Qualitative Evaluation of Coronary Flow during Anesthetic Induction Using Thallium-201 Perfusion Scans

Bruce Kleinman, M.D.,* Robert E. Henkin, M.D.,† Silas N. Glisson, Ph.D.,‡ Adel A. El-Etr, M.D.,§ MAMDouh Bakhos, M.D.,‡ Henry J. Sullivan, M.D.,** Alvaro Montoya, M.D.,†† Roque Pifarre, M.D.‡‡

Qualitative distribution of coronary flow using thallium-201 perfusion scans immediately postintubation was studied in 22 patients scheduled for elective coronary artery bypass surgery. Ten patients received a thiopental (4 mg/kg) and halothane induction. Twelve patients received a fentanyl (100 μg/kg) induction. Baseline thallium-201 perfusion scans were performed 24 h prior to surgery. These scans were compared with the scans performed postintubation. A thallium-positive scan was accepted as evidence of relative hypoperfusion. Baseline hemodynamic and ECG data were obtained prior to induction of anesthesia. These data were compared with the data obtained postintubation. Ten patients developed postintubation thallium-perfusion scan defects (thallium-positive scan), even though there was no statistical difference between their baseline hemodynamics and hemodynamics at the time of intubation. There was no difference in the incidence of thallium-positive scans between those patients anesthetized by fentanyl and those patients anesthetized with thiopental-halothane. The authors conclude that relative hypoperfusion, and possibly ischemia, occurred in 45% of patients studied, despite stable hemodynamics, and that the incidence of these events was the same with two different anesthetic techniques. (Key words: Anesthetics, intravenous: fentanyl. Anesthetics, volatile: halothane. Heart: coronary flow; ischemia; perfusion scans.)

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THE PERIOD of anesthetic induction in patients with coronary artery disease is associated with an increased risk of myocardial ischemia. The anesthesiologist is faced with the problems of deciding what monitoring devices to use to detect signs of early ischemia and what anesthetic agent to choose to reduce the incidence of ischemia. Previous studies have shown that ischemic events based on ECG criteria occur with some frequency during the induction of anesthesia.1 The period during which intubation is performed is particularly a high-risk interval.1,2

Although ECG, heart rate, and pulmonary capillary wedge pressure changes typically are used to identify ischemic events, the effectiveness of these variables in detecting early onset ischemic events is not fully known.

Nonhomogenous uptake of thallium-201 by the myocardium appearing as a defect on the scintigram means a decrease in relative perfusion of the myocardium. This has been shown to occur in the setting of acute ischemia, acute infarction, or scar formation.3 Serial scanning enables one to distinguish whether a defect is permanent and, therefore, due to scar or myocardial infarction or transient.4 Using serial thallium-201 perfusion scans, this study was designed to investigate whether there is a difference in the incidence of perfusion defects during anesthetic induction with fentanyl or halothane, and whether standard monitoring techniques such as V5 ECG tracings and pulmonary capillary wedge pressure measurements predict or detect these events during anesthetic induction and intubation.

Methods

SUBJECTS

After approval from the Institutional Review Board, informed consent was obtained from 22 patients scheduled for elective coronary artery bypass surgery. The patients were in good health aside from their coronary artery disease, and had hemoglobin levels ranging from 13 to 15.4 g·dl−1. All of the patients had significant coronary artery disease, as defined by obstruction of at least 75% or greater of one or more major coronary arteries. All patients also had significant obstructive disease in the left anterior descending coronary artery. No patient had obstructive disease of the left main coronary artery. All patients had normal left ventricular function, as defined by the ejection fraction calculated from the left ventricular angiogram in the right anterior oblique projection at the time of catheterization. The mean ejection fraction was 0.72 (range 0.65–0.89). No chronic medications were discontinued before surgery, and all patients received their medications on the morning of surgery. All patients

* Assistant Professor of Anesthesiology and Medicine.
† Professor of Radiology; Director of the Department of Nuclear Medicine.
‡ Associate Professor of Anesthesiology and Pharmacology.
§ Professor of Anesthesiology; Chairman, Department of Anesthesiology.
†† Associate Professor of Surgery, Division of Cardiovascular Surgery.
** Associate Professor of Surgery, Division of Cardiovascular Surgery.
‡‡ Associate Professor of Surgery, Division of Cardiovascular Surgery.
††† Professor of Surgery; Chairman, Division of Cardiovascular Surgery.

