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LOCALIZATION OF BARORECEPTORS AND GAIN OF THE BARORECEPTOR–HEART RATE REFLEX IN THE LIZARD *TRACHYDOSAURUS RUGOSUS*

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SUMMARY

1. The location of arterial baroreceptors was studied in unanaesthetized *Trachydosaurus rugosus* by inflation of strategically placed perivascular cuffs. There was no evidence for baroreceptors at the site homologous with the carotid sinus of mammals or at the site homologous with the aortic arch baroreceptors of mammals and birds. The baroreceptors of *T. rugosus* probably lie in the truncus arteriosus.

2. When the sensitivity or gain of the cardiac response to changes in systemic arterial pressure was expressed as change in heart period per unit change in mean aortic blood pressure, the value for 6 animals was 1370 ms/kPa (100 ms/mmHg). This is very much greater than reported values for rabbits. If, instead, gain was calculated as the percentage increase in heart rate per unit fall in mean aortic pressure, a mean gain of 98%/kPa (7·2%/mmHg) was obtained. It is suggested that the latter method provides the better comparative value.

3. Adrenergic neurone blockade with bretylium and muscarinic blockade with atropine or hyoscine indicated that sympathetic fibres probably play the dominant role in generating the gain of the cardiac baroreflex.

INTRODUCTION

The existence of baroreceptors and baroreflexes in reptiles has been demonstrated by the observation that blood pressure is restored towards normal after being disturbed experimentally in iguanas (Hohnke, 1975) and snakes (Seymour & Lillywhite, 1976; Lillywhite & Seymour, 1978). On the efferent side of the baroreflex, many studies have shown that the heart and blood vessels receive an autonomic innervation which is capable of changing vascular resistance and the rate and force of beating of the heart (Berger & Burnstock, 1979). The afferent and central components, by contrast, have received little attention, so that nothing is known about the sensitivity of the reptilian baroreflex relative to that of mammals. In addition to providing this information, the present investigation sought to localize the baroreceptors of the lizard *Trachydosaurus rugosus*.

The method selected was to produce changes in arterial pressure by inflation of

perivascular cuffs. With this technique the gain of the cardiac component of the baroreflex can be calculated just as it is in mammals (Korner *et al.* 1972). The technique can also be used to determine the location of the baroreceptors. Inflation of cuffs placed around central arteries reveals, by its effect upon heart rate, whether the baroreceptors lie proximal or distal to the cuffs. Cuffs were strategically placed to test whether baroreceptors lie in any of three different locations chosen on the basis of homology and published reports on baroreceptor nerves in reptiles.

The mammalian carotid body–carotid sinus is homologous with that region in reptiles where the embryonic common carotid artery divides into the internal and external branches (Adams, 1958, 1962). This region was the first to be tested. The second site to be tested was the region in reptiles homologous with the aortic arch baroreceptive region of mammals. In mammals, Nonidez (1935*a*, 1937) reported that these receptors lie on the persistent aortic arch, near the position of the embryonic ductus arteriosus. The ductus arteriosus or its remnant lies on each lateral aorta in reptiles at quite a distance from the truncus arteriosus (O'Donoghue, 1917, 1920; Burggren, 1976). The third possible baroreceptive region examined was the truncus arteriosus, which is innervated in reptiles by branches of the vagus nerve (Gaskell & Gadow, 1884; Adams, 1962). From previous work it is not clear whether the branches contain baroreceptor afferent fibres. Fedele (1937) observed a consistent depressor response on central stimulation of the truncal nerves, whereas other workers did not (Kazem-Beck, 1885, 1888; Mills, 1885).

METHODS

Experiments were performed on a total of 14 lizards of either sex weighing 500–730 g. The surgical implantation of inflatable cuffs and blood pressure cannulae was carried out under barbiturate anaesthesia (Nembutal: Abbott Laboratories). The dose of anaesthesia required to prevent a righting reflex, limb withdrawal reflex or arching of the hindlimbs in response to prodding of the vent, ranged from 20–45 mg/kg (i.p.) and was independent of body mass. An initial dose of 20 mg/kg was supplemented after 30 min by a further dose of 10 mg/kg if required. During abdominal surgery, and usually for a short time afterwards, animals were ventilated with a small animal respirator. All experiments were carried out during spring and summer, apart from one which was performed in autumn.

