Chapter 2

EFFECTS OF DECREASED PARTIAL PRESSURE OF OXYGEN ON RESPIRATORY PHYSIOLOGY

It is recognized generally that the most serious, single danger for the flier is the decreased partial pressure of oxygen encountered at low barometric pressures which, without the proper use of oxygen equipment and cabin pressurization, can quickly lead to incapacitation or even death, depending on the flight altitude. This type of hypoxia (insufficient oxygen in the inspired air) ranges from moderate to severe, to fulminating at altitudes between 10,000 and 35,000 feet and higher. At least 75 hypoxic fatalities occurred in the European Theater during World War II at altitudes between 17,000 and 31,000 feet. In these cases, the duration of the hypoxic exposure prior to death varied between less than 3 minutes (five cases) to more than an hour, with 27 fatalities reportedly occurring within 10 minutes or less, as estimated by fellow crewmembers. Two deaths occurred at altitudes between 17,000 and 20,000 feet, a fact which emphasizes that, under no conditions can the lethal effects of acute altitude hypoxia be underestimated, particularly in view of the current routine flight altitudes above 40,000 feet for commercial and military aircraft and manned space flights.

Man's tolerance to hypoxia has not changed since World War II, but the altitude capability of high performance aircraft and the requirement for adequate protective equipment and strict oxygen discipline have all continued to increase in importance and significance. Even hypoxic episodes that lead only to mental confusion or unconsciousness, but not necessarily death, may result ultimately in the total loss of the aircraft, crew, and passengers, because of the mental disorientation during and following the episode and the consequences stemming from uncontrolled aircraft at possibly supersonic speeds.

To understand and appreciate the nature of altitude hypoxia requires primarily an understanding of the physiology of respiration under both normal and abnormal environmental conditions.

The chief purpose of the respiratory process is to supply the lungs and, consequently, the blood and tissues with adequate oxygen and to eliminate the carbon dioxide that is generated by the metabolism of the body tissues—thus a homeostatic state is maintained in spite of a wide variety of conditions and activities. The respiratory process, in conjunction with the renal system, also plays a role in maintaining the acid-base balance of the body within narrow limits under normal environmental conditions, however, in chronic hypoxic environments, the kidney is the major factor in this regard and is important in the process of altitude acclimatization.

Respiration may be divided into three general categories—namely, the pulmonary phase, the blood transport phase, and the tissue phase. The pulmonary phase involves the exchange of gases between the external or ambient atmosphere and the alveolar air, and between the alveolar air and the blood in the pulmonary capillaries. The transport phase depends on an adequate cardiovascular system and blood constituents for transporting the respiratory gases in adequate quantities between the lungs and tissues. The

tissue phase of respiration involves the exchange of gases between the cells of the body and the blood in the tissue capillaries.

Pulmonary Phase of Respiration

The total volume of air in the lungs (total lung capacity) is subdivided as shown in figure 2–1. These subdivisions are important in the study of pulmonary function in health, in disease, and under abnormal environmental conditions, such as pressure breathing. The end of a quiet expiration is the usual reference point when making quantitative measurements of these subdivisions.

At the end of quiet expiration, the elastic recoil force of the lung is approximately balanced by the expansile tendency of the chest wall. It, therefore, is often called the equilibrium point. There are four primary lung volumes, as shown in the related illustration. Combinations of two or more primary lung volumes are known as lung capacities. These sometimes reflect the functional compartments of the lung more accurately, than do the lung volumes.

The definitions and average normal values (measured in the resting state, BTPS (gas volume in the lung existing at *B*ody *T*emperature and atmospheric *P*ressure and completely *S*aturated with water vapor at body temperature)) for the primary lung volumes are as follows:

a. *Tidal volume* is the volume of air exchanged in one breath. The resting tidal volume is about 500 cc.

b. *Inspiratory reserve volume* is rarely referred to since it is quite variable, depending on the amount of the tidal volume. It is the maximum amount of air that can be inspired at the end of a resting inspiration.

c. *Expiratory reserve volume* is the maximum amount of air that can be forcibly expired following a normal expiration. The average value is about 1200 cc.

d. *Residual volume* is the amount of air

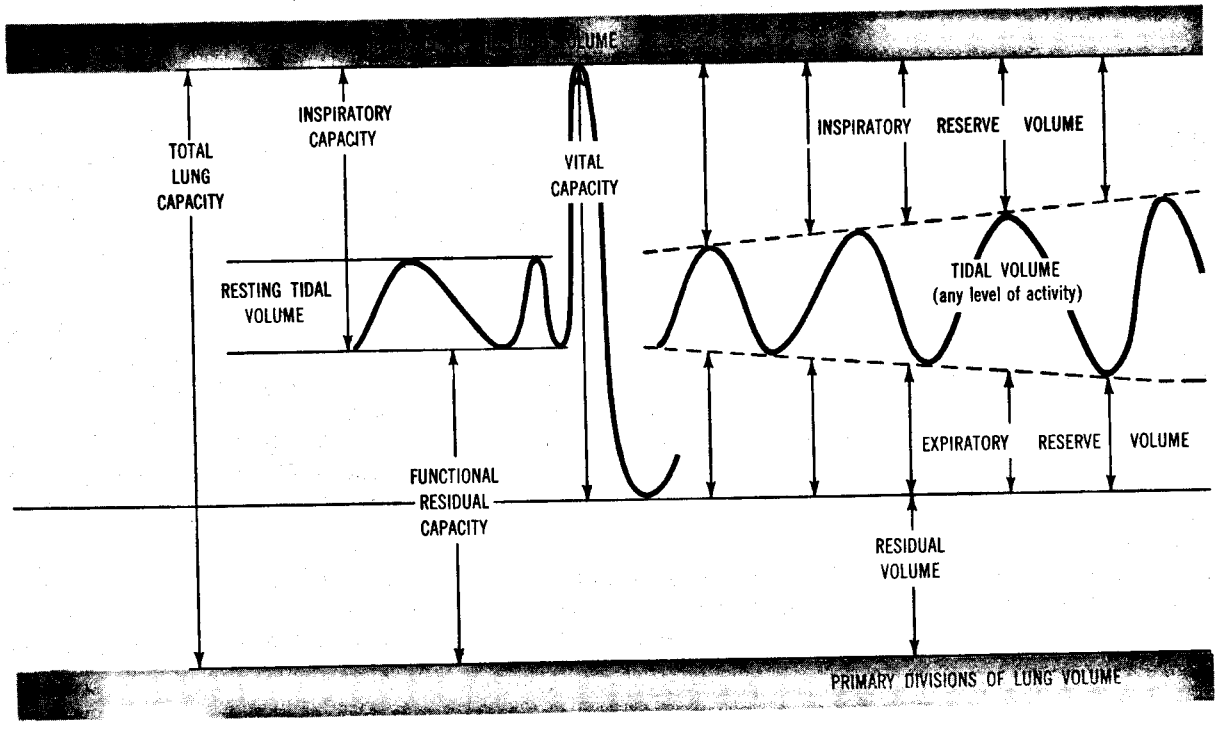


Figure 2–1. Standardization of Terms Used in Respiratory Physiology.

remaining in the lungs following a maximum expiratory effort. The average value is about 1200 cc, and constitutes 20 to 25% of the total lung capacity.

There are also four major lung capacities:

(1) *Total lung capacity* is the sum of all four of the primary lung volumes and averages about 6000 cc.

(2) *Inspiratory capacity* is the maximum volume of air that can be inhaled from the end of a quiet expiration (the sum of the tidal volume and inspiratory reserve volume). The average value is about 3600 cc.

(3) *Vital capacity* is the maximum amount of air that can be exhaled from the lungs following a maximum inspiration. The average value is about 4800 cc. It is the sum of the inspiratory reserve volume, tidal volume, and expiratory reserve volume.

(4) *Functional residual capacity* is the amount of air remaining in the lungs following a normal tidal expiration.

Measurements of all lung volumes and capacities can be made with a spirometer or similar calibrated recording device. The values given for the lung volumes and capacities are approximations only. The values are affected by the age, sex, height, and weight of the subject, and more accurate values may be calculated for individual subjects by using regression formulas which take these variables into account. When regression formulas are used to predict the normal values, the results are often given as "percent of predicted normal."

Knowledge of pulmonary physiology has increased rapidly in recent years. A significant number of advances have resulted from research associated with aviation physiology. A brief summary of some important concepts follows:

The major physiologic functions of the lungs can be grouped under three main headings, namely, *ventilation, diffusion,* and *perfusion.*

(a) *Ventilation* may be defined as the mass movement of air in and out of the lungs or the process by which alveolar air is periodically mixed with atmospheric air. Adequate ventilation is dependent upon the creation of a pressure gradient between the alveoli and the external atmosphere by the bellows action of the chest and diaphragm acting upon the lung. The patency of the airways, the integrity of the "respiratory center" in the medulla, the strength of the intercostal and abdominal muscles and the diaphragm, and the elastic characteristics of the lung and thorax systems are important factors in the maintenance of adequate ventilation. In addition, distribution of the inspired gases throughout the lung is of great importance.

The presence of bronchial secretions, bronchiolar narrowing, or masses occluding some of the airways will cause the alveoli to be unevenly ventilated. Some will be normally ventilated or hyperventilated, and others will be underventilated. Uneven distribution of inspired air may cause the lung to function as a group of compartments, each ventilating at its own rate. For example, about 50% of normal people show relatively slow ventilation of a lung compartment equalling 10 to 50% of the functional residual capacity. In individuals with severe obstructive emphysema, as much as two-thirds of the functional residual capacity may receive only 10% of the total ventilation.

(b) *Diffusion* of gases across the alveolar-capillary wall refers to the mechanism by which the respiratory gases are transferred from the alveolar air to the blood in the pulmonary capillaries and vice versa. Carbon dioxide diffuses across the alveolar wall about 20 times as rapidly as oxygen. However, the pressure gradient of oxygen across the alveolar membrane is normally about 10 times as great as the pressure gradient of CO_2. The presence of fibrosis, granuloma, edema, or exudate in the alveoli or in the alveolar-capillary wall interferes markedly with the process of diffusion and may result in hypoxia or CO_2 retention, or both. Certain types of diffusion abnormalities, such as granulomatous involvement of the alveolar wall in pulmonary sarcoidosis, are referred to as "alveolar-capillary block syndromes."

In diseases leading to abnormalities of diffusion, oxygen diffusion is generally impaired earlier and to a greater degree than is CO_2 diffusion. This is due chiefly to the much greater diffusibility of CO_2 across the alveolar-capillary membrane. Thus, by means of increasing the minute volume of ventilation, normally functioning areas of lung may compensate for CO_2 retention in diseased areas. On the other hand, for end-capillary blood in the pulmonary circulation to become adequately saturated with oxygen, the oxygen must diffuse across the alveolar membrane, through the interstitial fluid and the capillary endothelium. Within the capillary, the dissolved oxygen must then diffuse through the plasma, the red blood cell membrane, and the intracellular fluid within the red cell to combine with the hemoglobin. Thus, oxygen must diffuse from a gaseous state in the alveoli to a dissolved state within the alveolar membrane and the pulmonary-capillary tissues and fluids. The solubility of a gas, as well as its partial pressure, greatly influences its diffusion characteristics. Carbon dioxide is about 25 times more soluble than oxygen in pulmonary tissues and fluids and, as indicated above, its capacity for diffusion is about 20 times greater than oxygen.

Figure 2–2 compares the different rates of oxygen uptake by the blood when breathing low and high levels of oxygen. The average time for the passage of blood through the lung capillaries for the exchange of respiratory gases has been estimated by Roughton to be about 0.75 second when at rest, and about 0.33 second or faster during heavy exercise. (For a detailed and quantitative discussion of the respiratory functions of the blood, refer to chapter 5 (Roughton), *Handbook of Respiratory Physiology*, listed under References at the end of this chapter.)

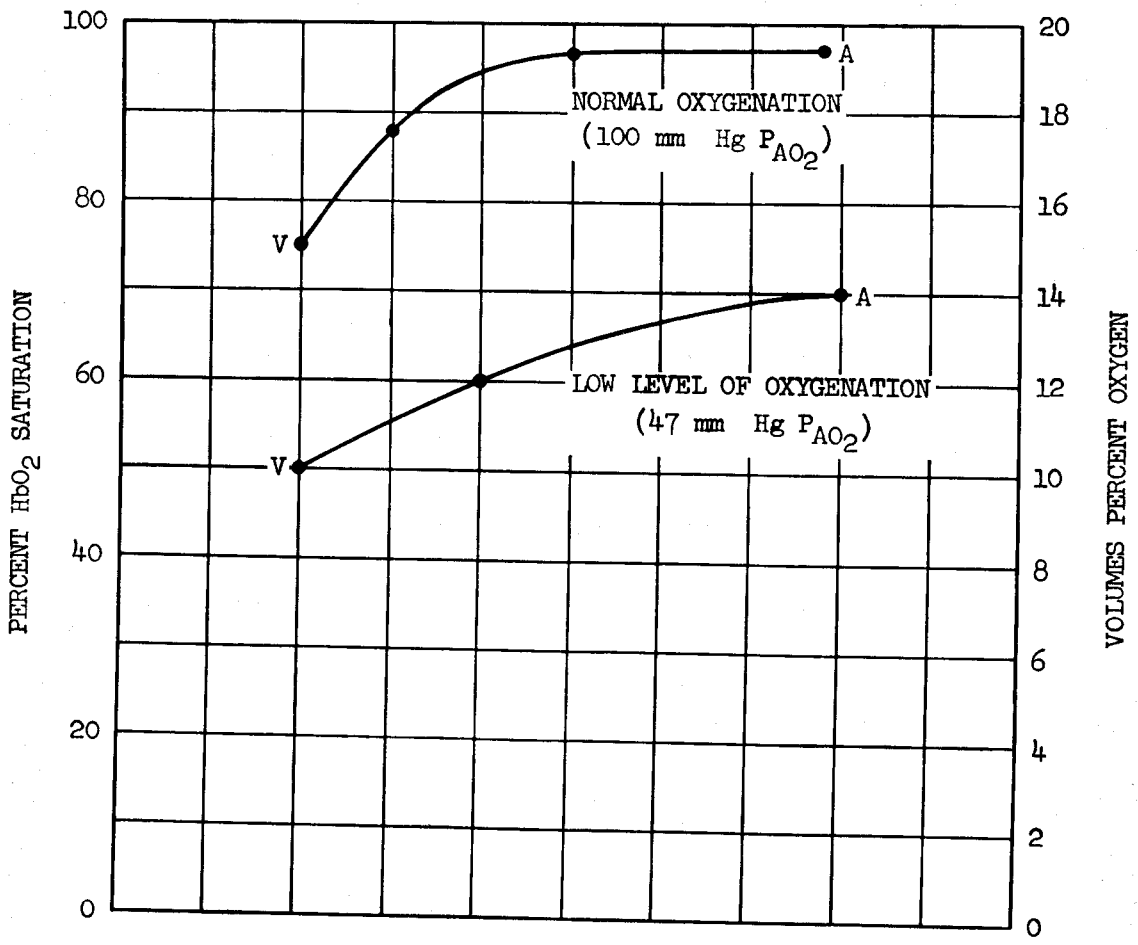
(c) *Perfusion* of blood through the lung capillaries is not always uniform, even in normal individuals. In various disease states, blood flow through the lung may vary greatly from one area to another. Uneven perfusion of the lungs with blood may become a very serious matter when combined with uneven ventilation of the lung. Some areas of lung may be well-ventilated but poorly perfused. These areas merely increase the dead space and do not contribute to gas exchange. Other areas may be well-perfused but poorly ventilated. These areas act virtually as right-to-left vascular shunts since the blood flowing through them retains its venous character.

Disturbances of ventilation-perfusion relationships may occur during flight when "G" forces act on the lung during acceleration, causing redistribution of the blood flow to the lungs. *For example*, during exposure to footward "G" forces, the lower lobes would become somewhat engorged. During exposure to headward G forces, the apical regions would become engorged. Other ventilation-perfusion disturbances can result from pressure breathing and from such dysbaric syndromes as the "chokes" or any abnormal alterations in the pulmonary circulation.

Composition of Respired Air

Dry atmospheric air contains 20.95% oxygen, 79.02% nitrogen, and 0.03% carbon dioxide by volume. Included with the nitrogen are small amounts of rare gases that apparently have no physiological significance. The relative percentage composition of dry atmospheric air does not vary appreciably with altitudes up to 80,000 feet or about 15 miles. Above these altitudes, the percentage of oxygen very gradually decreases and the percentage of the trace gas helium increases slightly because of their molecular weights and the influence of gravity. There are no significant variations with latitude (see table 2–1).

Quantities of gas at various altitudes expressed in percentages of the atmosphere have little significance, for percentage represents the relative volume of a gas and not its molecular concentration. Since molecular concentration determines the availability of the gas to the body, the actual concentration of any gas is best expressed in terms of "partial pressure."



	PERCENT HbO_2 SATURATION	VOLUMES PERCENT OXYGEN
	LOW LEVEL	
1.	50	10
2.	60	12
3.	70	14
	NORMAL LEVEL	
1.	75	15
2.	88	17.6
3.	96	19.2
4.	97	19.4

Changes in percent oxygen saturation as blood passes along a lung capillary under conditions of normal alveolar oxygen tension (100 mm Hg) and alveolar hypoxia (47 mm Hg). A=Arterial; V=Mixed venous blood.

Figure 2-2. Blood Oxygen Saturation Values in a Lung Capillary.

