Dr. Downes Deposition Exhibits

Marked on October 7, 1981

re: General

Extra

Downes Ex #1
10/7/81
JT

THE CHILDREN'S HOSPITAL OF PHILADELPHIA
ONE CHILDREN'S CENTER
34TH STREET AND CIVIC CENTER BOULEVARD
PHILADELPHIA, PA 19104
(215) 596-9387

DEPARTMENT OF ANESTHESIA
JOHN J. DOWNES, M.D.
ANESTHESIOLOGIST-IN-CHIEF
RUSSELL C. RAPHAELY, M.D.
ASSOCIATE DIRECTOR

K. W. TERRY LEE, M.D.
A. MICHAEL BROENNLE, M.D.
EUGENE K. BETTS, M.D.
ROBERT G. KETTRICK, M.D.
JOSEPHINE J. TEMPLETON, M.D.
RODOLFO I. GODINEZ, M.D., PH.D.
DAVID B. SWEDLOW, M.D.
THOMAS P. KEON, M.D.
MARK S. HEISER, M.D.
NORIG ELLISON, M.D.
DAVID R. JOBES, M.D.
ALAN J. SCHWARTZ, M.D.
GERALD S. LEFEVER, M.D., PH.D.

October 5, 1981

Mr. John Connors
Mr. Carroll E. Dubuc
Haight, Gardner, Poor and Havens
Federal Bar Building
Washington, D.C. 20006

Re: FFAC v. Lockheed Aircraft Corporation

Dear Sirs:

I have reviewed the material which you forwarded to me regarding the extraordinary decompression accident involving a C-5A transport on April 4, 1975 near Saigon, South Vietnam. From our conversations and the data at hand, it appears that an unspecified number of Vietnamese refugee children were subjected to effects of sudden decompression from a cabin altitude of 5,000 ft. to the aircraft altitude of 23,400 ft.

It is my understanding that the plaintiffs allege that subsequent and present neurologic and mental handicaps resulted from the hypoxia and other effects of this acute decompression. In this regard, I see two key issues: 1) to estimate the degree of arterial hypoxemia and its duration from the time of the decompression until the aircraft had descended to a safe altitude; and, 2) to assess whether the cardiorespiratory reflex responses to hypoxia in infants and children between 9 months and 2 years (the ages involved) are comparable to, better than, or less effective than in adults.

In order to address issue #1 certain assumptions must be made and the evidence presented to me makes these seem reasonable.

1. No serious intrinsic cardiopulmonary disease prior to the decompression.

2. No serious disease of the brain stem or neuromuscular system prior to the decompression.

3. A hemoglobin concentration of 10 gm/dl; normal blood volume for age (approximately 75 ml/kg); hemoglobin capacity for O_2 of 1.34 ml/gm.

4. Normal arterial pressure (50-60 mmHg) and cardiac index (3.0 - 3.5 L/min/M_2)

Given these assumptions, one can presume that the infants had a normal alveolar and arterial PO_2 and PCO_2 at sea level prior to the flight, and reduction in alveolar PO_2 and alveolar PCO_2 reflected the decrease in barometric pressure to approximately 632 mmHg at a cabin altitude of 5,000 ft.; one expects a 2 mmHg reduction in P_ACO_2 associated with the mild hyperventilation due to the drop in the P_AO_2 (See Table). Assuming an alveolar-arterial tension difference ($AaDO_2$) for O_2 of 10 mmHg at cabin altitude, one would anticipate an arterial oxygen tension (PaO_2) of 74 mmHg (oxygen saturation, SaO_2 94%) and a negligible alveolar-arterial carbon dioxide tension difference resulting in a $PaCO_2$ of 33 mmHg. When the decompression occured, within fifteen seconds two events can be expected to occur: 1) a fall in P_AO_2 from 84 to 31 mmHg associated with a fall in PaO_2 to approximately 25 mmHg ($AaDO_2$ would be decreased because of hyperventilation in response to the hypoxia), and 2) the P_ACO_2 and the $PaCO_2$ would decrease from 33-28 mmHg because of the chemoreceptor stimulus to increase ventilation secondary to the drop in PaO_2. At a PaO_2 of 25 mmHg and $PaCO_2$ of 28 mmHg, the SaO_2 would be 51%.

This degree of hypoxemia was endured for less than 1.75 minutes as the aircraft descended to 20,000 ft., and the P_AO_2 could be expected to rise to 38 mmHg; P_ACO_2 falls because of sustained hyperventilation to 25 mmHg with an increase in PaO_2 to approximately 30 mmHg (SaO_2 53%). At these two levels of PO_2, the oxygen content of arterial blood would be 5.6 ml/dl at 23,400 ft., and 6.8 ml/dl at 20,000 ft.; in both instances oxygen extraction from the blood down to a PO_2 of 18 mmHg (SaO_2 30%), giving an arterio-venous oxygen content difference of 2.8 ml/dl and 3.5 ml/dl respectively, should occur. With the expected 50 to 100% increase in cerebral blood flow that occurs in adults, and the higher flows observed at normoxia in children (see below), sufficient oxygen delivery to maintain cell integrity of the brain for periods of 3 to 5 minutes at rest can be expected.

Data from the literature on cerebral blood flow in children (Kennedy C, and Sokoloff L: J Clin Invest 36:1130, 1957, and Kety SS: J Chron Dis 3:478, 1956) indicates that cerebral vascular resistance in children is approximately half and cerebral blood flow nearly double that of adults under comparable resting conditions in normal individuals. The basal cerebral metabolic rate for oxygen ($CMRO_2$) is increased by approximately 25% in children when compared with healthy young adults (same references). Thus, it can be said that children have the luxury of greater cerebral blood flow in relation to O_2 demand than observed in adults, probably to meet the child's long term needs for sustained growth and development of the brain. However, in the acute hypoxic event, the infant and child would appear to be protected against brain anoxia when compared to the adult. In addition, I would expect the respiratory and cardiovascular responses to hypoxia to be intact in infants and children who do not have severe preexisting central nervous system or cardiopulmonary disease. In such an instance, the cerebral vascular resistance might well decrease with a sudden hypoxic stimulus such as developed in the acute decompression in this case; although the children would also respond with hyperventilation resulting in a decrease in $PaCO_2$, with consequent stimulus for cerebral vasoconstriction, it would seem probable that the hypoxic stimulus would dominate as it does in the healthy young adult.

I have been in the practice of pediatric anesthesia and critical care since 1963. During that time I have observed many episodes of acute hypoxemia in previously non-hypoxemic infants and children. In certain instances the magnitude of hypoxemia was similar to that which may have occured in the infants and children involved in this case, but of much longer duration. The cardiorespiratory and central nervous system responses of these infants and children which I observed and cared for were qualitatively, and insofar as we could determine quantitatively, similar to that reported in adults suffering a comparable degree of hypoxia. Thus, based on the facts which you have provided, the literature, my personal experience, and some assumptions about the cardiopulmonary status of the infants and children in question, I can state with reasonable medical certainty that the hypoxic event which occured in these infants and children would not account for prolonged significant central nervous system damage.

I have appended a table of my calculations of alveolar gas tensions and the appropriate reference citations. I hope this information is of assistance.

Sincerely,

John J. Downes, M.D.
Anesthesiologist-in-Chief and Director,
DEPARTMENT of ANESTHESIA and CRITICAL CARE
The Children's Hospital of Philadelphia

Professor of Anesthesia and Pediatrics
University of Pennsylvania School of Medicine

JJD:cp

TABLE: AVERAGE ALVEOLAR GAS TENSIONS
IN ACUTE DECOMPRESSION AT ALTITUDE

ALTITUDE (ft.)	P_B	P_IO_2	ADULT[1] P_AO_2	ADULT[1] P_ACO_2	INFANT/CHILD[2] P_AO_2	INFANT/CHILD[2] P_ACO_2	Time (min.)
23,400	303	54	26	28	26	28	0.25
20,000	349	64	33	30	38	25	1.75
15,000	429	80	46	33	51	28	4.75
10,000	523	100	61	36	66	31	TOTAL - 7.88
5,000	632	122	79	38	84	33	
Sea Level	760	149	103	40	108	35	

Cabin altitude before decompression: 5,000 ft.

Calculations based on: 1) Alveolar air equation:

$$P_AO_2 = \left(P_B - 47\right)\left(F_IO_2\right) - P_ACO_2 \left[F_IO_2 + \frac{1 - F_IO_2}{R}\right]$$

in which F_IO_2 is 0.2094, R is 0.79

2) Arterial oxygen content (CaO_2) in ml/dl:

CaO_2 = Hb (gm/dl) x 1.34 (ml/gm) x SaO_2 + PaO_2 (mmHg) x 0.3 (ml/dl).

[1] Luft UC: Altitude Sickness (Ch. 9) in Armstrong HG (ed): Aerospace Medicine. Williams and Wilkins, Baltimore, 1961, pp 120-142, as cited in Randel HW (ed): ibid, 2nd edition, 1971, p. 62.

[2] Data from: 1) Albert MS, Winter RW: Pediatrics 37:728, 1966

2) Levison H, Featherby EA, Weng TR: Am Rev Resp Dis 101:274, 1970

3) Roughton FJW: Transport of oxygen and carbon dioxide. Ch. 31 in Fenn WD, Rahn H (eds): Handbook of Respiration, Vol. I, Amer Physiol Soc, Washington, D.C., 1964

4) Severinghaus J: Blood O_2 Dissociation Line Charts: Man. Handbook of Respiration, National Academy of Sciences, W.B. Saunders, Philadelphia, 1958, p. 73.

CAPS

Downes C # 2
10/7/81

Table: 1

Average Alveolar Gas Tensions in Acute Decompression At Altitude

			Adult (1)		Infant/child (2)		
Altitude (ft)	P_B	P_{IO_2}	P_{AO_2}	P_{ACO_2}	P_{AO_2}	P_{ACO_2}	Time
23, 400	303	54	26	28	26	28	0.25
20, 000	349	64	33	30	38	25	1.75
15, 000	429	80	46	33	51	28	4.75
10, 000	523	100	61	36	66	31	7.88
5, 000	632	122	79	38	84	33	
sea level	760	149	103	40	108	35	

Cabin altitude before decompression: 5,000 ft.

Calculations based on: 1) Alveolar air equation

$$P_{AO_2} = (P_B - 47)(F_{IO_2}) - P_{ACO_2}\left[F_{IO_2} + \frac{1 - F_{IO_2}}{R}\right]$$

in which F_{IO_2} is 0.2094,
R is 1.0

At 23,400

$$P_{AO_2} = (303 - 47)(.21) - 28\left(.21 + \frac{1 - .21}{0.79}\right)$$
$= ~~53.7 - 28.2~~$
$= 25.6$

At 20,000

$$P_{AO_2} = (349 - 47)(.21) - 25\left(.21 + \frac{1 - .21}{.79}\right)$$
$= ~~63.4 - 25.2~~$
$= 38.2$

(1) Luft, UC: Altitude Sickness (Ch. 9) in Armstrong HG (ed): Aerospace Medicine Williams and Wilkins, Baltimore, 1961 pp 120-142 as cited in Randel HW (ed): ibid (2nd edition, 1971) p. 62

(2) Data from: 1) Albert, MS; Winter RW Pediatrics 37: 728, 1966
2) Levison H, Featherby EA, Weng TR: Am Rev Resp Dis 101:274, 1970

Table 2 Estimated Average Blood Gas Values in Children During Acute Decompression At Altitude

Document #

(Infant/Child Values)

Altitude ft	Time min	$P_{A,a}CO_2$ mm Hg	P_{AO_2} mm Hg	$P_{a}O_2$ mm Hg	$P_{jv}O_2$ mm Hg	SaO_2 %	$SjvO_2$ %	CaO_2 ml/dl	$CjvO_2$ ml/dl	Ca-jv C ml/dl
23,400	:25	28	26	24	18	51	30	6.8	4.0	2.8
20,000	1.75	25	38	30	20	63	35	8.2	4.7	3.5
15,000	4.75 Total 7.88	28	51	42	25	82	46	10.7	6.2	4.5
10,000		31	66	56	27	89	51	11.9	6.9	5.0
5,000		33	84	74	30	94	57	12.6	7.6	5.0

Assumptions Hb = 10gm/dl

Hb Cap O_2 = 1.34 ml/gm

P_{a,O_2} 23 = pH 7.52 } 23,400

V 30 = 7.44 }

a 25 = 7.49 } 20,000

V 32 = 7.42 }

CaO_2 19.6 (17.3-22.

jv-RA = 41 { C_vO_2 12.9 (11.0-15.

C_TO_2 15.7

Jugular 18-20 P_{O_2} mm

Downes Ex #4
10/7/81
[illegible]

Plaintiff's witness

BUSBY COMPUTATIONS BASED ON
SCHNEIDER TRIAL TESTIMONY

SEA LEVEL:

760 x .21 = 159 Ambient O_2	
- 47	
713	tracheal
x .21	
149	tracheal O_2
- 40	CO_2
109	alveolar
- 09	alveolar arterial grad
100	arterial PO_2

23,400 FEET:

303 x .21 = 63 Ambient O_2	
- 47	
256	tracheal pressure
x .21	
~~53.7~~	tracheal O_2
- 46	CO_2 (due to sleep)
7.7	

.05 x 303 = 15 ?

why 46 torr - that's at sea level ? because they decomp. from 5000 ft. not sea level

Add increased breathing effect (this is at 22,000 feet, not 23,400) but is figure used by Busby)

7.7	
+ 20.0	
27.7	
????	Decreased gradient

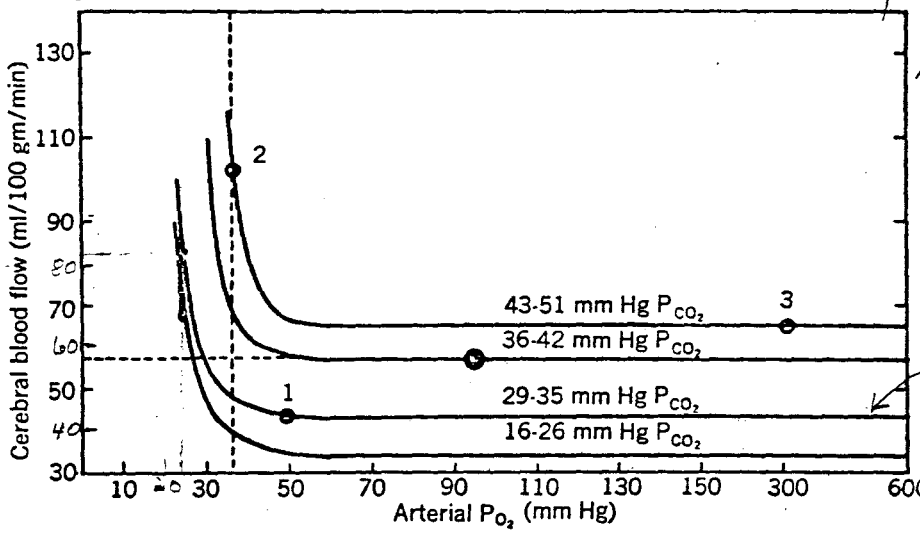


Fig. 62-28. Relationships among arterial P_{O_2}, arterial P_{CO_2}, and rate of brain blood flow (based on 56 measurements in seven normal men). • represents normal blood flow and arterial gas tensions for air breathing at sea level. Above about 50 mm Hg arterial P_{O_2}, O_2 tension appears to exert no important effect on blood flow. Vasodilator effect of low arterial P_{O_2} becomes increasingly prominent as arterial P_{O_2} falls below 50 mm Hg. By varying arterial P_{CO_2}, wide fluctuations in brain blood flow can be produced both in absence and in presence of anoxemia; actually hypocapnia is capable of reversing and hypercapnia of exaggerating cerebral vasodilatation normally associated with anoxemia. Illustration shows that particular rate of cerebral blood flow can be obtained over wide range of variation of arterial P_{O_2} and P_{CO_2}. Numbered open circles indicate conditions to be expected in, *1*, hyperventilation of altitude hypoxia; *2*, hypoxia and hypercapnia of insufficient pulmonary ventilation; and *3*, administration of O_2 to patient with pulmonary insufficiency. (From Lambertsen.[105])

related events beginning with an increase in the amount of physically dissolved O_2 in blood. This results in diminished reduction of hemoglobin, decreased availability of base for CO_2 transport, and an increase in central P_{CO_2} and pH[27, 103, 111, 113, 114] (Figs. 60-9 and 60-10). Probably this central acidosis is responsible for the respiratory stimulation that lowers arterial $[H^+]$.[114] When this slight arterial hypocapnia is prevented by use of devices for artificially controlling alveolar P_{CO_2},[115] no cerebral vasoconstrictor effect of O_2 is seen.[104, 105] For this reason the cerebral vasomotor actions of O_2 excess and O_2 lack do not appear to be part of a smoothly continuous physiologic effect of change in P_{O_2}. More likely, high O_2 tensions produce little or no effect on adult vessels, and hypoxic vasodilatation represents a pathologic failure of the normal mechanisms for maintenance of vascular tone.[45, 105] This possibility must be examined in the light of clear demonstration that O_2 administration does cause constriction of the ductus arteriosus in the newborn lamb.[30]

O_2 administration aids respiratory and brain circulatory studies by making it possible to produce central *hypercapnia* concurrently with arterial *hypocapnia*.[104] The situation resembles that to be expected if the rate of metabolic CO_2 production by the brain cells were grossly increased. Since hypercapnia limited to the venous or tissue side of the brain circulation leads not to dilatation but to constriction of brain vessels (Table 62-2), it is unlikely that the brain blood flow is regulated by local metabolite production. To account for the increases in local circulation demonstrated to occur with exaggerated functional activity,[175, 186] metabolic factors other than P_{CO_2} have to be evaluated. One such metabolic factor is a localized diminution of P_{O_2}. However, even a fall in P_{O_2} during increased metabolism is an unlikely basis for the dilatation of vessels in the activated region since the P_{O_2} of arterial blood must be reduced nearly 50 mm Hg below normal before cerebral vasodilatation becomes evident[105] (Fig. 62-28). Thus, although local response to increased functional activity is almost certainly a feature of cerebral circulatory control, it is more likely that

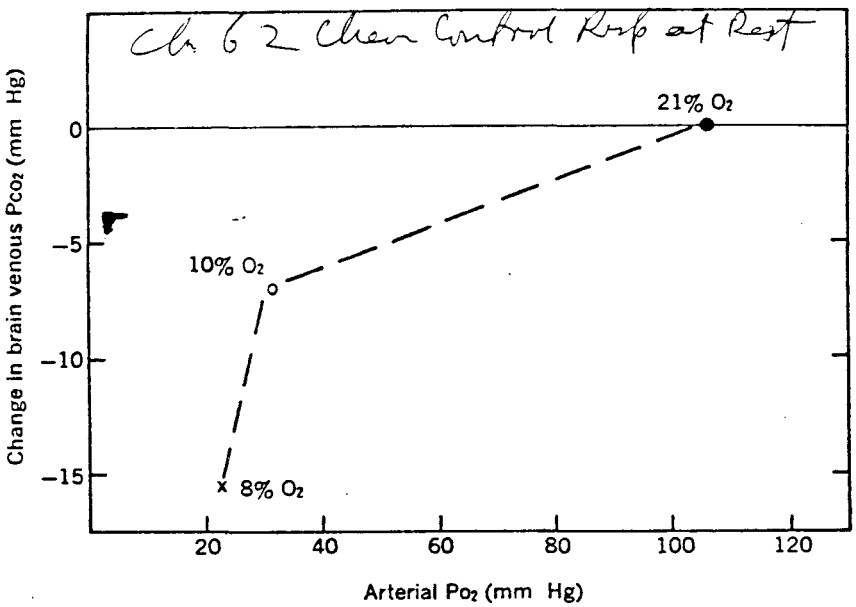


Fig. 62-24. Effect of low inspired O_2 tension on P_{CO_2} of brain venous blood.[96, 112] Breathing 10% and 8% O_2 in N_2 at sea level lowers central P_{CO_2} and $[H^+]$ by (1) respiratory stimulation (which lowers arterial P_{CO_2}) and (2) anoxemic dilatation of cerebral vessels (which by increasing blood flow further lowers central P_{CO_2} and increases pH). Hypoxia therefore lowers concentration of central respiratory stimulus at the same time that it induces chemoflex stimulation.

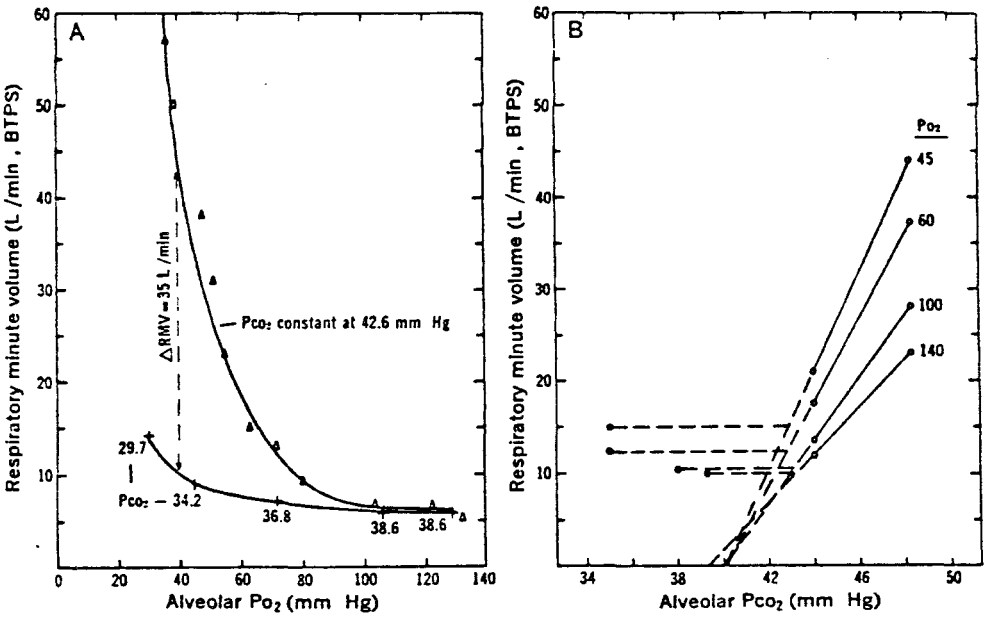


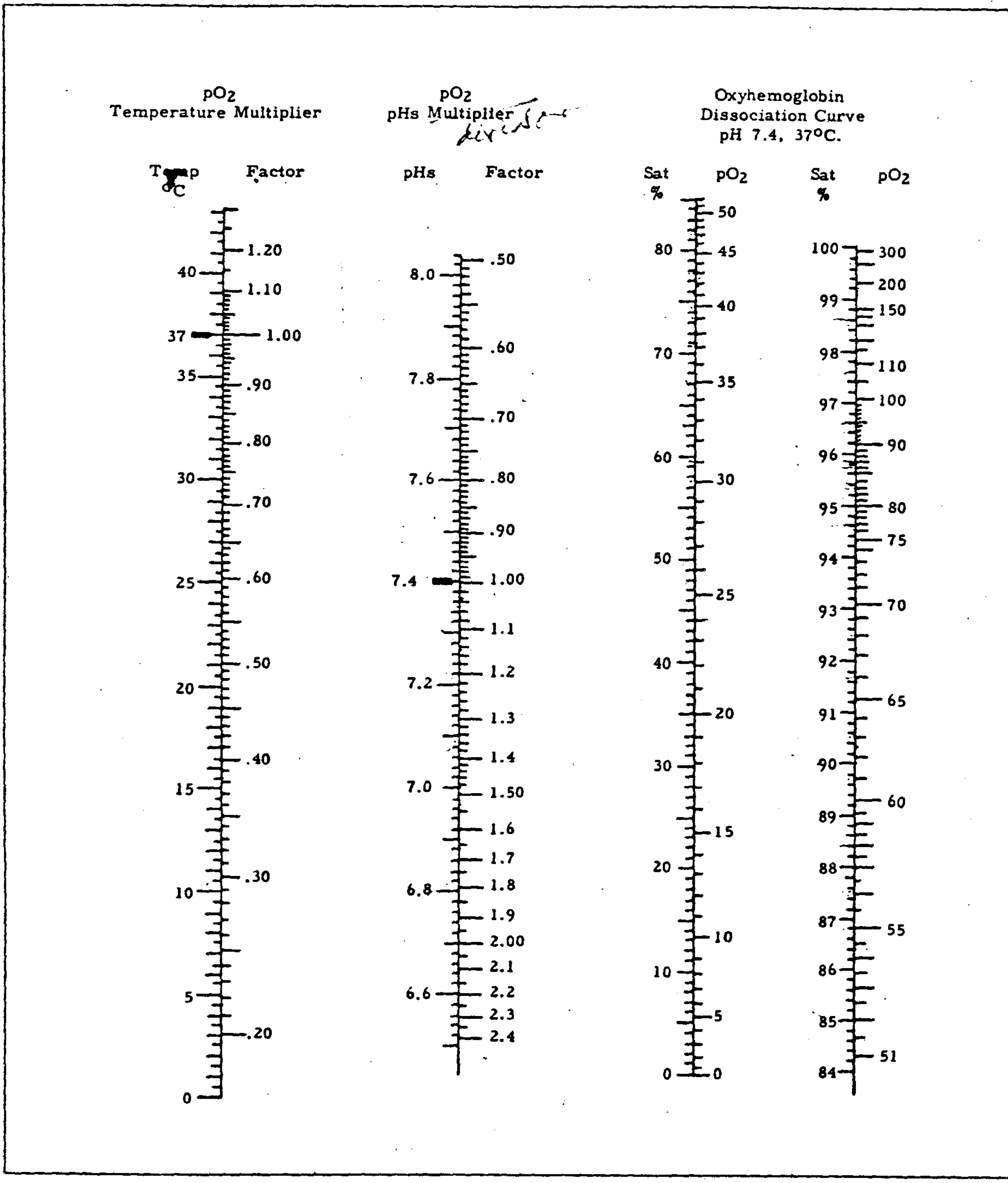
Fig. 62-25. Interaction of P_{O_2} and P_{CO_2} in respiratory control.

A, Influence of P_{CO_2} on respiratory response to O_2 lack. Lower curve shows that progressive lowering of alveolar P_{O_2} produces only slight respiratory stimulation which, apparently by lowering alveolar and central P_{CO_2}, tends to be self-limiting. Hypocapnia thus may mask anoxic drive. Upper curve, obtained over same range of hypoxia, but *while holding alveolar P_{CO_2} essentially constant*, indicates true capacity of chemoreceptor influence on respiratory control system. Difference between curves, e.g., 35 L/min at 40 mm Hg P_{O_2}, represents loss of reactivity of centers resulting from effects of difference in alveolar P_{CO_2} of approximately 8 mm Hg.

B, Influence of P_{O_2} on respiratory response to CO_2. Response to change in alveolar P_{CO_2} is steeper at low than at normal P_{O_2}. Horizontal, dashed lines are employed to suggest that level of ventilation during zero inspired P_{CO_2} will be unchanged by addition of CO_2 until threshold for CO_2 stimulation is reached.[126, 130, 153]

(Modified from Loeschcke and Gertz.[130])

Severinghaus

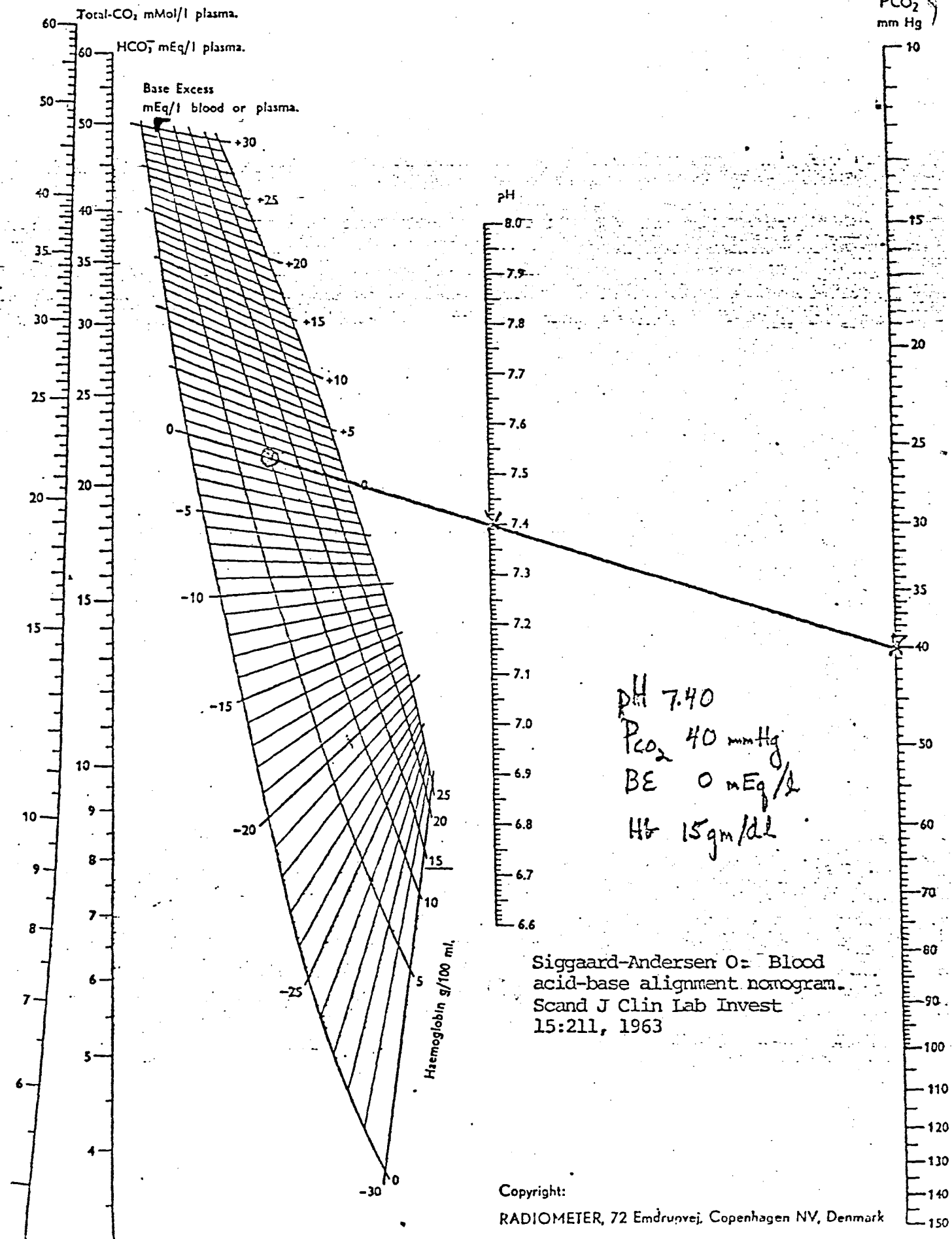


Contributor: Severinghaus, J. W.

[illegible] Dissociation Curve ([illegible]):
at pH 7.4, [illegible]
[illegible] at pH 7.6; /

Handbk Resp.
NAS/NRC WB Saunders 1958

SIGGAARD-ANDERSEN ALIGNMENT NOMOGRAM



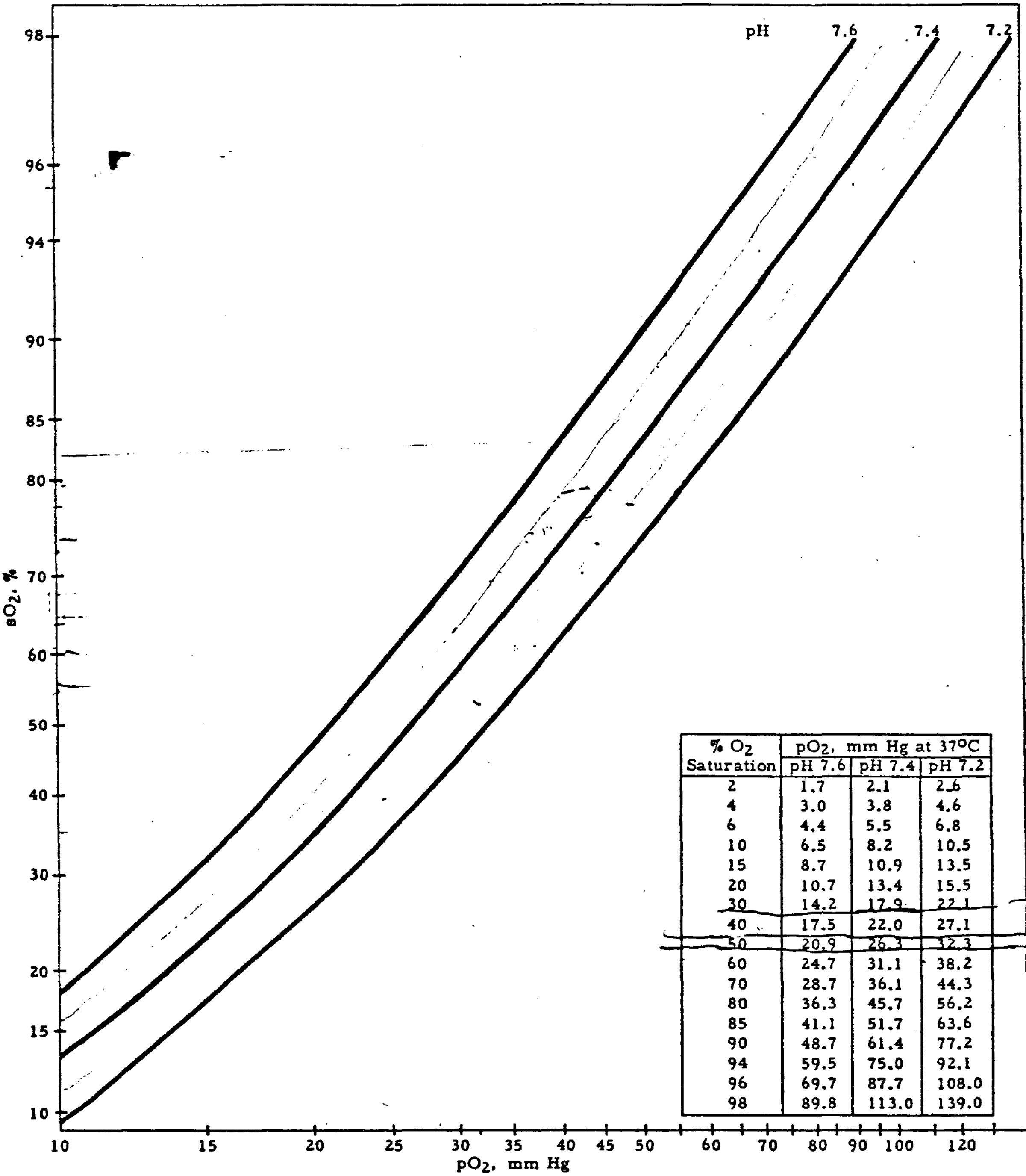
Siggaard-Andersen O: Blood acid-base alignment nomogram. Scand J Clin Lab Invest 15:211, 1963

Dill

71. BLOOD O_2 DISSOCIATION CURVES: MAN

Part I: AT VARIOUS pH VALUES

Theory and method of development of straight line curves given in headnote and in Parts I and II of Table 72.



% O_2 Saturation	pO_2, mm Hg at 37°C		
	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	15.5
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

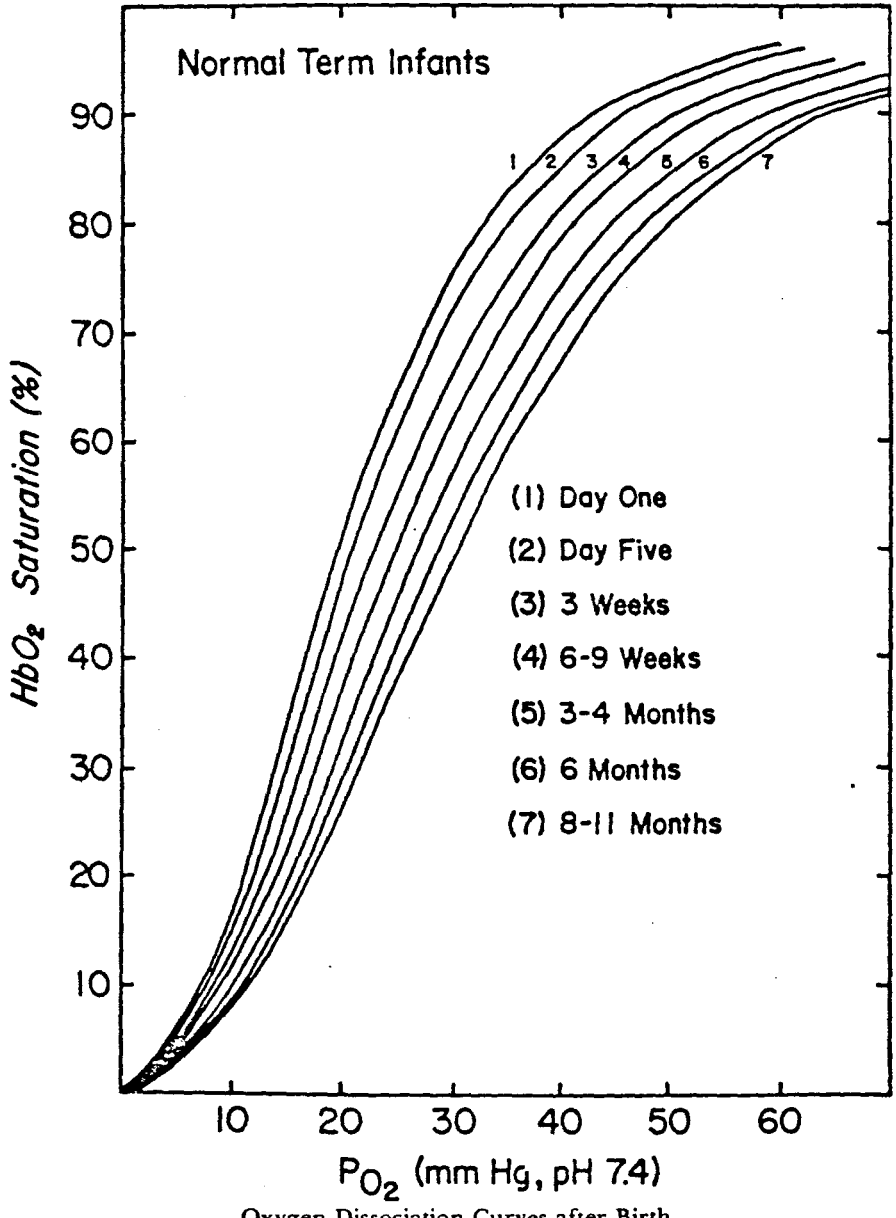
minimal $P_I O_2$

P_{50}

Contributors: Bartels, H., and Opitz, E.

Reference: Dill, D. B., in "Handbook of Respiratory Data in Aviation," Committee on Medical Research, Washington, 1944.

DPG fraction," which is the product of the concentration of adult hemoglobin and that of DPG in the red cells (25, 26, 42). The position (and therefore slope) of the dissociation curve is often expressed as the "P_{50}," or oxygen pressure in mm Hg exerted by 50 percent oxygen-saturated hemoglobin at 37° C, pH 7.4. The P_{50} of



Oxygen Dissociation Curves after Birth

Figure 3-6. The P_{50} on Day 1 is 19.4 ± 1.8 mm Hg and has shifted to 30.3 ± 0.7 at 11 months of age. (From Oski et al.—*J Pediatr*, 77:941, 1970) (42).

3.0 Methods and Normal Values

METHOD	NORMAL MEAN VALUES (+2SD) AT 37°C			
	Newborn[1] (age 24 hrs.)	Infant[2] (1-24 Mos.)	Child[3] (7-19 Yrs.)	Adult[4]
pH - Glass electrode	7.37 (.06)	7.40 (.06)	7.39 (.02)	7.40 (.03)
$PaCO_2$ - CO_2 electrode	33 (6)	34 (8)	37 (3)	39 (5)
- Interpolation (Astrup)				
- Riley bubble				
- Nomogram + T_{CO_2}, pH				
BE - Nomogram + pH, PCO_2	-6.0* (3)	-3.0 (3)	-2.0* (2)	0.0 (2)
T_{CO_2} - Van Slyke	21* (3)	21 (4)	23* (2)	26 (4)
- Kopp-Natelson				
- Nomogram + pH, PCO_2				
(HCO_3^-) - Calculation (T_{CO_2} - .03 P_{CO_2})	20 (3)	20* (4)	22* (2)	25* (4)
- Nomogram + pH, P_{CO_2}				

1. Koch, G. and Wendel, H.: Biol. Neonat. 12:136, 1968

2. Albert, M.S. and Winters, R.W.: Pediatrics 37:728, 1966

3. Levison, H., Featherby, E.A., and Weng, T.R.: Am. Rev. Resp. Dis. 101:274, 1970

4. Siggaard-Andersen, O.: The Acid-Base Status of the Blood. Williams and Wilkins, Baltimore, 1964 (2nd ed.).

* Values calculated from author's data.

