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A QUANTITATIVE ANALYSIS OF VENTILATION TACHYCARDIA AND ITS CONTROL IN TWO CHELONIANS, *PSEUDEMYS SCRIPTA* AND *TESTUDO GRAECA*

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SUMMARY

1. In both the turtle, *Pseudemys scripta*, and the tortoise, *Testudo graeca*, lung ventilation is closely accompanied by a tachycardia of predictable magnitude and duration.

2. Efferent vagal activity progressively decreases as heart rate increases with the onset of lung ventilation. Atropine increases heart rate during apnoea to those levels observed during prolonged breathing series when vagal tone is negligible. β-adrenergic blockade of the heart does not affect the development, duration or magnitude of ventilation tachycardia. It is thus concluded that heart rate change during chelonian lung ventilation is mediated solely by alterations in vagal tone.

3. Peripheral sensory reflexes involving pulmonary stretch receptors, arterial chemoreceptors and baroreceptors, and receptors stimulated by water immersion do not affect heart rate during breathing. It is suggested that ventilation tachycardia in these chelonians is the result of the spread of activity between the respiratory and cardiac centres of the medulla.

INTRODUCTION

The basic pattern of lung ventilation of many aquatic and terrestrial reptiles consists of periods of apnoea of variable duration which are interrupted by shorter intervals of active inspiration and expiration. Most intermittently ventilating vertebrates utilize distinctive circulatory adjustments during prolonged periods of apnoea to both protect those tissues least resistant to hypoxic damage and to conserve cardiac energy expenditure (see Andersen, 1966; Angell James & Daly, 1972, for reviews). Nervous and cardiac tissues, which are particularly susceptible to hypoxia in other vertebrates, are surprisingly resistant to the damaging affects of hypoxia and even anoxia during enforced apnoea in reptiles. This has been attributed to the potential of reptilian tissues to sustain levels of anaerobic metabolism unparalleled among the vertebrates (Belkin, 1963; Robin *et al.* 1964; Belkin, 1968; Penney, 1974). However, during normal voluntary apnoea the tissues of reptiles apparently continue to metabolize aerobically (Baldwin, 1926; Belkin, 1963; 1968) and circulatory adjustments similar to those evident in other intermittently breathing vertebrates,

including decreased systemic blood flow due to peripheral vasoconstriction, marked reduction of pulmonary blood flow, and a profound bradycardia, often develop in reptiles as apnoea progresses (Andersen, 1961; Belkin, 1964; Millen *et al.* 1964; White & Ross, 1966; Millard & Johansen, 1974). The present paper describes changes in heart rate which occur during intermittent lung ventilation and voluntary apnoea in two chelonian reptiles; the turtle *Pseudemys scripta*, and the tortoise *Testudo graeca*. Although a number of observations suggest that in reptiles this chronotropic cardiac reflex is neurally mediated (Belkin, 1964; White, 1969; Gaunt & Gans, 1970; White, 1970), few detailed studies of heart rate regulation have been carried out on conscious, intact reptiles. This investigation was, therefore, undertaken to determine what neural mechanisms and physiological factors control and influence heart rate during lung ventilation and non-enforced apnoea in chelonians.

MATERIALS AND METHODS

Experiments were performed on 24 individuals of *Pseudemys scripta* and 12 of *Testudo graeca* weighing between 0·95 and 1·80 kg. Apparently healthy animals obtained from commercial suppliers were used for this study. All experiments were performed at 16–18 °C.

To record an electrocardiogram, three 1·5 cm square brass plate electrodes coated with electrode gel were fastened with elastic bands along the midventral line of the plastron over the heart at points approximately 1 cm apart. The most posterior plate served to ground the animal. The electrodes were connected to an a.c. amplifier whose output was displayed on a Sanborn 966 six-channel pen recorder writing on rectangular co-ordinates. The QRS complex of the e.c.g. wave form triggered a Devices 2751 Instantaneous Ratemeter which was modified to compute rate over a signal frequency range of 4–50 cycles/min.

To monitor ventilation frequency, a large rubber sleeve tapering at one end to a small orifice fitted with a pneumotach screen was pulled over the anterior third of the animal's carapace. The head and forelimbs thus became enclosed within an airtight chamber whose only opening to the atmosphere was via the pneumotach lumen. Although forelimb movement was somewhat restrained the animal was free to extend its head for some distance out of the shell. Ventilation of the lungs caused displacement of gas within the chamber. The resultant momentary pressure differential between the two sides of the pneumotach membrane was monitored by a Sanborn 270 B3 air transducer and displayed on the recorder. Because the bases of the front legs expanded outwards at inspiration and as they were included within the pneumotach chamber, the apparatus did not give an accurate measure of ventilation volume but was adequate to indicate breathing frequency. The gas content of the inspired atmosphere was controlled by passing a steady flow of the appropriate gas into a cannula which was fastened into the pneumotach chamber. The gas escaped through the pneumotach, producing a stable shift of the baseline in the respiratory recordings which was larger than the normal inspiratory excursion but which could be eliminated electrically. A flow of approximately 1 l/min was sufficient to maintain the desired experimental atmosphere even during prolonged and rapid lung ventilation. Each turtle or tortoise, fitted with e.c.g. electrodes and pneumotach chamber, was

suspended in a horizontal plane from an overhead bar by elastic bands passing around its carapace. This denied the animal a substrate for locomotion, yet did not enforce an unnatural limb or body posture. Animals rarely struggled when suspended in this fashion and usually remained quiet for several hours while the experiment proceeded. The parameters measured (respiratory frequency, apnoea duration, and heart rate) using this apparatus showed no significant difference from those recorded in completely unrestrained animals using other techniques (Burggren, in preparation).

