Functional Morphology of the Heart in Fishes

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SYNOPSIS. The systemic heart of fishes consists of four chambers in series, the sinus venosus, atrium, ventricle, and conus or bulbus. Valves between the chambers and contraction of all chambers except the bulbus maintain a unidirectional blood flow through the heart. The heart is composed of typical vertebrate cardiac muscle, although there may be minor differences in the distribution of spontaneously active cells, the rate and nature of spread of excitatory waves, and the characteristics of resting and action potentials between different fish and other vertebrates. Cholinergic fibers innervate the heart, except in hagfish which have aneural hearts. Fish hearts lack sympathetic innervation. The level of vagal tone varies considerably, and is affected by many factors. In some fish the heart is essentially aneural (without vagal tone) during exercise and may resemble an isolated mammalian ventricle with increased venous return causing increased cardiac output. There are many mechanisms that could increase venous return in exercising fish. $\beta$-adrenergic receptors have been located on the hearts of some fish, and changing levels of catecholamines may play a role in regulating cardiac activity. Changes in cardiac output in fish are normally associated with large changes in stroke volume and small changes in heart rate.

The systemic (branchial) heart of fishes consists of four chambers in series (Fig. 1). Venous blood enters the sinus venosus from the liver and ducts of Cuvier, is pumped first into the atrium, then into the ventricle, and finally into the ventral aorta via a conus (elasmobranchs) or bulbus arteriosus (teleosts). All chambers except the bulbus are normally contractile and are separated by valves at the sinoatrial and atrioventricular junctions and at the junction of the ventricle and conus or bulbus. These valves maintain a unidirectional flow of blood through the heart. The contractile conus containing cardiac muscle fibers, may have up to seven transverse rows of valves along its length, whereas the bulbus, elastic and non-contractile, has only a single pair of valves guarding the exit of the ventricle.

The sinus venosus is generally only weakly contractile; contractions of the atrium move blood into the ventricle, which is the main propulsive chamber of the heart. The contractions of the conus in elasmobranchs serve to increase the efficiency of conal valve action rather than to move blood into the ventral aorta (Satchell and Jones, 1967). The volume of the atrium in fishes, unlike that in mammals, is approximately that of the ventricle, a significant fact when considering the role of atrial contraction in ventricular filling.

The structure of the heart of many fishes has been described in detail (Robb, 1965; Mott, 1957), including the hagfish (Johansen, 1960, 1963; Jensen, 1965), many elasmobranchs (Satchell and Jones, 1967; Sudak, 1965 a,b), and teleosts (Saxena and Bakhshi, 1965; Nawar, 1955; Singh, 1960). The heart of fish is made up of what appear to be typical vertebrate cardiac muscle fibers. It is possible that fish cardiac cells are somewhat smaller than those in mammals, but the paucity of data precludes making such a generalization (Perks, personal communication). Jensen (1965) reported that the diameters of atrial and ventricular fibers in the hagfish heart were.

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FIG. 1. Diagrams of the heart in a trout (teleost) and a shark (elasmobranch).

6.1μ and 7.1μ, respectively. Atrial and ventricular fiber diameters in the dog heart are 10μ and 16μ, respectively (Hoffman and Cranefield, 1960). Small fiber diameter may in part explain why so few transmembrane potentials have been recorded from the hearts in fish.

The form of the action potential and the electrocardiogram are similar to those recorded from other vertebrate hearts (Fig. 2). As in all vertebrates there is some interspecific variability in the characteristics of the action and resting potentials recorded from the heart (Kuriyama, et al, 1960; Seyama and Irisawa, 1967; Jensen, 1965). Jensen (1965) recorded cardiac resting and action potentials from a number of fish including three species of hagfish, an elasmobranch, and two marine teleosts. He recorded resting potentials of between 40 and 60 mV in atrial and ventricular fibers in the hearts of these fishes. Investigators other than Jensen (1965) have recorded resting potentials close to 70 mV from fibers in atrial strips from the skate heart (Seyama and Irisawa, 1967), in the goldfish atrium and ventricle (Kuriyama, et al, 1960), and in the trout ventricle (Fig. 2).

