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Oxygen transport and cardiovascular responses in skipjack tuna *(Katsuwonus pelamis)* and yellowfin tuna *(Thunnus albacares)* exposed to acute hypoxia

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Summary. Responses to acute hypoxia were measured in skipjack tuna *(Katsuwonus pelamis)* and yellowfin tuna *(Thunnus albacares)* ($\approx$1–3 kg body weight). Fish were prevented from making swimming movements by a spinal injection of lidocaine and were placed in front of a seawater delivery pipe to provide ram ventilation of the gills. Fish could set their own ventilation volumes by adjusting mouth gape. Heart rate, dorsal and ventral aortic blood pressures, and cardiac output were continuously monitored during normoxia (inhalant water ($PO_2 > 150$ mmHg) and three levels of hypoxia (inhalant water $PO_2 \approx 130$, 90, and 50 mmHg). Water and blood samples were taken for oxygen measurements in fluids afferent and efferent to the gills. From these data, various measures of the effectiveness of oxygen transfer, and branchial and systemic vascular resistance were calculated. Despite high ventilation volumes (4–7 l · min^{-1} · kg^{-1}), tunas extract approximately 50% of the oxygen from the inhalant water, in part because high cardiac outputs (115–132 ml · min^{-1} · kg^{-1}) result in ventilation/perfusion conductance ratios (0.75–1.1) close to the theoretically ideal value of 1.0. Therefore, tunas have oxygen transfer factors (ml O_2 · min^{-1} · mmHg^{-1} · kg^{-1}) that are 10–50 times greater than those of other fishes. The efficiency of oxygen transfer from water in tunas ($\approx$65%) matches that measured in teleosts with ventilation volumes an order of magnitude lower. The high oxygen transfer factors of tunas are made possible, in part, by a large gill surface area; however, this appears to carry a considerable osmoregulatory cost as the metabolic rate of gills may account for up 70% of the total metabolism in spinally blocked (i.e., non-swimming) fish. During hypoxia, skipjack and yellowfin tunas show a decrease in heart rate and increase in ventilation volume, as do other teleosts. However, in tunas hypoxic bradycardia is not accompanied by equivalent increases in stroke volume, and cardiac output falls as HR decreases. In both tuna species, oxygen consumption eventually must be maintained by drawing on substantial venous oxygen reserves. This occurs at a higher inhalant water PO_2 (between 130 and 90 mmHg) in skipjack tuna than in yellowfin tuna (between 90 and 50 mmHg). The need to draw on venous oxygen reserves would make it difficult to meet the oxygen demand of increasing swimming speed, which is a common response to hypoxia in both species. Because yellowfin tuna can maintain oxygen consumption at a seawater oxygen tension of 90 mmHg without drawing on venous oxygen reserves, they could probably survive for extended periods at this level of hypoxia.

Key words: Hypoxia – Cardiovascular – Oxygen transport – Skipjack tuna *Katsuwonus pelamis* – Yellowfin tuna, *Thunnus albacares*

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Abbreviations: BP_{da}, BP_{va} dorsal, ventral aortic blood pressure; C_aO_2, C_vO_2 oxygen content of arterial, venous blood; DO_2 diffusion capacity; E_b, E_w effectiveness of O_2 uptake by blood, and from water, respectively; Hct hematocrit; HR heart rate; PCO_2 carbon dioxide tension; P_aCO_2, P_vCO_2 carbon dioxide tension of arterial and venous blood, respectively; PO_2 oxygen tension; P_aO_2, P_vO_2, P_iO_2, P_eO_2 oxygen tension of arterial blood, venous blood, and inspired and expired water, respectively; pHa, pHv pH of arterial and venous blood, respectively; P_{w-b}, effective water to blood oxygen partial pressure difference; ΔPg partial pressure (tension) gradient; $\dot{Q}$ cardiac output; R vascular resistance; SV stroke volume; SEM standard error of mean; TO_2 transfer factor; U utilization; $\dot{V}_g$ ventilation volume; $\dot{V}O_2$ oxygen consumption

Introduction

Anatomical, physiological, and biochemical adaptations enable tunas to consume oxygen at rates that are unmatched by other teleosts (Brett 1972; Gooding et al. 1981; Brill 1987). As a consequence of their high aerobic

demand even at low swimming speeds (Gooding et al. 1981; Jones et al. 1990), the distribution of tunas has been hypothesized to be determined in large measure by ambient oxygen levels (Barkley et al. 1978; Sund et al. 1981). Early laboratory and field studies on the hypoxia sensitivity of tunas have focused on changes in swimming speed (Dizon 1977), time to mortality (Gooding et al. 1981), or fisheries catch data (Ingham et al. 1977; Hanamoto 1987). Results from these studies suggest that hypoxia tolerance is quite variable among tuna species (Sharp 1978).

More recent work investigating the ambient oxygen sensitivity of yellowfin tuna *(Thunnus albacares)* and skipjack tuna *(Katsuwonus pelamis)* (Bushnell et al. 1990; Bushnell and Brill 1991a) indicates that both species respond to hypoxia by increasing gape, $\dot{V}_g$ and swimming speed, while reducing HR and ultimately $\dot{Q}$. Based on when cardiorespiratory responses are initiated (P_iO_2 110–139 mmHg), Bushnell et al. (1990) have demonstrated that tunas are no more sensitive to hypoxia than other marine fishes. Although the observed physiological and behavioral changes could be presumed to be aimed at maintaining oxygen supply to the tissues, none of the previous studies have demonstrated how effective they are in achieving this. Also, no previous studies have measured the effects of hypoxia on arterial or venous blood pressures, blood gases, or blood acid-base status in tunas.

The primary objectives of this study were to determine how well the cardiorespiratory systems of skipjack and yellowfin tunas function in normoxia compared with other fish species, and how well they are able to maintain oxygen delivery to the tissues during hypoxia. To meet these objectives, HR, $\dot{Q}$, P_iO_2, P_cO_2 and P_aO_2, P_vO_2, C_aO_2 and $C_vO_2$were measured. When combined in appropriate equations with estimates of $\dot{V}_g$, these variables are used to calculate standard measures of performance of the cardiorespiratory system in normoxia and hypoxia.

Materials and methods

Ten yellowfin tuna (1.42 ± 0.2 kg) and nine skipjack tuna (1.64 ± 0.3 kg) were used. Fish were captured by local commercial fishermen and maintained in shore-side tanks at the Kewalo Research Facility (Southwest Fisheries Science Center Honolulu Laboratory, National Marine Fisheries Service, NOAA). Fish maintenance and handling procedures at this laboratory are described in Nakamura (1972). Food was presented daily, but withheld for 24 h prior to an experiment to allow sufficient time for gut clearance (Magnuson 1969).

Surgical and instrumentation procedures. Fish were netted in their holding tank and anesthetized by being placed in a plastic bag containing oxygenated seawater and MS222 ($1\ g \cdot l^{-1}$) buffered with an equimolar concentration of $NaHCO_3$. Fish were then quickly moved to a surgery table where they were force-ventilated with oxygenated seawater containing a maintenance dose of $NaHCO_3$-buffered MS222 ($0.1\ g \cdot l^{-1}$). Subjects were instrumented with a pulsed Doppler cardiac output probe, four exhalant water sampling catheters, and dorsal and ventral aorta cannulae as described in Jones et al. (1986, 1990), Bushnell et al. (1990), and Bushnell and Brill (1991a).

Following surgery, which generally took 50–60 min, fish were spinally blocked with an injection of lidocaine HCl and placed ventral side up in a restraining apparatus described in Bushnell et al. (1990). They were then positioned immediately in front of a pipe delivering seawater at approximately $35\ l \cdot min^{-1}$. Tunas are obligate ram ventilators, and this system provides adequate ventilation while allowing fish to set their own $\dot{V}_g$ (Bushnell et al. 1990). Fish were left undisturbed for at least 1 h after the completion of surgery to recover from anesthesia.

Measurement and data recording procedures. The analyses of water PO_2, blood gases, and blood oxygen content were performed using three Radiometer PHM 73 blood gas analyzers. The first was connected to an oxygen probe mounted in a water-jacketed, flow-through cuvette. Cuvette temperature was maintained at 25 °C. The oxygen probe, calibrated with standard zero-PO_2 solution and air-saturated seawater, was used to measure P_iO_2 and P_eO_2. A second blood gas analyzer was connected to a BMS3/MK2 Blood Micro System (also maintained at 25 °C) and was used to measure pH, PO_2, and PCO_2 of dorsal aorta (arterial) and ventral aorta (venous) blood samples (pHa, pHv, P_aO_2, P_vO_2, P_aCO_2, and P_vCO_2, respectively). The PO_2 electrode was calibrated with zero PO_2 solution and air-saturated saline, the PCO_2 electrode with precision-mixed gases (PCO_2 4.0 and 23.6 mmHg), and the pH electrode with pH 7.00 and 7.80 buffers. The third blood gas analyzer was used to measure blood oxygen content as described by Tucker (1967).

