Transactions of the American Fisheries Society 130:783–795, 2001

Cardiac Response to Variable Forced Exercise at Different Temperatures: An Angling Simulation for Smallmouth Bass

JASON F. SCHREER,* STEVEN J. COOKE,[1] AND R. S. MCKINLEY

Department of Biology, University of Waterloo, Waterloo, Ontario N2L 3G1, Canada

Abstract.—Longer angling durations (capture by hook and line) and higher water temperatures are generally thought to be more detrimental to angled fish. Here, this concept was investigated in smallmouth bass *Micropterus dolomieu* by monitoring cardiac output (CO) and its components, heart rate (HR) and stroke volume (SV), before, during, and after a simulated angling event in a Blazka-type respirometer. Fish (total $n = 31$) were acclimated to 12, 16, or 20°C and exposed to conditions that elicited repeated burst swimming either briefly (20 s) or to exhaustion (120–180 s). Resting CO and HR increased significantly with increasing temperature ($Q_{10} \sim 2$), indicating temperature conformity, whereas SV was not affected by temperature. Recovery times (time after angling until cardiac parameters returned to resting levels) ranged from 0 to 85 min (mean ~ 40 min) for briefly angled fish and from 20 to 210 min (mean ~ 105 min) for exhaustively angled fish. These recovery times increased significantly with angling duration but were not affected by water temperature. Almost all of the increase in CO during recovery was attributable to increased HR. At 20°C, the increase in HR was sufficient to result in an increase in CO despite a decrease in SV during the initial portion of recovery. During recovery, both CO and HR increased to approximately 1.8 times and as much as 2.65 times the resting values, whereas SV increased to approximately 1.2 times the resting value. At 20°C, SV typically decreased to 0.8 times the resting value for the initial part of recovery. Recovery intensities (maximum increases in cardiac values) were generally not affected by angling duration. Fish acclimated to 16°C generally had larger recovery intensities than fish at 12°C and 20°C, indicating that for smallmouth bass, 16°C may be an optimal temperature relative to the other two temperatures. From a management perspective, the results suggest that (1) angling duration should be minimized, (2) angling at high as well as low temperatures may be detrimental, and (3) even when fish are angled very quickly, they still undergo a period of increased CO that can last as long as 1 h.

The physiological disturbances from short periods of anaerobic activity, such as those incurred during angling (capture by hook and line), can require several hours (Wood et al. 1983; Tufts et al. 1991; Booth et al. 1995) or even days (Wydoski et al. 1976; Haux et al. 1985) to return to resting values. During this time, the rate of oxygen consumption may remain above resting rates for several hours (Scarabello et al. 1991). In many cases, severe exercise may result in mortality afterwards, whether immediate or delayed (Black 1958; Bennett 1978; Wood et al. 1983).

Because of the importance of recreational fishing and the resultant removal of a substantial number of fish, numerous management strategies have been implemented to maintain populations of game fish (e.g., length-limit regulations, specific angling seasons, and catch-and-release fishing [Barnhart 1989]). The introduction of mandatory and voluntary catch-and-release angling has led to speculation about the recovery and eventual survival of fish after release. Although both acute and delayed mortality are obvious indicators of the success or failure of this management technique (Muoneke and Childress 1994), more subtle factors such as the underlying mechanisms associated with physiological disturbance are much more difficult to monitor, and relevant information is limited. Such disturbances may lead to decreases in individual survival (e.g., predator avoidance, foraging success) and in reproductive success (e.g., mate acquisition, fecundity, parental care) and are of paramount importance in evaluating management strategies.

Several methods are currently used to monitor physiological disturbances resulting from angling, but all have limitations (Cooke et al. in press). Hematological analyses (e.g., Wydoski et al. 1976; Gustaveson et al. 1991) have been used widely and very successfully, but the volume of blood that can be taken from a single fish is limited. Sampling

* Corresponding author: jfschree@sciborg.uwaterloo.ca

[1] Current address: Department of Natural Resources and Environmental Sciences, University of Illinois and Center for Aquatic Ecology, Illinois Natural History Survey, Champaign, Illinois, 61820 USA.

Received September 13, 1999; accepted January 15, 2001

too much blood from an individual can itself result in a host of physiological responses. Also, for both hematological and tissue analyses (e.g., Booth et al. 1995; Kieffer et al. 1995), samples typically are taken terminally from different individuals, which means the resolution of these studies is usually in the order of several hours.

Current telemetric devices allow in situ monitoring of physiological disturbances, but all have severe limitations when applied to angling. Electromyogram activity (Kaseloo et al. 1992) and tailbeat frequency (Johnstone et al. 1992) have been used to monitor metabolic activity, but often fish will not or cannot swim after rigorous angling. Therefore, these techniques would provide little information under these conditions (but see Cooke et al. 2000). Heart rate alone has been used as an indicator of metabolic rate during angling recovery (Anderson et al. 1998), but that may not be suitable for certain species. For example, most fish increase their cardiac output (CO) primarily by increasing stroke volume (SV) rather than heart rate (HR; Farrell and Jones 1992; Thorarensen et al. 1996). Therefore, a more accurate measure of oxygen consumption requires measurement of CO, which is a function of both HR and SV (i.e., CO = HR × SV) (Thorarensen et al. 1996; Webber et al. 1998).

For fish, angling requires maximum power output from locomotory muscles that involve white muscle fibers and the anaerobic metabolism of glycogen. After this event, fish face a period of recovery marked by the repayment of an oxygen debt associated with the clearance of by-products produced during angling (Wood et al. 1983; Gaesser and Brooks 1984). Consequently, by monitoring the repayment of this oxygen debt through measuring such features as CO, the response and recovery of fish to angling can be assessed.