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Address reprint requests to Dr. Kleinman.

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TABLE 1. Study Group Demography Comparing Those Patients Receiving a Halothane induction versus those Receiving Fentanyl

<table>
<thead>
<tr>
<th></th>
<th>Halothane</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Females</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>56.5</td>
<td>55.4</td>
</tr>
<tr>
<td>Vessel Involvement (&gt;75% obstruction)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Vessel</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2 Vessel</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>3 Vessel</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrates</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Beta blockers and calcium channel blockers</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stable angina</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>Prior infarction</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>+ Stress test</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

were premedicated with 10 mg morphine intramuscularly prior to their arrival to the operating room. Further characteristics of the study group are seen in table 1.

SCINTIGRAPHY

Thallium scan studies were performed with either a Siemens LISA® 7-inch field-of-view camera or an El Scint Apex 215M® with an 8-inch field of view. Preoperative, postintubation, and postoperative scanning studies were performed on the same imaging device in the majority of patients. All scans were obtained within 5 min of injection of thallium-201 (NEN®-Dupont).

Preoperative thallium scans were obtained with a dose of 2 mCi of 201 thallous chloride. Anterior, 45-degree left anterior oblique, 60-degree left anterior oblique, and left lateral views were obtained. These views all contained an excess of 250,000 counts/view and approximated 1,000 counts/cm² over the myocardium.

Two mCi of thallous chloride was injected during intubation. Scanning was begun 5 min later. Technically comparable views to the preoperative studies were obtained in the anterior, 45-degree, and 60-degree left anterior oblique projections.

Postoperative scans were performed similarly to the preoperative scans prior to discharge from the hospital. For all scans, the mercury x-rays of 201 thallium were imagined (69–80 KEV), as well as the photo peak at approximately 169 KEV. Dual analyzers were used.

Data were analyzed in either the El Scint® or Medical Data Systems® computers using an automated-analysis program. Review of the results at the conclusion of the project indicated that the automated-analysis program did not achieve any better results in identifying perfusion defects than did simple visual interpretation.

All images were photographed on transparencies or Polaroid® film for display. Interpretation was by a single experienced observer (R.H.), without knowledge of scan sequence or of the catheterization or operative results. Films were interpreted on two different occasions separated by 2 months to assure consistency of interpretation.

DESIGN AND PROCEDURES

Due to the 7 h needed to effect thallium-201 clearance from the myocardium, baseline thallium scans were done the day before surgery. Prior to induction of anesthesia, an arterial cannula and pulmonary artery catheter (#7 F American-Edwards®) were inserted into all patients. The V5 ECG lead was continuously monitored and recorded during sampling periods (Gould 2400® ink brush recorder). Hemodynamic measurements, which included heart rate, arterial blood pressure, and pulmonary capillary wedge pressure were recorded (Gould 2400® ink brush recorder). All pressures were transduced with a TXX Gould® disposable transducer and connected to the patient with no more than 4 feet of arterial tubing. Resonant frequency has been determined to range between 20–25 Hz. Cardiac outputs were measured by thermodilution prior to induction and immediately after injection of thallium. The average of three measurements was taken.

Anesthetic induction was achieved in 12 patients using fentanyl (100 μg/kg), 100% O₂, and pancuronium (0.15 mg/kg) in the following manner: one-half the total fentanyl dose was infused over a period of 10 min and the other half was injected slowly over a period of 3–5 min. Prior to infusing the fentanyl, 1 mg of pancuronium was injected into a peripheral iv. The rest of the pancuronium was injected slowly, as the fentanyl was infusing, over a period of 3–5 min. When the patient lost consciousness, ventilation was controlled with bag and mask at 100% F₃O₂. Ten patients received a thiopental, halothane–O₂ induction in the following manner: thiopental was infused into a central vein over a period of 5–8 min for a total dose of 4 mg/kg. When the patient lost consciousness, the halothane vaporizer was turned on and the patient was ventilated with oxygen and gradually increasing concentrations of halothane. The end-expiratory halothane concentration was measured using a mass spectrometer (Perkins-Elmer®) and ranged between 1–1.5%. During this time, pancuronium (0.15 mg/kg) was slowly injected. When the attending anesthesiologist deemed the patient fully anesthetized and stable, the patient was intubated. Whether a patient underwent a fentanyl or halothane induction depended on the time constraints and operating
room scheduling on the day of surgery, because halothane inductions took a longer period of time.