A Valpey-Fischer[1] Model VF-1 pulsed Doppler system was used to measure $\dot{Q}$ [theory of operation described in Hartley et al. (1978)]. A single naked crystal (1 mm diameter) was embedded in a drop of silicon rubber sealant which was trimmed to a smooth oval shape (0.5 cm × 0.75 cm). The embedded crystal was glued with Vetbond tissue adhesive to the thin membrane in the gill cavity overlying the ventral aorta as described in Bushnell et al. (1990).

The mean $\dot{Q}$ signal from the Doppler flow system was calibrated immediately following the 1 h allowed for recovery from surgery and anesthesia by simultaneous measurement of $\dot{Q}$ using dye dilution. Indocyanine green dye solution (0.1–0.2 ml, $\approx 2\ mg \cdot ml^{-1}$) was injected into the ventral aorta via the indwelling cannula. Concentration of the dye in blood downstream of the heart was determined by withdrawing a subsample of dorsal aortic blood through a densitometer cuvette connected to a Waters D–400 Densitometer. Data recording and calculation of $\dot{Q}$ from dye dilution curves were performed using standard procedures as described in Bushnell (1988) and Brill and Bushnell (1989). Five to seven measurements of $\dot{Q}$, with 5-min intervals between each, were made on every fish.

BP_{da} and BP_{va} were monitored with U-Onics P–106 Physiological Pressure Transducers connected to Gould amplifiers. The transducers were calibrated daily with a water manometer. In most cases, the BP_{va} signal triggered a cardiotachograph. The output signals from pressure transducers, the Radiometer measuring P_iO_2, the pulsed Doppler flow meter (mean and pulsatile $\dot{Q}$), and cardiotachograph were simultaneously recorded on a Gould 260 6-channel pen recorder. In addition, a Dianachart A/D converter and an IBM AT computer recorded mean BP_{va} and BP_{da}, $\dot{Q}$, HR, and P_iO_2 every 5 s. These data were stored on disk for later analysis. Data presented here were taken from the digitized records with their accuracy checked against the chart records.

No attempt was made to measure $\dot{V}_g$ in this study, because preliminary experiments had shown that having both the dye diffuser and the dorsal aorta catheter in the mouth would interfere with ventilatory water flow. $\dot{V}_g$ was therefore estimated from regression equations of $\dot{V}_g$ on P_iO_2 based on data obtained previously under similar circumstances (Bushnell 1988; Bushnell et al. 1990).

[1] Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA

For yellowfin and skipjack tunas, respectively, the regression equations used were:

$$\dot{V}_g = \left(\frac{P_iO_2 - 154}{154}\right) \cdot (-7.16) + 3.9; \quad r^2 = 0.92 \tag{1}$$

$$\text{and } \dot{V}_g = \left(\frac{P_iO_2 - 154}{154}\right) \cdot (-5.67) + 6.7; \quad r^2 = 0.98 \tag{2}$$

where $\dot{V}_g$ is in $l \cdot min^{-1} \cdot kg^{-1}$ and P_iO_2 in mmHg.

Experimental protocol. Fish were exposed to three levels of hypoxia ($P_iO_2 \approx 130$, 90, and 50 mmHg), presented in random order. Control measurements of cardiorespiratory variables were made during the normoxic period ($P_iO_2 > 150$ mmHg) immediately preceding the hypoxic episode. HR, BP_{va}, BP_{da}, $\dot{Q}$, and P_iO_2 were continuously recorded, exhalant water samples were taken (for P_eO_2) and stored in 5-ml plastic syringes, and dorsal and ventral aortic blood samples taken in precooled glass syringes and stored on ice. P_iO_2 was then reduced to the preselected level and held there for 2 min before the second series of blood and exhalent water samples were taken. The P_iO_2 was then returned to normoxic levels and the fish left undisturbed for 1 h. During this time, the eight water and four blood samples were analyzed. Hct was also determined.

At the conclusion of the experiment, fish were sacrificed, weighed, and necropsied to record the position of the dorsal and ventral aortic catheters and the pulsed Doppler blood flow probe.

Calculations. The Fick principle was used to calculate the following three metabolic rates:
Whole animal $\dot{V}O_2$ ($= \dot{V}O_2$-total) in ml $O_2 \cdot min^{-1} \cdot kg^{-1}$;

$$\dot{V}O_2\text{-total} = \dot{V}_g \cdot ((P_iO_2 - P_eO_2) \cdot \alpha_w) \tag{3}$$

where α_w = solubility coefficient of oxygen in seawater (ml $O_2 \cdot l^{-1} \cdot mmHg^{-1}$) at 25 °C;
$\dot{V}O_2$ of all tissues excluding the gills[2] ($= \dot{V}O_2$-body) in ml $O_2 \cdot min^{-1} \cdot kg^{-1}$;

$$\dot{V}O_2\text{-body} = \dot{Q} \cdot \left(\frac{C_aO_2 - C_vO_2}{100}\right) \tag{4}$$

where $\dot{Q}$ is in $ml \cdot min^{-1} \cdot kg^{-1}$, and C_aO_2 and C_vO_2 are in ml $O_2 \cdot dl^{-1}$;
and $\dot{V}O_2$ of the gills ($= \dot{V}O_2$-gill) in ml $O_2 \cdot min^{-1} \cdot kg^{-1}$

$$\dot{V}O_2\text{-gill} = (\dot{V}O_2\text{-total} - \dot{V}O_2\text{-body}) \tag{5}$$

Data from other measured variables were used to calculate the following standard indicators of cardiorespiratory function:
Oxygen delivery to all tissues excluding the gills (O_2 delivery, in ml $O_2 \cdot min^{-1} \cdot kg^{-1}$);

$$O_2 \text{ delivery} = \dot{Q} \cdot (C_aO_2 \cdot 100) \tag{6}$$

Ventilation/perfusion conductance ratio ($\dot{V}_g/\dot{Q}$ conductance);

$$\frac{\dot{V}_g}{\dot{Q}} \text{ conductance} = \frac{\dot{V}_g \cdot \alpha_w}{\dot{Q} \cdot \alpha_b} \tag{7}$$

where α_b is the solubility coefficient of blood at the prevailing P_aO_2 (i.e., C_aO_2/P_aO_2);
Utilization (U, %) or the fraction of oxygen content removed from the water;

$$U = \left(\frac{P_iO_2 - P_eO_2}{P_iO_2}\right) \cdot 100 \tag{8}$$

E_w (%), which is the ratio of the actual rate of removal of oxygen from the water ($\dot{V}O_2$) and the theoretical maximum rate possible (Hughes and Shelton 1962);

$$E_w = \left(\frac{P_iO_2 - P_eO_2}{P_iO_2 - P_vO_2}\right) \cdot 100 \tag{9}$$

Effectiveness of oxygen uptake by blood (E_b, %) which is the ratio of the actual rate of oxygen uptake by the blood and the theoretical maximum rate possible;

$$E_b = \left(\frac{C_aO_2 - C_vO_2}{C_{i_{eq}}O_2 - C_vO_2}\right) \cdot 100 \tag{10}$$

where $C_{i_{eq}}O_2$ is the oxygen content that would be achieved if P_aO_2 was equal to P_iO_2;
Transfer factor (TO_2, in ml $O_2 \cdot min^{-1} \cdot mmHg^{-1} \cdot kg^{-1}$) which is the rate of O_2 transfer from the water to the blood per unit partial pressure difference (ΔPg) between P_iO_2 and P_vO_2 (Randall et al. 1967; Piiper and Scheid 1984);

$$TO_2 = \frac{\dot{V}O_2\text{-water}}{\Delta Pg} \tag{11}$$

where

$$\Delta Pg = \tfrac{1}{2} \cdot (P_iO_2 + P_eO_2) - \tfrac{1}{2} \cdot (P_aO_2 + P_vO_2) \tag{12}$$

This calculation assumes a linear dissociation curve, and its use has been criticized by Piiper and Baumgarten-Schumann (1968). Therefore, we also calculated diffusion capacity (DO_2, in ml $O_2 \cdot min^{-1} \cdot mmHg^{-1} \cdot kg^{-1}$) which is similar to TO_2 but accommodates the properties of hemoglobin (Scheid and Piiper 1976)

$$DO_2 = \frac{\dot{V}O_2\text{-total}}{P_{w-b}} \tag{13}$$

where

$$P_{w-b} = \frac{(P_iO_2 - P_aO_2) - (P_eO_2 - P_vO_2}{\ln\left[\frac{(P_iO_2 - P_aO_2}{(P_eO_2 - P_vO_2)}\right]} \tag{14}$$

[2] $C_{i_{eq}}O_2$ was calculated based on the yellowfin and skipjack tuna blood oxygen dissociation curves presented in Fig. 1 [data from Brill and Bushnell (1991)]. Regression lines were fitted to these data using an iterative least-squares technique (Sigmaplot ver. 4.1) and the following non-linear equation

$$\text{Saturation (\%)} = \frac{a-d}{\left[1+\left(\frac{PO_2}{c}\right)^b\right]^e} + d$$

The fitted parameters from skipjack tuna blood were:

$a = 101.93887$, $b = -1.45585$, $c = 8.15136$, $d = -1.80961$, and $e = 3.41219$

and for yellowfin tuna blood were:

$a = 103.40217$, $b = -3.29641$, $c = 44.91710$, $d = 1.10164$, and $e = 0.27497$.

