The oculocardiac reflex : an electrocardiographic study with special reference to the differences between right and left vagal and ocular pressures in tabetics and non-tabetics / Samuel A. Levine, M.D. Boston.

THE OCULOCARDIAC REFLEX

An Electrocardiographic Study with Special Reference to the Differences between Right and Left Vagal and Ocular Pressures in Tabetics and Non-Tabetics

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THE OCULOCARDIAC REFLEX

AN ELECTROCARDIOGRAPHIC STUDY WITH SPECIAL REFERENCE TO THE DIFFERENCES BETWEEN RIGHT AND LEFT VAGAL AND OCULAR PRESSURES IN TABETICS AND NON-TABETICS *

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INTRODUCTION AND HISTORICAL REVIEW

In 1908 B. Aschner[1] first observed that pressure on the eyeball caused slowing of the pulse and decrease in the depth of respirations. He found that it would arouse stuporous, anesthetized and unconscious patients, and enable them to respond to questions. He also showed that in narcosis the oculocardiac reflex lasts longer than the corneal or pupillary reflexes. Grossmann and Miloslavich[2] in 1912 made similar observations, and Fabre and Petzetakis,[3] working six years later than Aschner, confirmed the observation that the oculocardiac reflex persists even under deep ether or chloroform anesthesia, and can be elicited after the corneal reflex has gone. It was demonstrated by Aschner that the afferent impulse of the reflex passes through the trigeminal nerve to its nucleus in the midbrain, and that the efferent passes by way of the pneumogastric nerve. By cutting the third, fourth, sixth, seventh or eighth cranial nerves he could not destroy the reflex, but by cutting the fifth the reflex was destroyed. He also showed that the inhibition of the heart was not due to increased intracranial pressure, for the relief of pressure by trephining the skull did not abolish the reflex.

Milian,[4] overlooking Aschner's discovery of five years before, stated in 1913 that the phenomenon of pulse-slowing resulting from ocular pressure should be called *"la signe de Gautrelet"* after one of his pupils who had been studying this reflex in his hospital for two years. This

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* From the Hospital of the Rockefeller Institute for Medical Research, New York.

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1. Aschner, B.: Ueber einen bisher noch nicht beschriebenen Reflex vom Auge auf Kreislauf und Atmung, Wien. klin. Wchnschr., 1908, xliv, 1529.

2. Grossmann, J., and Miloslavich, E.: Ueber die Beeinflussung der Herztätigkeit durch Bulbusdruck, Wien. klin. Rundschau, 1912, xii, 177.

3. Fabre and Petzetakis: Persistance du réflex oculo-cardiaque pendant l'anesthésie générale, Compt. rend. Soc. de biol., 1914, lxxvi, 343.

4. Milian: Du ralentissement du pouls radial au cours de la compression oculaire dans la maladie de Basedow, Bull. et mém. Soc. méd. d. hôp. de Paris, 1913, No. 14, p. 878.

would bring back Gautrelet's first observation to three years after Aschner's discovery. Milian found that one could obtain the normal oculocardiac reflex in a dog breathing normally, whether anesthetized or not. But if the thorax were opened and artificial respiration instituted, no slowing of the pulse followed ocular pressure, but instead the tone of the ventricles was increased. This is the experimental evidence for the existence of the reflex and the basis of its mechanism. Many reports dealing with it from a clinical aspect have now been published.

Loeper and Mougeot[5] investigated the condition of the oculocardiac reflex in certain varieties of gastric neurosis, namely, hypervagotonic and the hypersympathicotonic. The former are recognized by the presence of some of the following symptoms: paleness of the face, tendency to myopia, bradycardia, low blood-pressure, moist skin, asthma, gastric hypersecretion, hyperchlorhydria, rapid gastric motility, spasmodic constipation, etc. The latter present the opposite set of symptoms. The former react to pilocarpin with sweating and salivation, but do not react to epinephrin. The latter do not react to pilocarpin, but react to epinephrin with tachycardia, hypertension and glycosuria. They found that the oculocardiac reflex was exaggerated in the hypervagotonic cases and diminished or absent in the hypersympathicotonic. Similar results were obtained in an elaborate investigation by Gautrelet.[6] In several instances they claim to have accelerated the pulse rate by ocular pressure, and they explain this unusual result by stating that the reflex which is generally conveyed by the vagus at times passes over the sympathetic nerve. They consider that an individual whose pulse rate is accelerated or slowed more than ten beats per minute by pressure is abnormal, in the sense that he belongs to one or the other of the two groups named. This is a point to which reference will be made again later.

Lesieur, Vernet, and Petzetakis[7, 8, 9] have studied the condition of the oculocardiac reflex in epileptics. This reflex, as well as tendon and

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5. Loeper, M., and Mougeot, A.: Le réflexe oculo-cardiaque dans le diagnostic des nevroses gastriques, Bull. et mém. Soc. méd. d. hôp. de Paris, 1913 No. 14, p. 865.

6. Gautrelet, J.: Le réflexe oculo-cardiaque, Paris méd., Nov. 29, 1913, p. 583.

7. Lesieur, Ch.: Vernet, M., and Petzetakis: Sur un cas d'arrêt total du coeur par réflexe oculo-cardiaque chez un épileptique, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 9, p. 394.

8. Lesuier, Ch., Vernet, M., and Petzetakis: Contribution à l'étude du réflexe oculo-cardiaque. Son exagération dans l'épilepsie, les variations sous l'influence d'actions médicamenteuses ou toxiques, Bull. et. mém. Soc. méd. d. hôp. de Paris, 1914, No. 9, p. 440.

9. Lesieur, Ch., Vernet, M., and Petzetakis: Considerations sur les modifications des refléxes pendant la compression oculaire chez certains épileptiques, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 11, p. 510.

cutaneous reflexes, was found to be exaggerated. In their experience it was found that bromid treatment not only lessened the frequency of epileptic attacks, but also diminished the oculocardiac reflex by decreasing vagal irritability. In the same study they found that the right eye responded more strikingly and on slighter pressure than did the left. Usually, when atropin was injected subcutaneously, it abolished the oculocardiac reflex, but ocular pressure continued to inhibit the respiratory motions of the patient just as it did when no atropin was given. This they called the oculophrenic reflex. Later, the same authors[10] found that ocular pressure caused glycosuria in three of their six cases of epilepsy. Four showed albuminuria and all had polyuria. This reflex, they presumed traveled over the sympathetic nerve and not the vagus, because in Claude Bernard's experiments in which he punctured the floor of the fourth ventricle, he observed glycosuria in animals when the vagi were divided, but not when all the sympathetic fibers were cut.

Guillain and Dubois,[11] in a case of double athetosis, noticed that the athetoid movements of the face, tongue and extremities ceased during ocular pressure, but increased during any other medical manipulation. Obstinate yawning and hiccough were observed by Loeper and Mlle. Weil[12] to be influenced favorably by ocular pressure. In six cases of pseudobulbar paralysis, Guillain and Dubois[13] found that four had lost the oculocardiac reflex, and that the other two responded by an acceleration of the pulse rate. The condition of the oculocardiac reflex in various tremors has been studied by Lesieur, Vernet and Petzetakis.[14] Of sixteen cases of Parkinson's disease the reflex was absent in fifteen. Of four cases of multiple sclerosis the reflex was absent in one, accelerated in two and normal in one. The reflex was normal in two cases of alcoholic tremor and in two cases of senile tremor. Of six cases of general paresis the reflex was exaggerated in four and normal

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10. Lesieur, Ch., Vernet, M., and Petzetakis: Glycosurie, albuminurie, polyurie provoquées par la compression oculaire, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 11, p. 515.

11. Guillain, G., and Dubois, J.: Action inhibitrice de la compression oculaire sur les mouvements anormaux dans un cas d'athetose double, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 16, p. 850.

12. Loeper, M., and Mlle. Weil: Action favorable de la compression oculaire sur certaines manifestations nerveuses et en particulier sur le hoquet, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 13, p. 631.

13. Guillain, G., and Dubois, J.: L'abolition et l'inversion du réflex oculo-cardiaque dans les paralysies pseudo-bulbaires, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 12, p. 584.

14. Lesieur, Ch.: Vernet, M., and Petzetakis: Le réflexe oculo-cardiaque chez les sujets atteints de divers tremblements, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 12, p. 593; Réflexe oculo-cardiaque et maladie de Parkinson, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 14, p. 599.

in two. They concluded that the oculocardiac reflex might aid in determining whether a nervous lesion was central or peripheral. It does not seem clear, however, how the differentiation is to be made, for a reflex may be either exaggerated or destroyed by a lesion of the afferent limb, center, or efferent limb of a reflex arc. The condition alone of a reflex will scarcely determine the site of a lesion.

In the study of the reflex in organic diseases of the central nervous system, Miloslavich[15] in 1910 was the first to observe that the reflex was absent in tabes dorsalis. Loeper and Mougeot,[16, 17] and Lesieur, Vernet and Petzetakis[18] have shown that in a majority of such patients the pulse rate is either not slowed at all by ocular pressure, or only slightly affected. Lesieur, Vernet and Petzetakis found the reflex to be absent when the Argyll Robertson phenomenon had not yet developed. In syphilis of the nervous system, other than tabes dorsalis, Loeper, Mougeot and Vahram[19] found that the reflex was also absent in thirty of their forty cases.[20] None had Argyll Robertson pupils. These patients were generally in the tertiary stage. The reflex, then, is lost both in tabes and in other syphilitic diseases of the nervous system. But there need be no necessary relation between its loss and the presence or absence of the Argyll Robertson pupil. Different results were obtained in this study concerning the condition of the reflex in syphilitic patients who are not tabetic. Further reference will be made to this later.

The condition of the reflex in Basedow's disease has aroused a great deal of interest. Grossmann and Miloslavich,[2] Milian,[4] Sainton,[21] and Lesieur, Vernet and Petzetakis[14] have shown that it is generally exaggerated in this condition. They attribute this to the hypervagotonicity present in these patients.

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15. Miloslavich, E.: Ueber Trigeminus-vagus Reflex, Wien. med. Wchnschr., 1910, lx, 3051.
16. Loeper, M., and Mougeot, A.: L'absence du réflexe oculo-cardiac dans le tabes, Progrès méd., 1913, No. 52, p. 675.
17. Loeper, M., and Mougeot, A.: Absence fréquente du réflexe oculo-cardiaque dans le tabes, Bull. et mém. Soc. méd. d. hôp. de Paris, 1913, No. 39, p. 942.
18. Lesieur, Ch.: Vernet, M., and Petzetakis: Note sur l'abolition fréquente du réflexe oculo-cardiaque dans le tabes, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 9, 446.
19. Loeper, M., Mougeot, A., and Vahram: Abolition fréquente du réflexe oculocardiaque chez les syphilitiques, Progrès méd., 1914, No. 14, p. 157.
20. The authors give no reason why the cases should be considered syphilis of the nervous system and not merely tertiary syphilis. They conclude, however, that the frequent absence of the oculocardiac reflex is evidence that the nervous system is early involved.
21. Sainton: Le réflexe oculo-cardiaque dans la syndrome de Basedow, Bull. méd., 1913, No. 60, p. 701.

