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HE ARTERIAL SYSTEM

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INTRODUCTION

The primary circulation of fishes follows the typical vertebrate attern in that a heart forces blood into a ventral aorta, which then ves off paired vessels (afferent branchials), which arch upward be- een successive gill clefts and rejoin (efferent branchials) to form the rsal aorta, which is typically paired anteriorly and a single vessel steriorly. The dorsal aorta distributes blood to the vascular beds in e tissues where exchange of metabolites, nutrients, and waste prod-

ucts occur. A unique feature of the piscine circulation is that the gas exchanger (gills) and systemic capillaries are perfused in series. Hence the piscine circulation is an arterial portal system in which the whole cardiac output ($\dot{Q}$) flows through two successive vascular beds of comparable resistance, and each influences the perfusion and hemodynamics of the other (Langille *et al.*, 1983). The nature and regulation of blood flow through the gills has been the subject of extensive review in the previous volume of this series (Volume XI), and further discussion would be repetitive and probably redundant.

A further embellishment of the arterial system exists in those fish having arterial retia interspersed between the gills and some tissue vascular beds. The descriptor *rete mirabile* refers to a blood vessel that divides into many small vessels and then rejoins to form a single vessel (Fange, 1983). Retial systems can be found in many teleosts and elasmobranchs. In most cases arterial and venous retia are closely apposed and form countercurrent systems that concentrate gases (swim bladder retia or choroid retia in the eye), metabolites (lactate in the swim bladder rete), or heat (heat-exchanger retia). The contribution of retia to vascular resistance is small since the retial vessels are resistances in parallel, although in those fishes with retial exchangers protecting large areas of the body (i.e., muscle) changes in blood flow distribution subserving thermoregulation may have a major influence on circulatory dynamics. Retia associated with swim bladders are well studied, and reviews by Blaxter and Tytler (1978) and Fange (1983) provide an in-depth discussion. Further, the anatomy and physiology of cephalic retial systems have been reviewed extensively by Block and Carey (1985) and Block (1991), whereas anatomical descriptions of muscle and visceral retia date back 150 years and are summarized in a previous volume of this series (Volume VII) and in reviews by Carey (1982) and Satchell (1991). Consequently, our discussion will focus on the physiology of countercurrent retial systems that trap heat (heat exchange retial systems or heat exchangers), concentrating on contributions made in recent years.

The primary circulation is remarkably constant in general form in cyclostomes, elasmobranchs, and teleosts. However, major modifications of the posterior branchial arteries occur in air-breathing fishes. These modifications have been reviewed by, for instance, Fishman *et al.* (1989) and Satchell (1975) and will not be dealt with in the present account. The primary circulation is, in many vascular regions, paralleled by a secondary system of arteries, capillaries, and veins which is discussed in Chapter 4 by Steffensen and Lomholt. The role of the conus and bulbus arteriosus, two structures that are found within the

pericardium in all fishes except cyclostomes, is discussed in this chapter. Regardless of whether the origin of the conus or bulbus arteriosus is cardiac or arterial both of these structures have a marked influence on pressure-flow relationships in the arterial system and consequently, that influence is described here.

In this chapter circulatory dynamics and regulation will be emphasized. Consequently, we will look at the "nature" of the fluid flowing in the vessels and effect of blood viscosity on cardiac energetics. The "windkessel" function of the major arteries is also of great importance because of influences on both cardiac work and central and peripheral blood flow. Arterial elasticity is the major contributor to the "windkessel" by storing blood in systole, when the walls are stretched by the blood pressure, and maintaining blood flow in diastole by their passive recoil. Hence, the arteries are more than just conduits for blood from the heart to the peripheral blood vessels. Control of flow distribution resides in the arterioles that terminate the arterial system and their role in regulating blood flow in response to exercise, hypoxia, and for thermoregulation is discussed in the final section.

II. PHYSICAL FACTORS AFFECTING BLOOD FLOW

Blood flow in major blood vessels of fishes will be laminar with turbulence occurring, if at all, only during peak systolic ejection in the ventral aorta of the largest animals (Langille *et al.* 1983). Hence if flow is laminar then the pressure gradient for steady flow can be obtained from Poiseuille's Law

$$F = \frac{(P_i - P_e)\,\pi\, r^4}{8\, L\, \mu} \tag{1}$$

where F = flow ($cm^3 \cdot S^{-1}$), P_i = pressure at the upstream point (dynes $\cdot$ cm^{-2}), P_e = pressure at the downstream point (dynes $\cdot$ cm^{-2}), L = distance between the two measuring points (cm), r = radius of the vessel (cm), and μ = viscosity (Poise). Hence, there will be a pressure drop whenever flow occurs, energy being dissipated in overcoming the "inner friction" or viscosity of the blood.

Both pressure and flow in the major arteries are pulsatile, and due to inertia within the blood, the flow amplitude may no longer be related to the pressure gradient, so that Poiseuille's Law will not apply.

The deviation from Poiseuille's Law and the extent of the phase lag is determined by a nondimensional constant α where

$$\alpha = r\sqrt{\frac{2\pi f\rho}{\mu}} \qquad (2)$$

where r = radius of the vessel, ρ density (gm · cm^{-3}), and f = frequency (Hz). If α is less than unity, then instantaneous flow rate will vary by less than 2% from that predicted by Poiseuille's Law, and the phase difference between pressure gradient and flow will be less than 10%. Assuming viscosity to be constant, then the same α may arise from a low frequency in a wide tube or a high frequency in a narrow tube. The small heart and narrow blood vessels of fish, combined with low frequencies of contraction, mean that α will be small in the vast majority of species.

This discussion highlights the importance of viscosity to the dynamics of blood flow in fishes. Blood viscosity is related to the fraction of red blood cells in the blood, so from Poiseuille's Law [Eq. (1)], vascular resistance is also related to hematocrit (Hct). Furthermore, since the heart must generate sufficient blood pressure to overcome vascular resistance, blood pressure is in turn linked to Hct. The complex and, as yet, incompletely understood interrelationships between blood viscosity, Hct, cardiac performance, and O_2 transport are represented globally by the optimal hematocrit concept.

Blood viscosity increases with both increasing Hct and decreasing temperature (Fig. 1A and B). In addition, blood is a non-Newtonian fluid, that is, its viscosity is shear-rate dependent (Fig. 1C) and increases exponentially at low shear rates. Consequently, blood viscosity will be highest in fishes that have high Hcts and low blood velocities and live in cold water. The lowest viscosities will be found in warm-water fish with low hematocrits and high flows. The viscosity of plasma is also shear-rate dependent (Fig. 1D) and, in some species, can contribute over 50% to the whole blood viscosity, even at high temperatures. Interestingly, in rainbow trout the viscosity of plasma is independent of shear rate (Fletcher and Haedrich, 1987).

The shear-rate dependency of fish blood has been examined in a number of species, and it appears that it is less in active fishes (Graham and Fletcher, 1985; Fletcher and Haedrich, 1987; Wells and Baldwin, 1990). However, at very high shear rates these species differences are considerably reduced. It is important to note that the major site of vascular resistance is the arterioles, and the shear-rate value in arterioles will have the greatest influence on the overall vascular resistance. However, shear rate is inversely proportional to radius of the

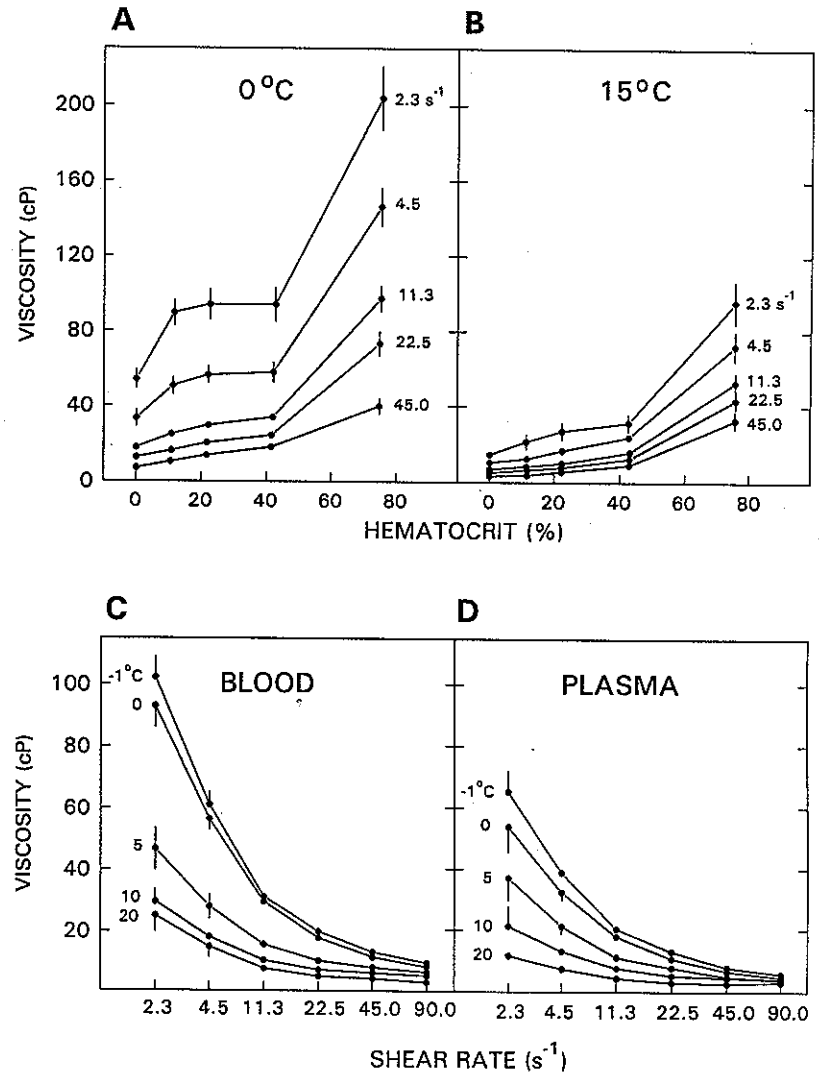

Fig. 1. The effect of hematocrit on viscosity at 0°C (A) and 15°C (B) at shear rates indicated to the immediate right of each line describing a viscosity–hematocrit relation. Average hematocrits and mean cellular hemoglobin concentrations, respectively, are as follows: 0% (plasma); 11.5% and 24.8; 22.3% and 20.6; 43.1% and 26.3; and 75.0% and 25.7. The effect of temperature (indicated in °C to the left of each line) and shear rate on the viscosity of whole blood (C) and plasma (D). For C and D, total protein concentration, 4.32 ± 0.12 g · 100 ml^{-1}; average Hb concentration, 4.93 ± 0.18 g · 100 ml^{-1}, average hematocrit, 22.26 ± 0.20%. Redrawn from Graham and Fletcher (1983).

blood vessel, so shear rate in arterioles would be expected to be high (Graham and Fletcher, 1983).

The optimal hematocrit concept recognizes that, in terms of cardiac work per unit of internal O_2 convection, there is a trade-off between increasing Hct to increase the O_2-carrying capacity of the blood and decreasing the Hct to reduce the work of the heart generating blood pressure. The upper limit for Hct may, therefore, be set by the species-specific maximal blood pressure that the heart can develop (see Chapter 3), and the exceptional increase in blood viscosity at Hct values around 50–80%. Conversely, the lower limit for Hct may be set by the maximum $\dot{Q}$ of the heart. One would further expect these limits to be affected by temperature, not only because of the effect of Hct on blood viscosity, but also because the demand for internal O_2 convection is temperature dependent.

Wells and co-workers (Baldwin and Wells, 1990; Wells and Baldwin, 1990; Wells and Weber, 1991) examined the optimal hematocrit concept in a number of fish species by measuring blood viscosity with a cone-plate viscometer and relating this to the O_2-transport capacity (OTC) as calculated by

$$\mathrm{OTC} = \frac{1.3\,[\mathrm{Hb}]}{\mu} \qquad (3)$$

where [Hb] = hemoglobin concentration. Reasonably discreet peaks for the calculated optimal Hct were obtained and these ranged among various species from 20 to 40%. The range for calculated optimal hematocrit clearly encompasses measured Hct values of many teleosts and elasmobranchs. However, for individual species examined, there were discrepancies between the calculated optimal Hct and the measured Hct. For example, calculated optimal Hct values were much higher than Hct values actually measured in rainbow trout (*Oncorhynchus mykiss*) and a variety of tropical elasmobranchs. Furthermore, in tropical reef teleosts, measured Hct was greater than the calculated optimum in an active species, but measured values were lower than the calculated optimum of two less-active species (Wells and Baldwin, 1990).