H-11's data Ch. 31 Handbk Resp. I
Transport O2/CO2
FJW Roughton

A. HILL'S EQUATION. This equation was first put forward in 1910 before the molecular weight and number of iron atoms in hemoglobin were known. It was assumed that the molecule of hemoglobin contained n atoms of iron, and that the equilibrium could therefore be formulated by the equation

$$Hb_n + nO_2 \rightleftharpoons (HbO_2)_n$$

Application of the law of mass action then led to the equation

$$\frac{y}{100} = \frac{KP^n}{1 + KP^n} \tag{11}$$

where y is the per cent saturation with oxygen, P is the partial pressure of oxygen, and K is an equilibrium constant. Similarly for the equilibrium between carbon monoxide and reduced hemoglobin

$$\frac{z}{100} = \frac{Lq^n}{1 + Lq^n} \tag{12}$$

where z is the per cent saturation with carbon monoxide, q is the partial pressure of carbon monoxide, and L is an equilibrium constant.

The early data of Barcroft were well fitted by this equation, the value of n lying between 2 and 3. These data, however, were subsequently found to contain certain systematic errors; when these are allowed for, the fits are much less satisfactory. Hill's equation was, furthermore, deprived of its theoretical basis when it was discovered that the molecular weight of hemoglobin was 64,800 and therefore that n should have been 4, instead of the value between 2 and 3 which empirically fitted the early dissociation curves. Hill's equation is still, however, much used as an empiric relation, since it has the convenience of containing only two arbitrary constants, i.e., K and n. Its advantages and limitations must accordingly be examined further.

Equation 11, by simple algebraic rearrangement, can be changed to the form

$$\frac{y}{100 - y} = KP^n \tag{13}$$

Taking logarithms of both sides of equation 13 gives

$$\log\left(\frac{y}{100 - y}\right) = \log K + n \log P \tag{14}$$

A simple plot of log $(y/[100 - y])$ against log P should therefore yield a straight line making an angle, with the log P axis, of tangent equal to n, and an intercept on the log $(y/[100 - y])$ axis equal to log K. In this way the values of the two constants n and K are simply determinable from a series of points on the oxyhemoglobin dissociation.

TABLE 2. *Application of Hill's Equation to Oxyhemoglobin Dissociation Curve Data on Human Blood at pH 7.4, 37 C*

y (% Saturation)	P (Oxygen Pressure)	Log $\left(\frac{y}{100-y}\right)$	Log p
2	2.1	$\bar{2}.310$	0.322
4	3.8	$\bar{2}.620$	0.580
6	5.5	$\bar{2}.805$	0.740
10	8.2	$\bar{1}.046$	0.914
15	10.9	$\bar{1}.247$	1.037
20	13.4	$\bar{1}.398$	1.127
30	17.9	$\bar{1}.632$	1.253
40	22.0	$\bar{1}.824$	1.342
50	26.3	0.000	1.420
60	31.1	0.176	1.493
70	36.1	0.368	1.557
80	45.7	0.602	1.660
85	51.7	0.753	1.713
90	61.4	0.954	1.788
94	75.0	1.195	1.875
96	87.7	1.380	1.943
98	113.0	1.690	2.053

Table 2 shows the application of this procedure to the data on the dissociation curve at pH 7.4, which have already been plotted in figure 6. The values of log $(y/100 - y)$ are then graphed against log p as shown in figure 12*A*. The complete set of points so obtained do not fall on a straight line, although they do so reasonably well within the range of 20 to 98 per cent saturation—i.e., the part of the dissociation curve that is of main physiological interest. Within this range n is approximately equal to 2.7, which is close to the original value given by Hill. Below 20 per cent saturation, however, the points diverge increasingly from the $n = 2.7$ line and, indeed, tend to approach a line corresponding to $n = 1$. The dotted extension of figure 12*A* above 98 per cent saturation is derived not from direct determinations but from calculations based on Adair's intermediate compound theory, described in the next section. According to this theory the curve should approximate to a straight line, corresponding to $n = 1$, not only in the bottom range but also in the top range. It is not at present experimentally possible in the physiological pH range (7.0 to 7.6) to work at high enough saturations to demonstrate the effect directly in the top range, but it can be—and has been—verified by especially accurate work at alkaline pH, as figure 12*B* for 4 per cent sheep hemoglobin at pH 9.1, 19 C shows. The failure of Hill's equation at one or both ends of the curve is

Downes Ex # 6
10/7/81
JJM.

JAMES G. GAUME, M.D.
CONSULTANT IN HUMAN FACTORS
JOHN J. DOWNES, M.D. 1517 ESPINOSA CIRCLE
PALOS VERDES ESTATES, CALIFORNIA 90274
(213) 375-6607

G force - med

August 31, 1981

Carroll E. Dubuc, Edq.
HAIGHT, GARDNER, POOR & HAVENS
Federal Bar Building
1819 H Street, N.W.
Washington, D.C. 20006

RE: FFAC v. Lockheed Aircraft Corporation
Your File No. 2041-1278-2S

Dear Mr. Dubuc,

In accordance with your original request, I have reviewed the testimony given by Dr. Busby in the Schneider trial, in which he stated that, as the editor of selected published papers in the Proceedings of the XVIII International Congress of Aviation and Space Medicine, it did not mean necessarily that he agreed with the concept of the "Time of Safe Unconsciousness following Decompression," which I proposed in that paper which was published in his "Proceedings." Other knowledgeable aerospace medical experts, however, did agree with the TSU concept as it was presented in 1969. Apparently, at a later date, when he was at the Civil Aeromedical Institute in Oklahoma City, OK, Dr. Busby saw fit to consult me by telephone regarding the experimental design of altitude chamber experiments, which he was planning, on the ability of female flight attendants to perform physical workload at cabin altitude and during a decompression and the accompanying hypoxia. His results were later published in Aerospace Medicine.

At a later date, you requested that I perform my own calculations with regard to three factors involved in the C5A SN68-218 crash in Saigon on April 4, 1975:

1) The injury potential to the orphans involved in the crash landing and deceleration of the C5A aircraft;

2) The significance of the total pressure change and rate of change during the decompression from 5,000 ft. to 23,500 ft.;

3) The import of the hypoxia resulting from the decompression on the passengers in the troop compartment.

Also, you requested that I review other testimony, documents, calculations and statistics which you supplied to me. I have researched these data, performed the analyses and calculations, and hereby submit my report in three sections. For Section A on decelerations, I asked the assistance of Mr. Roy Jablonsky, P.E., a recognized

expert on accident analysis and reconstruction, to calculate the G-forces involved. I also asked him to calculate the G-forces of selected amusement park rides which impose G-forces in the same direction on the rider as those imposed in the decelerations for the occupants of the troop compartment. His calculations are very close to those of John Edwards'. I have examined both calculations and I adopt those of Edwards and Jablonsky and base my opinion on those calculations.

Section A of my reports deals with the decelerations experienced by the occupants of the troop compartment.. Mr. Jablonsky's calculations for the G-forces imposed during the amusement park rides are also in Attachment A-1. As you can see, the G-forces for the rides, experienced by literally thousands of amusement park patrons every year, are far in excess of those felt by the occupants of the troop compartment. In my opinion, the G-forces imposed in the G_Z (vertical) or the G_X (horizontal - transverse to the long axis of the body) were <u>not</u> injurious to any of the orphans in the troop compartment, seated in rear-facing seats and fully supported by the seat-back and restrained adequately by seatbelts and pillow padding, to a reasonable medical certainty.

Section B of my report considers the total pressure change, and the rate of change, experienced by the orphans and adults in the troop compartment. Attachment B-1, from the book <u>Aerospace Medicine</u>, by Armstrong, a recongized expert in that field, states that the pressure change is not responsible for the physiological effects of decompression, but to oxygen deprivation (see Section C-Hypoxia Effects). In my opinion, the total pressure change, the rate of change, and the duration of change, did not produce any harmful, lasting physiological effects, to a reasonable medical certainty, on anyone in the troop compartment.

Section C of my report analyzes the physiological effects of the hypoxia imposed by this decompression, and details the reasons why no significant effects were endured by those subjected to the event. Because of the compensatory, protective mechanisms inherent in the human body, in infants as well as in adults, the increased blood supply to the brain prevented any brain damage, to a reasonable medical certainty. Attachments C-1, -2, -3 and -4, provide ample support for this opinion.

I appreciate the opportunity to be of service in this matter. Should additional assistance be required, please feel free to call on me.

Sincerely,

James G. Gaume, M.D.
Human Factors Consultant

JAMES G. GAUME, M.D.
CONSULTANT IN HUMAN FACTORS
1517 ESPINOSA CIRCLE
PALOS VERDES ESTATES, CALIFORNIA 90274
(213) 375-6607

REPORT A

ANALYSIS OF THE ACCELERATIONS INVOLVED IN THE DECOMPRESSION AND CRASH LANDING EVENT OF C5A SN68-218 ON 4 APRIL 1975

During the very rapid decompression and the descent to the ground, there were no significant accelerations. At first touchdown of the aircraft, the rear main landing gear wheels dug three feet into the soft ground and were wiped off, but the impact was barely noticeable by those in the troop compartment and in the flight deck. The aircraft was in contact with the ground during this touchdown for a distance of approximately 1100 ft., then became airborne again, flew 2700 ft. through the air and contacted a 5 ft. dike at the far edge of the Saigon River. The front wheels of the main landing gear passed through the dike and were wiped off. Again, the impact was barely noticeable by those in those same compartments. The aircraft then settled into the ground, slid for some distance, at which point the troop compartment separated from the fuselage, the plane broke into four main segments, each going in slightly different directions. The troop compartment traveled a total of 2012 ft. before coming to rest, right side up. The average G-force experienced by the people in the troop compartment was 1.6 G transverse to the bodies of those in the compartment. This is about twice the G-force felt by passengers in a jet airliner as it accelerates down the runway for a normal takeoff, and is in the same direction on the body as the G-force experienced by those

in the troop compartment, from front to back, or $+G_x$.

These G-forces are considerably lower than those experienced by riders of a number of amusement park rides. For example, a ride called the ROTOR is a vertical, 14 ft. diameter cylinder containing a floor on which people stand. The cylinder (cage) is spun up to a maximum of 35 rpm, and the floor is dropped down 3 to 4 ft. The centrifugal force flattens the rider's back against the outer wall of the cage and is strong enough to keep him there, and the G-force is calculated to be 2.89G's. Another ride, variously called the ELECTRIC RAINBOW or the ROUNDUP, contains the cars on the end of a 20 ft. arm which rotates around the center hub, moving up and down as it rotates at a miximum of 15 rpm, producing a force on the passenger of 1.53G's. Another ride which puts the car through a loop the loop, starts the ride with a catapult thrust producing 4.5 G's on the passenger. All three of these rides apply the G's in the same direction on the body as the 1.6 G's experienced by the orphans and adults in the troop compartment in this case. The beginning of the loop in the last ride mentioned produces a vertical G-force of 6.2G's, and the average vertical G at the bottom of many of the newer roller coaster dips is well over 3G's. (See Attachment A_1)

The catapult on an aircraft carrier which launches a jet fighter applies 5.57 G's to the pilot, which is 3.45 times

the average 1.6 G's felt by the occupants of the troop compartment.

James G. Gaume M.D.

James G. Gaume, M.D.
Human Factors Consultant
30 August 1981

ATTACHMENT A-1

R. D. JABLONSKY, INC.
CONSULTING ENGINEER
POST OFFICE BOX 672
ALTADENA, CALIFORNIA 91001
798-6100 · 681-8444

August 31, 1981

Dr. J. G. Gaume
1517 Espinoza Circle
Palos Verdes, California 90274

Re: Deceleration Analysis
C-5A Serial No. 68-218
April 4, 1975

Dear Dr. Gaume:

In accordance with your request, an analysis has been made of the data which you furnished which described the descent profile, flight information and crash scene information concerning the crash landing of the C-5A, Serial No. 68-218 which occurred on April 4, 1975. The purpose of the analysis was to determine the probable level of the accelerations experienced by persons seated in the troop cargo compartment. The analysis considered the descent from an altitude of approximately 23,400 feet to the point of first contact with the ground as one part and as a second part the trajectory from first point of contact with the ground to the point of rest of the troop compartment. The information which you furnished and upon which my analysis was made is herewith attached as Appendix A.

According to the altitude time history supplied in graphical form the aircraft descended from an altitude of approximately 23,400 feet to approximately 600 feet in approximately 15 minutes.

During this interval of time there were fluctuations in the descent rate. To determine the vertical accelerations experienced as a result of the recorded fluctuations in descent rate the incremental variations in vertical velocity, vertical acceleration and vertical rate of onset were calculated. From a study of the altitude time history curve a time interval of 7½ seconds was selected as a basis for calculating the velocity, acceleration and rate of onset from the available data. Using a shorter time interval as a basis for calculations would not have yielded any more meaningful information from the graphical data available. The results of this analysis showed that the maximum vertically up acceleration experienced was approximately 10.7 feet per second/per second (0.33g) occurring at approximately 8,900 feet altitude and the maximum downward acceleration experienced was 14.22 feet per second/per second (0.44g) occurring at approximately 7,800 feet. The maximum rate of offset experienced was no greater than 0.1g's/second. The results of these calculations are herewith included in Attachment 1. This attachment sets forth the numerical results as provided by the altitude time history curve at 7½ second intervals. In addition to the vertical velocity, acceleration and rate of onset the tabulation also provides the total atmospheric pressure and the partial pressure due to oxygen. These pressures are given in millimeters of mercury. The altitude pressure relationship was based upon standard atmospheric conditions. The partial pressure due to oxygen is based upon an oxygen percentage of 20.95.

Between the first and second points of contact with the ground the aircraft traveled a total distance of 2,700 feet. Reportedly,

there was no significant change in air speed (310 mph - 455 ft/sec) between the first and second points of contact with the ground. At the reported speed this distance was traveled in approximately 6 seconds. It is my understanding that the engines could not be effective within this interval of time. Thus, between and including the first and second points of ground contact to be consistent with the constant air speed no significant decelerations were experienced.

The wreckage diagram depicts the section of cargo floor coming to rest at a point approximately 1,400 feet from the second impact location. Reportedly, at 1,200 feet from the second impact location break-up of the aircraft occurred. Thus, the troop compartment and the cargo floor decelerated at the same rate from the second impact position to the point of break-up (1,200 feet). As previously noted the cargo floor moved an additional 400 feet. The troop compartment moved an additional 812 feet (2012-1200 = 812). From this information deceleration rates from the second impact location can be calculated. The analysis shows that during the 1,200 feet from second impact location to the point of break-up the average rate of deceleration was 74 feet per second/per second (2.30g's). The time elapsed to traverse this distance at the computed rate of deceleration was approximately 5.69 seconds. At the end of this time when break-up occurred the velocity had decreased to 172 feet per second (117 mph). From the point of break-up the troop compartment traveled an additional 812 feet to its point of rest. The constant rate of acceleration necessary to traverse this distance from the speed of 172 feet per second was approximately 18 feet per second/per second (0.57g's). The time for the troop

compartment to traverse the final 812 feet based upon the average deceleration rate of 18 feet per second/per second was approximately 9.44 seconds. The calculations yielding the above-mentioned figures are included in Attachment 2.

The vertical acceleration rates experienced during the flight descent and the horizontal deceleration rates experienced after ground contact were compared to accelerations and decelerations in these directions by thrill ride apparatus commonly found in amusement parks. Several different types of rides were considered. In the typical roller coaster at the bottom of the dips between 2.5 and 3g's vertical acceleration is experienced. At the top of the curve the negative acceleration is usually approximately 1g. Due to the vertical radius of curvature of the track and the speed traveled the rates of onset are usually in excess of 3g's per second.

An amusement ride consisting of a 14 foot diameter cylinder which turns on its vertically positioned axis at a speed of 35 revolutions per minute the floor can be lowered after the speed has been reached. The centrifugal force causes the occupants to be forced against the inside wall of the cylinder. The force is sufficient such that the frictional resistance will prevent the occupants from sliding vertically downward. Thus, the floor can be lowered and the occupants are held against the wall of the cylinder as a result of the centrifugal force. The centrifugal acceleration developed results in 2.89g's. The duration of the force is usually more than 60 seconds.

In a roller coaster-type ride in which the track makes a complete vertical circle the car is accelerated to approximately 4.2g's reaching a speed of between 50 and 60 miles per hour within a distance of approximately 160 feet. This section of the track is horizontal. Thus, the acceleration is in the direction of travel. In traveling through the vertical curve the centrifugal acceleration attained is 6.5g's.

In a ride in which the occupants sit in a car located at the end of a 20 foot radius arm the arm makes 15 revolutions per minute. There are several different varieties of this type of ride. In some instances the car will oscillate in a vertical plane and in others the oscillation of the car will be in the horizontal plane as it rotates. Thus, the occupants will experience the cnetrifugal force through an infinite number of horizontal body positions. Typically the centrifugal acceleration for rides of this type is 1½g's.

Calculations based upon several rides found in amusement parks in the Southern California area are included in Attachment 3.

C O N C L U S I O N

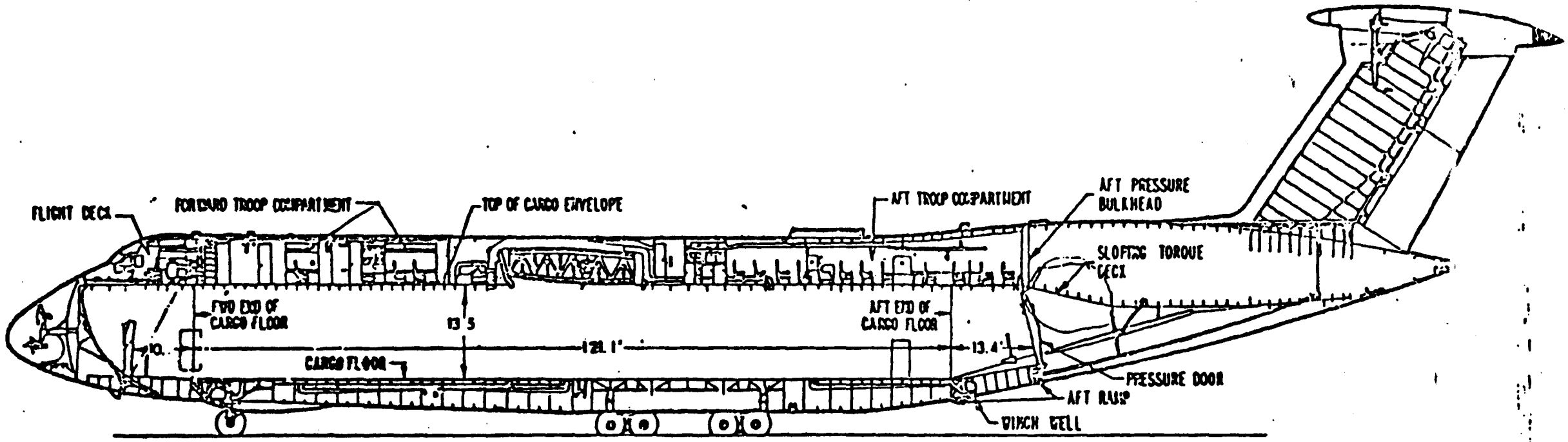
The dynamic forces experienced by the occupants in the cargo compartment during the flight descent phase were probably less than those necessary to be sensed by the occupants. During the crash-landing and the deceleration of the aircraft to the points of rest the rates of onset and the deceleration levels reached by

the troop compartment were significantly less than those experienced in thrill rides commonly found in amusement parks.

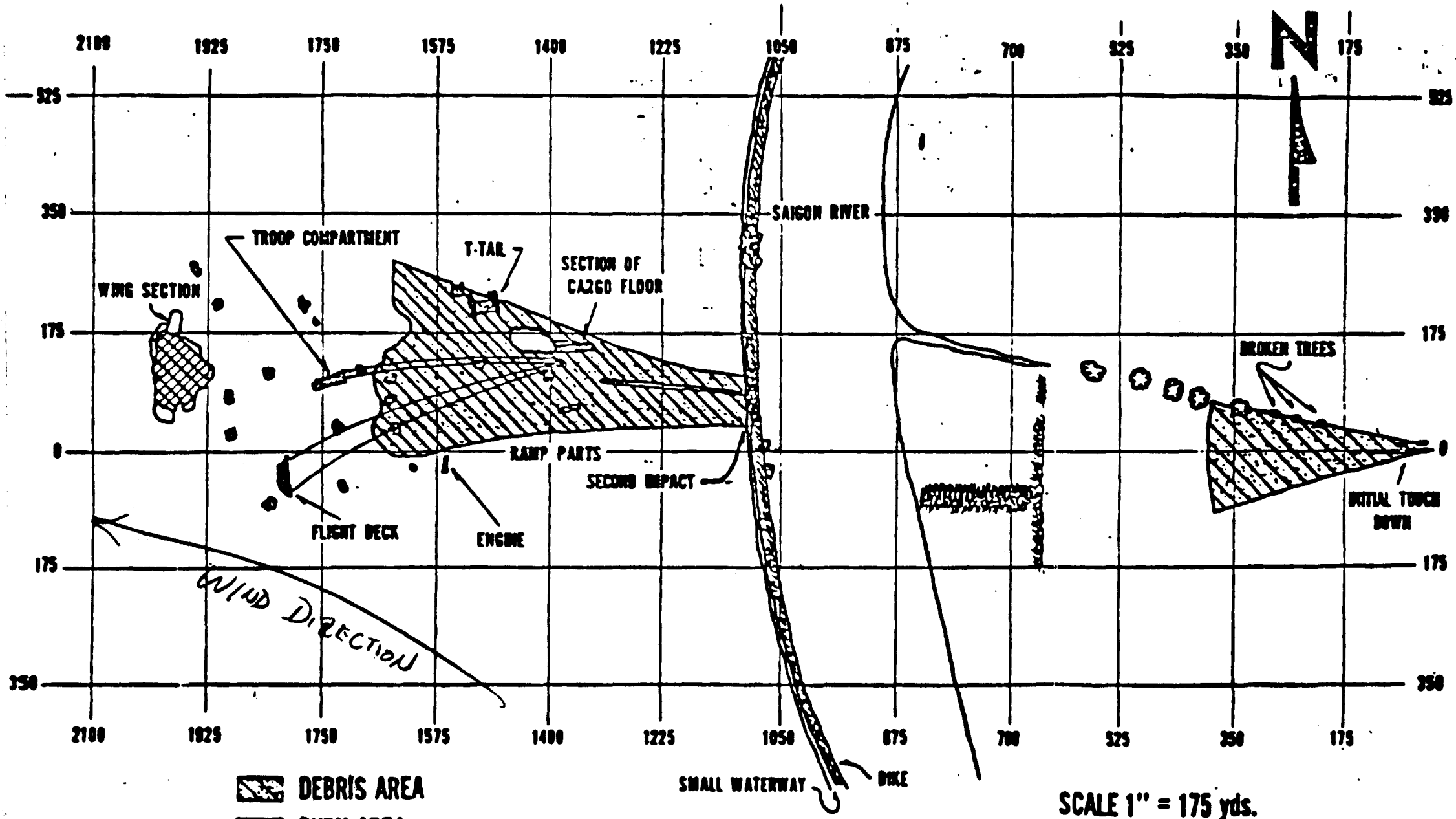
Respectfully submitted,

R. D. Jablonsky, P.E.
Registered Professional Engineer
California License No. 3775

RDJ/dh


FLIGHT DECK
FORWARD TROOP COMPARTMENT
TOP OF CARGO ENVELOPE
AFT TROOP COMPARTMENT
AFT PRESSURE BULKHEAD
SLOPING TORQUE DECK
FWD END OF CARGO FLOOR
13'5
AFT END OF CARGO FLOOR
10.
121.1'
13.4'
CARGO FLOOR
PRESSURE DOOR
AFT RAMP
WINCH WELL

WRECKAGE DIAGRAM
C-5A SN 68-218 4 APRIL 1975



APPENDIX A

For Roy — C5A Crash - Saigon 1975

1. Airspeed @ 1st touchdown = 270 knots (310 mph)
   " @ 2nd " = 270 knots

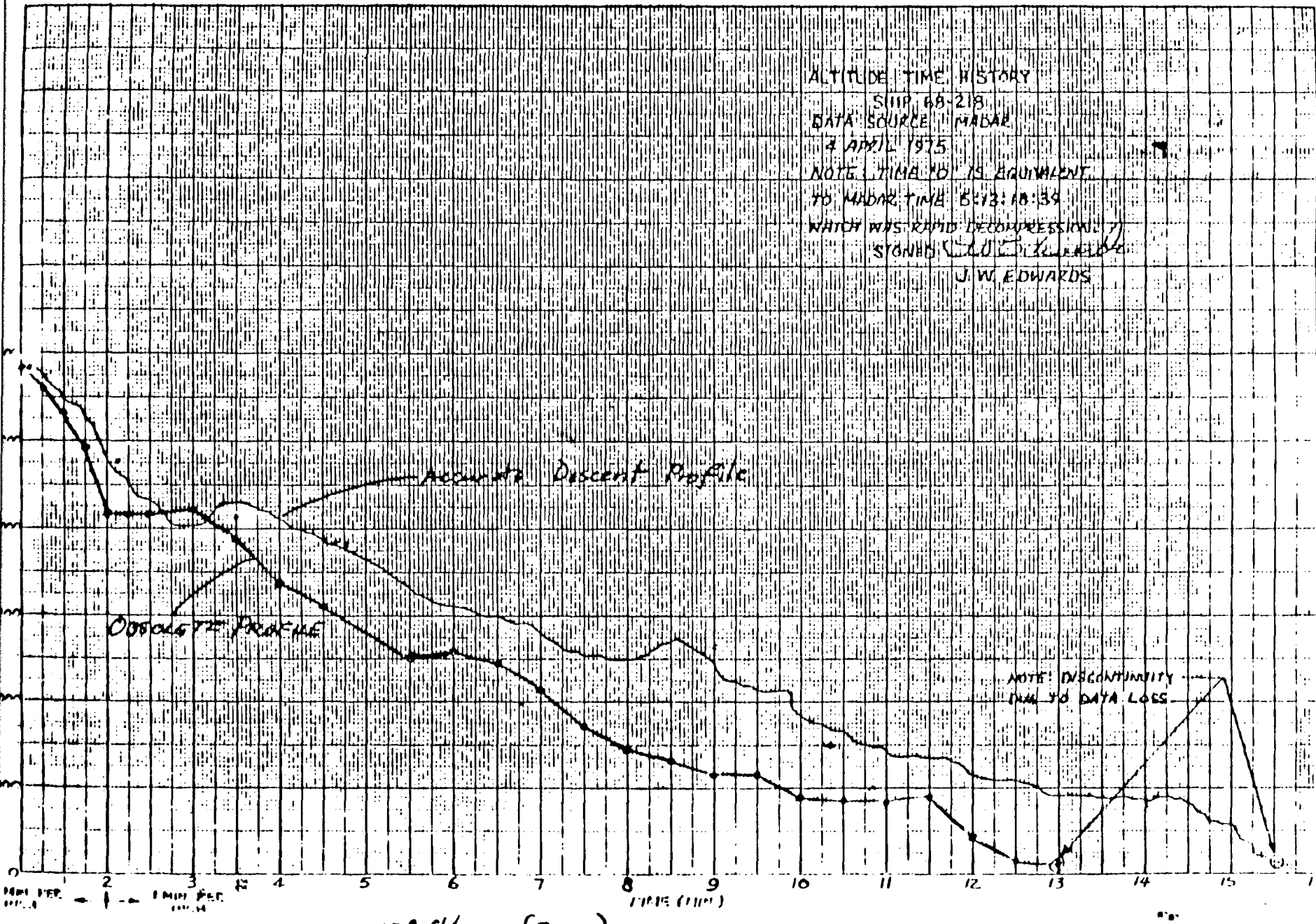
2. Descent rate (prior to flare) = 500 - 600 ft/min
   Flare due to air cushion (ground effect) under wing

3. 1st ~~Impact~~ touchdown — rear main landing gear dug 3ft into grassy ground, deceleration forces low (G_x axis?), rolled & skidded 1000 ft, & became airborne, climbed at 12° angle, travelled 2700 ft & impacted dike on far side of river, lost rear main landing gear on impact.

4. 2nd ~~impact~~ touchdown — Front m.l.g. cut thru 5' hi & 5' wide dike on far side of river & was wiped off A/C. No evidence of G_z accelerations on front m.l.g. on inspection. A/C contacted ground (rice paddy) & skidded 1200 ft where it broke into 4 major sections — troop cmpt, flight deck, wing, & tail + smaller sections of cargo cmpt. Troop compt came to rest at 2012 ft after 2nd touchdown.

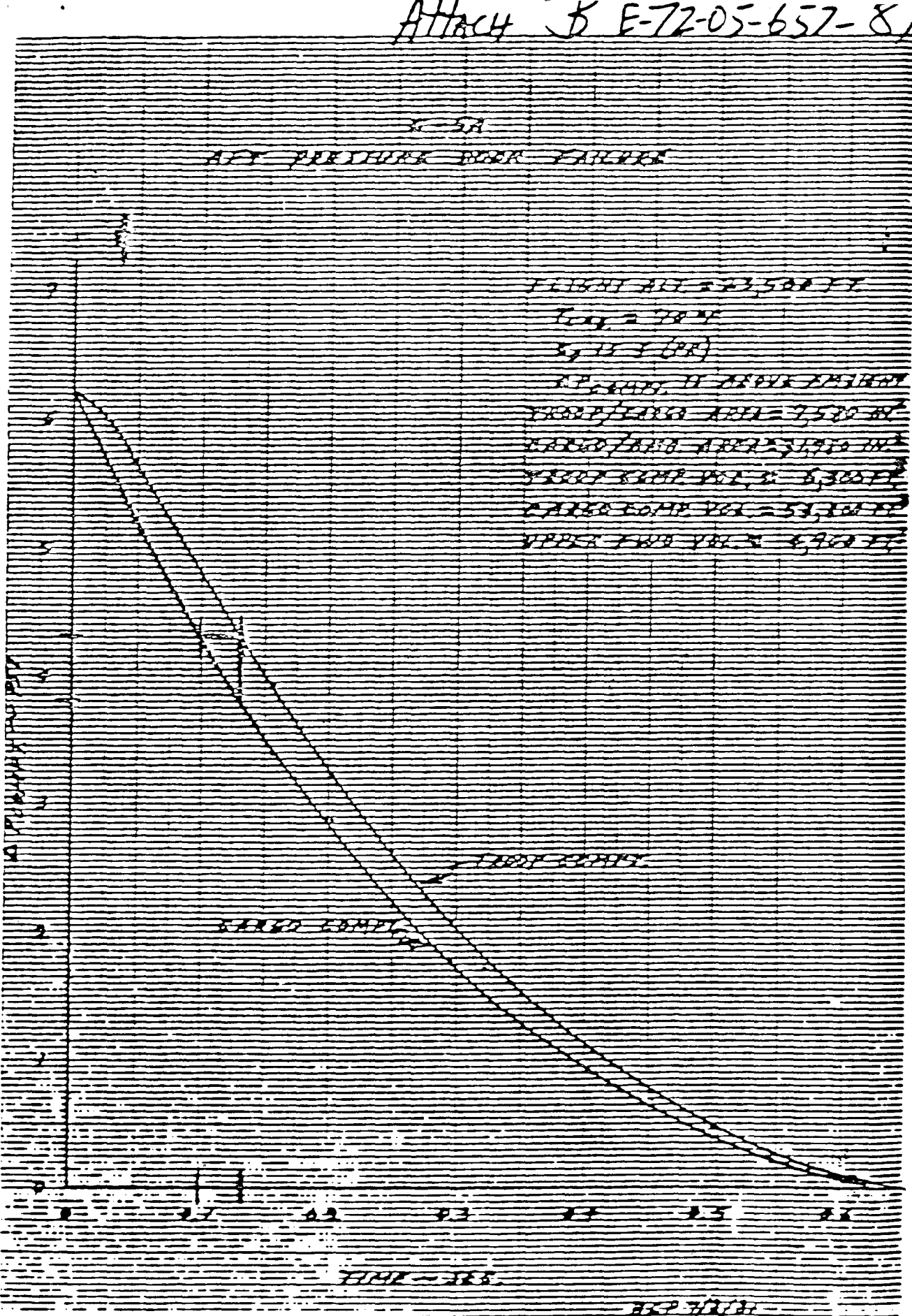
5. Aircraft Wt =

6. Troop Compartment litt - Unknown



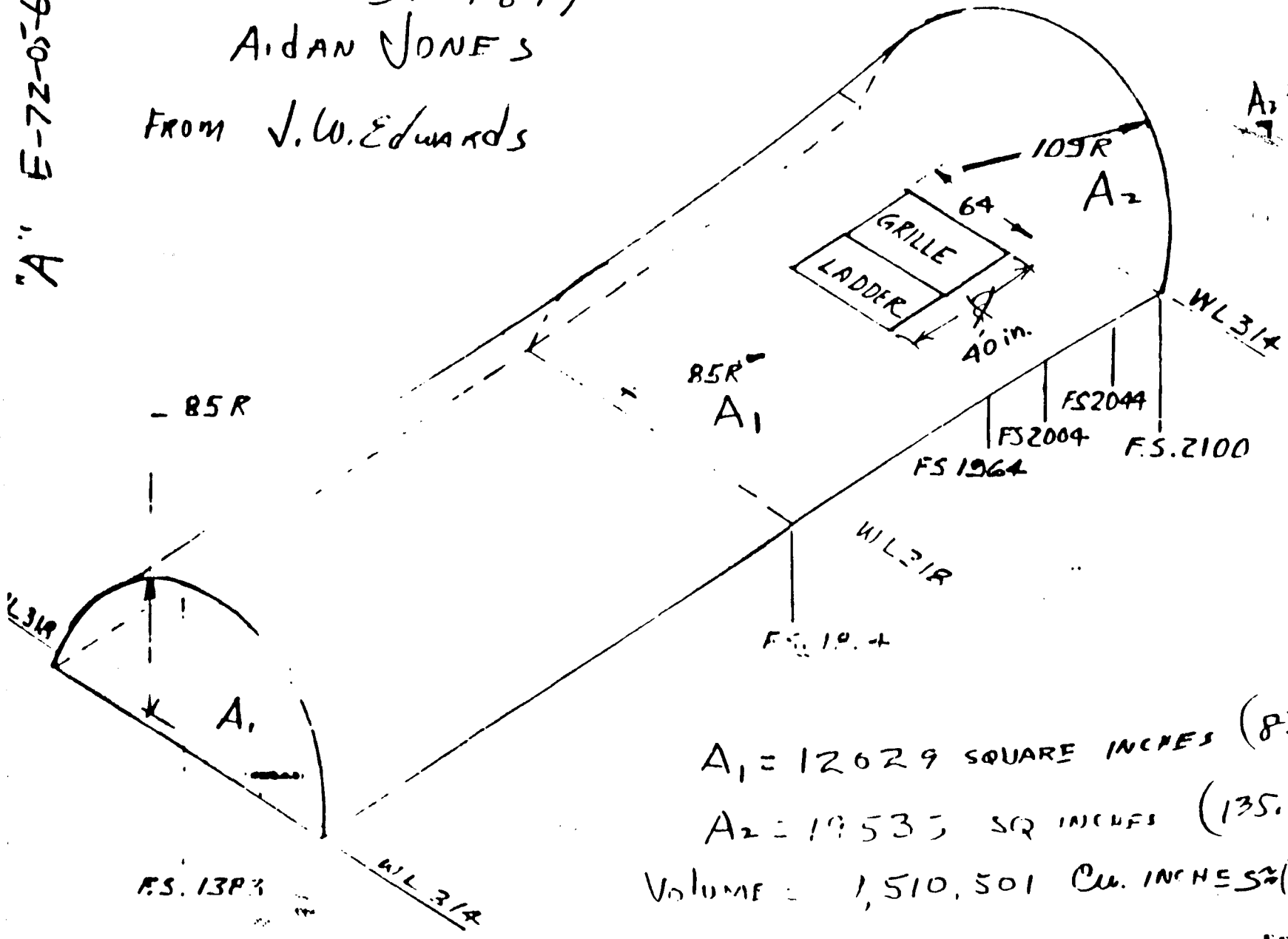
Av. Descent Rate 1st 3 min = 2,400 ft/min (approx)
3-8 min = 1035 ft/min (")
8-10(15) = 1300 ft/min (")

Attach B E-72-05-657-81



"A" E-72-05-637-W

To:
202-737-7849
AIDAN JONES
FROM J.W. Edwards

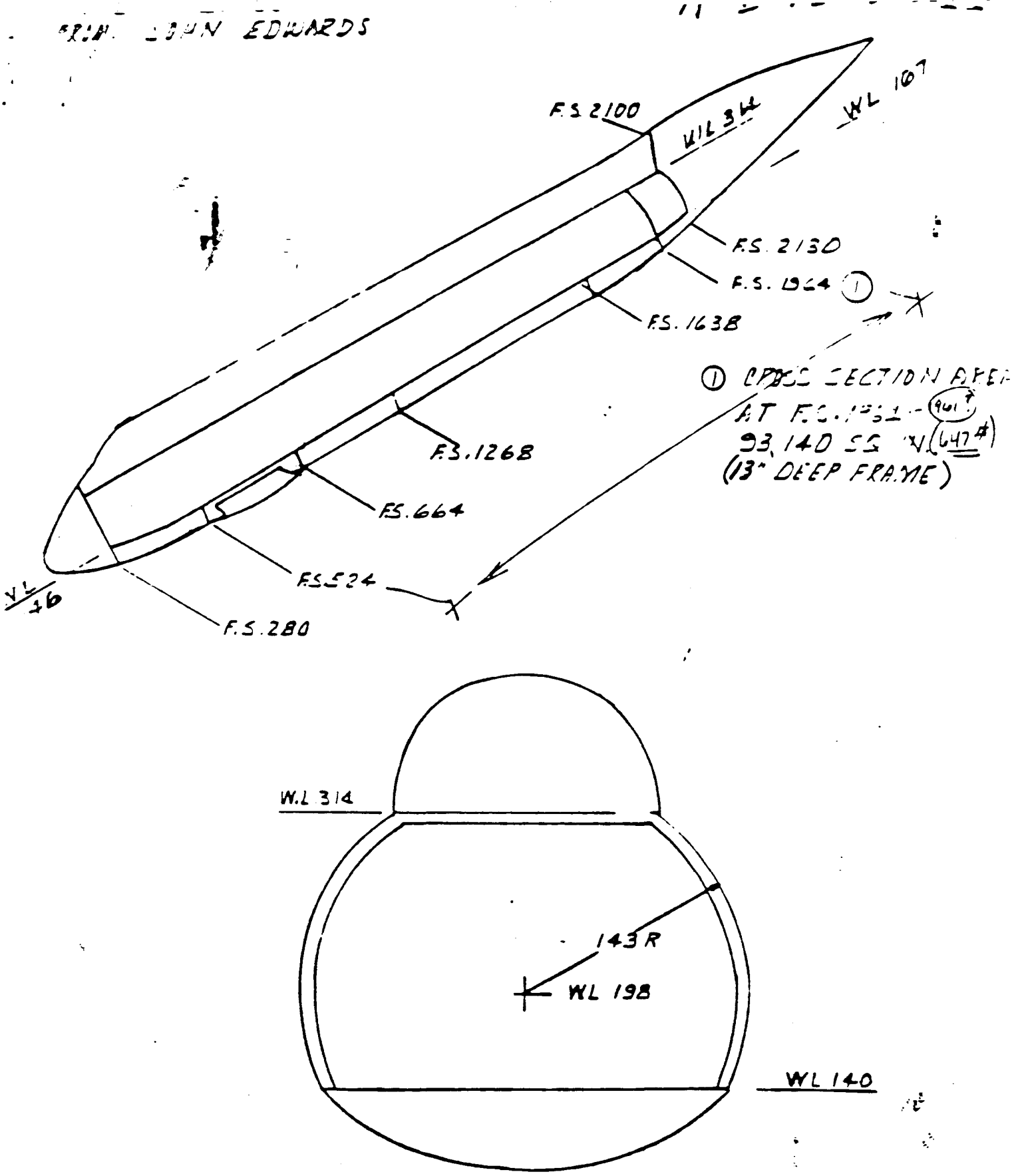


A_1 = 12029 SQUARE INCHES (83.5 ft²) 1:1.6

A_2 = 19535 SQ INCHES (135.7 ft²)

VOLUME = 1,510,501 CU. INCHES ≈ (875 cu ft)

