Transactions of the American Fisheries Society 134:1457–1470, 2005

DOI: 10.1577/T04-235.1

[Article]

Physiological Effects of Swim Bladder Overexpansion and Catastrophic Decompression on Red Snapper

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Abstract.—The commercial and recreational harvests of red snapper *Lutjanus campechanus* in the Gulf of Mexico have declined over the past five decades, prompting strict regulations. Release mortality associated with catastrophic decompression (CD) is a possible cause for the continuing decline, although to date no physiological data exist to support this assumption. Using a flow-through high-pressure chamber, subadult red snapper were acclimated to 101.2, 405.3, 608.0, and 1,215.9 kPa, simulating depths typical of their distribution (as deep as 200 m), and then decompressed at a rate of 10.1 kPa/s. Lateral and dorsal X-ray imaging in combination with necropsy showed that swim bladders expanded in a predictable manner. Ventral expansion into the caudal body cavity space occurred at lower pressures, whereas expansion into the cranial portion of the body cavity occurred at the highest pressure. Expansion patterns resulted in over 70 different overexpansion injuries, the most severe being to vital organs. Our results suggest a specific suite of clearly identifiable injuries associated with CD that increase in number and severity as retrieval depth increases. A more thorough understanding of catastrophic decompression syndrome will provide insight into the declining fishery and aid in developing effective physiology-based management strategies.

Commercial and recreational harvests of red snapper *Lutjanus campechanus* from the Gulf of Mexico account for 8 million pounds of production annually but have been plagued by a steady population decline over the past 25 years (Schirripa and Legault 1999). Although overfishing was initially suspected as the major cause of the population decrease (Goodyear 1995; NOAA 1997; Gregg and Fogt 2000), the implementation of increasingly stringent fishing regulations beginning in the early 1990s has failed to reverse this trend (Schirripa and Legault 1999). Consequently, biologists have begun to examine other factors that may play a role in the fishery decline. Shrimp trawl bycatch, for example, is believed to be a major cause of juvenile red snapper mortality (Moe 1963; Bradley and Bryan 1976; Gutherz and Pellegrin 1988; Nichols 1989; Schirippa and Legault 1999). Nichols (1989) estimated that at least 20 million juvenile red snapper were killed in Gulf of Mexico shrimp trawls in 1989 alone. Another damaging practice is capture and release of undersized red snapper (<40 cm total length [TL]) by both commercial and recreational anglers. Schirippa and Legault (1999) found that 52% of all red snapper caught by fishermen were below the legal size limit and, therefore, were released. While release mortality of undersized shallow-water game fish is probably low (Pelzman 1978; Crabtree et al. 1998; Kaimmer and Trumble 1998; Pope and Wilde 2004), deepwater species such as red snapper often suffer pressure-related injuries (Harden-Jones 1952; Bruesewitz and Coble 1993; Parrish and Moffitt 1993; Gitschlag and Renaud 1994; Wilson and Burns 1996) that may increase mortality rates of released fish.

Red snapper rapidly brought to the surface from deep water experience catastrophic decompression (CD), a phenomenon characterized by a sudden decrease in ambient pressure that causes swim bladder overexpansion and potential damage to associated internal organs (Schmidt-Nielson 1997). In severe cases, the decompression event can be lethal, resulting in high release mortality in the fishery. Fish suffering from CD usually exhibit characteristic symptoms, including exopthalmia (bulging eyes), external hemorrhaging, everted stomach, intestinal protrusions, and loss of equilibrium (Harden-Jones 1952; Bruesewitz and Coble 1993; Parrish and Moffitt 1993; Gitschlag and Renaud 1994; Wilson and Burns 1996). The consistent pattern of symptoms seen in fish brought up from high depths may be aptly described as catastrophic decompression syndrome (CDS). While severe CDS may be acutely lethal, less se-

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Received December 23, 2004; accepted July 20, 2005
Published online November 3, 2005

vere decompression events may also result in mortality. Tytler and Blaxter (1977) found that, upon rapid decompression, fish display positive buoyancy along with equilibrium loss and are often unable to return to the depth at which they were captured. Consequently, released fish are left to struggle on the surface where they are vulnerable to predators (Blaxter and Batty 1984; Keniry et al. 1996) and unfavorable temperatures (Keniry et al. 1996; Bettoli and Osborne 1998; FSG 1999).

Puncturing the swim bladder to vent excess gas (Keniry et al. 1996; FSG 1999; B. Jones, National Marine Fisheries Service, personal communication) or allowing fish sufficient time to equalize swim bladder pressures by slowing retrieval rates (Parrish and Moffitt 1993; Gitschlag and Renaud 1994; Keniry et al. 1996) may minimize or prevent CDS, but the efficacy of mitigation protocols is vigorously debated (B. Jones, personal communication). Wilson and Burns (1996) argued that vented fish have a higher survival rate relative to unvented fish; however, several other studies found that venting does not significantly enhance survival (Gotschall 1964; Bruesewitz and Coble 1993; Render and Wilson 1993, 1994, 1996) and may even result in higher release mortality as a result of inaccurate puncturing techniques that can cause infection or damage internal organs (Harden-Jones 1957; Parrish and Moffitt 1993; B. Jones, personal communication). Field mark and recapture studies designed to resolve this controversy have been hindered by poor return numbers (less than 2%; Render and Wilson 1994; Keniry et al. 1996) or uncertainties about capture depth (Gotschall 1964; Fable 1980; Gitschlag and Renaud 1994), and preliminary results may be spurious owing to high secondary stresses such as temperature shock and handling time (Bettoli and Osborne 1998). Slowing retrieval from capture depth or holding captured fish at an intermediate depth in an attempt to let fish equalize swim bladder pressure as they are brought to the surface is equally contentious (Wilson and Smith 1985; Haight 1989; Keniry et al. 1996). Limitations encountered in descriptive field-based studies, including uncontrollable outside variables (temperature or capture depth) or experimental artifacts (unnatural conditions, tagging, or handling stress), make it difficult to achieve a clear consensus as to the efficacy of CDS mitigation protocols. A better approach may be an extensive physiology-based CDS assessment that could be used as a predictive tool to assess red snapper responses under many field conditions (Matthews and Reavis 1990; Gitschlag and Renaud 1994; Render and Wilson 1994).

