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EFFECTS OF VAGAL STIMULATION ON SWIMBLADDER BLOOD FLOW IN THE EUROPEAN EEL *ANGUILLA ANGUILLA*

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Summary

The influence of the vagus nerve on swimbladder blood flow in the European eel (*Anguilla anguilla*) was characterized by recording the changes in blood flow rate and blood pressure following stimulation of the vagus nerve. After electrical stimulation, blood flow in the swimbladder artery increased from 0.9 ml min^{-1} to 2.1 ml min^{-1}. Video recordings of small vessels on the caudal side of the rete mirabile revealed an increase in erythrocyte velocity combined with a small vasodilation. This effect could not be blocked by injection of the α-adrenergic antagonist phentolamine, the β-adrenergic antagonist propranolol or the muscarinic cholinoceptor antagonist atropine. In all preparations with a high initial flow rate (>1.9 ml min^{-1}), vagotomy resulted in a marked decrease in blood flow (by approximately 80 %). This effect was not observed in preparations with a low initial swimbladder blood flow. Stimulation of the vagus nerve produced a decrease, and vagotomy produced an increase, in perfusion pressure in blood-perfused swimbladder preparations. Histological studies revealed the presence of a ganglion in the vagus nerve located on the anterior part of the resorbing section of the swimbladder close to the origin of the ductus pneumaticus, which is probably associated with swimbladder function. These results suggest that swimbladder blood flow, at least to some extent, is under vagal tonic control. The effects do not, however, appear to involve the classical α- and β-adrenergic or muscarinic cholinoceptor functions.

Key words: European eel, *Anguilla anguilla*, vagal stimulation, blood flow, swimbladder, microcirculation, circulatory system.

Introduction

A gas-filled swimbladder that functions as a hydrostatic organ is common in teleost fish. To maintain neutral buoyancy, a highly sophisticated control system has evolved to keep the swimbladder volume constant despite the changes in hydrostatic pressure encountered during vertical migrations. Afferent information is provided by stretch receptors in the swimbladder wall or by effects on the balance organs (Qutob, 1962; Tytler and Blaxter, 1973; Fänge, 1983). The efferent part of the system consists of nervous control of the relative surface areas of the resorbent and the secretory parts of the swimbladder wall, the control of blood flow through the swimbladder and regulation of the metabolic activity of swimbladder gas gland cells (Pelster and Scheid, 1992).

The surface area of the secretory and the resorbent parts of the swimbladder, and also the vasculature of this organ, appears to be under nervous control. In the Atlantic cod *Gadus morhua*, electrical stimulation of the right vago-sympathetic trunk caused a slight reduction in the gas gland blood flow, concurrent with an opening of the 'oval', an effect that could be inhibited by the ganglionic blocking agent chlorisondamine (Wahlquist, 1985). However, in the European eel *Anguilla anguilla*, vagotomy induced a vasoconstriction in swimbladder vessels and a decrease in the rate of gas secretion (Fänge, 1953). In a more recent study, Pelster and Scheid (1992) demonstrated that the rate of gas secretion into the swimbladder decreased with reduced blood flow to the swimbladder.

Blood flow to the swimbladder also appears to be modulated by both cholinergic and adrenergic mechanisms. The study of Pelster (1994) suggests that α-adrenoceptor-controlled resistance vessels are located close to the rete mirabile, probably at the arterial entrance of the rete. Injection of acetylcholine into the dorsal artery resulted in an instant decrease in swimbladder blood flow (Schwerte and Pelster, 1995).

In the eel swimbladder, three capillary beds are arranged in series: the arterial capillaries of the rete mirabile, the capillary network of the swimbladder epithelium and the venous capillaries of the rete mirabile. Because of this rather complex structure, the question arises of where the possible control sites are located. While humoral and neuronal effects can achieve vasoconstriction, no mechanism inducing vasodilation has yet

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been identified. Furthermore, the results concerning the influence of the vagus nerve on swimbladder blood flow are equivocal. The purpose of the present study was to analyze further the influence of the vagus nerve on the control of swimbladder blood perfusion in the European eel (*Anguilla anguilla*).

Materials and methods

Specimens of the European eel *Anguilla anguilla* L. (body mass 350–500 g) were obtained from a local supplier and kept in sea water (30 ‰) at 10 °C until used in the experiments. All experiments were performed at room temperature (20–22 °C).

