Dr. Gaume Deposition Exhibits

re: General
Marked on September 25, 1981

D1302, D1270, Gaume Ex.#1

Original, Annotated, Extra Set

(pulled)

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

August 31, 1981

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

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

Dear Mr. Dubuc,

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

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

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

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

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

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

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

Section A of my reports deals with the decelerations experienced by the occupants of the troop compartment.. Mr. Jablonsky's calculations for the G-forces imposed during the amusement park rides are also in Attachment A-1. As you can see, the G-forces for the rides, experienced by literally thousands of amusement park patrons every year, are far in excess of those felt by the occupants of the troop compartment. In my opinion, the G-forces imposed in the G_Z (vertical) or the G_X (horizontal - transverse to the long axis of the body) were 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 recongized expert in that field, states that the pressure change is not responsible for the physiological effects of decompression, but to oxygen deprivation (see Section C-Hypoxia Effects). In my opinion, the total pressure change, the rate of change, and the duration of change, did not produce any harmful, lasting physiological effects, to a reasonable medical certainty, on anyone in the troop compartment.

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

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

Sincerely,

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

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

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

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

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

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

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

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

ATTACHMENT A-1

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

August 31, 1981

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

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

Dear Dr. Gaume:

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

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

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

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

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

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

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

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

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

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

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

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

C O N C L U S I O N

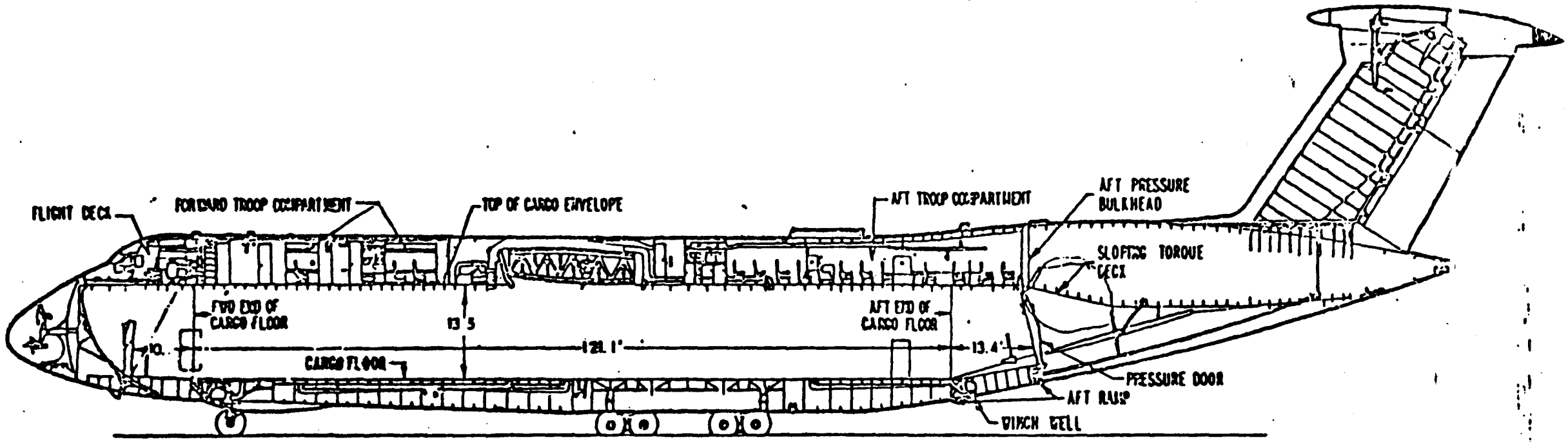
The dynamic forces experienced by the occupants in the cargo compartment during the flight descent phase were probably less than those necessary to be sensed by the occupants. During the crash-landing and the deceleration of the aircraft to the points of rest the rates of onset and the deceleration levels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

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

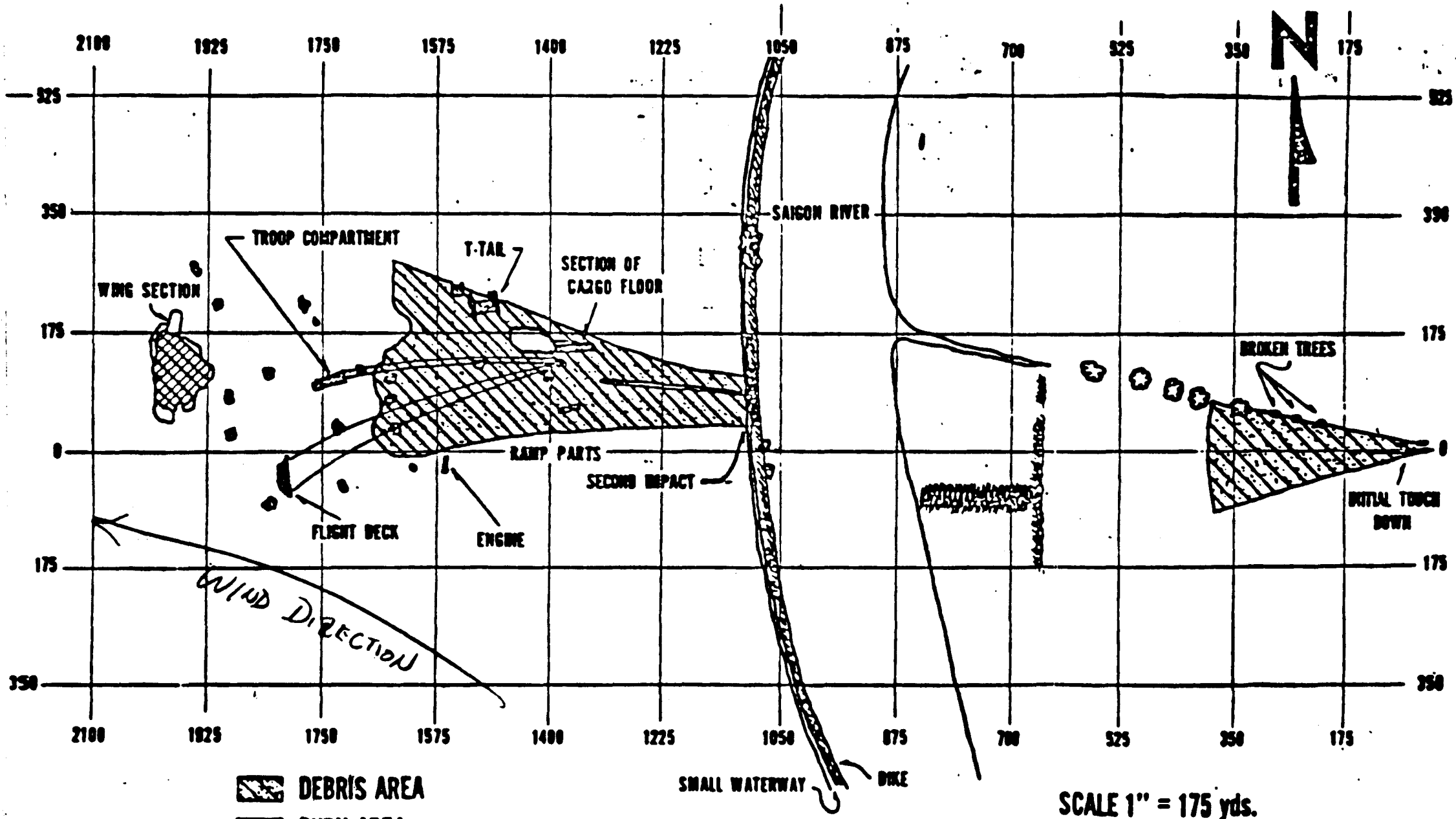
Respectfully submitted,

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

RDJ/dh


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

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



For Roy — C5A Crash - Saigon 1975

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

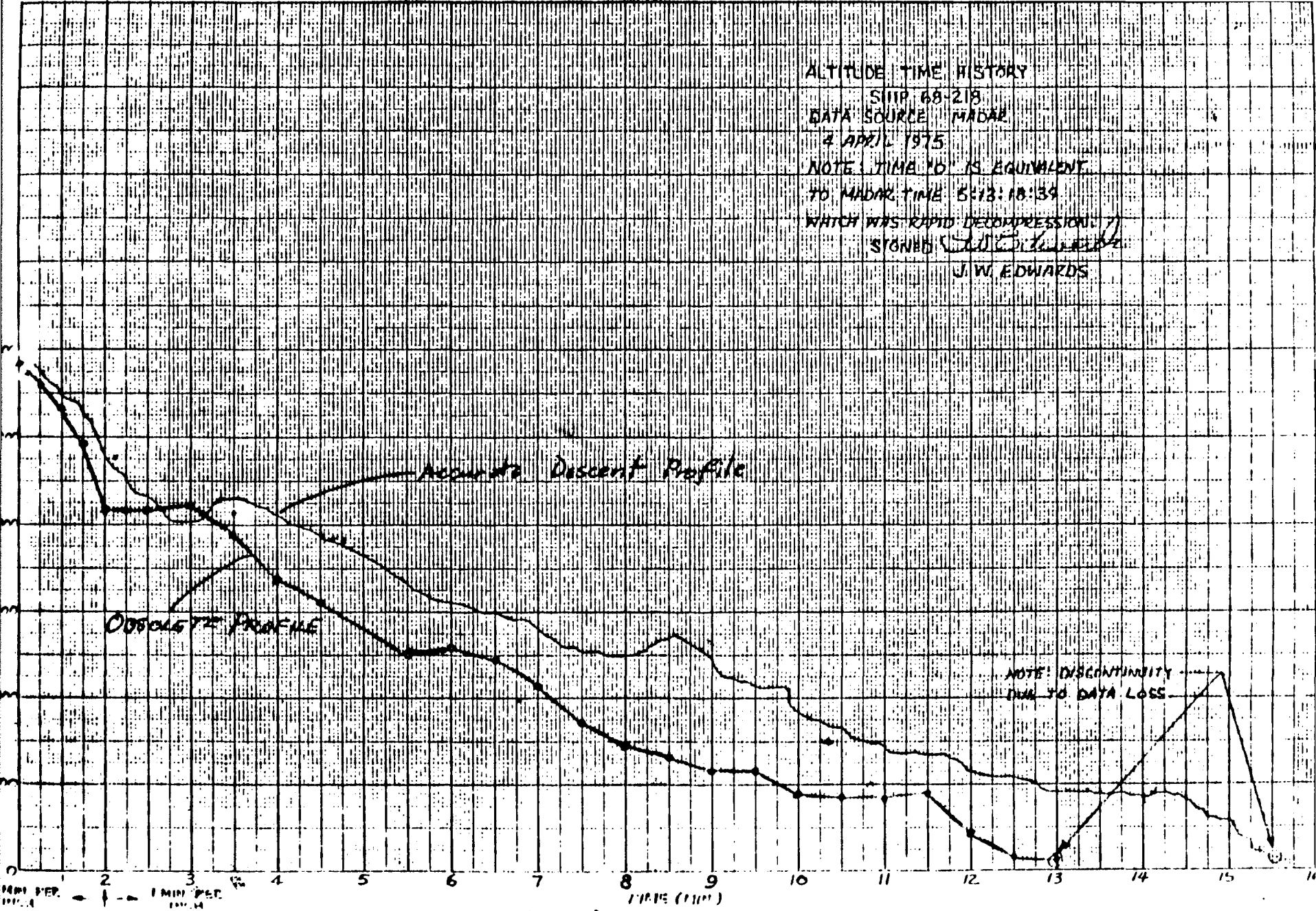
2. Descent rate (prior to flare) = 500 - 600 ft/min
Flare due to air cushion (ground effect) under wing