The steady decline in the Gulf of Mexico red snapper population, despite relatively low natural mortality (Schirripa and Legault 1999) and increasingly rigorous protection that includes season closures, bag limits, and minimum size limits, strongly suggests that the fate of the released red snapper is integral to the design of future management strategies (Gitschlag and Renaud 1994; Render and Wilson 1994). The purpose of our research is to assess the type, extent, and pattern of physiological and anatomical injury incurred by red snapper exposed to controlled CD from specific depths simulated in the laboratory. Laboratory-based assessment would be useful in uncoupling the effects of rapid pressure change from other confounding factors in the environment and could be helpful in developing effective regulation and management strategies to ameliorate release mortality rates for the Gulf of Mexico red snapper.

Methods

Red snapper were collected by hook and line between 15 April and 31 October 2000, 2001, and 2002 at depths of 30–60 m from reef sites southeast of Destin Pass in Okaloosa County, Florida (15.9 ± 6.66 km offshore). All captured fish were observed for postdecompression trauma and vented by puncturing the swim bladder 3–4 scales posterior to the pectoral fin (FSG 1999) with a sterile 18-gauge hypodermic needle (Bruesewitz and Coble 1993). Excess swim bladder gas was collected into a water-filled, inverted graduated cylinder with an 18-gauge hypodermic needle connected to a three-way stopcock. Fish were then measured (mm TL), tagged, and transferred to covered 150-L Rubbermaid containers filled with clean seawater that was circulated and aerated by submersible pumps (Rule Industries, Gloucester, Massachusetts; Model 1200). Capture temperatures were estimated from body core temperatures of three larger fish (see methodology of Cook and Crist 1979). Holding temperatures were maintained within ±2.0°C of capture temperature. Fifteen haphazardly selected red snapper (TL = 324.9 ± 8.10 mm [mean ± SD]; wet weight = 480.8 ± 36.95 g) were vented, measured, and necropsied immediately upon capture. Remaining fish were transported in a 380-L fiberglass container filled with clean, aerated seawater to the Marine Ecological–Physiology Laboratory at the University of West Florida.

Maintenance of red snapper.—Red snapper were treated prophylactically with 50.00 mg/L nitrofurazone, dipped in 0.30 mg/L $CuSO_4$ for 60 min, and quarantined for 5 d in 0.05 mg/L Dylox and 2.50 mg/L Marex to eliminate bacteria, trematode, and *Amylodinium* spp. infestation (Chitwood and Lichtenfels 1972; Toft and Edstrom 1980). Fish were held in 2,000-L biologically filtered tanks at densities no greater than 1 fish/50 L of water and allowed to recover from swim bladder puncture for at least 14 d before pressure trials (Bruesewitz and Coble 1993). Fish were maintained at 23.1 ± 0.86°C and held under a 12:12 light–dark photoperiod. Water quality parameters were monitored every other day to assure accepted limits of ammonia (0.1 ± 0.04 mg/L), nitrite (0.1 ± 0.02 mg/L), nitrate (34.4 ± 16.69 mg/L), pH (7.9–8.2), and salinity (32.2 ± 1.74 mg/L). Water changes (~33% of total volume) were performed every 2 weeks or as dictated by water quality parameters. Fish were fed squid, shrimp, or both daily until sated, but were not fed 24 h before or during experimental trials.

X-ray imaging and analyses.—Predecompression status of each fish was assessed roentgenologically before experimental trial at ambient pressure with a Faxitron X-ray machine (Hewlett-Packard Company, Palo Alto, California; Model 43855A) set at 70 kilovolt peak (kVp) with 5-s exposure (dorsal images) or at 65 kVp with 5-s exposure (lateral images; Tytler and Blaxter 1977) resolved on type M Structurix ReadyPak (Agfa-Gevaert Group, Mortsel, Belgium) X-ray film. The process was repeated for each fish after controlled decompression. Using the width, height, and length measures from lateral and dorsal aspect images, pre- and postdecompression swim bladder volume estimates were determined (Davenport 1999). Expansion patterns pre- and posttrial were used to evaluate the progression of CDS and were calculated according to changes in swim bladder volume in cranial and caudal cavities. Pelvic and anal fin insertion points were identified on each X-ray, and a solid vertical line placed at 10% of the distance between insertion points in the caudal direction represented the boundary between cranial and caudal body cavities. Body cavity space to the left of the solid line was referred to as cranial cavity and body cavity space to the right was referred to as caudal cavity.

Flow-through high-pressure chamber and equipment.—A 28-L steel flow-through high-pressure chamber (Figure 1) was used to simulate pressures fish would experience at selected depths. The chamber was pressurized with a Myers–APEX 220-V electric reciprocating pump (Pentair Pump Group, Ashland, Ohio) at a flow rate of 7 L/min, and pressure was regulated with a brass T-handle 0.00–21.09 kilogram-force per square centimeter pressure relief valve (McMaster-Carr Supply Company, Elmhurst, Illinois; Model 9763 K51). Water temperatures in the chamber were monitored with a Traceable digital thermometer (Control Company, Friendswood, Texas; Model 15-078-38) attached to a type-K thermocouple probe. Water circulated through the chamber was collected in a 160-L reservoir, where temperature and water quality were closely monitored and adjusted as necessary. Observations were made through two pressure-tolerant sight glass view ports.

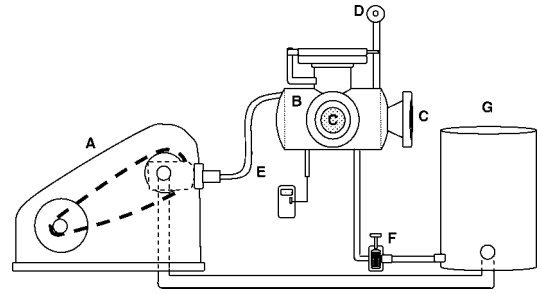

FIGURE 1.—Schematic drawing of the flow-through high-pressure chamber used to expose Gulf of Mexico red snapper to simulated depths to assess catastrophic decompression-related physiological and anatomical injury. Water is pumped from a Myers–APEX reciprocating pump (A) into the 28-L high-pressure chamber (B) equipped with sight glass view ports (C), pressure indicator gauge (D), digital thermometer (E), and pressure-regulating valve (F). Water exits the chamber into a 160-L recirculating water reservoir (G) before being pumped back into the chamber.

