Rarely are the capillaries equally distended throughout a given field, and frequently the corpuscles will appear to remain stationary in one or more of them for a brief period and then to move on again. The whole picture manifestly shows that the passage through the capillaries is attended with considerable difficulty. The resistance to the flow of the blood caused by friction is small in the large arteries; it increases gradually as they divide, but receives its chief and most important addition in the minute arteries and capillaries, especially in the former, on account of the flow being more rapid in them. This constitutes what is so frequently referred to as the "peripheral resistance" and is one of the important physical factors in explaining the circulation.

The high pressure and pulsatile flow in the arteries, the steady stream through the capillaries, and the low pressure and uniform flow in the veins can all be explained on purely physical principles. In the systole of the ventricle (1), driving a certain quantity of blood into the aorta at regular intervals and with a certain force; the long stretch of elastic branching arteries (2), and the peripheral resistance (3) are provided all the conditions which were shown above to be necessary for the conversion of an intermittent into a continuous current, while the veins afford a free and easy passage for the blood back to the heart when the greater part of the energy imparted by it to the blood has been expended in passing the peripheral resistance. The circulation in the vessels at any moment represents a state of dynamic equilibrium which is subject to constant variations. The energy of the heart's action may be altered by variations in the rate, volume, and force of its strokes. Again, the peripheral resistance may vary in consequence of changes in the calibre of the vessels. Constriction on their part over any considerable area will raise, widespread dilatation will lower, general arterial pressure.

Changes in both heart and arteries are constantly taking place, and indeed are necessary and essential. A fixed and rigid condition of the circulatory apparatus, devoid of adjustments within wide limits to adapt the blood flow to the varying needs of the several organs, would be worse than useless; it would be positively harmful. The mechanisms by which these changes are effected will be considered later on.

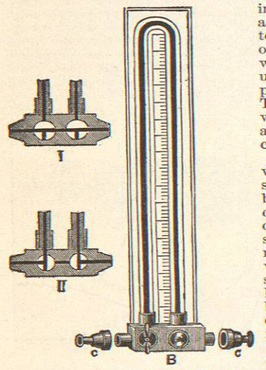

FIG. 1335.—Volkmann's Hæmodromometer. I, Blood flows from artery to artery; II, blood must pass through the glass tube of B; c, c, cannulæ for the divided artery.

A third factor which may exert some influence on the blood pressure is the quantity of blood contained in the vessels. The amount normally present would be able, in a state of static equilibrium, the heart being at rest, to produce a uniform mean pressure of only 5 to 10 mm. Hg throughout the vascular system in dogs.[6] But during life the heart maintains a continuous circulation by accumulating the blood in the arteries under a positive pressure, thanks to the peripheral resistance and the elasticity of their walls, while the veins are emptied to a corresponding extent and the pressure within them reduced below the mean. Experimental investigation as to what would be the effect of increasing or diminishing the quantity of the circulating medium has shown that variations in both directions and within considerable limits have a surprisingly little effect on the arterial pressure, compensating mechanisms being provided for quickly restoring it to, and preserving it at, the normal level. These mechanisms are manifold and complex, and a brief enumeration is all that can be attempted here. Taking the case of a copious intravenous injection, say of defibrinated blood or normal saline solution, the increased volume of blood is first provided for by relaxation and dilatation of the vessels. The veins especially expand to accommodate the increasing mass, and when the infusion becomes large enough the main veins are seen to be overfilled and the liver becomes as hard as a board. By this means and by partial failure of the heart's action from overdistention of its cavities the blood pressure tends to remain at or near the normal. More permanent relief, however, is obtained by other means and with striking rapidity. Transudation into the tissues removes a part of the injected fluid from the vessels, while the secretory activity of the kidneys and intestinal mucosa is enormously heightened and thus disposes of another portion. The character of the injection naturally exercises a decided influence on the relative share of these several mechanisms in the total result. In the case of hemorrhage, of course, the reverse series of changes are noted. There is a more complete emptying of the heart at each systole, a general contraction of the vessels, a restricted secretion from the kidneys, salivary and other glands, and a rapid absorption of lymph into the vascular system.

