Respiratory, cardiovascular and metabolic adjustments during steady state swimming in the green turtle, *Chelonia mydas*

P.J. Butler, W.K. Milsom*, and A.J. Woakes
Department of Zoology and Comparative Physiology, University of Birmingham, Birmingham B15 2TT, England
Accepted August 1, 1983

**Summary.** Heart rate and pulmonary artery blood flow of resting green turtles, *Chelonia mydas*, at 29 °C increased with lung ventilation (heart rate from 24 ± 5 to 51 ± 8 beats min). When swimming at 0.6 m s⁻¹ in water at 30 °C, oxygen uptake was 2.83 times and respiratory frequency was 2.75 times the resting values. Heart rate was 1.33 times that during ventilation at rest but 2.83 times that at the end of a breath hold at rest. Partial pressures of oxygen and carbon dioxide, lactic acid concentration and pH of arterial blood, when swimming at 0.5 m s⁻¹, were similar to those soon after ventilation at rest. Pulmonary blood flow did not decline to low levels between breaths, when the animals were swimming, as it did when they were at rest.

In active turtles it appears that pulmonary perfusion remains elevated, supplying oxygen to the locomotory muscles at a sufficiently high rate to support the complete aerobic production of energy, and that respiratory frequency is kept as low as possible, as surfacing for air increases the metabolic cost of swimming.

**Introduction**

Many studies on amphibians and reptiles have emphasised how the incompletely divided ventricle is ideally suited to animals which ventilate their lungs intermittently (Shelton 1976; White 1976). During breath holding, constriction occurs somewhere in the pulmonary circuit thus causing a large proportion of the blood leaving the heart to bypass the lungs (a R → L shunt). During lung ventilation, however, pulmonary vasodilation occurs and perfusion of the lung increases, thus matching the level of ventilation. It is also clear that pulmonary perfusion is not always related directly to ventilation. Changes in partial pressures of oxygen in the lungs and arterial blood of *Pseudemys scripta* during voluntary dives of long (e.g. 3 h) duration indicate that, while pulmonary perfusion may decline at the beginning of such a dive, it may increase at various times during the dive (Burggren and Shelton 1979). It has also been demonstrated for *Chelonia mydas*, *Testudo graeca* and *P. scripta* that pulmonary perfusion increases during locomotor activity in the absence of lung ventilation (Johansen et al. 1970; Shelton and Burggren 1976).

This latter observation, in particular, is important with respect to those reptiles, such as *Chelonia mydas*, which swim submerged and surface intermittently to breathe. The only study on the energetics of swimming in *C. mydas* concludes that, despite the ability of the animal to metabolize anaerobically when submerged and inactive, the anaerobic contribution to swimming is insignificant (Prange 1976). The same study also points out that the act of lifting the head into the air to breathe increases the energetic cost of swimming. It would seem sensible, therefore, for the green turtle to utilise as much oxygen in the lung as possible between breaths and keep respiratory frequency to a minimum, particularly during its long migrations (Carr and Goodman 1970). Unfortunately, Prange (1976) did not measure any cardiovascular or respiratory variables (except oxygen uptake) leaving this suggestion speculative.

Thus, the present study was undertaken in order to determine the changes in blood gases, lactic acid, heart rate and pulmonary blood flow that occur in green turtles at rest and while swimming at various velocities.
Materials and methods

Experiments were performed on juvenile green turtles, Chelonia mydas, of undetermined sex whose mass ranged from 0.92 to 1.32 kg. The animals were obtained from Cayman Turtle Farm Limited, Grand Cayman, where they had been bred from captive stock. They were sent by airfreight to the U.K. and kept in a tank (160 x 80 x 30 cm deep) of circulating, filtered sea water at 28 °C for at least 2 weeks before use. They were fed trout pellets at a ration of 1.2% of their body weight per day.

All operations were performed on animals anaesthetized with 2-3% Halothane (I.C.I. Limited) in oxygen-enriched air (i.e. 29% O₂, 71% N₂). Recovery took from 5-60 min and the animals were left for at least 18 h before being experimented upon. Heart rate was measured from the electrocardiogram which was obtained by way of a purpose-built PIM radiotransmitter (Butler and Woakes 1982). For placement of the transmitter a rectangular portion of the plastron was cut with a scalpel and removed. The 2 separate electrodes of the transmitter were positioned either side of the heart and the body of the transmitter was inserted into the abdominal cavity. The piece of plastron was glued or stitched back in place and the whole area was covered by a piece of thick latex rubber held in place by cyanoacrylate adhesive (Radiospares Limited). The rubber patch was smeared with silicone grease, thus giving a watertight seal. The transmitted signal was received by a Sony CRF 5090 receiver, demodulated by a purpose-built demodulator (Woakes 1980) and displayed on a pen recorder (Ormed Ltd.).

