Comp. Biochem. Physiol. 1977, Vol. 58A, pp. 185 to 191. Pergamon Press. Printed in Great Britain

PULMONARY STRETCH RECEPTORS REGULATE HEART RATE AND PULMONARY BLOOD FLOW IN THE TURTLE, *PSEUDEMYS SCRIPTA**

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(*Received* 4 *February* 1977)

Abstract—1. The coupling of ventilatory and circulatory activity present during normal periodic breathing in turtles was produced experimentally during artificial ventilation via lung cannulae. Heart rates and left pulmonary minute flow of 25–30 beats/min and 5–6 ml/min associated with voluntary breathing changed to 3–10 beats/min and 0.1–0.5 ml/min during apnoea. Artificial tidal ventilations with air promptly brought heart rate and pulmonary flow back to 75% of the values during voluntary breathing.

2. Step-wise imposed changes in lung volume caused reductions in heart rate and pulmonary flow on withdrawals and prompt restoration of initial values on air injection.

3. Lung injection of N_2, O_2 or CO_2 enriched gas gave responses similar to injection of air and the involvement of pulmonary chemo-sensitive (CO_2) receptors cannot be important in eliciting the cardio-respiratory coupling. Measurement of intrapulmonary pressure revealed the circulatory changes to be intimately related to the recorded pressure and/or the associated pulmonary volume change.

4. A change in intrapulmonary pressure and/or volume produced by placing turtles at variable depths in a water column caused graded changes in heart rate and pulmonary flow with depth. Atropinization completely abolished the coupling between pulmonary stretch and cardiovascular changes.

5. The presence of a pulmonary stretch receptor function is discussed in a phylogenetical perspective. Its importance is amplified in periodic breathers like chelonians and especially in aquatic forms where lung volume changes occur with depth and during breathholding due to aquatic CO_2 elimination through the skin.

INTRODUCTION

Aquatic reptiles typically ventilate their lungs in a periodic fashion. Ventilatory activity naturally ceases during diving or experimental submersion but also when removed from water aquatic reptiles show non-ventilatory periods interrupted by bursts of breathing (McCutcheon, 1943; Randall *et al.*, 1944; Burggren, 1975). Resumption of breathing is typically accompanied by striking increases in heart rate (ventilation tachycardia) (Belkin, 1964; Millard & Johansen, 1974; Burggren, 1975) and an increased blood flow to the lungs (White & Ross, 1966; Johansen *et al.*, 1970; Shelton & Burggren, 1976), a response also characteristic of habitual divers among birds and mammals (see Andersen, 1966; Angell James & Daly, 1972 for review). The physiological implications of the coordination of ventilatory and circulatory activities in relation to respiratory gas exchange and transport and to cardiac energy expenditure are now well appreciated in reptiles (White, 1976) and some aspects of their neural basis, particularly the efferent reflex pathways, have been revealed (Burggren, 1975). However, there is a paucity of information on what stimuli, receptor types and afferent reflex pathways elicit and coordinate the changes in lung ventilation, heart rate and lung perfusion. Evidence suggests that the development of ventilation tachycardia may depend in part on a direct interaction between CNS regions controlling cardiac and ventilatory activity (Huggins *et al.*, 1970, for crocodilians, and Burggren, 1975, for chelonians). However, the processing of sensory information eliciting such coordinated changes are not well understood. Similarly, changes in heart rate sometimes develop in anticipation of breathing (Belkin, 1964; Boyer, 1963; Gaunt & Gans, 1969; Johansen *et al.*, 1970; Burggren, 1975), and the neural events underlying that response are unknown. Also, potential peripheral sites for receptors and their modes of stimulation are not well documented for reptiles. The lung parenchyma itself may be the site of mechanoreceptors, or chemoreceptors may screen changes in gas composition of the lungs or the gas composition or pH of pulmonary venous blood.

The present study attempts to assess the role of intrapulmonary pressure for the elicitation of cardiovascular changes associated with voluntary and artificial breathing in the turtle, *Pseudemys scripta*. Intrapulmonary pressure will determine the stretch in the pulmonary parenchyma and bear a relation to the volume of the lung itself. Since the lungs of an aquatic animal will be externally compressed during diving a variable mechanical stimulus will affect the lungs. The study therefore also inquires about how external compression during diving affects heart rate and lung perfusion.

