

Metabolic and respiratory status of stranded juvenile loggerhead sea turtles (*Caretta caretta*): 66 cases (2008–2009)

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Objective—To document venous blood gas, acid-base, and plasma biochemical values for stranded juvenile loggerhead turtles at admission to a rehabilitation facility, compare these values among stranding causes, investigate differences in these values for turtles that survived versus those that died, and establish the baseline values for successfully rehabilitated loggerhead turtles (*Caretta caretta*).

Design—Retrospective case series.

Animals—66 stranded juvenile loggerhead turtles that were hospitalized between 2008 and 2009.

Procedures—Venous blood gas, acid-base, and plasma biochemical values at the time of admission were compared retrospectively among turtles with different stranding causes. Initial results were compared between turtles that survived and turtles that died. Results for survivors were compared between the time of admission and time of release.

Results—57 (86.36%) turtles had various types of acid-base disorders at the time of admission to the rehabilitation facility. Of these, 33 (57.9%) had mixed acid-base disorders and 24 (42.1%) had primary acid-base disorders. All acid-base disorders were classified as mild to moderate, except 1 case of severe metabolic and respiratory acidosis. Except for the debilitated turtles (in which the mean initial glucose concentration was much lower than that observed for the rest of turtles), there was no difference in initial values when comparing stranding causes. Turtles that died during rehabilitation had significantly higher initial anion gap and osmolality, compared with turtles that survived.

Conclusions and Clinical Relevance—Acid-base disorders were present in most stranded juvenile loggerhead turtles. Evaluation of accurately obtained, temperature-corrected venous blood gas, acid-base, and plasma biochemical values can provide important clinical and prognostic information and a valuable basis for the implementation of adequate and rapid treatment for stranded loggerhead turtles admitted to rehabilitation facilities. (*J Am Vet Med Assoc* 2013;242:396–401)

Veterinary care in sea turtle rehabilitation facilities is an essential component of sea turtle conservation around the world. Clinical and pathological studies are continuing to contribute to a better understanding of problems in stranded sea turtles and provide a basis to guide conservation efforts. Five species of sea turtles have been reported around the Canary Islands: loggerhead (*Caretta caretta*), green turtle (*Chelonia mydas*), leatherback (*Dermochelys coriacea*), hawksbill (*Eretmochelys imbricata*), and olive ridley (*Lepidochelys olivacea*).^{1,2} However, loggerhead turtles are the most common species found around the Canary Islands coming from the US western Atlantic by the Gulf Stream.^{1,3}

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ABBREVIATION

SCL Straight carapace length

Diseases and causes of death among sea turtles stranded in the Canary Islands have been described.^{4,5} Variations of the different plasma biochemical and hematologic parameters of loggerhead sea turtles according to the cause of stranding or disease have also been reported.⁶ However, reports^{7–9} on blood gas and acid-base status of loggerhead turtles, particularly stranded juvenile animals, are limited. The goals of the study reported here were to document venous blood gas, acid-base, and plasma biochemical values for stranded juvenile loggerhead turtles at the time of admission to our rehabilitation facility; compare these values among stranding causes; investigate differences in these values between turtles that survived and those that died; and establish baseline values for rehabilitated juvenile loggerhead turtles.

Materials and Methods

Sea turtle rehabilitation at the Tafira Wildlife Rehabilitation Center was conducted with authorization of

the Wildlife Department of the Canary Islands Government, in compliance with guidelines of the Tafira Wildlife Rehabilitation Center Animal Care Committee.

Criteria for selection of cases—All turtles selected for this study were identified as juvenile specimens on the basis of SCL.^{10,11} Medical records for all live, stranded juvenile loggerhead turtles admitted to the Tafira Wildlife Rehabilitation Center between 2008 and 2009 were reviewed. Data collected included date and cause of stranding; weight; SCL; cloacal temperature at the time of venous blood sample collection; success or failure of rehabilitation; venous pH, PO_2 , and PCO_2 values; anion gap; osmolality; and concentrations of lactate, sodium, potassium, chloride, glucose, BUN, and HCO_3^- at the time of admission (before administration of any drugs) and, where applicable, immediately before the release of rehabilitated turtles into the wild (without drug administration during the last 7 days of hospitalization).

