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Serological Changes Associated with Gill-Net Capture and Restraint in Three Species of Sharks

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Abstract.—To investigate the biochemical effects of capture and restraint on sharks, 17 serum constituents were measured in three species (bonnethead shark *Sphyrna tiburo*, blacktip shark *Carcharhinus limbatus*, and bull shark *C. leucas*) after gill-net capture. The relative degree of capture effects on each animal was judged using an index of behavioral response devised for use in tag−recapture studies. Serum from each shark was assayed for glucose, creatinine, uric acid, sodium, chloride, potassium, inorganic phosphate, total and ionized calcium, total protein, albumin, globulin, alkaline phosphatase, lactate, lactate dehydrogenase, aspartate aminotransferase, and total iron. In addition, hematocrit was measured from whole blood for each shark. When correlated with the relative degree of capture effects, there were significant intraspecific changes in the concentration of potassium, lactate, inorganic phosphate, uric acid, alkaline phosphatase, aspartate aminotransferase, total and ionized calcium, and glucose. Significant interspecific differences in the concentration of sodium, chloride, potassium, total protein, albumin, globulin, lactate dehydrogenase, aspartate aminotransferase, ionized calcium, alkaline phosphatase, and glucose in minimally stressed animals also were observed. The study suggests that the deleterious effects of gill-net capture and restraint probably involve respiratory and metabolic acidosis and hypoglycemia as well as cellular damage. Species-specific and individual differences in the mortality of sharks caught in gill nets are likely related to an animal's respiratory physiology and degree of struggling upon capture as well as to the extent of net entanglement around the gill area.

Catch-and-release fishing has become popular among recreational fishermen and, with the implementation of size limits, quotas, and prohibitions on taking certain species in commercial fisheries, it is increasingly practiced by commercial fishermen as well. To understand the effects of capture and handling on individual fish and the subsequent impact on fish populations, studies have been conducted on hooking mortality (e.g., Wertheimer 1988; Wertheimer et al. 1989; Schaefer 1989; Bendock and Alexandersdottir 1993; Meals and Miranda 1994), delayed mortality (e.g., Bennett et al. 1989; Bugley and Shepherd 1991; Gjernes et al. 1993), handling mortality (e.g., Wassenberg and Hill 1989; Pierce and Tomcko 1993), and the effects of specific gear types (e.g., Chai et al. 1992).

Recent declines in the abundance of sharks worldwide (NMFS 1993; Bonfil 1994) and the increasing practice of catch-and-release shark fishing call for studies examining the specific physiological responses of sharks caught in fishing gear. When sharks are caught and restrained, a complex combination of physiological responses in the animals may result, including exhaustive exercise, stress response, and effects on ventilatory and metabolic processes. Different methods of capture may lead to different combinations of these responses, and both stress-induced mortality and the serological responses to capture and restraint may differ markedly among species (Cliff and Thurman 1984; Jones and Andrews 1990; Smith 1992). The few published studies on shark stress have dealt with changes in blood chemistry and hematology resulting from environmental changes, such as in degree of salinity or those resulting from confinement (Butler et al. 1979; Mandrup-Poulsen 1981; Bedford 1982; Torres et al. 1986b) or from surgical stress (Torres et al. 1986a). In contrast, the consequences of exhaustive exercise associated with capture, handling, and restraint have rarely been studied. Several previous studies of shark capture suggest that marked changes in hematological constituents, including blood chemistry and blood oxygen transport systems, may be typical responses to capture and restraint (Cliff and Thurman 1984;

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Wells and Davie 1985; Wells et al. 1986; Smith 1992).

The purpose of this study was to determine the serological changes in sharks following gill-net capture and restraint. Gill nets are used throughout the world in coastal and pelagic fisheries, and sharks are commonly caught in this gear type as targeted or bycatch species (Bonfil 1994). In the United States, gill nets are commonly used in the coastal zone to catch a variety of fishes, and a number of fisheries operate within shark nursery areas (Hueter and Manire 1994). Where fishery regulations preclude the retention of sharks captured by gill net, there is concern that the released sharks may experience substantial physiological trauma that results in poor postrelease survival. This is of special concern when gill nets are deployed in shark nurseries, where juvenile or small adult sharks may be a substantial part of the bycatch.

To evaluate some of the impacts of catch-and-release gill-net fishing on small sharks, we sought to understand the physiological effects of this practice on these animals. To accomplish this, we examined the serum constituent values in three species of sharks (bonnethead shark *Sphyrna tiburo*, blacktip shark *Carcharhinus limbatus*, and bull shark *C. leucas*) that were affected to varying degrees by gill-net capture and restraint. These sharks were chosen because previous research had found that the mortality rates associated with gill-net capture and restraint were species specific, with relatively higher mortality in blacktip and bonnethead sharks and lower mortality in bull sharks (Hueter and Manire 1994). Although stress indices normally are plotted against time beginning with the initial application of a stressor, this was not feasible in our study as it was impossible to determine the exact time of initial capture of each fish. For this study, therefore, we utilized an index of relative behavioral response to capture and restraint that was developed for shark tag-and-release studies (Hueter and Manire 1994) and that has been used to estimate postrelease (cryptic) mortality (author's unpublished data). The use of this index in the current study made it possible to evaluate serological changes over a range of relative effects of capture and restraint and to determine correlations between the sharks' physiological and behavioral responses to gill-net capture.