The gas composition of lung air varies with breathing and particularly large variations will attend the first breath following a non-ventilatory period. Secondarily, changes in lung gas composition will alter blood gases and pH. Pulmonary CO_2 receptors are well documented in birds (Fedde & Petersen, 1970)

* This study was supported by a grant from the Danish Natural Science Research Council.

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and their presence in reptiles have recently been reported (lizards, Gatz *et al.*, 1974; chelonians, Milsom & Jones, 1976). This study additionally was designed to determine what role sudden changes in lung gas composition have on pulmonary blood flow and heart rate.

MATERIALS AND METHODS

The experimental animals used were healthy specimens of the freshwater turtle, *Pseudemys scripta*, weighing between 500 and 900 g. All experiments were performed at 25°C.

Lung cannulation was performed on animals anaesthetized with cold torpor (1°C for 12 hr) as described by Burggren (1975). A conical hole (maximum dia 1 cm) was drilled in the carapace over the anterior region of the lung. The pleura and lung wall were exposed, and the tip of a PE60 cannula was advanced through a small perforation in the lung wall into the lumen of an anterior lateral chamber. The walls of the lung and pleura were tied securily around the cannula which was led out through the hole in the carapace. A rapidly-setting epoxy was applied to seal the opening in the carapace around the cannula. Both the left and right lung of each animal was cannulated and the two cannulae connected by a T joint, the free end of which was connected to a 3-way tap fitted with one or two syringes. The syringes, which could be filled with air or other gas mixtures, could be used to make stepwise injections or withdrawals of gas or to ventilate the lungs in a tidal fashion. Since the T provided an external low resistance connection between the two lungs, injection or withdrawal of gas was evenly distributed between both lungs. In most experiments intrapulmonary pressure was also monitored. The side arm of a second T joint placed between the lungs and the tap was connected to a Statham P23Dd pressure transducer, whose output was displayed on a Brush 260 recorder. The transducer was calibrated with static water columns.

In addition to the lung cannulation, a cuff-type electromagnetic flow transducer (Statham) was placed around the proximal portion of the left pulmonary artery of each turtle. A 3 cm square piece of the plastron of an anaesthetized animal was excised with a cast-cutter directly over the heart and central arteries in preparation for the flow probe placement. The left pulmonary artery was exposed, and a flow transducer of an appropriate diameter, usually with a 1.0–1.5 mm lumen, was placed around the vessel. The transducer leads were guided out through a notch cut in the excised piece of plastron, which was scaled back into position with rapidly-setting epoxy. A Statham 2202 electromagnetic blood flow meter with an electrical zero function was used to monitor blood flow. The output from this meter was also displayed on the Brush recorder. Stroke flow was derived by integration of flow profiles using weighing or square counting methods. At the termination of each experiment the pulmonary artery was excised and the flow transducer was calibrated *in vitro* with reptile saline delivered from a reservoir placed approximately 40 cmH_2O above the artery.

Animals were returned to room temperature (25°C) after surgery, and were allowed a 12–24 hr period of recovery before experiments were performed. The turtles showed no apparent effects of the lung cannulation and flow transducer implantation. Dissection of animals sacrificed 1 week or more after surgery revealed no evidence of gross infection, pulmonary edema or hemorrhage and no lesions of the pulmonary artery wall.

Each turtle complete with lung cannulae and pulmonary flow transducer was suspended in water without restraint of the head or limbs. This was accomplished by glueing the end of a vertical 1.5 m long rod to the top of the turtle's carapace. The animal attached to the end of the rod was then positioned at the desired depth in a 1 m deep container filled with water. In some experiments, the animals were placed just below the surface of the water, so that they were free to lift their heads above the surface and voluntarily ventilate their lungs. In other experiments, dives were simulated by raising or lowering the animals in steps through the column of water. It was essential that these involuntary dives should not be accompanied by any vibration, noise or other undesirable disturbance. This was best accomplished by using a finely controlled hydraulic jack to smoothly raise or lower the container of water over the turtle. In this fashion, dives to a specific depth in the water column between 0 and 1.0 m could be readily produced without mechanically disturbing the turtle itself. Animals were left at a specific depth at least until a stable cardiovascular state was evident, usually occurring after 1–2 min.