During induction, patients were manually ventilated with bag and mask to maintain normocarbia. Arterial oxygen partial pressure ranged between 283–502 mmHg. During intubation, hemodynamic variables were remeasured and recorded. Thallium-201 was injected into a central vein when the endotracheal tube was placed in the trachea. Scanning was begun shortly thereafter. Six to seven days after surgery, follow-up scans were obtained on all but two patients.

The attending anesthesiologist (who was not an investigator) was instructed to anesthetize the patient as he normally would. If he had to use a vasodilator such as nitroglycerin or an inotropic such as dobutamine because of the monitored hemodynamics, he was advised to do so. If this occurred, an attempt would be made to inject the thallium prior to the initiation of these agents.

**Statistical Analysis**

In comparing baseline hemodynamic and ECG measurements to the measurements during intubation for each anesthetic group, a paired t-test was performed. Within each anesthetic group, a nonpaired, t-test was also performed in comparing baseline hemodynamic and ECG measurements to the measurements during intubation between patients with a thallium-positive versus those patients with a thallium-negative scan. When measurements were compared between all patients with a thallium-negative scan during intubation to all patients with a thallium-positive scan at the time of intubation, a nonpaired t-test was used. For both tests, a probability of <0.05 was considered significant. Fisher's exact test was performed to analyze the frequency of relative hypoperfusion events (thallium-positive scans) between the two anesthetic groups. All values are expressed as mean ± SEM.

**Results**

In the left anterior oblique (LAO) projection, one is able to visualize parts of the septal wall, apex, inferior wall, and posterolateral wall (fig. 1). A normal scan would show uniform uptake of tracer. An example of a perfusion defect is shown in figure 2.

Five of the 10 halothane patients had abnormal thallium scans immediately after intubation. Four of these patients had normal preoperative scans. One patient had a septal perfusion defect preoperatively and developed an additional defect in the posterolateral and inferior walls at the time of intubation. Four of the five patients had normal postoperative scans. One patient with a normal preoperative scan developed a septal perfusion defect postintubation and maintained that defect postoperatively (table 2).

Five of the 12 fentanyl patients had abnormal thallium scans immediately after intubation. Four of these had normal preoperative scans. One had a preoperative inferior perfusion defect that became much larger during intubation. Three patients with abnormal scans following intubation had normal scans postoperatively. One patient with a septal perfusion defect postintubation developed a posterolateral perfusion defect postoperatively. One patient with an apical perfusion defect postintubation refused follow-up scanning (table 2). One patient had a normal preoperative and postintubation scan, but developed an abnormal postoperative scan (apical perfusion defect).

Overall, ten of 22 patients (45%) developed perfusion defects, which were not present preoperatively, following intubation. Two of these had perfusion defects postoperatively. One patient refused follow-up study. There was no significant difference between anesthetic groups ($P = 0.75$, Fisher's exact test).

The hemodynamic and ECG data showed that, among the ten patients who developed postintubation scan defects, no difference existed between measurements taken prior to induction of anesthesia and those same measurements during intubation (table 3). This was so whether a patient was anesthetized by halothane or fentanyl (table 4).

Those patients with thallium-negative scans did show statistical differences in heart rate, cardiac index, and rate–pressure product between baseline and intubation,
whether examined as a single group (table 3) or according to anesthetic (table 4).

