

## SOME OBSERVATIONS ON HEART-BLOCK \*

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The subject of heart-block has held a prominent position in recent medical literature. Certain features of the following cases of auricular-ventricular block appeal to me as deserving of record and discussion. They are taken from the records of the University Hospital and presented in this form on the recommendation of Dr. A. W. Hewlett, to whom I am indebted for many valuable suggestions.

*CASE 1.—History.*—B. E. M., 28, student, was admitted May 20, 1909, complaining of dizziness and dyspnea on exertion. His family history and habits were good. Two years previously he suffered from a very severe tonsillitis followed by acute rheumatism. During the second week of his illness the heart-rate increased out of proportion to his illness, rising to 170 on sitting up. There was no pain but excessive nervousness and a small goiter developed. He improved on thyroidectin after seven months in bed, the pulse-rate dropping to 80-90 at the end of that time. This rate has persisted since then though he has had occasional attacks of "heart hurry." Two weeks previous to admission he had tonsillitis with little fever, and a week later the tachycardia returned with dizziness and dyspnea. After two days the heart-rate fell to 50, rose gradually to 90 and then suddenly to 160.

*Examination.*—Color good. Skin moist and warm. A small soft goiter, no thrill, no murmur, no eye signs. Slight lagging over the right upper thorax, auscultation of lungs negative. Some evidence of cardiac enlargement; a faint diastolic murmur noted in the pulmonary area. Radial pulse soft, large, and quick, with no irregularity.

*Course of Disease.*—Within a few hours after admission a partial heart-block was noticed with variable ratio between the auricular and ventricular rates, at one time a 2-1 rhythm persisted for a short time, but usually the ratio was 3-2. The *a-c* intervals in seconds were estimated as follows: 0.27-0.36-0.44-block, 0.26-0.36-0.40-block, etc. The shortest measured interval was 0.25 second and the longest 0.57 second. Following 1/50 gr. atropin the pulse became regular with an *a-c* interval of 0.27-0.30 second. Twenty minutes later there were five intermissions in one minute and five hours later the former arrhythmia had been resumed. The following morning 1/100 gr. atropin was given and the pulse became and remained regular.

Oct. 25, 1909: All symptoms had disappeared, the orthodiagram showed a reduction of 9 per cent. in the area of the heart shadow and there was no murmur.

Oct. 19, 1910: The patient returned with an afternoon temperature, a chronic cough and definite signs on the left upper chest behind. No tubercle bacilli were found. He now complained of intermissions in the pulse associated with a thump in the pericardiac region causing a cough. These were due to ventricular extrasystoles. The pulse-rate ranged from 80 to 100 per minute.

The heart-block in this case may be ascribed either to nervous influences or to the infection, both of which are commonly recognized as

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causes of dissociation. It is to be noted, however, that the irregularity followed a period of rapid pulse-rate which in itself may lead to partial heart-block as has been shown by Erlanger and Hirschfelder.<sup>1</sup>

The prompt improvement under the administration of atropin is convincing proof of the presence of some vagus effect. The action of the drug was not the usual one, however, as may be seen from the following:

Date	Time	Auricular Rate	Rhythm
5/20/09	At entrance	110	2-1 and 3-2
5/21/09	Before atropin	110	3-2
5/21/09	2 hrs. after atropin	95	Regular or occasional block
5/21/09	5 hrs. after atropin	90	4-3
5/22/09		82	Occasional block
5/22/09		72	Regular

The atropin produced a disappearance of the block with an improvement in the transmission time but the auricular rate has fallen rather than increased as is the usual effect. A similar experience in a case of partial block is recorded by Griffin and Cohn.<sup>2</sup>

**CASE 2.—History.**—S. M. B., April 12, 1910, was admitted to the otological clinic for the radical mastoid operation. Except for chronic alcoholism his previous history was negative. On April 18, operation was deferred because of delirium tremens.

**Operation.**—On May 9 patient was operated on under ether anesthesia. The lateral sinus was exposed, also the dura over the roof of the antrum low down.

**Course of Disease.**—The temperature fell to normal on the following day. On May 15 the patient complained of sharp darting pains in the head and over the face. The temperature rose to 102.2 and there was some evidence of infection in the wound. An irregular temperature persisted until the 24th. On the 23d the pulse-rate fell to 48 in the presence of a temperature of 101.2 F. and for four days the rate never rose above 56. A neurological examination on this date showed nothing. On May 26 it was noted that the pulse was irregular as well as infrequent and a tracing was taken showing an incomplete block with a 3-2 and 4-3 rhythm. The range of the *a-c* intervals was 0.27-0.42 second.

Before the action of atropin or mechanical stimulation of the vagus could be tried the pulse became regular and remained so thereafter. Tracings taken the following day were normal with an average *a-c* interval of about 0.27 second. During the several months' residence in the hospital the arrhythmia was never again observed.

The arrhythmia in this case, as in the preceding one, was probably due to vagus influence plus infection. In the absence of neurological evidence we are scarcely justified in assuming a local meningitis, or irritation of the vagus nerve. Unfortunately we were unable to apply the atropin test before recovery although we were saved, thereby, from a

1. Erlanger and Hirschfelder: Physiology of Heart-Block in Mammals, *Am. Jour. Physiol.*, 1906, xv, 153.

2. Griffith and Conn: Remarks on the Study of a Case Showing a Greatly Lengthened *a-c* Interval with Attacks of Partial and of Complete Heart-Block, with an Investigation of the Underlying Pathological Conditions, *Quart. Jour. Med.*, 1910, iii, 126.

probable misinterpretation of our results as *propter hoc* instead of *post hoc* in case it had resulted favorably.

CASE 3.—*History*.—March 13, 1909, a musician, aged 54, came complaining of shortness of breath and edema. He had all the features of a chronic interstitial nephritis with cardiac failure, relative murmurs, enlarged liver, chronic emphysema and a systolic pressure of 210 mm. Hg. As his condition was urgent, he was given 1 mg. of strophanthin and its action controlled by plethysmographic records. No irregularity was noted during the short period preceding administration of the drug. Within two or three minutes after this the tracings became slow and markedly irregular, enough to give rise to considerable alarm on the part of the attendants. The patient was not aware of any change in his condition.

At 5:30 (an hour later) phlebograms were taken. At the time these could not be interpreted, but they were later and in the light of clearer tracings recognized as those of partial block with a 3 to 1 rhythm; by the 17th tracings were obtained which were recognized as those of a partial heart-block. On the 20th 1/100 gr. atropin was given without recognizable influence on the arrhythmia. Mechanical and thermic stimuli produced no definite change. On March 22 digitalis was resumed in doses of 80 drops of the tincture per day for five days. The block still continued to come and go. It did not seem to be aggravated by digitalis.