The condition of the reflex in tachycardias and bradycardias has been studied by Mougeot[22] and Loeper and Mougeot.[23] They believe, in general, that in tachycardias and in bradycardias of nervous origin, the reflex is preserved, while in those of myocardial origin, it is lost. They suggest this difference as a method for distinguishing the two. Fabre and Petzetakis[24] observed that in the bradycardias occurring during the puerperium, ocular pressure caused auriculoventricular dissociation. Dufour and Legras[25] reported the case of a young woman who, having had a miscarriage a few months before, suddenly developed hypo-ovarian symptoms; that is to say, amenorrhea and loss of hair. Later she developed hyperthyroidism, indicated by the presence of exophthalmos and tachycardia. Epileptiform attacks, due, it is said, to cerebrospinal hypertension, supervened. During this time ocular pressure caused no slowing of the heart. Later, when the hair began to return, the reflex reappeared, so that during ocular pressure complete auriculoventricular dissociation took place. No mention was made as to which eye caused the dissociation. The authors do not clearly state whether the reflex was absent before hyperthyroidism developed, during it, or after the symptoms had quieted down. It does not seem likely that the occurrence of hyperthyroidism should make the reflex disappear. Its absence was more probably due to the hyposecretion of the ovaries; for the reflex reappeared when the ovaries functioned normally.

The influence of drugs on the condition of the reflex has been studied by Petzetakis[26] and Mougeot.[27] They found that atropin

22. Mougeot, A.: Tachycardie paradoxale des hypertendus et réflexe oculo-cardiaque, Progrès méd., 1913, No. 51, p. 663; Le réflexe oculo-cardiaque dans les tachycardies permanentes sans arythmie, Compt. rend. Soc. de Biol., 1914, No. 5, p. 205.

23. Loeper, M., and Mougeot, A.: Le réflexe oculo-cardiaque dans le diagnostic de la nature des bradycardies, Compt. rend. Soc. de biol., 1914, No. 3, p. 104.

24. Fabre and Petzetakis: De la bradycardie et automatisme ventriculaire provoqué dans les suites de couches par la compression oculaire, Réunion Obstétrical et Gynecologique de Lyon, January 19, 1914, p. 37.

25. Dufour and Legras: Réflexe oculo-cardiaque provoquant l'arrêt du coeur. l'automatisme ventriculaire et la dissociation auriculo-ventriculaire, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 14, p. 686.

26. Petzetakis: L'épreuve de l'atropine, du nitrite d'amyl et de la compression oculaire dans les bradycardies totales, Compt. ren. Soc. de biol., 1913, lxxv, p. 677; De l'automatisme ventriculaire provoqué par la compression oculaire et l'atropine dans les bradycardies totales, Compt. rend. Soc. de Biol., 1914, No. 1, p. 15; L'épreuve de la compression oculaire du nitrite d'amyl et de l'atropine dans le diagnostic des bradycardies totales d'origin nerveuse, Presse méd., 1914, No. 17, p. 161; Abolition du réflexe oculo-cardiaque par l'atropine; son exagération par la pilocarpine; sa persistance pendant l'épreuve du nitrite d'amyl, Compt. rend. Soc. de biol., 1914, No. 6, p. 247; Le réflexe oculo-cardiaque chez les sujets normaux non bradycardiques, Bull. et mém. Soc. d. hôp. de Paris, 1914, No. 12, p. 562.

27. Mougeot, A.: Supression constante par l'atropine de réflexe oculo-cardiaque, Compt. rend. Soc. de biol., 1914, No. 4, p. 162.

0.02 mg.[28] injected subcutaneously abolished the reflex for a period of one to three hours. In a small portion of cases atropin exaggerated the reflex or excited automatic ventricular beats. These are the cases in which atropin retards rather than accelerates the heart rate. Pilocarpin, 0.01 gm. generally exaggerated the reflex. When amyl nitrite was given ocular pressure slowed the accelerated heart, but inhibition began after a longer latent period than occurs normally.

Various studies of the effect of the ocular pressure on the mechanism of the heart itself have been made. Mougeot[29] found that the reflex was generally not abolished during alternation of the ventricle. In some cases of uremia and other intoxications in which ventricular alternation was present, the reflex was lost. Its reappearance was taken to indicate amelioration in the underlying condition. It has been suggested that the loss of the reflex depends on an intoxication of the vagus nuclei in the medulla, by means of which the path of the reflex is cut. In ten of seventy-five cases, Petzetakis[26, 30] obtained transient dissociation of the auricles and ventricles by ocular pressure. The same arhythmia was also observed by Gallavardin, Dufour and Petzetakis.[31] No mention was made of any difference between the effects produced by pressure on the right and left eyes. A little later Petzetakis[32] was able to conclude from a study of two cases that pressure on the left eye is more apt to cause disturbance in auriculoventricular conduction, while pressure on the right eye has a greater influence on rate. His explanation for the difference in effect between the two is that the right vagus is more sensitive than the left. He found two patients in whom ocular pressure caused partial heart-block and later complete auriculoventricular dissociation. The arhythmia was prevented from appearing in these cases by the injection of atropin. Petzetakis[33] reported one case in which atropin had the reverse effect. In this case the injection *slowed the pulse* and permitted the appearance

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28. This seems to be a small dose.

29. Mougeot, A.: Le réflexe oculo-cardiaque dans l'alternance ventriculaire, Compt. rend. Soc. de biol., 1914, No. 12, p. 541.

30. Petzetakis: Réflexe oculo-cardiaque et dissociation auriculo-ventriculaire, Compt. rend. Soc. de biol., 1914, No. 10, p. 409; Automatisme ventriculaire intermittent provoqué à l'état normal, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 14, p. 727.

31. Gallavardin, Dufour and Petzetakis: Automatisme ventriculaire intermittent, spontané ou provoqué par la compression oculaire et l'injection d'atropine dans les bradycardies totales, Arch. de mal. du coeur, 1914, vii, 1.

32. Petzetakis: Block auriculo-ventriculaire provoqué par la compression oculaire, Bull. et mém. Soc. méd. d. hôp. de Paris, 1914, No. 14, p. 739.

33. Petzetakis: L'épreuve paradoxale de l'atropine. Son action ralentissante sur le rhythme cardiaque, Bull. et mém. Soc. méd. hôp. de Paris, 1914, No. 12, 567.

of the reflex, which had not been present before. In most instances diagnoses of the disorders of the heart mechanism were made by means of polygraphic tracings. This method does not give conclusive information on some points of cardiac arhythmias, and when the tracings are made from patients disturbed by manipulation, they involve some doubt.

Pressure over the vagus nerves in the human subject has been studied particularly by Robinson and Draper.[34] They found that vagal pressure caused slowing of the auricles and ventricles and depressed conductivity in cases having normal hearts. The *Q-B* interval, i. e., the time between the *Q* wave in the electrocardiogram and the footpoint of the brachial pulse, was sometimes diminished. The right vagus had a greater effect on the force and rate of the heart, while the left vagus had a more pronounced effect on the conduction of impulses from auricles to ventricles. The heart was found to respond more quickly to right than to left vagal pressure. Right vagal pressure caused an increase in the action current of the ventricles and a decrease of the current of the auricles, i. e., the height of the *R* waves was increased and the height of the *P* waves was diminished. Left vagal pressure diminished the height of the *R* waves, while the *P* waves were unaffected. In cases of auricular fibrillation, vagal pressure slowed the ventricles without affecting the auricles. Reference will later be made to these results.

METHOD

In this investigation, patients were placed in the galvanometer circuit, and electrocardiograms were made to serve as controls. An assistant at the bedside, in continuous telephonic communication with the operator at the galvanometer, exerted pressure on the neck, or on the eye, as the case might be. Enough pressure was exerted on the neck entirely to obliterate the carotid pulse. No accurate measurement of the degree of pressure exerted on the eye was possible. An attempt was made later to determine the amount of pressure, in millimeters of mercury, used on the eyes in order to obtain a standard for comparison with other observers. As nearly as could be judged, a similar degree of pressure was applied to the distended air-tight bag of a blood-pressure apparatus. By this method the pressure employed was found to be approximately 30 mm. of mercury. The eyelid was first closed and the

34. Robinson, G. C., and Draper, G.: Action of Vagus Nerve on Human Heart, Jour. Exper. Med., 1911, xiv, 217; Studies with the Electrocardiogram on the Action of the Vagus Nerve on the Human Heart, Jour. Exper. Med., 1912, xv, 14.

operator's thumb applied under the supraorbital ridge, but not directly over the cornea. The pressure on the two eyes was made as nearly equal as possible. Before pressure was made a strip of curve of seven or eight seconds duration was taken. A signal registered the onset of pressure on the tracings. Pressure continued for about six or seven seconds, the offset being marked by the second signal in the tracing. The exposure was continued about eight or ten seconds longer to include the return of the heart's action to normal. In most cases the second lead was taken (right arm to left leg), but in some instances the first lead (right arm to left arm) was tried, to obtain, if possible, more prominent auricular waves.

Pressure was usually made first on the right vagus, then on the left vagus, then on the right eye and last on the left eye. Two records of each pressure (eight in all) were obtained. Four minutes were permitted to elapse between any two pressures, in order to exclude the summed influence of one on the succeeding. Occasionally, the left vagus was pressed on first and sometimes the left or right eye, to be certain to exclude confusion occasioned by fatigue phenomena. The same patient was in a few instances examined on two different days, and the order of pressure was reversed. Different degrees of pressure were used in the same patient at different times in order to determine whether the amount of slowing varied with the intensity of the pressure. Finally, the symptoms of the patient during pressure were recorded. The signs found included flushing of the face, inhibition of respirations, expressions of pain and the movements of deglutition, etc.

PROTOCOLS[35]

Case 1.—Schoolboy, aged 9 years. He had a rheumatic history. Pupils react normally.

Clinical Diagnosis: Chorea, rheumatic fever, chronic valvular disease.

Case 2.—Schoolboy, 11 years old. Pupils react normally.

Clinical Diagnosis: Chronic endocarditis.

Case 3.—Clerk, 49 years old. No rheumatic history. Pupils react normally.

Clinical Diagnosis: Rheumatic fever, myocardial insufficiency, auricular fibrillation.

Case 4.—Printer, 49 years old. No rheumatic history. Pupils equal, oval, and react sluggishly to light. Reaction to accommodation is greater and more rapid.

Clinical Diagnosis: Tabes dorsalis.

Case 5.—Business man, 38 years old. Pupils are equal and react sluggishly to light. Both react well in accommodation.

Clinical Diagnosis: Syphilitic aoritis and syphilis of the spinal cord (syphilitic myelitis).

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35. The protocols have been compiled to report the sex, age, occupation, previous diseases having a possible influence on the cardiovascular system, the state of the pupils at the time of examination, and the clinical diagnosis.

CASE 6.—Male, 49 years old. Instructor in physical culture. Pupils are equal, small, do not react to light, prompt reaction in accommodation.

Clinical Diagnosis: Tabes dorsalis.

CASE 7.—Coachman, 45 years old. Pupils unequal, right is 2 mm., left is 5.5 mm. in diameter. Neither reacts to light and both have sluggish reaction in accommodation.

Clinical Diagnosis: Syphilis of the brain and spinal cord (probably early paresis or taboparesis).

CASE 8.—Salesman, 46 years old. Pupils unequal, right slightly larger than left. Both irregular. React slightly but sharply to light and normally in accommodation.

Clinical Diagnosis: Tabes dorsalis.

CASE 9.—Blacksmith, 58 years old. Rheumatic fever four years ago. Hard chancre thirty years ago. Pupils react normally.

Clinical Diagnosis: Acute rheumatic fever, acute pericarditis, cardiac arhythmia, transitory auricular fibrillation, serofibrinous pleurisy and latent syphilis.

CASE 10.—Laborer, 31 years old. Rheumatic fever seven years ago, and two attacks since. Pupils react normally.

Clinical Diagnosis: Chronic valvular disease.

CASE 11.—School girl, 11 years old. Had one attack of rheumatic fever. Pupils react normally.

Clinical Diagnosis: Chronic mitral regurgitation. Chronic cardiac hypertrophy and dilatation.

CASE 12.—School girl, 9 years old. Rheumatic fever five years ago. Pupils react normally.

Clinical Diagnosis: Acute rheumatic fever, chronic valvular disease, mitral regurgitation, aortic regurgitation and aortic stenosis.

