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THE EFFECTS OF HIGH ALTITUDE ON MAN*

BY

B. H. C. MATTHEWS, C.B.E., Sc.D., F.R.S.

LECTURE I

Dr. Oliver founded these lectures in memory of William Sharpey "to promote physiological research by observation and experiment and to encourage the application of physiological knowledge to prevention and cure of disease and the prolongation of life."

The subject of the present lectures is the application of physiological knowledge to the saving of life and to increasing the efficiency of flying personnel. As an example, enemy fire is an obvious risk to which aircrews are often exposed, but, flying at 25,000 feet, many thousands of men are constantly exposed to other less obvious physiological dangers to life. At this height the atmosphere is lethal to men acclimatized to sea level, so that they must be protected by oxygen equipment. The intense cold is another danger, from which they are protected by special clothing and equipment. The physiological risk, anoxia, is an unseen enemy, present continually, from which aircrews must be protected not only by oxygen equipment but also by training to make them realize the nature of this danger and its effects. If they have to crash-land, the

FIG. 1.—Paul Bert's decompression chamber.

hazards of sudden deceleration can be greatly reduced by the application of physiological knowledge embodied in crash procedure and body harnesses. In tight turns the effects of centrifugal forces on the circulation can be minimized.

Research on physiological problems of flying has been carried out intensively in all Allied countries; the Germans also were early in the field, and large funds and facilities were available for the purpose from 1933 onwards. The Flying Personnel Research Committee, under the chairmanship of Sir Edward Mellanby, was set up by the Secretary of State for Air before the outbreak of the war; it initiated intensive investigations for the R.A.F. by R.A.F. medical officers and civilians, working both in the laboratory and in the field, to carry out basic

research and organize its application to aircrews. The Americans gave much help before Pearl Harbour, but since then they have provided almost unlimited facilities for research in the physiology of flight. Research in Canada and Australia has been very active, and the results are of great value. It is impossible to give credit here to the many research workers involved. Free interchange of information has taken place between the Allies, and much of the resulting success has come from team-work by men from all the Allied nations.

The field of aviation physiology is wide, and here two subjects are chosen: (1) the effects of high-altitude flying; (2) the effects of mechanical stress.

Physiological Effects of High Altitude

These can be divided into: (1) the effects of lowered partial pressure of oxygen at high altitude; (2) the effects of the lowered atmospheric pressure. The first leads to incomplete saturation of the blood with oxygen, and may indirectly disturb the carbon dioxide content of the blood. The second may lead to bubble formation within blood vessels and tissues, and also to effects following expansion of the gas present in the viscera, sinuses, middle ear, and, after chest injuries, of air within the pleura.

FIG. 2.—R.A.F. decompression chamber.

Anoxia

Anoxia is not a new problem; the effects of oxygen lack on man were encountered both on mountains and in balloon ascents before the beginning of the last century, but modern research on this subject was initiated by Paul Bert after the disaster in 1875 to the balloon "Zenith," in which Tissandier's two companions lost their lives from anoxia, although oxygen for breathing was carried in the balloon. Paul Bert (1878) built the first decompression chamber (Fig. 1), in which low-pressure air conditions could be studied on the ground, and this was followed by the work of Haldane, Barcroft, Henderson, and Flack, which, in addition to elucidating the nature of anoxia, shed much light on the normal processes of respiration. While many physiological studies have been carried out in flight, the greater part of our exact knowledge comes from studies made in decompression chambers (Fig. 2) at sea level or in moun-

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tain expeditions, as the conditions for research and experiment are not easily met with during flight. It is necessary to draw a sharp distinction between the mountaineer who has had days or weeks to acclimatize to low air pressure and the aviator who usually starts from ground level and is rarely away from it for more than 12 hours. An altitude of 28,000 feet has been reached on Mount Everest by men not using oxygen equipment; the same height may prove fatal to an aviator in about half an hour.

