Technical Report Documentation Page

1. Report No.	2. Government Accession No.	3. Recipient's Catalog No.
FAA-AM-77-8		

4. Title and Subtitle	5. Report Date
A STUDY OF EFFECTS OF HYPERTHERMIA ON LARGE, SHORT-HAIRED MALE DOGS: A SIMULATED AIR TRANSPORT ENVIRONMENTAL STRESS	March 1977
	6. Performing Organization Code
	8. Performing Organization Report No.

7. Author(s) G. D. Hanneman, D.V.M., E. A. Higgins, Ph.D., G. T. Price, D.V.M., G. E. Funkhouser, P. M. Grape, and L. Snyder

9. Performing Organization Name and Address

FAA Civil Aeromedical Institute
P.O. Box 25082
Oklahoma City, Oklahoma 73125

10. Work Unit No. (TRAIS)	11. Contract or Grant No.

12. Sponsoring Agency Name and Address	13. Type of Report and Period Covered
Office of Aviation Medicine Federal Aviation Administration 800 Independence Avenue, S.W. Washington, D.C. 20591	OAM Report
	14. Sponsoring Agency Code FAA

15. Supplementary Notes

This research was conducted under Task AM-A-74-VM-1 and AM-E-75-VM-1.

16. Abstract

When dogs are shipped by air transport, they can encounter environmental temperatures as high as 130.0° F during the summer months. Normally, little concern is given to the effects of hyperthermia on animals; however, heat-induced hyperthermia can be a major problem in dogs. To assess some aspects of the heat stress problem, 20 dogs were exposed to an ambient temperature of 130.0° F for 30 minutes--10 dogs at 15-percent relative humidity and 10 at 35-percent relative humidity. Transient and permanent changes were seen; however, no dogs died from exposure. All dogs exhibited increases in heart rate, rectal temperature, blood pH, hemoglobin, packed cell volume, and red blood cell count while body weight and blood carbon dioxide decreased. There were also differences between the two groups for blood pH, blood carbon dioxide, rectal temperature, and weight loss. The major histological tissue changes attributed to hyperthermia were fragmentation of the myocardium, acute cortical necrosis in the kidneys, and marked degenerative changes in the cerebellum and cerebral cortex that were considered severe and permanent.

17. Key Words		18. Distribution Statement
Hyperthermia Environmental Stress Dogs Transportation	Histopathology Blood Chemistry Humidity	Document is available to the public through the National Technical Information Service, Springfield, Virginia 22161.

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified		

Form DOT F 1700.7 (8-72) Reproduction of completed page authorized

A STUDY OF EFFECTS OF HYPERTHERMIA ON LARGE, SHORT-HAIRED MALE DOGS: A SIMULATED AIR TRANSPORT ENVIRONMENTAL STRESS

I. Introduction.

In the early 1970's, the general public let it be known that they were concerned about the safe, efficient, and humane treatment of animals during shipments by air freight. In 1973 the Special Studies Subcommittee of the Committee on Government Operations, House of Representatives, conducted hearings on problems in air shipment of domestic animals.[17] Testimony before this committee made quite evident that of the environmental stresses animals can be exposed to during shipment, heat is the most serious. It has been reported that in an air carrier on the ground in the summer, temperatures may reach 130.0° F or higher in the cargo compartment.[1 17 18]

When animals are exposed to ambient temperatures that exceed their normal body temperatures, they may experience hyperthermia.[13] The degree of hyperthermia in an animal depends on such factors as humidity, ventilation, and the animal's muscular exertion, hair coat, and physical condition. During an episode of hyperthermia, an animal may show little clinical evidence of harm; however, transient and permanent changes can occur. Because of public concern more attention has been given to the dog and its well-being than to other domestic small animals.

The dog, unlike man, produces little sweat but promotes heat loss through panting. By panting, the dog is able to evaporate moisture from its tongue and upper respiratory tract. However, as the relative humidity (RH) increases or when ventilation is inadequate, this process becomes less effective in dissipating heat. Prolonged exposure to these factors can tax a dog's ability to maintain a normal body temperature, and heat hyperpyrexia or heatstroke can result.[2]

In the past decade, little progress has been made in studying the biochemical and histopathological effects of hyperthermia on animals. It is a question, so far unanswered, of what degree and duration of hyperthermia will damage the central nervous system.[13] In 1927, Hall and Wakefield[4] studied hyperthermia in female dogs in an environment in which the dry-bulb temperature ranged between 131.0° and 141.0° F and the wet-bulb temperature ranged between 95.0° and 115.0° F. After 20–75 minutes of exposure, the dogs' rectal temperatures ranged between 106.0° and 113.4° F. They noted increases in blood urea nitrogen (BUN), sugar, chlorides, lactic acid, creatinine, and calcium (Ca) and decreases in plasma carbon dioxide (CO_2) combining power, inorganic phosphate, and serum pH. Histopathology showed cellular degeneration in the lung, kidney, liver, heart, and brain.