Lung cannulation was performed on animals after anaesthesia was induced by 15 min of exposure to Halothane vapour in a dessicator (after Gans & Hughes, 1967). A hole of 1 cm diameter was drilled in the carapace over the anterior lobe of either the right or left lung. The lung epithelium was drawn to the surface of the hole with forceps and perforated with a needle. The tip of a polyethylene cannula (pp90) was passed through the perforation into the lumen of the anterior lobe and the cannula was sewn into place in the lung wall. The hole in the carapace, with the cannula passing out through it, was then sealed with rapidly-setting cement. Chronically-implanted cannulae usually remained open for several days and dissection of animals killed after experimentation revealed little or no pulmonary damage in the area of cannulation.

Cannulation of the femoral vein for the injection of drugs into the vascular system was also performed under Halothane anaesthesia. An incision was made on the dorsal surface of a hind limb, the femoral vein exposed and occlusively cannulated in a downstream direction. The cannula (pp90), which was filled with heparinized saline, was led out of the incision and the wound closed with interrupted sutures. Experiments were performed 24–36 h after surgery. Drugs injected were L-adrenaline bitartrate, DL-propranolol hydrochloride, and atropine sulphate.

Electrophysiological recordings were made from the exposed vagus nerves in the neck of unanaesthetized *Pseudemys scripta*. Turtles which had been refrigerated at 1 °C for 12–15 h to induce cold torpor were restrained ventral side up. The head was extended and held with neck outstretched by a non-constrictive yoke that was placed around the base of the skull. The single finger of a rubber glove fitted with the pneumotach to monitor lung ventilation was placed over the head of the turtle. The right or left vagus nerve was exposed at the base of the neck and carefully separated from the adhering carotid artery and sympathetic trunk. The turtles were then allowed to warm to room temperature (16–18 °C) and regain consciousness. There was no significant difference in the intervals between breathing series and the number of breaths in a breathing series in intact animals and those prepared for neurophysiological recording from a vagus nerve. Small fibres were carefully separated from the intact vagus nerve or from its distal or medial end after severing and were laid over two silver hook electrodes. To prevent their desiccation, the exposed vagus trunk and the nerve fibres on the electrodes were covered with liquid paraffin. Action potentials were amplified with an a.c. amplifier whose continuous output was stored on magnetic tape for later analysis and photography.

Table 1. *Apnoea duration and heart rate during apnoea and lung ventilation in* Pseudemys scripta *and* Testudo graeca

(Data presented are mean values ± standard deviation.)

Species	*N*	Apneoa duration (min)	Number breaths/breathing series	Heart rate during apnoea (beats/min)	Heart rate during lung ventilation (beats/min)
Pseudemys scripta	16	3·8 ± 4·0	7·1 ± 6·8	7·8 ± 2·0	17·9 ± 4·0
Testudo graeca	10	0·4 ± 0·3	—	8·2 ± 1·0	15·1 ± 1·5

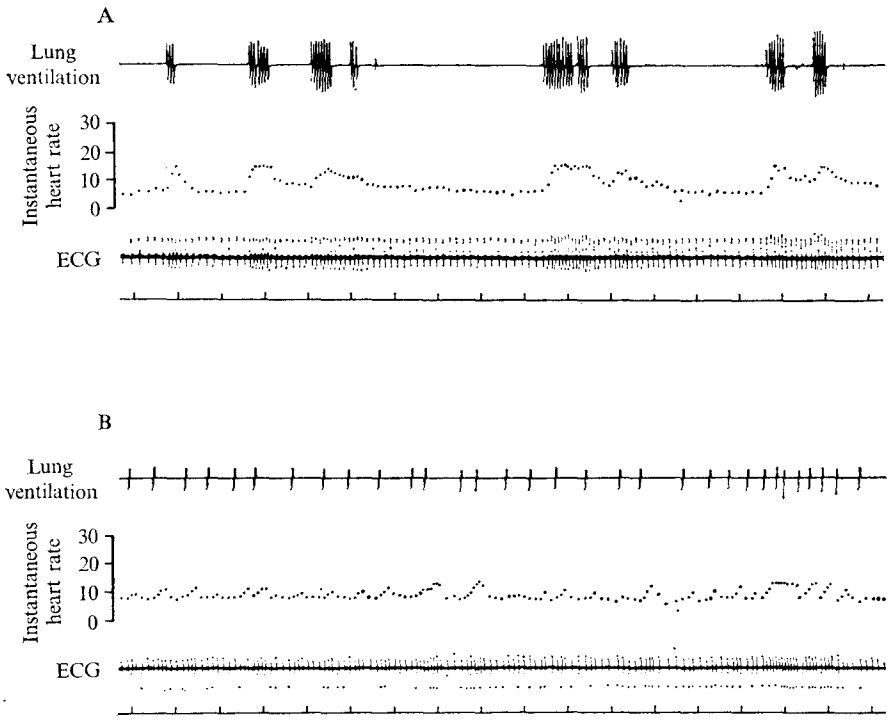

Fig. 1. Representative patterns of lung ventilation (vertical deflections in upper trace) and accompanying heart rate changes in *Pseudemys scripta* (A) and *Testudo graeca* (B).

