Vagal control of pulmonary vascular resistance in the turtle *Chrysemys scripta*[1]

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Received September 16, 1976

MILSOM, W. K., B. L. LANGILLE, and D. R. JONES. 1977. Vagal control of pulmonary vascular resistance in the turtle *Chrysemys scripta*. Can. J. Zool. **55**: 359–367.

We have directly examined the control of pulmonary vascular resistance in the turtle *Chrysemys scripta* to determine the way in which pulmonary vasoregulation is achieved. The pulmonary circulation of the turtle *Chrysemys scripta* receives a strong excitatory cholinergic innervation from the vagus nerve. The major site of vasoconstrictor activity is in the extrinsic pulmonary artery proximal to the lung with only weak constrictor activity evident in the intrinsic arteries and arterioles within the lung parenchyma. No cholinergic innervation is evident in the segment of the extrinsic pulmonary artery proximal to the origin of the arterial ligament (ligamentum Botalli) and all vagally induced changes in flow resistance reside in the much narrower segment distal to this site. Vagal stimulation in an intact preparation produces sufficient constriction in the distal segment of extrinsic pulmonary artery to totally occlude pulmonary flow. The pulmonary arteries appear to be devoid of sympathetic innervation.

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On a étudié, par méthode directe, le contrôle de la résistance vasculaire pulmonaire chez la tortue *Chrysemys scripta*. La circulation pulmonaire, chez cette tortue, reçoit du nerf vague une innervation cholinergique excitatoire très forte. L'activité vasoconstrictrice a son principal siège dans l'artère pulmonaire extrinsèque près du poumon, alors qu'il semble y avoir peu d'activité constrictrice dans les artères intrinsèques et les artérioles du parenchyme pulmonaire. On n'a pas de preuve de l'existence d'une innervation cholinergique dans le segment de l'artère pulmonaire extrinsèque près de l'origine du ligament artériel (ligamentum Botalli) et tous les changements de résistance à l'écoulement contrôlés par le nerf vague se manifestent dans le segment, très étroit, distal par rapport à ce point. La stimulation par le vague, dans une préparation intacte, produit une constriction suffisante dans le segment distal de l'artère pulmonaire extrinsèque pour enrayer complètement la circulation pulmonaire. Les artères pulmonaires semblent démunies de toute innervation sympathique.

[Traduit par le journal]

Introduction

In almost all vertebrates that have been examined the larger, more compliant pulmonary arteries appear to be the major site of changes in pulmonary vascular tone rather than the smaller arterioles or resistance vessels. Vasoconstrictor control at such a high level in the pulmonary vascular circuit has been demonstrated in the dog (Szidon and Fishman 1969), duck (Aakhus and Johansen 1964), lizard (Berger 1972), tortoise (Berger 1972), and toad (Smith 1976) with the crocodile the only noted exception (Berger 1974). Despite this consistent pattern the neural control of pulmonary vascular resistance shows marked differences in various species. The pulmonary sympathetic nerves provide the excitatory innervation to the pulmonary artery in the dog (Szidon and Fishman 1969) and there is no trace of any vagal or cholinergic innervation. In total contrast, the sympathetic nerves in the lizard (Berger 1972) and toad (Campbell 1971) supply the pulmonary artery with β-adrenergic inhibitory fibres and the vagus provides cholinergic excitatory innervation.

In the turtle and tortoise, no sympathetic innervation to the pulmonary artery has ever been found (Luckhardt and Carlson 1921; Berger 1972) and reports on the effects of sympathomimetic drugs on the pulmonary artery are conflicting. In a series of careful experiments on turtles (*Chrysemys* and *Malaco-clemys*), Luckhardt and Carlson (1921) demonstrated pulmonary vasodilatation in response to small doses of epinephrine and vasoconstriction in response to larger doses. Woodbury

[1]Supported by grants from the National Research Council of Canada and the British Columbia Heart Fund.

and Robertson (1942), also working on turtles (genus not given), deduced from pressure profiles that epinephrine constricted the larger extrinsic pulmonary artery but had no effect on the smaller intrinsic pulmonary arteries. A recent study, however, failed to show any effect of norepinephrine, isoprenaline, or phenylephrine on pulmonary arterial tone in the tortoise (*Chelodina*) (Berger 1972).

