Noninvasive Diagnosis of Coronary Artery Disease: The Cardiokymographic Stress Test

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SUMMARY Stress-induced abnormalities of regional left ventricular wall motion were assessed by cardiokymography (CKG) during the course of maximal treadmill exercise tests in 157 patients, of whom 122 subsequently underwent coronary angiography. Seventy patients had significant angiographic coronary artery disease and 52 were normal. Forty-one of the 70 patients developed >0.1 mV ST-segment depression (ECG sensitivity 59%) and 52 of 70 patients developed abnormal systolic outward motion by CKG (CKG sensitivity 74%). Among the 52 normals, 36 had negative ECG stress tests (ECG specificity 69%) and 49 had normally sustained systolic inward motion by CKG (CKG specificity 94%). The stress CKG was normal in 15 of the 16 false-positive stress ECGs; the stress ECG was correctly normal in two of the three false-positive stress CKG tests. Only one normal patient had concordantly false-positive ECG and CKG tests. The predictive accuracy of concordant ECG and CKG interpretations was, therefore, higher than either test alone.

These data suggest that regional wall motion abnormalities, which are sensitive and specific markers of myocardial ischemia, may be detected noninvasively by CKG. We concluded that CKG helps identify false-positive and false-negative ECG stress tests and improves the diagnostic accuracy of stress testing for detection of coronary artery disease.

THE STANDARD electrocardiographic treadmill exercise test is an established clinical procedure for detecting and evaluating patients with coronary artery disease. As a diagnostic tool, however, the procedure is limited by the somewhat low sensitivity and specificity of electrocardiographic ST-segment depression as a marker of ischemia. Thus, when the procedure is used in a population with a low prevalence of disease, a very high false-positive rate is inevitably observed. A technique that uses a second, independent marker of regional myocardial ischemia and is readily applicable during ECG stress testing could potentially enhance the diagnostic accuracy of the test procedure.

Several studies have shown that regional left ventricular wall motion abnormalities are an additional sensitive and specific marker of regional ischemia that may be induced by exercise or atrial pacing. Until recently, however, suitable noninvasive methods for the clinical detection of wall motion abnormalities have been limited.

The cardiokymograph (CKG) is a relatively simple electronic device capable of detecting an analog representation of regional wall motion noninvasively and was first described in 1967 as the displacement cardiograph. Previous studies have compared the noninvasively recorded CKG signal with simultaneous direct recordings from a contiguous epicardial length gauge in the open-chest dog before and after coronary occlusion. Both methods showed the same predictable sequence of ischemic abnormalities with a high degree of correlation. CKG recordings have also been shown to be predictive of ventriculographic wall motion abnormalities. Because the CKG is potentially applicable to routine stress testing, the purpose of this study was to determine whether cardiokymography can detect exercise-induced wall motion abnormalities and to evaluate its usefulness as a noninvasive adjunct to electrocardiographic exercise testing in the clinical diagnosis of ischemic heart disease.

Methods

The Cardiokymograph

The CKG consists of two components, a probe and a signal-conditioning unit. The CKG probe uses a 5-cm, circular, flat capacitive plate (formerly an inductive coil) as part of a high-frequency, low-power oscillator. The plate is mounted in a plastic ring and strapped to the chest, with an air gap separating the coil from the skin. Activation of the plate by battery current causes it to emit a low-energy (0.5 mW/cm) electromagnetic field (1.5 amps/m). This field is capable of tissue penetration like any electromagnetic field. Motion within the field causes a change in effective capacitance, producing a change in the frequency of the oscillator. This change in frequency is converted by the signal-processing unit into a change in voltage proportional to the original motion, which is amplified and displayed on a multichannel recorder as a continuous analog signal. The frequency response of the system is flat ±10% from 0.1–90 Hz with a phase shift.
of ± 20° at these frequency extremes. Details of the electronics and measurement characteristics of the CKG have been published. The CKG is frequently thought to resemble a conventional apex cardiogram (ACG), or the less well known kinetocardiogram (CKG). However, the CKG is different from conventional apex cardiography. The ACG can only record chest wall vibrations by direct contact in a small focal area over the cardiac apex. The ACG specifically records motion of the transducer diaphragm relative to the rim of the sensing head, and not absolute motion. Direct contact with the chest wall introduces distortions by variations in transducer pressure and by variations in the tissue characteristics of the thorax. The conventional ACG recording demonstrates a sustained outward systolic motion representing the complex thrusting motion of the cardiac apex, but cannot be used to analyze segments remote from the apex. It has, therefore, not found a clinical application in the evaluation of patients with ischemic heart disease. On the other hand, the CKG tracing is similar to the tracing obtained by the technique of kinetocardiography, which records absolute chest wall motion by direct contact over a wide precordial area. The main limitation of the KCG is its elaborate and cumbersome apparatus, which is not portable, and the direct chest wall contact of the probe, which introduces the same distortions that affect apex cardiography. The CKG records absolute motion without contact with the chest, eliminating the problem of distortion inherent in the ACG and KCG. Like the KCG, the CKG records motion over the entire precordium, but because of the considerable tissue penetration of the electromagnetic field, the CKG responds to deeper cardiac movement, as well as to precordial surface movement.

