Materials Reviewed in Preparation

- many recently-acquired photographs, in particular those in color, of the accident scene / wreckage etc.
- two films, one short, one long, of the accident scene / wreckage, etc
- a letter from John J. Downes, M.D. to Mr. John Connors, dated October 5, 1981
- a report from James G. Caume, M.D. to Mr. Carroll E. Dubuc, dated August 31, 1981
- wreckage diagram - updated
- a report from C. Keith Connors, Ph. D., to Mr. Charles R. Wark.
- an "Accident Report" unsigned and undated, but recently prepared in the offices of Lewis. Wilson, Lewis and Jones.
- personal notes relating to pressure differentials that could have built up in the infant's lungs during the decompression
- personal notes relating to partial pressures of oxygen in the lung that could have resulted from the decompression, and subsequantly.
- the reference made by Dr. Downes to the Luft chapter in Armstrong
- the "locations diagram" - defendant's exhibit D1210
- report of Mr. John W. Edwards (partially)

DEFT.EX. ~~DD~~ Busby Dep Exh. 1
DATE: 12/18/81
REPORTER: ALBERT J. GASDOR

Accident Report

On April 4, 1975 a Lockheed C-5A aft ramp together with the attached pressure door failed at approximately 23,000 ft. The structural failure resulted in a sudden decompression of the aircraft, and partial loss of control by the cutting of the number 1 and 2 hydraulic lines, the control cables to the tail, and the alternate electric trim and rudder yaw, Even though number three hydraulic system was not damaged, primary pitch and yaw control were lost due to damage to the control cables to the tail. Using the remaining controls available to them, right aileron, spoilers, and engine thrust; the pilots were able to maintain control of the aircraft by a combination of banking aircraft and thrusting the engines in order to keep a quasi-level descent for an emergency landing. A quasi-level descent being a series of dives and pull-ups until the aircraft was at landing altitide. The aircraft approached its first touch down point on the east side of the Saigon River. Just before touch down the engines were at full throttle to reduce the descent rate and the aircraft was at a slight roll angle. As the aircraft was touching down the engines were retarded to idle. The velocity of the aircraft as recorded by the MADAR DATA was approximately 270 knots (456 ft./sec.) approximately 3-4 seconds. The average wind velocity recorded at Saigon Airport at the time of the crash was 15 knots. The direction of the wind was approximately to the west, therefore the ground airspeed of the C-5A was about 283 knots (478 ft./sec.). The velocity is about 2-1/2 times the aircraft's normal landing velocity. It will be demonstrated later that the C-5A came to a complete stop in a shorter distance (~1900 ft.).

283 K → 1900'
110 K → 2300'

than it does when it lands at its normal landing velocity (~2300 ft.). Therefore the aircraft impacted at 283 knots and stopped in approximately 1900 ft. A normal landing would be at approximately 110 knots. (190 ft./sec.) and would stop in about 2300 ft.

Impacts on the East Side of the Saigon River

As the C-5A approached its first impact point it had a velocity of 283 knots and the pilots had limited control of the aircraft. There was no record of the descent speed. The initial impact occurred when the aft landing gear struck a dike. The aircraft still lofting above the ground struck another dike this time more severely than the first. The landing gear dug into the soil for a short distance. The aircraft bounced up again and then settled back down hitting a third dike. The landing gear again dug into the soil. It is probable that two complete sets of landing gear were lost during or shortly after this impact. Photographs show wheels and pieces of the landing gear spread throughout this area. The C-5A bounced up again and made several small ruts with its engines or wing tips. The aircraft then hit another dike. Finally the C-5A became airborne again slicing several treetops off with its starboard wing. From the films it appears that there were at least eight or more distinct impact points east of the Saigon River. These multiple impacts all occurred in a distance of about 350 yards. The impacts were of sufficient magnitude (snapped off several pieces of landing gear) to have weakened part or all of the C-5A structure.

Impacts on the West Side of the Saigon River

The C-5A crossed the Saigon River at a probable velocity of 283 knots. This velocity may not be precisely relied upon and is believed to somewhat less because of the series of impacts encountered on the east side. The angle of attack of the aircraft (nose up or down) also cannot be estimated because of the lack of in-flight data. It is to be emphasized that the pilots had no control of the C-5A during any of the impacts.

The aircraft impacted on the west side of the Saigon River breaking the remainder of its landing gear off. The C-5A went into a sliding skid for about 175 yards. After this point the skid marks disappeared indicating that the aircraft may have lifted off the ground. The C-5A traveled about 150 yards and broke into four separate sections: the T-tail, the aft troop compartment, the flight deck and the complete wing structure. At this point of impact, large amounts of debris were found and a large section of the cargo floor was located. This is also the area where almost all of the dead were located. Northwest and about 100 yards away from the last impact point, the T-tail was found. The T-tail had a clean fracture indicating a sudden separation from the fuselage. It appears the tail was thrown over to its location as a result of the impact. The flight deck moved in a south-west direction and traveled approximately 400 yards from the impact. It appears that the flight deck traveled about 150 yards in the air and skidded to a stop in the remaining 250 yards. The wing structure also detached during the impact and through a combination of inertial (96,000 lbs. fuel) and lift forces was propelled approximately

525 yards from the point of last impact. The aft troop compartment became detached from between the wing section and the T-tail, and was propelled from the impact primarily by inertial forces and possibly some lift force. The troop compartment began digging into the ground approximately 175 yds. from the point of last impact. The aft troop compartment then came to a sudden stop after hitting an elevation. The total distance the troop compartment dug into the ground was approximately 2 lengths of the structure or about 40 yards.

The velocity of the four major sections were equal at the point of break-up. The velocity at this point has been estimated (see Appendix I) as 200 knots, (338 ft./sec.). The estimated "G" forces for the aft troop compartment, flight deck and the T-tail are summarized below (see Appendix I for details):

1. T-tail: After close examination of the photographs, it was concluded that the T-tail had been sheared off during the last impact. The minimum "G" force range required to break the tail off according to our engineering analysis with data from Lockheed reports is 11 to 15.

G break 11-15

2. Flight Deck: Given the initial velocity as 200 knots, and the measured slide path of the flight deck, an average "G" force range of 6-8 has been estimated.

Average G 6-8

3. Aft Troop Compartment: The aft troop compartment had an initial height at the point of break-up. The height combined with a velocity of 200 knots turned the aft troop compartment into a projectile. The troop compartment was airborne as indicated by the photographs for approximately 175 yards, and smashed down onto the ground at the end of its trajectory. The average vertical "G" force range was estimated to be 10_6-30_6. The aft troop compartment then started digging into the ground and came to a sudden stop by hitting a small hill. The average estimated horizontal "G" force range during the deceleration was 7-13. At the point of impact with the hill, the estimated horizontal "G" force range was 220 to 480. It is obvious from the engineering analysis that the "G" force environment in the aft troop compartment was extremely complex and severe.

Troop compt
175 yards
airborne
vert g 10-30
Horiz g 7-13
Strike hill 220-480g

Summary

In conclusion the C-5A had an approach speed of 2-1/2 times its normal landing speed. The pilot only had limited control of the aircraft before the crash and no control during the crash landing. The C-5A structure experienced a series of 8-12 impacts, some sufficiently severe to break off landing gear, on the east side of the Saigon River. Approaching the west side of the river the aircraft had a velocity of approximate-

bounced into the air again and impacted again about 400 feet away, breaking up into four major sections, each moving at a velocity of approximately 200 knots. The aft troop compartment experienced a severe and extremely complex "G" force environment. The engineering analysis of Appendix I demonstrates the complexity involved in attempting to calculate the "G" force environment. The assumption of constant deceleration over the complete crash site cannot be used. The average "G" force ranges in Appendix I are all based on conservative assumptions. Peak "G" forces, greater than what is calculated, undoubtly exist; but cannot be calculated.

Appendix I

Estimation of Deceleration Levels

In order for some of the passengers and crew to survive the crash, the airframe and ground had to absorb the energy of the airframe/passengers/crew in a manner that made their survival possible while removing the danger of a post landing fire. This energy absorbtion was accomplished over several definable periods of time/distances. One method of characterizing the events that occurred during the time from aircraft touchdown to points where the various parts came to rest would be to develop the deceleration time history. As indicated by the location of the major parts in the photographs of the crash site, the aircraft was subjected to complex set aerodynamic, inertia, and frictional forces. These complex forces thus would yield an equally complex deceleration time history. Since only the final position and an estimation of the initial conditions are known, it is not possible to evaluate but the simplest assumptions (constant deceleration) without some additional data. Even though not valid, the assumption of a constant deceleration for both the flight deck and aft troop compartment may be made after the aircraft breaks up. This assumption will yield a lower bound on the estimated maximum "G" load. Since only the initial conditions are known, additional data (structural failure) is needed to determine the intermediate conditions. Use of structural failures will only yield a lower bound on the applied loads/maximum "G's" since the rate of failure is not known.

The first step is to determine the form of the deceleration while the aircraft is intact, thus developing lift. The deceleration force is given by:

$$F = \mu (W-L) + D - T - D_p$$

Where: μ = Coefficient friction

W = Weight

L = Lift = $1/2\rho V^2 C_L S$

D = Drag = $1/2\rho V^2 C_d S$

T = Thrust

D_p = Drag of landing gear post

ρ = Density of air

v = Velocity

C_L = Coefficient of lift

C_d = Coefficient of drag

S = Wing area.

The deceleration is given by:

$$a = 1/m\{F\} = 1/m\{\mu(W-L) + D - T - D_p\}$$

where : m = mass of aircraft

The deceleration can be written as:

$$\frac{dv}{dt} = a$$

or :

$$\frac{m \cdot dv}{\mu (W-L) + D - T - D_p} = dt$$

since L, D and D_p will be a function of velocity. The aircraft may be pitching, therefore both C_L and C_d may be a function of time, but they are assumed to change much slower than the velocity. Intergrating the above will allow an estimation of the form of deceleration versus time. A cubic variation of deceleration with time would be a good approximation for the above equation using the stated assumptions:

$$\frac{dv}{dt} = a = ct^3$$

where c = constant

yielding:

$$V_F - V_I = \frac{ct^4}{4}$$

where V_F = final velocity

V_I = Initial

and,

$$S_F - S_I = \frac{ct^5}{20} + V_I t.$$

where S_F = final position

S_I = Initial position

Next, the equations can be developed for the region in which a constant deceleration is to be assumed, as with the structural failure which is to be used with the above equation, this assumption will yield a lower bound on the maximum "G" estimate. For this assumption:

$$\frac{dv}{dt} = a$$

yielding:

$$V_F - V_I = at$$

and,

For the above formulations the distances are obtained from the referenced reports, velocities from referenced reports and calculations. The calculation of an intermediate velocity is made by assuming a minimum deceleration to fail parts of the structure. This must be done since there is one more unknown than equations available. The tail failure was selected because the normal flight loads on the tail are seen as bending movements/axial loads on the fuselage. The deceleration loads are also seen as bending movements/axial loads on the fuselage, thus, the loads needed to fail the fuselage are known (Figure I and Lockheed Reports). Since the axial force is now higher, several calculations on the magnitude of the failure load were made. The calculated "G's" for the failure load is 13 plus or minus 2. For the estimate "G" an intermediate velocity of 360 ft./sec. plus or minus 20 ft./sec. is obtained (Figure II). The aft troop compartment will require about one and a half seconds to travel from the break up point to its final touch down point, about the same time to travel from its break up height to the ground. At the point of final impact the aft troop compartment will experience 10-30 vertical maximum "G's". The horizontal maximum "G's" will depend on the method used. If it is assumed that the aft troop compartment would have gone as far as the forward flight deck if the hill had not been present, then the average "G's" would have been about 7 (Figure III) with a much higher "G" level when the hill is impacted, (Figure IV). If the average "G" is calculated then the "G" is about 13. Since some of the seats containing children failed, it is possible to estimate a level of horizontal "G" loading in the aft troop compartment. Depending on the weight of the children, the horizontal loading would range from

Summary

Within the assumptions made, the following maximum "G" loadings have been calculated, these would be mimimum values since the time rate of failure is not known.

Horizontal

11 - 15 before break-up (use of structural failure)

7 - 13 after break-up (integration)

60 - 80 after break-up (use of structural failure)

Within the assumptions made, the following "G" loadings has been estimated for various impacts.