The partial pressure of a gas, in a mixture of gases not interacting with one another, is equal to that pressure which the particular gas would exert if it alone occupied the space taken up by the mixture (Dalton's Law). The "total pressure" of a mixture of gases is, therefore, the sum of the pressures of the individual gases composing the mixture. For moist air, this law can be represented by the formula:

$$P_B = P_{O_2} + P_{N_2} + P_{CO_2} + P_{H_2O}$$

where P_B is the total barometric pressure, and P_{O_2}, P_{N_2}, P_{CO_2}, and P_{H_2O} are the partial pressures of oxygen, nitrogen, carbon dioxide, and water vapor, respectively.

The total standard pressure (barometric pressure) of the atmosphere at sea level is 760 mm Hg (14.7 psi). When the air is assumed to be dry, the partial pressure exerted by oxygen at sea level is:

TABLE 2–1. CHEMICAL COMPOSITION OF THE ATMOSPHERE (DRY) AT SEA LEVEL.

Constituent	*Molecular Weight*	*Percent by Volume*
Nitrogen (N_2)	28.016	78.09
Oxygen (O_2)	32.000	20.95
Argon (A)	39.944	0.93
Carbon dioxide (CO_2)	44.010	0.03
Neon (Ne)	20.183	1.8×10^{-3}
Helium (He)	4.003	5.24×10^{-4}
Krypton (Kr)	83.7	1.0×10^{-4}
Hydrogen (H_2)	2.016	5.0×10^{-5}
Xenon (Xe)	131.3	8.0×10^{-6}
Radon (Rn)	222.	6.0×10^{-18}
Dry Air	28.966	100.00

$$\frac{20.95}{100} \times 760 = 159 \text{ mmHg (3.1 psi)}.$$

The partial pressure exerted by nitrogen at sea level is:

$$\frac{79.02}{100} \times 760 = 601 \text{ mmHg (11.6 psi)}.$$

The partial pressures of the other gases may be similarly calculated.

Table 2–2 summarizes the barometric pressure of the atmosphere at various altitudes.

Composition of Pulmonary Air

The atmospheric air that is drawn through the nasal passages into the trachea becomes saturated with water vapor. Furthermore, it mixes with the alveolar air. One must visualize in the alveoli, an interface across which gaseous interchange occurs between the air previously present in the alveoli and that which has newly entered. The newly entered air "delivers" oxygen and "receives" carbon dioxide, whereas that already present in the alveoli "receives" oxygen and "yields" carbon dioxide. Therefore, expired air contains less oxygen and more carbon dioxide than does inspired air which normally is essentially free of carbon dioxide. Expired air does not give a true picture of the conditions that exist in the alveoli, since it is a mixture of air from the alveoli and from the dead space. The partial pressure of oxygen in the alveoli determines how much oxygen reaches the blood and tissues. The partial pressures of the gases in the alveoli at sea level and at various altitudes when breathing air and when breathing 100% oxygen are shown in table 2–3, which provides a comparison of the equivalent altitudes at which the alveolar gas compositions are essentially the same.

When man is breathing pure oxygen at 33,700 feet, the partial pressure of oxygen in the alveoli is the same as the pressure at sea level when breathing air. Above 34,000 feet, the partial pressure of oxygen in the lungs begins to fall below the pressure at sea level, even though 100% oxygen is breathed. At altitudes greater than 40,000 feet, the partial pressure of oxygen decreases rapidly

TABLE 2–2. BAROMETRIC PRESSURE AND TEMPERATURE CHANGES WITH THE GEOMETRIC ALTITUDE.

US Standard Atmosphere (1962) (1 torr = 1 mm Hg)

	Pressure			*Temperature*	
Alt (Feet)	*Torr*	*in.Hg*	*PSIA*	*°F.*	*°C.*
Sea Level	760.00	29.92	14.70	59.0	15.0
500	746.37	29.38	14.43	57.2	14.0
1000	732.93	28.86	14.17	55.4	13.0
1500	719.70	28.33	13.92	53.7	12.0
2000	706.66	27.82	13.66	51.9	11.0
2500	693.81	27.32	13.42	50.1	10.0
3000	681.15	26.82	13.17	48.3	9.1
3500	668.69	26.33	12.93	46.5	8.1
4000	656.40	25.84	12.69	44.7	7.1
4500	644.30	25.37	12.46	43.0	6.1
5000	632.38	24.90	12.23	41.2	5.1
5500	620.65	24.43	12.00	39.4	4.1
6000	609.09	23.98	11.78	37.6	3.1
6500	597.70	23.53	11.56	35.8	2.1
7000	586.49	23.09	11.34	34.0	1.1
7500	575.45	22.66	11.13	32.3	0.1
8000	564.58	22.23	10.92	30.5	−0.8
8500	553.88	21.81	10.71	28.7	−1.8
9000	543.34	21.39	10.51	26.9	−2.8
9500	532.97	20.98	10.31	25.1	−3.8
10000	522.75	20.58	10.11	23.4	−4.8
10500	512.70	20.19	9.91	21.6	−5.8
11000	502.80	19.80	9.72	19.8	−6.8
11500	493.06	19.41	9.53	18.0	−7.8
12000	483.48	19.03	9.35	16.2	−8.8
12500	474.04	18.66	9.17	14.5	−9.8
13000	464.76	18.30	8.99	12.7	−10.7
13500	455.62	17.94	8.81	10.9	−11.7
14000	446.63	17.58	8.64	9.1	−12.7
14500	437.79	17.24	8.47	7.3	−13.7
15000	429.08	16.89	8.30	5.5	−14.7
15500	420.52	16.56	8.13	3.8	−15.7
16000	412.10	16.22	7.97	2.0	−16.7
16500	403.82	15.90	7.81	0.2	−17.7
17000	395.67	15.58	7.65	−1.6	−18.7
17500	387.65	15.26	7.50	−3.4	−19.6
18000	379.77	14.95	7.34	−5.1	−20.6
18500	372.02	14.65	7.19	−6.9	−21.6
19000	364.40	14.35	7.05	−8.7	−22.6
19500	356.90	14.05	6.90	−10.5	−23.6
20000	349.53	13.76	6.76	−12.3	−24.6
20500	342.29	13.48	6.62	−14.0	−25.6
21000	335.17	13.20	6.48	−15.8	−26.6
21500	328.16	12.92	6.35	−17.6	−27.6
22000	321.28	12.65	6.21	−19.4	−28.5
22500	314.51	12.38	6.08	−21.2	−29.5
23000	307.86	12.12	5.95	−22.9	−30.5
23500	301.33	11.86	5.83	−24.7	−31.5
24000	294.91	11.61	5.70	−26.5	−32.5
24500	288.60	11.36	5.58	−28.3	−33.5
25000	282.40	11.12	5.46	−30.0	−34.5
25500	276.31	10.88	5.34	−31.8	−35.5
26000	270.32	10.64	5.23	−33.6	−36.4

TABLE 2–2. Continued.

Pressure				*Temperature*	
Alt (Feet)	*Torr*	*in.Hg*	*PSIA*	*°F.*	*°C.*
26500	264.44	10.41	5.11	−35.4	−37.4
27000	258.67	10.18	5.00	−37.2	−38.4
27500	253.00	9.96	4.89	−38.9	−39.4
28000	247.43	9.74	4.78	−40.7	−40.4
28500	241.96	9.53	4.68	−42.5	−41.4
29000	236.59	9.31	4.57	−44.3	−42.4
29500	231.31	9.11	4.47	−46.1	−43.4
30000	226.13	8.90	4.37	−47.8	−44.4
30500	221.05	8.70	4.27	−49.6	−45.3
31000	216.06	8.51	4.18	−51.4	−46.3
31500	211.16	8.31	4.08	−53.2	−47.3
32000	206.35	8.12	3.99	−54.9	−48.3
32500	201.63	7.94	3.90	−56.7	−49.3
33000	197.00	7.76	3.81	−58.5	−50.3
33500	192.46	7.58	3.72	−60.3	−51.3
34000	188.00	7.40	3.64	−62.1	−52.3
34500	183.62	7.23	3.55	−63.8	−53.2
35000	179.33	7.06	3.47	−65.6	−54.2
36000	170.99	6.73	3.31	−69.2	−56.2
37000	163.00	6.42	3.15	−69.7	−56.5
38000	155.37	6.12	3.00	−69.7	−56.5
39000	148.11	5.83	2.86	−69.7	−56.5
40000	141.18	5.56	2.73	−69.7	−56.5
41000	134.58	5.30	2.60	−69.7	−56.5
42000	128.29	5.05	2.48	−69.7	−56.5
43000	122.30	4.81	2.36	−69.7	−56.5
44000	116.58	4.59	2.25	−69.7	−56.5
45000	111.13	4.38	2.15	−69.7	−56.5
46000	105.94	4.17	2.05	−69.7	−56.5
47000	100.99	3.98	1.95	−69.7	−56.5
48000	96.27	3.79	1.86	−69.7	−56.5
49000	91.77	3.61	1.77	−69.7	−56.5
50000	87.49	3.44	1.69	−69.7	−56.5
51000	83.40	3.28	1.61	−69.7	−56.5
52000	79.51	3.13	1.54	−69.7	−56.5
53000	75.79	2.98	1.47	−69.7	−56.5
54000	72.25	2.84	1.40	−69.7	−56.5
55000	68.88	2.71	1.33	−69.7	−56.5
56000	65.67	2.59	1.27	−69.7	−56.5
57000	62.60	2.46	1.21	−69.7	−56.5
58000	59.68	2.35	1.15	−69.7	−56.5
59000	56.89	2.24	1.10	−69.7	−56.5
60000	54.24	2.14	1.05	−69.7	−56.5
61000	51.71	2.04	1.00	−69.7	−56.5
62000	49.30	1.94	9.53^{-1}	−69.7	−56.5
63000	47.00	1.85	9.09	−69.7	−56.5
64000	44.80	1.76	8.66	−69.7	−56.5
65000	42.71	1.68	8.26	−69.7	−56.5
66000	40.72	1.60	7.87	−69.6	−56.4
67000	38.82	1.53	7.51	−69.1	−56.1
68000	37.02	1.46	7.16	−68.5	−55.8
69000	35.30	1.39	6.83	−68.0	−55.5
70000	33.66	1.33	6.51	−67.4	−55.2
71000	32.10	1.26	6.21	−66.9	−54.9

TABLE 2–2. Continued.

	Pressure			Temperature	
Alt (Feet)	*Torr*	*in.Hg*	*PSIA*	*°F.*	*°C.*
72000	30.62	1.21	5.92	−66.3	−54.6
73000	29.20	1.15	5.65^{-1}	−65.8	−54.3
74000	27.86	1.10	5.39	−65.2	−54.0
75000	26.57	1.05	5.14	−64.7	−53.7
76000	25.35	9.98^{-1}	4.90	−64.2	−53.4
77000	24.19	9.52	4.68	−63.6	−53.1
78000	23.08	9.09	4.46	−63.1	−52.8
79000	22.02	8.67	4.26	−62.5	−52.5
80000	21.01	8.27	4.06	−62.0	−52.2
81000	20.05	7.90	3.88	−61.4	−51.9
82000	19.14	7.54	3.70	−60.9	−51.6
83000	18.27	7.19	3.53	−60.3	−51.3
84000	17.44	6.87	3.37	−59.8	−51.0
85000	16.65	6.55	3.22	−59.3	−50.7
86000	15.89	6.26	3.07	−58.7	−50.4
87000	15.17	5.97	2.93	−58.2	−50.1
88000	14.49	5.70	2.80	−57.6	−49.8
89000	13.83	5.45	2.67	−57.1	−49.5
90000	13.21	5.20	2.55	−56.5	−49.2
91000	12.61	4.97	2.44	−56.0	−48.9
92000	12.05	4.74	2.33	−55.4	−48.6
93000	11.51	4.53	2.22	−54.9	−48.3
94000	10.99	4.33	2.13	−54.4	−48.0
95000	10.50	4.13	2.03	−53.8	−47.7
96000	10.03	3.95	1.94	−53.3	−47.4
97000	9.58	3.77	1.85	−52.7	−47.1
98000	9.15	3.60	1.77	−52.2	−46.8
99000	8.75	3.44	1.69	−51.6	−46.5
100000	8.36	3.29^{-1}	1.62^{-1}	−51.1	−46.2
101000	7.99	3.14	1.54	−50.6	−45.9
102000	7.63	3.01	1.48	−50.0	−45.6
103000	7.29	2.87	1.41	−49.5	−45.3
104000	6.97	2.75	1.35	−48.9	−45.0
105000	6.66	2.62	1.29	−48.4	−44.7
106000	6.37	2.51	1.23	−47.4	−44.1
107000	6.09	2.40	1.18	−45.8	−43.2
108000	5.82	2.29	1.13	−44.3	−42.4
109000	5.57	2.19	1.08	−42.8	−41.6
110000	5.33	2.10	1.03	−41.3	−40.7
120000	3.45	1.36	6.67^{-2}	−26.1	−32.3
130000	2.27	8.92^{-2}	4.38	−10.9	−23.8
140000	1.51	5.95	2.92	+4.3	−15.4
150000	1.02	4.02	1.97	+19.4	−7.0
160000	6.97^{-1}	2.75	1.35	+27.5	−2.5
170000	4.78	1.88	9.23^{-3}	+27.5	−2.5
180000	3.26	1.28	6.31	+18.9	−7.3
190000	2.21	8.70^{-3}	4.27	+8.1	−13.3
200000	1.48	5.85	2.87	−2.7	−19.3
210000	9.85^{-2}	3.88	1.91	−22.0	−30.0
220000	6.41	2.52	1.24	−43.5	−41.9
230000	4.08	1.60	7.88^{-4}	−64.9	−53.9
240000	2.53	9.95^{-4}	4.89	−86.4	−65.8
250000	1.53	6.01	2.95	−107.8	−77.7
260000	8.92^{-3}	3.51	1.73	−129.3	−89.6

TABLE 2-2. Continued.

Pressure				*Temperature*	
Alt (Feet)	*Torr*	*in.Hg*	*PSIA*	*°F.*	*°C.*
270000	5.09	2.00	9.85^{-5}	−134.5	−92.5
280000	2.90^{-3}	1.14^{-4}	5.62^{-5}	−134.5	−92.5
290000	1.66	6.52^{-5}	3.20	−134.5	−92.5
300000	9.49^{-4}	3.74	1.84	−126.8	−88.2
350000	8.52^{-5}	3.35^{-6}	1.65^{-6}	−24.5	−31.4
400000	1.60	6.30^{-7}	3.10^{-7}	233.9	112.2
450000	6.31^{-6}	2.48	1.22	734.1	390.1
500000	3.50	1.38	6.78^{-8}	1203.8	651.0
600000	1.50	5.92^{-8}	2.91	1647.2	897.3
700000	7.42^{-7}	2.92	1.44	1835.7	1002.1
800000	3.95	1.56	7.64^{-9}	1964.3	1073.5
900000	2.22	8.74^{-9}	4.29	2053.4	1123.0
1000000	1.30	5.13	2.52	2124.6	1162.5
1100000	7.92^{-8}	3.12	1.53	2160.3	1182.4
1200000	4.96	1.95	9.59^{-10}	2189.3	1198.5
1300000	3.19	1.25	6.16	2214.6	1212.5
1400000	2.10	8.25^{-10}	4.05	2217.2	1214.0
1500000	1.40	5.52	2.71	2221.2	1216.2
1600000	9.55^{-9}	3.76	1.85	2232.1	1222.3
1700000	6.61	2.60	1.28	2233.7	1223.1
1800000	4.62	1.82	8.93^{-11}	2232.9	1222.7
1900000	3.26	1.29	6.31	2241.4	1227.4
2000000	2.33	9.17^{-11}	4.50	2250.8	1232.7

TABLE 2-3. PULMONARY GASES AT EQUIVALENT ALTITUDES WHEN BREATHING AIR OR PURE OXYGEN.

Equivalent Altitudes		*Breathing*	*Tracheal Inspired* Po_2 *mm Hg*	*Alveolar*		*R**
Feet	*mm Hg*			Po_2 *mm Hg*	Pco_2 *mm Hg*	
Sea Level	760	air	149	103	40	.85
34,000	188	oxygen	141	101	40	
5,000	632	air	123	80	38	.87
36,500	167	oxygen	120	82	38	
10,000	523	air	100	61	36	0.90
39,500	145	oxygen	98	62	36	
15,000	429	air	80	46	33	0.95
42,000	128	oxygen	81	48	33	
18,000	380	air	70	38	31	0.98
44,000	117	oxygen	70	39	31	
20,000	350	air	64	34	30	1.00
45,000	111	oxygen	64	34	30	
22,000	321	air	57	30	28	1.05
46,000	106	oxygen	59	30	29	

* R = Respiratory Exchange Ratio ($\dot{V}co_2/\dot{V}o_2$)

and falls below the limit that maintains the body in a physiologically safe condition.

For the unacclimatized man, an alveolar oxygen tension of less than 50 mm Hg is considered as approaching a severe state of hypoxia and an oxygen tension of 30 mm Hg is not adequate for supporting consciousness, and collapse is imminent. Theoretically, at a barometric pressure of 87 mm Hg (50,000 feet), with a normal carbon dioxide tension in the lungs of 40 mm Hg plus the water vapor tension of 47 mm Hg, even when breathing pure oxygen, the alveolar oxygen tension is reduced to zero and approaches a true state of anoxia. At 63,000 feet where the barometric pressure is 47 mm Hg, the lungs are completely filled with water vapor, theoretically, leaving no available room for other gases. Actually, under such a condition as this, not only is there the theoretical tendency for the body fluids and venous blood to boil, but outgassing of all dissolved gases in the venous blood, including oxygen, carbon dioxide and nitrogen will proceed outward at a vigorous rate through the lungs. This outgassing process becomes most extreme under conditions of a vacuum, such as in space flight conditions where the ambient barometric pressure is essentially zero. The term *ebullism* has been suggested for this unusual boiling phenomenon and unique medical syndrome.