RESULTS

Initial experiments were performed to determine whether a rhythmic tidal ventilation with air through the lung catheters would alter heart rate and lung perfusion of turtles in a diving position. These experiments would mimic voluntary breathing in terms of transient lung volume and pressure changes and in terms of changes in lung gas composition. Figure 1 shows the results of a representative experiment on a *Pseudemys* which was suspended below the water surface, yet which could extend its head to the surface and breathe. Voluntary breathing in this animal was rapidly accompanied by marked cardiovascular adjustments. Maximum heart rate and left pulmonary minute flow were 25–30 beats/min and 5–6 ml/min, respectively, at the end of each breathing series, compared to approximately 3–10 beats/min and 0.1–0.5 ml/min during periods of apnoea. Artificial ventilation, consisting of repeatedly withdrawing 10 ml of lung gas followed by immediate replacement of this volume with air, was initiated after 15 min of voluntary apnoea when heart rate was approximately 2–4 beats/min and minute flow in the left pulmonary artery approximately 0.1 ml/min. Within seconds from the onset of artificial ventilation both heart rate and pulmonary flow increased conspicuously, and after 8 artificial "breaths" heart rate was 16–18 beats/min and left pulmonary flow was approximately 3 ml/min. With the termination of artificial ventilation, pulmonary stroke flow and, to a lesser extent, heart rate began to decline towards pre-ventilation levels. Artificial ventilation with air generally produced maximal heart rate and blood flow responses which were about 75% of the maximum responses accompanying voluntary breathing.

Step-wise imposed changes in lung volume also consistently produced striking changes in heart rate and pulmonary perfusion. Figure 2 illustrates the effect of withdrawing lung gas in steps during a period of voluntary apnoea. Both heart rate and left pulmonary artery stroke volume decreased immediately upon each consecutive withdrawal of lung gas. Injection of a volume of air equivalent to the total volume of removed lung gas rapidly returned heart rate as well as pulmonary blood flow to very near control levels. Similarly, if the 3-way tap connected to the lung was opened to the atmosphere, allowing intrapulmonary pressure to fall towards ambient hydrostatic pressure

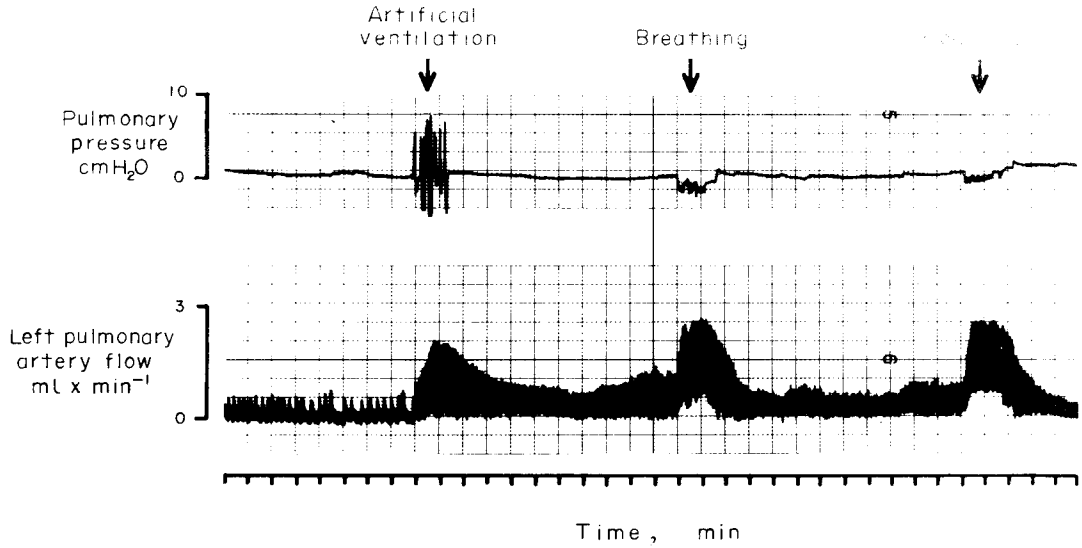

Fig. 1. Intrapulmonary pressure and left pulmonary artery blood flow during artificial ventilation and voluntary breathing in *Pseudemys scripta*.

