AN ELECTROCARDIOGRAPHIC STUDY OF THE ANAPHYLACTIC RABBIT.*

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Plates 33 to 37.

INTRODUCTION.

The subject of cardiac disturbances in serum anaphylaxis has not received much attention from investigators. Gay and Southard,1 in their extensive histological studies, noted that the heart of the anaphylactic guinea pig often shows hemorrhages; they did not, however, observe any alteration in the functional activity of this organ, as far as we are aware. The first definite observation of a pathological alteration of the heart's activity during serum anaphylaxis was made by Auer and Lewis,2 who observed that the heart of a guinea pig dying from anaphylaxis showed several abrupt diminishations in rate, and that these rates were in simple ratio to each other. This observation was made by inspection of the exposed heart and by examination of graphic records. The block was thought to be due to asphyxia. This interpretation is in accord with the results Sherrington3 obtained with decapitated, atropinized cats, where asphyxia produced an abrupt halving of the cardiac rate; it also agrees with the work of Lewis and Mathison,4 who demonstrated the occurrence of heart-block in cats during asphyxia by means of the electrocardiograph.

During the anaphylactic reaction of the rabbit, one of us5 has noticed heart-block, both by inspection of the exposed heart and by a graphic method. Further and more detailed evidence will be given in this paper.

Cardiac disturbances during serum anaphylaxis of the cat have been described by Schultz,6 but his results are difficult to interpret with certainty. In the cat matters are complicated by the fact that horse serum exerts a strongly toxic effect in the unsensitized animal; as little as 0.25 c.c. per kilo usually kills young

*A preliminary note was published in the Zentralblatt für Physiologie, 1913, xxvii, 1. Received for publication, July 1, 1913.
3 Sherrington, C. S., Jour. Physiol., 1909, xxxviii, 381.
4 Lewis, T., and Mathison, G. C., Heart, 1910, ii, 47.
5 Auer, J., Jour. Exper. Med., 1911, xiv, 483 (see plate 44 and plate 47, figure 4).
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cats, while adult cats are strongly affected but "frequently recover" from this dose.⁷ Even 0.1 c.c. per kilo gives a fall in blood pressure both in normal and sensitized cats.⁸ The ordinary reinjection dose employed by Schultz seems to have been 0.2 c.c. per kilo. Moreover, Schultz observed traces of blood clot in the hearts of some anaphylactic cats.⁹ The cat thus differs strikingly from guinea pigs, rabbits, and dogs in its reaction to horse serum, for the latter animals show no definite toxic effects when comparatively large quantities of not too fresh horse serum are injected intravenously. A normal guinea pig tolerates 5 c.c., a normal rabbit 15 c.c., and a normal dog at least 30 c.c. of horse serum intravenously without any symptoms which even remotely resemble those occurring in anaphylaxis.¹⁰ A consideration of these data forces us to the conclusion that the cardiac disturbances noted by Schultz are not necessarily anaphylactic phenomena.

The anaphylactic dog also shows heart-block and other cardiac disturbances demonstrable by the electrocardiograph. A detailed statement of these changes will be found in another paper.¹¹

This brief review of the literature concerning heart disturbances in serum anaphylaxis of the intact animal shows definitely that alterations of the functional activity of this organ are common in this state and apparently form an integral part of anaphylaxis. In the following pages we shall give an account of the main changes which the heart of the anaphylactic rabbit reveals when examined by the electrocardiograph. Details regarding the process of sensitization, which is by no means as certain in the rabbit as in the guinea pig, will be found in a former paper by one of us.

EXPERIMENTAL PART.

The cardiac changes of anaphylactic reaction were studied in twenty-four rabbits. Young animals weighing about one kilo were sensitized by repeated subcutaneous injections of horse serum. About four weeks after the last injection, the animals were given the final, toxic dose of about 9 c.c. into a marginal ear vein, and the cardiac disturbances were studied by means of electrocardiograms taken at short intervals immediately before, during, and after the injection. The records were made with the large Edelmann string galvanometer. The animals were connected with the instrument by electrodes.

