THE FAVORABLE PROGNOSIS OF AURICULAR FIBRILLATION*

T. HOMER COFFEN, M.D.
PORTLAND, ORE.

Auricular fibrillation in man has generally been considered a result of myocardial damage.[1] Observations in many cases by many observers make it appear that heart function may not be seriously impaired by this rhythm. Whether this abnormal cardiac rhythm, resulting from profound disturbance in the auricles, throws an added burden on the heart's efficiency, or whether it is one of several unimportant signs of impaired cardiac function, is worthy of consideration. Symptoms and signs of decreased cardiac reserve are found in hearts without this rhythm; etiologic and predisposing factors may be the same as are found in auricular fibrillation, and yet the patients present very

* Read before the Section on Practice of Medicine at the Seventy-Fourth Annual Session of the American Medical Association, San Francisco, June, 1923.

1. Years ago, physiologists noted that there was fibrillation of the ventricles just before death in experimental animals, and that fibrillation of the auricles was less apt to be fatal, since the auricles recover normal rhythm more easily than the ventricles. "The auricles, when in a state of fibrillation, present an entirely different aspect than during normal action; the walls of the auricle stand in diastolic position; systole, either complete or partial, is never accomplished; the wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement; rapid, minute and constant twitching or undulatory movements are observed in a multitude of small areas upon its surface" (James Mackenzie: Diseases of the Heart, 1914, p. 216). "The movement as a whole is more or less incoordinate, and therefore ineffectual" (Thomas Lewis: Mechanism and Graphic Registration of the Heart Beat, 1920, p. 444). The story of the discovery that this form of arrhythmia is due to fibrillation of the auricle is nothing short of dramatic. Lewis' explanation of a circuituous pathway of the contraction impulse in the auricular wall, and the more recent use of quinidin, have reawakened interest in the subject. While there are now numerous references to the use of quinidin in auricular fibrillation, the following are among the first to appear: Levy, R. L.: Restoration of the Normal Cardiac Mechanism in Auricular Fibrillation by Quinidin, J. A. M. A. **76**: 1289 (May 7) 1921. Lewis, Drury, Iliescu and Wedd: Restoration of Normal Cardiac Mechanism in Auricular Fibrillation, Brit. M. J. **2**: 514 (Oct.) 1921. White, P. D.; Marvin, H. M., and Burwell, C. S.: The Action of Quinidine Sulphate in Heart Disease to Abolish the Circus Movement of Auricular Flutter and Fibrillation, M. Rec. **185**: 647 (Dec. 1) 1921. Lewis, Thomas: The Value of Quinidine Sulphate in Cases of Auricular Fibrillation, Am. J. M. Sc. **163**: 781 (June) 1922; The Action of Atropine and Quinidine in Fibrillation of the Auricles, ibid. **164**: 1 (July) 1922.

similar clinical courses. Some hearts develop this rhythm so insidiously that the patient is but little inconvenienced, even though it becomes permanent. Others, after a period of decompensation coincident with the inception of the new rhythm, are tided over and compensate well for years. Still others show brief paroxysms of this arrhythmia, with spontaneous restoration of normal rhythm. Again, it may appear in young hearts in the course of decompensation incidental to acute endocarditis, disappearing with a return of compensation.[2] These facts make it appear that the condition, per se, may not be the cause or the effect of myocardial disease.

INCIDENCE

Auricular fibrillation is the most frequent of all irregularities of the heart.[3] From 60 to 90 per cent. of

"Arrhythmia perpetua" had been noted years before, and Nothnagel described a case in 1878, which we now recognize as auricular fibrillation, as noted by Hamburger (The Recognition and Treatment of Different Forms of Auricular Fibrillation, M. Clin. N. Am. **5**: 1705 [May] 1922). The condition was recognized as an entity by Mackenzie as early as 1890, but he thought it due to irregular simultaneous contractions of auricles and ventricles from a stimulus arising in the auriculoventricular node, and so termed it "nodal rhythm." He noted the disappearance of the auricular wave in jugular tracings. Cushny, in 1906, noted the similarity between tracings from dogs with auricular fibrillation and human tracings of paroxysmal irregularities, and suggested that this might be a factor of clinical importance; on a visit to Mackenzie, he suggested that auricular fibrillation might be identical with "nodal rhythm." Mackenzie published polygraph tracings with this explanation in 1907, but stated that he "failed to appreciate what auricular fibrillation really was." Lewis had been investigating cardiac irregularities clinically, and experimentally producing fibrillation of the auricles in the dog, and noted in 1909 that with the onset of fibrillation the venous curves changed from the auricular to the ventricular form, and the electrocardiograms, especially in Lead III, showed certain oscillations during ventricular diastole, produced by the fibrillating auricles. Examining some of the electrocardiograms of typical cases of "nodal rhythm" sent him by Mackenzie, he found these oscillations present and demonstrated their correspondence with the small fibrillation waves. Rothberger and Winterberg also showed the electrocardiographic relation between fibrillation in man and animals in the same year.

2. An illustration of the insidious development of auricular fibrillation, with no subjective or objective symptoms or signs, is seen in Case 1. Fibrillation developing during an acute cardiac breakdown incidental to chronic endocarditis is seen in Case 27, while fibrillation found a year after an acute breakdown is seen in Case 26. Case 17 is probably of fifteen years' duration, and yet the patient, now 69 years old, is comfortable and ambulatory. Cases 15 and 35 illustrate paroxysmal auricular fibrillation, with restoration of normal rhythm between attacks; in the first, a comparatively young man, there are but few signs of disturbed cardiac function, while in the second, a man of 69, pulmonary edema of alarming severity has attended the attacks. Mackenzie (Diseases of the Heart, pp. 217 and 229) refers to a patient, a physician, aged 50, seen in a transient attack. He was under observation for ten years, remaining well and with a regular heart. Bishop (J. A. M. A. **79**: 196 [July] 1922) refers to patients under observation fifteen years in comparative comfort with permanent fibrillation.

3. According to various authors, the arrhythmias are found in the following frequency: auricular fibrillation, 48 per cent.; premature contractions, 44 per cent.; sinus irregularities, 14.4 per cent.; disorders of conduction, 4.15 per cent.; flutter of auricles, 1.1 per cent. According to Lewis, the last three types of arrhythmia form about 25 per cent. of all.

decompensated hearts, and most irregular hearts with a rate of 120 or over, are probably in this condition.[4] Flint observes that when there is no history of rheumatism, the condition is associated with advancing years, occurring most frequently in the sixth to the seventh decades. It is the usual arrhythmia accompanying decompensation in rheumatic mitral stenosis.[5]

TABLE 1.—*Relation of Fibrillation to Sex, According to Various Authors*

Author	Men, Per Cent.	Women, Per Cent.
Lewis (Cohn, in Nelson's Loose Leaf Medicine 4: 333, 1920) 189 cases	60	40
Semerau (Cohn, in Nelson's Loose Leaf Medicine 4: 333, 1920) 193 cases	70.15	29.85
Willius (Clinical Electrocardiography, 1922, p. 66) 500 cases	53.6	46.4
Levine (Am. J. M. Sc. 154: 43 [July] 1917) 128 cases	50	50

TABLE 2.—*Relation of Various Diseases, According to Various Authors*

Author	Exertion, per Cent.	Articular Rheumatism, per Cent.	Syphilis, per Cent.	Infective Disease per Cent.	Atherosclerosis, per Cent.	Renal Disease, per Cent.	Exophthalmic Goiter, per Cent.	Adenoma Thyroid, per Cent.
Lewis (after Cohn) 152 cases	....	66.3	0.7	0.7	17.7	8.5	....	....
Frey (after Cohn) 100 cases	....	31.0	....	4.0	32.0	9.0	5.0	....
Semerau (after Cohn)	6.3	26.1	6.3	3.6	36.1	4.5	0.9	....
Willius* 500 cases	....	30.6	....	....	69.4	....	27.9	20.5

* He estimates (Clinical Electrocardiography, p. 66) that 9 per cent. of exophthalmic goiter and 70 per cent. of toxic thyroid adenomas show auricular fibrillation.

It is found more commonly in men than in women (Table 1). If one divides the cases into rheumatic and nonrheumatic groups, the sexes are about equally divided, because mitral stenosis is frequent in rheumatic female subjects, and atheroma in men.

4. Flint, H. L.: The Heart: Old and New Views, New York, Paul B. Hoeber, 1921, p. 148. Mackenzie, James: Diseases of the Heart, p. 217.