as the lung deflated, it caused heart rate and pulmonary flow to decrease sharply (Fig. 3). If air was then injected back into the lungs in amounts sufficing to restore pulmonary pressure at the level prevailing before opening the tap, heart rate and left pulmonary stroke volume again returned to values close to the control.

These data demonstrate that cardiovascular changes can be elicited by artificial tidal ventilation with air or by step-wise, imposed lung volume changes, but they do not reveal to what extent these responses are mediated by pulmonary stretch receptors or by peripheral chemoreceptors located in the lungs or arterial circulation. Hence, the experiments were repeated using 100% N_2, 100% O_2 or 5% CO_2 in air instead of air alone for either tidal ventilation or the step-wise withdrawal reinjection experiments. Changes in heart rate and pulmonary blood flow produced by artificial tidal ventilation during voluntary apnoea with each of these experimental gas mixtures were similar to those produced by ventilation with air, although maximum responses with air ventilation often were 10–20% higher than with other gas mixtures. The effects of various gas mixtures on these cardiovascular parameters during step-wise lung volume changes were also very similar to the experiments with air. Changes in heart rate in experiments when 30 ml of lung gas was withdrawn during apnoea and subsequently reinjected using an equivalent volume of experimental gas are given in Table 1. In all experiments the injection of N_2, O_2 or CO_2 enriched gas into the lungs following removal of air

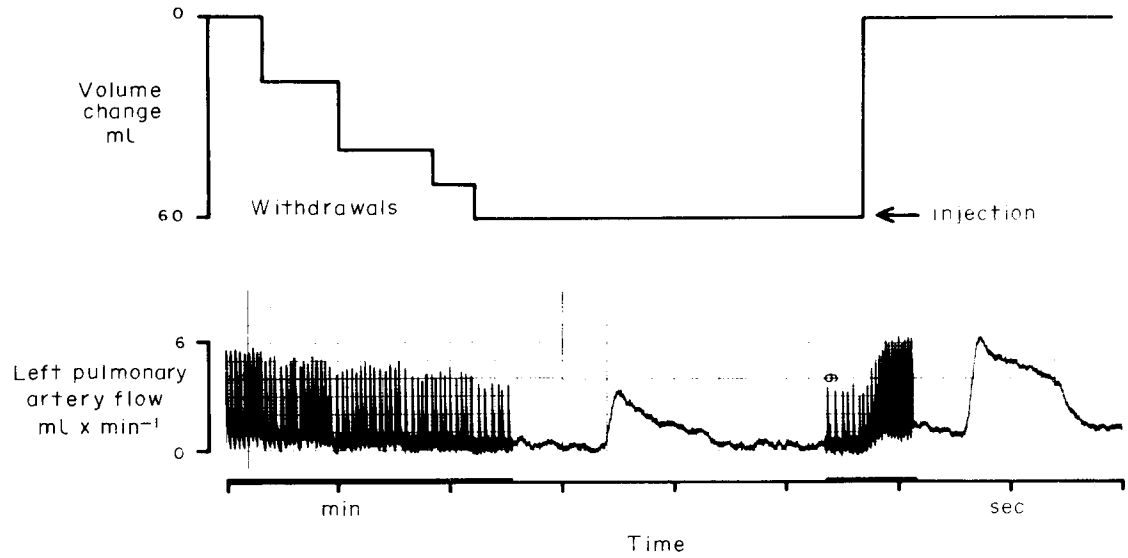

Fig. 2. Changes in left pulmonary artery blood flow during step-wise withdrawals of gas from the lungs of *Pseudemys*. Replacement of the lung gas promptly returns heart rate and pulmonary blood flow to pre-withdrawal values.

