Noninvasive Detection of Periinduction Ischemic Ventricular Dysfunction by Cardiokymography in Humans: Preliminary Experience

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Regional wall motion analysis provides a sensitive and selective means for detection of myocardial ischemia. 1-4 Animal experiments have demonstrated that anesthetic maneuvers may produce regional ventricular dysfunction limited to an area supplied by a critically constricted coronary artery 5 and that the dysfunction is correlated with decreases in blood flow to the affected region of the ventricle. 6 Furthermore, ischemia is not detected reliably by electrocardiographic or hemodynamic measurements, particularly if the ischemia is limited to a modest region of the ventricle. 7 Regional ischemic dysfunction following endotracheal intubation in humans with CAD (coronary artery disease) previously has been documented by mode echocardiography. 8

Most clinical methods of estimating regional wall motion are either complex (such as echocardiography), involve highly invasive techniques (such as ventriculography), or require radioisotopes (such as scintigraphy). The cardiokymograph (CKG) is a noninvasive electromagnetic device providing an easily interpretable recording of anterior left ventricular wall motion in the closed chest. 8-12 It does not emit radiation or require the use of radioisotopes.

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The purpose of this study was to establish in humans with CAD whether cardiokymography would detect wall motion abnormalities during anesthesia, before or in the absence of electrocardiographic evidence of ischemia. We studied two groups of patients in the periinduction period, one with angiographically documented CAD and a second group free of evidence of ischemic heart disease, in order to document the incidence of abnormal CKG recordings during anesthesia in humans without CAD.

METHODS

Patients were studied after giving informed consent to a protocol approved by the Subcommittee of Human Studies. Group 1 consisted of 24 patients with angiographically proven left anterior descending (LAD) CAD and normal or moderately impaired anterior left ventricular wall motion as documented by contrast ventriculography, in whom a normal CKG (i.e., Type I; vide infra) could be obtained when awake. All patients were scheduled for coronary artery bypass graft surgery and monitored by our standard practices, including ECG leads II and V5, continuous intraarterial blood pressure, central venous pressure, and pulmonary artery pressure recording and intermittent pulmonary capillary wedge pressure measurements. No attempt was made to influence anesthetic selection or management. Group 2 consisted of 25 ASA Class 1 patients scheduled to undergo dental extraction under general endotracheal anesthesia, in whom a normal CKG could be obtained while they were awake. All Group 2 patients received thiopental for induction of anesthesia and succinylcholine for facilitation of endotracheal intubation. Monitoring in Group 2 consisted of a single precordial electrographic lead, blood pressure determination by cuff and Korotkoff sounds, and heart rate derived from the ECG.

In both groups, cardiokymograms were obtained prior to induction of anesthesia, after induction of anesthesia, and 1–3 min after endotracheal intubation. In no case was the responsible anesthetist a member of the research team.

The Cardiokymograph. The cardiokymograph 8-10 uses a transducer consisting of a 5-cm circular flat capacitive plate as part of a high-frequency (10 MHz) low-power (10 mW) 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/cm²) electromagnetic field (1.5 amps/m). The electromagnetic field can penetrate totally through the body. Motion within the field causes a change in effective capacitance and produces a change in the frequency of the oscillation. This change in frequency is converted into a change in voltage proportional to the original motion and amplified and recorded as an analog signal. The frequency response of the system is flat ±10% from 0.1 to 90 Hz, with a phase shift of ±20° at these frequency extremes. The cardiokymograph is highly focal and can detect motion only with a vectorial component perpendicular to and directly under its coil. Therefore, motion on the surface parallel to the coil, or perpendicular to the coil but beyond its outer extent, produces no output. In clinical use, the cardiokymograph records both anterior heart wall motion and chest wall motion. Chest wall motion is minimized by the configuration of the transducer holder and by recording kymograms during apnea at end-expiration. Since the output of the cardiokymograph decreases exponentially with the distance from the moving surface, the analog signal detects primarily the anterior left ventricular wall motion when the transducer is placed appropriately. The electronic features of the cardiokymograph have been reported in detail, and validation of its ability to record anterior left ventricular wall motion has been performed extensively in open- and closed-chest animals and humans with and without ischemic heart disease.