5. Neuhof, S.: Clinical Cardiology, New York, the Macmillan Company, 1917, p. 104.

Table 3.—Summary of Cases

Group	Case	Sex *	Age	Duration	Period of Observation	Onset: Gradual	Onset: Insidious	Onset: Effort	Onset: Postinfluenzal	Decompensation: Severe	Decompensation: Mild	Decompensation: Recurrent	Decompensation: At Present	Improved with Treatment	Downward Course	Rheumatic History	Valve Lesions: Mitral Insufficiency	Valve Lesions: Mitral Stenosis	Valve Lesions: Aortic	Valve Lesions: Atherosclerosis	Symptoms and Signs During Decompensation: Dyspnea	Gastro-Intestinal	Palpitation	Pain	Cough	Dizziness	Edema	Heart Borders	Enlarged Liver	Pulmonary Edema	Present Condition: Good	Fair	Poor	Dead	
	1	♂	50	?	3 yr.	+	+	—	—	—	±	—	0	+	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	+	—	—	—	
Nonrheumatic Group	2	♀	62	1 yr.	1 yr.	..	..	+	..	±	—	—	0	++	—	—	—	—	—	±	+	—	+	—	—	±	—	+	—	—	+	—	—	—	
Nonrheumatic Group	3	♂	34	2½ yr.	2½ yr.	—	—	++	? —	3 m. +	—	—	0	++	—	—	—	—	—	—	+	+	+	—	+	—	+	..	..	—	—	+	—	—	
Nonrheumatic Group	4	♂	40	1 yr.	1 yr.	—	—	+	—	±	—	—	++	+	—	—	—	—	—	—	—	—	—	—	—	—	—	½″	—	—	..	+	..	..	
Nonrheumatic Group	5	♀	70	1½ yr.	1 da.	+	+	—	—	++	—	+	D	±	+	—	—	—	—	+	+	+	+	—	—	..	+	Mid. ax.	4 fb.	—	—	—	—	+	
Nonrheumatic Group	6	♂	74	4 mo.	3 wk.	+	—	—	—	++	—	—	D	±	+	—	—	—	—	+	+	+	+	—	—	—	+	Mid. ax.	4 fb.	—	—	—	—	+	
Nonrheumatic Group	7	♂	63	1 yr.	Short	+	—	—	+	—	+	—	+	+	—	—	—	—	—	+	+	—	—	—	—	—	—	⅛″	—	—	+	—	—	—	
Nonrheumatic Group	8	♂	56	3 mo.	Short	+	—	—	++	—	+	—	—	+	+	—	—	—	—	?	+	—	—	—	—	—	—	½″	?	—	—	+	—	—	
Nonrheumatic Group	9	♀	70	3 mo.	Short	+	—	—	+	+	—	+	..	+	+	—	—	—	—	—	+	Gas+	+	—	—	—	++	1″	2 fb.	—	—	+	—	—	
Nonrheumatic Group	10	♂	56	6 yr.	6 yr.	—	—	+	+	—	—	—	—	+	—	—	—	—	—	+	+	—	—	—	+	—	..	+	..	—	+	—	—	—	
Nonrheumatic Group	11	♂	60	2 mo.	3 yr.	+	—	—	—	—	+	—	—	+	—	—	—	—	—	+	—	—	—	—	—	..	..	1″	+	—	+	—	—	—	
Nonrheumatic Group	12	♂	66	4 yr.	Short	+	—	—	+	+	—	—	±	±	+	—	—	—	+	++	++	+	—	—	+	—	—	Mid. ax.	—	—	—	+	—	—	
Nonrheumatic Group	13	♂	72	1 yr.	Short	+	—	—	—	—	+	—	+	+	+	—	—	—	—	+	+	—	+	—	+	—	—	Ant. ax.	—	—	—	+	—	—	Prostatic operation
Nonrheumatic Group	14	♂	47	+ 3 mo.	Short	+	—	—	—	—	+	—	0	+	—	—	—	—	—	+	++	..	..	..	+	..	..	2″	—	+	+	—	—	—	

Rheumatic Group	15	♂	44	Short	2 yr.	+	—	—	—	—	+	..	—	+	—	—	?	?	—	±	At-tack	+	+	—	—	—	—	1″	—	—	+	—	—	—	Paroxysm'l auricular fibrillation
	16	♂	72	Yrs.	3 yr.	+	—	—	—	+	—	+	0	+	—	25 y. ++	+	+	+	++	+	+	+	—	—	—	—	Ant. ax.	4 fb	—	+	—	—	—	
	17	♀	69	15 yr.	8 yr.	+	—	—	—	+	—	+	±	+	—	+	+	—	—	±	+	+	+	—	—	—	—	2″	1 fb.	—	+	—	—	—	
	18	♀	65	8 yr.	Short	+	—	—	—	+	—	+	D	—	—	+	+	+	+	+	+	+	+	—	—	—	+	Ant. ax.	3 fb.	—	—	—	—	+	
	19	♀	62	Yrs.	8 yr.	+	—	+	—	+	—	+	—	+	+	+	+	—	—	+	+	+	+	—	+	+	+	Ant. ax.	3 fb.	—	—	—	—	?	
	20	♀	46	3 yr	—	+	—	—	++	+	—	+	D	+	+	7 y.	+	—	+	+	+	+	+	—	—	—	+	Ant. ax.	+ 3 fb.	—	—	—	—	+	
	21	♀	38	8 yr.	10 da.	+	—	—	—	+	—	—	D	+	+	+	+	—	—	—	++	+	+	..	..	..	..	Ant. ax.	+	—	—	—	—	+	
	22	♀	26	3 mo	Short	+	—	—	—	++	—	+	D	—	+	+	++	—	—	—	++	+	+	+	—	+	—	Mid. ax.	4 fb.	—	—	—	—	+	
	23	♂	73	6 mo.	1 yr.	+	—	—	—	+	—	+	D	+	+	+	+	—	+	++	++	+	+	—	—	+	+	Ant. ax	3 fb.	—	—	—	—	+	
	24	♀	49	4 yr.	3 mo.	+	+	—	—	+	—	+	D	+	+	+	+	..	+	—	+	+	—	—	—	—	—	Ant. ax.	3 fb.	—	—	—	—	+	Cancer uterus
	25	♂	21	3 mo.	1 mo.	+	—	—	—	++	—	+	—	+	—	+	+	+	—	—	+	—	—	—	—	—	..	1″	—	—	—	+	—	—	
	26	♀	60	Less than 1 yr.	1 yr.	+	—	—	—	++	—	+	—	++	..	++	+	—	—	+	+	+	—	+	—	—	—	Ant. ax.	2 fb.	—	+	—	—	—	
	27	♀	57	+ 3 yr.	3 yr.	+	—	—	—	+	—	+	—	++	—	+	+	+	—	—	+	+	+	—	—	—	—	½″	1 fb.	—	+	—	—	—	
	28	♀	66	+ 2 yr.	5 mo.	+	—	—	—	+	—	—	D	±	+	+	+	+	..	+	+	+	..	+	+	..	+	Ant. ax.	3 fb.	—	—	—	—	+	
	29	♂	49	7 yr.	6 yr.	—	—	+	—	+	—	+	—	++	..	+	+	+	—	+	+	+	—	—	—	—	—	Ant. ax.	3 fb.	—	±	—	—	—	
	30	♀	40	Yrs.	1 yr.	+	—	—	—	—	+	—	±	+	—	+	+	+	—	—	±	+	—	—	—	—	+	1″	1 fb.	—	+	—	—	—	
	31	♂	45	2 yr.?	8 mo.	+	—	—	—	—	+	—	+	+	—	+	+	+	—	+	+	—	+	++	—	—	—	2″	—	—	—	+	—	—	
	32	♂	50	Yrs.?	9 mo.	+	—	+	—	—	+	+	±	++	—	+	—	+	—	+	++	—	+	+	—	—	—	1″	1 fb.	—	+	—	—	—	
	33	♂	50	?	3 mo.	+	—	—	—	—	+	—	+	±	+	Tons	+	—	+	±	+	+	+	+	—	—	..	3″	—	—	+	—	—	—	
	34	♀	36	Short	—	—	—	—	—	±	—	+	—	+	—	+	+	—	—	—	+	+	+	..	+	..	+	Mid. ax.	4 fb.	—	+	—	—	—	Pregnancy, hypertension, albuminuria
	35	♂	69	Short	8 mo.	—	—	+	—	+	—	+	±	++	—	+	+	—	+	+	+	+	++	+	—	+	++	2″	2 fb.	++	+	—	—	—	Paroxysmal auricular fibrillation
	36	♀	45	Yrs.	Short	—	—	—	—	+	—	+	±	++	—	+	—	+	—	—	+	—	+	+	..	..	+	1½″	1	+	+	—	—	—	
	37	♂	35	3 yr.	Short	+	—	—	—	+	+	+	±	+	—	—	+	—	—	—	+	+	+	—	—	—	±	±	—	—	+	—	—	—	

* In this column, ♂ indicates male, and ♀ female.

ETIOLOGIC AND PREDISPOSING FACTORS

Besides rheumatism with valvular endocarditis and arteriosclerosis, both of which are apt to be accompanied by myocardial weakness, various other factors are of prognostic significance. Among these are prolonged effort, infective diseases, thyroid disease and renal disease [6] (Table 2). Effort, sometimes slight and sometimes violent, may produce it. This occurs most often in middle life or in rheumatic hearts.[7]

Digitalis may produce it,[8] while, on the other hand, Eyster and Fahr [9] refer to rare cases in which auricular fibrillation is not accompanied by demonstrable valvular or myocardial change, and in which no serious break in compensation has occurred.

