Cardiac preload and venous return in swimming sea bass (Dicentrarchus labrax L.)

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Summary

Cardiac preload (central venous pressure, PCV), mean circulatory filling pressure (MCFP), dorsal aortic blood pressure (PDA) and relative cardiac output (\( Q \)) were measured in sea bass (Dicentrarchus labrax) at rest and while swimming at 1 and 2 \( BL \) s\(^{-1} \). MCFP, an index of venous capacitance and the upstream venous pressure driving the return of venous blood to the heart, was measured as the plateau in PCV during ventral aortic occlusion. Compared with resting values, swimming at 1 and 2 \( BL \) s\(^{-1} \) increased \( Q \) (by 15±1.5 and 38±6.5%, respectively), PCV (from 0.11±0.01 kPa to 0.12±0.01 and 0.16±0.02 kPa, respectively), MCFP (from 0.27±0.02 kPa to 0.31±0.02 and 0.40±0.04 kPa, respectively) and the calculated pressure gradient for venous return (\( \Delta P_V \), from 0.16±0.01 kPa to 0.18±0.02 and 0.24±0.02 kPa, respectively), but not PDA. In spite of an increased preload, the increase in \( Q \) was exclusively mediated by an increased heart rate (fH, from 80±4 beats min\(^{-1} \) to 88±4 and 103±3 beats min\(^{-1} \), respectively), and stroke volume (\( V_s \)) remained unchanged. Prazosin treatment (1 mg kg\(^{-1} \) \( M_b \)) abolished pressure and flow changes during swimming at 1 \( BL \) s\(^{-1} \), but not 2 \( BL \) s\(^{-1} \), indicating that other control systems besides an \( \alpha \)-adrenoceptor control are involved. This study is the first to address the control of venous capacitance in swimming fish. It questions the generality that increased \( Q \) during swimming is regulated primarily through \( V_s \) and shows that an increased cardiac filling pressure does not necessarily lead to an increased \( V_s \) in fish, but may instead compensate for a reduced cardiac filling time.

Key words: cardiac preload, cardiac output, exercise, heart rate, mean circulatory filling pressure, prazosin, sea bass, stroke volume, teleost, venous capacitance, venous return.

Introduction

Although it is well known that fish increase cardiac output (\( Q \)) during exercise to meet increased metabolic demands, there is a controversy concerning the basic hemodynamic mechanisms underlying this increase. One view is that stroke volume (\( V_s \)) and, to a lesser extent, heart rate (fH) are responsible for the rise in \( Q \) in swimming fish, especially in salmonids (Dunnall and Schreer, 2003; Farrell, 1991; Farrell and Jones, 1992; Jones and Randall, 1978; Kiceniuk and Jones, 1977). This view has been questioned by others, who suggest that increased fH is the primary means of increasing \( Q \) during swimming (Altimiras and Larsen, 2000; Axelsson et al., 1994; Cooke et al., 2003; Korsmeyer et al., 1997; Lêfrancois et al., 1998; Priede, 1974). While this difference in opinion may exist because different species do indeed have different strategies for increasing \( Q \), we know little of one of the primary determinants of \( V_s \) in fish, i.e., cardiac preload (= central venous pressure, PCV). Ventricular end-systolic volume, at least in rainbow trout, is close to zero and hence cannot be reduced much further during exercise (Forster and Farrell, 1994; Franklin and Davie, 1992). Thus, any increase in \( V_s \) has to come about by increasing end-diastolic volume, which must come about through increased PCV and a concurrent increase in the myocardial force of contraction due to the Frank-Starling relationship (Farrell, 1991; Farrell and Jones, 1992). Consequently, without information on the changes in PCV during swimming it is difficult to evaluate fully the role of increasing \( V_s \).

This line of logic ignores the fact that under steady-state conditions, \( Q \) equals venous return and the heart can pump only what it gets back from the venous circulation. Moreover, any increase in PCV during exercise will reduce the pressure gradient for venous return (flow) to the heart from the venous periphery, if end-capillary blood pressure and venous resistance remain unaltered. Thus, in addition to increasing cardiac filling pressure, a proportional increase in peripheral venous pressure is expected to ensure that venous return can
match the increase in $Q$. Venous return can be estimated from measurements of $PCV$ and venular blood pressures.

