Physiology. — "*On the ventricle-electrogram of the Frogs' heart.*" By Dr. S. DE BOER. (Communicated by Prof. G. VAN RIJNBERK.[1])

(Communicated in the meeting of October 27, 1917).

It has appeared from my former investigations that we can admit the width of the R-oscillation of the ventricle-electrogram as a measure for the velocity, with which the impulse is transmitted through the ventricle.[2]) A decrease of the velocity of impulse-transmission is expressed by a widening of the R-oscillation, whilst a narrower R-oscillation betrays an increase of the velocity of impulse-transmission through the ventricle.

If now we want to trace the influence that the velocity of impulse-transmission has on the shape of the ventricle-electrogram, then we can consequently conclude from the width of the R-oscillation, whether the impulse is transmitted with more or less velocity through the ventricle. Now we can make the velocity of impulse-transmission decrease by exciting an extra-systole of the ventricle at an anticipated moment of the heart-period. If we wish however to compare the electrogram of such an anticipated extra-systole of the ventricle with those of the normal periodical systoles of the ventricle, then the requirement must be satisfied, that this anticipated ventricle-systole is brought about by an impulse, that reaches the ventricle along the atrio-ventricular systems of connection. The place where the impulse hits the ventricle at such an anticipated ventricle-systole must be the same as at the normal periodical ventricle-systoles. Only then we can make a comparison. Otherwise the modification of the shape of the ventricle-electrogram might be attributed to the fact, that the impulse proceeded from another place of the ventricle (e.g. at the surface of the ventricle as at extra-stimulation of this part of the heart). We apply consequently an extrastimulus to the auricle at an anticipated moment of the heart-period. After the extrasystole

[1]) These investigations were likewise communicated in the meeting of the Biological section of the Genootschap ter bevordering van Natuur- Genees- en Heelkunde (Physiologendag) held on the 20th of December 1917.

[2]) Zeitschrift für Biologie Bd. 65 Seite 128 and Journal of Physiology. Vol 49 page 310.

of the auricle, obtained in this way, the impulse continues along the atrio-ventricular systems, and causes an anticipated ventricle-systole. We can compare the electrogram of this systole with those of the normal periodical ventricle-systoles. It had appeared to me already during my investigations into this subject in 1914[1]), that the electrograms of those anticipated ventricle-systoles showed R-oscillations, of which the width, compared with those of the normal periodical ventricle-systoles, had increased. At the same time I stated that the T-oscillations of these anticipated ventricle-systoles had changed in a negative sense.[2])

We may expect, that after the compensatory pause during the post-compensatory systole the velocity of impulse-transmission had increased, and a decrease of the width of the R-oscillation of the electrograms belonging to it proved indeed, that this was the case. The T-oscillation of these electrograms has changed in a positive sense (a negative T-oscillation of the periodical ventricle-systoles had decreased, a positive T-oscillation had increased).

Modifications of the velocity with which the impulse was transmitted through the ventricle were consequently expressed by the width of the R-oscillation and by the dimension and the direction of the T-oscillation. During the last year I continued these investigations and systematically observed the changes that occurred in the ventricle-electrogram, when I modified the velocity of impulse-transmission. This continued investigation consists of 3 parts.

1. In the first place I caused the velocity of impulse-transmission to decrease by poisoning with digitalis or antiarine. Before the poisoning first a photogram was made, and then, whilst the poisoning continued, constantly with definite pauses a photogram was made, till the halving of the ventricle-rhythm set in. As the velocity of impulse-transmission suddenly increased again after the halving of the ventricle-rhythm, directly an other photogram was made. In this way I could compare the electrograms of the frogs' hearts before the poisoning with those that were made after the poisoning, and even before disturbances of rhythm set in,

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[1]) Zeitschr. für Biologie, Bd. 65, Seite 428, 1915.

[2]) By the change of the T-oscillation in a negative sense is meant, that a positive T-oscillation of the electrograms of the periodical ventricle-systoles decreases during an anticipated ventricle-systole or changes into a negative T-oscillation. If however the T-oscillation of the periodical ventricle-systoles is already negative, then an increase of it during an anticipated ventricle-systole means also a change in a negative sense.

The ventricle-electrograms produced after the halving of the ventricle-rhythm were compared in the first place with the electrograms taken immediately before the halving, and at the same time with all the previously registered ventricle-electrograms.

2. A second series of experiments was made with frogs' hearts in which, after the application of the mentioned poisons, halving of the ventricle-rhythm had set in already. This halved ventricle-rhythm was thereupon converted into the normal twice as quick one by an induction-stimulus, as was mentioned in my previous essays[1]). The normal ventricle-rhythm was converted again into the halved one. In this way I obtained in one photogram the ventricle-electrograms of the normal ventricle-rhythm and those of the halved one. During the normal ventricle-rhythm the impulse is transmitted much slower through the ventricle than during the halved one, because the number ot ventricle pulsations in the first rhythm is twice as great as in the second. Occasionally a spontaneous modification of rhythm of the not-poisoned heart was registered.

3. In a third series of experiments anticipated ventricle-systoles were excited in the not-poisoned frog's heart by applying extra-stimula to the auricle. I caused then in the beginning of the irritable ventricle-period and at a later period anticipated ventricle-systoles. As I explained already before, the electrograms of these anticipated ventricle-systoles could be compared with those of the periodical ventricle-systoles. The electograms of the anticipated ventricle-systoles were also mutually compared. During the ventricle-systoles that were excited in the beginning of the irritable ventricle-period the impulse was transmitted slower through the ventricle than during the ventricle-systoles that were excited at a later period of the irritable ventricle-period.

These 3 series of experiments produced me a rich material for the study of the influence of the velocity of impulse-transmission on the shape of the ventricle-electrogram. I shall first discuss these 3 series of experiments successively, guided by some photograms, and afterwards communicate my conclusions in a theoretical explanation, and add to these conclusions a few consideration concerning the signification of the views obtained for the electrophysiology of the heart.