In situ *experiments*

Under anaesthesia, the animals were quickly immobilized by penetrating the skull with a fine needle and spinal pithing. The animals were placed into an eel-holder (Pelster, 1994), and the gills were irrigated with well-aerated sea water (20–22 °C) at a constant flow rate of approximately 1.5–2 l min^{-1}. The body wall was opened ventrally. The swimbladder, the vagus nerve close to the ductus pneumaticus and a small area of the capillary system on the secretory part of the swimbladder were carefully exposed and freed of connective tissue. Blood vessels from other tissues entering the vein leaving the retia were ligated.

Erythrocyte velocity was measured using a Doppler flow probe (0.5–1 mm i.d.), which was placed on the artery entering the rete mirabile. The flow probe was connected to a Doppler flowmeter (Bioengineering, Iowa, USA). The dorsal artery was non-occlusively cannulated (PE 50) for measurement of blood pressure and injection of antagonists. The cannula was connected to a pressure transducer (Gould, Statham, BD 23 ID). The Doppler flow signal and the arterial pressure were continuously recorded on a computer system using a sampling rate of 20 Hz and the software package BrainWave (Broomfield, Colorado, USA). The signal from the flow probe was calibrated *in situ* at the end of each experiment. For this purpose, the swimbladder artery was occlusively cannulated with a PE 20 cannula (with 100 i.u. ml^{-1} heparin in the saline) and perfused using a peristaltic pump at constant flow rate; blood was drawn from the dorsal artery of the experimental animal. The pressure transducer was calibrated using a static water column. Small areas of the capillary system behind the rete mirabile were recorded using a CCD camera (Hamamatsu, C2400-77) and a digital video tape recorder PC card (Miro DC20). The general anatomy of the swimbladder, the location of the various sites of measurement and stimulation and the nomenclature used for the swimbladder blood vessels are presented in Fig. 1.

Blood-perfused swimbladder preparations and antagonist application

To investigate the effects of vagus nerve stimulation and vagotomy on blood-perfused swimbladder preparations, blood drawn from the non-occlusively cannulated (PE 50) dorsal artery was directed into the occlusively cannulated swimbladder artery using a peristaltic pump. The swimbladder was perfused at a constant flow rate of 0.2–0.4 ml min^{-1} (approximately 4–8 % of the cardiac output), and the perfusion pressure was measured using a pressure transducer (Gould, Statham, BD 23 ID) connected to the swimbladder artery cannula. The total volume of the cannula loop was approximately 1 ml. When used, the α- and β-adrenergic antagonists, phentolamine and propranolol (60 nmol kg^{-1} body mass), respectively, and the muscarinic receptor antagonist atropine (200 nmol kg^{-1} body mass) were injected into the perfusion cannula.

