Auricular fibrillation

Ross C. King
University of Nebraska Medical Center

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Part of the Medical Education Commons

Recommended Citation
https://digitalcommons.unmc.edu/mdtheses/558

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.
AURICULAR FIBRILLATION

by

Ross C. King

1932
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Introduction</td>
<td>1</td>
</tr>
<tr>
<td>2. History</td>
<td>2--7</td>
</tr>
<tr>
<td>3. Mechanism</td>
<td>8--13</td>
</tr>
<tr>
<td>4. Etiology</td>
<td>14--22</td>
</tr>
<tr>
<td>5. Pathology</td>
<td>23--25</td>
</tr>
<tr>
<td>6. Symptoms and Signs</td>
<td>26--29</td>
</tr>
<tr>
<td>7. Prognosis</td>
<td>30--33</td>
</tr>
<tr>
<td>8. Treatment</td>
<td>34--37</td>
</tr>
<tr>
<td>9. Bibliography</td>
<td>38--42</td>
</tr>
</tbody>
</table>
Introduction.

I became interested in this subject three years ago through one of the Staff members who aroused my interest in electrocardiographic work. For the past three years I have been reading some of the literature and looking up hospital reports on the subject.

I attempted to cover a portion of the available literature. I have tried to bring out the development and modern conception of auricular fibrillation, and to show the possibilities that are left for the work to be done in the future to bring the subject to complete understanding.

Auricular fibrillation should be a subject of vital interest to all medical men. It is considered to be one of the most common cardiac conditions. At least fifty per cent of all cardiac irregularities (15a) have auricular fibrillation at one time or another, and is responsible for from sixty to seventy per cent of all serious cardiac failures (47).

In this paper there will be a series of fifty cases taken from the University Hospital records after the electrocardiogram was installed. All cases have an electrocardiogram to demonstrate the fibrillation. In these fifty cases an attempt was made to determine the etiology. Etiology will be compared in general with cases of the University Hospital and cases reported from the United States and England.
History.

One of the commonest, most interesting and most important disorders of cardiac rhythm is auricular fibrillation, fundamentally a disturbance of auricular origin and attended usually by absolute irregularity of ventricular action.

Sir James Mackenzie (47) states: "In the history of medical progress it is of interest to note that while some discoveries are recognized at once and an exalted view taken of their possibilities, which a fuller experience may or may not endorse, other discoveries are not appreciated till long years after, when experience gradually reveals their significance. The condition which at one time passed under such terms as paralysis of the auricle, and nodal rhythm (27a, 5), and is now called auricular fibrillation, belongs to the latter group".

For (47) more than one hundred years clinical observers were dimly conscious of a peculiar condition of the heart which produced ill health in certain people. The description of Withering (47) of the effect of digitalis, shows that he recognized a form of heart failure which was peculiarly susceptible to the drug. After this time, when physicians recognized mitral stenosis, they came to associate it with a peculiar pulse which frequently was present in this condition called the mitral pulse. As long as forty or fifty years ago, some physicians recognized that in mitral stenosis the presystolic murmur sometimes disappeared.
The arterial pulse of auricular fibrillation is grossly irregular. It has been recorded by Marey, Somerbrodt, Riegel, Herring and others (5). In 1899 Cushing (5) described the effect of experimental faradization of the auricles in animals, which was known to produce auricular fibrillation. It had been described briefly by MacWilliams (28) in 1887. Cushing (5) noted the complete irregularity of the arterial pulse and pointed out its similarity to the completely irregular arterial pulse observed clinically. He suggested that the clinical condition is due to fibrillation of the auricles, and repeated his suggestion in a paper with Edmunds (15a) in 1907.

The venous pulse of auricular fibrillation is characterized by the absence of the "a" wave of auricular systole and the presence of a large wave due to ventricular systoles. Prominent systolic pulsations of the veins in the neck were reported by Skoda (5) in 1850 and Bomberger (5) in 1857. It remained, however, for Mackenzie (5, 27b, 47) to point out in 1902, that the ventricular form of venous pulse is associated with complete irregularity of the arterial pulse, which at this time he thought was due to paralysis of the auricle. Mackenzie still adhered to auricular paralysis in 1904. In 1907 (27b) he came out with the theory of simultaneous contractions of the auricles and ventricles with the impulse arising in the junctional tissues. He later abandoned nodal rhythm when true cases of such were demonstrated.

The small auricular waves occurring in diastole were described in 1907 by Wenckebach and 1908 by Herring. Herring contributed the cause to tricuspid insufficiency. The small auricular waves in the venous

(3)
pulses have been recorded by Lewis, Jolly and Ritchie, Rihl, Wiggers, Niles and Wiggers, and others (5, 15c). Lewis, Jolly and Ritchie, and Rihl attributed them to auricular fibrillation, but Wiggers and Niles attributed them to other mechanical phenomena because they did not correspond to the intra-auricular pressure curves (38).

Auricular fibrillation was first recorded electrocardiographically by Einthoven in 1906. Characterized by absence of normal "p" wave, presence of small auricular oscillations, complete irregularity of the ventricles, and ventricular complexes of supraventricular type. The small auricular oscillations were attributed to fibrillation of the auricles and were shown to be independent of somatic tremor. Drury and Iliescu (11) subsequently demonstrated that, although they varied considerably from one instant to another, the presence and amplitude of such oscillations were independent of respiration (21). Jolly and Ritchie (5) estimated the rate of auricular oscillations to be 390 to 522 per minute in human beings, while Drury and Iliescu (11) found it to vary from 300 to 600 with an average of 473.

Lewis and Rothberger and Winterberg (46) independently and almost simultaneously presented evidence which demonstrated conclusively that complete irregularity of the heart is due to fibrillation of the auricle. P. D. Whites(46) translation of Rothberger and Winterbergs article reads that: "Although the pulsus irregularis perpetuum has been known to clinicians a long time, it has only been described in recent years by Herring (1903), as a special form of cardiac arrhythmia, characterized by particular features".

"The normal venous pulse curve shows with each heart beat three characteristic upstrokes, the first which proceeds the apex Impulse,
that is, the pulse in the greater arteries, and represents auricular systole.

"In arrhythmia perpetua this wave which gives us evidence of activity of the right auricle, is entirely missing, and the phlebogram shows the characteristics of the so called positive or ventricular venous pulse."

"There has long been known in animal experimentation a very irregular heart rhythm, namely auricular fibrillation, which is identical in all details with the signs of arrhythmia perpetua."

"In auricular fibrillation more or less delicate fibrillary movements occur which are of no significance for the movement of blood; as a result in the phlebogram the pre-ventricular wave is missing and a positive venous pulse develops."

"Auricular fibrillation is accompanied by absolutely irregular ventricular action, transmitted to the pulse; this arrhythmia is of exactly the same character as that which we find in arrhythmia perpetua."

The authors then described electrocardiograms from experimental animals and patients, pointing to the three points of similarity.

1. Absolute ventricular arrhythmia; 2. absence of "P" waves; 3. presence of irregular oscillations of the galvanometer string due to the fibrillary waves themselves.

Lewis (5, 24b) based his conclusions on clinical and experimental observations which were in close agreement; in the experiments fibrillation of the auricles was known to be present. In each the radial
pulse was completely irregular, both rhythm and force; the venous pulse was of the ventricular type; the "a" waves were absent, and rapid small undulations were present in the longer diastoles; the electrocardiogram showed the ventricles to be irregular with complexes of the supraventricular type, the absence of "P" waves, and the presence of continuous oscillations were shown to be of auricular origin. Lewis (5, 24a) had the opportunity to examine the heart of a horse with an irregular irregular pulse immediately after it was killed and witnessed the auricles fibrillating. Cutler, Levine and Beck (5) witnessed the fibrillation of the auricles in a human and described it as irregular convulsive contractions.