(1) *Assessment of the gain of the reflex*

The aorta and posterior vena cava were exposed by a mid-ventral abdominal incision. A cuff was placed around the posterior vena cava just rostral to the liver and its inflation tube was led out through a stab wound in the lateral abdominal wall. A second cuff was installed around the aorta just rostral to the coeliaco-mesenteric artery. About 1 cm rostral to the aortic cuff a non-occlusive polyethylene cannula (i.d. 0·50 mm; o.d. 0·90 mm) was inserted through a very fine puncture in the aorta and advanced a few cm towards the heart. The cannula usually had a closed, angled tip with one or two apertures slightly behind the tip.

Gain of the baroreceptor-heart rate reflex was calculated as the change in hear

period induced by a unit change in mean systemic arterial pressure. Heart period is given by:

$$\text{HP} = \frac{60}{\text{heart rate (beats/min)}} \times 1000 \text{ ms.}$$

Mean arterial pressure was calculated as:

$$\bar{P}_{art} = \frac{P_{systolic} + (2 \times P_{diastolic})}{3} \text{ kPa.}$$

Changes in arterial pressure were induced by graded inflations of the arterial and venous cuffs. In mammals such a procedure produces a sigmoid relationship between HP and $\bar{P}_{art}$ (Korner *et al.* 1972). In the lizard it is difficult to induce high pressures by aortic occlusion. Thus the upper plateau of this supposedly sigmoid relationship is not always apparent and the most common finding is that points obtained at low and high pressures are clustered into two groups with few points between. For this reason it was necessary to define gain as the slope of a regression line fitting all available points. In situations where a lower plateau was evident all points more than 1 kPa lower than the right-most point of the lower cluster were arbitrarily rejected. The slope of the resulting line of best fit was calculated by the method of least squares to give gain in ms/kPa.

(2) *Localization of the baroreceptors*

(*a*) *Test of carotid vasculature*

A mid-ventral incision was begun at the level of the forelimbs and continued forwards for a distance of about 3 cm. The anterior portion of the pericardium was exposed and the right and left common carotid arteries were separated from the closely adherent lateral aortae at their origin from the truncus. A cuff was placed around each carotid artery in this position (A, Fig. 1). Each ductus caroticus was approached through a lateral incision in the neck and tied firmly. In an attempt to avoid damaging any innervation of the carotid trifurcation the ties were made at the junction of the duct and the lateral aorta. To monitor the effect of cuff inflation the ductus caroticus of one side was cannulated towards the trifurcation.

(*b*) *Test of lateral aortic vasculature*

After exposing the anterior pericardium the lateral aorta of each side was gently separated from the common carotid artery and the pulmonary artery close to the truncus. A cuff was placed around each lateral aorta (B, Fig. 1) and each ductus caroticus was ligated close to the carotid trifurcation. In this way the possibility of damage to any innervation of the lateral aortae was minimized.

(*c*) *Test of truncus arteriosus*

The pericardium was opened in the midline and a large cuff was placed around the truncus arteriosus as distal as possible to the ventricle (C, Fig. 1). In one animal a cannula was inserted through a fine stab wound into the carotico-aortic trunk (see Fig. 1). The cannula was advanced towards the ventricle until its tip lay proximal to the cuff and therefore monitored the effect of cuff inflation on intra-truncal pressure.

