
Elizabeth J. Chittick, DVM, MS, DACZM; M. Andrew Stamper, DVM, DACZM; Jean F. Beasley; Gregory A. Lewbart, VMD, MS, DACZM; William A. Horne, DVM, PhD, DACVA

Objective—To determine safety and efficacy of an anesthetic protocol incorporating medetomidine, ketamine, and sevoflurane for anesthesia of injured loggerhead sea turtles.

Design—Retrospective study.

Animals—13 loggerhead sea turtles.

Procedure—Anesthesia was induced with medetomidine (50 µg/kg [22.7 µg/lb], IV) and ketamine (5 mg/kg [2.3 mg/lb], IV) and maintained with sevoflurane (0.5 to 2.5%) in oxygen. Sevoflurane was delivered with a pressure-limited intermittent-flow ventilator. Heart rate and rhythm, end-tidal partial pressure of CO₂, and cloacal temperature were monitored continuously; venous blood gas analyses were performed intermittently. Administration of sevoflurane was discontinued 30 to 60 minutes prior to the end of the surgical procedure. Atipamezole (0.25 mg/kg [0.11 mg/lb], IV) was administered at the end of surgery.

Results—Median induction time was 11 minutes (range, 2 to 40 minutes; n = 11). Median delivered sevoflurane concentrations 15, 30, 60, and 120 minutes after intubation were 2.5 (n = 12), 1.5 (12), 1.25 (12), and 0.5% (8), respectively. Heart rate decreased during surgery to a median value of 15 beats/min (n = 11). End-tidal partial pressure of CO₂ ranged from 2 to 16 mm Hg (n = 8); median blood gas values were within reference limits. Median time from atipamezole administration to extubation was 14 minutes (range, 2 to 84 minutes; n = 7).

Conclusions and Clinical Relevance—Results suggest that a combination of medetomidine and ketamine for induction and sevoflurane for maintenance provides safe, effective, controllable anesthesia in injured loggerhead sea turtles. (J Am Vet Med Assoc 2002;221:1019–1025)
duration and depth of anesthesia. Like the injectable agents, however, they display markedly different pharmacokinetic properties in reptiles, as compared to mammals, and may be associated with prolonged recovery periods, particularly in sea turtles. Following awake intubation in Kemp's ridley sea turtles, induction of anesthesia with isoflurane (3.4%) took only 7 minutes, but recovery times after 2 hours of anesthesia with 2% isoflurane averaged 4 hours.1 In a separate report,2 mask induction of an injured green sea turtle with 4% isoflurane took nearly 2 hours, and following surgical repair of a depressed skull fracture, recovery lasted 18 hours. We have had similar experiences of prolonged induction and recovery periods with the use of isoflurane in injured sea turtles in our hospital. As a result, we have elected to use sevoflurane for inhalant anesthesia of sea turtles. With its low solubility in blood, sevoflurane provides for fast anesthetic induction and rapid, smooth recovery.3 Sevoflurane has been used as an alternative to isoflurane in desert tortoises. Following awake intubation, induction of anesthesia with sevoflurane (3 to 7%) took only 3 minutes, and recovery times after 105 minutes of anesthesia with 2.5 to 3.75% sevoflurane averaged 28 minutes.4,5 An important disadvantage to the use of sevoflurane, compared with isoflurane, however, is that sevoflurane is currently 4 times as expensive.

The purpose of the study reported here was to develop an anesthetic protocol for anesthesia of injured loggerhead sea turtles. Specifically, we wanted to determine whether anesthetic induction with a combination of medetomidine and ketamine and maintenance with sevoflurane would be safe and effective.

**Criteria for Selection of Cases**

Medical records of injured loggerhead sea turtles (*Caretta caretta*) admitted to the North Carolina State University College of Veterinary Medicine between 1996 and 2000 were reviewed. Turtles were eligible for inclusion in the study if they had been anesthetized with a combination of medetomidine, ketamine, and sevoflurane.