The pressure of oxygen in the alveoli varies with the percentage of oxygen in the inspired air and the barometric pressure and, consequently, is subject to variations as either of these two factors changes. The carbon dioxide tension will decrease as the individual begins to hyperventilate with the onset of hypoxia, but the range of variation is small as compared to the extensive changes in alveolar oxygen tension. The water vapor in the alveolar air remains constant. At sea level, the alveolar partial pressures when breathing air are as follows:

P_{O_2} = 103 mm Hg;
P_{CO_2} = 40 mm Hg;
P_{H_2O} = 47 mm Hg; and
P_{N_2} = 570 mm Hg.

The equation for calculating the alveolar oxygen tension in mmHg, when inspired carbon dioxide is essentially zero, is:

$$P_{A_{O_2}} = (P_B - 47)\, F_{I_{O_2}} - P_{A_{CO_2}} \left(F_{I_{O_2}} + \frac{1 - F_{I_{O_2}}}{R}\right)$$

Where $P_{A_{O_2}}$ = Alveolar oxygen tension
P_B = ambient barometric pressure
$F_{I_{O_2}}$ = fraction of inspired oxygen (0.209 for air)
$P_{A_{CO_2}}$ = alveolar carbon dioxide tension
R = respiratory exchange ratio
47 = vapor tension of water 37° C.

When R is unity, this equation reduces to:

$$P_{A_{O_2}} = (P_B - 47)\, F_{I_{O_2}} - P_{A_{CO_2}}$$

When breathing 100% oxygen at any altitude, the above equation resolves to a simpler form:

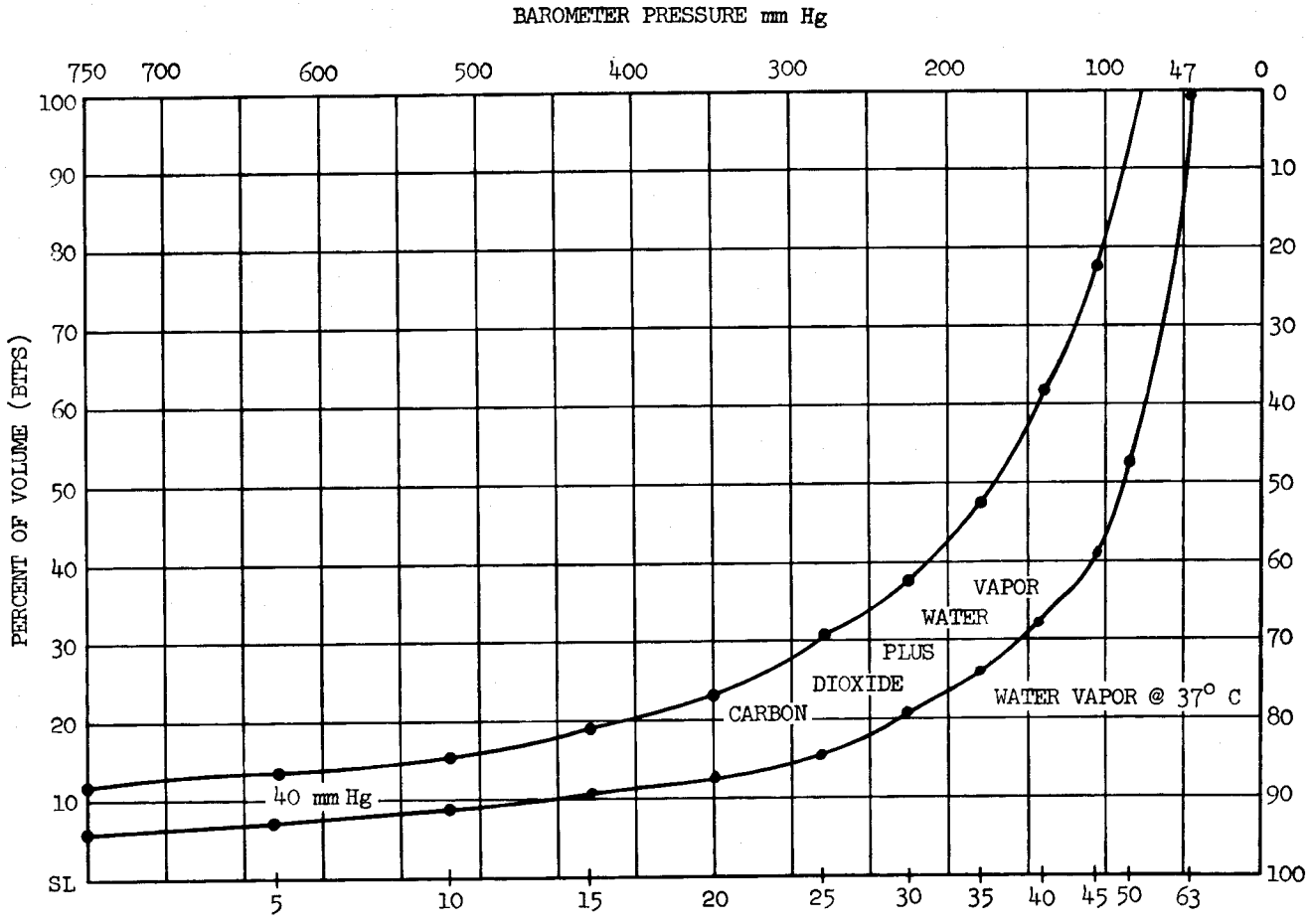
$$P_{A_{O_2}} = (P_B - 47) - P_{A_{CO_2}}$$

At sea level, the combined pressure of P_{CO_2} and P_{H_2O} is 87 mm Hg and occupies $\frac{87}{760}$ or 11% of the lung volume; oxygen occupies $\frac{103}{760}$ or 14%; and nitrogen, 75%.

At 18,000 feet, P_{CO_2} equals 31 mm Hg, P_{H_2O} remains 47 mm Hg, and the barometric pressure is 380 mm Hg. At this level $\frac{31 + 47}{380}$ or 21% of the lung volume is occupied by carbon dioxide and water vapor.

Figure 2–3 shows the increasing percentage of the lung volume occupied by water vapor and by both water vapor and carbon dioxide with decreasing barometric pressures (in which it is assumed that the P_{CO_2} remains constant at 40 mm Hg). It can be seen that water vapor alone occupies about 50% of the lung volume at an altitude of 47,000 feet and, without hyperventilation at this altitude, less than 15% of the volume is available for oxygen.

Between 30,000 and 40,000 feet, the automatic pressure-demand oxygen regulators (MD–1 or MD–2) are designed to deliver 100% oxygen under a slight "safety" pressure (3 to 4 mm Hg) to prevent inboard mask leakage. At altitudes above 40,000 feet, the positive pressure delivered to the mask increases with increasing altitude, as shown in table 2–4.



PERCENT OF VOLUME FOR AIR OR OXYGEN

	ALT IN THOU	PER VOL BTPS $CO_2+H_2O(g)$
1.	SL	12
2.	5	14
3.	10	16
4.	15	20
5.	20	23
6.	25	31
7.	30	38
8.	35	48
9.	40	62
10.	45	78
11.	50	100

	ALT IN THOU	PER VOL BTPS $H_2O(g)$ @ 37° C
1.	SL	6
2.	5	7
3.	10	9
4.	15	11
5.	20	13
6.	25	16
7.	30	21
8.	35	26
9.	40	33
10.	45	41
11.	50	54
12.	63	100

The percent of the lung volume occupied by water vapor and carbon dioxide at various altitudes, assuming a constant alveolar carbon dioxide tension of 40 mm Hg.

Figure 2–3. Lung Volume Occupied by Water Vapor and Carbon Dioxide at Various Altitudes.

TABLE 2–4. TRACHEAL PARTIAL PRESSURE OF OXYGEN, PRESSURE BREATHING 100 PERCENT OXYGEN.

Altitude (Feet)	*Barometric Pressure (mm Hg)*	*Tracheal Po_2 100% O_2 (mm Hg)*	*Breathing Pressure Using Standard Air Force Oxygen Equipment (mm Hg)*	*Tracheal Po_2 Using Standard Air Force Oxygen Equipment*
40,000	141	94	4–8	98–102
43,000	122	75	11–14	86–89
45,000	111	64	15–18	79–81
48,000	96	49	23	72
50,000	87	40	30	70

It can be noted that, at 50,000 feet, a positive pressure of about 30 mm Hg is transmitted to the mask, resulting in a tracheal Po_2 of about 70 mm Hg, provided there is no excessive mask leakage. This is equivalent to breathing oxygen at 44,000 feet or breathing air at 18,000 feet. In all of these cases, this represents a severe degree of hypoxia, being compounded still further at 50,000 feet by the high degree of unsupported pressure breathing and its effect on the cardiovascular system. For these reasons, this type of pressure breathing at altitudes above 45,000 feet provides inadequate protection except for brief periods, in extreme emergencies, followed by immediate descent. For adequate protection, the pressure suit is mandatory to effectively bring the equivalent altitude for pulmonary oxygenation below 40,000 feet.

Oxygen Transport and Tissue Phase of Respiration

The quantity of gas that goes into solution, temperature remaining constant, is dependent on its solubility characteristics and is proportional to the partial pressure of the gas concerned (Henry's Law). However, far greater quantities of oxygen and carbon dioxide are carried in the blood than can be present in simple solution in the plasma. At sea level, when air is breathed, only 0.24 cc of oxygen and 2.5 cc of carbon dioxide is carried in 100 cc of blood in simple solution. Under the same conditions, however, 100 cc of blood actually contains about 18 to 20 cc of oxygen and 40 to 50 cc of carbon dioxide. This is 100 times the amount of oxygen and 20 times the amount of carbon dioxide that can be carried in simple solution. The ability of the blood to carry such a great load of oxygen is due to the hemoglobin contained in the red blood cells. Carbon dioxide is carried largely in the form of bicarbonate ions in the plasma and in the red blood cells.

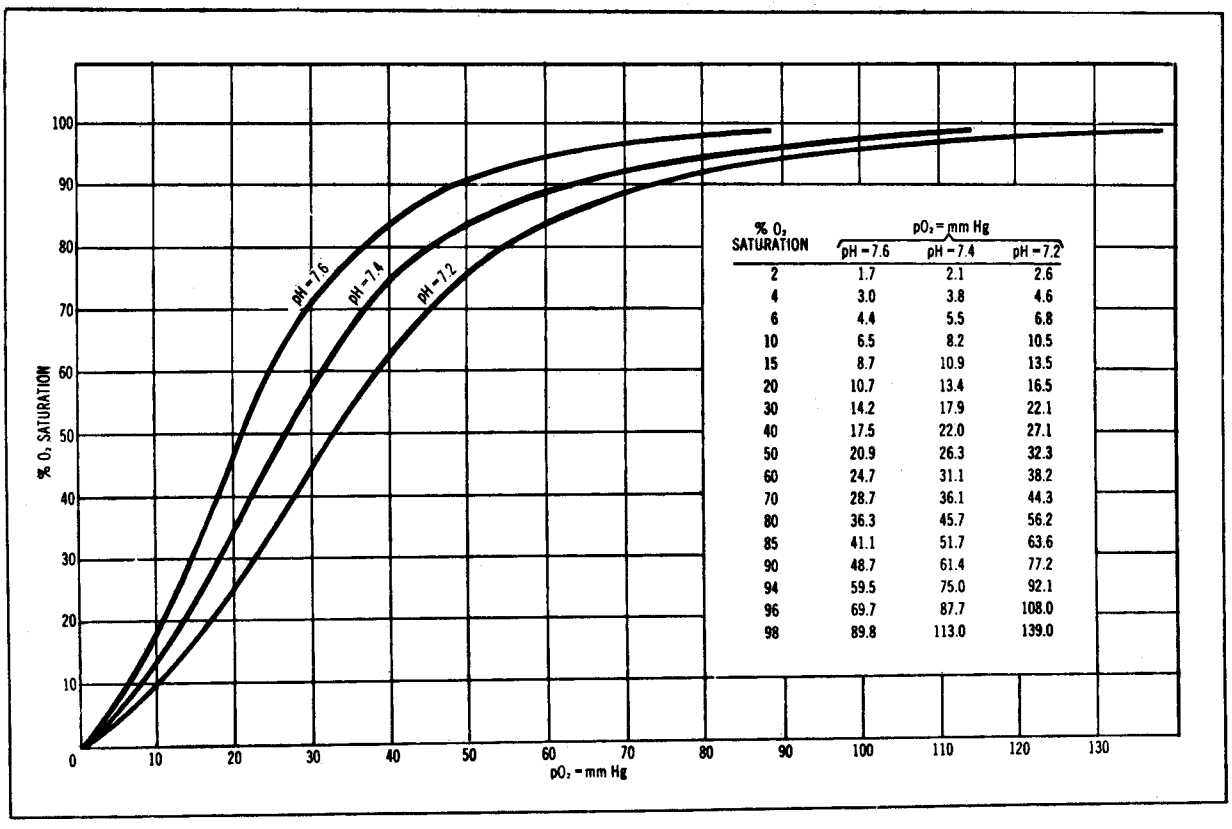
Oxygen combines reversibly with hemoglobin in a unique manner to form oxyhemoglobin. This is discussed in connection with the curves of dissociation of oxyhemoglobin in figure 2–4.

The combination of hemoglobin with oxygen is influenced by the partial pressure of oxygen in the surrounding medium. This has a direct effect on the amount of oxygen delivered to the tissues of the body at various altitudes.

It has been noted that, as the partial pressure of oxygen is lowered, little oxygen is released from hemoglobin until a partial pressure of 60 mm Hg or less is attained (figure 2–4, O_2 dissociation curve, pH 7.4). At this point, a more rapid evolution of oxygen commences and continues until 0 mm Hg is reached. The same observation can be made in the reverse direction—*i.e.*, the greatest absorption takes place between 0 to 40 mm Hg.

The oxygen-carrying capacity of hemoglobin is very sensitive to changes in the pH and Pco_2 of the blood (Bohr effect) as is apparent in figure 2–4.

For example, at an oxygen tension of 50 mm Hg, pH 7.2, the oxygen saturation is 75%, while at pH 7.6, the oxygen saturation is 90%. Carbon dioxide has a major influence



% O_2 SATURATION	pO_2 - mm Hg		
	pH - 7.6	pH - 7.4	pH - 7.2
2	1.7	2.1	2.6
4	3.0	3.8	4.6
6	4.4	5.5	6.8
10	6.5	8.2	10.5
15	8.7	10.9	13.5
20	10.7	13.4	16.5
30	14.2	17.9	22.1
40	17.5	22.0	27.1
50	20.9	26.3	32.3
60	24.7	31.1	38.2
70	28.7	36.1	44.3
80	36.3	45.7	56.2
85	41.1	51.7	63.6
90	48.7	61.4	77.2
94	59.5	75.0	92.1
96	69.7	87.7	108.0
98	89.8	113.0	139.0

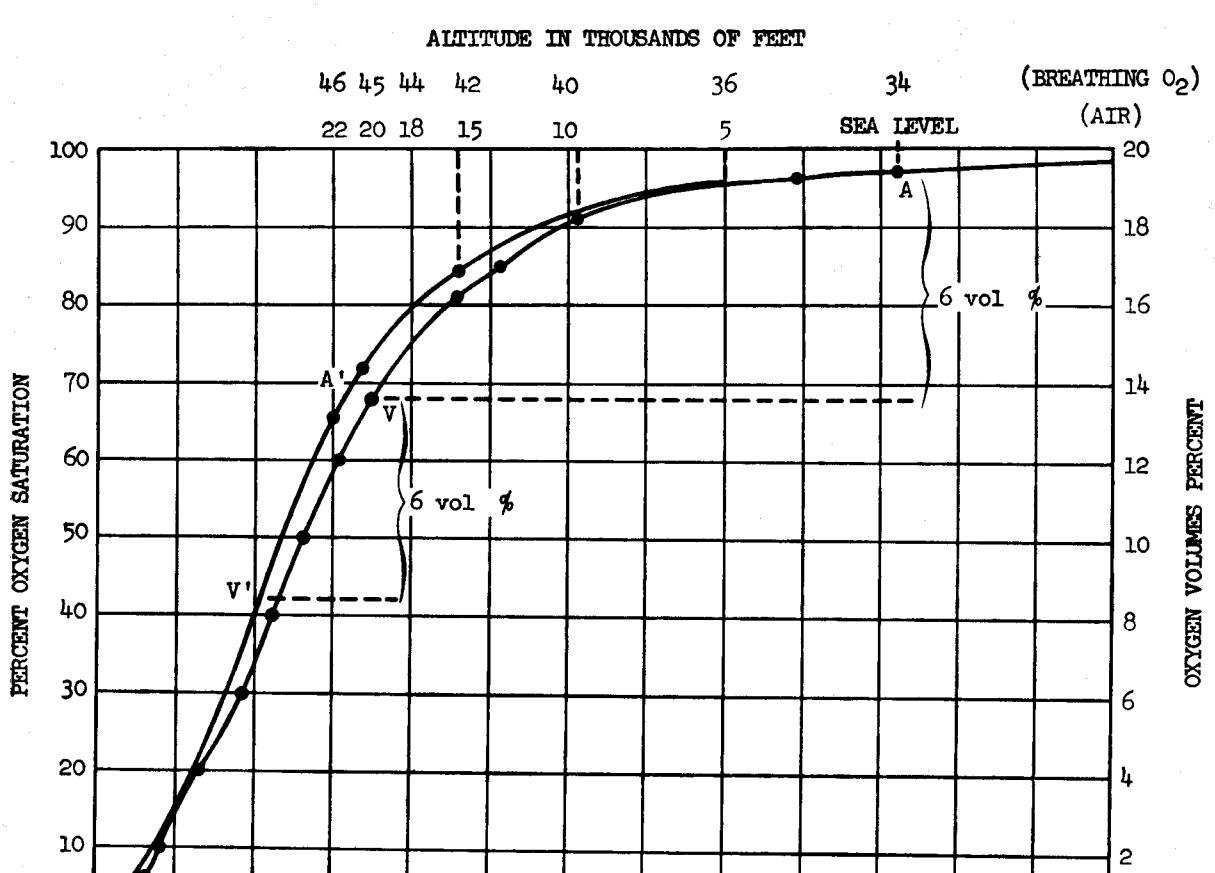
Figure 2–4. Oxygen Dissociation Curves for Human Blood.

on blood pH and on the oxygen dissociation curve. An increase in P_{CO_2} shifts the curve to the right toward the acid side (low pH) and, conversely, a low P_{CO_2} shifts the curve to the left toward the alkaline side (high pH).