CASE 13.—School boy, 12 years old. Pupils react normally.

Clinical Diagnosis: Chronic valvular disease, mitral regurgitation and stenosis, chronic cardiac hypertrophy and dilatation.

CASE 14.[36]— Tailor, 30 years old. Right pupil larger than left. Reaction to light and accommodation is active in both eyes.

Clinical Diagnosis: Secondary syphilis, syphilitic periostitis of the frontal bone, periostitis of ulna; multiple, gonorrheal, periurethral abscesses.

CASE 15.—School boy, 14 years old. Rheumatic fever. Pupils react normally.

Clinical Diagnosis: Acute rheumatic fever and acute endocarditis.

CASE 16.—School girl, 11 years old. Chorea and rheumatic fever. Pupils react normally.

Clinical Diagnosis: Lobar pneumonia.

CASE 17.—Male, 9 years old. School boy. No history of rheumatic fever. Pupils react normally.

Clinical Diagnosis: Lobar pneumonia and empyema.

CASE 18.—Cloakmaker, 40 years old. Pupils slightly dilated and unequal. Left is 4.5 mm., right is 4 mm. in diameter. Both irregular and react in accommodation but not to light.

Clinical Diagnosis: Tabes dorsalis.

CASE 19.—Bookkeeper, 63 years old. No history of rheumatic fever. Pupils equal and react in accommodation but not to light.

Clinical Diagnosis: Tabes dorsalis.

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36. Case 14 has been omitted because he began to show signs of early tabes, although the oculocardiac reflex was present.

CASE 20.—Electrician, 32 years old. Pupils unequal. Right is twice the size of the left. Both react slightly to light and well in accommodation.
Clinical Diagnosis: Tabes dorsalis.

CASE 21.—Business man, 26 years old. Rheumatic history eight years ago. Pupils slightly irregular in outline but react normally.
Clinical Diagnosis: Secondary syphilis. Syphilis of cerebrospinal meninges.

CASE 22.—Girl, 17 years old. Machine operator. Pupils react normally.
Clinical Diagnosis: Diabetes mellitus.

CASE 23.—Man, 53 years old. Actor. Pupils are unequal; right 3 mm., left 2.25 mm. in diameter. Neither reacts to light, but both react in accommodation.
Clinical Diagnosis: Tabes dorsalis.

CASE 24.—Reporter, 34 years old. Pupils are equal, irregular in outline, react normally to light and in accommodation.
Clinical Diagnosis: Secondary syphilis, syphilis of the cerebrospinal meninges, syphilis of the auditory and facial nerves.

CASE 25.—Tailor, 53 years old. Pupils equal, slightly irregular, do not react to light; both react slightly in accommodation.
Clinical Diagnosis: Tabes dorsalis.

CASE 26.—Porter, 35 years old. Pupils react normally.
Clinical Diagnosis: Lobar pneumonia and lung abscesses.

CASE 27.—Boy, 18 years old. Factory hand. Pupils react normally.
Clinical Diagnosis: Lobar pneumonia.

CASE 28.—Salesman, 24 years old. Pupils react normally.
Clinical Diagnosis: Lobar pneumonia.

CASE 29.—Diamond cutter, 60 years old. Had syphilis thirty-eight years ago. Pupils are somewhat dilated. Left does not react to light as completely and quickly as right. Both react well in accommodation.
Clinical Diagnosis: Transient heart-block. Syphilis of the heart.

CASE 30.—Farm hand, 26 years old. Pupils react normally.
Clinical Diagnosis: Acute rheumatic fever, acute myocarditis, lobar pneumonia.

GENERAL RESULTS AND OBSERVATIONS

The material for this study consisted of eight cases of tabes dorsalis, one case of taboparesis, five cases of syphilis which were non-tabetic, nine cases of chronic endocarditis (having a normal rhythm), one case of chronic endocarditis with auricular fibrillation, five cases of lobar pneumonia, and one case of diabetes mellitus. The five non-tabetic syphilitics included two cases of syphilis of the cerebrospinal meninges, one of syphilitic myelitis, one of syphilis of the heart, and one patient who has been observed during the secondary stage and who now suggests signs of early tabes.

The results of ocular and vagus pressure from four (Cases 4, 18, 20 and 25) of the eight cases of *tabes dorsalis* are shown in detail in figures 1a-d. Figure 1a gives the results of right vagus pressure, 1b of right ocular pressure, 1c of left vagus pressure and 1d of left ocular pressure. In one instance (Figure 1c, Tracing 157) there is marked slowing on vagus pressure. The degree of inhibition of the heart produced by vagus pressure was greater in this case than in any of the

other cases (tabetic or non-tabetic.)[37] This is a significant point, because although the oculocardiac reflex will be shown to be generally absent in tabetics and present in non-tabetics, the greatest degree of slowing by direct vagal pressure was obtained in a tabetic. The centrifugal path of the reflex was intact. One (Case 6) of the tabetics, whose pupils were rigid and did not react to light, developed a moderate amount of slowing as a result both of right and left ocular pressure. In some of the other tabetics, where the oculocardiac reflex was absent, the pupils reacted slightly to light. All the other tabetics gave no slowing on ocular pressure. In eight cases of tabes dorsalis, then, one showed a moderate oculocardiac reflex whereas it was absent in the other seven. There is, also, no exact parallelism between the Argyll Robertson reaction and the oculocardiac reflex. There was no difference in the effect produced by pressure on the two eyes. The only change noticed in the electrocardiograms was a slight increase in the prominence of the *P*-waves in two instances on right vagal pressure. There was no marked change in the conduction time or in the form of the ventricular complex.

In the second group of cases are included the four *syphilitics*[38] who were *not tabetic*. (Figs. 2a-d.) In three of the four cases, the oculocardiac reflex was preserved. The results obtained here differ, therefore, from those obtained by Loeper, Mougeot and Vahram.[19] They found that in 75 per cent. of their cases of syphilis the oculocardiac reflex was *absent*. The reflex was *present* in 75 per cent. of the cases in this study. In two (Cases 21 and 24) of the four cases (Fig. 2b, Tracings 166 and 187, and Fig. 2d, Tracings 167 and 183) there was complete inhibition of the heart for periods ranging from 3.31 seconds to 10.92 seconds (Fig. 5). In a third case (No. 29) there was a moderate amount of slowing, that is to say, a slight reaction. The fourth (Case 5) did not react at all to ocular pressure. This case was one of syphilis of the spinal cord and syphilitic aortitis. He showed an Argyll Robertson pupil, so that it is not at all unlikely that he had early tabes. The slowing in these cases was more pronounced during pressure on the right ocular bulb than on the left. There were no changes in the electrocardiograms. In Case 21 an escaped ventricular beat occurred 0.79 second after the preceding pause of 3.67 seconds. Another long pause of 2.40 seconds followed. The escaped ventricular beat was of normal form, probably supraventricular in origin. In this case all the waves

37. The term non-tabetic is used throughout to include all the cases in the series except those of tabes dorsalis.

38. Case 14, originally included here, has been omitted because he began to show signs which may be those of early tabes, although the oculocardiac reflex was present.

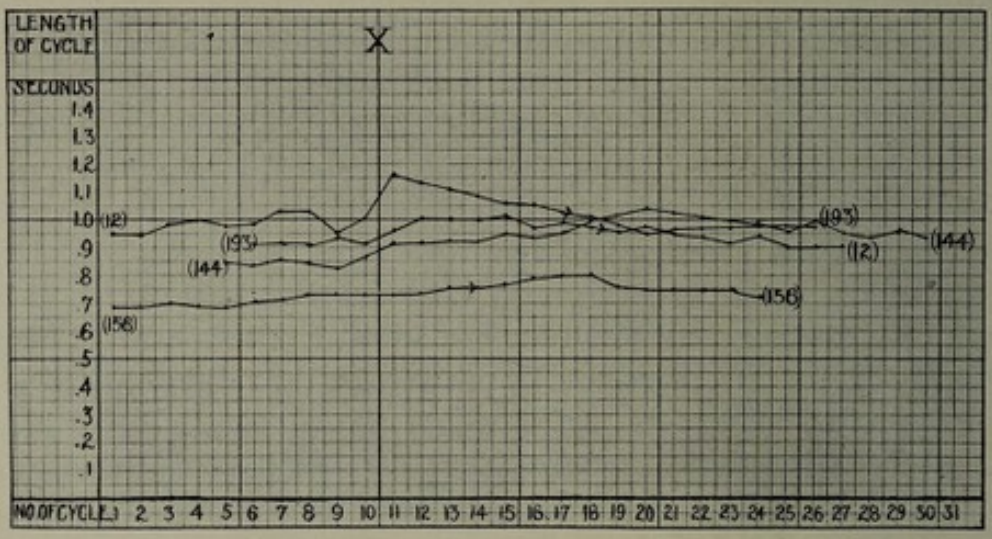

Figure 1a

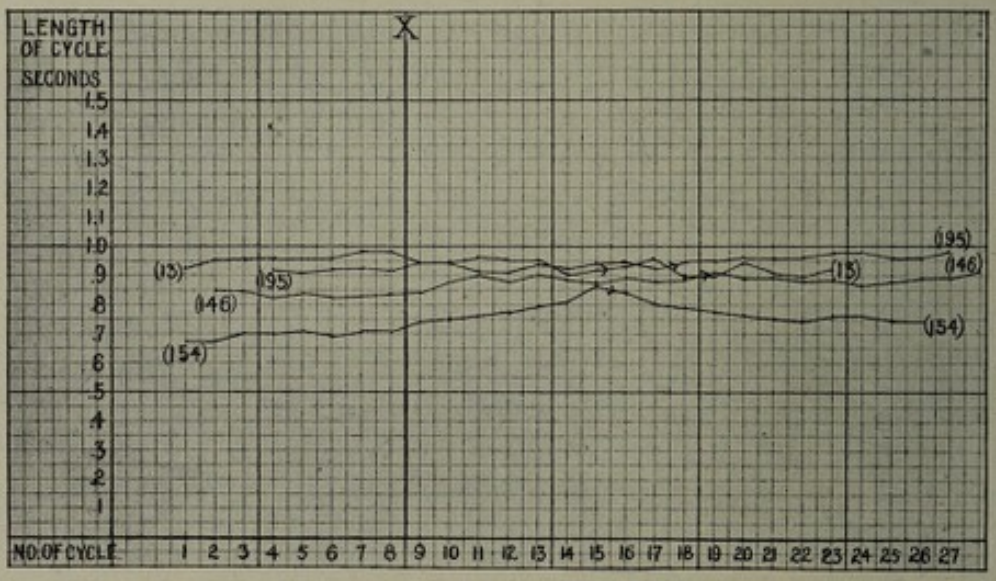

Figure 1b

Figures 1, 2, 3 and 4 detail the duration (on the ordinate) of every cardiac cycle (on the abscissa) calculated from electrocardiograms taken during a given digital (vagal or ocular) pressure. X indicates the onset of pressure; the arrow (→) the time of the offset. The figures marked *a* show the results