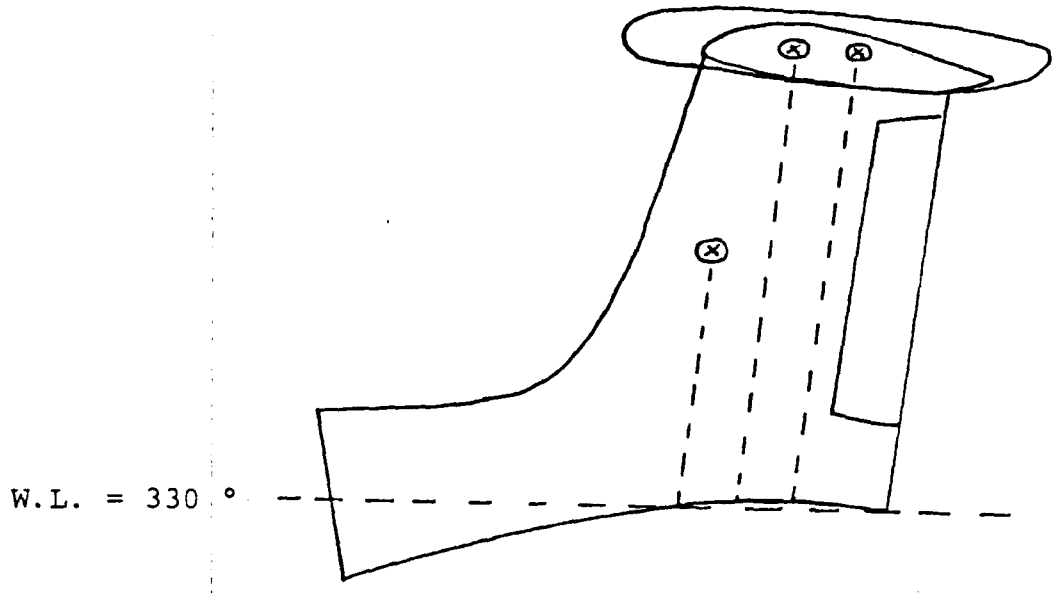
Horizontal

60 - 80 after break-up (use of structural failure)

220 - 480 after break-up (integration)

Vertical

10 - 30 after break-up (integration)



Item	Weight	$\bar{X}$	$\bar{Y}$	$\bar{Z}$	Mass
Bullet	769.4	2895.6	0.0	786.6	23.9
H.S.	3275.4	2921.3	140.1	787.5	101.7
V.S.	6151.0	2786.6	-0.1	633.1	191.02

$F = m(a) = [\ 23.9 + 203.4 + 196]\ a = 428.3(a)$ 15.

$M = 23.9\ (456.6)\ a + (2)\ (101.7)\ (457.5)\ a + (191.02)\ (303.1)a$

$M = 1.619 \times 10^5\ (a)$

Figure I - Sample Calculation

Deceleration given by

$$a = ct^3$$

where $a = -13$ "G's" $= -416$ ft./sec^2

$$V_F - V_I = \frac{ct^4}{4} = \frac{-416t}{4} = -104t$$

$$V_F = V_I + \frac{ct^4}{4}$$

$$S_F - S_I = V_I t + \frac{ct^5}{20} = V_I t - \frac{416t^2}{20}$$

$$-20.8t^2 + V_I t - (S_F - S_I) = 0$$

$$t = \frac{-V_I \pm \sqrt{(V_I)^2 - (4)(20.8)(S_F - S_I)}}{(-20.8)(2)}$$

, where $S_I = 0$

$S_F \approx 500$ ft.

$V_I \approx 463$ ft./sec,

$$t = \frac{V_I \pm \sqrt{(V_I)^2 - (4)(20.8)(S_F)}}{41.6}$$

$t \approx 1.2$ sec. $V_F \approx 360$ ft./sec.

Seat is designed to transfer:

2500 lb horizontal

1250 lb vertical

375 lb lateral

to the floor beam without failing. For seat to fail in the horizontal direction with children, the "G" would be given by

$$G = \frac{2500}{(N)W_c}$$

N = number of children

W_c = Weight per child.

$$G = 60 - 85,$$

actual value would depend on the weight of the children. Note: the moment arm has not been adjusted for the children so the above estimates would be less than the actual values.

Figure V - Sample Calculations

$$"G" = \frac{(V_I)^2}{64.4(\Delta X)}$$

ΔX = penetration into hill

ΔX = 2, 3, 4 ft.

then $"G_2" = 460 \pm 20$

$"G_3" = 320 \pm 20$

$"G_4" = 240 \pm 20$

$$t_2 = \frac{V_I}{(32.2)\ "G_2"} =$$

$$t_3 = \frac{V_I}{(32.2)\ "G_3"} =$$

$$t_4 = \frac{V_I}{(32.2)\ "G_4"} =$$

Figure IV - Sample Calculations

$$V_F - V_I = at \qquad \text{, where } V_F = 0$$

$$V_I = 360$$

$$S_F - S_I = \frac{at^2}{2} + V_I t \qquad S_F \approx 750 \text{ ft.}$$

$$S_I = 0 \text{ ,}$$

$$S_F - S_I = \frac{a}{2} \frac{V_I^2}{a^2} - \frac{V_I^2}{a}$$

$$a = \frac{-(V_I)^2}{(2)(S_F - S_I)}$$

Deceleration would be $7.0 \pm .5$

$$t = \frac{V_I \pm \sqrt{(V_I)^2 + 2a(S_F - S_I)}}{a}$$

$$t \approx 0.5 \text{ .}$$

Velocity at Impact : $V_F = V_I + at$

$$V_F = 250 \text{ ft./sec.} \pm 10 \text{ ft./sec.}$$

Figure III – Sample Calculations

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
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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)

check these with Cohen

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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

this is an adapted data line

get reference

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:

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

in which F_IO_2 is 0.2094, R is 0.79

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

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

(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.

Name: [illegible] 10/2/81

Topic: Average Alveolar Partial Pressures: Acute Decompression & Altitude

Altitude (ft)	P_B	P_{IO_2}	Adapt (+)(1) $P_{A_{O_2}}$	Adapt (+)(1) $P_{A_{CO_2}}$	Infant/Child(2) $P_{A_{O_2}}$	Infant/Child(2) $P_{A_{CO_2}}$	Time (m)
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	102	35?	

↖ adapted levels?

Calculate altitude basis decompression: 5,000 ft.

Calculation based on: $P_{A_{O_2}} = (P_B - 47)(F_{IO_2}) - P_{A_{CO_2}}\left[F_{IO_2} + \frac{1 - F_{IO_2}}{R}\right]$

in which F_{IO_2} is 0.20#,

R is 1.0 ?

At 23,400 $\begin{cases} P_{A_{O_2}} = (303 - 47)(.21) - 28\left(.21 + \frac{1-.21}{0.79}\right) \\ = 53.7 - 28.2 \\ = 25.6 \end{cases}$

At 20,000 $\begin{cases} P_{A_{O_2}} = (349 - 47)(.21) - 25\left(.21 + \frac{1-.21}{.79}\right) \\ = 63.4 - 25.2 \\ = 38.2 \end{cases}$

(1) Luft, UC: Altitude tolerance (6.1) in
Dinsdale, HB (ed): Aerospace [illegible]
Williams & Wilkins, Baltimore, MD, pp 125-142
as cited in Kendal HW (ed): [illegible], 1971) p. 62

(2) Dalton [illegible] Albert MS, Winter PW Pediatrics 37: 728, 1966
[illegible] EP Jr. Tr: [illegible] 101: 274, [illegible]

Table 2 Estimated (Average) [illegible] in Children [illegible]
During Acute [illegible]

(Patient / [illegible] Values)

Altitude	Time	$P_{A,a}CO_2$	$P_{A}O_2$	$P_{a}O_2$	$P_{\bar{v}}O_2$	S_aO_2	$S_{\bar{v}}O_2$	C_aO_2	$C_{\bar{v}}O_2$	$C_{a-\bar{v}}O_2$
ft	min	mm Hg	mm Hg	mm Hg	mm Hg	%	%	ml/dl	ml/dl	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	4:10 7:00	22	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

[illegible] Hb = 10 gm/dl
Hb Cap O_2 = 1.34 ml/gm

[illegible] pH 1.52 } [illegible]
[illegible] 7.41 }
[illegible] 25 7.41 } [illegible]
[illegible] 13 7.4 }

$CaCO_2$ 17.5 (17.5-22.
$C_{\bar{v}}CO_2$ 48.2 (46-52)
$C_{\bar{v}}O_2$ 10.7 (10-16)

[illegible] Hgb was 18-23 Bl/dm

JAMES G. GAUME, M.D.
CONSULTANT IN HUMAN FACTORS
1517 ESPINOSA CIRCLE
PALOS VERDES ESTATES, CALIFORNIA 90274

(213) 375-6607

August 31, 1981

DEFT.EX. ~~DD~~-Busby Dep Exh. 2/
DATE: 12/18/81
REPORTER: ALBERT J. GASDOR

Carroll E. Dubuc, Esq.
KNIGHT, 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.

evidence

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

DEFENDANT'S EXHIBIT
D 1302

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 not 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 Aerospace Medicine, by Armstrong, a recognized 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

APPENDIX A

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 truck and the speed traveled the rates of onset are usually in excess of 3g's per second.

note: no jolt calcs

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

doesn't really go into rates of onset!

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

JAMES G. GAUME, M.D.
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Doesn't address jolt.

REPORT A

ANALYSIS OF THE ACCELERATIONS INVOLVED IN THE DECOMPRESSION AND CRASH LANDING EVENT OF CSA 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 mixiumum 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)

- jolt not considered

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

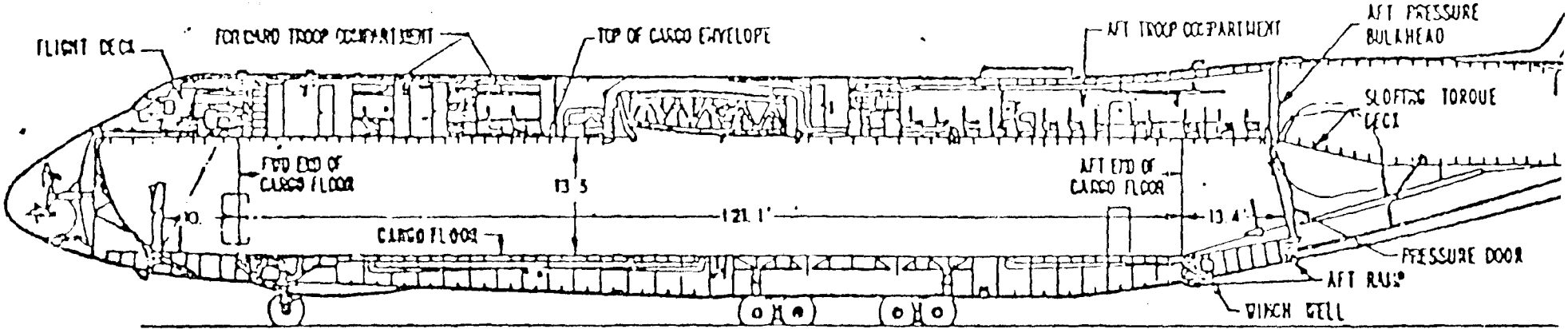
Analysis of Accelerations
C5A SN68-218, 4 April 1975

James G. Gaume, M.D.

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


FLIGHT DECK
FOR CARGO 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
DITCH WELL

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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.

5-10

?

James G. Gaume, M.D.

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

ATTACHMENT B-1, J.C.G.

old
what about
1971 edition!!

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

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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 effects 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

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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

Analysis of Hypoxia
C5A SN68-218, 4 April 1975

James G. Gaume, M.D.

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

Analysis of Hypoxia
C5A SN68-218, 4 April 1975

James G. Gaume, M.D.

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.
Human Factors Consultant
30 August 1981

ATTACHMENT C-1, J.G.G.

LAB DATA SHEET

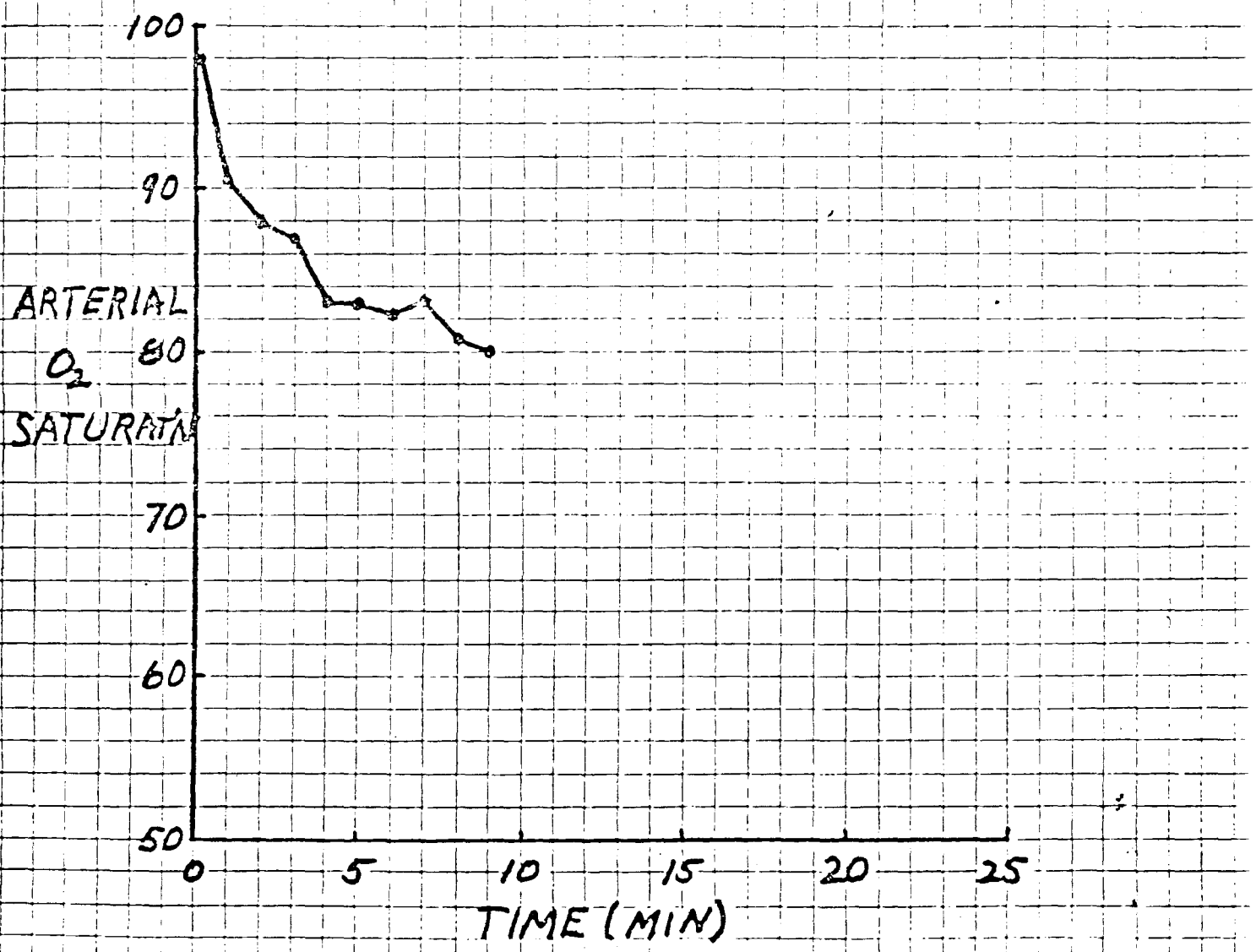
NAME: JGG. AGE: 65

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.