RESULTS

Breathing patterns

The breathing pattern in *Pseudemys* was characterized by relatively long periods of apnoea between breathing series which were made up of a number of breathing cycles (Table 1, Fig. 1A). The periods of apnoea in *Testudo*, on the other hand, were much shorter and were punctuated in most instances by only a single breath (Fig. 1B). As the standard deviations in Table 1 show, there is a wide variation from the mean duration of apnoea in both species. The intervals between breathing series ranged from a few seconds in both species, to 59 min in *Pseudemys* and 9 min in *Testudo*. In both species the breathing activity almost always ended on inspiration and began again after a period of apnoea with an expiration of gas from the lungs.

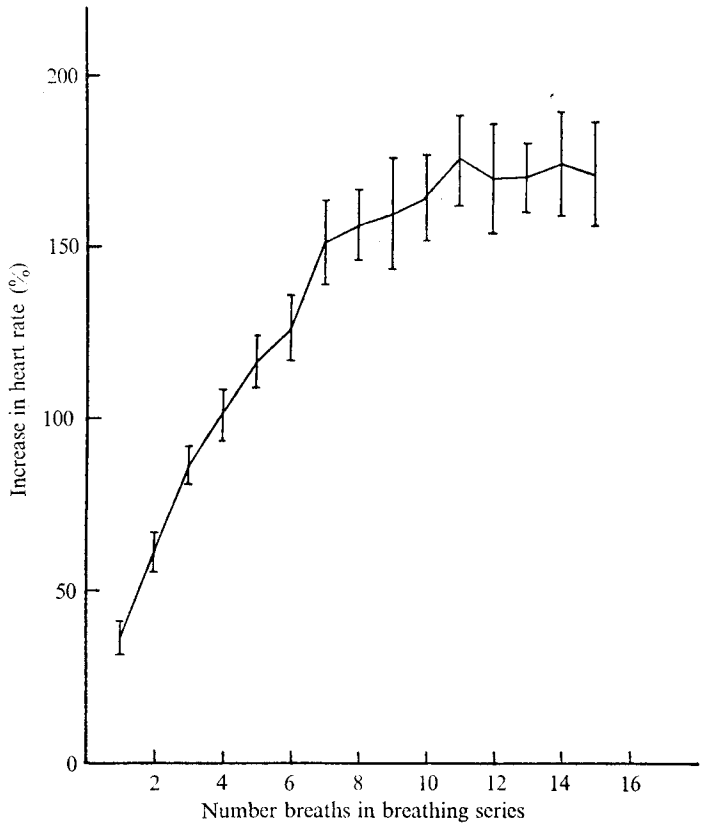

Fig. 2. Mean increase in heart rate (±S.E.) of *Pseudemys scripta* with increasing size of breathing series (N = 16).

Heart rate during apnoea and breathing

Difficulty arises in assigning a 'normal' rate of heart beat to an animal whose heart rate is subject to great variation depending on physiological and behavioural state. That heart rate is low during apnoea ('diving bradycardia' of White, 1969) and high during lung ventilation ('ventilation tachycardia' of Belkin, 1964; Gaunt & Gans, 1970) has been well established in the chelonians, although not without contention as to what the phenomenon should be termed. Breathing in intact undisturbed *Pseudemys* and *Testudo* normally occupies less than 15% of their total activity and as a consequence the overall mean heart rate closely approximates to the heart rate during apnoea rather than during breathing. In the present study the increase in heart rate above that observed during undisturbed apnoea is referred to as ventilation tachycardia with the rather arbitrary justification of convenience.

The mean heart rate during apnoea for both *Pseudemys* and *Testudo* was approximately 8 beats/min (Table 1). Although there was some individual variation, heart rate during periods of apnoea was relatively constant in any one individual. Occasionally rhythmic oscillations in heart rate of an amplitude of 2–3 beats/min were observed due to a slight elongation of every third or fourth diastole. However, the marked groupings of several rapid heart beats followed by a long diastolic period which has