Contrary to the view of muscle damage as the cause of fibrillation, it is thought by some to be due to abnormal stimulus production in the sinus node, or multiple foci in the auricular wall, or that the disorganization of auricular activity is a defect in conduction, the sinus node maintaining its activity or some other auricular focus taking it over.[10]

---

6. S. A. Levine (Am. J. M. Sc. **154**: 146, 1917) found, in eighteen cases of transient auricular fibrillation, one during lysis of pneumonia similar to one described by Krumbhaar (Transient Auricular Fibrillation, an Electrocardiographic Study, Arch. Int. Med. **18**: 263 [Aug.] 1916); one in hyperthyroidism; one after operation; two during acute rheumatic fever; four following auricular fibrillation; four as a result of active digitalis therapy, and six spontaneous or idiopathic. He also refers to Cohn as finding transient auricular fibrillation in twelve of 123 cases of pneumonia, and two cases in healthy, athletic young men.

7. This is well seen in Cases 10, 3, 4, 29 and 34 of this series.

8. White and Burwell (The Clinical Significance of Changes in the Form of the Electrocardiogram, M. Clin. N. Am. **4**: 1843 [May] 1921) cite a case of auricular fibrillation (paroxysmal) assumed to be due to digitalis. The patient was admitted with normal rhythm but with cardiosclerosis and moderate decompensation, and precordial pain on exertion from cardiac symptoms, and he then had auricular fibrillation. Digitalis was stopped, and a week later he had normal rhythm. He was under observation for six years. Three years after the time he was first seen, fibrillation was present and permanent. The steady, slow, downward course in an old man with marked arteriosclerosis and final permanent fibrillation was not accompanied by any period of acute heart failure, the patient being able to work constantly on his farm. Progressive changes in the electrocardiogram were characteristic of advancing myocardial involvement.

9. Eyster, J. A. E., and Fahr, C. E.: Observations on the Use of Quinidin in Auricular Fibrillation, Arch. Int. Med. **29**: 59 (Jan. 16) 1922. "While acutely developing auricular fibrillation undoubtedly causes considerable mechanical deficiency of the heart and is probably not infrequently the cause of cardiac decompensation, the heart may compensate for this as it does for valve injury, particularly when it is assisted by the protective influence of ventricular stimulation by digitalization."

10. Cohn thus summarizes a discussion of the subject: "On account of the view generally held of the function of heart muscle and because of the transient nature of the irregularity in many instances, there seems to be little reason for laying undue stress on structural alteration of the muscle to account for this abnormal rhythm" (Nelson's Loose Leaf Medicine **4**: 335).

PROGNOSTIC FACTORS

In spite of its frequency, its etiologic relation to antecedent rheumatic involvement with endocarditis or to cardiosclerosis, and the ease of recognition of auricular fibrillation,[11] it is too frequently overlooked, or when it is suspected or recognized, a serious prognosis is too often given. While there are numerous contributions to the subject in the literature, they have dealt largely with diagnosis, discussion of its mechanism, or response to quinidin, and not with prognosis. Pardee,[12] however, pointed out that an irregular heart does not of necessity cripple the individual, and that breaking down of cardiac reserve takes a variable amount of time, the variation depending on the quality of heart muscle, on the demands the patient makes on his heart, or on the associated pathologic conditions.

Whether auricular fibrillation depends on diseased myocardium or to changes in stimulus production or conduction, prognosis depends primarily on the degree and duration of impaired cardiac efficiency and the heart's ability to function under the new rate and rhythm. If decompensation is severe, moderate or mild, the future of the patient will depend also on his age, etiologic and predisposing factors, associated pathologic conditions and response to treatment. The outlook in a patient of 30, with antecedent attacks of acute arthritis and endocardial damage, is obviously quite different from that in a patient of 60, with thickened peripheral arteries, an enlarged heart and a story of gradually failing circulation, incidental to long continued arterial tension; in the former, the young myocardium has remarkable powers of recuperation, while

11. Auricular fibrillation is detected without instruments by: 1. The fact that it is the usual type of arrhythmia, especially in the conditions cited. 2. The complete arrhythmia, no two successive beats being the same in rhythm or force. This is contrary to the next frequent arrhythmia, due to premature contractions, in that in the latter there are runs of normal rhythm followed by what appears to be a dropped beat at the radial pulse, but which in reality is a beat appearing earlier than normal and followed by a compensatory pause. The premature beat is usually too weak to come through to the wrist, but can be heard over the heart with the stethoscope. 3. The deficit between the radial count and that over the heart as heard with the stethoscope. This is due to many weak ventricular contractions which do not come through to the radial artery. This may also be present in frequent ectopic beats when some are too weak to appear at the radial artery. 4. The history of impaired cardiac efficiency, as shown by the presence of antecedent endocarditis and valvular lesions, especially of the mitral valve. 5. History of long continued heart strain from arterial hypertension and accompanying cardiosclerosis.

12. Pardee, H. E. B.: The Prognosis of Auricular Fibrillation, J. A. M. A. **64**: 2057 (June 19) 1915.

in the latter, evidences of myocardial weakness may be obvious. On the other hand, irrespective of age or predisposing factors, auricular fibrillation may be attended by profound circulatory failure, the heart responding promptly to treatment, even a return to normal rhythm; or, in spite of treatment, there is a fatal termination.

Consideration of factors leading to serious decom-

TABLE 4.—*Fatal Cases*

Case	Duration of Auricular Fibrillation	Recurrent Decompensation	Chronic Endocarditis	Atheroma	Duration of Decompensation Before Death	Age, Years
5	1½ yrs.	+	—	+	6 mos.	70
6	4 mos.	—	—	+	4 mos.	74
18	8 yrs.	+	+	+	2 mos.	65
20	3 yrs.	+	+	+	6 mos.	46
21	3 yrs.	—	+	—	3½ yrs.	38
22	3 mos.	+	+	—	6 mos.	36
23	6 mos.	+	+	+	6 mos.	73
24	4 yrs.	—	+	—	5 wks.	49
28	2 yrs.	—	+	+	1½ yrs.	66

TABLE 5.—*Findings of Willius*

	Cases	Aur. Fib. Mortality, per Cent.	Control Mortality, per Cent.
Complete series	500	41.3	13.6
Uncomplicated	367	36.9	16.2
With premature ventricular beats	89	42.0	15.9
Aberrant Q R S in all derivations	33	63.4	54.2
Aberrant Q R S and premature ventricular beats	11	87.5	87.5
In mitral regurgitation	72	51.9	27.8
In mitral stenosis	27	47.1	22.2
In mitral regurgitation and stenosis	40	53.4	34.6
In aortic regurgitation	13	33.7	71.4
In chronic myocarditis	81	43.3	43.3
In myocarditis secondary to hypertension	98	54.7	53.8
In myocarditis secondary to exophthalmic goiter	97	34.2	17.1
In myocarditis secondary to thyroid adenoma and hyperthyroidism	71	30.0	8.9

pensation and death is of prognostic value. A downward course, in spite of treatment, is evidence of failing heart muscle whose reserve is exhausted. It will be noted in the cases here cited that duration of fibrillation is not necessarily a factor. Recurrent decompensation was found in more than half the cases. Rheumatic history and chronic valvular lesions, or atheroma are definite factors. The duration of decompensation before death illustrates the progress of cardiac failure (Tables 3 and 4).

Electrocardiographic records in auricular fibrillation have been emphasized by White,[13] who found that aberrant ventricular complexes or ectopic ventricular contractions have a prognosis twice as grave as uncomplicated auricular fibrillation because these indicate

TABLE 6.—*Relation of Clinical Groups to Present Condition and to Sex*

	Condition Good	Condition Fair	Died	Males	Females
No endocarditis or rheumatic history	6	6	2	11	3
Endocarditis and rheumatic history	13	2	7	10	13

TABLE 7.—*Relation of Onset to Etiologic Factors*

Effort	Gradual or Insidious	Postinfluenzal	With Atheroma and Hypertension	With Endocarditis	Endocarditis and Atheroma
8	29	6	9	23	13

TABLE 8.—*Duration*

	Number
15 years	1
8 years	1
7 years	1
6 years	1
4 years	2
3 years	2
2 years	2
"Years"	7
Less than 1 year	13
Paroxysmal	2

TABLE 9.—*Degree and Recurrence of Decompensation*

Former Attacks	Number of Cases	Present Condition		
		Good	Fair	Dead
Severe	24	9	5	10
Moderate	11	8	3	..
Recurrent	19	8	2	7

serious myocardial damage or irritability of the ventricle, which does not stand up under the strain as relatively healthy muscle does. Willius, in 500 cases, found 133 patients with premature beats,

13. P. D. White (Prognosis in Heart Disease in Relation to Auricular Fibrillation and Alternation of the Pulse, Am. J. M. Sc. **157**:5 [Jan.] 1919) showed that in pulsus alternans the prognosis is much more grave than in auricular fibrillation, but that fibrillation as such adds little, if anything, to the gravity of prognosis in a case of heart disease. Forty-eight per cent. of 100 patients with fibrillation and 47 per cent. of 100 patients with similar hearts, but without fibrillation, died.

aberrant Q-R-S in all derivations, or aberrant Q-R-S with premature ventricular beats (Table 5). In the latter, the mortality was 87.5 per cent. He concludes that mortality attending fibrillation doubles and in some groups trebles that occurring in similar types of heart disease not complicated by this arrhythmia, but that it is not the arrhythmia per se which is the determining factor, but the integrity of the myocardium and proper treatment.

