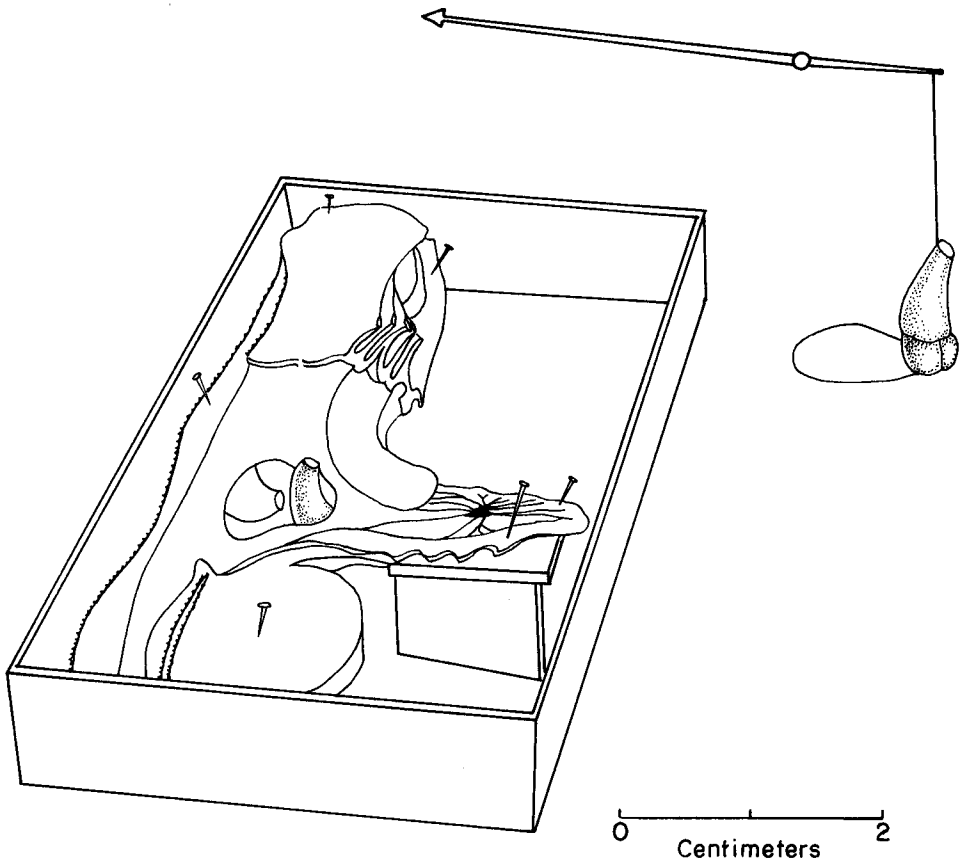

FIGURE 225.—Oka's method of exposing the visceral ganglion for study of heart stimulation in the oyster. Reproduced from Oka, 1932.

the underlying theory of temperature coefficients of biological reactions (Bělehrádek, 1935).

In experiments with *C. virginica* at the Woods Hole laboratory Federighi (1929) found the values of μ equal to 16,000 and 13,600. It is rather difficult to convert his data into conventional terms of number of beats per minute since his experimental results are presented only as plots of logarithms of the frequencies (time required for 10 beats) multiplied by 100 against the reciprocals of absolute temperature. At my request Federighi in a personal communication supplied excerpts from his laboratory notes which show the following rates:

Temperature	Beats per minute	Temperature	Beats per minute
25°	47	16°	21
21°–22°	35	10°	11

The rates appear to be much higher than those observed by others. In Federighi's experiments the upper critical temperature above which there was rapid decline in pulse rate was approximately 30° C.

In Koehring's (1937) observations on *C. virginica* the heart rate averaged 20 beats per minute at 20°. She found also that in the oysters with one valve completely removed the heart action was inhibited for several hours and there was no ciliary motion of the gill epithelium. Inhibition of the heart's activity when the shells are closed was reported by Stauber (1940) in oysters uninjured except for perforation of both valves. He found that the heart rhythm of *C. virginica* slowed down and became irregular when the oyster closed the valves. In some of the closed oysters the heart remained inactive for 2 to 3 minutes, then resumed beating at low frequencies of about two to three times per minute, only rarely exceeding six beats per minute at the temperature of 17.5° C. As the valves began to open, the heart beat increased to 14 to 16 times per minute. These results are in accord with observations on *Anodonta* and

Sphaerium (Cyclas) by Gartkiewicz (1926), who described the suppression of heart beat and of ciliary motion during the periods of shell closures. Because of the high transparency of the shell of *Sphaerium* the behavior of the heart of this mollusk could be observed under normal conditions. Gartkiewicz calls the inhibition of cardiac and ciliary activity the "sleep" of the bivalves. The cause of the heart's inhibition is not known; it is probable that in the case of *Sphaerium* the lowered pH of body fluids and the accumulation of carbon dioxide may have contributed to the suppression of cardiac activities. This, however, does not account for the observed temporary cessations of heart beats in the oysters and clams kept in sea water but with their valves partly removed. Apparently the stoppage associated with the contraction of the adductor muscle was due to inhibition originated from the nervous system.

The heart beat in *O. circumpicta* of Japan reaches a maximum of 30 beats per minute at 35° C. and slows down to three beats per minute at 5° C. and to 14 at 40° C. No heart action was recorded by Takatsuki (1927) at 0° and at 45° C. Climatic conditions apparently influence the heart rhythm since it was shown by the same author (Takatsuki, 1929) that the heart pulsation of *O. circumpicta* P. from the waters of the northern part of Japan (Anomori Prefecture) is about 14 times per minute at 20° C. In contrast, the pulse of *O. dendata* Kuster from the bay of Palau, South Sea Islands, where the temperature ranges from 28° to 29° C. throughout the year, was only eight times per minute, and the maximum rate of 22 times per minute was observed in the laboratory at 45° C. The pulsation in the northern species at temperature of 28° to 29° C. was 24 times per minute, and the critical temperature was 35° C. These observations may indicate differences in thermic adjustments of oysters inhabiting cold and warm waters. No general conclusions can be drawn at present from Takatsuki's observations because other factors such as degree of sexual maturity and general conditions of the oyster, which were not reported, may affect the heart beat.

