

Update to Practice Standards for Electrocardiographic Monitoring in Hospital Settings

A Scientific Statement From the American Heart Association

Endorsed by the American College of Cardiology, American Association of Critical-Care Nurses, and Pediatric and Congenital Electrophysiology Society

BACKGROUND AND PURPOSE: This scientific statement provides an interprofessional, comprehensive review of evidence and recommendations for indications, duration, and implementation of continuous electrocardiographic monitoring of hospitalized patients. Since the original practice standards were published in 2004, new issues have emerged that need to be addressed: overuse of arrhythmia monitoring among a variety of patient populations, appropriate use of ischemia and QT-interval monitoring among select populations, alarm management, and documentation in electronic health records.

METHODS: Authors were commissioned by the American Heart Association and included experts from general cardiology, electrophysiology (adult and pediatric), and interventional cardiology, as well as a hospitalist and experts in alarm management. Strict adherence to the American Heart Association conflict of interest policy was maintained throughout the consensus process. Authors were assigned topics relevant to their areas of expertise, reviewed the literature with an emphasis on publications since the prior practice standards, and drafted recommendations on indications and duration for electrocardiographic monitoring in accordance with the American Heart Association Level of Evidence grading algorithm that was in place at the time of commissioning.

RESULTS: The comprehensive document is grouped into 5 sections: (1) Overview of Arrhythmia, Ischemia, and QTc Monitoring; (2) Recommendations for Indication and Duration of Electrocardiographic Monitoring presented by patient population; (3) Organizational Aspects: Alarm Management, Education of Staff, and Documentation; (4) Implementation of Practice Standards; and (5) Call for Research.

CONCLUSIONS: Many of the recommendations are based on limited data, so authors conclude with specific questions for further research.

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The goals of electrocardiographic monitoring have expanded from simple heart rate and basic rhythm determination to the diagnosis of complex arrhythmias, the detection of acute and often silent myocardial ischemia, and the identification of drug-induced prolonged QT interval. The first American Heart Association (AHA) scientific statement on practice standards for electrocardiographic monitoring in hospital settings was published in 2004¹ and provided an interprofessional, comprehensive review of evidence and recommendations for continuous electrocardiographic monitoring of hospitalized patients.

Since then, however, further data and new issues have emerged that need to be more fully addressed: overuse of arrhythmia monitoring among a variety of patient populations, underuse of QT-interval and ST-segment monitoring among select populations, alarm fatigue, and documentation in electronic health records. For this document, the writing group reviewed research published since 2004 to provide updated recommendations for indications, duration, and implementation indications for continuous electrocardiographic monitoring in hospitalized patients. This document does not contain recommendations for other forms of electrocardiographic monitoring, including the static 12-lead ECG, exercise testing, or ambulatory electrocardiographic (Holter) monitoring.

ORGANIZATION OF THE WRITING GROUP

The 2004 practice standards¹ were commissioned by the AHA Manuscript Oversight Committee on the basis of a proposal from the AHA's Council on Cardiovascular Nursing, along with support from the Council on Cardiovascular Disease in the Young and the Council on Clinical Cardiology. The aim was to encompass all areas of hospital electrocardiographic monitoring, including arrhythmia, ST-segment ischemia, and QT-interval monitoring in both children and adults. The current update to practice standards was similarly approved, with appointments of interprofessional experts from the AHA's Council on Cardiovascular and Stroke Nursing, Council on Cardiovascular Disease in the Young, and Council on Clinical Cardiology. A member of the American College of Cardiology (ACC) was formally appointed for the update to practice standards. The current writing group is composed of experts from general cardiology, electrophysiology (adult and pediatric), and interventional cardiology and included experts in alarm management. The writing group included nurses, cardiologists, and a hospitalist.

METHOD AND EVIDENCE REVIEW

Experts from the writing group were asked to perform a literature review of select topics and inpatient

populations, to evaluate the strength of evidence for electrocardiographic monitoring, and to provide recommendations on indications for electrocardiographic monitoring. Data on costs for electrocardiographic monitoring are limited at this time; therefore, the main outcomes of the current practice standards will continue to be the degree to which evidence exists to support the usefulness and effectiveness of continuous electrocardiographic monitoring. As more studies are published that include financial analyses, these data can be reviewed in future practice standards.

Searches were extended to studies, reviews, and previous related scientific statements or guidelines from the AHA, ACC, American Association of Critical-Care Nurses, or Heart Rhythm Society that were published in English and accessible through PubMed Medline, CINAHL, Cochrane, and other selected relevant databases. Key search words included *arrhythmia*, *dysrhythmia*, *ST-segment monitoring*, *QTc monitoring*, and *torsade de pointes*, among other terms. *ECG monitoring* was searched in the context of patient populations such as *acute coronary syndrome (myocardial infarction, STEMI, NSTEMI, therapeutic hypothermia, targeted temperature management, angina, unstable angina, chest pain, vasospastic angina, percutaneous coronary intervention, open heart surgery)*. *Arrhythmia* was searched with other key words (*ventricular arrhythmias, atrial arrhythmias, sinus bradycardia, atrioventricular block*). Further searches included *ECG monitoring* and diagnoses such as *syncope, heart failure, endocarditis, stroke, drug overdose, electrolyte abnormalities, hemodialysis, and pediatric*. Procedures searched in regard to electrocardiographic monitoring and arrhythmias included *ventricular assist device, ablation, transcatheter valve replacement, pacemakers (transcutaneous, transvenous, permanent), and implantable cardioverter-defibrillator*.

For the organizational aspects of monitoring, literature search terms included *electrocardiography, arrhythmias, cardiac, monitoring (physiologic), nursing assessment, nursing staff, hospital, nursing care, nurse's role, and clinical competence*. Key words/phrases included *in-service, competency, or education*. In addition, the subheadings *education, nursing, standards, diagnosis, cardiac or physiologic monitoring and equipment alarms, and alarm fatigue* were used. Finally, additional articles were garnered from reference lists of literature identified from the initial review.

The recommendations and levels of evidence used in writing the current practice standards were developed by the ACC Foundation/AHA Task Force on Practice Guidelines for Applying Classification of Recommendation and Level of Evidence² and were in effect at the time of the commissioning of this statement (Table 1). Class of Recommendation (COR) according to size of treatment effect is described as COR I (should be performed), IIa (is reasonable to perform), IIb (may be considered), III (no benefit; is not recommended),

Table 1. Applying Classification of Recommendations and Level of Evidence

| | | SIZE OF TREATMENT EFFECT | | | |
|---|--|--|---|--|---|
| | | CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/administered | CLASS IIa <i>Benefit >> Risk</i> Additional studies with <i>focused objectives needed</i> IT IS REASONABLE to perform procedure/administer treatment | CLASS IIb <i>Benefit ≥ Risk</i> Additional studies with <i>broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment MAY BE CONSIDERED | CLASS III <i>No Benefit or CLASS III Harm</i> |
| ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT | LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Sufficient evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Sufficient evidence from multiple randomized trials or meta-analyses |
| | LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Evidence from single randomized trial or nonrandomized studies |
| | LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Only expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Only diverging expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Only diverging expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Only expert opinion, case studies, or standard of care |
| Suggested phrases for writing recommendations | | should be recommended | is reasonable | may/might be considered | COR III: No Benefit |
| Comparative effectiveness phrases [†] | | treatment/strategy A is recommended/indicated in preference to treatment B | treatment/strategy A is probably recommended/indicated in preference to treatment B | may/might be reasonable | COR III: Harm |
| | | treatment A should be chosen over treatment B | it is reasonable to choose treatment A over treatment B | usefulness/effectiveness is unknown/unclear/uncertain or not well established | is not recommended |
| | | | | is not indicated | should not be performed/administered/other |
| | | | | is not useful/beneficial/effective | causes harm associated with excess morbidity/mortality |
| | | | | | should not be performed/administered/other |

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations such as sex, age, history of diabetes mellitus, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

or III (harm; is potentially harmful and should not be performed). Level of Evidence (LOE) for estimates of certainty (precision) of treatment effect are classified as Level A, B, or C (Table 1). It must be noted that a recommendation with LOE C does not imply a weak recommendation.² Some interventions may prove difficult or unethical to test in a randomized design, but there may be a very clear clinical consensus that a particular therapy is useful.

Some interventions have become firmly established as standard of practice without a randomized controlled trial (RCT) and thus seldom receive investigation. This is particularly notable in the case of electrocardio-

graphic monitoring. Electrocardiographic monitoring quickly became the accepted standard of practice in all intensive care units (ICUs) and for many patients on a step-down/progressive care unit. Thus, the standard of care for electrocardiographic monitoring was firmly established before RCTs for almost all electrocardiographic monitoring. For example, patients immediately after myocardial infarction (MI) received electrocardiographic monitoring without patients being randomized to electrocardiographic monitoring versus no monitoring. Thus, the vast majority of studies of patients after MI are not directly evaluating the intervention of electrocardiographic monitoring but clearly demonstrate

that select groups of patients are at risk for significant arrhythmias, ischemia, or QT prolongation. Because data on monitoring as a specific intervention are few (with several exceptions), the writing group considered the vast majority of recommendations for continuous electrocardiographic monitoring to be LOE C (standard of care, consensus opinion of experts). Following the precedent set with the 2004 electrocardiographic monitoring practice standards, the writing group included studies that describe mortality, morbidity, and cost (when available) and the incidence or prevalence of arrhythmias, ST-segment events, or QT prolongation among patient populations, as well as data from experimental studies when available. Certain hospitalized patient populations have not been included in studies of electrocardiographic monitoring. In these populations, the writing group attempted to be clear that recommendations were based primarily on expert consensus. In some cases, the writing group simply stated its inability to provide recommendations because of insufficient data.

Although the original AHA scientific statement¹ presented practice standards within CORs, the current practice standards present recommendations by patient population. The goal is ease of inclusion in routine hospital order sets and quicker access to recommendations for practicing clinicians for day-to-day decision making. This pragmatic organization also allows attention to be directed to inpatient populations for whom electrocardiographic monitoring needs further study.

DOCUMENT REVIEW AND APPROVAL

The completed document was sent for external peer review by experts in the field, including representatives from the AHA and ACC. After passing peer review, it was reviewed for final approval to publish by the AHA's Science Advisory and Coordinating Committee, the highest science body within the association.

PRESENTATION OF CONTENT

This comprehensive document is grouped into 5 sections, the first being Overview of Arrhythmia, Ischemia, and QTc Monitoring. Because continuous electrocardiographic monitoring has 3 main purposes, the writing group provides an overview of each. Because clinicians may be less familiar with continuous ST-segment and QTc monitoring, the overview sections for these topics contain more detail than the overview section for arrhythmia monitoring. The next section is Recommendations for Indication and Duration of Electrocardiographic Monitoring, which we present by patient population to allow practicing clinicians access to recommendations for day-to-day decision

making and to facilitate inclusion in hospital order sets. Pediatric considerations are included when evidence is sufficient to make recommendations or when practice standards are notably different for pediatric patients. Following the recommendations for electrocardiographic monitoring is Section 3, Organizational Aspects: Alarm Management, Education of Staff, and Documentation related to electrocardiographic monitoring, which includes recommendations for these updated and pragmatic areas. The next section, Implementation of Practice Standards, summarizes results of hospitals that have evaluated implementation of the 2004 practice standards, allowing us to learn from their experience. The final section, Call for Research, targets specific areas of highest priority for future research to address current gaps in evidence for electrocardiographic monitoring.

SECTION 1: OVERVIEW OF ARRHYTHMIA, ISCHEMIA, AND QTc MONITORING

Overview of Arrhythmia Monitoring

Background

There are 4 broad rationales for arrhythmia monitoring. The first is immediate recognition of sudden cardiac arrest to improve time to defibrillation. Delayed defibrillation is associated with significantly decreased survival after in-hospital cardiac arrest,³ and occurrence of arrest in an unmonitored patient doubles the likelihood of delayed defibrillation.³ Nurse-to-patient ratios have been identified as the greatest predictor of survival after in-hospital arrest.⁴ It is likely that adequate staffing improves nurses' ability to respond rapidly to monitor-identified alarms. Identification of hospitalized patient populations at sufficient risk of arrest to warrant inpatient monitoring is critical to improve survival.

The second rationale is recognizing deteriorating conditions (ie, development of early afterdepolarizations or nonsustained arrhythmias) that may lead to a life-threatening, sustained arrhythmia and thereby prompting treatment that may prevent or mitigate the effects of a cardiac arrest. Hospitals with the lowest arrest incidence also have the greatest arrest survival.⁴ Although many hospital factors may underlie this association, it is likely that early recognition of patients at risk for arrest improves overall survival. Third, arrhythmia monitoring will, in many situations, facilitate management of arrhythmias even if not immediately life-threatening. Finally, arrhythmia monitoring can facilitate diagnosis of arrhythmias or cause of symptoms (eg, syncope and palpitations) and subsequently guide appropriate management.

Implementation of Arrhythmia Monitoring

Two key points when implementing continuous arrhythmia monitoring are accurate electrode placement and selection of the appropriate leads to monitor.

Accurate Electrode Placement

Unlike the standard 12-lead ECG in which limb electrodes are placed on the arms and legs, limb electrodes for hospitalized patients receiving continuous electrocardiographic monitoring are placed on the torso to allow patient movement while reducing artifact.¹

Accurate electrode placement is key to correct identification of monitored arrhythmias. Incorrect diagnosis of ventricular tachycardia (VT; which was caused by artifact possibly related to a misplaced electrode) among hospitalized inpatients and emergency department (ED) patients has resulted in unnecessary interventions ranging from intravenous antiarrhythmic agent to diagnostic catheterizations and even implantation of a pacemaker and implantable cardioverter-defibrillator (ICD).⁵ Correct placement of electrocardiographic electrodes for both arrhythmia monitoring and continuous ST-segment monitoring cannot be overemphasized. Frequent inaccurate placement of electrodes, particularly the precordial lead, has been reported in a multisite study.⁶

Appropriate Lead Selection

For arrhythmia monitoring, V₁ is commonly selected because of its helpfulness in distinguishing between VT and aberrancy.^{1,7}

Pediatric Considerations

Although V₁ is commonly used for arrhythmia monitoring in adults, lead II is commonly selected as a primary lead for continuous monitoring in the pediatric population because supraventricular arrhythmias are more common than ventricular arrhythmias, and P waves are often best visible in the inferior leads.^{7,8} Age-related variations are found in pediatric ECGs and include heart rate, axis, intervals, and voltage criteria.⁹

Among children, respiratory causes of arrest are more common than cardiac causes; however, hypoxia may lead to bradycardia and subsequent asystole. Cardiac arrest data from 36 902 adults and 880 children (neonatal intensive care and delivery unit excluded) from 253 US and Canadian hospitals revealed that the first documented pulseless arrest rhythm was most commonly asystole or pulseless electrical activity in both children and adults.¹⁰

Overview of Continuous ST-Segment Ischemia Monitoring

Background

The ST segment is the portion of the surface ECG that is coincident with ventricular repolarization. Although

electrocardiographic abnormalities associated with myocardial ischemia or MI may be seen in the PR segment or the QRS complex, the earliest manifestations of myocardial ischemia are usually T-wave and ST-segment changes.¹¹

Because the static 12-lead provides only ≈10 seconds of electrocardiographic waveforms, an extension of this technology has been continuous ST-segment monitoring available for patients most at risk for ischemia. Given the benefits of early management of MI, continuous ST-segment monitoring may facilitate early recognition of ischemic events, potentiating rapid medical treatment and revascularization to avoid permanent myocardial damage in patients presenting with signs and symptoms suggestive of acute coronary syndrome (ACS). Continuous ST-segment monitoring has the potential to identify ischemia before the onset of symptoms. This may be particularly important for patients who are unable to perceive angina symptoms^{12,13} (eg, patients with diabetes mellitus who did not experience angina with a prior MI) or patients who cannot communicate that they are having angina symptoms^{14–17} (eg, patients who are intubated and sedated or with impaired mental status). Alternatively, the absence of dynamic changes on continuous ST-segment monitoring after revascularization can help provide reassurance that chest discomfort does not represent coronary reocclusion.^{1,15} Descriptive studies consistently highlight the prevalence of transient ischemia among patients in ICUs and step-down units.^{18,19}

Verification of Perfusion and Prognostic and Clinical Significance

Despite lack of an RCT evaluating the clinical benefit of continuous ST-segment monitoring in hospitalized patients, many prospective and comparative studies report the clinical and prognostic impact associated with continuous ST-segment monitoring (Table 2). Continuous ST-segment monitoring has been used as a marker of ischemia and infarction in a variety of settings. As early as 1999, investigators prospectively evaluated 100 patients with chest pain and an ECG nondiagnostic of acute MI, finding that transient ST-segment elevation or depression ≥1 mm occurred in 15.9% of the patients, was an independent risk factor for cardiac death or MI, and should be considered an early risk stratification tool.²⁸ A later study of 237 patients admitted for ACS and on a telemetry monitor revealed that 17% had ischemia per continuous ST-segment monitoring. These patients with ischemia were 8.5 times more likely to have in-hospital complications.³⁰

Akkerhuis et al³⁴ conducted a meta-analysis of 3 multisite trials of patients with non-ST-segment-elevation MI (NSTEMI; the Netherlands and United States).