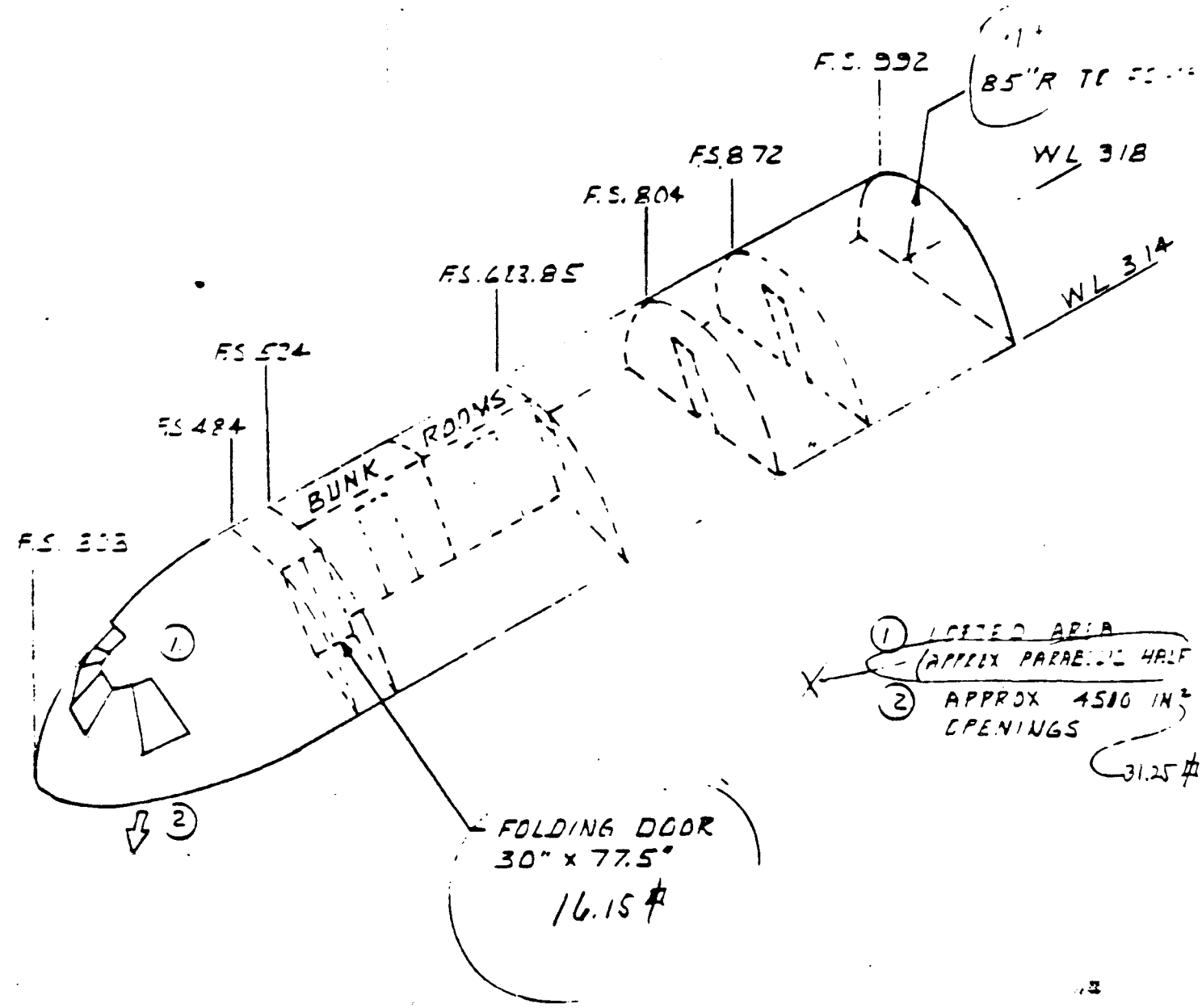
AFT TROOP COMPARTMENT



CARGO COMPARTMENT

TEL. 332-737-7839
AIDAN JONES
FROM JOHN EDWARDS

A E-7205-657-8



C-5A FLIGHT STATION

ATTACHMENT 1

VERTICAL VELOCITY, ACCELERATION AND RATE OF ON SET CALCULATION FROM ALTITUDE HISTORY OF SHIP 68-218, GRAPH OF J W EDWARDS NUMERICAL DIFFERENTIATION BASE UPON 7.5 SECOND TIME INTERVALS

(MINUS INDICATES VERTICALLY UP)

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC.	ACCELERATION FT/SEC^2	ON SET RATE g/SEC
0	0	23300	−6.67	−.89	—
.13	7.5	23350	−6.67	0	0.004
.25	15.0	23400	13.33	2.67	0.01
.38	22.5	23300	40.00	3.56	0.004
.50	30.0	23000	26.67	−.78	0.02
.63	37.5	22800	40.00	1.78	0.01
.75	45.0	22500	26.67	−1.78	0.01
.88	52.5	22300	53.33	3.55	0.02
1.00	60.0	21900	13.33	−5.33	0.04
1.13	67.5	21800	26.67	1.78	0.10
1.25	75.0	21600	26.67	0	0.007
1.38	82.5	21400	53.33	3.55	0.01
1.50	90.0	21000	26.67	−3.55	0.03
1.63	97.5	20800	66.67	5.33	0.04
1.75	105.0	20300	93.33	3.55	0.007
1.88	112.5	19600	66.67	−3.55	0.03
2.00	120	19100	73.33	0.89	0.02
2.13	127.5	18550	60.00	−1.78	0.01
2.25	135.0	18100	93.33	4.44	0.03
2.38	142.5	17400	33.33	−8.00	0.05
2.50	150.0	17150	46.67	1.78	0.04
2.63	157.5	16800	73.33	3.55	0.007
2.75	165.0	16250	13.33	−8.00	0.05
2.88	172.5	16150	0	−1.78	0.03
3.00	180.0	16150	−20.00	2.90	0.02
3.13	187.5	16300	−66.67	6.22	0.01
3.25	195.0	16800	−40.00	−3.56	0.04

TIME, MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC	ACCELERATION FT/SEC^2	ONSET RATE g/SEC
3.38	202.5	17100	-13.33	-3.56	0
3.50	210.0	17200	13.33	3.56	0.03
3.63	217.5	17100	26.67	1.78	0.007
3.75	225.0	16900	13.33	-1.78	0.01
3.88	232.5	16800	53.33	+5.33	0.03
4.00	240.0	16400	46.67	-0.89	0.02
4.13	247.5	16050	20.00	-3.56	0.02
4.25	255.0	15900	13.33	-0.89	0.03
4.38	262.5	15800	53.33	5.33	0.04
4.50	270.0	15600	20.00	-4.44	0.02
4.63	277.5	15250	20.00	0	0.01
4.75	285.6	15100	40.00	2.67	0.02
4.88	292.5	14800	20.00	-2.67	0.03
5.00	300.0	14650	46.67	3.56	0.02
5.13	307.5	14300	40.00	-0.89	0.01
5.25	315.0	14000	53.33	1.78	0.01
5.38	322.5	13600	40.00	-1.78	0.01
5.50	330.0	13300	46.67	0.89	0.01
5.63	337.5	12950	40.00	-0.89	0.01
5.75	345.0	12650	20.00	-2.67	0.01
5.88	352.5	12500	13.33	-0.89	0.01
6.00	360.0	12400	6.67	-0.89	0.01
6.13	365.5	12350	20.00	1.78	0.01
6.25	375.0	12200	26.67	0.89	0.02
6.38	382.5	12000	0	-3.56	0.03
6.50	390.0	12000	26.67	3.56	0.02
6.63	397.5	11800	13.33	-1.78	0.01
6.75	405.0	11700	13.33	0	0.02
6.88	412.5	11600	53.33	5.33	0.02
7.00	420.0	11200	53.33	0	0.01
7.13	427.5	10800	40.00	-1.78	0.01
7.25	435.0	10500	13.33	-3.56	0.02
7.38	442.5	10400	33.33	2.56	0.03
7.50	450.0	10150	0	-4.44	0.02
7.63	457.5	10150	13.33	1.78	0.01
7.75	465.0	10050	6.67	-0.89	0.01
7.88	472.5	10000	0	-0.89	0.01

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC	ACCELERATION FT/SEC²	ONSET RATE g/SEC
8.00	480.0	10000	0	0	0.01
8.13	487.5	10100	−13.33	1.78	0
8.25	495.0	10300	−26.67	1.78	0
8.38	502.5	10600	−40.00	1.78	0
8.50	510.0	10800	−26.67	−1.78	0.01
8.63	517.5	10800	0	−3.56	0.02
8.75	525.0	10500	40.00	5.33	0.04
8.88	532.5	10200	40.00	0	0.02
9.00	540.0	9700	66.67	3.56	0
9.13	547.5	9000	93.33	3.56	0.06
9.25	555.0	8900	13.33	−10.67	0.04
9.38	562.5	8800	13.33	0	0.01
9.50	570.0	8600	26.67	1.78	0.01
9.63	577.5	8500	13.33	−1.78	0.02
9.75	585.0	8550	−6.67	−2.67	0.07
9.88	592.5	7800	100.00	14.22	0.07
10.00	600.0	7150	86.67	−1.78	0.03
10.13	607.5	7050	13.33	−9.78	0.04
10.25	615.0	6950	13.33	0	0.01
10.38	622.5	6700	33.33	2.67	0.03
10.50	630.0	6650	6.67	3.55	0.01
10.63	637.5	6200	60.00	7.11	0.01
10.75	645.0	6050	20.00	−5.33	0.05
10.88	652.5	5900	20.00	0	0.02
11.00	660.0	5700	26.67	0.89	0.01
11.13	667.5	5450	33.33	0.89	0
11.25	675.0	5450	0	−4.44	0.02
11.38	682.5	5500	−6.67	0.89	0.02
11.50	690.0	5400	13.33	2.67	0.01
11.63	697.5	5400	0	−1.78	0.01
11.75	705.0	5300	13.33	1.78	0.04
11.88	712.5	5000	40.00	3.56	0.01
12.00	720.0	4650	46.67	0.89	0.01
12.13	727.5	4400	33.33	−1.78	0.01
12.25	735.0	4350	6.67	−3.55	0.02
12.38	742.5	4350	0	−0.89	0.01
12.50	750.0	4300	6.67	0.89	0.01

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC	ACCELERATION FT/SEC^2	ONSET RATE g/SEC
12.63	757.5	4200	13.33	0.89	0.01
12.75	765.0	4050	20.00	0.89	0.01
12.88	772.5	3700	46.67	3.56	0.02
13.00	780.0	3650	6.67	-5.33	0.04
13.13	787.5	3650	0	-0.89	0.02
13.25	795.0	3650	0	0	0.01
13.38	802.5	3650	0	0	0
13.50	810.0	3550	13.33	1.78	0.01
13.63	817.5	3550	0	-1.78	0.01
13.75	825.0	3450	13.33	1.78	0.01
13.88	832.5	3450	0	-1.78	0.01
14.00	840.0	3400	6.67	0.89	0.01
14.13	847.5	3550	-20.00	3.56	0.01
14.25	855.0	3600	-6.67	-1.78	0.02
14.38	862.5	3550	6.67	1.73	0.01
14.50	870.0	3300	33.33	3.55	0.02
14.63	877.5	2850	60.00	3.56	0
14.75	885.0	2450	53.33	-0.89	0.02
14.88	892.5	2400	6.67	-6.22	0.02
15.00	900.0	2350	6.67	0	0.03
15.13	907.5	1550	106.67	13.33	0.10
15.25	915.0	1350	26.67	-10.67	0.07
15.38	922.5	800	73.33	6.22	0.01
15.50	930.0	550	33.33	5.33	—

NUMERICAL VALUES OF ALTITUDE AND O_2 PARTIAL PRESSURE

BASED ON ALTITUDE TIME HISTORY --- SHP 68-2.8

GRAPH OF J W EDWARDS

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	TOTAL PRESSURE mm Hg.	PARTIAL PRESSURE O_2 mm Hg.
0	0	23300	303.59	63.60
.13	7.5	23350	302.94	63.47
.25	15.0	23400	302.26	63.32
.38	22.5	23300	303.59	63.60
.50	30.0	23000	307.53	64.43
.63	37.5	22800	310.18	64.98
.75	45.0	22500	314.19	65.82
.88	52.5	22300	316.88	66.39
1.00	60.0	21900	322.33	67.53
1.13	67.5	21800	323.70	67.82
1.25	75.0	21600	326.47	68.39
1.38	82.5	21400	329.24	68.98
1.50	90.0	21000	334.86	70.15
1.63	97.5	20800	337.70	70.75
1.75	105.0	20300	334.88	72.25
1.88	112.5	19600	355.15	74.40
2.00	120.0	19100	362.62	75.97
2.13	127.5	18550	370.84	77.69
2.25	135.0	18100	377.97	79.18
2.38	142.5	17400	389.01	81.50
2.50	150.0	17150	393.45	82.43
2.63	157.5	16800	398.69	83.53
2.75	165.0	16250	407.92	85.46
2.88	172.5	16150	409.70	85.83
3.00	180.0	16150	409.70	85.83
3.13	187.5	16300	406.91	85.25
3.25	195.0	16800	398.64	83.53
3.38	202.5	17100	393.83	82.51
3.50	210.0	17200	392.37	82.17
3.63	217.5	17100	393.83	82.51

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	TOTAL PRESSURE mm Hg	PARTIAL PRESSURE O_2 mm Hg.
3.75	225.0	16900	397.07	83.19
3.88	232.5	16800	398.69	83.53
4.0	240.0	16400	405.25	84.91
4.13	247.5	16050	410.97	86.10
4.25	255.0	15900	413.57	86.64
4.38	262.5	15800	415.25	87.00
4.50	270.0	15400	422.03	88.42
4.63	277.5	15250	424.43	88.92
4.75	285.5	15100	427.17	89.49
4.88	292.5	14800	432.37	90.58
5.00	300.0	14650	434.85	91.10
5.13	307.5	14300	441.13	92.42
5.25	315.0	14000	446.46	93.53
5.38	322.5	13600	453.65	95.04
5.50	330.0	13300	459.10	96.18
5.63	337.5	12950	465.58	97.54
5.75	345.0	12650	47066	98.60
5.88	352.5	12500	473.90	99.28
6.00	360.0	12400	475.78	99.68
6.13	365.5	12350	476.76	99.98
6.25	375.0	12200	479.55	100.47
6.38	382.5	12000	483.34	101.24
6.50	390.0	12000	483.34	101.24
6.63	397.5	11800	487.16	102.06
6.75	405.0	11700	489.08	102.46
6.88	412.5	11600	491.01	102.87
7.00	420.0	11700	491.91	103.06
7.13	427.5	10800	506.63	106.14
7.25	435.0	10500	512.59	107.39
7.38	442.5	10400	514.59	107.81
7.50	450.0	10150	519.68	108.87
7.63	457.5	10150	519.68	108.87
7.75	465.0	10050	521.72	109.30
7.88	472.50	10000	522.65	109.50
8.00	480.0	10000	522.65	109.50
8.13	487.5	10100	520.63	109.07

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	TOTAL PRESSURE mm Hg	PARTIAL PRESSUR O_2 mm Hg
8.25	495.0	10300	516.60	108.23
8 38	502.5	10600	510.60	106.97
8.50	510.0	10800	506.63	106.14
8 63	517.5	10800	506. 63	106. 14
8.75	525.0	10500	512 59	107 39
8.88	532.5	10200	518.61	108 65
9.00	540.0	9700	528.77	110.78
9.13	547.5	9000	543.26	113.81
9.25	555.0	8900	545.35	114.25
9.38	562.5	8800	547.46	114.69
9.50	570.0	8600	551.68	115. 58
9.63	577.5	8500	553.80	116.02
9.75	585.0	8550	552.70	115.79
9.88	592.5	7800	568.64	119.17
10.00	600.0	7150	583. 18	112. 18
10.13	607.5	7050	585 47	112. 66
10.25	615.0	6950	587.76	123. 13
10.38	622.5	6700	593.15	124. 26
10.50	630.0	6650	594.36	124.52
10.63	637.5	6200	604.47	126.64
10.75	645.0	6050	607. 82	127.34
10.88	652.5	5900	611.34	128.08
11.00	660.0	5700	615.97	129.04
11.13	667.5	5450	621.54	130 24
11.25	675.0	5450	621 54	130. 24
11.38	682.5	5500	620.61	130.02
11.50	690.0	5400	622.95	130.51
11.63	697.5	5400	622.95	130 51
11.75	705.0	5300	625.29	131.00
11.88	712.5	5000	632.36	132.48
12.00	720.0	4650	640.59	134.20
12.13	727.5	4400	646 68	135.48
12.25	735.0	4350	647.95	135.75
12.38	742.5	4350	647 95	135.75
12.50	750.0	4300	649.10	135 99
12.63	757.5	4200	651.52	136. 49
12.75	765.0	4050	655.07	137 24

TIME MINUTES	TIME SECONDS	ALTITUDE ~~FEET~~	TOTAL PRESSURE mm Hg	PARTIAL PRES O_2 mm Hg
12.88	772.50	3700	663.73	139.05
13.00	780.0	3650	664.97	139.31
13.13	787.5	3650	664.97	139.31
13.25	795.0	3650	664 97	139.31
13 38	802.50	3650	664 97	139.31
13.50	810.0	3550	667.21	139.79
13.63	817.5	3550	669.21	139.79
13.75	825.0	3450	669.80	140.32
13.88	832.5	3450	669.80	140.32
14.00	840.0	3400	671.15	140.6
14.13	847.5	3550	669.26	139.79
14.25	855.0	3600	666.20	139.57
14.38	862.5	3550	669.26	139.79
14 50	870.0	3300	673.64	141.13
14.63	877.5	2850	684.78	143.46
14.75	885.0	2450	694.94	145.59
14.88	892.5	2400	696.36	145.89
15.00	900.0	2350	697.48	146.12
15.13	907.5	1550	718.31	150.49
15.25	915.0	1350	723 90	151.06
15.38	922.5	800	738.28	154.67
15.50	930.0	550	745.02	156.08

ATTACHMENT 2

DECELERATION ANALYSIS

FROM 2ND GROUND CONTACT TO POINT OF REST

PLANE BREAKUP OCCURRED 1200' FROM 2ND GROUND CONTACT.

CARGO COMPARTMENT FLOOR MOVED 1400' FROM 2ND GROUND CONTACT TO ITS POINT OF REST.

PLANE VELOCITY AT 2ND GROUND CONTACT LOCATION 455 FT/SEC. (270 KNOTS – 310 MPH)

BASED UPON CONSTANT DECELERATION RATE

$V^2 = 2as$

V = VELOCITY FT/SEC

a = DECELERATION FT/SEC^2

S = DISTANCE FT

$$a = \frac{V^2}{2s}$$

$$a = \frac{455^2}{2 \times 1400} = 74 \ FT/SEC^2 = 2.30 \ g$$

THE TROOP COMPARTMENT SEPARATED FROM THE CARGO FLOOR AFTER TRAVELING 1200' FROM 2ND GROUND CONTACT LOCATION

THE TOTAL DISTANCE TRAVELED BY THE TROOP COMPARTMENT FROM 2ND GROUND CONTACT LOCATION WAS 2012'.

THUS THE TROOP COMPARTMENT TRAVELED 812' AFTER BREAKUP (2012 – 1200)

THE VELOCITY OF THE TROOP COMPARTMENT AT POINT OF BREAK-UP:

$V_I^2 - V_B^2 = 2as$

V_I = IMPACT VELOCITY

V_B = BREAKUP VELOCITY

$$455^2 - V_b^2 = 2 \times 74 \times 1200$$

$$V_b = \sqrt{455^2 - 2 \times 74 \times 1200}$$

$$V_b = 172 \text{ FT/SEC} \quad (117 \text{ mph})$$

FROM THE VELOCITY OF 172 FT/SEC THE TROOP COMPARTMENT TRAVELED 812' TO ITS POINT OF REST

$$a = \frac{V^2}{2 \times S}$$

$$a = \frac{172^2}{2 \times 812} = 18.22 \text{ FT/SEC} \quad (0.57\,g)$$

TIME FOR TROOP COMPARTMENT TO MOVE FROM POINT OF BREAK UP TO POINT OF REST

$$S = \frac{1}{2} a t^2$$

S = DISTANCE TRAVELED

a = DECELERATION RATE

t = TIME

$$t = \sqrt{\frac{812 \times 2}{18.22}}$$

$$t = 9.44 \text{ SECONDS}$$

TIME FOR PLANE TO MOVE FROM 2ND GROUND CONTACT LOCATION TO POINT OF BREAKUP

$$t = \sqrt{\frac{1200 \times 2}{74.}} = 5.69 \text{ SECONDS}$$

TOTAL ELAPSED TIME FROM 2ND GROUND CONTACT LOCATION TO POINT OF REST OF TROOP COMPARTMENT

$$T = t_B + t_A$$

$$T = 5.69 + 9.44 = 15.13 \text{ SECONDS}$$

t_B = TIME BEFORE BREAKUP

t_A = TIME AFTER BREAKUP

NOTE:

THE CARGO FLOOR AND TROOP COMPARTMENT WERE AT SAME FORWARD VELOCITY UP TO INSTANT OF BREAK UP.

THE MAJOR COMPONENT TRAVELING THE SHORTER DISTANCE WILL REPRESENT THE DECELERATION RATE OF THE AIRCRAFT PRIOR TO BREAK UP.

DISTANCE BETWEEN 1ST AND 2ND GROUND CONTACT LOCATION = 2700'

BEFORE BECOMING AIR BORNE TO 2ND GROUND CONTACT LOCATION PLANE SKIDDED 1000'

THUS PLANE WAS AIR BORNE FOR 1700'

BASED UPON CONSTANT SPEED OF 455'/SEC THE TIME ELAPSED BETWEEN 1ST AND 2ND POINTS OF GROUND CONTACT WAS:

$$\frac{2700}{455} = 5.93 \text{ SECOND}$$

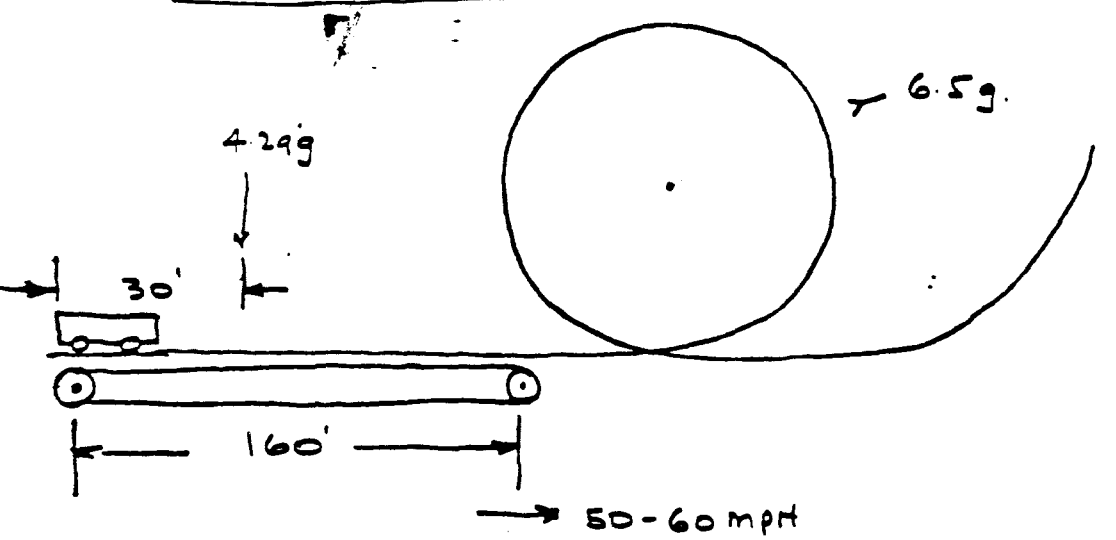
THE PLANE WAS AIRBORE

$$\frac{1700}{455} = 3.74 \text{ SECONDS}$$

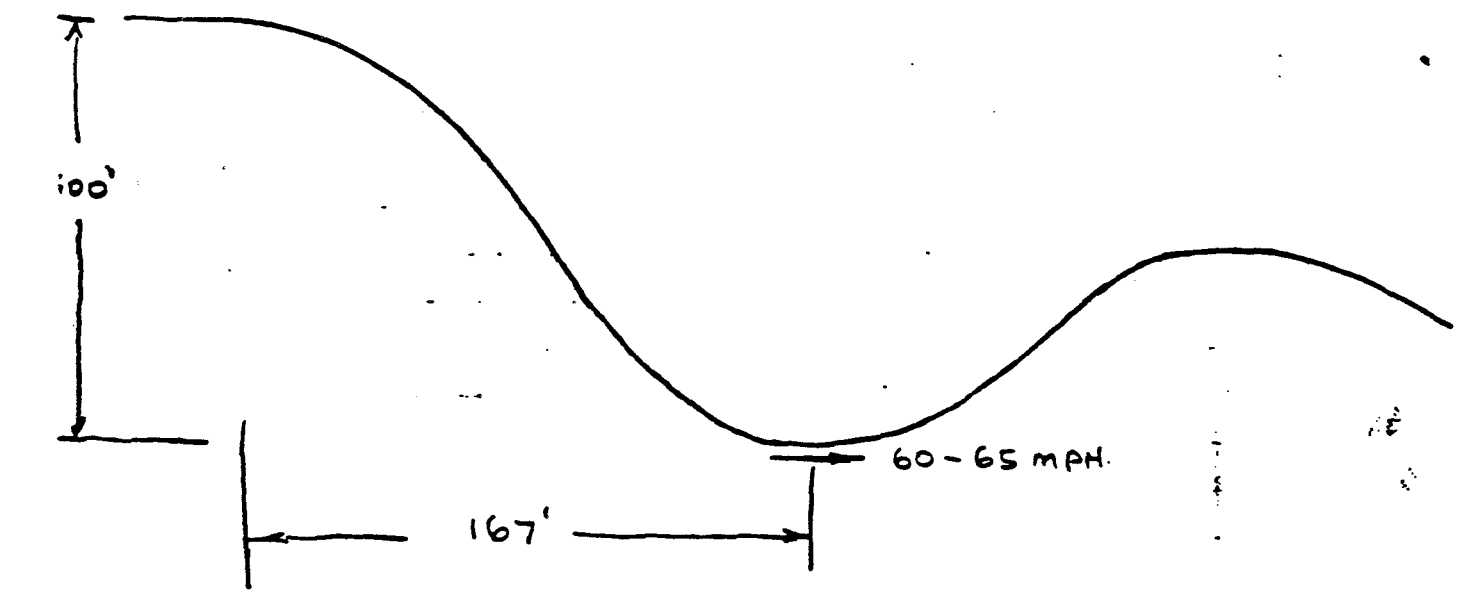
ATTACHMENT 3

AMUSEMENT RIDE ANALYSIS

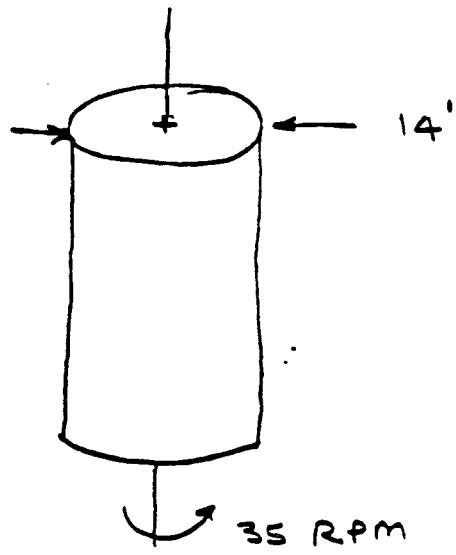
MONTEZUMA'S REVENGE



ROLLER COASTER



ROTOR



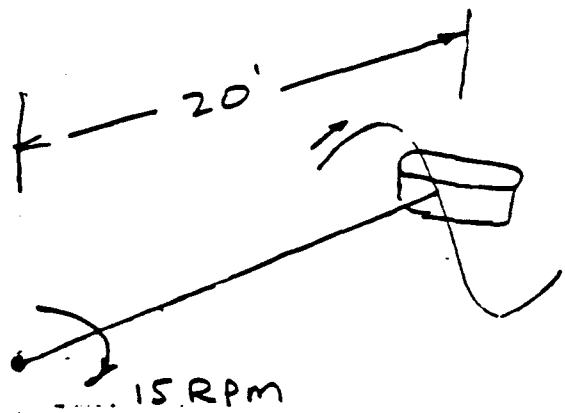
$14\pi = 43.98'$ CIRCUM

$\frac{43.98}{1.72} = 25.51$ FT/SEC

CENTRIFUGAL ACCELERATION $= \frac{V^2}{R}$

$\frac{25.51^2}{7} = 92.96$ FT/SEC^2 $= 2.89$ g

ROUND-UP



$40\pi = 125.66'$ CIRCUMFERENCE

15 RPM = 0.25 RPS = 4 SECONDS/REVOLUTION

$\frac{125.66}{4} = 31.42$ FT/SEC

$\frac{31.42^2}{20} = 49.35$ FT/SEC/SEC $= 1.53$ g

JAMES G. GAUME, M.D.
CONSULTANT IN HUMAN FACTORS
1517 ESPINOSA CIRCLE
PALOS VERDES ESTATES, CALIFORNIA 90274
(213) 375-6607

REPORT B

ANALYSIS OF THE PHYSIOLOGICAL EFFECT OF THE CHANGE OF PRESSURE DURING THE DECOMPRESSION EVENT OF C5A SN68-218 4 APRIL 1975

The difference in the time of decompression between the cargo and the troop compartments was minimal -- a matter of milliseconds -- because of the size of the openings, made up by the ladder well and the grille, total approximately 18 sq. ft. in area. When the pressure of 302.8 mm Hg (5.85 psia) in less than one second (approximately 0.6 second or 600 milliseconds). The total pressure change was 329.8 mm Hg (6.39 psia). In the 0.6 second, the cargo compartment was at the ambient pressure. However, as soon as the pressure began reducing in the cargo compartment, the pressure in the troop compartment began to reduce also. As stated above, the troop compartment lagged behind the cargo compartment approximately 0.03 second (30 milliseconds). The total pressure reduction during this 30 milliseconds was approximately 25 mm Hg. Both the time difference and the pressure difference in this period are insignificant with regard to the physiological effects, because the response time of the body to the pressure change is much slower than the pressure difference in that period of time. Attachement B_1, page 147, from Armstrong's book, Aerospace Medicine, states that "the physiological effects of loss of pressurization of jet transports

will not be caused by explosive decompression, but to the effect of acute oxygen deprivation." In this case, however, the period of oxygen deprivation was too brief and too mild to have any lasting, serious consequence.

The "bends" would have been the earliest symptom to develop on decompression, but these do not usually appear until 10-15 minutes after the decompression and therefore did not have time to develop. Bends would be unlikely at 23,400 ft., however.

James G. Gaume, M.D.

JAMES G. GAUME, M.D.
Human Factors Consultant
30 August 1981

aero-space medicine

EDITED BY

Maj. Gen. Harry G. Armstrong, USAF (Ret.)

Formerly, Surgeon General, United States Air Force

Baltimore 1961

THE WILLIAMS & WILKINS COMPANY

Library of Congress Catalog Card Number 60-10173

Composed and printed at the

Waverly Press, Inc.

Baltimore 2, Maryland, U.S.A.

required at rest. As a consequence the amount of oxygen which must be added to the inspired air in flight must be varied according to the ventilation rate in order to keep the oxygen percentage constant.

A further and very important consideration is the fact that each method of administration varies tremendously in its efficiency and, in most cases, the amount of oxygen supplied to the individual is no criterion of the amount available to him for respiration. In practice the only satisfactory means of determining the amount of oxygen required for any particular piece of equipment is to determine experimentally the flow necessary to give a sea level value to the partial pressure of the oxygen in the lungs or in the blood.

Oxygen Utilization in Flight

All high altitude military airplanes are provided with oxygen equipment and military personnel are required to utilize oxygen at all times while participating in flight above 10,000 feet. One of the first indications of incipient oxygen lack occurs at night where a measurable reduction in night vision usually occurs at altitudes as low as 5000 feet. The decision establishing the mandatory altitude at which military personnel must use oxygen equipment is based on the factor of dispensing with the annoyance of the use of oxygen equipment until an altitude is reached where hypoxia may create an equal or greater handicap. The physiologic changes caused by the development of minor hypoxia from sea level to 10,000 feet are of a moderate nature. In most cases the airmen are unaware of them. They consist of a slight increase of pulmonary ventilation resulting from an increase in the rate and depth of breathing. There is a slight to moderate increase of blood pressure and pulse rate. In military aircraft capable of flight above 35,000 feet, the cockpits are usually pressurized. Pressurization varies from 12,000 to 18,000 feet. In aircraft capable of flight above 35,000 feet, positive pressure breathing equipment is used. In military aircraft capable of flight above 55,000 feet, full or partial pressure suits with their ancillary oxygen equipment are required. Individuals are encouraged to use oxygen at lower altitudes than those prescribed whenever it is deemed necessary by reason of low altitude tolerance, undue physical activity in flight or other circumstance which cannot be covered by general regulation.

In commercial aviation, oxygen equipment is installed in accordance with civil aeronautics regulation. In commercial carriers with unpressurized cabins, a separate oxygen system is maintained for the crew and passengers, respectively. The passenger oxygen equipment requirement consists of a 10 per cent passenger availability at 8000 to 14,000 feet to 100 per cent equipment availability for passengers above 15,000 feet for the duration of the flight. Pressurized cabin commercial carriers are covered by additional civil air regulations. At present, the average commercial carrier flies at a maximum altitude of 20,000 feet with an internal pressurization of 8000 feet. Under these circumstances civil aeronautics regulation requires that crew members be provided with oxygen equipment for the duration of the flight above 10,000 feet. Ten per cent of the passengers will be provided with oxygen equipment with 30 minutes capacity if the altitude does not exceed 25,000 feet.

Jet transports flying at altitudes of 40,000 feet will have an internal pressurization of 8500 feet. In view of the possibility of failure of plane pressurization of jet aircraft which for economical operation must invariably fly above 25,000 feet altitude, the existing civil aeronautics regulation stipulates oxygen equipment for all passengers. In addition, the pilot will wear an oxygen mask at all times above 25,000 feet. Automatic presentation systems are installed in this type of commercial carrier. With this system the pilot can make the masks available to passengers in case of emergency by simply pressing a button. The passenger then holds a rubber cup over his nose and mouth until subsequent descent to safe levels has been accomplished.

The physiologic effects of loss of pressurization of jet transports will not be caused by explosive decompression, but to the effect of acute oxygen deprivation. The onset of hypoxia will depend upon the type of equipment failure and the altitude of the plane. In the case of compressor malfunction the internal pressurization will drop slowly and corrective measures will be less urgent. In

JAMES G. GAUME, M.D.
CONSULTANT IN HUMAN FACTORS
1517 ESPINOSA CIRCLE
PALOS VERDES ESTATES, CALIFORNIA 90274
(213) 375-6607

REPORT C

ANALYSIS OF THE HYPOXIA CAUSED BY THE DECOMPRESSION EVENT OF C5A SN68-218 4 APRIL 1975

At 23,400 ft. the alveolar pO_2 (oxygen pressure) is approximately 28 mm Hg. On a sudden decompression from 5,000 ft. to 23,400 ft. in less than 0.6 second, hypoxia could be evident to the observer in a few (2-3) minutes. The subject would feel hypoxic in 1.5-2.0 minutes, but the feeling (of air hunger) passes within one minute after onset, and breathing is relatively easy again until 5-6 total minutes have passed. The reasons for this effect are: (1) An increase in pulmonary ventilation takes place automatically and the subject takes in a greater volume of air per breath and per minute. The blood pO_2 has already been reduced, so that as the greater volume of air is breathed, more oxygen (O_2) is extracted from the inspired air, raising the arterial pO_2 by perhaps 20%; and (2) Hypoxia is a potent cerebral vasodilator which increases the volume of blood flowing through the brain, thereby again increasing the O_2 available to the brain by as much as 35% at 23,400 ft., which would raise the pO_2 of the cerebral blood to more than 40 mm Hg. This would be equivalent to the arterial pO_2 expected at less than 18,000 ft. At 18,000 ft., it is expected that the average person would have a TUC of 30 minutes. However, by the time that this 18,000 ft.

pO_2 equivalent was attained (due to the combined spontaneous increase in pulmonary ventilation and the hypoxia effect), the aircraft had already descended to an altitude of approximately 16,000 ft., according to the descent profile indicated by the MADAR data. This is an easily survivable altitude without any physiological damage. (See Attachments C1,2,3)

These normal, physiological, compensatory, protective mechanisms which came into play, activated by the extremely rapid decompression, constituted the factors which prevented the occupants of the troop compartment of the C5A from becoming unconscious, and therefore, from sustaining any brain damage as a result of hypoxia. The hypoxia was too mild and too transient to be of any serious import. An example of this is illustrated by the incident described by Charles A. Lindbergh, involving himself, as detailed in the Foreword of the Handbook of Respiratory Physiology (Attachments C4, p. vi). As indicated by this example, had anyone in the C5A become unconscious from lack of O_2, they would have recovered consciousness within 2-3 minutes more, because again, according to the MADAR data, they were down to 16,000 ft. in 3.0 minutes from the moment of decompression. According to the various testimonies perviously given, no one became unconscious.

Another decompression event, involving a National Air Lines DC 10 over Albuquerque, NM, took place at 39,000 ft. The

cabin altitude reached 31,600 ft. altitude and was above 16,000 ft. for 5.5 to 6.0 minutes. Three or four passengers and flight attendants became unconscious from lack of oxygen, as a matter of record, but all regained consciousness at approximately 15,000 ft. without any harm.

Totally unacclimatized people are transported to the tops of Mt. Evans and Pike's Peak in Colorado, both of which have altitudes of more than 14,000 ft., and stay there for hours, walking around, climbing small elevations, without harm. Others have flown over the "Hump" in Asia, and have been without O_2 for as much as thirty minutes, without ill effect except for headache.

The Cuban Stowaway, who stowed away in the wheel well of a DC 8 as it took off from Havana to Spain, was without O_2 at 29,000 ft. for more than 7 hours, and survived with no apparent harm. His case was thoroughly documented by Spanish physicians when he reached Spain. Houdini could stay under water for 4 minutes without breathing either air or oxygen. There is a case on record wherein a man diving has remained under water without breathing apparatus, merely by holding his breath, for 13 minutes without any air except that which he had in his lungs when he submerged, and he had repeated this feat a number of times.

Therefore, because the human body has a number of protective mechanisms, all of which were activated and came into

play at the time of the decompression, the people in the troop compartment of the C5A in question survived without harm, to a reasonable medical certainty. The calculations normally used which consider only the reduction of pO_2 available, by virtue of subtracting the partial pressure values for carbon dioxide and water vapor in the lungs, do not tell the whole story. They do not consider the dilatation of the cerebral arteries and the resultant increase in blood flow and O_2 to the brain, and the consequent reduction in altitude equivalent caused by this normal compensatory mechanism. Increased pulmonary ventilation has been considered, but little or nothing has been said about the increase in heart rate which also accompanies hypoxia, and helps to provide an increase in blood flow and O_2 to the brain.

Attachment C5 from the book, Hypoxia, by Van Liere and Stickney, pp 284,285, "Ability of Young Animals to Withstand Asphyxia and Hypoxia," quotes the work of several investigators who all say that newborn human infants are able to withstand considerable periods of hypoxia (24, 71, 104). This appears to be true of the infants of most mammals, most likely another compensatory, protective mechanism to assure survival of the species.

James G. Gaume, M.D.

James G. Gaume, M.D.
Human Factors Consultant
30 August 1981

ATTACHMENT C-1, J.G.G.