Clinical Groups.—As pointed out by others, auricular fibrillation cases fall into certain clinical groups, the most definite being those with and without rheumatic endocarditis. In the latter, syphilis is a relatively unimportant factor.[14] There are two distinct clinical groups as to etiology. The relation of these groups to present condition and to sex is shown in Table 6.

The onset of the irregular rhythm was insidious or gradual in twenty-nine cases, dated by the patient to effort in eight, to influenza in six, and seemed to be a result of atheroma associated with hypertension in twenty-three. It was associated with chronic valvular lesions in twenty-three, and both endocarditis and atheroma were found in fourteen.

The duration of auricular fibrillation is often difficult to determine accurately, but in many cases the patient is aware of the irregularity (Table 8). The degree and recurrence of decompensation from the history and from observation give some idea of the severity of myocardial weakness (Table 9).

Response to treatment, with consideration of the frequency, severity and duration of attacks of decompensation, gives a measure of prognosis. Many patients with a heart rate over 120 make quick recovery on digitalis and may be able to undertake rather heavy physical work so long as the rate is kept down by digitalis.

Brachmann [15] emphasizes cardiac and mental rest.

14. Levine, S. A.: Auricular Fibrillation: Some Clinical Considerations, Am. J. M. Sc. **154**: 43 (July) 1917. In 128 cases, Levine found only eleven that showed syphilis, while 12 per cent. of all cases admitted to the medical wards showed syphilis.

15. Brachmann, D. S.: Auricular Fibrillation, from Observations and Deductions from a Series of Four Hundred Cases, Lancet **1**: 374 (Feb. 19) 1921. The effect of digitalis is explained by exhaustion "brought about by the ventricular rate being stimulated to too great activity, and the slowing enables the ventricle to get more rest and so regain a measure of strength. From this result the condition of the muscle is determined, and one can estimate, within certain limits, the amount of healthy muscle by the degree of recovery."

Hamburger [16] states that it is not so much the heart rate observed at first as the heart rate observed after treatment that is important. Cohn [17] emphasizes the prognostic value of the pulse deficit, the disparity between the heart rate and the pulse rate decreasing as the patient improves and as the heart slows. Christian,[18] comparing digitalis effect in chronic cardiac cases with and without fibrillation, found that when he got good results from digitalis the heart slowed, irrespective of rhythm.[19]

Valvular lesions were demonstrated clinically in all of Group 2, usually evidenced by a mitral systolic murmur. The cases have been so grouped with a full realization that such a murmur may appear in cardiosclerosis or in a dilated and weakened heart. The history of acute rheumatism, sometimes recurrent, the recollection on the part of the patient that there were accompanying valvular lesions, and evidence of such a lesion at the time of observation have all been taken into account. The mitral stenotic murmur was found in ten cases. This has given rise to much discussion, since its presence has been taken as evidence of auricular contraction.[20]

---

16. Hamburger, W. W.: Treatment of Auricular Fibrillation, M. Clin. N. Am. **5**: 1716 (May) 1922.

17. Cohn, A. H., in Nelson's Loose Leaf Medicine **4**: 333.

18. Christian, H. A.: Digitalis Effect in Chronic Cardiac Cases with Regular Rhythm in Contrast to Auricular Fibrillation, M. Clin. N. Am. **5**: 1173 (March) 1922.

19. Quinidin was not used in this series because the patients were not under hospital and electrocardiographic control.

20. According to Flint (The Heart: Old and New Views, p. 148), two factors are involved in the variability of murmurs in this condition: The auricle does not contract as a chamber but remains in the position of diastole; the murmurs which occur during the diastolic period in mitral stenosis are dependent on the rate of flow of blood from auricle and ventricle. With auricular contraction, the intra-auricular pressure is greatest during the active contraction of this chamber and again at the opening of the mitral avalve. The two varieties of diastolic murmurs in mitral stenosis are due to these two causes. The position of the murmurs caused by the contraction of the auricle depends on the time relation of this contraction to the diastole of the ventricle. This murmur, which is crescendo in character and called auriculosystolic, disappears in fibrillation, whereas the other diastolic murmur remains unaffected by fibrillation and bears a fixed relation to the second sound of the heart. The amount of diastolic period which the latter murmur occupies will depend on the rate of the heart and the degrees of stenosis. Cohn (p. 335) discusses it as follows: "This disappears necessarily at onset of fibrillation. It is made when the mechanism of the heart is normal by the contraction of the left auricle; when the auricles fibrillate, the possibility of creating the murmur by their contraction no longer exists. The auricular-ventricular murmur accordingly ceases, but in the normally beating heart blood flows from the left auricle to the left ventricle during the whole of the ventricular diastolic period, not merely at the terminal portion of it during the contraction of the auricle. The flow of blood into the ventricle which takes place in the first portion of ventricular diastole likewise produces a murmur, often of rumbling character, in passing the stenotic valve. This murmur is present irrespective of the nature of auricular activity, whether coordinate or fibrillating."

The measure of cardiac efficiency has the same significance in auricular fibrillation as in other cardiac conditions. No one sign determines prognosis, but the composite of many symptoms and signs which indicate weakened heart muscle. In 100 patients without fibrillation, the early symptoms and signs were: dyspnea, 92; palpitation and consciousness of arrhythmia, 66; pain, 50; dizziness, 40; cough, 31; gastro-intestinal, 19; pulmonary edema, 10; heart enlargement, 74; albuminuria, 38; edema, 17; liver enlargement, 9, and disturbance in rhythm, 36.

It will be noted in Table 3 how frequently the heart and liver borders were increased in mild decompensation. This seems to indicate that the heart adjusts itself to the combined factors which increase the demands on it by hypertrophy, and that the liver, at an earlier stage often much enlarged from passive congestion, remains so, though function is not much embarrassed.

GROUP 1.—CASES WITHOUT RHEUMATIC HISTORY OR ENDOCARDITIS; ONSET GRADUAL, INSIDIOUS, FOLLOWING INFLUENZA OR EFFORT

CASE 1.—Mr. R. H. B., aged 50, seen, May 21, 1920, because that morning during examination for life insurance, the heart was found irregular, had no symptoms and did not know how long the heart had been irregular; he had never been aware of it. The patient appeared healthy, with no cyanosis, edema, dyspnea or other evidences of circulatory disturbance. The systolic blood pressure was 135; diastolic, 85. The heart was very irregular in force and rhythm. There was a pulse deficit of 30; a pulse rate of 120. The heart borders were increased; roentgenograms made at a distance of 2 meters showed an increased shadow of both auricles, particularly the left. The heart area, as compared with the normal, was:

	Age	Height Inches	Weight Pounds	Area Sq. Cm.	Diameter Cm.
Observed	50	53	186	148	15
Normal	50	73	187	142	14
Normal	50	74	199	148	14.4

Polygrams at this time showed auricular fibrillation.

Standardized tincture of digitalis, 15 minims, was ordered every four hours, and when the patient was seen two days later the pulse at the radial was 90, with occasional large beats. The heart rate was 140. Digitalis was ordered in 20 drop doses for three days. The rate over the heart was 152,

pulse 102. The patient was feeling very well. He had been away on a business trip by automobile, and walked considerably, with little difficulty. Digitalis was continued for two days longer, when the heart rate was 130, and the pulse 86. There were no evidences of overdigitalization. Digitalis was continued in 30 drop doses for the next three days, when the heart rate was 140 and the pulse 90. June 5, after continuous digitalis, there was no particular effect on the heart. He was seen from that time on until March, 1921, when he said that he was feeling a little bit tired, with some shortness of breath on exertion, but he was under a business strain. The radial count was 76; the heart, 160. He was seen again in April, when he said that for a few weeks he had not been feeling just right; he was depressed and awakened early the last few mornings, which he attributed to business worries. He thought that he had been slightly jaundiced; examination showed him to be definitely so; the liver was palpable two finger breadths below the costal margin and tender; the tongue was moderately coated; the heart was very irregular; the rate over the heart was 120, and the wrist, 95. He was not seen again until August, when he was feeling quite well, and was able to do moderate physical exertion without any difficulty. The pulse at the wrist was 67, and at the heart, 134. An electrocardiogram showed typical auricular fibrillation. He was again given digitalis, 15 drops, for fifteen doses, and then 5 drops for thirteen doses. In October he was feeling well and was walking to his office three times in the last week, which was about 3 miles, walking the distance in fifty minutes and having no distress of any kind.