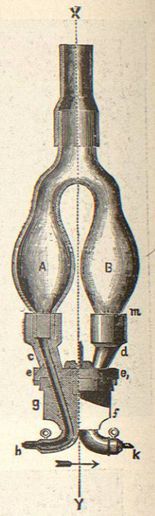

FIG. 1336.—Ludwig and Dogiel's Rheometer. X, Y, Axis of rotation; A, B, glass bulbs; h, k, cannulæ inserted in the divided artery, e, e, rotates on g, f; c, d, tubes.

In conclusion, two general facts in regard to the blood pressure deserve special mention: First, its remarkable constancy under ordinary circumstances in spite of many modifying influences; second, the important and extensive variations, seemingly out of all proportion to the exciting causes, occasionally produced by trifling and insignificant stimuli.

3. Velocity of the Blood Current. In considering the rate of flow in the vessels it is important to distinguish between the average velocity for a given period of time and the actual velocity which constantly oscillates above and below this mean with every phase of the heart's beat and with every variation of the peripheral resistance.

Volkmann first measured the mean velocity in the arteries in 1846 with his hæmodromometer.

The principle of the method is simply to determine the volume of blood flowing through an artery in a given period and dividing by its cross section. The quotient represents the length of a cylindrical column of blood with a base equal to that cross section, and therefore measures the average velocity in that time. Ludwig afterward devised a more convenient instrument, the stromuhr or rheometer, by means of which numerous data have been collected.

The results are not entirely exact, on account of many imperfections in the method, but they may be regarded as approximately true. Even in the course of a single experiment the mean velocity in the carotid or other

large artery is found to vary considerably for reasons which will be stated presently. Disregarding these variations for the moment, the rate of flow in the carotid of the dog or horse, and probably also of man, may be placed at from 300 to 500 mm. per second. The rate diminishes in the smaller arteries the further one goes out from the heart, and becomes slowest in the capillaries. As measured in the web of a frog's foot under the microscope, it is about 0.5 mm. per second. There are reasons for supposing it somewhat greater in the capillaries of mammalia, where it has been estimated at about 0.8 mm. per second. On leaving the capillaries the flow is quite slow in the venous radicles emerging from them, but it increases as these join into larger trunks, until finally it attains a velocity approximately equal to one-half that with which it started in the aorta. These changes in the mean velocity along the system are determined according to one of the simple laws of hydrodynamics stated in the preceding section. A continuous circulation in a closed system implies that an equal quantity of fluid passes each cross section in a given time. Now the quantity which passes may be expressed as the product of the velocity by the cross section, thus $Q = V \times A$. And similarly $Q' = V' \times A'$, etc. But Q, Q', etc., being equal to one another $V \times A = V' \times A'$, hence $V : V' :: A' : A$, or the velocity varies inversely as the cross section along the system.

It was stated above that the arterial tree widens out steadily from the aorta to the capillaries (see Fig. 1330). Comparison of the respective velocities at these two points now shows that the amount of this increase must be something like five hundred times. For a graphic representation of the velocities in the vascular system and the relation to the sectional area see Fig. 1334 above.

This dependence of the velocity on the width of the bed applies of course only to the general rate of the whole system, and many special and transient variations constantly occur. Since the real cause of the flow from one point to another is the difference of pressure between those points, the actual velocity at each moment must depend on the amount of this difference of pressure and the resistance against which it is working. A change in either of these factors will necessarily affect the velocity. An illustration of this is found in the following observations on the rate of flow in the carotid of the dog by Dogiel:

Weight in kilograms.	Period of observation, in seconds.	Rate of flow in millimetres, varied.
23.3	80	From 733 to 349
12.1	127	" 520 " 243
3.2	63	" 458 " 411
3.6	45	" 339 " 204

The slowing of the current observed in each case is not referable to incipient coagulation or other experimental error, at least not to any great extent; on the contrary, it is due to changes in the condition of the peripheral area supplied by the carotid artery. Blood pressure remaining the same, the rate of flow becomes essentially a function of the peripheral resistance. The temporary interference with the circulation during the adjustment of the instrument destroys the tone of the vessels beyond, and lowers the resistance, so that the observed rate is at first too high. With circulation re-established the normal calibre is regained, the resistance increases, and a corresponding retardation is observed.