A number of reasons can be advanced for these equivocal findings in respect to the validity of the optimal hematocrit concept. On the one hand, the plasma may or may not make a significant contribution to measured viscosity whereas, on the other hand, variations in erythrocyte size and rigidity of the red blood cell membranes may decrease or increase whole blood viscosity. Also, the concentration of hemoglobin

packed into each blood cell will affect differentially oxygen transport capability even when Hcts are similar. Furthermore, red blood cells have functions other than O_2 transport and the measured Hct is likely to reflect compromises with these functions. Finally, an optimal Hct for O_2 transport may be more relevant to a fish's physiology when internal O_2 convection is pushed near its limit, i.e., near maximum $\dot{V}O_2$. For example, prolonged and especially burst swimming are accompanied by increased Hct as a result of splenic contraction releasing stored red blood cells. The most extreme example of this is observed in the red-blooded Antarctic fish, *Pagothenia borchgrevinki*, in which Hct almost doubles when they are stressed (Davison *et al.*, 1988; Franklin *et al.*, 1991). These fish live in frigid waters and have low Hct at rest, so there will be an impact of the increased Hct on blood viscosity. In view of these considerations, it is more likely that optimal Hct spans a broad range and attempts to define *the* optimal Hct should recognize the importance of many other factors.

Antarctic fishes are an extreme example of how blood viscosity influences overall cardiovascular design. Blood viscosity is exceptionally high around 0°C, and Antarctic fishes living exclusively at temperatures of −1.7° to 2.0°C have either fewer or no red blood cells compared with temperate teleost species (see MacDonald *et al.*, 1987). This appears to be an effective strategy to reduce blood viscosity, since the viscosity of the blood of hemoglobin-free Chaenichthyids at 0°C (2.9–3.9 centipoise; Hemmingsen and Douglas, 1972; Wells *et al.*, 1990) is comparable to that at much higher temperatures in temperate water species of fish. While low or zero Hcts reduce blood viscosity, they also reduce blood O_2 carrying capacity. To compensate, both hemoglobin-free and red-blooded Antarctic fishes have a high $\dot{Q}$ for internal O_2 convection. The immediate advantage of this strategy is not clear in terms of cardiac work because high $\dot{Q}$ values increase cardiac work, thereby negating some or all of the benefits accrued by reducing blood viscosity. Further, since most cardiac work is done in generating tension within the muscle and not as external work, then Laplacian relationships dictate that a large-volume, low-frequency pump will operate at a low mechanical efficiency. However, the low blood pressures of Antarctic fishes will tend to offset the effects of a large ventricular volume on wall tension. Perhaps the most important advantage of the low Hct and high $\dot{Q}$ strategy for internal O_2 convection in Antarctic fishes is to minimize the shear-rate effect on blood viscosity (Wells *et al.*, 1990).

Considerable modifications of the cardiovascular system of Antarctic fishes are necessitated by having a high $\dot{Q}$. To achieve high $\dot{Q}$

values, the ventricle is enlarged and accommodates an exceptional stroke volume (e.g., 10 ml · kg^{-1} in *Chionodraco hamatus*; Tota *et al.*, 1991). Because of the high resting stroke volume, large increases in stroke volume are unlikely so heart rate rather than stroke volume increases with swimming. The increase in heart rate during exercise is brought about by a marked reduction in the high resting vagal tonus (Axelsson *et al.*, 1992). Further, stroke volume is particularly sensitive to increases in diastolic output pressure in *Chionodraco hamatus* (Tota *et al.*, 1991), indicating a limited capacity for homeometric regulation. Antarctic fishes also have large capillaries which, combined with a low Hct, yields an unusually low vascular resistance (Axelsson *et al.*, 1992). One disadvantage of low Hct blood is that venous O_2 content will be low. Consequently, myocardial O_2 demand will be a major proportion of the O_2 normally contained in venous blood (see Chapter 3). The long residence time of blood in the heart as a result of slow heart rates will favor O_2 extraction, whereas the low dP/dt due to low arterial blood pressures, will be advantageous in terms of myocardial O_2 demand. However, low venous P_{O_2} in hemoglobin-free fishes may seriously limit myocardial O_2 delivery and thereby limit exercise capability.

III. ROLES OF THE CONUS AND BULBUS ARTERIOSUS

For efficient gas exchange, water flow over the gills and blood flow through the gills should be continuous. Continuous blood flow may be achieved by the elastic recoil of blood vessel walls stretched during systolic ejection. The best estimate of the extent of this "windkessel" function of the ventral aortic system is from flow measurements made on the main vessel either just outside the conus or bulbus or between pairs of branchial arteries. Recordings from these situations show major differences between flow patterns in cyclostomes and elasmobranchs on the one hand and teleosts on the other, although these flow patterns may not be truly indicative of gill blood flow. In the ventral aorta of elasmobranchs, flow recorded either just outside the pericardium or between the second and third pairs of branchial arteries, stops or even reverses at some stage during diastole (Fig. 2; Satchell and Jones, 1967; Butler and Taylor, 1975; Short *et al.*, 1977; Metcalfe and Butler, 1982; Abel *et al.*, 1987). In contrast, flow in the teleost ventral aorta is usually continuous during disatole, a testament to the extreme capacity and compliance of the bulbus arteriosus (Fig. 5; Johansen, 1962; Stevens *et al.*, 1972; Farrell, 1981; Hipkins, 1985; Axelsson *et al.*, 1989; Jones *et al.*, 1992a).

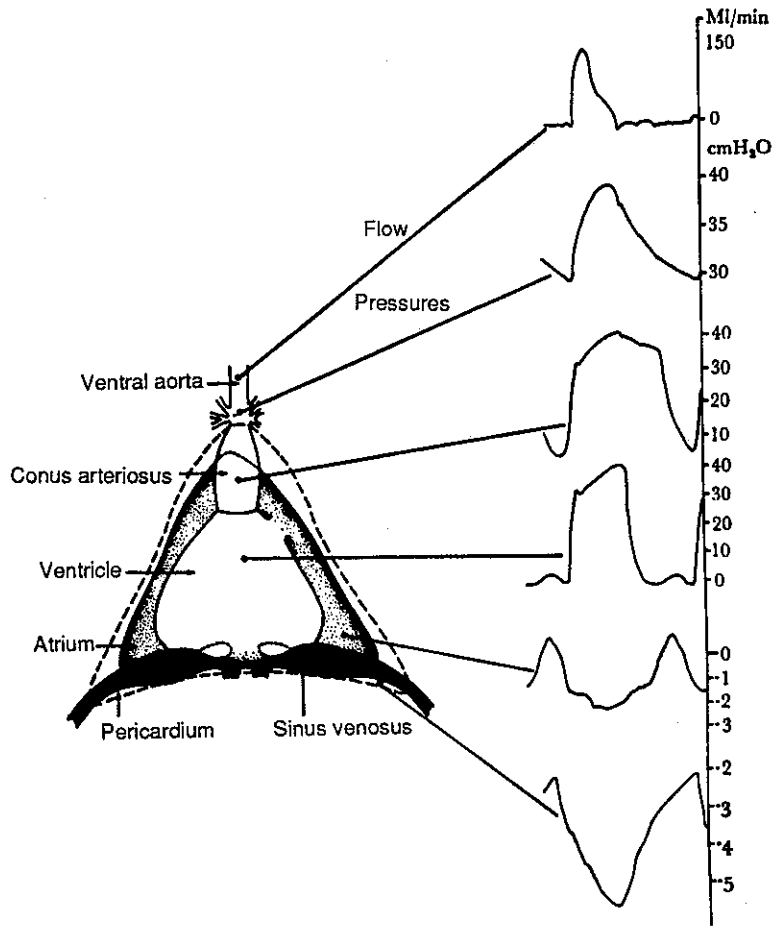

Fig. 2. Schematic representation of heart structure and pressure within the pericardium, atrium, ventricle, and conus, aligned with ventral aortic pressure and flow of the Port Jackson shark, *Heteredontus portusjacksoni*. Not all pressures were recorded simultaneously. Figure from Satchell (1971), with permission.

A. Role of the Conus Arteriosus

At present, the role of the elasmobranch conus arteriosus is obscure. Nevertheless, many aspects of its function are clear. There is no doubt that ventricular systole is prolonged by contraction of the conus. However, the conal contribution to flow is slight (Johansen *et al.*, 1966) since its volume is so much smaller than that of the ventricle. Ideas that conal contraction can provide an extra, active addition of propulsive

power to that released by the ventricle (Johansen, 1965) have never been substantiated. The contrary idea that the conus could "depulsate" or reduce the peak systolic pressure also seems a doubtful proposition. "Depulsation" results from the elasticity of the central arterial system and since the conus is contracting during ventricular ejection (March *et al.*, 1962; Sudak, 1965), its walls will be stiff and the conal contribution to overall elasticity will be small.

Conal contraction ensures the competency of the lower (proximal) tiers of conal valves. In the isolated, relaxed conus of *Heterodontus portusjacksoni* only the top (distal) valves remain competent against back flow. The middle or lower tiers are not large enough to bridge the relaxed conus and must, therefore, depend on conal contraction to reduce conal diameter and bring them into contact with one another (Satchell and Jones, 1967). Satchell and Jones (1967) envisage a "peristaltic" wave moving up the conus and closing each tier of valves in turn. Certainly, the conus contracts with a time delay with respect to the ventricle, which allows the conus to distend with the initial ventricular ejection so that outflow is not impeded. Nodal tissue is present at the ventricular–conal junction and the electrocardiogram (ECG) shows a delay between deflections caused by contraction of the ventricle and conus (Satchell, 1991). Furthermore, the conduction velocity of the conal depolarization wave is some 20 times slower than the ventricular wave of depolarization (Tebécis, 1967), slow enough in fact (2–4 cm · sec^{-1}) to provide the required to proximal distal wave of contraction.

Satchell and Jones (1967) envisaged the primary role of the conus as postponing valve closure until the nadir of pericardial negative pressure, caused by ventricular ejection, was passed, reducing ventral aortic backflow. However, in their traces, marked aortic backflow was associated with closure of the lower not the upper set of valves (Fig. 2). It seems strange that this backflow would not also passively shut the upper conal valves, which are competent in relaxed preparations. Furthermore, there seems no intrinsic reason why negative external pressures should have more effect on valve competency than positive internal pressures. In fact, Satchell and Jones (1967) were unable to control arterial and pericardial pressures independently, and the only conclusion that can be drawn from their experiments is that aortic backflow increases when transmural pressures increase. Finally, pericardial pressures in Horn sharks (*Heterodontus francisci*) are not always negative, indicating that further research on the outflow dynamics of the elasmobranch heart should prove worthwhile (Abel *et al.*, 1986).

B. Role of the Bulbus Arteriosus

Depulsation and prolongation of aortic flow during diastole are undoubted functions of the teleostean bulbus. The bulbus of rainbow trout, yellowfin tuna (*Thunnus albacares*), and carp (*Cyprinus carpio*) are extremely elastic, with those of the latter two species being exceptionally extensible over pressure ranges to which the vessels are subjected *in vivo* (Fig. 3A; Licht and Harris, 1973; Priede, 1976; Jones *et al.*, 1992b). Licht and Harris (1973) report that the bulbus of carp is 30 times more distensible than the human aorta over a pressure range of 7.3 to 33 mm Hg. One of the reasons for the high distensibility may be

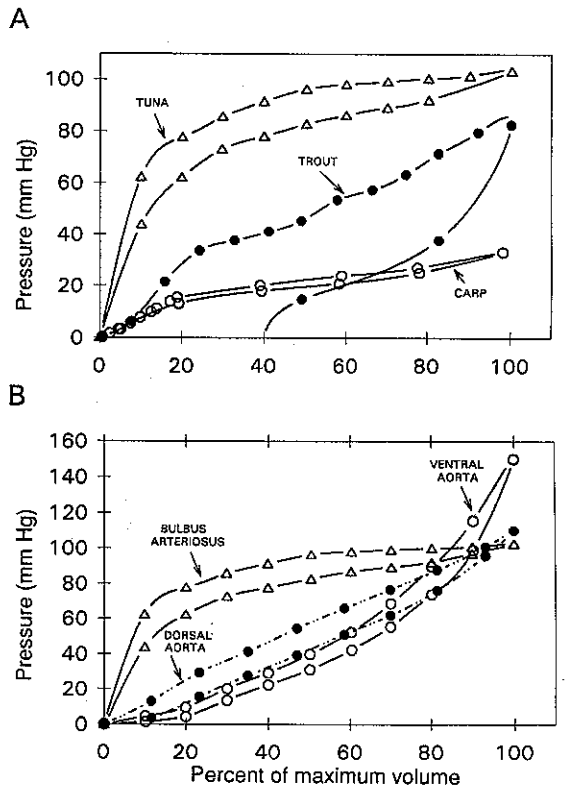

Fig. 3. (A) Quasi-static pressure–volume curves for the bulbus arteriosus of tuna (Jones *et al.*, 1992b), trout (Priede, 1976), and carp (Licht and Harris, 1973). Redrawn from published traces, scaling the ordinate (volume) to 100% at maximal volume. (B) Quasi-static pressure–volume curves for the bulbus arteriosus and dorsal and ventral aortas of tuna. Ordinate scaled to 100% at maximal volume.

that teleost elastin has fewer cross-links than that of mammals, so it is more elastic (Serafini-Fracassini *et al.*, 1978). Further, the "higgledy-piggledy" arrangement of elastic fibrils in the bulbus may allow the fibers to slide between one another, allowing for greater distension (Benjamin *et al.*, 1983, see also Chapter 3 of this volume). Finally, the elastin : collagen ratio usually gives a good indication of blood-vessel stiffness. The larger the ratio, the more elastic the tissue. In the trout bulbus this ratio is about 14 (Serafini-Fracassini *et al.*, 1978), compared with 1.5 in the proximal mammalian aorta (McDonald, 1974), and 0.4 in the frog aorta (Gibbons and Shadwick, 1991a).

