Staff Meeting Bulletin Hospitals of the » » » University of Minnesota

Aviation Medicine

Volume XV | Friday, January 21, 1944 | Number 13

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
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Volume XV | Friday, January 21, 1944 | Number 13

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William A. O'Brien, M.D.

I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS
January 24 - 29, 1944

Visitors Welcome

Monday, January 24

9:00 - 10:00	Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff, Todd Amphitheater, U. H.
9:00 - 11:00	Obstetrics and Gynecology Conference; J. L. McKelvey and Staff, Interns Quarters, U. H.
12:30 - 1:30	Pediatrics Seminar; Physiological and Clinical Tests of Autonomic Function and Autonomic Balance; A. V. Stoesser, W-205 U. H.
12:30 - 1:30	Pathology Seminar; Lantern slide demonstration of some Tropical Diseases; E. T. Bell, 104 I. A.
7:30	Cancer Biology Seminar; Influence of Castration on the Development of Mammary Cancer on Mice; Staff, 116 M. S.

Tuesday, January 25

8:00 - 9:00	Surgery Journal Club; O. H. Wangensteen and Staff, Main 515, U. H.
9:00 - 10:00	Roentgenology-Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff, Eustis Amphitheater, U. H.
11:00 - 12:00	Urology Conference; C. D. Creevy and Staff, Main 515, U. H.
12:30 - 1:30	Pathology Conference; Autopsies. Pathology Staff, 104 I. A.
12:30 - 1:30	Physiology-Pharmacology Seminar; Isotope Studies of Transport across the Intestinal Epithelium; M. B. Visscher, 214 M. H.
4:30 - 5:30	Obstetrics and Gynecology Conference; J. L. McKelvey and Staff, Station 54, U. H.
4:00 - 5:00	Pediatrics; Grand Rounds; I. McQuarrie and Staff; W-205 U. H.
5:00 - 6:00	Roentgen Diagnosis Conference; Annette Stenstrom, Lewis Bixler, M-515 U. H.

Wednesday, January 26

11:00 - 12:00	Pathology-Medicine- Surgery Conference; Acute Pyelonephritis, Possible Pancreatic Calcification; E. T. Bell, C. J. Watson, O. H. Wangensteen and Staff, Todd Amphitheater, U. H.
12:30 - 1:30	Pharmacology Seminar; Blackwater Fever: J. T. Litchfield, Jr., 105 M. H.
4:15 - 6:00	Obstetrics and Gynecology Journal Club; J. L. McKelvey and Staff; Station 54, U. H.

4:30 - 5:30 Neurophysiology Seminar; Release of Acetycholine at Voluntary Nerve Endings; Frederic Kottke, 129 M. H.

8:00 Interdepartmental Seminar; The Isolation and Identification of Steroid Compounds from the Urine of Patients with various derangements of the Adrenal Gland: H. L. Mason, Mayo Foundation; The Influence of Dietary Restriction on Fertility: Zelda Ball, Cancer Biology; The Effect of Lithosperm ruderale on the Sexual Cycle of Mice: Elizabeth M. Cranston, Pharmacology and Bureau of Plant Industry, U.S.D.A.; Certain Biochemical Reactions of Vitamin E: Richard H. Barnes, Walter O. Lundberg and Geo O. Burr, Physiological Chemistry. Eustis Amphitheatre.

Thursday, January 27

9:00 - 10:00 Medicine Case Presentation; C. J. Watson and Staff, Todd Amphitheater, U. H.

10:00 - 12:00 Medicine Rounds; R. J. Watson and Staff, East 214 U. H.

12:30 - 1:30 Physiological Chemistry Seminar; Oral and Dental Biochemistry; W. D. Armstrong, 116 M. H.

12:30 - 1:30 Poliomyelitis Seminar; Muscular Coordination; Starke Hathaway, 113 Mo. S.

5:00 - 6:00 Roentgenology Seminar; Reviews of Recent Radiological Literature; Staff, M-515 U. H.

4:30 - 5:30 Bacteriology Seminar; Evidence Concerning the Etiology of Colds, Infectious Mononucleosis and Herpes Zoster; Jeanne Khanke, 129 M. H.

Friday, January 28

9:00 - 10:00 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

8:30 - 10:00 Grand Rounds; I. McQuarrie and Staff.

10:00 - 12:00 Medicine Ward Rounds; C. J. Watson and Staff; East 214 U. H.

11:45 - 1:15 University of Minnesota Hospitals General Staff Meeting: Rational Therapeutics; R. N. Bieter, Powell Hall Recreation Room.

1:00 - 2:00 Medicine Case Presentation; C. J. Watson and Staff; Main 515, U. H.

1:00 - 2:30 Dermatology and Syphilology; Presentation of selected Cases of the Week; Henry E. Michelson and Staff; W-306 U. H.

1:30 - 3:00 Roentgenology-Neurosurgery Conference; H. O. Peterson, W. T. Peyton, and Staff, Todd Amphitheater, U. H.

Saturday, January 29

9:00 - 11:30 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U. H.