**Procedures**

Anesthetic protocol—Upon arrival, sea turtles were manually restrained for examination, radiography, and weighing. Blood samples collected from the dorsal cervical sinus4 were submitted for a CBC and plasma biochemical analyses.1 In preparation for anesthesia, the turtles were positioned in ventral recumbency. Anesthesia was then induced with medetomidine4 (50 µg/kg [22.7 mg/lb]) and ketamine1 (5 mg/kg [2.3 mg/lb]) administered IV over 15 seconds in the dorsal cervical sinus (in some cases, medetomidine was administered IM 20 minutes prior to IV administration of ketamine). Once the jaws were fully relaxed, a bite block (6 × 1.25 inch PVC T-piece) was inserted to hold the beak open and provide access to the glottis. Closed curved hemostats were used to gently open the glottis for placement of a cuffed endotracheal tube.1 Tube size varied according to patient weight. For turtles weighing between 20 and 30 kg (44 and 66 lb), tubes with an internal diameter of 6 or 7 mm were used; for turtles weighing between 30 and 40 kg (66 and 88 lb), tubes with an internal diameter of 7 or 8 mm were used; for turtles weighing > 40 kg, tubes with an internal diameter of 9 or 10 mm were used. Because sea turtles have closed tracheal rings, care was taken to avoid overinflating the cuff. The time from injection of medetomidine and ketamine to endotracheal intubation was recorded as the induction time.

Once intubated, turtles were connected to a pressure-limited, intermittent flow ventilator3 that delivered sevoflurane in oxygen. The vaporizer7 was initially set to deliver 3 to 7% sevoflurane; the vaporizer setting was adjusted over time to deliver the minimum concentration required to prevent purposeful movement in response to surgical stimulation. The ventilator was initially set at 4 to 8 breaths/min with an inspiratory flow rate of 10 to 20 L/min (depending on weight) and peak inspiratory pressure (PIP) of 10 to 15 cm H₂O. Once turtles had reached a surgical plane of anesthesia, the respiratory rate was decreased to 1 to 2 breaths/min. In some turtles, a 16-gauge, 8-inch polyurethane catheter1 was placed in the dorsal cervical sinus for delivery of fluids intraoperatively. Either a balanced electrolyte solution5 or lactated Ringer's solution10 was administered at a rate of 5 to 10 mL/kg/h (2.3 to 4.5 mL/lb/h). In most instances, the catheters were removed immediately after surgery. After induction of anesthesia, turtles were positioned as necessary to allow for surgical access.

Heart rate and rhythm, end-tidal partial pressure of CO₂ (PetCO₂), and cloacal temperature were monitored throughout surgery, and venous blood gas analyses were performed intermittently. An electrocardiograph2 was used to monitor heart rate and rhythm; adhesive lead pads1 were placed directly on the carapace. A lead-II or lead-III electrocardiogram was obtained by positioning the leads over the right and left shoulders and left femur. Cloacal temperature was obtained with a long, flexible temperature probe attached to the electrocardiograph. A qualitative assessment of pulse intensity was obtained periodically by placing a Doppler probe7 over the ventrolateral cervical region. End-tidal partial pressure of CO₂ was monitored continuously by use of mainstream capnography7. Venous blood samples for blood gas analyses were collected in heparinized syringes through a 20-gauge needle or through the indwelling catheter. All samples were analyzed immediately.8