Procedures—A standardized method of blood sample collection and analysis was used for this study.¹² To minimize artifactual changes in hematologic variables due to animal handling, 1 mL of venous blood was anaerobically collected from the cervical sinus of each turtle into a non-heparinized syringe immediately at time of admission. Each sample was immediately analyzed via a portable electronic blood analyzer^a for pH, PO_2 , PCO_2 , and lactate concentration with cartridges^b for those analytes and for concentrations of sodium, potassium, chloride, glucose, and BUN with cartridges^c for those analytes. Because the portable electronic blood analyzer performs analysis of samples at 37°C (98.6°F), the temperature of each turtle was taken to correct the parameters with equations and values considered more appropriate for sea turtles than the portable electronic blood analyzer human-derived algorithms.^{13–15} The body temperature of each turtle was recorded with a digital thermometer^d inserted at least 10 cm into the cloaca and placed against the cloacal wall. The pH,^{8,13} PCO_2 ,^{8,14,16,17} and PO_2 ^{8,9,14,17} were corrected for the turtle's body temperature via the following equations:

$$\begin{aligned}\text{Temperature-corrected pH} &= \text{pH} + 0.014(\Delta T) \\ \text{Temperature-corrected } \text{PCO}_2 &= \text{PCO}_2(10^{-0.019\Delta T}) \\ \text{Temperature-corrected } \text{PO}_2 &= \text{PO}_2(10^{-0.0058\Delta T})\end{aligned}$$

where ΔT is 37°C – body temperature.

The temperature-corrected HCO_3^- concentration was calculated via the Henderson-Hasselbach equation. The solubility coefficient for CO_2 (αCO_2) and the pKa (logarithmic acid dissociation constant) value were calculated for each patient via species-specific equations for sea turtles.^{8,15} The αCO_2 ranged from 0.039 to 0.049, and the pKa value ranged from 6.15 to 6.20.

Anion gap (mmol/L)¹⁸ and osmolality (mOsm/kg)¹⁹ were calculated via the following equations:

$$\text{Anion gap} = (\text{sodium concentration} + \text{potassium concentration}) - (\text{chloride concentration} + \text{temperature-corrected } \text{HCO}_3^- \text{ concentration})$$

$$\text{Osmolality} = 2 (\text{sodium concentration} + \text{potassium concentration}) + (\text{glucose concentration}/18) + (\text{BUN concentration}/2.8)$$

Rehabilitation sea turtles were placed individually in outdoor pools with a continuous flow of sea water, capacity of 10,000 L, and depth of 1 m, providing plenty of room for swimming. Sea turtles were fed fresh or frozen fish (sardine, Atlantic mackerel, and Atlantic horse mackerel) once a day. Natural light periodicity ranged from 8 hours in the winter to 14 hours in the summer. Clinical evaluation, including physical examination, evaluation of swimming activity, core body temperature (measured from the cloaca), food ingestion, weight, and SCL, was performed daily following a complete clinical assessment protocol.^{20–22} Treatments included, when necessary, amputation of the injured flipper, surgical removal of fish hooks, (tube) feeding, cleaning and debridement of the external traumatic injuries, antimicrobial treatment, fluid therapy, correction of floating problems (lung injury or intestinal impaction), and mineral oil gavages. Each turtle was released when it was determined to be convalescent on the basis of clinical parameters and to be in good physical condition. Immediately prior to release, a blood sample from each turtle was analyzed for the same parameters that had been analyzed at the time of admission.

Statistical analysis—Statistical analysis was performed with a commercial statistics program.^e To determine whether the data were normally distributed, the Kolmogorov-Smirnov test was used. Paired data were statistically compared via a paired *t* test for data normally distributed or via a Wilcoxon signed rank test for data that were not normally distributed. Also, initial and convalescent data were statistically compared according to SCL of the turtles via a Mann-Whitney signed rank test for independent data that were not normally distributed. Similarly, initial data from turtles that survived were compared with those from turtles that died. To evaluate the correlation and dependence between 2 random variables, the Pearson correlation coefficient was used. Values of $P < 0.05$ were considered significant. Because of the small sample size, the initial data set according to the cause of stranding or disease was examined by means of descriptive statistical analysis.

