Metabolic and respiratory derangements associated with death in cold-stunned Kemp's ridley turtles (*Lepidochelys kempii*): 32 cases (2005–2009)

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Objective—To assess selected clinicopathologic variables at hospital admission (day 1) for cold-stunned Kemp's ridley turtles (*Lepidochelys kempii*) that died during the first 3 days after admission (nonsurvivors) and turtles that survived (survivors) and to determine the percentage change of each variable from day 1 to day of death (nonsurvivors) or to day 2 or 3 of hospitalization (survivors).

Design—Retrospective case-control study.

Animals—64 stranded, cold-stunned Kemp's ridley turtles hospitalized from October 2005 through December 2009.

Procedures—Blood gas, pH, Hct, and selected biochemical values in blood samples determined on day 1 and day of death (nonsurvivors; n = 32) or day 2 or 3 of hospitalization (survivors; 32) were obtained from medical records. For each variable, initial values and percentage changes (from initial values to values at the day of death or day 2 or 3 of hospitalization) were compared between survivors and nonsurvivors.

Results—Compared with blood analysis findings for survivors, nonsurvivors initially had significantly higher potassium concentration and Pco_2 and significantly lower Po_2, pH, and bicarbonate concentration than did survivors. For the first 2 or 3 days of hospitalization, percentage changes in potassium, lactate, and ionized calcium concentrations were significantly higher and percentage changes in pH and plasma glucose and bicarbonate concentrations were significantly lower in nonsurvivors.

Conclusions and Clinical Relevance—At hospital admission, cold-stunned Kemp's ridley turtles were affected by metabolic and respiratory derangements; severe derangements were associated with death. Evaluation of blood gas, pH, Hct, and selected clinicopathologic variables provided useful clinical and prognostic information during rehabilitation of cold-stunned Kemp's ridley turtles. (*J Am Vet Med Assoc* 2012;240:317–323)

Kemp's ridley turtle (*Lepidochelys kempii*) is the smallest and rarest species of sea turtle. It is listed as critically endangered by the World Conservation Union and as endangered by the US Endangered Species Act.[1] Adult Kemp's ridley turtles are usually found in the Gulf of Mexico, although juveniles of this species commonly travel to the northeastern coast of the United States to feed during the summer.[2,3] Juveniles leave these summer foraging grounds in autumn when the water temperature begins to decrease. Turtles that fail to migrate to warmer waters may become cold-stunned at water temperatures < 10°C (50°F).[4–7] Cold-stunned juvenile Kemp's ridley turtles are often found stranded on beaches of New York and Massachusetts from October through December of each year.[4–7] Such strandings are thought to be caused by a combination of geographic, oceanographic, and meteorologic conditions.[6]

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Abbreviations

iCa	Ionized calcium
iCa_{cor}	pH-corrected ionized calcium
iMg	Ionized magnesium
iMg_{cor}	pH-corrected ionized magnesium
Pco_{2TC}	Temperature-corrected Pco_2
pH_{TC}	Temperature-corrected pH
Po_{2TC}	Temperature-corrected Po_2

In Massachusetts, cold-stunned juvenile Kemp's ridley turtles that are found alive are recovered by a network of volunteers and staff of the Massachusetts Audubon Society and transported to the hospital at the New England Aquarium, Boston, for medical care and long-term rehabilitation. Details of medical management of cold-stunned turtles have been previously described.[8] Briefly, turtles are gradually warmed over several days and treated for dehydration, acid-base and electrolyte derangements, cardiorespiratory depression, and concurrent pathological conditions such as pneumonia or traumatic injuries.[8] During the first few days of hospitalization, treatments are selected on the basis of physical examination, clinicopathologic,

and radiographic findings. These treatments often include mechanical ventilation; administration of fluids that may contain additional potassium, calcium, dextrose, and bicarbonate; and administration of atropine, doxapram, and antimicrobials.[8] Results of hematologic, blood biochemical, and plasma biochemical analyses in cold-stunned Kemp's ridley turtles have been described.[9–12]

Abnormalities in blood gas, pH, lactate, and electrolyte values have been extensively evaluated in domestic animals, and results of these analyses aid clinicians in the determination of prognosis and selection of proper treatments such as mechanical ventilation and administration of fluids.[13,14] Blood gas and pH data for Kemp's ridley turtles that survived cold stunning were recently described[9]; however, to the authors' knowledge, similar values have not been reported for turtles that did not survive cold stunning. The purpose of the study reported here was to assess values of selected clinicopathologic variables, including blood gas and pH values, at the time of hospital admission (day 1) for cold-stunned Kemp's ridley turtles that died during the first 3 days of hospitalization (nonsurvivors) and turtles that survived (survivors) and to determine the percentage change of each variable from day 1 to day of death (nonsurvivors) or to day 2 or 3 of hospitalization (survivors).