Hysteresis is the proportion of energy lost through viscous processes during inflation and deflation and is evidenced by a pressure difference occurring at similar volumes during the increasing and decreasing limbs of a volume cycle (Fig. 3A). On a pressure–volume loop, the area within the loop as a proportion of the area under the inflation part of the cycle, is a measure of hysteresis. For the carp, hysteresis is negligible, whereas for the rainbow trout, it is substantial (Fig. 3A). The reason for this difference is obscure. Vascular smooth muscle is the main contributor to hysteresis in the mammalian arterial wall (Dobrin, 1978), and it is plentiful in the bulbi of both carp and rainbow trout. Furthermore, smooth muscle cells in bulbi of both rainbow trout and carp are firmly joined to one another by desmosomes. Lack of hysteresis in carp probably results from insufficient conditioning of the vessel. The first 4 to 6 volume cycles do not give identical results, and this probably obscured the hysteresis since Licht and Harris (1973) averaged values of 51 volume cycles done on bulbi from six fish.

Bulbi are round, swollen proximally, and tapering distally to meet the ventral aorta (Santer, 1985). The functional significance of differing bulbar shapes is unknown, although Priede (1976) has discussed some hydromechanical advantages and disadvantages. Basically the bulbus expands in preference to the ventral aorta and this seems to be achieved by the bulbus having a much larger diameter and different wall construction. Dead space within the expanded bulb is reduced by trabeculae in rainbow trout, yellowfin tuna (*Thunnus albacares*), turbot (*Scophthalmus maximum*), and other less phylogenetically advanced teleosts. The trabeculae may be irregular and anastomosing (Santer, 1985) or may be regularly disposed into longitudinal and radial elements as in rainbow trout and tuna species (Priede, 1976; Jones *et al.*, 1992b). The longitudinal and radial elements provide for strain equalization within the bulbar wall. When the bulbus is expanded there is far more strain on the inner than the outer bulbar wall,

but the inner longitudinal elements do not follow the outer wall during expansion (see Chapter 3). The longitudinal elements arch outward and support the outer wall by means of the obliquely inserted radial elements (Fig. 4) (Priede, 1976). A smooth bulbar lumen represents the apogee of teleostean phylogenetic advancement implying more differentiation in strain-limiting wall structure than in that of the conus of more primitive forms. Cyclostomes with a smooth inner wall to the conus appear to present an exception. However, the conus in cyclostomes has features that suggest it is an isolated evolutionary experiment rather than a predecessor of the conus or bulbus in the elasmobranchs and teleosts.

Collagen, which is relatively inexpansible, is usually confined to the pericardial layer of the wall and will check bulbar expansion. Hence, collagen and elastin form a two-phase system in the bulbus similar to their role in blood vessels. Further, although it is the pericar-

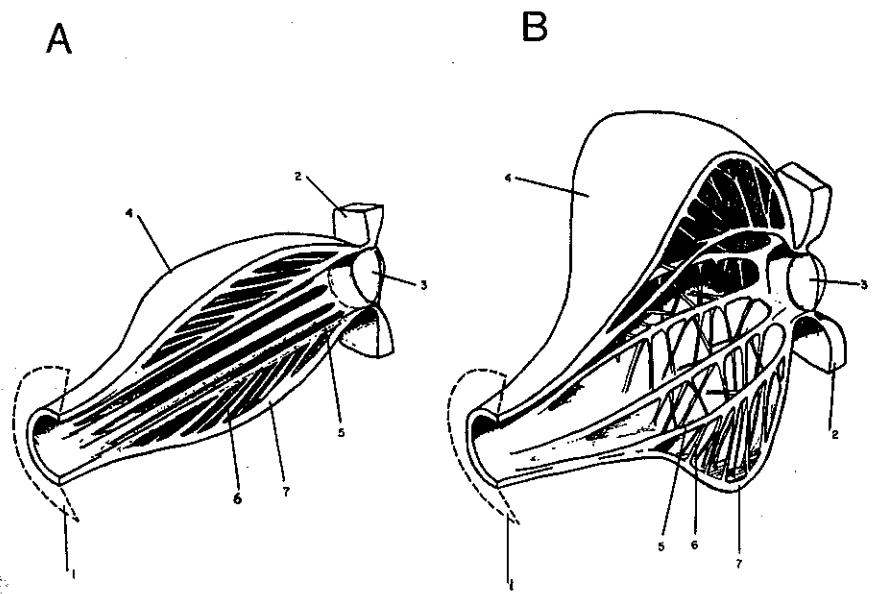

Fig. 4. Isometric projection diagrams of the bulbus arteriosus as reconstructed in the contracted (A) and expanded (B) states. 1, Anterior wall of pericardial cavity; 2, wall of ventricle; 3, ventriculo-bulbar valve; 4, adventitia composed of pericardial elements and epicardium; 5, longitudinal elements; 6, radial elements; 7, compact layer of media with predominantly circumferential fibers. Caption and figure from Priede (1976), with permission.

dial confinement of the bulbus that allows expansion in the first place, ultimately the pericardial wall itself will limit bulbar enlargement.

The discussion so far has been restricted to circumferential and not longitudinal enlargement of the bulbus. The attachment of the pericardium to the distal end of the bulbus would appear to constrain bulbar lengthening *in vivo*. However, there is nothing to stop an elongating bulbus from pushing the ventricle backward. Hence, it is no surprise that collagen fibers in the outer wall may be oriented to resist longitudinal much more than circumferential stretch. For instance, in bigeye tuna (*Thunnus obesus*), the outer wall is twice as extensible in the circumferential as in the longitudinal direction (Jones *et al.*, 1992b).

Concentration of the major portion of arterial distensibility just outside the heart is more effective in reducing peak systolic pressure than a similar compliance distributed throughout the arterial system (Campbell *et al.*, 1981; Jones, 1991). On the other hand, diastolic pressures will be raised equally regardless of the location of the compliance. In other words, the heart "sees" all of the compliance added on the outflow tract, whereas compliance added at other locations is effectively hidden from the heart during systolic ejection.

Hence, the bulbar "windkessel" not only creates steady flow in the aorta, and presumably enhances gas exchange at the gills, but also reduces peak systolic pressures (Fig. 5). As the majority of cardiac O_2 uptake is utilized for developing tension, generating the blood pressure, any reduction in cardiac tension will bring about an improvement in cardiac efficiency. Efficiency is the quotient of external work divided by total energy transformed and, like that of most biological systems, the efficiency of the fish heart is low (Farrell *et al.*, 1985; Farrell and Milligan, 1986). Hence, a reduction in peak systolic pressure will have a marked impact on the efficiency of heart function, much more so than improving external work performance, since so little of cardiac oxygen consumption appears as external work. Nevertheless, compromise must remain an important feature of cardiovascular function. For instance, increases in input frequency to the "windkessel" will further reduce pulsatility around the mean pressure, and promote continuous flow, but energetic savings will be compromised by the increase in the proportion of time that the cardiac muscle is generating tension.

Priede (1976) calculated that 25% of the stroke volume in a trout could be held in the bulbus. Pressure–volume loops suggest that this may be an underestimate. For instance, in 1- to 2-kg tuna, stroke volumes vary from 0.3 to 1.3 ml · kg^{-1} (Bushnell and Jones, 1992) and the bulbus can contain up to 0.5 to 1 ml · kg^{-1} at normal ventral aortic

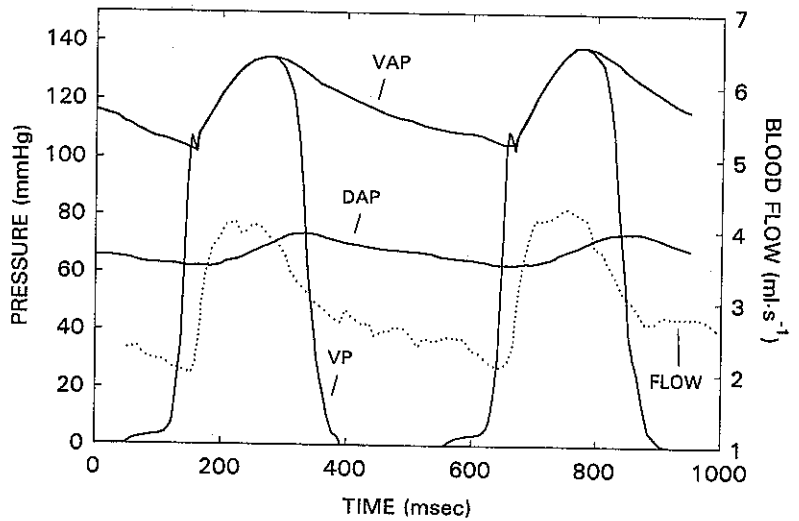

Fig. 5. Pressures in the ventricle (VP), ventral (VAP) and dorsal (DAP) aortas and ventral aortic flow (FLOW) of yellowfin tuna (*Thunnus albacares*). Superimposed from original traces in Jones *et al.* (1992a).

blood pressures when examined using quasi-static pressure–volume loops (Fig. 3A and B; Jones *et al.*, 1992b). However, *in vivo*, expansion of the bulbus will be limited owing to its position within the pericardium. Furthermore, there is a lot of smooth muscle in the bulbar wall, individual cells being tightly bound together by desmosomes. The muscle cells are innervated by autonomic nerves at their outer ends, not laterally, and low-resistance gap junctions connect the cells in a cablelike electrical syncytium (Watson and Cobb, 1979). Hence, contraction or relaxation of smooth muscle will considerably reduce the capacity and compliance of the bulbus. Acetylcholine causes contraction of spiral strips of trout bulbus (Klaverkamp and Dyer, 1974) and considerably stiffens the wall of the isolated bulbus and ventral aorta in dynamic pressure–volume tests (Farrell, 1979). On the other hand, adrenergic agents and hypoxic exposure cause an increase in compliance in dynamic pressure–volume tests (Farrell, 1979). *In vivo*, these changes would tend to increase depulsation by accentuating the reduction in systolic pressure.

No ventral aortic back-flow is measured during valve closure in fish with a bulbus arteriosus (Fig. 5). It is possible that backflow occurs in the region of the ventriculobulbar valves but, because of the com-

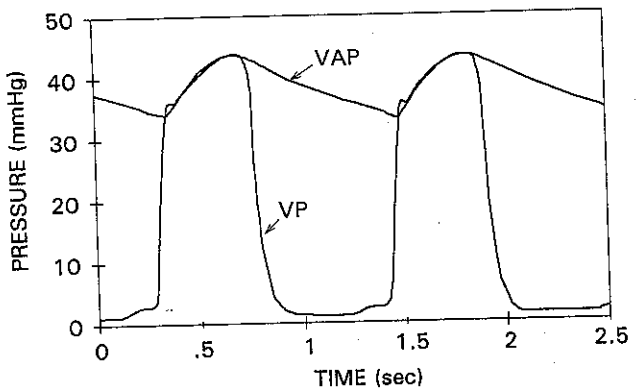

Fig. 6. Superimposed traces of ventricular (VP) and ventral aortic (VAP) pressures in the carp (*Cyprinus carpio*) from original traces in Ngan *et al.* (1974).

pliance of the bulbus, backflow is not transmitted to the ventral aorta. During ventricular ejection, vortices may be set up between the valve cusp and the wall so that as ventricular output slows, the pressure difference between the vortex and decelerating blood is sufficient to close the ventriculobulbar valves. This idea, first attributed to Leonardo da Vinci, seems to hold for mammals, although it is doubtful that this mechanism will apply in fish owing to much lower rates of acceleration and deceleration during ventricular ejection.

A notable feature of high-fidelity pressure and flow recordings in teleosts (i.e., carp, Ngan *et al.*, 1974; yellowfin tuna, Jones *et al.*, 1992a) is that ventricular pressure generation rises throughout the ejection phase, and aortic and ventricular pressures part company, indicating valve closure, at or soon after peak pressure (Fig. 5 and 6). This is unusual in vertebrates, for valve closure usually occurs on the descending limb of the ventricular pressure profile. In fact, the situation in teleosts is suggestive of active processes being involved in valve closure. In some teleosts, there is a muscular ring of tissue at the junction between the bulbus and ventricle and its activation could bring about valve closure. The muscular ring may represent a vestige of the more phylogenetically ancient conus. Alternatively, it may be that the valve is actively held open during ejection and closes passively when the ventricular muscle relaxes. Sanchez-Quintana and Hurle (1987) have described the insertion of cardiac muscle fibers on the bulboventricular ring that could actively open the valve. These are superficial fibers in swordfish (*Xiphias gladius*) and deep fibers in

Atlantic bluefin tuna (*Thunnus thynnus*). Nonpassive cardiac valving is a common feature among the vertebrates, and a careful hemodynamic analysis of the cardiac outflow tract in teleosts should be most rewarding.