A large number of fibers in the hagfish heart show a slow depolarization (pace-maker potential) during diastole (Jensen, 1965) indicating a widespread distribution of spontaneously active cells. McWilliam (1885) and Kisch (1948) have demonstrated that many regions of the heart contain fibers with the capacity to act as a pacemaker, although the initiation of the heart beat normally occurs at the sinoatrial node (Mott, 1957).

The wave of excitation spreads over the heart from the pacemaker region in a caudal direction (Chiesa, Noseda, and Marchetti, 1962; Marchetti, Noseda, and Chiesa, 1962). There are probably differences in the rate of spread of the wave of excitation over the heart (Bennion, personal communication). Direct visual observation of the contracting ventricle of the heart in a trout indicates that the apex of the ventricle (Fig. 1) contracts before portions of the ventricle closer to either the atrium or bulbus. The wave of contraction then moves from the apex of the ventricle towards the bulbus and atrium, but as the atroventricular valves are closed blood is...
FIG. 3. Pressures recorded from the chambers of the heart in the lingcod (Ophiodon elongatus). The traces have been superimposed upon one another in the diagram on the right to illustrate the relationships of the pressure changes in the various chambers. (Stevens and Bennion, unpublished data).

forced only into the aorta via the bulbus. Such an arrangement, where the apex of the ventricle contracts before its other parts, is only possible if there are fast conducting pathways which transmit the wave of excitation from the atrium to the apex of the ventricle, before other points in the ventricle closer to the atrium are excited.

In general it appears that conduction velocities of the wave of excitation over the atrium and ventricle of the trout heart are slower than in mammalian hearts (Bennion, personal communication).

The electrocardiogram has been recorded from a number of fish (Serfaty and Raynaud, 1956, 1957; Kisch 1948; Oets, 1950; Robertson, et al, 1966). Typically there is a P wave followed by a QRS complex and a T wave (Fig. 2). However Oets (1950) also recorded a V wave, just preceding the P wave and associated with the contraction of the sinus venosus of the heart in the eel. The V wave persisted even after removal of the atrium and ventricle.

There are very few recordings of pressure changes occurring in the heart during a single contraction (Johansen, 1962; Robertson, et al, 1966; Johansen and Hanson, 1967; Satchell and Jones, 1967).

Pressures recorded from the sinus venosus (Johansen and Hanson, 1967) and atrium (Fig. 3) are generally of the order of −2 to +5 mm Hg compared with atmospheric pressure. Negative pressures have been recorded in the sinus venosus of elasmobranchs (Sudak, 1965 a,b; Johansen and Martin, 1965). This negative pressure appears to be caused at least in part by contractions of the ventricle within a rigid pericardium. Negative pressure within the pericardium presumably aids venous return and increases cardiac output. Indeed Hanson (1967) found that opening the pericardial cavity in the ratfish decreased the cardiac output. Satchell and Jones (1967), however, were unable to detect any change in cardiac output in the Port Jackson shark when the pericardium was opened. The pericardium in teleosts is not rigid, and similar mechanisms do not seem to be operating to increase venous return.

In the lingcod (Fig. 3) atrial pressures only exceed those in the ventricle during or just prior to atrial systole. This, plus the fact that atrial volume is approximately the same as that of the ventricle, indicates that atrial systole plays a significant role in ventricular filling (see also Sudak, 1965a). Although in mammals the major portion of ventricular filling is due to direct venous inflow, the data presented in Figure 3 indicate that atrial contraction plays a more prominent role in fish.