Materials and Methods

Case selection—Medical records of all live, cold-stunned Kemp's ridley turtles that were hospitalized at the New England Aquarium from October 2005 through December 2009 were reviewed. Turtles were included in the nonsurvivor group if they died of natural causes within the first 3 days of hospitalization. For each nonsurvivor, a successfully rehabilitated turtle that had been hospitalized closest in time to the nonsurvivor (within a maximum of ± 2 days) was selected for inclusion in a comparison survivor group. This method was chosen to maximize the likelihood that survivors and nonsurvivors had been exposed to similar environmental (ie, weather and oceanographic) conditions prior to hospitalization.

Medical records review—For each patient, information obtained from the medical records included date of admission to the hospital (day 1); weight, straight carapace length, and cloacal temperature at the time of admission; environmental temperature at which the turtle was maintained; outcome of the first 3 days of hospitalization (survival or death); and date of death (ie, day of hospitalization) for nonsurvivors. Results of Hct and blood gas, acid-base, and blood biochemical analyses determined on the first day of hospitalization (day 1) and day of death for nonsurvivors and on corresponding days of hospitalization for matched survivors were recorded. Variables of interest included blood pH, P_{CO_2}, P_{O_2}, Hct, anion gap, osmolality, and iCa, iMg, sodium, potassium, chloride, glucose, lactate, bicarbonate, and BUN concentrations.

Procedures—Rehabilitation of Kemp's ridley turtles at the New England Aquarium was conducted with authorization of the US Department of the Interior Fish and Wildlife Service and the US Department of Commerce National Marine Fisheries Service. For each turtle, blood sample collections were performed by use of a standard method during the period of interest in this study. For each sample, blood was collected from an external jugular vein into a 1- or 3-mL heparinized syringe.[9] The volume of blood collected on day 1 was usually 3 mL, and the sample that remained after analysis was archived for future use. The volume of blood collected on day 2 or day 3 was usually 0.5 mL. Biochemical analysis of whole blood was performed by use of a point-of-care analyzer[a] in accordance with the manufacturer's guidelines; blood was transferred directly from the syringe to the analyzer. The Hct was manually determined as previously described.[10] Because the blood analyzer operated at 37°C (98.6°F), pH, P_{CO_2}, and P_{O_2} values were corrected for the patient's temperature (cloacal temperature [used in analysis of blood samples on day 1] or environmental temperature [used in analysis of blood samples on all subsequent days]).[15–20] Published equations were used for temperature correction, pH correction, and bicarbonate, anion gap, and osmolality calculations[13,15–24] (**Appendix**). Calculation of the iCa_{cor} concentration and the iMg_{cor} concentration was performed by use of the pH_{TC}.[21,22] Bicarbonate concentration in blood samples was calculated by use of the Henderson-Hasselbalch equation, pH_{TC}, and $P_{CO_{2TC}}$. The αCO_2 and pK values were calculated by use of previously described species-specific equations for use in analysis of blood samples of Kemp's ridley turtles.[23] The percentage changes in the Hct and blood gas, acid-base, and biochemical variables for nonsurvivors that died on day 2 or 3 were calculated by use of values from day 1 and from day of death. For comparison, the percentage changes in the values of these variables were calculated for survivors by use of results from day 1 and results from day 2 or 3 (whichever day corresponded to the day of death of the matched nonsurvivor).

Statistical analysis—Results of analyses of blood samples obtained on day 1 and value of percentage change in each variable were compared for survivors versus nonsurvivors by use of a 1-way ANOVA.[b] Data that failed to meet assumptions of normality and equal variance were compared by use of a nonparametric Kruskal-Wallis 1-way ANOVA on ranks.[b] For 1 nonsurvivor that had a blood lactate concentration and P_{CO_2} value that were higher than the analytic range of the analyzer, the maximum recorded values of these 2 variables that were found in the other turtles in this study were assigned. Values of $P < 0.05$ were considered significant.