Protocol

Studies were performed during the course of diagnostic treadmill exercise testing on 157 patients from two populations: 27 apparently healthy volunteers and 130 patients with suspected coronary artery disease who subsequently underwent diagnostic coronary angiography within 2 days. Patients with either left bundle branch block or left ventricular hypertrophy on the resting 12-lead ECG were excluded from the study because they might bias the comparison against the ECG. Other patients were excluded if they had a clinical contraindication to treadmill exercise. The remaining subjects were studied consecutively. In the catheterized group there were 96 men and 34 women (mean age 52 years, range 34–72 years). Twenty-three percent were asymptomatic, 28% had atypical chest pain and 49% had typical angina pectoris. In the subgroup of 52 patients with normal coronary angiography, 85% were asymptomatic or had atypical chest pain. In all cases, the decision for coronary angiography was made independently by the patient’s attending physician. The indication for coronary angiography in the asymptomatic patients was either a previously positive stress test, an abnormal ECG, previous symptoms or other clinical circumstances requiring a diagnosis.

Each subject underwent a resting 12-lead ECG. Cardiokymographic tracings were recorded in the supine position at voluntarily held end-expiration over standard precordial electrocardiographic positions V1 to V5, with additional tracings recorded one interspace above and below these positions. A transthoracic electrocardiographic lead, an indirect carotid pulse tracing and a phonocardiogram were recorded simultaneously for timing purposes on an Electronics for Medicine VR6 physiologic recorder at a paper speed of 100 mm/sec. The most “normal” CKG recording from this scan was chosen as the control and the position was marked on the chest. If all of the resting CKG recordings were normal, the CKG recorded at the fourth interspace in the V5 line was chosen as control, because fluoroscopically this position was most frequently centered over the anterior left ventricle. The criteria for “normal” and “abnormal” CKGs were empirically based on the analog representation of segmental wall motion as determined by direct myocardial gauges and shown to be identical to the simultaneously recorded noninvasive CKG before and after a coronary occlusion. The normal CKG configuration, which is morphologically analogous to the left ventricular time-volume curve, is shown in figure 1. During ejection, the normal CKG (and normal length gauge) shows an initial upward motion, followed by a predominate downward motion throughout ejection, without paradoxical bulges. Certain variations from this typical pattern are known to occur when using a variety of other noninvasive techniques. Figure 2 shows the range of patterns considered atypical but still normal; each pattern is characterized by relatively continuous downward (i.e., inward) motion during systolic ejection (defined from the onset of the delay-corrected carotid upstroke to the phonocardiographic second heart sound). These “normal” morphologies were designated type I patterns.