9:00 - 10:00 Medicine Case Presentation; C. J. Watson and Staff; Main 515, U. H.

10:00 - 12:00 Medicine Ward Rounds: C. J. Watson and Staff; E-214, U. H.

11:30 - 12:30 Anatomy Seminar; Review of Cushing's "Bibliography of Vesalius"; S. P. Miller, 226 I. A.

II. SOME ASPECTS OF AVIATION MEDICINE

George N. Aagaard

The prominent part aviation plays in military life today, and the increasing part it will play in transportation in the post-war world, make it essential for all physicians to have a knowledge of the principles of aviation physiology and medicine. In this article an attempt is made to discuss some of the more important of these principles and their application. Obviously, it would be impossible to cover the subject thoroughly in the limited time and space.

The term anoxia as used in aviation medicine is intended to mean only oxygen want or deficiency, not a complete absence of oxygen as the word implies. The classification of anoxia was mentioned in a recent issue of this bulletin. Of the four types of anoxia aviation deals chiefly with anoxic anoxia, or anoxemia. This is due to a deficient supply of oxygen in the inspired air with a resultant decrease in the oxygen tension in the arterial blood. The deficiency of oxygen at high altitudes is not due to a decreased percentage of oxygen in the atmosphere, since the percentage of all the gases in the atmosphere has been shown to be constant up to altitudes of 70,000 feet or more. The partial pressure of oxygen is the all-important factor. The accompanying table shows the partial pressure of oxygen at selected altitudes.

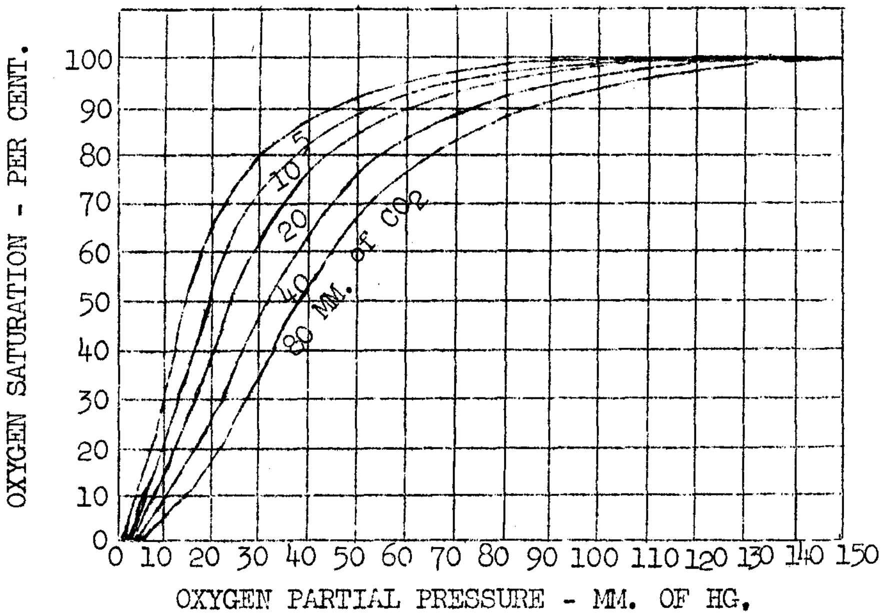
Altitude-pressure table based on the United States standard atmosphere--feet-millimeters

ALTITUDE	PRESSURE	ALTITUDE	PRESSURE	ALTITUDE	PRESSURE	ALTITUDE	PRESSURE
feet	mm. Hg	feet	mm. Hg	feet	mm. Hg	feet	mm. Hg
0	760	21,000	334.8	41,000	134.2	61,000	51.6
5,000	632.4	26,000	269.8	46,000	105.7	66,000	40.7
10,000	522.6	31,000	215.4	51,000	83.2	71,000	32
15,000	428.8	36,000	170.4	56,000	65.5	76,000	25.2
20,000	349.2						

(Taken from Principles and Practice of Aviation Medicine by Harry G. Armstrong, Williams and Wilkins Company, Baltimore, 1939.)

Since it is the partial pressure of oxygen that determines the degree of oxygen saturation in the arterial blood, it is evident that there will be an increasing deficiency of oxygen in the blood as one ascends above sea-level. Increasing the percentage of oxygen in the inspired mixture will help to correct this deficiency and equipment to accomplish this has long been in use in commercial and military aviation. However, such methods of increasing the percentage of oxygen in the inspired air are effective only to a certain point. Even if oxygen makes up one hundred percent of the inspired mixture, it will not be effective if the atmospheric pressure is below that required to maintain a minimum essential level of oxygen saturation in the hemoglobin. The accompanying graph (Fig. 1) shows the relationship of the percentage saturation of hemoglobin with oxygen to the partial pressure of oxygen in the blood.

*From the department of Medicine, University of Minnesota.



(Taken from Principles and Practice of Aviation Medicine by Harry G. Armstrong, Williams and Wilkins Company, Baltimore, 1939.)

