tidal and arterial carbon dioxide tensions. Lambertsen and his colleagues (Lambertsen, Smyth, Semple and Gelfand 1958 (179); Lambertsen and Benjamin 1959 (178) have performed extensive comparisons between the end-expiratory carbon dioxide tension and the simultaneous arterial carbon dioxide tension. They found excellent agreement between these two measurements when the tidal volume exceeded 0.6 litre. When, however, the tidal volume was as low as 0.36 litres they found that the end-tidal carbon dioxide tension was 2.9 mmHg less than that of the arterial blood. This discrepancy was due to the failure of the small tidal volume to wash out the dead space during expiration. Thus the carbon dioxide tension of the gas leaving the respiratory tract at the end of expiration

is equal to the arterial carbon dioxide tension, provided that the tidal volume is at least o.6 litre. This requirement was fulfilled in virtually all the present experiments. The agreement between the values of carbon dioxide tension obtained by end-expiratory Haldane-Priestley sampling and by the determination of the end-tidal carbon dioxide tension from a continuous record which was found in the present series of experiments confirmed the value of both these techniques as a measure of the alveolar carbon dioxide tension.

The reduction of the alveolar carbon dioxide tension induced by pressure breathing confirmed the conclusions drawn from the measurements of the respiratory gas exchange discussed earlier in this chapter. Although there was a considerable variation in the response of different subjects to the same intensity of pressure breathing this manoeuvre always produced a reduction of the alveolar carbon dioxide tension. Since in these experiments there was no change in the absolute pressure within the respiratory tract with the induction of pressure breathing, the changes observed must have been due to the effect of pressure breathing upon the gaseous exchange between the blood and the environment. As has been seen the carbon dioxide output in the expired gas was actually increased during pressure breathing so that the fall of alveolar carbon dioxide tension must have been due to an overall hyperventilation of the lungs. Further evidence for this increase of alveolar ventilation in pressure breathing was obtained by calculating the alveolar respiratory exchange ratio for each of the discrete samples of alveolar gas.

The alveolar respiratory exchange ratio was calculated from the tensions of carbon dioxide and oxygen in each Haldane-Priestley sample using the following form of alveolar air equation (103) (245):

$$R = \frac{P_{ACO_2}(1 - F_{IO_2})}{P_{IO_2} - P_{AO_2} - F_{IO_2} \times P_{ACO_2}}$$

R = respiratory exchange ratio

F_{IO_2} = fractional concentration of oxygen in the inspired gas

P_{IO_2} = partial pressure of oxygen in the moist inspired gas

P_{AO_2} = partial pressure of oxygen in the alveolar gas

P_{ACO_2} = partial pressure of carbon dioxide in the alveolar gas.

The time course of the mean alveolar respiratory exchange ratio in each of the three pressure breathing conditions investigated is presented in Table 5-12. The alveolar exchange ratio was increased above the resting value during pressure breathing, the greatest increase occurring at the beginning of the exposure. The ratio declined slowly during the exposure, but it did not regain the control value.

Immediately after the cessation of pressure breathing the alveolar exchange ratio fell below the control value and then increased towards the resting level. The changes of the alveolar exchange ratio offered further evidence, therefore, that pressure breathing induced a true hyperventilation with removal of carbon dioxide from the body stores of this gas. These results demonstrated in greater detail the course of this hyperventilation than did the mean values of the expired respiratory exchange ratio obtained during the study of the total gaseous exchange in pressure breathing. Thus, although during the exposure to pressure breathing the pulmonary ventilation generally declined after the initial increase the alveolar respiratory exchange ratio declined only slowly

TABLE 5-12

THE ALVEOLAR RESPIRATORY EXCHANGE RATIO
IN PRESSURE BREATHING AT GROUND LEVEL

Positive breathing pressure	Control Time (min.)	Mean alveolar respiratory exchange ratio							
		Pressure breathing				Recovery			
		0-1	1-2	2-3	3-4	0-1	1-2	2-3	
30 mmHg without counterpressure	0.83	1.21	1.11	1.08	1.03	0.65	0.70	0.73	
50 mmHg with trunk counterpressure	0.82	0.97	0.94	0.94	0.89	0.72	0.75	0.78	
80 mmHg with trunk counterpressure	0.83	1.12	1.08	1.04	0.98	0.69	0.73	0.76	

TABLE 5-13

THE APPARENT DIFFUSING CAPACITY MEASURED BY THE BREATH-
HOLDING TECHNIQUE IN SUBJECTS SEATED AT REST BREATHING AIR

No. of Subjects	Apparent diffusing capacity (ml min. mmHg)		Reference
	Mean	Standard error	
8	32.1	± 0.84	McNeill, Rankin & Forster (1958) (225)
9	33.5	± 3.09	Apthorpe & Marshall (1961) (6)
11	27.1	± 2.68	Ogilvie, Forster, Blakemore & Morton (1957) (228)
14	30.0	± 1.56	Cadigan, Marks, Ellicott, Jones & Gaensler (1961) (52)
7	30.4	± 3.3	Roughton & Forster (1957) (254)

and did not return to the control value during the pressure breathing period
in any of the experiments. Throughout the pressure breathing period, there-
fore, the sizes of the various gas stores of the body were undergoing continual
change. The studies of the changes in the gas stores during hyperventilation
performed by Farhi and Rahn 1955 (100) suggests that most of the increase
in the respiratory exchange ratio during pressure breathing was due to
.emoval of carbon dioxide from the body store.

Pressure breathing without counterpressure at a positive pressure of
30 mmHg produced the greatest reduction of the alveolar carbon dioxide
tension of the three conditions of pressure breathing studied in this investiga-
tion. Breathing at positive pressures of 50 and 80 mmHg with trunk counter-
pressure reduced the alveolar carbon dioxide tension to a small degree, the
magnitude of the reduction being proportional to the breathing pressure.
These findings confirmed the conclusions drawn earlier in this discussion that,
even with the full trunk counterpressure afforded by the pressure jerkin,
pressure breathing induced a true hyperventilation in subjects who had con-
siderable experience of this manoeuvre. The reduction of the arterial carbon
dioxide tension produced by pressure breathing was of significance since
hypocapnia induces important changes in the cardiovascular system, such as
the redistribution of the systemic blood flow and in the central nervous system.
These effects are discussed later in this chapter and in Chapter 6.

Diffusing Capacity of the Lungs – The measurement of the apparent
diffusing capacity of the lungs by the breath-holding technique originally
developed by Krogh and Krogh 1910 (174) and by Krogh 1915 (176) and
modified by Forster, Fowler, Bates and Van Lingen 1954 (109) has been
used by many investigators to study the effects of various environmental
changes and disease processes upon the exchange of gas between the alveolar
gas and the blood flowing through the alveolar capillaries. This method
using carbon monoxide was chosen in the present study since the duration of
the actual experimental determination is relatively short; it does not require
a steady state of respiratory gas exchange and it may be performed in the
resting subject. The values obtained for the pulmonary diffusing capacity of
resting healthy subjects do, however, vary from one laboratory to another.
This difficulty appears to be related primarily to the deficiencies of present
methods of determining the absolute concentration of carbon monoxide in
respiratory gas mixtures (68).

In the present study repeated checks of the linearity of the infra-red carbon
monoxide analyzer were made. Further, in this investigation direct com-
parisons were made between the diffusing capacity under various environ-
mental conditions in the same subject. Errors in the estimation of the
absolute as opposed to the relative concentration of carbon monoxide, would
have, however, resulted in errors in the estimated values for the diffusing
capacity of the pulmonary membrane and the pulmonary capillary blood
volume. The mean value of the pulmonary-diffusing capacity of the resting
subjects at an alveolar oxygen tension of 95 to 115 mmHg obtained in the
present study was 31.7 ml per minute per mmHg. It may be seen from
Table 5–13 that this mean value agrees well with the values obtained in
similar circumstances by other investigators using this technique. Thus it
would appear that the details of the procedure employed for the measurement

of the pulmonary diffusing capacity in the present study were reasonably satisfactory. The reproducibility of the measurement in a given subject under the same conditions was also acceptable.

The results of the measurements of the apparent diffusing capacity performed upon three subjects demonstrated that pressure breathing caused a consistent reduction of the diffusing capacity at a given alveolar oxygen tension (Table 5-7). Ogilvie, Forster, Blakemore and Morton 1957 (228) found that a rise in the intrapulmonary pressure during the period of breath-hold reduced the apparent diffusing capacity in two out of three subjects. The maximum reduction of diffusing capacity of 17"„ was produced by a positive intrapulmonary pressure of 44 mmHg. A tendency for the diffusing capacity to be reduced by continuous pressure breathing at 15 mmHg was reported by Cadigan, Marks, Ellicott, Jones and Gaensler 1961 (52) although in their experiments the alveolar volume during the determination of the diffusing capacity was greatly increased by pressure breathing. In the present experiments where the diffusing capacity was not measured until the subject had been pressure breathing for at least one minute and the alveolar volume was virtually constant it was found that the effects of pressure breathing were more consistent and more profound.

The various factors which can influence the magnitude of the apparent diffusing capacity as measured by the breath-holding technique have been investigated in detail by Ogilvie, Forster, Blakemore and Morton 1957 (228), Forster, Roughton, Cander, Briscoe and Kreuzer 1957 (110) and Cadigan, Marks, Ellicott, Jones and Gaensler 1961 (52). Krogh 1915 (176) using her original technique reported that the diffusing capacity of the lungs increased in proportion to the alveolar volume above the mid-capacity. Although Ogilvie, Forster, Blakemore and Morton 1957 (228) ·vere unable to demonstrate this effect, Marks, Cugell, Cadigan and Gaensler 1957 (201) confirmed Krogh's original observations. Cadigan, Marks, Ellicott, Jones and Gaensler 1961 (52) studied the effect of variations of alveolar volume in a large group of subjects and found that the diffusing capacity was related linearly to alveolar volume over the range from functional residual capacity to almost full lung capacity. At very high volumes the diffusing capacity was increased disproportionately. The regression coefficient of diffusing capacity upon lung volume was approximately 4.2 units of diffusing capacity per litre of alveolar volume.

Inspection of Table 5-8 shows that the alveolar volume was generally slightly greater during the measurement of the diffusing capacity in pressure breathing than when the measurement was made at rest. The largest increase in the alveolar volume during breath-holding was associated with a positive breathing pressure of 80 mmHg. The mean increase in this situation amounted to 0.51 litre B.T.P.S. On the basis of the data of Cadigan, Marks, Ellicott, Jones and Gaensler 1961 (52) this increase of alveolar volume of itself would have increased the apparent diffusing capacity by about 2.1 ml per minute per mmHg, whereas in fact breathing at a positive pressure of 80 mmHg induced a mean decrease of diffusing capacity of 9.7 ml per minute per mmHg.

The duration of the breath hold has been shown to influence the value obtained for the pulmonary diffusing capacity (109). A breath-holding period

of ten seconds was used in the present study and the variation from this did not exceed ±1.0 second. The duration of both the period of inspiration of the carbon monoxide-helium mixture and of sample collection affect the final alveolar concentration of carbon monoxide (165). The method of allowing, for these effects suggested by Jones and Meade (165) was employed in the present study. The durations of inspiration and of the delivery of the alveolar sample were similar in the resting and pressure breathing periods.

The influence of changes of pulmonary ventilation and cardiac output upon the diffusing capacity are of interest since pressure breathing produced hyperventilation and a reduction of the cardiac output. Ogilvie, Forster, Blakemore and Morton 1957 (228) and Ross, Frayser and Hickham 1959 (252) have investigated the effect of hyperventilation upon the diffusing capacity as measured by the modified Krogh breath-holding technique. A considerable degree of hyperventilation amounting to a trebling of the resting pulmonary ventilation had no effect upon the value of the diffusing capacity in resting subjects. Ross, Frayser and Hickham 1959 (252) also demonstrated that a doubling of the resting cardiac output by the intravenous infusion of adrenaline, noradrenaline or atropine or by re-active hyperaemia in the lower limbs had no significant effect upon the diffusing capacity. Turino, Brandfonbrener and Fishman 1959 (277) reduced the blood flow to one lung in supine subjects by partial occlusion of one branch of the pulmonary artery. They found that the diffusing capacity of the lung was not reduced until the blood flow was decreased to less than half the control value. Rosenberg and Forster 1960 (251) studied the effects of pulmonary blood flow upon the diffusing capacity in isolated cat lungs. They found that, provided the pressure across the walls of the pulmonary vessels was unchanged, the diffusing capacity was constant over a wide range of pulmonary blood flows. Thus it would appear that the reduction of the diffusing capacity of the lungs induced by pressure breathing was not to be explained by either the hyperventilation or the reduction of pulmonary blood flow which were also produced by this manoeuvre.

The analysis of the apparent diffusing capacity into its two components, the diffusing capacity of the pulmonary membrane and the rate of uptake of carbon monoxide in the pulmonary capillary blood as developed by Roughton and Forster 1957 (254) was used in order to analyze the changes underlying the observed reduction of the apparent diffusing capacity in pressure breathing. Implicit in this theoretical analysis is the assumption that there is uniform distribution of diffusing capacity and capillary blood volume in relation to alveolar volume. Experimental evidence obtained by Forster, Fowler, Bates and Van Lingen 1954 (109) suggested that in normal resting subjects this assumption is not precisely true and the results of the measurements of the anatomical and physiological dead space in pressure breathing suggest that this manoeuvre causes an alteration in the distribution of the pulmonary capillary blood flow to ventilated alveoli. In the analysis of the present experimental data, however, the basic assumption made by Roughton and Forster 1957 (254) was adopted as it was considered that the error introduced by so doing would be small. The plot of the reciprocal of the apparent diffusing capacity against the reciprocal of the rate of uptake of carbon monoxide by the pulmonary capillary blood at the corresponding mean

capillary oxygen tensions for each subject in each experimental situation was found to be virtually linear. The linearity of these plots showed that the conditions produced by exposure to a given level of pressure breathing were reproducible since each point was obtained in a separate exposure to pressure breathing.

The absolute values obtained for the diffusing capacity of the pulmonary membrane and for the pulmonary capillary blood volume from the data for the apparent diffusing capacity at various mean pulmonary capillary oxygen tensions were determined by the relationship between θ and the mean pulmonary capillary oxygen tension used in the calculations. The relationship between θ and the oxygen tension in the plasma surrounding the red cells is determined by the ratio of the permeability of the red cell membrane to the permeability of the red cell interior for oxygen (λ). In the original description of this technique Roughton and Forster 1957 (254) used values of θ corresponding to two extremes of red cell membrane permeability $(\lambda = 1.5, \lambda = \alpha)$ and then they averaged the values of the diffusing capacity of the pulmonary membrane and the pulmonary capillary blood volume so obtained. They showed that the values obtained for the pulmonary capillary blood volume were relatively insensitive to the value of λ employed. The values obtained for the diffusing capacity of the pulmonary membrane were, however, sensitive to the chosen value of the red cell membrane permeability. In the present investigation the procedure devised by McNeill, Rankin and Forster 1958 (225) of using an average value of red cell permeability $\lambda = 2.5$ was adopted. Although this assumption may have affected the calculated absolute values of the diffusing capacity of the pulmonary membrane and the capillary blood volume, it did not affect their relative values.

The results of the analysis of the apparent diffusing capacity into its components demonstrated that pressure breathing did not change the diffusing capacity of the pulmonary membrane (Table 5-8). The reduction in the apparent diffusing capacity of the lungs was due to a decrease in the rate of the uptake of carbon monoxide by the red cells in the pulmonary capillaries. The rate of uptake of carbon monoxide by unit volume of blood is directly proportional to the concentration of haemoglobin in it (254). Thus the calculated reduction in the rate of uptake of carbon monoxide by the red cells in the pulmonary capillaries produced by pressure breathing could have been due to a reduction of either the pulmonary capillary blood volume or of the concentration of haemoglobin in it. Pressure breathing would have to produce a very large decrease (approximately 50%) in the haematocrit of the pulmonary capillary blood in order for this change to account for the observed change in the diffusing capacity. In fact the haematocrit of the venous and systemic arterial blood is increased by pressure breathing (146).

It may be concluded, therefore, that the reduction of the diffusing capacity produced by pressure breathing was due to a reduction of the volume of blood in the pulmonary capillaries. The pulmonary capillary blood volume during pressure breathing varied inversely with the magnitude of the breathing pressure (Table 5-8). In the previous chapter spirometric and radiological evidence was presented which suggested that at least a fraction of the blood which was displaced from the trunk into the limbs by pressure breathing originated in the lungs. Although the pulmonary capillary blood volume

forms only a small fraction of the total volume of blood contained within the thorax it probably shared in this general shift of blood from the viscera to the limbs. Such an association has been demonstrated to occur during changes in posture and during the Muller manoeuvre. Thus the reduction of the diffusing capacity associated with changing from the supine to the erect posture (Bates and Pearce 1956) (26) has been shown to be due to a reduction of the pulmonary capillary blood volume (185). Such a change of posture is known to produce a marked shift of blood from the heart and lungs into the lower limbs (270). Cotes, Snidal and Shepard 1960 (69) found that, in one subject, a reduction of the alveolar pressure to 24 mmHg below that of the environment during the period of breath-holding increased the pulmonary capillary blood volume at rest from 73 to 132 ml. Negative pressure breathing is known to increase the blood content of the lungs. Central venous engorgement produced by the inflation of a bladder around the lower half of the body also resulted in an increase in the pulmonary capillary blood volume (253). Thus there is considerable evidence that the blood in the pulmonary capillaries participates in any general shift of blood out of or into the thorax. It may be concluded, therefore, that the reduction of pulmonary capillary blood volume produced by pressure breathing was a part of the general shift of blood from the central part of the circulation which was induced by this procedure.

Alveolar Gas Tension at Reduced Environmental Pressure – Sampling of the alveolar gas following rapid decompression to a low pressure was performed with four different combinations of final environmental pressure and positive breathing pressure so that the absolute intrapulmonary pressure maintained following the decompression also varied with the positive breathing pressure (Fig. 5–15). Further, whilst in three of these conditions trunk counterpressure was used, no respiratory counterpressure was applied at a positive pressure of 30 mmHg, the lowest breathing pressure studied. These particular conditions were chosen since they represented certain pressure breathing systems which had been used, or were proposed for use in high altitude aircraft. Thus a system based upon a positive breathing pressure of 30 mmHg at an altitude of 50000 ft (barometric pressure – 87 mmHg) and using no respiratory counterpressure was in current use in the Royal Air Force, whilst the system maintaining an absolute intrapulmonary pressure of 141 mmHg as was provided by a breathing pressure of 80 mmHg at a simulated altitude of 57500 ft was considered adequate to prevent significant hypoxic effects upon the central nervous system.

The time course of the changes of the alveolar carbon dioxide tension obtained during pressure breathing following rapid decompression differed markedly from the course of the changes found during pressure breathing under comparable conditions at ground level. During pressure breathing at ground level (Fig. 5–8) the carbon dioxide tension fell progressively, the change being rapid at first and then slower. When pressure breathing was induced by a rapid decompression to simulated high altitude the alveolar carbon dioxide tension was lowest immediately after the decompression and it increased during the exposure, rapidly at first and then more slowly (Figs. 5–11 and 5–12). Although the values of the alveolar carbon dioxide tension immediately after the decompression were similar in the four different
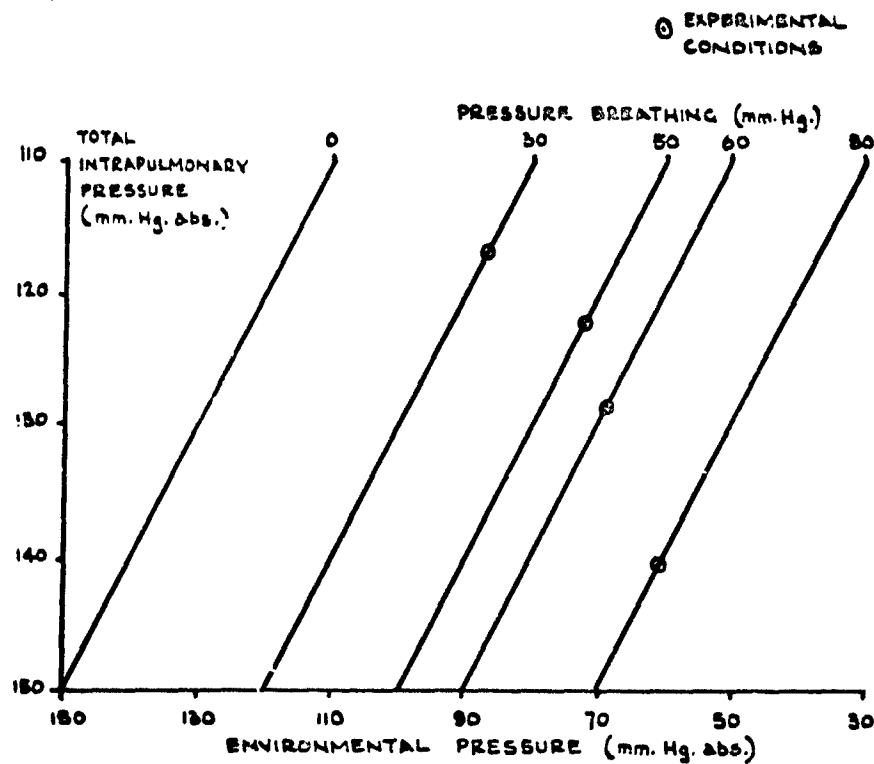
FIG. 5-15 The relationships between environmental pressure, positive breathing pressure and total intrapulmonary pressure used in the study of alveolar gas tensions during pressure breathing at high altitude

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pressure breathing conditions studied, the rate at which the carbon dioxide tension increased during the exposure varied with the experimental condition. When oxygen was breathed at the pressure of the environment (Fig. 5-13) the alveolar carbon dioxide tension immediately after the decompression was reduced to between 17 and 20 mmHg, which was very similar to the range of values obtained when the pressure breathing was induced following rapid decompression. Subsequently, the alveolar carbon dioxide tension rose more rapidly than in the pressure breathing experiments. The values of the carbon dioxide and oxygen tensions obtained after breathing 100% oxygen for one and a half to two minutes at the various absolute pressures used in this study are plotted in Fig. 5-16 together with the corresponding values obtained after one and a half to two minutes exposure to pressure breathing. The carbon dioxide tensions in the alveolar gas when oxygen was breathed at the pressure of the environment were uniformly greater than those found when pressure breathing was performed at the same absolute intrapulmonary pressure.

The sudden reduction of the alveolar carbon dioxide tension which occurred with the rapid decompression from a simulated altitude of 25 000 ft was a direct effect of the reduction of the absolute intrapulmonary pressure produced by the fall of environmental pressure (196) (95). As the environmental pressure fell during the rapid decompression the alveolar gas expanded, increasing the lung volume and passing through the mouth and nose to the environment until the absolute pressure within the respiratory tract equalled that delivered by the pressure demand oxygen regulator (99). In the present experiments where the duration of the decompression was approximately two seconds the time course of the absolute intrapulmonary pressure was very similar to that of the environmental pressure until the absolute pressure equalled that delivered by the oxygen regulator at which level the intrapulmonary pressure was maintained until recompression occurred (Ernsting, unpublished observation). The partial pressures of the alveolar gases were reduced as the total intrapulmonary pressure fell. Since the alveolar lining is moist and the lungs together with the blood flowing through them have a high heat capacity the expansion of alveolar gas under these conditions of decompression was probably isothermal so that at the end of the decompression the alveolar gas was fully saturated with water vapour at body temperature. When the assumption that no gaseous exchange occurred between the alveolar gas and the blood flowing through the pulmonary capillaries during the decompression was made, it was possible to calculate the expected tensions of alveolar gases immediately after the decompression from the corresponding gas tensions which existed before decompression and the initial and final absolute intrapulmonary pressures using the relationship:

$$P'_{Ax} = \frac{(P^{\bullet}_B - 47)}{(P'_B - 47)} \times P'_{Ax}$$

P'_{Ax} = alveolar tension of gas X before decompression
$P^{\bullet}_{Ax}$ = alveolar tension of gas X immediately after decompression
P'_B = total intrapulmonary pressure before decompression (mmHg)
$P^{\bullet}_B$ = total intrapulmonary pressure after decompression (mmHg).
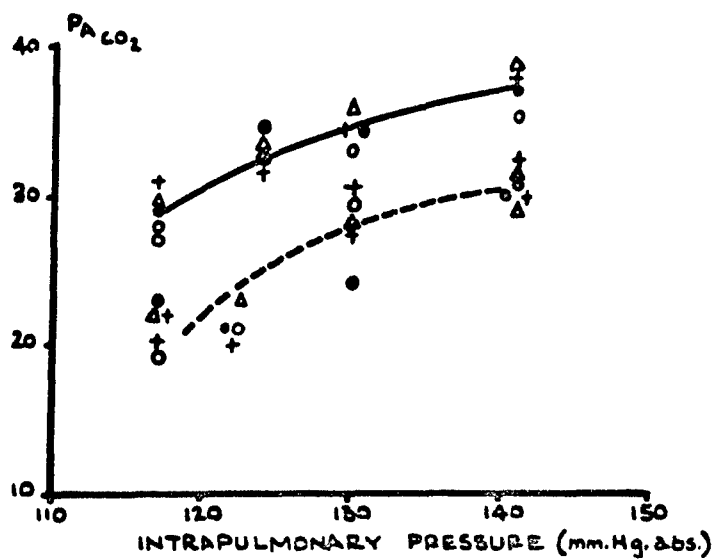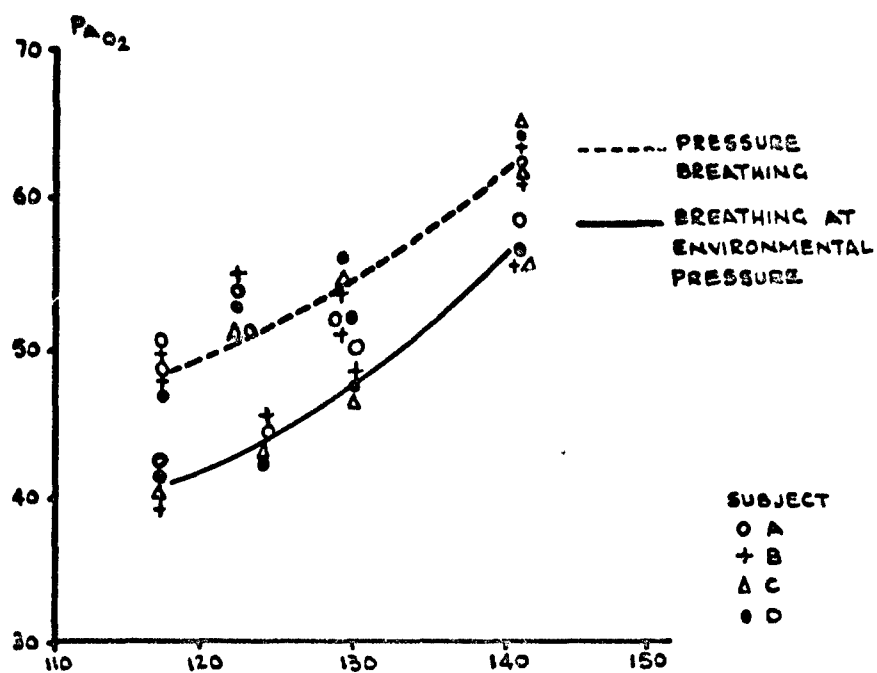
FIG. 5-16 The relationship between alveolar gas tensions and absolute intrapulmonary pressure whilst breathing oxygen at the environmental pressure (solid line) and during pressure breathing (interrupted line)

Such calculations have been made using the mean values of the alveolar gas tensions obtained before decompression at a simulated altitude of 25000 ft and the highest and lowest intrapulmonary pressures studied in this investigation (Table 5-14). The carbon dioxide tensions in the alveolar samples obtained two to five seconds after decompression in the present series of experiments were, however, not as low as the values calculated in Table 5-14. The assumption that no gaseous exchange occurred between the pulmonary capillary blood and the alveolar gas during the decompression was not strictly true. In fact as the alveolar carbon dioxide tension fell during the decompression the carbon dioxide tension gradient between the mixed venous blood and the alveolar gas rose progressively so that the rate at which carbon dioxide passed from the blood into the alveolar space was increased. Since the carbon dioxide tension of the mixed venous blood does not change until about twelve seconds after a sudden reduction of the arterial carbon dioxide tension Ernsting (1963) (93) the mixed venous-alveolar carbon dioxide tension gradient was increased to about five times the resting value immediately after the decompression. The rate of exchange of carbon dioxide between the pulmonary capillary blood and the alveolar gas is so rapid that even with the greatly increased carbon dioxide tension gradient at the beginning of the pulmonary capillaries there was probably almost complete equilibrium between the carbon dioxide tension of the blood leaving the pulmonary capillaries and that of the alveolar gas. This mechanism is supported by the detailed studies of the behaviour of the alveolar carbon dioxide tension and arterial pH during and following rapid decompression over a larger pressure range performed by Ernsting and McHardy 1962 (96). The pH of the arterial blood was recorded continuously and the measured changes of pH produced by rapid decompression from 560 to 140 mmHg absolute in 1.5 sec. were consistent with those predicted from the change of the alveolar carbon dioxide tension assuming that the end pulmonary capillary carbon dioxide tension equalled that of the alveolar gas. The very rapid passage of carbon dioxide into the alveolar gas reduced the fall of the tension of this gas during the decompression and brought about the rapid rise of carbon dioxide tension after the decompression.

The composition of the alveolar gas following rapid decompression when oxygen was breathed throughout the exposure at the pressure of the environment (Fig. 5-13) reflected the interaction of several factors. The alveolar tension of carbon dioxide was reduced by the rapid decompression and then it rose progressively to reach a level which was related to the absolute intrapulmonary pressure (Fig. 5-16). As the alveolar carbon dioxide tension increased in any given exposure the corresponding alveolar oxygen tension decreased since the sum of the partial pressures of these two gases was constant. The level of the alveolar oxygen tension over the whole range of intrapulmonary pressures was such that it constituted a stimulus to an increase of pulmonary ventilation at the resting subject (42) (242). The alveolar oxygen and carbon dioxide tensions obtained at the various absolute intrapulmonary pressures used both decreased as the total pressure was reduced. The relationships between the alveolar carbon dioxide and oxygen tensions which were operative two minutes after the decompression were similar to those obtained after considerably longer periods of breathing air at

TABLE 5-14

THE CALCULATED ALVEOLAR GAS TENSIONS FOLLOWING RAPID
DECOMPRESSION FROM A SIMULATED ALTITUDE OF 25 000 FT
WHILST 100% OXYGEN IS BREATHED — ASSUMING NO GASEOUS
EXCHANGE BETWEEN THE BLOOD AND THE GAS IN THE LUNGS
DURING DECOMPRESSION

	Alveolar gas tensions (mmHg)	
	Carbon dioxide	Oxygen
A. Before decompression		
Breathing 100% O_2 at 25 000 ft	40	195
B. After decompression		
(i) Pressure breathing at 30 mmHg at 50 000 ft (P^* = 117 mmHg)	12	58
(ii) Pressure breathing at 80 mmHg at 57 500 ft (P^* = 141 mmHg)	16	78

* Total absolute intrapulmonary pressure

reduced barometric pressure (40). Thus the values of the alveolar tensions of oxgyen and carbon dioxide obtained after two minutes exposure to reduced pressure appeared to be the result principally of the interaction between the hypoxic drive to ventilation of the low alveolar oxygen tension and the reduction of the normal carbon dioxide drive to ventilation. Even after this time, however, although the rates of change of the alveolar gas tensions were markedly reduced the gas stores of the body, particularly the carbon dioxide store, were far from an equilibrium state (100). It is impossible, therefore, to state with certainty the relative contributions of the various factors controlling the pulmonary ventilation in these circumstances.

It has been seen that the mean alveolar carbon dioxide tension at a given absolute intrapulmonary pressure two minutes after decompression was considerably less in the pressure breathing experiments than when oxygen was breathed at the pressure of the environment (Fig. 5-16). Since the absolute intrapulmonary pressures after decompression were strictly comparable, the reduction of the alveolar carbon dioxide tension produced by the decompression must have been similar in the two conditions. The slower rate of increase of alveolar carbon dioxide tension after decompression in the pressure breathing experiments must have been due to a greater alveolar ventilation and perhaps a lower pulmonary capillary blood flow during pressure breathing than when oxygen was breathed at the pressure of the environment. It was seen earlier in this chapter that pressure breathing at ground level with a normal alveolar oxygen tension produced a true alveolar hyperventilation. Further, the pulmonary ventilation associated with pressure breathing at reduced barometric pressure was found to be greater than the ventilation produced by pressure breathing at the same pressure at ground level (Table 5-9). The lowest alveolar carbon dioxide tension found in the present study was produced by pressure breathing at a positive pressure of 30 mmHg without respiratory counterpressure at a simulated altitude of 50000 ft which gave an intrapulmonary pressure of 117 mmHg absolute. Pressure breathing at this level without counterpressure produced a very marked increase of pulmonary ventilation, even at ground level and in spite of the very low alveolar carbon dioxide tension the alveolar oxygen tension was the lowest encountered during pressure breathing. In the other three conditions, in which trunk counterpressure was employed, the absolute intrapulmonary pressure was increased as the positive breathing pressure was increased (Fig. 5-15). The increase of the absolute intrapulmonary pressure caused a corresponding rise of the alveolar oxygen tension.

The most important finding obtained from the analysis of the alveolar gas samples obtained during pressure breathing at reduced environmental pressures was the low tension of carbon dioxide in the alveolar gas under these conditions. As a direct result of this reduction of the alveolar carbon dioxide tension below the normal level, the alveolar oxygen tension was raised. Further, since the pH of the blood flowing through the pulmonary capillaries was increased by the reduction of the alveolar carbon dioxide tension and by the reduction of the oxygen saturation of the blood (61) the concentration of oxygen in the arterial blood at a given oxygen tension was also raised. In addition, the reduced arterial carbon dioxide tension will have had important effects upon the cardiovascular system, in particular the cerebral circulation.

Arterial Blood Gas Tensions – The efficiency with which these pressure breathing systems prevented hypoxia at low environmental pressures was assessed finally by determining the composition of the arterial blood under these conditions. All the measurements were made following rapid decompression to a simulated altitude of 56000 ft ($P_B = 65.7$ mmHg). The total pressure of the arterial gases during pressure breathing at a pressure of 60–80 mmHg at this simulated altitude was considerably greater than the environmental pressure, so that under these conditions bubbles were expected in arterial blood withdrawn by the usual technique. An experiment in which a technical failure occurred and the arterial blood sample was taken at the pressure of the environment confirmed this prediction. The technique whereby the sampled arterial blood was maintained at an absolute pressure of greater than 141 mmHg (Fig. 5–14) was developed to avoid the errors which could arise from the formation of discrete gas bubbles in a blood sample before analysis. Preliminary experiments demonstrated the importance of maintaining the pressure in the whole of the sampling system considerably above that of the environment. Originally it was intended that the sampling system should be flushed by opening the side arm of the tap attached to the syringe to the environment. Directly this manoeuvre was performed at a simulated altitude of 56000 ft, however, large bubbles of gas formed in the blood in the sampling system. The waste bottle was therefore pressurized as well as th box containing the sampling syringe. The final technique was found to be very satisfactory and blood samples completely free of gas bubbles were obtained in all the experiments, the results of which are presented in Table 5–10.

The pressure clothing assemblies used in this part of the investigation differed from those employed during the sampling of alveolar gases at reduced environmental pressure. Standard pressure breathing masks and pressure helmets were worn and counterpressure was also applied to the lower limbs by means of an anti-g suit. These assemblies represented those which it was proposed should be used in flight. The use of an oronasal mask for the delivery of a positive breathing pressure of 60–63 mmHg introduced the discomforts which have been discussed in Chapter 3. The application of counterpressure to the lower limbs reduced the volume of blood displaced from the trunk into the periphery by the raised intrapulmonary pressure (Chapter 6). There was a small variation in the pressure maintained in the breathing cavity of the mask or helmet by the pressure demand regulator (Mark 20 or 21) used in these experiments (Table 5–10). This variation was due principally to the presence of outboard leakage of oxygen from the mask or pressure helmet.

