REPORT

of

Committee on Thesis

The undersigned, acting as a Committee of the Graduate School, have read the accompanying thesis submitted by Fredrick Arthur Willius for the degree of Master of Science in Medicine. They approve it as a thesis meeting the requirements of the Graduate School of the University of Minnesota, and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science in Medicine.

H. A. Plummer
Chairman

Walter M. Boothby

William A. Lemon

S. Marx White

W. Wilson

THE UNIVERSITY OF MINNESOTA

GRADUATE SCHOOL

Report

of

Committee on Examination

This is to certify that we the undersigned, as a committee of the Graduate School, have given Frederick Arthur Willius final oral examination for the degree of Master of Science in ~~Medicine~~. We recommend that the degree of Master of Science in Medicine be conferred upon the candidate.

Minneapolis, Minnesota

May 25 1920

H. S. Plummer
Chairman

S. Marx White

Walter M. Boothby

M. Sehaus

Statement Concerning Life of Candidate for Master's Degree (to be read by Chairman at the final oral examination; copy to be filed in the Dean's Office).

1. Name of candidate (in full):

Fredrick Arthur Willius.

2. Place and Date of birth:

St. Paul, November 24, 1888.

3. Preliminary education, secondary and collegiate:

University of Minnesota, 1908-1910.
Medical School, University of Minnesota, 1910-1914.

4. Degrees obtained, with dates and names of institution:

B. S., University of Minnesota, 1912.
M. D., University of Minnesota, 1914.

5. Language examinations passed:

Unnecessary.

6. Minor line of work, with date of final written examination:

Physiology, No examination necessary.

7. Major line of work, with date of final written examination:

Medicine. Examination May 13, 1920. Grade B.

8. Thesis subject, with date of final approval:

Observations on negativity of the final ventricular wave T of the electrocardiogram.

9. Degree applied for:

Master of Science in Medicine.

Graduate School, University of Minnesota.

May 22, 1920.

This is to certify that Fredrick Arthur Willius, a candidate for the degree of Master of Science in Medicine, has passed the final written examination for the major in the Department of Medicine.

H. S. Plummer

For the Major Department.

Graduate School, University of Minnesota.

May 22, 1920.

This is to certify that Fredrick Arthur Willius, a candidate for the degree of Master of Science in Medicine, has satisfactorily completed the requirements for the minor in the Department of Physiology.

F. C. Mann

For the Minor Department.

THESIS

OBSERVATIONS ON NEGATIVITY OF THE FINAL VENTRICULAR WAVE T OF THE ELECTROCARDIOGRAM

Fredrick Arthur Willius

Submitted to the Graduate Faculty of the University of Minnesota in partial fulfillment of the requirements for the Degree of Master of Science in Medicine

May, 1920

A review of the literature on electrocardiography, both experimental and clinical, at once reveals a variance in views as to the interpretation of the normal electrocardiogram. The two views that have gained broadest recognition are: (1) All waves are manifestations of excitation and contraction of heart muscle, and (2) the waves result from electrical changes accompanying conduction of the impulse and contraction of the muscle.

Einthoven assumed that the right ventricle represents the cardiac base and the left ventricle the apex, and that the dominance of negativity in the right ventricle causes an upward deflection while dominance in the left causes a downward deflection of the galvanometer. Thus the R wave is ascribed to contraction of the right heart, the S wave to contraction of the left heart, and the horizontal S-T interval to neutralization of basal and apical negativity. The T wave represents contraction of the right ventricular base outlasting that of the left.

Eppinger and Rothberger object to Einthoven's assumption in ascribing the rôle of cardiac base to the relatively weak right ventricle and regarding the left ventricle with its massive muscle bulk as the apex.

The views of Kraus and Nicolai are based on the structural arrangement of the ventricular musculature into systems. Following auricular contraction the impulse passes through the auriculoventricular bundle and its contiguous structures. The long P-R interval is explained by slow conduction

and the absence of appreciable action currents at this time due to the small muscle mass. The R wave is ascribed to primary activity of the basal portions of the papillary muscles. As the excitation wave spreads toward the apex the termination of the R wave occurs. The S-T interval is explained by the absence of potential between base and apex. The T wave results from late return of negativity to the base.

The views of Einthoven, Kraus and Nicolai agree in general in emphasizing the antagonistic action of electrical potential between base and apex.

In distinct opposition to the views already expressed, Hoffmann concluded that the electrocardiogram results from two actions, impulse conduction, and muscle contraction. The Q R S complex results from passage of the impulse through the ventricular conduction system and the S-T interval and the T wave from electrical potential caused by the contracting ventricle. Hoffmann produced standstill of the frog heart by the application of muscarin, obtained simultaneous electrocardiographic and ventricular suspension curves, and found that the electrocardiogram of the nonbeating heart differs from the normal only in the absence of the T wave. When the muscarin effect was abolished by atropin and the beats returned the T wave reappeared.

Eyster and Meek, as a result of their experimental work on the relation of the line of isopotential to the formation of the electrocardiogram, and their critical review of the literature, in general agree with Hoffmann's theory. They believe the R wave to be concerned with conduction but they do not ascribe definite structures as conducting mediums. The T wave is the expression of preponderance of contraction on one side of the line of equipotential. Eyster and Meek further show the differences between physiologic

curves of conduction and contraction. When a nerve is stimulated where conduction alone occurs, a single monophasic or diphasic electrical response occurs. In skeletal muscle this rapid electric change is followed by a slower and more prolonged electric variation. This conforms with the general contour of the electrocardiographic deflections; the R wave is abrupt and steep, the T wave blunt and prolonged.

Cardiac events and the electrocardiogram

The relationship of the waves of the electrocardiogram to definitely known cardiac events strongly supports the "conduction-contraction" theory. By a consideration of accepted relationships of the normal heart sounds a working basis for comparison is established.

Einthoven, Flohil and Battaerd have shown that the first sound begins at the initiation of ventricular systole and lasts from 0.07 to 0.10 seconds, is followed by a pause varying from 0.15 to 0.25 seconds, and then succeeded by the second sound. These observations have been confirmed by other investigators.[1,10,35] The second sound follows closely on the closure of the semilunar valves,[37] begins simultaneously with the rise of intraventricular pressure,[14] and gains its maximum amplitude during this period.[33] That the second sound is an early diastolic event has been shown clearly.[26,36]

Kahn has demonstrated that the first sound falls in the pause between the R and the T waves and begins at the moment the R wave disappears and a short time before the rise of the T wave. The second sound begins 0.05 seconds after the end of the T wave. This relationship reveals the fact that the R wave is completed before ventricular contraction begins and indicates

conduction rather than contraction. The T wave definitely occupies the period associated with actual ventricular contraction.

In clinical studies of the abnormalities of the Q R S group they are largely ascribed to disease of the ventricular conduction system.[3,24,34,38,39] It must be recognized, however, that the graphic representation of contraction in the electrocardiogram is the expression of changes in electrical potential and not the translation of actual contraction. The iso-electric portion of the intervals S-T and a portion of T-P are not latent in the sense of a refractory phase.

Consideration of electropotential

Waller and Reid demonstrated a line of equipotential passing through the heart from base to apex in relation to any two derivations from the extremities. A preponderance of negativity above this line, representing the cardiac base, caused deflection of the galvanometer connected to both upper extremities in a manner to indicate relative negativity of the electrode connected with the right arm. The arm becomes relatively negative in derivations from an arm and a leg. Dominance of negativity below the equipotential line deflects the galvanometer in the opposite direction.

Hypothesis of T wave negativity

The three derivations of the electrocardiogram possess symbols of definite electropotential in relation to their electrodes. In the normal electrocardiogram Derivation 1 bears symbols as follows : right arm -, left arm +; Derivation 2, right arm -, left leg +; and Derivation 3, left arm -, left leg + (Fig. 1). This arrangement implies positive or upright deflections

Positive T wave in all Derivations (normal)

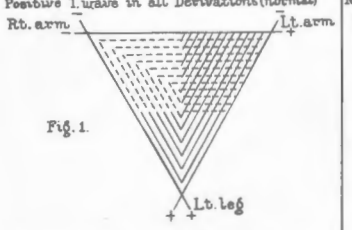


Fig. 1.

Negative T wave in Derivation I.

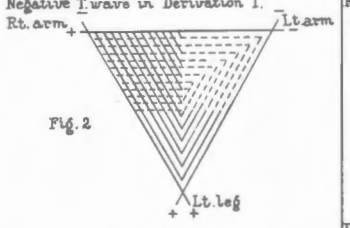


Fig. 2

Negative T wave in Derivation III.

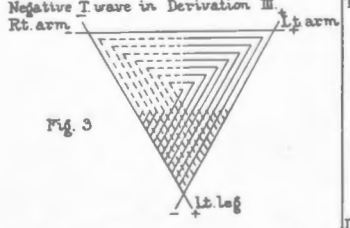


Fig. 3

Negative T wave in Derivations I and II.

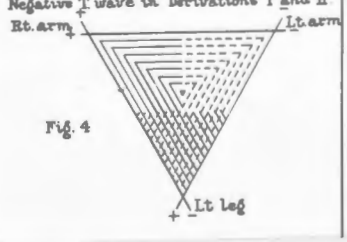


Fig. 4

Negative T wave in Derivations II and III.

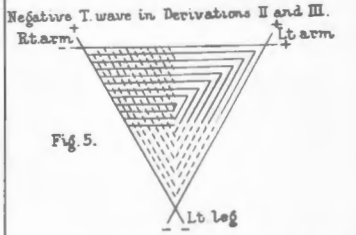


Fig. 5.

Negative T wave in Derivations I, II and III

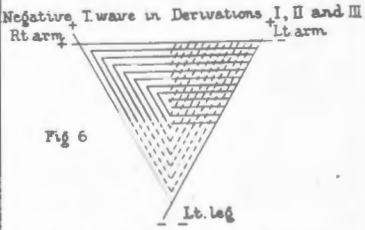


Fig 6

Diffuse iso-electric state of heart if negative T wave in Derivation II occurred.

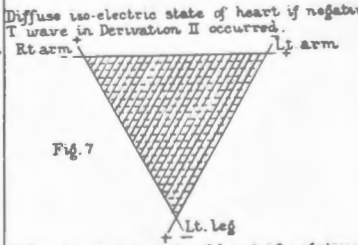


Fig. 7

Diffuse iso-electric state of heart if negative T wave in Derivations I and III occurred.

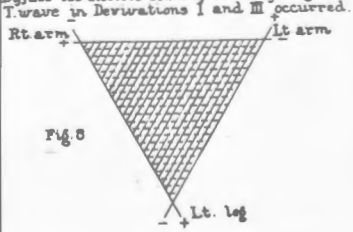


Fig. 8

in all derivations of the electrocardiogram. If it is assumed that the "conduction-contraction" theory is correct, the T wave is the expression of preponderance of contraction on one side of the line of equipotential. T wave negativity (inversion) therefore results from changes in contraction preponderance. The negativity of this wave in certain isolated or combined derivations of the electrocardiogram is indicative of definite potential changes affecting contraction preponderance in various regions of the cardiac musculature.

In the normal heart, therefore, according to standard derivations, the T wave in all derivations is positive (upright), the right upper zone of potential is strongly electronegative to the apical zone, while the left upper zone is iso-electric. This potential arrangement is illustrated in Figure 1.

For reasons of simplification I have represented the three derivations by the sides of an equilateral triangle. To prevent misunderstanding it should be stated that the schematic figure employed, divided into zones of electropotential, is not based on mathematical consideration. The right upper zone in general corresponds to the sinus region of the heart and in the normal potential arrangement, is electronegative.

Confirmation of this is found in the researches of Keith and Flack, Wybauw, Lewis, Oppenheimer and Oppenheimer, Brandenburg and Hoffmann, and Ganter and Zahn, who have shown that the cardiac impulse takes its origin in a collection of specialized tissue, a remnant of the primordial sinus. This structure lies in the sulcus terminalis at the juncture of the superior vena cava and the right auricular appendage , and is the seat of primary cardiac

negativity.

Changes in the normal potential distribution produce T wave negativity in isolated or combined derivations of the electrocardiogram. ~~These changes are the result of electrode potentials~~ Reversal of potential in one derivation alters cardiac potential so that T wave negativity in that derivation occurs.

T wave negativity

Many opinions have been expressed as to the significance of negativity or inversion of the T wave in isolated or combined derivations of the electrocardiogram.

The effect of digitalis on the heart as manifested by negativity of the T wave is well known[4]; it has been ascribed to muscular ventricular redistribution or possibly to alteration in muscular contractility. These changes are not permanent.

Numerous statements may be found in which myocardial damage is ascribed to T wave negativity in certain derivations[3,18,19,25] and again these occurrences have been noted in apparently normal hearts[13,20,31], affecting largely Derivation 3.

Smith, during his experimental work on coronary ligations, observed interesting changes in the T wave. The most constant changes in the electrocardiogram following ligation of any branch of the left coronary artery affected the T wave. A strongly positive to a markedly negative wave resulted fairly constantly with a slower return to positive or iso-electric. The negativity was usually ovserved within twenty-four hours after ligation and lasted for from three to four days. The duration seemed to bear a relationship to the size of the artery ligated. This work offers a tangible basis in

directing attention to changes in the intrinsic blood supply of the heart as evidenced by greater or lesser derangements of cardiac function. Morison has shown that blood volume alterations may produce changes detectable in the electrocardiogram.

Increased general cellular function implies increased blood volume for the maintenance of normal tissue metabolism. In a specialized organ this augmentation is manifested by an increase in its function. In the heart increase in the blood volume beyond physiologic limits increases contraction. I refer particularly to increase of ventricular blood volume since coronary volume is largely dependent on this factor. Because of impairment or disease of certain intrinsic channels of blood supply, the affected muscle does not receive the requisite amount of blood properly to maintain function while the unaffected muscle demands greater blood volume for relatively more efficient contraction. This is an explanation for T wave changes in isolated or combined derivations occurring permanently or temporarily.

The action of the cardiac nerves on T wave negativity

Stimulation of the cardiac vagus has been shown to produce negativity of the T wave,[5,29] and the same observation is noted following stimulation of the left cervical sympathetic branches.[28]

A series of clinical observations were conducted on patients having negative T waves in isolated or combined derivations of their electrocardiograms. Following the initial tracing pressure was applied in turn to the right vagus region in the neck, to the left vagus region, and finally to the right eye ball (oculocardiac reflex), and the respective electrocardiograms

obtained. In no instance was any change noted in the negative T wave or in the positive T wave of the unaffected derivations. Atropin (gr. 1/150) was then administered subcutaneously and records obtained every ten minutes for forty minutes. Again no effect on the T wave was noted. No change occurred following the subcutaneous administration of adrenalin (0.5 c.c. of a 1 to 1000 solution). These clinical procedures, however, are obviously not so accurate as direct experimental stimulation.

The present status of cardiac histopathology fails to explain many derangements of function. Histologic studies do not indicate why auricular fibrillation occurs in one heart and auricular flutter in another. Disease of the cardiac conduction system is more definite histologically. Involvement below the auriculoventricular bundle is often associated with unquestionable intrinsic vascular changes.

There has been no adequate explanation of the occurrence of the negative T wave in isolated or combined derivations of the electrocardiogram. In the Mayo Clinic we have repeatedly observed the negative T wave in Derivation 1, in Derivation 3, in combined Derivations 1 and 2, in combined Derivations 2 and 3, and in combined Derivations 1, 2, and 3. No instances of negativity in Derivation 2 or in combined Derivations 1 and 3 are recorded. This observation in 7,000 electrocardiographic examinations eliminates the element of coincidence. There is a definite reason why these changes do not occur. Lewis states that the T wave is always upright in Derivation 2 alone.

T wave negativity in Derivation 1, Figure 2.- Figure 2 represents the arrangement of potential existing with this abnormality. The left arm becomes electronegative to the right while the other signs remain unchanged. The right

upper zone, instead of being electronegative, becomes iso-electric while the left upper becomes electronegative to the apex. The occurrence of an iso-electric state in the right upper zone is a marked departure from the normal in that the area of primary electronegativity is altered.

T wave negativity in Derivation 3, Figure 3.- The left leg becomes electronegative to the left arm; the other derivations remain unchanged. The right upper zone remains electronegative with reference to the left upper. The apical zone becomes iso-electric. This distribution is but a slight deviation from normal in that the right upper zone remains electronegative.

T wave negativity in combined Derivations 1 and 2, Figure 4.- The left arm becomes electronegative to the right arm and the left leg electronegative to the right arm. The third derivation remains unchanged. The left upper zone becomes electronegative to the right, and the apical zone becomes iso-electric. This arrangement again deviates from the normal in altering the area of primary electronegativity; it is a lesser change in that the right upper zone becomes electropositive instead of iso-electric.

T wave negativity in combined Derivations 2 and 3, Figure 5.- The left leg becomes electronegative to both arms. Derivation 1 remains unchanged. The apical zone becomes electronegative to the left upper while the right upper zone becomes iso-electric. This arrangement as in Derivation 1 is a marked change from normal, in that the area of primary electronegativity becomes iso-electric.

T wave negativity in combined Derivations 1, 2, and 3, Figure 6.- All derivations reverse their signs in relation to each other. The apical zone becomes electronegative to the right upper while the left upper zone

becomes iso-electric. The upper zones assume just the opposite relationship to each other that occurs in combined Derivations 2 and 3. Here again the potential distribution is disturbed, the area of primary electronegativity becoming electropositive, approaching the changes accompanying T wave negativity in combined Derivations 1 and 2.

Figures 7 and 8 illustrate the reasons why T wave negativity in Derivation 2 and in combined Derivations 1 and 3 does not occur. In Derivation 2 the left leg would be electronegative to the right arm while the other derivations would remain unchanged. This distribution would imply a diffuse iso-electric cardiac state and would indicate that cardiac contraction had ceased. In combined Derivations 1 and 3 the left arm would be electronegative to the right and the left leg to the left arm. This arrangement likewise would indicate a diffuse iso-electric state.

The greater the deviation from the normal potential distribution the greater the significance of the disorder responsible for the change. The greatest change occurring which is compatible with life is the iso-electric state occupying the right upper zone. Therefore T wave negativity in Derivation 1 and in combined Derivations 2 and 3 should be associated generally with grave heart disease. Next in significance should be those distributions of potential rendering the left upper zone electronegative to the right and represented by T wave negativity in combined Derivations 1 and 2 and in combined Derivations 1, 2, and 3.

In the distribution which most closely approximates normal the T wave is negative in Derivation 3. In these cases the normal potential relationship of the right upper zone is maintained.

Clinical consideration of T wave negativity

This study comprises 1106 cases of T wave negativity in the electrocardiograms. One hundred forty instances (12.6 per cent) were noted in Derivation 1, 688 (62.2 per cent) in Derivation 3, 62 (5.6 per cent) in combined Derivations 1 and 2, 171 (15.5 per cent) in combined Derivations 2 and 3, and 45 (4.1 per cent) in combined Derivations 1, 2, and 3. There was no instance of T wave negativity in Derivation 2 nor in combined Derivations 1 and 3. Patients who had had digitalis within six weeks of the time of electrocardiographic examination were not included in this series.

T wave negativity and ventricular preponderance

In the complete series about half (46.6 per cent) of the electrocardiograms were associated with preponderance of the left ventricle. Preponderance of the right ventricle is evidenced by a negative R wave in Derivation 1 and by a positive R wave in Derivation 3. In cases of preponderance of the left ventricle these changes are reversed. Right preponderance was infrequent (17.3 per cent) and no unbalance was present in 36.1 per cent of the electrocardiograms. Preponderance of the left ventricle occurred most often associated with T wave negativity in Derivation 1 and in combined Derivations 1 and 2. No instance of right preponderance was noted with negativity in combined Derivations 1, 2, and 3. These observations are summarized in Table 1.

Table 1

T WAVE NEGATIVITY AND VENTRICULAR PREPONDERANCE

Derivation	Total	Left preponderance	Percentage	Right preponderance	Percentage	None	Percentage
1	140	120	85.7	5	3.6	15	10.7
3	688	274	39.8	132	19.2	282	41.0
1 and 2	62	52	83.9	4	6.4	6	9.7
2 and 3	171	43	25.2	51	29.8	77	45.0
1, 2, and 3	45	27	60.0	0	0	18	40.0
Total	1106	516	46.6	192	17.3	398	36.1

Table 2

GRAVE HEART DISEASE ASSOCIATED WITH T WAVE NEGATIVITY

Derivation	Total	Arborization block	Delayed auriculo-ventricular conduction	Complete auriculo-ventricular dissociation	Auricular fibrillation	Auricular flutter	Ventricular tachycardia	Aortic disease	Angina pectoris
1	140	53	3	3	19	1	1	16	9
3	688	3	2	0	11	0	0	8	21
1 and 2	62	14	2	0	5	0	1	6	1
2 and 3	171	11	3	1	24	0	1	13	3
1, 2 and 3	45	3	3	0	11	0	0	8	2
Total	1106	84	13	4	70	1	3	51	36

Associated diseases

T wave negativity in Derivation 1. (140 cases).- Myocardial degeneration associated with the hypertension group occurred with greatest frequency (38.6 per cent) in the patients having T wave negativity in Derivation 1 of their electrocardiograms. Three factors are probably responsible for the myocardial changes accompanying hypertension: (1) The cause or causes primarily responsible for the constitutional disorder; (2) the action on the myocardium of the retention products or of the intermediate products of incomplete metabolism or toxic agents resulting from imperfect renal or tissue function, and (3) the increased cardiac work, affecting largely the myocardium, resulting from the hypertension per se and the alterations in cardiovascular balance.

Chronic endocarditis occurred second in order of frequency (22.1 per cent of the cases). The myocardial damage attending this disorder occurs concomitant with or secondary to the endocardial invasion (Table 3). In no instance was the cardiac examination negative. In the majority of instances grave heart disease was present.

Fifty-three patients had arborization block, 3 had delayed auriculoventricular conduction, 3 had complete auriculoventricular dissociation, 19 had auricular fibrillation, 1 had auricular flutter and 1 had ventricular tachycardia. Sixteen patients had aortic disease. Nine had angina pectoris.

The high incidence of grave heart disease in this group verifies my previous statement regarding the potential distribution responsible for this negativity as being the greatest departure from normal. The right upper zone is iso-electric instead of electronegative.

Table 3

ASSOCIATED DISEASES

NEGATIVE T WAVE IN DERIVATION 1

Decade	Total	Degenerative processes			Infections			Local nutritional disturbances			No cardiac findings	Inconclusive cardiac findings.
		Hypertension with and without clinical nephritis	Exophthalmic goiter	Adenomas with hyperthyroidism	Chronic endocarditis	Chronic myocarditis	Syphilis	Arterio-sclerosis	Angina pectoris	Congenital heart disease		
11 - 20	2	0	0	0	2	0	0	0	0	0	0	0
21 - 30	5	0	0	0	4	1	0	0	0	0	0	0
31 - 40	7	1	0	0	3	2	0	0	0	0	0	1
41 - 50	28	10	2	2	7	1	5	1	1	0	0	0
51 - 60	40	16	1	2	6	6	2	7	4	0	0	0
61 - 70	54	25	0	2	9	7	0	11	4	0	0	0
71 - 80	4	2	0	0	0	2	0	0	0	0	0	0
Total	140	54	3	6	31	19	7	19	9	0	0	1
		45.0%			40.7%			13.6%				0.7%

T wave negativity in Derivation 3 (688 cases).- The relative frequency of T wave negativity occurring in Derivation 3 is at once apparent.

The myocardial degeneration secondary to exophthalmic goiter was the most frequently associated condition (19.2 per cent). Myocardial damage due to exophthalmic goiter involves the cellular action of thyroxin (thyroid active principle)[17] on the myocardium[27], and the increased cardiac work accompanying the rise of the basal metabolic rate.

Chronic endocarditis (16.9 per cent) and chronic myocarditis (14.8 per cent) occurred about equally often. By chronic myocarditis I refer to those cases of inflammatory origin in contradistinction to the cases of myocardial degeneration. Myocardial degeneration associated with the hypertension group occurred in only 10.8 per cent of cases (Table 4).

Grave heart disease is relatively infrequent in this group; 3 patients had arborization block, 2 had delayed auriculoventricular conduction, and 11 auricular fibrillation. Eight patients had aortic disease and 21 had angina pectoris. Twenty per cent of the patients had no demonstrable evidence of organic heart disease. Of this number 40 per cent had cardiac neurosis.

The relative infrequency of grave heart disease and the high percentage of apparently normal hearts in this group are in marked contrast to the findings associated with T wave negativity in Derivation 1. These findings are in accord with the hypothetical significance of T wave negativity in Derivation 3. The potential distribution in this T wave negativity is illustrated in Figure 3. The right upper zone remains electronegative, the other zones reverse their potential, establishing an arrangement which does not materially depart from the normal.