¹⁰ These doses do not represent the maximal quantities which are tolerated; they are quoted because such doses have been injected in normal animals during the course of other experiments.
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usually attached to the right fore leg and left hind leg (the second lead). The string of the galvanometer was arranged so that a deviation of 1 cm. in the curve represents the passage of 1 millivolt. No ether was given during the experiment, and the slight operative procedures hardly demanded it. In eleven of the sensitized rabbits both vagi were cut before the toxic dose of serum was injected and after tracheotomy had been performed. Horse serum was also injected intravenously into two non-sensitized rabbits, from which electrocardiographic records were obtained in the same way as from the others.

The rate of the heart-beat, the relation of auricular to ventricular contractions, and the form of the electrocardiograms were especially considered in studying the records obtained in these experiments. Definite abnormalities in the heart-beat were observed in twenty-two of the twenty-four experiments in which sensitized rabbits were used, and in ten experiments the anaphylactic reaction was fatal, the rabbits dying in from two to nineteen minutes. The average time of death was seven minutes after the beginning of the intravenous injection. In these experiments it is necessary to separate the cardiac disturbances which resulted primarily from the anaphylactic reaction, from those which occurred as phenomena of the dying heart, and cessation of respiration was therefore used as the index of death. Changes in the heart-beat, which occurred after respiration had ceased, were not considered as phenomena of the anaphylactic reaction, although in some instances the electrocardiograms returned to normal after respiration had ceased, while they had been markedly abnormal before.

The intravenous injection of horse serum into two non-sensitized normal rabbits was followed by no such changes in the electrocardiograms as were seen in the sensitized animals. In one control experiment the records indicated that premature ectopic ventricular beats began to occur occasionally about thirty seconds after the beginning of the injection of horse serum and before it was completed. The ectopic beats became more numerous, and one minute after the beginning of the injection they alternated with the normal beats. These ectopic ventricular contractions gradually became less numerous, and in three and a half minutes after the beginning of the injections the records show that they had ceased to occur. Three days later this rabbit was again attached to the string galvanometer and given an intravenous injection of nine cubic centimeters of
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normal saline solution. No disturbances of the cardiac mechanism occurred. In the second control experiment the records show that no cardiac disturbances followed the intravenous injection of horse serum.

A second intravenous injection of horse serum was given to seven sensitized rabbits after the effects of the first injection had passed off. All but one of the rabbits in these experiments had shown marked cardiac disturbances after the first injection. After the second injection no disturbances of the heart-beat or other symptoms occurred except in one experiment in which ectopic ventricular contractions appeared. These animals were thus in the anti-anaphylactic state at this time. The fact that ectopic ventricular contractions occurred after the second injection in one of these animals and also in one of the non-sensitized animals after a single injection of horse serum, indicates that the occurrence of such beats probably cannot be considered as significant when they occur during the anaphylactic reaction.

The condition of the various experiments and the results may be summarized briefly as follows:

