AHA Scientific Statement

Practice Standards for Electrocardiographic Monitoring in Hospital Settings

An American Heart Association Scientific Statement From the Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young

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Abstract—The goals of electrocardiographic (ECG) monitoring in hospital settings have expanded from simple heart rate and basic rhythm determination to the diagnosis of complex arrhythmias, myocardial ischemia, and prolonged QT interval. Whereas computerized arrhythmia analysis is automatic in cardiac monitoring systems, computerized ST-segment ischemia analysis is available only in newer-generation monitors, and computerized QT-interval monitoring is currently unavailable. Even in hospitals with ST-monitoring capability, ischemia monitoring is vastly underutilized by healthcare professionals. Moreover, because no computerized analysis is available for QT monitoring, healthcare professionals must determine when it is appropriate to manually measure QT intervals (eg, when a patient is started on a potentially proarrhythmic drug). The purpose of the present review is to provide 'best practices' for hospital ECG monitoring. Randomized clinical trials in this area are almost nonexistent; therefore, expert opinions are based upon clinical experience and related research in the field of electrocardiography. This consensus document encompasses all areas of hospital cardiac monitoring in both children and adults. The emphasis is on information clinicians need to know to monitor patients safely and effectively. Recommendations are made with regard to indications, timeframes, and strategies to improve the diagnostic accuracy of cardiac arrhythmia, ischemia, and QT-interval monitoring. Currently available ECG lead systems are described, and recommendations related to staffing, training, and methods to improve quality are provided. (*Circulation.* 2004;110:2721-2746.)

Key Words: AHA Scientific Statements ■ pediatrics ■ electrocardiography ■ torsade de pointes ■ myocardial infarction ■ tachyarrhythmias ■ ischemia ■ antiarrhythmic agents ■ long-QT syndrome

Since the introduction of electrocardiographic (ECG) monitoring in hospital units >40 years ago,[1] the goals of monitoring have expanded from simple tracking of heart rate and basic rhythm to the diagnosis of complex arrhythmias, the detection of myocardial ischemia, and the identification of a prolonged QT interval. During the same 4 decades, major improvements have occurred in cardiac monitoring systems, including computerized arrhythmia detection algorithms, ST-segment/ischemia monitoring software, improved noise-reduction strategies, multilead monitoring, and reduced lead sets for monitoring-derived 12-lead ECGs with a minimal number of electrodes.[2,3]

Despite these advances in technology, the need for human oversight in the interpretation of ECG monitoring data is as important today as it was 40 years ago for the following reasons. First, cardiac monitor algorithms are intentionally set for high sensitivity at the expense of specificity. As a result, numerous false alarms occur that must be evaluated by healthcare professionals so that overtreatment of patients will not occur. Examples of overtreatment are reported in the

literature and include unnecessary cardiac catheterization because of false ST-segment monitor alarms,[4] as well as unnecessary electrophysiology testing and device implantation because of muscle artifact simulating ventricular tachycardia.[5]

Second, in the past several decades, it has been well established by numerous clinical trials that a more aggressive approach to the treatment of myocardial ischemia in acute coronary syndromes improves patient outcomes. Strategies to prevent infarction or to reduce infarct size rely heavily on the ability of healthcare professionals to identify myocardial ischemia in emergency departments (EDs) and to provide ongoing surveillance for recurrent ischemia in coronary intensive care units and intermediate care (telemetry) units.

Third, a plethora of electrophysiological interventions have been introduced during the past several decades, including catheter ablation, biventricular pacing, and implantable cardioverter defibrillators. Such complex device technology calls for healthcare professionals with expertise in analyzing ECG monitoring data for evidence of device malfunction and nonoptimal device programming.

Fourth, drugs have been introduced that have the potential to cause prolongation of ventricular repolarization. The failure of healthcare professionals to recognize a prolonged QT interval and prodromal polymorphic ventricular ectopy may result in sudden death because of the malignant ventricular arrhythmia torsades de pointes.

Fifth, and possibly most important, only humans, not cardiac monitors, can determine the goals of monitoring for an individual patient. For example, a young patient without heart disease who is monitored after uncomplicated catheter ablation does not need to be monitored for ST segment changes of ischemia or for QT interval prolongation (if no proarrhythmic drugs are being administered); however, a patient with acute myocardial infarction (MI) given an antiarrhythmic agent requires monitoring for arrhythmias, ischemia, and QT prolongation.

Although the need for skilled healthcare professionals with expertise in electrocardiography and cardiac monitoring has never been greater, the supply of such expert clinicians has never been smaller. The reasons for the short supply of expert clinicians include the following: First, the concept of a pure coronary care unit with nurses specially trained in ECG interpretation has been replaced in many US hospitals by multipurpose critical care units. Second, the critical shortage of nurses has meant that nurses with little or no intensive care clinical experience are working in monitored hospital units. The lesson learned by Day[6] when he originally attempted to place cardiac monitors on a medical ward to monitor patients with acute MI in 1960 still rings true today: "We had no nurses who could correctly interpret the electrocardiographic patterns or fathom the alarm systems and the proper application of electrodes ... The rapid turnover of nurses and part-time help on the medical floors made it impossible to develop adequately trained personnel." Third, medical students, residents, and cardiology trainees often are inadequately trained in ECG interpretation and they learn even less about cardiac monitoring leads and technology or interpretation of monitoring data. Mirvis[7] remarked: "In recent years,

the practice and science of electrocardiography have fallen from a distinguished position. Cardiology trainees are less interested and often must be obliged to read ECGs. Basic and clinical research in electrocardiography is sporadic, and health care financing agencies attempt to withdraw reimbursement."

Despite its devaluation by financing and training institutions, the ECG contains a wealth of diagnostic information routinely used to guide clinical decision making in hospitalized patients. For some conditions, such as transient myocardial ischemia, the ECG remains the gold standard for diagnosis, in spite of the advance of many other diagnostic techniques. The ECG is a noninvasive technique that is inexpensive, simple, and reproducible.[8] Moreover, the ECG is one of the most commonly used diagnostic tests that can be rapidly recorded with extremely portable equipment and is in general always obtainable.[9]

For all of these reasons, experts from the American Heart Association's Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young and the International Society of Computerized Electrocardiology felt compelled to develop a position paper to provide recommendations for best practices in hospital ECG monitoring. It was noted that prior ECG monitoring guidelines are either outdated[10,11] or limited in scope to one aspect of cardiac monitoring such as arrhythmia,[10,11] ST segment,[12] or QT interval[13] monitoring. Moreover, previous guidelines focused solely on adults; no attempt has been made to address the pediatric population requiring ECG monitoring. Hence, a comprehensive document is needed.

Published clinical trials in hospital cardiac monitoring are almost nonexistent. For this reason, it is not possible to develop a formal guideline with levels of evidence supported by published research. Nonetheless, it was deemed appropriate, timely, and valuable by members of the present writing group to provide expert opinions based on clinical experience and related research in the field of electrocardiography. It is understood that recommendations based on expert opinion require frequent updating as more definitive research data become available.

Therefore, this consensus document represents the first attempt in the literature to encompass all areas of hospital cardiac monitoring, including arrhythmia, ischemia, and QT interval monitoring in both children and adults. This report focuses on real-time ECG monitoring; hence, it does not address the recording of standard "snapshot" 12-lead ECGs in hospital settings or Holter monitoring, which is not performed for prospective clinical decision making. The emphasis here is on the information clinicians need to know to monitor patients safely and effectively.

The rating system used in this statement was devised by the American College of Cardiology Emergency Cardiac Care Committee[11] and consists of the following categories:

Class I: Cardiac monitoring is indicated in most, if not all, patients in this group.

Class II: Cardiac monitoring may be of benefit in some patients but is not considered essential for all patients.

Class III: Cardiac monitoring is not indicated because a patient's risk of a serious event is so low that monitoring has no therapeutic benefit.

Cardiac Arrhythmia Monitoring

Class I

Class I includes all patients at significant risk of an immediate, life-threatening arrhythmia. If a patient is required to leave the monitored unit for diagnostic or therapeutic procedures, then cardiac monitoring should be continued with a portable, battery-operated monitor-defibrillator used by a healthcare provider who is skilled in ECG interpretation and defibrillation. These patients are divided into 16 subcategories.

Patients Who Have Been Resuscitated From Cardiac Arrest

The patient resuscitated from outpatient or inpatient cardiac arrest is at high risk for recurrence of that event and should continue to be monitored in an intensive care unit while being evaluated for the cause of the event (eg, hyperkalemia, acute myocardial ischemia) and while corrective/preventive treatment is being instituted. ECG monitoring should continue until an implantable cardioverter defibrillator (ICD) is implanted, unless the patient had a clearly transient, reversible, preventable, and now-corrected cause of the cardiac arrest. Such transient situations are relatively rare.

Patients in the Early Phase of Acute Coronary Syndromes (ST-Elevation or Non–ST-Elevation MI, Unstable Angina/"Rule-Out" MI)

Much of the published data on ECG monitoring of patients with acute MI were collected during an era when treatment, and therefore the natural history, was different from treatment today. Factors such as early mechanical revascularization, nitrates, aspirin and other antiplatelet and antithrombotic agents, β-blockers, and angiotensin-converting enzyme inhibitors have revolutionized care and have greatly reduced the incidence and time course of complicating arrhythmias. For example, a patient with acute MI who presents early after onset of symptoms to an institution with an immediate percutaneous coronary intervention protocol may receive a definitive therapy (eg, a stent to an occluded vessel) and be sent home the next day. At the other end of the spectrum are acute MI patients who do not have such definitively successful reperfusion outcomes or who have a more complicated course because of comorbidities, advanced age, or other factors. Thus, one finds a wide range of recommended ECG monitoring time frames, from 24 hours in the former case to ≥72 hours in the latter case.

It is recommended that monitoring begin as soon as the patient presents to the ED and continue uninterrupted for a minimum of 24 hours for uncomplicated acute MI. Because of the possibility of malignant reperfusion arrhythmias, all patients who receive early reperfusion therapy should undergo uninterrupted ECG monitoring, including during intrahospital transport. Bonnemeier et al[14] reported that in patients with a first MI, those with elevated initial troponin values are more likely than those with normal initial troponins to experience malignant reperfusion arrhythmias after primary percutaneous coronary interventions. In patients with a more complicated course, such as those with ongoing or recurrent ischemia, development of acute heart failure or cardiogenic shock, and arrhythmias requiring an intervention such as temporary pacing, defibrillation, or intravenous antiarrhythmics, monitoring should continue for 24 hours after complications have resolved. Patients with unstable angina or "rule-out" MI should undergo cardiac monitoring until infarction has been ruled out and signs (transient ST-T–wave changes) and symptoms (chest pain or anginal equivalent) of myocardial ischemia have been absent for 24 hours.

Patients With Unstable Coronary Syndromes and Newly Diagnosed High-Risk Coronary Lesions

ECG monitoring is indicated for patients with newly diagnosed critical left main coronary artery disease or its equivalent (eg, proximal left anterior descending and circumflex disease) who are candidates for urgent revascularization. Monitoring should continue uninterrupted while these patients await intervention.

Adults Who Have Undergone Cardiac Surgery

ECG monitoring should be performed after uncomplicated cardiac surgery for a minimum of 48 to 72 hours. For patients at high risk for developing postoperative atrial fibrillation, monitoring should continue until hospital discharge. Risk factors for the development of postoperative atrial fibrillation include advanced age, history of atrial fibrillation, presence of valvular disease, and preoperative β-blocker withdrawal.[15,16] Creswell et al[17] reported that the incidence of postoperative atrial fibrillation in a sample of >4000 patients is 32% after coronary artery bypass surgery, 64% after combined bypass and mitral valve replacement surgery, 49% after combined bypass and aortic valve replacement, and 11% after heart transplantation. The incidence of postoperative atrial fibrillation in minimally invasive coronary bypass procedures is not significantly different than it is with traditional techniques.[18–20]

The onset of atrial fibrillation typically occurs on the second to fourth postoperative day. Funk and coworkers[21] recently reported that the development of atrial fibrillation after cardiac surgery is not uncommon after hospital discharge. These investigators found that 14% of 302 patients developed atrial fibrillation in the 2 weeks after hospital discharge and that 69% of these episodes were asymptomatic. A predictor of postdischarge atrial fibrillation was a recorded episode of atrial fibrillation while the patient was hospitalized, which provides a rationale for ECG monitoring throughout the entire hospital stay. Other arrhythmias that occur after cardiac surgery are ventricular tachycardia and fibrillation, atrioventricular (AV) block, and sinus node dysfunction.[22,23]

A recommendation for the improvement of the diagnostic accuracy of postoperative tachyarrhythmias is to take advantage of atrial epicardial pacemaker leads that often are left in place after surgery.[24] When atrial fibrillation has a ventricular response >150 bpm, the R-R intervals vary less noticeably than they do after the ventricular rate is slowed. Thus, clinicians may fail to note the random R-R irregularity that is characteristic of atrial fibrillation, and the rhythm may be misdiagnosed as paroxysmal supraventricular tachycardia. Likewise, atrial activity may not be obvious on the surface

ECG in patients who develop atrial flutter. Furthermore, in a patient with preexisting bundle-branch block, the development of a postoperative supraventricular tachyarrhythmia may be difficult to distinguish from ventricular tachycardia. In all of these situations, an accurate diagnosis can be readily made if an atrial electrogram is recorded. The technique for recording an atrial electrogram is described in the subsequent section on cardiac monitoring lead systems.