I. *Comparison of the ventricle-electrograms of frogs' hearts before and after the poisoning with digitalis.*

The experiments were made in the following manner. The frog

[1]) Archives Néerlandaises de Physiologie de l'homme et des animaux Tome I, p. 271 et 502.

was extended on a cork-plate, and thereupon the heart was laid bare in the usual way and suspended at the point. The oscillations of the lever were photographed on the sensitive plate beside the electrograms that were obtained after placing one unpolisarable electrode on the heart point and one on the auricle. The time was likewise indicated in all photograms in $^1/_5$ second. The experiments of the second and the third series were arranged in the same way, but for these a stimulator was moreover placed against one of the parts of the heart, and the moment at which the stimulus was applied, was indicated on the sensitive plate by a signal that was linked in the primary current-circuit of the induction-apparatus [1]).

In this series of photograms as well as in the two following ones we shall trace in the first place the width (duration) of the R-oscillation, then the extent and the direction of the T-oscillation. We call a T positive, when its direction is equal to that of the R-oscillation, and negative when it is opposite to the latter. Thereupon we consider the line of connection between the R- and the T-oscillation. When this line of connection is removed in the direction of the R-oscillation, it rises, it lowers, when it is removed in a direction opposite to that of the R-oscillation. In this communication I shall consequently explain more accurately of the photograms represented only these 3 parts of the ventricle-electrograms.

In order to avoid occupying too much room for the figures, I shall restrict myself to reproduce five photograms, one taken before and four after the poisoning with digitalis dialysate (GOLAZ).

In Fig. 1 the suspension-curves of a frog's heart are reproduced before the poisoning and likewise the electrograms (deduction auricle-point). The T-oscillation is positive, the line of connection between the R- and the T-oscillation is above the line indicating the position of rest of the string. Then I inject under the skin of the thigh 12 drops of digitalis dialysatum. Another photogram is taken under equal conditions fifteen minutes after the injection (Fig. 2). If we compare the width of the R-oscillation of this photogram with that in Fig. 1, we see that it has considerably increased. This teaches us that the velocity of impulse-transmission through the ventricle has decreased. The T-oscillation is still positive, but has become very little and the line of connection between the R-oscillation and the T-oscillation coincides now almost with the position of rest of the string.

[1]) When the primary current-circuit was closed the signal moved downward, when opened upward. The closing induction shocks were blended off, the opening induction shocks directed towards the frog's heart.

Fig. 3 has been taken 15 minutes after Fig. 2. The width of the R-oscillation has still increased very much [1]). The T-oscillation has now become strongly negative and the line of connection between

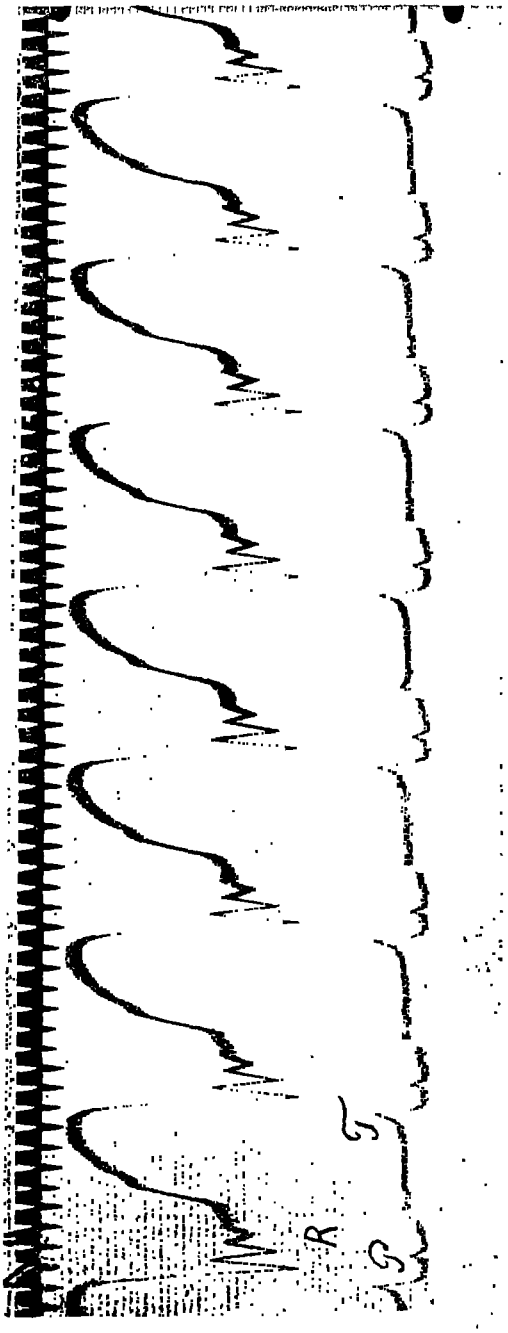

Fig. 1.

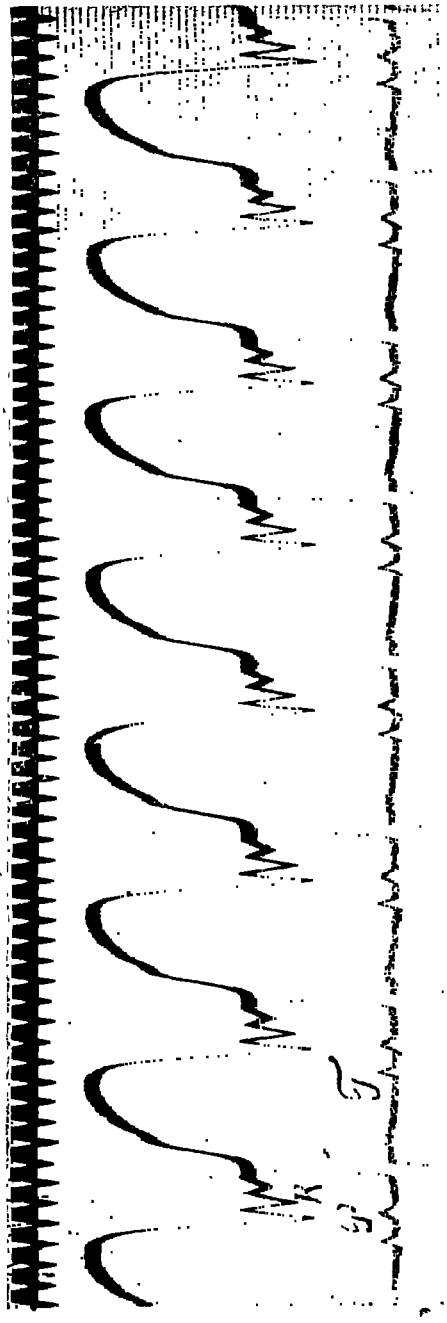

Fig. 2.