<table>
<thead>
<tr>
<th>No. of experiment</th>
<th>Result</th>
<th>Vagi.</th>
<th>Complex changes before cessation of respiration.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Fatal</td>
<td>Intact</td>
<td>Marked. Temporary partial block. Slight changes in form.</td>
</tr>
<tr>
<td>3</td>
<td>Fatal</td>
<td>Intact</td>
<td>Marked. Temporary partial block. T wave much increased.</td>
</tr>
<tr>
<td>6</td>
<td>Fatal</td>
<td>Intact</td>
<td>Marked. Abnormal relation of P and R waves. S wave developed.</td>
</tr>
<tr>
<td>8</td>
<td>Fatal</td>
<td>Intact</td>
<td>Marked. Temporary block twice. Marked S and negative T waves developed.</td>
</tr>
<tr>
<td>10</td>
<td>Fatal</td>
<td>Cut</td>
<td>Marked. S wave and negative T wave developed.</td>
</tr>
<tr>
<td>17</td>
<td>Fatal</td>
<td>Cut</td>
<td>Marked. P waves disappear. Possible auricular fibrillation.</td>
</tr>
<tr>
<td>19</td>
<td>Fatal</td>
<td>Cut</td>
<td>Marked. T partly fused with R waves.</td>
</tr>
<tr>
<td>4</td>
<td>Not fatal</td>
<td>Intact</td>
<td>Moderate. P wave disappears. T wave becomes absent.</td>
</tr>
<tr>
<td>9</td>
<td>Not fatal</td>
<td>Intact</td>
<td>Marked. Abnormal relation of P and R waves. T wave disappears.</td>
</tr>
<tr>
<td>11</td>
<td>Not fatal</td>
<td>Intact</td>
<td>Marked. Abnormal relation of P and R waves. Large S and negative T waves.</td>
</tr>
</tbody>
</table>
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<table>
<thead>
<tr>
<th>No. of experiment</th>
<th>Result</th>
<th>Vagi</th>
<th>Complex changes before cessation of respiration</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>Not fatal</td>
<td>Intact</td>
<td>Slight. S wave develops. T wave becomes negative.</td>
</tr>
<tr>
<td>14</td>
<td>Not fatal</td>
<td>Intact</td>
<td>Slight. S wave develops. T wave disappears.</td>
</tr>
<tr>
<td>18</td>
<td>Not fatal</td>
<td>Cut</td>
<td>Marked. Prominent S wave develops and large T waves.</td>
</tr>
<tr>
<td>21</td>
<td>Not fatal</td>
<td>Cut</td>
<td>Marked. R waves broad. T waves negative.</td>
</tr>
<tr>
<td>22</td>
<td>Not fatal</td>
<td>Cut</td>
<td>Marked. Broad, deep S wave.</td>
</tr>
<tr>
<td>23</td>
<td>Not fatal</td>
<td>Cut</td>
<td>Moderate. S wave develops. T wave disappears.</td>
</tr>
<tr>
<td>26</td>
<td>Not fatal</td>
<td>Cut</td>
<td>No definite changes.</td>
</tr>
<tr>
<td>15</td>
<td>Control</td>
<td>Intact</td>
<td>No changes except ectopic ventricular beats.</td>
</tr>
<tr>
<td>16</td>
<td>Control</td>
<td>Intact</td>
<td>No definite changes.</td>
</tr>
</tbody>
</table>

The experiments may be grouped into those in which the anaphylactic reaction was fatal, and those in which the animals recovered. These groups may be subdivided according to whether the vagi were intact or cut.

The changes in cardiac rate may be considered, however, independent of these groups, for seventeen of the twenty-four experiments showed an alteration of rate which bore apparently no relation to the ultimate outcome of the experiments, nor to whether the vagi were intact or not. This change consisted of a slowing of twenty-five beats or more during the first minute after beginning the injection, and then an increase to a rate twenty-five beats or more per minute faster than that present before the injection. This change of rate may be illustrated by a chart constructed from experiment 12, a non-fatal experiment with the vagi intact (text-figure 1). The same initial slowing of the heart is shown in blood pressure records.\(^\text{12}\)

The first group consists of six experiments in which the final injection of horse serum was fatal and in which the vagi were intact. In all but one of these cases marked disturbances of the heart-beat developed swiftly, often before the end of the serum injection, and at a time when the respiration was practically undisturbed. The

most striking disturbance was partial heart-block, which occurred in three of the cases. This condition was always temporary, and the normal sequential beat was established before the changes of the dying heart occurred. In experiment 8 partial heart-block occurred one and a half minutes after the beginning of the injection (figures 1 and 2). The normal sequential beat returned forty-five

![Text-fig. 1. Experiment 12. The changes in the cardiac rate in anaphylactic shock.](image-url)
seconds later with an increase in rate and a moderate change in the form of the complexes (figure 3). Partial heart-block appeared again in one and a quarter minutes, when the complexes had changed markedly in form (figure 4). The block had disappeared five minutes after the beginning of injection (figure 5), but returned again as a phenomenon of the dying heart in less than one minute (figures 6 and 7). In experiment 3 (figure 8) a partial heart-block was present one minute and a half after the injection (figure 9), while in three minutes complete dissociation of auricles and ventricles developed (figure 10). The sequential beat had returned at four minutes after the injection, but with a marked delay in the conduction time (figure 11). Half a minute later partial block had again set in (figure 12). The various electrocardiograms obtained from this animal showed marked changes in form, and the record obtained shortly after the injection shows ventricular complexes very much of the same form as those taken eleven minutes after the injection and one minute after respiration had ceased (figure 13). In each, the sharp, rather prominent T waves are close to the R waves, the down strokes of which are slow and do not reach the base line. This form of ventricular complex seems to be characteristic of the dying heart, as shown by one of us (Robinson). In experiment 2 partial heart-block was present from two to four and a half minutes after the injection and returned again after the normal sequential beat had been present for half a minute, continuing until respiration ceased ten minutes after the injection.