Vagus nerve stimulation

The vagus nerve was carefully lifted from the gut, and a pair

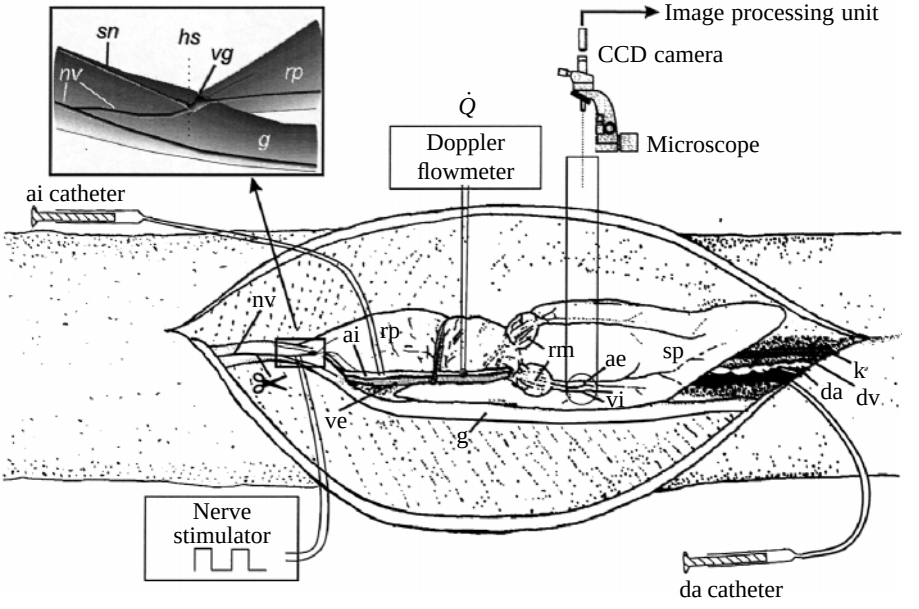

Fig. 1. Schematic drawing of the eel swimbladder showing the locations of the flow probe and of the various cannulae. The inset shows an enlarged view of the swimbladder ganglion. ai, arterial influx; ae, arterial efflux from the rete; da, dorsal artery; dv, dorsal vein; g, gut; hs, location of the histological section shown in Fig. 7; k, kidney; nv, vagus nerve; rm, rete mirabile; rp, resorbing part of the swimbladder; sn, second nerve, with ganglion cells next to the swimbladder ganglion of the vagus (stimulation of this second nerve did not modify swimbladder blood flow); sp, secretory part of the swimbladder; ve, venous efflux; vi, venous influx to the rete; vg, swimbladder ganglion of the vagus nerve; the scissors indicates the site of vagotomy. $\dot{Q}$, blood flow rate.

of unshielded platinum hook electrodes was placed under the nerve distal to the branch supplying the swimbladder using a micromanipulator; the nerve was isolated with paraffin oil and then cut anterior to the electrodes (Fig. 1). The stimulation voltage (*U*) used was 1–5 V. The stimulation frequency (*f*) was varied between 1 and 15 Hz, with a pulse width (*d*) of 10 ms and a duration (*t*) of 2–30 s. The nerve stimulator (Grass S44) was electrically isolated from any additional electrical equipment. To analyze the type of innervation, the α- and β-adrenergic antagonists, phentolamine and propranolol (60 nmol kg^{-1} body mass), and the muscarinic receptor antagonist atropine (200 nmol kg^{-1} body mass) were injected into the dorsal artery.

All drugs used were obtained from Sigma Chemical Company.

Histochemistry

The vagus nerve was carefully isolated for histochemical examination. The tissue was spread out on dental wax, fixed in buffered formalin and stained using Azan. The stained preparation was embedded in paraffin and sectioned (10 µm cross sections) from cranial to caudal over a distance of 2 mm. The sections were analyzed using a Zeiss Diaplan microscope.

Data analysis

The velocity of the erythrocytes caudal to the rete mirabile was calculated by tracking them visually along a defined distance. The distances were calibrated using an object micrometer.

Statistically significant differences in the observations were evaluated using a one-way analysis of variance (ANOVA) followed by a multiple-comparison procedure (Bonferroni, SigmaStat). Significance was accepted when $P<0.05$. Data are presented as mean ± S.E.M.

Results

Vagal effects on swimbladder blood flow rate were tested *in situ* by stimulating the vagus nerve (*t*=10 s; *U*=2.5 V; *d*=10 ms; *f*=9 Hz) close to the sphincter of the pneumatic duct while recording blood flow rate and blood pressure in the swimbladder artery and the erythrocyte velocity on the caudal side of the rete mirabile. Vagus nerve stimulation resulted in a transient small decrease in arterial blood pressure and a marked increase in swimbladder blood flow rate (Fig. 2). The effect was dependent on the frequency and duration of the stimulation. A maximal response was achieved using a frequency of 9 Hz and a duration of 10 s. The response was easily reproducible and did not decrease even after repeated stimulation over a period of 3–4 h. In some experiments, in which the vagus nerve was repeatedly stimulated for up to 5 min (stimulation regime, *t*=3 s; *U*=2.5 V; *d*=10 ms; *f*=9 Hz; followed by a stimulation-free interval of 5 s), an increase in blood flow was observed as long as the stimulation persisted (data not shown). Analysis of the video recordings of the artery leaving the rete mirabile showed that the small vessels behind the retia experienced a significant vasodilation (Fig. 3A), while the erythrocyte velocity increased from 0.6 to 1.2 mm s^{-1} ($P<0.05$; Fig. 3B) after vagal stimulation. Vasodilation was not seen close to or within the rete mirabile. Neither treatment with the α- and β-adrenergic antagonists, phentolamine and propranolol, nor with the muscarinic cholinoceptor antagonist atropine could abolish the effect (Fig. 4).