The analog tracing of the CKG resembles that derived from pairs of piezoelectric signals implanted in the ventricular wall (sonomicrography) with several additional deflections, which are caused by movements of the whole heart in relation to the chest wall (fig. 1). There are small inward and outward motions during isovolumic systole. With onset of ventricular ejection, there is an initial outward motion peaking at the “11” point, probably representing a changing contour of the heart and rotation of the apex. The CKG events affected by myocardial oxygen balance occur primarily during ejection following the “11” point. A normal CKG ("Type I") demonstrates predominantly inward motion during ejection, without systolic outward motion. A CKG demonstrating dyssnergy ("Type II") displays a reduced amplitude of systolic inward motion, with the maximum inward motion occurring during the first half of systole, and with significant paradoxical outward motion. A dyskinetic CKG ("Type III") is characterized by holosystolic outward motion and lack of systolic inward motion. Abnormal CKGs are, therefore, analogous to the sonomicographic tracings described for moderate ("Type II") and severe ("Type III") ischemia in our previous animal experiments.

**RESULTS**

In Group 1, eight of 24 patients (33%) demonstrated abnormal wall motion at some time during the periinduction period. A representative example is shown in figure 2. This patient demonstrated transient early systolic
outward motion 1 min after endotracheal intubation, despite having what usually would be considered well-controlled hemodynamics and an unchanged ECG. The eight patients demonstrating positive CKGs were managed by a variety of anesthetic techniques considered acceptable for patients with CAD. Table 1 presents the ECG and hemodynamic data at the time ischemia first was detected and the PCW values prior to anesthesia. Only one patient demonstrated change in the ST segment > 1 mV suggestive of myocardial ischemia. Only three of the eight patients, including the one with ECC changes, sustained elevation of PCW > 4 mmHg, and only two had absolute PCW values of greater than 15 mmHg.

In contrast to the patients of Group 1, only one of 25 patients (4%) without overt coronary artery disease demonstrated an abnormal CKG recording. The difference in incidence of abnormal perinduction cardiokymograms between the groups is highly statistically significant (chi-square test: P < .01). The hemodynamic data from Group 2 is presented in Table 2. Sixteen of 25 patients had heart rates greater than 100 beats/min, and 15 had systolic blood pressure of 140 mmHg or greater. Thus, they failed to demonstrate abnormal wall motion, despite poorly controlled hemodynamics.

**Discussion**

This study documents a high incidence of abnormal cardiokymograms during the perinduction period in patients with angiographically documented left anterior descending coronary artery disease, despite anesthetic management employing invasive hemodynamic monitoring and, in most cases, hemodynamics and ECC not suggestive of myocardial ischemia. In contrast, abnormal cardiokymograms are unusual in patients without evidence of coronary artery disease undergoing anesthesia, despite a high incidence of hemodynamic changes associated with increased myocardial oxygen demand. This suggests that the CKG provides a noninvasive method for early detection of myocardial ischemia during anesthesia.

The cardiokymograph previously has been evaluated extensively and validated as a monitor of anterior left ventricular wall motion. For instance, in the open-chested dog, the CKG was shown to correlate closely with recordings from epicardial mercury and silastic length gauges. In studies of patients with CAD subjected to atrial pacing or treadmill stress testing, the CKG has been shown to be both more sensitive and more specific an indicator of the presence of coronary artery disease as documented by coronary angiography than the electrocardiogram.

Ventricular wall motion during induction of anesthesia and endotracheal intubation in patients with CAD has been studied previously by Elliott et al. using M-mode echocardiography. Ten of 24 patients demonstrated abnormal wall motion, interpreted as ischemic dysfunction, whereas the ECG was indicative of ischemia in only one. These results are strikingly similar to those we observed using the CKG. The incidence of ischemia is also similar to that reported by Wilkinson et al., using myocardial lactate production and ECC changes as criteria. Previous work by others in children and in adult patients without heart disease has shown that inhalation anesthesia is associated with depression of wall motion but not with changes indicative of ischemia.

Giles et al. demonstrated that left ventricular ejection fraction estimated by the computerized nuclear probe (nuclear stethoscope) is decreased following laryngoscopy and endotracheal intubation in patients both with and without ischemic heart disease. The duration of decreased ejection fraction is significantly greater in patients with ischemic heart disease than in patients without heart disease. The authors note that the delayed recovery of ventricular function may indicate either myocardial ischemic damage or altered ventricular reserve following afterload stress in the absence of ischemia. Thus, this method of monitoring, besides requiring radioactivity, may not differentiate reliably between myocardial ischaemic values.