It will be noticed that this quickening influence of a local dilatation is in no sense a contradiction to the general principle that the rate of flow is inversely proportional to the width of the bed. The local change is not extensive enough to affect the total cross section of that level appreciably, but by reducing the resistance along one of numerous alternative paths increases the flow in that direction at the expense of the others. In such cases the flow of blood through the widened artery or arteries is for the time being increased in rapidity, not only in spite of, but actually in consequence of the artery being widened. The influence of changes in the blood pressure on the velocity is shown in the effect produced on the latter by each beat of the heart. When the velocity of flow is recorded by means of an instrument which will register its finer variations, say

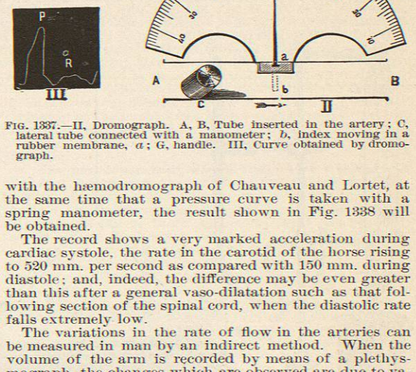

FIG. 1337.—II, Dromograph. A, B, Tube inserted in the artery; C, lateral tube connected with a manometer; *b*, index moving in a rubber membrane, *a*; G, handle. III, Curve obtained by dromograph.

with the hæmodromograph of Chauveau and Lortet, at the same time that a pressure curve is taken with a spring manometer, the result shown in Fig. 1338 will be obtained.

The record shows a very marked acceleration during cardiac systole, the rate in the carotid of the horse rising to 520 mm. per second as compared with 150 mm. during diastole; and, indeed, the difference may be even greater than this after a general vaso-dilatation such as that following section of the spinal cord, when the diastolic rate falls extremely low.

The variations in the rate of flow in the arteries can be measured in man by an indirect method. When the volume of the arm is recorded by means of a plethysmograph, the changes which are observed are due to variations in the size of the arteries and indicate corresponding variations in the rate at which the blood enters them, the outflow from the veins being uniform. From the volume curve (see Fig. 1340) Fick constructed the curve of velocity, and found that it resembles closely the curve of Lortet obtained by the direct method on the horse.

4. The circulation in the veins is influenced by a number of accessory factors, whose combined effect renders it

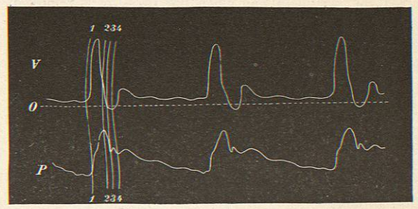

FIG. 1338.—Curves of Velocity (V) and Pressure (P) from the Carotid of the Horse. (According to Lortet.) The lines 1, 2, 3, 4 mark corresponding points in the two curves. The ventricular systole begins at 1; the diastole begins somewhere between 3 and 4.

somewhat irregular perhaps, but which on the whole promote and quicken it. Among these secondary helps to the circulation the following are usually included, although the fact must not be lost sight of that the real cause of the venous flow is the ventricular stroke, enough of whose energy remains, after driving the blood past

the peripheral resistance, to carry it along the veins back to the heart.

a. Aspiration of the Thorax.—It has been stated above that the pressure in the great veins near the heart sinks below zero or atmospheric pressure. This is due to the permanent negative pressure in the thorax; and since every inspiration increases and every expiration diminishes the amount of this negative pressure, the breathing movements assisted by the arrangement of the valves constitute a virtual pump, which sucks the blood from the venæ cavæ and promotes the flow of blood in the direction of the normal circulation.

β. Contraction of the Skeletal Muscles.—Many of the veins possess valves which open freely toward the heart but effectually block the passage of the blood in the opposite direction. External pressure on such veins forces their contents along toward the heart while they fill again from the peripheral end when the pressure is removed. Something of this sort occurs in all bodily movements in which the muscles by their alternate contraction and relaxation provide the intermittent compression. It has been suggested that the pulsation of the arteries may similarly possess a mechanical function from the regular rhythmic shocks against the closely applied venæ comites.