In all preparations with a high initial flow rate (>1.9 ml min^{-1}), vagotomy resulted in a substantial decrease in blood flow rate to the swimbladder (Fig. 5). In these preparations, brief mechanical squeezing of the vagus nerve also evoked a transient decrease in blood flow. The blood pressure in the dorsal aorta was not significantly affected. These effects were not observed in preparations with low swimbladder blood flow rate.

In blood-perfused swimbladder preparations, vagal stimulation caused a significant decrease (approximately 10 %) in perfusion pressure, which then slowly returned to pre-injection levels. Fig. 6A shows a single experiment with typical changes in perfusion pressure induced by vagal

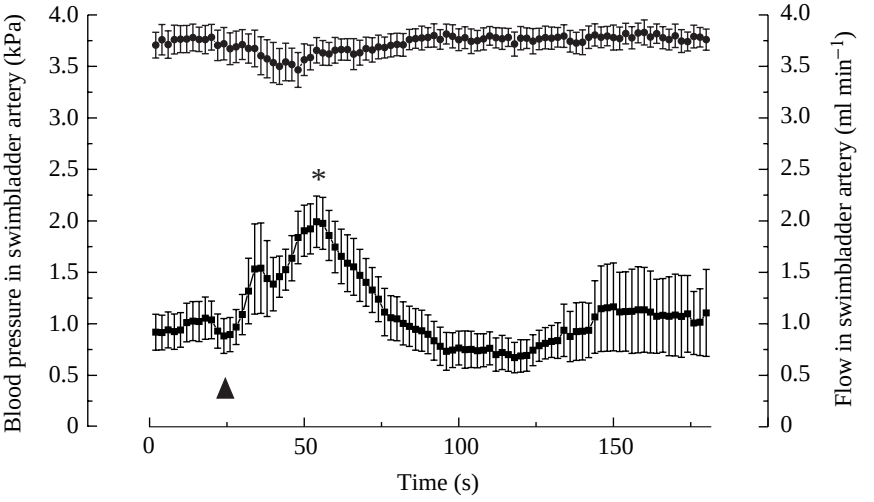

Fig. 2. Changes in blood pressure (circles) and flow rate (squares) in the swimbladder artery following stimulation of the vagus nerve. Values are mean ± S.E.M., *N*=11. The asterisk indicates a significant difference compared with the control value before vagus nerve stimulation. Stimulus duration, 10 s; frequency, 9 Hz; pulse width, 10 ms; voltage, 2.5 V. The onset of stimulation is indicated by the arrowhead.

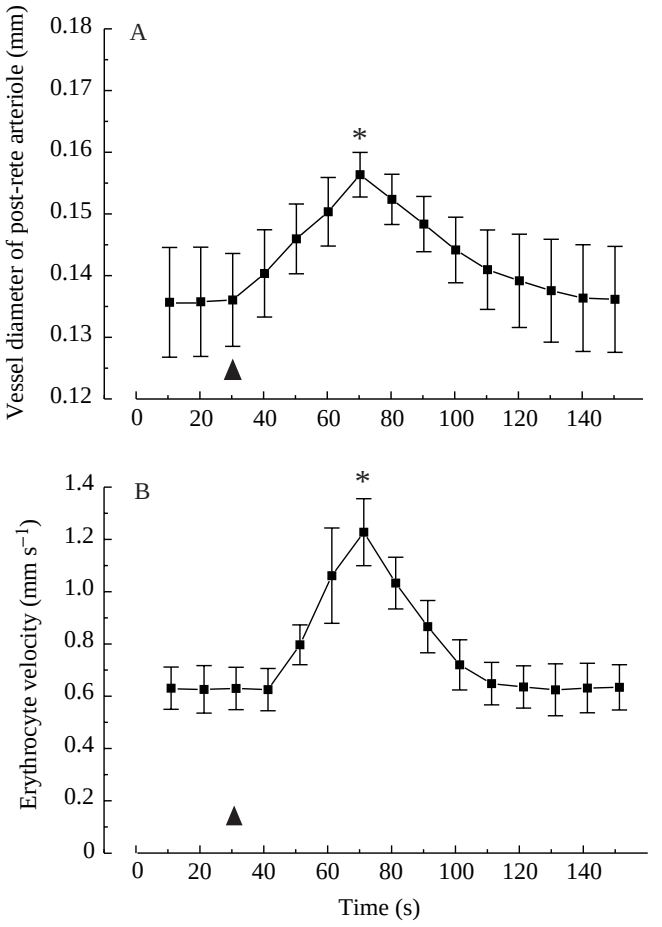

Fig. 3. Changes in post-rete arteriole vessel diameter (A) and erythrocyte velocity (B). Values are mean ± S.E.M., *N*=5. Asterisks indicate significant differences compared with control values before vagus nerve stimulation. Stimulus voltage, 2.5 V; stimulus duration, 10 s; frequency, 9 Hz; pulse width, 10 ms. The onset of stimulation is indicated by the arrowhead.