Methods

Sharks were collected using anchored gill nets (11.75- or 15.25-cm stretch mesh) in inshore waters of southwestern Florida, namely, Pine Island Sound (26°27′N, 82°08′W) and Tampa Bay (27°51′N, 82°38′W), between April and July 1994. All sharks were captured in waters less than 3 m deep. Nets were fished for 45–60 min and total sampling time never exceeded 3 h. Although it was not usually possible, general observations of behavior were occasionally made when sharks were seen encountering the net.

After capture, each shark was removed from the net, its gender was determined, and it was measured, weighed, and tagged with nylon-barbed dart tags (Hallprint, Australia). A blood sample (5–7 mL) then was taken via caudal venipuncture. All blood samples except those from some bull sharks were taken within 1 h of the time the shark first struck the net. A portion of each blood sample was drawn into a microhematocrit tube that was then sealed, and the remainder of the sample was placed in a 10-mL sealed tube. Blood tubes were kept on ice (up to 6 h) until further processing.

After the handling of each shark was completed, its relative condition was assessed using a ''vitality code'' based on behavior at release, a method developed by Hueter and Manire (1994) that is similar to that of Wertheimer et al. (1989). The condition of each animal was assigned to one of five categories, ranging from condition 1 (good) to condition 5 (moribund or dead) using the following criteria:

Condition 1 (good): No revival time required when the shark was returned to the water; rapid swimming upon release, usually with a vigorous splash (i.e., still capable of burst as well as maintenance swimming).

Condition 2 (fair): No revival time required; slow but strong swimming upon release (i.e., incapable of burst swimming but still capable of maintenance swimming).

Condition 3 (poor): Short revival time (up to 30 s) required; once revived, slow and sometimes atypical swimming upon release (i.e., incapable of normal maintenance swimming but still exhibiting some directed swimming).

Condition 4 (very poor): Long revival time (>30 s) required; once revived, limited or no swimming observed upon release but respiration functional (i.e., incapable of directed swimming but still alive).

Condition 5 (moribund or dead): Dead upon removal from gear or moribund and unable to revive even after a long submergence time.

Sharks in condition 5 were used only if they were still alive at the time blood was drawn (that

is, moribund but not yet dead); sharks in this category were not released. This method of rating release condition has proven useful in determining postrelease (cryptic) mortality in a shark tagging study (author's unpublished data). That study indicates that postrelease mortality increases as release condition deteriorates, suggesting that the animal's physiological state after capture and restraint is correlated with these behavioral conditions as well.

Upon return to the laboratory, microhematocrit tubes were centrifuged at 5,000 rpm (1,286 × gravity) for 10 min, and hematocrit was determined using a microcapillary reader. After coagulation on ice for 3–6 h, blood samples were centrifuged at 5,000 rpm (1,286 × gravity) for 10 min. Serum was separated from sedimented cells and frozen at −20°C. Serum samples were transported on dry ice to a commercial pathology laboratory for subsequent analysis of glucose, creatinine, uric acid, sodium, chloride, potassium, inorganic phosphate, total and ionized calcium, total protein, albumin, globulin, alkaline phosphatase (ALP), lactate, lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and total iron, along with visual determination of hemolysis (slight, moderate, or heavy). Lactate concentrations were reported by the commercial laboratory up to 12 mmol/L; higher values were reported simply as being greater than 12 mmol/L, in which case a value of 12 mmol/L was used for data analysis.

As with any study of wild animals, true ''unstressed'' baseline values for serum constituents (Love 1970; Love 1980) could not be obtained in the present study, but condition-1 sharks were considered to have experienced ''minimal stress'' (nonexhausted). The serological values from these minimally stressed animals were compared with those from animals that were more highly stressed or exhausted (conditions 2–4) or moribund (condition 5) to determine whether changes in serum variables were associated with capture and restraint in these three species. Minimal stress values were also compared among the three species to determine interspecific differences in response.

Statistical analyses were performed using SigmaStat (Jandel Scientific 1997). Most data were analyzed using a Kruskal−Wallis one-way analysis of variance (ANOVA) of ranks test, followed by a Dunn's multiple-range test to examine differences between the median values at different levels of relative effect of capture and restraint. Pearson's product-moment coefficient correlation was used to test correlations. Significance was determined at $\alpha < 0.05$.

TABLE 1.—Capture and restraint effect condition of netted and restrained sharks. Values are the number of sharks collected and the percentages of the total collection of that species. Definitions of the various conditions are given in the text.