Engelmann (5) in 1895 established that ventricular fibrillation was due to multiple foci in the muscle of the ventricle and applied the same theory to the auricles. Winterberg, Lewis, Rothberger and Winterberg, held to this same theory as to mechanism. Lewis then developed his idea in the theory of heterogenesis, while Rothberger and Winterberg gave it the name of tachysystole. In 1912 and 1913 Lewis (5, 24b) pointed out that auricular extrasystoles, paroxysmal auricular tachycardia, auricular flutter and fibrillation had certain features in common. He stated in the conclusions, that (1) "single premature auricular contractions, (2) small groups of the same, (3) paroxysms of tachycardia from single auricular foci, (4) auricular flutter, (5) paroxysms of tachycardia from two or more auricular foci, and (6) auricular fibrillation arise essentially in the same manner; namely, through the pathological of heterogenetic origin of new impulses in the auricle are clearly suggested by the facts at our disposal". In 1914 Rothberger and Winterberg attribu-
ted auricular fibrillation to a pronounced acceleration of the auricles to rates of 3000 to 3500 per minute, but Lewis believes that they failed to distinguish between fibrillation and the state of rapid re-excitation.
Mechanism.

Our knowledge of fibrillatory contraction of heart muscle as produced experimentally, begins with the report of Hoffa and Ludwig (15c) in 1850. Ventricular fibrillation was produced by them in cold blooded and mammalian hearts by electrical stimulation, strong, rapidly repeated shocks being delivered by a rotation machine in an endeavor to produce tetanus, or rather a condition of persisting contraction immediately about the electrodes; but in the rest of the heart contractions were recorded which were weak and absolutely irregular. Einhodt's (15c) description in 1874 is essentially that of Ludwig.

Fibrillation was referred to by Heidenhain (15c) as a tumultuous tetanus. Schleski (15c) speaks of it as a tetanus with intermission. Both Goltz and Cyon (15c) refer to it as tetanus, although Cyon also refers to fibrillation in the frog's ventricle as a peristaltic contraction. The impossibility of a heart tetanus disposed of that conception.

Vulpian (15c) in 1874 describes the result of direct faradization of the ventricles of a dog in which he obtained fibrillation. He saw only the ventricles fibrillate while the auricles beat rhythmically.

Albert and Dehn (15c) (1874) believed that potassium salts caused fibrillation by injury to a "co-ordination center", a nervous structure in the heart. Kronecker and Schmey (15c) punctured the heart and destroyed the co-ordinating center and the blood supply and stressed these points as a cause of fibrillation. MacWilliams (15c) duplicated their
experiment and disproved their theory by obtaining recovery from fibrillation.

Cohnheim (15c) explained the delay in the onset of fibrillation on coronary ligation as due to slow development of a "metabolic poison" which acted as a unidentified stimulus.

Romanes (15c)(1876) noted the rhythmic contractions of the umbrella of the jelly fish. MacWilliams (28, 15c) (1887) discussed the fibrillar contractions of the heart and showed the impulse to pass through the muscle cell. MacWilliams explained fibrillation with increased and with decreased excitability. He brought out clearly that usually conduction is decreased, that the impulse progresses at different rates and reaches adjacent muscle bundles at different points of time, and, since these are connected with one another by anastomosing branches, the contraction would naturally be propagated from one contracting fiber to another over which the contraction wave had already passed. There would evidently be a more or less rapid series of contractions in each muscle bundle in consequence of the successive contraction waves reaching that bundle from different directions along its anastomoses with other bundles.

Porter (15c) (1894) came out with his block hypothesis. He postulated the development of intramuscular blocks which served to deflect the course of the impulse and to conduct it into abnormal and devious paths, so that it may be conceived as shuttling about or weaving its way through the mass of cardiac tissue.

Struck by the fact that different parts of the fibrillating heart chamber are not in the same phase of contraction, Engelmann (15c) (1895)
proposed that, in fibrillating contraction, the different heart fibers become independently rhythmic, and that each is a focus of impulse formation. When we conceive that these independent, multiple ectopic foci have inherently different rhythms, and that in addition, each may be beating at its maximal rate (Hoffman-1905) (15c) we have an idea of the degree of inco-ordination which is believed to prevail in atricular fibrillation. This theory was elaborated by Winterberg (46) in 1906 and adopted by many prominent workers including Rothberger and Winterberg, Herring, Rihl, Haberland and Lewis, and for this reason has dominated the literature for years.

Mayer (1908) demonstrated a single contraction wave can be made to pass in one direction continuously around a ring cut from the disc of a jelly fish. Mines (15b, 15c, 5) 1913 confirmed Mayer's observation with rings cut from the auricle of a ray fish and suggested this as the basis of delerium cordis.

Garrey (15b, 15c, 5) (1914) disproved the acceptable theory of the day a stated: "the persistance of cardiac fibrillation is, other things being equal, directly proportional to the size of the tissue masses involved whether the pieces are cut from hearts already fibrillating or are faradically stimulated to start the process in them". The form of the tissue is important; for long narrow or thin pieces recover promptly, and narrow strips when connected with a fibrillating mass or when faradically stimulated do not fibrillate, but beat co-ordinately. Tissue rings cut from fibrillating hearts of marine turtles ceased fibrillating, but the contraction waves continued, repeating the circuit about the ring in coordinate "circular contractions". Sufficiently nar-
row bridges of any portion of the musculature of auricles or ventricles will prevent the extension of the fibrillar process and act thus like the auriculo-ventricular conducting bundle. When fibrillation is induced by localized faradization this focus may be subsequently excised, its inco-ordination will cease, just as will that of any other piece of similar size, while that of the remaining (larger) mass will continue, showing that the process, therefore, involves the whole tissue mass and is not sustained by impulses arising in any definite location.

The experiment supports the block hypothesis and suggests that the blocks probably result in intramuscular ringlike circuits with resulting "circus contractions" which are fundamentally essential to the fibrillar process. Such ring circuits can exist in large masses but not in sufficiently small ones.

It remained for Lewis and collaborators (5, 11, 24d, 25, 54) in 1920 and 1921 to prove the mechanism of auricular fibrillation. Circus movement in the auricles is described as a wave traveling in a circular path, the crest of the wave advancing into responsive muscle, but followed by a wake of refractory muscle. Between the retreating wake of refractory muscle and the crest of the advancing wave is a gap of responsive muscle. The presence of this gap of responsive muscle in a circle of refractory muscle is essential for the maintenance of the circus movement. If the gap of responsive muscle should be abolished the crest of the advancing wave meeting refractory tissue which could not receive it, the circus movement would then cease unless a longer path were open to it. The extent of the gap depends upon three factors:
(1) length of the path, (2) the rate of conduction, (3) and the duration of the refractory period. The gap is increased if the path becomes longer, if conduction becomes slower, or if the refractory period becomes shorter; it is decreased or abolished if the path becomes shorter, if conduction becomes more rapid, or if the refractory period becomes longer. These factors are in themselves closely interdependent.