Percent saturation at a given P_aO_2 was calculated from these equations and maximum blood oxygen content estimated by extrapolating to 100% saturation from measured C_aO_2. Percent saturation at a given P_iO_2 was then calculated and $C_{i_{eq}}O_2$ estimated based this value and maximum blood oxygen content.

Data from measured variables were also used to calculate the following standard indicators of cardiovascular function:

SV (in ml · beat^{-1} · kg^{-1});

$$SV = \frac{\dot{Q}}{HR} \quad (15)$$

Resistance to blood flow offered by the gills (R_{branch}, in mmHg · ml^{-1} · min^{-1} · kg^{-1});

$$R_{branch} = \frac{BP_{va} - BP_{da}}{\dot{Q}} \quad (16)$$

Resistance to blood flow offered by the systemic circulation (R_{system}, in mmHg · ml^{-1} · min^{-1} · kg^{-1});

$$R_{system} = \frac{BP_{da} - BP_{v}}{\dot{Q}} \quad (17)$$

Total resistance to blood flow offered by the gill and systemic circulations (R_{total}, in mmHg · ml^{-1} · min^{-1} · kg^{-1});

$$R_{total} = \frac{BP_{va} - BP_{v}}{\dot{Q}} \quad (18)$$

where venous pressure (BP_v) is assumed to be zero in both cases; Cardiac power output (mW · kg^{-1});

$$\text{cardiac power output} = (BP_{va} \cdot \dot{Q}) \cdot 2.23 \cdot 10^{-3} \quad (19)$$

where $2.23 \cdot 10^{-3}$ converts mmHg · ml · min^{-1} to mW.

Statistical analysis procedures. As stated, experiments consisted of three hypoxia trials during which fish were exposed to P_iO_2s of approximately 130 mmHg, 90 mmHg, or 50 mmHg. Changes occurring during hypoxia were calculated by comparing values measured during the normoxic period (1–3 min) immediately preceding a hypoxic episode with those measured during the hypoxic episode. The 95% confidence intervals were used to determine statistically significant changes occurring during hypoxia. Inter-trial comparison of variables in the three control or hypoxia groups, as well as comparisons with data collected in previous studies, were made using analysis of variance (ANOVA) and a multiple-range test. Interspecific comparisons, or comparisons of data collected in only one other study, were made with an unpaired Student's *t*-test. In all

Table 1. Mean values (± SEM) of cardiorespiratory variables measured in normoxia and significant changes occurring during hypoxia in yellowfin tuna *(Thunnus albacares)*

Variable	Control	Hypoxia		
		($P_iO_2 \approx 130$ mmHg)	($P_iO_2 \approx 90$ mmHg)	($P_iO_2 \approx 50$ mmHg)
P_iO_2 (mmHg)	154.1 ± 0.6 (10)	−23.6 ±1.8 (8)	−65.5 ±2.2 (10)	−103.1 ± 1.9 (7)
P_eO_2 (mmHg)	75.9 ± 3.7 (10)	NS	−16.8 ±4.4 (10)	na
P_aO_2 (mmHg)	74.3 ± 6.3 (10)	NS	−23.6 ±4.3 (10)	− 40.5 ± 7.8 (7)
P_vO_2 (mmHg)	32.3 ± 3.2 (9)	− 1.2 ±0.5 (7)	− 4.5 ±1.0 (9)	− 8.7 ± 0.8 (6)
C_aO_2 (ml · dl^{-1})	13.6 ± 1.2 (10)	− 0.9 ±0.4 (8)	− 1.7 ±0.7 (10)	− 2.4 ± 0.5 (7)
C_vO_2 (ml · dl^{-1})	9.0 ± 0.8 (9)	NS	NS	− 2.4 ± 0.4 (6)
$C_aO_2 - C_vO_2$ (ml · dl^{-1})	4.9 ± 0.8 (9)	− 1.1 ±0.4 (7)	NS	NS
P_aCO_2 (mmHg)	3.3 ± 0.4 (8)	− 0.3 ±0.1 (7)	NS	− 0.8 ± 0.3 (7)
P_vCO_2 (mmHg)	3.8 ± 0.5 (8)	NS	NS	− 0.6 ± 0.2 (6)
pHa	7.82± 0.03 (10)	+ 0.02±0.01 (8)	+ 0.05±0.01 (10)	+ 0.06± 0.01 (7)
pHv	7.83± 0.02 (9)	NS	NS	NS
pHa − pHv	− 0.01± 0.02 (9)	NS	+ 0.04±0.01 (8)	+ 0.06± 0.01 (6)
O_2 delivery (ml · min^{-1} · kg^{-1})	14.5 ± 1.5 (8)	NS	NS	− 6.3 ± 1.7 (7)
$\dot{V}O_2$-body (ml · min^{-1} · kg^{-1})	4.7 ± 0.4 (7)	− 1.5 ±0.4 (6)	NS	− 1.3 ± 0.2 (6)
$\dot{V}O_2$-total (ml · min^{-1} · kg^{-1})	10.5 ± 0.5 (10)	+ 1.1 ±0.5 (8)	NS	na
$\dot{V}O_2$-gill (ml · min^{-1} · kg^{-1})	5.9 ± 0.8 (7)	+ 2.5 ±0.7 (6)	NS	na
$\dot{V}_g$ (l · min^{-1} · kg^{-1})	3.9 ± 0.1 (10)	+ 1.1 ±0.8 (8)	+ 3.0 ±0.1 (10)	+ 5.1 ± 0.3 (8)
$\dot{Q}$ (ml · min^{-1} · kg^{-1})	115.4 ±17.4 (8)	NS	NS	− 40.9 ±17.4 (6)
$\dot{V}_g/\dot{Q}$	43.4 ± 8.8 (8)	+11.5 ±2.6 (7)	+39.2 ±6.7 (8)	+ 86.8 ± 7.8 (6)
$\dot{V}_g/\dot{Q}$ conductance	0.73± 0.10 (8)	+ 0.18±0.08 (7)	+ 0.26±0.10 (8)	+ 0.58± 0.08 (6)
ΔPg (mmHg)	62.3 ± 4.4 (9)	− 9.3 ±2.2 (7)	−27.6 ±3.4 (9)	na
TO_2 (ml · min^{-1} · mmHg^{-1} · kg^{-1})	0.17± 0.02 (7)	+ 0.04±0.01 (7)	+ 0.22±0.06 (9)	na
DO_2 (ml · min^{-1} · mmHg^{-1} · kg^{-1})	0.18± 0.02 (9)	+ 0.05±0.01 (7)	+ 0.23±0.07 (9)	na
U (%)	50.8 ± 2.3 (10)	− 7.5 ±1.8 (8)	−18.0 ±4.0 (10)	na
E_w (%)	62.6 ± 2.7 (7)	− 7.4 ±2.7 (7)	−15.3 ±5.3 (9)	na
E_b (%)	89.3 ± 0.4 (7)	NS	−23.7 ±7.8 (9)	− 17.3 ± 7.0 (6)
HR (beates · min^{-1})	96.7 ± 5.8 (10)	NS	NS	− 26.8 ± 4.3 (7)
SV (ml · beat^{-1} · kg^{-1})	1.3 ± 0.2 (8)	NS	NS	NS
BP_{va} (mmHg)	89.7 ± 8.1 (10)	NS	NS	NS
BP_{da} (mmHg)	32.6 ± 2.4 (10)	NS	NS	NS
R_{branch} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	0.71± 0.06 (8)	NS	NS	+ 0.25± 0.11 (6)
R_{system} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	0.33± 0.02 (8)	NS	NS	+ 0.12± 0.04 (6)
R_{total} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	1.05± 0.06 (7)	NS	NS	+ 0.37± 0.13 (6)
Cardiac power output (mW · kg^{-1})	27.2 ± 4.3 (8)	NS	NS	− 11.7 ± 4.6 (6)
Hct (%)	26.7 ± 1.2 (10)	NS	NS	NS

Number of fish is in parentheses; NS = not significantly different from normoxia values; na = data not available

cases a P-value lower than 0.05 was taken as the fiducial limit of significance. The grand means for the normoxic measurements were calculated by averaging data collected during the control period from all three trials for each individual to arrive at a mean value for each fish. These values were, in turn, averaged to determine the grand mean for the species. All values in the text, tables, and figures are expressed as mean ± standard error of the mean (SEM).

Results

Normoxia

There were no significant differences in mean control values measured immediately before each level of hypoxia. This indicates that both tuna species remained physiologically stable throughout the course of an experiment, or that randomizing the sequence of presentation of the three levels of hypoxia removed any effects of physiological changes occurring over time. Most of the 35 measured or calculated variables (Tables 1, 2) were similar in skipjack and yellowfin tunas, with the exception of $\dot{V}O_2$-total, $\dot{V}O_2$-gill, $\dot{V}_g$, TO_2, DO_2, HR, and Hct, which were significantly higher in skipjack tuna, and E_b, which was significantly lower.