Results

Of the 173 cold-stunned Kemp's ridley turtles that were admitted to the hospital at the New England Aquarium during the period of interest in the present study, 32 met the criterion for inclusion in the nonsurvivor group and 32 were selected for the survivor group (**Table 1**). Four turtles were evaluated in 2005, 18 in 2006, 2 in 2007, 16 in 2008, and 24 in 2009. Of the 32 nonsurvivors, 12 died within the first 24-hour period, 13 died within the second 24-hour period, and 7 died within the third 24-hour period after admission. Analysis of blood samples was not performed on the day of death for 1 turtle that died on day 2. Therefore, data from 19 turtles were used to calculate the percentage change between initial values and values at day 2 or 3 for Hct and blood gas, acid-base, and biochemical variables.

Assumptions that data were normally distributed and equal in variance were met for Hct, anion gap, and iCa_{cor}, chloride, BUN, and bicarbonate concentrations in samples collected on day 1; data for all other variables did not meet these assumptions. The pH_{TC} ($P = 0.001$),

Table 1—Weight, straight carapace length, and cloacal temperature (determined on the day of hospital admission [day 1]) for 32 cold-stunned Kemp's ridley turtles that died during the first 3 days of hospitalization (nonsurvivors) and 32 turtles that survived (survivors).

Variable	Survivor group (n = 32)		Nonsurvivor group (n = 32)	
	Mean ± SD	Range	Mean ± SD	Range
Weight (kg)	2.48 ± 0.86	0.90–4.60	2.72 ± 1.09	1.25–7.02
Straight carapace length (cm)	25.3 ± 3.3	18.8–31.9	26.6 ± 3.5	20.3–37.0
Cloacal temperature (°C)	12.3 ± 2.7	8.0–17.0	11.6 ± 3.2	5.3–18.6

For each nonsurvivor, a successfully rehabilitated turtle that had been evaluated closest in time to the nonsurvivor (within a maximum interval of ± 2 days) was selected for inclusion in a comparison survivor group.

Table 2—Values of selected clinicopathologic variables determined on day 1 in blood samples of survivor and nonsurvivor cold-stunned Kemp's ridley turtles described in Table 1.

Variable	Survivor group (n = 32)				Nonsurvivor group (n = 32)			
	Mean ± SD	Median	Range	10th–90th percentile range	Mean ± SD	Median	Range	10th–90th percentile range
Hct (%)	42 ± 9	40	29–71	32–50	46 ± 11	47	15–65	32–58
pH_{TC}*	7.53 ± 0.17	7.57	7.05–7.79	7.29–7.70	7.35 ± 0.20	7.35	6.90–7.75	7.09–7.60
Pco_{2TC} (mm Hg)*	27.5 ± 16.4	22.4	13.5–84.0	16.6–41.2	34.3 ± 13.7	35.4	15.1–80.7	17.0–46.0
Po_{2TC} (mm Hg)*	56.0 ± 25.6	50.4	13.8–129.7	23.9–85.3	39.6 ± 16.9	33.4	17.9–80.6	19.1–63.5
Bicarbonate concentration (mmol/L) *	30.3 ± 6.9	31.3	17.4–43.6	21.5–39.4	25.3 ± 8.5	25.7	11.7–42.8	18.1–35.9
Sodium concentration (mmol/L)	157.6 ± 5.9	157.9	146.3–166.7	149.7–165.9	161.7 ± 12.2	160.8	138.5–190.9	147.5–178.2
Potassium concentration (mmol/L)*	3.97 ± 0.91	3.90	2.61–6.33	3.01–5.31	5.44 ± 1.56	5.03	3.03–9.38	3.71–7.59
Chloride concentration (mmol/L)	117.2 ± 5.4	116.5	107.1–125.3	110.3–123.9	121.0 ± 8.4	121.7	105.3–140.0	109.0–130.6
iCa_{cor} concentration (mmol/L)	0.72 ± 0.14	0.70	0.52–1.09	0.56–0.90	0.76 ± 0.16	0.73	0.36–1.27	0.59–0.95
iMg_{cor} concentration (mmol/L)	0.84 ± 0.17	0.85	0.61–1.27	0.64–1.04	1.01 ± 0.40	0.94	0.42–2.09	0.63–1.51
Glucose concentration (mg/dL)	121 ± 90	110	17–381	35–242	127 ± 85	107	22–364	33–248
Lactate concentration (mmol/L)	9.0 ± 5.2	8.0	2.3–22.5	3.9–16.5	10.0 ± 4.5	9.7	2.4–25.2	5.8–14.9
BUN concentration (mg/dL)	15 ± 6	15	5–30	8–23	17 ± 9	17	7–42	9–31
Osmolality (mOsm)	313.7 ± 13.3	312.9	294.1–340.9	295.7–336.7	322.5 ± 24.2	318.5	274.9–370.9	293.9–360.1
Anion gap (mmol/L)	12.89 ± 7.30	12.58	1.80–30.30	4.95–21.96	16.38 ± 9.54	17.26	–0.10 to 32.90	3.03–28.41