Table 4

ASSOCIATED DISEASES

NEGATIVE T WAVE IN DERIVATION 3

Decade	Total	Degenerative processes			Infections			Local nutritional disturbances			No cardiac findings	Inconclusive cardiac findings
		Hypertension with and without clinical nephritis	Exophthalmic goiter	Adenomas with hyperthyroidism	Chronic endocarditis	Chronic myocarditis	Syphilis	Arterio-sclerosis	Angina pectoris	Congenital heart disease		
1 - 10	10	0	0	0	4	0	0	0	0	4	0	2
11 - 20	62	2	20	0	13	2	1	0	0	0	22	2
21 - 30	190	6	55	3	40	19	2	0	0	1	46	18
31 - 40	161	12	39	5	32	15	6	0	0	0	35	17
41 - 50	138	28	16	5	18	25	5	4	4	1	19	17
51 - 60	83	21	2	9	5	27	5	7	6	0	1	6
61 - 70	39	4	0	2	4	12	0	12	10	0	2	3
71 - 80	5	1	0	0	0	2	0	2	1	0	0	0
Total	688	74	132	24	116	102	19	25	21	6	125	65
		33.4%			34.4%			3.7%		0.9%	18.2%	9.4%

The potential distribution occurring in such a relatively high percentage of apparently normal hearts and the frequent transient T wave negativity in Derivation 3 make functional myocardial fatigue a causative possibility in a certain number of cases at least.

T wave negativity in combined Derivations 1 and 2, (62 cases).- Myocardial degeneration associated with the hypertension group occurred in half (50 per cent) of the patients having T wave negativity in combined Derivations 1 and 2. Chronic endocarditis was present in 20.9 per cent of the cases. In no instance was the cardiac examination negative. These findings are summarized in Table 5. About half (46.7 per cent) of the cases were associated with grave heart disease. Fourteen patients had arborization block, 2 had delayed auriculoventricular conduction, 5 had auricular fibrillation, and 1 had ventricular tachycardia. Six patients had aortic disease and one of these, had an aortic aneurysm. One patient had angina pectoris.

Reference to the potential distribution responsible for T wave negativity in this derivation combination (Fig. 4) shows that the normal potential (electronegativity) of the right upper zone is disturbed. The left upper zone becomes electronegative to the right upper. Although this is a distinct departure from normal, it is less marked than those states in which the right upper zone becomes iso-electric.

T wave negativity in combined Derivations 2 and 3,(171 cases).- Chronic endocarditis occurred with greatest frequency (26.9 per cent) in those patients having T wave negativity in combined Derivations 2 and 3. In order of frequency followed myocardial degeneration associated with exophthalmic goiter (21.0 per cent), chronic myocarditis (17.5 per cent), and myocardial degeneration associated with the hypertension group (15.8 per cent).

Table 5

ASSOCIATED DISEASES

NEGATIVE T WAVE IN DERIVATIONS 1 AND 2

Decade	Total	Degenerative processes			Infections			Local nutritional disturbances				
		Hypertension with and without clinical nephritis	Exophthalmic goiter	Adenomas with hyperthyroidism	Chronic endocarditis	Chronic myocarditis	Syphilis	Arterio-sclerosis	Angina pectoris	Congenital heart disease	No cardiac findings	Inconclusive cardiac findings
21 - 30	7	0	0	0	6	1	0	0	0	0	0	0
31 - 40	5	3	0	0	2	0	0	0	0	0	0	0
41 - 50	13	7	0	1	1	3	0	0	0	0	0	1
51 - 60	20	14	1	0	2	1	1	1	1	0	0	0
61 - 70	15	7	0	1	2	1	1	3	0	0	0	0
71 - 80	2	0	0	0	0	1	0	1	0	0	0	0
Total	62	31	1	2	13	7	2	5	1	0	0	1
		54.8%			35.5%			8.1%				1.6%

A large percentage of the patients had grave heart disease. Eleven patients had arborization block, 3 had delayed auriculoventricular conduction, 1 had complete auriculoventricular dissociation, 24 had auricular fibrillation, and 1 ventricular tachycardia. Thirteen patients had aortic disease and 3 had angina pectoris. In 9 cases the cardiac examination was negative. These observations are illustrated in Table 6.

Again the reader is referred to Figure 5, which illustrates the potential distribution in T wave negativity in this derivation combination. The similarity to the potential arrangement attending T wave negativity in Derivation 1 may be noted.

T wave negativity in combined Derivations 1,2,and 3 (45 cases).- T wave negativity in Derivations 1, 2, and 3 constituted the smallest group, comprising only 4.1 per cent of the total series.

Myocardial degeneration associated with the hypertension group occurred most often (35.5 per cent) and in order of occurrence chronic endocarditis (24.4 per cent) and chronic myocarditis (20.0 per cent). Every patient in this group had definite clinical evidence of heart disease (Table 7).

Three of the patients with the graver forms of heart disease had arborization block, 3 delayed auriculoventricular conduction, and 11 auricular fibrillation. Eight patients had aortic disease and 2 had angina pectoris. If the potential distribution in this disorder is taken into consideration the apical zone will be found electronegative to the right upper, while the left upper zone will be iso-electric (Fig. 6). This arrangement is similar to that attending T wave negativity in combined Derivations 1 and 2 in that the right upper zone is electropositive. It likewise is a distinct deviation from normal.

Table 6

ASSOCIATED DISEASES

NEGATIVE T WAVE IN DERIVATIONS 2 AND 3

Decade	Total	Degenerative processes			Infections			Local nutritional disturbances		Congenital heart disease	No cardiac findings	Inconclusive cardiac findings
		Hypertension with and without clinical nephritis	Exophthalmic goiter	Adenomas with hyperthyroidism	Chronic endocarditis	Chronic myocarditis	Syphilis	Arterio-sclerosis	Angina pectoris			
11 - 20	12	1	4	0	5	2	0	0	0	0	0	0
21 - 30	45	1	13	1	16	6	1	0	0	1	3	3
31 - 40	39	4	10	0	14	4	0	0	0	0	5	2
41 - 50	34	5	8	3	6	8	1	0	0	2	1	0
51 - 60	28	10	0	1	4	8	0	4	2	0	0	1
61 - 70	12	5	1	0	1	2	1	2	1	0	0	0
71 - 80	1	1	0	0	0	0	0	0	0	0	0	0
Total	171	27	36	5	46	30	3	6	3	3	9	6
		39.8%			46.2%			3.5%		1.7%	5.3%	3.5%

Table 7

ASSOCIATED DISEASES

NEGATIVE T WAVE IN DERIVATIONS 1, 2, AND 3

Decade	Total	Degenerative processes			Infections			Local nutritional disturbance			No cardiac findings	Inconclusive cardiac findings
		Hypertension with and without clinical nephritis	Exophthalmic goiter	Adenomas with hyperthyroidism	Chronic endocarditis	Chronic myocarditis	Syphilis	Arterio-sclerosis	Angina pectoris	Congenital heart disease		
11 - 20	1	0	0	0	1	0	0	0	0	0	0	0
21 - 30	4	0	0	0	2	1	0	0	0	1	0	0
31 - 40	7	1	0	0	4	1	1	0	0	0	0	0
41 - 50	9	2	1	2	0	3	1	0	0	0	0	0
51 - 60	12	8	0	0	2	0	0	2	1	0	0	0
61 - 70	12	5	0	0	2	4	0	1	1	0	0	0
Total	45	16	1	2	11	9	2	3	2	1	0	0
		42.2%			48.8%			6.6%		2.2%		

Table 8

ELECTROCARDIOGRAPHIC CHANGES ASSOCIATED WITH T WAVE NEGATIVITY

Derivation	Total	T wave negativity unattended	Sinus arrhythmia	Auricular premature contractions	Auricular and nodal premature contractions	Auricular and ventricular premature contractions	Nodal premature contractions	Nodal premature contractions and sinus arrhythmia	Ventricular premature contractions	Ventricular and nodal premature contractions	Ventricular premature contrations and sinus arrhythmia	Auricular fibrillation	Auricular fibrillation and ventricular premature contractions	Auricular fibrillation and aberrant Q R S complexes in isolated derivations	Auricular fibrillation and aberrant Q R S complexes in isolated derivations and ventricular premature contractions	Arborization block	Arborization block and auricular premature contractions	Arborization block and ventricular premature contractions	Arborization block and auricular fibrillation	Arborization block and delayed auriculoventricular conduction	Aberrant Q R S complexes in isolated derivations	Aberrant Q R S complexes in isolated derivations and auricular premature contractions	Aberrant Q R S complexes in isolated derivations and ventricular premature contractions	Delayed auriculoventricular conduction	Delayed auriculoventricular conduction and sinus arrhythmia	Delayed auriculoventricular conduction, aberrant Q R S complexes in isolated derivations and ventricular premature contractions	Complete auriculoventricular dissociation	Auricular flutter	Nodal tachycardia	Ventricular tachycardia
1	140	41	0	2	0	6	0	0	11	0	0	8	6	1	0	36	3	8	4	2	2	1	2	1	0	0	3	1	1	0
3	688	550	24	14	1	8	4	1	40	0	1	17	4	0	1	10	0	2	0	0	10	0	1	0	0	0	0	0	0	0
1 and 2	62	32	0	2	0	2	1	0	2	1	0	3	1	0	0	11	0	1	1	1	2	0	0	1	0	0	0	0	0	0
2 and 3	171	112	1	5	0	1	1	0	10	1	0	20	4	0	0	4	0	5	1	1	1	0	0	1	1	0	1	0	0	0
1, 2 and 3	45	21	0	2	0	0	0	0	4	0	0	8	3	0	0	2	0	1	0	0	0	0	0	0	2	0	1	0	0	0
Total	1106	756	25	25	1	17	6	1	67	2	1	56	18	1	1	63	3	17	6	4	16	1	3	5	1	1	4	1	1	3

Table 9

VALVULAR DISEASE ASSOCIATED WITH T WAVE NEGATIVITY

Derivation	Total	Mitral disease						Aortic disease						
		Mitral regurgitation	Mitral stenosis	Double mitral lesion	Mitral and polyvalvular regurgitation	Double mitral lesion and aortic regurgitation	Mitral stenosis and aortic regurgitation	Aortic regurgitation	Aortic stenosis	Double aortic lesion	Aortitis	Aortic regurgitation and aortitis	Aortic and polyvalvular regurgitation	Aortic and mitral regurgitation
1	37	16	0	2	1	2	0	3	2	2[f]	2[ab]	5[cd]	2[e]	0
3	122	64	27	7	0	2	0	10[ghi]	1	2	1[l]	2[jk]	0	6
1 and 2	14	5	0	1	1	1	1	2	1	2[m]	0	0	0	0
2 and 3	49	22	7	5	2	0	0	4[n]	1	3	2[o]	1[p]	2	0
1, 2 and 3	12	4	0	0	0	0	0	3	0	2	0	3[q]	0	0
Total	234	111	34	15	4	5	1	22	5	11	5	11	4	6

Note: a, b, c, etc. = syphilitic lesions

Cardiac mortality and T wave negativity

T wave negativity in Derivation 1.- Information has been received concerning 117 patients having T wave negativity in Derivation 1 of their electrocardiograms. Seventy-eight (66.6 per cent) have died from heart disease during a period of four and one half years(Table 10). The mortality in every decade is high; the lowest percentage (45.8) occurred between the ages of 41 and 50. Thirty-three patients are alive and 10 of these report their conditions worse, 15 are improved,and 8 are unchanged. None of the patients was without cardiac complaint.

The high cardiac mortality in this group is in accord with the hypothetical and clinical significance, previously emphasized, which attends this T wave negativity. To prevent misunderstanding, it may be stated that the negative T wave per se is only the manifestation of serious underlying myocardial disorder. Changes in cardiac function affecting contraction preponderance, resulting from organic or functional myocardial fatigue, alter electropotential, which produces T wave negativity.

T wave negativity in Derivation 3.- Information has been received concerning 487 patients having T wave negativity in Derivation 3 of their electrocardiograms. Forty-six (9.4 per cent) have died from heart disease during a period of four and one half years. The data are summarized in Table 11. This relatively low cardiac mortality is sharply contrasted with the mortality of the foregoing group. It is in agreement, however, with the hypothetical and clinical significance accorded T wave negativity in Derivation 3. The potential distribution producing this negativity is but a slight departure from

Table 10

CARDIAC MORTALITY

T WAVE NEGATIVITY IN DERIVATION 1

Decade	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
11 - 20	2	2	2	0	1	50.0	1	0	1	0	0
21 - 30	5	5	3	2	3	60.0	2	0	0	2	0
31 - 40	7	6	6	0	4	66.6	2	1	1	0	0
41 - 50	28	24	13	11	11	45.8	11	2	6	3	0
51 - 60	40	32	22	10	21	65.6	9	2	5	2	0
61 - 70	54	44	33	11	35	79.5	8	5	2	1	0
71 - 80	4	4	4	0	3	75.0	0	0	0	0	0
Total	140	117	83	34	78	66.6	33	10	15	8	0

Table 11

CARDIAC MORTALITY

T WAVE NEGATIVITY IN DERIVATION 3

Decade	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
1- 10	10	8	3	5	2	25.0	5	0	3	2	3
11 -20	62	42	9	33	2	4.7	40	7	20	13	17
21- 30	190	139	22	117	5	3.5	127	30	53	44	37
31- 40	161	113	27	86	9	7.9	99	23	33	43	25
41- 50	138	89	24	65	9	10.1	78	18	30	30	20
51- 60	83	68	37	31	13	19.1	53	13	18	22	3
61- 70	39	24	15	9	5	20.8	16	6	3	7	1
71- 80	5	4	3	1	1	25.0	2	0	1	1	0
Total	688	487	140	347	46	9.4	420	97	161	162	106

Table 12

CARDIAC MORTALITY

T WAVE NEGATIVITY IN COMBINED DERIVATIONS 1 AND 2

Decade	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
21 - 30	7	5	5	0	4	80.0	1	0	0	1	0
31 - 40	5	3	3	0	3	100.0	0	0	0	0	0
41 - 50	13	11	9	2	7	63.6	3	0	2	1	1
51 - 60	20	19	13	6	12	63.1	7	1	5	1	0
61 - 70	15	12	8	4	7	58.3	5	0	5	0	0
71 - 80	2	2	1	1	2	100.0	0	0	0	0	0
Total	62	52	39	13	35	67.3	16	1	12	3	1

normal. Four hundred twenty patients are alive, 97 report their conditions worse, 161 are improved, and 162 are unchanged. One hundred six patients report no cardiac complaints.

T.wave negativity in combined Derivations 1 and 2.- Of the 52 patients having T wave negativity in combined Derivations 1 and 2 concerning whom we have heard, 35 (67.3 per cent) have died from heart disease during four and one half years. This mortality is greater than was anticipated in that the potential distribution resulting in this T wave negativity was not the greatest departure from normal. In Figure 4, in which the potential distribution is represented, the right upper zone becomes electropositive to the left, instead of electronegative as in the normal, but this is a lesser departure than iso-electric. The fact that the group is relatively small may be a factor in obtaining a high cardiac mortality. Sixteen patients are alive, 1 reports his condition worse, 12 are improved, and 3 are unchanged. Only one patient reports no cardiac complaints.

T wave negativity in combined Derivations 2 and 3.- We have learned of the condition of 135 patients having T wave negativity in combined Derivations 2 and 3. Thirty-five (25.9 per cent) have died from heart disease during a period of four and one half years (Table 13). In contradistinction to the foregoing group, the mortality was lower than expected from a hypothetical consideration of potential distribution and associated heart disease. A possible explanation for this discrepancy rests in the fact that 23.3 per cent of the patients were examined during the last year of the series, and the time element, therefore, is too short to embrace a true mortality average. Eighty-eight patients are alive, 25 report their conditions worse, 37 are

Table 13

CARDIAC MORTALITY

T WAVE NEGATIVITY IN COMBINED DERIVATIONS 2 AND 3

Decade	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
11 - 20	12	9	0	9	0	0	9	2	4	3	2
21 - 30	45	34	11	23	9	26.4	22	7	10	5	6
31 - 40	39	32	9	23	5	15.6	24	7	9	8	7
41 - 50	34	27	16	11	8	29.6	17	4	7	6	1
51 - 60	28	20	15	5	6	30.0	12	2	7	3	0
61 - 70	12	12	9	3	6	50.0	4	3	0	1	0
71 - 80	1	1	1	0	1	100.0	0	0	0	0	0
Total	171	135	61	74	35	25.9	88	25	37	26	16

improved, and 26 are unchanged. Sixteen patients report no cardiac complaints; 7 of these had had thyroidectomies for hyperthyroidism and were cured.

T wave negativity in combined Derivations 1, 2, and 3.- Patients having T wave negativity in all derivations comprised a relatively small group. Nineteen of the 38 (50.0 per cent) on whom we have had reports, have died from heart disease during four and one half years. (Table 14). This mortality agrees fairly well with hypothetical considerations of potential distribution and associated grave heart disease (Fig. 6). Fifteen patients are alive, 5 report their conditions worse, 8 are improved, and 2 are unchanged. No patient was without cardiac complaint.

In the complete series of T wave negativity, regardless of derivation grouping, information has been received concerning 829 patients. Two hundred thirteen (25.6 per cent) have died from heart disease (Table 15).

A group of cases was compiled including those cases in which there was T wave negativity without other electrocardiographic abnormalities except ventricular preponderance. This was done with the idea of excluding disorders of cardiac action known in themselves materially to influence cardiac mortality, especially auricular fibrillation and flutter, ventricular tachycardia, delayed auriculoventricular conduction, complete auriculoventricular dissociation, and arborization block. Of the 559 patients in this group of whose condition we have learned, 92 (16.4 per cent) have died from heart disease (Table 15). The cardiac mortality in this group is 9.2 per cent less than that of the complete series.

Table 14

CARDIAC MORTALITY

T WAVE NEGATIVITY IN COMBINED DERIVATIONS 1, 2 AND 3

Decade	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
11 - 20	1	1	0	1	1	100.0	0	0	0	0	0
21 - 30	4	2	2	0	1	50.0	1	0	1	0	0
31 - 40	7	5	3	2	2	40.0	2	0	1	1	0
41 - 50	9	9	6	3	1	11.1	6	0	5	1	0
51 - 60	12	10	10	0	9	90.0	1	1	0	0	0
61 - 70	12	11	8	3	5	45.5	5	4	1	0	0
Total	45	38	29	9	19	50.0	15	5	8	2	0

Table 15

CARDIAC MORTALITY IN COMPLETE SERIES

Derivation	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
1	140	117	83	34	78	66.6	33	10	15	8	0
2	688	487	140	347	46	9.4	420	97	161	162	106
1 and 2	62	52	39	13	35	67.3	16	1	12	3	1
2 and 3	171	135	61	74	35	25.9	88	25	37	26	16
1, 2 and 3	45	38	29	9	19	50.0	15	5	8	2	0
Total	1106	829	352	477	213	25.6	572	138	233	201	123

Table 16

CARDIAC MORTALITY

T WAVE NEGATIVITY WITHOUT OTHER ELECTROCARDIOGRAPHIC ABNORMALITIES

Derivation	Total	Patients heard from	Males	Females	Cardiac deaths	Percentage	Living	Worse	Improved	Unchanged	No cardiac complaints
1	41	32	21	11	17	53.1	13	4	7	2	0
3	550	394	86	308	33	8.3	349	76	132	141	83
1 and 2	32	29	23	6	17	58.6	12	1	8	3	1
2 and 3	112	86	33	53	17	19.7	61	18	28	15	14
1,2 and 3	21	18	15	3	8	44.4	7	2	4	1	0
Total	756	559	178	381	92	16.4	442	101	179	162	98

Conclusions

1. The ventricular complexes of the electrocardiogram are the expressions of two distinct processes, impulse conduction, and muscle contraction.

2. The T wave is the expression of preponderance of contraction on one side of the line of equipotential (Eyster and Meek).

3. T wave negativity results from alteration in potential distribution from changes in contraction preponderance. Changes in contraction preponderance may result from changes in blood volume, and from organic or functional myocardial fatigue.

4. The occurrence of T wave negativity in certain isolated and combined derivations of the electrocardiogram bears a fairly definite relationship to degrees of cardiac damage.

5. The significance of T wave negativity in this series as evidence of heart disease in order of gravity is in (1) combined Derivations 1 and 2, (2) Derivation 1, (3) combined Derivations 1, 2, and 3, (4) combined Derivations 2 and 3, and (5) Derivation 3.

6. T wave negativity in Derivation 2 and in combined Derivations 1 and 3 has not been observed in the Mayo Clinic, and according to the hypothesis cannot exist. Such negativity would indicate a diffuse iso-electric state of the heart which would mean cardiac standstill.

BIBLIOGRAPHY

1. Barker, L.F.: Electrocardiography and phonocardiography. Bull. Johns Hopkins Hosp., 1910, xxi, 358-389.

2. Brandenburg, K. and Hoffmann, P.: Forschungsergebnisse aus Medizin und Naturwissenshaft. Wo entstehen die normalen Bewegungsreize im Warmbluterherzen und welche Folgen für die Schlagfolge hat ihre reizlose Ausschaltung? Med. Klin., 1912, viii, 16-21.

3. Carter, E.P.: Further observations on the aberrant electrocardiogram associated with sclerosis of the atrioventricular bundle branches and their terminal arborizations. Clinical and histologic report of a case in which such aberrant complexes were obtained. Arch. Int. Med., 1918, xxii, 331-353.

4. Cohn, A.E., Fraser, F.R., and Jamieson, H.A.: The influence of digitalis on the T wave of the human electrocardiogram. Jour. Exper. Med., 1915, xxi, 593-604.

5. Dale, Dorothy and Mines, G.R.: The influence of nerve stimulation on the electrocardiogram. Jour. Physiol., 1913, xlvi, 319-336.

6. Einthoven, W.: Le telecardiogramme. Arch. internat. de physiol., 1906, iv, 132-165.

7. Einthoven, W., Flohil, A. and Battaerd, P.J.T.A.: Die Registrierung der menschlichen Herztöne mittels des Saitengalvonometers. Arch. f. d. ges. Physiol., 1907, cxvii, 461-472.

8. Eppinger, H. and Rothberger, E.J.: Zur Analyse des Elektrokardiogramms. Wien. klin. Wchnschr., 1909, xxii, 1091-1098.

9. Eyster, J.A.E. and Meek, W.J.: The interpretation of the normal electrocardiogram. A critical and experimental study. Arch. Int. Med., 1913, xi, 206-247.

10. Frank, O. and Hess, O.: Ueber das Cardiogramm und dan ersten Herzton. Verhandl. d. Kong. f. innere Med., 1908, xxv, 283-291.

11. Ganter, G. and Zahn, A.: Über Reizbildung und Reizleitung im Saugetierherzen in ihrer Beziehung zum spezifischen Muskelgewebe. Zentralbl. f. Physiol., 1911, xxv, 782-784.

12. Hoffmann, A.: Zur Deutung des Elektrokardiogrammes. Arch. f.d.ges.Physiol., 1910, cxxxiii, 552-578.

13. James, W.B. and Williams, H.B.: The electrocardiogram in clinical medicine. I. The string galvanometer and the electrocardiogram in health. Med. and Surg. Report of Presbyterian Hosp. New York City, 1912, ix, 17-59.

14. Kahn, R.H.: Beiträge zur Kenntniss des Elektrokardiogrammes. Arch. f. d. ges Physiol., 1909, cxxvi, 197-224.

15. Kahn, R.H.: Weitere Beiträge zur Kenntniss des Elektrokardiogrammes. Arch. f.d.ges. Physiol., 1909, cxxix, 291-328.

16. Keith, A. and Flask, M. The form and nature of the muscular connections between the primary divisions of the vetebrate heart. Jour. Anat. and Physiol., 1906-1907, xli, 172-189.

17. Kendall, E.C.: The active constituent of the thyroid, its isolation, chemical nature, and physiologic action. Collected Papers of The Mayo Clinic. Philadelphia, Saunders, 1916, viii, 513-527.

18. Kraus, F. and Nicolai, G.: Ueber das Elektrokardiogramm des gesunden und kranken Menschen. Leipzig, 1910.

19. Krumbhaar, E.B.: The electrocardiograph in diagnosis and prognosis. New York Med. Jour., 1916, cvi, 1065-1069.

20. Krumbhaar, E.B. and Jenks, H.H.: Electrocardiographic studies in normal infants and children. Heart, 1917, vi, 189-197.

21. Lewis, T.: Clinical electrocardiography. London, Shaw, 1913, p.20.

22. Lewis, T., Oppenheimer, B.S., and Oppenheimer, Adele.: The site of origin of the mammalian heart-beat. The pacemaker in the dog. Heart, 1910-1911, ii, 147-169.

23. Morison, R.A.: Changes in the electrocardiogram due possibly to alterations in blood volumes. Proc. Soc. Exper. Biol. and Med., 1917, xiv, 69.

24. Oppenheimer, B.S. and Rothschild, M.A.: Electrocardiographic changes associated with myocardial involvement and with special reference to prognosis. Jour.Am. Med.Assn.,1917, lxix, 429-431.

