J Comp Physiol B (2007) 177:623–630
DOI 10.1007/s00360-007-0160-9

ORIGINAL PAPER

# Metabolic and respiratory status of cold-stunned Kemp's ridley sea turtles (Lepidochelys kempii)

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Received: 14 January 2007 / Revised: 3 March 2007 / Accepted: 12 March 2007 / Published online: 13 April 2007


**Abstract** ''Cold-stunning'' of sea turtles has been reported as a naturally occurring stressor for many years; however, the physiologic status of cold-stunned turtles has only been partially described. This study investigated initial and convalescent venous blood gas, acid-base, and critical plasma biochemical data for 26 naturally cold-stunned Kemp's ridley sea turtles (Lepidochelys kempii) from Cape Cod, MA, USA. Samples were analyzed for pH, $pCO_2$, $pO_2$, bicarbonate, plasma osmolality, sodium, potassium, chloride, ionized calcium, ionized magnesium, glucose, lactate, and blood urea nitrogen using a clinical point-of-care analyzer. Data were corrected for the patient's body temperature using both species-specific and more general correction methods. In general, venous blood gas, acid-base, and plasma biochemical data obtained for surviving cold-stunned Kemp's ridley sea turtles were consistent with previously documented data for sea turtles exposed to a wide range of temperatures and physiologic stressors. Data indicated that turtles were initially affected by metabolic and respiratory acidosis. Initial pH-corrected ionized calcium concentrations were lower than convalescent concentrations, and initial pH-corrected ionized magnesium concentrations were higher than convalescent concentrations.

**Keywords** Kemp's ridley sea turtle · Lepidochelys kempii · Cold-stunning · Acid-base status · Blood gas

Communicated by H.V. Carey.

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## Introduction

Kemp's ridley turtle (Lepidochelys kempii) is the smallest and rarest of the sea turtles (Márquez et al. 2005). While adult Kemp's ridley sea turtles reside primarily in the Gulf of Mexico, juveniles may migrate seasonally to foraging grounds along the northeast coast of the United States (Lazell 1980; Morreale and Standora 2005). Juveniles that do not leave these northern waters in autumn are exposed to decreasing water-temperatures and may become ''cold-stunned'' (Burke et al. 1991; Morreale et al. 1992; Gerle et al. 2000; Still et al. 2002, 2005; Dodge et al. in press). Cold-stunned sea turtles cease swimming, float on the surface of the water, and often become stranded (Schwartz 1978). In Massachusetts, cold-stunned Kemp's ridley sea turtles are commonly found stranded on beaches during the months of October through December, when water temperatures drop below 10°C (Still et al. 2002, 2005; Dodge et al. in press). Unfortunately, 35-85% of cold-stunned sea turtles are found dead (Gerle et al. 2000; Bentivegnal et al. 2000; Turnbull et al. 2000). Live turtles are collected by a network of volunteers and transported to the New England Aquarium, Boston, MA, USA for rehabilitation (Wyneken et al. 2006).

Several studies have documented temperature-dependent physiologic responses of sea turtles (Kraus and Jackson 1980; Lutz and Dunbar-Cooper 1987; Lutz et al. 1989; Moon et al. 1997). The effect of a variety of stressors, including forced submergence, experimentally induced hibernation, general anesthesia, and trawling have been evaluated in several sea turtle species (Stabenau et al. 1991; Moon and Stabenau 1996; Moon et al. 1997; Chittick et al. 2002; Harms et al. 2003). Several authors have reported hematologic and selected biochemical data for living and deceased cold-stunned sea turtles; however, the

blood gas and acid-base status of cold-stunned sea turtles has not been thoroughly described (Carminati et al. 1994; Turnbull et al. 2000; Smith et al. 2000). This study presents initial and convalescent venous blood gas, acid-base, and critical plasma biochemical data for cold-stunned, juvenile Kemp's ridley sea turtles that were successfully rehabilitated and released to the wild.

