THE DETAILS OF THE PHOTOGRAPHICALLY RECORDED VENOUS PULSE IN AURICULAR FIBRILLATION.

BY WALTER L. NILES, M.D., AND CARL J. WIGGERS, M.D.

(From the Second Medical Division of Bellevue Hospital and the Physiological Laboratory of Cornell University Medical College, New York City.)

PLATES 1 TO 4.

(Received for publication, September 15, 1916.)

INTRODUCTION.

The condition of auricular fibrillation is easily diagnosed by the aid of arterial and jugular tracings or by electrocardiograms, and in most cases it can be recognized by careful physical examination alone. It is not possible, however, to interpret all the waves found in the venous records. Generally, these tracings may be placed in one of two classes; (a) those in which prominent systolic waves, differing in contour from the small, impact waves of the normal phlebogram, predominate or occur alone, and (b) those in which large or small diastolic waves recur occasionally, in groups, or in a continued series averaging 250 to 500 per minute.

The prominent systolic waves, designated as ventricular waves, have generally been attributed to an associated tricuspid regurgitation. Lewis¹ states: "The ventricular form of venous pulse is a term which expresses the only fixed quality manifested by graphic records taken from the jugular veins in these cases. . . . The older conception, that the prominence of the venous pulsation is an index of the degree of tricuspid reflux, is not without a definite foundation." The inference follows that tricuspid regurgitation is a frequent accompaniment of this form of arrhythmia. It is of interest to reexamine to what extent this is true.

The diastolic waves are often so recurrent and numerous that it is impossible to account for them under the assumption that the auricle is in a dilated and finely fibrillating state, the condition to which the term auricular fibrillation was first applied. Many electrocardiograms also show definite variations resembling P waves or diastolic wave groups which are interspersed between easily recog-

nized ventricular complexes. Consequently it has been suggested (Hewlett and Wilson\textsuperscript{2}) that in some cases a condition of coarse fibrillation obtains, or that the condition is associated with a state of rapid coordinated contractions designated as auricular tachyrythmia, or flutter (Jolly and Ritchie\textsuperscript{3}). Concerning the diagnostic differentiation between flutter and fibrillation, Ritchie states:

"If polygraph tracings reveal a rhythmic series of large auricular waves in the jugular veins at times when the ventricles are in diastole, the condition is clearly flutter and not fibrillation. On the other hand, if the waves are small, irregular in rhythm, and very rapid, and if at the same time there is little uniformity in the grouping of arterial pulse beats, the auricles are either in fibrillation or in the form of activity representing simultaneous flutter and fibrillation."

Hewlett and Wilson,\textsuperscript{2} in 1915, reviewed the experimental relation between fine and coarse fibrillation and comment upon the rarity with which large recurrent diastolic waves are found in venous tracings. They conclude that as a rule clinical fibrillation is of the fine type, but present a case, fully studied by photographic registration, in which distinct diastolic waves, which have no relation to electrocardiogram wavelets, were present. This they interpreted as a condition of coarse fibrillation.

A more comprehensive attempt to establish the extent to which coarse and fine fibrillation occur clinically and to what degree these in turn are associated with flutter is of apparent interest. For these reasons an objective analysis of the jugular and other records taken by photographically recording capsules, was undertaken, and their interpretation harmonized with the case histories and other physical signs present at the time.

\textit{Method of Procedure.}

Records of the venous pulse were taken simultaneously with the subclavian and radial pulses and with apex beats or electrocardiograms.\textsuperscript{4} The superiority of capsules photographically recording the dynamic changes by reflecting beams of light need not be reviewed. The photographic records were obtained by mounting the Frank segment capsules, illuminating lamp, and photokymograph on a

\begin{itemize}
  \item \textsuperscript{2} Hewlett, A. W., and Wilson, F. N., \textit{Arch. Int. Med.}, 1915, xv, 786.
  \item \textsuperscript{3} Jolly and Ritchie, \textit{Auricular Flutter}, New York, 1914, 125.
  \item \textsuperscript{4} We are indebted to Dr. Alexander Lambert for placing the electrocardiograph in Bellevue Hospital at our service and to Dr. Josephine B. Neal for valuable technical assistance in securing the clinical electrocardiograms.
\end{itemize}
table which could be wheeled to the bedside (Wiggers\(^8\)). When simultaneous electrocardiograms were taken the table was also aligned with the projection system of a Cambridge model of Einthoven's string galvanometer. The movements of the segment capsule mirrors were thus photographed simultaneously with the shadow of the galvanometer string. Tracings of the jugular and radial pulses were also taken by a Jacquet polygraph for comparison with the photographic records.