The mean circulatory filling pressure (MCFP) is an index of venous capacitance. It is measured as the venous pressure after a short (5–10 s) cardiac arrest and also represents the upstream venous (venular) pressure that drives venous return (Pang, 2001; Rothe, 1986, 1993; Sandblom and Axelsson, 2005). MCFP can increase due to an increased smooth muscle tone and/or a decreased compliance in venous capacitance vessels (Conklin et al., 1997; Hoagland et al., 2000; Olson et al., 1997; Pang, 2001; Rothe, 1986, 1993; Zhang et al., 1998). While comprehensive studies have examined the nervous and humoral control of venous capacitance in unaesthetized fish under resting conditions (Conklin et al., 1997; Hoagland et al., 2000; Olson et al., 1997; Sandblom and Axelsson, 2005; Zhang et al., 1998), none have considered the changes in venous capacitance that are likely to occur during exercise. Furthermore, basic information on $PCV$ during exercise is limited to a few studies and what data exist are compromised by noisy signals and/or experiments on a small number of animals (Jones and Randall, 1978; Kiceniuk and Jones, 1977; Stevens and Randall, 1967).

The primary objective of this study was therefore to measure changes in $PCV$ and MCFP in a fast swimming teleost, the European sea bass ($Dicentrarchus labrax$ L.). By combining $PCV$ and MCFP measurements, it was also possible to assess the degree to which the pressure gradient for venous return, venous capacitance and cardiac preload change during the periods of increased $Q$ associated with exercise. Also, the role of $\alpha$-adrenoceptor control of these responses was examined, since Zhang et al. (1998) identified that venous capacitance in resting rainbow trout ($Oncorhynchus mykiss$) can be altered by $\alpha$-adrenergic mechanisms.

**Materials and methods**

**Animals**

Results from 13 sea bass (279–648 g and 31–38 cm) are presented in this study. The fish were obtained from the Ferme Marine des Baleines (Ile de Ré, France) and maintained at CREMA under a natural photoperiod in indoor 400 l fibre glass tanks supplied with recirculating, biofiltered seawater at ambient temperature (21–24°C). The fish were fed commercial dry pellets on a regular basis.

**Surgical procedures**

Prior to surgery the fish were anaesthetized in seawater containing MS-222 (approx 100 mg l$^{-1}$) and placed on watersoaked foam on a surgery table. During surgery, the fish was covered with wet tissue paper and the gills were continuously irrigated with aerated, chilled (-11–16°C) seawater containing MS-222 (-50 mg l$^{-1}$). The ventral aorta was exposed with an incision on the right side of the isthmus and dissected free. A Perspex cuff-type 20 MHz Doppler flow probe (Iowa Doppler products; Iowa City, IA, USA), with an inner diameter of 1.8–2.0 mm, was positioned around the aorta proximal to the bulbus to measure relative changes in $Q$ (Fig. 1). Also, a cuff-type vascular occluder (i.d. 2.5–4.3 mm) was positioned posterior to the flow probe to obtain zero-flow during measurement of MCFP. The occluder was constructed from Perspex, a heat-flared water-filled PE-50 catheter and a piece of latex rubber (model Thin, Dental Dam, Coltène/Whaledent Inc, USA and Canada). The rubber was tied with a 4–0 suture holding the vessel. A 4–0 suture was used to close the vessel wall was lifted with forceps and secured with a 4–0 suture, allowing the vessel to be gently lifted during the cannulation procedure. This procedure prevents blood loss, and is an improvement on the method used by Farrell and Clutterham (2003). A PE-50 catheter, with 2–3 side-holes to keep it patent and a bubble 1 cm from the tip, was inserted into the ductus of Cuvier through a small incision made close to the suture holding the vessel. A 4–0 suture was used to close the vessel wall around the catheter, leaving the bubble located on the luminal side of the vessel wall. The catheter was secured with silk sutures to the skin. The third efferent branchial artery on the left side was occlusively cannulated to measure dorsal aortic pressure ($P_{DA}$). To do this, the first two gill arches were retracted to expose the third gill arch, which was gently