Administration of sevoflurane was discontinued 30 to 60 minutes prior to the end of surgery, and turtles were ventilated with 100% oxygen until extubated. The time from intubation to the time that administration of sevoflurane was discontinued was recorded as the sevoflurane maintenance interval. At the completion of the surgical procedure, atipamezole2 was given IV slowly; the dose of atipamezole was 5 times the dose of medetomidine. Ventilation was decreased at this time to 1 breath every 1 to 2 minutes to stimulate spontaneous breathing. Turtles were extubated when spontaneous respiration was accompanied by sustained voluntary movements of the head and flippers. Times from discontinuation of sevoflurane administration to atipamezole administration and from atipamezole administration to extubation were recorded.
Data analysis—Venous blood gas analyses were performed at 37°C, and measured values were corrected to 25°C with computer software. The corrected pH was calculated as pH at 37°C + (0.014°C X AT). The factor 0.014°C represents the ΔpH/ΔT value for green sea turtles in the 25 to 35°C temperature range. The corrected pH was calculated as pH at 37°C + (0.014°C X AT). The corrected PbCO2 was calculated as PbCO2 at 37°C X 10^(-0.019 X T). Corrected bicarbonate concentrations were calculated by incorporating corrected pH and PbCO2 values into the Henderson-Hesselbach equation HCO3- = 0.041 X PbCO2 X 10^(pH-6.175), where 0.041 and 6.175 are values for αCO2 and pKa, respectively, for sea turtle plasma at 25°C. Temperature-corrected estimates for PbO2 were calculated as PbO2 at 37°C X 10^(-0.014°C X T). The percentage of tidal volume that was physiologic dead space (VDS/VT) was calculated as (PbCO2 – PETCO2)/PbCO2 X 100.

Results

Thirteen loggerhead sea turtles met the criteria for inclusion in the study. All 13 turtles had been rescued along the coast of North Carolina; 6 were stranded on the beach, 5 were floating in open water, and 2 were trapped in nets. The turtles had a variety of skull, shell, and soft tissue traumatic injuries requiring surgery, including carapace fractures from propeller (n = 5), skull fractures (3), plastron fractures (1), spinal cord injury (1), a severed hind limb (1), an intestinal foreign body (fish hook; 1), and prolapse and traumatic amputation of the oviduct (1). Twelve turtles were juveniles of undetermined sex weighing between 17 and 49 kg (37.4 and 107.8 lb; median, 33 kg [72.6 lb]). The remaining turtle was a 120-kg (264-lb) adult female.

Results of preoperative bloodwork were available for 11 turtles (Table 1). Results of plasma biochemical analyses were abnormal for most of the 11, as determined by comparison with previously determined reference values. The most frequently encountered disorders were hypoalbuminemia (<1.2 mg/dL; n = 7), hypocalcemia (<6 mg/dL; 5) and hypophosphatemia (<6 mg/dL; 5). Seven turtles had hyperglycemia (>145 mg/dL). Two turtles had severe anemia (PCV = 9 and 11%), and 1 had severe hyponatremia (115 mEq/L). Three turtles had creatine kinase activities > 5,000 U/L. All other serum enzyme activities were within reference limits. Preoperative test results did not preclude us from proceeding immediately to surgery with any of the turtles.

Dorsal cervical sinus administration of medetomidine and ketamine induced heavy sedation and profound muscle relaxation, allowing for rapid and safe endotracheal intubation. Induction time was recorded in 11 turtles and ranged from 2 to 40 minutes (median, 10 minutes). Nine of the 11 turtles were intubated within 14 minutes. A surgical plane of anesthesia was generally reached in 5 to 10 minutes after administration of sevoflurane at a concentration of 3 to 4% was begun. Two turtles with severe, presumably painful wounds required initial sevoflurane concentrations of 6 to 7% for 30 and 15 minutes. Once a surgical plane of anesthesia was reached, however, anesthesia could be maintained with lower sevoflurane concentrations. For added analgesia and sedation, 3 turtles received an additional dose of medetomidine (25 to 50 µg/kg [11.4 to 22.7 µg/lb]) 1 to 2 hours after anesthetic induction. Median vaporizer settings for delivered concentration of sevoflurane 15, 30, 60, and 120 minutes after induction of anesthesia were 2.5 (n = 12), 1.5 (12), 1.25 (12), and 0.5% (8), respectively. The lowest delivered concentration for all 13 turtles was 0.5%. Sevoflurane maintenance times were recorded for 9 turtles and ranged from 70 to 315 minutes (median, 104 minutes).