Analysis (11, 150, 240) of auricular disorders produced experimentally and yielding electrocardiograms similar to those of clinical fibrillation of the auricle shows a condition in which a single excitation wave circulates continuously through the auricular muscle. The path taken both by the central and centrifugal parts of this wave is sinuous and varies in greater or lesser degree from cycle to cycle. Thus auricular fibrillation, as it occurs clinically, is an advanced variety of impure flutter. The sinuous course of the waves is attributed to barriers of refractory tissue; the chief difference between slightly impure flutter on the one hand and fibrillation on the other is that in the latter the barriers are of greater extent and of more frequent occurrence. In pure flutter the effective refractory period is somewhat longer than in impure flutter and fibrillation.

Therefore, the mechanism of auricular fibrillation and auricular flutter, as described by Lewis (240), is due to a disturbance in the pace maker of the heart, whereas normally the impulses regulating auricular contraction arise in the sinus node. During auricular flutter and fibrillation a circus movement has been established in the circular muscle which surrounds the superior and inferior vena cava. In
auricular flutter the impulses responsible for auricular stimulation come from the circus at very rapid but regular intervals due to a uniformity in the refractory period of the circular muscle. In auricular fibrillation the impulses come from the circus at irregular intervals thus causing the auricles to contract rapidly but irregularly.
Etiology

Auricular fibrillation is a frequent cause of heart failure; according to Mackenzie (47, 27a) the most frequent cause. Once auricular fibrillation occurs it generally remains established for the rest of the patient's life. Occasionally it is paroxysmal in character, lasting for a few hours or few days only, but recurring.

Among the fifty cases presented we are attempting to show some of the etiological factors of auricular fibrillation.

In 17 of the 50 cases there was definite cardiac decompensation. Heart symptoms had been present, as far as the patient was able to remember, from nine months to 43 years in 15 of the cases. One case was very indefinite and thought that probably she had had a bad heart all her life. Another case absolutely had no knowledge of the duration of her heart symptoms previous to her entrance to the hospital. The average age of these 17 cases was 67 years, the range of age was from 51 to 78. These cardiac decompensation cases were all probably on a myocardial basis.

There were 14 cases which clinically showed either a mitral stenosis or mitral insufficiency or both. The cardiac symptoms ranged from one month to 30 years. The average age was 42 years, the range of age was from 28 years to 70 years. These cases fall under the rheumatic group.

There were 14 cases of thyroid of a toxic nature with an average age of 54 years and a range of age from 37 to 66 years. The duration of cardiac symptoms in this connection ranged from two months to 11 years.
Three cases of nephritis, one case presented no cardiac symptoms and the other two cases had symptoms referable to the heart for one year duration.

The group falling under syphilis had cardiac symptoms for two years in one case and no symptoms in the other case.

A tabulation of the etiology of the 50 cases of auricular fibrillation at the University of Nebraska Hospital is:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Average Age</th>
<th>Cases</th>
<th>Range of Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial Degeneration</td>
<td>67</td>
<td>17</td>
<td>51--78</td>
</tr>
<tr>
<td>Rheumatic Valvular Disease</td>
<td>42</td>
<td>14</td>
<td>28--70</td>
</tr>
<tr>
<td>Toxic Thyroid</td>
<td>54</td>
<td>14</td>
<td>37--66</td>
</tr>
<tr>
<td>Arterial Disease and Hypertension</td>
<td>67</td>
<td>3</td>
<td>51--79</td>
</tr>
<tr>
<td>Syphilis</td>
<td>60</td>
<td>2</td>
<td>57--53</td>
</tr>
</tbody>
</table>

A tabulation of the etiology of auricular fibrillation taken from Campbell's article in Guy's Hospital Report (48) shows the following:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Average Age</th>
<th>Cases</th>
<th>Range of Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatic Valvular</td>
<td>36</td>
<td>53</td>
<td>18--56</td>
</tr>
<tr>
<td>Diseases of Thyroid</td>
<td>45</td>
<td>11</td>
<td>35--50</td>
</tr>
<tr>
<td>Hypertension</td>
<td>61</td>
<td>12</td>
<td>54--72</td>
</tr>
<tr>
<td>Arterial Disease</td>
<td>63</td>
<td>14</td>
<td>52--76</td>
</tr>
<tr>
<td>Various: Bronchitis and Syphilis</td>
<td>58</td>
<td>4</td>
<td>56--53</td>
</tr>
<tr>
<td>No Apparent Disease</td>
<td>49</td>
<td>6</td>
<td>41--54</td>
</tr>
</tbody>
</table>

Lewis (24b) gave an account of 114 cases of complete irregularity of the heart. Of the 114 cases he found 57 or 50 per cent of the cases to be auricular fibrillation. In basing his report on etiology he used
73 of the 114 cases and found 43 were males and 30 were females with age limits ranging from 13 to 84 with an average age of 41.9 years. He also found that 26 of the cases had a previous history of rheumatic fever or chorea. A summation of his work is:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Rheumatic or Family Rheumatic History</th>
<th>No Rheumatism</th>
<th>Not Noted</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral Stenosis</td>
<td>21</td>
<td>1</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Complete Irregularity</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Arterial Disease</td>
<td>6</td>
<td></td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Pericardial Adhesions</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Gramular Kidney</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Aortic Disease</td>
<td>2</td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Remainder</td>
<td></td>
<td>8</td>
<td>3</td>
<td>11</td>
</tr>
</tbody>
</table>

Brachman (7) in studying 400 cases of auricular fibrillation in out patient and in patient clinics shows:

<table>
<thead>
<tr>
<th>Age and Sex.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>16-20</td>
</tr>
<tr>
<td>Out-Patients; M.</td>
<td>3</td>
</tr>
<tr>
<td>F.</td>
<td>4</td>
</tr>
<tr>
<td>In-Patients; M.</td>
<td>0</td>
</tr>
<tr>
<td>F.</td>
<td>1</td>
</tr>
</tbody>
</table>

Preexisting Disease:
A-Rheumatic fever; B-No previous illness; C-Influenza; D-Rheumatism; E-Chorea; F-Scarlet Fever; G-Graves Disease; H-Enteric Fever; I-Small Pox; J-Diphtheria; K-Syphilis.
Cookson (9b) states there are three common etiological groups in which auricular disorder is persistent; (1) Rheumatic fever heart disease, (2) age or arteriosclerotic, (3) exophthalmic goitre or some toxic thyroid state. In the London Hospital 361 cases of auricular fibrillation were studied, 16 per cent had rheumatic heart disease or a history of rheumatic fever, 79 per cent non-rheumatic type, 7 per cent exophthalmic goitre, 2 per cent syphilitic aortitis.

On close observation it will be noted that in cases reported there were none under the age of ten. Up till 1925 there was only five cases reported in a child below ten. Ogden (29) reviewed the literature up to this time. Fifteen cases of auricular fibrillation were then discovered in the London Hospital after close check up was made in the children below ten. The greatest per centage of these cases were on a rheumatic basis although one was thought to be due to diphtheria. There were two cases of persistent auricular fibrillation in children of six and four respectively due to rheumatic fever (9a, 26, 33). In a study of the cases in children with a rheumatic fever history there seems to be three types of fibrillation; (1) paroxysmal, (2) terminal, (3) chronic.

From the articles read it may be stated that auricular fibrillation in children is mostly due to rheumatic fever. There were no cases report
ed in the list taken from the University Hospital.