Results

One hundred two live loggerhead turtles were admitted to the Tafira Wildlife Rehabilitation Center during the study period. Of these 102 sea turtles, 66 loggerhead turtles were chosen on the basis of our selection criteria (juvenile loggerhead), and initial blood values at admission were compared with those of convalescent values in 60 turtles. Six turtles that had initial data collected did not have convalescent data because during the hospitalization period, they died of sepsis (Table 1) subsequent to severe fracture of carapace ($n = 1$), severe lesions caused by hooks (2), and severe debilitation (3). Median rehabilitation times for different stranding categories were 46 days (range, 14 to 306 days) for fishing net entanglement (48 [72.97%] turtles), 17 days (range, 2 to 38 days) for hook ingestion (6 [9.1%]), 30 days (1 to 127 days) for debilitation (5 [7.6%]), 80.5 days (range, 37 to 124 days) for traumatic injury caused by boat strike (2 [3%]), 31 days for buoyancy

Table 1—Mean (median) ± SD (range) values for physical characteristics, days of hospitalization, and cloacal temperatures for 66 stranded juvenile loggerhead turtles.

Variable	Survived (n = 60)	Died (n = 6)
Body weight (kg)	6.43 (5.12) ± 4.83 (1.03–20.6)	14.11 (8.40) ± 17.01 (0.66–39)
SCL (cm)	32.93 (32.75) ± 8.74 (18–50)	42.10 (39) ± 16.59 (18–61)
Hospitalization time (d)	57.89 (45) ± 45.77 (8–306)	13 (7) ± 16.75 (1–37)
Initial temperature (°C)	22.47 (23.16) ± 1.99 (17.1–24.7)	21.53 (21.35) ± 2.77 (18.2–25.1)
Convalescent temperature (°C)	21.77 (22.40) ± 1.84 (17.3–24.8)	NM

NM = Not measured.

Table 2—Mean (median) ± SD (10th and 90th percentiles) temperature and pH-corrected initial and convalescent blood gas, acid-base, and plasma biochemical values for juvenile loggerhead sea turtles.

Variable	Initial values (n = 60)		Convalescent values (n = 60)		P value
	Mean (median) ± SD	10th percentile–90th percentile	Mean (median) ± SD	10th percentile–90th percentile	
pH*	7.50 (7.50) ± 0.09	7.38–7.59	7.56 (7.56) ± 0.04	7.49–7.63	< 0.001
Pco ₂ (Torr)*	31.07 (30.40) ± 5.45	25.67–39.89	33.45 (33.70) ± 4.94	27.36–41	< 0.05
Po ₂ (Torr)	70.98 (70.72) ± 14.14	56.56–87.53	61.64 (59.16) ± 17.67	47–71.78	< 0.001
HCO ₃ ⁻ (mmol/L)*	28.83 (28.69) ± 5.98	21.86–34.78	36.72 (36.54) ± 5.21	29.5–44.16	< 0.001
Lactate (mmol/L)	3.08 (2.02) ± 3.47	0.33–7.20	0.55 (0.30) ± 0.48	0.30–1.01	< 0.001
Sodium (mmol/L)	148.52 (148) ± 4.29	144–153	149.54 (149) ± 3.21	146–153	< 0.01
Potassium (mmol/L)	3.35 (3.40) ± 0.47	2.80–3.90	3.28 (3.20) ± 0.45	2.70–4	0.227
Chloride (mmol/L)*	113.74 (113) ± 5.96	107–120	110.64 (111) ± 4.50	105–118	< 0.001
Anion gap (mmol/L)	9.25 (9.38) ± 4.75	3.66–14.43	5.41 (5.42) ± 4.91	0.60–11.37	< 0.001
Glucose (mg/dL)*	109.89 (112.50) ± 43.43	51.80–152.30	109.31 (110) ± 16.15	85–130	0.599
BUN (mg/dL)†	62.63 (56) ± 25.79	33.60–103.40	131.76 (140) ± 19.36	106–140	< 0.001
Osmolality (mOsmol/kg)†	332.08 (330.64) ± 14.58	314.53–349.10	358.85 (360.62) ± 10.61	344.88–369.62	< 0.001

*Paired data set was normally distributed. †Mean and SD convalescent BUN and osmolality values underestimate the true values because BUN values exceeded the analytic range of the analyzer (140 mg/dL) in 45 sea turtles. Six turtles that had initial data collected did not have convalescent data because they died during the hospitalization period as a result of sepsis subsequent to severe fracture of the carapace (n = 1), severe lesions caused by hooks (2), and severe debilitation (3).

disorder (1 [1.5%]), 14 days for plastic ingestion (1 [1.5%]), and 39 days (range, 37 to 41 days) for unidentified causes (3 [4.5%]).