Table 2. Selected Studies of Continuous ST-Segment Monitoring for Verification of Perfusion and Clinical and Prognostic Significance

| |
|--|
| Verification of myocardial perfusion |
| By thrombolytics (Krucoff et al, ²⁰ Langer et al, ²¹ Maas et al, ²² Cruden et al ²³) |
| By percutaneous intervention (Krucoff et al, ²⁰ Terkelson et al ²⁴) |
| By anticoagulants (Jernberg et al ²⁵) |
| By platelet inhibitors (Klootwijk et al ²⁶) |
| By intensive insulin therapy (Stefanidis et al ²⁷) |
| Clinical significance |
| Among patients with chest pain and an ECG nondiagnostic of acute MI, transient ST-segment elevation or depression ≥ 1 mm occurred in 15.9% of the patients and was an independent risk factor for cardiac death or MI (Jernberg et al ²⁸). |
| A 6-h rule-out protocol using cardiac markers and continuous ST-segment monitoring for MI among patients in a chest pain unit demonstrated ST-segment changes on continuous monitoring for 6 patients, leading to early identification of an evolving MI for 2 of these who subsequently received thrombolytics (Herren et al ²⁹). |
| 17% of telemetry patients admitted for ACS had ischemia per continuous monitoring; these patients were 8.5 times more likely to have in-hospital complications (Pelter et al ³⁰). |
| ICU patients with ST-segment depression demonstrated a 4.7-fold higher risk for troponin elevation (Landesberg et al ³¹). |
| After adjustment for risk scores, presence of ST-segment shifts on continuous electrocardiographic monitoring was a stronger independent predictor of mortality than the admission 12-lead ECG (Yan et al ³²). |
| The variability of ST-segment shifts during continuous monitoring in the first 4–24 h of an MI predicted mortality within 5 y, suggesting that continuous ST-segment monitoring during the first 24 h of an MI is a valuable tool for differentiating high- and low-risk patients (Ottander et al ³³). |
| Prognostic significance |
| Variables of continuous ST-segment recovery were predictive for both mortality and the composite risk of mortality, reinfarction, and heart failure (Maas et al ²²). |
| Retrospective, blinded meta-analysis of 3 multisite trials of patients with NSTEMI identified recurrent ischemia episodes in 27% of patients; number of ischemic episodes in 24 h was directly proportionate to the probability of cardiac events at 5 and 30 d. After controlling for known baseline predictors for worse outcomes, each ischemic event predicted a 25% increase for death/MI at 5 and 30 d (Akkerhuis et al ³⁴). |

ACS indicates acute coronary syndrome; ICU, intensive care unit; MI, myocardial infarction; and NSTEMI, non-ST-segment-elevation myocardial infarction.

The 3 clinical trials had evaluated glycoprotein IIb/IIIa inhibitors; a subgroup of patients (n=995) in these sites received additional continuous 12-lead ST-segment monitoring. In a retrospective, blinded analysis, recurrent ischemia episodes were identified in 27% of patients. The number of ischemic episodes in 24 hours was directly proportionate to the probability of cardiac events at 5 and 30 days. At 30 days, the composite end points of MI or death were met by 19.7% of patients with ST-segment episodes compared with only 5.7% of those without ST-segment episodes. After known baseline predictors for worse outcomes were controlled for, each ischemic event predicted a 25% increase in death/MI at 5 and 30 days.

Implementation of Continuous ST-Segment Ischemia Monitoring

It is important to clarify that diagnosis of an MI is not made by continuous ST-segment monitoring. Rather, continuous ST-segment monitoring is used as part of a comprehensive assessment for MI. A 12-lead ECG is done to verify findings of continuous monitoring.³⁵ The 2004 practice standards¹ recommended 1-mm ST-segment deviation as clinically significant for patients in critical care units at high risk of ACS. However, 1- to 2-mm ST-segment elevation or depression that lasts at least 1 minute (with or without symptoms) may be clinically significant, and further clinical assessment is warranted.^{14,18,19,36} Further study is needed to guide optimal alarm thresholds on the basis of several considerations: the patient's potential for developing ischemia, a pragmatic plan for whether this ischemia would be addressed with a feasible and appropriate intervention, the type of nursing unit, and tolerance for potential false alarms, which case reports and studies have revealed to be attributable to patient movement.^{37,38}

Methods Used to Measure ST-Segment Changes

The technology for real-time monitoring for ischemia has been available since the mid-1980s, and practical clinical guidelines were published as a consensus statement in 1999¹⁴ and again within the practice standards in 2004.¹ The 2004 practice standards¹ recommended that aging monitors at end of life be replaced with monitors with automated ischemia monitoring capability.

Unlike arrhythmia monitoring, the software for ischemia monitoring is not automatically enabled by the monitor manufacturers. A thoughtful decision by nurses, physicians, and biomedical engineers at each hospital is critical to identifying an interprofessional protocol for ischemia monitoring, including identification of which hospital units commonly admit the patient populations who may benefit from continuous ischemia monitoring.³⁹ Some hospitals have chosen to have ST-segment monitoring as a default for all patients in ICUs and telemetry units; unfortunately, this is likely to result in overuse of ST-segment monitoring because busy nurses may neglect to turn off the function when a patient does not meet criteria. To reduce unnecessary monitoring and alarms, the writing group recommends setting the ST-segment monitoring default to "off" because only a select group of patients will potentially benefit from this monitoring. Each institution should have clear guidelines and a protocol for nurses to efficiently enable continuous ST-segment ischemia monitoring in patients for whom it is indicated and turn it off when it is no longer indicated for the patient. A physician or advanced practice clinician may indicate when a patient

has transitioned from a clinical condition warranting ischemia monitoring (eg, potential ACS) to no longer needing ischemia monitoring (eg, ACS ruled out). However, because nurses are the caregivers managing the continuous electrocardiographic monitoring 24 hours a day, it is most often the nurse who most quickly identifies when a patient transitions into and out of a paced rhythm or right bundle-branch block (BBB) and can turn off ST-segment monitoring immediately to avoid false alarms. Thus, interprofessional management and communication are essential.

Accurate Electrode Placement and Interpretation

As previously discussed (in Overview of Arrhythmia Monitoring), accurately placed electrocardiographic electrodes are essential to avoid inaccurate diagnoses and potentially inappropriate treatment. The necessity for correct electrocardiographic electrode placement also applies to continuous ST-segment monitoring. Continuous ST-segment monitoring may differ in measurement method compared with exercise stress testing and core laboratories in that a measurement at 60 milliseconds beyond the J point is commonly reported for studies or protocols specific to continuous ST-segment monitoring,^{19,31,39} in an attempt to reduce the potential for false alarms that may occur if the measurement is taken at 80 milliseconds beyond the J point where the upslope of the T wave begins.⁴⁰ Further study is needed.

Appropriate Lead Selection

Ideally, ST-segment monitoring software should have the capability of monitoring all 12 leads simultaneously. In practice, monitoring system capabilities vary, with some monitoring systems able to monitor only 1 precordial lead at a time. Thus, the decision about which precordial lead is selected should be based on the coronary artery or surface known or suspected to be affected by the ischemic process. The principle of using the “ST-segment fingerprint” supports lead selection for noninvasive monitoring. This principle is based on validation of noninvasive ST-segment changes occurring during invasive angiography as being able to accurately signal occlusion via balloon inflation and spontaneous ischemia during early MI.²⁰ Thus, if an artery is identified on angiography to be occluded and leading to ischemia, this ST-segment fingerprint should guide lead selection for postangiographic monitoring. Similarly, if an initial 12-lead ECG demonstrates that a particular lead suggests ischemia, that same lead should be prioritized for continuous ischemia monitoring. Recommendations have differed over the years as to which lead is of most benefit in identifying ischemia in the circumflex artery: I, aVL, V₅ or V₆⁴¹; III or aVF¹⁵; or V₆ for ST-segment elevation and V₁ and V₃ for ST-segment depression⁴² in the circumflex. The disagreement may be attributable to heterogeneity of study

samples, with ischemia in distal versus proximal areas of the circumflex artery and either elevation or depression of the ST segment. Further studies with larger sample sizes may be helpful.

For patients at risk for both arrhythmia and ischemia, selection of the ideal precordial lead can be challenging because at least 1 precordial lead (V₁ or V₆) is needed to assess for ventricular arrhythmias. Thus, it is imperative that if a patient is to receive continuous electrocardiographic monitoring, it is individualized by a clinician competent in ST-segment monitoring.

Nonischemic Causes of ST-Segment Changes

Continuous ST-segment monitoring may not be appropriate for particular patient populations for 2 main reasons: Their clinical condition does not warrant it, or their baseline ECG is abnormal and does not allow valid measurement of the ST segment. A number of conditions other than myocardial ischemia can cause ST-segment changes and trigger ST-segment alarms. For example, changes on the ECG may occur among patients who are admitted with hyperkalemia; their admission “baseline” waveform may include ST-segment elevation with peaked T waves and widening of the QRS complex, often (but not always) throughout the limb and precordial leads.⁴³ As hyperkalemia resolves and the patient’s ST segment returns to baseline, the ST-segment alarm may be triggered because the baseline ST segment stored in the memory of the monitor demonstrated ST-segment elevation, rather than the patient’s outpatient baseline before hyperkalemia.

Similarly, patients who are hypothermic as a result of accidental hypothermia or targeted temperature management may exhibit Osborn waves, which may manifest as prominent J-point elevation mimicking ST-segment–elevation MI (STEMI).^{44,45} Although ST-segment monitoring is recommended for a patient who sustained an ACS-associated arrest and is undergoing therapeutic hypothermia, the ST segment returning to baseline on rewarming may trigger the ST-segment alarm, although this ST resolution to baseline represents a normal physiological response to rewarming. Finally, up to a quarter of patients after defibrillation may exhibit ST-segment elevation, but this usually decreases within 5 minutes.⁴³ The clinician can adjust alarm settings for the patient’s new baseline during resolution of hyperkalemia or hypothermia or after resuscitation to avoid unnecessary alarms.

Primary repolarization abnormalities occur independently, without changes in QRS depolarization,^{40,42} and include the following:

- Drugs and toxins (eg, prolonged use of digitalis may result in what has been described by clinicians as a “scoop” or “soup ladle” ST-segment depression)
- Electrolyte abnormalities (eg, serum calcium and potassium)

- Pericarditis or myopericarditis, often with diffuse changes on the ECG, including ST-segment elevation (or, more frequently, PR depression) in multiple leads, representing inflammation rather than true ischemia⁴³

Secondary repolarization abnormalities may also manifest as ST-segment and T-wave abnormalities. They occur with changes in sequence or duration of ventricular depolarization^{40,42} and include the following:

- Right or left BBB
- Paced rhythms
- Ventricular hypertrophy
- Ventricular pre-excitation

Several other patient populations that present with electrocardiographic waveforms displaying abnormal repolarization are particularly challenging for the clinician on initial admission to the hospital if the patient's history is not well known and no baseline ECG is available. Expert consultation may be indicated. These conditions follow:

- Early repolarization pattern, described as widespread and consistent ST-segment elevation at the J point, with characteristic QRS slurring or notching (a positive deflection on terminal QRS complex); preservation of the initial concave up-sloping; and prominent T waves in at least 2 contiguous leads⁴³
- Chronic or evolutionary ST-segment elevation caused by ventricular aneurysmal dilatation from a previous infarction⁴³
- Brugada syndrome, manifested by right BBB and ST-segment elevation in leads V₁ through V₃ among patients who, despite absence of chest pain, are at high risk for syncope or death resulting from VT or ventricular fibrillation (VF)^{43,46}

Because there are so many patient populations for whom either continuous ST-segment monitoring is not valid or the ST segment is difficult to measure accurately, this monitoring should be considered an add-on rather than a default for in-hospital cardiac monitoring. In contrast, there are discrete patient populations for whom ST-segment monitoring may provide valuable prognostic and clinical data.

Changes to Previous Recommendations for ST-Segment Monitoring

The authors of the 2004 practice standards¹ gave a COR I recommendation for continuous ST-segment monitoring to 4 patient populations (early phase of ACS; chest pain or angina-equivalent symptoms in patients presenting to the ED; after nonurgent percutaneous coronary intervention (PCI) with suboptimal angiographic results; and possible variant angina caused by coronary vasospasm), all of whom were at significant risk of myocardial ischemia, which, if sustained, could result in acute MI.

After considerable discussion resulting in consensus, the current writing group has given these patients a

COR IIa recommendation, not because we doubt the potential for this technology but because recent literature demonstrates a very serious problem with false and nonactionable alarm signals.^{47–53} False and nonactionable alarm signals have led to alarm fatigue and resulted in sentinel events.^{49,54} False alarm signals occur when there is no valid triggering event, whereas nonactionable alarm signals correctly sound but for an event that has no clinical relevance.

False ST-segment alarms^{37,38} were described in early reports and continue to be an area needing priority attention. Investigators evaluated standardization of monitor software and nurse tailoring of patient alarms using a 12-lead hardwire monitoring system in a 15-bed medical progressive care (telemetry) unit over an 18-day period, reporting that after intervention, the number of ST-segment alarms (9647) remained high and needed further follow-up interventions.⁴⁷ In 5 ICUs in a single hospital during a period of 31 days, Drew et al⁵⁰ reported that 91% of the 6196 alarms for ST-segment changes were considered nonactionable. Unfortunately, until this unacceptable rate of false and nonactionable alarms can be addressed, we can no longer give a COR I recommendation because false and nonactionable alarm signals distract the nurse, bother the patient, and desensitize clinicians to respond to alarms.^{47,49,50,55} ST-segment monitoring software in its current state contributes far too many false and nonactionable alarm signals that constrain its usefulness.

Exacerbating the problem of false and nonactionable alarm signals is the lack of studies on the process and outcomes of continuous ST-segment monitoring by staff nurses in telemetry settings. The majority of studies evaluating benefits of continuous ischemia monitoring were done by researchers who used a 12-lead monitoring system.^{18,19,28,30,34} These important studies provided a foundation for others; unfortunately, very few studies were forthcoming to evaluate real-time workflow process and real-time outcomes for the patients as a result of caregivers using their standard continuous monitoring equipment. Ischemia monitoring in non-ICU settings (eg, telemetry units) has not been adequately studied. Literature on appropriate workflow and protocols is rarely published; reports of the impact of protocols for real-time continuous electrocardiographic monitoring are sorely needed. Earlier writing groups provided recommendations during a time when some hospitals still did not have equipment to perform continuous ST-segment monitoring; at that time, hospital leaders were advised that when their cardiac monitors needed to be replaced, they should be replaced with monitors capable of ischemia monitoring.^{1,14} Since that time, most hospitals have had monitoring capability for ischemia; however, the recent practice of unnecessary arrhythmia monitoring combined with suboptimal technology and

Table 3. Recommendations for Continuous ST-Segment Monitoring of Hospitalized Adult Patients

| |
|---|
| Class of Recommendation I |
| None |
| Class of Recommendation IIa |
| Continuous ST-segment monitoring is reasonable for: |
| Early-phase ACS (<24 h) for intermediate to high risk NSTEMI-ACS or STEMI, while receiving definitive diagnosis, initiating immediately and continuing uninterrupted ≥24–48 h (or until ruled out; negative biomarkers) (Level of Evidence B) |
| After MI without revascularization or with residual ischemic lesions (initiating immediately; continuing ≥24–48 h until no evidence of ongoing modifiable ischemia or hemodynamic or electric instability) (Level of Evidence C) |
| Newly diagnosed left main coronary artery lesion (until revascularized) (Level of Evidence C) |
| Vasospastic angina (can be useful to document transient ST-segment changes until diagnosed and stabilized) (Level of Evidence C) |
| After nonurgent PCI with complications or suboptimal results (for ≥24 h or until complication resolved; expert judgment is needed for type of complication) (Level of Evidence C) |
| Open heart surgery (intraoperatively) (Level of Evidence B) |
| Class of Recommendation IIb |
| Continuous ST-segment monitoring may be considered for: |
| After MI with revascularization of all ischemic lesions (initiating immediately; continuing ≥12–24 h after revascularization; duration of monitoring may be shorter or longer, depending on how quickly patient was revascularized, cardiac biomarker levels, and clinical condition) (Level of Evidence B) |
| Apical ballooning (stress cardiomyopathy): until symptoms resolved (Level of Evidence C) |
| During targeted temperature management (therapeutic hypothermia) procedure based on presumed cause of arrest (Level of Evidence C) |
| Open heart surgery: immediately postoperatively in intubated and sedated patients until able to recognize and report new or ongoing ischemia (Level of Evidence B) |
| ADHF: only if possible ischemic origin and evaluable ST segment (until precipitating event is successfully treated) (Level of Evidence C) |
| Stroke: only in patients with acute stroke at increased risk for cardiac events with evaluable ST segments (24–48 h) (Level of Evidence C) |
| Class of Recommendation III: No Benefit; Level of Evidence C |
| Continuous ST-segment monitoring is not beneficial for: |
| Fully awake and alert patients able to recognize and verbalize angina symptoms |
| After nonurgent PCI without complications |
| After routine coronary angiography (no further monitoring beyond femoral sheath removed and immediate postprocedure area) |
| Low-risk and noncardiac chest pain (risk score derived from established scoring tool) |
| Class of Recommendation III: Harm; Level of Evidence C |
| Continuous ST-segment monitoring is potentially harmful because it will likely trigger false or nonactionable alarms that may disturb patients, distract nurses, or lead to unnecessary treatment for: |
| Condition-specific changes in repolarization |
| Myopericarditis |
| Chronic “scooped” ST segment caused by prolonged digitalis use |
| LBBB, RBBB (unless advanced interpretation skills are present) |