Anderson (2) in 129 cases reports of hyperthyroidism with auricular fibrillation states: "it is a early occurrence for auricular fibrillation to begin early in the disease and if not treated will be very apt to become a fixed fibrillation". Swan (40) reports a case with myxedema. The patient was given thyroid extract. The dose was given till the patient showed signs of improvement. At the stage of maximum dosage the patient had auricular fibrillation. The medication was stopped and the fibrillation ceased. The course of medication was administered till three different occasions the patient went into fibrillation.

Hay and Jones (16) report cases in which there were no heart symptoms until a strain, mental and physical developed an auricular fibrillation. They reported five cases of fibrillation following mental shock, electric shock, lifting of a heavy box, fright from a dog and running for a street car respectively. In Smith's (36) reports of auricular fibrillation he found that by removing foci of infection such as teeth and tonsils that in one case fibrillation ceased after removal of the infection. Fowler and Baldridge (12) report cases after an abdominal operation starting fibrillation, who did not have any previous heart trouble. They explained this as a basis of vagus stimulation.

In conclusion, the most common etiological factors are rheumatic valvular disease, arteriosclerotic, age, or myocardial degeneration, toxic thyroid, and syphilis respectively. There are various sundry causes such as operations, trauma, medication, and foci of infection which seem to play their role. In comparison, the University of Nebraska reports run in close harmony with the English and American reports submitted to the literature. All literature seems very scanty on reports of children below ten years of age.

(18)
<table>
<thead>
<tr>
<th>Hosp. No.</th>
<th>Age</th>
<th>Cardiac Sym. Prev. to Admittance</th>
<th>Clinical Diagnosis other than Heart</th>
<th>Cardiac Vascular Diagnosis</th>
<th>Outcome</th>
<th>Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>26618</td>
<td>72</td>
<td>4-5 years</td>
<td>Epithelioma</td>
<td>Arteriosclerosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27495</td>
<td></td>
<td></td>
<td>Spastic Entropium</td>
<td>Decompensation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rt. Bd. Br. Block</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nodal Rhythm</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bredycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27690</td>
<td>87</td>
<td>43 years</td>
<td>Chronic Hypertension</td>
<td>Myocardial Degeneration</td>
<td>Removed</td>
<td></td>
</tr>
<tr>
<td>28452E</td>
<td>78</td>
<td>All of life</td>
<td>Retroce and Prolapse</td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>28707</td>
<td>51</td>
<td>10 years</td>
<td></td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>29817</td>
<td>71</td>
<td>5 years</td>
<td></td>
<td>Coronary embolus</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td>Dropped Beats</td>
<td></td>
</tr>
<tr>
<td>30977</td>
<td>75</td>
<td>Unknown</td>
<td>Acute Cholecystitis</td>
<td>Arteriosclerosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>30819</td>
<td>79</td>
<td>1 year</td>
<td>Head injury</td>
<td>Myocardial failure</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>30820</td>
<td>87</td>
<td>2 years</td>
<td></td>
<td>Chronic myocarditis</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>30898</td>
<td>67</td>
<td>1 year</td>
<td>Senile Dementia</td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Traumatic Ulcer--Leg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30935</td>
<td>88</td>
<td>3 months</td>
<td>Carcinoma of Gall Bladder</td>
<td>Myocarditis</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>32469</td>
<td>70</td>
<td>3 months</td>
<td>Varicose Ulcers</td>
<td>Cardiac Decompensation</td>
<td>Dismissed not fibrillating</td>
<td></td>
</tr>
<tr>
<td>32614</td>
<td>70</td>
<td>35 years</td>
<td></td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>34171</td>
<td>57</td>
<td>1 year</td>
<td>Pharyngitis</td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>34436</td>
<td>75</td>
<td>1 year</td>
<td></td>
<td>Chronic Myocarditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Coronary Thrombosis</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>34181</td>
<td>57</td>
<td>1 year</td>
<td>Tonsillitis</td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td>Unimproved</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thrombosis left Leg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34665</td>
<td>69</td>
<td>9 months</td>
<td>Endarteritis Obliterans</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>35305</td>
<td>65</td>
<td>5 years</td>
<td>Cardiac Decompensation</td>
<td>Dismissed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hosp. No.</td>
<td>Age</td>
<td>Years</td>
<td>Cardiac Symp. Prev.</td>
<td>Clinical Diagnosis other than Heart</td>
<td>Cardio Vascular Diagnosis</td>
<td>Outcome</td>
</tr>
<tr>
<td>----------</td>
<td>-----</td>
<td>-------</td>
<td>-------------------</td>
<td>------------------------------------</td>
<td>---------------------------</td>
<td>---------</td>
</tr>
<tr>
<td>27167</td>
<td>46</td>
<td>3</td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>27242</td>
<td>54</td>
<td>3-4</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Arterial Embolism</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>27576</td>
<td>23</td>
<td>6</td>
<td>Pulmonary T.B.</td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic Cholecystitis</td>
<td></td>
<td>Aortic Insufficiency</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>27711</td>
<td>46</td>
<td>3</td>
<td>Chronic Rheumatism</td>
<td></td>
<td>Chronic Rheu. Endocarditis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>28170</td>
<td>39</td>
<td>30</td>
<td>Epistaxis</td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral insufficiency</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>30150</td>
<td>41</td>
<td>1</td>
<td></td>
<td></td>
<td>Mitral stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>30824</td>
<td>35</td>
<td>6</td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Hypertrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral Insufficiency</td>
<td></td>
</tr>
<tr>
<td>30653</td>
<td>44</td>
<td>12</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral Insufficiency</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>31623</td>
<td>62</td>
<td>4</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td>31742</td>
<td>70</td>
<td>1</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Chronic Myocardial Degen.</td>
<td></td>
</tr>
<tr>
<td>32414</td>
<td>35</td>
<td>14</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fibrillating</td>
<td></td>
</tr>
<tr>
<td>32917</td>
<td>43</td>
<td>43</td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Fibrillation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fibrillating</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Dismissed</td>
<td></td>
</tr>
</tbody>
</table>
### Mitral Stenosis and Insufficiency Continued

<table>
<thead>
<tr>
<th>Hosp. No.</th>
<th>Age</th>
<th>Cardiac Sympt. Prev. to admittance</th>
<th>Clinical Diagnosis other than heart</th>
<th>Cardio Vascular Diagnosis</th>
<th>Outcome</th>
<th>Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>32861</td>
<td>66</td>
<td>1 year</td>
<td></td>
<td>Mitral Insufficiency</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral Stenosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac Decompensation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Antepulse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32829</td>
<td>65</td>
<td>3 years</td>
<td></td>
<td>Mitral Stenosis</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mitral Insufficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Auricular Fibrillation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Toxic Thyroid