[1]) The time has not been reproduced in this photogram, but the velocity of the fall of the plate was the same as in the former photograms.

he R-oscillation and the T-oscillation lies now below the position of rest of the string.

Fig. 4 was again photographed 15 minutes after Fig. 3. The R-oscillation has now become exceedingly wide. The T-oscillation is

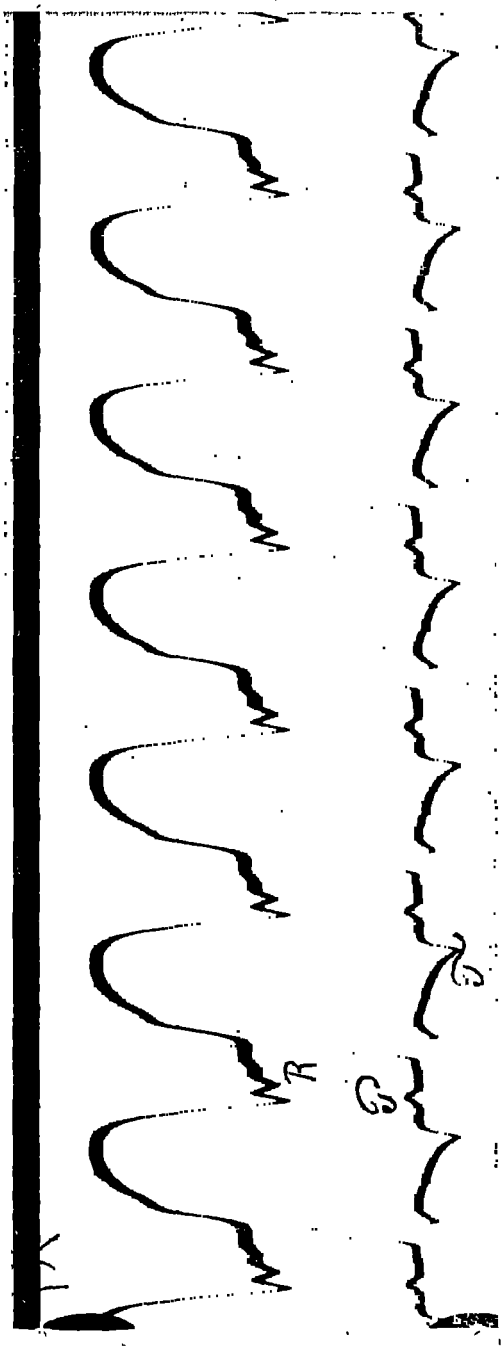

Fig. 3.

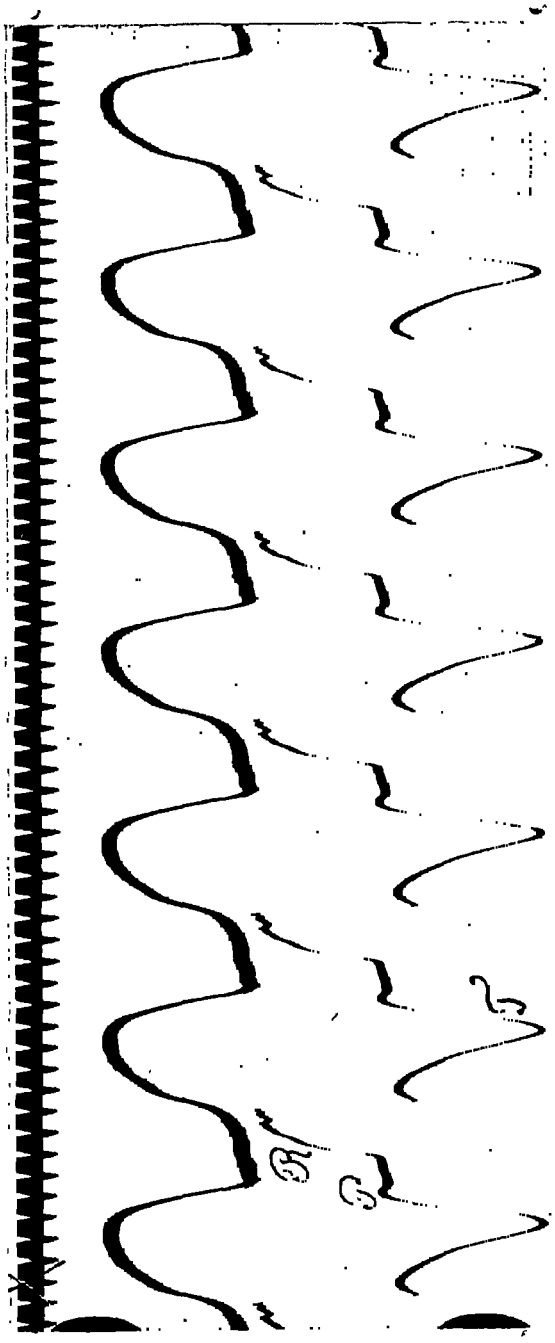

Fig. 4.

now very strongly negative, and the line of connection between the R-oscillation and the T-oscillation has descended more than in the former photogram.