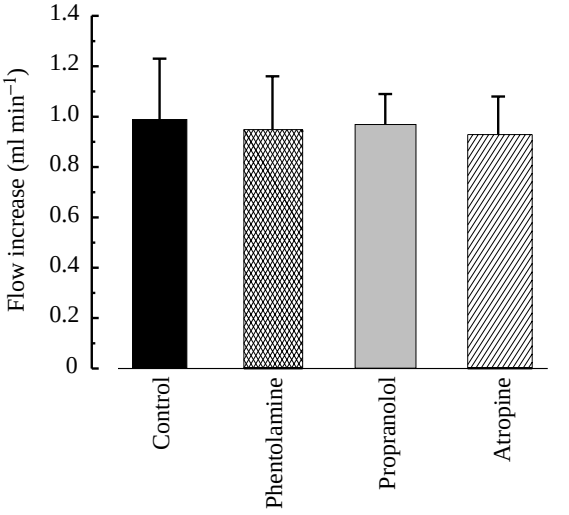

Fig. 4. Mean values for the increase in blood flow rate in the swimbladder artery induced by stimulation of the vagus nerve after preincubation with the α-adrenergic antagonist phentolamine, the β-adrenergic antagonist propranolol (both 60 nmol kg^{-1} body mass) or the muscarine receptor antagonist atropine (200 nmol kg^{-1} body mass). Values are means + S.E.M., *N*=6. The values do not differ significantly.

stimulation and by vagotomy. The changes in perfusion pressure after vagotomy were observed in approximately 30–40 % of all preparations. Fig. 6B presents the mean values for seven responding preparations after vagus nerve stimulation. Vagal stimulation evoked an immediate transient decrease in perfusion pressure. Again, the effect was not abolished after pretreatment with phentolamine, propranolol or atropine (results not shown). The injection of these antagonists caused a transient decrease in perfusion pressure, possibly reflecting the transient decrease in haematocrit. The same transient decrease in perfusion pressure was observed after

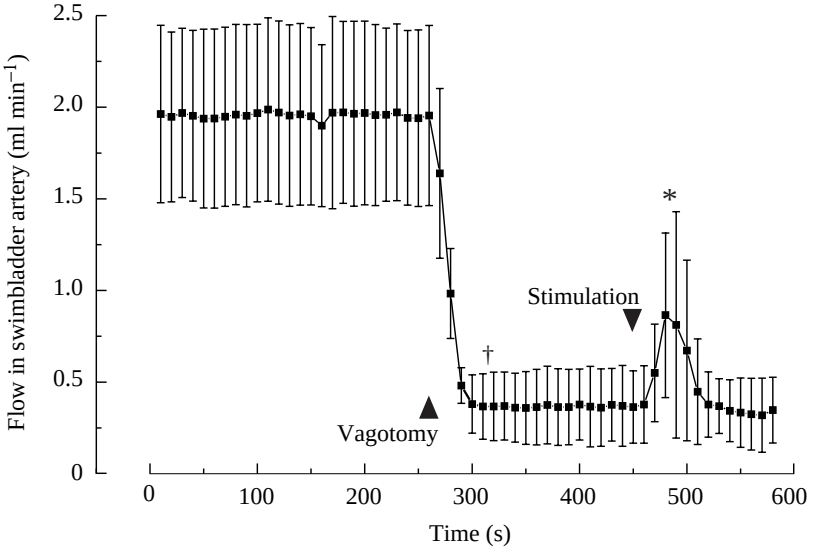

Fig. 5. Mean changes in swimbladder artery blood flow rate after vagotomy and subsequent vagus nerve stimulation. Stimulus duration, 5 s; frequency, 9 Hz; pulse width, 10 ms; voltage, 2.5 V. Values are means ± S.E.M., *N*=5. The dagger indicates a significant difference compared with the control value before vagotomy; the asterisk indicates a significant difference compared with the mean blood flow rate after vagotomy.