In this study our aim was to monitor the angling response and recovery of smallmouth bass *Micropterus dolomieu* continuously in real –time, which we accomplished by measuring CO and its components HR and SV before, during, and after a simulated angling event. Also, because longer angling durations and warmer water temperatures have been suggested to increase mortality and physiological disturbance (Gustaveson et al. 1991; Muoneke and Childress 1994; Kieffer et al. 1995), we tested the hypotheses that (1) cardiac recovery time increases with increasing angling duration and water temperature and (2) the magnitude or intensity of the cardiac response increases with increasing angling duration and water temperature. Lastly, we considered the results of this study with reference to management strategies for catch-and-release angling.

Methods

Study animals.—Smallmouth bass (n = 31), ranging in size (total length) from 260 to 343 mm, were angled from Lake Erie and transported to holding facilities at the University of Waterloo. Water temperatures at time of capture were 14–18°C. Fish were held in flow-through tanks supplied with oxygenated well water and acclimated to 12, 16, and 20°C for at least 4 weeks before experimentation. The study temperatures were chosen to reflect a range of temperatures at which smallmouth bass are commonly angled, both throughout their distributional range and during several different seasons (Coble 1975; Armour 1993). Fish were held under 12 h light/dark conditions and fed emerald shiners *Notropis atherinoides* to satiation every 3 d. Before surgery and experimentation, the fish were fasted for 72 h. Sample sizes for the various groups were 5, 6, and 7 for exhaustively angled fish and 3, 3, and 7 for briefly angled fish acclimated to 12, 16, and 20°C, respectively.

Surgery and instrumentation.—Fish were anesthetized before surgery with 60 ppm clove oil (emulsified with ethanol, 9:1 ethanol:clove oil) for about 5 min, until the subjects had lost equilibrium and were nonresponsive (Anderson et al. 1997). The fish were placed on one side on a wetted sponge operating sling. The anesthetized state was maintained during surgery by irrigating the gills with water containing a maintenance concentration of anesthetic (30 ppm clove oil). An oval-shaped plastic cover was placed behind the first gill arch, and the gills and operculum were held in an open position to expose the area underneath. The ventral aorta was usually visible just caudal to the first gill arch at the point where the gill arch begins to run anterior-caudally. The connective and adipose tissues surrounding the vessel were carefully removed with blunt forceps. A flexible silicone cuff–type Doppler flow probe (subminiature 20-MHz piezoelectric transducer; Iowa Doppler Products, Iowa City, IA), sized to match the diameter of the vessel, was placed around the aorta. The internal diameter of the cuffs ranged from 1.2 to 2.0 mm. Using a flowmeter (545C–4 Directional Pulsed Doppler Flowmeter; Bioengineering, the University of Iowa, Iowa City) and a digital strip-chart recorder (LabVIEW, Version 4.0.1; National Instruments Corporation, Austin, Texas), we checked

the cuff for adequate signal strength. A single suture surrounding the cuff secured the probe to the vessel and ensured that the diameter of the lumen would remain nearly constant. The lead wire from the probe was then sutured to the skin just anterior to the origin of the pectoral fin. The length of lead wire from the cuff to this first suture was left slack so that muscular movements would be unlikely to alter the position of the cuff. Several additional sutures were used to secure the wire to the body wall. The entire procedure took 15–30 min.

Cardiac parameter measurement and calculation.—The Doppler transducer emits a pulsed sonic signal, and because of the Doppler shift when the signal is reflected from a moving object in the blood (e.g., a red blood cell), a shift in the signal frequency is observed. This shift in frequency represents a velocity and is measured as a change in voltage (0.5 V = 1 kHz; Operating and Service Manual, 545C–4 Directional Pulsed Doppler Flowmeter). Peaks in voltage/velocity represent a heart beat; counting peaks per unit time yields HR. The mean voltage per unit time is an index of flow, or CO (flow can also be calculated directly in milliliters per unit time; see Postmortem calibration). The quotient of CO divided by HR yields SV.

Before experimentation, we focused the sonic signal in the center of the vessel by maximizing the signal strength (adjusting the range). To ensure accurate and consistent measurements, we checked signal strength four times throughout the experiment: (1) after surgery and placement in the respirometer, (2) after recovery and before experimentation, (3) after euthanasia (fish intact), (4) just before calibration measurements. Typically no adjustment was needed once the range was set.

Angling simulations.—After surgery, individual fish were immediately placed into a 120-L Blazka-type respirometer (see Booth et al. 1997 for details) and monitored until they had regained equilibrium and were holding station within the tube. Fish were then allowed to recover from surgery and acclimate to the respirometer for at least 12 h at water velocities of less than 0.2 m s^{-1}. Slow, near-zero velocities were chosen to simulate the typical lentic conditions where smallmouth bass from this population often reside (Coble 1975). A darkened area at the front of the swim tube provided cover and ensured that the fish were not disturbed by general laboratory activity. During the recovery period, the respirometer was continuously supplied with aerated well water at the appropriate acclimation temperature.

Cardiac parameters were recorded continuously for at least 1 h before the angling simulation (resting), during the angling simulation, and for at least 5 h during recovery. All experiments were conducted at the same time of day (simulated angling event at ~1000 hours). After resting recordings, fish were exposed to either a brief (for 20 s) or an exhaustive (for 120–180 s, and until the fish would no longer swim, or had lost equilibrium, or both) simulated angling event. This event consisted of a series of velocity bursts up to 1.5 m s^{-1} that attempted to mimic some of the conditions fish experience during angling. Velocity bursts were adjusted so that fish could not hold station and would be pushed backwards so that their tail touched the back grate of the tube. This caused the fish to perform a series of vigorous swimming bursts. The intensity and duration of the velocity bursts were adjusted up or down depending on the ability of the fish so that each subject was ''angled'' under similar conditions and ended up in a similar state of fatigue (within each duration, brief or exhaustive). After this period, velocity was returned to near zero during the recovery phase. This angling simulation probably elicited minimal physiological disturbances, because only one of the three main stressors associated with catch-and-release angling—variable forced exercise (the other two being the hooking injury [Muoneke and Childress 1994] and air exposure [Ferguson and Tufts 1992; Cooke et al. 2001])—was simulated. However, related work in which electromyogram signals (Cooke et al. 2000) and catecholamines and cortisol concentrations (Butler et al. 1986; Lowe and Wells 1996) were monitored during angling and simulated angling conditions suggests that even this limited angling simulation elicits a physiological response similar to that in ''real'' hook and line angling and more rigorous angling simulations.