Acclimation to pressure and controlled decompression.—Individual red snapper were exposed to 101.3 (ambient), 405.3, 608.0, or 1,215.9 kPa, simulating depths of 0, 30, 50, or 110 m, respectively. Pressure treatments approximated the range in which red snapper are commonly captured by commercial and recreational anglers (Allen and Tashiro 1977; Klima 1977; Workman and Foster 1994; Goodyear 1995; Manooch et al. 1998). For each trial, a fish was placed into the pressure chamber and allowed to acclimate to its surroundings for 2–3 h before pressure was increased. Pressures were then increased within the chamber at 101.3 kPa/12 h, a rate slow enough to minimize stress and assure continuous swim bladder filling. Acclimation was determined to be complete when a fish achieved neutral buoyancy (Wilson and Smith

TABLE 1.—Frequency of external injuries (%) comprising lesions (L), exopthalmia in one eye (EX1), exopthalmia in both eyes (EX2), sustained stomach eversion (SE), brief stomach eversion (BE), sustained prolapse (SP), brief prolapse (BP), and hemorrhaging (H) observed in red snapper exposed to simulated depths to assess catastrophic decompression-related physiological and anatomical injury in control (101.2 kPa), low (405.3 kPa), medium (608.0 kPa), and high (1,215.9 kPa) laboratory groups and the only field group (405.3–608.0 kPa).

		Body surface		Eyes				Mouth				Anus			
Group	*n*	L	H	L	EX1	EX2	H	L	SE	BE	H	L	SP	BP	H
Control	13	30.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.7	0.0	0.0
Low	12	41.7	0.0	0.0	8.3	0.0	0.0	0.0	8.3	33.3	0.0	0.0	0.0	66.7	66.7
Medium	12	41.7	0.0	0.0	25.0	8.3	0.0	0.0	0.0	58.3	0.0	0.0	8.3	41.7	75.0
High	12	50.0	0.0	0.0	41.7	41.7	0.0	0.0	0.0	75.0	0.0	0.0	41.7	50.0	91.7
Field	15	26.7	0.0	0.0	26.7	0.0	0.0	0.0	0.0	12.5	0.0	0.0	75.0	25.0	62.5

1985), displaying minimal fin movement to maintain its position in the water column (Harden-Jones and Scholes 1985). Swim bladder filling rates were estimated from time needed to reach complete acclimation to a given simulated depth. After attaining neutral buoyancy, fish were held for an additional 12 h before undergoing forced decompression. Fish were decompressed at an average rate of 10.1 kPa/s, a rate simulating pressure change experienced during retrieval from depth during recreational fishing, which we determined as the average retrieval with typical recreational gear through personal observation. Following pressure release, fish were examined for trauma. X-ray imaging was repeated and then each fish's swim bladder was vented into the gas collection apparatus previously described. Fish were then sacrificed and a complete necropsy was performed.

Necropsy procedures.—Field and laboratory necropsy protocols were identical. First, the spinal cord was severed anterior to the first dorsal fin spine. Then, each fish was opened with a ventral midsaggital incision from the anus to the pectoral girdle. Gastrointestinal system components from anus to esophagus were carefully examined for injury in each fish with the aid of a binocular dissecting microscope if needed. Gastrointestinal system injuries were categorized as displacement, stretching, bruising, damage to peritoneal connections, hemorrhaging, hematomas, volvulus (a condition marked by twisting of the gut), intussusception (telescoping of the gut), and perforation. We identified gross morphological damage to the liver, gallbladder, spleen, heart, peritoneal connections, swim bladder, and gas gland and recorded evidence of holes, tears, stretching, bruising, hemorrhaging, or hematomas observed.

Statistical analyses.—Where one-way analysis of variance (ANOVA) determined differences between treatment groups, posthoc statistical relationships among individual mean values were made with a Student–Newman–Keuls (SNK) test. Injury frequencies were compared between treatment groups with a type III contingency test. Data are presented as mean $\pm$ SD, except grand means, which are presented as $\mu \pm$ SE. All statistical decisions are based on $\alpha = 0.05$.

Results

Of the 132 red snapper collected in the field captured between October 2001 and July 2003, over 80% showed external signs of CDS regardless of capture depth. The most common injuries observed were exopthalmia (14%), stomach eversion (19%), prolapse (59%), external lesions (3%), bleeding from the anus (7%), and flared opercula (13.0%). Approximately 23% of all fish collected and vented onboard the fishing vessel died within 1 h after the procedure; an additional 10% of captured red snapper died during transport or within 12 h after returning to the laboratory. These counts differ only slightly from the proportions of injuries observed in the 15 red snapper necropsied in the field (Table 1). External lesions (26.7%), exopthalmia in one eye (26.7%), brief stomach eversion (12.5%), brief anal prolapse (25%), sustained anal prolapse (75%), and bleeding from the anus (62.5%) were the most common injuries observed in these 15 fish. Overambient swim bladder gas volume for fish vented in the field averaged 52.0 $\pm$ 20.86 mL (Table 2). Differing capture depth (29.6 $\pm$ 6.74 m) and body size (TL = 324.9 $\pm$ 8.10 mm; wet weight = 401.3 $\pm$ 36.95 g) probably accounts for variations in overambient (vented) swim bladder volume (Table 2). External symptoms in laboratory-decompressed red snapper (Figure 2), such as exopthalmia, stomach eversion, and sustained prolapse, paralleled injuries documented during field necropsies. The relative frequency of external injuries for the 15 fish necropsied in the field tallied only slightly higher than frequencies for the low-pressure laboratory group

TABLE 2.—Swim bladder vented volume and size relationships for Gulf of Mexico red snapper in field groups and in groups exposed to simulated depths to assess catastrophic decompression-related physiological and anatomical injury. Mean values (±SD) are listed for control (101.2 kPa), low (405.3 kPa), medium (608.0 kPa), and high (1,215.9 kPa) laboratory groups and the only field group (405.3–608.0 kPa). Values with different letters indicate significant differences between mean values within that row (one-way ANOVA and Student–Newman–Keuls test; $\alpha = 0.05$; na = not available).

	Laboratory groups														
	Control			Low			Medium			High			Field group		
Measurement	$\bar{x}$	SD	*n*	$\bar{x}$	SD	*n*	$\bar{x}$	SD	*n*	$\bar{x}$	SD	*n*	$\bar{x}$	SD	*n*
Vented volume (mL)		na		29.3 z	9.48	12	30.4 z	9.59	12	41.0 y	9.48	12	52.0	20.86	15
Total geometric volume, mL (before)	20.3 z	5.10	10	26.2 z	10.70	8	21.3 z	7.21	7	22.8 z	7.94	9		na	
Total geometric volume, mL (after)	21.0 z	9.14	10	50.6 y	14.86	8	49.4 y	22.20	7	65.4 x	13.19	9		na	
% Available total cavity (before)	57.6 z	6.42	11	54.7 z	8.10	10	58.7 z	5.26	9	59.3 z	2.68	9		na	
% Available total cavity (after)	50.3 z	8.93	11	29.3 y	8.24	10	25.2 y	9.20	9	19.1 x	2.76	9		na	
% Available caudal cavity (before)	44.7 z	6.77	11	46.4 z	9.29	10	49.1 z	6.56	9	51.3 z	5.26	9		na	
% Available caudal cavity (after)	36.5 z	12.49	11	13.5 y	8.07	10	13.6 y	9.66	9	8.2 x	8.16	9		na	
% Available cranial cavity (before)	72.4 z	3.50	11	71.5 z	5.90	10	71.7 z	5.54	9	70.7 z	7.85	9		na	
% Available cranial cavity (after)	69.6 z	5.78	11	47.7 y	10.50	10	45.8 y	16.29	9	41.2 x	18.96	9		na	

(Figure 2). A type III contingency analysis showed that injury frequency in laboratory fish became higher with increasing pressure, the highest percentage of external injury occurring in the high-pressure laboratory group ($P < 0.05$; Figure 2).