In this patient, irregularity of the heart came on without his being aware of any cardiac distress or signs of cardiac strain. It has continued now for three years and the heart is functionally normal. Digitalis did not restore normal rhythm.

Case 2.—Mrs. R. G. R., aged 62, seen in 1919, when she complained of "rheumatism" and some neuritis, had a thyroidectomy in 1909, when menstruation stopped; otherwise she had always been quite well. She had no cardiac symptoms, and heart examination was completely normal at that time, with a blood pressure of 130 systolic and 80 diastolic. She was not seen again until July 7, 1922, when she complained of considerable dyspnea and, the night before, considerable orthopnea. She dated her trouble to May, two months before, when she was very busy with house cleaning and noted that her heart was irregular. Examination showed a completely irregular heart, with a pulse rate of 133 and ventricle contractions of 145. The blood pressure was 130 systolic and 95 diastolic; the heart was slightly dilated. Electrocardiograms showed auricular fibrillation. There was some notching of the R. She improved rapidly on digitalis, and is now quite well.

CASE 3.—Mr. F. A. H., aged 34, seen, Sept. 29, 1921, had been troubled with his heart since January, 1921, coming on after extreme exertion, the symptoms then being dyspnea and weakness. He was in the hospital for three months. He had more or less broken compensation, but, on the whole, he was quite comfortable. The heart was completely irregular, and electrocardiograms showed typical auricular fibrillation. This patient has gone on since in comfort, occasionally requiring digitalis to tide him over periods of broken compensation.

CASE 4.—Mr. W. A. W., aged 40, seen, Aug. 15, 1922, a stationary engineer, had not been able to work since February. He was extremely nervous. His heart was very irregular. Slight effort, as walking fast, even a short distance, produced extreme palpitation, after which the precordium was very sore. The onset occurred in February, after lifting heavy objects while working on the railroad. He thought he had had an irregular heart for some years. He had been hoarse for some months. He had diphtheria years ago, no severe rheumatism, and no other illness. In 1902, when he had an operation, there was some trouble with the heart, but never an acute breakdown, dyspnea or cyanosis. He was well developed; he did not look sick. The pulse was: wrist, 110; heart, 120; the systolic blood pressure, 120; diastolic, 80. The cervical veins were slightly distended, the left heart border one-half inch outside the normal border. The valve sounds were clear. Electrocardiograms showed typical auricular fibrillation. The heart has improved but is still irregular.

CASE 5.—Mrs. T. H. M., aged 70, seen, June 20, 1919, for cystitis, had an irregular heart, which irregularity came on insidiously. Polygrams showed auricular fibrillation. She was not seen again until Sept. 26, 1920; she had not been well since March, being in bed two thirds of the time with broken compensation, digestive disturbances and enlarged liver, which was tender, four finger breadths below the costal margin; there was edema of the ankles, and some fluid in the abdomen. The heart was dilated to the midaxillary line. The sounds were clear. The pulse was 130; the heart, 160. The systolic blood pressure was 190; diastolic, 105. Polygrams showed auricular fibrillation. The patient died suddenly the day following.

CASE 6.—Mr. C. W. D., aged 74, seen, Oct. 20, 1920, complained of heart trouble for four months, weakness, dyspnea, nausea and edema. He was cyanotic, the liver was tender, palpated four finger breadths below the costal margin, and the heart was dilated to the midaxillary line, and very irregular. The heart rate was 150; radial pulse, 100. The systolic blood pressure was 195; diastolic, 100. The heart sounds

were clear. Polygrams showed auricular fibrillation. The patient died ten days later.

CASE 7.—Mr. O. A. D., aged 60, seen, Dec. 20, 1919, had been troubled with the heart since having influenza in November, 1918. There had been no symptoms for four or five months after the influenza, but gradual increasing weakness and dyspnea on exertion. The condition had been getting worse for the past two months. He had had no former illness. The systolic blood pressure was 195; diastolic, 100. The left heart border was 1 inch to the left of the midclavicular line in the sixth space. The sounds were clear; the heart rate, 110; the pulse, 92. The condition was recognized as auricular fibrillation from the polygrams; digitalis was given, with steady improvement. December 24, the heart rate was 56; the pulse, 44. In February, on returning from California, the patient was comfortable. The heart was still irregular.

CASE 8.—Mr. W. A. A., aged 56, seen, May 10, 1922, complained of dyspnea on exertion, and soreness of the calves of the legs; he was subject to frequent colds; his heart, which he knew was irregular, was worse with cough. He dated his trouble from influenza in February, 1922. He was obese (weight, 285 pounds [129 kg.]; height, 5 feet 11 inches [180 cm.]). He had always been a heavy eater, smoked to excess, and always worked hard in a paper mill. The left border of the heart was one-half inch outside the normal limits; the sounds were distant but clear, and the rate was completely irregular; heart, 92; wrist, 72. The blood pressure was: systolic, from 145 to 155; diastolic, from 75 to 85. Electrocardiograms showed auricular fibrillation. When seen two months later, he was better, but still had dyspnea on unusual effort and his heart still showed fibrillation.

CASE 9.—Mrs. E. M. T., aged 70, seen, May 31, 1922, complained of "asthma" for two years, but since an attack of influenza three months before, when she was in bed for three weeks, she had swelling of the ankles and feet, gas in the abdomen, palpitation, throbbing of the vessels in the neck, and orthopnea. She was very obese, and had considerable respiratory effort; the external jugulars showed marked filling and collapse, the latter especially with respiration. There was much edema and marked cyanosis. The left border of the heart was 1 inch outside the normal limits, and was completely irregular; there were no murmurs. The systolic blood pressure was 140; diastolic, 85. The heart rate was 120; radial pulse, 100. The liver was tender, palpated two finger breadths below the costal margin. An electrocardiogram showed typical fibrillation with ectopic beats. The patient has not been seen since, but reports that she has not improved much. One cannot say how long the fibrillation has been present.

CASE 10.—Mr. H. L. S., aged 56, seen, Aug. 31, 1921, knew that his heart had been irregular for the past four years. The onset occurred with sudden fright and exertion; his house caught fire, he had considerable trouble in awakening his wife, and then he ran to a fire alarm box. His physician gave him digitalis because he was quite dyspneic and there were apparently evidences of heart failure. Nine months ago he had influenza with bronchitis and possible pneumonia; the heart was then enlarged and it was irregular. It improved with digitalis. He appeared healthy; he was of large frame and well nourished; there was a tendency to obesity. There was no cyanosis and no dyspnea. The heart was quite irregular, the rate at the wrist 87 and over the heart 108. There were a few fine râles heard at the bases of both lungs posteriorly. The heart borders were difficult to outline, but apparently increased. The sounds were distant but clear. The arteries were moderately thickened, and the blood pressure was: systolic, 120; diastolic, 85. Electrocardiograms showed typical auricular fibrillation. In the next two months he improved steadily, requiring occasional digitalis, and troubled occasionally with cough and sore throat, sleeplessness and nervousness. October 27, he was feeling well and has continued so since; he is able to be up and about his business, walking without difficulty, and exerting himself in an ordinary way without signs of heart strain.

In this man of 56, the onset seems to date definitely to the time of fright and severe exertion, when he had an acute heart breakdown of moderate severity which responded to digitalis. The heart was probably irregular at that time and has been so since. At present, nearly five years after the onset, the man is efficient and able to attend to his business with no marked evidences of heart strain.

CASE 11.—Mr. J. H. H., aged 60, seen, Aug. 30, 1921, two months before began to tire easily, and lost 20 pounds (9 kg.) in two months. There was some dyspnea and palpitation. Examination showed severe secondary anemia. The heart borders were increased, the left border being 1 inch outside the midclavicular line. The liver was slightly enlarged, and there were a few râles at the bases of both lungs, posteriorly. The picture was that of moderate cardiac failure. Electrocardiograms showed definite auricular fibrillation with ectopic beats. He was given tincture of digitalis, 25 drops, every four hours for eight doses. When seen, September 2, he was not feeling very much better, but the heart was perfectly regular and electrocardiograms showed normal P-R-T sequence. September 6, he was better and the heart was regular, but, on the 7th, auricular fibrillation was again present. Digitalis was continued for the next two months, the patient feeling very much better, although his heart remained irregular. The electrocardiograms still showed auricular fibrillation. He has been at work since.

CASE 12.—Mr. C. J. A., aged 66, seen, Aug. 29, 1922, complained of marked dyspnea, digestive disturbances, cough and moderate sputum in the morning, and rather frequent hiccup. He dated his trouble from moderately severe influenza in 1918 and again in 1921. Since then there had been increasing difficulty with breathing, and other evidences of gradual circulatory breakdown. The patient was ambulatory; he showed very little cyanosis, but some respiratory effort. The systolic blood pressure was 225; diastolic, 150; the emphysematous contour of the chest was marked; the lungs were clear except for some râles at the bases posteriorly. The heart was completely irregular, the rate over the heart being 160 and at the wrist 140. The impulse was forcible. There was a systolic and a faint diastolic murmur over the aortic area, and a soft blowing systolic murmur at the apex. The left border of the heart was out to the midaxillary line.