Survival—Initial and convalescent temperature- and pH-corrected blood gas, acid-base, and plasma biochemical values were measured from turtles that were successfully rehabilitated (Table 2). Initial values for the same parameters were measured from turtles that died during the rehabilitation period (Table 3). When comparing initial values at the time of hospital admission of turtles that died with those of turtles that survived, turtles that died during convalescence had significantly ($P < 0.05$) higher initial osmolality and initial temperature-corrected anion gap (350.82 ± 25.15 mOsm/kg and 13.74 ± 6.03 mmol/L, respectively; Mann-Whitney Z value, -2.584) than did turtles that survived (332.08 ± 14.58 mOsm/kg and 9.25 ± 4.75 mmol/L, respectively; Mann-Whitney Z value, -2.108). No significant difference was found when comparing the rest of the hematologic variables.

Acid-base disorders—To evaluate imbalances in the acid-base status of the turtles, data obtained from 60 turtles were analyzed by comparing initial and convalescent temperature-corrected pH, HCO₃⁻ concentration, and Pco₂. Regarding the 6 turtles that died during the convalescence period, initial data for these parameters were compared with the initial values of those that survived. In addition, lactate concentration, tem-

Table 3—Mean (median) ± SD (range) temperature and pH-corrected initial blood gas, acid-base, and plasma biochemical values for juvenile loggerhead sea turtles that died during convalescence.

Variable	Initial values (n = 6)	
	Mean (median) ± SD	Range
pH	7.5 (7.56) ± 0.23	7.05–7.68
Pco ₂ (Torr)	30.01 (30.36) ± 8.42	16.25–41.08
Po ₂ (Torr)	53.28 (54.83) ± 11.73	38.52–68.37
HCO ₃ ⁻ (mmol/L)	29.63 (33.85) ± 10.24	11.05–39.62
Lactate (mmol/L)	5.55 (2.94) ± 6.71	0.30–18.74
Sodium (mmol/L)	155.17 (153) ± 8.70	146–171
Potassium (mmol/L)	3.20 (3.15) ± 0.72	2.40–4.30
Chloride (mmol/L)	115 (112) ± 13.80	102–140
Anion gap (mmol/L)	13.74 (12.34) ± 6.04	7.47–24.25
Glucose (mg/dL)	104.83 (66) ± 103.34	51.80–152.30
BUN (mg/dL)	79.17 (73) ± 24.63	33.60–103.40
Osmolality (mOsmol/kg)	350.82 (342.53) ± 25.15	330.64–400.21

perature-corrected anion gap, and electrolyte concentrations were also considered for a complete interpretation of the results.²³ Fifty-seven (86.4%) turtles had some type of acid-base disorder at the time of hospital admission. Of these, 33 (57.9%) had mixed acid-base disorders and 24 (42.1%) had primary acid-base disorders. All acid-base disorders were classified as mild to moderate, except 1 case of severe metabolic and respiratory acidosis (pH, 7.05; temperature-corrected HCO₃⁻ concentration, 11.05 mmol/L; temperature-corrected Pco₂, 35.3 Torr). Forty-nine (74.2%) turtles had some type of metabolic acidosis, including metabolic and respiratory acidosis (26 [39.4%]), metabolic acidosis

with complete respiratory compensation determined on the basis of detection of low P_{CO_2} and subsequent normalization of pH (12 [18.2%]), metabolic acidosis with partial respiratory compensation (7 [10.6%]), and mixed metabolic acidosis and respiratory alkalosis (4 [6.06%]). The remaining mixed acid-base disorders were associated with alkaline imbalances ($n = 3$ [4.5%]) and respiratory acidosis (5 [7.6%]). No biochemical abnormalities related to acid-base disorders were detected in 9 turtles.

Glucose concentration—The mean \pm SD initial glucose concentration for debilitated turtles ($n = 5$) was much lower (52.80 ± 43.01 mg/dL; median, 31 mg/dL; range, 20 to 123 mg/dL) than the mean initial glucose concentrations of stranded turtles in all other stranding categories (114.57 ± 40.34 mg/dL; median, 118 mg/dL; range, 20 to 276 mg/dL).

Influence of size and weight—When comparing all blood values according to the SCL, we observed a significant difference for the lactate concentration. Turtles with an SCL ≥ 42 cm (juvenile turtles¹¹) had significantly higher initial lactate concentrations (5.11 ± 3.56 mmol/L; median, 4.81 mmol/L; range, 1.65 to 18.74 mmol/L) than did turtles with an SCL < 42 cm (juvenile pelagic turtles: 2.16 ± 2.12 mmol/L; median, 1.62 mmol/L; range, 0.30 to 11.92 mmol/L) ($P < 0.01$; Mann-Whitney Z value, 0.303).

