

Electrofishing-induced cardiac disturbance and injury in rainbow trout

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Cardiac output (CO), heart rate (HR) and stroke volume (SV) were monitored in rainbow trout *Oncorhynchus mykiss* electroshocked (pulsed DC) with various voltage, frequency, pulse width and shock duration settings. Additionally, behavioural recovery times and internal haemorrhaging were examined. During electroshock, heart function became erratic and typically ceased for much of the event (cardiac arrest). Following electroshock, CO increased almost entirely due to an increase in SV. Cardiac function generally returned to resting levels within 2–3 h. Behaviourally, recovery was much more rapid, typically taking only a few minutes for fish to regain equilibrium and to begin swimming normally, and only rarely was >1 h. Internal injury ranged from 0 to 7 cm² of haemorrhaging along the spine and surrounding musculature, although only 4% of the fish had corresponding damage to the vertebrae. Comparisons across the various electrofisher settings indicate that response and injury are highly variable, but generally increased with more intense settings. Higher frequencies and voltages appear to most negatively affect behavioural recovery and injury while longer shock durations increased the length of cardiac arrest and the duration of cardiac recovery. Because of the variability in results and small sample sizes, however, the strongest conclusion that can be drawn from this work is that electrofishing, regardless of the settings, has a considerable negative impact on rainbow trout that is often not apparent externally. Additionally, this study has shown how electrofishing directly effects fish physiologically through impairment of cardiac function. Studies examining the physiological and behavioural response of fishes and subsequent recovery will be necessary for the development of electrofishing guidelines which minimize the disturbance to fishes.

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Key words: cardiac output; electrofishing; heart rate; injury; stroke volume.

INTRODUCTION

Electrofishing is often used by fishery biologists to sample fish populations in small freshwater streams and rivers. The popularity of electrofishing is due in large part to the absence of alternative collection techniques that are efficient for small lotic systems (Cooke *et al.*, 1998). Harmful effects due to electrofishing, however, were noted for salmonids since the late 1940s (Hauck, 1949; Pratt,

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1955). Subsequent studies have reported effects ranging from negligible mortality and injury rates (McCrimmon & Bidgood, 1965; Hudy, 1985) to serious spinal damage and haemorrhaging (Sharber & Carothers, 1988*a,b*; Sharber *et al.*, 1994; Thompson *et al.*, 1997*a*). Other potential negative effects endured by fishes due to electroshock include: reduced swimming stamina (Horak & Klein, 1967; Mitton & McDonald, 1994*a*), muscle, nerve and tissue damage (McMichael, 1993; Hollender & Carline, 1994) and physiological and behavioural disturbances (Mitton & McDonald, 1994*a*; Schreck *et al.*, 1976; Mesa & Schreck, 1989; Cho *et al.*, 2002).

Harm due to electroshock, both incidence and severity, are related to the equipment and type of electrical output [*e.g.* AC (alternating current), continuous DC (direct current), pulsed DC, voltage (V), frequency (Hz) and pulse width (ms)] from the electrofisher (Whaley *et al.*, 1978; Sharber & Carothers, 1988*b*; McMichael, 1993; Sharber *et al.*, 1994; Dalbey *et al.*, 1996). For example, continuous or low frequency pulsed DC has been shown to reduce spinal injury rates compared to higher frequencies (Whaley *et al.*, 1978; McMichael, 1993; Sharber *et al.*, 1994; Dalbey *et al.*, 1996).

There is a general consensus that electrofishing does inflict some level of harm to fishes (Snyder, 1992, 1995). This could bias mark-recapture studies by overlooking potential cumulative effects of exposure to electroshock and therefore affect subsequent capture (Sharber & Carothers, 1988*a,b*). Since this capture technique is used extensively for fishery research and management, it is very important to understand the extent of harm inflicted on individual fish. Many researchers report the incurring injury, usually spinal, experienced by fishes due to electroshock (Sharber & Carothers, 1988*a*; Dalbey *et al.*, 1996; Kocovsky *et al.*, 1997; Ainslie *et al.*, 1998), although there is little information on less severe injury or disturbance. While haemorrhaging is often mentioned anecdotally, it is rarely quantified (Sharber *et al.*, 1994; Dalbey *et al.*, 1996; Kocovsky *et al.*, 1997; Ainslie *et al.*, 1998). Further, although fishes may appear physically 'normal' following electrofishing, the physiological disturbance may last for several hours (Schreck *et al.*, 1976; Mitton & McDonald, 1994*a,b*). Because various electrofisher settings can affect fishes in numerous ways, it is necessary to assess these effects at various biological levels using ecologically relevant indicators.

The objective of this study was to determine how various pulsed DC electrofisher settings affect and injure rainbow trout *Oncorhynchus mykiss* (Walbaum) and how long it takes these fish to recover from the short-term physiological disturbances. Direct impacts were assessed by monitoring cardiac function during and following electroshock. Recovery was determined by monitoring the length of time it took for fish to regain equilibrium following electroshock and how long cardiac function remained altered. Injury was assessed by quantifying the area of haemorrhage in the musculature around the spinal column. The working hypothesis for this study was that fish would be more negatively affected by higher electrofisher settings and longer shocking durations. Therefore, responses would be more intense, recoveries would be longer and injuries would be more extreme with larger frequencies, voltages, pulse widths and longer electroshock durations. The aim was to provide new information on the sublethal effects of electrofishing, including insight into what is physically

occurring to the rainbow trout during the shock, and to provide managers with information on how to optimize the various electrofishing settings to minimize injury and physiological disturbance.

MATERIALS AND METHODS

To study the effects of various electrofishing settings on the behaviour and physiology of fish, a three component study was undertaken. Physiological responses were monitored by measuring blood flow and cardiac performance, behaviour was assessed by visually observing fish during and after electrofishing, and injury was determined by standard *post mortem* examinations for spinal injury and external and internal haemorrhaging.

STUDY ANIMALS

In the winter of 1999 hatchery rainbow trout (Rainbow Springs Fish Hatchery, Thamesford, Ontario, Canada) were transported to holding facilities at the University of Waterloo, Ontario. Fish were held for at least 2 weeks in flow-through 1000 l tanks that were oxygenated and supplied with 11°C well water. Fish were held under a 12L:12D photoperiod and fed to satiation daily with 5 mm floating trout chow (Martin Feedmills, Elmira, ON, Canada). Prior to surgery and experimentation fish were fasted for 24 h. A total of 75 rainbow trout (mean \pm s.e. total length, $L_T = 355 \pm 4$ mm and mass, $M = 576 \pm 13$ g) were used for this study.