(Continued)

Table 3. Continued

| |
|---|
| Paced rhythms |
| General principles and alarm management |
| General principles |
| Assess if ST-segment monitoring is indicated for patient |
| Monitor via 12-lead ECGs; select display leads on the basis of priority purpose of monitoring |
| Assess alarm parameters on the basis of patient’s baseline and purpose of monitoring; adjust as appropriate |
| Continue monitoring until indication no longer relevant |
| Reassess daily |
| If unable to resolve continuous false alarms, discontinue continuous ST-segment monitoring to avoid alarm fatigue; collaborate with the care team to decide whether other assessments are needed (eg, agitated patient with delirium: consider daily ECGs rather than continuous ST-segment monitoring) |
| Document the continuous ST-segment monitoring in patient’s medical record at baseline and then at least every 8–12 h |
| Document waveform strip on ECG with any signs or symptoms of angina or angina equivalent |
| In ACS, continue ST-segment monitoring until MI has been ruled out or other diagnosis is made (eg, myopericarditis) |
| If ACS confirmed, continue continuous ST-segment monitoring using guidelines provided in these practice standards |
| Alarm management |
| Have interprofessional policy in place at institution for use of continuous ST-segment monitoring |
| In responding to alarms, first assesses: |
| Is patient appropriate for continuous ST-segment monitoring (eg, verify rhythm is not paced)? |
| Are electrodes placed correctly? |
| Did ST-segment alarm continue despite change to supine position? |
| After the above are confirmed, obtain 12-lead ECG; qualified clinician should evaluate 12-lead ECG to confirm presence of ST-segment changes in contiguous leads |

ACS indicates acute coronary syndrome; ADHF, acute decompensated heart failure; LBBB, left bundle-branch block; MI, myocardial infarction; NSTEMI-ACS, non-ST-segment elevation acute coronary syndrome; PCI, percutaneous coronary intervention; RBBB, right bundle-branch block; and STEMI, ST-segment elevation myocardial infarction.

suboptimal management of alarms has resulted in an unacceptable rate of false and nonactionable alarms.

Although the number of studies addressing implementation of practice standards for electrocardiographic monitoring has increased in the past few years, the focus has been on arrhythmia monitoring, not continuous ST-segment monitoring.^{56–59} Unfortunately, this has resulted in a continued lack of studies evaluating continuous ST-segment monitoring by telemetry, which often includes only 5 electrodes (7 leads) rather than 10 electrodes (12 leads). Research is ongoing on the practical use of reduced lead sets with capability for 12-lead monitoring directly by staff nurses.⁶⁰

The highest current recommendation for ischemia monitoring is COR IIa, LOE C for intermediate- to high-risk patients in the early phase of ACS, including

those being evaluated for vasospastic angina; post-MI patients without revascularization or with residual ischemic lesions; and newly diagnosed patients with a high-risk lesion such as left main blockage in the setting of a nursing unit with technology for continuous 12-lead ST monitoring and related education and protocols that facilitate reduction of false and nonactionable alarm signals (Table 3). A COR IIb, LOE B recommendation is given for ischemia monitoring immediately postoperatively after open heart surgery, which is congruent with guidelines published in 2011.⁶¹

Technology and protocols for continuous ST-segment monitoring must be improved. The full potential of continuous ST-segment monitoring has not been realized. Monitor manufacturers have a responsibility to work with clinicians to optimize this potentially beneficial technology. Specific recommendations to improve ST-segment technology have been suggested,⁵⁰ and further remedies for addressing alarm fatigue are given in Section 3 of this article; specific recommendations for further studies on ST-segment monitoring are listed in Section 5.

Pediatric Considerations

In neonates and infants, the TP segment is the preferred reference for the isoelectric line.⁶² Changes of ≥ 1 mm above the isoelectric line may be considered clinically significant; however, this is uncommon in the newborn. During the first week of life, variability in T waves is normal. After ≈ 1 week, the T wave is generally negative in V_1 and positive in V_5 and V_6 .⁶²

ST-segment monitoring for primary ischemia is less common among children than adults, although children with congenital heart defects such as Fontan circulation may benefit.^{63,64} In 30 children with Kawasaki disease (a leading acquired heart disease in children in developed nations^{65,66}), ST-segment monitoring via Holter successfully demonstrated ischemic ST-segment depression in all patients with angiographically identified left main coronary artery occlusion.⁶⁷ In children, secondary ischemia resulting from myocardial demand exceeding supply may include those receiving high-dose epinephrine or isoproterenol, infants with prenatal exposure to cocaine,⁶⁸ or infants with cardiotoxicity during treatment of severe asthma.⁶⁹ Thus, further study of ST-segment monitoring among these pediatric populations has been proposed, with clinical recommendations similar to those for adults in terms of degree of elevation/depression, duration of at least 1 minute, and confirmation by a 12-lead ECG.⁶⁷ Further study is needed to support specific recommendations for use among select patient populations, method of monitoring, normal versus abnormal measurements, and effectiveness of interventions.

Table 4. Examples of Drugs Available on US Market With Known Risk for TdP

| Drug Class | Examples |
|----------------|---|
| Antiarrhythmic | Amiodarone, disopyramide, dofetilide, ibutilide, sotalol |
| Antibiotic | Azithromycin, ciprofloxacin, erythromycin, levofloxacin, moxifloxacin |
| Antidepressant | Citalopram, escitalopram |
| Antiemetic | Droperidol, ondansetron |
| Antifungal | Fluconazole |
| Antipsychotic | Haloperidol, chlorpromazine |
| Opiate | Methadone |

TdP indicates torsade de pointes.

Adapted from <http://www.crediblemeds.org>.⁷⁶ Used with permission from AZCERT Inc.

Overview of QTc Monitoring

Background

Acute QT prolongation is associated with increased risk for torsade de pointes (TdP), a rare but potentially fatal VT. QT-interval prolongation is attributable to abnormal structure or function of ion channels and related proteins responsible for cardiac cellular repolarization. In the congenital form of long-QT syndrome (LQTS), these abnormalities result from genetic mutation, whereas in acquired LQTS, ion channel function is altered by acute medical conditions or the action of drugs. Emerging evidence indicates that some instances of drug-induced acquired LQTS and TdP may be related to previously undetected genetic factors.^{70–72} Prevalence of QTc prolongation is difficult to determine because of inconsistent definitions and differing research methods. Investigations have reported prevalence of QTc prolongation >500 milliseconds in monitored patients ranging from 2.6% to 24%.^{73–75}

A major risk factor for acquired LQTS and TdP in hospitalized patients is the initiation, increased dose, or overdose of QT-prolonging drugs. Drugs from a wide range of classes have been implicated (Table 4). AZCERT, Inc, an independent nonprofit organization, maintains a continually updated lists of drugs with known, possible, and conditional risk for causing TdP on its website.⁷⁶ This resource provides a conservative approach to management and is used by clinicians in evaluating the risks and benefits of listed drugs. Ongoing studies of these drugs, as well as new agents, will provide evidence for future recommendations.

Examples of drug classes known to present potential risk for prolonged ventricular repolarization and TdP are listed (Table 4). The degree of risk is variable, and data on the incidence of TdP are best documented for the antiarrhythmic agents. Among the antiarrhythmics, older drugs such as disopyramide, procainamide, and quinidine, as well as dofetilide, ibutilide, and sotalol, have been found to have a TdP incidence of 1% to 10%.⁷⁷ Although amiodarone frequently causes

Table 5. General Risk Factors and Indicators for Impending TdP

| | Examples |
|--|--|
| General risk factors | |
| Older age | |
| Female sex | |
| Heart disease | Left ventricular hypertrophy Low left ventricular ejection fraction Myocardial ischemia |
| Bradycardia | Pause after conversion from AF or flutter to sinus rhythm Compensatory pauses after PVCs Sinus pauses Mobitz II or complete heart block with ventricular rate <40 bpm |
| Electrolyte abnormalities | Hypokalemia (moderate to severe) Hypomagnesemia (moderate to severe) Malnutrition electrolyte disorders |
| Metabolic impairment (acquired or genetic) | Renal failure Hepatic failure |
| Genetic predisposition to QT prolongation | Unexplained QT prolongation in patient or family member Family history of syncope, sudden death, LQTS |
| Concomitant use of drugs that prolong QT or impair their metabolism | |
| Electrocardiographic indicators of impending TdP | |
| Sudden bradycardia or long pauses (eg, compensatory pauses after ventricular ectopy) | |
| Enhanced U waves | |
| T wave alternans | |
| Nonsustained polymorphic VT | |

AF indicates atrial fibrillation; LQTS, long-QT syndrome; PVC, premature ventricular contraction; TdP, torsade de pointes; and VT, ventricular tachycardia. Adapted from Drew et al.⁸⁰ Copyright © 2010, American Heart Association, Inc.

marked QTc interval prolongation, it is thought to less frequently result in TdP.⁷⁸

Administration by intravenous route and rapid infusion of culprit drugs has been demonstrated in animal studies to be more likely to cause arrhythmia.⁷⁹ Other reported risk factors (Table 5) include female sex, family history of congenital LQTS, and underlying conditions such as electrolyte abnormalities, renal or hepatic dysfunction, hypothyroidism, heart disease, and bradycardic episodes.⁸⁰ QT prolongation has also been observed in patients undergoing therapeutic hypothermia after cardiac arrest.^{81,82}

Although assessing the QT interval has become a standard part of the in-hospital monitoring routine, consensus is lacking about specific processes such as measurement methods (manual, semiautomated, fully automated continuous), heart rate correction methods, frequency of QT measurement, and patient selection. Because of the dearth of definitive research findings, many of the recommendations made here are based on expert opinion.

Implementation of QTc Monitoring

Implementation of QTc monitoring involves several considerations: selection of the method to measure the QTc, selection of the appropriate lead to monitor, measurement of the length of the QT interval in seconds or milliseconds, and correction of the QT interval for heart rate (QTc).

Methods Used to Measure the QTc Interval

In monitored hospitalized patients, QT and QTc interval duration can be determined by 3 methods: manual measurement with handheld calipers, a semiautomated approach with digital calipers built into the electrocardiographic monitoring system, and fully automated continuous QTc monitoring. A specific recommendation for 1 of the previously mentioned 3 methods cannot be made at the present time because hospitals have different monitoring systems, some more sophisticated than others, and no studies to date have determined the best method for in-hospital monitoring of QT/QTc. Most institutions currently do not have automatic continuous QT correction in their monitoring systems. Thus, knowing how to measure QT intervals and calculate the corrected QT manually is important and provides human oversight to automatically calculated readings.

Calculating the QTc in patients with atrial fibrillation (AF) is particularly challenging with manual measurement because of the constantly changing RR interval. Although there is no consensus on the best method, >1 approach has been suggested. One method takes the longest and shortest QTc interval in an electrocardiographic recording and calculates an average. Another method takes the average of multiple QTc measurements (up to 10) in a recording.⁸³ Methods that rely on only a single QT measurement are less likely to accurately represent the actual repolarization duration.

Newer-generation hospital electrocardiographic monitoring systems include electronic calipers. Electronic calipers are positioned to measure both the QT and previous RR interval and the values entered. The system then uses an automated QT correction to calculate the QTc. The most recent development is fully automated QTc monitoring, in which QT/QTc intervals are measured every 5 minutes for display, alarms, and trending.^{84,85} The fully automated method has the advantage of measuring every QT interval from all monitoring leads and uses a representative heart rate for correction.

Caution should be used in comparing serial QTc measurements on recordings from bedside monitors with those from standard 12-lead ECGs. Monitor measurements, especially from fully automated continuous QTc monitoring, should not be considered equivalent to, or used interchangeably with, standard 12-lead ECGs for serial comparison.⁸⁶ However, nurses monitoring QTc for at-risk patients every 8 hours via telemetry are able to report increasing measurements, having identified an increase in QTc for which the prescriber would

want to be notified (eg, reaching 500 milliseconds), at which point the prescriber may decide whether a 12-lead ECG is needed to confirm the measurement and adjust treatment accordingly. Patients can be monitored for response to Class III antiarrhythmic drugs (eg, call prescriber if QTc increases $\geq 25\%$ from baseline). Guidelines for the frequency of QTc measurement and notification of prescriber may vary according to patient characteristics and drug.

Appropriate Lead Selection

The AHA/ACC Foundation/Heart Rhythm Society recommendations for the standardization and interpretation of the ECG (2009) recommend selecting the electrocardiographic lead with the longest T wave when monitoring the QT interval and avoiding a lead with U waves.⁴⁰ The same electrocardiographic lead should be used over time for the same patient because QT length varies across the 12 leads. If a lead change after QT monitoring has started is unavoidable, then that lead change should be clearly documented along with the QT measurement.

Measurement of the QT Interval

The QT interval is measured from the onset of the QRS complex to the end of the T wave. If the QRS should become prolonged, for example, with a new BBB, the resultant increase in QT interval should not be interpreted as acquired LQTS.⁸⁷ This situation can be handled by subtracting the increased QRS length from the QT interval.^{40,80} An alternative method is to substitute measurement of the JT interval (from the end of the QRS to the end of the T wave), thereby eliminating the confounding widened QRS.^{40,80} Either method must be documented and used consistently to detect valid ventricular repolarization changes over time. With notched or biphasic T waves or superimposed U wave, the end of the entire T-wave complex should be considered the end of the QT interval. If a U wave is discrete, that is, occurring after the T wave returns to the baseline, then it should not be included in the QT measurement.

Correction of the QT for Heart Rate

Because the QT interval lengthens with slow heart rates and shortens with fast heart rates, it is necessary to correct the QT interval for heart rate to accurately detect repolarization changes over time. The Bazett formula, by which the measured QT interval is divided by the square root of the R-R interval (in seconds), is the most commonly used QT correction method. However, multiple studies have demonstrated that the Bazett method overestimates QTc values at faster heart rates. Numerous alternatives, including the Hodges, Framingham, Fridericia, and subject-specific formulas, have been shown to be more accurate.^{88–91} Although there is currently a lack of consensus on a single optimal formula,

some of these alternatives may be used more frequently in the future.

The QTc interval is considered prolonged when it is >450 milliseconds for male patients and >460 milliseconds for female patients; the difference for sex decreases at ≈ 40 years of age.⁴⁰ Review of studies and expert opinion highlights the clinical significance for QTc duration of >500 milliseconds as being associated with higher risk for TdP.^{80,92–96} Although QTc prolongation criteria have been recommended, there is no firmly established threshold below which QTc prolongation is considered free of proarrhythmic risk.⁸³

General Recommendations for QTc Monitoring

Regardless of the method used, it is essential that all clinicians responsible for electrocardiographic monitoring share a consistent method and procedure within their hospital. Such a QTc protocol should include measurement equipment, electrocardiographic lead selection criteria, use of a consistent lead in the same patient over time, method to identify QRS onset and T-wave offset, QT correction formula, frequency of measurement, and documentation procedure.

It is important to remember that a goal of QTc monitoring is to assess the safety of QT-prolonging medications (eg, Class III antiarrhythmic agents) and to avoid TdP. Thus, astute arrhythmia monitoring must be done concurrently with QTc monitoring. Evidence suggests that a combination of clinical risk factors and QT-prolonging medications may present increased risk for TdP.⁹⁷ Among inpatients with observed prolonged QTc interval, monitoring is a high priority if they exhibit any of the risk factors or specific electrocardiographic indicators of impending TdP (Table 5).

Among patients for whom the provider has selected inpatient initiation, for those with risk factors for TdP (Table 5), including baseline QTc prolongation, who are being started on nonantiarrhythmic drugs with known, possible, or conditional risk for TdP, QT monitoring is recommended. Nonantiarrhythmic drugs with known, possible, or conditional risk for TdP, including many antipsychotic agents, are generally initiated in the outpatient setting without electrocardiographic monitoring. Because studies of nonantiarrhythmic drugs with known risk for TdP consist largely of case reports and small series, data on incidence of TdP are limited.