Since each arterial sample was drawn at a constant rate over a two minute period its carbon dioxide tension represented approximately the mean value of the arterial carbon dioxide tension over this period. The values obtained during the control period with the subject breathing oxygen at a simulated altitude of 25000 ft gave a mean value of 39.6 mmHg for the arterial carbon dioxide tension. This did not differ significantly from the mean value of 40.3 mmHg obtained for the alveolar carbon dioxide tension determined under the same conditions by Haldane-Priestley sampling. The normal venous admixture contributed by the true anatomical right to left shunts and by alveoli with very low ventilation-perfusion ratios Riley and Cournand

1949 (244) was responsible for the slight desaturation of the arterial blood obtained under these circumstances, in spite of the high alveolar oxygen tension.

Sampling of arterial blood during pressure breathing was not started until at least one minute had elapsed following the rapid decompression so that the gross disturbances of gas exchange induced by the sudden reduction of the environmental pressure had partially subsided. The arterial carbon dioxide tension was considerably reduced and the values obtained in these experiments (Table 5–16) were similar to the values of the alveolar carbon dioxide tension obtained by Haldane-Priestley sampling under comparable conditions. The arterial carbon dioxide tension was consistently lower when pressure breathing was performed with an oronasal mask jerkin and anti-g suit than when a pressure helmet, jerkin and anti-g suit were worn. In these experiments the discomfort of breathing at a positive pressure of 60 mmHg with an oronasal mask was added to the lower intrapulmonary pressure (127 mmHg absolute) employed when this assembly was used. Although a higher positive breathing pressure (79 mmHg) was experienced in the series in which a pressure helmet was worn, the absence of discomfort in the head and neck and the higher intrapulmonary pressure (145 mmHg absolute) resulted in a smaller degree of hypocapnia one to three minutes after the decompression.

The oxygen saturation of the arterial blood obtained during pressure breathing with an intrapulmonary pressure of 145 mmHg absolute was consistently greater than that of the blood obtained when intrapulmonary pressure was only 127 mmHg absolute. The mean increase in the percentage saturation associated with the 17.2 mmHg increase in the total intrapulmonary pressure in the eight pairs of experiments was 6.3%. Of greater interest, however, was perhaps the associated increase in the tension of oxygen in the arterial blood (Table 5–15). The oxygen tension of each blood sample was calculated using Dill's oxygen dissociation curves for whole blood (74). The accuracy of this indirect technique of assessing the oxygen tension of a blood sample varied with the actual value of the oxygen saturation because of the shape of the oxygen dissociation curve. The slope of the dissociation curve changes markedly over the range of saturations recorded in the present experiments (Table 5–16) so that the accuracy with which the oxygen tension could be predicted from the oxygen saturation was reduced by more than three-fold as the saturation increased from 82.5% to 92.5%. An increase of the pH of the blood from 7.40 to 7.50 slightly decreased the slope of the dissociation curve over the range of interest in the present context. Since the difference between duplicate analyses of the oxygen saturation of the same blood sample did not exceed 1%, the probable error of the predicted oxygen tension varied from about 1.2 mmHg at a saturation of 82.5% to 4 mmHg at 92.5%. The accuracy with which the pH of each blood sample was measured was such that the inaccuracy in the predicted oxygen tension due to variations in this factor was negligible. A further variation was introduced by the use of a standard oxygen dissociation curve rather than the curve for the subject's own blood. The magnitude of the error introduced in this manner was probably small.

The sum of the partial pressures of oxygen, carbon dioxide and water vapour in the arterial blood sampled during pressure breathing have been

TABLE 5-15

THE ARTERIAL GAS TENSIONS DURING PRESSURE BREATHING AT A SIMULATED ALTITUDE OF 56 000 ft (P$_B$ = 65.7 mmHg)

Subject	Positive breathing pressure (mmHg)	Arterial gas tensions Carbon dioxide (mmHg)	Oxygen (mmHg)	Alveolar – arterial total pressure difference (mmHg)
Pressure breathing with mask, jerkin and anti-G suit				
	63	26.5	51.0	4.2
	61	29.4	45.0	5.3
	60	31.0	54.0	2.7
	60	29.2	44.0	5.5
	63	26.6	47.0	8.1
	60	28.3	43.5	6.9
	63	30.2	46.0	5.5
	61	28.5	44.5	6.7
Mean	61.4	28.7	45.8	5.6
Standard error		±0.56	±0.85	±0.59
Pressure breathing with helmet, jerkin and anti-G suit				
	79	29.0	63.0	5.7
	80	32.5	60.0	6.2
	80	31.5	62.0	5.2
	78	34.5	57.0	5.2
	77	30.2	62.5	3.0
	80	30.6	62.0	6.1
	77	34.5	56.0	5.2
	80	29.5	60.5	8.7
Mean	78.9	31.5	60.4	5.7
Standard error		±0.23	±0.92	±0.56

TABLE 5-16

THE SLOPE OF THE OXYGEN DISSOCIATION CURVE FOR HUMAN BLOOD AT 37°C AT VARIOUS LEVELS OF OXYGEN SATURATION (DILL, 1944)

Oxygen saturation (%)	Slope of dissociation curve (mmHg per % saturation)	
	pH = 7.4	pH = 7.5
70–80	0.96	0.86
80–85	1.20	1.08
85–90	1.94	1.73
90–95	4.00	3.53

compared with the corresponding total intrapulmonary pressure (Table 5–15). In every experiment the sum of the arterial gas tensions was less than the corresponding absolute intrapulmonary pressure and although there was a very considerable variation from one experiment to another the mean value of the difference in all the experiments amounted to 5.6 mmHg Apart from experimental errors several physiological factors could have contributed to this difference. Any nitrogen present would have been included in the difference since the tension of nitrogen was taken into account in the alveolar gas but not in the arterial blood. In every experiment the subject had breathed oxygen containing less than 0.5"₀ nitrogen for at least one hour before the exposure to pressure breathing so that the alveolar and arterial nitrogen tensions were probably less than 1.5 mmHg. Evidence has already been presented to support the assumption that the arterial carbon dioxide tension represented the mean alveolar carbon dioxide tension in pressure breathing.

Thus most of the observed difference between the sums of the gas tensions in the alveolar air and in the arterial blood was due to the difference between the alveolar tension of oxygen and the arterial tension of this gas. An alveolar-arterial oxygen tension difference may be due to a finite resistance to the diffusion of oxygen from the alveolar air into the pulmonary capillary blood (diffusion component), the passage of venous blood through channels not exposed to alveolar gas (shunt component) and an uneven distribution of alveolar ventilation to pulmonary blood flow (distribution component). Farhi and Rahn 1955 (101) showed theoretically that when nitrogen is eliminated from the inspired gas the distribution component of the gradient becomes negligible. The major fraction of the alveolar-arterial oxygen tension difference found in the present experiments with an alveolar oxygen tension between 50 and 65 mmHg was probably due to the presence of a finite resistance to overall diffusion since the contribution of the shunt component would have been greatly reduced as compared to normal by the small difference between the oxygen tensions of the mixed venous and of the arterial blood in this situation. Although the measurements of the alveolar-arterial oxygen tension gradient during pressure breathing were subject to considerable technical errors, the results support the conclusion that pressure breathing does not impair the uptake of oxygen by the blood flowing through the pulmonary capillaries.

The calculated values of the arterial oxygen tension obtained during pressure breathing exhibited considerable variation from one exposure to another in the same series of experiments (Table 5–15). The major portion of this variation was contributed by variations in the arterial carbon dioxide tension rather than by changes in the alveolar-arterial oxygen tension difference. The mean arterial oxygen tension achieved with a positive breathing pressure of 61 mmHg and an intrapulmonary pressure of 127 mmHg absolute was 45.8 mmHg. In the second series of experiments in which an absolute intrapulmonary pressure of 145 mmHg was attained by the use of a positive breathing pressure of 79 mmHg the mean arterial oxygen tension was 60.4 mmHg. Thus a difference of 17.5 mmHg in the total intrapulmonary pressure was associated with a difference of 14.6 mmHg between the means of the arterial oxygen tensions attained in the two conditions. It may be seen,

however (Table 5-16) that the arterial oxygen tensions found at the greater intrapulmonary pressure were associated with slightly higher values of the arterial carbon dioxide tension. The difference between the mean values of the sums of the individual values of the oxygen and carbon dioxide tensions in each series was 17.3 mmHg. Thus the difference between the sum of the arterial oxygen and carbon dioxide tensions in the two series of experiments was equal to the difference between the respective intrapulmonary pressures.

Previous investigations of the composition of the arterial blood during pressure breathing at low environmental pressures have been restricted to the study of the effects of positive breathing pressures of up to about 30 mmHg. Dill and Penrod 1948 (75) found that breathing oxygen at the environmental pressure at a simulated altitude of 44 800 ft (environment pressure = 112 mmHg) gave, in a group of eight subjects, a mean arterial oxygen saturation of 66.5"₀ and a mean arterial carbon dioxide tension of 26 mmHg. Barach, Eckman, Eckman, Ginsburg and Rumsey 1947 (16) studied the effect of a positive breathing pressure of 15 mmHg at this simulated altitude (45 000 ft) which gave an intrapulmonary pressure of 125 mmHg absolute and found that it produced a mean arterial saturation in five subjects of 80.0"₀ and a mean arterial carbon dioxide tension of 32.9 mmHg. They found further that breathing oxygen at the pressure of the environment at a simulated altitude of 42 300 ft (126 mmHg absolute) produced a mean arterial oxygen saturation of 80.6"₀ in a group of five subjects and a mean arterial carbon dioxide tension of 35.2 mmHg. Thus even a positive breathing pressure of 15 mmHg induced a certain degree of hyperventilation and produced the arterial oxygen saturation obtained when oxygen was breathed at an environmental pressure equal to the total intrapulmonary pressure which existed during pressure breathing.

Taylor, Marbarger and Power 1948 (275) investigated, in a group of three subjects, the composition of the arterial blood during pressure breathing with trunk counterpressure at a positive pressure of 32 mmHg at a simulated altitude of 50 000 ft which gave an intrapulmonary pressure of 119 mmHg absolute. The mean arterial oxygen saturation attained under these conditions was 77.5"₀, the mean arterial carbon dioxide tension was 32 mmHg and the mean arterial oxygen tension was 40 mmHg. A comparison of the results obtained in the present study with those of previous investigators shows that at similar absolute intrapulmonary pressures the arterial carbon dioxide tension was considerably lower in the present experiments. The experimental conditions employed by Taylor, Marbarger and Power 1948 (275) and Barach, Eckman, Eckman, Ginsburg and Rumsey 1947 (16) differed in three important respects from those of the present study. The positive breathing pressures used in the latter were between twice and four times those investigated by Taylor and Barach. The low environmental pressure was attained in the experiments of both Taylor et al and Barach et al by a relatively slow reduction of pressure and the arterial blood was sampled between two and fifteen minutes of the exposure whereas in the present experiments the exposure was commenced with a rapid decompression and sampling was performed much earlier. These differences in the experimental conditions all tended to produce lower values of the arterial carbon dioxide tension in the

arterial blood as sampled in the present experiments as compared with the values obtained by the earlier investigators.

In some respects it was desirable that the sampling of arterial blood should have been performed when a respiratory "steady state" had been attained during pressure breathing at reduced environmental pressure. It was not possible, however, to expose the subjects to a prolonged period of pressure breathing at the high positive pressures used in this study. The severe cardio-vascular disturbances induced by pressure breathing at these pressures limited the acceptable duration of an exposure to a simulated altitude of 56000 ft to less than four minutes. Since each arterial sample was taken over a two minute period after an initial delay of one minute the time available for the induction of pressure breathing was short. Thus the environmental pressure was reduced over two seconds in order to produce a rapid onset of pressure breathing. This form of induction of pressure breathing at reduced environmental pressure was also of practical interest since failure of the pressure cabin of an aircraft during flight may occur very rapidly.

The relatively high values of arterial oxygen tension and saturation found during pressure breathing at simulated high altitude with an intrapulmonary pressure of 127 mmHg absolute were due to the low value of the alveolar carbon dioxide tension which existed in this condition (Table 5–15). Thus it may be calculated (Table 5–17) that if, in these circumstances, the alveolar carbon dioxide tension had been 40 mmHg, the arterial oxygen saturation would have been only 67"₀ as compared with the value of 84.3"₀ obtained experimentally. The reduction of the alveolar carbon dioxide tension produced this effect by two mechanisms. Firstly the alveolar oxygen tension was raised from the value which would have existed with an alveolar carbon dioxide tension of 40 mmHg by 11 mmHg to about 50 mmHg. Secondly, the associated increase in the pH of the pulmonary capillary blood raised the oxygen saturation of the arterial blood by 5"₀. At the higher intrapulmonary pressure of 145 mmHg absolute the calculated arterial oxygen saturation produced by an alveolar carbon dioxide tension of 40 mmHg was 85.5"₀ so that the observed degree of hypocapnia raised the arterial oxygen saturation by 6"₀. Thus the observed hypocapnia contributed only about a third of the increase of the arterial oxygen saturation at an intrapulmonary pressure of 145 mmHg absolute as compared with that induced by a similar degree of hypocapnia at an absolute intrapulmonary pressure of 127 mmHg. This difference was due to the increased steepness of the oxygen dissociation curve at the lower arterial oxygen tension.

The primary purpose of pressure breathing at high altitude is the maintenance of normal cerebral activity. The function of the central nervous system depends upon its oxygen supply which is determined by the oxygen saturation and tension of the arterial blood and the blood flow which it receives. The effect of pressure breathing under the conditions used in these experiments upon the cerebral blood flow has not been measured. In Chapter 6 it will be shown, however, that pressure breathing with counterpressure applied to the trunk and lower limbs produces only a small change in the cerebral arterio-venous vascular pressure difference. It would appear unlikely, therefore, that pressure breathing under these conditions causes any gross change of cerebral blood flow by a mechanism involving a change in driving

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TABLE 5-17

THE PREDICTED EFFECT OF AN ALVEOLAR CARBON DIOXIDE TEN-
SION OF 40 mmHg UPON THE ARTERIAL GAS TENSIONS AND THE
MEAN CEREBRAL CAPILLARY OXYGEN TENSION DURING PRESSURE
BREATHING AT 61 mmHg WITH AN INTRAPULMONARY PRESSURE OF
127 mmHg ABSOLUTE

	Breathing air at ground level	Pressure breathing with an intra-pulmonary pressure of 127 mmHg	
		With observed alveolar carbon dioxide tension	With assumed alveolar carbon dioxide tension
Alveolar gas tensions			
Carbon dioxide (mmHg)	40	29	40
Oxygen (mmHg)	100	51	40
Arterial blood			
Carbon dioxide tension (mmHg)	40	29[1]	40
Oxygen tension (mmHg)	90	46[1]	35
Oxygen saturation (%)	97	84.3[1]	67
Cerebral metabolism			
Oxygen consumption (ml/min./100g brain)	3.3	3.3	3.3
Blood flow (ml/min./100g brain)	60	40	72
Cerebral blood oxygen tension			
Internal jugular (mmHg)	40	22	24.5
Mean cerebral capillary (mmHg)	57	30	28

[1] Experimentally determined values

pressure across the cerebral vascular bed. The tensions of carbon dioxide and oxygen in the arterial blood do, however, play an important part in the control of the cerebral vascular resistance. Hypocapnia causes constriction of the cerebral vessels whilst moderate or severe hypoxia induces a vasodilatation (170). Thus at a constant arterial oxygen tension above 40 mmHg a reduction of the arterial carbon dioxide tension from the normal level of 40 mmHg to 30 mmHg decreases the cerebral blood flow by one third (170, 177). At a constant arterial carbon dioxide tension hypoxaemia does not change the cerebral blood flow until the arterial oxygen tension is reduced below 50 mmHg. The increase in cerebral blood flow induced by a reduction of the arterial oxygen tension to 45 mmHg amounts to less than 5", of the resting value (177).

Thus the mean arterial carbon dioxide and oxygen tensions (29 and 46 mmHg respectively) found during pressure breathing at a simulated altitude of 56000 ft with an intrapulmonary pressure of 127 mmHg absolute would have reduced the cerebral blood flow to about two-thirds of the normal resting value. If, however, the arterial carbon dioxide tension had been normal (40 mmHg) the arterial oxygen tension would have been about 35 mmHg at the same total intrapulmonary pressure (Table 5–18) and these conditions would have resulted in an approximately 20", increase of the cerebral blood flow above the normal resting level. Thus, although the hypocapnia found during pressure breathing at reduced environmental pressure following a rapid decompression resulted in a considerably higher arterial oxygen saturation than would have occurred if the arterial carbon dioxide tension had been 40 mmHg, the blood flow to the brain would have been some 75", greater if no hypocapnia had occurred. The experimental results upon which these calculations are based were all obtained in the steady state. The velocity at which these changes of cerebral vascular resistance occur when the arterial gas tensions are suddenly changed is uncertain. Studies using radioactive krypton suggest, however, that cerebral vascular responses to alterations of carbon dioxide tension occur within one minute of a change of arterial gas tension (186).

The relative effects of these changes of arterial oxygen saturation and blood flow upon the oxygen supply to the cerebral tissues may be expressed quantitatively by using Barcroft's concept (24) of a mean tissue capillary oxygen tension. This is the tension which, if it existed along the whole length of all the capillaries of the tissues, would result in the transfer of oxygen at the rate at which it actually occurs. If a steady state of respiratory gas exchange within the brain is assumed then it is possible to calculate the mean capillary oxygen tension corresponding to the two conditions which have been considered during pressure breathing (Table 5–17). Thus, although as compared with the state which obtained with an arterial carbon dioxide tension of 40 mmHg the observed hypocapnia increased the arterial oxygen saturation from 67", to 84", and the arterial oxygen tension from 35 to 46 mmHg, it was associated with an increase of only 2 mmHg in the mean cerebral capillary oxygen tension. Although several of the assumptions made in these calculations have not been tested experimentally, the results serve to emphasize that the observed hypocapnia produced a relatively small increase in the tension at which oxygen was delivered to the brain.

SUMMARY

The investigations described in this chapter confirmed that the most important disturbance of pulmonary gas exchange induced by pressure breathing was an increase of pulmonary ventilation. Although this manoeuvre increased the physiological dead space the increment in the total pulmonary ventilation was such that the alveolar ventilation was also increased. There was a reduction of the alveolar carbon dioxide tension which was more profound during pressure breathing in the presence of mild hypoxia at reduced environmental pressure than at ground level. The distribution of the inspired gas within the lungs was not affected by pressure breathing but evidence was obtained which suggested that this procedure disturbed the distribution of the pulmonary blood flow and reduced the volume of the blood within the pulmonary capillaries. There was, however, no impairment of diffusion across the alveolar capillary membrane proper. Although pressure breathing produced a disturbance of the relationships between alveolar ventilation and pulmonary capillary blood flow in the lungs, the increase of pulmonary ventilation prevented any significant impairment of the overall exchange of carbon dioxide and oxygen between the inspired gas and the blood flowing through the lungs. The vascular changes were thought to be the result of the shift of blood to the periphery which was produced by pressure breathing. The alveolar hyperventilation during pressure breathing at simulated high altitude with an alveolar oxygen tension of the order of 50–60 mmHg resulted in a considerably higher arterial oxygen saturation than would have otherwise occurred.

THE CARDIOVASCULAR EFFECTS OF HIGH
PRESSURE BREATHING

INTRODUCTION

The early investigations (119) (30) of the effects of pressure breathing at positive pressures above 30 mmHg showed that the time for which this manoeuvre could be performed was limited by the occurrence of syncope. The application of counterpressure to the chest or trunk allowed the use of greater positive breathing pressures but the time for which pressure breathing could be performed without circulatory collapse was found to be progressively reduced as the breathing pressure was raised. Thus the practical use of positive pressure breathing with oxygen as a means of preventing serious hypoxia at altitudes above 40000 ft was primarily limited by the occurrence of syncope.

Direct observation of the superficial veins of the limbs shows that one of the most important effects of positive pressure breathing is the displacement of blood into the peripheral capacity vessels. Limited studies of the amount of blood displaced in this manner were made by Henry 1951 (146) who found that pressure breathing with counterpressure applied to the chest at a positive pressure of 40 mmHg displaced about 300 ml of blood into the lower limbs in the erect posture. In the present investigation using the complete trunk counterpressure afforded by the pressure jerkin, the volume of blood displaced into various segments of the upper and lower limbs has been determined at positive breathing pressures of up to 130 mmHg. The progressive increase of limb volume subsequent to the displacement of blood into the part at the beginning of pressure breathing has also been studied as a measure of the increase in the volume of extravascular fluid within the limb. The influence of these disturbances of the distribution and the volume of the circulating blood induced by positive pressure breathing upon the cardiovascular system have been investigated by measuring the heart rate and the pressures at various sites within the circulation. The nature, incidence and causation of the syncope which arises during pressure breathing has also been examined. The effects of the application of various degrees of counterpressure to the limbs upon the cardiovascular disturbances produced by pressure breathing have been determined together with the limits of the protection afforded by various combinations of pressure garments. When the degree of positive pressure which can be employed with a given pressure breathing system is limited as it is in a system using an oronasal mask, the maximum altitude to which this system can be used safely will depend upon the minimum absolute intrapulmonary pressure which can be tolerated. The latter in turn depends upon the degree of hypoxia which is acceptable under the conditions in which the system will be used. Experiments have been performed in order to determine the effects of various degrees of hypoxia upon the cardiovascular responses to positive pressure breathing.

Changes of Limb Volume – In separate experiments the volume of the hand, forearm, thigh or calf was recorded continuously using a water-filled plethysmograph. The subject was secured in the ejection seat by means of the standard seat harness in order to reduce to a minimum the movements of the limb segment under study with the induction and cessation of pressure breathing. The upper limb plethysmographs were placed at such a height that the mid-plane of the segment within the plethysmograph lay 10 cm below the sternal angle. When the volume of a lower limb segment was measured the limb was supported in the horizontal position with the hip joint flexed to 90°. The changes in the impedance between the electrodes of the plethysmograph produced by alterations in the level of water within it were fed on to a galvanometer of a photographic recorder by means of a suitable amplifier. The subject wore a Type D partial pressure headpiece and a pressure jerkin which was supplied by a demand regulator, the outlet pressure of which could be varied between 0 and 150 mmHg gauge. The pressure in the headpiece was measured by means of an unbonded strain gauge pressure transducer, the output of which was fed on to the second galvanometer of the photographic paper recorder.

In any given experiment the behaviour of only one limb segment was studied. In most of the experiments a series of four exposures to various positive breathing pressures was made. The duration of an exposure to pressure breathing was varied inversely with the positive breathing pressure from five minutes at a pressure of + 50 mmHg to two minutes at a pressure of + 130 mmHg. Following an exposure to pressure breathing a period of at least ten minutes was required before the limb volume had returned to its resting value. In a few of the experiments in this series in which the volume of the forearm was measured, the pressure in an antecubital vein was recorded by means of a capacitance manometer. Two individuals who were experienced in pressure breathing were used for the majority of the experiments in this study. A series of experiments was also performed on a further group of four subjects trained in pressure breathing. In this series only the forearm volume was recorded.

Results – The general shape of the curve relating limb segment volume to time before, during and after pressure breathing was found to be independent of the site of the limb segment and of the positive breathing pressure at which the curve was obtained. A record obtained in a typical experiment in which forearm volume, pressure in the homolateral antecubital vein and headpiece pressure were measured is presented in Fig. 6–1. At the beginning of pressure breathing there was a rapid increase both in limb volume and of venous pressure over a period of ten to twenty seconds. The rate of increase of limb volume then declined progressively until a relatively constant minimum value was attained. This minimum rate of increase of volume was maintained for as long as pressure breathing was continued. The rate of rise of venous pressure at the beginning of pressure breathing increased progressively until a value which was very nearly equal to the positive breathing pressure was attained. The venous pressure then remained constant except for small fluctuations which were respiratory in timing until pressure breathing was terminated. The time at which the venous pressure reached the final plateau value was the same as that at which the rate of increase of limb volume
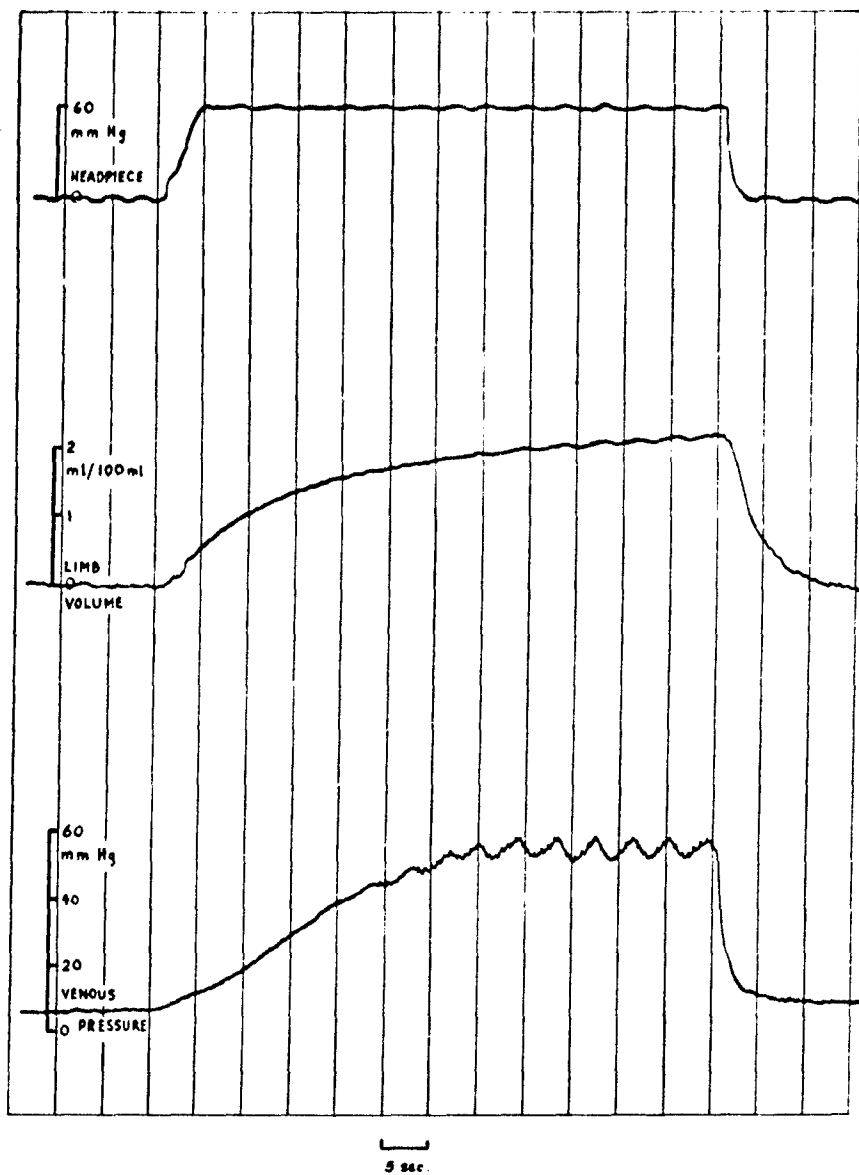
FIG. 6-1 The effect of pressure breathing at a positive pressure of 60 mmHg with trunk counterpressure upon forearm volume (middle trace). Peripheral venous pressure (bottom trace) and headpiece pressure (top trace)

reached a minimum value. When pressure breathing stopped both the limb volume and the venous pressure fell rapidly, the decrease of pressure being more rapid than that of limb volume. Even one minute after the cessation of pressure breathing the segment volume was always greater than the resting value. Subsequently there was a slow decline of limb volume until five to ten minutes later the volume had regained its original value. The volume pulse of the limb segment was reduced during pressure breathing as compared with the magnitude of this variation of volume in the resting state. Directly pressure breathing was terminated the pulse volume of the limb increased to a value which was considerably greater than the resting value, particularly after pressure breathing at positive pressures greater than 50 mmHg.

It was assumed that the initial rapid increase of limb volume was due to an increase in the blood content of the part. The magnitude of this increase of the blood volume was determined from the record of limb volume by measuring the increment of volume up to the point at which the rate of increase of volume reached a constant minimum value (Fig. 6-2). In order to define this point more clearly a straight line was drawn through the latter part of the limb volume curve. The increase of the limb volume up to this point was then expressed as a proportion of the resting volume of the segment of the limb within the plethysmograph which had been measured at the end of the experiment. The relationships between the increase of the blood content of the limb segment studied produced by positive pressure breathing and the corresponding positive breathing pressures for the two subjects studied in detail are presented in Figs. 6-3 and 6-4. The increase of blood content per unit volume of limb segment at a given positive breathing pressure is considerably greater in the upper limb than in the lower. The results of the experiments performed on the other four subjects are presented in Table 6-1.

The slow increase of limb volume during pressure breathing subsequent to the initial rapid increase due to the displacement of blood into the region was measured for each record. The difference between the limb volume one minute after cessation of pressure breathing and that in the resting state was also measured. The relationship between these two volume changes is presented in Fig. 6-5. Although there was a considerable scatter between individual values these two volumes were approximately equal in any given experiment. The slow rate of increase of limb volume during pressure breathing was measured for each record and expressed as the increase of limb volume per unit volume of limb segment per minute. The relationships between the rate of increase of limb volume and the corresponding positive breathing pressures are presented in Figs. 6-6 and 6-7 for each of the limb segments studied. There is a virtually linear relationship between the rate of increase of limb volume and the positive breathing pressure. At a given positive breathing pressure the rate of increase of volume is greater in the upper limb segments than in the lower limb segments.

The Distensibility of the Vessels of the Hand – The distensibility of the capacity vessels of the hand was measured during pressure breathing and during simple venous congestion in order to determine whether pressure breathing induced any change of vascular distensibility.

The subject, wearing a Type D partial pressure headpiece and a pressure jerkin was secured in an ejection seat. The volume of one hand was recorded

TABLE 6-1

THE INCREASE IN BLOOD CONTENT OF THE FOREARM DURING PRESSURE BREATHING

Subject	Positive breathing pressure (mmHg)	Increase of blood content (ml/100 ml forearm)
C	50	1.9
	100	2.3
	130	2.6
D	40	1.4
	60	1.7
	110	2.4
E	50	1.6
	80	2.0
	120	2.5
F	30	1.0
	60	1.8
	90	2.2

TABLE 6-2

SYSTEMIC ARTERIAL PULSE PRESSURE DURING PRESSURE BREATHING

Positive breathing pressure (mmHg)	Subject	Arterial pulse pressure (mmHg)	
		Rest	Pressure breathing
A. No counterpressure			
30	A	55	20
35	C	60	25
50	D	45	15
B. Chest counterpressure			
60	A	60	28
60	E	52	32
80	F	55	35
C. Trunk counterpressure			
60	B	60	35
60	C	55	30
80	A	55	25
80	B	62	33
80	D	53	32
100	C	58	25
100	B	60	22
120	A	57	28
120	B	65	30
D. Trunk and lower limb counterpressure			
60	F	55	40
60	A	53	38
80	F	60	41
80	E	50	28
80	D	50	25
100	A	56	35
100	D	54	22
120	E	55	25
120	F	63	25

LIMB VOLUME

SLOPE = RATE OF FORMATION
OF EXTRAVASCULAR FLUID

INCREASE OF
BLOOD CONTENT

START OF
PRESSURE BREATHING

TIME.
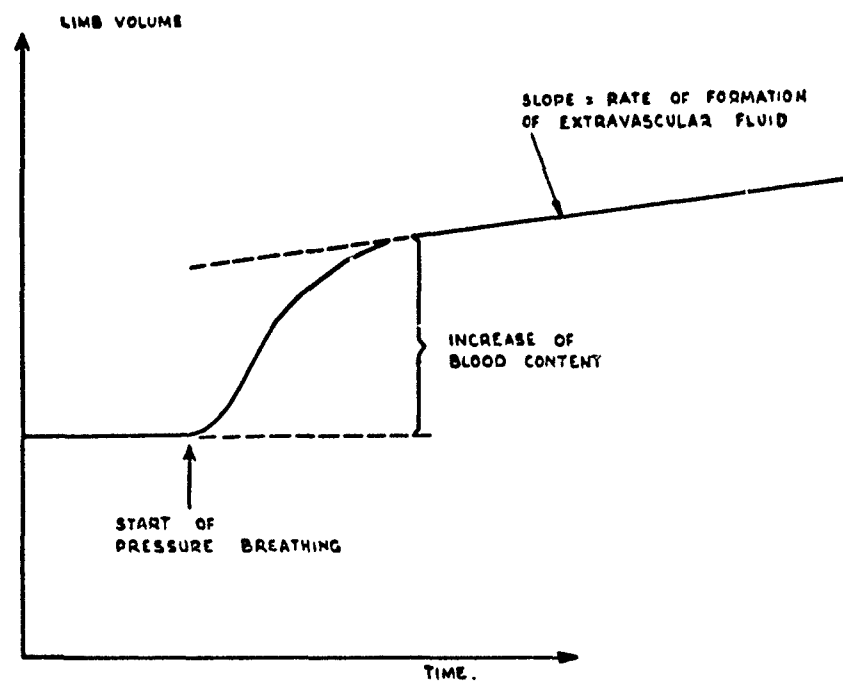
FIG. 6–2 The analysis of the limb volume changes induced by pressure breathing
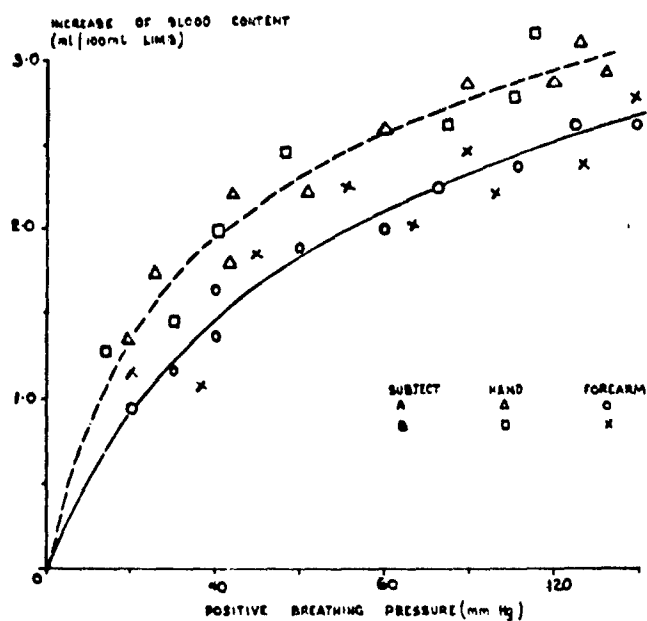
FIG. 6 3 The increase of the blood content of the hand and forearm induced by
pressure breathing with trunk counterpressure
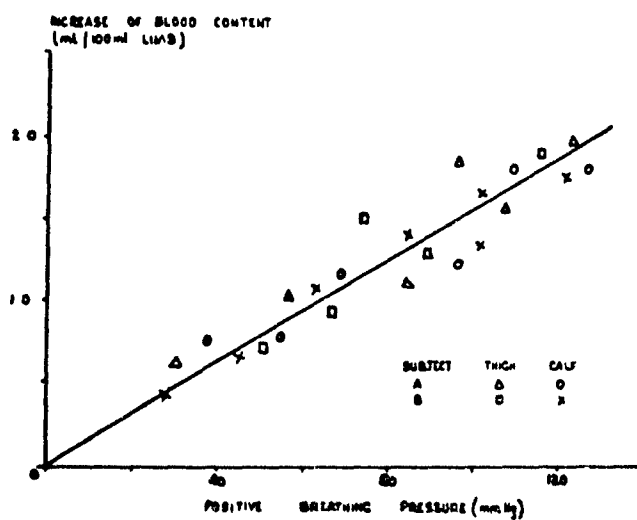
FIG. 6 4 The increase of the blood content of the thigh and calf induced by pressure
breathing with trunk counterpressure