The *a-c* interval was carefully estimated in a considerable number of tracings. The following are the averages for different rhythms:

During regular rhythm.....	.17-.22 sec. average .20
Occasional block .....	.13-.22 up to .35-block
3-2 rhythm .....	.13-.24 block
2-1 rhythm .....	.17-block

It is worthy of note that the shortest *a-c* interval (following a block) is considerably shorter than the usually accepted normal interval and that the interval just preceding the blocked stimulus shows comparatively little increase over the normal.

James MacKenzie<sup>3</sup> first reported a partial heart-block after giving members of the digitalis group, and this was later confirmed by numerous observers. We believe this case unique in the suddenness of the onset.

The production of partial heart-block by digitalis is usually ascribed to the stimulation of the vagus inhibitory mechanism, especially where an abnormally long *a-c* interval previously existed (MacKenzie<sup>4</sup>). Cushny<sup>5</sup> suggests that direct action on the conductivity of the bundle of His may be an important factor since in animals a complete block induced by digitalis is often refractory to large doses of atropin. In partial block the vagus action in man has been shown to persist by the fact that block may be induced in some cases by vagus compression or even swal-

3. MacKenzie: Diseases of the Heart, London, 1908, p. 180.

4. MacKenzie: New Methods of Studying the Heart, Brit. Med. Jour., 1905, ii, 587, 702, 759; Clinical Methods of Recognizing Heart-Block, Brit. Med. Jour., 1906, ii, 1107.

5. Cushny: The Therapeutics of Digitalis and Its Allies, Am. Jour. Med. Sc., 1911, cxii, 469.

lowing. Rihl<sup>6</sup> and H. E. Herring<sup>7</sup> state that in all previously reported cases of digitalis block the auricular rate has been increased during the irregularity. The case of A. W. Hewlett<sup>8</sup> seems to be the only exception to this rule.

This case under discussion showed this increase in auricular rate in remarkable degree as may be seen from the following table:

Time	Auric. rate	Ventric. rate	Rhythm
At entrance—			
3/13/09 .....	125	125	Regular
After strophanthin—			
3/13/09 .....	162	82	2-1
3/15/09 .....	98	98	Regular
3/17/09 .....	120	80	3-2
3/18/09 .....	116	82-116	Variable
3/26/09 .....	110	110	Regular
3/27/09 .....	120	280	3-2

It will be observed that we have here the reverse of the paradoxical action of atropin recorded in our first case, in that a drug which usually slows the auricle by vagus inhibition caused an increased auricular rate. Moreover, the degree of the block varies with the auricular rate. Nor does the exhibition of atropin alter the picture to any considerable degree. It has been suggested that the mechanical effect of the auricular stasis stimulates the sinus to more frequent stimulus production. This by its tendency to aggravate the block establishes a vicious circle which may be interrupted either by improving conduction or slowing the auricle.

From the reports of Bachman<sup>9</sup> it appears that, in complete block, conditions are very different, since atropin in such cases increased the auricular without influencing the ventricular rate and the digitalis bodies slowed the auricular but increased the ventricular rate, that is, the usual vagus effect is obtained with both atropin and digitalis, while the increased ventricular rate with the latter is ascribed to direct muscular action.

CASE 4.—*History*.—A. B., 72, colored, came to the hospital April 29, 1910, complaining of shortness of breath and weakness. His first illness began seventeen years ago with symptoms of a urethritis and cystitis. Three years ago, a retention developed and suprapubic drainage for eight weeks was necessary. About a month previous to his entrance to the hospital, he suddenly became unconscious as he was sitting quietly. He fell to the floor and "believes he was paralyzed." Since then he had been short of breath, had coughed and grown weaker so that finally he went to bed. There had been some pain below the left clavicle on deep breathing. The left side had been tapped and considerable fluid drawn off; the patient did not know how much.

6. Rihl: Klinischer Beitrag zur Kenntnis der Ueberleitungsstörungen von der Bildungsstätte der Ursprungsreize zum Vorhof, *Deutsch. Arch. f. klin. Med.*, 1908, xciv, 286.

7. H. E. Hering: *Verhandl. d. Congr. f. inn. Medicin*, 23. Congr., 1905, p. 153.

8. Hewlett: *Digitalis Heart-Block*, *Jour. Am. Med. Assn.*, 1907, xlviii, 47.

9. Bachman: *Sphygmographic Study of Complete Heart-Block*, *THE ARCHIVES INT. MED.*, 1909, iv, 238.



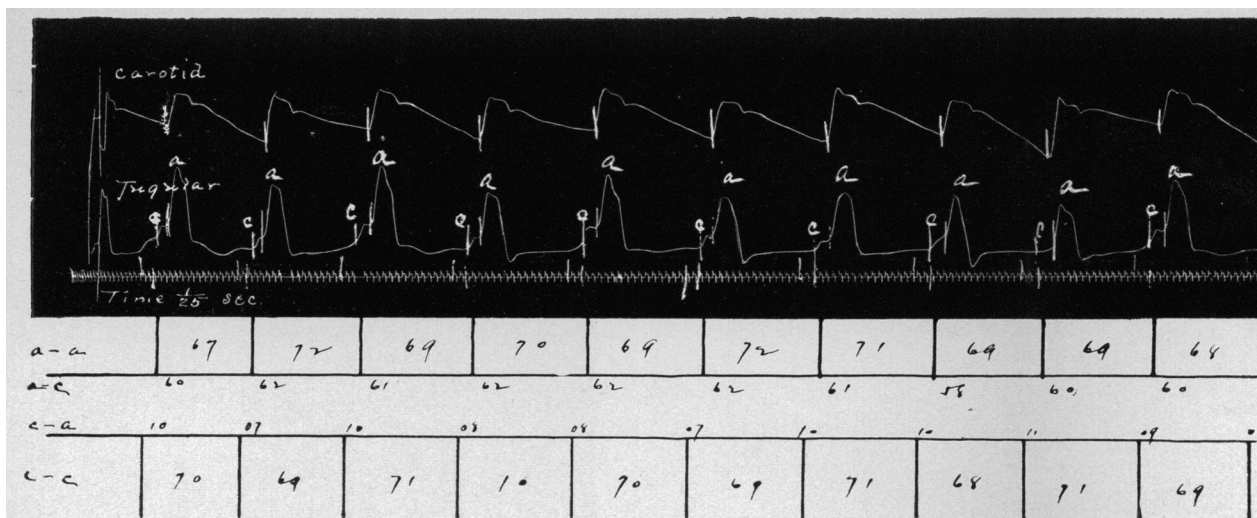


Fig. 1. Regular rhythm; the ventricles respond to the stimulus represented by the auricular systole occurring during ventricular systole.