Hypoxia: yellowfin tuna

During the 50 mmHg trial, P_eO_2 sometimes equaled or exceeded P_iO_2. Therefore, we assume that exhaled water samples became contaminated by surrounding water when mouth gape and opercular flair increased by a large degree. As a result, no P_eO_2s or associated calculations ($\dot{V}O_2$-total, $\dot{V}O_2$-gill, ΔPg, TO_2, DO_2, U, or E_w) are reported for this level of hypoxia.

Exposure of yellowfin tuna to hypoxia eventually resulted in significant changes in all measured and calculated variables except pHv, SV, BP_{va}, BP_{da}, and Hct (Table 1). When P_iO_2 was reduced to only 130 mmHg, P_aO_2, P_vO_2, and C_aO_2 were already significantly reduced,

Table 2. Mean values (± SEM) of cardiorespiratory variables measured in normoxia and significant changes occurring during hypoxia in skipjack tuna *(Katsuwonus pelamis)*

Variable	Control	Hypoxia		
		($P_iO_2 \approx 130$ mmHg)	($P_iO_2 \approx 90$ mmHg)	($P_iO_2 \approx 50$ mmHg)
P_iO_2 (mmHg)	152.5 ± 1.2 (9)	−22.3 ± 3.0 (8)	−66.9 ± 2.7 (6)	−101.3 ± 2.4 (6)
P_eO_2 (mmHg)	74.7 ± 3.1 (9)	NS	−23.5 ± 6.1 (7)	na
P_aO_2 (mmHg)	69.7 ± 7.1 (9)	− 7.1 ± 2.3 (6)	−26.7 ± 4.6 (8)	− 51.3 ± 6.2 (6)
P_vO_2 (mmHg)	36.7 ± 3.0 (9)	− 3.8 ± 0.6 (6)	−10.8 ± 1.9 (8)	− 17.6 ± 3.8 (6)
C_aO_2 (ml · dl^{-1})	15.1 ± 2.1 (9)	NS	− 1.9 ± 0.8* (8)	− 5.5 ± 1.7* (6)
C_vO_2 (ml · dl^{-1})	10.0 ± 1.5 (9)	NS	− 2.3 ± 0.5 (8)	− 3.9 ± 0.7 (6)
$C_aO_2 - C_vO_2$ (ml · dl^{-1})	5.1 ± 0.9 (9)	NS	NS	NS
P_aCO_2 (mmHg)	3.0 ± 0.4 (9)	NS	NS	− 0.8 ± 0.2
P_vCO_2 (mmHg)	3.8 ± 0.5 (8)	NS	NS	− 0.4 ± 0.2 (6)
pHa	7.86 ± 0.03 (9)	+ 0.03 ± 0.004 (6)	+ 0.06 ± 0.01 (8)	+ 0.12 ± 0.03 (6)
pHv	7.86 ± 0.04 (9)	NS	+ 0.03 ± 0.01 (8)	NS
pHa − pHv	− 0.01 ± 0.01 (9)	NS	+ 0.03 ± 0.01 (8)	+ 0.11 ± 0.02 (6)
O_2 delivery (ml · min^{-1} · kg^{-1})	18.2 ± 3.4 (9)	NS	− 5.8 ± 1.5 (8)	− 11.3 ± 2.3 (6)
$\dot{V}O_2$-body (ml · min^{-1} · kg^{-1})	5.8 ± 1.0 (9)	NS	NS	NS
$\dot{V}O_2$-total (ml · min^{-1} · kg^{-1})	18.2 ± 0.7# (9)	NS	NS	na
$\dot{V}O_2$-gill (ml · min^{-1} · kg^{-1})	12.4 ± 0.8# (9)	NS	NS	na
$\dot{V}_g$ (l · min^{-1} · kg^{-1})	6.8 ± 0.1# (9)	+ 0.8 ± 0.1 (5)	+ 2.4 ± 0.1 (8)	+ 3.7 ± 0.1 (7)
$\dot{Q}$ (ml · min^{-1} · kg^{-1})	132.3 ± 26.5 (9)	NS	− 27.0 ± 12.7 (8)	− 57.3 ± 16.4 (6)
$\dot{V}_g/\dot{Q}$	71.8 ± 13.3 (9)	NS	+ 44.0 ± 12.3 (8)	+ 99.6 ± 20.5 (6)
$\dot{V}_g/\dot{Q}$ conductance	1.09 ± 0.16 (9)	NS	NS	+ 0.53 ± 0.17 (6)
ΔPg (mmHg)	60.5 ± 4.8 (9)	NS	− 29.4 ± 5.3 (8)	na
TO_2 (ml · min^{-1} · mmHg^{-1} · kg^{-1})	0.32 ± 0.03# (9)	NS	+ 0.32 ± 0.10 (8)	na
DO_2 (ml · min^{-1} · mmHg^{-1} · kg^{-1})	0.35 ± 0.03# (9)	NS	+ 0.22 ± 0.07 (7)	na
U (%)	51.0 ± 2.0 (9)	− 10.2 ± 3.1 (8)	NS	na
E_w (%)	67.4 ± 2.9 (9)	− 12.3 ± 4.3 (6)	NS	na
E_b (%)	74.1 ± 1.5# (9)	NS	− 9.3 ± 4.5	− 26.7 ± 10.2 (6)
HR (beats · min^{-1})	125.9 ± 14.8# (9)	− 14.1 ± 4.5 (6)	− 31.5 ± 9.1 (8)	− 48.0 ± 9.7 (6)
SV (ml · beat^{-1} · kg^{-1})	1.1 ± 0.1 (9)	NS	NS	− 0.13 ± 0.06 (6)
BP_{va} (mmHg)	87.3 ± 5.4 (8)	NS	NS	NS
BP_{da} (mmHg)	40.2 ± 3.4 (9)	NS	− 4.0 ± 1.4 (7)	NS
R_{branch} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	0.43 ± 0.06 (8)	NS	+ 0.13 ± 0.06 (6)	+ 0.31 ± 0.08 (5)
R_{system} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	0.33 ± 0.07 (9)	NS	NS	NS
R_{total} (mmHg · ml^{-1} · min^{-1} · kg^{-1})	0.74 ± 0.16 (8)	NS	NS	+ 0.46 ± 0.12 (5)
Cardiac power output (mW · kg^{-1})	29.2 ± 0.9 (7)	NS	− 6.2 ± 2.8 (7)	− 11.4 ± 3.7 (5)
Hct (%)	33.9 ± 1.4# (8)	NS	NS	NS

# Denotes significant difference between skipjack and yellowfin tunas during normoxia
Number of fish is in parentheses; NS = not significantly different from normoxia values; na = data not available

and all continued to decrease as hypoxia became more severe. C_vO_2 was maintained at control values until P_iO_2 was reduced to 50 mmHg, by which time it had fallen significantly. C_aO_2–C_vO_2 difference was reduced at 130 mmHg, but not at 90 and 50 mmHg. A significant decrease in P_aCO_2 resulted in a significant increase in pHa at all levels of hypoxia. However, P_vCO_2 did not decrease until yellowfin tuna were exposed to the most intense level of hypoxia. As a result, pHv was maintained at control levels throughout, producing a significant pHa–pHv difference at 90 and 50 mmHg. The lack of bradycardia at 130 and 90 mmHg allowed O_2 delivery to be maintained at normoxic levels during moderate hypoxia, despite the slight fall in C_aO_2. At 50 mmHg, a significant bradycardia occurred and $\dot{Q}$ fell because SV did not increase; consequently, O_2 delivery fell. Blood pressure remained unchanged during a 36% fall in $\dot{Q}$ at 50 mmHg. This indicates that a general vasoconstriction had occurred, as shown by significant increases in R_{branch}, R_{system}, and R_{total}. The reduced $\dot{Q}$ at this level of hypoxia also resulted in a significant reduction in cardiac power output.

The C_aO_2–C_vO_2 difference, which was reduced at 130 mmHg because of the fall in C_aO_2, regained normoxic values at 90 and 50 mmHg because of a decline in C_aO_2. $\dot{V}O_2$-body was significantly lower at 130 mmHg as a consequence of the smaller C_aO_2–C_vO_2 difference; it returned to normoxic levels when P_iO_2 was reduced to 90 mmHg, as the C_aO_2–C_vO_2 difference also returned to control values. $\dot{V}O_2$-body was significantly reduced at 50 mmHg because $\dot{Q}$ fell. As a result of a significant increase in $\dot{V}O_2$-total concomitant with the decrease in $\dot{V}O_2$-body at 130 mmHg, $\dot{V}O_2$-gill was elevated at 130 mmHg, but returned to normoxic levels at 90 mmHg.