LAB DATA SHEET

NAME: J.G.G. AGE: 65

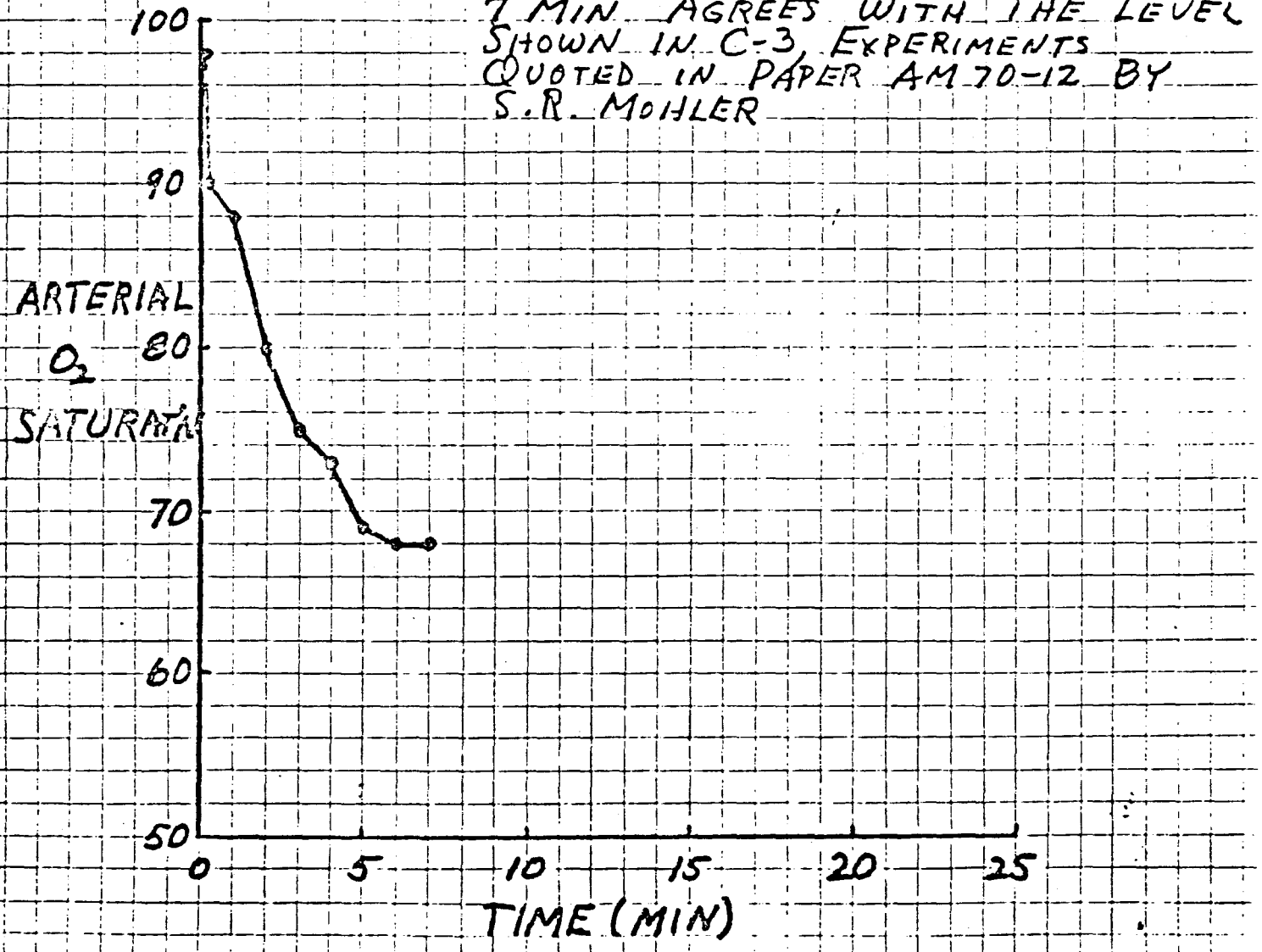
DATE: 4-6-81

HOUR: 1230

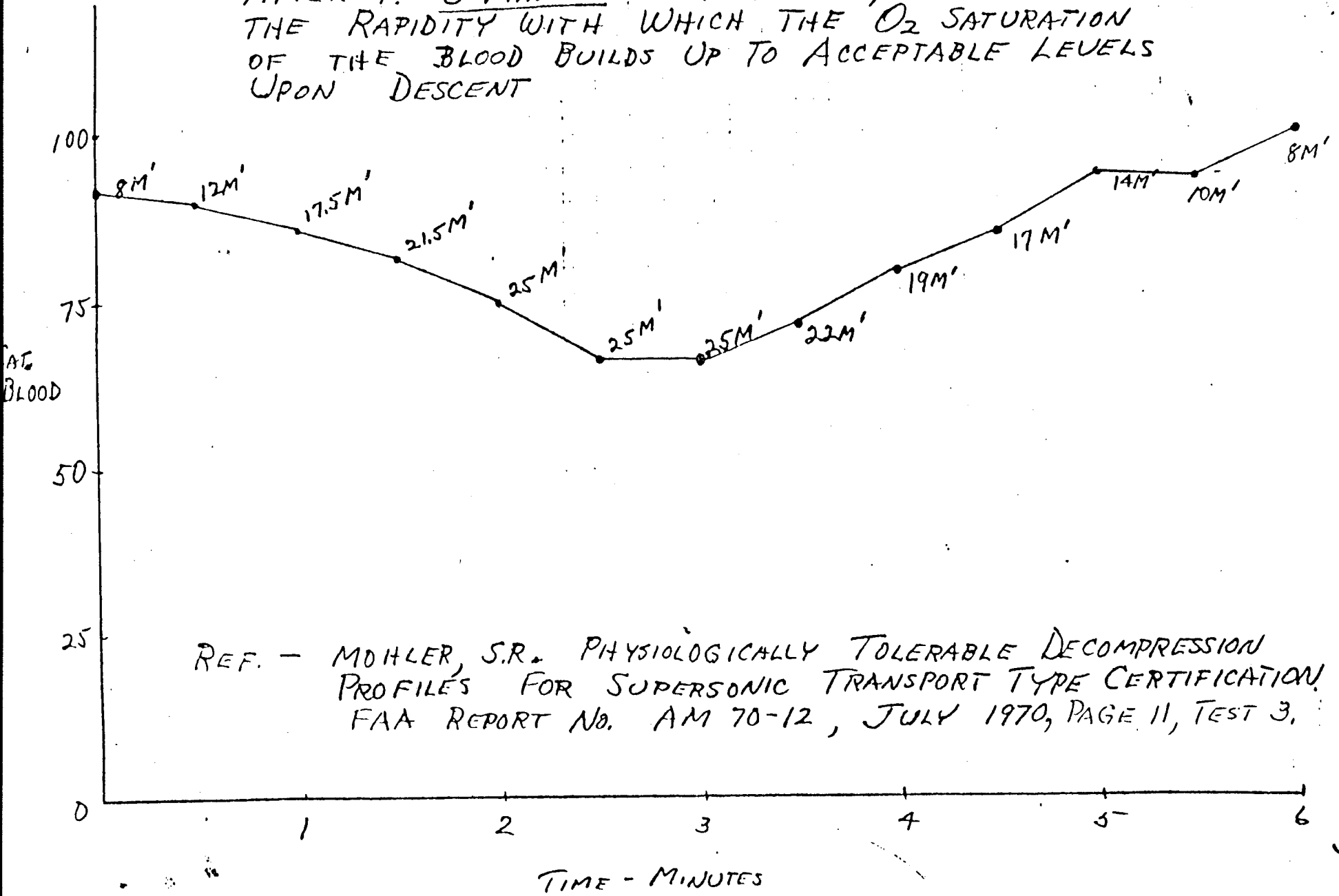
GAS MIXTURE: 33% ROOM AIR (7.5% O_2 EQUIVALENT)
67% N_2 (92.5% N_2)
24,000 FT. EQUIVALENT ALTITUDE

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.

ATTACHMENT C-3, J.G.G.

James G. Greene, 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

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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

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HYPOXIA

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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)[1] 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); Cirile 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

Anoxic 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 phosphocreatine 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, 101), on cats (35, 64, 90, 101), 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 level and the infant's ability to metabolize lactic and pyruvic acids to lipids.

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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.

APPENDIX I

For uniform (constant) deceleration the governing equation is:

$$G = \frac{V^2}{64.4S}$$

where:

V = Velocity in ft/sec
= 270 knots = 456 ft/sec

S = Deceleration distance = 1950 feet

The constant 64.4 is twice the acceleration due to gravity or $2g = 2 \times 32.2 = 64.4$ ft/sec^2.

Then:

$$G = \frac{(456)^2}{64.4\ (1950)} = 1.66$$

The 'Wreckage Diagram' for C-5A SN 68-218 shows a deceleration distance for the troop compartment of about 650 yards or 1950 feet as scaled from the diagram. For an initial speed of 270 knots or 456 ft/sec the average deceleration over this distance is 1.66g. In view of the nature of this accident it is the opinion of the author that the peak decelerations which occurred are probably not more than three (3) times this value or about 5g's. The reader should observe carefully the fact that such peaks cannot physically be applied for any appreciable period of time otherwise the aircraft would have to stop in much less than 1950 feet. [The value would be 646 feet at 5g's constant deceleration].

Contact No. 2

This ground contact occurred after the aircraft became airborne following the initial touchdown and crossed the Saigon River. Observation of the forward main gear tire marks relative to a small dike on the far bank of the river shows (together with the absence of nose gear marks) that the aircraft again touched down in level or slightly nose up attitude. The extended nose gear and extended main gear permitted the aircraft to pass over this dike, allowing failure of all of these remaining gears with little or no contact of the bottom of the fuselage with the dike. The decelerations here would again be no more than the values occurring in the first contact. Upon passage over the dike the bottom of the aircraft began a skidding and plowing run through wet and soft rice fields to the final points of rest. Observation of the accident photos and other evidence shows the following:

a) The troop compartment and the crew compartments remained essentially intact, maintaining living space for those occupants.

b) All seats remained attached to the floor and there were no seat belt or harness failures.

c) Seats in the troop compartment are 16g seats attached to the floor with a 9g restraint. All were rearward facing.

d) Skid tracks through the wet/soft marsh-like terrain are strongly indicative of long-duration, low-level, constant deceleration for the cockpit and troop compartments.

e) Break-up of the lower fuselage occurred in many relatively small pieces consistent with many successive failures, again indicative of continued and hence low level continuous deceleration.

f) The failure of the side walls of the lower (cargo) compartment ultimately resulted in the formation of two skids or runners for the troop compartment which guided that compartment in almost a straight track, reducing lateral loads to only those of vibratory nature and allowing the floor to remain intact.

g) Adult occupants seated or kneeling on the floor between rows of seats, without any kind of restraint other than holding by hand were able to stay in place throughout the complete impact sequence without serious injury. Cuts and bruises were reported. Only those occupants in line with an isle and holding by hand appear to have been unable to retain position. These occupants would have been in a condition similar to a 'free fall' at a somewhat elevated 'g' value of about 1.5 to 2.0g as they 'fell' longitudinally along the isle to impact at or near the front bulkhead. Their injuries thus occurred in this mode.

ACCIDENT SYNOPSIS

- The crash of this aircraft consisted of two ground contacts separated by approximately 875 yards of free flight. The analysis of the data available shows the following concerning these two contacts:

Contact No. 1

This contact has been characterized by several of those aboard the aircraft as 'a near normal touch down' or 'no more than a hard landing typical of military or commercial aircraft.' The sink rate was reported to be 500 to 600 feet per minute by one of the cockpit crew (Mayor Traynor), a fact in agreement with:

a) Extrapolation of the MADAR data.

b) The aircraft attitude and speed, i.e., nose up at touchdown. (It is noted that the nose gear did not contact the ground at this point).

c) The aircraft would have been in 'ground effect' as it approached the surface with resulting tendency to reduce any existing sink rate.