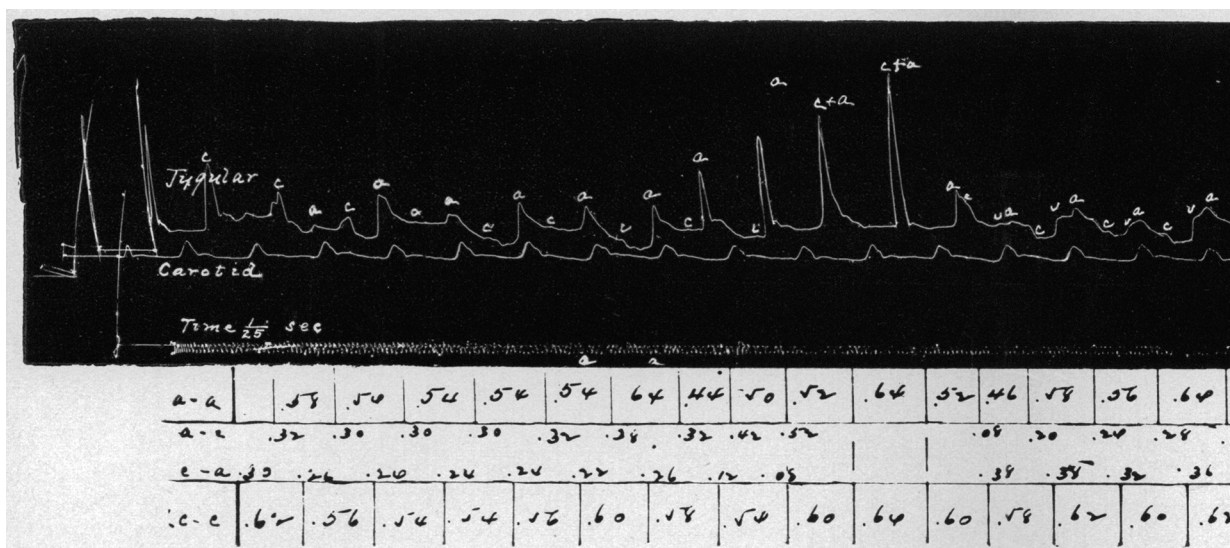


Fig. 3.—The ventricular rhythm is regular, the auricular irregular. The *a* wave is decidedly with no att

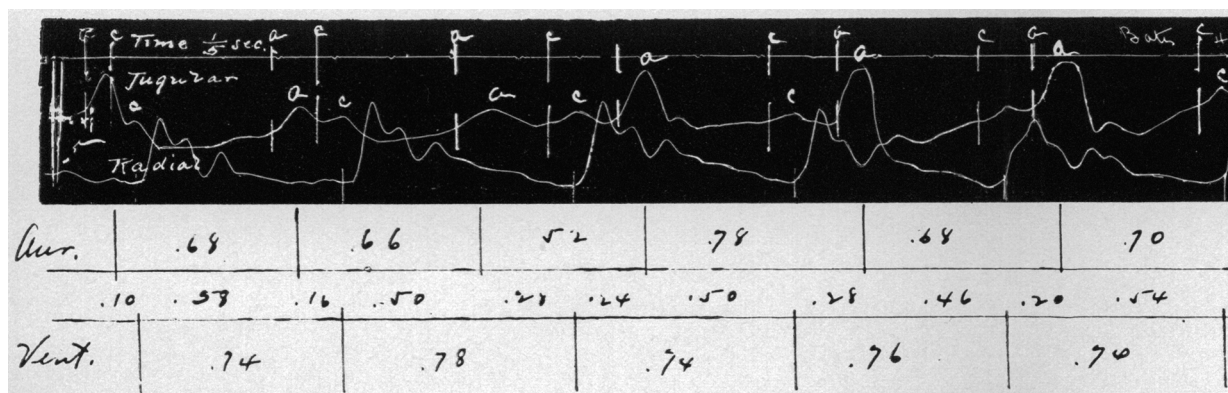
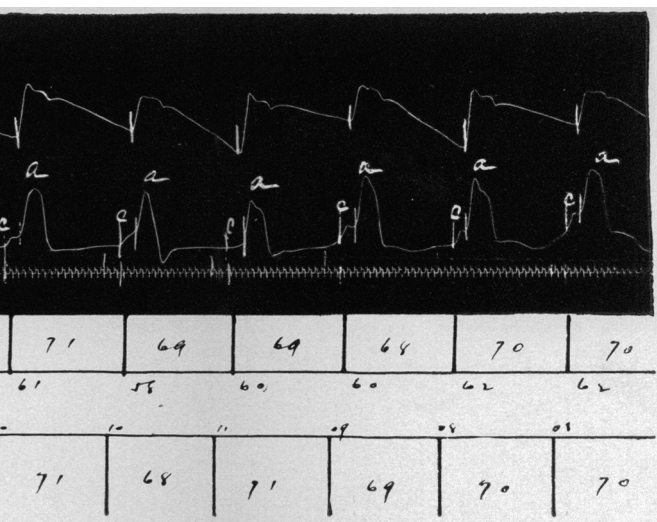