Each patient then performed a progressive, symptom-limited, maximal treadmill exercise test according to the Bruce protocol. Four ECG leads (X, Y, Z and V3) and cuff arterial pressure were recorded every minute during the exercise period and for a minimum of 10 minutes during recovery, while the transthoracic V5 ECG lead was continually monitored throughout the exercise and recovery periods. Exercise was terminated upon achieving 100% of the age-predicted heart rate, unless premature termination was indicated by development of chest pain, ventricular arrhythmia or exhaustion. The four-lead ECG, phonocardiogram, carotid pulse and CKG were recorded within 2 minutes of exercise termination and every minute thereafter during the recovery period. All CKG recordings after exercise were obtained over the single precordial position selected as the control and previously marked on the chest.
A normal cardiokymogram (CKG) with a phonocardiogram, an ECG and a carotid tracing for timing purposes. The "a" wave is a small, positive wave following the electrocardiographic "p" wave, representing the effect of atrial contraction. After the electrocardiographic QRS and before the first heart sound, the initial ventricular motion (IVM) wave is inscribed. The peak of this small positive wave usually coincides with the first vibrations of the first heart sound (i.e., the onset of isovolumic systole). During isovolumic systole there is a variable, sharp, downward deflection called preejection retraction (PER), with a subsequent upward motion that probably represents total heart movement. With the onset of ejection (20-50 msec before the carotid upstroke) there is an initial upward motion terminating in the "I" point, which probably represents a change in the contour of the heart and rotation of the apex. This positive wave is coincident with the palpable left ventricular precordial thrust. The "I" point represents the onset of the inward cardiac motion during systolic ejection (SIM). Inward motion terminates at the "O" point, often with a small notch in the tracing at the time of the second heart sound. The "O" point signifies the onset of the outward relaxation wave, which is passive and follows ventricular volume changes.

FIGURE 1. Variation in the normal cardiokymogram (CKG) pattern (type I patterns). Although there are differences in the height of the upward wave during early ejection, the depth of the isovolumic preejection retraction wave, the timing of the "O" point, and the presence or absence of positive waves during isovolumic relaxation, the common feature of all tracings is continuous inward motion during ejection.
Data Analysis

The exercise test was considered inadequate if the patient did not achieve 90% of his or her age-predicted maximum heart rate. The pre- and postexercise ECG and CKG were qualitatively analyzed by three and four different observers, respectively, without knowledge of the angiographic data. The criterion for a “positive” stress ECG was the development of at least 1.0 mm horizontal or downsloping ST-segment depression, 80 msec or more after the J point, either during exercise or recovery.

Two sets of criteria for an “abnormal” CKG pattern were defined in relation to the known effects of ischemia on regional wall motion. The first abnormality was defined as paradoxical systolic outward motion of an amplitude equal to or greater than that of initial inward motion, occurring during any part of systolic ejection. This pattern was termed type II or “dyssynergy.” The second abnormality was defined as the development of total absence of inward motion (“akinesis”) or holosystolic outward motion (“dyskinesis”). A variation of this pattern also included in this category was systolic outward motion occurring for less than the entire period of ejection, but not preceded by inward motion during the first half of the ejection period; i.e., the inward motion was delayed and only occurred during the last half of ejection or during diastole. These patterns were termed type III. A representation of all three types is shown in figure 3.

The resting CKG was classified as either type I (normal), type II (dyssynergy) or type III (dyskinesis), depending on the single control tracing. The postexercise data were similarly classified and the stress test was considered positive if a resting type I tracing became type II or III after exercise or a resting type II became type III after exercise and then returned to the control configuration during recovery.

Biplane left ventriculography and coronary angiography (Judkins technique) were analyzed by two observers without knowledge of the exercise data. The coronary angiogram was considered abnormal if a stenosis of greater than 50% of the luminal diameter was observed in at least one major coronary artery. Conflicting interpretations were resolved by group review without knowledge of the other findings. The results were correlated by chi-square analysis, using McNemar's test for correlated proportions, as well as tests for proportionality and the paired t test.

Results

A technically adequate CKG stress test was obtained in 96% of the 157 subjects studied. Because of the subjective nature of the CKG interpretations, we analyzed interobserver variability in the interpretations of the CKG by four independent readers. All four CKG readers completely agreed on their interpretations in 63% of the patients and in 80% of the tracings. There was unanimous interpretation of the postexercise response in 68% of the patients, and only one of the readers disagreed on the postexercise response in another 23% of the patients. Therefore, in 91% of the patients, at least three of the four readers had concordant interpretations. In 9% of the patients, two of the four readers had discordant interpretations from the other two; these were resolved by group review.

Normal Volunteers

The CKG was normal (type I) in the precordial position selected as the control resting tracing in all 27 apparently healthy volunteers. The location from which the control resting tracing was obtained was medial and superior to the cardiac apex, usually at, or one interspace below, electrocardiographic position V₅ or V₆. Variations in the normal configuration, however, were observed not only from patient to patient but also from position to position (fig. 2).