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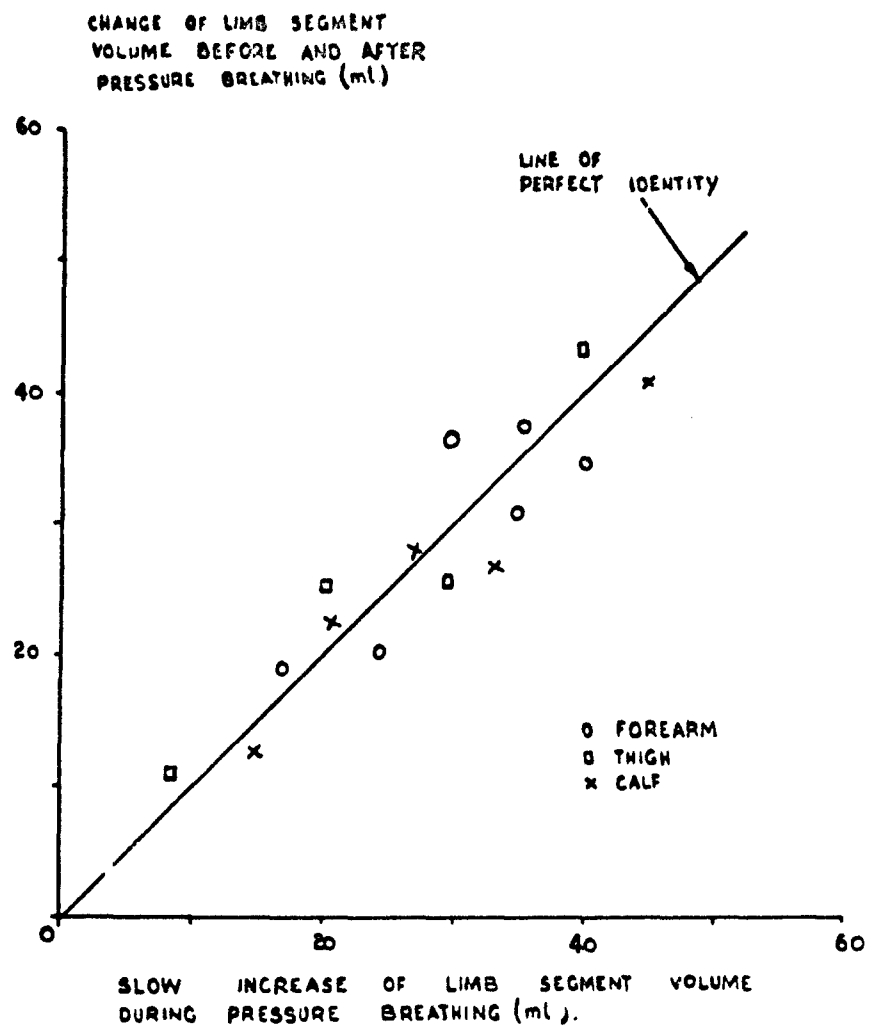
Fig. 6-5 The relationship between the increase of limb volume due to fluid filtration measured during pressure breathing and the change of limb volume between immediately before and one minute after pressure breathing

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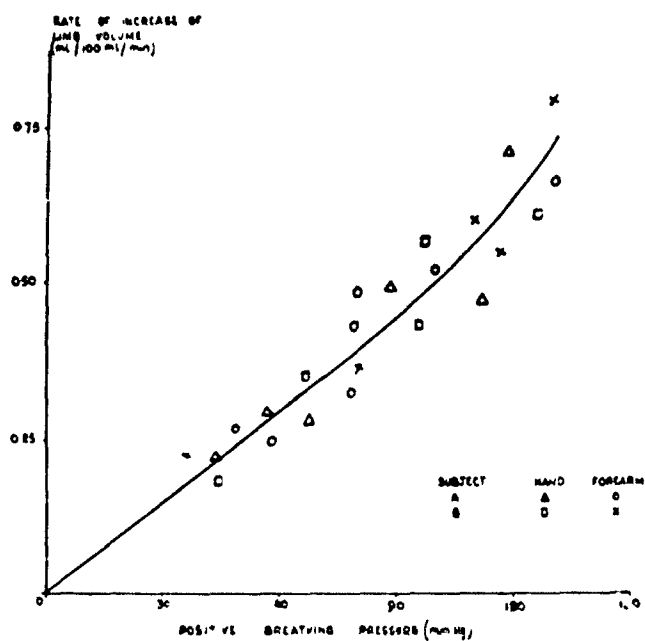
FIG. 6-6 The rate of increase of the volume of the hand and forearm due to fluid filtration during pressure breathing with trunk counterpressure
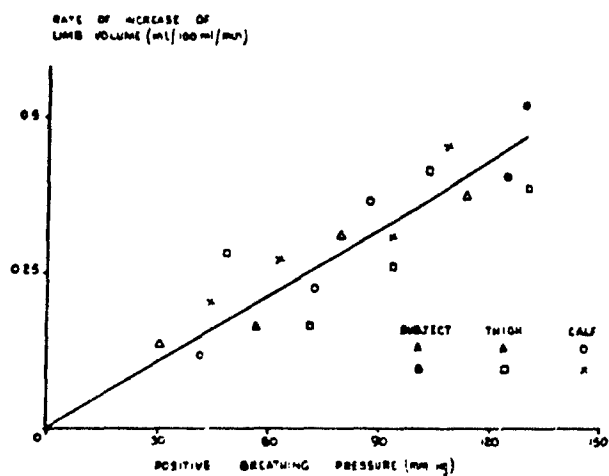
FIG. 6-7 The rate of increase of the volume of the thigh and calf due to fluid filtration during pressure breathing with trunk counterpressure

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continuously by means of a water-filled plethysmograph. The mid-plane of of the hand was 10 cm vertically below the sternal angle. The local venous pressure was recorded through a 20-gauge needle inserted in a suitable vein on the lateral aspect of the distal part of the forearm. The needle was connected by a polyethylene cannula to a capacitance pressure transducer which was placed in the horizontal plane which contained the tip of the intravenous needle. The electrical outputs from the amplifiers connected to the pressure transducer and the electrodes in the plethysmograph were fed on to two of the galvanometers of a photographic paper recorder. Simple vascular congestion of the hand was produced by inflating a standard sphygmomanometer cuff placed around the upper arm. Care was taken to ensure that when the cuff was uninflated it did not constrict the limb beneath it.

Two subjects, each of whom were experienced in pressure breathing, were studied. The blood content of the hand vessels was increased either by local congestion by the inflation of the sphygmomanometer cuff or by pressure breathing with or without trunk counterpressure. The positive breathing pressure used in a sphygmomanometer cuff were varied between 10 and 50 mmHg. Each exposure was maintained until the venous pressure had been constant for at least thirty seconds and each exposure was separated from the preceding one by a rest period of at least five minutes. The order of the exposures to the various procedures was randomized. In one experiment performed on each of the subjects, a total nerve block was performed at the wrist by infiltrating the median, ulnar and radial nerves and their branches with 2"₀ lignocaine hydrochloride. The nerve block produced complete analgesia below the wrist and paralysed the small muscles of the hand. Following the wrist block the hand was inserted in the plethysmograph and the needle placed in a vein just above the wrist and the blood content of the hand increased by local congestion and by pressure breathing.

Results – The increase in hand volume caused by the displacement of blood into the part was measured from each record from the start of the volume increase to the instant at which the venous pressure reached a plateau. The relationships between the increase of hand volume and the corresponding venous pressure obtained by local congestion, pressure breathing without respiratory counterpressure and pressure breathing with trunk counterpressure are presented in Fig. 6–8. The increase of hand volume associated with the rise of venous pressure to a given value was very similar when the distension was caused by pressure breathing with or without trunk counterpressure. During pressure breathing. however, the increase of hand volume associated with a given venous pressure was about 25"₀ less than the increase produced by local congestion. Very similar results were obtained with a second subject used in this investigation.

Following the nerve block at the wrist, the skin of the hand became warmer and bright red in colour. The rate of blood flow into the hand was calculated from the record of the increase of hand volume produced by the inflation of the sphygmomanometer cuff. The mean rate of blood flow was found to be 44 ml/100 ml of hand/min. in one subject and 40 ml/100 ml/min. in the other as compared with a mean blood flow of 5 ml/100 ml/min. in the non-nerve blocked hand. The response of the capacity vessels to local congestion obtained in the nerve-blocked hand is shown in Fig. 6–9. The resting venous
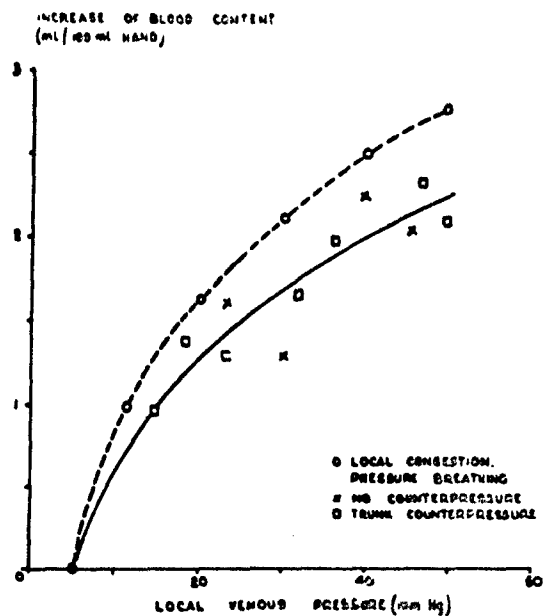
FIG. 6-8 The relationship between local venous pressure and the increase of the blood content of the hand during pressure breathing (solid line) and local congestion (interrupted line)
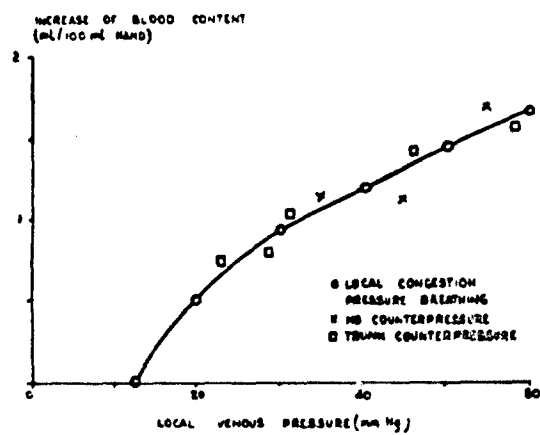

FIG. 6-9 The relationship between local venous pressure and the increase of the blood content of the hand during pressure breathing and local congestion following a total nerve block at the wrist

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pressure was greater than in the normal hand (Fig. 6-8). The relationships between the raised values of venous pressure and the corresponding increases of hand volume produced by pressure breathing with and without trunk counterpressure were very similar to that obtained with local congestion (Fig. 6-9). A nerve block at the wrist also abolished in the second subject the difference between the responses of the capacity vessels to pressure breathing and local congestion obtained in the normally innervated hand.

Peripheral Venous Pressure – The effects of pressure breathing without and with counterpressure applied to the trunk upon the peripheral venous pressure in the hand, arm and foot were studied in three subjects. The subject was fitted with a partial pressure headpiece and a pressure jerkin which were connected to a demand regulator, the outlet pressure of which could be varied between 0 and 150 mmHg. Venous pressure was measured through a 20-gauge needle introduced into the vein under study after the induction of local analgesia with 2"$_0$ lignocaine hydrochloride. The needle was connected by a 10 to 12 cm length of polyethylene cannula to a capacitance pressure transducer. The pressure transducer was placed in the same horizontal plane as that which contained the tip of the intravenous needle. The output of the pressure transducer was fed on to one galvanometer of a photographic recorder. The pressure in the headpiece was also measured and fed on to a second galvanometer of the recorder. In a few experiments the subject wore the modified pressure headpiece fitted with a mouthpiece so that the respiratory flow could be recorded with a Fleisch flow meter and a suitable pressure transducer placed between the mouthpiece and the valve box. The pressure in an antecubital vein was measured in the majority of the experiments when the subject was in the seated position. The arm was placed so that the tip of the needle was 10 cm vertically below the sternal angle. Care was taken to ensure that the arm did not move in relation to the sternal angle with the induction of pressure breathing. The remaining experiments in which records were obtained of venous pressure in the head, arm and foot were performed in a supine subject. A subject with a prominent superficial forehead vein was used for the study of the behaviour of the venous pressure in the head. In this instance an oronasal mask was used in place of a partial pressure helmet to deliver air at the desired positive breathing pressures. The venous pressure in the foot was recorded by means of a needle placed in a dorsal vein. On each occasion the desired positive breathing pressure was applied for ninety seconds and each exposure to pressure breathing was separated by a rest period of at least three minutes.

Results – The general behaviour of the peripheral venous pressure during pressure breathing was similar in the three sites from which it was recorded. Almost immediately after the beginning of pressure breathing, the venous pressure started to rise, the rate of rise increasing slightly with time until the pressure reached a value which was maintained throughout the remainder of the exposure (Fig. 6-10). The venous pressure returned very rapidly to the resting value directly pressure breathing ceased. The rate of rise of venous pressure at the beginning of pressure breathing in the forehead was considerably greater than that in the forearm which in turn was greater than that in the foot (Fig. 6-11).

Once the venous pressure had reached a plateau value during pressure

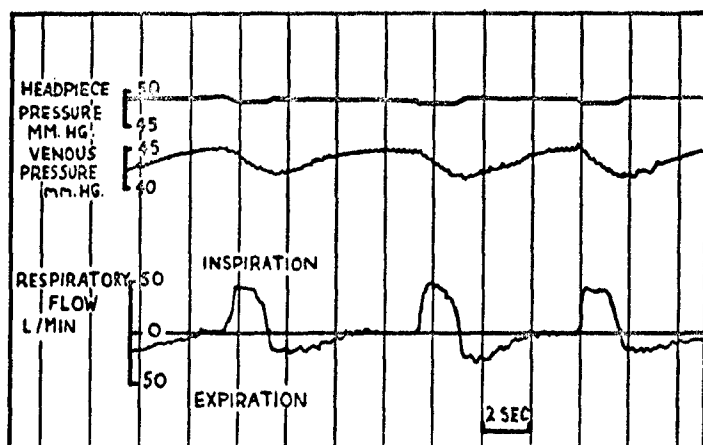FIG. 6-10 The effect of respiration, recorded as respiratory flow (bottom trace) upon pressure in the antecubital vein (middle trace) and headpiece pressure (top trace) during pressure breathing with trunk counterpressure at a positive pressure of 50 mmHg
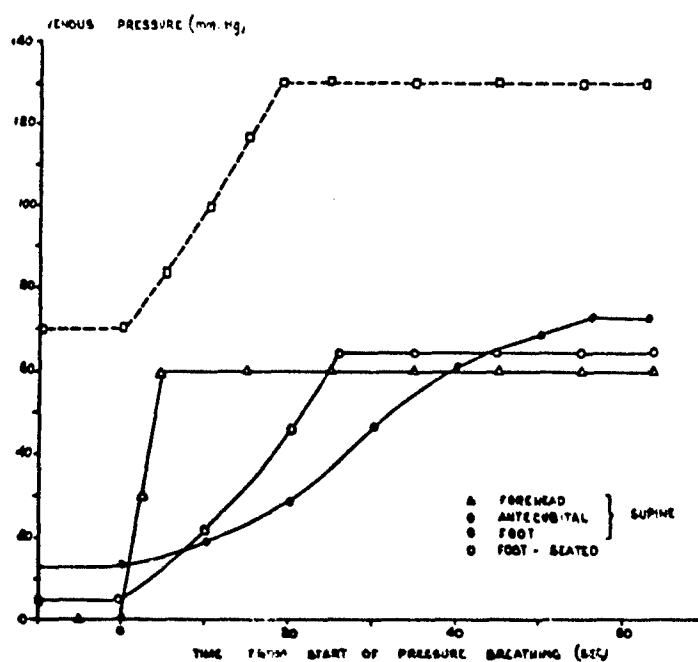

FIG. 6-11 The effect of pressure breathing with trunk counterpressure at a positive pressure of 65 mmHg upon the venous pressure in the forehead, forearm and foot

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breathing it exhibited respiratory fluctuations. The simultaneous records of mouthpiece pressure, respiratory flow and venous pressure (Fig. 6-10) demonstrated that the venous pressure fell during inspiration and rose during expiration. The total change of antecubital venous pressure during a respiratory cycle exceeded the corresponding change of mouthpiece pressure by about 2 mmHg during pressure breathing with trunk counterpressure and by about 4 mmHg during pressure breathing without respiratory counterpressure. The records of peripheral venous pressure obtained during pressure breathing also exhibited relatively small changes which were cardiac in timing (Fig. 6.10).

The increase of antecubital venous pressure measured as the difference between the resting value and the mean plateau value was determined for each exposure to pressure breathing and related to the corresponding positive breathing pressure. The results obtained from the three subjects for pressure breathing without respiratory counterpressure and for pressure breathing with trunk counterpressure are presented in Figs. 6-12 and 6-13 respectively. The relationship between the increase of venous pressure and the positive breathing pressure was more variable in the absence of respiratory counterpressure. For a given positive breathing pressure the rise of venous pressure was greater when trunk counterpressure was applied than when it was absent. With trunk counterpressure at a positive breathing pressure of 80 mmHg the rise of venous pressure was only 4 to 6 mmHg less than the positive breathing pressure.

Central Venous Pressure – The effect of pressure breathing with trunk counterpressure upon the pressure in the right atrium was determined in two subjects. With the subject in the supine position a large-bore needle was inserted into the median cubital vein of the right arm after the area had been infiltrated with local analgesic solution. One end of a one meter length of sterile fine polyethylene cannula (0.5 mm I.D.) was attached to a capacitance pressure transducer, the output of which was fed on to a cathode ray oscilloscope and a galvanometer of a photographic recorder. The measuring head of the pressure transducer and the polyethylene cannula were filled with sterile saline containing 2000 units of heparin per 100 ml of saline. The cannula was introduced under full aseptic conditions through the needle into the vein and the tip of the cannula advanced towards the heart with the subject's arm abducted to 90° and his head inclined towards his right shoulder. The position of the tip of the cannula was determined in two ways: The distance between the site of the venepuncture and the right atrium was measured from known topographical landmarks. It was assumed that when the length of cannula introduced into the venous system equalled this distance the tip lay within the right innominate vein, the superior vena cava or the right atrium. The position of the tip of the cannula was confirmed from the pattern of the pressure trace displayed on the screen of the oscilloscope. When the tip of the cannula was within the central venous region the characteristic right atrial pressure pattern was seen on the oscilloscope screen. When the position of the cannula was satisfactory the needle through which it had been introduced was withdrawn from the vein and the puncture site covered with a dressing. In order to prevent bleeding during pressure breathing a sphygmomanometer cuff was then wrapped around the elbow so that it covered the site of intro-
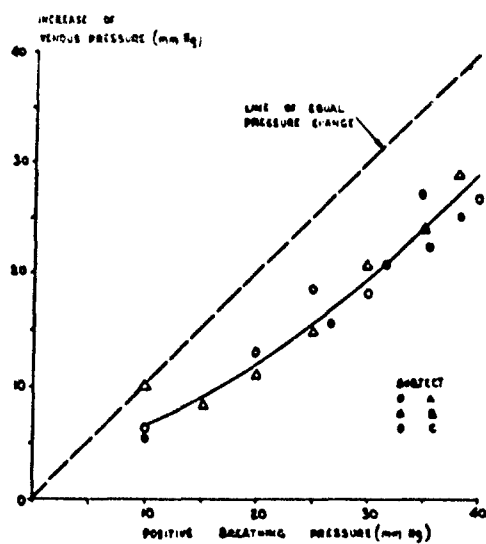
Fig. 6-12 The increase of forearm venous pressure induced by pressure breathing
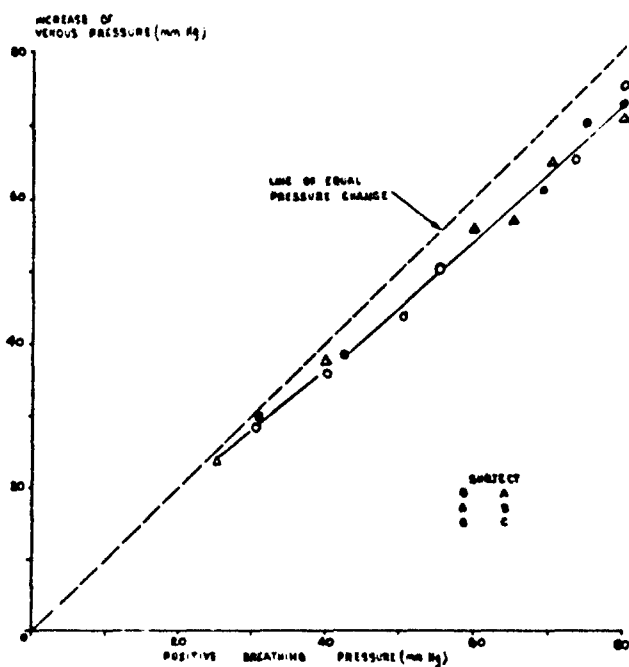without respiratory counterpressure

Fig. 6-13 The increase of forearm venous pressure induced by pressure breathing
with trunk counterpressure

duction of the polyethylene cannula. The bladder of the sphygmomanometer cuff was connected to the outlet of the regulator which was used to inflate the pressure headpiece and jerkin. After the introduction of the cannula the subject seated himself in an ejection seat, the harness of which was firmly secured in order to reduce the movement of the subject to a minimum. The pressure transducer was placed in a horizontal plane which was 5 cm vertically below the sternal angle.

The intraoesophageal pressure was measured by means of a standard balloon placed in a lower portion of the oesophaɡ   and connected to a suitable transducer. The right atrial and intraoesophageal pressures and the electrocardiogram were recorded at rest and during pressure breathing with a pressure headpiece and trunk counterpressure at positive pressures of 30, 60 and 80 mmHg. Each exposure to pressure breathing was limited to two minutes and it was separated from the one which preceded it by a rest period of at least ten minutes.

Results – The shape of the pressure changes recorded from the right atrium in the resting subject varied with respiration (Fig. 6-14). In most cardiac cycles it was possible to distinguish three positive waves, two of which were well defined. The first wave, the "a" wave, coincided with the P-R interval of the electrocardiogram and was produced by atrial systole. The descending limb of this wave generally had a small step which, in certain phases of the respiratory cycle could be distinguished as a positive wave. This wave, the "c" wave occurred during the ST segment of the electrocardiogram and was associated therefore with ventricular systole. The atrial pressure continued to fall following the "c" wave until a time which generally coincided with the T wave of the electrocardiogram when the pressure began to rise as the ascending limb of a third positive wave, the "v" wave. The "v" wave ended at the beginning of the next cardiac cycle. The right atrial pressure fell during inspiration and increased during expiration. The change in mean atrial pressure during the respiratory cycle amounted to about half the corresponding change of intraoesophageal pressure.

Pressure breathing caused a marked increase in the pressure in the right atrium (Fig. 6-14). The rate at which the right atrial pressure increased equalled that at which the pressure in the oesophagus was raised. The cardiac fluctuations in the right atrial pressure record were markedly reduced at the beginning of pressure breathing whilst the intraoesophageal pressure was rising. Pressure breathing induced an increase of heart rate and changed the appearance of the right atrial pressure record. In contrast to the relationship which existed during rest the respiratory fluctuations of right atrial pressure in pressure breathing were very similar in magnitude to the corresponding changes in intraoesophageal pressure. The pattern of the right atrial pressure fluctuations also varied markedly with respiration. During expiration the "a" wave was smaller, the increase of pressure which occurred towards the end of the previous cardiac cycle continuing at the same rate up to the peak of the "a" wave. The "a" wave was followed by a deep trough and then a well marked "c" wave. The "v" wave was small. During inspiration however, the amplitude of the "a" wave was increased and the "c" wave became less prominent.

The mean value of the right atrial pressure at the end of expiration was
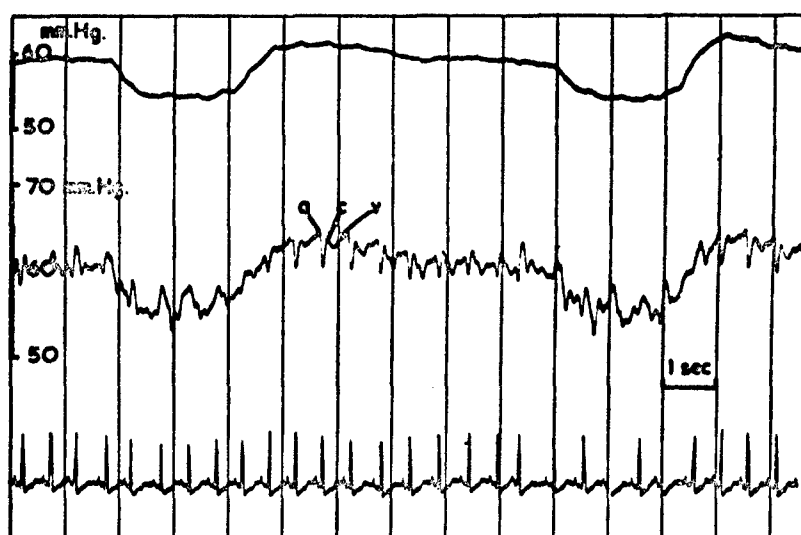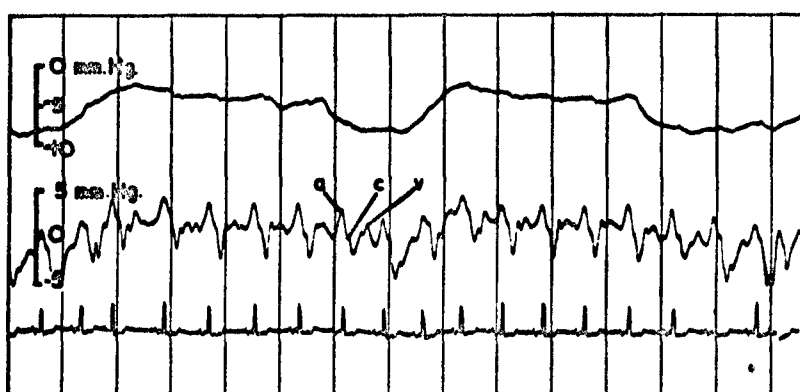
FIG. 6-14 Simultaneous records of the intraoesophageal pressure (top trace), right atrial pressure (middle trace) and the electrocardiogram (bottom trace) in a seated subject at rest and whilst pressure breathing at a positive pressure of 60 mmHg

determined in each condition studied. The mean atrial pressure over the cardiac cycle which coincided with the end of expiration was estimated by planimetric integration of the pressure curve for the cycle. This measurement was made for the five to eight respiratory cycles which occupied the last thirty seconds of each two minute exposure to pressure breathing. The corresponding values of the intraoesophageal pressure were determined from the record and the difference between each value of the right atrial pressure and the corresponding intraoesophageal pressure calculated. The mean of the five to eight values of the right atrial-intraoesophageal pressure difference (effective right atrial pressure) obtained for each experimental condition, was calculated and plotted against the corresponding positive breathing pressure (Fig. 6–15). In both subjects the effective right atrial pressure fell progressively as the positive breathing pressure increased.

Systemic Arterial Pressure – The effect of pressure breathing at various positive breathing pressures and with varying degrees of counterpressure applied to the trunk and limbs upon the systemic arterial pressure was measured by direct puncture of the brachial artery in several experiments on each of six subjects.

The subject, wearing the appropriate pressure clothing assembly, sat in an ejection seat, the harness of which was secured firmly in order to reduce to a minimum movement of the subject in inflation and deflation of the pressure clothing. The brachial artery pressure was measured by introducing a Riley needle into the artery and connecting it to a capacitance pressure transducer. The pressure transducer and the tip of the intra-arterial needle were placed in the same horizontal plane which was 10 cm vertically below the sternal angle. The pressure in the partial pressure headpiece was also measured with a strain gauge pressure transducer. The output of the transducer amplifiers were fed on to the galvanometers of a photographic recorder.

Pressure breathing was performed using a partial pressure helmet with either no counterpressure applied to the body or varying degrees of counterpressure applied to the surface of the body. In different exposures counterpressure was applied to the chest alone with the pressure breathing waistcoat, to the trunk alone with the pressure jerkin and to the trunk and lower limbs with a pressure jerkin and an anti-g suit. In the majority of experiments each exposure to pressure breathing was limited to one and a half minutes. On some occasions, however, the duration of the exposure was prolonged to four or five minutes. Each exposure to pressure breathing was separated from the preceding one by a rest period of at least three minutes duration.

Results – Pressure breathing always increased the systemic arterial blood pressure. The general form of the change was independent of the degree of positive breathing pressure employed and the counterpressure applied to the body. Both the systolic and diastolic pressures increased as the pressure in the headpiece was raised (Fig. 6–16). The arterial pressure was raised for as long as the positive pressure was applied to the respiratory tract. The respiratory fluctuations of arterial pressure were more prominent during pressure breathing. The heart rate increased with the induction of pressure breathing whilst the pulse pressure fell as the pressure in the headpiece was increased. The pulse pressure was reduced below the resting value throughout the pressure breathing period. The shape of the arterial pressure wave was changed by
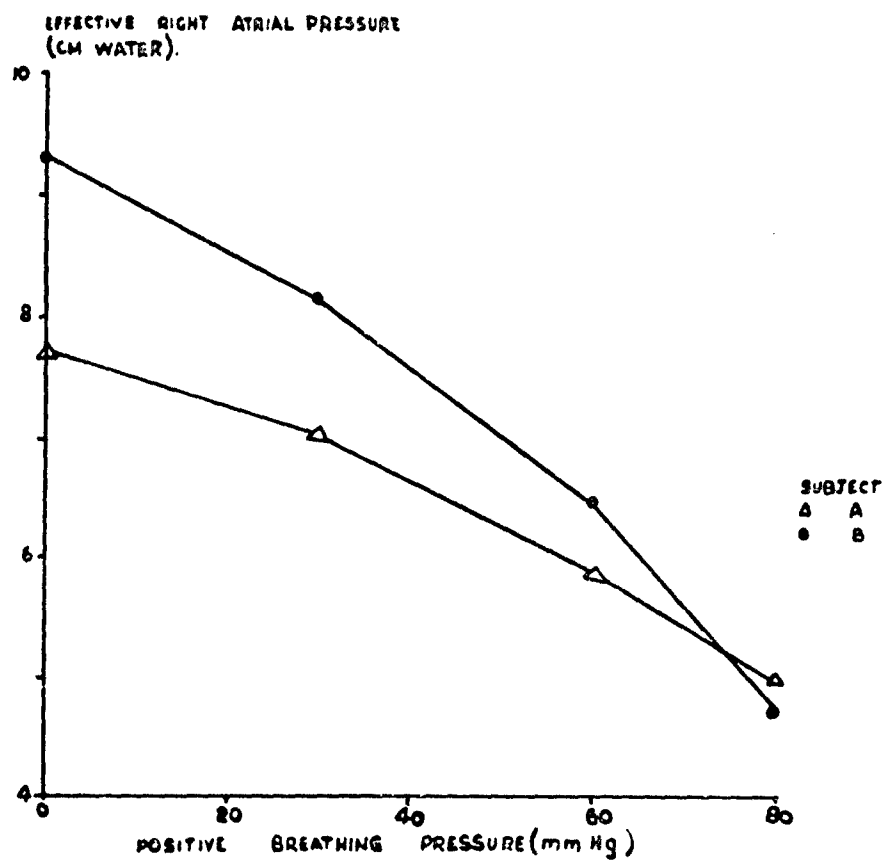
FIG. 6 15 The effect of pressure breathing with trunk counterpressure upon the 'effective' right atrial pressure (right atrial pressure minus intraoesophageal pressure) measured at the end of expiration

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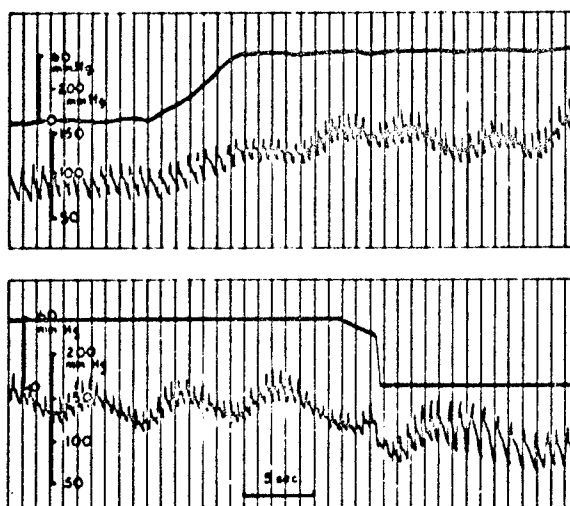
Fig. 6-16 The effect of pressure breathing with trunk counterpressure at a positive pressure of 60 mmHg upon the pressure in the brachial artery (lower trace). The headpiece pressure is displayed in the upper trace
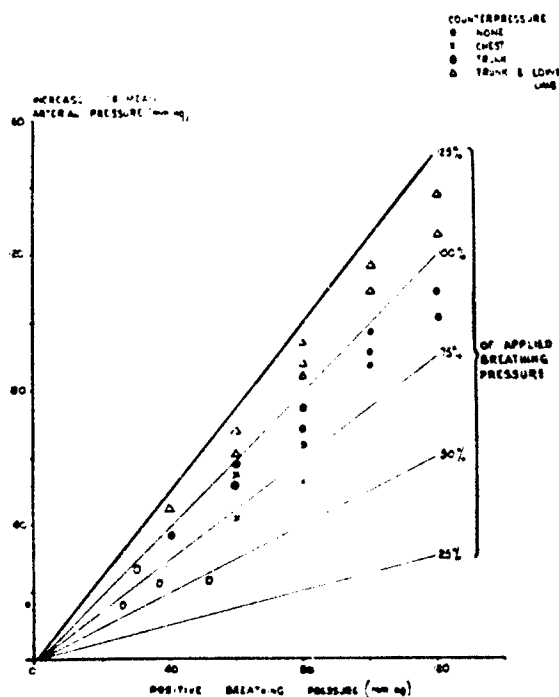
Fig. 6-17 The relationship between the positive breathing pressure and the increase in mean arterial pressure during pressure breathing with a pressure headpiece and various degrees of counterpressure applied to the body

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pressure breathing, the dicrotic notch and wave being much more prominent during pressure breathing than at rest. With the cessation of pressure breathing the arterial pressure fell rapidly with the pulse pressure remaining low for three to four beats (Fig. 6-16). This preliminary fall was followed by several beats in which the pulse pressure was very large. Thus following pressure breathing at a positive breathing pressure of 80 mmHg the maximum pulse pressure was 95 mmHg as compared with a resting pulse pressure of 45 mmHg. The very large pulse pressure which occurred three to five seconds after the cessation of pressure breathing was also associated with a marked cardiac slowing and a rise of the diastolic pressure. The pulse pressure and diastolic pressures then declined over twenty to thirty seconds to regain the values which existed before pressure breathing was started.

The magnitude of the increase of arterial pressure produced by pressure breathing varied with the positive breathing pressure and with the degree of counterpressure applied to the body. The mean arterial pressures over the last thirty seconds of rest and over the last thirty seconds of the period of exposure to pressure breathing were obtained from each record of arterial pressure. The increase of the mean pressure produced by pressure breathing was then related to the positive breathing pressure. There was only a small variation between the responses of different subjects to a given intensity of pressure breathing under the same conditions, so all the results obtained in this study are presented together in Fig. 6-17. For a given degree of counterpressure the increase of mean arterial pressure was directly proportional to the positive breathing pressure. At a specified positive breathing pressure the increase in mean arterial pressure was minimal with no counterpressure and rose progressively with pressure applied to chest, trunk and trunk with limbs, reaching a value which frequently exceeded the applied positive breathing pressure. The arterial pressure records were anyalyzed further by measuring the mean pulse pressure during the last thirty seconds of the rest and of the pressure breathing periods. The results of these calculations are presented in Table 6-2. Pressure breathing per se reduced the pulse pressure but the application of counterpressure raised the pulse pressure above the value achieved by pressure breathing alone.

**The Heart Rate** – One of the standard limb leads of the electrocardiogram, generally lead II, was recorded in many of the experiments in which subjects were exposed to pressure breathing at various positive breathing pressures and with various degrees of respiratory counterpressure. In all these experiments the subject was in the seated position and had rested for at least three minutes before pressure breathing was initiated. In a small series of experiments simultaneous records were taken of the three standard limb leads.