## Materials and methods

Cold-stunned Kemp's ridley sea turtles were admitted to the New England Aquarium Rescue and Rehabilitation Department in October and November, 2005. Daily average water temperature at 1 m depth in Massachusetts Bay during the stranding period was determined from the Gulf of Maine Ocean Observing System, buoy A01. Details of the medical management of cold-stunned sea turtles at New England Aquarium have been previously published (Wyneken et al. 2006). On the day of admission, each turtle was weighed, straight carapace length (SCL) was recorded, and the body temperature of the turtle (*T*p) was recorded using a digital thermometer inserted at least 10 cm into the cloaca. A venous blood sample was anaerobically collected from the cervical sinus of each turtle using a 1 ml heparinized syringe, and immediately analyzed for pH, $pCO_2$, $pO_2$, bicarbonate ($HCO_3$), plasma osmolality (Osm), sodium (Na), potassium (K), chloride (Cl), ionized Ca (iCa), ionized Mg (iMg), glucose (Glu), lactate (Lac), and blood urea nitrogen (BUN) using a clinical point-of-care analyzer (Critical Care Express, NOVA Biomedical, Waltham, MA, USA). Blood analysis was repeated periodically during the rehabilitation process. For this study, the ''convalescent'' sample was obtained from turtles that met all of the following criteria: swimming in water at 25°C for at least 10 days; discontinued from parenteral fluid therapy for at least 7 days; and eating voluntarily every day for at least 7 days. Due to the very small number of mortalities during the study period ($n$ = 2), only data from turtles that were successfully rehabilitated and returned to the wild are presented.

Because the blood analyzer warms samples to 37°C prior to analysis, the following parameters were corrected (equation given) for the patient's body temperature yielding a temperature-corrected (TC) value. In all cases, $\Delta T = 37 - T\text{p}$:

$$\text{pH}_{\text{TC}} = 0.015(\Delta T) + \text{pH} \quad \text{(Moon et al. 1997)}.$$

$$\text{pCO}_{2\text{TC}} = \text{pCO}_2(10^{-0.019\Delta T})$$

(Nunn et al. 1965; Ashwood et al. 1983; Chittick et al. 2002; Harms et al. 2003).

$$\text{pO}_{2\text{TC}} = \text{pO}_2(10^{-0.0058\Delta T})$$

(Roughton and Severinghaus 1973; Ashwood et al. 1983; Chittick et al. 2002; Harms et al. 2003).

$HCO_{3TC}$ was calculated using the Henderson–Hasselbalch equation, $pH_{TC}$, $pCO_{2TC}$, and values of $\alpha CO_2$ and pK calculated for each patient using species-specific equations for Kemp's ridley sea turtles (Stabenau and Heming 1993). Values for $\alpha CO_2$ ranged from 0.041 to 0.069. Values for pK ranged from 6.15 to 6.27.

Due to the effect of pH on iCa and iMg, pH-corrected (cor) values for these parameters were also determined.

$$\text{iCa}_{\text{cor}} = \text{iCa}(1 + 0.53(\text{pH} - \text{pH}_{\text{TC}})) \text{ (Fogh - Andersen 1981)}.$$

$$\text{iMg}_{\text{cor}} = \text{iMg}(10^{-0.2(\text{pH}_{\text{TC}} - \text{pH})}) \text{ (Ising et al. 1995)}.$$

Anion gap and osmolality were calculated by the following formulas:

$$\text{Anion gap (AGap)} = (\text{Na} + \text{K}) - (\text{Cl} + \text{HCO}_{3\text{TC}}) \quad \text{(Wellman et al. 2006)}.$$

$$\text{Osmolality (Osm)} = 1.86(\text{Na}) + (\text{Glu}) + (\text{BUN}) + 9 \text{ (Tietz 1986)}.$$

A total of 14 turtles were assessed with both initial and convalescent samples (paired data set). However, at each sample interval, a total of 20 turtles were assessed (full data set). Six turtles that had initial data collected did not have convalescent data collected as they were transferred to other institutions for further care prior to meeting the definition of convalescence. Six turtles that had convalescent data did not have initial data as the point-of-care analyzer was not available during the first 2 days of the stranding period.