Because of this, there must be a slight shift of the oxygen dissociation curve as blood passes through the arteries and veins between the lungs and the tissues. In the lungs, the uptake of oxygen and the release of carbon dioxide shift the curve to the alkaline side, permitting good oxygen loading. In the tissues, the uptake of carbon dioxide and release of oxygen tend to shift the curve to the acid side, favoring the unloading of oxygen at a higher oxygen tension. During muscular exercise, with an increase in carbon dioxide and acidity at the tissue level, this effect should provide a physiologic advantage for the delivery of oxygen. It has been shown, however, that the dilation and opening up of muscle capillaries and increased blood flow to the active muscles are much more significant in this respect.

Likewise, an increase in blood temperature shifts the curve to the right, again favoring the unloading of oxygen at metabolically active organs and tissues, while a decrease in blood temperature moves the dissociation curve to the left side of the diagram. This temperature effect can be an important consideration under conditions of hypo- or hyperthermia, cold or frozen extremities, etc.

The relationship between the oxygen dissociation curve, arterial and venous oxygen saturations, and equivalent altitudes in terms of the alveolar oxygen and carbon dioxide tensions is shown in figure 2–5 where the values from table 2–3 have been used. The normal oxygen dissociation curve (pH 7.4) indicates the arterial and venous points under sea level conditions and shows that,



OXY PRESS	OXY SAT
V	
1. 0	0
2. 2	2
3. 4	4
4. 6	6
5. 8	10
6. 13	20
7. 18	30
8. 22	40
9. 26	50
10. 31	60
11. 35	68
12. 46	81
13. 52	85
14. 61	91
15. 88	96
16. 103	97
17. 130	98

OXY PRESS	OXY SAT
V'	
1. 0	0
2. 30	64
3. 34	72
4. 46	84
5. 61	91
6. 88	96
7. 103	97
8. 130	98

The figure demonstrates the normal dissociation curve (pH 7.4) and the points on the shifted dissociation curve at various equivalent altitudes, breathing air or oxygen. The volumes percent scale is based on a blood oxygen capacity of 20 volumes percent (data from table 2-3).

Figure 2-5. Oxygen Dissociation Curves.

from arterial (A) to venous blood (V), the saturation decreases to approximately 70% with a decrease in oxygen content of about 6 volumes percent. At 20,000 feet while breathing air, or at 45,000 feet while breathing oxygen, the dissociation curve shifts slightly to the left because of the hypoxic hyperventilation which decreases carbon dioxide and increases pH. Here, the arterial saturation (A) is about the same as venous blood under sea level conditions. To deliver six volumes percent of oxygen to the tissues, blood flow remaining constant, the blood saturation must decrease to almost 40% saturation (V) with an oxygen tension of not much more than 20 mm Hg. By increasing the blood flow, *for example,* by doubling the cardiac output, the same quantity of oxygen can be delivered to the tissues with a net effect of decreasing the oxygen content by three volumes percent and raising the venous oxygen saturation and tension to about 55% and 26 mm Hg, respectively. Both the cardiovascular and ventilatory responses to hypoxia are important compensatory adjustments.

Furthermore, figure 2–5 shows that, at the critical hypoxia levels above 18,000 feet while breathing air and 44,000 feet while breathing oxygen, the arterial saturation points lie on the steep portion of the dissociation curve where the oxygen tensions are between 30 and 40 mm Hg. It is in this region of the curve that even a small decrease in the barometric pressure can result in a striking decrease in the arterial oxygen saturation. This is particularly true when breathing pure oxygen above 44,000 feet, for here each mm Hg change in the barometric pressure represents the same change in the oxygen tension, and a change of only 2 to 3 mm Hg can be the difference between consciousness and unconsciousness. This same concept holds true when pressure breathing at these or higher altitudes with a tight or leaky oxygen mask. Under these conditions, every mm Hg of oxygen counts, and even a slight drop in the mask pressure will immediately cause the arterial oxygen saturation to slide sharply down the dissociation curve to dangerously low levels. It is interesting to compare this type of situation with a change in altitude from sea level to 10,000 feet, *for example,* breathing air. In this case, a decrease of 40 mm Hg in oxygen tension has only a slight effect on the arterial oxygen saturation which decreases to about 90%. This important characteristic permits man to function effectively in a fairly broad pressure environment and is the very basis for the design of cabin pressurization systems permitting considerable latitude in the selection and control of pressure differentials for the cabin.

Figure 2–6 is a composite picture of both phases of respiration at rest, with the values of the gases involved at ground level. Hypoxia, discussed later, may result from a deficiency in either of the two phases of respiration.

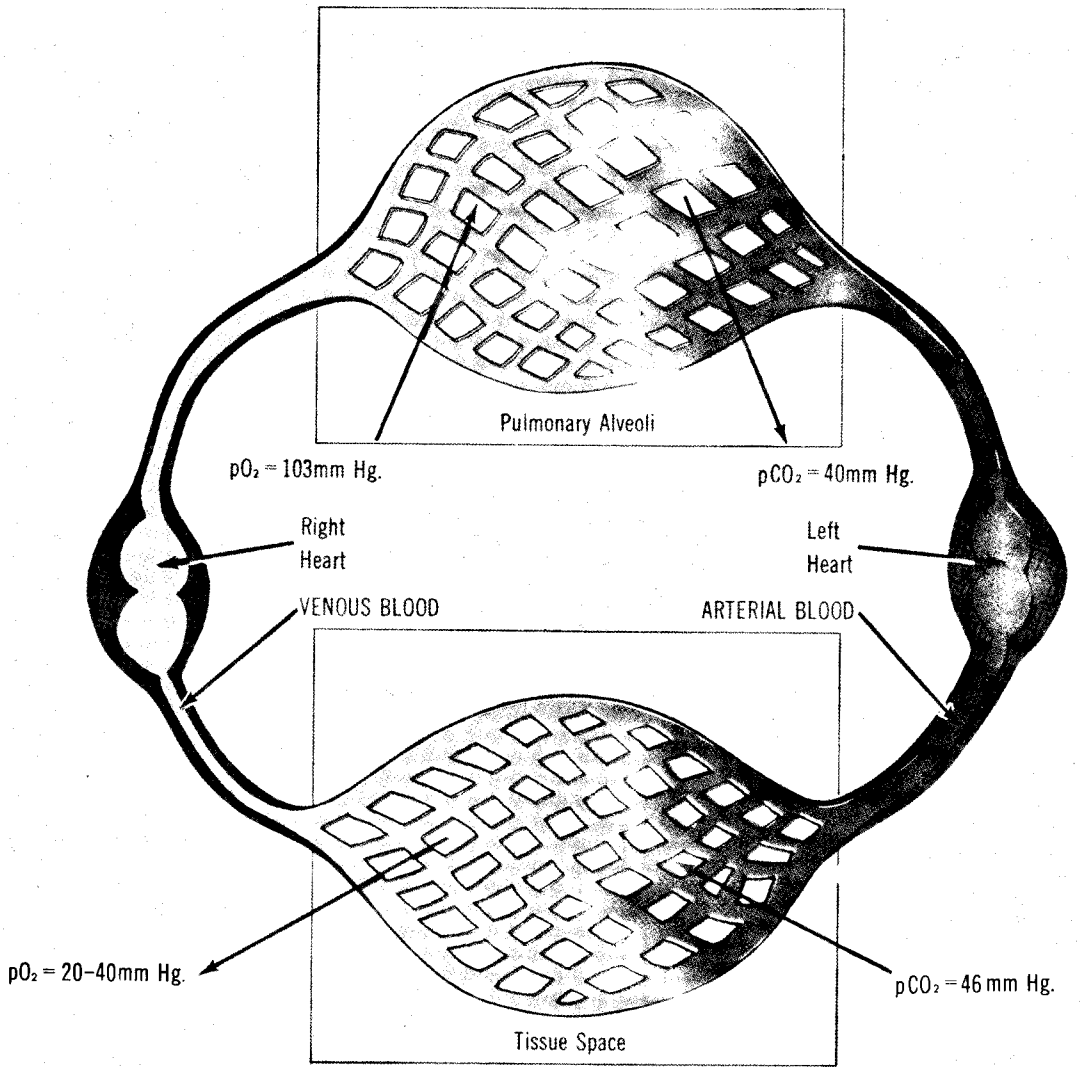
Control of Ventilation

The control of respiratory rate and depth is complicated, but two of the important factors that influence the process are the chemical and psychic stimuli. An individual at rest ordinarily breathes at a rate of 12 to 16 times per minute with no conscious effort. The result is an exchange of 6 to 8 liters per minute (minute respiratory volume). With exercise, there is an increased demand for oxygen and for elimination of carbon dioxide. The body responds to this demand by an increase in ventilation and cardiac output. Figure 2–7 illustrates the changes in ventilation during work at ground level and altitudes up to 40,000 feet, while the subject breathes from an oxygen system.

When hypoxia is not present, the minute respiratory volume does not change significantly with altitude up to 30,000 feet in the resting subject, but each increase in workload causes a rise in ventilation. Note that the curve remains rather constant for each workload up to 30,000 feet, and then there is a marked rise in ventilation between 30,000 and 40,000 feet. At 40,000 feet, even though 100% oxygen is breathed, the barometric pressure is so low that the alveolar oxygen

	Mixed Venous Blood
O_2	
Content	14 vol %
Tension	36mm Hg
Physical Sol	0.1 vol %
CO_2	
Content	54 vol %
Tension	46mm Hg
Physical Sol	3.0 vol %
Hemoglobin	
Content	15 gram %
Oxy Hgb	70%
Hgb	30%

	Arterial Blood
O_2	
Content	19 vol %
Tension	100mm Hg
Physical Sol	0.24 vol %
CO_2	
Content	49 vol %
Tension	40mm Hg
Physical Sol	2.5 vol %
Hemoglobin	
Content	15 gram %
Oxy Hgb	96%
Hgb	4%



Mechanics of gas exchange - Internal and external respiration

Figure 2-6. Phases of Respiration.

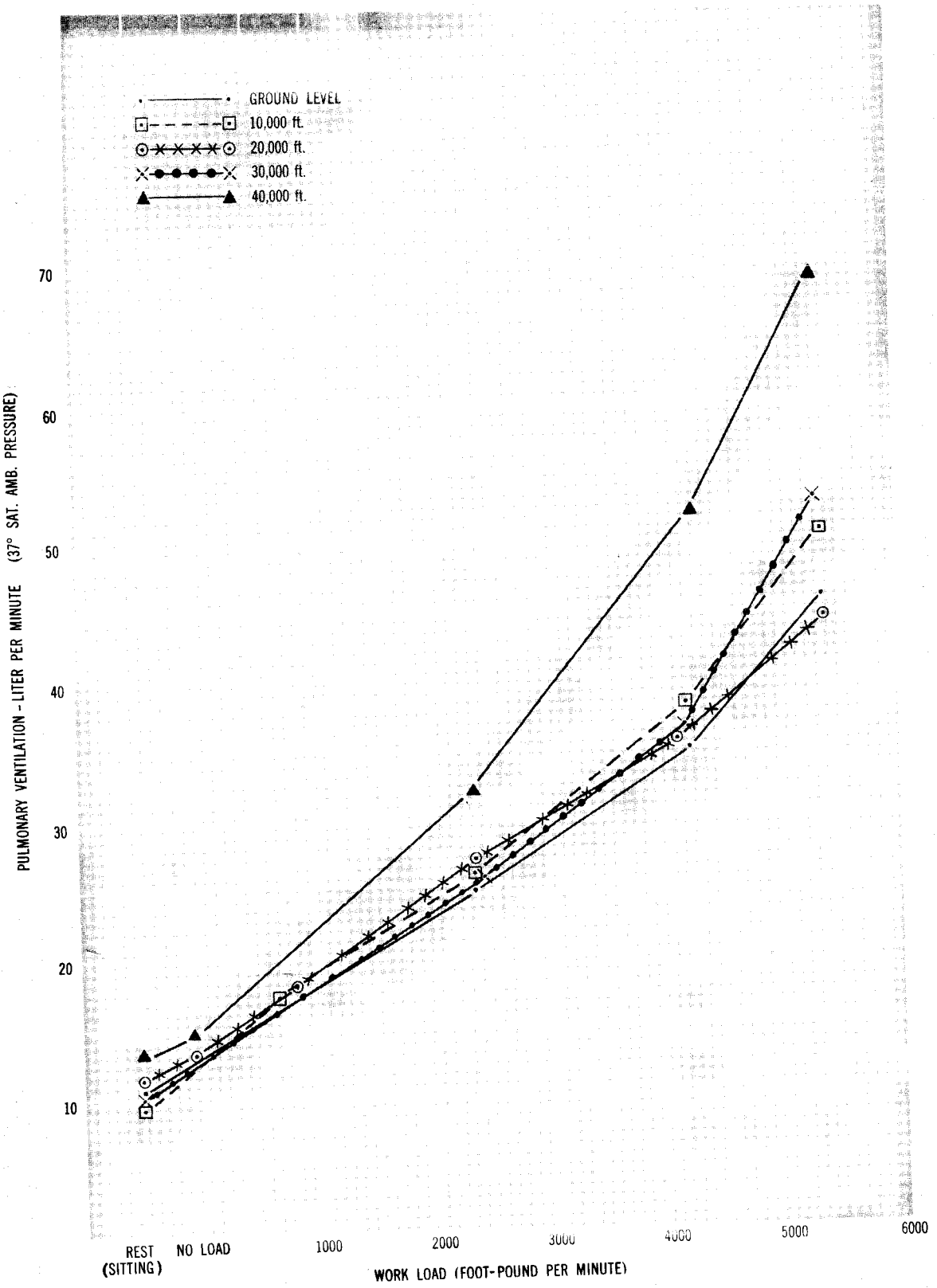


Figure 2–7. The Relation of Ventilatory Volume to Work at Various Altitudes.

tension is the same as at 10,000 feet while breathing ambient air. The disproportionate rise in minute respiratory volume for a given workload at 40,000 feet with the subject breathing 100% oxygen can be related to hypoxia.

Measurements taken on bomber crews and fighter pilots during flight showed minute respiratory volumes in the range of 7 to 15 liters per minute during periods of relative inactivity, the average being 12 liters per minute. When the subjects were active, the measurements ranged from 12 to 60 liters per minute, with an average of 25 liters per minute.

While breathing air at sea level, the partial pressure of oxygen in the arterial blood is normally about 100 mm Hg and oxygen lack (hypoxia) normally does not exist. The partial pressure of oxygen under this condition does not contribute to the control of respiration since the chemoreceptors in the aorta and carotid sinus primarily respond only to an abnormally low partial pressure of oxygen in the arterial blood. Instead, without hypoxia, the small fluctuations of carbon dioxide partial pressure, the pH of arterial blood, and the respiratory centers in the medulla play the major roles in regulating respiration under normal conditions at ground level. Not until the partial pressure of arterial oxygen has decreased below 80 mm Hg does a significant increase in ventilation begin while a person is at rest. This hypoxic stimulus for increased ventilation, however, results in the excessive blowing off of carbon dioxide which leads to hypocapnia, an increase in arterial pH, and an inhibitory effect on respiration. Thus, the net effect, even with severe hypoxia, is an increase in ventilation that is not nearly as great as it would be if the carbon dioxide and acid-base balance of the arterial blood were not concomitantly altered in a manner that antagonizes the hypoxic stimulus.

For the unacclimatized man, with ascent to altitudes above 8,000 feet while at rest, the oxygen tension in the inspired air is reduced sufficiently so that hypoxia begins to be a significant stimulus to respiration. Under conditions of exercise, this effect is much more pronounced and begins at lower altitudes. At altitudes where hypoxia is critical, the combination of hypoxia and the concomitant hyperventilation response usually presents a clinical picture that is difficult to differentiate from the hyperventilation syndrome seen; *for example,* in states of anxiety under normal oxygen conditions at ground level. This is characterized by lightheadedness, palpitation, and paresthesia of the extremities and perioral area. If excessive hyperventilation persists, carpopedal spasms, mental disorientation, and unconsciousness may occur. Often, the individual is not aware of overbreathing and may complain of a sensation of smothering. Under this condition of hyperventilation, particularly where hypoxia is not the primary contributing factor, the decrease in arterial carbon dioxide tension, the shift in pH, and the severe state of respiratory alkalosis result in a marked decrease in cerebral blood flow (cerebral vasoconstriction) and a decrease in brain oxygenation, even though the alveolar and arterial oxygen tensions may be high.