Fig. 4.—Similar to Figure 3. With the slower



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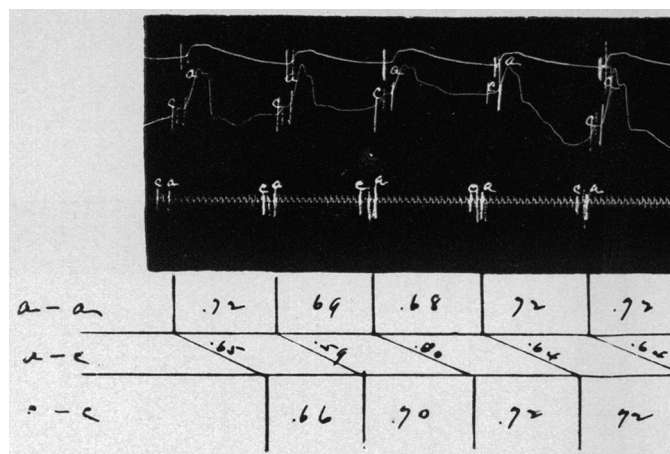
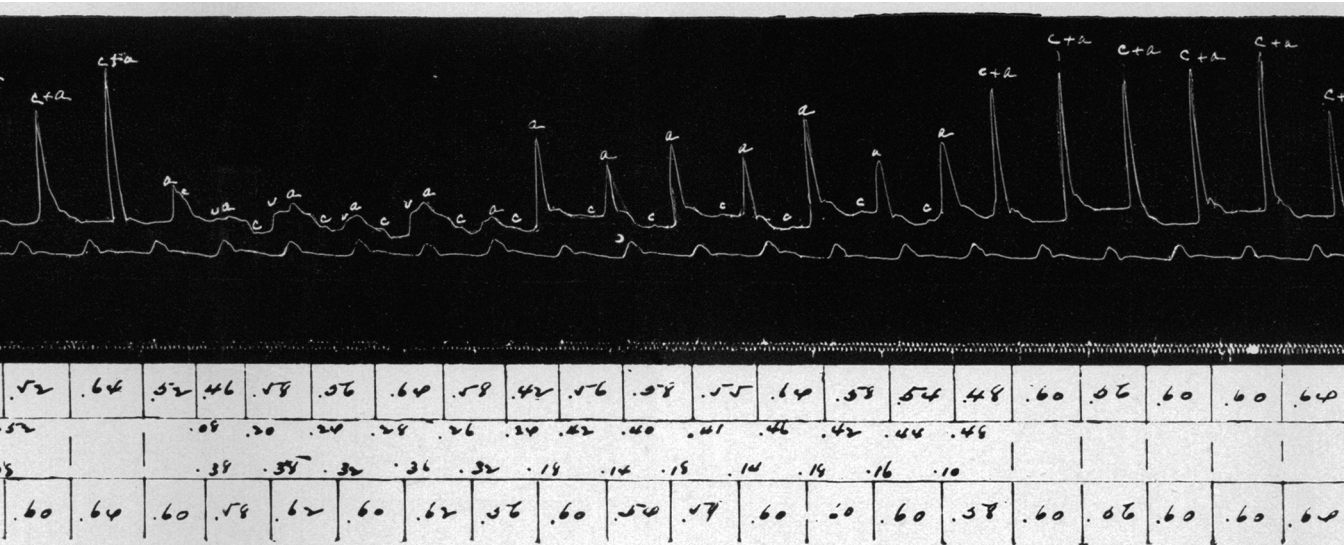
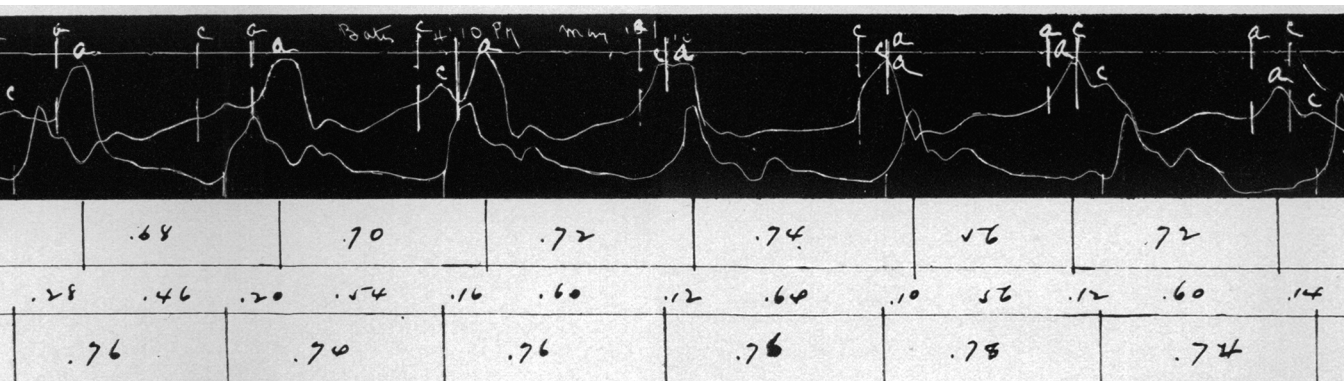


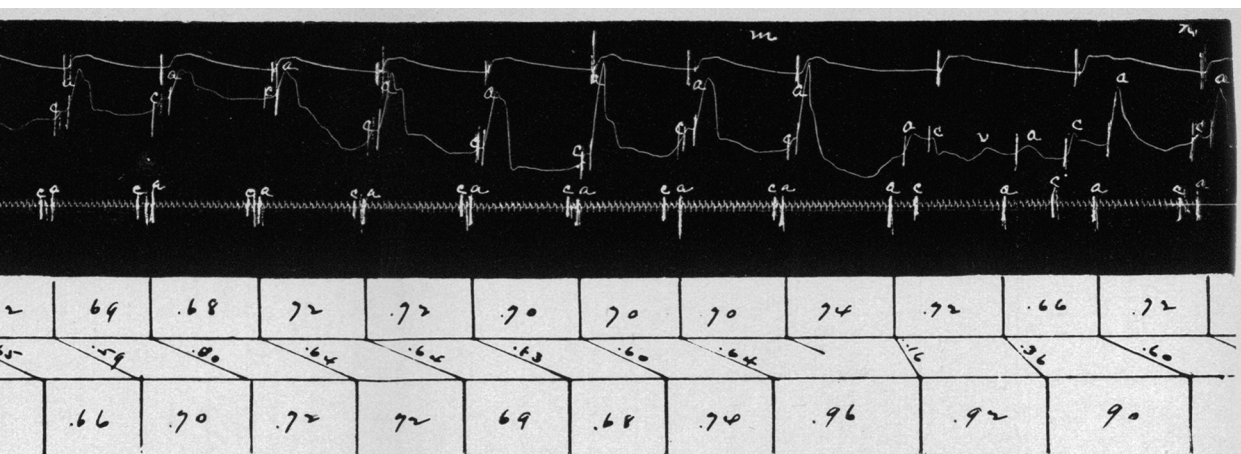
Fig. 2.—An occasional block at the end of 8 mm. of regul  
The auricles



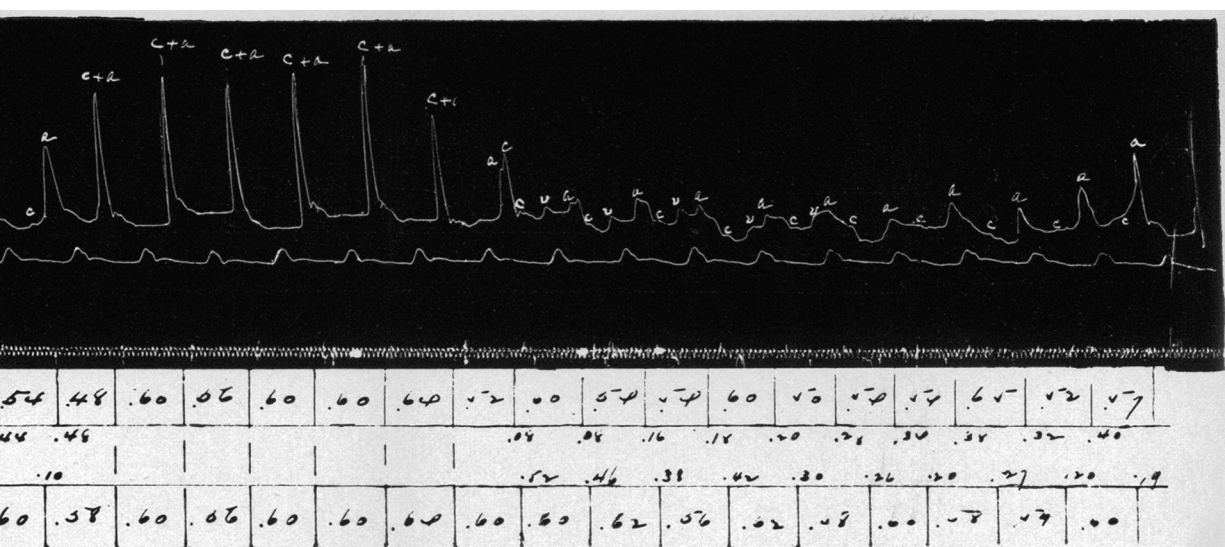
auricular irregular. The *a* wave is decidedly higher whenever it falls within 0.08 second before or 0.19 second after the beginn  
with no attempt to trace the transmissions through the bundle of His.