Temperature-corrected and pH-corrected data from the paired data set were statistically compared by a paired $t$-test for normally distributed data, or a Wilcox sign rank test (WSR) for data that were not normally distributed. Because 14 blood parameters were assessed, a protected $P = 0.0036$ was used for each statistical test to determine significance. To examine if the trends observed within the full data set were similar to those of the paired data set, the full data were analyzed with a $t$-test for normally distributed data, or a Mann–Whitney rank sum test (MWRS) for data that were not normally distributed, or had unequal variance. Power for the parametric tests was determined using $1 - \alpha$. Where the results for the statistical tests using the paired or full data sets were similar, only the results of the full data

analysis are provided. Discrepancies in tests using the two data sets are noted. Finally, to assess overall variability in initial versus convalescent values, the coefficient of variation (CV = standard deviation/mean) was calculated for each parameter, and this metadata was tested for statistical similarity with a WSR. The tables presented within utilize the data from all turtles, as they provide a better representation of the clinical range of values observed in all of the surviving turtles.

## Results and discussion

The turtles in this study were found stranded on the north side of Cape Cod between October 29 and November 29, 2005 (Fig. 1). The turtles stranded when water temperatures were between 8 and 11°C, and had experienced a 5°C drop in water temperature during the previous month (Fig. 1). Initial and convalescent size, temperature, and rehabilitation status of the turtles are presented in Table 1. Initial and convalescent TC and pH-corrected blood gas, acid-base, and plasma biochemical data are presented in Table 2.

The mean initial $pH_{TC}$ of turtles in this study was very similar to both in vivo and in vitro values previously reported for sea turtles at 10–20°C under conditions of experimental cooling (Kraus and Jackson 1980; Lutz et al. 1989; Stabenau and Heming 1994; Moon et al. 1997). Mean convalescent $pH_{TC}$ was ~0.1 to 0.2 units higher than generally reported for sea turtles at 25°C (Kraus and Jackson 1980; Lutz et al. 1989; Stabenau et al. 1991; Stabenau and Heming 1994; Moon and Stabenau 1996; Moon et al. 1997; Chittick et al. 2002). The lack of significant difference between initial and convalescent $pH_{TC}$ (MWRS, $T = 501.5$, $P > 0.01$) is unexpected, as the venous pH of sea turtles generally tends to decrease as body temperature increases (Kraus and Jackson 1980; Lutz et al. 1989; Stabenau and Heming 1994; Moon et al. 1997). If the convalescent $pH_{TC}$ at 25°C (7.61) is considered to be ''normal'' for relatively healthy juvenile Kemp's ridley turtles, then the calculated $pH_{TC}$ for such turtles at 12°C should be ~7.8. As discussed below, the relatively low initial $pH_{TC}$ of the study turtles (7.65) most likely reflects a mild metabolic and respiratory acidosis.

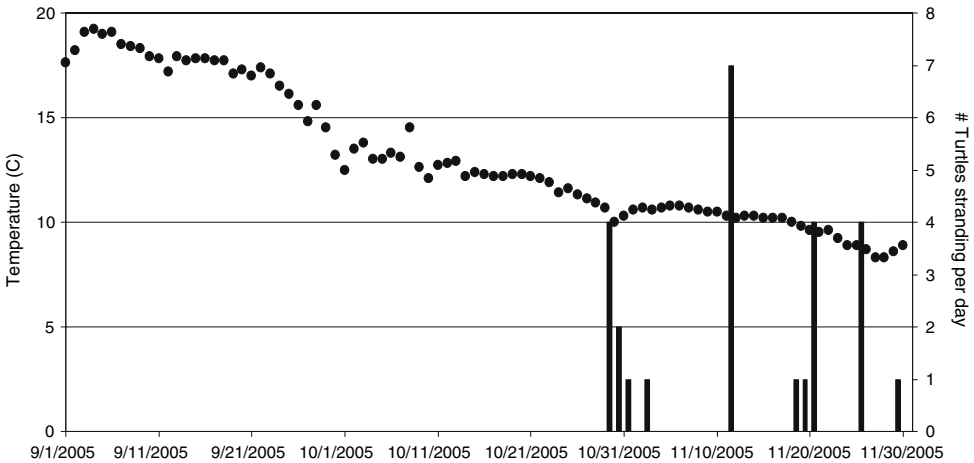


**Fig. 1** Daily average water temperature (*circles*) at 1 m depth in Massachusetts Bay (data from the Gulf of Maine Ocean Observing System, buoy A01, www.gomoos.org). *Bars* indicate the number of turtles stranding daily

