The comparative physiology and anatomy of the hagfish, a primitive, vertebrate, have been intermittently under study for about two centuries. In our own century, it was Greene's discovery that the branchial heart of the Pacific (California) hagfish (Eptatretus stoutii) is aneural that set the stage for renewal of interest in the animal. Greene's work, published in 1902, was confirmed by A. J. Carlson and, in recent years (in Myxine), by Augustinsson and co-workers. Greene reasoned that regulation of ventricular activity in the hagfish must depend upon the conditions which affect the muscle directly... The volume and pressure of the blood coming to the heart [and other factors]... must have a decided influence on the hagfish heart.' These neglected observations lie chronologically between Frank's work on the response of the frog heart to increased filling (1895), and Starling's investigations in dogs which were the basis for what we now call Starling's Law of the Heart (1915). In the years of controversy that followed Starling's sweeping generalizations, the relative roles of neural control factors, on the one hand, and of intrinsic myocardial response, on the other, have come under sharp scrutiny. Some workers hold that, in the intact mammal, neural control is dominant and that the role of intrinsic myocardial response is minimal. Others shift the emphasis to various degrees.

The hagfish circulatory system, uniquely incorporating a totally aneural ventricle, can hardly fail to be of interest to the circulatory physiologist and it seems curious that Greene's incisive comment was ignored so long. Renewed interest in the animal was stimulated in 1958 by Jensen who was led to the Pacific hagfish in part by Greene's observations. In 1961, Jensen concluded that... cardioregulation in the aneural branchial heart of the hagfish is accomplished by two major factors: tension as governed by venous return, and a biochemical factor produced in the heart.' He also found that the usual response of the branchial heart (or ventricle alone) to slight distention is to accelerate its rate but that... this heart has given no indication of contracting with greater force when the diastolic filling is increased..." The ventricle might, Jensen thought, even lessen its force of contraction under conditions of stress.

These results not only suggested the existence of a most unusual circulatory control system in which Starling's law had no part, but also raised the possibility that there may be a type of contractile tissue that does not exhibit the usual tension-length properties. The following study was undertaken in the hope of throwing light on some of these important questions.
FIGURE 1

The Pacific hagfish (Eptatretus stoutii). The head is to the left. The twelve external gill openings extend from the second to the ninth centimeter marks on the rule.

THE HAGFISH: GENERAL COMMENT

Myxine glutinosa, the Atlantic hagfish, was recognized by Linnaeus in 1758 but was grouped by him with the worms.8 A few years later (1765) Gunner described the animal as "a worm that resembles an eel" and commented on its extraordinary ability to produce slime.9 Home, in 1815, thought the hagfish to "... hold an intermediate place between ... the class pieces and the class vermes ..." (fig. 1). He studied a single specimen brought from the South Seas and correctly distinguished between what we now call Myxine, on the one hand, and Bdellostoma, Polistotrema, or Eptatretus8 on the other. Home's careful description of the structure of the gills and gill openings was especially valuable.16 (fig. 2). The circulatory anatomy of Myxine received the meticulous attention of Anders Adolf Retzius, Professor of Anatomy at the Karolinska Institute in Stockholm in 182217 and 1824.18 In 1841, Berlin's famous physiologist, Johann Müller, published an extensive account of the hagfish circulatory system based on his dissections of dead specimens. He described the branchial heart and its valves and confirmed Home's earlier description of the animal's gill system. He also observed a "heart-like" expansion of the portal vein (as had Retzius)19 but denied that it was independently contractile or that it functioned as anything more than a simple conduit.20 Several years later Müller observed that the expansion beats rhythmically in the living fish21 and subsequent workers showed that the portal expansion not only has muscle fibers in its walls but also possesses valves at inlet and outlet apertures. It is now recognized as a separate ampullary heart, possibly aneural, and is called the portal heart. A third heart of very unusual structure was described by Gustav Retzius, son of Anders Adolf, in 1890.22 This, the caudal heart, is actually a pair of flattened sacs between which is a cartilaginous plate (figs. 3 and 4).23 The sacs receive blood from subcutaneous sinuses and pass it on to veins which unite to form the caudal vein. The sacs have no muscle in their walls and are not in themselves contractile. Their pumping action is attributable, as shown by Greene,24 to rhythmic contraction of overlying muscle strips which bend the anterior end of the cartilaginous plate from side to side causing alternate filling and emptying of each sac. Valves at inlets and outlets of the chambers ensure forward passage of blood.25 The contractile elements are under reflex control and cease all movement if the adjacent spinal cord is destroyed.24

The three generic terms are now regarded as synonymous. Eptatretus8 dating from 1819, has chronologic priority. Bdellostoma10 was introduced in 1834 and Polistotrema12 in 1881. The most recent synonym is Dodecatrema.13 The Pacific species (Eptatretus stoutii)14 was named for Dr. Arthur B. Stout, a practicing physician in San Francisco who served as corresponding secretary for the California Academy of Science from 1875 to 1878.