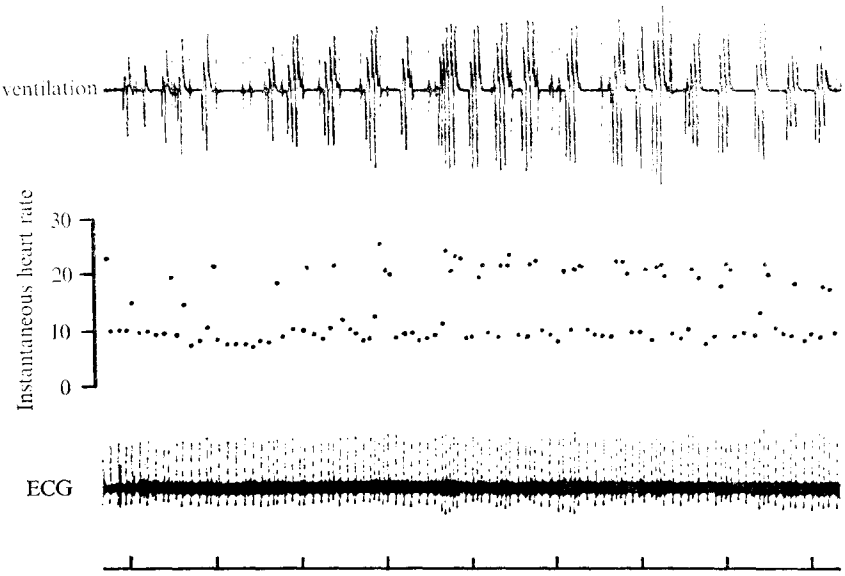

Fig. 3. Heart rate changes during rapid lung ventilation (vertical deflections in upper trace) in *Pseudemys scripta*. Time marker in minutes.

been reported to occur in other reptiles during apnoea (Johansen, 1959; White, 1970) was not seen in either *Pseudemys* or *Testudo*.

The mean heart rate in *Pseudemys* during lung ventilation was approximately 18 beats/min, an increase of 130% above the mean pre-ventilation heart rate (Table 1). Heart rate invariably increased within 1–2 beats of the first ventilatory movement (Fig. 1). In *Pseudemys* the rate continued to rise as a breathing series progressed, reaching a maximum of 18–25 beats/min after some 8–12 breaths (Fig. 2). In breathing series of more than 12 breaths this level was usually maintained until ventilation stopped, though on a few occasions there was a slow decline from the peak rate. When lung ventilation stopped the heart rate dropped rapidly, regardless of whether the breathing series had been long enough for the peak rate to be developed. Heart rate in *Pseudemys* usually returned to the pre-ventilation level within one or two heart beats of the final respiratory movement (Fig. 1 A). The mean heart rate in *Testudo* during lung ventilation was approximately 15 beats/min, an increase of 85% above the mean pre-ventilation heart rate (Table 1). In *Testudo* the maximum heart rate also did not develop immediately, but rose during several heart beats following the single breathing cycle (Fig. 1 B). If the periods of apnoea intervening between breaths were very short then there was often a cumulative effect on heart rate though it seldom attained the levels seen in *Pseudemys*. In both species the highest rate recorded during normal breathing at rest (18–25 beats/min) was often considerably less than the values observed during locomotor activity. Breathing series after exceptionally prolonged apnoea could also cause heart rate to increase above the normal maximum.

An effect on heart rate was usually seen in both species no matter how short the period of apnoea preceding a breath. As Fig. 3 shows, a pause in lung ventilation of less than 10 sec was accompanied by a decrease in heart rate which was quickly

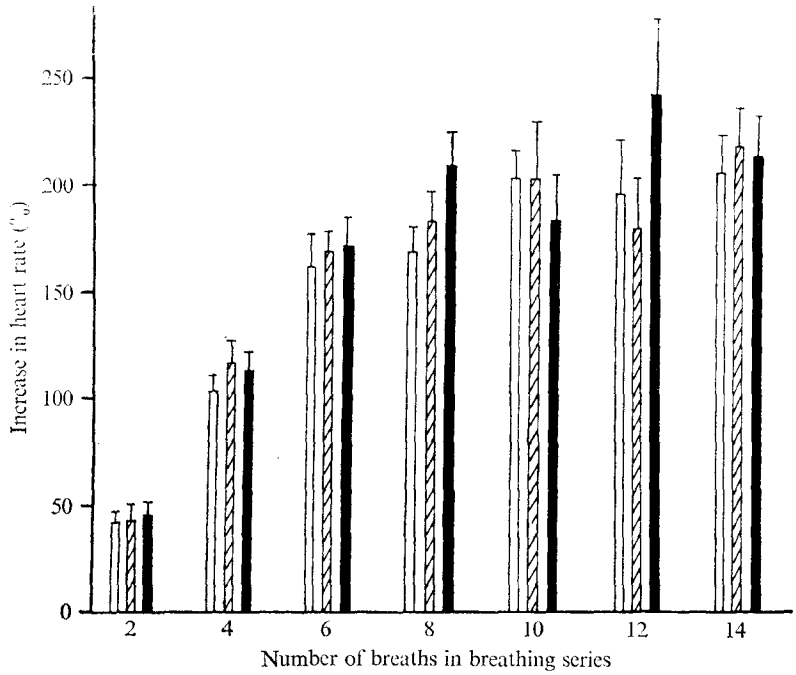

Fig. 4. Mean increases in heart rates (±S.E.) with increasing size of breathing series in *Pseudemys scripta* during lung ventilation in air (open bars), 100% N_2 (diagonals) and 10% CO_2 (solid bars) (N = 7).

reversed at the onset of the next breathing movement. In some animals the heart rate occasionally increased before lung ventilation started, suggesting that there is more than a simple causal relationship between ventilation and the tachycardia.