*Details of the Normal Photographic Curves.*

Although the details of the photographic venous pulse have often been described (Edens and Wartensleben,\(^6\) Ohm,\(^7\) Van Zwaluwenburg and Agnew,\(^8\) and Wiggers\(^9\)), our analysis of the photographic tracings in auricular fibrillation will be facilitated by a brief reconsideration of its chief features, especially as related to the photographic apex curves.

The two tracings shown in Fig. 1 give the essential characteristics of normal photographic records from the apex region and supraclavicular fossa. Experience has shown that there is nothing distinctive about the contour of the apex curve, which may in fact be positive or negative according to the position of the patient and placement of the tambour (Wiggers\(^10\)). A detailed description is therefore superfluous. There is, however, one feature of these tracings which makes them valuable, for unless the chest wall is too thick or emphysematous lung overlies the heart, they show clear indications of the heart sounds (S\(_1\) and S\(_2\)) which enables a positive determination of the beginning and end of ventricular systole. The photographic venous pulse always shows three essential waves; to these a fourth wave is


\(^7\) Ohm, R., *Venepuls- und Herzschallregistrierung als Grundlage für die Beurteilung der Mechanischen Arbeitsleistung des Herzens nach eigenen Methoden*, Berlin, 1914.


sometimes added when the cardiac cycle is long. The waves differ from polygraph curves in that each wave has a distinctive contour and that the relative prominence of the waves is different.

Since the records taken from cases of auricular fibrillation have so little in common with normal curves, it was found desirable to discontinue the commonly accepted terminology (a, c, v waves) introduced by Mackenzie. All records may readily be divided into systolic and diastolic portions. This is usually accomplished by the use of the two sound vibrations previously referred to in the apex tracing. Occasionally these vibrations are transmitted to the neck and superimposed on the venous tracings as well (Fig. 1, S'). The waves are best designated as presystolic, systolic, and diastolic. In the normal cycle each portion has, as a rule, a single wave. The presystolic wave (P) is associated with auricular systole. The systolic wave (S), occurring a considerable interval after the first sound, is synchronous with the subclavian rise of pressure and is, therefore, due to an arterial impact. The third wave (D1), commonly called v wave, is entirely diastolic in time, and is probably due to the cessation of stasis, as the tricuspid valve opens at its summit. When the cardiac cycle is long, a second diastolic wave (D2) is often found. Its significance cannot be positively given at present, but its presence in long cycles should be kept in mind in connection with fibrillation waves.

The following are average durations found for these waves:

<table>
<thead>
<tr>
<th>Wave Type</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presystolic wave (P), or a wave</td>
<td>0.09-0.10</td>
</tr>
<tr>
<td>Systolic wave (S), or c wave</td>
<td>0.28-0.32</td>
</tr>
<tr>
<td>First diastolic wave (D1), or v wave</td>
<td>0.15-0.24</td>
</tr>
<tr>
<td>Second diastolic wave (D2), or h wave</td>
<td>0.21-0.22</td>
</tr>
</tbody>
</table>

**Types of Waves in Auricular Fibrillation.**

*Method of Analysis.*—The photographic tracings taken from cases of auricular fibrillation were analyzed in the same manner as normal records. The beginning and end of each ventricular systole must first be established. In many instances the first and second sound vibrations of the jugular or apex tracing satisfactorily determine these places; in others, where irregular cardiac action occurs, only a single sound is recorded in the jugular or apex tracing. Since
there is nothing distinctive about heart sound vibrations, it is occasionally impossible in auricular fibrillation to determine whether the vibrations represent the end of a premature but ineffective ventricular systole, a third sound, or a short diastolic murmur. The addition of an electrocardiograph record serves to elucidate these cases since the presence or absence of an R wave at these times differentiates them. Combined simultaneous records of the jugular pulse, apex beat, and electrocardiograms were therefore made in most cases, but as the electrocardiograms taken on bromide paper are not sufficiently clear to reproduce well, they have been omitted in the reproductions.