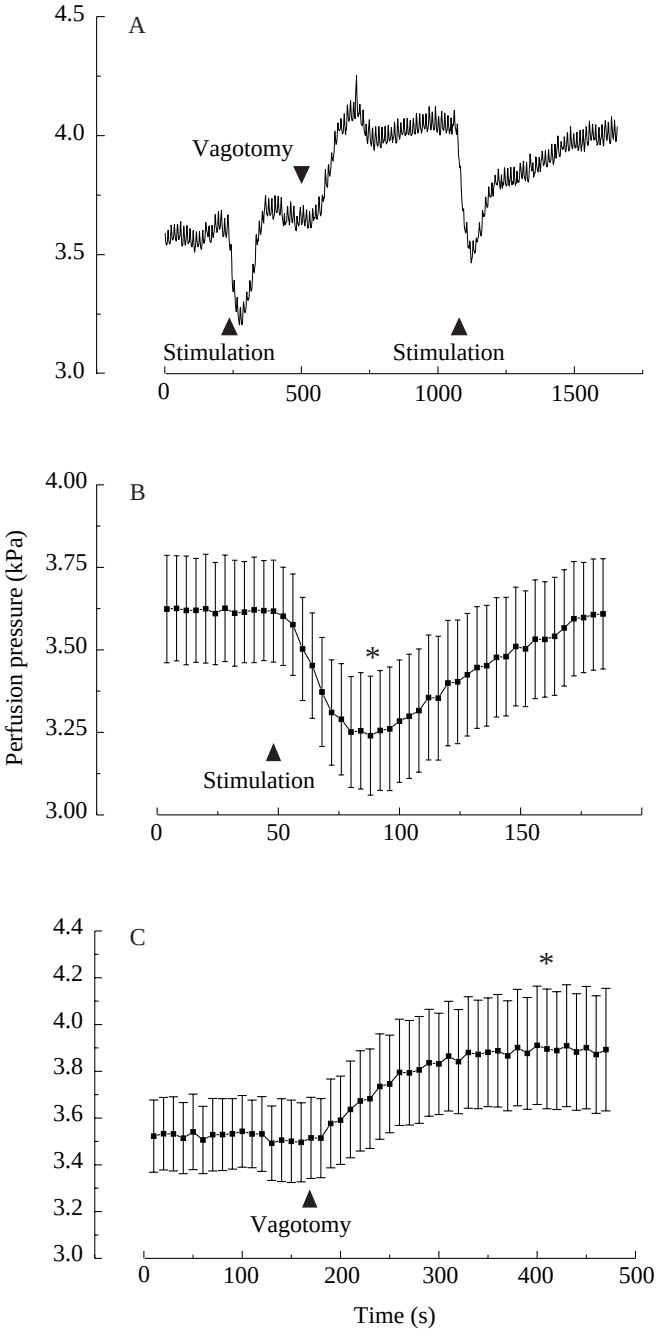

Fig. 6. (A) A single experiment with a blood-perfused swimbladder preparation showing the typical changes in perfusion pressure induced by vagus stimulation and by vagotomy. (B) Mean changes (N=7) in perfusion pressure in blood-perfused swimbladder preparations after vagus nerve stimulation. Stimulus duration, 10 s; frequency, 9 Hz; pulse width, 5 ms; voltage, 2.5 V. (C) Mean changes in perfusion pressure in blood-perfused swimbladder preparations after vagotomy (N=5). Values are mean ± S.E.M. Asterisks indicate significant differences compared with control values before vagotomy.

sham injections. Vagotomy induced a slow but significant increase in perfusion pressure of approximately 10 % (Fig. 6C).