25. Pardee, H.E.B.: The field of the various methods of cardiac diagnosis. New York Med. Jour., 1917, cvi, 1065-1069.

26. Piper, H.: Die Blutdruckschwankungen in den Hohlräumen des Herzens und in den grossen Gefässen. Arch. f. Anat. u. Physiol.,1912, 343-383.

27. Plummer, H.S.: Personal communication to the author.

28. Rothberger, J. and Winterberg, H.: Ueber die Beziehungen der Herznerven zur Form des Elektrokardiogramms. Arch.f.d.ges. Physiol., 1910, cxxxv, 506-558.

29. Samojloff, A.: Weitere Beiträge zur Elektrophysiologie des Herzens. Arch. f. d. ges. Physiol., 1910, cxxxv, 417-468.

30. Smith, F.M.: The ligation of the coronary arteries with electrocardiographic study. Arch. Int. Med., 1918, xxii, 8-27.

31. Smith, S.C.: The clinical value of electrocardiography. Penn. Med.Jour., 1917, xxi, 10-18.

32. Waller, A.D. and Reid, E.W.: On the action of the excised mammalian heart. Phil. Tr. Roy. Soc. London, 1887, clxxviii, 215-256.

33. Weber, A. and Wirth, A.: Zur Registrierung der Hertztöne nach O.Frank . Deutsch. Arch. f. klin. Med., 1912, cv, 562-575.

34. Wedd, A.M.: The clinical significance of slight notching of the R wave of the electrocardiogram. Arch. Int. Med.,1919, xxiii, 515-526.

35. Weiss, O. and Joachim, G.: Registrierung und Reproduktion menschlicher Hertztöne und Herzgeräusche. Arch. f.d.ges. Physiol., 1908, cxxiii, 341-386.

36. Wiggers, C.J.: The contour of the pressure curve in the pulmonary artery. Am.Jour. Physiol.,1914, xxxiii, 1-12.

37. Wiggers, C.J.: Modern aspects of the circulation in health and disease. Philadelphia, Lea and Febiger, 1915, 376 pp.

38. Willius, F.A.: Arborization block. Arch. Int. Med., 1919, xxiii, 431-440.

39. Willius, F.A.: Changes in the Q R S complex in isolated derivations of the human electrocardiogram. Arch. Int. Med. (In press).

40. Wybauw, R.: Sur le point de la systole cardiaque dan l'oreillette droite. Arch. internat. de physiol., 1910, x, 78-89.

ARBORIZATION BLOCK

F. A. WILLIUS, M.D.
MAYO CLINIC, ROCHESTER, MINN.

ARBORIZATION BLOCK

F. A. WILLIUS, M.D.
MAYO CLINIC, ROCHESTER, MINN.

Arborization block or impaired intraventricular conduction is dependent on graphic records for its recognition. It is now generally accepted to indicate disease of the subendocardial myocardium[1] and evidences serious functional cardiac disturbance.[2] The involvement occurs in the subendocardial or Purkinje plexus.

The deflections constituting the initial ventricular complex of the electocardiogram are termed Q, R, S, and indicate the passage of the electrical impulse through the main divisions of the auriculoventricular bundle and their arborizations. These deflections comprise a graphic record which is upright in all leads, is abruptly pointed, and has a narrow base. The normal base width does not exceed 0.10 second.[3]

Arborization block is recognized by abnormal deviations of the Q, R, S group. These are increased width, notching of the apex, and splintering of the ascending and descending limbs.

The bizarre complex of arborization block is probably due either to impulse transmission through circuitous and aberrant paths or to delayed transmission through normal channels. Experimental and clinical evidence supports the former view. The abnormal complex, constituting the ventricular premature contraction (extrasystole), is well recognized, as is the complex of ventricular tachycardia and the idioventricular complex of complete auriculoventricular dissociation, which simulate the notched and widened Q, R, S group of arborization block. These we know result from ectopic stimuli which arise somewhere in the ventricular musculature and traverse aberrant paths to provoke ventricular systoles. The constancy in form of the deflections of the normal electrocardiogram make the abnormal complexes stand out as striking entities.

The present study was undertaken to determine, if possible, the significance of this disordered mechanism with especial reference to life expectancy.

---

1. Carter, E. P.: Further Observations on the Aberrant Electrocardiogram Associated with Sclerosis of the Atrioventricular Bundle Branches and Their Terminal Arborizations, Arch. Int. Med. **22**:331, 1918.

1. Oppenheimer, B. S., and Rothschild, M. A.: Electrocardiographic Changes Associated with Myocardial Involvement, J. A. M. A. **69**:429, 1917.

2. Robinson, G. C.: The Relation of Changes in the Form of the Ventricular complex of the Electrocardiogram to Functional changes in the Heart, Arch. Int. Med. **18**:830, 1916.

3. Lewis, T.: Clinical Electrocardiography, London, Shaw, 1913, p. 32.

One hundred and thirty-eight patients with arborization block have been examined. The electrocardiographic requirements warranting this diagnosis were, (1) notching of the apex R, (2) splintering of the ascending or descending limb, and (3) in complexes of normal contour, a base width exceeding 0.10 second. These changes are summarized in Table 1.

The electrocardiograms illustrate the types represented (Figs. 1 to 10). The tension of the galvanometer fiber influences the width of the unaltered complex; a loose fiber is capable of giving an increased base width.[4]

The disorders responsible for the development of subendocardial myocardial disease are, (1) infections, (2) degenerative processes, and (3) local nutritional disturbances. These observations are summarized in Table 2.

TABLE 1.—Q, R, S—Complex Changes

Decade	0.06 Sec.				0.07 Sec.				0.08 Sec.				0.09 Sec.				0.10 Sec.				0.11 Sec.				0.12 Sec.				0.13 Sec.				0.14 Sec.				0.15 Sec.				0.16 Sec.				0.17 Sec.				0.18 Sec.				Cases
	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	Unaltered	Notched	Splintered	Notched and Splintered	
11-20	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	1	0	0	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2
21-30	0	0	0	0	0	0	0	0	0	1	2	0	0	1	2	0	0	0	2	0	0	0	1	0	0	1	2	2	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10
31-40	0	0	1	0	0	1	1	0	0	1	2	0	0	2	1	0	0	1	3	0	0	0	1	0	0	3	4	2	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8
41-50	0	0	0	0	0	1	0	0	0	2	2	0	0	1	3	0	0	2	2	0	0	0	1	0	0	4	2	2	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	21
51-60	0	0	0	0	0	1	1	0	0	2	4	0	0	1	0	0	0	1	2	0	0	0	1	0	0	4	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	41
61-70	0	0	0	0	0	0	0	0	0	2	5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	20
71-80	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7
																																																			Total	138

Endocarditis was the most frequent causative disorder, and occurred in forty-nine of the 138 cases (35.5 per cent.). Its predominance in the earlier decades of life was anticipated; degenerative and local nutritional disturbances dominate the later decades. In order of frequency are cardiovascular-renal disease with hypertension, thyrotoxic adenomas and arteriosclerosis. Exophthalmic goiter occurred in five cases. Only four proved cases of lues were found. In twenty-seven instances no tangible histories or findings suggesting causative factors were obtained.

Exertion dyspnea was a complaint in all cases, and in thirty-one (22.5 per cent.) orthopnea was a dominant symptom. Palpitation on exertion was present in forty-seven instances (34 per cent.). Twenty-two patients (15.9 per cent.) had angina pectoris and in five of these this occurred in aortic disease. Edema of the lower extremities varying

4. Hirschfelder, A. D.: Personal communication to the author.

from slight pitting in most instances to definite swelling with glazed skin in a smaller number, was present in forty-two patients (30.4 per cent.). Only five cases of general anasarca are recorded in this series. Of the edema cases twenty-four (57.1 per cent.) occurred in patients with endocardial valvular disease. The relative infrequency of edema in grave heart disease is very interesting, and emphasizes the importance of adjunct methods in the thorough examination of patients suffering from cardiac disease.

Objectively, the striking feature present in practically all of the cases is the lack of definition of the heart sounds. They are muffled, the normal differentiation between the first and second sounds is absent, and the auscultatory findings of embryocardia are simulated. There was an increase in cardiac dulness in most of our cases, both to the right and to the left of the midsternal line. Auricular fibrillation was present in eighteen cases (13 per cent.), and occurred except in one instance in the later decades of life. Four patients had delayed auriculoventricular conduction, that is P-R intervals exceeding 0.22 second. The deflection amplitudes of the Q, R, S group showed that sixty-four patients (46.4 per cent.) had normal values (10 to 15 millivolts), sixty-one patients (44.2 per cent.) had high values, the greatest 39 millivolts, and thirteen patients (9.4 per cent.) had low values, the lowest of which was 5 millivolts. Deflections of high amplitude, largely diphasic, are believed by Carter to be indicative of a definite, totally obstructive, temporary or permanent lesion of one of the branches of the auriculoventricular bundle; those of low amplitude suggest diffuse sclerosis, although they do not preclude localized lesions of the main branch and its arborizations.

No striking changes in amplitude of the final T-wave of the ventricular complex were noted. This wave was negative in eighty-five

cases (63 per cent.), and occurred most frequently in Lead 1 alone, in forty-two cases (49.4 per cent.). Table 3 shows the T-wave negatively in this series. The inversion of the T-wave in Lead 1 is significant, I believe, and of itself indicative of myocardial changes, for in all the electrocardiograms studied in which this observation was noted,

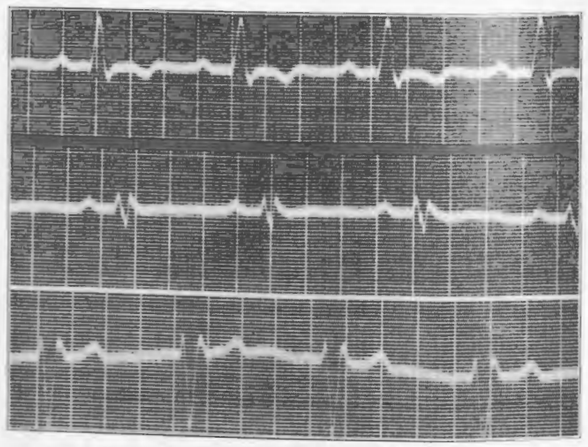


Fig. 1.—Rate 65. Q, R, S complex widened in Leads 1 and 3 0.11 sec. and splintered in Lead 2. Inverted T-wave, Lead 1. Left ventricular preponderance. Case 160997.

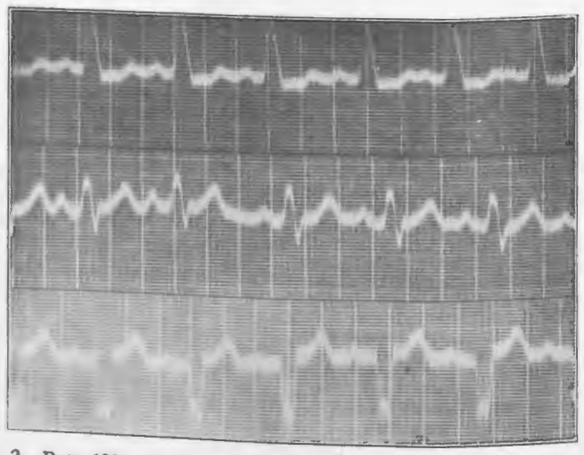


Fig. 2.—Rate 100. Q, R, S complex notched and widened 0.12 sec. Left ventricular preponderance. Case 213193.

the patients presented definite clinical evidence of myocardial insufficiency, except in one case, in which the conclusions were indefinite.

One hundred and twelve patients with arborization block have been heard from in answer to letters of inquiry. Seventy-eight (69.6 per cent.) of these have died; all except three died of heart disease. The average duration of life from the time of examination was eight

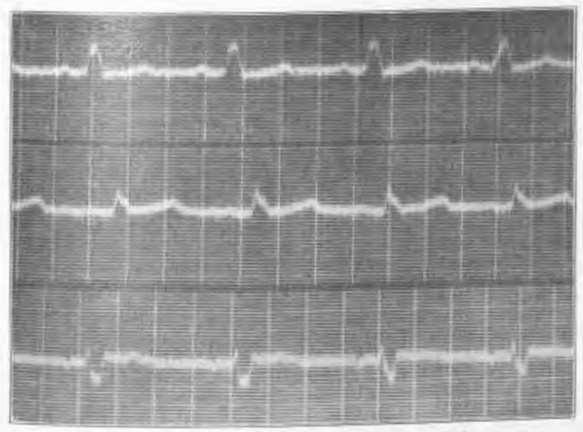


Fig. 3.—Rate 66. Q, R, S complex notched. Left ventricular preponderance. Case 154767.

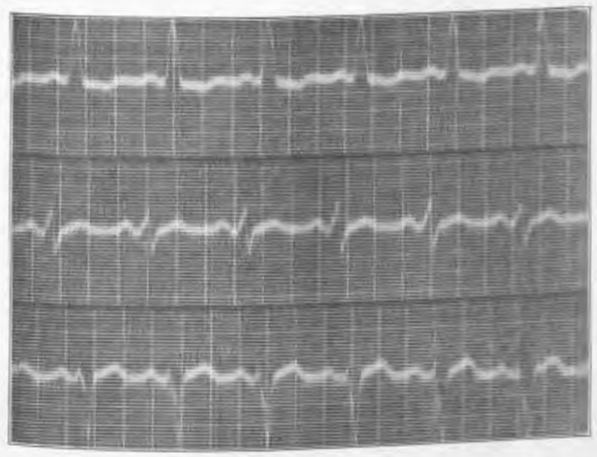


Fig. 4.—Rate 82. Q, R, S complex splintered and widened 0.11 sec. Left ventricular preponderance. Case 165664.

and one-half months. These statistics bear out the presumption that arborization block is a grave disorder. It is well recognized that disease involving the conduction system is a serious menace to life, but arborization block is attended by an earlier mortality than that caused by the lesions higher up. As life is directly dependent on ventricular action, any impairment of ventricular function is grave. The deaths are summarized in Table 4.

Thirty-four patients of the series are known to be alive; of these, seventeen are worse, four of them bed-ridden; nine report their conditions unchanged and eight report some improvement. We were

TABLE 2.—Etiologic Diseases

Decade	Endocarditis	[illegible]	Cardiovascular-Renal	Percentage	Thyrotoxic Adenomas	Percentage	Exophthalmic Goiter	Percentage	Arteriosclerosis	Percentage	Syphilis	Percentage	No Etiologic History
11–20	2	100.0	0	0	0	0	0	0	0	0	0	0	0
21–30	8	80.0	1	10.0	0	0	1	10.0	0	0	0	0	0
31–40	5	62.5	0	0	0	0	0	0	0	0	1	12.5	2
41–50	14	45.2	10	32.3	0	0	1	3.2	0	0	2	6.5	6
51–60	10	24.4	14	34.1	6	14.6	2	4.8	0	0	1	2.4	9
61–70	8	20.5	17	43.6	2	5.1	1	2.6	2	5.1	0	0	9
71–80	2	28.6	2	28.6	1	14.2	0	.0	1	14.2	0	0	1
	49		44		9		5		3		4*		27

* Three cases under syphilis classified under endocarditis.

TABLE 3.—T-Wave Negativity

Decades	Lead 1	Lead 2	Lead 3	Leads 1 and 2	Leads 2 and 3	Leads 1, 2 and 3	Total
11–20	1	0	0	0	0	0	1
21–30	0	1	2	2	1	0	6
31–40	1	0	3	1	0	0	5
41–50	8	0	1	2	2	0	13
51–60	14	0	3	3	6	1	27
61–70	16	0	3	5	1	4	29
71–80	2	0	0	1	1	0	4
	42	1	12	14	11	5	85

TABLE 4.—Summary of Deaths

Decade	Total Cases	Deaths
11–20	2	1
21–30	10	3
31–40	8	5
41–50	31	16
51–60	41	24
61–70	39	23* †
71–80	7	6*
Total	138	78

* Died of cancer. † Died of pneumonia.

afforded the opportunity of five necropsies; the cardiac findings are appended. No definite localizing lesions were found, but rather diffuse degenerative processes involving the myocardium.

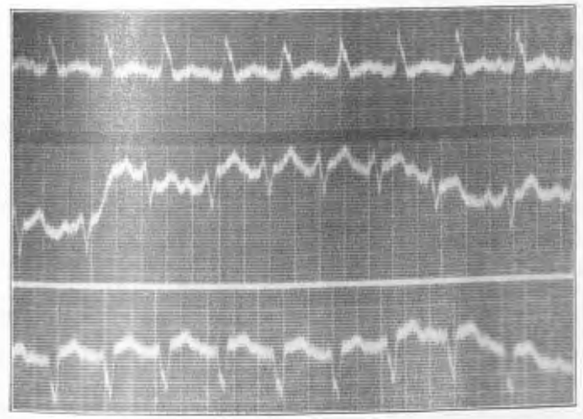


Fig. 5.—Rate 112. Q, R, S complex splintered and widened 0.12 sec. Left ventricular preponderance. Case 176302.

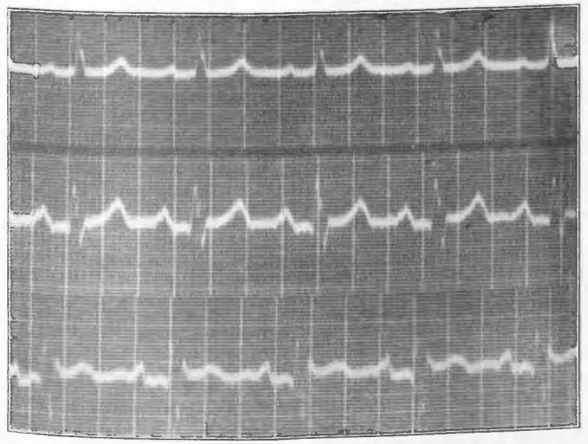


Fig. 6.—Rate 75. Q, R, S complex splintered 0.08 sec. Left ventricular preponderance. Case 162663.

Case 1 (130119).—Very marked fatty changes in the myocardium; marked dilatation of the aortic, mitral and tricuspid valvular rings of the heart; moderate nodular fibrous and fatty thickening of the lining of the aorta, and of the aortic and mitral leaflets of the heart; marked thinning of the myo-

cardium of the left ventricle; marked dilatation and engorgement of all of the chambers of the heart; moderate hydropericardium. Histologic Findings: Fragmentation and slight fatty changes.

Case 2 (147045).—Obliterative fibrous adhesive pericarditis; marked nodular sclerosis and fatty changes in the lining of the aorta and its main

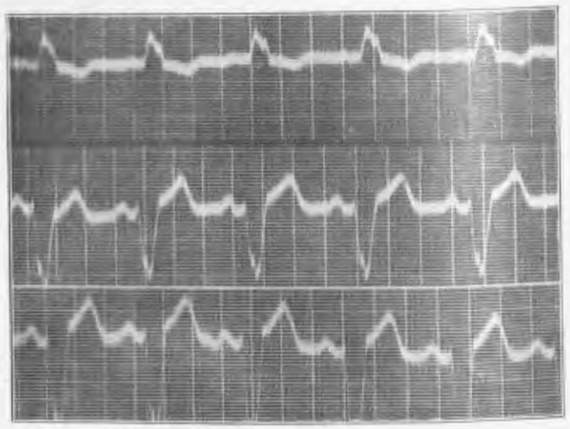


Fig. 7.—Rate 82. Q, R, S complex notched and widened 0.14 sec. Inverted T-wave, Lead 1. Left ventricular preponderance. Case 216281.

Fig. 8.—Rate 115. Q, R, S complex splintered and widened 0.16 sec. Inverted T-wave Lead 1. Left ventricular preponderance. Case 143010.

branches; marked calcareous sclerosis of the coronary arteries; huge spontaneous thrombosis of the dependent portion of the left ventricle; marked hypertrophy of the myocardium of the left ventricle; marked dilatation of all the chambers of the heart; moderate dilatation of the aortic and mitral valvular rings; marked diffuse thickening of the pulmonary artery. Histologic Findings:

The pericardium was thickened and adherent to the heart. There was a marked replacement of the heart muscle by fibrous tissue. Toward the lower portion was seen hyalinization of the muscle. The thrombus was made up of fibrin and in places showed a slight infiltration of leukocytes. With the fat stain considerable fat was found in the thrombus.

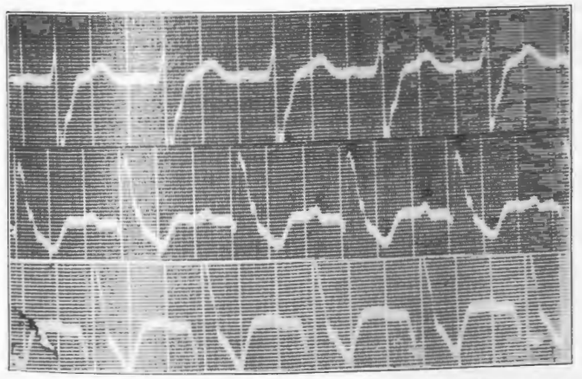


Fig. 9.—Rate 86. Q, R, S complex notched, splintered and widened 0.12 sec. Inverted T-wave, Leads 2 and 3. Right ventricular preponderance Case 142757.

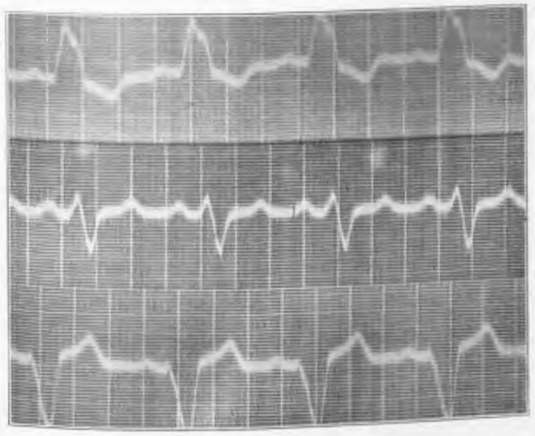


Fig. 10.—Rate 75. Q, R, S complex notched and widened 0.16 sec. Inverted T.-wave, Lead 1. Left ventricular preponderance. Case 154081.

Case 3 (161776).—Marked fatty degeneration of the myocardium; marked hydropericardium; moderate dilatation of the heart. Histologic Findings: In the heart there were marked fatty degeneration and fragmentation; moderate increase in fibrous connective tissue and hypertrophy of the muscle.

Case 4 (189701).—Acute dilatation of the heart; marked fatty and fibrous sclerosis of the lining of the aorta and of the aortic and mitral valvular leaf-

lets; petechial hemorrhages in the visceral pericardium. Histologic Findings: Moderate diffuse fatty degeneration of the myocardium. Aortitis probably luetic; fibrous and fatty changes in the intima and media, with round cell infiltration.

CASE 5 (197468).—Marked fatty and fibrous diffuse parenchymatous myocarditis; marked hypertrophy of the myocardium of the left ventricle; marked dilatation of all the chambers and valvular rings of the heart; spontaneous mural thrombosis of the left ventricle; slight hydropericardium. Histologic Findings: Fatty and fibrous degeneration of the myocardium.

SUMMARY

1. Arborization block is a grave disorder of the cardiac mechanism; it entails a large and early mortality (69.6 per cent.), in an average duration of eight and one-half months.

2. Disorders responsible for the development of this condition were found to be, in order of frequency, (1) infections, (2) degenerative processes, and (3) local nutritional disturbances.

3. The relative infrequency of edema was a striking observation.

4. The lack of definition and differentiation between the first and second heart sounds was constantly observed.

Reprinted from the Archives of Internal Medicine
April, 1919, Vol. XXIII, pp. 431-440

AMERICAN MEDICAL ASSOCIATION
FIVE HUNDRED AND THIRTY-FIVE NORTH DEARBORN STREET
CHICAGO

[Reprinted from the BOSTON MEDICAL AND SURGICAL JOURNAL, Vol. clxxviii, No. 2, pp. 40-44, January 10, 1918.]

PAROXYSMAL TACHYCARDIA OF VENTRICULAR ORIGIN.*

BY F. A. WILLIUS, M.D., ROCHESTER, MINN.,
The Mayo Clinic, Rochester.

DURING the last two years an interesting group of tachycardias has been observed in the Mayo Clinic, and the infrequent occurrence and the importance of recognition merits this report. The literature contains a wealth of material dealing with tachycardia of sinus and nodal origin, but few articles could be found relative to tachycardia having its origin in the ventricles.

The rhythmic cardiac impulse takes its origin in the sino-auricular node[5] or "pacemaker," a collection of specialized tissue lying in the sulcus terminalis at the juncture of the superior vena cava and the right auricular appendage. This has been established by the experimental work of Lewis,[6] Oppenheim and Oppenheim,[5] Eyster and Meek,[9] who found this structure to become electro-negative before the rest of the sinus region. The function of "pacemaker" may be assumed by other portions of the heart, either within or outside of the conduction system, with the establishment of an ectopic rhythm.