Excessive hyperventilation during flight can lead to serious and, possibly, disastrous consequences. On the other hand, under conditions of acute hypoxia, without the normal hypoxic stimulus for hyperventilation, man's altitude ceiling and the threshold for hypoxic unconsciousness, when breathing air, would be decreased from approximately 22,000 feet to 16,000 feet. As shown in table 2-3, the alveolar oxygen tension at 22,000 feet is approximately 30 mm Hg where unconscious collapse is imminent. At the same time, the alveolar carbon dioxide tension is reduced to about 28 mm Hg by the increased pulmonary ventilation. It can be shown by the alveolar equation that, if the ventilation did not increase and the alveolar carbon dioxide tension remained at 40 mm Hg, the alveolar oxygen tension would be reduced to 30 mm Hg at a barometric pressure of 412 mm Hg or 16,000 feet. In hypoxic situations such as this, a moderate hyperventilation response is not only normal and desirable but, in

critical instances, it can be the margin between consciousness and unconsciousness. Also, adaptation to chronic hyperventilation, with renal readjustment of the acid-base balance back toward normal, is one of the key factors in man's ability to acclimatize and to work and live at high altitudes. Thus, there are important advantages as well as serious disadvantages in hyperventilation, depending on the circumstances. The hyperventilation syndrome is most undesirable and dangerous when there is no physiologic need for it to increase the arterial oxygen tension, such as psychogenic hyperventilation induced by apprehension, fear, and anxiety. With proper indoctrination and training in the use of oxygen equipment, most hypoxic situations can be avoided except in unforeseen or unavoidable emergencies, and a vital physiologic requirement for hyperventilation should not occur normally when good oxygen discipline is maintained.

The Pattern of Respiration and Its Significance

Inhalation in the normal resting subject is an active movement, while exhalation is almost wholly passive. During exercise, not only do the rate and volume of respiration change, but so does the pattern. This difference is illustrated in figure 2–8. During a single inspiration of a resting individual, the "instantaneous flow rate" increases from zero, at the beginning, to 20 or 30 liters per minute near the midpoint of inspiration and returns to zero at the end. An individual who is exercising moderately may have a respiratory volume of 25 to 45 liters per

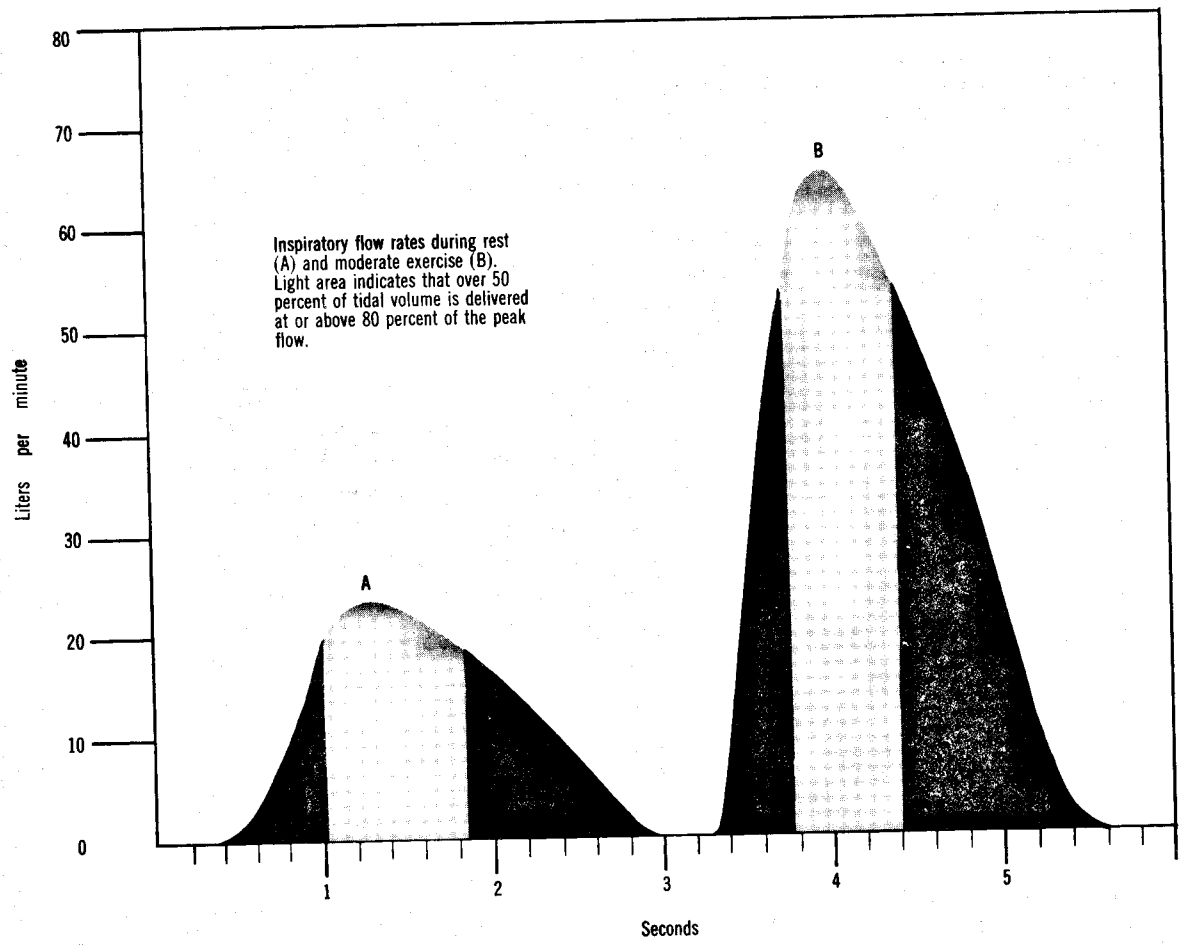


Figure 2–8. Changes in Respiratory Rate and Volume During Rest and Moderate Exercise.

minute and an instantaneous flow rate as high as 65 to 90 liters per minute. In general, to obtain the approximate maximal inspiratory or peak flow rates, minute respiratory volume (stated at STP) may be multiplied by 3.7 for a subject at rest and by 2.8 for an individual who is exercising.

Hypoxia

Altitude sickness in aviation is a syndrome that is usually acute and results from inadequate oxygenation of tissues secondary to a decreased partial pressure of oxygen in the inspired air. A syndrome, anoxia, meaning literally "without oxygen," is sometimes erroneously used to denote a deficiency rather than a lack of oxygen in the tissues. At altitudes below 55,000 feet, it is more exact to use the term "hypoxia," for even in such cases of acute altitude sickness, the tissues are never entirely without oxygen. At increasingly higher altitudes above 50,000 feet, however, tissue hypoxia verges into

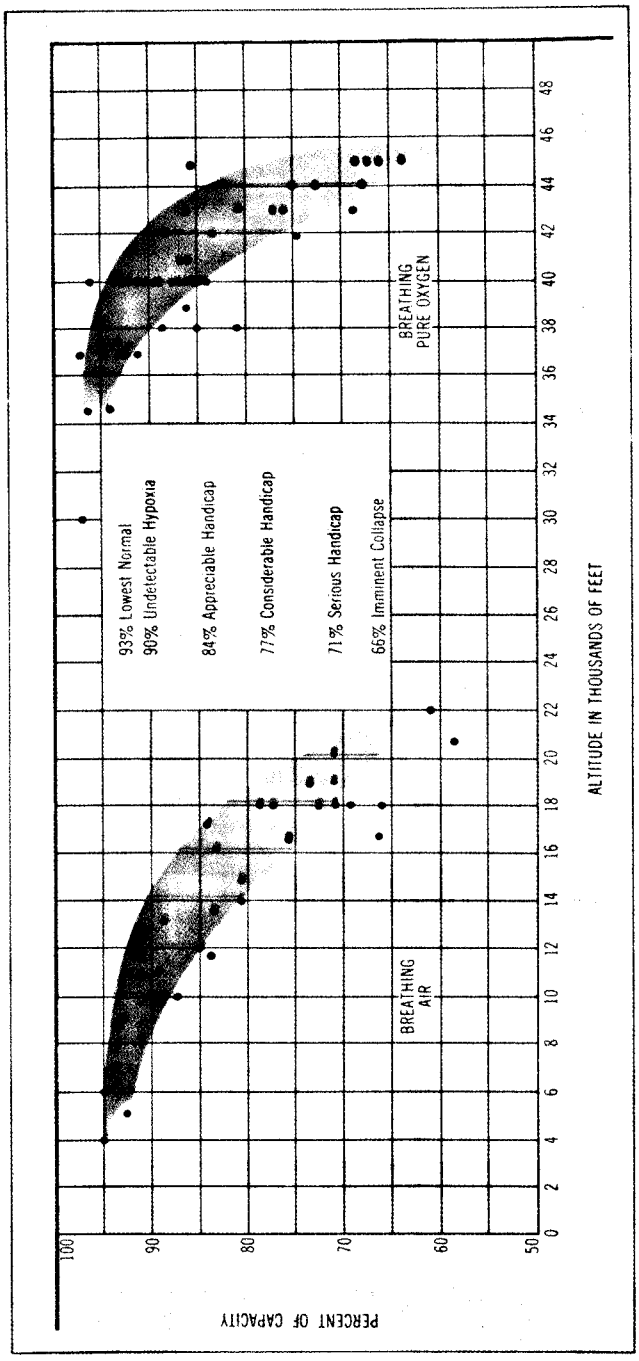


Figure 2–9. Oxygen Saturation (Percent of Capacity) of Arterial Blood and Range of Performance at Various Altitudes in Subjects Breathing Air and in Subjects Breathing Oxygen.

essentially true anoxia, regardless of whether ambient air or pure oxygen is being breathed.

a. Classifications of Hypoxia. Generally, there are four different classifications of hypoxia: *hypoxic, hypemic* or *anemic, histotoxic,* and *stagnant* hypoxia.

(1) *Hypoxic hypoxia* is caused by a decrease of the O_2 pressure in the inspired air or in the lungs, or by conditions which prevent or interfere with the diffusion of O_2 across the alveolar membrane. Examples of hypoxic hypoxia are: (a) A reduced atmospheric pressure and, therefore, a reduced alveolar Po_2 at altitude; (b) interference with respiration, as in asthma (where the tracheal or bronchial cartilage is constricted, thereby impeding proper lung ventilation), in pneumonia (where the collection of fluid in the alveoli hinders O_2 diffusion across the alveolar-capillary membrane), and in obstruction of air passages, such as by tumors or strangulation; (3) arterial venous shunts, as in congenital cardiovascular cases (see figure 2–9).

(2) *Hypemic or anemic hypoxia* is caused by a reduction in the capacity of the blood to carry a sufficient amount of oxygen because of a decreased hemoglobin content. *For example,* 1 Gm of hemoglobin normally carries 1.34 cc of oxygen, and a normal healthy man has the capacity to transport 20 cc of oxygen per 100 cc of blood. If the same individual were wounded and his hemoglobin reduced by one-half, due to blood loss, then he could transport only 10 cc of oxygen per 100 cc of blood. In the latter instance, there may be insufficient oxygen for the tissues, even though the blood is fully saturated with oxygen and no cyanosis is present. For cyanosis to occur, over 6 Gm of reduced hemoglobin per 100 cc of blood must be present in the capillaries of the skin. Carbon monoxide, nitrites, sulfa drugs, etc., cause the same type of hypoxia by forming compounds with hemoglobin and reducing the amount of hemoglobin available to form oxyhemoglobin.

(3) *Histotoxic hypoxia* ensues when the utilization of O_2 by the body tissues is interfered with. Alcohol, narcotics, and certain poisons, such as cyanide, interfere with the ability of the cells to make use of the O_2 available to them, even though the supply is normal in all respects. During histotoxic hypoxia, the venous O_2 HHb saturation is higher than normal because the O_2 is not being unloaded at the tissues. The tissues are unable to metabolize the delivered O_2.

(4) *Stagnant hypoxia,* like hypemic hypoxia, is due to a malfunction of the circulatory system, but differs in certain respects. While the oxygen-carrying capacity of the blood is adequate, there is an inadequate circulation of the blood. Such conditions as heart failure, arterial spasm, occlusion of a blood vessel, and the venous pooling encountered during positive G maneuvers and pressure breathing would predispose to stagnant hypoxia.

It is evident that all of these forms of hypoxia may become problems in flight. However, the most frequent and important type of hypoxia encountered is that caused by breathing air with a low partial pressure of oxygen. The result is the syndrome of "mountain" or "altitude" sickness.

Symptomatology of Hypoxia

The appearance of the signs and the severity of the symptoms of acute hypoxic hypoxia depends upon the following variables:

a. Absolute altitude.
b. Rate of ascent.
c. Duration at altitude.
d. Ambient temperature.
e. Physical activity.
f. Individual factors:
   (1) Inherent tolerance.
   (2) Physical fitness.
   (3) Emotionality.
   (4) Acclimatization.

Although it might seem that the higher the altitude the more marked the symptoms, it has been observed that, at rapid rates of ascent, still higher altitudes can be reached for brief periods of time before serious symptoms appear. Length of exposure is an important variable. Thus, while an altitude

of 18,000 feet can be tolerated by most healthy persons for 30 minutes, severe symptoms may appear much sooner. A high surrounding temperature and physical exertion favor the development of symptoms at lower altitudes. Physical fitness and acclimatization from residence at high altitude raise an individual's "ceiling," while apprehension and lack of adequate physiological compensation by the respiratory and circulatory systems lower it.

For convenience, the symptomatology of hypoxia may be divided into stages related to the approximate pressure, the altitudes, and the oxygen saturation of the blood. As shown in table 2–5, the stages of hypoxia are:

(a) *Indifferent Stage.* The only adverse effect is on dark adaption, which is manifest at altitudes as low as 5,000 feet. It emphasizes the need for oxygen from the ground up during night flights, especially in the case of fighter pilots. Electrocardiographic changes may occur at altitudes as low as 5,000 feet; there is also an increase in the pulse rate.

(b) *Compensatory Stage.* Physiological compensations provide some defense against hypoxia so that effects are reduced unless the exposure is prolonged or unless exercise is undertaken. Respiration may increase in depth or slightly in rate, whereas the pulse rate, the systolic blood pressure, the rate of circulation, and the cardiac output increase.

(c) *Disturbance Stage.* In this stage, the physiological compensations do not suffice to provide adequate oxygen for the tissues—latent oxygen want becoming manifest. Subjective symptoms may include fatigue, lassitude, somnolence, dizziness, headache, breathlessness, and euphoria. Occasionally, there are no subjective sensations up to the time of unconsciousness. Objective symptoms include:

1. Special Senses. Both the peripheral and central vision are impaired and visual acuity is diminished. Extraocular muscles are weak and incoordinate, and the range of accommodation is decreased. Touch and pain are diminished or lost. Hearing is one of the last senses to be impaired or lost.

2. Mental Processes. Intellectual impairment is an early sign and makes it improbable for the individual to comprehend his own disability. Thinking is slow, and calculations of a navigator or bombardier are unreliable. Memory is faulty, particularly for events in the immediate past. Judgment is poor. Reaction time is delayed.

3. Personality Traits. There may be a release of basic personality traits and emotions as with alcoholic intoxication. There may be euphoria, elation, pugnaciousness, overconfidence, or moroseness.

4. Psychomotor Functions. Muscular coordination is decreased, and delicate or fine muscular movements may be impossible. This results in stammering, illegible handwriting, and poor coordination in aerobatics and in formation flying.

5. Hyperventilation Syndrome.

6. Cyanosis.

(d) *Critical Stage.* This is the stage in which consciousness is lost. This may be the result of circulatory failure ("fainter") or

TABLE 2–5. STAGES OF HYPOXIA.

Stage	*Altitude in Feet*		*Arterial Oxygen Saturation %*
	Breathing Air	*Breathing 100% Oxygen*	
Indifferent	0 to 10,000	34,000 to 39,000	95 to 90
Compensatory	10,000 to 15,000	39,000 to 42,500	90 to 80
Disturbance	15,000 to 20,000	42,500 to 44,800	80 to 70
Critical	20,000 to 23,000	44,800 to 45,500	70 to 60

of central nervous system failure ("non-fainter," unconsciousness with maintenance of blood pressure). The former is more common with prolonged hypoxia; the latter with acute hypoxia. With either type, there may be convulsions and eventual failure of the respiratory center, followed by cardiovascular failure and death.

Symptoms of Hypoxia

Unfortunately, man does not possess a built-in warning system to alert him to the onset of hypoxia and the danger of an hypoxic environment similar to the pain sensations of heat, cold, and certain noxious gases. On the contrary, hypoxia is painless, often resulting in a sense of well-being and the inability to recognize subjectively the onset of incapacitation. For these reasons, hypoxia in aviation is doubly insidious and dangerous.

Headache and lethargy are common complaints after a prolonged period of hypoxia. The headache is of general distribution, but is particularly acute in the frontal region. The best cure is sleep, but the administration of 100% oxygen is advisable if headache is severe. These symptoms have been explained on the basis of edema of the tissues, particularly the cerebral tissues, as a consequence of an increased permeability of the capillaries caused by the hypoxia. Nausea, vomiting, and severe prostration may also occur, but these usually clear up in 24 to 48 hours. Permanent cerebral damage resulting from altitude hypoxia has been comparatively rare, with only a few authenticated cases on record.

Individual variation in the ability to withstand hypoxia is considerable and accounts for variations in "ceiling." A large part of the tolerance is based on the adequacy of physiological adjustments, especially in breathing. The immediate result of deeper breathing is an increase in the pressure of oxygen in the lungs and increased alkalinity of the blood, owing to the hyperventilation effect. The latter favors uptake of oxygen by the hemoglobin. At such extreme altitudes as 40,000 feet where 100% oxygen must be breathed, the total pressure in the alveoli equals the sum of the partial pressures exerted by water vapor, carbon dioxide, and oxygen. The pressure of the water vapor is relatively constant, tending to correspond to a saturated state of 37°C. Consequently, lowering of the partial pressure of carbon dioxide, such as occurs in deep breathing, will increase the partial pressure of oxygen in the lungs by an approximately equivalent amount.