Although these populations are discussed further in the Monitoring Recommendations by Patient Populations section, patients most likely to benefit (COR I recommendation) from QTc monitoring while hospitalized are highlighted in Table 6.

Pediatric Considerations

The above recommendations may also be applied to pediatric populations, with several caveats noted. The normal upper limit for QTc among children 11 days to

Table 6. Recommended QTc Monitoring of Hospitalized Adult Patients by Population*

| Patient Population/Indication | Recommendations |
|---|--|
| Drug initiation* | |
| Patients with or without risk factors for TdP† who are started on antiarrhythmic drugs with known risk for TdP Medications include dofetilide,‡ ibutilide,‡ sotalol, disopyramide, procainamide, quinidine | QTc monitoring is recommended: For dofetilide (Class I; Level of Evidence B)§ For others (Class I; Level of Evidence C)§ |
| | Factors determining duration of QTc monitoring: QTc return to baseline Drug half-life Time to drug elimination dependent on hepatic or renal function Presence of QT-related arrhythmias Continue QTc monitoring for 48–72 h for patients initiating or increasing dose of disopyramide, procainamide, quinidine, and sotalol |
| Patients with or without risk factors for TdP† who are started on antiarrhythmic drugs with possible risk for TdP Medications include amiodarone, dronedarone, flecainide | QTc monitoring may be reasonable (Class IIb; Level of Evidence C)§ |
| Patients with history of prolonged QTc or with general risk factors for TdP† who are started on nonantiarrhythmic drugs with risk for TdP Drugs with known risk Drugs with possible or conditional risk | QTc monitoring is recommended (Class I; Level of Evidence C)§ QTc monitoring is reasonable (Class IIa; Level of Evidence C)§ |
| Patients without history of prolonged QTc or without general risk factors for TdP† who are started on nonantiarrhythmic drugs with risk for TdP Drugs with known risk Drugs with possible or conditional risk | QTc monitoring is not recommended Class III: No Benefit; Level of Evidence C Class III: No Benefit; Level of Evidence C |
| General principles | |
| For patients with Class I indication for QT monitoring, document the QTc, including rhythm strip, in patient's medical record at baseline and then at least every 8–12 h If QTc prolongation occurs during administration of drug, more frequent measurement may be needed Document QTc before and after increases in dose of QT-prolonging drug In patients who develop QTc >500 ms, discontinue causative drug and continue QTc monitoring until drug washes out and QTc is documented to be decreasing Decision to hold drug will vary on the basis of drug (eg, may not need to hold amiodarone or dronedarone); consult an expert on whether to continue drug when QT prolongation is observed | |

(Continued)

Table 6. Continued

| Patient Population/Indication | Recommendations |
|--|--|
| Targeted temperature management (therapeutic hypothermia) | |
| Patients undergoing targeted temperature management | QTc monitoring is recommended until: Temperature normalized QTc interval is in normal range No evidence of QT-related arrhythmias (Class I; Level of Evidence C)§ |
| Congenital LQTS | |
| Patients with inherited long QT who: Present with unstable ventricular arrhythmias and/or Have medically or metabolically induced prolongation of QTc interval | QTc monitoring is recommended until: Stabilization of ventricular arrhythmias Exacerbating medical or metabolic condition is reversed QTc interval returns to baseline (Class I; Level of Evidence C)§ |
| Electrolyte disorders | |
| Patients with moderate to severe hypokalemia or hypomagnesemia, in combination with other general risk factors for TdP† | QTc monitoring is recommended until: Electrolytes normalized No evidence of QT-related arrhythmias (Class I; Level of Evidence C)§ |
| Drug overdose | |
| Patients with overdose of drug with known TdP risk or with overdose of unknown drug(s) | QTc monitoring is recommended until: QT-prolonging drug levels have decreased Unknown drug has been identified as non-QT-prolonging QTc interval is in normal range No evidence of QT-related arrhythmias (Class I; Level of Evidence C)§ |
| Acute neurological event | |
| Patients with acute neurological events and no baseline QTc prolongation | QTc monitoring is not recommended (Class III: No Benefit; Level of Evidence C) |

LQTS indicates long-QT syndrome; and TdP, torsade de pointes.

*These recommendations apply to patients who are hospitalized. Different recommendations may apply to patients in an outpatient setting. Classification of known, possible, or conditional risk is per <http://www.crediblemeds.org>.⁷⁶

†Risk factors are listed in Table 5.

‡US Food and Drug Administration guidelines apply.

§Document the QTc, including rhythm strip, in the patient's medical record at baseline and then at least every 8 to 12 hours.

Data derived from Zipes et al,⁷⁷ Malik and Camm,⁷⁸ Drew et al,⁸⁰ Storm et al,⁸¹ Riaz et al,⁸² Beach et al,⁹³ and Machado et al.⁹⁸

16 years of age was identified as <450 milliseconds.⁹ A patient with LQTS and a resting QTc of ≥500 milliseconds is generally considered at increased clinical risk for a significant arrhythmia. A shortened QTc interval (ie, <300 milliseconds) may be associated with malignant arrhythmias.⁹⁹ An estimated 10% of sud-

den infant death syndrome is thought to be a result of hereditary QT prolongation. Therefore, in attempt to reduce further QT prolongation by QT-prolonging medications in the setting of existing congenital QT prolongation, clinicians have implemented automated, continuous QT-interval surveillance in the neonatal ICU.¹⁰⁰

SECTION 2: RECOMMENDATIONS FOR INDICATION AND DURATION OF ELECTROCARDIOGRAPHIC MONITORING

Monitoring Recommendations by Patient Populations

The following sections provide a review of evidence for electrocardiographic monitoring in discrete patient populations, with recommendations for electrocardiographic monitoring when sufficient evidence to provide recommendations exists, including a summary of each patient population for which recommendations are provided in the text in Table 7.

Chest Pain and Coronary Artery Disease

Cardiac arrest is the leading cause of death in US adults and the most common cause of death after MI.^{101,102} Although the rate of sudden cardiac death (SCD) has decreased with advanced reperfusion and medical and secondary prevention therapies, it still accounts for 24% to 40% of total mortality after MI,^{101,103} with an overall incidence of 2% to 4% per year.¹⁰⁴ The highest absolute rates of SCD occur within the early hours after MI and during the initial hospitalization, a relatively brief period that accounts for 17% of the sudden deaths likely to occur within the first 30 days after infarction.¹⁰⁴ The substantial risk of death in the early hours and days after MI is a primary motivation for the early recognition, evaluation, and electrocardiographic monitoring of patients with acute ischemic cardiac events.

All patients presenting for urgent assessment of chest pain or symptoms of acute ischemic cardiac disease should be rapidly assessed and treated within the construct of a predetermined chest pain protocol, including prompt initiation of electrocardiographic monitoring and acquisition of a static 12-lead ECG, with early triage of patients with evidence of STEMI to urgent reperfusion therapy.¹⁰⁵ For all patients, appropriate use of electrocardiographic monitoring is essential because the large number of patients admitted for chest pain has an important impact on the availability of monitored beds in some institutions. Indeed, heavy use of telemetry monitoring and ICU admissions is an important driver of increased costs without clear substantiation of improved outcomes in many subsets of patients.^{106–108}

Electrocardiographic monitoring should be purposeful.^{109–111} Thus, researchers are working to differentiate patients whose chest pain is unlikely to be cardiac in origin in an attempt to reduce unnecessary inpatient admissions.

In the absence of STEMI, assessment tools include elements of the history, physical examination, static 12-lead ECG, and cardiac biomarkers to stratify the likelihood of ischemia as high, intermediate, or low. Several authors^{105,112–114} have described the likelihood of signs and symptoms that represent ACS secondary to coronary artery disease. A variety of scoring tools have been developed to assist in identifying patients at presentation to the ED who are at increased likelihood for the presence of ischemia and therefore at higher risk for adverse outcome,^{115–120} although the superiority of these scores beyond clinical judgment is not established. Among patients whose presenting symptoms are found to be the result of a noncardiac cause or deemed to be low likelihood for an ischemic cardiac condition, further evaluation and treatment can be directed in a more appropriate manner without continued electrocardiographic monitoring.

In contrast, when the initial assessment is consistent with an intermediate or high likelihood of ischemic chest pain, further evaluation and management for potential cardiac ischemic conditions are warranted. Risk assessment tools such as the TIMI¹²¹ (Thrombolysis in Myocardial Infarction) risk score, the PURSUIT¹²² (Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy) score, the GRACE¹²³ (Global Registry of Acute Coronary Events) score, or the NCDR-ACTION (National Cardiovascular Data Registry-Acute Coronary Treatment and Intervention Outcomes Network) registry score¹²⁴ incorporate historical, risk factor, biochemical, and electrocardiographic data and have demonstrated ability to predict short-, intermediate-, and/or long-term risk for adverse outcome. Therefore, when applied to patients with ischemic cardiac symptoms, these scores are useful in guiding the intensity of therapeutic management, including level of nursing and monitoring intensity, anticoagulation therapy, and accelerated invasive assessment and revascularization for high-risk patients.¹¹⁴ The following section contains evidence for electrocardiographic monitoring of patients when they are at moderate to high risk for ACS.

Early Phase of ACS (<24 Hours)

Although the incidence of malignant ventricular arrhythmias occurring in the setting of ACS historically has varied considerably from 2% to 20%,^{125–129} limited recent research indicates that the incidence of malignant ventricular arrhythmias in the setting of ACS is less than that reported decades ago.¹³⁰ Nonetheless, because ventricular arrhythmias are known to occur

Table 7. Recommended Electrocardiographic Monitoring of Hospitalized Adult Patients by Population

| Patient Population/Indication | Arrhythmia Monitoring Recommendations | Continuous ST-Segment Ischemia Monitoring Recommendations | QTc* |
|---|--|---|------|
| Chest pain/coronary artery disease | | | |
| Early-phase ACS (<24 h) for intermediate- or high-risk NSTEMI-ACS or STEMI | Should be initiated immediately, continuing uninterrupted ≥24–48 h (or until ruled out; negative biomarkers) (Class I; Level of Evidence B) | Is reasonable to initiate immediately, continuing uninterrupted ≥24–48 h (or until MI ruled out; negative biomarkers or successful reperfusion/revascularization) (Class IIa; Level of Evidence B) | |
| After MI, with revascularization of all ischemic lesions | Should be initiated immediately, continuing uninterrupted ≥12–24 h after revascularization (duration of monitoring after PCI may be shorter or longer, depending on how quickly patient was revascularized, cardiac biomarker levels, and clinical condition) (Class I; Level of Evidence B) | May be considered for immediate initiation, continuing uninterrupted ≥12–24 h after revascularization (duration of monitoring after PCI may be shorter or longer, depending on how quickly patient was revascularized, cardiac biomarker levels, and clinical condition) (Class IIb; Level of Evidence B) | |
| After MI, without revascularization or with residual ischemic lesions | Should be initiated immediately, continuing uninterrupted ≥24–48 h until no evidence of ongoing modifiable ischemia or hemodynamic or electric instability (Class I; Level of Evidence C) | Is reasonable to initiate immediately, continuing uninterrupted ≥24–48 h until no evidence of ongoing modifiable ischemia or hemodynamic or electric instability (Class IIa; Level of Evidence C) | |
| Targeted temperature management | Class I; Level of Evidence C | Decision must be based on presumed cause of arrest (Class IIb; Level of Evidence C) | * |
| Vasospastic angina (ie, Prinzmetal) | Until symptoms resolved (Class I; Level of Evidence C) | Can be useful in patients to document transient ST-segment changes until clinical syndrome diagnosed and stabilized (Class IIa; Level of Evidence C) | |
| Apical ballooning syndrome (stress cardiomyopathy) | Until symptoms resolved (Class I; Level of Evidence C) | May be useful to document until symptoms resolved (Class IIb; Level of Evidence C) | |
| Newly diagnosed left main coronary artery lesion | Until revascularized (Class I; Level of Evidence C) | Until revascularized (Class IIa; Level of Evidence C) | |
| After nonurgent PCI, with complications | For ≥24 h or until complication resolved (Class IIa; Level of Evidence C) | For ≥24 h or until complication resolved (Class IIa; Level of Evidence C) | |
| After nonurgent PCI, without complications | No further monitoring beyond femoral sheath removal and immediate postprocedure area (Class III: No Benefit; Level of Evidence C) | No further monitoring beyond femoral sheath removal and immediate postprocedure area (Class III: No Benefit; Level of Evidence C) | |
| After routine diagnostic coronary angiography | No further monitoring beyond immediate postprocedure area (Class III: No Benefit; Level of Evidence C) | No further monitoring beyond immediate postprocedure area (Class III: No Benefit; Level of Evidence C) | |
| Low-risk and noncardiac chest pain (risk score derived from established scoring tool) | If normal ECG and negative biomarkers (Class III: No Benefit; Level of Evidence B) | If normal ECG and negative biomarkers (Class III: No Benefit; Level of Evidence B) | |
| Major cardiac interventions | | | |
| Open heart surgery | | | |
| Uncomplicated: 48–72 h | Class I; Level of Evidence B | Intraoperatively (Class IIa; Level of Evidence B) and postoperatively in intubated and sedated patients until able to recognize and report new or ongoing ischemia (Class IIb; Level of Evidence B) | |
| High risk for AF: monitor until discharge from acute care unit | Class I; Level of Evidence B | | |
| Mechanical circulatory support | | Only if patient meets respective criteria (ie, signs and symptoms of angina) | |
| Clinically significant cardiovascular or hemodynamic deterioration | Class I; Level of Evidence C | | |
| Immediately after implantation | Class I; Level of Evidence C | | |
| Admitted with noncardiac problems | Class IIa; Level of Evidence C | | |
| Admitted to a rehabilitation facility | Class III: No Benefit; Level of Evidence C | | |

(Continued)

Table 7. Continued

| Patient Population/Indication | Arrhythmia Monitoring Recommendations | Continuous ST-Segment Ischemia Monitoring Recommendations | QTc* |
|--|--|---|------|
| Major cardiac interventions Continued | | | |
| Transcatheter structural interventions | | Not indicated unless ischemic origin is suspected; then follow indications and duration per ischemia criteria | |
| After TAVR, particularly with periprocedural conduction abnormalities | ≥3 d after procedure (<i>Class I; Level of Evidence C</i>) and after day 3 (<i>Class IIa; Level of Evidence C</i>) | | |
| Other transcatheter interventions (eg, VSD, ASD, valvuloplasty) | Duration of monitoring varies with procedure, device, and patient factors (<i>Class I; Level of Evidence C</i>) | | |
| Arrhythmias | | | |
| VTs; postresuscitation from VT/VF cardiac arrest or hemodynamically unstable VT | Until ICD implanted or underlying problem resolved (<i>Class I; Level of Evidence C</i>) | For all arrhythmias, add ST-segment monitoring only if ischemic origin is suspected; then follow indications and duration per ischemia criteria | |
| Nonsustained VT | <i>Class IIb; Level of Evidence C</i> | | |
| Atrial tachyarrhythmias | | | |
| New or recurrent AF: monitor until treatment strategy determined | <i>Class I; Level of Evidence C</i> | | |
| Hemodynamically unstable or symptomatic AF | <i>Class I; Level of Evidence C</i> | | |
| Ongoing rate control management | <i>Class I; Level of Evidence C</i> | | |
| Initiation of new antiarrhythmic agent† | See text; QTc monitoring may be indicated for hospitalized patients | | * |
| Chronic AF | | | |
| If admitted for reason other than arrhythmia or rate and patient are hemodynamically stable | <i>Class III: No Benefit; Level of Evidence C</i> | | |
| If medical condition affects ventricular rate or patient is unstable | <i>Class IIa; Level of Evidence C</i> | | |
| Sinus bradycardias | | | |
| Symptomatic | <i>Class I; Level of Evidence C</i> | | |
| Asymptomatic, significant bradycardia with negative chronotropic medications initiated | <i>Class IIa; Level of Evidence C</i> | | |
| Asymptomatic, hemodynamically stable, admitted for other indication | <i>Class III: No Benefit; Level of Evidence C</i> | | |
| Atrioventricular block | | | |
| Symptomatic second- or third-degree atrioventricular block of any anatomic origin | <i>Class I; Level of Evidence C</i> | | |
| Asymptomatic second- or third-degree block caused by distal conduction system disease | <i>Class I; Level of Evidence C</i> | | |
| Third-degree atrioventricular block caused by intranodal disease | <i>Class I; Level of Evidence C</i> | | |
| Asymptomatic Wenckebach or transient atrioventricular block of vagal origin | <i>Class III: No Benefit; Level of Evidence C</i> | | |
| Congenital or genetic arrhythmic syndromes (eg, WPW, Brugada, LQTS) | | | |
| Hemodynamically unstable, recurrent syncope, increased arrhythmia susceptibility | Until appropriate therapy is delivered (<i>Class I; Level of Evidence C</i>) | | |
| WPW with rapid conduction via accessory pathway during atrial arrhythmia | Until therapy such as antiarrhythmic medication or ablation is delivered (<i>Class I; Level of Evidence C</i>) | | |
| Congenital long QT with unstable ventricular arrhythmias or further QT prolongation induced medically or metabolically | Until stable, exacerbating cause reversed, QTc returned to baseline (<i>Class I; Level of Evidence C</i>) | * | |

