Electrokymographic Studies of the Left Atrium in Normal and Diseased Hearts

By J. B. McKinnon, M.D., and Ben Friedman, M.D.

Electrokymographic tracings of left auricular pulsations in normal and diseased states are described. A characteristic abnormality observed in persons with organic mitral valve insufficiency is believed to be graphic evidence of regurgitation of blood from ventricle into atrium. Electrokymographic evidence of regurgitation was found in persons with mitral stenosis without an audible systolic murmur but instances of mitral stenosis without insufficiency were recorded. Patients with Austin Flint murmurs and individuals with murmurs mimicking mitral insufficiency showed uniformly normal atrial pulsations.

The ELECTROKYMOGRAPH is an instrument which makes continuous recordings of pulsations observed under the fluoroscope. The mechanical aspects of this device and its clinical application have been the subjects of recent reviews. Interpretation of the records is frequently difficult due to vigorous positional movements of the heart which obscure the actual volume changes. This difficulty is especially prominent in the movements of the right atrium and the ventricles. The left atrium is so situated that it partakes to a lesser degree of the vigorous positional movements of the heart. In our experience, tracings can be obtained of this chamber which appear to show changes associated with events in the cardiac cycle. It must be pointed out, however, that extreme care has to be employed in the interpretation of the records. Many artifacts may arise by improper positioning of the recording slit, resulting in changes which are due to mixed pulsations of a variety of structures within the chest. Errors will occur less frequently in the hands of a fluoroscopist who has experience in the use of the electrokymogram.

The pulsations of the left atrium may be recorded in either the right or the left anterior oblique position. There is some difference in the appearance of the tracings; the systolic filling wave recorded in the right oblique position is more prominent than that taken in the left oblique position. The tracings discussed in this paper were taken with the patient erect in the right anterior oblique position, employing a 60-cycle half wave fluoroscope and a Cambridge electrokymograph and recording device. In this position a downward deflection in the curve represents volume contraction or movement of the heart in the direction of the apex, while upward deflection will result from atrial expansion or movement of the heart in the direction of the base.

The normal left atrial curve usually consists of three waves: one in atrial systole and two in atrial diastole (fig. 1). In some instances the two diastolic waves tend to merge. The first wave begins with the onset of the first heart sound and consists of a small upward deflection of brief duration, which returns promptly to the baseline. This is followed by progressive elevation in the baseline (wave 2) which continues until immediately after the second heart sound, at which time it drops off sharply until about the middle of diastole. At this point it may level again or actually rise somewhat, and, shortly thereafter, decline again (wave 3) because of active atrial contraction.

The small upward deflection coincident with first sound (wave 1) is believed to be due to two factors: (1), a hypothetically small amount of regurgitation as the mitral valve is closing, and (2), the elastic effect of the sudden stretch of the mitral valve with the beginning of the isometric phase of ventricular systole. Evidence

From the Veterans Administration Hospital, McKinney, Texas.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the results of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.
in favor of the latter interpretation is afforded by the study of a patient who had auricular fibrillation without demonstrable organic cardiac disease (fig. 2). In this tracing the size of the first wave varies directly with the force of the ventricular contraction, as measured by the height of the carotid pulse wave. The downward deflection of the first wave occurs in the ejection phase of ventricular systole and is believed to represent movement of the atrioventricular septum and the attached atrium toward the apex. It varies in depth and duration depending on whether atrial filling or ventricular contraction dominates the movement. Atrial filling, which occurs simultaneously with ventricular systole but results in diametrically opposite deflection in the atrial tracing, is mainly responsible for the upward deflection of the second wave. The point of change in direction at the peak of wave 2 occurs shortly after the second heart sound and indicates the opening of the mitral valve. The subsequent decline in the curve represents emptying of the atrium during the course of passive filling of the left ventricle. This continues until the middle of ventricular diastole at which point the atrial and ventricular pressures approximate each other and emptying of the atrium stops. The atrial volume remains unchanged or may even be increased slightly until active contraction completes the emptying of the atrium. The third wave is absent in cases of auricular fibrillation (fig. 3). Volume changes as a result of active atrial contraction appear generally to be
small. In some normal patients wave 3 may be absent or appear to blend with the preceding wave. Study of the left atrial curve in one patient with complete heart block similarly demonstrated that the volume of ejection of the left atrium during atrial contraction is extremely small in proportion to that occurring during the passive filling phase of the cycle. These observations are in conformity with the conclusions based on the direct measurements of atrial and ventricular pressure and volume changes.