Although cholinergic, excitatory innervation from the vagus to the pulmonary artery has been well established in the turtle and tortoise (Krogh 1906; Luckhardt and Carlson 1921; Berger 1972) neither the extent of vagally induced changes in pulmonary vascular resistance nor the precise site where constriction occurs are clear. Studies on another reptile, the lizard (Berger 1972), indicate that the most likely site for vasoconstrictor control is the distal portion of the pulmonary artery extrinsic to the lung. In the turtle, however, this portion of the pulmonary artery has been described as thin walled with a reduced amount of fibrous elastic and muscle tissue (Woodbury and Robertson 1942). Berger (1972) initially described the extrinsic pulmonary artery in the tortoise (*Chelodina*) as weakly contractile and attributed increased pulmonary resistance during vagal stimulation to the intrinsic pulmonary vasculature. In a later, brief series of experiments on two tortoises he illustrated a strong constrictor effect of vagal stimulation on the extrinsic pulmonary artery but did not clearly resolve the discrepancy between these results (Berger 1974).

A knowledge of pulmonary vasoregulation in turtles is important in understanding cardiovascular adaptations to environmental changes, particularly the net right to left vascular shunting that occurs through the incompletely divided ventricles during thermoregulation and submergence (White 1976) and the net left to right vascular shunting that occurs during lung ventilation (Shetton and Burggren 1976). In view of the confusion that exists in the literature, we have reinvestigated the control of pulmonary vascular resistance in the aquatic turtle *Chrysemys scripta*.

Materials and Methods

Turtles (average weight 1.4 kg) were sedated by refrigeration at −5 °C for several hours and then either pithed or, in some instances, decerebrated. A tracheostomy was performed and turtles were tidally ventilated with room air using a constant-volume positive-pressure respiration pump (40 ml, 8 cm H_2O pressure). The vagus nerve below the vagal ganglion was exposed high in the neck on either side of the body, dissected free, and sectioned. The peripheral end was passed through silver ring electrodes shielded in moulded epoxy resin and connected to a Grass S-4 stimulator. On several occasions the cervical sympathetic nerve was prepared in the same manner. The turtles were restrained on their backs and a window was cut into the plastron above the heart and great vessels using a necropsy saw. The pulmonary artery was clamped and sectioned just past the bifurcation of the main pulmonary trunk on the side on which the vagus nerve was exposed and a polyethylene cannula was tied into the peripheral end. Perfusion began immediately using Ringer's solution containing glucose. In 14 preparations a second cannula was inserted with the opening directed centrally into the distal portion of the same pulmonary artery at a point where the artery entered the lung parenchyma and this now isolated segment of pulmonary artery perfused *in situ*. Perfusion was by means of a constant-flow infusion pump (Harvard Apparatus model 901) at flows between 0.38 and 3.8 ml per minute, which generated perfusion pressures between 5 and 15 cm H_2O under control conditions. The pressure in the upstream perfusion cannula was recorded with a Statham P23V pressure transducer and monitored on a Beckman dynograph writing on curvilinear coordinates. Drugs used were acetylcholine chloride (ACh), atropine sulphate, and epinephrine chloride dissolved in distilled water of which 0.1-ml volumes were injected into the perfusion line as close as possible to the preparation. Control injections of 0.1-ml saline were also given.

In four preparations the distal cannula was inserted into the pulmonary vein and the proximal cannula was fed along the pulmonary artery in small steps until it reached the level of the lung hilus and withdrawn again to its point of insertion near the bifurcation of the main pulmonary trunk. This preparation, which included the entire pulmonary circulation on one side of the body, was also perfused *in situ*. The back pressure to infusion was monitored continuously and vagal stimulation was conducted after each step movement of the proximal cannula.

In a further four preparations the pulmonary arteries were neither cannulated nor perfused but were carefully cleared for observation and photographic recording. In these preparations, constriction of the pulmonary arteries could alter blood flow through the pulmonary circuit by shunting through the incompletely divided ventricles. A cannula was placed in the jugular vein, high in the neck, for drug infusion and nerve stimulation was performed as described above.

