RHEOGRAPHY

BY

K. POLZER, F. SCHUHFRIED, AND H. HEEGER

From the Cardiac Department of the Hanusch-Hospital, Vienna

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Rheography records the changes of electrical conductivity of the human body caused by the blood flow and is practically applicable to any part of the body. This study has been undertaken to show the clinical value of this new method. The striking changes of the rheographical curve in various clinical conditions are described.

Efforts have been made for some time to register fluctuations in blood flow by means of alternating current conductivity measurements (Holzer *et al.*, 1945). By using an electronic amplifying apparatus it has been possible to increase the sensitivity of this method, named by us *Rheography*, to an adequate degree. The difficulty lies in the fact that fluctuations in blood flow, as they occur with the rhythm of the pulse wave, cause in general an alteration in conductivity of only about one per cent. Thus, the method only became routinely practicable when it became possible to record a rheogram of sufficient amplification and yet free from interference. It was found to be expedient to use large or small electrodes, according to the choice of the zone to be examined, and this requires conformity between the measuring apparatus and the size of the electrode. In this paper the practicability of this method in cardiovascular diagnosis as a result of advances in the physics of the problem will be demonstrated.

This method is applicable to every region of the body, yields strictly reproducible curves, is capable of standardization and calibration, and is practically independent of the pressure of the electrode. It causes the patient no discomfort that could play a disturbing role in the appraisal of blood flow disturbances.

TECHNIQUE

As a measuring device, the familiar Wheatstone bridge is adapted to the special requirements of rheography. In Fig. 1 the measuring device is shown. The vascular section of the patient is connected to one arm of the Wheatstone bridge. The patient must be considered electrically as a combination of resistance and capacitive reactance (Schaefer, 1940). The arm of the Wheatstone bridge (between points A and B) is therefore equipped with a variable resistor and a variable capacitive reactance. It is in this way possible to reconstruct the patient electrically. By variation of this arm of the bridge the patient's capacitive reactance and resistance can be measured. In particular the value of the resistance gives a guide to the volume of blood flow of the vascular section examined, when a peripheral rheogram is being recorded. In a further arm of the Wheatstone bridge lies a resistor that can be short-circuited by the aid of a balance key, the purpose of the which is to produce a certain defined imbalance. This is done for the following reason. At two points facing each other on the bridge voltage is fed in, and led off at two other points. The voltage led off is to be modulated by the fluctuations in resistance in the patient's circuit. In order to resolve such a modulation linearly a carrier voltage must be present that must be sufficiently large with respect to the modulation.

Calibration. A further important attachment for the calibration of the rheogram is the calibration key. In series with the patient lies a resistance, which through connection with further resistors can be decreased with the help of a key, whereby a control wave occurs in the curve. The height of the rheogram can thus be expressed in fractions of ohms. It was found to be expedient to make this calibration height variable,

FIG. 1.—Rheograph. (For explanation, see text).

since the height of the rheogram is exposed to large variations depending on the zone to be measured and the condition of the blood vessels. In general a height of 0.1Ω is recommended, in certain cases 0.5Ω . In special cases a control wave as large as 1Ω or as small as 0.05Ω may be desirable.

Balance of the Wheatstone Bridge. Because of its freedom from inertia, a magic eye, such as is used for supervising modulation in wireless, is recommended for checking bridge balance. In bridge balance, when the bridge yields only a small voltage or practically no voltage, the magic eye shows no deflection, and the unlit part is at its largest. After balancing of the bridge is accomplished the balance key is released. The bridge will thus be brought into imbalance and yields a voltage that is modulated by conductivity fluctuations in the patient. The voltage will be increased and rectified by an amplifier and then led to an electrocardiograph for graphic registration of the curve. Since fluctuations in conductivity are proportional to blood flow fluctuations, the rheogram can be used for recording such fluctuations.