Changes in Body Cavity Dimensions During CD

Average time to acclimation (68.1 ± 2.78, 98.1 ± 4.40, 174.3 ± 2.22 h for low-, medium-, and high-pressure treatment groups, respectively) and vented volumes (Table 2) demonstrated a positive relationship with increasing pressure. Acclimation times for all groups were statistically distinct (one-way ANOVA: $P < 0.01$; SNK at $\alpha = 0.05$). Control fish showed no significant overinflation volume; however, volume for low-, medium-, and high-pressure treatment groups averaged 29.3 ± 9.48, 30.4 ± 9.59, and 41.0 ± 9.48 mL, respectively (Table 2); red snapper in the high-pressure treatment group had significantly higher vented volumes than fish from low- and medium-pressure treatment groups (one-way ANOVA: $P < 0.01$; SNK at $\alpha = 0.05$). Vented volumes of fish from low- and medium-pressure treatment groups did not differ from one another. Similarly, mean calculated geometric swim bladder volumes from X-rays taken before and after CD were increased in postexperimental values in all treatments except the control (Table 2; Figure 3). Swim bladder volumes in low-, medium-, and high-pressure treatment groups showed pre- to postexperimental increases of 51.3 ± 14.64%, 55.9 ± 13.55%, and 71.5 ± 5.94%, respectively (Figure 3). Swim bladder volumes from low- and medium-pressure treatment groups differed by less than 5% and formed a statistically homogeneous group; however, control and high-pressure volumes were each statistically distinct from all other experimental groups (one-way ANOVA: $P < 0.01$; SNK at $\alpha = 0.05$; Table 2).

Increases in swim bladder volumes resulted in progressive reductions in body cavity space available to other internal organs (Table 2). Preexperimental (i.e., ambient pressure at 101.30 kPa) X-

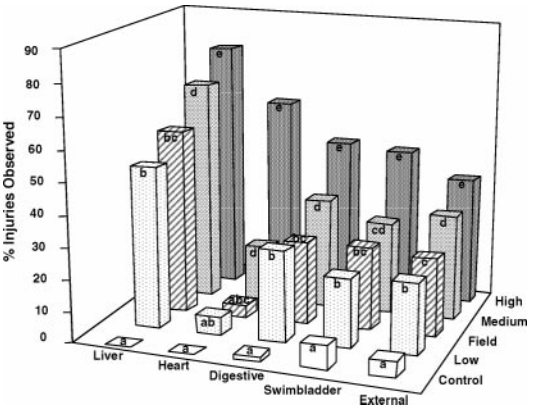

FIGURE 2.—Frequency histogram of injuries occurring within five major organ systems of Gulf of Mexico red snapper in field and control (101.3 kPa), low (405.3 kPa), medium (608.0 kPa), and high (1,215.9 kPa) laboratory groups exposed to laboratory-controlled simulated depths to assess catastrophic decompression-related physiological and anatomical injury. Values with different letters indicate significant differences between field, control, and low-, medium-, and high-pressure groups within each organ system (one-way ANOVA and Student–Newman–Keuls test; $\alpha = 0.05$).

rays of all fish found nearly 60% of the body cavity available to internal organs other than the swim bladder and no significant difference was observed between treatment groups (one-way ANOVA: $P = 0.510$). After decompression, however, fish in low-, medium-, and high-pressure treatment groups exhibited respective losses of 25.4 ± 13.97%, 36.7 ± 11.70%, and 40.6 ± 5.23% of body cavity space. Low- and medium- as well as medium- and high-pressure treatment group means formed statistically indistinguishable groups (one-way ANOVA: $P < 0.01$; SNK at $\alpha = 0.05$).

Interestingly, X-ray imaging also revealed that body cavity loss was not uniform; the swim bladder first impinged the caudal body cavity space and expanded into the cranial portion where it became progressively more pronounced as decompression depth increased (Figure 3). Of the total area available in the caudal body cavity space alone, 47.9 ± 1.45% is unoccupied by the swim bladder and available to other internal organs (Table 2). Preexperiment X-rays found no significant differences in this value between group means (one-way ANOVA: $P = 0.360$). After CD, available caudal cavity space fell to 13.5 ± 8.07%, 13.6 ± 9.66%, and 8.2 ± 8.16% in the low-, medium-, and high-pressure treatments groups, respectively (Table 2). Caudal body cavity means for control and high-pressure treatment groups were each individually distinct from all other groups; however, low- and medium-pressure treatment groups were not significantly different from one another (one-way ANOVA: $P < 0.010$; SNK at $\alpha = 0.05$). In the cranial cavity space, approximately 70.00% of the area remained unoccupied by the swim bladder in nondecompressed fish from all treatment groups (one-way ANOVA: $P = 0.970$). After decompression, available space in the cranial region was reduced to 47.7 ± 10.50%, 45.8 ± 16.29%, and 41.2 ± 18.96% in low-, medium-, and high-pressure treatment groups, respectively (Table 2). Control and low-pressure treatment groups were not significantly different from one another but were significantly different from both medium- and high-pressure treatment groups, which were also individually distinct (one-way ANOVA: $P < 0.01$; SNK at $\alpha = 0.05$; Table 2).

Effects of CD on Internal Organs

Digestive system injuries for red snapper decompressed experimentally increased significantly with treatment pressures and frequency of injury differed significantly between all laboratory- and field-decompressed fish (type III contingency analysis: $P < 0.05$; Figure 2). Injuries proximal to the anus were associated with low- and medium-pressure laboratory groups, where injuries to the lower-intestine portion of the digestive system were strongly correlated with low-pressure treatment (Table 3). As decompression pressures increased, however, gastrointestinal tract injuries progressed in the rostral direction from anus to esophagus; these injuries were more closely associated with the high-pressure laboratory group. Consequently, fish in the high-pressure treatment group not only sustained injuries common to lower-pressure treatment groups but also exhibited a new suite of digestive system injuries corresponding to areas closer to the esophagus (Table 3). Not surprisingly, injuries to the digestive organs were more obvious and more frequent in the high-pressure treatment group (Figure 2).