All experiments were conducted at room temperature (23 °C) and heart rates were recorded with copper electrodes connected to a Tektronix FM122 preamplifier and monitored on the second channel of the Beckman dynograph.

In eight specimens both extrinsic pulmonary arteries were dissected free after experimentation, fixed in 10% buffered formalin, and embedded in paraffin. Serial cross sections (10 μm) were stained with haematoxylin and eosin for light microscopic examination.

Results

Morphology

The pulmonary trunk arises from a residual bulbus cordis (March 1961) and bifurcates about 2 cm from the heart into right and left pulmonary arteries each of which consists of a short proximal segment ($\simeq$2 cm) narrowing at the site of origin of the arterial ligament or ligamentum Botalli (the remnant of the ductus arteriosus) and continuing as a much narrower vessel to the lung (Fig. 1*a*). The origin of the arterial ligament on either side is obscured, in ventral view, by the great systemic arteries on the right side of the body and by the arched course of the left pulmonary artery as it passes dorsally towards the lung. Fibres arise from the vagus nerve as it passes dorsal to the aortic arch on either side and run through the membrane accompanying the arterial ligament onto the surface of the pulmonary artery at the site of origin of the ligament. No vagal fibres joined the pulmonary artery proximal to this point. The pulmonary artery distal to this segment, however, was densely innervated by fibres that arose from the vagus in the vicinity of the origin of the cardiac and pulmonary branches of this nerve.

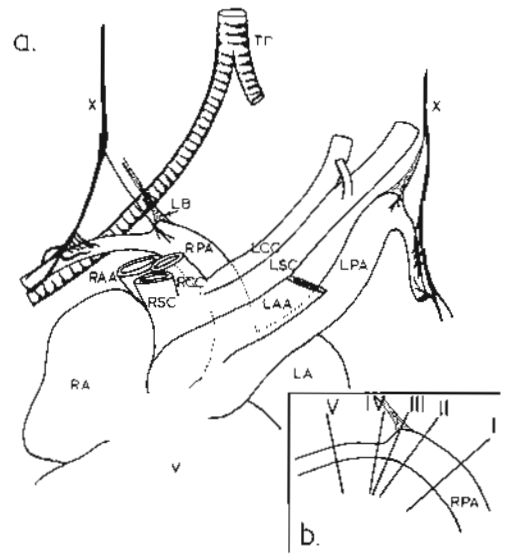

FIG. 1 (*a*) Diagram of the central arteries in the turtle showing the vagal innervation of the pulmonary arteries in ventral view. (*b*) Diagram of the right pulmonary artery indicating areas from which histological sections were taken. LA, left atrium; LAA, left aortic arch; LB, ligamentum Botalli; LCC, left common carotid; LPA, left pulmonary artery; LSC, left subclavian; RA, right atrium; RAA, right aortic arch; RCC, right common carotid; RPA, right pulmonary artery; RSC, right subclavian; Tr, trachea; V, ventricle; X, vagus nerve.

Histological sections (Fig. 2) selected from several sites along the pulmonary artery (Fig. 1*b*) indicate that smooth muscle is abundant throughout the length of the extrinsic pulmonary artery. The proximal segment, however, is relatively thin walled and thus smooth muscle is more preponderant in the narrower distal segment. The transition from a large, thin-walled artery to a narrow, thick-walled artery occurs across the segment where the arterial ligament originates. A dense thickening of the smooth muscle is normally associated with this region (Fig. 2*b*). In a few individuals (Fig. 2*a*), however, little or no reduction in arterial dimensions occurred along the extrinsic pulmonary artery although traces of a remnant of the arterial ligament still appeared (Fig. 2*a*, IV). In others (Fig. 2*c*) a conical pocket remained of the ductus arteriosus and this region was heavily vested with a thickening of smooth muscle. These latter cases represent the extremes of individual variability that we witnessed in *Chrysemys scripta* and are not surprising in view of reported cases of the occurrence of a patent ductus arteriosus in some young turtles and total absence of even an arterial ligament in others (see O'Donoghue 1917 for review).