Gas exchange variables were also sensitive to changes in P_iO_2. As $\dot{V}_g$ increased during hypoxia, the $\dot{V}_g/\dot{Q}$ ratio rose significantly through all levels of hypoxia to a high of 125 at 50 mmHg. Although a reduction in U occurred as a consequence of the increased $\dot{V}_g$, it was not enough to prevent the $\dot{V}_g/\dot{Q}$ conductance ratio from increasing as hypoxia became more severe. Despite a significant increase in TO_2 and DO_2 at 130 and 90 mmHg, E_w fell at 130 and 90 mmHg. E_b remained at normoxic levels at 130 mmHg, but was significantly reduced at both 90 and 50 mmHg.

Hypoxia: skipjack tuna

As in yellowfin tuna, exhaled water samples apparently became contaminated by surrounding water during the 50 mmHg hypoxia trial. Therefore, no P_cO_2s or associated calculations ($\dot{V}O_2$-total, $\dot{V}O_2$-gill, ΔPg, TO_2, DO_2, U, or E_w) are reported for this level of hypoxia.

Changes believed to be indicative of failure in the oxygen extraction and delivery systems occurred at moderate levels of hypoxia (Table 2). Significant bradycardia occurred at 130 mmHg and became more severe as hypoxia deepened. This resulted in a significant reduction in $\dot{Q}$ at 90 mmHg which, along with a decrease in C_aO_2, caused O_2 delivery to begin to decline. Other aspects of the skipjack tuna's inability to tolerate hypoxia included a relatively slow increase in TO_2 and DO_2, neither of which changed until P_iO_2 was reduced to 90 mmHg. Interestingly, E_w was significantly reduced at 130 mmHg but not at 90 mmHg, while E_b was unchanged at 130 mmHg, but significantly reduced at 90 mmHg and further reduced at 50 mmHg. The increase in $\dot{V}_g/\dot{Q}$ ratio observed at every level of hypoxia was accompanied by an increase of $\dot{V}_g/\dot{Q}$ conductance only when P_iO_2 had fallen to 50 mmHg.

Because the decrement in C_aO_2 was accompanied by a fall in C_vO_2, the arterial-venous oxygen content difference never varied significantly from control measurements. P_aCO_2 was reduced at all levels of hypoxia. Therefore, pHa was significantly increased, and the pHa–pHv difference became significant at 90 and 50 mmHg.

BP_{da} was essentially maintained despite significant reductions in $\dot{Q}$ at 90 and 50 mmHg. However, statistically significant increases in R_{branch} and R_{total} occurred only at a P_iO_2 of 50 mmHg. Cardiac power output was reduced at 90 mmHg because of the lowered $\dot{Q}$ and was reduced by approximately 60% at 50 mmHg because of the steep reduction in $\dot{Q}$.

Discussion

Cardiovascular function during normoxia. The P_aO_2 values of skipjack and yellowfin tunas (69.7 and 74.3 mmHg, respectively) in this study are similar to those found in paralyzed force-ventilated albacore *(Thunnus alalunga)* [62 mmHg, White et al. (1988)] but are lower those of lightly anesthetized, force-ventilated skipjack tuna [90 mmHg, Stevens (1972)]. More important, our values nearly match those found in free-swimming kawakawa *(Euthynnus affinis)* [63 mmHg, Jones et al. (1986)], indicating that the P_aO_2 values for the fish in this study were not abnormal nor an artifact caused by surgical and other manipulations. P_aO_2 values in tunas are, surprisingly, much lower than those in conscious but sedentary rainbow trout [130 mmHg, Holeton and Randall (1967b), Kiceniuk and Jones (1977)]. The reasons for these differences are unknown. Note however, that during normoxia arterial blood is more than 80% saturated in skipjack tuna and more than 90% saturated in yellowfin tuna (Fig. 1).

P_vO_2 values are similar to those of force-ventilated skipjack tuna [32 mmHg, Stevens (1972)] and rainbow trout [30 mmHg, Holeton and Randall (1967a) and Kiceniuk and Jones (1977)] but are higher than those of force-ventilated albacore [12 mmHg, White et al. (1988)] and free-swimming kawakawa [13 mmHg, Jones et al. (1986)]. This is presumably due to oxygen demands being higher in the albacore which had been recently boated and were recovering from the stress of capture, and in the swimming kawakawa which had towed a number of catheters.

Arterial blood oxygen content in most other teleosts ranges from 4 to 10 ml · dl^{-1}, and those of tuna were thought to be much higher (Randall 1970). An unusually high C_aO_2 of 21.7 ml · dl^{-1} was reported by White et al. (1988) in stressed, freshly boated albacore that had Hcts of over 50%. We found the Hct in skipjack and yellowfin tunas to be substantially lower (27–33%) and similar to that (35%) reported by Jones et al. (1986) in free-swimming kawakawa. As a result, the C_aO_2s reported here are also lower than that found by White et al. (1988). Because we often saw Hcts of over 50% in yellowfin and skipjack tunas that had been netted and had blood sampled by cardiac puncture, we believe the reports of unusually high Hct and oxygen-carrying capacity of tuna blood to be a result of either the release of red cells into the circulation from the spleen or hemoconcentration. Both occur during capture or exercise in other teleosts (Yamamoto and Itazawa 1989).

The spinally blocked (i.e., non-swimming) tunas in this study maintained a significantly higher venous oxygen reserve (C_vO_2 was approximately 40–60% of C_aO_2) than observed in other teleosts. For example, in rainbow trout C_vO_2 is only 14% of C_aO_2 (Kiceniuk and Jones 1977). A high C_vO_2 level also been observed in paralyzed, force-ventilated albacore (White et al. 1988). However, the lower P_vO_2 seen in swimming kawakawa (Jones et al. 1986) implies that the high venous reserve seen in non-swimming tunas may be due to low oxygen consumption of the swimming muscles.

P_aCO_2 and P_vCO_2 recorded previously in tunas [4.1–5.5 and 5.5 mmHg, respectively; (Jones et al. 1986; White et al. 1988)] are only slightly higher then those reported here (3.0–3.3 and 3.8 mmHg, respectively). The kawakawa from which Jones et al. (1986) collected blood were free swimming, yet the investigators thought the fish were under ventilated and therefore had significantly elevated blood CO_2 levels. In light of our data, this apparently is not the case. White et al. (1988) found elevated P_vCO_2 (9.0 mmHg) and low pHv (7.55) in albacore, presumably because these fish were still recovering from the stress of capture. Finally, studies by Perry et al. (1985) and Brill et al. (1991) have shown that blood from skipjack and yellowfin tunas has a very high non-bicarbonate buffering capacity; therefore, the lack of an arterial to venous pH difference in our study is not surprising.

$\dot{Q}$ measured in this study (115–132 ml · min^{-1} · kg^{-1}) were significantly above those recorded in albacore [29.4–36.1 ml · min^{-1} · kg^{-1}; Lai et al. (1987), White et al. (1988)], although part of this difference may be explained by the albacore being much larger (7.4–11.2 kg body weight) than the fish studied here. Surprisingly, our measures of $\dot{Q}$ are close to that for skipjack tuna calculated using the Fick equation [80–100 ml · min^{-1} · kg^{-1}; (Stevens 1972; Brill et al. 1978)] despite the fact that the large gill $\dot{V}O_2$ of tunas (discussed below) would cause the Fick method to over estimate $\dot{Q}$ (Metcalf and Butler 1982). This apparent paradox remains to be resolved. Although affected by differences in body size and measurement temperature, the $\dot{Q}$ of skipjack and yellowfin tuna are approximately 3–10 times those recorded in other teleosts such as rainbow trout [17.6 ml · min^{-1} · kg^{-1}, Kiceniuk and Jones (1977)], cod [*Gadus morhua*, 19.2 ml · min^{-1} · kg^{-1}, Fritsche and Nilsson (1989)] or eel [*Anguilla anguilla*, 11.5 ml · min^{-1} · kg^{-1}, Peyraud-Waitzenegger and Soulier (1989)].

HRs measured in this study were similar to those recorded previously (Bushnell et al. 1990). In spite of the higher HR of skipjack tuna, SV values were similar (1.1–1.3 ml · beat^{-1} · kg^{-1}) in both species and comparable to that calculated for skipjack tuna from data presented in Stevens (1972). SV in skipjack and yellowfin tunas, however, are substantially higher than those recorded by White et al. (1988) and Lai et al. (1987) in 9- to 10-kg albacore (0.33–0.36 ml · beat^{-1} · kg^{-1}). Both HR and SV in skipjack and yellowfin tuna are also significantly higher than those of other teleosts such as rainbow trout [37.8 beats · min^{-1} and 0.46 ml · beat^{-1} · kg^{-1} (Kiceniuk and Jones 1977)], cod [41.4 beats · min^{-1} and 0.51 ml · beat^{-1} · kg^{-1} (Fritsche and Nilsson 1989)] or eel [37.1 beats · min^{-1} and 0.29 ml · beat^{-1} · kg^{-1} (Peyraud-Waitzenegger and Soulier 1989)].