In this man of 66, a farmer who had done heavy physical work, the onset of the circulatory failure seems to date to mild attacks of influenza, the first one being four years ago. In spite of the marked dilatation of his heart and the complete arrhythmia, he did not have orthopnea, and his cardiac failure was only moderate. The arteries were extremely tortuous and thickened. In spite of these factors, this man has probably gone on for two or three years with a completely irregular heart. His physician at home told him that his heart was irregular during the influenza. The patient was under observation for four weeks. There was marked improvement in the heart; he was symptomatically relieved, but, since returning home, to a higher altitude, he has not been so well, and the outlook is not favorable.

CASE 13.—Mr. W., aged 72, seen, Sept. 18, 1922, had moderate circulatory failure. He had been in bed for some weeks following a prostatic operation. He now had an infected bladder and pyelonephrosis. He had been a very active man, in spite of his age, spending long hours at his business. The heart was dilated within 1 inch of the anterior axillary line, and was completely irregular. While there was no orthopnea, there was some dyspnea and cough. The heart was completely irregular, and showed auricular fibrillation, with a pulse deficit of 40, the heart rate being 160. He improved under digitalis, although the heart never became regular and the bladder and prostatic trouble still persisted. On account of the man's age and infection the prognosis is, of course, unfavorable, irrespective of the cardiac condition.

CASE 14.—Mr. F. G., aged 47, a laborer, seen, Oct. 18, 1922, had been in the hospital in July on account of an accident. He had what he called asthma in April. Since July he had had cough, a little sputum, and shortness of breath on the slightest effort. He did not know that he had any irregularity of the pulse. He was very well nourished, with no cyanosis and only slight respiratory effort. The left border of the

heart was felt in the sixth space about 2 inches from the mid-clavicular line. The heart sounds were distant, and no murmurs were heard. The heart rate was 140, and the pulse rate, 95. The liver was not enlarged. Electrocardiograms showed typical auricular fibrillation. The systolic blood pressure was 180; diastolic, 100. He was given digitalis, to which he responded readily, so that now he is quite comfortable.

GROUP 2.—WITH RHEUMATIC HISTORY AND CHRONIC ENDOCARDITIS

CASE 15.—Mr. J. A. E., aged 44, seen, Dec. 15, 1920, complained of palpitation and irregularity of the heart for about three months. He had had acute rheumatic fever and chorea at 12 years of age. Years ago he had had liver trouble, with edema of the feet, and formerly he was an immoderate user of alcohol and tobacco. There were occasional ectopic beats; the heart was slightly enlarged; there was a very soft distant blowing murmur at the apex. He was not seen again until Oct. 20, 1922, when he said he had had six attacks with his heart since he had been seen before, characterized by extreme palpitation, usually of sudden onset, lasting from a few hours to two or three days, without apparent cause. The left heart border was 1 inch outside the normal limits; the pulse rate was 190. Electrocardiograms showed paroxysmal auricular fibrillation. He had been taking 10 drops of digitalis three times daily for ten months. When seen next day the heart was regular, and the electrocardiograms showed normal rhythm. Since that time he has had four or five attacks, but has never been seen in any of them.

In this man of 44 with cardiac history, dating back to the age of 12, cardiac symptoms have not been prominent until the last two or three years, characterized by paroxysmal tachycardia due to auricular fibrillation, which disappears between attacks, when cardiac efficiency is good. He is able to work in a lumber mill.

CASE 16.—Mr. F. E. H., aged 72, seen, Jan. 5, 1921, had broken compensation, moderate dyspnea and some orthopnea; and digestive distress and "palpitation" were troublesome symptoms. He was laid up in bed twenty-five years ago for thirteen weeks with acute rheumatism; his heart was probably involved then. There had not been recurrence except for an attack a year ago, with some rheumatism in the knees. He thinks the heart was irregular for many years. The heart was dilated to the anterior axillary line, and very irregular in force and rhythm; the rate was 160. There was a pulse deficit of 70. There were definite aortic systolic and mitral systolic murmurs. The systolic blood pressure was 190; diastolic, 100. The peripheral arteries were tortuous. The liver was tender, extending four finger breadths below the costal margin in the mammillary line. Digitalis produced a marked improvement in ten days, and from that time on there was

slow, though progressive, improvement. He is now up and about and comfortable, able to get about on street cars and automobiles. When last seen, Aug. 21, 1922, his heart was compensating well. There was a definite late diastolic or crescendo presystolic murmur at the apex in addition to the mitral and aortic systolic murmurs. He has learned to use digitalis in from 5 to 10 drop doses for dyspnea or beginning symptoms of cardiac decompensation.

CASE 17.—Mrs. H. M. E., aged 61, seen, Sept. 7, 1915, in broken compensation, had had rheumatism. She had been in bed since April, the trouble having begun with seasickness. Her chief complaint at that time was gas, indigestion and pain under the heart, and a fluttering sensation in the abdominal aorta. She had had "indigestion" for years, and a frontal sinus infection for three years. At the time of examination her heart was very irregular, with a pulse deficit. When questioned, she volunteered the information that her heart had been irregular, she knew, for years, probably for fifteen years. The left border was 2 cm. outside the normal borders, with a soft blowing murmur at the apex. The systolic blood pressure was 120; diastolic, 80. The liver was felt one finger breadth below the costal margin, and was tender. The lungs showed some râles at the bases posteriorly. Polygrams made at that time showed auricular fibrillation. The patient improved so much in a month that she was able to come to the office, having dyspnea only on unusual effort. She was then seen irregularly for a year, with gradual improvement but occasional breaks in compensation, which were mild. In October, 1916, she had mild lobular pneumonia and was in bed a month. Her heart responded to digitalis and gave no especial concern. She was not seen from January, 1917, to February, 1922, when she was 68 years old. In the meantime she had been very well, although using digitalis or strophanthus occasionally. She volunteered that strophanthus often helped her more than digitalis. Her chief complaint was gas in the stomach, with a feeling of pressure on the heart which made it flutter or palpitate. Her heart was compensating at that time; there was a definite systolic murmur at the apex, which had been heard seven years before, and a pulse deficit of 20. The liver was palpated about one finger breadth below the costal margin. Auricular fibrillation was demonstrated by electrocardiograms.

This woman of 68 was known to have auricular fibrillation seven years before, when she had been in bed most of the time for five months. For almost a year following there were recurring breaks in compensation, usually of moderate severity, which were always aided by digitalis or strophanthus. She was lost sight of for five years, during which time she has been very comfortable, up and about doing her housework, her chief complaint being gas and palpitation. In

spite of fibrillation, she went through a moderately severe attack of bronchopneumonia in 1916.

CASE 18.—Mrs. S. J. F., aged 65, seen, Aug. 8, 1922, complained of dyspnea and constipation. She had been troubled with her heart for six or eight years, and frequent tonsillitis and more or less "rheumatism" for years, but had never been laid up in bed. Her tonsils were about twice the normal size, and were cryptic. The heart was dilated 8 cm. to the left, and there was moderate decompensation; the rate was 120 with a pulse deficit of 10. At the apex there was a soft blowing systolic murmur and a faint, though definite, presystolic murmur. Over the aortic area there was a soft systolic murmur; the arteries were moderately tortuous and thickened. The systolic blood pressure was 190; diastolic, 120. The liver was tender, three finger breadths below the costal margin. Electrocardiograms showed auricular fibrillation, with occasional ventricular ectopic beats. She did not respond to digitalis; she could not arrange for bed rest, and it is reported that she died six months later.

CASE 19.—Mrs. M. McC., aged 62, seen in February, 1915, had dyspnea, palpitation and broken compensation with dilatation, dating to overexertion. The heart was irregular; rate, 140; wrist, 100. The systolic blood pressure was 190; diastolic, 105. The liver was three finger breadths below the costal margin. Polygrams showed auricular fibrillation. The urine showed excessive albumin and casts. There was marked and steady improvement on digitalis and rest. The patient has been seen irregularly since, and is fairly comfortable, though she has occasional breaks in compensation. She had had "heart trouble" for years, her first attack being at 18 years of age, when she was in bed for three months with inflammatory rheumatism.

CASE 20.—Mrs. W. H. McG., aged 46, seen, March 19, 1922, in broken compensation, had not so much dyspnea as extreme weakness and palpitation. The heart was completely irregular, and electrocardiograms showed typical auricular fibrillation. Years ago she had rheumatism, when she was confined to bed for a short time. Her present trouble she dated to tonsillitis seven years ago, and an attack of influenza in January of this year. When first seen, she had just returned from a trip to California, where she had an acute cardiac breakdown, with dyspnea, cyanosis, digestive disturbance, swollen liver and irregular heart action. Examination showed the heart to be dilated to the anterior axillary line, and very irregular in force and rhythm. There was a very definite systolic murmur over the apex; at the base, near the pulmonic area, there was both a systolic and a soft diastolic murmur. The liver was enlarged and tender, three finger breadths below the costal margin. There was no edema. Convalescence was extremely slow in spite of digitalis and bed rest,

and at times the prognosis looked very serious, particularly since mental disturbances appeared, such as hallucinations and delusions; at times the patient became almost violent. These gradually cleared up with improvement in the heart, and, five months after the onset, the patient was gradually getting up and about. After undue effort there was another break in compensation, attended by extreme mental confusion, sleeplessness, orthopnea and Cheyne-Stokes breathing, followed by death, Aug. 17, 1922.