Peak systolic intraventricular pressures are of the order of 50 mm Hg in the lingcod. Ventral aortic blood pressures of 70 mm Hg systolic recorded from the trout (Holeton and Randall, 1967a) indicate, however that intraventricular pressures may be higher in some teleosts (Fig. 3). Intraventricular pressures recorded from elasmobranchs appear to be somewhat lower than those recorded from the lingcod (Satchell and Jones, 1967; Sudak, 1965 a, b).

Both the bulbus and conus in teleosts and elasmobranchs, respectively, play a role in maintaining blood flow into the ventral aorta during diastole (Satchell and Jones, 1967; Satchell, 1960; Sudak, 1965a; Johansen and Martin, 1965). Satchell and Jones (1967) have shown that the lower
FIG. 4. The effect of changing heart rate on the stroke volume and velocity of blood flow in the ventral aorta of a 2.7 kg lingcod (Ophiodon elongatus). At low heart rates the blood flow falls to zero during diastole. At high heart rates the elastic rebound of the bulbus maintains blood flow in the ventral aorta during diastole.

and middle conal valves of the Port Jackson shark are incompetent in the absence of conal systole, and it appears that contractions of the conus do not aid the ventricle in forcing blood into the ventral aorta but rather assist in the closure of the conal valves. Both the conus and bulbus (Fig. 3) appear to function as distended elastic reservoirs which passively empty through the peripheral vessels at the end of ventricular systole. Thus, both the conus and the bulbus maintain blood flow into the ventral aorta during ventricular relaxation due to their elastic properties. This role is assumed by the aorta and arteries in mammals, but special structures are presumably necessary in fish to protect the gill capillaries, which are in close proximity to the heart, from large oscillations in pressure and flow. Conal systole occurs in elasmobranchs to assist in closure of the conal valves despite a negative pressure within the rigid pericardium which tends to distend the conus (Satchell and Jones, 1967).

In the lingcod, blood flow in the ventral aorta can be related to three phases of the cardiac cycle, ventricular systole, ventricular diastole, and the elastic rebound of the distended bulbus (Fig. 4). There is a rapid increase in blood flow as the ventricle contracts, a rapid decrease as the ventricle relaxes, and finally a slow decrease in the rate of flow as the volume of the bulbus decreases (Fig. 4). The elastic rebound of the bulbus maintains flow in the ventral aorta of the lingcod for about 1 sec after ventricular relaxation, so that only when the heart rate falls below about 40/min-ute does blood flow in the ventral aorta fall to zero during diastole. There does not appear, in either the lingcod or shark (Satchell and Jones, 1967) to be any reversal of flow in the ventral aorta during diastole similar to that seen in mammals when the semilunar valves close. This is undoubtedly due to the presence of the conus and bulbus in elasmobranchs and teleosts, respectively.

In Figure 4A the minimum flow rate of blood in the ventral aorta of the lingcod is between 9 and 12 ml per minute. The mean blood flow is 15 ml/minute, and the period of time when the maintenance of blood flow in the ventral aorta is due to the elastic rebound of the bulbus is 44% of the total time. This means that blood flow in the ventral aorta due to the elastic rebound of the bulbus represents about 29% of total cardiac output. At lower heart rates the proportion of blood flowing during this period is undoubtedly larger. Thus, the bulbus plays a significant role in dampening oscillations in blood flow imposed upon the ventral aorta by contractions of the ventricle.

Reported values for cardiac output from fishes cover a wide range (Hart, 1943; Burger and Bradley, 1951; Mott, 1957; Johansen, 1962; Goldstein, et al, 1964; Holeton and Randall, 1967b; Stevens and Randall, 1967b). These variations may be due to differences in the techniques used, in the prevailing environmental parameters, in the physiological state of the ani-
mal, or in actual interspecific variations between fishes.

An increase in temperature causes a marked increase in cardiac output (Fig. 5), due almost entirely to an increase in heart rate (Grodzinski, 1955; Labat, et al, 1961; Laurent, 1962; Wilber, 1961). Temperature presumably acts directly on the pacemaker cells of the heart (Laurent, 1962), altering membrane permeability and increasing the intrinsic rate of these cells. Surprisingly, temperature has little or no effect on stroke volume in the lingcod (Fig. 5).