(Continued)

Table 7. Continued

| Patient Population/Indication | Arrhythmia Monitoring Recommendations | Continuous ST-Segment Ischemia Monitoring Recommendations | QTc* |
|--|--|--|------|
| Syncope of suspected cardiac origin | | | |
| Meeting admission criteria for syncope, cause of syncope suspected to be cardiac | Monitor ≥24 h; until cause and treatment identified; then follow indications and durations per criteria in these practice standards (<i>Class I; Level of Evidence B</i>) | Not indicated unless ischemic cause is suspected; then follow indications and duration per ischemia criteria | |
| After electrophysiology procedures/ablations | | | |
| Uncomplicated SVT ablation | Can be discontinued after immediate postprocedure area (<i>Class IIb; Level of Evidence C</i>) | For signs and symptoms of ischemia, follow indications and duration per ischemia criteria | |
| Complex ablation (pulmonary vein isolation) or serious comorbidities (eg, heart failure) | Monitor for 12–24 h (duration of monitoring varies with procedure, vascular access, and patient factors) (<i>Class I; Level of Evidence C</i>) | | |
| Atrioventricular nodal ablation after incessant tachycardia and after chronic AF with concomitant pacemaker implantation | Monitor for 12–24 h (<i>Class I; Level of Evidence C</i>) | | |
| After pacemaker or ICD implantation procedures | | | |
| Transcutaneous pacing pads | Monitor until pacing is no longer necessary and the device is removed or replaced with a permanent device (<i>Class I; Level of Evidence C</i>) | <i>Class III: Harm; Level of Evidence C</i> | |
| Standard temporary transvenous pacing wires | Monitor until pacing is no longer necessary and the device is removed or replaced with a permanent device (<i>Class I; Level of Evidence C</i>) | | |
| Semipermanent transvenous pacing | | | |
| Day 1 | <i>Class IIa; Level of Evidence C</i> | | |
| After day 1 | <i>Class IIb; Level of Evidence C</i> | | |
| Permanent pacemaker or ICD | | | |
| Pacemaker dependent | For 12–24 h (<i>Class I; Level of Evidence C</i>) | | |
| Not pacemaker dependent | For 12–24 h (<i>Class IIb; Level of Evidence C</i>) | | |
| Generator change | In immediate postprocedure period (<i>Class IIb; Level of Evidence C</i>) | | |
| Preexisting rhythm devices | | | |
| ICD shocks, requiring hospital admission | For duration of related hospitalization until precipitating event treated (<i>Class I; Level of Evidence C</i>) | <i>Class III: No Benefit; Level of Evidence C</i> | |
| ICD or pacemaker, admission for unrelated indication | <i>Class III: No Benefit; Level of Evidence C</i> | | |
| Stable with wearable defibrillator, admission for unrelated indication | <i>Class III: No Benefit; Level of Evidence C</i> | | |
| Other cardiac conditions | | | |
| Acute decompensated heart failure | Until precipitating event (eg, volume overload; ischemia; anemia; progressive ventricular, respiratory, or renal failure; hypertension; exacerbation of comorbidities; new-onset AF; or infection) is successfully treated (<i>Class I; Level of Evidence B</i>) | Only if possible ischemic origin and in the setting of evaluable ST segments (<i>Class IIb; Level of Evidence C</i>) | |
| Infective endocarditis | Until clinically stable (<i>Class IIa; Level of Evidence C</i>) | <i>Class III: No Benefit; Level of Evidence C</i> | |
| Noncardiac conditions | | | |
| Postconscious sedation | May be of benefit until patients are breathing per baseline and hemodynamically stable; consider that monitoring other than ECG may be more appropriate (eg, oximetry, end-tidal CO ₂) (<i>Class IIb; Level of Evidence C</i>) | Decision based on preoperative cardiac risk assessment | |

(Continued)

Table 7. Continued

| Patient Population/Indication | Arrhythmia Monitoring Recommendations | Continuous ST-Segment Ischemia Monitoring Recommendations | QTc* |
|---|--|--|------|
| Noncardiac conditions Continued | | | |
| Noncardiac surgery | Not indicated among asymptomatic postoperative patients; postoperative patients with angina equivalent symptoms or rhythm changes should be treated according to chest pain/coronary artery disease standards above (Class III: No Benefit; Level of Evidence C) | Only if specific practice standard met (Class III: No Benefit; Level of Evidence C) | |
| Noncardiac major thoracic surgery | After noncardiac major thoracic surgery such as pulmonary resection to identify AF through postoperative day 2–3 and may be helpful until discharge from acute care (Class IIa; Level of Evidence B) | | |
| Medical conditions | | | |
| Stroke | Monitor 24–48 h (Class I; Level of Evidence B) Monitor longer if cryptogenic stroke (to assess for intermittent AF and asymptomatic rapid ventricular response) (Class IIa; Level of Evidence B) | ST-segment monitoring should be considered only in patients with acute stroke at increased risk for cardiac events with evaluable ST-segments (24–48 h) (Class IIb; Level of Evidence C) | |
| Moderate to severe imbalance of potassium or magnesium | Until normalization of electrolytes (Class I; Level of Evidence B) In less severe electrolyte abnormalities, if 12-lead ECG at time of abnormal laboratory result demonstrates electric abnormalities, consider continuous electrocardiographic monitoring | Class III: No Benefit; Level of Evidence C | * |
| Drug overdose | Monitor until free of the influence of the drug(s) and clinically stable (Class I; Level of Evidence B) (see specific recommendations for QTc monitoring in Table 6) | Class III: No Benefit; Level of Evidence C | * |
| Hemodialysis | Efficacy is not well established for most patients receiving chronic hemodialysis unless they have another indication (eg, hyperkalemia, arrhythmia) (Class IIb; Level of Evidence B) (see specific recommendations for QTc monitoring in Table 6) | Class III: No Benefit; Level of Evidence C | * |
| DNR/DNI | | | |
| When data gained from monitoring would trigger interventions consistent with patient wishes (eg, rate control if symptomatic) | Follow practice standards for related conditions | Follow practice standards for related conditions | |
| When data will not be acted on and comfort-focused care is the goal | Class III: Harm; Level of Evidence C | Class III: Harm; Level of Evidence C | |

Need for continuous electrocardiographic monitoring should be reevaluated at least every 24 to 48 hours.

Patients in an intensive care unit and immediate postprocedure area (eg, catheterization laboratory) will have continuous electrocardiographic monitoring.

Patients with Class I indications for arrhythmia monitoring who need to be transported off the unit should have continuous electrocardiographic monitoring via a portable monitor–defibrillator/pacemaker with a healthcare provider skilled in use of the equipment and in electrocardiographic interpretation.

For chest pain/coronary artery disease, complications such as cardiogenic shock or recurrent angina or angina-equivalent syndromes require continued arrhythmia monitoring beyond 24 to 48 hours.

For chest pain/coronary artery disease, reapplication of ischemia monitoring should be considered in previously stable patients who experience recurrent signs/symptoms of ischemia.

For continuous ST-segment monitoring, monitor all 12 leads in the setting of a nursing unit with technology, education, and protocols that facilitate reduction of false and nonactionable alarm signals; not appropriate for patients with uninterpretable ECG (ST segments).

ACS indicates acute coronary syndrome; AF, atrial fibrillation; ASD, atrial septal defect; DNR/DNI, do not resuscitate/do not intubate; ICD, implantable cardioverter-defibrillator; LQTS, long-QT syndrome; MI, myocardial infarction; NSTEMI-ACS, non-ST-segment-elevation acute coronary syndrome; PCI, percutaneous coronary intervention; STEMI, ST-segment-elevation myocardial infarction; SVT, supraventricular tachycardia; TAVR, transcatheter aortic valve replacement; VF, ventricular fibrillation; VSD, ventricular septal defect; VT, ventricular tachycardia; and WPW, Wolff-Parkinson-White.

*QTc monitoring indicated; see comprehensive QTc monitoring recommendations in Table 6.

†For patients who are hospitalized.

in the setting of ACS, most commonly after STEMI or NSTEMI, and have been associated with increased risk of in-hospital mortality, patients in the early phase of ACS should receive arrhythmia monitoring.

Although cardiac risk is highest within the first hours to days of ACS, diagnostic findings, including ST-segment deviation on ECG and biomarker elevation, may not manifest until after the initial presentation with symp-

toms suggestive of unstable angina or non–ST-segment elevation ACS. Therefore, a high index of suspicion must be maintained in patients at increased risk until a diagnosis of NSTEMI can be verified or excluded.¹⁰⁵ All patients at intermediate or high likelihood of ACS should be evaluated rapidly and receive electrocardiographic monitoring without delay.^{131–133} Electrocardiographic monitoring for arrhythmia and ischemia should continue uninterrupted for a minimum of 24 to 48 hours, including during transportation within the hospital, until a definitive noncardiac diagnosis has been established or until appropriate reperfusion or therapy has been provided. This includes patients evaluated in chest pain units for probable or likely ACS who remain at intermediate risk for adverse events after initial evaluation. Arrhythmia and ischemia monitoring are integral components of these short-stay units, which can provide a cost-effective means for the management of patients in the early stages of evaluation for ACS.^{112–114,134,135} Regardless of the patient population, the need for monitoring should be reassessed every 24 hours on the basis of clinical and diagnostic findings and response to therapy, including the absence of major complications such as sustained VT or VF, high-degree atrioventricular block or other evidence of electric instability, or evidence of recurrent or persistent ischemia or hemodynamic instability, including mechanical complication.

Static 12-lead ECGs record only several seconds of an often dynamic physiological process. Because changes on the ECG are associated with increased risk of adverse outcome but may be transient or manifest after presentation, acquisition of a serial 12-lead ECG may improve both diagnostic sensitivity and specificity when performed at 15- to 30-minute intervals in patients with an initial nondiagnostic ECG during evaluation for ongoing symptoms.^{136–138} Alternatively, in patients at increased risk for ACS, continuous ST-segment monitoring may provide additional prognostic information by detection of this transient or evolving electrocardiographic finding. Refractory or recurrent ischemia identified in this way has been shown to offer incremental prognostic information and can serve as a trigger to optimize therapeutic direction.¹³⁸ In a study of 234 intermediate- to high-risk patients with ACS, 23.1% demonstrated ischemic changes identified on continuous ST-segment monitoring. These documented ischemic episodes were associated with an increase in adverse outcome and provided additional prognostic information to the TIMI and PURSUIT risk scores.¹³⁹ Herren et al²⁹ evaluated a 6-hour rule-out protocol using cardiac markers and continuous ST-segment monitoring among patients in a chest pain unit. Among 383 consecutive patients with chest pain, 6 demonstrated ST-segment changes on continuous monitoring, leading to early identification of an

evolving MI for 2 of these who subsequently received thrombolytics.²⁹

Among 968 patients with ACS (the majority of whom had NSTEMI or unstable angina), the conventional 12-lead ECG lacked sensitivity, failing to detect T-wave or ST-segment changes in 80% of these patients.¹⁴⁰ By implementation of a 12-lead ECG from 5 electrodes and continuous ST-segment monitoring transmitted via telephone to the local EDs in northern California, patients (n=4219) with symptoms of ACS had a faster time to first intravenous drug; among patients with STEMI, there was a trend toward faster door-to-balloon time and lower mortality risk.¹⁴¹ In another prospective study of 678 patients with chest pain with suspected ACS, 26 patients had their therapy changed as a consequence of new injury or ischemia identified early through continuous ST-segment monitoring.¹⁴² It is reasonable to implement continuous ischemia monitoring with 12 leads to augment troponins in units with staff who are equipped with the appropriate education, protocol, and resources such as the ED and coronary care unit to improve early risk stratification for select patients with intermediate to high risk of ACS.³⁴

Recommendations

- 1. Arrhythmia monitoring should be initiated immediately in the early phase of evaluation and management of patients at intermediate or high risk of ACS and those with documented STEMI and continue uninterrupted ≥ 24 to 48 hours (or until ruled out; negative biomarkers) (Class I; Level of Evidence B).**
- 2. Continuous ischemia monitoring is reasonable using 12-leads immediately in the early phase of evaluation and management of patients at intermediate or high risk of ACS and those with documented STEMI, continuing uninterrupted ≥ 24 to 48 hours (or until ruled out; negative biomarkers or successful revascularization) (Class IIa; Level of Evidence B).**

After MI, With Revascularization

Continuous arrhythmia and ST-segment ischemia monitoring is an essential component of post-MI care. Identification of ischemic changes or rhythm abnormalities provides important prognostic information and often results in targeted therapeutic modifications during the early period of increased risk.^{143–147} Ischemia monitoring allows recognition of ongoing or recurrent ischemia in a broad segment of patients with ACS while providing important ancillary information in patients during and after reperfusion and revascularization therapy, including culprit artery patency after thrombolytic therapy.^{22,148–151}

and primary PCI.²⁰ Because early, persistent resolution of ischemia is associated with optimal long-term results, identification of recurrent ischemia during early postinfarction monitoring is a critical component of care. Even after angiographically successful primary PCI, failure of ST-segment resolution as a result of poor myocardial perfusion or evidence of recurrent ST-segment elevation caused by reocclusion or infarction extension is associated with worse outcome and should prompt additional evaluation.¹⁵²

Lack of signs of ischemia on continuous monitoring has been suggested as a criterion for low risk and early hospital discharge after MI.¹⁵³ Although the safety of early discharge in select patients after MI has been documented after thrombolytic therapy (GUSTO [Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries]) and primary PCI,^{154,155} early discharge is offered to only a fraction of patients meeting low-risk criteria.¹⁵⁴ Continuous ambulatory ischemia monitoring has been suggested as a readily available prognostic device in the identification of low-risk patients suitable for early hospital dismissal. Bogaty and colleagues¹⁵³ randomized 120 low-risk patients after infarction in a 2:1 fashion to a short hospital course or usual care. Patients assigned to a short hospital course were evaluated with continuous ambulatory electrocardiographic monitoring during hospitalization hours 48 to 72, including a supervised walk up 2 flights of stairs. If ischemic symptoms or ST-segment shifts were identified during this 24-hour monitoring period (21% of randomized patients), early discharge plans were canceled, and these patients were evaluated accordingly. With this strategy, mean hospital length of stay was shortened from 6.9 to 3.5 days without any increase in adverse events or adverse psychosocial effects.

In patients who have undergone successful reperfusion and amelioration of any ischemic substrate, real-time studies in which nurses continuously monitor for ST-segment changes are lacking, and the duration for ischemia monitoring is unclear. Although ischemia monitoring should not be discontinued in patients with recurrent ischemic signs or symptoms or other evidence of continued ischemia, continued ST-segment monitoring in an unselected cohort of patients after infarct may result in a significant burden of nonactionable or false alarms and a potentially hazardous increase in diagnostic and therapeutic actions. Particularly in alert patients in the active recovery phase of MI, asymptomatic ST-segment shifts may occur as a result of position, activity, or even evolutionary changes associated with large infarctions or axis shifts, leading to unnecessary testing and associated cost. Therefore, it is reasonable to consider ST-segment monitoring ≥ 12 to 24 hours after reperfusion, but the duration of monitoring may be shorter

or longer, depending on how quickly the patient was revascularized, cardiac biomarker levels, and clinical condition (COR IIb, LOE B). Reapplication of ischemia monitoring can be considered in previously stable patients after MI who experience recurrent signs or symptoms of ischemia.