Visual observations can be carried on for short periods of time only, and their usefulness is, therefore, rather limited although their distinct advantage is that the heart is not affected by experimental manipulations. The pulse curve of the heart beating inside the intact pericardium may be obtained by the sphygmograph tambour technique. Continuous recording may be made for several hours before the heart is fatigued by the weight of the writing lever pressing on the pericardium wall and the rhythm and amplitude decrease.

The wave-line curve shown in figure 226 represents the changes in the hydrostatic pressure inside the pericardium, the increase in pressure corresponding to systolic contraction of the ventricle which is followed by the falling of pressure during the diastole when the auricles expand and are gradually filled with blood. The method is not sensitive enough to record separately the contractions of the auricles, which beat shortly before the contraction of the ventricle. In the experiment shown in figure 226 the oyster was kept in about 3 l. of sea water at 22.5° C.; its pulse rate was 18 to 20 times per minute.

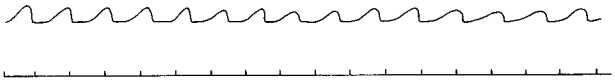

FIGURE 226.—Pulse of an adult *C. virginica* at 22.5° C. recorded by transmitting the motion of the pericardium membrane to the writing lever. Time interval: 3 seconds.

The contractions of auricles interposed between the two ventricular contractions are clearly seen on the tracings of the beats of an exposed heart with the hook connecting the writing lever placed under the auriculo-ventricular junction (fig. 227, two lower lines). In the upper line, the hook was under the ventricle near the emergence of the aorta and the auricular contractions were not registered. The increase in frequency of beat shown in the third (lowest) curve was due to an increase in the water temperature from 20.5° to 24.5° C.

Tracings obtained with the excised heart are similar to those made by the heart in situ with the hook under the ventricle since no contraction of the auricles can be registered in such preparations (fig. 228).

EXTRACARDIAC REGULATION

Carlson (1905a, 1905b, 1905c, 1906a, 1906b, 1906c, 1906d, 1907) has shown that stimulation of the visceral ganglion of *Cardium*, *Pecten*, *Mytilus*, and other bivalves produces an inhibitory effect on the heart. Using faradic stimulation, Diederichs (1935) demonstrated that a single shock applied to the visceral ganglion of *Mytilus* produces diastolic arrest. By separating the ganglia he obtained evidence that both the ac-

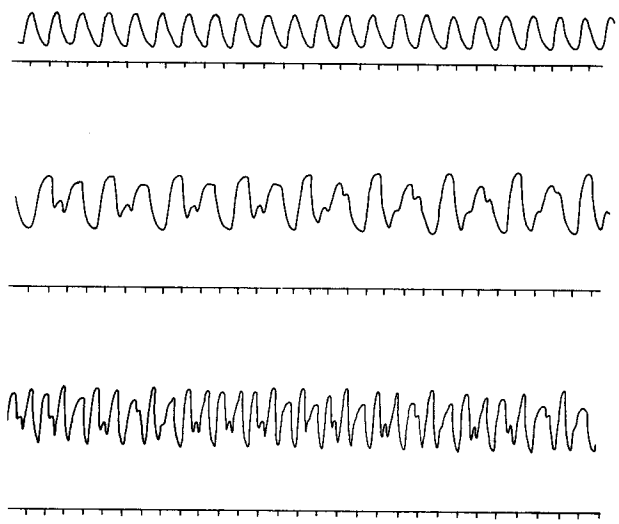

FIGURE 227.—Three records of heart beat of *C. virginica* in situ. The upper curve was obtained by placing the connecting hook of a kymograph lever under the ventricle. The two lower curves were made when the hook was placed under the auriculo-ventricular junction. Increased frequency of the lowest curve is associated with an increase of temperature of sea water from 20.5° to 24.5° C. Time interval, 2 seconds.

celerating and inhibiting nerves lead from the visceral ganglion to the heart and that the two other ganglia affect the heart by way of the visceral ganglion. Oka (1932) found that stimulation of the visceral ganglion inhibits both auricular and ventricular rhythms, and Irisawa, Kobayashi, and Matsubayashi (1961) determined the action potentials in *O. laperousi* and found that oyster heart relaxes through anodal current.

The cardiac nerve is a small branch of the visceral nerve which emerges from the cerebro-visceral connective near the visceral ganglion. Its branches enter the auricles at their base and regulate only the auricular rhythm. The ventricular rhythm, according to Oka's view, is regulated by the cardiac nerves which enter the ventricle at the aorta end. This finding is not in agreement with Carlson's observations that the cardiac nerves enter the heart of a bivalve at the base of the auricles and not at the aortic end. Experimentation with the oyster heart is difficult because exposure of the ganglion causes profuse bleeding and collapse of the heart. Furthermore, the cardiac nerves in *C. virginica* are extremely small and difficult to observe in the living tissue.

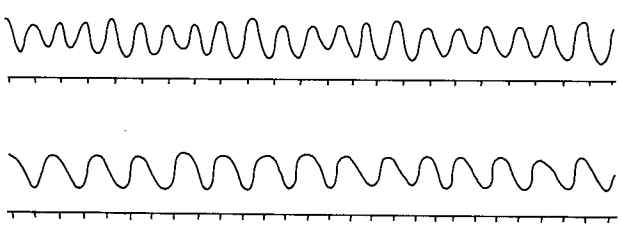

FIGURE 228.—Tracings of the beating of the excised heart of *C. virginica* at 20° C. Salinity 31.7 °/oo. Time interval, 5 seconds.