*Values are significantly ($P < 0.05$) different between survivors and nonsurvivors.

Table 3—Percentage change in selected clinicopathologic variables determined in blood samples of the survivor and nonsurvivor cold-stunned Kemp's ridley turtles described in Tables 1 and 2 between day 1 and the day of death (nonsurvivors) or corresponding day of hospitalization (day 2 or 3; survivors).

Variable	Survivor group (n = 32)				Nonsurvivor group (n = 32)			
	Mean ± SD	Median	Range	10th–90th percentile range	Mean ± SD	Median	Range	10th–90th percentile range
Hct	–4.7 ± 20.4	–4.3	–44.7 to 37.2	–27.3 to 20.2	–4.7 ± 29.8	0.9	–100.0 to 15.4	–14.9 to 13.4
pH_{TC}*	1.8 ± 3.9	1.0	–2.2 to 15.1	–1.6 to 5.1	–1.6 ± 4.3	–3.1	–7.1 to 8.2	–6.7 to 3.7
Pco_{2TC}	0 ± 31.3	–0.2	–61.4 to 61.0	–33.8 to 40.6	37.3 ± 60.7	40.7	–64.1 to 180.2	–37.9 to 91.8
Po_{2TC}	50.4 ± 88.9	25.2	–26.8 to 335.0	–17.1 to 134.1	12.1 ± 59.1	3.5	–82.7 to 168.0	–56.9 to 75.1
Bicarbonate concentration*	2.2 ± 20.8	0.2	–47.4 to 44.1	–15.7 to 28.0	–12.6 ± 30.6	–18.0	–62.4 to 84.3	–44.4 to 9.2
Sodium concentration	–0.8 ± 2.1	–1.2	–4.9 to 3.1	–3.3 to 1.8	0.3 ± 3.6	–0.6	–3.5 to 12.0	–2.8 to 3.4
Potassium concentration*	–12.3 ± 18.3	–14.4	–51.7 to 14.8	–34.2 to 9.4	37.6 ± 46.3	44.3	–55.7 to 122.7	–28.2 to 80.9
Chloride concentration	0.3 ± 2.5	0.9	–4.7 to 4.1	–2.9 to 2.7	1.2 ± 2.8	0.8	–5.2 to 4.6	–2.0 to 4.1
iCa_{cor} concentration*	12.3 ± 37.1	6.3	–13.6 to 159.7	–7.9 to 18.6	15.1 ± 13.9	14.2	–7.8 to 43.2	–2.7 to 32.5
iMg_{cor} concentration	9.5 ± 56.6	–7.3	–27.9 to 166.4	–23.2 to 51.9	5.7 ± 20.2	1.4	–23.1 to 60.6	–15.7 to 25.5
Glucose concentration*	65.0 ± 93.0	45.6	–33.8 to 343.8	–12.2 to 178.4	–13.6 ± 34.4	–18.3	–71.2 to 45.7	–59.2 to 25.3
Lactate concentration*	–34.8 ± 64.7	–47.1	–87.5 to 202.6	–80.2 to 1.3	70.5 ± 98.3	45.0	–63.2 to 233.3	–42.3 to 222.1
BUN concentration	9.9 ± 15.9	9.2	–31.6 to 47.4	0 to 26.7	17.4 ± 33.9	7.1	–20 to 115.6	–5.8 to 51.8
Osmolality	–0.1 ± 2.2	0.1	–4.2 to 3.1	–3.0 to 2.6	0.2 ± 3.9	–0.9	–5.2 to 11.5	–3.7 to 3.7
Anion gap	12.6 ± 68.9	–16.1	–42.8 to 223.2	–37.6 to 53.4	46.4 ± 136.1	24.2	–91.9 to 501.8	–85.7 to 97.8

*Values are significantly ($P < 0.05$) different between survivors and nonsurvivors.