This brings up another important and not generally appreciated fact. Since the arterial blood receives its oxygen from the air in the alveolae, it is the oxygen tension in the alveolar air which, in the final analysis, determines the oxygen tension in the blood. Alveolar air contains water vapor at a pressure of 47 mm. Hg. This is constant at all altitudes. As the atmospheric pressure decreases, it is evident that the proportion of the total pressure taken up by water vapor increases significantly and limits the pressure available to oxygen. Carbon dioxide is also present in the alveolar air in amounts much greater than in atmospheric air and also decreases the oxygen partial pressure in the alveolae in a similar fashion. Armstrong (1) has stated that water vapor and carbon dioxide together total 86 mm. Hg. pressure in the alveolar air. Therefore at an altitude of 50,313 feet where the atmospheric pressure is 86 mm. Hg. the oxygen tension in the alveolar air would be zero. Actually, however, a reduction in carbon dioxide tension due to increased pulmonary ventilation permits an increase in oxygen pressure. It is generally considered that the highest altitude at which humans can survive while breathing pure oxygen is 42,000 to 45,000 feet if some method of increasing the pressure is not used. Pressure cabins, pressure suits, and pressure masks by providing an increased atmospheric pressure, enable humans to go to higher altitudes with adequate partial pressure of oxygen.

Any type of anemia with the resultant decrease in oxygen carrying capacity will limit the altitude to which ascent can safely be made. In military evacuation work in which hemorrhage plays an important part this is a paramount consideration. Carbon monoxide poisoning which produces anoxia by reducing the oxygen carrying capacity of the blood must be guarded against in aviation by proper measures to keep aircraft cabins free of engine exhaust gases.

Chemical Changes in the Blood During Anoxia

For many years it was thought that anoxia caused acidosis. Accumulation of lactic acid and other metabolic products was held to be the cause of this bodily change. Haldane showed that there was a decrease in the excretion of acid in the urine and that the urine became alkaline. There is general agreement today, that moderate degrees of anoxia by increasing pulmonary ventilation, cause an excessive loss of carbon-dioxide and a tendency to alkalosis (25). This is compensated by an increased excretion of base by the kidneys. However, anoxia of extreme degree may produce acidosis by impaired oxygenation in the tissues and the accumulation of acid products; plus the failure of the respiratory center and a diminution in breathing, with a resultant accumulation of CO_2. Prolonged anoxia is followed by a decrease in the alkaline reserve due to the increased excretion of base mentioned above.

There is considerable evidence at present to indicate that the blood sugar rises in normal individuals during anoxia. This is thought to be due to increased adrenal activity.

The Heart and Circulation During Anoxia

The heart rate increases with increases in altitude (2, 13, 14). This increase is more pronounced when exercise is attempted. Exercise which causes only a slight increase of heart rate at sea-level will cause a marked increase at an altitude of 15,000 feet or greater (23). Blood pressure has been shown to change but little until an altitude of 15,000 feet is reached (13). At that level there most often is a slight rise in systolic pressure and also a slight fall in the diastolic pressure with a resultant increase in pulse pressure. In extreme anoxia blood pressure may fall to such an extent that syncope occurs.

The minute output of the heart has been shown by Wiggers (27) to be increased at altitudes up to 15,000 feet largely by an increase in the heart rate. Circulation to vital regions may be increased further by regional constriction and dilatation of the blood vessels. At altitudes from 15,000 feet to 22,000 feet which are equivalent to oxygen percentages of 12 to 9%, respectively, there is an increase in the diastolic heart volume and an increase in the stroke output which further increases the minute output. At altitudes exceeding 30,000 feet a circulatory crisis with fall in blood pressure and decreased output is likely to occur.

Anoxia has been shown to increase coronary flow (8, 27). It has been stated that anoxia is the most effective coronary dilator known. Wiggers has expressed the view that it is this increased coronary flow which enables the heart to perform increased work under anoxic conditions. In fact he feels that it is only when the anoxia becomes so marked that further coronary dilatation is not possible, that the circulatory crisis occurs.

Many workers have shown that anoxia causes changes in the electrocardiogram (10). The most frequently observed changes are flattening or inversion of the T waves and depression of the ST segment. Changes in conduction, although less frequent in occurrence, are noted in more severe degrees of anoxia and are revealed by increase in the PR interval and changes in the QRS complex.