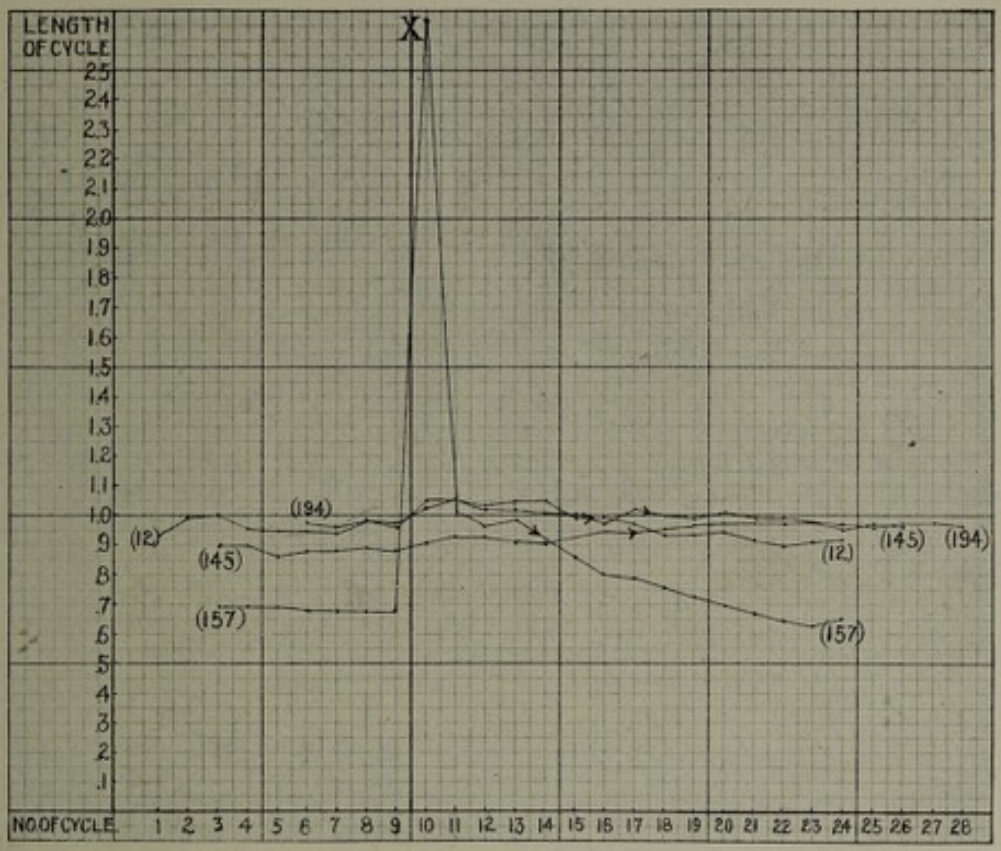

Figure 1c

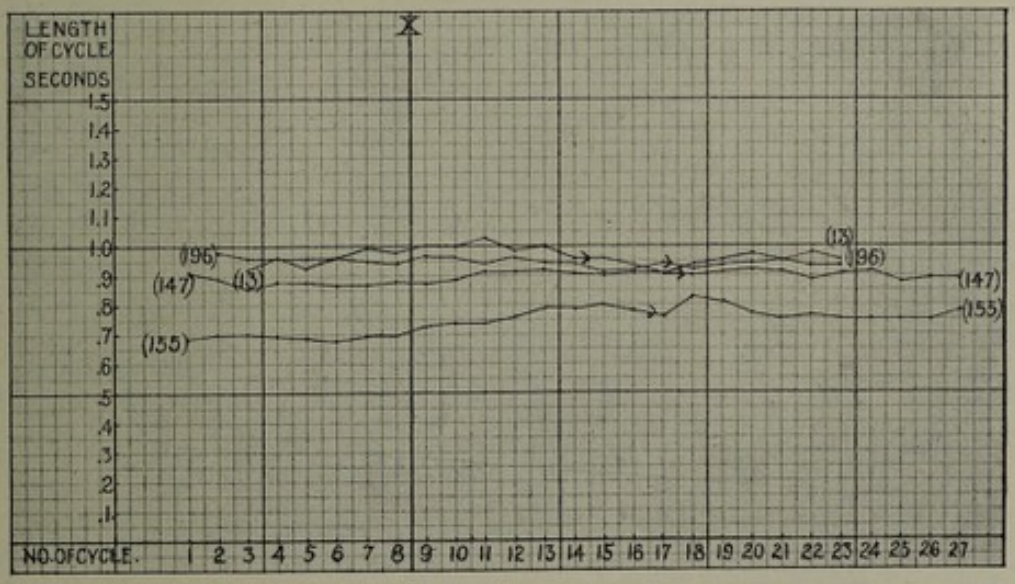

Figure 1d

of right vagus pressure: *b*, right ocular pressure; *c*, left vagus pressure; *d*, left ocular pressure. The numbers at the beginnings and ends of the curves indicate, in the accompanying table, the patients to whom the records refer.