Spontaneous Activity

Under control conditions the pressure measured in the perfusion cannula increased with increasing flow but at any given flow, perfusion pressure usually remained constant throughout an experiment. Some preparations, however, frequently showed spontaneous activity that could generate changes in pressure of less than one to more than 20 cm H_2O pressure under constant perfusion. The amplitude and frequency of the spontaneous activity were not constant and there was no close correlation between the amplitude of the pressure change and the control perfusion pressure or rate of flow in the artery (Fig. 3, middle and lower traces). Spontaneous activity usually became more prevalent after several hours of perfusion.

Effects of Stimulating the Vagus Nerve

Stimulation of the cervical vagus nerve in the turtle always caused constriction of the pulmonary artery. The response had a latency of

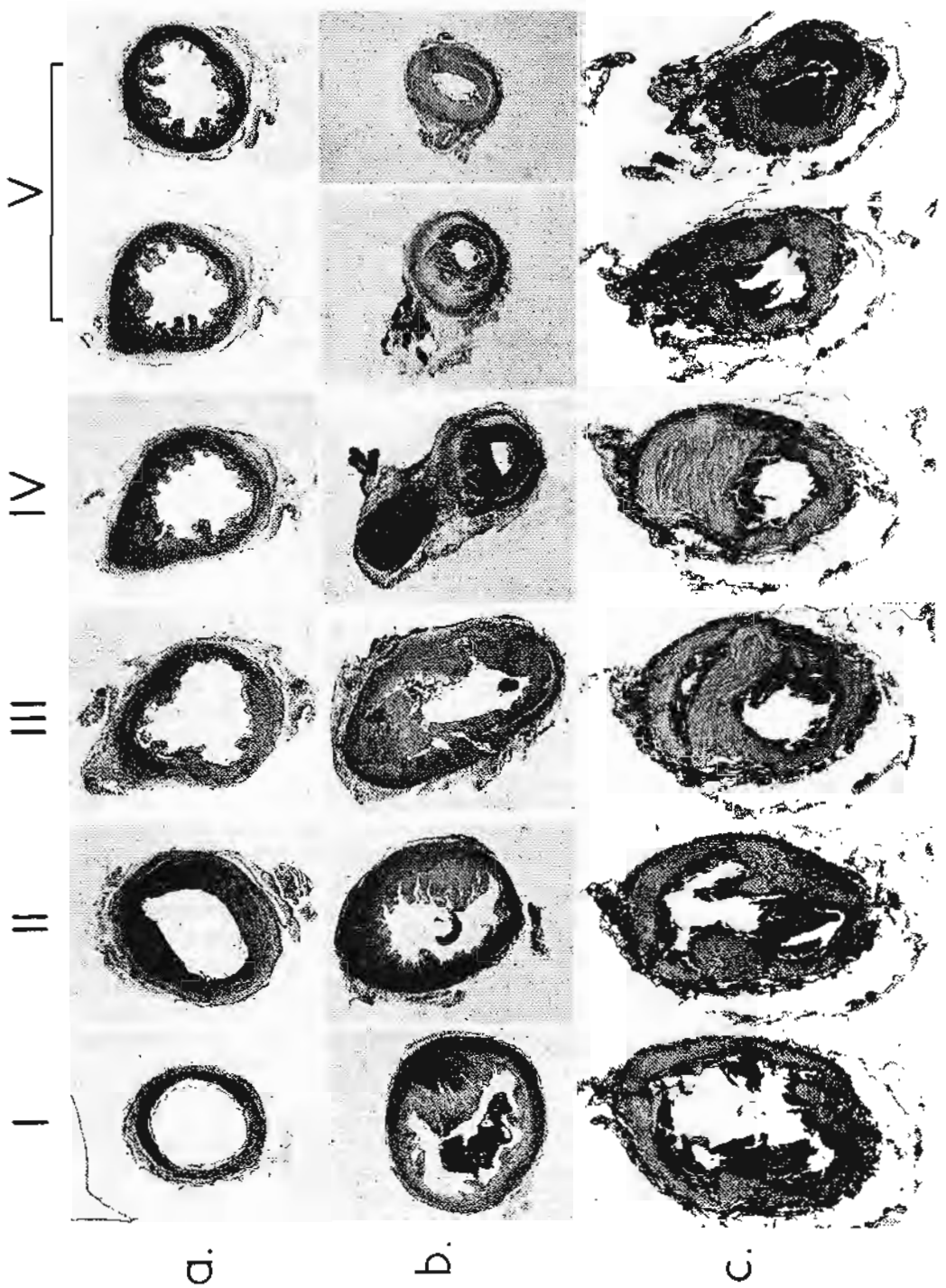

FIG. 2. Photomicrographs of the right pulmonary artery from three individuals (*a–c*) taken from the sections (I–V) indicated in Fig. 1*b*.