All 27 volunteers had negative ECG stress tests. Two minutes after exercise, two of the 27 subjects demonstrated a type II pattern and one a type III pattern. Four minutes after exercise, 26 of the 27 tracings were normal; only one showed a type III pattern that did not return to control by 10 minutes. A typical example of a normal test is given in figure 4. The criterion for a CKG stress test abnormality applied to the interpretation of the group undergoing coronary arteriography was defined, therefore, as exercise-induced dyssynergy or dyskinesis (type II or III response) that persisted for at least 4 minutes and subsequently returned to the control pattern.

![Figure 3](https://example.com/figure3.png)

**Figure 3.** Criteria for the three fundamental types of cardiokymographic tracings. Type I (normal) exhibits systolic inward motion (SIM) throughout ejection, without systolic outward motion (SOM), and with the “0” point occurring near the end of ejection. Type II (“dyssynergy”) displays a reduced amplitude SIM associated with significant paradoxical SOM, and with the “0” point occurring during the first half of ejection. Type III (“dyskinesis”) is characterized by a holosystolic outward motion without early SIM.
Sensitivity and Specificity for Detection of Angiographic Coronary Artery Disease

Of the 130 patients evaluated by angiography, eight were excluded from the study. In seven, a resting normal pattern was not obtained at any precordial position (two for technical reasons) and in one patient a postexercise tracing could not be obtained until after the fourth minute of recovery due to inability to voluntarily breath hold. The analysis of the treadmill results was limited, therefore, to the remaining 122 subjects. The sensitivity and specificity of ECG and CKG stress testing for the diagnosis of angiographically demonstrable coronary disease are summarized in table 1.

Seventy of 122 patients (57%) had significant coronary artery disease. The ECG stress test was abnormal in 41 (sensitivity 59%) while the CKG stress test was abnormal in 52 (sensitivity 74%; p = 0.05). There were 21 type III responses and 31 type II responses. Eleven of the 29 false-negative ECG stress tests occurred in patients in whom the exercise was considered inadequate because of failure to achieve 90% of the age-predicted maximum heart rate. The lack of an ischemic ECG response in these 11 patients does not, therefore, constitute a real false-negative stress test. If we exclude the 11 patients with inadequate exercise, the ECG sensitivity would increase from 59% to 69%, and the CKG sensitivity would increase from 74% to 76% (p = NS). Nevertheless, the stress CKG was correctly positive in seven of these 11 negative but inadequate ECG stress tests. Therefore, we include these patients because the results suggest that an inadequate stress for the ECG response may not be an inadequate stress for the more sensitive CKG response. Moreover, four of the false-negative CKG stress tests were abnormal immediately after exercise but normal 4 minutes after exercise and were therefore considered “negative” by the time criterion defined in the

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<tr>
<th>Table 1. Electrocardiographic and Cardiokymographic Correlation with Coronary Angiography: Sensitivity and Specificity</th>
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<td>Coronary angiogram</td>
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*Inadequate stress (subjects reached < 90% of predicted heart rate).

Abbreviation: CKG = cardiokymogram.

**Figure 4.** A concordantly negative ECG and cardiokymographic (CKG) stress test in a patient without coronary artery disease. Both tracings are normal at rest and after exercise. In fact, the slope and amplitude of the CKG ejection wave increases, and there is an accentuation of the preejection retraction and the early systolic upward motion representing increased total heart motion. The carotid pulse and phonocardiogram are recorded simultaneously. LAD = left anterior descending coronary artery; TM ECG = treadmill electrocardiogram; Ant. LV = anterior left ventricle.
volunteer population. The CKG sensitivity would increase to 80% by eliminating this time criterion, although specificity would decrease slightly. Typical responses are shown in figures 5-7.

Fifty-two of the 122 patients had no significant coronary artery disease by angiography. The ECG stress test was negative in 36 (specificity 69%), while the CKG was negative in 49 (specificity 94%; \( p < 0.001 \)). The stress CKG was correctly normal in 15 of the 16 false-positive stress ECGs (fig. 8). The stress ECG was correctly normal in two of the three false-positive stress CKG tests.

**Figure 5.** A concordantly positive ECG and cardiokymographic (CKG) stress test in a patient with severe triple-vessel coronary artery disease. Both tracings are normal at rest, but 1.5 minutes after exercise, 2 mm of downsloping ST-segment depression and an abnormal (type III, "dyskinetic") CKG pattern develops. This holosystolic outward motion diminishes by 2 minutes and then returns to normal by 5 minutes after exercise.