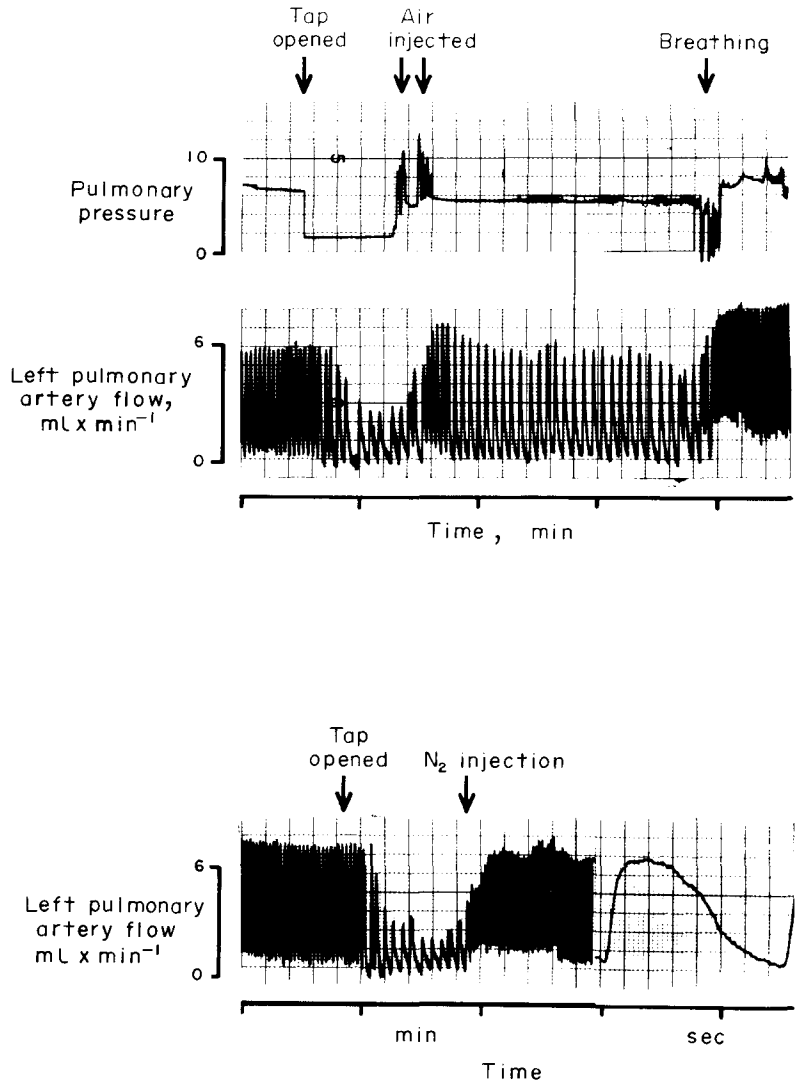

Fig. 3. Intrapulmonary pressure and left pulmonary artery blood during artificial deflation of the lungs and subsequent reinjection of air and N_2 in *Pseudemys*. Voluntary breathing is effective in restoring heart rate and pulmonary blood flow.

caused heart rate to return towards control levels. However, even reinjection of air or the sample of lung gas previously withdrawn usually returned heart rate to only approximately 90% of its control level, compared to 77–85% by the 3 experimental gases (Table 1).

During diving in natural environments turtles will experience changes in the hydrostatic pressure acting upon their lungs, which in turn may significantly affect both intrapulmonary pressure and volume. Experiments were designed to reveal whether heart rate and pulmonary blood flow would be affected not by

Table 1. Effect of withdrawal of 30 ml of lung gas and replacement with 30 ml of N_2, O_2 or 5% CO_2 on heart rate during diving in *Pseudemys scripta*

	Heart rate before lung gas withdrawal (beats/min)	Heart rate after lung gas withdrawal (beats/min)	Heart rate after injection of experimental gas (beats/min)	Heart rate after injection as % of control heart rate
Air	30 ± 5	14 ± 3	27 ± 5	90%
100% N_2	31 ± 3	14 ± 6	25 ± 6	81%
100% O_2	27 ± 2	15 ± 3	23 ± 4	85%
5% CO_2 in air	31 ± 4	20 ± 3	24 ± 5	77%