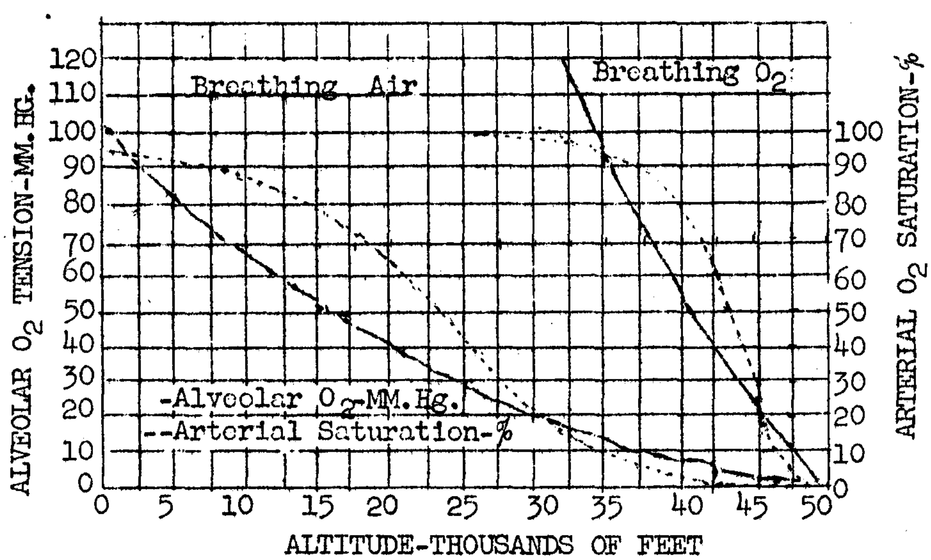
Patients with chronic valvular or hypertensive heart disease in whom cardiac enlargement of moderate or marked degree, and/or reduction of the cardiac reserve has already taken place, should not be subjected to anoxia. Similarly, patients with coronary arteriosclerosis in whom the compensatory increase in coronary flow might not be forthcoming would be poor risks. However, such patients should be able to travel at altitudes presently used by commercial aircraft if oxygen can be administered.

Anoxia and Respiration

Schneider, who with his co-workers, has done much investigative work on this subject, found that an increase in the depth of respiration occurred in most individuals at altitudes of 4,000 to 6,000 feet (14, 15). The minute volume volume of respiration was increased with increasing altitude only by this means until an altitude of 15,000 feet was reached. Above this level, the rate of respiration was also increased. Exercise at high altitude causes great increases in both the rate and depth of respiration. Somervell (23) in one of the Mt. Everest expeditions noted that from seven to ten complete respiratory cycles were required for each step. Schneider states (21) that when the increased respiratory volume observed at high altitudes is corrected for temperature and pressure (to equivalent volume at 0 C., and 760 mm. Hg. pressure) it is actually from 10 to 78% lower than that to be expected at the same conditions at sea level.

It is well known that the respiratory center is relatively insensitive to decreases in oxygen tension and very sensitive to increases in carbon dioxide tension. The increases in depth and rate of respiration associated with anoxia cannot be explained by direct action of the respiratory center. Comroe and Schmidt (19, 20) have shown that chemoreceptors located in the aortic and carotid bodies are very sensitive to decreased oxygen tension and stimulate the respiratory center. This is fortunate since, as has been shown above, there is a marked decrease in carbon dioxide tension and therefore a decreased stimulation to the center in anoxic anoxia. In fact, without the chemoreceptors in the carotid body the respiratory center is depressed by oxygen lack. Comroe and Schmidt have mentioned the following interesting possibility; in anoxic anoxia the respiratory center might be depressed by oxygen lack and only the stimulation received from the carotid and aortic bodies might be maintaining it in action. If under such conditions, the concentration of oxygen is suddenly increased as by applying a mask, the stimulation from the chemoreceptors would suddenly be stopped and there might not be enough carbon dioxide in the blood to stimulate the depressed respiratory center and respiration might cease.

It has been shown that alveolar carbom dioxide tension decreases with increasing altitude (15, 22). This is, of course, caused by increased loss of the gas due to the increased respiratory volume. Since the decreased partial pressure of carbon dioxide permits an increased partial pressure of oxygen in the alveolar air, this becomes a matter of considerable importance when considering the use of pure oxygen at high altitudes.



(Taken from Principles and Practice of Aviation Medicine by Harry G. Armstrong, Williams and Wilkins Company, Baltimore, 1939.)

Figure 2 shows the alveolar oxygen tension at increasing altitudes breathing air and breathing 100% oxygen. It can be seen that when breathing 100% oxygen the tension of oxygen in the alveolar air is maintained at normal values up to approximately 33,000 feet. At higher altitudes the oxygen tension in the alveolar air and consequently the percentage saturation of hemoglobin falls abruptly, until both reach zero at 50,000 feet. In going from 40,000 feet to 45,000 feet the oxygen saturation of the hemoglobin falls from approximately 80% to 30%. It is probably in this range that the absolute ceiling of most normal individuals lies, when breathing 100% oxygen without some method of increasing pressure. Usually, however, 37,000 feet is given as the upper limit of flight with oxygen equipment.

It is interesting to note that hyperventilation and alkalosis may occur at altitudes used in commercial aircraft during the pre-war period. Uihlein and Boothby (24) have reported two such cases. In one of the patients tetany with carpopedal spasm was observed. Apprehension during flight in nervous passengers, when added to the above mentioned increase in ventilation might be sufficient to produce this state. Hinshaw and Boothby (9) have suggested that under unfavorable weather conditions even the most experienced pilot might hyperventilate. Loss of judgement and, "freezing to the controls" under such conditions are offered as a possible explanation for some of the aviation accidents that are laid to pilot failure. In military aviation especially during the strain and tenseness prior to and during hazardous missions the same problem is likely to arise.