Postmortem calibration.—After experimentation, the fish were killed with an overdose of anesthetic (180 ppm clove oil), and a postmortem calibration was made to convert Doppler shift (measured in V) to flow (mL/min). The head, including the pericardial cavity, was separated from the rest of the fish. The pericardial cavity was opened with a caudal/ventral incision exposing the heart. The sinus venosus, atrium, and ventricle were removed and the bulbus arteriosus was catheterized with PE-100 tubing for perfusion of the ventral aorta. With use of a constant infusion pump (Harvard Apparatus, South Natick, MA), anticoagulated blood (cows' blood containing 2 g of sodium oxalate, 0.4 g of sodium chloride, and 40 mL

of distilled H_2O per liter) was perfused through the aorta to calibrate the probes over a range of flow rates encompassing those recorded during the trials. Reference flow rates were analyzed with linear least-squares regression (mean $R^2 = 0.980$, $n = 31$) in SYSTAT, Version 7.

Data analysis.—To determine recovery times, traces for CO, HR, and SV, adjusted to resting (100%), were plotted for each fish and analyzed visually. A fish was considered to be recovered when values returned to resting or became stable (in some cases cardiac function stabilized above the preangling values [~10%] because of a slight diurnal effect in activity [i.e., more active during the day: Reynolds and Casterlin 1976; Schreer and Cooke, unpublished data]). The magnitude or intensity of recovery was determined as the highest value, for that particular cardiac parameter, attained during the recovery period. A minimum value was also determined for SV because values often decreased to below resting values during the recovery period.

Differences across the various groups were compared by analysis of variance (ANOVA; SYSTAT, version 7). Resting cardiac values across temperatures were compared with a one-way ANOVA. Temperature (12, 16, or 20°C) and angling duration (brief or exhaustive) effects on cardiac recovery and intensity were determined with a two-factorial ANOVA. When ANOVAs were significant, effects were compared, a posteriori, with a Tukey pairwise comparison. Significance was set at $\alpha = 0.05$.

Results

As shown in Figure 1, resting values (mean ± SE) for CO (mL min^{-1} kg^{-1})—12°C = 19.7 ± 2.1, 16°C = 29.8 ± 3.7, 20°C = 33.7 ± 3.5—and HR (beat min^{-1})—12°C = 26.7 ± 3.6, 16°C = 34.5 ± 2.7, 20°C = 52.8 ± 2.5—increased with temperature (CO ANOVA, $F = 4.202$, $P = 0.024$; CO post hoc, 20°C > 12°C, $P = 0.019$; HR ANOVA, $F = 22.779$, $P < 0.001$; HR post hoc, 20°C > 12°C and 16°C, $P < 0.001$ for each). The increase in rate with a 10°C increase in temperature (i.e., Q_{10}) for $CO_{12–16°C}$, $CO_{16–20°C}$, and $CO_{12–20°C}$ was 2.84, 1.36, and 1.96, respectively. Q_{10} values for $HR_{12–16°C}$, $HR_{16–20°C}$, and $HR_{12–20°C}$ were 1.90, 2.89, and 2.34, respectively. SV (mL kg^{-1})—12°C = 0.78 ± 0.06, 16°C = 0.87 ± 0.09, 20°C = 0.64 ± 0.06—was not affected by temperature, although 20°C fish had a marginally lower SV than 16°C fish (ANOVA, $F = 2.959$, $P = 0.067$; post hoc, $P = 0.059$).

The angling event elicited an initial bradycardia lasting from 5 to 20 s (Figure 2). Brief periods of bradycardia were also observed throughout the exhaustive angling events (Figure 2B) and corresponded to periods of burst swimming. Recovery from the angling event was marked by an increase in CO that was almost entirely attributable to an increase in HR (Figures 3 and 4). Similarly, for the 20°C fish, the increase in HR was sufficient to result in an increase in CO despite a decrease in SV for part of the recovery period (Figure 4). Recovery times ranged from 0 min for SV to 85 min for CO and HR of briefly angled fish and from 20 min for SV to 210 min for CO and HR of ex-

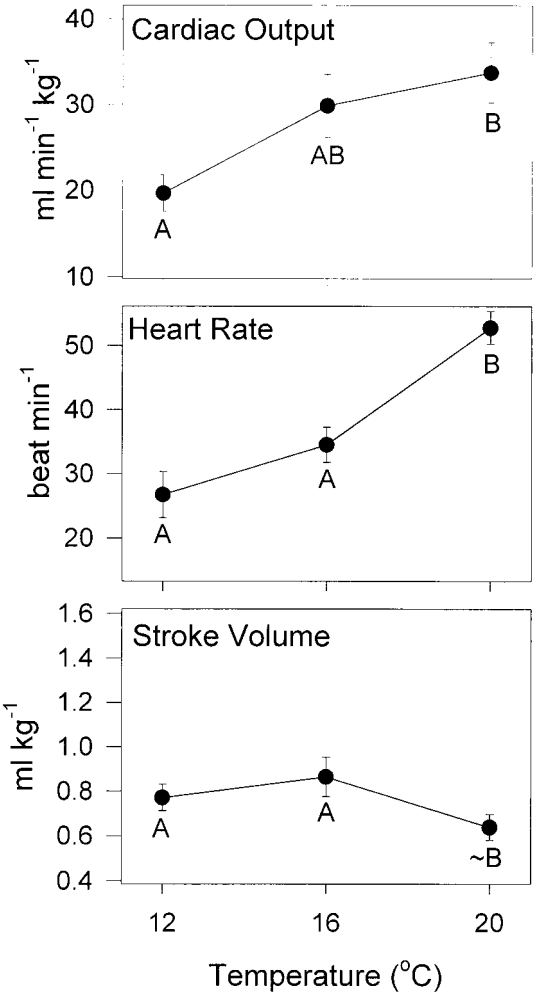

FIGURE 1.—Influence of temperature on resting values for cardiac parameters of smallmouth bass. Scales for *y*-axes are relative to (i.e., 0.5× to 2.1×) the 12°C values. Fish were acclimated for more than 4 weeks at 12°C ($n = 8$), 16°C ($n = 9$), and 20°C ($n = 14$). Points within a panel with different letters were significantly (~ = marginally) different.