Life-threatening tachyarrhythmias and bradyarrhythmias are known complications in patients with STEMI or NSTEMI. Several prospective and retrospective studies have sought to describe the incidence, timing, and predictors of ventricular arrhythmias. Among 3065 patients with STEMI, Mehta et al¹²⁷ found that 133 (4.3%) undergoing primary PCI had VT/VF during the procedure. Also among patients with STEMI, Piccini et al¹²⁹ reported a very low rate of ventricular arrhythmias (1.5%) during hospitalization that were as likely to occur after 48 hours as within 48 hours. However, most studies have reported that ventricular arrhythmias occur early during hospitalization, often before and during revascularization.^{127,156,157}

Piccini et al¹⁵⁷ reported a 5.2% incidence of sustained VT and VF before revascularization in a 3-year retrospective study of patients with acute MI; in-hospital mortality risk was significantly increased in patients with sustained VT or VF (16.3% versus 3.7% in those without VT/VF). Although successful PCI was associated with a reduction in mortality (41% to 14%), patients with VT/VF still had a significantly higher in-hospital mortality than those with no VT/VF. Analysis of data from the Canadian GRACE (Global Registry of Acute Coronary Events) and CANRACE (Canadian Registry of Acute Coronary Events) registries revealed that the rate of sustained VT in patients with STEMI who underwent primary PCI did not differ significantly ($P=0.54$) from the rate in those who received fibrinolysis alone or in combination with rescue PCI or urgent/elective PCI.¹⁵⁸

In patients with STEMI undergoing primary PCI, the incidence of malignant ventricular arrhythmias is $\approx 5\%$ (4.7%–5.7%), with 60% to 64% of ventricular arrhythmias occurring within the first 24 hours of admission and 90% to 92% occurring within 48 hours of PCI.^{128,159} Rahimi et al¹⁶⁰ reported a lower incidence of malignant ventricular arrhythmias in a sample of patients with NSTEMI undergoing PCI (2.6%), with 80% occurring during the first 12 hours and none after 48 hours. The overall incidence of ventricular arrhythmias in patients with acute MI undergoing PCI has been reported as 2.6% to 5.7%.^{127,128,159,160} Because the majority occur within 12 to 48 hours after presentation with STEMI or NSTEMI-ACS, arrhythmia monitoring should be initiated immediately on presentation and continue uninterrupted for 12 to 24 hours after reperfusion therapy (PCI or thrombolytic therapy), including during transportation within the hospital. The appropriateness of early discharge versus continued monitoring needs

to be considered in the context of how quickly the patient was revascularized, cardiac biomarker levels, and clinical condition. The need for monitoring should be reassessed every 24 hours, with a goal of continued monitoring until the patient has been event free for 12 to 24 hours. Duration of monitoring after PCI may be shorter or longer, depending on how quickly the patient was revascularized, cardiac biomarker levels, and clinical condition.

Recommendations

- 1. Arrhythmia monitoring should be initiated immediately on presentation with MI, continuing uninterrupted for ≥ 12 to 24 hours after reperfusion (PCI or thrombolytic therapy) (Class I; Level of Evidence B).**
- 2. Ischemia monitoring may be considered immediately on presentation with MI, continuing uninterrupted for ≥ 12 to 24 hours after reperfusion (PCI or thrombolytic therapy) (Class IIb; Level of Evidence B).**

After MI, Without Reperfusion or Revascularization

Reperfusion and revascularization is the preferred therapeutic course for the vast majority of patients with MI. Despite the recommendation of the ACC/AHA guidelines for revascularization in these patients, nearly 20% of patients with non-ST-segment elevation ACS and either 3-vessel disease or left main disease identified with coronary angiography during hospitalization, who are known to benefit from revascularization, were managed medically.¹⁶¹ During the first 24 hours of admission for patients with ACS, of whom 73% did not undergo PCI, Winkler et al¹³⁰ found that potentially life-threatening arrhythmias occurred rarely: <1% developed asystole, TdP, or VF, and only 1% had sustained VT. In this sample, 8.63% were diagnosed with STEMI, 26.62% with NSTEMI, and 64.75% with unstable angina. The majority (94%) did not require treatment with antiarrhythmic medication, suggesting that even when early reperfusion is not used, life-threatening arrhythmias do not occur with great frequency.

Using data from the MERLIN-TIMI 36 trial (Metabolic Efficiency With Ranolazine for Less Ischemia in Non-ST-Elevation Acute Coronary Syndrome—Thrombolysis in Myocardial Infarction 36), Scirica et al¹⁶² found that nonsustained VT occurred commonly after non-ST-segment elevation ACS: 56.4% had at least 1 episode of at least 3 consecutive beats. Continuous event recorders captured episodes of nonsustained VT for the first 7 days after randomization, and subjects were followed up for 1 year. Although there was no difference in the risk of SCD between patients with ventricular triplets and no VT, 4 to 7 and ≥ 8 consecutive beats

of nonsustained VT were independently associated with an increased risk of SCD, particularly when it occurred after 48 hours. It is noteworthy that having ≥ 8 beats in the first 24 to 48 hours was not associated with a significant risk of SCD. The prognostic impact of nonsustained VT for mortality is well known if left ventricular ejection fraction is <40%, and electrophysiology studies may be considered, or revascularization may be considered for an NSTEMI. However, there is no guideline-based recommendation for continuous electrocardiographic monitoring in the setting of nonsustained VT.

Mehta et al¹²⁸ similarly found that late VT was associated with poorer outcomes and noted that postprocedural TIMI flow <3, lack of β -blocker use on admission, and ST-segment resolution <70% were correlated with late VT/VF. Factors including prior MI/Q waves on admission, previous heart failure, brain natriuretic peptide >80 pg/mL, use of diuretics/hypokalemia, heart rate >100 bpm, left ventricular ejection fraction <40%, lower systolic blood pressure, chronic kidney disease/elevated creatinine, chronic obstructive pulmonary disease (COPD), elevated white blood cell count, older age, and higher Killip class were associated with mortality.^{157,159,160,163}

There is disagreement about the need to continue electrocardiographic monitoring of patients with ACS beyond 24 to 48 hours. Although several studies have found that ventricular arrhythmias occur infrequently beyond 48 hours after MI,^{159,160} data in this setting are conflicting. It is recommended that these patients be monitored for at least 24 hours and until there is no evidence of modifiable ongoing ischemia or hemodynamic or electric instability. Because factors associated with greater mortality have been identified, it is reasonable to consider continuous electrocardiographic monitoring 24 to 48 hours for patients who manifest an ongoing remediable problem that is amenable to further therapeutic intervention. As important as it is to identify patients who require increased (longer) monitoring, it is equally important to identify low-risk patients who may be candidates for early discharge.

Recommendations

- 1. Arrhythmia monitoring should be initiated immediately after MI when there is no reperfusion or revascularization, continuing uninterrupted for ≥ 24 to 48 hours and until there is no evidence of ongoing modifiable ischemia or hemodynamic or electric instability (Class I; Level of Evidence C).**
- 2. Continuous ischemia monitoring is reasonable for patients with ongoing or untreated myocardial ischemia or infarction for ≥ 24 to 48 hours, continuing uninterrupted until**

there is no evidence of ongoing modifiable ischemia or hemodynamic or electric instability (Class IIa; Level of Evidence C).

Targeted Temperature Management

Ischemia monitoring may be useful for patients treated with therapeutic hypothermia who are at high risk for the development of recurrent ischemia after cardiac arrest. However, careful assessment is needed to differentiate between new ischemic or injury patterns and the effects of hypothermia on ST-segment morphology to avoid unnecessary and potentially harmful diagnostic and therapeutic responses.

Mild therapeutic hypothermia is recommended to improve outcome after resuscitated cardiac arrest.^{164–166} Although mild hypothermia has been defined as a target body temperature of 32°C to 33°C and has been associated with improved neurological outcome and survival in this population, this degree of cooling slows impulse conduction through all cardiac tissue, resulting in prolongation of all electrocardiographic intervals and elevation of the J point, resulting in characteristic Osborn waves, with the height of the Osborn wave proportional to the degree of hypothermia.^{167–169} Additional changes on the ECG have also been recorded in response to both accidental and therapeutic hypothermia, including AF in up to 50% of patients,¹⁷⁰ ST-segment depression,¹⁷¹ ST-segment elevation,¹⁷⁰ Brugada syndrome morphology,¹⁷² and QT prolongation.^{81,82} All these findings may confuse interpretation of the ECG in patients treated with therapeutic hypothermia with core body temperatures <35°C. Because mechanical ventilation and sedation are essential components of the care process in patients being treated with therapeutic hypothermia, arrhythmia and ischemia monitoring can herald the otherwise silent occurrence or recurrence of arrhythmia, myocardial ischemia, and myocardial injury. However, because of the frequent changes on the ECG attributable to the hypothermic state, including potentially confounding Osborn waves and other ST-segment deviations in >30% of patients undergoing therapeutic hypothermia,¹⁷³ the relative benefit of ischemia monitoring in this patient population needs to be weighed against the potential for misidentifying Osborn waves for ST-segment deviations caused by ischemia.

Recommendations

1. Arrhythmia monitoring, including QTc monitoring, is indicated in patients being treated with targeted temperature management (Class I; Level of Evidence C).
2. The decision for ischemia monitoring must be based on the presumed cause of the arrest (Class IIb; Level of Evidence C).

Vasospastic Angina

Coronary spasms occur as transient contractions in coronary arteries with highly variable amounts of sclerosis, including arteries in which sclerotic lesions are visible only with intravascular ultrasound.¹⁷⁴ Variant angina is a type of vasospastic angina and has been characterized by ST-segment elevation during angina attacks.^{174–177} Variant angina is more common among women and is not associated with typical coronary artery disease risk factors except smoking. Cocaine has been identified as a trigger.⁴³ Rare case studies have reported variant angina among adolescents.^{178,179} Recent attention has been directed to the contribution of an allergic (hypersensitivity) response.¹⁸⁰

Electrocardiographic manifestations and severity of angina vary for vasospastic angina (also called coronary spastic angina). Partial or complete occlusion may result in anginal pain with ischemia in the region perfused by the artery (ST-segment elevation), but if collateral flow is available, ST-segment depression may be visible instead.¹⁷⁴ In the CASPAR study (Coronary Artery Spasm in Patients With Acute Coronary Syndrome) of 488 consecutive patients with suspected ACS, 25% of patients had no culprit lesion, and epicardial coronary spasms were established for 50% of these patients.¹⁸¹ Investigators suggest that coronary spasm should routinely be considered as a differential diagnosis in ACS.

Patients with variant angina have electrocardiographic changes that are difficult to capture on a static 12-lead ECG because the ischemia is transient. They may have asymptomatic ischemia episodes, or they may be symptomatic with syncope that occurs as a result of severe arrhythmias, including VT, VF, and high-degree atrioventricular block.^{176,182} Untreated variant angina can result in MI, fatal arrhythmias, and sudden death,¹⁷⁶ so early treatment is imperative. Guidelines for the diagnosis and treatment of patients with vasospastic angina, including calcium channel blockers and nitrates, are available.¹⁷⁴ Continuous ambulatory electrocardiographic monitoring has been recommended for identifying frequency and duration of vasospasm leading to MI.¹⁸³ However, no studies were identified that used in-hospital ST-segment monitoring. Thus, for patients admitted with chest pain for whom vasospastic angina is a differential diagnosis, continuous ST-segment monitoring is reasonable and may be helpful in early detection of ischemia caused by spasms not documented on 12-lead ECG to facilitate diagnosis and to initiate treatment.

Recommendations

1. Arrhythmia monitoring for patients with vasospastic angina should be performed until symptoms resolve (Class I; Level of Evidence C).

2. Ischemia monitoring can be useful for patients with vasospastic angina until diagnosis is made and symptoms resolve (*Class IIa; Level of Evidence C*).

Apical Ballooning Syndrome (Stress Cardiomyopathy)

Apical ballooning (also called stress cardiomyopathy or takotsubo cardiomyopathy) is an acute cardiac syndrome manifested by transient wall-motion abnormalities (typically apical) that are most often exhibited in postmenopausal women with significant ST-segment elevation or T-wave abnormalities despite no significant obstructive coronary disease on angiography and only mild troponin elevation.¹⁸⁴ Current understanding of the pathogenesis is evolving on the basis of an exaggerated sympathetic stimulation often associated with a significant emotional crisis; coronary spasms have been implicated.¹⁸⁴ Case studies and case series reports¹⁸⁵ describe dynamic ST-segment and T-wave changes that increase and decrease rapidly. The clinical utility of continuous ST-segment ischemia monitoring has not been studied in this population. If continuous ST-segment ischemia monitoring is used, caution must be exercised because excessive alarms could occur if ST-segment alarm parameters are not adjusted on the basis of the unique characteristics of this dynamic condition.

Recommendations

1. Arrhythmia monitoring is recommended for patients with left ventricular apical ballooning until related symptoms resolve (*Class I; Level of Evidence C*).
2. Ischemia monitoring may be considered until symptoms resolved (*Class IIb; Level of Evidence C*).

Newly Diagnosed Left Main Coronary Artery Lesion

In a retrospective study¹⁸⁶ of >1700 patients undergoing coronary artery bypass graft (CABG) surgery, 97 had angiographically documented clinically significant ($\geq 50\%$) left main coronary artery stenosis. Among those patients with angiographically significant left main disease, 4 had acute MI or unstable angina before undergoing angiography and experienced cardiac events (3 had life-threatening ventricular arrhythmias, 1 had NSTEMI) while awaiting surgery. All events occurred within the first 3 days, but none occurred in the first 24 hours.¹⁸⁶ Although serious cardiac events occur infrequently in patients with left main stenosis awaiting CABG, ventricular arrhythmias are the most frequently occurring event, and patients with ACS may be at highest risk.

Recommendations

1. Arrhythmia monitoring is indicated in patients who have newly recognized critical left main

coronary artery stenosis or its equivalent while awaiting revascularization (*Class I; Level of Evidence C*).

2. Ischemia monitoring is reasonable for patients who have newly recognized critical left main coronary artery stenosis or its equivalent while awaiting revascularization (*Class IIa; Level of Evidence C*).

After Nonurgent PCI, With Complications or Suboptimal Results

In high-volume centers, complications after PCI are relatively infrequent. However, several complications of coronary intervention have been identified that warrant careful observation and a higher level of management after PCI: persistent chest pain with changes on the ECG, hypotension, severe arrhythmia, and angiographic evidence of significant dissection or remaining thrombus.¹⁸⁷ For suboptimal PCI results, including coronary artery complications such as vessel dissection or thrombus or procedural complications such as under-expansion or incomplete stent apposition, it is reasonable to continue monitoring for at least 24 hours given the risk of abrupt closure and other ischemic complications of dissection, including chest pain, changes on the ECG, MI, and death.¹⁸⁸ Although acute closure is most likely to occur within minutes of balloon inflation, sub-acute closure can occur later, with a median of 24 hours reported by Cheneau et al.¹⁸⁹ Continuous electrocardiographic monitoring can be useful for patients who experience complications during angiography or coronary intervention, including vessel dissection or no reflow, or who have suboptimal interventional outcomes.

Recommendations

1. After nonurgent PCI with complications or suboptimal results, it is reasonable to monitor for arrhythmia, beginning immediately and continuing for ≥ 24 hours or until complication is resolved (*Class IIa; Level of Evidence C*).
2. After nonurgent PCI with complications or suboptimal results, it is reasonable to monitor for ischemia, beginning immediately and continuing for ≥ 24 hours or until complication is resolved (*Class IIa; Level of Evidence C*).

After Nonurgent PCI, Without Complications

In 1994, Li et al¹⁸⁷ established that the majority of severe complications after angioplasty occur during the procedure itself or are immediately evident from the angiographic result (poor or no flow). Thus, continuous electrocardiographic monitoring would be unlikely to improve outcomes when there is no chest pain, change on the ECG, or symptomatic arrhythmia. In a review of nearly 20 000 PCIs in patients without cardiogenic shock, Addala et al¹⁵⁶ observed VF during PCI in

164 (0.84%). All were immediately defibrillated, and all were discharged without neurological sequelae. More recently, Spoon et al¹⁹⁰ noted that in-hospital mortality has decreased for patients undergoing PCI for stable angina. Improved treatment (stenting and optimal anticoagulation) and reduced vascular complications (closure devices and transradial approach) have led to a reduction in major complications and safe same-day discharge in low-risk patients after PCI. The risk of major complications is highest immediately after PCI; most major complications occur during the first 6 hours after PCI and decline thereafter.^{191–193} Despite the low likelihood of a serious event, vasovagal responses with symptomatic bradycardia can occur at the time of femoral sheath removal, so it is reasonable to consider continuous electrocardiographic monitoring immediately after intervention until femoral sheaths are removed. Ischemia monitoring may provide assurance that postprocedural chest discomfort is from stent manipulation, not angina.

Recommendation

- 1. In the absence of complications, continuous monitoring for ischemia and arrhythmia beyond femoral sheath pull in the immediate postprocedural area is unlikely to benefit after nonurgent PCI and therefore not recommended (Class III: No Benefit; Level of Evidence C).**

After Routine Diagnostic Coronary Angiography

The risk of complication after routine coronary angiography is <2% overall.^{194,195} Ventricular arrhythmias seldom occur during coronary angiography, and if they do occur, it is frequently when the catheter is manipulated or in response to high osmolar ionic contrast dye, used infrequently today.^{195,196} Major complications, including death, MI, or embolization during or after diagnostic cardiac angiography, are extremely rare (<1%), and procedural mortality has been reported to be 0.1%.^{195,197} It is increasingly common to perform diagnostic catheterizations in the ambulatory setting, and patients are often discharged several hours after uncomplicated coronary angiography.

Recommendation

- 1. In the absence of complications, continuous electrocardiographic monitoring (beyond that which is provided in immediate post-procedure area) after routine angiography is not indicated in low-risk patients (Class III: No Benefit; Level of Evidence C).**

Low-Risk and Noncardiac Chest Pain

Various strategies for assessment of patients presenting with chest pain share a common goal of early identifica-

tion of those at moderate to high risk of suffering ACS caused by obstructive coronary disease. Patients with a low likelihood of an active ischemic coronary syndrome are at low risk of adverse outcomes and thus may be safely discharged to outpatient follow-up without the need for ongoing inpatient evaluation and monitoring.¹¹² In a number of studies, investigators have questioned the value of continuous monitoring of patients presenting with chest pain and a low risk of ACS while in the ED, during transport, and after admission for workup and observation.^{106,107,198–201} The goal in each of these investigations was to ensure the safe allocation of monitoring resources so that these resources were available for patients with a clear, evidence-based need without unnecessary risk to the population of patients with chest pain in general.