SURGERY AND INSTRUMENTATION

A complete description of surgical procedures and the Doppler measurement theory is given by Schreer *et al.* (2001). Briefly, fish were anaesthetized prior to surgery with 60 ppm clove oil (emulsified with ethanol, 9:1 ethanol:clove oil) and this state was maintained during surgery by irrigating the gills with water containing a maintenance concentration of anaesthetic (30 ppm clove oil). A flexible silicone cuff-type Doppler flow probe (sub-miniature 20 MHz piezoelectric transducer: Iowa Doppler Products, Iowa City, IA, U.S.A.), sized to match the diameter of the vessel, was placed around the ventral aorta. Internal diameter of the cuffs ranged from 1.1 to 1.7 mm. A single suture surrounding the cuff secured the probe to the vessel and the lead wire from the probe was then sutured to the skin just anterior to the origin of the pectoral fin and to several other locations leading up to the dorsal fin. The entire procedure took 15–30 min. A flowmeter (545C-4 Directional Pulsed Doppler Flowmeter: Bioengineering, The University of Iowa, Iowa City, IA, U.S.A.) and a digital strip-chart recorder (LabVIEW, Version 4.0.1, National Instruments Corporation, Austin, TX, U.S.A.) were used to monitor blood flow.

The cardiac output (CO) was calculated by averaging the flow index per unit time and heart rate (HR) was determined by counting peaks. The quotient of CO divided by HR yielded stroke volume (SV). Where possible, actual flow rates were calculated directly through a *post mortem* calibration (Schreer *et al.*, 2001).

HOLDING

Following surgery, individual fish were immediately placed into black containers (68 cm long \times 40–45 cm wide \times 30 cm high) filled with 65 l of water. Conductivity of the water was $1300 \mu\text{S cm}^{-1}$ measured at 11°C. A 15 \times 40 cm hole was cut in the lids of the containers to allow for viewing while at the same time providing the fish with 15 cm of cover around the sides of the container. Twelve containers in total were used so that 12 fish could be tested simultaneously while being held independently. The containers had an inflow hole at one end and a drain at the other end creating a directional flow-through system. The inflow was such that the water in the container was being replaced every

c. 0.5 h. Water was maintained at 11°C and the water was oxygenated with an air-stone held behind a grate to prevent entanglement of hard-wired fish. Fish were permitted to recover for at least 12 h prior to experimentation.

ELECTROFISHING

Electrodes were constructed from aluminum plates (40–45 cm wide × 24 cm high × 0.667 mm thick) and were placed at both ends of the containers so that they were c. 68 cm apart. Electrical wire was connected to each aluminum plate with a stainless steel bolt. The wire from one metal plate could be fixed to the anode of the electrofisher using an alligator clip and the wire from the other metal plate could be fixed to the cathode. This set-up allowed the metal plates to be connected to the electrofisher to electroshock each container separately without disturbing the fish. The electrofishing was conducted with a pulsed DC Smith-Root Model 12-A pDC backpack electrofisher that had been recently tuned (Smith-Root Inc., Vancouver, WA, U.S.A.). Verification of electrofisher function was conducted with a digital oscilloscope.

ELECTROFISHING TREATMENTS

Following resting recordings, 75 fish were electroshocked using one of 11 different treatments. All electrofishing occurred at c. 0900 or 1600 hours. The frequency of the electroshock was either 10, 30, 90 or 80.8 Hz. The 80.8 Hz setting is a pulse pattern which starts at a rate of 80 Hz and decreases to a rate of 8 Hz. This setting produced an exponential change in the applied power as the frequency was changed. The voltage of the electroshock was either 100, 200 or 400 V. The pulse width of the electroshock was either 2, 4 or 8 ms. The duration of the shock was either 2, 8 or 32 s in length. When one of the variables was changed, the others were kept constant. These constants were 30 Hz for frequency, 100 V for voltage, 2 ms for pulse width and 8 s for the duration of the shock. This resulted in 10 different electrofishing treatments (Table I). The eleventh treatment was a control group. For the control, the electrofisher was set at 100 V,

TABLE I. Electroshock treatments. The varied setting is in bold. Of the 75 rainbow trout used for this study, 73 were used for the comparison of behaviour, injury and cardiac arrest (B) and 57 for comparison of cardiac function (CF)

Treatment	Settings				<i>n</i>	
	Frequency (Hz)	Voltage (V)	Pulse width (ms)	Duration (s)	B	CF
Standard or 30 Hz or 100 V or 2 ms or 8 s	30	100	2	8	7	5
10 Hz	10	100	2	8	7	5
90 Hz	90	100	2	8	6	4
80.8 Hz	80.8	100	2	8	6	5
200 V	30	200	2	8	6	5
400 V	30	400	2	8	7	4
4 ms	30	100	4	8	6	6
8 ms	30	100	8	8	7	5
2 s	30	100	2	2	7	7
32 s	30	100	2	32	8	5
Control	–	–	–	–	6	6

30 Hz and 2 ms and turned on for 8 s. But because the wires leading to the metal plates were not clipped to the anode or the cathode, an electric current was not produced in the tank. This was also verified with an oscilloscope.

DATA COLLECTION

Cardiac response

Cardiac variables were recorded continuously for at least 1 h prior to manipulation (resting), during the electrofishing, and for at least 5 h during recovery.

Behaviour

Fish were visually observed in the tanks immediately before, during and after electroshocking, and a behavioural recovery time was obtained for each fish. Fish were deemed behaviourally recovered when they had regained equilibrium and could maintain this position for a prolonged period (*i.e.* >10 min).

Injury

Approximately 24 h following electroshock fish were euthanized with an overdose of anaesthetic (180 ppm clove oil). External injuries were noted including bruising or branding and haemorrhages in the eyes and fin rays. The head and pericardial cavity were removed for cardiac calibration and the body of the fish was put on ice for 30 min in order to coagulate the blood prior to necropsy. Fish were then filleted from the left side in order to locate and identify any internal injuries (Reynolds, 1996). Obvious spinal injuries and haemorrhages were noted for approximate location and total area (cm²). A spinal injury was indicated by a misalignment or compression of one or more vertebrae as described by Thompson *et al.* (1997b) and Reynolds (1996). Photographs were taken of all fish for later reference.

DATA ANALYSIS

To determine recovery times, traces for CO, HR and SV, adjusted to resting (100%), were plotted for each fish and analysed visually. A fish was considered to be recovered when values returned to resting or became stable. The magnitude or intensity of recovery was determined as the highest value, for that particular cardiac variable, attained during the recovery period. Cardiac arrest during electroshock was assessed by visually analysing raw voltage outputs for blood flow and measuring periods of 0 flow.

Values for each setting were compared to a control of 100% (for cardiac intensity) or 0 (for all other comparisons) using the 95% CI. Differences across the various settings (frequency, voltage, pulse width and duration of the shock) were compared separately for: 1) cardiac intensity (per cent resting for CO, HR and SV), 2) cardiac recovery time (CO, HR and SV in min), 3) cardiac arrest (s), 4) behavioural recovery (min) and 5) injury (total internal haemorrhage area in cm²) with one-way ANOVA. Controls were excluded from these analyses because all effects were negligible and variances were very near zero. When ANOVAs were significant, effects were compared, *a posteriori*, with a Tukey pair-wise comparison. All tests were performed in SAS (Version 8.01) and significance was set at $\alpha = 0.05$.