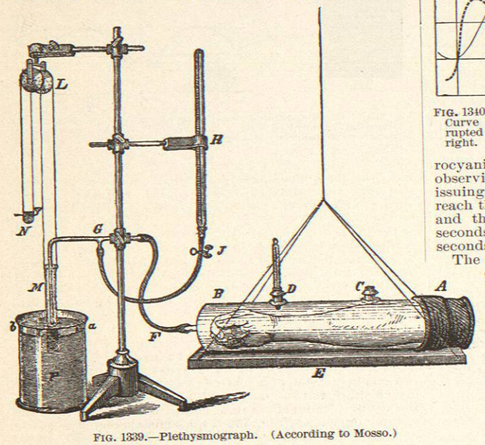

FIG. 1339.—Plethysmograph. (According to Mosso.)

γ. Posture.—Changes in the position of the limbs and body also affect the pressure in the veins by altering the tension of their walls (Braune). Thus in walking, the femoral vein where it passes under Poupart's ligament collapses when the leg is rotated backward and outward, but fills again when the leg returns to its first position, the whole movement making a kind of suction and pressure apparatus.

δ. Gravity.—In the upright position of the body, the difference of hydrostatic pressure in the vessels of the head and feet amounts to 1.75 metres of water or 129 mm. mercury. This naturally opposes an upward current and assists a downward one, so that the effect on the arteries and veins is equal but opposite in direction. The result is that it modifies the distribution rather than the rate of flow of the blood, as may be seen on comparing the pale, anæmic condition of the hand when the arm is raised with its full, red appearance when the arm hangs down.

When from any combination of circumstances the blood returns to the right side of the heart in excessive and dangerous amounts the liver is supposed to play the rôle of a shunt by taking up a considerable quantity of blood, much in the same way as it does after profuse intravenous injections (see above).

5. The Time of a Complete Circulation. This was first determined by Hering (1829) by injecting potassium ferrocyanide into the central end of the jugular vein and observing the time of its first appearance in the blood issuing from the peripheral end of the same vessel. To reach the latter point a complete circuit had to be made, and the time thus determined was found to be 31.5 seconds in the horse; 16.7 seconds in the dog; 7.79 seconds in the rabbit.

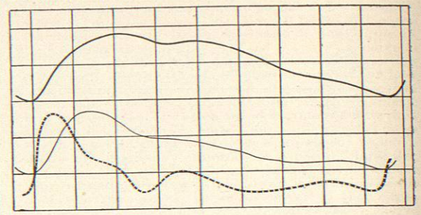

FIG. 1340.—Plethysmographic Curve (Upper Continuous Line), Pulse Curve (Lower Continuous Line), and the Velocity Curve (Interrupted Line) in Man. (According to Fick.) To be read from left to right.

The time is not much affected by the length of the course in the same animal, because the difference between the longest and shortest path would be made up mostly of the larger trunks, where the rate of flow is most rapid. Dividing the time in seconds given above by the length of the cardiac cycle for the corresponding animal, it appears that the circulation time covers from 26 to 28 heart beats. The same rule applied to man would give as the circulation time:

$$27 \times \tfrac{4}{5} = 22.5 \text{ seconds.}$$

These measurements possess only a relative value, because the method does not take into account the error arising from the diffusion of the salt during the time of its transit and from the fact that the first appearance of the salt is really determined by the maximal or axial velocity of the current. The mean time should therefore be taken as at least twice as great as the observed time.

6. The Pulse. The intermittent character of the circulation in the arteries has been referred to in more than one connection. With each systole of the ventricle a temporary increase of pressure, size, and velocity above the mean results from the sudden accession of the extra volume of blood to that already present in this part of the system. The pressure oscillations are readily felt in superficial arteries, and the resulting expansion has long been known technically as the "pulse." It can be graphically recorded, as Vierordt first showed (1855), and Marey soon afterward (1860) devised his well-known sphygmograph in order to obtain an accurate reproduction of the movement in all its phases. When recorded

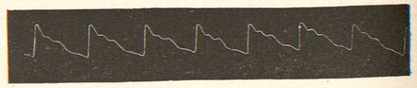

FIG. 1341.—Sphygmogram taken from the Radial Artery with a Marey Sphygmograph. (According to Langendorff.) Read from left to right.

on a travelling surface, the curve or sphygmogram like that represented in Fig. 1341, is obtained.