Fig. 5 was taken 15 min. after Fig. 4. In the mean time the rhythm of the ventricle has halved, but after every large ventricle-systole still an abortive systole of the ventricle occurs. This abortive ventricle-systole gives a little nearly triangular electrogram (*a*). During the halved ventricle-rhythm the velocity of impulse-transmission through the ventricle has again considerably increased. In accordance with this fact the R-oscillation has become again much narrower. The T-oscillation is still negative, but has become considerably smaller than in the former photogram. The line of connection between the R-oscillation and the T-oscillation lies for a part somewhat above the position of rest of the string. If we compare Fig. 5 and Fig. 3, then in Fig. 5 the R-oscillation is narrower than in Fig. 3. In accordance with this fact the T of Fig. 5 is likewise smaller than that of Fig. 3, and the line of connection between the R-oscillation and the T-oscillation in Fig. 5 lies at a higher level than in Fig. 3. After the application of the two poisons mentioned above these results were constantly obtained by me. As long as the poisoning continues, and still before the halving of the ventricle-rhythm has set in, the velocity of the impulse-transmission through the ventricle decreases. The width of the R-oscillation increases accordingly, the T-oscillation changes in a negative sense and the line of connection between the R-oscillation and the T-oscillation descends[1]). As soon as halving of the ventricle-rhythm has set in the velocity of impulse-transmission increases again; the width of the R-oscillation decreases, the T-oscillation changes in a positive sense, and the line of connection between the R-oscillation and the T-oscillation rises[2]).

II. *Artificial and spontaneous modifications of rhythm.*

If we poison a frog's heart with veratrine, digitalis or antiarine

[1]) If after the poisoning ventricle alternation appears, then the proportions through the partial ventricle-systole during the little ventricle-systoles are of course different (vide report of the Physiologendag, 20 Dec. 1917). More extensively about this subject afterwards.

[2]) From Fig. 1 to Fig. 5 included the width of the P-oscillation increases through the poisoning, whilst the auricle-rhythm remains constant. The width of the R-oscillation has consequently decreased in Fig. 5 in consequence of the halving of the ventricle-rhythm, but the width of the P-oscillation has increased, as the rhythm of the auricle has remained unaltered.

the rhythm of the ventricle halves after some time, because the duration of the refractory stage of the ventricle increases. We can then convert the halved ventricle-rhythm into the normal twice as quick rhythm by applying an extra-stimulus to the ventricle at the

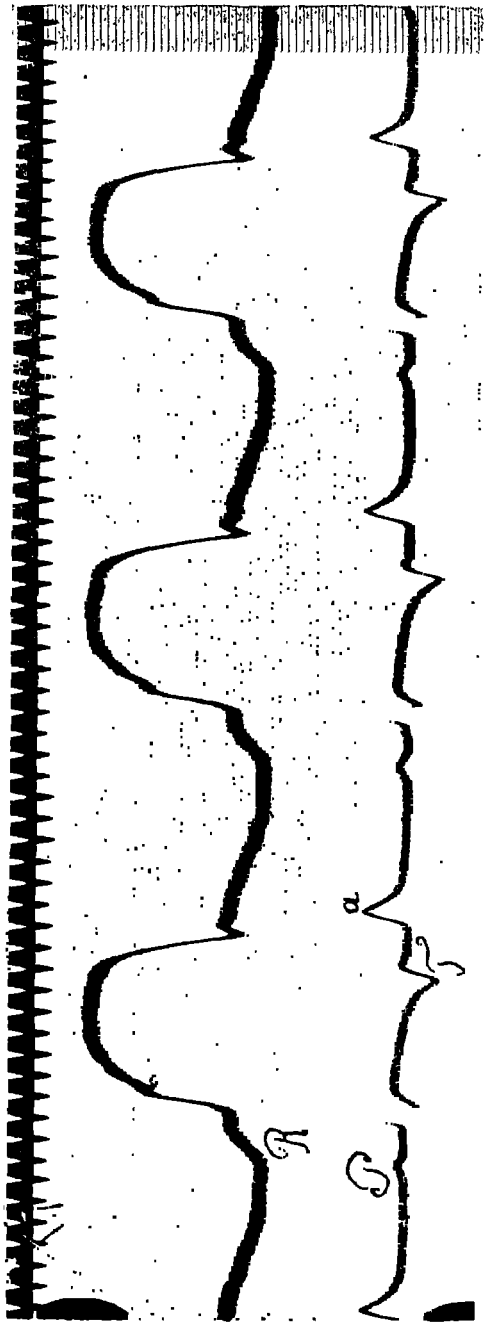

Fig. 5.

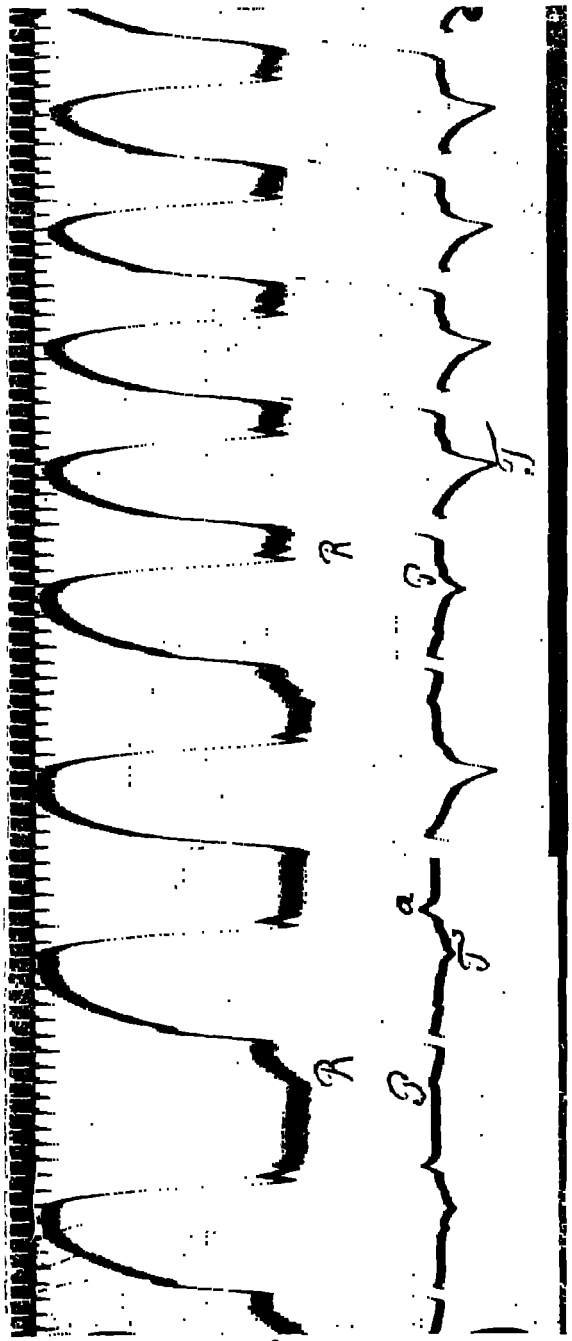

Fig. 6.