Two specific series of experiments were performed on six subjects. In the first series of experiments each subject was exposed on a number of occasions to a positive breathing pressure of 60 mmHg for a period of three minutes using a partial pressure helmet. Duplicate experiments were performed with each subject wearing four different combinations of pressure clothing, viz., the pressure breathing waistcoat, the pressure jerkin, the pressure jerkin and the anti-g suit and the arm jerkin and anti-g suit. The second series of experiments consisted of exposing each of the subjects whilst wearing a pressure jerkin on two separate occasions to three different positive breathing

pressures. The positive breathing pressures used were 40, 70 and 100 mmHg and the duration of each exposure to the two lower pressures was three minutes whilst the exposure to the highest pressure was limited to one and a half minutes. In both series of experiments the order of exposures was randomized with regard to positive breathing pressure and combination of garments.

Results – In general no gross electrocardiographic abnormalities were seen during positive pressure breathing at ground level unless syncope occurred. In subjects who exhibited an occasional premature systole at rest this abnormality did not arise during pressure breathing. When lead II of the electrocardiogram was recorded only small changes were seen in the shape of the Q.R.S. complex during pressure breathing but records of the three standard limb leads showed that in certain circumstances pressure breathing caused distinct changes in the shape of the Q.R.S. complex (Fig. 6–18). With the induction of pressure breathing the R wave in lead I became smaller, whilst in lead III the height of the S wave was decreased. At a given positive breathing pressure these electrocardiographic changes were most marked when pressure breathing was performed without respiratory counterpressure and they were least apparent when counterpressure was applied to the whole trunk (Fig. 6–18). With a given degree of counterpressure the changes became more prominent as the positive breathing pressure was increased.

The magnitude of the effect of pressure breathing upon the heart rate in a given individual varied with the positive breathing pressure employed, the duration of the exposure and the degree of counterpressure applied to the body. At low positive breathing pressures and where counterpressure was applied to the greater part of the body the heart rate was either unchanged or only slightly increased by pressure breathing. At positive breathing pressures above 40 mmHg pressure breathing with either no counterpressure or only trunk counterpressure always caused an increase of heart rate. Thus pressure breathing at 60 mmHg with trunk counterpressure caused an almost immediate increase of the heart rate (Fig. 6–19). The heart rate was increased rapidly during the first thirty seconds of pressure breathing and then the rate of increase generally fell. Generally, however, when the positive breathing pressure exceeded 40 mmHg and only trunk counterpressure was applied, the heart rate continued to rise throughout the exposure to pressure breathing.

The electrocardiographic records obtained in the two series of specific experiments were analyzed by counting the number of heart beats in each thirty second period and calculating the corresponding heart rate in beats per minute for each of the intervals. The mean values for the heart rate for each thirty second interval over the last minute of the rest period, during pressure breathing and for the first two minutes of the recovery period for the two series of experiments are presented in Figs. 6–19 and 6–20. The rate of in-increase of heart rate and the maximum heart rate induced by pressure breathing at 60 mmHg were greatest when counterpressure was applied to the chest alone and were minimal when counterpressure was applied to the trunk and upper and lower limbs Fig. 6–19. When trunk counterpressure was used the rate of rise and the maximum value of the heart rate both increased as the positive breathing pressure was increased Fig. 6–20.

Two subjects were exposed on one occasion each to a positive breathing pressure of 130 mmHg whilst wearing a partial pressure headpiece, pressure
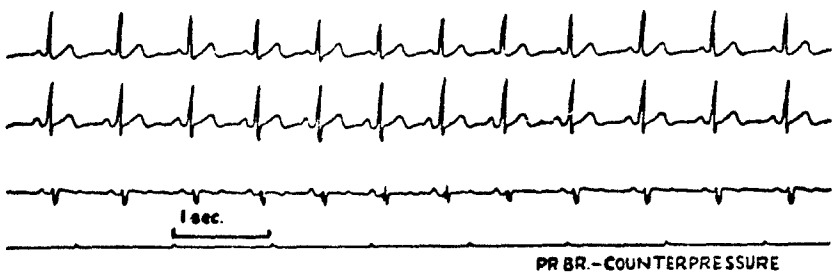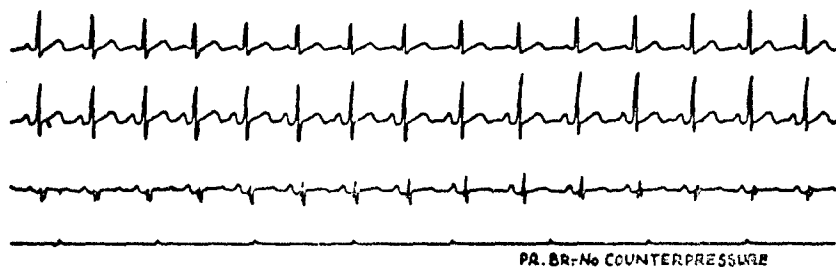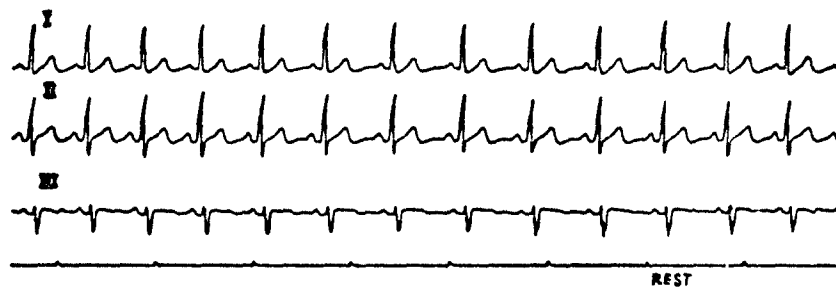
FIG. 6-18 The effect of pressure breathing at a positive pressure of 40 mmHg with and without trunk counterpressure upon the three standard limb leads of the electrocardiogram
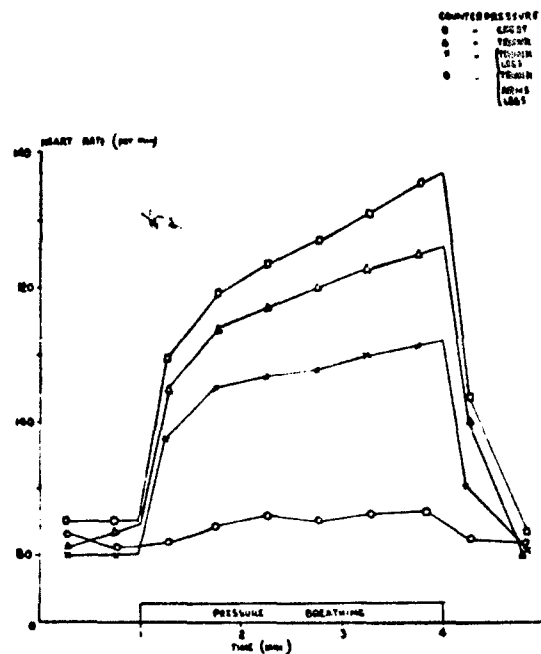
Fig. 6-19 The effect upon the heart rate of pressure breathing at a positive pressure of 60 mmHg with a pressure headpiece and with varying degrees of counterpressure applied to the body. Each point is the mean of the values obtained in duplicate experiments on each of six subjects
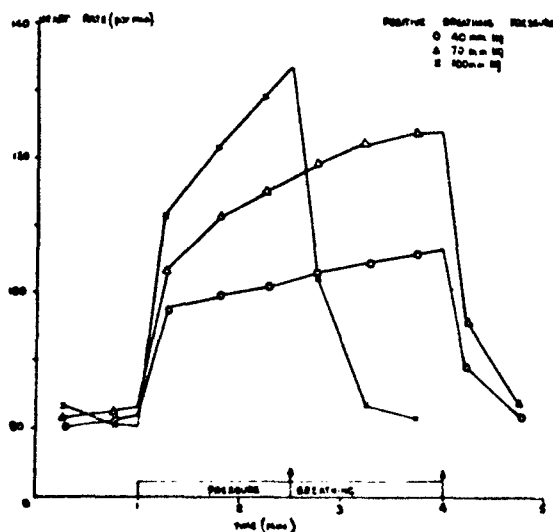
Fig. 6-20 The effect upon heart rate of pressure breathing with trunk counterpressure at various positive breathing pressures. Each point is the mean of the values obtained in duplicate experiments on each of six subjects

jerkin and anti-g suit. This procedure rapidly produced a very high heart rate (Fig. 6-21). In an attempt to reduce arm discomfort the subject periodically tensed the muscles of the upper limbs and flexed the elbows. This manoeuvre was associated with a marked but temporary reduction of the heart rate.

Duration of Protection at Ground Level – Several series of exposures to positive pressure breathing were performed at ground level with various degrees of counterpressure applied to the body in order to define the time course of the cardiovascular effects in a number of subjects. Two different groups of subjects were used in these experiments. One group consisted of members of the staff of the Institute of Aviation Medicine who had considerable previous experience of pressure breathing. The other group consisted of aircrew volunteers who had no previous experience of pressure breathing at positive breathing pressures above 30 mmHg. The latter group was always given a series of exposures to pressure breathing over one or two days before the final experimental determination of their tolerance to high positive pressure breathing. This training was performed using the assembly of pressure clothing which was to be worn in the final experiment. It consisted of familiarization with the equipment and adjustment of fit so as to ensure the maximum possible degree of comfort. The positive breathing pressure and the duration of exposure to it were gradually increased in this series of preliminary experiments.

The subject was strapped in an ejection seat and was at rest for at least five minutes before the exposure to pressure breathing was started. The experiments were performed in a room, the temperature of which varied between 18° and 20°C. The duration of the exposure to a given breathing pressure was varied with the degree of counterpressure worn by the subject (Table 6-3).

In each experiment lead II of the electrocardiogram was recorded continuously and the arterial blood pressure was measured by the indirect technique using a sphygmomanometer cuff and a piezoelectric transducer.

An exposure to pressure breathing was terminated if the subject signified that symptoms of an impending collapse had arisen. The subject was closely observed and pressure breathing was stopped directly there was any impairment of consciousness as indicated by a failure of the subject to respond to a simple command. An exposure was also terminated if the heart rate suddenly fell to a value of less than eighty beats per minute or if the systolic pressure fell by more than 30 mmHg. In the event of a collapse the subject remained seated in the ejection seat until his heart rate and arterial pressure approached the resting value. The pressure headpiece or oronasal mask was usually removed directly pressure breathing was stopped. Facilities were available for placing the subject supine with his head 20 cm lower than his feet should he fail to regain consciousness within ten seconds of the cessation of pressure breathing. A subject was not exposed to pressure breathing for at least twenty-four hours following a syncopal attack.

Results – The overall results of these experiments are summarized in Table 6-3. Most of the subjects, both experienced and inexperienced, successfully completed the various exposures to pressure breathing. The cause of a premature termination of an exposure to pressure breathing was always the development of a syncopal attack.

TABLE 6-3

INCIDENCE OF SYNCOPAL ATTACKS DURING PRESSURE
BREATHING AT GROUND LEVEL

Positive breathing pressure (mmHg)	Duration of exposure (min)	Previous experience	Number of subjects	Syncopal attacks	
				Number	Proportion of subjects
A. Helmet and Jerkin					
80	4	Considerable	9	0	0
80	4	Minimal	15	2	14"₀
80	2	Moderate	20	0	0
B. Helmet, Jerkin and anti-G suit					
80	7½	Considerable	25	0	0
80	7½	Minimal	50	5	10"₀
80	5	Moderate	50	0	0
107	5	Considerable	12	2	17"₀
107	3	Moderate	15	0	0
107	2	Minimal	50	0	0
120	3	Considerable	9	1	11"₀
133	2	Moderate	15	0	0
C. Helmet, Arm Jerkin and anti-G suit					
80	10	Considerable	10	0	0
110	6	Moderate	20	0	0
110	4	Minimal	20	1	5"₀
140 for 1 min. decaying to 0 in 5 min.		Minimal	20	1	5"₀
		Moderate	12	0	0
D. Mask, Jerkin and anti-G suit					
60	6	Considerable	6	0	0
60	2	Minimal	22	0	0
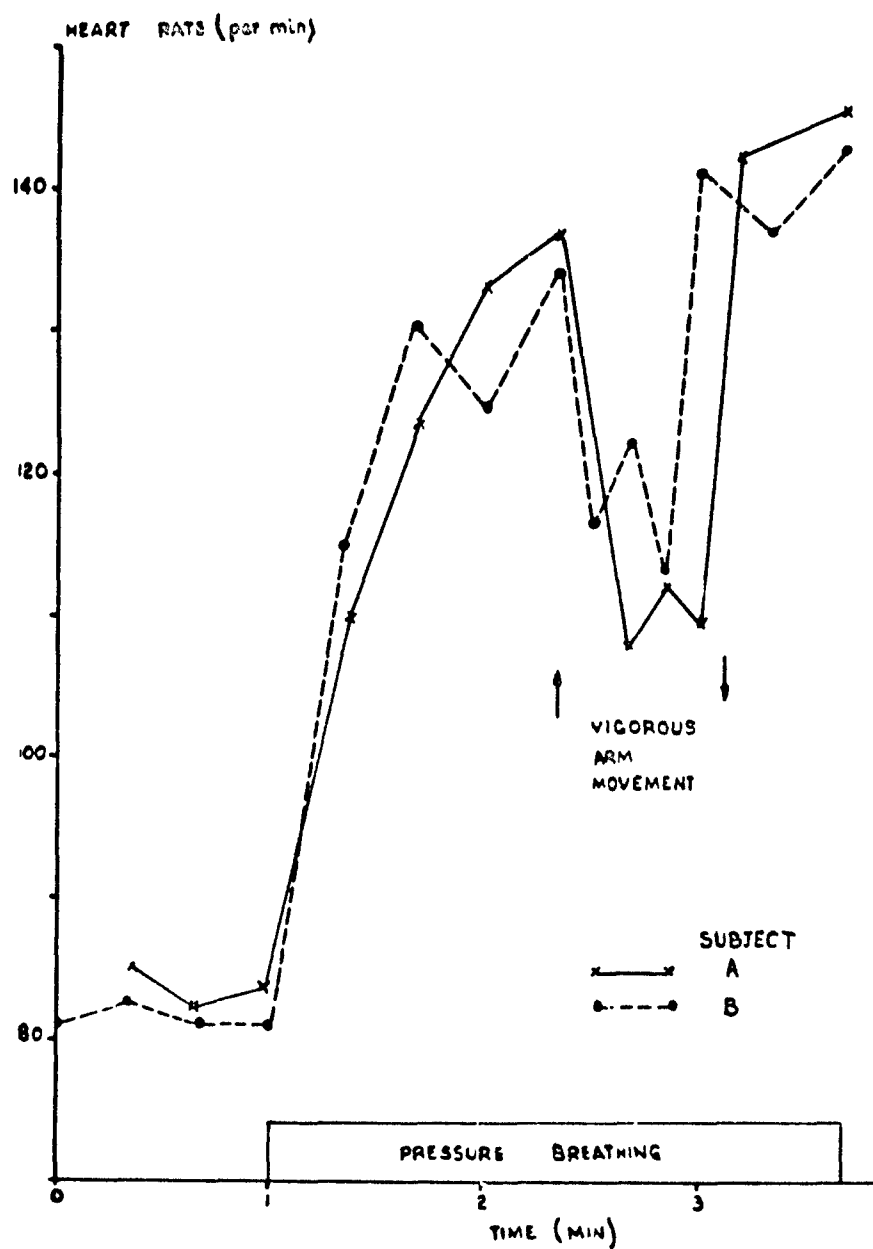
FIG. 6-21 The effect of upper limb movement during pressure breathing at a positive pressure of 130 mmHg with trunk and lower limb counterpressure upon the heart rate
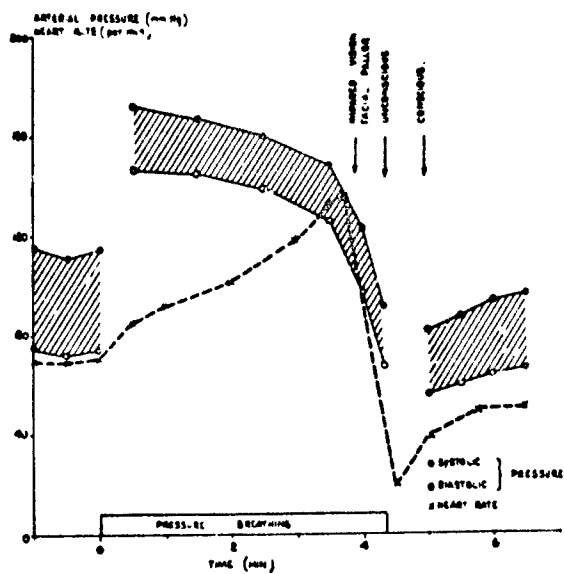
FIG. 6-22 The arterial blood pressure and heart rate during an exposure to pressure breathing with trunk counterpressure at 80 mm/Hg which was terminated by a syncoptal attack
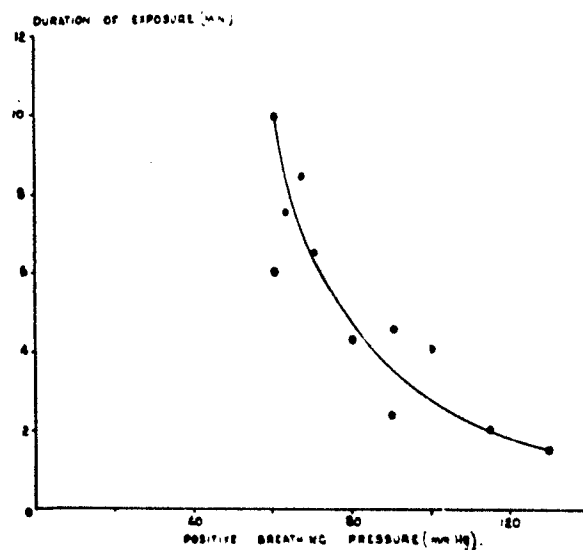
FIG. 6-25 The relationship between the time at which syncope occurred and the positive breathing pressure during pressure breathing with trunk counterpressure

The clinical features of syncope induced by pressure breathing varied only slightly from one individual to another. The onset of syncope was generally heralded by a feeling of nausea and uneasiness. These symptoms were usually preceded or accompanied by the sudden onset of sweating, especially on the face and hands. At this stage there was marked facial pallor. The symptoms were rapidly followed by dimming of vision and then complete loss of vision. Unconsciousness occurred within a further few seconds. Loss of consciousness was sometimes accompanied by a diminution of postural tone, particularly in the limbs. On several occasions unconsciousness was associated with spasmodic movement of the limbs which were sometimes followed by a major epileptiform convulsion. Recovery of consciousness followed rapidly upon cessation of pressure breathing. Following syncope the subject usually exhibited facial pallor and often complained of slight nausea from half to one hour. Although pressure breathing was nearly always stopped when the subject developed facial pallor and dimming of vision, consciousness was lost in about half of the instances in which syncope terminated an exposure to pressure breathing.

The changes of heart rate and arterial blood pressure which occurred during the development of syncope were very similar on all occasions. The heart rate and arterial blood pressure during a typical syncopal attack are presented in Fig. 6-22. During one or two minutes before syncope occurred the heart rate increased rapidly to attain a value of between 130 and 170 beats per minute when collapse was imminent. Over the same period the arterial pressure, both systolic and diastolic declined. The rate of fall of arterial pressure suddenly accelerated and the heart rate suddenly fell, usually to a rate of less than 60 beats per minute. These sudden cardiovascular changes were associated with the impairment and loss of vision and finally loss of consciousness already described. At this point pressure breathing was always terminated. This was followed by a slow increase of heart rate and a rise of arterial blood pressure towards the control values which pertained before pressure breathing was commenced.

The primary factors which determined the incidence of syncope during pressure breathing were the positive breathing pressure, the duration of the exposure and the degree of counterpressure applied to the body (Table 6-3). The incidence of syncope was also influenced by the previous experience of the subject of high pressure breathing. Other factors being equal the incidence of collapse was lower in experienced subjects than in those who had only a very limited experience of this procedure. Two-thirds of the subjects who had a syncopal attack experienced severe discomfort or frank pain during the exposure to pressure breathing (Table 6-4). Whilst 67"„ of the subjects who developed pressure breathing syncope had a previous history of vasovagal syncope, only 15"„ of the subjects who did not develop pressure breathing syncope had a previous history of syncope.

A number of experiments were performed in which three subjects, each of whom had had considerable previous experience of pressure breathing, were exposed to various positive breathing pressures whilst using trunk counterpressure alone. Particular care was taken in these experiments to ensure that the pressure headpiece and jerkin fitted the subject correctly. Each of these exposures was continued until the subject developed symptoms of an impending

TABLE 6–4

CIRCUMSTANCES OF SYNCOPAL ATTACKS
DURING PRESSURE BREATHING AT GROUND LEVEL

Positive breathing pressure (mmHg)	Previous experience	Duration of exposure (min.)	Previous syncopal attacks	Special factors present
A. Helmet and Jerkin				
80	Minimal	2½	nil	Headpiece discomfort
80	Minimal	3½	††	Hyperventilation and arm pain
B. Helmet, Jerkin and anti-G suit				
		4½	††	Intense dislike of headpiece
		4½	nil	Marked hyperventilation
80	Minimal	5½	†	Hyperventilation
		6	††	----
		6½	nil	Arm pain
107	Considerable	3½	†	Arm pain and head pain
		4	†	Arm pain: hyperventilation
120	Considerable	2½	nil	Severe arm pain
C. Helmet, Arm Jerkin and anti-G suit				
110	Minimal	3½	†	Headpiece pain
140 decaying to 0	Minimal	3	††	Hyperventilation and arm pain

† –→ †† increasing incidence of syncopal attacks

TABLE 6–5

SOURCES AND INCIDENCE OF DISCOMFORT
AND PAIN DURING PRESSURE BREATHING

Positive breathing pressure (mmHg)	Incidence of discomfort or pain		
	Upper arm	Neck	Head
A. Helmet, Jerkin with or without anti-G suit			
80	60"„ discomfort 15"„ pain	15"„ discomfort 5"„ pain	5"„ discomfort
107	50"„ discomfort 30"„ pain	20"„ discomfort 20"„ pain	10"„ discomfort
B. Helmet, arm jerkin and anti-G suit			
110	10"„ discomfort	25"„ discomfort 20"„ pain	10"„ discomfort
140 decaying to 0	20"„ discomfort	30"„ discomfort 25"„ pain	50"„ discomfort

collapse. In every instance the electrocardiogram showed that the symptoms were accompanied by a sudden and profound bradycardia. In no instance did the subject suffer any severe discomfort or pain during the exposure to pressure breathing. The relationship between the positive breathing pressure and the duration of the exposure when syncope occurred is presented in Fig. 6-23. The duration of an exposure to pressure breathing when syncope arose decreased as the positive breathing pressure was increased.

A considerable proportion of the subjects experienced some discomfort during pressure breathing, particularly at the higher positive breathing pressures. The incidence of the main types of discomfort which occurred during positive pressure breathing with either a standard pressure jerkin or an arm jerkin and a partial pressure headpiece and anti-g suit are presented in Table 6-5. There were two main sites of severe discomfort, namely the neck and upper limb. The pain in the neck arose in association with the absence of counterpressure to the lower part of this region. Severe pain generally arose when the lower edge of the neck bladder of the headpiece lay across the larynx. At positive breathing pressures above 110 mmHg discomfort occurred in association with a tendency of the headpiece to rise up over the head. In the absence of counterpressure to the upper limbs most subjects complained of some discomfort in the upper arm during exposure to positive breathing pressures above 60 mmHg lasting for more than one minute. The discomfort was generally located on the medial aspect of the upper part of the upper arm. It was of the ill-localized sickening type. The intensity and incidence of this discomfort increased as the positive breathing pressure was raised above 80 mmHg. A proportion of subjects experienced frank pain in the upper arms when pressure breathing at positive breathing pressures above 80 mmHg. The occurrence of severe pain was sometimes associated with a syncopal attack. The discomfort or pain in the upper limbs could be relieved temporarily by tensing the muscles of the upper arms.

In these experiments the subject's skin was also inspected following each exposure to pressure breathing. When the positive breathing pressure exceeded 60 mmHg pressure breathing produced petechial haemorrhages in the skin, the incidence of petechial haemorrhages depending upon the magnitude of the positive breathing pressure and the duration of the exposure. Thus few petechiae were seen after pressure breathing at 40 mmHg for ten minutes whilst an exposure to a positive breathing pressure of 80 mmHg for five minutes always produced large showers of minute haemorrhages in the skin. At a positive breathing pressure of 130 mmHg many petechiae were present after an exposure lasting only one minute. The distribution of these haemorrhages was relatively constant. They were most numerous in the skin at the upper border of the pressure jerkin beyond the armholes and above the neck of the garment. The proximal border of the rash was clearly defined and coincided with the line of reflection of the bladder of the jerkin off the skin of the trunk. Distally the density of the petechiae fell off so that few were seen beyond the deltoid muscle or on the upper part of the neck. Petechiae were only seen in the forearm and hand at positive breathing pressures above 120 mmHg. No petechiae occurred in the lower limbs. Occasionally at positive breathing pressures in excess of 100 mmHg petechiae were found on

the trunk beneath the pressure jerkin. The distribution of these haemorrhages coincided with the folds in the inner layer of the garment.

The Effect of Hypoxia – The reactions of the individual to pressure breathing in the presence of hypoxia were investigated using two techniques. In one group of experiments acute hypoxia was produced by exposing the subject who was breathing air to an absolute pressure of either 349 or 380 mmHg. Pressure breathing was then induced without changing the absolute intrapulmonary pressure. In the other group of experiments hypoxia was produced by exposing the subject, who was breathing 100"₀ oxygen, to pressure breathing with an intrapulmonary pressure of less than 190 mmHg absolute.

Pressure Breathing with Air – Each of the thirteen subjects used in this study had had considerable experience of high-pressure breathing. The subject wore a pressure jerkin and a partial pressure headpiece and was secured in an ejection seat placed in one compartment of the decompression chamber. The inlet hose to the pressure jerkin and pressure headpiece was connected to a two-way tap by means of which the subject could be made to breathe from either of the two compartments of the decompression chamber. The pressure in the pressure headpiece relative to that in the subject's compartment was measured by means of a mercury manometer. The electrocardiogram (lead II) was recorded continuously for one minute before and throughout the exposure to pressure breathing. Two observers were in the decompression chamber with the subject throughout the experimental procedure. The observers breathed oxygen when the pressure within the decompression chamber was reduced to below 520 mmHg absolute.

The effect of pressure breathing at a positive breathing pressure of 52 mmHg was investigated in each subject at three different absolute intrapulmonary pressures:

(a) 740–770 mmHg absolute (inspired oxygen tension calculated for gas saturated with water vapour at body temperature of 144–153 mmHg.) The pressure in the subject's compartment was reduced to 52 mmHg less than the prevailing barometric pressure, whilst the subject was breathing air from within the compartment. Pressure breathing was instituted by turning the tap so that the jerkin and headpiece were connected to the other compartment which was at atmospheric pressure. Pressure breathing was continued for two minutes.

(b) 380 mmHg absolute (inspired oxygen tension, calculated for gas saturated with water vapour at body temperature of 69 mmHg.) The subject breathed air throughout the procedure. The doors which separated the two compartments were open and the pressure within the compartments reduced to 380 mmHg absolute in four minutes. The recording of the electrocardiogram was started immediately the pressure reached 380 mmHg absolute and the doors between the compartments were closed. One minute after a chamber pressure of 380 mmHg absolute was attained the pressure headpiece and jerkin were connected to the second compartment and the pressure in the subject's compartment reduced rapidly by a further 52 mmHg (the induction of pressure breathing took five seconds). Pressure breathing was continued for two minutes unless unconsciousness occurred or syncope was imminent. At the end of the pressure breathing period the subject was given oxygen to breathe.

(c) 349 mmHg absolute (inspired oxygen tension, calculated for gas saturated with water vapour at body temperature of 63 mmHg). The technique employed was similar to that described in the preceding paragraph, except that the absolute pressure used in the second compartment was 349 mmHg absolute.

The order of the exposures to the three levels of partial pressure of oxygen in the inspired gas was randomized. The degrees of hypoxia induced by the two levels of absolute intrapulmonary pressure used in this study were determined by taking end-expiratory Haldane-Priestley samples of alveolar gas. The technique by which these samples were obtained was similar to that employed to obtain samples during pressure breathing at simulated altitudes above 50 000 ft (Chapter 5).

Results – The results of this group of experiments are presented in Table 6-6. The mean values of the heart rate before and during the two minute period of pressure breathing at a positive pressure of 52 mmHg are presented with their respective standard errors in Fig. 6-24. Each subject successfully completed an exposure to pressure breathing at ground level. The mean increase of heart rate over this period was 27.6 beats per minute. Eleven of the thirteen subjects exposed to pressure breathing at an intrapulmonary pressure of 380 mmHg absolute successfully completed the required two minutes. Most of these, however, were moderately cyanosed during this period. The rise of mean heart rate over the pressure breathing period was nineteen beats per minute. The remaining two subjects became nauseated and suffered dimming of vision thirty and one hundred seconds respectively after the beginning of pressure breathing. There was facial pallor and a marked bradycardia during the collapse in each case.

Twelve subjects performed pressure breathing with an absolute intrapulmonary pressure of 340 mmHg. Ni subjects completed the exposure success. " . ough they exhibited well-marked cyanosis throughout the time spent at a chamber pressure of 349 mmHg absolute. The mean heart rate was increased by 22.9 beats per minute over the pressure breathing period. Two of the three other subjects were restless and severely confused throughout the exposure to pressure breathing. Both these subjects had rapid bounding radial pulses whilst pressure breathing. The remaining subject became unconscious fifteen seconds after the beginning of pressure breathing and had a major convulsion. There was marked bradycardia and the pulse was impalpable at the wrist during the collapse. Pressure breathing was ceased immediately and oxygen was administered. Recovery followed rapidly.

The results of the analyses of the end expiratory Haldane-Priestley samples of alveolar gas obtained under the conditions of the experiments performed at reduced environmental pressure in relation to the period of pressure breathing are presented in Fig. 6-25. The alveolar oxygen tension varied between 37 and 42 mmHg during pressure breathing with an absolute intrapulmonary pressure of 380 mmHg. At an intrapulmonary pressure of 349 mmHg absolute the alveolar oxygen tension lay between 35 and 40 mmHg.

Effect of Hypoxia upon Limb Volume   The effect of hypoxia upon the changes of forearm volume induced by pressure breathing was investigated

TABLE 6-6

PRESSURE BREATHING AT 52 mmHg FOR TWO MINUTES WITH VARYING DEGREES OF HYPOXIA

Subject	Response to pressure breathing with air at absolute intrapulmonary pressure of		
	740–770 mmHg	380 mmHg	349 mmHg
A	S	S	Confused
B	S	S	S
C	S	S	S
D	S	Syncope	Convulsion and syncope
E	S	S	Confused
F	S	S	S
G	S	Syncope	–
H	S	S	S
J	S	S	S
K	S	S	S
L	S	S	S
M	S	S	S
N	S	S	S
Incidence of failures (%)	Nil	15%	25%

S = Successful completion of exposure

TABLE 6-7

EFFECT OF HYPOXIA UPON INCREASE OF BLOOD CONTENT OF FORE-ARM DURING PRESSURE BREATHING AT 60 mmHg

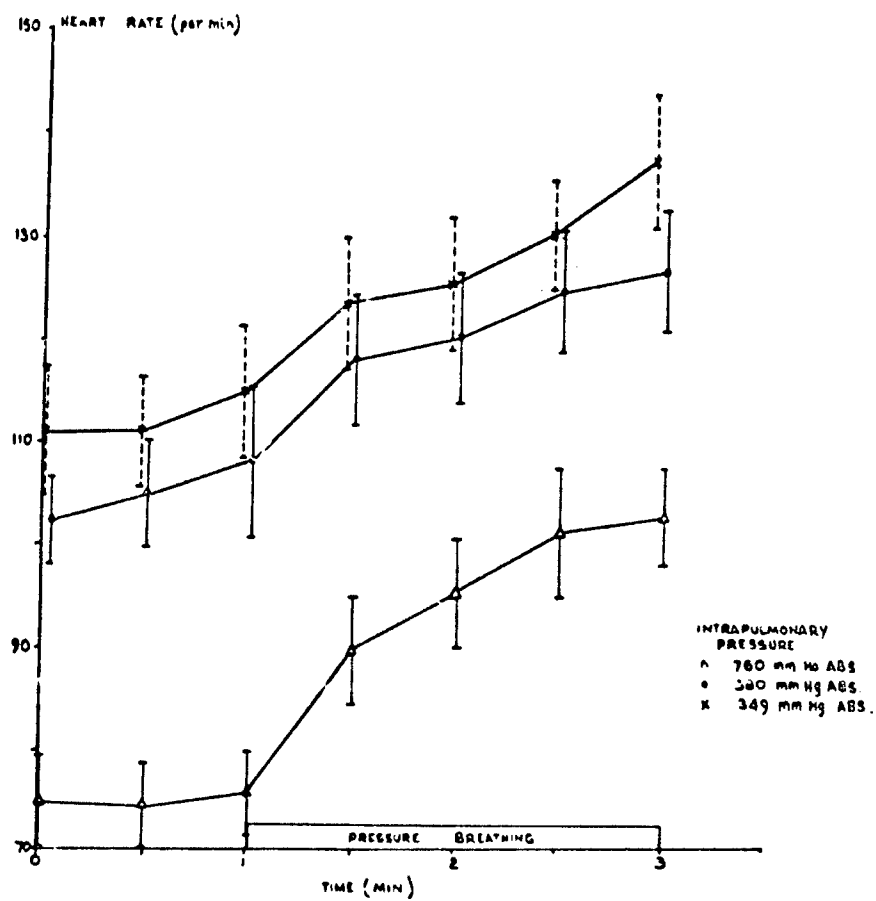| Subject	Increase of blood content of forearm (ml/100 ml)	
	Breathing 100% oxygen	Breathing air at 380 mmHg absolute
A	1.75	1.70
	1.83	1.90
B	1.80	1.96
	1.90	1.75
C	1.65	1.69
	1.78	1.61

FIG. 6 24 The effect of hypoxia upon the heart rate during pressure breathing with trunk counterpressure at a positive pressure of 52 mmHg (mean values ± 1 S.E.)
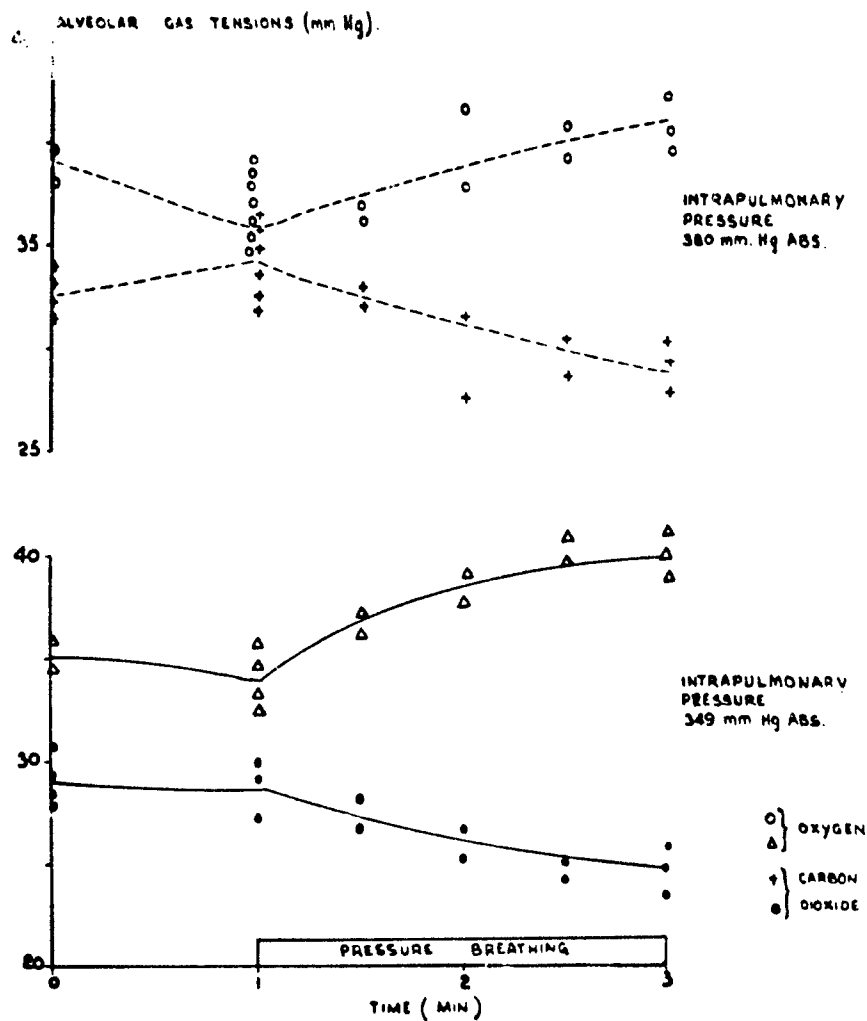
FIG. 6·25 The alveolar oxygen and carbon dioxide tensions during pressure breathing with air at a positive pressure of 52 mmHg at various absolute intrapulmonary pressures

all three subjects experienced in pressure breathing. The subject wore a pressure headpiece and pressure jerkin and he was secured in an ejection seat placed within one compartment of a decompression chamber. Pressure breathing and hypoxia were induced by a technique similar to that used in the previous series of experiments. The subject was exposed to a positive breathing pressure of 60 mmHg at an intrapulmonary pressure of 380 mmHg absolute. The subject breathed either air or 100% oxygen for five minutes before and throughout the subsequent two minutes exposure to pressure breathing. During a single experiment each subject was exposed to pressure breathing on four occasions. The order in which air or oxygen was breathed was randomized. One upper limb was supported on a table in a comfortable position with rubber pads placed beneath the elbow and wrist. The forearm lay in an horizontal plane which was about 7 cm below the sternal angle. The circumference of the limb was recorded continuously by a mercury-in-rubber strain gauge (286) placed around the upper part of the forearm. The output of the bridge amplifier to which the gauge was connected was fed on to a galvanometer of a photographic recorder.