The rise of the curve is quite sudden, while the downstroke is more gradual and interrupted by one or more secondary waves. The phenomenon of the pulse is entirely explained by the interaction of a number of physical factors, resulting as it does from the sudden injection of the contents of the ventricle into the elastic arteries, which are already full and whose outlet is partially blocked by the peripheral resistance. The actual height and form of a given pulse-curve depends much on the adjustment of the instrument; but if a series of curves are taken from different arteries under the same conditions, it is found that their character undergoes definite changes according to the distance of the corresponding arteries from the heart. The carotid curve, for example, rises higher and more suddenly than the radial curve; this in turn is higher and steeper than the tibial pulse curve. In other words, the pulse curve grows lower and flatter toward the capillaries, and under ordinary conditions it disappears entirely by the time they are reached.

The more rapid rise and greater amplitude of the pulse near the heart is the immediate result of the suddenness with which the ventricle is emptied. The additional quantity of blood thus thrown into the arteries starts a wave of positive pressure down the arterial tree, but on account of the resistance ahead it first expands the aorta and its largest branches. The distant arteries, therefore, do not feel the full force of the systole as would be the case in a system of rigid tubes; when the wave reaches them it has been damped by a longer or shorter stretch of elastic arterial wall. The recoil of this wall is less sudden than the contraction of the heart, and so the expansion of the successive segments becomes more gradual.

The loss in amplitude is also readily explained by the elastic character of the vessels. The energy of the wave is gradually spent in spreading over an increasing area of elastic tubes every portion of which for a brief period remains distended a little more than before. Each section of artery thus makes a levy on the extra expansion as it is passed along, and transmits it to the next section diminished by that much. This is, in fact, the only way in which the mean arterial pressure can be produced and maintained. This process, of course, is further accelerated by the rapid increase of elastic surface as the bed widens, not to mention the loss resulting from partial reflections with consequent interferences of the wave at every bifurcation of the vessels.

Under certain circumstances the pulse may pass through the capillaries into the veins beyond. The increased vascularity of organs during periods of heightened functional activity is secured by a wide dilatation of their small arteries. The peripheral resistance, so essential in bringing the elasticity of the arteries into play, is thereby locally diminished and the dilated vessels cease to do their share toward destroying the remnant of the pulse which comes down to them from the larger arteries. The blood flows more readily through the widened channels, and frequently issues from the corresponding veins in a rapid, pulsating stream.

This true venous pulse, appearing under special conditions in the small peripheral veins, must be distinguished from the pulsations which are sometimes observed in the great veins near the heart. The latter are caused by changes of pressure transmitted backward from the heart and thorax, and are often described as constituting a "venous pulse."

The special features of the pulse curve, including the number and character of the secondary elevations, are interesting on account of the suggestions and indications they give concerning the action of the heart, the condition of the vessels, and the state of the blood pressure. For these the reader is referred to the special article on the pulse (see *Sphygmography*).

The rate of transmission of the pulse wave, which must not be confounded with the onward movement of the blood itself, is found by taking simultaneous records of the pulse from arteries unequally distant from the heart. If the distance of the two points from the heart is known, the rate of propagation is easily calculated, and it has been found to vary normally between 5 and 10 m. per second. The rate depends mainly on the degree of rigidity of the arteries, the wave travelling slowly when the mean blood pressure is low and in the soft distensible arteries of children, while its velocity is increased by high arterial pressure and the hardening of the arteries in old age. In any case the pulse wave moves many times faster than the blood itself, being, as we have seen, merely a wave of pressure which is started in the aorta and which travels over the blood exactly as a water wave passes over the surface of a stream independently of the rate and direction of the latter's own current. The ordinary pulse curve does not return to the base line from which it started until the rise due to the next wave begins, indicating that the wave requires the entire time of a cardiac cycle to pass a given point. With a heart beating at the rate of 72 per minute, this would equal $\frac{4}{5}$ second, and taking the velocity of the wave at 6 m. per second, we would have as the length of the wave $6 \times \frac{4}{5} = 5$ m. This is several times greater than the distance of the farthest capillaries from the heart, showing that the beginning of the pulse wave is lost at the periphery some time before the end of it leaves the aorta.