LAB DATA SHEET

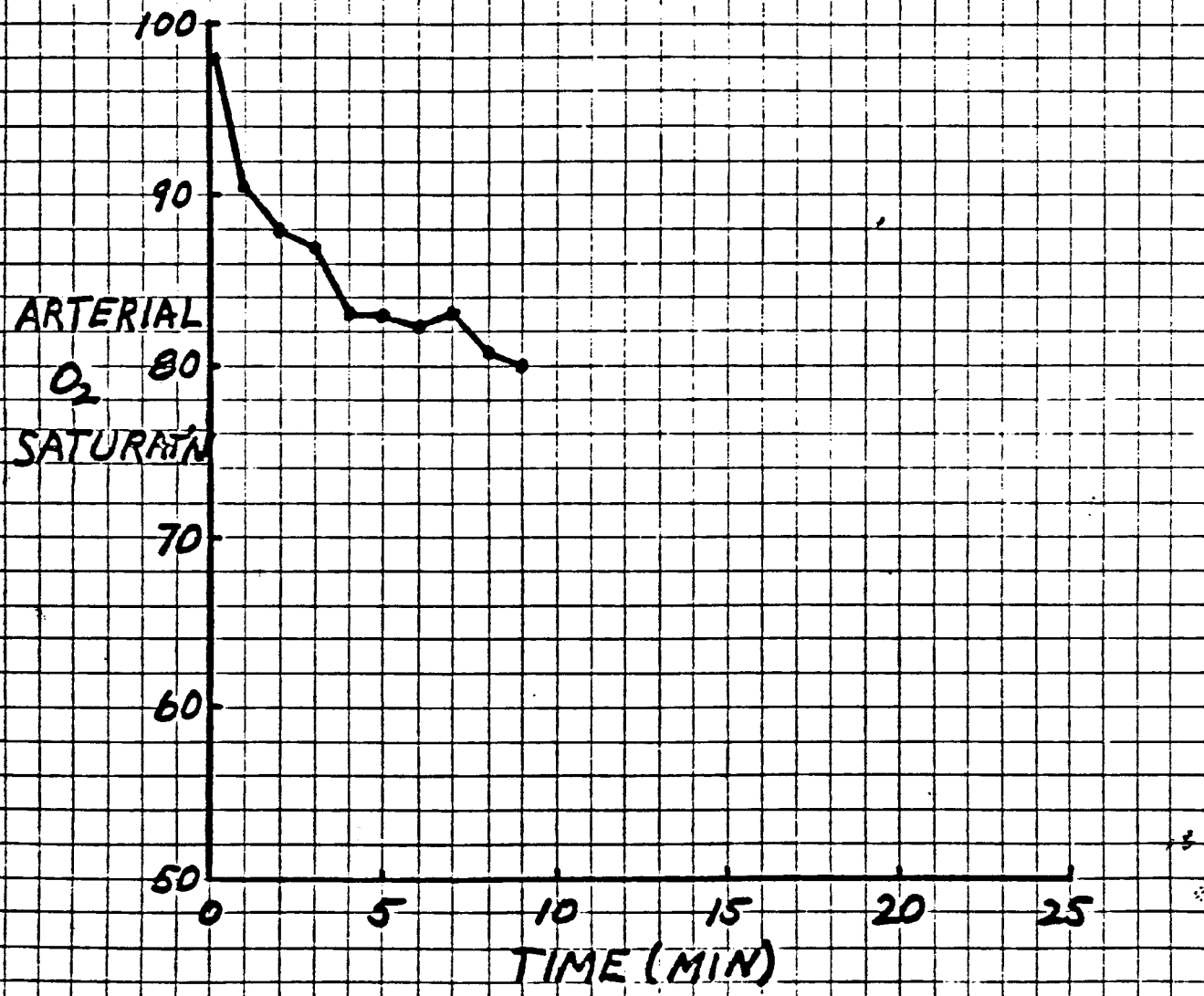
NAME: JGG. AGE: 65

DATE: 4-6-81

HOUR: 1200

GAS MIXTURE: 50% ROOM AIR (10.3% O_2 EQUIVALENT)
50% N_2 (89.1% N_2)
18,000 FT. EQUIVALENT ALTITUDE

REMARKS: AIR HUNGER @ 2 MIN. - PASSED QUICKLY. AIR HUNGER AGAIN @ 8-9 MIN.



ATTACHMENT C-2, JGG

LAB DATA SHEET

NAME: J.G.G. AGE: 65

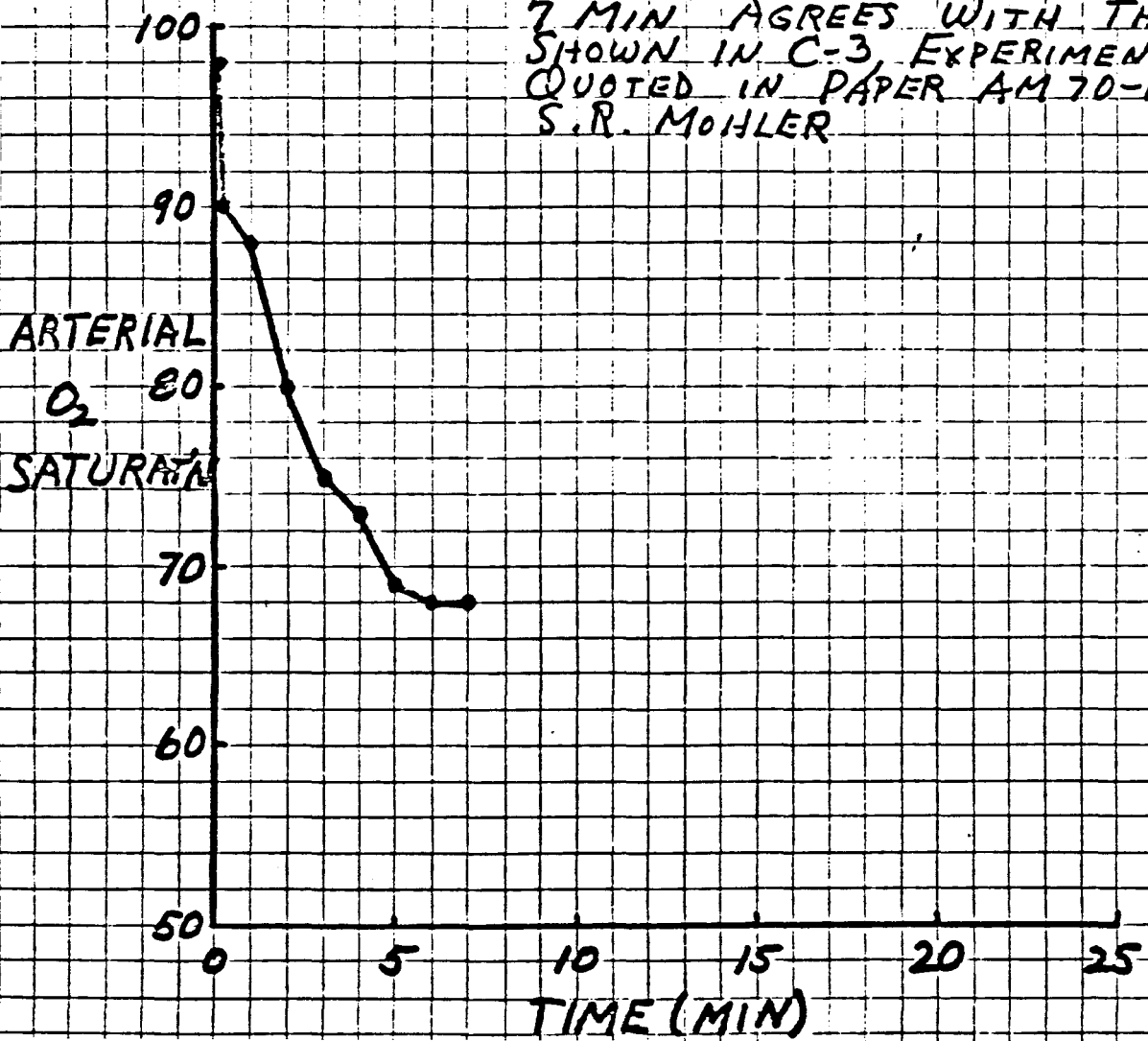
DATE: 4-6-81

HOUR: 1230

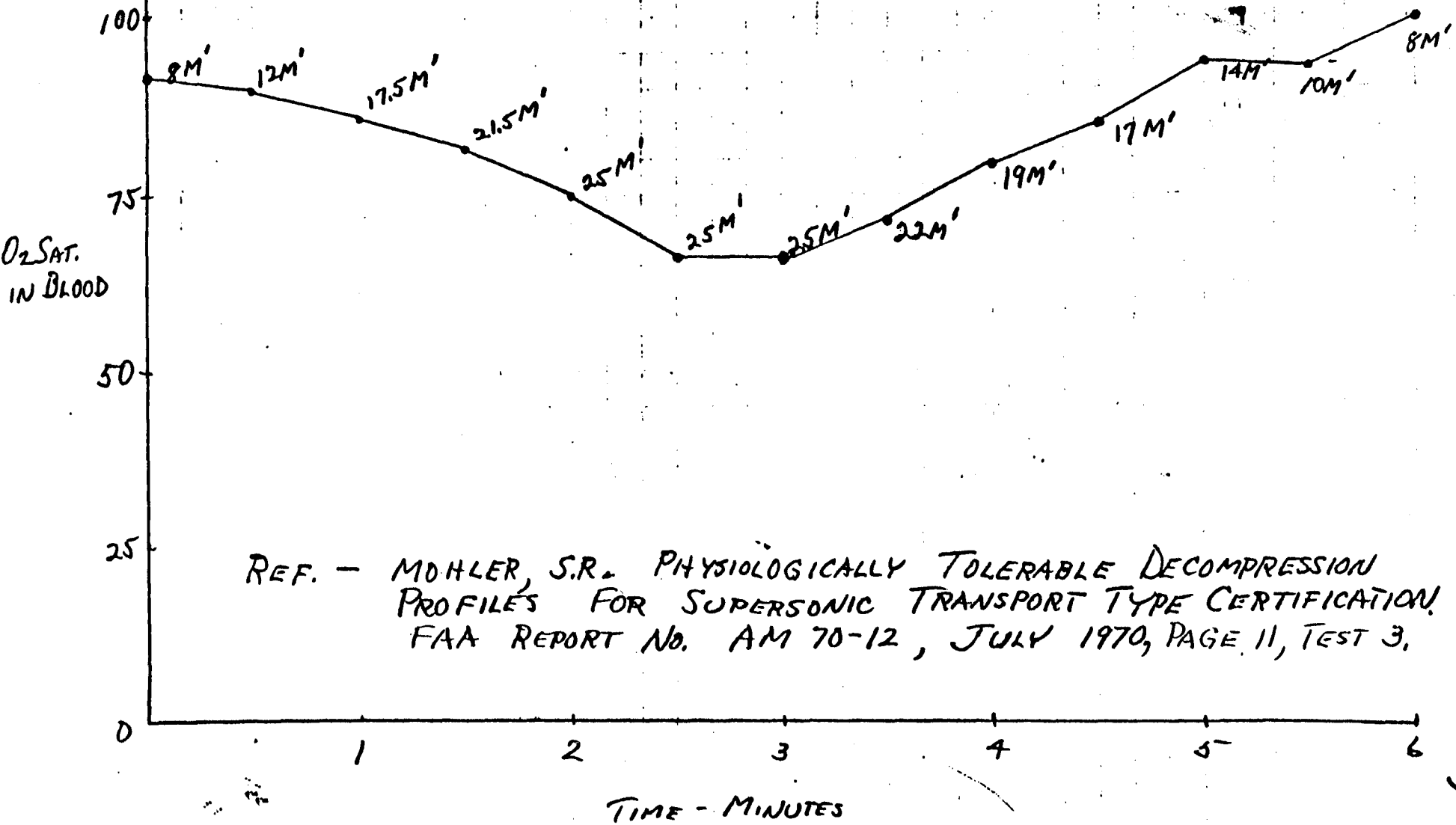
GAS MIXTURE: 33% Room Air (7.5% O_2 Equivalent)
67% N_2 (92.5% N_2)
24,000 Ft. Equivalent Altitude

REMARKS: Air Hunger @ 1.5 Min - Passed Quickly
Air Hunger Again at 6-7 Min.

This O_2 Saturation Level @ 7 Min Agrees With The Level Shown In C-3, Experiments Quoted In Paper AM 70-12 By S.R. Mohler



EXPERIMENTS AT 25,000 FT. ALTITUDE, BREATHING AIR, AND DESCENT RATE OF APPROXIMATELY 3000 FT/MIN AFTER A 3-MINUTE STAY AT 25,000 FT. SHOWING THE RAPIDITY WITH WHICH THE O_2 SATURATION OF THE BLOOD BUILDS UP TO ACCEPTABLE LEVELS UPON DESCENT



REF. — MOHLER, S.R. PHYSIOLOGICALLY TOLERABLE DECOMPRESSION PROFILES FOR SUPERSONIC TRANSPORT TYPE CERTIFICATION. FAA REPORT NO. AM 70-12, JULY 1970, PAGE 11, TEST 3.

James G. Laume, M.D.

ATTACHMENT C-4

HANDBOOK OF RESPIRATORY PHYSIOLOGY

AIR UNIVERSITY
USAF SCHOOL OF AVIATION MEDICINE
Randolph Air Force Base, Texas

RESPIRATORY PHYSIOLOGY IN AVIATION

Edited by

WALTER M. BOOTHBY, M. D.

The Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico

Air University
USAF School of Aviation Medicine
Randolph Field, Texas
September 1954

TRAINING FOR THE RECOGNITION OF OXYGEN EMERGENCIES IN HIGH-ALTITUDE FLYING

Charles A. Lindbergh

Just as primary training in stick-rudder-throttle technique is essential to a pilot of the most advanced types of airplanes, primary training in oxygen technique is essential to the high-altitude crew. Modern, scientific safeguards do not remove the importance of a thorough understanding of the ABC's in each field.

Emergencies can result as fatally today, but the value of training in oxygen technique was probably more apparent during the years before pressure masks and pressurized fuselages came into service use. Troubles were then encountered more frequently, and methods of combating them were less advanced. In the early days of altitude flying, a pilot operating much above 30,000 feet was always in a more or less hypoxic condition.

The lessons I learned from high-altitude test flying during World War II all point to this primary requirement: *Learn to recognize hypoxia quickly.* Then, you have time to do something about it before you lose consciousness.

This might be called the *B* in the *ABC's* of oxygen technique. The *A* relates to having emergency equipment available and in condition for use. The *C* demands a considered plan for using it. You can spend plenty of time arranging A and practicing C; your error is likely to come in the B of recognition. I shall try to emphasize and clarify the problem by examples from my own experience.

My first obvious contact with hypoxia came in 1927, while I was flying the *Spirit of St. Louis* at an altitude of about 20,000 feet over the mountains of Colorado. The plane carried no oxygen, and during the latter part of the slow climb I grew aware of an increasing vagueness of perception. The simplest problems of addition and subtraction, in connection with my navigation, became difficult.

My first rough experiments in oxygen technique were carried out in a P-36, in 1939, at altitudes of slightly over 30,000 feet. In this plane, an oxygen supply was available through a wooden tube at the end of a rubber hose. I studied the dulling and sharpening effects on my senses when the tube was removed from its normal position between my teeth, and when it was replaced. Pilots' tales of mysterious high-altitude effects on mind and body cautioned me in these experiments.

In 1942, at Willow Run, I undertook a project in which high-altitude breakdown tests were to be run on the ignition system of an R-2800 engine

in a P-47 fighter. The cockpit was unpressurized, and a pressure mask was not available at the time. Flights were to be made as far above 40,000 feet as possible. (By stripping the plane of all removable military equipment, I finally attained a maximum indicated altitude of 43,000 feet.)

Before starting this project, I flew to Rochester, Minnesota, for two weeks of simulated high-altitude operation in the altitude chamber at Dr. Boothby's Aero-Medical Unit of the Mayo Clinic. Chamber tests soon showed that at 40,000 feet I could expect approximately 15 seconds of reasonably clear consciousness following a complete oxygen failure — slightly more or slightly less, depending on the abruptness of the failure and my physical condition at the moment. Fifteen seconds gave little more than enough time to transfer from the plane's oxygen system to a jump-bottle oxygen system. And 15 seconds would be available *only if I discovered an oxygen failure immediately upon its occurrence.*

The general opinion prevailing among flying personnel, in 1942, was to the effect that you could not train your senses to become aware of a hypoxic condition in time to take conscious action to overcome it. My own experience led me to doubt the validity of this opinion. Working with Dr. Boothby and his staff, I arranged a system whereby the oxygen supply to my mask, in the altitude chamber, could be cut off without my knowledge. Another mask, with a full supply of oxygen, was laid at my side. It was my job to learn to detect hypoxia quickly enough to change the masks without assistance. Several trials taught me to make the change with a number of the originally available seconds of consciousness still in reserve.

This training may well have saved my life in the test flights with the P-47 which followed. On one of these flights, my oxygen gage read 50 pounds high and I ran short of oxygen without warning, at 36,000 feet during a descent from higher altitude. I noticed the effects of hypoxia in plenty of time, but I made an error in what I call here the C of oxygen technique. Instead of changing immediately to the jump-bottle system, I nosed my fighter down into a dive toward denser air. Of course, in a dive from 36,000 feet, I had more than 15 seconds of consciousness available; but it was not enough. The dials in front of me faded. My mind became too dull to think of the jump-bottle system. From somewhere above 30,000 feet to somewhere below 20,000 feet, I remember only a great shriek outside my cockpit and my determination to increase the angle of dive regardless of consequences. The P-47 almost certainly went through a compressibility condition, but it was fully controllable again when the instrument-board dials began to clarify, at about 17,000 feet, and as my senses regained their normalcy with the increasing density of air.

That P-47 flight produced excellent examples of proper recognition of an oxygen emergency and improper action following the recognition. It pointed up the value of adequate altitude-chamber training. Good B technique compensated for bad C technique. The flight took place ten years prior to the writing of this chapter; but regardless of the improvement in emergency equipment and procedure, the ability to recognize hypoxia quickly still remains essential to the safety of the high-altitude crew. You should be able to recognize the symptoms of anoxia even when your mind is concentrating on the duties of your mission. The procedure

to be followed thereafter depends on such variable factors as the cause of oxygen failure, the type of your aircraft, and the mission you have been assigned to.

Altitude-chamber training for the recognition of hypoxia is simple. It is applicable to group instruction. It saves lives.

ATTACHMENT C-5

EDWARD J. VAN LIERE
J. CLIFFORD STICKNEY

HYPOXIA

C-39332

THE UNIVERSITY OF CHICAGO PRESS
CHICAGO AND LONDON

ATTACHMENT C-5, J.G.S.

To WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE,
which provides the climate for creative work

Library of Congress Catalog Card Number: 63-16722

THE UNIVERSITY OF CHICAGO PRESS, CHICAGO & LONDON
The University of Toronto Press, Toronto 5, Canada

CHAPTER XVIII

EFFECT OF HYPOXIA ON THE NERVOUS SYSTEM

Of all the tissues in the body, nervous tissue is the least capable of withstanding oxygen want. Whereas cartilage tissue, for example, may withstand total deprivation of oxygen for several hours without suffering any apparent deleterious effects, nervous tissue can withstand deprivation of oxygen for only a few minutes. Since nervous tissue is so sensitive to oxygen want, it is obvious that the effect of hypoxia on the central nervous system of the intact organism is of paramount importance.

BLOOD SUPPLY TO THE BRAIN

The literature on cerebral circulation was reviewed by Wolff (109) in 1936. In 1943 Schmidt (86) published a monograph on cerebral circulation. The effect of hypoxia on cerebral circulation was reviewed by Opitz (76) in 1950, by Kety (62) in 1958, and by Lassen (65) in 1959. The reader is referred to these reviews for details of this important subject.

Schmidt (85) and Schmidt and Pierson (87) showed that oxygen deficiency produces vasodilatation and an increased volume of blood flow to the medulla oblongata and hypothalamus. A number of investigators in the early 1930's (20, 67, 110) also demonstrated that hypoxia produces dilatation of the pial vessels. These findings have been confirmed by later workers (65).

Wolff (109) stated that inhalation of carbon dioxide produces a more marked vasodilatation of the vessels which supply the brain than does oxygen want. If this were true, there would be a greater dilatation of the cerebral vessels during asphyxia than during anoxic hypoxia. On the other hand, Dumke and Schmidt (31) in 1943 observed that both hypoxia and hypercapnia increased cerebral blood flow but that the effect of hypoxia was more striking than that produced by carbon dioxide.

The consensus is that slight variations of oxygen tensions do not affect cerebral blood flow; however, a moderate decrease in oxygen tension may produce a significant increase. Courtice (23) in 1941, working with chloralosed cats, found that there was no increase in cerebral circulation until the inspired air contains less than 15 per cent oxygen. Kety and Schmidt (63) in 1948 reported that in subjects breathing 10–13 per cent oxygen the cerebral blood flow increased about 35 per cent. Lassen (65) reported similar findings. The latter worker has emphasized that the pronounced vasodilatory response to oxygen lack means that a greater degree of arterial oxygen unsaturation can be tolerated than would be the case if this response did not occur.

Opitz and Schneider (76) reported in 1950 that cerebral blood flow increased by anemia and that vasodilatation commences when the pO_2 of the cerebral venous blood falls to about 28 mm. Hg.

Although there is sound evidence that anoxic hypoxia and probably hemic hypoxia cause an increased blood supply to the brain, it is likely that in spite of this the diminished oxygen tension during hypoxia produces a deficient oxygen supply to the brain. It is generally conceded that during anoxic hypoxia the brain is one of the first organs to be affected.

SURVIVAL TIME OF DIFFERENT NERVE TISSUES DEPRIVED OF BLOOD

It has been known for a long time that different parts of the nervous system are more sensitive to deprivation of blood supply, that is, stagnant and hemic hypoxia, than are others. According to Heymans[1] (49), Stenon (93) in 1667 and Legallois (66) more than a century and a half later, were the first to investigate this important problem.

Many workers have experimentally produced anemia of the brain by occluding the arterial supply; among the early investigators were: Cooper (22) in 1836; Hill (51) in 1896 and in 1900 (52); Crile and Dolley (25) in 1908; and Pike, Guthrie, and Stewart (78) also in 1908. Others have reported studies on the effect of acute anemia on nervous centers (4, 16, 17, 25, 28, 39, 43, 50, 60, 61, 72, 77, 94, 95, 103).

Cannon and Burkett (19) in 1913 reviewed the literature of the

[1] C. Heymans in 1950 reviewed the literature concerning survival and revival of nervous tissues after arrest of circulation. The reader is referred to this extensive review which lists 246 references. (C. Heymans, *Physiol. Rev.*, 30 [1950], 395.)

effect of anemia on nerve cells of different classes. Table 10, which was compiled by Drinker (30) from the literature cited by Cannon and Burkett, shows the survival time of different nerve tissues when completely deprived of blood.

TABLE 10

SURVIVAL TIME OF DIFFERENT NERVE TISSUES COMPLETELY DEPRIVED OF BLOOD*

Tissue	Survival Time (Minutes)
Cerebrum, small pyramidal cells	8
Cerebellum, Purkinje's cells	13
Medullary centers	20–30
Spinal cord	45–60
Sympathetic ganglia	60
Myenteric plexus	180

* From W. P. Drinker, *Carbon Monoxide Asphyxia* (New York: Oxford University Press, 1938), p. 133.

Drinker, interestingly enough, has pointed out that Table 10 indicates that individuals who have suffered from severe hypoxia, such as may be produced by carbon monoxide poisoning, may be practically decerebrated.

Heymans *et al.* (50) in 1937 studied the effect of acute anemia on the nerve centers by perfusion of the isolated head of the dog. The circulation was interrupted for varying periods of time, and the ability of the centers to revive after the circulation had been completely interrupted was noted.

Table 11 shows that the cortical regions are the most sensitive to oxygen want. It is of interest that Davies and Bronk (26*a*), in studies

TABLE 11

ABILITY OF CENTERS AT VARIOUS LEVELS OF THE NERVOUS SYSTEM TO WITHSTAND COMPLETE INTERRUPTION OF BLOOD SUPPLY*

Interruption of Central Circulation up to	Cortical	Palpebral Pupillary	Cardio-regulatory	Vaso-motor	Respiratory
1–5 min.	+	+	+	+	+
5–10	−	+	+	+	+
10–15	−	−	+	+	+
15–30	−	−	+	+	+
30	−	−	−	−	−

* From W. P. Drinker, *Carbon Monoxide Asphyxia* (New York: Oxford University Press, 1938), p. 134.

on oxygen tension in the mammalian brain, reported that the cortex (at least locally) is on the verge of oxygen insufficiency even in its normal state. Actually the cortex has but a small reserve of dissolved oxygen should the circulation fail completely. Their experiments suggest, however, that the cortex ought to function normally as long as its oxygen tension is well above 5 mm. Hg.

It is of especial interest (Table 11) that the respiratory center, which is generally regarded as being extremely sensitive to oxygen want, may be revived after it has been deprived of its circulation for a considerable time. Heymans *et al.* (50) pointed out that their experiments demonstrated that the respiratory and circulatory centers possessed great resistance to hypoxia and could be revived after the circulation had been arrested for as long as thirty minutes. They stated, however, that certain centers, which probably were situated in the cerebrum, were more sensitive to anemia and were irreparably damaged if the circulation were arrested for more than five minutes.

Arrest of circulation in spinal cord.—As early as 1667 Stenon (93) reported that anemia of the spinal cord produces paralysis at the end of one minute and suppression of sensitivity and motor functions after three minutes. Legallois (see 66) in 1830 reported that ligation of the abdominal aorta produced paralysis of motor spinal functions but that the spinal centers may recover their function if the circulation has not been obstructed too long.

Since this early work a number of investigators (12, 13, 14, 15, 21, 36, 69, 84, 92, 97, 100) have reported the effects of interruption of the circulation of the spinal cord. Many of these studies were made following obstruction of the abdominal aorta.

HISTOLOGIC STUDIES OF STRUCTURAL CHANGES

*Anqxic hypoxia.*Thorner and Lewy (96) in 1940 reported experiments performed on guinea pigs and cats which had been subjected to complete hypoxia by being placed in an environment of pure nitrogen for various periods of time. These workers found that exposures to sublethal periods of pure hypoxia produced vascular and degenerative changes in the central nervous system. It was emphasized that some of these changes were irreversible and became summated in animals repeatedly subjected to hypoxia.

Following fatal cases of nitrous oxide-oxygen anesthesia, lesions of the brain, especially in the cortex and basal ganglia, have been observed (41, 70). These changes have been attributed to anoxic hypoxia.

It has been suggested by van der Molen (98) that cortical cell changes occur at partial pressures of oxygen equivalent to an altitude of 28,000 feet (8,535 meters) and, moreover, that some of these changes might be irreversible. It will be remembered, however, that the average unacclimatized individual cannot live much beyond an

altitude of 25,000 feet (7,620 meters). Only individuals thoroughly acclimated could withstand an altitude of 28,000 feet; it is known, of course, that several members of the various Mount Everest expeditions were reasonably well acclimated to this great height.

Windle and his co-workers (105, 106, 107, 108), during the early 1940's, carried out extensive researches on the central nervous system of full-term guinea pig fetuses which had been subjected to severe grades of hypoxia (and of asphyxia). (Some of these animals were resuscitated and later subjected to learning tests.) Controlled histopathologic studies were made. Neuropathologic changes of various degrees of severity were observed, which were not necessarily related to the duration of the hypoxia. Among the changes noted were capillary hemorrhages, clouding of Nissl substances, shrinkage of the neuron, and loss of stainability. In some instances, there was a generalized necrosis of the brain and spinal cord with chromatolysis and edema. Glial proliferation and loss of nerve cells, especially in the pyramidal layers of the cerebral cortex, were also found.

Morrison (74) in 1946 made comprehensive histologic observations on twenty-five dogs and ten monkeys which had been subjected to various degrees of hypoxia. He observed that a single exposure to a simulated altitude of 32,000 feet (9,755 meters) for twenty-five minutes produced extensive lamina necrosis in the cortex of the monkey.

Repeated exposures of moderate hypoxia (12–13 volumes per cent of oxygen in the blood) showed that the first histologic changes occurred in the cell bodies of the cortical gray matter. When 10 volumes per cent oxygen were used, and the animals subjected to repeated exposures, the white matter became involved, demyelinization appearing in the corpus callosum and centrum semiovale.

It was observed further that during severe hypoxia the frontal lobe was most often, and the temporal lobe least often, involved. The cerebellum was more often affected than the basal ganglion. The spinal cord and medulla were not affected by hypoxia compatible with life.

In 1945 Hoff, Grenell, and Fulton (57), working with guinea pigs, reported that hypoxia caused marked changes in the cell, which involved the cytoplasm, nuclei, and Nissl substance. Damaged cells were found in various locations of the brain, but those in the medulla and cerebellar cortex were especially involved.

Metz (73) in 1949, after subjecting several different species of vertebrates (goldfish, frogs, turtles, pigeons, and rats) to severe grades of hypoxia, commented on the fact that he did not see much histologic nerve damage. He emphasized the possibility that the changes which may have occurred were not morphologic in nature but rather were biochemical phenomena at a submicroscopic level. This is an interesting observation and suggests further researches along this line.

Recently Hager *et al.* (46) studied electron-microscopic changes in brain tissue of hamsters following acute hypoxia. The studies suggested that there is a rise of intracellular osmotic pressure and disintegration in both the perikaryon and the mitochondria.

Gerard (38), from his studies on hypoxia and neural metabolism, has concluded that one of the functions of oxygen is to keep the cell membrane polarized and, further, that proteolytic processes are initiated by complete hypoxia. It is thought that the accumulation of lactic acid in severe degrees of hypoxia may be partially responsible for this reaction.

Gellhorn *et al.* (37) have suggested that hypoxia and hypoglycemia have a similar physiologic action on the central nervous system and that they act synergistically in the production of convulsive seizures. Sugar and Gerard (95) have also suggested that hypoglycemia acts much like hypoxia on the function of the brain, since it leads to interference with oxidation in that organ.

Hemic and stagnant hypoxia.—Histologic studies of nervous tissue have been made on the differential effects of hypoxia following anemia. Gomez and Pike (41) in 1909, working with cats, reported histologic changes in nerve cells brought about by total anemia of the central nervous system. The order of susceptibility of the cells of the central nervous system to oxygen want, as shown by histological studies, was as follows: small pyramidal cells, Purkinje cells, cells of the medulla oblongata, cells of retina, cells of cervical cord, cells in lumbar cord, and sympathetic ganglionic cells.

Gildea and Cobb (39) in 1930, studying pathologic effects of cerebral anemia, observed nonspecific cortical lesions, such as focal areas of necrosis and swollen and shrunken ganglion cells. The most pronounced effect was noted in the cells of lamina III and IV of the cortex.

In 1938 Greenfield (42) reviewed previous work on neuronal damage from stagnant and anoxic hypoxia. He emphasized that there are considerable differences in the responses of different nerve cells.

Weinberger *et al.* (101) in 1940, working with cats, produced temporary anemia by occluding the pulmonary artery. At the end of three minutes and ten seconds, permanent and severe pathologic changes were found in the cerebral cortex. Longer periods of hemic

hypoxia produced lesions in the Purkinje cells of the cerebellum and in nerve cells in the basal ganglion.

Effect of anemia on cells of spinal cord: A number of investigators (34, 47, 81, 92, 99) have made histologic studies of certain nerve cells after the circulation of the spinal cord had been partially or totally arrested. For the most part, severe anemia (ischemia) produced grave damage to the cells, and in some instances necrosis and destruction occurred. The amount of damage, of course, depended upon the severity and duration of the anemia. Some cells—for example, those of the spinal ganglia—withstood anemia much better than others.

These studies on the cells of the spinal cord have important clinical significance. They are especially pertinent in surgical operations involving important blood vessels, particularly the aorta. Recently, however, the use of extracorporeal circulation has removed many dangers in this area.

As might be expected, arrest of circulation produces grave organic changes in the cells of the central nervous system within a relatively short time. It has been emphasized by Sugar and Gerard (95), however, that while the damages which follow sudden anemia are primarily due to hypoxia, there are other important contributing factors. Those which they mention are hypoglycemia, hypercapnia, and the increased extracellular potassium.

Carbon monoxide poisoning.—The effect of carbon monoxide on the nervous system has engaged the attention of numerous workers (53, 54, 58, 75, 91, 96, 111). Not only has necrosis of nerve fibers in the brain been observed, but necrosis in the peripheral nerves, as well (58, 91).

In 1934 Yant *et al.* (111) made extensive investigations of histologic changes produced in the central nervous system of dogs following administration of carbon monoxide; various pronounced lesions were found.

In 1946 Lhermitte and De Ajuriaguerra (68) reported that if death rapidly followed carbon monoxide poisoning, hemorrhages, necrosis, and edema occurred. These changes primarily involved the lenticular nuclei but the subcortical white matter, the hippocampus, the substantia nigra, and the cerebellum also were affected. If carbon monoxide poisoning is continued for long, changes appear in the vascular network with infiltration of the walls by neutral lipids and other substances, such as ferric salts and calcium.

These authors suggest that a toxic factor in addition to the anoxic factor in carbon monoxide poisoning affects the neuroglia and the vascular network with specific involvement of the basilar region and

the white fibers of the centrum ovale. In this connection, Thorner and Lewy (96) in 1940 raised the interesting question whether the cerebral changes in carbon monoxide poisoning are actually typical of hypoxia or are caused by other factors.

Dutra (32) in 1952, studying the brain of man, reported that cerebral lesions which occur as residua of carbon monoxide poisoning consist essentially of dilatation of blood vessels, edema, perivascular hemorrhages, degeneration and death of ganglionic cells, focal demyelinization, and foci of necrosis. He felt that these lesions were either directly or indirectly caused by diminution of the supply of oxygen.

Obviously, carbon monoxide poisoning is capable of producing severe damage to nervous tissue. Some of the histologic changes following severe poisoning are irreversible, so that permanent damage has been done, and as Drinker has pointed out, individuals may be practically decerebrated.

CHEMISTRY OF THE BRAIN

During the past two decades or so, considerable research has been done on the chemistry of the brain during hypoxia. Several investigators (6, 7, 44, 45) have found an increase in lactic acid during anoxic hypoxia. Gurdjian *et al.* (44, 45) in 1944 reported that cerebral lactic acid rose when the oxygen content of inspired air fell to 10–13 per cent. Criscuolo and Biddulph (26) in 1958, working with rats, found that adrenalectomy prevented an increase in lactic acid of the brain during hypoxia. If, however, epinephrine were administered, the usual rise of lactic acid during hypoxia was observed. The authors felt that this finding suggested that blood sugar is the substrate for lactic acid.

There is evidence that hypoxia causes a decrease in phosphocreatine. Gurdjian *et al.* (44) reported a decrease of phosohocreatine when animals breathed 7 per cent oxygen. No change, however, was noted in cerebral adenosine triphosphate. In 1953 Albaum *et al.* (2), working with rabbits, subjected them to progressive stages of hypoxia and correlated the chemical changes in the brain with electrical measurement of function. Moderate decreases of adenosine triphosphate, creatine phosphate, and glycogen were observed. These decreases, however, were not noted until the stage of inexcitability had been reached.

Welsh (102) subjected rats to anoxic hypoxia (200–100 mm. Hg

barometric pressure) for one to two hours and observed that the acetylcholine in the brain was decreased by approximately one-third to one-half. Insulin hypoglycemia was found to cause a greater decrease in acetylcholine than anoxic hypoxia. It was suggested that the decline in free acetylcholine might account for the decrease in excitability of the cortex under conditions of hypoxia and of hypoglycemia.

Dixon (29) in 1949 studied changes in the concentration of potassium in slices of rabbit cerebral cortex, which were bathed in a bicarbonate-Ringer's solution. In the absence of glucose a loss of potassium from the tissues was noted. With active utilization of glucose, however, there was an increase in the uptake of potassium. In this respect brain tissue resembles other tissues of the body.

The chemistry of the brain during hypoxic states obviously needs further investigation. Studies which correlate the chemical changes with electrical activity of the brain are especially needed.

ABILITY OF YOUNG ANIMALS TO WITHSTAND ASPHYXIA AND HYPOXIA

It has been known for well over two centuries that young animals are considerably less susceptible to asphyxia than adults. As early as 1725 Robert Boyle (10) commented on the resistance of kittens to asphyxia, and Paul Bert (5) in 1870 called attention to the fact that newborn animals were capable of withstanding prolonged asphyxia. Since that time many observers have reported studies on asphyxia and also on hypoxia in young animals and have confirmed and extended the earlier work.

Studies have been made on rats (1, 8, 9, 11, 18, 27, 35, 48, 55, 83, 88, 89, 90, 104), on dogs (33, 35, 40, 55, 61, 64, 88, 90, 104), on guinea pigs (18, 35, 40, 104), on rabbits (35, 40, 88, 90, 104), on cats (35, 64, 90, 104), and on mice (3, 59, 79, 80). A few observations have also been made on chicks and ducklings (82) and on the opossum (64). Newborn human infants, too, are capable of withstanding considerable periods of hypoxia; several workers have emphasized this (24, 71, 104).

Space does not permit giving details concerning all these experiments. Suffice it to say that the problem has been approached in numerous ways, and various grades and different types of hypoxia were used; the length of exposure was also varied. A few typical experiments may be cited.

Kabat (60) in 1940, studying resistance of very young puppies to arrest of brain circulation, found they were much more resistant to acute hypoxia than adult animals. The respiratory center in the newborn animal continued to function seventeen times as long as in the adult. The newborn also achieved complete functional recovery much more quickly than did the adult animal. At the age of four months, the resistance was diminished to the adult level.

Fazekas, Alexander, and Himwich (35) in 1941 studied the tolerance of the adult and infant of various species (rat, dog, cat, rabbit, and guinea pig) to hypoxia. The newborn exhibited a much greater tolerance to hypoxia than adults. Tolerance varied in the different species; for example, tolerance was longest in the physiologically immature newborn rats and shortest in the comparatively mature guinea pig. The authors suggested that in the newborn puppy and rat the factor permitting survival was poikilothermia, the fall of temperature diminishing the metabolic demands. It has also been demonstrated that in these two animals there is a lower cerebral metabolic rate.

Glass, Snyder, and Webster (40) in 1944, working with dogs, rabbits, and guinea pigs, subjected to pure nitrogen, concluded that tolerance to hypoxia is related to the stage of development rather than to environment. Interesting results were obtained with suckling rabbits breathing pure nitrogen. The survival period at one week was ten minutes; at two weeks, four minutes; and at three weeks, one and a half minutes, the last value being the same as that of the matured animal. These authors emphasized that the defense of the fetus against asphyxia is important because of the increased hazard of respiratory failure during the terminal phase of intrauterine life and the early neonatal period.

Selle (89) has pointed out that the increased tolerance of young animals to hypoxia is apparently due to several factors: (*a*) a low metabolic rate of the central nervous system, (*b*) poikilothermia, and (*c*) an anaerobic source of energy. Kabat (60) and Jelinek (59) also feel that the newborn can obtain anaerobic energy from glycolysis to a greater extent than adults. It has been shown by Himwich and his associates (55) that insulin reduces, and glucose increases, the survival of young animals placed in pure nitrogen. He and his coworkers (56), studying the survival of young animals which had been given sodium cyanide (which inhibits the cytochrome system), demonstrated clearly that anaerobic energy is available to young animals.

De Haan and Field (27) in 1959, working with rats, felt that young

animals can withstand hypoxia better than adults because of high glycogen levels and the infant's ability to metabolize lactic and pyruvic acids to lipids.

REFERENCES

1. Adolph, E. F. 1948. *Amer. J. Physiol.*, 155: 366.
2. Albaum, H. G.; Noell, W. K.; and Chinn, H. I. 1953. *Amer. J. Physiol.*, 174: 408.
3. Avery, R. C., and Johlin, J. M. 1932. *Proc. Soc. Exp. Biol. Med.*, 29: 1184.
4. Battelli, F. 1900. *J. Physiol. Path. Gen.*, 2: 443.
5. Bert, P. 1870. *Physiologie de la respiration*. Paris.
6. Biddulph, C., *et al.* 1958. *Amer. J. Physiol.*, 193: 345.
7. ———. 1959. *J. Appl. Physiol.*, 13: 486.
8. Bollman, J. H.; Fazio, A. N.; and Faulconer, A. 1951. *Anesthesiology*, 12: 420.
9. Borgard, W., and Hoffman, F. 1939. *Arch. Gynaek.*, 168: 873.
10. Boyle, R. 1725. *The Philosophical Works of Boyle*. London: W. and J. Innys.
11. Britton, S. W., and Kline, R. F. 1945. *Amer. J. Physiol.*, 145: 190.
12. Brown-Sequard, E. 1851. *C. R. Soc. Biol. (Par.)*, 32: 855.
13. ———. 1855. *Ibid.*, 41: 628.
14. ———. 1855. *Ibid.*, 45: 562.
15. ———. 1858. *J. Physiol. Homme*, 1: 95, 117, 353.
16. Brukhonenko, S., and Tchetchuline, S. 1929. *J. Physiol. Path. Gen.*, 27: 64.
17. Bunce, D. F. M. 1961. *Fed. Proc.*, 20: 100.
18. Cameron, J. A. 1941. *J. Cell. Comp. Physiol.*, 18: 379.
19. Cannon, W. B., and Burkett, I. R. 1913. *Amer. J. Physiol.*, 32: 347.
20. Cobb, S., and Freemont-Smith, F. 1931. *Arch. Neurol. Psychiat.*, 26: 731.
21. Colson, C. 1890. *Arch. Biol. (Par.)*, 10: 431.
22. Cooper, A. 1836. *Guy Hosp. Rep.*, 7: 457.
23. Courtice, F. C. 1941. *J. Physiol.*, 100: 198.
24. Crehan, E. L.; Kennedy, R. L. J.; and Wood, E. H. 1950. *Proc. Mayo Clinic*, 25: 392.
25. Crile, G., and Dolley. 1908. *J. Exp. Med.*, 10: 782.
26. Criscuolo, D., and Biddulph, C. 1958. *Proc. Soc. Exp. Biol. Med.*, 98: 118.

26a. Davies, P. W., and Bronk, D. W. 1957. *Fed. Proc.*, 16: 689.