Investigations by Carlson did not demonstrate the presence of acceleratory nerves in the hearts of bivalves. Oka (1932) thinks that possibly both kinds of nerves, the acceleratory and the inhibitory. are present in the heart of *O. circumpicta* but that the action of the inhibitory nerve predominates. The suggestion is based on the observation of old heart preparations of lowered vitality in which the beat of the auricles was slightly accelerated by stimulation of the ganglion. The evidence is not convincing and requires verification.

Krigsman and Divaris (1955) arrive at the following conclusions which appear to be applicable to the oyster heart: 1) The systolic mechanism is situated in the heart's muscle fibers; and 2) extrinsic regulatory nerves influence the pacemaker system. The inhibiting fibers are probably cholinergic, and the accelerating fibers may have adrenergic properties. The latter statement needs further verification.

EFFECTS OF MINERAL SALTS AND DRUGS

Bivalve hearts respond readily to changes in the chemical composition of water and to the presence of low concentrations of various drugs and poisons. Because of this sensitivity the hearts of several common species such as *Anodonta, Mya, Mercenaria, Ostrea,* and others often have been used in pharmacological bioassays. The test is usually made with a preparation of an excised entire heart (or ventricle) in the perfused chamber. Increased acidity slows the beat of the excised heart of *C. virginica;* a pH of 4.0 and lower causes diastolic arrest and from pH 4 to 9 the rate increases with the increase of pH values. Above pH 9 the contractions become irregular (Otis, 1942).

A change in the balance of metallic ions in the surrounding water affects cardiac activity. Small excesses of potassium stimulate the heart by increasing the frequency of beat (positive chronotropic effect) and by changing the tonus (tonotropic effect) of the myocardium (Jullien and Morin, 1930, 1931b).

The action of sodium is similar to that of the potassium, but response is less pronounced. Small excesses of calcium cause negative chronotropic and positive tonotropic effects, and magnesium acts in a way similar to that of calcium, i.e., produces negative chronotropic effect and causes diastolic arrest of the heart. Lack of magnesium results in a systolic arrest (Jullien and Morin, 1931b; Jullien, 1936a).

Among the effects of various drugs the most interesting is that of acetylcholine, a chemical agent in neuromuscular transmission which depresses heart action of oysters and other mollusks (Jullien, 1935c; Jullien and Vincent, 1938; Jullien, Vincent, Vuillet, and Bouchet, 1939; Prosser and Prosser, 1938; Prosser, 1940, 1942; and Wait, 1943) and is particularly effective on the heart of the clam (*Mercenaria mercenaria*). Prosser (1940) has shown that inhibition of the heart of this species can be obtained with a concentration as low as 10^{-12}. Recent investigations by Pilgrim (1954) and Greenberg and Windsor (1962) showed that in the hearts of many bivalves acetylcholine produces a "combination response", depressing the cardiac activity in low concentrations and exciting it at high concentrations. The authors used ventricle strip preparations of the hearts of 40 American (in the Greenberg and Windsor experiments) and 8 New Zealand species (in Pilgrim's tests). Preparations which remained quiescent when first set up attained regular rhythm in 2 to 3 hours, a condition which was also observed in tests made in the Woods Hole laboratory on *C. virginica*. In Greenberg's and Windsor's experiments the quiescent preparations were induced to beat with 10^{-7} to 10^{-5} molar concentrations of 5-hydroxytryptamine.

There exists great variability in the responses of different bivalve species to acetylcholine. In some of them only the depressing effect of the drug was recorded. This group includes oysters (*C. virginica* and *C. gigas*), several clams of the family Veneridae (*Mercenaria mercenaria, Tapes philipinarum, Saxidomus giganteus*, and others), *Mya arenaria, Entoderma saxicola*, and *Prododesmus macroschisma*. The excitor effect was demonstrated for *Mytilus californianus* and *M. canaliculus* (in Pilgrim's tests), thus confirming previous observations on Mytilidae by Jullien and Vincent (1938). In Pectinidae, Matridae, Carditidae, and other families, both types of responses were recorded.

The following explanation of the "combination response", i.e., depression in low concentration and excitation in high concentration, was suggested by Pilgrim (1954): the low concentration tends to inhibit pacemaker activity; at high concentration, while the pacemaker is inhibited, the drug acts directly on the muscle causing a steady contraction. Further research is needed to corroborate this hypothesis.

Greenberg and Windsor (1962) remark that "a reasonable mode of acetylcholine action on bivalve hearts should involve either two separate sites of action or two modes of attachment to the same site at high and low concentrations".

Sensitivity of bivalve hearts to acetylcholine varies in different species. The most sensitive ones, reported by Pilgrim, are *Dosinia, Amphiderma*, and *Mercenaria mercenaria*. Oysters are less responsive to the drug. Jullien (1935c) reported that in *C. angulata* the frequency and the amplitude of heart beat are decreased in a concentration of 10^{-5} with diastolic arrest following at two times 10^{-5} to two times 10^{-4} concentration. In New Zealand species, *Ostrea hefferdi*, the cardiac activity is depressed with a diastolic arrest at concentrations varying from 10^{-8} to 10^{-5} (Pilgrim, 1954). In *C. virginica* the decrease in the frequency and amplitude of isolated heart was apparent at concentration 10^{-5} (fig. 229) and the effect persisted for several minutes after the preparation was flushed with fresh sea water (second line). The effect of the drug can be noticed even in extremely low concentrations of 10^{-8} and 10^{-9}. Under normal conditions the hearts of bivalves contain little acetylcholine (Jullien and Vincent, 1938), but the heart of the gastropod *Murex* is very rich in this compound.