Mean values ± 1 S.D. ($n = 5$).

withdrawing or adding gases, but simply by inducing dives to different depths in a water column. Changes in heart rate and pulmonary blood flow produced solely by variations in hydrostatic pressure during 5 experimental dives in *Pseudemys* to a depth of 75 cm are illustrated in Fig. 4. Intrapulmonary pressure increased in proportion to the depth in water but tended to be 5–10 cmH_2O higher than the ambient hydrostatic pressure, suggesting that an active constriction of the lung prevails. As the depth of descent and intrapulmonary pressure increased, heart rate and pulmonary perfusion both fell markedly. At a depth of 75 cm heart rate was approximately 40% and left pulmonary artery minute flow was approximately 20% of the levels prevailing when the turtle was at the water surface. As with the deflation–inflation experiments, there was evidence of a hysteresis in the cardiovascular responses as the animals descended and then ascended. This phenomenon is illustrated in Fig. 5. In these experiments the maximum depth of the dives was only 15 cm, as reflected in the low intrapulmonary pressures. Nonetheless, the magnitudes of change in heart rate and pulmonary perfusion were in these experiments similar to those occurring during deeper dives in other turtles. Heart rate and left pulmonary artery stroke flow after the dive and completion of the ascent usually returned to levels which were 10–20% lower than pre-dive levels. Generally, this hysteresis was evident regardless of pre-dive levels of heart rate or pulmonary flow.

Changes in intrapulmonary volume and pressure which occur during experimental manipulation of lung volume or natural diving may have a direct mechanical effect on pulmonary blood vessel diameter. This will result in changes in pulmonary impedance, and so ultimately affect blood flow through the lung. Experiments were performed to examine this possibility, and, if changes in pulmonary perfusion and heart rate during artificial ventilation and diving proved to be instead neurally mediated, they would be important in elucidating the type of innervation involved. Animals subjected to cholinergic blockade produced by injection of 0.2 mg/kg body weight of atropine sulphate into the femoral vein were both artificially ventilated and submerged in diving experiments identical to those in control turtles. Tidal ventilation or step-wise volume changes with any gas mixture, or deflation of the lung produced by opening the tap to the atmosphere produced no changes whatsoever in either heart rate or left pulmonary artery flow in atropinized turtles. In Figs 4 and 5 are also presented data from two atropinized turtles during involuntary descent and ascent. Intrapulmonary pressure closely followed that of non-atropinized animals, but cholinergic blockade obviated the attendant changes in heart rate and pulmonary perfusion.

DISCUSSION

A coupling of ventilatory and circulatory events has been demonstrated to occur in gills of fishes (Satchell, 1960, 1971), airbreathing organs of fishes and lungfishes (Johansen, 1970) and the lungs of all tetrapods (Shelton, 1970; White, 1976; Jones & Johansen, 1972;