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

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

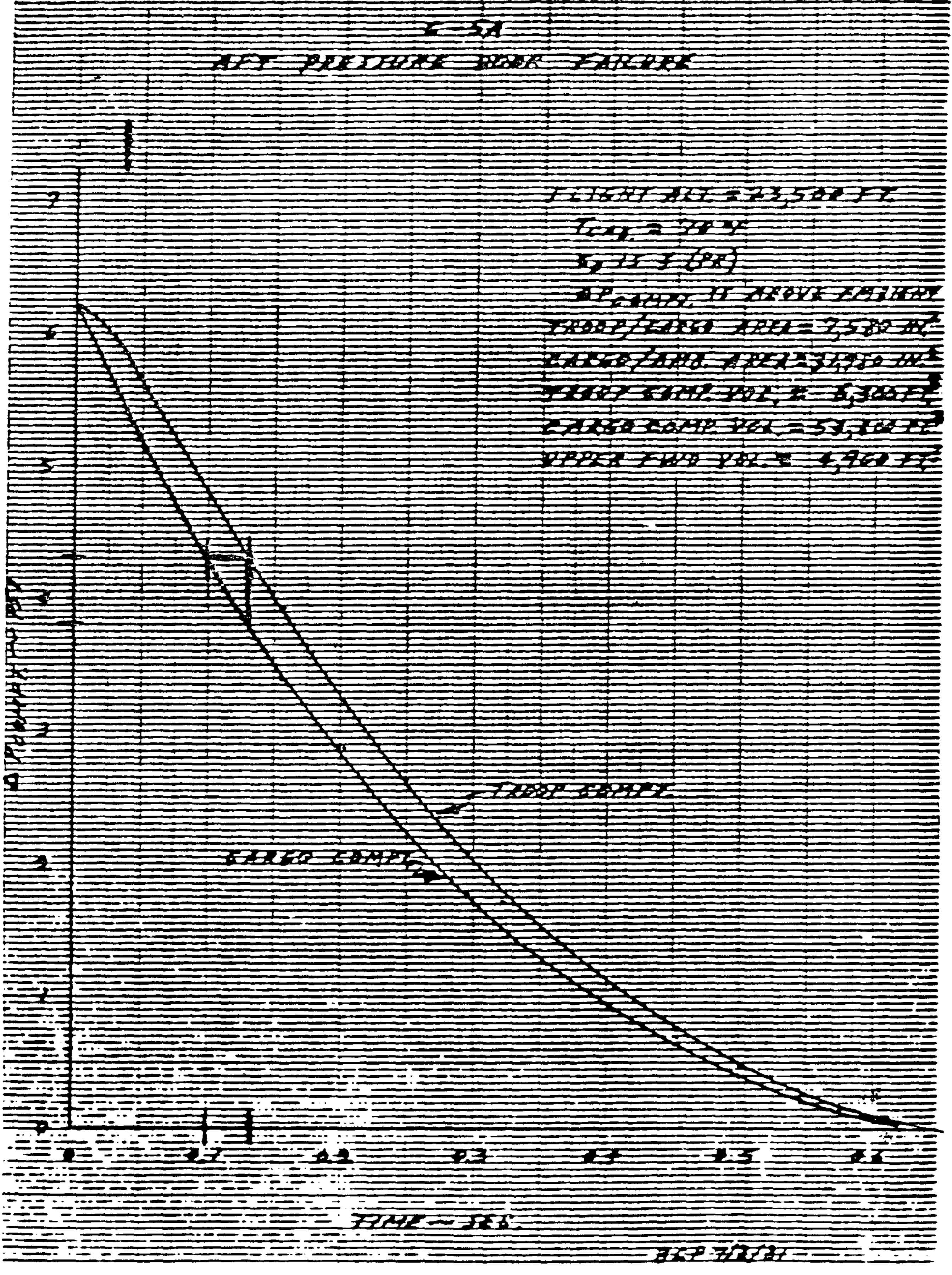
5. Aircraft Wt =

6. Troop Compartment Wt - Unknown



Av. Descent Rate 1st 3 min = 2,400 ft/min (approx)
3-8 min = 1,035 ft/min (")
8-11(16) = 1,300 ft/min (")