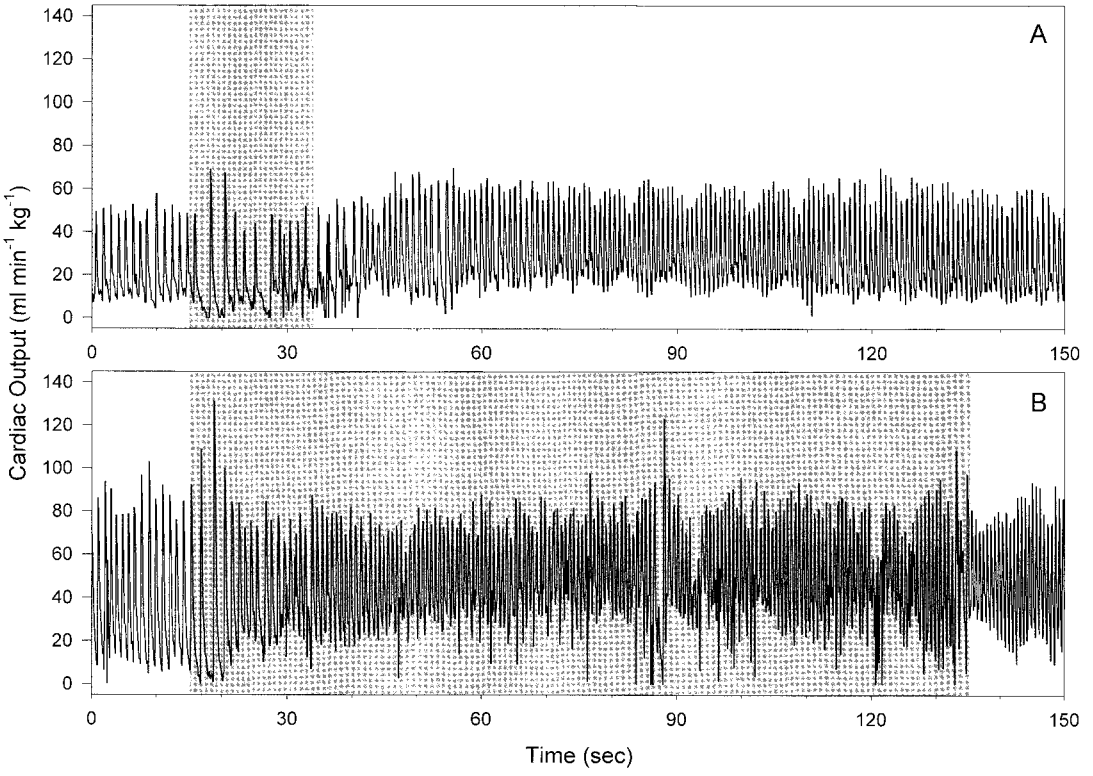

FIGURE 2.—Examples of cardiac output during the simulated angling event. Readings were taken every 0.1 s. Fish were acclimated for more than 4 weeks at 20°C and angled either briefly (A, 20 s) or to exhaustion (B, 120 s). The shaded areas denote the simulated angling event.

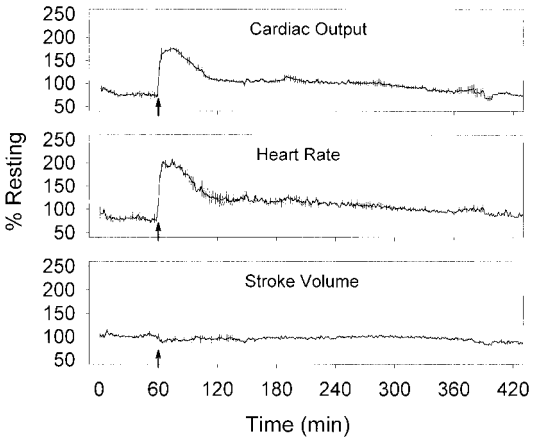

FIGURE 3.—Cardiac response to exhaustive simulated angling in smallmouth bass (n = 6) acclimated to 16°C. Fish were acclimated for more than 4 weeks and angled for 120–180 s. All values are mean (1 min) ± SE percent resting (averaged for the 6 fish), with resting equal to 100%. The arrows denote the beginning of the angling event, which always occurs at time = 60 min.

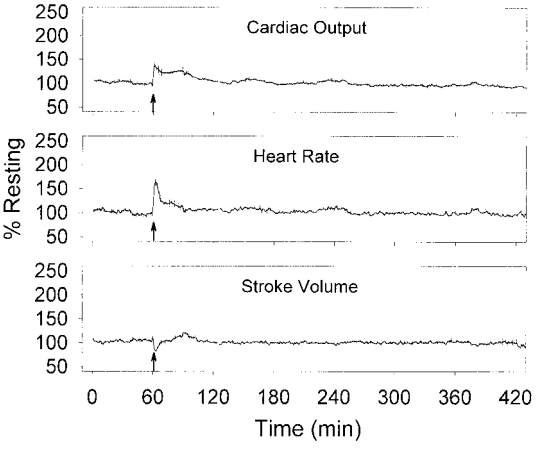

FIGURE 4.—Cardiac response to brief simulated angling in smallmouth bass (n = 7) acclimated to 12°C. Fish were acclimated for more than 4 weeks and angled for 20 s. All values are mean (1 min) ± SE percent resting (averaged for the 7 fish), with resting equal to 100%. The arrows denote the beginning of the angling event, which always occurs at time = 60 min.

haustively angled fish. The magnitude of recovery ranged from 100% of resting (i.e., no response, equal to resting) for SV to 265% of resting for CO of both briefly and exhaustively angled fish. Means for both recovery time and intensity are presented in Figure 5.