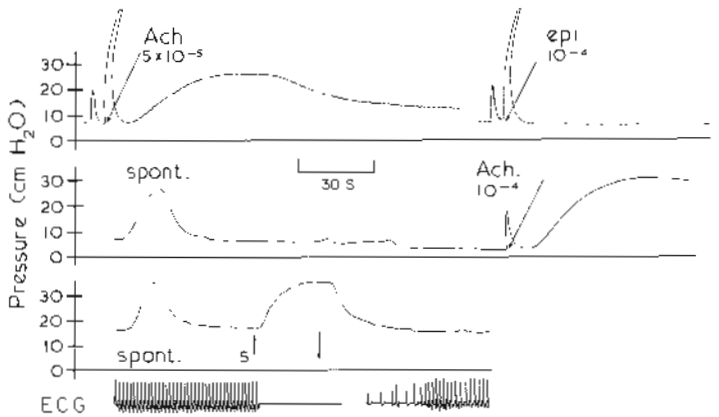

FIG. 3. Effect of various disturbances on constant-flow perfusion pressure in the isolated extrinsic pulmonary artery. Upper pressure trace: effects of infusion of 0.1 ml of acetylcholine (Ach) and epinephrine (epi), flow = 0.38 ml/min. Middle pressure trace: spontaneous activity and effects of infusion of 0.1 ml acetylcholine, flow = 0.76 ml/min. Lower pressure trace: spontaneous activity and effects of vagal stimulation (between arrows) at 1.5 V, 25 Hz, 10 ms, flow = 1.9 ml/min. Electrocardiogram (ECG) shows simultaneous bradycardia resulting from vagal stimulation. (Sharp peaks accompanying drug infusions are injection artefacts.)

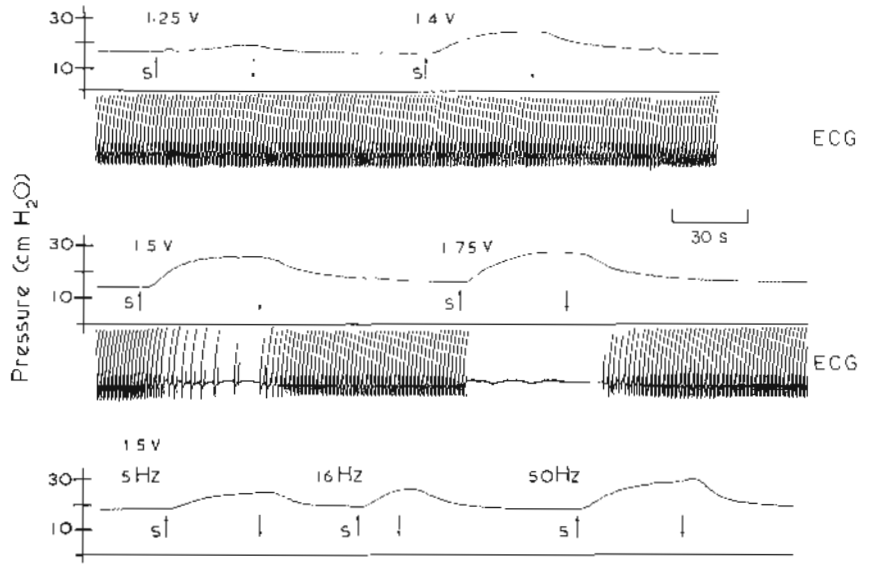

FIG. 4. Effects of vagal stimulation on the constant-flow perfusion pressure in the extrinsic pulmonary artery and on the heart rate. Each electrocardiogram accompanies the pressure trace above it. Vagal stimulation occurs between each pair of arrows and is at 50 Hz frequency in the upper two pairs of traces. All other stimulus frequencies and strengths as indicated. Flow = 0.38 ml/min throughout.