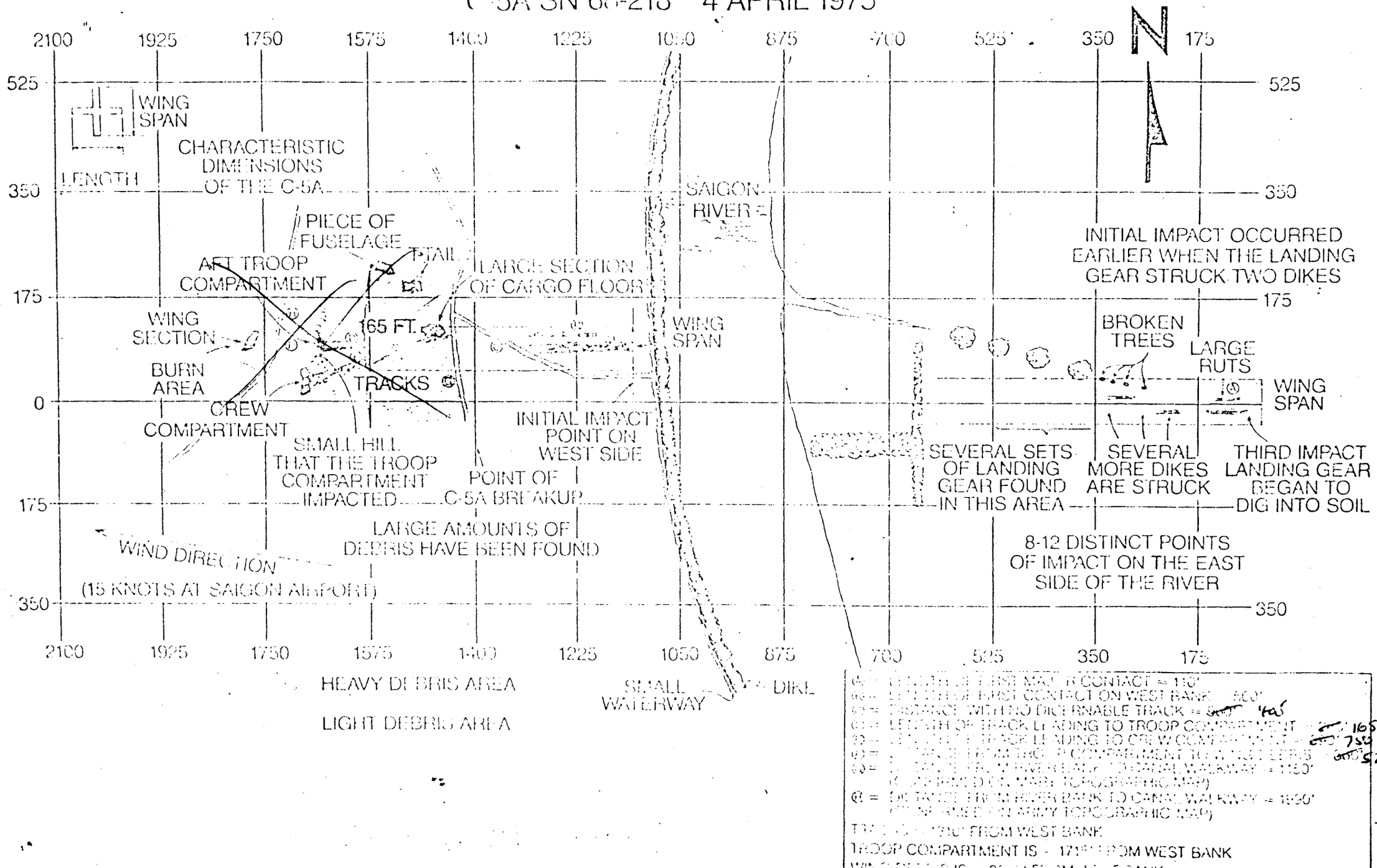
d) Statements of other crew, for example: Capt. Harp said in the Schneider Trial, page 2143, line 4: 'I would say there were hardly any G forces on the first landing.'

The primary structural failure at this first contact was removal of the rear sets of landing gears, probably due to the landing on a less than normally firm runway and to the above normal touchdown speed of 270 knots, both of which could be expected to increase the drag forces on the gear.

Since the ultimate design load for each gear does not exceed 240,000 lbs, and assumption of full design load being developed on the rearmost gears, plus a limit load of 160,000 lbs on each of the forward main gears, gives a total load of 800,000 lbs. This would load the 450,000 lb aircraft to no more than 1.78g's along the longitudinal axis of the aircraft. The vertical loads would have been very consistent with those occuring for a landing at near or lower than normal sink rate. Vibratory oscillations would have been induced into the structure due to failure of the gear, however these, being of high frequency, would have been more of an 'audible' nature to passengers of the troop compartment rather than of a nature such as to produce a displacement or impact type response of those passengers.

No hazard to the occupants of either the cockpit or troop compartments can thus be expected from this contact.

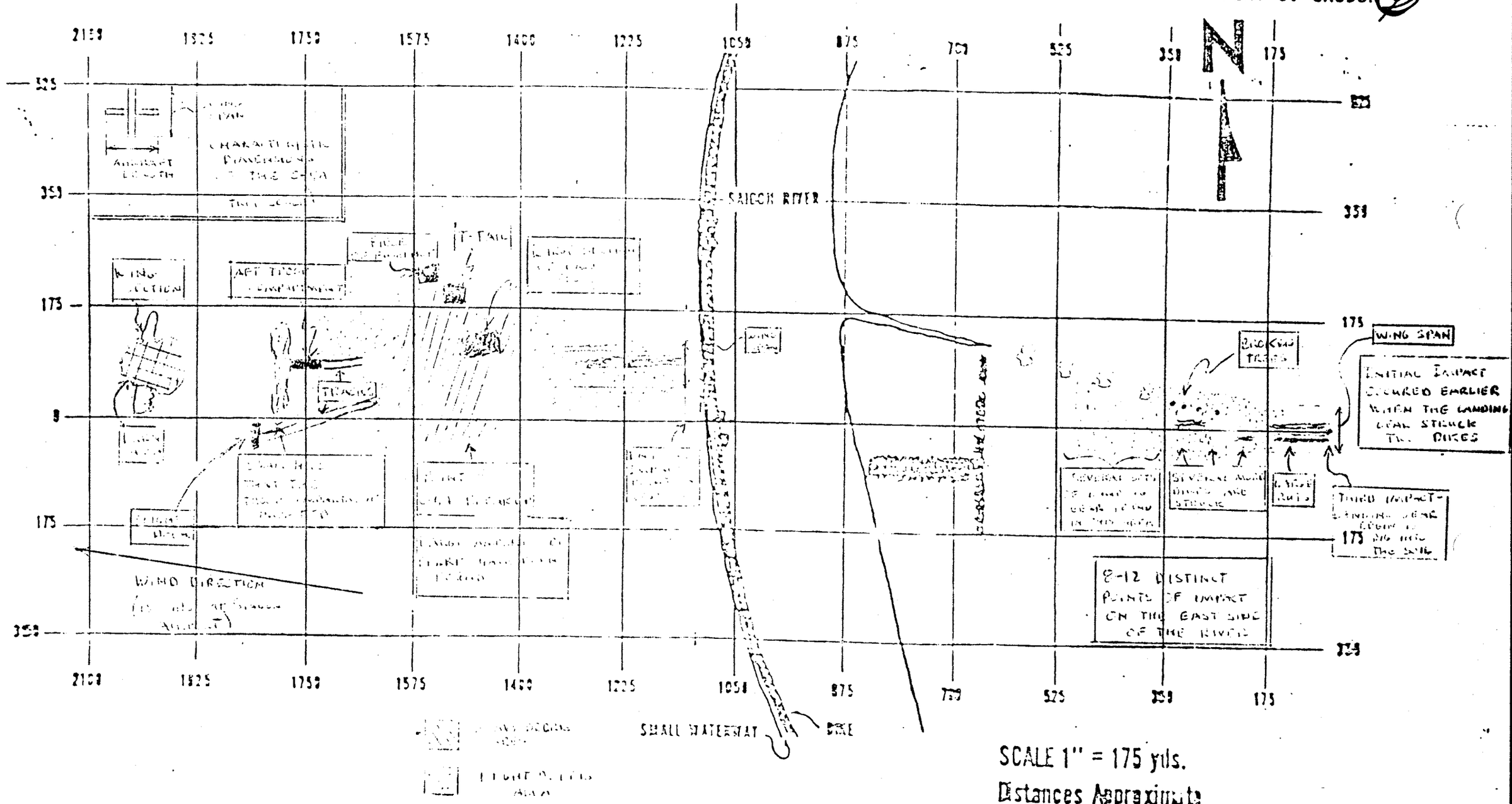
WRECKAGE DIAGRAM
C-5A SN 68-218 4 APRIL 1975



DEFT. EX. DB-Bunday Dep. Exh. 5
DATE: 12/10/81
REPORTER: ALBERT J. GASDOR

DEFT.EX. DD- Busby Dep Exh. 5-A
DATE: 12/18/81
REPORTER: ALBERT J. GASDOR

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



CRASH OF AF68-218 C-5A ON 4 APRIL 1975

By: John W. Edwards

Supervisor: LAC Technical Team
Serving Aircraft
Accident Board

AF 68-218 departed Ton Son Nhut Airport Saigon S. Viet Nam c. 4 April 1975 and crash landed approximately 28 minutes later in a rice paddy while approaching the runway from which it had just de-parted.

At the time of the decompression the aircraft had climbed to an altitude of 23,200 feet approximately. The aircraft continued its climb for an additional nine (9) seconds to a maximum altitude of 23,424 feet at which time it began a descent. According to the on board data recording system, MADAR, the aircraft first reached 10,000 feet altitude approximately seven (7) minutes and 51 seconds later. Attachment 5 dipicts the altitude time history.

1.0 The g'loads on the occupants at the decompression were essentially negligible as substantiated by the following information:

1.1 The Engineering analysis exhibit D-2 page 90 fifth paragraph indicates structural responses rather than airplane motions.

1.2 All crew statements described the decompression in terms of noise only, i.e., "Loud Pop" rather than airplane motion. Example: Harriett Mary Neill court testimony page 174 (Aimmerly case) last paragraph. "I remember the first thing I was aware of was that there was a loud pop and ---".

2.0 Regarding the effects of Hypoxia, it should be noted that the altitude of 68-218 at 23,424 feet was more than a mile lower than Mt. Everest which has been climbed by man many times.

2.1 Chanute AFB in Illinois has a Physiological Training Unit which publishes an Atmospheric Pressure Table which advises that the time to parachute from 20,000 feet to 10,000 feet is 6 minutes and 30 seconds whereas the time of useful consciousness is over twice that or 15 minutes for a working crew member. A person at rest would consume less oxygen. Attachment 5 shows that the time from rapid decompression until the aircraft descended to 10,000 feet was 7' 45" approximately. This table is repeated below for convenience.

U. S. STANDARD

ATMOSPHERIC PRESSURE TABLE

PREPARED

BY THE

PHYSIOLOGICAL TRAINING UNIT

CHANUTE AFB, ILLINOIS

DO	DON'T
1. Get annual physical within 60 days of birthday. (AFR 160-10)	1. Fly while you have a cold or are fatigued.
2. Have base line ECG in records. (AFR 160-121)	2. Donate blood unless emergency -- no flying for 72 hours after donation. (AFR 160-53)
3. Get physiological training refresher course every 3 years. (AFR 50-27)	3. Fly without flight clearance from Flight Surgeon when reporting to new station. Time will not be logged. (ATC Sup 1 to AFM 160-5)
4. Hand carry medical and dental records to new station. (ATC Sup 1 to AFM 160-5)	4. Fly for 24 hours following ingestion of drugs such as antihistamines (PBZ, benadryl, etc), narcotics or alcohol, etc. (Check with Flight Surgeon.)
5. Eat before flying -- prevents hypoglycemia.	5. Fly after injection treatment or drugs from Dentist.
6. Pilots should not eat at same facility to avoid food poisoning.	

PREVENT BURNS: Always wear coverall and gloves. Do not wear nylon undergarments or socks. Wear helmet and oxygen mask at all times in jet aircraft.

DON'T DOCTOR YOURSELF: Don't take any medicine unless your Flight Surgeon advises it. If you are sick, see your Flight Surgeon -- he is interested in your health.

OXYGEN LACK AND BAIL-OUT

ALTITUDE (feet)	TIME OF USEFUL CONSCIOUSNESS	TIME TO FREE FALL TO 10,000 FT	TIME (MIN) TO FALL TO 10,000 FT WITH 25 FT CANOPY OPEN	SYMPTOMS OF OXYGEN LACK
10,000	Many hrs	0:00	0:00	Fatigue, headache, drowsiness.
20,000	15 min	0:49	6:50	No sense of time, over-confidence, poor judgment, impaired vision, faulty reasoning, unconsciousness.
30,000	1 1/2 min	1:31	12:36	
40,000	15 sec	2:05	16:54	If any of these symptoms appear, use 100% O_2 or Pressure. Check "P. D. McCripe" for oxygen leak!
50,000	9 sec	2:32	22:24	
60,000	9 sec	3:02	over 28:00	

P - Pressure R - Regulator
D - Diaphragm Reg. I - Indicator
M - Mask P - Portable oxygen
C - Clamps E - Emergency oxygen
C - Connections

1. Standard bail-out bottle lasts 9 - 12 minutes.
2. Temperature - 67° F at 40,000 ft and above.
3. Free fall mandatory above 30,000 ft. due to extreme opening shock of parachute, temperature and oxygen lack.