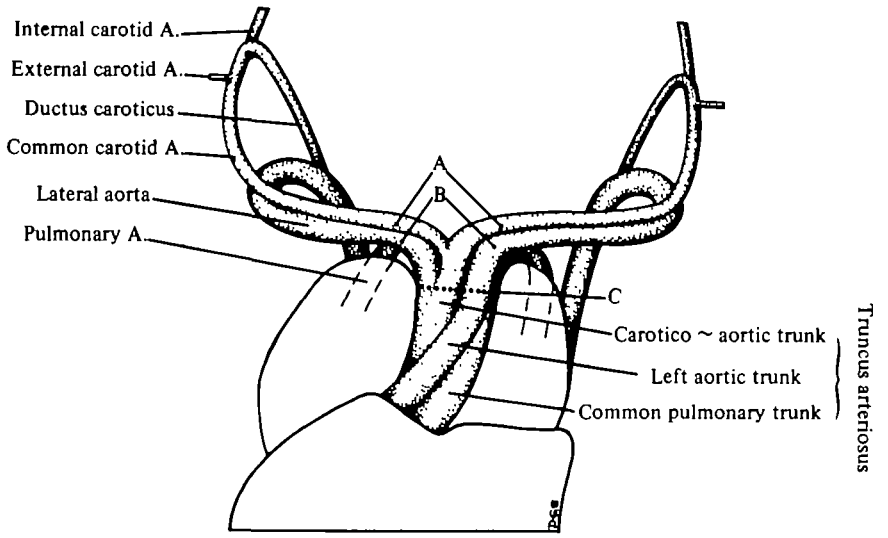

Fig. 1. Drawing of the central arteries of *T. rugosus*. Note the existence of a ductus caroticus on each side of the animal running from the carotid trifurcation to the lateral aorta. Carotid arterial, lateral aortic and truncal cuffs were installed as indicated by letters A, B and C respectively.

(3) *Protocol*

During an experiment the surgically-prepared animal was placed in a narrow darkened box. Most animals remained quite still for many hours. Room temperature was maintained at about 25 °C. Pressures were measured by connecting the cannulae filled with heparinized saline (100 i.u./ml) to Statham P23DI pressure transducers calibrated against a static pressure head. Heart rate was obtained by using an electrocardiographic signal as the trigger for a Grass Model 7P44 tachograph. All records were displayed on a Grass Model 79D polygraph. Drugs used were hyoscine hydrobromide (David Bull), atropine sulphate (Sigma) and bretylium tosylate (Burroughs-Wellcome). Each drug was administered by intra-arterial injection over a period of 2–3 min.

RESULTS

In all 14 animals studied, tachycardia and hypertension persisted for several hours following anaesthesia. Moreover, maximal cardiac responses to cuff inflation were usually not obtained until 24 h after anaesthesia. Thus all results presented here were collected at least 24 h after surgery.

Resting heart rate in conscious lizards ranged from 20–30 beats/min and resting systemic arterial pressure was 6·0 kPa systolic (range 5·1–6·6 kPa) over 4·4 kPa diastolic (range 3·9–4·9 kPa).

Baroreflex and gain

In all animals studied, inflation of the aortic cuff caused a rise in systemic arterial pressure and a prompt bradycardia which usually reached its maximum within 10 s (Fig. 2A). Inflation of the posterior vena caval cuff invariably reduced systemic arterial pressure and increased heart rate (Fig. 2B). The time required to reach the

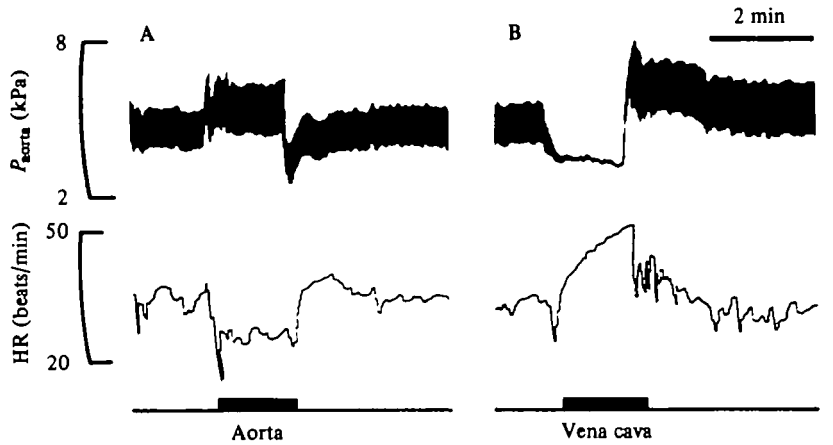

Fig. 2. Effect upon aortic blood pressure (P_{aorta}, kPa) and heart rate (HR, beats/min) of inflating a cuff around the aorta (in A) or the posterior vena cava (in B) for the period marked by the bar. Note that bradycardia developed more quickly than tachycardia, and after deflation of the cuff heart rate returned to the control level more quickly after bradycardia than after tachycardia.

Table 1. *Gain of the cardiac component of the baroreflex in ms/kPa*

The correlation coefficient of the least-squares line whose slope corresponds to gain is included in brackets. In animals 2 and 5 two plateau points were excluded from the analysis of control gain.