**Table 1** Initial and convalescent size, temperature, and rehabilitation (rehab) status of cold-stunned Kemp's ridley turtles for which blood gas, acid-base, and plasma biochemical data were collected

| | Initial | | Convalescent | |
|---|---|---|---|---|
| | Mean | Range | Mean | Range |
| Weight (kg) $n = 20$ (all data) | 2.65 | 1.6–4.64 | 2.54 | 1.5–5 |
| Weight (kg) $n = 14$ (paired data) | 2.53 | 1.7–4.64 | 2.56 | 1.5–5 |
| SCL (cm) $n = 20$ | 26 | 22.2–34.5 | NR | |
| Temperature (C) $n = 20$ | 12.3 | 8.3–16.7 | 24.5 | 22.2–25 |
| Day of rehab $n = 20$ | 1 | NA | 28 | 18–73 |
| Days eating $n = 20$ | NA | | 19 | 8–67 |
| Days off fluid therapy $n = 20$ | NA | | 17.6 | 7–63 |
| Days at 25°C $n = 20$ | NA | | 24.8 | 11–71 |

*NA* not applicable, *NR* not recorded

**Table 2** Temperature and pH-corrected initial and convalescent blood gas, acid-base, and plasma biochemical values for cold-stunned Kemp's ridley sea turtles (mean, median, standard deviation, 10th and 90th percentiles)

| | Initial ($n$ = 20) | | Convalescent ($n$ = 20) | |
|---|---|---|---|---|
| | Mean (median) ±SD | 10th percentile–90th percentile | Mean (median) ± SD | 10th percentile–90th percentile |
| Na (mmol/l) | 153 (152) ± 5.4 | 146–158 | 150 (150) ± 3.1 | 146–153 |
| K (mmol/l) | 3.3 (3.3) ± 0.7 | 2.3–4.3 | 3.6 (3.6) ± 0.4 | 3.3–4.0 |
| Cl (mmol/l) | 118 (118) ± 4.6 | 112–123 | 115 (115) ± 3.4 | 111–119 |
| Glu (mmol/l) | 8.8 (7.6) ± 3.7 | 5.4–13 | 6.8 (6.9) ± 0.8 | 5.8–7.7 |
| Lac (mmol/l)[a] | 6.2 (5.3) ± 5.1 | 1.0–10.9 | 1.9 (1.5) ± 1.4 | 0.6–3.7 |
| $pCO_{2TC}$ (torr)[a] | 20.7 (18.6) ± 6.4 | 15.1–27.9 | 30.4 (30.9) ± 3.6 | 25.9–33.8 |
| $pO_{2TC}$ (torr) | 79.4 (80) ± 21.6 | 54.0–109.5 | 84.9 (82) ± 16.5 | 68.5–101 |
| $pH_{TC}$ | 7.65 (7.68) ± 0.16 | 7.59–7.77 | 7.61 (7.61) ± 0.05 | 7.55–7.67 |
| $HCO_3$ (mmol/l) | 34.1 (31.1) ± 9.4 | 23.4–46.6 | 36.6 (35.9) ± 5.4 | 31.1–44.5 |
| $iCa_{cor}$ (mmol/l)[a] | 0.67 (0.69) ± 0.12 | 0.49–0.81 | 0.92 (0.90) ± 0.07 | 0.85–1.02 |
| Agap (mmol/l) | 2.8 (1.6) ± 6.8 | –5.7–12.8 | 2.3 (3.2) ± 3.0 | –1.2–4.4 |
| $iMg_{cor}$(mmol/l)[a] | 0.85 (0.88) ± 0.23 | 0.59–1.08 | 0.62 (0.61) ± 0.14 | 0.44–0.77 |
| BUN (mmol/l)[ab] | 5.7 (5.3) ± 1.8 | 3.5–8.2 | 28 (28.5) ± 2.5 | 27.7–28.5 |
| Osm (mOsm)[ab] | 307 (307) ± 10 | 296–320 | 321 (323) ± 8 | 308–329 |