Children Who Have Undergone Cardiac Surgery
In contrast to adults, children who undergo cardiac surgery, typically to repair congenital cardiac defects, are not particularly at risk for postoperative atrial fibrillation. Arrhythmias that are more commonly observed in the pediatric age group are atrial flutter and junctional ectopic tachycardia.[25] In addition, ventricular tachycardia may occur after procedures that involve ventriculotomy or after coronary reimplantation in the arterial switch procedure for transposition. Recording the atrial electrogram using temporary epicardial pacemaker leads may be especially useful for diagnosing arrhythmias in children after congenital heart surgery. For example, an atrial electrogram is valuable in distinguishing junctional ectopic tachycardia from sinus tachycardia.

Patients Who Have Undergone Nonurgent Percutaneous Coronary Intervention With Complications
ECG monitoring is indicated for patients with coronary angioplasty, stenting, or both who experience complications in the catheterization laboratory such as vessel dissection or no reflow or who have less-definitive interventional outcomes. Monitoring should be initiated immediately postprocedure and continue for 24 hours or longer if arrhythmias or ST-segment–deviation events occur.

Patients Who Have Undergone Implantation of an Automatic Defibrillator Lead or a Pacemaker Lead and Are Considered Pacemaker Dependent
Pacemaker dependency is an unstable or absent spontaneous rhythm with hemodynamic instability in the absence of pacing. Lead dislodgement is a well-known although uncommon early complication after insertion of pacemakers, defibrillators, and (more commonly) biventricular pacemakers.[26–29] Another less common cause of loss of capture is a sudden increase in pacing threshold. Such threshold increases have been largely eliminated with the widespread use of steroid-eluding leads. Another pacemaker problem that can be identified with ECG monitoring and corrected with noninvasive reprogramming includes the failure to sense (in the atrium or ventricles). ECG monitoring of the patient is recommended for 12 to 24 hours after implantation.

Patients With a Temporary Pacemaker or Transcutaneous Pacing Pads
Temporary transvenous pacemakers are associated with a higher risk of loss of capture than are permanent pacemakers. Temporary transvenous lead wires are stiffer than permanent lead wires to facilitate rapid insertion from remote venous access points. In addition, they lack active and passive fixation mechanisms of permanent leads. This makes lead perforation (through the right ventricular free wall or interventricular septum) or lead dislodgement more likely. In

addition, no pacemaker output may occur if lead wires become separated from the external pacemaker generator, batteries become depleted, or oversensing occurs because of large P or T waves or extraneous electrical potentials such as muscle artifact or nearby faulty electrical equipment. Therefore, it is recommended that all patients with temporary pacemakers be monitored until pacing is either no longer necessary and the device is removed or replaced with a permanent device. Transcutaneous pacing is subject to the same concerns as those for other temporary pacemakers. In addition, because the pacing artifact is large, it may obscure or mimic the QRS complex, making it difficult to determine the presence of ventricular capture. In such instances, different ECG monitoring leads should be tried to identify a lead that minimizes the pacemaker artifact and maximizes the QRS complex. If no such lead can be identified, then concomitant monitoring with a non-ECG method is recommended (eg, arterial pressure, pulse oximetry monitoring, or both).

Patients With AV Block
Monitoring is indicated for patients with Mobitz II block, advanced (2:1 or higher) second-degree AV block, complete heart block, or new-onset bundle-branch block in the setting of acute (especially anterior) MI. Sir Thomas Lewis's "law of the heart" states that natural pacemakers from more distal sites in the conduction system tend to be slower and less reliable. Mobitz II AV block, especially with a wide QRS complex, typically results from disease in the distal (ie, His-Purkinje) system, and thus if complete block develops, then the escape pacemakers tend to be slow and unreliable. Therefore, patients with Mobitz II AV block require intensive monitoring. Mobitz I (Wenckebach) AV block with a narrow QRS complex is typical of a proximal (ie, AV nodal) site of block, and thus if complete block develops, then the escape pacemakers are faster and more reliable. Because one cannot always predict the outcome of Mobitz I block, these patients should be monitored unless it has been established that the block is a stable long-term condition.

Second-degree 2:1 AV block or AV block with consecutive blocked P waves is not categorized as Mobitz I or II because it does not allow inference about the proximal versus distal site of block. Because some of these rhythms reflect His-Purkinje system disease, monitoring is recommended. For patients with Mobitz II advanced second-degree AV block, or complete heart block, ECG monitoring should be continued until the block resolves or until a definitive therapy (usually implantation of a permanent pacemaker) is implemented.

Patients With Arrhythmias Complicating Wolff-Parkinson-White Syndrome With Rapid Anterograde Conduction Over an Accessory Pathway
Sudden cardiac death in Wolff-Parkinson-White (WPW) syndrome is strongly associated with rapid anterograde conduction over the accessory pathway, typically during atrial fibrillation. Other factors that have been implicated include a family history of WPW, syncope, use of digitalis, and presence of multiple accessory pathways. Therefore, monitoring of patients with arrhythmias exhibiting rapid antero-

grade conduction over an accessory pathway is recommended until a definitive therapy (usually an ablation procedure) is established.

Patients With Long-QT Syndrome and Associated Ventricular Arrhythmias

Torsades de pointes[30] is a life-threatening, hemodynamically unstable polymorphic ventricular tachycardia that is associated with a prolonged QT interval and is typically triggered by a ventricular premature beat arising out of a pause-dependent increase in U wave amplitude. Prolonged runs may degenerate to ventricular fibrillation. The prolonged QT interval, pause-dependent increases in U wave amplitude, polymorphic ventricular premature beats, or ventricular bigeminy often precede by minutes or even hours polymorphic couplets, triplets, and eventually longer runs. Therefore, strict monitoring of these patients is required. A complete discussion of QT interval monitoring is provided in a later section.

Patients Receiving Intraaortic Balloon Counterpulsation

In addition to the need to monitor all patients who are hemodynamically unstable, patients with a balloon pump may benefit from the recognition of and intervention for arrhythmias that may make tracking by the device difficult and thus decrease its effectiveness. ECG monitoring should be continued until the patient is weaned from the intraaortic balloon pump.

Patients With Acute Heart Failure/Pulmonary Edema

A variety of arrhythmias may contribute to or be the primary cause of acute cardiac decompensation (eg, the development of atrial fibrillation with an uncontrolled ventricular response). Acute heart failure also is a major risk factor for atrial and ventricular arrhythmias. In addition, some therapies for heart failure, especially intravenous positive inotropic drugs (eg, milrinone, dobutamine), have significant proarrhythmic properties.[31,32] Because B-type natriuretic peptide (nesiritide) is an arterial and venous dilator that inhibits sympathetic activity, it may be less arrhythmogenic than positive inotropic agents. Burger et al[33] reported that patients with heart failure who were treated with nesiritide were less likely to experience sustained ventricular tachycardia or cardiac arrest than were patients who were treated with dobutamine. Monitoring is valuable for detecting sinus tachycardia that may signal hypotension during administration of nesiritide. Therefore, continuous monitoring is recommended for all patients until the signs and symptoms of acute heart failure have resolved and cardiac monitoring reveals no hemodynamically significant arrhythmias for at least 24 hours.

Patients With Indications for Intensive Care

ECG monitoring is recommended for patients with major trauma, acute respiratory failure, sepsis, shock, acute pulmonary embolus, major noncardiac surgery (especially in older adult patients with a history of coronary artery disease or coronary risk factors), renal failure with electrolyte abnormalities (eg, hyperkalemia), drug overdose (especially from known arrhythmogenics, eg, digitalis, tricyclic antidepressants, phenothiazines, antiarrhythmics), and other illnesses. It is estimated that ≈1 in 5 patients admitted to intensive care

will develop significant arrhythmias, most commonly atrial fibrillation or ventricular tachycardia.[34] Clinically significant arrhythmias have been reported in a variety of surgical populations requiring intensive care, for example, patients undergoing major noncardiothoracic surgery,[35] colorectal surgery,[36] and pulmonary surgery.[37] ECG monitoring should be continued until patients are weaned from mechanical ventilation and are hemodynamically stable.

Patients Undergoing Diagnostic/Therapeutic Procedures Requiring Conscious Sedation or Anesthesia

Numerous procedures requiring conscious sedation are performed in hospital settings (eg, electrocardioversion). ECG monitoring is indicated for all such procedures and should be continued until patients are awake, alert, and hemodynamically stable.

Patients With Any Other Hemodynamically Unstable Arrhythmia

It is important to point out that arrhythmias that are considered benign in an individual without heart disease may be lethal in a patient with significant heart disease. For example, the development of atrial fibrillation in a patient with critical aortic stenosis or hypertrophic cardiomyopathy may cause immediate hemodynamic deterioration. Therefore, a Class II indication for arrhythmia monitoring may appropriately be a Class I indication for patients with heart disease.

Diagnosis of Arrhythmias in Pediatric Patients

In general, the mechanisms of arrhythmias are the same in children as they are in adults; however, the appearance of the arrhythmias on the ECG may differ because of developmental issues such as heart size, baseline heart rate, sinus and AV node function, and autonomic innervation. For example, the distinction between wide and narrow QRS tachycardia must be altered to take into account a child's age. Although a QRS width of >0.12 second defines wide QRS tachycardia in adults, the upper limit of normal in infants is ≈0.08 second.[38] This discrepancy means that ventricular tachycardia in an infant with a QRS duration of 0.09 second may be misdiagnosed as supraventricular tachycardia, if adult criteria are used. Similarly, the definition of tachycardia based on rate is also age dependent, with the upper limit of typical being higher in infants (158 bpm) as compared with that in teenagers (120 bpm). These differences present significant issues for the computerized arrhythmia detection algorithms in cardiac monitoring systems, as well as for the clinicians who interpret arrhythmias. Typical age-based ECG standards are shown in Table 1.[38]

Class II

ECG monitoring may be beneficial in some patients, but it is not considered essential for all. Cardiac monitoring is helpful in the clinical management of Class II patients, but it is not expected to save lives. Cardiac monitoring often takes place in an intermediate care (telemetry) unit. These patients are divided into 10 subcategories.

Patients With Postacute MI

The decision whether to continue monitoring acute MI patients 24 to 48 hours after admission is controversial. On

TABLE 1.   Normal ECG Standards for Children by Age

	0–1 d	1–3 d	3–7 d	7–30 d	1–3 mo	3–6 mo	6–12 mo	1–3 y	3–5 y	5–8 y	8–12 y	12–16 y
Heart rate/min	94–155 (122)	91–158 (122)	90–166 (128)	106–182 (149)	120–179 (149)	105–185 (141)	108–169 (131)	89–152 (119)	73–137 (109)	65–133 (100)	62–130 (91)	60–120 (80)
PR interval lead II, s	0.08–0.16 (0.107)	0.08–0.14 (0.108)	0.07–0.15 (0.102)	0.07–0.14 (0.100)	0.07–0.13 (0.098)	0.07–0.15 (0.105)	0.07–0.16 (0.106)	0.08–0.15 (0.113)	0.08–0.16 (0.119)	0.09–0.16 (0.123)	0.09–0.17 (0.128)	0.09–0.18 (0.135)
QRS interval lead V_5, s	0.02–0.07 (0.05)	0.02–0.07 (0.05)	0.02–0.07 (0.05)	0.02–0.08 (0.05)	0.02–0.08 (0.05)	0.02–0.08 (0.05)	0.03–0.08 (0.05)	0.03–0.08 (0.06)	0.03–0.07 (0.06)	0.03–0.08 (0.06)	0.04–0.09 (0.06)	0.04–0.09 (0.07)

All values 2nd–98th percentile; numbers in parentheses, means. Adapted from *Pediatr Cardiol.* 1979;1:123.

the one hand, analysis of the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO-III) study data shows that patients who have late ventricular arrhythmias (>48 hours after hospital admission) have a higher mortality at 1 month and 1 year than do patients who have early arrhythmias.[39] Thus, ECG monitoring past 48 hours would likely help to identify a high-risk group that may benefit from more aggressive therapy and closer postdischarge follow-up. On the other hand, although ventricular arrhythmias after 48 hours post-MI have prognostic significance, they seldom occur. Thus, many patients need to be monitored to identify just 1 of these high-risk patients. Most of the risk for major ventricular arrhythmias in the 15 059 GUSTO-III patients occurred during the first 24 hours, after which the hazard curve was flat.[40] Moreover, 95% of major adverse outcomes (death, stroke, or shock) occurred within the first 24 hours.

Predictors of in-hospital sustained ventricular arrhythmias (ventricular tachycardia and fibrillation) have been reported recently for patients with post–ST-elevation MI[39] and post–non–ST-elevation MI.[41] These predictors include previous hypertension, chronic obstructive pulmonary disease, previous MI, ST-segment changes at presentation, higher Killip class, and lower initial systolic blood pressure. Thus, presently, it seems reasonable to continue to monitor post-MI patients with any of these predictors beyond 48 hours until hospital discharge.

Patients With Chest Pain Syndromes

Patients who present to the ED with chest pain but who do not have diagnostic ECG findings or elevated biomarkers often are admitted to a telemetry unit while repeat troponins and signs and symptoms of myocardial ischemia are monitored. Recently, this practice has been questioned. For example, Snider et al[42] reported that in a total of 414 patients consecutively admitted from the ED to a telemetry unit for suspected acute coronary syndromes, 37% had atypical chest pain and normal ECG findings. Arrhythmias were observed in only 8% of this subgroup and only 4 patients had arrhythmia events that led to an intervention. These investigators concluded that patients with atypical chest pain and a normal ECG in the ED were at low risk of life-threatening arrhythmias and that the use of telemetry monitoring in this group should be reevaluated. Estrada et al[43] assessed the role of telemetry in guiding patient management decisions in 2240 patients admitted to a telemetry unit. They reported that patients admitted for syncope or chest pain syndromes had lower rates of unexpected intensive care transfer and most were unrelated to arrhythmic conditions. They concluded that telemetry was less valuable for clinical decision making in patients with

chest pain syndromes. Estrada et al[44] used the same cohort of 2240 patients to determine whether the American College of Cardiology cardiac monitoring guidelines[11] accurately stratified patients according to their risks for developing clinically significant arrhythmias in non–intensive care settings. They concluded that patients with chest pain should be moved from Class I to Class II and patients with arrhythmias should be moved from Class II to Class I.