**Figure 6.** An example of a concordantly positive stress test with a type II ("dyssynergic") response induced by exercise. The arrow indicates the abnormal systolic outward motion induced by exercise. Ant. LV = anterior left ventricle; CKG = cardiokymogram; LAD = left anterior descending coronary artery; TM ECG = treadmill electrocardiogram.
FALSE NEGATIVE TREADMILL ECG WITH TRUE POSITIVE CKG

Influence of Disease Prevalence and Symptoms on Test Accuracy

In table 2, patients are subdivided into groups based on their presenting symptom. Patients with atypical chest pain had a low (30%) prevalence of angiographic coronary artery disease, and the CKG had a significantly higher sensitivity, specificity and predictive accuracy than the ECG. Asymptomatic patients had a 64% prevalence for coronary artery disease and patients with typical angina had a 91% prevalence for coronary artery disease. There were no statistically significant differences between the ECG and CKG in these two groups. When asymptomatic patients are combined with patients presenting with atypical chest pain, the prevalence for coronary artery disease is 45%, and the CKG has a significantly higher specificity and predictive accuracy than the ECG.

In the entire study population, the total predictive accuracy (percentage of correct responses) of the treadmill CKG was 83%, compared with 63% for the treadmill ECG (p < 0.01).

Exercise-induced CKG abnormalities persisted significantly longer than the ECG abnormalities during the recovery period. Thus, the ECG returned to baseline in the 41 patients with a true-positive test within 9.4 ± 5.1 minutes (sd) and in the 16 with a false-positive test in 6.9 ± 6.0 minutes (p = NS), while the CKG returned to normal in the 52 true-positive CKG tests in 13.6 ± 7.9 minutes (p < 0.001).

FIGURE 7. A false-negative stress ECG associated with a true-positive response on the cardiokymogram (CKG). The CKG changes from a type I to a type III pattern with exercise and then reverts to normal 30 minutes after exercise. There are no ST-segment changes, although the patient had significant coronary artery disease.

FIGURE 8. A false-positive stress ECG test associated with a true-negative response on the cardiokymogram (CKG). This patient had normal coronaries but developed 4 mm of ST-segment depression, although the CKG was normal (type I) before and after exercise.
TABLE 2. Sensitivity, Specificity and Total Predictive Accuracy of the Electrocardiogram and the Cardiokymogram in Different Subgroups, Divided by Presenting Symptoms

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<tr>
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<th>Asymptomatic</th>
<th>Atypical chest pain</th>
<th>Typical angina</th>
<th>Asymptomatic and atypical pain</th>
<th>Total</th>
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<tr>
<td></td>
<td>Sn (%)</td>
<td>Sp (%)</td>
<td>TPA (%)</td>
<td>Sn (%)</td>
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<td>ECG</td>
<td>71</td>
<td>50</td>
<td>64</td>
<td>63</td>
<td>59</td>
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<td>CKG</td>
<td>71</td>
<td>88</td>
<td>78</td>
<td>67</td>
<td>59</td>
</tr>
<tr>
<td>CAD prevalence</td>
<td>(64%)</td>
<td>(100%)</td>
<td>(30%)</td>
<td>(91%)</td>
<td>(57%)</td>
</tr>
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* p < 0.05.
† p < 0.01.
Abbreviations: Sn = sensitivity; Sp = specificity; TPA = total predictive accuracy; CKG = cardiokymogram; CAD = coronary artery disease.

Influence of Severity and Distribution of Coronary Artery Disease on Test Responses

Abnormal exercise CKG responses were related more to the presence of anterior descending and/or main left coronary artery stenosis than to the number of diseased vessels. Moreover, the frequency and type of abnormal CKG response was related to the magnitude of anterior coronary stenosis (fig. 9). Regardless of associated disease, only one of 36 patients without any anterior descending or main left stenosis had an abnormal CKG response (type II) and only four of 23 patients with less than 50% stenosis had a similar response, one of which was type III. In contrast, 13 of 23 patients with 50-75% stenosis and 37 of 40 patients with more than 75% stenosis had an abnormal response, 21 of which were type III.

Thus, 15 of the 18 false-negative CKG responses occurred in patients with less than high-grade (>75%) anterior descending or main left coronary artery stenosis. Only two of 28 patients with isolated circumflex and/or right coronary stenosis had abnormal responses, and both were type II.