	Gain (r value) in msec/kPa		
Animal	Control	After Atropine (2·9 mmol/kg) or Hyoscine (2·3 mmol/kg)	After Bretylium (2·4 mmol/kg)
1	620 (0·98)	460 (0·99)	—
2	990 (0·95)	—	—
3	1400 (0·90)	1150 (0·88)	—
4	2050 (0·97)	—	80 (0·56)
5	690 (0·93)	—	80 (0·97)
6	2470 (0·99)	—	290 (0·90)
	Mean gain 1370		

maximum increase in heart rate ranged from 90–200 s. Recovery of heart rate to resting levels required up to 10 min, with recovery from tachycardia taking longer than from bradycardia. The mean gain of the baroreceptor-heart rate reflex in 6 lizards was 1370 ms/kPa (see Table 1).

Effects of drugs on the baroreflex

(*a*) *Atropine or hyoscine*

Injection of atropine (2·9 mmol/kg; $N = 1$) or hyoscine (2·3 mmol/kg; $N = 2$) caused an immediate increase in heart rate of 10% (27–30 beats/min), 25% (20–25 beats/min) and 60% (22–35 beats/min) in the three animals tested. Muscarinic blockade also abolished the often very pronounced beat-to-beat variation in resting

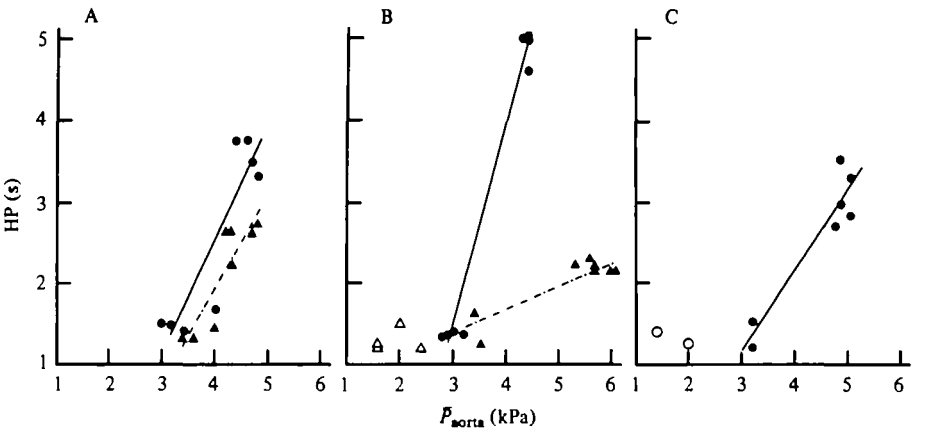

Fig. 3. Plot of heart period (HP, s) versus the experimentally-induced mean aortic blood pressure ($\bar{P}_{aorta}$, kPa). In A, B and C circles represent points obtained under control conditions and these are connected by solid lines calculated by least-squares regression. Note that all control points fall on the line in A and B whereas the 2 open circles in C clearly lie on a plateau. In A the triangles represent points obtained after treatment with hyoscine (2·3 mmol/kg i.a.) while, in B, points marked by triangles were obtained after injection of bretylium (2·4 mmol/kg i.a.). The broken lines were calculated by least-squares regression. Open circles and triangles represent points excluded from the least-squares analysis.