![Fig. 1. Arrangement of the Doppler flow probe (Q) and vascular occluder (VO) on the ventral aorta, and catheters for blood pressure measurements in sinus venosus (PCV) and the efferent branchial artery (PDA).](image-url)
Venous return in swimming sea bass

retracted. The branchial artery was dissected free close to the angle of the branchial arch, occluded upstream with a 4–0 silk suture and cannulated downstream with a tapered PE-50 catheter (Fig. 1). The catheter was secured with 3–0 silk sutures around the gill arch and secured to the skin with 3–0 silk sutures. The catheters and the lead from the flow probe were collectively secured with a 3–0 suture to the back of the fish. Both blood pressure catheters were filled with physiological saline (0.9% NaCl). Following surgery, the fish were revived in fresh seawater, placed in plastic floating tubes in the holding tanks and allowed a 24·h recovery before experiments commenced.

Experimental protocol

A stainless steel, Brett-type swimtunnel respirometer was used in the present study. This tunnel had been designed to exercise individual fish in a non-turbulent water flow with a uniform cross-sectional water velocity. The total water volume was 48·l and the swim chamber had a square cross-sectional area of 290·cm². A propeller downstream of the swim chamber generated water flow. The flow in the swimtunnel was calibrated (Marsh-McBirney 200 flow meter; Frederick, MD, USA) in cm s⁻¹, which was converted to swimming speeds in body lengths s⁻¹ (BL s⁻¹). The respirometer was thermostatted by immersion in a large outer stainless steel tank that received a flow of aerated water. Since venous pressure is low readings can be affected by any changes in the pressure head on the propeller in the swim-channel when water velocity is changed.

To minimise this problem the lid to the swim chamber was removed. Water pressure was measured with a saline-filled catheter immersed in the channel, and no pressure fluctuations were observed at the swimming speeds used (up to 2 BL s⁻¹). The swim channel was covered with an opaque black plastic sheet to avoid visual disturbance of the fish.

All experiments were conducted at a temperature of 22°C. The experimental protocol started with a 2 ·min recording at rest, i.e., the fish oriented into a low water velocity and maintaining position without swimming. MCFP was measured at rest by occluding the ventral aorta for 8·s. Water velocity was then gradually increased over a 5·min period until the fish reached a swimming speed of 1·BL·s⁻¹, which was maintained for 15·min. MCFP was remeasured at the end of this period. The same procedure was used for a swimming speed of 2·BL·s⁻¹. Water velocity was then gradually increased over a 5 min period until the fish reached a swimming speed of 1·BL·s⁻¹, which was maintained for 15 min. MCFP was remeasured at the end of this period. The same procedure was used for a swimming speed of 2·BL·s⁻¹. Water velocity was then reduced to the resting condition over a 2·min period and an α-adrenoceptor antagonist (prazosin, 1·mg·kg⁻¹ Mb; Pfizer, Sandwich, UK) was administered via the venous catheter. The entire protocol was repeated 1.5–2.0·h later. Preliminary experiments had revealed that an exercise period of 15·min was well beyond the time necessary to establish stable cardiovascular measurements in untreated fish.

Successful ventral aortic occlusion always resulted in a rapid fall in $P_{DA}$, a rise in $P_{CV}$ and a complete cessation of ventral aortic flow (Fig. 2). Although the ventral aorta was easily accessible, the vessel proved to be relatively fragile as compared with rainbow trout and several sea bass died due to

![Fig. 2. Representative original recordings during ventral aortic occlusion in a resting 530 g sea bass (Dicentrarchus labrax). Before (left-hand panel) and 1.5–2 h after (right-hand panels) prazosin treatment (1 mg kg⁻¹ Mb). (A) Dorsal aortic pressure ($P_{DA}$); (B) central venous pressure ($P_{CV}$); (C) relative cardiac output in kHz ($Q$); and (D) heart rate ($f_H$). Mean circulatory filling pressure (MCFP) was calculated from the average venous plateau pressure between the 5th and 7th seconds during the ventral aortic occlusion.](image-url)
fatal rupture of the ventral aorta during occlusion. This generally occurred after the first occlusion and might have been due to mechanical abrasion from the vascular occluder during recovery. Another unusual finding was the low occurrence of blood clotting in the catheters during routine surgery and this was the reason why heparin was omitted from the saline.