Pulmonary conditions which decrease the vital capacity and contraindicate or limit flying include chronic tuberculosis, emphysema, pneumoconiosis, and bronchiectasis. Conditions of the lung which might be made worse under anoxia include pneumonia, bronchitis, and other infectious diseases including tuberculosis. Expansion of confined air would constitute a hazard in pneumothorax, lung cysts, and lung abscesses in which there is not free egress of gas from the lesion to the bronchial tree. The problem of hydrothorax has intrigued the author. If the atmospheric pressure is decreased sufficiently, would gases come out of solution and hydropneumothorax result? Thus far I have found no report of such an occurrence in the literature.

Anoxia and the Nervous System

Of the many changes caused by anoxia none are more interesting than those found in the central nervous system. Spinal fluid pressure and the volume of the brain have been shown to increase during anoxia. This has been shown to occur when the oxygen percentage is reduced by increasing the nitrogen percentage and maintaining the mixture at atmospheric pressure. Landis (11) found that anoxia caused an increase in capillary permeability both for plasma and for proteins. It has been suggested that by this means cerebral edema occurs and the volume of the brain is increased. The headaches, dizziness and fatigue which often persist for several days after high altitude flights may perhaps be caused by some such mechanism. In carbon monoxide poisoning in which anoxia is the chief change, it has been shown that increased spinal fluid pressure occurs. (5)

The effects of anoxia on the intellectual processes are of great interest. Barcroft (3) in an early report of such observations stated that an effect similar to acute alcoholic intoxication occurs. He found first, a sense of stimulation with a sense of superiority, loss of judgement, and fixity of purpose. The importance of such changes in military and commercial aviation is obvious. Later, depression with lack of interest, and inability to realize impending unconsciousness is noted. The higher mental powers as noted above are depressed early, whereas the sensory and motor functions are effected much later. Several observers have, however, reported that when they realized their need of oxygen they were almost unable to take the physical action necessary to start it flowing. McFarland (16,17,

18) has shown that at an altitude of 15,000 feet there is definite increase in reaction time. Visual acuity and depth perception have been shown to decrease at altitudes at which commercial planes fly under certain circumstances.

Oxygen Equipment

Since lack of oxygen may cause difficulty in so many ways one can rightly ask, should we fly at such high altitudes that we become anoxic. The reason is, that even in commercial aviation the advantages of more constant weather conditions and better performance obtained at high altitudes are great. In military aviation the advantage of greater surprise must be added to these. Therefore, means have been sought to combat anoxia. Inhalation of oxygen by means of a mask is at present the most commonly used method. It is important that the mask fit tightly or the oxygen will be diluted by the atmosphere. Oxygen systems may be divided into two chief types: The constant flow and demand types. In the constant flow system oxygen flows into the mask at whatever rate is set and remains at that rate unless the setting is changed. Obviously this is wasteful unless the flyer is constantly changing the setting as he descends. The demans system gives oxygen only in inspiration and is shut off on expiration. In addition an aeronoid valve automatically permits a variable amount of the atmosphere to be inhaled with each inspiration. The proportion of atmospheric air inspired is automatically decreased as one ascends until finally pure oxygen is delivered.

Army Air Force regulations as recently stated by General Grant (7), the Air Surgeon, provide for oxygen under the following circumstances: all flights of one hour duration at altitudes of 10,000 feet or more; all flights over 15,000 feet; from the ground up when the rate of climb is 2,000 feet per minute; all night flying.

The Effects of Decreased Atmospheric Pressure

Gases which are confined within the body cavities will follow Boyles law and expand as the pressure is reduced. This becomes a matter of extreme importance when one considers the volume of gas which may be present at times in the gastro-intestinal tract. A volume of gas will increase to five times its sea-level volume when an altitude of 38,389 feet is reached. Individuals who find it impossible to belch will often experience serious pain, and in our brief experience we have had to discontinue the flight on two occasions because of extreme abdominal distension. In one of these candidates a large amount of carbonated beverage had been consumed shortly before the ascent. Discomfort is often first noted at about 12,000 feet but it is usually easily controlled by belching or the passing of flatus. If the ascent is slow enough, there will be ample time for intestinal gases to work up or down to the nearest orifice and be eliminated, but an excessive amount of gas or a rapid ascent may cause large amounts of gas to be trapped in the segments of the gut where escape is impossible and severe pain may result. The importance of avoiding high altitude flying in patients who habitually have large amounts of gas in their gut is obvious. The use of air ambulances to evacuate the wounded should probably be avoided during the immediate post-operative period in laporotomized patients. Patients whose vital capacity is already reduced from pulmonary disease might well have difficulty if the diaphragms were elevated by abdominal distension.

Lovelace (12) and his co-workers have emphasized the dangers which may be encountered in taking a patient with pneumothorax up even to altitudes which are commonly used in commercial air traffic today. With decreasing atmospheric pressure the volume of the air in the pleural space will increase at the expense of the lung with reduction of the vital capacity and possible tearing of pleural adhesions. The frequent use of artificial pneumothorax in modern therapy of tuberculosis and the frequent occurrence of chest wounds in modern military operations make this a matter of importance to civilian and military physicians alike.