7. The Pulmonary Circulation and its Relation to the Systemic.

a. The maintenance of the circulation as a whole implies that the average output of the two ventricles of the heart is the same. Considering the relatively small capacity of the pulmonary circuit, which normally contains only one-twelfth of the total blood (Hegel and Sprehl), the time spent in this part of the vascular system must be much shorter than that taken for the major circuit. The pressure in the pulmonary arteries has been measured upon various animals by numerous observers with very discordant results; in general it seems to range between one-third and one-fifth of the aortic pressure, being given at 20 to 25 mm. Hg. for the dog.[7] This indicates a low resistance in the pulmonary capillaries, and the great distensibility and slight tone of the pulmonary vessels are perhaps the most striking features of the lesser circulation.

The influence of the respiratory movements on the pulmonary circulation is a complex one and depends on several factors. The expansion of the lungs in a normal inspiration has been shown to dilate the pulmonary vessels. This diminishes the resistance and tends to lower the pressure in the pulmonary artery. At the same time the output of the right ventricle is augmented by the increased aspiratory action of the thorax, which we have seen hastens the return of the blood to the right auricle and so increases its diastolic distention. This factor would tend to raise pulmonary pressure, and actual measurement alone can determine which of the opposing tendencies will have the upper hand. In certain cases, at least, it has been shown that the dilatation more than compensated for the increased output of the heart and the pressure in inspiration fell (Talma). In natural expiration the conditions are reversed and the pressure again rises. The course of events is entirely different during artificial respiration, when the lungs are inflated under positive pressure with the thorax open.

The great range of adaptibility, as regards its capacity, shown by the pulmonary circuit deserves special mention. In dogs about three-fourths of this vascular area was occluded without producing any effect on aortic blood pressure (Lichtheim). Since the systemic venous pressure in this experiment also remained normal, the amount of blood passing through the lungs was evidently not reduced by this extensive narrowing of the path. The pressure in the remaining pulmonary artery was, however, raised by the operation, and it is supposed that this rise of pressure distended the open channels sufficiently to explain the undiminished flow through the lungs. It is doubtful whether this observation, made under abnormal conditions, can be applied generally: at least in rabbits with pleural cavities intact clamping of one pulmonary

artery caused a decided drop in the systemic blood pressure. It is merely given to illustrate the possibilities of capacity change in the lung vessels. They can easily be distended so as to retain enough blood to lower the carotid pressure to zero, as shown in some experiments on differential respiration.[8]

The pulmonary circulation is affected not only by the condition of the systemic circuit at its venous end, but

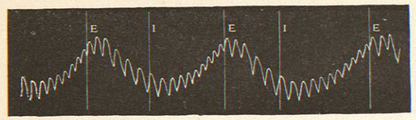

FIG. 1342.—Carotid Blood-Pressure Tracing of Dog. Vagi not divided. I, Inspiration; E, expiration. (Stirling.)

also by that of the arterial side. An excessive transfusion or a powerful general constriction may so increase the work of the left ventricle that it fails to empty itself completely at each systole. A stagnation extending from the left ventricle to the left auricle and thence to the pulmonary veins results, and the pressure in the lesser circuit is raised. The threatened danger from this source to the right ventricle with its thin walls is averted by the power of expansion which has been dwelt upon so fully; so that the lungs may be said to play the same rôle in regulating the blood flow to the left ventricle as that ascribed to the liver for the right ventricle (Tigerstedt).

β. Respiratory changes of blood pressure in the systemic arteries. The pulmonary circulation reacts upon the systemic pressure by modifying the inflow to the left auricle. It has been stated that the pulmonary vessels expand during inspiration and constrict during expiration. While the distention is in the act of developing, the blood flow is momentarily retarded and the left heart receives a smaller charge; but once the maximal dilatation has been reached, the resulting diminution of the resistance will augment the flow and the left heart receives a more abundant supply. On the contrary, expiration primarily increases the flow by narrowing the calibre of the dilated vessels and forcing on their contents, and secondarily diminishes it by increasing the resistance. The effect of these changes in the lungs on the pressure in the aorta depends largely on the rate and depth of the respiratory movements. When these are very shallow, no effect whatever is observed in the systemic arteries. When they are both quick and strong, only the primary effects appear, and the aortic pressure falls in inspiration and rises in expiration. The typical result is seen only in ordinary breathing when the rate is slow enough to permit both the primary and the secondary changes to produce their proper effect (see Fig. 1342). In this case the pressure first falls during inspiration to a minimum (primary effect) and then rises again (secondary effect); in the succeeding expiration the rise continues for a short time to the maximum (primary effect) and then the pressure falls again (secondary effect). The main effect of inspiration, therefore, is to raise the pressure and the main effect of expiration to lower it. The curve frequently shows also changes in the rate of the heart which beats slower during expiration and faster in inspiration. These changes are brought about through nervous influences that must be considered later, and it need only be stated in this connection that they are not of sufficient moment to affect the general course of the phenomena here described.