Hemorrhaging or hematomas of the liver were evident in all fish exposed to above-ambient pressures (Table 4). Liver injury frequency was significantly distinct in all laboratory groups (type III contingency analysis: $P < 0.05$; Figure 2), but the low-pressure laboratory and field groups were not significantly different from one another (type III contingency analysis: $P > 0.05$). Hemorrhaging, hematomas, and bruising of the liver were most

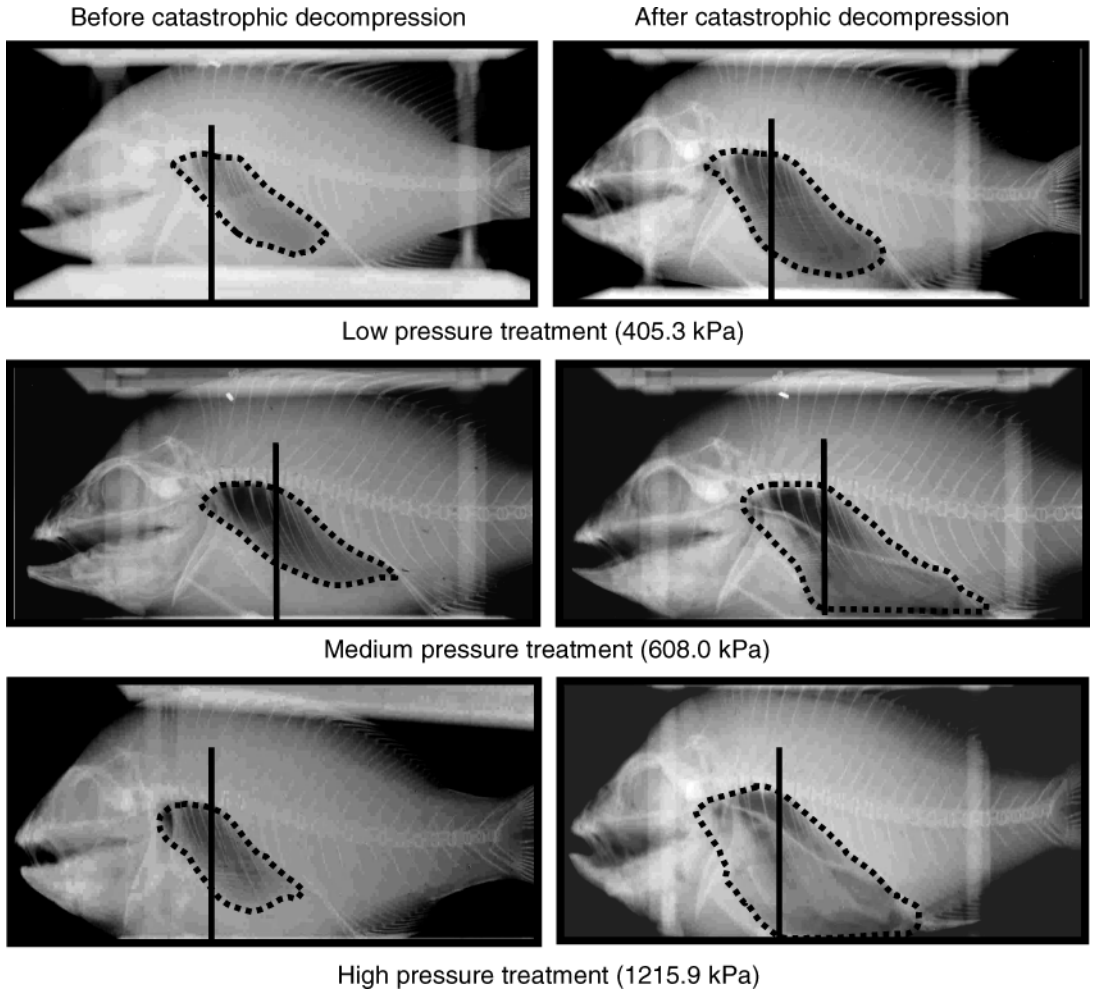

FIGURE 3.—Representative lateral aspect X-ray images of Gulf of Mexico red snapper before and after catastrophic decompression from low-, medium-, and high-pressure laboratory groups. Dashed lines represent swim bladder boundaries and vertical lines demarcate the cranial–caudal body cavity boundary.

commonly observed in the medium- and high-pressure treatment groups.

Frequency of cardiac injury (Table 4) was greatest in fish decompressed from higher pressure. Significantly fewer injuries to the heart were seen in the medium- and low-pressure treatment groups, which formed a statistically homogenous group (type III contingency analysis: $P < 0.05$; Figure 2). Likewise, the incidence of cardiac injury in control-, low-pressure, and field-decompressed fish were statistically indistinguishable (type III contingency analysis: $P < 0.05$). Cardiac injuries occurred in less than 1.0% of control or low-pressure treatment groups; however, symptoms of severe cardiac damage were common in medium- and high-pressure treatment groups. Hematomas and loss of pericardial integrity or ruptures in the bulbous arteriosis accounted for the majority of cardiac injuries observed. Hematomas, while occurring infrequently, were only observed in fish decompressed in the high-pressure treatment group.

Injuries to the swim bladder were also most numerous and severe in the high-pressure treatment group (Table 4). Frequency of swim bladder injury in control and high-pressure-treated fish were statistically distinct, whereas the remaining treatments and field-collected fish formed a homogenous group (type III contingency analysis: $P < 0.05$; Figure 2). Less than 1% of control fish displayed swim bladder abnormalities. Swim bladder wall injuries sustained by fish in the high-pressure

TABLE 3.—Frequency of gastrointestinal injuries comprising stretching (ST), misshaping (M), bruising (B), hematomas (HA), hemorrhaging (H), torn peritoneum (TP), volvulus (V), intussusception (INT), prolapse (PRO), invagination (INV), and perforation (PERF) observed in Gulf of Mexico red snapper in the field (n = 15) and at simulated depths (n = 13, 12, 12, and 12) to assess catastrophic decompression-related physiological and anatomical injury. Injuries were documented only if they occurred. See Table 2 for descriptions of treatment groups.

