

# DILATED CARDIOMYOPATHY ASSOCIATED WITH HYPOVITAMINOSIS E IN A CAPTIVE COLLECTION OF FLYING FOXES (*PTEROPUS* SPP.)

Darryl J. Heard, B.V.M.S., Ph.D., Claus D. Buergelt, D.V.M., Ph.D.,  
Patti S. Snyder, D.V.M., M.S., Andra K. Voges, D.V.M., and Ellen S. Dierenfeld, Ph.D.

**Abstract:** Seven captive adult flying foxes (*Pteropus* spp.) from the same facility developed dilated cardiomyopathy during a 2-yr period. The physical, clinicopathologic, radiographic, and ultrasonographic findings showed congestive heart failure secondary to dilated cardiomyopathy. Pathologic findings and micronutrient evaluation of the diet, blood, and tissues were indicative of hypovitaminosis E.

**Key words:** Bat, flying fox, *Pteropus*, dilated cardiomyopathy, hypovitaminosis E, nutrition.

## INTRODUCTION

Idiopathic cardiomyopathy leading to congestive heart failure was described in a 16-yr-old Indian flying fox (*Pteropus giganteus*).<sup>13</sup> Primary cardiomyopathy in association with hypovitaminosis E has been reported in several captive animals, including artiodactylids,<sup>7-9</sup> primates,<sup>8,9</sup> and birds.<sup>9</sup> The following is a description of several cases of dilated cardiomyopathy associated with possible hypovitaminosis E in a captive collection of flying foxes.

## MATERIALS AND METHODS

Between June 1992 and July 1994, six island flying foxes (*Pteropus hypomelanus*) and one Rodriguez Island flying fox (*P. rodricensis*) were diagnosed with dilated cardiomyopathy (Table 1). All bats were housed in indoor/outdoor flight enclosures at the Lube Foundation, a private breeding and research facility in north-central Florida. All but one of the affected bats (no. 7) were male. In March 1993, bats 2, 3, and 4 had undergone abdominal surgery for implantation of telethermometers, which were removed 1-2 mo later.

All affected island flying foxes, along

with eight other adult males and 30 females, had been captured in Indonesia and imported in June 1990. After arrival, they were randomly subdivided into several separately housed breeding groups. The Rodriguez Island flying fox was captive bred and imported in September 1990 from the Jersey Zoo with four other adult males and five females. The Rodriguez Island flying foxes were maintained as a single breeding group. Other flying fox species housed at Lube in 1993 included golden-mantled (*P. pumilus* 8.9.1), giant (*P. vampyrus* 5.15), dog-faced (*Cynopterus brachyotis* 2.3.1), and grey-headed (*P. poliocephalus* 2.1) flying foxes.

All bats were fed a mixture of fruits and vegetables supplemented with commercial primate diets (Zu/Preem Canned Marmoset and Hill's New World Monkey Chow, Hill's Pet Products, Topeka, Kansas 66601, USA) and a vitamin supplement (Vionate Vitamin-Mineral Powder, ARC Laboratories, Atlanta, Georgia 30340, USA). The fruits and vegetables were processed into 2- × 2-cm chunks with a commercial food slicer, and all ingredients were mixed and distributed in 170-ml cups placed in multiple positions throughout each pen. Each bat was offered about half its bodyweight in food daily. The diet initially contained a calculated 56 IU/kg (dry basis) of vitamin E. At the beginning of 1994, the amounts of both vitamin supplement and primate diets were

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From the Departments of Small Animal Clinical Sciences (Heard, Snyder, Voges) and Pathobiology (Buergelt), College of Veterinary Medicine, University of Florida, Gainesville, Florida 32610, USA; and the Department of Nutrition, Wildlife Conservation Society, Bronx, New York 10460-1099, USA (Dierenfeld).

**Table 1.** Species, identification, sex, dates of diagnosis and death, and clinical signs for seven captive flying foxes that spontaneously developed dilated cardiomyopathy.