Results – The increase of forearm circumference due to the peripheral displacement of blood was measured from the photographic record by the technique described earlier in this chapter. The actual increase of volume per unit volume of forearm was calculated (286). The results of these experiments are presented in Table 6 7. There was no significant difference between the initial increase of forearm volume induced by pressure breathing under hypoxic conditions as compared with the increase when the subject breathed 100% oxygen.

Pressure Breathing with oxygen at low environmental pressures – These experiments were performed in the small compartment of the decompression chamber. All the subjects had successfully completed a programme of training in pressure breathing at ground level before the exposure to pressure breathing at low environmental pressure was carried out. The subject, wearing the assembly of pressure clothing under investigation, was secured in the seat fitted in the decompression chamber. The oronasal mask or pressure headpiece and the pressure clothing were connected to an automatic pressure demand regulator (Mark 20 or 21) set to deliver 100% oxygen throughout the experiment. The pressure at the lips was measured relative to that within the subject's compartment of the decompression chamber by means of a mercury manometer. The subject was decompressed to an initial pressure altitude between 25000 and 27000 ft and after a period of one to two minutes he was decompressed in one second to the final altitude. The oxygen regulator automatically delivered oxygen at the required positive breathing pressure as the decompression occurred. The final altitude was maintained for a period which varied between a half and two minutes.

The exposure to pressure breathing at simulated high altitude was terminated by recompression to below a pressure altitude of 40000 ft. The rate of recompression varied from very rapid (time to return to a pressure-altitude of 40000 ft of less than five seconds) to controlled rates of recompression which simulated rates of descent of either 10000 ft or 15000 ft per minute to a pressure-altitude of 40000 ft. The electrocardiogram (lead II) was recorded during all the exposures whilst in many the arterial pressure

was also measured indirectly using a sphygmomanometer cuff and piezoelectric transducer. An exposure to simulated high altitude was terminated immediately if the subject complained of any of the symptoms of an impending collapse or the E.C.G. record showed a bradycardia which exceeded 30 beats per minute or a fall of arterial pressure which exceeded 30 mmHg. The combinations of pressure clothing investigated, the duration of the exposure to the maximum simulated altitude, the absolute intrapulmonary pressure and the positive breathing pressure during that period and the rate of recompression to a simulated altitude of 40000 ft are presented in Table 6–8.

Results – The overall results for the experiments in which subjects were exposed to pressure breathing with oxygen at environmental pressures of less than 140 mmHg absolute are presented in Table 6–8. The features of the experiments in which the subject did not successfully complete the intended exposure are presented in Table 6–9. When the intrapulmonary pressure was 126 mmHg absolute or greater the incidence of unsuccessful experiments lay between 4 and 6%, whilst with an absolute intrapulmonary pressure of 121 mmHg, there was a disturbance of consciousness in a quarter of the subjects.

In those experiments in which the absolute intrapulmonary pressure during pressure breathing was between 126 and 155 mmHg the two causes of an unsuccessful completion of exposure were abdominal pain and syncope. (Table 6–9). The abdominal pain occurred immediately after decompression and often the subject had complained of mild abdominal discomfort before the decompression occurred. The syncopal attacks were heralded by nausea, sweating and dimming of vision. The electrocardiogram showed a marked bradycardia at this time. The pressure in the chamber was always increased as rapidly as possible to 140 mmHg absolute at this point but in two of the instances the subject lost consciousness for fifteen to twenty seconds.

The mean value of the heart rate and the mean arterial pressure obtained in three of the eleven series of experiments are presented in Figs. 6–26, 6–27 and 6–28. In each of the Figures the mean values of heart rate and arterial pressure during the exposure of the same subjects to a similar positive breathing pressure-time relationship whilst breathing air at ground level are also given. In each series of experiments the heart rate during pressure breathing was consistently greater in the altitude experiments as compared with the values obtained at ground level. There was, however, no such difference in the values of the mean arterial pressure.

Marked swelling of the back of the hand occurred fifteen to thirty seconds after the decompression in all the subjects exposed to a pressure altitude of 70000 ft. This swelling was crepitant but there was no impairment of function. The swelling resolved when the pressure within the decompression chamber was increased. There were no residual symptoms or impairment of sensory or motor function subsequent to the exposure.

DISCUSSION
Displacement of blood into the limbs – The inception of positive pressure breathing caused a progressive increase of the volume of each of the segments of the upper and lower limbs examined in this study. Direct observation of the superficial veins showed that the blood content of these vessels was

TABLE 6-8

EXPOSURE TO PRESSURE BREATHING
AT REDUCED ENVIRONMENTAL PRESSURE

Regulator type	Maximum altitude (feet)	Time at maximum altitude (min.)	Rate of descent (ft./min.)	Intrapulmonary pressure at maximum altitude (mmHg abs.)	Number of subjects	Unsuccessful attempts Number	Proportion
A. Headpiece and pressure jerkin							
18	56000	1	10000	145	10	0	0
B. Headpiece, pressure jerkin and anti-G suit							
18	60000	1	10000	145	20	0	0
20	60000	1	10000	155	100	5	5%
18	70000	1	10000	145	12	0	0
C. Mask and pressure jerkin							
Special	55000	1	rapid	121	20	5	25%
Special	55000	1-2	rapid	120	5	1	20%
D. Mask, pressure jerkin and anti-G suit							
21	56000	½	10000	126-128	22	0	0
21	56000	½	10000	126-128	100	4	4%
E. Headpiece, arm jerkin and anti-G suit							
18	100000[1]	1	15000	118	17	1	6%
18	70000[1]	5	rapid	145	10	0	0
20	60000	1	10000	155	100	2	2%

[1] Pressure gloves worn

TABLE 6-9

CAUSES OF FAILURES DURING PRESSURE BREATHING
AT SIMULATED HIGH ALTITUDE

Altitude (feet)	Number of subjects exposed	Number of failures	Cause of failure
B. Headpiece, Jerkin and anti-G suit			
60000	1000	5	3 – abdominal pain 2 – syncope 1 min. and 1½ min.
C. Mask and Jerkin			
55000[1]	20	5	2 – severe confusion 3 – mild confusion
55000[2]	5	1	1 – syncope after 1 min.
D. Mask, Jerkin and anti-G suit			
56000	100	4	1 – syncope after 65 sec. 3 – abdominal pain
E. Headpiece, Arm Jerkin and anti-G suit			
100000	17	1	1 – syncope after 3½ min.
60000	100	2	2 – abdominal pain

[1] Intrapulmonary pressure 121 mmHg abs.
[2] Intrapulmonary pressure 120 mmHg abs.

FIG. 6-26 The effect upon heart rate and arterial pressure of pressure breathing with a headpiece and trunk and lower limb counterpressure at ground level and at 60000 ft mean values for twenty subjects

Fig. 6 27 The effects of on heart rate and arterial pressure of pressure breathing with an oronasal mask and trunk and lower limb counterpressure at ground level and at 56000 ft mean values for twenty-two subjects
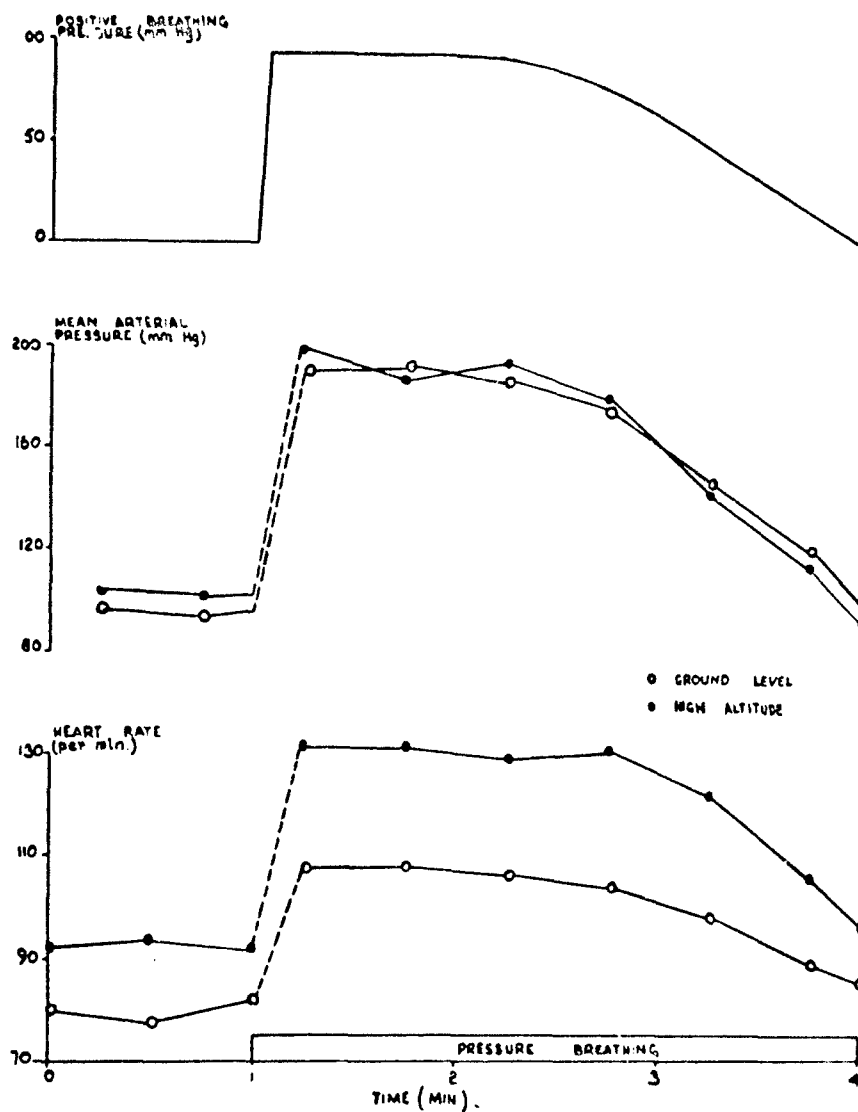
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FIG. 6-28 The effects upon the heart rate and arterial pressure of pressure breathing with a headpiece and trunk, upper and lower limb counterpressure at ground level and at 70000 ft mean values for ten subjects
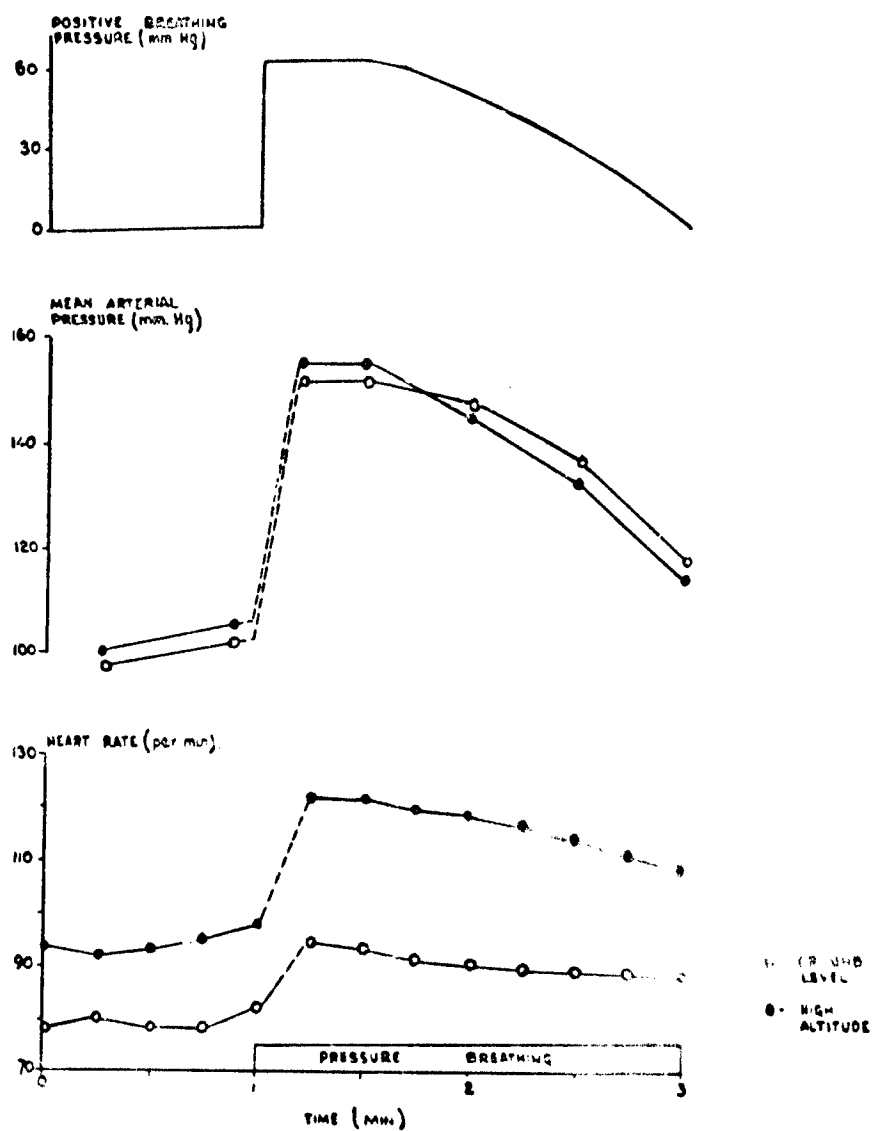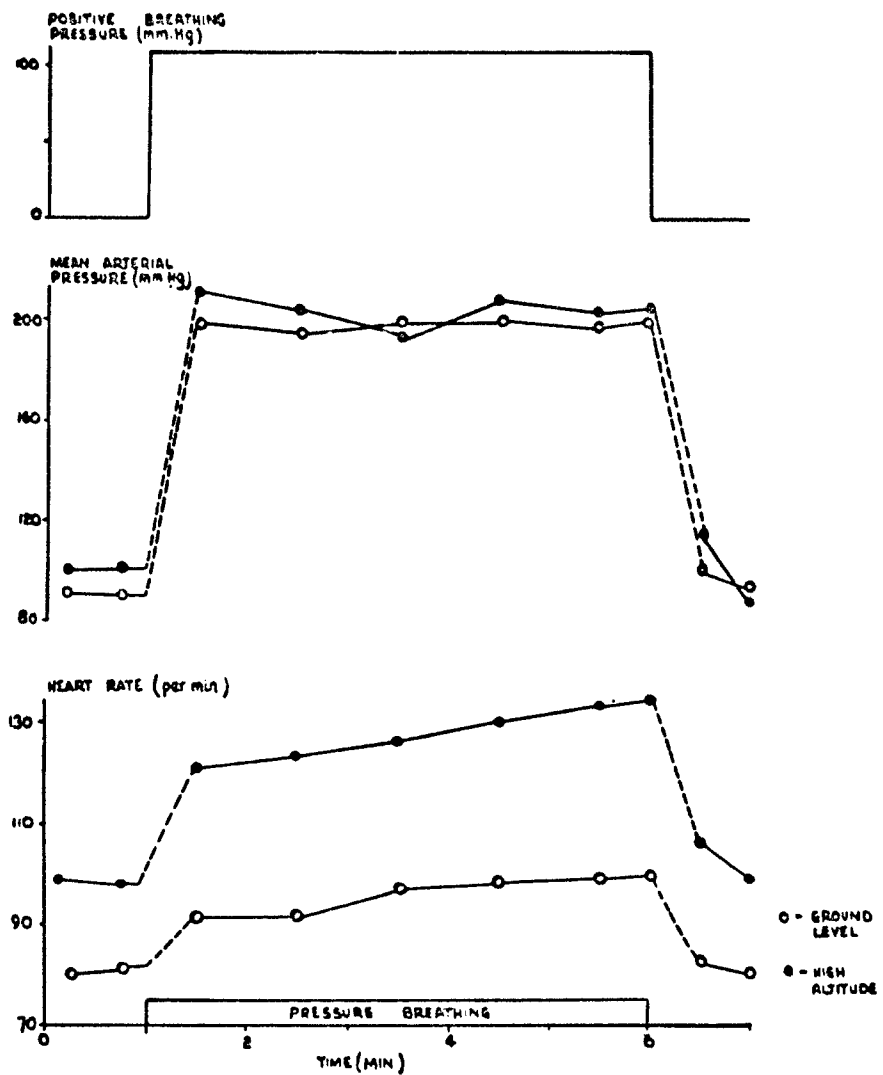
increased during pressure breathing. The relatively large increase of limb volume which occurred at the beginning of pressure breathing was due to an increase in the volume of blood contained within the peripheral vascular tree. The central venous pressure was increased directly the intrapulmonary pressure was raised (Fig. 6-14) so that the pressure gradient which normally existed between the peripheral and central parts of the venous system was reversed. The venous valves prevented any retrograde flow of blood. Since, however, arterial flow into the limb continued, blood collected in its vascular bed. The vessels were progressively distended by the inflow of blood. There was a consequent rise of pressure throughout the vascular bed until the venous pressure in the limb exceeded the central venous pressure and blood started to flow again from the limb. The relationship between the increase of the volume of blood in the limb and the corresponding pressures within the vascular bed reflected the distensibilities of the various components of the bed. The rate of increase of limb volume was high at the beginning of pressure breathing and it fell progressively over the ensuing ten to twenty seconds to reach a minimum rate which was maintained for as long as the raised intrapulmonary pressure remained constant.

Simultaneous records of forearm volume and the pressure in the corresponding antecubital veins (Fig. 6-1), showed that, in the forearm at least, the initial large increase of limb volume was associated with a low venous pressure. As the pressure in the vascular bed increased the increase of limb volume per unit rise of venous pressure fell progressively. Thus the distensibility of the vascular bed fell as the pressure within it was increased. Directly the pressure within the capillaries of the vascular bed of a limb was increased excess fluid passed from the blood into the extravascular space. Even if it is assumed that the rate of increase of extravascular fluid during the period in which blood was accumulating in the limb was as high as the rate measured after the changes of blood volume were completed, it may be calculated that the increase of extravascular fluid volume was less than 8"₀ of the total increase of limb volume over this period. The error associated with assuming that all the initial increase of limb volume in pressure breathing was due to an increase in the volume of blood in the part was therefore small.

The relationship between the volume of blood displaced into a limb and the corresponding positive breathing pressure is determined by a number of factors. The most important factors are the magnitudes of the increases of the pressure differences across the walls of the various components of the peripheral vascular bed and the distensibilities of these components. The increase of the transmural pressure produced by a given positive breathing pressure is a function of the effect of pressure breathing upon the intravascular and extravascular pressures in the limb. The response of the vessels to a given increase of transmural pressure may be modified by local factors such as the temperature and activity of the tissues, by humoral influences and by nervous reflexes.

Pressure breathing with the trunk counterpressure applied by the pressure jerkin increased the mean intra-arterial pressure by 80 to 100"₀ of the applied positive breathing pressure (Fig. 6-17). When this garment was used the rise of peripheral venous pressure virtually equalled the corresponding positive breathing pressure (Fig. 6-13). Thus when counterpressure is applied to the

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trunk the pressure within the whole peripheral vascular bed is raised by an amount which is nearly equal to the positive breathing pressure.

In order to define the effect of pressure breathing upon the extravascular pressure the tissue pressure was measured during pressure breathing by determining the pressure required to introduce a minute quantity (1.7 cu. mm.) of saline into the tissue through a hypodermic needle (91). These experiments showed that the pressure in the subcutaneous tissues of the limbs and the relaxed muscles of the upper limb was not significantly increased by the induction of pressure breathing at positive breathing pressures of up to 100 mmHg. The maximum increase of tissue pressure found in these regions after pressure breathing for five minutes was 5 mmHg. Whilst the pressure in the anterior tibial and soleus muscles behaved in the same manner as the muscle pressure in the upper limb, the pressure in the quadriceps femoris and gastrocnemius muscles increased by between 30 and 45", of the applied positive breathing pressure within twenty seconds of the commencement of pressure breathing. The increase of tissue pressure induced by pressure breathing in these muscle groups of the lower limb was believed to reflect the properties of the thick indistensible fascial layers which envelop them. The distensibilities of the fascial sheaths which surround these muscle groups are probably comparable with the distensibilities of the vascular beds contained within the muscles. When blood collects in the vessels of these regions the distension of the vascular bed is restricted by the enveloping fascia and this causes the observed increase of tissue pressure. Wells, Youmans and Miller 1938 (284) found similar changes in the tissue pressures at various sites in the upper and lower limbs when the vascular pressure was increased by venous congestion. Thus the tissue pressure in the relaxed upper limb and in the lower limb, with the exception of the quadriceps and soleus muscle groups, was only slightly increased so that the vessels in these regions were subjected to an increase of transmural pressure which virtually equalled the rise of intravascular pressure. In the deep muscle groups of the lower limb, however, there was a considerable rise of tissue pressure at the beginning of pressure breathing, so that the transmural pressures of the vascular beds in these muscles was only increased by 55 to 70", of the increase of intravascular pressure.

It is possible to estimate approximately the proportion of the overall increase of the volume of blood in the limb which is contained by each component of the peripheral vascular bed. The volume elasticity coefficients of the arteries have been investigated extensively in vitro. Hallock and Benson 1937 (139) found that there was an approximate linear relationship between the increase of the capacity of an artery and its transmural pressure over the range of pressures of interest in the present study. The volume elastic coefficients of the arteries of the upper and lower limbs is approximately 1", increase in volume per mmHg increment of transmural pressure. Since the volume of the arterial vessels of the upper limb amounts to about 10 ml (29) it can be calculated that the additional volume of blood contained by the upper limb arteries during pressure breathing lies between 5 and 10", of the total increase of the volume of blood in the limb. The arteries of the lower limbs take up a similar proportion of the total increase of the blood content of these parts. The part played by the capillary bed is difficult to assess. The

capillaries contain a relatively small proportion, about one fifth, of the blood volume of the resting limb (128). The bed is, however, capable of very large increases of capacity under conditions of work and presumably of raised transmural pressure (173). The veins contain the major fraction, about two thirds of the blood content of the peripheral vascular bed at rest. At low transmural pr   res the distensibility of the venous portion of the system is very high. When the veins are collapsed a very large increase in capacity is produced by a small increase of transmural pressure. Thus in isolated veins the distensibility is great at low intraluminal pressures, but at a pressure of about 4 mmHg an inflection occurs in the pressure-volume diagram and at higher pressures the distensibility is much reduced (257). The distensibility of the veins at transmural pressures above 4 mmHg is, however, far greater than the distensibility of a comparable artery over the same pressure range (63). The veins constitute the major portion of the capacity of the peripheral vascular bed and these vessels probably accommodate most of the increase of the blood content of the limbs induced by pressure breathing.

Studies of the pressure volume characteristics of the vascular bed of the forearm (131) and of the calf (193) (64) have shown that the distensibility falls markedly as the transmural pressure is increased. The results of the present study appear to be in conflict with the findings of other workers, particularly in the lower limb where the relationship between the increase of blood volume and the applied positive breathing pressure was found to be virtually linear (Fig. 6–4). In the experiments performed by previous investigators, however, the subjects were recumbent and the limb under study was placed either at or above heart level. In the present study the primary interest was the increase of the blood content of the limbs induced by pressure breathing in the seated subject. Both the upper and lower limbs were placed considerably below heart level and the resting venous pressures were of the order of 10 and 30 mmHg respectively. It was not possible to study the calf with the long axis of the part vertical so that the venous pressure in this segment was somewhat less than the venous pressure which normally exists when the feet are dependant. When allowance is made for the higher resting values of the venous pressures which existed in the present experiments, the shapes of the pressure-distension curves for the forearm and calf obtained during pressure breathing are comparable with those obtained by the application of subatmospheric pressures to these regions (131) (64).

The increment of blood content produced by a given positive breathing pressure was somewhat greater in the hand than in the forearm (Fig. 6–3). This difference may have been a reflection of the differences in the proportion of skin and muscle in the two regions and their respective vascularities. Thus whilst by volume the hand contains approximately 55"$_0$ skin and bone and 15"$_0$ muscle (1) the forearm contains 8"$_0$ skin and 60"$_0$ muscle (66). This finding suggests that the skin has a greater vascular capacity than muscle. The vascularity of the skin varies from one region to another, however, and the relative importance of these two tissues as sites for the accommodation of blood during pressure breathing cannot be assessed from the present data.

The thigh and calf had very similar vascular distensibilities during pressure breathing. The mean distensibility of the lower limb amounted to 0.016 ml/100 ml of limb per mmHg positive breathing pressure. These results may

be compared with that obtained by Henry 1951 (146) who determined the increase of the volume of the lower limbs produced by pressure breathing with chest counterpressure. His subjects stood in a plethysmograph which was filled with water to the level of the top of the thighs. A positive breathing pressure of 40 mmHg increased the volume of the lower limbs by 140 ml which gives a calculated distensibility of approximately 0.018 ml/100 ml of limb per mmHg positive breathing pressure. Although Henry's subjects were standing, the water in the plethysmograph prevented virtually any increase of transmural pressure due to gravity below the surface of the water. Thus the transmural pressures of the lower limb vessels were very similar in Henry's and the present study. The value for the overall distensibility of the vessels obtained in the present study agreed well with the value obtained by Henry.

In the present investigation care was taken to maintain a constant thermal environment. Further, the temperature of the water in the plethysmograph was maintained at the value at which Barcroft and Edholm 1943 (21) showed resulted in the same vascular behaviour as that exhibited by the normally clothed part at a room temperature of 18 to 22°C. The effect of changes of limb temperature upon the quantity of blood displaced into the lower limbs was investigated in a few experiments by Henry 1951 (146). He found that increasing the temperature of the water of the plethysmograph in which the lower limbs were immersed from 25° to 40°C increased the quantity of blood displaced by a given positive breathing pressure, by 60%. Greenfield and Patterson 1956 (131), however, found that the capacity of the forearm vessels defined as the volume of blood in the part when a subatmospheric pressure of 100 mmHg was applied to it, was unchanged by raising the temperature of the water in the plethysmograph or by general heating of the body.

Similar results were obtained by Wood and Eckstein 1958 (289) who measured the additional volume of blood held by the vessels of the forearm when the venous pressure was raised by 30 mmHg using local congestion. This additional volume of blood was not increased by either the local or general application of heat. These investigators did find, however, that reducing the temperature of the environment below the comfort level resulted in a decrease in the distensibility of the forearm veins. Although the results obtained by Greenfield and Patterson 1956 (131) and by Wood and Eckstein 1958 (289) suggest that an increase in the temperature of the environment above the comfortable range does not increase distensibility of the capacity vessels of the forearm, they do not necessarily conflict with the results obtained by Henry 1951 (146). The studies were performed on different limb segments. Further there is evidence that pressure breathing causes a reflex reduction of the distensibility of the capacity vessels. It is conceivable that this reflex reduction of distensibility may be impaired by a rise of the environmental temperature.

It is possible to estimate approximately the total quantity of blood displaced into the four limbs at the beginning of pressure breathing with trunk counterpressure from the measurements made on the individual limb segments. The amount of blood pooled in certain limb segments, namely the upper arm and the foot was not determined in the present study. It would appear likely, however, that the distensibility of the capacity vessels of the

upper arm is very similar to that of the vessels of the forearm. Thus it has been assumed that the upper limb, with the exception of the hand, may be treated as a single region with a vascular distensibility equal to that found in the forearm. Although no measurements were made on the foot, the volume of this region is less than 10%, of the volume of the lower limb so that the overall error introduced by assuming that the distensibility of the vessels of the foot is similar to that of the rest of the lower limb will be small. The measurements of the change in the blood content of the thigh and calf produced by pressure breathing were performed with the subjects in the seated position with the limb horizontal. When the knee is flexed and the foot is placed on the floor the vascular pressures in the calf will be increased above those which were operative in the present study. The present results and those obtained by Coles, Kidd and Patterson 1956 (64) at venous transmural pressures of up to 200 mmHg show, however, that at the vascular pressures concerned the pressure-volume curve of the vessels of the calf is virtually a straight line. Thus it is unlikely that the quantity of blood displaced into the calf by pressure breathing at a given positive breathing pressure with the subject in the normal seated position will differ very much from that found with the lower limb horizontal in the seated subject. In order to calculate the total volume of blood displaced by a given positive breathing pressure from the results given in Figs. 6-3 and 6-4 and Table 6-1, the total volumes of each segment must be known. These volumes were measured for the subjects used in this study. (Table 6-10). The relationship between the quantity of blood displaced into all four limbs of a seated subject and the corresponding positive breathing pressure is presented in Fig. 6-29.

The magnitude of the volume of blood displaced into the limbs by pressure breathing in the seated position may be compared with the effects of venous congestion and changes of posture upon the volume of blood in the limbs. Thus Ebert and Stead 1940 (87) measured the effect in supine subjects of displacing blood into one upper limb and both lower limbs by inflating cuffs around the proximal ends of the limbs to diastolic pressure (70 to 80 mmHg) for ten minutes upon the volume of blood in the remainder of the body (measured by a dye dilution technique). They found that this degree of venous congestion reduced the volume of blood in the remainder of the body by about 700 ml. Nearly a third of this reduction was due to the passage of fluid into the intravascular spaces of the congested limbs. The greater increase of the blood content of the limbs produced by venous congestion than that produced by a comparable positive breathing pressure is primarily due to the difference in the posture of the subjects in the two series of experiments. The initial venous distension was considerably less in the experiments performed by Ebert and Stead 1940 (87) than in the present pressure breathing experiments. Asmussen 1943 (9) studied the amount of blood contained in the lower limbs under various conditions by occluding the circulation through these parts and measuring the remaining blood volume by a carbon monoxide technique. He found that the volume of blood in the lower limbs at rest in the horizontal position averaged 0.72 litre and that tilting the subject 60° head-up increased this volume by 0.35 litre. The blood displaced by pressure breathing is, however, in addition to that pooled in the lower limbs by the assumption of a seated posture. The total volume of blood displaced
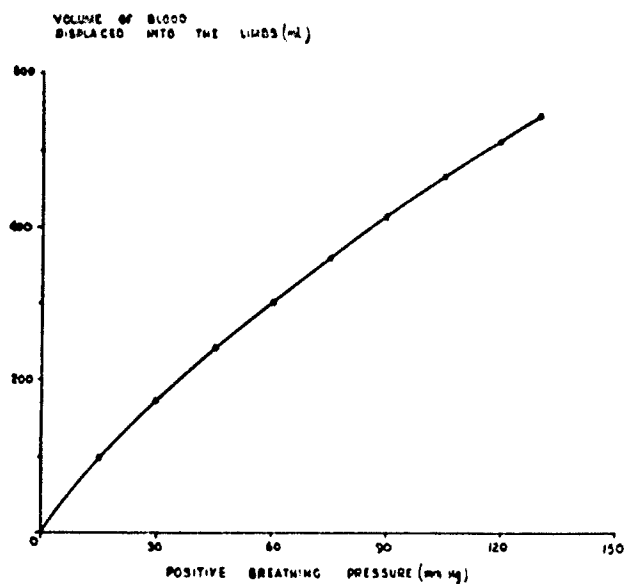
FIG. 6-29 The volume of blood displaced into the limbs by pressure breathing with trunk counterpressure

FIG. 6-30 The reduction of effective blood volume induced by pressure breathing with trunk counterpressure

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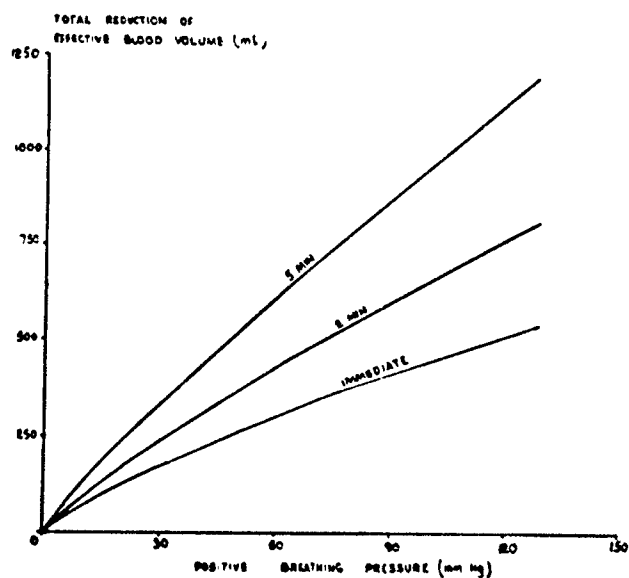
TABLE 6-10

THE VOLUMES OF LIMB SEGMENTS OF SUBJECTS USED IN STUDY OF
LIMB VOLUMES IN PRESSURE BREATHING

| Segment | Volume (litre) | |
	Subject A	B
Hand	0.45	0.53
Upper limb to shoulder	2.95	3.10
Lower limb to symphysis pubis	9.50	10.08

into the limbs by the assumption of the seated position and pressure breathing at a positive breathing pressure of 60 to 80 mmHg is similar to that produced by venous congestion of the limbs induced in the horizontal position as in Ebert and Stead's experiments.

Reflex Changes of Vascular Distensibility – The effect of the central disturbances induced by pressure breathing upon the distensibility of the peripheral vasculature was investigated by comparing the increase in hand volume produced by pressure breathing with that produced by local venous congestion. The increase of the volume of blood in the hand produced by a given increase of venous pressure was greater when the rise of venous pressure was produced by local congestion than when it was induced by pressure breathing (Fig. 6–8). Thus the distensibility of the vessels of the hand was reduced by pressure breathing. Since the arterial pressure was raised during pressure breathing but not during local venous congestion the quantity of blood contained in the arterial portion of the vascular bed of the hand was probably greater during pressure breathing than during local congestion. Following a complete nerve block at the wrist there was no difference in the increase of hand volume produced by a given rise of venous pressure whether it was induced by pressure breathing or by local congestion. The reduced distensibility of the capacity vessels of the hand produced by pressure breathing under normal conditions was mediated therefore by efferent nerves to the vessels of the hand.