end of the diastole or the pause. This was already explicitly discussed by me with regard to veratrine and digitalis to which I refer here [1]). The same holds likewise for antiarine, about which I intend to publish a more extensive communication. We can then convert again the normal ventricle-rhythm into the halved one by applying an extra-stimulus to the auricle or to the ventricle-basis in the beginning of the ventricle-systole.

During the normal rhythm the impulse is transmitted slower through the ventricle than during the halved rhythm of the ventricle. It is of course clear that the conductivity inside the ventricle during the normal ventricle-rhythm, in which in the same time twice as many systoles of the ventricle take place than during the halved ventricle-rhythm, is worse than after the halving of the ventricle-rhythm.

In Fig. 6 I reproduce an example of such an artificial modification of the rhythm. In the beginning of the fig. (the first two ventricle-systoles) the rhythm of the ventricle is halved. After every large ventricle-systole occurs another extremely little abortive ventricle-systole, the little triangular electrograms of which are indicated by an *a*. Both these little ventricle-systoles of the halved rhythm show little negative T-oscillations, and the line of connection between the R- and the T-oscillation lies just below the position of rest of the string. At the rising of the signal the ventricle-basis receives an extra-stimulus towards the end of the pause, after which a great ventricle-systole follows. Thereupon the normal rhythm of the ventricle is restored. The first ventricle-systole of this normal ventricle-rhythm succeeds still after a rather long pause, so that the impulse-transmission through the ventricle is now only unimportantly retarded (compare the width of the R-oscillation of this systole with that of the two preceding systoles of the halved ventricle-rhythm). This slight retardation is however already expressed in an enlargement of the negative T-oscillation and in a descent of the line of connection between the R-oscillation and the T-oscillation. The pauses between the succeeding ventricle-systoles are considerably shortened, and now the width of the R-oscillations has remarkably increased. The ventricle-electrograms show likewise large negative T-oscillations, and the lines of connection between the R- and the T-oscillations have descended considerably, and are gradually converted into the T-oscillations.

In Fig. 7 the halved rhythm of the ventricle was by an

[1]) Arch. Néerl. de Physiol. loc. cit.

extra-stimulus on the basis ventriculi converted into the normal one, and the latter again into the halved ventricle-rhythm. The halved ventricle-rhythm was here also obtained by poisoning with antiarine.

The first ventricle-systole of the figure belongs still to the halved rhythm. A short time after the end of the diastole the ventricle-basis receives an extra-stimulus, through which the halved ventricle-rhythm is converted into the normal twice as quick rhythm.

When we compare now the ventricle-electrograms of these two rhythms, we are immediately struck by the fact that the R-oscillations during the normal ventricle-rhythm are wider than those during the halved rhythm.

In the halved ventricle-rhythm the T-oscillations are negative but very little, and the line of connection between the R and the T lies just above the position of rest of the string. In the normal ventricle-rhythm the T-oscillations are likewise negative but rather large, and the line of connection between the R and the T lies now below the position of rest of the string.

The basis ventriculi receives another stimulus at the 2nd rising of the signal, which gives rise to a little abortive systole. After the compensatory pause the ventricle is fixed again into the halved rhythm through the post-compensatory systole. The ventricle electrograms have likewise again obtained the same shape as in the beginning of the figure.

In Fig. 8 the ventricle pulsates in the beginning likewise in the halved rhythm (after poisoning with antiarine). At 1 the ventricle-basis receives an extra-stimulus, causing an extra-systole of the ventricle. After this the halved ventricle-rhythm continues however. When thereupon at 2 the extra-stimulus is repeated a little earlier in the ventricle-period, the conversion into the normal ventricle-rhythm succeeds, but after 3 systoles it changes again into the halved one. During the halved rhythm again little abortive ventricle-systoles occur, the triangular electrograms of which are indicated by an *a*. During the halved ventricle-rhythm the T-oscillation is positive, and the line of connection between the R and the T is above the position of rest of the string. At the quicker normal ventricle-rhythm the R-oscillations are considerably widened, the T is strongly negative and the line of connection between the R and the T has descended far below the position of rest of the string.

We find these proportions not only with poisoned hearts, but not poisoned frogs' hearts show the same phenomena. Fig. 1 of one of