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$P_{O_{2TC}}$ ($P = 0.004$), and bicarbonate concentration ($P = 0.017$) were significantly lower on day 1 in nonsurvivors than in survivors (**Table 2**). The $P_{CO_{2TC}}$ ($P = 0.006$) and potassium ($P < 0.001$) concentrations were significantly greater on day 1 in nonsurvivors than in survivors.

Compared with blood analysis findings in survivors, there were significantly greater percentage changes (from initial values to values at day 2 or 3) for iCa_{cor} ($P = 0.032$), potassium ($P < 0.001$), and lactate ($P = 0.002$) concentrations, and there were significantly lower percentage changes for pH_{TC} ($P = 0.005$) and glucose ($P = 0.001$) and bicarbonate ($P = 0.007$) concentrations in nonsurvivors (**Table 3**). Assumptions that data for percentage change in each variable were normally distributed and equal in variance were met for the percentage changes in Hct and osmolality; data for all other variables did not meet these assumptions.

Discussion

Physiologic responses of sea turtles to various natural and experimental stressors, including exposure to cold, have been described.[9–12,15–17,25–27] Several studies[9–12] have investigated hematologic, blood biochemical, and plasma biochemical values of cold-stunned Kemp's ridley turtles, one of which provided blood gas, pH, and blood biochemical data for turtles that were successfully rehabilitated after cold-stunning events.[9] However, to the authors' knowledge, the present study is the first in which blood gas and pH values for cold-stunned turtles that subsequently died were assessed and in which those values were compared with values for cold-stunned turtles that survived.

On day 1, nonsurvivors had a significantly lower pH_{TC}, $P_{O_{2TC}}$, and blood bicarbonate concentration and a significantly higher $P_{CO_{2TC}}$ value than did survivors. This indicated that many cold-stunned turtles were affected by hypoxemia and a mixed (respiratory and metabolic) acidosis. The mean pH_{TC} in blood samples collected on day 1 from survivors was 7.53 in the present study and 7.65 in another study.[9] Homeostatic mechanisms in healthy vertebrates (including sea turtles) maintain relatively alkaline blood pH, and as body temperature decreases, the pH of blood increases.[17,27] In the present study, the mean blood pH_{TC} in the nonsurvivors on day 1 was 7.35, which was unexpectedly low for the mean cloacal temperature (11.6°C [52.9°F]) of these turtles and indicated acidosis. In addition, turtles that died on day 2 or 3 of hospitalization had a lower blood pH_{TC} value and bicarbonate concentration on the day of death, compared with values on day 1. This indicated that acidosis was exacerbated prior to death. In contrast, the pH_{TC} value and bicarbonate concentration remained stable or increased in survivors during the first 2 or 3 days of hospitalization.

In contrast to pH, the P_{CO_2} in the blood of turtles decreases as body temperature decreases.[17,27] Thus, a P_{CO_2} value that is within the reference range in a turtle that has a cloacal temperature of 25°C (77°F) is considered to be high in a turtle that has a cloacal temperature of 10°C. For example, the mean $P_{CO_{2TC}}$ in the blood of rehabilitated Kemp's ridley turtles is 30.4 mm Hg in those that have a cloacal temperature of 24.5°C (76.1°F) and

is 20.7 mm Hg in those that have a cloacal temperature of 12.3°C (54.1°F).[9] Similarly, the median $P_{CO_{2TC}}$ in the blood of survivors on day 1 in the present study was 22.4 mm Hg (mean cloacal temperature, 12.3°C). Thus, the median $P_{CO_{2TC}}$ value (35.4 mm Hg) in nonsurvivors having a mean cloacal temperature of 11.6°C on day 1 in the present study was more than 50% greater than the median P_{CO_2} value that would be expected in a healthy turtle at that temperature (ie, this P_{CO_2} value would be considered only mildly increased in a turtle that had a cloacal temperature of 25°C). These findings highlight the importance of temperature correction for pH and blood gas values of ectotherms.