<table>
<thead>
<tr>
<th>Hosp. No.</th>
<th>Age</th>
<th>Cardiac Sympt. Prev. to admittance</th>
<th>Clinical Diagnosis other than heart</th>
<th>Cardio Vascular Diagnosis</th>
<th>Outcome</th>
<th>Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>27503</td>
<td>66</td>
<td>11 years</td>
<td>Bronchial Pneumonia</td>
<td>Cardiac Decompensation</td>
<td>Died</td>
<td>Chronic Myocarditis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Toxic Thyroid</td>
<td>Auricular Fibrillation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27657</td>
<td>57</td>
<td>1 year</td>
<td>Tox Adenomatous Thyroid</td>
<td>Cardiac Decompensation</td>
<td>Died</td>
<td>Lt. Vent. Hypert. Inst. Fibres of heart</td>
</tr>
<tr>
<td>28175</td>
<td>68</td>
<td>3 years</td>
<td>Lobar Pneumonia</td>
<td>Toxic Myocarditis</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Interstitial Nephritis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Goitre</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29526</td>
<td>46</td>
<td>2 years</td>
<td>Tox Adenoma</td>
<td>Cardiac Decompensation</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29735</td>
<td>59</td>
<td></td>
<td>Toxic Thyroid</td>
<td>Cardiac Decompensation</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fracture-Lit. Femur</td>
<td>Auricular Fibrillation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Eyeliditis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30632</td>
<td>58</td>
<td>5 years</td>
<td>Tox Adenoma Thyroid</td>
<td>Endocarditis</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>30493</td>
<td>63</td>
<td></td>
<td>Chronic Tonsillitis</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>32996</td>
<td>60</td>
<td>8 years</td>
<td>Gen. Crema Rt. Index</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>34049</td>
<td>47</td>
<td>3 years</td>
<td>Hyperthyroidism</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>30166</td>
<td>37</td>
<td>3 months</td>
<td>Exophthalmic Goitre</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>30585</td>
<td>58</td>
<td>5 years</td>
<td>Tox Adenoma</td>
<td>Myocarditis</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>34049</td>
<td>47</td>
<td>3 years</td>
<td>Hyperthyroidism</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>51580</td>
<td>42</td>
<td>1 year</td>
<td>Hyperplastic Thyroid</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>54727</td>
<td>51</td>
<td>2 months</td>
<td>Toxic Thyroid</td>
<td>Auricular Fibrillation</td>
<td>Dismissed</td>
<td></td>
</tr>
<tr>
<td>Hosp. No.</td>
<td>Age</td>
<td>Cardiac Sym.</td>
<td>Prev. to Admittance</td>
<td>Clinical Diagnosis other than Heart</td>
<td>Cardio Vascular Diagnosis</td>
<td>Outcome</td>
</tr>
<tr>
<td>-----------</td>
<td>-----</td>
<td>--------------</td>
<td>---------------------</td>
<td>-----------------------------------</td>
<td>--------------------------</td>
<td>---------</td>
</tr>
<tr>
<td>28698</td>
<td>24</td>
<td>None</td>
<td>Multiple Sclerosis</td>
<td>Nephritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30826</td>
<td>68</td>
<td>1 Year</td>
<td>Chronic Vascular Nephritis</td>
<td>Chronic Vascular Hypertension Incomp. Rt. Bd. Br. Block</td>
<td>Arteriosclerosis</td>
<td>Dismissed</td>
</tr>
<tr>
<td>34436</td>
<td>75</td>
<td>1 Year</td>
<td>Chronic Nephritis</td>
<td>Chronic Myocarditis</td>
<td>Coronary Thrombosis</td>
<td>Died</td>
</tr>
</tbody>
</table>

### SYPHILITIC

<table>
<thead>
<tr>
<th>Hosp. No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>28561</td>
<td>63</td>
<td>Post Traumatic Headache</td>
<td>Dismissed</td>
</tr>
<tr>
<td>33697</td>
<td>57</td>
<td>Syphilis</td>
<td>Dismissed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cerebral Embolism</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arteriosclerosis</td>
<td></td>
</tr>
</tbody>
</table>
Pathology

As attention was drawn successively to the auricular ventricular node, and sino-auricular node, investigators sought in the morbid anatomy of these structures the explanations of the various disorders of the heart beat. The lesions which were found did not always have the meanings at first attributed to them. Nevertheless these observations demonstrated that anatomical changes are frequently present in the auricular muscle of patients with auricular fibrillation. Müller (1905) and Wenckebach (1907) reported lesions of the auricular muscle. Schönberg in 1909 (5, 14) described areas of lymphocytic infiltration and of fibrosis in the auricular muscle of cases of auricular fibrillation, but found similar lesions in cases without fibrillation. In 1910 Hedinger found lymphocytic infiltration and granulation tissue in the auricular walls in cases of fibrillation, and Koch described sclerotic changes in the sino-auricular node. Mackenzie (47, 5) stated in 1913 that, "while no lesion is constantly associated with auricular fibrillation, and the special tissue is not necessarily affected, yet anatomical changes, myocardial degeneration, cellular infiltration, or anemic infarction due to coronary disease are often found. In 1914 Jarish (5) reviewed the literature and reported frequent occurrence of fibrous changes or cellular infiltration in the auricular wall in cases of auricular fibrillation. Lenoble in 1921 described fibrosis of the auricular muscle. Freethingham (46) was unable to establish any relationship between anatomical changes in the auricle and the presence of auricular fibrillation. Cowan and Ritchie (5) have summarized the subject as follows:
"Our own histological observations and those of others indicate that
the auricular muscle is usually affected by a diffuse or patchy fib-
rosis, with or without cellular infiltration and other signs of sub-
acute or chronic myocarditis. The essential lesions must be sought
for in the auricular walls rather than in the primitive tissue, be-
cause the sinus node is not necessarily involved in the inflammatory
process, whereas it may be inflamed although the auricular contract-
ions are co-ordinate and rhythmic".

There remains a group of cases of auricular fibrillation in which
no demonstrable anatomical changes are found in the auricular muscle.
One is reminded of the occasional patient with abundant clinical evi-
dence of myocardial disease, who may even die of myocardial failure,
in whom the pathologist find no abnormality of the heart muscle. The
answer is to be found in the pathological physiology of the tissue
cells, in physico-chemical changes that defy detection by our present
anatomical methods. In 1910 Lewis (24b) suggested that local anemia
of the auricular muscle might be a factor in the production of auricular
fibrillation and he and Mathison showed that in cats asphyxia slows the
sinus rhythm and depresses the auricular ventricular conduction. Wil-
son (5) in 1923 stated that, "in persistent auricular fibrillation is
invariably associated with changes in the heart muscle, which may, how-
ever, be physico-chemical in nature and impossible of demonstration at
post mortem. The studies of Carter, Andrus and Dieunade (5) are of
importance in this connection. They found that anoxemia in contractile
tissue increases the hydrogen ion concentration, and that an increase
in the hydrogen ion concentration increases the refractory period and
decreases conduction. This may lead to the appearance of ectopic rhythms,
re-entrant waves and circus movements may be accounted for by regional or diffuse accumulations of hydrogen ions. Resnik (32) reported that when vagal tone is removed and the auricles are stimulated by a faradic current, the early effects of anoxemia is to predispose the auricles to fibrillation; the late effect is to inhibit the development of fibrillation.

From a study of all these cases it was evident that there was no typical lesion in the auricles that could be looked on as the cause of auricular fibrillation. A slight degeneration lesion was found in the muscle fibers of the auricles much more frequently with auricular fibrillation than in regular beating hearts of the same age. It is interesting to speculate whether the lesion was the result of the irregularity of rhythm or a factor in its production, by causing some defect in the conduction of impulses or in the ability of the muscles to respond to the impulses.
Symptoms and Signs.

It must be acknowledged that subjective manifestations are few in number (15a, 15b) and that they may be almost entirely absent. A certain degree of shortwindedness (21) during or after exertion, an occasional fluttering in the neck or chest may be experienced. A general feeling of ill health, often associated with easy exhaustion, is not uncommon. Gastric discomfort and loss of appetite are not infrequent. The symptoms are prominent in nervous subjects. Such are the main disturbances in cases in which the ailment is of short standing.