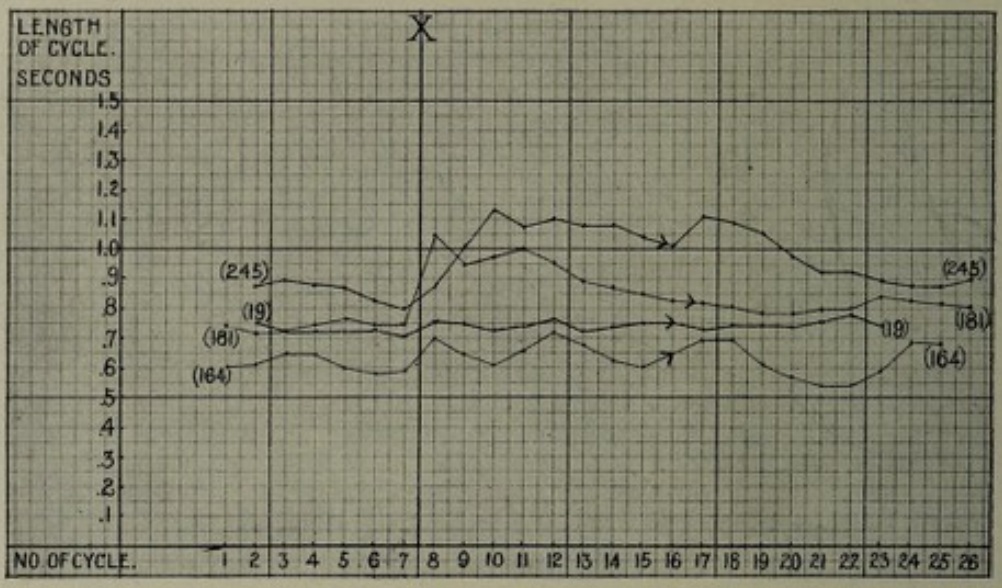

Figure 2a

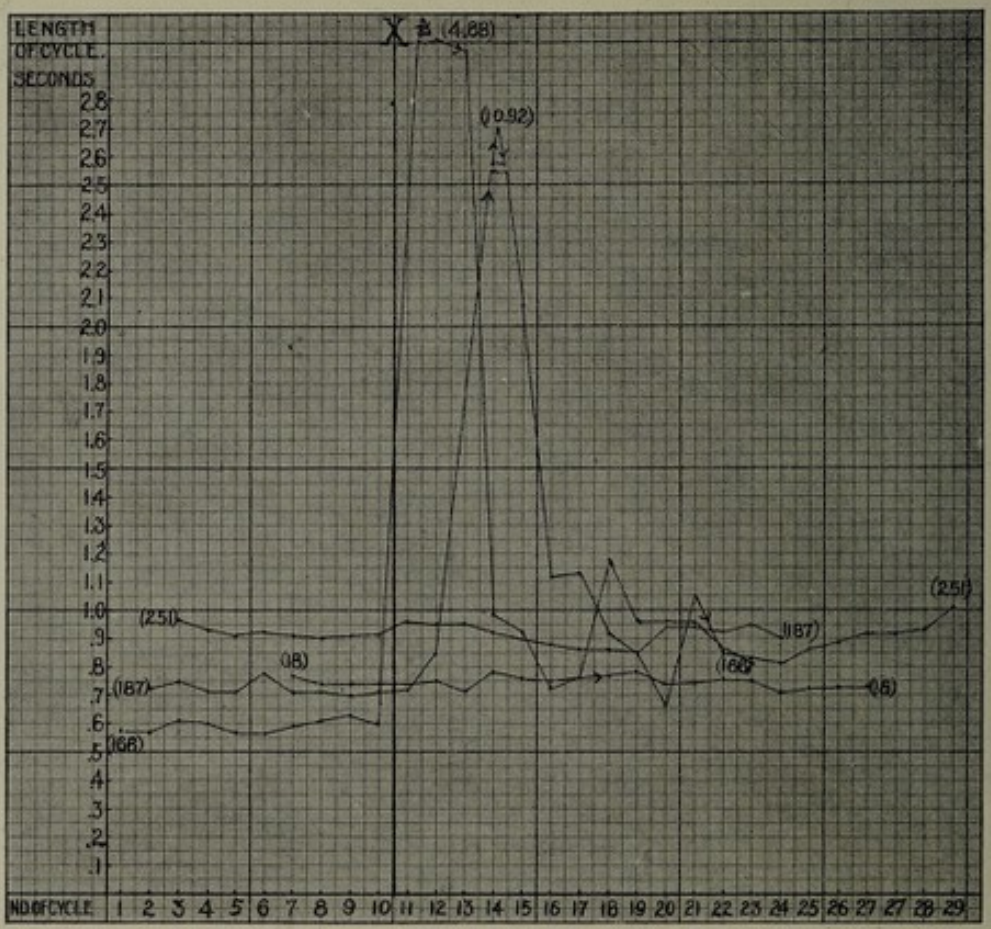

Figure 2b

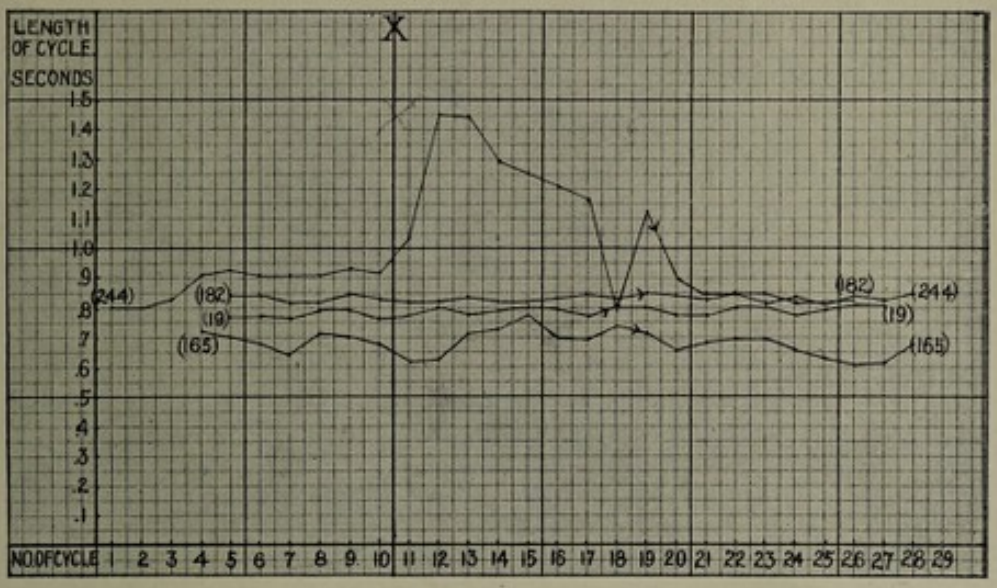

Figure 2c

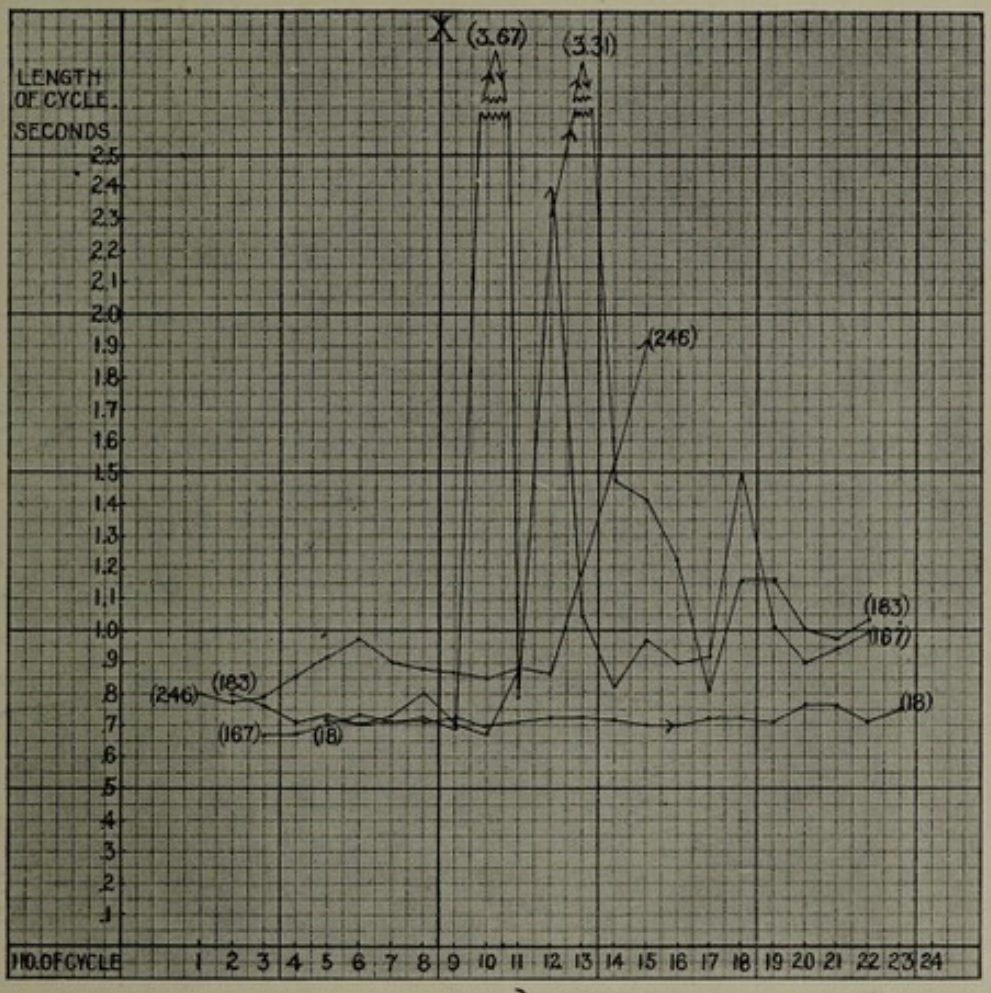

Figure 2d

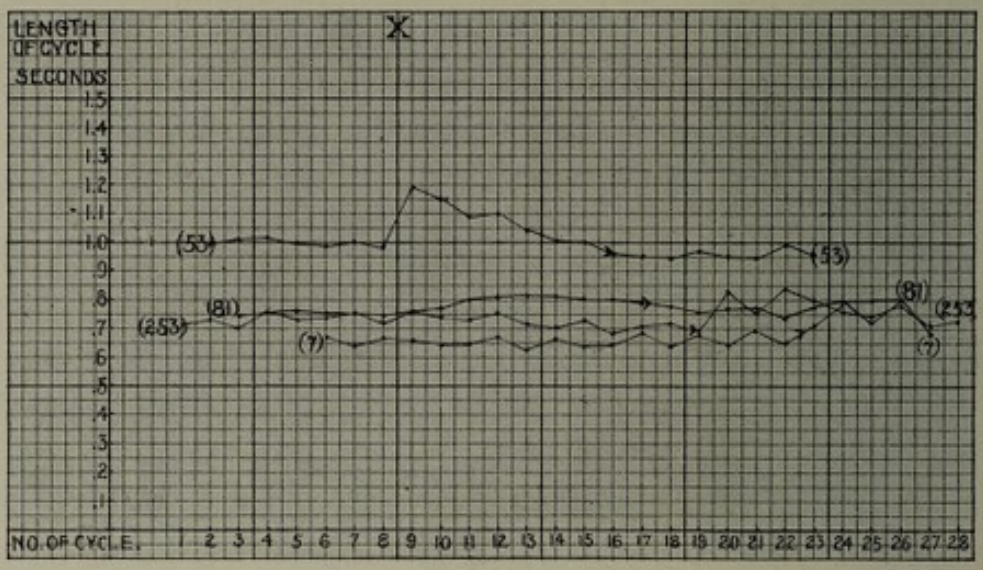

Figure 3a

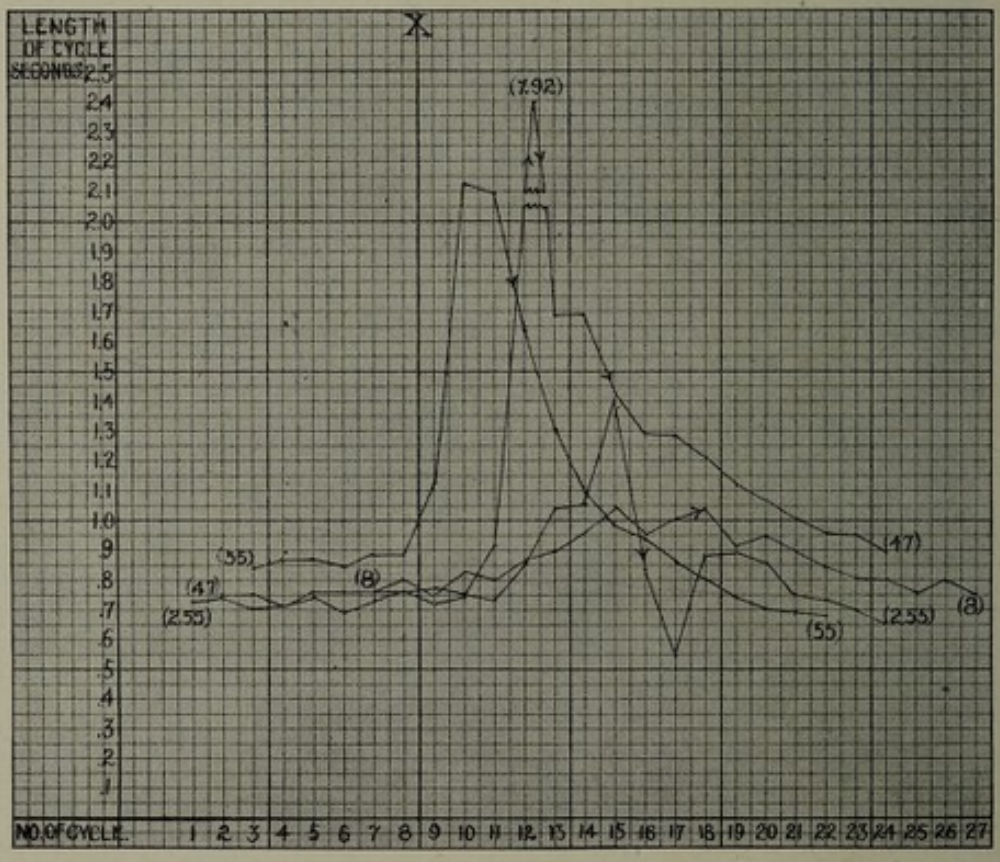

Figure 3b

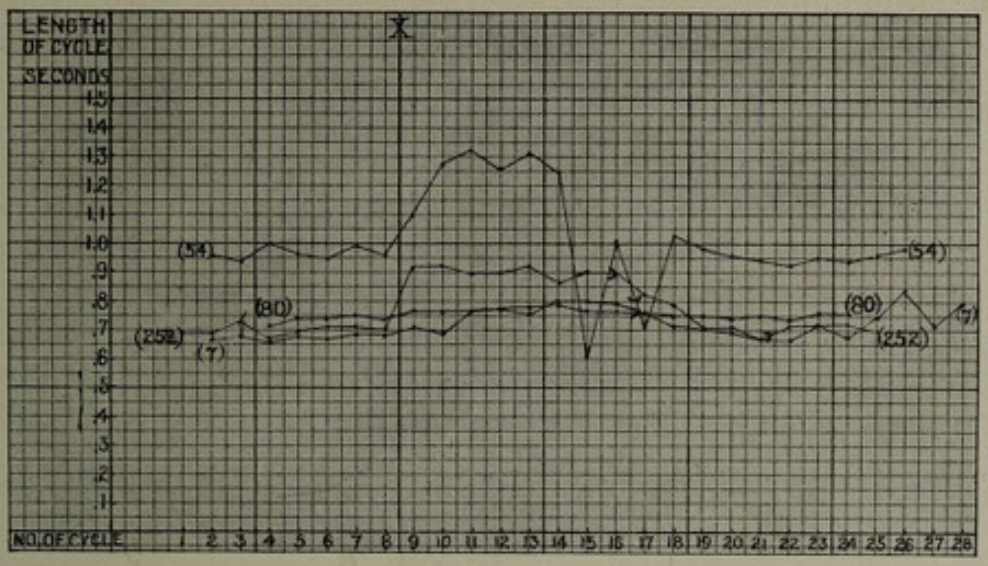

Figure 3c

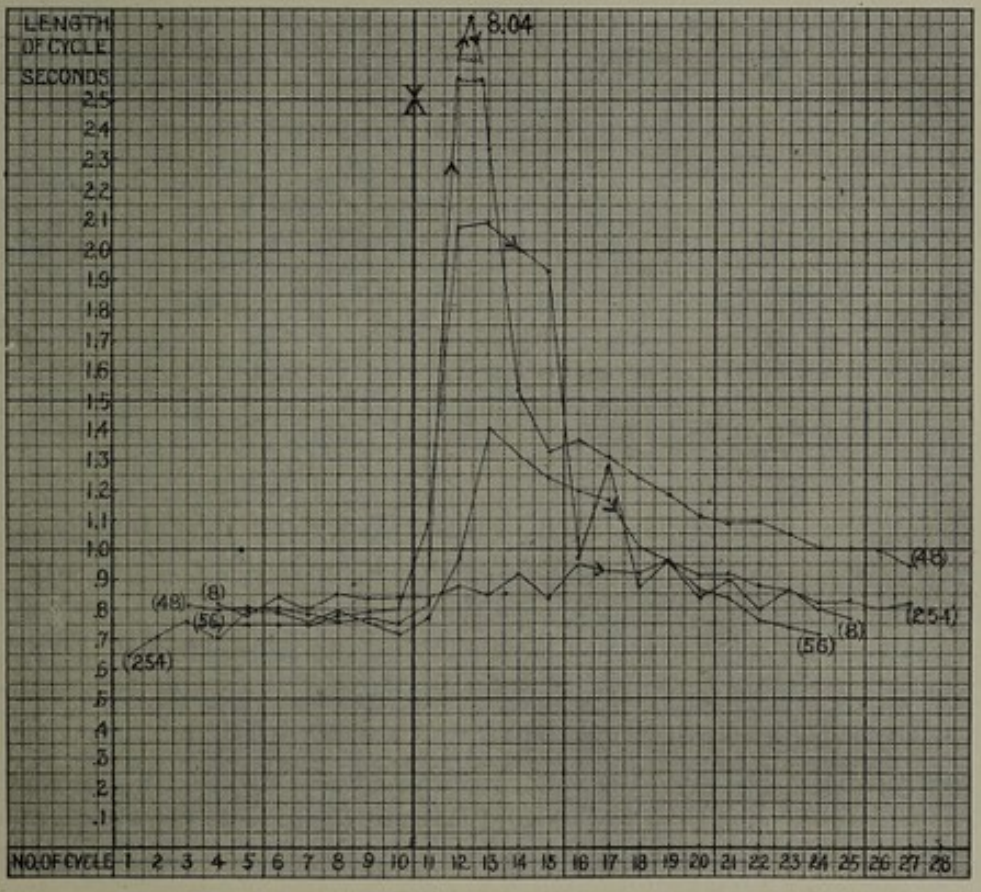

Figure 3d

were greatly diminished in height during ocular pressure, so that it was difficult to differentiate a ventricular contraction from the oscillations that are sometimes observed in electrocardiograms during stimulation of the vagus nerves. Case 24 was very sensitive to ocular pressure. The pacemaker was entirely inhibited by right ocular pressure for 10.92 seconds (Fig. 5). Auriculoventricular conduction was lengthened considerably during pressure on the left eye. The effect on conduction, however, was not limited to pressure on the left eyeball, for the P-R time following the pause of 10.92 seconds was 0.24 second.

