

THE FISH BULBUS ARTERIOSUS IS NOT MERELY A "WINDKESSEL"

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The bulbus arteriosus (BA) connects the cardiac ventricle of teleosts and lampreys to the ventral aorta; it expands during cardiac systole and rebounds, pushing blood into the ventral aorta, during diastole. The result of this "windkessel effect" is a smoothing of blood flow to the delicate gill vessels. The BA is extremely distensible (30 X the mammalian aorta) and may store transiently 25-100% of the cardiac output (e. g., Olson, K. in: *"The Physiology of Fishes"*, Ed., D.H. Evans, CRC Press, pg. 129-154, 1998). The BA is innervated (Holmgren, S., *Acta Physiol. Scand.* 99: 62-74, 1977; Watson, A.D. and Cobb, J.L.S., *Cell Tiss. Res.* 196: 337-346, 1979), and adrenergic and cholinergic agonists increased and decreased, respectively, the compliance of the BA of trout (Farrell, A., *J. Exp. Zool.* 209: 169-173, 1979). In addition, acetylcholine has been shown to contract trout BA spiral strips (Klaverkamp, J.F. and Dyer, D.C. *Fur. S. Pharmac.* 28: 25-34, 1974). Immunoreactive ANP has been localized in the BA of a variety of freshwater fishes (Kim, S.Z. et al., *Comp. Biochem. Physiol. [B]* 100: 575-578, 1991), and a high density of receptors for natriuretic peptides has been described in the BA of the conger eel (Cerra, M.C. et al., *J. Exp. Zool.* 263: 215-219, 1992). These studies suggest that at least the teleost BA is controlled by a variety of neurotransmitters and paracrine and may be more than a simple windkessel. To test this hypothesis, we used techniques described previously (e.g., Evans, D.H. and Gunderson, M.P., *Am. J. Physiol.* 274: R1050-R1057, 1998; Evans, D.H. and Gunderson, M.P. *Bull. MDIBL* 37: 107, 1998; Evans, D.H. and Harrie, A.C., this volume) for the capture and maintenance of lampreys (*Petromyzon marinus*) and eels (*Anguilla rostrata*) and preparation of vascular rings from the BA. Resting tensions of 500 mg were set and maintained in each species before the sequential addition of putative vasoactive agonists. Data are expressed as mean tension change \pm S.E. (N).

Agonist	ET-1 0.1 μ M	ACh 0.1 mM	SNP 0.1 mM	NO 10 μ M Initial	NO Final	pCNP 0.1 μ M	Carb 1 μ M	PGE1 1 μ M
Lamprey	976 \pm 162 (20)	61.6 \pm 1 1 (15)	36.1 \pm 8.5 (10)	19.4 \pm 6.5 (16)	-54.9 \pm 12 (16)	-319 \pm 143 (11)	-46.7 \pm 14 (12)	-1141 \pm 185 (6)
Eel	646 \pm 80 (8)	(8) no response	-48 \pm 28 (8) (NS)	-28 \pm 8.9 (8)	no change	-178 \pm 37** (8)	-4.6 \pm 3.1 (8) (NS)	-377 \pm 55 (8)

ET = endothelin; ACh = acetylcholine; SNP = sodium nitroprusside; NO = nitric oxide, pCNP = porcine C-type natriuretic peptide; Carb = carbaprostacyclin; PGE1 = prostaglandin E; ** eel-atrial natriuretic peptide

It is clear that the bulbus arteriosus in both species is sensitive to a wide variety of substances that are vasoactive in arteries and veins in vertebrates, including fishes. ET is especially constrictive, producing 100-200% increases in tension in the eel and lamprey, respectively. ACh produced small, but significant, contraction in the lamprey BA, but had no effect on the eel BA. ACh did contract the eel ventral aorta (Evans, D.H. and Gunderson, M.P. *Bull. MDIBL* 37: 107, 1998). The NO-donor, sodium nitroprusside, contracted the lamprey aorta slightly, but significantly, and the response to NO itself was biphasic, with an initial contraction, followed by a more significant dilation. This biphasic response to NO was also seen in the lamprey aorta (Evans, D.H. and Harrie, A.C., this volume) and is unexplained. SNP did not produce significant dilation in the eel BA, but NO did, and the response was not biphasic. pCNP produced significant dilation in the lamprey BA, as it did in the aorta (Evans, D.H. and Harrie, A.C., this volume). The eel BA dilated significantly when eel ANP was applied. The BA's of both species were only marginally sensitive to the PGI₂-agonist carbaprostacyclin, but extremely sensitive to the E-type prostaglandin, PGE₁. (Supported by NSF IBN-9604824 and REU NSF BIR 9531348)