The Origin of the Variations of Body Impedance Occurring during the Cardiac Cycle

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If the trunk is placed between two electrodes of a high frequency circuit, changes in impedance occur during the cardiac cycle. Experiments are presented which show that these variations do not result from the actual volume changes of the heart, as has been suggested in the literature. The changes in impedance are caused by the rhythmic variations in blood content of the vessels.

The well known fact that a change in volume or in shape of a body placed between two electrodes in a high frequency circuit influences the impedance and may therefore be recorded continuously was used by Atzler and Lehmann, Cremer, Rosa, Holzer and Polzer, Donzelot and Milovanovich in their attempts to study the volume changes of the heart.

In their first publication on this subject Nyboer and his associates also suggested that their "radiocardiogram" is a volume curve of the heart. In later papers, however, Nyboer apparently changed his point of view. The spontaneous rhythmic changes in body impedance are regarded as originating solely from the rhythmic fluctuations in caliber of the body vessels caused by the cardiac cycle. For this reason the method is called "impedance plethysmography." The prime importance of the cardiac volume changes has been criticized by Kedrov, Vetter and in a more recent paper by Polzer and Schuhfried. No systematic investigation has, however, been conducted to evaluate both influences which may contribute in the production of the rhythmic variations in body impedance during the cardiac cycle. This has been attempted in the experiments to be reported in the present paper.

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Method

If two electrodes of a high frequency circuit are applied to different parts of the body, fluctuations of the electric impedance during the cardiac cycle may be recorded by the apparatus to be described presently. In the production of these the two above-mentioned factors (changes in the volume of the beating heart itself and the rhythmic fluctuations in caliber of the blood vessels) may play a role. The discrimination between these has been attempted in two different ways. In one series of experiments either the heart or the lungs was electrically isolated by putting it in a rubber bag. In model experiments an envelope of this kind had proved to be quite an efficient insulator. The second set of experiments was started shortly after the death of the animal. By inserting cannulas into the aorta and the pulmonary artery (a) the heart's ventricles, (b) the lesser, and (c) the systemic circulation could be connected separately with a mechanical fluid pump. In this way the effect of volume changes of the heart and those resulting from rhythmic perfusion of the blood vessels could be studied separately.

Dogs were used in all experiments. After an initial dose of either morphine (2 mg. per kilogram), Chlorosan (50 mg. per kilogram) and Nembutal (20 mg. per kilogram) or morphine (2 mg. per kilogram), scopalamine (0.2 mg. per kilogram) and Nembutal (30 mg. per kilogram) prolonged anesthesia was obtained by occasional additional injections of Nembutal (10 mg. per kilogram). The animal was tracheotomized and connected with a Starling respiration pump. The spontaneous changes in electrical impedance occurring during the cardiac cycle were recorded during apnea of the animal in order to eliminate the fluctuations caused by breathing. The thorax was opened in the midline by elevating the sternum; the pericardium was incised and the heart exposed. In order to evaluate the effect of this procedure on the records, the lungs were inflated fully to cover the heart and the chest was temporarily...
closed again. Commonly no appreciable differences were noticed between the records taken from the intact dog and after the closure of the thorax. The next step consisted in the electrical isolation of the heart by enclosing it in a rubber bag, which was followed by the wrapping of both lungs in rubber sheeting. At each step a record was made. These were taken after inflation of the lungs, closure of the thorax and during apnea of the animal.

After an intravenous injection of 1.5 cc. of heparin the animal was bled to death from the carotid artery. Immediately thereafter the chest was opened again and both the aorta and the pulmonary artery were transected above the semilunar valves, and brass cannulas were pushed into the ventricles and ligated. The cannulas were both connected with a valveless motor-driven pump, and, by adjusting a screwclamp, the amounts of fluid (gum acacia-saline solution) passing to the ventricles could be equalized. A strong ligature had been placed around the base of the heart across both atria in order to prevent any backflow of fluid. Records were then made in which the pressure in the system was registered by a Statham gage for comparing the impedance changes with the pump cycle.

The chest was now again opened and glass cannulas inserted into the peripheral ends of both the aorta and the pulmonary artery. As the ligature around the atria had been removed and the pump had been provided with a set of valves and connected with a container filled with the gum acacia-saline mixture, the fluid could be driven either through the aorta into the systemic circulation and could leave the body by the right heart and the cannula in the right ventricle or through the pulmonary artery into the lesser circulation and out again by the left heart and the cannula in the left ventricle. On these records, too, both the impedance and the pressure changes resulting from the rhythmic perfusion were registered.

The electrodes (1.5 by 2.5 cm.) were applied to the right foreleg and the left hindleg ("standard derivation") or on each side of the thorax in the axis of the two main branches of the pulmonary artery ("side derivation").