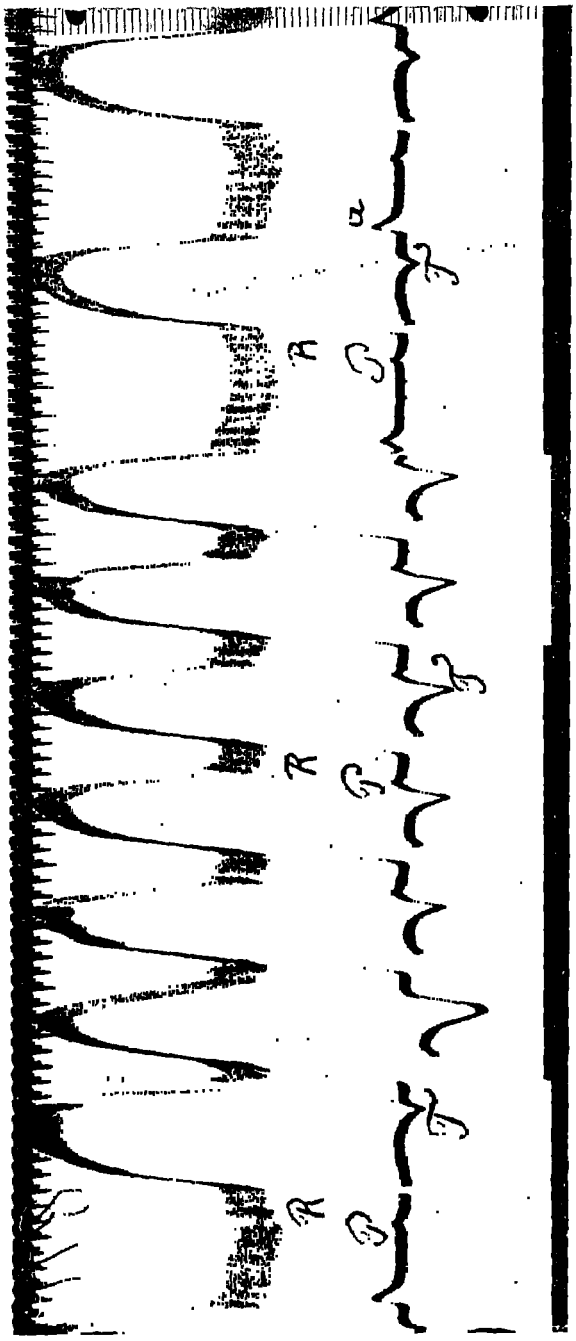

Fig. 7.

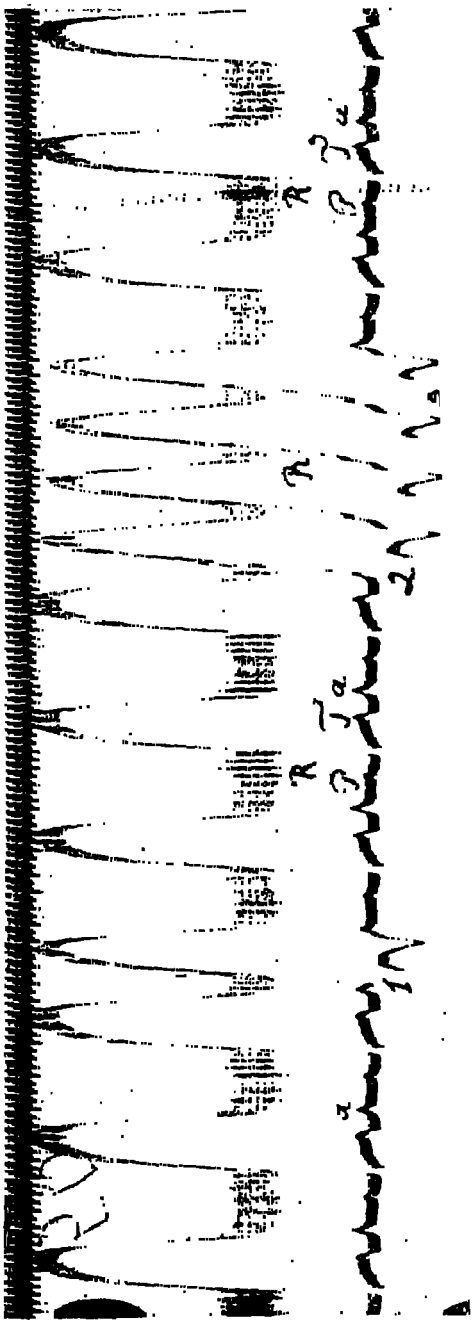

Fig. 8.

my former communications [1]) teaches us so. In this figure we see a spontaneous conversion of the normal ventricle-rhythm into the

[1]) Koninkl. Akademie van Wetenschappen at Amsterdam. Verslag van de gewone vergadering der Wis- en Natuurk. Afdeeling van 30 Juni 1917 Deel XXVI blz. 424 and Proceedings. Vol. XX, page 404.

halved one in a not-poisoned heart. In both rhythms the T-oscillations are positive but those of the halved rhythm are larger than those of the twice as quick normal one.

During the halved rhythm the width of the R-oscillation in this

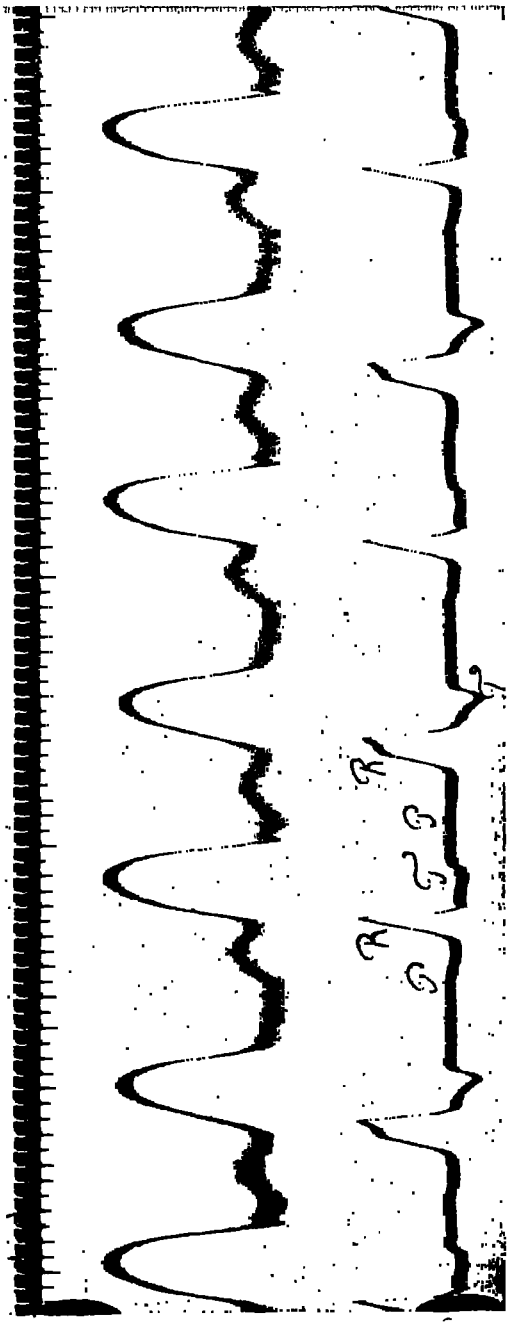

Fig. 9.

figure decreases from the first systole to the third included, and the height of the T-oscillation increases. A line of connection between the R and the T is not to be observed in this figure, because the T is immediately connected with the R.