Eserine causes periodical alterations in the amplitude of heart beat and slight increase in the rate of beating (fig. 230). The significance of the

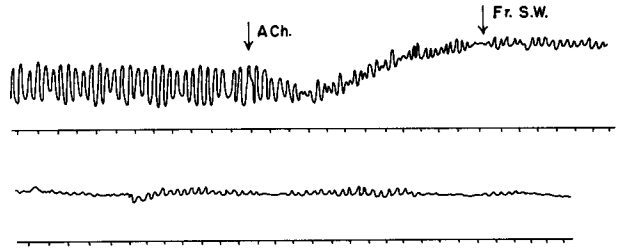

FIGURE 229.—Effect of acetylcholine in the concentration 10^{-5} on the beat of isolated heart of *C. virginica*. ACh—acetylcholine added; Fr.S.W.—perfusion chamber flushed with fresh sea water. Temperature 23.7° C. Time interval, 5 seconds.

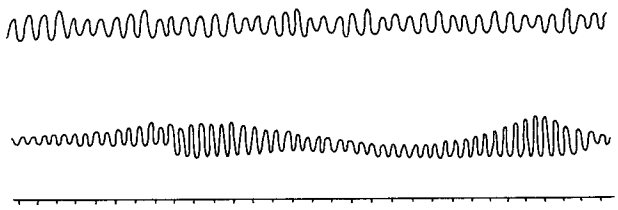

FIGURE 230.—Tracings of the heart beats (in situ) of *C. virginica* in sea water (upper line) and after the addition of eserine, (second line) in concentration of 10^{-4}, to the pericardial chamber. Temperature 21.5° C. Time interval, 5 seconds.

drug in heart physiology is the fact that it prevents the destruction of acetylcholine by the enzymes of the organism.

Veratrine has a temporary stimulating effect on the heart of *O. edulis* (Jullien, 1936a). In my experiments with isolated heart of *C. virginica*, a slight stimulating effect on the frequency of ventricular contraction was recorded in the concentration of veratrine of 1:10,000. Within a few seconds the number of beats increased from 12 to 18 and 20 times per minute at 20.5° C. (fig. 231). Navez (1936) described the depressive action of pilocarpine on the heart of *Anomia*.

High concentrations of curare inhibit the heart activity of the oyster; in lower concentrations the drug has a strong positive tonotropic effect (Jullien, 1936a) and also counteracts the inhibitory effect of acetylcholine. Jullien found that heart action stopped by acetylcholine was restored by subsequent applications of curare.

Adrenaline accelerates the heart beat of *O. circumpicta*, (Takatsuki, 1933) in a concentration of about 1.8 times 10^{-7}. Similar activating action has been reported for *C. virginica* (Otis, 1942) and for *O. edulis* (Jullien, 1935d, 1936a, 1936c). Stronger concentrations produce irregular beating and some times systolic arrest.

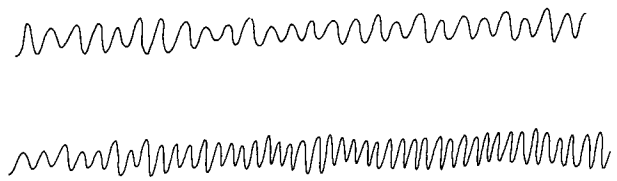

FIGURE 231.—Effect of veratrine (conc. 1:10,000) on ventricular contractions of the isolated heart of *C. virginica*. Temperature 20.5° C. Time interval, 5 sec. Upper line—in sea water; lower line—immediately after the perfusion with veratrine in sea water.

BLOOD VESSELS

Lack of continuity between the arteries and veins due to the presence of sinuses is the characteristic feature of the open circulatory system of bivalves. The spaces which function as capillaries have no distinct walls, are of irregular shape, and appear as slits in the tissue (fig. 79). Their presence imposes difficulty in the maintainance of effective circulation of blood through the organs and tissues. The deficiency is partially overcome by the presence of pulsating vessels and accessory hearts, which assist in the moving of blood through the mantle.

All blood vessels of the oyster have very thin and delicate walls that are easily ruptured by a slight increase in pressure. In anatomical preparations of the circulatory system, it is, therefore, difficult to obtain complete penetration of arterial and venous systems by injection. Partial success may be obtained by using a warm gelatine solution stained with appropriate dyes; by injecting borax or lithium carmine and immediately placing the preparation into 95 percent alcohol in which the stain is precipitated; or by injecting vinyl resin solution diluted with acetone (Eble, 1958). For more detailed study the preparation may be dehydrated and clarified in oil of cloves or in cedarwood oil. Very small vessels may be injected through a capillary tubing using aquaeous solution of methylene blue, toluidin blue, or some other suitable dye. Although no permanent preparation can be obtained in this way, the method is useful for tracing the connection between the small vessels.

Because the injection of the venous system is even more difficult than that of the arteries, knowledge of venous circulation in bivalves is less complete than that of the arterial system. Attempts to observe the movement of blood inside the veins usually are not successful because the tissues are either too contractible or contain so much glycogen that the vessels are obscured. The description of the principal blood vessels of the oyster given below is based on the examination of many specimens injected by various methods and studied under a low power of magnification.

THE ARTERIAL SYSTEM

The arteries can be recognized in microscopic preparations by their well-developed walls lined with a single layer of flattened endothelial cells

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(fig. 81). They have a distinct layer of circular and longitudinal muscles surrounded by connective tissue.

The arterial system described here is shown diagrammatically in fig. 232 from the right side, after the partial removal of the mantle and some of the visceral mass. The right wall of the pericardium is cut off to expose the heart. This diagrammatic drawing is based on examination of several specimens injected through the ventricle.