Analysis of Combined Test Results

Because the two tests were performed simultaneously with the aim of establishing a single diagnosis (the presence or absence of coronary disease), the combined results were analyzed as a single test (table 3). Only 76 of the 122 test results (62%) were concordant (33 positive and 43 negative). The predictive accuracy of a concordantly positive test for coronary artery disease was 97% (32 of 33), and 79% (34 of 43) for a concordantly negative test for absence of coronary artery disease. The overall predictive accuracy of a discordant result was 87% (66 of 76), which was greater than for either test alone.

The remaining 46 tests were discordant. The overall predictive accuracy of a discordant test result for coronary artery disease was 76% (35 of 46) for the CKG, compared with only 24% (11 of 46) for the ECG (p < 0.001).

Discussion

Regional left ventricular contraction abnormalities accompanying ischemic heart disease have been recognized since the nineteenth century.23,24 Recently, studies have established that these changes in regional wall motion are highly sensitive to the development of myocardial ischemia, occurring within 5 seconds of acute coronary occlusion6-8 and preceding or occurring without detectable electrocardiographic ST-segment changes.6,9 Such abnormalities are often dynamic, being provoked by stresses that increase myocardial oxygen demand5,11-13 and improved by interventions that tend to relieve the imbalance between supply and demand.7,25,26 Application of regional wall motion analysis, therefore, to clinical stress testing might improve the diagnostic accuracy of the

Table 3. Correlation of Coronary Angiography with Combined Electrocardiographic and Cardiokymographic Analysis

| Coronary angiography | ECG | CKG | + | - |
|----------------------|-----|-----|---|---|------|
| Concordant           |     |     | + | + | 32 
| n = 76 (62%)         |     |     | - | - | 9 34|
| Discordant           |     |     | - | + | 20 
| n = 46 (38%)         |     |     | + | - | 9 15|
| Total                |     |     | 70| 52| (n = 122) |

Abbreviations: + = positive; - = negative.
procedure. In this regard, both two-dimensional echocardiography and gated blood pool scintigraphy are potentially suitable techniques for noninvasive, clinical evaluation of regional wall motion, but the associated expense and complexity of these techniques limit widespread applicability to diagnostic stress testing.

The results of this study suggest that cardiokymography may be particularly suited to the noninvasive diagnosis of coronary artery disease when used in conjunction with routine ECG stress testing. In this study, exercise-induced CKG abnormalities were at least marginally more sensitive and more specific than ST-segment depression for the diagnosis of coronary artery disease, and was most sensitive in detecting disease within the distribution of the left anterior descending coronary artery, consistent with our anterior transducer placement. In addition, the pattern of CKG response appeared to be related to severity of disease, as the majority of patients (eight of 12) with left main or equivalent disease had type III CKG responses. This diagnostic accuracy probably relates directly to the superior sensitivity and specificity of regional dysfunction as a marker of acute ischemia. Because of interference by respiratory motion, the CKG tracing is not obtainable during exercise without signal averaging. However, exercise-induced CKG abnormalities persisted longer during recovery than electrocardiographic changes and occurred more often than ST depression in patients who achieved less than adequate levels of exercise.

Although the CKG had a higher sensitivity and specificity than the ECG for detection of coronary artery disease in this study, this result may partly reflect selection bias in the population studied rather than major differences in diagnostic value. It is likely that the patients with normal coronary arteries who were referred for testing were biased toward those with a prior false-positive stress ECG. This possibility is supported by the lower ECG specificity in our population than that routinely observed in our laboratory or reported by others. The specificity of the CKG was significantly superior, however, compared with the higher specificity reported in the literature. Furthermore, the CKG had a significantly higher diagnostic accuracy in the low-prevalence subgroup with atypical chest pain (table 2). This is precisely the group with the lowest ECG predictive accuracy (in this study as well as in others) and yet with the greatest need for an accurate diagnosis. Patients with typical angina have a high prevalence of disease (90%), and therefore do not ordinarily require a stress test for a diagnosis.

Because the ECG and CKG detect different manifestations of regional myocardial ischemia (electrical and mechanical), the cause of false-positive and false-negative responses might differ. Thus, the diagnostic accuracy of stress testing was substantially improved by using cardiokymography in conjunction with electrocardiography. Despite the high rate of false-positive ECG stress tests, only one of the 79 normal subjects (52 angiographically normal, 27 apparently healthy volunteers) had concordantly false-positive ECG and CKG results. This concordant specificity substantially exceeds that of any single noninvasive diagnostic technique.