Carapace lead placement allowed for consistent monitoring of heart rate and rhythm (Fig 1). Preoperative heart rates were recorded in 3 turtles and ranged from 20 to 30 beats/min (Table 2). Heart rates began to decline immediately after anesthetic induction in all turtles and typically stabilized within 15 to 20 minutes. Other than bradycardia, no arrhythmias were detected. The adult female loggerhead had the lowest heart rate (4 to 8 beats/min) during the first hour of anesthesia. This may have been in response to the high concentration of sevoflurane (4%) required to keep the turtle anesthetized. As the sevoflurane concentration was gradually reduced to 0.5%, heart rate

Table 1—Results of preoperative clinicopathologic testing for 11 injured loggerhead sea turtles.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Measured value</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg/dL)</td>
<td>167</td>
<td>66–297</td>
</tr>
<tr>
<td>Albumin (µg/dL)</td>
<td>1.1</td>
<td>0.6–1.9</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>26</td>
<td>9–34</td>
</tr>
<tr>
<td>Urea nitrogen (mg/dL)</td>
<td>69</td>
<td>16–166</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>0.4</td>
<td>0.3–0.5</td>
</tr>
<tr>
<td>Uric acid (mg/dL)</td>
<td>0.6</td>
<td>0.4–1.6</td>
</tr>
<tr>
<td>Calcium (mg/dL)</td>
<td>6.1</td>
<td>4.3–7.9</td>
</tr>
<tr>
<td>Magnesium (mg/dL)</td>
<td>4.1</td>
<td>2.5–6.8</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>151</td>
<td>115–162</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>3.5</td>
<td>2.4–4.0</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>114</td>
<td>96–127</td>
</tr>
<tr>
<td>Total CO2 (µEq/L)</td>
<td>21</td>
<td>14–40</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>37</td>
<td>32</td>
</tr>
<tr>
<td>Aspartate aminotransferase (U/L)</td>
<td>177</td>
<td>81–368</td>
</tr>
<tr>
<td>Lactate dehydrogenase (U/L)</td>
<td>118</td>
<td>314</td>
</tr>
<tr>
<td>Creatine kinase (U/L)</td>
<td>2,132</td>
<td>252–17,120</td>
</tr>
</tbody>
</table>

Figure 1—Electrocardiogram obtained from a loggerhead sea turtle before (awake) and after (anesthetized) induction of anesthesia. Lead pads were placed directly on the carapace. P waves, QRS complexes, and T waves are indicated by arrows. Chart speed = 25 mm/s.
increased to 15 beats/min and remained constant for the rest of the procedure. Heart rates of the juvenile turtles ranged from 10 to 20 beats/min during anesthesia, regardless of the duration of the procedure. Following extubation, heart rate ranged from 20 to 30 beats/min.

Median intraoperative cloacal temperature was 25°C (77°F; range, 23.5 to 26.5°C [74.3 to 79.7°F]). Compared with preoperative values, median pH and \( PV_O_2 \) increased slightly after induction of anesthesia (Table 2). In contrast, median PVCO2 and HCO3– concentration decreased after induction of anesthesia, with the lowest values recorded 1 to 2 hours after anesthetic induction (Fig 2). Four of the turtles had an immediate 50 to 75% decrease in PVCO2, compared with preoperative values, after induction of anesthesia that was sustained throughout the anesthetic period. Median PVCO2 and pH values for these turtles during the first and second hours after induction of anesthesia were 21 mm Hg and 7.64 and 18 mm Hg and 7.85, respectively. In contrast, median PVCO2 and pH values for the remaining 3 turtles during these periods were 33 mm Hg and 7.51 and 34 mm Hg and 7.58, respectively. The discrepancy in PVCO2 values between the 2 groups of turtles could not be attributed to differences in ventilator settings; however, review of the case records revealed that the 4 turtles with the lowest PVCO2 values had extensive shell fractures, whereas the 3 turtles with the higher PVCO2 values did not. The lowest PVCO2 values were recorded from a turtle that had sustained carapace and plastron injuries that penetrated the coelomic cavity.