Attach B F-72-05-657-8/

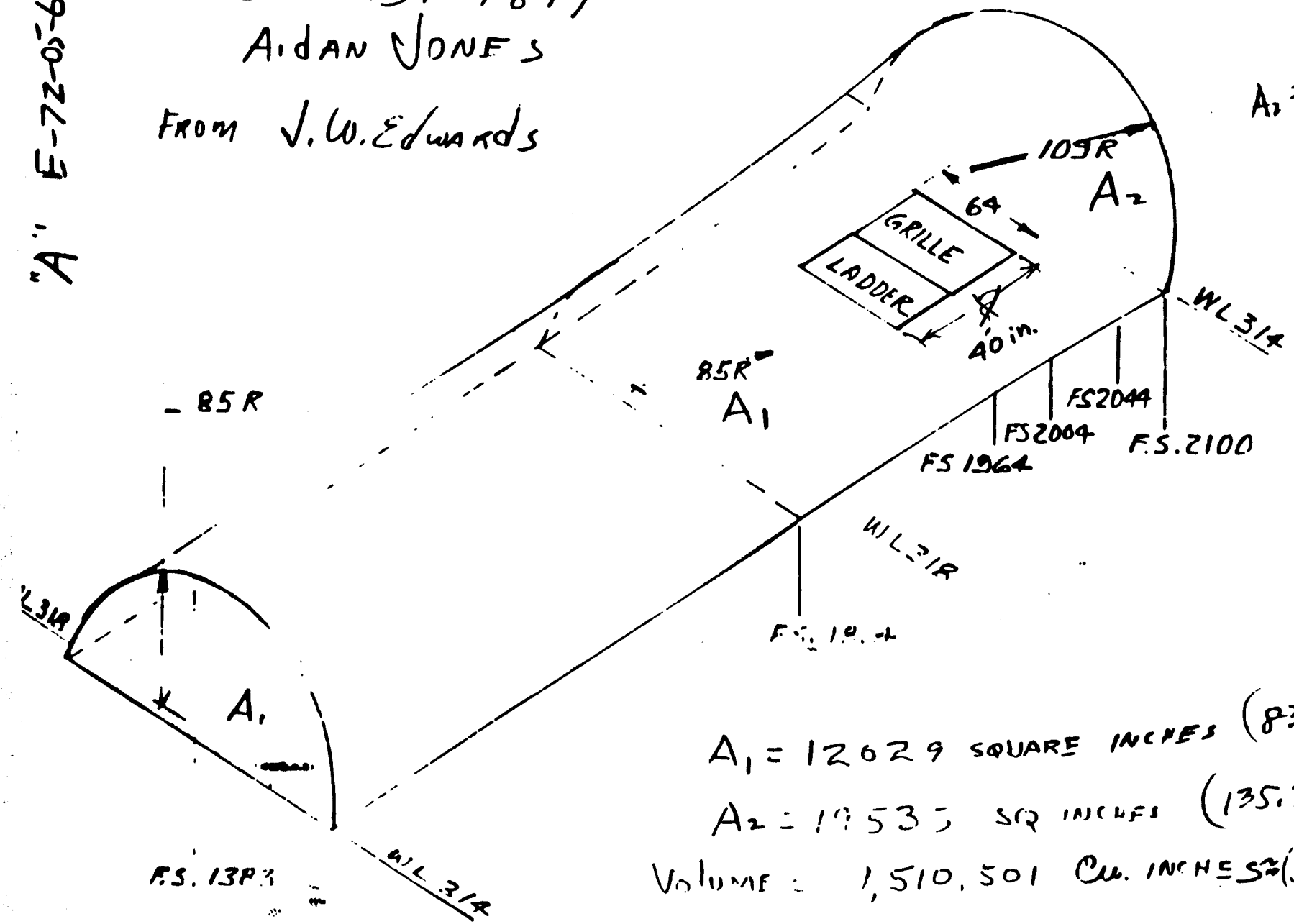


APPENDIX A

"A" E-72-05-637-B

To:
202-737-7849
AIDAN JONES

FROM J. W. Edwards



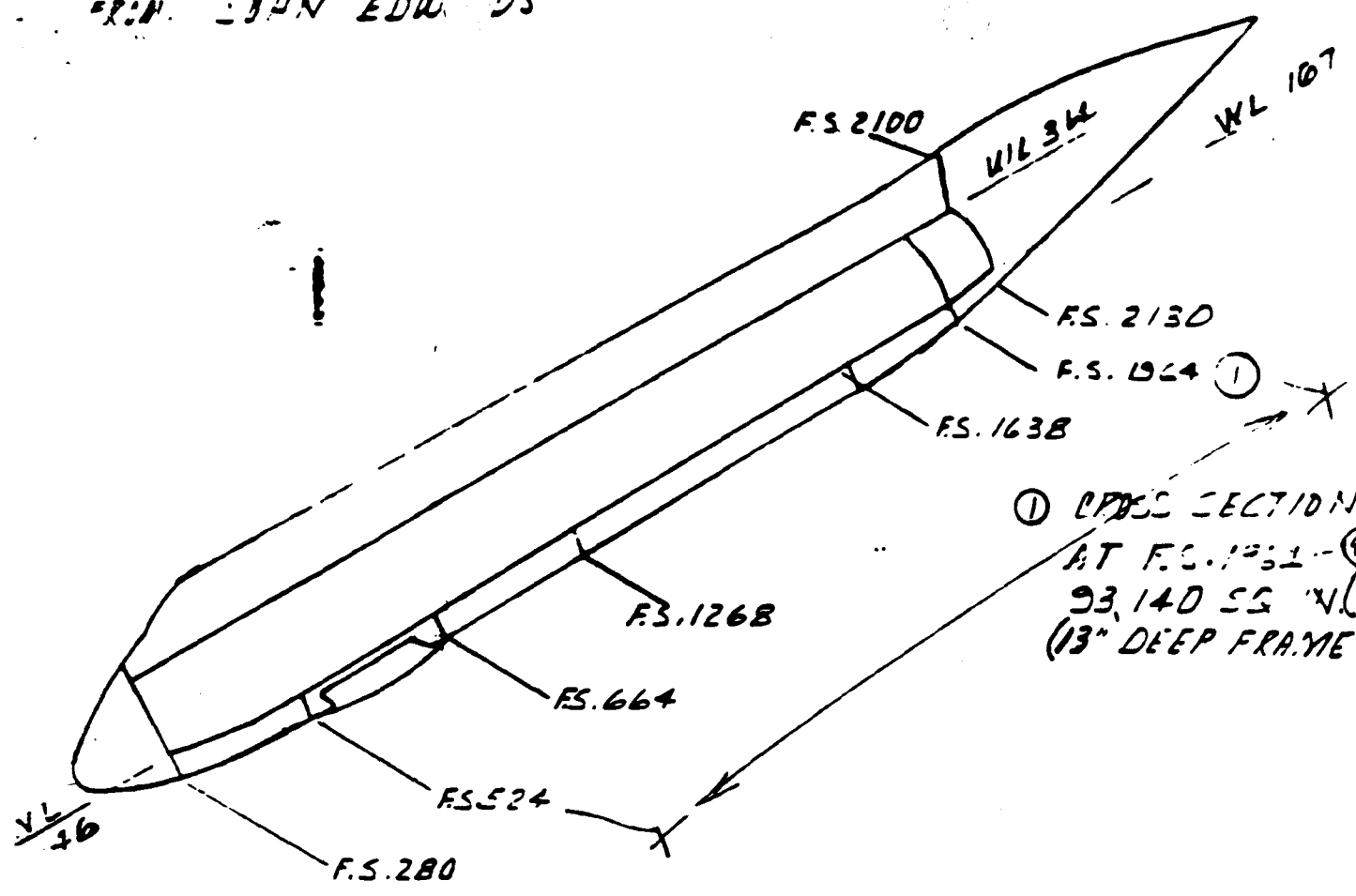
A_1 = 120.29 SQUARE INCHES (83.5 #) 1:1.6

A_2 = 195.35 SQ INCHES (135.7 #)

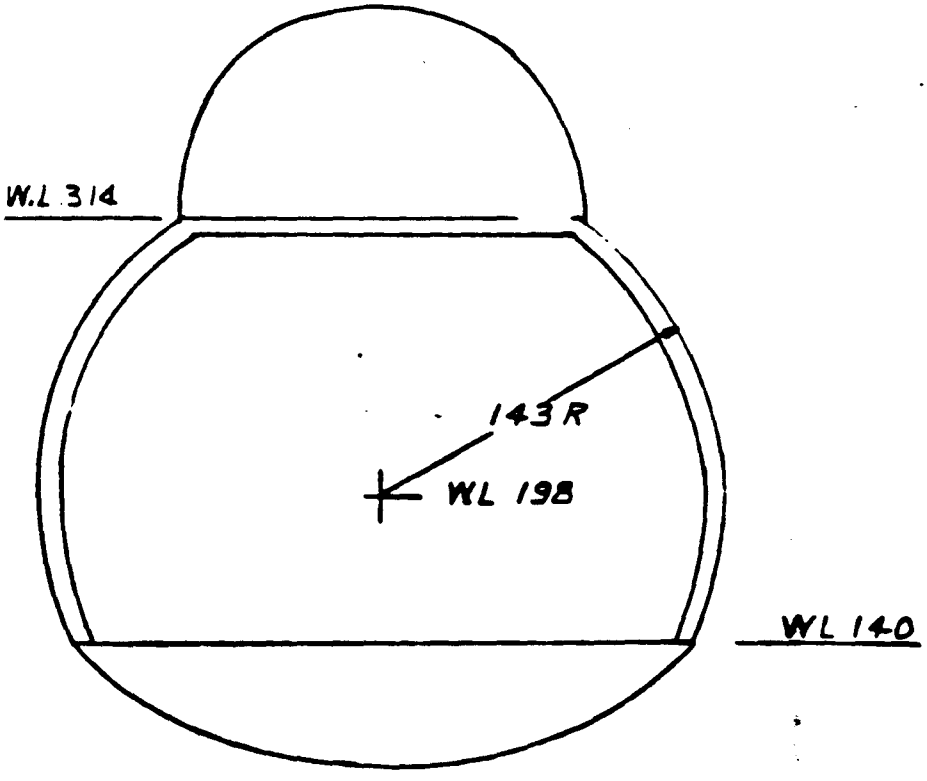
Volume = 1,510,501 Cu. INCHES ≈ (875 cuft)

AFT TROOP COMPARTMENT

FROM JOHN EDWARDS



1 CROSS SECTION AREA AT F.S. 1931 - 961 ?
93.140 SQ IN. (647 #)
(13" DEEP FRAME)

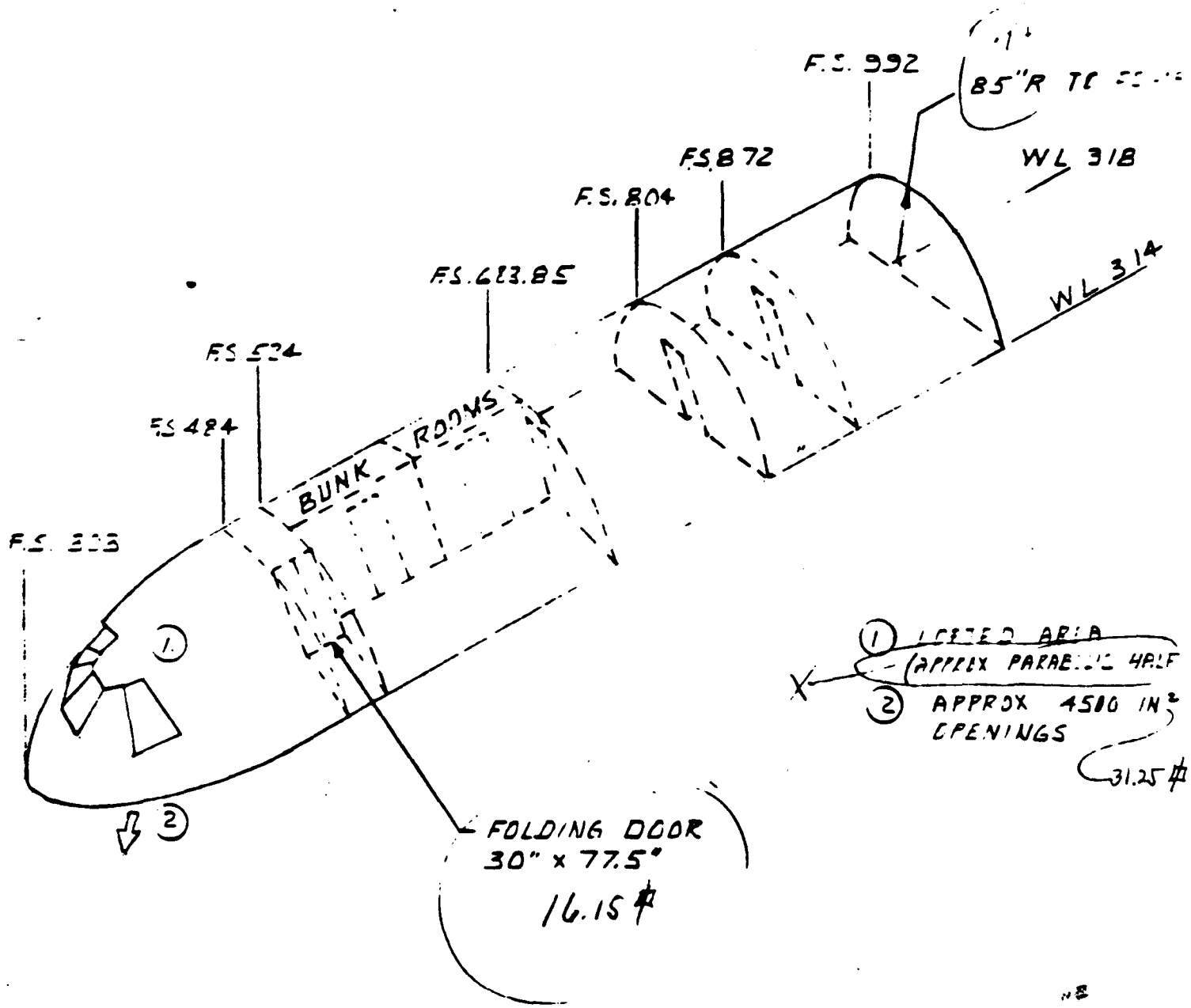


CONSTANT F.S. 524 TO 1964

CARGO COMPARTMENT

TO: 202-767-7629
AIDAN JONES
FROM JOHN EDWARDS

A E-7205-657-8



C-5A FLIGHT STATION

ATTACHMENT 1

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC	ACCELERATION FT/SEC^2	ONSET RATE g/SEC
3.38	202.5	17100	-13.33	-3.56	0
3.50	210.0	17200	13.33	3.56	0.03
3.63	217.5	17100	26.67	1.78	0.007
3.75	225.0	16900	13.33	-1.78	0.01
3.88	232.5	16800	53.33	+5.33	0.03
4.00	240.0	16400	46.67	-0.89	0.02
4.13	247.5	16050	20.00	-3.56	0.02
4.25	255.0	15900	13.33	-0.89	0.03
4.38	262.5	15800	53.33	5.33	0.04
4.50	270.0	15400	20.00	-4.44	0.02
4.63	277.5	15250	20.00	0	0.01
4.75	285.6	15100	40.00	2.67	0.02
4.88	292.5	14800	20.00	-2.67	0.03
5.00	300.0	14650	46.67	3.56	0.02
5.13	307.5	14300	40.00	-0.89	0.01
5.25	315.0	14000	53.33	1.78	0.01
5.38	322.5	13600	40.00	-1.78	0.01
5.50	330.0	13300	46.67	0.89	0.01
5.63	337.5	12950	40.00	-0.89	0.01
5.75	345.0	12650	20.00	-2.67	0.01
5.88	352.5	12500	13.33	-0.89	0.01
6.00	360.0	12400	6.67	-0.89	0.01
6.13	365.5	12350	20.00	1.78	0.01
6.25	375.0	12200	26.67	0.89	0.02
6.38	382.5	12000	0	-3.56	0.03
6.50	390.0	12000	26.67	3.56	0.02
6.63	397.5	11800	13.33	-1.78	0.01
6.75	405.0	11700	13.33	0	0.02
6.88	412.5	11600	53.33	5.33	0.02
7.00	420.0	11200	53.33	0	0.01
7.13	427.5	10800	40.00	-1.78	0.01
7.25	435.0	10500	13.33	-3.56	0.02
7.38	442.5	10400	33.33	2.56	0.03
7.50	450.0	10150	0	-4.44	0.02
7.63	457.5	10150	13.33	1.78	0.01
7.75	465.0	10050	6.67	-0.89	0.01
7.88	472.5	10000	0	-0.89	0.01