The major fraction of the capacity of the peripheral vasculature resides in the vei ..s. Goltz 1864 (124) demonstrated that the capacity of the veins may be changed independently of the pressure existing within them and this finding has been confirmed repeatedly. Donegan 1921 (76) found that the capacity of various veins in the dog, cat and rabbit could be changed by stimulation of certain afferent nerve trunks. Exposure to a low environmental temperature was shown by Doupe, Krynauw and Snodgrass 1938 (79) to increase the pressure in an isolated segment of forearm vein. Page, Hickham, Sieker, McIntosh and Pryor 1955 (234) demonstrated that tilting from the horizontal position to the erect increased the pressure in an isolated segment of the forearm vein. They also found that the Valsalva manoeuvre was a potent stimulus to active veno-constriction. Wood and Eckstein 1958 (289) studied the effect of displacing blood into the lower limbs by the inflation of cuffs placed around the upper thighs upon the distensibility of the forearm vasculature as measured by the increase of limb volume produced by raising the effective local venous pressure by 30 mmHg. They found that pooling blood in the lower limbs reduced the distensibility of the forearm vessels by about 20"₀.

Sharpey-Schafer 1961 (263) developed a technique for measuring venous tone by relating the rate of increase of volume to the rate of increase of venous pressure rise when a venous congestion cuff was inflated around the upper arm. He demonstrated a very marked increase of venous tone during the Valsalva manoeuvre. Blair, Glover and Kidd 1959 (37) were unable, however, to show any change in the distensibility of the forearm vessels during pressure breathing at a positive pressure of 15 mmHg. In the present experiment positive breathing pressures of up to 50 mmHg were employed and a significant reduction of the distensibility of the vasculature of the hand was

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found during pressure breathing. The behaviour of the capacity vessels of the hand may differ, however, from that of the forearm vessels in pressure breathing. The intramusculature veins are thin and have a paucity of muscle fibres in their walls as compared with the superficial veins (114). Also Donegan 1921 (76) found in animal experiments that whilst the calibre of the superficial veins of the limbs could be altered readily by stimulating the sympathetic chain, the intramusculature veins were not affected by this procedure. Studies of the effects of the peripheral pooling of blood have shown, however, that the distensibility of the capacity vessels of the forearm can change (289). It would appear likely, therefore, that pressure breathing induces a reflex reduction of the distensibility of the capacity vessels of the arm and lower limb as well as those of the hand.

It is of interest to consider the nature of the stimulus which may be responsible for the reflex reduction of the distensibility of the peripheral capacity vessels induced by positive pressure breathing. Relatively little is known of the reflex mechanism which controls venous tone. Goltz 1864 (124) demonstrated that the normal tone of the intestinal veins depended upon an intact medulla and spinal cord. Heymans, Bouckaert and Doutrebande 1931 (155) showed that innervated perfused mesenteric veins constricted when the pressure in the carotid sinus was reduced. The reflex venoconstriction which occurs in man when blood is displaced in the periphery by tilting into the erect posture (265) or by congesting the lower limbs (289) is not usually accompanied by a fall of arterial blood pressure so it is unlikely that the observed reflex venoconstriction arises from the carotid or aortic baroreceptors. Reflex venoconstriction occurs during the Valsalva manoeuvre in which the transmural pressure of the carotid sinus is increased, although the aortic transmural pressure is generally decreased by this procedure. Pressure breathing with trunk counterpressure and a pressure headpiece which applies counterpressure to the skin overlying the carotid bifurcation induces little or no change in the transmural pressures of either the carotid sinus or the arch of the aorta. In all these situations, however, there is a reduction of the effective right atrial pressure. It is probable, therefore, that the receptors responsible for the reflex venoconstriction produced by these various procedures lie in the low pressure region of the intrathoracic vascular bed, probably in the right atrium or the venae cavae. Roddie and Shepherd 1956 (247) have postulated that receptors in this region are responsible for the reflex arteriolar vasodilatation which occurs in the forearm when blood is emptied from the legs into the chest. It is also probable that the peripheral arteriolar vasoconstriction induced by positive pressure breathing arises from receptors in the low pressure portion of the intrathoracic circulation.

A further possible cause of the reduction of the distensibility of the peripheral capacity vessels in pressure breathing which should be considered is hyperventilation. Thus Page, Hickham, Sieker, McIntosh and Pryor 1955 (234) found that overbreathing induced a rise of the pressure in a temporarily isolated segment of forearm vein. The distensibility of the forearm vascular bed was studied in voluntary hyperventilation by Eckstein, Hamilton and McGammond 1958 (89) by determining the increase in the blood content of the forearm associated with increasing the effective venous pressure from zero to 30 mmHg. They found that a reduction of the end-expiratory carbon

dioxide tension from 43 to 25 mmHg was associated with a 24% decrease of the distensibility of the capacity vessels of the forearm. They further demonstrated that a considerable proportion of this change of distensibility was due to the mechanical effects within the thorax of the hyperventilation since the effect was only partially abolished when carbon dioxide was added to the inspired gas in order to prevent hypocapnia. Although pressure breathing does produce an increase of pulmonary ventilation and a reduction of the alveolar carbon dioxide tension, the magnitude of these changes at positive breathing pressures of up to 50 mmHg when trunk counterpressure was used is small (Chapter 5). Thus the hypocapnia induced by pressure breathing probably plays only a minor role in the production of the reflex reduction of the distensibility of the capacity vessels found in the present study.

Fluid Filtration -- The volume of each limb segment studied during pressure breathing continued to increase after the local venous pressure had become constant. This rate of increase of limb volume remained unchanged through an exposure to pressure breathing. The actual increase of limb volume produced by pressure breathing in addition to that associated with the displacement of blood into the part was measured from the experimental records. It was found that after the circulatory changes associated with the termination of pressure breathing had subsided the limb segment volume was always greater than the volume of the part immediately before the exposure to pressure breathing. It was found that the magnitude of the increase of limb volume during pressure breathing measured from the point at which the rate of increase of limb volume became constant was approximately equal to the difference of limb volumes immediately before and immediately after the exposure to pressure breathing (Fig. 6–5). This increase of limb volume was due to a nett passage of fluid from the blood into the tissues of the limb. Starling 1909 (272) demonstrated that the intracapillary pressure was the most important single factor governing the exchange of fluid between the blood and the tissues. The rise of capillary pressure induced by pressure breathing disturbs the normal balance between the hydrostatic and osmotic forces across the walls of the capillaries so that there is a considerable hydrostatic force driving fluid from the capillaries into the surrounding tissues. Although there was some variability in the rates of increase of volume in pressure breathing, the relationship between the rate of filtration and the positive breathing pressure was virtually linear both in the upper and lower limbs (Figs. 6–6 and 6–7).

Starling 1909 (272) and later Landis and Gibbon 1933 (182) demonstrated that the rate of filtration of fluid is closely related to the venous pressure. Pappenheimer and Soto-Rivera 1948 (235) showed in the perfused hind limb of the cat and dog that increasing the venous pressure by 0.5 mmHg produced a readily detectable movement of fluid from within the circulation into the extravascular space. Further they found that in order to produce a given filtration rate the arterial pressure must be raised by five to ten times the increase of venous pressure required to induce the same rate of filtration. Landis and Gibbon 1933 (182) measured the rate of increase of forearm volume during venous congestion produced by a cuff placed around the upper arm by means of a pressure plethysmograph. They found that the rate of filtration was linearly related to the venous pressure and that the mean

rate of increase of filtration amounted to 0.0045 ml/min./100 ml of forearm per mmHg increase of venous pressure. This value may be compared with the mean rate of increase obtained in the present pressure breathing experiments which amounted to 0.0048 ml/min./100 ml of forearm per mmHg increase of positive breathing pressure.

The rate of filtration of fluid from the circulation during pressure breathing at a given positive breathing pressure was some 30% slower in the lower limb than in the upper. The studies Ernsting 1955 (91) of the behaviour of the tissue pressure during pressure breathing already described show that in the deep muscle groups of the lower limbs the tissue pressure was raised by between 30 and 45% of the applied positive breathing pressure. Thus in these regions the transmural pressure of the tissue capillaries was increased by only 55 to 70% of the positive breathing pressure. The hydrostatic pressure driving fluid into these tissues was therefore considerably less than the force acting in the superficial tissues of the lower limbs and in the muscles and superficial tissues of the upper limbs. The measurements of tissue pressure also showed that the pressure in the tissues beneath the deep fascial layers of the lower limbs increased progressively during pressure breathing. This increase of tissue pressure was due to the increase of extravascular fluid beneath the deep fascial layers. The rise of tissue pressure observed over five minutes amounted to 10–15 mmHg at a positive breathing pressure of 60 mmHg. The consequent further decrease of transmural pressure of the capillaries in these regions would be expected to reduce the rate of filtration of fluid into the deep muscles. It is unlikely, however, that this rise of tissue pressure would result in a detectable reduction of the overall rate of increase of limb volume. Indeed, in the present experiments no reduction in the rate of increase of limb volume was found.

The measurements of the rates at which fluid passed from the circulation into the tissues of individual limb segments have been used to calculate the overall loss of fluid from the circulation associated with pressure breathing with trunk counterpressure. These calculations were based upon the assumptions already described in the calculation of the volume of blood displaced into the limbs at the beginning of pressure breathing. The overall rate of filtration of fluid from the circulation into the tissues of the limbs calculated from the measurements made on the individual limb segments was 0.99 ml/min./mmHg of positive breathing pressure. Since trunk counterpressure was used in these experiments, the increase of peripheral venous pressure was very nearly equal to the applied positive breathing pressure. Landis and Hortenstine 1950 (183) calculated that a general rise of venous pressure of 10 cm water throughout the body would filter about 250 ml of fluid from the plasma in the first ten minutes. This calculation gives a rate of filtration of fluid from the circulation of 3.4 ml/min./mmHg rise of venous pressure. This value is nearly four times as great as the rate of filtration found in the present investigation. In pressure breathing with trunk counterpressure, however, the vascular transmural pressure is only raised in the limbs whereas the rate of filtration calculated by Landis and Hortenstine applies to a rise of venous pressure throughout the body.

Henry, Hendrickson, Movitt and Meehan 1945 (149) and Henry 1951 (146) investigated the loss of fluid from the circulation during pressure

breathing by making serial measurements of the haematocrit. The subjects were exposed to positive breathing pressures of up to 55 mmHg with either no respiratory counterpressure or counterpressure applied to the chest and upper abdomen by means of a vest. After a rest period which lasted for thirty minutes, each subject was exposed, in the seated position, to pressure breathing for a period of thirty minutes. Blood for the determination of the haematocrit was taken from either a dorsal vein of the hand, which had been previously heated or in a few experiments from an artery. No difference was found between the values given by the arterial and arterialized venous blood. When the vest was used the mean reduction of the blood volume induced by pressure breathing for thirty minutes at a positive pressure of 50 mmHg was 9%. Assuming a resting blood volume of 5.5 litre this represented a loss of about 500 ml of fluid from the circulation and hence a rate of loss of fluid of 0.33 ml/min./mmHg of positive breathing pressure. The rate of filtration of fluid from the circulation induced by pressure breathing obtained by Henry was only one third of the rate of filtration calculated from the rates of increase of limb volume found in the present investigation.

There are a number of possible explanations for the discrepancy between the overall rates of filtration found during pressure breathing by Henry and in the present study. Firstly the methods used in the two studies for the measurement of the rate of loss of fluid from the circulation were dissimilar. Henry et al 1943 (144) carefully considered the errors which could have arisen from using the change of haematocrit to predict the loss of fluid from the circulation. Simultaneous measurements of the change of haemoglobin concentration showed that the total red cell volume calculated from the haemoglobin concentration and the haematocrit was unchanged by pressure breathing. In the present study it was assumed that the blood content of the limb remained constant following the initial displacement of blood at the beginning of pressure breathing. Although the venous pressure remained unchanged after the initial rise it is conceivable that the distensibility of the capacity vessels increased with the duration of the exposure to pressure breathing was, however, greater than the resting volume by an amount which on average equalled the increase of limb volume during the period of pressure breathing measured from the point at which it was assumed that the blood content of the limb had become constant (Fig. 6–5). Thus although the estimation of the fluid loss from the circulation into the limbs was made by an indirect method, it is unlikely that the calculated rate of filtration was much in error.

Secondly the degree of respiratory counterpressure used by Henry was somewhat less than that provided by the pressure jerkin. Thus the volume of tissue into which filtration occurred was greater with the vest than with the pressure jerkin. The magnitude of the rise of venous pressure induced by a given positive breathing pressure was, however, less in Henry's experiments since the vest did not prevent an increase of the functional residual capacity. At a positive breathing pressure of 50 mmHg the difference in the rise of venous pressure associated with the vest and pressure jerkin was probably of the order of 6 to 8 mmHg. Although this difference in the effect of a given positive breathing pressure upon the peripheral venous pressure would result in a lower filtration rate in Henry's experiments the magnitude of this effect

is too small to account for the difference between the calculated overall filtration rates.

Finally, the duration of the exposure to pressure breathing over which the measurements of filtration rate were made differed widely in the two series. All Henry's measurements were made over a period of thirty minutes whereas in the present study pressure breathing was only performed for five minutes. The accumulation of fluid in the extravascular space would be expected to reduce the rate of filtration over a period as long as thirty minutes. Thus Landis and Gibbon 1933 (182) found that the rate of filtration in the forearm when the venous pressure was raised to a constant value for thirty minutes, declined progressively with time. Moreover, an examination of the details of the results obtained by Henry, Hendrickson, Movitt and Meehan 1945 (149) shows that the rate of increase of haematocrit during pressure breathing fell progressively. Thus in one experiment over half the total change of haematocrit found in a thirty minute exposure occurred in the first five minutes. The haematocrit change in this experiment gave an overall rate of fluid loss of approximately 1.0 ml/min./mmHg positive breathing pressure over the first five minutes of the exposure. In the other experiment, of which adequate experimental details were given, the rate of fluid loss over the first ten minutes of pressure breathing was 0.62 ml/min./mmHg of positive breathing pressure. Thus the discrepancy between the results of the present experiments and those obtained by Henry is due principally to the differences in the duration of the pressure breathing exposure over which the measurements were made. There is reasonable agreement when similar periods of exposure are compared. This comparison emphasizes the fact that the rate of filtration induced by pressure breathing at a given positive breathing pressure declines as the duration of the exposure is lengthened.

The Effective Blood Volume – It has been seen that pressure breathing produces a displacement of blood into the peripheral capacity vessels and a loss of fluid from the blood into the extravascular spaces of the limbs. Both these changes reduce the volume of blood in the central part of the circulation. The concept of the "effective blood volume" Landis and Hortenstine 1950 (183) is of value in this context. In many situations the total blood volume may be normal but an increase in the volume of some or all of the peripheral vessels can reduce the volume of blood available to the heart per unit time for active recirculation. The "effective" blood volume is the volume of blood available at a given instant for the maintenance of the circulation. Although no satisfactory method is available as yet for measuring the absolute value of the effective blood volume, it is frequently possible to estimate the change of this quantity induced by a given procedure. Thus during pressure breathing the effective blood volume is reduced by the accumulation of blood in the peripheral vessels and by the passage of fluid from the capillaries in regions in which there is an increase of the vascular transmural pressure.

The blood content of a region will be increased and fluid will be lost from the capillaries of its tissues as long as the increase in tissue pressure produced by pressure breathing is less than the corresponding increase in central venous pressure. Within the thorax the tissue pressure will be raised by an amount which equals the increase of the intrapleural pressure, whilst within the abdomen the increase of tissue pressure will be only slightly less. Thus little or

no pooling of blood or increase of extravascular fluid will occur within the thoracic or abdominal cavities during pressure breathing. In the absence of counterpressure to the trunk an increase of the blood content and rate of fluid filtration will occur in the skin, subcutaneous tissues and muscles outside these cavities. When trunk counterpressure is employed, however, little pooling or filtration of fluid from the circulation will occur within the trunk. Pressure breathing does not increase the transmural pressures of the capillaries or of the capacity vessels of the central nervous system since the pressure of the cerebro-spinal fluid is increased by an amount which equals the rise of venous pressure. Blood pooling and fluid filtration will occur into those superficial tissues of the head and neck to which counterpressure is not applied. When a pressure headpiece is worn only a small portion of the neck and the crown of the head are unpressurized. Thus the blood and fluid displaced into the limbs represents virtually all the reduction of the effective blood volume produced by pressure breathing, particularly when trunk counterpressure is used.

The amount by which the effective blood volume is reduced by pressure breathing with trunk counterpressure has been estimated from the measurements of the volume of blood displaced in the limbs by various positive breathing pressures (Fig. 6–29), and the overall rate of filtration of fluid into the tissues of the limbs (0.99 ml/min./mmHg positive breathing pressure). The results of these calculations are presented in Fig. 6–30. At a given positive breathing pressure the rate of reduction of effective blood volume caused by the immediate displacement of blood into the limbs is approximately doubled by the loss of fluid from the circulation induced by pressure breathing for five minutes. The effects of the reduction of the effective blood volume induced by pressure breathing are manifold and are considered later in this discussion.

Peripheral Venous Pressure – The time taken for the venous outflow from a region to restart at the beginning of pressure breathing and hence for the peripheral venous pressure to become stabilized at a raised value depends upon the relationship between the rate of flow of blood into the region and the distensibility of its capacity vessels. The behaviour of the peripheral venous pressure during pressure breathing was investigated in three regions, namely the forehead, the upper limb and the lower limb. The venous pressure in the forehead increased to a steady value within five seconds of the beginning of pressure breathing (Fig. 6–11) whilst the pressure in an antecubital vein did not achieve a steady value until twenty seconds later. The blood flow to the skin of the head is relatively high (Kerslake, personal communication) while the veins of the scalp probably have a relatively low distensibility. The rapid rise of forehead venous pressure is also related to the valveless connections through the orbit between the intracranial and extracranial venous systems (167). The venous pressure within the skull rises very rapidly at the beginning of pressure breathing because the intracranial circulation lies within an indistensible container and the rise of venous pressure within the skull is transmitted to the extracranial veins of the forehead.

In the supine posture the pressure within a dorsal vein of the foot did not reach a steady value until nearly sixty seconds had elapsed from the commencement of pressure breathing. The time taken for the venous pressure in the foot to attain a steady value was reduced to fourteen seconds by the

assumption of the seated posture (Fig. 6-11). The increased rate of rise of venous pressure associated with the seated position was probably due to the postural reduction in the distensibility of the capacity vessels of the lower limb. In a relaxed subject the venous pressure at a given horizontal level in the lower limb is the pressure exerted by the column of blood between that level and the right atrium. When seated, therefore, the veins are already subjected to a high transmural pressure and the distensibility will be much less than in the supine posture when many of the veins are collapsed. The increase in the rate of rise of the venous pressure in the foot at the beginning of pressure breathing when a subject is in the seated position is unlikely to be due to an increase of the rate of blood flow into the limb. The assumption of the seated or erect posture is associated with a generalized arteriolar vasoconstriction (45) so that the arterial inflow into the lower limb will be lower in the seated position than in the supine, at least in the resting state before the commencement of pressure breathing.

After the initial increase in pressure at the beginning of pressure breathing the peripheral venous pressure always exhibited obvious variations which coincided in time with respiration. In some experiments small respiratory fluctuations were seen at rest but these always disappeared during the period in which the venous pressure was rising at the beginning of pressure breathing. Detailed analysis of these pressure fluctuations showed that the pressure in the antecubital vein fell during inspiration and rose in expiration both at rest and during pressure breathing (Fig. 6-10). These fluctuations of peripheral venous pressure are a reflection of the changes of the right atrial pressure during the respiratory cycle. Changes of intrapleural pressure are transmitted directly through the walls of the heart to the blood within its cavities. During inspiration the intrapleural pressure falls and thus induces a similar change in the mean pressure in the right atrium which, under certain circumstances, is reflected in the peripheral venous pressure. The peripheral venous pressure will only follow changes in the right atrial pressure when there is a continuous column of blood between the peripheral site of measurement and the right atrium. Thus if the dependent arm is placed in a suitable position respiratory fluctuations are apparent in the peripheral venous pressure.

At the beginning of pressure breathing the valves in the veins at the root of the limb are shut since the central venous pressure exceeds the peripheral venous pressure. Once, however, the peripheral venous pressure has risen so that venous return recommences, the peripheral veins are widely distended by the high transmural pressure and changes of right atrial pressure are reflected in the peripheral veins. The magnitude of the respiratory fluctuations seen in the records of peripheral venous pressure in the upper limb were always greater during pressure breathing than at rest. This difference was due to the considerably lower distensibility of the peripheral veins when the pressure within them was raised and to the increase of the respiratory fluctuations of intrapleural pressure associated with the hyperpnoea of pressure breathing. The part played by the reduction of the venous distensibility at increased venous pressure was confirmed by the decrease in the fluctuations of limb volume produced by pressure breathing (Fig. 6-11). When the upper limb was placed in a suitable position at rest, fluctuations in the volume of the forearm occurred which were respiratory in timing. In these circumstances

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the magnitude of the fluctuations of limb volume due to respiration was reduced during pressure breathing, whereas the fluctuations in local venous pressure were increased.

In pressure breathing the peripheral venous pressure measured in the antecubital veins exhibited small fluctuations, the frequency of which was equal to the heart rate. These particular pressure changes persisted when a sphygmomanometer cuff, placed around the upper arm, was inflated to a pressure 20 mmHg greater than the applied positive breathing pressure. The fluctuations did not depend, therefore, upon the presence of a continuous column of blood from the peripheral veins to the right atrium. Hence they were not a reflection of the pressure changes in the right atrium during the cardiac cycle. Very similar fluctuations in peripheral venous pressure were produced by local venous congestion of the upper limb in a subject at rest. Simultaneous measurements of the pressures in the brachial artery and the antecubital vein of the same limb showed that the increase in venous pressure with each cardiac cycle coincided with the systolic rise of arterial pressure. The cardiac fluctuations which occurred in the peripheral venous pressure during pressure breathing are due, therefore, to the direct transmission of the arterial pulse to the blood in the venae comitantes of the arterial system in the limb. This transmission occurs in pressure breathing and with local venous congestion because the veins accompanying the arteries are distended and the volume pulse in the arterial tree is transmitted to their contents.

The relationship between the increase of peripheral venous pressure (measured at the end of expiration) produced by pressure breathing and the corresponding positive breathing pressure, varied with the degree of counter-pressure applied to the trunk (Figs. 6-12 and 6-13). In the absence of respiratory counterpressure the difference between the positive breathing pressure and the corresponding increase of peripheral venous pressure increased progressively with the magnitude of the applied breathing pressure (Fig. 6-12). At positive breathing pressures above 25 mmHg this difference was relatively constant at approximately 12 mmHg. Similar results were obtained by Otis, Rahn and Fenn 1946 (232) who also studied the effects of pressure breathing upon the peripheral venous pressure. The primary cause of the discrepancy between the applied positive pressure and the consequent increase of venous pressure is the increased elastic recoil of the lung associated with the raised functional residual capacity which occurs in pressure breathing without respiratory counterpressure. The magnitude of the difference between the positive breathing pressure and the change of venous pressure was of the same order as the change in expiratory intraoesophageal pressure produced by the same positive breathing pressure (Table 6-11). The importance of the increase of the functional residual capacity was confirmed by the marked reduction of the difference between the applied breathing pressure and the corresponding increase of venous pressure produced by the application of counterpressure to the trunk during pressure breathing (Fig. 6-13). At higher positive breathing pressures the rise of venous pressure was 4 to 6 mmHg less than the corresponding increase of intrapulmonary pressure when trunk counterpressure was employed. This difference was greater than the corresponding change of end-expiratory intraoesophageal pressure induced by pressure breathing with trunk counterpressure (Table 6-11).

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TABLE 6-11

THE EFFECT OF PRESSURE BREATHING UPON PERIPHERAL VENOUS
PRESSURE AND MOUTH-INTRAOESOPHAGEAL PRESSURE DIFFERENCE

Positive breathing pressure (mmHg)	Difference between positive breathing pressure and change* of venous pressure (mmHg)	Change* of mouth-intraoesophageal pressure difference (mmHg)
A. No counterpressure		
10	3.2	3.5
20	8.0	7.3
30	10.5	11.0
B. Trunk counterpressure		
20	0.5	0.3
40	4.0	2.0
60	6.0	2.5
80	8.0	2.8

* Measured from the value in the resting state

TABLE 6-12

THE RELATIONSHIP BETWEEN REDUCTION OF EFFECTIVE RIGHT
ATRIAL PRESSURE AND REDUCTION OF EFFECTIVE BLOOD VOLUME
DURING PRESSURE BREATHING

Positive breathing pressure (mmHg)	Reduction of right atrial pressure (cm of water)		Reduction of effective blood volume (ml)	Mean reduction of right atrial pressure per 100 ml reduction of blood volume	
	Subject A	Subject B		Subject A	Subject B
30	0.8	1.1	230	0.35	0.48
60	1.9	2.9	430	0.45	0.67
80	2.8	4.6	540	0.52	0.85

Since the peripheral veins were distended and the rate of blood flow through the limbs was reduced during pressure breathing, the pressure gradient between the peripheral veins and the right atrium was probably reduced by this manoeuvre. Thus the increase of peripheral venous pressure induced by a given positive breathing pressure was probably slightly less than the corresponding rise of right atrial pressure. Further, the induction of pressure breathing increased the right atrial pressure by an amount which was slightly less than the corresponding rise of intraoesophageal pressure (Fig. 6–15). Both these factors would tend to reduce the rise of peripheral venous pressure for a given increase of intraoesophageal pressure and they probably account for the difference observed experimentally between the changes in these two pressures (Table 6–11).

The increase of peripheral venous pressure induced by pressure breathing at a given positive pressure without respiratory counterpressure was considerably increased by the application of counterpressure to the trunk (Figs. 6–12 and 6–13). Thus at a given positive pressure the use of trunk counterpressure increased the amount of blood displaced into the limbs and the rate of loss of fluid from the circulation. If it is assumed that the application of counterpressure to the trunk does not change the distensibility of the peripheral capacity vessels, it is possible to calculate the increase in the reduction of the effective blood volume associated with the use of this counterpressure. Thus at a positive breathing pressure of 30 mmHg the application of counterpressure to the trunk will increase the immediate reduction of the effective blood volume from 125 ml to 175 ml and the subsequent rate of reduction of blood volume from 20 ml/min. to 30 ml/min. The use of trunk counterpressure during a five minute exposure to a positive breathing pressure of 30 mmHg will cause a reduction of the effective blood volume which is 50% greater than that produced by pressure breathing without respiratory counterpressure. This greater reduction of the effective blood volume associated with the use of counterpressure to the trunk is a distinct disadvantage. The reduction of respiratory fatigue which trunk counterpressure affords, however, outweighs the increased circulatory embarrassment, particularly at positive breathing pressures greater than 30 mmHg.

Central Venous Pressure – During diastole the relationship between the pressures in the right atrium and the pleural cavity (effective right atrial pressure) is determined by the tone of the atrial wall and the rate of the venous return to the heart. If these factors remain constant then a given rise of intra-pleural pressure will produce an equal rise of pressure in the right atrium. At rest the amplitude of the respiratory fluctuations of right atrial pressure amounted to about half the corresponding changes of intraoesophageal pressure (Fig. 6–14). A similar result was obtained by Bloomfield, Lawsen, Cournand, Breed and Richards 1946 (38) who recorded simultaneously the pressure changes in the right atrium and in a small pneumothorax. The smaller amplitude of the respiratory fluctuations of right atrial pressure was probably due to an increase in the venous return during inspiration which increased the effective right atrial pressure.

The relatively large increase of intrapleural pressure associated with the induction of pressure breathing was transmitted directly to the blood within the heart so that there was an equally rapid rise of right atrial pressure at the

beginning of pressure breathing. At this time the fluctuations of right atrial pressure during the cardiac cycle were markedly reduced. This diminution of the cardiac fluctuations of pressure was due to a reduction in the venous return to the heart since the central venous pressure was raised above the venous pressure in the limbs. The tissue pressure within the thorax, the skull and, to a considerable extent, the abdomen increases with the central venous pressure at the beginning of pressure breathing so that there is little or no impairment of venous return from these regions, except that which arises reflexly. At rest about 65% of the total venous return to the right heart comes from the organs within the thorax, skull and abdomen (280). Thus, if no reflex changes in the arteriolar and venous tone occur, the venous return to the heart would be reduced by at least one third directly pressure breathing was commenced. It has been shown, however, that pressure breathing induces constriction of the resistance vessels in both the skin (102) and the muscle (37) of the limbs. Such constriction if localized to the limbs, would reduce the magnitude of the fall of the venous return since it would divert the cardiac output to regions in which there is no increase in the capacity of the vascular bed.

Following the recommencement of venous return from the limbs, there was, however, a reduction in the effective right atrial pressure as compared with that which existed at rest (Fig. 6–15). The pattern of the right atrial pressure changes during the cardiac cycle and the modifying effects of respiration were also altered in pressure breathing. During expiration the amplitude of the "a" wave, which is produced by atrial systole was much smaller during pressure breathing than at rest (Fig. 6–14). The fall of mean right atrial pressure induced by inspiration was greater in pressure breathing and the amplitude of the "a" wave was markedly increased. These more profound changes produced by respiration during pressure breathing suggest that respiration exerts a greater influence on the venous return during pressure breathing than it does at rest.

The fall of the effective right atrial pressure induced by pressure breathing was a result of the reduction of the effective blood volume which is produced by this manoeuvre. The changes of the effective blood volume produced by pressure breathing for two minutes at the positive pressures used in the studies of right atrial pressure have been determined from the curves of Fig. 6–30 and are presented in Table 6–12, together with the corresponding changes of the effective right atrial pressure. The reduction of effective right atrial pressure per unit reduction of effective blood volume may be compared with those obtained when the blood volume is reduced by venesection or by the application of venous torniquets to the limbs. McMichael and Sharpey-Schafer 1944 (224) used both these procedures and measured the corresponding changes of right atrial pressure by means of a cardiac catheter. They found that a venesection of 420 ml reduced the right atrial pressure by 2.5 cm water, whereas venous congestion of the lower limbs, which reduced the effective blood volume by about 700 ml, reduced the right atrial pressure by 5 cm water.

Gauer, Henry and Sieker 1953 (121) bled six subjects and determined the effect of this procedure upon the right atrial pressure. The fall of right atrial pressure was directly proportional to the volume of blood removed and

amounted to 0.7 cm water per 100 ml blood removed. Thus the relationship between reduction of effective blood volume and the consequent fall of effective right atrial pressure found during pressure breathing was similar in one subject (subject B, Table 6–12) to that obtained by McMichael and Sharpey-Schafer 1944 (224) and Gauer, Henry and Sieker 1953 (121). In the other subject (subject A, Table 6–12) the fall of right atrial pressure induced by pressure breathing was somewhat less than that found in haemorrhage. Although the number of experiments in which the effective right atrial pressure was measured in pressure breathing was small, the results obtained support the concept that the effective right atrial pressure was reduced by the peripheral displacement of blood and the loss of circulating fluid into the tissues of the limbs.

Cardiac Output – A fall of the filling pressure of the right atrium was shown by Knowlton and Starling 1912 (172) to reduce the output of the heart in the heart and lung preparation. Sarnoff and Mitchell 1961 (258), who repeated Starling's experiments, confirmed in the isolated dog's heart the relationship between atrial pressure and ventricular output embodied in Starling's law. In 1944 McMichael and Sharpey-Schafer (224) showed that in the intact man there was a broad direct relationship between right atrial pressure and cardiac output when the former was varied by venesection and by the infusion of saline. There was, however, considerable variation in response of the cardiac output to a given change of right atrial pressure from one experiment to another. Part of this at least was due to the variability in the response of the heart rate to the changes of right atrial pressure. Recently Braunwald, Bloodwell, Goldberg and Morrow 1961 (43) showed that the human heart obeys Starling's law when nervous influence is reduced by ganglion blocking agents. In the intact individual however, the sympathetic and vagal efferent nerves to the heart modify its response to a given change of right atrial pressure. It is probable, however, that the very marked fall of effective right atrial pressure induced by pressure breathing would lead to a reduction of the cardiac output.

Although no direct measurements of cardiac output were made in the present study, other investigators have shown that pressure breathing reduces the output of the heart. Using a ballisto-cardiographic method Otis, Rahn, Brontman, Mullins and Fenn 1946 (231) found that in trained individuals pressure breathing at a positive pressure of 22 mmHg reduced the cardiac output by 14%. The stroke volume was decreased by 26% but there was a simultaneous increase in the heart rate. A similar reduction of cardiac output was found by Barach, Eckman, Ginsburg, Rumse Corr, Eckman and Besson 1946 (17) who determined the change of cardiac output produced by pressure breathing using the direct Fick method. Cain and Mahoney 1953 (53) also used a ballisto-cardiograph to determine the changes of cardiac output induced by pressure breathing. They found a positive breathing pressure of 40 mmHg reduced the cardiac output by 35 to 50% when no respiratory counterpressure was used and by 30 to 40% when a vest was employed to apply counterpressure to the upper part of the trunk. Using the acetylene method developed by Grollman 1929 (133), Parry (personal communication) measured the cardiac output during pressure breathing with and without trunk counterpressure at a positive pressure of 30 mmHg. In

the absence of counterpressure pressure breathing reduced the cardiac output by 30%. When counterpressure was applied to the trunk the reduction of cardiac output induced by a positive breathing pressure of 30 mmHg amounted to between 15 and 20%. It would appear likely, therefore, that pressure breathing with respiratory counterpressure at a positive pressure of 30 mmHg reduces the cardiac output by about 30%. Apart from a few determination made by Cain and Mahoney 1953 (53) in which their subjects wore the capstan partial pressure suit, there have been no measurements of the effects of pressure breathing upon the cardiac output at positive pressures above 40 mmHg. It would appear probable, however, that the cardiac output falls as the positive breathing pressure is increased.

The Arterial Pressure – The systemic arterial pressure was always increased by pressure breathing, although the relationship between the increase of arterial pressure and the positive breathing pressure varied with the degree of counterpressure applied to the surface of the body. In the experiments in which a bladder garment was used the pressure in the headpiece did not achieve the desired value for about five seconds and the arterial pressure increased simultaneously with the rise of intrathoracic pressure (Fig. 6–16). When no counterpressure was used, however, the headpiece pressure increased abruptly at the beginning of pressure breathing and there was an equally rapid increase of arterial pressure. The magnitude of this increase of arterial pressure was equal to the rise of intrathoracic pressure. This increase of arterial blood pressure which was maintained for only two or three beats, was followed by a reduction of the mean pressure and of the pulse pressure. The mechanism underlying the initial rapid rise of arterial pressure in pressure breathing is analogous to the initial increase of arterial pressure produced by the Valsalva manoeuvre (264). It is due to the direct transmission of the rise of intrapleural pressure to the left ventricle and the systemic arteries within the thorax and abdomen. The decline of the arterial pressure and the reduction of the pulse pressure which followed the initial increase of arterial pressure is a reflection of the decrease in the venous return to the right atrium. Although these two phases in the arterial pressure response were clearly discernible when the intrathoracic pressure was raised rapidly by pressure breathing without counterpressure, the initial rapid increase became submerged in the subsequent fall of pressure when the intrathoracic pressure was increased more slowly over four to five seconds. Thus the initial changes of arterial pressure in pressure breathing are due to the direct transmission of the rise of the intrapleural pressure to the intrathoracic vessels and the subsequent decrease in the venous return to the right side of the heart.

Following the initial changes associated with the increase of intrathoracic pressure the arterial pressure usually became steady until ten to fifteen seconds after the beginning of pressure breathing. At this point the arterial pressure frequently increased by a small amount to reach a level which was maintained, with respiratory fluctuations, for the remainder of the exposure to pressure breathing. This small additional increase of arterial pressure occurred at a time when the venous return from the limbs restarted. It was probably due, therefore, to the consequent increase in the cardiac output. Sharpey-Schafer 1955 (264) suggests that the increase of mean arterial pressure and pulse pressure seen five to six seconds after the beginning of the

Valsalva manoeuvre is due to the onset of arteriolar constriction. It is probable that such peripheral vasoconstriction also played a part in this rise of arterial pressure seen ten to fifteen seconds after the beginning of pressure breathing.