A major limitation of these investigations[42–44] is that ST-segment monitoring was not performed in the study's telemetry units. Recently, Pelter et al[45] conducted continuous 12-lead ST-segment monitoring in 237 patients who were treated on a telemetry unit for postacute MI or chest pain syndromes. Thirty-nine patients (17%) had ≥1 episode of transient myocardial ischemia (Figure 1). Serious in-hospital consequences (ie, death, major arrhythmia, cardiogenic shock, acute pulmonary edema, abrupt reocclusion after percutaneous coronary intervention, MI after telemetry admission, or unplanned transfer to the intensive care unit) occurred in 46% of the group with transient myocardial ischemia as compared with 10% in the group without ischemia ($P<0.001$). Patients with transient myocardial ischemia were 8.5 times more likely than those without ischemia to have in-hospital complications (95% CI, 3.7 to 19.7) after investigators controlled for other predictors of adverse out-
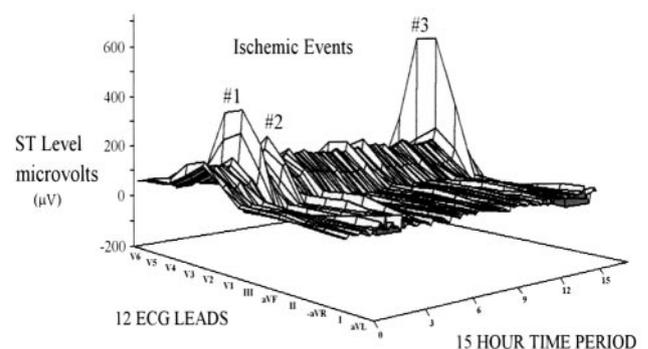

Figure 1. 12-lead ST segment monitoring data recorded in a 78-y-old man admitted to telemetry unit with chest pain syndrome and negative troponins. Vertical (Y) axis shows ST amplitudes in μV measured at J+60 ms. Negative numbers reflect ST depression; positive numbers, ST elevation. 12 ECG leads displayed in Cabrillo format on X axis and time on Z axis. ST events 1 and 2 went unnoticed by clinicians because patient did not complain of chest pain. ST event 3 was associated with chest pain, and patient was taken to cardiac catheterization laboratory and treated with stent to occluded LAD coronary artery. Subsequently, troponins were elevated and diagnosis made of acute ST elevation MI occurring after hospital admission. Reprinted with permission from *Heart Lung.* 2003;32:71. Copyright 2003, Elsevier Science.

come (advanced age, radiographic evidence of heart failure, previous MI). In a companion study, Pelter et al[46] reported that the incidence of transient myocardial ischemia in telemetry units is the same as it was in coronary care units (CCUs) in 1999 and that the vast majority of these ST events are clinically silent (proportion of silent ST events: 71% in the telemetry group, 58% in the CCU group).

A more rational approach to making a decision about which patients presenting to the ED with chest pain should be treated in a hospital unit with ECG monitoring is to use an evidence-based prediction tool. The Goldman risk-assessment tool categorizes patients into a high-, moderate-, low-, or very-low-risk group based on initial ECG and history and physical examination findings.[47] Goldman et al found 5 variables to be valuable in predicting the risk of a major adverse event in a large cohort of >10 000 chest pain patients. These predictors were (1) suspected MI on initial ECG (ST-segment elevation of ≥1 mm or pathological Q waves in ≥2 leads), (2) suspected ischemia on initial ECG (ST-segment depression of ≥1 mm or T wave inversion in ≥2 leads), (3) systolic blood pressure <110 mm Hg, (4) rales heard above the bases bilaterally, and (5) history of unstable ischemic heart disease (worsening of previously stable angina, new onset of post-MI angina, angina after a coronary revascularization procedure, or pain that is the same as that associated with a previous MI).

Recently, 2 studies reported using the Goldman risk score to determine which ED patients should receive inpatient monitoring on a telemetry unit. Durairaj et al[48] found that among the 318 patients with chest pain who were classified in the very-low-risk category, 0 suffered a major in-hospital complication. Likewise, Hollander et al[49] found that among 1029 patients who had a low Goldman risk score and negative initial biomarkers, 0 suffered cardiovascular death or a life-threatening ventricular arrhythmia during hospital telemetry monitoring.

In the absence of a prospective randomized clinical trial to determine whether telemetry-guided management improves patient outcomes, it seems reasonable to recommend inpatient ECG monitoring for patients with any sign of ischemia or infarction on the initial ECG, as well as for patients with ≥1 evidence-based risk factor (low systolic blood pressure, pulmonary rales, or exacerbation of ischemic heart disease). ECG monitoring should be continued for 12 to 24 hours until acute MI has been ruled out by negative biomarkers.

Patients Who Have Undergone Uncomplicated, Nonurgent Percutaneous Coronary Interventions
Monitoring in patients who have undergone uncomplicated, nonurgent percutaneous coronary interventions (ie, not for acute MI) should begin immediately postintervention, but it need not continue after 6 to 8 hours if patients received a stent. Patients who undergo coronary angioplasty without stenting should be monitored for 12 to 24 hours because of the higher incidence of abrupt closure.

Patients Who Are Administered an Antiarrhythmic Drug or Who Require Adjustment of Drugs for Rate Control With Chronic Atrial Tachyarrhythmias
The potential benefits of monitoring include (1) detection of a prolonged QT interval response to the drug, (2) assessment of sinus node function after initiating a drug with negative chronotropic properties, especially when the integrity of the sinus node is uncertain, (3) detection of hemodynamic deterioration after initiating an antiarrhythmic drug with negative inotropic properties, especially in patients with compromised left ventricular function (ejection fraction <40%), and (4) assessment of the efficacy of the drug to control the ventricular rate in chronic atrial fibrillation or flutter, especially with increasing patient activity. It should be pointed out that for patients who are administered certain antiarrhythmic drugs with a known high risk of proarrhythmia, ECG monitoring should be considered a Class I rather than a Class II indication (see "QT Interval and ECG Monitoring for Detection of Proarrhythmia").

Patients Who Have Undergone Implantation of a Pacemaker Lead and Are Not Pacemaker Dependent
Patients who are not pacemaker dependent have a spontaneous rhythm in the absence of pacing that does not cause hemodynamic instability. Thus, the goal of monitoring pacemaker function in these patients is not to detect and treat life-threatening bradyarrhythmias but to detect pacemaker failure to capture, pace (no output), or sense appropriately. To confirm that pacing function and programming are appropriate, 12 to 24 hours of postprocedural ECG monitoring is recommended.

Patients Who Have Undergone Uncomplicated Ablation of an Arrhythmia
Patients undergoing ablation procedures are typically discharged after a short observation period. AV block is a rare complication of radiofrequency ablation for AV nodal reentrant tachycardia, and it often resolves without permanent pacing.[50] Therefore, it is no longer routine practice to monitor such patients. Patients who may benefit from postprocedural ECG monitoring are those who have experienced prolonged rapid heart rates from an incessant tachycardia because they may develop prolonged QT interval and torsades de pointes after ablation therapy.[51] Likewise, torsades de pointes has been reported in patients with chronic atrial fibrillation who have undergone AV junction ablation with the implantation of a pacemaker.[52] Although pacemaker programming to maintain relatively high paced rates is thought to decrease the incidence of this complication, 12 to 24 hours of ECG monitoring is recommended. In addition, patients with significant organic heart disease who undergo ventricular tachycardia ablation warrant postprocedural monitoring for 12 to 24 hours.

Patients Who Have Undergone Routine Coronary Angiography
When vascular closure devices are used to seal the groin puncture, patients often can ambulate and be discharged several hours after uncomplicated diagnostic coronary angiography. ECG monitoring may be indicated immediately after the procedure, however, because vasovagal reactions causing symptomatic bradycardia are not uncommon in this setting.

Patients With Subacute Heart Failure

The role of telemetry monitoring in this patient population is unclear. Opasich et al[53] reported on 711 inpatients with heart failure, 199 of whom underwent telemetry monitoring. The decision to use telemetry was related to known arrhythmia (n=82), electrolyte disturbances (n=20), atrial fibrillation (n=12), symptoms (n=48), intravenous dobutamine (n=13), drug control (n=16), or device control (n=8). The investigators determined that treatment was guided by telemetry in only 33 patients (17%). The physicians' perception was that telemetry monitoring was helpful in 70% of patients, however. One reason for this discrepancy may have been that the investigators considered telemetry important in guiding treatment only if it resulted in a change in treatment. It could be argued that telemetry monitoring may have provided documentation for and reassurance about the efficacy of the treatment plan and that no changes in treatment were warranted. In the absence of randomized clinical trials, it seems reasonable to perform ECG monitoring in the subacute phase of acute heart failure while medications, device therapy, or both are being manipulated.

Patients Who Are Being Evaluated for Syncope

Many patients with syncope in whom a careful history is taken do not require hospitalization. Patients with syncope of truly unknown origin should have ≥24 hours of inpatient monitoring. The diagnostic yield of ECG monitoring in patients with syncope may be low in the absence of a high amount of suspicion about an arrhythmic cause.[54] Kapoor[55] emphasized that in patients with syncope, heart disease is the major predictor of risk for death or significant arrhythmia. When suspicion arises about an arrhythmic cause for the syncope or in patients who have primary electrophysiologic disorders (eg, conduction system disease, nonsustained ventricular tachycardia, possible pacemaker malfunction), inpatient monitoring is indicated for 24 to 48 hours, or until an arrhythmic cause has been ruled out by invasive cardiac electrophysiological testing.

Patients With Do-Not-Resuscitate Orders With Arrhythmias That Cause Discomfort

Terminally ill patients experiencing palpitations, shortness of breath, anxiety, or all of these symptoms may require arrhythmia management as part of palliative care provision. The goal of cardiac monitoring in these patients is not to prevent or treat life-threatening arrhythmias, but rather to assist in titrating antiarrhythmic drugs for optimum rate control. ECG monitoring can be discontinued when rate control has been achieved.

Class III

The patients included in this class are postoperative patients who are at low risk for cardiac arrhythmias (eg, young patients without heart disease who undergo uncomplicated surgical procedures); obstetric patients, unless heart disease is present; patients with permanent, rate-controlled atrial fibrillation; patients undergoing hemodialysis (in general, hemodialysis is performed in outpatient settings [the National Kidney Foundation does not mention the need for ECG monitoring during dialysis; see http://www.kidney.org/professionals/kdoqi/index.cfm]); however, when patients have a Class I or II indication and undergo dialysis in the hospital, ECG monitoring is recommended); and stable patients with chronic ventricular premature beats. Malignant ventricular arrhythmias are unlikely to be triggered by ventricular premature beats in the absence of major modulating factors such as acid-base imbalance, electrolyte abnormality, or myocardial ischemia.

ST-Segment Ischemia Monitoring

Beginning in the mid-1980s, cardiac monitoring companies began adding special ST-segment analysis software to their equipment. Although the current generation of monitors provides for computerized ischemia monitoring, many hospital units still lack this capability. It is also important to point out that in most monitors with computerized ischemia monitoring software, a nurse must activate the software for it to work. Therefore, unlike computerized arrhythmia monitoring that is automatically performed, ST-segment ischemia monitoring must in general be manually enabled. Unfortunately, even in hospital units with computerized ischemia monitoring capability, ST-segment monitoring is widely underused. The results of a recent national random survey of 192 nurse leaders in hospital cardiac units revealed that 46% did not use ST-segment monitoring for the detection of myocardial ischemia in patients admitted with acute coronary syndromes.[56] The primary reason listed for nonuse was "lack of physician support." Other reasons included a high number of false ST alarms and lack of education about how to use the technology and what to do in response to ST alarms.

It is important to point out that no randomized clinical trials have been conducted to determine whether the addition of computerized ST-segment ischemia monitoring improves patient outcomes. Thus, the assignment of the following clinical situations to each of the categories (Class I, II, III) is not based on research but rather on the opinions of the expert writing group. In the absence of such research, it would be inappropriate to state that hospitals without ST-segment monitoring capability are delivering substandard care; however, in the opinion of the expert writing group, when aging cardiac monitors need to be replaced, automated ischemia monitoring capability should be considered, especially for hospitals that provide care for a large number of patients with acute coronary syndromes.