Data acquisition, calculations and statistical analysis

Both blood pressure catheters were connected to pressure transducers (model DPT-6100, pvb Medizintechnik, Kirchseeon, Germany), calibrated against a static water column with the zero reference pressure. The signals from the pressure transducers were amplified with a 4ChAmp amplifier (Somedic, Hörby, Sweden). Relative cardiac output ($Q$) was recorded with a directional-pulsed Doppler flow meter (model 545C-4, University of Iowa, USA). The digital signals were fed into a portable computer running a custom made program, General Acquisition (Labview version 6.01, National Instruments, Austin, TX, USA).

Heart rate ($f_H$) was obtained from either pulsatile pressure or flow records. Relative changes in cardiac stroke volume ($V_s$) were calculated as $Q/f_H$. Total systemic resistance ($R_{sys}$) was calculated from the pressure drop in the circulation divided by cardiac output $R_{sys}=(P_{DA}–P_{CV})/Q$. For the $V_s$, $Q$ and $R_{sys}$ calculations raw data was used and the initial untreated resting value for each fish was arbitrarily set to 100%. The plateau pressure in $P_{CV}$ during ventral aortic occlusion was assumed to equal mean circulatory filling pressure (MCFP), and the average pressure between the 5th and 7th seconds of ventral aortic occlusion is reported here. This time interval is sufficient to obtain reasonably steady plateau values, while minimising the compromising effects of barostatic reflexes (Sandblom and Axelsson, 2005). A similar method has been used previously to measure MCFP in the trout (Hoagland et al., 2000; Zhang et al., 1998). No correction for inequalities in blood remaining in the

![Fig. 3.](image)
Venous return in swimming sea bass

arterial circulation after occlusion was performed, because the difference was assumed to be negligible due to the large differences in compliances and volumes of the arterial and the venous compartments (Rothe, 1993; Zhang et al., 1998). The pressure gradient ($\Delta P$) for venous return was calculated as

$$\Delta P_V = MCFP - P_{CV}. \quad (1)$$

Resistance to venous return ($R_v$) was calculated as

$$R_v = (MCFP - P_{CV})/Q, \quad (2)$$

assuming that $\dot{Q}$ equals venous return.

Data (mean values ±S.E.M.) are presented only for fish that performed steady state swimming at both water velocities. Partial data from fish that exhibited either erratic swimming behaviours, burst swimming, died after prazosin treatment, lost a catheter, or the ventral aorta ruptured were discarded. Cardiovascular data were taken from the last 90 s of each exercise period. For the calculations of venous variables, i.e. Fig. 4, $P_{CV}$ was taken as the average of a 30 s period before ventral aortic occlusion. Wilcoxon matched pairs signed-ranks test, with a fiduciary level of $P_{1/100}$ was used to evaluate statistically significant differences in cardiovascular variables at different swimming speeds and between treatments. To compensate for multiple two-group comparisons, a modified Bonferroni-test was applied (Holm, 1979).

**Results**

**Cardiovascular responses to swimming in untreated fish**

Fig. 2 shows representative resting cardiovascular recordings for an individual fish during ventral aortic occlusion. Fig. 3 summarises the mean data for fish at rest and during steady state swimming at 1 and 2 $BL$ s$^{-1}$, while Table 1 shows changes in $\dot{Q}$, $V_s$, and $f$ for individual fish. Exercise increased $\dot{Q}$ by 15±1.5% and 38±6.5% of the resting value, respectively, at 1 and 2 $BL$ s$^{-1}$. These increases in $\dot{Q}$ were mediated by significant increases in $f$ from 80±4 beats·min$^{-1}$ at rest to 88±4 beats·min$^{-1}$ at 1 $BL$ s$^{-1}$ and 103±3 beats·min$^{-1}$ at 2 $BL$ s$^{-1}$. By contrast, $V_s$ remained unaltered during exercise. Nevertheless, the increases in $\dot{Q}$ were associated with significant rises in $P_{CV}$ from 0.11±0.01 ·kPa at rest to 0.12±0.01·kPa at 1 $BL$ s$^{-1}$ and 0.16±0.02 ·kPa at 2 $BL$ s$^{-1}$. However, $P_{DA}$ and $R_{sys}$ were unchanged during exercise.