about 2 s and continued 3 or 4 s beyond the period of stimulation if a stable response was allowed to develop (Figs. 3, 4). Maximum response to vagal stimulation was obtained with stimulus strengths around 1.5 V and a stimulus frequency of 40–50 Hz (Fig. 4). For all stimulations the pulse duration was 10 ms. Under these conditions, perfusion pressure could rise by 35 cm H_2O above the control level. The functional threshold of vagal fibres to the pulmonary artery is lower than that of the cardiac fibres as seen by the initiation of contraction in the pulmonary artery before any signs of stimulation-induced bradycardia. Max-

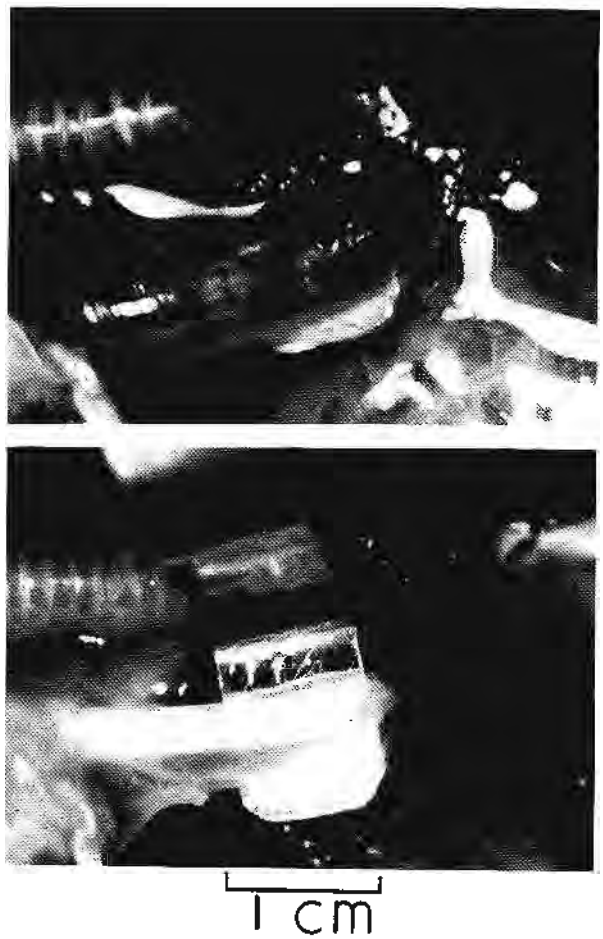

FIG. 5. Effect of vagal stimulation on the pulmonary artery in vivo. The upper photograph shows the artery under control conditions (blood flows from left to right). The lower photograph illustrates the totally constricted artery during vagal stimulation (1.5 V, 50 Hz) with full occlusion of pulmonary flow.

imum constrictor response was obtained from the pulmonary artery at high levels of bradycardia but before stimulation-induced cardiac arrest (Fig. 4).

To evaluate the extent to which vagal stimulation could reduce pulmonary blood flow in vivo, the right pulmonary artery in four specimens was carefully cleared for observation and photographic recording of the distal segment of the extrinsic pulmonary artery during vagal stimulation. Using 1.5-V, 50-Hz stimulation the vasoconstrictor response was sufficient in every case to totally occlude the vessel (Fig. 5) throughout the entire cardiac cycle. By changing stimulus strength or frequency, varying degrees of reduction in pulmonary flow could be obtained.

The entire pulmonary circuit on one side was isolated in four specimens to establish the contribution of the various levels of the pulmonary arterial system in the vasoconstrictor response to vagal stimulation. A cannula was inserted retrograde into the pulmonary vein to drain the circuit and the perfusion cannula was inserted into the pulmonary artery at its origin from the main pulmonary trunk. The perfusion cannula was then advanced distally along the pulmonary artery in small steps and the ipsilateral vagus stimulated at 1.5 V and 50 Hz at the end of each step. This was continued until the cannula was advanced to the lung hilus and then the same procedure was followed in reverse until the cannula was withdrawn again to its site of insertion. There were no differences in the increased perfusion pressure resulting from vagal stimulation regardless of where the

perfusion cannula was positioned in the segment of the extrinsic artery proximal to the arterial ligament. Once the cannula was advanced beyond the site of narrowing of the pulmonary artery, near the origin of the arterial ligament, the increment in perfusion pressure accompanying vagal stimulation was much reduced (Fig. 6). This change in response to vagal stimulation developed along the distal segment of the extrinsic pulmonary artery. As the cannula was advanced into the hilus of the lung, the remaining small increase in pulmonary resistance on vagal stimulation remained unchanged. After withdrawal of the cannula into the proximal segment of the extrinsic pulmonary artery, the full vasoconstrictor response could again be elicited (Fig. 6, lower trace).