Two large arteries emerge from the posterio-dorsal side of the ventricle. The largest one is the anterior aorta (ant.ao.), which upon leaving the heart forms a short enlargement or a bulb leading to the large visceral artery (visc.a.) with its numerous branches and small pericardial artery (small unmarked vessel under the visceral artery), which supplies blood to the wall of the pericardium. The much smaller posterior aorta (post.ao.) supplies blood to the adductor muscle and rectum (r.). Near the point of emergence of the posterior aorta it gives off a small rectal artery (r.a.), which follows the wall of the rectum.

The visceral artery (visc.a.) emerges from the anterior aorta as a wide vessel that supplies blood to the organs of the visceral mass. Its upper branch reaches the level of the labial palps and of the cephalic hood. The lower branch extends along the wall of the crystalline sac and forms the reno-gonadial artery (r.g.a.); numerous small branches of this vessel supply blood to gonads and kidneys.

In its course toward the anterior part of the body, the anterior aorta (ant.ao.) passes under the intestinal loop (not shown in fig. 232) and gives off several small vessels which bring blood to the digestive diverticula (gastric arteries, g.a.), mantle, and the labial palps. At the anterior end of the body the aorta forms a common trunk of the pallial artery (co.p.a.), which divides into two short branches corresponding to the left and right side of the body, each branch giving rise to the ventral and dorsal circumpallial arteries (cr.p.a.). Each of these continues along the periphery of the mantle lobes, supplying blood to the mantle through a large number of short vessels which end in the mantle lacunae. A very small subligamental artery emerges from the end of the common pallial artery and leads to the subligamental gland (fig. 78). The cephalic artery (cph.a.) and labial artery (l.a.) supply blood to the anterior end of the body and to the right and left labial palps.

THE VENOUS SYSTEM

Since the presence of irregular sinuses prevents the filling up of the entire venous system with one injection it is necessary to make separate injections of the principal vessels and to supplement the study with an examination of sectioned material. The course of small veins may be traced by injecting a water soluble dye and watching its penetration in the tissues of the visceral mass and gills.

The venous system comprises the sinuses, afferent and efferent veins and small vessels of the gills. It is diagrammatically shown in figure 233. Ramifications of the vessels are omitted for the sake of clarity.

The sinuses occur throughout the entire visceral mass, in the pallium, along the adductor muscle, and around the kidneys. Their outlines are highly irregular, and the area they occupy varies, depending on the degree of distension by blood. The renal sinus (r.s.) consists of several smaller sinuses which surround the main part of the kidneys and open into the efferent branchial vessel and into the sinuses between the adductor muscle and the heart at the posterior side of the body. The renal sinus spreads into the connective tissue of the adjacent area and is in communication with the inter-nephridial passages leading to the pericardium. The renal vein (r.v.) carries blood from the sinus into the common afferent vein. The visceral sinus, v.s., not definitely outlined in the diagram, spreads over the surrounding tissues and drains its blood through the gastric (g.v.), hepatic (h.v.), and other veins into the common afferent vein (c.af.v.). The muscle sinus (m.s.) is a small area below the renal sinus on the surface of the adductor muscle under the pyloric region. The system of afferent veins consists of a single common afferent vein (c.af.v.) and two lateral afferent veins, l. af. v. (fig. 233 and fig. 73). The common afferent vein runs on the ridge formed by the fusion of the two inner ascending lamellae of the gills. The blood received by this vein comes from the deeper parts of the body and is brought by a number of veins which can be identified as the cephalic veins (c.v.) from the cephalic region; the labial veins (l.v.); the gastric and hepatic veins (g.v., h.v.); the network of small reno-gonadial veins (r.g.v.); short renal vein (r.v.) and the adductor muscle vein (not shown in the diagram). In thin, watery specimens most of these veins can