RESULTS

Of the 75 rainbow trout used for the study, 73 fish were used for the comparison of behaviour, injury and cardiac arrest and 57 for comparisons of cardiac function. Two fish were excluded from experimentation because of death and a poor signal following surgery. An additional 16 fish were excluded

from comparisons of cardiac function because of poor signals following experimentation. This resulted in group sizes ranging from six to eight for behaviour, injury and cardiac arrest comparisons and four to seven for cardiac function comparisons (Table I). There were no significant differences in mass across any of the 11 groups.

Behavioural responses during electroshock were variable and generally independent of the type of shock. As expected, electrotaxis (forced swimming towards the anode plate) was observed followed by tetany (muscle rigidity and seizures) and narcosis (muscle relaxation or sleep-like state) (Reynolds, 1996). Fish characteristically convulsed or exhibited involuntary jerking with flared gills. Usually within 1–3 s after the shock fish lost equilibrium and became supine for the duration of the shocking period. Most fish ceased ventilating during electroshock and some coughed prior to regaining equilibrium. Following the shock approximately half of the fish regained equilibrium immediately while the other fish ranged between 1 and 83 min to regain equilibrium. A total of 17 fish took ≥ 10 min to regain equilibrium. One fish never regained equilibrium after 12 h. A number of fish (28%) hit the anode or cathode during the electroshock, but there were no significant differences in any of the variables measured between these fish and fish that had no contact with the conductors. Control fish showed none of the above effects.

Injury from electroshock was highly variable. External bruising or branding was observed on 10 fish. These bruised areas were dark in colour and located just anterior to, or directly under, the dorsal fin extending ventrally across the lateral line. Two fish had haemorrhages in the eye and one fish had haemorrhaging at the base of the pectoral fin. The highest incidence of external bruising or haemorrhaging was found in the 80–8 Hz group (50%), followed by the 30 Hz, 400 V, 4 ms groups (29%), the 90 Hz group (14%), and the 2 s group (12.5%). All fish in the other groups did not show any external injury. Internal injuries were also highly variable. Only three fish had obvious spinal injuries. One of these fish (2 s) died and the other two appeared normal at the termination of the experiment (400 V, 4 ms). The 400 V fish was swimming on its side immediately after electroshock, but within 15 min was upright and swimming normally. The 2 s fish appeared normal within minutes of shocking, but was found dead 4 h later. The 4 ms fish took almost 2 h to fully regain equilibrium and during this time had difficulty swimming, was struggling to regain equilibrium, and was gasping at the surface of the water. Large haemorrhages (1.5–3.5 cm²) were associated with all spinal injuries. Few fish had no internal haemorrhaging. Only 17 fish (excluding the controls) showed no indication of internal haemorrhaging. None of the treatments had 0% haemorrhaging, but ranged from 29 to 100%. The area of haemorrhaging ranged, on average, from 0.07 to 4.30 cm². No control fish showed any kind of external or internal injuries.

Resting cardiac values (mean \pm s.e.) for rainbow trout at 11°C were 17.72 ± 0.84 ml min⁻¹ kg⁻¹, 67.55 ± 1.08 bpm (beats min⁻¹) and 0.27 ± 0.02 ml kg⁻¹ for CO, HR and SV, respectively (calculated from 51 successful calibrations with a mean r^2 of 0.98). During electroshock a period of cardiac arrest occurred that was somewhat dependent on the duration of the shock (Fig. 1). Immediately following the shock, most fish had a period of arrhythmia lasting anywhere from a few seconds to several minutes (Fig. 1).

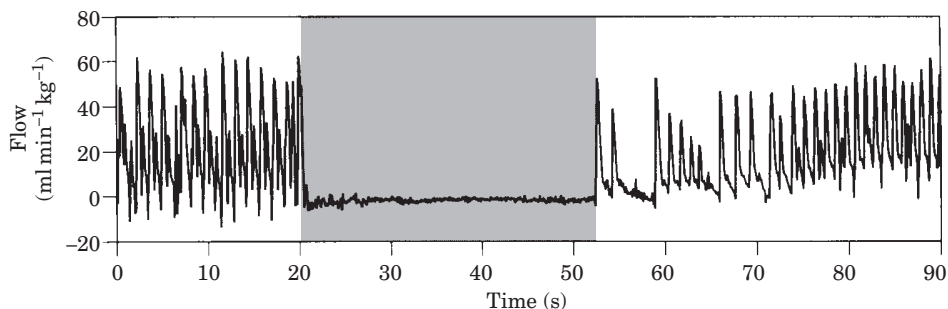


FIG. 1. Example of the cardiac response during electroshock. The fish was electroshocked for 32 s at 100 V, with a pulse width of 2 ms and a frequency of 30 Hz. The shaded area is the shocking period.

During recovery from the shock all cardiac variables were elevated and, typical for salmonids (Farrell & Jones, 1992), most of the increase in CO was due to an increase in SV while HR only increased slightly or in some cases actually decreased (Fig. 2). Cardiac output was elevated on average 165–189% of resting values and returned to resting levels over the next 100–186 min. This increase in CO was due to an even larger increase in SV ranging on average from 193 to 252% of resting values that returned to resting levels within 98–166 min. Heart rate increased only slightly following electroshock (on average 108–132% of resting values) and returned to resting levels within 40–114 min. Controls showed little change from resting values; CO and SV increased 113 and 116% of resting values and returned to resting values within 15 min, HR was unchanged from resting levels.

Comparisons across electrofisher settings indicated that there was a weak trend towards a more intense response for higher electrofisher settings (Figs 3–6). Most of these trends, however, were not significant. Most cardiac function variables were significantly different than control values with heart rate intensity as an exception that was only occasionally different from controls. Excluding controls, there were typically no significant differences across electrofisher settings for any of the cardiac function variables. A notable exception was for the duration treatment where fish exposed to 32 s of electroshock took longer to recover than those shocked for 2 s. The length of cardiac arrest for shocked fish was always significantly greater than controls. There were also several significant increases with higher settings for frequency, voltage and most noticeably duration. Behavioural recovery times increased with higher electrofisher settings, but these trends, due to large variation, were only significant for frequency and duration. The total area of haemorrhaging generally increased with higher electrofisher settings, but like behavioural recovery, these trends were typically not significant due to large variation. The strongest trends for per cent injury with increasing settings were for frequency and voltage.