Although there was no significant ($P = 0.056$) difference in mean percentage change of $P_{CO_{2TC}}$ in survivors versus nonsurvivors in the present study, survivors generally had a P_{CO_2} value on day 2 or 3 that was lower than or equal to their P_{CO_2} value on day 1, and nonsurvivors generally had a P_{CO_2} value on the day of death that was higher than their P_{CO_2} value on day 1. Interpretation of these data suggests that an increase in an already high P_{CO_2} value in a cold-stunned Kemp's ridley turtle that has received medical treatment indicates a poor prognosis. Although standard treatments for cold-stunned turtles are intended to improve the patient's ventilation, tissue perfusion, and buffering capacity, the present study has provided data that may be useful for improvement of these treatments.

As has been found in other studies,[10,12] many cold-stunned turtles in the present study had hyperkalemia, and blood potassium concentrations on day 1 were higher in nonsurvivors than in survivors. Potassium concentrations increased over time in nonsurvivors but remained stable or decreased over time in survivors. The cause of hyperkalemia in cold-stunned turtles is unclear, but it is likely multifactorial; possible causes include impaired renal function, dehydration, and a compensatory response to acidosis.[10,27] Other authors have reported[27] that the plasma potassium concentration in loggerhead turtles (*Caretta caretta*) increases as the pH of blood decreases in response to a decrease in environmental temperature, and they suggested that this is caused by a shift of hydrogen ions into cells and a shift of potassium ions out of cells in an attempt to maintain physiologically normal pH. It is reasonable to suggest that a similar situation may exist in cold-stunned Kemp's ridley turtles. Concurrent acidosis ($pH_{TC} < 7.5$) and hyperkalemia (blood potassium concentration > 4.0 mmol/L) were detected on day 1 in 14 nonsurvivors and 7 survivors in the present study (data not shown). Severe hyperkalemia is associated with bradyarrhythmias and death in many species.[28,29] Consistent with previous findings[10] in cold-stunned Kemp's ridley turtles, severe hyperkalemia was associated with a poor prognosis in the present study, and all 8 turtles with a blood potassium concentration > 6.4 mmol/L died within 72 hours after admission to the hospital. Of the Kemp's ridley turtles evaluated in another study,[10] only 2 of the 142 turtles that survived cold stunning had a plasma potassium concentration > 6.4 mmol/L in the initial blood samples (unpublished data).

It has been suggested[8] that gradual warming (eg, 2° to 3°C [3.6° to 5.4°F] increase/d for 5 to 7 days) is

more successful than rapid warming (eg, 15°C [27°F] increase over 1 day) for successful rehabilitation (survival) of cold-stunned turtles. Although the reason for this clinical observation has not been experimentally determined, it is possible that rapid warming of cold-stunned turtles exacerbates metabolic and respiratory derangements (ie, a further decrease in blood pH and a further increase in P_{CO_2} and blood potassium concentration) that may cause death as a result of severe acidosis or hyperkalemia. However, if a patient is gradually warmed over several days and medically treated to correct metabolic and respiratory derangements, the homeostatic response to the derangements may be more effective. As such, we continue to recommend gradual warming of cold-stunned turtles, especially those that are severely acidotic, hypercapnic, or hyperkalemic.

Investigators in another study[10] found that sodium and chloride concentrations are higher in plasma of nonsurviving versus surviving cold-stunned turtles, but those results were not supported by the present study's findings. In addition, there was no significant difference between survivors and nonsurvivors with regard to the mean percentage change of sodium or chloride concentrations in blood samples during the first 2 or 3 days of hospitalization in the present study. The discrepancy in findings between the other study[10] and the present study may be explained by the differing methods of case selection between the 2 studies. In the other study,[10] data that were obtained up to the first 12 days of hospitalization were used as initial values for comparisons of hematologic and plasma biochemical variables over time, whereas in the present study, comparisons were made only for values that were obtained up to the first 3 days of hospitalization. Thus, analysis of results in the other study[10] was more likely than analysis of results in the present study to reveal a change in the turtles' clinical status and response to treatment. Although blood sodium and chloride concentrations on day 1 were not significantly different between nonsurvivors and survivors in the present study, all turtles with sodium concentrations > 167 mmol/L (n = 10 turtles) or chloride concentrations > 125.3 mmol/L (7) died within 2 days after admission to the hospital. These findings suggest that some cold-stunned turtles were affected by dehydration and possibly salt gland dysfunction and that turtles with severe derangement of electrolyte concentrations may have a poor prognosis, as has been reported.[10]