Condition	Bonnethead sharks	Blacktip sharks	Bull sharks
1. Good	4 (10%)	9 (27%)	11 (41%)
2. Fair	10 (26%)	4 (12%)	9 (33%)
3. Poor	6 (15%)	7 (21%)	2 (7%)
4. Very poor	7 (18%)	5 (15%)	None collected
5. Moribund	12 (31%)	8 (25%)	5 (19%)
Totals	39	33	27

Results

Blood samples were collected from 39 bonnethead sharks (14 male, 25 female), 33 blacktip sharks (13 male, 20 female), and 27 bull sharks (13 male, 14 female)(Table 1). Both juvenile and adult bonnethead sharks (54–108 cm total length [TL]) were captured, but only juvenile blacktip (54–86 cm TL) and bull sharks (68–110 cm TL) were collected.

Although the moment of capture was not usually observed, when it was, behavioral differences were noted among the three species' responses to gill-net capture. In general, bonnethead and blacktip sharks struggled dramatically upon encountering the net, became tightly entangled (especially in the head and gill regions), and experienced some skin abrasions and fin damage. In contrast, bull sharks did not appear to struggle for long or to become entangled in the gill region (more in the fin and tail areas) and subsequently exhibited little surface damage. In addition to these behavioral differences among the species, interspecific differences in immediate (observable) mortality rates were seen. These mortality rates were consistent with those reported by Hueter and Manire (1994), with bonnethead and blacktip sharks exhibiting relatively higher observable mortality than bull sharks (Table 1) under similar capture conditions.

Interspecific differences in several serum constituents were found when minimally stressed animals were compared (Table 2). Statistically significant differences among species were found in several electrolytes (sodium, potassium, chloride, and ionized calcium) and enzymes (ALP, LDH, and AST), as well as in a number of other serum constituents (glucose, total protein, albumin, and globulin).

TABLE 2.—Interspecific comparison of various serum constituents and hematocrit in minimally stressed (condition 1) sharks. Values are the medians (25th–75th quartiles).

Variable	Bonnethead sharks (n = 4)	Blacktip sharks (n = 9)	Bull sharks (n = 11)	Significance[a]
Glucose (mg/dL)	183 (175–192)	62 (48.5–67.3)	54.5 (41–64)	P = 0.005; BH > BT, BU
Creatinine (mg/dL)	0.2 (0.2–0.3)	0.2 (0.2–0.3)	0.2 (0.2–0.3)	NS
Uric acid (mg/dL)	0.35 (0.20–0.40)	0.10 (0.10–0.13)	0.20 (0.10–0.60)	NS
Sodium (mEq/L)	312 (306–317)	321 (313–329)	288 (285–294)	P < 0.001; BT > BU
Potassium (mEq/L)	6.4 (6.0–7.1)	4.0 (3.7–4.3)	6.3 (5.7–6.7)	P < 0.001; BH, BU > BT
Chloride (mEq/L)	207 (206–209)	208 (207–212)	202 (201–204)	P < 0.001; BT > BU
Total calcium (mg/dL)	16.5 (15.9–17.2)	17.1 (16.3–17.7)	17.3 (16.0–17.6)	NS
Inorganic phosphate (mg/dL)	7.4 (7.0–8.0)	8.0 (7.6–8.5)	8.1 (6.9–8.8)	NS
Ionized calcium (mg/dL)	10.9 (10.3–11.1)	12.1 (11.8–12.9)	11.7 (10.7–11.9)	P = 0.003; BT > BH, BU
Total protein (g/dL)	3.2 (3.0–3.3)	2.2 (2.2–2.6)	2.9 (2.8–2.9)	P = 0.002; BH, BU > BT
Albumin (g/dL)	1.0 (0.9–1.0)	1.0 (1.0–1.1)	1.1 (1.0–1.2)	P < 0.05; BU > BH
Globulin (g/dL)	2.3 (2.1–2.4)	1.2 (1.2–1.4)	1.8 (1.6–1.8)	P < 0.001; BH, BU > BT
ALP (μg/L)	5.0 (3.0–8.5)	2.0 (1.0–2.3)	2.0 (1.0–4.8)	P < 0.05; BH > BT
LDH (μg/L)	2.0 (1.0–3.0)	6.0 (3.5–7.0)	21 (10.5–42.0)	P = 0.002; BU > BT, BH
AST (μg/L)	88.5 (68.5–108)	28 (25.0–29.3)	16 (6.0–22.0)	P < 0.001; BH > BU
Lactate (mmol/L)	3.9 (2.2–5.3)	4.7 (4.1–6.4)	6.3 (4.5–11.8)	NS
Total iron (mEq/L)	39 (25.5–53.0)	19 (17.3–29.5)	19 (15.8–40.5)	NS
Hematocrit (%)	29.6 (27.3–30.0)	27.5 (25.0–29.5)	30.0 (28.0–30.0)	NS

[a] Abbreviations are as follows: BH = bonnethead, BT = blacktip, BU = bull.