DISCUSSION

This study has shown how electroshock can affect rainbow trout sublethally at many different levels. And while some of these findings were ambiguous and

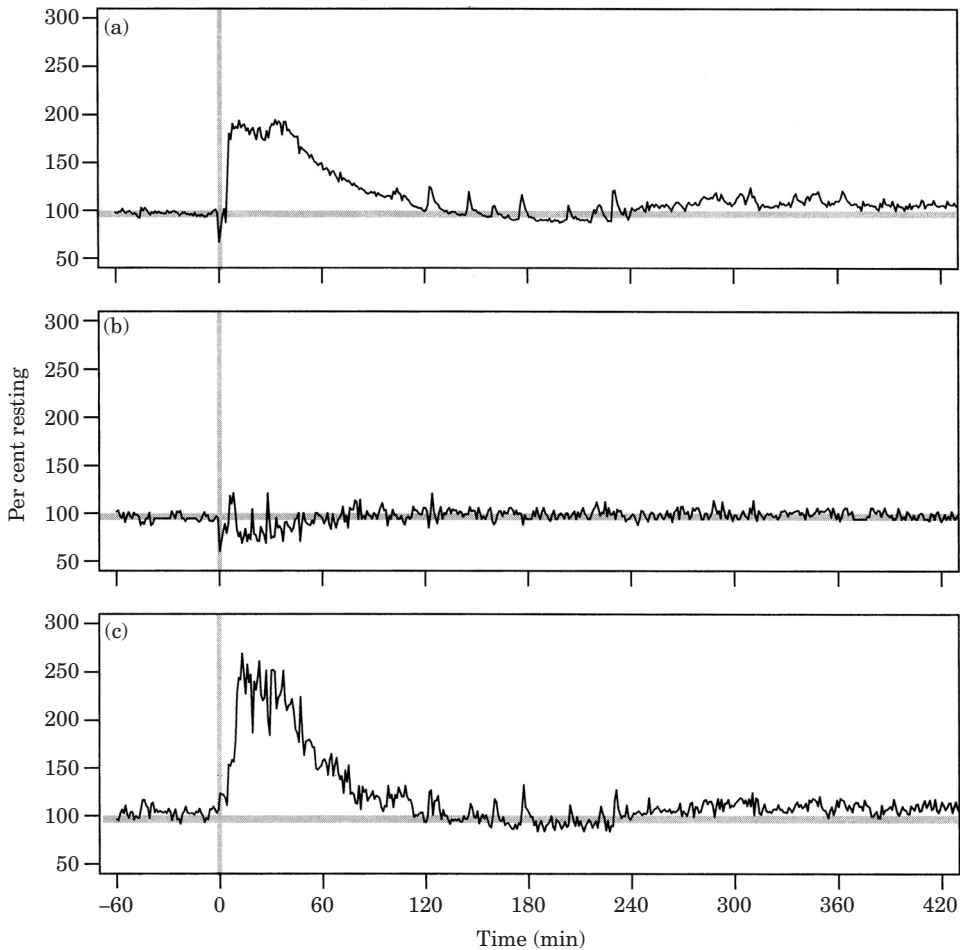


FIG. 2. Example of the cardiac response of a rainbow trout to electroshock: (a) cardiac output, (b) heart rate and (c) stroke volume. The fish was electroshocked for 8 s at 100 V, with a pulse width of 2 ms and a frequency of 80–8 Hz (frequency was reduced from 80 to 8 Hz over the 8 s shock). The shaded vertical lines are the shocking period and horizontal vertical lines are the 100% resting level.

trends were not significant, there were many patterns that can be useful in understanding the effects of various electrofisher settings on fish behaviour, physiology and injury.

When a fish was electroshocked it typically went into tetany or narcosis almost immediately and lost equilibrium. During the shock ventilation ceased and the heart went into arrest for the duration of the shock. The length of the cardiac arrest also increased moderately with increasing frequency and voltage, but was most strongly dependent on the duration of the shock. Different pulse widths had little affect on the duration of cardiac arrest. Following the shock approximately half of the fish regained equilibrium immediately while the rest took between 1 and 83 min to regain equilibrium. Time to regain equilibrium increased considerably with higher frequencies and voltages (although these trends were not significant due to large variation) and moderately for longer