Region affected and injury	Injury frequency (%)				
	Control	Low	Medium	High	Field
Colon					
ST	0.0	50.0	58.3	75.0	100.0
M	15.4	25.0	58.3	83.3	100.0
B	0.0	33.3	50.0	83.3	100.0
H	0.0	75.0	66.7	83.3	100.0
V	0.0	8.3	8.3	41.7	16.7
PRO	0.0	33.3	58.3	75.0	83.3
Lower intestine					
ST	7.7	83.3	75.0	100.0	75.0
M	0.0	16.7	8.3	33.3	66.7
H	0.0	50.0	66.7	83.3	75.0
TP	0.0	66.7	66.7	66.7	75.0
V	0.0	75.0	33.3	75.0	66.7
INT	0.0	8.3	8.3	25.0	33.3
Upper intestine					
ST	0.0	25.0	33.3	75.0	0.0
M	0.0	8.3	33.3	33.3	0.0
H	0.0	66.7	75.0	75.0	33.3
TP	0.0	16.7	16.7	16.7	0.0
V	0.0	25.0	41.7	66.7	12.5
INT	0.0	16.7	25.0	41.7	12.5
Pyloric caeca					
ST	0.0	25.0	66.7	75.0	12.5
M	0.0	8.3	16.7	33.3	12.5
B	0.0	18.2	9.1	0.0	0.0
H	0.0	16.7	41.7	83.3	12.5
V	0.0	50.0	25.0	33.3	0.0
Cardiac stomach					
M	7.7	50.0	58.3	91.7	25.0
H	0.0	25.0	50.0	66.7	12.5
TP	0.0	8.3	8.3	50.0	0.0
V	0.0	0.0	0.0	16.7	0.0
INV	7.7	33.3	58.3	91.7	25.0
PERF	0.0	0.0	8.3	33.3	0.0
Pyloric stomach					
ST	0.0	0.0	25.0	83.3	0.0
M	0.0	25.0	66.7	75.0	12.5
H	0.0	25.0	33.3	75.0	12.5
V	0.0	33.3	41.7	83.3	25.0
INV	7.7	25.0	0.0	16.7	0.0
PERF	0.0	8.3	16.7	16.7	0.0
Esophagus					
ST	0.0	25.0	58.3	91.7	12.5
HA	0.0	16.7	33.3	66.7	0.0
H	0.0	41.7	58.3	83.3	12.5

TABLE 4.—Frequency of internal injuries comprising hematomas (HA), hemorrhaging (H), torn peritoneum (TP), pericardial damage (PD), volvulus (V), lesions (L), stretching (ST), one hole (HOLE), multiple holes (HOLES), and bulbous arteriosis damage (BAD) observed in Gulf of Mexico red snapper in the field (n = 15) and at simulated depths (n = 13, 12, 12, and 12) to assess catastrophic decompression-related physiological and anatomical injury. Injuries were documented only if they occurred. See Table 2 for descriptions of treatment groups.

Region affected and injury	Injury frequency (%)				
	Control	Low	Medium	High	Field
Gallbladder					
H	0.0	0.0	33.7	16.7	0.0
V	15.4	25.0	58.3	91.7	83.3
Liver					
HA	0.0	33.3	66.7	83.3	50.0
H	0.0	75.0	83.3	91.7	75.0
Spleen					
L	0.0	0.0	41.7	91.7	0.0
Heart					
H	0.0	25.0	50.0	50.0	0.0
PD	0.0	50.0	25.0	33.3	0.0
ST	0.0	8.7	33.3	66.7	12.5
BAD	0.0	0.0	8.3	25.0	0.0
Swim bladder					
H	0.0	25.0	50.0	50.0	0.0
TP	0.0	16.7	66.7	83.3	50.0
L	0.0	25.0	25.0	50.0	25.0
ST	7.7	25.0	33.3	66.7	12.5
HOLE	0.0	75.0	58.3	83.3	87.5
HOLES	0.0	0.0	16.7	33.3	12.2
Gas gland					
H	0.0	33.3	58.3	83.3	0.0

treatment group, including left-ventral tears and multisite ruptures, were more severe and numerous than seen in other treatment groups. Overall, 80% of injuries to the swim bladder upon CD were observed in the high-pressure treatment group. Damage to the peritoneum, stretching, right-ventral tears, and damage to the gas gland were observed in both high- and medium-pressure treatment groups. Interestingly, symptoms of bleeding in the vicinity of the swim bladder corresponded with the medium-pressure treatment group. Fish in the medium- and low-pressure treatment groups displayed the fewest number of injuries, and no injuries were limited strictly to the low-pressure treatment group.

Patterns of Organ Damage Relative to Pressure Group

Gross anatomical injuries resulting from CD were readily observed in nearly all internal organ

systems, and external injuries were common in all above-ambient pressure groups as well. Internal injuries showed clear patterns of progression and severity that were directly related to decompression pressure. Digestive system injuries were found in most fish; however, fish from different pressure treatments presented with a dose–response injury pattern such that injuries increased in numbers, severity, type, and location as pressures increased. In addition, secondary effects, such as intussusception, volvulus, and displacement, became more frequent at high pressures. Frequency of liver injuries showed a similar graded response in that the suite of injuries remained constant and severity gradually increased with pressure. Relatively severe swim bladder decompression injuries abruptly appeared in fish as pressure reached 608 kPa. At or above this pressure level, minor injuries rapidly shifted toward major insult. Similar threshold responses were seen in the heart, where nearly all observed cardiac injuries occurred in fish acclimated to high-pressure. When evaluated concurrently, internal anatomical injuries to red snapper progressed in a distinctive pattern, increasing in total number and type either incrementally (i.e., dose–response) or acutely (i.e., threshold) and moving rostrally with increasing decompression pressure.

Discussion

Depending on decompression depth, expansion of the red snapper swim bladder during CD is a process that results in two different categories of injury. Displacement injuries are the first to occur as the expanding swim bladder increases in volume and begins to contact internal organs and systems. Continued decompression results in more severe compaction injuries as the swim bladder becomes confined by the body wall and pressure on organs increases dramatically (Davenport 1999). It seems likely that injuries sustained during decompression would have variable effects on fish. The most severe injuries (e.g., puncture or rupture of vital organs) may prove acutely lethal to fish. However, less severe injuries (e.g., marked compaction of organ systems) may have lingering chronic lethal effects. Even minor insult, such as displacement or bruising, could potentially inhibit performance and, ultimately, survival. The progressive decrease in available body cavity space seen in red snapper exposed to decompression does not affect all organ systems simultaneously. Necropsy data and X-ray imaging reveal a distinctive pattern of swim bladder expansion and related injury, beginning with ventral expansion in the caudal direction and progressing into the cranial regions of the body cavity. Normally, the swim bladder occupies nearly half of the caudal cavity; however, following CD, significant portions of space available to organs (up to 45%) can be lost to the expanding swim bladder, even in low-pressure treatment groups. In contrast, the swim bladder occupies only a small portion of the cranial region of the body cavity at ambient pressure. While up to 51% of the available space can be lost after extreme decompression events, damage to cranially positioned organs only begin to manifest themselves in fish from high-pressure treatment groups. Clearly, red snapper captured from relatively deeper waters can be expected to show increasing acute mortality as body cavity space decreases, internal pressures rise, and displacement and compaction injuries become more frequent and severe.