27. De Haan, R. L., and Field, J. 1959. *Amer. J. Physiol.*, 197: 445.
28. d'Hallium, M. 1904. *Presse Med.*, 12: 345.
29. Dixon, K. C. 1949. *Biochem. J.*, 44: 187.
30. Drinker, C. K. 1938. *Carbon Monoxide Asphyxia*, p. 133. New York: Oxford University Press.
31. Dumke, P. R., and Schmidt, C. F. 1943. *Amer. J. Physiol.*, 138: 421.
32. Dutra, F. R. 1952. *Amer. J. Clin. Path.* 22: 925.
33. Ederstrom, H. E. 1959. *Proc. Soc. Exp. Med. Biol.*, 100: 741.
34. Ehrlich, P., and Brieger, L. 1884. *Z. Klin. Med.*, 8 (Suppl.): 155.
35. Fazekas, J. F.; Alexander, F. A. D.; and Himwich, H. E. 1941. *Amer. J. Physiol.*, 134: 281.
36. Gelfan, S., and Tarlov, I. M. 1953. *Fed. Proc.*, 12: 50.
37. Gellhorn, E.; Ingraham, R. C.; and Moldavsky, L. 1938. *J. Neurophysiol.*, 1: 301.
38. Gerard, R. W. 1938. *Arch. Neurol. Psychiat.*, 40: 985.
39. Gildea, E. F., and Cobb, S. 1930. *Arch. Neurol. Psychiat.*, 23: 876.
40. Glass, H. G.; Snyder, F. F.; and Webster, E. 1944. *Amer. J. Physiol.*, 140: 609.
41. Gomez, L., and Pike, F. H. 1909. *J. Exp. Med.*, 11: 257.
42. Greenfield, J. G. 1938. *J. Neurol. Psychiat.*, 1: 306.
43. Grenell, R. G. 1946. *J. Neuropath. Exp. Neurol.*, 5: 131.
44. Gurdjian, E. S.; Stone, W. E.; and Webster, J. E. 1944. *Arch. Neurol. Psychiat.*, 51: 472.
45. Gurdjian, E. S.; Webster, J. E.; and Stone, W. E. 1949. *Amer. J. Physiol.*, 156: 149.
46. Hager, H.; Hirschberger, W.; and Scholz, W. 1960. *Aerospace Med.*, 31: 379.
47. Haggquivst, G. 1940. *Acta Med. Scand.*, 104: 8.
48. Herrlich, H. C.; Fazekas, J. F.; and Himwich, H. E. 1941. *Proc. Soc. Exp. Biol. Med.*, 48: 466.
49. Heymans, C. 1950. *Physiol. Rev.*, 30: 395.
50. Heymans, C., *et al.* 1937. *Arch. Neurol. Psychiat.*, 38: 304.
51. Hill, L. 1896. *The Cerebral Circulation*. London: Churchill.
52. ———. 1900. *Trans. Roy. Soc., London*, B, 193: 69.
53. Hill, L., and Semerak, C. B. 1918. *J.A.M.A.*, 71: 649.
54. Hiller, F. 1924. *Z. Ges. Neurol. Psychiat.*, 93: 594.
55. Himwich, H. E.; Alexander, F. A. D.; and Fazekas, J. F. 1941. *Amer. J. Physiol. (Proc.)*, 53: 193.
56. Himwich, H. E., *et al.* 1942. *Amer. J. Physiol.*, 135: 387.
57. Hoff, E. C.; Grenell, R. G.; Fulton, J. F. 1945. *Medicine*, 24: 161.
58. Hsu, Y. K., and Cheng, Y. L. 1938. *Brain*, 61: 384.
59. Jelinek, V. 1950. *Biol. Listy* (Prague), 31: 76.
60. Kabat, H. 1940. *Amer. J. Physiol.*, 130: 588.
61. Kabat, H.; Dennis, C.; and Baker, A. B. 1941. *Amer. J. Physiol.*, 132: 737.
62. Kety, S. S. 1958. *In: Circulation (Proc. Harvey Tercentary Congress)*, p. 331. Oxford: Blackwell.
63. Kety, S. S., and Schmidt, C. F. 1948. *J. Clin. Invest.*, 27: 484.
64. Kline, R. F., and Britton, S. W. 1945. *Fed. Proc.*, 4: 41.

65. Lassen, N. A. 1959. *Physiol. Rev.*, 39: 183.
66. Legallois, cited by Heymans. 1950. *Physiol. Rev.*, 30: 381.
67. Lennox, W. G., and Gibbs, E. L. 1932. *J. Clin. Invest.*, 11: 1155.
68. Lhermitte, J., and Ajuriaguerra, De. 1946. *Sem. Hôp. Paris*, 22: 1945.
69. Litten, M. 1880. *Z. Klin. Med.*, 1: 131.
70. Lowenberg, K.; Waggoner, R. W.; and Zbinden, T. 1936. *Ann. Surg.*, 104: 801.
71. Mabry, C. D. 1959. *J. Pediat.*, 55: 211.
72. Mayer, S. 1878. *Med. Centralbl.*, 16: 579.
73. Metz, B. 1949. *Fed. Proc.*, 8: 109.
74. Morrison, L. R. 1946. *Arch. Neurol. Psychiat.*, 55: 1.
75. Neighbors, D., and Garrett, C. C. 1931. *Texas J. Med.*, 27: 513.
76. Opitz, E., and Schneider, M. 1950. *Ergebn. Physiol.*, 46: 126.
77. Petroff, J. R. 1931. *Z. Ges. Exp. Med.*, 75: 1.
78. Pike, F. H.; Guthrie, C. C.; and Stewart, G. N. 1908. *J. Exp. Med.*, 10: 490.
79. Reiss, M. 1931. *Z. Ges. Exp. Med.*, 79: 345.
80. Reiss, M., and Haurowitz, F. 1921. *Klin. Wschr.*, 8: 743.
81. Righetti, H., cited by Heymans. 1950. *Physiol. Rev.*, 30: 375.
82. Rostorfer, H. H., and Rigdon, R. H. 1947. *Biol. Bull.*, 92: 23.
83. Samson, F. E., Jr., and Dahl, N. 1956. *Fed. Proc.*, 15: 161.
84. Schiffer. 1869. *Centralbl. Med. Wissensch.*, Nos. 37 and 38: 579, 593.
85. Schmidt, C. F. 1928; 1932; 1934; 1936. *Amer. J. Physiol.*, 84: 202; 102: 94; 110: 137; and 114: 572.
86. ———. 1943. *The Cerebral Circulation in Health and Disease.* Springfield, Ill.: Thomas.
87. Schmidt, C. F., and Pierson, C. J. 1934. *Amer. J. Physiol.*, 108: 241.
88. Selle, W. A. 1941. *Proc. Soc. Exp. Biol. Med.*, 48: 417.
89. ———. 1944. *Amer. J. Physiol.*, 141: 297.
90. Selle, W. A., and Witten, T. A. 1941. *Proc. Soc. Exp. Biol. Med.*, 47: 495.
91. Shaffer. 1903. *Centralbl. Nervenk. Psychiat.* (new series), 14: 485.
92. Spronck, C. H. D., cited by Heymans. 1950. *Physiol. Rev.*, 30: 381.
93. Stenon, N., cited by Heymans, *ibid.*
94. Stratton, G. M. 1919. *Sci. Monthly*, 8: 421.
95. Sugar, O., and Gerard, R. W. 1938. *J. Neurophysiol.*, 1: 558.
96. Thorner, M. W., and Lewy, F. H. 1940. *J.A.M.A.*, 115: 1595.
97. Tureen, L. L. 1936. *Arch. Neurol. Psychiat.*, 35: 789.
98. van der Mole, H. R. 1939. *Ned. T. Geneesk.*, 83: 4921.
99. Van Harreveld, A., and Marmont, G. 1939. *J. Neurophysiol.*, 2: 101.
100. Vulpian, A., cited by Heymans. 1950. *Physiol. Rev.*, 30: 381.
101. Weinberger, L. M.; Gibbon, M. H.; and Gibbon, J. H., Jr. 1940. *Arch. Neurol. Psychiat.*, 43: 615, 961.
102. Welsh, J. H. 1943. *J. Neurophysiol.*, 6: 329.
103. Werthiemier, E., and Dubois, C. 1911. *C. R. Soc. Biol.* (*Par.*), 70: 304.
104. Wilson, J. L., *et al.* 1948. *Pediatrics*, 1: 581.
105. Windle, W. F. 1944. *Psychosom. Med.*, 6: 158.
106. Windle, W. F., and Becker, R. F. 1942. *Proc. Soc. Exp. Biol. Med.*, 51: 213.
107. ———. 1943. *Amer. J. Obstet. Gynec.*, 45: 183.
108. Windle, W. F.; Becker, R. F.; and Weil, A. 1944. *J. Neuropath. Exp. Neurol.*, 3: 224.
109. Wolff, H. G. 1936. *Physiol. Rev.*, 16: 545.
110. Wolff, H. G., and Lennox, W. G. 1930. *Arch. Neurol. Psychiat.*, 23: 1097.
111. Yant, W. P., *et al.* 1934. *Pub. Health Bull.* (U.S. Public Health Service), No. 211.

CEREBROSPINAL FLUID

Pressure

In 1960 Small *et al.* (20) reported the effect on anesthetized dogs of the inspiration of 8 per cent oxygen in nitrogen mixtures. Cerebrospinal fluid, arterial blood, and central venous pressures were all measured simultaneously with modern pressure transducers. The peak increase in cerebrospinal fluid pressure, occurring at four minutes on the average, was 108 per cent over the control. Mean arterial blood pressure increased 31 per cent and venous pressure 69 per cent at the same time. Vasodilation in the brain as well as increased blood pressures, both arterial and venous, were suggested as the causes of the rise in cerebrospinal fluid pressure. Earlier experimenters have reported similar findings in both dogs and cats. Most have found an early rise in short bouts of severe hypoxia (2, 15, 17). With longer exposures the terminal increase may be lessed marked or absent (1, 7, 23, 25). Edstrom and Essex (3) found the rise occurring for thirteen to thirty-three minutes following the breathing of pure nitrogen gas until near collapse.

According to present concepts (10, 21), hypoxia can cause cerebral vasodilation and increased cerebral blood flow. Since brain and cerebrospinal fluid are incompressible, in order for the cranium to accommodate the extra volume of blood there must be a shift of fluid from the cranial cavity. In the process cerebrospinal fluid pressure is apparently elevated, and cerebrospinal fluid absorption into the venous outflow is probably increased temporarily until a new equilibrium is reached.

PHYSIOLOGICAL TRAINING 1980

DEPARTMENT OF TRANSPORTATION
FEDERAL AVIATION ADMINISTRATION
MIKE MONRONEY AERONAUTICAL CENTER
CIVIL AEROMEDICAL INSTITUTE
PHYSIOLOGICAL OPERATIONS

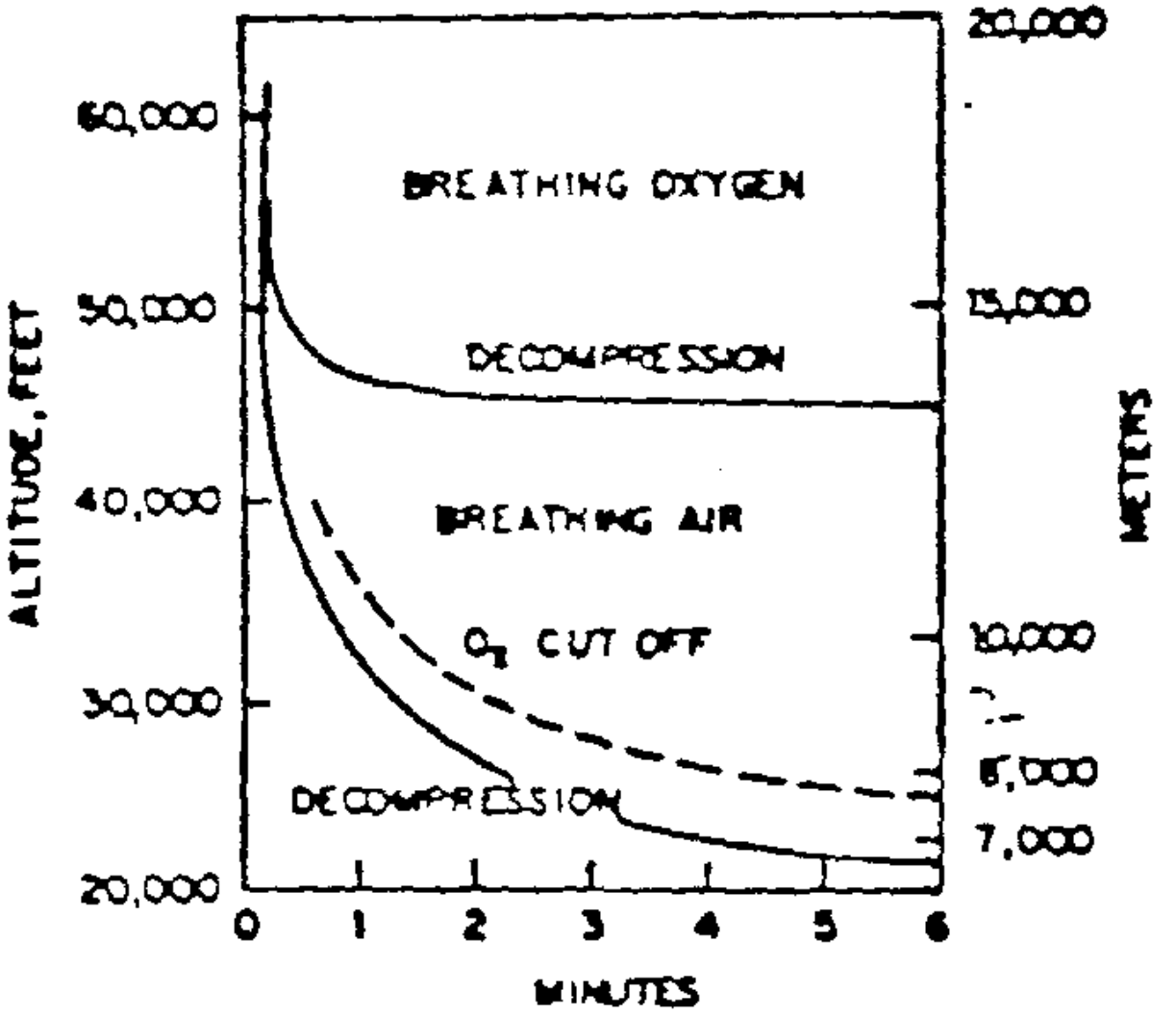


FIG. 33. Time of useful consciousness at altitude. *Solid curve below*, after rapid decompression from sea level while breathing air. *Solid curve above*, after rapid decompression from 10,058 m (33,000 ft) breathing 100% oxygen. *Dotted curve*, after separation from oxygen supply while at altitude. After Luft (50).

INTRODUCTION TO PHYSIOLOGICAL TRAINING

Humans have a remarkable ability to adapt to their surroundings. The human body makes adjustments for changes in external temperature, acclimates to barometric pressure variations from one habitat to another, compensates for motion in space and postural changes in relation to gravity, resists toxic agents and diseases, and performs all these adjustments while meeting changing energy requirements for varying amounts of physical and mental activity. The human body does adjust to acute and chronic reductions in its' oxygen supply by increasing respiration, chemical changes in the blood, and by increasing production of red blood cells; however, a complete absence of oxygen will cause death in approximately five to eight minutes.

In aviation, the demands upon the compensatory mechanisms of the body are numerous and of considerable magnitude. The environmental changes of greatest physiological significance involved in flight are: marked changes in barometric pressure, considerable variation in temperature, and movement at high speed in three dimensions.

The advances in aeronautical and mechanical engineering in the past decade have resulted in the development of highly versatile aircraft. Since we are essentially ground creatures, we must learn how to adjust to the low pressures and temperatures of flight, and the effects of acceleration on the body. Low visibility with its concomitant problems of disorientation, and problems related to the general physical and mental stress associated with flight, should also be considered. Humans cannot operate these machines at full capacity without physical aids, such as a supplemental supply of oxygen and pressurized cabins for use at altitudes starting as low as 10,000 feet.

We must overcome the handicaps imposed by nature on an organism designed for terrestrial life. In particular, the limiting factors in adjustment of the human body to flight must be appreciated. The extent to which these limiting factors are alleviated by available equipment must be understood clearly. Indifference, ignorance, and carelessness can nullify the foresight, ingenuity, and effort involved in supplying the pilot with efficient equipment.

An effort is made in the following pages to outline some of the important factors regarding physiological effects of flight, and to describe the devices and procedures that will contribute to the safety and efficiency of all who fly.

FOREWORD

Aviation Physiology deals with the physical and mental effects of flight on air crew personnel and passengers. Study of this booklet will familiarize you with some of the physiological problems of flight, and will instruct you in the use of some devices that Aviation Physiologists and others have developed to assist in the compensation for the numerous environmental changes that are encountered in flight.

For most of you, Aviation Physiology may be an entirely new field. To others, it is something that you were taught while in the military service or elsewhere.

It is our sincere hope that we can enlighten, stimulate, and assist you during your brief stay with us. After you have returned to your regular routine, remember that the Physiological Operations & Training Section of the Civil Aeromedical Institute strives to assist with problems concerning Aviation Physiology.

Inquiries should be addressed to:

Mike Monroney Aeronautical Center
Civil Aeromedical Institute
Physiological Operations & Training Section, AAC-143
P.O. Box 25082
Oklahoma City, Oklahoma 73125

Phone: (405) 686-4837
FTS: 732-4837

PHYSIOLOGICAL TRAINING

PHYSICS OF THE ATMOSPHERE

Perhaps the primary problem of flight related to physiology has to do with the fact that the pressure of the gases in the atmosphere change as we ascend and descend. Thus, it is essential that we have an understanding of the gases found in the atmosphere and their effects upon the body. There are other factors such as temperature change which also need to be understood if we are to protect ourselves from potential hazard.

COMPOSITION OF THE ATMOSPHERE

The atmosphere is a mixture of gases. It is composed primarily of nitrogen (N_2) and oxygen (O_2). The gases other than nitrogen and oxygen are so low in percentage that they are considered to be negligible from the standpoint of aviation physiology. Therefore, we usually refer to the percentages of nitrogen and oxygen as approximately 80% and 20% respectively, rather than their true percentages of 78.08% and 20.95%.

A. Nitrogen — 78.08%

This gas is responsible for the major portion of the total atmospheric pressure or weight. The gas itself is inert as far as the human body is concerned. Once the body becomes saturated, the same amount of nitrogen is exhaled as was inhaled. In other words, the body utilizes none of the nitrogen, although it is saturated with it.

B. Oxygen — 20.95%

This gas is essential for life. When the body is deprived of oxygen, death follows a short time later. Each time we breathe, about 21% of that breath is oxygen. In the lungs this gas is absorbed into the blood stream and is carried by the blood to all parts of the body. It is used to burn or oxidize food material and for the production of heat and kinetic energy.

METHODS OF EXPRESSING ALTITUDE

The gaseous atmosphere surrounding the earth is affected by the gravitational pull of the earth. This gravitational pull is expressed as weight per unit area and is referred to as standard atmospheric pressure. This pressure is 14.7 pounds per square inch (p.s.i.), 29.92 inches of mercury (in. Hg) or 760 millimeters of mercury (mm Hg). Standard atmospheric conditions exist at mean sea level in mid-latitudes at plus fifteen degrees Centigrade (+15°C) or (+59°F). The weight or pressure of the gases decreases as altitude increases.

PHYSIOLOGICAL DIVISIONS OF THE ATMOSPHERE

The divisions of the atmosphere are primarily physical or meterological in nature. Most people are familiar with the troposphere and the stratosphere. They exist and are important for the aviator to know. However, from the point of view of the physiological effects associated with flight, the following divisions give a better view of the problem areas:

A. Physiological Zone

This area extends from sea level to about 12,000 feet and represents that area of the atmosphere to which the human body is more or less adapted. Only minor physiological problems exist when flying within this zone, e.g., middle ear and sinus trapped gas difficulties. Shortness of breath, dizziness, and headache are common symptoms noticed by individuals who go to higher than acclimatized levels and either exert themselves or stay there too long. Above this zone, we are in a foreign environment to which the human body is unaccustomed, unless we have become acclimatized. The process of acclimatization will be discussed later. The barometric pressure drops from 760 mm Hg to 483 mm Hg in this zone.

B. Physiological Deficient Zone

This zone extends from about 12,000 feet to 50,000 feet and represents, along with the preceding zone, the area where the majority of flying is accomplished at the present time. Because of the reduced atmospheric pressure, this is the zone where oxygen deficiency becomes an ever increasing problem as we ascend. Trapped gases in the intestinal tract and evolved gas problems may also occur within this zone. The pressure changes from 483 mm Hg at 12,000 feet to 87 mm Hg at 50,000 feet.

C. Partial Space Equivalent Zone

From the point of view of extent in miles this is an overwhelming area, but from the point of view of pressure change, it is small. The extent of this zone is from 50,000 feet to 120 miles, the pressure change represented is just a little over 1 p.s.i. The peculiar problems of flight over 50,000 feet are essentially the same as for humans existing in total space. The need for sealed cabins, pressure suits, and the problems of blood and body fluids boiling above 63,000 feet plus the gravitational changes on the body make this a space-equivalent zone. Advances in technology have reached a point to where supersonic transports (Concorde) are presently operating safely in this zone.

D. Total Space Equivalent Zone

This zone extends from 120 miles on and represents what might be called true space. However, except for a few physiological and psychological problems peculiar to space travel, the physiological problems of this zone are identical with those of the preceding zone.

PHYSICAL GAS LAWS

A human body rapidly exposed to an altitude of 45,000 feet clothed in everyday street apparel could become unconscious in 9-12 seconds, may be reduced to a vegetable in a few minutes, and perhaps be dead a few minutes later. The dangerous element here is the reduced oxygen pressure found at this level. Since air is a mixture of gases, it behaves as such and is subject to those laws which govern gases. The following laws help to explain the effects of reduced barometric pressure and its interplay on the human body.

A. Graham's Law

This law states that a gas of high pressure exerts a force toward a region of lower pressure, and that if an existing membrane separating these regions of unequal pressure, is permeable or semi-permeable, the gas of higher pressure will pass (or diffuse) through the membrane into the region of low pressure. This process, which occurs in milliseconds, continues until the unequal regions are nearly equal in pressure. This law explains the transfer of oxygen, carbon dioxide, and other gases from one part of the body to another. This law is also thought to be a contributing factor in the processes of Boyle's Law and Henry's Law.

B. Boyle's Law

Boyle's Law states the volume of a gas is inversely proportional to the pressure, temperature remaining constant; or, if the pressure on a gas decreases, its volume increases and vice versa. This law, when applied to your body, explains the expansion of gases trapped within the body in such areas as the middle ear, sinuses, teeth, stomach, and the intestines.

C. Henry's Law

This law states that the amount of gas in solution varies directly with the pressure of that gas over the solution; or, when the pressure of a gas over a certain liquid decreases, the amount of that gas dissolved in the liquid will also decrease (or vise versa). This gas law affects the body in that gases, primarily nitrogen, will come out of solution when the body is exposed to reduced atmospheric pressure. This occurs because the pressure of nitrogen is reduced proportionately as the total atmospheric pressure is reduced (see Dalton's Law). This "evolved" gas phenomenon may lead to disorders similar to the "Bends" which a deep sea diver experiences as a result of rapid ascent to the surface (going from an area of high external pressure, therefore high N_2 saturation, to an area of lower external pressure and lower nitrogen saturation, possibly resulting in nitrogen bubble formation throughout the body).

D. Charles' Law

This law states that if the volume of a gas remains constant, the pressure will vary directly with the temperature. This law has no direct physiological significance since body temperature remains fairly constant. It does, however, explain the fact that pressure within supplemental oxygen cylinder(s) will decrease if the ambient temperature surrounding the storage cylinder(s) decreases. Since we may expect a 2°C or 3½°F drop in temperature per 1,000 feet as we ascend to altitude (stabilizing at approximately 35,000'), we should notice a drop in indicated pressure on the supplemental oxygen cylinder(s)' supply gage. This is a normal occurrence, and no reason to suspect the oxygen is being depleted. Rather, all the stored supplemental oxygen is still there and readily available for use if needed. The oxygen is simply more "compact" (less molecular activity) now due to the location of the storage containers in an unheated area of the aircraft. If the stored oxygen is carried in the passenger or pilots area of the aircraft, there is no need to even recall this particular gas law.

E. Dalton's Law

This law states that the total pressure of a mixture of gases is equal to the sum of the pressures of each gas in that mixture. Each gas exerts its own pressure, depending on the percentage of that gas in the mixture. The pressure of each gas in the mixture is expressed as the partial pressure (p) of that gas, i.e., pO_2 (for oxygen). The application of this law to aviation is that even though the percentage of oxygen in the atmosphere is constant (21%), its pO_2 will decrease proportionately as atmospheric pressure decreases. This reduction of pO_2 by ascent to altitude explains "altitude (or mountain) sickness." Later in this booklet, this "sickness" will be referred to to Hypoxic Hypoxia.

TABLE OF U.S. STANDARD ATMOSPHERE					
Feet	*In. of Hg*	*Mm of Hg*	*PSI*	C°	F°
0	29.92	760.0	14.69	15.0	59.0
2,000	27.82	706.7	13.66	11.0	51.9
4,000	25.84	656.3	12.69	7.1	44.7
6,000	23.98	609.1	11.91	3.1	37.6
8,000	22.23	564.6	11.77	–0.8	30.5
10,000	20.58	522.7	10.10	–4.8	23.4
12,000	19.03	483.4	9.34	–8.8	16.2
14,000	17.58	446.5	8.63	–12.7	9.1
16,000	16.22	412.0	7.96	–16.7	1.9
18,000	14.95	379.7	7.34	–20.7	–5.1
20,000	13.76	349.5	6.76	–24.6	–12.3
22,000	12.65	321.3	6.21	–28.6	–19.4
24,000	11.61	294.9	5.70	–32.5	–26.5
26,000	10.64	270.3	5.22	–36.5	–33.6
28,000	9.74	237.4	4.78	–40.5	–40.7
30,000	8.90	226.1	4.37	–44.4	–47.8
32,000	8.12	206.3	3.99	–48.4	–54.9
34,000	7.40	188.0	3.63	–52.4	–62.0
36,000	6.73	171.0	3.30	–55.0	–69.7
38,000	6.12	155.5	3.00	–55.0	–69.7
40,000	5.56	141.2	2.73	–55.0	–69.7
42,000	5.05	128.3	2.48	–55.0	–69.7
44,000	4.59	116.6	2.25	–55.0	–69.7
46,000	4.17	105.9	2.05	–55.0	–69.7
48,000	3.79	96.3	1.86	–55.0	–69.7
50,000	3.44	87.4	1.70	–55.0	–69.7
55,000	2.71	68.8	1.33	TEMPERATURE	
60,000	2.14	54.4	1.05	REMAINS CONSTANT	
63,000	1.95	46.9	.907		
64,000	1.76	44.7	.86		
70,000	1.32	33.5	*PSF* 113.2		
74,000	1.09	27.7	77.3		
80,000	.82	20.9	58.1		
84,000	.68	17.3	47.9		
90,000	.51	13.0	35.9		
94,000	.43	10.9	29.7		
100,000	.33	8.0	22.3		

Source: The ARDC Model Atmosphere – 1959

In. of Hg = Inches of Mercury
Mm of Hg = Millimeter of Mercury
PSI = Pounds per square inch
C° = Centigrade
F° = Fahrenheit
PSF = Pounds per Square Foot

RESPIRATION AND CIRCULATION

When the human organism is exposed by aerial flight to various stresses, both physiological and psychological, all body functions are affected. However, the areas of the body which are affected most directly are the respiratory and circulatory systems. Therefore, it is important for the individual to be familiar with the actions and limitations of human respiratory and circulatory systems.

RESPIRATION

A. *The Concept of Respiration*

Respiration is defined as the exchange of gases between the organism and its environment. The more obvious features of this process are the absorption of oxygen from the atmosphere and the elimination of some carbon dioxide from the body.

Cells in the body require oxygen for the burning of food material and the production of heat and energy. Carbon dioxide (CO_2) is produced from these reactions and excesses must be removed from the body. The lungs receive oxygen from the atmosphere which diffuses through the lungs into the blood. The blood at the same time releases carbon dioxide into the lungs and it is then expelled to the outside air. The oxygen absorbed by the blood is transported to nearly every cell in the body. The blood then receives carbon dioxide from the cells and carries it to the lungs for release.

The respiratory system is made up of the lungs, a series of conducting tubes called bronchi, the windpipe or trachea, and the mouth and nose. Air first enters the nasal passages where it is warmed, moisturized, and filtered. It then passes down the throat through the windpipe into the bronchial tubes and into the lungs. Once inside the lungs, the large tubes branch into many thousands of smaller tubes. Located at the very end of each individual branch is an air sac. These air sacs (alveoli) are very small, the total number in the lungs is estimated to be three hundred million. Tiny blood vessels (capillaries) surround the thin moist walls of each air sac. Because of these thin walls (1/50,000th of an inch) gases can diffuse back and forth into and out of the blood which flows constantly through the capillaries.

The chest cavity is surrounded by the ribs on the sides and separated from the abdominal cavity by a large flat sheet of muscle (diaphragm). Since the chest is a closed cavity with only one opening to the outside, any change in total volume ventilates the airspaces in the lungs. The chest size is altered by muscular action which raises and lowers the diaphragm and by contraction and relaxation of muscles between the ribs. Inspiration is the active phase of lung ventilation. Expiration is a passive phase resulting from the relaxation of chest muscles and diaphragm.

B. *Movement of Gases in the Respiratory System*

1. External Respiration

External respiration is the exchange of gases between the lungs and the surrounding atmosphere.

The pO_2 forces oxygen through the air sacs into the blood stream. The partial pressure of oxygen at ground level is about 21% of the total atmospheric pressure. When a breath of air is taken into the lungs you would expect the partial pressure of oxygen in the lungs to be about 159 mm Hg. However, the lungs also contain other gases which exert a fairly constant pressure (water vapor 47 mm Hg, and carbon dioxide 40 mm Hg). Therefore, these gases reduce the partial pressure of the oxygen entering the air sacs to about 102 mm Hg at ground level.

Due to the function of Graham's Law the high partial pressure of oxygen (102 mm Hg) now diffuses through the air sac wall and enters the blood stream. This will raise the partial pressure of oxygen in the blood from approximately 40 mm Hg to approximately 100 mm Hg. At the same time this is happening, the high pressure of carbon dioxide (47 mm Hg, CO_2) in the blood will cause some of the CO_2 to diffuse into the air sac where the CO_2 pressure is about 40 mm Hg.

2. Internal Respiration

Internal respiration is the exchange of gases between the blood and the body cells.

The same principle that applies to external respiration is found in this phase of respiration except that the partial pressures of the gases involved are now reversed. High partial pressure oxygen diffuses from the circulatory system into the body tissues and excess partial pressure of carbon dioxide diffuses out of the tissue into the blood stream for return to the heart and lungs some of which is to be exhaled from the body.

CIRCULATION

A. Function

The circulatory system is concerned with the transportation of blood throughout the body. Blood carries food and oxygen to the tissues and waste material, including carbon dioxide, from the tissues to the organs of excretion which include the lungs, kidneys, liver, sweat glands, and skin. Blood has additional functions of maintaining body heat and fluid balance.

B. Structure

The segments of the body which comprise the circulatory system are the heart, arteries, veins, and capillaries.

The heart is a pumping organ capable of forcing the blood through the vessels as tissue requirements dictate. The interior of the heart is divided into right and left halves and each half has two cavities.

The arteries are the vessels which normally carry oxygenated blood away from the heart. The elastic walls are muscular and strong, permitting the arteries to vary their carrying capacity. Small arteries connect large arteries to capillaries. The capillaries convey blood from the arteries to the veins. They are very small, thin walled, and usually form a network in the tissues in which the exchange of all gases between tissues and blood takes place.

The veins are the vessels which carry deoxygenated blood back to the heart. They have thinner walls and are less elastic than the corresponding sized arteries. When the blood enters the veins from the capillaries it is under low pressure. Therefore, some method is necessary to get the blood back to the heart, especially from the lower regions of the body. The muscles around the veins produce a milking action of the veins forcing the blood toward the heart. Backflow of blood is prevented primarily by valves located in the veins.

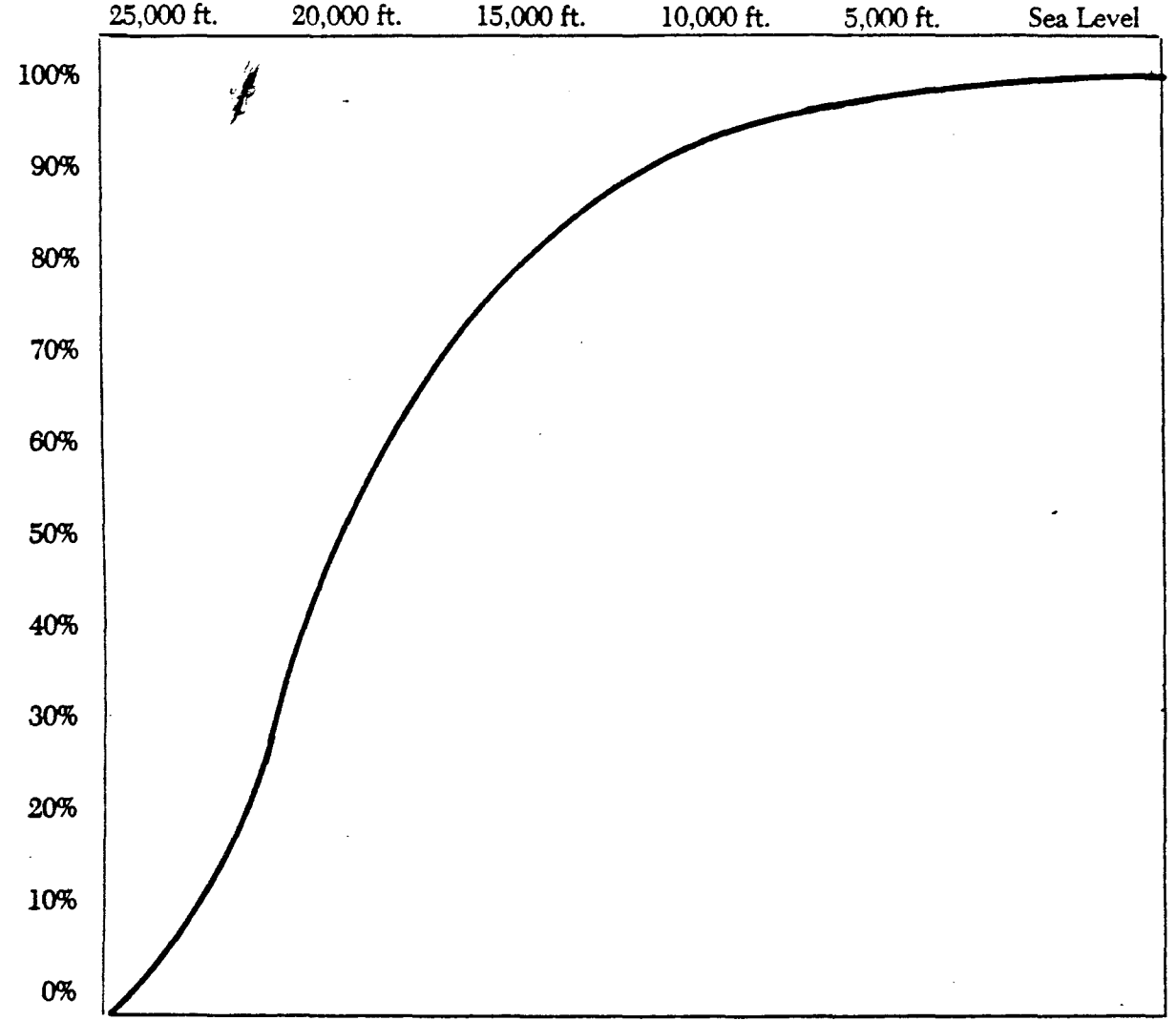
C. *Control of the Heart*

Carbon dioxide has an important effect on the heart. An increased concentration of CO_2 results in acceleration of the heart. In exercise there is not only a strong heart beat but also a faster rate because of the increased amount of carbon dioxide and need for oxygen in the blood. A decrease in carbon dioxide tends to slow the heart rate until the body again reaches its normal CO_2 quantity. Lack of oxygen causes an increase in the heart rate and has a definite effect upon the heart's contraction. It will increase the rate transporting a greater volume of blood to the lungs to obtain oxygen thus alleviating tissue deficiency. The heart has influence on blood pressure in that the faster the rate and the greater the force of the heart beat, the higher will be the arterial pressure. Because the heart is a pump, any change in its action will affect the pressure of the fluid pumped. There are other factors, chemical and otherwise, which affect the heart's actions and several of these will be covered in more detail later.

D. *Composition of the Blood*

Blood is made up of two parts, plasma and solids. Approximately 90% of the plasma is water, in which many substances are dissolved or suspended. The solid part of the blood is composed mainly of white and red blood cells. White blood cells are composed primarily of substances which act as antibodies to assist in fighting disease and infections in the body. The red blood cells are formed in the bone marrow and contain a colored substance called hemoglobin. Hemoglobin is an iron-containing compound. The iron in the hemoglobin molecule is responsible for the chemical affinity of hemoglobin for oxygen. About 95% of the oxygen is transported by hemoglobin and the remainder is in simple solution. It can readily be seen that a person who is anemic, or for any other reason does not have enough functioning red blood cells, will begin to suffer from lack of oxygen at relatively low altitudes. The blood of the average person contains about 15 grams of hemoglobin per 100 ml (milliliter). Each gram of hemoglobin is capable of combining with 1.34 ml of oxygen so the blood could contain 20 ml of oxygen per 100 ml of blood or 20 volumes percent if it were completely saturated. Normal arterial saturation is about 95-97 percent and the oxygen content is 19 volumes percent. The ability of hemoglobin to take up or release oxygen is not a linear function of the partial pressure of oxygen. However, the relationship is well defined and is usually shown in the form of the oxygen dissociation curve. Venous or return blood has a normal oxygen tension of 40 mm Hg, contains 14 volumes percent of oxygen, and is 65-75% saturated.

OXYGEN DISSOCIATION CURVE



1. Altitude. EPT decreases at higher altitudes.

2. Rate of Ascent. In general, the faster the rate, the shorter the EPT.

3. Physical Activity. Exercise decreases EPT considerably.

4. Day-to-Day Factors. Physical fitness, diet, rest, drugs, smoking, illness, and other factors may change your ability to tolerate hypoxia from day to day, therefore, changing your EPT.

METHODS TO COMBAT HYPOXIA

1. Hypoxic Hypoxia

A realization or recognition of your hypoxia symptoms should be followed by immediate use of supplemental oxygen if you are to combat hypoxic hypoxia successfully. Individuals who have difficulty recognizing their hypoxia symptoms must check their oxygen system periodically to assure an adequate supply and proper function. Frequency of checks will depend upon the cabin altitude of the aircraft. Refer to Effective Performance Time list on previous page for guidance on this matter. In most cases, recovery is rapid and the individual usually regains full faculties within fifteen seconds after oxygen is administered. The procedures employed in checking your oxygen equipment will be discussed in subsequent chapters. In aircraft where supplemental oxygen is not available, an emergency descent to altitudes below 10,000 feet generally will have the same effect as supplemental oxygen. Federal Aviation Regulation 91.32 states that the flight crew shall: use supplemental oxygen from above 12,5000 feet and up to 14,000 feet, if they remain in that altitude range longer than 30 minutes; use supplental oxygen above 14,000 feet regardless of time above 14,000 feet. For flight altitudes at and above 15,000 feet (regardless of time at or above 15K') each occupant (flightcrew and passengers) must be provided with supplemental oxygen (no requirement stating clearly that the passengers shall use the supplemental oxygen provided).

2. Histotoxic Hypoxia

This type hypoxia will not be corrected by the use of supplemental oxygen because the uptake of oxygen is impaired at the tissue level regardless of the oxygen saturation of the blood. The only solution to this type of hypoxia is abstinence from alcohol prior to flight. Remember it takes the body approximately 3 hours to eliminate 1 ounce of alcohol in the body. Only medications (drugs) prescribed by a flight surgeon or an aviation medical examiner may be taken prior to or during flight.

Hrs. since 1st drink . . .	1	2	3	4	5	6
SUBTRACT	.015%	.030%	.045%	.060%	.075%	.090%

THE REMAINDER IS AN ESTIMATE OF THE PERCENT OF ALCOHOL IN YOUR BLOOD.

Example – 160 lb. man, 8 drinks in 6 hours
.188% minus .090% = .098%

INTERPRETATION OF RESULTS

Percent of Blood-Alcohol	Intoxicated?	If You Are a Pilot
.001 to .050	You Are Not	Don't Fly
.050 to .100	You May Be	Don't Fly
.100 to .150	You Probably Are	Don't Fly
.150 and Above	YOU ARE	Don't Fly

Courtesy: Dr. Stanley R. Mohler, FAA – Chief, Aero-Medical Applications Div.