[a] Statistically significant differences between initial and convalescent data

[b] Mean and SD convalescent BUN and Osm underestimate the true values because BUN values exceeded the analytical range of the analyzer in 11 patients (see text)

Mean initial $pCO_{2TC}$ was significantly lower than convalescent values ($t$-test, $t = -5.81$, $P < 0.001$, power = 1.0) and was similar to that reported previously for Kemp's ridley and loggerhead sea turtles under experimental exposure to 10–15°C (Lutz and Dunbar Cooper 1987; Moon et al. 1997), and only slightly lower than that of green turtles under experimental cooling to 15°C (Kraus and Jackson 1980). Our data agree with a number of studies that have demonstrated that arterial and venous $pCO_2$ of turtles decreases with temperature (Kraus and Jackson 1980; Lutz et al. 1989; Moon et al. 1997). While it seems paradoxical that $pCO_2$ would be lower under conditions of bradypnea/apnea, similar observations have been made for Kemp's ridley sea turtles under simulated hibernation (Moon et al. 1997). We agree with Moon et al. (1997) that these observations may be partly explained by increased $CO_2$ solubility and decreased tissue production of $CO_2$ at lower temperatures. However, the initial $pCO_{2TC}$ in this study (20.7 Torr) was higher than that which would be predicted (17.2 Torr) assuming that the convalescent $pCO_{2TC}$ (30.4 Torr) represents a ''normal'' value for relatively healthy juvenile Kemp's ridley turtles. This relatively elevated initial $pCO_2$ is likely due to hypoventilation and decreased perfusion under conditions of hypothermia, and is likely partly responsible for the initial acidosis. Mean convalescent $pCO_{2TC}$ in this study is very similar to green and loggerhead sea turtles at 25°C (Kraus and Jackson 1980; Lutz et al. 1989; Chittick et al. 2002), but lower than that reported for Kemp's ridley sea turtles at 25°C (Stabenau et al. 1991; Moon et al. 1997).

Several previous studies have documented that blood $pO_2$ of sea turtles, like $pCO_2$, decreases with temperature (Kraus and Jackson 1980; Lutz et al. 1989; Moon et al. 1997). However, in this study there was no difference in mean initial versus convalescent $pO_{2TC}$ ($t$-test, $t = -0.91$, $P > 0.36$). $pO_{2TC}$ values were generally higher than those previously reported for sea turtles across a range of temperatures (Lutz et al. 1989; Stabenau et al. 1991; Moon and Stabenau 1996; Moon et al. 1997; Chittick et al. 2002; Harms et al. 2003). The reason for this relatively high $pO_2$ is unclear. While artifactual increases of $pO_2$ due to inappropriate sample handling (e.g., delay of sample processing, mixing of air with the sample, etc.) would tend to increase $pO_2$, we believe our methodology avoided such factors. It should be noted that the present $pO_{2TC}$ values are more similar to values reported in previous studies that used the same temperature correction formula as used in this study (Chittick et al. 2002; Harms et al. 2003), compared to directly measured $pO_2$ (Kraus and Jackson 1980; Lutz et al. 1989) and $pO_{2TC}$ calculated using an alternative correction method (Stabenau et al. 1991; Moon et al. 1997).

Mean initial and convalescent bicarbonate values were statistically equivalent (MWRS, $W = 307$, $P > 0.2$) and

were similar to those previously reported for Kemp's ridley and loggerhead sea turtles across a range of temperatures (Lutz et al. 1989; Stabenau et al. 1991; Chittick et al. 2002; Harms et al. 2003). The inverse relationship of bicarbonate concentration and temperature that was noted for loggerhead sea turtles (Lutz et al. 1989) was not seen in this study.