The impedance between a pair of electrodes was measured by a modified Wheatstone bridge which was fed by a generator (14,000 cycles per second). The tissue impedance and a series resistor of 0.5 ohm were placed in one branch and were compared with a fixed impedance in the other branch. Since a change in body impedance affects the balance of the bridge, the bridge voltage may be detected and suitably amplified in one channel of an electrocardiograph. The amplification factor was always kept at the same value. In all records a decrease in impedance corresponds to a rise of the curve. The series resistor of 0.5 ohm could be short-circuited for calibration purposes.

In the experiments on the living animal the electrocardiogram was obtained by the use of a second independent channel of the electrocardiograph after filtering out the high frequency modulation. With the "standard derivation" for the impedance measurement an electrocardiogram was taken from the right and the left foreleg; with the "side derivation" the left side of the chest and the right foreleg were used. In the experiments on the dead animal the second channel recorded the pressure responses of the Statham gage.

Results

In the experiments on the living animal the attempt was made to withdraw either the heart or the pulmonary vascular bed from the flow of electric current between the electrodes by wrapping the heart or the lungs in rubber sheeting. The efficiency of this method had been tested by placing a piece of rubber sheet between two flat electrodes; no current was found to pass. The spontaneous fluctuations in body impedance related to cardiac activity prove not to be influenced at all by wrapping the heart in a rubber bag, at least when the "standard derivation" (right foreleg-left hind-leg) is used. The effectiveness of the insulating envelope is obvious from the reduction in size of the ventricular complex of the electrocardiogram. The absence of any notable effect on the impedance record suggests that the volume changes of the vascular bed and not those of the heart are mainly responsible for the spontaneous impedance fluctuations registered from the "standard" leads.

The position of the leads however proves to be very important. If the same experiment is repeated with an electrode placed on each side of the thorax, the curves shown in figures 1 A and B are obtained.

Since, after the isolation of the heart, the excursions become appreciably greater, it may be concluded that at least under this condition both the volume changes of the heart and the blood content of the vessels play a role. Even under these circumstances, however, when the heart lies between the electrodes, the vascular component prevails. This is clearly shown by comparison of figures 1 A and C. In the later experiment not the heart but both lungs were wrapped in sheet rubber. The exclusion of the pulmonary vascular bed greatly reduces the
exursions of the impedance curve, so that only small atypical fluctuations are left.

In the experiments on the dead animal the effect of artificially induced volume changes of the heart was compared with that of rhythmic perfusion of either the systemic or the lesser circulation. When a valveless mechanical pump is connected with both ventricles and the resistance of the tubes to them is so adjusted that the ventricles receive a nearly equal amount of blood, after closure of the thorax a record is obtained in which the upper curve gives the changes in impedance between the right foreleg and the left hindleg, the lower one those in pressure (figure 2 A).

Since in this experiment the stroke volume was maintained at 60 cc. it is plain that pure volume changes of the heart play a very minor role indeed. The experiment was repeated after placing the electrodes at the sides of the chest (fig. 2 B). In this situation in which the heart effectively intercepts the flow of the electric current between the electrodes, the influence of the changes in heart volume is well marked.

The perfusion of the systemic circulation and the exclusion of the heart and the lesser circulation was accomplished by connecting the aorta with a mechanical pump provided with valves and by letting the fluid leave the body by the cannula in the right ventricle.

The resulting impedance and pressure records are reproduced in figure 3 A. Changing the electrodes from the “standard position” to

![Figure 1](image1)

**Fig. 1.** A. Thoracotomized dog: (a) impedance record (both sides of the chest); (b) electrocardiogram (left side of chest-right foreleg).
B. Same dog with heart enveloped in rubber bag.
C. Same dog with lungs wrapped up in rubber sheeting.

![Figure 2A](image2)

**Fig. 2.** Artificially induced volume changes of the heart in the dead animal. Stroke volume 60 cc.: (a) impedance record (right foreleg-left hindleg); (b) pressure in ventricles.
B. Same dog: (a) impedance record (both sides of the chest); (b) pressure in ventricles.

the sides of the chest greatly lessens the extent of the excursions, as is shown in figure 3 B. This effect strongly indicates that the caliber
changes of the systemic vessels are mainly responsible for the impedance fluctuations shown in the former picture. As the extent of the systemic vascular bed within the chest is relatively small, the "side leads" are only able to pick up small impedance variations (fig. 3B).

This being the case, the opposite effect might be expected in those experiments in which only the lesser circulation was perfused. By connecting the outflow tube of the pump with the pulmonary artery and letting the fluid out by a tube in the left ventricle records like those of figures 4A and B have been obtained. At the "standard position" of the leads the electric current flows between the right foreleg and the left hindleg and so has to pass the thoracic organs. These, however, only represent a fraction of the total impedance between the electrodes and so the fluctuations caused by the rhythmic perfusion of the lesser circulation are small though well marked (fig. 4A).