The accompanying table sets out the major effects of oxygen lack as they are seen in the aviator. There are three primary factors determining the intensity of symptoms: (1) the oxygen partial pressure of exposure; (2) the duration of exposure;

the fuselage. In fighter aircraft the high-altitude pilot carries in his parachute packing an emergency oxygen set which can not only be used in emergency but which goes with him to supply oxygen during descent, should he have occasion to use his parachute. Otherwise he may become unconscious while falling and fail to pull his parachute rip-cord.

Anoxia of gradual onset is extremely insidious in that the subject is usually quite unaware that anything is wrong. His behaviour appears normal to himself, but to others may be that of a drunken man. In the last war, cases of the bombing of friendly targets occurred owing to anoxia. It is for this reason that the R.A.F. have put into operation large numbers of mobile decompression chambers. In these, aircrews can

Effects of Oxygen Lack on Man

I	II	III	IV	V	VI	VII
Altitude; Oxygen p.p., mm. Hg.	Approx. Blood Saturation (%)	Effect	Time for Full Development	Unconsciousness	Death	Approx. O ₂ Percentage in Inspirate for Normality
5,000-10,000 ft. 126.5-104.5	95-90	Reduced night vision	Over 1 hour	Never	No	28%
10,000-15,000 ft. 85.8	90-78	Fatigue, headache, errors of judgment, and indifferent mental condition.	Over 1 hour	Very uncommon, and only with hard work	No	35%
15,000-18,000 ft. 77.5	80-68	More pronounced mental errors. Reduced capacity for physical work	30 min.	Incidence less than 1% if at rest	Only in pathological subjects	42%
18,000-22,000 ft. 64	70-55	Gross mental abnormality. Euphoria or drowsiness and lethargy. Loss of neuromuscular co-ordination	15 min.	Unconsciousness may occur above 20,000 ft. Dangerous, especially in single-seater aircraft	Rare, but possible with other adverse conditions (cold, etc.)	50%
22,000-27,000 ft. 51	Less than 65	Rapid loss of muscular control, often with uncontrollable twitching, especially if some hyperventilation	10-2 min.	Individual variation in time for onset of unconsciousness, 5-30 min.	Death likely at 27,000 ft. in less than 1 hour	70%
27,000-30,000 ft. 45		Rapid loss of consciousness, 1-5 min.			Death probable in 20-60 min.	80%
30,000-37,000 ft. 32		Unconsciousness in $\frac{1}{2}$ to 2 minutes			Death to be expected in less than 30 min.	90-100%
37,000-44,000 ft. 24		Breathing 100% oxygen, anoxia present. Severe at 44,000 ft.				
Over 44,000 ft.		Only safe with pressure cabin or pressure suit				

(3) the muscular activity of the subject. At heights up to 35,000 feet all symptoms are entirely prevented by breathing pure oxygen or air enriched with oxygen (Col. VII of table).

The effect of changing from oxygen to air at 35,000 feet is to produce unconsciousness in less than a minute. Respiratory failure is to be expected after about 7 to 8 minutes and death in about 10 minutes. At 25,000 feet the same change leads to a progressive development of symptoms. The first symptoms seen are cerebral; subjects are often euphoric within two minutes. Failure of the nervous and muscular co-ordination follows. The electro-encephalogram (Beigel *et al.*, 1942) parallels the clinical reaction to anoxia; slow waves are observed as the subject becomes unconscious, and spike activity during convulsive seizures. There are often intensive peripheral vasodilatation and great acceleration of the heart just before unconsciousness occurs; alternatively, some subjects show collapse of the cardiovascular system before becoming unconscious. Unconsciousness and loss of muscular tone may ensue in about 5 minutes. Death is unlikely to occur in less than 30 minutes, but there is a possibility that it may follow longer exposure.

The danger of an oxygen breakdown and death of a member of the crew in large R.A.F. aircraft is reduced by telephonic communication, maintained at regular short intervals from the captain to all his men, and very few deaths attributable to anoxia have occurred. In the U.S.A.A.F., which has had to operate under different conditions, the considerable number of deaths from this cause enables lethal exposure to be deduced to an approximation. Death is unlikely to occur below 22,000 feet and may occur in a few minutes above 27,000 feet (see Col. VI of table). A number of necropsies have been performed on those who have died from anoxia, and 27 are described in a recent paper by Kitzler (1944). These show little pathological change except a great congestion of the lungs, viscera, and, particularly, the kidneys.