Comparisons across the various groupings (Figure 5 and see Table 1 for *F*-ratios and *P*-values) indicated that fish angled to exhaustion had significantly longer recovery times for all cardiac parameters than briefly angled fish. However, the magnitude of the response was generally not affected by angling duration. The difference in HR intensity for briefly versus exhaustively angled fish was significant, although for the 16°C fish the values were identical. The difference between CO intensity for fish briefly and exhaustively angled at 12°C was also significant, but not for any other temperature. Differences in recovery times across temperatures were small, although a trend for exhaustively angled fish could be seen for CO, HR, and SV, with 16°C fish having shorter recovery times than the 12°C and 20°C fish. The temperature effect on recovery time was weakly significant for SV, although none of the pairwise comparisons were significant.

Several differences in magnitude were significant across temperatures, but the trend varied with the cardiac parameter and the angling duration. Only CO recovery intensity had a significant interaction between temperature and angling duration. For exhaustively angled fish, the magnitude of the CO response decreased significantly as the temperature increased. However, for briefly angled fish, 16°C fish had a significantly more intense CO response than did the 20°C fish and a marginally significantly more intense CO response than the 12°C fish. A similar significant trend was seen for HR, for which 16°C fish of both angling durations had a significantly more intense response than the 12°C and 20°C fish. The magnitude of recovery for SV showed no significant trends.

When decreases in SV were compared, a more apparent trend was observed. As temperature increased, SV values decreased significantly for both briefly and exhaustively angled fish. Moreover, the decrease in SV was marginally greater for exhaustively angled fish.

Discussion

Resting Values

Resting values for CO, HR, and SV from different studies are highly variable even within a single species under similar conditions (Farrell and Jones 1992). The values presented here are the first direct measurements for smallmouth bass or any centrarchid and fit well within the ranges observed for other temperate water teleosts. As well, the CO values are appropriately 2–3 times that of the sluggish hagfish *Eptatretus cirrhatus,* about 0.25 as much as that of the extremely active tunas (e.g., the skipjack tuna *Katsuwonus pelamis*), and similar to that of the moderately active rainbow trout *Oncorhynchus mykiss* at comparable temperatures (Farrell 1991a). Cardiac output and SV values estimated for the closely related largemouth bass *M. salmoides* by a whole-body thermodilution technique (Reynolds and Casterlin 1978) were higher than the values presented here ($44_{\text{largemouth}}$ vs. $20_{\text{smallmouth}}$ mL min^{-1} kg^{-1} and $1.46_{\text{largemouth}}$ vs. $0.77_{\text{smallmouth}}$ mL kg^{-1} for CO and SV, respectively, at 12°C), although Farrell and Jones (1992) suggested that indirect methods can substantially overestimate CO.

Fish acclimated to warmer temperatures had greater CO values. Within limits, most processes accelerate with increased temperatures (Schmidt-Nielsen 1990). This typically results in an increase in metabolic rate, which generally produces a proportional increase in CO (Mirkovic and Rombough 1998). An increase in CO with increasing temperature has been demonstrated for several fish species and is due in part to an increase in HR as a result of an increase in membrane permeability of pacemaker fibers (Randall 1968, 1970; Farrell and Jones 1992) and changes in neural and humoral regulation (Farrell 1984). Illustrated weakly for smallmouth bass here, SV may increase with decreasing temperature as a result of ventricular hypertrophy (Graham and Farrell 1990; Sephton and Driedzic 1991) or increased ventricular filling time because of decreased HR (Graham and Farrell 1990) or both. The increase in CO and HR noted here ($Q_{10} = \sim 2$) is similar to previous findings for several species (winter flounder *Pleuronectes americanus* [Cech et al. 1976], dogfish *Scyliorhinus acanthias* [Butler and Taylor 1975], sea raven *Hemitripterus americanus* [Graham and Farrell 1985], and rainbow trout [Barron et al. 1987]) and is indicative of temperature conformity, whereas $Q_{10} = 1$ would indicate complete temperature compensation (Farrell and Jones 1992).

Relative Contribution of HR and SV to Increased CO

Angled fish generally perform a series of locomotory bursts utilizing anaerobic metabolism