IV. PRESSURE AND FLOW RELATIONSHIPS IN THE VENTRAL AND DORSAL AORTAS

Blood vessel structure appears to be similar from cyclostomes to the most advanced teleosts. The wall consists of three layers or tunics. The luminal tunic (intima) is endothelial, and the endothelium may be drawn into long spindle-shaped folds that run parallel to the aortic axis (Leknes, 1985). The middle tunic (media) is largely smooth muscle with elastic fibers interspersed between the smooth muscle cells. The outer tunic (adventitia) is mostly collagen. The collagenous layer is believed to be much thicker in the dorsal than in the ventral aorta (Lander, 1964).

In the ventral aorta the smooth muscle cells are often loosely arranged with respect to the long axis of the vessel. However, in some teleosts smooth muscle is more organized, and in trout, for example, it is arranged both circumferentially and longitudinally (Serafini-Fracassini *et al.*, 1978).

Elastic fibrils are often arranged randomly although grouping into distinct laminae, resembling amorphous elastin of mammalian vessels, occurs in carp, eel, rainbow trout, and yellowfin and skipjack (*Katsuwonus pelamis*) tuna (Dornesco and Santa, 1963; Isokawa *et al.*, 1988, 1990; Jones *et al.*, 1992b). Large wall strain due to the continuous pounding of high blood pressures may be a prerequisite for laminae formation, which explains their absence in fishes with low blood pressures. However, no determinations have been made of either the requisite wall strain or the period over which strain must be exerted to promote laminae formation. Leknes (1986) reports that no laminae occur up to 21 days posthatching in the guppy (*Poecilia reticulata*), although some alignment of fibrils is observed.

A unique feature of cyclostome blood vessels (and the cyclostome conus) is that the fibrillar network in the media does not consist of elastin (Wright, 1984; Isokawa *et al.*, 1989). In lamprey, the ventral aorta has a thicker wall than the hagfish, although there is a much higher collagen content in the wall of the latter (Wright, 1984). Interestingly, the New Zealand hagfish (*Eptatretus cirrhatus*) has a paired

ventral aorta, whereas *Myxine glutinosa* has a single ventral aorta (Axelsson *et al.*, 1990; Forster *et al.*, 1992).

In elasmobranchs, elasticity measurements indicate that the ventral aorta is some six times more distensible than the dorsal aorta (Lander, 1964). However, this does not appear to be the case in at least one teleost, the yellowfin tuna (Jones *et al.*, 1992b). Pressure–volume loops show that the compliance of the ventral or dorsal aortae are similar. In the ventral aorta, distensibility is high up to the physiological pressure range but above that range, the pressure–volume curve increases sharply as the wall becomes stiffer (Fig. 3B, Jones *et al.*, 1992b). The shape of this curve is generally attributed to the interaction of elastin and collagen, in that the highly distensible portion is dominated by the contribution of elastin, whereas the stiff portion is predominantly the result of collagen fibers. In contrast, pressure–volume loops for the dorsal aorta are nearly linear (Fig. 3B) over the range of pressures experienced physiologically. Both loops show hysteresis, which is more marked for the dorsal than ventral aorta. Hence, viscous losses in each cycle are higher for the dorsal aorta.

Pressure and flow relationships in arteries are usually analyzed using an analog (pressure = flow × resistance) of Ohm's law for direct current electrical circuits. However, both pressure and flow are pulsatile, and hemodynamic analysis is better served by considering not only the mean values of pressure and flow but also the sine wave harmonics of the pulsatile waveforms. The harmonics are obtained from a Fourier analysis of the original pressure and flow waveforms (McDonald, 1974; Jones, 1991). The quotient of dividing oscillatory pressure by oscillatory flow is termed impedance. Further, it can be seen from traces presented in Fig. 5 (Jones *et al.*, 1992a) that in the ventral aorta, flow leads pressure. Consequently, the phase angle between any pair of harmonics of pressure and flow of the same frequency will usually be negative.

Impedance modulus and phase differences for pressures and flows in the ventral aorta of a yellowfin tuna are shown in Fig. 7 (Jones *et al.*, 1992a). It is noteworthy that the shape of the modulus and phase curves is unchanged by a doubling of heart rate. It can be seen from the modulus curve that zero-frequency impedance is some 3 to 10 times greater than that for the first and succeeding harmonics. What this means from the fish's point of view is that the energetic costs of pulsatile pumping are much lower than they would be if impedance remained at steady flow values. The phase curves in Fig. 7 show that flow leads pressure at all pulsatile harmonics, which is a characteristic feature of a "windkessel" system. During the past 20 years, electrical,

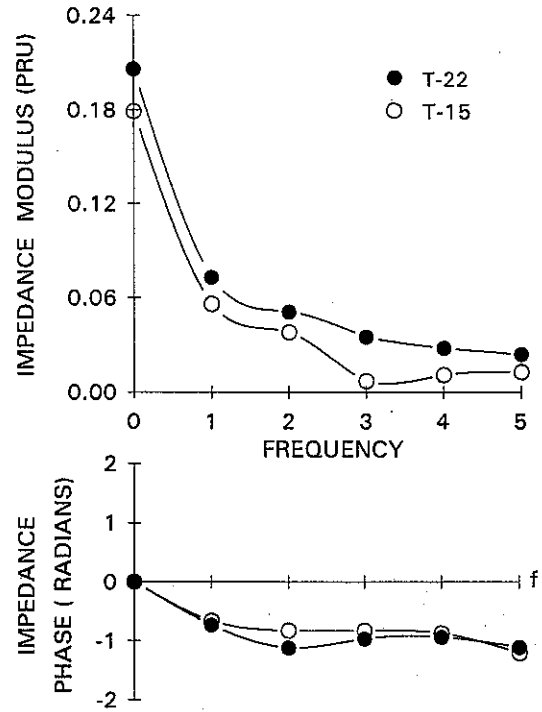

Fig. 7. Input impedance modulus and phase of the ventral aortic circulation in a yellowfin tuna (*Thunnus albacares*). For T-22 heart rate was 120 min^{-1} and for T-15 heart rate was 58 · min^{-1}. Figure and caption from Jones *et al.* (1992a).

mechanical, and mathematical models have been used to supplement recordings of pressure and flow in fish to better understand and attempt to explain pressure and flow relationships in the ventral and dorsal aortae. Models, consisting of two sets of compliant and resistive elements coupled in series (Satchell, 1971; Jones *et al.*, 1974) have been analyzed in great detail by Langille *et al.* (1983) and consequently will not be dealt with here.

The analysis of vascular impedance shown in Fig. 7 was limited by only being taken to five harmonics but, even at heart rates in excess of 2 Hz, there are no signs of the oscillations in modulus and phase that are so apparent in analyses of mammalian circulations. Pulse wave velocities will be slow, although accelerating toward the gills owing to the increased stiffness of the distal ventral aorta. Nevertheless, it is unlikely that pulse transmission will occupy a large enough portion of the cardiac cycle (10–20%) for significant phase changes to occur be-

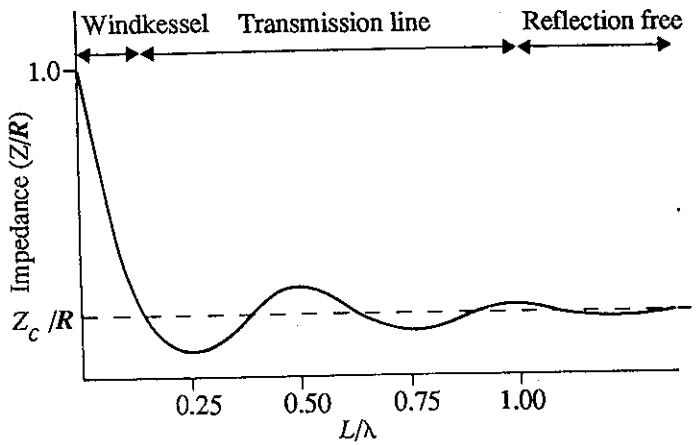

Fig. 8. A generalized curve showing impedance amplitude as a function of frequency expressed as the ratio of the arterial tree length (L) to the fundamental pressure wavelength (λ). A short arterial tree and/or a low heart rate shift the impedance spectrum to the left, making the system function as a "windkessel." A very long arterial tree or a very high heart rate shifts the spectrum to the right, making it more like a reflection-free transmission line. Impedance modulus (Z) is normalized by dividing by peripheral resistance (*R*). Z_c is the characteristic impedance. Figure and caption from Gibbons and Shadwick (1991b), by permission.

tween pressure and flow pulses at different arterial sites. These phase changes can lead to marked deviations between pulses expected in a "windkessel" and those actually recorded, and are caused by wave propagation effects.

In mammals, wave-propagation effects cause amplification of the pressure pulse as it moves toward the periphery, distortion of the pulse profile, and the appearance of diastolic secondary waves. These features result from both geometric and elastic tapering of the arteries as the periphery is approached, as well as discrete reflections either from discontinuities in the arterial system or in the terminal impedance (Taylor, 1964; McDonald, 1974; Gibbons and Shadwick, 1991b; Jones, 1991). However, reflection effects in mammals are more pronounced the higher the terminal resistance. The terminal resistance for the ventral aorta is the gill resistance, which is generally less than that of the systemic resistance. Consequently, a number of factors contribute to a lack of wave transmission effects in the ventral aortic circulation of teleosts, and it is entirely possible that circumstances may exist (i.e., an elongated ventral aorta, extremely high heart rate, low pulse wave velocity, and high gill resistance) to promote considerable deviation from the "windkessel" model.

Pressure and flows in the dorsal aortic circulation are pulsatile, albeit with some loss of the high-frequency components of the waveform seen in the ventral aorta. Unfortunately, simultaneous recordings of pressure and flow have been made only in Atlantic cod (*Gadus morhua*) and yellowfin tuna (Jones *et al.*, 1974; 1992a), although the numerous recordings of pressures made in the postbranchial circulation suggest that neither cod nor tuna are in any way unique. Dorsal aortic flow pulsatility as a function of mean flow, is about 2.5 to 4 times pressure pulsatility (as a function of mean pressure). If the dorsal aortic circulatory system were purely resistive, then pulse flow and pulse pressure would be equal percentages of their means. Hence, there is considerable compliance in the dorsal aortic system, which subserves a "windkessel" function. Even if the vessels of the dorsal aortic circulatory system are stiffer than those of the ventral, as seems to be the case in elasmobranchs (Lander, 1964), their considerable length means that compliance of this part of the circulation will be substantial.

Analysis of electrical and mechanical models of the fish circulation by Langille *et al.* (1983) shows that dorsal and ventral aortic pressure oscillations are inversely proportional to dorsal aortic compliance, whereas ventral aortic flow oscillations are directly proportional. However, the effect of increasing dorsal aortic compliance on ventral aortic pressure pulsatility is rather small, so energetic savings in terms of reduced cardiac work will not be that big (Jones *et al.*, 1974). On the other hand, a large dorsal aortic compliance may have a deleterious effect on gas exchange. With a large dorsal aortic compliance, cardiac stroke flow rushes through the gills to charge the dorsal aortic capacitance, increasing flow oscillations not only in the ventral aorta but also at the gas-exchange surface of the gills.

Since the dorsal aorta is long, a significant proportion of the cardiac cycle may be taken by the pulse wave in traveling along it. Hence, one of the basic tenets of the "windkessel," that pressure changes generated by cardiac contraction occur simultaneously throughout the arterial system, will not be fulfilled. Thus, under certain conditions, wave transmission effects might be expected in the dorsal circulation. For instance, body length is related to body mass to the minus one-third power, whereas the correlation between heart rate and body mass in fish (if one exists) has not been determined. Only if heart frequency decreases in proportion to the change in aortic length will haemodynamics of large and small fish be the same. But, if there is no heart frequency : body mass relation, and if both large and small fish have similar heart rates, then wave transmission effects can be expected in the former but not in the latter. Also, heart rate may increase with

increase in temperature and with exercise, so it is possible that wave-transmission effects will become more significant in fish exercising in warm waters.

A major benefit of wave transmission phenomena is that the high terminal impedance, represented by the vascular beds, is essentially decoupled from the input to the arterial system. This represents a considerable energetic saving, which is totally realized when the ratio of the length (L) of the arterial tree to the fundamental pressure wavelength (λ) is one-quarter (Fig. 8). λ is the pulse wave velocity divided by the repetition (heart) rate. However, λ is not fixed and varies directly with wall stiffness, which can be changed as a result of neural, humoral, and hormonal influences. Furthermore, an increase in blood pressure that occurs particularly during initial stages of exercise, will increase stretch of the arterial walls and make them stiffer.