Values for PETCO2 varied from 2 to 16 mm Hg throughout the intraoperative period, and PETCO2 did not appear to vary with PVCO2. For example, 2 turtles with PETCO2 of 12 mm Hg had PVCO2 of 21 and 36 mm Hg. One turtle positioned in dorsal recumbency with PVCO2 between 30 and 48 mm Hg had PETCO2 of 2 mm

**Table 2—Heart rate, venous blood gas values, and end-tidal partial pressure of CO₂ (PETCO₂) before, during, and after surgery in loggerhead sea turtles**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before surgery</th>
<th>During surgery (hours after intubation)</th>
<th>After surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>25 (20–30)</td>
<td>15 (10–22) 11</td>
<td>14 (9–20) 7</td>
</tr>
<tr>
<td>pH</td>
<td>7.48 (7.42–7.54)</td>
<td>7.51 (7.40–7.81) 4</td>
<td>7.50 (7.37–7.88) 5</td>
</tr>
<tr>
<td>PVCO2 (mm Hg)</td>
<td>34 (34–49)</td>
<td>29 (29–43) 6</td>
<td>21 (17–40) 4</td>
</tr>
<tr>
<td>PETCO2 (mm Hg)</td>
<td>NA</td>
<td>8 (2–16)</td>
<td>8 (2–16)</td>
</tr>
</tbody>
</table>

Anesthesia was induced with medetomidine (50 µg/kg [22.7 µg/lb], IV) and ketamine (5 mg/kg [2.3 mg/lb], IV) and maintained with sevoflurane (0.5 to 2.5%). Intermittent positive-pressure ventilation was performed during surgery. Turtles received atipamezole (0.25 mg/kg [0.11 mg/lb], IV) at the end of surgery. For heart rate, high and low values for each turtle in each time interval were used to determine median and range. All blood gas values were measured at 37°C and corrected to 25°C.

*Calculated using temperature-corrected pH and PVCO2 values and pKa and αCO2 values for sea turtle plasma at 25°C.

Hg. In some turtles, PETCO2 decreased after anesthetic induction and increased toward the end of surgery as the sevoflurane concentration was lowered. Intraoperative VdSV/Vt was estimated in 5 turtles and ranged from 40 to 80%.

Plasma lactate concentrations measured prior to anesthesia in 2 turtles were 1.59 and 2 mmol/L. Lactate concentration 1 to 2 hours after induction of anesthesia ranged from 0.6 to 2.8 mmol/L (n = 3). In a fourth turtle that had sustained carapace and plastron injuries, lactate concentrations 1 to 2 hours and 3 to 5 hours after anesthetic induction were 5.7 and 5.3 mmol/L, respectively. The PVO2 in this turtle decreased from 86 to 21 mm Hg during this time, and pH
inhalant anesthetic agents, use of this anesthetic of barbiturates alone, ketamine alone, or other loggerhead sea turtles. Compared with administration of medetomidine and ketamine for induction were typically delayed until the third such episode. The time from atipamezole administration to the turtle recovered within 5 minutes following IV administration of the turtle with a prolonged recovery, atipamezole had mistakenly been given IM. The turtle recovered within 5 minutes following IV administration of a second dose of atipamezole.

No anesthetic complications developed in any of the turtles during the first 24 hours after surgery. Three turtles died after the first week of postoperative care. Two of these turtles had been examined because of head trauma, and 1 of the 2 had had severe anemia. The third turtle had sustained multiple carapace and plastron fractures and was also severely anemic. Of the 10 turtles that survived, 9 were released to the wild following 1 to 2 years of supportive care, and 1 was scheduled to be released.