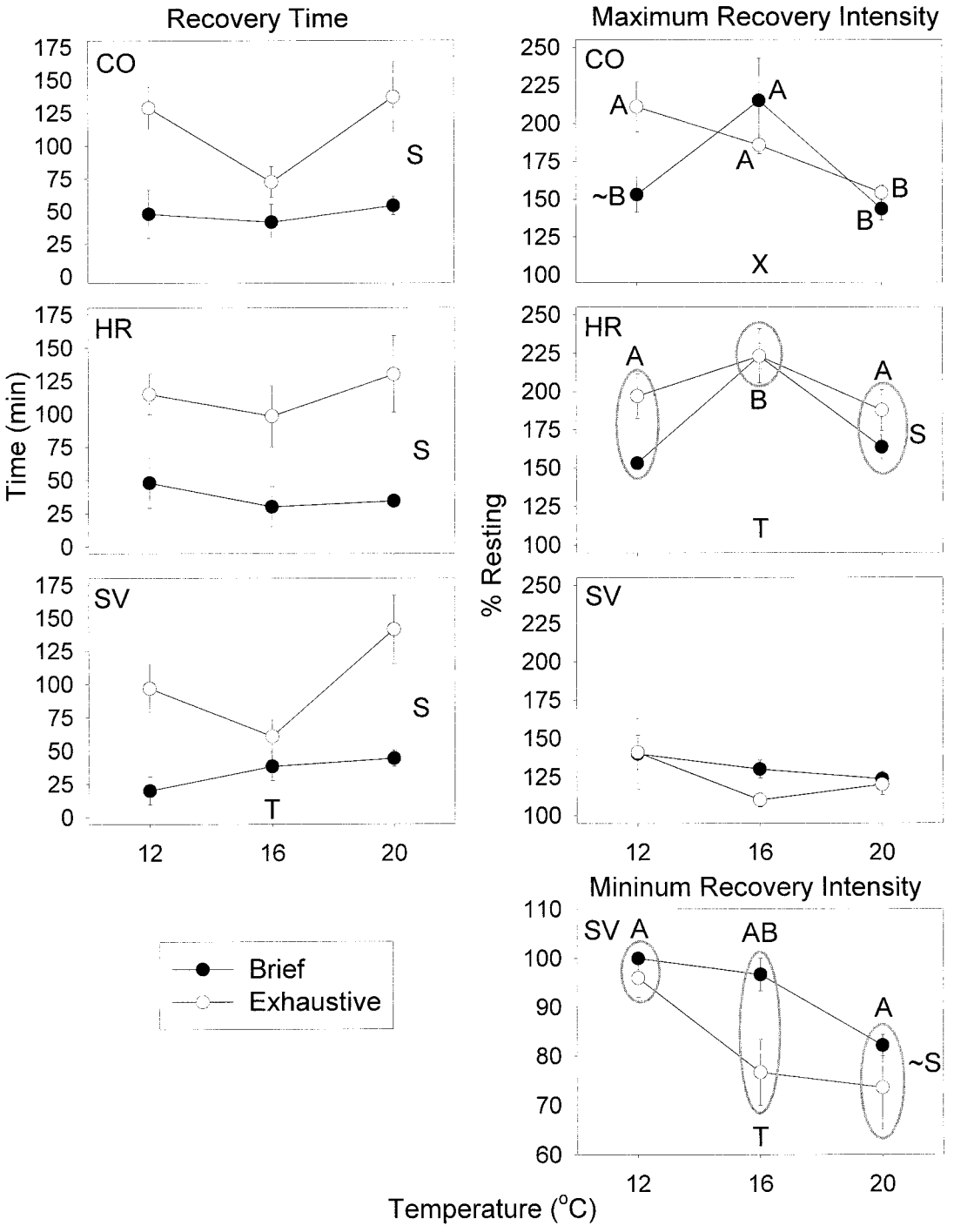

FIGURE 5.—The influence of temperature and simulated angling duration on recovery time and intensity (maximum increase) of cardiac parameters in smallmouth bass. The graph on the bottom right shows the decrease in stroke volume during recovery. Fish were acclimated for more than 4 weeks at 12 (n = 8), 16 (n = 9), and 20°C (n = 14). Brief = 20 s of simulated angling. Exhaustive = 120−180 s of simulated angling and until the fish could no longer swim or had lost equilibrium or both. See Table 1 for F-ratios and P-values. S, simulated angling effect; T, temperature effect; X, interaction with temperature and angling. Points within a panel with different letters were significantly (~ = marginally) different. Circled values were pooled for posthoc Tukey pairwise comparison because they did not interact.

TABLE 1.—Significance levels (*F*-ratios [*P*-values]) for the influences of temperature and angling duration on recovery time and intensity (two-factorial analysis of variance with post hoc Tukey pairwise comparison). Brief angling = 20 s, exhaustive angling 120–180 s and until fish could no longer swim or had lost equilibrium or both. The following abbreviations are used: CO = cardiac output, HR = heart rate, and SV = stroke volume. Maximum and minimum refer to recovery intensity (see text). Significance was set at $\alpha = 0.05$. Marginally significant differences are also shown.

Comparison	Temperature	Angling	Temperature × angling
CO recovery time		16.051 (<0.001)	
CO maximum	12.056 (<0.001)		6.002 (0.007)
Post hoc analysis			Exhaustive–12°C > Brief–12°C ($P = 0.038$) Brief–16°C > Brief–12°C ($P = 0.052$), Brief–20°C ($P = 0.004$) Exhaustive–12°C > Exhaustive–20°C ($P = 0.007$)
HR recovery time			
HR maximum	8.898 (0.001)		
Post hoc analysis	16°C > 12°C (0.005), 20°C (0.002)	16.614 (<0.001) 4.674 (0.040)	
SV recovery time	3.376 (0.050)	17.255 (<0.001)	
SV maximum		4.105 (0.054)	
SV minimum	5.104 (0.014)		
Post hoc analysis	12°C > 20°C ($P = 0.011$)		

(Tufts et al. 1991; Kieffer et al. 1995). The bradycardia induced by this activity is thought to be necessary to prevent hypertension as violent muscle contractions shunt peripheral blood flow (Farrell and Jones 1992). Recovery from burst exercise, specifically dealing with oxygen debt and lactate clearance, results in an increased metabolic rate and consequently an increase in CO and in one or both of its components (HR and SV) (Farrell and Jones 1992). The increase in CO during angling recovery for smallmouth bass was almost entirely attributable to an increase in HR. For the 20°C fish the increase in HR was sufficient to produce an increase in CO despite a decrease in SV. This is unlike most fish species, for which increases in CO are primarily the result of increases in SV rather than in HR (Farrell 1991a; Farrell and Jones 1992; Thorarensen et al. 1996), and is similar to mammals, birds, reptiles, and amphibians, in which CO is primarily modulated by HR (Farrell 1991a). In fact, Farrell (1991a) suggests that vertebrates exhibit an evolutionary trend from volume-modulated to frequency-modulated cardiac output. However, the extremely active tunas are an exception to this, as apparently are the smallmouth bass. Resembling the tuna in being able to increase HR by twofold over resting values (Farrell 1991a), the smallmouth bass would have no need to increase SV during maximum metabolic activity.