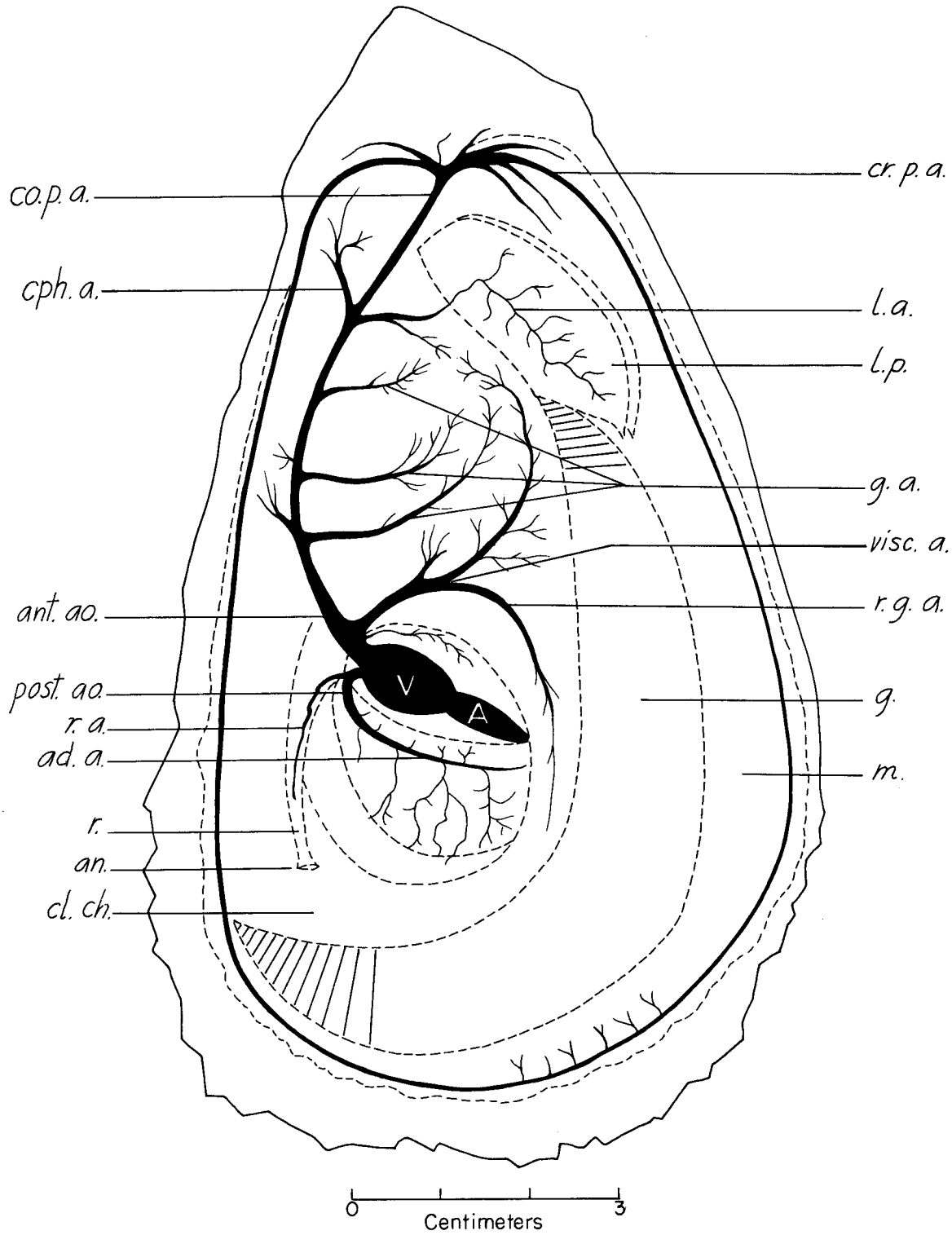

FIGURE 232.—Diagram of the arterial system of *C. virginica*. A—right auricle; ad.a.—adductor muscle artery; an.—anus; ant.ao.—anterior aorta; cl.ch.—cloacal chamber; co.p.a.—common pallial artery; cph.a.—cephalic artery; cr.p.a.—circumpallial artery; g.—gills; g.a.—gastric arteries; l.a.—labial palp artery; l.p.—labial palps; m.—mantle; post.ao.—posterior aorta; r.—rectum; r.a.—rectal artery; r.g.a.—reno-gonadial artery; visc.a.—visceral artery. For the sake of clarity profuse ramifications of the vessels are not shown.