Nevertheless, the major determinant of the properties of the dorsal aortic circulation is L/λ (Fig. 8; Gibbons and Shadwick, 1991b). A short dorsal arterial vessel or low heart rate will shift the curve left, making the system function as a "windkessel" (Fig. 8). A long vessel and high heart rate confer the benefits of wave transmission effects. However, in extremely long fish, these benefits may be lost as the system will function as a reflection-free transmission line (Fig. 8).

V. BLOOD-FLOW DISTRIBUTION AND VASCULAR RESISTANCE

Blood-flow distribution is regulated by smooth muscle activity in the resistance vessels induced neurally, humorally, or locally. However, the basal vascular tone is set by myogenic activity within electrically coupled smooth muscle cells, contracting to counteract stretch caused by the blood pressure. In addition, contraction of smooth muscle cells can be modified by endothelial-derived substances (Vanhoutte *et al.*, 1986; Miller and Vanhoutte, 1986). Hence blood flow distribution is affected not only against a background set by the basal vascular tone, but also modulation by endothelial-derived factors.

In fish the branchial and systemic vascular beds represent the two major sites of resistance in the circulatory system. Approximately 30% (range 18–40%) of the resting total peripheral resistance (TPR) occurs in the gills, while the remaining 70% (range 60–82%) resides in the visceral and somatic vasculature (Table I). In the few species of tuna in which it has been examined, branchial resistance (R_g) is significantly elevated (41–65% of TPR), presumably as a result of their much larger

gill surface area. The innervation and control of the branchial vasculature has been reviewed in a previous volume (XA) in this series (Nilsson, 1984a) and elsewhere (Nilsson, 1983; 1986; Nilsson and Axelsson, 1987) and will not be considered here. Further, nonadrenergic and noncholinergic (NANC) neurotransmitters such as bombesin, neurotensin, and vasoactive intestinal peptide (Holmgren and Nilsson, 1983a,b) are considered in detail in Part B, Chapter 5. This section will emphasize adrenergic and cholinergic control and their effects on distribution of systemic blood flow.

A. Blood Flow Distribution at Rest

Although little is known about the autonomic innervation of the systemic vasculature in cyclostomes, anatomical and histochemical evidence suggest that there is some adrenergic spinal innervation to the blood vessels in lampetroids (Leont'eva, 1966). In addition, infusion of adrenaline and acetylcholine into hagfish, *Myxine glutinosa*, produces a small reduction (10–30%) in systemic resistance (R_s), whereas adenosine reduces R_s by almost 50% (Axelsson *et al.*, 1990). In spite of unusually low dorsal (P_{da}) and ventral (P_{va}) aortic pressures (Table I), TPR in *M. glutinosa* is similar to teleosts because of a correspondingly low $\dot{Q}$.

The autonomic innervation of the arterial system in elasmobranchs has not been extensively studied. Histochemical and *in vitro* studies have demonstrated the existence of adrenergic innervation of systemic arteries in *Squalus acanthias* and *Scyliorhinus canicula* (Nilsson *et al.*, 1975). However, direct neurogenic control of R_s has not been demonstrated. Instead, *in vivo* and *in vitro* experiments suggest that control of R_s at rest is mainly via circulating catecholamines (Short *et al.*, 1977; Butler *et al.*, 1978; Opdyke *et al.*, 1981, 1982). Injection of catecholamines *in vivo* produces a large α-adrenoceptor mediated systemic vasoconstriction, as well as a smaller β-adrenoceptor-mediated vasodilation (Kent and Pierce, 1978; Opdyke *et al.*, 1982). Certainly, indirect neurogenic control of vascular resistance may exist, however, since ganglionic blockade of pre- and postsynaptic transmission has been shown to reduce the release of catecholamines from chromaffin cells in *S. acanthias* (Opdyke *et al.*, 1983).

Histochemical, *in vitro*, and *in vivo* evidence shows that the major systemic arteries and arterioles in teleosts receive widespread adrenergic innervation (Nilsson, 1983; 1984b). While both α- and β-adrenoceptors are present, the α-adrenergic constrictor mechanism dominates the β-adrenergic dilator mechanism in the systemic vascu-

Table I

Systemic and Vascular Resistance Calculated from Data Measured in Fish at Rest and during Exercise (cm · s^{-1}) or Hypoxia[a]

Species	Mass (kg)	Condition	$\dot{Q}$ ($ml^{-1} \cdot min^{-1} \cdot kg^{-1}$)	P_{va} (mmHg)	P_{da} (mmHg)	Resistance: Branch (mmHg · $ml^{-1} \cdot min^{-1} \cdot kg^{-1}$)	Resistance: System (mmHg · $ml^{-1} \cdot min^{-1} \cdot kg^{-1}$)	Resistance: Total (mmHg · $ml^{-1} \cdot min^{-1} \cdot kg^{-1}$)	Branch (% of total)	System (% of total)	Reference[b]
Myxine glutinosa	.55–.91	rest	8.7	7.8	5.8	0.23	0.66	0.90	26.0%	74.0%	1
		hypoxia-15	8.7	9.4	6.8	0.30	0.78	1.08	28.0%	72.0%	
Scyliorhinus stellaris	2.8	rest	52.5	25.2	18.5	0.13	0.35	0.48	26.6%	73.4%	2
		~24 cm · s^{-1}	89.2	25.5	17.5	0.09	0.20	0.29	31.4%	68.6%	
S. canicula	0.75	rest	32.1	38.0	29.0	0.28	0.90	1.18	23.7%	76.3%	3
		hypoxia-77	35.7	32.0	24.0	0.22	0.67	0.90	25.0%	75.0%	
Anguilla anguilla	0.51	rest	11.8	37.9	25.0	1.12	2.17	3.29	34.0%	66.0%	4
		hypoxia-40	7.8	32.3	17.6	1.88	2.26	4.14	45.3%	54.7%	
A. australis	0.62	rest	11.3	38.6	23.5	1.34	2.08	3.41	39.1%	60.9%	5
		15 cm · s^{-1}	11.3	39.0	23.6	1.36	2.09	3.45	39.4%	60.6%	
		24 cm · s^{-1}	10.3	46.8	21.8	2.42	2.11	4.53	53.4%	46.6%	
A. japonica	0.3–0.6	rest	11.0	24.2	16.9	0.66	1.54	2.20	29.8%	70.2%	6
		hypoxia-80	9.5	23.3	17.4	0.62	1.83	2.45	25.2%	74.8%	
		hypoxia-40	4.8	14.3	9.7	0.97	2.05	3.02	32.1%	67.9%	
Gadus morhua	0.4–0.8	rest	17.3	36.8	24.0	0.74	1.39	2.12	34.7%	65.3%	7
		26 cm · s^{-1}	25.4	46.5	30.0	0.65	1.18	1.83	35.5%	64.5%	
G. morhua	0.4–1.3	rest	19.2	36.8	23.3	0.70	1.21	1.91	36.7%	63.3%	8
		hypoxia-35	19.2	47.3	37.5	0.51	1.95	2.46	20.6%	79.4%	
Hemitripterus americanus	0.67–1.4	rest	18.8	28.5	23.3	0.28	1.24	1.52	18.4%	81.6%	9
		30 cm · s^{-1}	30.9	35.3	26.3	0.29	0.85	1.14	25.5%	74.5%	
Ophiodon elongatus	3.8–6.5	rest	11.2	38.0	28.3	0.87	2.53	3.39	25.5%	74.5%	10
		hypoxia-75	9.9	39.2	30.2	0.91	3.05	3.96	23.0%	77.0%	
		hypoxia-35	7.7	33.8	23.9	1.29	3.10	4.39	29.3%	70.7%	
Oncorhynchus mykiss	0.9–1.5	rest	17.6	38.8	31.0	0.44	1.76	2.20	20.1%	79.9%	11
		44 cm · s^{-1}	28.4	40.2	30.0	0.36	1.06	1.42	25.4%	74.6%	
		63 cm · s^{-1}	34.8	48.7	30.0	0.45	0.95	1.40	32.3%	67.7%	
		73 cm · s^{-1}	42.9	52.2	33.7	0.43	0.79	1.22	35.1%	64.6%	
O. mykiss	0.1–0.7	rest	36.7	31.2	25.4	0.16	0.69	0.85	18.8%	81.2%	12
Thunnus albacares	1.4	spinal block	115	89.7	32.6	0.49	0.28	0.78	63.7%	36.3%	13
		hypoxia-130	115	89.7	32.6	0.50	0.28	0.78	63.7%	36.3%	
		hypoxia-90	115	89.7	32.6	0.50	0.28	0.78	63.7%	36.3%	
		hypoxia-50	74.1	89.7	32.6	0.77	0.44	1.21	63.7%	36.3%	
T. albacares	1.0–2.0	anesthetized	61.9	84.4	44.9	0.64	0.73	1.36	46.8%	53.2%	14
T. alalunga	7.8–10.7	spinalectomy	29.4	84.2[b]	48.1	1.23	1.64	2.86	42.9%	57.1%	15
Katsuwonus pelamis	1.6	spinal block	132.3	87.3	40.2	0.36	0.30	0.66	54.0%	46.0%	13
		hypoxia-130	132.3	87.3	40.2	0.36	0.30	0.66	54.0%	46.0%	
		hypoxia-90	105.3	87.3	36.2	0.49	0.34	0.83	58.5%	41.5%	
		hypoxia-50	75.0	87.3	40.2	0.63	0.54	1.16	54.0%	46.0%	

[a] PO_2 given in mmHg. Central venous pressure was assumed to be zero in all conditions.

[b] Mean ventricular systolic pressure, (1) Axelsson *et al.*, 1990; (2) Piiper *et al.*, 1977; (3) Short *et al.*, 1979; (4) Peyraud-Waitznegger and Soulier, 1989; (5) Davie and Forster, 1980; (6) Chan, 1986; (7) Axelsson and Nilsson, 1986; (8) Fritsche and Nilsson, 1989; (9) Axelsson *et al.*, 1989; (10) Farrell, 1982; (11) Kiceniuk and Jones, 1977; (12) Wood and Shelton, 1980; (13) Bushnell and Brill, 1992; (14) Jones *et al.*, 1992a; (15) Lai *et al.*, 1987.

lature (Wood, 1976; Farrell, 1981; Nilsson and Holmgren, 1985). Although a cholinergic innervation of major arteries in eel and trout was postulated by Kirby and Burnstock (1969), experimental evidence has not substantiated the idea of primary cholinergic vascular control in the systemic circuit (Nilsson, 1983). The relative importance of neural versus humoral control of resting R_s has been a matter of some controversy (Wahlqvist and Nilsson, 1977; Smith, 1978). It is now generally agreed that in rainbow trout (Smith, 1978; Wood and Shelton, 1980), resting R_s is maintained by a neurally mediated adrenergic tonus. This was also thought to be the case in Atlantic cod (Smith *et al.*, 1985), but recent evidence suggests that the neural adrenergic influence on systemic vasculature in Atlantic cod is extremely low, verging on the nonexistent (Fritsche and Nilsson, 1990). In eels (*Anguilla australis*), adrenergic innervation of dorsal aorta, visceral arteries, and small arterioles has been demonstrated histochemically (Gannon, 1972), and both adrenaline and spinal stimulation increase R_s, yet pharmacological blockade of α-adrenoceptors fails to reduce R_s. Hence, it appears that in eels, unlike in rainbow trout, there is no resting adrenergic tonus to the systemic circulation (Hipkins *et al.*, 1986).

Due to the difficulty of measuring blood flow and pressure in visceral and somatic vascular beds, R_s is usually considered a lumped parameter. A few studies, however, have attempted to study the distribution and control of blood flow to individual vascular beds within the systemic circuit using radioactive microspheres. In this technique, activity of tissue samples following injection of microspheres is taken as a measure of relative tissue perfusion and converted to absolute blood flow by multiplying by measured or assumed $\dot{Q}$. Results indicate that blood flow distribution to muscle and visceral organs in albacore tuna, *Thunnus alalunga* (White *et al.*, 1988), rainbow trout (Barron *et al.*, 1987), and arctic grayling, *Thymalus arcticus* (Cameron, 1975) are similar (Fig. 9A). A high proportion of $\dot{Q}$ in all three species is distributed to the red (10–38%) and white (28–50%) swimming muscles, which agrees with earlier findings of flow distribution in the dogfish, *S. acanthias* (Kent *et al.*, 1971, 1973). Among the visceral organs, most of the blood flow in the tuna and grayling goes to the liver and kidney, whereas in the rainbow trout the kidney and pyloric caeca (not shown in Fig. 9A) receive the highest percentage of $\dot{Q}$. Although the white muscle in all three species receives a large percentage of $\dot{Q}$, the actual perfusion rate (ml · min^{-1} · g^{-1}) is low when compared with red muscle, liver, and kidney (Fig. 9B).