Class I

Patients in the Early Phase of Acute Coronary Syndromes (ST-Elevation or Non–ST-Elevation MI, Unstable Angina/"Rule-Out" MI)

Patients with acute coronary syndromes are the highest-priority candidates for ST-segment monitoring. They should be monitored for a minimum of 24 hours and until they remain event-free for 12 to 24 hours. The potential benefits in patients with acute MI include the ability to (1) assess patency of the culprit artery after thrombolytic therapy[57–61]; (2) detect abrupt reocclusion after primary angioplasty[62]; (3) detect ongoing ischemia (ie, failed reperfusion therapy), recurrent ischemia, and infarct extension; and (4) detect transient myocardial ischemia. ST-segment monitoring stud-

TABLE 2.   Prognostic Significance of Transient Myocardial Ischemia With ST-Segment Monitoring in Patients Hospitalized for Unstable Angina

Investigator, Year	n	Incidence of Transient Ischemia (%)	% of Silent Events	% With Adverse Outcome (death or MI)
Gottlieb et al				
1986[63]	70	53	90	at 30 d: + ischemia=16, − ischemia = 3, $P<0.01$
1987[64]				at 2 y: + ischemia=27, − ischemia = 3, $P<0.01$
Nademanee et al, 1987[65]	49	59	91	at 3–6 mo: + ischemia=17, − ischemia = 5
Krucoff, 1988[66]	282	23	84	Hospital: + ischemia=31, − ischemia = 0
Langer et al, 1989[67]	135	66	92	Hospital: + ischemia = 16, − ischemia = 4, $P<0.05$
Larsson et al, 1992[68]	198	23	94	at 30 d: + ischemia=17, − ischemia = 3, $P<0.01$
Amanullah, Lindvall, 1993[69]	43	98	>90	>3 y: + ischemia=18, − ischemia = 0
Bugiardini et al, 1995[70]	104	93	Not reported	Hospital+30 d: + ischemia=38, − ischemia = 0

ies of patients hospitalized with unstable angina show that although 80% to 90% of transient ischemic events are asymptomatic, they are nonetheless significant markers for unfavorable short- and long-term outcomes (Table 2).[63–70]

Patients Who Present to the ED With Chest Pain or Anginal Equivalent Symptoms

It is not uncommon for patients with acute ST-elevation MI to have an initial ECG that is nondiagnostic for acute ischemia. Investigators who use continuous monitoring have shown that the ST segment often is dynamic in the early hours of acute MI. This pattern of dynamic ST-segment elevation has been termed "intermittent reperfusion" and is thought to represent cycles of thrombotic occlusion and spontaneous reperfusion in early infarction. Seven studies have reported on the frequency of intermittent reperfusion in acute ST-elevation MI.[58–60,71–74] A meta-analysis of these studies indicates that the frequency is 34% to 40% (95% CI).[75] When ST-segment elevation is dynamic, an initial ECG may not exhibit ST-segment elevation if the patient is in a period of resolving ST segments when the standard 12-lead ECG is recorded. It is important to point out that a standard 12-lead ECG provides only a 10-second period of ECG information. Thus, unless continuous ST-segment monitoring is instituted in the ED, it is likely that some patients who would benefit from early reperfusion therapy will go untreated. Tatum et al reported that 1% to 2% of the 3 million chest pain patients sent home from the ED annually may have been discharged in error, and these "missed MI" patients have a mortality rate almost twice that of the chest pain patients who are admitted to the hospital.[76]

ST-segment monitoring for 8 to 12 hours in combination with testing serum biomarkers of injury may be a cost-effective way to triage patients who present to the ED with chest pain.[77–81] Because many of these patients do not really suffer from acute coronary syndromes, ST-segment monitoring in the ED may be less costly if it results in fewer "rule-out" MI patients being admitted to a monitored hospital unit.

Patients Who Have Undergone Nonurgent Percutaneous Coronary Intervention With Suboptimal Angiographic Results

This group includes patients with coronary angioplasty, stents, or both who experience complications in the catheterization

laboratory such as vessel dissection or thrombosis or who have less-definitive interventional outcomes. Monitoring should be initiated immediately postprocedure and continue for ≥24 hours if ST events occur. Abrupt reocclusion is most likely to occur early after the procedure, often before the patient has left the cardiac catheterization laboratory or within the first several hours after transfer to a monitored unit.[82] When multilead ECG monitoring is performed during the intervention, documentation of ST-segment deviation during catheter balloon occlusion improves both sensitivity and specificity of interpretation of ST events postintervention.[62,83]

Patients With Possible Variant Angina Resulting From Coronary Vasospasm

The potential benefits of ST-segment monitoring include the ability to (1) confirm the diagnosis by observing transient ST-segment elevation, (2) predict the culprit artery and proximity of site of vasospasm (if multilead or 12-lead monitoring is being performed), (3) assess the risk for malignant ventricular arrhythmias during vasospasm, and (4) assess the efficacy of therapy with a calcium-channel blocker. ST monitoring should continue until therapy has been initiated and the patient has been ST event-free for 12 to 24 hours.

Class II

Patients With Postacute MI

ST monitoring should not be discontinued in patients who have experienced recurrent chest pain or anginal symptoms or who have had a second elevation in cardiac enzymes indicating infarct extension until they have experienced a 24-hour-long ST event–free period. If the patient has recurrent symptoms of ischemia after ST monitoring is discontinued, then ST monitoring should be restarted. A potential benefit of ST monitoring in the postacute MI period is to assess a patient's readiness for early mobilization and discharge from the hospital. The absence of ischemic events with increasing physical activity in the hospital provides justification for the efficacy of the antianginal regimen and for early discharge of the patient.

Patients Who Have Undergone Nonurgent Uncomplicated Percutaneous Coronary Intervention

Although not mandatory for stable patients, if cardiac monitors are equipped with ST monitoring in the postprocedure

unit, ST monitoring should be activated in the immediate postintervention period and continued for 4 to 8 hours. To evaluate the need for postangioplasty cardiac monitoring, Li and coworkers[84] reported on the clinical outcome of consecutive patients who were monitored postintervention. ECG monitoring of 135 patients yielded 23 significant findings (eg, death, emergency bypass operation, or acute MI). Of the 23 patients with adverse hospital outcomes, 22 had a complicated or an unsuccessful intervention. In the 122 patients with successful coronary angioplasty without angiographic evidence of vessel complications or clinical symptoms at the end of the procedure, no significant arrhythmia or acute MI occurred. These investigators concluded that ECG monitoring is not required after successful, uncomplicated coronary angioplasty. Li's study was conducted in the early 1990s, and the subsequent introduction of stents has made the complication of early abrupt vessel closure even rarer. Thus, cardiac monitoring is not considered mandatory for stable postpercutaneous coronary intervention patients, especially those with only stented vessel(s).

An important potential benefit of ST monitoring in the postintervention period is the ability to evaluate chest pain. In a small cohort of patients, Jeremias et al[85] found that ≈41% of stent patients and 12% of angioplasty patients experienced postintervention chest pain. Noncardiac chest pain may be caused by stretching the coronary vessel during high-pressure balloon inflations or stent deployment.[85,86] Benign chest pain, nausea, or other nonspecific symptoms also may result from gastrointestinal causes brought on by fasting or esophageal reflux after eating in a near-supine position. The absence of ST-segment deviation in these situations may provide reassurance that such symptoms are not likely ischemic in nature.

Patients at High Risk for Ischemia After Cardiac or Noncardiac Surgery

The potential benefits of ST monitoring after cardiac surgery are to (1) distinguish incisional from ischemic chest pain, (2) assess graft patency and detect reocclusion, and (3) determine whether postoperative cardiac complications (eg, arrhythmias, heart failure) have an ischemic basis. It is important to point out that experience with ST monitoring after cardiac surgery is limited. Moreover, few if any clinical studies exist to guide clinicians in distinguishing the gradual diffuse ST-T–wave changes that are frequently observed after pericardiotomy from changes that are indicative of acute myocardial ischemia.

The potential benefit of ST monitoring after noncardiac surgery is to detect perioperative ischemia in older adult patients who are at risk of cardiac complications (eg, patients with left ventricular hypertrophy, coronary artery or peripheral vascular disease, or cardiac risk factors).[87,88] The American College of Cardiology/American Heart Association guideline for perioperative cardiovascular evaluation for noncardiac surgical patients[89] supports intraoperative and postoperative ST-segment monitoring in high-risk situations, which they define as patients with emergent major operations (particularly older adults), aortic and other major vascular surgeries, peripheral vascular surgery, and anticipated prolonged surgical procedures associated with large fluid shifts, blood loss, or both.

Mangano et al[90] reported a high-risk period immediately after surgery when the patient emerges from anesthesia and experiences postoperative pain. Such arousal of the sympathetic nervous system is accompanied by an increased heart rate. Therefore, the mechanism of ischemia in the early postoperative period often results from myocardial oxygen demand that exceeds blood flow capability rather than from a coronary occlusion process.

Any adult who is critically ill (especially older adults) and has a high cardiovascular demand may develop myocardial ischemia and associated cardiac complications. Booker et al[91] reported that of 76 patients admitted to an intensive care unit for noncardiac reasons (after noncardiac surgery or other major illness), 8 developed transient myocardial ischemia with 12-lead ST-segment monitoring, and of these, 6 also developed elevated serum troponin levels. The 8 patients with transient ischemia experienced a total of 37 ST events (average of 9 events per patient during a 24-hour monitoring period). Only 2 ST events were accompanied by chest pain (95% were clinically silent). Of the 8 patients with transient ischemia, 6 experienced cardiac complications, including non–ST-elevation MI, acute heart failure, and symptomatic arrhythmia, and 1 patient died.

Several studies of ST-segment monitoring in patients being weaned from mechanical ventilation have shown an increased failure to wean as well as an increased risk of cardiac complications in patients with ischemic events as compared with those without ischemic events.[92–95] Therefore, ST-segment monitoring should be considered intra- and postoperatively, continuing for 24 to 48 h, in patients in any of these high-risk categories.

Pediatric Patients at Risk of Ischemia or Infarction Resulting From Congenital or Acquired Conditions

The use of ST-segment monitoring in the pediatric population has not been extensively studied or documented[96]; however, ischemic mechanisms have been reported in children. These mechanisms include (1) prenatal exposure to cocaine causing coronary vasospasm in infants,[97] (2) cardiotoxicity during the treatment of severe childhood asthma,[98] (3) intraoperative hypoxia during repair of congenital defects,[99] (4) blunt chest trauma,[96] (5) coronary artery disease from Kawasaki disease,[100] (6) acute myocarditis,[101] and a diverse range of other cardiac conditions.[102]

It may not be feasible to perform ST-segment monitoring in hospitalized children because neonatal and pediatric intensive care units may not be equipped with cardiac monitors that have ST-segment measurement software. In addition, little information can be found about the best lead systems for detecting ischemia in the pediatric population or what ECG criteria should be used. For example, the rapid heart rates that are normally observed in pediatric patients may produce nonspecific ST–T-wave changes. Johnsrude et al[102] studied 96 children with documented MI and reported that ST-segment elevation >2 mm was valuable in making the diagnosis. It remains to be seen whether ST-segment monitoring will have a place in pediatric hospital units.

Class III

Patients With Left Bundle-Branch Block

Patients with left bundle-branch block have ST-T waves that markedly deviate in a positive or negative direction, depending on the ECG lead. The steeply sloping ST segments in these patients cause ST amplitude, which usually is measured at a fixed interval after the J point (eg, 60 milliseconds), to vary with heart rate. Because ST-segment monitoring software triggers an alarm for a change in ST amplitude, such patients have frequent false ST alarms, and this leads to staff fatigue and disenchantment with the technology. Patients with right bundle-branch block usually can be monitored successfully because the ST-T wave is not so extremely deviated; however, patients with frequent intermittent right bundle-branch block should not be monitored because of false ST alarms whenever the block appears or disappears.

Patients With Ventricular Pacing Rhythm

QRS morphology in right ventricular pacing rhythm is similar to the pattern of left bundle-branch block. Thus, the same rationale for not monitoring patients with left bundle-branch block applies to patients with ventricular pacemakers, especially those with rate-adaptive pacing (variable heart rates). Patients especially prone to false ST alarms are those who fluctuate between spontaneous rhythm (with a more typical ST segment) and pacing rhythm (with a deviated ST segment).

Patients With Other Confounding Arrhythmias That Obscure the ST Segment

Patients with coarse atrial fibrillation or flutter may have fluctuating ST-segment amplitudes because of chaotic atrial activity that is measured in the ST segment. Intermittent accelerated ventricular rhythm also may interfere with ST monitoring. This rhythm is not uncommon in patients with ischemic heart disease, and episodes may last for 30 to 90 seconds, which is long enough to trigger an ST alarm.

Patients Who Are Agitated

Patients who are restless and confused are difficult to monitor because of frequent false ST alarms that result from a noisy signal.

QT Interval and ECG Monitoring for Detection of Proarrhythmia

Introduction

The QT interval is an indirect measure of ventricular repolarization. Acute increases in the QT interval can be observed in multiple clinical situations and are associated with an increased risk of syncope and sudden death from torsades de pointes ventricular tachycardia. Clinical situations that may lead to QT prolongation include initiation, increased dosage or overdosage of QT-prolonging drugs, ischemia/infarction, electrolyte disorders, sudden decreases in heart rate, and acute neurologic events.

General Considerations in QT Interval Monitoring

The literature lacks consensus about many aspects of QT interval monitoring. For example, it is unclear how the QT interval measurement should be made, what QT interval threshold should be considered dangerously prolonged, whether corrected QT interval measurements are more efficacious in determining risk for torsades de pointes than uncorrected values, what is the best correction formula to use in clinical practice, and much more. Thus, in the section that follows, the recommendations of the present writing group often are based on expert opinion rather than on proven empirical evidence. More important than QT interval monitoring is continuous ECG monitoring with immediate access to defibrillation because certain conditions pose significant risk of life-threatening arrhythmias and cardiac arrest.

The QT interval should be measured from the beginning of the QRS complex to the end of the T wave. Although the onset of the QRS complex is usually readily apparent, the end of the T wave can be difficult to determine. It can be useful to draw a tangent to the steepest downslope of the T wave and define the intersection of this line with the baseline as the end of the T wave. If the T wave is notched, then the end of the T wave should be considered the end of the entire complex. Discrete U waves, which arise after the T wave has returned to baseline, should not be included in the QT interval. It may be difficult to distinguish a prominent U wave fused with the T wave from a bifid T wave that is characteristic of a congenital long QT syndrome.

Because ventricular repolarization time typically increases with slow heart rates and decreases with fast rates, it is assumed that the QT interval should be corrected for heart rate (QT_C) to assess trends in a given patient over time. However, it is important to point out that the QT_C interval has never been validated as a predictor for torsades de pointes. If a patient has an uncorrected QT interval of 0.44 second before initiation of a potentially proarrhythmic agent and has the same value 8 hours later, then the QT_C at these 2 points may be vastly different if the heart rate is different. In this example, if the predrug heart rate were 60 and the postdrug heart rate were 80, then the QT_C measurement before and after the drug would be 0.44 and 0.52 second, respectively.