Effect of Sympathetic Stimulation and Infusion of Various Drugs

Stimulation of the cervical sympathetic nerve caused no change in perfusion pressure in all animals studied. In addition epinephrine (1–100 μg) was without effect upon the artery (Fig. 3) either in the perfused preparations or the in vivo preparations.

The response to ACh infusion (1–10 μg), however, mimicked the full response to vagal stimulation (Fig. 3). The excitatory responses to acetylcholine and vagal stimulation were rapidly blocked with atropine (10 μg). Atropine did not alter the responses to sympathetic stimulation or infusion of epinephrine, indicating that the negative responses were not due to the simultaneous presence of active cholinergic constriction.

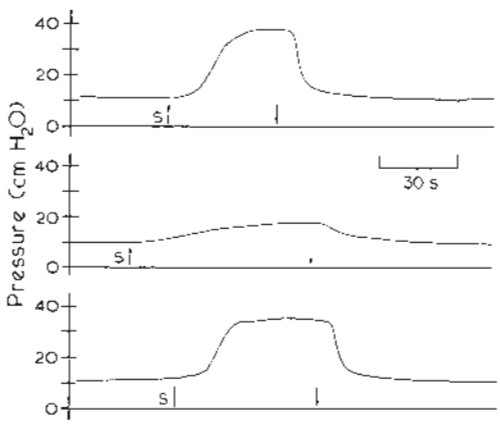

FIG. 6. Effect of vagal stimulation on constant-flow perfusion pressure generated in the right pulmonary vascular circuit. Vagal stimulation (1.5 V, 50 Hz) occurs between each pair of arrows. Upper trace shows response to stimulation during perfusion of entire circuit (perfusion cannula at origin of right pulmonary artery). Middle trace shows response of intrinsic pulmonary arteries, only, to stimulation (perfusion and recording cannula now advanced to near lung hilus). Lower trace shows response after the cannula is withdrawn back to the origin of the right pulmonary artery. Flow = 0.38 ml/min throughout.

Discussion

Krogh (1906) established that vasomotor fibres in the vagosympathetic nerve of the turtle were tonically active and that vagal section led to vasodilatation of the pulmonary artery on the ipsilateral side of the body. These results were later confirmed by Luckhardt and Carlson (1921), who further showed that this activity could be blocked by atropine, which established the cholinergic nature of these fibres. Berger (1972) has demonstrated that the same innervation exists in the lizard and is capable of constricting the pulmonary artery sufficiently to produce extremely large changes in pulmonary vascular resistance. The results presented here demonstrate that this also is true of the vagal innervation to the pulmonary artery in the turtle. Increasing strength of vagal stimulation produced a graded pressure rise in the pulmonary artery when perfused under constant flow (Fig. 4) and the constriction that is elicited is sufficient to occlude the pulmonary artery under physiological conditions as demonstrated by the in vivo experiments (Fig. 5). These results were reproduced by infusion of ACh (Fig. 3) and both the constriction caused by vagal stimulation and ACh infusion were totally blocked by infusion of atropine, confirming that cholinergic fibres within the vagosympathetic trunk are the vasomotor fibres. This strong constrictor response to vagal stimulation and ACh infusion in *Chrysemys scripta* confirms recent results obtained by Berger (1974) in a brief series of experiments on two tortoises (*Chelodina longicollis*) and is in marked contrast to the detailed account of a weak and functionally insignificant vagal cholinergic constrictor response published earlier (on *Chelodina longicollis*) (Berger 1972).