In three turtles the right carotid artery was cannulated to allow the measurement of blood pressure, and the taking of arterial blood samples. It was necessary to flare the end of the cannula (to provide good anchorage) and to stretch the central end of the blood vessel so that when the neck shortened in the conscious animal, the wall of the artery did not have a valving effect at the end of the cannula. Blood pressure was monitored by a Bell and Howell 4-327-L21 transducer and displayed on a pen recorder (Ormed Ltd.). Blood gases and pH were monitored by BMS 3 Mk II and PHM 73 analysers (Radiometer Ltd.). The O₂ and CO₂ electrodes were calibrated using humidified gases prepared by gas mixing pumps (Wösthoff, Bochum) and the pH electrode by precision buffers. All electrodes were at the temperature of the water containing the experimental animal. Blood oxygen content was measured by a Lex-Oz-Con analyser (Cavitron Ltd.), haematocrit was determined by a microhaematocrit centrifuge (Hawksley) and lactic acid concentration was assayed enzymatically using a standard kit (Sigma) and a spectrophotometer (Model 25, Beckman).

Because of the shape of the coracoids and of the position of their associated muscles, it was not possible to expose the heart and major blood vessels from a ventral approach, as has previously been the case with other chelonians (Shelton and Burggren 1976), so in two turtles two oblong holes were cut, with a necropsy saw, in the carapace either side of the mid line at the level of the second neural plate. The lungs were briefly deflated while the aortic arch was exposed through the left and the pulmonary arch through the right opening. An electromagnetic flow probe (3.0 or 3.5 mm diameter – Biotronex Ltd.) was placed around each of these blood vessels and the lungs reinflated. The leads from the probes were anchored to nearby connective tissue, the pieces of carapace replaced and the two areas were each covered with latex rubber as described previously. Post mortem inspection revealed that the aortic arch curved in front of the probe which was well positioned and fitted snugly around the vessel in each animal. Zero flow was apparent from the diastolic portion of flow traces for the aortic arch (cf. Shelton and Burggren 1976). The only accessible portion of the pulmonary artery was curving from the heart to the lung, so the probe on this artery did not have such a good fit and zero flow was not so easy to determine. During very low heart rates the base-line drifted, presumably as the walls of the artery collapsed away from the probe (cf. Shelton and Burggren 1976). Smaller probes occluded the artery too much. The method described by Shelton and Burggren (1976) was therefore adopted, viz. a constant level of trace immediately before systole was assumed to be zero. Because of the small number of animals that were available for this part of the study and because of the uncertainties of the zero level of the traces from the pulmonary artery, these data were not quantified. However, both animals showed similar responses during the experiments so the data are qualitatively useful and calibration bars are given in Fig. 4 for information. In vitro calibration of the flow probes was carried out at the end of each experiment using the relevant excised pieces of artery from the experimental animal and saline from a reservoir set at a height equivalent to mean blood pressure recorded from the other turtles. The outputs from the flow probes were displayed, via a pulsed logic electromagnetic flow meter (Model BL 610, Biotronex), on two channels of a rectilinear pen recorder (Ormed Ltd.).

A state of rest was obtained for all turtles by placing them individually in fresh water at approximately 28 °C in a darkened glass tank 50 x 47 x 36 cm deep. Five turtles were trained to surface at a hole, 9 cm diameter in a perspex sheet covering all of the water at a depth of 1 cm. The hole itself was covered by a dome-shaped chamber of 150 ml volume. Air was pumped through this chamber at a rate of 10 l min⁻¹. Gas leaving the chamber passed through a pneumotachograph (Mercury Electronics Ltd.) which was connected to a differential pressure transducer (Model 270, Hewlett Packard Ltd.). This enabled air flow during lung ventilation to be measured (cf. Brett and Shelton 1979). The standing voltage resulting from the continuous air flow was backed off so that the air flow caused by lung ventilation could be integrated to give tidal volume (see Fig. 1). The composition of the effluent gas was continuously