Measurements taken during intubation showed no difference in mean pulmonary capillary wedge pressure, double product, heart rate, cardiac index, diastolic and systolic pressures, or ST segments between those patients with a positive thallium scan versus those with a negative scan (table 4). One patient with a normal preoperative and postintubation scan had ST-segment depression during induction with fentanyl and developed an apical defect postoperatively. Nitroglycerin administration resulted in rapid normalization of the ST segments. In this patient, injection of thallium occurred during intubation when the ST segments had already normalized. This was the only patient in whom a cardioactive drug was administered during induction.

Only one patient had ECG and enzyme evidence for a perioperative myocardial infarction. This patient had a normal preoperative scan, but developed a septal perfusion defect immediately after intubation. Postoperatively, the patient had a persistent posterolateral perfusion defect and resolution of the septal defect.

Discussion

The clinician typically uses ECG, heart rate, or capillary wedge pressure change as markers of myocardial ischemia. Whether these variables can detect early-onset imbalance in the oxygen supply/demand ratio has not been adequately demonstrated.

This study measured electrocardiographic and hemodynamic variables before and during halothane or fen-
TABLE 2. Patients with Abnormal Thallium Scans Immediately Postintubation

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>Intubation</th>
<th>Postop</th>
<th>Site of Coronary Lesions*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane n = 5</td>
<td>NL</td>
<td>Septal</td>
<td>NL</td>
<td>LAD, LCX</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Septal</td>
<td>Septal</td>
<td>LAD, LCX, RCA</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Posterior</td>
<td>NL</td>
<td>LAD</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Septal</td>
<td>NL</td>
<td>LAD, LCX, RCA</td>
</tr>
<tr>
<td></td>
<td>Septal</td>
<td>Posterior</td>
<td>NL</td>
<td>LAD, LCX, RCA</td>
</tr>
<tr>
<td>Fentanyl n = 5</td>
<td>Inferior</td>
<td>Inferolateral</td>
<td>NL</td>
<td>LAD, LCX</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Septal</td>
<td>NL</td>
<td>LAD</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Apical</td>
<td>_</td>
<td>LAD, LCX, RCA</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Inferoapical</td>
<td>NL</td>
<td>LAD, RCA</td>
</tr>
<tr>
<td></td>
<td>NL</td>
<td>Septal</td>
<td>Posterior</td>
<td>LAD, RCA</td>
</tr>
</tbody>
</table>

Abbreviations: NL, normal; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery.

* Anatomic locations of obstructive lesions identified by angiography are listed for comparison to the scintigraphic results.

tanyl anesthetic induction and compared the results with evidence of myocardial perfusion homogeneity obtained concomitantly using thallium-201 scans.

Thallium-201 is a monovalent potassium analog that is rapidly cleared from the blood by many organs of the body, including the myocardium. Thallium-201 is a perfusion marker and not a metabolic one. Therefore, the regional concentration of thallium-201 in the myocardium is largely related to its regional perfusion.4,5 The myocardial half-life of thallium is 7 h.6 Only 5% of the total injected thallium dose is extracted by the myocardium. The initial distribution of thallium-201 in the myocardium depends on the perfusion at the time of injection.6 The scan results only determine the homogeneity of myocardial perfusion, identifying those areas of relative hypoperfusion. It is possible that areas interpreted to reflect hypoperfusion may in fact represent areas of normal flow surrounded by areas of supernormal flow. Identified areas of hypoperfusion may or may not be associated with ischemia. However, myocardial perfusion defects have been demonstrated during acute ischemia and myocardial infarction.5

Nonhomogenous uptake of thallium-201 by the myocardium is interpreted by the authors to indicate areas of relative hypoperfusion, not supernormal flow. Halothane has been shown to be a weak coronary vasodilator.7 However, studies in humans demonstrate that coronary flow decreases with halothane in accordance with its effects on the indices of myocardial oxygen demand.8,9 It has also been shown with high-dose fentanyl that, as myocardial oxygen consumption decreases, myocardial blood flow also decreases.10 Therefore, it would be unlikely that nonhomogeneity of tracer uptake is due to supernormal flow in normal coronary arteries.