Many environmental parameters affect the activity of the heart (Labat, 1966) including light flashes (Otis, Cerf, and Thomas, 1957; Labat, Peyraud, and Serfaty, 1962), mechanical vibrations (Bianki and Vinnitski, 1965), salinity changes (Serfaty and Labat, 1960, 1961; Labat and Serfaty, 1961), atmospheric pressure (Labat, Demers, and Aureille, 1965), anoxia (Satchell, 1960, 1961; Randall and Shelton, 1963; Shelton and Randall, 1962; Randall, 1966; Randall and Smith, 1967; Serfaty, Labat, and Bernat, 1965), removal from water (Leivestad, Anderson, and Scholander, 1957; Serfaty and Raynaud, 1957 a,b, and 1958; Garey, 1962), and touch (Serfaty and Labat, 1960). Most of these parameters probably affect the heart indirectly, mainly by altering the level of vagal inhibition on the heart.

With the exception of that of the hagfish, all fish hearts are innervated by a branch of the vagus nerve (Ripplinger, 1950; Mott, 1957; Laurent, 1962; Randall, 1966). There appear to be no sympathetic fibers innervating the fish heart (Coutdeaux and Laurent, 1958) and all efferents are assumed to be cholinergic (Laurent, 1962; Randall, 1966) and can be blocked with atropine (Mott, 1957). The level of vagal tone to the heart varies in different species of fish and in the same species under different conditions (Stevens and Randall, 1967a). There appears to be no vagal tone in resting salmonids but a high level in some resting cyprinoids. Hypoxia or exposure of the fish to air causes an increase in vagal tone and a marked bradycardia in both elasmobranchs and teleosts (Satchell, 1961; Garey, 1962; Randall and Shelton, 1963; Randall, 1966; Randall and Smith, 1967). It has generally been supposed that this hypoxic bradycardia indicates a decreased cardiac output; however in the trout, hypoxia, although associated with a bradycardia, does not cause any marked changes in cardiac output (Fig. 6). This fish changes from a condition of high rate and low stroke-volume to a state of low rate and high stroke-volume with the onset of hypoxia (Holeton and Randall, 1967b). The heart beat is co-ordinated with breathing during hypoxia, and the condition of low rate and high stroke-volume may allow a longer time for blood to remain in the gills during the periods of maximal water flow (Hughes, 1964; Hughes and Shelton, 1962; Satchell, 1961). Thus, the significance of the bradycardia in this instance may be in relating the flows of blood and water at the respiratory surface to increase the effectiveness of gaseous exchange (Randall, Holeton, and Stevens, 1967).
Sinus arrhythmia maintained by vagal inhibition during certain phases of the breathing cycle has been observed in fish in aerated conditions (Lyon, 1926; Lutz, 1930; Satchell, 1960; Shelton and Randall, 1962) and, as Satchell (1960) has suggested, may also serve to correlate maximum flows of blood and water at the respiratory surface.

Stimulation of a large number of sensory systems causes a general increase in vagal tone resulting in bradycardia. The extent to which various species of fish respond to stimulation in terms of an increase in vagal tone appears variable. In the lingcod almost any external stimulus will elicit bradycardia. Even when the animal swims, the heart rate decreases due to an increase in vagal tone (Stevens and Bennion, personal communication). In the sucker (Catostomus macrocheilus) swimming is associated with a release of vagal tone and a marked increase in heart rate. In the trout with no resting vagal tone there are only small increases in heart rate associated with swimming. If, however, swimming occurs during hypoxia, when there is a high level of vagal tone, activity is associated with a marked increase in heart rate in the trout due to a release of the hypoxia-induced vagal tone. I have only observed bradycardia in trout when the animal is in an hypoxic environment or exposed to air. General disturbances cause either no change or a small increase in heart rate in salmonids (Randall and Stevens, 1967).