There appears to be a special symptomatology, or, speaking more correctly perhaps, a more profound disturbance at the time of the onset of the irregularity. The actual onset has probably never been recorded, but the symptoms at observations at or about the onset all point to its being as sudden as it is in case of regular paroxysmal tachycardia. In one case the irregularity commenced while the patient lay in bed and shortly after waking. He was seized by violent palpitation in the chest, a choking feeling in the throat, and inability to "catch his breath".

The symptomatology in the majority of patient's cases in which obvious valvular disease or dilatation is present, is that of decompensation in its various degrees. It is not in any way indentified with the condition of the irregularity itself.

Brachman (7) in a series of 400 cases compares 200 cases with fibrillation and 200 cases without fibrillation as to symptomatology. A-Dyspnea; B-Pain; C-Palpitation; D-Vertigo; E-Cough; F-Weakness; G-Insomnia; H-Nervousness; I-Choking Feeling; J-Fainting (Unconscious) K-Irrregularity.
The radial pulse irregularity is of the most varied description. The pulse may be slow or fast, and the variation in rate is great (23). Variation is 30 to 200 per minute. The beats may be of small excursion, more commonly there is a haphazard intermingling of forcible and weak contractions and the latter are often markedly dicrotic (44). The radial pulse is often but an indifferent index of the rate of the ventricle (10) many beats are not transmitted. The pulse rate may be considerably reduced, either as a result of "dropped" beats or as a consequence of the actual slow speed of the ventricle. The beats may show coupling over short or long stretches of curves. The fast types are the commonest, and in those the usual rate of the ventricle is approximately double the normal rate (110—150). It is usually at these fast rates that the disorderly character of the pulsation is so prominent, nevertheless, it is always present, a fact which can be determined by careful measuring of the tracings (15b). Where the grade of disorder of irregularity is high the condition may be recognized by feeling the pulse; and with experience even the lesser grades of disorder, for example those met with in cases where the pulse is slower, can be identified by similar means, though the method is inevitably uncertain. The disorder may be recognized by two criteria. First the most important is the absolute character of the arrhythmia. The heart rhythm is never regular, and seldom or never do two beats of the same character of length succeed each other. In a long curve it is rare to find any two short sections of
tracing which have a superficial resemblance to each other. The pauses between the beats bear no relationship to one another. The second criterion consists in the absence of a definite and continued relationship between the strength of a beat and the length of the pause which precedes it. A strong beat may follow a short pause and a weak beat may succeed a long pause.

In the normal tracing taken over the jugular vein (37) the so called c wave, which corresponds in time with the carotid pulse is preceded by an a wave due to the contraction of the auricle. In similar tracings (18a) taken from patients with absolute irregularity the "a" wave (27b) is absent and the tracing rises abruptly without any preceding auricular wave. It is now generally conceded that the first wave on such abnormal tracings, though corresponding in time with the carotid pulse, is not due to a transmitted arterial pulse, but is sent back into the vein from the heart.

It is believed the "v" wave is due to the beginning of ventricular contraction. It can be demonstrated in tracings taken directly over the normally acting auricle, either internally from the esophagus (39) or externally when the bony chest wall is deficient (18a). In normal jugular tracings this wave is generally diminished or absent because it is taken up by the diastole of the auricle; but when the auricular systole is absent, it is transmitted back through the filled auricle and becomes a marked feature of the venous pulse.

Mackenzie(47) has called attention to the remarkable change that takes place in the murmur of mitral stenosis when the heart assumes an absolutely irregular rhythm. The characteristic presystolic accentuation
heard ordinarily is caused by the contraction of the auricle which in-
creases the rate of blood flow through the constricted mitral orifice
just previous to ventricular systole. When a patient develops an ab-
solutely irregular rhythm the mitral stenosis murmur may disappear en-
tirely. If it persists it loses its presystolic accentuation.

According to Erachman (7), the three most constant and diagnosable
signs found in auricular fibrillation are (1) the irregular irregularity
of the heart, (2) enlarged transverse diameter of the heart, (3) a dif-
ference in the apex and pulse rate, pulse deficit.

Electrocardiograms (24a) taken from patients exhibiting the irreg-
ularity show a number of irregular waves apart from the ventricular
curve; they are more clearly defined in diastole. They are found in
no other disorder of the heart's action. They disappear when, in a
paroxysmal case, the irregularity vanishes, and are therefore due to a
temporary and disorderly action of some part of the auricular wall.
Cardiographic curves give no evidence of such disordered action in the
ventricles. Fibrillation of the auricle yields curves which are ident-
ical in every respect, and no such curves have been obtained by any
other experimental means. Further the waves on the experimental electro-
cardiogram can be shown to correspond to fibrillary movements in the
auricle, by means of synchronous tracings.

The electrocardiogram (11, 15c) shows persistent oscillations of
the string which are irregular in tempo and magnitude; like the venous
pulse variations they occur throughout the cycles but are best noted in
the period of ventricular rest. These oscillations are definitely due
to the continuous activity of the auricle, but no "p" wave marks a def-
inite auricular systole.

(29)
Prognosis

It is impossible to overestimate the importance of auricular fibrillation in diseases of the heart, for more than half of the cases dying of heart failure show this abnormal rhythm at some time or other before death (20). The prognosis in fibrillation, therefore, is of some moment, especially as there are such wide differences in the time lived by patients suffering from this irregularity—some dying within a few hours of the abnormal rhythm, while others live for many years without any marked impairment of their activities.

The dominant factor in the prognosis (8) is the condition of the heart muscle, and it must be the aim in forming a judgment in these to find out as much as can be with regard to its condition. The evidence will naturally group itself into two headings; (1) the clinical evidence, (2) electrocardiographic findings.

The onset of auricular fibrillation is always sudden, and in making a prognostic judgment the symptoms which accompany the onset are extremely important, and give valuable clinical evidence of the state of the functioning power of the heart. If the patient is quite unaware when the change from the normal to the abnormal rhythm takes place it shows, as a rule, that the heart muscle is fairly good, and the outlook is much more favorable than if the change was accompanied by severe distress and evidence of sudden heart failure. This sudden heart failure is the result of the rapid and disorderly action of the heart caused by fibrillation. When there is much distress at the onset the outlook is grave, and indicates a considerable impairment of heart muscle, while occasionally the symptoms are so severe that the patient may only survive a few hours.

(30)
There is one type of auricular fibrillation in which the change from the normal to the fibrillation is not accompanied by marked distress, though the outlook is unsatisfactory, when cardiac failure occurs, and that is the type in which the heart rate remains persistently slow during the fibrillation when not under the influence of digitalis. In this type there is usually marked myocardial damage giving rise to delay in the junctional tissues and therefore partial heart block. The slow rate, with fibrillation, is for the time an advantage for the heart, but when these cases commence with symptoms of cardiac failure the outlook is very bad as they respond so unfavorably to treatment with digitalis or strophanthin.

In order to get a clear idea how this variability in the symptoms is bought about it is necessary to consider the forces at work in the heart.

Mackenzie (47) divided these into "rest force" or force necessary to maintain the circulation when the patient is at rest, and the "reserve force" which is drawn upon when any effort is undertaken, however slight.

<table>
<thead>
<tr>
<th>Rest Force</th>
<th>Reserve Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral Stenosis</td>
<td>Reserve Force</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>Auricular Fibrillation Reserve Force</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>Auricular Fibrillation Reserve Force</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>Auricular Fibrillation Reserve Force</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>Auricular Fibrillation Reserve Force</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>Auricular Fibrillation No Reserve Force</td>
</tr>
</tbody>
</table>
After fibrillation has once set in the outlook depends on a number of factors independent of the abnormal rhythm.