Discussion

Results of the present study suggest that use of a combination of medetomidine and ketamine for induction and sevoflurane for maintenance provides safe, effective, controllable anesthesia in critically injured loggerhead sea turtles. Compared with administration of barbiturates alone, ketamine alone, or other inhalant anesthetic agents, use of this anesthetic combination provided for rapid anesthetic induction, long procedure times, and fast anesthetic recovery. In addition, recovery time was not affected by duration of anesthesia or the surgical procedure. Two turtles with prolonged induction and recovery times may have received partial doses of medetomidine and ketamine or of atipamezole. Factors that likely contributed to the faster recovery time included the reversibility of medetomidine, the use of a low dose of ketamine, and the low solubility of sevoflurane in blood. The shortened recovery times were of special importance in these clinical cases, as they allowed for rapid transfer of the turtles to a separate rehabilitation facility located several hours away.

Clinicopathologic findings consistent with poor nutrition were evident in several turtles in this study. Hypoproteminemia and hypocalcemia raise concerns about plasma oncotic pressure, drug binding, cardiac contractility, and hemostasis. However, no guidelines currently exist for the lower limits of protein and calcium concentrations in sea turtles below which anesthesia should not be performed. Because of concerns about potential intraoperative fluid loss and lactic acidosis, we chose to administer a crystalloid solution without lactate to most of these turtles. The acetate and gluconate in this solution are considered to be more appropriate buffers for reptiles, as lactate metabolism in these species does not necessarily result in bicarbonate formation. This solution does not contain calcium, but because we did not have a means to measure blood pressure, we chose not to administer additional calcium, as we would not be able to assess its effects. Further studies are required to determine the optimal methods for managing fluid and electrolyte disturbances in sea turtles.

Induction of anesthesia with medetomidine and ketamine reduced the handling risks associated with endotracheal intubation in these turtles and eliminated the effect of breath holding on overall induction time. Although intubation is possible in some chelonians while awake, it is not a safe practice when working with larger sea turtle species that have powerful jaws capable of crushing thick-shelled mollusks. The dorsal cervical sinus, however, is readily accessible for IV administration of induction agents and catheter placement. Dosages of medetomidine and ketamine used in this study were much lower than those previously reported for reptiles in which either drug was used alone. When medetomidine was administered IM alone, a dose of 150 µg/kg (68.2 µg/lb) was required for light to heavy sedation of desert tortoises. Administration of ketamine at a dose of 8 to 18 mg/kg (3.6 to 8.2 mg/lb), IV, was necessary for intubation of sea turtles. Our results, on the other hand, suggest that, as in mammals, the use of medetomidine and ketamine in combination in sea turtles was more effective than the use of either drug alone. Moreover, it was our clinical impression that the use of medetomidine and ketamine for anesthetic induction allowed for extended periods when sevoflurane could be administered at low concentrations. This was especially apparent in the 3 turtles that were given additional doses of medetomidine for intraoperative pain relief.

