Kinetocardiographic Changes in Ischemic Heart Disease

By E. E. Eddleman, Jr., M.D.

In 1955 Vakil reported the presence of an abnormal outward precordial pulsation in patients with myocardial infarction.1 The kinetocardiogram (graphic representation of low-frequency displacement precordial movements) offers a means by which these changes can be objectively evaluated. Thus, a number of reports have defined some of the abnormalities of precordial pulsations encountered in these groups of patients.2-6 The following report presents a review of the kinetocardiographic patterns encountered in patients with ischemic heart disease.

Technics

Low-frequency precordial movements (kinetocardiograms) were recorded with use of the method previously described.7-9 The apparatus consists of a flexible metal bellows connected by a short piece of Tygon tubing to a low-pressure strain-gage (Statham P-5A). The output is registered on an oscillographic recorder (Electronics for Medicine DR-8). A sensing probe, approximately 7 mm in diameter, and attached on the closed side of the bellows, can be placed perpendicular to any point on the chest wall. The entire set-up is mounted on a cross bar above the patient in order to record absolute chest wall movements and not relative interspace motion. The significant characteristics of the system are as follows:

1. The bellows senses true displacement of the precordium because of the external frame of reference, whereas the apexcardiogram as usually taken senses relative interspace motion.

2. The system is linear for displacement movements up to 25 cycles per second within acceptable physical limits (1 DB).

3. The rigidity of the bellows system does not significantly alter the pattern as the records are similar, if not identical, to those taken with a photo-electric cell.4

4. The records are highly reproducible from day to day in the same patient if taken from identical precordial positions.

5. The system described is sufficiently sensitive so that records can be obtained from many points over the precordium and not just at the apex. Many of the abnormalities noted in patients with heart disease involve marked changes in precordial movements in areas other than at the apex.

Nomenclature

The term “kinetocardiogram” is used to define records of precordial motion as obtained with a displacement pick-up mounted from a fixed point above the chest wall. This distinguishes the records from those of the apexcardiogram, since that technic records relative interspace motion and not true displacement movements of the chest wall. In addition, the kinetocardiogram is recorded from many points over the chest wall, and therefore the term “apexcardiogram” is not applicable. The letter “K” is used as an abbreviation for the kinetocardiogram, and the location of the point where the traces are recorded is referred to by subscripts (K14). The first digit indicates the vertical line as used in precordial electrocardiography, and the second subscript refers to the intercostal space. Thus, K14 is the kinetocardiographic record taken from the right parasternal line in the fourth intercostal space and K24 is from the left parasternal line in the fourth intercostal space. Records are obtained from points corresponding to the precordial “V” leads and from the left, right, and mid-epigastric areas just beneath the costal margin. These records are designated “KEL,” “KER,” and “KEM,” respectively.

Normal Kinetocardiograms

Figure 1A presents a normal kinetocardiogram taken from K14 position. There is an initial systolic outward movement in the K14.
 SYMPOSIUM: CORONARY ARTERY DISEASE

The upper tracing labeled “C” is the carotid pulse. The arrows labeled “P” and “QRS” indicate the onset of the P wave and the QRS complex in the electrocardiogram. The first curved arrow points to the atrial movements that occur after the P wave and before the QRS complex. The second curved arrow points to the marked inward movement during ejection. The third curved arrow points to initial outward ventricular movement, which is presumably due to right ventricular activity. Note that the major movement is inward, occurring during ejection. Time lines are 0.02 second apart.

The normal K₁₄ (right parasternal) record from a normal subject. The upper tracing labeled “C” is the carotid pulse. The arrows labeled “P” and “QRS” indicate the onset of the P wave and the QRS complex in the electrocardiogram. The thrust in normal subjects is of relatively small amplitude and of brief duration as measured on an arbitrary base line.*

The records from K₂₄ and K₃₄ have a contour somewhat intermediate between the K₁₄ and K₄₅ record. The normal upper limit in duration of the left ventricular thrust and the initial outward movement in the K₂₄ trace is 0.085 second as measured along the base line. This figure represents two standard deviations above the mean. Thus, any outward movement 0.09 second and greater in duration should be considered abnormal.

Atrial Movements

Atrial systole (between the onset of the P-wave and QRS complex in the electrocardiogram) is usually associated with a small outward and then inward movement (fig. 1A, first arrow). Occasionally there is a terminal outward movement that may last as long as 0.04 second after the onset of the QRS complex. The amplitude of these movements seldom exceeds one third of the total amplitude of the trace in normal subjects. Atrial movements may become relatively larger in ischemic heart disease.