Another factor to consider when assessing the relative contributions of HR and SV to CO is the type of exercise. In a few cases, investigators have suggested that after anaerobic, burst exercise, increases in CO are the result of increased HR (Lucas et al. 1991; Farrell and Jones 1992). Because the exercise during angling is anaerobic, an increase in CO during recovery that is predominantly attributable to an increase in HR may not be such an anomaly. However, HR in Atlantic salmon *Salmo salar* after angling increased only 15–30% above resting (Anderson et al. 1998), compared with the greater than 100% increase in HR for smallmouth bass after simulated angling.

A final possible explanation for the relative contributions of HR and SV in increased CO is the recovery time after the surgical procedure used to affix the monitoring device (Webber et al. 1998). Webber et al. (1998) suggest that HR in cod *Gadus morhua* remains above resting values for as long as 8–10 d after surgery. Therefore, if the relative contributions of HR and SV to increases in CO during exercise are examined within this recovery period, above-normal postsurgery resting HR values may prevent the typical increases in HR. However, even though the smallmouth bass in the present study were allowed to recover for less than 24 h (but more than 12 h) after surgery, increases in CO were almost entirely the result of increases in HR. That these fish had abnormally high resting HR values, therefore, is highly unlikely. Further, we have held smallmouth bass for extended periods (2 weeks) and did not observe any further decrease in cardiac parameters (Schreer and Cooke, unpublished results), contrary to the suggestion of Webber et al. (1998).

Recovery Time and Intensity

All recovery times were within 3.5 h for exhaustively angled fish and were typically less than 2 h. This is considerably shorter than estimated recovery times for HR in Atlantic salmon, in which values remained increased for 16 h (Anderson et al. 1998). Angling resulted in increases of various hematological parameters in largemouth bass for 8–24 h (Gustaveson et al. 1991), in muskellunge *Esox masquinongy* for 12–18 h (Beggs et al. 1980), in rainbow trout for up to 72 h (Wydoski et al. 1976), and in perch *Perca fluviatilis* for 48–96 h (Haux et al. 1985). However, other studies have shown similar rapid recovery times for white muscle pH, lactate, ATP, and phosphocreatine in Atlantic salmon, with values returning to resting levels within 2–4 h, although muscle glycogen remained increased for 12 h (Booth et al. 1995). Moreover, blood pH in exhaustively exercised Atlantic salmon returned to resting values within 4 h, although blood lactate remained increased for 8 h (Tufts et al. 1991).

Cardiac parameters for all briefly angled fish returned to resting values within 1.5 h and typically within 1 h. Although these recovery times are relatively short, it is interesting to note that recovery lasts about 1 h for fish angled under what are generally considered nonrigorous angling conditions (Kieffer et al. 1995). In fact, some studies (Gustaveson et al. 1991) have used angling durations (15 s) similar to this as controls. However, from our results, even when fish are angled very quickly, they still have a period of metabolic disruption that may contribute to increased mortality, nest abandonment, and nest predation after release.

As expected, fish angled to exhaustion had much longer recovery times than those angled only briefly. A similar result has been seen previously in numerous studies of angling stress in black bass *Micropterus* spp. as well as other fish species. Physiological disturbances (increases in muscle lactate, metabolic protons, and partial pressure of carbon dioxide and decreases in pH and energy reserves) were more severe in nesting male smallmouth bass played to exhaustion (2 min) than those played briefly (<20 s) (Kieffer et al. 1995). Moreover, time to return to their nests was fourfold longer for black bass angled to exhaustion than for briefly angled fish (Kieffer et al. 1995; Philipp et al. 1997). In largemouth bass, physiological disturbances (increases in plasma glucose, chloride, and osmolarity and blood lactate) were generally more severe with increasing time on hook (Gustaveson et al. 1991).

Surprisingly, however, cardiac recovery time did not increase with increasing temperature, and the magnitude of the response was generally not affected by angling duration. Previous work on the effect of temperature on angling disturbance has been contradictory. Angling disturbance is generally thought to increase with water temperature. High temperatures have been blamed for greater angling mortality in smallmouth bass (Cooke and Hogle 2000), largemouth bass (Welborn and Barkley 1974), rainbow trout (Dotson 1982), brook trout (Nuhfer and Alexander 1992), cutthroat trout *Oncorhynchus clarki* (Hunsaker et al. 1970; Marnell and Hunsaker 1970), and seatrout *Salmo trutta trutta* (Matlock and Dailey 1981). A similar trend was suggested for physiological disturbances (increases in plasma glucose, chloride, and osmolarity and blood lactate) with no mortality in largemouth bass (Gustaveson et al. 1991), but several of the trends were weak. Despite the wealth of information suggesting that angling disturbance increases with temperature, several studies have shown no relationship. In Atlantic salmon, time to recovery for HR was similar for fish at 8°C and 16°C (Anderson et al. 1998). In lake trout *Salvelinus namaycush* (Loftus et al. 1988) and channel catfish *Ictalurus punctatus* (Ott and Storey 1993), hooking mortality was not related to temperature. Similarly, for largemouth bass, U_{crit} values were found to be thermally insensitive for certain size classes and temperature ranges (Beamish 1970; Kolok 1991). Maintaining the same level of cardiovascular or swim performance over a range of temperatures presumably allows fish to tolerate thermal variation in water temperature during summer (Kolok et al. 1993). This may also explain why the recovery times we observed did not vary with temperature.