Species	Animal no.	Sex	Diagnosis <sup>a</sup>	Clinical signs	Death <sup>b</sup>
<i>Pteropus rodricensis</i>	1	M	ND	ND	15 Jun 92
<i>Pteropus hypomelanus</i>	2	M	9 Apr 93	hepatomegaly, hypothermia, lethargy, tachyarrhythmia, venocollapse	18 Aug 93
	3	M	ND	ND	11 Sep 93
	4	M	27 Aug 93	hepatomegaly, lethargy, pale mucous membranes, venocollapse	13 Sep 93 (E)
	5	M	29 Sep 93	cachexia, cranial edema, lethargy, pale mucous membranes, tachyarrhythmia, venocollapse	1 Nov 93 (E)
	6	M	23 Sep 93	anorexia, cranial edema, hepatomegaly, hypothermia, lethargy tachyarrhythmia, venocollapse	24 Sep 93 (E)
	7	F	11 Jul 94	abortion, cranial edema, heart murmur, hepatomegaly, lethargy	14 Jul 94 (E)

<sup>a</sup> Based on thoracic radiographic and ultrasonographic findings. ND = not determined.

<sup>b</sup> E = euthanasia.

increased to give a calculated vitamin E content of 641 IU/kg (dry basis). Drinking water was available ad lib.

The bats were anesthetized and maintained with isoflurane (Aerrane, Anaquest, Madison, Wisconsin 53713-2318, USA) in oxygen for all diagnostic procedures. Blood was collected from five bats (nos. 2, 4, 5, 6, 7) either at initial diagnosis of dilated cardiomyopathy or just prior to euthanasia and was submitted for hematologic and plasma biochemical analysis (Table 2). Thoracic and abdominal radiography and

ultrasonography were performed in five affected bats (nos. 2, 4, 5, 6, 7) and eight healthy male adult wild-caught island flying foxes. Plasma samples from three affected and 11 unaffected island flying foxes were submitted for detection of antibodies against encephalomyocarditis (Dr. Jack Gaskin, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, Florida 32610, USA). Plasma samples were collected from bats 4, 5, and 6 immediately prior to euthanasia and submitted for taurine (Veterinary Med-

**Table 2.** Selected hematologic and plasma biochemical values from blood collected at the time of either diagnosis or euthanasia for five captive, wild-caught adult island flying foxes that spontaneously developed dilated cardiomyopathy.

Variable	Animal no. (blood collection date)					Reference ranges ( $\bar{x} \pm SD$ ) ( $n = 75$ )
	(6/16/93)	(8/27/93)	(9/29/93)	(9/24/93)	(7/12/94)	
WBC ( $\mu\text{l}^{-1}$ )	ND <sup>a</sup>	ND	5,200	2,900	2,000	13,340 $\pm$ 5.48
RBC ( $\mu\text{l}^{-1}$ )	ND	ND	7.3	5.3	7.11	9.03 $\pm$ 1.36
Hemoglobin (g/dl)	ND	ND	11.8	7.5	10.6	15.4 $\pm$ 2.5
Packed cell volume (%)	ND	ND	34.5	22.8	31.8	47.1 $\pm$ 8.5
Glucose (mg/dl)	121	93	203	59	94	152 $\pm$ 35
Potassium (mEq/L)	4.4	4.5	4.6	6.0	5.8	3.4 $\pm$ 0.4
Blood urea nitrogen (mg/dl)	9.4	10	43	50.7	56	4.4 $\pm$ 3.6
Creatinine (mg/dl)	ND	0.4	0.5	0.6	0.5	0.6 $\pm$ 0.1
Calcium (mg/dl)	7.7	7.8	7.8	7.8	7.7	8.5 $\pm$ 0.6
Phosphorus (mg/dl)	ND	5.6	5.3	7.8	6.9	5.5 $\pm$ 2.4
Protein (g/dl)	ND	5.3	4.7	4.5	4.3	7.2 $\pm$ 0.6
Albumin (g/dl)	ND	2.7	2.5	2.4	2.3	4.2 $\pm$ 0.3
Aspartate transaminase (U/L)	102	154	170	520.5	376	41 $\pm$ 22
Alanine transaminase (U/L)	48	58	127	113.1	86	10 $\pm$ 7

<sup>a</sup> ND = not determined.

icine Molecular Biosciences, University of California, Davis, California 95616, USA), L-carnitine (Metabolic Analysis Labs, Madison, Wisconsin 53713, USA), selenium, copper, cobalt, magnesium, and zinc (Animal Health Diagnostic Laboratory, Lansing, Michigan 48909, USA), and vitamin E concentration determinations (Table 3). Vitamin E levels were determined by high-power liquid chromatography (HPLC) with fluorescence detection using parameters previously described.<sup>15</sup> Plasma samples from unaffected male ( $n = 6$ ) and female ( $n = 6$ ) wild-caught island flying foxes were submitted for comparison.