3. Hypemic Hypoxia

Contamination of the blood with gases (Carbon Monoxide,CO) other than oxygen is the source of this form of hypoxia. The correction is usually in locating and eliminating the source of the contaminating gases. In general aviation aircraft this means a careful check of the heater and exhaust manifold equipment prior to flight. Abstinence from smoking during and for a reasonable period prior to flight is also recommended. If hypoxia symptoms are recognized, and if supplemental oxygen and/or descent do not seem to relieve the symptoms, suspect carbon monoxide poisoning. Should this occur, ventilate the cabin continuously, land, get medical attention, and have aircraft checked as soon as possible.

4. Stagnant Hypoxia

Sudden incapacitation of the aircrew member is one of the chief concerns of the FAA medical standards. Any evidence of coronary/artery disease is grounds for immediate denial of medical certification.

Stresses of "G" forces encountered in aerobatic maneuvers are usually anticipated and so brief as not to cause prolonged pooling of blood.

Prolonged periods of pressure breathing required at the higher altitudes may result in stagnant hypoxia due to the distention of the alveoli sac caused by the oxygen pressure. The distention may partially restrict the flow of venous blood through the lungs resulting in a decreased outflow of oxygenated blood.

OXYGEN REQUIREMENTS

AIRCRAFT ALTITUDE	BAROM. PRESS. mmHg	H_2 PRESS. (BODY)	TRACH. PRESS. mmHg	% 0_2 INSP. AIR	TRACH. PRESS. $p0_2$	AVEOLAR $PC0_2$	AVEOLAR $p0_2$ mmHg	% 0_2 SAT. Hb.	% SUPPLEM. 0_2 REQUIR. INSP. AIR	TRACH. PRESS. $p0_2$	% 0_2 SAT. Hb.	CONSTANT FLOW 0_2 SYSTEM
Sea Level	760	47	713	.21	149	40	103	96%	(.21) 21%	149	96%	NTPD*
5,000 ft.	632	47	585	.21	122	38	78	94%	(.250 25%	149	96%	.5 LPM
10,000 ft.	523	47	476	.21	100	36	61	90%	(.31) 31%	149	96%	1.0 LPM
15,000 ft.	429	47	382	.21	80	33	46	70%	(.40) 40%	149	96%	1.5 LPM
20,000 ft.	349	47	302	.21	63	30	33	62%	(.49) 49%	149	96%	2.0 LPM
25,000 ft.	282	47	235	.21	49				(.63) 63%	149	96%	2.5 LPM
					Totally inadequate							Rebreather Type Equip. Not Recomm. Above 25,000
30,000 ft.	225	47	178	.21	37	40	103	96%	(.84) 84%	149	96%	3.0 LPM
35,000 ft.	179	47	132	.21	28	39	93	95%	(1.00) 100%	132	95%	3.5 LPM
40,000 ft.	141	47	94	.21	20	35	59	87%	(1.00) 100%	94	87%	4.0 LPM 4.1 LPM IS MAXIMUM FLOW MAXIMUM CERT. ALTITUDE OF 41,000 ft. Must be a Phase Sequential Type Mask.
PRESSURE DILUTER DEMAND OXYGEN EQUIPMENT												
40,000 ft.	141	47	94	.21	20							
				(x 1.00)	94	Inadequate						
				(+8mmHg)	102	35	59	87%	100% (1.00)	94	87%	
						36	66	92%	100% + Pres	102	92%	
42,000 ft.	128	47	81	.21	17							
				(x 1.00)	81	33	48	71%	100%	97		
				(+16mmHg)	97	36	61	90%	+ Pos. Pres	97	90%	
45,000 ft.	111	47	64	.21	13							
				(x 1.00)	64	30	34	62%	100%			
				(+33mmHg)	97	36	61	90%	+ Pos. Pres	97	90%	

HYPERVENTILATION

Respiratory controls of the body react to the amount of carbon dioxide (CO_2) found in the blood stream. In a physically relaxed state, the amount of carbon dioxide in the blood stimulates the centers and breathing rate is stabilized at about 12 to 16 breaths per minute. When physical activity occurs, the body cells use more oxygen and more carbon dioxide is produced. Excessive carbon dioxide enters the blood and subsequently the respiratory center responds to this, and breathing increases in depth and rate to remove the oversupply of carbon dioxide. Once the excess CO_2 is removed, the respiratory center causes the breathing rate to change back to normal.

The same process is involved when a maximum effort is made to hold the breath. While the breath is being held, the body cells continue to manufacture carbon dioxide which enters the blood. The amount in the blood finally becomes so great that, in spite of conscious efforts, the respiratory center overrides it and breathing is resumed.

SYMPTOMS AND TREATMENT OF CONDITIONS PRODUCED BY HYPERVENTILATION

Hyperventilation, or over breathing, is a disturbance of respiration that may occur in indivduals as a result of emotional tension or anxiety. Under conditions of emotional stress, fright, or pain, lung ventilation may increase, although the carbon dioxide output of the body cells remains at a resting level. As a result, carbon dioxide is washed out of the blood. This results in an excessive loss of carbon dioxide from the lungs, lowering the carbon dioxide tension below the normal 40 mm Hg. The most common symptoms are dizziness, hot and cold sensations, tingling of the hands, legs, and feet, tetany, nausea, sleepiness, and finally unconsciousness. Unconsciousness is due to the respiratory centers overriding mechanism to regain control of breathing by reducing blood flow to the brain (Cerebal Stagnant Hypoxia).

After becoming unconscious, the breathing rate will be exceedingly low until enough carbon dioxide is produced to stimulate the respiratory center. Hyperventilation also occurs as a result of the body's normal compensatory response to hypoxia. However, excessive breathing does little good in overcoming hypoxia.

Should symptoms occur which cannot definitely be identified as either hypoxia or hyperventilation, the following steps should be taken:

1. Check oxygen equipment immediately and put the regulator auto-mix lever on 100% oxygen (demand or pressure demand system). Continuous flow system – check oxygen supply and flow mechanism.

2. After three or four breaths of oxygen, the symptoms should improve markedly, if the condition experienced was hypoxia. (Recovery from hypoxia is extremely rapid.)

3. If the symptoms persist, consciously slow your breathing rate until symptoms clear and then resume breathing at a normal rate. Breathing can be slowed by breathing into a bag, or talking aloud.

Several aircraft accidents have been traced to probable hyperventilation. It is recommended that you induce hyperventilation by voluntarily breathing several deep breaths at an accelerated rate. You will begin to get some of the symptoms mentioned previously. Once you experience several of these symptoms, return to your normal rate of breathing. After you become familiar with the early warnings your body gives you, the likelihood of an accident caused by hyperventilation will be reduced. Caution: Do not hyperventilate while alone or in a standing position. You may fall and injure yourself.

DECOMPRESSION SICKNESS – DYSBARISMS

Increased or decreased pressure can cause other effects on the body. These undesirable effects are called decompression sickness or dysbarisms. They are caused by gas expanding or evolving within the body and may be divided into two groups:

A. Trapped Gas

During ascent and descent, free gas expands or contracts in certain body cavities. A person's inability to equalize with pressure changes may cause abdominal pain, toothache, or pain in ears and sinuses. These gases obey Boyle's Law.

B. Evolved Gas

This condition is produced by the low atmospheric pressure of high altitude, primarily above 30,000 feet. However, we should be aware of the fact that such problems have occured as low as 18,000 feet. Gases escaping from solution in the blood and other body tissues may be responsible for such conditions as bends, chokes, paresthesias, and central nervous system problems. These gases or bubbles consist mainly of nitrogen, with some oxygen, carbon dioxide, and water vapor. The formation of these bubbles is explained by Henry's Law.

TRAPPED GASES – CAUSE, EFFECTS, PREVENTION, AND TREATMENT

A. Ear Block — Barotitis Media

The ear is composed of three sections: the outer ear, the middle ear, and the inner ear. The outer ear includes the auditory canal, which ends at the eardrum. The eardrum is a thin membrane about 0.004 inches thick. The middle ear is located within the temporal bone of the skull and is separated from the outer ear by the eardrum. A short slit-like tube that connects the middle ear cavity and the back wall of the throat is called the eustachian tube. The inner ear is used for both hearing and certain equilibrium senses.

During ascent or descent, air must escape or be replenished through the eustachian tube to equalize the pressure in the middle ear cavity with that of the atmosphere. If you are unable to equalize this pressure because of a head cold or an infection in the tubes, pain and discomfort will result.

Normally, there is little difficulity equalizing pressure during descent because this can be accomplished by swallowing, yawning, or tensing the muscles of the throat at intervals. During sleep, the rate of swallowing slows down. For this reason it is advisable to awaken sleeping passengers prior to descent for the purpose of permitting them to ventilate their ears. Infants should be given a bottle or pacifier. Small children should avoid difficulity by chewing gum. If the preceding actions fail to equalize the pressure, you should valsalva. The valsalva procedure is done by closing the mouth, holding the nose and blowing. This will force air up the eustachian tube and into the middle ear. This is not a dangerous procedure and should not be delayed until the pressure in the ears becomes painful, otherwise it may be extremely difficult to *open* the eustachian tube. Painful ear block generally occurs when descent is made too rapidly. To relieve this pain, ascent to higher altitude is recommended. This should be followed by a

slower descent. During the second descent, close attention should be given to the prompt use of clearing techniques. Prudent use of nasal inhalants such as Benzedrex, Afrin, or Neosynephrin may also prove to be very helpful but should be used sparingly due to their compounding effect with hypoxia.

After a flight in which you use 100 percent oxygen, the valsalva procedure should be accomplished several times to ventilate the middle ear. This is recommended because the middle ear will be filled with pure oxygen, which is then gradually absorbed by the tissue of the middle ear. This causes a reduction in pressure which may become painful later in the day or night.

B. Sinus Block — Barosinusitis

The sinuses present a condition in flight similar to that of the middle ear. The sinuses are air filled, rigid, bony cavities lined with mucous membrane. They connect with the nasal cavity by means of one or more small openings. When these openings into the sinuses are normal, air passes out of and into these cavities without difficulty at any moderate rate of ascent or descent. If the openings of the sinuses are obstructed by the swelling of the mucous membrane lining, ready equalization of pressure becomes difficult. When the maxillary sinuses are affected, the pain will probably be felt on either side of the nose, in the cheek bones. Maxillary sinusitis may produce pain referred to the teeth of the upper jaw and may be mistaken for a toothache. When the frontal sinuses are affected, the pain will be located above the eyes and usually is quite severe. This type of sinus problem is the most common.

Equalization of pressure to relieve pain in the sinuses is best accomplished by use of the valsalva procedure, and/or inhalants previously mentioned in conjunction with ear block. Reversing the direction of pressure change as rapidly as possible may be necessary to clear severe sinus blocks.

C. Toothache — Barodontalgia

A toothache may occur at altitude when we fly. The pain may or may not become more severe as altitude is increased, but descent almost invariably brings relief. The toothache often disappears at the same altitude at which it was first observed on ascent.

Common sources of this difficulty are abscesses, mechanically imperfect fillings, inadequately filled root canals, and pulpitis.

Anyone who experiences a toothache at altitude should see a dentist without delay for examination and treatment. Maxillary sinus discomfort may be misinterpreted as a toothache.

D. Gastrointestinal Pain

Gastrointestinal pain is the discomfort caused by the expansion of gas within the digestive tract during ascent into the reduced pressure found at altitude. Fortunately, the symptoms are not serious in most individuals. In flights above 25,000 feet, enough distention may occur to produce severe pain.

The stomach, small and large intestines normally contain variable amounts of gas with pressure approximately equivalent to that of the ambient atmosphere. The chief sources of this gas are swallowed atmospheric air and, to a lesser extent, gas formed as a result of digestive processes.

As gases in the stomach and intestines expand during ascent, relief is ordinarily obtained by belching or by passing flatus.

Gas pains of even moderate severity may result in lowered blood pressure. Shock will be the eventual result if relief from distention is not obtained. Immediate descent from altitude should be made in order to obtain relief.

EVOLVED GASES – CAUSE, EFFECTS, PREVENTION, AND TREATMENT

Evolved gases are called decompression sicknesses and are caused by the same things that cause bends in caisson workers or deep sea divers. The formation of gas bubbles within the body resembles the release of bubbles in a carbonated beverage, such as soda pop or beer, when the cap is removed.

Nitrogen, always present in body fluids, comes out of solution and forms bubbles if the pressure on the body drops sufficiently. Fatty tissue contains many times more nitrogen than other tissue, making an overweight person more susceptible to evolved gas decompression sicknesses. The action of these bubbles on various tissues of the body are thought to cause various types of evolved gas sicknesses.

A. Bends

This is characterized by pain in and about the joints, and may be mild at the onset. Failure to descend from altitude following the onset of bends may result in deep, gnawing, and penetrating pain which becomes intolerable in severity. Ordinarily, the pain is progressive and becomes worse if ascent is made to a higher altitude. Severe pain can cause loss of muscular power of the extremity involved, and if the pain is allowed to continue, collapse may result. The pain may diffuse from the joint over the arm or leg as a whole, or over the entire area of a long bone. Joints, such as those of the knee and shoulder, are most frequently affected.

B. Chokes

Chokes, the common term for symptoms referable to the chest, probably are caused in part by blocking of the smaller pulmonary blood vessels by innumerable small bubbles. This, at first, may cause a deep burning sensation underneath the sternum. As the condition progresses, the pain may become more severe, may be stabbing in character, and may be markedly accentuated upon deep inhalation. It is necessary to take short breaths to avoid distress. There is an uncontrollable desire to cough, but the cough is ineffective and non-productive. Finally, there is a sensation of suffocation, breathing becomes progressively more shallow, and there may be cyanosis. Immediate descent is imperative when these symptoms occur. This condition, if allowed to progress, frequently results in collapse and unconsciousness. Fatigue and weakness, as well as soreness in the chest, may persist for several hours after descent to ground level.

C. Paresthesia

Another sign of decompression sickness is called paresthesia. Symptoms of paresthesia are tingling, itching, and cold and warm sensations. These symptoms are thought to be caused by the occurrence of bubbles locally or in the central nervous system where they may involve nerve tracts leading to the affected areas in the skin.

A mottled red rash may appear on the skin and, more rarely, a welt accompanied by a burning sensation. Skin manifestations of paresthesia are not in themselves incapacitating or critical.

D. Central Nervous System Disturbances (CNS)

Central nervous system disturbances include visual disturbances which are similar to the symptoms of hypoxia or the pulling of positive "G's." Lines or spots before your eyes may be seen. Some parts of the field of vision may disappear or blur. Dull and persistent headaches are commonly associated with visual symptoms. Other comparatively rare effects are partial paralysis, sensory disturbances, and aphasia, all of which are usually transient. These nerve manifestations occur primarily during or after descent and are generally relieved upon reaching ground level although in rare cases they may persist for a long period of time.

E. Complications

One of the outcomes of dysbarisms may be a case of shock. It consists of faintness, dizziness, nausea, or even loss of consciousness, accompanied by pallor and sweating. This reaction is your body's protest against a disturbance in the circulation of your blood. This form of shock may be experienced during the time you are at high altitude. However, symptoms occasionally persist even after you return to the ground or may reappear several hours after you land. This is why you should treat decompression sickness with respect. The shock reaction makes it dangerous. Prompt recognition of symptoms and treatment (usually by recompression) results in an almost 100% cure.

If you have symptoms of dysbarism or decompression sickness, remain quiet, keep the affected area immobile, and descend. It may prove to be crippling and the resultant lack of efficiency when you have a severe attack can be extremely dangerous. Pain is usually relieved after descent. Statistically speaking, the hazard is small. You should minimize this hazard by contacting the closest aviation medical examiner (AME) in the event of an occurence of this type. He will know how to treat the problem, and he will know where the closest recompression chamber is located.

SCUBA DIVING AND FLYING

Many flying personnel are also diving enthusiasts. SCUBA (Self Contained Underwater Breathing Apparatus) is an exhilarating hobby; however, recent research has shown that diving and flying shortly thereafter can have marked effects on an individual. The SCUBA diver uses compressed air in the breathing tanks. When dives are made to a depth of approximately 30 feet, the body will absorb about twice as much nitrogen as it had at the surface. Generally, the diver has no problems when returning to the surface, provided the recommended stops as prescribed in the diving manual have been followed. However, the problem is compounded if that individual decides to fly an aircraft immediately after diving. A person flying an aircraft to altitude in excess of 8,000 feet following SCUBA diving is in the same predicament the non-diver is when flying at 40,000 feet unpressurized. In other words, we subject our body to altitudes where evolved gases can and do occur.

The recommended waiting time before flight to cabin pressure altitudes of 8,000 feet is at least 4 hours after diving which has not required controlled ascent (non-decompression stop diving), and at least 24 hours after diving which has required controlled ascent (decompression stop diving). The waiting time before flight to cabin pressure altitudes above 8,000 feet should be at least 24 hours after any SCUBA diving.

NOTE:

If you or a member of your flight experience evolved gas type decompression sickness such as bends, CNS, or chokes, you should contact the School of Aero-Space Medicine, Brooks AFB, Texas, at once. The USAF School of Aerospace Medicine maintains a 24-hour "hot line" for consultation in suspected evolved gas disorders. Their phone number is (512) 536-3278. Delays may cause the situation to worsen and collapse may occur. There are numerous treatment facilities over the world where these manifestations can be treated by skilled personnel and return to normal will be expedited if prompt action is taken. The local flight surgeon or aviation medical examiner will have current information about these treatment facilities.

CABIN PRESSURIZATION AND DECOMPRESSION

Cabin pressurization is the maintenance of a cabin altitude lower than the actual flight altitude. This is accomplished by compressing air in the aircraft cabin. Pressurized aircraft reduce the physiological problems at altitude and increase the effectiveness and comfort of the aircrew member and passengers. However, when pressurized to an altitude lower than actual flight, the possibility of sudden loss of this pressurization exists. If you are prepared for this eventuality, the effects will be minor.

ADVANTAGES OF CABIN PRESSURIZATION SYSTEMS

Flights to altitude may be made without the use of supplemental oxygen. However, the oxygen ceiling, in relation to cabin altitude, must still be observed. Additional precautions should be taken to see that oxygen equipment is readily available in the event of a sudden loss of pressure.

Decompression sicknesses are prevented or made less serious because the body is not exposed to extremely low barometric pressure.

Heat and ventilation can be controlled more satisfactorily.

Cabin pressurization is especially advantageous in transport aircraft on long flights at medium altitudes. Eliminating the need for an oxygen mask contributes to the comfort of all crew members and increases their efficiency by enabling them to move more freely about the aircraft.

Pressurization of most aircraft is accomplished by maintaining a sea level environment up to engineering limitations. Once a maximum pressure differential is reached, a constant differential is maintained. Cabin pressure altitudes are then limited by the actual flight altitude of the aircraft.

PHYSIOLOGICAL EFFECTS OF DECOMPRESSION

Decompression is defined as the inability of the aircraft's pressurization system to maintain its designed pressure schedule. This can be caused by a malfunction in the pressurization system or structural damage to the aircraft. Physiologically, decompressions fall into two categories:

1. *Explosive Decompression*

   Explosive decompression is defined as a change in cabin pressure faster than the lungs can decompress. Therefore, it is possible that lung damage may occur. Normally, the time required to release air from the lungs where no restrictions exist, such as masks, etc., is 0.2 seconds. Most authorities consider any decompression which occurs in less than 0.5 seconds as explosive and potentially dangerous.

2. *Rapid Decompression*

   Rapid decompression is defined as a change in cabin pressure where the lungs can decompress faster than the cabin. Therefore, there is no likelihood of lung damage.

Experiments have been conducted in altitude chambers where normal, healthy persons were decompressed from 5,000 feet to 35,000 feet in 0.7 seconds. There were no ill effects from these decompressions. Almost all large pressurized aircraft decompress at a relatively slow rate (10 seconds or more). Thus, anyone exposed will, in all probability, experience a rapid decompression rather than an explosive decompression. This is not necessarily true for small volume pressurized aircraft. The use of safety belts becomes much more critical when seated near exits or windows.

During a decompression there may be noise, and for a split second one may feel dazed. The cabin air will fill with fog, dust, or flying debris. Fog occurs due to the rapid drop in temperature and the change of relative humidity. Normally, the ears clear automatically. Belching or passage of intestinal gas may occur. Air will rush from the mouth and nose due to the escape of air from the lungs, and may be noticed by some individuals.

The primary danger of decompression is hypoxia. Unless proper utilization of oxygen equipment is made when the aircraft is flying above 30,000 feet, unconsciousness will occur in a very short time. The period of useful consciousness is considerably shortened when a person is subjected to a rapid decompression. This is due to the rapid reduction of pressure on the body. Thus, oxygen in the lungs is exhaled rapidly. This in effect reduces the partial pressure of oxygen in the blood and thus may reduce the effective performance time by 1/3 to 1/2 its normal time. It is for this reason the oxygen mask should be worn on the face when flying at very high altitudes, regardless of the cabin pressure altitude.

Another potential hazard of high altitude decompressions is the possibility of evolved gas decompression sickness. Exposure to windblast and extremely cold temperatures are other hazards one might have to face.

Rapid descent from altitude is indicated if these problems are to be minimized. Automatic visual and aural warning systems should be included in the equipment of all pressurized aircraft so that slow decompressions will not occur and overwhelm the occupants before being detected.

PRINCIPLES AND PROBLEMS OF VISION

Of the various human sensory equipment, none is more important for flying than the eyes. Good vision, in spite of radar, GCA, and other electronic devices, remains of primary importance in the judgment of distance, depth, and position in flight, and the reading of instruments and maps.

ANATOMY AND PHYSIOLOGY OF THE EYE

The eye is an organic camera with an almost inexhaustible supply of film. Both the eye and a camera have a shutter, diaphragm, lens, method of focusing, and film, all arranged in a light-tight container.

Objects that reflect or emit light project an image of themselves on the retina of the eye. The retina is an interior coating of the eye, located around the sides and to the back. It contains the light-sensitive cells known as rods and cones. These connect to the cells of the optic nerve which transmit messages directly to the brain.

The greatest concentration of cones is in the center portion of the retina in a slight depression known as the fovea. There are a great many cones and they excite the interconnecting neurons on a one to one basis. This is the reason why the cones are able to detect fine detail. The cones are also color sensitive.

The rods are found primarily in the peripheral areas of the eye with a zone between this area and the fovea occupied by both rods and cones. Although there are many rods in the peripheral areas they are less numerous than the cones. The rods also are not sensitive to color and do not detect light in wave lengths greater than red. The rods are very valuable as they are much more sensitive to light than cones. Their function is based upon the buildup of a chemical known as visual purple.

REACTIONS TO ILLUMINATION LEVELS AND TECHNIQUES OF SEEING

Rods are activated by an amount of light equal to starlight found on a clear night. Cones on the other hand require at least half the intensity of moonlight in order to function. Four factors which influence visual acuity are: size and detail, brightness, contrast, illumination and exposure time.

We have one blind spot where the optic nerve enters the eye. A person does not normally notice this as the visual fields of both eyes overlap and, in effect, eliminate this optic disc.

At below one-half moon illumination there are two blind spots; the optic disc and the fovea which contains only daylight receptors. An object cannot be seen at night under low light conditions by looking directly at it. By looking 4° to 12° away from an object, it can be seen since the image will then fall on the rods. This technique is called off-center vision and can be developed with practice.

Rods are not always instantly ready for use. During daylight hours, a lack of visual purple makes these cells partly inoperative. Generally, 30 minutes is required to properly night adapt your eyes. Wear sunglasses in the daytime and do not expose yourself to bright light if you want to see better on night flights.

INSTRUMENT LIGHTING

A number of visual problems arise in night lighting of instruments in the cockpit. The purpose of such lighting is to make the instruments easily readable. Because of other considerations, illumination needed for optimum readability is not always practical. We shall discuss three types of instrument lights.

A. Ultraviolet Flood Lighting of Fluorescent Instrument Markings

The instruments are marked with a paint which is fluoresced with a bluish-green color. This is probably the most unsatisfactory color for preservation of dark adaptation. The ultra-violet lighting system illuminates only the fluorescent instrument markings. Everything else is black. The advantage of this system is the readability of instrument. The disadvantages of this system are: difficulity in keeping the instruments in focus, stray untraviolet light may reach the eyes and cause fogging of vision, irritation of the eyes and loss of dark adaptation.

B. Red Lights

Both indirect lighting and flood lighting can be provided. The advantage of the indirect system is that light is confined to the instrument face and does not flood the cockpit. The disadvantages of this system are: difficulty in obtaining uniform light distribution over all parts of the instrument, no illumination of knobs and switches located on the panel, and finally, obstructing portions of the dials. Strip lighting will help in identifying knobs and switches.

Flood lighting systems provide red light for the entire instrument panel. These lights are mounted in the cockpit and the reflection from the panel is directed toward the floor. The advantage of this system is that all knobs, switches, and instruments are illuminated. The disadvantages are that large amounts of light are scattered within the cockpit and the difficulty in finding a good place to mount the fixture. Also, although red light is the most desirable for preservation of dark adaptation, it disturbs our normal color relationship. All color differences are lost. The coloration shown on maps is useless and information printed in red become unreadable.

C. White Lights

The latest type of lighting being used in aircraft is low density white light. The advantage of this system is that a more normal instrument picture is presented to the pilot. Readability of instruments is excellent; however, the dark adaptation is somewhat impaired. Regular white light, such as a flashlight, will destroy night adaptation and should be used sparingly. The low density white light can be regulated so that instruments are easily read and the tendency towards disorientation is eliminated. The concensus of pilots flying today is that the white lights are the best.

ADVERSE WEATHER LIGHTING

The common complaint pilots have about anticollision lights is annoyance, with strobe lights being the most irritating. There can be no doubt as to their effectiveness; however, when flying near or through thunderstorms it is recommended that the anticollision light be turned off if it interferes with the pilots orientation.

THUNDERSTORMS

Lightning flashes encountered when flying near or through thunderstorms create a special lighting problem. To make continued reading of instruments possible under such conditions a higher than normal instrument lighting intensity is required. This can be obtained easier by using white light rather than red lights.

NOISE AND THE GENERAL AVIATION PILOT

CAUSES OF NOISE

Cockpit noise in light aircraft has been around so long that we all seem to accept it as an inevitable part of flying, like carburetor ice and turbulence.

If noise were merely a nuisance, if all it did was to briefly interrupt our routines, perhaps we could learn to tolerate it.

Of course, it is to be expected that some hearing loss will be experienced by every person during an average lifetime, either through aging or through environmental noise. But for those who fly for either business or pleasure, this problem may occur sooner, unless personal protection is used. Most long-time pilots have a mild loss of hearing. Many pilots report unusual amounts of fatigue after flights in particularly noisy aircraft. Many pilots have temporary losses of hearing sensitivity after flights; and many pilots have difficulty understanding transmissions from the ground.

Noise exposure has harmful effects that are cumulative and so produces an adverse effect on the listener, both as sound intensity and length of time are increased. The Occupational Safety and Health Act places strict restrictions on the amount of noise a worker will be subjected to during an 8 hour work shift. No such restrictions, however, are imposed on the general aviation pilot.

At the same time that general aviation aircraft manufacturers have developed more powerful engines, they have not given the occupants better noise protection and control, so that today's aircraft are more powerful, yet some are noisier than ever. Still, the levels of sound associated with powered flight are high enough for general aviation pilots to be concerned about participating in continuous operations. In basic terms, propeller-driven aircraft are noisy, with helicopters and planes with open cockpits being the noisiest.

Recent tests conducted by the Civil Aeromedical Institute (CAMI) demonstrated that all propeller-driven, fixed-wing aircraft and all helicopters are potential sources of damaging noise intensities for the flight crew. The protection afforded by the cockpit is not enough to keep most active pilots from being overexposed. Part of the problem is explained by another CAMI study that showed the fatiguing effects of noise to be increased in a listener, such as a pilot, who is mentally active rather than resting. Since a pilot-in-command cannot rest safely during flight, the noise may affect him more than, for instance, a passenger who is relaxing.

The amount of hearing problem likely to be engendered by a given amount of noise is a function of the susceptibility of the listener, and of the amount of time exposed. A comprehensive investigation would probably show that the most exposed people are aerial-applicators, followed by flight instructors, helicopter pilots, business and other commercial pilots, flight attendants, airline pilots, and flight engineers.

The primary sources of noise in a light aircraft are:

1. Squeaks and rattles.
2. Exhaust.
3. Propellers.
4. Ventilation system.
5. Air turbulence around fuselage.

1. Squeaks and rattles – Squeaks and rattles may be remedied by welding rather than riveting. However, there still would not be much difference in sound intensity after it was done.

2. Exhaust noise – Exhaust noises are now being muffled, but mufflers add weight to the aircraft.

3. Propellers – Propeller-driven aircraft predominately produce low-frequency noise with the most intense portion of the noise at the pilot's position. The higher speed of propeller rotation will result in a slight upward shift in the predominant frequency range. The prop noise ranges from 89 to 113 dB with the average being about 106 dB.

4. Ventilation systems – In light aircraft tested, ventilation noise turned out to be as noisy as all the other items put together. However, if manufacturers quieted the ventilation systems, it would only decrease the sound by 3 decibles (dB).

5. Air turbulence around fuselage – Another source of noise in aircraft is air flow or air turbulence around the fuselage. This is vividly demonstrated when an engine quits and the aviator still hears noise around the aircraft.

Turbulence noise may be reduced by streamlining of the aircraft. You can also eliminte a lot of the noise by acoustical treatment of the walls. Rugs, curtains, and acoustical tiles are ***NOT*** good for eliminating noise as they only cut down on echo. It is not effective in reducing transmitted sound. The only solution to the problem is to increase the mass with something like lead blankets. Light aircraft could not be treated in this manner as it would not be conducive to flight.

Some type of honeycomb structure might do the same, but it would change the aerodynamic property of the aircraft so it wouldn't fly.

EXPOSURE -vs- HEARING LOSS

Different people have different responses to noise. Even for one person, sensitivity may be different in each ear. Because of the individual differences in sensitivity, we have to talk about the average case.

In light twin engine planes, the average person may develop a significant hearing loss by flying more than about 8 hours a week. If you fly 8 hours a week for 10 years in a light twin, you can expect to have enough hearing loss to begin having trouble understanding speech.

If you fly more than 5 hours a week in a light single-engine plane, in 10 years you should expect to have trouble understanding speech.

In open cockpit planes, leaning the head over the side 30 seconds a week, over a 10-year period could produce a significant hearing loss. The Civil Aeromedical Institute in Oklahoma City has never tested a crop-duster pilot who did *not* have a hearing loss and some of those tested had only been exposed for 1½ years. They fly as many days a year as they can and often are in the air 14 hours a day.

One of the main problems with hearing loss is that it generally occurs very slowly over a period of time. The loss is so slow that aviators are not aware of the problem until speech intelligibility becomes very difficult. By the time this problem is noticed, a permanent hearing loss has probably occurred.

Therefore, personal protection early in a person's flying career is the only practical answer to hearing loss in aviation.

Another problem associated with noise is speech intelligibility. Cockpit noise is particularly detrimental to the understanding of speech because the engine and exhaust noises are at a maximum in the same frequency range where speech has its maximum energy. Pilots often report that although the volume or gain control on the receiver is turned all the way up, tower transmissions are garbled or covered up, masked, by the engine noise. Tests at CAMI showed that, under full power takeoff conditions, the intelligibility of the tower controller can sometimes fall from 100 percent to zero.

EAR PLUGS

Problems concerning noise in flight are fairly easy to solve. Earplugs or similar hearing protection will prevent almost all the problems heretofore mentioned. Earplugs or earcaps are devices that are inserted in or pressed against the external ear canal to reduce the effect of ambient sound on the auditory system. Because every ear is unique in shape and size, several approaches to solving the problem of designing adequate earplugs have been taken. As a result, commercially available earplugs may be:

1. Premolded
2. Moldable
3. Custom molded

1. Premolded earplugs – includes varieties that are vented and varieties in which a headband presses a cap across the open end of the canal as well as plugs that insert more or less deeply to block the canal opening completely.

2. Moldable earplugs – includes impregnated and non-impregnated porous materials, expandable foams, and putty-like substances. Plain, unimpregnated cotton is useless as a hearing protector, so a commercial earplug should be used.

3. Custom molded earplugs – are either made by the manufacturer from an ear impression or the ear impression itself becomes the earplug. Since every ear is unique in shape and size, one might assume that a standard, off-the-shelf earplug would not protect as well as a personalized or custom-fitted earplug. Intuition says that a custom-fitted plug should provide a better, more precise seal within the ear canal, should do so through most the the length of the inserted segment, and should be more comfortable and easier to insert. Although they are generally the most expensive, if all the assumptions about comfort, acoustic seal, and ease of use are true, then personalized earplugs would be a bargain despite their higher costs. Tests conducted at CAMI indicated that many of the specially fitted plugs are not great bargains at all; in general, they are more expensive, less comfortable, and less effective than premolded or moldable types. Custom molded earplugs may be the answer for those who will not wear earplugs unless they can be convinced that the expense makes them the best available and as such a status symbol.

All earplugs are worthless in your pocket or in the glove compartment. Under any circumstances, a poorly fitted noise-protection device is worse than none because it gives the user a false sense of security. Similarly, loose-fitting earplugs or headsets are not at all helpful. An earplug that is "so comfortable that I can hardly feel it" is not doing any good. It should not be uncomfortable, but it must be snug. The usual technique of wearing only one earphone is not recommended to the pilot because of possible damage to the unprotected ear.

A very common question concerning the wearing of earplugs is "How do you hear what you need to hear if you wear earplugs?" Hearing protectors work much the same way sunglasses do. If you walk into a dark room with sunglasses on, you bump into things. If you wear them when there is a lot of

sunlight, you will be able to see better than you did without them. The same analogy applies to earplugs. If you wear them where it is quiet, you will not hear much of anything. If you wear them where it is noisy (say a car running at 40 mph with the windows open), the plugs would be very beneficial. You will hear everything you need to hear and you'll hear it better. Speech intelligibility is improved tremendously by using earplugs in the cockpit.

When you wear earplugs, people will have trouble hearing you! Without background noise to compete against, you will have a tendency to talk softer and lower and people will have difficulity hearing you. The background noise is still present to everyone not wearing earplugs, but it will not be as noticeable to you. You probably won't remember this statement until you begin using earplugs and the tower or your flying buddy will say: "Say again."

The only people who will not hear speech better in noise while wearing earplugs are those who already have a very severe high-tone hearing loss. These are mostly people who are particularly susceptible to the deafening effects of noise, and who therefore need to be especially careful to protect themselves from further exposures to noise. Wearing of earplugs for these people will help protect them against further damage.

Earplugs can be bought for as little as ten cents a pair for wax-impregnated cotton or for up to $30 a pair for custom made ear inserts. The most common varieties range between $.75 and a $1.50 a pair and can be used countless times.

RECOMMENDATIONS CONCERNING EARPLUGS

1. Use earplugs whenever flying.

2. Use earplugs while wearing earphones.

3. Be certain that earplugs fit snugly and that headsets are adjusted to cover both ears tightly.

4. Use real earplugs; plain absorbent cotton does not work.

5. Talk a little louder to the passengers and into the microphone; remember that the noise is still there and that one must speak loudly enough to overcome its effects.

6. Check the fit of the ear protector by pressing earplugs with the forefinger or earmuffs with the palms. If fitted correctly, there will be no change in the amount of sound that is still getting through. If the noise decreases or gets louder, the earplugs are not the right size or improperly fitted. Too much pressure applied manually on the ears may distort the earplugs and the noise becomes louder. Earplugs present no problem when descending and when cabin pressure increases.

7. Demonstrate the effectiveness of the earplugs to yourself by wearing only one plug during a flight of an hour or more. Immediately after shutting down the engine, remove the plug. The difference in hearing in the two ears will almost make it seems as if the ear that was unprotected during the flight is now quite deaf. Of course, it is not. It is only less sensitive because of the exposure to noise and will recover with time. *Repeated* exposure can produce permanent damage to one's hearing.

8. Less fatigue should be noted after a cross-country flight. With less noise, one can fly more comfortable, further, and safer. Try some earplugs, you'll be amazed at the results!

For additional information, the aviator is referred to:

"Cockpict Noise Intensity: Fifteen Single-Engine Light Aircraft" by J. V. Tobias, FAA Office of Aviation Medicine Report AM-68-21, and "Cockpit Noise Intensity: Eleven Twin-Engine Light Aircraft," same author OAM Report 68-25. Also Advisory Circular AC-91-35 is very helpful.

SPATIAL DISORIENTATION
SENSORY ILLUSION OF FLIGHT
VERTIGO

The terms listed in our title are often used interchangeably even though their exact meanings differ somewhat.

Sensory Illusion – A false or misinterpreted sensory impression; a false interpretation of a real sensory image.

Vertigo – A hallucination of movement. A sensation of rotary motion of the external world or of the individual.

Disorientation – Loss of proper bearings, state of mental confusion as to position, location, or movement.

Sensory apparatus in various parts of the body provides the brain with information about your position in relation to your environment. The eyes, inner ears, and muscle sense, in other words, literally tell you *which end is up*. In flying, many conditions you encounter can cause conflicts or illusions in these sensory functions. Cockpit confusion might be another term for disorientation since the information from your senses and from your flight instruments may be contradictory.

To understand the functions of the organs of equilibrium and how interpretations of these senses may lead to sensory illusions and spatial disorientation is a complex undertaking, but rewarding.

SENSORY SYSTEMS INVOLVED IN EQUILIBRIUM

The sensory organs of the body associated primarily with maintaining body equilibrium are the eyes, the semicircular canals of the inner ear, and the perceptors of the skeletal muscles.

A. The Eyes

The eye acts as the receptor organ for visual sensations. These sensations establish impulses in the rods and cones and the impluses travel the optic nerve to the brain for interpretation. The eye is very reliable for orientation provided adequate reference points are available. When flying, however, you are at a disadvantage when trying to interpret purely visual clues. Objects seen from the air often look quite different from objects seen from the ground. Also, you are used to having the ground extend to the horizon. In the air you lack the visual clues that a continuous background provides for recognizing objects and deciding their size and distance. A very common mistake is in interpreting the lights that you see at night. Pilots sometime become confused about the relation between their own motion and the false motion of fixed lights on the ground. Thus, a pilot may decide that a fixed light on the ground is another airplane traveling in the opposite direction.

If you cannot see the horizon, you may mistakenly choose some other line as a reference and, for example, may fly parallel to a tilted cloud bank instead of the ground. Consider what happens when no clouds are present and the horizon is obscured by haze or adverse lighting conditions. In such a situation

you are apt to be completely without reference, which amounts to *flying blind*. In Alaska and other similar areas, this problem is particularly severe due to haze and light reflected from snow covered ground. Under such conditions, sensory illusions in flight are only part of the problem; a noticeable loss of depth perception increases the hazard.

All these illusions are mistakes in interpretation caused by inadequate information on which to establish a reference. Your eyes are reporting correctly to your brain, but they don't give it enough information to work on.

This situation is worse at night than during the day, for your eyes are furnishing less information. Under such conditions your eyes can send false messages to the brain.

Have you ever been stopped at a traffic signal and then had another automobile pull along side? Although you were stopped, did you have the illusion that you were backing up slowly and found that you were indeed stopped and the other car was moving forward very slowly? This is only one simple illustration of sensory illusions. There are numerous others that can and do occur while flying aircraft in both VFR and IFR conditions.

B. Perception of the Skeletal Muscles

The tension of the various muscles in your body assist you in determining your position within the frame of reference, as well as any motion with respect to this reference. Compared to your eyes and equilibrium senses, however, the muscles play a very small part in determining such things.

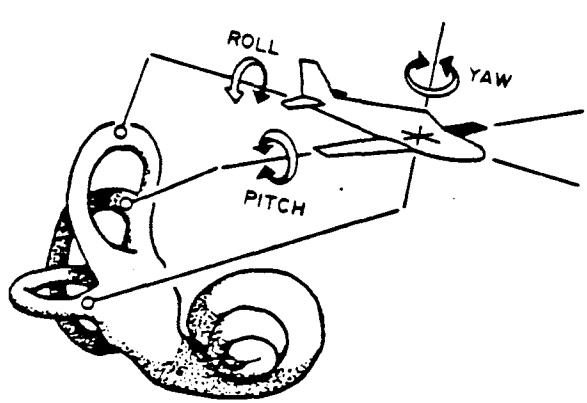
However, no matter how small a part the muscle sense plays in determining direction in the air it can give some indication of your body position in the seat in the aircraft and the sensation that gravity is being applied along a line from the earth passing vertically through the seat. This feeling can occur regardless of the aircraft's reference position to the earth. This means that even though muscle sense indicates to the pilot the aircraft is flying a straight and level course, the aircraft may actually be in a coordinated turn.

Trust your instruments.

Letting your senses take over and putting your trust in them may or may not cause an illusion. You may have absolutely no idea what the attitude of the aircraft is or its position in reference to the ground.