It is interesting to note that the decreased density of the air at higher altitudes decreases the effort required in respiration. Somervell (23), mentioned that even though his respirations were extremely rapid at 28,000 feet the effort involved was extremely small. Asthmatics may experience relief in flying at moderate altitudes while breathing 100% oxygen due to the decreased amount of nitrogen in the inspired air mixture.

The ear and the nasal sinuses are greatly effected by changes in the atmospheric pressure. In making an ascent, the intra-aural pressure exceeds the atmospheric pressure. Automatically, and periodically air is forced out into the nasopharynx through the Eustachian tube. This is usually signalled to the flyer by a clicking sound in the ear. This automatic adjustment of intra-aural pressure to environmental pressure does not occur on descent however. The Eustachian tube with its fibrous collapsible medial portion acts as a flutter valve and permits the free passage of air from within out, but not from without in. An active opening of the Eustachian tube by the musculature of the nasopharynx is required to permit the air to enter the middle ear from the naso-pharynx. Yawning, screaming and the familiar Valsalva technique are methods of equalizing intra-aural pressure.

If the pressure of the atmosphere exceeds the intra-aural pressure by more than 80 to 90 mm. Hg. it becomes impossible for voluntary action to open the collapsed tube (14). Under such circumstances it is necessary to ascend again sufficiently to decrease the differential in pressure so that voluntary action may complete the equalization. Inability to maintain the equalized pressure in the ears is of common occurrence in inexperienced personnel and in our work with indoctrination flights we have frequently found it necessary to resort to ascent and more gradual descent. Such symtoms usually do not occur until an altitude of 25,000 to 15,000 feet is reached on a descent from an altitude of 40,000 feet. It is interesting to note that experienced workers in low pressure chambers are able to hold the Eustachian tube patent continuously and thus descend from altitudes of 40,000 feet to sea level in 30 seconds or less. The nasal sinuses react similarly to the middle ear in changes of altitude. Conditions in the nasopharynx which would impair such adjustments of pressure as have been noted above would disqualify for high altitude.

When the middle ear is improperly ventilated on descent from high altitude, severe pain, dizziness, loss of hearing and even nausea and vomiting may occur. The ear drum becomes red, swollen and retracted and fluid may accumulate in the middle ear. This condition has become known as aero-otitis. Prevention of it may often be accomplished if all who complain of persistent pain in their ears on reaching ground level are immediately seen by one competent in such work. By prompt shrinkage of the nasal mucosa and forcing of air into the middle ear either by some modification of the Valsalva method or catheterization of the Eustachian tube, immediate relief of pain and prevention of the acute changes noted above may be accomplished.

Behnke (4) has called attention to the development of aero-otitis 18 to 24 hours after descent in flyers who had no distress upon reaching the ground. He suggests that the pure oxygen breathed during high altitude flights may fill the middle ear and during sleep be absorbed leaving a decreased pressure within the middle ear. The sleeper may not be aware of such a change and the pressure differential may be so great when the discomfort finally awakens him that voluntary effort would no longer suffice. The practical importance of this matter is readily seen in sleeper planes where it has been found necessary to awaken passengers on each descent. This also must be considered in transportation of any unconscious patient in air ambulances.

Aero-Embolism

Aero-embolism (1,6) is aviation's counterpart to caisson disease found in divers, tunnel-workers and the like. Since the amount of a given gas will go into solution in a liquid is directly proportional to the pressure of the gas on the liquid, it can be seen that in going from sea-level to high altitudes gases will go out of solution and tend to form bubbles, unless eliminated rapidly enough. Since nitrogen makes up such a large part of the atmosphere and is inert in the body it becomes the most important gas to be considered in this respect, although oxygen and carbon dioxide also play a part in the formation of such bubbles. At sea-level each 100 cc. of blood will contain 1.5 cc. of nitrogen. The fact that nitrogen is several times more soluble in fatty tissues than it is in blood increases its importance. With a decrease of tension of nitrogen in the lungs the nitrogen in solution in the blood will tend to come out of solution in the form of bubbles and the nitrogen in the tissues will come out of the tissues and go into the blood as rapidly as possible. The fatty tissues which hold the most of the gas are usually those with the poorest blood supply and thus one would expect to find the formation of bubbles most commonly around the joints and subcutaneous tissues. This is the case and is the reason for the popular name of "bends".

The most frequent symptoms of aero-embolism are pains and bubbles appearing around the joints. Pruritis, formication and urticaria are common skin manifestations of the condition. Neurological manifestations include pain along the nerve trunks, paralysis, and convulsions and coma. A burning sensation in the chest is said to warn of pulmonary involvement and pulmonary edema has been described. One should expect to find pulmonary infarction rather than pulmonary edema since the bubbles are formed in the tissues and are carried to the heart and thence to the pulmonary circulation where it is conceivable that obstruction of arterial circulation might result.

It is generally believed that the atmospheric pressure may be decreased by one half before formation is likely. Thus in the tunnel worker the pressure may be decreased from six atmospheres to three without danger. This has led to the use of stage decompression in the prevention of decompression sickness in such workers. In aviation one should be able to go from one atmosphere at sea level to one-half atmosphere at 18,000 feet with impunity. Practical experience has shown, however, that symptoms of aero-embolism are very unlikely to occur at altitudes below 30,000 feet no matter how rapid the ascent.