Failure to evoke any response from cervical sympathetic nerve stimulation confirms the work of previous investigators (Luckhardt and Carlson 1921; Berger 1972). The lack of response of the extrinsic pulmonary artery to infusion of epinephrine even in large doses, alone or after

atropine, is consistent with the lack of response to other sympathomimetic drugs (norepinephrine, phenylephrine, isoprenaline) reported by Berger (*Chelodina longicollis*) (1972). Although Luckhardt and Carlson (1921) recorded dilatation and constriction of the pulmonary arteries to various doses of epinephrine (*Chrysemys elegans*, *Malacoclemys lesuerii*), their results are based on direct observations of the small pulmonary arteries on the surface of the lung and measurements of flow through the entire pulmonary circulation. This suggests that sympathomimetic drugs act on the smaller intrinsic pulmonary arteries. The argument for vasoconstriction of the extrinsic pulmonary artery by epinephrine presented by Woodbury and Robertson (1942) is unfortunately based on indirect evidence and since the species of turtle these investigators examined is not given it may be that this discrepancy arises from a species difference.

Despite the abundance of smooth muscle throughout the extrinsic pulmonary artery (Fig. 2), the distal segment from site of origin of the arterial ligament to the lung hilus is the major area of change in pulmonary vascular resistance on vagal stimulation (Fig. 6). The absence of any significant pressure fall as the perfusion cannula was advanced along the proximal pulmonary artery during vagal stimulation under conditions of constant flow suggests that this segment does not contribute to changes in pulmonary resistance. There was, however, a rapid pressure drop across the area of origin of the arterial ligament and a continuing fall in pressure along the distal segment of extrinsic artery to the lung hilus under these conditions, confirming the work of Berger (1974). Consistent with this are anatomical observations, during careful gross dissection, of vagal innervation throughout the distal segment of the extrinsic pulmonary artery (Fig. 1) and no vagal innervation of the proximal arterial segment. Severing the vagal branches to the distal segment of the pulmonary artery led to a reduction in the resistance to flow developed on vagal stimulation similar to that seen by advancing the perfusion cannula beyond this segment (Fig. 6). In the region of the lung hilus the pulmonary artery rapidly becomes thin walled and elastic, suggesting that the small pulmonary vascular resistance distal to this site is spread throughout the smaller arterial branches as is typical of the blood supply to other organs.

Occlusion of the distal extrinsic pulmonary artery in vivo by vagal stimulation was easily and repeatedly produced in our experiments (Fig. 5) yet Luckhardt and Carlson (1921) observed that vagally induced constriction was rarely sufficient to obliterate the lumen of arteries on the surface of the lung (*Chrysemys* and *Malacoclemys*). This is understandable in light of the minor contribution made to changes in pulmonary vascular resistance by the intrinsic pulmonary arteries that these researchers were observing. Much of the change in lumen diameter that they observed must in fact have been due to passive factors stemming from the reduction in pulmonary pressure and flow produced proximal to their observation site.

Aakhus and Johansen (1964) report that a common feature of the pulmonary artery during submersion in the duck was the presence, in diastole, of a particularly narrow segment about midway between the base of the artery and the site where the artery entered the pulmonary parenchyma. It would appear that this distal segment of the extrinsic pulmonary artery is retained as a major control site for changes in pulmonary vascular resistance even in some species where all trace of the ductus arteriosus or arterial ligament are absent. Further, owing to the nature of the incompletely divided ventricles in non-crocodilian reptiles, changes in resistance of the pulmonary and systemic vasculature play an important role in determining the distribution of blood on ventricular ejection. Evidence suggests that shunt flow through the chelonian ventricle is predominantly controlled by resistance changes in the pulmonary vasculature beyond the pulmonary outflow tract (White 1968, 1970; Johansen *et al.* 1970; Shelton and Burggren 1976). Our findings suggest that the efferent limb of these reflex changes in pulmonary vascular resistance in the turtle *Chrysemys scripta* is the distal segment of the extrinsic pulmonary artery and the cholinergic vasomotor fibres supplying it from the vagosympathetic trunk.

Acknowledgements

We express our gratitude to Professor F. N. White, Department of Physiology, University of California at Los Angeles Medical School,

for generously allowing us access to unpublished material as well as for his helpful suggestions and encouragement. The technical assistance of Ms. Daphne Hards in preparing histological samples is also gratefully acknowledged.

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