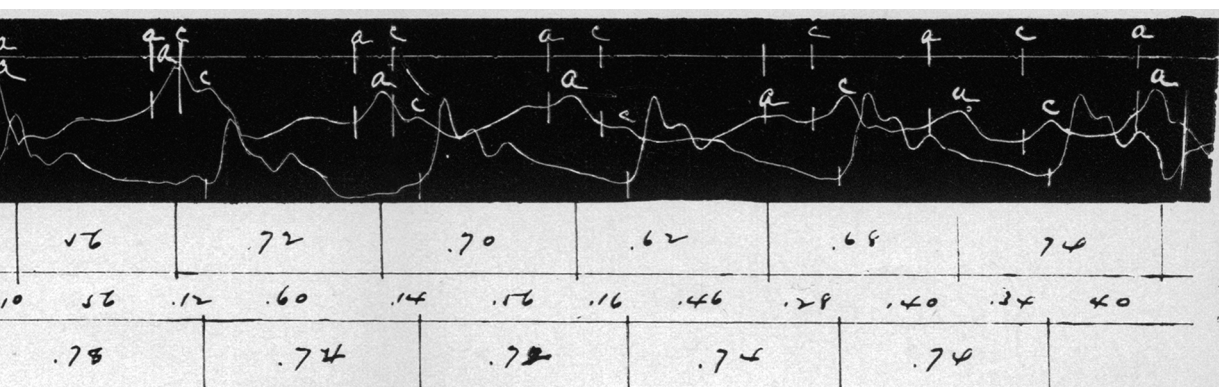
4.—Similar to Figure 3. With the slower rate the values between which summation of *a* and *c* wave occur, are somewhat great



sional block at the end of S mm. of regular rhythm. The diagram assumes a partial block immediately following m. The auricles remain regular throughout.



and before or 0.19 second after the beginning of the c wave. The diagram gives the time relations the bundle of His.



of a and c wave occur, are somewhat greater.