CASE 21.—Mrs. S. J., aged 38, seen, July 15, 1919, for three and a half years had palpitation and "severe pains in her heart," and could not sleep because of orthopnea. She had had frequent courses of digitalis, which now nauseated her. She had had repeated attacks of acute rheumatism eight years before. The heart was dilated outside the axillary line—true delirium cordis. The heart rate was 140; wrist, 80; there was a mitral systolic murmur. There was gradual improvement on strophanthus and digitalis, but after two weeks there was another breakdown from which the patient died in ten days. Mental confusion was a prominent symptom. Polygrams showed definite auricular fibrillation.

CASE 22.—Mrs. D., aged 36, seen, July 22, 1919, had had extreme decompensation for three or four months with remissions and recurrences, and rheumatism at 14, 19 and 25 years of age, when she was in bed from two to five months. The heart was dilated to the midaxillary line; there were mitral insufficiency and auricular fibrillation. The patient died in three months.

CASE 23.—Mr. H. C. W., aged 73, seen in April, 1921, had extreme dyspnea, dilatation of the heart and a completely irregular pulse. Electrocardiograms showed typical auricular fibrillation. The systolic blood pressure was 190; diastolic, 100. There was a pulse deficit of 30. The arteries are extremely tortuous; there was much infection about the teeth. The patient improved markedly on digitalis, but his course has been progressively downward, with periods of broken compensation. There was no history of rheumatism, but one of chronic endocarditis; over the aorta there was a roughened systolic murmur, and the second sound at the aorta was accentuated; there was marked thickening of the peripheral arteries. After recurrent cardiac breakdowns, he died in October.

CASE 24.—Mrs. C. A. N., aged 49, seen, Jan. 5, 1922, had had chronic arthritis and tonsil infection. Examination showed a mitral systolic murmur and, at the aortic area, a rather high pitched diastolic murmur. The heart was dilated to within 1 inch of the anterior axillary line. There were moderate dyspnea and cyanosis. The systolic blood pressure was 110; diastolic, 65. The liver was large, and was felt three finger breaths below the costal margin in the mid-

clavicular line. For a few years she had frequent menstruation and a year ago she had radium treatment in Chicago, for severe hemorrhages. At that time she required three blood transfusions in five weeks. For the last four years her heart has given considerable trouble, so that at one time she was in bed for two months. Polygrams were characteristic of auricular fibrillation. She died in five weeks as the result of intestinal obstruction due to carcinoma of the ovary.

In this patient there was a history of rheumatism and tonsillitis with probable involvement of the heart extending back several years, with more or less broken compensation over the last four years. During our observations she did not have any particular distress from the heart, and died as a result of pelvic carcinoma. Just how long the auricular fibrillation had been present it is difficult to say. Digitalis was of definite help while she was in the hospital.

CASE 25.—Mr. M. M. C., aged 21, seen, Oct. 6, 1921, said that his present trouble dated to May; he had very little cardiac distress, but knew that his heart was irregular. He said he had the same trouble before the tonsils were out in 1916. The pulse rate at the wrist was 72; over the heart, 124. The left border of the heart was about 1 inch outside the normal limits; on auscultation there was a low, rumbling, presystolic murmur in the sixth space; there was a soft, blowing, systolic murmur heard at the apex; the second pulmonic sound was accentuated. Electrocardiograms showed typical auricular fibrillation. He has been comfortable since, but the fibrillation persists.

CASE 26.—Mrs. D. W. C., aged 60, was seen in May, 1921, in an acute cardiac breakdown. The heart was quite regular, and there was acute pain in the region of the spleen, which was thought to be due to infarction. She had acute articular rheumatism twelve years ago which lasted four years, and she was in bed several weeks; she had another attack eight years ago which lasted over a year; she was in bed for several weeks, and now had it off and on, but not severely. The heart was bad during the rheumatic attacks. When seen a year ago, the heart was not irregular but showed mitral insufficiency. July 11, 1922, when she was able to come to the office, the heart was completely irregular, and the electrocardiograms showed auricular fibrillation.

The onset of her present trouble was with an acute broken compensation, which was controlled by a long rest in bed and very careful attention. Compensation is established so that the patient is quite comfortable.

CASE 27.—Mrs. J. E. W., aged 64, seen, Oct. 27, 1919, had broken compensation of one week's duration. Her heart was completely irregular, the rate at the heart being 102, and at the wrist, 66. There was a double mitral murmur. She stayed in bed a month, responded quickly to digitalis, and

when seen, Jan. 26, 1920, at the office, was completely comfortable, though the left heart border was one-half inch outside its normal limits, and the heart was completely irregular. In January, 1922, she went through a cholecystectomy with no heart symptoms, though auricular fibrillation still persists, with no cardiac symptoms.

CASE 28.—Mrs. C. J. S., aged 66, seen, Aug. 23, 1921, complained of "bronchitis" for about a year, fluttering of the heart, choking sensations at times producing shortness of breath, swelling of the face and of the ankles, and occasional nausea. The present trouble dated to December, 1920. She said, however, that two years ago she had a bad cold and did not recover for a long time. Examination showed the heart to be very irregular in force, volume and rhythm; the rate was 130; at the wrist, 90. The systolic blood pressure was 185; diastolic, 120. The heart was dilated to the anterior axillary line. There was a shrill systolic murmur at the apex, and the liver was three finger breadths below the costal margin. Electrocardiograms showed typical auricular fibrillation. Her course was progressively downward, and she died within five months. Digitalis, however, was of definite value, frequently tiding over periods of broken compensation.

CASE 29.—Mr. P. H., aged 49, seen, Dec. 9, 1917, was in moderate broken compensation, dating to October, 1916, when, duck shooting, he overexerted after a hearty meal. His pulse was then 160, and "he could not breathe." He had formerly had rheumatism, and had been in bed for weeks occasionally. He had been up and about the house for about a month. The heart was dilated to the anterior axillary line, and was extremely irregular; the heart rate was 140; wrist, 90. There was a mitral systolic murmur and a definite diastolic murmur. Polygrams showed auricular fibrillation. There was slow improvement on rest and digitalis. February 16, a definite, though faint diastolic bruit was noted. He continued to improve, and on April 5, walked about twelve blocks up hill, when he fainted; this was followed by two weeks' decompensation, gradually improving to August, when he was very comfortable. He was not seen then until Aug. 16, 1922, when, after driving his automobile 40 miles on a hot day and lifting a 100 pound sack from it, he suddenly became unconscious; when seen three hours later he appeared moribund, with slow, shallow respiration and very irregular, weak pulse. He had been thrashing about in bed, requiring a restraining jacket. A digitalis preparation brought improvement, and in the morning, ten hours later, he was entirely conscious and quiet. The heart was now 110; the radial pulse, 90; the heart dilated 1½ inches to the left; and there was very little cyanosis, and no dyspnea. He was kept in bed a week, improving steadily on digitalis. Electrocardiograms, August 22, showed fibrillation.

CASE 30.—Mrs. M. M. K., aged 40, seen, Aug. 30, 1922, knew that she had had heart trouble since she was 16, though the cardiac symptoms were not marked. In spite of this, she said that, last summer, at a height of 7,000 feet, she was very well and able to undergo effort without difficulty. Her complaint was that she was nervous and had headaches and dizziness frequently, some edema about the ankles in warm weather, and considerable digestive distress. She was undernourished and rather pale. There was no cyanosis, and very little respiratory effort. The heart was completely irregular; the rate at the heart was 120, and at the wrist, 92. The left border of the heart was in the midaxillary line in the sixth space. A very definite rough systolic murmur was heard over the pulmonary area. In the fifth space, near the sternum, there was a very soft diastolic murmur. The liver was felt two finger breadths below the costal margin.

In this woman of 40, with a cardiac history since she was 16 years old, auricular fibrillation was found, and there was very little evidence of heart failure. There was no history of acute rheumatism, but her tonsils were infected and she had had repeated attacks of tonsillitis with fever. She did not know how long the heart had been irregular, although she says that her liver has been enlarged for three or four months and that she had it formerly, off and on. In spite of chronic endocarditis and auricular fibrillation she has very little evidence of heart weakness, and was even able to dance last summer at an elevation of 7,000 feet, without difficulty. She knows that her heart was irregular at that time.