Laurent (1962) concluded that there are afferent fibers innervating receptors in the atrium and ventricle. These afferents are active during atrial and ventricular contractions. Randall (1966) was unable to detect any afferent activity in the cardiac branch of the vagus.

There is a paucity of data concerning the changes in cardiac output that occur during exercise in fish. In the trout, exercise is associated with large increases in stroke volume and small changes in heart rate (Stevens and Randall, 1967 a, b). During moderate swimming there is a four-fold increase in stroke volume and a 15% increase in heart rate above the resting level (Fig. 7). There is no vagal tone to the hearts of salmonids during rest or exercise as long as the fish is in a well aerated environment. The vagal tone to the heart...
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is reduced to zero during exercise in the sucker (Stevens and Randall, 1967a). Therefore, any changes that occur in cardiac output in these fish cannot be explained in terms of activity levels in the nerves innervating the heart. Thus, like the hagfish (Chapman, et al, 1963) some teleosts at least have an essentially aneural heart during exercise (Stevens and Randall, 1967b). An obvious exception to this is the lingcod which increases vagal tone and decreases cardiac output during short swimming bursts (Stevens and Bennion, personal communication). In the ratfish swimming is associated with an initial increase in vagal tone and a decrease in heart rate. With more prolonged periods of swimming, however, there is a subsequent increase in heart rate and cardiac output which follows the bradycardia seen at the onset of swimming (Hanson, 1967). This is also true in the lingcod (Stevens and Bennion, op. cit.).

The hagfish heart is generally considered to be aneural (Greene, 1902; Augustinsson, et al, 1956), but the recent work of Hirsch, Jellinek, and Cooper (1964) has raised some doubt. They concluded that there were nerve fibers and ganglionic cells in the myocardium; however as Jensen (1965) points out, no one has observed any connections between the hagfish heart and the central nervous system, and therefore the balance of evidence is still in favor of considering the hagfish heart to be aneural.

The in situ aneural heart of the hagfish behaves like an isolated mammalian ventricle in that it responds to increased filling by increasing its force of contraction (Chapman, 1963; Chapman, Jensen, and Wildenthal, 1963; Chapman, Wildenthal, and Jensen, 1968). Similar mechanisms may be operating on the hearts in teleosts and elasmobranchs. Indeed Johansen (1962) observed an increase in stroke volume with increasing venous return in the cod, and the changes in cardiac output occurring in the trout during exercise may be related in part to an increase in venous return to the heart (Fig. 7).

There is a whole variety of mechanisms for increasing or regulating venous return in fish. Hagfish possess several accessory hearts, namely the portal, cardinal, and caudal hearts (Chapman, Jensen, and Wildenthal, 1963; Jensen, 1963). The action of these hearts plus the contractions of skeletal muscle around the gills aid the circulation of the blood and regulate venous return to the systemic (branchial) heart.

Valves have been demonstrated in the segmental arteries and veins of some elasmobranchs (Satchell, 1965). These valves are so arranged that contractions of muscles during swimming force blood from the arterial to the venous side of the circulation, thus increasing venous return during swimming. Venous valves also appear to be present in teleosts (Dornesco and Santa, 1963), and muscular action may play a role in increasing venous return to the heart. In many teleosts there is an elastic ligament running down the middle of the dorsal aorta (Dornesco and Santa, 1963; de Kock and Symmons, 1959). This rather stiff ligament, extending from the basioccipital to the caudal region, may aid in the propulsion of blood down the dorsal aorta as waves of muscular contraction pass down the body during swimming. This ligament, therefore, may represent a mechanism in teleosts for increasing venous return during swimming.

There may be some mobilization of blood from storage organs during swimming. In teleosts, swimming is associated with a decrease in splenic volume (Stevens, 1968), and hepatic sphincters may regulate venous return to the heart and the volume of blood in the liver during exercise (Johansen and Hanson, 1967). Lowering of the dorsal fin in the ratfish decreases the amount of blood in the dorsal fin sinus (Hanson, personal communication), and such a mechanism could increase venous return to the heart. The ratfish, however, does not appear to lower the dorsal fin at the onset of swimming.