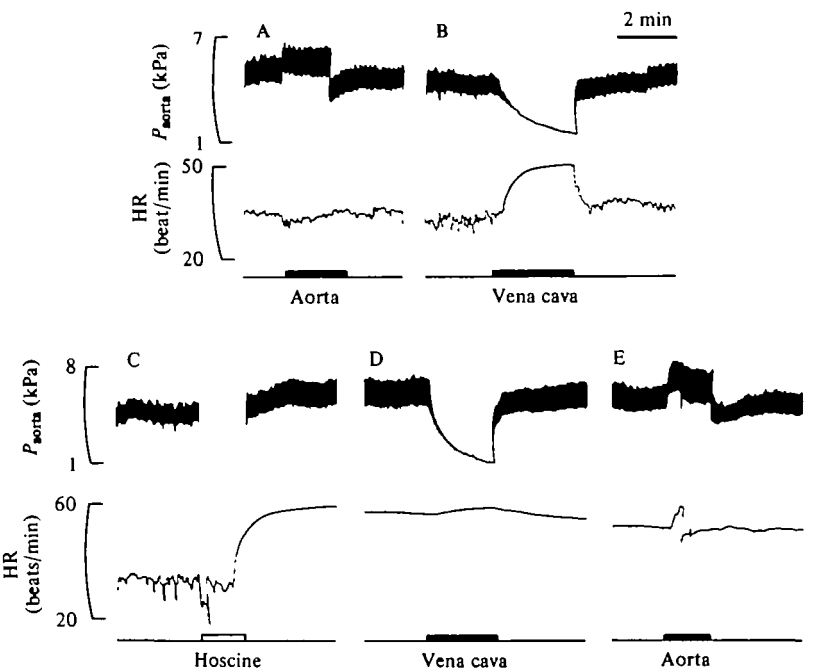

Fig. 4. Effect of inflating a cuff on the aorta (A) and posterior vena cava (B) upon aortic blood pressure (P_{aorta}) and heart rate (HR) in an animal injected 3 h earlier with bretylium (2·4 mmol/kg). Note that, following injection of hyoscine (2·3 mmol/kg), the variability in beat-to-beat interval was greatly reduced (C), as were the responses to vena caval (D) and aortic (E) cuff inflation.

heart rate. After either drug, aortic cuff inflation usually still caused a slight reduction of heart rate although never to the level attained during a control response. Posterior vena caval cuff inflation caused heart rate to rise to approximately the same level before and after atropine or hyoscine. In the two animals in which all the necessary measurements were made, muscarinic blockage caused a moderate reduction of the gain of the baroreflex (Fig. 3A; Table 1).

(b) *Bretylium*

Injection of bretylium (2·4 mmol/kg) caused an immediate increase in blood pressure and heart rate in the three animals tested. After approximately 3 h, blood pressure returned to the control level, while heart rate was still somewhat elevated. Beat-to-beat fluctuations in heart rate persisted after bretylium treatment (Figs. 4A, B), and cardiac responses were still obtained to inflation of the cuffs around the aorta (Fig. 4A) and posterior vena cava (Fig. 4B): but the gain of the reflex was greatly reduced (Fig. 3B; Table 1). In all three animals treated with bretylium, subsequent injection of hysocine (2·3 mmol/kg) elevated heart rate to a level in excess of that achieved during a post-bretylium venous cuff inflation (Fig. 4C). In addition, after treatment with bretylium plus hysocine only very small responses were obtained on inflation of either cuff, and beat-to-beat fluctuations in heart rate were abolished (Figs. 4D, E). The early tachycardia to aortic cuff inflation in Figure 4E is reminiscent of the Bainbridge reflex, and a similar response has been reported after parasympathetic blockade, or after a combination of parasympathetic and sympathetic blockade, in the dog (Glick & Braunwald, 1965).

Localization of baroreceptors

Two animals were prepared with a cuff around each common carotid artery; in one animal the resting heart rate was within the range quoted above, whereas the second animal had a resting heart rate substantially above that range (about 40 beats/min). In both animals, simultaneous inflation of the cuffs around the right and left common carotid arteries caused a fall in pressure in the ductus caroticus, while having no persistent effect on the pressure measured in the aorta (Fig. 5A). This procedure had no effect upon heart rate, even though a posterior vena caval cuff inflation produced a substantial tachycardia in the same animals (Fig. 5B).

Inflation of cuffs around the lateral aortae in two animals caused a fall in aortic pressure and a bradycardia that was similar to that observed when the aortic cuff was inflated. A fall in central arterial pressure induced by vena caval cuff inflation in these animals led to the expected tachycardia.

Inflation of the cuff around the truncus arteriosus caused a marked elevation of systolic pressure measured in the truncus, while diastolic pressure either remained at the control level or fell substantially (Fig. 6). It is difficult to explain the fall sometimes observed in diastolic pressure, but it is possible that the tip of the cannula sometimes interfered with normal closure of the ventricular valves. In any case, a bradycardia invariably occurred during the period of truncal cuff inflation in 21 tests in 2 animals (e.g. Fig. 6). In one animal, heart rate always returned to the control level on deflation of the cuff. In the second animal studied, heart rate either returned to a level close to control (Fig. 6A), or underwent a lengthy overshoot. This secondary tachy-