Sir Everard Home, John Hunter's brother-in-law, may in fact have utilized some of Hunter's drawings and writings in preparing the description. After Hunter's death, Home destroyed a large number of Hunterian manuscripts, of which he was curator. Among those destroyed was a volume on the anatomy of fish and works on eels and earthworms. Clift, Hunter's amanuensis, later accused Home of destroying the manuscripts in order that his plagiarisms could never be established.30 The specimen described by Home was brought from the South Seas by Sir Joseph Banks, Hunter's pupil and friend, who accompanied Captain James Cook on his first voyage (1768-1771).
Home's drawing of the gill structure of Eptatretus (left), and Myxine (right). Note the separate external gill openings of Eptatretus and the paired, combined ones in Myxine.
The arrangement of the various hearts in the hagfish appears, therefore, to be one in which the main, or branchial, heart (atrium and ventricle) is served by two lesser pumping mechanisms, both concerned with venous return.* Only the caudal pump is known to be under neural control.

The environment in which the hagfish appears to develop optimally is difficult or impossible to simulate in the laboratory. Said by Stensiö to be a degenerate lineal descendant of the earliest vertebrate, it has no jaws and probably cannot seize living prey for food. Although it has on occasion been found in very shallow water, it usually lies virtually immobile in lightless, frigid marine canyons for very long periods of time (weeks or months). When, however, dead or dying marine animals are brought within range of its keen olfactory perception, it is capable of vigorous and effective action. Once it has fed—and in captivity it lives for up to eight months without food intake—it again appears to settle into a state of marine hibernation. The extent to which metabolic and circulatory activities are slowed at such times is not known, but unless disturbed by light or mechanical stimuli, the slowing must be profound. Maxwell has shown that the animal possesses a hyperbolic hemoglobin dissociation curve and that 95% saturation occurs at oxygen tensions of less than 40 mm Hg. Under "normal" conditions, however, its arterial blood (dorsal aorta) is about 50% saturated and venous blood almost totally deoxygenated. The implications are that it can live actively at ambient oxygen tension levels of 10 to 20 mm Hg and that its tissues are acclimated to tensions of no more than 2 to 3 mm.

According to McFarland and Munz the Pacific hagfish is "... unique among vertebrates in being isotonic to sea water and having the osmotic concentration of the blood composed almost entirely of ionic constituents." Serum sodium and chloride account for 88% of its total osmotic pressure. The animal has few, if any, natural enemies and does not appear to be equipped to combat mi-

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* Cole describes paired contractile venous expansions far forward in the head region and calls them the fourth heart.
Circulation in the Hagfish

crobial attacks or to heal wounds. As Papermaster and co-workers have recently shown, it forms no antibodies when injected with substances that are antigenic in higher species. Its chief defense mechanisms appear to be its ability to move about with great vigor and its remarkable capacity to secrete enormous quantities of protective slime.

The rather complex anatomical relations between the branchial and the portal hearts are shown in figure 5. The branchial heart and its connections are depicted in figure 6. The ventral aorta (e), separated from the branchial ventricle (d) by a bicuspid valve, leads venous blood to the 12 pairs of gills. Thence the oxygenated blood is collected in the dorsal aorta which distributes it to the tissues and to the subcutaneous sinuses. Return is via the venous system to the sinus venosus (a). The caudal hearts, whose function is to move blood from posterior subcutaneous sinuses to the caudal vein, have already been described. The portal heart, receiving most of its blood from mesenteric veins, also has a connection with the systemic venous system via the right anterior jugular vein. Figure 7 shows the general pattern of the circulatory system.

Microscopically, it is of significance that the thick branchial ventricular wall is loosely arranged and possesses no coronary system. It is also of interest that the tissue contains large amounts of epinephrine and norepinephrine which are distributed in specific granular cells similar to those of the mammalian adrenal medulla.