Although high when compared with those in salmonids [30–70 mmHg (Holeton and Randall 1967a; Randall 1970)], the BP_{va} values in yellowfin and skipjack tunas in our study (87–90 mmHg) are in good agreement with those of Lai et al. (1987) for albacore. Interestingly, White et al. (1988) reported similar mean BP_{va} values in albacore, but mean BP_{da} values that were only 1–5 mmHg lower. In this study, blood pressure drop across the gills was 56 and 47 mmHg in yellowfin and skipjack tunas, respectively. Muir and Brown (1971) speculated that the oblique blood channels through the secondary lamellae of tuna are an adaptation to reduce the blood pressure drop across the gills. In support of this argument, they used morphometric and physiological data to calculate the $BP_{va} - BP_{da}$ differences when blood was flowing along the transverse length of the secondary lamellae or the shorter oblique channels. The drop in blood pressure predicted for the long transverse channels (68 mmHg) is close to that recorded here and by Lai et al. (1987), while the 3.9 mmHg reduction, calculated to result from blood flowing through the shorter oblique channels, is close to that observed by White et al. (1988). Typically, the branchial vasculature resistance represents 20–40% of the total vascular resistance in fishes. The R_{branch} in our study was 60% and 68% of R_{total} in skipjack and yellowfin tunas, respectively, but was only 26% and 4% of R_{total} using data for albacore presented in Lai et al. (1987) and White et al. (1988), respectively. Whether tunas can rearrange blood flow within the gill to bring about a significant modification of vascular resistance, as these data imply, remains to be determined.

Because of the high $\dot{Q}$ and BP_{va}, the cardiac power output of yellowfin and skipjack tunas exceeds that of other active species (e.g., rainbow trout) by approximately an order of magnitude (Brill and Bushnell 1991b).

Cardiorespiratory function during normoxia. Despite differences of an order of magnitude or more in $\dot{V}_g$ (and $\dot{V}O_2$) of starry flounder [*Platichthys stellatus*; $\dot{V}_g = 0.111 \cdot min^{-1} \cdot kg^{-1}$ (Wood et al. 1979)], tench [*Tinca tinca*; $\dot{V}_g = 0.121 \cdot min^{-1} \cdot kg^{-1}$ (Eddy 1974)], rainbow trout [$\dot{V}_g = 0.171 \cdot min^{-1} \cdot kg^{-1}$; Cameron and Davies (1970)], and eel [$\dot{V}_g = 0.084 l \cdot min^{-1} \cdot kg^{-1}$ (Peyraud-Waitzenegger and Soulier 1989)], skipjack tuna ($6.8 l \cdot min^{-1} \cdot kg^{-1}$) and yellowfin tuna ($3.9 l \cdot min^{-1} \cdot kg^{-1}$) (Stevens 1972; this study), all utilize approximately 50% of the oxygen in the water passing over the gills.

The $\dot{V}_g/\dot{Q}$ ratio in teleosts other than tunas ranges from 1.04 and 2.94 in the rainbow trout and starry flounder, respectively (Cameron and Davis 1970; Wood et al. 1979) to 7.34 and 8.25 in the eel and tench, respectively (Eddy 1974; Peyraud-Waitzenegger and Soulier 1989). The $\dot{V}_g/\dot{Q}$ ratio of skipjack and yellowfin tunas (Tables 1, 2) is much higher because of their higher $\dot{V}_g$. However, $\dot{V}_g/\dot{Q}$ conductance ratio, which takes into account differences in blood oxygen-carrying capacity, indicates that the ventilation/perfusion systems are well matched in skipjack tuna (1.09), while yellowfin tuna are slightly over-perfused (0.73). Previous studies in teleosts and elasmobranchs have found $\dot{V}_g/\dot{Q}$ conductance to vary from 0.42 in the larger spotted dogfish, *Scyliorhinus stellaris*, and 1.8 in the dogfish, *S. canicula* (Short et al. 1979), to 1.2 in rainbow trout (Randall et al. 1967). Based on theoretical grounds, a slight over-ventilation is considered advantageous for maintaining a high P_aO_2 (Scheid and Piiper 1976).

The TO_2s measured in our study (0.32 and $0.17 ml \cdot min^{-1} \cdot mmHg^{-1} \cdot kg^{-1}$ in skipjack and yellowfin tunas, respectively) are the highest recorded in any species to date and are similar to those made by Stevens (1972) for force-ventilated skipjack tuna. In contrast, the TO_2s of eel, tench, starry flounder, dogfish, and rainbow trout are only 0.0045 (Peyraud-Waitzenegger and Soulier 1989), 0.0061 (Eddy 1974), 0.0069 (Wood et al. 1979), 0.013 (Short et al. 1979), and 0.016 (Cameron and Davis 1970) $ml \cdot min^{-1} \cdot mmHg^{-1} \cdot kg^{-1}$, respectively. The diffusion capacity (DO_2), a related measure of oxygen transfer that incorporates the properties of hemoglobin (Eqs. 11, 12), was not significantly different from TO_2 in skipjack and yellowfin tunas, as was also found by Short et al. (1979) in the dogfish. Unfortunately, there are few reported measures of DO_2 in other species.

TO_2 and DO_2 depend primarily on the gill surface area available for exchange, and diffusion distance between blood and water (Randall et al. 1967). When corrected for size, a 200-g skipjack tuna has a gill surface area of 2051 mm^2 compared to a similarly sized rainbow trout which has a gill surface area of only 206 mm^2. In general, tunas have gill surface areas 5–10 times larger than other active teleosts (Hughes 1984a, b). In addition, the mean total water-blood distance in the secondary lamellae of tuna gills is very small, being only 0.60 and 0.53 μm in skipjack and yellowfin tunas, respectively, but 6.37 μm in rainbow trout (Hughes 1984a).

The fact that the P_aO_2 did not equal P_iO_2 suggests that the gills are not acting as perfect counter-current gas exchangers for oxygen. Indications of where departures from the ideal occur are provided by measurements of E_w and E_b. E_w values in both skipjack and yellowfin tunas (67% and 63%, respectively) were lower than previously estimated for skipjack tuna [90% (Stevens 1972)], but higher than those for rainbow trout [30–58% (Randall et al. 1967; Cameron and Davis 1970)], and dogfish [29% (Short et al. 1979)], and roughly equal to that of a starry flounder [75% (Wood and Shelton 1979)]. The average E_w of 65% in the tunas means that at least 65% of the water flowing past the gills was effectively "in contact" with blood in the gills, or conversely, only 35% of the water was "shunted" into pathways that did not participate in oxygen exchange (Piiper and Scheid 1984). With such a large $\dot{V}_g$, the fact that E_w is not lower is an indicator of the efficiency of tuna gills as a gas exchanger.

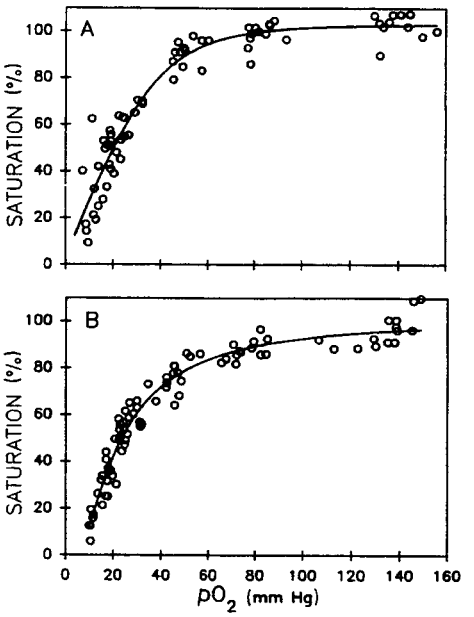

Fig. 1A, B. Blood oxygen dissociation curves for yellowfin tuna (**A**) and skipjack tuna (**B**). Data are from blood equilibrated at 20 and 30 °C with 0.5% CO_2 (i.e., arterial PCO_2) and various oxygen tensions. The P_{50}s for yellowfin and skipjack tuna were 20.5 and 22.5 mmHg, respectively. The plasma pHs at P_{50} were 7.876 and 7.925, respectively. Data were taken from the study on the effects of temperature on tuna blood oxygen binding characteristics by Brill and Bushnell (1991)

E_bs in starry flounder, tench, rainbow trout, and dogfish are 57, 81, 90, and 91%, respectively (Randall 1967; Randall et al. 1967; Short et al. 1979, Wood et al. 1979); in this study E_bs in skipjack and yellowfin tunas were 74 and 89%, respectively. The fact that E_b was relatively low in skipjack tuna indicates that measured C_aO_2s were substantially less than they would have been if P_aO_2 equaled P_iO_2 and blood leaving the gills was nearly saturated (see Eq. 10). In most fishes, blood leaving the gills is over 90% saturated, and the hemoglobin is functioning on the top (i.e., relatively flat) portion of the

oxygen dissociation curve (Cameron 1971; Eddy 1973). Therefore, large P_aO_2–P_iO_2 differences have little effect on C_aO_2. However, based on the blood oxygen dissociation curves (Fig. 1), blood leaving the gills in skipjack tuna (but not yellowfin tuna) is operating near the shoulder or steep portion of the dissociation curve, where C_aO_2 falls rapidly with PO_2. This may be due to the high oxygen demand of the gill tissue itself (discussed in the following section). In other words, the oxygen needed to meet the energy demands of the gill tissue in skipjack tuna may be taken from the blood as well as the water. However, it more likely that the differences in E_b between skipjack and yellowfin tunas is due to the differences in shapes of the blood oxygen dissociation curves at their upper ends. Yet to be determined is whether this situation changes during the increased periods of oxygen demand during high speed swimming and oxygen debt repayment.