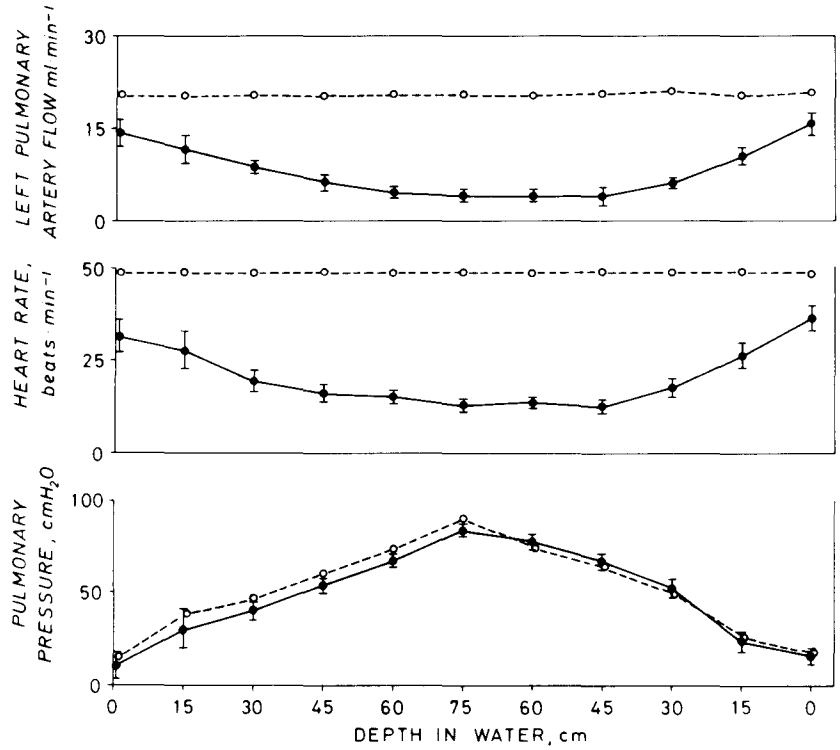

Fig. 4. Changes in heart rate and left pulmonary blood flow in *Pseudemys* in relation to altered intrapulmonary pressure brought about by experimental dives to graded depths. Solid symbols represent average values ± 1 S.D., $n = 5$. Open symbols and broken lines show changes in the same parameters after atropinization.

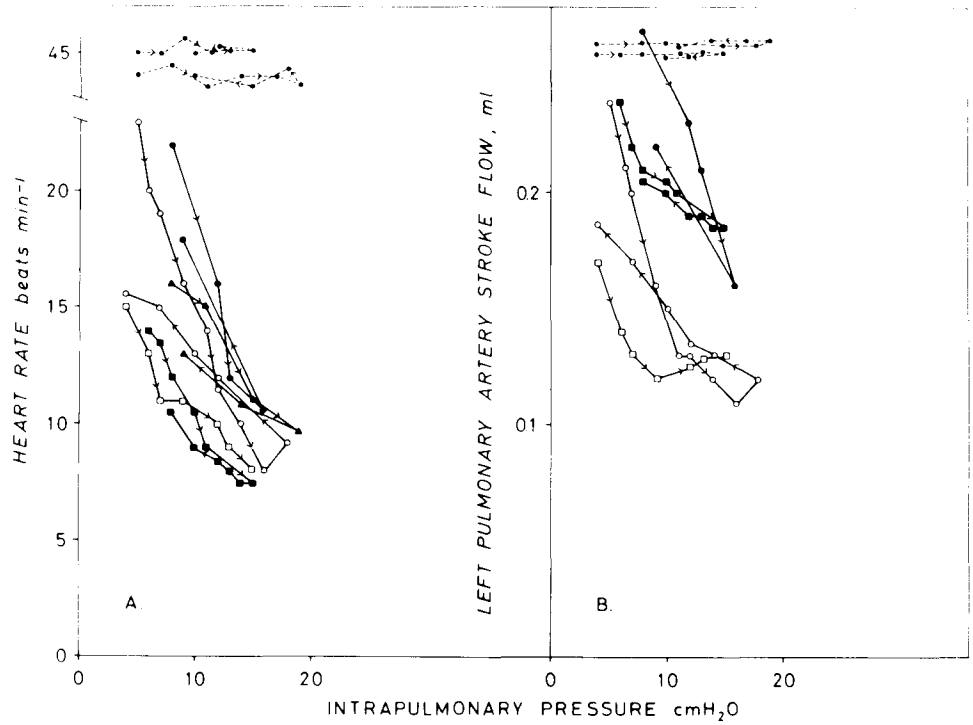

Fig. 5. Heart rate and left pulmonary artery stroke flow in relation to intrapulmonary pressure during descents and ascents of artificial dives of *Pseudemys*. Smaller symbols on top show changes after atropinization.

McCready *et al.*, 1966). The functional significance of such coupling lies in matching the convective volumes of respiratory gases in water or air externally with those in the blood internally. Airbreathing and lung ventilation in vertebrates except birds and mammals are often periodic, and periods of apnoea are separated by single breathing cycles or bursts of repetitive ventilations. For animals practising periodic breathing ventilation–circulation coupling must have particular significance for efficient gas exchange.