The results of vagal and ocular pressure were also studied in five cases of *pneumonia* (Cases 17, 26, 27, 28, 30). The curves plotted from electrocardiograms taken during vagal pressures rose very slightly, that

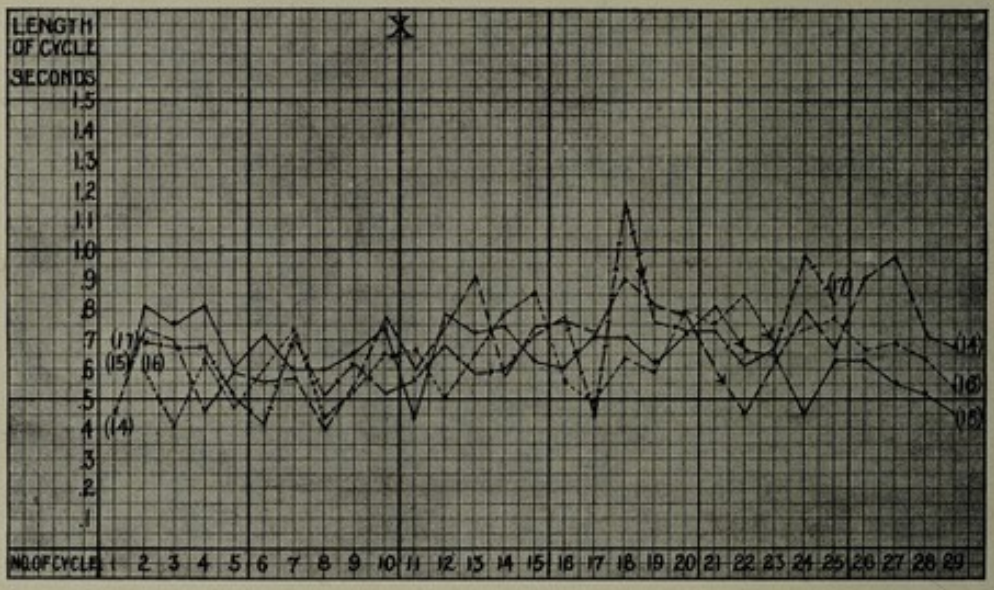

Figure 4a

Figure 4.—a and b give the results of pressure in a case (No. 3) of auricular fibrillation, before (a) and during (b) the administration of digitalis; a 14 + b 201 —— —— —— right vagus; a 15 + b 208 ——— right eye; a 16 + b 202 — — — — left vagus; a 17 + b 209 x——x——x left eye.

is to say, pressure did not slow the heart very much. The curves of ocular pressure, on the other hand, rose abruptly after a latent period of from one to three beats. This demonstrates strikingly how much more effective ocular pressure is in slowing the heart than direct vagal pressure. Another point to be observed is that the pulse rate did not drop back to normal after pressure was released, but in fact was still quite slow after ten seconds, and in some instances after thirty seconds. This occurrence emphasizes the importance of waiting between succeeding pressures on the same patient, to guard against either fatigue phenomena or summation of stimuli. One of the pneumonia patients, Case 17, showed only slight slowing. The reason for this is, probably, that not enough pressure was exerted.

Three (Cases 17, 28 and 30) of the five pneumonia cases showed no changes in the form or sequence of the curves, except the slowing. In the other two, several interesting changes were observed. In Case 26 left ocular pressure caused considerable delay in conduction time. In places auricular waves cannot be distinguished at all, and yet the ventricular complexes are of normal contour. The auricular representation is iso-electric.

The last of the pneumonia cases (No. 27) presented varied changes during ocular pressure. Figure 8 shows three inverted *P*-waves, one of which was blocked. (Inverted *P*-waves were seen in one other instance during left ocular pressure.) The same tracing showed a conduction time of 0.28 second. On five different occasions left ocular

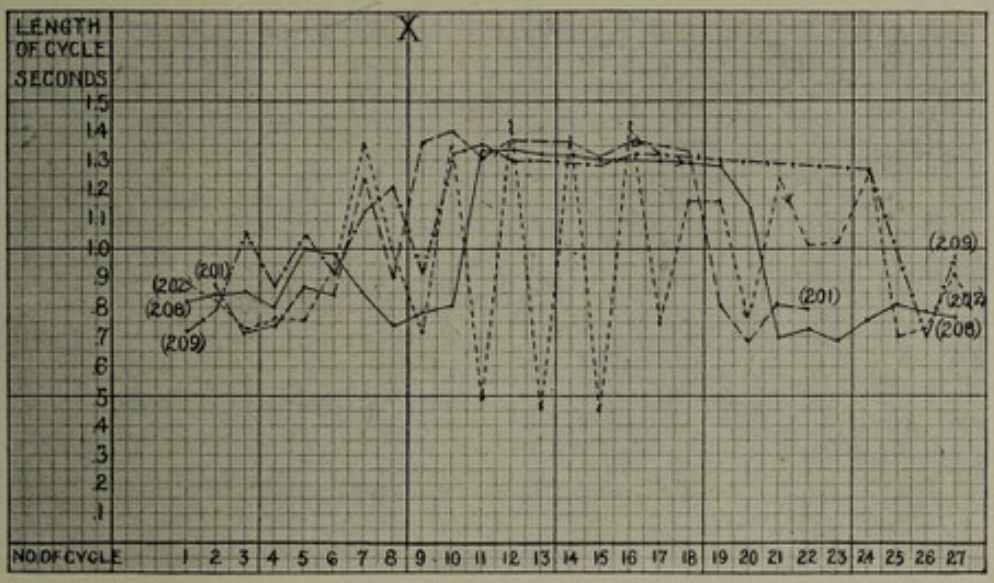

Figure 4b

pressure caused delayed conduction, the *P-R* interval rising to 0.24 and 0.32 second. In none of the four times when the right eye was pressed in this case was there any delay in conduction. Figure 6 shows partial heart-block as a result of left ocular pressure. Finally, in one instance, pressure on the left bulb caused an increase in the height of the *P*-waves, an occurrence observed also by Robinson and Draper[34] as a result of direct pressure on the left vagus nerve. In this case, then, right ocular pressure merely slowed the heart and had no effect on conduction. Pressure on the left eye, however, caused ectopic auricular contractions, delayed conduction, partial heart-block, and an increase in the height of the *P*-waves. These different effects appeared in the same individual under similar circumstances. It seems reasonable to believe that the duration and the force of the pressure, which must have varied, were important factors in determining the degree of inhibition that took place. Just as increasing strengths of faradic currents applied

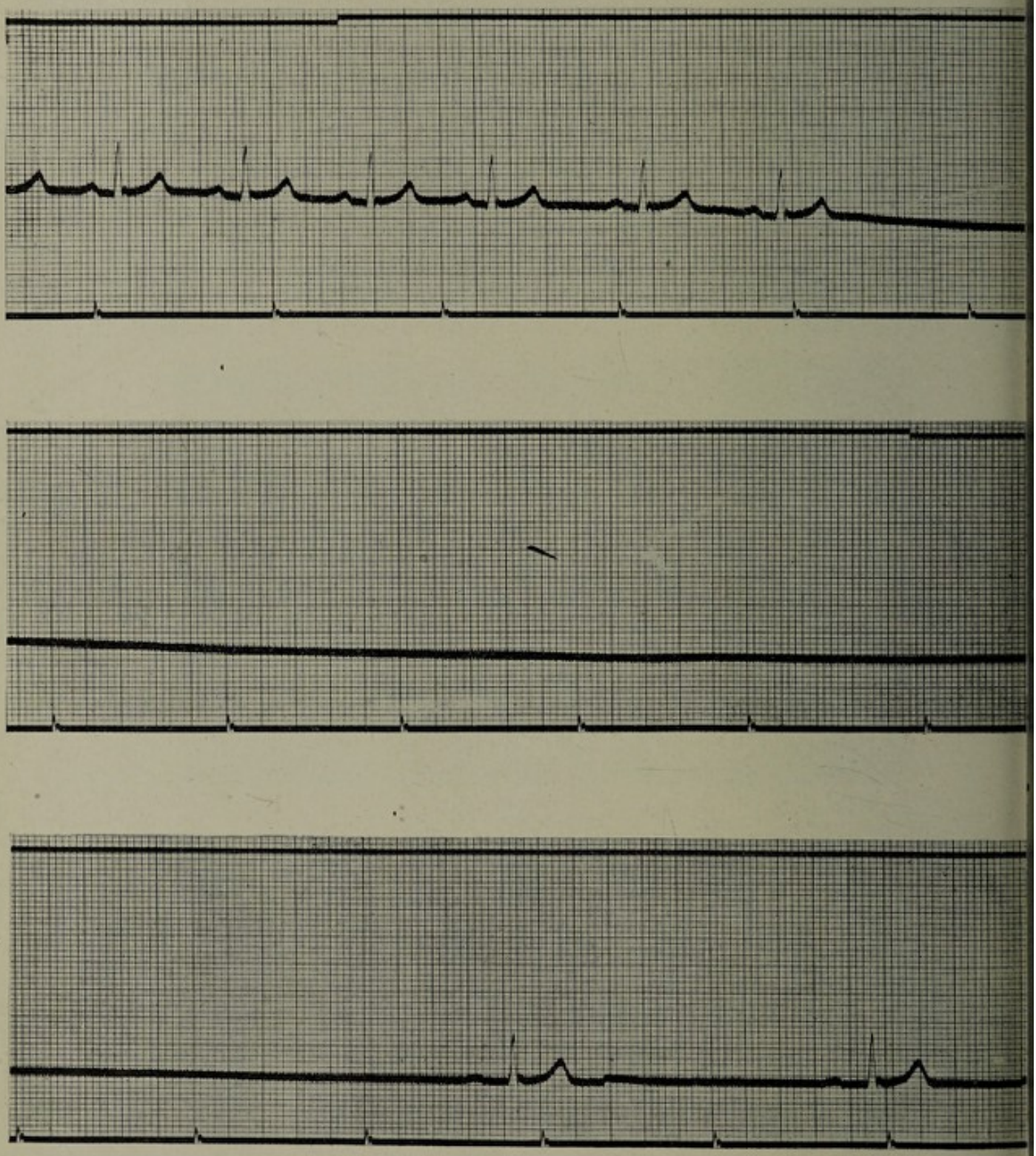

Fig. 5.—Case 24. Right ocular pressure. An electrocardiogram showing total inhibition of all bers of heart for 10.92 seconds. The *P-R* time of the beat after the long pause is lengthened to 0.24 s The slowing continued for twenty-four seconds after the release of pressure. In all curves, divisio the abscissa equal 0.04 second. Above is a signal indicating the duration of digital pressure. Below signal marking one-second intervals.

to exposed nerves in experiments on animals are accompanied by increasing effects, so may increasing pressures produce increasing effects on the rate and on conduction. Mild pressures may slow the rate and perhaps slightly affect conduction; slightly greater pressures may block auricular beats, and great pressures may stop the heart. Under certain circumstances, possibly during removal of the pressure, complete auriculoventricular dissociation might occur. It is therefore important to standardize the degree and duration of pressure. In addition, the resistance offered by the eye is probably a factor involved in eliciting the reflex.