**Effects of $\alpha$-adrenoceptor blockade on cardiovascular performance during swimming**

Blockade of $\alpha$-adrenergic receptors with prazosin showed that the cardiovascular system is at least partially controlled via $\alpha$-adrenoceptors, both at rest as well as during exercise (Figs 3 and 4). At rest, prazosin treatment significantly decreased $R_{sys}$, producing a significant arterial hypotension while $P_{CV}$ increased significantly.

During swimming after prazosin treatment, the increases in $P_{CV}$ and $f$ were absent at 1 $BL$ s$^{-1}$, but not at 2 $BL$ s$^{-1}$. In fact, the increase in $P_{CV}$ was accentuated significantly at 2 $BL$ s$^{-1}$.

### Table 1. Changes in cardiac output, heart rate and stroke volume from nine individual sea bass, *Dicentrarchus labrax*, at rest and while swimming at 1 and 2 $BL$ s$^{-1}$

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<tr>
<th>Fish</th>
<th>$\dot{Q}$ (% Rest)</th>
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<th>$\dot{Q}$ (% 2 $BL$ s$^{-1}$)</th>
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after prazosin. Prazosin also significantly accentuated the decrease in $R_{sys}$ at $2\text{ BL s}^{-1}$ (from the resting value of $85.7\pm3.2$ to $73.5\pm3.6\%$) and the corresponding hypotension ($P_{DA}$ from $2.9\pm0.1$ to $2.5\pm0.1$ kPa). Again, $V_s$ remained unchanged throughout the swim challenges after prazosin treatment despite the increase in $P_{CV}$ (Fig. 3). After prazosin, MCFP also remained unaltered at $1\text{ BL s}^{-1}$, but increased significantly from $0.28\pm0.02$ kPa at rest to $0.40\pm0.05$ kPa during swimming at $2\text{ BL s}^{-1}$, but no more so than before prazosin treatment (Fig. 4). As a result $\Delta P_{V}$ increased significantly compared with the resting value at $2\text{ BL s}^{-1}$, although this response was not as pronounced as pre-prazosin treatment. $R_{v}$ remained unaltered throughout the experiment (Fig. 4).

**Discussion**

In its natural pelagic habitat, sea bass are subjected to strong water currents requiring long periods of sustained exercise. This ecological fact makes sea bass an excellent experimental animal for exercise studies. However, existing cardiovascular data of sea bass is sparse. Axelsson et al. (2002) reported a resting $f_{H}$ of $51$ beats min$^{-1}$ at $16^\circ\text{C}$. In the present study at $22^\circ\text{C}$, $f_{H}$ was $80$ beats min$^{-1}$ at $16^\circ\text{C}$. In the present study at $22^\circ\text{C}$, $f_{H}$ was $80$ beats min$^{-1}$, a difference that would represent a $Q_{10}$ of $2.1$ between the two studies and suggests that the difference was simply a temperature effect. Furthermore, judging from the variable heart rate in resting fish (Fig. 2), it is likely that the fish had a functional cholinergic tone, which is indicative of an acceptable decay in post-surgical stress (Campbell et al., 2004).

**Hemodynamics of venous return and cardiac filling pressure in sea bass**

The present study is the first to demonstrate an active control of the venous vasculature during exercise in any species of fish.