The patients response to effort is important and gives as a rule one of the best indications of the functioning power of the heart (42). Where the response is considerable curtailed, especially when this limitation is rapidly increasing, the outlook is very grave.

Marked cardiac enlargement is another unfavorable factor in prognosis, and indicates a considerable myocardial damage in addition to the fibrillation.

The presence of a considerable degree of valvular disease also marks the outlook less favorable, and at the same time renders the attempt to establish the normal rhythm with quinidine more dangerous, especially in mitral stenosis.

In fibrillation it is extremely difficult to give a definite prognosis until one had had the opportunity of observing the patients response to treatment. This consists, as a rule, in attempting to limit the heart rate with digitalis or strophanthin, or trying to convert the fibrillation back to a normal rhythm with quinidine sulphate.

In giving a prognosis in all cases of auricular fibrillation it must be remembered that ventricular fibrillation is liable to occur at any time with the consequence of sudden death of the patient.

In conclusion (20), auricular fibrillation is of more serious consequence in senile than in rheumatic heart disease. In cases of mitral stenosis the presence of fibrillation makes little difference to the expectation of life. In cases of aortic regurgitation the expectation is better when fibrillation is present than with a normal rhythm. The prog-
nosis is better with aortic regurgitation than with mitral stenosis. With marked cardiac enlargement the outlook is more grave. The expectation of life is longer in females than in males. In the electrocardiographic findings the presence of bizarre Q R S wave is of most significance in the prognosis. The mortality of cardiac disease is doubled and tripled in conditions with auricular fibrillation (45).

There remains a type of patient who fibrillates only at intervals or occasionally, the transient fibrillators (15, 17, 18b, 22, 30, 31). The prognosis of these cases is usually good with the careful watching and restricting of their duties. The may go into persistent fibrillation and their prognosis would then fall under the above conditions.
Treatment

Before the addition of quinidine sulphate to the treatment of heart disease, auricular fibrillation was recognized as a permanent change in the mechanism of the heart beat which was to persist, except in the rare instances of paroxysmal fibrillation, as long as the patient lived. Under ordinary circumstances, the ventricular rate could be controlled more or less successfully with digitalis, but the tumultuous beating of the heart with its distressing subjective sensations are readily induced by exertion(6). This (5) can be explained on the basis of digitalis making fibrillation more fixed by shorting the refractory period and lengthening the conduction. Digitalis (24e) produces a heart block at the auricular ventricular node so that the auricular impulsed do not pass through to the ventricle. Digitalis (32) does not effect the fibrillation in a means of stopping it, it does strengthen the force of the ventricular beat. Haver (49e) reports a case of Stokes Adams Syndrome from digitalis. Jensen (19) reports giving large doses of digitalis with no nausea and vomiting to a patient with auricular fibrillation without heart failure. The dose was one dram to one and a half dram every six hours for three doses and repeated again in forty eight hours. Patients all felt better, only sign was a coupled beat. There was no effect upon the urinary output.

It has been suggested that the restoration of normal sinus rhythm by the use of quinidine sulphate by abolishing the irregular heart action and putting the normal pacemaker in control, would relieve the patients of the distress incident to the irregularity of fibrillation. The evidence so far accumulated shows clearly that auricular fibrillation can
be abolished by quinidine in at least 50 per cent (5, 49b) of cases and while the normal mechanism prevails, the patients are undoubtedly relieved of the discomfort incident to irregular ventricular action and their exercise tolerance is thereby increased. Quinidine sulphate acts on the muscle tissue of the heart. The refractory period is lengthened and the conduction is shortened. Conduction is depressed from the auricle to the ventricle. The advantages of quinidine over digitalis are (5): (1) marked subjective improvement of the patient under normal mechanism, (2) great increase in cardiac reserve, (3) improved pulmonary ventilation, (4) improvement in the oxygen saturation of the venous blood, and on the basis of the coefficient of utilization, improvement in the peripheral systemic blood flow.

When (49e) there is little evidence of heart disease other than arrhythmia, quinidine should always be used. It is safe and effective remedy and normal rhythm may be maintained for years with a remarkable freedom from symptoms. On the other hand when fibrillation has been established for more than six months, and there is a large heart and gross congestive failure or when there is active rheumatic or syphilitic infection, there is more danger in using quinidine and less chance of lasting success. Digitalis is the drug of choice and quinidine should seldom if ever be used.

The drug (49a, 49d) must be employed with caution for toxic symptoms may ensue, besides idiosyncrasies. Toxic symptoms are indications for the interruption of treatment. The earliest of these symptoms are diarrhea, fullness of the head, epigastric pain, nausea with or without vomiting. Later manifestations are palpitation, precordial pain or tenderness, cyanosis and marked increase in ventricular rate.
There is no set rule as to the dosage of the two drugs, digitalis and quinidine. Some like to give massive doses of digitalis till the heart beat is regular and the remaining doses equivalent to keep the patient fully digitalized. Quinidine is usually administered in 0.2 gram doses every two to four hours till the heart beat is regular, then the administration is usually cut down to three times a day with doses large enough to maintain the regular rhythm.

Whiting (45) tried many drugs in fibrillation. Among them are strophanthus (1), apocynum, squill, digitalis and spartein. He found in order of toxicity apocynum, strophanthus, digitalis and squill. In order of potency he determined strophanthus, digitalis, squill, apocynum and spartein. Agassiz (1) used strophanthins with a large amount of toxicity.

Wedd (41) in his experience with veratum viride concludes: (1) "Alcoholic solutions of veratum viride when given to patients suffering from auricular fibrillation produce slowing of both the auricular and ventricular rates of beating and a fall in blood pressure. These auricular changes are independent of the general toxic effects. (2) Veratum viride, as the experimental observations show has, in addition the vagal action already observed, a direct action, similar to quinidine, upon the auricular muscle of a dog. This direct action will, on the circus movement theory of auricular fibrillation tend to enhance the rate of the auricular oscillations. (3) The slowing of the ventricular rate by veratum viride given orally occurs much earlier that that following digitalis bodies. However, as a therapeutic agent veratum is somewhat handicapped by uncertainty and irregularity of action. It may be useful in cases in which simultaneous slowing of the ventricular
rate and lowering of the blood pressure is desired. Dosage of the
drug is 30 to 75 minims.

It is to be remembered that the main object to be achieved when
treating auricular fibrillation is to adjust the rate of the ventricles
so as to produce an adequate circulation without undue ventricular
work. The greatest therapeutic agent is rest—physical, mental, and
cardiac.
Bibliography.

1. Agassiz, G.D.S.
Observations upon the Effects of Strophanthin in Cases of Auricular Fibrillation.
Heart—1912—3—353.

2. Anderson, J.P.
Auricular Fibrillation Associated with Hyperthyroidism.

3. Andrus, E.C. and Carter, E.P.
The Refractory Period of the Normal Beating Dogs Auricle: With a
note on the occurrence of Auricular Fibrillation following a

4. Barber, E.W. and Middleton, R.P.

5. Barker, P.S.
Auricular Fibrillation.
American Heart Journal—1926—2—72.

6. Blumgart, H.
The Reaction to Exercise of the Heart Affected by Auricular Fibrillation.
Heart—1924—11—49.

7. Breachman, D.S.
Auricular Fibrillation; Some Observations and Deductions from a
Series of Four Hundred Cases. Lancet—1921—1—374.