Types of Waves Occurring during Ventricular Systole.

In experimental auricular fibrillation many ventricular systoles occur that are too weak to open the semilunar valves, and hence fail to elevate aortic pressure, or elevate it so slightly that a pulse wave is not propagated to the more peripheral arteries (Wiggers\textsuperscript{11}). The frequent occurrence of a pulse deficit is evidence that this also occurs in clinical fibrillation. The ventricular systoles may, therefore, be divided into effective and ineffective groups. Effective systoles may be recognized by the presence of a corresponding pulse wave in the subclavian or radial tracings. Ineffective systoles are often indicated by a single sound in the apex curve, but more certainly by the presence of a ventricular complex in the electrocardiogram without a corresponding arterial wave.

Types of Waves during Effective Systoles.—The different types of waves occurring during effective systoles are illustrated by the actual records shown in Figs. 2 to 9, but for a description, reference may be made to the transferred waves shown in the curves of Text-fig. 1.

The systolic waves may resemble typical impact waves such as are found in normal pulses. As in Type I, the rise is usually preceded by a short isometric period (A–B), during which vibrations of the first sound are discernible. The wave terminates in the vibrations of the second sound at C. The impact wave may, as shown in Type II, rise and fall sharply, which is also a frequent nor-

\textsuperscript{11} Wiggers, Arch. Int. Med., 1915, xv, 77.
TEXT-FIG. 1. I to VI represent different types of systolic waves occurring in auricular fibrillation. VII to X represent diastolic waves. Transcribed from the original curves and reduced about one-third.

Type I. Normal and intensified impact wave, often the only characteristic feature, and sometimes interpreted as the ventricular type of the venous pulse.

Type II. Peaked impact wave followed by systolic drop. It occurs when the tambour is pressed lightly and intravenous pressure is low during systole.

Type III. Intra-auricular type of venous pulse. It most nearly resembles the intra-auricular pressure curve and is only recorded when the arterial impact is feeble or absent.

Type IV. Double systolic waves both completed during systole. Probably due to tug or position change of contracting ventricle.

Type V. Systolic impact and stasis wave. Double systolic wave differing from Type IV in that the summit of the second wave terminated at the end of systole.

Type VI. Regurgitation wave due to tricuspid insufficiency.
normal variation (Fig. 1) when the tambour is very lightly applied. A
direct impact may be almost or completely absent, as in Type III,
in which a marked fall similar to that observed within the auricle,
is the predominant feature. This is followed by a systolic rise (C–D),
which also resembles the auricular pressure curve. This probably
as nearly corresponds to a pure venous curve as it is possible to ob-
tain and probably occurs only when the arterial waves are not
vigorous enough to superimpose an arterial impact on the tambour
record. A fourth type of wave is shown in Type IV where an impact
oscillation (A–B–C) is followed by a second systolic oscillation (C–D–E).
Its occurrence before the end of systole cannot be questioned, as the
vibrations of the second sound occur after it terminates. Its signi-
ficance cannot be fully explained. The possibilities that it may
represent a tricuspid regurgitation late in systole, or an auricular
stasis, do not entirely accord with the fact that it falls before dias-
tole begins. The possible explanations to be considered are: (a)
a ventricular position change which modifies auricular pressure and
hence the jugular curve; and (b) a tug of the contracting ventricle
on the superior vena cava. This is made more probable by the fact
that waves of this type were particularly marked in two of our cases,
both of which had definite signs of adhesive mediastino-pericarditis,
and pericardial adhesions may well result in an unusual traction
upon the superior attachment of the sac. A second type of double
systolic wave is shown as Type V. This differs from Type IV in
that the summit of the second wave terminates at the beginning of
diastole.

What may be designated as regurgitation waves were also found.
These waves have a distinctive contour—entirely different from
the rounded ventricular waves described by Mackenzie and others
in polygraph curves. These waves (Type VI) resemble waves found
within the auricle when a relative insufficiency occurs during fibril-
lation of the auricles. Early in systole they rise sharply (A–B),
then gradually rise further or remain sustained until the onset of
diastole (C). Particularly characteristic are small undulations or
vibrations superimposed on their summits which represent murmur
vibrations transmitted to the supraclavicular region and generally
audible there.
Waves Occurring during Ineffective Systoles.—During ineffective systoles the venous pulse may show nothing except a gradual stasis (Type VII) terminating at the end of systole; or the isometric period may be followed by a double vibration similar to, but smaller than those sometimes found in effective systoles (Type VIII). These waves may occasionally be shorter and more prominent (Type IX), and evidence of a small regurgitant wave is sometimes found (Type X).