Bats 4 and 5 were treated with the angiotensin-converting enzyme inhibitor, enalapril (Vasotec, Merck & Co., West Point, Pennsylvania 19486, USA; 0.5 mg p.o. every 72 hr). Treatment of bat 7 consisted of furosemide (Geneva Pharmaceuticals, Broomfield, Colorado 80020, USA; 1.5 mg/kg p.o. b.i.d.) with the addition of digoxin (Roxane Laboratories, Columbus, Ohio 43216, USA; 0.005 mg p.o. b.i.d.) after 2 days.

All seven bats were necropsied, and representative tissues were examined histopathologically. Tissue samples from bats 4,

5, and 7 were collected at necropsy and frozen at  $-70^{\circ}\text{C}$  for later analysis of vitamin E ( $\alpha$ - and  $\gamma$ -tocopherol) concentrations using HPLC with fluorescence detection.<sup>4</sup> These tissue samples were compared with tissues from three bats that died acutely from traumatic injuries in 1994 (Table 4). Myocardial samples from bat 6 were placed in Trump's solution and submitted for transmission electron microscopic examination.

## RESULTS

### Clinical signs

Two flying foxes (nos. 1, 3) were found dead. During routine physical examination, two bats (nos. 2, 4) appeared to have cyanotic mucous membranes and poor peripheral perfusion, making venipuncture difficult. Bat 5 was identified as ill during thoracic radiography and ultrasonography of the remaining imported island flying fox males. Bats 6 and 7 were examined because of lethargy and cranial swelling.

The clinical signs associated with dilated cardiomyopathy in these bats were consistent with decreased myocardial function and development of congestive heart failure

**Table 3.** Plasma levels of micronutrients from three adult wild-caught island flying foxes that spontaneously developed dilated cardiomyopathy and from 12 bats with no evidence of cardiomyopathy.

Animal no.	Sex	Date	Vitamin E (µg/ml)	Cholesterol (mg/dl)	Selenium (ng/ml)	Copper (ppm)	Cobalt (ppm)	Magnesium (ppm)	Zinc (ppm)	Taurine (nmoles/ml)	L-carnitine (µmoles/sL)			
											Total	Free	Esters	
<b>Affected</b>														
4	M	9/13/93	0.00	9	75	1.55	<0.125	22.0	1.20	ND <sup>a</sup>	26.0	22.4	3.6	0.16
5	M	9/25/93	0.00	4	71	0.846	<0.111	32.3	4.34	45	61.6	53.4	8.2	0.15
6	M	9/25/93	0.00	7	83	1.10	<0.125	30.0	1.65	118	67.5	57.2	10.3	0.18
<b>Unaffected</b>														
631	M	10/22/93	0.44	6	63	1.25	<0.125	21.1	4.12	14	25.5	20.8	4.7	0.23
664	M	10/22/93	0.03	1	83	0.865	<0.111	22.9	3.73	41	19.2	17.4	1.8	0.10
627	M	10/22/93	0.00	2	60	1.46	<0.111	29.4	5.01	ND	ND	ND	ND	ND
674	M	10/22/93	0.00	11	90	1.17	<0.111	21.2	3.10	ND	ND	ND	ND	ND
635	M	10/22/93	0.00	6	87	1.60	<0.125	27.1	2.24	37	25.5	23.0	2.5	0.11
630	M	10/22/93	0.00	8	70	0.824	<0.111	23.2	3.87	55	24.6	21.6	3.0	0.14
639	F	10/22/93	0.19	18	80	1.21	<0.111	22.3	2.35	ND	28.3	24.0	4.3	0.18
718	F	10/22/93	0.00	12	68	1.2	<0.111	19.8	4.5	33	12.3	11.2	1.1	0.10
688	F	10/22/93	0.00	32	83	1.59	<0.111	23.6	1.86	47	25.1	21.6	3.5	0.16
690	F	10/22/93	0.57	ND	111	1.42	<0.125	18.9	1.8	ND	ND	ND	ND	ND
633	F	10/22/93	0.14	43	80	1.41	<0.111	20.9	1.48	48	27.4	23.0	4.4	0.19
666	F	10/22/93	0.03	21	100	1.36	<0.111	19.4	2.29	48	23.7	20.4	3.3	0.16

<sup>a</sup> ND = not determined.