Inexperienced personnel collapse more frequently at intermediate altitudes than do experienced persons. The factors involved in such collapse are primarily psychogenic. The hyperventilation produced by hypoxia ordinarily lowers alveolar carbon dioxide enough to produce only minor symptoms, such as dizziness. However, a person who is apprehensive may hyperventilate to a greater extent and produce a degree of hypocapnia associated with more marked symptoms. Such hypocapnia, added to the splanchnic vasodilation, which is a frequent response to fear, may bring about collapse.

Prophylaxis and Treatment of Hypoxic Hypoxia

The treatment of hypoxia requires the administration of 100% oxygen by inhalation. If respiration has ceased, artificial respiration, along with the simultaneous use of 100% oxygen is indicated. If peripheral circulatory failure persists, the type must be determined and treatment given accordingly.

The prevention of hyperventilation in flying personnel is largely a matter of indoctrination. This is accomplished by instructing personnel in the proper use of oxygen equipment. The principal types of this equipment in the Air Force are described and illustrated on the pages that follow.

Recovery from hypoxia is usually rapid when sufficient oxygen is supplied. An individual on the threshold of unconsciousness may regain his full faculties within 15 seconds after he receives an abundance of oxygen. Experience has shown that, if a hypoxic patient breathes oxygen deeply, he may occasionally experience a flash of dizziness, but

this usually passes immediately and is followed by complete restoration of normal function. Known as the "oxygen paradox," this effect is probably caused by the cessation of the hypoxic respiratory drive that is mediated through the aortic and carotid bodies (chemoreceptors) with the first breath of oxygen. The normal respiratory drive, maintained predominately at the respiratory centers in the medulla by pH and P_{CO_2}, is now absent, due to the recent hypoxic hyperventilation and the concomitant alkalosis and hypocapnia.

Pressure Breathing

To prevent the effects of hypoxia above 40,000 feet some method of increasing the partial pressure of alveolar oxygen must be used. One method is positive pressure breathing by using an oxygen system that delivers 100% oxygen at greater than ambient pressures. Special pressure breathing regulators, mask, and mask valves are required.

In continuous positive pressure breathing, the process of breathing becomes more difficult at increasingly higher breathing pressures. The normal process of involuntary active inspiration and passive expiration is changed to a relatively passive, yet conscious inspiration and a very active expiration. Intermittent positive pressure breathing can partially compensate for this change in breathing mechanics by using a regulator which delivers oxygen under pressure only during inspiration. Therefore, both inspiration and expiration become passive phenomena. This quite commonly leads, however, to a symptomatic hyperventilation, which is one of the major disadvantages of intermittent positive pressure breathing.

Another major drawback is that intermittent positive pressure breathing provides a much lower mean mask pressure when compared with continuous positive pressure breathing systems. This means that the positive pressure portion of the intermittent cycle must be set at a higher level to bring the mean pressure up to the required magnitude. The *mean* mask pressure that is maintained during the complete respiratory cycle by positive pressure breathing is the important point of reference for evaluating alveolar oxygen pressure and the effectiveness of arterial oxygenation. Continuous positive pressure breathing provides a mean mask pressure that is nearly equal to the pressure delivered by the regulator, depending mainly on the tightness of the mask fit and the amount of mask leakage. Intermittent positive pressure breathing, on the other hand, can maintain a mean mask pressure that is only about one-third to one-half the peak pressure. For these reasons, continuous positive pressure breathing is the method of choice for use at high altitudes.

Breathing against continuous positive pressure is fatiguing and, because of the abnormal pressure relationships that are established between the lungs and the rest of the body, man's practical tolerance to pressure breathing and excessively high intrapulmonic pressures is limited and depends, in part, on his subjective response, his physical fitness, and the extent of cardiovascular compensations that can be brought into play for maintaining an adequate circulating blood volume. In this regard, the most efficient type of breathing pattern is worth consideration. If a rapid inspiration is followed by a prolonged expiration, the mean mask and intrapulmonic pressure of oxygen can be maintained at a high level, but there will also be an increase in the mean intrathoracic (intrapleural) pressure, somewhat similar to a valsalva maneuver. This restricts normal venous return to the thorax and flow of blood through the lungs, and results in an increased venous pressure and decreased cardiac output. On the other hand, if the breathing pattern is changed to a relatively slow inspiration, followed by a rapid expiration, the mean mask and intrapulmonic pressures will be only slightly lower, but the mean intrathoracic pressure will be considerably reduced, since considerably less pressure is transmitted across the elastic, expanding lungs to the heart and great vessels within the thorax. This will allow higher oxygen pressures to be used and, at the same time, tend to lessen somewhat the

undesirable circulatory effects. This, however, should not imply that, under the conditions of positive pressure breathing, *complete* relaxation of the thoracic and abdominal muscles during inspiration is desirable or even possible. The tensed abdominal muscles provide considerable support for the diaphragm, the venous vascular system, and venous return to the thorax. If allowed to relax completely, excessive pooling of blood below the diaphragm can result in a marked decrease in venous return and cardiac output. Likewise, the tense thoracic and diaphragmatic musculature helps to prevent excessive overdistention of the lungs and helps to maintain, as well as possible, the lung volumes within their relatively normal relationships, as shown in figure 2–1.

Ordinarily, mask pressures of 15 to 30 mm Hg (8 to 15 inches water) can be tolerated for limited periods. Above 30 mm Hg pressure, it has been shown that, besides fatigue caused by pressure breathing at these higher levels, other symptoms may occur. At these pressures, subjects commonly complain of being overinflated and, concurrently, a feeling of congestion in the region of the frontal sinuses. At higher pressures, there may be pain occurring in the ears and in the posterior pharynx as a result of overdistention.

This pain in the posterior pharynx commonly has been the cause for termination of flight when attempts were made to pressure breathe against 60 mm Hg. At pressures between 60 and 100 mm Hg, there is the great danger of parenchymal lung damage secondary to overexpansion unless counterpressurization is applied. Perhaps the greatest limitation to pressure breathing is the effect on the cardiovascular system. Increasing the intrathoracic pressure by pressure breathing results in an increased intrapleural pressure which tends to compress lung tissue. This offers considerable resistance to the flow of blood through the lungs which, in turn, increases the venous pressure. As a result, cardiac output is decreased and a pooling of blood occurs in the extremities, but more extensively in the larger vessels in the abdomen.

It is known that blood displacement by pressure breathing may lead to a loss of consciousness. This effect upon the cardiovascular system is the greatest limiting factor in pressure breathing. Therefore, for flights of any duration over 40,000 feet, it is necessary to pressurize the person either by means of a counterpressurization suit or a pressurized cabin. The latter has the advantage of making it possible to fly unencumbered ("shirt sleeve") above 50,000 feet without the continuous use of oxygen equipment, provided the cabin pressure is kept under 10,000 feet (523 mm Hg). According to current directives, it is not permissible to fly above 50,000 feet, regardless of cabin altitude, unless the flier is protected by a pressure suit or capsule in the event cabin pressure is lost.

In general, the pressurization of aircraft cabins during high altitude flights affords a safe and comfortable environment for the crews and passengers, but the extent to which an aircraft cabin can be pressurized is limited, primarily, by engineering, mechanical, and structural considerations. For these reasons, an ideal sea level pressure within an aircraft cabin at high altitudes is either prohibitive or impractical. Instead, a compromise must be made between this ideal pressure and the physiologic limits beyond which a flier cannot be safely exposed. The maximum differential pressure between the cabin and ambient pressures is limited by the type of aircraft and the type of mission for which it is designed to fly, such as transport and commercial airliners, high altitude bombers, and fighter aircraft. These limiting pressure differentials range from about 9 psi for commercial and other comparable military aircraft to 5 psi and 2.75 psi for military aircraft, depending on the flight situation. Because of this, as shown in figure 2–10, the higher the flight altitude the higher also must be the cabin altitude if the limiting pressure differential is not to be exceeded. The minimum pressure differential of 2.75 psi permits flights to any altitude with a

cabin altitude that will not exceed 40,000 feet. In this situation pressure breathing is not required but pure oxygen must be breathed to avoid hypoxia, and decompression sickness is a serious possibility without adequate denitrogenation before ascent. With a 5 psi differential pressure, regardless of the flight altitude, the cabin will always remain below an altitude of 30,000 feet, thereby reducing the probability of developing decompression sickness.

One potential hazard inherent in pressurized cabins is a sudden loss of pressure due to mechanical or structural defects or damage by gunfire, that may rapidly subject the pilot, crew, and passengers to altitudes where useful consciousness and life cannot be sustained. For this reason, there must be provided emergency oxygen and protective equipment which is adequate within the altitude profile of the aircraft. Between

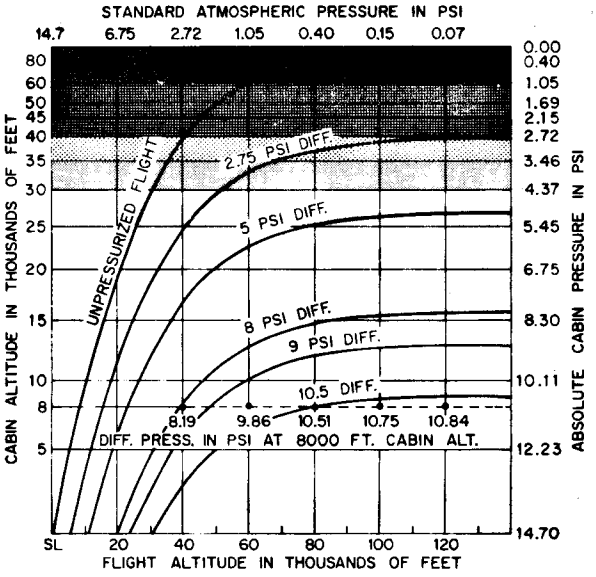
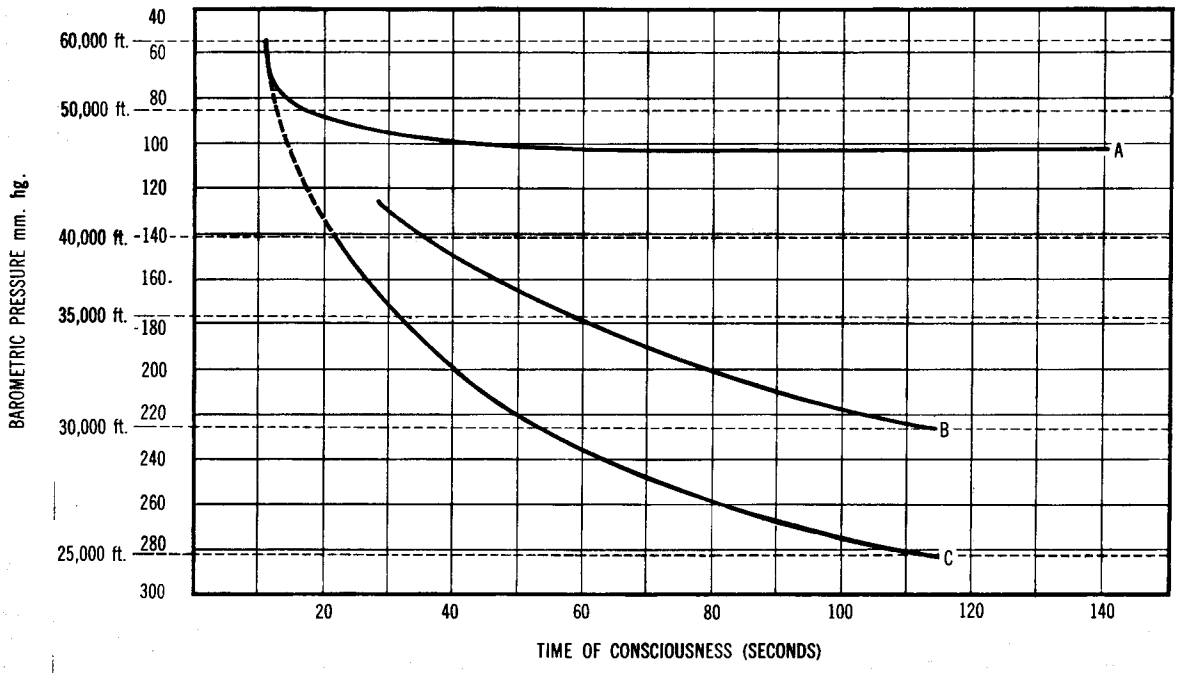


Figure 2–10. Cabin Pressure Schedules and Physiologic Thresholds at Various Flight Altitudes.



A. BENZINGER - 100% Oxygen at ambient pressure (rapid decompression from 40,000 ft.)

B. HALL - Air breathing (mask removal at altitude)

C. WILSON and COMFORT - Air breathing (rapid decompression from 10,000 ft.)

Figure 2–11. Time of Consciousness With Varying Types of Exposure at High Altitude.

40,000 and 50,000 feet, pressure breathing regulators will supply enough pressure to the pilot and/or crew for them to remain conscious during an immediate emergency descent to below 40,000 feet. However, sustained flight at these altitudes cannot be safely accomplished by pressure breathing.

Above 50,000 feet, pressure breathing is of little value since it is impossible to tolerate the amount of positive pressure necessary to prevent severe hypoxia without counterpressure. It is for this reason that all pilots and crews flying in aircraft without capsules above 50,000 feet are required to wear a counterpressurization garment of some type. These suits must inflate automatically in the event of a loss of cabin pressurization, and current suit types must offer the possibility of mission completion.

The likelihood of a sudden exposure to "critical altitudes" necessitates familiarity with the use of oxygen equipment and current pressure suits since, without this equipment, less than 15 to 30 seconds may elapse between the time of the decompression and loss of consciousness. Figure 2–11 shows the times of consciousness with varying types of exposure at high altitudes.

Oxygen Equipment

From the preceding discussion of the principles of respiratory physiology in aviation, it is evident that to produce efficient protective equipment for flight at high altitudes, engineers and physiologists must work together. With the evolution of oxygen equipment, man's altitude ceiling has been increased progressively.

In general, an oxygen system in an aircraft consists of containers for storing the oxygen supply, tubing to conduct the oxygen from the main supply to a metering device, a metering device to control the flow of oxygen, and a mask to direct the oxygen to the flier's respiratory system.

The first equipment used by the Air Force to protect fliers against reduced partial pressure of oxygen at high altitude was a simple tube connected through a valve type of metering device to a cylinder containing oxygen under high pressure. The maximum pressure in the oyxgen bottle was 1,800 psi. This tube was fitted with a pipe stem through which a continuous flow of oxygen was delivered into the user's mouth.

Although the system served its purpose by improving the flier's condition up to about 20,000 feet, it had the following disadvantages:

a. It did not adequately protect the normal nose-breathing individual.

b. Oxygen delivered during expiration, which is about half of the total time, was wasted.

c. The pipe stem was uncomfortable to hold between the teeth for long periods, especially in the unheated aircraft cockpits of the early days.

Continuous-Flow Oxygen System

In military aircraft, there is an obvious advantage in keeping the weight and space requirements of the oxygen system to a minimum. This increases the tactical efficiency of the aircraft by affording space for additional fuel to prolong the range of flight, for additional bomb loads, or for increasing the maneuverability essential in fighter aircraft. To economize on weight and space and to increase the altitude ceiling, a method was needed to restrict oxygen flow to a volume approaching the actual needs of an individual for a given altitude.

This requirement was quickly met by the design of a lightweight oronasal mask (figure 2–12), through which a continuous flow of oxygen was delivered to the flier. Attached to this mask was a rebreather bag (rubber bag of about 800 cc capacity) into which the first part of the exhaled gas was directed. Since this gas was from the upper part of the respiratory system, it consisted primarily of unused oxygen.

The rebreather system economizes by:

a. Reusing the oxygen contained in the respiratory dead space.

b. Supplying a reservoir to collect the volume of gas that flows from the oxygen cylinders during the expiratory phase of the breathing cycle.