Recently, direct measurements of blood flow to the viscera in the

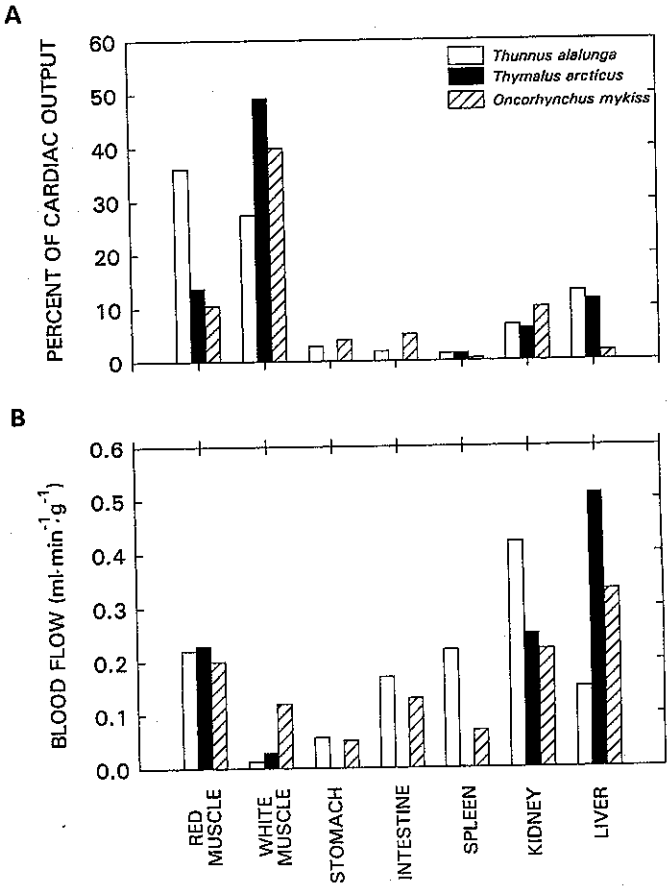

Fig. 9. The percentage distribution of cardiac output (A) and absolute blood flow (B) to selected organs and tissues of albacore tuna, *Thunnus alalunga* (open bars) (White *et al.*, 1988), arctic grayling, *Thymalus arcticus* (solid bars) (Cameron 1975), and rainbow trout, *Oncorhynchus mykiss* (striped bars) (Barron *et al.*, 1988) as determined by the injection of radioactive microspheres.

sea raven, *Hemitripterus americanus*, have been made by placing electromagnetic flow cuffs around the celiac artery, a major branch of the celiaco-mesenteric artery, which provides blood to approximately 50% of the gut (Axelsson *et al.*, 1989). Blood flow in the artery of unfed, resting fish was 2.9 ml · min^{-1} (15% of $\dot{Q}$) and doubled after feeding to reach approximately 30% of $\dot{Q}$. There appeared to be a tonically active adrenergic innervation of the visceral vasculature since administration

of phentolamine, an α-adrenoceptor blocking agent, resulted in a quadrupling of resting celiac artery blood flow. Although the exact site of visceral resistance could not be established, pressure and flow measurements demonstrated that in sea raven, at least, resistance changes did not occur upstream or proximal to the measuring site. This contradicts suggestions made for rainbow trout (Smith, 1978; Randall and Daxboeck, 1982) that the celiaco-mesenteric artery is a major control site for R_s. Resistance changes, presumably at the arteriolar level of the gastrointestinal circuit, make an important contribution to R_s, since changes in celiac blood flow are accompanied by opposite and proportional changes in dorsal aortic pressure (Axelsson *et al.*, 1989).

Doppler blood flow measurements in the celiac and mesenteric arteries of resting Atlantic cod showed that gastrointestinal (stomach, pyloric caeca, liver, and intestine) blood flow accounted of approximately 40% (7.5 ml · min^{-1}) of $\dot{Q}$ (Axelsson and Fritsche, 1991). Control of resistance to blood flow in celiac and mesenteric arteries in resting cod is unclear, however. Whereas portions of Axelsson and Fritsche's (1991) study could find no evidence for adrenergic tonus acting on the gut vessels, a second portion of the same study clearly demonstrated resting humoral and neuronal adrenergic tonus on the celiac artery. Postprandial blood flow to the gut increased to 52% of $\dot{Q}$ but did not change after the injection of the α-adrenoceptor blocker, phentolamine, which contrasts with that of the sea raven, in which blood flow increased substantially after phentolamine treatment. The difference may reflect the fact that resting adrenergic tonus is already reduced to elevate postprandial blood flow in the Atlantic cod.

B. Blood Flow during Exercise

Brett (1964) divided swimming into three categories, burst, prolonged, and sustained, in order to reduce confusion that was associated with the many types of swimming behavior. Burst swimming is high-speed anaerobic exercise lasting < 20 sec, whereas sustained swimming is aerobic exercise lasting > 200 min. Between these two extremes is prolonged swimming, which may be composed of aerobic and anaerobic components. The description that follows will focus on cardiovascular events that occur during prolonged or sustained swimming.

Cardiovascular responses to exercise are surprisingly variable among fish species. In dogfish for instance, $\dot{Q}$ increases by 70% with little change in P_{da} and P_{va}. Thus R_g falls by 43% while R_s falls by 40% (Table I). In the short-finned eel (*Anguilla australis*), on the other

hand, $\dot{Q}$ and P_{da} remain the same or decrease slightly (Table I), while P_{va} increases significantly. This is indicative of a substantial increase in R_g and virtually no change in R_s (Davie and Forster, 1980). Responses to exercise in the sea raven, cod, and rainbow trout are similar to each other, and one might suggest that they are more typical of the cardiovascular adjustments to exercise in teleosts. Increasing swimming speeds (Table I) generally cause an increase in $\dot{Q}$, an increase in P_{da} and P_{va}, and a decrease in R_s (Kiceniuk and Jones, 1977; Randall and Daxboeck, 1982; Axelsson *et al.*, 1989; Axelsson and Fritsche, 1991). The increase in P_{va} and P_{da} is biphasic in response to a step increase in swimming speed and consists of an initial increase in pressure, which peaks in 10–15 min, followed by a slow decline, which stabilizes at a value slightly higher than before the increase in swimming speed (Kiceniuk and Jones, 1977; Randall, 1982). In exercising cod, R_s has been shown to be under adrenergic neuronal control since pharmacological blockade eliminates or reverses the changes in P_{da} and P_{va} seen in untreated fish (Axelsson and Nilsson, 1986). However, Axelsson and Fritsche (1991) found that α-adrenoceptor blockade following neuronal blockade further reduced P_{da}, indicating either a possible role for circulating catecholamines in exercise or a compensatory release of circulating catecholamines in response to the pharmacological neural blockade.

Although plasma levels of adrenaline and noradrenaline were thought to increase 8- to 26-fold in response to exercise (Nakano and Tomlinson, 1967), recent work has shown this to be a result of "stress" due to how the exercise was induced, rather than a direct result of exercise *per se*. When dogfish, trout, or cod are allowed to swim spontaneously or are induced to exercise without violence (i.e., no prodding, handling, or shocking) exercise levels of circulating catecholamines are only slightly higher (two to three times) or no different than resting values (Ristori and Laurent, 1985; Butler *et al.*, 1986; Hughes *et al.*, 1988). In fact, when proper care is taken, fish can be swum to exhaustion without raising plasma catecholamine levels at all (Butler *et al.*, 1989). Therefore, although plasma catecholamine levels can be significantly elevated during repeated burst exercise, for instance, their role in controlling R_s during normal increases in swimming speed is considered to be physiologically insignificant (Butler, 1986).

The distribution of systemic blood flow is significantly altered in exercise when compared with that in rest. In exercising rainbow trout, estimates of blood flow using radioactive microspheres show that red muscle blood flow increases from 9% (rest) to 42% of $\dot{Q}$ (Randall and Daxboeck, 1982) and remains elevated for at least 2 hr postexercise

(Neumann *et al.*, 1983). The mechanism behind the dramatic increase in blood flow to the swimming muscles is still unclear, although vasodilation resulting from locally produced metabolites (CO_2, H^+, K^+, lactic acid) has been suggested (Randall and Daxboeck, 1982; Canty and Farrell, 1985). Redistribution of blood flow to swimming muscle comes at the expense of blood flow to visceral organs. In rainbow trout, for instance, blood flow to the liver, spleen, and intestine is curtailed when muscle flow is enhanced (Randall and Daxboeck, 1982). In exercising cod, blood flow in the celiac and mesenteric arteries was significantly reduced (29% and 36%, respectively) as a result of a 75% increase in vascular resistance of the celiac and 101% increase in the mesenteric circuit (Axelsson and Fritsche, 1991). Pharmacological studies indicated that resistance in the mesenteric artery was controlled by an adrenergic tonus that has nervous and humoral components, whereas no adrenergic control of the celiac artery could be demonstrated. In Atlantic cod, therefore, the blood vessels to the gut respond to adrenergic as well as nonadrenergic mechanisms. In this regard, it should be mentioned that the gut vasculature has a significant innervation with fibers that contain NANC neurotransmitters (Holmgren and Nilsson, 1983a,b). Although we do not know much about their role in controlling R_s and blood flow during exercise, their potential contribution should not be ignored.

C. Blood Flow during Hypoxia

Few studies have made the necessary measurements required to properly evaluate changes in blood-flow distribution in response to environmental hypoxia. Nevertheless, when it has been studied, R_s has been reported to increase (Japanese eel, *Anguilla japonica*, Atlantic cod, lingcod), decrease (dogfish, yellowfin tuna) or remain unchanged (European eel, hagfish, skipjack tuna) (see Table I for references). The changes in R_s are reflected in equally varied changes in $\dot{Q}$, P_{va}, and P_{da}. Although some of the alterations in R_s are simply the result of passive changes in vessel resistance (Farrell, 1982), other factors have been implicated. In Atlantic cod, for instance, $\dot{Q}$ does not change during hypoxia (water P_{O_2} = 30–40 mm Hg) while P_{va} and P_{da} increase (Fritsche and Nilsson, 1989, 1990). Pharmacological blockade of adrenergic nerves significantly reduces hypoxia-induced hypertension in the ventral and dorsal aortas. Subsequent treatment with α-adrenoceptor blockade further reduces P_{va}, which led the authors to conclude that arterial hypertension in hypoxia was primarily a result of increased nervous adrenergic tone combined with a small, but significant, tonus resulting from circulating catecholamines (Fritsche and Nilsson, 1990).

Although levels of circulating catecholamines do not increase substantially in exercise, levels of adrenaline and noradrenaline increase markedly in hypoxia. A number of studies with rainbow trout (Tetens and Christensen, 1987; Boutilier *et al.*, 1988; Ristori and Laurent, 1989) have shown that while concentrations of both catecholamines increase 300–400% during deep hypoxia (water P_{O_2} = 20–40 mm Hg), noradrenaline becomes the predominant catecholamine, attaining levels as high as 20.5 n$M \cdot l^{-1}$ (Ristori and Laurent, 1989). However, in Atlantic cod subjected to 30 to 40 mm Hg hypoxia (Axelsson and Fritsche, 1991) both noradrenaline and adrenaline reach similar levels (30 n$M \cdot l^{-1}$, noradrenaline; 50 n$M \cdot l^{-1}$ adrenaline). Although it is clear that plasma catecholamine levels rise in response to hypoxia, *in vitro* studies indicate that they do not appear to reach a level high enough to alter R_s. In Atlantic cod, rainbow trout, and short-finned eel tail preparations, significant changes in R_s occur only when catecholamine levels reach 100 to 300 n$M \cdot l^{-1}$ (Wood and Shelton, 1975; Wahlqvist, 1980; Davie, 1981).

In contrast to the redistribution that occurs during exercise, distribution of blood flow to muscles and visceral organs in the arctic grayling remains unchanged when ambient oxygen is reduced to 55 mm Hg (Cameron, 1975). However, when Atlantic cod are exposed to hypoxia (water P_{O_2} = 30–40 mm Hg), mesenteric artery blood flow decreases 62% and celiac artery decreases 42%, in spite of a 32% increase in $\dot{Q}$ (Axelsson and Fritsche, 1991). The increases in vascular resistance in both arteries were eliminated by adrenergic nerve blockade with bretylium, and further reduced by application of the α-adrenoceptor blocker phentolamine. These results agree with earlier findings (Fritsche and Nilsson, 1989, 1990) that vascular resistance in the Atlantic cod is controlled by both neuronal and humoral adrenergic mechanisms during hypoxia.

It is obvious from the preceding discussion that our understanding of the distribution of systemic blood flow is extremely limited. Obviously there are marked species differences, not only in the control of resting blood pressure and resistance, but also in control of responses to hypoxia and exercise. Even within a species, responses to similar experimental conditions differ. In Atlantic cod, for example, exposure to hypoxia (water P_{O_2} = 30–40 mm Hg) results either in an increase in $\dot{Q}$ and a decrease in R_s (Axelsson and Fritsche, 1991) or an increase in R_s, while $\dot{Q}$ remains unchanged (Fritsche and Nilsson, 1989). Standardized procedures, a wider variety of species, and more sophisti-

cated techniques will be required before we can begin to make the most basic generalizations about the regulation of blood flow in fish.