Within each species, several serum constituents changed significantly with increasing stress levels (Tables 3–5). One biologically significant change was the increase in serum lactate in all three species. Lactate concentrations tripled in bonnethead sharks (increasing from 3.9 to at least 12.0 mmol/L; Table 3) and blacktip sharks (4.7 to at least 12.0 mmol/L; Table 4) and doubled in bull sharks (6.3 to at least 12.0 mmol/L; Table 5) with increasing relative effects of capture and restraint. A number of other significant changes also occurred. Serum glucose decreased significantly in both bonnethead ($P < 0.001$) and bull ($P < 0.05$) sharks, with values declining 33% and more than 50%, respectively. Inorganic phosphate increased significantly in all three species, and AST increased in bull sharks ($P = 0.009$).

There were no significant changes in the hematocrit of any species associated with condition factor (overall median [25th−75th quartiles]: bonnethead sharks, 29.6% [27.3–30.0%]; blacktip sharks, 27.5% [25.0–29.5%]; bull sharks, 30.0% [28.0–30.0%]). Thus, there was no evidence of hemodilution or hemoconcentration in response to capture and restraint, at least not in the hour following the initial capture event. Extensive hemolysis was evident only in samples from bonnethead sharks, where it was associated with a significant ($P < 0.001$) increase in potassium; this would be expected with the dumping of intracellular potassium from the erythrocytes into the serum. However, all three species had significant potassium increases, even though blacktip and bull shark serum exhibited very little

TABLE 3.—Comparison of various serum constituents and hematocrit in bonnethead sharks at five different capture effect levels (conditions). Values are medians (25th–75th quartiles).

Variable	Condition 1 (n = 4)	Condition 2 (n = 10)	Condition 3 (n = 6)	Condition 4 (n = 7)	Condition 5 (n = 12)	Significance[a]
Glucose (mg/dL)	183 (175–192)	169 (156–188)	188 (179–199)	154 (131–163)	125 (115–141)	P < 0.001; 1, 2, 3 > 5
Creatinine (mg/dL)	0.2 (0.2–0.3)	0.2 (0.2–0.3)	0.2 (0.2–.03)	0.2 (0.2–0.3)	0.3 (0.2–0.3)	NS
Uric acid (mg/dL)	0.35 (0.20–0.40)	1.15 (0.30–1.60)	0.80 (0.30–1.05)	1.8 (1.33–2.30)	1.9 (1.20–2.40)	P = 0.009; DU
Sodium (mEq/L)	312 (306–317)	307 (287–315)	318 (291–326)	298 (291–303)	301 (286–306)	NS
Potassium (mEq/L)	6.4 (6.0–7.1)	8.8 (7.0–11.0)	7.5 (6.8–8.5)	10.4 (9.8–12.1)	16.1 (11.4–17.5)	P < 0.001; 5 > 1, 2, 3
Chloride (mEq/L)	207 (206–209)	209 (207–209)	205 (201–208)	203 (202–210)	210 (205–213)	NS
Total calcium (mg/dL)	16.5 (15.9–17.2)	16.8 (15.6–17.4)	17.0 (15.7–18.3)	16.1 (15.3–16.8)	16.8 (15.8–17.5)	NS
Inorganic phosphate (mg/dL)	7.4 (7.0–8.0)	9.5 (8.3–10.5)	8.4 (7.5–9.9)	10.5 (9.8–12.4)	13.8 (10.5–16.3)	P = 0.001; 5 > 1
Ionized calcium (mg/dL)	10.9 (10.3–11.1)	10.8 (10.6–11.1)	11.2 (10.1–12.2)	10.8 (9.6–10.9)	9.9 (9.2–10.4)	NS
Total protein (g/dL)	3.2 (3.0–3.3)	3.1 (2.9–3.9)	3.1 (2.9–3.4)	3.6 (2.7–3.8)	3.9 (3.7–4.3)	NS
Albumin (g/dL)	1.0 (0.9–1.0)	1.0 (0.9–1.1)	1.0 (0.9–1.1)	1.1 (0.9–1.1)	1.3 (1.1–1.3)	NS
Globulin (g/dL)	2.3 (2.1–2.4)	2.1 (2.0–2.6)	2.2 (1.8–2.4)	2.4 (1.8–2.7)	2.7 (2.5–3.0)	NS
ALP (μg/L)	5.0 (3.0–8.5)	8.5 (3.0–31.0)	2.0 (1.0–7.0)	11.0 (3.3–25.8)	41.5 (10.0–47.5)	P < 0.05; DU
LDH (μg/L)	2.0 (1.0–3.0)	2.0 (2.0–4.0)	2.0 (2.0–4.0)	2.0 (0.5–7.5)	2.0 (0.0–3.0)	NS
AST (μg/L)	88.5 (68.5–108.0)	82.0 (50.0–137.0)	65.5 (51.0–105.0)	34.0 (24.3–56.0)	61.5 (36.5–87.5)	NS
Lactate (mmol/L)	3.9 (2.2–5.3)	7.5 (5.0–9.1)	9.1 (6.6–11.2)	8.1 (6.7–9.1)	12.0 (12.0–12.0)	P = 0.001; 5 > 1, 2
Total iron (mEq/L)	39.0 (25.5–53.0)	52.5 (39.0–78.0)	29.0 (24.0–32.0)	52.0 (31.3–98.0)	69.0 (44.0–103.5)	NS
Hematocrit (%)	28.5 (27.5–30.0)	30.0 (28.5–31.8)	25.5 (23.0–28.0)	29.0 (29.0–29.0)	29.5 (28.5–31.5)	P < 0.05; 2 > 3

[a] Abbreviations are as follows: DU difference undetected (Dunn).

hemolysis. Concentrations of sodium, chloride, creatinine, total protein, albumin, globulin, LDH, and total iron did not change significantly in any of the three species.