Diastolic Waves.

The diastolic waves occurring during auricular fibrillation may be classed as large and small. The large oscillations are frequently found just before the systolic wave. In contour and duration they cannot be distinguished from normal presystolic (a) waves. Similar waves are also found during mid-diastole as shown at D in Fig. 9, and when this event is long they may occur in numbers. They apparently correspond to the waves found in Hewlett and Wilson's reported case. Instead of being a rare event, however, we find that they are a very common occurrence in photographically recorded venous pulses from cases of auricular fibrillation.

Three possible explanations are suggested for these waves, but it is not possible to decide between them on the basis of clinical experiment alone. It is conceivable that they may be produced by a coordinated mechanical contraction of a considerable portion of the auricular musculature which is sufficient to inaugurate a pressure wave within the auricle. According to this view, we must assume that the incoordinated fractionate contractions of individual units of cardiac tissue happen to be so related that from time to time their interference produces an actual shortening of a certain section of the auricle. If this were the case we would expect simultaneous waves in the electrocardiogram which resemble normal P waves. Such waves are occasionally to be found, but the synchronism is by no means frequent.

It is possible that these waves indicate incoordinate coarse fibrillations of the auricle, as suggested by Hewlett and Wilson, since

\[12\] The term “fractionate contraction” has been defined by one of us as the interval that any unit of auricular tissue continues to shorten \(\text{(Am. J. Physiol., 1916, xl, 222)}\).
they are irregular in size and bear no relation to smaller variations of the electrocardiogram. If this interpretation is true, then coarse fibrillations are far more common clinically than is generally believed.

Inefficient contractions of the ventricle might produce such waves. The possibility has been shown in animal experiments by one of us, and indeed was at the same time considered the most probable cause of these waves. Though virtually diastolic waves, they would really be due to unrecognized systoles. If this were true the electrocardiograms would show distinct ventricular complexes synchronous with them. A careful study of our records shows, however, that such inefficient systoles are rarely present in clinical fibrillation, and that very few such waves can be accounted for upon this basis.

The resemblance of the presystolic waves found in fibrillation to the second diastolic wave of the normal venous pulse has led Lewis and others to attribute it, as has been done in the normal pulse, to a floating together of the tricuspid valves. Neither in the normal or fibrillating pulses does this interpretation appeal to us. The facts (a) that the wave is not associated with the third sound in cases where the latter has been recorded (Einthoven, Eyster), (b) that the valves do not come together sharply at the beginning of diastole, but apparently gradually float together throughout diastole (Dean), (c) that, on dynamic principles, their closure is probably caused by a pressure difference rather than a wave of pressure within the auricle, all lead one to question such an origin in normal pulses. The fact that several such waves occur during long diastolic intervals in fibrillation would necessitate the assumption that the valves move into their position of closure several times, while an isolated presystolic wave occurring at the end of a long diastole would necessitate the assumption that in these beats the closure was, for some unaccountable reason, greatly delayed. That these waves are similar to the late diastolic waves normally found in long cycles cannot be denied; that either owes its origin to a valve movement seems improbable.

13 Lewis, Mechanism of the Heart Beat, London, 1911, 204.
14 Einthoven, W., Arch. ges. Physiol., 1907, cxx, 31.
They may be due to a position change of the relaxing or filling ventricle which exerts a pressure traction upon the auricles or large veins or perhaps causes a variation of intrathoracic pressure which might modify the influx and efflux of venous blood in the extrathoracic veins.

Records of Representative Cases.

As it is manifestly impossible to reproduce the many hundred yards of records taken from a total of 25 cases in the Second Medical Division of Bellevue Hospital, a description of a few short segments from representative cases must suffice for presentation. In this selection have been included (1) cases in which systolic waves predominate including (a) slow rhythms and (b) rapid rhythms, and (2) cases in which diastolic waves predominate (slow rhythms).