8. Coffen, T.H.
The Favorable Prognosis of Auricular Fibrillation.

9. Cookson, H.
A) Auricular Fibrillation in Children.
Lancet—1929—2—1139
B) The Etiology and Prognosis of Auricular Fibrillation.
Quarterly Jour. of Med.—1930—25—309.

10. Dock, W. and Levine, S.A.
The Occurrence of Regular Ventricular Rhythm with a Rate of over
Fifty in cases of Auricular Fibrillation.

11. Drury, A.M. and Iliescu, G.C.
Observations upon Flutter and Fibrillation. The Electrocardiograms
of Clinical Fibrillation.
Heart—1921—8—171.

12. Fowler, W.W. and Baldridge, G.W.
Auricular Fibrillation as the only Manifestation of Heart Disease.
Amer. Heart Jour.,—1920—5—185.
13. Fox, G.H.
The Clinical Significance of Transitory Delirium Cordis.
Amer. Jour. of Med. Science--1910--140--615

14. Frothingham, C.
The Auricles in Cases of Auricular Fibrillation.
Arch. of Int. Med.,--1925--36--437

15. Garrey, W.H.
A) The Suppression of Cardiac Fibrillation by Section.

B) The Nature of Fibrillary Contraction of the Heart--Its relation
to Tissue Mass and Form.
Amer. Jour. of Physiol.--1914--33--397.

C) Auricular Fibrillation.
Physiological Reviews--1924--4--215.

16. Hay, J. and Jones, H.W.
Trauma as Cause of Auricular Fibrillation. Its Medical-Legal Significance

17. Heard, J.D. and Caldwell, A.H.
Transient Auricular Fibrillation.

18. Hewlett, A.W.
A) Clinical Observations on Absolutely Irregular Hearts.

B) Auricular Fibrillation Associated with Auricular Extrasystoles.
Heart--1910--2--107.

19. Jensen, J.
Massive Doses of Digitalis in Auricular Fibrillation without Heart Failure.
Lancet--1924--1--747.

20. Jones, H.W.
Some Points in the Prognosis of Auricular Fibrillation.
Lancet--1926--2--640.

21. Kilgore, E.S.
Time Relations of Heart Beats. Respiratory Variations of Heart Rate in
the Presence of Auricular Fibrillation.
Heart--1919--7--47.

22. Levy, R.L.
Auricular Fibrillation with Regular Ventricular Rhythm, and Rate over
Sixty.
Arch. of Int. Med.,--1926--38--116

(39)
24. Lewis, T.
   B) Auricular Fibrillation and Its Relationship to Clinical Irregularity
      of the Heart.
      Heart—1909—1—306.
   C) Observations upon Disorders of the Heart's Action.
      Heart—1912—3—279
   D) Observations upon Flutter and Fibrillation. The Nature of Auricular
      Fibrillation as It Occurs in Patients.
      Heart—1921—8—193.

25. Lewis, T., Drury, A.N. and Iliescu, G.C.
   A Demonstration of Circum Movement in Clinical Fibrillation of the Auricles
   Heart—1921—9—361

26. Legs, D.G. and Russel, H.B.
   A Case of Persistent Auricular Fibrillation in a Child of Six.

27. Mackenzie, J.
   A) The Inception of the Rhythm of the Heart by the Ventricles as the
      Cause of Continuous Irregularity of the Heart.
      Brit. Med. Jour.—1904—1—529
   B) The Interpretation of the Pulsations in the Jugular Veins.

28. McWilliams, J.A.
   Fibrillar Contractions of the Heart.
   Jour. of Physiol.—1887—3—296.

29. Ogden, R.T.
   Amer. Jour. of Dis. of Children—1925—29—767

30. Parkinson, A. and Campbell, M.
   Paroxysmal Auricular Fibrillation. A Record of 200 Patients.

31. Patterson, R.V.
   Transient and Recurrent Auricular Fibrillation.

32. Resnik, W.H.
   Transient Auricular Fibrillation Following Digitalis Therapy with
   Observations upon the Action of Atropine.
   Jour. of Clinical Investigation—1924—1—181.
33. Rosnik, W.H. and Scott, W.I.
Auricular Fibrillation Report of Case in Child Age 4 Years.
Amer. Jour. of Disease of Children--1926--65--1611.

34. Eoes, J.
Auricular Fibrillation in Domestic Animals.
Heart--1924--11--1

35. Schwartz, S.F. and Weiss, M.M.
Auricular Fibrillation in Children. Its Relation to Rheumatic Heart Dis.

36. Smith, F.M.
Clinical Observations on Paroxysmal Auricular Fibrillation and Flutter.

37. Stewart, H.J., Crawford, J.H., & Hastings, A.B.
The Effect of Tachycardia on the Blood Flow in Dogs. The Effect of
Rapid Irregular Rhythm as Seen in Auricular Fibrillation.

38. Stewart, H.J., Crawford, J.H., and Gilchrist, A.R.,
Studies on the Effect of Cardiac Irregularity on the Circulation. The
Relation of Pulse Deficit to Rate of Blood Flow in Dogs Subject to
Artificial Auricular Fibrillation and to Regular Tachycardia.

39. Stewart, H.J. and Gilchrist, A.R.
Studies on the Effect of Cardiac Irregularity on the Circulation. The
Estimation of Cardiac Output in Dogs Subject to Artificial Auricular
Fibrillation.
Jour. of Clin. Invest.--1928--5--335.

40. Swan, J.M.
A Case of Auricular Administration Occurring During the Administration
of Thyroid Substance. Annals.

41. Wedd, A.M.
Veratrum Viride in Auricular Fibrillation.
Heart--1926--12--307

42. White, P.D.
Prognosis in Heart Disease in Relation to Auricular Fibrillation and Alteration of the Pulse.

43. Whiting, A.J.
On the Comparative Value of the Digitalis Series of Remedies in the
Heart Failure of Auricular Fibrillation and Changes in the Clinical Features of Mitral Stenosis after Fibrillation of the Auricle.
Proc. of the Royal Society of Medicine--Sect. of Therapy and Pharm.
London--1918--11--1
44. Wiggers, C.J.
Studies on the Pathological Physiology of the Heart. Intra-auricular
Intra-ventricular and aortic Pressure Curves and Auricular Fibrillation.
Arch. of Int. Medicine—1915—15—77

45. Willius, F.A.
Auricular Fibrillation and life Expectancy.

46. White, P.D.
Auricular Fibrillation and Auricular Flutter
Heart Diseases—Pg. 642.

47. MacKenzie, Sir James
Auricular Fibrillation.

48. Guy's Hospital Report—1929—9—117

49. Tice.
A) Oille, J.A. and Jamieson, Ross A.
Quinidine Sulphate in Auricular Fibrillation.

B) Maynard, E.P.
Five Years Experience in the Treatment of Chronic Auricular Fibrillation
with Quinidine Sulphate.
Amer. Jour. of Med. Science—1928—175—55

C) Hewer, T.F.R.
Stokes Adams Syndrome produced by Digitalis in a case of Auricular
Fibrillation.
The Bristol Medico-Chirurgical Jour.—1930—47—135.

D) Hurxthal, L.M.
Auricular Fibrillation in Patients with Goitre.

E) Porter, William B.
The Rational use of Quinidine in the Treatment of Auricular Fibrillation
Southern Medicine and Surgery.—1930—92—321