Myocardial Infarction

Definition of Changes

Patients with myocardial infarctions often

* The base line is defined as a horizontal line which is placed on the curve at a point 0.04 second after the onset of the QRS complex (fig.1).

brief duration as measured above the base line (0.06 second). The major record in systole is still a retraction or an inward movement during ejection.
normal subjects. This paradoxic or outward movement has been arbitrarily divided into several categories according to its duration and configuration. An outward systolic movement measured 0.09 second to 0.20 second in duration as measured on the base line is considered as "an abnormal movement" (fig. 2). When the duration is 0.20 second or greater it is classified as a "bulge." In addition, there are two configurational changes. The most common is an "early systolic bulge," which is so designated because it begins before ejection (fig. 3). The other less common type is a mid or late systolic "bulge" (fig. 4). starting after the onset of the carotid upstroke.

**Mechanism**

In 1933 Wiggers and Tennant ligated a coronary artery in dogs and noted that the ischemic area contracted poorly during systole and ballooned out with the rise in intraventricular pressure. Presumably these outward movements are transmitted to the precordium and recorded as "bulges." Since the precordial location of the bulges compares generally well to the anatomic location of the infarction, this explanation appears to be the most plausible; however, an abnormal

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**Figure 2**

A kinetocardiogram taken from a patient who had an old myocardial infarction and angina pectoris. The upper curve is the carotid pulse. The vertical time lines are 0.02 second apart. The onset of the QRS complex in the electrocardiogram is indicated by arrow labeled "QRS." Note the pressure of an abnormally prolonged early systolic outward movement (curved arrows). Its duration above the dotted base line is 0.12 second. This is prolonged above normal (0.085 second) but not as sustained as a "bulge" as defined.

show (68 per cent) a paradoxic systolic outward movement of longer duration than the brief outward movement as noted in

**Figure 3**

A typical kinetocardiogram showing a "bulge" from a patient with a myocardial infarction. The vertical time lines are 0.02 second apart. The upper tracing is the carotid pulse. The arrows labeled "QRS" represent the onset of the QRS complex in the electrocardiogram. There is a sustained outward movement throughout ejection, beginning shortly after ventricular excitation (bulge), in contrast to the pronounced increased movement during ejection in a normal subject.

**Figure 4**

A kinetocardiograph tracing from a patient with an old myocardial infarction and angina pectoris. The vertical time lines are 0.02 second apart. The upper tracing is the carotid pulse. The ones labeled "P" and "QRS" represent the onset of the P wave and the QRS complex in the electrocardiogram. Note the outward movement during mid and late systole (curved arrow), which represents a "late systolic bulge."

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movement of the entire heart cannot be excluded as a cause of the bulge. The fact that the "bulge" may be localized to only one precordial position also supports the first hypothesis.

Left and right ventricular hypertrophy can also produce paradoxic outward movements of the precordium. However, bulges cannot be attributed to ventricular hypertrophy if they occur in patients with normal size hearts without left or right ventricular enlargement. The character and location of the impulse is of some help in distinguishing left or right ventricular hypertrophy from ischemic "bulges."

**Location and Characteristics**

Bulges may occur at any position over the precordium and in the epigastric areas; however, they are exceedingly uncommon to the right of the sternum. The impulse is usually diffuse and not so well localized as the apex thrust in normal subjects or as in patients with left ventricular hypertrophy. On the other hand, they are not usually so diffuse as the parasternal lift in patients with right ventricular hypertrophy. The point where the bulge is largest in amplitude is of some help in distinguishing the impulse from right and left ventricular hypertrophy. Table 1 lists location of the maximum outward movement above the base line in 69 of 102 patients with myocardial infarctions who had bulges. No patient with an infarction had a bulge in the K14 area, whereas 22 per cent of the 46 patients with mitral stenosis had the largest impulse at this point. In addition, no patient with left ventricular hypertrophy had the largest outward movement at the V2 or K24 area, whereas 17 per cent of the patients with bulges had the largest impulse at this point.

**Incidence of Bulges**

Table 2 lists the incidence of abnormal movements and bulges in patients with myocardial infarctions, as previously reported. The incidence is higher in acute infarctions than in old infarctions. In fact, a previous study indicated that essentially all patients with acute infarctions have "bulges" present sometime during the first 6 weeks if frequent or daily traces are taken. A subsequent follow-up on these patients indicated that about 30 per cent disappeared or remained as only an "abnormal early systolic movement."

This is in keeping with the over-all incidence in old infarctions (table 2).

**Significance**

Although the location of the largest bulge is of some help in the differential diagnosis, other clinical information is necessary in evaluating these changes. For example, if a patient has never had a cause for left ventricular hypertrophy and is seen with a chest pain compatible with an infarction, a sustained outward movement at the K45 point can be interpreted as an ischemic bulge. If no history or other information is available, there is no way of distinguishing the bulge from the impulse of left ventricular hypertrophy. On the other hand, bulges may be diagnostic in a rare instance. They have been recorded

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**Table 1**

<table>
<thead>
<tr>
<th>KCG Location</th>
<th>Myocardial Infarction (69 Patients)</th>
<th>Aortic Valvular Disease and Hypertensive Cardiovascular Disease (100 Patients)</th>
<th>Mitral Stenosis (46 Patients)</th>
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<tbody>
<tr>
<td>K14</td>
<td>0</td>
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<td>22%</td>
</tr>
<tr>
<td>K24</td>
<td>17%</td>
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</tr>
<tr>
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</tr>
<tr>
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</tr>
<tr>
<td>KER</td>
<td>3%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>KEM</td>
<td>7%</td>
<td>0</td>
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as well as felt at the bedside in patients with atypical pain of infarction who are known not to have either left ventricular hypertrophy or right ventricular hypertrophy. Only later did the electrocardiogram confirm the diagnosis of a myocardial infarction.