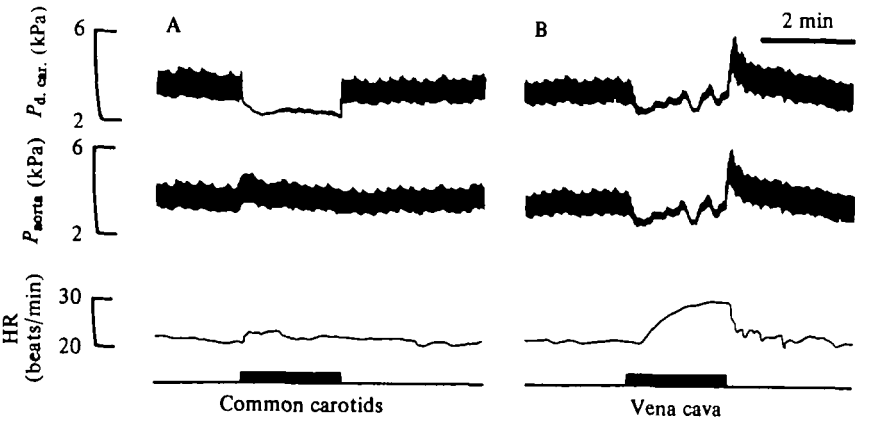

Figure. 5. (A) Effect of inflation of cuffs around the right and left common carotid arteries upon pressure recorded from the ductus caroticus ($P_{d.car.}$) and the aorta (P_{aorta}) and upon heart rate (HR). (B) Effect of posterior vena caval cuff inflation.
Note that each ductus caroticus was tied near the lateral aorta.

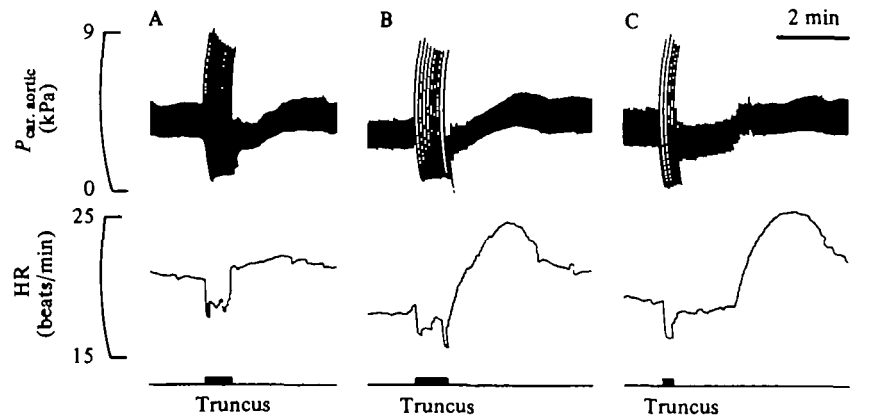

Fig. 6. Effect of inflation of a cuff around the distal truncus arteriosus upon intra-truncal pressure ($P_{car. aortic}$) and heart rate (HR). In A, heart rate returned to about the control level on deflation of the cuff, in B heart rate overshot the control level just after deflation, while in C tachycardia occurred some time after deflation.

cardia was always associated with struggling body movements which occurred either near the end of the period of cuff inflation (Fig. 6B) or soon after deflation of the cuff (Fig. 6C). Struggling in this animal always resulted in a substantial and lenghty tachycardia.

DISCUSSION

Changes in arterial pressure in *Trachydosaurus rugosus* elicited cardiac responses similar to those observed in mammals (Korner *et al.* 1972), indicating the existence of pressure-sensitive receptors. Since lizards have an undivided ventricle an increase or decrease in central systemic arterial pressure is probably accompanied by like changes in central pulmonary arterial pressure. Thus, the observation of heart-rate responses to posterior vena caval cuff inflation, and to aortic cuff inflation, could be explained by

the presence of baroreceptors in the systemic or pulmonary arterial circuit, or both. It is also possible that the reflexes were mediated by receptors in the heart or central veins. A further point to be borne in mind is that, while cuff inflation experiments can be used to localize the receptor site which has the most powerful influence over heart rate, they cannot eliminate the possibility that subsidiary receptor sites exist elsewhere.