Initial lactate concentrations were higher than convalescent concentrations for the full data set, but not for the paired data set (MWRS, $W = 225$, $P < 0.003$; paired $t$-test, $t = 2.51$, $P < 0.03$, power = 0.54). This discrepancy may result from the use of a conservative $P$-value in determining significance for the paired data set. The mean initial lactate concentration is within the range reported for sea turtles exposed to moderate stressors such as pound net capture and general anesthesia (Moon and Stabenau 1996; Chittick et al. 2002; Harms et al. 2003). Mean initial lactate concentration was lower, however, than seen in sea turtles under more physiologically demanding stressors such as long voluntary dives, trawl net capture, or experimental forced submergence (Berkson 1966; Wood et al. 1984; Lutz and Bentley 1985; Stabenau et al. 1991; Harms et al. 2003). Since most cold-stunned Kemp's ridley sea turtles show severe bradypnea/apnea and bradycardia at the time of admission, elevated initial lactate concentrations are likely due to anaerobic metabolism caused by hypoventilation and poor perfusion, representing a metabolic acidosis. Moderately elevated lactate concentrations were seen in healthy loggerhead turtles under experimental exposure to water at 10°C (Lutz et al. 1989). Convalescent lactate concentrations in this study are similar to those of healthy sea turtles at 25°C (Berkson 1966; Hochachka et al. 1975; Wood et al. 1984; Lutz and Bentley 1985; Lutz et al. 1989; Stabenau et al. 1991).

Mean initial sodium, potassium and chloride values were not significantly different than respective convalescent values (Na and K, MWRS, $T > 401.5$, $P > 0.13$; Cl, $t$-test, $t = 2.89$, $P < 0.006$, power = 0.77). Initial glucose values did not differ from convalescent values (MWRS, $T = 213$, $P > 0.06$). While initial glucose values were often higher than convalescent values, several turtles were markedly hypoglycemic upon admission.

While anion gap values for sea turtles have not previously been reported, our values are similar to values calculated using the Kemp's ridley sea turtle data of Stabenau et al. (1991). There was no significant difference between initial and convalescent samples (MWRS, $W = 369$, $P > 0.6$). The utility of anion gap in defining the acid-base status of sea turtles requires further investigation.

Electrolyte and glucose values in this study were very similar to previously published values for healthy and cold-stunned Kemp's ridley sea turtles and loggerhead sea turtles (Lutz and Dunbar Cooper 1987; Stabenau et al. 1991; Carminati et al. 1994; Whitaker and Krum 1999; Turnbull et al. 2000; Chittick et al. 2002; Stamper et al. 2005). Turtles in this study did not show elevated electrolyte levels as previously reported for some cold-stunned sea turtles (George 1997). It is possible that this discrepancy reflects variability in the physiologic derangements in cold-stunned sea turtles. Turtles in this study survived rehabilitation, indicating that they may have been generally healthier than some turtles described in previous studies. While several studies have noted significant decreases in potassium at lower temperatures (Lutz and Dunbar-Cooper 1987; Lutz et al. 1989), this effect was not seen in this study. In previous studies, temperature-related potassium changes were believed to occur in response to temperature-related pH changes. Since turtles in this study did not show significant temperature-related pH differences, temperature-related variation of potassium levels would not be expected. Turtles in this study population also did not show the severe hyperkalemia that has been described in freshly dead, cold-stunned Kemp's ridley sea turtles, and Kemp's ridley sea turtles subjected to trawling (Stabenau et al. 1991; Carminati et al. 1994).