C. *The Mechanics of the Heart.*—The circulation of the blood through the vessels is maintained by the energy of the beating heart. Before considering the causes which generate this beat and which determine its character, force, and frequency, it will be convenient to take up a number of mechanical problems which the heart presents when regarded as a valvular pump in action.

1. External Phenomena of the Beat. *α.* When the living heart is observed directly, either in a mammal, or, better still, in a cold-blooded animal like the frog or terrapin, each beat is seen to begin with a contraction of the great veins in the neighborhood of the heart. The contraction proceeds as a peristaltic wave whose progress can be readily followed in the cold-blooded heart as it spreads first over the auricles and then over the ventricles. In the mammal the wave-like character of the beat is difficult to recognize on account of the great rapidity of the movement. Here the auricles appear to contract suddenly over their whole extent, the auricular appendages being drawn inward and the rest of the wall pressing toward the auriculo-ventricular orifices, without, however, completely obliterating their cavities. As soon as the contraction of the auricles is completed the ventricles immediately follow with theirs. The contraction of each section of the heart is spoken of as its systole, and the succeeding relaxation as the diastole. A complete beat or "cardiac cycle" comprises an auricular and a ventricular systole and a heart pause, during which all parts of the heart are at rest. The systole and diastole of the two auricles are perfectly synchronous in a normal heart as are also those of the two ventricles, the synchronism resulting from the peculiar arrangement of the muscle fibres in their walls.

In the auricles these consist of a superficial set common to both sides and of deeper fibres proper to each. The superficial fibres run transversely, being best developed on the anterior surface, and some of them turn in at the auricular septum. Of the deep fibres, some pass over the auricle in the form of an open loop to be attached at both extremities to the auriculo-ventricular ring; these are called the "looped" fibres. Others, known as "annular" fibres, encircle the auricular appendages and surround the openings of the venæ cavæ and pulmonary veins.

The complex nature of the arrangement of the fibres in the ventricles appears from the fact that a vertical

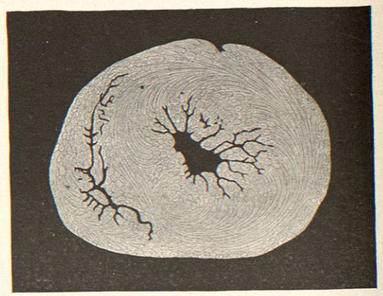

FIG. 1343.—Cross Section of a Completely Contracted Human Heart, at the Level of the Lower and Middle Third. (According to Krehl.)

section of their walls taken at almost any point shows the fibres on the outer surface crossing the direction of those on the inner, while between these every intermediate direction may be represented. The left ventricle possesses a much more powerful musculature than the right ventricle, but, as in the case of the auricles, a large number of fibres are common to both; in fact, the outer wall of the right ventricle is so largely made up of fibres

coming from the left that the former appears in sections of the heart like a cleft or pocket in the wall of the latter (see Fig. 1343).

Schematically the fibres may be described as falling into a number of more or less well-defined groups according to the general direction of their course. The superficial oblique fibres constitute the first of these groups. They take their origin in the fibrous rings at the base of the heart and have a general trend obliquely downward and to the left. They form a thin, superficial stratum on the wall of the right ventricle, but on the left side they increase greatly in number, and on reaching the apex many of them turn in to form the whorl, whence they ascend on the inner surface to terminate in the papillary muscles and chordæ tendineæ or to pass all the way up to the auriculo-ventricular ring.