**Table 4.** Plasma vitamin E and tissue vitamin E ( $\alpha/\gamma$  tocopherol) levels in island flying foxes (*Pteropus hypomelanus*) with and without dilated cardiomyopathy. The tissue vitamin E levels for animal no. 7 may be inaccurate because of dehydration prior to analysis.

Animal	Plasma vitamin E ( $\mu\text{g/ml}$ )	$\alpha/\gamma$ tocopherol ( $\mu\text{g/g}$ wet)		
		Heart	Liver	Kidney
4	0.00	0.60/0.00	0.51/0.00	0.41/0.00
5	0.00	ND <sup>a</sup>	0.74/0.00	0.54/0.00
6	0.00	ND	ND	ND
7	0.06	4.04/ND	2.19/ND	1.26/ND
623	ND	ND	7.71/0.22	4.81/0.45
661	ND	ND	12.5 /0.53	ND
699	ND	ND	11.32/0.61	10.76/1.12

<sup>a</sup> ND = not determined.

(Table 1). These signs included lethargy, reluctance to fly, exercise intolerance, hypothermia, tachyarrhythmias, and, in the later stages, anorexia/cachexia, hepatomegaly, and cranial edema (Fig. 1). Additionally, bat 7 had an auscultatable murmur and had aborted a late-term fetus 1 mo prior to presentation.

### Hematologic and plasma biochemical values

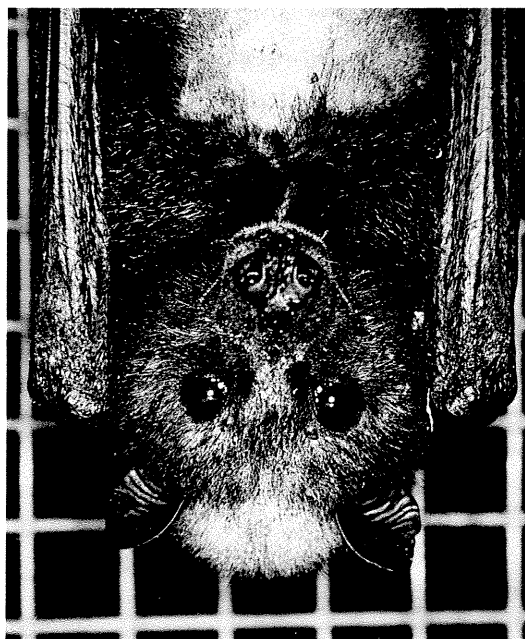
Significant findings included leukopenia, anemia, hyperkalemia, uremia, hypoproteinemia, hypoalbuminemia, and elevated aspartate transaminase (AST) and alanine transaminase (ALT) (Table 2).