In the steady state the increase of arterial pressure induced by pressure breathing was less than the positive breathing pressure even when counter-pressure was applied to the whole of the surface of the trunk (Fig. 6–17). However, there was a marked decrease of the arterial pulse pressure (Table 6–2). If no circulatory disturbances occurred, a given rise of intrapleural pressure should produce an equal increase in the arterial pressure with no change of pulse pressure. The primary factor responsible for the failure of the arterial pressure to follow precisely the increase of intrapleural pressure in pressure breathing is the reduction of the effective blood volume and the consequent reduction of the cardiac output induced by this procedure. Counterpressure to the limbs serve to reduce the fall in the effective blood volume and so the mean arterial and pulse pressures were raised (Fig. 6-17, Table 6–2). The application of respiratory counterpressure also increases the arte.ial pressure at a given positive breathing pressure by reducing the lung distension and hence producing a greater increase of intrapleural pressure that would otherwise occur. This latter mechanism is of particular significance at positive breathing pressures less than 30 mmHg since at this level a large fraction of the applied breathing pressure may be expended in overcoming the elastic recoil of the lungs.

The increase of arterial pressure and the decrease of pulse pressure observed during pressure breathing is not, however, simply an expression of the interaction between the rise of intrapleural pressure and the fall of cardiac output. Pressure breathing induces a marked increase of peripheral resistance as does the Valsava manoeuvre (37) (142). Thus the peripheral resistance in the forearm is approximately doubled by pressure breathing at a positive pressure of 60 mmHg when trunk counterpressure is employed (Ernsting, unpublished observation). A similar arteriolar constriction may well occur in the abdominal viscera during this procedure, although no direct measurements have been made of the blood flow through these organs in this condition. Such a generalized arteriolar constriction would tend to maintain the arterial pressure during pressure breathing in the face of a decrease in the cardiac output. The importance of this mechanism in maintaining the arterial pressure when the intrathoracic pressure is raised is shown by the effect of sympathetic ganglion blocking agents upon the response to the Valsalva manoeuvre. Thus, when the Valsalva manoeuvre is performed following the administration of a ganglion blocking agent such as tetraethylammonium chloride, the arterial pressure continues to fall following the initial rise at the beginning of the manoeuvre.The normal secondary increase of arterial pressure which occurs whilst the intrathoracic pressure is raised is absent after the administration of such a blocking agent (130).

The reflex constriction of the peripheral resistance vessels induced by pressure breathing is an important factor in the maintenance of an effective systemic arterial pressure during this manoeuvre. There are several possible sites for the receptors which are responsible for this reflex. A decrease of the activity of the carotid and aortic baroreceptors results in reflex peripheral

vasoconstriction. The activity of these receptors depends upon the vascular transmural pressure. When a pressure headpiece, which applies counter-pressure to the skin overlying the carotid sinus, is used the vascular trans-mural pressures of both the carotid sinus and the aorta are reduced by the induction of pressure breathing since the rise of arterial pressure is less than the increase of the pressure applied to the neck by the headpiece and the rise of intrapleural pressure. Not only is the mean arterial transmural pressure reduced by pressure breathing, but probably of greater importance, with respect to baroreceptor activity, is the concomitant reduction of the arterial pulse pressure. Thus Ead, Green and Neil 1953 (86) have shown that the reflex effect upon the systemic pressure of a pulsatile pressure in the carotid sinus is much greater than that of a steady pressure at the same mean value.

Stimulation of receptors in the low pressure regions of the intrathoracic vascular bed can also produce reflex changes of peripheral vascular resistance. Thus raising the pressure in the right side of the heart in the dog produced a reflex bradycardia and peripheral arteriolar dilatation (12) although the actual site of the receptors underlying these reflex changes is uncertain. There is also a considerable body of evidence (70) (12) that reflex changes of peri-pheral resistance can arise from receptors situated in the walls of the pul-monary veins and the left atrium. In man, Roddie and Shepherd 1956 (247) have demonstrated reflex changes in the tone of the resistance vessels of the forearm as a result of alterations of the volume of blood within the chest. These changes occurred without any significant change of systemic arterial pressure so it is unlikely that they were due to alterations in arterial baro-receptor activity. The reflex increase of peripheral resistance induced by pressure breathing probably arises, therefore, from changes in the activity of receptors situated both in the low pressure regions of the intrathoracic circula-tion and the aortic and carotid sinus regions.

In most of the exposures to pressure breathing in which counterpressure was applied to the lower limbs as well as to the trunk the increase of mean arterial pressure actually exceeded the positive breathing pressure (Fig. 6-17). Even with this degree of counterpressure, however, the arterial pulse pressure was less during pressure breathing than at rest (Table 6-2) which suggests that the stroke volume of the heart was reduced by pressure breathing under these conditions. This increase of arterial pressure relative to the intrapleural pressure during pressure breathing was due primarily, therefore, to a marked increase of peripheral resistance, although the associated tachycardia would also have contributed to the increase of blood pressure in view of the reduction of the stroke volume.

The rapid decrease of the arterial pressure when the headpiece pressure was reduced to zero at the end of a period of pressure breathing (Fig. 6-16) was a direct effect of the reduction of the intrathoracic pressure. The arterial pulse pressure which remained less than normal for several beats after the reduction of the headpiece pressure increased to a value which was con-siderably greater than the pulse pressure in the resting state. This increase was due primarily to the rise in stroke volume of the left ventricle which occurred at this time. As the intrapleural pressure fell the venous return to the right side of the heart was greatly augmented by the release of the blood held in the widely distended capacity vessels during pressure breathing. Although

this influx of blood occurred very rapidly there was a delay of about two seconds before the stroke volume of the left ventricle was increased because of the capacity and resistance of the intervening pulmonary vascular bed. The persistence of the increase in peripheral resistance induced by pressure breathing into this period also played a part in the genesis of the large arterial pulse pressure and the increase in mean arterial pressure which occurred after the cessation of pressure breathing. This response is analogous to the "overshoot" of arterial pressure which follows cessation of the Valsalva manoeuvre. The increase of arterial pressure which follows release of raised intrapulmonary pressure is abolished by the administration of a ganglion blocking agent which prevents peripheral vasoconstriction (130). The cardiac slowing associated with the large increase of arterial pulse pressure which followed cessation of pressure breathing probably arose from stimulation of the carotid and aortic baroreceptors.

The blood flow through an organ is determined by the difference between the local arterial and venous pressures and the resistance to flow offered by the vascular bed. Although in pressure breathing the arterial pressure is raised considerably, in most situations the peripheral venous pressure is raised by an amount which either equals or exceeds the increase of arterial pressure after the initial fifteen to twenty seconds of pressure breathing. Thus the arterio-venous pressure difference is slightly reduced by pressure breathing so that in the absence of any change of peripheral resistance the regional blood flow would be reduced. Although there is an overall increase of peripheral resistance in pressure breathing, only limited studies of regional blood flow have been performed in this condition. There is strong evidence that arteriolar vasoconstriction occurs in both the skin and muscles of the limbs with a consequent reduction of blood flow (102) (37); Ernsting, personal observation. No direct measurements have been made of cerebral blood flow during pressure breathing at high positive pressures, although it is known that the most important factors controlling blood flow through the brain are the tensions of oxygen and carbon dioxide in the arterial blood. The significance of this control mechanism during pressure breathing was discussed in the previous chapter. Nothing is known concerning blood flow through other regions during pressure breathing, although there must be an overall reduction of blood flow since the cardiac output is reduced.

Heart Rate – The magnitude of the increase of the heart rate induced by pressure breathing depended upon the positive breathing pressure, the area of the body to which counterpressure was applied and the duration of the exposure (Figs. 6–19 and 6–20). Thus the greater the positive breathing pressure and the longer the time for which it was operative, the higher was the heart rate. The cardiac acceleration generally commenced within five seconds of the beginning of an exposure to pressure breathing, particularly at the higher positive pressures. The reflexes underlying the tachycardia induced by pressure breathing probably arise from receptors similar or identical to those which are responsible for the reflex peripheral vasoconstriction which also occurs in this condition. Thus a reduction in the effective filling pressure of the right or left side of the heart will give rise to tachycardia (12). Furthermore a reduction of the mean and pulse pressure in the carotid baroreceptor region causes a reflex cardiac acceleration (86). The observed rise of the heart

rate with either an increase in the positive breathing pressure or the prolongation of an exposure was probably due to the associated decrease of the effective blood volume and hence of the effective filling pressure of the heart The effects of applying counterpressure to a large proportion of the surface of the body is probably also explicable on the basis of the increase in the effective blood volume associated with the application of counterpressure. The temporary reduction of heart rate associated with contraction of the muscles of the upper limbs (Fig. 6-21) was due to a transient increase of the venous return to the heart. This increase in venous return was produced by the rise of tissue pressure in the limbs associated with muscular contraction.

The electrocardiographic changes produced by pressure breathing (Fig. 6-18) indicated that there was an alteration of the electrical axis of the heart. A detailed analysis of these changes was not undertaken but the recordings of the standard limb leads showed that pressure breathing rotated the electrical axis of the heart in the frontal plane in a clockwise direction. Since the application of counterpressure to the trunk reduced the magnitude of this rotation (Fig. 6-18) the rotation was probably associated with the respiratory disturbance induced by pressure breathing, in particular the increase of the volume of gas within the respiratory tract. A similar clockwise rotation of the electrical axis of the heart was produced by a deep inspiration. In both instances the mechanism underlying the rotation of the electrical axis was a clockwise rotation of the heart itself in the frontal plane. This rotation was induced by descent of the diaphragm.

Pressure Breathing Syncope – Under certain circumstances the subject exposed to pressure breathing may suffer a syncopal attack. The clinical features of the syncopal attacks which were observed in the present investigation were remarkably uniform and they conformed closely to those of vasovagal syncope. Lewis 1932 (187) studied the mechanism of fainting and he demonstrated that the acute fall of arterial pressure which characterizes this condition persisted when the associated bradycardia was abolished by the administration of atropine. He introduced the term "vasovagal" to emphasize the importance of peripheral vasodilatation as well as bradycardia in the genesis of the acute hypotension which characterizes a faint.

Interest in vasovagal syncope was intensified during World War II since it frequently occurs in individuals who have suffered a haemorrhage. Thus Wallace and Sharpey-Schafer 1941 (281) showed that the incidence of fainting increased with the volume of blood withdrawn from the circulation. Although McDowall 1938 (217) had suggested that John Hunter's observation that the blood flowing from a vein became bright red when a patient fainted indicated that peripheral vasodilatation occurred in fainting, no direct measurements of peripheral blood flow were made in this condition until 1944. Barcroft, Edholm, McMichael and Sharpey-Schafer 1944 (23) found that there was a decrease of vascular resistance in the forearm during a faint and Barcroft and Edholm 1945 (22) showed that this was due to active vasodilatation in muscles and a constant feature of vasovagal syncope whatever the precipitating cause. In several experiments the change of peripheral resistance was determined during pressure breathing syncope by the technique developed by Hayter and Sharpey-Schafer 1958 (142). In each instance of syncope in these experiments the fall of arterial pressure was

associated with a marked decrease of peripheral resistance in the forearm (Ernsting, personal observation). Thus the cardiovascular changes which have been observed during pressure breathing syncope are exactly similar to those which characterize vasovagal syncope due to haemorrhage, prolonged standing in the upright posture emotional disturbance and hypoxia.

A reduction of the effective blood volume, whether produced by haemorrhage or by venous congestion of the limbs (87) will, if of sufficient magnitude, induce a vasovagal faint. Since pressure breathing reduces the effective blood volume it would appear likely that this disturbance may be responsible for syncope in pressure breathing. This manoeuvre does, however, produce other disturbances which are known to precipitate vasovagal syncope in susceptible subjects. These include emotional disturbances such as dislike of wearing a headpiece, which may occur in an inexperienced subject, and discomfort or frank pain due to an ill-fitting garment. In certain circumstances in which experienced subjects developed syncope during pressure breathing, none of these additional disturbances was evident and it is probable that syncope under these circumstances was due primarily to a reduction of the effective blood volume. Where experiments were performed with trunk counter-pressure, it is possible to estimate from the positive breathing pressure and the duration of the exposure when syncope occurred the approximate reduction of the effective blood volume using the curves presented in Fig. 6-30. The reduction of effective blood volume which had been produced in each of these exposures when syncope occurred lay between 660 and 900 ml (Table 6-13).

These values may be compared with the relationship between the volume of blood withdrawn from semi-reclining subjects and the incidence of fainting found by Wallace and Sharpey-Schafer 1941 (281). Approximately 20% of their subjects fainted when 700 ml of blood was withdrawn whereas syncope occurred in 50% when 1000 ml was removed. Thus the reductions of the effective blood volume induced by pressure breathing when syncope occurred were of the same order as that which induced vasovagal syncope in 20% to 40% of normal subjects. Furthermore, Ebert and Stead 1940 (87) found that the application of venous tourniquets to one upper and both lower limbs for ten minutes, which reduced the volume of blood circulating in the head, trunk and one arm by an average of 720 ml, induced symptoms of vasovagal syncope in four out of seven subjects. This evidence supports the hypothesis that pressure breathing syncope in instances where there is no discomfort or emotional disturbance is due primarily to a reduction of the effective blood volume.

However, many incidents of pressure breathing syncope occurred, where the reduction of effective blood volume was not so great and the presence of other precipitating factors had to be considered (Table 6-4). Since in the majority of these syncopal attacks counterpressure was also applied to the lower limbs by means of an anti-g suit, it is necessary to consider the effect of this counterpressure upon the displacement of blood and fluid into the lower limbs. When a jerkin and anti-g suit are used during pressure breathing, counterpressure is applied to between 75 and 80% of the surface of the lower limbs. Measurement of the tissue pressure beneath an anti-g suit has shown that this garment raises the pressure in the underlying tissues by an amount which equals the gas pressure in the suit bladder, Ernsting 1955 (91). It has

TABLE 6-13

THE REDUCTION OF EFFECTIVE BLOOD VOLUME ASSOCIATED WITH SYNCOPE DURING PRESSURE BREATHING WITH TRUNK COUNTERPRESSURE IN EXPERIENCED SUBJECTS IN THE ABSENCE OF DISCOMFORT AND PAIN

Positive breathing pressure (mmHg)	Duration of exposure when syncope occurred (min.)	Reduction of effective blood volume (ml)
60	10	900
60	6	660
64	7.5	800
67	8.4	850
70	6.5	790
80	4.3	730
90	4.5	830
90	2.4	630
100	4.0	880
115	2.0	740
130	2.5	720

TABLE 6-14

THE REDUCTION OF EFFECTIVE BLOOD VOLUME WHEN SYNCOPE OCCURRED DURING PRESSURE BREATHING

Positive breathing pressure (mmHg)	Duration of exposure when syncope occurred (min.)	Reduction of effective blood volume (ml)	Contributing factors Experience	Others
A. Helmet and jerkin				
80	2.5	550	Minimal:	Headpiece discomfort
80	3.5	620	Minimal:	Hyperventilation and arm pain
B. Helmet, jerkin and anti-G suit				
80	4.5	360	Minimal:	Intense dislike of headpiece
80	4.5	360	Minimal:	Marked hyperventilation
80	5.5	400	Minimal:	Hyperventilation
80	6.0	420	Minimal:	—
80	6.5	440	Minimal:	Arm pain
107	3.5	390	Considerable:	Arm and head pain
107	4.0	420	Considerable:	Arm pain: hyperventilation
120	2.5	390	Considerable:	Severe arm pain
C. Helmet, arm jerki and anti-G suit				
110	3.5	210	Minimal:	Headpiece pain
140 decreasing to 0 in 5 min.	3.0	230	Minimal:	Hyperventilation and arm pain

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been assumed, therefore, that the inflation of the anti-g suit to a pressure (gauge) which equals the positive breathing pressure reduces the volume of blood displaced and the rate of loss of fluid into the tissues of the lower limbs to 25% of the values which were measured during pressure breathing without counterpressure. The results of the calculations of the reduction of the effective blood volume produced by pressure breathing when syncope occurred are presented in Table 6–14. These calculations show that syncope occurred in more than half the subjects when the reduction of effective blood volume did not exceed 400 ml. In each of these instances there was an obvious disturbance in addition to that of pressure breathing. The potentiating effect of emotional disturbances, pain and hypoxia upon the development of vaso-vagal syncope due to a small reduction of effective blood volume is well documented (90). Hypocapnia which occurred frequently during pressure breathing in inexperienced subjects also increased the likelihood of pressure breathing syncope. Thus pressure breathing with trunk counterpressure at a positive pressure of 60 mmHg induced collapse in three out of six subjects when the alveolar carbon dioxide tension was reduced to 25 mmHg whereas none of the subjects collapsed when the alveolar carbon dioxide tension remained above 35 mmHg (Ernsting, Green, McHardy and Wagner, un-published observation).

The reflexes which give rise to the cardiovascular changes which occur in pressure breathing syncope are probably the same as those which occur in vasovagal syncope due to haemorrhage, emotional disturbances, etc. Whilst it is known that the vagal fibres to the heart and the sympathetic vasodilator fibres to the vessels of the skeletal muscle form the efferent limbs of these reflexes the receptors and afferent pathways have not been defined. The evidence which is available suggests that the receptors concerned lie within the heart. Thus the left atrium contains receptors, the rate of firing of which depends upon the degree of distension of these cavities (150). Sharpey-Schafer, Hayter and Barlow 1958 (266) have suggested that the large pressure transients which occur in the left ventricle during systole when the volume of blood in it is reduced to virtually zero may stimulate receptors which are responsible for the vasovagal reflexes. This hypothesis is supported by the absence of fainting in patients in heart failure following large venesections (159). In such patients even a large venesection does not reduce the filling pressure of the heart to a point at which the left ventricle is emptied com-pletely during systole. It is likely, therefore, that when syncope occurs in pressure breathing the reduction of effective blood volume induced by the manoeuvre has decreased the effective filling pressure of the heart to such an extent that the consequent change in the pattern of afferent impulses from receptors within the heart fires the central mechanism which induces peri-pheral vasodilatation and bradycardia.

Duration of Protection – The occurrence of syncope forms the absolute limit to the time for which pressure breathing can be performed. Although the primary cause of syncope during pressure breathing is the reduction of the effective blood volume beyond a certain value, in many instances before this value was reached syncope was precipitated by emotional disturbances, the presence of discomfort or pain and by hypocapnia. The several groups of experiments which were performed at ground level (Table 6–3) gave an

indication of the relationship between the time for which pressure breathing could be performed without the occurrence of syncope, the positive breathing pressure and the degree of counterpressure applied to the surface of the body. Although the number of subjects used in some of these experiments was relatively small it is possible to draw certain conclusions with regard to the time for which pressure breathing can be performed using a particular assembly of pressure clothing. Thus, at a positive breathing pressure of 80 mmHg the duration of protection against syncope afforded to inexperienced subjects by the jerkin alone is less than four minutes whilst the addition of the counterpressure given to the lower limbs by the anti-g suit prevents syncope arising within this time. At a higher positive breathing pressure (107 mmHg) syncope occurred within four minutes even when the anti-g suit was used with the pressure jerkin. In nearly all the exposures to this positive breathing pressure the subjects experienced upper arm discomfort (Table 6-5) and the duration of protection given by this assembly is limited to about two minutes. The application of counterpressure to the upper limbs by means of the arm jerkin in addition to the lower limbs extended the duration of protection afforded against a positive breathing pressure of 110 mmHg to at least four minutes.

These experiments also demonstrated the influence of previous experience of pressure breathing at positive pressures of the order of 60–80 mmHg upon the incidence of syncope during a given exposure. The main factors responsible for the decrease in the incidence of pressure breathing syncope with training are familiarity with the equipment, with the discomfort, particularly that arising in the upper arm and the adoption of a more nearly normal breathing pattern. Many subjects hyperventilate markedly during their first few exposures to pressure breathing. Most subjects, however, rapidly adapt to the unusual sensations associated with high positive breathing pressures and exhibit only a small degree of hyperventilation after training. With a moderate degree of training consisting of approximately six exposures to positive breathing pressures above 50 mmHg the incidence of syncope was significantly reduced as compared with the incidence when the subjects had had no previous experience of the manoeuvre.

The time for which pressure breathing was performed by moderately experienced subjects without the occurrence of syncope is summarized in Table 6-15, in relation to the positive breathing pressure and the degree of counterpressure applied to the surface of the body. After adequate training the principal factors precipitating syncope during pressure breathing were severe discomfort or frank pain in the upper arm, neck or head. When the standard pressure jerkin was used the incidence of severe upper arm discomfort became considerable at positive breathing pressures above 80 mmHg, whilst pain was common when the positive pressure was greater than 107 mmHg (Table 6-5). The pain in the upper arm was relieved by the application of counterpressure to this region. It probably arose from the extreme distension of the veins on the medial aspect of the upper arm which occurred at the higher positive breathing pressures. Discomfort in the neck and head was generally absent at positive breathing pressures of up to 80 mmHg. At a positive pressure of 107 mmHg about one fifth of the subjects had severe neck discomfort when the standard pressure helmet was employed. Subsequent

experiments in which a pressure helmet fitted with a lengthened neck bladder was used showed that the incidence of this pain could be reduced to an insignificant level by the application of adequate counterpressure to the neck. This increased comfort during pressure breathing was, however, associated with a reduction of head mobility. Although it was possible to reduce some of the head discomfort induced by the pressure helmet at the highest positive breathing pressure investigated, many subjects experienced discomfort under these conditions.

The reduction of the effective blood volume associated with pressure breathing for the time which did not give rise to syncope in moderately experienced subjects, has been calculated for each of the conditions in which the standard jerkin was used (Table 6–15). It was also possible to estimate the reduction of effective blood volume which occurred when the arm jerkin and anti-g suit were used to apply counterpressure to the body. The bladders in the sleeves of the arm jerkin cover about 75"₀ of the upper limbs above the wrists, and no counterpressure was applied to the hands. The results of these calculations, which are only approximate, suggest that the reduction of the effective blood volume induced by pressure breathing for the periods given in Table 6–15 did not exceed 400 ml except when the anti-g suit was not worn. It is of interest that the reductions of effective blood volume produced by the various combinations of positive breathing pressure and duration of exposure which were found to be acceptable for moderately experienced subjects when counterpressure was applied to the trunk and either the lower or all four limbs were very similar. This finding lends support to the concept that the magnitude of the reduction of the effective blood volume may be used as an indication of the degree of stress imposed by a given exposure to pressure breathing. Wallace and Sharpey-Schafer 1941 (281) found that the incidence of syncope when 400 ml of blood was withdrawn from resting subjects was less than 2"₀. Thus the proportion of subjects exhibiting a collapse during pressure breathing with counterpressure to the trunk and the lower limbs within the limits given in Table 6–15 would be expected to be very low.

The experimental results and calculations presented in Table 6–15 demonstrate the marked reduction in the cardiovascular stress during pressure breathing associated with the application of counterpressure to the lower limbs by means of the anti-g suit. In practice, no additional disadvantages accrue from the use of the anti-g suit in this manner since this garment must be worn in order to gain protection against the effects of positive accelerations. When the pressure jerkin is used alone at a positive breathing pressure of 80 mmHg the reduction of effective blood volume, which is considerable after two minutes, reaches 700 ml during a four minute exposure (Fig. 6–30). Thus at positive breathing pressures above 50 mmHg counterpressure should be applied to the lower limbs by means of an anti-g suit as well as to the trunk by means of the pressure jerkin. The application of counterpressure to the upper limbs by the bladders of the arm jerkin reduced even further the cardiovascular stress induced by a given positive breathing pressure. The bladders in the sleeves of the arm jerkin do, however, restrict movement when they are inflated so that there are practical disadvantages to the use of this garment. The experimental results showed that the combination of standard

TABLE 6-15

THE DURATION OF PROTECTION AFFORDED BY VARIOUS PRESSURE
CLOTHING ASSEMBLIES AGAINST SYNCOPE DURING PRESSURE
BREATHING IN MODERATELY EXPERIENCED SUBJECTS

Pressure clothing assembly	Positive breathing pressure (mmHg)	Duration of pressure breathing without syncope (min.)	Reduction of effective blood volume (ml)
Helmet Jerkin	80	2	540
Helmet	80	5	380
Jerkin	107	3	380
Anti-g suit	133	2	400
Helmet Arm jerkin	110	6	390
Anti-g suit	140 decaying to 0 in 5 min.		240[1]

[1] Maximum reduction

jerkin and anti-g suit provided adequate counterpressure at positive breathing pressures of up to 107 mmHg for exposures lasting for three minutes. Above this breathing pressure, however, the incidence of severe arm pain was high (Table 6-5) and the time for which pressure breathing could be performed with this combination was reduced to a value which was of little practical use. Thus counterpressure must be applied to the upper limbs as well as the trunk and lower limbs at positive breathing pressures above 107 mmHg.

Effects of Hypoxia upon the Cardiovascular Responses – Since the reduction of effective blood volume and the intensity of the subjective disturbances induced by pressure breathing increased as the positive breathing pressure rose it was clearly desirable that the positive pressure used at a given altitude should be the minimum which could be accepted. This minimum is set by the maximum degree of hypoxia which could be tolerated following the induction of pressure breathing at high altitude. Two considerations determine the maximum degree of hypoxia which can be allowed in this situation. The first is the influence of hypoxia upon the cardiovascular responses to pressure breathing. The second consideration is the effect of hypoxia upon the higher functions of the central nervous system and the degradation of performance which ensues. The former can be determined by measuring the time of incidence of syncope and the latter by performance tests.

The cardiovascular effects of hypoxia during pressure breathing were studied in subjects breathing air at reduced environmental pressure. This method was used since it simplified the experimental procedure and allowed immediate treatment to be given in the event of a collapse since a medical officer accompanied the subject in the decompression chamber. It was assumed that changes of absolute intrapulmonary pressure per se had no specific effect upon the tolerance to pressure breathing and that any effects found with a reduction of absolute intrapulmonary pressure were due to the associated reduction of the alveolar oxygen tension. It was known that breathing oxygen at an environmental pressure of 117 mmHg (equivalent to an altitude of 44 000 ft) which resulted in an alveolar oxygen tension of 35-40 mmHg, rapidly produced a detectable impairment of performance. It was considered, therefore, that this absolute intrapulmonary pressure was probably the lowest which would be acceptable during pressure breathing of oxygen at high altitude. Calculations suggested that the absolute intrapulmonary pressure which would result in a similar value of the alveolar oxygen tension when air was breathed was 380 mmHg. Since the effects of hypoxia could be determined with greater safety at this absolute pressure, hypoxia was induced by the subject breathing air at an absolute pressure of 380 mmHg (equivalent to an altitude of 18 000 ft). In order to allow an assessment of the effects of a more profound degree of hypoxia an intrapulmonary of 349 mmHg absolute (equivalent to an altitude of 20 000 ft) was also used as a few preliminary experiments had shown that pressure breathing with an intrapulmonary pressure of 321 mmHg (equivalent to an altitude of 22 000 ft) regularly produced unconsciousness. The alveolar gas samples obtained during pressure breathing with air at reduced environmental pressure (Fig. 6-25) showed that with an intrapulmonary pressure of 380 mmHg absolute the alveolar oxygen tension lay between 35 and 42 mmHg and was on the

average some 2 mmHg lower when the intrapulmonary pressure was 349 mmHg absolute. The relatively higher alveolar oxygen tensions which were obtained at a pressure of 349 mmHg absolute were due to the greater degree of hyperventilation induced at this pressure and the consequent lowering of the alveolar carbon dioxide tension.

The control series of experiments confirmed that pressure breathing without hypoxia at a positive pressure of 52 mmHg for two minutes with trunk counterpressure does not induce any serious cardiovascular stress (Fig. 6-24). The degrees of hypoxia used in these studies induced a considerable tachycardia in the resting state. Thus the mean heart rate of the resting subjects was increased from 75 per minute at ground level to 108 per minute at an intrapulmonary pressure of 380 mmHg absolute. The induction of pressure breathing increased the heart rate both at ground level and when hypoxia was present. The increase of heart rate produced by pressure breathing was greater when the alveolar oxygen tension was normal than when hypoxia was present. The final heart rate, achieved after pressure breathing for two minutes was, however, considerably greater in the hypoxic state than at ground level (Fig. 6-24). The heart rate changes suggest, therefore, that the cardiovascular stresses induced by hypoxia and pressure breathing are additive. Two types of failure occurred when pressure breathing was performed with a low alveolar oxygen tension. In one type loss of consciousness was associated with arterial hypotension and bradycardia (vasovagal syncope) whilst in the other type the subject became confused whilst his pulse remained rapid and bounding. The cerebral disfunction which occurred in the latter instances was not due to a sudden fall of cerebral blood flow as occurred in the syncopal attacks. It was produced by the low oxygen content of the arterial blood whilst the cerebral circulation was maintained.

This group of experiments demonstrated that the degree of hypoxia associated with an alveolar oxygen tension of the order of 35–40 mmHg caused syncope during pressure breathing under circumstances in which syncope did not occur when the alveolar oxygen tension was within normal limits. Anderson, Aden, Barcroft, Edholm and Manning 1946 (5) studied the effect of displacing blood into the lower limbs by means of congestion cuffs placed around the upper thighs upon the manner in which consciousness was lost when the oxygen tension in the inspired gas was progressively reduced. They found that whilst three of their thirteen subjects lost consciousness by vasovagal syncope when there was no displacement of blood into the lower limbs, ten of the subjects developed vasovagal syncope when hypoxia was induced after the application of congestion cuffs to the thighs. The application of congestion cuffs to the thighs did not of itself induce vasovagal syncope in these subjects. These experiments showed that vasovagal syncope is much more likely to occur when a reduction of the effective blood volume is combined with hypoxia than when hypoxia is absent. This is a similar effect to that found during pressure breathing. The manner in which hypoxia renders a subject more liable to vasovagal syncope during pressure breathing is uncertain. The hypoxia might act peripherally, in that it accentuated the circulatory disturbances induced by the raised intrapulmonary pressure. Thus the volume of blood displaced into the limbs or the filtration of fluid from the vessels into the tissues of the limbs might be increased by hypoxia.

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The increased susceptibility to syncope could, however, be due to central effects in that hypoxia might render the central nervous mechanism underlying vasovagal syncope more sensitive to the peripheral circulatory disturbances induced by pressure breathing.

The possibility that hypoxia of the degree associated with an alveolar oxygen tension of 35–40 mmHg might affect the distensibility of the peripheral capacity vessels was investigated in a small series of experiments (Table 6–6). It was found that, at least in the forearm, hypoxia did not increase the volume of blood displaced into the limb by a given positive breathing pressure. Confirmatory evidence that hypoxia does not affect the distribution of blood within the circulation is given by the results of the studies of the effect of hypoxia on the pulmonary circulation performed by Fritts, Odell, Harris, Braunwald and Fishman 1960 (116). They found that the central blood volume was unchanged by moderate hypoxia and that there was no movement of the centre of gravity of subjects lying on a teeter-board when hypoxia was induced. It would appear probable, therefore, that the volume of blood displaced into the limbs by a given positive breathing pressure is not affected by the presence of moderate hypoxia.

Severe anoxia has been shown to damage the walls of tissue capillaries and to render them permeable to plasma proteins so that oedema follows a period of anoxia (181). Furthermore, Maurer 1940 (206) demonstrated that severe hypoxia increases the flow of lymph from the lung. McMichael and Morris 1936 (223) studied the effect of breathing 9.5% oxygen in nitrogen upon the rate of increase of volume of the congested forearm. They found, however, that the degree of hypoxia induced by this gas mixture did not change the rate of fluid filtration in this region. The effect of acute anoxia upon capillary permeability in man was investigated by Henry, Goodman and Meehan 1947 (147) who measured the concentration of plasma protein in venous blood from a congested arm and compared it with the protein concentration in blood from a control arm both at sea level and whilst the subject breathed air at a simulated altitude of 20 000 ft. They found that when the venous oxygen tension was reduced to below 15–25 mmHg there was a significant increase in the permeability of the capillary walls to protein. The oxygen tension in the venous blood from the control arm did not reach this low level during air breathing at 20 000 ft. The lower tension in the venous blood from the experimental arm was produced by the congestion and consequent reduction of local blood flow. It is unlikely, however, that the venous oxygen tension fell as low as this level during pressure breathing with air at an intrapulmonary pressure of 380 mmHg absolute. The rate of filtration of fluid into the tissues of the limbs was probably not increased by the level of hypoxia used in the pressure breathing experiments.

It would appear improbable, therefore, that the hypoxia associated with an alveolar oxygen tension of 35 to 40 mmHg either increases distensibility of the peripheral capacity vessels or the rate of fluid filtration during pressure breathing. The experimental studies of the amount of blood displaced in the forearm by pressure breathing were, however, very limited in that only three subjects were investigated and no measurements were made before or during a syncopal attack. Although the possibility that hypoxia increases the likelihood of a syncopal attack during pressure breathing by increasing the volume

of fluid displaced into the unpressurized regions cannot be completely excluded, the evidence available suggests that this mechanism is improbable. It may be concluded tentatively that hypoxia acts by increasing the sensitivity of the central nervous mechanism responsible for the vascular and endocrine changes which underly vasovagal syncope to the circulatory disturbances induced by pressure breathing.

Acceptable Degree of Hypoxia during Pressure Breathing – The experiments discussed in the previous paragraphs showed conclusively that hypoxia of the degree associated with an alveolar oxygen tension of 35 to 40 mmHg increased the possibility of syncope occurring during pressure breathing. They also confirmed that, in a proportion of subjects, even a short duration exposure to this degree of hypoxia gave rise to mental confusion. It was concluded, therefore, that the absolute intrapulmonary pressure during pressure breathing with oxygen at high altitude must be considerably greater than 117 mmHg since the alveolar oxygen tension at this intrapulmonary pressure would be of the order of 40 mmHg unless gross hyperventilation occurred. On the other hand oxygen can be breathed at an absolute pressure of 141 mmHg (equivalent to an altitude of 40 000 ft) with virtually no impairment of performance. It appeared probable that an absolute intrapulmonary pressure of 141 mmHg was the lowest which could be used during pressure breathing with oxygen at high altitude without untoward effects arising due to hypoxia. Furthermore, this absolute pressure had been used in other high altitude partial pressure assemblies such as the capstan partial pressure suit (162) (157). The pressure demand oxygen regulator (Mark 18) used in the initial assessment of the headpiece and pressure jerkin assemblies in the decompression chamber delivered a slightly higher absolute pressure, 145 mmHg (Table 6-8).

The subjects used in these experiments, each of whom had had an extensive experience of hypoxia, did not detect any impairment of consciousness. The simple tests of performance, such as the ability to perform mental arithmetic which were used at this time, showed no impairment during the exposures to pressure altitudes of 60 000 and 70 000 ft. In addition, none of the experienced subjects used in these preliminary studies developed any symptoms or signs of syncope whilst pressure breathing at pressure altitudes above 40 000 ft. It was decided, therefore, that the intrapulmonary pressure used with assemblies incorporating a headpiece should not be less than 141–145 mmHg absolute. For various technical reasons the pressure demand regulator (Mark 20) developed for use in the Royal Air Force maintains an absolute pressure of the order of 155 mmHg at altitudes above 38 000 ft. The subsequent experience gained in the training of aircrew using this regulator in the use of either the standard jerkin or the arm jerkin in combination with a headpiece and anti-g suit showed that no impairment of performance occurs due to hypoxia during pressure breathing when intrapulmonary pressure is of the order of 155 mmHg absolute (Table 6-8). There was a small incidence of syncope during the exposure of these aircrew to a simulated altitude of 60 000 ft in the jerkin, headpiece, anti-g suit combination (Table 6-9), but in both instances the subjects exhibited gross hyperventilation whilst pressure breathing.

The experiment described in Chapter 3 had shown that when an oronasal

mask was used during pressure breathing the maximum positive pressure which could be tolerated lay between 60 and 65 mmHg. Thus an assembly emloying an oronasal mask could only be used to an altitude of 52 000 ft if the intrapulmonary pressure was not to be reduced below 141 mmHg absolute. The acceptability of a slightly greater degree of hypoxia when an oronasal mask was used with a pressure jerkin was investigated. In the first group of experiments of this series twenty subjects were exposed whilst wearing an oronasal mask and a pressure jerkin to a positive breathing pressure of 52 mmHg at a pressure altitude of 55 000 ft (an environmental pressure of 69 mmHg absolute) which gave an intrapulmonary pressure of 121 mmHg. The hypoxia associated with this intrapulmonary pressure resulted in mental confusion in five out of the twenty subjects during an exposure lasting one minute, although none of the subjects exhibited any symptoms or signs of pressure breathing syncope (Table 6–9). The results of the alveolar gas sampling given in Chapter 5 showed that in these exposures the alveolar oxygen tension lay between 50 and 55 mmHg and the alveolar carbon dioxide tension between 18 and 22 mmHg. Thus, although the alveolar oxygen tension was considerably greater than that which was found to induce syncope and confusion during pressure breathing with air, it was accompanied by a more profound degree of hypocapnia as compared with that which occurred in the air breathing experiments.