VI. HEAT-EXCHANGE RETIAL SYSTEMS

Endothermy, the ability to maintain elevated body temperature by trapping metabolic heat with vascular retia has evolved independently three times in the family Scombridae and once in the elasmobranch family Lamnidae (Block, 1991). In nonendothermic fish, heat produced by metabolism is lost to the surrounding water as blood flows through the gills. As a result, the largest steady-state excess temperature that can be maintained is about 0.5°C (Carey, 1982). Heat-exchange retial systems effectively uncouple the heat-production and heat-loss pathways and allow body temperature to exceed ambient water temperature by 5° to 20°C (Carey and Teal, 1969a,b; Graham, 1973). Vascular heat exchangers are found in three locations: the swimming muscles, the viscera, and the cranial cavity (Table II). Benefits of endothermy include niche expansion (Carey *et al.*, 1971; Graham, 1975) and increases in muscle power output (Carey *et al.*, 1971), digestion rate (Stevens and McLeese, 1984), metabolite and oxygen flux rate (Stevens and Carey, 1981), visual threshold (Block and Carey, 1985), and recovery from burst activity (Stevens and Neill, 1978).

A. Anatomy of Heat-Exchange Retia

Vascular heat exchangers, consisting of small interdigitating arteries and veins, are associated with swimming muscles in all true tunas (Scombridae), lamnid sharks (Carey, 1982), and swordfish (Block, 1991) (Table II). A single, large rete is located centrally in the hemal canal of tropical species of tuna. However, temperate and subtropical tuna, lamnid sharks, and swordfish lack a central rete. In the latter, a pair of well-developed lateral or cutaneous retia are present on the dorsal and ventral surface of the red swimming muscles (Fig. 10) (Stevens and Neill, 1978; Carey, 1982). The lateral heat exchanger's effectiveness in limiting heat loss in cold water (7°C) is exemplified by the giant bluefin tuna in which core temperatures of large (200–400 kg) tuna can be as high as 29°C (Carey and Teal, 1969b). Although both location and basic anatomy of heat-exchanging retia in lamnid sharks are similar to those of the bluefin tuna (Carey and Teal, 1969a,b), the retia in mako and great white shark (*Cacharodon carcharias*) do not

Table II
The Occurrence of Retial Heat-Exchange Systems in Billfish, Tuna, and Sharks

Common name	Scientific name	Swimming muscle[a]			
		Cranial	Central	Lateral	Visceral
Billfish[b,c]					
Blue marlin	*Makaira nigricans*	X	O	O	O
Black marlin	*M. indica*	X	O	O	O
White marlin	*Tetrapterus albidus*	X	O	O	O
Striped marlin	*T. audax*	X	O	O	O
Long bill spearfish	*T. pfluegeri*	X	O	O	O
Short bill spearfish	*T. angustirostris*	X	O	O	O
Sailfish	*Istiophorus platypterus*	X	O	O	O
Swordfish[d]	*Xiphias gladius*	X	X	O	O
Tuna[c,e,f]					
Bullet tuna	*Auxis rochei*	?	X	X	O
Frigate tuna	*A. thazard*	?	X	X	O
Kawakawa	*Euthynnus affinis*	?	X	X	O
Little tunny	*E. allatteratus*	?	X	X	O
Black skipjack	*E. lineatus*	X	X	X	O
Skipjack tuna	*Katsuwonus pelamis*	X	X	X	O
Longtail tuna	*Thunnus tonggol*	?	X	X	O
Blackfin tuna	*T. atlanticus*	?	X	X	O
Yellowfin tuna	*T. albacares*	X	X	X	O
Albacore	*T. alalunga*	X	O	X	X
Bigeye	*T. obesus*	X	O	X	X
Atlantic bluefin tuna	*T. thynnus thynnus*	X	O	X	X
Pacific bluefin tuna	*T. t. orientalis*	X	O	X	X
Southern bluefin tuna	*T. maccoyii*	?	O	X	X
Butterfly mackerel[g]	*Gasterochisma melampus*	X	O	O	O
Sharks[c]					
Mako	*Isurus oxyrhinchus*	X	O	X	X
Long-finned mako	*I. paucus*	X	O	X	X
Porbeagle	*Lamna nausas*	X	O	X	X
Salmon shark	*L. ditropis*	X	O	X	X
White shark	*Carcharodon carcharias*	X	O	X	X
Big-eyed thresher	*Alopias superciliosus*	X	O	X	O
Thresher	*A. vulpinus*	O	?	?	X

[a] X, present; O, absent, ?, unknown.
[b] Block (1986).
[c] Carey (1982).
[d] Carey (1990).
[e] Collette (1978).
[f] Sharp and Pirages (1978).
[g] Block (pers. comm.).

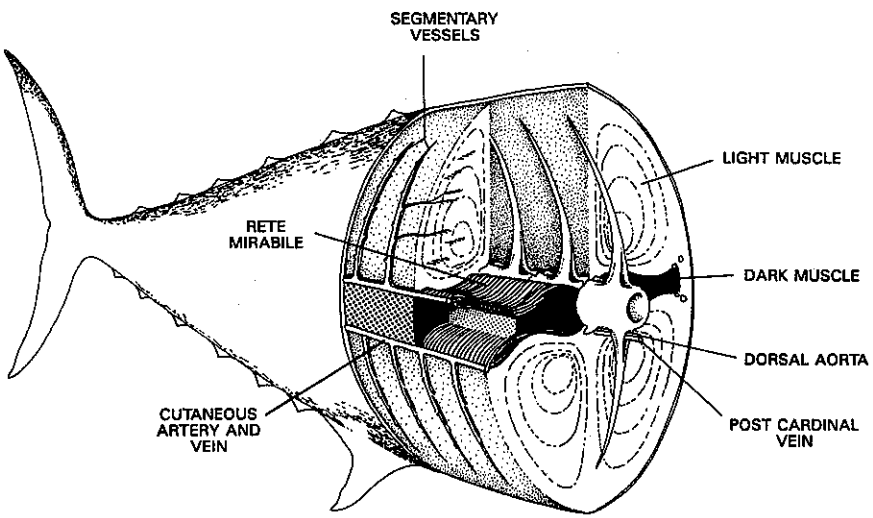

Fig. 10. A schematic diagram of the blood supply to muscle of bluefin tuna. Four pairs of cutaneous vessels branch into many smaller vessels and form the counter-current heat exchange retia above and below the red swimming (dark) muscle. The white (light) muscle is supplied by bands of alternating arteries and veins that pass through the muscle from the segmental vessels, thus acting as small heat exchangers. Other non-heat-exchanging arteries and veins (not shown) run out from dorsal aorta and post cardinal vein, respectively. Redrawn from Carey (1973).

form a compact mass of tissue, but are dispersed throughout the red muscle (Carey and Teal, 1969b).

A second, smaller rete, formed by approximately 20 vessels that branch to form triads of two arterioles and one venule, provides blood to the white swimming muscles in both tuna and sharks, except in the mako and great white sharks. In the latter, ribbons of vessels, arising from the rete, course through the white muscle (Carey and Teal, 1969b).

Among tuna species, only bluefin, albacore, and bigeye have a visceral rete (Table II). It is well developed, and visceral temperature is as high as the temperature of the swimming muscles (Carey *et al.*, 1971). The single rete is located on the dorsal side of the liver and consists of small veins that intermingle with small arteries branching from the celiomesenteric artery (Carey *et al.*, 1971). Billfish do not have a visceral rete. In contrast, porbeagle, mako, and great white sharks have paired visceral or suprahepatic retia, which are located on the ventral and lateral surfaces of the esophagus (Burne, 1923, Carey *et al.*, 1971). Unlike the red muscle retia, the suprahepatic rete in sharks consists of a spongelike meshwork of arteries, which fills the lumen of a venous sinus. Warm venous blood from the hepatic circulation fills the sinus bathing the arterial rete. The venous sinus drains into the Cuvierian ducts.

Cranial retia that protect the eyes and brain from heat loss are found in tuna, lamnid sharks, and billfish. Brain and eye temperature in bluefin tuna swimming in 20°C water can be as high as 27°C (Linthicum and Carey, 1972), and despite large fluctuations in ambient water temperature of 7° to 23°C, brain and eye temperature change by only 6° to 7°C. In tuna and lamnid sharks the heat-exchange retia are paired structures located dorsal and anterior to the first efferent branchial arteries (Stevens and Fry, 1971; Linthicun and Carey, 1972).

Brain and eye temperatures of lamnid sharks are often 5°C above ambient water temperature (Block and Carey, 1985). A pair of cranial heat-exchange retia, consisting of an arterial plexus passing through a venous sinus in the orbit, is found in the porbeagle shark (Burne, 1923), salmon shark (*Lamna ditropis*), shortfin mako (*Isurus oxyrhinchus*), longfin mako (*I. paucus*), and great white shark (Block and Carey, 1985). Recent evidence (Wolf *et al.*, 1988) suggests that in lamnid sharks, brain metabolism alone is not sufficient to account for the reported brain and eye temperatures. Heat may be imported to the brain via a "red muscle vein," which carries blood 0.3° to 4.5°C warmer than ambient water temperature from the red swimming muscle to the plexus of veins in the membrane covering the brain (Wolf *et al.*, 1988).

In this respect, the carotid retia located in the crania of billfish, swordfish, and butterfly mackerel are associated with thermogenic tissues derived from skeletal muscles attached to the eyeball (Block, 1986). Although the heater tissue has few myofibrils and is incapable of force generation, it has many mitochondria, which are used for heat generation (Block, 1986, 1987; Block and Franzini-Armstrong, 1988). The paired carotid retia at the base of the heater organs are formed by extensive branching of carotid arteries and veins. Carotid artery blood is warmed as it passes through the retia and is delivered to a fine network of capillaries on the ventral surface of the retina (Block, 1986).

B. Blood Flow in Heat-Exchange Retia

The heat exchanger's effectiveness in trapping heat is determined, in part, by three factors; the shunt fraction (how much blood bypasses the exchanger), the surface area of the rete available for heat exchange, and rate of blood flow through the rete (Graham, 1983). A measure of

effectiveness that is often used is heat exchanger efficiency (H.E.), defined mathematically as

$$\text{H.E.} = \frac{T_{xm} - T_{xvb}}{T_{xm}} \quad (4)$$

where T_{xm} = the difference between red muscle and ambient water temperature and T_{xvb} = the difference between the temperature of venous blood after passing through the heat exchanger and ambient water temperature (Brill *et al.*, 1992).

A high- and fixed-efficiency rete can be detrimental as well as advantageous. During bouts of strenuous activity, for example, the inability to shed heat can lead to overheating (Sharp and Vlymen, 1978). Because core temperature rises as a function of size, as well as activity (Neill *et al.*, 1976), the distribution of larger fish could be limited to colder waters (Barkley *et al.*, 1978). Finally, since heat exchangers reduce heat gain as well as heat loss between the fish and the surrounding water, cool endothermic fish returning to warm surface water after a vertical migration will warm at a much slower rate than nonendothermic fish (Brill *et al.*, 1992). The control of heat-exchange efficiency through blood-flow changes, therefore, has physiological, as well as ecological implications.

The extent of blood-flow rearrangement necessary to significantly alter body temperature depends on heat-exchanger efficiency. A hypothetical model of steady-state (heat production equals heat loss) muscle temperature in tuna with countercurrent heat exchangers (Brill *et al.*, 1992) shows that excess muscle temperature [T_{xm}, Eq. (4)] is related to heat-exchanger efficiency in a highly nonlinear manner (Fig. 11)

$$\text{H.E.} = \frac{(T_{xm} - 0.42)}{T_{xm}} \quad (5)$$

(Graham, 1983; Brill *et al.*, 1992). At high efficiencies (>95%), a 3% reduction in efficiency is predicted to result in a 6°C decrease in body temperature. Calculations based on body temperature and metabolism estimate heat-exchanger efficiency in skipjack and albacore tuna to be between 95 and 98% (Neill *et al.*, 1976; Graham, 1983). Direct measurements, however, show efficiency in kawakawa (Brill *et al.*, 1992) and skipjack tuna (Stevens and Neill, 1978) to be only 70%. Owing to the flatness of the efficiency–excess temperature curve below 80% efficiency, large changes in efficiency would be required to cause significant changes in body temperature.