Nine cases of *chronic valvular disease* (Patients 1, 2, 9, 10, 11, 12, 13, 15, 16), all of whom had hearts actuated by a normal rhythm, were studied. Figures 3a-d show the results in four of them (1, 2, 9 and 10). Only one (Case 10) of the nine cases had a moderate reaction to *vagal pressure,* while another (Case 1) showed a slight effect. All the others were little, if at all, affected by either right or left vagal pressure. On *ocular pressure,* eight (Nos. 1, 9, 10, 11, 12, 13, 15 and 16) reacted strikingly, and the other one (No. 2) moderately. The patient (No. 2) who reacted with moderate slowing was not pressed very vigorously; he was one of the first patients studied. The first (No. 1) patient studied showed no slowing whatever on moderate ocular pressure, but on greater pressure he had a good reaction. In this case *P*-waves diminished in height during right ocular pressure, and during left ocular pressure *P*-waves were blocked. Left ocular pressure caused delayed conduction (P-R interval 0.44 second in one instance). Right vagal pressure caused a slight delay in conduction on one occasion. There was no noticeable difference on the heart rate between the effects of right and left ocular pressure in these cases.

The results observed in one patient (Case 3) whose heart showed the rhythm of *auricular fibrillation* are included in the study. Observations were made before and during the administration of digitalis. Figure 4a shows the results of ocular and vagal pressures before digitalis was given. There was no evidence of slowing during pressure. Figure 4b, which gives the results after a course of digitalis therapy, shows a distinct slowing on pressure. There was no difference in effect between the right and left sides or between ocular and vagal pressure either before or during digitalis treatment. All other non-tabetic cases showed a distinct difference in reaction between ocular and vagal pressure. Many of them manifested differences in the effect of pressure on the right and left eyes. But here the result was the same whether ocular or vagal pressure was exerted or whether it was the left or right side that was used. The up and down swings in Figure 4b, Curve 202, are due to three premature ectopic ventricular beats alternating with

the normal ventricular contractions. There is a striking tendency for all normal heart cycles in this chart to be of the same length during pressure. Digitalis, it seems, changed the condition of the oculocardiac reflex. Wenckebach[39] and v. Hoesslin[40] have similarly shown that digitalis produces a heightening of the efficiency of vagus pressure.

One case of diabetes mellitus was studied (Case 22). Tracings were taken two different times at an interval of fifteen days. At neither time was there more than the slightest slowing either during vagal or ocular pressure. There has been no report in the literature of the condition of the oculocardiac reflex in this disease. A study of more cases is required to decide whether the condition of the reflex found in this case is typical.[41]

The sensations experienced and the objective signs displayed during pressure have been studied to determine if differences between tabetics and non-tabetics were present, and to see if they were constant. Of the

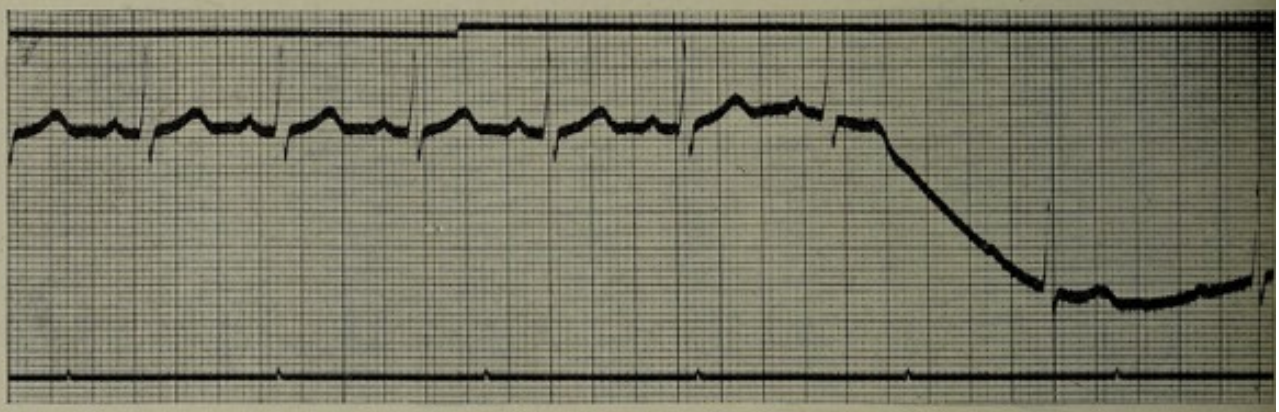

Figure 6

Fig. 6.—Case 27. Left ocular pressure. Moderate slowing of the heart is shown. There is a decrease in the size of the *P* wave. Conduction time is delayed and is gradually increased until an auricular beat is blocked. The *P-R* interval following the blocked beat is short, 0.15 second.

thirty patients studied, six had unusual sensations of one kind or another (Table 1). The others felt nothing but the pressure and the pain, when there was any. An examination of Table 1 shows that *vagus pressure* had little effect on either tabetics or non-tabetics. Occa-

39. Wenckebach, K. F.: Die Unregelmässige Herztätigkeit und ihre klinische Bedeutung, W. Engelmann, Leipzig and Berlin, 1914, p. 172.

40. Von Hoesslin, H.: Beobachtungen über den Einfluss des Vagus auf das menschliche Herz, Deutsch. Arch. f. klin. Med., 1914, cxiii, 537.

41. Since these observations were made, three cases of diabetes in the medical wards of the Peter Bent Brigham Hospital were investigated. In one case the oculocardiac reflex was absent on two different occasions. In the other two there was a moderate slowing on right ocular pressure, but none on the left.

sionally a patient complained of slight pain; very rarely there was flushing or inhibition of respiration; on a few occasions the patient swallowed during or immediately after pressure. Deglutition might very well have resulted from the discomfort of the pressure on the neck. *Ocular pressure,* on the other hand, produced a marked difference in the two groups of cases. In the tabetics, three[42] had a moderate amount of pain (marked +), three had a slight or very slight pain (marked v. sl.) and three had no pain whatever (marked —). In five cases there was slight flushing and in four none at all. Respiration was checked during pressure on only two occasions and at no time was there any impulse toward swallowing. In contrast to this are the results in non-tabetics. The pain from ocular pressure was quite severe in all but two cases. One of these was a case of syphilitic myelitis (there is evidence that he is developing tabes), and the second was subjected to much lighter pressure than any of the others. There was

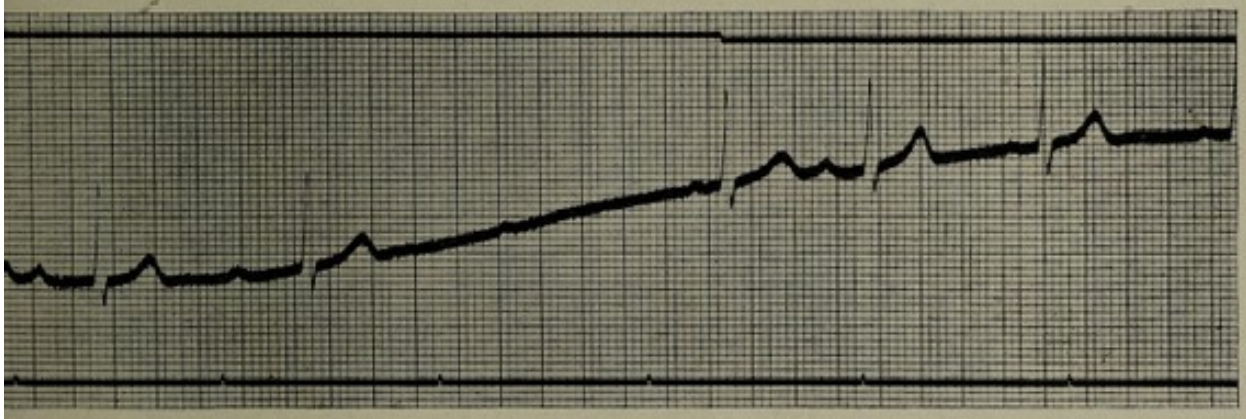

Figure 6—Continued

flushing of the face in all but two cases. Respiration was checked in fifteen of the twenty cases. It is evident that pain, flushing of the face and respiratory inhibition generally occur during ocular pressure in non-tabetics, and to a much less extent in tabetics.

DISCUSSION AND SUMMARY

The oculocardiac reflex (inhibition of the heart produced by ocular pressure) is a normal but variable reflex. The statement that a heart is abnormal if its rate is slowed or accelerated by more than ten beats a minute, made by Loeper and Mougeot[5] is not substantiated by the observations made in this study. Some individuals normally have a more active oculocardiac reflex than others, just as some have more active tendon and superficial reflexes. In fact, when the reflex is con-

42. I have included under tabetics Case 7, which is probably a case of early tabo-paresis.

TABLE 1.—Sensations Elicited by Pressure in Tabetics and Non-Tabetics

Case	Vagus				Eye				Subjective Symptoms
	Pain	Flushing	Inhibition of Respiration	Deglutition	Pain	Flushing	Inhibition of Respiration	Deglutition	
TABETICS									
4	—	+	—	+	+	sl.	—	—	Felt slight pain with ocular pressure, especially right.
6	—	—	—	—	—	—	—	—	Felt nothing but the pressure. No pain.
7	—	—	—	—	+	—	—	—	Slight pain in eyes. Pins and needles sensation in right eye.
8	—	—	—	—	—	—	—	—	Had no other feeling than that of pressure.
18	—	—	+	—	—	sl.	—	—	Merely felt pressure.
19	—	—	—	—	sl.	sl.	+	—	Merely felt pressure.
20	—	+	—	—	+	sl.	—	—	Pressure on left vagus made him feel faint.
23	—	—	—	—	vsl.	—	—	—	Felt nothing except slight pain in eyes.
25	—	—	—	—	sl.	sl.	—	—	Felt very slight pain in the eyes.
NON-TABETICS									
1	—	—	—	—	+	+	—	+	Pain in the eyes.
2	..	..	..	..	..	..	..	..	No record obtained.
3	—	sl.	—	—	++	+	—	—	Felt faint, choking sensation, cramp in belly when eyes were pressed.
5	—	—	—	—	—	—	—	—	Felt nothing peculiar. (See protocol.) (May be tabetic.)
9	—	+	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
10	—	—	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
11	—	—	+	—	++	—	+	—	Felt nothing but pain from pressure on eyes.
12	—	—	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
13	+	+	+	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
14	—	—	—	+	++	+	+	—	Felt nothing but pain from pressure on eyes.
15	—	—	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
16	—	—	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
17	+	—	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
21	rt. +	rt. +	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
22	—	sl.	—	—	++	+	+	—	Felt nothing but pain from pressure on eyes.
24	—	—	—	—	++	+	+	—	Felt slow electric current going through body when eyes were pressed. It caused him much pain.
26	—	—	—	—	++	+	+	—	Left eye pained more than right.
27	—	—	—	—	++	+	+	—	Left eye pained more than right.
28	—	—	—	—	++	+	+	—	Felt nausea when right eye was pressed, in addition to pain.
29	—	—	—	—	++	+	—	—	Merely felt pain.
30	—	—	—	—	++	+	—	—	When right eye was pressed right leg felt dead and it lifted itself up from bed.

sidered absent there is often a slight inhibition of the heart, as is shown by a tendency for the curves in Figures 1b and 1d to rise during pressure. It seems highly probable that different effects may be produced in the same individual by changing the duration and the degree of ocular pressure. This is shown in several cases described in this investigation. The comparison which was instituted made it clear that inhibition of the heart could much more easily be obtained by ocular than by direct vagal pressure. A difference as striking as that which has been observed between the two can only be obtained if the eyes are pressed quite firmly, causing the patient slight distress. Ocular pressure brings out disturbances in the heart mechanism which were not obtained by direct vagal pressure.