Lewis[7] has classified these abnormal rhythms as homogenetic and heterogenetic. The former is characterized by a relatively slow rate, the onset of the rhythm is gradual, the seat of impulse

* Submitted Nov. 5, 1917, for publication.

production is probably always within the system of specialized tissue (conduction system) and the heart is under control of its extrinsic nerves. He believes this type to be due to exaggerated physiologic processes.

In contradistinction to this, the heterogenetic type presents a rapid pulse and rapid onset; the seat of impulse production may be within the system of specialized tissue or without, and the heart is not under control of its extrinsic innervation. This type is believed to result only from pathologic processes.

Paroxysmal ventricular tachycardia is heterogenetic and, as far as we know, is the result of myocardial disease. The recognition of this condition is of the utmost importance and can be made with certainty only by means of graphic records. The introduction of the electrocardiograph has made possible the identification of obscure tachycardias.

Experimental studies have not only clarified the mechanism of this disorder but have suggested etiologic processes. When a single induction shock is applied to any portion of the ventricle during its resting period a single premature contraction occurs.[6] The contraction evoked is not proportionate to the stimulus applied but always maximal,[1] constituting the well-known "all or none" law of Bowditch, and does not occur when the muscle is in the state of contraction[8] (refractory phase).

Regular series of suitably arranged induction shocks produce series of premature ventricular contractions simulating the graphic records of ventricular tachycardia. Lewis[6] produced premature ventricular contractions by ligation of the coronary arteries constantly, by tying off the left descending branch and in most instances

by impairing the circulation in the right vessel. As the nutrition of the ventricle became progressively impaired, series of heterogenetic contractions occurred, the sequence becoming longer as the nutritional changes became more marked.

By the intravenous injection of salts, Rothberger and Winterberg[11] produced this tachycardia in dogs. They found that combined stimulation of the vagi and accelerators caused cessation of the heart beat, but after injection of 5-10 mg. of barium chlorid in 1% aqueous solution, premature ventricular contractions occurred. With doses of 25 to 50 mg., minus accelerator stimulation, ventricular tachycardia was produced, and at times a transient arrhythmia. Calcium chlorid 100 to 200 mg. in 10% aqueous solution, produced similar results. They concluded that these salts increase the ventricular irritability, but stated that the nodal tissues are not appreciably influenced.

The electrocardiogram exhibits series of premature ventricular contractions, the complex forms varying with the point of origin in the ventricles. Identification of auricular contractions during the tachycardia is frequently difficult, but careful measurement shows that retrogression does not occur, as the first auricular complex of the normal rhythm falls at the proper point.

Lewis[6] maintains that the auricles and ventricles contract at the same rate, for each complex is identical to the adjacent one, and if auricles and ventricles were contracting at independent rates, the auricular complex would at times be superimposed and destroy the contour of the general curve. A case of ventricular tachycardia is reported by Palfrey[10] with poly-

graphic tracings in which the ventricular rate exceeded the auricular. This did not occur in any of the cases reported in this paper.

One other case of ventricular tachycardia is reported in the literature.[2]

Two of our cases revealed impairment of conduction, one in the junctional tissues and one beyond the main branch of the bundle of His.

Five cases of paroxysmal tachycardia of ventricular origin have come under the writer's observation during the last two years, and this disorder has occurred only in .047% of the abnormal cases. Three cases have occurred in males and two in females—the youngest one 21 years, the oldest 62 years, with an average age of 41.4 years. Four of the patients gave definite histories of previous infection with the streptococcus group. Syphilis could not be determined in any case.

The symptomatology in all cases was strikingly uniform, all histories revealing distressing palpitation with tachycardia, induced by exertion or excitement. The paroxysms had sudden onset, stopped abruptly and lasted from several minutes to several weeks. Vertigo attended the paroxysms in three cases, and two patients complained of nervousness. Exertion dyspnea was a constant symptom. One case presented slight pitting edema of the lower extremities.

Objectively the cardiac examinations revealed little of significance. In all cases there was slight increase in the dulness to the left (one-half to three-fourths inches) and valvular disease was not demonstrated in a single instance.

The lowest pulse rate during the paroxysms was 109, the highest 267, and the average of all recorded pulse readings was 174.

The pathologic changes in ventricular tachycardia cannot be identified as entities as no reported cases were disclosed in a search of the literature. The experimental work of Lewis,[6] however, suggests obliterative coronary disease and its attendant nutritive changes as a hypothetical pathologic picture.

One of our patients died a suicidal death, and we were afforded the opportunity of a necropsy. The left coronary artery was distinctly atheromatous, which is very significant in view of Lewis' work. The myocardium of the ventricles presented a few areas of fibrosis, the mitral and tricuspid leaflets were thickened, but apparently competent, and atheroma of the aortic valves was found. The thoracic and abdominal aorta were atheromatous.

In all probability, any condition increasing ventricular irritability is a potential factor in the production of this rare condition, and until more autopsy material is available, the conclusions as to lesion types must remain hypothetical. The gravity of the condition depends, of course, on the degree of myocardial damage and the duration of the paroxysms. One patient gave a twenty-six year history of attacks, and the duration of paroxysms gradually increased with progressive evidence of myocardial insufficiency; the last one in which the patient was observed lasted six weeks. Cardiac reserve is, of course, the all-important factor in the consideration of heart disease, and this means largely myocardial quality. The maintenance of circulation is dependent on ventricular, and not auricular action, and obviously any abnormal ventricular rhythm must be regarded as potentially a grave disorder. Lewis[4] emphasized this point, stating that ventricular tachycardia bor-

ders on fibrillation, and ventricular fibrillation, as far as we know, is incompatible with life.

Little can be said relative to treatment. Two patients were treated, both being placed on the tincture of digitalis in doses varying from 1 to 3 cc. three times daily. The one patient was placed at rest in bed and digitalis was administered three times, to toxic effect, without any change in the abnormal rhythm. He was under observation twenty-eight days. The other patient was symptomatically improved, but his paroxysms had never exceeded a few hours.

In cases showing evidence of myocardial insufficiency digitalis should be employed, but it is very questionable whether the abnormal rhythm can be arrested by its administration. In two cases vagus pressure was applied without results. In another case the atropine test had no effect on the ectopic rhythm.

SUMMARY.

1. Paroxysmal tachycardia of ventricular origin is a rare condition, occurring in only .047% of all abnormal electrocardiograms recorded in the Clinic.

2. Two cases revealed conduction impairment.

3. As an etiologic factor, history of infection with the streptococcus group was elicited in four cases.

4. The symptomatology in all cases was very uniform, palpitation, tachycardia and exertion dyspnea being complained of by all the patients. Vertigo attended the paroxysms in three cases.

5. The average pulse rate during the paroxysms was 174.

6. One case coming to necropsy revealed distinct atheroma of the left coronary artery, which is very significant.

7. As life is dependent on ventricular, and not on auricular action, this condition must be considered potentially grave.

8. Digitalis medication in two cases treated did not affect the abnormal rhythm.

CASE 1 (70066). A male, 62 years of age, came to the Clinic June 28, 1915. Five-year history of paroxysms of rapid pulse and palpitation. This case not only presents coupled heterogenetic ventricular contractions and short paroxysms of ventricular tachycardia, but also impaired conduction through the junctional tissues, the P. R. interval being 0.25 seconds. There is hypertrophy of the left ventricle. The sinus rate is 86; the ventricular tachycardia rate is 150. (Plates I and II.)

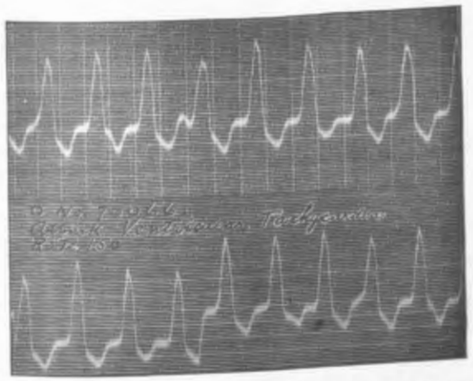


PLATE I.—No. 70,066. June 28, 1915. Leads I and III. Rate 150.

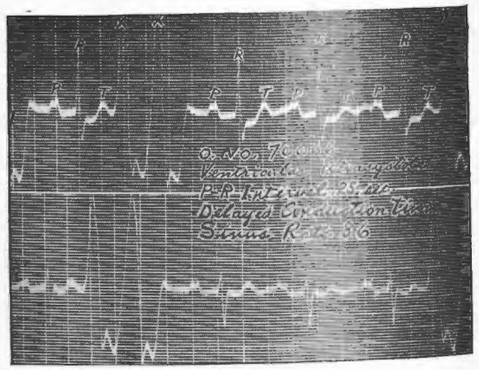


PLATE II.—No. 70,066. Aug. 31, 1915. Leads I and III. Sinus rate 86.

CASE 2 (185935). A female, 21 years of age, came to the Clinic Feb. 17, 1917. Three-year history of palpitation and tachycardia. Ventricular tachycardia. Rate, 120. In this instance the auricular complexes are evident and have the same rate as the ventricular. There is marked hypertrophy of the left ventricle. (Plate III.)

CASE 3 (194798). A male, 42 years of age, came to the Clinic May 5, 1917. Two-year history of spells of palpitation and tachycardia. Electrocardiograms show short paroxysms of ventricular tachycardia with intervening sinus rhythm. (Plates IV, V and VI.)

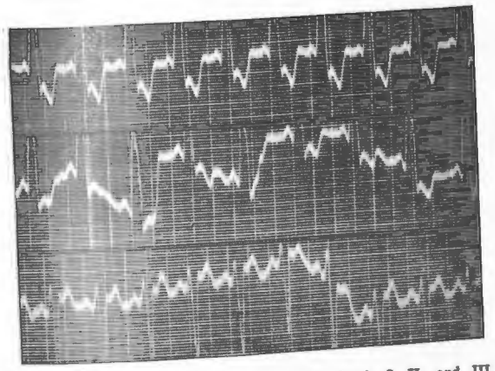


PLATE III.—No. 185,935. Feb. 19, 1917. Leads I, II, and III. Tachycardia rate 120.

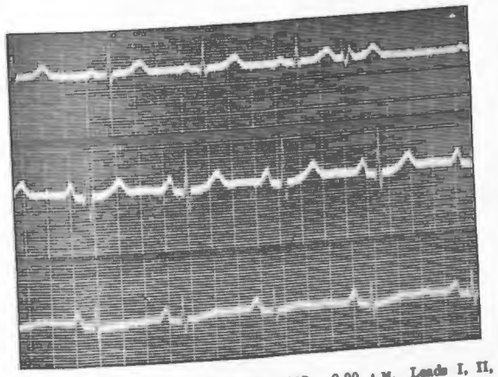


PLATE IV.—No. 194,798. June 18, 1917. 9.00 A.M. Leads I, II, and III. Rate 71. Ventricular premature contractions.

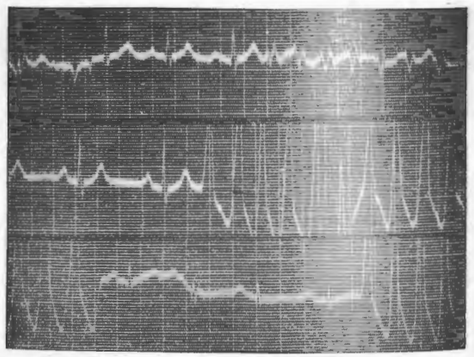
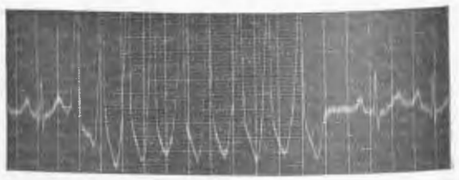
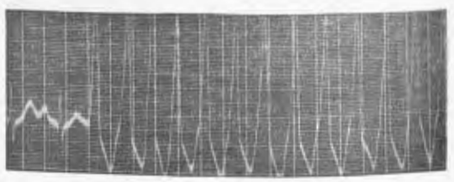


PLATE V.—No. 194,798. June 13, 1917, 3.45 P.M. Leads I, II, and III. Sinus rate 80 to 109. Short paroxysms of ventricular tachycardia rate 223 to 267.



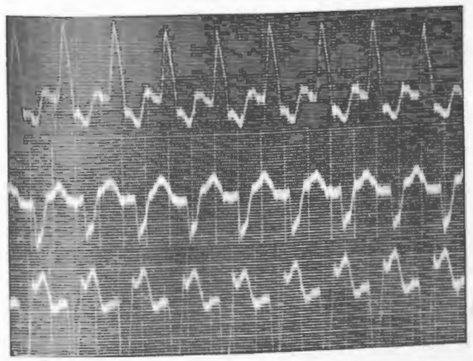
a.



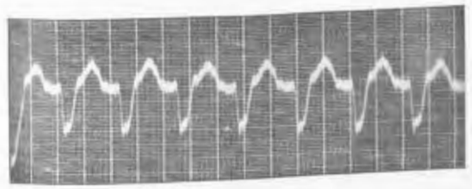
b.

PLATE VI.—(*a*) No. 194,798. June 13, 1917. Lead II. Sinus rate 100. Tachycardia rate 200. (*b*) No. 194,798. June 14, 1917. Lead II. Rate 220.

CASE 4 (200751). A female, 44 years of age, came to the Clinic July 11, 1917. Six-week history of palpitation and tachycardia. Ventricular tachycardia with rates varying from 120 to 125. At times the auricular complexes can be identified. This case presents evidence of arborization block. (Plate VII.)

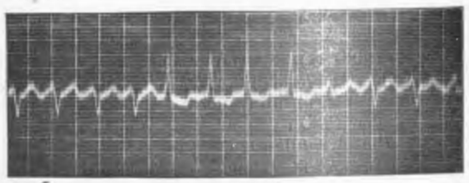


a.

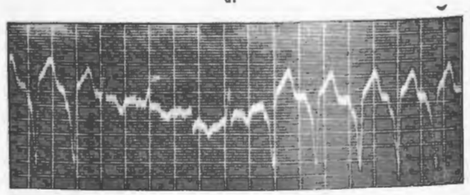


b.

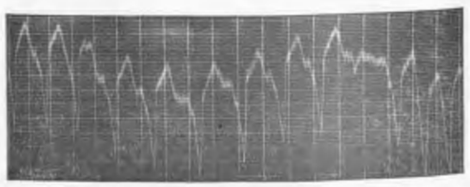
PLATE VII.—(*a*) No. 200,751. July 14, 1917. Leads I, II, and III. Rate 120 to 125. (*b*) No. 200,751. July 15, 1917. Lead II. Rate 125.



a.



b.



c.

PLATE VIII.—(a) No. 98,618. July 25, 1917. Lead II. Rate 172. Occasional nodal complexes. (b) No. No. 98,618. July 25, 1917. Lead III. Rate 172. (c) No. 98,618. July 25, 1917. Lead III. Rate 172.

CASE 5 (98618). A male, 38 years of age, came to the Clinic July 25, 1917. Twenty-six-year history of paroxysms of palpitation and tachycardia, increasing in frequency and duration. This case presents several interesting features. Occasional complexes are seen, arising probably in the junctional tissues. There is a constant difference in the general appearance of Lead II from all the other cases, and probably can be explained by the point of origin of the ectopic impulses. They arise from the basal portion of the left ventricle and from the direction of the heart's axis; Lead II

transects chiefly the "action currents" of the right heart. See Plate X. The lines x-y represent the transecting planes (Leads) by which the lines of force (action currents) are out. Plate XII shows the failure of vagus pressure to affect the tachycardia. A marked arrhythmia is present at times, and probably signifies variation in velocity of impulse discharge. (Plates VIII, IX, X, XI and XII.)

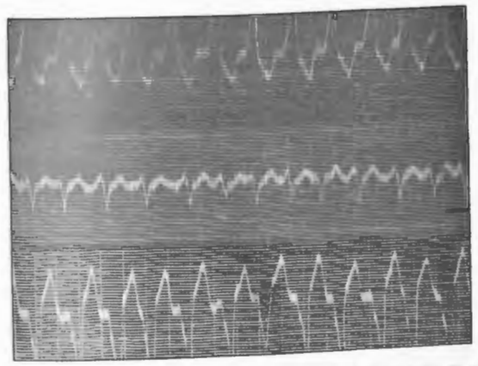


PLATE. IX.—No. 98,618. July 26, 1917. Leads I, II, and III. Rate 169 to 180.

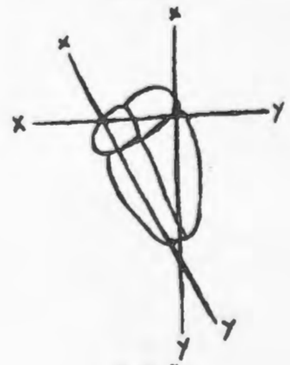
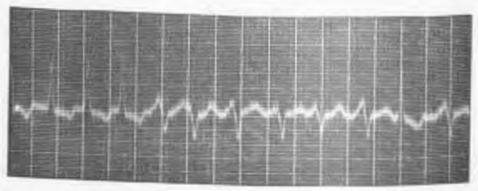


PLATE X.

a.

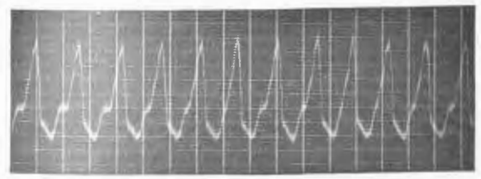


b.

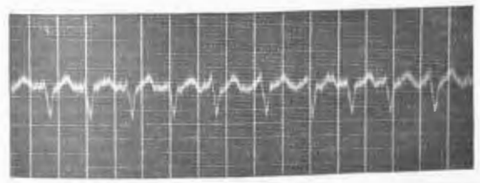
Rate 150 Rate 166

c.

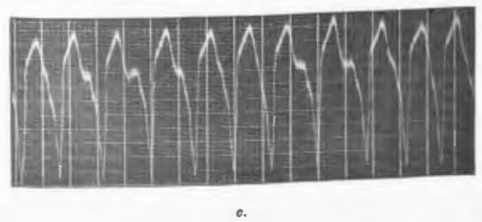
PLATE XI.—(*a*) No. 98,618. July 28, 1917. Lead II. Rate 150. Nodal tachycardia. (*b*) No. 98,618. July 28, 1917. Lead II. Rate 175. Nodal and ventricular tachycardia. (*c*) No. 98,618. July 28, 1917. Lead II. Failure of vagus pressure.



a.



b.



c.

PLATE XII.—(*a*) No. 98,618. Aug. 1, 1917. Lead I. Rate 192. (*b*) No. 98,618. Aug. 1, 1917, Lead II. Rate 175. (*c*) No. 98,618. Aug. 1, 1917. Lead III. Rate 180.

REFERENCES.

[1] Bowditch, H. P.: Ueber die Eigenthümlichkeit der Reizbarkeit, welche die Muskelfasern des Herzens zeigen. Arbeiten aus d. physiol. Anst. z. Leipzig, 1869 (published in 1872), 139-176.

[2] Hart, T. S.: Paroxysmal tachycardia; the paroxysms arise from the impulses of ventricular origin; the auricle responds to the ventricle; evidence of two points of abnormal ventricular irritability. Heart, 1912-1913, iv, 128-184.

[3] Keith, A., and Flack, M.: The form and nature of the muscular connections between the primary divisions of the vertebrate heart. Jour. Anat. and Physiol., 1906-1907, xli, 172-189.

[4] Lewis, T.: The experimental production of paroxysmal tachycardia and the effects of ligation of the coronary arteries. Heart, 1909-1910, i, 98-137.

[5] Lewis, T., Oppenheim, B. S., and Oppenheim, A.: The site of origin of the mammalian heart-beat; the pacemaker in the dog. Heart, 1910, ii, 147-169.

[6] Lewis, T.: Mechanism of the heart beat, London, Shaw, 1911, 87, 136, 158.

[7] Lewis, T.: Exceptional types of slow heart action, Quart. Jour. Med., 1912-1913, vi, 221-235.

[8] Marey, E.: Recherches sur les excitations electriques du coeur. Jour. d. l'Anat. e. d. Physiol., 1877, xiii, 60-83.

[9] Meek, W. J., and Eyster, J. A. E.: Experiments on the origin and propagation of the impulse in the heart. Am. Jour. Physiol., 1914, xxxiv, 368-388.

[10] Palfrey, F. W.: Paroxysmal tachycardia confined to the ventricles or to the auricles, with illustrative cases. Med. and Surg. Rep., Bost. City Hosp., 1913, 16, s. 182-189.

[11] Rothberger, C. J., and Winterberg, H.: Über die experimentelle Erzengung extrasystolischer ventrikulärer Tachykardie durch Acceleransreizung von Barium und Calcium. Arch. f. d. ges. Physiol., 1911, cxlii, 461-522.

PRESS OF JAMAICA PRINTING COMPANY, BOSTON, MASS.

The Operative Risk in Cardiac Disease.

By

F. A. WILLIUS, M.D.,

Mayo Clinic.

Rochester, Minn.

Reprinted from the

AMERICAN JOURNAL OF SURGERY

October, 1918.

go about in comfort, or those in whom this can be effected by medical treatment, are generally considered safe for operation.

In heart disease due to focal infection, such gratifying results are frequently seen following the removal of the focus, that the added risk seems justified. Another group of cases often shows marked cardiac improvement sufficient fully to justify the risk, namely, goiter, uterine fibroids and prostatic hypertrophy. Malignancy complicated by heart disease is generally considered operable if a fair chance for recovery is offered, and such cases often require palliative operations. In very few instances in which there has been urgent need of operation has it been refused on account of the cardiac condition, though in many instances, operation has only been undertaken after preliminary medical therapy.

In every case the decision is based on several factors: (1) The immediate operative risk, (2) the probable improvement of the heart following operation, (3) the patient's relative chance for length of life or general health with and without operation, and (4) in less serious conditions, whether or not the operative relief will justify the added risk. Experience in general has justified the taking of risks in cases demanding surgical intervention.

It is impossible to classify cases on a basis of valvular disease alone because the true index of cardiac efficiency is myocardial quality and this varies greatly in similar disease conditions. A classification based on cardiac reserve alone is also impossible because we have no accurate means of determining this factor and clinical impressions are variable.

Six groups of cases have been studied. These are generally recognized as bad risks, or the worst risks, if angina pectoris and aneurysm are excepted. The groups are: (1) Auricular fibrillation, (2) auricular flutter, (3) impaired auriculo-ventricular conduction, (4) impaired intraventricular conduction (arborization block) (5) mitral stenosis, and (6) aortic lesions including valvular disease, aortitis and dilatation (not aneurysmal.)

Auricular Fibrillation.—This disorder is now recognized as being due to rapid incoördinate contractions of the individual muscle bundles of the auricular wall. The auricles no longer contract, their walls dilated in diastole act as reservoirs in the general circulation. As the result of this disordered and inadequate stimulus production, the ventricular response is incoördinate and a pulse results which is usually totally arrhythmic. This condition may be chronic, intermittent or paroxysmal.

Exophthalmic Goiter.—Experience with exophthalmic goiter (hyperplastic toxic) has shown auricular fibrillation to be a frequent, disordered cardiac mechanism occurring in the course of the disease. Fibrillation occurs more frequently in the patient more than 40 years of age, and is often indicative of a relatively high degree of hyperthyroidism. The myocardiums of older patients, of course, do not tolerate toxic insults well, and fibrillation is very prone to be a permanent condition. The occurrence of this arrhythmia in young people usually evidences a high degree of hyperthyroidism, for the hearts of younger patients usually stand strain well. In this group, 104 patients have been operated on with 4 deaths. One patient died following a Porter hot water injection and 3 following thyroidectomy. Of the latter, 2 died of

hyperthyroidism and 1 of myocardial insufficiency on the second day after operation. Ten patients were under 30, twenty between 30 and 40 and seventy-four were more than 40 years of age. The operative mortality was 4 deaths in 104 cases (3.8 per cent) which compares favorably with the normal operative mortality of 2.6 per cent.

Thyrotoxic Adenomas.—Many patients having adenomas (simple goiter) for a certain number of years, develop symptoms of thyroid intoxication. The onset of symptoms is frequently insidious and the initial subjective complaints, those of a failing myocardium. These patients are usually older, beyond 40 years of age, and as I have mentioned, the heart muscle does not tolerate toxic influences well Added to this is an insidious onset. This group presents many cardiopaths. Experience has shown that such patients so often show striking cardiac improvement following thyroidectomy that the added risk seems fully justified. All patients with fibrillation are, however, subjected to preliminary medical treatment which is continued after operation, if the case demands it. Thirty-six patients with fibrillation had thyroidectomies with one operative death, giving a mortality of 2.7 per cent. The normal operative mortality in this group is 2.8 per cent. The favorable showing is owing largely to preoperative therapy and to the correlation between the surgical and medical services.