VERTICAL VELOCITY, ACCELERATION AND RATE OF ON SET CALCULATION FROM ALTITUDE HISTORY OF SHIP 68-218, GRAPH OF J W EDWARDS

NUMERICAL DIFFERENTIATIONS BASE UPON 7.5 SECOND TIME INTERVALS

(MINUS INDICATES VERTICALLY UP)

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC.	ACCELERATION FT/SEC^2	ON SET RATE g/SEC
0	0	23300	−6.67	−.89	—
.13	7.5	23350	−6.67	0	0.004
.25	15.0	23400	13.33	2.67	0.01
.38	22.5	23300	40.00	3.56	0.004
.50	30.0	23000	26.67	−.78	0.02
.63	37.5	22800	40.00	1.78	0.01
.75	45.0	22500	26.67	−1.78	0.01
.88	52.5	22300	53.33	3.55	0.02
1.00	60.0	21900	13.33	−5.33	0.04
1.13	67.5	21800	26.67	1.78	0.10
1.25	75.0	21600	26.67	0	0.007
1.38	82.5	21400	53.33	3.55	0.01
1.50	90.0	21000	26.67	−3.55	0.03
1.63	97.5	20800	66.67	5.33	0.04
1.75	105.0	20300	93.33	3.55	0.007
1.88	112.5	19600	66.67	−3.55	0.03
2.00	120	19100	73.33	0.89	0.02
2.13	127.5	18550	60.00	−1.78	0.01
2.25	135.0	18100	93.33	4.44	0.03
2.38	142.5	17400	33.33	−8.00	0.05
2.50	150.0	17150	46.67	1.78	0.04
2.63	157.5	16800	73.33	3.55	0.007
2.75	165.0	16250	13.33	−8.00	0.05
2.88	172.5	16150	0	−1.78	0 03
3.00	180.0	16150	−20.00	2.90	0.02
3.13	187.5	16300	−66.67	6.22	0.01
3.25	195.0	16800	−40.00	−3.56	0.04

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	VELOCITY FT/SEC	ACCELERATION FT/SEC^2	ONSET RATE g/SEC
8.00	480.0	10000	0	0	0.01
8.13	487.5	10100	−13.33	1.78	0
8.25	495.0	10300	−26.67	1.78	0
8.38	502.5	10600	−40.00	1.78	0
8.50	510.0	10800	−26.67	−1.78	0.01
8.63	517.5	10800	0	−3.56	0.02
8.75	525.0	10500	40.00	5.33	0.04
8.88	532.5	10200	40.00	0	0.02
9.00	540.0	9700	66.67	3.56	0
9.13	547.5	9000	93.33	3.56	0.06
9.25	555.0	8900	13.33	−10.67	0.04
9.38	562.5	8800	13.33	0	0.01
9.50	570.0	8600	26.67	1.78	0.01
9.63	577.5	8500	13.33	−1.78	0.02
9.75	585.0	8550	−6.67	−2.67	0.07
9.88	592.5	7800	100.00	14.22	0.07
10.00	600.0	7150	86.67	−1.78	0.03
10.13	607.5	7050	13.33	−9.78	0.04
10.25	615.0	6950	13.33	0	0.01
10.38	622.5	6700	33.33	2.67	0.03
10.50	630.0	6650	6.67	3.55	0.01
10.63	637.5	6200	60.00	7.11	0.01
10.75	645.0	6050	20.00	−5.33	0.05
10.88	652.5	5900	20.00	0	0.02
11.00	660.0	5700	26.67	0.89	0.01
11.13	667.5	5450	33.33	0.89	0
11.25	675.0	5450	0	−4.44	0.02
11.38	682.5	5500	−6.67	0.89	0.02
11.50	690.0	5400	13.33	2.67	0.01
11.63	697.5	5400	0	−1.78	0.01
11.75	705.0	5300	13.33	1.78	0.01
11.88	712.5	5000	40.00	3.56	0.01
12.00	720.0	4650	46.67	0.89	0.01
12.13	727.5	4400	33.33	−1.78	0.01
12.25	735.0	4350	6.67	−3.55	0.02
12.38	742.5	4350	0	−0.89	0.01
12.50	750.0	4300	6.67	0.89	0.01

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

NUMERICAL VALUES OF ALTITUDE AND O_2 PARTIAL PRESSURE

BASED ON ALTITUDE TIME HISTORY --- SHP 68-2-8

GRAPH OF J W EDWARDS

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	TOTAL PRESSURE mm Hg.	PARTIAL PRESSURE O_2 mm Hg.
0	0	23300	303.59	63.60
.13	7.5	23350	302.94	63.47
.25	15.0	23400	302.26	63.32
.38	22.5	23300	303.59	63.60
.50	30.0	23000	307.53	64.43
.63	37.5	22800	310.18	64.98
.75	45.0	22500	314.19	65.82
.88	52.5	22300	316.88	66.39
1.00	60.0	21900	322.33	67.53
1.13	67.5	21800	323.70	67.82
1.25	75.0	21600	326.47	68.39
1.38	82.5	21400	329.24	68.98
1.50	90.0	21000	334.86	70.15
1.63	97.5	20800	337.70	70.75
1.75	105.0	20300	334.88	72.25
1.88	112.5	19600	355.15	74.40
2.00	120.0	19100	362.62	75.97
2.13	127.5	18550	370.84	77.69
2.25	135.0	18100	377.97	79.18
2.38	142.5	17400	389.01	81.50
2.50	1500	17150	393.45	82.43
2.63	157.5	16800	398.69	83.53
2.75	165.0	16250	407.92	85.46
2.88	172.5	16150	409.70	85.83
3.00	180.0	16150	409.70	85.83
3.13	187.5	16300	406.91	85.25
3.25	195.0	16800	398.69	83.53
3.38	202.5	17100	393.83	82.51
3.50	210.0	17200	392.32	82.17
3.63	217.5	17100	393.83	82.51

TIME MINUTES	TME SECONDS	ALTITUDE FECT	TOOL PRESSURE mm Hg	PARTIAL PRESSURE O_2 mm Hg.
3.75	225.0	6900	397 07	83 19
3.88	232.5	16800	398 69	83 53
4.0	240.0	16400	405.25	84 90
4.13	247.5	16050	410.97	86 10
4.25	255.0	15900	413.57	86.64
4.38	2625	15800	415.25	87 00
4.50	270.0	15400	422.03	88 42
4 63	277.5	15250	424.43	88 92
4.75	285.5	15100	427.17	89 49
4.88	292.5	14800	432.37	90 58
5.00	300.0	14650	434.85	91.10
5.13	307.5	14300	441.13	92.42
5.25	315.0	14000	446.46	93.53
5.38	322.5	13600	453 65	95.04
5 50	330.0	13800	459.10	96.18
5.63	337.5	12950	465.58	97.54
5.75	345.0	12650	47066	98.60
5.88	352.5	12500	473.90	99.28
6.00	360.0	12400	475 78	99 68
6 13	365.5	12350	476 76	99.98
6 25	375.0	12200	479.55	100.47
6.38	382.5	12000	483.34	101.24
6 50	3900	12000	48334	101.26
6 63	397.5	11800	487.16	102.06
6.75	405.0	11700	489.08	102.46
688	412.5	11600	491.01	102.87
7.00	420.0	11200	491.91	103.06
7 13	427.5	10800	506.63	106.14
7.25	435,0	10500	512 59	107 39
7.38	442.5	10400	514.59	107.81
7.50	450.0	10150	519.68	108.87
7.63	457.5	10150	519.68	108.87
7.75	465.0	10050	52172	109 30
7.88	472.50	10000	522 65	109 50
8 00	4820	10000	522 65	109.50
8.13	487.5	10100	520.63	109 07

TIME MINUTES	TIME SECONDS	ALTITUDE FEET	TOTAL PRESSURE mm Hg	PARTIAL PRESSURE O_2 mm Hg
8.25	495.0	10300	516.60	108.23
8 38	502.5	10600	510.60	106.97
8.50	510.0	10800	506.63	106.14
8 63	517 5	10800	506. 63	106. 14
8.75	525.0	10500	512 59	107 39
8.88	532.5	10200	518.61	108 65
9.00	540.0	9700	528.77	110.78
9.13	547.5	9000	543.26	113.81
9.25	555.0	8900	545.35	114.25
9.38	562.5	8800	547.46	114.69
9.50	570.0	8600	551.68	115. 58
9.63	577.5	8500	553.80	116.02
9.75	585.0	8550	552.70	115.79
9.88	592.5	7800	568.64	119.17
10.00	600.0	7150	583.18	112. 18
10.13	607.5	7050	585.47	112. 66
10.25	615.0	6950	587.76	123. 13
10.38	622.5	6700	593.15	124. 26
10.50	630.0	6650	594.36	124.52
10.63	637.5	6200	604.47	126.64
10.75	645.0	6050	607.82	127.34
10.88	652.5	5900	611.34	128.08
11.00	660.0	5700	615.97	129.04
11.13	667.5	5450	621.54	1302
11.25	675.0	5450	621 54	130.21
11.38	682.5	5500	620.61	130.02
11.50	690.0	5400	622.95	130.51
11.63	697.5	5400	622.95	13051
11.75	705.0	5300	625.29	131.00
11.88	712.5	5000	632.36	132.48
12.00	720.0	4650	640.59	134.20
12.13	727.5	4400	646 68	135.48
12.25	735.0	4350	647.95	135.75
12.38	742.5	4350	647 95	135.75
12.50	750.0	4300	649.10	135 99
12.63	757.5	4200	651.52	136. 49
12.75	765.0	4050	655.07	137 24

