Electrocardiogram in Yellow Fever
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The variations of Electrocardiograms in Yellow Fever show a sufficient frequency to deserve a special study. They are more or less high, according to the severeness of the cases, and are generally sufficient to establish prognosis.

In cases which evolve toward cure, signs of degenerative processes of the cardiac muscle are rarely seen. Hardly some changes inherent to the influence of the vago-sympathetic system are observed, and never deformations of electrical complexes arise. In more severe incidences, chiefly in those which end in death, not only deformations and aberrations in the electric variations, but also progressive changes of determined waves are observed, the same corresponding to the degeneration process of the muscle. They can easily be accompanied by registration "en série" of Electrocardiograms.

Bradycardia, following the acute stage of the infection, chiefly convalescence, is always existing in unmortal cases. Sometimes highly emphasized, it is characterized in the Electrocardiogram, not only by the lengthened form of total diastole, as also by the increase of duration of all electric phenomenons and the interval which separates them. Observation N. 1 is a typical example of the above mentioned.

In some cases, scarce indeed, the influence of the vago-sympathetic system leads to auriculoventricular dissociation or to nodal rhythm. Such changes are always timely and disappear with the cure. Of this fact, observation 2 gives a striking example.

Rather frequent are deformations of auricular and ventricular complexes, consequent to degenerative processes of the myocardium, these changes leading to deficiency in the transmission of the contractile stimulus, by the different components of the heart. Sometimes such signs are obvious in clinical forms of middle or mild aspect, and there, they appear neither in the course of convalescence nor after the cure. Generally, however, they are found only in severe cases and appear at a certain stage of the infection. Their appearing can be deemed a bad prospect, as only in a single patient, that of observation N. 3, they did not mean to be a sure symptom of, if not impending, at least, a certain death. Observations 3 and 4 are examples of this affirmation.

Still as a meaning of a degenerative process of the cardiac muscle, we observed in cases of severe and fatal infection, a peculiar aspect of the electrocardiogram, which we always used to determine prognosis and probable evolution of the illness. This aspect is emphasized in the curves of observations N. 5, 6, 7, 8 and 9. As long as evolution is going on, even a long time before appearing any alarming clinical symptoms, one may
observe the increase in every direction and in all leads, of the T wave of ventricular complexes, with steady decrease in the extension of R wave and lengthening of the latter. The intersystolic interval also increases, so as to progressively deform the ventricular complexes which take really queer aspects in more advanced stages of the infection (Observations ns. 7, 8, and 9).

Not only ventricular variations are subject to changes; the auricular wave also is highly altered, however in a smaller number of patients.

In observation N. 7, beside a big deformation of the auricular complexes, and the increase of the intersystolic space (P−R), a few hours before the death, long crisis of extrasystolic heterotopic tachycardia of the ventricle could be observed.

In observations Ns. 8 and 9, the waves of the diagram were deformed in such a way, that differenciation could hardly be undertaken.

We must mention about observation N. 8, that, not only the ventricular complexes were deformed, as also auricular phenomenons had suffered serious alterations. The aspect of diagram, a few minutes before the death took place, would remind and even lead to think of the existence of a rhythm of heterotopic origin in the ventricle, but the thorough association of ventricular complexes brings a certainty of the homotopic origin of the ventricular excitation, and of its bad conductive power throughout the specific elements.

The graphic of observation N. 9 induces at first to believe in a rhythm of heterotopic excitation, but comparing it with diagrams obtained in the former observation, we should rather think it means only a deep deformation of homotopic ventricular complexes.

It is also of interest to mention a few observations gathered by the time of death. These verifications give an exact idea of the way in which the cardiac function ceases in this illness. Observations Ns. 8, 9, 10 and 11 are very curious examples, which deserve examination.

In the whole number of patients observed till death, the above mentioned deformation of ventricular complexes took place. At the final moment, all of them showed a period of ventricular extrasystoles, the said period having generally a variable duration and being followed by fibrillation of the heart and immediate stopping of the contractions.

The electrocardiogram in Yellow Fever has neither a characteristic nor a peculiar part to play with regard to the infection. It however registers in the most accurate manner, all stages of degeneration, which the cardiac muscle undergoes, and these alterations ought to deserve a more complete anatomical study. It means also an indication of some weight for prognosis, and a possibility of better judging the severeness of the various cases submitted to observation.

CLINICAL RECORDS.