Jacobsen and Hosoi[8] used radiothermy on dogs. Eleven dogs received single exposures ranging from 37 minutes to 12 hours with resultant maximum rectal temperatures of 108.0° to 111.0° F. Of the 11 dogs exposed, 6 died within 40 minutes after heating. Histopathology showed changes in the heart, lung, spleen, gastrointestinal tract, kidney, and brain. Knudson and Schaible[12] studied canine physiological and biochemical changes resulting from exposure to an ultrahigh frequency field. Exposure times ranging from 30 minutes to 5 hours 15 minutes resulted in rectal temperatures ranging from 104.0° to 109.9° F. Weight loss ranged from 1.9 percent to 11.1 percent in dogs that were given no water during treatment. Most dogs were exposed more than once. The biochemical changes in the blood were similar to those reported by Hall and Wakefield. Hartman and Major[5] reported pathological changes in female dogs resulting from 5 to 7 hours of exposure at 150.0° to 157.0° F and humidities of 30 percent to 40 percent. The

TABLE 1. Blood Constituents in Dogs as Affected by Exposure to 130.0° F and a Relative Humidity of 15 Percent or 35 Percent*

Items		Duration of Exposure (Min) at 15% RH						Duration of Exposure (Min) at 35% RH					
		0		15		30		0		15		30	
Hematologic													
pH		7.38	(0.03)	7.45	(0.05)	7.50	(0.07)**	7.38	(0.03)	7.56	(0.07)	7.64	(0.07)**
Pv_{CO_2}	(mm Hg)	35.23	(2.91)	28.58	(2.96)	24.69	(3.32)**	41.38	(5.94)	24.11	(7.83)	17.08	(4.89)
Pv_{O_2}	(mm Hg)	45.34	(6.88)	53.64	(7.94)	50.96	(8.92)	44.01	(6.11)	51.01	(12.96)	48.53	(16.59)
Hb	(gm/100 ml)	13.05	(1.54)	13.63	(1.52)	14.18	(1.53)**	12.80	(0.63)	13.82	(1.22)	14.93	(1.69)
PCV	(percent)	43.40	(3.44)	45.50	(4.38)	46.30	(4.97)**	47.60	(3.06)	50.00	(3.77)	52.80	(5.83)**
RBC	(10^6/cmm)	4.82	(0.49)	5.10	(0.45)	5.30	(0.65)**	5.15	(0.47)	5.47	(0.51)	5.68	(0.65)**
WBC	(10^3/cmm)	8.33	(1716)	8.09	(1507)	7.83	(1630)	8.41	(1503)	8.88	(1592)	8.83	(1547)
Serum Chemistries													
Glucose	(mg/100 ml)	68.20	(6.97)	74.20	(12.03)	76.00	(16.22)	66.90	(17.32)	75.00	(18.41)	71.70	(14.73)
Calcium	(mg/100 ml)	10.40	(0.98)	10.26	(1.09)	10.29	(1.06)	8.76	(0.56)	9.24	(0.55)	9.11	(0.65)
Bilirubin, Total	(mg/100 ml)	0.46	(0.20)	0.51	(0.27)	0.59	(0.29)	0.76	(0.33)	1.07	(0.58)	0.98	(0.48)
Cholesterol, Total	(mg/100 ml)	166.50	(26.88)	165.00	(34.48)	175.00	(31.97)	157.30	(29.71)	168.60	(29.50)	163.90	(27.47)
Lipids	(mg/100 ml)	380.70	(18.71)	384.00	(21.96)	389.70	(22.03)	390.10	(43.94)	383.00	(51.66)	388.10	(51.36)
BUN	(mg/100 ml)	10.95	(3.05)	11.70	(2.76)	13.26	(4.04)	12.44	(4.56)	12.46	(4.10)	11.74	(4.45)
Inorganic Phosphorus	(mg/100 ml)	1.64	(0.88)	1.35	(1.13)	1.75	(1.11)	2.60	(0.64)	2.26	(0.63)	2.19	(0.54)
Protein, Total	(gm/100 ml)	6.84	(0.67)	6.95	(0.56)	7.05	(0.52)	6.09	(0.76)	6.11	(0.69)	6.30	(0.92)
Albumin	(gm/100 ml)	1.59	(0.74)	1.50	(0.53)	1.61	(0.34)	2.88	(0.55)	2.93	(0.60)	3.00	(0.73)
Globulin	(gm/100 ml)	5.26	(0.67)	5.35	(0.54)	5.32	(0.57)	3.21	(0.33)	3.18	(0.26)	3.27	(0.28)
SGOT	(Karmen Units)	25.30	(4.08)	26.10	(6.85)	25.50	(4.88)	28.75	(9.22)	30.89	(10.60)	34.10	(11.31)
Alkaline Phosphatase	(I.U.)	15.22	(6.48)	13.44	(6.51)	13.90	(7.13)	10.10	(5.78)	9.67	(6.96)	9.40	(6.64)

* 10 dogs each at 15-percent and 35-percent RH
** $p \leq 0.01$ statistical test by analysis of variance

Table entries are means; standard deviations are given in parentheses.
The 15-min entries at 35-percent RH are the means of nine dogs.
Alkaline phosphatase at 15-percent RH at 0 and 15 min are means of nine and eight dogs respectively.