*Case H, 19.*—Male, aged 51 years. Rheumatic heart; no murmurs; compensating. Chronic nephritis. Systolic blood pressure, 165. (Fig. 2.)

The photographic record shows that systolic waves are predominant at the jugular region. Numerous attempts to obtain other venous waves of considerable size proved futile. Each systolic wave is nothing more than a transmitted arterial pulse. This is the characteristic record found in cases of auricular fibrillation accompanied by strong cardiac action and elevated blood pressure. The details of the central arterial pulse are readily discerned: 1–2, the initial vibrations; 3, the primary peak; and 4, the incisura with after vibrations. Their irregularity is the only conspicuous difference from normal arterial curves.

If we glance at the supraclavicular record taken by a Jacquet polygraph (Fig. 3) by way of comparison, it shows a series of systolic waves in every way typical of the ventricular type of venous pulse and referred to tricuspid regurgitation. It is clear that the deforming action of tambour levers may distort a perfectly normal arterial impact curve into the semblance of a regurgitation wave.

During the longer diastoles of Fig. 2 small waves occur (d–d) having a period of 0.076 to 0.128 of a second. Similar corresponding wavelets were also found in the apex curves.
Case G, 6 and H, 8.—Male, aged 44 years. Sclerotic heart; no murmurs; non-compensating. Chronic nephritis. Blood pressure, 220–150. (Fig. 4.)

This case showed in succession a variety of systolic wave types and in addition was characterized by numerous large diastolic waves during a certain period of his hospital stay. The cycles in this segment of record are numbered from 3 to 7 to facilitate description. Wave 3 represents a normal impact wave. The isometric period (a–b) is filled with fused preliminary vibrations after which the impact (b–c–d) occurs. Cycle 7 resembles this wave, but the systolic decline is sharper, placing it under Type II. Cycle 4 shows a bifurcated systolic wave (Type IV) which, to judge from the smaller amplitude of the corresponding radial beat, was produced by a less vigorous systole. Such smooth double vibrations were often found to accompany weak systoles in animal experiments previously reported by one of us. Cycle 5 would probably have shown a similar double wave had not a weak systole occurred at 6 and cut short the second vibration. After the first elevation of Cycle 6, the curve continues to rise owing to the stasis of blood in the auricle which had not been allowed to empty because of the practical absence of preceding diastole. This double vibration due to an ineffective systole corresponds to Type IX.

During diastole a series of large waves (D₁, D₂, and D₃) occurs. While the third oscillation, D₃, is at its crest, Systole 4 occurs. Without a careful analysis of the interval of systole, these systolic waves following might be attributed to the same cause as the diastolic waves D₁, D₂, and D₃ and held to indicate a rhythmic coarse contraction of the auricle.

Attention should be called to the presystolic wave, P, which precedes Systole 7. In time and general appearance it resembles a normal presystolic wave, or a wave due to a coordinated auricular activity. These waves occur so frequently that to say presystolic waves are absent in auricular fibrillation compels one to ignore many such waves not to be distinguished from normal a waves.

To summarize, this record shows in rapid sequence a variety of systolic waves when ventricular activity is irregular in force, and the presence of large diastolic waves having a rate of 5.5 per second, or 330 per minute.
Case H, 18.—Male, aged 51 years. Sclerotic heart; rate 140-150 per minute; relative mitral insufficiency. General anasarca. Deep, somewhat dyspneic breathing. (Fig. 5.)

When the heart rate is rapid the venous pulse is composed almost entirely of systolic waves. When, in addition, breathing is deep, they are not only superimposed upon the respiratory variations, but their contour changes with inspiration and expiration, as in this record. During inspiration the individual waves are small and scarcely recognizable (e.g., 2, 3, and 9). During expiration (Waves 1, 5, and 6) the waves are larger and have a distinctive contour which places them under Type II. The correspondence of the R peaks with each radial pulsation shows that each systole, even at that rapid rate, was effective.

Case H, 17.—Male, aged 35 years. Dilated, rheumatic heart; mitral regurgitation (Fig. 6); left-sided hypertrophy shown by electrocardiogram. A harsh systolic murmur was heard over the apex of the heart and in the right supraclavicular fossa.