Other Conditions.—There were 10 cases of fibrillation in patients less than 40 years of age and 20 in those more than 40 years of age, constituting a total of 30 cases in which operative measures were employed. The operations were as follows: 12 tonsillectomies, 4 excisions of epitheliomas (2 lower lip, 1 glands of the neck and 1 larynx), 1 excision

of glands for diagnosis (sarcoma), 3 cholecystectomies and appendectomies, 3 gastro-enterostomies (2 for ulcer and 1 for carcinomatous obstruction), 1 cholecystectomy, choledochotomy and appendectomy, 1 Talma-Morrison, 1 herniotomy, 1 cataract extraction, 1 suprapubic stab, 1 cauterization for urethral caruncle and 1 cystotomy and prostatectomy. There were two early deaths, one cardiac, following suprapubic stab, and one due to cholangitis following cholecystectomy.

Auricular Flutter.—This cardiac disorder is recognized as being due to rapid coördinate contractions of the auricles, stimulated by foci of irritation located in the auricular wall outside the normal pace-maker (sino-auricular node). The auricles contract at a rate of 200 to 380 per minute and the ventricles respond usually to one-half the auricular contractions, although any rhythm from a 1:1 association to a complete heart block may exist. The pulse is regular in one-half the reported cases and grossly irregular in the other half. The degree of block may vary from time to time, and most patients are subject to paroxysmal "weak spells," owing to sudden decrease in the degree of block which allows the ventricles to assume full auricular rate. The condition is usually chronic and may exist for years.

Four patients have been operated on, all included in the foregoing under fibrillation. These patients are of particular interest as apparently being the first proved cases of flutter coming to operation. A previous report[3] showed that these four patients were subjected to vigorous digitalis therapy, and rest until fibrillation was induced, and then operation was done.

Three of the patients had exophthalmic goiter,

though one had a cholecystectomy and tonsillectomy in the Clinic and a thyroidectomy, later, elsewhere. One other patient had had tonsillectomy. All the patients with exophthalmic goiter resumed a normal rhythm after operation, and two had no further cardiac symptoms. The last patient on whom a tonsillectomy was performed, reports himself greatly improved. Thus far there has been no mortality.

Partial and Complete Heart Block.—One patient with complete block has had three operations in 11 years; appendectomy, radical amputation of the breast for carcinoma, and excision of recurring nodules of the skin. An electrocardiogram was taken before the last operation. The pulse was recorded as being unusually slow at the previous examinations. The patient is alive and quite well.

Ten patients showing delayed conduction between auricles and ventricles, that is, auriculo-ventricular intervals of 0.22 to 0.28 of a second, have been operated on as follows: 4 tonsillectomies, 1 double ligation of the superior thyroid vessels for exophthalmic goiter, 1 double ligation and subsequent thyroidectomy for exophthalmic goiter, 2 thyroidectomies for thyrotoxic adenomas, 1 cholecystectomy and 1 prostatectomy. Six were more than 40 years of age and 4 were under 40 years. The patient on whom prostatectomy was done died on the fourth day, presenting the cardio-vascular renal syndrome.

Intraventricular or Arborization Block.—This condition is due to impaired conduction of the cardiac impulse after its passage through the bifurcation of the auriculo-ventricular bundle and evidences disease of the main bundle branches and the subendocardial plexus. Oppenheimer and Rothschild have emphasized the gravity of this condition and the early fatality which it often indicates.

The electrocardiogram reveals a prolonged Q. R. S. interval and variations from slight notching to the unusual complexes which are ascribed to branch bundle defects. A striking observation in this group of cases is the uniformity with which the clinical findings are substantiated by the graphic records in revealing serious myocardial disease. Twenty patients have been operated on, 7 under 40 years of age and 13 more than 40 years, without any operative mortality. There were 8 tonsillectomies, 5 thyroidectomies (3 for exophthalmic goiter and 2 for thyrotoxic adenoma), 1 salpingectomy, 1 cholecystectomy and appendectomy, 2 chest aspirations, 2 gland excisions for diagnosis (1 malignant and 1 inflammatory) and 1 posterior gastro-enterostomy for duodenal ulcer.

Mitral Stenosis.—Seventy-three cases of mitral stenosis are recorded in which operations were done. Twenty-five of the patients were under 40 years of age and 48 were more than 40 years. As previously stated, valvular disease alone cannot be satisfactorily grouped because of the difficulty in accurately classifying the degree of myocardial insufficiency. This mitral lesion is recognized as being serious owing to its tendency to progression, and therefore the cases have been included in this report. An attempt has been made to estimate, by clinical impressions, the degree of decompensation present, and, while obviously inaccurate, it is necessary in presenting the type of case represented in this study. The scale of 1 to 4 (minimum to maximum) has arbitrarily been used in denoting the degree of decompensation. The average in patients under 40 years of age was 2, in those more than 40, 2+. Ten patients showed auricular fibrillation; (vide supra) 9 of these were patients more

than 40 years of age. The operations are as follows: 39 tonsillectomies, 17 thyroidectomies (10 for simple goiter, 4 for exophthalmic goiter, and 3 for thyrotoxic adenomas), and 4 of these patients had secondary operations including 2 tonsillectomies, 1 appendectomy and 1 double cataract extraction. There were one double ligation of the superior thyroid vessels for exophthalmic goiter, 4 appendectomies, 1 cholecystostomy, 4 cholecystectomies and appendectomies, 1 choledochotomy, cholecystectomy and appendectomy, 1 subtotal abdominal hysterectomy, 1 perineorrhaphy, 1 trachelorrhaphy, 1 tumor excision (benign) 1 inguinal herniotomy and 1 thoracic paracentesis. There was no immediate operative mortality but one patient died two weeks later of cholangitis following a choledochotomy, cholecystectomy and appendectomy. The mortality in this group is 1.3 per cent. It is impossible accurately to state the normal mortality in such a protean surgical list but 1.5 per cent seems very conservative.

Aortic Lesions.—It has long been recognized that aortic disease needs no emphasis as regards its gravity. This group includes aortic valvular disease, aortitis and dilatation (not aneurysmal). Sixteen patients with aortic valvular disease have been operated on; 11 under 40 years of age and 5 more than 40 years. Six patients presented double aortic lesions, that is, insufficiency and stenosis, and 1 presented evidence of aortitis. One patient had aortic stenosis alone. These patients were all able to be up and about with relative comfort. Anginal pains were not elicited in a single instance. There were no operative deaths but 1 patient is reported dead from heart failure one year later (tonsillectomy). There were 12 tonsillectomies, 1 thyroidectomy for

adenomas, 1 cholecystectomy, 1 double herniotomy and appendectomy, and 1 chest aspiration. Two patients with aortitis (not including the aforementioned case) were operated on; both were more than 40 years of age. There were one exploration (general abdominal carcinosis) and one tonsillectomy. The latter patient died a cardiac death three months later. Four patients with dilatations of the aorta (not aneurysmal) underwent surgical procedures. The clinical diagnoses in these cases were verified by the fluoroscope. Three of the patients were more than 40 years of age. There were 2 thyroidectomies for exophthalmic goiter, 1 tonsillectomy, and 1 cystotomy and herniotomy. There were no deaths.

Summary.

1. The decision of operability in cardiac disease depends on factors as follows: (1) The immediate operative risk, (2) the probable improvement of the heart after operation, (3) the patient's relative chance for length of life or general health with and without operation, and (4) in less serious conditions, whether the operative relief will justify the added risk.

2. Cases in which the heart permits the patient to go about in relative comfort, or in which it can be sufficiently restored by treatment to allow this, usually are considered safe for operation.

3. Malignancy complicated by heart disease is usually considered operable if a fair hope of cure is offered.

4. The best measure of operative risk is a good clinical impression of the patients' ability to stand physical strain, supplemented by a careful history and a thorough physical examination.

5. Preoperative medical therapy and rest com-

April, 1919, No. 4, Vol. CLVII, p. 485

bined with surgical and medical correlation after operation, is of paramount importance.

6. The general tendency is to require too great a margin of cardiac safety in surgical work.

REFERENCES.

1. Blackford, J. M., Willius, F. A., and Haines, S. B.: Operative risk in cardiac disease. Jour. Am. Med. Assn., 1917, lxix, 2011-2014.
2. Blackford, J. M. and Willius, F. A.: Auricular flutter. Arch. Int. Med., 1918, xxi, 147-165.
3. Oppenheimer, B. S. and Rothschild, M. A.: Electrocardiographic changes associated with myocardial involvement. Jour. Am. Med. Assn., 1917, lxix, 429.

Congenital Dextrocardia

BY

F. A. WILLIUS, M.D.

MAYO CLINIC, ROCHESTER, MINNESOTA.

FROM THE
AMERICAN JOURNAL OF THE MEDICAL SCIENCES
April, 1919, No. 4, vol. clvii, p. 485

Extracted from the American Journal of the Medical Sciences,
April, 1919, No. 4, vol. clvii, p. 485.

CONGENITAL DEXTROCARDIA.

By F. A. Willius, M.D.,
MAYO CLINIC, ROCHESTER, MINNESOTA.

Anomalies of the heart, partly because of their infrequent occurrence and partly because of their occult manifestations, are of particular interest to the clinician. I have recently been afforded the opportunity of observing three cases of congenital dextrocardia. Two types of this anomaly are recognized: one associated with

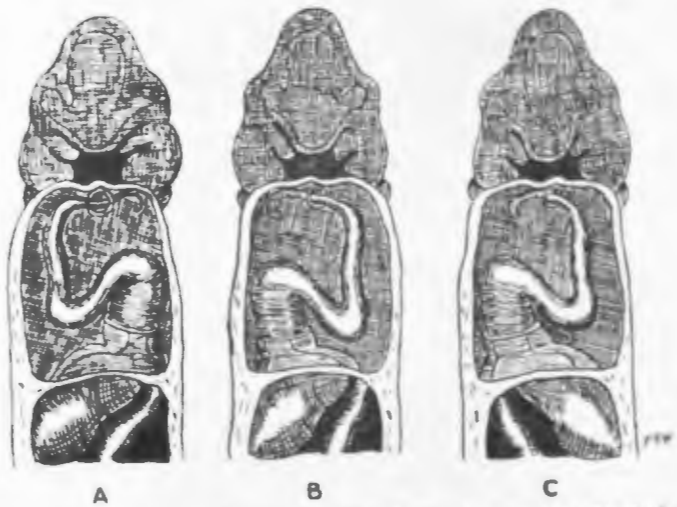


Fig. 1.—Transposition of the viscera in the embryo. *A*, normal; *B*, simple dextrocardia; *C*, complete situs transversus. Human embryo of about fifteen days. (After His.)

transposition of the abdominal viscera (*situs transversus*), and the other, in which the transposition affects only the heart and great vessels. At times anomalous arrangement of the venæ cavæ permits the admixture of arterial and venous blood; this gives rise to a clinical picture simulating the syndrome of congenital heart disease.

Recalling the embryological development of the heart, it is readily seen how transposition of this organ occurs. The two primitive cardiac tubes fuse into one about the fifteenth day[2] and an auricular, ventricular and bulbar subdivision becomes evident (Fig. 1). The tube soon becomes bent on itself, which determines largely the future axis of the heart. In congenital transposition the primitive tube bends into a contrasigmoid (S) instead of the normal sigmoid (S) manner. This has been explained[1] by assuming that the embryo lies in an abnormal position within the chorion, so that its right side instead of its left lies closer to the blood supply. The three patients whom I examined presented the most frequent anomaly, dextrocardia with *situs transversus*. In no instance was there any complaint referable to the abnormality.

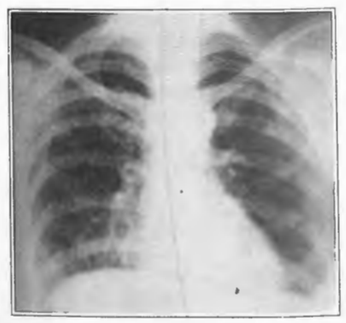


FIG. 2

REPORT OF CASES.

CASE I (222329).—A woman, aged forty years, presented herself for examination complaining of chest pains of the intercostal neuralgic type. The apex-beat of the heart was palpable in the fifth right intercostal space 7.5 cm. from the midsternal line. The cardiac dulness extended 9.0 cm. to the right and 1.5 cm. to the left of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found to be on the left side and gastric tympany on the right. The systolic blood-pressure was 152, the diastolic was 90. Radiograms of the chest showed the dextrocardia and a transposition of the stomach and colon (fluoroscopic colon). The electrocardiogram showed the heart-rate to be 94. Complete inversion of Leads I and II. The amplitude of the *R* waves in Leads I and II exceeded those in Lead III by one-third (Figs. 2, 3 and 4).

CASE II (224506).—A woman, aged thirty-seven years, presented herself for examination on account of a pelvic complaint. The apex-beat of the heart was palpable in the sixth right intercostal space,

FIG. 3

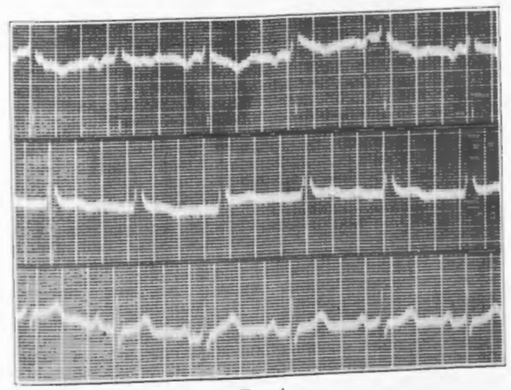


FIG. 4

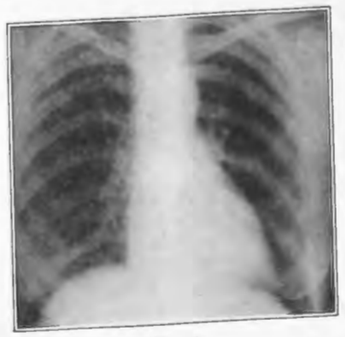


FIG. 5

9.0 cm. from the midsternal line. The cardiac dulness extended 11.0 cm. to the right of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found on the left side and gastric tympany on the right. A bilateral salpingitis and a cyst of

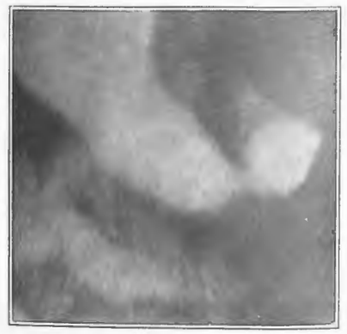


FIG. 6

the left ovary were palpated. The systolic blood-pressure was 120, the diastolic was 75. The radiograms revealed dextrocardia and transposition of the stomach and colon (fluoroscopic colon). The

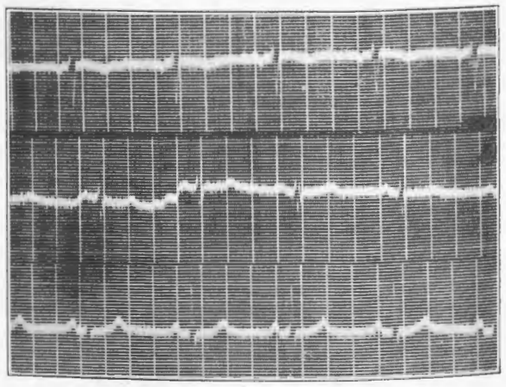


FIG. 7

electrocardiogram showed the heart-rate to be 75. There was complete inversion of Lead I. The amplitude of the *R* waves in Lead I were practically the same as those in Lead II and exceeded those in Lead III by slightly more than a third (Figs. 5, 6 and 7).

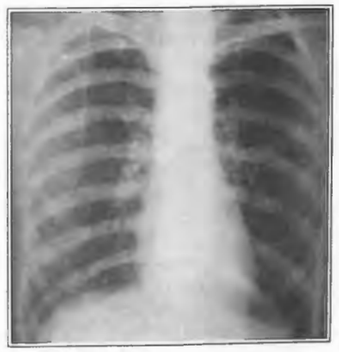


FIG. 8

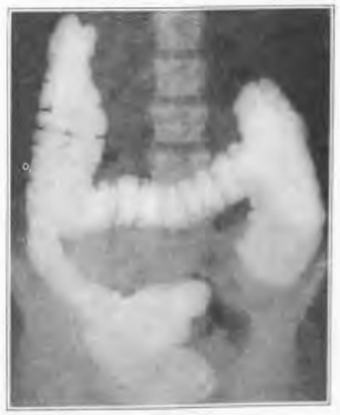


FIG. 9

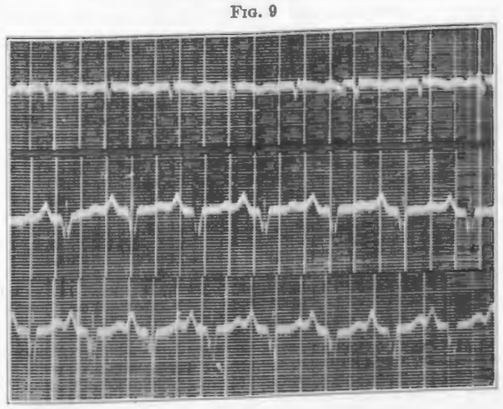
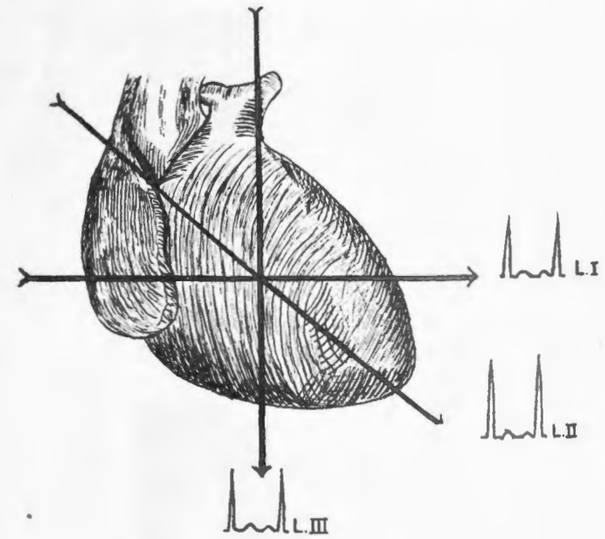
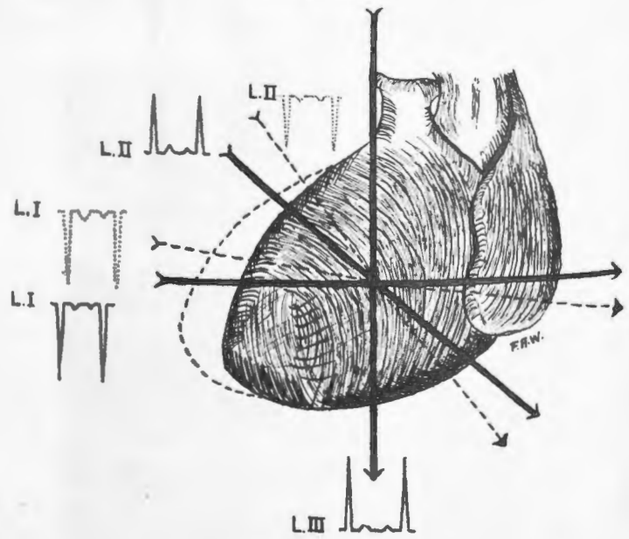


FIG. 10



A

Normal heart.



B

Congenital dextrocardia.

FIG. 11.—Schematic representations showing the angles produced by the direction of the leads and the resulting electrocardiograms. The dotted lines in Fig. *B* show the inclination to the right exaggerated and the inversion affecting Lead II.

CASE III (238633).—A woman, aged thirty-three years, presented herself for examination on account of goitre. The heart, as in the other cases, was found to be on the right side. The liver dulness was found on the left side and the gastric tympany on the right. The patient had a single adenoma of the right lobe of the thyroid 4.0 by 4.5 cm. The systolic blood-pressure was 112, the diastolic was 78. The radiogram revealed dextrocardia and transposition of the colon. The electrocardiogram showed the heart-rate to be 115; there was complete inversion of Lead I, and the amplitude of the *R* waves in Lead I were diminished to about one-half those of Lead III. The amplitude of the *R* waves in Lead III slightly exceeded those in Lead II. There was evidence of left ventricular preponderance (Figs. 8, 9 and 10).

The electrocardiograms of the last two cases essentially confirm the findings recorded in previous publications.[4 5 7 8] Lead I shows a complete inversion of all the deflections. Fig. 11 illustrates the angles produced by the direction of the leads and the resulting electrocardiograms. Case I (222329) shows the inversion also involving Lead II, and is explained by an exaggeration of the inclination of the cardiac axis to the right. The leads represent fixed planes of electrical potential, and changes in cardiac position or alterations in muscle bulk preponderance obviously affect the electrical currents, as expressed by the electrocardiograms.

It has been mentioned[6] that the *R* wave in Lead III becomes taller than in Lead II, but in these reported cases no constancy was observed. Hirschfelder mentions that the electrocardiographic curves sometimes are practically normal. Inversion of the deflections in Lead I is definite evidence of congenital dextrocardia with *situs transversus*, and we recognize electrocardiography as a valuable adjunct in the differential diagnosis of cardiac displacements.

REFERENCES.

1. Abbott, M. E.: Congenital cardiac disease. In: Mod. Med., Philadelphia, Lippincott, 1908, iv, 323.
2. Bryce, T. H.: Embryology. In: Quain's anatomy. New York, Longmans, 1908, i, p. 63.
3. Hirschfelder, A. D.: Diseases of the heart and aorta. Philadelphia, Lippincott, 1918, p. 562.
4. Hoke, E.: Ueber das Elektrokardiogramm eines Falles von Situs viscerum inversus totalis. München. med. Wchnschr., 1911, lviii, 802.
5. Lewis, T.: Electrocardiography and its importance in the clinical examination of heart affections. Brit. Med. Jour., 1912, i, 1421–1423.
6. Neuhof, S.: Clinical cardiology. New York, Macmillan, 1917, p. 32.
7. Nicolai, G. F.: Das Elektrokardiogramm bei Dextrocardie und anderen Lageveranderungen des Herzens. Berl. klin. Wchnschr., 1911, xlviii, 51–55.
8. Owen, S. A.: A case of complete transposition of the viscera, associated with mitral stenosis; including a description of the electrocardiographic tracings. Heart, 1911–1912, iii, 113–117.

Congenital Dextrocardia

BY

F. A. WILLIUS, M.D.

MAYO CLINIC, ROCHESTER, MINNESOTA.

FROM THE
AMERICAN JOURNAL OF THE MEDICAL SCIENCES
April, 1919, No. 4, vol. clvii, p. 485

Extracted from the American Journal of the Medical Sciences,
April, 1919, No. 4, vol. clvii, p. 485.

CONGENITAL DEXTROCARDIA.

By F. A. Willius, M.D.,
MAYO CLINIC, ROCHESTER, MINNESOTA.

Anomalies of the heart, partly because of their infrequent occurrence and partly because of their occult manifestations, are of particular interest to the clinician. I have recently been afforded the opportunity of observing three cases of congenital dextrocardia. Two types of this anomaly are recognized: one associated with

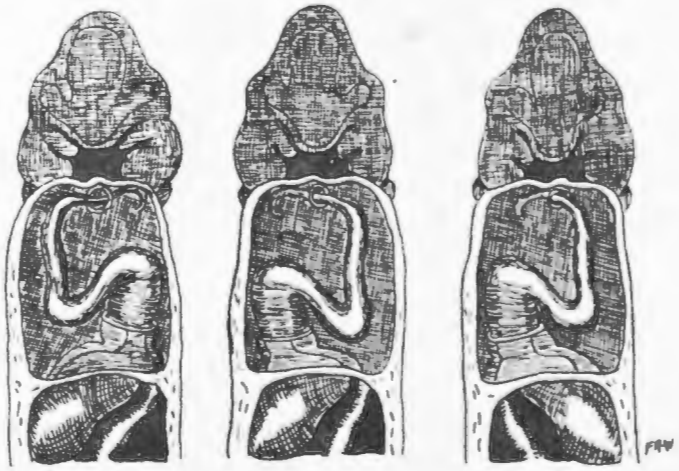


Fig. 1.—Transposition of the viscera in the embryo. *A*, normal; *B*, simple dextrocardia; *C*, complete situs transversus. Human embryo of about fifteen days. (After His.)

transposition of the abdominal viscera (*situs transversus*), and the other, in which the transposition affects only the heart and great vessels. At times[3] anomalous arrangement of the venæ cavæ permits the admixture of arterial and venous blood; this gives rise to a clinical picture simulating the syndrome of congenital heart disease.

Recalling the embryological development of the heart, it is readily seen how transposition of this organ occurs. The two primitive cardiac tubes fuse into one about the fifteenth day[2] and an auricular, ventricular and bulbar subdivision becomes evident (Fig. 1). The tube soon becomes bent on itself, which determines largely the future axis of the heart. In congenital transposition the primitive tube bends into a contrasigmoid (S) instead of the normal sigmoid (S) manner. This has been explained[1] by assuming that the embryo lies in an abnormal position within the chorion, so that its right side instead of its left lies closer to the blood supply. The three patients whom I examined presented the most frequent anomaly, dextrocardia with *situs transversus*. In no instance was there any complaint referable to the abnormality.