Placing of Electrodes. The alternating current used for purposes of measurement is applied to the patient's body with the aid of two electrodes. By choice of the position of these electrodes one can determine the zone to be examined. One is thereby independent of specified lead-off points, as, for instance, must be sought in the determination of pulse curves. For appraisal of peripheral blood flow ring-shaped electrodes are recommended. In a sectional examination we speak of a longitudinal rheogram, whereas a lead-off on two corresponding points lying at the same level transversely through the extremity is designated by Kaindl (1954) a transverse rheogram.

Beside these peripheral leads it is possible, by appropriate placing of the electrode, to register intra-thoracic blood volume fluctuations, liver volume fluctuations, etc., which will be discussed below.

Tin-plated lead, which can be made to conform easily to the form of the body, is recommended for the electrodes. The electrodes are surrounded by a good absorbent material and painted with electrode paste or soap solution.

THE PERIPHERAL RHEOGRAM

For examination of the extremities the peripheral rheogram in the longitudinal lead arrangement is recommended. For this one lays two ring-shaped electrodes proximal and distal to the vascular section to be examined. For the purpose of an over-all diagnosis one can transcribe a rheogram over the entire extremity; it is equally possible, also, to examine sectionally, upper arm, lower arm, hand, and finger, or partial sections therefrom. A record of analogous sections of the leg is equally possible. In the evaluation of these curves it is noticed that the rheogram of both the upper arm and upper leg is relatively small, of the order of 0.05Ω , that the rheogram of the lower leg and similarly the lower arm is larger (approximately 0.1Ω), while the rheogram of the finger and similarly the toe is from 0.2Ω to 0.4Ω (Fig. 2). The cause of this lies in the fact that in more peripheral vascular sections the shunt through the tissues of a non-pulsating nature is relatively smaller.

The peripheral rheogram has a characteristically steep ascent, which occurs with the arrival of the pulse wave in the particular zone examined by the electrode. The descent of the curve is correspondingly slower; in normal cases elastic after-waves are generally seen. In analysing the peripheral rheogram, attention should be given, not only to the amplitude of the curve and the resistance, but also to deformations in the curve which give information about the elasticity of the vessel walls. A sluggish ascent with a broad curved peak, with low amplitude and a gradual down-slope, is seen primarily in the arteriosclerotic type of curve.

Fig. 3, for instance, shows the peripheral rheogram of the right (upper curve) and of the left lower leg (lower curve) of a 62-year-old patient with severe arteriosclerosis. The blood flow in the right leg was worse than in the left. Accordingly the rheogram of the left lower leg displays an essentially steeper ascent, the curve peak is sharper and there are small elastic after-waves. Because of the more obstructed blood flow in the right leg the rheogram shows a more sluggish ascent, reaching its maximum later. The peak of the curve is broad and there is a sluggish down-slope too and elastic after-waves are absent. The amplitude measured by means of the calibration (0.1Ω) is 0.12Ω , smaller on the right leg than on the healthier left leg, where it is 0.17Ω . Where vessel wall tone is decreased, we find an abnormally high curve, which shows little differentiation into parts, and the widened rounded peak is reached with delay in this type. Where vessel wall tone is increased, we see a relatively steep ascent of the curve, the peak is reached earlier, and the curve is decreased in amplitude. The curve shows a narrow peak, which is followed by several after-waves. The analysis of the rheographic curve, according to its form and amplitude, thus amplifies vascular diagnosis, and with it a more exact supervision of vascular therapy may be possible.

THE SKULL RHEOGRAM

By placing the electrodes at the glabella and the retromastoid region an insight into the elasticity relationships of the skull is obtained. The method can be used to supervise therapeutic measures. In particular, the success or failure of response to stellate ganglion block is reflected in the rheogram (Polzer and Schuhfried, 1950).

THE RHEOCARDIOGRAM

Holzer *et al.* in 1945, applying leads to the right arm and left leg, called the procedure rheocardiography. By this arrangement the conductivity fluctuations in the thorax as well as in the periphery are registered. In order to try and differentiate the two, Polzer and Schuhfried (1949) attempted to register the filling fluctuations of the thorax alone by placing the electrodes on the apex of the heart and the right shoulder. They call this method the thoracic rheocardiogram (rcg)

FIG. 2.—Electrocardiogram (lead II) and peripheral rheogram of a 30-year-old subject (calibration 0.05Ω): (a) of the upper arm; (b) of the lower arm; and (c) of the finger.