Armstrong (1) showed the formation of gas bubbles in the spinal canal in goats which were subjected to decreased atmospheric pressure. Despite the fact that anoxia was prevented by oxygen administration through special masks, bubbles appeared in the spinal fluid manometer beginning at an altitude of 18,000 feet. Walsh and Boothby (26) repeated this experiment in man and found that bubbles appeared in all three subjects at altitudes of 10,000 to 12,000 feet. In the third case, denitrogenation was attempted by breathing 100% oxygen at rest and during exercise, but bubbles appeared at 12,000 feet none the less.

Rate of ascent and duration of exposure to high altitudes are both important factors in the development of aero-embolism. If the rate of ascent is sufficiently slow, the gases can be eliminated through the lungs as rapidly as necessary to prevent a super-saturation. Similarly, very short flights at high altitudes with a rapid rate of ascent rarely cause symptoms. Long flights at even moderate altitudes may cause symptoms to appear. Differences in individual susceptability even in persons of similar body build are apparent and thus far remain unexplained.

Aero-embolism can be prevented by a sufficiently slow rate of ascent. However in military aviation, particularly in interceptor planes, rapid ascent is essential for the successful performance of the mission. Another method which has been advocated is the breathing of pure oxygen for varying periods before ascent. Since the partial pressure of nitrogen in the alveolae is reduced, nitrogen should come out of solution and be largely eliminated before the flight begins if the period of denitrogenation is long enough. Exercise has been recommended to speed up circulation and thus remove larger amounts of nitrogen in a given period of time. Its effectiveness is, however, still a matter of debate.

Treatment of decompression illness is accomplished by re-compression. In aero-embolism descending to lower altitudes will usually bring about prompt relief of symptoms. Remaining at low altitudes or discontinuing the flight will give continued freedom from symptoms. In this respect the aviator is more fortunate than the diver who lives at the decompressed atmosphere and is subject to delayed attacks if the decompression is not adequate.

Our own experience in aviation medicine has been brief and limited. Recently the University was asked to determine the fitness for high altitude work of flight engineers of the Minneapolis Honeywell Regulator Company. This was possible because the department of aeronautical engineering recently obtained a low pressure chamber which was necessary for a final flight test and indoctrination procedure. Our work has been a cooperative enterprise involving the following departments of the University: Aeor-Engineering, medicine, the laboratory services of the University hospital, ophthalmology and otorhinolaryngology, neuropsychiatry and roentgenology. I wish to express my gratitude to the various members of these departments whose help and advice has been vital to the accomplishment of this work.

Candidates being considered for high altitude work were given an examination which included the following: ear, nose and throat examination including an audiogram and transillumination of the sinuses; a general medical history and physical examination with special attention being devoted to the circulatory and respiratory systems and including vital capacity determination; Minnesota Multi-phasic Personality Inventory; complete blood counts, Kline diagnostic and exclusion tests, blood grouping, and urinalysis; electrocardiogram with precordial leads; Roen Roentgen-kymogram, chest and sinus x-rays and fluoroscopy of the chest. Special studies were performed whenever indicated. These included stereoscopic chest x-rays, electrocardiograms after exercise, and consultation with men in various specialties.

Most of the men examined were in the age group from 25 to 35 years of age. College and university training or some training in a special trade school such as radio or electronics had been received by almost all of the men. Many of the men examined had already been doing considerable work in flight engineering but had begun this work before the program of examination had been started. Since the work of our subjects was concerned primarily in instructing army personnel in the operation and maintenance of their equipment and in checking the performance of such equipment, their visual qualifications were only that they be able to read print and ordinary instrument dials. The strict visual requirements which pertain to pilots do not apply to this group. For this reason there is is a great difference in the percentage of our subjects and none of them were rejected for visual defects. In army pilot selection examinations visual defects comprise the chief cause for rejection.

Of the first one hundred and fifty men examined, twenty were rejected, a total of 13 1/3%. The causes for rejection were as follows:

Ear, nose and throat pathology	13
Cardio-vascular	3
Pulmonary	3
Nervous and mental	3
Hernia	1
	23

One man had three and another two separate conditions which caused rejection. Deflected septums which were obstructive to a high degree and were associated with other pathology in the nose or sinuses were the most frequent disqualifying factor in the ear, nose and throat examination. X-ray evidence of changes in the sinuses without complaints or physical findings to give confirmation was not considered indicative of significant disease. The cardiovascular causes for rejection consisted of two cases of chronic rheumatic valvular heart disease previously unrecognized and moderately severe hypertension. The pulmonary cases were all tuberculous infections in which it could not be stated with certainty that the process had been healed or arrested for a sufficiently long period. It was not felt that inactive pulmonary tuberculosis should consititute a cause for rejection since with the use of 100% oxygen the oxygen content of the inspired air could be kept at normal levels.