A man breathing air at 30,000 feet remains conscious for little more than a minute, and it will be appreciated how rapidly aircrews must act if their oxygen equipment should fail at these altitudes. In large bomber aircraft the crew are provided with small portable oxygen sets so that they may carry out duties which necessitate walking any considerable distance in

be shown what happens if they neglect to use oxygen properly; this is done at ground level in safety under the supervision of a medical officer. This familiarization of aircrews with the proper use of their oxygen equipment probably accounts for the very low incidence of oxygen failures in the R.A.F.

Subjects vary greatly in their reaction to lack of oxygen, and these differences appear in part to be connected with the varying sensitivity of individual respiratory centres to oxygen lack. The respiratory centre and carotid sinus are primarily controlled by the carbon dioxide in the blood and, to a secondary and much more variable extent, by the oxygen tension. Some subjects placidly pass into coma and unconsciousness with increasing altitude with very little rise in the respiratory minute-volume and without muscular movement. Others show considerable hyperventilation, which often enables them to resist a greater degree of anoxia than the former class, but may then exhibit violent involuntary movements, sometimes extending to a generalized convulsion before they become unconscious. The lowering of carbon dioxide by hyperventilation in the latter class appears to be responsible for the very marked difference in their symptoms (Schmidt, 1943). When oxygen is administered to subjects who have become unconscious, unless a low flow is used at first they may pass through a phase of convulsion and twitching before returning to normality. Recovery can be very rapid, and the subject is practically unaware, a minute later, of what he has been through, provided the unconsciousness has been of short duration. Moreover, if pure oxygen is given to a subject on the verge of collapse it may produce complete collapse for some 30 seconds, followed by a very rapid recovery. While the biochemistry of this is not fully established, it probably is a reaction to the sudden change of acidity in the blood which will result from rapid conversion of reduced haemoglobin to oxyhaemoglobin (the Bohr effect).

In the last war, pilots often flew at 18,000 feet without using oxygen. Anoxia was then regarded as one of the normal stresses of flying, and pilots were required to pass an anoxia-tolerance test. In this they breathed from a closed spirometer, the carbon dioxide being absorbed and the oxygen tension falling progressively. Those who showed early pulse irregularities or fainting were rejected. This is now no longer necessary, as

anoxia is a stress which can be removed with proper oxygen equipment, and all who are fit on clinical examination are able to fly to at least 25,000 feet when using that gas. In R.A.F. aircraft the use of oxygen is compulsory above 10,000 feet, and below that at night for some aircrews. Between 10,000 and 15,000 feet there is no gross danger if not used; but experience has shown that not only is a man less mentally alert and his range of vision reduced if he does not have oxygen at these heights, but he subsequently suffers considerable fatigue if exposed to slight anoxia for several hours; this is avoided by the employment of oxygen. Moreover, without oxygen he "blacks out" more easily in a tight turn.

It is well known that prolonged oxygen-breathing at ground level may produce pulmonary oedema, and, as Paul Bert first showed, at pressures greater than atmospheric it may produce convulsions. The dangers of oxygen poisoning do not exist, as this gas is used in aviation at reduced pressures. No difficulties from this cause have arisen. Oxygen equipment for air use has to meet stringent requirements. It must be of minimum encumbrance to the wearer and require the minimum of his attention. All the Allied Air Forces use oxygen stored in cylinders, for although liquid oxygen in thermos containers can save weight, and was so employed both in the last war and subsequently by the R.A.F., its disadvantages—evaporative loss and the difficulty of controlling the flow to requirements—have rendered it less suitable than oxygen in cylinders for use in the rough-and-tumble of operational flying. The weight of the cylinders introduces the question of economy in the use of oxygen. All Air Forces employ systems in which the flow of oxygen to the mask is interrupted during expiration, as the consumption when this is done is little more than one-third of that needed with a constant flow. Apparatus must be able to supply a flow sufficient to cover the physiological requirement for any physical work that aircrews may have to undertake. A liberal rating is necessary to cover both this and the rapid ventilation that may occur during the stress of combat. Figures for ventilation rates of aircrews taken during bomber operations show an increase in areas of danger.