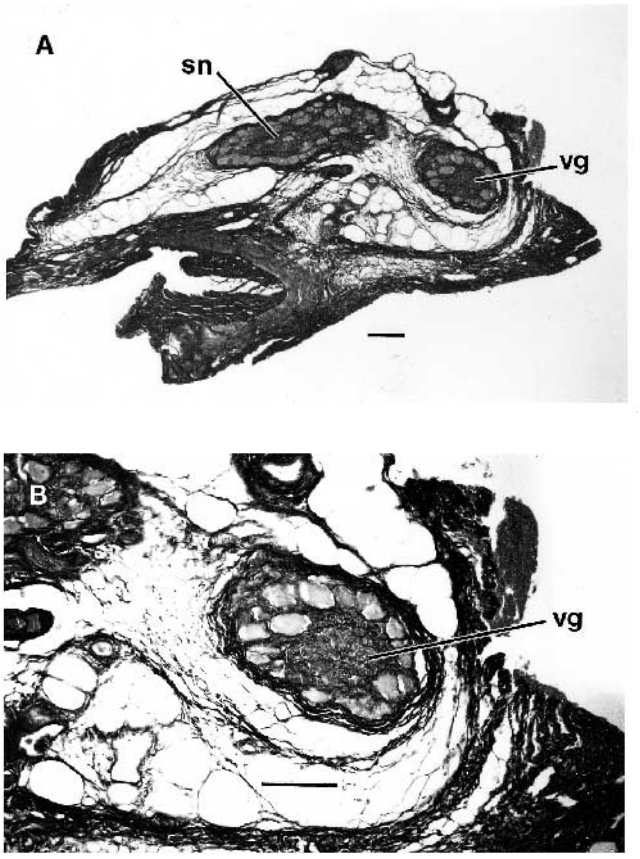

Fig. 7. (A,B) Photographs of Azan-stained 10 µm thick cross sections of the vagus nerve ganglion located at the proximal end of the resorbing part of the swimbladder (the location of this section within the ganglion is shown in the inset of Fig. 1). Owing to its location on the ventral side of the gut and the ventral view of the swimbladder obtained during preparation (see Fig. 1), the sections are presented with the ventral side up. (A) Overview; (B) close up of the vagus ganglion. sn, ganglion cells of the second nerve; vg, ganglion cells of the swimbladder ganglion of the vagus nerve. Scale bars, 100 µm.

The Azan-stained cross sections revealed a ganglion associated with the swimbladder (Fig. 7). The ganglion is located at the proximal end of the resorbing part of the swimbladder, at the connection of the ductus pneumaticus to the gut. On the basis of indirect evidence, we conclude that the vagal branch continuing to the swimbladder runs along the swimbladder artery and probably leaves the vessel approximately 8–10 mm in front of the rete mirabile. All attempts to place an occlusive cannula into the swimbladder artery at a distance of more than 8–10 mm from the rete mirabile caused a loss of the vagal stimulation effect, apparently because tying the cannula in place damaged the vagus nerve running next to the blood vessel. As shown in Fig. 7, the ganglion cells mix with ganglion cells of a second nerve and form a ganglion cell complex. Distal to the ganglion, the nerve fibres again separated from each other (Fig. 1). Stimulation of this second nerve did not evoke any changes in swimbladder blood flow or blood pressure (data not shown).

Discussion

This is the first study to use a combination of conventional pulsed Doppler flow measurements and vital video microscopy to study the control of swimbladder blood flow. Using these techniques, it was possible to study the effects of vagal stimulation on the blood flow pattern in the small vessels immediately distal to the rete mirabile.

Parasympathetic pathways typically have a ganglion close to, or even within, the effector organ. Histological examination of the vagus nerve of the European eel revealed a vagus ganglion on the anterior end of the resorbing swimbladder section. A peripheral vagal ganglion has also been described close to the swimbladder in the Atlantic cod *Gadus morhua* (Fahlén *et al.* 1965; McLean and Nilsson, 1981; Wahlqvist, 1985).

Electrical stimulation of the vagus nerve caused a vasodilation in the small blood vessels behind the rete mirabile. Accordingly, blood flow in the swimbladder artery and also in the capillary network behind the retia mirabilia increased during stimulation of the vagus. The decrease in perfusion pressure observed in blood-perfused swimbladder preparations during stimulation of the vagus also indicates a vasodilation of swimbladder vessels, resulting in a decrease in the resistance to blood flow. These results consistently indicate that electrical stimulation of the vagus causes a vasodilation of the swimbladder vessels coinciding with an increase in swimbladder blood flow. The slight pressure decrease observed in the dorsal aorta during vagal stimulation could therefore be a consequence of lower resistance in the swimbladder circulation.