A characteristic abnormality in the tracing recorded from the left atrium in cases of mitral valve disease has been described by Luisada

![Fig. 3. A 31 year old man with diastolic rumbling murmur at the apex, auricular fibrillation, congestive heart failure, marked left atrial enlargement and calcification of the mitral valve. A systolic murmur was not heard. Note curve of mitral regurgitation and absence of wave 3.](image)

**TABLE 1.** Type of Atrial Tracings in Selected Subjects

<table>
<thead>
<tr>
<th>Clinical Diagnosis</th>
<th>Number of Patients</th>
<th>Normal Tracing</th>
<th>Abnormal Tracing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart</td>
<td>25*</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>Organic mitral valve disease</td>
<td>8</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>9</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Mitral stenosis and insufficiency</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Syphilitic aortic insufficiency and Austin Flint murmur</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Functional mitral insufficiency with organic heart disease</td>
<td>14</td>
<td>14</td>
<td>0</td>
</tr>
</tbody>
</table>

* Two with apical systolic murmurs but no circulatory abnormalities.
† Two tracings are classified as borderline (see text).

![Fig. 4. Above: 25 year old student had an illness characterized by low grade fever, malaise, weakness, a changing P-R interval and favorable response to salicylates. A faint presystolic low pitched apical murmur was heard. No systolic murmur and no enlargement of cardiac chambers was detected. Note diminution in the downward deflection of wave 1. This is a transition to a positive systolic plateau (shown below).](image)

Below: 51 year old man with mitral stenosis and insufficiency, auricular fibrillation and congestive heart failure. The left atrium was enlarged. Note sustained elevation of atrial curve until a point beyond closing of the aortic valves. Carotid pulse tracing lags .03 to .04 sec. behind atrial electrokymogram.

and Fleischner. This consists of a marked elevation of the systolic wave, producing a systolic "plateau." They observed the characteristic changes in all cases of mitral valve disease, whether of mitral insufficiency, mitral stenosis, or stenosis and insufficiency combined.
We have examined 19 patients with mitral valve disease. On the basis of physical signs 9 of these subjects had mitral stenosis and insufficiency, 7 had mitral stenosis alone, and 2 individuals had mitral insufficiency alone.

In general, our findings have agreed with those of Luisada and Fleischner (table 1). Tracings recorded in 11 patients with organic mitral insufficiency were abnormal in 8, borderline in 2, and normal in 1 instance. The borderline tracings were obtained in 2 subjects with far-advanced mitral stenosis and congestive heart failure, the normal tracing in a young man with early mitral stenosis and a faint grade 1 systolic apical murmur. Five of the 8 patients presenting only apical diastolic murmurs without audible systolic murmurs showed abnormal records characteristic of mitral insufficiency (fig. 3). However, in 3 patients who had the physical signs of mitral stenosis without mitral insufficiency, a normal left atrial curve was recorded. In very early valve lesions (fig. 4, top) the first abnormality to be noted is moderate elevation of the first wave, which then levels off and becomes a slowly rising curve throughout the rest of systole. In more advanced valve lesions the regurgitation appears to persist throughout systole and sometimes beyond the closing of the aortic valves (fig. 4, bottom) as evidenced by sustained elevation of the systolic wave. In 2 patients with very far advanced mitral stenosis, cardiac enlargement, marked enlargement of the left atrium and congestive failure, a different configuration appears (fig. 5) in that the atrial filling curves tend to resemble those seen in the normal state.

Normal tracings were observed in each of 8 patients with so-called "functional" mitral insufficiency, as manifested by apical systolic murmurs, grades 2 to 3 in intensity, in the presence of nonvalvular heart disease with well-defined cardiac enlargement (fig. 6). The etiologic agents responsible for the heart disease in these 8 instances were coronary arteriosclerosis in 3 cases, hypertension in 2 patients, and thyrotoxicosis, diffuse myocarditis and anemia with hypertension of chronic nephritis each in one individual. Three patients with aortic insufficiency and an Austin Flint murmur showed no electrokymographic evidence of mitral insufficiency.

Normal atrial curves were recorded also in 2 healthy subjects with no clinical evidence of cardiac or circulatory abnormalities but with
apical murmurs of grade 1 to 2 intensity based on the classification of Levine. Six patients with cor pulmonale and 14 individuals with other types of organic cardiac disorders showed normal configurations in the atrial pulsation.

**Fig. 6. Above:** A 54 year old man with arteriosclerotic coronary disease, marked cardiac enlargement, congestive heart failure and a grade 2 to 3 systolic murmur at the apex. There was no hypertension, anemia or fever. Note normal atrial curve with presumed “functional mitral insufficiency.” **Below:** 26 year old man with hyperthyroidism (basal metabolic rate +42 per cent), moderate cardiac enlargement, grade 2 murmur at the apex.