Between the inner and outer layers of oblique fibres a second group proper to the left ventricle is interposed. The fibres of this class are more or less transverse in direction and may be described as the circular fibres of this chamber, of the thickness of whose wall they make

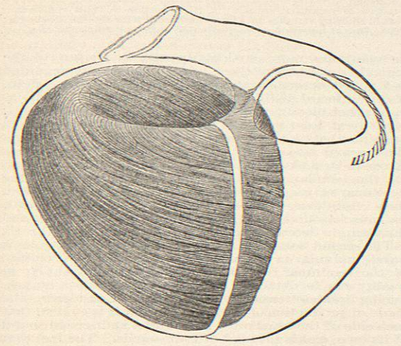

FIG. 1344.—Intermediate Layer of Circular Fibres of the Left Ventricle. The outer and inner fibres have been removed, and the outlines of the entire heart schematically indicated. (According to Krehl.)

up a considerable share. Taken as a whole, they form a truncated hollow cone through the narrow apical opening of which the fibres of the whorl enter.

The right ventricle also contains fibres of its own, and one set of these can be traced on the inner surface of its outer wall. These take their origin at the upper border of the septum, follow this down for a variable distance, then turning toward the lumen, stretch across to the outer wall. There they ascend in bundles, forming trabeculæ and columnæ carneæ, and end partly as papillary muscles, partly by short tendons in the fibro-tendinous ring at the base.

The portion of the right ventricle known as the conus arteriosus has its own special fibres, which in its outer wall arrange themselves into an inner longitudinal and an outer circular layer, the latter merging insensibly into the superficial oblique fibres of the general surface.

The anatomical basis for the observed synchronism of the two sides of the heart is thus provided for in the large number of fibres which pass across and are common to the chambers of the same name. The progressive character of the contraction, not easily recognized by direct observation, can be demonstrated by means of the accompanying electrical phenomena, which are similar in the case of the heart to those observed in skeletal muscle. An electrical change or current of action accompanies each heart beat; in the frog's heart the variation shown by the capillary electrometer is diphasic.

For the human heart the later work seems to show a triphasic current, first base negative to apex, then apex

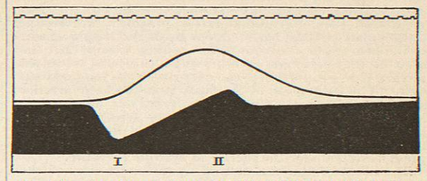

FIG. 1345.—Simultaneous Photogram of a Single Beat (Black Line) and of the Accompanying Electrical Change, indicated by the level of the black area, which shows the varying level of mercury in a capillary electrometer. I, First phase, base negative to apex; II, second phase, apex negative to base. (Waller.)

negative to base, and then again base negative to apex. The accompanying curve has been constructed to represent these electrical variations.

From them it is inferred that the contraction begins at the base of the heart and advances toward the apex; that for a time (the interval S to T in the curve) the entire ventricle is uniformly contracted; finally, that the contraction ends by disappearing first at the apex. It evidently follows the recurrent path of the superficial oblique fibres, beginning and ending at the auriculo-ventricular groove. Another deduction drawn from the electrical phenomena of the heart is that its contraction is comparable to a simple muscle twitch and is not to be regarded as a summated or tetanic contraction.

β. Changes in the form and position of the heart. In diastole the heart can hardly be said to possess a definite shape of its own, although it is frequently described as a hemi-spheroid with a rounded apex and somewhat flattened anterior and posterior surfaces. On account of its soft, flabby condition its shape changes according to the position of the body under the influence of its own weight. Not so in systole; the heart then becomes hard and rigid and its shape approaches that of a regular cone with a circular base. The changes in the three diameters during this transition must depend on its previous form in diastole. Under normal conditions in which the heart is supported by the lungs as by an elastic cushion, there is probably no extensive flattening in any one plane. In this case entrance into systole would involve a shortening in all directions while the apex became sharper or more pointed.

When the heart is examined in the ordinary way in animals with open thorax, it unquestionably flattens

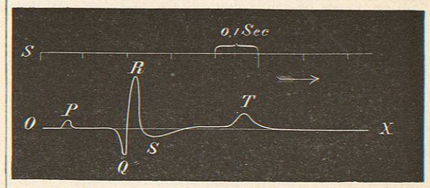

FIG. 1346.—Diagrammatic Curve of the Action Currents of the Human Heart. (Einthoven.) *R*, Beginning of ventricular contraction; *S*, apex negative to base; *T*, third phase, base negative to apex. The small diphasic variations represented by *P* and *Q* are caused by the auricular systole.

out during diastole, the transverse diameter elongates at the expense of the antero-posterior, and the base takes