The reflex was found to be generally absent in tabes dorsalis. The cause for this probably is a lesion in the brain itself. Although in all the cases of tabes the pupils were inactive to light or reacted sluggishly, there does not seem to be any constant relation between the occurrence of the Argyll Robertson phenomenon and the loss of the oculocardiac reflex. In fact, the only tabetic who had a moderate oculocardiac reflex, had no pupillary reaction to light whatever. These findings in tabetics are in accord with the results obtained by previous investigators. Tabetics do not complain of pain on ocular pressure to the same extent as non-tabetics, nor do flushing of the face and periods of apnea during pressure occur to as great a degree as in non-tabetics. It is not impossible that these reflex phenomena result from the sensation of pain. Of the five cases of syphilis (Cases 5, 14, 21, 24 and 29), if those patients are excluded who might possibly be developing tabes, namely, Cases 5 and 14, then three, or 100 per cent., had a normal oculocardiac reflex; and if the other two are included, all but one, Case 5, or 80 per cent., reacted normally. These results differ decidedly from those of Loeper, Mougeot and Vahram,[19] who found the reflex absent in 75 per cent. of their cases. One case of diabetes mellitus and one case of auricular fibrillation were found to have no oculocardiac reflex. No explanation for its absence can be given in the case of the diabetic. The patient with auricular fibrillation reacted after he was given digitalis. Of all the other cases in this series (including nine cases of chronic valvular disease, five of pneumonia, and five of syphilis) only one (Case 5) did not have an oculocardiac reflex, and he had signs of early tabes.

In tabetics there was no difference between the effects of pressure on the two eyes. In non-tabetics there was a distinct difference. The right reflex had a slightly greater effect on auricular contraction.[43] The

43. The greater slowing seen in the charts of pressures on the left side is only apparent and not real; it depends on the blocking of auricular beats which were slowed less by left than by right sided pressures. Rates, however, are counted from ventricular contractions.

different grades of inhibition caused by right ocular pressure were, first, a diminution in the height of the *P*-waves with only moderate slowing; second, disappearance of the *P*-waves and slowing of the rate of the ventricular contractions; third, complete inhibition of all the chambers of the heart. Left ocular pressure, on the other hand, may cause similar phenomena, but does not do so as often as right. The *P*-waves frequently could not be identified, but after pressure was released they gradually reappeared (Fig. 7). The ventricular representative, *R*, continued to have a normal form even though the ventricular rate was reduced. It is impossible in these cases to say whether the normal pacemaker was completely inhibited or whether the origin of the impulse for ventricular contractions resided in the junctional tissue. In most instances the pacemaking function probably continued to be at the sinus node. In any case the impulse was supraventricular in origin, for the form of the ventricular complexes remained normal.

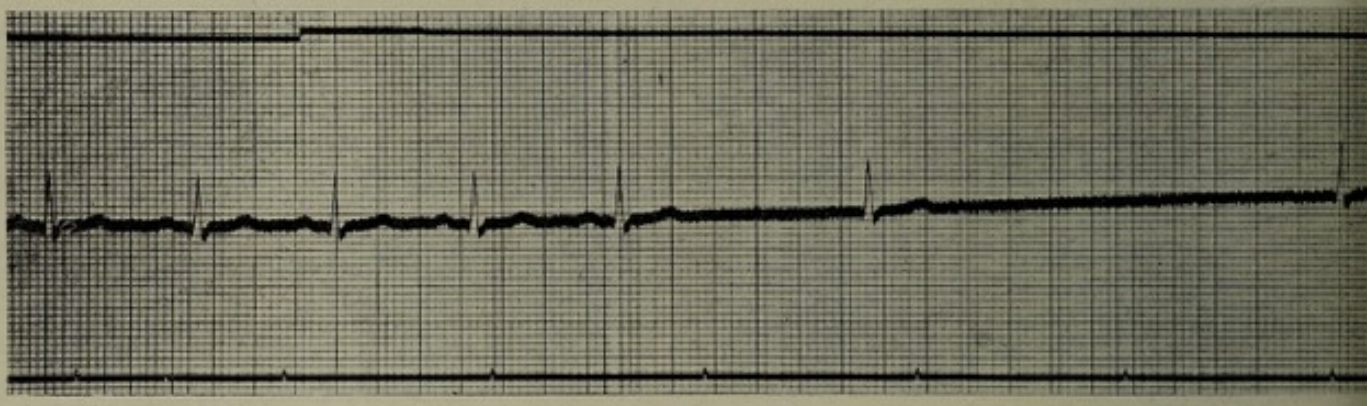

Figure 7

Fig. 7.—Case 26. Right ocular pressure. Total inhibition of the heart for periods of 2.27 seconds to 3.65 seconds is shown. *P*-waves are entirely absent; the ventricular complexes are of normal form. Beat V is an escaped ventricular contraction of normal form.

The change in the mechanism of the heart beat caused particularly by left ocular pressure were delayed conduction, partial heart-block and inversion of the *P*-waves. Complete inhibition of all the chambers of the heart also occurred. Ectopic ventricular beats occurred in two cases during left ocular pressure. Accurate measurements of the heights of the *P*- and *R*-waves before and during ocular or vagal pressure were not made. *P*-waves diminished in height in many instances during both left and right ocular pressure. One case showed a decrease in the height of the *P*-waves even before slowing of the heart began. Occasionally the height of the *P*-wave was increased by left ocular pressure.

Differences between right and left oculocardiac reflexes, such as were obtained by stimulation of the left and right vagus, were not as definite and striking as those obtained in experiments by Cohn.[44] The

TABLE 2.—Patients Referred to in the Curves

Patient 4	Patient 18	Patient 20	Patient 25	Patient 5	Patient 21	Patient 24	Patient 29	Patient 2	Patient 10	Patient 9	Patient 1
Fig.				Fig.				Fig.			
1a 12	144	156	193	2a 19	164	181	245	3a 7	53	81	253
b 13	146	154	195	b 18	166	187	251	b 8	55	47	255
c 12	145	157	194	c 19	165	182	244	c 7	54	80	252
d 13	147	155	196	d 18	167	183	246	d 8	56	48	254

fact that these differences were not so clearly demonstrated in the oculocardiac reflex must be due to the more complicated pathway pursued by the impulses through the brain. According to the experiments referred to, the right vagus has a greater influence on the pacemaker

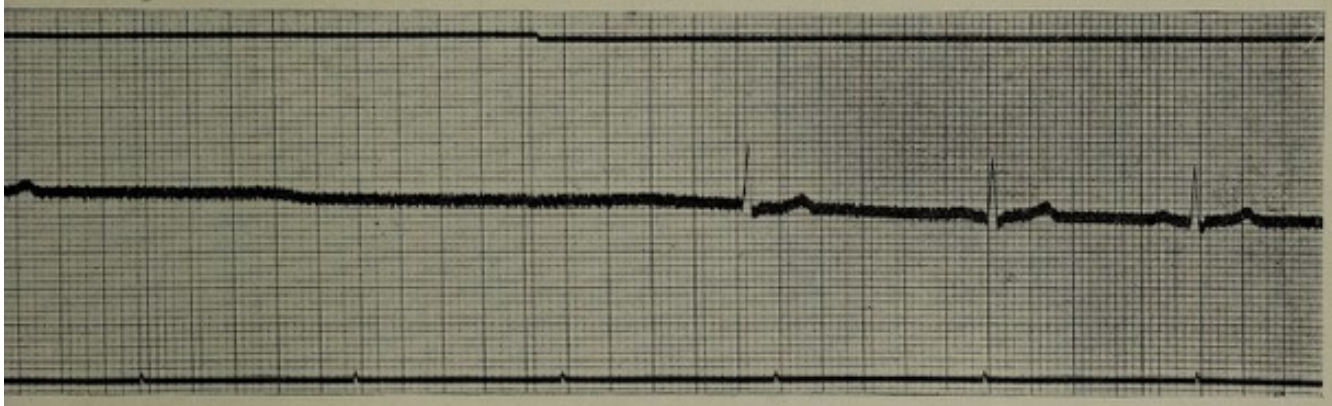

Figure 7—Continued

and the left a greater influence on auriculoventricular conduction. The difference between pressure on the two eyeballs compares satisfactorily, however, with that found by Robinson and Draper[34] in their vagus experiments.

CONCLUSIONS

1. Ocular pressure affords a simple and safe method of obtaining reflex vagus inhibition of the heart.

2. Inhibition of the heart by the oculocardiac reflex is much more profound and more frequently obtained than by pressure over the vagus nerves.

3. The oculocardiac reflex is generally absent in tabes dorsalis, present in pneumonia, syphilis (non-tabetic) and chronic valvular disease.

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44. Cohn, A. E.: On the Differences in the Effects of Stimulation of the Two Vagus Nerves on Rate and Conduction of the Dog's Heart, Jour. Exper. Med., 1912, xvi, 732.

4. The reflex was absent in one case of diabetes mellitus and also in one case of auricular fibrillation before digitalis treatment. It was present after digitalis was given.

5. Right ocular pressure has a slightly greater effect on the rate of the heart than left. It may stop the heart for a long period of time, relatively speaking, the *P*-waves are sometimes diminished in size and may become iso-electric. Occasionally the auriculoventricular interval is lengthened.

6. Left ocular pressure has a much greater effect on the conduction mechanism of the heart than right. It may lengthen auriculoventricular conduction, cause partial heart-block and result, possibly, in automatic ventricular rhythm. On two occasions inverted *P*-waves were seen. The height of the *R*-waves is sometimes increased, at other times

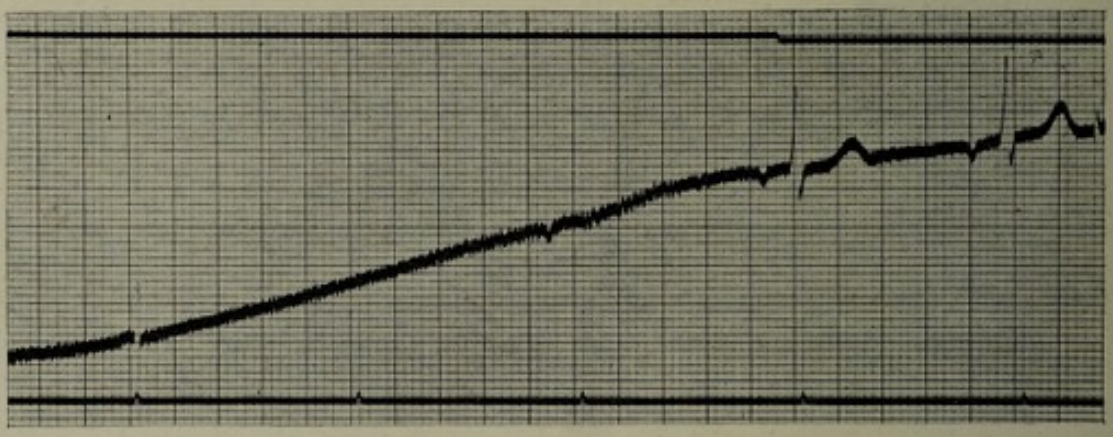

Fig. 8.—Case 27. Left ocular pressure. Inhibition of the ventricle is shown. Three inverted *P*-waves occurred one of which was blocked; in the other two the *P-R* time is short. After release from pressure the *P-R* time was 0.32 second.

diminished. Ectopic ventricular beats were twice observed. The *P*-waves are often diminished in size, but occasionally are increased. Escaped ventricular beats were seen both during right and left ocular pressure.

7. Pain, flushing of the face, and apnea during ocular pressure, are much less pronounced in tabetics than in non-tabetics.

8. The effects on the rate and on the rhythm of the heart produced by ocular pressures are not constant, differing in different individuals and in the same individual from time to time. The duration and the degree of pressure play an important part in the degree of inhibition.

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