A man at 18,000 feet, though still conscious, becomes very sluggish mentally. He is incapable of physical exertion and may become unconscious if he attempts it. In contrast to this, men who have become acclimatized over a period of weeks are able to lead a normal life at 17,000 feet and to carry out hard physical work. There are three major changes which make this possible:

1. The red blood corpuscle count rises by up to 50% and the oxygen-combining capacity of the blood increases with this.
2. The ventilation rate progressively increases, the alkalosis resulting is compensated by the kidneys, and the acclimatized man is able to breathe rapidly, with approximately normal pH of the blood.
3. There are changes in the tissue-oxidase systems which enable cells to work normally in spite of the reduced oxygen pressure in the blood reaching them. For example, in animals the concentration of myoglobin in the diaphragm is approximately doubled with acclimatization.

On return to sea level acclimatization is lost by a reversal of these changes, occupying a fortnight or more; artificially induced acclimatization (Rühl, 1943)—e.g., by ingestion of ammonium chloride—has not been very successful.

At heights above 18,000 feet little further acclimatization appears to take place, and the experience from mountain expeditions has been that above 20,000 feet men deteriorate in physical condition, sleep very little, are capricious in their diet, and always lose weight. It may be conjectured that the chemical processes of digestion and metabolism are unable to proceed normally at the low oxygen partial pressure which occurs under these conditions. Discontinuous exposure to altitudes of 10,000 to 14,000 feet, as may occur in pilots who are not using oxygen, does not lead to any very significant acclimatization. In one study of the blood of civil airline pilots not using oxygen a fall in the red blood count was found (Clinton and Thorn, 1943).

Nausea and respiratory distress, commonly seen in mountain sickness, are much less prominent in short exposure to oxygen lack: this fact appears to be due to slowness in lowering the CO_2 by hyperventilation in the early stages of acclimatization before Compensation 2, above, has occurred, and those symptoms must be attributed to this, acting concurrently with anoxia,

recalling Mosso's early hypothesis, now discarded, that loss of CO_2 was the primary cause of mountain sickness.

Effects of Low Pressure per se

The dangers of compressed-air illness have long been recognized among under-water and caisson workers, but it is only recently that the parallel effects that may occur at pressures below atmospheric have been recognized and studied. Symptoms occurring at low pressure that could not be attributed to lack of oxygen are found in the literature of exposure to low pressure (e.g., Jungblut), but were first fully described by Armstrong (1939). Intensive research has been carried out since then to elucidate the nature of decompression sickness as it occurs at reduced atmospheric pressure.

There is a great difference in the conditions leading to decompression sickness in the diver and the aviator. The diver spends his life at the lower pressure (atmospheric) and a short time at the higher pressure when under water. Consequently nitrogen reaches and goes into solution only in certain tissues readily accessible to the circulation, and does not attain a high pressure in inaccessible tissues—for example, the optical media of the eye. In diving, the pauses during ascent necessary to allow nitrogen to escape from the lungs are a small multiple of the total time spent submerged at high pressures. In contrast, the aviator spends his life at the high pressure (atmospheric), and his exposure takes place when he ascends to great altitudes. It is just from those tissues which do not become saturated with nitrogen in diving that escape of nitrogen is most difficult in the case of the aviator. One of the commonest sites of bubble formation at reduced pressure is the synovial fluid of joints, from which gas has difficulty in escaping owing to very low vascularity, and bubbles can often be seen by x-ray examination at low pressure. The first description of decompression sickness was by Robert Boyle, who saw a bubble within the eye of a viper when he decompressed it to a pressure equivalent to that at about 50,000 feet.