### Radiography and ultrasonography

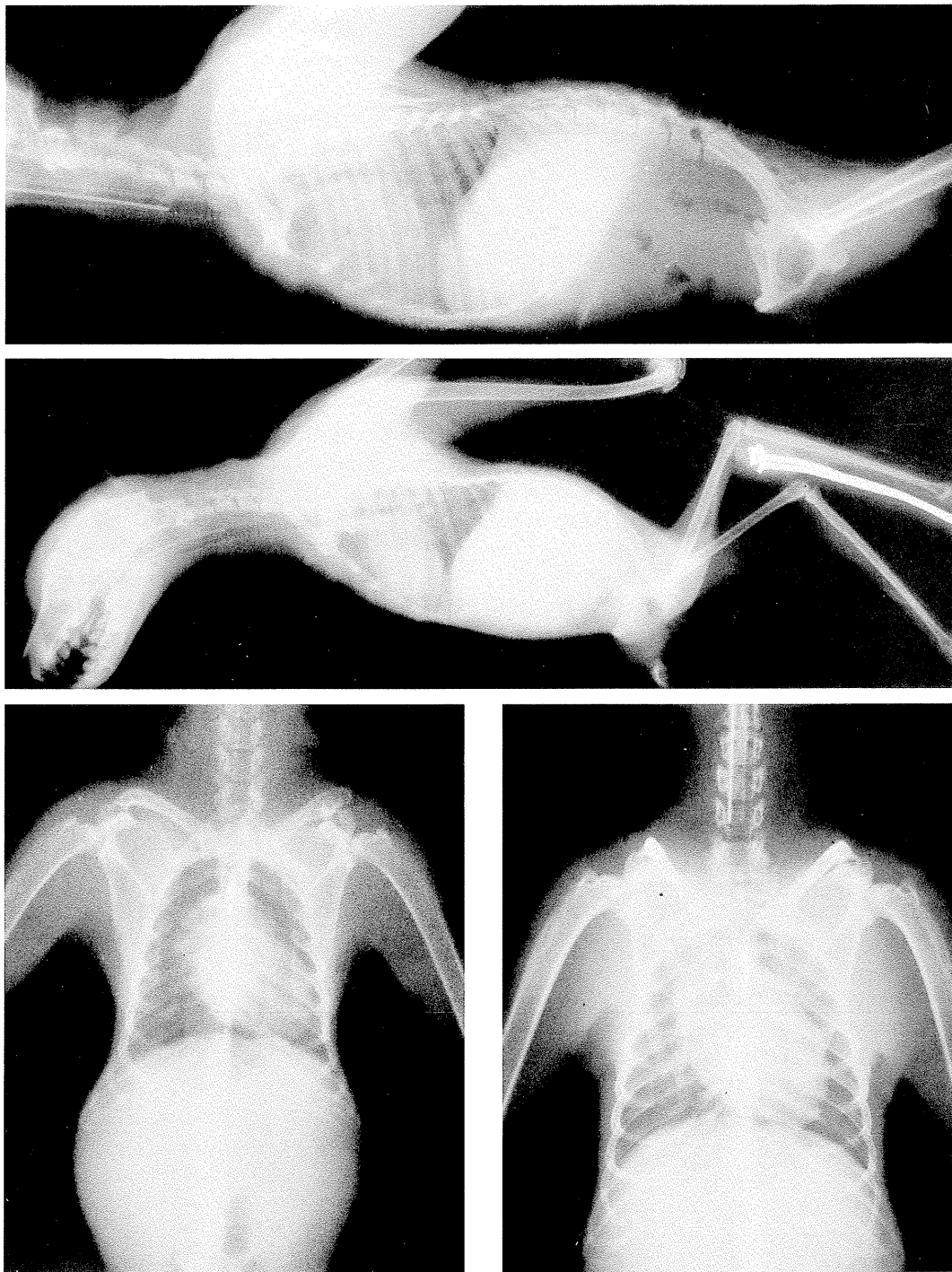
Thoracic radiographs showed generalized cardiomegaly and interstitial densities consistent with pulmonary edema (Fig. 2a, b). Two-dimensional echocardiography revealed ventricular and atrial dilatation, myocardial wall thinning, and poor contractility. Additionally, bat 7 had severe mitral valve insufficiency, as demonstrated by Doppler echocardiography.

### Gross and histopathologic findings

Gross necropsy confirmed cardiomegaly, cardiac dilatation, and hepatomegaly in the five bats diagnosed antemortem. Although both atria and ventricles appeared affected, dilatation was most common and severe in



**Figure 1.** Adult male wild-caught island flying fox (*Pteropus hypomelanus*) with cranial edema (left) due to congestive heart failure secondary to a dilated cardiomyopathy. A male without congestive heart failure (right) is shown for comparison.



**Figure 2.** Adult male wild-caught island flying fox (*Pteropus hypomelanus*). **a.** Lateral radiograph of male with congestive heart failure secondary to a dilated cardiomyopathy (top). Note the enlarged cardiac silhouette and pulmonary densities. A radiograph of a normal male (bottom) is shown for comparison. **b.** Ventrodorsal radiograph of male with congestive heart failure secondary to a dilated cardiomyopathy (right). A radiograph of a normal male (left) is shown for comparison. The radiodense objects overlying the spine are microchips implanted between the scapulae.

the right heart and was associated with apparent ventricular wall thinning (right ventricle wall thickness = 1 mm, left ventricle wall thickness  $\leq$  3 mm). In bats 1 and 7, marked pericardial effusion was also noted. In bat 6, an irregular 0.5-cm-diameter tan/purple mass was lightly adhered to the intima of the aortic outflow tract.