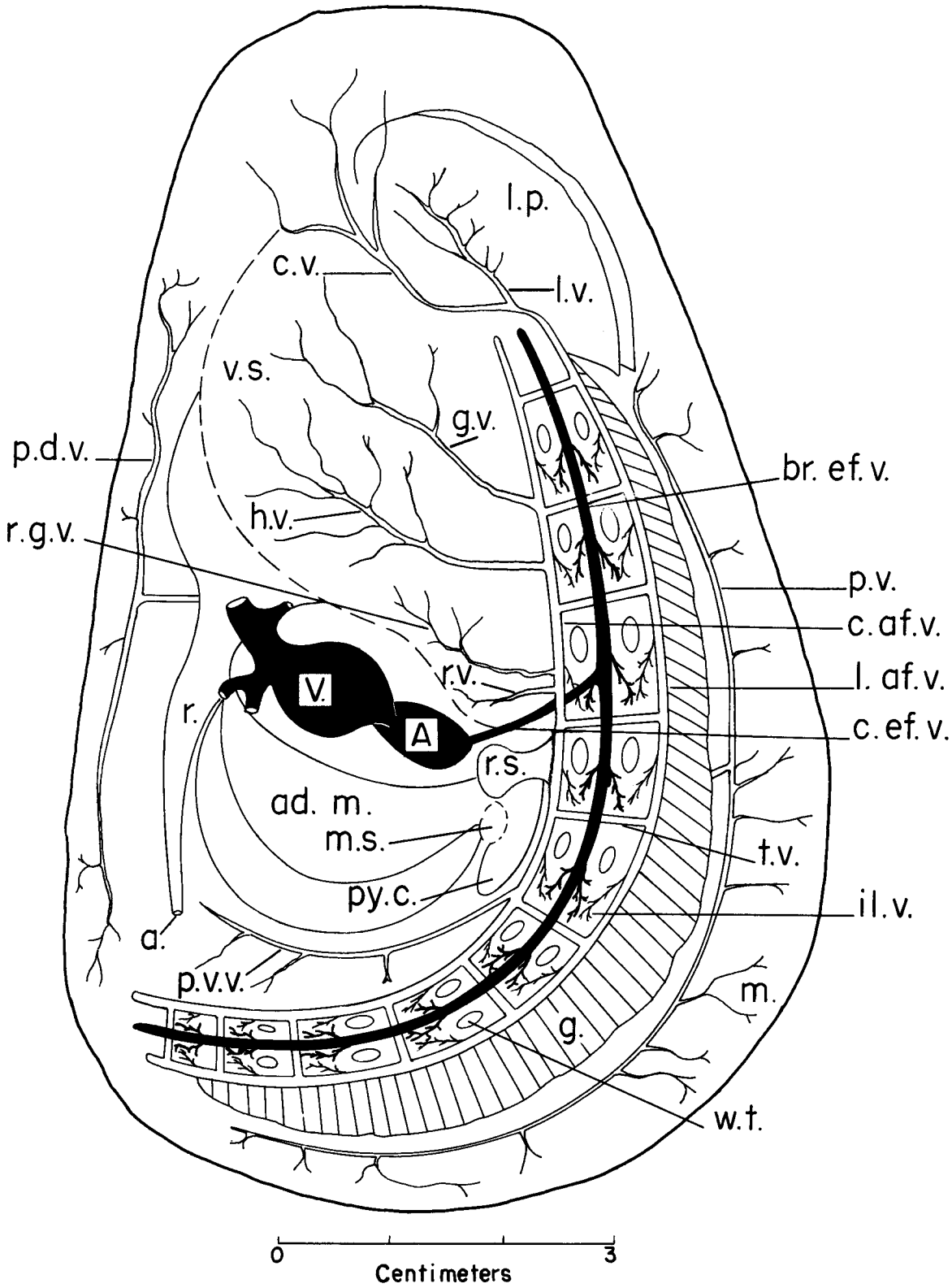

FIGURE 233.—Diagram of the venous system of *C. virginica* viewed from the right side. The right demibranch is open and pulled out to show the water tubes and the vessels of the descending and ascending lamellae. The left demibranch is not visible. Vessels carrying oxygenated blood are shown in solid black; others are open. The diagram

be seen from the surface. In sexually mature and "fat" oysters they are obscured by the deposits of glycogen and by the accumulation of sex cells. The paired lateral afferent veins (l.af.v.) are of smaller diameter than their common partner. They are located along the axis of the outer ascending lamella where the lamella fuses with the mantle lobe. The lateral afferent veins receive the blood from the mantle through the pallial veins (p.v.).

At regular intervals the common afferent vein is connected with the lateral veins by short transverse (horizontal) vessels (t.v.). These vessels can be seen in injected preparations of the gills and in sectioned material. The communication between the horizontal vessels in the gill tissues is maintained by means of vertical vessels which emerge from the walls of the three afferent veins as a series in a double row, one following the inner and the other the outer lamella of the demibranch. At each interfilamentar shelf the vertical vessels empty into a lacuna and eventually into the tubes of the gill filaments. There is no special path for the return of the blood from the interfilamentar lamellae and the tubes because the filaments end blindly. The walls of the common afferent vein contain a layer of elastic fibers arranged circularly; they are scarce in the walls of other veins. Endothelium is absent in all these vessels. The walls of the vertical vessels of the lamellae have a layer of muscular fibers which are able at intervals to constrict the lumen of the vessels along their length. In this way the flow of blood inside the gills is regulated (Elsey, 1935).

The blood channels in the interlamellar junctions are in communication with the vertical vessels and provide for the passage of blood from one lamella to the other. This rather inefficient circulation of the blood in the gill vessels is influenced by the contraction of the entire gill musculature and by contractions of the major afferent and efferent veins. The pulsations of these vessels have not been observed in vivo, but their histological structure suggests that they are capable of constricting their lumen. A tangential section of the common afferent vein preserved in a relaxed state (fig. 234) shows a well-developed layer of circular muscles flanked on both sides by thin bands of longitudinal muscles.

The system of efferent vessels comprises two short common efferent veins (c.ef.v.) which open into the auricles, a pair of branchial efferent veins (br.ef.v.) which run along the axis of the gill lamellae (fig. 73), pallial efferent veins (not shown in fig. 233), and the interlamellar and interfilamental vessels (il.v.) of the gills. The branchial efferent veins (br.ef.v.) run along the gill axis parallel to the branchial nerves (fig. 73) at the junctions of the ascending and descending lamellae. In their course they receive blood from the renal sinuses and empty into the common efferent vein. Blood which circulates in the mantle is carried to the heart through pallial sinuses and veins, but part of the blood from the posterior portion is drained back to the gills and to the branchial efferent vein (br.ef.v.).

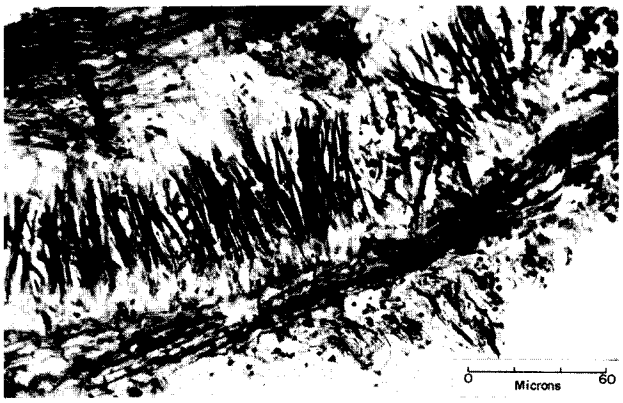

FIGURE 234.—Photomicrograph of a tangential section of the wall of the common afferent vein of *C. virginica* preserved in fully relaxed state. Narcotized in magnesium sulfate. Kahle, hematoxylin-eosin.

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was drawn from a number of preparations of partially injected venous system. Only the approximate position of various vessels is indicated. The diagram does not intend to show the actual appearance and distribution of veins. A—auricle; V—ventricle; a.—anus; ad. m.—adductor muscle; br. ef. v.—branchial efferent vein; c. af. v.—common afferent vein; c. v.—cephalic vein; c. ef. v.—common efferent vein; g.—gills; g. v.—gastric veins; h. v.—hepatic veins; il. v.—interlamellar veins of the gills; l. af. v.—lateral afferent vein; l. p.—labial palps; l. v.—labial vein; m —mantle; m. s.—mantle sinus; p. v.—pallial vein; py. c.—pyloric caecum; p. d. v.—posterior dorsal vein; p. v. v.—posterio-ventral vein; t. v.—transverse veins of the gills; r.—rectum; r. s.—renal sinus; r. g. v.—reno-gonadial veins; r. v.—renal vein; v. s.—visceral mass; w. t.—water tubes of the gills.