TIME MINUTES	TIME SECONDS	ALTITUDE ~~FEET~~	TOTAL PRESSURE mm Hg	PARTIAL PRESS O_2 mm Hg
12.88	772.50	3700	663.73	139.05
13.00	780.0	3650	664.97	139.31
13.13	787.5	3650	664.97	139.31
13.25	795.0	3650	664 97	139.31
13 38	802.50	3650	664 97	139.31
13.50	810.0	3550	667.22	139.79
13.63	817.5	3550	669.22	139.79
13.75	825.0	3450	669.80	140.32
13.88	832.5	3450	669.80	140.32
14.00	840.0	3400	671.15	140.6
14.13	847.5	3550	669.26	139.79
14.25	855.0	3600	666.20	139.57
14.38	862.5	3550	669.26	139.79
14 50	870.0	3300	673.64	141.13
14.63	877.5	2850	684.78	143.46
14.75	885.0	2450	694.94	145.59
14.88	892.5	2400	696.36	145.89
15.00	900.0	2350	697.48	146.12
15.13	907.5	1550	718.31	150.49
15.25	915.0	1350	723 90	151.06
15.38	922.5	800	738.28	154.67
15.50	930.0	550	745.02	156.08

ATTACHMENT 2

DECELERATION ANALYSIS

FROM 2ND GROUND CONTACT TO POINT OF REST

PLANE BREAKUP OCCURRED 1200' FROM 2ND GROUND CONTACT.

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

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

BASED UPON CONSTANT DECELERATION RATE

$V^2 = 2as$

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

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

V = VELOCITY FT/SEC

a = DECELERATION FT/SEC^2

S = DISTANCE FT

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

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

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

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

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

V_I = IMPACT VELOCITY

V_B = BREAKUP VELOCITY

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

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

$V_b = 172$ FT/SEC (117 mph)

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

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

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

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

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

S = DISTANCE TRAVELED

a = DECELERATION RATE

t = TIME

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

$t = 9.44$ SECONDS

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

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

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

$$T = t_B + t_A$$

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

t_B = TIME BEFORE BREAKUP

t_A = TIME AFTER BREAKUP

NOTE:

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

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

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

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

THUS PLANE WAS AIR BORNE FOR 1700'

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

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

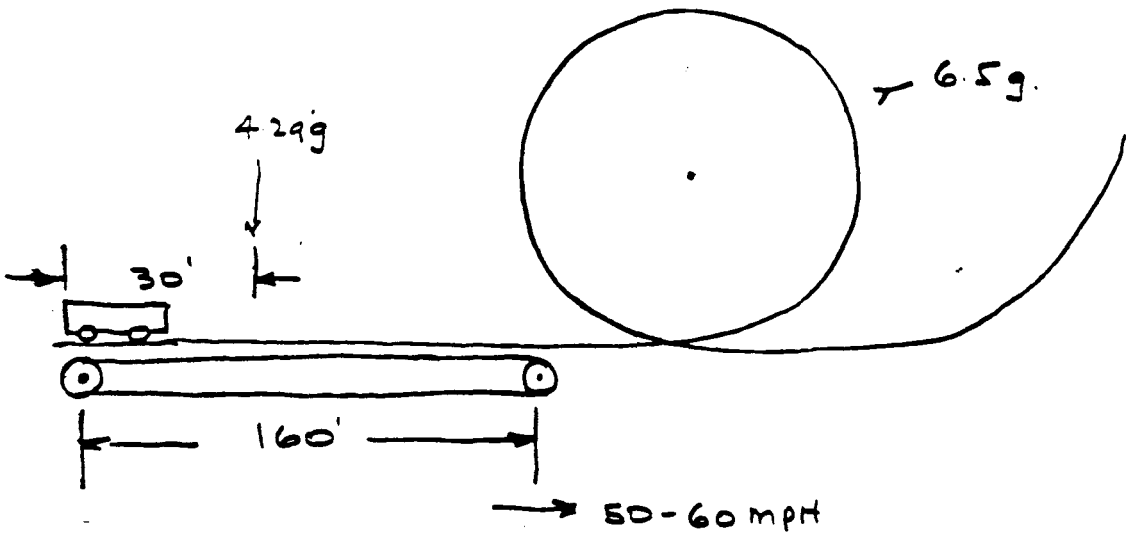
THE PLANE WAS AIRBORE

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

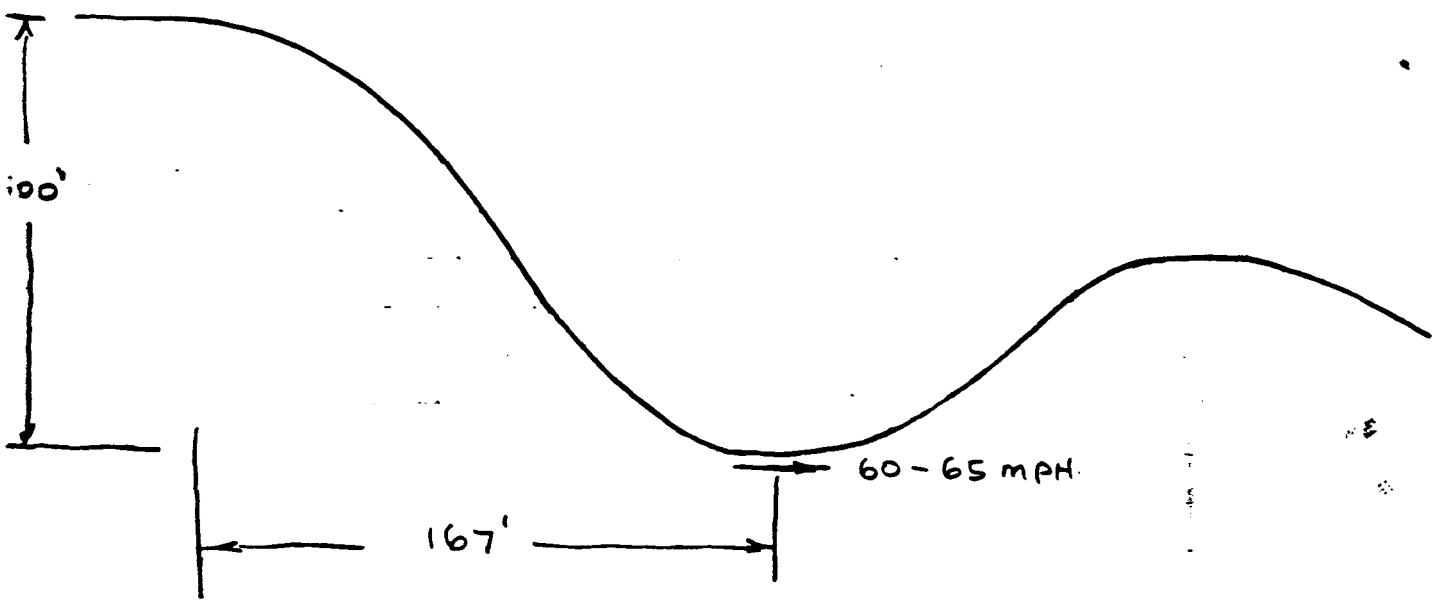
ATTACHMENT 3

AMUSEMENT RIDE ANALYSIS

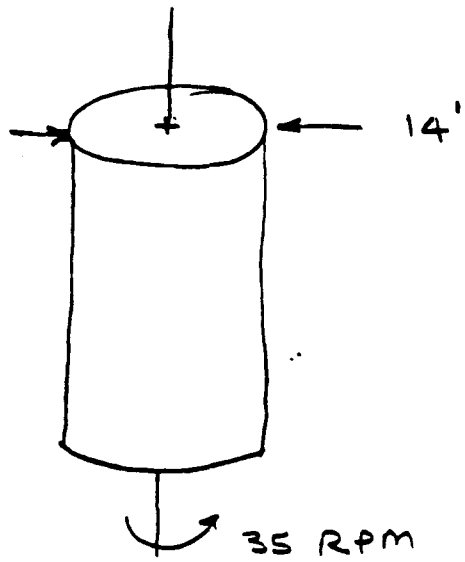
MONTEZUMA'S REVENGE



ROLLER COASTER



ROTOR



$14\pi = 43.98'$ CIRCUM

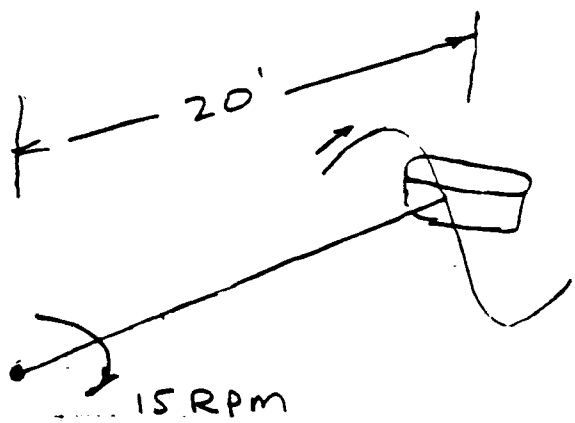
$\frac{43.98}{1.72} = 25.51$ FT/SEC

(0.58 RPS)
1.78 SEC/REV

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

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

ROUND-UP



$40\pi = 125.66'$ CIRCUMFERENCE

15 RPM = 0.25 RPS = 4 SECONDS/REVOLUTION

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

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

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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.

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30 August 1981

ATTACHMENT B-1, J.C.G.

aero-space medicine

EDITED BY

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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

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

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

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

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

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

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

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Human Factors Consultant
30 August 1981

ATTACHMENT C-1, J.G.G.