C. The Inner Ear

The inner ear consists of an auditory and non-auditory portion. The latter, primarily associated with equilibrium, contains the three semicircular canals. The semicircular canals are filled with fluid and are located at approximately right angles to each other.



One end of each canal is enlarged and in this area is found a mound of sensory hair cells. Angular movement or rotation of the body tends to move the fluid of the semicircular canal, thereby causing displacement of the hair cells and impulses are transmitted to the brain to be interpreted as motion about a known axis. The hairs which project into the fluid are extremely fine and light and bend with the fluids movement. They transmit messages to the brain telling which way they are displaced, and the brain figures out the direction of rotation. Since each canal lies in a different plane, the semicircular canals can report on rotation in three dimensions. This system works fine for short turns. But, if the turn continues at a constant rate for any length of time, the motion of the fluid in the canals catches up with the canal walls, the hairs are no longer bent, and the brain receives the completely false impression that the turning has stopped. Thus, after several seconds of motion at a constant rate it is quite impossible for the semicircular canals to detect a turn, especially if it is a gentle one.

Now, let's see what happens when you are turning rapidly and suddenly stop. The canal fluids continue to move and the hairs are bent in the opposite direction (due to the negative acceleration), indicating that you are turning in the opposite direction. As a result, you try to counteract this imaginary motion by turning in the original direction again.

If you are rotated rapidly to the right (positive acceleration) and are then suddenly stopped (negative accleration), you will get a senstion of turning to the left. In addition, you will see the lights move as if you were turning to the left if your vision is limited to say a single set of lights like those of another aircraft. The motion of the fluid in the semicircular canals creates impulses that affect your eyes. Even after rotation has been stopped, your eyes continue to make short "sweeps" as if you were rotating. This condition is called nystagmus.

To point out the danager existing with this type of illusion, just consider what happens in an airplane. You are flying at night and only a few lights are visible. You decide to make a turn to the right and as you begin to turn, your canals report (correctly) that you are rotating to the right. Now, especially if the turn is slow and the rate of turn becomes constant, the fluid in the canals will catch up with the motion of your body. When this happens, the hairs in the semi-circular canals are no longer affected and you get the erroneous impression that you have stopped turning.

At this point, you can get yourself into a lot of trouble with very little effort. If you believe that your canals are right, you will decide that you have stopped turning. Since you want to go the right, you are likely to swing right again and get into a much tighter turn.

Let's assume now that you don't receive the impression that you have stopped turning, or if you do, you are able to dismiss it as a result of checking your instruments. Finally, you decide to stop turning. The airplane and you stop rotating, but not the fluid in the canals. It continues and creates the impression that you are now turning to the left. To correct this, you make the error of beginning to turn to the right again. At this rate, you'll keep on turning until something drastic happens.

While turning under instrument conditions, if you should put your head down in a cockpit, a sudden stimulation of another semicircular canal just brought into this plane of motion may give you the sensation of an abrupt increase in the turn.

If an aircraft tips to one side quickly enough for the pilot to detect it, but is restored to straight and level flight so slowly that it is not detected, the pilot may believe that the aircraft is still tilted to that side. The sensation will be so convincing that the pilot will actually lean away from the supposed tilting even though the instruments may tell the pilot that the aircraft is in straight and level flight. Because of this tendency to lean from the side of the original tilt, this phenomenon, which is a common one during instrument flight, has come to be known as the *leans*. The opposite may happen. Your aircraft may tilt to one side and be undetected. If you recover from this tilted attitude abruptly, you will experience a roll to the opposite side even though what you have done is to level your aircraft. You might interpret

the situation as a wing low on the opposite side and may lean in order to align yourself with the supposed axis of the airplane.

Other sensing mechanisms located in the inner ear are called otolith organs. They are affected by straight line or linear accelerations. They register various "G" forces as they are applied to the body but are not able to give accurate body orientation information to the brain.

FLIGHT FACTORS CONTRIBUTING TO SENSORY ILLUSIONS

1. Changes in acceleration and deceleration.
2. Cloud layers.
3. Low level flight over water.
4. Frequent transfer from instruments to visual flight conditions. One must use either VFR or IFR, not oscillate from one type to the other.
5. Unperceived changes in flight attitude and/or altitude.

FACTORS CONTRIBUTING TO VISUAL ILLUSIONS

1. Optical characteristics of windshields.
2. Rain on windshields.
3. Effects of fog, haze, dust, and other such factors on depth perception.
4. Angle of glide slope.
   a. Steep angle would make apron appear to be farther away and runway longer.
   b. Shallow angle would make apron appear to be nearer and runway shorter.
5. Width of runway.

   Wide runways give the pilot feeling of being nearer to ground than actual position.
6. Variations in runway lighting systems.
   a. One row of lights brighter than other.
   b. Lights on one side of runway.
   c. Approach and runway light confusion.
   d. Width of runway lights.
7. Runway slope.
   a. Upslope – tendency to land short.
   b. Downslope – tendency to land long.

8. Terrain slope.

   a. Upslope – tendency to land long.

   b. Downslope – tendency to land short.

9. Landing over water.

   Tendency to land short due to lack of visual cues.

10. Autokinetic phenomenon.

    a. Apparent motion of a fixed light in the dark.

    b. Motion may be in any direction after a latent period of 6 to 12 seconds.

    c. This effect may be reduced by avoidance of staring at lights in the dark for prolonged periods and by reliance upon instruments.

WHAT YOU CAN DO TO BEAT SENSORY ILLUSIONS

From all this you can see that, individually treated, each type of illusion can cause a great deal of trouble. Since this is precisely what may happen if you are not careful, let's see what you can do to beat these illusions.

First of all, you probably appreciate the fact that sensory illusions or vertigo are problems that usually show up under conditions of poor visibility. Whenever the visibility is poor enough to prevent you from checking your equilibrium senses with your eyes, your equilibrium system is undependable. That is why your aircraft is provided with an artificial equilibrium system for indicating bank angle, aircraft altitude, rate of climb and so forth. This system is much more reliable than anything your are equipped with, but it's not easy to use, primarily because you weren't born with it. All your life on the ground you have been navigating by your eyes, and you are accustomed to doing what they tell you to do. Now, when you fly by instruments, you are told to ignore your natural senses and put your faith in dials and indicators.

There are several points to remember about instrument flying. The first is that you can learn to do it, but you have to use your head. Flying by instruments is a skill that can be highly developed. You have to read and interpret the instruments and act accordingly. At the same time, you must have confidence in the instruments and ignore any other signals your body gives you.

This procedure usually slows you down a bit. Tests show that flyers interpret the actual horizon about one-fifth of a second faster than they interpret instruments. Furthermore, pilots make a recovery from a dive about one and a half seconds faster under visual conditions than when they are on instruments. Pilots are also more susceptible than usual to the stresses of flight such as fatigue, oxygen lack, and anxiety. These stresses may reduce a pilot's ability to think straight, so there is the danger of forgetting to use the instruments the minute things get tough. Anything that produces an emotional upset is likely to disrupt conscious mental processes and make the pilot much more susceptible to the illusion of false sensations.

The second point to remember is that the illusions that have been described in this section are relatively rare. Believe it or not, this can actually be a disadvantage. You learn to adjust to the sensations of normal flight as you gain flying experience, but the possibility remains that you will suddenly encounter a vivid illusion that you have never experienced before. If you do not know what the illusion is or how

you can handle it, you are likely to get panicky and let your emotions take over. When this happens, you are putting your life in the hands of your senses, and under such conditions they may prove inadequate.

Last, but not least, remember that many accidents occur as a result of indecision about going on instruments. With poor visibility you may begin to go on instruments and then sensory illusions can make you believe your instruments are wrong.

There is just one way to beat false interpretation of motion. Put your faith in your instruments and not in your senses. Know what kind of tricks your senses can play on you, keep calm, and have confidence in your instrument panel. Once you have acquired this confidence, you can fly at night and in weather as easily as if you were following the railroad tracks, two creeks, and a cornfield back to the airport. The moral is simple: The transition from VFR to IFR must be a complete and trusting transition.

OXYGEN EQUIPMENT

The development of oxygen equipment has necessarily paralleled the progress in aircraft performance. Without protection from the physiological problems at altitude the human element becomes the limitation on how fast, how high, and how well an aircraft can perform. Oxygen equipment is just one area of development which has enabled us to fly in the hostile environment above 12,000 feet. In this chapter you will learn the basic principles of oxygen equipment. Emphasis will be placed on the equipment currently being used. The proper and effective use of oxygen equipment will be stressed and will include the methods of checking the oxygen equipment prior to and in flight. The opportunity to use and become familiar with this equipment will be afforded you during the chamber flight phase of your training.

OXYGEN STORAGE

Aircraft operators who fly routinely either pressurized or unpressurized at altitudes in excess of 10,000 feet commonly employ a fixed oxygen installation. This consists of containers affixed within the aircraft and serviced through an exterior fuselage valve.

Light aircraft operators who normally fly below 10,000 feet often prefer to use portable O_2 equipment consisting of a container, regulator, mask outlet, pressure guage, etc., as an integral unit which may be taken aboard the aircraft each time a flight is contemplated at altitude. Portable equipment, in order to avoid weight and bulk problems, is limited in oxygen supply duration. Typical breathing time for four people at 18,000 feet is in the range of 1-1/2 hours using a 22 cubic foot container. Fixed O_2 installations usually offer much longer duration times. Actual times will depend upon size of oxygen containers in the system, and the number of people using the system.

OXYGEN STORAGE METHODS

Gaseous oxygen is stored in the containers at a pressure of 1800-2200 PSI. This is termed a high pressure system. The high pressure system is used very extensively in general aviation and commercial aviation.

Latest development in oxygen systems for aircraft make use of chemical action and are termed solid state oxygen systems. Solid state oxygen has come into its own through its use in new jumbo jet transport. It has weight, duration, and storage advantages not found in systems currently in use.

REGULATORS AND MASKS

A. Continuous Flow

The continuous flow O_2 regulator provides a flow of 100% oxygen. The rate of flow is usually measured in liters per minute. Flow rate may be controlled by turning a valve to alter the flow rate. Several regulators are offered which employ an altitude sensing aneroid to change the flow rate automatically.

Continuous flow masks utilize an oronasal face piece to receive the oxygen flow. The face piece does not usually have an air tight or oxygen tight face seal. This permits the user to exhale around the face piece or through small face piece ports or openings designed to dilute the oxygen with ambient air.

Continuous flow masks in use today make use of a *rebreather* bag. This bag is attached to the mask and enables the wearer to reuse a part of the exhaled oxygen. Usually, there is a device in the oxygen hose which enables the wearer to see that oxygen is flowing through the system.

The design of continuous flow systems limits altitude range. In order to accommodate personnel flying in the higher altitude ranges several continuous flow masks are now being marketed that provide an air tight seal with exhalation valves which convert the rebreather bag into a reservoir bag. Very careful attention to system capabilities is required in use of this type equipment above 25,000 feet even though it has been certificated to 41,000 feet.

B. Demand and Pressure Demand

The demand regulator, as the name implies, operates to furnish oxygen only when the user inhales or *demands* it. A lever may also be employed to enable the regulator to automatically give either a mixture of cabin air and oxygen or 100% oxygen. This is referred to as the automix lever. The regulator is set up to give varying amounts of oxygen to the user depending upon the altitude attained.

The demand mask is designed to accommodate an air tight and oxygen tight seal to the face. This mask is expected to retain all of the oxygen thus inhaled into the mask by the user and not be diluted by entry of outside air. The demand regulator and mask provides a higher altitude capability than most continuous flow systems. It may be safely used to altitudes of 40,000 feet.

Pressure demand regulators are designed to furnish oxygen on inhalation either as a mixture of air and oxygen or 100% O_2. This regulator also provides a positive pressure application of oxygen to the mask face piece enabling the users lungs to be pressurized with oxygen. This is of great benefit at extreme altitudes such as 40,000 feet or higher. The oxygen pressure flow may be either manually controlled or function automatically on some regulators at a certain altitude through aneroid action.

The pressure demand masks are designed to create an air tight and oxygen tight seal. The inhalation and exhalation valves are specially designed to permit oxygen pressure build up within the mask face piece and thus supply oxygen under pressure to the lungs.

It is essential that demand and pressure demand masks be properly suspended by an adequate head harness and that the masks be afforded tension adjustments in order for the user to obtain a leak proof seal to the face. The higher you fly, the more critical this adjustment becomes.

PRE-FLIGHT OXYGEN EQUIPMENT CHECK

Prior to flight a person should locate the oxygen mask, practice donning it, and adjust head harness to fit; locate and check function of oxygen pressure gauges, flow indicators and connections; and check quantity of oxygen in the system. The mask should be donned and the O_2 system should be checked for function.

A physical check of the mask and tubing to spot any cracks, tears, or deterioration would also be indicated. If a person is using a mask connection to an individual regulator, check for regulator condition and lever or valve positions as required by that particular system.

GENERAL RULES FOR OXYGEN SAFETY

Do not inspect oxygen equipment with greasy hands. Do not permit accumulation of oily waste or residue in the vicinity of the oxygen system.

Do not use surplus oxygen equipment unless it is inspected by a certified FAA inspection station and approved for use.

Some military components use oxygen containers stressed for a pressure of 450 PSI (low pressure). Needless to say, a hazard exists if a person attempts to put 1800-2200 PSI O_2 pressure in this type container. High pressure O_2 containers should be marked to indicate *1800 PSI* before attempting to fill the container to this pressure. Most individuals do not posses the equipment necessary to fill an aircraft oxygen container from another source of high pressure oxygen. It is recommended that oxygen system servicing be done at FAA certified stations such as are located at some fixed base operations, terminal complexes, etc.

After any use of oxygen, careful attention should be given to ascertain that all flow is shut off before lighting cigarettes, etc.

Oxygen systems must be engineered to protect the individual to the maximum anticipated flight altitude of the aircraft. Before purchasing any oxygen equipment, it is recommended that you brief the distributor on such factors as peak altitude to be flown, number of persons who will use the oxygen system, expected oxygen breathing duration, range of the aircraft, and any other information you think will be helpful in designing a proper oxygen system. Do not make any modification to the system without consulting the supplier or distributor.

Do not place portable oxygen containers in the aircraft unless you fasten them securely to insure against displacement in the event of turbulence, unusual aircraft attitudes, etc.

SELF IMPOSED STRESS

This problem continues to be present in aviation because the flyer occasionally imposes stress on the body by doing some things or by omission of things that should be done. Examples of self imposed stresses are excessive or indiscriminate use of drugs, alcohol, or tobacco and failure to assure proper rest, diet, or physical fitness. Failure to adhere to a *common sense* approach in dealing with this many faceted problem could lead to a complete breakdown of ability to maintain physiological effectiveness under adverse conditions. Fatigue, as it relates to aircraft accidents, is a combination of physiological and psychological factors, most of which can be traced back to self imposed stress or the problem stresses of daily living.

ALCOHOL

Alcohol is eliminated from the body by a variety of methods and at an average rate of 0.015% per hour. There has been discussion regarding the so called "8-hour" or even "12-hour" rule governing the time from *bottle to throttle*. Considering the rate of elimination of alcohol, an average person (160 lbs.) at Sea Level, consuming 4 drinks (1½ oz. each) of 80 proof (40% alcohol) whiskey, could have a peak blood alcohol level of 0.10% (equal to the D.W.I. level). If this individual abstains from further alcohol consumption and remains safely on the ground, the blood alcohol level will drop to approximately 0.04% in 4 hours or "marginal" according to the National Transportation Safety Board (NTSB) criteria and is considered legally "sober" enough to drive in most states. However, even at this "marginal" level, if attempts are made to fly an airplane, there could be added difficulty or performance decrements at altitude possibly due to the compounding effect of mild hypoxia with prior alcohol consumption. Actually, some studies are indicating not just a compounding effect, but a synergism, that is, the combined action of the two: alcohol and altitude is greater than the sum of the action of one alone.

Among the first losses (or performance decrements) caused by alcohol are: judgment, comprehension, and fine attention. Decrements in vision and hearing first appear at blood levels as low as .01%. Reaction time is decreased by ten to thirty percent with blood levels of 0.10 to 0.20%. Alcohol, even in small amounts, produces, among other things, a dulling of critical judgment; a decreased sense of responsibility; diminished skill reactions and coordination; decreased speed and strength of muscular reflexes (even after one ounce of alcohol); decreases in efficiency of eye movements during reading (after one ounce of alcohol); increased frequency of errors (after one ounce of alcohol); constriction of visual fields; decreased ability to see under dim illuminations; loss of efficiency of sense of touch; decrease of memory and reasoning ability; increased susceptibility to fatigue and decreased attention span; decreased relevance of response; increased self confidence with decreased insight into immediate capabilities.

When a person undertakes to pilot an aircraft from a point on the surface of the earth, along a given course, to another point on the surface, hundreds of decisions must be made, some on the basis of incomplete information, some on the basis of unexpected information (adverse weather, etc.). Proper maneuvers must be accomplished and a return to the home station or a landing at an alternative point may be judicious. It can readily be seen that anything which impairs the pilot's ability to make successive decisions will lead to an increase in the accident probability. For example, an elderly pilot flew into a field which was usually uncontrolled, but which had a temporary control tower in use on the day in question. Evidence indicated that the .035% blood alcohol level found was sufficient to decrease the

pilot's ability to properly respond to the red signal from the tower. Result, a fatal accident. A businessman, who had consumed alcohol to the point that he markedly slurred speech in his communications with the tower, made repeated passes at a hard surface runway which was at least 4,000 feet long. Even with the assistance of the tower personnel, he was unable to land. He later had a fatal crash when the aircraft ran out of fuel.

It should be noted that the currency of a given pilot, the flight history of the pilot and the familiarity of the pilot with the aircraft and geographic area involved, bear importantly on the degree of adverse effects of alcohol on flight proficiency.

It should be stressed that the great majority of general aviation pilots are mature and dependable individuals, and operate their aircraft safely. However, through lack of understanding, proper education, or emotional problems, some pilots undertake flights while under the influence of alcohol and a certain percentage of these do have accidents.

Part 91 of the Federal Aviation Regulations, *General Operating and Flight Rules*, provides that no person may act as a crew member (or pilot in command) of a civil aircraft while under the *influence* of intoxicating liquor. The individual pilot must also be alert to sources of ethyl alcohol other than liquor (cough medicine, tonics, etc.). A good rule to follow is that if one is sick enough to require these substances, the individual is too sick to fly at that time.

It is also a good idea to allow a reasonable time from *bottle to throttle* although eight hours should be sufficient following light indulgence (4 ounces). Eight hours is the minimum time allowed by the Federal Aviation Administration for its' own pilots before flight can be undertaken following alcohol consumption. This rule has worked with apparent safety, within the context of FAA flight activities and through the exercise of judgment by FAA pilots.

Aircraft pilots who apply their knowledge of the potentially disastrous consequences of mixing alcohol and flying will do much to reduce the annual toll of life thus lost.

DRUGS – GENERAL

Drugs, as well as the conditions for which they are taken, can interfere with perception, decision making, and motor skills and can cause significant decrements in performance. Few activities are as dangerous as piloting an aircraft when such decrements occur.

The importance of flight personnel in aviation safety is illustrated by the fact that human factors are usually involved in eighty percent of all major aircraft accidents, both military and civilian. Investigation often reveals the presence of several human factors, each of which, when evaluated separately, might result in only a small, difficult-to-measure decrement. Among the more common findings are ingestion of drugs (including overdosage), and the symptoms or disease for which they were taken. All drugs are evaluated before they are allowed to be placed in use. However, very few of these drugs are evaluated as to their effect on the flying community, and even when they are evaluated, the test subjects usually are healthy, experienced, relaxed pilots who are generally given the dosage of the drug recommended by the manufacturer. Research by the Federal Aviation Administration's Civil Aeromedical Institute found that pilots' reactions at altitude to normal dosage of over-the-counter drugs were different from their reactions at sea level.

Recent investigations of general aviation accidents have revealed a significant incidence of drug and alcohol involvement. In many cases, self-medication with over-the-counter drugs is responsible for serious reactions among the flying community. A widely advertised, and apparently widely used, cold

remedy has caused incapacitating dizziness during flight in two cases that have been studied. Fortunately, a second pilot was present in each case to take over and land the aircraft. In one case, the second pilot was a student, which made the margin of safety rather thin. In both cases, twice the recommended dosage had been taken.

In other cases, doctors who are not pilots, flight surgeons, or designated Federal Aviation Administration Medical Examiners (AME's) have prescribed types of dosages of drugs which are extremely harmful and should not be taken if the user is to fly. In one such case, the pilot was flying below the top of surrounding low mountainous terrain during a thunderstorm and struck the ground. All four occupants were killed. Personnel at a nearby airport, from which the pilot had departed, reported that he had been in good spirits and had resisted suggestions that he delay his departure until the local thunderstorm activity had subsided. Three bottles of prescribed medication were found on the pilot's person. All pilots should have a greater awareness of and respect for the dangers of flying while taking drugs, particularly new drugs on the market. Each pilot should consult his AME physician when in doubt. The AME has received training as to which drugs are safe to take and fly.

DRUGS – OVER THE COUNTER

The following drugs can be bought over the counter and do not require a doctor's prescription. Only some of these drugs are discussed here. For full information concerning these and other drugs, consult your AME physician.

A. Ethyl Alcohol

This drug has been discussed at some length previously. It is suggested that you review the previous pages of this chapter for full details.

B. Tobacco

Some cigarettes contain ten to twenty milligrams of nicotine, of which up to 2.3 mg is absorbed if the smoke is inhaled. Up to 1.5 mg is absorbed through the mucous membranes of the mouth if the smoke is not inhaled. A 2.5 mg oral dose of nicotine can cause nausea and 50 to 60 mg is the lethal oral dose. The person who smokes two packages a day exceeds these amounts, but rarely reports any nicotinic effects because of rapid detoxification and the development of tolerance.

Carbon monoxide constitutes up to 2.5 percent of the volume of cigarette smoke and more in cigar smoke. If the smoke of three cigarettes is inhaled at sea level, a blood saturation of 4 percent CO may result.

This will reduce a person's visual acuity and dark adaptation to the extent of the mild hypoxia encountered in flight at 8,000 feet. Smoking at 10,000 feet produces effects equivalent to those seen at 14,000 feet without smoking. The effects of smoking at 20,000 feet are equivalent to the effects otherwise expected at 22,000 feet. With heavy smoking, blood saturations as high as eight percent are possible. This causes a corresponding drop in blood oxygen saturation and will lead to hypoxia.

Six Reasons for Dedicated Pilots Not to Smoke Cigarettes

1. Cigarette smoking markedly shortens life;

2. Cigarette smoking causes specific physiologic debilitations and diseases that are medically disqualifying for pilots;

3. Tobacco (nicotine) addicts lead chemically-centered lives, perpetually concerned about the hourly immediate availability of nicotine;

4. The nicotine addicted brain experiences withdrawal during sleep, resulting in poorer quality sleep and chronic insomnia;

5. The smoking pilot converts persons nearby into involuntary smokers, doing potential harm to them;

6. The smoking pilot damages gyroscopic instruments in those aircraft drawing flight deck air through the instruments, and fouls the outflow valves in pressurized aircraft.

C. Aspirin

Probably no over-the-counter drug is used more often or more indiscriminately than aspirin. Toxic effects are relatively rare and are almost associated with large doses. However, some serious reactions have been attributed to excessive use of aspirin.

Aspirin compounds containing aniline derivatives may cause a situation where the blood will not carry oxygen, if used indiscriminately. Excessive use of bromide containing compounds may cause psychological problems as well as an inflammation of the skin. Quinine containing preparations may cause vertigo, a ringing in the ears, deafness, or nausea.

Generally speaking, if you take aspirin in small dosage and have had no reactions in the past, it is probably safe to take this drug and fly.

D. Antihistamines

Undesirable effects which are possible with the use of antihistamines are drowsiness, inattention, confusion, mental depression, dizziness, vertigo, and impaired depth perception.

Because of the adverse effects of these symptoms on the safety of flight, pilots should not take *short-acting* antihistamines during the eight hours before flight or take the *long-acting* preparations within sixteen hours of flying.

E. Nasal Decongestants

Since these compounds can occasionally be used to advantage during flight (usually for the relief of a blocked ear or sinus during descent), their proper use in flight is not taboo. Indiscriminate use of these compounds can cause an increase in the heart rate, nervousness, tremor, incoordination, and dilation of the eyes with other visual disturbances.

F. Motion Sickness Remedies

Several types of drugs are used for the relief of motion sickness. Scopolamine is effective and has few reported side effects when used as directed. Antihistamines are used widely but often cause drowsiness, dizziness, and blurred vision. Barbiturates have been used but are seemingly of less value and definitely should not be used by pilots. Most pilot trainees who become airsick will have no difficulties after a number of flights. Therefore, some of these drugs may be prescribed for use in dual flights. Otherwise, the use of these preparations during flight or within eight to twelve hours before flight is generally not recommended.

C. Antidiarrhea Preparations

Drowsiness may occur with excessive use of paregoric. Visual disturbances may be noted with the use of antispasmodics. Aircraft accidents have been caused by the effects of preparations containing belladonna. Therefore, flying duties are generally not recommended for at least twelve hours after the use of these drugs.

DRUGS – PRESCRIPTION

When drugs are prescribed for the flyer, the major consideration should be whether the disease being treated is compatible with safe participation in aerial flight. Most diseases, however, must be evaluated on an individual basis by the physician. Often these diseases temporarily disqualify the person from flying. Only commonly used drugs for the symptomatic relief of realtively minor conditions appear here.

A. Amphetamines

While the source of amphetamines can often be traced to well-meaning spouses and friends, this agent does require a prescription. Amphetamines diminish a sense of fatigue and even delay its onset up to four hours; they tend to force the body beyond its natural capacities. Nervousness, impaired judgment, and a feeling of well being are sometimes reported, particularly with overdosage.

When amphetamines are taken in conjunction with a weight reduction program, low blood sugar may be present. The effects of this low blood sugar are additive to those of hypoxia. Amphetamines should not be used during flight.

B. Tranquilizers

With over 80 million prescriptions being written for tranquilizers each year, it is hard to believe that pilots are not among the users. In many cases, the condition involved is disqualifying for flying duties. However, some pilots often do not inform their physicians of their flying activities, thus adequate attention is not given to the undesirable effects of these drugs. While it may not be readily apparent, even the nonsedating tranquilizers usually have some measurable effect on judgment, alertness, efficiency, and over-all performance.

C. Sedatives

Sedatives have been used under controlled conditions to guarantee adequate rest before flight and alertness during flight. However, pilots should not take any sedatives for 12 to 24 hours before flight.

D. Antibiotics

When antibiotics are indicated, the airman is often too sick to fly. When in doubt, do not fly!

E. Cardiac Agents

Hypertension and most hypotensive agents are disqualifying for flying duties. Most medical authorities agree that sensible weight reduction programs are better for the pilot than medication.

F. Muscle Relaxants

These agent, with or without analgesic and tranquilizer actions cause sufficient weakness, sedation and vertigo to contraindicate flying duties for at least twelve hours after their use.

PHYSICAL FITNESS

Considerable efforts have been made by the President's Council on Physical Fitness, the Armed Forces, and other groups, to awaken the American people to the fact that all of us are in better health than ever before, but that we are also in poorer physical condition than ever before. Many of us wish we were in better shape, but we just cannot seem to find the time to spare on a program to attain our desires. Participation in outdoor activities, such as golf, hiking, swimming, and others is somewhat limited during winter months and some people just don't like these activities even if the weather is conducive. Therefore, it is to the desk ridden, snow bound, or otherwise indoor habitated person, that this portion of the handbook is devoted.

Noted scientists, such as Balke and others, state that a person's job efficiency, life expectancy, and general health are directly related to their physical fitness.

AN EXERCISE PROGRAM

NOTE: The first step in considering any physical fitness program should be a thorough medical examination and consultation with your physician.

There are many exercise programs available for those inclined to indulge in a more or less fixed program approach to physical fitness. They range anywhere from walking and running to a series of graduated exercises designed to affect specific muscle groups. It is important that each person assess individual needs (consultation with a physician, physical educator, and a full length mirror may help), and then map out a long range (would you believe, for life) program. As important as the exercise program itself is the manner in which it is approached. This should be done cautiously and progressively to attain maximum long range benefits. The following is an example of a simple but very effective training program as developed by Dr. Kenneth Cooper of the United States Air Force:

Exercise	*Point Value*
Running 1 mile in 7-9 minutes	5
Walking 1 mile at a 3.75 MPH rate	2
Cycling 1 mile at a 10-13 MPH rate	1
Swimming at a 75-90 feet per minute rate	½ point/ minute

Using this scale, the subject may select one exercise or a combination of several. Very slowly and comfortably, enter into the program by attempting to reach an average level of 10 points per week by the end of the sixth week. By the twelfth week, an average 20 points per week should be attained, and by the eighteenth to the twenty-fourth, 30 points per week should be the goal.

Week	*Points*
1 – 6 weeks	10
6 – 12 weeks	20
12 – 24 weeks	30

Maintaining a 30-point-per-week average will enable most people to be classified in the good to excellent physical fitness category.

During the initial stages of training, exercise should be taken 5 to 6 times a week and be *slowly* progressive by all means. With improvement, the exercise can be made more strenuous and the frequency reduced to 3 or 4 times a week.

There are other good exercises that have not been discussed. If a person exercises regularly using a different exercise and can perform at a level compatible with the good or excellent categories mentioned previously, this practice should continue. The level of fitness is what is needed regardless of how it is accomplished.

A. Conclusion

It is the abnormalities in the cardiovascular system that interfere so frequently with the continuation on flying status. Currently, exercises are being used to rehabilitate people who are already suffering from heart disease, high blood pressure, and many other potentially grounding medical problems. However, once they are removed from flying status because of one of these conditions, it becomes difficult to regain this status. A little preventive medicine practiced now will help to insure flying status for as long as an aviator desires to fly.

DIET

Flyers should use a diet that is properly balanced with respect to proteins, carbohydrates, and fats, and containing other essential nutrients such as vitamins and minerals. It is impossible to recommend a fixed caloric intake because of the wide range of sizes of flyers and their rate of body metabolism, again consultation with your physician is in order.

Over-eating should be avoided before and during flight to prevent excessive gas formation in the digestive system and drowsiness.

Gas expansion may result in severe pain at higher altitudes, and a sleepy pilot may become a hazard. Under-eating, on the other hand, may lead to a condition known as hypoglycemia or low blood sugar. Blood sugar is the fuel that keeps the body functioning at normal efficiency. Failure to eat two meals in a row, especially dinner and breakfast, can result in a gradual decline in muscular coordination, visual acuity, and proper judgment. While the degree of loss of efficiency may be small, it could be enough to get the pilot in trouble if other problems arise to complicate the total flying situation.

There are many sources of diet information available to us. A well balanced food intake program and attention to eating at regular intervals will prevent pilots from getting into difficulty while engaged in the performance of their aerial duties.

REST

As in the case of physical exercise, much has been written about the amount of rest or sleep an individual should strive to attain on a daily basis. Busy pilots frequently find themselves in a position in which sleep is sacrificed to some pressing business or personal activity. Management of time, therefore, becomes of prime importance if we are to meet our goals and yet maintain good health. Many people who are plagued by too few hours sleep at night have developed the habit of taking daytime naps. This is highly recommended when time and conditions permit. Individuals should also strive to ascertain their own requirements for sleep. On the average, most people feel best when they get from six to eight hours sleep nightly. Here, we have to compromise between what we know is good for our health and flight safety and the demands that life will try to make upon us. Physical and psychological stress as they are encountered from day to day should be taken into account to determine the amount of rest needed if we are to avoid chronic fatigue.

CIRCADIAN RHYTHMS

Cycles or *rhythms* can be defined as repetitions of situations, events, and levels of activity in regular intervals or periods of time. The most impressive cycle in the biological world is that associated with the earth's axial rotation in the powerful electromagnetic radiation field of the sun – the light and darkness cycle of the *day-night cycle*. This biologic rhythm of a period of 24 hours, with the phases of rest and activity, or sleep and wakefulness, is called the *circadian cycle* (from Latin, *circa* and *dies* – about 1 day). Its stability and changeability, particularly in reference to air travel, is the topic of this section.

A. Stability and Flexibility of the Circadian Cycle

The circadian cycle in humans and animals shows a certain degree of stability which can be expressed by the term Homeostasis. The normal physiological time requirements of the adult for sleep, or the duration of sleep is about 7 hours in every 24, plus a brief nap, in those people who are not under community, social or professional pressure. Most people, at the beginning of their nights sleep, fall immediately into a deep sleep for 2-3 hours, followed by a light sleep – the phase of dreaming and rapid eye movements. The stability of the circadian cycle is evidenced by the following facts:

1. It is impossible to break this cycle by ignoring sleep for a number of days. This leads to neurotic disorders, as proved by numerous sleep deprivation experiments.

2. The duration of the circadian rhythm can be shortened to 18 hours and extended to 28 hours by exposing the individual to artifical light-dark cycles. The physiological clock accepts these variations by adaptation. But going below this minimum or beyond this maximum is outside the clocks adaptability, and it continues to run at its routine 24-hour cycle.

3. The sleep and wakefulness cycle continues near its circadian pattern even in constant light environments, as observed in inhabitants of the subpolar twilight zones and in animals kept under similar constant conditions in the laboratory.

4. A shift in the phases of the circadian cycle is possible, but it requires a certain time for readjustment. This is a familiar problem in work time shifts in many occupations. Persons in these activities feel some inconvenience for a number of days after they have changed duty hours. There are, of course, differences in the sensitivity to a phase shift. A few people can sleep at any time, at any place, and under any conditions, but the majority is more or less sensitive in this respect.

In the above examples of work time shift the individual stays in the home time zone. With the development of fast-moving surface vehicles, and especially since the advent of the airplane, a new way of phase shift of the day-night cycle is experienced by millions of people, namely, by *time zone changes via air travel.*

B. Circadian Rhythm and Air Travel

As statistical studies in long distance flights have shown, most people are sensitive to this travel-produced phase shift, and experience some discomfort for several days. They become hungry, sleepy, or are awake at the wrong time with regard to the new local time. Their "head clock" and "stomach clock" and elimination system are confused. After transcontinental flights in the U.S. these conditions last from 3-4 days; after transatlantic flights, 5-6 days. Crossing 12 time zones, which leads to a complete reversal of the day-night cycle, resynchronization may take 10-12 days. As a general rule, most travelers adjust to a new circadian cycle at a rate of nearly 1 hour per day. Some people feel more easily adjusted after eastbound flights, others after westbound flights, and some when returning to their home time zone with its familiar climate and social order. There are, of course, a few people who are not particularly time sensitive at all.

The problem of circadian desynchrony is especially important for those whose occupation as such involves time zone changes. Aircrews of long distance air routes are in this category. They cross and recross a number of time zones several times a month or even fly around the world once every month. A too-frequent shift of the circadian cycles causes fatigue; this is well recognized by the pilot associations and medical directors of the airlines.

NOTE: Circadian Rhythms are not to be confused with the fad term "bio-rhythm" which refers to theoretical cycles in physical, emotional, and intellectual functions (usually about 1 month duration). Many studies have concluded that this theory has no real value in the aeromedical or physiological training application.

SURVIVAL

Perhaps the simplest way to address the subject of survival as it applies to modern day flying is to accept the most prevalent attitude concerning the subject – it can't happen to "me." In actuality however, it can and does happen to "me." Every time an aircraft flies over a remote or desolate area, the occupants of the aircraft are subjected to the possibility of an inflight emergency...and emergency landing...and the challenge of survival until rescue. For this reason, the subject must be addressed as sincerely as any other topic in this book. Travel from airport to airport may take you over hostile areas of dense or tropical forest, extreme temperature changes, mountains, swamps, deserts, or extended bodies of water. Though only minutes away from civilization by air, problems of survival in any of these hostile areas could result in death, even though a safe emergency or precautionary landing is executed.

PSYCHOLOGICAL ASPECTS

A human being's ability to survive is directly affected by the psychological aspects of survival and the will to survive. The basic difference between success or failure to survive under adverse conditions can often be attributed to an individual's ability to overcome stress and to continue to function effectively. A prior knowledge of the psychological factors and the importance of a strong will to survive, in addition to a knowledge of survival principles and techniques, is extremely important to a survivor.

Will power alone has often been the key factor in reported successful survival incidents. These reports are not held as classic examples of how to survive, but they do show that strong will power can often help one to conquer what appears to be an impossible obstacle.

It is not a sin to make an emergency or precautionary landing and survive, but there could be the tragedy of making a safe landing and not surviving because of lack of knowledge of your reactions under stress.

A knowledge of the "seven enemies of survival" will help anyone to be psychologically prepared to cope with them. They are common wants experienced in minor ways almost every day of our normal lives. When combined with other stresses of survival, these wants can become significant and of primary importance to us and could cause actions that would reduce our ability to survive. These seven enemies are:

1. Pain
2. Extreme temperature variation
3. Thirst
4. Hunger
5. Fatigue
6. Boredom
7. Loneliness

A survivor may have to function for long periods in spite of pain. A psychological adjustment is required to combat the effects of pain in such cases. Goals must be realigned. When in a survival situtation and it is impossible to eliminate pain, your goal must be altered to function effectively in spite of pain.

Extreme temperature and the psychological problems related to heat and cold will affect a survivor's capabilities. When extremely cold, there is a tendency to think of nothing but warmth. This must be altered to think of and engage in the activities necessary for getting and staying warm. In extremely hot climates, there is a tendency to think of nothing but cooling off. Again, this thinking must be altered and you must strive to decrease and maintain lower body temperature. In other words then, you must think in positive terms of "What can I do to combat the effects of extreme temperature," not just how hot or how cold it may be.

Thirst, and the effects of dehydration, may cause serious psychological problems as well as physiological problems. You must avoid depriving yourself of water if it is available, even when the supply is limited. If you are thirsty, drink as long as water is available. Do not deprive yourself, keep yourself fit. If rationing must take place, ration perspiration, not water intake.

Hunger, like thirst, is an enemy of survival because of its effect on you psychologically. Remember, in most instances, you can exist for an extended period of time without food, others have. Do not allow yourself to become controlled by the pangs of hunger.

Fatigue is also a problem. Fatigue reduces mental efficiency as well as physical efficiency. Do not become so involved in your attempt to survive that you forget that your body needs periodic rest. Fatigue can make you careless. Attempt to conserve your energy so that you will have enough in the event that you need to give yourself that "extra push" in order to accomplish some absolutely necessary activity.

Boredom is an unnecessary enemy of survival, but it attacks many. Simply fulfilling your basic needs should fill your time with meaningful activity that will eliminate boredom. Even when physical activity is impossible, boredom can be combated by other mental activities. A technique that might be used is to recall a book or movie in as much detail as possible, or formulate a mental plan to construct something. Relive portions of your life. Any mental endeavor to get your mind off your situation will be beneficial.

CLOTHING

In hot climates, survival is somewhat easier than in cold climates. Proper clothing is essential for protection against the sun, insects, and the hazards of terrain and vegetation. In cold climates the necessity for warm clothing is acute. It is almost impossible for us to have adequate clothing to protect us from prolonged exposure, especially if we are inactive; therefore, we must immediately provide shelter from the wind and weather. Snow is a good insulator and can be formed into a wind break or small shelter without too much difficulty. A sleeping bag provides excellent protection from extreme cold. We should, however, have brief periods of light activity to restore body temperature. We should rest as much as possible as this is a means to conserve body heat. We should be extra careful not to overexert or overheat ourselves, as this causes a wasteful loss of body energy and water. Freezing perspiration may also tend to expose us to frostbite or excessive loss of body heat. Severe exhaustion, malnutrition, or injuries may result in fatal exposure to cold if we do not adequately prepare ourselves for winter flying.

For overwater flights, we should have a life vest and raft as well as anti-exposure garments when cold weather is prevalent. Our survival time in water near the freezing level can be measured in minutes. This time can be increased many fold if we have the necessary protective equipment.

FOOD AND WATER

The importance of an individual's nutritional status in a survival situation is apparent. Check with a reliable supplier before making a decision on the kind and amount of food you intend to take with you on your flight. The Department of Defense has spent many years and countless dollars on survival foods.

We can profit from their experience. They have three main survival rations, each consisting of concentrated foods designed to sustain life under different climatic conditions. One packet contains some 1,700 calories and derives this from starch jelly bars, coffee, tea, and sugar. The second packet provides 2,100 calories and is designed for winter survival. The components of this packet are cereal bars, fruit cake bars, cheese bars, chocolate bars, starch jelly bars, sugar, soluble coffee, tea, and cream. They have another individual survival packet which has 3,500 calories and derives its energy from meat food product bars, fruit cake bars, cereal bars, tea, sugar, and seasonings.

All of the above packages are available through sporting goods stores, surplus stores, or from the manufacturer. Of course, some persons prefer to make their own food kits. To those persons, may we suggest you consider concentrated foods such as bouillon cubes or other items which have a high caloric content and do not occupy much space.