The venous tracing shows a series of prominent regurgitant waves, the characteristic features of which are a rapid rise during the onset of systole ending in a sustained ascending or descending plateau upon which a series of vibrations, probably resulting from the concurring murmur, are superimposed. In contour they correspond to intra-auricular waves found in experimental animals which have tricuspid regurgitation. The onset of systole as shown by the R wave of the accompanying electrocardiogram (not reproduced) is marked at S in each case. It is apparent that their exact contour depends to some extent upon the state of venous pressure at the onset of systole. Beat I represents a typical unaltered regurgitation curve. In Waves IV and V the onset of systole occurred at the summit of a diastolic stasis rise (S) and consequently the main rise of the wave occurred before the onset of systole. This is a frequent occurrence when the heart rate is rapid.

Case H, 11.—Male, aged 40 years. Rheumatic heart; very large. Systolic murmur over aortic area transmitted to the neck; diastolic murmur over sternum transmitted to the apex; systolic murmur at the apex transmitted to the axilla. (Fig. 7).
The record of Fig. 7 was taken on February 19, 1915. The pulse rate averaged 150 per minute. The venous pulse is chiefly made up of waves indicating a dilated right heart. Two types are prominent. Those marked Type V show after a primary systole wave, which is probably an impact, a considerable stasis rise. Several waves, owing to weaker systoles, show little of the first impact wave and consist essentially of the early systolic fall and subsequent rise. These are the waves that resemble the intra-auricular pressure curves (Type III). Between these waves occur typical regurgitant waves, which rise rapidly and remain sustained with numerous vibrations superimposed throughout systole (Type VI).

Another record shown as Fig. 8 was taken on March 1, 1915, after rest and digitalis treatment had slowed the pulse to an average of 86 per minute. This lengthened the diastolic interval which is consequently filled with many diastolic waves (d) varying considerably in amplitude and period. There are no corresponding waves found in the electrocardiogram record. Evidence of regurgitation waves is practically absent.

Case II, 1.—Male, aged 38 years. Infective endocarditis and mediastino-pericarditis; double mitral lesion; coarse systolic thrill over precordium (Fig. 9). Upper curve, supraclavicular venous pulse; lower curve, apex tracing.

During systole two distinct waves occur in the apex tracing, one (1–2–3) during the early half of systole; the second (3–4–5) during the latter half. Superimposed upon these waves occurs a series of finer waves extending throughout systole, as can be seen from the irregular width of the ascending line (3–4–5). They represent the first sound vibrations together with the systolic murmur. Systole ends with the few vibrations at S2.

Each systole produces in the jugular pulse two waves, 1–2–3 and 3–4–5, which, allowing for delay, correspond exactly to the apex waves. The probability that this double systolic wave (Type IV) is due to a position change or tug of the ventricle has been previously discussed.

Early in diastole of each cycle occurs a wave D similar to that sometimes found in the normal curve. When the cycles are long this wave may precede systole and simulate a presystolic wave, as in the third cycle shown.
### TABLE I.
Relative Frequency of Different Types of Waves.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal impacts</td>
<td>Type I and II</td>
</tr>
<tr>
<td>G, 1</td>
<td>32 Rheumatic heart; mitral stenosis.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 2</td>
<td>21 Rheumatic heart; mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 5</td>
<td>62 Sclerotic heart; relative mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 7</td>
<td>54 Rheumatic heart; double mitral lesion.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 9</td>
<td>26 Rheumatic heart; double mitral lesion.</td>
<td>Many.</td>
<td>Few.</td>
</tr>
<tr>
<td>G, 10</td>
<td>42 Rheumatic heart; mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 11</td>
<td>38 Rheumatic heart; mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>G, 14</td>
<td>26 Rheumatic heart; mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>H, 1</td>
<td>38 Infective endocarditis and mediastino-pericarditis; double mitral lesion.</td>
<td>Many.</td>
<td>Many.</td>
</tr>
<tr>
<td>H, 5</td>
<td>48 Postpneumonic mediastino-pericarditis; dilated heart; no murmurs.</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>H, 7</td>
<td>61 Sclerotic heart; dilated heart; no murmurs.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>H, 9</td>
<td>36 Rheumatic heart; mitral regurgitation.</td>
<td>&quot;</td>
<td></td>
</tr>
<tr>
<td>H, 10</td>
<td>30 <em>Streptococcus viridans</em> polyserositis; dilated heart; relative mitral regurgitation.</td>
<td>&quot;</td>
<td></td>
</tr>
</tbody>
</table>
WALTER L. NILES AND CARL J. WIGGERS