A further small group of experiments were performed with the oronasal mask and pressure jerkin in which the positive breathing pressure during the exposure to a pressure altitude of 55 000 ft was increased from 52 to 60 mmHg (Table 6–8). One of the five subjects used in these experiments developed a typical syncopal attack after one minute at 55 000 ft, although none of the remaining subjects had any subjective disturbance of consciousness (Table 6–9). This result suggested that, whilst the increase of intrapulmonary pressure might be adequate to prevent impairment of performance due to hypoxia during an exposure lasting one minute, the increase of the positive breathing pressure might be sufficient to induce pressure breathing syncope in the presence of this degree of hypoxia. It had been shown (Table 6–3) that syncope did not occur when subjects were exposed to a positive breathing pressure of 60 mmHg at ground level. In these latter experiments, however, counterpressure was also applied to the lower limbs by means of the anti-g suit. It was decided, therefore, that the decrease of the effective blood volume imposed by this positive breathing pressure at high altitude should be reduced by the use of an anti-g suit.

A more extensive group of experiments were performed in which pressure breathing was carried out with an oronasal mask and counterpressure was applied by means of the pressure jerkin and anti-g suit. A positive breathing pressure of 60–62 mmHg was used at a simulated altitude of 56 000 ft (an environmental pressure of 66 mmHg absolute). Since the previous experiments had shown that a significant impairment of consciousness could occur during pressure breathing at a positive pressure of 60 mmHg with a similar degree of hypoxia, the duration of the exposure to 56 000 ft was reduced from one minute to half a minute. The subsequent rate of descent to a pressure-altitude of 40 000 ft was, however, kept at 10 000 ft per minute. All the twenty-two subjects who underwent this exposure did so without any serious

disturbance of consciousness. The performance of each subject was assessed during pressure breathing by means of a tracking task. This test presented the subject with a task which was similar to that required of a pilot carrying out instrument flying (35). Immediately after decompression to 56 000 ft there was a small but significant impairment of performance at the task (32). The level of performance had, however, returned to the control level before the thirty seconds exposure to 56 000 ft was completed. This transient impairment of performance was probably caused by the disturbances associated with the sudden inflation of the pressure clothing and the difficulty in seeing the task instrument owing to the formation of a mist of water vapour within the decompression chamber. This mist formed at the instant of decompression and cleared during the subsequent fifteen seconds. Since the performance at the task had returned to the control level before descent was commenced it was concluded that the hypoxia associated with an intrapulmonary pressure of 126 to 128 mmHg absolute was within acceptable limits.

The conclusions drawn from this group of experiments using the mask/ jerkin/anti-g suit combination were confirmed by the results of training one hundred aircrew in the use of this assembly (Table 6–8). Only one of these subjects developed pressure breathing syncope. The time for which these subjects were exposed to a pressure-altitude of 56 000 ft was, however, relatively short, being only thirty seconds, although each subject was exposed to pressure breathing for a total time of two minutes. The effect of prolonging the duration of the exposure to a pressure-altitude of 56 000 ft to six minutes was investigated by Ernsting, Green, Nagle and Wagner 1960 (94). Two of their five subjects developed symptoms and signs of syncope about two and three-quarter minutes after the rapid decompression to 56 000 ft. All five subjects had previously completed a six-minute exposure to pressure breathing in the same equipment at ground level at a positive pressure of 60 mmHg without any suggestion of syncope. In an attempt to determine the relative parts played by the hypoxia and the disturbance associated with the use of an oronasal mask in the genesis of the syncopal attacks, each subject was also exposed to 56 000 ft for six minutes at the same intrapulmonary pressure whilst wearing a pressure headpiece, pressure jerkin and anti-g suit. None of the subjects developed any of the features of pressure breathing syncope during this latter exposure and the mean heart rates were consistently lower than the mean heart rates when the exposure was performed with an oronasal mask. The performance of the subjects at a tracking task was unimpaired after the first minute of the exposure to pressure breathing with a pressure headpiece at 56 000 ft. Furthermore, no impairment of performance was found in those subjects who successfully completed the full six minute exposure to 56 000 ft whilst using an oronasal mask.

These results showed that the hypoxia produced by pressure breathing at 56 000 ft with an intrapulmonary pressure of 126 mmHg absolute did not significantly impair performance even when the exposure was extended to six minutes. The occurrence of syncopal attacks during pressure breathing at the same positive pressure and absolute intrapulmonary pressure when an oronasal mask was used in place of a pressure headpiece was probably due to the summation of the effects of hypoxia with the cardiovascular disturbances and discomforts produced by pressure breathing with an oronasal mask. This

study confirmed that the length of time for which the mask, jerkin and anti-g suit combination, using a positive pressure of 60 mmHg, would protect against hypoxia at a pressure-altitude of 56000 ft was limited. When the descent from 56000 ft was commenced thirty seconds after the rapid decompression and the subsequent rate of descent to 40000 ft was 10000 ft per minute, syncope did not occur.

These experiments demonstrated that the degree of hypoxia associated with an intrapulmonary pressure of 126 to 128 mmHg absolute did not cause a significant impairment of performance during pressure breathing with oxygen at a positive pressure of 60 mmHg, provided that syncope did not occur. Even this degree of hypoxia did, however, increase the likelihood of syncope when pressure breathing was combined with additional stresses, such as the neck discomfort which occurred when an oronasal mask was used. Unless the duration of the exposure to pressure breathing under such additional stresses was severely limited the use of an intrapulmonary pressure of 126 to 128 mmHg resulted in a significant incidence of pressure breathing syncope. The effects of intrapulmonary pressures between 128 and 141 mmHg absolute upon the incidence of pressure breathing syncope were not investigated. The extensive series of exposures of both experienced and relatively inexperienced subjects of pressure breathing at pressures of up to 110 mmHg showed, however, that the use of an intrapulmonary pressure of the order of 145 to 155 mmHg absolute prevented any increase in the incidence of syncope over that associated with pressure breathing at ground level with an alveolar oxygen tension greater than 100 mmHg. When either the positive breathing pressure exceeds 60 mmHg or the time for which pressure breathing is performed at positive pressures above 50 mmHg exceeds one minute, the intrapulmonary pressure should be of the order of 141 mmHg absolute if syncope is to be avoided.

Pressure Breathing at Reduced Environmental Pressure – The pattern of the exposures to low environmental pressure used in this investigation was based primarily upon the form such an exposure would take should the pressure cabin of an aircraft fail during flight at high altitude. In flight such an exposure frequently occurs suddenly. Thus pressure breathing was initiated in the decompression chamber by a rapid decompression to the maximum pressure-altitude concerned. Since pressure breathing only provides protection against hypoxia, the pilot of an aircraft flying at high altitude would initiate descent immediately decompression occurred. Thus the exposure to the maximum altitude in these experiments was relatively short, either half or one minute and was followed by a descent at a controlled rate of either 10000 ft or 15000 ft per minute to below 40000 ft. The rate of descent used was that which could be attained by the aircraft in which the equipment was to be used.

Abdominal pain following rapid decompression was the commonest cause of failure of a subject to complete an exposure to pressure breathing in the decompression chamber (Table 6–9). The pain was generally referred to the periumbilical region or the epigastrium and always occurred immediately after the rapid decompression. It was relieved very rapidly by recompression to below a pressure-altitude of 25 000 ft. The pain arose from the effects of the expansion of gas within the gastrointestinal tract. Whilst expansion of gastro-

TABLE 6–16

THE INCIDENCE OF SYNCOPE DURING PRESSURE BREATHING AT
REDUCED ENVIRONMENTAL PRESSURE IN GROUPS EACH CONSISTING
OF 100 AIRCREW

Pressure clothing assembly	Form of exposure to reduced environmental pressure	Minimum intrapulmonary pressure (mmHg abs.)	Incidence of syncope
Mask Jerkin Anti-g suit	56000 ft for ½ min.; descent to 40000 ft at 10000 ft/min.	126–128	1%
Headpiece Jerkin Anti-g suit	60000 ft for 1 min.; descent to 40000 ft at 10000 ft min.	155	2%
Headpiece Arm jerkin Anti-g suit	60000 ft for 1 min.; descent to 40000 ft at 10000 ft/min.	155	2%

intestinal gas must have occurred in all the subjects, symptoms necessitating rapid recompression occurred only in about 3"„ of all the rapid decompressions performed in the present investigation. The incidence of severe abdominal symptoms could have been related either to the volume of gas within the gastrointestinal tract before decompression or to the site in which the gas expansion occurred, or a combination of both these factors. Thus, Bedell, Marshall, Du Bois and Harris 1956 (31) found that there were fairly large variations in the volume of gas in the abdominal cavity in normal subjects. The occurrence of severe abdominal pain following rapid decompression was, however, most probably related to the expansion of a volume of gas trapped in a loop of gut so that there was local distension of the region sufficient to give rise to the sensation of pain.

The other cause of failure during pressure breathing at low environmental pressure was syncope. As a result of the considerations presented in the previous section of this discussion, three assemblies of pressure clothing, based upon the pressure jerkin, were adopted for use in flight, and separate groups of aircrew were trained in the use of these assemblies. The incidence of syncope during pressure breathing at low environmental pressure is summarized in Table 6–16. The incidence of syncope in all three groups was very low. It may be concluded, therefore, that these assemblies of pressure clothing provide adequate protection against hypoxia within the altitude-time relationships used in these experiments.

The results of the measurements of the heart rate and arterial pressure made during certain of the exposures to reduced environmental pressure are of interest. In each of these three groups of experiments the heat rate and arterial pressure responses to pressure breathing at simulated high altitude were very similar in general form to those seen at ground level (Figs. 6-26, 6–27 and 6–28). No significant difference was found between the arterial pressure changes during pressure breathing at reduced environmental pressure and those produced by an exactly similar positive breathing pressure-time exposure at ground level with an alveolar oxygen tension greater than 100 mmHg. The results obtained with the mask, jerkin and anti-g suit assembly (Fig. 6–27) suggested that the arterial pressure response to a positive breathing pressure of 60 mmHg was not affected by a reduction of the alveolar oxygen tension from above 100 mmHg to approximately 50 mmHg. A certain degree of caution must be used, however, in accepting these conclusions since the arterial pressure measurements were made by an indirect method, the accuracy of which was probably of the same order as that of the standard sphygmomanometric technique.

In contrast to the arterial pressure responses the mean heart rates were consistently greater during pressure breathing at simulated high altitude than during an exposure to a similar positive breathing pressure-time relationship at ground level (Figs. 6-26, 6 -27 and 5 -28). The heart rates during the minute before the beginning of pressure breathing were some ten beats per minute greater at reduced environmental pressure than at ground level. The increase of heart rate produced by the induction of pressure breathing was also greater at reduced environmental pressure than at ground level. The relative tachycardia seen at reduced environmental pressure before the induction of pressure breathing was probably due to apprehension with

regard to the imminent rapid decompression. The subject was always aware that rapid decompression would follow within one to two minutes of the reduction of the pressure within the decompression chamber to a pressure-altitude of 25000 ft to 27000 ft. This tachycardia could not have been due to hypoxia as the alveolar oxygen tension at this time was of the order of 190 mmHg and the subject always breathed 100", oxygen during the ascent to this altitude. In each group of experiments this preliminary tachycardia was associated with a slightly higher mean arterial pressure than was obtained at ground level. These cardiovascular changes were typical of those which were produced by nervous apprehension. The greater increase of heart rate induced by pressure breathing at reduced environmental pressure was probably due to the addition of the effects of hypoxia, hypocapnia and nervous apprehension to the cardiovascular stresses induced by this manoeuvre.

SUMMARY

These investigations of the cardiovascular effects of pressure breathing at positive pressures of up to 130 mmHg showed that the rise of intrapleural pressure produced by this manoeuvre was transmitted to the venous and arterial parts of the circulation. At the beginning of pressure breathing the immediate increase of right atrial pressure prevented any venous return from the unpressurized regions of the limbs. Since the arterial flow into these parts continued, blood was displaced from the central reservoirs, primarily the heart and lungs, into the capacity vessels of the limbs. The accumulation of blood in the peripheral capacity vessels eventually resulted in the local venous pressure exceeding the raised right atrial pressure and the recommencement of venous return to the right side of the heart. The increase in the transmural pressures of the capillaries in the unpressurized regions resulted in the loss of circulating fluid into the extravascular space. The peripheral displacement of blood and the passage of fluid into the extravascular space reduced the effective blood volume. As the duration of an exposure to pressure breathing was prolonged this reduction of the effective blood volume increased, since the loss of circulating fluid continued at approximately the same rate for at least five minutes. The reflex responses to this reduction of effective blood volume were peripheral arteriolar and venous constriction together with tachycardia. The systemic arterial pressure was raised by an amount which varied with the degree of the lung expansion and the magnitude of the reduction of effective blood volume which were induced by pressure breathing.

When the duration of an exposure to pressure breathing was prolonged syncope occurred. These collapses had all the features of vasovagal syncope and evidence was obtained which suggested that a reduction of the effective blood volume was the primary cause of this syncope. A number of precipitating factors were also found. These included discomfort or pain, hypocapnia and hypoxia. There was no incidence of syncope when pressure breathing was performed with a headpiece and trunk counterpressure, at a positive pressure of 80 mmHg for two minutes. When either the positive breathing pressure or the duration of the exposure exceeded these values syncope began to appear. The magnitude of the reduction of the effective blood volume produced by an exposure to a given positive breathing pressure and the associated incidence of syncope were reduced by the application of counter-

pressure to the surface of the limbs. Since in practice an anti-g suit would also be worn, it was decided that counterpressure should be applied to the lower limbs with this garment whenever the positive breathing pressure exceeded 50 mmHg. The use of counterpressure to the trunk and lower limbs provided adequate protection against pressure breathing syncope for exposures lasting up to three minutes at positive breathing pressures of up to 107 mmHg. At positive breathing pressures above 110 mmHg the reduction of effective blood volume together with the occurrence of severe pain in the upper arm gave rise to syncope within this time and hence counterpressure must also be applied to the upper limbs at positive breathing pressures above this level.

Detailed experimental studies showed that hypoxia increased the incidence of syncope during pressure breathing as compared with the incidence which occurred during a similar exposure to pressure breathing with an alveolar oxygen tension of the order of 100 mmHg. The incidence of syncopal attacks was increased when the alveolar oxygen tension was reduced to between 45 and 50 mmHg, particularly when this hypoxia was associated with moderate or severe hypocapnia. The experimental exposure of subjects to pressure breathing with oxygen at reduced environmental pressure showed that if the intrapulmonary pressure was greater than 145 mmHg absolute, neither impairment of performance nor syncope occurred due to hypoxia. When the intrapulmonary pressure was reduced to 126 mmHg absolute pressure breathing syncope occurred after two and a half minutes when pressure breathing was combined with the discomforts associated with the use of an oronasal mask.

Thus the time for which the maintenance of an intrapulmonary pressure of the order of 145 mmHg absolute will provide protection against hypoxia at environmental pressures of less than 145 mmHg is determined by the occurrence of syncope. Syncope in these circumstances arises because of the associated reduction of effective blood volume, the magnitude of which depends upon the positive breathing pressure and the degree of counterpressure applied to the surface of the body. Respiratory considerations demand counterpressure to the trunk at positive breathing pressures above 30 mmHg. Counterpressure to the limbs is required when the positive breathing pressure exceeds 50 mmHg or the duration of the exposure exceeds about five minutes. Finally, counterpressure must be applied to the upper limbs as well as to the trunk and lower limbs when the positive breathing pressure exceeds 107 to 110 mmHg.

GENERAL SUMMARY AND CONCLUSIONS

Although pressure breathing with oxygen was proposed by Gagge in 1941 as a means of increasing the altitude tolerance of man at heights above 40 000 ft the possibilities of this technique were not fully exploited until nearly fifteen years later. During this interval pressurization of the aircraft cabin was adopted universally as the routine method for protecting aircrew against the 40 000 ft. Thus the role of pressure breathing in aviation changed from being essential for flight at altitudes above 40 000 ft to an emergency procedure whereby consciousness was maintained following failure of the aircraft pressure cabin. The possibility of breathing at positive pressures greater than 25 to 30 mmHg was introduced by Bazett who proposed the use of a bladder to apply counterpressure to the chest and suggested the use of the anti-g suit to apply counterpressure to the lower limbs. These proposals were studied extensively by Henry and his colleagues, and as has been seen, resulted eventually in the development of the capstan partial pressure suit. Pressure breathing with an intrapulmonary pressure of the order of 141 mmHg absolute does not, however, prevent the occurrence of decompression sickness. Furthermore the capstan partial pressure suit did not provide any protection against the effects of exposure to low temperature. Thus, although a suitable garment will permit pressure breathing at high positive pressures for a considerable period of time, the time for which pressure breathing may be used following loss of pressurization at high altitude is severely limited. These considerations led to the development of the British philosophy (225) which envisaged partial pressure garments as a means of providing short duration protection against hypoxia following loss of cabin pressure at altitudes above 40 000 ft. It is especially important from the practical point of view to limit the amount of special clothing to be worn by aircrew, especially pilots of interceptor aircraft. The realization of the severe restriction of the time for which pressure breathing with partial pressure suits could be used led to the proposal that counterpressure should be applied to as small an area of the surface of the body as was compatible with adequate protection against the effects of pressure breathing.

The aim of the work described in this thesis was to determine the degree of counterpressure which must be applied to the surface of the body in order that the physiological disturbances induced by the positive breathing pressure required at a given altitude were reduced to within acceptable limits. These limits were considered to be those which would allow 95% or more of the normal aircrew population to initiate and control the descent of an aircraft following loss of cabin pressure at high altitude. These manoeuvres require a moderate degree of skill and thus it was inferred that the pressure breathing equipment must prevent any impairment of consciousness at high altitude. Since some of the disturbances induced by high pressure breathing, particularly those in the cardiovascular system, increase with the duration of the

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exposure, it was considered important to define the time for which aircrew would be exposed to pressure breathing following depressurization of the cabin. The time required to initiate a controlled rapid descent in the type of aircraft for which this equipment was required was of the order of ten to fifteen seconds. It was considered, therefore, that the partial pressure equipment should be designed to allow a period of up to one minute for the initiation of descent. Since the aircraft concerned were capable of rates of descent considerably in excess of 10 000 ft/min. the estimation of the duration of the exposure to altitudes above 40 000 ft were based upon this descent rate.

The need for respiratory counterpressure – Previous experience of pressure breathing with an oronasal mask without counterpressure to the remainder of the body had shown that even a positive breathing pressure of 30 mmHg induced fatigue and that higher positive breathing pressures were poorly tolerated. In early experiments in the present investigation counterpressure was applied to the thorax by means of a bladder which was connected to the regulator delivery tube upstream of the pressure headpiece. Although this counterpressure reduced some of the lung distension normally induced by pressure breathing and reduced the conscious effort associated with pressure breathing at positive pressures of up to 40 mmHg it was inadequate at higher positive pressures. It was found that this inadequacy was due to the absence of support to the abdomen and the consequent descent of the diaphragm. Although the addition of counterpressure to part of the abdomen by means of the abdominal bladder of the anti-g suit reduced descent of the diaphragm, breathing remained difficult at positive pressures of the order of 80 mmHg. Subsequent experiments showed that the respiratory disturbances induced by positive breathing pressures above 40 mmHg could only be reduced to within acceptable limits by the application of counterpressure to virtually the entire surface of the trunk. The bladder garment which was developed for this purpose, the pressure jerkin, became the basic component of all the partial pressure assemblies subsequently introduced into the Royal Air Force.

Respiratory disturbances induced by pressure breathing – Although the pressure jerkin applied counterpressure to the whole surface of the trunk, pressure breathing with this garment produced certain disturbances of respiration. The increase in the functional residual capacity caused by pressure breathing was limited to about 500 ml at a positive pressure of 80 mmHg and at this breathing pressure the total lung capacity was only increased by 400 ml. A significant fraction of this increase of lung volume was due to a reduction in the volume of blood contained within the trunk. Pressure breathing with this degree of respiratory counterpressure caused an increase of pulmonary ventilation which was associated with alveolar hyperventilation and hypocapnia. The intensity of the hypocapnia varied with the magnitude of the positive breathing pressure and the duration of the exposure. The alveolar carbon dioxide tension did not, however, fall below 30 mmHg even at a positive breathing pressure of 80 mmHg at ground level. This degree of hypocapnia would not be expected to cause any significant impairment of performance and subsequent experiments showed that it did not affect the cardiovascular responses to high positive breathing pressures. Studies of the compliance of the lungs, of the resistance of the airways and of the work

performed upon the lungs demonstrated that there were minimal changes in the mechanics of respiration when trunk counterpressure was applied by means of the pressure jerkin during pressure breathing.

Detailed investigations of the gaseous exchange between the alveolar gas and the pulmonary capillary blood showed that during pressure breathing with trunk counterpressure the alveolar dead space was increased. Since there was no significant increase in the unevenness of the distribution of inspired gas within the lungs this increase of alveolar dead space was probably due to a disturbance of the distribution of the pulmonary capillary blood flow. It was found that the decrease of the apparent diffusing capacity produced by pressure breathing was due to a reduction of the pulmonary capillary blood volume. Both these changes were probably the result of the displacement of blood from within the thorax which was produced by pressure breathing. These changes did not, however, produce a detectable impairment of the overall function of the lung since the alveolar-arterial oxygen tension gradient during pressure breathing at a pressure-altitude of 55000 ft was within normal limits. The failure to demonstrate any significant change in the overall transfer of oxygen from the alveolar gas to the pulmonary capillary blood was probably due to the studies being performed in otherwise resting subjects. It is probable that the marked reduction of the pulmonary capillary blood volume produced by a positive breathing pressure of 80 mmHg would result in a detectable impairment of pulmonary gas exchange in an exercising subject. Thus extensive investigations should be performed before a pressure breathing system which has been shown to maintain adequate oxygenation in the resting subject is approved for use under conditions in which the subject performs vigorous muscular exercise.

Disturbances in the neck and head – Although the use of a pressure headpiece reduced the disturbance induced in the head and neck by pressure breathing, such equipment has obvious shortcomings in practical aviation. The limitations to the use of an oronasal mask for the delivery of positive pressures to the respiratory tract were found to be related primarily to the consequent distension of the mouth and pharynx. This distension gave rise to discomfort which, at positive breathing pressures above 65 mmHg, became frank pain. This experimental study showed that when an oronasal mask was used the maximum acceptable positive pressure was of the order of 65 mmHg. Even at this pressure, there was some evidence that the use of an oronasal mask in place of a pressure headpiece increased the incidence of syncopal attacks when the duration of the exposure was prolonged beyond three minutes. The absence of support to the floor of the mouth and neck during pressure breathing with an oronasal mask induced a large increase in the volume of the respiratory dead space. The increase of pulmonary ventilation produced by pressure breathing in these conditions, however, more than compensated for the increase of dead space and hypocapnia resulted. During pressure breathing at ground level there was usually no rise of pressure in the middle ear. Pressure breathing following a sudden decompression to high altitude, however, produced a considerable rise of middle ear pressure which resulted in an easily detectable, but acceptable, reduction of auditory acuity. The vessels of the conjunctivae, which are only poorly supported, were dilated by positive breathing pressures of the order of 60 mmHg although

actual rupture of a conjunctival vessel occurred only very rarely. Capillary rupture occurred, however, in the skin of the external auditory meatus at positive breathing pressures of the order of 80 to 100 mmHg.

The R.A.F. type of partial pressure headpiece used in this study did not cause a rise of pressure in the external auditory meatus. Although the use of an oronasal mask to deliver oxygen under pressure to the respiratory tract induced certain disturbances, it was concluded that these were within acceptable limits at positive pressures of up to 65 mmHg provided that the duration of the exposure was limited to four minutes. Subsequent experience gained in the training of 500 aircrew in the use of a pressure breathing assembly employing an oronasal mask to deliver positive pressures of up to 65 mmHg confirmed that the use of an oronasal mask under these circumstances was acceptable to over 97"ₒ of the subjects (Aikman, personal communication). When positive breathing pressures above 65 mmHg are required, some form of pressure headpiece which provides support to the greater part of the face and neck must be employed.

Cardiovascular disturbances – Measurements of the pressure in the right atrium showed that when pressure breathing was commenced there was an immediate rise of central venous pressure. The venous return from the un-pressurized regions of the body, in particular the limbs, ceased until sufficient blood had accumulated in the capacity vessels of these parts to raise the local venous pressure above the central venous pressure. Thus at the beginning of pressure breathing blood was displaced from within the thorax into the limbs. Circulating fluid was also lost into the extravascular spaces of these regions because of the increase in the capillary transmural pressure associated with the general rise of intravascular pressure. Thus pressure breathing, particularly when trunk counterpressure is employed, directly affects the effective circulatory blood volume and is a manoeuvre by which the effects of changes in the distribution of the blood volume within the body may be studied. The reduction in the effective pressure in the right atrium produced by pressure breathing was associated with a fall in the cardiac output although the heart rate was increased. When no counterpressure was applied to the limbs there was a fall in the effective systemic arterial pressure. The reduction of the effective blood volume was associated with reflex peripheral arteriolar and venous constriction.

As the positive breathing pressure was raised above 50 mmHg the factor which determined the maximum duration of a given exposure to pressure breathing with a pressure headpiece and trunk counterpressure was the incidence of syncope. The pressure breathing collapses had all the features of classical vasovagal syncope. The magnitude of the displacement of blood and of the loss of circulating fluid in the limbs was measured and was found to lie between 700 and 900 ml at the point at which syncope occurred in trained subjects. This reduction of effective blood volume was similar to that required to induce vasovagal syncope during a simple venesection. It was concluded that pressure breathing syncope was due primarily to a reduction of the effective blood volume. It was found that the occurrence of severe discomfort or pain during pressure breathing predisposed to syncope. Severe hypoxia or severe hypocapnia also rendered a subject more liable to syncope during pressure breathing.

Reduction of the peripheral displacement of circulating fluid normally produced by pressure breathing was shown to decrease the incidence of syncope and to increase the time for which a subject could be exposed to a given positive breathing pressure without the occurrence of a collapse. Since in practice the aircrew for whom this equipment was intended would wear anti-g suits, it was possible to apply counterpressure to the greater part of the surface of the lower limbs during pressure breathing without adding a further garment to the clothing which was required. Thus the counterpressure applied to the body during pressure breathing at positive pressures above 30 mmHg was extended to include most of the lower limbs as well as the trunk. It was important, however, to retain freedom of movement of the upper limbs during pressure breathing, but all the available methods of applying counterpressure to these regions produced some restriction of mobility. It was found that the limit to the positive breathing pressure which could be tolerated with counterpressure applied to the head, trunk and lower limbs, was determined by the occurence of discomfort and pain in the upper arm. Thus at positive pressures above 80 mmHg some subjects had pain in the upper arms and at positive breathing pressures above 110 mmHg there was a high incidence of severe pain. In certain subjects arm pain was associated with syncope during pressure breathing and at positive pressures above 110 mmHg pain from this region was a common cause of collapse. The maximum positive breathing pressure which could be employed for an exposure lasting three minutes without a significant incidence of collapse due to the absence of counterpressure to the upper limbs was 107 mmHg. The application of counterpressure to the upper arm and forearm prevented pain in this region. Thus at positive breathing pressures greater than 107 mmHg upper limb counterpressure was necessary in addition to counterpressure to the trunk and lower limbs.

Hypoxia and pressure breathing – Since the incidence of vasovagal syncope induced by congestion of the lower limbs had been shown to be increased by hopoxia (5), it appeared probable that hypoxia would increase the incidence of syncope during pressure breathing. It was found that with pressure breathing at pressures of 50 to 60 mmHg the incidence of syncope was increased if the alveolar oxygen tension was below 55 mmHg but not if the oxygen tension was greater than 55 mmHg. Furthermore, above this critical value there was no detectable impairment of performance. Measurements of the gas tensions in the alveolar gas and arterial blood showed that an absolute intrapulmonary pressure of 141 mmHg maintained an alveolar oxygen tension of 60 to 70 mmHg during pressure breathing with oxygen at altitudes above 40000 ft. It was decided, therefore, that the intrapulmonary pressure during pressure breathing with assemblies incorporating a pressure headpiece should not be less than 141 mmHg absolute at altitudes above 40000 ft. Thus since the maximum positive breathing pressure which was found to be acceptable when no counterpressure was applied to the upper limbs was 107 mmHg the maximum altitude to which the combination of pressure headpiece, pressure jerkin and anti-g suit could be used was 70000 ft (environmental pressure at 70000 ft is 34 mmHg plus a breathing pressure of 107 mmHg provides an intrapulmonary pressure of 141 mmHg absolute). In practice the oxygen regulators developed for use with this assembly

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provided an intrapulmonary pressure of 155 mmHg absolute, so that the maximum altitude to which it could be used was reduced to 66 000 ft.

The use of an intrapulmonary pressure of 141 mmHg absolute with an assembly based upon an oronasal mask would limit the maximum altitude to which this combination could be used to 53 000 ft (environmental pressure at 53 000 ft is 76 mmHg plus a breathing pressure of 65 mmHg providing an intrapulmonary pressure of 141 mmHg absolute). Since there was a well defined practical need for such an assembly to provide short duration protection against exposure to altitudes of up to 56 000 ft (environmental pressure = 66 mmHg) the influence of a slight degree of hypoxia upon the responses to pressure breathing was investigated. Provided that the total duration of the exposure to pressure breathing was limited to two minutes and that counterpressure was applied to the lower limbs as well as to the trunk, there was no impairment of performance when the intrapulmonary pressure was reduced to 126 mmHg absolute. This lower limb counterpressure was essential since it was found that the disturbances produced by the use of an oronasal mask at a positive breathing pressure of 60 mmHg induced syncope when a mild degree of hypoxia was induced in the absence of counterpressure to the lower limbs. An assembly consisting of an oronasal mask, pressure jerkin and anti-g suit was shown to provide adequate protection against hypoxia at altitudes of up to 56000 ft provided that descent was commenced within half a minute of the beginning of the exposure and that an altitude of 40000 ft was reached within a further one and a half minutes.

Concept of minimal counterpressure – As was demonstrated by Haldane in 1933, the hypoxia which ensues on exposure to altitudes greater than 40000 ft even when oxygen is breathed, can be prevented without any physiological disturbance by enveloping the entire individual within a gastight suit which is inflated with oxygen to an absolute pressure of at least 141 mmHg. The experimental studies described in this thesis showed that the high positive breathing pressures required to prevent hypoxia at altitudes between 50000 and 70000 ft are well tolerated over a period of several minutes even when counterpressure is applied to only a portion of the surface of the body. The minimum degree of body counterpressure which was found acceptable in relation to the time for which the subject was exposed to altitudes above 40000 ft was that provided by the pressure jerkin and anti-g suit. The three assemblies which have been introduced into the Royal Air Force have been based upon this combination of pressure garments (Table 7–1). No decompression sickness other than an occasional mild bend was experienced by the subjects who were exposed to pressure breathing at simulated altitudes of between 50000 ft and 100000 ft even when no preliminary breathing of oxygen was performed. Two of the subjects used most frequently in these investigations were, in fact, considerably more susceptible to decompression sickness than the great majority of aircrew. Neither of these subjects experienced any serious decompression sickness during short duration exposures to high altitude, although both developed severe bends after a ten minute exposure to a pressure altitude of 37000 ft. These observations lend strong support to the hypothesis that the incidence of serious decompression sickness during and following an exposure to an altitude greater than 30000 ft is negligible provided that the duration of the exposure is less than ten minutes.

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TABLE 7-1

PARTIAL PRESSURE ASSEMBLIES IN USE IN THE ROYAL AIR FORCE

Assembly	Oxygen Regulator (Mk)	Limits of protection
Headpiece Pressure Jerkin Anti-g suit	20	66000 ft for 1 min.: followed by descent to 40000 ft at 10000 ft/min.
Headpiece Arm Jerkin Anti-g suit	20	100000 ft for 1 min.: followed by descent to 40000 ft at 15000 ft/min.
Oronasal mask Pressure Jerkin Anti-g suit	21	56000 ft for $\frac{1}{2}$ min.: followed by descent to 40000 ft at 10000 ft/min.

TABLE 7-2

RESULTS OF TRAINING R.A.F. AIRCREW IN PARTIAL PRESSURE ASSEMBLIES

Assembly	Number of aircrew trained	Proportion developing syncope (%)
Oronasal mask Pressure Jerkin Anti-g suit	650	2.8
Headpiece Pressure Jerkin Anti-g suit	130	3.3

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Although from the point of view of practical aviation the advantages of reducing the area of the surface of the body covered by pressure clothing to the smallest possible value are considerable, the application of the concept of minimum counterpressure must introduce a degree of uncertainty with regard to the protection afforded against the effects of exposure to high altitude. The values of the heart rate recorded during the exposure of subjects to reduced environmental pressures in the decompression chamber suggested that pressure breathing at high altitude using various counterpressure assemblies places a certain degree of stress upon the cardiovascular system. In these exposures to high altitude the effective blood volume was decreased by 300 to 400 ml and this disturbance would be expected to lead to vasovagal fainting in a small proportion of subjects. Indeed, five cases of syncope occurred during the exposure of 300 aircrew to pressure breathing at simulated altitudes of either 56000 ft or 60000 ft (Table 6–16). Subsequent experience in the training of larger numbers of aircrew (Aikman, personal communication), the results of which are presented in Table 7–2, has confirmed that the incidence of syncope during such exposures is very low. It is impossible, however, to infer with certainty the effects of the circumstances surrounding an actual loss of cabin pressure during flight at high altitude upon the physiological responses to high pressure breathing. Such a decompression generally occurs without any preliminary warning and thus may well produce the mental and physiological disturbances associated with any sudden physical event.

The magnitude of these changes will be influenced greatly by the previous experience of the pilot and they will vary from surprise followed by the appropriate corrective procedure, to panic and perhaps loss of control of the aircraft. This situation is in distinct contrast to that which pertained in the experiments performed in the decompression chamber where the subject was always aware of when the decompression would occur. Furthermore the pilot of an aircraft will have to retain control and initiate descent during the period immediately following a decompression. In the decompression chamber, however, there was no such sense of urgency and in many experiments the subject had no specific task to perform following the decompression. In the decompression chamber experiments many of the subjects had a moderate tachycardia immediately before the decompression whilst some of them exhibited overt apprehension. These emotional disturbances probably accentuated the hyperventilation normally seen during pressure breathing and may have been a factor contributing to the incidence of syncope. In many subjects the sudden emergency arising during flight will probably produce a rise of cardiac output, a rise of arterial pressure and splanchnic vasoconstriction. These cardiovascular reactions will tend to reinforce the normal reactions to pressure breathing and hence to oppose the effects of the reduction of effective blood volume which is produced by this manoeuvre. The likelihood of syncope occurring during and following a rapid decompression during flight is very low provided that a rapid descent is initiated immediately, especially as in practice those aircrew who are particularly susceptible to pressure breathing syncope will have been excluded during training.

Limits of Partial Pressure Suits – This investigation has demonstrated, therefore, that pressure breathing with oxygen has a definite place in the

prevention of hypoxia following loss of cabin pressure at altitudes greater than 40000 ft. It is possible by this manoeuvre to prevent any serious impairment of consciousness at altitudes of up to 100000 ft. The cardiovascular disturbances which ensue when pressure breathing is performed at the high positive pressures necessary to prevent hypoxia with counterpressure applied to only part of the surface of the body imposes, however, a definite limit upon the time for which this procedure can be used. Furthermore, the absolute pressure which can be maintained in the body by this type of equipment is such that decompression sickness will occur if the exposure to high altitude is prolonged. The only satisfactory solution to the problem of providing protection against the effects of prolonged exposure to high altitude following loss of cabin pressurization is the full pressure suit.

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