CASE 31.—Mr. J. K., a railroad mechanic, aged 45, seen, Sept. 25, 1922, complained of moderate shortness of breath, considerable palpitation, and pain in the precordium. There was no abdominal distress or pain except when the heart had been bad, when the liver became swollen. He had "rheumatism" in his feet in 1914, when he was in bed for a few weeks. His heart was not affected at that time, according to his memory. In 1914 he had some fluttering about his heart; this was worse in 1916 so that he was laid up for awhile. In 1920 the fluttering was worse, and he had to give up for a while on account of this and weakness. He was very well developed and muscular. The heart was completely irregular; the heart rate was 146; at the wrist, 92. There were forcible irregular beats, visitble over the precordium. The left border was palpated about 2 inches outside the normal limits. The sounds were clear except for a soft systolic murmur over the mitral area. The blood pressure was: systolic, from 150 to 170; diastolic, from 95 to 105.

While this machinist of 45 is well developed and looks quite well, he in unable to follow his trade because of palpitation and moderate dyspnea on effort. The fibrillation has been present for two or three years. He had been taking digitalis for some months. Because of negative T-waves in

the electrocardiograms in all leads, it was thought that this might be a digitalis effect. Digitalis was withdrawn for two weeks when the T waves were positive.

CASE 32.—Mr. G. W., aged 50, seen, Oct. 31, 1922, complained of smothering sensations, particularly at night, so that he could not lie flat in bed, and shortness of breath with any exertion during the day. He had to give up his work, picking apples, two weeks before, on account of dyspnea. He had had inflammatory rheumatism of two months' duration, at the age of 15, 16 and 17, and he had some trouble in his joints for several years afterward, when he knew that he had some trouble about his heart because of fluttering and pain. Eight or nine years ago, he had a very severe spell of tonsillitis, which was probably a peritonsillar abscess. Eighteen years ago he had typhoid fever, when he was ill for seven months, and he says he has never fully recuperated. He was slightly cyanotic; there was considerable respiratory effort; the left border of the heart was only 1 inch outside the normal borders in the sixth space; the heart rate was 160; the pulse deficit, 45. The liver was felt one finger breadth below the costal margin; there was no edema. Over the apex there was a very definite, soft, rather roughened, diastolic murmur. The presystolic murmur was best heard between the longer pauses; the pulmonary and aortic sounds were normal. Electrocardiograms showed typical auricular fibrillation.

This laboring man of 50 has a history of severe inflammatory rheumatism in boyhood, and severe typhoid fever at 32. The heart is now completely irregular and has been for an indefinite time. His symptoms are those of mild cardiac weakness, and he is unable to stand slight effort without dyspnea. Digitalis produced a remarkably good effect.

CASE 33.—Mr. E. E. McC., aged 50, seen, Nov. 3, 1922, complained of dyspnea on exertion, with tightness through the chest. He had been a football coach and had played football continuously for some years. He had frequent trouble with tonsillitis, but was never laid up with rheumatism. At one time he had a very severe quinsy sore throat, and the tonsils had to be lanced. At this time he was ill two weeks. He was well developed and well nourished, and did not look ill. There was complete arrhythmia. The left border of the heart was 3 inches outside the normal limits; there was a definite systolic murmur at the apex and a rougher systolic murmur at the aortic area. The arteries were not particularly thickened. The liver was not enlarged. The urine showed a trace of albumin, and some granular and hyaline casts. He did not follow instructions, and his course has been progressively downward.

CASE 34.—Mrs. J. F. S., aged 36, seen in consultation, Aug. 23, 1922, complained of dyspnea, orthopnea, pain over the

heart, cough, and swelling of the legs and abdomen during pregnancy. She had had acute rheumatism with valvular lesions, and had had occasional broken compensation. Her physician had seen her first a week before when she had oliguria, albuminuria, headaches and a systolic blood pressure of 185, diastolic, 105. She thought her heart had been irregular for some months. The heart was completely irregular and dilated to the midaxillary line, and a faint systolic murmur was heard with the stronger beats. The heart rate was 130; pulse, 100. The liver was felt four finger breadths below the costal margin; there were marked edema and ascites. She responded favorably to digitalis.

CASE 35.—Mr. J. F. C., aged 69, seen, Nov. 8, 1923, said that the night before, after sitting for two hours in the cold at a stock show, he attempted to drive his car home, when he was seized with a sense of suffocation, with an increasing amount of frothy, blood-tinged sputum. This was so severe that he had to be taken home, the attack lasting for two or three hours. He had had a similar attack two months ago from no apparent cause. When seen the next day, the left heart border was 2 inches beyond the normal limits. It was slightly irregular; the rate was 120. There was a loud and shrill systolic murmur at the apex. The systolic blood pressure was 160; diastolic, 120. The liver was two finger breadths below the costal margin, and was moderately tender. There was considerable orthopnea. For the next week he improved steadily under digitalis and rest, and was able to come to the office on the 17th, when an electrocardiogram showed definite typical auricular fibrillation with auricular extopic beats. His improvement from this time on was steady. A second electrocardiogram made one week later showed normal rhythm with negative T in Lead 1, and with marked left ventricular preponderance (amplitude 5 cm.). Digestive symptoms (anorexia, slight nausea), weakness and dizziness, and extreme sensation of coldness in the extremities were present, although progress was satisfactory. December 27, he was up and about, although there was dyspnea on effort and weakness. Electrocardiograms made at this time showed a negative T in Lead 1, and left ventricular preponderance not so marked (3 cm.). Auricular ectopic beats were present. The left heart border was only 1 inch outside the normal limits. The apical systolic murmur had become softer and roughened, and there was a soft systolic murmur over the aortic area. He was taking digitalis in small doses. January 9, he went on a motor trip to California, standing the trip very well and returning much improved. Since then he has been able to attend to his business. May 2, he had another attack of pulmonary edema; he awoke at 2 a. m., and it lasted about two hours. Electrocardiograms made three days later showed normal rhythm.

In this man of 69, who looks well and now shows but little evidence of lessened cardiac reserve, there is a history of endocardial involvement, and he shows atheromatous changes. He has had at least four acute cardiac breakdowns, the chief symptom being pulmonary edema. In the first attack, auricular fibrillation seemed to be paroxysmal and lasted three or four days, while in the second attack it was apparently of short duration. Judicious use of digitalis has now brought about cardiac compensation.

CASE 36.—Mrs. C. V. D., aged 45, was seen, March 23, 1923, for examination. Compensation was good, although she knew that the heart had been involved for twenty-five years, because at that time she had acute rheumatism, being in bed for four months. She was told that there was valvular involvement. Two years later she had severe diphtheria, which affected her heart. She had recurrent attacks of mild decompensation, but has borne three children, now aged 18, 15 and 2 years. She had much trouble with her heart during her first confinement. Formerly she had much dyspnea, but there was now very little. She was now troubled with a dull, aching pain in the precordium, and there was some edema. There were no digestive symptoms. Examination showed the left heart border 1½ inches to the left of the midclavicular line in the sixth space. The heart was irregular in force and volume. The rate was 120, and the heart sounds were clear except for a presystolic rumble at the apex. The heart had probably been irregular for years, and she thought the physician said that she had mitral stenosis at the time of the confinements. The liver was one finger breadth below the costal margin, and was slightly tender.

This healthy appearing woman of 45 shows but little evidence of cardiac embarrassment in spite of a long-standing mitral stenosis and auricular fibrillation of some years' duration.

CASE 37.—Mr. F. W., aged 45, was seen, Jan. 27, 1923, with a heart attack. Electrocardiograms showed typical auricular fibrillation, with right ventricular ectopic beats. He dated his trouble back three years, when he had arthritis, some palpitation, and cough. One year ago his legs and ankles began to swell; he had dyspnea on the slightest effort. At the age of 15 he was in bed for four months with inflammatory rheumatism; he had a second attack ten years ago, lasting two weeks. The joints were still enlarged, but he did not suffer pain. Examination showed the left heart border just outside the normal limits. There was a noticeable pulsation in the fifth space just inside the nipple line; it was very irregular. At the apex there was a definite systolic murmur. The liver was not enlarged. He has improved so much that he is able to attend to his business.

SUMMARY

In a group of patients with auricular fibrillation, about three fourths are ambulatory, while one fourth have died. Half of the entire number are comfortable and able to carry on their daily activities without cardiac symptoms. Some of these have decreased reserve on unusual effort. About one fifth of the entire number have more marked limitation of cardiac reserve, and while ambulatory, are more or less incapacitated. Most of these have needed digitalis at one time or another, or have learned how to use it to safeguard the heart. The heart is able to adjust itself to auricular fibrillation, and to compensate as it does in valvular lesions. When both fibrillation and valvular lesions (or cardiosclerosis) are present it may still compensate well for months or years, the prognosis depending on the sum total of symptoms and signs which indicate cardiac function.

Evidences of myocardial weakness are largely clinical, but changes in the Q-R-S-T complexes, in repeated electrocardiograms, aid in measuring the progress of degenerative changes.

512 Journal Building.

Reprinted from The Journal of the American Medical Association
Aug. 11, 1923, Vol. 81, pp. 440-446