Preoperative heart rates (20 to 30 beats/min; n = 3) were similar to mean heart rates reported for awake, resting green (24 beats/min), Kemp’s ridley (34 beats/min), and leatherback (24 to 27 beats/min) sea turtles. Soon after anesthetic induction, however, heart rates of turtles in the present study decreased (median, 14 beats/min; n = 11). There are at least 2 explanations for this. First, 1 or all of the drugs used in the anesthetic protocol may have directly caused bradycardia. Medetomidine, a potent α2-adrenoceptor agonist, triggers a profound parasympathetic response and sustained bradycardia in dogs and seems to have similar effects in desert tortoises, although resting heart rates in the study in which this was reported were quite high relative to heart rates in a previous study. Sea
turtles anesthetized with inhalant agents alone have also developed bradycardia. Mean heart rate decreased 56% from preoperative values in Kemp’s ridley sea turtles anesthetized with isoflurane. In contrast, no significant change in heart rate was observed in desert tortoises anesthetized with sevoflurane, which in mammals has cardiovascular effects similar to those of isoflurane. Alternatively, the bradycardia observed in this study could have been a result of a dive response initiated by intubation or general anesthesia. This response results in shunting of pulmonary blood and preferential perfusion of other organs while minimizing energetic costs by lowering heart rate and cardiac output. Physiologic responses to diving were demonstrated in a recent study in which free-ranging leatherback sea turtles were equipped with electrocardiographs and time and depth recorders. While at the surface, these turtles had heart rates ranging from 24 to 27 beats/min. During routine dives, however, their heart rates decreased to 16 to 19 beats/min, comparable to the intraoperative heart rates observed in the present study. Similar results have been obtained in other diving sea turtles under laboratory conditions. Although the definitive cause of the bradycardia observed in the present study was not determined, the low heart rates may have been within the typical daily range for loggerheads, which spend 80 to 94% of their time submerged. Accordingly, we chose not to administer anticholinergic drugs to these patients.

Median values for results of venous blood gas analyses in this study were similar to those previously reported for arterial and venous blood from loggerhead sea turtles at 25°C. Blood pH, PCO₂, and HCO₃⁻ concentrations in these studies ranged from 7.4 to 7.60, 25 to 35 mm Hg, and 19 to 36 mmol/L, respectively. Relative to mammalian blood at 37°C, these values are more alkalemic. Intraoperative pH in these turtles ranged from 7.6 to 7.8 as the result of low partial pressures of CO₂. This may have been caused by inadvertent hyperventilation. Loggerheads normally breathe 2 to 8 times per minute while at the surface and have tidal volumes ranging from 20 to 50 mL/kg (9.1 to 22.7 mL/ib). Although ventilator settings were similar for all turtles, the tidal volume delivered to turtles with shell fractures could have been excessive, in that a change in compliance resulting from shell fractures and coelomic cavity penetration likely altered the normal pressure-volume relationship of the lungs. Given that the ventilator used in this study was pressure limited, rather than volume limited, an increase in compliance would result in an increase in delivered volume. Alternatively, it is possible that the lungs were damaged and leaking, although escaping air was never obvious in the surgical field. In addition, other investigators have found that fully inflated loggerhead lungs leak air even in the absence of external tears. It is also possible that these turtles had reduced pulmonary shunting relative to turtles without carapace injuries, allowing for more alveolar gas exchange, or that these turtles had reduced metabolism. Regardless of the mechanism, these results indicate that sea turtles with fractured shells may benefit from reductions in ventilator rate or PIP.

Sea turtles in the present study were mechanically ventilated with a unique, electronically controlled anesthetic delivery system that functions as a pressure-limited ventilator. On inspiration, it will continue to deliver an oxygen-sevoflurane mixture until a preset PIP is reached (10 to 15 cm H₂O in this study). Adjusting the inspiratory flow rate (10 to 30 L/min in this study, depending on patient weight) controls the inspiratory time. Once the inspiratory pressure is reached, the inspiratory valve closes while a separate expiratory valve opens to release exhaled gases. The respiratory rate is controlled by adjusting expiratory time. Thus, the ventilator works as a non-rebreathing circuit, but unlike traditional non-rebreathing systems, flow is intermittent, rather than continuous, and unidirectional, rather than to-and-fro. High oxygen-flow rates are therefore not required to avoid rebreathing CO₂. Intermittent, rather than continuous, flow is particularly well suited for use with sevoflurane, as it substantially reduces the amount of waste gas produced.