Effect of inspired gas composition on heart rate

To determine whether gas tensions in the lungs and/or 'breath by breath' alterations in the gas tension of the blood influenced ventilation tachycardia, changes in heart rate during the initial breaths taken in an atmosphere of 100% nitrogen or a 10% CO_2–90% air mixture were recorded in 10 *Pseudemys*. In all of these turtles tachycardia developed during lung ventilation in both the anoxic and the hypercapnic environments. There were no significant differences ($P > 0{\cdot}10$) between the tachycardia observed during air breathing and that which developed during nitrogen or carbon dioxide inhalation (Fig. 4). In each experimental situation a bradycardia was resumed after lung ventilation stopped. Although the initial inhalation of 100% N_2 or 10% CO_2 had no effect on the normal development of ventilation tachycardia, prolonged exposure (15–20 breaths) to an anoxic or hypercapnic environment usually led to the development of a more pronounced tachycardia which was not reversed during periods of apnoea and which persisted until the turtles were allowed to resume air breathing. Because of this effect of prolonged hypoxic or hypercapnic exposure on heart rate, as well as on ventilation frequency, all turtles were allowed at least three subsequent breathing series in air before further experiments were performed.

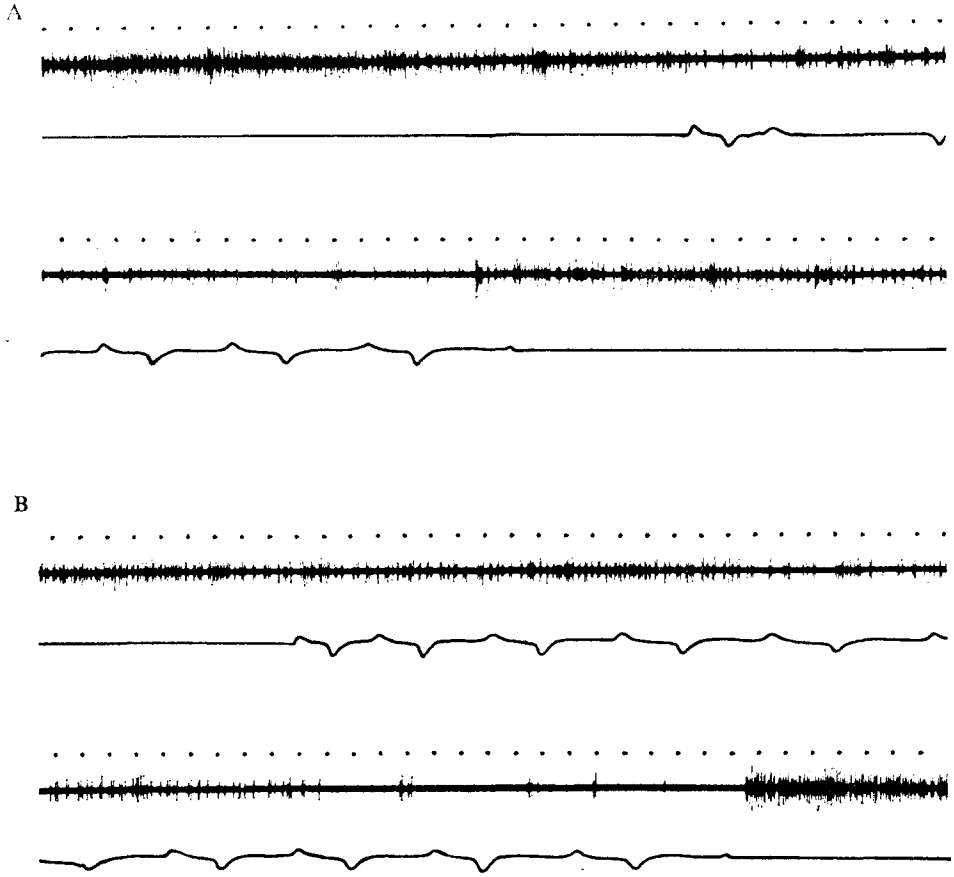

Fig. 5. Efferent vagal activity in an individual *Pseudemys scripta* during apnoea and during representative breathing series of less than 5 breaths (A), and more than 10 breaths (B). Time marker (upper trace) in seconds. Lung expirations are upward deflections in pneumotach record (bottom trace).

Effect of lung inflation on heart rate

To investigate the effect of lung inflation and deflation *per se* on the rate of heart beat, experiments were performed on 4 individual *Pseudemys* with chronically implanted lung cannulae. Each turtle's lungs could be inflated or deflated by the injection or withdrawal of gas from a syringe connected to the cannula. In all turtles it was found that either the injection or withdrawal of 5–30 ml of air had no effect whatsoever on heart rate. In one experiment, the lungs were inflated during a period of apnoea with 20 ml of air for approximately 2 min during which time no change in heart rate was observed. The turtle then spontaneously began ventilation of its lungs, beginning with expiration of the injected air, and the conventional tachycardia was immediately observed. Rhythmic artificial ventilation of the lungs at a frequency of 10, 15 and 20 cyc/min and with a 'tidal volume' of 5–30 ml of air had no effect on heart rate. The aforementioned experiments were all repeated using 100% N_2, 100% O_2 and 10% CO_2 in air. Again no changes in heart rate were observed, apart from those previously attributed to the long term effects of lung ventilation with N_2 and CO_2.