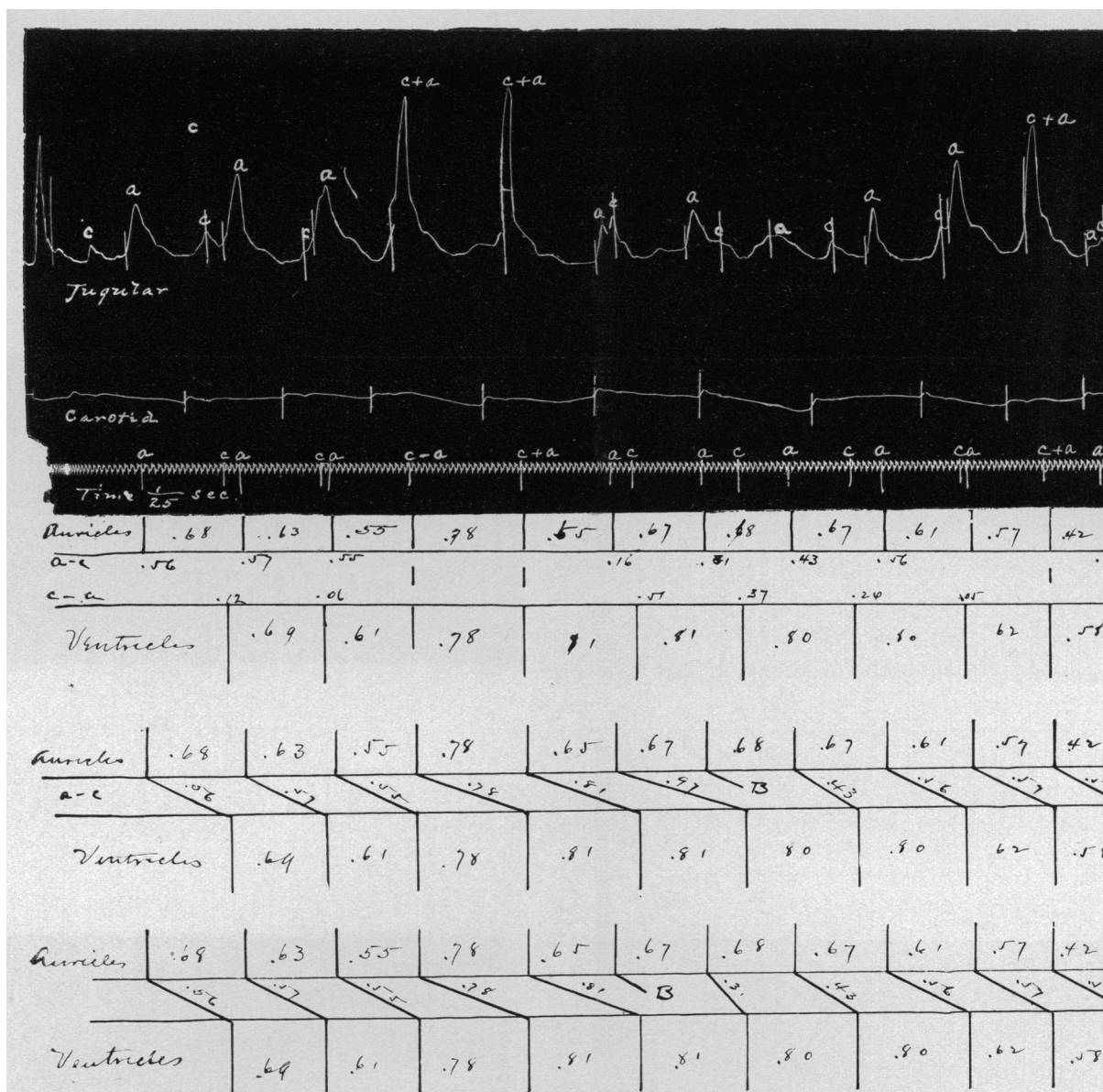
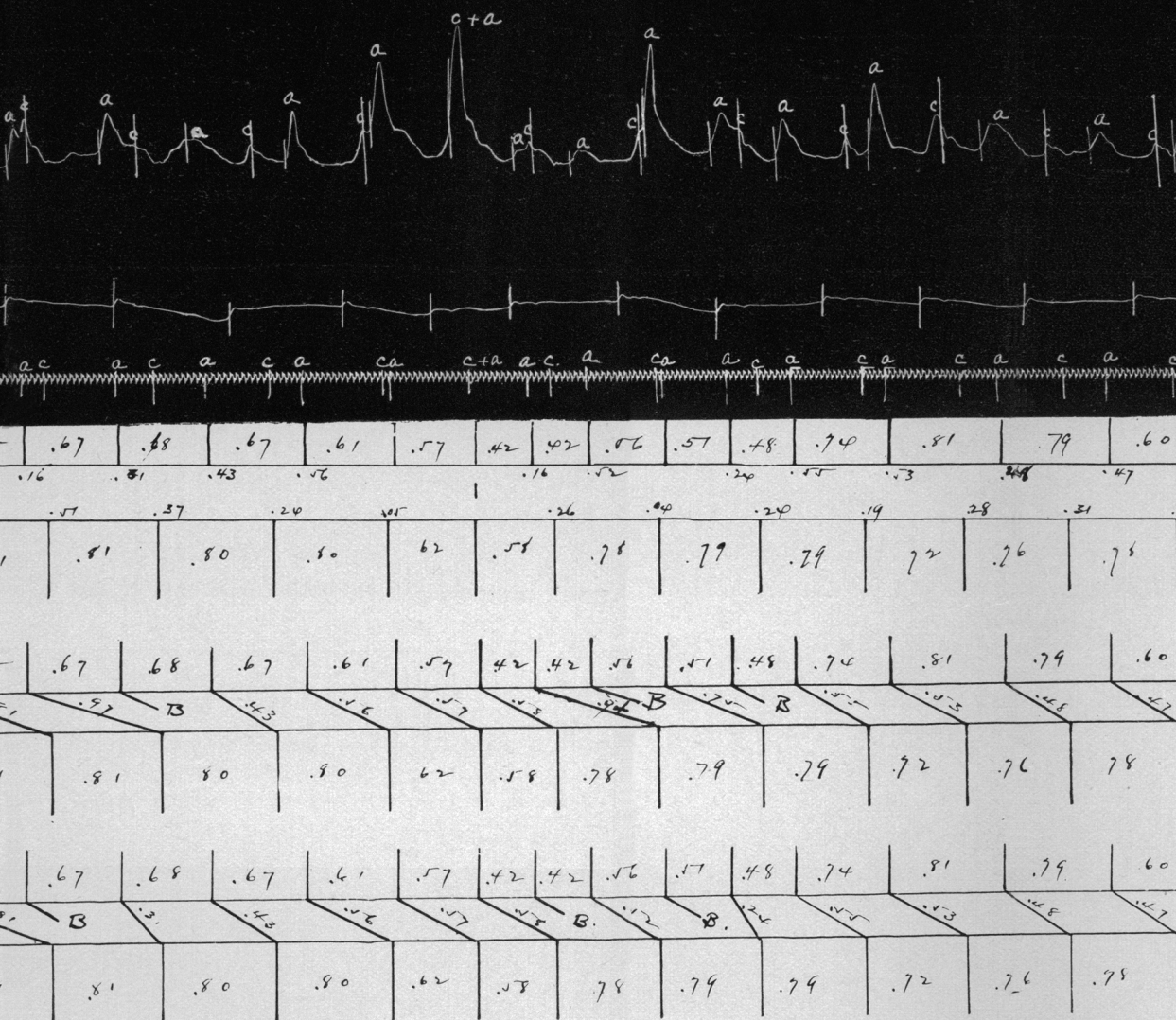
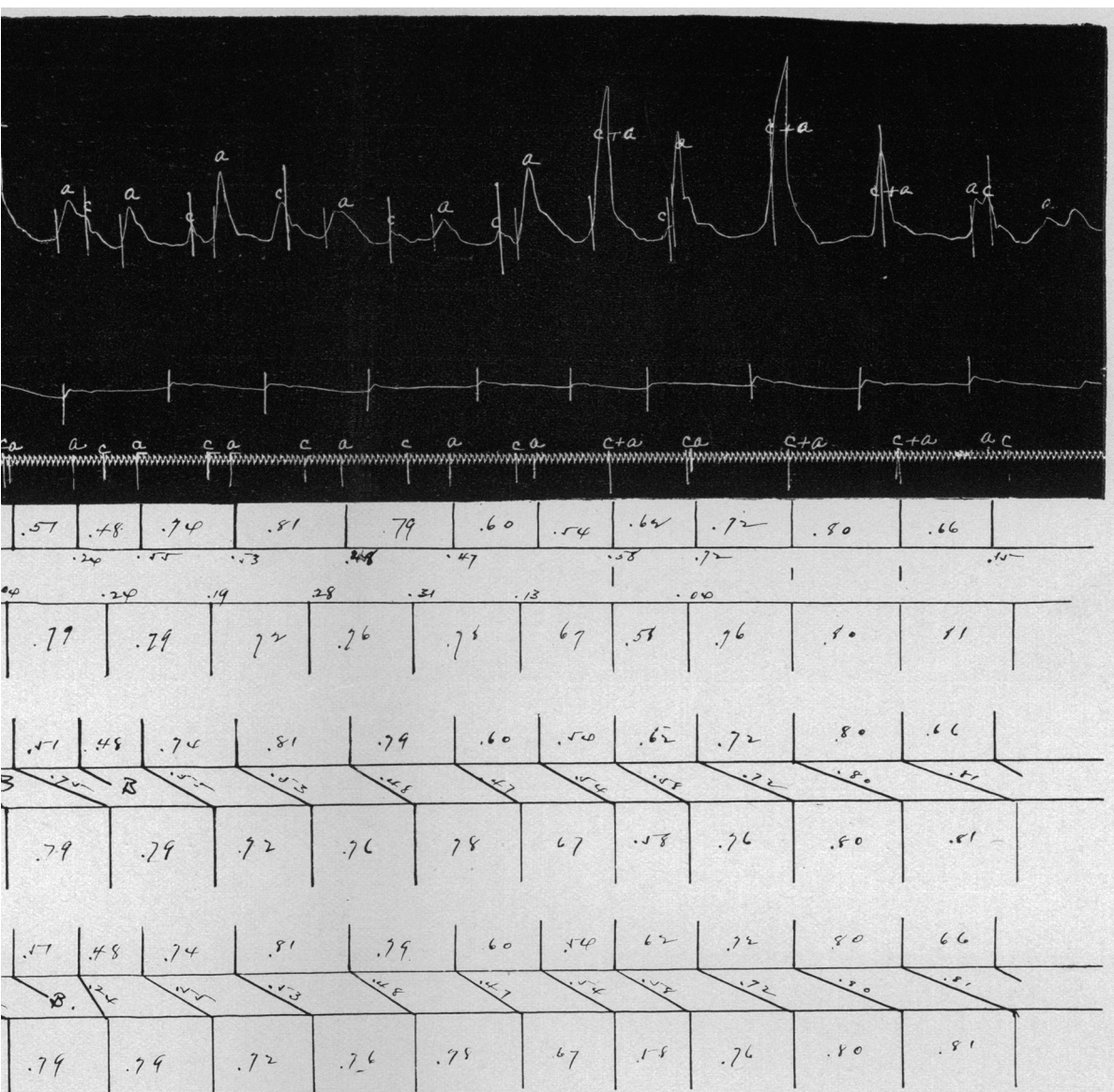