Fig. 9 may still find a place here. This figure shows bigeminus-groups, after poisoning with veratrine, resulting from the falling away of every third auricle- and ventricle-systole. During the second ventricle-systole of each group the impulse-transmission through the ventricle is retarded more considerably than during the first. This appears from the wider R-oscillation of the second ventricle-systole. But the T-oscillation is much more negative, and the line of connection between the R and the T has descended much lower.

The bigeminusgroups, which I published in 1915,[1]) show likewise analogous proportions. The R-oscillation of the 2nd systole is here wider, and the positive T-oscillation smaller than that of the 1st systole of each group.

III. *The electrograms of the anticipated ventricle-systoles.*

With regard to this series of experiments a short communication will be sufficient. In a former communication [2]) these were already mentioned and explained with figures. The anticipated ventricle-systoles were excited by extra-stimulation of the auricle, which brought about extra-systoles of these parts of the heart. After such an extra-systole the impulse proceeded along the atrio-ventricular systems of connection towards the ventricle, which consequently was brought to contraction at an earlier moment of the ventricle-period.

The place where the impulse enters into the ventricle at these anticipated ventricle-systoles, was consequently the same as for the normal periodical ventricle-systoles. For this reason there was no objection to compare the electrograms of these anticipated ventricle-systoles with those of the periodical ventricle-systoles. It is obvious, that the velocity of impulse-transmission through the ventricle during the anticipated ventricle-systoles was inferior to that of the periodical ventricle-systoles and the retardation was the more considerable according to a ventricle-systole being more anticipated. In accordance herewith the R-oscillation of the ventricle-electrograms of the anticipated ventricle-systoles was wider than that of the periodical

[1]) S. de Boer: Die Folgen der Extrareizung für das Elektrogramm des Froschherzens. Zeitschrift für Biologie, Bd 65, 1915, Seite 440, Fig. 8.

[2]) Koninklijke Akademie van Wetenschappen, Verslag van de gewone Vergadering der Wis- en Natuurk. afdeeling van 30 Juni 1917, Deel XXVI bldz. 422, and Proceedings Vol. XX page 404.

ventricle-systoles, and the wider in proportion as the ventricle-systole was more inticipated. The T-oscillation of an anticipated ventricle-systole changed in a negative sense, and the more so in proportion as the ventricle-systole was more anticipated. The line of connection between the R and the T had descended at an anticipated ventricle-systole, this descent was the more considerable in proportion as the ventricle-systole was more anticipated. At the post-compensatory systole these proportions were exactly the reverse. Then the velocity of impulse-transmission had improved, the R-oscillation was narrower, the T-oscillation had changed in a positive sense, and the line of connection between the R and the T had risen.

These short indications may be sufficient for the present. For further particulars one must compare the figures 6, 7, 8, 9 and 10 of my communication.[1])

IV. *Theoretical explanation.*

It has appeared most clearly from the three series of experiments described above, that there is a constantly occurring connection between the width (duration) of the R-oscillation (velocity of impulse-transmission through the ventricle) on one side and the dimension and direction of the T-oscillation and the level, on which the line of connection between the R and the T extends itself, on the other side. When the duration of the R-oscillation increases, then the T-oscillation changes in a negative sense, and the line of connection between the R and the T descends. If on the contrary the duration of the R-oscillation decreases, then the T changes in a positive sense, and the line of connection between the R and the T rises. The modifications that the T-oscillation is subject to, had already distinctly displayed themselves to me by the investigations I made in 1914. I think I am now likewise able to explain more explicitly the modifications, that the line of connection between the R and the T undergoes, and to bring in this way the above mentioned experiments under one point of view.

The normal ventricle-electrogram consists chiefly of an R- and a T-oscillation. Consequently we do not discuss here the Q- and S-oscillation, because the occurrence of these is of no importance whatever for our considerations. These R- and T-oscillations are caused

[1]) I intend to explain in a more circumstantial communication more elaborately the electrograms obtained after extra stimulation of the ventricle-basis and point. We can for this purpose compare the electrograms of the more and less anticipated systoles with each other, and not with those of the periodical ventricle-systoles (Vide Fig. 6, 7 and 10 of the former communication.)

by interference of the basal with the apical negativity. The upward-oscillation by which the ventricle-electrogram begins, originates, because the negativity of the basis begins or domineers in the beginning. A short time afterward the apical negativity begins (or the apical negativity increases) and brings the string back to the position of rest. Then there is for some time equilibrium between the basal and the apical negativity, and the string remains in the position of rest.

Thereupon the T-oscillation comes into existence; if this T-oscillation is positive, consequently in the same direction as the R-oscillation, this is caused by the fact, that the basal negativity lasts longer than the apical negativity, or because in the end the basal negativity domineers over the apical negativity. If the T-oscillation is negative, consequently in a direction opposite to that of the R-oscillation, then the apical negativity lasts longer than the basal negativity or then, in the end, the apical negativity domineers over the basal one. In Fig. 10 I have represented the origin of the R and the positive T by interference of the basal negativity *a—b—c* with the apical negativity *e—f—g*. When now the velocity of impulse-transmission decreases, then the apical negativity will begin (or increase) later after the beginning of the basal negativity, and bring the string back to the position of rest. On account of the retardation of the transmission the position of rest is now reached at a later period.