Localization of baroreceptors

It was shown conclusively by Hering (1923, 1924) that baroreflexes in mammals originate from the region where the common carotid artery divides. The homologous region in lizards with a ductus caroticus, such as *T. rugosus*, is the area of the carotid trifurcation (Adams, 1958). Inflation of cuffs on each common carotid artery, while producing a fall in pressure in the ductus caroticus, had no effect on heart rate (Fig. 5). Thus, the carotid 'sinus' is not the location of the baroreceptors in *T. rugosus*.

The aortic arch baroreceptors of mammals and birds are reported to lie near where the ductus arteriosus joins the surviving aorta (Nonidez, 1935*a*, *b*; Boyd, 1941). However, inflation of a cuff proximal to the homologous region in *T. rugosus* produced a bradycardia, indicating a pressure rise at the baroreceptors. The arterial regions in which pressure could be expected to rise during lateral aortic occlusion are the carotid arteries, already ruled out as a site of baroreceptors, the truncus arteriosus, and the pulmonary arteries. In view of the bradycardia elicited by inflation of a cuff around the distal truncus arteriosus, the pulmonary arteries can also be ruled out. The evidence therefore points to a truncal location, or to a cardiac or central venous location for baroreceptors in *T. rugosus*.

The possibility that the baroreceptors of *T. rugosus* lie in the ventricle, auricles, or central veins can only be assessed on indirect grounds. The atrial mechanoreceptors of mammals produce tachycardia when pressure rises (Ledsome & Linden, 1967), rather than the bradycardia observed in the present study. In addition, the cardiac baroreflex is likely to be involved in the regulation of arterial blood pressure, just as its functional homologue appears to be in mammals. Thus an arterial location for baroreceptors would provide more precise information on arterial pressure and its fluctuations than would receptors in sites isolated from the arteries. Finally, although venous, atrial, and ventricular mechanoreceptors exist in mammals, virtually the entire baroreflex cardiac response is mediated by receptors lying in the arteries (Korner *et al.* 1973).

A truncal location for baroreceptors is supported by anatomical evidence that nerves similar to the mammalian aortic arch depressor nerves run to the truncus from the vagus in reptiles (Gaskell & Gadow, 1884; Mills, 1885; Kazem-Beck, 1888; Fedele, 1937; Boyd, 1942; Khalil & Malek, 1952; Adams, 1962). Moreover, while there are physiological reports that the truncal nerves of reptiles do not mediate cardiovascular responses (Mills, 1885; Kazem-Beck, 1888), Fedele (1937) showed convincingly that in several reptilian species the truncal nerves contain afferent fibres capable of mediating reflexes on the heart. In an extremely detailed account Fedele traced the truncal nerves to their terminations and stated that the afferent fibres did not reach the ventricle. In view of the arguments outlined above it seems reasonable to conclude that the baroreceptors of *T. rugosus* are located in the truncus arteriosus.

It is somewhat surprising that the homologue of the mammalian carotid sinus has no baroreceptor role in *T. rugosus*. This is so, in particular, because the region is inner-

Table 2. *Values of gain of the cardiac component of the baroreflex in conscious animals*

Values shown for the mammals were calculated in the same manner as described for *T. rugosus* using data included in the sources quoted. The value obtained from Vatner & Boettcher (1978) relates to saline infusion experiments in conscious dogs. Values have been rounded off to the nearest whole number.

Animal	Gain % increase in heart rate/mm Hg fall in pressure	Source
T. rugosus	7	Present work
Dog	5	Table 3, Glick & Braunwald (1965)
	3	Vatner & Boettcher (1978)
Rabbit	2	Fig. 3A, Korner *et al.* (1972)
Man	3	Table 4, Robinson *et al.* (1966)

vated in *T. rugosus*, and in other lizards, by vagal branches and possibly by glossopharyngeal fibres (Terni, 1931; Adams, 1958; Chowdhary, 1950; Oelrich, 1956; Mehra, 1958; Rogers, 1967). And yet the present evidence is clear. It should be remembered that only in mammals is there convincing physiological evidence of a baroreceptive role for the region where the common carotid artery divides; perhaps the carotid sinus baroreceptor region developed *de novo* in mammals, or in their synapsid reptilian precursors. The present work failed to find evidence for baroreceptors in the lateral aortae of *T. rugosus;* thus baroreceptors appear to be absent from the position directly homologous with the aortic arch baroreceptor region of birds (Nonidez, 1935*b*; though see Jones, 1973) and mammals (Nonidez, 1935*a*, 1937; Boyd, 1941).