In general the symptoms are less severe and of slower onset than those seen in divers. Moreover, they disappear at once on descent to 20,000 feet. There are three main factors responsible for the formation of bubbles in the blood vessels and tissues at lowered pressure: (1) the ratio of pressures, and to a lesser extent the absolute magnitude of the pressure change; (2) the rate of change: this is of great importance in diving, but the rate to avoid aerial bends is so slow that gradual ascent to avoid symptoms is impracticable; (3) local conditions in the tissues which may precipitate formation of bubbles—e.g., exercise. While the bubbles in divers are primarily composed of nitrogen, those occurring in aviators contain a high proportion of carbon dioxide.

From theory and experiments on divers the late J. S. Haldane deduced that a change of pressure in a ratio of 2:1 at least was necessary before bubble formation could take place, and this appears true at reduced pressures, as decompression sickness is never seen below 20,000 feet. Above 30,000 feet it becomes increasingly common, and the symptoms are of increasing severity. The symptoms usually develop gradually, starting with muscular and joint pain, with pain in the chest followed by sensory symptoms, and anaesthesia of limbs; itching and tingling sensations are common. These may be followed by a collapse of central origin in which the subject suddenly becomes intensely white, the pulse may slow and become imperceptible, and consciousness is lost. On immediate increase of pressure to 20,000 feet recovery takes place in a few minutes, though delayed neural reactions, of which the commonest are visual scotomata (Engel *et al.*, 1944), and, rarely, epileptiform convulsions, may appear subsequent to return to ground level, and in rare cases death has occurred after severe chest symptoms (Goggio and Houck, 1945). In these, massive hydrothorax and pulmonary oedema have been found at necropsy.

The incidence of symptoms is extremely variable in the same subject between daily exposures, and between different individuals. This is a matter of great practical importance, but intensive research has so far failed to locate the fundamental biochemical factors involved. As in divers, overweight and obese body build are unfavourable. There is evidence of symptoms increasing with age (Thorne, 1943), but some subjects seem completely immune from bends at pressures corresponding to

40,000 feet. It is at present impossible to predict liability to bends from other physical characteristics, and it has therefore been necessary to select flying personnel for very high altitude work by tests carried out in a decompression chamber with two or three exposures on different days for each subject. At present we cannot foretell how long the selection will remain reliable. Protection against bends is possible by breathing oxygen before exposure to low pressure—to displace nitrogen from the blood and replace it by oxygen, which can be used up within the body when the pressure falls. By pre-breathing oxygen for several hours a very high degree of protection against bends is obtained. This has obvious practical difficulties for flying personnel, and can be employed only on missions working to a prearranged time-table. In practice, where available to crews, it is little used, as after selection they very rarely experience decompression symptoms in the air.

In early work the central effects were attributed to raised C.S.F. pressure resulting from bubble formation; but while the escape of gases from C.S.F. is very slow—as shown by McArdle (1945) on oxygen-breathing human subjects from whom C.S.F. was withdrawn for analysis by lumbar puncture—animal experiments have not revealed any great rise in C.S.F. pressure which can be attributed to internal bubble formation. Moreover, little rise in intracranial pressure occurs in man (Peterson *et al.*, 1944). It is most probable that the central effects follow local interruption of circulation by bubbles in blood vessels in the brain. That the peripheral symptoms of pain or itching have a peripheral origin was proved by Squad. Ldr. Pask in a number of cases in which the application of pressure to the affected limb with a special pressure bag removed the symptoms.

Change of Pressure

With rapid change of pressure the sinuses and middle ear must equalize their pressure with that outside; otherwise, symptoms may result from congestion and exudation with rising pressure, or cessation of circulation with falling pressure. This is a matter of great practical importance, as the maintenance of a healthy E.N.T. condition in aircrews is necessary for them to fly without any discomfort. The subject of otitic barotrauma has received much attention in the R.A.F. (Dickson *et al.*, 1943), but sudden change of pressure has no other physiological effects, and, provided the Eustachian tubes are patent, a fall of pressure of half an atmosphere in about half a second can be tolerated by man without any discomfort. Studies on this explosive decompression were first carried out by Armstrong and Heim, and later extended by the R.A.F. It was found that even such a severe change of pressure produced no discomfort in normal subjects. On the other hand, with increasing pressure air must be admitted to the middle ear by opening the Eustachian tube, and this cannot be done rapidly enough to tolerate descent at anything like this rate. Most aircrews can clear their ears by swallowing or by jaw movements, so that they can descend at 4,000 to 10,000 feet a minute without discomfort. (Expansion of gas in the viscera is rarely a practical source of difficulty in high flying.) All personnel can operate to 25,000 feet without oxygen; selected aircrews can operate to about 40,000 feet. Above this height it is essential to apply pressure all over the body surface either by a pressure suit (Marshall, 1937) such as was employed by Adam in 1937 when he took the altitude record to 54,000 feet, or a pressurized cabin or a pressure gondola such as was used in the U.S. Army high-altitude balloon flight to 72,000 feet.