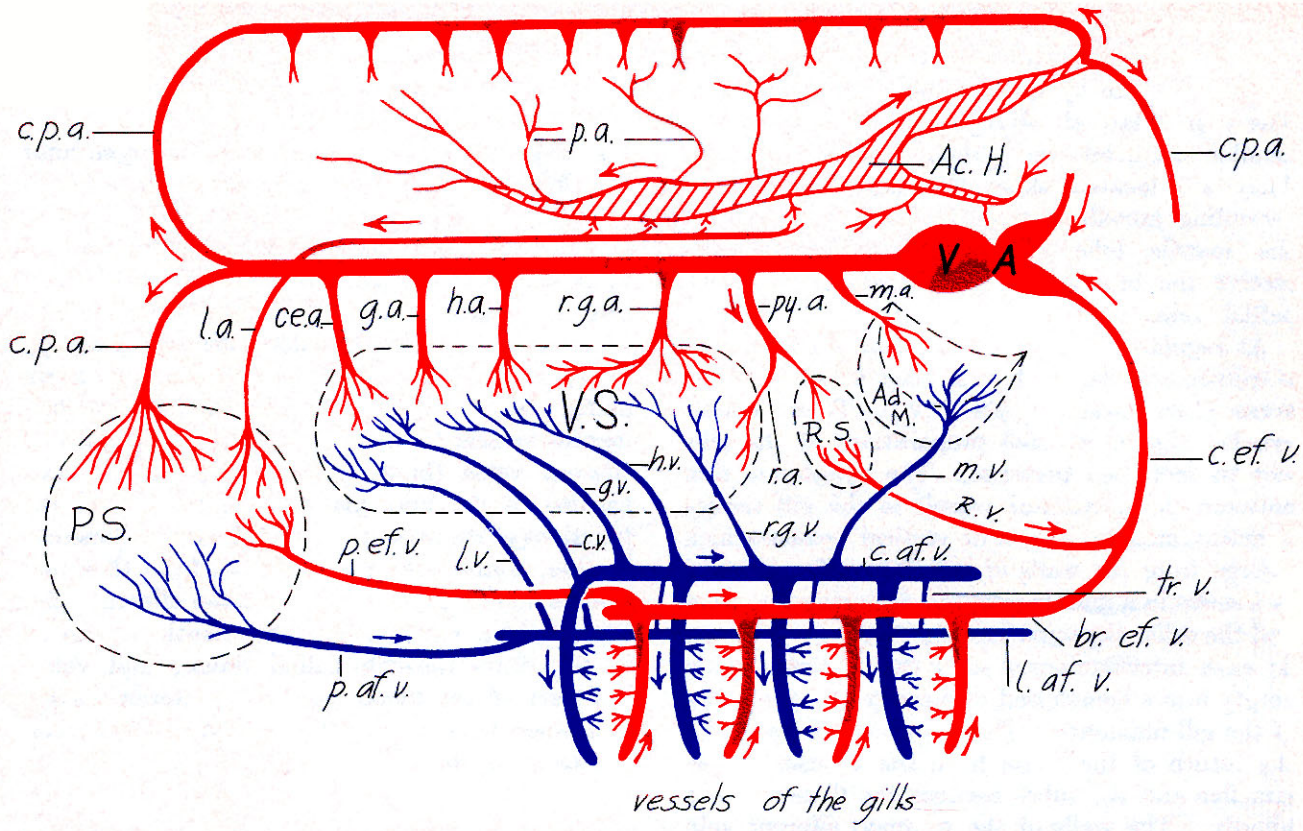

FIGURE 235.—Diagram of the circulation of blood in *C. virginica*. The position of various sinuses marked with capital letters is indicated by broken lines; only one demibranch and one accessory heart are shown. A—auricles; A.cH.—accessory heart of one side; P.S.—pallial sinuses; R.S.—renal sinuses; V—ventricle; V.S.—visceral sinuses; br.ef.v.—branchial efferent vein; c.af.v.—common afferent vein; c.ef.v.—common efferent vein; ce.a.—cephalic artery; cp.a.—circumpallial artery; c.v.—cephalic veins; ga.a.—gastric artery; g.v.—gastric vein; h.a.—hepatic artery; h.v.—hepatic vein; l.a.—labial artery; l.af.v.—lateral afferent vein; l.v.—labial vein; m.a.—adductor muscle artery; m.v.—adductor muscle vein; p.a.—pallial arteries; p.af.v.—pallial afferent vein; p.ef.v.—pallial efferent vein; py.a.—pyloric artery; r.a.—renal artery; r.g.a.—reno-geonadial artery; r.g.v.—reno-gonadial vein; r.v.—renal vein; tr.v.—transverse veins of the gills.

In visualizing the circulation of blood within the gills one must keep in mind the location of the five horizontal vessels at the top of the duplicated W-shaped junctions of the gill lamellae (fig. 73).

The course of circulation presented schematically in fig. 235 shows that the arterial blood goes to the sinuses (P.S., V.S., R.S.) and then is conveyed through the afferent veins to the gills and reaches the auricles via two common efferent veins. Some of the blood from the pallial sinuses (P.S.) and from the renal sinus (R.S.) bypasses the gills and is directly delivered to the auricles through the common efferent veins.

The deficiency in circulation caused by the presence of large sinuses is counteracted by the pulsations of radial vessels of the mantle and by a pair of accessory hearts (Ac.H.), which function independently of the principal heart of the oyster. The red and blue colors of the diagram show that only oxygenated blood fills the heart.

THE ACCESSORY HEART

The accessory heart is a paired tubular structure along the inner surfaces of the right and left mantle folds where they join together to form the cloacal chamber. Its position on the wall of the cloaca and its relation to the adjacent organs are shown in figure 236 drawn from life.

The accessory heart of the oyster is not the simple tubular structure described by Hopkins (1934, 1936) and Elsey (1935). It consists of three branches of almost equal size, joined together at a common center (fig. 237). The entire structure has the shape of the letter Y. The lower or ventral branch (v.br.) extends along the