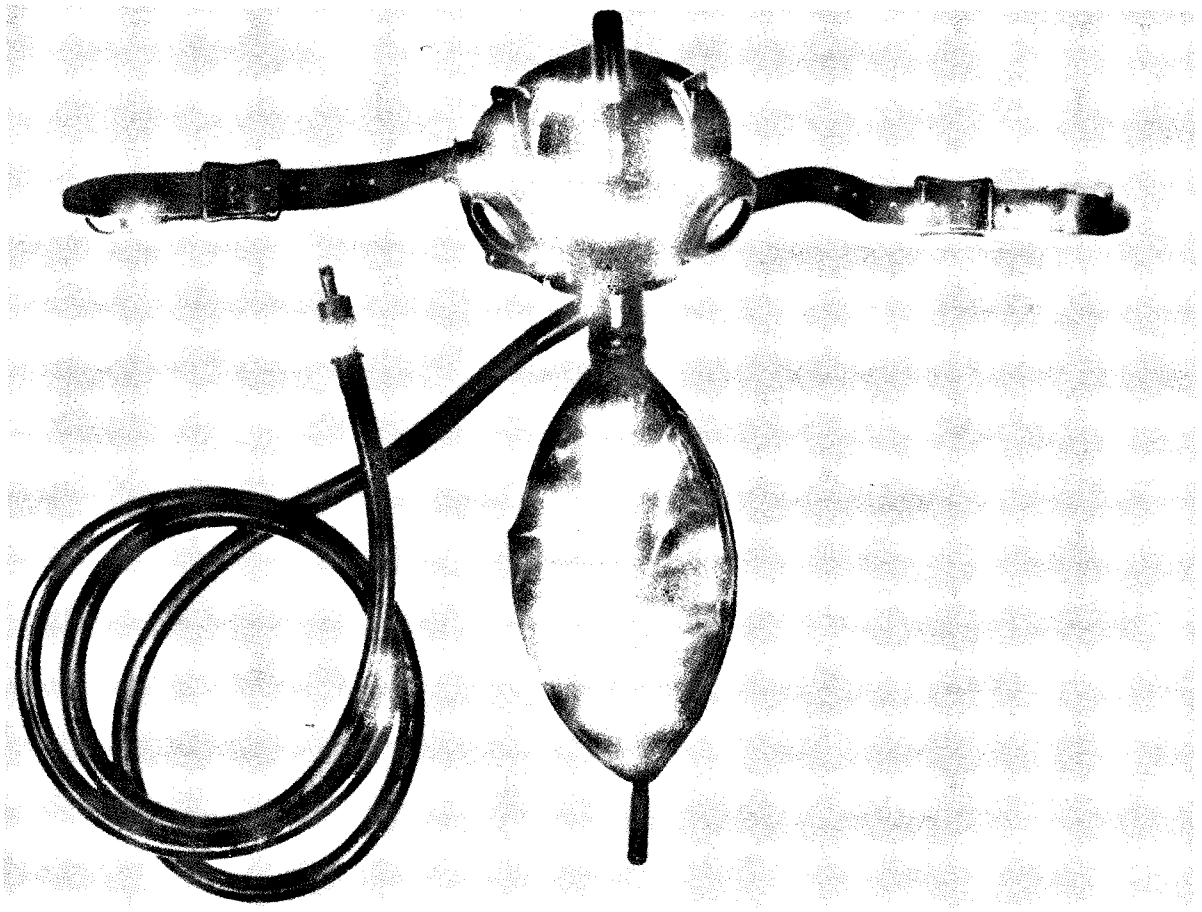


Figure 2–12. A–8B Constant Flow Oxygen Mask.

The mask is equipped with sponge-rubber discs that serve as valves, through which the latter portion of the exhaled air is blown off. They also serve as inspiratory ports for the entrance of ambient air when inspiration is not fully satisfied by the contents of the rebreather bag and the flow of oxygen from the regulator.

To maintain continuous flow of oxygen at various altitudes, the main oxygen supply is equipped with a metering device or oxygen regulator (figure 2–13). The regulator includes a valve that is opened or closed manually to compensate for changes in altitude. With this system, oxygen is delivered to the valve at a constant pressure and is unaffected by changing pressure within the cylinder as the supply is consumed. The flow gauge on the regulator is calibrated in thousands of feet. The flier sets the valve to correspond to his flight altitude and thus receives the proper amount of supplementary oxygen for that altitude. In another type of continuous-flow regulator, the A–11, the oxygen is controlled automatically rather than manually.

As discussed earlier in this chapter (see "Control of Ventilation"), under conditions of breathing 100% oxygen at ambient pressure, man can ascend to 40,000 feet and be in approximately the same condition as when breathing air at 10,000 feet. Since the continuous-flow system supplies 100% oxygen, it may be assumed that 40,000 feet is the upper limit for use of the equipment. Basically this is true, but the inherent characteristics of the standard form of the system make it necessary to limit this assump-

tion to conditions of rest. Therefore, it is adequate for passengers in transport aircraft below 30,000 feet because of the marginal degree of safety at higher altitudes in terms of mask fit and mask leakage.

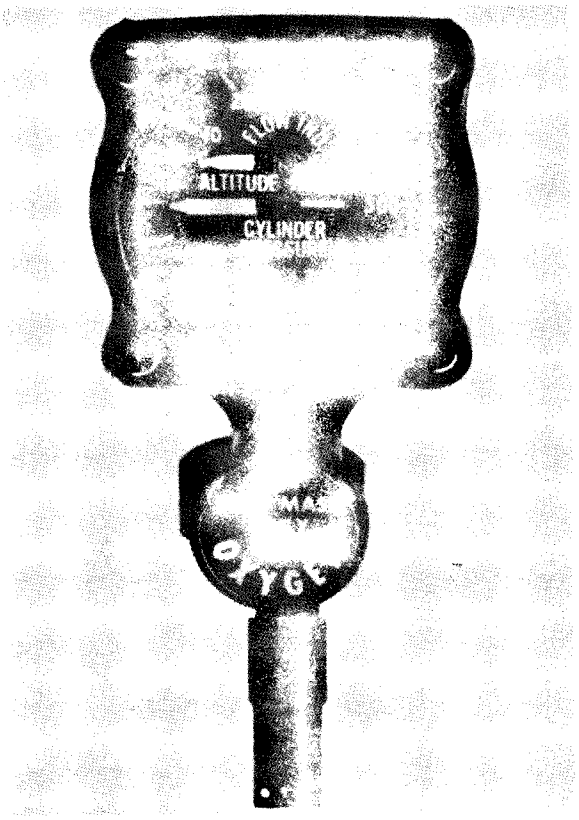


Figure 2–13. Oxygen Regulator (Limited Standard).

Additional activity required of crew members in military aircraft may create a greater demand for supplemental oxygen than that supplied by the regulator and result in excessive inspiration of ambient air through inspiratory ports, thus producing hypoxia. A mask fit which will prevent inboard leakage is difficult to obtain when the crewmember is active, and this becomes an important factor at higher altitudes where any leakage around the edge of the mask will result in dilution of the oxygen concentration supplied. In view of the practical experience in military aviation, an arbitrary altitude ceiling of 25,000 feet has been established for this type of equipment.

The continuous-flow system, commonly, is installed in cargo, air evacuation, and transport aircraft where it serves as a simple system that can be operated successfully, even by individuals who have not been especially trained in its use. As long as the limitations discussed above are observed, it is a good method of supplying oxygen for certain types of flights at moderate altitudes.

In the early part of World War II, it became apparent that aircraft would be operating in the altitude range above 25,000 feet. Above this critical altitude, the introduction of even small amounts of ambient air into the respiratory passages was hazardous. Thus, it became necessary to attack the problem of inboard mask leakage if flights above 25,000 feet were to be feasible.

Demand-Type Oxygen System

A major improvement over the continuous-flow oxygen system is the demand-type oxygen system. This system delivers oxygen only during the inspiratory phase of the breathing cycle. Extensive physiological and engineering tests indicated that this method of delivering oxygen at high altitudes assured the flier of receiving 100% oxygen as long as an airtight face-to-mask seal could be maintained. Many design and engineering problems were involved in developing this system to a stage where it was satisfactory for use in military aircraft. The pressure of wartime and the urgent necessity for standardization of this equipment made it possible to install the first form of the demand system in combat aircraft in a remarkably short time. Improvements were made as the need for them became apparent during service tests under combat conditions, until the present demand-oxygen system was finally evolved.

The demand-oxygen mask is a simple mechanism designed to fit comfortably over the face of the wearer (figure 2–14). It contains a single flapper valve, seated in the base of the mask facepiece, which permits all of the expired air to be blown off to the

outside atmosphere. During inspiration, the flapper seals tightly against the valve seat and no ambient air can be admitted to the mask through this channel.

The metering system that controls the flow of oxygen to the mask is called the diluter demand regulator (figure 2–15). This regulator is a fairly simple mechanism operated by normal changes in pressure occurring during the breathing cycle. Basically, it consists of a round box with a thin rubber diaphragm stretched across the front. Attached to the diaphragm is the lever of a valve that opens or closes the port that leads to the pressure reduction stage of the regulator.

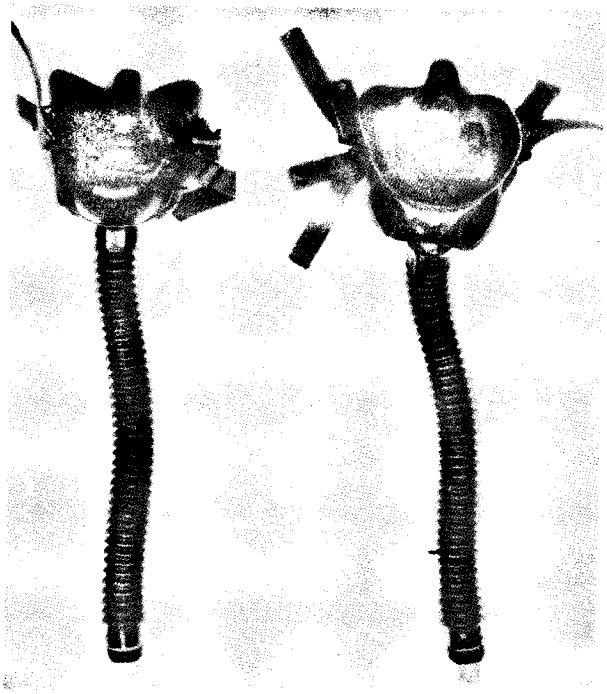


Figure 2–14. A–14B Oxygen Mask (Limited Standard).

When the mask is attached to the regulator through a hose connection and the wearer inhales, a slight negative pressure is created within the regulator. This negative pressure draws the diaphragm in and changes the position of the valve so that an opening is created to the pressure reduction stage containing oxygen under pressure, and this oxygen flows through the regulator. The initial chamber of the regulator is a pressure reduction stage where oxygen cylinder pressure is reduced to a lower constant pressure, providing a relatively fixed pressure behind the diaphragm-operated valve.

Barring mechanical difficulties in the regulator itself, the only site of inward air leakage is around the face-to-mask seal. A number of different mask designs were tried until one was standardized which met the requirements for comfort and effective seal on the majority of types of faces. However, no matter how carefully such a mask is fitted, a minute leakage seems to be inevitable. This leakage factor makes it necessary, in the interest of safety, to limit the use of the diluter-demand oxygen system to altitudes below 35,000 feet.

Cylinder oxygen is further conserved in the demand system by making use of the oxygen present in ambient air at altitudes up to 34,000 feet where 100% oxygen is required. The diluter mechanism that mixes air and oxygen consists of a metal bellows attached to valves over both air and oxygen ports. As the flight altitude increases, trapped air in the bellows expands causing the bellows to operate these two valves. Thus, during ascent, the oxygen valve opening is gradually increased while the air inlet is reduced, thereby increasing proportionately the concentration of oxygen delivered to the mask.

The percentage of oxygen required for any given altitude has been determined by physiological experiments, and the design of the diluter mechanism of the regulator is based upon these data. A gradually changing mixture of air and oxygen is delivered up to about 30,000 feet, at which altitude the air inlet is closed completely and 100% oxygen is delivered. The user can change, at any time, the dilution lever on the side of the regulator from the "normal" diluting position to the "100% oxygen" position which will close off the air inlet and provide 100% oxygen at any altitude.

For the treatment of mild hypoxia, below 35,000 feet, the demand regulator is provided

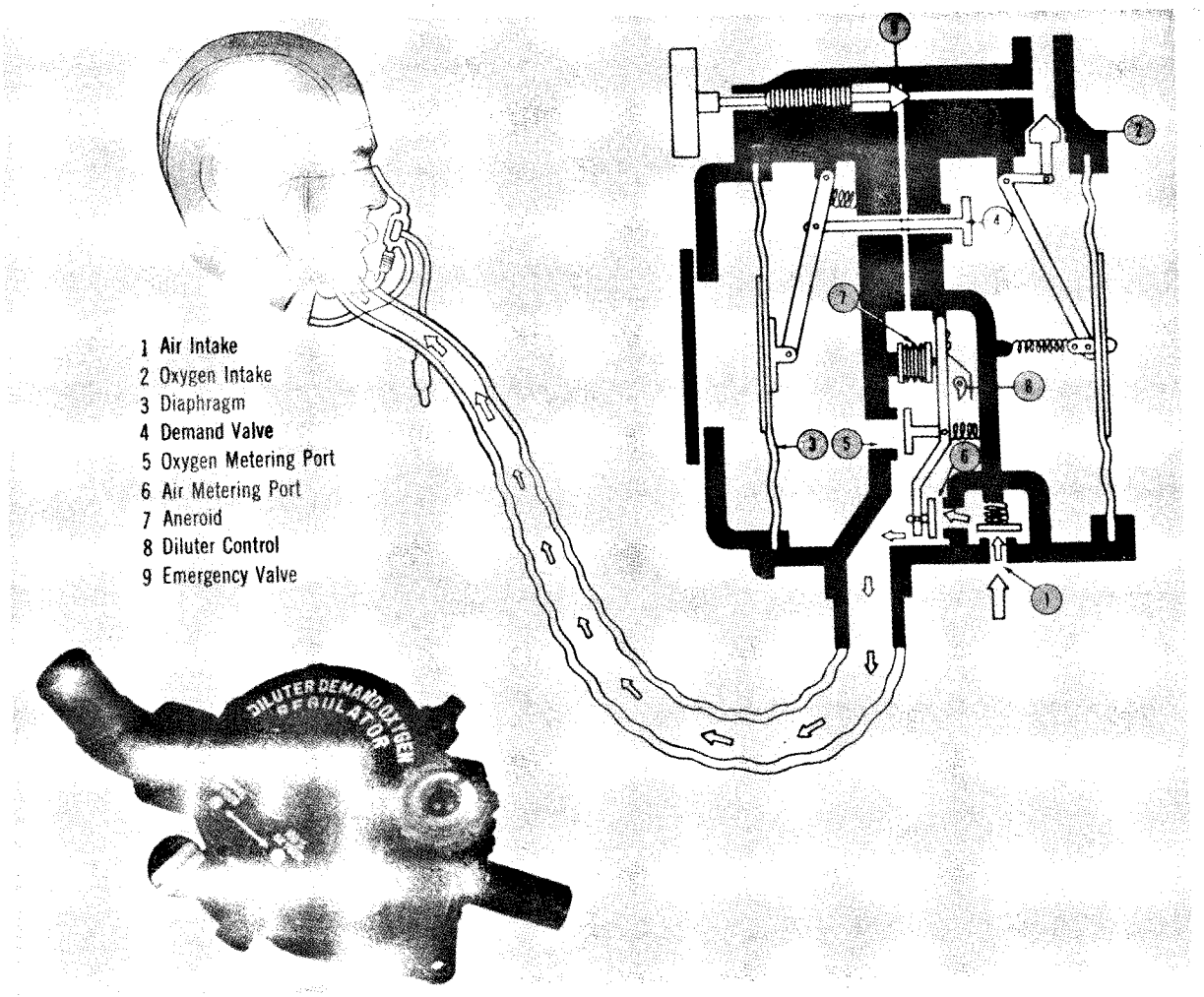


Figure 2–15. Diluter Demand Oxygen Regulator.

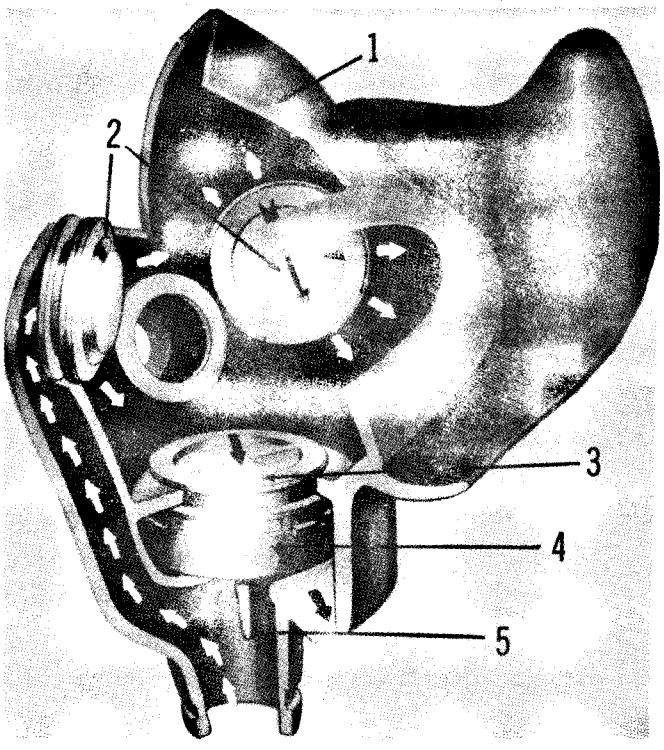
with an emergency valve. When this valve is opened, a large volume of oxygen in a continuous flow may be obtained quickly. It is sufficient to revive an hypoxic individual in a few seconds under most conditions, and maintain normal arterial oxygen saturation, even with a pulmonary ventilation rate equivalent to that of moderate exercise. It should be kept in mind that indiscriminate and excessive use of the emergency valve is extremely wasteful of oxygen and, if left open unnecessarily, can deplete the aircraft oxygen supply quickly.

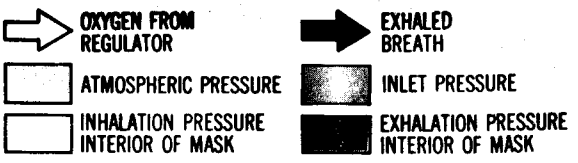
To maintain fliers in a normal condition at altitudes above 34,000 feet, two requirements must be fulfilled:

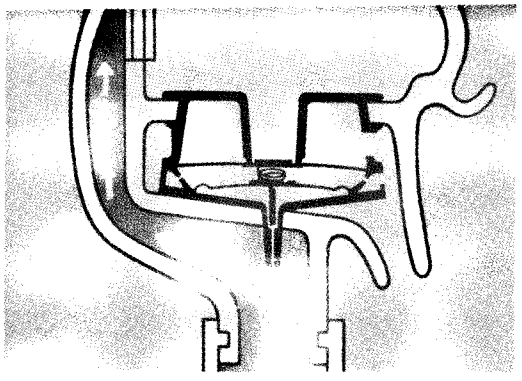
a. Up to 40,000 feet, inboard mask leakage must be eliminated so that 100% oxygen can be delivered to the lungs with every breath.

b. Above 40,000 feet, oxygen must be delivered to the mask at pressures in excess of ambient. If sufficient pressure is added to the mask, positive pressure breathing will maintain a normal alveolar partial pressure of oxygen.