A normal QT_C is <0.46 second in women and <0.45 second in men. A QT_C >0.50 second in either sex has been shown to correlate with a higher risk for torsades de pointes.[13,103,104] Reported cases of drug-induced torsades de pointes indicate that the vast majority occur in patients with QT_C >0.50 second.[105] It is important to point out that this rule has exceptions. For example, amiodarone causes marked prolongation of the QT interval but is not associated with a high risk for proarrhythmia. Another problem in recommending a QT prolongation criterion for clinical practice is that no threshold has been established below which QT prolongation is considered free of proarrhythmic risk.[106]

The most commonly used QT correction formula in clinical practice is the one introduced by Bazett,[107] QT_C = QT interval divided by the square root of the R-R interval measured in seconds. The adequacy of Bazett's formula has been questioned because some evidence exists that the formula overcorrects the QT interval at fast heart rates and undercorrects it at low heart rates.[108] In a recent report on the value of QT_C in predicting coronary heart disease in 14 548 healthy men and women, only minor differences were seen in the risk stratification provided by 3 rate correction methods,
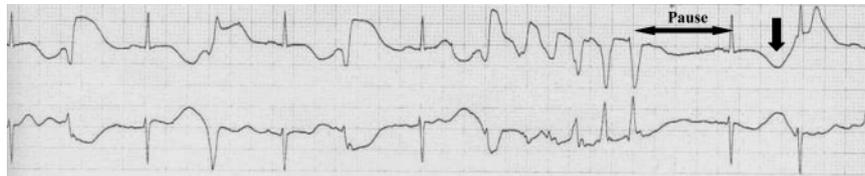
Figure 2. Arrhythmias associated with prolonged QT interval that place patient at immediate risk for developing torsades de pointes. ECG characteristics include underlying prolonged QT interval, T wave alternans, polymorphic ventricular premature beats that fall near T-U portion of repolarization, pause-dependent enhancement of QT interval (arrow), and nonsustained polymorphic ventricular tachycardia.

with the Bazett correction providing slightly better separation.[109] This finding supports the continued use of the Bazett correction method in clinical practice. If a healthcare professional is uncertain about how to calculate QT_C, a standard 12-lead ECG can be recorded. Standard ECG algorithms provide both uncorrected and corrected QT intervals. If the computer measurement of the uncorrected QT interval is confirmed by manual measurement, then healthcare professionals can trust the corrected value of the algorithm.

Because the end of the T wave often is obscure, cardiac monitors do not have algorithms to measure QT intervals and sound an alarm for QT prolongation. Thus, manual measurement by a healthcare professional is necessary.[104] Lead selection for QT interval monitoring should be made by noting which lead of the patient's standard 12-lead ECG has the most well-defined T wave end. The longest QT interval across 12 leads usually is in a mid-precordial lead (typically V_3 or V_4), presumably because these leads are in close proximity to the heart and thus have large amplitude T waves.[110] Lead II is a commonly used lead in the research literature for measuring QT intervals, and if the patient has a normal T wave axis, then a prominent positive T wave will be present in this lead. Moreover, when U waves are present, they often are separated from the T wave in lead II so that QT measurement rather than QT_U measurement is possible.

Regardless of the choice of lead for cardiac monitoring in an individual patient, it is important to make QT measurements in the same lead over time. When monitoring a patient for drug-induced prolonged QT, the clinician should document QT_C in the patient's medical record by using a rhythm strip example before the drug is initiated and thereafter at least every 8 hours. In addition, the QT_C should be documented before and after increases in drug dosage.

Risk Factors for Torsades de Pointes
For the subsequent Class I, II, and III categories, QT interval monitoring is a higher priority if the patient has risk factors for torsades de pointes. Risk factors include older age, female sex, heart disease (especially left ventricular hypertrophy, ischemia, or low left ventricular ejection fraction), slow heart rate, electrolyte abnormalities (especially hypokalemia or hypomagnesemia), starvation diet, acquired or genetic metabolic impairment, genetic predisposition to QT prolongation (as detected by baseline QT prolongation or family history of syncope, sudden death, or long QT syndrome), and the concomitant use of other drugs that prolong the QT interval or impair their metabolism.[104] In addition, patients with an increased QT interval are at immediate risk of torsades de pointes if they exhibit QT-related arrhythmias including

sudden bradycardia or long pauses (eg, compensatory pauses after ventricular ectopy), enhanced U waves, T wave alternans, polymorphic ventricular premature beats, couplets, and nonsustained polymorphic ventricular tachycardia (Figure 2).

Class I

Patients Administered an Antiarrhythmic Drug Known to Cause Torsades de Pointes
Table 3 lists potentially proarrhythmic drugs generally accepted by authorities to have a risk of causing prolonged

TABLE 3. Drugs With Risk of Torsades de Pointes

Generic Name	Brand Name(s)	Clinical Use
Amiodarone	Cordarone Pacerone	Antiarrhythmic
Arsenic trioxide	Trisenox	Cancer/leukemia
Bepridil	Vascor	Antianginal
Chlorpromazine	Thorazine	Antipsychotic
Cisapride	Propulsid	GI stimulant
Clarithromycin	Biaxin	Antibiotic
Disopyramide*	Norpace	Antiarrhythmic
Dofetilide*	Tikosyn	Antiarrhythmic
Domperidone	Motilium	Antiemetic
Droperidol	Inapsine	Sedative, antiemetic
Erythromycin	E.E.S. Erythrocin	Antibiotic
Halofantrine	Halfan	Antimalarial
Haloperidol	Haldol	Antipsychotic
Ibutilide*	Corvert	Antiarrhythmic
Levomethadyl	ORLAAM	Opiate agonist
Mesoridazine	Serentil	Antipsychotic
Methadone	Dolophine Methadose	Opiate agonist
Pentamidine	NebuPent Pentam	Anti-infective Pneumocystitis
Pimozide	Orap	Antipsychotic
Procainamide*	Pronestyl Procan	Antiarrhythmic
Quinidine*	Quinaglute Cardioquin	Antiarrhythmic
Sotalol*	Betapace	Antiarrhythmic
Sparfloxacin	Zagam	Antibiotic
Thioridazine	Mellaril	Antipsychotic

*Requires hospital ECG monitoring. Remaining drugs require hospital monitoring if patient has risk factors for torsades de pointes. Adapted from http://torsades.org/medical-pros/drug-lists/drug-lists.htm.

ventricular repolarization and torsades de pointes. The antiarrhythmic agents that are the most likely to cause proarrhythmia include quinidine, procainamide, disopyramide, sotalol, dofetilide, and ibutilide. Amiodarone often causes marked QT interval prolongation; however, it has a low frequency of torsades de pointes.[111] The recommended time frames for ECG QT interval monitoring include 48 to 72 hours for patients initiating or increasing therapy with quinidine, procainamide, disopyramide, sotalol, and dofetilide, and 4 to 5 hours for patients who are being treated with ibutilide. In patients who receive ibutilide for the treatment of atrial fibrillation, the most likely time for torsades de pointes to occur is at the time of conversion to sinus rhythm when a pause occurs. Locati et al[112] analyzed the Holter monitor recordings of 12 patients who developed drug-induced torsades de pointes and found that all episodes were preceded by a short-long-short cycle length sequence. In patients who develop a prolonged QT_C >0.50 second, the offending drug should be discontinued and ECG monitoring should continue until the agent washes out and the QT_C is observed to decrease.[13,113]

Patients Who Overdose From a Potentially Proarrhythmic Agent
ECG monitoring of the QT interval should continue until drug levels have decreased and evidence of marked QT prolongation or associated arrhythmias is no longer found.

Patients With New-Onset Bradyarrhythmias
Patients who develop complete heart block or long sinus pauses with sick sinus syndrome are prone to develop torsades de pointes, including those who have undergone ablation of the AV junction to produce complete heart block to counteract uncontrolled rapid heart rates.[51] Monitoring should continue until the bradyarrhythmia has resolved or definitive treatment (eg, permanent pacing) has been instituted.

Patients With Severe Hypokalemia or Hypomagnesemia
Patients with severe electrolyte disorders, especially when other risk factors for torsades de pointes are present, should be monitored until the disorder is corrected and no QT-related arrhythmias are present.

Class II
Patients Who Require Treatment With Antipsychotics or Other Drugs With Possible Risk of Torsades de Pointes
Drugs with moderate QT prolonging potential are generally initiated in the outpatient setting. In those rare individuals with a history of QT prolongation but in whom the addition of these drugs is judged necessary, in-hospital cardiac monitoring may be recommended. These antipsychotics are listed on the University of Arizona Center for Education and Research on Therapeutics web site (http://torsades.org/medical-pros/drug-lists/drug-lists.htm).

Patients With Acute Neurological Events
Patients with subarachnoid hemorrhage are especially prone to QT prolongation; however, they rarely develop torsades de pointes. Sommargren et al[114] analyzed nearly 90 000 12-lead ECGs from 227 patients with subarachnoid hemorrhage monitored continuously in the neurological intensive care

unit. During an average of 114 hours of continuous 12-lead ECG monitoring, a prolonged QT_C was present in 73% of the patients and abnormal U waves were present in 20%; however, only 1 patient developed torsades de pointes. Therefore, patients being monitored in a neurological intensive care unit who have a normal QT_C do not require frequent QT interval measurement. Those with a QT_C >0.50 second should be monitored for QT-related arrhythmias and further prolongation of the QT interval.

Class III
Healthy Patients Administered Drugs That Pose Little Risk for Torsades de Pointes
ECG monitoring is unnecessary in patients without baseline QT prolongation or other risk factors for torsades de pointes. The drugs that are unlikely to cause torsades de pointes are listed on the University of Arizona Center for Education and Research on Therapeutics web site.

Cardiac Monitoring Lead Systems
Standard 12-Lead ECG
Although the standard 12-lead ECG is not used for continuous patient monitoring, it is important to appreciate the basics of this type of ECG and how cardiac monitoring leads differ. A total of 10 electrodes are required to record the standard ECG: 1 on each wrist and ankle and 6 across the precordium. Essentially 2 types of leads are recorded. One type is the bipolar leads that measure the potential difference between 2 electrodes (a positive and a negative electrode); I, II, and III are the 3 bipolar leads. The second type is the unipolar leads that measure the potential variation at a single electrode with respect to a reference potential with constant potential obtained by averaging the potentials at the right wrist, left wrist, and left ankle; aVR, aVL, aVF, and V_1 to V_6 are the 9 unipolar leads. Hence, 12 waveforms are derived from the 10 electrodes. It is important to understand that any variation in electrode positioning from that of the standard 12 lead positions will result in altered waveforms. This is of little consequence for rhythm monitoring but it is important when measuring amplitudes for any reason (eg, ST-segment deviation).

Electrode Positioning for Cardiac Monitoring
In contrast to the standard 12-lead ECG in which limb electrodes are placed on wrists and ankles, bedside cardiac monitoring limb electrodes are placed on the torso to reduce muscle artifact during limb movement and to avoid tethering the patient. Therefore, in all subsequent descriptions of lead systems, the right arm (RA) electrode is placed in the infraclavicular fossa close to the right shoulder, the left arm (LA) electrode is placed in the infraclavicular fossa close to the left shoulder, and the left leg (LL) electrode is placed below the rib cage on the left side of the abdomen. The ground or reference electrode (RL) can be placed anywhere, but it is usually placed on the right side of the abdomen.

Currently Used Hospital Monitoring Lead Systems
Simple 3-Electrode Bipolar Lead Monitoring
The oldest and simplest of all cardiac monitoring lead systems are bipolar leads, which as the name suggests, record the
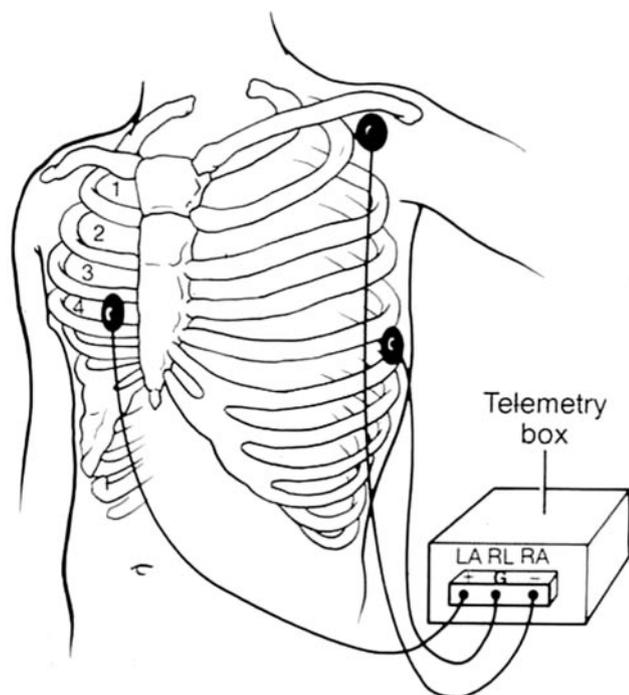
Figure 3. Simple 3-electrode bipolar lead system showing electrode placement for recording single MCL-1 lead. Positive electrode placed in V_1 location and negative electrode placed in left infraclavicular fossa. Reference (ground) electrode shown here in V_6 location; however, it can be placed in any convenient position.

potential difference between 2 electrodes. Leads that can be monitored using this system are lead I (positive electrode, LA; negative electrode, RA), lead II (positive electrode, LL; negative electrode, RA), lead III (positive electrode, LL; negative electrode, LA), or a modified chest lead such as MCL_1 (Figure 3).