1st. Diagram. Acute stage of the infection (fig. 1) normal rhythm slightly accelerated.
2nd. Diagram. Convalescent from the illness (fig. 2). Normal Rhythm very slow.

\[
\begin{align*}
P - R &= 0.12 \\
P &= 0.06 \\
R - T &= 0.32 \\
R &= 0.04 \\
S &= 0.02 \\
T &= 0.12
\end{align*}
\]


1st. Diagram. Acute stage of the infection (fig. 3). Very deep respiratory arhythmia, with periods of dissociation and other of nodal rhythm.

This irregularity was registered, at times, in the way of P—R dissociation, otherwise by the appearing of a few nodal excitations which are plainly shown in fig. 3. Thus, the interval P—R of the 3 first cycles is 0.20 and the wave R measures, in these same cycles, 1 mvt. and 7/10 mvt. After a longer diastolic stage, the third cycle of the diagram is 0.04 and the wave R with 1 mvt. and 9/10 mvt. The 4th and 5th cycles of the diagram are similar to the three first ones, the 6th showing a new nodal excitation with interval P—R equal to 0.12. P with 2/10 mvt. and R with 1 mvt. and 7/10 mvt.

In this incidence, stages of plain auriculoventricular dissociation with ventricular bradyrhythmia, could be seen, down to 32 excitations per minute, of which however we were unable to get a sufficiently plain graphic to allow being printed.

2nd. Diagram. Convalescent from the illness. Normal rhythm. The intersystolic interval remains permanent at 0.20 (fig. 4).


1st. Diagram. Acute stage of the illness. Normal rhythm. Slight febrile tachycardia:

\[
\begin{align*}
P - R &= 0.08 \\
P &= 0.04 \\
R - T &= 0.30 \\
R &= 0.02 \\
S &= 0.01 \\
T &= 0.12
\end{align*}
\]
2nd. Diagram. After the acute stage of the infection. Frequent ventricular complexes of the nodal type are observed, but of quite abnormal conductive power. Besides this, the sinusal complexes even had suffered a modification. Thus:

\[
\begin{align*}
P - R &= 0.12 \\
P &= 0.06 \\
R &= 0.02 \\
R - T &= 0.32 \\
T &= 0.12 \\
S &= 0
\end{align*}
\]

The deformed complexes of this period measured:

\[
\begin{align*}
R &= 0.08 \\
R - T &= 0.40 \\
T &= 0.16 \\
P - R &= 0.02
\end{align*}
\]

The extension of wave R in the diagram of fig. 5 is 1 mvt. and 1/10 mvt. and in fig. 6, 8/10 mvt. The waves R. of the aberrant complexes had but 5/10 mvt.


1st. Diagram on the fifth day of the illness. (Fig. 7). Regular Rhythm.

<table>
<thead>
<tr>
<th>DI</th>
<th>DII</th>
<th>DIII</th>
</tr>
</thead>
<tbody>
<tr>
<td>P = 0.04 2/10 mvt.</td>
<td>P = 0.06 2/10 mvt.</td>
<td>P = 0.06 2/10 mvt.</td>
</tr>
<tr>
<td>P - R = 0.12</td>
<td>P - R = 0.12</td>
<td>R = 0.02 4/10 mvt.</td>
</tr>
<tr>
<td>R = 0.02 3/10 mvt.</td>
<td>R = 0.02 4/10 mvt.</td>
<td>S = 0.02 3/10 mvt.</td>
</tr>
<tr>
<td>S = 0.02 5/10 mvt.</td>
<td>S = 0.03 4/10 mvt.</td>
<td>R - T = 0.24</td>
</tr>
<tr>
<td>R - T = 0.24</td>
<td>R - T = 0.20</td>
<td>T = 0.04 1/10 mvt.</td>
</tr>
<tr>
<td>T = 0.12 2/10 mvt.</td>
<td>R = 0.08 3/10 mvt.</td>
<td>T - P = 0.14</td>
</tr>
<tr>
<td>T - P = 0.14</td>
<td>T - P = 0.14</td>
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</tbody>
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By the occasion in which this diagram was taken, the clinical condition of the patient was of course, already serious, but of still very uncertain prognosis. By then, the marked development of wave T was already noticeable.

2nd. Diagram on the seventh day of the illness (fig. 8).