Initial BUN concentrations were significantly lower than convalescent concentrations (MWRS, $T = 210$, $P < 0.001$). The mean convalescent BUN cited in this study underestimates the true mean, as 11 turtles had convalescent levels above the range of the analyzer (28.5 mmol/l). However, convalescent minimum BUN levels in this study were generally consistent with previously published values for healthy and rehabilitated Kemp's ridley sea turtles and loggerhead sea turtles (Carminati et al. 1994; Whitaker and Krum 1999; Turnbull et al. 2000; Stamper et al. 2005). Initial BUN levels in this study were also consistent with those that have been previously documented in ill sea turtles, including cold-stunned Kemp's ridley turtles (Carminati et al. 1994; Turnbull et al. 2000; Chittick et al. 2002). Decreased BUN levels were also noted in healthy loggerhead sea turtles experimentally exposed to low temperatures (Lutz et al. 1989). This reduction in BUN may represent decreased urea production due to decreased protein intake or decreased hepatic function. Dilution of plasma samples should be considered to allow more precise determination of convalescent BUN using this point-of-care analyzer.

Initial calculated osmolality was significantly lower than convalescent values ($t$-test, $t = -4.27$, $P < 0.001$, power = 0.99). This was expected in light of the significant difference in initial versus convalescent BUN. Mean initial calculated osmolality was similar to that calculated using previously published data on cold-stunned Kemp's ridley sea turtles, and similar to that reported for loggerhead sea turtles at 18°C (Lutz and Dunbar-Cooper 1987; Carminati

et al. 1994; Turnbull et al. 2000). As discussed for BUN, the convalescent calculated osmolality values underestimate the true values, but were generally consistent with previously published data for healthy and rehabilitated Kemp's ridley and loggerhead sea turtles at 24–27°C (Lutz and Dunbar-Cooper 1987; Lutz et al. 1989; Carminati et al. 1994; Turnbull et al. 2000).

Initial $iCa_{cor}$ concentrations were lower than convalescent concentrations (MWRS, $T = 219$, $P < 0.001$). Initial and convalescent $iCa_{cor}$ concentrations were lower than those reported for several other reptile species (Silver and Jackson 1986; Rooney et al. 1999; Sleeman and Gaynor 2000; Dennis et al. 2001). Several authors documented that Kemp's ridley and loggerhead sea turtles have lower total plasma calcium concentrations than many other vertebrates (Lutz and Dunbar Cooper 1987; Lutz et al. 1989; Carminati et al. 1994; Whitaker and Krum 1999; Turnbull et al. 2000; Chittick et al. 2002). However, iCa does not generally correlate well with total plasma calcium, making it difficult to conclude that the generally low iCa values reflect low total Ca (Ladenson et al. 1978, 1979; Schenck and Chew 2005). Additional studies that evaluate iCa levels and total Ca levels in larger numbers of healthy and ill sea turtles may help to better define normocalcemia.

This study provides the first measurements of iMg in a reptile. Initial $iMg_{cor}$ values were higher than convalescent values (MWRS, $T = 542$, $P < 0.001$), and were above the normal range of iMg in humans (Ising et al. 1995). Elevated total magnesium concentrations have previously been reported in ill loggerhead sea turtles; however, the etiology of the hypermagnesemia was unclear (Wyneken et al. 2006). Mean convalescent $iMg_{cor}$ in the turtles was at the upper end of the normal range of iMg in humans (Ising et al. 1995). As with iCa, additional iMg data on larger numbers of healthy sea turtles may help to better define normal and abnormal magnesium states.

For all blood parameters, there was a wider range of initial values compared to convalescent values. The CV averaged 15% lower at convalescence than at admission (WSR, $W = -105$, $P < 0.001$). This likely represents improved homeostasis upon convalescence.

Temperature and pH correction of blood gas and plasma biochemical results of ectotherms remains problematic when using clinical analyzers designed for mammals. As such, we have provided the raw data generated by the analyzer at 37°C (Table 3), as well as details of the calculations used to correct the results to the patients' body temperature. It was likely that the temperature and pH-corrected data were more representative of the true physiological status of the patient. For temperature correction of pH, and calculation of $HCO_3$, we have used formulas based on species-specific data for Kemp's ridley sea turtles. For temperature correction of $pCO_2$ and $pO_2$ we have used formulas derived mathematically from several earlier works (Nunn et al. 1965; Roughton and Severinghaus 1973; Ashwood et al. 1983). These formulas have been recently applied to temperature correction of loggerhead sea turtle blood gas values (Chittick et al. 2002; Harms et al. 2003). For pH correction of iCa and iMg, it was necessary to use formulas developed for humans. While these formulas may not be quantitatively precise for sea turtles, we feel that it is important to apply a pH correction, rather than only reporting values obtained at an artifactually low pH. Further work to validate methods of pH correction of iCa and iMg values of ectotherms is warranted.