The number of samples obtained for each gender at each condition was insufficient for a robust statistical comparison between sexes due to high variability and uneven numbers. An ANOVA on ranks revealed no differences between adults and juveniles in bonnethead sharks, and there were no significant correlations between either weight or length (age) and any of the serum constituents measured in any species.

Discussion

Capture and restraint of an animal elicits a complex set of responses that, when combined, may lead to mortality. These responses will involve the circulatory, respiratory, endocrine, and muscular systems at the very least and at the worst every tissue in the body. The degree to which blood variables are affected will depend upon the extent to which each system or tissue is affected. Although this study was not designed to separate out each process involved, it does help explain some of the effects on the organism as a whole.

Interspecific Differences

Several of the observed interspecific differences in the serum values of minimally stressed sharks are probably due to ambient salinity differences. The bull sharks from the study area were generally found in salinities ranging from 3.0‰ to 28.5‰ (mean, 24.6‰), while bonnethead sharks were found at 15.4–36.1‰ (mean, 29.7‰) and blacktip sharks at 15.8–38.1‰ (mean, 28.2‰). Interspecific comparisons reveal that bull sharks had significantly ($P < 0.009$) lower serum sodium and

TABLE 4.—Comparison of various serum constituents and hematocrit in blacktip sharks at five different capture effect levels (conditions). Values are medians (25th–75th quartiles).

Variable	Condition 1 (n = 9)	Condition 2 (n = 4)	Condition 3 (n = 7)	Condition 4 (n = 5)	Condition 5 (n = 8)	Significance
Glucose (mg/dL)	62.0 (48.5–67.3)	65.5 (55.0–86.0)	59.0 (52.5–68.0)	57.0 (44.8–81.3)	44.0 (31.5–60.0)	NS
Creatinine (mg/dL)	0.2 (0.2–0.3)	0.3 (0.25–0.30)	0.3 (0.3–0.3)	0.2 (0.2–0.3)	0.3 (0.30–0.35)	NS
Uric acid (mg/dL)	0.1 (0.10–0.13)	0.4 (0.10–0.75)	0.8 (0.20–1.18)	0.6 (0.43–0.75)	1.15 (0.95–1.20)	$P < 0.001$; 5 > 1
Sodium (mEq/L)	321 (313–329)	336 (328–350)	325 (321–338)	325 (315-328)	328 (321–333)	NS
Potassium (mEq/L)	4.0 (3.7–4.3)	4.9 (4.3–5.4)	6.0 (4.6–7.7)	5.9 (5.2–6.5)	7.3 (7.0–9.7)	$P < 0.001$; 5 > 1
Chloride (mEq/L)	208 (207–212)	214 (211–215)	209 (208–210)	210 (205–213)	212 (210–214)	NS
Total calcium (mg/dL)	17.1 (16.3–17.7)	17.8 (17.6–18.2)	17.5 (17.1–18.8)	18.3 (17.7–18.4)	19.9 (18.4–21.7)	$P = 0.009$; 5 > 1
Inorganic phosphate (mg/dL)	8.0 (7.6–8.5)	9.0 (7.4–9.3)	8.7 (8.0–9.1)	9.2 (8.6–9.7)	10.3 (8.5–11.7)	$P < 0.05$; 5 > 1
Ionized calcium (mg/dL)	12.1 (11.8–12.9)	13.0 (12.5–13.5)	12.6 (11.9–13.3)	12.9 (12.3–13.2)	13.6 (13.3–15.3)	$P < 0.05$; 5 > 1
Total protein (g/dL)	2.2 (2.2–2.6)	2.3 (2.1–2.4)	2.5 (2.2–2.5)	2.2 (2.1–2.7)	2.4 (2.1–2.7)	NS
Albumin (g/dL)	1.0 (1.0–1.1)	1.1 (1.1–1.2)	1.1 (1.0–1.1)	1.1 (1.0–1.1)	1.1 (1.0–1.2)	NS
Globulin (g/dL)	1.2 (1.2–1.4)	1.2 (1.0–1.3)	1.4 (1.2–1.5)	1.1 (1.1–1.6)	1.4 (1.1–1.6)	NS
ALP (μg/L)	2.0 (1.0–2.3)	1.0 (0.0–4.5)	3.0 (2.3–3.0)	1.0 (1.0–3.0)	4.0 (4.0–6.0)	$P < 0.05$; 5 > 1
LDH (μg/L)	6.0 (3.5–7.0)	12.0 (6.0–20.0)	4.0 (0.5–9.8)	11.0 (5.8–18.3)	3.0 (2.0–6.0)	NS
AST (μg/L)	28.0 (25.0–29.3)	56.0 (40.5–86.5)	42.0 (31.0–50.5)	47.0 (13.8–82.8)	37.0 (19.5–59.5)	NS
Lactate (mmol/L)	4.7 (4.1–6.4)	6.6 (5.2–7.3)	8.6 (7.2–12.0)	11.1 (10.1–11.6)	12.0 (11.2–12.0)	$P = 0.007$; 5 > 1
Total iron (mEq/L)	19.0 (17.3–29.5)	18.0 (15.0–25.0)	23.0 (19.0–32.0)	21.0 (18.5–32.3)	29.0 (27.5–41.5)	NS
Hematocrit (%)	25.0 (25.0–27.0)	29.5 (27.0–32.0)	29.5 (27.0–30.0)	28.5 (26.5–30.0)	26.0 (24.8–28.5)	NS