In the present study, mean concentrations of iCa_{cor} in blood samples on day 1 were not significantly different between survivors and nonsurvivors and were similar to previously reported initial iCa_{cor} concentrations for cold-stunned Kemp's ridley turtles.[9] However, mean concentrations of iCa_{cor} in blood samples on day 1 in the present study were lower than values reported for convalescent cold-stunned Kemp's ridley turtles[9] and for other reptile species.[30–35] The pathophysiologic cause of hypocalcemia in cold-stunned sea turtles is unknown. It is possible that transient hypocalcemia may be caused by a homeostatic response to elevated circulating concentrations of other divalent cations (eg, magnesium). A reduction in plasma iCa concentration in association with hypermagnesemia in humans has

been reported and may be attributable to magnesium-induced inhibition of parathyroid hormone secretion.[36] In the present study, the mean concentration of iMg_{cor} in blood samples obtained on day 1 was elevated, compared with the iMg_{cor} concentration reported for healthy Kemp's ridley turtles,[10] and was similar to blood iMg_{cor} concentrations in cold-stunned Kemp's ridley turtles reported in another study.[9] The causes of hypermagnesemia in stranded sea turtles are unknown, but likely include ingestion or aspiration of seawater and impaired renal function.[8] It is interesting that concentrations of iCa_{cor} in blood samples increased over the first 2 or 3 days of hospitalization to a significantly greater degree in turtles that died, compared with results for turtles that survived. Possible explanations for this finding include impaired renal function, dehydration, loss of cation homeostasis, lactic acidosis, or more aggressive administration of calcium in nonsurvivors than in survivors. Increased plasma calcium and magnesium concentrations have been detected in freshwater turtles during experimentally induced anoxia and lactic acidosis.[37] It is thought that the plasma concentrations of these ions increase as calcium and magnesium carbonates (which serve as buffers during lactic acidosis) are released from bone.[37] Similar mechanisms may be involved in Kemp's ridley turtles. Additional investigations of the clinical importance and appropriate medical management of iCa and iMg concentration derangements in sea turtles are warranted.

We detected substantial variation in the concentration of glucose in the blood of both survivors and nonsurvivors in the present study; some turtles had hypoglycemia, and some had hyperglycemia. Authors of other studies[10–12] have described this same finding in cold-stunned sea turtles. Although speculative, possible causes of hypoglycemia in these turtles include anorexia, exhaustion, and sepsis. Possible causes of hyperglycemia in reptiles include physiologic responses to stress, overcompensation by gluconeogenic mechanisms, disease of the liver or pancreas, or administration of dextrose.[38] Hyperglycemia of idiopathic origin has been described in reports of debilitated loggerhead sea turtles.[39,40]

Prior to completion of this study, it was the authors' clinical impression that nonsurviving cold-stunned Kemp's ridley turtles initially had higher circulating lactate concentrations than did their surviving counterparts. However, there was no significant difference in blood lactate concentration in day 1 samples of survivors versus those of nonsurvivors in the present study. The mean lactate concentration in blood samples collected from survivors on day 1 in this study was higher than that determined in cold-stunned Kemp's ridley turtles that survived in another study[9] and was higher than concentrations determined in sea turtles exposed to moderate stressors (eg, pound net capture and general anesthesia).[15,16,41] However, the mean lactate concentration in blood samples collected on day 1 in the present study was similar to concentrations detected in blood samples collected from sea turtles exposed to more severe stressors (eg, trawl net capture, experimental forced submergence, or long-duration voluntary dives).[15,25,26,42–44] Blood lactate concentration increased by nearly 50% between day 1 and the day of death in nonsurvivors and decreased by approximately 50% in survivors over the corresponding

period in the present study. Similar changes in blood lactate concentration in dogs that survived or died during hospitalization for gastric dilatation–volvulus syndrome have been reported.[14] Data from the present study suggest that cold-stunned Kemp's ridley turtles with a blood lactate concentration that increases during the first 3 days of hospitalization (despite treatment) have a poor prognosis.