That the magnitude of recovery was not affected by angling duration indicates cardiac scope (i.e., maximum CO and HR; Farrell 1991b) may be a limiting factor in the replacement of an oxygen debt associated with the clearance of by-products produced during angling. A threefold increase in CO during exercise is considered extreme (Farrell 1991a) and less than a twofold increase is more the norm (Farrell and Jones 1992). Because smallmouth bass increase CO, almost solely because of a similar increase in HR of 1.5–2 times after even a brief angling period, a further increase in CO may not be available after a more exhaustive activity period. Therefore, the oxygen debt associ-

ated with a more severe stressor cannot be repaid at a faster rate (i.e., greater CO intensity) but only by increasing CO to whatever the threshold may be for a longer time.

The magnitude of recovery across temperature was greatest for CO of briefly angled 16°C fish and for HR of briefly and exhaustively angled 16°C fish. Combined with the nonsignificant trends towards shorter recovery times for 16°C fish across most parameters and angling conditions, this suggests that 16°C may represent an optimal temperature for cardiac recovery (as suggested in Farrell et al. 1996) in comparison with 12°C and 20°C. At 12°C, smallmouth bass are generally less active, and investigators have suggested that as temperature decreases from 20°C to 5°C, activity levels are reduced until the fish eventually enter a state of torpor (Coble 1975; Sephton and Driedzic 1991). This is probably an important component of an overwintering strategy. Because the fish are less active at this temperature, the intensity of their cardiac response to angling may be less, which results in longer recovery times. A temperature of 16°C is common during spawning for smallmouth bass (Armour 1993) and therefore is a temperature at which these fish are likely to have maximal activities (Hinch and Collins 1991). Consequently, smallmouth bass may be able to deal with a stressor most efficiently at this temperature. At 20°C smallmouth bass may be pushing their thermal maximal limit (for this Lake Erie population; many southern populations can reside at much higher temperatures). Increases in resting values and limits on maximal rates may again limit cardiac response intensity at warmer temperatures, resulting in longer recovery durations.

A very interesting anomaly from this trend was a decrease in the magnitude of CO with increasing temperature for exhaustively angled fish presumably as a result of the metabolic scope of this species. All species have an upper limit of metabolism, regardless of exogenous and endogenous factors. Because resting CO and HR increased with temperature, more of this scope is being used by fish acclimated to warmer temperatures. Therefore, these higher-temperature fish would have a lower ceiling (above resting) for any increase and this may explain why CO recovery intensity decreases with increasing temperature. The mechanism behind this relationship can be seen in the mechanical limits of the components of CO, HR and SV. Aside from tuna, the suggested upper limit of cardiac frequency in lower vertebrates is 120 beats/min (Farrell 1991a). Because resting HR increases with temperature, and was approximately 75% higher for 20°C fish than for 12°C fish, the interval between resting and maximum frequency becomes smaller. However, HR recovery intensity did not decrease with increasing temperature. Therefore, CO intensity did not decrease with increasing temperature as the result of a decrease in HR intensity. Similarly, SV intensity did not significantly decrease with increasing temperature, although there was a trend in that direction, and therefore also does not explain the observed decrease in CO. However, when considering that SV actually decreased below resting values for part of the recovery period in some fish, the explanation becomes clearer. The decreases in SV during recovery showed a significant trend towards a more intense decrease with increasing temperature. Cardiac ejection is much higher in fish than mammals (Farrell 1991a). In rainbow trout, this value can be nearly 100% of ventricular volume. Consequently, SV is almost solely determined by end-diastolic volume and therefore is limited by cardiac filling. As HR increases, cardiac filling time decreases, causing SV to decrease, and CO to plateau.

Management Implications

Regulations mandating the release of numerous fish species, particularly black bass, have increased since the inception of catch-and-release angling in 1954 (Barnhart 1989) and more so in recent years (Quinn 1996). Even voluntary catch-and-release angling (see review by Quinn 1996) has increased substantially. If management agencies are to encourage or mandate catch-and-release angling, the assumption that a large proportion of these fish will survive with no measurable impact on individual fitness or the population must truly be the case (Cooke et al. in press). Clearly evident in our results are several implications for managers, competitive angling event organizers, and anglers with respect to minimizing the physiological disturbance on smallmouth bass. The results suggest that (1) angling duration should be kept as short as possible, (2) angling at both low and high temperatures may be detrimental, and (3) even when fish are angled very quickly, the period of cardiac recovery can still last as long as 1 h. During this period of cardiac disturbance and recovery is when fish have less metabolic scope available for such fitness-related activities as predator avoidance and parental care.

In conclusion, after exposure to a simulated angling event, smallmouth bass increase cardiac out-

put twofold over resting values. Almost all of the increase in CO is the result of an increase in HR; SV either increased very little, did not change, or actually decreased during recovery. Recovery times increased with longer angling durations but were not affected by temperature. The intensity of recovery did not increase with angling duration, which indicates both that the cardiac response is maximized even with brief disturbances and that more rigorous disturbances cannot be dealt with by a more intense response but simply by extending recovery over a longer period. Intensity trends across temperature show that fish at moderate temperatures (16°C) had more intense and perhaps shorter recoveries and suggest that this temperature may be optimal compared with 12°C and 20°C. From a management perspective, this research provides managers with some of the first data on the real-time recovery and metabolic consequences of catch-and-release angling. Further work with this methodology, as well as increasing the sample sizes examined (a limitation of this study), will provide more definitive information on how fish respond to various natural and anthropogenic perturbations.

Acknowledgments

This work was supported by the University of Waterloo, the Department of Fisheries and Oceans, and the Natural Sciences and Research Council of Canada. Thanks are extended to Amy Dick for assistance in the laboratory and data handling, Ken Chandler for assistance in the field, and R. S. McKinley for financial support and access to equipment and laboratory space. The manuscript benefited from reviews by Anthony P. Farrell and anonymous reviewers. J.F.S. thanks X.O.Q.

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