chloride concentrations than the other two species. Mandrup-Poulsen (1981) found that both sodium and chloride increased with increasing salinity in bonnethead sharks, and Piermarini and Evans (1998) found similar results in Atlantic stingrays *Dasyatis sabina*.

Other interspecific differences may be the result of different diets (e.g., glucose, total protein, albumin, potassium, and ionized calcium) or unknown biochemical differences (e.g., ALP, LDH, and AST). It is also possible that some of these differences (especially ALP and LDH) are due to ''minimal stress'' (i.e., that gill-net capture alone may elevate some values above true baselines, which are virtually impossible to obtain from wild animals). It is unlikely that any of these interspecific differences would account for the observed differences in mortality.

Time Frame of Change

The time frame of the change is critical to understanding the results of the present study. In general, the longer a shark was in the net, the worse its condition was. However, even condition-5 animals (except bull sharks) were sampled less than 1 h following initial contact with the gill net. Therefore, the values seen in this study may be less than those seen in other studies, in which fish were exercised to exhaustion and then allowed to recover (i.e., with more time elapsing between the stress and blood sampling). Holeton and Heisler (1983) found maximum blood lactate concentrations in larger spotted dogfish *Scyliorhinus stellaris* 4–8 h after exercise and the acid–base status restored to normal 10–14 h after exercise. Milligan (1996) found arterial blood pH to be disrupted for up to 4 h after exercise in rainbow trout *Oncor-*

TABLE 5.—Comparison of various serum constituents and hematocrit in bull sharks at four different capture effect levels (conditions). Values are medians (25th–75th quartiles).

Variable	Condition 1 (n = 11)	Condition 2 (n = 9)	Condition 3 (n = 2)	Condition 5 (n = 5)	Significance[a]
Glucose (mg/dL)	54.5 (41.0–64.0)	64.0 (57.3–72.5)	64.0 (49.0–79.0)	22.0 (11.5–32.3)	$P < 0.05$; $2 > 5$
Creatinine (mg/dL)	0.2 (0.2–0.3)	0.3 (0.2–0.3)	0.25 (0.2–0.3)	0.3 (0.28–0.33)	NS
Uric acid (mg/dL)	0.2 (0.1–0.6)	0.7 (0.18–0.90)	0.7 (0.2–1.2)	0.3 (0.18–0.73)	NS
Sodium (mEq/L)	288 (285–294)	291 (286–301)	286.5 (285–288)	283 (278–291)	NS
Potassium (mEq/L)	6.3 (5.7–6.7)	6.8 (6.5–7.6)	7.3 (7.0–7.6)	10.1 (9.4–12.9)	$P = 0.001$; $5 > 1$
Chloride (mEq/L)	202 (201–204)	204 (201–209)	200 (199–201)	199 (198–200)	NS
Total calcium (mg/dL)	17.3 (16.0–17.6)	18.8 (17.1–19.5)	18.1 (16.9–19.2)	18.6 (17.2–19.0)	NS
Inorganic phosphate (mg/dL)	8.1 (6.9–8.8)	9.1 (8.7–10.5)	8.7 (7.4–10.0)	13.7 (10.8–14.2)	$P = 0.004$; $5 > 1$
Ionized calcium (mg/dL)	11.7 (10.7–11.9)	12.2 (11.8–12.7)	12.1 (10.6–13.5)	12.9 (11.9–13.1)	NS
Total protein (g/dL)	2.9 (2.8–2.9)	2.9 (2.7–3.1)	3.0 (2.5–3.5)	2.6 (2.3–2.8)	NS
Albumin (g/dL)	1.1 (1.03–1.18)	1.1 (0.98–1.20)	0.95 (0.90–1.00)	1.1 (1.0–1.13)	NS
Globulin (g/dL)	1.8 (1.63–1.80)	1.8 (1.68–2.05)	2.05 (1.50–2.60)	1.4 (1.25–1.78)	NS
ALP (μg/L)	2.0 (1.0–4.8)	3.0 (2.0–4.0)	1.0 (1.0–1.0)	7.0 (3.8–7.8)	NS
LDH (μg/L)	21 (11–42)	35 (21–72)	59 (52–66)	78 (22–99)	NS
AST (μg/L)	16 (6–22)	23 (16–47)	51 (43–58)	66 (27–97)	$P = 0.009$; DU
Lactate (mmol/L)	6.3 (4.5–11.8)	12.0 (9.6–12.0)	11.4 (10.7–12.0)	12.0 (12.0–12.0)	$P < 0.05$; DU
Total iron (mEq/L)	19.0 (15.8–40.5)	32.0 (16.8–48.8)	11.0 (10.0–12.0)	41.0 (21.3–59.0)	NS
Hematocrit (%)	30.0 (28.0–31.0)	32.0 (29.5–32.4)	27.0 (23.0–31.0)	28.0 (22.5–30.8)	NS