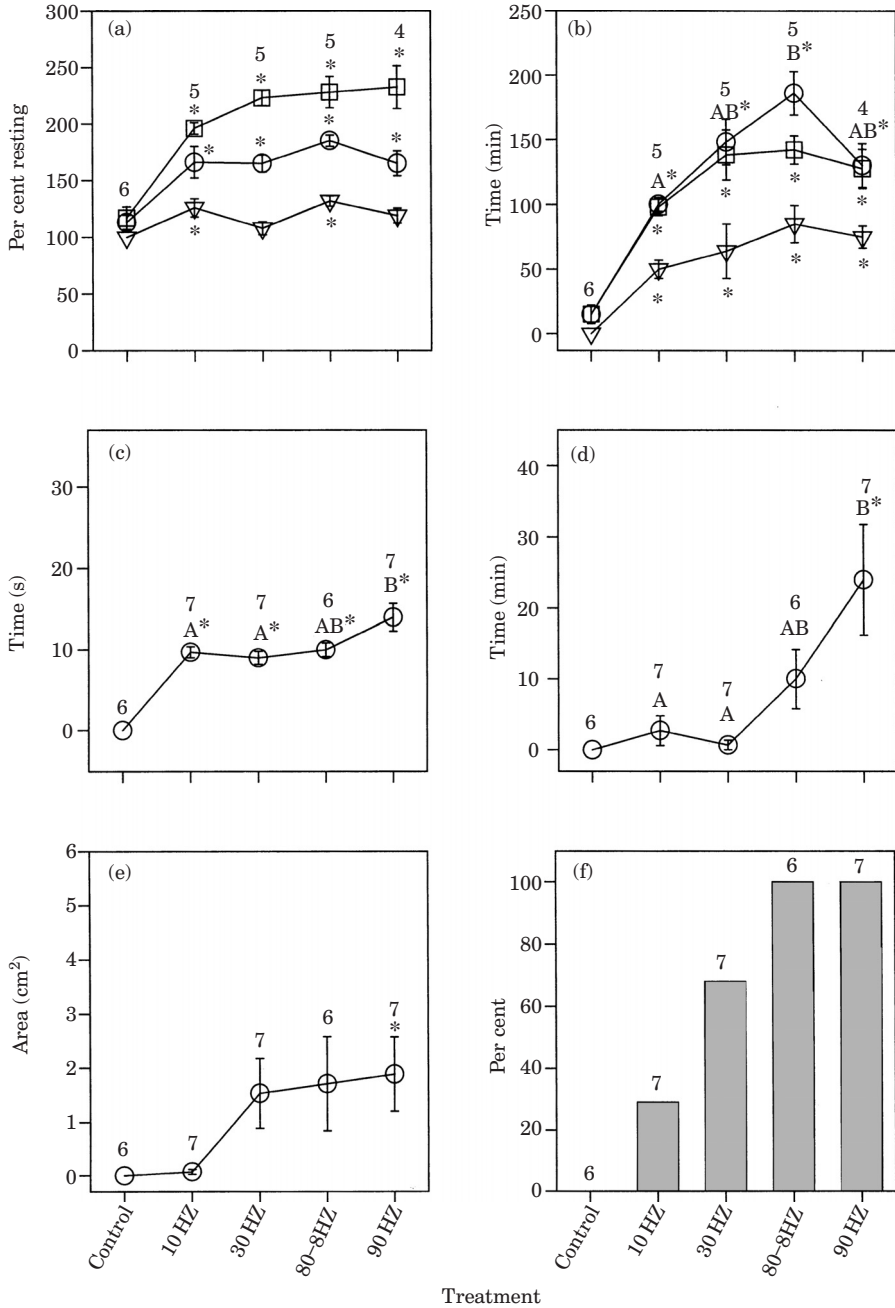


FIG. 3. Comparisons of (a) cardiac intensity (○, cardiac output; ▽, heart rate; □, stroke volume), (b) cardiac recovery, (c) cardiac arrest, (d) behavioural recovery, (e) injury area and (f) per cent injury of rainbow trout following electroshock at different frequencies. Fish were electroshocked for 8 s at 100 V, with a pulse width of 2 ms, and at 10, 30, 90 or 80-8 Hz (frequency was reduced from 80 to 8 Hz over the 8 s shock). Values (a)–(e) are presented as mean ± s.e. Numbers are sample sizes. *, the 95% confidence interval does not overlap with 0 (or 100 for cardiac intensity). Differences across treatments are denoted with different letters.

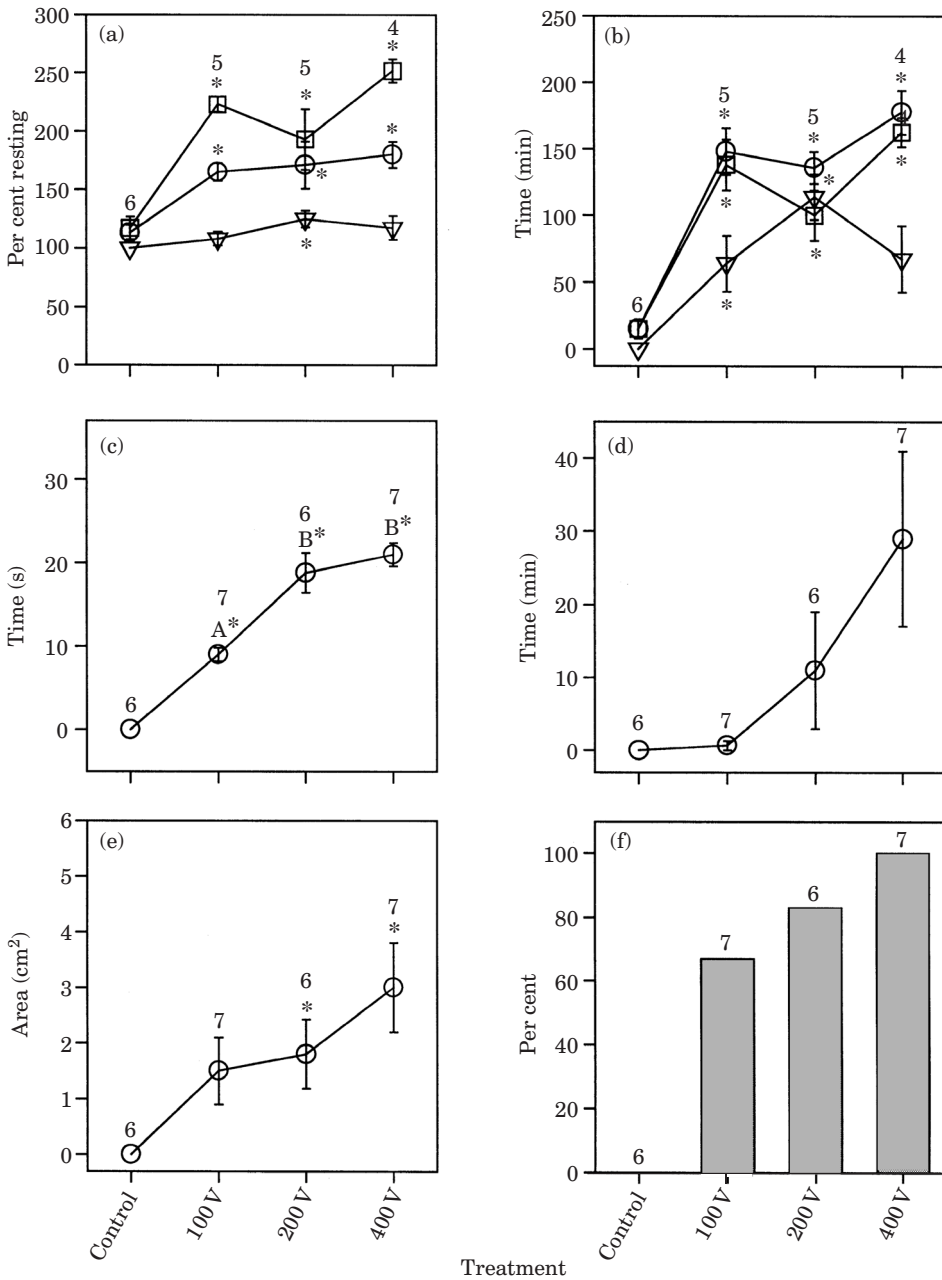


FIG. 4. Comparisons of (a) cardiac intensity (○, cardiac output; ▽, heart rate; □, stroke volume), (b) cardiac recovery, (c) cardiac arrest, (d) behavioural recovery, (e) injury area and (f) per cent injury of rainbow trout following electroshock at different voltages. Fish were electroshocked for 8 s with a pulse width of 2 ms, at a frequency of 30 Hz, and at 100, 200 and 400 V. Values (a)–(e) are presented as mean ± s.e. Numbers are sample sizes. *, the 95% CI does not overlap with 0 (or 100 for cardiac intensity). Differences across treatments are denoted with different letters.

shock durations. Cardiovascularly, these fish were in an altered state for 2–3 h following the shock. During this time CO was elevated to nearly 200% of resting values due to an increase in SV to >200% of resting levels. Heart rate only increased slightly during recovery. There were no consistent trends across the various settings for cardiac recovery except for an increase in CO recovery time with longer durations of electroshock. Approximately 24 h following the electroshock most fish had internal haemorrhaging around the spine. Interestingly, most of these fish showed no external branding or haemorrhaging. Trends in injury area and % injury across electrofisher settings suggest that injury may increase with higher frequencies and voltages (although none of these trends were significant).