**Fig. 4.** Upper trace shows a representative recording of venous pressure during a full swim protocol (~30 min) in an untreated sea bass, *Dicentrarchus labrax*. Ventral aortic occlusions for MCFP measurements at the end of each swim speed (rest, $1\text{ BL s}^{-1}$ and $2\text{ BL s}^{-1}$, respectively) are marked with vertical arrows. Lower panels (A–D) show mean values (+ S.E.M.) of measured and calculated venous variables at rest and while swimming at $1$ and $2\text{ BL s}^{-1}$. Filled bars represent untreated fish ($N=8–9$) and open bars ($N=7$) represent fish after treatment with prazosin ($1\text{ mg kg}^{-1} \text{Mb}$). The variables are $A$, central venous pressure ($P_{CV}$); $B$, mean circulatory filling pressure (MCFP); $C$, pressure gradient for venous return ($\Delta P_{V}$) and $D$, resistance to venous return ($R_{v}$). *Statistically significant difference ($P<0.05$) from resting values within each treatment and statistically significant difference ($P<0.05$) between values before and after prazosin treatment. For a detailed description on calculations of mean values, see Materials and methods section.
affected MCFP as well. The importance of an α-adrenergic tone on the arterial side of the circulation has been previously demonstrated at rest and during swimming in other fish species (Axelsson and Fritsche, 1991; Axelsson and Nilsson, 1986; Smith, 1978), and was confirmed here because after prazosin treatment sea bass could not maintain $R_{sys}$ at $2 \text{BL s}^{-1}$ and suffered a major systemic hypotension. In view of this, it could be argued that the increase in $Pcv$ was only a consequence of the reduction in $R_{sys}$, but then MCFP would not have increased. Instead, the accentuated reduction in arterial pressure at $2 \text{BL s}^{-1}$ possibly triggered the activation of some unknown vasoactive system, acting primarily on the venous vasculature. One concern with the present study is that an increased adrenergic tone on resistance vessels may have counteracted a further decrease in $R_{sys}$ during exercise and resulted in an unaltered resistance in untreated fish. It is unknown whether the adrenergic control of MCFP is mediated by adrenergic nerves and/or circulating catecholamines. In cod (Axelsson and Nilsson, 1986; Butler et al., 1989; Smith et al., 1985) and trout (Smith, 1978) it has been demonstrated that the increase in arterial blood pressure observed during moderate exercise is exclusively mediated by adrenergic nerves. Whether the same is true for the venous circulation in fish is not yet known.

A possible consequence of a decreased venous capacitance, manifested as the increase in MCFP, is that blood volume from the venous compartment is redistributed to other parts of the circulation, such as muscle capillary beds, respiratory organs and central veins (Pang, 2001). It is possible that much of the blood redistributed from the venous system in the sea bass during exercise, in addition to increasing cardiac preload, served to fill gill vasculature and muscle capillary beds. In mammals the splanchnic circulation has a high capacitance and is the primary reservoir for blood volume mobilization during exercise (Pang, 2001; Rothe, 1986). To what extent splanchnic venous blood volume is mobilized during exercise in fish is unclear, even though Stevens and Randall (1967) demonstrated that blood flow in the subintestinal vein (e.g. portal vein) decreased and venous pressure increased in rainbow trout. Albeit somewhat meager evidence, the observations are consistent with blood volume mobilization from the splanchnic venous compartment during exercise in fish. As judged from the drop in $PDA$ and $R_{sys}$ during swimming at $2 \text{BL s}^{-1}$ after prazosin, it is possible that the gut circulation continued to be perfused (unlike the normal decrease with exercise) as perfusion of locomotory muscles increased (Axelsson and Fritsche, 1991; Axelsson et al., 2000; Farrell et al., 2001; Thorarensen et al., 1993). Further research in this area is clearly needed.

**Heart rate versus stroke volume regulation during exercise**

Increased $Q$ observed after force feeding in sea bass was due primarily to tachycardia (Axelsson et al., 2002). Similarly, in the present experiments, sea bass increased $Q$ through tachycardia with no significant change in $V_s$, despite the fact that cardiac preload increased significantly (Fig. 3, Table 1). This shows that an increased cardiac preload does not
necessarily lead to an increased \( V_s \) in fish, but may instead compensate for a reduced cardiac filling time associated with an increase in \( f_t \). Thus, within the scope of the present exercise challenge, \( \dot{Q} \) in sea bass was frequency regulated.