[a] Abbreviations are as follows: DU = difference undetected (Dunn).

hynchus mykiss. Therefore, the results of this study provide neither maximum values nor recovery values.

Respiratory and Metabolic Acidosis

All the sharks in this study likely experienced some degree of respiratory hypoxia in response to gill-net capture and restraint, which was exacerbated when they were removed from the water. It has been reported that some continuously swimming sharks, which rely almost exclusively on ram ventilation, will suffocate when prevented from swimming forward (Clark and Kabasawa 1977). In contrast, other sharks are known to swim only periodically and can maintain their resting ventilation rate by the action of respiratory muscles (Piiper et al. 1977). We are unaware of any reports of bonnethead or blacktip sharks that were observed in the wild resting motionless on the bottom while healthy, and both species are known to swim continuously in captivity. In contrast, bull sharks will routinely cease activity and rest on the bottom in captivity (C. Manire, personal observation). Based on these behavioral patterns, bonnethead and blacktip sharks would be expected to be more susceptible to oxygen deprivation when restrained because they require a minimal swimming speed to adequately oxygenate their tissues (Gruber and Keyes 1981). In such animals, total restriction from movement would quickly lead to oxygen deprivation at the cellular level. Therefore, bull sharks have an advantage over bonnethead and blacktip sharks when movement is restricted by netting (Clark and Kabasawa 1977). Hence, bonnethead and blacktip sharks would be expected to undergo respiratory acidosis to a greater extent than bull sharks when captured in a gill net. This alone would make these two species more susceptible to

mortality. Likewise, the bonnethead and blacktip sharks became entangled in the gill region much more frequently than the bull sharks, which probably increased the extent to which they experienced respiratory acidosis and mortality.

Other behavioral differences among the three species likely contributed to the different mortality rates observed. Both bonnethead and blacktip sharks thrashed violently while in the nets, as evidenced by their degree of entanglement when removed. This period of hyperactivity certainly increased energetic requirements, thereby exacerbating the respiratory acidosis as well as initiating metabolic acidosis (Milligan 1996). In contrast, judging from direct visual observations and the degree of net entanglement, bull sharks did not exert intense efforts to escape; this probably allowed these sharks to remain in the nets for an extended period before experiencing either respiratory or metabolic acidosis.

All three species in our study experienced significant increases in serum lactate with increasing effects of capture and restraint. The sharks underwent a period of hyperactivity when struggling in the net that increased their energetic demands. This increased demand for energy is partially met through anaerobic respiration in white muscle, with the concomitant production of lactic acid (Bone 1988). This mechanism has been demonstrated in the epaulette shark *Hemiscyllium ocellatum* (Wise et al. 1998) and in the small spotted dogfish *Scyliorhinus canicula* (Butler and Taylor 1975; Butler et al. 1979) under hypoxic conditions. The increase in serum lactate that was observed in all three species in this study indicates the presence of a portion of the dissociation products of lactic acid (i.e., lactate and H^+ ions), which were apparently diffused out of the white muscle cells into the blood, adding to acidemia. Wood et al. (1983) and Wood (1991) suggested that only about 20% of the postexercise lactate and proton load in fish white muscle mass is released into the blood, with 80% remaining within the cells to be slowly removed by metabolic processes in situ. Although pH was not measured directly here, the significant increase in lactate in each species likely would have altered the blood pH.

Species-specific responses to increased lactate concentrations are suggested by previous studies. Cliff and Thurman (1984) noted that dusky sharks *C. obscurus* experienced a sharp decline in bicarbonate ions during the first 10 min of hyperactivity induced by capture and transport. In contrast, Holeton and Heisler (1978) suggested that branchial uptake of bicarbonate ions from the external environment by the larger spotted dogfish may improve the buffering capacity of its blood. The acid–base regulation in this species was accomplished via branchial HCO_3^-/Cl^- and H^+/Na^+ ion exchange mechanisms. In our study no changes were observed in either serum sodium or chloride in any species. Two possible explanations are inadequate time for the serum levels to have changed and restriction of water circulation through the gills, which would have inhibited the ion exchange process.

Another factor that may be related to the relative mortality of the three species of shark is water temperature. Hueter and Manire (1994) found that mortality from gill-net capture increased with water temperature. Heisler (1988) found that although the arterial pH of the larger spotted dogfish is tightly regulated in steady-state conditions, the pH of arterial blood and body fluids decreases with increasing temperature. This would exacerbate the acidemia when sharks are captured in warmer water, thereby making the animals more susceptible to the deleterious effects of respiratory and metabolic acidosis of capture and restraint.