TABLE 2. Heart Rates and Rectal Temperatures in Dogs as Affected by Exposure to 130.0° F and a Relative Humidity of 15 Percent or 35 Percent*

Items	Duration of Exposure (Min) at 15% RH							Duration of Exposure (Min) at 35% RH						
	0	5	10	15	20	25	30	0	5	10	15	20	25	30
Heart Rate (beats/min)	85 (27)	95 (32)	97 (26)	102 (33)	107 (28)	113 (33)	111** (25)	111 (24)	134 (19)	135 (22)	135 (21)	143 (28)	147 (23)	149** (22)
Rectal Temperature (° F)	101.5 (0.28)	101.6 (0.26)	101.8 (0.21)	102.2 (0.17)	102.7 (0.15)	103.2 (0.25)	103.9** (0.49)	101.7 (0.35)	102.0 (0.44)	102.4 (0.51)	103.0 (0.50)	103.7 (0.53)	104.5 (0.58)	105.3** (0.67)

* 10 dogs each at 15-percent and 35-percent RH

** $p \leq 0.01$

Table entries are means; standard deviations are given in parentheses.

were suggestive of early cortical necrosis. In the tissues showing tubular necrosis, the changes were present as early as 28 h and as late as 100 h postexposure. In one dog necropsied at 28 h, the changes were so profound that, in our experience, if the dog had been allowed to live it would probably have experienced renal failure.

The lung from all the exposed dogs showed some degree of pathology. Those necropsied at 4, 28, and 52 h revealed more lung changes than those sacrificed at 100 and 196 h. The predominant histopathological findings were thickening and congestion of the alveoli septi, hemorrhage and/or edema in the alveolar spaces, and engorgement of the blood vessels. Occasionally, a specimen had hemorrhage or debris in a few of the bronchi.

IV. Discussion.

The results of this study showed significant ($p \leq 0.01$) changes in heart rate, rectal temperature, Pv_{CO_2}, blood pH, Hb, RBC, PCV*, and weight that can be related to the exposure environment. The effects of humidity produced significant differences between the 15-percent-RH and 35-percent-RH groups for Pv_{CO_2}, blood pH, rectal temperature, and weight loss. Peak heart rate increase occurred in 5 of the 10 dogs of the 15-percent-RH group before the end of the 30-min exposure whereas 7 of the 10 in the 35-percent-RH group peaked before the end of exposure. Although no significant change was found in BUN, 2 of 10 dogs showed a decrease in BUN at the 15-percent-RH exposure whereas 7 of 10 at the 35-percent-RH exposure showed a decrease. Some of the remaining parameters showed changes that were not significant.

Hemolysis was also noted in samples from a few of the dogs exposed to the 35-percent humidity, but the quantity was not measurable. Iampietro *et al.*[7] showed that hemolysis occurred in dogs exposed to temperatures ranging from 70.0° to 120.0° F at 50-percent RH for up to 60 min. He states that hemolysis might have been the result of extremely low blood CO_2 and/or high pH brought about by hyperventilation and/or high body temperature.

Throughout the available literature, we found few reports that histology had been performed on surviving victims of hyperthermia. Hartman and Major[5] reported histological changes in anesthetized dogs exposed to temperatures of from 150.0° to 175.0° at 30- to 40-percent RH. Histological changes in animal tissues have been reported by Jacobsen and Hosoi[8] and by Mortimer[15] in studies of experimental hyperthermia induced by ultrahigh frequency oscillators or high frequency current. Hall and Wakefield[4] showed that degenerative changes occurred in the brain, heart, kidney, and lung of dogs that died from experimental heatstroke. Malamud *et al.*[14] studied 125 fatal cases of heatstroke in military personnel who had undergone strenuous muscular exercise at high temperatures. According to their description, the histopathological changes of the brain and viscera in these cases closely parallel the changes found in our dogs. Freeman and Dumoff,[3] in their report on cerebellar syndrome following heatstroke in man, state that periods of exposure must be brief because at high temperatures the pathological alterations in the body cells rapidly become irreversible.