In two experiments an abnormal relation between the P and R waves occurred in the electrocardiograms. This consisted of a temporary shortening of the conduction (P–R) time. Thus in experiment 6 the P–R time was 0.08 of a second before the injection (figure 14), while it became reduced to 0.033 of a second two minutes after the injection was begun (figure 15). In half a minute the P–R time had lengthened to 0.068 of a second. In experiment 5 the P–R time was diminished at forty-five seconds after the onset of the injection, and remained so for one and a quarter minutes. The P–R relation was then normal for one minute, when

\[^{18}\text{Robinson, G. C., Jour. Exper. Med., 1912, xvi, 291.}\]
the P and R waves were again abnormally near each other and partially fused. This same change in the relation of the P and R waves occurred in the electrocardiograms from five other experiments in which the anaphylactic shock was not fatal, three in which the vagi were intact, and two in which they had been cut.

In the four experiments in which a fatal anaphylactic shock occurred after the vagi had been cut, definite abnormalities in the forms of the electrocardiograms appeared in forty-five seconds, one and a half minutes, two minutes, and two and a half minutes respectively after the beginning of the injection. In one experiment respiration ceased at about the same time that the electrocardiograms became abnormal, but in the others intervals of two, four, and eight minutes respectively separated the appearance of abnormalities in the electrocardiograms and the cessation of respiration. The changes in form of the electrocardiograms from these experiments were various. In two the P wave disappeared, and in one of these the electrocardiograms suggest indefinitely that auricular fibrillation was present for a short time. The T waves of ventricular complexes changed definitely in three of the experiments, in two occurring close to or partly fused with the R wave. In the other experiment which showed abnormal T waves, they gradually diminished in size and then became definitely negative or downwardly directed, this change beginning two and a half minutes after beginning the injection and reaching its maximum one and a half minutes later (figures 16 and 17). Thus it is seen that changes occurred in the electrocardiograms from this group of experiments which indicate that both the auricular and the ventricular activity were disturbed, while no definite disturbances of conduction took place.

The experiments in which the anaphylactic shock was not fatal, are conveniently divided into those in which the vagi were intact and those in which the vagi were cut. There were seven experiments with intact vagi, and the electrocardiograms from all but one showed changes which were marked in three, moderate in one, and slight in two. The abnormalities in the electrocardiograms were first seen in from one to three and a half minutes after the beginning of the injection. In all these experiments but one, the electo-
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cardiograms returned to the form seen before the injection, the time varying from seven and a half to twenty-one minutes. These changes vary in the different experiments. Those which occurred in experiment II are shown in figures 18 to 21; before injection the complexes are well formed, the P–R time is 0.07 and the rate 281; three and a quarter minutes later the R wave is followed by a negative phase, the P–R time is unchanged, while the rate has decreased to 174 beats per minute; fifteen seconds after this a marked arrhythmia with varying electrocardiograms set in, which disappeared in another fifteen seconds, when very abnormal electrocardiograms occurred. This form, consisting mostly of a broad negative wave, persisted until five and a half minutes after the injection, when it began to return gradually to the form seen before the injection.

The T waves were changed in four of these seven experiments, disappearing in three, and in one becoming downwardly directed. The P and R waves had an abnormal relation, temporarily, in three experiments, becoming approximated as in experiment 6 (figures 14 and 15), and in one experiment the P wave disappeared.

Of the seven non-fatal experiments in which the vagi were cut, five showed marked changes in the form of the electrocardiograms; in one there were moderate changes, and in one no changes were seen. The changes appeared after one to five minutes, and the abnormalities lasted from two and a half to five minutes. The changes in this group resemble those which occurred in the non-fatal experiments where the vagi were not cut. In four experiments large, broad, downwardly directed S waves appeared. The T wave changed in three, once increasing in size, once disappearing, and once becoming downwardly directed. In two experiments the P approximated the R waves, and in one the P waves disappeared. An example of the marked changes which occurred in the electrocardiograms of this group of cases is given in figures 22 to 24.