Of the remaining 130 subjects, 117 were given tests in the low pressure chamber. This test was given for the purpose of acquainting the men with the oxygen equipment and the need for it, and also to observe their gross reactions to simulated conditions of high altitude. The pressure was gradually reduced to that at an altitude of 40,000 feet after the subject had breathed oxygen for a period of 20 to 30 minutes. The average "flight" was of 45 minutes duration about 20 minutes being devoted to ascent and another 20 minutes to descent. Because of the short exposure, no conclusions can be drawn as to the individual susceptibility to aeroembolism. Of the group tested three developed symptoms and signs of aero-otitis media. All three responded quickly to treatment by vasoconstrictors applied to the mucosa of the nasopharynx followed by forcing air into the middle ear by one of the methods mentioned above. Many of the subjects experienced difficulty in equalizing intra-aural pressure as rapidly as was necessary for the rate of descent. Stopping of the descent and a short ascent permitted them to catch up and further descent could usually be made without difficulty. It was impossible to predict from the results of the nose and throat examination which subjects would have difficulty with their ears. It was our opinion that previous experience was more important than minor abnormalities in determining a subject's ability to take care of his ears. Many subjects who had entirely normal nose and throat examinations found it difficult to adadequately adjust to the pressure changes while others with marked pathology and many hours of flying had no difficulty. An outstanding example of this was the man who had previously served as a dive bomber pilot in the Navy reserve and despite a septum which was almost completely obstructive had no difficulty. Abdominal cramps followed by syncope of 40,000 feet occurred in one subject and was thought to be due to excessive amounts of gas in the intestinal tract with resultant elevation of the diaphragm.

Summary

Some of the important aspects of aviation physiology and medicine are presented. Our own experience in selection of candidates for high altitude work is reviewed.

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III. GOSSIP

Mild weather continues as Snow Week is again cancelled. Minnesota natives have bragged about their rugged climate so long, that some appear disappointed with this extraordinary weather....This was a busy week in War Time Graduate Medical Meetings, with Drs. Braasch and Creevy assigned to Fort Snelling and Wold Chamberlain on Tuesday, J. A. Myers to Fort Des Moines and Camp Dodge on Wednesday, and E. H. Rynearson to Schick General Hospital today. Each group now has a meeting every other week and everyone seems to enjoy it. ...On Monday to Shakopee to speak to the high school boys on their health. This enterprising school features a Health Week in its program of instruction. The children prepare special articles, which they deliver with great gusto. Physicians from the community speak on various subjects, and prominent citizens take part. The idea is the "brain child" of Superintendent John A. Metcalf, energetic school director....The Minnesota State Medical Association radio talk in the tropical medicine series, which drew the largest comment, was the one on filariasis. It has been demonstrated that 70% of the cases in the male show some degree of genital pathologic change, even though, superficially, it is not apparent during the early course of the disease. This produces marked emotional change, and feelings of insecurity. Our men see the advanced changes in the natives, and assume the chronically infested native is a mirror prediction. Unless the patient is given special handling, and protection from remarks by his mates, he will make decisions as to his future which are not wise. It is believed that in the majority of instances that little permanent deformity will follow, as it is proportional to the extent of mechanical obstruction to the lymphatics. This is another example of a disease in which the psychosomatic features outweigh the pathologic changes. Best response to the radio talk came from agencies dealing with troubled relatives in our city who had received discouraging news from infected patients....Malaria is the ranking disease as far as interest in tropical medicine by the public, is concerned. Specific warnings concerning patients with falciparum malaria are now being broadcast. Patients who have been exposed to possible malarial infection, who show stupor, coma, convulsions, confusion, restlessness, negativism, mild nausea to intractible vomiting, moderate to severe diarrhea, localized or generalized abdominal pain, passage of dark red, brown, or black urine, or even respiratory infections should be suspected of having malaria. If the diagnosis of malaria cannot be made, and the patient progress unfavorably, specific treatment should be instituted. Everyone is urged to be on the alert, for it is now known that malaria has replaced syphilis as the great mimic of other diseases....Louie Daniels who went so suddenly during the holiday season, left behind many examples of his exquisite writing. I have one before me now which tells of the efforts his father made to prevent his family from contracting colds. His father built a sleeping porch where the kids had lots of fun, but did not get very much sleep. Next they tried cold baths. He remembers them because everybody slapped and snorted, and shrieked in his turn, and then waited to hear the next victim. The children caught their father using warm water one morning, which caused the system to break down. When Louie grew older and left home, the only thing he did about colds was to carry a handkerchief. Now he finds himself confronted with the same problem that his father had, for his wife insists that something should be done to keep the children from catching colds. Being a physician, he is supposed to know something about it. He thinks about sun lamps, cod liver oil, vaccines, and sleeping with the windows closed, and wonders if it wouldn't be a good idea to go back to the sleeping porch so that his kids could take up the family pillow fights where they left off in 1910. He says that he doesn't remember any colds then, nor neither does he remember much of anything else. Louie never wrote anything which did not bring joy to the recipient. I have a small treasure of his writing which he sent when our Patrick arrived on St. Patrick's day, six years ago. He was good medicine for all....