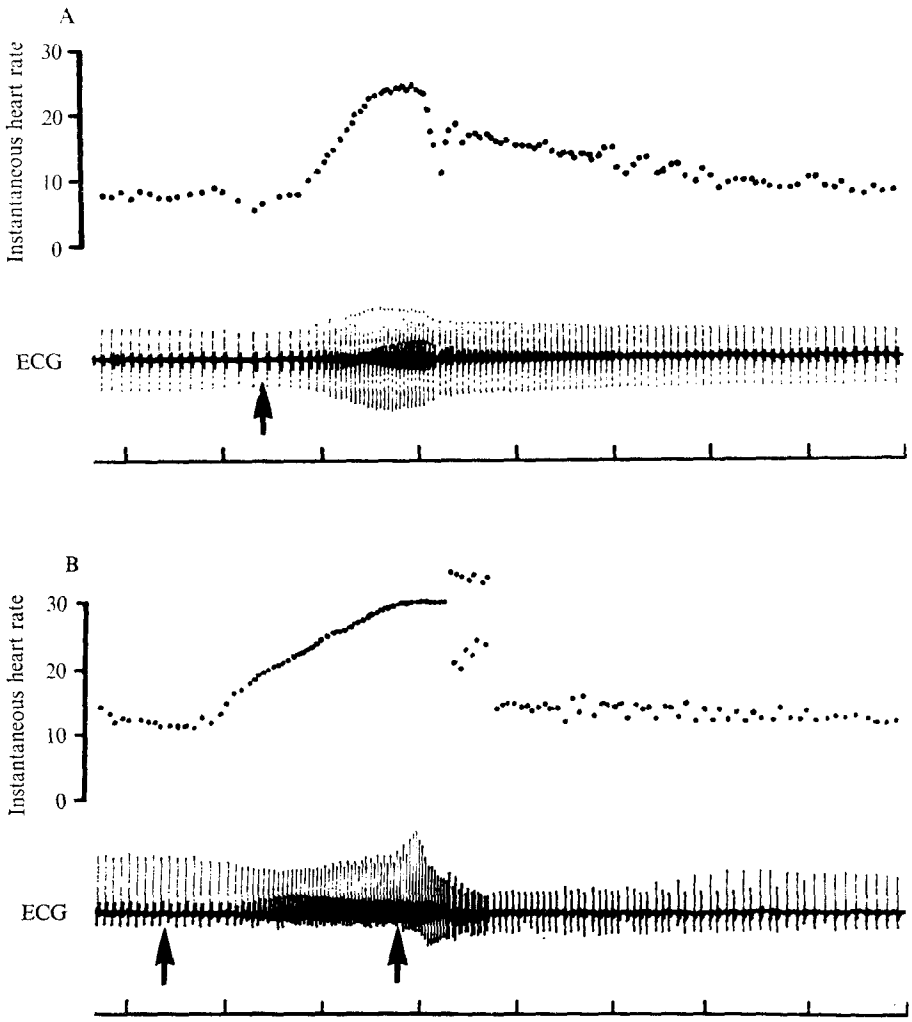

Fig. 6. Effect of adrenaline on heart rate in *Testudo graeca* (A). Injection of 5 μg adrenaline at arrow. Time marker in minutes. Effect of propranolol on adrenaline-induced tachycardia in *Testudo graeca* (B). 5 μg adrenaline injected at first arrow, followed at second arrow by the injection of 1·0 mg of propanolol. Time marker in minutes.

Electrophysiological recordings

Efferent activity was recorded from the proximal end of severed nerve fibres which were detached from the main vagus trunk in 4 individual *Pseudemys*. Several different units were consistently observed to be discharging at a relatively uniform rate during periods of apnoea. On most occasions efferent discharge began to decrease with the onset of lung ventilation and progressively fewer spikes were observed with each subsequent breath (Fig. 5A, B). Towards the end of a long ventilation series vagal discharge was almost completely absent (Fig. 5B). Immediately upon cessation of the last ventilation cycle efferent vagal discharge returned, often with a momentary burst at a frequency greater than the pre-ventilation frequency. Rarely, efferent