Hypoglycemia

Although hyperglycemia is expected with stress in many teleosts (Mazeaud et al. 1977; Specker and Schreck 1980; Wells et al. 1986) and burst exercise alone does not affect serum glucose levels (Moyes and West 1995), both the bonnethead and bull sharks exhibited significant declines in serum glucose with increased relative effects of capture and restraint. It should be noted that, although the differences were not statistically significant, all three species had higher serum glucose concentrations in either condition 2 or 3 than in condition 1. It is possible that the initial response was an increase in glucose that was later followed by a decrease, but a more controlled study would be necessary to determine this. This finding is consistent with Cliff and Thurman's (1984) suggestion that large decreases in glucose concentration may contribute to death in some sharks. However, in our study the bonnethead sharks had the highest glucose levels at all conditions, and yet they experienced the highest mortality. In fact, moribund (condition-5) bonnethead sharks had significantly more glucose than the other two species at any condition level. The decrease in glucose levels between conditions 1 and 5 was also greater in bonnethead sharks.

Cellular Damage

Capture and restraint disrupted the serum concentrations of potassium in all three species, leading to hyperkalemia, a condition that, regardless of its cause, may be deleterious to an animal. Cliff and Thurman (1984) attributed large increases in extracellular potassium in dusky sharks to leakage from muscle cells induced by intracellular acidosis. The correlation of potassium with lactate in our study would support this hypothesis. A portion of the potassium increase may also be attributed to the depletion of blood glucose. Shifts in potassium from the intracellular to the extracellular compartments in response to changes in blood glucose have also been observed in teleosts (Houston et al. 1971).

In this study, serum inorganic phosphate levels were elevated by capture and restraint, most likely due to leakage from damaged muscle cells. Inorganic phosphate and proteins account for most of the intracellular buffering in the white muscle of fish (Okuma and Abe 1992). The diffusion of inorganic phosphate into the blood would decrease the buffering capacity within the muscle cells. As most of the lactic acid produced in white muscle may be retained in the cell (Wood et al. 1983; Wood 1991), this diffusion of inorganic phosphate into the extracellular fluid may exacerbate a severe intracellular acid–base imbalance.

The enzyme aspartate aminotransferase (AST) increased significantly with capture and restraint only in bull sharks. In vertebrates, AST elevations are known to occur with liver damage (Price and Stevens 1982), cardiac tissue damage (Martini 1995), and physical damage to other tissues. When subjected to severe skin abrasions during transport, channel catfish *Ictalurus punctatus* exhibit large increases in AST (Bentinck-Smith et al. 1987), and serum AST levels are known to increase due to release from hemolyzed blood cells in teleosts (Stoskopf 1993). The lack of AST elevation in bonnethead sharks would indicate that it is not released from hemolyzed blood cells in this species. Gelsleichter et al. (1998) demonstrated oxytetracycline-induced increases in serum AST in the nurse shark *Ginglymostoma cirratum* and suggested that it may have indicated hepatic damage. Similarly, the elevated levels of serum AST that we observed in bull sharks indicate cell damage of some type, but the particular tissue damaged and the exact meaning of the elevated AST are unknown. It is likely that the lack of elevated AST in the other two species was due to the fact that they succumbed to capture much more quickly than the bull sharks so that there was not adequate time for AST to become elevated.

Alkaline phosphatase (ALP) is an enzyme originating mainly in the bone, liver, placenta, and, to a lesser extent, the kidneys of many vertebrates, and it is normally present in the blood in negligible concentrations (Price and Stevens 1982). Although age is an important determinant of ALP in mammals, there was no correlation with age (total length) in the bonnethead sharks, the only one of the three species with a range of age-classes represented. Serum ALP concentration is positively correlated with hepatic lesions in yellowfin tuna *Thunnus albacares* (Sandnes et al. 1988), but it normally does not increase as part of the stress response in fish (Bourke et al. 1987). The increases observed in the bonnethead and blacktip sharks are likely a product of internal damage caused by net capture and restraint, but an exact etiology remains to be determined.

Conclusions

The relatively simple process of gill-net capture and restraint of sharks leads to a complex set of responses that may involve exercise to exhaustion, stress, hypoxia, respiratory and/or metabolic acidosis and the resultant acidemia, as well as cellular damage. Although this study was not designed to separate out these various components, it has provided evidence of their occurrence. It is apparent that differences in the ways in which species respond may reduce or exacerbate mortality. Experimental evidence to separate out the different components is needed to understand their relative involvement in the mortality caused by the gill-net capture and restraint of sharks. However, it is clear that capture in gill nets affects individual sharks in major ways and thus that gill-net fisheries may impact shark populations. Whether these fisheries are directed at the sharks or the sharks are taken as bycatch, the physiological and behavioral impacts on individual animals can be catastrophic. It is hoped that this study will lead to other investigations examining the effects of catch-and-release fishing techniques in the recreational and commercial shark fisheries.

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