Histologic examination of the seven hearts showed moderate to severe acute myocyte degeneration and chronic, multifocal myocardial fibrosis. Additionally, bat 3 had a mild, multifocal lymphocytic myocarditis. The mass in the aortic outflow tract of bat 6 was identified as a thrombus, and left atrial thrombosis was also identified in bat 1. In bats 5 and 7, there was minimal multifocal acute myofiber degeneration of the tongue. In the pectoral muscles of bats 4, 5, and 7, there was also acute to chronic, mild multifocal myodegeneration. Hepatomegaly was caused by marked congestion. Despite the use of special stains, neither bacteria nor fungi were identified in any of the histologic sections. Protozoa and parasites were not observed.

#### **Electron microscopic findings**

Ultrastructural studies of the heart from bat 6 revealed myocyte degeneration that included mitochondrial swelling, interruption of contractile bands, myocytolysis with myofilaments scattered throughout the sarcoplasm, and replacement of myocytes by collagen-producing fibroblasts. These alterations were considered compatible with acute primary myocyte degeneration and chronic postnecrotic fibrosis. No viruses or other infectious agents were identified.

#### **Viral antibody status**

All samples were negative for antibodies to encephalomyocarditis virus.

#### **Micronutrient and toxicologic analysis**

Significant findings in 1993 included unmeasurable plasma levels of vitamin E for three affected bats and low to unmeasurable levels in many bats without dilated car-

diomyopathy ( $0.10 \pm 0.18 \mu\text{g/ml}$ ; range, 0.00–0.57;  $n = 12$ ) (Table 3). Random plasma samples from island flying foxes in April ( $2.02 \pm 2.00 \mu\text{g/ml}$ ; range, 0.20–6.12;  $n = 8$ ) and August 1994 ( $0.31 \pm 0.50 \mu\text{g/ml}$ ; range, 0.01–2.11;  $n = 18$ ), following diet improvements, were greater than the 1993 values. Random plasma vitamin E concentrations were also determined for golden-mantled ( $1.20 \pm 1.10 \mu\text{g/ml}$ ; range, 0.19–4.27,  $n = 18$ ), giant ( $0.87 \pm 1.13 \mu\text{g/ml}$ ; range, 0.05–4.92,  $n = 20$ ), and Rodriguez Island ( $0.81 \pm 0.85 \mu\text{g/ml}$ ; range, 0.00–3.00;  $n = 12$ ) flying foxes in late 1993 and early 1994. Tissue levels of  $\alpha$ -tocopherol were much lower in the affected than in the unaffected bats (Table 4). Additionally,  $\gamma$ -tocopherol levels were undetectable in affected bats. Several bats with and without cardiomyopathy had moderately elevated zinc levels (Table 3).

Plasma taurine levels in all but one bat were well above the 20 nmol/ml concentration associated with feline cardiomyopathy (Table 3).<sup>14</sup> Plasma total L-carnitine concentrations in all bats were lower than those considered normal in humans ( $90 \pm 6 \mu\text{moles/L}$ )<sup>1</sup> but were either within or higher than normal ranges for dogs (12–40  $\mu\text{moles/L}$ )<sup>6</sup> and cats.<sup>5</sup> The highest plasma L-carnitine concentrations (62 and 68  $\mu\text{moles/L}$ ) occurred in two bats (nos. 5, 6) with cardiomyopathy.

### **DISCUSSION**

All the flying foxes described here had myocardial degeneration and fibrosis associated with dilatation and eventual congestive heart failure. Dilated cardiomyopathy may result from genetic, infectious, toxic, traumatic, endocrine, nutritional, and idiopathic conditions.<sup>16,17</sup> Because fibrosis is a common end point for many myocardial diseases, more than one etiology was perhaps responsible for the disease in these bats.<sup>17</sup> However, the clustering of cases at the same facility over a 2-yr period is suggestive of a single cause.

A genetic etiology is unlikely because all

affected animals were adults and had been obtained from several different geographical regions in Indonesia. Infectious causes of myocarditis in animals include viruses (e.g., coxsackie, encephalomyocarditis, canine parvovirus), bacterial (e.g., *Bacillus piliformis*), protozoal (e.g., toxoplasmosis, trypanosomiasis), and fungal (e.g., aspergillosis).<sup>16</sup> Although there was no evidence of an infectious agent in these bats, myocyte death may have been caused by an agent that had been eliminated by the time dilated cardiomyopathy was diagnosed. Similarly, antibodies to viruses perhaps were undetectable or had waned by the time of sampling.

