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Marine Turtle Newsletter

Resuscitation of Sea Turtles

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Several techniques have been described for resuscitation of comatose sea turtles. These include

a. electrical stimulation of the pectoral region (Shoop, 1982),
b. periodic compression (pumping) of the plastron with the turtle in a supine position (Hopkins and Richardson, 1984), and
c. insertion of a plastic tube into the trachea followed by gentle blowing into the tube at irregular intervals (Balazs, 1986).

Given the potential for adverse effects, electrical shock cannot be recommended for routine resuscitation of sea turtles. Pumping of the plastron is also a questionable resuscitation technique. Balazs (1986) reported that plastral pumping does not ventilate the lungs with air because of glottal lock (i.e., airway closure). In addition, placing turtles in a supine position during plastral pumping causes the viscera to compress the dorsally located lungs, thereby reducing lung volume and hindering lung inflation. We describe herein a modification of the resuscitation technique of Balazs (1986) that has been used successfully with Kemp's ridley sea turtles (*Lepidochelys kempi*) recovering from general anesthesia and anoxic submergence.

The well-accepted ABC's of resuscitation are to preserve

a. a clear **A**irway,
b. normal **B**reathing, and
c. **C**ardiac function.

In our experience, airway closure resulting from glottal lock and cessation of normal ventilatory patterns (rather than cardiac dysfunction) limit the recovery of comatose sea turtles. Nevertheless, inadequate gas exchange will eventually compromise normal cardiac function and decrease the survival potential of comatose turtles. Therefore, the present technique was designed to resuscitate the turtle by manually or mechanically increasing pulmonary gas exchange during the comatose period. In our procedure, the turtle is maintained in a prone position out of water. A mouth gag is used to hold the jaws open and permit easy access to the glottis, which is located posterior to the tongue on the floor of the mouth. Ideally, the mouth gag should be constructed from wood or polyvinylchloride (PVC) in order to minimize damage to the keratinous sheaths that cover the jaws.

The turtle is intubated with an appropriately-sized endotracheal tube with a low pressure cuff. Endotracheal tubes that are too large damage the tracheal mucosa, whereas tubes that are too small increase the resistance of gas flowing through the tube and decrease effective respiratory resuscitation. Suitable endotracheal tubes for 4-23 kg Kemp's ridley turtles are 5-10 mm outside diameter (3-7 mm inside diameter). The endotracheal tube is carefully inserted through the glottis into the trachea, to a depth of approximately 5-8 cm. The consequence of inserting the tube past the tracheal bifurcation is inflation of only one lung, and therefore, inadequate gas exchange and possible barotrauma. Once the tube is in position, the low pressure cuff is inflated to prevent air leakage on inspiration. The turtle is then ventilated with air repeatedly and at regular intervals. Ventilation frequency depends on the duration of the comatose period. Minimum ventilation rate should be 2-4 breaths per minute and should be increased the longer the turtle has been comatose.

Lung volumes in reptiles vary as a function of body weight (Tenney and Tenney, 1970). However, the requisite lung volume of individual sea turtles may be unknown at the time of intubation. Generally, the procedure for small turtles is to gently inflate the lungs until the carapace begins to move. Inspiratory duration should be between 1-2 seconds and lung inflation should be slow and gradual throughout the inspiratory period. The preferred method is to underinflate the lungs with less than maximal volumes of air, while augmenting gas exchange by increasing the ventilatory frequency. This ensures that the lungs are not overinflated, which may cause significant and irreparable lung damage. We recommend ventilating the turtle with a hand-held resuscitator bag in cases where mechanical ventilation is unfeasible (i.e., in the field), rather than the technique of blowing into the tube for two reasons. First, human-exhaled gases contain less O_2 (17-18%) and more CO_2 (about 5%) than air (about 21% O_2, 0.03% CO_2). Second, zoonotic diseases may be contracted by the person performing the oral resuscitation procedure.

The low pressure cuff on the endotracheal tube should be deflated when the turtle begins to revive. In our experience with comatose Kemp's ridleys, immediate post-revival characteristics include movement of the pectoral and pelvic muscles, hyperventilation (6 to 10 breaths per minute), and significant increases in heart rate. Heart rate (non-invasive femoral artery ultrasonic Doppler flow probes) and cloacal temperature should be monitored during the entire resuscitation procedure. We have found that revival of comatose turtles is adversely influenced by cloacal temperatures outside the range 25-30 °C. Revival takes longer at temperatures below 25 °C. Temperatures above 30 °C may approach the critical maximum. In all cases, comatose and/or revived turtles should be transported to knowledgeable authorities (e.g., local veterinarian, zoo) for continued treatment and monitoring. Manual ventilation must be continued during transport.

To date, we have found no resuscitation technique that will work with "wet drowned" sea turtles. One 20 kg Kemp's ridley was found to "wet drown" following a period of anoxic submergence. The turtle was conscious and active upon surfacing but quickly became lethargic. After one hour of intermittent unassisted ventilation with no improvement in blood gasses or pH, the turtle ceased ventilating and its heart rate fell from 42 to 6 beats per minute. At that point, the turtle was intubated and its lungs mechanically ventilated with room (ambient) air. Seawater was collected from the lungs via the intubation tube, providing clear evidence that the turtle had inhaled water and "classically" drowned. Continued mechanical ventilation did not improve blood gases (although heart rate increased six-fold), consistent with the view that the inhaled seawater had irreparably damaged the blood-gas barrier, hindering gas exchange. Unfortunately, the turtle died within 24 hours. Conversely, a comatose Kemp's ridley survived severe anoxic acidosis ("dry drowning" where, as a result of glottal lock, the turtle does not inhale water), but required mechanical ventilation for 48 hours before it

would respond to tactile stimulation and ventilate unassisted. The turtle was returned to water and did not demonstrate any adverse side effects from the anoxia or the ventilation procedure.

The technique we have described for resuscitation of comatose sea turtles is easy to perform, can be used efficiently in the laboratory or in the field, and does not require substantial investment in equipment. Endotracheal tubes and manual resuscitators can be purchased for less than US$ 70 from commercial suppliers of hospital equipment. Cloacal thermometers and ultrasonic Doppler probes are useful but optional equipment.

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