Altitude (Feet)	Pressure mm. Hg.	p.s.i.
38000	154.5	2.99
38500	151.3	2.92
39000	147.5	2.85
39500	144.1	2.79
40000	140.7	2.72
40500	137.4	2.66
41000	134.1	2.59
41500	131.0	2.53
42000	127.9	2.47
42500	124.9	2.42
43000	121.9	2.35
43500	119.0	2.30
44000	116.3	2.25
44500	113.5	2.19
45000	110.9	2.14
45500	108.3	2.09
46000	105.8	2.04
46500	103.1	1.99
47000	100.7	1.95
47500	98.3	1.90
48000	96.0	1.85
48500	93.7	1.81
49000	91.5	1.77
49500	89.4	1.73
50000	87.3	1.69
50500	85.2	1.65
51000	83.2	1.61
51500	81.3	1.57
52000	79.3	1.53
52500	77.4	1.50
53000	75.6	1.46
53500	73.8	1.43
54000	72.1	1.39
54500	70.4	1.36
55000	68.8	1.33
55500	67.1	1.30
56000	65.5	1.27
56500	64.0	1.24

Altitude (Feet)	Pressure mm. Hg.	p.s.i.
19000	354.0	7.04
19500	356.5	6.89
20000	349.1	6.75
20500	341.8	6.61
21000	334.5	6.47
21500	327.5	6.33
22000	320.8	6.20
22500	314.0	6.07
23000	307.4	5.94
23500	300.8	5.82
24000	294.4	5.70
24500	288.0	5.57
25000	281.8	5.45
25500	275.8	5.33
26000	269.8	5.22
26500	263.8	5.10
27000	258.0	4.99
27500	252.4	4.88
28000	246.8	4.77
28500	241.4	4.67
29000	236.0	4.56
29500	230.8	4.46
30000	225.6	4.36
30500	220.4	4.26
31000	215.4	4.17
31500	210.4	4.07
32000	205.6	3.98
32500	201.0	3.89
33000	196.2	3.80
33500	191.8	3.71
34000	187.2	3.62
34500	183.0	3.54
35000	178.7	3.46
35500	174.4	3.37
36000	170.3	3.29
36500	166.3	3.22
37000	162.4	3.14
37500	158.5	3.07

Altitude (Feet)	Pressure mm. Hg.	p.s.i.
0	760.0	14.70
500	746.4	14.43
1000	732.9	14.17
1500	719.7	13.92
2000	706.8	13.66
2500	693.8	13.42
3000	681.1	13.17
3500	668.8	12.93
4000	656.5	12.69
4500	644.2	12.46
5000	632.3	12.23
5500	620.6	12.00
6000	609.0	11.78
6500	597.6	11.55
7000	586.4	11.34
7500	575.3	11.12
8000	564.4	10.91
8500	553.7	10.71
9000	543.2	10.50
9500	532.8	10.30
10000	522.6	10.11
10500	512.6	9.91
11000	502.6	9.72
11500	492.8	9.53
12000	483.2	9.35
12500	473.8	9.16
13000	464.5	8.98
13500	455.4	8.81
14000	446.4	8.63
14500	437.5	8.46
15000	428.8	8.29
15500	420.2	8.13
16000	411.8	7.96
16500	403.5	7.80
17000	395.3	7.64
17500	387.3	7.49
18000	379.4	7.34
18500	371.7	7.19

Altitude (Feet)	Pressure mm. Hg.	p.s.i.
57000	62.4	1.21
57500	61.0	1.18
58000	59.5	1.15
58500	58.1	1.12
59000	56.8	1.10
59500	55.4	1.07
60000	54.1	1.05
60500	52.8	1.02
61000	51.6	0.998
61500	50.4	0.975
62000	49.2	0.951
62500	48.0	0.928
63000	46.9	0.907
63500	45.8	0.886
64000	44.7	0.864
65000	40.6	0.7861
66000	36.9	0.7145
70000	33.8	0.6494
72000	30.4	0.5905
74000	27.7	0.5365
76000	25.3	0.4876
78000	22.9	0.4430
80000	20.8	0.4028
82000	18.9	0.3661
84000	17.2	0.3327
86000	15.6	0.3024
88000	14.2	0.2749
90000	12.9	0.2496
92000	11.7	0.2270
94000	10.7	0.2063
96000	9.7	0.1875
98000	8.8	0.1704
100000	8.0	0.1549
120000	3.34	0.0628
140000	1.51	0.0293
160000	0.787	0.0153
180000	0.433	0.0083
200000	0.258	0.0046

CONVERSION FACTORS

I. PRESSURE:

1 atmosphere = 14.696 psi = 760 mm Hg = ~~29.92~~ 29.92 in Hg = 1013.3 mb.

1 mm Hg = 13.595 mm H_2O = 0.535 in. H_2O = 0.0193 psi

1 psi = 51.715 mm Hg

1 in. H_2O = 1.865 mm Hg

II. ALTITUDE:

1 foot = 0.3048 meter

1 meter = 3.2808

1 mile (US) = 1.9093 kilometer

1 kilometer = 0.62137 miles

III. VOLUME:

1 cu ft = 28.316 liters

1 liter = 0.03532 cu ft = 61.025 cu in

1 cubic meter = 35.314 cu ft

IV. TEMPERATURE:

Deg. Centigrade (°C) for Deg. Fahrenheit (°F)

°C = 5/9 (T°F - 32)

Deg. Fahrenheit for Deg. Centigrade

°F = (9/5 T°C) + 32

Temp. absolute (K) from Centigrade

K = T°C + 273.18

2.2 The medical attendants in the aft troop compartment reported no signs of Hypoxia. Example: Court testimony of Mary Neill (formerly Ms Goffenett) page 176 (Zimmerly case).

3.0 The vertical g' loads at the first impact were essentially negligible as substantiated by the following data.

3.1 Capt Traynor noted that the sink rate was between 500 and 600 feet per minute as documented in his court testimony page 90 (Marchetti case) second answer.

3.2 The engineering analysis section of the Accident Report notes that the landing gear would have failed at 11 to 16 feet per second rate of sink at the gross weight of 450,000 pounds due to the high vertical load which did not happen since only the two aft main gears failed at the first impact and broke in a drag load direction as a pencil would when held tightly in the fist and thrust across the table striking a heavy object.

3.3 The marks in the soil showed indentations of only the aft gear which broke and the stubs of these broken gear then plowed into the soft farm land digging up furrows too narrow for the entire gear. Reference Photograph 3G.

3.4 The remaining forward main gear and the nose gear were carried by the aircraft to the second impact point across the river.

3.5 Capt Traynor's court testimony on page 89 (Marchetti case) describes the first impact as "--- normal or less than normal rate of descent ---".

3.6 Harriett Mary Neill (Goeffenett) court testimony on page 180 (Zimmerly case) describes the first impact as "--- a firm commercial airliner landing ---".

4.0 The longitudinal decelerations at the first impact were essentially negligible as substantiated by the following data.

4.1 The engineering analysis, exhibit D-2, on page 90 third paragraph, shows the airpseed 3.6 seconds prior to first impact as 270 knots or 455 feet per second. On the fifth paragraph of the same page, the airpseed just prior to the second impact is listed as the identical 270 knots (455 feet per second), therefore, the speed did not reduce noticeably.

4.2 The crew statements as summarized in 3.5, 3.6 above also pertain to the absence of longitudinal deceleration since no sudden "bumps" were discussed.

4.3 Calculations of the change in aircraft velocity due the energy absorbed by breaking one of the aft main gear as detailed in attachment #1 show that the aircraft would slow down by .2 feet per second from its initial velocity of 455 feet per second. The second aft main gear would have broken later with an additional .2 feet per second slow down.

4.4 Exhibit 3g which is a color photograph of the first impact point clearly shows:

A. The left aft gear striking the ground first - rolling a few feet and breaking off at about the same time the right aft gear strikes the ground rolls for a short distance and also breaks off as evidenced by a discontinuation of the tire marks. The aircraft in a left wing low attitude, continues to settle and the broken stub of the aft main gear starts plowing through the soft farm land. At the same time the two left engines come close enough to the ground to "vacuum" up the soft dust and rice straw as evidenced by the two clean streaks

widely spaced at the same spacing as the two left engines on the C-5. The inboard door of the left aft gear, now being free to swing downward because of the broken gear strikes the ground just to the right of the deep darker plow mark and scrapes the surface of the soil resulting in a wider mark but less dark in color due to the lesser penetration of the soil. At this time the aircraft starts to gain altitude since no engine "vacuum" marks are in evidence. The aircraft still being close to the ground continues to blow away surface dust and straw.

The left aft gear tumbles free and stops to the left of the flight path near the vegetation that runs lateral to the flight path. The right aft gear ends up near the line of palm trees just to the right of the line of flight. The aircraft continues on toward the river with the right wing cutting off four (4) small palm trees in an ascending manner.

what!

5.0 The vertical g loads on the occupants at the second impact were essentially negligible as shown by the following data.

5.1 The aircraft was very close to the ground as shown by color photograph exhibit 3F which shows the effects of the broken stubs of the aft main gear plowing through the vegetation on the river side of the dike. It is very noteworthy that the nose gear was above this vegetation on the river side of the dike since no middle plow mark was left by the nose gear. This nose up attitude of the aircraft allowed the main fuselage to clear the dike therefore the

aircraft literally flew onto the rice paddy severing the nose gear and then the remaining two forward main gear at the dike. Again the color photograph indicates the aircraft was in contact with the rice paddy very shortly after crossing the dike.

5.2 The plow marks on the river side of the dike are essentially uniform from the river to the dike as shown by exhibit 3F indicating that the aircraft was not descending rapidly. An appreciable descent rate would have shown a widening and deepening of these plow marks.

5.3 The pilot of the aircraft, Capt Traynor, in his court testimony on page 2215 (Schneider case) describes the second impact as "This time it shook the aircraft a little bit more" and "well, it was a vibration like I had blown a tire or run off a runway."

5.4 Neither pilot nor co-pilot mentions being bounced up and down which would be indicative of vertical g loads.

5.5 In the aft crew compartment all adult occupants were in positions other than normal seats. Mrs Neill (formerly Goffenett) was in between two rows of seats with her arms spread over the seats she was facing. None of these occupants were dislodged from their position despite the lack of normal seat belt restraints except Mrs. Neill who was between rows of seats 2 and 3 from the front, as evidenced by her court testimony on page 87 (Marchetti case). Mrs. Neill stated that after the first impact she " must have let loose", "and the second impact, I was thrown forward against the forward bulkhead".

Doctor Stark in his court testimony (Marchetti case) on page 25 stated that none of the adults had seats. Because of his concern for the impending landing, Doctor

Stark had sat down between two rows of seats (page 30) and braced himself against the seat at his back and was not dislodged from this position during the entire sequence. Also, on page 30 he describes the sequence "--- and there was certainly a very definite impact but everything remained pretty much as it was." On page 34 he refers to the condition of the children as "---essentially, unchanged from the time they were aboard the plane, as near as I could determine."

Capt Marcia Gray Tate was on her knees between two rows of seats leaning forward over the seats in front of her as discussed in her court testimony (Marchetti case) on page 33. On page 35 she testified that she stayed in that position until the aircraft came to a complete stop. Also, on page 35 she described the landing as "--- bumpy but it was not particularly violent", and compared the landing on page 36 as "yes, there was -- very similar to me -- to a rough landing in a commercial aircraft that I had been in previous to that."

Lieutenant Aune was sitting in the aisle with her legs crossed and bracing herself with the seats according to her court testimony (Marchetti case) on page 1914. Also on page 1915 it is noted that she was not dislodged except as a result of turning loose to grab someone's ankle who was standing. At this time she went sliding along the floor to the front.

These statements together with the physical evidence and photographs indicate that the vertical g loads were negligible.

6.0 The longitudinal decelerations were fairly uniform and of a relatively low magnitude for an airplane crash.

6.1 The g loads as computed by using velocity and distance were 1.6 average for the occupants of the aft troop compartment and 1.46 for occupants of the flight deck. Refer to Attachment 2.

6.2 The expected variation in this average would be a peak of 3.91 g's as scaled from 27 different tests of rocket sled test by the Air Force in 1951 used to develop restraint systems. The cover sheet and tabular data sheet is listed as Attachment 3.

6.3 The terrain was flat, wet, grassy and free of any obstructions such as trees or rocks.

6.4 The aircraft sliding in essentially a straight line stayed in contact with the ground at all times as shown by photographs 3E, 3F, and 3B.

6.5 The occupants were seated in rear-ward facing seats which means that the occupant is pressed into the seat cushions by the decelerations.

6.6 The occupants are seated approximately 20 feet above the bottom of the fuselage which means that the soft aluminum structure absorbs and cushions the shock loads in a manner similar to the soft body structure of a racing car which is termed " deformable" by race car drivers.

The errosion of this structure by the scrubbing action of the rice paddy would be felt as vibration and noise rather than a shock due to the cushioning action of the structure between the occupant and the ground.