The metabolic needs of the heart are closely linked to its blood flow, because the resting myocardium extracts such a high percentage of oxygen from its arterial blood that there is virtually no venous oxygen reserve. The only way the myocardium can meet increased oxygen demands is to increase its blood flow. A consequence of this fact is that, if myocardial oxygen demands remain the same, decreased blood flow may be associated with ischemia. This is why thallium scans that show areas of decreased uptake are considered to represent potential areas of ischemia.11

TABLE 3. Comparison of Baseline Hemodynamics to Hemodynamics at Intubation Between Thallium-positive and Thallium-negative Patients*

<table>
<thead>
<tr>
<th></th>
<th>Thallium + (n = 10)</th>
<th>Thallium – (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Intubation†</td>
</tr>
<tr>
<td>PCW mmHg</td>
<td>11 ± 1.0</td>
<td>13 ± 1.0</td>
</tr>
<tr>
<td>HR beats·min⁻¹</td>
<td>66 ± 3.0</td>
<td>71 ± 4</td>
</tr>
<tr>
<td>CI l·min⁻¹·m⁻²</td>
<td>2.8 ± 0.2</td>
<td>3.1 ± 0.1</td>
</tr>
<tr>
<td>SBP mmHg</td>
<td>133 ± 6.0</td>
<td>128 ± 8.0</td>
</tr>
<tr>
<td>DBP mmHg</td>
<td>72 ± 3.0</td>
<td>76 ± 5.0</td>
</tr>
<tr>
<td>RPP beats·min⁻¹·mmHg</td>
<td>8770 ± 559</td>
<td>9186 ± 841</td>
</tr>
<tr>
<td>V₅</td>
<td>NL</td>
<td>NL</td>
</tr>
</tbody>
</table>

Abbreviations: PCW, pulmonary capillary wedge pressure; HR, heart rate; CI, cardiac index, SBP, systolic blood pressure; DBP, diastolic blood pressure RPP, rate pressure product; NL, normal. Statistical probabilities comparing baseline to intubation for each thallium group are shown. NS, nonsignificant.

† Time of scan: immediately postintubation.

* Time of scan: immediately postintubation.

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Areas of decreased myocardial uptake of thallium-201, representing possible ischemia but not necrotic myocardium, will appear to be normal after a relatively short period of time. This is true even if the area in question remains hypoperfused. This is known as the phenomenon of thallium redistribution. It is for this reason, following injection of thallium, that scanning must be done shortly thereafter.

Although the patient groups were small, there was no difference in the incidence of perfusion defects between those patients anesthetized with thiopental and halothane versus those with fentanyl. This is consistent with the results of one study that compared halothane versus morphine anesthesia, wherein no difference in myocardial oxygen balance was found. However, one study showing ischemia during induction with halothane and nitrous oxide has been published.

It has been reported that monitoring pulmonary capillary wedge pressures can detect early ischemia. However, no significant difference in pulmonary capillary wedge pressure measurements existed between the thallium-positive patients and the thallium-negative patients in our study. The pulmonary capillary wedge pressure measurements in our study are considerably less than those reporting the relationship of ischemia with elevated pulmonary capillary wedge pressures. Our data imply that either normal wedge pressure does not rule out early ischemic events, or the patients were not ischemic, despite the perfusion defects.

Studies in dogs suggest that, as blood flow is progressively reduced, the first sign of ischemia is metabolic (i.e., regional lactate production). Wall-motion abnormalities appear with further reduction of blood flow. It is only with severe depression of coronary blood flow that global left-ventricular dysfunction occurs. Ischemic regional ventricular dysfunction may occur in dogs despite near-normal systemic hemodynamics and normal global left-ventricular function. Studies with two-dimensional, transesophageal echocardiography in patients undergoing supraceliac aortic occlusion for resection of abdominal aortic aneurysms showed left-ventricular wall motion abnormalities despite pulmonary artery wedge pressures in the normal range. Therefore, it should not be surprising that indices that measure global myocardial function, such as the pulmonary capillary wedge pressure measurement, may not detect early ischemia because this is a regional occurrence.