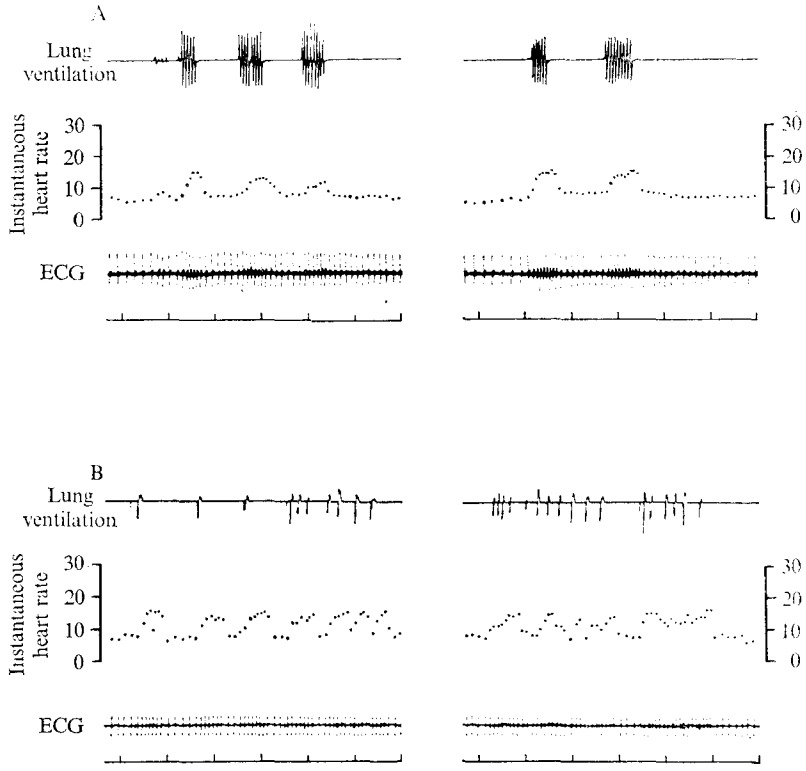

Fig. 7. Lung ventilation and accompanying heart rate changes in *Pseudemys scripta* (A) and *Testudo graeca* (B) before (left) and 5 min after (right) β-adrenergic blockade by propranolol. Time marker in minutes.

activity at pre-ventilation frequencies was briefly resumed during a breathing series.

Pharmacology of ventilation tachycardia

To determine the relative contributions of sympathetic adrenergic and parasympathetic cholinergic fibres in producing ventilation tachycardia, experiments using atropine, adrenaline, and propranolol, a β-adrenergic blocking agent, were performed on 6 turtles and 2 tortoises. (It has been demonstrated by Van Harn, Emaus & Meester (1973) that adrenergic responses of the turtle heart are mediated through β-receptors rather than α-receptors, so only propranolol was used for adrenergic blockade.) Injection of adrenalin (5 μg) into the vascular system via the femoral vein resulted in a pronounced (30–40 beats/min) and often prolonged tachycardia (Fig. 6A). The injection of propranolol produced an action antagonistic to the action of adrenaline (Fig. 6B). The experimental protocol in adrenergic blocking experiments was first to observe and quantify the conventional ventilation tachycardia. A brief tachycardia was then induced by the injection of 5 μg of adrenalin to confirm the presence of active adrenergic receptors influencing heart rate. Blockade of β-adrenergic receptors after the injection of 1·0 mg propranolol was then confirmed by the failure of subsequent adrenalin injection (10 μg) to produce heart rate changes. Lung ventilation, which was apparently unimpaired in all animals suffering

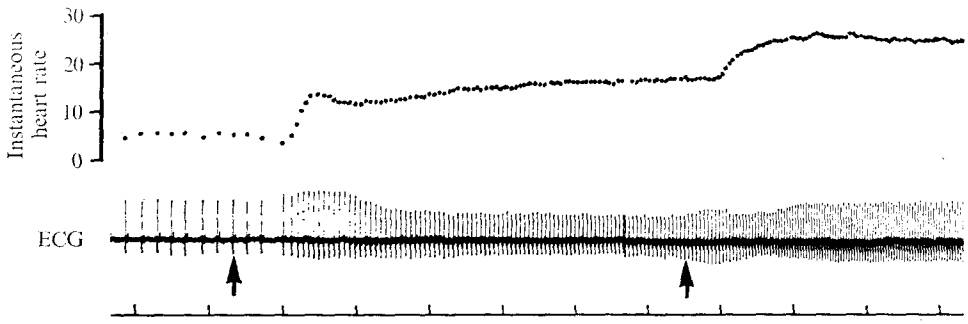

Fig. 8. Effect of adrenaline injection into atropinized *Pseudemys scripta*. At first arrow 1 mg atropine was administered followed at second arrow by 5 μg adrenaline. Time marker in minutes.

β-adrenergic blockade by propranolol, induced a ventilation tachycardia which was identical, both qualitatively and quantitatively, to the ventilation tachycardia observed in these animals before adrenergic blockade (Fig. 7). The additional tachycardia elicited by locomotor activity was never observed in those animals whose β-receptors were blocked by propranolol.

The injection of 1 mg of atropine produced initially one or two markedly long diastoles which were followed by a gradual increase in heart rate with eventual stabilization at approximately 18–25 beats/min in both *Pseudemys* and *Testudo* (Fig. 8). Heart rate did not decrease from this level between subsequent breathing series nor did relatively long breathing series increase it above this level in atropinized animals. The injection of adrenaline into an atropinized turtle invariably induced a further increase in heart rate up to approximately 30–35 beats/min (Fig. 8).