Pseudemys scripta studied presently exhibits a distinctly periodic breathing, both in relation to voluntary diving and when it is removed from water (Burggren, 1975). Our results confirm earlier studies by demonstrating a profound change in heart rate and blood flow to the lung during voluntary breathing (White & Ross, 1966; Johansen *et al.*, 1970; Shelton & Burggren, 1976).

The present investigation has demonstrated that artificially produced changes in lung volume and pressure can to a large extent reproduce the response in heart rate and lung perfusion which occurs at the resumption of normal voluntary breathing following apnoea. Coordination of the ventilatory and circulatory processes in the turtle lung under the described conditions is hence governed by peripheral stimuli acting on the lung itself or on receptors sensing blood borne stimuli. The appropriate stimulus could be mechanical or chemical, but our results exclude that the composition of the gas used to inflate the lungs exerts any significant effect on the rapidly developing responses. A gradual longer-termed chemoreceptor influence on heart rate and blood flow distribution during breathholding is likely, but has not been investigated in this study.

Our data show that a change in the state of pulmonary stretch or volume is the eliciting stimulus to the marked cardiovascular changes recorded in *Pseudemys*. That the efferent neural pathway for this response is cholinergic, most certainly vagal, is clear from the atropinization experiments. That cholinergic nerves are without importance in determining the compliance of the lung is apparent from the similarity in the intrapulmonary pressure changes during gradual immersion to equal depths before and after atropinization (Fig. 4).

Pulmonary inflation receptors have been described for all tetrapods and current knowledge on the subject has recently been reviewed by Paintal (1973). In the mammal the responses of the receptors were mapped by Adrian (1933) and their natural stimulus appeared to be the volume of air entering the lungs on inspiration, although Davis *et al.* (1956 suggest that in the mammal the excitatory stimulus is more closely linked with the transpulmonary pressure change rather than with the actual volume of lung gas. The present experiments in *Pseudemys* suggest that restoration in intrapulmonary volume rather than pressure readjusts cardiovascular performance to initial values.

Conclusive proof of a receptor function based on volume change must depend on actual measurement of lung volume. This was not done in our study but the supposition that volume change may be the eliciting stimulus receives support from Figs 3 and 4. On the one hand, opening the tap on the cannula or withdrawing gas from the lung decreased both intrapulmonary pressure and lung volume, and a decrease in heart rate and pulmonary blood flow developed. However, increased hydrostatic pressures during

... diving resulted in an intrapulmonary pressure increase but a lung volume decrease due to the compressing force. Heart and pulmonary blood flow were still diminished. Hence, in both types of experiment a decrease in lung volume was accompanied by a decrease in heart rate, while intrapulmonary pressure changed in opposite directions. A sensing system for lung volume rather than intrapulmonary pressure may be of more direct benefit to a diving animal that commonly utilizes active changes in lung volume to adjust buoyancy over a wide range of intrapulmonary pressures (Milsom & Johansen, 1975).

Pulmonary stretch receptors in mammals are known to mediate increases in heart rate and reductions in peripheral vascular resistance. However, their influence over cardiovascular function in most adult mammals is considered relatively minor, and is mainly concerned with regulation of tidal volume and respiratory frequency (see Paintal, 1973, for review).

In the lower tetrapods and especially in aquatic forms among amphibians and reptiles the inflation reflexes take on much greater importance. The intermittent breathing in these animals calls for regulatory mechanisms to insure a coupling of cardiovascular function to the breathing activity. Efficient use of oxygen stores during diving and above all a recovery and quick repletion following dives will crucially depend on such coordination. The state of pulmonary stretch is also likely to give diving reptiles and amphibians information as to the rate of O_2 depletion from the lungs since CO_2 elimination occurs to a great extent directly to the water, resulting in a decrease in lung volume in proportion to O_2 depletion during breathholds (Standaert & Johansen, 1974).

Our results show that the diving turtle ascending or descending vertically through the water column will experience heart rate and pulmonary blood flow changes tuned to its depth and consequent compression of its lungs. This response may serve to explain earlier recordings of changes in heart rate and blood flow in anticipation of breathing during ascent (Johansen *et al.*, 1970). The findings of the present investigation also require consideration in future studies of the changes in ambient hydrostatic pressure which a reptile is subjected to during diving.

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