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

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

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October 5, 1981

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

Re: FFAC v. Lockheed Aircraft Corporation

Dear Sirs:

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

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

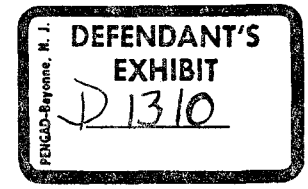
In order to address issue #1 certain assumptions must be made and the evidence presented to me makes these seem reasonable.

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

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

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

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



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

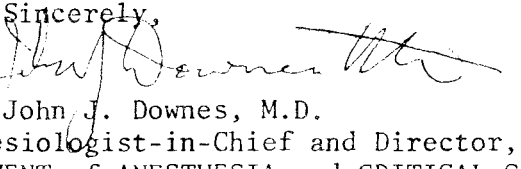
This degree of hypoxemia was endured for less than 1.75 minutes as the aircraft descended to 20,000 ft., and the P_AO_2 could be expected to rise to 38 mmHg; P_ACO_2 falls because of sustained hyperventilation to 25 mmHg with an increase in PaO_2 to approximately 30 mmHg (SaO_2 53%). At these two levels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

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

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

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

Sincerely,



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

Professor of Anesthesia and Pediatrics
University of Pennsylvania School of Medicine

JJD:cp

TABLE: AVERAGE ALVEOLAR GAS TENSIONS
IN ACUTE DECOMPRESSION AT ALTITUDE

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

Cabin altitude before decompression: 5,000 ft.

Calculations based on: 1) Alveolar air equation:

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

in which F_IO_2 is 0.2094, R is 0.79

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

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

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

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

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

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

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