Metabolic costs of large gill surface areas. The gills are the main sites of passive water and ion movements and are also the main organ of osmoregulation (Evans 1979). Therefore, fish (such as tunas) with large gill surface areas are likely to have high osmoregulatory costs (Stevens 1972; Brill 1987), much of which will occur at the gills. The cost of osmoregulation has been estimated to account for 27–50% of the standard metabolic rate (Rao 1968; Nordlie and Leffler 1975), and Daxboeck et al. (1982) estimated that the metabolism of the gills alone accounts for 27% (range 19–75%) of the standard metabolic rate of rainbow trout. In our study, $\dot{V}O_2$-gill accounted for 68% and 54% of $\dot{V}O_2$-total in skipjack and yellowfin tunas, respectively. When expressed as oxygen demand per unit gill surface area of a 1-kg fish (Hughes 1984a), the $\dot{V}O_2$-gill of skipjack and yellowfin tunas ($6.70 \cdot 10^{-6}$ and $4.43 \cdot 10^{-6}$ ml · min^{-1} · mm^{-2}, respectively) are substantially higher than that of (freshwater) rainbow trout [$0.77 \cdot 10^{-6}$ ml · min^{-1} · mm^{-2} (Daxboeck et al. 1982)] but equivalent to that of (sea water) Atlantic cod, *Gadus morhua* [$8.59 \cdot 10^{-6}$ ml · min^{-1} · mm^{-2}; (Johansen and Pettersson 1981)].

Cardiovascular and cardiorespiratory function during hypoxia. Studies using spinally blocked (force-ventilated) and free-swimming skipjack and yellowfin tunas showed that both species increased gape and $\dot{V}_g$ in response to hypoxia (Bushnell et al. 1990; Bushnell and Brill 1991). Although $\dot{V}_g$ could not be quantified in this study, an increase in mouth gape occurred, and it is assumed that $\dot{V}_g$ increased during hypoxia in a manner similar to that recorded previously.

Stevens (1972) observed a significant negative correlation between $\dot{V}_g$ and U in force-ventilated skipjack tuna during normoxia, as did Bushnell and Brill (1991) in free-swimming yellowfin tuna during hypoxia. Therefore, increased $\dot{V}_g$ probably accounts for some of the observed 14–35% decrease in U observed in this study during hypoxia. During an approximate doubling of $\dot{V}_g$ in response to hypoxia, U decreased 17–35% in flounder, rainbow trout, and European eel (Kerstens et al. 1979; Smith and Jones 1982; Steffensen et al. 1982; Le Moigne et al. 1986). A decrease in U, concomitant with a increase in $\dot{V}_g$ during hypoxia is not a universal response in fishes, however. In studies of spiny dogfish (Short et al. 1979), catfish *Ictalurus punctatus* (Burggren and Cameron 1980), plaice *Pleuronectes platessa* (Steffensen et al. 1982), and European eel (Peyraud-Waitzenegger and Soulier 1989), no changes in U were found during hypoxia despite increases in $\dot{V}_g$.

Significant bradycardia occurred at P_iO_2 values of 90 and 50 mmHg in skipjack tuna, but not until P_iO_2 reached 50 mmHg in yellowfin tuna. These data agree with those previously obtained for spinally blocked and swimming skipjack tuna, although the HR response of yellowfin tuna in the present study appeared less sensitive to hypoxia than that reported in Bushnell et al. (1990) and Bushnell and Brill (1991).

$\dot{Q}$ decreased by 19% and 42% in skipjack tuna at P_iO_2 values of 90 and 50 mmHg, respectively, and by 36% in yellowfin tuna at a P_iO_2 of 50 mmHg. The reductions in $\dot{Q}$ at a P_iO_2 of 50 mmHg are similar to those observed in lingcod *(Ophiodon elongatus)* and European eel (31% and 37% reduction in $\dot{Q}$, respectively) at similar levels of hypoxia (Farrell 1982; Peyraud-Waitzenegger and Soulier 1989). However, Atlantic cod and rainbow trout maintain $\dot{Q}$ down to a P_iO_2 of 30–40 mmHg (Wood and Shelton 1980; Fritsche and Nilsson 1989) and dogfish sharks down to a P_iO_2 of 77 mmHg (Short et al. 1979), in spite of significant bradycardia, by increasing in SV. In contrast, skipjack and yellowfin tunas showed no increases in SV.

The increase in SV during hypoxia-induced bradycardia has been attributed to a Starling response and an increase in circulating catecholamines compensating for the depressant effect of hypoxia on myocardial contractility (Short et al. 1977; Farrell 1985). In eels studied by Peyraud-Waitzenegger and Soulier (1989), there was no increase in circulating catecholamines during hypoxia and no increase in SV. The same situation could be occurring in tunas during hypoxia; however, another explanation is more likely: recent studies by Farrell et al. (1990) on the isolated, perfused hearts of skipjack and yellowfin tunas indicated that tuna hearts normally function on the upper (i.e., flat) portion of their Starling curves, and therefore the increased filling time during bradycardia can not result in compensatory increases in SV.

In both skipjack and yellowfin tunas, blood pressures remained essentially unchanged at all levels of hypoxia because of a general vasoconstriction (i.e., increases in R_{total}, R_{branch}, and R_{system}). In yellowfin tuna, the percentage drop in $\dot{Q}$ was similar to the increases in R, suggesting that increases in R may have resulted from passive "collapse" of elastic blood vessels (Farrell 1984). However, reflex vasoactivity may also have occurred in which the reflex arc involved afferent information from baroreceptors and efferent information to branchial and systemic receptors as has been observed in other teleosts (Farrell 1984; Fritsche and Nilsson 1990). Direct hypoxic vasoconstriction of the gills may also occur in tunas as in other teleosts (Pettersson and Johansen 1982). Blood

pressure responses to hypoxia appear to be species specific, however, because increases (Holeton and Randall 1967b; Fritsche and Nilsson 1989, 1990), decreases (Farrell 1982; Peyraud-Waitzenegger Soulier 1989), and no change (Fritsche 1990) have been observed.

The dramatic reduction in cardiac power output in skipjack tuna at a P_iO_2 of 50 mmHg may be indicative of general myocardial failure. Nothing is known about the hypoxia tolerance of tuna myocardium, although our data suggest that it is relatively hypoxia intolerant. The high myoglobin levels observed in tuna hearts (Giovane et al. 1980) may, therefore, not function to maintain contractility in the face of low extracellular oxygen as has been suggested for other teleosts (Dreidzic 1983, 1988); rather, high myoglobin levels may serve to ensure adequate rates of oxygen delivery to the mitochondria, as they apparently do in tuna red muscle (Stevens 1972).

$\dot{T}O_2$ in skipjack tuna remained unchanged at a P_iO_2 of 130 mmHg, but doubled when P_iO_2 was reduced to 90 mmHg. In yellowfin tuna, $\dot{T}O_2$ increased by 23% at 130 mmHg and 122% at a P_iO_2 of 90 mmHg. In spiny dogfish, $\dot{T}O_2$ showed no change during hypoxia, but in European eel it increased 66% at a P_iO_2 of 40 mmHg. $\dot{D}O_2$ increased in both tuna species during hypoxia. The increase, which occurred at a higher P_iO_2 in yellowfin tuna (130 mmHg) than in skipjack tuna (90 mmHg), was a result of a decrease in the mean water to blood PO_2 gradient (ΔPg), which also appears to account for the entire change in $\dot{T}O_2$. The fact that an already high normoxic $\dot{T}O_2$ or $\dot{D}O_2$ was increased further is indicative of the excellent gas-exchange capabilities of the gills of tunas.

Increases in $\dot{T}O_2$ and $\dot{D}O_2$ imply an increase in the effective gill surface area, or a reduction in the diffusion distance between blood and water, or both. The effective area of the gills can be increased by lamellar recruitment, redistribution of blood flow, or distention of secondary lamellae due to increase in systolic or pulse pressures (Randall et al. 1967; Jones and Randall 1978; Booth 1979; Farrell 1980; Davie et al. 1982; Pettersson and Johansen 1982). At present it is not known whether lamellar recruitment can occur in tunas. An increase in blood pressure thought to promote lamellar recruitment (Booth 1979) was not seen in our study, however. Hypoxic bradycardia per se may also be partially responsible for the observed increases in $\dot{T}O_2$ and $\dot{D}O_2$. Abolition of the hypoxic bradycardia with atropine abated the increases observed in dogfish at a P_iO_2 of 50 mmHg (Taylor and Barrett 1985).