**Methods**

Specimens of *Eptatretus stoutii* were collected in a tributary of the San Diego trough and were kept in a refrigerator at about 10°C in buckets of fresh sea water. The animals used were from 35 to 45 cm long and weighed 60 to 100 g. The wet weight of the branchial ventricle varied between 75 and 120 mg.

For most experiments, the animals were lightly anesthetized by putting them into a sea water environment containing small amounts of tricaine methane sulfonate. Catheters were then tied firmly into the nostril so that the gills could be perfused at regular intervals but continuous perfusion could not be carried out under the circumstances. The possibility that the tissues of the hearts were sometimes hypoxic cannot be ruled out but was
minimized by the frequency of perfusion and by the constant application of aerated sea water directly to the exposed organs. The branchial and portal hearts were approached from the ventral aspect through a midline incision, care being taken not to injure the branchial vessels. Catheters were carefully lined up with the axes of the vessels to be entered (ventral and dorsal aortas and posterior cardinal vein) and their distal ends fixed with pins to the dissecting board. The tips were then introduced into the desired vessel and were tied in place. For taking pressures directly from the branchial ventricle, atrium, or portal heart, short lengths of catheters tipped with No. 24 hypodermic needle points were used. The needles were inserted directly through the walls of the chambers to be studied.

It is hardly possible to place a catheter or needle directly into the lumens of the caudal heart sacs. Instead, needles were inserted so that their open ends lay just over the sac itself. While no direct pressures could be obtained in this way, very satisfactory records of caudal pump activity were nevertheless recorded.

When catheters were in place, the animal and the board on which it rested were transferred to a table where strain gauges were already mounted. The heights of the gauges were then adjusted and connection with the catheters was established; great care had to be taken to avoid pulling catheter tips out of the vessels in which they rested. Temperatures were controlled approximately by frequent applications of sea water held at 8° to 10°C.

Isolated branchial heart preparations were prepared by passing catheters into the ventricular lumen through the ventral aorta for pressure measurements. A second catheter was passed into the ventricle via the atrium and was tied in place with a ligature in the A-V groove. The cannulated heart was then removed and suspended in cold sea water. The pressure cannula was connected to a strain gauge and the ventricular catheter to an injection device for delivering precisely measured quantities of cold sea water.

**Results**

The rates of branchial and portal hearts appear to be dependent on temperature, as shown earlier by Jensen. In the present work, four isolated hearts held at 8° to 15°C showed rates of 5 to 12 per minute. Raising the temperature to 20°C or higher brought rates up to 24 per minute.

Under laboratory "resting" conditions, rates for the three hearts in four different "intact" specimens of *Eptatretus stoutii* were as shown in table 1.

It was repeatedly noted that in isolated heart preparations and in the "intact" animal, rate always slowed and sometimes stopped if return flow was at a very low level. The easiest way to restart a quiescent heart was to perfuse it. And reperfusion of a heart beating at a very slow rate usually, but not always, caused the rate to increase.

Of great interest was the fact that branchial ventricular rate seemed to depend to an important extent on the degree of A-V block at any particular time. Degrees of block up to 6:1 were observed and, in general, the degree of block tended to increase when the ventricle was overdistended (fig. 8). Very high degrees of block often indicated deterioration of the preparation. In other instances, where the ventricle was not being perfused

---

**TABLE 1**

Rates Per Minute of Various Hearts in Four Specimens of *Eptatretus stoutii* with "Intact" Circulatory Systems Under Laboratory Conditions. No A-V Block Was Present.

<table>
<thead>
<tr>
<th>Experimental time</th>
<th>Temp °C</th>
<th>Branchial ventricle</th>
<th>Portal heart</th>
<th>Caudal heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 min</td>
<td>11</td>
<td>18</td>
<td>24</td>
<td>0</td>
</tr>
<tr>
<td>10 min</td>
<td>16</td>
<td>24</td>
<td>30</td>
<td>0</td>
</tr>
<tr>
<td>0 min</td>
<td>16</td>
<td>23</td>
<td>30</td>
<td>88 (or 44)</td>
</tr>
<tr>
<td>5 min</td>
<td>16</td>
<td>25</td>
<td>25</td>
<td>88 (or 44)</td>
</tr>
<tr>
<td>10 min</td>
<td>16</td>
<td>24</td>
<td>26</td>
<td>88 (or 44)</td>
</tr>
<tr>
<td>18</td>
<td>22</td>
<td>20</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>26</td>
<td>30</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
Schematic drawing of circulatory apparatus of Eptatretus stoutii.