**DISCUSSION**

The essential feature of the abnormality in the left atrial volume curve in persons with mitral valve disease is an increased filling at the beginning of the isometric contraction phase of ventricular systole. It can be attributed neither to artefacts due to filling of pulmonary veins or aorta nor to transmitted ventricular thrusts, for it precedes these events in the cardiac cycle by at least .06 second. Moreover, it does not appear in tracings taken in normal subjects or in patients with nonvalvular heart disease. The possibility exists that the right ventricular hypertrophy which usually accompanies mitral valve disease might be responsible for a positional change by reason of more forceful contraction. Thus the typical deformity in the tracings might conceivably have no relation to regurgitation of blood but might be related to secondary changes incidental to right ventricular strain. To evaluate this possibility, we examined 6 patients with varying degrees of right ventricular strain secondary to chronic pulmonary disease. In none of these patients was an abnormal atrial curve observed. We believe, therefore, that this abnormality of the atrial filling curve seen in persons with mitral valve disease does actually represent regurgitation of blood through an insufficient valve. The abnormal tracing resembles the auricular volume curves recorded by Wiggers and Feil in experimental acute mitral valve insufficiency.

There are two possible explanations for the tendency for the appearance of a normal pattern in persons with far advanced mitral stenosis: (1) When the mitral orifice is greatly narrowed, there may be very little regurgitation. This seemed to be the situation in one of the patients who came to autopsy (Fig. 6). (2) The high atrial pressure and reduced left ventricular pressure which accompanies advanced mitral stenosis results in a decline in pressure differential between the left ventricle and left atrium. This factor probably plays a minor role, since the ventriculo-atrial pressure gradients in congestive failure are still very high during the phase of ventricular ejection.

The evidence supports the belief that mitral stenosis does occur without regurgitation, but that the absence of an audible systolic murmur in persons with mitral stenosis does not rule out the simultaneous existence of mitral regurgitation.

The apical systolic murmurs which appear in persons with left ventricular dilatation due
to nonvalvular heart disease have been commonly attributed to functional regurgitation as a result of dilatation of the mitral ring or to displacement of the papillary muscles and chordae tendinae to which the valve cusps are attached. The failure to demonstrate abnormal atrial pulsations in conditions where functional mitral insufficiency would be anticipated may be due to the insensitivity of the method of detecting small degrees of regurgitation. In persons with known mitral valve disease, electrokymographic evidence of regurgitation has been detected in 5 instances without an audible systolic murmur and in others with murmurs of grade 1 or 2 intensity. The finding of normal atrial pulsations in individuals with nonvalvular heart disease and loud systolic murmurs suggests that the murmurs are not due to mitral insufficiency or else that, if actual regurgitation is present, it is slight and not related to the intensity of the murmur.

The clinical diagnosis of mitral insufficiency has been based heretofore on indirect evidence, dependent principally upon the presence of an apical systolic murmur. The diagnosis is frequently in doubt, particularly when there is no evidence of associated mitral stenosis. Any procedure which reflects hemodynamic changes related directly to valvular insufficiency should prove to be of great value. Alteration in the electrokymographic tracings of left auricular movement in persons with diseased mitral valves are of a character which can be explained only on the basis of actual regurgitation of blood from the ventricle into the auricle.

This new tool should prove to be of value in the study of a variety of problems. These include: (a) evaluation of the significance of apical systolic murmurs, (b) the progression of mitral valve disease, (c) detection of mitral stenosis in the rare cases when the diastolic murmur is inaudible, and (d) differentiation of the Austin Flint murmur from that of organic mitral stenosis.

**Summary**

(1) The normal left atrial pulsation, as recorded by the electrokymograph, has been described.

(2) A characteristic abnormality in the tracing was observed in cases of mitral valve insufficiency.

(3) This abnormality is not due to forceful contraction of the right ventricle, but is believed to be related to actual regurgitation of blood into the left atrium. The abnormality of the curve tended to be less pronounced in two subjects with very far advanced mitral stenosis and congestive failure. This is attributed mainly to marked diminution in size of the mitral orifice and hence in the degree of regurgitation.

(4) Electrokymographic evidence of regurgitation was found in persons with mitral stenosis without a systolic murmur. Instances of mitral stenosis were observed without clinical or electrokymographic evidence of regurgitation.

(5) Patients with the Austin Flint murmur and subjects with nonvalvular heart disease with or without apical systolic murmurs showed uniformly normal atrial tracings. The absence of confirmatory evidence of regurgitation in persons with nonvalvular heart disease and loud apical systolic murmurs cast considerable doubt on the concept of functional mitral insufficiency as the cause of the murmurs.

**REFERENCES**

Electrokymographic Studies of the Left Atrium in Normal and Diseased Hearts

J. B. MCKINNON and BEN FRIEDMAN

Circulation. 1950;2:572-577
doi: 10.1161/01.CIR.2.4.572

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1950 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/2/4/572