FIG. 3.—Electrocardiogram (lead II), and peripheral rheogram of the right and left leg of a 62-year-old male patient with arteriosclerosis (calibration 0.1Ω).

FIG. 4.—Electrocardiogram (lead II), thoracic rheo-cardiogram and phonocardiogram of a 26-year-old subject.

FIG. 5.—Lead II, thoracic rheo-cardiogram (upper curve) and rheogram of the right upper arm (lower curve) and phonocardiogram of a 67-year-old woman with tricuspid regurgitation.

and contrast it with the peripheral rheogram (rg) which is obtained by placing two electrodes on an extremity. The thoracic rheocardiogram (Fig. 4) normally shows a slight hump during atrial systole. During the isometric phase, the beginning of which coincides with the Q wave of the electrocardiogram, at the time of displacement of the valvular plane of the heart, there occurs a drop in the curve corresponding to sucking in of blood into the heart from the great veins. At the beginning of the ejection period, that is with the opening of the semilunar valves, a steep ascent appears, which breaks off sharply from the rest. The steep ascent corresponds to the rapid filling of the arteries near the heart. In the further course of this phase of systole there occurs a preponderance of outflow of blood into the periphery over the further expulsion of blood from the heart. Accordingly the curve sinks after the first third of the ejection period somewhat more sluggishly. The second heart sound indicates the end of systole. With the valves all closed, the congestion of blood awaiting entry into the ventricles produces rheocardiographically a new ascent of the curve. The continuing diastolic ascent of the curve falls in the rapid filling phase of the ventricles and is explicable by increasing filling of the venæ cavæ in this period. This phenomenon may be attributed to increased pressure on vein walls as a result of increased circulation rate. The results of the electrokymographic investigations of Salans *et al.* (1950) are in agreement with the results of our investigations.

In conformity with their arrangement we call the first wave the arterial or systolic wave, the second rheographic wave the venous or diastolic wave. In the normal rheocardiogram the height of the zenith of the venous wave coincides roughly with that of the arterial wave.

The thoracic rheocardiogram, besides affording an exact measurement of the phases of the cardiac cycle on the basis of characteristic changes in the form of the curve, also gives an appraisal of disturbed hæmodynamics when there is regurgitation.

Holzer *et al.* (1945) emphasized that they had obtained an abnormally high venous wave in all cases of tricuspid regurgitation. Later a secondary venous wave was determined also in the purely peripheral rheogram record. Further investigations demonstrate that this diastolic secondary wave is detectable not only in tricuspid regurgitation, but also in extreme venous congestion in right heart failure, without tricuspid regurgitation: Grabner *et al.* (1955) also emphasize this.

The synchronous registration of the jugular pulse curve with the rheogram, and in particular the peripheral rheogram (Fig. 9), seems to endorse the view of Bloomfield *et al.* (1946) and Lagerlöf and Werkö (1948), who assumed that ventricular filling in tricuspid regurgitation to be predominantly protodiastolic, whereas they recorded extensive repression of the venous inflow in later diastole. Accordingly in the late diastole, as a result of this repression of the venous return, there is successive congestion of the systemic veins: this is characterized in the peripheral rheogram by the appearance of the secondary venous wave. The abnormally high diastolic wave in the rheocardiogram would, on this assumption, be explained similarly, because it seems chiefly determined by the flow of venous return. The simultaneous registration of the thoracic rheocardiogram and the peripheral rheogram, as for example on the right upper arm (Fig. 5) yields a delay of the venous secondary wave in the periphery of approximately 100 msec. This delay in the secondary wave shows that this cannot be determined by the regurgitation of blood in systole, but must rather be interpreted as a venous congestion wave. If it were the result of regurgitation, it would occur synchronously with the "cv" wave of the jugular pulse. This, however, occurs always in the systole. Further, since by applying pressure greater than the venous pressure to a point proximal to the rheograph lead, it is possible to make the secondary wave disappear, the venous origin of the wave seems to be well established.