For civil use high-altitude flying has many advantages. It enables aircraft to operate fast and economically, to fly over the weather, and to fly in smooth air. It seems certain that pressure-cabin aircraft will ultimately be employed for all long-distance travel. Passengers inside will not then need to wear oxygen masks to avoid anoxia; nor would they incur the risks of decompression sickness or experience the discomforts of rapid pressure change on their ears. It therefore becomes a matter of engineering to overcome the physiological stresses of altitude to which man has been subjected since the start of flying.

It is interesting to turn to the work of Paul Bert, written in 1877, to see his recommendations on oxygen for aeronauts. "Aeronauts planning very lofty ascents ought in the preceding days to avoid nervous and intellectual fatigue. Before starting they should take meals of substantial food and take with them

some cheering victuals to eat frequently on the way. To be safe it is necessary only to breathe the air whose oxygen content rises proportionately as the pressure falls, and I cannot recommend too strongly that at 5,000 or 6,000 metres a direct and compulsory connexion be made between the oxygen bags and the mouths of the aeronauts." Much of the physiological work for the R.A.F. during this war has been directed towards putting Paul Bert's recommendations into effect.

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IRREPARABLE VESICO-VAGINAL FISTULA IN IRAQ

TREATMENT BY URETEROCOLOSTOMY

BY

DONALD M. DOUGLAS, M.B.E., M.B., Ch.M.,
M.S.Minn., F.R.C.S.

Professor of Clinical Surgery, Royal Faculty of Medicine, Baghdad,
Iraq; First Assistant in Surgery, British Postgraduate Medical
School

Thirty centuries before the birth of Christ the Sumerians, living on the banks of the River Tigris, recorded their deeds in writing, maintained an army for their own defence, and drank a "liquid closely resembling beer" (Lloyd, 1943). But in spite of this admirable beginning Iraq was but an outlying province of the decadent Ottoman Empire at the outbreak of the First Great War. She was loosely governed by corrupt officials, who were satisfied in the discharge of their duties if a sufficient revenue was extorted from the country and a sufficient quota of conscripts was obtained for the Turkish Army. The real power in the country lay in the hands of the great Kurdish and Bedouin sheikhs, to whom social reform and social services made no appeal.

To-day, a mere 20 years since the foundations of independent Iraq were laid, the structure of a universal health service is well established, its two main pillars a thriving medical school and a school of nursing. But a sad legacy of centuries of ignorance and neglect remains. Tuberculosis is rife, and in every town and village the hideous ravages of smallpox, trachoma, leishmaniasis, and venereal disease are evident. The infantile mortality in country districts is over 50%.

It is with the results of neglect in pre-natal and natal care that this paper deals. In the smaller towns and villages, and in the desert, women are attended during childbirth by their relatives. Hospital care, even when available, is not used unless the relatives consider that life is in danger. This is in part due to distrust of hospitals and in part to the great difficulties of transport in the desert. As a result women may lie in childbirth for seven to ten days without any skilled attention whatsoever, and large fistulae between the bladder and anterior vaginal wall may occur from prolonged pressure of the foetal head.

Material of the Present Series

During the last three years 29 cases of vesico-vaginal fistula have been admitted to the gynaecological wards of the Royal Hospital, Baghdad. Of the 29 cases 25 came from outlying villages or the desert. In all of them the infant was stillborn. The average time in labour was about 120 hours, but six women were known to have been in labour for over 168 hours and one for 240 hours. As a rule the infant was extracted manually by