Mean BUN concentration in turtles in the present study was lower than concentrations typically found in healthy Kemp's ridley turtles,[9,10] which is consistent with other published data for debilitated sea turtles.[9–12] The pathophysiologic cause of this finding is unknown. However, impaired hepatic function, anorexia, or other metabolic disturbances have been proposed as contributing factors to low BUN concentrations in cold-stunned turtles.[10] Specific clinicopathologic markers of liver disease in sea turtles have not been described, to the authors' knowledge. Results of 1 study[45] indicate that measurement of circulating bile acids concentration may be useful in freshwater turtles. Therefore, measurement of circulating bile acids concentration should be considered in future studies of debilitated sea turtles. The mean Hct on day 1 was higher in cold-stunned turtles in the present study than in healthy Kemp's ridley turtles.[10] This finding may be attributable to dehydration. A wide range of Hct values was detected in blood samples of turtles in the present study (severe hemoconcentration [Hct, 71%] to moderate anemia [Hct, 15%]). Anion gap values were somewhat higher than those previously reported for Kemp's ridley turtles.[9,25] The reason for the discrepancy between findings of the present study and other studies is unclear, but it may be related to the high sodium concentration and low bicarbonate concentration in blood that were found in many turtles in the present study. The mean osmolality of blood was similar to previously published values for cold-stunned Kemp's ridley turtles.[9] Because of a low BUN concentration, the osmolality of blood in cold-stunned sea turtles before treatment and rehabilitation may be lower than that in convalescent turtles.[9]

The methods that were used for blood collection and processing and for correction of results for temperature and pH in the present study are consistent with the methods used in other studies[9,15,16] on the physiologic state of sea turtles, including Kemp's ridley turtles. Although the equations that were used in the present study to correct results of blood analyses for temperature and pH have not been validated in Kemp's ridley turtles, they are believed to provide data that are more relevant to the physiologic status of sea turtles than are data that have not been corrected for temperature and pH (ie, raw data from analyses performed at 37°C).[15] In the present study, the values of pH in blood samples were corrected for patient temperature in accordance with the findings of other researchers that the pH of Kemp's ridley turtle blood increases approximately 0.015 U for every 1°C (1.8°F) decrease in environmental temperature within a specific range of temperatures (ie, actual pH value of blood > pH value of blood measured at 37°C).[17] Because of this relationship between measured pH and temperature, the iCa and iMg concentrations in blood analyzed at 37°C are higher than the actual iCa and iMg concentrations

in blood at lower temperatures.[21,22] Therefore, the pH_{TC} was used to calculate iCa_{cor} and iMg_{cor} concentrations in the present study. Similarly, blood gas values were corrected for patient temperature in accordance with other researchers' findings that the actual values of P_{CO_2} and P_{O_2} in Kemp's ridley turtle blood at cloacal or environmental temperatures < 37°C are less than the values of these variables when they are measured at 37°C.[15,16,18–20]

Analysis of results of the present study indicated that cold-stunned Kemp's ridley turtles were often affected by metabolic and respiratory derangements and that the more severe derangements were associated with death. Thorough clinical assessment of cold-stunned Kemp's ridley turtles should include serial evaluation of venous blood pH, blood gas, and selected hematologic and blood biochemical variables. Such evaluations may provide useful clinical and prognostic information for clinicians and rehabilitators.

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AQUATIC ANIMALS

Appendix

Equations used in calculation of metabolic and respiratory variables for cold-stunned Kemp's ridley turtles.

Calculation category	Equation	References
Correction for temperature	$pH_{TC} = (0.015 \times \Delta T) + pH$	17
	$P_{CO_{2TC}} = P_{CO_2} \times 10^{(-0.019 \times \Delta T)}$	15, 16, 18, and 19
	$P_{O_{2TC}} = P_{O_2} \times 10^{(-0.0058 \times \Delta T)}$	15, 16, 19, and 20
Correction for pH	iCa_{cor} concentration = iCa concentration $\times (1 + [0.53 \times \{pH - pH_{TC}\}])$	21
	iMg_{cor} concentration = iMg concentration $\times 10^{(-0.2 \times [pH_{TC} - pH])}$	22
Henderson-Hasselbalch	HCO_{3cor} concentration = $\alpha CO_2 \times P_{CO_{2TC}} \times 10^{(pH_{TC} - pK)}$	23
Additional calculations	Anion gap = (Na^+ concentration + K^+ concentration) – (Cl^- concentration + HCO_3^- concentration)	13
	Osmolality = ($1.86 \times Na^+$ concentration) + (Glu concentration/18) + (BUN concentration/2.8) + 9	24

$\Delta T = 37$ – patient temperature (in °C), where patient temperature is the temperature (cloacal [day 1] or environmental [day 2 or 3]) of the turtle. Glu = Glucose.