AB DATA SHEET

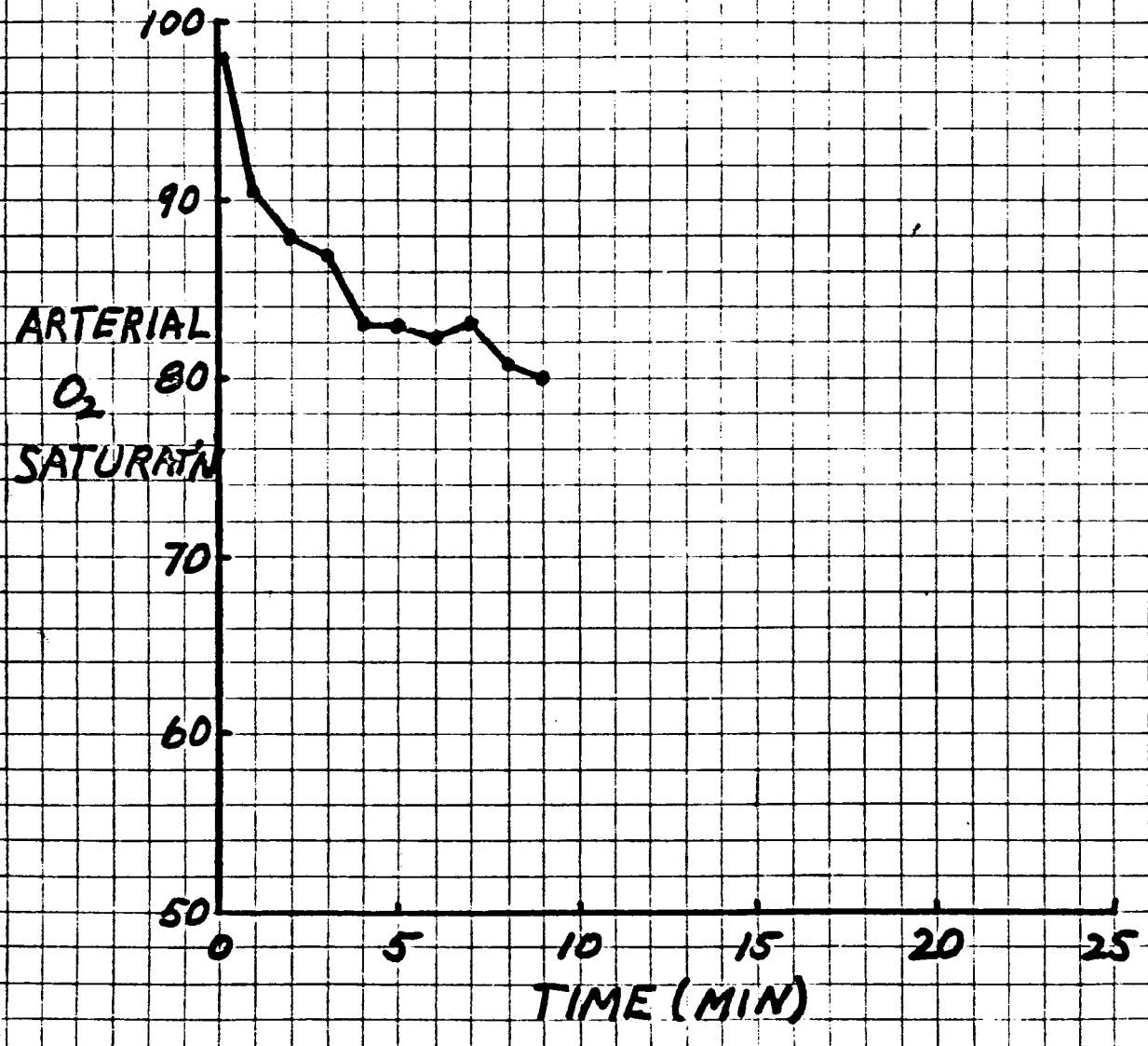
NAME: JGG. AGE: 65

DATE: 4-6-81

HOUR: 1200

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

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



ATTACHMENT C-2, JGG

AB DATA SHEET

NAME: J.G.G. AGE: 65

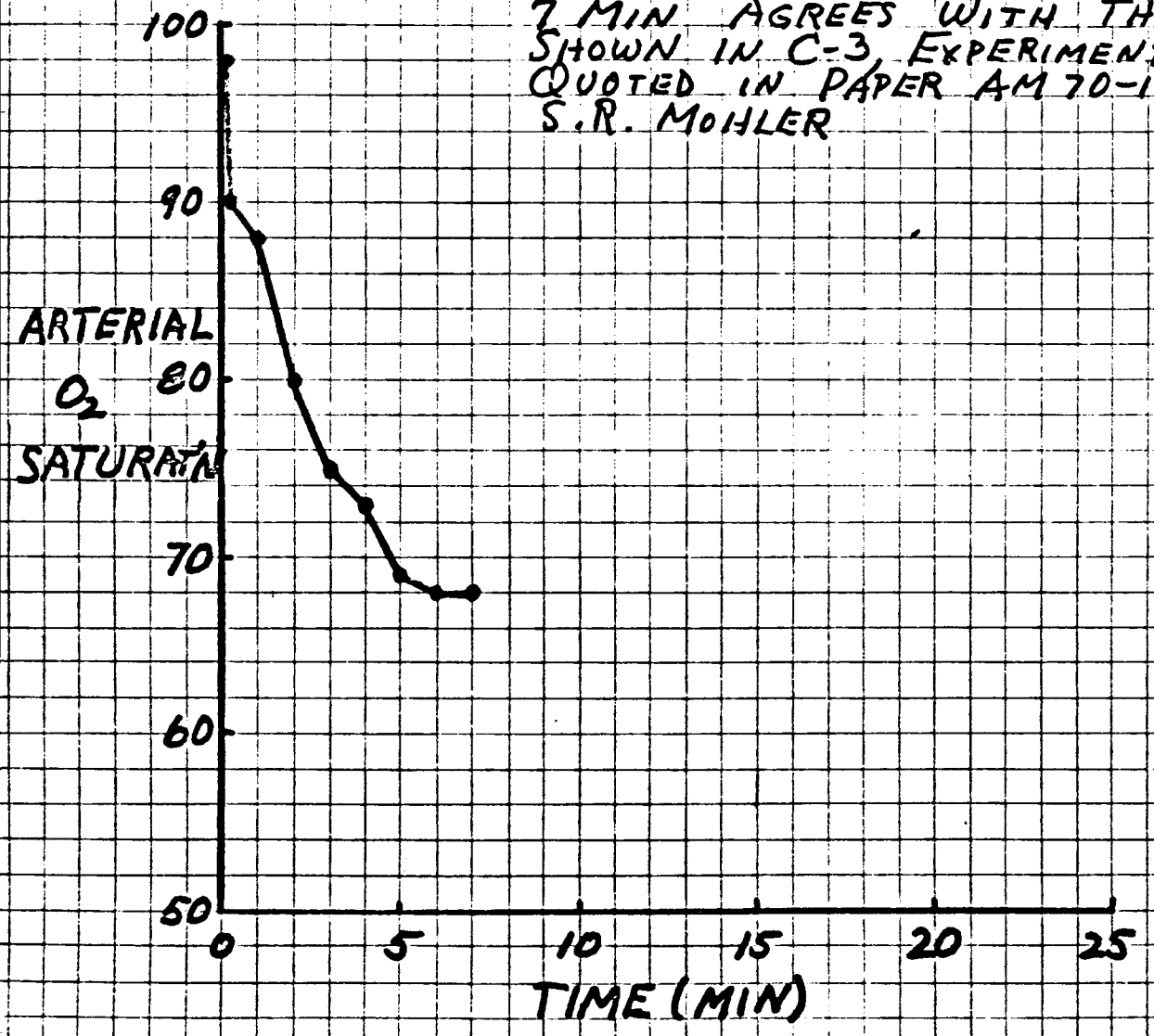
DATE: 4-6-81

HOUR: 1230

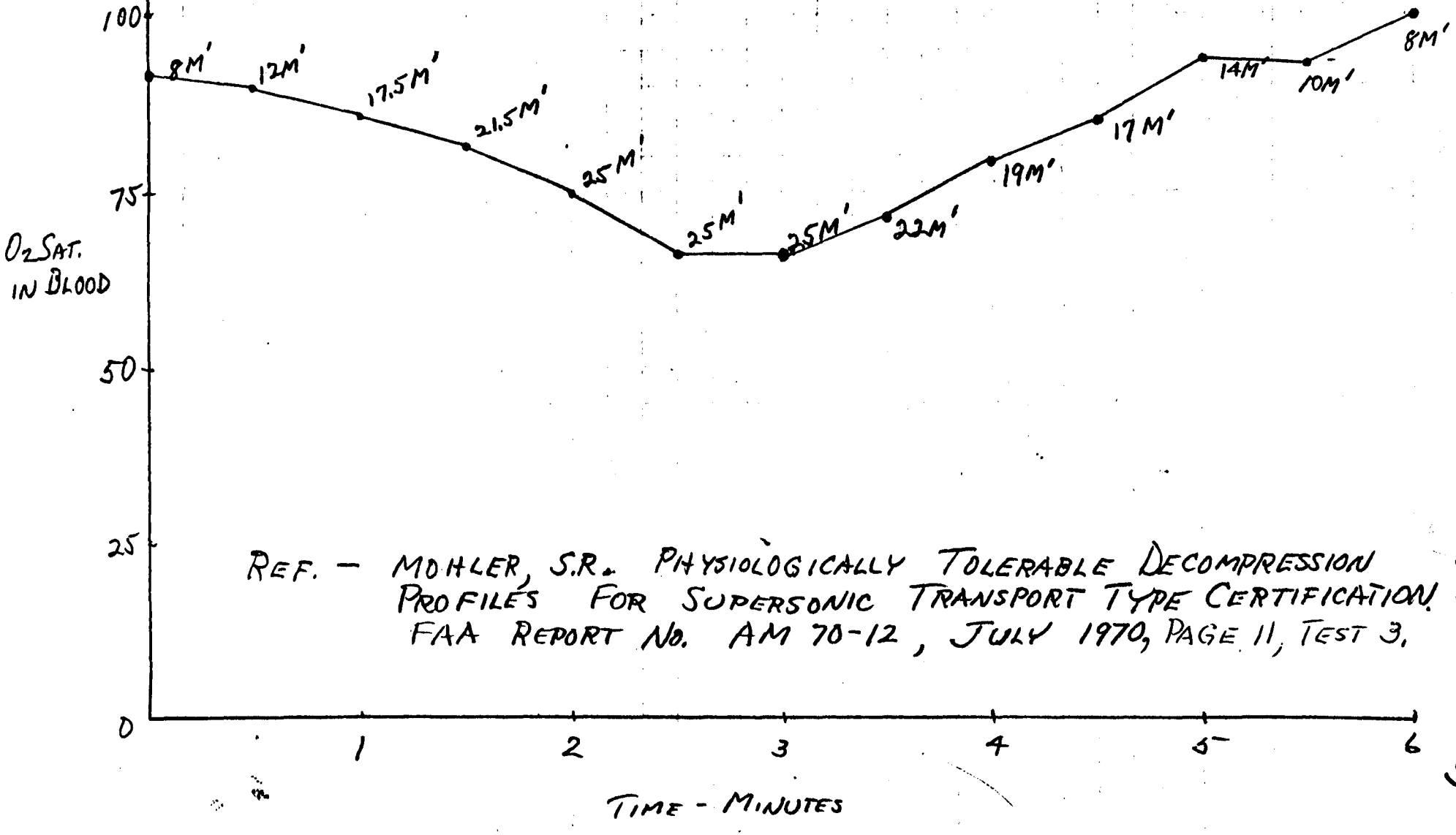
GAS MIXTURE: 33% ROOM AIR (7.5% O_2 EQUIVALENT,
67% N_2 (92.5% N_2)
24,000 FT. EQUIVALENT ALTITUDE

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



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ATTACHMENT C-3, J.G.G.