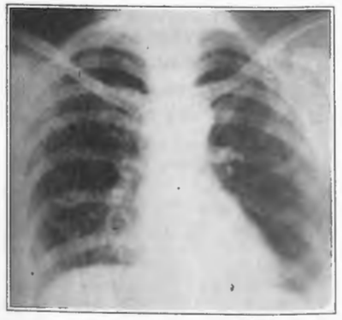


FIG. 2

REPORT OF CASES.

CASE I (222329).—A woman, aged forty years, presented herself for examination complaining of chest pains of the intercostal neuralgic type. The apex-beat of the heart was palpable in the fifth right intercostal space 7.5 cm. from the midsternal line. The cardiac dulness extended 9.0 cm. to the right and 1.5 cm. to the left of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found to be on the left side and gastric tympany on the right. The systolic blood-pressure was 152, the diastolic was 90. Radiograms of the chest showed the dextrocardia and a transposition of the stomach and colon (fluoroscopic colon). The electrocardiogram showed the heart-rate to be 94. Complete inversion of Leads I and II. The amplitude of the *R* waves in Leads I and II exceeded those in Lead III by one-third (Figs. 2, 3 and 4).

CASE II (224506).—A woman, aged thirty-seven years, presented herself for examination on account of a pelvic complaint. The apex-beat of the heart was palpable in the sixth right intercostal space,

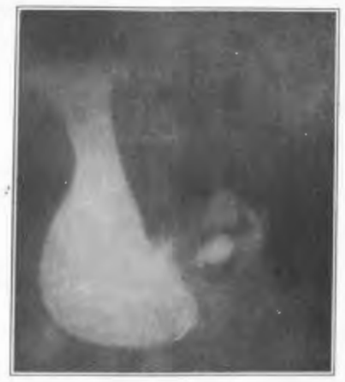


FIG. 3

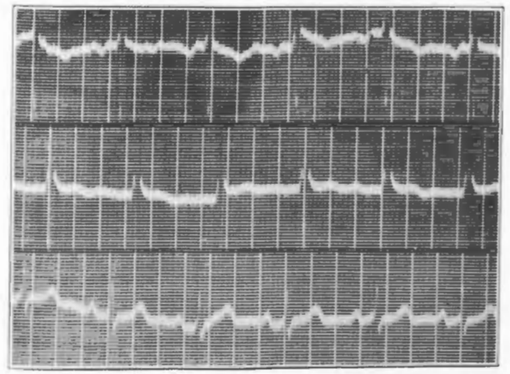


FIG. 4

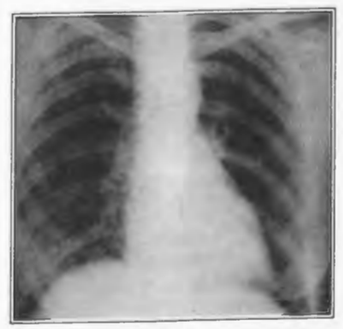


FIG. 5

9.0 cm. from the midsternal line. The cardiac dulness extended 11.0 cm. to the right of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found on the left side and gastric tympany on the right. A bilateral salpingitis and a cyst of

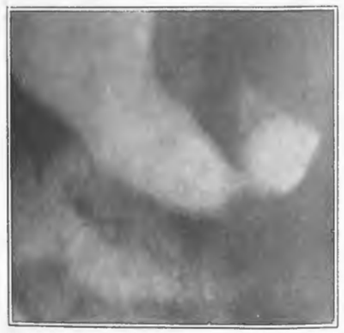


Fig. 6

the left ovary were palpated. The systolic blood-pressure was 120, the diastolic was 75. The radiograms revealed dextrocardia and transposition of the stomach and colon (fluoroscopic colon). The

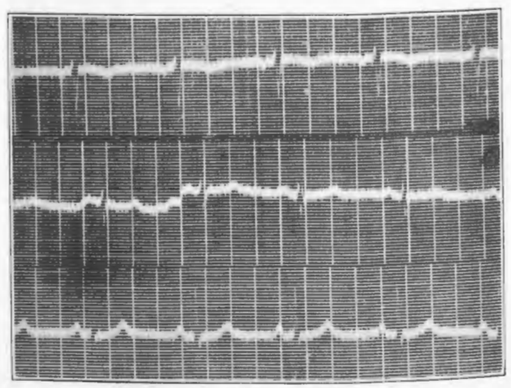


Fig. 7

electrocardiogram showed the heart-rate to be 75. There was complete inversion of Lead I. The amplitude of the *R* waves in Lead I were practically the same as those in Lead II and exceeded those in Lead III by slightly more than a third (Figs. 5, 6 and 7).

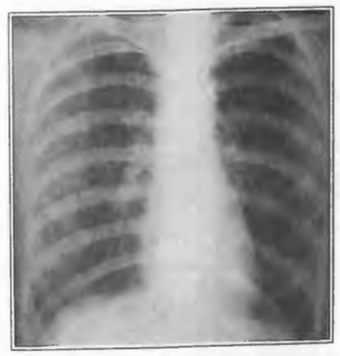


Fig. 8

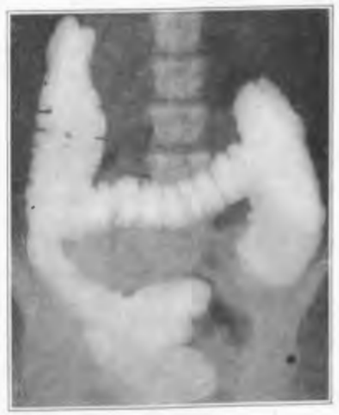


Fig. 9

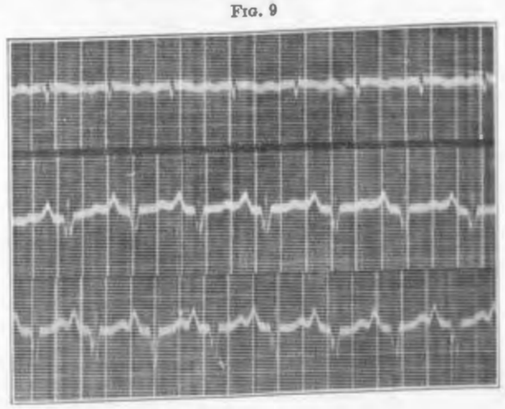
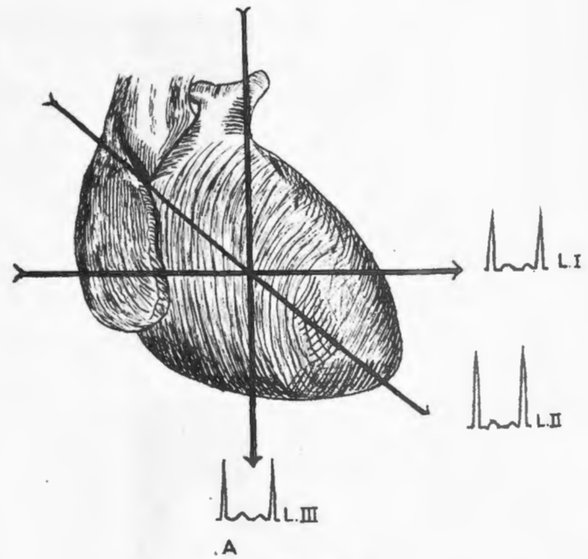
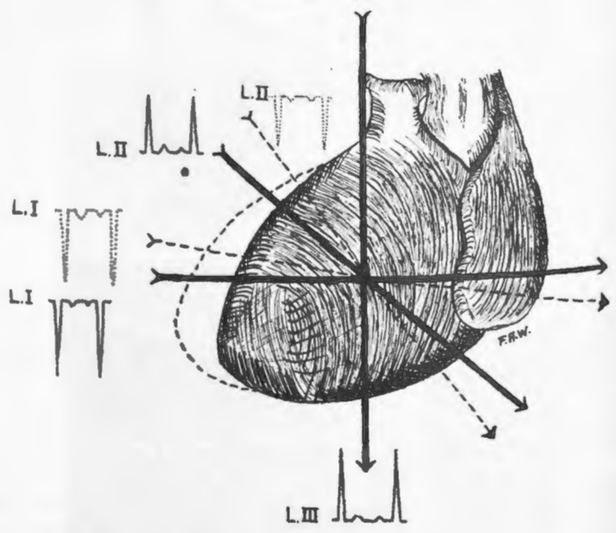


Fig. 10



A

Normal heart.



B

Congenital dextrocardia.

FIG. 11.—Schematic representations showing the angles produced by the direction of the leads and the resulting electrocardiograms. The dotted lines in Fig. *B* show the inclination to the right exaggerated and the inversion affecting Lead II.

CASE III (238633).—A woman, aged thirty-three years, presented herself for examination on account of goitre. The heart, as in the other cases, was found to be on the right side. The liver dulness was found on the left side and the gastric tympany on the right. The patient had a single adenoma of the right lobe of the thyroid 4.0 by 4.5 cm. The systolic blood-pressure was 112, the diastolic was 78. The radiogram revealed dextrocardia and transposition of the colon. The electrocardiogram showed the heart-rate to be 115; there was complete inversion of Lead I, and the amplitude of the *R* waves in Lead I were diminished to about one-half those of Lead III. The amplitude of the *R* waves in Lead III slightly exceeded those in Lead II. There was evidence of left ventricular preponderance (Figs. 8, 9 and 10).

The electrocardiograms of the last two cases essentially confirm the findings recorded in previous publications.[4 5 7 8] Lead I shows a complete inversion of all the deflections. Fig. 11 illustrates the angles produced by the direction of the leads and the resulting electrocardiograms. Case I (222329) shows the inversion also involving Lead II, and is explained by an exaggeration of the inclination of the cardiac axis to the right. The leads represent fixed planes of electrical potential, and changes in cardiac position or alterations in muscle bulk preponderance obviously affect the electrical currents, as expressed by the electrocardiograms.

It has been mentioned[6] that the *R* wave in Lead III becomes taller than in Lead II, but in these reported cases no constancy was observed. Hirschfelder mentions that the electrocardiographic curves sometimes are practically normal. Inversion of the deflections in Lead I is definite evidence of congenital dextrocardia with *situs transversus*, and we recognize electrocardiography as a valuable adjunct in the differential diagnosis of cardiac displacements.

REFERENCES.

1. Abbott, M. E.: Congenital cardiac disease. In: Mod. Med., Philadelphia, Lippincott, 1908, iv, 323.
2. Bryce, T. H.: Embryology. In: Quain's anatomy. New York, Longmans, 1908, i, p. 63.
3. Hirschfelder, A. D.: Diseases of the heart and aorta. Philadelphia, Lippincott, 1918, p. 562.
4. Hoke, E.: Ueber das Elektrokardiogramm eines Falles von Situs viscerum inversus totalis. München. med. Wchnschr., 1911, lviii, 802.
5. Lewis, T.: Electrocardiography and its importance in the clinical examination of heart affections. Brit. Med. Jour., 1912, i, 1421–1423.
6. Neuhof, S.: Clinical cardiology. New York, Macmillan, 1917, p. 32.
7. Nicolai, G. F.: Das Elektrokardiogramm bei Dextrocardie und anderen Lageveranderungen des Hersens. Berl. klin. Wchnschr., 1911, xlviii, 51–55.
8. Owen, S. A.: A case of complete transposition of the viscera, associated with mitral stenosis; including a description of the electrocardiographic tracings. Heart, 1911–1912, iii, 113–117.

Congenital Dextrocardia

BY

F. A. WILLIUS, M.D.
MAYO CLINIC, ROCHESTER, MINNESOTA.

FROM THE
AMERICAN JOURNAL OF THE MEDICAL SCIENCES
April, 1919, No. 4, vol. clvii, p. 485

Extracted from the American Journal of the Medical Sciences,
April, 1919, No. 4, vol. clvii, p. 485.

CONGENITAL DEXTROCARDIA.

By F. A. Willius, M.D.,
MAYO CLINIC, ROCHESTER, MINNESOTA.

Anomalies of the heart, partly because of their infrequent occurrence and partly because of their occult manifestations, are of particular interest to the clinician. I have recently been afforded the opportunity of observing three cases of congenital dextrocardia. Two types of this anomaly are recognized: one associated with

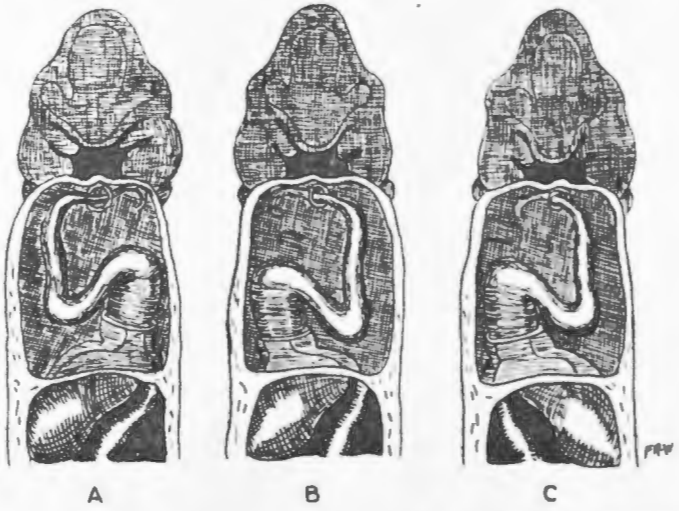


Fig. 1.—Transposition of the viscera in the embryo. *A*, normal; *B*, simple dextrocardia; *C*, complete situs transversus. Human embryo of about fifteen days. (After His.)

transposition of the abdominal viscera (*situs transversus*), and the other, in which the transposition affects only the heart and great vessels. At times[3] anomalous arrangement of the venæ cavæ permits the admixture of arterial and venous blood; this gives rise to a clinical picture simulating the syndrome of congenital heart disease.

Recalling the embryological development of the heart, it is readily seen how transposition of this organ occurs. The two primitive cardiac tubes fuse into one about the fifteenth day[2] and an auricular, ventricular and bulbar subdivision becomes evident (Fig. 1). The tube soon becomes bent on itself, which determines largely the future axis of the heart. In congenital transposition the primitive tube bends into a contrasigmoid (S) instead of the normal sigmoid (S) manner. This has been explained[1] by assuming that the embryo lies in an abnormal position within the chorion, so that its right side instead of its left lies closer to the blood supply. The three patients whom I examined presented the most frequent anomaly, dextrocardia with *situs transversus*. In no instance was there any complaint referable to the abnormality.

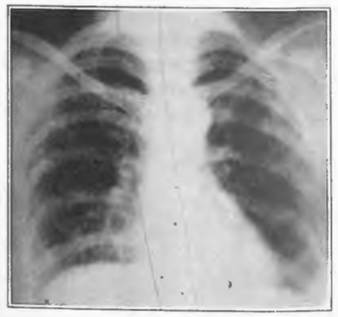


FIG. 2

REPORT OF CASES.

CASE I (222329).—A woman, aged forty years, presented herself for examination complaining of chest pains of the intercostal neuralgic type. The apex-beat of the heart was palpable in the fifth right intercostal space 7.5 cm. from the midsternal line. The cardiac dulness extended 9.0 cm. to the right and 1.5 cm. to the left of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found to be on the left side and gastric tympany on the right. The systolic blood-pressure was 152, the diastolic was 90. Radiograms of the chest showed the dextrocardia and a transposition of the stomach and colon (fluoroscopic colon). The electrocardiogram showed the heart-rate to be 94. Complete inversion of Leads I and II. The amplitude of the *R* waves in Leads I and II exceeded those in Lead III by one-third (Figs. 2, 3 and 4).

CASE II (224506).—A woman, aged thirty-seven years, presented herself for examination on account of a pelvic complaint. The apex-beat of the heart was palpable in the sixth right intercostal space,

FIG. 3

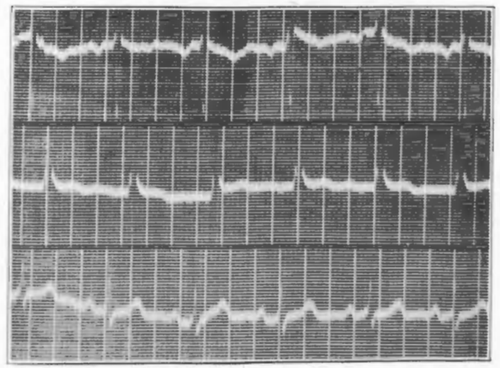


FIG. 4

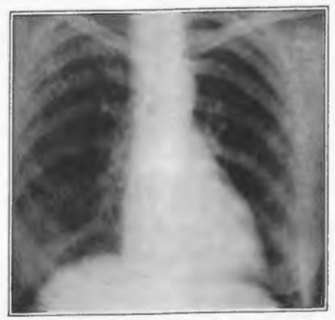


FIG. 5

9.0 cm. from the midsternal line. The cardiac dulness extended 11.0 cm. to the right of the midsternum. The heart sounds were best heard at the apex. Liver dulness was found on the left side and gastric tympany on the right. A bilateral salpingitis and a cyst of

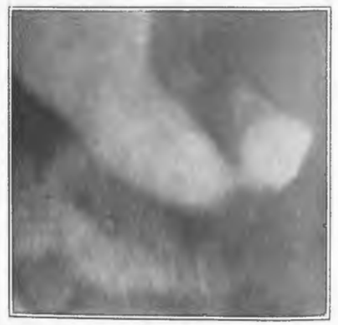


FIG. 6

the left ovary were palpated. The systolic blood-pressure was 120, the diastolic was 75. The radiograms revealed dextrocardia and transposition of the stomach and colon (fluoroscopic colon). The

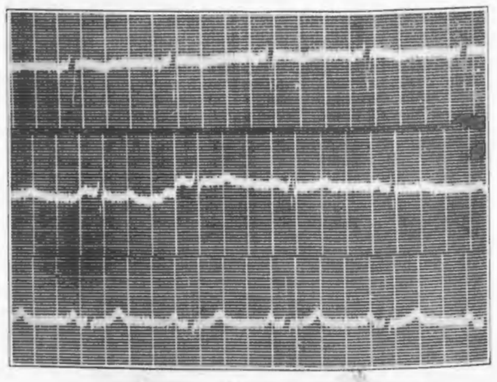


FIG. 7

electrocardiogram showed the heart-rate to be 75. There was complete inversion of Lead I. The amplitude of the *R* waves in Lead I were practically the same as those in Lead II and exceeded those in Lead III by slightly more than a third (Figs. 5, 6 and 7).

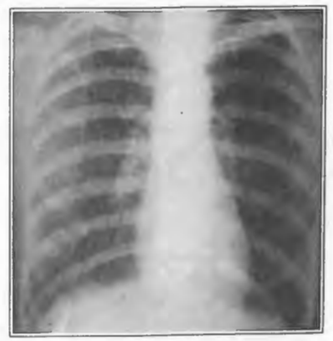


FIG. 8

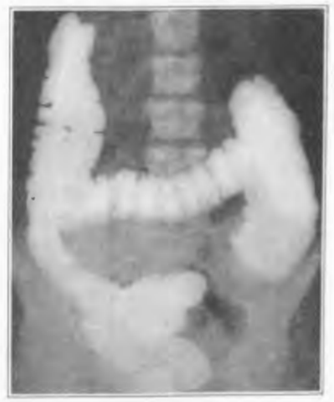


FIG. 9

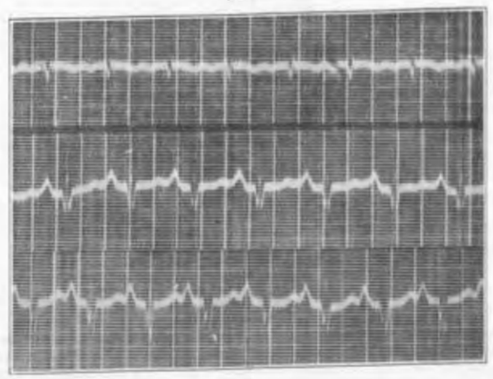


FIG. 10

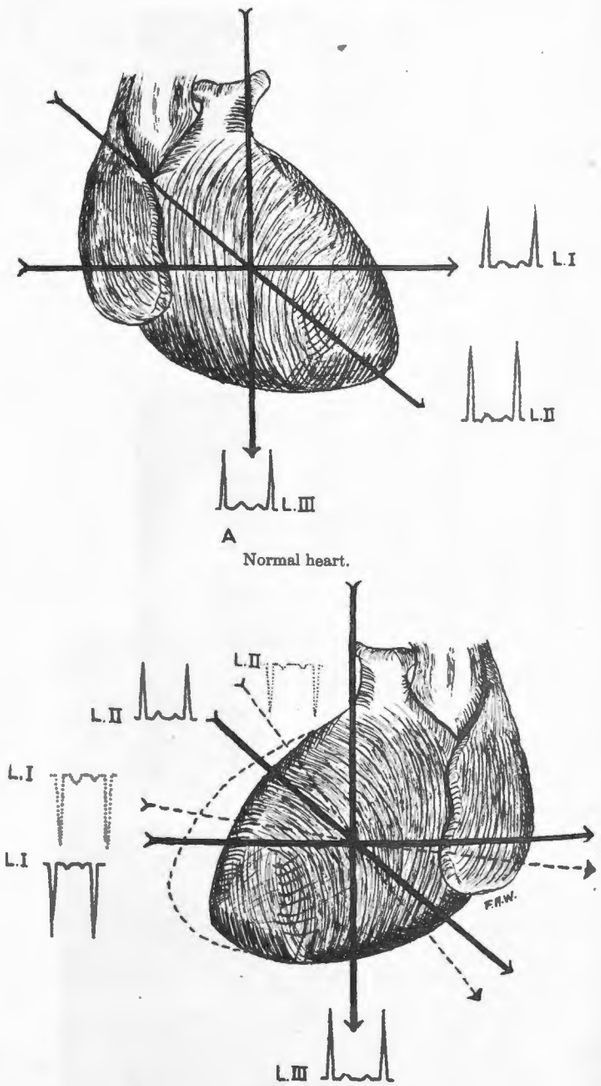


FIG. 11.—Schematic representations showing the angles produced by the direction of the leads and the resulting electrocardiograms. The dotted lines in Fig. *B* show the inclination to the right exaggerated and the inversion affecting Lead II.

CASE III (238633).—A woman, aged thirty-three years, presented herself for examination on account of goitre. The heart, as in the other cases, was found to be on the right side. The liver dulness was found on the left side and the gastric tympany on the right. The patient had a single adenoma of the right lobe of the thyroid 4.0 by 4.5 cm. The systolic blood-pressure was 112, the diastolic was 78. The radiogram revealed dextrocardia and transposition of the colon. The electrocardiogram showed the heart-rate to be 115; there was complete inversion of Lead I, and the amplitude of the *R* waves in Lead I were diminished to about one-half those of Lead III. The amplitude of the *R* waves in Lead III slightly exceeded those in Lead II. There was evidence of left ventricular preponderance (Figs. 8, 9 and 10).

The electrocardiograms of the last two cases essentially confirm the findings recorded in previous publications.[4 5 7 8] Lead I shows a complete inversion of all the deflections. Fig. 11 illustrates the angles produced by the direction of the leads and the resulting electrocardiograms. Case I (222329) shows the inversion also involving Lead II, and is explained by an exaggeration of the inclination of the cardiac axis to the right. The leads represent fixed planes of electrical potential, and changes in cardiac position or alterations in muscle bulk preponderance obviously affect the electrical currents, as expressed by the electrocardiograms.

It has been mentioned[6] that the *R* wave in Lead III becomes taller than in Lead II, but in these reported cases no constancy was observed. Hirschfelder mentions that the electrocardiographic curves sometimes are practically normal. Inversion of the deflections in Lead I is definite evidence of congenital dextrocardia with *situs transversus*, and we recognize electrocardiography as a valuable adjunct in the differential diagnosis of cardiac displacements.

REFERENCES.

1. Abbott, M. E.: Congenital cardiac disease. In: Mod. Med., Philadelphia, Lippincott, 1908, iv, 323.
2. Bryce, T. H.: Embryology. In: Quain's anatomy. New York, Longmans, 1908, i, p. 63.
3. Hirschfelder, A. D.: Diseases of the heart and aorta. Philadelphia, Lippincott, 1918, p. 562.
4. Hoke, E.: Ueber das Elektrokardiogramm eines Falles von Situs viscerum inversus totalis. München. med. Wchnschr., 1911, lviii, 802.
5. Lewis, T.: Electrocardiography and its importance in the clinical examination of heart affections. Brit. Med. Jour., 1912, i, 1421–1423.
6. Neuhof, S.: Clinical cardiology. New York, Macmillan, 1917, p. 32.
7. Nicolai, G. F.: Das Elektrokardiogramm bei Dextrocardie und anderen Lageveranderungen des Hersens. Berl. klin. Wchnschr., 1911, xlviii, 51–55.
8. Owen, S. A.: A case of complete transposition of the viscera, associated with mitral stenosis; including a description of the electrocardiographic tracings. Heart, 1911–1912, iii, 113–117.