Venous partial pressure of CO₂ was consistently higher than PETCO₂ in turtles in the present study, with the discrepancy between values ranging from 15 to 65 mm Hg. This disparity indicates that PETCO₂ is not a reliable indicator of ventilation adequacy in aquatic reptiles, as reported previously for anesthetized Kemp’s ridley sea turtles. With the substantial shunting of pulmonary blood that occurs with the dive response, positive pressure ventilation increases physiologic dead space. The net effect is a dilution of expired CO₂ proportional to the degree of shunting. Interestingly, right-to-left shunting in diving reptiles acts to reduce the acidity of pulmonary blood and increase the acidity of arterial blood. This serves to promote oxygen storage in the pulmonary vasculature and increase oxygen delivery to tissues, respectively. Approximate physiologic dead space in this study, expressed as a percentage of tidal volume, ranged from 40 to 80%.

Median PVO₂ values in this study exceeded the value (40 mm Hg) for dorsal cervical sinus blood from loggerhead sea turtles breathing ambient air at 25°C. In contrast, Kemp’s ridley sea turtles maintained on isoflurane in 40% oxygen in a previous study became hypoxemic and developed lactic acidosis. Loggerheads in the present study were maintained on 100% oxygen until extubation, and the lowest intraoperative pH recorded was 7.37. In fact, in 1 turtle in which plasma lactate concentration remained between 5 and 6 mmol/L throughout a 5-hour surgical procedure, the pH remained close to 7.6. The fact that the Kemp’s ridley sea turtles in the present study were all positioned in dorsal recumbency may account for differences between the 2 studies. In this position, the dorsally situated lungs are compressed by overlying body organs, and greater pressure is required for lung inflation. This may enhance ventilation-perfusion mismatching and dilution of oxygenated blood. Venous partial pressure of O₂ decreased in 2 of 4 turtles that were positioned in dorsal recumbency in this study, but neither
turtle became hypoxemic. However, we were not monitoring arterial blood gas partial pressures, and P\textsubscript{O\textsubscript{2}} values were corrected to a temperature of 25°C, not directly measured. We could not, therefore, estimate shunt fractions in these patients.

The present study raises an important issue with regard to temperature correction of blood gas values in reptiles. Most clinical analyzers measure pH, P\textsubscript{CO\textsubscript{2}}, and P\textsubscript{O\textsubscript{2}} at 37°C and then use formulas developed for humans to correct these values to patient temperature.\textsuperscript{13} In addition, values for H\textsubscript{CO\textsubscript{3}} concentration, base excess, and oxygen-hemoglobin saturation are all calculated on the basis of assumed values for the carbonic acid dissociation constant, the buffering capacity of human plasma, and the heat required for oxygenation of human hemoglobin. In many cases, however, values for these parameters in ectotherms differ considerably from those for humans. On the basis of comparisons of the response of pH and P\textsubscript{CO\textsubscript{2}} to temperature changes in mammals,\textsuperscript{12,20} green sea turtles,\textsuperscript{11} and other reptiles,\textsuperscript{21} we conclude that corrections made by the analyzer used in this study were adequate for pH and P\textsubscript{CO\textsubscript{2}} in the range of 25 to 37°C. At lower temperatures, however, pH changes in loggerhead sea turtle blood occur at a much steeper rate.\textsuperscript{22} We also concluded that corrections to P\textsubscript{O\textsubscript{2}} made by the analyzer were inaccurate, as the formulas assumed a much higher enthalpy of formation required for oxygenation of hemoglobin than has been measured in loggerheads.\textsuperscript{22} Studies\textsuperscript{20,26} have shown that sea turtle hemoglobin is relatively insensitive to pH, temperature, and allosteric effectors such as adenosine triphosphate and 2,3 diphosphoglycerate. This molecular adaptation allows O\textsubscript{2} delivery to tissues regardless of water temperature at various latitudes and depths.\textsuperscript{26} In light of these unique features of sea turtle hemoglobin, we conclude that temperature corrections for P\textsubscript{O\textsubscript{2}} in sea turtles should be based solely on solubility changes, not changes in affinity for hemoglobin.\textsuperscript{12}

References


Scientific Reports: Retrospective Study

1025