THE HEPATORHEOGRAM

Heeger and Polzer (1956) endeavoured to achieve a rheographic registration of liver pulsations. For this purpose two small electrodes (2×2.5 cm.) were attached at a distance of 6–8 cm. from each

FIG. 6.—Lead II and hepatorheogram of a normal subject.

FIG. 7.—Lead II and hepatorheogram of a 54-year-old woman with tricuspid regurgitation.

FIG. 8.—Lead II and normal rheogram of the jugular veins (31-year-old subject).

FIG. 9.—Lead II, rheogram of the jugular veins (upper curve), rheogram of the right upper arm (lower curve) and phonocardiogram of a 62-year-old woman with tricuspid regurgitation.

FIG. 10.—Lead II, normal heart proximal rheocardiogram and phonocardiogram of a 21-year-old man.

FIG. 11.—Lead II, heart proximal rheocardiogram and phonocardiogram of a 32-year-old woman with pure mitral stenosis.

other parallel to the costal arch over the liver. While the hepatorheogram (hrg) in the normal case (Fig. 6) shows merely a slight systolic arterial rise of the base line, and the curve in diastole runs nearly on the zero line, in the presence of tricuspid regurgitation (Fig. 7), besides the small arterial rise, a strikingly high diastolic wave is formed. We therefore assume in the presence of tricuspid regurgitation a diastolic increase in liver volume and deny systolic regurgitation of blood as far back as the liver. These diastolic filling fluctuations of the liver, which will be interpreted in the sense of pulsating congestion, are ultimately determined by the primarily protodiastolic ventricular filling found in tricuspid regurgitation. For, because of the small filling capacity of the right ventricle which is already filled with residual blood, the blood flow to the heart will be obstructed, which leads to a diastolic increase in volume of the congested liver. Proof of such a diastolic increase in liver volume with tricuspid regurgitation is at variance with the assumption made up to the present that the expansile pulsation of the liver is systolic in time. The liver pulse registered by a pulse recorder according to the method of Boucke-Brecht is quite comparable to the jugular pulse and is therefore interpreted as the expression of systolic blood regurgitation. Actually the liver pulse shows when compared with the hepatorheogram an exactly opposite course. On the other hand, the so-called liver pulse curve in tricuspid regurgitation is like a mirror image of the registrable chest wall pulsations over the left lateral thoracic wall. We have therefore attributed the so-called liver pulse to the clearly demonstrable rocking movements of the thorax in tricuspid incompetence. The liver pulse is thus according to our view determined by mechanical impulses imparted by the heart, and should not be regarded as a sign of regurgitation of blood into the liver, whereas the hepatorheogram reflects the volume fluctuations.

VARIX PULSATIONS

On the basis of these examinations of the liver we assumed that the familiar varix pulsation in this valvular deficiency were also diastolic in time. The phenomenon of pulsating varices is interpreted by numerous authors as systolic and attributed to regurgitation through the incompetent tricuspid valve, although Joachim (1910), through graphic registrations of such varix pulsations, had already determined that the varix pulsations occurred 0.04 sec. before the arterial pulse.

By registration of a transverse rheogram aimed at the level of a pulsating varix node it is possible to determine a venous wave, which comes into view just before the arterial rise. We have attributed this venous wave to the secondary wave of the peripheral rheogram. The slow progress of this congestive wave explains the fact that it is recorded just before the arterial displacement of the following systole. In the presence of distinct varices, the marked state of venous congestion accompanying tricuspid regurgitation will become, under certain conditions, even visible and palpable in diastole. The diastolic origin of such varix pulsations makes it understandable that, even without the presence of tricuspid regurgitation, in extreme right heart failure, varices may pulsate sometimes. This is illustrated by a case described by Hallock and Clarke (1941), in which distinct pulsations of varices above the knee were observed for years but at autopsy only mitral valve disease, with extreme right heart failure, but without tricuspid regurgitation, was found.