6.7 The average g' load of 1.6 would be only one-tenth that of the average of the 43 rocket sled test in the attachment 3 report. In all these cases the deceleration distance was from 24.6 feet to 47.1 feet with a velocity change of ranging from 77 feet per second to 181.5 feet per second.

6.8 The peak deceleration of 3.91 g's is about one-half of what one experiences in an amusement part ride which range from 1.53 g's to 6.2 g's.

6.9 The aircraft skidded through the wet rice paddy in a slightly nose up attitude as indicated by the front end of the aircraft being more intact - that is the entire circumference of the nose section, although severely damaged was with the crew compartment as shown in photo 3C. The crew compartment actually skidded on the lower portion - and after coming to a stop - rolled over on the right side since no mud is noted on the cab top.

6.10 The copilot, Capt Harp, actually described the stopping of the aircraft in his court testimony on page 2143 (Schneider case) as "It seemed like we were sliding through a bog. The slide itself was relatively smooth."

7.0 As the aircraft slide through the rice paddy, the errosion of the lower fuselage up to the cargo floor severely diminished the structural integrity of the aircraft. This scrubbing action tore off pieces of structure - absorbing the speed of the aircraft and opening up cracks in the structure.

7.1 When the errosion penetrated the cargo floor at about 800 feet from the dike the cracks opened up the sides of the fuselage and the wing - still having lifting power due to the remaining velocity, actual broke free from the flight deck in front of the wing and the aft troop compartment aft of the wing, and literally flew off separately. The wing landed a football field and a half in front of the aft troop compartment where a fire broke out consuming most of the wing.

7.2 At about the same time the empennage, due to the structural cracks formed by the scrubbing away of the aft fuselage, and having lifting power also, broke loose and flew separately off to the right side.

7.3 The flight deck, with the lower portion more intact, continued to slide through the wet rice paddy and curved off to the left. At the end of the slide the flight deck turned over on its right side. The total distance traveled by the flight deck was scaled from the wreckage diagram attachment 7 as 2,209 feet which computes to an average g load of 1.46 in the longitudinal or X axis and the lateral movement was scaled as 607.89 feet from the time of separation. This computes to 1.07 g's in the lateral or Y axis as shown in attachment 4.

7.4 The aft troop compartment, at the time of separation from the wing and empennage, continued to slide through the rice paddy. Since the lower portion of the fuselage under this troop compartment was not intact, the friction was greater and the distance traveled was less - actually scaled as 2,012 feet computing 1.60 g's in the X axis. The lateral movement was also less - actually scaling at 121.56 feet which computes at .29 g's in the Y axis.

7.5 The aft troop compartment was now open at the front end due to the departure of the wing. This opening allowed relatively warm 100°F airto enter this compartment which was previously cooled at about 70°F. This sudden inrush of warm air would have been noticed by the occupants. The aft troop compartment came to a rest about 150 yards from the burning wing, pointing almost directly at the fire area. Although the wind was blowing in a direction to carry the smoke and fumes away from the aft troop compartment, undoubtedly some fire oder would have been noticed even 150 yards away.

7.6 There was no fire in the area of the aft troop compartment as evidenced by color photograph 3B and my own personal observations at the site.

7.7 There was no fire in the area of the crew compartment as evidenced by photographs 3C and my own personal observations at the site.

7.8 The only fire was in the wing area as shown by 3A.

The foregoing opinion is based on a total assessment of all the available evidence and information and includes; actual on the site participation in the search for pertinent aircraft parts, an examination of the wreckage, evaluation of recorded data on the on-board recording system MADAR, evaluation of all crew statements made to the Collateral Board, evaluation of statements made by crew members in depositions, evaluation of statements made by some crew members in court testimony, evaluation of aerial photographs, and on the site knowledge of the type of terrain.

The preponderance of evidence leads to a reasonable engineering conclusion that the occupants of this aircraft were not harmed by the g' loads at either the Rapid Decompression or the impacts with the ground.

Further it is a considered engineering opinion that the occupants were subjected to far less severe conditions of "thin" air than that which is expected to be harmful.

John W. Edwards
Chief Project Engineer
Lockheed-Georgia Company

JWE:bg

Determine Aircraft Velocity Change due to breaking of main gear strut due to drag loads:

KE_1 = before 1st impact

$$KE_1 = 1/2\ MV_1^2 = 1/2\ \frac{451000}{32.2} \times 455^2$$

$$KE_1 = 1.4498 \times 10^9$$

Energy absorbed by breaking one gear:
Assume gear picked up drag load for 10 feet starting at 0 drag and increasing to 250000 at 10 feet

$\therefore$ Average distance is 5 feet

$\therefore$ $Fxd = 250000 \times 5 = 1.250 \times 10^6$ foot pounds

KE_2 = energy left after breaking first gear.

$$KE_2 = KE_1 - 1.25 \times 10^6$$

$$KE_2 = 1449.8 \times 10^6 - 1.25 \times 10^6$$

$$KE_2 = 1448.55 \times 10^6$$

Velocity after 1st impact:

$KE_2 = 1/2\ MV_2^2$ or

$$V_2^2 = \frac{2 \times KE_2}{M}$$

$$= \frac{2 \times 1.44855 \times 10^9}{\frac{451000}{32.2}}$$

$$= 2.89710 \times 10^9 = \frac{2.8971 \times 10^9}{1.40062NO^4}$$

$$V_2^2 = 2.068441 \times 10^5$$

V_2 = 454.80 feet per sec*

The aircraft would travel this 10 feet in $\frac{10}{455}$ or .022 sec., therefore, Velocity change .2 feet sec. in .022 sec.

$$V = AT \text{ or } A = \frac{.2}{.022} = 9.09$$

$$g's = \frac{A}{32.2} \qquad g's = .28$$

*This is for one gear - the second gear would impart a similar .2 ft decel at a later time.

AF TECHNICAL REPORT No. 5915, Part 2 December 1951

HUMAN EXPOSURES TO LINEAR DECELERATION

Part 2. The Forward-Facing Position and the Development of a Crash Harness

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Run No.	Subject	Initial Velocity (ft/sec)	Final Velocity (ft/sec)	Velocity Change (ft/sec)	(g's)	Duration (seconds)	Individual Stopping Distance (feet)	Calculated Slope (ft/sec)	Calculated Plateau (g's)	Weight of Subject (pounds)	Force (g x W) (pounds)	Harness Area (sq. in.)	Harness Pressure (psi)
		Configuration No. 1 Deceleration Distance, 47.1 feet											
		Active Brake Units, 1,2, ——— 12, 13, ——— 23, 24											
96	JTB	217	115	102	69.5	.30	12.6	1170	11.3	172	1940	137.5	14.1
101	JTB	No displacement record											
119	JTB	202.5	95	107.5	72.6	.33	15.6	1060	10.9	174	1893	217.5	8.7
120	JTB	No displacement record											
121	WAR	200.5	72	128.5	87.6	.37	20.5	575	11.9 —	152.5	1811	207.5	8.7
		Configuration No. 2 Deceleration Distance 35.4 feet											
		Active Brake Units 1,2,3, ——— 12, 13 24, ——— 23, 24											
94	JTB	216	128	88	61	.21	8.5	1055	15.0	172	2575	221	9.9
95	JTB	No displacement record											
97	WAR	226	149	77	52.3	.21	4.1	1079	15.0 —	155	2310	241.5	9.5
98	WAR	211	116.1	97.9	66.4	.21	11.0	1250	16.8 —	155	2586	207.5	12.5
102	RCA	216.2	121	95.2	65	.21	10.0	1265	16.7	170	2840	245.5	11.7
106	JTB	220	126	94	64.2	.21	9.0	1170	16.6	176	2920	221.0	13.8
113	RCA	220	115.5	104.5	70.7	.21	11.9	980	13.9	170	2366	204.0	11.6
114	JTB	202	113.5	88.5	60.4	.22	10.6	923	14.3	177	2522	238.0	10.6
115	JTB	215.5	123	92.5	63.3	.235	26.5	970	17.5	197	3445	198.2	17.4
118	JTB	223.5	141	82.5	56.1	.22	4.4	935	16.9	208	3500	205.7	17.6
122	WAR	145	92	53	70	.22	15.2	940	15.0	(208+22)	3445	198.0	17.2
		Configuration No 3. Deceleration Distance 29.7 feet											
		Active Brake Units, 1,2,3,4, ——— 12,23, 34											
99	JTB	216.2	126	90.2	61.5	.16	6.5	1155	21.2 —	149	3150	201.3	15.7
100	WAR	220	129.5	90.5	61.8	.16	6.0	1230	20.4 —	155	3225	205.5	15.2
110	WAR	232	135	97.0	59.4	.16	5.1	1150	21.2 —	155	3280	176.5	18.6
113	JTB	219	128	91.0	62.1	.16	6.2	1215	23.5	149	3500	170.3	20.6
116	WAR	220	131	89.0	60.8	.16	5.7	1022	23.3 —	155	3610	176.3	20.5
117	JTB	210	108	102	69.6	.175	7.8	1000	21.1	149	3590	170.3	21.2
128	WAR	No displacement record											
132	JTB	No displacement record											
163	JTB	132	0	132	90.4	.23	18.5	894	32.0	(170+22)	6222	186.0	32.9
165	JTB	206	126	80	54.8	.17	5.3	934	25.0	(170+22)	3436	186.0	18.5
166	JTB	200	107	90	61.6	.175	8.0		19.4	170	3298	186.0	17.7
		Configuration No 4 Deceleration Distance 29.7 feet											
		Active Brake Units, 1,2,3,4,5 ——— 12,23,34											
103	JTB	221	110	111	69.3	.16	9.3	1110	25.8	155	4000	240.3	16.7
104	WAR	209.6	122.2	86.4	59.	.16	6.2	975	24.3 —	152	3695	207.5	17.8
105	RCA	No displacement record											
122	JTB	201	120	78	53.0	.16	5.6	980	25.0	152	3800	201.3	18.9
		Configuration No. 5 Deceleration Distance 24.8 feet											
		Active Brake Units, 1,2,3,4,5,6 ——— 12,13											
107	JTB	218	134	84	57.3	.16	3.2	966	27.0	149	4030	240.3	16.8
108	WAR	211	129	83	56.7	.16	4.0	900	25.9 —	153	3902	207.5	18.1
109	RCA	213	123	90	61.6	.155	2.4	990	28.2	170	4800	204.0	26.9
110	JTB	226	142	84	57.4	.15	2.0	1150	31.8	172.5	5490	217.5	25.2
111	JTB	211	104	107	73.0	.15	5.0	1170	34.6	152	5255	240.3	21.8
123	RCA	197.6	106	91.6	62.6	.16	7.7	910	28.5	172.5	4899	204.0	23.8
124	JTB	210	131	79	53.7	.16	3.2	900	26.7	172.5	4599	217.5	21.1
129	WAR	No displacement record											
130	JTB	201	124	77	52.5	.16	4.8	796	24.8	153	3771	240.3	15.8
		Backward Facing, Seated Position •											
113	WAR	206	90	116	72.2	.16	8.2	1156	35.0 —	152	5320	260	20.5
134	JTB	208	96	112	76.1	.16	9.2	1160	34.8	153	5324	252	21.1
		Configuration No. 6. Deceleration Distance 24.8 feet											
133	JTB	213	87	126	86	.155	11.1	1370	36.6	177	6839	238	28.7
135	JTB	220	105.5	114.5	78.9	.16	7.7	1344	34.1	172	6553	217.5	30.2
		Configuration No. 7 Reduced Brake Pressure											
		Deceleration Distance, 39.5 feet. 38 consecutive brakes at 150 to 200 p.s.i. closing pressure											
210	PMS	210	116	94	64.2	.237	12.0	881	13.9	306	2844	280	10.2
		Deceleration Distance, 36.4 feet 35 consecutive brakes at 200 to 250 p.s.i. closing pressure											
211	JTB	218	123	95	64.8	.235	9.9	288	20.6	175	3604	217.5	16.6
		Deceleration Distance, 34.3 feet 33 consecutive brakes at 250 to 300 p.s.i. closing pressure											
212	PMS	210	89.5	120.5	82.2	.217	11.9	300	32.7	206	6735	280	24.1
		Deceleration Distance, 36.4 35 consecutive brakes at 250 to 300 p.s.i. closing pressure											
213	JTB	224	71.0	143	97.5	.250	18.7	344.5	36.5	175	6386	217.5	29.4
		Deceleration Distance, 36.4 35 consecutive brakes at 300 to 350 p.s.i. closing pressure											
214	PMS	222.5	121.0	1256	123.8	.263	21.8	331	38.62	306	7938	250	28.35
		Deceleration Distance 31.2 feet 30 consecutive brakes at 350 to 400 p.s.i. closing pressure											
215	JTB	226	50.2	175.8	120.	.228	19.7	493.5	45.4	175	7942	217.5	36.5

A. Velocity at entry to brakes
B. Velocity at exit from brakes
C. Velocity change in miles per hour and feet per second
D. Duration of deceleration
E. Calculated equivalent stopping distance for the observed deceleration if final velocity were zero
F. Initial slope of deceleration-time curve calculated from displacement-time record.
G. Plateau of trapezoidal deceleration-time curve calculated from displacement-time record.
H. Weight in pounds of subject just prior to run.
I. The product of calculated plateau g-times the weight of the subject, from columns F and G.
J. Measured area of harness webbing impinging on the subject in the forward seated position.
K. The force in column K, divided by the harness area column I, to give average harness pressure

• Measured head area of subject against seat.