DISCUSSION.

The experiments which have been described show that in the anaphylactic reaction produced by intravenous injections of horse serum into sensitized rabbits, disturbances of the heart occur in a
large majority of cases. These disturbances are seen in non-fatal as well as in fatal anaphylaxis, and after the vagi are cut as well as when they are intact. There were but two negative experiments in twenty-four, and in these the rabbits showed no signs of anaphylaxis. It is thus evident that cardiac disturbances are practically constantly present in rabbits during the anaphylactic reaction, in contrast to dogs, where changes in the heart-beat were noted in but six of twelve experiments, although a marked fall of blood pressure was constant. An intravenous injection of horse serum does not cause such cardiac disturbances in non-sensitized rabbits, nor in rabbits in the anti-anaphylactic state.

The cardiac disturbances vary considerably. They may consist of disturbances in conduction, giving rise to partial heart-block, which occasionally goes on to complete dissociation of auricles and ventricles. Partial heart-block occurred in our series only in experiments in which there was fatal anaphylaxis and in which the vagi were intact. The number of experiments is too small, however, to indicate whether or not it is only under such conditions that partial heart-block may develop. That the relationship between auricular and ventricular activities may become disturbed is indicated by the abnormal proximity of the P and R waves (figure 15). This change, which was seen only once in the series on dogs, occurred in seven experiments of this series. Its significance has been discussed in our paper on cardiac anaphylaxis in the dog; it probably represents a change in the position of the point at which the stimulus of the heart-beat arises.

The changes in form of the electrocardiograms, especially of the portions representing ventricular systole, are various. The waves most frequently altered are the T waves, which disappeared in six experiments, became negative or downwardly directed in five, and increased in size in four. In eleven experiments, negative or downwardly directed S waves developed during the anaphylactic reaction. The changes in the form of the electrocardiograms suggest damage to the ventricles, which renders their contractions abnormal. A discussion of the various forms encountered is not in the scope of this paper. It may be said, however, that changes occur similar

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\textsuperscript{14} Robinson, G. C., and Auer, J., \textit{loc. cit.}
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to those obtained by Eppinger and Rothberger\textsuperscript{15} following the injections of toxic substances directly into the ventricular musculature of the dog.

The cardiac disturbances in sensitized rabbits occur very soon after the intravenous injections of serum, in some experiments before the end of the injection. They were usually observed from thirty seconds to three minutes after the beginning of the injection, although in a few experiments five minutes elapsed before cardiac disturbances occurred.

The heart apparently recovers quickly in the non-fatal experiments, and electrocardiograms similar to those obtained before the injection appeared from two and a half to eighteen minutes after the abnormalities set in. The oscillation between normal and abnormal forms of the electrocardiograms was usually gradual, but in several experiments marked abnormalities occurred or disappeared suddenly. In the fatal cases it was also noted that early occurring abnormalities tended to disappear before the changes of the dying heart set in. This was seen especially well in those experiments where partial heart-block occurred, for the normal sequential beat was usually reestablished once or even twice before the final changes of cardiac death appeared.

**SUMMARY.**

Electrocardiographic examination of rabbits during the anaphylactic reaction revealed marked and various changes of the heart’s activity in twenty-two out of twenty-four animals. Changes occurred in fatal as well as in non-fatal cases, after the vagi were cut as well as when they were intact. Cardiac disturbances are thus a practically constant result of serum anaphylaxis in the rabbit. It therefore is possible that anaphylaxis plays a rôle in the causation of certain cardiac derangements in man.

EXPLANATION OF PLATES 10

PLATE 33.

Fig. 1. Experiment 8. Electrocardiogram taken 1 minute after the beginning of the injection. Rate = 233. P-R time = 0.075 of a second.

Fig. 2. Experiment 8. ½ minute later, and before the end of the injection. Auricular rate = 225. Ventricular rate = 112. Partial heart-block.

Fig. 3. Experiment 8. 3½ minutes after the beginning of the injection. Rate = 300. P-R time = 0.06 of a second. Partial heart-block has disappeared. T wave has diminished. S wave has developed.