There are several other impressions concerning the interpretation of bulges that are of sufficient interest to mention. The majority of patients who have bulges distributed over the entire precordial appear to have a poor prognosis. Conversely, most of those with a transient bulge or a bulge that is localized to only one precordial area do well, and many have an excellent exercise tolerance. This would suggest that the extent of the bulge bears some correlation to the size and severity of the infarction. However, the over-all use of the kinetocardiographic records in relation to the prognosis of myocardial infarctions must await further documentation. It should be mentioned that the presence of a "bulge" does not distinguish between a true ventricular aneurysm or only a physiologic ballooning during systole. Bulges that appear identical have been noted at autopsy in patients with true ventricular aneurysm as well as in patients with no aneurysm and only myocardial infarction.

**Angina**

Patients with angina pectoris without known or proven infarctions may exhibit bulges (fig. 5). These can be either early or late systolic bulges. In all likelihood, these are due to infarctions which have been unrecognized, either clinically or electrocardiographically. In a recent study of 11 patients who gave no history of an infarct and who had no abnormality in the electrocardiogram, definite bulges were present in only two; however, abnormal systolic movements were present in seven of the 11. Thus, some abnormalities in the kinetocardiogram records occur frequently; however, true bulges are relatively rare except during the pain or after an infarction. At times, the improvement or lack of improvement in the bulge after nitroglycerin may be useful in distinguishing an ischemic bulge from a bulge due to an infarction.

**Effect of Exercise**

**Effect on Bulges**

Exercise which does not produce anginal pain in patients with resting bulges may

<table>
<thead>
<tr>
<th>Table 2</th>
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<tbody>
<tr>
<td><strong>Incidence of Bulges in Myocardial Infarctions</strong></td>
</tr>
<tr>
<td>Acute myocardial infarctions (posterior and anterior)</td>
</tr>
<tr>
<td>60</td>
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<tr>
<td>Old myocardial infarctions (posterior and anterior)</td>
</tr>
<tr>
<td>All infarctions</td>
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</tbody>
</table>

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**Figure 5**

A kinetocardiographic tracing from a patient with only angina pectoris. There was no history or electrocardiographic evidence of a myocardial infarction. The upper tracing is the carotid pulse. The vertical time lines are 0.02 second apart. The arrows labeled "P" and "QRS" represent the onset of the P wave and the QRS complex in the electrocardiogram. Note that after the P wave there is a large outward atrial movement followed by an inward atrial movement extending about 0.05 second after the onset of the QRS complex. The main movement during systole is a sustained outward movement or a "bulge" (curved arrows). This represents a "bulge" in a patient with only angina pectoris.
minimize the bulge or rarely cause it to disappear entirely. On the other hand, patients with angina and no bulge at rest may develop a bulge during exercise, especially when pain occurs. The latter change occurs in approximately 30 per cent of the patients and may aid in diagnosis but obviously cannot as yet be used as a sole means of establishing the diagnosis of angina pectoris.

**Exercise and Atrial Movements**

Skinner et al.\(^5\) reported an increase in amplitude of precordial atrial movements following exercise in patients with angina. The ratio of the amplitude of the atrial movements divided by the total or largest amplitude in the entire complex increased with exercise (the normal ratio is rarely over 30 per cent). Subsequently, Benchimol and Dimond,\(^12,13\) using the apexcardiographic technic, reported abnormally high atrial ratios in patients with angina pectoris after exercise. They suggested that this procedure could be employed as a diagnostic method for angina. Results from this laboratory indicate that the separation of normal subjects from patients with angina is not so reliable as would be desired. Further studies are needed before this can be accepted as a diagnostic method for angina. Skinner et al.\(^14\) demonstrated that exercise increases the atrial ratio in patients with heart failure, regardless of etiology. Thus, an abnormally high resting or postexertional ratio is not specific for the diagnosis of angina.

**Summary**

Patients with myocardial infarctions may show a paradoxic precordial outward systolic movement that can be recorded by the kinetocardiographic technic and often is felt at the bedside.

Three types of changes occur. The first is an early systolic outward movement, which is abnormally long in duration but not sustained throughout systole. The second is a sustained outward movement during systole called a “bulge,” and the third is a late systolic outward movement.

Bulges can be recorded in approximately two thirds of the patients with known infarctions with the largest outward movement most frequently noted in or near the V\(_3\) area.

A few patients with angina may have a bulge at rest; however, apparently about 30 per cent develop a bulge on exercise.

**References**

11. Unpublished data.
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