RUN #	A AVERAGE g (1)	B PLATEAU (TABLE II)	C PLATEAU 218 (2)	D PEAK g - SEAT (3)	E PEAK g -SEAT 218 (4)
96	10.56	11.3	1.71	11.0	1.67
119	10.12	10.9	1.72	13.5	2.13
121	10.79	11.9	1.76	15.5	2.30
94	13.01	15.0	1.84	27.8	3.42
97	11.39	15.0	2.11	*	*
98	14.48	16.8	1.86	*	*
102	14.08	16.7	1.90	21.5	2.44
106	13.90	16.6	1.91	24.5	2.82
117	11.06	13.9	2.01	19.0	2.75
118	12.49	14.3	1.83	*	*
149	12.22	17.5	2.29	*	*
150	11.65	16.9	2.32	*	*
164	14.54	15.0	1.65	*	*
99	17.51	21.2	1.94	25.5	2.33
100	17.57	20.4	1.86	26.5	2.41
142	16.89	21.2	2.01	*	*
143	17.66	23.5	2.13	*	*
146	17.27	23.3	2.16	*	*
147	18.10	24.1	2.13	*	*
163	17.82	32.1	2.87	*	*
165	14.61	18.0	1.97	*	*
166	15.97	19.4	1.94	*	*
103	16.30	25.8	2.53	35.0	3.44
104	16.77	24.3	2.32	34.0	3.24
122	15.14	25.0	2.64	18.0	1.90
107	16.30	27.0	2.65	35.0	3.44
108	17.18	28.9	2.69	28.5	2.65
109	16.03	28.2	2.81	35.3	3.52
110	17.39	31.8	2.93	38.0	3.50
111	21.74	34.6	2.55	44.5	3.28
123	17.78	28.5	2.56	22.0	1.98
124	14.75	26.7	2.90	36.0	3.91
130	14.95	24.8	2.65	*	*
113	22.52	35.0	2.49	38.5	2.74
114	21.74	34.8	2.56	29.9	2.20
133	25.25	38.6	2.51	*	*
135	22.22	38.1	2.74	*	*
210	12.32	13.9	1.81	19.9	2.58
211	13.72	20.6	2.40	23.0	2.68
212	17.25	32.7	3.03	31.0	2.88
213	17.76	36.5	3.29	36.0	3.24
214	19.92	38.6	3.10	38.5	3.09
215	23.95	45.4	3.03	47.0	3.14
TOTAL	694.67		100.11		75.68
AVERAGE	16.16		2.33		2.80

* CURVE FOR SEAT DECEL NOT GIVEN IN REF REPORT

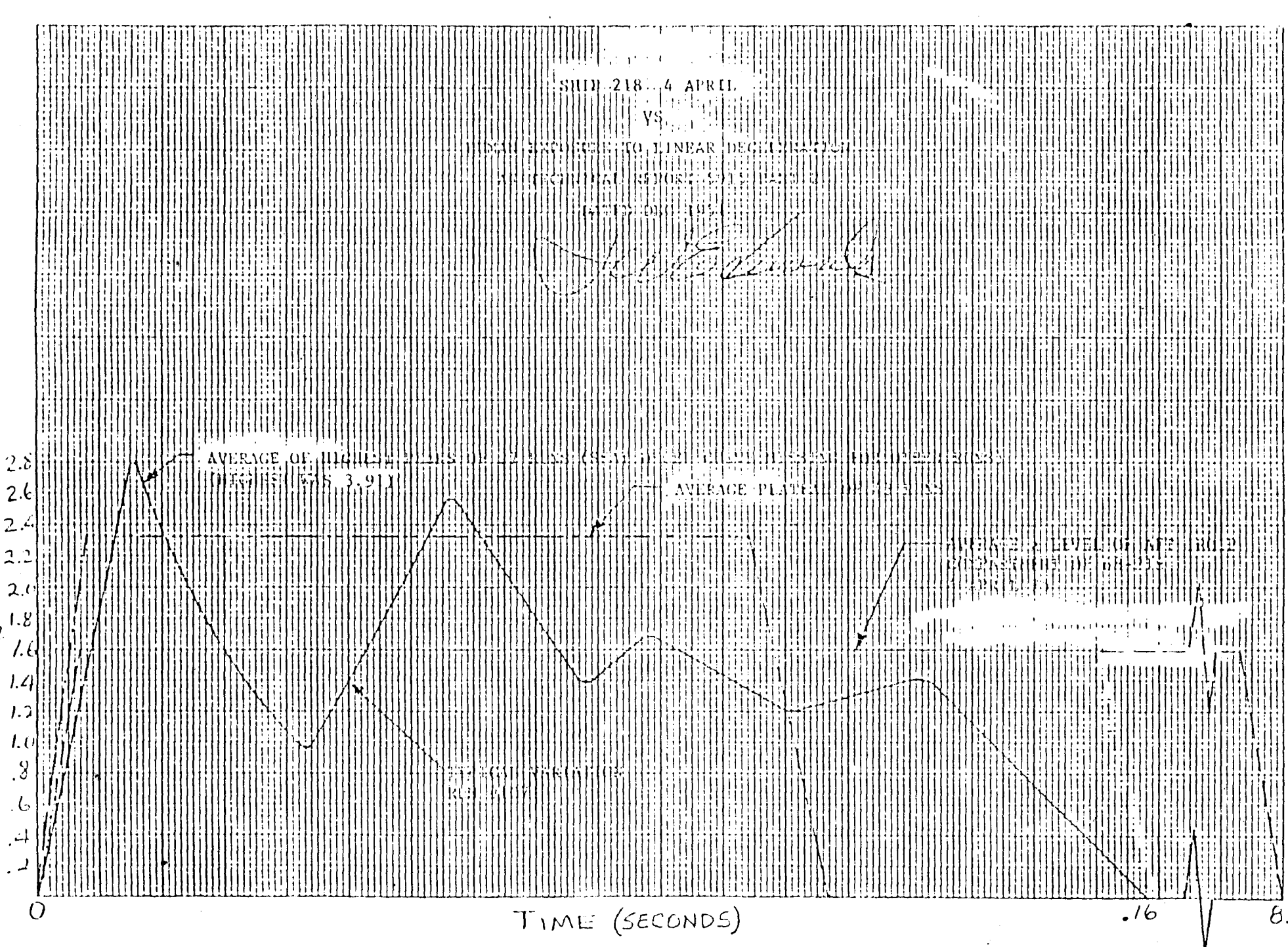
REF: HUMAN EXPOSURE TO LINEAR DECELERATION AF 5915 PART 2 DATED DECEMBER 1951
TABLE II, PAGE 20

(1) Velocity change divided by duration divided by 32.2

(2) Divide Column B by Column A and multiply by 1.6 in order to ratio the sled decels to the airplane average decel.
2.33 (average plateau for the airplane) is used to construct the curve.

(3) Scaled from seat decel curves in referenced report

(4) Divide Column D by Column A and multiply by 1.6 in order to ratio the sled seat decels to the airplane average decel.
The 2.80 average was used for the highest peak on the variable curve which was patterned to resemble Run #107 seat curve whose average decel is close to Column A Average.



ATTACHMENT #3

"Y" AXIS g LOADS ON PASSENGER COMPARTMENTS

Note (1) Troop compartment traveled 121.58 feet in the "y" axis after separation from the aircraft.

o Total travel time $\frac{455}{51.56} = 8.82$

T = 5.083 for 1159 feet

o $d = 1/2\ AT^2$ or $A = \frac{2d}{T^2}$

o $A = \frac{2 \times 121.58}{(5.083)^2} = 9.41$

$g's = \frac{9.41}{32.2} = .29$

(2) Flight Deck traveled 607.89 feet in the "y" axis after separation from the aircraft.

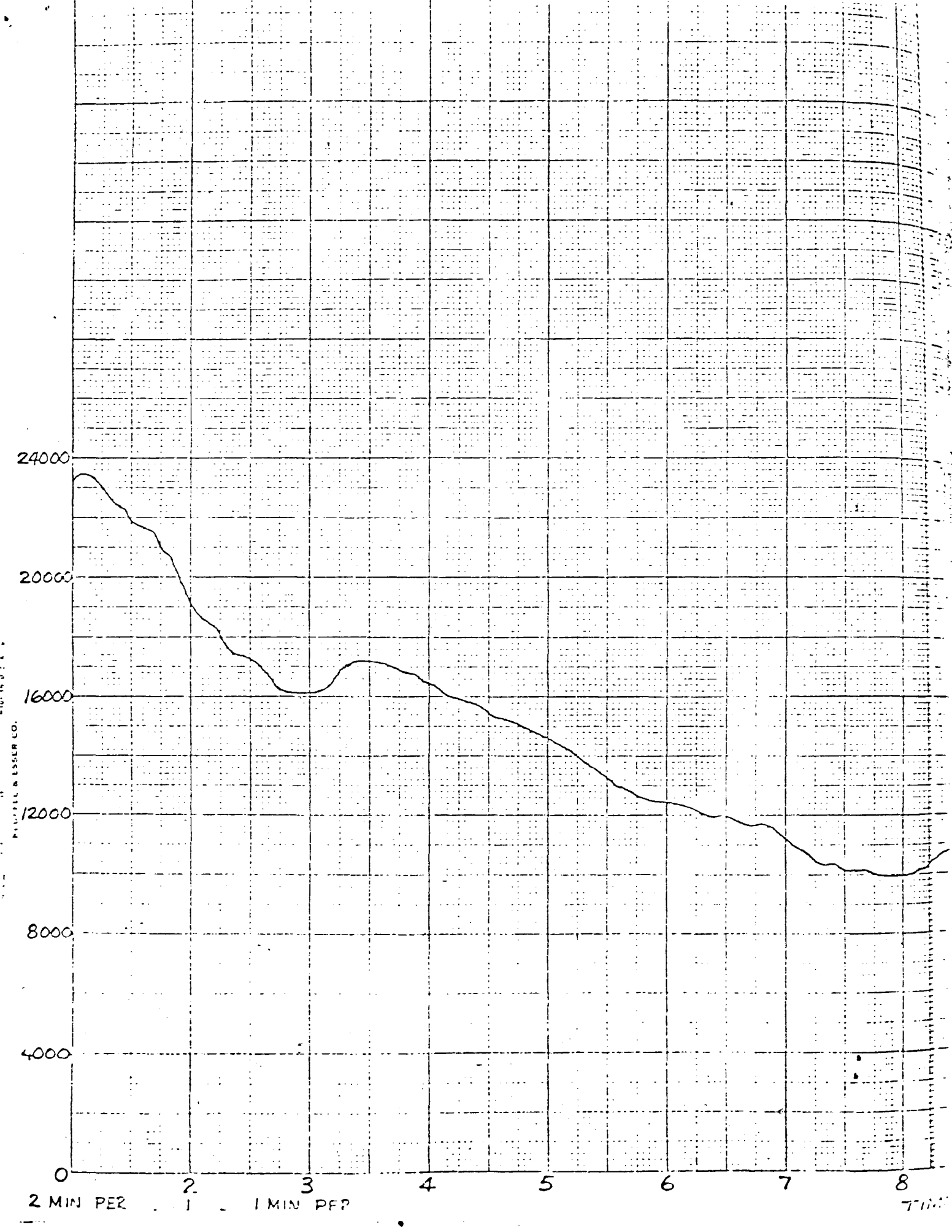
o Total travel time $\frac{455}{46.96} = 9.69$

T = 5.94 sec for 1356 feet

$A = \frac{2 \times 607.89}{(5.94)^2} = 34.45$

$g^1 = \frac{34.45}{32.2} = 1.07$

J. E. Edwards


24000
20000
16000
12000
8000
4000
0
2
3
4
5
6
7
8
2 MIN PER
1 MIN PER

ATTACHMENT 5

ALTITUDE TIME HISTORY

SHIP 68-218

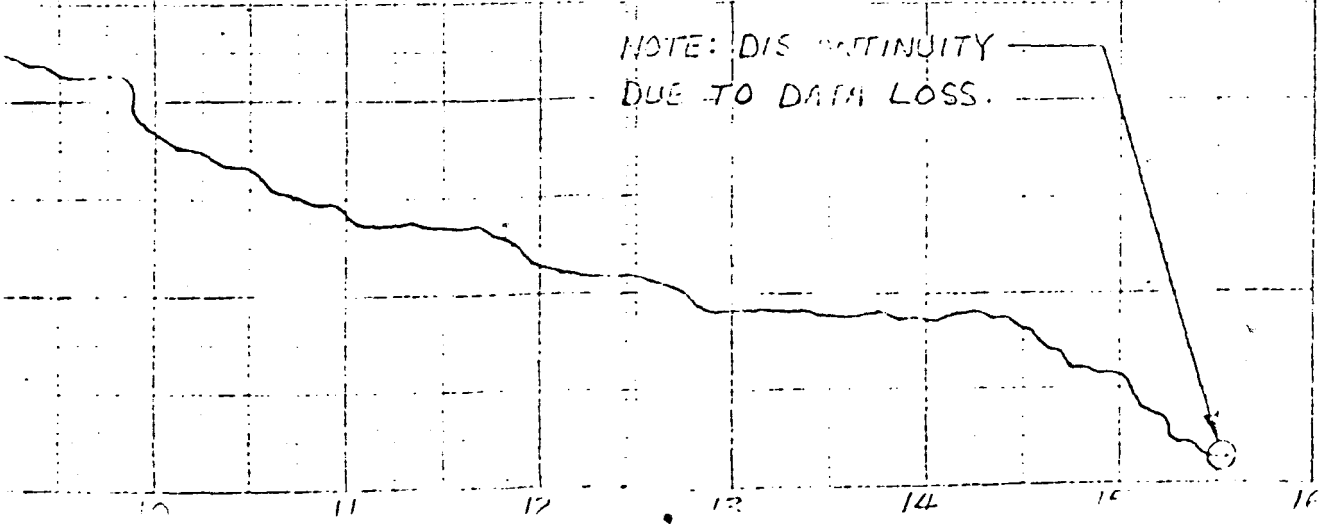
DATA SOURCE - MADAR

4 APRIL 1975

NOTE: TIME "0" IS EQUIVALENT TO MADAR TIME 5:13:18:39 WHICH WAS RAPID DECOMPRESSION.