A far greater effect results if under identical conditions the electrodes are applied to the sides of the chest (fig. 4B). In this case the rhythmically perfused lungs constitute the major part of the impedance between the leads and the extent of the excursions in figure 4B clearly shows the great influence of plethysmographic changes.

**DISCUSSION**

The results indicate that the fluid content of the vascular bed between the electrodes is the principle factor in determining the extent of the impedance. The simultaneously registered electrocardiogram and pressure record show that increase in blood content of the tissues goes with increase of current flow and
therefore with decrease of tissue impedance. According to the data on specific tissue impedance of Holzer and of Rajewski as reported by Holzer and Polzer and the slightly different values of Burger and van Milaan, the conclusion seems to be warranted that of all tissues the blood offers the least resistance to the electric current. The replacement of blood by gum acacia-saline solution (0.6 per cent sodium chloride and 6 per cent gum arabic) will have increased the impedance fluctuations recorded in the postmortem experiments by the factor 2, as the specific impedance of the solution amounts to about half that of blood.

Assuming that the blood content of the tissue between the electrodes determines the extent of the recorded impedance fluctuations, we may now attempt an interpretation of the results of these experiments in which the heart beat was imitated by rhythmic pumping of the ventricles. Examples are given in figures 2 A and B. In the former record “standard leads” were used and so the impedance record fluctuates but little as the change of cardiac volume contributes only a fraction to the total impedance of the tissue between the right foreleg and the left hindleg. The much larger excursions of figure 2 B are readily explained by the position of the electrodes. With the leads fastened to the sides of the thorax the heart constitutes an appreciable fraction of the tissue through which the current has to pass, especially in the comparatively narrow-chested dog. For this reason the cardiac volume changes must show up better in the impedance record. In these experiments a rather remarkable phase shift manifests itself as the rise in impedance appears to be retarded with regard to the increase in pressure. In our opinion this phenomenon may be explained by the fact that some head of pressure has to be built up for the dilatation of the ventricular wall and that the inflow of fluid can only occur thereafter. For this reason the start of the ventricular filling is probably more accurately marked by the notch present on the ascending limb of most pressure records instead of by their footpoints.

In the spontaneously beating heart we may also expect that the increase in volume lessens the impedance and so increases the flow of the electric current through the heart. The reverse must occur during systole. As the contraction of the heart tends to increase the impedance but simultaneously decreases it by the expulsion of blood into the vessels of the lesser and systemic circulations, the changes in total impedance must amount to the difference between the cardiac and the vascular factor. This is borne out by comparing figures 1 A and B. The exclusion of the cardiac factor by wrapping the heart in a rubber bag proves to increase the excursions of the impedance record. As in this instance “side leads” have been used, the volume changes of the heart are mainly compared with the effect of the pulsations of only the pulmonary vascular bed. Even under these favorable conditions the direct influence of the beating heart appears to be very small.

This may sound rather surprising considering that the amount of blood thrown into the lesser circulation cannot be more than about half of the total volume change of the heart during systole. In part the phenomenon may be explained by the physical fact that the decrease in impedance resulting from the inflow of a given amount of fluid depends largely upon the distribution. If this fluid is equally distributed over the electric field between the electrodes, maximum variation will result whereas relatively little change will be seen when the fluid remains accumulated in a small sphere. On the other hand little is known about the spread of the current within the thorax. While it might be argued that with two relatively small electrodes at the sides of the chest the fluctuations in blood content of the whole pulmonary vascular bed must also have been inadequately reproduced, it might have been that in the actual position of the electrodes the cardiac factor was particularly poorly represented in the record.

In order to test this supposition the electrodes were applied to a great number of opposite spots on the chest wall. In the dog no significantly different results were obtained when one or both of the side electrodes were shifted. On the other hand remarkable differences were seen when one electrode was placed on the sternum and the other on the vertebral column.
The interpretation of the resulting curves however was found to be very difficult. It was expected that the shape of the thorax, the situation of the heart near the anterior wall and the absence of a cardiac lobe would make the human subject more suited for studying the impedance changes of the heart. However in most of these cases rather complicated curves were obtained so that a discussion of these had better be postponed.

SUMMARY

The impedance of the body has been described as fluctuating with the beat of the heart. By electrical isolation of the heart or the lungs of the living dog and by artificially induced volume changes of the heart and rhythmic perfusion of the systemic and of the lesser circulation in the dead animal these fluctuations were found principally to result from the expulsion of the blood from the heart during the ventricular contraction. The volume changes of the heart itself generally play a very minor role.

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