Fig. 4. Experiment 8. 4 minutes after the beginning of the injection. Auricular rate = 300. Ventricular rate = 150. Partial heart-block has returned. P waves indefinite. Well-marked negative or downwardly directed T waves.

Fig. 5. Experiment 8. 5 minutes after the beginning of the injection. Rate = 205. Partial block has again disappeared. P-R time = 0.072 to 0.08 of a second. Respiration ceased half a minute later.

Fig. 6. Experiment 8. 5½ minutes after the beginning of the injection. Auricular rate = 312. Partial heart-block developing.

Fig. 7. Experiment 8. ½ minute later. Partial heart-block. Auricular rate = 300. Ventricular rate = 150.

10 Time is marked in 1/5 and 1/50 of a second.
PLATE 34.

Fig. 8. Experiment 3. Electrocardiogram taken before injection. Rate = 300. P-R time = 0.066 of a second.

Fig. 9. Experiment 3. 1½ minutes after the beginning of the injection. Auricular rate = 280. Ventricular rate = 140. Partial heart-block. The ventricular complexes have the form often caused by dying hearts.

Fig. 10. Experiment 3. 3 minutes after the beginning of the injection. Auricular rate = 105. Ventricular rate = 108. Complete dissociation of auricles and ventricles.

Fig. 11. Experiment 3. 4 minutes after the beginning of the injection. Rate = 261. Heart-block has disappeared. P-R time = 0.11 of a second.

Fig. 12. Experiment 3. 4½ minutes after the beginning of the injection. Auricular rate = 255. Partial heart-block. 2:1 rhythm changing over to 3:1 rhythm.

Fig. 13. Experiment 3. 11 minutes after the beginning of the injection. 1 minute after respiration had ceased. The form of the ventricular portions of the electrocardiograms resembles those seen in figure 9. Partial heart-block.
PLATE 35.

Fig. 14. Experiment 6. Electrocardiogram obtained before injection. Rate = 214. P-R time = 0.08 of a second.

Fig. 15. Experiment 6. 2 minutes after the beginning of the injection. Rate = 212. P-R time = 0.033 of a second. Abnormal relation of auricular and ventricular activity.

Fig. 16. Experiment 10. Before injection. Rate = 270. P-R time = 0.064 of a second. T wave well formed.

Fig. 17. Experiment 10. 5 minutes after the beginning of the injection. Rate = 265. P-R time = 0.055 of a second. T wave has become negative or downwardly directed.
Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

(Auer and Robinson: Electrocardiographic Study of the Anaphylactic Rabbit.)
Plate 36.

Fig. 18. Experiment 11. Before injection. Rate = 281. P-R time = 0.07 of a second.

Fig. 19. Experiment 11. 3½ minutes after the beginning of the injection. Rate = 174. P-R time = 0.07 of a second. The form of the ventricular portions of the electrocardiograms is changed, the R wave becoming larger and being followed by a broad depression.

Fig. 20. Experiment 11. 5½ minutes after the beginning of the injection. Rate = 279. P-R time = 0.05 of a second. Ventricular portions of electrocardiograms markedly abnormal.

Fig. 21. Experiment 11. 20 minutes after the beginning of the injection. Rate = 250. P-R time = 0.07 of a second. The electrocardiograms resemble those of the control curve. Rabbit recovered.
Fig. 18.

Fig. 19.

Fig. 20.

Fig. 21.

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PLATE 37.

Fig. 22. Experiment 24. Before injection. Rate = 271. P-R time = 0.06 of a second. The electrocardiogram is normal.

Fig. 23. Experiment 24. 2 minutes after the beginning of the injection. Rate = 281. P-R time = 0.04 of a second. Ventricular portions of the electrocardiograms become suddenly very abnormal.

Fig. 24. Experiment 24. 5 minutes after the beginning of the injection. Rate = 262. P-R time = 0.06 of a second. The electrocardiograms return suddenly to the form obtained before the injection.
Fig. 22.

Before injection. Rate: 271

Fig. 23.

2 minutes after injection. Rate: 291

Fig. 24.

5 minutes after injection. Rate: 263

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