Symptomatic presentation of CDS is similar across species (Parrish and Moffitt 1993; Gitschlag and Renaud 1994; Keniry et al. 1996); however, the relationship between depth and external symptoms is weak and may not accurately reflect the extent of internal injury. We consistently observed bleeding, contusions, prolapse, eversion, or exopthalmia in all laboratory and field red snapper. Other studies also document typical CDS symptoms, including stomach eversion (Harden-Jones 1952; Bruesewitz and Coble 1993; Parrish and Moffitt 1993; Gitschlag and Renaud 1994; Keniry et al. 1996; Davenport 1999), prolapse (Bruesewitz and Coble 1993; Gitschlag and Renaud 1994; Keniry et al. 1996; Davenport 1999), distortion of scales and flesh (Parrish and Moffitt 1993), and exopthalmia (Bruesewitz and Coble 1993; Parrish and Moffitt 1993; Gitschlag and Renaud 1994). It is noteworthy that external CDS symptoms are present in Hawaiian snapper *Pristipomoides filamentosus* (Parrish and Moffitt 1993), burbot *Lota lota* (Bruesewitz and Coble 1993), red snapper (Gitschlag and Renaud 1994), and Atlantic cod *Gadus morhua* (Harden-Jones and Scholes 1985) from depths as shallow as 10 m (burbot) to over 100 m (Hawaiian snapper). The consistent presence of similar external injuries in a variety of species argues that CD has similar effects on most (if not all) fish possessing swim bladders, even when they are retrieved from relatively shallow depths. While injuries in red snapper were more frequent after CD from higher pressure, all fish in noncontrol pressure groups displayed one or more of the external symptoms, suggesting that external CDS indicators in red snapper are not closely linked to

capture depth. It is unclear what, if any, effect external injury may have on red snapper survival; however, bleeding and contusions may be avenues for infection, prolapse and eversion may indicate more extensive internal injury, and exopthalmia may promote vision problems. In the absence of accurate external CDS predictors and the ambiguous nature of external insult, the only reliable way to assess the magnitude of CD damage in red snapper is to inspect the type and extent of internal injuries directly by necropsy.

Gastrointestinal Tract and Gallbladder

Injuries to the intestine and gallbladder were ubiquitous, no doubt because they occur in the caudal region of the body cavity where the expanding swim bladder first displaces and then compacts internal organs. The caudal region is the first to be occupied by the swim bladder and contains portions of the colon and large intestine that are easily displaced, resulting in injuries to ligaments and connecting peritoneal membranes (Sood et al. 2000). Stretching or tearing of membranes may have chronic repercussions, including intussusception and volvulus. The former occurs as the intestine telescopes upon itself, leading to inflammation, prolapse, decreased blood flow to the intestines, or even gangrene (Argyle 1986; Conners et al. 1999). The latter is a twisting of the gut, which can result in dysphagia, digestion problems, gut perforations, peritonitis, and sepsis (Godshall et al. 1999; Shivanand et al. 2003). In addition to conspicuous severe injuries, mild gastrointestinal damage can have long-term effects that hinder fish performance and survival. For example, 29 previously decompressed fish captured for this study stopped eating after weeks of normal feeding behavior and apparently died from starvation. Upon necropsy, intussusception and volvulus were apparent in 25 of these fish. It is probable that fish released by anglers succumb to similar latent effects of CD. These types of injuries would not be immediately evident and may take weeks or even months to appear. As a result, previous studies evaluating short-term survival of red snapper (Burns and Restrepo 2002) may greatly overestimate survival rates of released fish. Furthermore, latent effects of CDS may help explain low recapture numbers in mark and recapture studies.

Liver, Spleen, and Heart

Our data indicate that potentially lethal decompression injuries to the liver, spleen, and heart occur in red snapper retrieved from deeper reefs. Injuries in the cranial region of the body cavity were always observed in fish from the high-pressure treatment group (1,215.9 kPa), where they occurred in conjunction with severe compaction damage to the caudal cavity organ systems. With the exception of stomach eversion (a displacement injury), damages in the cranial region were primarily compaction injuries. The liver occupies the site where swim bladder expansion shifts from caudal to cranial displacement and injuries become more severe. Consequently, damage to the liver was limited to bruising and hematomas, conditions somewhat less severe than the lacerations and active bleeding observed in the spleen and heart. Untreated splenic laceration in vertebrates results in hemodynamic instability and hypotension from the hemorrhaging, peritonitis, and septic conditions that are acutely lethal (Argyle 1986; Kuehnert 1993). Likewise, active bleeding or structural damage to the heart from swim bladder compaction can be immediately fatal (Nehoda et al. 2001). Red snapper suffering from splenic or cardiac involvement after CD probably experience similar consequences, which, if extrapolated to the field, would argue that release mortality might be as high as 90% for fish retrieved from 110 m or deeper.

Swim Bladder

Unlike other organ systems that may exhibit displacement or compaction injury, swim bladder damage is always a displacement phenomenon. Most swim bladder damage occurred in fish from medium- and high-pressure treatment groups and included holes or tears in the swim bladder wall and damage to arterial connections, the gas gland, or the rete mirabile. Tears in the swim bladder wall were most frequently observed in the highest-pressure treatment group. Ruptured swim bladders are known to be lethal to deep-sea Pacific grenadier fish *Coryphaenoides acrolepis* (Wilson and Smith 1985) and European perch *Perca fluviatilis* (Harden-Jones 1952). Damage to red snapper gas glands or retia was also more common at higher pressures. Unless or until the damage is repaired, gas secretion into the swim bladder will be disrupted, resulting in an inability to precisely control buoyancy. Lack of buoyancy control may, in turn, decrease maneuverability, making red snapper more susceptible to predation and less efficient at prey capture (Gitschlag and Renaud 1994). Subtler swim bladder injury may include damage to mechanoreceptors located in the swim bladder membrane wall that may affect pressure sensitivity of

released fish (Tytler and Blaxter 1977; Gee 1981). Therefore, red snapper taken from depths greater than 50 m may experience long-term impaired performance and continued risk of mortality from swim bladder injuries.