Another important determinant of gas transfer efficiency is the coupling of the respiratory and circulatory systems, as reflected in the $\dot{V}_g/\dot{Q}$ and the $\dot{V}_g/\dot{Q}$ conductance ratios. Both increased at all levels of hypoxia in yellowfin tuna, which actually become over-ventilated (i.e., $\dot{V}_g/\dot{Q}$ conductance ratio >1). Skipjack tuna, in contrast, showed significant increase in $\dot{V}_g/\dot{Q}$ at P_iO_2 levels of 90 and 50 mmHg, whereas $\dot{V}_g/\dot{Q}$ conductance ratio increased only at a P_iO_2 of 50 mmHg. The doubling of the $\dot{V}_g/\dot{Q}$ observed at a P_iO_2 of 50 mmHg is slightly less than the threefold increase recorded previously by Bushnell et al. (1990), but is much less than the 13-fold increase recorded in rainbow trout under similar conditions (Holeton and Randall 1967a; Wood and Shelton 1980).

$\dot{V}_g/\dot{Q}$ conductance did not rise more rapidly during hypoxia because the increase in the ventilatory oxygen delivery was offset for a time by the increase in the oxygen solubility of the blood. As the P_aO_2 fell during hypoxia, the blood began to function on the shoulder or steeper portion of the dissociation curve. As a consequence, large changes in oxygen content occurred in association with small changes in P_aO_2, and the solubility coefficient increased. The largest increase in $\dot{V}_g/\dot{Q}$ conductance occurred at a P_iO_2 of 50 mmHg when the concomitant reduction in $\dot{Q}$ reached its maximum.

E_w decreased 17% and 12% at a P_iO_2 of 130 mmHg in skipjack and yellowfin tunas, respectively. It declined 24% at a P_iO_2 of 90 mmHg in yellowfin tuna, but was unchanged at this level of hypoxia in skipjack tuna. This occurred in spite of predicted $\dot{V}_g$ being significantly increased at both levels of hypoxia in both species. In comparison, E_w in trout (Randall et al. 1967), dogfish (Short et al. 1979), and European eel (Peyraud-Waitzenegger and Soulier 1989) remains unchanged during hypoxia. E_b decreased during hypoxia (at 90 and 50 mmHg) in both skipjack and yellowfin tunas. Hypoxia (P_iO_2 80 mmHg) has been shown to decrease E_b in dogfish, an effect which was attributed to fact that arterial blood was functioning below the shoulder of the oxygen dissociation curve (Short et al. 1979). Based on P_aO_2s recorded during hypoxia and the blood oxygen dissociation curves shown in Fig. 1, it is obvious that a similar situation is occurring in both skipjack and yellowfin tunas during hypoxia.

O_2 delivery (i.e., $C_aO_2 \times \dot{Q}$) in yellowfin tuna remained at control levels until P_iO_2 had fallen to 50 mmHg, whereas it began to decline at a P_iO_2 of 90 mmHg in skipjack tuna. In addition to the early decline, the fall of O_2 delivery in skipjack tuna was steeper, as evidenced by the approximately 63% drop in O_2 delivery at a P_iO_2 of 50 mmHg compared to a 44% drop in yellowfin tuna at the same P_iO_2. The declines resulted from decreases in both C_aO_2 and $\dot{Q}$.

Other fish species show equivalent declines in O_2 delivery during hypoxia. Dogfish shark show a 43% decrease at a P_iO_2 of 80 mmHg (Short et al. 1979) and eel a 70% decline at a P_iO_2 of 40 mmHg (Peyraud-Waitzenegger and Soulier 1989). However, the decline in O_2 delivery occurs in the former solely because of a decline in C_aO_2, and in the latter because of a decline in C_aO_2 and $\dot{Q}$. Rainbow trout show no decreases in $\dot{Q}$ at P_iO_2 levels as low as 50 mmHg (Wood and Shelton 1980). O_2 delivery, therefore, is directly proportional to C_aO_2 which decreases by 13% at a P_iO_2 of 90 mmHg and 38% at a P_iO_2 of 50 mmHg (Boutilier et al. 1988). In contrast, winter flounder (*Pseudopleuronectes americanus*) make up for decreasing C_aO_2 by increasing $\dot{Q}$ such that there is no decline in O_2 delivery at P_iO_2 as low as 60 mmHg (Cech et al. 1977). The most relevant comparison is probably the responses of tunas and rainbow trout, since both are active, as opposed to benthic, species. C_aO_2 decreases with hypoxia are roughly equivalent in tunas

and trout (Boutilier et al. 1988) but, because hypoxic bradycardia is not accompanied by increases in SV in tunas as it is in trout, tunas are forced to use venous reserves to support $\dot{V}O_2$-body.

$\dot{V}O_2$-body in yellowfin tuna remained at near-normoxic levels through a P_iO_2 of 90 mmHg without a concurrent reduction in the venous reserve. However, skipjack tuna suffered a significant reduction in both C_aO_2 and $\dot{Q}$ during moderate hypoxia. Although they were also able to maintain $\dot{V}O_2$-body through a P_iO_2 of 90 mmHg, it was at the expense of the venous reserve, as C_vO_2 began to decrease at a P_iO_2 of 90 mmHg. At the most severe levels of hypoxia, neither tuna species were able to supply enough oxygen to meet demand, and $\dot{V}O_2$-body fell by about 27% in yellowfin tuna and 55% in skipjack tuna.

Of the species showing decreases in O_2 delivery (dogfish shark, eel, rainbow trout, skipjack tuna, and yellowfin tuna), only rainbow trout are able to fully counteract the decreases in O_2 delivery by reductions in C_vO_2 (i.e., venous O_2 reserves). $\dot{V}O_2$-total decreases 53% at a P_iO_2 of 40 mmHg in eels and 28% at a P_iO_2 of 80 mmHg in dogfish shark. $\dot{V}O_2$-body ($\dot{V}O_2$-total could not be measured at the P_iO_2 of 50 mmHg in tunas) decreased 55% and 27% in skipjack and yellowfin tunas, respectively, at a P_iO_2 of 50 mmHg. Rainbow trout show no change in $\dot{V}O_2$-total down to a P_iO_2 of <40 mmHg (Holeton and Randall 1967a).

In general, then, it appears that skipjack tuna are less hypoxia tolerant than yellowfin tuna. This conclusion is based primarily on the fact that O_2 delivery is maintained at normoxic levels through 90 mmHg in yellowfin tuna, whereas it is significantly reduced at that level in skipjack tuna. $\dot{V}O_2$-body of both species was maintained equally well through 90 mmHg; the collapse, when it occurred, was more severe in skipjack tuna. These data also agree with those of Graham et al. (1989) who found the oxygen uptake rate of swimming albacore declined with ambient oxygen levels below P_iO_2 of 100 mmHg.

Perhaps more important than when the collapse in O_2 delivery occurred, was how metabolism was maintained. Many of the cardiorespiratory adjustments important for maintaining arterial saturation were made at a higher P_iO_2 in yellowfin tuna than in skipjack tuna. Improvements in $\dot{V}_g/\dot{Q}$, $\dot{V}_g/\dot{Q}$ conductance, TO_2, and DO_2 were made at 130 mmHg in yellowfin tuna while they did not occur in skipjack tuna until P_iO_2 had fallen to 90 mmHg. Changes that were seen first (i.e., at higher P_iO_2) in skipjack tuna were generally detrimental in nature. The most important, in terms of O_2 delivery, was the reduction in $\dot{Q}$ occurring at a P_iO_2 of 90 mmHg in skipjack tuna and at a P_iO_2 of 50 mmHg in yellowfin tuna. Because O_2 delivery to the tissues was reduced as a consequence of the lowered $\dot{Q}$, skipjack tuna were forced to maintain $\dot{V}O_2$ by drawing upon the venous oxygen reserve. In spinally blocked (i.e., non-swimming) fish, this is not critically important as metabolic rate is stable. This, however, is not the case in the wild. A reduction in activity, the common response to hypoxia in many teleosts, is not available to free-swimming tunas because they are obligate ram ventilators. Also, a common response to hypoxia in tunas, as documented in Dizon (1977) and Bushnell and Brill (1991), is an increase in swimming speed. Skipjack tuna in moderate hypoxia will be able to meet this increased metabolic oxygen demand only by drawing further on their venous oxygen reserve. In light of the higher metabolic rate of skipjack tuna, higher minimum swimming speed, and larger increase in $\dot{V}O_2$ per increase in unit speed (Boggs 1984; Boggs and Kitchell 1991) compared to yellowfin tuna, the increased speed could not be supported aerobically for very long. However, yellowfin tuna should be able to maintain aerobic metabolism for a much longer period. The intolerance of skipjack tuna at this moderate level of hypoxia has been experimentally corroborated by Gooding et al. (1981) who found minimum oxygen tolerance level for skipjack tuna to be between 75 and 90 mmHg.

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