Fig. 5.—Both auricles and ventricles are irregular with fairly close relations between the *a-a* and *c-c*





ular with fairly close relations between the  $a-a$  and  $c-c$  intervals when not complicated by a block. This is best shown in



when not complicated by a block. This is best shown in the later portion of the hypothetical diagrams.

*Examination.*—On entrance the bedside chart showed a temperature with a maximum of 100.4 F., pulse 92, respirations 22. The patient was orthopneic with marked Cheyne-Stokes breathing, cyanosis of the mucous membranes and full throbbing vessels. There were signs of fluid in the left pleural cavity, as high as the third rib in front with numerous râles over the remainder of both lung areas. The cardiac impulse was diffuse and might be seen as high as the manubrium and on the right side. The apex-beat could not be located by sight or touch. A slight diastolic shock was noted in the second left intercostal space. The left border of the heart dulness was lost in the dulness of the fluid. The right border was 6 cm. to right of the median line. Both sounds were distant at the apex and without murmurs. The aortic second was accentuated, also the pulmonic second. The radial was thickened, large, full and incompressible. Systolic blood-pressure was 195 mm. Hg. The liver was enlarged, firm but not tender or pulsating. There was no edema. Deep reflexes were normal. The urine showed fairly large amount of albumin, and microscopic pus. Red blood-cells numbered 4,810,000, white blood-cells 4,450; and hemoglobin was 80 per cent.

*Course of Disease.*—Three days later a diagnostic puncture furnished a light yellow fluid, specific gravity 1.017 with 750 white blood-cells per c.mm., nearly all of which were lymphocytes, 40 per cent. showing badly degenerating nuclei. The pleural fluid gradually decreased under depleting measures so that on the thirteenth day in the hospital the apex could be located in the fifth left intercostal space within the nipple line. The following extract is taken from a note made by Dr. Hewlett on that day: "There is a very unusual variation in the quality of the first sound, most marked at the apex but heard over other parts of the heart. At times it is loud and ringing, at others soft and muffled so that it can hardly be heard. This does not seem to depend on respiratory movements. . . . The heart is quite irregular with no relation between this and respiration. The irregularity is much less noticeable at the wrist and might easily be overlooked."

On the seventeenth day (May 5, 1910) a retention developed calling for catheterization. The urine became scanty and highly albuminous and the patient died in uremia on the twenty-eighth day (May 26, 1910).

Autopsy was refused.

The numerous polygrams from this patient fell into four classes according to the time of rhythm.

Figure 1 represents the most frequent and persistent type and is perfectly regular. It is part of a long record which remained uniform for eight minutes. In this tracing the average *c-c* interval calculated from the carotid pulse measures 0.70 second with a minimum of 0.68 second and a maximum of 0.71 second. The phlebogram shows a positive venous pulse, but the true character of the systolic elevation is not apparent until the irregular period at the end of eight minutes is studied.

This is shown in Figure 2, and represents the second type of arrhythmia, viz., with the ventricles irregular and the auricles regular. The *a* and *c* waves are here easily recognized and from the portion of the tracing immediately preceding and following this irregular stage the relation of the *a* and *c* wave is easily made out. Throughout the records the *c* wave of the phlebogram slightly precedes the systolic rise on the carotid, i. e., the venous *c* wave is "protosystolic" (Bachman<sup>10</sup>). The irregularity cov-

10. Bachman: Interpretation of the Venous Pulse, Am. Jour. Med. Sc., 1908, cxxxvi, 674.

ers the period between the points marked *m* and *n*, including four ventricular and five auricular systoles. It will be noted that there is little change in the *a-a* intervals. The value of the third *a-a* interval is estimated at 0.66 second, although the summation of the *a* and *v* waves at this point makes its determination somewhat problematic. On the other hand the *c-c* intervals have suddenly increased from an average of 0.70 second to 0.96, 0.92 and 0.90 second respectively. These values show a difference which is very near the limit of error and do not exceed the difference between successive values elsewhere in the tracing.

Figure 3 is a reproduction of a tracing taken while the pulse-rate was considerably elevated. It represents the third type of tracing with the auricles irregular and the ventricles regular. The *c-c* interval is regular with an average of about 0.58 second corresponding to a ventricular rate of about 104. The phlebogram shows a striking alternation of high and low waves. The former occur where the period of auricular systole overlaps that of ventricular systole. Where this does not occur the excursion is comparatively slight and the waves well differentiated. In the twenty-three seconds represented in the tracing there are three more auricular than ventricular waves representing an auricular rate of about 113, as against a ventricular of 104. The *a-a* interval varies considerably (from 0.42 to 0.65). The same condition is more convincingly shown in Figure 4, taken on the previous day, when the pulse-rate was less elevated. The average *c-c* interval is 0.75 second, corresponding to a rate of eighty per minute. The average *a-a* interval is 0.68 second, equivalent to ninety per minute. Moreover, the *a-a* intervals vary from 0.52 to 0.78 without any corresponding variation in the *c-c* intervals.

Figure 5 is an example of the fourth type of tracing, in which the auricle and ventricle are both irregular. The patient had received 60 drops of tincture of digitalis during the previous thirty hours but similar tracings were obtained several days previously. Next to the regular type of tracing illustrated in Figure 1, it is the most frequent form. It will be noted that the *c-c* intervals vary from 0.61 to 0.81 second in a rhythmical manner independent of the respiratory rhythm while the *a-a* interval is subject to sudden and irregular changes.

Except in tracings of the first type it is apparent that we are dealing with some form of heart-block. Whether the block is complete or partial is not so evident.

In the light of subsequent tracings, it is easily seen that in Figure 1 we have a regular pulse with an unusually long *a-c* interval as a result of which the *a* wave falls on the *c* wave of the next preceding ventricular contraction. The transition is well marked in Figure 2, and allows of no other interpretation.