The width of the R-oscillation increases thereby. But the other part of the ventricle-electrogram is likewise greatly influenced by the

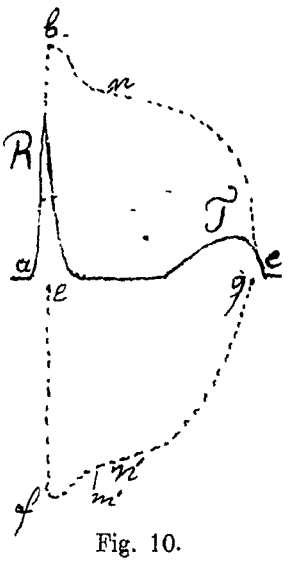

Fig. 10.

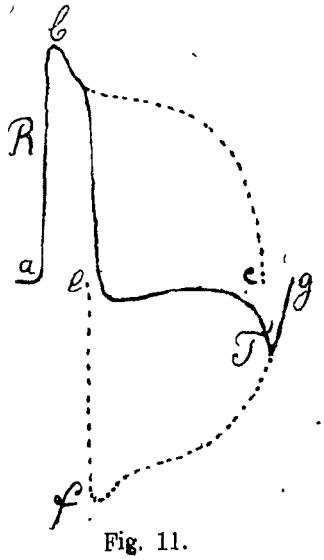

Fig. 11.

retardation of the transmission. The scheme of Fig. 11 may explain this fact. The basal and the apical negativity consist in this scheme of the same curves as those of Fig. 10, but the apical negativity has now removed more backward.

Point e is now much farther removed from a than in Fig. 10. What is now the consequence of this removal of the apical negativity? In the first place that at the end of the electrogram the apical negativity begins to domineer, and consequently the T becomes negative. If the retardation of the transmission had been less important, then this would only have reduced the positive T somewhat. But the line of connection between the R and the T has likewise descended. This is also to be understood. Whilst in Fig. 10 at a given moment the basal negativity n interferes with an equally strong apical negativity n' the string remains thereby in the position of rest.

When now, on account of the retardation of the transmission, the apical negativity is removed to the end of the electrogram, then the basal point n no longer interferes with n' but with m', which is removed farther from the position of rest. This holds now for all points of the basal negativity after retardation of transmission. These interfere consequently all with stronger apical negativities than before the retardation. This is the reason why the line of connection between the R and the T descends. This simple construction teaches us, why at retardation of transmission not only the R-oscillation widens, but also the T changes in a negative sense, and the line of connection between the R and the T descends.

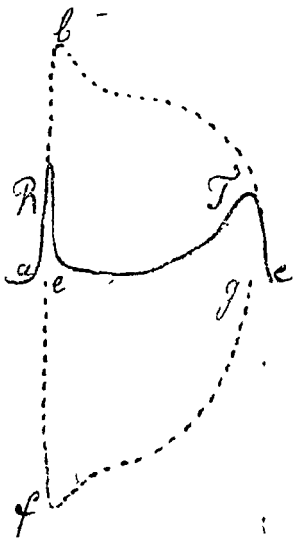

Fig. 12.

A clear illustration of the experimental data.

At an acceleration of impulse-transmission the R-oscillation will on the contrary become narrower and remove the apical negativity in a contrary direction i.e. to the front. Then each point of the curve of the basal negativity will interfere with a less important apical negativity than before the acceleration. The result is then a rising of the line of connection between the R and the T and an enlargement of the T as the scheme of Fig. 12 indicates. [1])

Still a few words about the height of

[1]) I have used in the scheme of Fig. 11 and 12 for the basal and the apical

the R. When the velocity of the impulse-transmission of the ventricle is so great, that apical negativity has already brought back the initial oscillation of the string to the position of rest, before the full basal negativity has developed itself, then the height of the R-oscillation possibly increases at a retardation of the transmission. These proportions are also reproduced in this way in the scheme Fig. 10.

If on the contrary the maximal basis-negativity has already been reached, before the apical negativity brings the string back to the position of rest, then a retardation of the conductivity will no more increase the height of R, but only widen its top. We find these proportions in the frog's heart after the hemorrhage. [1]) I hope to come back to this subject more elaborately in a more extensive communication.

It stands to reason that the shape of the ventricle-electrogram is *not only determined by the velocity of impulse-transmission.* In a former communication of mine I indicated the partial asystole of the ventricle as the cause of the modification of the shape. [2]) Its shape can likewise change besides by the more or less monophasical deduction (by killing the heart-tissue under one deducting electrode).

I shall restrict myself in this short communication to a few remarks concerning the consequences of the views developed above.

In the first place about the atypical ventricle-electrograms. In these the proportions are as in Fig. 11 viz. a high, wide R, descent of the line of connection between R and T and a negative T. In such an electrogram the apical negativity has consequently been removed backward [3]). This can be caused by retardation of velocity, but in casu the longer distance that the impulse has to cover, will most likely be the cause. In the light of these experiments the shape of the atypical electrograms is conspicuous to us.

components the same as in Fig. 10. It is obvious, that at modification of the velocity of impulse transmission these two components are likewise modified. As these modifications are for in an equal sense, the results are after all as reproduced in Fig. 11 and 12.

[1]) Zeitschrift für Biologie, Bd. 65, Seite 428.

[2]) Archives Néerlandaises de Physiologie de l'homme et des animaux, Tome I, p. 29, 1916 and Zentralblatt für Biologie, Bd. 30, Seite 149, 1915.

[3]) For the question under consideration it is of no importance, whether we have to do here either with the basal and apical negativity or with the negativity of the left- and the right-ventricle. When the two negativities coincide less, because one of the two commences later, then the atypical shape of the ventricle-electrogram sets in.

In the second place vigorous hearts have a large positive T-oscillation. This is likewise easy to understand, as vigorous hearts possess a good conductivity.

There exists moreover a strong overlapping of the basal and the apical-negativity, and consequently the basal negativity is at the end strongly expressed.

Finally the considerable variability of the T-oscillation is determined in this sense by the velocity of impulse-transmission as has been explained above.

These short remarks may suffice here. I intend to explain these and further results of these experiments in an ulterior more elaborate communication.

Amsterdam, Oct. 1917. *Physiological Laboratory.*