Although the sensitivity of concordant testing was only 46%, the high specificity imparted a predictive accuracy greater than that of either procedure alone and allowed identification of 94% of the false-positive ECG responders. Only 62% of the patients had such concordant test results, however, and the predictive accuracy of a discordant result was substantially lower (63%). This group might benefit most from additional, more extensive (and expensive) procedures such as thallium scintigraphy. We have had limited experience with this application. In 16 patients with discordant ECG and CKG test results, thallium exercise scintigraphy correctly identified the presence or absence of significant coronary artery disease in 11 patients (81%). This approach, therefore, may provide a cost-effective basis for serial diagnostic testing.

Limitations

The criteria used for the performance and interpretation of the CKG in this study were empirically based on the known effects of ischemia on regional left ventricular wall motion, modified by experience gained from applying the procedure during the investigation. Thus, the selection of an appropriate control tracing and the interpretation of test results requires training, experience and operator interaction similar to that of many other noninvasive techniques in which knowledge of typical patterns is applied not only to interpretation but also to the initial data collection.

Furthermore, an important limitation in the interpretation of the CKG signal relates to the complexity of cardiac motion, which the device records. The CKG recording probably represents a vectorial sum of the true motion of the segment just beneath the probe, as well as the translational motion of the entire heart and transmitted chest wall vibrations. The magnitude of contribution of each of these movements to the summed CKG recording undoubtedly varies with each person, with the physiologic state and with the precordial chest position from which the recording is obtained. Thus, a recording obtained over the cardiac apex (identical to a conventionally recorded ACG) often reveals outward systolic motion not readily distinguishable from the holosystolic outward motion associated with severe ischemia. This motion represents the upward lift of the cardiac apex toward the chest wall during systole and is not a regional contractile event. Chest wall motion appears to predominate relative to segmental wall motion at the point of maximal impulse, whereas segmental motion predominates at areas remote from the apex. Therefore, we avoid placing the probe over the cardiac apex.

Immediately after exercise, normal subjects may show a rapid inward systolic motion that terminates very early in systole and is followed by an outward motion during the major portion of ejection in areas
remote from the cardiac apex. This pattern has been observed with other techniques and has been attributed to the reduced cardiac volume, resistance to ejection and hyperdynamic contractile response associated with tachycardia in a healthy heart.46 After exercise, the ventricular volume may change significantly, and therefore the myocardial segment moving within the electromagnetic field may not be identical to the segment recorded during the control state, even if the CKG probe remains in an identical location on the chest wall. For example, a shift in the position of the apex at high rates could appear as an artificial development of outward motion and thus cause a false-positive response if the CKG probe was in a para-apical position. This may also apply to placement of the transducer over a normal zone immediately adjacent to a dysyncergenic segment.

The interpretive criteria developed in this study, therefore, should be viewed as a basis for broader application, which will more precisely define specific patterns representing the range of normality and severity of disease. In this regard, certain patterns similar to those observed with kinetocardiography appear typical of left ventricular hypertrophy and may be distinguishable from myocardial asynergy. Furthermore, tracings in the presence of both left or right bundle branch block remain normal in the absence of other associated disease (Silverberg RA, Diamond GA, Charuzi Y, Vas R, Tzivoni D, Berman D, Forrester JS: unpublished data). Thus, the technique is potentially applicable to stress tests in patients with abnormalities that limit the value of electrocardiographic monitoring.1

In summary, the CKG produces a complex analog wave form that, although not fully understood, correlates with dynamic changes in regional left ventricular mechanical function. The data in this study suggest that the CKG is a useful noninvasive technique applicable to routine treadmill exercise testing where its use as an adjunct to electrocardiography may improve the overall accuracy of the procedure.

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References

28. Ascoop CA, Simoons ML, Egmne WG, Bruschke AV: Ex-
exercise test, history and serum lipid levels in patients with chest pain and normal electrocardiogram at rest: comparison to findings at coronary arteriography. Am Heart J 82: 609, 1971
45. Rushmer RF: Cardiovascular Dynamics. Philadelphia, WB Saunders, 1970
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R A Silverberg, G A Diamond, R Vas, D Tzivoni, H J Swan and J S Forrester

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