TABLE I—Continued.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal impacts.</td>
<td>Intra-auricular</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Types I and II</td>
<td>curves, feebly</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ventricular</td>
</tr>
<tr>
<td>H, 12</td>
<td>48 Rheumatic heart; double mitral lesion.</td>
<td>All.</td>
<td>Few.</td>
</tr>
<tr>
<td>H, 13</td>
<td>37 Rheumatic heart; mitral regurgitation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H, 16</td>
<td>24 Rheumatic heart; double mitral lesion; Graves' disease.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H, 17</td>
<td>35 Rheumatic heart; mitral regurgitation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H, 18</td>
<td>51 Sclerotic heart; relative mitral regurgitation.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>H, 19</td>
<td>51 Rheumatic heart; no murmurs; chronic nephritis.</td>
<td>Many.</td>
<td></td>
</tr>
<tr>
<td>H, 20</td>
<td>38 Rheumatic heart; double mitral lesion.</td>
<td>All.</td>
<td></td>
</tr>
<tr>
<td>H, 21</td>
<td>55 Sclerotic heart; relative mitral regurgitation.</td>
<td>Few.</td>
<td></td>
</tr>
<tr>
<td>H, 23</td>
<td>34 Rheumatic heart; double mitral lesion.</td>
<td></td>
<td>Many.</td>
</tr>
</tbody>
</table>

In the apex curve as well as the jugular curve occur small wavelets (d-d) whose period averages 0.13 to 1.1 seconds. Their rate averages about 460 per minute.

This case, illustrating a fibrillating auricle with slow ventricular
rhythm, shows in the jugular a typical double systolic wave, a normal diastolic variation, and, in addition, small periodic diastolic waves.

The electrocardiogram showed similar diastolic waves of considerable amplitude, which did not correspond definitely with the waves in the phlebogram.

The relative frequency with which the various types of waves occurred in our cases is shown in Table I.

By far the most frequent form of systolic wave is the normal impact wave (Types I and II), which was present in all but three cases. They often constitute the most conspicuous, and in certain cases (twelve of our series) the only characteristic feature of the venous pulse curves. They may resemble each other from one cycle to another, which is usually the case when the heart is slow and comparatively regular, or they may show rhythmical variations in size with respiration, especially when the heart is rapid yet fairly regular, or when moderate dyspnea is present. They may vary from beat to beat both in contour and amplitude, these being determined, as has been previously analyzed by one of us, by (a) the vigor of ventricular systole, (b) the height of aortic pressure at the onset of systole, (c) the duration of the previous diastolic interval, and (d) the pressure of the recording tambour.

When the cardiac contraction is particularly vigorous the systolic wave may resemble a typical central arterial pulse terminating in a deep incisure. The important observation was made that when this occurs the venous pulse recorded by the polygraph is unusually distorted and often resembles the ventricular waves of Mackenzie, which are supposed to indicate regurgitation. So called ventricular waves of polygraph curves may thus be produced by perfectly normal arterial impacts and are not indicative of tricuspid regurgitation; on the contrary, they may signify exceptional heart action.

Next in frequency is the stasis curve (Type V), being found in eight cases. In only one instance were they the only curves present, and, as might be anticipated, they were most frequent when the heart was incompetent and venous stasis was evident in the pulmonary or portal circulations.

The type of wave which we attribute to a systolic tug or position change of the heart (Type IV) was present in four cases. In two of
these cases well marked signs of pericardial adhesions were noted, while in another case though no physical signs of the condition were recorded, the rheumatic origin of the heart lesions makes its presence not improbable. In the fourth instance pericardial adhesions were not suspected.

Intra-auricular pressure waves (Type III) were present in only two cases. Regurgitation waves (Type VI) were present in five cases, and in one of these they disappeared during the course of hospital observation.

This observation discredits the idea that tricuspid regurgitation is a frequent accompaniment of auricular fibrillation.

**SUMMARY.**

While auricular fibrillation is easily recognized by arterial and jugular tracings or by electrocardiograms, it is not possible to interpret all the waves found in these records.