Catecholamines are known to affect the hearts of fishes (Ostlund, 1954) and could play some role in the regulation of cardiac output in fish. Eptatretin from the hagfish
heart (Jensen, 1963), a potent cardiac stimulant, may also play a role in the regulation of cardiac output in the hagfish. It has not been found in any other animal.

High levels of catecholamines have been found in cyclostome hearts (Augustinsson, 1956; Bloom, et al, 1961; Hirsch, Jellinek, and Cooper, 1964). Much lower levels of adrenaline and noradrenaline have been demonstrated in the hearts of other fishes (von Euler and Fänge, 1961), and adrenaline usually represents more than 50% of the total catecholamines present. Catecholamines cause an increase in the rate and force of the heart beat in teleosts and elasmobranchs, but rather surprisingly have little or no effect on the branchial heart of the hagfish (Ostlund, 1954). β-adrenergic receptors have been demonstrated in the plaice heart (Falck, et al, 1966). In the intact salmon, Randall and Stevens (1967) found that adrenaline either had no effect or increased the heart rate even after blocking β-adrenergic receptors with either dichloroisoproterenol or propranolol. This does not mean, however, that β-receptors are not present in the salmon heart, as the changes in rate after blocking the β-receptors could be due to a secondary effect of adrenaline on some other part of the circulatory system.

Levels of catecholamines have been measured in the blood of salmon and carp (Fontaine, Mazeaud, and Mazeaud, 1963; Mazeaud, 1964) and are known to increase during activity in the trout (Nakano and Thomlinson, 1967). Curves of ventricular function relating venous filling pressure to stroke work have not been constructed for any fish, but changing levels of catecholamines are known to alter the shape of these curves in mammals (Sarnoff and Berglund, 1954). Changing levels of catecholamines in the blood of the trout during swimming would affect the rate and strength of contraction of the heart and may alter the relationship between venous filling and the force of contraction.

The balance of evidence indicates that changes in cardiac output in fish result from changes in stroke volume (Hanson, 1967; Johansen, 1962; Stevens and Randall, 1967b). There is some adjustment of heart rate produced via cholinergic nerves innervating the heart, changes in temperature, and perhaps changing levels of catecholamines in the blood. Stroke volume increases when venous return to the heart increases. There is a variety of ways for increasing venous return to the heart in fish and these may be operative during activity, when the heart of many fish appears to be essentially aneural. Under these circumstances it may be behaving in situ like an isolated mammalian ventricle in that it obeys Starling's law of the heart.

Changing levels of catecholamines probably affect the relationship between venous filling and stroke volume during exercise in at least some teleosts. Changes in rate occurring in the heart during exercise may be related to a release of vagal tone, increased levels of catecholamines, or to an increase in venous pressure (Labat, Raynaud, and Serfaty, 1961).

The denervated mammalian heart in situ responds to exercise with large changes in stroke volume and heart rate (Shepherd, 1965), changes which are in many ways similar to those observed in the trout. Increases in cardiac output during exercise in intact mammals are usually associated with changes in heart rate, stroke volume remaining fairly constant (Rushmer, 1965). This would indicate that the evolution of a vagal and sympathetic supply to the heart in vertebrates is primarily for the more precise control of heart rate, such that heart rate can be regulated to change cardiac output while maintaining a constant stroke volume. It seems that regulation of heart rate in fish is not very precise, and changes in cardiac output are associated with large changes in stroke volume. There is however some development of neural regulation in the fish heart with the development of vagal tone (except in the hagfish), but refinements like a sympathetic innervation to the heart have not occurred and appear to be a special property of tetrapods. It may be some consolation to fish, however, that Shepherd's (1965) dogs
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ran just as well even after complete denervation of the heart.

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