In contrast to our results, Stray-Pedersen (1970) observed a vasoconstriction of the vessels in the secretory part of the swimbladder of the European eel during electrical stimulation of the ramus intestinalis of the vagus. In Stray-Pedersen's study, the period of stimulation was relatively long (10–30 s), with a voltage of 10–20 V. Fänge (1953) demonstrated that electrical stimulation of the vagus over several minutes caused a contraction of the secretory mucosa, while the mucosa of the pneumatic duct relaxed. This suggests that prolonged stimulation of the vagus may provoke a contraction of the smooth muscle cells of the muscularis mucosa, causing a decrease in blood flow due to vascular compression. In the present study, we used a much shorter stimulation regime, and in our experiments contractions of the mucosa were not observed. Vasodilation, however, was seen within seconds after stimulation, which suggests a direct effect on vascular smooth muscle cells. Thus, the explanation for the differences between our results and those of Stray-Pedersen (1970) may well be the differences in the stimulation regime.

While stimulation of the vagus nerve caused a vasodilation, vagotomy *in situ* caused a striking decrease in blood flow in preparations with a high initial blood flow and an increase in vascular resistance, as indicated by the increased perfusion pressure in the blood-perfused preparations. A convincing explanation emerging from these results is that a vagal vasodilator tonus affects the swimbladder blood flow rate and that the blood vessels constrict in the absence of that tonus.

In the *in situ* preparations, as well as in the blood-perfused preparations, electrical stimulation of the vagus always induced a vasodilation, but it was not possible to achieve the prevagotomy levels of swimbladder blood flow or perfusion pressure (Fig. 5). The reason for this could be that electrically stimulating the whole fibre bundle *in situ* is too different from the *in vivo* signal controlling the swimbladder vessels.

The observation that neither the α- and β-adrenergic antagonists, phentolamine and propranolol, nor the muscarinic receptor antagonist atropine abolished the effects of vagal stimulation indicates that the vagal innervation is neither adrenergic nor cholinergic. Immunohistochemical as well as physiological studies using strips of smooth muscle tissue have previously demonstrated the importance of non-adrenergic, non-cholinergic (possibly peptidergic) innervation in the control of swimbladder function (Lundin and Holmgren, 1989; Lundin, 1991*a*,*b*). Evidence for the presence of substance-P-like material has been found in several teleosts. Substance P is a potent vasodilator but, applied exogenously to swimbladder smooth muscle, it has an excitatory effect (Lundin, 1991*a*,*b*). Vasoactive intestinal polypeptide (VIP) induced a relaxation of swimbladder smooth muscle in cod and eel, for example, but no VIP-immunoreactive fibres were observed close to the blood vessels (Lundin, 1991*a*,*b*). VIP induced only a slight vasodilation in saline-perfused preparations of the cod swimbladder (Lundin and Holmgren, 1984). Preliminary experiments also demonstrated only a slight vasodilation in swimbladder vessels of the European eel following VIP application, and similar effects were obtained with the NO donator sodium nitroprusside (T. Schwerte and B. Pelster, unpublished results). Surprisingly, however, VIP application *in vivo* impaired swimbladder filling (Lundin and Holmgren, 1991); vasodilation would be expected to have the opposite effect. The actual contribution of VIP to swimbladder control *in vivo* clearly requires further clarification.

These pharmacological results again stress the differences between our study and those using prolonged electrical stimulation. While neither cholinergic nor adrenergic antagonists could block the vagus-induced vasodilation in our study, previous studies have shown that prolonged electrical stimulation of the vagus branches caused mainly adrenergic effects and perhaps some weak cholinergic effects (Stray-Pedersen, 1970; Fänge, 1953).

The physiological role of the vagus in the control of swimbladder function was clearly demonstrated by Bohr (1894) in studies showing a decrease in the rate of gas deposition into the swimbladder following vagotomy (see also Jakobs, 1930, 1933; Fänge 1953, 1966). This result has been interpreted as a putative direct effect of cholinergic innervation on the gas gland cells, increasing the metabolic activity of these cells (Ewart and Driedzic, 1990). The results of the present study provide further details of the mechanism involved. Vagotomy effectively reduces the swimbladder blood flow rate, which is an important variable determining the rate of gas

deposition into the swimbladder (Pelster and Scheid, 1992). Furthermore, the vagus tonus, causing a decrease in vascular resistance, antagonizes the α-adrenergically controlled resistance increment described by Pelster (1994). Besides this vagus tonus, which may be a very effective vasodilator, only weak vasodilatory effectors have been described for swimbladder vessels. It is therefore possible that a vasodilation of the swimbladder vessels, which is essential for the filling of the swimbladder with gases, is achieved mainly *via* the tonus of the vagus nerve.

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