Standard and precordial ECG leads are reported not to identify early ischemia. Monitoring of the V5 ECG lead in our investigation also did not correlate with onset of perfusion-scan defects. None of the patients who developed an abnormal postintubation thallium scan developed ST-segment changes suggestive of ischemia either at or before intubation. One patient did develop ST-segment depression, while showing a normal postintubation scan. This presumably was due to the fact that a nitroglycerin infusion was started prior to the injection of thallium. By the time the thallium was given, ST segments had normalized with the resultant normal scan. Because we are dealing with regional disease and regional perfusion defects, it is not surprising that V5 monitoring was insensitive. ST-segment changes due to ischemia of the
 inferior wall will be evident in leads II, III, and AVF, but not $V_{5}$. Septal events that normally would be detected with leads V1 or V2 may not be detected by lead $V_{6}$. Out of the ten patients with abnormal thallium scans at the time of intubation, five had septal perfusion defects, while two had perfusion defects involving the inferior wall (table 2). Monitoring additional leads, such as lead II, may be helpful.

Most of our patients had rate-pressure products less than the suggested critical level of 12,000 beats·min$^{-1}$ · mmHg. Despite this fact, ten patients developed abnormal scans immediately after intubation. Assuming the abnormal scans reflected ischemia, this could be due to one of two reasons: either ischemia may occur even in the face of controlling the indices of myocardial oxygen demands, or the rate-pressure product does not reflect myocardial oxygen demands under anesthesia. There is much evidence in support of the latter.

In the ten patients with thallium-positive scans, heart rate and systolic pressure did not change significantly during intubation. This suggests that, despite fairly constant myocardial oxygen demand, a high percentage of patients may develop ischemia. This finding may reflect the fact that one can have ischemia due, not to changes in myocardial oxygen demand, but to changes in oxygen supply.

Statistically significant changes in heart rate, cardiac index, and rate-pressure product were seen in those patients with thallium-negative scans. From a clinical point of view, the changes in heart rate were not large. The cardiac-index change was due to the increase in heart rate. The fact that the change in rate-pressure product was significant in this group of patients is further evidence for its lack of predictive value during anesthesia.

One might question whether the mild vasodilator activity of halothane contributed to the relative hypoperfusion observed. The role of vasodilators in reducing flow was addressed in an interesting study in swine using regional lactate production as the marker for ischemia. The results showed that a vasodilator such as adenosine will cause a decrease in flow to an area of myocardium supplied by a stenosed coronary artery. This decrease in flow is associated with regional lactate production, i.e., ischemia. However, if the experiment is repeated using a vasodilator such as nifedipine that also decreases myocardial oxygen demand via its negative inotropic effect, then the decreased flow will not be associated with lactate production. The decrease in myocardial oxygen consumption with nifedipine was presumed to be greater than the decrease in myocardial blood flow. Therefore, there was no ischemia. Halothane is a mild coronary artery vasodilator that can decrease myocardial oxygen demand through its negative inotropic effects. Therefore, it is possible, at least in those patients anesthetized with halothane, that the abnormal thallium scans merely represent perturbations in flow without ischemia. It is possible that the flow abnormalities started well before induction. It has also been demonstrated that transient defects can occur on resting thallium scans in patients without other evidence of an acute ischemic process or previous myocardial infarction.

Two out of ten patients with abnormal postintubation scans had abnormal postoperative scans. One of the two had ECG and enzyme evidence of perioperative myocardial infarction. The significance of the defect in the other patient is unknown. Without further scanning, we cannot tell whether the defect was transient, perhaps an ischemic episode, or permanent and, therefore, representing a myocardial infarction.

In summary, abnormal thallium scans performed immediately after intubation were noted in 45% of patients scheduled for elective coronary artery bypass surgery. Differences in anesthetic agents did not affect this incidence. Evidence of myocardial hypoperfusion occurred despite no overall significant changes in heart rate, systolic or diastolic blood pressure, rate-pressure product, mean pulmonary capillary wedge pressure, cardiac index, and $V_{5}$ ECG lead trace. This suggests, but does not prove, transient ischemia in the presence of stable hemodynamics. Further studies with metabolic scintigraphic markers will be needed to prove whether this is, in fact, ischemia.

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References


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