Effect of overdistention of the branchial heart by continuous injection of sea water into the posterior cardinal vein, beginning at zero time. Note the initial slowing (owing to increase in AV block) and the appearance of bigeminy. (Retouched.)

Alternating caudal heart beat (below). The small upstroke just before the main upstroke of brachial ventricular pressure is due to atrial contraction.

Nonalternating caudal heart beat (negative deflections) (below). Branchial and portal heart rates are unusually rapid.

Spontaneous cessation of caudal heart beat (below). The isolated beat was followed by six minutes of inactivity.
(and may, therefore, have been hypoxic) the degree of block was reduced when even small amounts of fluid were presented to the ventricle. The result was usually an increase in rate.

*Portal heart rate* ranged between 15 and 60 per minute but usually fell below 30. There was some evidence that overdistention of the chamber produced marked irregularity in its rhythm.

*The caudal heart*, in the undisturbed "intact" preparation, is often completely inactive. When in action, its rate is usually faster than that of the branchial or portal hearts. Frequently, alternans patterns (fig. 9) were obtained owing to the reciprocal action of the two pumps. In other tracings, a clear single impulse is seen (fig. 10). Irregularities of the caudal rhythm and rate are common. Slowing for a few seconds often precedes total cessation of activity (fig. 11). When conditions are ripe for resumption of activity, the hearts usually start abruptly at or near the rate that prevailed in the last period of activity. Restarting of the caudal heart beat often followed perfusion of the gills, washing of the tail region with cold water, and pricking the intact skin with a pin. Periods of quiescence, in the absence of external stimulation, lasted from two to thirteen minutes.

*Resting systolic pressures* in the branchial ventricle and portal heart usually were less than 1 mm Hg. In the branchial ventricle, a resting range of 0.7 to 5.9 mm Hg was noted.

Under conditions of maximal or near-maximal load, the branchial ventricle, whether isolated or in situ, frequently drove pressures up to 20 mm Hg and on one occasion an isolated heart reached 30.8 mm Hg. Similarly, the branchial atrium and the portal heart, when moderately or maximally distended, were consistently capable of developing pressures up to about 2.0 mm Hg.

The response of the isolated branchial ventricle to filling is shown clearly in figure 12, which represents a repetition of Frank's classical experiments with the frog heart. It will be seen that the isolated ventricle, contracting virtually isovolumically, develops higher pressure with each increase in amount of sea water injected (0.015 ml increments). There was, however, no descending limb. In figure 13, one sees the effect of injecting 0.03 ml of sea water into the posterior cardinal vein of an "intact" preparation. The ventricular systolic pressure more than doubles between beats. Continuous injection of sea water into the posterior cardinal vein (2.0 ml over a 30-second period) may cause ventricular irregu-
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Family of pressure curves produced by isolated branchial ventricle, contracting isovolumically. Curve 1 was a control beat; Curve 2 followed the injection of 0.015 ml of sea water. The preparation was then allowed to return to zero diastolic pressure and 0.030 ml was injected (Curve 3). Curve 4 resulted from an injection of 0.045 ml, Curve 5 from 0.060 ml, and so on. Curve 9 resulted from an injection of 0.120 ml. Temperature 11°C throughout, (Redrawn, from original recordings).

Epinephrine in various concentrations, injected into the posterior cardinal vein or into the isolated ventricle produced no effect beyond that attributable to volume alone (fig. 16). Reserpinization produced very little change in the isovolumic response of the branchial ventricle to volume stress (table 4). There was some evidence, however, that the ventricular response declined in some animals after reserpinization and that it was partly restored toward control values by the use of epinephrine (fig. 17).

### Table 2

Response of Branchial Ventricle (in Terms of Maximal Aortic Pressure) to Injection of 0.075 ml of Sea Water Every 30 Seconds.