Bipolar lead monitoring often is used for portable monitor-defibrillators. The goals of such monitoring are to track heart rate, detect R waves for synchronized direct-current shock in electrocardioversion, and detect ventricular fibrillation. This type of monitoring is inadequate for sophisticated arrhythmia monitoring because a "true" V_1 lead is not available with this system. Lead V_1 is considered the best lead for diagnosing right and left bundle-branch block, to confirm proper right ventricular pacemaker location in temporary transvenous pacing, and to distinguish ventricular tachycardia from supraventricular tachycardia with aberrant ventricular conduction. The bipolar substitute for lead V_1 (MCL_1) has been shown to differ in QRS morphology in 40% of patients with ventricular tachycardia and as such is not recommended for diagnosing wide QRS complex tachycardia.[115] Bipolar lead monitoring also is inadequate for ST-segment monitoring because it does not provide multilead monitoring or precordial leads, which often are the most sensitive for detecting ischemia.

Common 5-Electrode Limb Leads Plus 1 Precordial Lead Combination

A commonly used lead system in current clinical practice is one in which 5 electrodes are used (Figure 4). The 4 limb electrodes are placed in the LA, RA, LL, and RL positions so
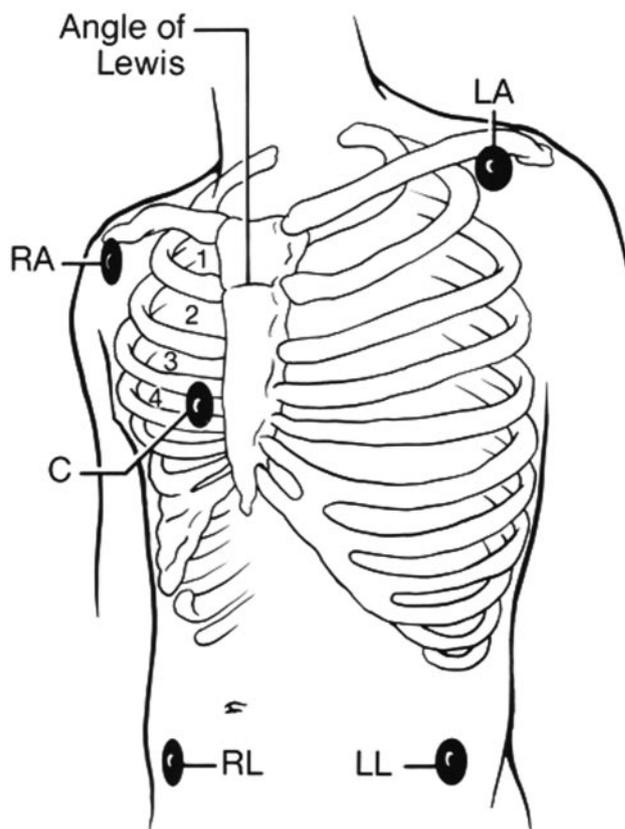

Figure 4. Commonly used 5-electrode lead system that allows for recording any of the 6 limb leads plus 1 precordial (V) lead. Shown here is lead placement for recording V_1. A limitation of this system is that only 1 precordial lead can be recorded.

that any of the 6 limb leads can be obtained (leads I, II, III, aVR, aVL, or aVF). A fifth chest electrode can be placed in any of the standard V_1 to V_6 locations, but in general V_1 is selected because of its value in arrhythmia monitoring. Cardiac monitors with this lead system often have 2 channels generally for ECG display so that 1 limb lead and 1 precordial lead can be displayed simultaneously.

An advantage of this 5-electrode lead system is that it allows the recording of a true V_1 lead, and this prevents the inaccuracy that comes from monitoring with MCL_1. A limitation of this lead system is that >1 V lead cannot be recorded simultaneously. Often, >1 V lead is indicated. For example, although V_1 is an excellent lead for diagnosing arrhythmias with a wide QRS complex (bundle-branch blocks, ventricular pacemaker rhythms, and wide QRS tachycardias), it is insensitive for detecting acute myocardial ischemia.[83,116,117]

10-Electrode Mason-Likar 12-Lead ECG System

In 1966, Mason and Likar[118] introduced a variation on the positioning of the standard limb electrodes specifically designed for 12-lead ECG exercise stress testing (Figure 5). To avoid excessive movement in the lead wires attached to the 4 recording points on the limbs, they suggested that the RA electrode be shifted to the right infraclavicular fossa medial to the border of the deltoid muscle. Similarly, a corresponding position in the left infraclavicular fossa was suggested for the LA electrode. The LL electrode was shifted to the left iliac
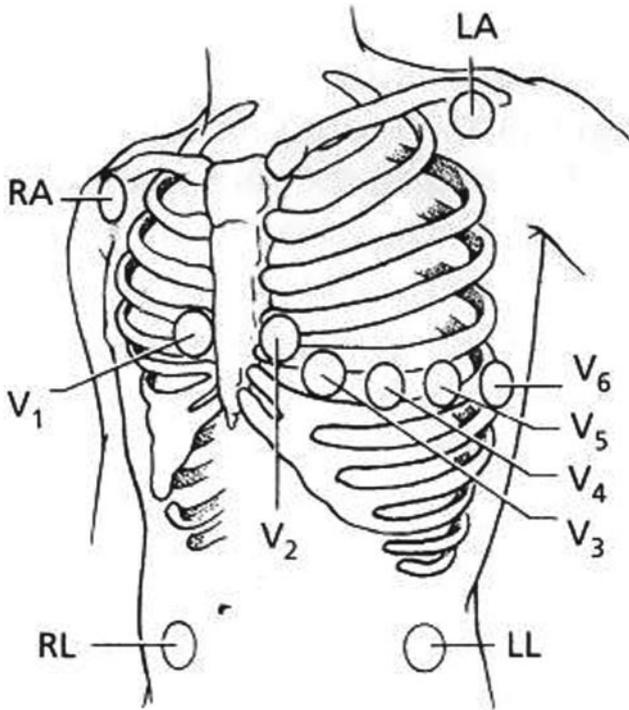
Figure 5. Mason-Likar 12-lead ECG system. Anatomic locations for precordial leads are the same as for recording a standard 12-lead ECG, including V_1 in 4th intercostal space at right sternal margin, V_2 in 4th intercostal space at left sternal margin, V_3 midway between V_2 and V_4, V_4 in 5th intercostal space at left midclavicular line, V_5 in left anterior axillary line at V_4 level, V_6 at left midaxillary line at V_4 and V_5 levels. Anatomic locations for limb leads are relocated from standard 12-lead ECG positions on wrists and ankles to torso.

Figure 6. Frank vectorcardiographic lead configuration. Eight electrodes are required, including 2 on back (behind neck and between scapulae). It is not practical for continuous hospital monitoring because patient must lie on 2 electrodes when supine, which may be uncomfortable and may cause noisy signal.

fossa. The RL electrode could be placed anywhere, but it was usually placed on the right iliac fossa for symmetry.

Several points should be emphasized about this revised version of lead positioning for recording a 12-lead ECG. Limb lead QRS complexes are slightly different in amplitude and axis when extremity electrodes are repositioned on the torso. Precordial leads also may vary slightly because they use the Wilson central terminal as the indifferent electrode, which is made up of LA, RA, and LL leads. Krucoff et al[119] reported that ST-segment measurements were only incidentally affected when electrodes were moved from standard wrist/ankle positions to the torso. Nevertheless, caution should be exercised when comparing serial 12-lead ECGs that include both standard and Mason-Likar recordings.

A major advantage of cardiac monitors using the Mason-Likar 12-lead system is that ST-segment monitoring software has been developed to analyze all 12 leads and to sound an alarm for ST-segment changes, whether or not multiple leads are being displayed on the bedside or central monitor. Therefore, if lead II is being displayed but the patient has a transient ischemic event involving lead V_5, an ST alarm would be triggered. Not all manufacturers that offer the Mason-Likar lead system perform full 12-lead ST-segment analysis nor do they store all 12 leads for printing at a later time. These features should be explored when deciding which cardiac monitors to purchase for a hospital unit. Another advantage of the Mason-Likar lead system is that >1 pre-
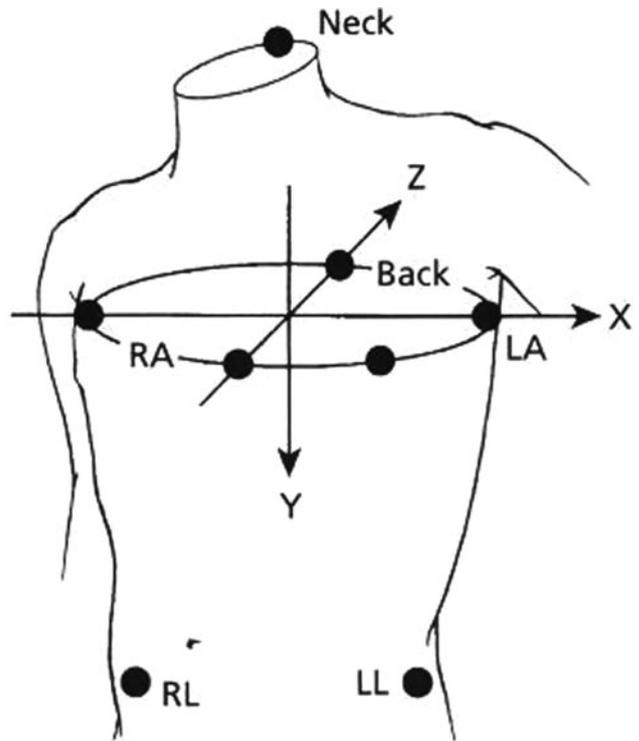
cordial lead can be displayed at the same time. For example, a patient with wide QRS complex tachycardia being monitored after angioplasty of the left anterior descending coronary artery can be monitored with lead V_1 (for arrhythmia) and V_3 (for ischemia).

The disadvantage of the Mason-Likar lead system for cardiac monitoring is that 10 electrodes are required and the 6 precordial electrodes often interfere with diagnostic (eg, echocardiograms, chest x-rays) and emergency (defibrillation sites) procedures. In addition, the precordial sites are difficult to maintain in women with large breasts and men with hairy chests.

8-Electrode Vectorcardiographic Lead System
The first properly designed orthogonal lead system was introduced by Frank.[120] This system provided 3 leads for measuring components of the electrical activity of the heart in 3 mutually perpendicular dimensions: right to left (X), head to foot (Y), and front to back (Z). The positioning of the electrodes is shown in Figure 6.

The Frank lead system is used extensively in Sweden for hospital cardiac monitoring of patients with acute coronary syndromes.[73,121,122] With continuous recordings of the 3 X, Y, and Z leads, a computerized system calculates 2 vectorcardiographic parameters and compares reference ECG waveforms with current waveforms in the 3 leads to display evolutionary QRS and ST-segment trends over time. The 2 vectorcardiographic parameters that are displayed are the QRS vector difference, which quantifies the total changes
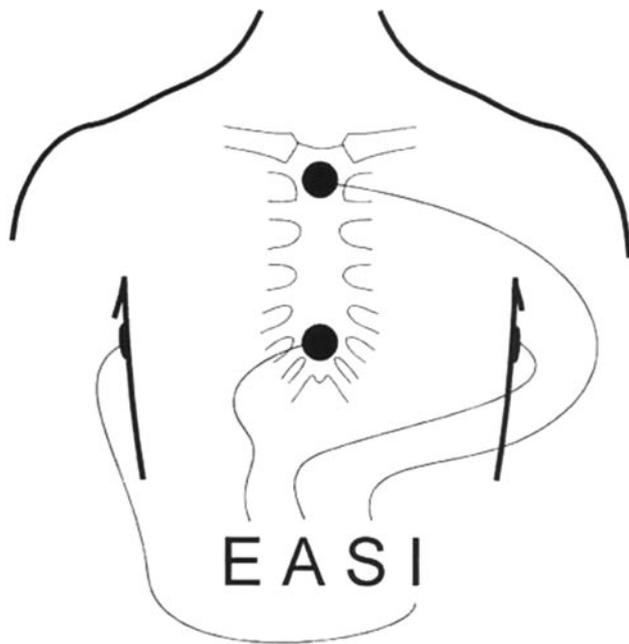
Figure 7. Lead configuration introduced by Dower et al[2] was the first reduced lead set incorporated into cardiac bedside and telemetry monitors. Fifth electrode required for ground electrode. Three leads recorded: lead AS (A positive, S negative), lead ES (E positive, S negative), and lead AI (A positive, I negative). From these 3 directly recorded leads, a 12-lead ECG is derived with same approach to derive 12 leads from 3 vectorcardiographic (X, Y, Z) leads. Adapted from *J Electrocardiol*. 1988;21:S182.

within the QRS complex, and the ST vector magnitude, which quantifies the total ST segment changes measured 20 milliseconds after the QRS complex.

Derived 12-Lead ECG

A synthesized 12-lead ECG that is similar but not identical to the standard 12-lead ECG has been derived from the Frank X, Y, Z leads by Dower et al.[123] Each of the 12 leads in this approach is calculated from a linear combination of the 3 X, Y, Z leads. For example, at any instant the potential in lead I can be determined from the equation $I = aX + bY + cZ$, where a, b, c are fixed coefficients and X, Y, Z represent the potentials recorded in leads X, Y, Z at the same time. Similar equations apply to all 12 leads but with different values of a, b, c. It cannot be stressed too highly that this derived 12-lead ECG is an approximation of the standard 12-lead ECG, and although a close correspondence may exist in many individuals, differences may be found in others. Further examples of derived 12-lead ECGs are given below. Caution always should be exercised when comparing serial ECGs that include both standard and derived ECGs recordings.