<table>
<thead>
<tr>
<th>DI</th>
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<tbody>
<tr>
<td>P = Double 0.08 1/10 mvt.</td>
<td>P = 0.08 2/10 mvt.</td>
<td>P = 0.08 Double</td>
</tr>
<tr>
<td>P - R = 0.14</td>
<td>P - R = 0.16</td>
<td>P - Q = 0.16</td>
</tr>
<tr>
<td>R = 0.02</td>
<td>R = 0.02 3/10 mvt.</td>
<td>Q = 0.02 2/10 mvt.</td>
</tr>
<tr>
<td>S = 0.08</td>
<td>S = 0.08 2/10 mvt.</td>
<td>R = 0.08 6/10 mvt.</td>
</tr>
<tr>
<td>R - T = 0.30</td>
<td>R - T = 0.28</td>
<td>Q - T = 0.28</td>
</tr>
<tr>
<td>T = 0.12</td>
<td>T = 0.12 4/10 mvt.</td>
<td>T = 0.08 1/10 mvt.</td>
</tr>
<tr>
<td>T - P = 0.06</td>
<td>T - P = 0.06</td>
<td>T - P = 0.06</td>
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In this incidence, in proportion symptoms were becoming more serious, and the death was nearer, there appeared signs of defective conduction in the ventricles, specially of the left ventricle.


1st. Diagram on the day before the death (fig. 9). Regular rhythm rather accelerated. One sees already a large increase of wave T and deficient conduction in ventricles. These signs become exaggerated on the following day, a few hours before death.

<table>
<thead>
<tr>
<th>DI</th>
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<th>DIII</th>
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<tbody>
<tr>
<td>P = 0.02 2/10 mvt.</td>
<td>P = 0.08 2/10 mvt.</td>
<td>P = 0.02 2/10 mvt.</td>
</tr>
<tr>
<td>P–R = 0.10</td>
<td>P–R = 0.12</td>
<td>P–R = 0.12</td>
</tr>
<tr>
<td>R = 0.02 3/10 mvt.</td>
<td>R = 0.06 8/10 mvt.</td>
<td>R = 0.06 6/10 mvt.</td>
</tr>
<tr>
<td>S = 0.02 1/10 mvt.</td>
<td>S = 0.06 4/10 mvt.</td>
<td>S = 0.06 4/10 mvt.</td>
</tr>
<tr>
<td>R–T = 0.24</td>
<td>R–T = 0.24</td>
<td>R–T = 0.24</td>
</tr>
<tr>
<td>T = 0.08 3/10 mvt.</td>
<td>T = 0.08 8/10 mvt.</td>
<td>T = 0.12 6/10 mvt.</td>
</tr>
<tr>
<td>T–P = 0.12</td>
<td>T–P = 0.10</td>
<td>T–P = 0.10</td>
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2nd. Diagram (Fig. 10).

<table>
<thead>
<tr>
<th>DI</th>
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<tbody>
<tr>
<td>P = 0.08 2/10 mvt.</td>
<td>P = 0.08 2/10 mvt.</td>
<td>P = 0.08 1/10 mvt.</td>
</tr>
<tr>
<td>P–R = 0.12</td>
<td>P–R = 0.10</td>
<td>P–R = 0.10</td>
</tr>
<tr>
<td>R = 0.04 8/10 mvt.</td>
<td>R = 0.04 7/10 mvt.</td>
<td>R = 0.06 8/10 mvt.</td>
</tr>
<tr>
<td>S = 0.08 6/10 mvt.</td>
<td>S = 0.08 5/10 mvt.</td>
<td>S = 0.06 6/10 mvt.</td>
</tr>
<tr>
<td>R–T = 0.24</td>
<td>R–T = 0.24</td>
<td>R–T = 0.26</td>
</tr>
<tr>
<td>T = 0.08 7/10 mvt.</td>
<td>T = 0.10 9/10 mvt.</td>
<td>T = 0.12 8/10 mvt.</td>
</tr>
<tr>
<td>T–P = 0.08</td>
<td>T–P = 0.08</td>
<td>T–P = 0.08</td>
</tr>
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</table>


1st. Diagram on the third day of the illness (Fig. 11). Regular rhythm.