<table>
<thead>
<tr>
<th>Total injected ml</th>
<th>Maximal pressure mm Hg</th>
<th>Rate beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>7.0</td>
<td>21</td>
</tr>
<tr>
<td>0.075</td>
<td>20.4</td>
<td>24</td>
</tr>
<tr>
<td>0.150</td>
<td>21.2</td>
<td>24</td>
</tr>
<tr>
<td>0.225</td>
<td>20.4</td>
<td>24</td>
</tr>
<tr>
<td>0.300</td>
<td>19.4</td>
<td>27</td>
</tr>
<tr>
<td>0.375</td>
<td>18.6</td>
<td>30</td>
</tr>
<tr>
<td>0.450</td>
<td>18.1</td>
<td>30</td>
</tr>
<tr>
<td>0.525</td>
<td>17.8</td>
<td>30</td>
</tr>
<tr>
<td>0.600</td>
<td>17.1</td>
<td>30</td>
</tr>
</tbody>
</table>

Response, in terms of ventral aortic pressure, to injection of 0.030 ml sea water into posterior cardinal vein at Δ.
Maximal pressure response to serial injections of increasing volumes of sea water into posterior cardinal vein of “intact” preparation. Isotonic contraction. Lower curve represents response when increments of 0.030 ml were used. Upper curve shows response to 0.060 ml increments from the third to the eighth injections.

Resistance to flow across the aortic valve seemed to be negligible as judged by the pressure drop from ventricle to aorta. Under “resting” conditions it was not measurable with accuracy and when perfusion (via the posterior cardinal vein) was maximal, it was no more than 1 or 2 mm Hg (fig. 18). Resistance to flow in the gill vascular system was greater but highly variable. Under “resting” conditions, pressure drop from ventral to dorsal aorta was regularly 2 to 3 mm Hg and was greatly increased when the branchial heart (“intact” preparation) was perfused (fig. 19). When perfusion was maximal, the drop from ventral to dorsal aorta rose as high as 12 mm Hg but tended to fall as perfusion was continued.
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FIGURE 18
Pressure drop from branchial ventricle to ventral aorta. Ventricular calibration is at left, aortic at right. At A, 0.30 ml of sea water was injected into posterior cardinal vein. Note the effect on atrial pressure (small rise at the beginning of the ventricular upstroke) as well as on ventricular response.

The electrocardiogram varies according to placement of the electrodes. Using needle electrodes placed on each side of the branchial ventricle (RA on the right, LA on the left), dipolar tracings were obtained as shown in figure 20. The main deflections (QRS) and the preceding P waves are easily seen. Another deflection, diphasic in form, stems from the portal heart. PR intervals were highly variable but were usually about one second. In some instances they were much longer. It proved to be impossible to obtain caudal electrocardiograms.

Discussion
The key finding in the present study is that the aneural branchial ventricle, which is the primary driving force of the hagfish circulatory system, responds to increased filling by increasing its force of contraction precisely as does the isolated frog or mammalian ventricle. The unperfused hagfish ventricle, whether isolated or in situ, tends to beat very slowly or to stop altogether for relatively long periods. When reperfused, it readily begins beating again but may require several seconds to return to its previous level of activity. This phenomenon was also observed by Jensen and may account in part for the discrepancy between his earlier work and the present study.

The nearest approach to “resting” physiological conditions under laboratory conditions was achieved by Johansen who worked with unanesthetized Myxine glutinosa at temperatures of 4° to 7°C. The rates of branchial...

TABLE 3
Response of Isolated Branchial Ventricle (Maximal Pressure in mm Hg) to Increasing Increments of Filling at Various Temperatures. The Experiments at 13° and at 21° Were Done on the Same Heart. Those at 11° and 14° Were Done on Different Hearts.

<table>
<thead>
<tr>
<th>Increment of filling (ml)</th>
<th>11°</th>
<th>13°</th>
<th>14°</th>
<th>21°</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>5.6</td>
<td>2.0</td>
<td>1.8</td>
<td>1.2</td>
</tr>
<tr>
<td>.015</td>
<td>10.3</td>
<td>11.8</td>
<td>2.4</td>
<td>5.2</td>
</tr>
<tr>
<td>.030</td>
<td>15.2</td>
<td>12.4</td>
<td>10.3</td>
<td>11.6</td>
</tr>
<tr>
<td>.046</td>
<td>16.5</td>
<td>16.1</td>
<td>17.3</td>
<td>17.5</td>
</tr>
<tr>
<td>.060</td>
<td>18.7</td>
<td>18.8</td>
<td>21.0</td>
<td>19.2</td>
</tr>
<tr>
<td>.075</td>
<td>20.2</td>
<td>21.7</td>
<td>23.0</td>
<td>19.8</td>
</tr>
</tbody>
</table>

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and portal hearts he observed were quite similar to those found in the present study. The participation of periodic contraction of gill musculature in the propulsion of blood from gill capillaries through the systemic arterial tree, as reported by Johansen, was not substantiated. It is possible that the anesthetic abolished the effect but it is also possible that the unanesthetized animal produced so much slime that external gill apertures became occluded; periodic contraction of gill musculature may, therefore, have been directed at clearing the external openings rather than at the propulsion of blood.