Reduced Lead Sets for Obtaining a Derived 12-Lead ECG

Reduced lead set technology is a method of deriving 12 ECG leads from a small number of leads/electrodes. It should be appreciated that given any 2 limb leads, such as I and II, the remaining 4 limb leads can be calculated accurately from these leads. The greater problem is reconstructing precordial

leads from a small number of chest electrodes. Nevertheless, the attraction of minimizing the number of electrodes on the precordium for monitoring purposes to make equipment less bulky and expensive and to simplify matters for patients is clear.

5-Electrode 12-Lead ECG System

Dower et al[2] introduced a reduced lead configuration that requires 4 recording electrodes (plus a fifth ground electrode) as shown in Figure 7. Three leads are recorded with this 5-electrode configuration from which 12 leads are derived similar to the configuration described above for deriving 12 leads from the 3 X, Y, Z leads of the Frank system.[123] As with any 12-lead ECG that differs in electrode number and placement from the standard 12-lead ECG, caution must be exercised when comparing serial ECGs that combine conventionally recorded 12-lead ECGs and those derived from a reduced lead system because QRS, ST, and T waves may differ between the 2 systems.

The 12-lead ECG derived by Dower has been investigated in a series of studies that conclude it is comparable to the standard 12-lead ECG for the diagnosis of wide QRS complex tachycardias[124] and acute myocardial ischemia.[125–127] These investigators also conducted a clinical trial that compared the reduced lead system with routine monitoring for detecting acute myocardial ischemia in 422 patients admitted to the CCU with acute coronary syndromes. They reported that of 463 ischemic events detected with ST-segment monitoring using the reduced lead ECG, 67% showed no evidence of ischemia in routine CCU monitoring leads (V_1 and II), and 80% of the episodes were asymptomatic.[128]

6-Electrode 12-Lead ECG Systems

Drew et al[3] evaluated a new reduced lead system that uses 6 standard electrode sites (Mason-Likar limb leads plus V_1 and V_5) from which the remaining 4 precordial leads are constructed (V_2, V_3, V_4, and V_6). These investigators compared standard and 6-electrode 12-lead ECGs using data from 2 prospective clinical trials involving 649 patients evaluated for chest pain in the ED (ischemia group, n=509) and tachycardias in the cardiac electrophysiology laboratory (arrhythmia group, n=140). They reported that the 6-electrode 12-lead ECG was comparable to the standard ECG for diagnosing multiple cardiac abnormalities, including wide QRS complex tachycardias and acute myocardial ischemia. A practical advantage of this reduced lead set is that standard electrode sites are used, which means that clinicians do not have to learn an unfamiliar lead placement. In addition, the 8 Mason-Likar leads provided in this system probably provide a closer match to the standard 12-lead ECG than if all 12 leads were synthesized.

A second 6-electrode 12-lead ECG system uses 6 standard electrode-monitoring sites (Mason-Likar limb leads plus V_2 and V_5) from which the remaining 4 precordial leads are constructed (V_1, V_3, V_4, and V_6). Thus, similar to the reduced lead set described above, 8 Mason-Likar leads can be obtained. A difference in this reduced lead set is that a "patient-specific" derivation of the 12-lead ECG is an option, which is likely to improve the match with the standard 12-lead ECG.[129] In contrast, other reduced lead sets use fixed

coefficients to derive 12 leads. This patient-specific option requires an initial recording of a standard 12-lead ECG with a cardiograph made by the same manufacturer to calculate the patient-specific coefficients that are necessary to estimate the derived leads. The system defaults to a fixed coefficient method for deriving 12 leads if the prerequisite ECG machine is unavailable.

Recording an Atrial Electrogram

Waldo et al[24] recommended obtaining an atrial electrogram on all postoperative cardiac surgery patients who develop a tachycardia because of the value of seeing unobscured atrial activity in diagnosing the tachycardia mechanism. An atrial electrogram can be recorded with the bedside monitor or with a standard 12-lead ECG machine.[130] The simplest way to record an immediate atrial electrogram at the bedside is to unsnap the chest (V) lead wire from the patient's chest and hold it against the tip of an atrial epicardial pacemaker lead wire so that metal is touching metal. A 15- to 30-second rhythm strip can be printed out, which is in general long enough to diagnose the rhythm. Dual-channel ECG rhythm strips will display a selected limb lead on 1 channel and the atrial electrogram on the "V" channel. Rubber gloves should be worn when handling epicardial pacemaker leads because a small amount of current traveling up the wire directly to the heart can induce ventricular fibrillation in a vulnerable patient. Thus, for electrical-safety reasons, hospitals should develop a policy for recording atrial electrograms.

Staffing, Training, and Methods Improving Quality of ECG Monitoring

Use of Dedicated Monitor Watchers

Any consideration of staffing must begin with a discussion of whether dedicated monitor watchers are necessary. Early analog monitors were not equipped with memory and required constant surveillance. The position of dedicated monitor watcher was created because of the need for continual observation of the central banks of analog monitors on units where the nurse-to-patient ratio was >1:2 or 1:3.[131] Today's totally automated computerized monitoring systems still have not achieved a level of accuracy that is sufficient to eliminate the need for human surveillance. Alarms must be recognized, interpreted, and acted on by a knowledgeable person in a timely fashion.[132] Although the present monitoring systems have improved regarding the detection of arrhythmias and ischemia, the question still remains: Is it necessary for someone to watch the monitors at all times or is it sufficient for nurses to rely on alarms to alert them to problems?[133]

Employing a dedicated monitor watcher has a number of potential benefits.[132,134,135] First, alarms can be immediately reviewed and validated by patient assessment. Second, a monitor watcher may detect subtle warning signs that may not trigger an alarm but may be a harbinger of more serious events (eg, new-onset bundle-branch block during acute MI may precede complete heart block). Third, monitor watchers may be likely to ensure proper lead placement, signal quality, and setting of alarm parameters. Problems such as a lead not directly adhering to the body or a dead battery usually are

lower-grade alarms that do not transmit a continuous audible or visual alarm. If these conditions are not corrected promptly, however, then a patient could experience a serious arrhythmia or ischemic event that would go undetected. Fourth, monitor watchers can free nurses from many activities, such as running rhythm strips and mounting them in the medical record, keeping the equipment in running order, and maintaining an inventory of monitor supplies, which allows nurses to spend more time with patients. Fifth, it is costly and probably not feasible to educate every nurse on the unit to use sophisticated monitoring equipment to its fullest potential. Sixth, in the absence of a dedicated monitor watcher, monitors cannot be continually observed by staff nurses because of their direct patient care responsibilities.

A number of arguments also exist against employing monitor watchers.[132,134,135] First, employing dedicated monitor watchers is costly and may be superfluous considering the increasingly sophisticated monitoring technology available. Money may be better spent on updating monitoring equipment and hiring additional nurses to care for patients. Second, with the advent of improved technology, monitors themselves may be able to do a better job than monitor watchers of notifying the nurse of an arrhythmia or ischemic episode via remote alarms, pagers with the capacity to display a rhythm, or bed-to-bed communications. Third, most monitor watchers must view several screens, each displaying many ECG waveforms, which can be difficult for the human mind to absorb. Watching multiple screens also can have a mesmerizing effect, possibly causing fatigue and decreased vigilance. Fourth, the presence of a monitor watcher may shift the responsibility for detection of arrhythmias away from the nursing staff, thus fostering dependence and impeding the development of their expertise.

Stukshis et al[131] compared the detection accuracy of 4 categories of clinically important arrhythmias for 7 weeks with a monitor watcher and 7 weeks without a monitor watcher. Using a computerized arrhythmia storage system as the gold standard for arrhythmia occurrences, they found that the presence of a dedicated registered nurse monitor watcher on a cardiac progressive care unit was associated with significantly ($P<0.001$) improved accuracy in the detection of nonsustained ventricular tachycardia, supraventricular tachycardia, and pauses. In addition, the detection of life-threatening rhythms (ventricular fibrillation, sustained ventricular tachycardia, other significant tachycardias, severe bradycardia, and asystole) was correct a higher percentage of the time with a monitor watcher (95% versus 88% without a monitor watcher).

In a companion study, Funk et al[133] examined outcomes in 2383 patients on the same cardiac progressive care unit during a 9-month period with a dedicated nurse monitor watcher and a 9-month period without a monitor watcher. They found no significant difference in mortality, frequency of unplanned transfer to an intensive care unit, or occurrence of most life-threatening arrhythmias. In this sample of almost 2400 acutely ill cardiac patients, adverse outcomes occurred with unexpectedly low frequency: 10 deaths, 7 instances of asystole, and 6 episodes of ventricular fibrillation. The small number of these outcomes provided insufficient statistical

power to detect differences in their occurrence with and without a monitor watcher. Of importance, however, was that the presence of a dedicated monitor watcher was associated with significantly fewer episodes of sustained ventricular tachycardia (adjusted OR 0.64, 95% CI, 0.46 to 0.90). The authors suggested that the fewer episodes of sustained ventricular tachycardia may have occurred because a monitor watcher could detect the less-serious precursor rhythms (eg, lengthening QT interval, increase in the number of ventricular premature beats, and nonsustained ventricular tachycardia) and initiate the interventions necessary to prevent sustained ventricular tachycardia.

The current relevance of the findings of these 2 studies, which were conducted in the mid-1990s, is questionable. Although they showed that the presence of a dedicated nurse monitor watcher was associated with greater accuracy in the detection of clinically important arrhythmias and a reduced incidence of sustained ventricular tachycardia, both the clinical environment and monitoring technology have changed. In addition, computerized ST-segment monitoring was not available in the study units. As shown recently,[45,46] silent myocardial ischemia is not uncommon in patients treated on a telemetry unit and is strongly linked to adverse outcomes. Thus, a monitor watcher who is skilled in the interpretation of ST-segment changes of ischemia may provide an even stronger justification for staffing this position.

Some hospitals combine the monitors of several units with 1 monitor watcher at a remote location who can telephone the unit or page a nurse to report a critical rhythm. This arrangement is not ideal and is recommended only if the expertise of the remote monitor watcher is superior and training cannot be provided to nurses on each monitored unit. Other alternatives to dedicated monitor watchers are to have nurses carry pagers that signal when an alarm goes off and display the rhythm strip or to post multiple monitor screens around the unit. Investing in a state-of-the-art monitoring system and educating nurses to use the technology to its fullest potential also could ensure that patient outcomes are not compromised in the absence of a dedicated monitor watcher.

Qualifications of Staff

Cardiac monitoring is performed in a wide variety of hospital units, including critical care, progressive or step-down, EDs, medical-surgical, high-risk obstetrics, cardiac catheterization and electrophysiology laboratories, operating rooms, and postanesthesia recovery. The goals of monitoring are different for each unit. For example, in EDs, ST-segment monitoring is important so that patients with acute coronary syndromes will not be inadvertently sent home. The medical and nursing leadership in each hospital unit with cardiac monitoring should determine what staff proficiencies are required to monitor patients safely and effectively, given the types of patients cared for in a particular unit. Ideally, nursing staff should understand specific ECG abnormalities (Table 4) and general electrophysiologic concepts (Table 5) and be proficient in monitoring skills (Table 6) to work in units in which ECG monitoring is a high priority.

TABLE 4.   Specific ECG Abnormalities

Normal rhythms
    Sinus rhythm
    Sinus bradycardia
    Sinus arrhythmia
    Sinus tachycardia
Intraventricular conduction defects
    Right and left bundle-branch block
    Aberrant ventricular conduction
Bradyarrhythmias
    Inappropriate sinus bradycardia
    Sinus node pause or arrest
    Nonconducted atrial premature beats
    Junctional rhythm
    AV blocks
        1st degree
        2nd degree
            Mobitz I (Wenckebach)
            Mobitz II
            Advanced ($\geq$2:1)
        3rd degree (complete heart block)
    Asystole, pulseless electrical activity
    Sinoventricular rhythm (in severe hyperkalemia)
Tachyarrhythmias
    Supraventricular
        Paroxysmal supraventricular tachycardia (AV nodal reentrant, AV reentrant)
        Atrial fibrillation
        Atrial flutter
        Multifocal atrial tachycardia
        Atrial tachycardia with 2:1 block
        Junctional ectopic tachycardia
    Ventricular
        Accelerated ventricular rhythm
        Nonsustained/sustained monomorphic ventricular tachycardia
        Nonsustained/sustained polymorphic ventricular tachycardia
        Prolonged QT interval-associated ventricular ectopy, torsades de pointes
        Ventricular fibrillation
Premature complexes
    Supraventricular (atrial, junctional)
    Ventricular
Pacemaker electrocardiography
    Failure to capture
    Failure to pace (no pacer output)
    Failure to sense
    Failure to capture both ventricles in biventricular pacing
ECG abnormalities of acute myocardial ischemia
    ST-segment elevation, depression
    T-wave inversion
Muscle or other artifacts simulating arrhythmias

TABLE 5.   General Electrophysiologic Concepts

Automaticity
  Physiologic pacemakers
  Overdrive suppression
Excitation
  Refractory periods
Conduction
  Conduction velocity
  Concealed conduction
  Anterograde and retrograde conduction
Sinus node physiology
  Normal ranges of sinus rate with age
  Effects of autonomic tone
    Vasovagal reactions
    Resting/sleep
    Activity/exercise
  Effects of drugs
AV node physiology
  Effects of atrial rate
  Effects of autonomic tone
    Resting/sleep
    Activity/exercise
  Effects of drugs
Wide vs narrow QRS complexes
QT/U intervals
  Relation to rate
  Gender differences
  Drug effects
  Pause dependency
Observations with arrhythmias
  Sustained vs nonsustained
  Monomorphic vs polymorphic
  Hemodynamically stable vs unstable
  Symptomatic vs asymptomatic
  Association with heart disease vs no heart disease
Hemodynamic effects of arrhythmias
  Influence of rate
  Influence of heart disease
  Influence of A-V synchrony
  Influence of left ventricular synchrony
Implantable devices
  Function of electronic pacemakers, including biventricular pacemakers
  Function of automatic defibrillators
Acute myocardial ischemia
  ST-elevation MI (anterior, inferior, right ventricular; ST recovery indicative of successful reperfusion; reperfusion arrhythmias; intermittent reperfusion; ECG leads related to occlusion of 3 main coronary arteries)
  Non–ST-elevation MI
  Transient myocardial ischemia (effects of body position changes mimicking ischemia)
Syncope
Effects of common antiarrhythmic drugs, rate control vs rhythm control