AURICULAR FLUTTER

JOHN M. BLACKFORD, M.D.
SEATTLE, WASH.
AND
FRED A. WILLIUS, M.D.
MAYO CLINICS, ROCHESTER, MINN.

Sixteen cases of auricular flutter have been observed in the Mayo Clinic by us during the last thirty months. In the study of these cases a review of the literature revealed certain features which made it seem desirable to record our findings. The paucity of case reports in the literature is ample evidence that the condition is frequently overlooked.

Definition.—Auricular flutter may be described as an acceleration of the auricles to a rate beyond 200 per minute.[1] In all reported cases such acceleration has been accompanied by a partial heart block, giving a ventricular rate of one-half, one-third or one-fourth of the auricular rate, or a total dissociation of rhythm (complete heart block); or the degree of block may vary between the auricular beats, giving a gross ventricular arrhythmia. The partial block is apparently due to the inability of the auriculoventricular bundle to conduct impulses so rapidly, or to the inability of the ventricle to respond so rapidly. There is no reason to suppose that organic disease exists in the bundle except in those few cases (two, one our own, reported to date[2]), in which there is evident complete dissociation; and in a small group of ventricular bradycardias in which the auriculoventricular bundle may at least be questioned. In the paroxysmal attacks, when the ventricles assume the full auricular rate, we have evidence of a temporary increase in irritability of the ventricle or the auriculoventricular bundle.

There is no known pathologic difference between an auricular rate of less than 200 and one at which the rate exceeds this figure, yet the clinical manifestations are so different as to justify the classification of flutter as a clinical entity.[3] The fundamental clinical differences

1. Hertz, A. F., and Goodhart, G. W.: The Speed-Limit of the Human Heart. Quart. Jour. Med., 1908-1909, **2**, 213.
2. Jolly, W. A. and Ritchie, W. T.: Auricular Flutter and Fibrillation. Heart, 1910-1911, **2**, 177.
3. Lewis, T.: Observations on a Curious and Not Uncommon Form of Extreme Acceleration of the Auricle. "Auricular Flutter." Heart, 1912-1913, **4**, 171.

lie in the fact that flutter tends to persist indefinitely, whereas auricular paroxysmal tachycardia rarely reaches so rapid a rate and the attack stops after a relatively short period. In flutter the auricles continue their rapid rate when the ventricles are slower, while in auricular paroxysmal tachycardia 1-1 rhythm is always present and the sinus rhythm is restored between attacks.

Experimental.—Auricular flutter was produced experimentally by MacWilliam in 1887[4] by mild faradization of the auricles of exposed animal hearts. Lewis (1912-1913) observed the same condition after the intravenous injection of glyoxylic acid.[5] Hirshfelder[6] (1908) produced it by ligation of the coronary arteries, and similar observations have been made after cooling the auricles and during chloroform anesthesia. In our laboratories, working with Kendall, we produced, experimentally, hyperthyroidization in the goat by a large injection of the thyroid active principle, alpha-iodin.[7] We have observed auricular flutter as one of the cardiac phenomena shown by practically continuous electrocardiographic tracings over several hours preceding death.

Mechanism.—Flutter is caused by focus of stimuli in the wall of the auricular muscle at a point outside the normal pacemaker or sinus node (ectopic stimuli), the discharge of stimuli being at a rate so rapid and continuous as to submerge the sinus activity. This conclusion is based on the fact that the P wave is found to have an abnormal form in the clinical electrocardiogram and that in the experimental study the P wave approaches the normal contour as the stimulus is applied nearer the sinus node.

A statement of methods by which flutter can be produced in the laboratory will help to visualize the subject. A single shock applied with the stimulating electrode to any point in the wall of the auricle causes an auricular extrasystole, providing the stimulus is applied when the muscle is not contracting. A continued mild faradization applied to the same point causes similar contractions, but each contraction is maximal, and hence only when the muscle begins to relax, or pass out of the "refractory phase," is further stimulation effective; then another contraction is caused by succeeding stimulus, etc. In

4. McWilliam, J. A.: Fibrillar Contraction of the Heart. Jour. Physiol., 1887, **8**, 296.

5. Lewis, T.: The Mechanism of the Heart Beat with Special Reference to the Clinical Pathology. London, Shaw, 1911, p. 311.

6. Hirschfelder, A. D.: Contributions to the Study of Auricular Fibrillation, Paroxysmal Tachycardia, and the so-called Auriculo-(atrio) Ventricular Extrasystoles. Bull. Johns Hopkins Hosp., 1908, **19**, 322.

7. Kendall, E. C.: The Isolation in Crystalline Form of the Compound Containing Iodin which occurs in the Thyroid; its Chemical Nature and Physiological Activity. Tr. Assn. Am. Phys., 1915, **30**, 420.

other words, the auricle is contracting as rapidly as possible—a state of "flutter" during the time continuous faradization is applied.

Thus, if asphyxia is allowed to act, a visible change is found in auricular activity. Suddenly the auricle dilates and ceases coordinate contraction, but each little individual muscle bundle begins to contract regardless of the muscle mass; that is, incoordinated contraction. In other words, multiple foci of irritability occur throughout the auricular mass due to asphyxiation. The dilated auricle as a whole is functionless. It acts only as a reservoir, but close inspection reveals the fibril-

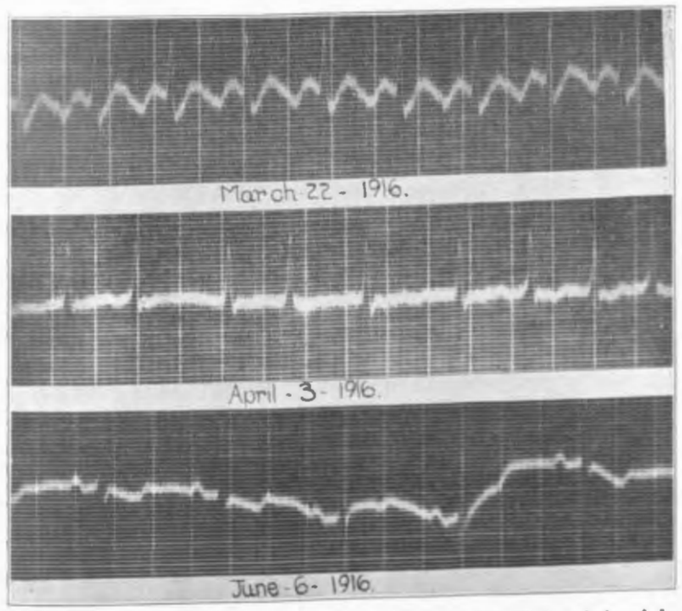


Fig. 1 (146153).—1. Typical auricular flutter. 2. Fibrillation induced by digitalis. 3. Normal rhythm following thyroidectomy.

lating muscle twitching which is characteristic of auricular fibrillation, and the total arrhythmia of the ventricular action is at once apparent.

In the human heart the irritable focus causing flutter must be the result of disease. It cannot be too strongly emphasized that flutter, per se, is only objective evidence of localized irritability in the auricular wall; and that any other organic cardiac disease may exist in the same heart.

It is evident that at present no clear distinction, mechanical or organic, can be given as differentiating paroxysmal auricular tachycar-

dia from auricular flutter. The difference is largely a well-grounded clinical conception based on a different symptomatology. The only objective distinction is one of auricular rate, and that a partial block usually exists in flutter cases.

Pathology.—It is already evident that auricular flutter is not a pathologic entity, for we often see auricular extrasystoles, flutter fibrillation and a sinus rhythm in a single case within a relatively short time. The literature contains only six necropsy reports in unquestioned cases; our series contains two others. Ritchie[8] has reported a lymphocytic infiltration of the epicardium, most marked in the region of the sinus node; and he thinks this may have depressed sinus activity. The cases of Gulland and Mackenzie,[9] and Hume's[10] first case add nothing significant to these findings. The pathologic findings are at present unimportant, since so little has been recorded.

The irritable auricular focus is the essential feature, and our study must include all causes of localized injury or irritability to heart muscle. Such causes may be classified under three heads: (1) infections causing localized injury; (2) general and local myocardial degeneration from any cause, as hypertension, valvular disease, goiter, etc.; and (3) localized malnutrition of the auricular wall as in coronary sclerosis, etc.

We do not know why in certain cases a localized injury should be selected from more extensive myocardial damage to become a source of irritation, and to send forth such rapid impulses as to submerge the sinus rate and establish flutter. That such functional pathology exists, however, is evident.

We have no evidence that flutter can be purely of neurogenic origin. In all the reported cases and in our own cases there was either objective evidence of other cardiac damage or a history indicating infectious, toxic, myocardial or coronary etiology.

Etiology.—Auricular flutter occurs four times as often in men as in women, counting the reported cases and our own. The average age of the patients was 47 years, the youngest 6 years and the oldest 82 years. The condition is most frequent between the ages of 40 and 60 years, but in our series more cases[7] occurred between 30 and 40 years of age.

8. Ritchie, W. T.: Further Observations on Auricular Flutter. Quart. Jour. Med., 1913-1914, **7,** 1.

9. Mackenzie, J.: Diseases of the Heart. Ed. 3, London, 1913, p. 105.

10. Hume, W. E.: A Polygraphic Study of Four Cases of Diphtheria with a Pathological Examination of Three Cases. Heart, 1913, 1914, **5,** 25.

Our cases at once call attention to an etiology of infection, since antecedent diseases of probable streptococcic origin were noted with remarkable frequency, namely, 1 rheumatic fever, 5 tonsillitis, 6 bad teeth, 6 "grippe," 2 pneumonia. In 3 cases the patient dated his symptoms from one of these infections. All the patients in our series gave histories of one or more of the foregoing diseases. In 59 reported cases there is little data on this phase of the subject, but when given, the streptococcus group predominates; thirteen histories of rheumatic fever are recorded.[11] Venereal disease plays no evident part.

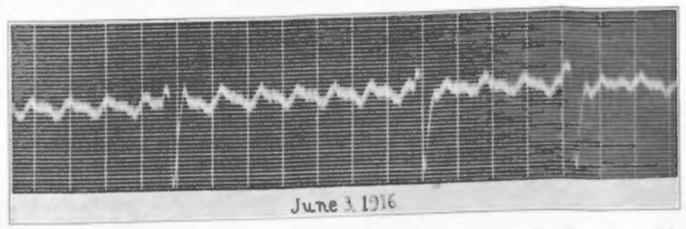


Fig. 2 (161186).—Auricular flutter with heart block; auricular rate 260; ventricular rate 43.

None of our patients was syphilitic, though three cases are noted in the literature.[12] We noted four histories of typhoid fever; other reports contained one case.[13]

Exophthalmic goiter was definite in four of our cases and was believed to be the probable etiologic factor. One other such case is reported.[14] Mitral disease was observed in but one of our sixteen

11. Mathewson, G. D.: A Case of Auricular Flutter. Edinburgh Med. Jour., 1913, **11,** 500. Ritchie, W. T.: Further Observations on Auricular Flutter. Quart. Jour. Med., 1913-1914, **7,** 1. Gunson, E. B.: Auricular Flutter Followed by Paroxysmal Auricular Fibrillation. Lancet, London, 1914, **2,** 151. Levine, S. A., and Frothingham, Jr., C.: A Study of a Case of Auricular Flutter. The Archives Int. Med., 1915, **16,** 818. Neuhof, S.: Auricular Flutter Accompanying Acute Endopericarditis. Med. Rec., New York, 1915, **88,** 995. Tallman, M. H.: Auricular Flutter. Northwest Med., 1916, **15,** 145. Sutherland, G. A.: Auricular Flutter in Acute Rheumatic Carditis. Brit. Jour. Child. Dis., 1914, **11,** 337. Ritchie, W. T.: Auricular Flutter. Edinburgh, Green, 1914, p. 33. Mackenzie, J.: Diseases of the Heart. Ed. 3, London, 1913. Appendix, Cases 68 and 69. Quoted by Ritchie, Footnote 14.

12. Gunson, E. B.: Auricular Flutter Followed by Paroxysmal Auricular Fibrillation. Lancet, London, 1914, **2,** 151. Ritchie, W. T.: Auricular Flutter. Edinburgh, Green, 1914, p. 33. Cowan, J.: Diseases of the Heart. London, Arnold, 1914, p. 205.

13. Tallman, M. H.: Auricular Flutter. Northwest Med., 1916, **15,** 145.

14. Ritchie, W. T.: Auricular Flutter. Edinburgh, Green, 1914, p. 33. Sutherland, G. A.: Auricular Flutter in Acute Rheumatic Carditis. Brit. Jour. Child. Dis., 1914, **11,** 337.

patients, though the literature[15, 2] reports ten cases of stenotic or double mitral lesions.

Relative Incidence.—We examined electrocardiographically 3,500 patients and observed 16 auricular flutter records. There were 363 patients with auricular fibrillation, 160 showing auricular extrasystoles, 316 showing ventricular extrasystoles and 5 auricular paroxysmal tachycardia. These figures are doubtless far from a fair average, because we examined a great number of patients suffering from toxic goiter (both hyperplastic and nonhyperplastic) and fibrillation is very common in such cases. The proportion of 16 flutters to 363 fibrillations is probably a fair average.

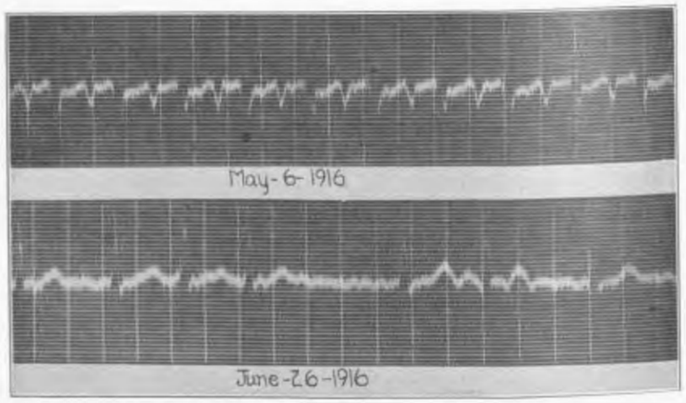


Fig. 3 (158953).—1. 2-1 flutter; auricular rate 316; ventricular rate 158. 2. Fibrillation induced by digitalis.

Symptoms.—The symptoms of the condition depend essentially on the ventricular rate and the cardiac compensation. The symptoms do not depend on the auricular rate alone, for the auricles may be found at 320 and the patient may not be aware of serious trouble, or the auricles may be dilated and functionally inactive (fibrillation) yet with good cardiac compensation and with little or no discomfort. Symptoms are further confused by disease to which the flutter is incidental or terminal, as in mitral disease, arteriosclerosis or chronic nephritis. In

15. Lewis, T.: Observations on a Curious and Not Uncommon Form of Extreme Acceleration of the Auricle. "Auricular Flutter." Heart, 1912-1913, **4**, 171. Mackenzie, J.: Diseases of the Heart. Ed. 3, London, 1913, p. 105. Ritchie, W. T.: Auricular Flutter. Edinburgh, Green, 1914, p. 33. Rihl, J.: Klinische Beobachtungen über atrioventriculäre Automatie mit Bradykardie. Ztschr. f. exper. Path. u. Therap., 1911, **9**, 496. Gibson, G. A.: A Discussion on Some Aspects of Heart Block. Brit. Med. Jour., 1906, **2**, 1113.

such cases flutter is clearly a manifestation of serious nutritional disturbance in the auricular wall and should be regarded only as a symptom worthy of relief.

Flutter cases may be conveniently classified as paroxysmal or chronic, depending on the duration of the disorder. We use the term "paroxysmal" in cases in which the normal rhythm is restored between attacks lasting a few hours or days and "chronic" when the condition tends to persist.

Paroxysmal flutter is not clearly defined from auricular paroxysmal tachycardia, as before mentioned. We have observed it only as a disorder incidental to evident myocardial disease; it is serious because of the great strain on the myocardium. Short paroxysms of flutter occur in which the auricular rate is between 200 and 380 and the ventricular rate bears a definite or indefinite ratio to the auricular. The attack gives symptoms of cardiac embarrassment varying in degree with the cardiac compensation and the length of the attack. Palpitation, tachycardia, flushing, breathlessness, weakness, flatulence, pallor, vertigo, polyuria, faintness and syncope come on as the attack progresses, though sudden relief may come at any time from cessation of the attack. We have seen, alternately, attacks of flutter and fibrillation in the same patient.

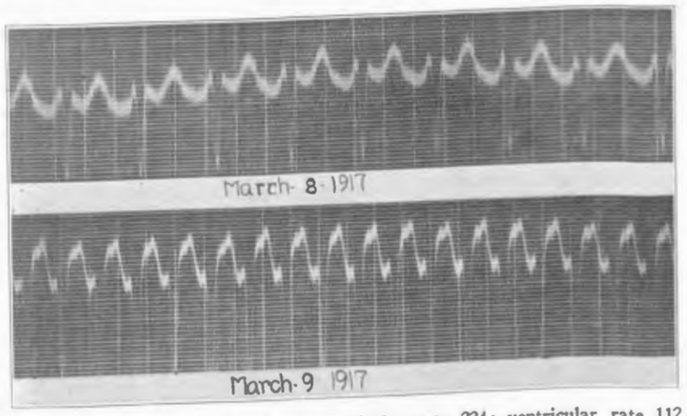


Fig. 4 (187575).—1. 2-1 flutter; auricular rate 224; ventricular rate 112. 2. Paroxysm of 1-1 flutter; rate 232.

Chronic flutter should always be recognized, for it can usually be relieved. The flutter lasts for long periods, for weeks or even years, and can be detected by proper tracings at any time during its course. The ordinary auriculoventricular ratio is 2-1 and the pulse is usually

100 to 180, but any degree of block may exist. The pulse, therefore, may vary from idioventricular rhythm, 32, to the full auricular rate, 320.

The most constant symptoms in our cases have been persistent and obscure tachycaria and weakness. Most of the patients are subject to "weak spells"—violent paroxysmal attacks of tachycardia with acute cardiac insufficiency brought on usually by exertion and by stopping suddenly. In such paroxysms the ventricles assume or approximate the auricular rate. Certain patients have described it as "a feeling like a bird fluttering in the chest," which is probably a fairly characteristic sensation. An occasional patient is very little inconvenienced by the paroxysm and in such instances the trouble is most likely to be overlooked.

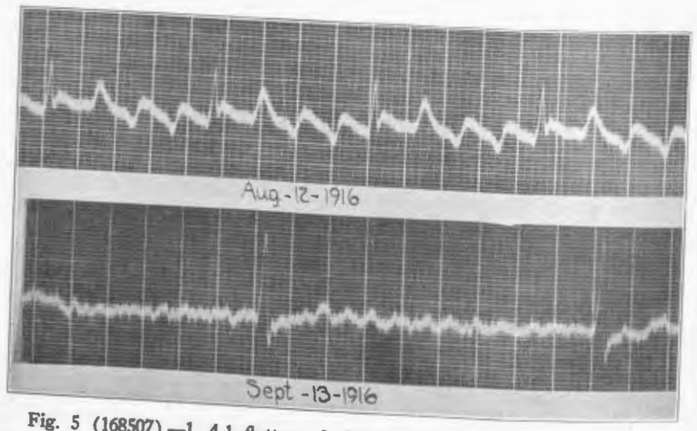


Fig. 5 (168507).—1. 4-1 flutter. 2. Fibrillation with complete heart block under digitalis.

Objectively, the heart may be normal except for weak sounds and a tick-tack rhythm. Usually there is a tachycardia more or less marked, and often there is mitral stenosis. In one half of our cases the pulse has been regular and in the others markedly irregular, owing to regular or irregular conduction through the auriculoventricular bundle. Rapid, regular venous pulsation in the neck is indicative of the disorder; and any patient more than 30 years of age with a tachycardia unaccounted for, and particularly if he is subject to "weak spells," should be under suspicion.

Vagal Pressure.—In certain cases vagal pressure will promptly reduce the ventricular rate by increasing the degree of block; but the auricular rate is not affected by this procedure, contrasting sharply

with the cases of auricular tachycardia in which sinus rhythm is suddenly restored by vagal pressure. The slowing of the ventricle is but transitory, and is recognized as due to increasing the degree of block temporarily by causing vagal depression of the auriculoventricular bundle.

The diagnosis rests finally on graphic tracings, and we believe the electrocardiograph to be far the most satisfactory. While we disclaim expert knowledge of the polygraph, we feel sure that the findings with this instrument may easily be misinterpreted and are not invariably conclusive.

SUMMARY OF CASES

Following is a brief summary emphasizing the interesting features in our cases:

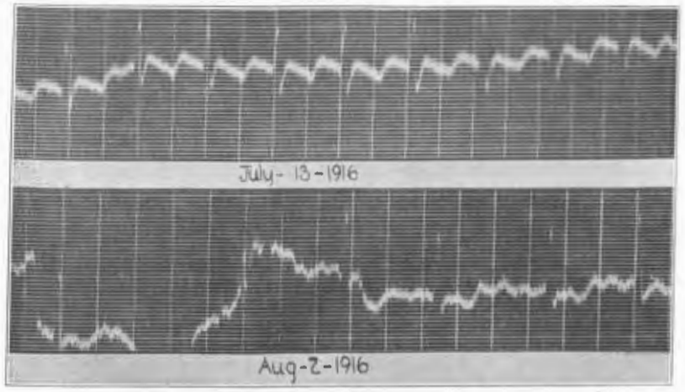


Fig. 6 (92324).—1. Auricular flutter; auricular rate 292; ventricular rate 146. 2. Fibrillation induced by digitalis; rate about 120.

Case 119641.—A man, aged 39, came for examination Nov. 24, 1914. He had suffered with hay-fever for years, and with asthma during the past year. He came to the clinic during a paroxysm of tachycardia which had existed for two days. The pulse was 212 constantly, with marked evidence of cardiac embarrassment. These attacks had occurred twice within the year and had lasted six and eight days, beginning and terminating abruptly. He was unable to retain any medicine, and in spite of the treatment died on the fifth day of his attack, three days after his arrival. Before death the heart was greatly dilated and the pulse varied instantly from slow to rapid rhythm. No tracings were possible after the first day. No valvular lesion was demonstrated clinically or pathologically.

Case 142236.—A man, aged 55, was admitted to the clinic Sept. 28, 1915. The patient had been subject to "grippe," and five years before coming for examination had noticed dyspnea and spells of syncope with sudden onset. He would fall whenever and wherever the spells came on. At this time he rested six weeks and improved. One year later (July, 1913) he had another attack which

lasted six weeks and which necessitated two weeks in bed. April, 1914, another attack lasted a month, after which his health was good until July 7, 1915, when a sudden attack came on while he was eating supper. This had persisted since with violent attacks of syncope. During the first examination in the clinic the patient suddenly went into collapse. He became cyanotic, pulseless and unconscious; there was extreme pallor and cold sweat. Death seemed imminent, when the idea of vagal pressure suddenly occurred to one of us. Pressure on the right vagus suddenly reduced the pulse rate (see cardiograms of similar pressure in another attack) to about 55, with prompt recovery of consciousness. Fibrillation was induced three times during two months by digitalis therapy, only to have the flutter recur after its discontinuance. The patient's fortitude in attempting a fourth course of treatment was rewarded by a return to a sinus rhythm on Christmas eve, and eighteen months later he reported that he was

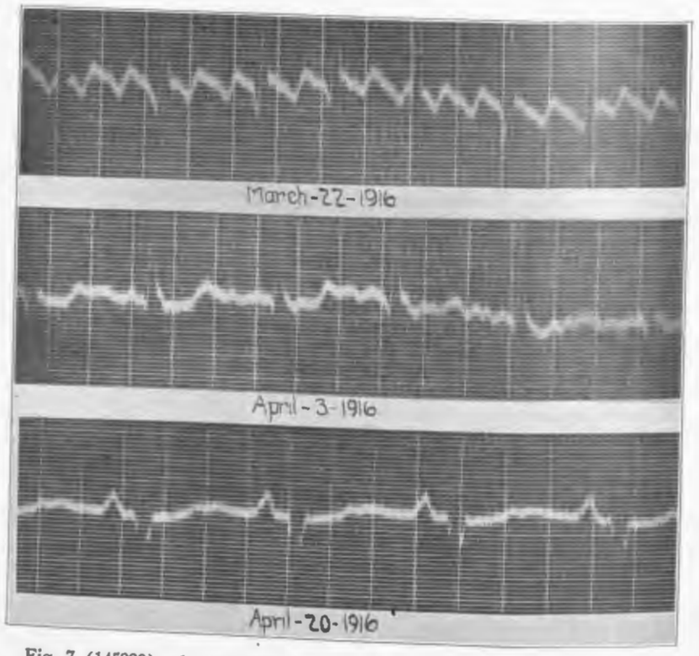


Fig. 7 (145990).—1. Auricular flutter; 2-1 rhythm; auricular rate 340; ventricular rate 170. 2. Fibrillation induced by digitalis. 3. Sinus rhythm following thyroidectomy; rate 92.

doing light farm work. The heart was normal objectively between attacks except for occasional auricular extrasystoles.