Other studies on fish with various life-strategies also suggest that control of \( \dot{Q} \) by \( f_t \) during exercise, might be more important than previously thought (Farrell, 1991; Farrell and Jones, 1992; Jones and Randall, 1978). Korsmeyer et al. (1997) found in the highly active yellowfin tuna (\( Thunnus albacares \)) that \( \dot{Q} \) increased by 13.6% during exercise at 24°C exclusively through tachycardia and, in fact, \( V_s \) decreased. Similarly, during forced swimming at 0°C the Antarctic borch (\( Pagothenia borchgrevinki \)) increased \( \dot{Q} \) by 75% by doubling \( f_t \) (Axelsson et al., 1992). Furthermore, Cooke et al. (2003) investigated the relative contribution of \( V_s \) and \( f_t \) to maximum cardiac output at 3°C in three North American species with various degrees of winter quiescence. Largemouth bass (\( Micropterus salmoides \)), a winter active species, increased \( \dot{Q} \) by means of a 124% increase in \( f_t \) with a 24% reduction in \( V_s \). Similarly, the intermediately active black crappie (\( Pomoxis nigromaculatus \)) increased \( \dot{Q} \) by a 156% increase in \( f_t \) with a 56% reduction in \( V_s \). By contrast, in the winter active white bass (\( Morone chrysops \)) maximum \( \dot{Q} \) was attained by a 45% increase in \( f_t \) and a 55% increase in \( V_s \). Within a species, temperature may modulate the relative contributions of \( f_t \) and \( V_s \) during exercise. For example, for maximum \( \dot{Q} \) at 5°C and 10°C in largescale suckers (\( Catostomus macrocheilus \)), increased \( V_s \) contributed 58% and 62%, respectively (Kolok et al., 1993), whereas at 16°C \( f_t \) contributed 70% of maximum \( \dot{Q} \) during exercise. Altogether these results indicate the importance of frequency regulation of \( \dot{Q} \) in various fish species under various conditions but, of course, contrasts with several studies (mainly on salmonids) where increased \( V_s \) was the major means of increasing \( \dot{Q} \) during exercise (Dunmall and Schreeer, 2003; Farrell, 1991; Farrell and Jones, 1992; Jones and Randall, 1978; Kiceniuk and Jones, 1977).

Under the present experimental conditions, exercising sea bass used only tachycardia to increase \( \dot{Q} \), but to what degree \( V_s \) might be modulated in sea bass at higher swimming speeds and different temperatures awaits further study. By performing the study at 22°C, which is in the upper range of the temperature preferendum for sea bass (Claireaux and Lagardere, 1999), it is possible that at lower water temperatures \( V_s \) could increase during swimming. Another concern is that fish only swim to 2 \( BL \) \( s^{-1} \) and this resulted in a relatively small increase in \( \dot{Q} \) (38%). Therefore, it is possible that at higher swimming speeds further increases in \( \dot{Q} \) could occur through increased \( V_s \).

In conclusion, this is the first study to measure variables related to venous return and cardiac filling pressure during exercise and to provide evidence for an active involvement of the venous vasculature during exercise in any species of fish. An \( \alpha \)-adrenoeceptor mediated control system was partially responsible for a decrease in venous capacitance, as reflected as an increase in MCFP in the swimming sea bass. This control would serve to: (1) maintain or increase the pressure gradient for venous return, thus matching venous return and cardiac output; (2) increase central venous blood volume, and consequently cardiac preload; and (3) possibly redistribute venous blood to other parts of the circulation, such as the gills and muscle capillaries. These results highlight the fact that an increased cardiac preload does not necessarily result in an increased \( V_s \), but can instead compensate for the reduced filling time when \( f_t \) is increased, thereby offsetting potential decreases in \( V_s \). In fact, under the present conditions, the exercise-induced increase in \( \dot{Q} \) in sea bass was exclusively mediated by tachycardia.

Symbols

\[
\begin{align*}
\dot{f}_t & \quad \text{heart rate} \\
M_b & \quad \text{body mass} \\
\text{MCFP} & \quad \text{mean circulatory filling pressure} \\
P_{CV} & \quad \text{central venous pressure} \\
P_{DA} & \quad \text{dorsal aortic blood pressure} \\
\Delta P_V & \quad \text{pressure gradient for venous return} \\
\dot{Q} & \quad \text{relative cardiac output} \\
R_{SYS} & \quad \text{total systemic resistance} \\
R_V & \quad \text{resistance to venous return} \\
U_{\text{crit}} & \quad \text{critical swimming speed} \\
V_s & \quad \text{stroke volume}
\end{align*}
\]

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References


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