TABLE 6.   Specific Monitoring Skills

Operation of monitoring system used in hospital unit (arrhythmia, ST monitoring)
Recognition of limitations of computer algorithms
Proper skin preparation for applying electrodes
Accurate electrode placement for system used (eg, reduced lead sets)
Setting heart rate, ST alarm parameters appropriately
Measurement of heart rate
Measurement of intervals (use of ECG calipers)
Recognition of atrial activity
Evaluating pauses
Diagnosis of specific rhythms
Recording of standard 12-lead ECG, landmarks for and importance of accurate lead placement
Recording from postoperative epicardial wires (including electrical safety)
Ability to intervene (unit protocols for responding to, reporting, and documenting)
  Defibrillation/cardioversion
  Patient with bradycardia
  Patient with tachycardia
  Patient with syncope
  Patient with cardiorespiratory arrest
  Patient with implanted device (new or chronic)
  Patient with temporary transvenous pacemaker
  Patient with transcutaneous pacemaker

Staff members who are responsible for ECG monitoring should receive formal orientation and training that is specific to the type of monitoring system being used and to the goals of monitoring for the patient. Appropriate training must include both didactic content and hands-on practice with return demonstration. Being able to demonstrate accurate electrode placement is especially important because inaccurate lead placement is common in hospital units[136] and can result in misdiagnosis (Figure 8).[137]

Documentation

Special attention should be paid to the documentation of the onset and offset of tachycardias because diagnostic clues to arrhythmia mechanism often become evident at those times. Clinicians can use alarm parameters to ensure such documentation by setting the low-rate alarm just below the tachycardia rate. All symptomatic tachy- or bradyarrhythmias and all rhythms that require immediate treatment should be documented in a patient's permanent record. An atrial electrogram should be documented in all postcardiac-surgery patients who develop a tachycardia of unknown origin and who have atrial epicardial pacemaker leads in place. Additional documentation may include extremes of rapid or slow heart rate and representative examples, supplemented by trend reports or statements about the frequency with which such events are observed. Notations on the trend reports about the timing of drugs or other therapies are desirable. When mounting rhythm strips in the medical record, the staff member doing so should place significant changes (eg, onset/offset of a tachycardia) in the center of the page, with adequate time
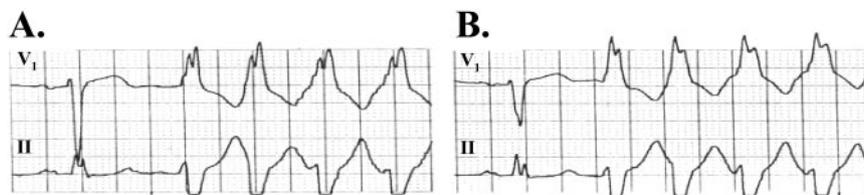
Figure 8. (A) Onset of wide QRS complex tachycardia shows a taller right peak pattern in lead V_1, unhelpful in distinguishing between ventricular tachycardia and supraventricular tachycardia with aberrant conduction. Examination of the patient revealed V_1 electrode misplaced to 5th rather than 4th intercostal space. (B) After lead placement corrected, another episode of wide QRS complex tachycardia showed a taller left peak pattern in lead V_1, strongly suggestive of ventricular tachycardia.[137] Subsequent invasive cardiac electrophysiological study confirmed patient had ventricular tachycardia.

preserved before and after the event to allow a clear understanding of the mechanism. Folding or winding rolls of ECG strips into the chart is not recommended because data are lost when the chart is copied or scanned.

Computerized medical records must be designed to preserve and display the original ECG waveforms at a resolution that is consistent with published guidelines for ECG data quality. It is not acceptable to substitute written diagnostic statements for the tracings themselves.

Documentation With a "Stat" Standard 12-Lead ECG
Information often is contained in the standard 12-lead ECG that is not evident on a monitor rhythm strip of 1 to 2 leads. Medical and nursing leadership in each unit should establish guidelines for what changes in rhythm or patient status should trigger the acquisition of a 12-lead ECG. For units with continuous 12-lead ECG monitoring using Mason-Likar or reduced lead configurations, a 12-lead ECG can be printed out at any time. If comparison with previous standard 12-lead ECGs is important for diagnosis, then a separate recording of a standard 12-lead ECG is necessary.

Methods to Improve the Quality of ST-Segment Ischemia Monitoring
Because ST-segment monitoring is relatively new and vastly underused, clinicians are less skilled in its use. For this reason, the expert writing group devised the following list of strategies to improve the diagnostic accuracy of ST-segment monitoring.

1. **Identification of ST-Segment Fluctuations Resulting From Body Position Changes.** Some patients experience sizable changes in ST-segment amplitudes when they change their body position.[138,139] Such fluctuations are more difficult to distinguish from ischemia than are permanent baseline ST-segment abnormalities because, like ischemic episodes, they are transient. Drew et al[140] reported that a change in body position was the most common cause of false ST alarms. Positional ST-segment changes also have been a cause of unnecessary cardiac catheterization and percutaneous coronary intervention.[4] The most likely explanation for these ST-segment fluctuations is a slight change in heart position relative to the monitoring electrode.[141] A telltale sign of a positional ST-segment change is to observe an associated QRS change. To minimize the risk of possible overtreatment in these patients, healthcare professionals should be taught to evaluate the ST segment in the supine state. Hence, when an ST alarm sounds and a

patient is found in a side-lying position, he or she should be returned to the supine position. If the ST-segment deviation persists in the supine position, it should be considered indicative of myocardial ischemia.

2. **Importance of Careful Skin Preparation.** Because the amplitudes of clinically significant ST changes are as small as 1 mm, a noisy signal presents a major obstacle to an accurate diagnosis. A careful skin preparation that includes shaving electrode sites and removing skin oils and cutaneous debris with alcohol and a rough washcloth is worth the extra minutes because of the time saved in responding to false ST alarms.[142,143]

3. **Importance of Consistent Lead Placement.** It is advantageous to mark electrode locations with indelible ink so that when electrodes are removed (eg, precordial electrodes often are removed during recording of echocardiograms), they can be replaced in the same location. Electrodes located in close proximity to the heart (ie, precordial leads) are especially prone to waveform changes when electrodes are relocated as little as 1 cm away from their original location. A major advantage of continuous ST-segment monitoring as compared with serial 12-lead ECGs is that electrodes stay in place and do not vary as do standard 12-lead ECGs when recorded in different hospital units by different personnel.[144] Nonetheless, when it is necessary to change lead placement (eg, because of the breakdown of skin under an electrode), healthcare professionals should be careful to document such changes on rhythm strips placed in the patient's medical record. When evaluating ST-segment trends graphically, one should note that a clue that an ST change is the result of electrode repositioning is that the change follows a straight-line period when the patient is off the monitor to have electrodes replaced.

4. **Importance of Tailoring ST Alarm Parameters to Patient's Baseline ST Level.** Many current cardiac monitors with ST-segment monitoring software allow clinicians to set the alarm parameters manually. Most patients do not have perfectly isoelectric ST segments; therefore, if alarm parameters are set 1 to 2 mm around the isoelectric line rather than at the patient's baseline ST level, then frequent false alarms will occur. Drew et al[140] reported that in 159 patients admitted to the hospital for nonurgent diagnostic cardiac catheterization, 100 (63%) had a baseline ST-segment deviation of ≥ 100 μV (≥ 1 mm), revealed by ST-segment monitoring. None of the 159 patients was considered to be experiencing acute myocardial ischemia at the time of the ECG recording. The reasons for these baseline ST-segment abnormalities included (1) early repolarization normal variant, (2) left intraventricular conduction

delay, (3) right bundle-branch block, (4) left ventricular hypertrophy "strain" pattern, (5) digitalis effect, and (6) nonspecific ST-T–wave abnormalities. It is recommended that alarm parameters be set at 1 mm above and below the baseline ST level in patients at high risk for ischemia and at 2 mm in more stable patients. The rationale for wider ST alarm parameters in more stable patients is that it greatly reduces the number of false ST alarms, which can occur frequently in more active patients.

5. **Importance of Understanding the Goals of Monitoring in the Individual Patient.** The goal of ST monitoring immediately after thrombolytic therapy for ST-elevation acute MI is to document the rapid recovery of ST-segment deviation. Rapid ST-segment recovery indicates a patent infarct-related artery.[59,145,146] For example, ≥50% reduction in the peak ST elevation lead within 1 hour of thrombolytic therapy indicates a patent vessel.[146] Such rapid changes in the ST segment will trigger alarms, which should be considered "good" alarms. Conversely, a silent ST monitor after thrombolytic therapy suggests no ST-segment recovery and may warrant a more aggressive therapeutic approach.[147] In contrast, the goal of ST monitoring of a patient 48 hours after acute MI is to detect recurrent ischemia. During this period, an ST alarm should be considered a "bad" alarm.

6. **Importance of Analyzing ECG Printout Rather Than Just Graphic Trends.** Most cardiac monitors with ST-segment monitoring software provide displays of ST-segment trends in a single lead or summated leads. Although such graphic trend information is convenient for quickly identifying potential ischemic events, it is important to print out the ECG tracing in question to confirm that the ST-segment changes are the result of ischemia rather than of a transient arrhythmia (eg, an accelerated ventricular rhythm or new bundle-branch block).

Quality Improvement Programs

Medical and nursing professionals in positions of unit and hospital leadership are responsible for the maintenance and improvement of quality in cardiac monitoring. Quality assurance is accomplished, in part, by establishing protocols that govern the roles and responsibilities of all levels of staff regarding cardiac monitoring, documentation of ECG changes, periodic documentation that alarms are set appropriately, and response to emergency and nonemergency cardiac events.

Healthcare professionals also can achieve quality assurance by instituting a mandatory comprehensive orientation and training program that includes all staff involved in cardiac monitoring. Such an educational program should result in staff demonstrating competence in specific cardiac-monitoring skills. Measures of quality, in addition to training and testing, might include such items as time to first shock for life-threatening arrhythmias, diagnostic accuracy of rhythm interpretations, adequacy of staffing (both numbers and training), appropriateness and quality of 12-lead ECGs recorded in response to detected rhythms, timeliness of human review of computer-generated alarms and rhythm strips, and incorporation of clinically significant rhythm strips into the permanent medical record.

After initial orientation and training, a mandatory periodic competency evaluation of all staff should be performed to ensure continued proficiency in critical elements of cardiac monitoring. This evaluation also could include periodic audits of electrode placement and rhythm strip interpretation. Continuing education to reinforce current knowledge and update staff on research findings and techniques should be encouraged and supported.

A periodic review of unit protocols, initial training, staff competency level, and ongoing education efforts should be undertaken at designated intervals to determine whether they continue to meet staff and patient needs. This analysis should include reviews of staff performance, critical events, and patient outcomes.

Conclusion

Cardiac monitoring was introduced >40 years ago; hence, a body of clinical knowledge and research guides best practices in hospital settings. Moreover, it is a well-established fact that arrhythmia monitoring with immediately available defibrillation has improved survival and patient outcomes. In contrast, less is known about the efficacy of ST-segment ischemia monitoring or QT interval monitoring. A consensus of experts who manage patients with acute myocardial ischemia and proarrhythmia is not a substitute for carefully conducted randomized clinical trials. Still, important clinical decisions are made every day with cardiac monitoring data. For this reason, the present consensus document represents the best currently available sources to guide clinical practice in hospital settings with respect to ECG monitoring in children and adults.

Disclosure

Writing Group Member Name	Research Grant	Speakers Bureau/Honoraria	Stock Ownership	Consultant/Advisory Board	Other
Dr Barbara J. Drew	NHLBI; Medtronic Physio-Control; Inovise Medical Philips Medical; General Electric Medical System	None	None	None	None
Dr Robert M. Califf	COR Therapeutics; Daiichi; Merck; Novartis	None	Nitrox LLC	Aventis; Bristol Myers Squibb; Inspire Pharmaceuticals; Johnson & Johnson; King Pharmaceuticals; Millennium; Novartis; Ortho Biotech; Proctor and Gamble; Quintiles	None
Dr Marjorie Funk	None	None	None	None	None
Dr Elizabeth Kaufman	AHA	None	None	None	None
Dr Mitchell W. Krucoff	Medtronic; General Electric Medical Systems; Philips Medical Systems; Northeast Monitoring; Siemens Medical Systems; Quinton Medical; Mortara Instrument; RECOM; Vivometrics; Guidant; St. Jude Medical; Angelmed	None	None	Medtronic; General Electric Medical Systems; Philips Medical Systems; Northeast Monitoring; Siemens Medical Systems; Quinton Medical; Mortara Instrument; RECOM; Vivometrics; Guidant; St. Jude Medical; Angelmed	None
Dr Michael M. Laks	None	None	None	Phillips Medical Systems; Recom, Inc.	None
Dr Peter W. Macfarlane	None	None	None	Quinton Medical Device Manufacturer; Medtronic PhysioControl; Draeger Medical	None
Dr Claire Sommargren	None	None	None	None	None
Dr Steven Swiryn	None	None	Medtronic; Walgreen	None	None
Dr George Van Hare	Medtronic	None	None	Medtronic	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit.

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