In addition, a positive correlation ($r = 0.478$; $P < 0.001$) was observed between turtle weight and initial lactate concentrations. When studying the association between SCL and other variables, we only observed significant differences for the BUN concentration. We observed a negative association between SCL and BUN concentrations ($r = -0.376$; $P < 0.01$).

Values generated by the portable electronic blood analyzer at 37°C—Because results from temperature correction of blood gas and plasma biochemical analysis of ectothermic animals remain problematic when clinical portable analyzers designed for mammals such as the portable electronic blood analyzer are used, the raw data generated by the analyzer at 37°C are provided (Table 4). The pH measured by the portable electronic blood analyzer at 37°C was 2.65% lower than the pH after temperature correction (Wilcoxon Z value, -7.009 ; $P < 0.001$). The P_{CO_2} and P_{O_2} measured by the portable electronic blood analyzer at 37°C were 46.15% (-32.84 ; $P < 0.001$) and 15.78% (Wilcoxon Z value, -7.001 ; $P < 0.001$) higher than those after temperature correction, respectively. However, the temperature-corrected portable electronic blood analyzer values for pH (-1.377 ; $P = 0.173$) and P_{CO_2} (0.250 ; $P = 0.804$) were similar enough to our cal-

culated values to be clinically useful, whereas the portable electronic blood analyzer-corrected values for P_{O_2} differed by 46.9% from our calculated values (Wilcoxon Z value, -6.791 ; $P < 0.001$).

Discussion

In the present study, acid-base disorders were present in most stranded juvenile loggerhead turtles. Turtles that died during rehabilitation had significantly higher initial anion gap and osmolality, compared with turtles that survived. Evaluation of accurately obtained, temperature-corrected venous blood gas, acid-base, and plasma biochemical values can provide important clinical and prognostic information and a valuable basis for the implementation of adequate and rapid treatment for stranded loggerhead turtles admitted to rehabilitation facilities.

Although hematologic and plasma biochemical values from stranded juvenile loggerhead turtles have been described,^{6,12} reports⁷⁻⁹ of blood gas and acid-base status of stranded juvenile loggerhead turtles are limited. Acidosis was detected in most turtles examined in the present study. Mean initial temperature-corrected pH, P_{CO_2} , and HCO_3^- concentration values were significantly lower than convalescent values, whereas mean initial lactate concentration and temperature-corrected anion gap were higher than convalescent values. There were no significant differences between initial temperature-corrected pH, P_{CO_2} , and HCO_3^- concentration for turtles that died and initial values for those that survived, except for a turtle with severe metabolic and respiratory acidosis that died. In addition, turtles that died had significantly higher initial osmolality and temperature-corrected anion gap than did survivors. Acidosis has also been described in loggerhead turtles experimentally subjected to involuntary submergence²⁴ and captured by trawl⁹ and in cold-stunned Kemp's ridley turtles.¹⁷

Metabolic acidosis in the turtles of the present study was most likely caused by accumulation of lactic acid, as revealed by the increased anion gap.²⁵ Potential causes of acid gain include poor tissue perfusion due to shock, extreme exercise trying to get out of fishing nets, involuntary submergence, capture by nets, and decreased acid excretion due to renal dysfunction. In addition, the positive correlation between turtle weight and initial lactate concentration was also observed in loggerhead turtles captured by trawl, suggesting a greater degree of anaerobic metabolism during trawl capture in larger turtles.⁹ Mean initial temperature-corrected P_{CO_2} was significantly lower than mean convalescent temperature-corrected P_{CO_2} in this study. This could be due to

Table 4—Mean (median) \pm SD (10th and 90th percentiles) initial and convalescent pH, P_{CO_2} , and P_{O_2} for juvenile loggerhead turtles measured with a portable electronic blood analyzer at 37°C (98.6°F).