DISCUSSION

Both *Pseudemys* and *Testudo* show a relatively exact and predictable tachycardia which is closely related to lung ventilation. Differences in ventilation patterns between the terrestrial tortoise (which breathes fairly regularly with single breaths) and the semi-aquatic turtle (which, even in air, breathes in short bursts separated by long and more irregular intervals of apnoea) are reflected in differences in the tachycardia patterns. The effect of intermittent breathing, together with the accompanying cardiovascular adjustments including tachycardia, increased cardiac output and both pulmonary and systemic vasodilation (White & Ross, 1966), is to cause fluctuations in the alveolar and arterial concentrations of the respiratory gases. The extent of these fluctuations is related, as might be expected, to the duration of the periods of apnoea. Because the intervals between breathing are much shorter in *Testudo*, the extent of the cardiovascular responses required to alleviate tissue hypoxia and hypercapnia after a normal period of apnoea in *Testudo* will be much less than in *Pseudemys*, as is reflected in the consistently smaller magnitude of the tortoise's ventilation tachycardia. In *Pseudemys* the cardiovascular responses including ventilation tachycardia required after a short period of apnoea will be correspondingly less than after a longer period of apnoea. It would appear that in the turtle the gradual

increase in heart rate with progressively longer breathing series represents a graded cardiac response which enables a delicate balance to be maintained between the minimization of cardiac energy expenditure and the demand for increased systemic and pulmonary vascular bed perfusion. Similar profiles of heart rate changes during breathing have been reported in other terrestrial and aquatic reptiles including *Pseudemys concinna* (Belkin, 1964), *Chelydra serpentina* (Gaunt & Gans, 1970), and *Tropidonotus natrix* (Johansen, 1959), although in *Caiman sclerops* and *Alligator mississipiensis* there is a less precise relationship between heart and lung ventilation (Andersen, 1961; Huggins, Hoff & Pēna, 1970).

In spite of both qualitative and quantitive differences in ventilation tachycardia, its mechanisms of control in the two chelonians appears to be same. Pharmacological evidence indicates that ventilation tachycardia is produced entirely by release of vagal inhibition, and although efferent vagal activity recorded in the neck of *Pseudemys* could be destined for other organs, the very close correlation of recorded vagal tone with the development and extent of cholinergic cardiac inhibition suggests that the observed activity may be representative of that in the cardiac branch of the vagus. Vagal release is complete during the more prolonged breathing series in *Pseudemys*, thus enabling heart rate to reach the same elevated levels as that recorded in atropinized or vagotomized animals. In *Testudo*, however, such a degree of vagal release is not often observed. In both species locomotor activity may cause the heart rate to go above the rate seen in atropinized animals due to sympathetic activity.

The experiments to determine which of the many changes in physiological state associated with lung ventilation were significant in influencing the development of ventilation tachycardia were confined for the most part to *Pseudemys*. Since heart changes during lung ventilation and apnoea occur in the turtle even when permanently in air, it would appear that stimulation by water immersion of the trigeminal nerve reflexes, which have been implicated in diving responses of other vertebrates (Angell James & Daly, 1972), is not essential in the development of ventilation tachycardia. Although little is known of arterial chemoreceptor function in reptiles, it seems unlikely that the initiation, maintenance, or termination of ventilation tachycardia can be attributed to sensory input from arterial chemoreceptors responding to 'breath to breath' changes in blood O_2, CO_2, or pH. Indeed, the initial inhalation of 100% N_2 or 10% CO_2 in no way alters the tachycardia associated with lung ventilation in *Pseudemys*. Changes in heart rate with lung ventilation in both *Pseudemys* and *Testudo* appear to be too rapid to be mediated by chemoreceptor reflexes. However, the more profound tachycardia which often develops after 15–20 breaths in these highly experimental conditions probably does reflect cardio-vascular reflexes involving arterial chemoreceptors. It has been suggested that these chemoreceptors exist at the bifurcation of the common carotid artery of *Pseudemys scripta* (Frankel *et al.* 1969). Although White (1970) concluded that the mechanical state of inflation of the lungs or thorax is of some importance in initiating cardiovascular reflexes in the alligator, the present study has been unable to establish any relationship between the state of lung inflation and heart rate in the turtle. There is a substantial body of evidence demonstrating the existence of pulmonary stretch receptors in chelonians (Coombs, 1920; Carlson & Luckhardt, 1920; Frankel *et al.* 1969) but they seem to have no importance in the development of ventilation tachycardia. Arterial baro-

receptors may be involved in heart rate reflexes in chelonians, but preliminary measurements of blood pressure and heart rate have not revealed any clear relationship between these parameters.

There seems to be little reason, therefore, to implicate any of the more obvious sensory pathways in the direct co-ordination of the vagally mediated ventilation tachycardia in chelonians, as Huggins *et al.* (1970) have similarly suggested for crocodilians. It appears likely that a spread of excitatory activity within the medulla between the centres controlling breathing and heart functions rather than the action of a peripheral sensory reflex may effect cardiac vagal tone during lung ventilation in *Pseudemys* and *Testudo*.

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