Among patients determined to be at low risk for ACS on the basis of well-defined risk stratification schemes, significant monitor alarms are very uncommon. In the ALARMED (Adverse Events in Low Risk Patients With Chest Pain Receiving Continuous Electrocardiographic Monitoring in the Emergency Department)²⁰⁰ pilot study, 72 patients admitted to the ED with chest pain potentially of ischemic origin were monitored for 371 hours, and 1762 alarms were recorded. Of those alarms, 99.4% were false alarms. Eleven were significant alarms, 3 of which required a change in management. None of the 3 that required a change in management was in the low- or very-low-risk group.

Similarly, Hollander and colleagues¹⁰⁶ conducted a prospective cohort study of patients admitted from the ED to a non-ICU monitored bed with a diagnosis of chest pain. These patients had a Goldman Risk Score¹¹⁵ of <8%, a negative troponin I level (<0.3 ng/mL), and a normal initial creatine kinase-MB level (<5 ng/mL). Of the 1750 patients admitted to a non-ICU monitored bed for chest pain, 1029 met the criteria for low risk. The primary outcomes were cardiovascular death and life-threatening ventricular arrhythmia during telemetry. No patients had sustained VT/VF requiring treatment. There were 2 deaths, neither of which was cardiovascular or potentially preventable by electrocardiographic monitoring.

Both the ALARMED²⁰⁰ and Hollander et al¹⁰⁶ studies failed to show benefit in detecting or predicting any lethal arrhythmias, sudden death, MI, or hemodynamic instability related to heart rhythm among patients in whom ACS was ruled out and risks were low on the basis of established criteria. The low-risk designation makes continuous electrocardiographic monitoring unlikely to be helpful and potentially harmful in diverting scarce resources in an unproductive manner. In a study of patients determined to be primarily at low to intermediate risk of ACS by the HEART (history, ECG, age, risk factors, and troponin)^{118,202} score, Bovino et al²⁰³ found that continuous ST-segment monitoring in the ED detected only 3 episodes of ST-segment deviation, none of

which was associated with a diagnosis of ACS, and only 9% of patients were ruled in for ACS. Thus, their findings did not support the use of continuous ST-segment monitoring in the ED in this lower-risk population.

Recommendation

- 1. Continuous arrhythmia and ST-segment monitoring provides no benefit and is not indicated for patients with symptoms suggestive of ACS determined to be low risk or noncardiac in origin (Class III: No Benefit; Level of Evidence B).**

Major Cardiac Interventions

Open Heart Surgery

Arrhythmias are common after open heart surgery. AF is a common arrhythmia after any type of cardiac surgery, including CABG surgery or surgical valve replacement/repair.^{204–209} New-onset AF in CABG may occur at any point from the time of the procedure, usually 2 to 4 days postoperatively with peak occurrence at 2 days.²⁰⁶ AF occurs in 28% to 33% of patients undergoing CABG.²⁰⁷

Approximately 33% to 49% of patients undergoing surgical valve replacement/repair develop AF, and if the surgery is a combination of valve and CABG, the incidence rises to 60%.²¹⁰ AF is present at surgery in 30% to 40% of patients, and ≈8.5% convert to sinus rhythm after surgery. It is unclear how many remain in sinus rhythm. The onset of AF is associated with hemodynamic instability and increased risk of stroke, although up to 69% of episodes of AF are not associated with symptoms.²⁰⁵ Identification of new-onset AF when it occurs early in the postoperative period increases the likelihood that it will be treated before any hemodynamic or thromboembolic complications. Risk factors for developing AF after open heart surgery include older age, left atrial enlargement, mitral valve disease, heart failure, hypertension, and history of AF.²⁰⁸

Ventricular arrhythmias are common in the immediate postoperative period and are related to hypothermia, ischemia, and electrolyte abnormalities.²¹¹ Risk decreases over time but is never eliminated. These rhythms have the potential to affect hemodynamic stability and to cause sudden death.

Permanent pacemaker implantation is required in 1.5% of cases for postoperative atrioventricular block after cardiac surgery.²¹² In a study by Huynh and colleagues,²¹³ among the 207 patients who underwent surgical valve replacement/repair, 7.2% required permanent pacing postoperatively. Predictors included preoperative first-degree atrioventricular block with and without left anterior fascicular block and/or intraventricular conduction delay, postoperative cardiac arrest, and combined mitral and aortic valve replacements.

The greatest risk of ventricular arrhythmia and sudden death occurs in the immediate postoperative period in the ICU where monitoring is the standard of care, and although risk does not completely disappear, it decreases rapidly once the patient is ready for discharge from the ICU. Similarly, the risk of heart block requiring temporary or permanent pacing is higher in the immediate postoperative period. Bradycardia with or without third-degree atrioventricular block has significant hemodynamic consequences; therefore, the patient benefits from early recognition of the rhythm. AF may occur at any time in the postoperative period and has both potential hemodynamic consequences (diastolic heart failure, rapid ventricular response) and embolic consequences (transient ischemic attack, stroke).

In addition to arrhythmia monitoring, ischemia monitoring has shown utility in identifying myocardial ischemia in the intraoperative period^{214,215} and was given a COR IIa, LOE B recommendation in published guidelines.⁶¹ Ischemia monitoring can also be helpful in the early postoperative period,^{216–219} with potential to guide targeted therapeutic efforts, including surgical revision or early angiography and percutaneous revascularization,^{220,221} and was given a COR IIb, LOE B recommendation in published guidelines.⁶¹

Recommendations

- 1. For patients whose open heart surgery was uncomplicated, arrhythmia monitoring is recommended for a minimum of 48 to 72 hours postoperatively (Class I; Level of Evidence B).**
- 2. For patients at high risk for AF, postoperative arrhythmia monitoring is recommended for the duration of their hospitalization in an acute care unit (Class I; Level of Evidence B).**
- 3. Ischemia monitoring is reasonable intraoperatively (Class IIa; Level of Evidence B).**
- 4. Ischemia monitoring may be considered in the immediate postoperative setting for the detection of ongoing or new ischemia in intubated and sedated patients and those in the early recuperative phase who may have difficulty recognizing and reporting new or ongoing ischemia (Class IIb; Level of Evidence B).**

Pediatric Considerations

Arrhythmias are common in children after surgical repair of congenital heart disease. Virtually all of these patients will have an electrocardiographic monitor in place as part of the admission to an ICU. Because of developmental characteristics of the patient and alterations in the conduction system secondary to the underlying structural disease, the mechanism can be challenging to diagnose.^{222,223} The arrhythmias are com-

monly atrial or junctional in origin,^{224,225} with the focus difficult to distinguish.

Tachycardia that is atrial in origin is commonly attributable to an atrial macroreentrant circuit or is a focal tachycardia caused by a gain of automaticity. The P wave can be difficult to see because at higher rates it may be buried in the preceding T wave. In addition, in patients who have a history of a total cavopulmonary anastomosis (all of the systemic venous return bypasses the heart and drains directly into the pulmonary arteries) and have undergone an atrial reduction, there may not be much atrial tissue to generate voltage that is easily seen on surface leads.^{226,227} Junctional ectopic tachycardia is thought to originate from a gain of automaticity of the bundle of His. In this arrhythmia, the P wave is not seen via surface leads because it is masked by the QRS complex. Recording of the atrial depolarization by either an epicardial or a transesophageal lead may be necessary to distinguish the mechanism. VT is also seen after congenital heart surgery.^{222,228} These arrhythmias may be the result of surgical incisions, coronary manipulation, electrolyte imbalance, or infusion of vasoactive medication. Whether the mechanism of a tachycardia is ventricular or supraventricular, it can have a substantial negative impact on the patient's hemodynamic status. It is critical for these rhythm disturbances to be detected and treated appropriately in the postoperative period. The duration of monitoring is determined by the clinical stability of the patient.

After pediatric cardiac surgery, high-grade atrioventricular nodal block can be seen and may be transient or permanent. If the expectation is that atrioventricular nodal function will return, some recommend allowing 7 days for assessment of atrioventricular nodal function before placement of a permanent pacing system.²²⁹ During the period before return of atrioventricular nodal function or placement of a permanent pacemaker, the patient is typically supported with temporary epicardial pacing wires connected to an external pulse generator. Pacing capture thresholds can change rapidly, necessitating continuous electrocardiographic monitoring. Although continuous arrhythmia monitoring until discharge from an acute care unit is common practice in pediatrics, current data are lacking to provide specific recommendations for practice.

Mechanical Circulatory Support

Mechanical circulatory support devices include the total artificial heart, extracorporeal life support, ventricular assist device (VAD), and intra-aortic balloon pump.

Clinically Significant Cardiovascular or Hemodynamic Deterioration. These patients are hemodynamically unstable; therefore, it is standard of care that they will be in an ICU, where they will receive electrocardiographic monitoring to allow the recognition and treatment of arrhythmias. Further monitoring for ischemia

is necessary only if the patient meets criteria (eg, signs and symptoms of angina). Further monitoring for QTc is indicated only if the patient meets the respective QTc monitoring criteria (eg, initiating dofetilide).

Recommendation

- 1. For hemodynamically unstable patients with immediate need for mechanical circulatory support, continuous arrhythmia is indicated (Class I; Level of Evidence C).**

Immediately After Implantation. VADs are increasingly being used long term for patients with advanced heart failure. It is standard of care that in the immediate postoperative period, these patients will be an ICU with continuous electrocardiographic monitoring. After the immediate postoperative period, patients with VADs can be successfully cared for outside of an ICU. Patients have been considered stable for telemetry units if they are extubated, are weaned from vasopressors, have stable vital signs and heart rhythm, and can be cared for by staff competent in care of patients with VADs.^{230,231}

After the immediate period, ≈20% of patients with left VADs have atrial arrhythmias, which are most common within the first 60 days of implantation. Patients with left VADs who had atrial arrhythmias had a worse unadjusted quality of life and a decreased rate of improvement in a 6-minute walk test over 6 to 24 months after implantation.²³² Although ventricular arrhythmias are common in patients with continuous-flow left VADs, occurring in about one third of patients, these arrhythmias may not be life-threatening.^{233,234}

Recommendation

- 1. Arrhythmia monitoring is indicated for patients in the postoperative period after VAD implantation (Class I; Level of Evidence C).**

Admitted With Noncardiac Problems. Hemodynamically stable patients with VADs admitted for noncardiac problems (eg, gastrointestinal bleed) will usually be on telemetry units. Continuous electrocardiographic monitoring for hospitalized patients with VADs is considered standard of care because many patients have a pulse that is difficult or impossible to palpate. In addition, arrhythmias may provide insight into the hemodynamics of the VAD, indicating a need to increase or decrease pump speed to optimize hemodynamic function. It is important that patients with a VAD are cared for by personnel trained in VAD care,²³⁵ and this level of expertise is most often found in telemetry units in the hospital. However, if staff members have competency in the basic management of VADs elsewhere, telemetry may not be required for stable patients. Thus, it is

reasonable to admit patients with acute care needs to telemetry units; however, patients with nonacute, noncardiac problems may be admitted to nonmonitored units (eg, a mental health unit) if appropriate VAD management can be provided.

Recommendation

- 1. Arrhythmia monitoring can be beneficial for patients with VADs admitted with noncardiac problems but may not be needed in all circumstances if appropriate VAD care can be provided (Class IIa; Level of Evidence C).**

Admitted to a Rehabilitation Facility. Rehabilitation facilities where staff is educated on the basic care of patients with VADs may be safe environments for these patients, even without providing continuous electrocardiographic monitoring.

Recommendation

- 1. Arrhythmia monitoring is not recommended for patients with VADs admitted to a rehabilitation facility where basic VAD care is available (Class III: No Benefit; Level of Evidence C).**

Pediatric Considerations. At this point in the development of the clinical practice of caring for left VADs in children, most are housed in ICUs or step-down units. As a result of their location, they are typically monitored.

Transcatheter Structural Interventions

Transcatheter structural interventions represent a heterogeneous group of interventions with a heterogeneous group of devices. Some, such as closure of septal defects and transcatheter aortic valve replacement (TAVR), have specifically described procedure-related risk of arrhythmias. For others, such as repair of perivalvular leak or pseudoaneurysm repair, arrhythmia risk will depend more on patient comorbidities than on the specific surgical procedure. All patients undergoing transcatheter interventions require arrhythmia monitoring after the procedure.

After TAVR. TAVR has emerged as an alternative to open heart surgery for patients with severe aortic stenosis who are at high risk for surgical replacement,²³⁶ and indications may expand to those at moderate risk as experience with this procedure grows. Atrioventricular block requiring pacemaker implantation is reported in 1% to 8% of patients with the Edwards SAPIEN valve and 19% to 42% with the Medtronic CoreValve using currently available devices.²³⁶ The need for pacemaker implantation did not influence subsequent survival after TAVR in some studies,²³⁷ although others have shown increased mortality in those receiving a new pacemaker.²³⁸ Clinical predictors of complete heart

block²³⁹ include characteristics of patient status (valve calcification and preexisting right BBB)²⁴⁰ and comorbidities, as well as characteristics of the procedure (depth of implantation, device profile)²⁴¹ and balloon sizing.²⁴² However, sensitivity and specificity are not high except possibly for right BBB. AF is also common after TAVR.^{243,244} Current consensus documents recommend continuous monitoring early after the procedure (at least 3 days).²³⁶ In a recent analysis²⁴⁵ of 32 studies of TAVR, including >5000 patients without pacemaker before the procedure, the need for a pacemaker was reported in 25% of Medtronic CoreValve and in 6% of the Edwards SAPIEN valve recipients. In the 5 trials reporting timing of the development of atrioventricular block, 63% occurred during or within 24 hours of TAVR, and another 32% occurred within 1 week, leading these authors to recommend 1 week of in-hospital monitoring after TAVR.²⁴⁵ Among patients who do not require a permanent pacemaker by 48 hours after TAVR, 5% of patients with persistent left BBB (versus 2% without left BBB) are likely to require a permanent pacemaker.²⁴⁶ Longer monitoring could be considered for patients with evidence of periprocedural conduction abnormalities.

Electrocardiographic monitoring may also reveal AF after TAVR. Amat-Santos and colleagues²⁰⁹ studied the incidence and implications of AF in the TAVR population. AF had not been considered a significant risk for TAVR patients in the past.²⁴⁷ However, findings from the study reported by Amat-Santos et al²⁰⁹ suggest that the incidence of new-onset AF may be high in the first 30 days after TAVR. They studied 138 consecutive patients with no history of AF who had TAVR and were monitored until hospital discharge. New-onset AF occurred in 44 patients (31.9%) at a median time of 48 hours after TAVR and was associated with a higher incidence of stroke at 30 days and 1 year.

The following recommendations are given. Duration of monitoring varies with procedure, device, and patient factors.

Recommendations

- 1. After TAVR, at least 3 days of postprocedural arrhythmia monitoring is recommended (Class I; Level of Evidence C).**
- 2. Longer monitoring periods may be beneficial for patients undergoing TAVR, particularly those with periprocedural conduction abnormalities (Class IIa; Level of Evidence C).**

Other Transcatheter Interventions: Ventricular Septal Defect Closure. Percutaneous device closure of perimembranous ventricular septal defect is increasingly used as a successful alternative to surgical closure.²⁴⁸ Arrhythmic complications, including BBB and

atrioventricular block, occur in 11% during the procedure²⁴⁸ and in 15% after the procedure,²⁴⁹ with a 1% to 5.7% incidence of high-grade atrioventricular block. Time to the development of atrioventricular block ranged from intraprocedural to up to 7 days after the procedure, with most occurring by 3 days. Although atrioventricular block resolution often occurs with steroids, certain characteristics of the ventricular septal defect and placement of the occluder²⁴⁹ have been identified as risk factors for atrioventricular block. Thus, all patients should be monitored after percutaneous ventricular septal defect closure.

Other Transcatheter Interventions: Atrial Septal Defect Closure. Atrial septal defect transcatheter occlusion techniques have emerged as an alternative to surgery. Both atrial arrhythmias and heart blocks have been reported.²⁵⁰ In a meta-analysis of 172 series comprising >13 000 patients, the incidence of atrioventricular block was 0.4%.²⁵¹ The overall risk of major periprocedural complications was 0% to 9.4% in the studies, with a pooled risk of 1.6%. In a recent series of 706 patients undergoing atrial septal defect closure with an Amplatzer device, the risk of atrioventricular block was 0.85%. Age, size of the atrial septal defect, and characteristics of the occluder were predictive of atrioventricular block.²⁵²

Recommendation

1. All patients undergoing transcatheter interventions require arrhythmia monitoring after the procedure (**Class I; Level of Evidence C**). Duration of monitoring varies with procedure, device, and patient factors.