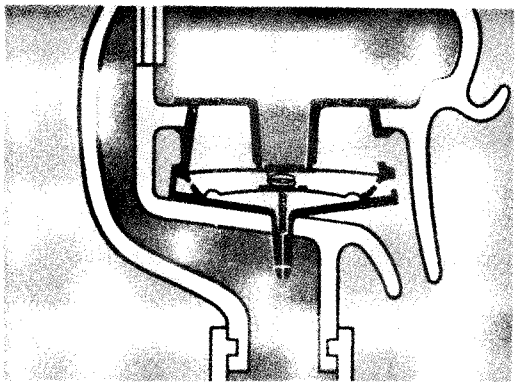
From a mechanical standpoint, the same modifications fulfill these two requirements. It is possible to eliminate inboard mask leakage by supplying oxygen at a small positive pressure—about 2 inches of water. Under these conditions, if the mask-to-face



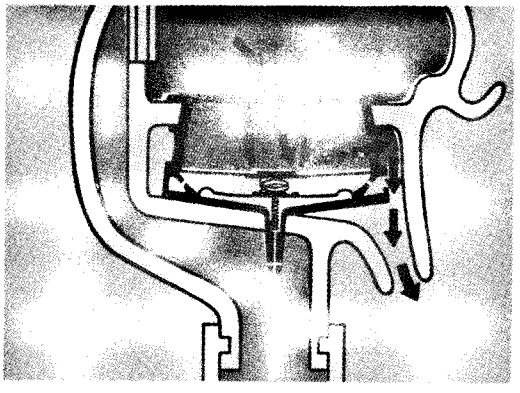




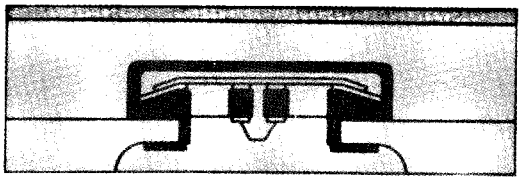
INHALATION. The inlet pressure is greater than the pressure inside the mask during inhalation. The compensating diaphragm is pushed up against the main diaphragm, closing the exhalation valve.

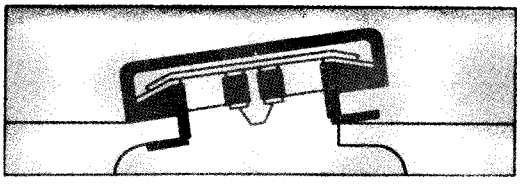


PAUSE BETWEEN INHALATION AND EXHALATION. The inlet pressure and the pressure inside the mask are momentarily equal. The valve remains closed.



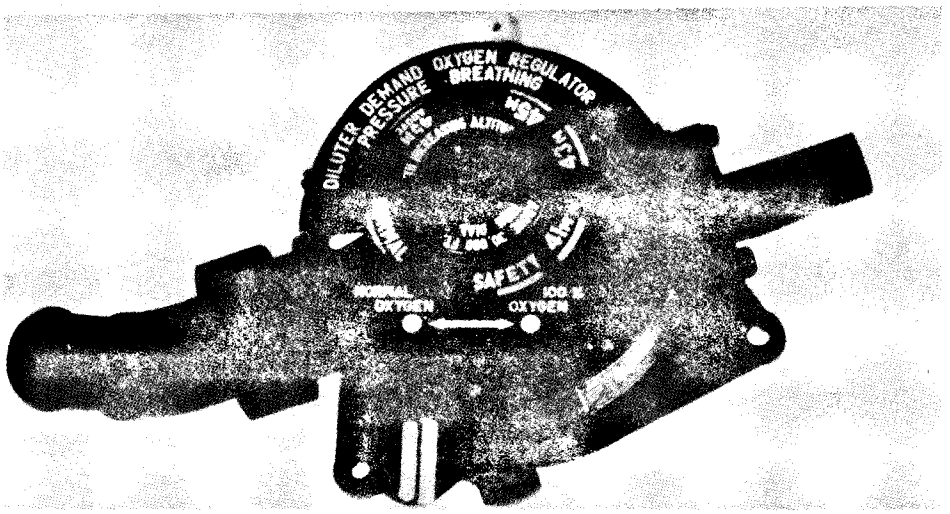
EXHALATION. The pressure inside the mask momentarily increases slightly above the inlet pressure, so that the main diaphragm and the compensating diaphragm are pushed down, permitting the exhaled breath to pass through the valve and the outlet.

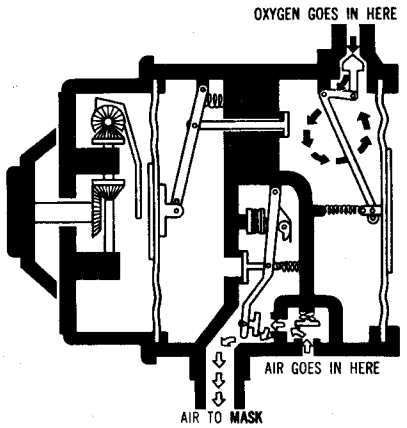




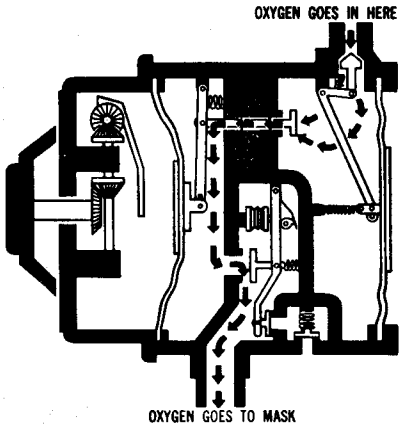
IF THESE INLET CHECK VALVES ARE NOT SET INTO PLACE PROPERLY, THE MASK WILL NOT WORK

Figure 2–16. Components of Type MS–22001 Pressure Breathing Oxygen Mask.

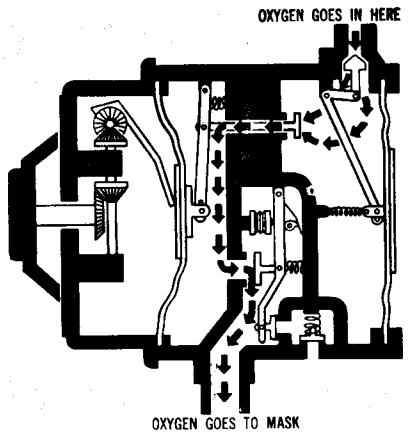




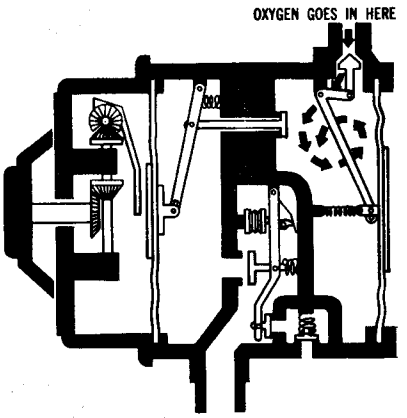
REGULATOR DURING INHALATION AT SEA LEVEL. Oxygen valve is closed; air valve is open, and you breathe air only



REGULATOR DURING INHALATION AT 34,000 FEET. Air valve is closed; oxygen valve is open, and you breathe 100% oxygen.



REGULATOR DURING INHALATION WITH PRESSURE BREATHING. Spring presses down on diaphragm, opening de mand valve, and forcing oxygen into the mask under pressure



REGULATOR DURING EXHALATION WITH PRESSURE BREATHING. As you exhale, you momentarily raise the pressure, forcing the diaphragm up against the spring tension. The demand valve closes and no oxygen flows.

Figure 2-17. A-14 Manual Pressure Breathing, Diluter Demand Oxygen Regulator.

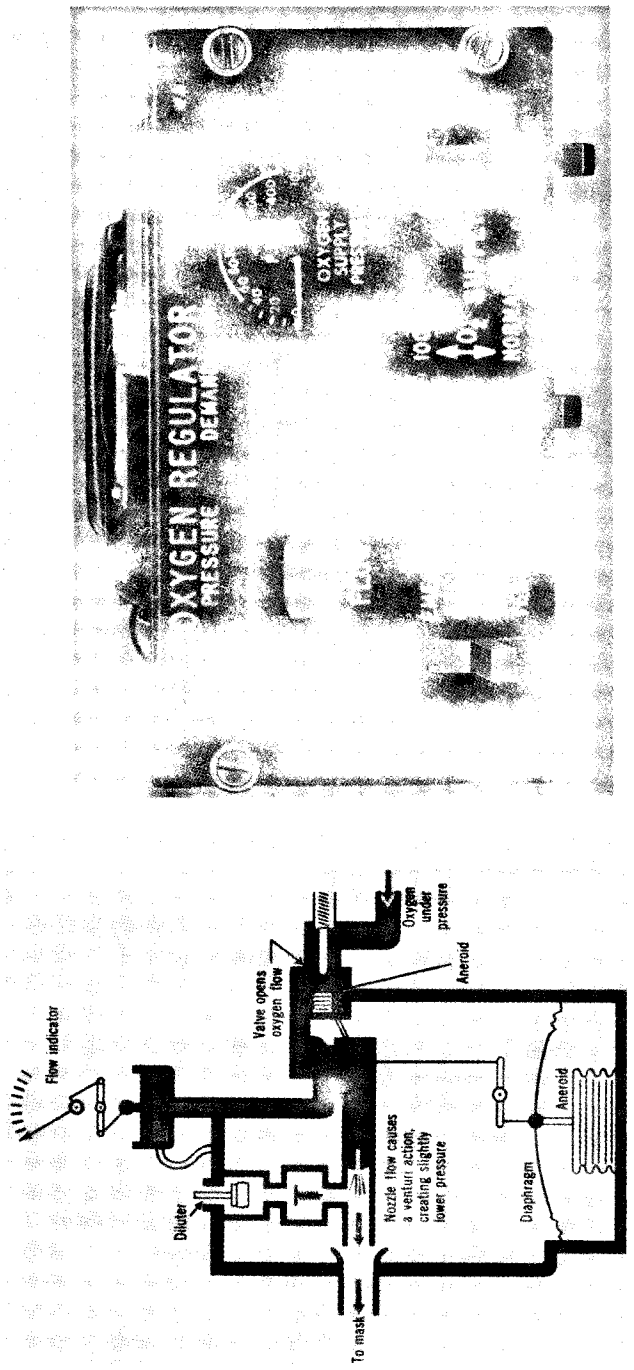


Figure 2–18. MD–1 Automatic Pressure Breathing, Diluter Demand Oxygen Regulator (Limited Standard).

seal is broken, there would be loss of oxygen to the outside atmosphere, but the slightly greater pressure within the mask would prevent ambient air from being drawn in during inspiration. This concept of "safety" pressure has been applied to standard pressure-demand oxygen systems and is used when the person is at an altitude between 30,000 and 40,000 feet.

Adjustment of the seal of the pressure-breathing mask upon the face makes it possible to increase its pressure-holding capacity and to permit breathing oxygen at pressures up to 30 mm Hg.

Basically, only two changes have been made in the demand-oxygen mask to convert it to a pressure-breathing system. They are shown in the illustration of the operation of the pressure-compensated valve (figure 2–16). The mask is molded with an inner flap (1) that tends to seal against the face when oxygen is delivered at positive pressures. In addition, it was necessary to alter the valve system because the standard demand-mask exhalation valve (3) opened at a very small positive pressure. A direct connection was made between the underside of the exhalation valve (4) and the incoming oxygen line (5). The pressure of the oxygen delivered by the regulator is, thus, effective in closing the exhalation valve.

To open the exhalation valve, then, it is necessary to exceed the pressure in the incoming oxygen line by exhaling with a greater force than that of the incoming oxygen. Check valves (2) over the oxygen inlet ports in the mask make it possible to develop the necessary exhalation pressure within the mask.

A spring applied to the diaphragm converts the standard demand regulator to the pressure-demand system (figure 2–17). The pressure-demand system may be activated automatically by an aneroid mechanism. The automatic type of control is essential for emergency situations arising in aircraft with operating ceilings up to 50,000 feet. Figure 2–18 illustrates a recent type of automatic regulator. For specific details on all current types of oxygen equipment, refer to the

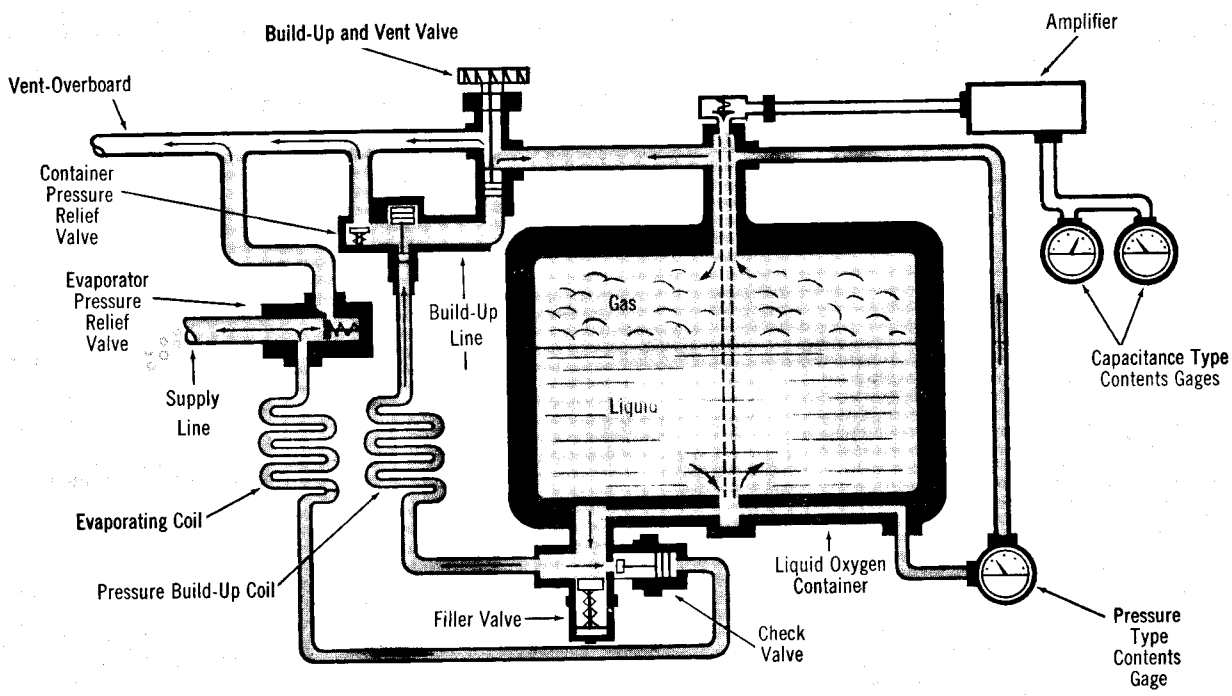
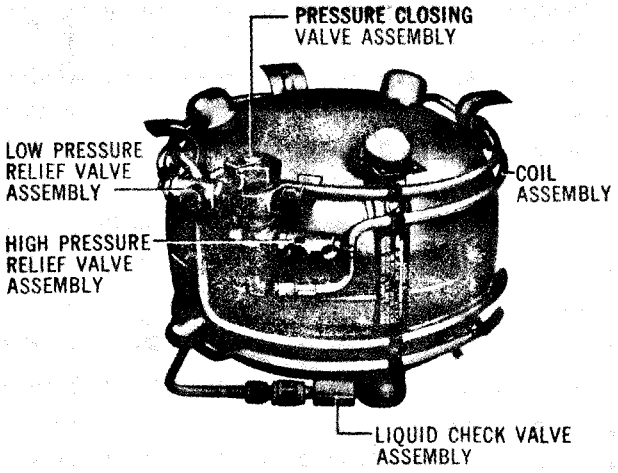


Figure 2–19. Schema of Liquid Oxygen System Components.



Capacity	5 liters
Operating Pressure	70 psi
Filling Pressure	20 psi
Filling Time	5 min
Build-Up Time	10 min
Weight (Empty)	14.0 lb.
Weight (Full)	26.5 lb.
Width (Overall)	12⅞ in.
Height (Overall)	9⅜ in.
Cubage (Maximum)	1,370 cu. in.

Figure 2–20. USAF Type A–3 Five-Liter Liquid Oxygen Converter.

current Technical Order listed in the references.

Liquid Oxygen Systems

The development of inflight refueling techniques and more effective aircraft and personal pressurization systems have extended the performance capabilities of current aircraft. Concurrent with these capabilities is an increased need for crew oxygen supplies due to the increased flight length.

This need was solved by the development of an entirely different, more complex oxygen system that used liquid oxygen (LOX) in place of gaseous oxygen. Even though there are disadvantages in this system, the LOX system more than offsets these disadvantages by storing large quantities of breathing oxygen in a compact, lightweight unit. Figure 2–19 shows the components of a LOX system and their relative positions. This schema is not typical of any particular aircraft installation, but is intended to indicate the operating characteristics of LOX systems in general. Figure 2–20 is a USAF Type A–3 five-liter liquid oxygen converter. One liter of liquid oxygen will provide approximately 800 liters of gaseous oxygen at normal sea level conditions. In comparison, a one-liter gaseous oxygen cylinder, pressurized to 450 psi, will provide only about 30 liters under the same conditions.

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