Mitigation Strategies

Allowing fish to equalize swim bladder pressures from depth by slowing retrieval times has been suggested as one possible way to mitigate CDS. Parrish and Moffitt (1993) had 100% survival in Hawaiian snapper retrieved from 65 to 100 m if fish were only retrieved to 30 m for the tagging procedure. Numerous factors determine the rate at which a fish is able to compensate for buoyancy, including species (Alexander 1993), size (Gee 1977), temperature, and pressure (Overfield and Kylstra 1971; Harden-Jones and Scholes 1985). Alexander's (1966) observations of deepwater fishes that make large, vertical migrations led him to conclude that, even under the best conditions, fish will require between 4 and 48 h to make necessary adjustments to swim bladder volume. Slow secretion and resorption rates have since been determined for species such as Atlantic cod (1.0 m/h; Harden-Jones and Scholes 1985), deep lake polystigma cichlid *Haplochromis polystigma* and Livingston's cichlid *H. livingstonii*, (0.21 m/h; Ribbink and Hill 1979), bluegill *Lepomis macrochirus*, and Mozambique tilapia *Oreochromis mossambicus* (1.0 m/h; Alexander 1972), and it is now accepted that buoyancy regulation probably does not exceed rates greater than 2.5 m/h (Wittenberg and Wittenberg 1974). Red snapper secretion rates in our study (mean = 0.52 m/h) were similar to rates quantified for other species. While secretion is fundamentally different from resorption, it is unlikely that red snapper can resorb swim bladder gas fast enough so that slow retrieval would be a practical mitigation strategy for most fishing protocols (also see Wilson and Smith 1985; Haight 1989; Keniry et al. 1996).

Venting has been promoted as another way to mitigate release mortality in red snapper (Keniry et al. 1996; FSG 1999), but the efficacy of this technique is questionable. Indeed, venting allows fish to return to depth without having to overcome resistance of an overexpanded swim bladder (FSG 1999), but it is unclear as to the number of fish that actually survive. Few field-based studies have directly assessed the effects of venting and those that have are hampered by low return rates (less than 2%; Keniry et al. 1996), variations in capture depth (Render and Wilson 1996), and species and size differences (Bruesewitz and Coble 1993; Parrish and Moffitt 1993; Wilson and Burns 1996). As a result, conclusions are often in direct conflict and controversial. While a few researchers and organizations endorse venting (Keniry et al. 1996; FSG 1999), the technique fails to ameliorate internal injuries associated with the decompression event (Harden-Jones 1957). Venting requires increased handling time, knowledge of internal anatomy, and repositioning of everted internal organs (Harden-Jones 1957; Keniry et al. 1996; B. Jones, personal communication; B. LaRoche, Virginia Department of Game and Inland Fisheries, personal communication). Even with knowledge of red snapper internal anatomy, accepted venting procedures, and sterile equipment, survival for vented fish in our study was less than 75%. Our data did not speak to the survivability of fish subjected to the venting procedure, but it was clear the procedure did not change any of the injuries that were observed. In cases where fish had been vented previously to our capturing them, there was evidence of scarring on the swim bladder and puncture wounds, both of which may persist well beyond 2 weeks (personal observation) and can cause potentially fatal infections (B. Jones, personal communication).

While mitigation protocols such as slow retrieval rates and venting techniques are largely ineffectual, releasing fish that have not been vented is equally untenable. An overexpanded swim bladder leaves fish prone to higher temperatures that may prove lethal (Bettoli and Osborne 1998; B. LaRoche, personal communication). In addition, fish may be vulnerable to echolocating predators because the large swim bladder enhances returning echoes (Blaxter and Batty 1984). In the field, it was not uncommon to see bottlenose *Tursiops truncates* and Atlantic spotted dolphins *Stenella frontalis* preying on unvented red snapper left floating on the surface. It is clear that both vented and unvented red snapper suffer high release mortality and that slow retrieval rates are not feasible. In light of the ineffective mitigation protocols, it follows that reducing CDS mortality in red snapper will lie with management strategies rather than on mitigation-based techniques.

High mortality of released red snapper by anglers, coupled with the lack of effective mitigation strategies, suggest that a new approach to managing the declining fishery is needed. Our data demonstrate that physiological and anatomical effects of CD are more widespread than previously believed and that released fish that do not die im-

mediately may later succumb to latent effects of CDS. While many injuries may not be immediately lethal, the fact that they often occur in conjunction with more severe damage is a point that should be carefully considered by fisheries managers. The most common suggestion for ameliorating release mortality is to restrict fishing to shallower depths or modify regulations so that fewer fish are released when survival is unlikely (B. Jones, personal communication). Burns and Restrepo (2002) suggested that fishing should be limited to depths shallower than 50 m. However, our data as well as those collected on other game species clearly demonstrate that both acute and chronic lethal injuries associated with CDS occur at depths much shallower than 50 m. In addition, many of these injuries manifest themselves through longer periods than previously suspected, often in the absence of immediate, obvious signs of injury. Even assuming an optimistic survival rate for vented fish of 50%, it becomes clear that many fish will die upon release in addition to those that die from harvesting. A better approach may be to manage the fishery based solely on bag limits (i.e., every fish retrieved is kept, regardless of size, until the bag limit is met) rather than in conjunction with minimum-size limits. This strategy would, in theory, eliminate release of fish unlikely to survive. Red snapper is the most commercially and recreationally sought after species in the Gulf of Mexico, so the use of physiological and anatomical data to develop sustainable management practices may make it possible to eliminate both ineffective mitigative practices as well as an important source of population mortality.

Acknowledgments

We gratefully acknowledge statisticians Dr. R. Amin and H. Bears, Capt. J. Westbrook and the crew of the *New Florida Girl*, Dr. J. Sparks and the mechanical engineers at Systems Engineering Research Facilities, Cantonment, Florida, and D. Moss and staff at Claude Peteet Mariculture. We also thank M. Reaid, B. Hohmann, A. Jimenez, S. Hanna, M. Cook, and N. Fangue for technical contributions, C. J. Brauner, University of British Columbia, for financial support to J. L. Rummer during the writing portion of this project, and finally, two anonymous referees for their helpful suggestions. This project was executed under Florida state permits 02R-523 and 01S-523 and a Federal Letter of Agreement under 50 CFR 600.10, which made us exempt from 50 CFR 622.

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