James G. Gaume, M.D.

ATTACHMENT C-4

HANDBOOK OF RESPIRATORY PHYSIOLOGY

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

RESPIRATORY PHYSIOLOGY IN AVIATION

Edited by

WALTER M. BOOTHBY, M. D.

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

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

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

Charles A. Lindbergh

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

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

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

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

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

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

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

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

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

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

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

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

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

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

ATTACHMENT C-5

EDWARD J. VAN LIERE
J. CLIFFORD STICKNEY

HYPOXIA

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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); Crile and Dolley (25) in 1908; and Pike, Guthrie, and Stewart (78) also in 1908. Others have reported studies on the effect of acute anemia on nervous centers (4, 16, 17, 25, 28, 39, 43, 50, 60, 61, 72, 77, 94, 95, 103).

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

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

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

TABLE 10

SURVIVAL TIME OF DIFFERENT NERVE TISSUES COMPLETELY DEPRIVED OF BLOOD*

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

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

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

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

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

TABLE 11

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

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

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

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

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

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

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

HISTOLOGIC STUDIES OF STRUCTURAL CHANGES

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

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

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

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

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

ABILITY OF YOUNG ANIMALS TO WITHSTAND ASPHYXIA AND HYPOXIA

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

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

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

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

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

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

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

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

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

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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.

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Professional Affiliations

Registered Professional Engineer California,	I 003775	Industrial Engineering
Registered Professional Engineer California,	MT001611	Metallurgical Engineering
Registered Professional Engineer California,	SF003109	Safety Engineering
Registered Professional Engineer Missouri,	1276	Engineering
Certified Safety Professional	3869	

Education

Washington University, B.S. (Electrical Engineering)	1940
West Coast University, M.S. (Systems Engineering)	1969
West Coast University, M.S. (Computer Systems)	1970
University of Southern California, M.S. (Safety)	1973

Professional Experience

Union Electric Company: Test Engineer - electrical controls, turbo generators, protective relays (to 1941).

Curtis-Wright Corporation: Materials and Process Engineer - chemical treatment of metals, design and application of welding equipment, metallurgical examination of materials, design and manufacture of aircraft (to 1945).

ACF Industries: General Manager - design and manufacture of railway equipment and aircraft air frame structures (to 1953).

Centrifugal and Mechanical Industries: Works Manager - design and manufacture of ore processing equipment (to 1955).

U.S. Industries: Chief Engineer - design, manufacture and test of electric motors and generators, fans and blowers, gear reducers, clutches and brakes (to 1957).

Giannini Controls Corporation: Chief Engineer - design, manufacture and test of pressure measuring instruments, AC and DC motors, potentiometers, temperature sensors, gyroscopes, accelerometers and associated equipment (to 1962).

Delta Semiconductor: Works Manager - design and manufacture of diodes and semiconductor devices.

Ralph M. Parsons Company - Engineering Manager - design and test of telemetry systems.

Electro-Optical Systems: Engineering Manager - design and test of semiconductor pressure and temperature instruments (to 1965).

Truesdail Laboratories, Inc.: Consulting Engineer - design and validation of experiments, experimental testing, accident reconstruction and analysis for causal effects (to 1976).

R. D. Jablonsky, Inc. - Consulting Engineer - industrial and safety systems.

Scientific Affiliations

Acoustical Society of America
American Institute of Industrial Engineers
American Society of Metals
American Society of Non-Destructive Testing
American Society of Safety Engineers
American Society of Testing Materials
Human Factors Society
Illuminating Engineering Society
Institute of Electrical and Electronic Engineers
National Fire Protection Association
Society of Automotive Engineers
Society of Photographic Scientists and Engineers
Sigma Xi

9/2/81 [illegible]

LIST OF DOCUMENTS/MATERIALS I HAVE REVIEWED-

1. Dimensions drawings of:
   a. Aft troop cmpt.
   b. Cargo cmpt.
   c. Cockpit & relief crew cmpt.

2. IDC Huie to Perry - Cabin P diffs during climb, 4-28-75
   Exhibit D-1

3. Task No. 5 - Attachm. F to Request 0001 of IDC Huie to Perry of 4-18-75 re: Cargo Cmpt Dec.

4. Trial testimonies, Schneider & Marchetti, of Maj. Dennis Traynor, Capt. Tilford Harp

5. Trial test. of J. Edwards in Schneider & Marchetti trials

6. Wreckage Diagram - Exh. D-9

7. Deposit. testimony of J. Edwards 4-28-80 & Deposit Exhibit 1-8 (2,3 & 8 are wreck. diagr. & computats, Edwards derivat. of G-forces formulae & Edwards computat.s G-loads on passengers.

8. USAF AAR Narrative Summary (Exhib D-3)

9. Aerospace Articles List

10. Busby Calculations based on Schneider Trial Testimony

11. Ltr from James Piper, Esq. 6-1-78 to Richard Jones, Esq. re: MADAR data from AF68-218 4-4-75 from point after R.D. to below 10M' (Exhib D-43)

12. Ltr of O. Lewis, Esq. of 6-1-78 to ~~Richard Jones, Esq.~~ Itzhak Brook MD. of 5-18-78, purportedly setting forth considerations re: hypoxia from the above accident (Exhib. D-42)

13. Ltr from I. Brook, MD. to O. Lewis, Esq. of 5-25-78 responding to above lttr (12).

14. Ltr of I. Brook, MD. to Marianne Schuelein, MD., 1-19-79, (DD-239)

15. Ltr of M. Schuelein, MD to I. Brook, MD. of 1-24-80 (D-250)

16. Ltr of Harry Gibbons, MD to C. Dubuc, Esq. 2-2-80

17. Transcript of trial testimony (Schneider) of following witnesses:
Ms. Lievermann (3-18-80, 303-367)
Lt. Aune (3-27-80, 1895-1952)
Lt. Tate (3-27-80, 1950-1990)
Dr. Stark (3-28-80, 2031-2122)
Lt. Neill (3-31-80, 2373-2403)
Dr. Gibbons (4-1-80, 2556-2674)
Mr. Parker (4-9-80, 3583-3595)
Prof. Harper (4-9-80, 3595-3621)
Wm. Timm (3-17-80, 158-175, 244-288, 388-479)

18. Affidavit of Patricia Quinn, M.D. 6-23-80

19. Ltr of 1975, FFAC to Adoptive Parents (DD-2)

20. Side-view diagram of the C5A (D-4)

21. Busby prelim. injunct. testimony of 6-25-80

22. Randel, Aerosp Med chapt. on Hypoxia

23. Bio A. Data Book 2nd Ed. Chapt. on Atmosphere

24. Physiolog. Trng, FAA, 1980

25. Physiolog. Trng. FAA (Undated)

26. J. Edwards calculation of vertical G-forces felt by C5A survivors

27. Deposition & trial testimony for:
Regina Aune
Tilford Harp
Christine Lievermann
Keith Malone
Harriet (Goffinet) Neill
Merritt Stark
Marcia (Wirtz) Tate
Dennis Traynor

27. Sworn statements of:
Regina Aune
Tilford Harp
Christine Lievermann
Keith Malone
Marcia (Wirtz) Tate

28. Collateral Report

29. Copies of data on file re: anoxia, hypoxia & near-drowning in infants & children

30. Flight descent profile as time taken from MADAR data.

31. Data duplicating resulting 9pm flight profile
(Exhib. D 1222 – 1226) the actual

32. Sworn statement of Harriet (Goffinet) Neill.

Reports of Edwards Dr. James Turnbow (D 1298). (D 1303) &

Drs. Jefferson Harry Gibbons (D-1304) Davis (D-16-1), Chas. Berry (D-1306)

Gaume Ex D 1270
9/25/81
JTM

RESUME

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EDUCATIONAL BACKGROUND

1975-present	Continuing Medical Education Requirements
1978-1980	Psychology Graduate Studies, University of Southern California
1960-1964	Aerospace Medicine Lectures, USAF School of Aviation Medicine, Brooks AFB
1950-1953	Graduate Studies, University of Kansas Medical Center
1943	USAF School of Aviation Medicine, Randolph Field, San Antonio, Texas
1943	US Army Medical Field Service School, Carlisle Barracks, Pennsylvania
1936-1940	M.D. Creighton University School of Medicine, Omaha, Nebraska
1932-1936	B.S., Chemistry, Kansas State University, Manhattan, Kansas

PROFESSIONAL HISTORY

McDONNELL DOUGLAS CORPORATION: 1964 to present

Present Position: Principal Staff Engineer for Aviation Medicine and Safety Research, Human Factors Engineering
Douglas Aircraft Company, Long Beach, California

Responsible for biomedical and Human Factors research and development and management. Also responsible for design problem analysis and solution in relation to Human Factors in support of various programs in the Division.

NORTHROP SPACE LABORATORIES: 1962-1964. Hawthorne, California

Director of Life Sciences. Research and development in biodynamics, bioastronautics, behavioral sciences and human engineering.

MARTIN-MARIETTA CORPORATION: 1958-1962. Denver, Colorado

Chief, Space Medicine and Biotechnology. Responsible for overall development and director of space biotechnology research program. Directed conceptual research on development of life support systems for manned space operations. Hydroponic food production.