Case 142802.—A woman, aged 32, was admitted to the clinic Oct. 6, 1915. She had had repeated tonsillitis and a probable exophthalmic goiter. She came for relief from biliary colic. For several months she had been conscious of a "fluttering heart" and had had several spells of violent palpitation, usually following exertion. Electrocardiograms showed a 320-160 rate ordinarily. While under observation she came into the office with a ventricular rate of 320 (counted

by stethoscope) during a paroxysmal attack lasting nearly two hours. She had walked several blocks to the office and was not sufficiently inconvenienced during the first hour to make complaint. The rapidity was discovered on examination, when the ventricular rate was found to have fallen to 300. She walked up one flight of stairs for a cardiographic examination during the attack, which stopped suddenly while the examination was in progress and the usual 2-1 rhythm was reestablished. This case is unique in that it is the fastest human

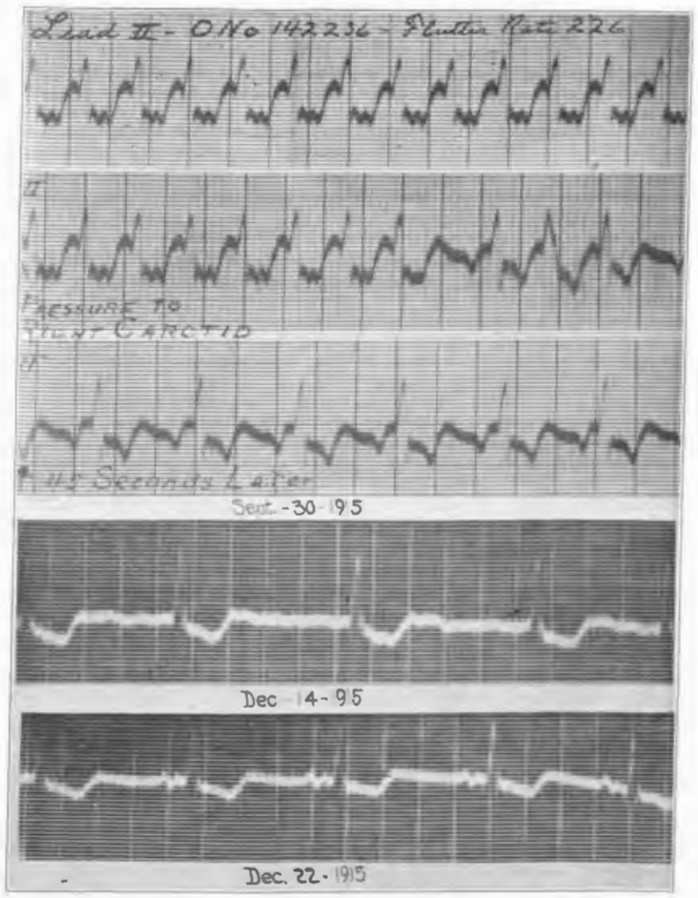


Fig. 8 (142236).—1. Paroxysm of 1-1 flutter with 45 second intervals between the three strips; rate 224; pressure applied to right vagus indicated by arrow at beginning of second strip; 2-1 flutter established within a few seconds, as indicated in third strip. 2. Fibrillation induced by digitalis; rate; 88. 3. Sinus rhythm restored; rate 71; definite abnormal auricular rate.

ventricular rate yet recorded. The patient said she had suffered from such attacks repeatedly and that the present attack was milder than many of the others.

Under digitalis medication fibrillation was induced but on its withdrawal flutter returned. A second trial resulted similarly; a third attempt was made, and the patient while fibrillating was referred for cholecystectomy, which was successfully accomplished. A sinus rhythm was established some days later (rate 120) and persisted for four months, until the flutter returned following heavy work cleaning house. We obtained further tracings May 1, 1916, showing flutter, but the patient could not remain for treatment.

This case had been diagnosed as exophthalmic goiter before the patient came to us and she was anxious to have a thyroidectomy. We wished to defer operation on account of the heart condition and the questionable diagnosis of hyperthyroidism, and thyroidectomy was performed elsewhere (November, 1916). Pathologic examination of the tissue, which the surgeon kindly sent to us, showed the typical hyperplastic changes of exophthalmic goiter. The patient reports a pulse practically normal and health restored since operation.

Case 145990.—A woman, aged 41, came for examination March 21, 1916. The patient had had tonsillitis repeatedly. She had had the classical symptoms

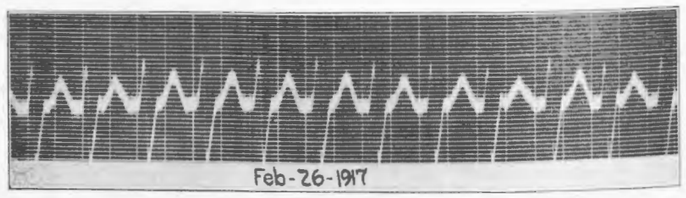


Fig. 9 (130119).—Auricular flutter; auricular rate 300; ventricular rate 150.

of exophthalmic goiter for four years. Three months previously ligations had been done in the clinic. No cardiac disorder was then suspected. On returning for thyroidectomy she was prostrated by a prolonged paroxysm of tachycardia. The cardiogram revealed flutter. She was treated with digitalis, and thyroidectomy was done while the heart was fibrillating. Sinus rhythm was restored soon after operation. She gave the interesting history of a prolonged paroxysm of tachycardia the year before, during which syncope was so complete that she was thought to be dead. Aug. 10, 1917, the patient reported marked improvement in general health and no more spells of tachycardia.

Case 146153.—A man, aged 26, entered the clinic March 18, 1916. There was no history of previous infections, except typhoid in 1912. The tonsils appeared large and unhealthy. Classical exophthalmic goiter with symptoms had existed for nine years; relatively mild symptoms until the last two years. He had had three spells of vomiting and prostration, probably due to flutter. The heart action had been irregular and rapid but no record was taken before the ligations were done here Nov. 26, 1915. At the present examination the cardiogram showed typical auricular flutter. Heavy digitalis medication, 29 c.c. in ten days, brought on fibrillation. Thyroidectomy was performed April 10, 1917, and sinus rhythm was restored shortly after. The patient reports Aug. 7, 1917, "I have forgotten all about my heart."

Case 168507.—A man, aged 43, was examined Aug. 9, 1916. The patient gave a history of typhoid and he was subject to tonsillitis. He came to the clinic because of recurrent attacks of appendicitis. He appeared healthy. A cardiographic examination was made on account of the irregular pulse. After discovering the flutter we elicited a good history of sudden attacks of mild

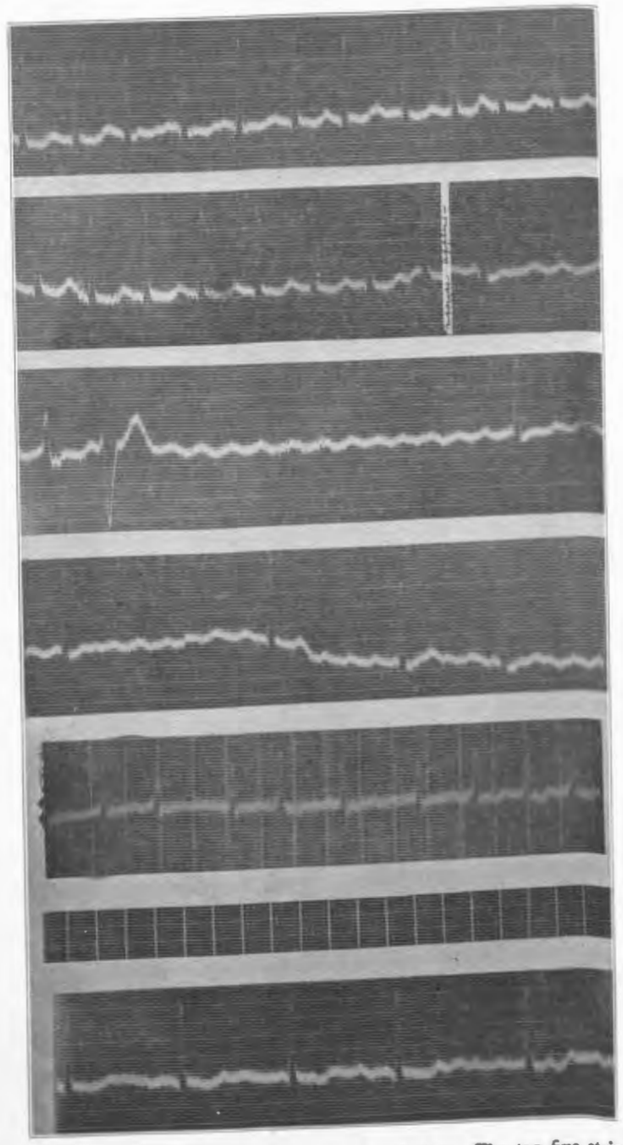


Fig. 10 (195883).—1. Auricular flutter; 2-1 rhythm. The top five strips are continuous tracings cut for reproduction. Pressure applied at point indicated by interruption of light; cessation of ventricular action 4.5 seconds in third strip on release of pressure; series of ventricular extrasystoles as shown in fifth strip before resumption of normal rhythm. 2. Lowest strip shows fibrillation under digitalis.

syncope with rapid heart action, brought on by marked exertion during the previous five years. Mild chronic dyspnea was also admitted. The patient was treated thirty days, taking 100 c.c of digitol before complete heart block and fibrillation were induced. He left us then, but he has reported through his brother (Aug. 12, 1917) that he feels well. The appendix has not been removed.

Case 158953.—A man, aged 61, was examined May 5, 1916. Twelve months before admission, following grippe and mumps, the patient had had "nervous prostration," chief symptoms, nervousness, weakness and palpitation. His physician told him the rapid pulse was due to "nervousness." November, 1915, there was again a sudden onset of rapid heart and weakness which has persisted to date. He had "weak spells" and was dizzy. With rapidity of the heart hoarseness was noticed. He was treated with 69 c.c. digitalis twenty-three days before the onset of fibrillation. Flutter recurred and the treatment was again instituted for a few days until the onset of fibrillation. A few days later there was a second recurrence of flutter and again fibrillation was induced by digitalis. Tonsillectomy was performed July 10 without incident. The patient continued

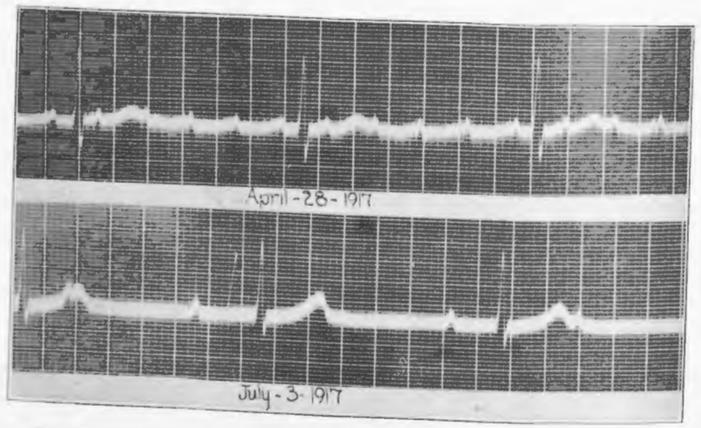


Fig. 11 (192392).—1. Auricular flutter with complete heart block; 5-1 rhythm. 2. Complete heart block.

fibrillation without cardiac symptoms up to the last report. From subjective sensations he was able to say accurately whether flutter or fibrillation was present. No evidence of cardiac hypertrophy or valvular disease was obtainable at any time.

Case 161186.—A man, aged 75, a feeble, stiff, fat old man with marked sclerosis of the peripheral vessels and evident cardiac insufficiency, was examined May 31, 1916. He dated rheumatic symptoms back to rheumatic fever twelve years and four years previously. Dropsy had been noted for a year but not much dyspnea or palpitation, probably because the rheumatism made exertion difficult. Blood pressure 170-82 and pulse varying from 44 to 68, indicating impaired auriculoventricular conduction. There was no valvular disease evident and the heart was not definitely large. Treatment by rest and digitalis at home was recommended.

Case 92324.—A woman, aged 39, was admitted for examination July 12, 1916. Definite exophthalmic goiter; ligation performed in the clinic, October, 1913. At this time the pulse was regular, 95 to 118, and the heart greatly dilated.

Thyroidectomy was performed, February, 1914, after marked improvement in the heart condition. The patient returned the second time for observation. All of the previous winter she suffered from frequent colds and grippe. There was no history of syncope. The first cardiogram taken showed flutter with 2-1 rhythm, ventricular rate 196. Fibrillation was readily brought on with digitalis and continued during fifty-six days of observation.

Case 130119.—A man, aged 52, was admitted for examination Feb. 17, 1917. The patient had been operated on in the clinic, May 6, 1915, under local anesthesia for a small cyst of the tongue, but no general examination had been made at that time. The patient was very obese, with evident marked cardiac

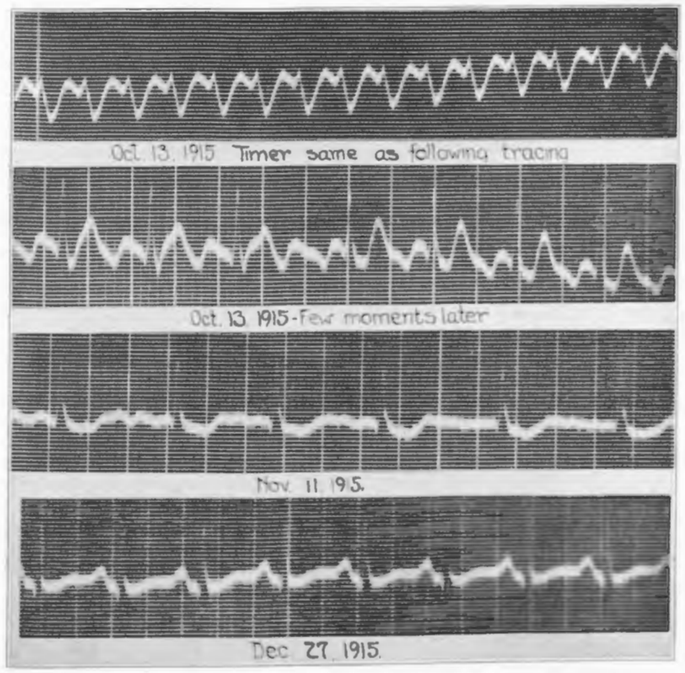


Fig. 12 (142802).—1. Paroxysm of auricular flutter showing ventricular rate slightly above 300; timer same as next tracing. 2. A few seconds later showing break in attack during the time necessary to change plates. 3. Auricular fibrillation induced by digitalis. 4. Sinus tachycardia; rate 130 following cholecystectomy and tonsillectomy. This patient was operated on subsequently elsewhere for exophthalmic goiter four months following and at present reports normal heart action and pulse rate.

insufficiency, and the roentgen ray showed diffuse dilatation of the aorta and the heart greatly enlarged. Dyspnea had been noted twelve years, but marked cardiac insufficiency followed grippe last fall. The cardiogram showed flutter. The patient died suddenly on the fourth day in a coughing attack. Necropsy revealed a fatty, dilated heart, and fatty sclerosis and dilatation of the aorta.

CASE 195883.—A woman, aged 43, was admitted for examination July 1, 1917. The patient was subject to grippe. She had had ten children. The rapid heart action had been first noticed fourteen years previously during pregnancy. There had been palpitation since on exertion, and last year there were repeated spells of very rapid heart action with palpitation. Objectively the patient showed marked tachycardia (192). Pressure on the right vagus immediately slowed the pulse to about 60; releasing the pressure permitted the pulse go back to 192. She was treated with large doses of digitalis, and fibrillation was brought on in a few days. Fibrillation continued since. A slower pulse is definite evidence of double mitral lesion.

CASE 187575.—A man, aged 45, was admitted for examination March 8, 1917. He had had typhoid twenty-seven years previously. For two years he had been subject to attacks of dyspnea and pain in the precordium radiating to the epigastrium. Worse attacks lasted one-half hour, causing complete incapacity. The heart enlarged objectively but no valvular defect was noted. The electrocardiogram showed typical flutter, auricles 224, ventricles 112. The second examination showed ventricles 224, and a 1-1 rhythm. The patient was unable to remain for treatment.

CASE 192392.—A man, aged 37, was admitted for examination Aug. 25, 1917. He gave a history of a questionable sore on the penis at the age of 17, but we were unable to establish syphilis on a most searching investigation. Apparently the man was strong and healthy. There had been mild dyspnea on exertion for two years, but no definite attacks. A month previously a sudden syncope followed very heavy lifting and the "right arm went dead for a little while." The heart was objectively negative except for a slow pulse (46). The electrocardiogram showed flutter, auricles 225 and ventricles 46. To rule out syphilis a therapeutic test was given, with no result after three weeks of treatment. Digitalis was started and the next morning a second cardiogram showed a sinus rhythm with complete disassociation of ventricles (46) and auricles (72). The digitalis was stopped at once. The condition had persisted since (three months). No further medication was used except potassium iodid for empirical reasons. The patient suffered no subjective inconvenience and had no evidences of a Stokes-Adams syndrome.

CASE 145519.—A thin, asthenic woman, aged 30, was admitted for examination Aug. 2, 1917. Following pneumonia five years previously she had suffered with repeated weak spells associated with rapid heart action. The pulse was always rapid, and at the examination averaged 133. Electrocardiogram showed auricular flutter, auricles 266, ventricles 133. This patient is to return for treatment.

CASE 204974.—A man, aged 37, was admitted for examination Aug. 17, 1917. The patient had been asthmatic all his life, but we were unable to elicit a history of previous infection. He was a laborer and looked robust. The heart had been very rapid for three years when at work, but there was no history of weak spells. He was prepared for a cardiogram on account of irregular pulse. Auricles 258. Ventricles 172. This patient is to return for treatment.

CASE 203875.—A thin man, aged 52, was admitted for examination Aug 6, 1917. He was subject to colds. Eight years previously he had an attack called "bilious" for three weeks. The muscles were contracted and there was debility and weakness with sudden onset. Morphin relieved the attack. Since then repeated lighter "bilious spells" occurred and morphin was given to prevent the spasm. These attacks occurred at intervals of weeks or months. Podophyllum or morphin relieved the attacks. There was mild chronic dyspnea and the electrocardiogram showed flutter. The patient continued under treatment.

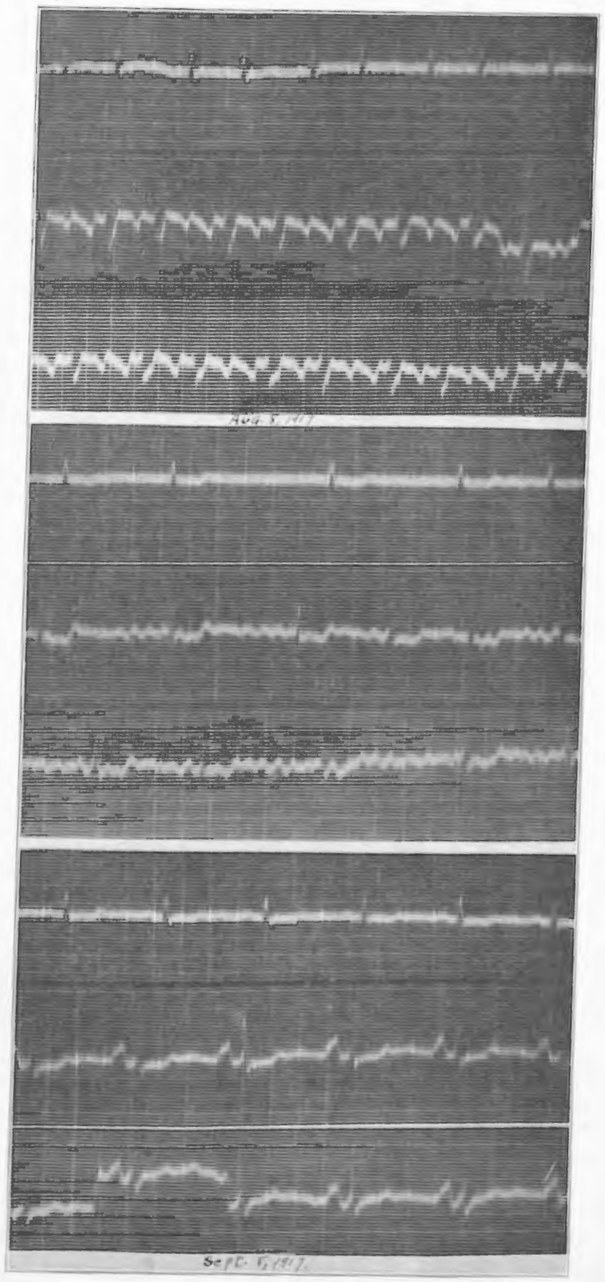


Fig. 13 (203875).—1. Paroxysm of auricular flutter showing ventricular rate of 120. 2. Twenty-six days later showing auricular fibrillation after heavy digitalis dosage. 3. Sinus rhythm five days later.

Treatment and Results.—As Lewis has shown, digitalis has proved to be the sovereign remedy in cases of flutter. We have used the drug in large doses, and have treated ten patients, not counting the two who were moribund and who died before any effect of medication was possible. Two patients (Cases 161186 and 192392) were not treated for obvious reasons, and two (Cases 204974 and 187575) were unable to stay for treatment.

The amounts of digitalis administered and a summary of the results are given in the accompanying table. All the patients were in the hospital at complete rest during the digitalis medication. Digitalis broke the flutter in all ten patients, and four finally resumed and held a normal rhythm. The others were markedly improved subjectively by the onset of fibrillation.

TABLE SHOWING AMOUNTS OF DIGITALIS GIVEN AND SUMMARY OF RESULTS

Case	Onset of Fibrillation		Onset of Toxic Effects	
	Days	Amount, C.c.	Days	Amount, C.c.
142236	75	280	51	185
142802	42	220	26	180
145090	60	94	0	0
146153	8	29	0	0
158953	51	100	22	70
922324	4	12	0	0
168507	23	100	0	0
195833	16	51	0	0
203875	24	190	22	190

The most important point in the treatment is to use enough digitalis. We have run up the dose rapidly to the physiologic tolerance of the patient. Our maximum dosage was 10.5 c.c. daily for ten days before toxic effects were evident. We have seen no bad results from such massive dosage except the temporary toxic symptoms, and we feel sure that most patients require massive dosage to obtain the desired result. Further, we have found better results from pushing the drug to physiologic complete block if the patient tolerates it to this point; that is, far beyond the point of fibrillation in most instances. In all our patients we have produced marked poisoning before discontinuing the drug. In the four cured patients we repeatedly had the fibrillation break back to a flutter until digitalis poisoning was produced.

The after-treatment is important. We discontinue medication for cured patients, but have advised in fibrillating cases that digitalis be used, if necessary, to keep the pulse averaging below 80 when at rest. The usual general advice to cardiopaths should always be emphasized.

Operability.—One operation is reported in the literature—a death on the table under chloroform anesthesia attributed to flutter. We believe that if surgical treatment is indicated, particularly the removal of a probable source of infection or toxemia, the risk should be accepted. All operations have been performed after inducing fibrillation by digitalis and while the patient was under medication. We have been careful not to have atropin administered before operation, since this would temporarily abolish the vagus stimulation of the digitalis.

Three of our patients had exophthalmic goiter, and in two of these sinus rhythm and apparently normal health were regained after thyroidectomy. The third was a bad cardiopath, but sinus rhythm was restored, though the patient still has some symptoms of cardiac insufficiency.

Removal of the tonsils seemed advisable in cases in which there was evident focal infection, and this was done in two instances. One patient improved greatly; the other is subjectively cured. Cholecystectomy was done in one case.

SUMMARY

Auricular flutter occurs as a mechanical disorder in certain diseased hearts, and in our experience has been most frequently associated with exophthalmic goiter.

The paroxysmal attacks noted in fourteen of our sixteen cases are dangerous to health and even to life. Two patients died in such attacks and two others appeared so nearly dead as to deceive competent observers. The patient with flutter is always in danger of such attacks. He should always have the disorder arrested as soon as possible after its discovery.

In our experience efficient treatment may be relied on to cause the onset of fibrillation and greatly to relieve the patient. None of our patients is known to have had a recurrence of flutter after his dismissal.

Three patients were operated on under ether anesthesia for exophthalmic goiter after fibrillation was established, and one of these and one other have had tonsillectomies (local anesthesia) performed without incident. Cholecystectomy has been performed in one case.

All of the ten treated patients are alive and much improved or cured with treatment. Five report that they cannot detect any cardiac symptoms.

Reprinted from the Archives of Internal Medicine
January, 1918, Vol. XXI, pp. 147-165

AMERICAN MEDICAL ASSOCIATION
FIVE HUNDRED AND THIRTY-FIVE NORTH DEARBORN STREET
CHICAGO