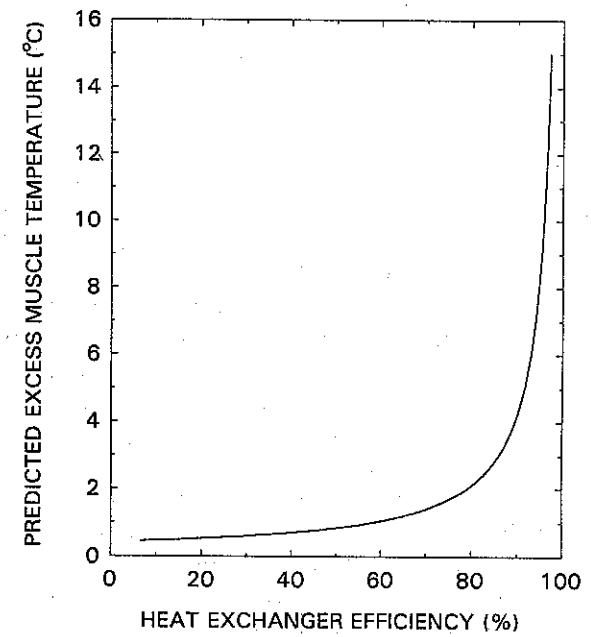

Fig. 11. Hypothetical steady state (heat production equals heat loss) muscle temperature of a tuna with a range of counter-current heat-exchanger efficiencies. Redrawn from Brill *et al.* (1992).

Anatomical studies have shown that in most fish, alternate routes exist that allow blood to reach organ systems (red swimming muscles, brain, eyes, or viscera) without passing through retia. In fish with lateral heat exchangers, for example, cool blood from the gills can be delivered to the swimming muscles via dorsal aortic branches that are not associated with retial systems (Graham, 1975; Graham and Dickson, 1981). In tuna and swordfish that have centrally located heat exchangers and poorly developed or nonexistent lateral heat exchangers, blood shunted laterally instead of centrally effectively alters the thermal conductivity of the fish (Dizon and Brill, 1979a; Carey, 1990). Alternatively, whole-body thermal conductivity can also be changed by modifying the perfusion of red muscle relative to white muscle because, although blood flow to the white muscle passes through a small rete of its own, it is not as large and well developed as those surrounding the red muscle.

Shunts bypassing arterial and venous sides of the suprahepatic retia also exist. The celio-mesenteric, lieno-gastric, and spermatic arteries

act as shunt vessels around the arterial side of suprahepatic retia (Carey *et al.*, 1981). The venous side of the rete can be bypassed via the hepatic sinus, a pathway that returns blood directly to the heart. The internal carotid arteries act as shunt vessels around the carotid retia in tuna and lamnid sharks, although the internal carotids are quite small (Block and Carey, 1985). The red muscle vein in lamnid sharks may also be used to regulate brain temperature, since warm blood flowing to the brain through this vessel can be diverted away from the brain to the lateral cutaneous rete (Wolf *et al.*, 1988).

Although it has never been demonstrated experimentally, changes in the relative resistance of retial and shunt vessels would be an effective mechanism for altering heat-retaining efficiency of the rete. Carey *et al.* (1981), for example, estimated that if venous flow through the hepatic sinus shunt (around the suprahepatic rete) increased from 0 to 20%, heat-exchanger efficiency would fall from 96 to 77%, thereby reducing visceral temperature from 8° to 1.5°C above ambient.

The effective surface area of the heat-exchange retia could also be altered by changing the resistance of vessels within the retia. Experimental and anatomical studies suggest that catecholamines may be important in altering resistance in and around the retial vascular beds. Although no direct innervation has been demonstrated, both Stevens *et al.* (1974) and Dickson (1988) have confirmed the existence of smooth muscle in the walls of arterial retia in skipjack and albacore tuna. Injecting circulating catecholamines into skipjack tuna dramatically reduces heat exchanger efficiency (Brill *et al.*, 1992), but the mechanism of action is unknown.

Heat-exchanger efficiency is inversely proportional to blood flow through the retial system (Mitchell and Meyers, 1968). Theoretically, it is possible that in heat exchangers operating at close to 100% efficiency, additional heat generated by exercise might be dissipated by increased flow through the rete. Carey and Teal (1969a) reported that violent struggling by captured bluefin tuna reduced, rather than increased, deep body temperature, yet laboratory experiments have failed to demonstrate any relationship between body temperature and exercise in skipjack tuna (Dizon and Brill, 1979a).

To date, there is no *direct* evidence that blood flow through, or bypassing, any retia occurs in response to external or internal perturbations. However, there is at least an anatomical basis for believing that fish with heat-exchange retia may be able to control rates of heat loss to, or heat gain from, the environment (i.e., phyysiologically thermoregulate). Unfortunately, even *indirect* laboratory and field evidence to support the concept of physiological thermoregulation is sparse because changes in body temperature are difficult to interpret. Alternative explanations for the observed alterations in body temperature (i.e., changes in activity, metabolism, body size, thermal inertia) must be eliminated before physiological thermoregulation can be cited as an explanation.

The above notwithstanding, physiological thermoregulation has been demonstrated, indirectly, using body-temperature measurements, in free-swimming bluefin tuna (Carey and Lawson, 1973), bigeye tuna (Holland *et al.*, 1992), and swordfish (Carey, 1990). However, in at least one case (Carey and Lawson, 1973), the authors' conclusions were challenged (Neill and Stevens, 1974) because mathematical models of passive thermal inertia accounted for most of the change in body temperature.

Evidence of physiological thermoregulation is often based on measures of the animal's whole body thermoconductivity (k), an empirically derived parameter in the non-steady-state heat-transfer equation [Eq. (6)] which applies to cases in which heat production and heat loss are not equal and body temperature is changing

$$dT_b/dt = (k \times (T_b - T_a)) + T_o \quad (6)$$

where dT_b/dt = the instantaneous rate of body temperature change, k = an empirically derived parameter describing whole body thermal conductivity, T_b and T_a = body and ambient water temperature, respectively, and T_o = the temperature increase due to heat production of the animal (Brill *et al.*, 1992). This equation is often used to mathematically describe heat transfer (gain or loss) with the environment occurring when fish make large and rapid movements up and down in the water column. Figure 12 presents body-temperature data from free-swimming fish equipped with ultrasonic temperature and depth transmitters. As expected, body temperature in the blue shark (Fig. 12A), an elasmobranch lacking heat exchangers associated with the swimming muscles, closely follows changes in ambient water temperature. The swordfish (Fig. 12B), on the other hand, has a small lateral heat exchanger and warms approximately 10 times more quickly than it cools (Carey, 1990). Although k was not calculated, the data suggest that swordfish are making circulatory adjustments in blood flow to swimming muscles and heat-exchange retia that increase whole-body thermal conductivity as they rise through the water column into warm water, and reduce thermal conductivity when they return to the colder depths (Carey, 1990).

In contrast to the fairly long-term (hours) physiological thermal regulation demonstrated by the swordfish in Fig. 12B, field data from

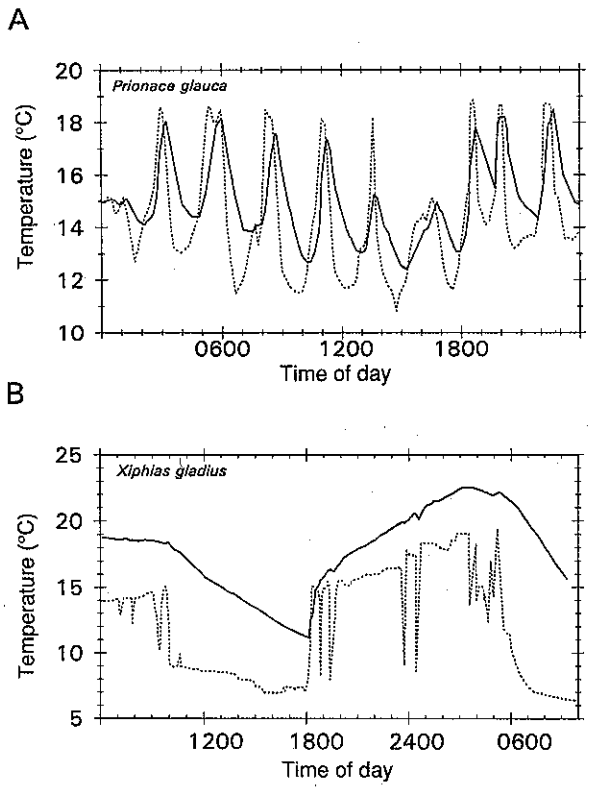

Fig. 12. Muscle temperature of a blue shark, *Prionace glauca* (A), and swordfish, *Xiphias gladius* (B), measured telemetrically. (A) Muscle temperature (heavy line) in the blue shark changes rapidly with water temperature (dotted line). The blue shark is a poikilotherm and equilibrates with water after an hour or so, as is seen at the beginning of the record. Redrawn from Carey, (1982). (B) Muscle temperature (heavy line) cools at a slower rate than ambient water temperature (dotted line) in the swordfish, which has a small lateral heat exchanger. The muscle rewarms very rapidly when the fish returns to warm water. The rate of warming is more than 10 times as fast as the rate of cooling. When the fish reentered cool water on the second day, it again cooled very slowly. Redrawn from Carey, (1990).

bigeye tuna (Fig. 13; Holland *et al.*, 1992) suggest rapid, short-term (minutes) changes in whole-body thermal conductivity. The rate of warming is approximately 50 to 200 times faster than of cooling (Fig. 13). Although metabolic heat (T_o) is expected to increase warming and decrease cooling rates, [see Eq. (6)] its contribution to body temperature was calculated to be only 0.02°C · min^{-1}, and therefore, insignifi-

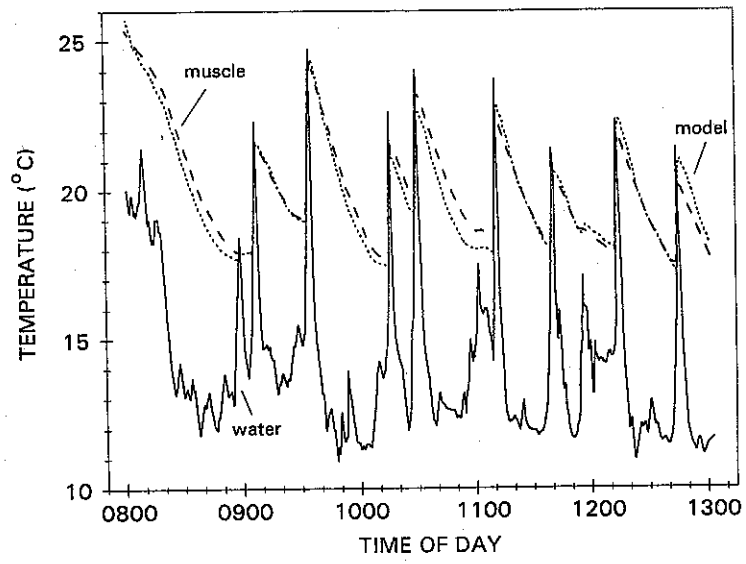

Fig. 13. Records of water temperature (water) and swimming muscle temperature (muscle) in a bigeye tuna (*Thunnus obesus*) obtained telemetrically. As for the swordfish (Fig. 12B), tuna cool much more slowly than they warm. The rapid warming and slow cooling curves generated by computer modeling (model, dotted line) closely match the observed data. Whole-body thermal conductivity [k, Eq. (6)] during warming (high k) and cooling (low k) can change by over two orders of magnitude. Redrawn from Holland *et al.* (1992).

cant. Computer modeling of body temperature fluxes (Fig. 13), which utilized a variable whole-body thermal conductivity, fitted the observed data quite well, indicating that k could change by as much as two orders of magnitude.

Whereas field data suggest that physiological thermoregulation occurs, there is little supporting evidence from controlled laboratory experiments. Dizon *et al.* (1978) and Dizon and Brill (1979a,b) studied thermoregulatory behavior when temperature and activity were closely regulated or monitored. Under these conditions, twofold to fourfold changes in whole-body thermal conductivity were recorded in swimming skipjack and yellowfin tuna. The pattern of change in core temperature of restrained albacore tuna subjected to acute temperature change suggest that they are also capable of physiological thermoregulation (Graham and Dickson, 1981).

Most recently, Dewar *et al.* (1991) and Brill *et al.* (1992) have minimicked the temperature changes encountered by tuna swimming

up and down in the water column in a large swim tunnel. Tunas with thermocouples inserted into red swimming muscle were exposed to abrupt step changes in temperature (3° to 5°C) while swimming at a constant speed [i.e., T_o, Eq. (6) is constant]. Under these conditions, thermal conductance changed rapidly, varying inversely with ambient water temperature (Dewar *et al.*, 1991; Brill *et al.*, 1992). Whole-body thermal conductivity also changed when tuna were held at a constant temperature and briefly forced to swim at higher speeds. In this case, an increase in metabolism was reflected by an increase in body temperature. The rate of body temperature increase at 25°C was seven to eight times higher than at 30°C, which suggests that at 30°C, the additional heat load resulting from increased metabolism is being dissipated, perhaps through control of heat-exchange efficiency (Dewar *et al.*, 1991).

Results presented in this discussion suggest that physiological thermoregulation occurs in endothermic fish. This, presumably, is a result of changes in the heat-exchange efficiency of the retial systems resulting from alterations in blood flow through or around the retia. Unfortunately, there is no direct evidence for neuronal and humoral control of blood flow in retia. Future research will make an important contribution toward our understanding of thermoregulation effected through changes in blood flow.

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