DII

<p>| |</p>
<table>
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<tbody>
<tr>
<td>P = 0.06 2/10 mvt.</td>
</tr>
<tr>
<td>P–R = 0.12</td>
</tr>
<tr>
<td>R = 0.02 3/10 mvt.</td>
</tr>
<tr>
<td>S = 0.02 4/10 mvt.</td>
</tr>
<tr>
<td>R–T = 0.28</td>
</tr>
<tr>
<td>T = 0.08 3/10 mvt.</td>
</tr>
<tr>
<td>T–P = 0.12</td>
</tr>
</tbody>
</table>
2nd. A diagram obtained four hours before death (fig. 12).

DII
\[
P = 0.08 \text{ 2/10 mvt. Double}
R = 0.12
S = 0.04 \text{ 7/10 mvt.}
T = 0.12 \text{ 7/10 mvt.}
T-P = 0.06
\]

In this case, signs of degeneration of the cardiac muscle did also appear.


1st. Diagram on the fourth day of the illness. Regular rhythm slightly accelerated (Fig. 13). The diagram taken on the 5th day showed already a considerable increase in the extension of wave T, and a sensible decrease in R. The interval R-R of 0.12 had already grown up to 0.16 on the following day, say on the sixth day in the morning; these signs showed a still higher increase (Figs. 14—16), the rhythm being frequently interrupted by more or less long crisis of heterotypic ventricular excitations, summed up, real crisis of extrasystolic, paroxystic tachycardia.

Curious it is to note that, for a short period after the tachycardiac crisis, the amplitude of wave T was considerably reduced (fig. 17). Fig. 18 shows diagram taken a few minutes before the death.

N. 8. J. 13 years. Masculine. Mestiço. Brazilian. Profession unknown. The patient did not give any information about the number of days he had felt ill before entering the hospital. He remained interned there a few hours only, died after that time.

1st. Diagram taken by the time of his admission (fig. 19) great tachycardy. Temperature 37.2. Great increase of wave T, which showed higher than R.

2nd. Diagram taken two hours before death, when the patient was seized by a great agitation and had abundant blood vomiting. The diagram shows a wave R exceedingly reduced; wave P twofold, deformed and very ample, and wave T, the greatest of all, considerably increased in relation with the foregoing diagram. (fig. 20).

Fig. 21 shows the diagram registered by the moment of the death. The auricular complexes are enlarged and deformed the ventricular complexes deeply aberrant, reminding the type of variations, subsequent to heterotopic excitation of the ventricles. Meanwhile, by comparing with diagrams obtained in the same stage, and at less advanced periods of other patients, we believe they represent the very homotopic excitation, deeply troubled in its transmission.


Remained in the Hospital for three hours only, and died after that time. No information about how many days of illness. Entered in severe condition, with very frequent blood vomiting. During the whole permanency
in the hospital, showed the rhythm represented in fig. 22. There may be seen a diagram similar to that of fig. 20, but with more accelerated rhythm. The great waves represent the variations T of the ventricular complexes, and the small loops which are seen immediately in front of same are the variations R of the same complexes. The auricular phenomena P, are mixed within the lowering branch of each one of the waves T.


The general condition of the patient was a relatively favourable up to the fifth day. The first diagram (fig. 23) was taken on the fourth day of the illness and shows a double P in DII. In all, it is perfectly normal. On the fifth day of the illness, the diagram of fig. 24 was taken. There, already, the great increase of wave T and sensible decrease of wave R was noticeable. The auricular variation P showed irregular, at times positive, otherwise negative. On the sixth day, few moments before death, the diagrams of figs. 25, 26 and 27 were taken. Fig. 25 shows a regular rhythm, slow, with the ventricular complexes of quite abnormal type and the auricular complexes, double and negative.

The diagram of fig. 26, few minutes before death, show the same alteration which account for the complexes of fig. 25, however at a more advanced stage. By the moment of death, the deformation of the electric variations of the ventricle increased more and more till the appearing of fibrillation, foregoing to stop of the heart. (Fig. 27)


The first diagram taken on the third day of infection, when general condition was rather favorable, was perfectly normal. (Fig. 28). On the sixth day, after plain symptoms of cardiac insufficiency had appeared the day before, the diagrams took the aspect of fig. 29. Ventricular phenomena in great decrease, with wave T diphasic. This aspect prevailed up to a collapse crisis with apnea, meaning death coming on. By this opportunity, the electro-cardiograms took successively the aspects as shown in Figs. 30, 31, 32 and 33. At first the ventricular excitations, absolutely abnormal, growing little by little more deformed until a slow and unifocal rhythm of the ventricles appears, to which fibrillation and death immediately follow.