The venous tracings may in general be placed in one of two classes: (a) those in which prominent systolic waves predominate or occur alone; and (b) those in which large or small diastolic waves occur occasionally, in groups, or in a continued series averaging 250 to 500 per minute. The prominent systolic waves have generally been attributed to tricuspid regurgitation, the inference being that tricuspid regurgitation is a common state in auricular fibrillation. The diastolic waves are so numerous and so many electrocardiograms show diastolic waves that it is impossible to account for them on the assumption that the auricle is in a dilated and finely fibrillating state. It has been suggested that in these instances a condition of coarse fibrillation, which is closely allied to auricular flutter, obtains. The systolic and diastolic waves of the venous pulse of twenty-five clinical cases of auricular fibrillation, recorded by photographic methods, were studied.

Six types of systolic waves (Text-Fig. 1) were found: (1) an intensified impact wave, the most common and often the only characteristic feature, indicating vigorous ventricular action; (2) a peaked impact followed by a rapid systolic drop due to light pressure of the tambour; (3) the intra-auricular type of systolic variation, so called from its resemblance to intra-auricular pressure
curves found in animals, occurring in clinical cases only when ventricular systole is weak; (4) double systolic waves, attributed to a systolic tug of the ventricle on the auricles and large veins; (5) a systolic impact followed by a stasis wave, present when intravenous pressure is high; (6) a regurgitation wave composed of a steep rise continued into a systolic plateau with murmur vibrations superimposed.

Our study showed (1) that tricuspid regurgitation, as indicated by the presence of regurgitation waves, is a rare accompaniment of auricular fibrillation; and (2) that the contrary opinion, arrived at by the frequent presence in polygraph tracings of ventricular types of waves, is due to the fact that the contour of intensified impact waves is distorted by polygraph levers so that they simulate regurgitation waves.

Recurrent diastolic waves were frequently present in our records. Their relative size depended, to a considerable extent, on the pressure of the tambour. There was no constant relation to similar waves in the recorded electrocardiogram, nor is it proven that they are indicative of a coarse type of fibrillation or an associated flutter.

EXPLANATION OF PLATES.

PLATE 1.

Fig. 1. Curve showing the character of the normal apex and venous pulses photographically recorded. Reduced nine-tenths.

Fig. 2. Curves of supraclavicular and radial pulses from a case of auricular fibrillation in which prominent arterial impact waves predominate. During diastole small waves (d-d) sometimes recurred. Reduced six-tenths.

Fig. 3. Venous tracing taken by a Jacquet polygraph from the same case as Fig. 2, showing the so-called ventricular type of the venous pulse.

PLATE 2.

Fig. 4. Curves of supraclavicular and radial pulses from a case of auricular fibrillation showing the variety of systolic waves and numerous large diastolic waves. Reduced eight-tenths.

Fig. 5. Supraclavicular and radial pulses in auricular fibrillation with rapid heart rate and deep breathing. The lines correspond to the rise of the R waves of the electrocardiogram simultaneously recorded but not reproduced. Reduced nine-tenths.
Plate 3.

Fig. 6. Supraclavicular and radial pulses in auricular fibrillation. The former shows regurgitant waves almost exclusively. Lines are drawn to correspond to R waves of the electrocardiogram simultaneously recorded, but not reproduced.

Fig. 7. Supraclavicular and radial pulses from a case showing a gradation of systolic types of waves. Marked according to the scheme shown in Text-fig. 1. Reduced one-half.

Plate 4.

Fig. 8. Venous and radial pulses from the same case as Fig. 7 after rest and digitalis medication. The absence of regurgitation waves and the presence of diastolic waves is shown. Reduced nine-tenths.

Fig. 9. Venous and apex curves, showing similarity of waves (p. 13). Reduced about one-half.
FIG. 1.

FIG. 2.

FIG. 3.

(Niles and Wiggers: Venous Pulse in Auricular Fibrillation.)
Fig. 4.

Fig. 5.

(Niles and Wiggers: Venous Pulse in Auricular Fibrillation.)
Fig. 6.

Fig. 7.

(Niles and Wiggers: Venous Pulse in Auricular Fibrillation.)
Fig. 8.

Fig. 9.

(Niles and Wiggers: Venous Pulse in Auricular Fibrillation)