SIGNED

J. W. EDWARDS



AGRAM
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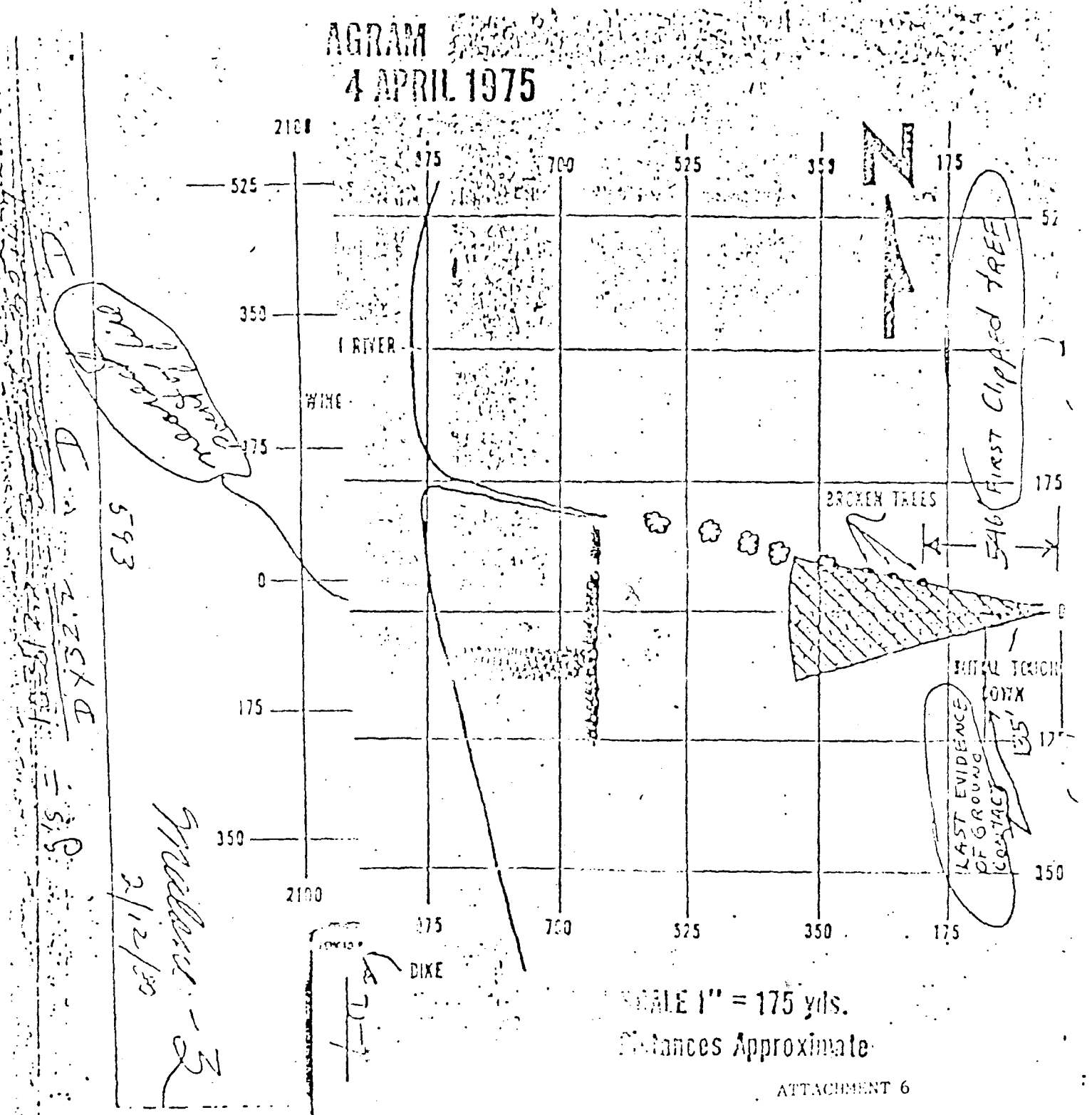

ATTACHMENT 6

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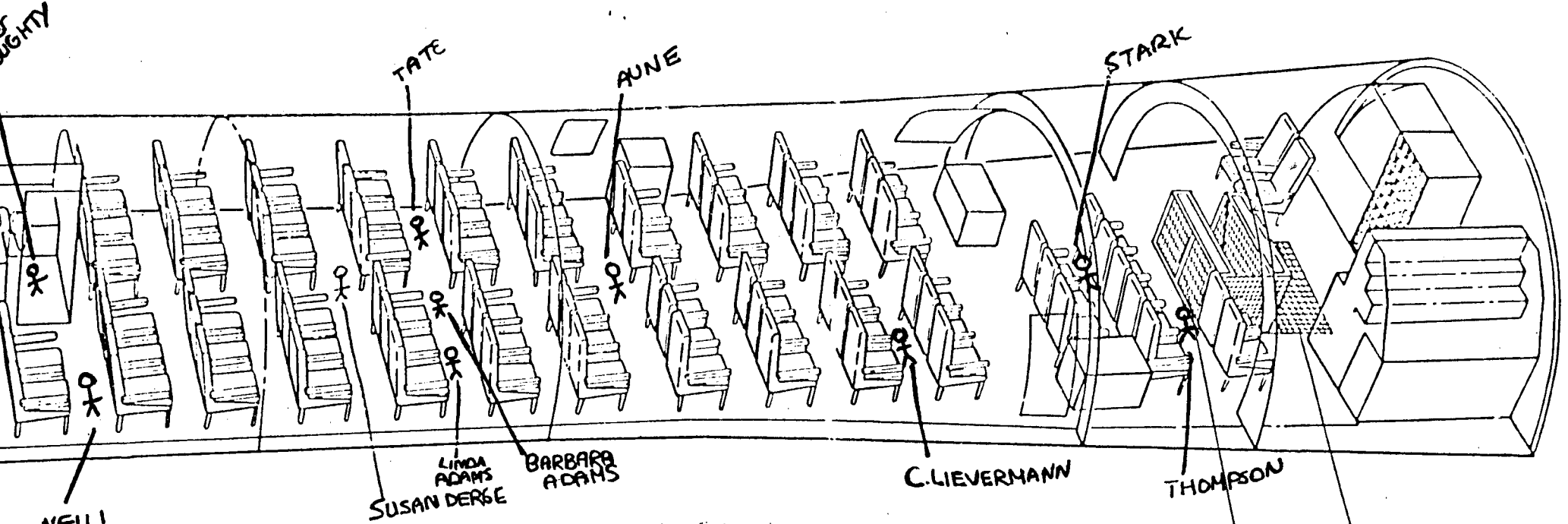
"Response of a Seat-Passenger System to Impulsive Loading," (with J.A. Collins), Proceedings of Symposium on the Dynamic Response of Structures, Pergamon Press, 1972.



(5AM-4

DEFT.EX. ~~DD~~ Bentley 7

DATE: 12/18/81

REPORTER: ALBERT J. GASDOR

J. Robert Cromack, P.E.
Cromack Engineering Association, Inc.
Post Office Box 28243
Tempe, Arizona 85282

Mr. Cromack is expected to testify regarding the calculation of "G" forces in this accident; he will describe the limitations and potentials of such calculations in the understanding of impacts and potential injury such forces can cause to the human child. Mr. Cromack will testify concerning the accident of the C5A and will indicate the limitations and difficulties encountered in proper calculation of the specific "G" forces in this accident. Mr. Cromack will specifically discuss in detail the unreasonable approach to this question by Lockheed Aircraft Corporation, as indicated by prior testimony of Mr. Edwards and Dr. Gibbons. (45 minutes)

J. Kenneth Mason, M.D.
Regius Professor of Forensic Medicine
University of Edinburgh
66 Craiglee Drive
Edinburgh, Scotland

Dr. Mason is expected to discuss aviation pathology and the relationship between the C5A accident and the pathological development of the plaintiff, as well as the other C5A surviving children that have been examined. Dr. Mason is expected to express the opinion that, based on his experience as an aviation pathologist, the method by which to evaluate the injury-producing capacity of the C5A accident environment should be conducted from the viewpoint of the current condition and immediate post-accident condition of the children on board the C5A. Dr. Mason is expected to testify about the limitations and potentials of correlating the accident environment to the understanding of the current condition of the survivors of the accident. Dr. Mason is expected to testify that the accident environment of the C5A was sufficient to cause MBD. (45 minutes)

DEFT.EX. DD-Busby Exh. 8

Douglas E. Busby, M.D.

the medical findings for the plaintiff and other children aboard the C5A. Dr. Busby is expected to discuss the relationship between explosive decompression, hypoxia, deceleration, and impact and fume and smoke inhalation and the development of brain injury in the C5A surviving children; and, specifically, Dr. Busby is expected to testify that in his opinion, the current findings in the surviving children of the C5A accident are sufficiently explained by the accident environment. Dr. Busby will testify that the C5A accident environment was sufficient to cause severe injury to multiple systems of the human body. Dr. Busby will explain in detail the methods employed in accident investigations when correlations are required, comparing the findings of the survivors with the accident environment. Dr. Busby is specifically expected to testify that the method usually employed in the evaluation of survivors of aircraft accidents precedes the correlation of such findings to the accident environments, and that following the evaluation of the survivors of a serious accident environment, one would then determine whether or not the correlation exists between the accident environment and the medical findings. Dr. Busby will testify that in his opinion the accident environment of the C5A starting at the time of the explosive decompression and ending with the impacts and crash of the C5A had sufficient physical and biomechanical forces present to cause the current findings and conditions in the surviving children of that accident. Dr. Busby is expected to testify that within reasonable medical probability, the combination of explosive decompression, hypoxia, deceleration and impact, coupled with other factors in the accident environment, such as fumes and psychological exposure, could sufficiently explain current findings in the surviving plaintiffs. (45 minutes)

C. Keith Conners, Ph.D.
Professor of Psychiatry
George Washington University Medical Center
Professor of Psychiatry and Neurology
Director of Research
Children's Hospital National Medical Center
111 Michigan Avenue, N.W.

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

$$= (303-47).2095 - 40\left(.2095 + \frac{1-.2095}{.83}\right)$$

$$= 256 \times .2095 - 40\left(.2095 + \frac{.7905}{.83}\right)$$

$$= 53.6 - 40\,(.2095 + .9524)$$

$$= 53.6 - 40\,(1.16)$$

$$= 53.6 - 46.4$$

$$= \sim 7.2 \text{ mm Hg}$$

Douglas E. Busby M.D.

12/18/81.

DEFT.EX. Busby # 9
DATE: 12-18-81
REPORTER: ALBERT J. GASDOR

DEFT.EX. DD-2546
DATE: 12/18/81
REPORTER: ALBERT J. GASDOR

E.D. - expansion gas → bradycardia

G. Troop compt

175 yards airborne

vent g 10-30

hang 7-13

stop/hill 220-480

Total pressure change 330 mm Hg

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

hyp - alt - pressure table $\dot{V}_{O_2}$ at SL 149, at 6000' 118, 4000' 127

NATO 24,000' 30 C when report. breathing altern adapt 17,000' modern aircraft

30 mm Hg

Boothby curve (from left) - P_{CO_2} little change 6000' and thickens Priority method - 10 min after reaching sampling altitude

No measurable increase in $\dot{V}$ until $P_{A_{O_2}} < 60$

Brain damage anoxic considerations

- body temp
- blood pressure - sitting, sleeping - hypovolemia
- expansion gas in gut → bradycardia
- hypoxic insult → CBF (↑ lactic acid) | will ↓/0 in parachute

(altered right)

- impact or deceleration insult / direct trauma
- toxic insult / ? further hypoxia
- further ↑ pressure
- open fontanelles distensibility of brain ventricles

blood pressure ↓ → further hypoxia.

50 mm Hg intra & intracellular pyruvate & lactate increase

Reagan - dusky appearance of children after decompression (nurse report to him)

Collect report

- stories on fire
- 150 - 9 injured, extent unknown
- large amts of mud
- heat loading
- children 'dazed' (nurse - often crash & at hospital)
- difficulties getting masks in place pulling off + not enough masks

DEFT.EX. Busby #12

DATE: 12-18-81

REPORTER: ALBERT J. GASDOR