In our study, the histopathological changes in the brain, heart, kidney, and lung examined provided the most interesting and valuable information. It is our opinion that most of the changes in the cerebellum and cerebral cortex are considered severe and permanent and that the neuronal changes developed during the period of hyperthermia. We believe the fragmentation of the myocardium, loss of myofibrillar striations, and pyknotic nuclei are attributable to hyperthermia. In those dogs whose kidneys showed acute cortical necrosis, this was, we believe, related to hyperthermia in that none of the dogs experienced any clinical signs of shock. In general, the changes observed in the lungs were minimal to moderate with those necropsied at 100 and 196 hours showing signs of recovery. We were unable to detect any qualitative or quantitative relationship in tissue changes between the two humidity groups. Although all dogs survived acute hyperthermia with no signs of illness, there were histological changes in the tissues examined.

V. Conclusions.

In view of the results obtained from this limited study, we should remember that 130.0° F is on the high side of the ambient temperature scale to which dogs could be exposed during air transport. With a dog's average rectal temperature being 101.5° F, the question remains: At what ambient temperature will a dog sustain permanent damage from hyperthermia? We should not assume that all dogs will respond to a given temperature in the same manner. Aside from health problems that can influence a dog's ability to withstand various temperatures, there are at least three anatomical characteristics that should be considered. First, there is a wide range of size in breeds weighing from 3 to 175 pounds and, therefore, considerable variation in the area of body surface exposed. Second, the hair coats of various breeds differ in density, length, and texture. Third, the brachycephalic breeds of dogs have short, narrow nasal passages, pendulous soft palates, and heavily muscled throats—characteristics that increase the effort of accelerated breathing during panting. These factors should be studied further before the FAA can recommend thermal limits for the air transportation of dogs.

References

1. American Airlines: Freight Service Manual, Freight Edition, 4, August 31, 1973.
2. Armistead, W. W.: Heat Stroke and Heat Prostration. In *Canine Medicine*. Haskins, H. P., J. V. Lacroix, and K. Mayer (Ed.), 2d ed., American Veterinary Publications, Inc., Santa Barbara, Calif., 708–712, 1971.
3. Freeman, W., and E. Dumoff: Cerebellar Syndrome Following Heat Stroke, ARCH. NEUROL. PSYCHIATRY, 51:67–72, 1971.
4. Hall, W. W., and E. G. Wakefield: A Study of Experimental Heat-Stroke, JAMA, 89:177–182, 1927.
5. Hartman, F. W., and R. C. Major: Pathological Change Resulting From Accurately Controlled Artificial Fever, AM. J. CLIN. PATHOL., 5:392–410, 1935.
6. Higgins, E. A.: Some Physiological Responses of Dogs to Heat and Humidity, Ph.D. Thesis, University of Oklahoma, Norman, Okla., 1966.
7. Iampietro, P. F., M. J. Burr, V. Fiorica, J. M. McKenzie, and E. A. Higgins: pH-Dependent Lysis of Canine Erythrocytes, J. APPL. PHYSIOL., 23:505–510, 1967.
8. Jacobsen, V. C., and K. Hosoi: The Morphologic Changes in Animal Tissues Due to Heating by an Ultrahigh Frequency Oscillator, ARCH. PATHOL., 11:744–759, 1931.
9. Kanter, G. S.: Effect of Heat on Regulation of Body Fluids and Electrolytes in Dogs, AM. J. PHYSIOL., 178:259–262, 1954.
10. Kanter, G. S.: Cause of Hypoglycemia in Dogs Exposed to Heat, AM. J. PHYSIOL., 196:619–624, 1959.
11. Kanter, G. S., and R. H. Lubinski: Effect of Heat on Inorganic Phosphate Regulation in Dogs, AM. J. PHYSIOL., 180:559–562, 1955.
12. Knudson, A., and P. J. Schaible: Physiologic and Biochemical Changes Resulting From Exposure to an Ultrahigh Frequency Field, ARCH. PATHOL., 11:728–743, 1931.
13. Leithead, C. S., and A. R. Lind: *Heat Stress and Heat Disorders*, F. A. Davis Company, Philadelphia, Pa., 1964.
14. Malamud, N., W. Haymaker, and R. P. Custer: Heat Stroke. THE MILITARY SURGEON, 431, November 1946.
15. Mortimer, B.: Experimental Hyperthermia Induced by the High Frequency Current, RADIOLOGY, 16:705–718, 1931.
16. Nemoto, E. M., and H. M. Frankel: Cerebral Oxygenation and Metabolism During Progressive Hyperthermia, AM. J. PHYSIOL., 219:1784–1788, 1970.
17. Special Studies Subcommittee, Committee on Government Operations, House of Representatives: Problems in Air Shipment of Domestic Animals, US Government Printing Office, Washington, D.C., Hearings held September 26–28, 1973.
18. Zachary, M.: Air Shipment of Pets, VET. MED. SMALL ANIM. CLIN., 925–928, August 1975.