A continuous supply of drinking water is essential to human life. Man can live about 30 days without food, but can live for only about 3 days without water. Most land areas of the world provide sufficient amounts of drinking water for survival; however, deserts, arctic regions, and the oceans do not. If we fly over deserts, there is little or no water available; therefore, we must carry our water with us or materials needed to make solar stills. Cans of water can be bought at many stores in most cities. The Civil Defense authorities in your town may be able to suggest a source of food and water suitable for equipping your aircraft.

In arctic regions water exists, but generally is in the form of snow or ice. We must carry along fuel to melt these forms of water. Ice is better to melt than snow because there is less air in the ice, therefore, requiring less fuel. Snow or ice melted in the mouth takes large quantities of body heat; therefore, we should do this sparingly.

On the ocean, water may be obtained by desalting or distilling sea water. Sea water must not be drunk, even if diluted, because the salt concentrations produce a greater load on the kidneys and result in a greater loss of fluids in the urine. Desalting kits as well as solar stills can be purchased. The latter requires sunlight to operate.

If water supplies are limited, diet should be light and composed mostly of sugars, as proteins and fats increase the amount of waste products the kidneys must secrete. There are two ways to conserve water:

1. Rest and avoid physical exertion which results in sweating.

2. Keep the body cool in hot climates by seeking shade, wear light weight clothing, and avoid travel while the sun is hot.

SURVIVAL KITS

Some typical items which may be carried in a survival kit are:

1. Knife, either a sheath knife or a *Boy Scout* knife.

2. Small compass. (There is nothing like walking in one direction to be found.)

3. Matches. Suggest wood matches and dip the heads in paraffin wax. There is a round match container which is excellent for a survial kit. It has a piece of flint on one end and with the aid of a knife and a small piece of cotton a fire can be readily started.

4. Signaling equipment such as a signal mirror, flashlight, whistle, signaling flares, and smoke bombs are also items to consider. Recent developments have made these quite effective.

5. Medical items such as a first aid kit. This kit should have, among other items:

a. Two bandages, Compress 4 x 4 inches.
b. Two bandages, Compress 2 inches x 6 yards.
c. Bandage, Triangular, Compress, 37 x 37 x 52 inches.
d. Tourniquet, Elastic, 2¼ inches x 5 feet.
e. Gauze, Petrolatum, 3 x 36 inches.
f. Package of band-aids.
g. Small bottle of methiolate.
h. Water purification tablets.
i. Small bottle of petrolatum jelly.
j. Chloroquine and bug repellents.

6. Fish hooks, snares, and other items for procurement of food.

8. File, pliers, hatchet, wire saw, and small whetstone. Depending upon the geographical location, number of people, and duration of survival situations, food and water should also be considered in any survival kit.

Other recommended items include a razor with several additional blades, tooth brushes, soap, and toilet paper.

SOURCES OF INFORMATION

There are many sources of information available to you concerning the subjects discussed in this chapter. It is important that you seek out one or more of these sources and have such information available in the event that you need it. Better yet, look at the information periodically, review the basics of survival before the emergency, not after the emergency.

The military services have manuals covering the many aspects of survival. These may be purchased from the U.S. Government Printing Office, Washington, D.C. Flying organizations that produce a pilot's handbook usually include a chapter on survival. Also, there are many commercial publications available in both hard cover and paperback editions. Check your local book store. Don't overlook the Boy Scouts of America Field Manual. Many of you may have a copy stored in your bookcase. In the event that it does happen to "me" (that is you find yourself in a survival situation) don't fail to meet the goal – *rescue*, for lack of a little knowledge.

ALTITUDE CHAMBER FLIGHT

1. Pre-flight briefing.

2. Mask fitting and operation of oxygen regulator and intercom system in chamber.

3. Ascent to 6,000 feet and descent to ground level to determine proper clearing of ear and sinus cavities. Rate of ascent and descent is 3,000 feet per minute.

4. Reascend to 8,000 feet at a rate of 3,000 feet per minute.

5. Rapid decompression (8-10 seconds) from 8,000 feet to 18,000* feet without the use of supplemental oxygen. Masks will be readily available at all times and will be used as instructed.

6. Ascent to 25,000 feet at the rate of 3,500 feet per minute.

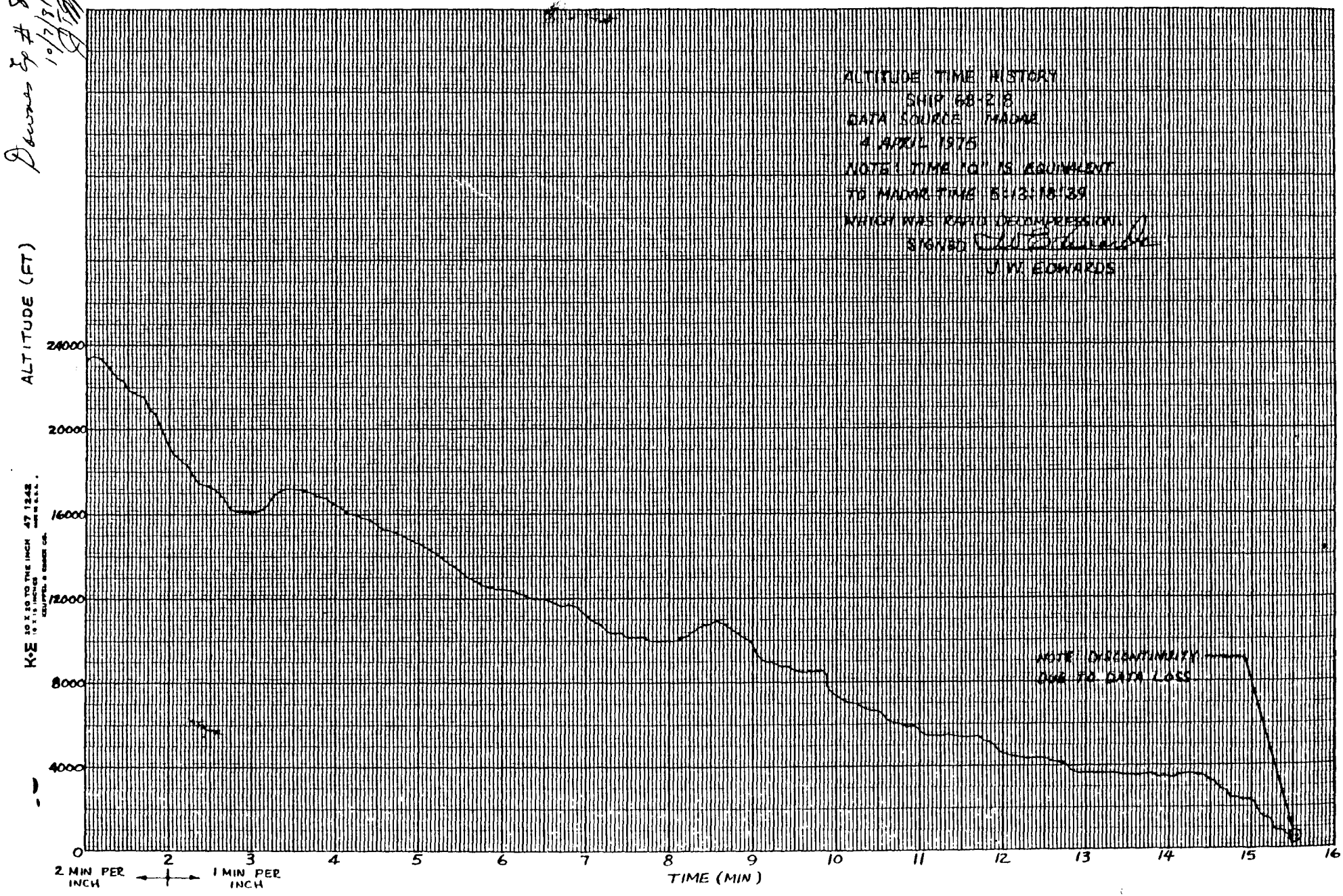
7. Removal of masks at 25,000 feet to enable trainees to experience symptoms of hypoxia. Five minutes time limit off supplemental oxygen.

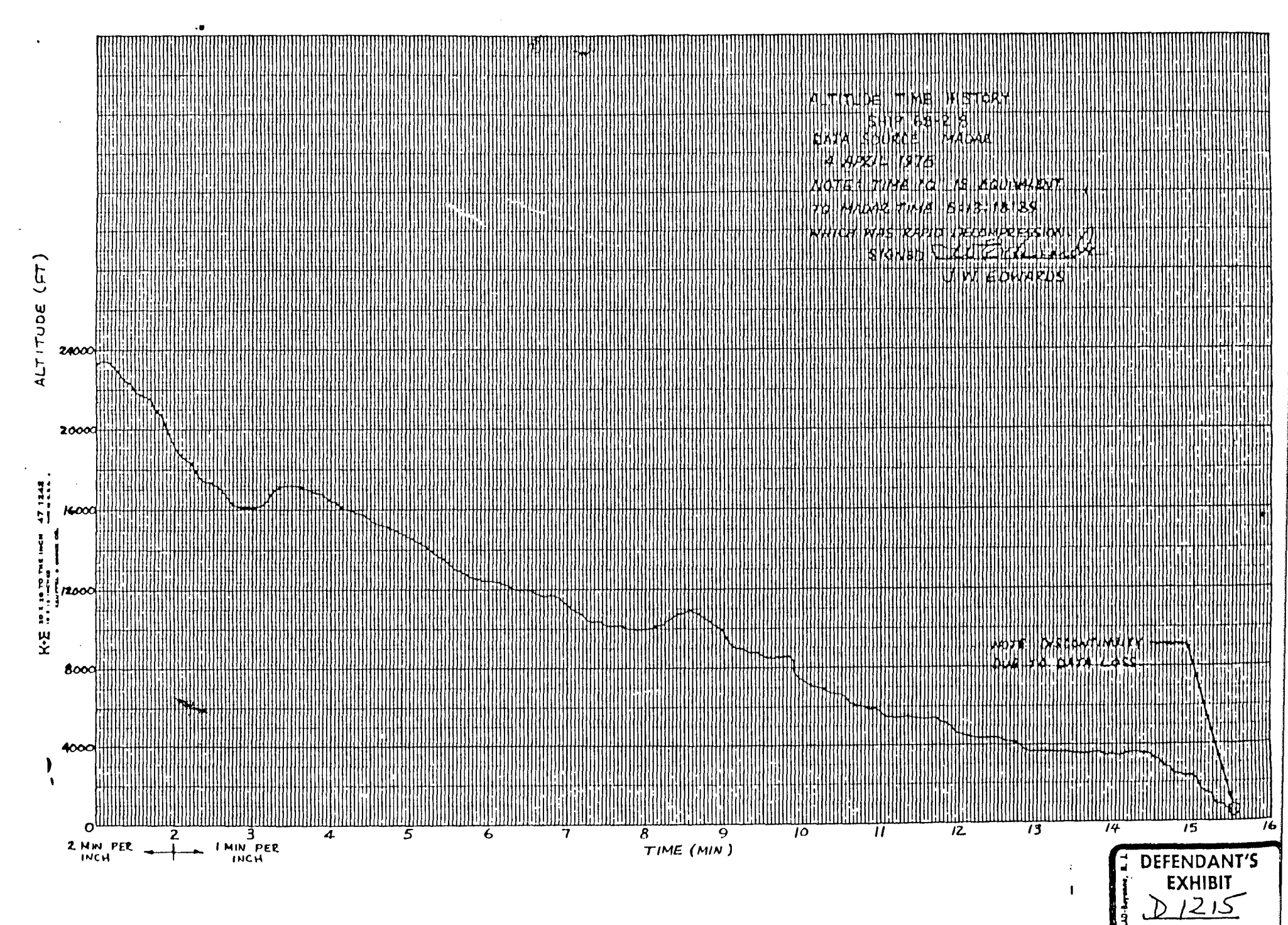
8. Descent to ground level at the rate of 3,500 feet per minute. Trainees will practice pressure breathing and discuss their hypoxia symptoms on descent.

*This procedure is used at altitude chambers which are equipped with independent vacuum accumulators. Other chambers which use the main portion of the chamber itself for an accumulator, may have to return to ground level before proceeding with the rapid decompression.

Flight Profile Chart

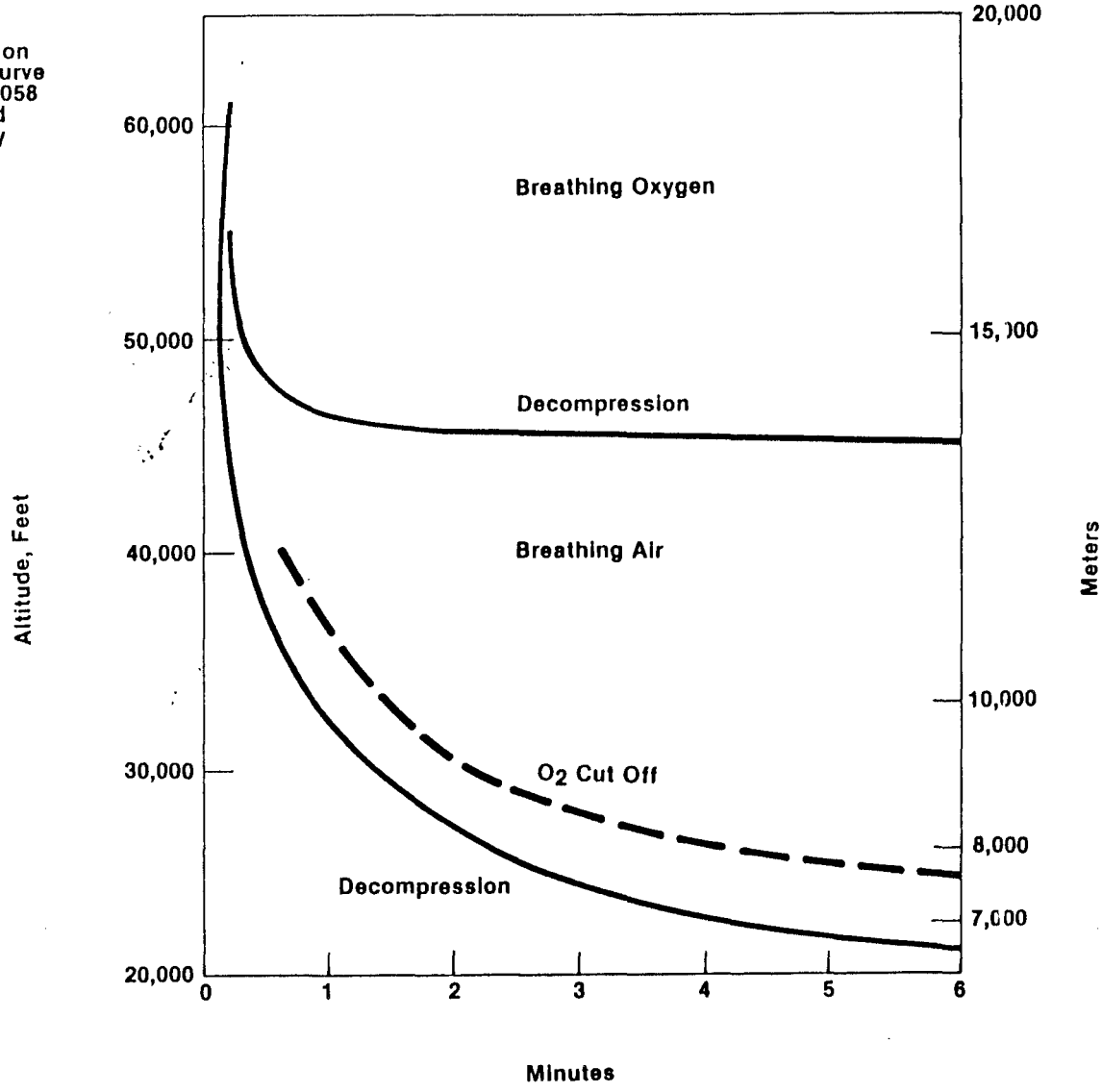
Drawing Ex # 8
10/2/91




ALTITUDE TIME HISTORY
SHIP 68-2-8
DATA SOURCE MADAR
4 APRIL 1976
NOTE: TIME 20 IS EQUIVALENT
TO MADAR TIME 6:18:18.89
WHICH WAS RAPID DECOMPRESSION
SIGNED
J W EDWARDS
ALTITUDE (FT)
24000
20000
16000
12000
8000
4000
0
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
TIME (MIN)
2 MIN PER INCH
1 MIN PER INCH
NOTE DISCONTINUITY
DUE TO DATA LOSS
DEFENDANT'S EXHIBIT
D 1215

Time of Useful Consciousness at Attitude

Solid Curve Below, After Rapid Decompression From Sea Level While Breathing Air. Solid Curve Above, After Rapid Decompression From 10,058 m (33,000 ft) Breathing 100% Oxygen. Dotted Curve, After Separation From Oxygen Supply While at Altitude. After Luft (50).



Downes Ex #10
10/7/81
FTR

UNIVERSITY OF PENNSYLVANIA - SCHOOL OF MEDICINE

Curriculum Vitae

JOHN J. DOWNES, M.D.

Home Address:	18 Merion Road Merion, Pennsylvania 19066
Office Address:	Department of Anesthesia The Children's Hospital of Philadelphia 34th Street and Civic Center Boulevard Philadelphia, Pennsylvania 19104
Date of Birth:	September 14, 1930
Place of Birth:	Pittsburgh, Pennsylvania
Marital Status:	Married - JoAnn Splon
	Children: Margaret Sarah July 7, 1957
	Katherine Rachel April 15, 1959
	John Matthew January 11, 1962
	Peter James March 12, 1964

Education:

1949	Campion Jesuit High School
1949-1951	Loyola University, Chicago, Illinois
1951-1952	B.S., St. Louis University, St. Louis, Missouri
1952-1956	M.D., Loyola University Stritch School of Medicine, Chicago, Illinois

Postgraduate Training and Fellowship Appointments:

1956-1957	Rotating Internship, Indianapolis General Hospital, Indianapolis, Indiana
1959-1963	Anesthesia Residency, University of Pennsylvania, Philadelphia, Pennsylvania
1961-1963	Clinical Pharmacology Fellowship, University of Pennsylvania, Philadelphia, Pennsylvania

Military Service:

1957-1959	U.S. Public Health Service, Division of Indian Health

Faculty Appointments:

1959-1961	Assistant Instructor in Anesthesia, Department of Anesthesia, University of Pennsylvania
1961-1963	Instructor in Pharmacology, Department of Pharmacology, University of Pennsylvania
1961-1966	Instructor in Anesthesia, Department of Anesthesia, University of Pennsylvania
1963-1966	Instructor in Pediatrics, Department of Pediatrics, University of Pennsylvania
1966-1971	Assistant Professor of Anesthesia, Department of Anesthesia, University of Pennsylvania
1966-1971	Assistant Professor of Pediatrics, Department of Pediatrics, University of Pennsylvania

Faculty Appointments (Continued):

1971-1973	Associate Professor of Anesthesia, Department of Anesthesia, University of Pennsylvania
1971-1973	Associate Professor of Pediatrics, Department of Pediatrics, University of Pennsylvania
1973-present	Professor of Anesthesia, Department of Anesthesia, University of Pennsylvania
1973-present	Professor of Pediatrics, Department of Pediatrics, University of Pennsylvania
1975-present	Fellow Faculty of Anesthesia, Department of Anesthesia, Royal College of Surgeons, Dublin, Ireland
1977-present	Fellow Faculty of Anesthesia, Department of Anesthesia, Royal Australasia College of Surgeons, Australia

Hospital and Administrative Appointments:

1963-1967	Assistant Anesthesiologist, The Children's Hospital of Philadelphia
1963-1967	Project Director and Co-Principal Investigator, U.S.P.H.S., Grant N.B. 04828
1965-1968	Project Director and Principal Investigator, U.S.P.H.S., Grant H.D. 01412
1967-1968	Associate Anesthesiologist, The Children's Hospital of Philadelphia
1967-1972	Director, Intensive Care Unit, The Children's Hospital of Philadelphia
1968-1970	Assistant Physician to the Hospital (Neonatalogy) Pennsylvania Hospital
1968-1970	Project Director, Studies of Acid-Base Metabolism, Abbotts Laboratories Grant
1968-1971	Co-Investigator, U.S.P.H.S., Grant H.D. 00201
1968-present	Senior Anesthesiologist, The Children's Hospital of Philadelphia
1969-1971	Project Director, Studies of Intravenous Isoproterenol in Asthma Winthrop Laboratories Grant
1972-present	Director, Department of Anesthesia, The Children's Hospital of Philadelphia
1973-1974	Co-Investigator, U.S.P.H.S. - National Institutes of Health Training Grant
1975-present	Co-Investigator, National Institutes of Health Head Trauma Center Grant, University of Pennsylvania and The Children's Hospital of Philadelphia

Committees:

1967-1971	Committee on Acute Medicine, American Society of Anesthesiologists
1967-1971	Program Chairman, Section of Anesthesiology, American Academy of Pediatrics
1968-1974	Secretary, Committee Z-79 (Equipment for Anesthesia and Resuscitation), American National Standards Institute

Committees (Continued):

1968-present	Member, American Delegation, International Standards Organization, Technical Committee 121
1969-1970	Committee on Respiration, American Society of Anesthesiologists
1970-1972	Chairman, Sub-Committee on Guidelines, Society for Critical Care Medicine
1971-1972	Chairman, Sub-Committee on Clinical Care, American Society of Anesthesiologists
1971-1977	Council, Society for Critical Care Medicine
1972-present	Member, Committee on Mechanical Equipment, American Society of Anesthesiologists
1974-1977	Chairman, Committee on Mechanical Equipment, American Society of Anesthesiologists
1975-1977	Chairman, American Delegation, International Standards Organization
1976-1977	Member, Committee on Pediatric Anesthesia, American Society of Anesthesiologists
1976-1977	Member, Committee on Respiration, American Society of Anesthesiologists
1977-1978	Secretary, Steering Committee of the Medical Faculty, University of Pennsylvania
1977-1980	Member, Anesthesiology Devices Advisory Board, Bureau of Medical Devices of the Food and Drug Administration, U.S.

Committees at The Children's Hospital of Philadelphia:

1965-1972	Intensive Care Unit Committee
1967-1969	Research Committee
1969-1970	Credentials Committee
1970-1971	Patient Care Committee, Chairman, Sub-Committee on Respiration
1971-present	Executive Committee of the Medical Staff
1972-1974	Facilities Planning and Coordination Committee
1975-1976	Vice President of the Medical Staff
1976-present	Chairman, Ad Hoc Committee on Brain Death
1978-1980	Secretary, Executive Committee of the Medical Staff
1980-present	President of the Medical Staff
1980-present	Chairman, Hospital Planning Medical Staff Advisory Group
1980-present	Quality Assurance Committee

Visiting Professorships:

April, 1969	University of California
February, 1970	University of West Virginia
February, 1971	Albert Einstein School of Medicine, New York
November, 1971	University of the West Indies, Jamaica
March, 1973	Stanford University, Palo Alto, California
March, 1975	University of Florida, Gainesville
May, 1975	Royal College of Surgeons, Dublin, Ireland
November, 1977	Case Western University, Cleveland, Ohio
August, 1977	Australian/New Zealand Anesthesia Societies
January, 1978	Johns Hopkins University, Baltimore, Maryland

Visiting Professorships (Continued):

February, 1978	Hershey Medical Center, Pennsylvania State University
November, 1978	McGill University, Montreal, Canada
March, 1979	Columbia University, New York, New York
May, 1979	University of Illinois, Chicago
June, 1980	Japanese Society of Anesthesiologists, Tokyo, Japan

Consultant Appointments:

1971-1972	Consultant in Anesthesiology, Project Hope, Washington, D.C.
1972-present	Consultant, National Center for Health Services, Research and Development, U.S.P.H.S.
1975-present	Residency Reviewer, American Medical Association - American Board of Anesthesiology
1975-present	Associate Examiner, American Board of Anesthesiologists
1976-1977	Consultant in Anesthesiology, Naval Regional Medical Center, Philadelphia, Pennsylvania
1977-present	Food and Drug Administration Panel Member, Bureau of Medical Devices, Anesthesiology Panel

Certifications and Licensure:

1957	Indiana State Board, #18134
1961	Pennsylvania, #6262-E
1963	Certified, American Board of Anesthesiology (3000)
1964	Fellow, American Academy of Pediatrics
1977	Diplomate, American Board of Anesthesiology
1977	Fellow, American College of Anesthesiology

Awards, Honors and Membership in Honorary Societies:

1956	The Mosby Medical Graduate Achievement Award
1956	Alpha Sigma Nu Scholastic Honorary Fraternity
1971	Association of University Anesthetists
1975	Fellow, Faculty of Anesthesia, Royal College of Surgeons, Ireland
1977	Fellow, Faculty of Anesthesia, Royal Australasian College of Surgeons, Australia
1979	Loyola University of Chicago, Pediatric Anesthesia Award

Memberships in Professional and Scientific Societies:

1961-present	American Medical Association
1961-present	Pennsylvania State and Philadelphia County Medical Societies
1964-present	American Society of Anesthesiologists
1964-present	Philadelphia Society of Anesthesiologists
1964-present	Sigma-Xi (Honorary Scientific Association)
1964-present	American Academy of Pediatrics
1967-present	American Association of University Professors
1968-present	American Association for the Advancement of Science
1970-present	Society for Critical Care Medicine (Founding Member)
1971-present	Association of University Anesthetists
1972-present	Philadelphia Pediatric Society
1977-present	College of Physicians of Philadelphia

BIBLIOGRAPHY:

Original Papers:

1. Downes, J.J.: Primary diptheritic otitis media. AMA Arch Otolaryng 70: 27-31, 1959.

2. Downes, J.J., et al: Apnea, suction, hyperventilation - effect on arterial oxygen saturation. Anesthesiology 22: 29, 1961.

3. Bonica, J.J., et al (including Downes, J.J.): Effects of surgical pneumothorax on pulmonary ventilation. Anesthesiology 22: 966, 1961.

4. Downes, J.J., Wood, D.W.: Mechanical ventilation in the management of status asthmaticus in children. Science and Practice in Anesthesia, J.B. Lippincott Co., Philadelphia, 1965.

5. Downes, J.J., Lambertsen, C.J.: Dynamic characteristics of ventilatory depression in man on abrupt administration of oxygen at 1.0 atmosphere. J. Appl. Physiol. 21: 447, 1966.

6. Downes, J.J., Wood, D.W., Striker, T.W.: Diagnosis and treatment: Advanced in the management of status asthmaticus in children. Pediatrics 38: 286, 1966.

7. Downes, J.J.: A physiologic basis for diagnosis and management of the respiratory distress syndrome. Inhalation Therapy 11: 84, 1966.

8. Downes, J.J., Striker, T.W.: Acute respiratory failure in infants and children with status asthmaticus, bronchiolitis, and pneumonitis. Acta Anesthesiologic Scand (Suppl) 23: 747, 1966.

9. Striker, T.W., Stool, S., Downes, J.J.: Prolonged nasotracheal intubation in infants and children. Arch. Otolaryng. 85: 106, 1967.

10. Downes, J.J., Kemp, R.E., Lambertsen, C.J.: Magnitude and duration of respiratory depression due to fentanyl and meperidine in man. J. Pharm. Exper. Therap. 158: 516, 1967.

11. Downes, J.J., Arya, S., Morrow, G., Boggs, T.R.: Transient respiratory distress syndrome in the newborn. Arch. Dis. Child 42: 659, 1967.

12. Bachman, L., Downes, J.J., Richards, C.C., Coyle, D., May, E.: The organization and function of an intensive care unit in a children's hospital. Anesthesia and Analgesia 46: 570, 1967.

13. Downes, J.J., Wood, D.W., Striker, T.W., Haddad, C.: Acute respiratory failure in infants with bronchiolitis. Anesthesiology 29: 426, 1968.

14. Downes, J.J., Wood, D.W., Pittman, J.C.: The arterial blood gas and acid-base disorders in infants and children with status asthmaticus. Pediatrics 42: 238, 1968.

BIBLIOGRAPHY (Continued):

Original Papers (Continued):

15. Wood, D.W., Downes, J.J., Lecks, H.I.: The management of respiratory failure in childhood status asthmaticus. Experience with 30 episodes and evolution of a technique. J. Allergy 42: 261, 1968.

16. Downes, J.J.: Resuscitation and intensive care in the newborn. International Anes. Clin. 6: 911, 1968.

17. Parks, C.D., Nicodemus, H., Downes, J.J., Waldhausen, J.A.: Changes in pulmonary vascular resistance following closure of ventricular septal defects. Circulation (Suppl) 39: 193, 1969.

18. Downes, J.J., Bachman, L.: Preoperative and postoperative care and cardiopulmonary resuscitation. In: Textbook of Pediatrics, Edited by W.E. Nelson (9th Edition), W.B. Saunders Co., Philadelphia, 1969.

19. Downes, J.J., Nicodemus, H.: Preparation for and recovery from anesthesia. Ped. Clin. N. Amer. 16: 601, 1969.

20. Nicodemus, H.F., Downes, J.J.: Alterations in ventilation associated with Tetralogy of Fallot. Anesthesiology 31: 265, 1969.

21. Stool, S.E., Downes, J.J., Wood, D.W.: Airway management. In: Practice of Pediatrics (Chapter 29), Edited by Brenneman, Harper and Row, Hagerstown, Maryland, 1969.

22. Downes, J.J., Nicodemus, H.F., Pierce, W.S., Waldhausen, J.A.: Acute respiratory failure in infants following cardiovascular surgery. J. Thor. Cardiovasc. Surg. 59: 21, 1970.

23. Downes, J.J., Vidyasagar, D., Boggs, T.R.: Respiratory distress syndrome of the newborn: A clinical scoring with acid-base and blood gas correlations. Clin. Pediat. 9: 325, 1970.

24. Vidyasagar, D., Downes, J.J., Boggs, T.R.: Respiratory distress syndrome of the newborn: Umbilical artery catheterization. Clin. Pediat. 9: 332, 1970.

25. Downes, J.J.: Mechanical ventilation of the newborn (editorial). Anesthesiology 34: 116, 1971.

26. Downes, J.J.: Pediatric and infant intensive care. Mod. Med. 39: 122, 1971.

27. Pierce, W.S., Raphaely, R.C., Downes, J.J., Waldhausen, J.A.: Cardiopulmonary bypass in infants: Indications, methods, and results in 32 patients. Surgery 70: 839, 1971.

BIBLIOGRAPHY (Continued):

Original Papers (Continued):

28. Wood, D.W., Downes, J.J., Lecks, H.I.: A clinical scoring system for the diagnosis of respiratory failure. Amer. J. Dis. Child 123: 227, 1972.

29. Downes, J.J., Fulgencio, T., Raphaely, R.C.: Acute respiratory failure in infants and children. Ped. Clin. No. Amer. 19: 423, 1972.

30. Downes, J.J., Chairman: Guidelines for Organization of Critical Care Units. Report of the Committee on Guidelines. JAMA 222: 1532, 1972.

31. Downes, J.J., Wood, D.W., Sheinkopf, H.N.: Effects of intravenous isoproterenol infusion in children with severe hypercapnea to status asthmaticus. Crit. Care Med. 1: 63, 1973.

32. Raphaely, R.C., Downes, J.J.: Congenital diaphragmatic hernia: Prediction of survival. J. Pediat. Surg. 8: 815, 1973.

33. Wood, D.W., Downes, J.J.: Intravenous isoproterenol in the treatment of respiratory failure in childhood status asthmaticus. Am. Allergy 31: 607-610, 1973.

34. Downes, J.J., Raphaely, R.C.: Pediatric anesthesia and intensive care. Penna. Med., 1973.

35. Aberdeen, E., Downes, J.J.: Artificial airways in children. Surg. Clin. No. Amer. 54: 1155-1170, 1974.

36. Downes, J.J.: Intensive post-operative care for infants and children following cardiovascular operations. In: Proceedings of USA-USSR Joint Symposium on Congenital Heart Disease. Edited by D. Sharma, DHEW Publication No (NIH) 74-613,

37. Downes, J.J.: Basic Principles in the Use of Respirators. Chapter in The Anesthesiologist, Mother, and Newborn. Edited by F. Moya, Williams and Wilkins Co., Baltimore, 1974.

38. Downes, J.J., Raphaely, R.C.: Pediatric intensive care. Anesthesiology 43: 238-250, 1975.

39. Edmunds, L.H., Downes, J.J.: Assisted ventilation in infants. Chapter 9 in Gibbon's Surgery of the Chest. Edited by D. Sabiston, F. Spender, Third Edition, W.B. Saunders Co., Philadelphia, 1975, pp 224-238.

40. Wood, D.W., Downes, J.J.: The management of respiratory failure in childhood status asthmaticus. Advances in Asthma and Allergy, 1975.

BIBLIOGRAPHY (Continued):

Original Papers (Continued):

41. Fox, W.W., Berman, L.S., Downes, J.J., Peckham, G.J.: The therapeutic application of end-expiratory pressure in the meconium aspiration syndrome. Pediatrics 56: 214, 1975.

42. Berman, L.S., Fox, W.W., Raphaely, R.C., Downes, J.J.: Optimum levels of CPAP for tracheal extubation of newborn infants. J. Pediatrics 89:109-112, 1976.

43. Downes, J.J.: CPAP and PEEP, a perspective. Anesthesiology 44:1-5, 1976 (Editorial).

44. Downes, J.J.: Newborn respiratory care. ASA Refresher Courses, Volume 2, J.L. Lipincott, Philadelphia, 1977.

45. Schwartz, A.J., Downes, J.J.: Hazards of a simple monitoring device - the esophageal stethoscope. Anesthesioogy 47: 64-65, 1977.

46. Downes, J.J., Betts, E.K.: Anesthesia for the critically ill infant. Refresher Courses in Anesthesiology, 5: 47-70, 1977.

47. Betts, E.K., Downes, J.J., Schaffer, D.B., Johns, R.: Retrolental fibroplasia and general anesthesia. Anesthesiology 47: 518-520, 1977.

48. Downes, J.J., Goldberg, A.I.: Airway management, mechanical ventilation, and cardiopulmonary resuscitation. Chapter in Pulmonary Disease in the Newborn Fetus and Child. Edited by E. Scarpelli, R. Auld, Lea and Febiger, Philadelphia, 1978.

49. Downes, J.J., Raphaely, R.C.: Pediatric anesthesia and intensive care. Chapter in Pediatric Surgery. Edited by M. Ravitch, Year Book, Chicago, 1979.

50. Downes, J.J., Raphaely, R.C.: Pre-operative and post-operative care and cardiopulmonary resuscitation. Chapter in Nelson's Textbook of Pediatrics. Edited by V.C. Vaughn, R.J. McKay, 11th Edition, W.B. Saunders Co., Philadelphia, 1979.

51. Downes, J.J., Godinez, R.I.G.: Acute airway obstruction in the child. Refresher Course Lectures in Anesthesiology, 8: 29-48, 1980.

52. Loeber, N.V., Downes, J.J.: Pulmonary function in chronic respiration failure in infancy. Crit. Care Med. 8: 596-601, 1980.

53. Harrington, J., Raphaely, R.C., Downes, J.J.: Pathophysiology of respiratory failure in congenital diaphragmatic hernia. In press. Anesthesiology, 1981

BIBLIOGRAPHY (Continued):

Original Papers (Continued):

54. Downes, J.J., Raphaely, R.C.: Pediatric intensive care. Chapter in Fundamentals of Anesthesiology. Edited by C.P. Larson, H. Wollman, W.B. Saunders Co., Philadelphia, In press, 1980.

56. Downes, J.J.: Management of respiratory failure in children with status asthmaticus. Loyola University of Chicago Pediatric Anesthesia Symposium. Academic Press, New York, 1980, in press.

57. Loomis, J., Rosen, J., Downes, J.J.: Intravenous isoproterenol in children with acute hypercapnia due to status asthmaticus - experience in 103 episodes. In preparation, 1981.

58. O'Keeffe, C.M., Bruce, D.A., Downes, J.J.: ICP and cardiovascular function in pediatric head trauma. Anesthesiology 51:5270, 1979

59. Keon, T.P., Downes, J.J.: Cardiovascular effects of relaxants in infants. In preparation, 1981.

60. Lee, K.W., Templeton, J.J., Dougal, R.M., Downes, J.J.: Selection of endotracheal tubes in infants and children. In preparation, 1981.

61. Morray, J.P., Fox, W.W., Kettrick, R.G., Downes, J.J.: Clinical correlates of successful weaning from mechanical ventilation in severe bronchopulmonary dysplasia. Submitted for publication, 1980.

62. Morray, J.P., Fox, W.W., Kettrick, R.G., Downes, J.J.: Improvement in lung mechanics as a function of age in the infant with severe bronchopulmonary dysplasia. Submitted for publication, 1980.

63. Raphaely, R.C., Swedlow, D.B., Downes, J.J., Bruce, D.A.: The management of severe pediatric head trauma. Ped. Clin. N. Amer. 27:3, 1980.

64. Morray, J.P., Fox, W.W., Kettrick, R.G., Downes, J.J.: Pulmonary function in bronchopulmonary dysplasia (BPD). Crit Care Med 8:228, 1980.

BIBLIOGRAPHY (Continued):

Abstracts:

1. Downes, J.J., Lambertsen, C.J.: The magnitude and time course of transient respiratory depression by oxygen at controlled $PaCO_2$. Fed. Proc. 23: 259, 1964.

2. Gelfand, R., Lambertsen, C.J., Downes, J.J.: Use of CO_2 induced respiratory transients in analysis of respiratory control mechanisms. The Physiologist 7: 211, 1964.

3. Downes, J.J., Wood, D.W., Nightingale, D.A., Bachman, L.: Arterial blood gas and acid-base derangements in children with status asthmaticus. Anesthesiology 26: 245, 1964.

4. Downes, J.J., Arya, S.C., Morrow, G., Boggs, T.R.: Transient respiratory distress in premature newborns. J. Pediat. 69: 964, 1966.

5. Downes, J.J., Johnson, L., Arya, S.C., Boggs, T.R.: Energy substrates in the respiratory distress syndrome. J. Pediat. 69:968, 1966.

6. Wood, D.W., Downes, J.J., Lecks, H.I.: Hypoxemia caused by perfusion non-ventilated lung in children with status asthmaticus. Abstracts of the Society for Pediatric Research, 1966.

7. Vidyasagar, D., Downes, J.J., Johnson, L., Boggs, T.R.: Energy substrates and acid-base balance in the normal premature infant. Pediatric Research 1: 219, 1967.

8. Haddad, C., Downes, J.J., Richards, C.C.: Effect of mechanical ventilation on the physiologic dead-space of infants with normal lungs. Anesthesiology 29: 294, 1968.

9. Pannathpur, J., Downes, J.J., Vidyasagar, D., McNair Scott, T.F. Boggs, T.R.: Long term study of respiratory distress syndrome (RDS) of the newborn. Abstracts of the Society for Pediatric Research, 1968.

10. Downes, J.J., Wood, D.W., Sheinkopf, H.N.: The effects of intravenous isoproterenol infusion in children with severe hypercapnia due to status asthmaticus. American Society of Anesthesiologists Annual Meeting, October, 1971.

11. Oh, T.H., Downes, J.J.: Comparison of intemittent positive pressure breathing (IPPB) and powered nebulizer to administer racemic epinephrine in croup. American Society of Anesthesiologists Annual Meeting, October, 1977.

12. Betts, E.K., Downes, J.J., Schaffer, D.B., Johns, R.: Retrolental fibroplasia and O_2 administration during general anesthesia. American Academy of Pediatrics Annual Meeting, New Orleans, April 1977.

BIBLIOGRAPHY (Continued):

Abstracts (Continued):

13. Betts, E.K., Downes, J.J., Morriss, F., Ellison, N.: Effects of hemodilution in children undergoing correction of scoliosis. American Society of Anesthesiologists Annual Meeting, New Orleans, October, 1977.

14. Loomis, J.C., Raphaely, R.C., Downes, J.J.: Treatment with intravenous isoproterenol infusion in 107 episodes of refractory status asthmaticus in children. American Society of Anesthesiologists Annual Meeting, Chicago, October 1978.

15. Harrington, J.F., Raphaely, R.C., Downes, J.J.: Relationship of cardiopulmonary shunting to survival in diaphragmatic hernia of the newborn. American Society of Anesthesiologists Annual Meeting, Chicago, October, 1978.

16. Morray, J.P., Kettrick, R.G., Downes, J.J.: Clinical correlates of successful weaning from mechanical ventilation in severe bronchopulmonary dysplasia (BPD). Third World Congress on Intensive and Critical Care Medicine, Washingon, D.C., May, 1981.

17. Kettrick, R.G., Downes J.J., Donar M.E.: Transfer of life support systems to the home in ventilator-dependent children. Third World Congress on Intensive and Critical Care Medicine, Washington, D.C., May, 1981.

18. Loeber, N.V., Downes, J.J.: Lung function in chronic respiratory failure in infancy. Anesthesiology 51:S328, 1979.