A large group of toxic agents (e.g., cobalt, catecholamines, antihypertensive and antineoplastic agents, ethanol) are known to produce myocardial injury.<sup>16,17</sup> However, the bats described here had no known exposure to such toxic agents. Although in several animals, zinc levels were moderately elevated relative to values in other mammals,<sup>12</sup> there was no apparent relationship to affected bats. Furthermore, zinc toxicity has not previously been associated with cardiomyopathy in either animals or humans.<sup>16,17</sup> However, zinc is known to interfere with the gastrointestinal uptake of selenium and may thus exacerbate deficiency.<sup>16</sup> The elevated zinc levels may have been caused by ingestion of rainwater in contact with the corroded galvanized-iron wire on the bats' enclosures.

Nutritional deficiencies known to be associated with cardiomyopathy include those of selenium, vitamin E, potassium, copper, iron, thiamine, magnesium, protein, tryptophan, choline, taurine, and L-carnitine.<sup>16,17</sup> The plasma selenium, potassium, copper, iron, magnesium, protein, taurine, and L-carnitine concentrations in these bats were within normal mammalian ranges.<sup>5,6,12</sup> Plasma thiamine, tryptophan, and choline levels were not evaluated.

The observed myodegenerative changes and low plasma and tissue vitamin E levels in the cardiomyopathic bats are consistent with hypovitaminosis E.<sup>2,3,16</sup> Confirmation of

a direct relationship would require controlled nutritional studies using diets of known vitamin E content. This clinical study is further limited by point sampling of vitamin E levels. Vitamin E levels normally may fluctuate in flying foxes. Further information is required on vitamin E nutrition and blood and tissue levels in free-ranging flying foxes to more accurately interpret the observations described in this study.

Vitamin E is synthesized only by plants,<sup>1</sup> and natural sources include grains and green leafy plants. The original bat diet of primarily fruits and vegetables was low in these sources. Although large flying foxes have traditionally been thought to feed exclusively on fruits and/or flowers, it has recently been shown that some species chew leaves and ingest the fiber-free juices.<sup>11</sup> These leaves may be an important source of essential nutrients lacking in a purely frugivorous diet. The Lube Foundation keepers have observed several species of flying foxes crawling to the ground to consume or chew grasses.

Traumatic causes of myocardial injury include central nervous system injury, stress, exertional rhabdomyolysis, radiation, electrical defibrillation, and hemorrhagic shock.<sup>16</sup> Of these, only stress could be implicated in the development of cardiomyopathy in these bats. Stress-induced cardiac necrosis without accompanying skeletal muscle lesions has been observed in a variety of small mammals and primates.<sup>16</sup> The presence of cardiomyopathy in adult male wild-caught animals, three of which had undergone surgery just months before presentation, is suggestive of stress as a contributing factor. An argument against this is normal food intake and high fecundity in the wild-caught breeding groups as well as absence of overcrowding and abnormal behaviors. Stress can exacerbate and/or precipitate cardiomyopathy in bats with marginal to deficient tissue vitamin E levels. There was no evidence in these bats of endocrine causes of myocardial injury, such as glucocorticoid excess, functional phoe-

chromocytomas, diabetes mellitus, hyperthyroidism, hypothyroidism, and growth hormone excess.<sup>16</sup>

The abnormal clinicopathologic changes are consistent with organ dysfunction secondary to congestive heart failure. Moderate to severe uremia and hyperkalemia with normal creatinine probably reflect decreased renal blood flow and impaired function. The elevated AST and ALT levels indicate hepatic injury, possibly due to severe congestion. The anemia may be due to either hemodilution from fluid retention and/or impaired red cell production secondary to hypovitaminosis E.<sup>1</sup>

No attempt was made to parenterally administer high doses of vitamin E to affected bats because of the delay in determining a possible cause. The efficacy of this approach is questionable because the damage that produces cardiac dilatation is probably irreversible by the time of diagnosis. Vitamin E treatment is, however, indicated to prevent further myocardial injury. The change in the diet at the beginning of 1994 appears to have increased the dietary intake of vitamin E sufficient to produce tissue levels within normal mammalian ranges. No further cases of dilated cardiomyopathy have been diagnosed since July 1994. The diet has recently been modified by decreasing the vitamin supplement to give a calculated vitamin E content of 240 mg/kg dry weight, in balance with other nutrients. This change was made to reduce the dietary intake of some micronutrients (e.g., iron) present in the vitamin supplement.

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