Several basic findings can be drawn from these results. During electroshock when a fish becomes immobilized, its heart may stop for the duration of the shock or even longer. Following the shock many fish will regain some swimming function immediately, while others will be seriously impaired for >1 h. Cardiovascularly, even fish that regain equilibrium immediately will have elevated levels of blood flow for up to 3 h following the shock. Furthermore, most of these fish will have some internal haemorrhaging. Different electrofisher settings may impact the severity of the effects, but even at very low settings, impairments can be considerable.

Of the four electrofisher variables compared in this study, it appears that frequency and voltage most negatively affected behavioural recovery, injury and to a lesser degree cardiac arrest while the duration of the shock had the greatest impact on cardiac arrest. Cardiac recovery, both intensity and time, from electroshock were independent of the magnitude of settings, although longer shocking durations caused an increase in recovery time. Pulse width had the weakest impact on all the variables measured.

When a fish is exposed to an electrical field of adequate intensity it causes a series of neuromuscular responses (Vibert, 1967). AC is thought to elicit the most extreme amount of tetany and the most severe disturbance while continuous DC generally results in narcosis and a more mild disturbance (Spencer, 1967; Reynolds, 1983; Bayley *et al.*, 1989; Hollender & Carline, 1994; Mitton & McDonald, 1994b). AC is also not preferred because it does not elicit electro-taxis and therefore is an inefficient capture technique (Vibert, 1963). The pulsed DC used in this study, and most commonly used in recent electrofishing work because of its lower power requirements (Sharber *et al.*, 1994), is thought to be intermediate and results in immobilization by tetany (Mitton & McDonald, 1994b; Dalbey *et al.*, 1996; Ainslie *et al.*, 1998).

Initially when a fish is exposed to a DC electric field it swims towards the anode (electrotaxis) (Reynolds, 1996). As the distance between the fish and the anode decreases, the voltage gradient across its body increases exponentially (Novotny & Priegel, 1974). When the fish is very near the anode, it enters a state of narcosis and any increase in the voltage gradients beyond this point results in tetany (Vibert, 1967; Reynolds, 1996).

Originally it was thought that the severe muscular contractions during tetany were responsible for the high incidence of internal haemorrhaging (Cox & Lamarque, 1990). Additional work, however, has suggested that injuries can also occur at voltage gradients below the level required to produce narcosis (Sharber & Carothers, 1988a; Sharber *et al.*, 1994; Sharber & Black, 1999).

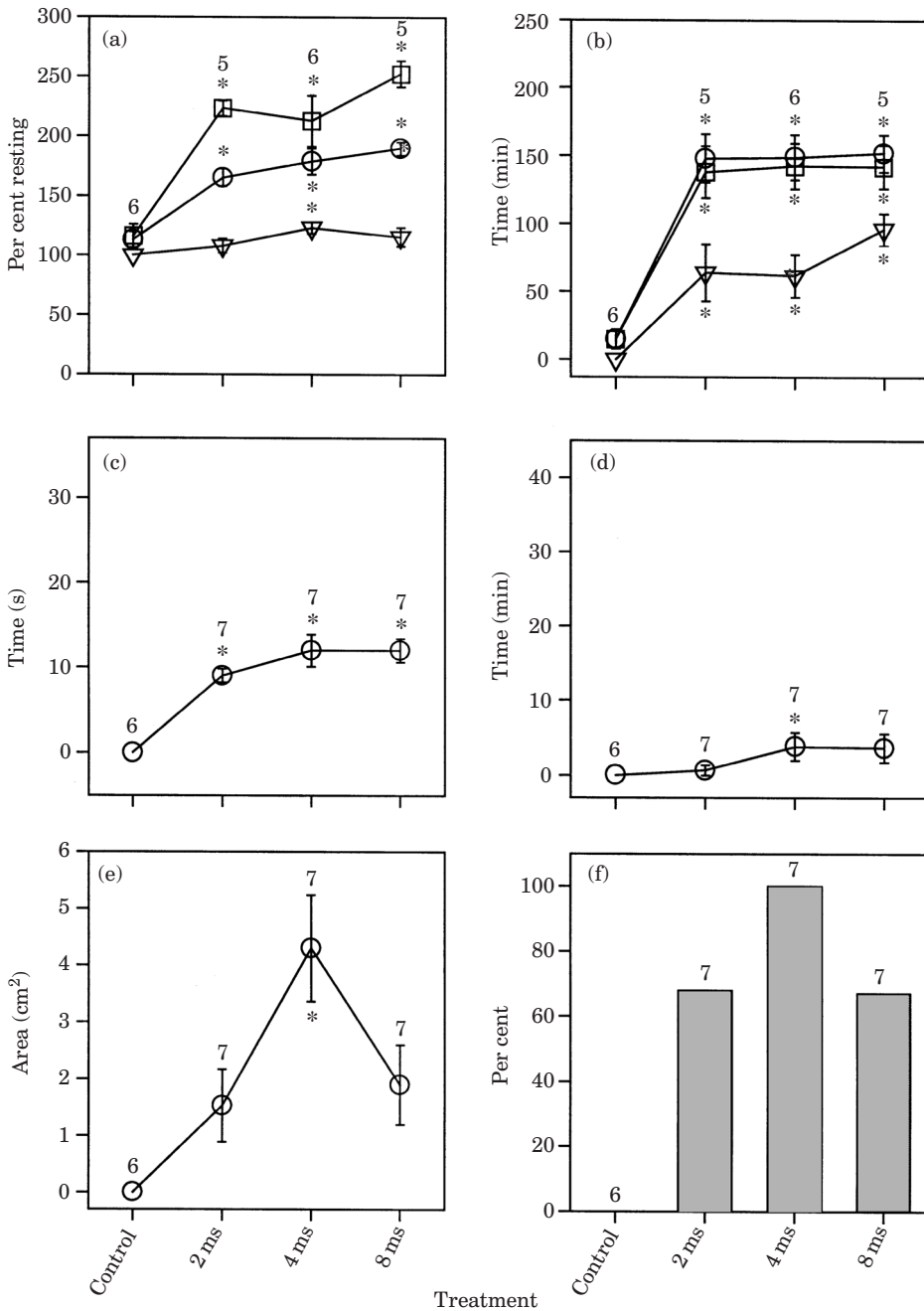


FIG. 5. Comparisons of (a) cardiac intensity (○, cardiac output; ▽, heart rate; □, stroke volume), (b) cardiac recovery, (c) cardiac arrest, (d) behavioural recovery, (e) injury area and (f) per cent injury of rainbow trout following electroshock at different pulse widths. Fish were electroshocked for 8 s at a frequency of 30 Hz, at a voltage of 100 V, and with pulse widths of 2, 4 and 8 ms. Values (a)–(e) are presented as mean ± s.e. Numbers are sample sizes. *, the 95% CI does not overlap with 0 (or 100 for cardiac intensity). There were no significant differences across treatments.