Variable	Initial values (n = 66)		Convalescent values (n = 60)	
	Mean (median) \pm SD	10th percentile–90th percentile	Mean (median) \pm SD	10th percentile–90th percentile
pH	7.31 (7.32) \pm 0.08	7.22–7.40	7.35 (7.35) \pm 0.04	7.29–7.40
P_{CO_2}	55.8 (54.6) \pm 9.63	43.7–70.34	64.99 (63.3) \pm 10.58	53.2–81.2
P_{O_2}	84.87 (84) \pm 17.21	66.2–104.8	73.56 (72) \pm 12.2	57.8–89.1

moderate hyperventilation at the time of admission as a compensatory mechanism to counteract the metabolic acidosis. Another study²⁶ found CO₂ concentrations to be significantly higher in stranded turtles versus foraging and nesting turtles, most likely because of decreased respiratory ventilation in stranded turtles. However, only 5 stranded loggerheads were included in that study.²⁶

Although an increase of potassium concentration has been observed in loggerhead turtles with acute metabolic acidosis,²⁴ we did not observe a significant difference between initial and convalescent potassium concentrations. Different etiologic pathways may explain why 7 of 60 turtles had severe hypoglycemia but 10 of 60 had moderate hyperglycemia. We believe that the high initial glucose concentration may be a result of stress associated with capture and transport before clinical examination. Stress-associated hyperglycemia has been reported in sea turtles.^{27,28} Other causes for hyperglycemia include liver disease, pancreatic disease, and overcompensation of gluconeogenic mechanisms.²⁹ Hypoglycemia in reptiles may be attributed to prolonged anorexia, severe hepatobiliary disease, and septicemia.³⁰ The mean initial glucose concentration for turtles hospitalized because of debilitation was much lower than that for the rest of turtles. Only 1 turtle hospitalized because of debilitation had normal glucose values. Anemia, hypoproteinemia, and hypoglycemia have been reported in debilitated wild sea turtles.⁶

The convalescent temperature-corrected pH, HCO₃⁻ concentration, Pco₂, and Po₂; concentrations of blood lactate, electrolytes, glucose, and BUN; and osmolality were similar to those reported for loggerhead turtles.^{7-9,12,22,24,26,31-34} The mean convalescent BUN concentration and osmolality reported in our study underestimated the true mean because 45 turtles had convalescent BUN concentrations above the range of the analyzer. Convalescent temperature-corrected anion gap in our study was lower than that reported for healthy loggerhead turtles.³² Hypoalbuminemia can cause decreased anion gap; however, albumin concentration was not measured in the study reported here. Because studies^{17,32} on anion gap in sea turtles are scarce, further investigations on anion gap and clinical diagnosis in sea turtles are needed.

The portable electronic blood analyzer has been used to determine the venous blood gases in marine turtles.^{8,9,16,35} However, correction for body temperature of pH, Po₂, Pco₂, and HCO₃⁻ concentration of blood samples of sea turtles with portable analyzers has not always been made.^{8,9,16} Our results support studies¹⁷ that found temperature- and pH-corrected acid-base values to be more representative of the true physiologic status of the turtle. Use of noncorrected values undoubtedly leads to erroneous conclusions in the diagnosis of the metabolic and respiratory status of the turtles because the noncorrected pH is lower than after temperature correction, whereas the noncorrected Pco₂ and Po₂ are higher than after temperature correction. In addition, temperature corrections generated by the portable electronic blood analyzer were not valid for Po₂, differing by 46.9% from our calculated values. Corrections to Po₂ made by the portable electronic blood analyzer were inaccurate because the formulas assumed a much higher

enthalpy of formation required for oxygenation of hemoglobin than has been measured in loggerheads.^{8,36} Thus, temperature-based adjustment is essential to obtain accurate values for sea turtles.

Several studies^{7,13,17,31,37} have been reported of the effect of temperature on some physiologic functions of sea turtles, such as ventilation and acid-base balance. It has been observed that the blood pH of sea turtles decreases as body temperature increases.^{7,13,15,17,37} Several studies found that the venous Pco₂^{7,13,17,37} and Po₂^{7,13,37} decrease as body temperature decreases. No temperature effect on the interpretation of the results was observed in our study because of the low difference between initial temperature (22.7°C [72.8°F]) and convalescent temperature (21.8°C [71.9°F]). In addition, the range of temperature for the turtles of our study was within that described in another study¹³ (15° to 25°C [59° to 77°F]), in which pH, Pco₂, and HCO₃⁻ concentration did not change significantly with the body temperature of the turtles.

This study shows that accurately obtained, temperature-corrected venous blood gas, acid-base, and plasma biochemical values provide essential information for veterinarians in the treatment of stranded loggerhead turtles.

- a. i-STAT, Heska, Loveland, Colo.
- b. CG4+ cartridges, Heska, Loveland, Colo.
- c. EC8+ cartridges, Heska, Loveland, Colo.
- d. Digi-Sense Thermocouple T, Cole-Parmer Instrument Co, Vernon Hills, Ill.
- e. PASW, version 18.0, SPSS Inc, Chicago, Ill.

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