The problem of gain comparisons between species

The gain of the cardiac component of the baroreflex of *T. rugosus* is very high when compared with that in the rabbit, cf. a mean value of 1300 ms/kPa (100 ms/mm Hg) *v.* 5·2 ± 1·0 ms/mm Hg (Korner *et al.* 1972). *Trachydosaurus rugosus* has approximately 1/10th the resting heart rate of a rabbit so that given the hyperbolic relationship between heart rate and heart period, it is unreasonable to compare the gain value of Table 1 with that reported by Korner *et al.* (1972). However, if gain is defined in percentage terms then differences in resting heart rate play no role in the value obtained. Accordingly, experimentally-induced heart rates were expressed as a percentage increase over the minimum heart rate that could be elicited by aortic cuff inflation. Gain was then recalculated as the percentage change in heart rate per unit change in mean arterial pressure. The revised mean gain for the six lizards comprising this study becomes 98%/kPa or 7·2%/mmHg. Similar gain values, dervied from previously published reports on conscious mammals, are comparable to that of *T. rugosus* (see Table 2).

The physiological significance of gain depends upon how heart rate relates to cardiac output in mammals and birds, and, in lower vertebrates with an undivided ventricle, to total heart output (see Johansen, 1972). Stroke volume is extremely labile in fish and it seems to be the prime determinant of the increased output of the heart during exercise (Jones & Randall, 1979). Little is known of the situation in amphibians,

reptiles or birds. Even in mammals there is no general agreement on the relationship between heart rate and cardiac output. However, recent work suggests that, in unanaesthetized, undisturbed dogs, changes in cardiac output are brought about mostly by changes in heart rate (Vatner & Boettcher, 1978; Boettcher *et al.* 1978; Anderson, Yingling & Sagawa, 1979). If this finding is true for all tetrapods, then the baroreflex cardiac gain would provide a reasonable index of the comparative ability of animals to increase or decrease the output of the heart in response to arterial pressure disturbances. Concerning the comparative ability of animals to regulate blood pressure, a more useful gain to measure is that relating changes in central arterial pressure and changes in pressure at the isolated baroreceptor site. In this value, total peripheral resistance and heart output both participate. Considering the location of baroreceptors in *T. rugosus*, this would be a difficult value to acquire.

Effects of Autonomic Blocking Drugs

As in all tetrapods the cardiac pacemaker of *T. rugosus* is innervated by vagal cholinergic and sympathetic adrenergic fibres (Berger, 1971). Muscarinic blockade with either atropine or hyoscine produced a substantial rise in heart rate, and beat-to-beat fluctuations in heart rate disappeared. Thus, it seems likely that resting heart rate is maintained by modulating efferent vagal discharge in *T. rugosus*. From the present work it is impossible to judge whether a resting sympathetic discharge occurs in *T. rugosus*. Lillywhite & Seymour (1978) showed that in the tiger snake, *Notechis scutatus*, β-adrenoreceptor blockade with propranolol produced a bradycardia. They concluded that the heart of the tiger snake was normally under the influence of excitatory sympathetic tone. However, propranolol blocks the effects of circulating, as well as neurally released, catecholamines. Thus Lillywhite and Seymour's observations do not necessarily indicate the existence of a resting discharge in cardiac sympathetic nerves.

An attempt was made to determine the contribution of vagal and sympathetic fibres to the gain of the baroreceptor-heart rate reflex by using hyosciene or atropine, and the adrenergic neurone blocking compound bretylium. The results summarized in Table 1 suggest that sympathetic fibres are more important in the cardiac baroreflex than vagal fibres. However, this conclusion requires qualification. It is possible that blockade of one group of fibres leads to a compensating change in the activity of the remaining group. In fact there is evidence that this occurs, particularly after bretylium treatment. Some 3 h after bretylium injection, aortic cuff inflation produced little reduction in heart rate and hyoscine injection led to a large increase in heart rate. The contribution of the vagus to gain might therefore be poorly estimated by the use of bretylium. In spite of this reservation, the combined evidence from muscarinic blockade and adrenergic neurone blockade indicates that in T. *rugosus* sympathetic fibres play the dominant role in generating the response of the heart to pressure disturbances.

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