Tracings showing a similar incidence of the *a* waves on the *c* waves have been reproduced by Wenckebach,<sup>11</sup> Griffith and Cohn,<sup>2</sup> Thayer and Peabody<sup>12</sup> and others. Wenckebach<sup>13</sup> has applied the name of *Vorhof-Pfropfung* to this condition and finds the cause in the relation of the *a-c* to the *a-a* intervals. Thus it occurs whenever these become approximately equal whether from lengthening of the former in disturbed conductivity or abbreviation of the latter in tachycardia. Similar phenomena occur throughout the tracings from this patient.

It is possible in any case of auricular-ventricular dissociation in which the auricular exceeds the ventricular rate to construct a diagram showing the time relations of auricles and ventricles and by connecting judiciously selected points by slanting lines to assume the block of a number of auricular impulses equal to the difference between the two rates. Such a diagram cannot be taken as evidence of the transmission of any of the auricular stimuli to the ventricle since the relationship may be purely accidental. In a partial block we expect to find (a) the blocked impulse represented by a long interval in the carotid pulse; (b) the *a-c* interval increasing by a progressively smaller increment up to the following block; and (c) the "allorhythmia" of Wenckebach in the radial in which the first *c-c* interval following the block is the longest of the transmitted intervals.

Applying these criteria to the diagram in Figure 2, we find the block represented by a *c-c* interval of 0.96 second, which is within reasonable errors the same as the following intervals; the increase in the *a-c* intervals following are 0.20 and 0.24 respectively (increasing increments), and the second long beat is approximately equal to the first and third. Two interpretations are here possible, viz., the block is partial and the *c-c* intervals are so nearly equal by the accidental increase of the *a-c* interval, just sufficient to produce this, or the block is complete and the ventricles have taken an automatic regular rhythm.

A somewhat similar tracing showing only two long radial pulse periods was reproduced by Joachim<sup>14</sup> and interpreted by the former hypothesis. Estimated from these tracings as reproduced the longest *a-c* interval is about one second while the preceding intervals are not much above the normal. Both Wenckebach<sup>11</sup> and Rihl<sup>6</sup> have questioned this interpretation and published similar instances as showing automatic ventricular contraction.

11. Wenckebach: Beiträge zur Kenntnis der menschlichen Herztätigkeit, Arch. f. Anat. u. Physiol., 1908, Abtlg., Supplement Band 53.

12. Thayer and Peabody: A Study of Two Cases of Adams-Stokes Syndrome with Heart-Block, THE ARCHIVES INT. MED., 1911, vii, 289.

13. Wenckebach: Ueber eine kritische Frequenz des Herzen bei paroxysmaler Tachycardie, Deutsch. Arch. f. klin. Med., 1910, ci, 402.

14. Joachim: Ein atypischer Fall von Störung der Reizleitung in Herzmuskel, Berl. klin. Wechnshr., 1908, xlv, 911.

Gibson and Cohn<sup>2</sup> report a somewhat similar case with alternative diagnosis, selecting as most probable the one assuming an *a-c* interval exceeding one second. Thayer and Peabody's<sup>12</sup> tracings are somewhat similar and are interpreted as due to automatic ventricular contraction.

In the case under consideration the indicated *a-c* interval of 0.90 second is not greater than occurs elsewhere where no other interpretation is possible, while an automatic rate of sixty-six per minute, which is equivalent to the rate of the long periods, is exceptionally high except under toxic doses of digitalis (Cushny<sup>5</sup>). The tracings of Hewlett and Barringer<sup>15</sup> are interpreted as such effects.

The third type of tracings with irregular auricle and apparently regular ventricle is still more involved. In Figure 3, there is absolutely no indication in the carotid of the location of the blocked stimuli. The *a-c* interval fluctuates although tending to increase to an uncertain limit. Where an *a-c* interval is shorter, however, than its predecessor, it is found to correspond to a longer *a-a* interval, possibly because the His bundle has had the longer time to recover. The contrary is also found. It seems improbable that these two factors should be so perfectly balanced as to leave no evidence of the auricular irregularity in the carotid tracing. The difficulty of recognizing the blocked auricular impulse is not removed by a study of the values of the *a-c* intervals since such short intervals as 0.08 second shown in the diagram are quite incompatible with the interval of 0.61 second during the regular periods (see Fig. 1). The inference is that the ventricle in such cases responds to the stimulus of the next preceding beat. But where the series of *a-c* readings is 0.08, 0.08, 0.16, 0.18, 0.20, 0.28, 0.34, 0.38, as occurs in the latter portion of Figure 3, there is no good indication for selecting any stimulus as the one that is blocked. The alternative of a complete block with an independent regular rhythm is very attractive in this connection, except for the unusually high frequency of the automatic ventricular rates of 113 per minute which it implies. With the exception of the case reported by Hewlett and Barringer<sup>15</sup> the rate of sixty-six in a case of complete heart-block reported by Windle<sup>16</sup> is the highest which has come to our notice.

In connection with a tracing showing similar rhythmical variations in the size of the auricular wave, Wenckebach<sup>11</sup> has observed a variation in the intensity and quality of the heart sounds in every way similar to that noted by Dr. Hewlett in the history and ascribes the *Vorhof-Pfropfung* above described as the cause.

In Figure 5 the difficulty of identifying the blocked stimulus is nearly as great. There is, however, an added irregularity of the carotid which reflects more or less perfectly the irregularity of the auricle plus

15. Hewlett and Barringer: Effect of Digitalis on the Ventricular Rate in Man, *THE ARCHIVES INT. MED.*, 1910, v, 1.

16. Windle: Permanent Complete Heart-Block, *Heart*, 1910, ii, 102.

an occasional block. Two alternative diagrams are given as plausible interpretations of this tracing although others are possible. The relationship between the two rhythms is too close to allow of interpretation as complete block—it would be inconceivable that two independent rhythms should show similar fluctuations for any considerable length of time. The long periods of regular rhythm are also incompatible with an independent ventricular rhythm.

## SUMMARY

1. Two cases of partial heart-block due to vagus stimulation or to acute infections or to a combination of these.

2. One case of partial block following the injection of 0.001 gm. of strophanthin.

3. One case of partial block apparently cured by the use of atropin. Two cases in which such injections produced no notable effect on the block.

4. When the block followed strophanthin the auricular rate was increased and when the block disappeared after atropin the auricular rate was lessened.

5. The last case showed partial block with the following peculiarities:

A. Long periods of regular rhythm during which the auricles contracted during ventricular systole. In such cases the ventricles responded to the stimuli coming from the previous auricular contractions, the *a-c* interval being about 0.60 second.

B. Repeated *a-c* intervals of more than 0.80 second.

C. Auricular irregularity with such relations between the *a-c* intervals and the *a-c* increments due to fatigue that the ventricles showed no evidence of the auricular irregularity.

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