**Table 1. Hemodynamic Values and Electrocardiogram (ECG) When Abnormal Cardiokymograph (CKG) First Was Recorded* in Eight Patients with Coronary Artery Disease**

<table>
<thead>
<tr>
<th>Pulmonary Arterial Wedge Pressure (mmHg)</th>
<th>ECG Depression (mm)</th>
<th>Heart Rate (beats/Min)</th>
<th>Systolic/Diastolic (mmHg)</th>
<th>Mean (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 (10)†</td>
<td>0</td>
<td>77</td>
<td>125/70</td>
<td>85</td>
</tr>
<tr>
<td>11 (8)†</td>
<td>0</td>
<td>72</td>
<td>110/70</td>
<td>80</td>
</tr>
<tr>
<td>28 (7)†</td>
<td>1</td>
<td>58</td>
<td>200/100</td>
<td>130</td>
</tr>
<tr>
<td>12 (5)†</td>
<td>0</td>
<td>80</td>
<td>140/85</td>
<td>100</td>
</tr>
<tr>
<td>22 (8)†</td>
<td>0</td>
<td>50</td>
<td>90/55</td>
<td>60</td>
</tr>
<tr>
<td>10 (10)†</td>
<td>0</td>
<td>57</td>
<td>145/75</td>
<td>95</td>
</tr>
<tr>
<td>6 (4)†</td>
<td>0</td>
<td>130</td>
<td>190/95</td>
<td>125</td>
</tr>
<tr>
<td>11 (11)†</td>
<td>0</td>
<td>65</td>
<td>100/60</td>
<td>60</td>
</tr>
</tbody>
</table>

* Since the CKG was recorded intermittently, onset of abnormalities may have occurred sooner.
† PCW values prior to anesthetic induction.

**Table 2. Perinduction Hemodynamic Values in 25 ASA Class I Patients* (Group 2)**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>(beats/min)</td>
<td>(mmHg)</td>
<td>(mmHg)</td>
</tr>
<tr>
<td>80 ± 16</td>
<td>118 ± 13</td>
<td>71 ± 10</td>
</tr>
<tr>
<td>87 ± 16</td>
<td>108 ± 11</td>
<td>66 ± 7</td>
</tr>
<tr>
<td>103 ± 19</td>
<td>137 ± 12</td>
<td>85 ± 15</td>
</tr>
<tr>
<td>100 ± 18</td>
<td>129 ± 12</td>
<td>79 ± 12</td>
</tr>
</tbody>
</table>

* Mean ± SD.
emia and myocardial depression. In contrast, the CKG, which examines wall motion, appears to have a high specificity for ischemia.

Our 4% incidence (i.e., 1 out of 25) of abnormal CKGs in patients without coronary artery disease is consistent with the literature.\textsuperscript{1,11,12} This may represent an episode of ischemia in a patient with undiagnosed coronary artery disease or a false positive. The CKG will generate a tracing mimicking abnormal ventricular wall motion if the transducer is located above the cardiac apex or atria, thus detecting apical or atrial motion. In our case we hypothesize that the single abnormal CKG in Group 2 was due to detection of apical motion when the heart size decreased with the tachycardia accompanying endotracheal intubation. However, we cannot rule out other causes.

One disadvantage of the cardioiomyogram is the inability to record during respiration, since this causes the transducer position to move relative to the position of the heart. In awake patients, this problem is overcome by recording during voluntary breath holding at end expiration. In patients undergoing anesthesia with controlled ventilation, provision of sufficient apnea to record three beats of the CKG poses no problem. Another disadvantage is the difficulty in locating the transducer position above the anterior left ventricle. In our experience, proper transducer placement, as judged by obtaining a normal CKG recording, usually takes less than 2 min after an individual has used the instrument in 25 subjects. Inability to use this technique in patients with abnormal anterior left ventricular wall motion is another disadvantage, since this may rule out this type of monitoring for many individuals with previous myocardial infarction. The ability to record only anterior heart wall motion is a further disadvantage. Cardiomyography is insensitive to changes in motion of the inferior, posterior, and lateral left ventricular walls. However, the left anterior descending coronary artery, which supplies the anterior left ventricular wall, is the most commonly diseased coronary artery. Advantages of the CKG include safety, relative simplicity of operation, ease of interpretation, and its noninvasive nature.

We made no attempt to control or unify the type of anesthetic being administered in patients with CAD, since our purpose was to determine whether abnormal wall motion would be detected by cardiomyography, rather than to compare different strategies of anesthetic management. Our study suggests that the CKG provides a sensitive, noninvasive method for early detection of anterior left ventricular wall ischemia during anesthesia and that the incidence of false-positive recordings is small. We believe the CKG warrants serious consideration as a monitor of myocardial ischemia in clinical anesthetic practice.

References

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