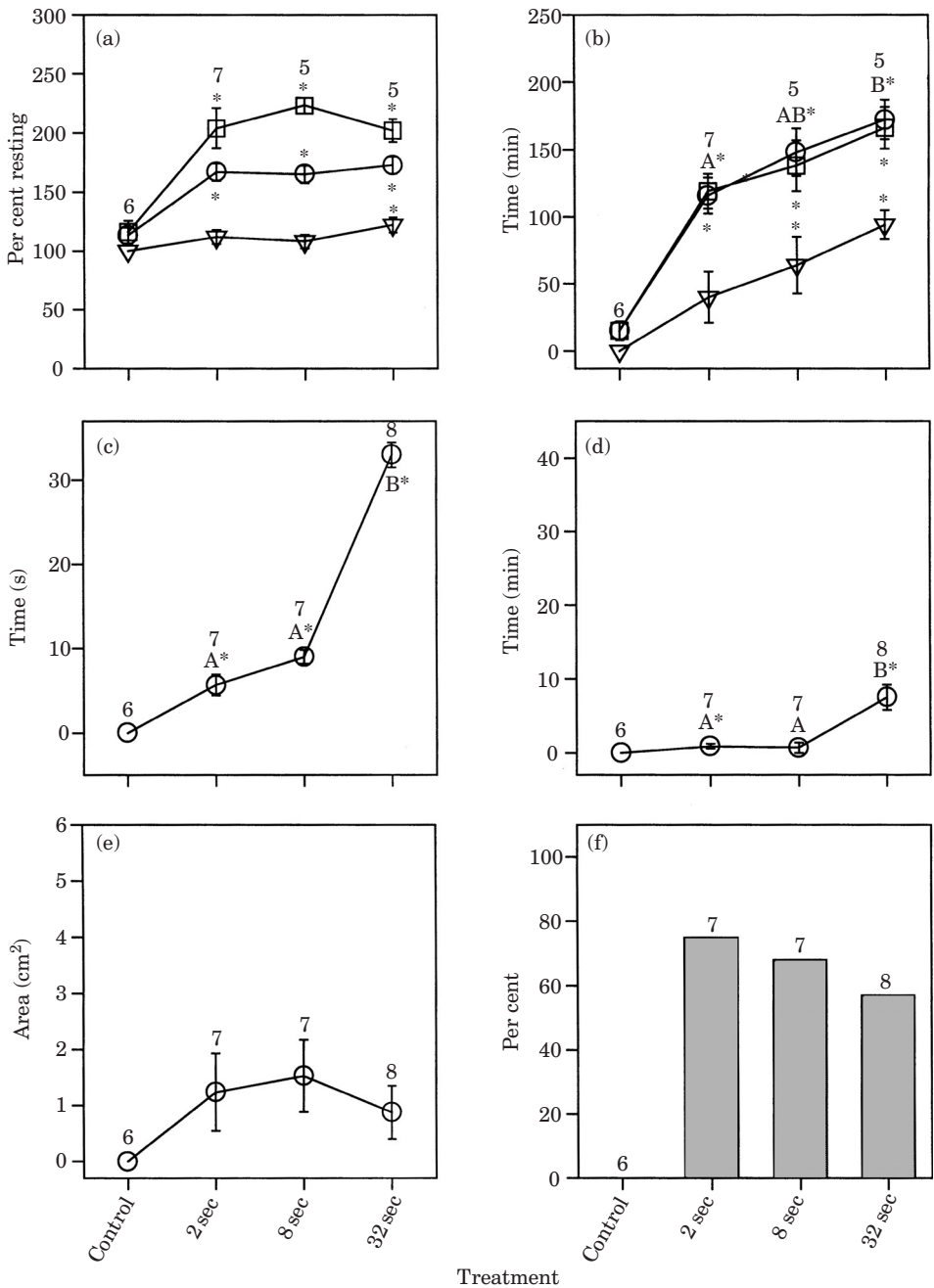


FIG. 6. Comparisons of (a) cardiac intensity (○, cardiac output; ▽, heart rate; □, stroke volume), (b) cardiac recovery, (c) cardiac arrest, (d) behavioural recovery, (e) injury area and (f) per cent injury of rainbow trout following electroshock for different durations. Fish were electroshocked with a pulse width of 2 ms at a frequency of 30 Hz, at a voltage of 100 V, and for 2, 8 and 32 s. Values (a)–(e) are presented as mean ± S.E. Numbers are sample sizes. *, the 95% CI does not overlap with 0 (or 100 for cardiac intensity). Differences across treatments are denoted with different letters.

In fact, Sharber *et al.* (1994) and Sharber & Black (1999) suggest that spinal injuries can occur at the first sign of twitching or re-orientation towards the anode because of myoclonic jerks (contraction of parallel muscle myotomes) of the white muscle on either side of the spine. When the nervous system is overloaded by a stimulus, albeit electrical, chemical or other stimuli, this often manifests itself in epileptic seizures (Penfield & Jasper, 1954; Delgado-Escueta *et al.*, 1986). Sharber and Black (1999) have now taken this concept one step further and propose that all fish reactions to electrofishing can be directly compared to the various categorizations of epilepsy (*e.g.* taxis = automatism, narcosis = petit mal seizure and tetany = grand mal seizure).

Although myoclonic jerks appear to be all that is necessary for injury to occur, it is the narcosis and tetany that cause the muscle of fishes to become essentially nonfunctional and therefore result in the loss of equilibrium and cardiac arrest. Cardiac slowing and arrest has also been observed in stinging catfish *Heteropneustes fossilis* (Bloch), Mozambique tilapia *Oreochromis mossambicus* (Peters) and freshwater prawn *Macrobrachium rosenbergii* exposed to electroshock (Biswas & Karmarker, 1979). Cardiac arrest has also been observed under more natural conditions when chum salmon *Oncorhynchus keta* (Walbaum) contract trunk musculature to discharge gametes (Uematsu *et al.*, 1983). While cessation of cardiac flow results in a reduction of oxygen transport to the tissues and consequently a decrease in aerobic metabolism, it has been suggested that this decrease in flow is a reflex response of the cardiovascular system to prevent hypertension and a similar explanation was proposed for bradycardia during periods of burst swimming (Farrell & Jones, 1992). Thus, bradycardia and even cardiac arrest may be an adaptive response mediated by the central nervous system.

Electrofishing induced cardiac arrest, however, is not an adaptive stress response, but instead represents a forced and extended contraction of the cardiac musculature that interrupts the transport of blood in the cardiovascular system. During cardiac arrest, it is unclear how much blood remains in the heart. The persistent contraction of the heart would halt the movement of blood through the vasculature. If this cardiac arrest were to persist for extended periods of time resulting in a severe depression in aerobic metabolism, the heart and other tissues not capable of anaerobic metabolism may become hypoxic and eventually anoxic. If prolonged, even tissues that can temporarily function anaerobically may become hypoxic or anoxic leading to tissue damage. Many fishes, including rainbow trout, possess coronary circulation (Farrell & Jones, 1992) but this also would be halted due to the cardiac arrest. It is unclear how long different tissues can remain without replenishment of more richly oxygenated blood, but what is clear is that the longer the duration of electroshock, the increased potential for permanent tissue damage. In this study, 32 s was the maximal period of electroshock and therefore periods of cardiac arrest for at least this duration were encountered. This duration of shocking is by no means excessive in terms of field applications. When electrofishing fluvial systems, it is typical to sweep the anode from side to side, often applying current constantly (Cooke *et al.*, 1998). When more complex habitat or deeper water is encountered, typically additional effort is expended, repeatedly exposing fishes in a confined area to the electric field (Cooke *et al.*, 1998).