In any event, the branchial ventricle proved to be able to deal quite effectively with relatively enormous volume loads and, when contracting isovolumically, to develop extremely high pressures. There is little evidence, therefore, that it requires a booster effect in the outflow (arterial) circuit.

It proved to be difficult to produce the descending limb described by Frank and Starling; the hagfish branchial ventricle usually responded to stepwise increase in filling by increasing its force of isovolumic contraction until it suddenly stopped altogether. On being allowed to empty, it usually began beating again without difficulty. In the "intact" preparation, ventricular arrhythmia frequently occurred as the ventricle became overloaded but failure in the usual sense was not observed.

In agreement with Bloom et al., the branchial heart proved to be quite insensitive to epinephrine, even in high concentrations. Pretreatment with reserpine seemed to have very little effect on ventricular sensitivity to epinephrine. It is possible that reserpine caused a diminution in the vigor with which the ventricle responded to increased filling and that subsequently, epinephrine merely restored it to its normal functional status. In any case, the response to epinephrine in no way resembled that found in the frog heart by Segall and Anrep.

The response of the portal heart resembles that of the branchial in that it increases its force of contraction as filling is increased. It must, therefore, respond, under normal conditions, in the same way to increased filling from mesenteric veins after the animal has fed. Its rate is more constant than is that of the other hearts and not infrequently it continued to beat when the other hearts were temporarily quiescent. When overloaded it, like the branchial ventricle, sometimes beats irregularly.

The caudal hearts, as intimated earlier, are quite unpredictable under laboratory conditions. Greene's demonstration that destruction of the cord causes permanent cessation of caudal heart activity, plus the observation...
that various external stimuli seem to be able to bring about resumption of activity, support the concept that the caudal hearts are reflexly controlled. It seems likely that distention of the lateral sinuses, among other stimuli, can activate the paired pumps although this is not certain.

The circulatory control system of the hagfish appears, therefore, to embody both neural and myocardial features. The animal is unique primarily in that the two control principles are anatomically separate. Neural mechanisms in this very primitive, elongate creature operate primarily on the venous side of the circulation, presumably making caudal and possibly portal hearts responsive to venous over- or underfilling. They, in turn, control the branchial heart by altering the amount of return flow. The branchial ventricle thus seems to be controlled solely by volume signals and the hagfish circulatory apparatus seems to utilize the Frank-Starling principle as its primary means of response to stress. Neural, and possibly hormonal, factors come to bear on the property of intrinsic myocardial response less directly than in the mammal. In higher members of the animal kingdom, both neural and myocardial mechanisms come to be intricately intertwined in the myocardium itself; in the hagfish they are neatly separate and distinct.

Finally, the astonishing facility with which the hagfish circulatory system can change from almost total quiescence to vigorously effective activity richly deserves serious study. The loose anatomical arrangement of myocardial fibers, permitting easy perfusion from either side of chamber walls, is probably part of the explanation and, in a sense, renders the ventricle more adaptable and less vulnerable than those that require coronary circulations. But there is also the possibility that the nature of the contractile tissue is different and that, as Jensen intimates, special hormonal factors play an important role. In any event, studies on the species suggest that a great deal is to be gained from revival of the comparative physiological approach to circulatory function and control.

Conclusions

1. The branchial atrium and ventricle, and the portal heart of Eptatretus stoutii (the Pacific hagfish) respond to increased filling by increased force of contraction as judged by maximal pressures developed.

2. The branchial ventricle is capable of increasing maximal pressure from less than 1 mm Hg to well over 20 mm Hg within several beats.

3. Greene's suggestion that the caudal hearts are under reflex control is borne out, if not actually proved, by the action of certain external somatic stimuli. The quiescent caudal heart is often restored to vigorous activity by such stimulation.

4. Factors controlling the portal heart are not understood but it seems to be the most constant, with regard to rate, of any of the hearts.

5. The probability that neural factors act on ampullary (venous return) hearts and that the branchial ventricle responds solely to volume stimuli is supported by present findings.

Acknowledgment

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