RHEOGRAM OF THE JUGULAR VEINS

The jugular veins are a further field of application for rheography. By placing the instrument upon the right and left jugular veins the measuring current flows through both these veins as well as the vena anonyma which connects them. If the lead-off points are chosen as far as possible from the carotid arteries, the rheogram of the jugular veins thus elicited shows a close agreement with the venous pulse registered in the conventional way. Corresponding to the normal jugular vein pulse, the rheogram of a 32-year-old subject (Fig. 8) shows a presystolic, a systolic, and a diastolic wave as well as a distinct systolic and diastolic collapse. In the presence of tricuspid regurgitation one finds in the rheogram of the jugular veins (Fig. 9), as well as in the jugular pulse, a broad systolic plateau corresponding to the insufficiency wave and a depressed diastolic curve drop. In those

cases especially, where for anatomical reasons the registration of the jugular pulse seems difficult, a rheographic display of volume fluctuations of the neck veins is indicated.

THE HEART-PROXIMAL RHEOCARDIOGRAM IN MITRAL STENOSIS

Recently one of us (Heeger, 1957) attempted to register the pathological fluctuations in heart volume in mitral stenosis by means of a heart-proximal rheocardiogram. For this purpose a small electrode (1.0×1.5 cm.) was attached over the cardiac apex and a second electrode of the same size at the insertion point of the fourth rib at the left sternal border. The rheographic curve thus obtained (Fig. 10) reflects the changing volume of blood in atria and ventricles and is similar to a curve of the electrokymogram of the individual heart compartments. It is characterized by a distinct decrease in systolic conductivity as a result of the systolic drop in ventricular volume. The ascent of the curve beginning in diastole is determined by the increase in conductivity in the atria when filled during ventricular systole. After opening of the atrio-ventricular valves there occurs a slow drop in the curve as a result of the decrease in atrial volume. By virtue of the increase of ventricular volume there occurs later in diastole an ascent of the curve. The contraction of the atria is expressed in a curve drop of changeable intensity, according to the drop in atrial volume.

In contradiction to this, in the presence of mitral stenosis the heart-proximal rheocardiogram is characterized by a change in the course of the diastolic curve (Fig. 11). After the systolic drop of the rheographic curve there occurs at the time of the second heart sound a steep ascent of the curve, which is interrupted, synchronously with the mitral opening snap, by a distinct break in the curve. Subsequently it shows a further conductivity increase in the course of diastole. We assume now that this diastolic ascent of the heart-proximal rheocardiogram curve is determined by particular hæmodynamic changes in mitral stenosis. The curve break at the time of the mitral opening snap could be caused by displacement of the stenotic mitral valve funnel toward the left ventricle, which would be precipitated by the pressure gradient between the left atrium and the left ventricle. The further diastolic ascent of the rheographic curve could be attributed to the obstruction to outflow by the stenotic mitral valve and the associated congestion in the pulmonary veins. According to the degree of stenosis of the mitral valve, the heart-proximal rheocardiogram shows certain changes in the curve break and the course of the diastolic curve.

SUMMARY

By registering the fluctuations of electrical conductivity, it is possible to record fluctuations in flow in the blood vessels of any chosen area by appropriate placing of the electrodes. Placing the electrodes on the thorax (rheocardiogram) reflects hæmodynamics on the basis of intrathoracic conductivity fluctuations. In particular, venous congestion in right heart failure yields characteristic curves which may help in differential diagnosis.

By placing the electrodes on an extremity (rheogram) the changes in form and amplitude of the inscribed curves indicate blood flow, give information about the elasticity of the vessel walls, and help in the supervision of vascular therapy.

Further, strict reproducibility, the possibility of standardization and calibration of the curves, as well as independence from circumscribed points for placing of the electrode are emphasized as advantages of this method.

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