Following the shock, the fish is impaired due to muscle fatigue and oxygen debt. The muscle fatigue is caused by a depletion of energy stores within the muscle and because of intra- and extra-cellular acidosis (Mitton & McDonald, 1994a). This muscle fatigue itself would result in an oxygen debt and is compounded by the acute impairment of circulatory efficiency during the shock when the heart stops. Therefore, following electroshock fishes are recovering from extreme muscular exertion and hypoxia similar to a 2–3 min bout of exhaustive exercise (Mitton & McDonald, 1994b). The results of the current study show that cardiac function may be impaired for up to 3 h following electroshock. The cardiac disturbance associated with electroshock is similar in duration to those determined for smallmouth bass *Micropterus dolomieu* Lacepède and rock bass, *Ambloplites rupestris* (Rafinesque) exposed to simulated angling events (Schreer *et al.*, 2001; Cooke *et al.*, 2001). The up to 3 h increase in plasma lactate in rainbow trout following electroshock indicates the severity of anaerobic muscle activity and the similarity to exhaustive exercise (Schreck *et al.*, 1976; Mitton & McDonald, 1994b).

Internal haemorrhaging present in most fish 24 h after the shock indicates that some physical impairment will continue for at least several days. Also, the relatively low incidence of external injury (bruising and branding) confirms findings of other studies that a higher percentage of fishes are injured by electrofishing than would be suspected from external examination (Thompson *et al.*, 1997a; Kocovsky *et al.*, 1997).

In considering the negative effects of electrofishing due to different electrofisher settings in this study, a clear pattern is not evident, except that all types of electric shock resulted in considerable impairment. Higher voltages and frequencies most consistently showed an increase in cardiac arrest, behavioural recovery (not significant for voltage) and injury (not significant). More violent and more frequent muscle contractions due to higher voltages (Snyder, 1992) and frequencies (Whaley *et al.*, 1978; Sharber *et al.*, 1994) increase the likelihood of internal injury and therefore long-term damage. More rapid development of tetany at higher frequencies may be a major contributor to the damage (Sharber *et al.*, 1994). The similarity in results for 80–8 Hz and 90 Hz indicates that most of the impact results from the initial frequency and reducing the frequency during the shock improves conditions minimally. At higher voltages, fishes are exposed to higher voltage gradients and therefore the severity of the muscular contractions during tetany will be more severe (Vibert, 1967). In the present study, this trend could be seen for a few of the variables measured, but several were not significant. Some studies have found similar results for injury (Spencer, 1967), although others have shown that higher voltages do not affect injury (Hudy, 1985; Sharber *et al.*, 1994).

Longer durations of electrofishing have more of a negative impact on short-term physiological recovery. Basically the longer periods of tetany, and consequently muscle fatigue and cardiac arrest, result in a more severe reduction in aerobic metabolism and a larger oxygen debt. The fact that cardiac intensity during recovery was similar across all settings indicates that rainbow trout are increasing CO to maximal levels even for less severe oxygen debt. Therefore, when the oxygen debt is more extreme the only option available to deal with this debt is to increase recovery time. This has also been observed for smallmouth

bass exercised for different durations and consequently to different levels of oxygen debt (Schreer *et al.*, 2001). An alternative explanation is that acidosis reduces the force of ventricular contraction resulting in a reduction in CO (Farrell & Jones, 1992). Therefore, CO and SV intensity during recovery may have been tempered by higher electrofisher settings and the consequent increase in acidosis and reduction in muscle force.

Different pulse widths had the least impact on impairment. Only injury area suggested some increase with higher settings, although the highest injury was in the moderate pulse width category. Results from the literature for pulse width are contradictory with some studies showing no effect (Collins *et al.*, 1954) while others found higher injury with longer pulse durations (Lamarque, 1990).

In putting results from the study in appropriate context, there are many factors that must be considered and how they impact electrical fields (*e.g.* distance from and orientation to the electrodes, temperature and conductivity of the water, length and condition of the fish, species and pulse shape). In this study an attempt was made to control for most of these so that the impacts across settings could be compared. Findings from this study need to be taken in the context that fish were very close to the anode or cathode (a limitation of the laboratory-based study) and this may have increased the severity of the results. Because all fish were exposed to similar conditions, however, this should not have an impact on the comparisons across settings.

One clear recommendation arising from the results of this study is that the duration of electroshock encountered by individual fish should be minimized. The duration of the shock appears to have the strongest effect on short-term (cardiovascular) recovery by directly affecting oxygen debt (although this was not measured directly). This means that repeated and continuous application of electroshock to a specific habitat (*e.g.* pool) should be avoided or if essential, kept to minimal duration. Furthermore, when using the depletion removal method for population estimates it is important to recognize the possible compounding effects from repeated electrofishing over a short period. A second recommendation, although the trends supporting this are not very strong, echoes those of several other studies suggesting that frequency and voltage should be kept to a minimum to reduce long-term recovery through injury (Whaley *et al.*, 1978; Snyder, 1992, 1995; Sharber *et al.*, 1994; Dalbey *et al.*, 1996). A final recommendation is that simple visual observations are not good indicators of the effects of electrofishing. In this and other studies (Dalbey *et al.*, 1996; Thompson *et al.*, 1997b; Kocovsky *et al.*, 1997) with low mortality, there was still significant long-term injury and short-term physiological impairments. As well, external injury (bruising or branding) and behavioural status are not good indicators of internal injuries (Thompson *et al.*, 1997b; Kocovsky *et al.*, 1997) or physiological disturbance. Changes in pulse width had the smallest impact and therefore no recommendations regarding this variable can be made from these findings.

In conclusion, it is very important that fisheries biologists do not overlook the potential effects of exposure to electroshock. This could bias mark-recapture and radiotelemetry studies and add additional pressure to declining or endangered fish populations. To date the best course of action to take is to set electrofisher settings that minimize all levels of impairments without compromising

efficiency too greatly. From this work it appears that voltage and frequency should be minimized to reduce injury while the duration of the shock should be reduced to minimize cardiovascular recovery. The data, however, only weakly support these recommendations. A stronger conclusion that can be drawn from this study is that regardless of the settings, electrofishing causes physiological impairment (specifically to the heart) and internal injury that may be considerable even in fishes that appear normal.

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