The point-of-care analyzer used in this study calculates TC values for pH, $pCO_2$, and $pO_2$ using National Committee for Clinical Laboratory Standards formulas. Since we have chosen to correct for temperature using methods that differ from those of the analyzer, we have not reported the analyzer-corrected values. While the analyzer-corrected values for pH, $pCO_2$, and $pO_2$ are different than our calculated values, we agree with Chittick et al. (2002) and Harms et al. (2003) that the analyzer-corrected values for pH and $pCO_2$ are similar enough to our calculated values to be clinically useful. For example, the analyzer-corrected pH and $pCO_2$ values varied from our calculated values by no more than 0.2%. However, also in agreement with previous reports, we found that the analyzer-corrected values for $pO_2$ do not agree well with our calculated values, differing by up to 70%. This is likely due to the relatively reduced effect of temperature on oxyhemoglobin dissoci-

**Table 3** Initial and convalescent blood gas, pH, ionized calcium, and ionized magnesium values for cold-stunned Kemp's ridley sea turtles, generated at 37°C

| | Initial ($n = 20$) | | Convalescent ($n = 20$) | |
|---|---|---|---|---|
| | Mean (median) ± SD | 10th percentile–90th percentile | Mean (median) ± SD | 10th percentile–90th percentile |
| PH | 7.28 (7.29) ± 0.16 | 7.19–7.39 | 7.42 (7.43) ± 0.05 | 7.36–7.49 |
| $pCO_2$ (torr) | 63.1 (54.8) ± 22 | 41.2–88.7 | 52.5 (52.7) ± 6.5 | 44.8–58.6 |
| $pO_2$ (torr) | 111.2 (112.5) ± 31.6 | 73.1–155.8 | 100.29 (96.7) ± 19.49 | 81.0–119.4 |
| iCa (mmol/l) | 0.83 (0.86) ± 0.15 | 0.61–0.99 | 1.02 (1.01) ± 0.07 | 0.94–1.13 |
| iMg (mmol/l) | 1.0 (0.88) ± 0.28 | 0.59–1.08 | 0.67 (0.67) ± 0.16 | 0.48–0.84 |

ation in sea turtles (Giardina et al. 1992; Lutcavage and Lutz 1997; Chittick et al. 2002).

In summary, this study provides data on initial and convalescent venous blood gas, acid-base, and plasma biochemical status of successfully rehabilitated, cold-stunned Kemp's ridley sea turtles. These data offer insight into the physiological response of sea turtles to reduced body temperature under natural conditions. In general, the data were consistent with previous works that have experimentally evaluated the physiological response of sea turtles to a range of temperatures. It is important to note that this study reports values only for surviving turtles, thus it does not adequately describe the complete breadth of physiologic derangements that may be seen in cold-stunned sea turtles. Evaluation of these parameters in more severely affected, non-surviving cold-stunned cases is required to further define the metabolic causes of mortality in cold-stunned sea turtles. The results of this study indicate that cold-stunned juvenile Kemp's ridley sea turtles that survive rehabilitation may present initially with mild to moderate respiratory and metabolic acidosis, hypocalcemia, and hypermagnesemia. As such, evaluation of blood gas, acid-base, and plasma biochemical status is important in developing management plans for cold-stunned turtles.

**Acknowledgments** Diagnostic blood collection from sea turtles at the New England Aquarium is performed with authorization of the United States Fish and Wildlife Service and the National Marine Fisheries Service. We thank the staff and volunteers of the Rescue and Rehabilitation Department and Animal Health Department of the New England Aquarium, and the Massachusetts Audubon Wellfleet Sanctuary for turtle recovery and care. We thank Elizabeth Small for helpful insight on temperature-correction formulas. The editorial comments of Dr. John Mandelman improved this manuscript.

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