USAF SCHOOL OF AEROSPACE MEDICINE: 1956-1958. San Antonio, Texas

Research Scientist in Department of Space Medicine. Worked directly with Dr. Hubertus Strughold, world-renowned pioneer in Space Medicine. Subject and monitor of experiments in zero-gravity and underwater weightlessness.

PRACTICE OF MEDICINE: 1946-1956 and 1941-1942. Ellinwood, Kansas

General practice of medicine and surgery. Staff physician, St. Rose Hospital, Great Bend, Kansas, and Ellinwood District Hospital. Industrial medicine for oil companies. Member, Barton County Medical Society, Kansas State Medical Society and American Medical Association.

MILITARY SERVICE: 1942-1946. United States Air Force Medical Corps

Flight Surgeon. Served in North Africa, Italy, Burma, China and India. Entered October 1, 1942 as First Lieutenant and separated May 10, 1946 as Major.

PRIVATE CONSULTING PRACTICE: 1972 to present.

Human Factors in Accident Reconstruction and Investigation in association with:

Roy D. Jablonsky, P.E., Inc.
1829 Pepper Drive
Altadena, California

Cases have included injuries/deaths due to: Gunshot wounds; roller coaster accidents; medical equipment-related accidents; automobile and motorcycle accidents; and recreational vehicle accidents.

PROFESSIONAL AFFILIATIONS

Aerospace Medical Association: Fellow, 1972
President, Space Medicine Branch, 1964-1965
American Institute of Aeronautics and Astronautics, Associate Fellow
American Academy of Family Physicians
California Academy of Family Physicians
Editorial Advisory Board, Journal of Combustion Toxicology
Who's Who in the West
Advisory Board Member, Donald Douglas Museum and Library, Santa Monica, CA
ASTM E5.21.03 Task Group on Toxicity Practice
FAA Special Aviation Fire and Explosion Reduction (SAFER) Committee: Member, Subgroup on Toxicity
Kansas State Board of Healing Arts, Certificate No. 8376

TEACHING APPOINTMENTS

University of Southern California, Graduate School, Department of Industrial and Systems Engineering, Adjunct Professor, 1967-present.
St. Rose Hospital, Ellinwood, Kansas, Instructor in Obstetrics, 1950-1956.
St. Francis Hospital, Wichita, Kansas, Instructor in Anatomy and Human Physiology, 1940-1941.
Kansas State University, Manhattan, Kansas, Instructor, Human Physiology, 1935-1936.

PUBLICATIONS

"Man's Flight Into Space: Making the World of Tomorrow," International Relations Institute Publication, St. Mary's University, San Antonio, Texas, Sept. 1957.

"Plants as a Means of Balancing a Closed Ecological System," Advances in Astronautical Sciences, Vol. 1, 107-116, Dec. 1957.

"Design of an Algal Chamber Adaptable to a Space Ship Cabin," Fourth Annual Meeting of American Astronautical Society, New York, Advances in Astronautics, Vol. 2, 222 Jan 1958.

"Nutrition in Space Operations," Annual Meeting of Institute of Food Technologists, Chicago, Illinois, Food Technology, XII, No. 9, 433-435, Sept. 1958.

"Life Support Systems for the Lunar Base," Semi-Annual Meeting of American Rocket Society, Preprint No. 1227-60, May 1960.

"Lunar Life Support Systems,"Missile Design and Development, Vol. 7, No. 1, 46, January 1961.

"Physiopathologic Implications of Chronic Weightlessness," Proceedings of First USAF Latin American Medical Conference, Albrook AFB, Panama Canal Zone, Office of Surgeon General, USAF, publishers, March 1962.

"Effects of Chronic Lunar Gravity on Human Physiology," Lunar Missions Meeting of American Rocket Society, Cleveland, Ohio, July 1962; Technology of Lunar Exploration-Progress in Astronautics and Aeronautics, Vol. 10, 381-411, Academic Press, 1962, with W. Kuehnegger.

"Space Cabin Life Support Systems Engineering and Development Tests in a Manned Space Laboratory Simulator," Volume II, Douglas Aircraft Company Report No. SM--49256, Aerospace Medicine/Man Systems Date, May 1966.

"Space Cabin Life Support Systems Engineering and Development Tests in a Manned Space Laboratory Simulator" Volume IV, Douglas Aircraft Company Report No. DAC-49256, Aerospace Medicine/Man Systems Date, with C. R. Adams, Nov. 1966.

"Evaluation of an Exercise System for Nul-Gravity Applications" Douglas Aircraft Company Report No. 60623, with D. L. Carpenter, Jan 1967.

"Some Results of Using Helium as a Space Cabin Atmosphere Diluent," Douglas Aircraft Company Report No. 4270, with C. R. Adams, Sept 1967.

"Crashworthiness Program - Detailed Analysis and Significance of the Gas Measurement," Data from the AIA Cleveland Fire Tests of 1967, Report No. SR-76, June 1968.

"Life Support Systems Technologies for Sealed Environments," Douglas Aircraft Company Report No. 5041, July 1968. Presented as Keynote Address at CYRO/68 Exposition, Chicago, Illinois; Applications of Cryogenic Technology, Chapter 5, R.W. Vance and A. Weinstock, Editors, Tinnen-Brown, 1969.

"Notes on the "E" Wave," Report No. SR-86, October 1968.

PUBLICATIONS (Continued)

"Analysis of Requirement for Individual Air Outlets on the DC-10," Report No. SR-90 November 1968.

"A Method of Estimating the Dynamic Envelope of the Seated, Lap-belt restrained Human Body Under Forward Linear Decelerations," Report No. SR-95, March 1969.

"Safe Animal Transportation Criteria in 'Class D' Baggage Compartments," Douglas Aircraft Company Report No. 67949, with P. Bartek, April 1969.

"Theoretical Determination of the Time of Useful Function (TUF) on Exposure to Combinations of Toxic Gases," Aerospace Medicine, 40:12, 1354-1357, Dec 1969.

"Experimental Technique for TUF Studies Under Mixed Contaminants," McDonnell Douglas Report No. MDC J0617, January 1970.

"Factors Influencing the Time of Safe Unconsciousness (TSU) for Commercial Jet Passengers Following Cabin Decompression":
Free Communications Volume of XVIII International Congress of Aerospace Medicine 445-456, September 1969;
Aerospace Medicine, 41:4, 382-385 April 1970;
Recent Advances in Aerospace Medicine, Proceeding of XVIII International Congress of Aviation and Space Medicine, D. E. Busby, Editor, Amsterdam, 193-203, D. Reidel Publishing Company, Dordrecht, Holland, 1969.

"Dynamics Analysis for Time of Useful Function (TUF) Predictions in Toxic Combustive Environments, Aerospace Medicine, 41:12, 1392-1395, December 1970.

"Experimental Results on Time of Useful Function (TUF) After Exposure to Mixtures of Serious Contaminants" Aerospace Medicine, 42:9, 987-990, September 1971.

"Physiological Conditioning for Minimizing Aircrew Fatigue" McDonnell Douglas Report No. MDC J1076, August 1972.

"Pilot Incapacitation: An Expression of Convergent Factors"(Co-author), Aerospace Medicine 43:9, 974-977, September 1972.

"Advances in Physiological Conditioning and Exercise Monitoring Procedures" (Co-author), McDonnell Douglas Report No. MDC J5789, April 1973.

"Analysis of the Effect of Increased Production Rate in DC-10 Paint Operations on Atmospheric Concentrations of Chemicals Used for Stripping" (Co-author), McDonnell Douglas Report No. MDC J5986, May 1973.

"Physiological Conditioning for Minimizing Aircrew Fatigue: Phase II-Application to DAC Flight Crews," McDonnell Douglas Report No. MDC J6664, Autust 1974.

"Mental Workload Assessment II. Physiological Correlates of Mental Workload: Report of Three Preliminary Laboratory Tests," McDonnell Douglas Report No. MDC J7023/01, with R. T. White, January 1976.

PUBLICATIONS (Continued)

"Mental Workload Assessment III. Laboratory Evaluation of One Subjective and Two Physiological Measures of Mental Workload," McDonnell Douglas Report No. MDC J7024/01, with R. T. White, January 1976.

"Animal Exposure During Burn Tests," Final Report, Contract No. NAS 2-8668, NASA Ames Research Center, January 1976.

"Bioassay Technologies," McDonnell Douglas Report No. MDC J7453, February 1977.

"Work/Rest Cycles for SIGINT and Other Long-Term Missions," McDonnell Douglas Report No. MDC J7904/01, May 1978.

"Biomedical Hazards Analysis for the KC 10," McDonnell Douglas Report No. MDC J8160, May 1978.

"Instrumented Animal Systems for Toxic Threat Assessment of Materials," Journal of Combustion Toxicology, May 1981. Presented at the California Conference on Fire Toxicity, August 1979.

"Further Development of an Instrumented Animal System for Toxic Threat Assessment of Materials," Proceedings of Fifth International Conference on Fire Safety, San Francisco, January 1980.

ADDITIONAL RECENT PAPERS

"Initial Tests of the ECG/R/T_i Animal Exposure System Using Carbon Monoxide," Journal of Combustion Toxicology, (In Press).

"Cardiac Responses of the Rat to Various Levels of CO," Presented at the Aerospace Medical Association Annual Scientific Meeting, Anaheim, California, May 1980.

"Comparison of the Cardiac Arrhythmia (C_a) and the Time to Incapacitation (T_i) Under Carbon Monoxide Exposures," Presented at the Aerospace Medical Association Annual Scientific Meeting, San Antonio, Texas, May 1981.

"Animal Responses in the BART Full Scale Vehicle Fire Tests," Presented at the Third Annual California Conference on Fire Toxicity, Stanford Research International, Menlo Park, California, July 1981.

"Physical Conditioning for the Prevention of Pilot Fatigue," Proceedings of the Human Factors Workshop of the Fifth International Conference of the System Safety Society, Denver, Colorado, July 1981.

PRESENTATIONS AT SCIENTIFIC MEETINGS (Not Published)

In addition to the presentations listed, I have been a guest speaker and lecturer at more than 254 meetings of various scientific, military and civic organizations and universities. These have included:

American Rocket Society
American Association for the Advancement of Science
American Astronautical Society
American Medical Association
American College of Physicians
Nebraska Academy of Science
Colorado University Medical Center
Medical Education for National Defense Program, Creighton University
Air Force Academy - First Lecturer in Space Medicine
Sheppard AFB Missile Training School

and many other local, regional and national meetings of medical educational and engineering societies. I have also participated in several television panel discussions and educational series.