Arrhythmias

As a general rule, patients with arrhythmias that are life-threatening or potentially life-threatening and those who require ongoing management should be monitored. Monitoring is not required for patients with asymptomatic, non-life-threatening arrhythmias that do not require ongoing management.

Ventricular Tachycardias

Patients resuscitated from cardiac arrest or unstable VT have a high risk of recurrent arrest.²²⁹ Most of these patients will require an ICD. While hospitalized for evaluation and before implantation of an ICD or other devices (eg, wearable cardiac defibrillator, VAD), these patients should have arrhythmia monitoring.

Recommendations

1. These patients should receive arrhythmia monitoring until ICD implantation. Patients after cardiac arrest believed to be attributable to a transient and reversible cause should be

monitored until the underlying problem is resolved (**Class I; Level of Evidence C**).

2. After implantation, if further therapy is ongoing (ie, medications, ablation), monitoring should be continued until adequate suppression of arrhythmia as determined by the treating team is achieved (**Class I; Level of Evidence C**).

Ventricular tachyarrhythmias resulting in ICD shock are discussed in Section 2, Preexisting Rhythm Devices.

Nonsustained VT

Premature ventricular contractions (PVCs) and nonsustained VT may be associated with occult structural heart disease and with long-term reduced survival in unselected populations.²⁵³ Even in otherwise normal hearts, PVCs and nonsustained VT may over time lead to tachycardia-induced cardiomyopathy.²⁵⁴ Further evaluation and treatment may be warranted in some situations²⁵⁴ such as determining the origin of the VT, which may help guide further therapy such as ablation.

Recommendation

1. PVCs and nonsustained VT are not immediately life-threatening, and in the absence of other indications for monitoring in hospitalized patients, continued arrhythmia monitoring may be considered but is not required (**Class IIb; Level of Evidence C**).

Atrial Tachyarrhythmias

Atrial arrhythmias include new-onset or recurrent paroxysmal or intermittent-persistent AF or other atrial tachyarrhythmias.²⁵⁵ For unidentified supraventricular arrhythmias, arrhythmia monitoring will aid in diagnosis. For AF, monitoring will aid in determining the burden of AF and the adequacy of rate control. Although rapid AF is rarely life-threatening, it can cause symptoms and, in some cases, hemodynamic deterioration.

Recommendations

1. Patients admitted for new-onset or recurrent atrial tachyarrhythmias, including AF, should receive arrhythmia monitoring while planned evaluation is underway and the treatment strategy is determined (**Class I; Level of Evidence C**).
2. Patients with hemodynamically unstable or symptomatic atrial arrhythmias should receive arrhythmia monitoring until hemodynamically stable (**Class I; Level of Evidence C**).
3. If rate control in the hospital is deemed necessary, arrhythmia monitoring is beneficial in management and may accelerate appropriate treatment (**Class I; Level of Evidence C**).

Although strategies of attempting to maintain sinus rhythm through the use of antiarrhythmic drugs have not been shown to improve mortality, antiarrhythmic drug therapy may be used to decrease the frequency of symptoms and to improve quality of life in selected patients.²⁵⁶ In a 2006 meta-analysis of 44 trials including >11 000 patients,²⁵⁷ Class IC (flecainide and propafenone) and Class III drugs (amiodarone, sotalol, dofetilide; dronedarone is not approved) reduced recurrences of atrial tachyarrhythmias after cardioversion of AF.

Except for amiodarone and propafenone, all antiarrhythmics have increased proarrhythmias (number needed to harm, 17–119), with risk most commonly occurring during initiation.²⁵⁶ Previous guidelines have not addressed inpatient versus outpatient electrocardiographic monitoring for the initiation of antiarrhythmic agents.²⁵⁶ Dofetilide carries a risk of TdP, and inpatient electrocardiographic monitoring is required by the US Food and Drug Administration for 3 days during initiation because of the risk of QT prolongation and ventricular arrhythmias.²⁵⁶ Sotalol also prolongs the QT interval.²⁵⁷ Some experts have suggested that outpatient initiation is safe if the patient is in sinus rhythm and the QT interval and electrolytes are normal,²⁵⁸ although others suggest inpatient monitoring.²⁵⁶ Initiation and dose escalation during hospitalization with electrocardiographic monitoring should be considered; the package insert for sotalol has a corresponding black box warning.²⁵⁶ One small study suggested that outpatient, unmonitored initiation of flecainide and propafenone is safe,²⁵⁹ but data are insufficient for a definitive recommendation. Data supporting the safety of the initiation of antiarrhythmic drug therapy without monitoring are best established for amiodarone and dronedarone,²⁵⁶ although both can lead to bradycardia,²⁵⁷ so monitoring may be considered for initiation, particularly in patients currently in AF whose sinus node function may put them at risk for postconversion pauses.

Recommendations

1. Arrhythmia monitoring is recommended for initiation of dofetilide (*Class I; Level of Evidence B*).
2. Among patients for whom the provider has selected inpatient initiation, arrhythmia monitoring is recommended for the initiation of sotalol, flecainide, and propafenone (*Class I; Level of Evidence B*).
3. Among patients for whom the provider has selected inpatient initiation, arrhythmia monitoring may be considered for the initiation of amiodarone and dronedarone (*Class IIb; Level of Evidence B*).

Patients receiving QTc monitoring, as described earlier in these practice standards, should be monitored for arrhythmias.

Chronic AF

Patients with long-standing or permanent AF who are admitted with other medical indications do not routinely need arrhythmia monitoring. For patients with chronic AF who are admitted for a medical condition that affects ventricular rate, arrhythmia monitoring is reasonable.

Recommendations

1. Patients with known permanent AF do not need ongoing monitoring once rate control is determined to be adequate (*Class III: No Benefit; Level of Evidence C*).
2. Arrhythmia monitoring is reasonable in patients with chronic AF if a medical condition may affect their ventricular rate (eg, results in changes in autonomic state or inability to take medications) or if their heart rate may affect their clinical stability (*Class IIa; Level of Evidence C*).

Sinus Bradycardias

The clinical manifestations of sinus node dysfunction, implying slow impulse generation from the sinus node or pauses in sinus node firing, are many, ranging from syncope to chronotropic incompetence to the incidental finding of bradycardia in the asymptomatic patient. Untreated, sinus node dysfunction does not influence survival,²⁶⁰ and asymptomatic sinus bradycardia is not an indication for pacing.²²⁹ Thus, asymptomatic sinus bradycardia does not require in-hospital monitoring. Patients with symptomatic sinus bradycardia awaiting pacemaker implantation should be monitored. Many drugs can slow the heart rate, and in patients with significant sinus bradycardia in whom negative chronotropic medications are being initiated, monitoring may be considered on an individual basis.

Recommendations

1. Arrhythmia monitoring should be used for patients with symptomatic bradycardia such as syncope (*Class I; Level of Evidence C*).
2. Arrhythmia monitoring may be beneficial for patients undergoing initiation of negatively chronotropic medications, in whom worsening of a baseline sinus bradycardia may be a concern (*Class IIa; Level of Evidence C*).
3. Finally, arrhythmia monitoring is not recommended for asymptomatic, hemodynamically stable patients with bradycardia (*Class III; Level of Evidence B*).

Atrioventricular Block

Atrioventricular block is classified by the pattern of conduction from the atria to the ventricles. In first-degree atrioventricular block, the PR interval is prolonged but 1:1 conduction remains; in second-degree block, some P waves are conducted and some are not; in third-degree block, the atrioventricular block is complete. Advanced atrioventricular block refers to blocking of >1 consecutive P wave. Second-degree atrioventricular block is further classified on the basis of the pattern of the PR interval, which generally correlates with the anatomic site of block. Mobitz type I, or Wenckebach, is characterized by a progressive lengthening of the PR interval before the block and generally reflects disease within the atrioventricular node, whereas Mobitz type II is characterized by unchanging PR intervals before the block and is generally associated with infranodal (distal) conduction system disease. Complete heart block is not defined on the basis of type but can often, although not always, be diagnosed as intranodal (proximal) versus infranodal (distal) on the basis of the characteristics of the escape focus, as well as the preceding rhythms.

Early studies showed that atrioventricular block resulting from distal disease could progress rapidly and unpredictably and has been associated with sudden death.^{229,261} Thus, these patients require arrhythmia monitoring until a pacemaker is implanted. In contrast, patients with type I second-degree atrioventricular block (Wenckebach) generally have a benign prognosis.²⁶² Wenckebach is common and benign in athletes and during sleep.²⁶³ Monitoring may be considered in patients with Wenckebach but is generally not required. Complete heart block caused by atrioventricular nodal disease will have a stable, junctional escape and is not immediately life-threatening. Monitoring of patients with atrioventricular nodal third-degree block should be considered on an individual basis.

Recommendations

1. Patients with symptomatic second- or third-degree atrioventricular block of any anatomic origin should have arrhythmia monitoring (*Class I; Level of Evidence C*).
2. Patients with asymptomatic second- or third-degree atrioventricular block caused by distal conduction system disease should have arrhythmia monitoring (*Class I; Level of Evidence C*).
3. Patients with third-degree atrioventricular block should have arrhythmia monitoring (*Class I; Level of Evidence C*).
4. Asymptomatic patients with Wenckebach and those with transient atrioventricular block of any degree determined to be of

vagal origin do not benefit from arrhythmia monitoring (*Class III; Level of Evidence C*).

Pediatric Considerations. Third-degree atrioventricular nodal block can be seen in infants and children in the absence of cardiac surgery. The decision for permanent pacing is based on escape loci and heart rate, as well as symptoms associated with the bradycardia.²²⁹ For newborns, the ability to feed without signs of hemodynamic compromise is often used in determining the need for pacing. During that assessment period, they are often in a neonatal ICU where electrocardiographic monitoring is standard of care.

Congenital or Genetic Arrhythmic Syndromes

Among the numerous conditions that can predispose patients to life-threatening arrhythmias are many inherited disorders such as LQTS and Brugada syndrome and those characterized by abnormal conduction such as Wolff-Parkinson-White syndrome.²⁶⁴ Determining which of these patients are at higher risk of sudden death and require therapeutic intervention can be challenging.

Hemodynamically Unstable. Patients with these syndromes who present with signs of unstable ventricular arrhythmias (eg, recurrent syncope or worsening ventricular ectopy) or clear worsening of the underlying arrhythmic susceptibility (eg, during uncontrolled fevers with Brugada syndrome or with metabolically induced prolongation of the QT interval in LQTS) should have arrhythmia monitoring until appropriate therapy is delivered. Worrisome findings on the ECG such as a significant augmentation of QT prolongation or the development of a type I Brugada pattern on the ECG during a febrile illness should also be considered in the decision to provide electrocardiographic monitoring.

Recommendation

1. Hemodynamically unstable patients with congenital or genetic arrhythmic syndromes should have arrhythmia monitoring until appropriate therapy is delivered (*Class I; Level of Evidence C*).

Wolff-Parkinson-White Syndrome With Rapid Conduction via an Accessory Pathway.

Patients with Wolff-Parkinson-White syndrome who demonstrate rapid conduction via an accessory pathway during an atrial arrhythmia, typically with shortest pre-excited RR intervals <250 milliseconds, are at greater risk of developing VF.²⁶⁵

Recommendation

1. Arrhythmia monitoring is indicated in patients with Wolff-Parkinson-White syndrome with

rapid conduction via an accessory pathway until therapy such as antiarrhythmic medication or ablation is delivered (Class I; Level of Evidence C).

Congenital Long QT. Patients with inherited long QT who present with unstable ventricular arrhythmias or have medically or metabolically induced prolongation of the QTc interval should have electrocardiographic monitoring.

Recommendation

- 1. Arrhythmia and QTc monitoring is indicated in unstable patients with congenital long QT until stabilization of ventricular arrhythmias, reversal of exacerbating medical or metabolic condition, and return of the QTc interval to baseline (Class I; Level of Evidence C).**

Syncope of Suspected Cardiac Origin

Syncope, defined as a transient, self-limited loss of consciousness, is among the most common reasons for visiting an ED.²⁶⁶ There are 2 reasons to evaluate syncope. The first is to determine the pathogenic mechanism to prevent future episodes, which are most often not life-threatening but may have an impact on quality of life. The second is to identify the less common patient whose syncopal episode represents a risk for death. Several predictive algorithms such as the San Francisco²⁶⁶ and Rose²⁶⁷ rules have been developed to determine prognosis on the basis of clinical factors at presentation and thus guide necessity for inpatient admission; other studies are ongoing.²⁶⁸ These and other algorithms have identified age, structural heart disease, abnormal ECG, absence of prodromal symptoms, and other factors as predictive of higher risk in patients presenting with syncope.²⁶⁶ Recent studies suggest electrocardiographic findings predictive of serious cardiac outcomes, which include Mobitz 2 or complete atrioventricular block, severe conduction system disease, new ischemic changes, nonsinus rhythm, left axis deviation, or other abnormalities noted from electrocardiographic monitoring in the ED.²⁶⁹

The ACC/AHA/Heart Rhythm Society 2017 guideline for the evaluation and management of patients with syncope²⁷⁰ provides recommendations for hospital admission for syncope. This guideline also states that patients with a suspected cardiac cause of their syncope are vulnerable to arrhythmias and thus should undergo electrocardiographic monitoring.

For those patients admitted for treatment of syncope with an identified cause, such pathogenesis should guide the use of arrhythmia monitoring. Occurrence of symptoms correlating with a documented arrhythmia on monitoring is considered a gold standard for the diagnosis of arrhythmia, but asymptomatic findings on monitoring, including asystole >3 seconds, Mobitz 2

atrioventricular block, ventricular arrhythmia, or rapid supraventricular arrhythmias, when associated with syncope at other times, have also been considered diagnostic.²⁷¹ In contrast to a 5% diagnostic yield of inpatient telemetry monitoring in unselected populations admitted for syncope of undetermined origin,²⁷² predetermined algorithms based on predictive characteristics documented a 16% to 18% diagnostic yield with inpatient monitoring.^{273,274} In a study of telemetry use, syncope was one of the major diagnoses for which telemetry monitoring influenced management decisions.⁵⁸

Recommendation

- 1. Arrhythmia monitoring for at least 24 hours is recommended for those patients who, on the basis of presentation characteristics, are admitted for syncope with a suspected cardiac origin (Class I; Level of Evidence B).**

Pediatric Considerations. Syncope is a common presenting complaint for pediatric patients in the ED and often associated with high patient and parental anxiety. Although syncope in pediatric patients is often attributable to a benign cause,²⁷⁵ in some patients, syncope may herald a serious medical condition such as obstruction to blood flow, myocardial dysfunction, or syncope that originated from a primary arrhythmic source. Algorithms using a detailed medical history, family history, physical examination, and 12-lead ECG have been developed to help distinguish between benign syncope and malignant cardiac conditions requiring further evaluation.^{270,276,277} If a patient is believed to have a cardiac condition that requires admission, consideration should be given to arrhythmia monitoring.

Postelectrophysiology Procedures/Ablations

The practice of ablation is rapidly evolving with a paucity of published research related to postprocedural arrhythmia monitoring. Although the incidence of major complications has been reported, the timing of occurrence is often not described in detail.²⁷⁸ Clearly, a major complication such as hemodynamic instability from pericardial effusion or a thromboembolic event such as stroke is an indication for arrhythmia monitoring. Therefore, the following recommendations are based primarily on expert opinion. Investigators are encouraged to assess and report the timing of both minor and major complications that require arrhythmia monitoring so that recommendations can have a stronger evidence base.

Uncomplicated Supraventricular Tachycardia Ablation

The technologies and techniques for catheter-based ablations of cardiac arrhythmias are advancing at a great pace. Modern tools allow the practitioner to deliver targeted lesion sets and to perform more controlled transseptal punctures. Rates of postoperative complications after ablation for simple supraventricular tachycardias

(SVTs; atrioventricular nodal reentry tachycardia, atrioventricular reentry tachycardia, atrial flutter, and atrial tachycardia) have improved over the past several years. The rate of atrioventricular block, for example, after slow pathway ablation for atrioventricular nodal reentry tachycardia is now <1%.²⁷⁹ The duration of observation of patients with uncomplicated SVT ablation without evidence of transient atrioventricular block is not well defined in the medical literature; some clinicians discharge patients after ≈4 hours of arrhythmia monitoring.

Recommendation

1. Patients with uncomplicated SVT ablation (eg, no transient atrioventricular block) may be discharged from arrhythmia monitoring after a short observation period in a post-procedure area. Additional arrhythmia monitoring after the postprocedure area may be considered (Class IIb; Level of Evidence C).

Complex Ablations or Serious Comorbidities

Investigators²⁷⁸ prospectively evaluated 1676 consecutive ablations (non-AF SVT, AF, and VT) for major adverse postprocedural events such as life-threatening cardiac perforation, stroke, or death, with the following findings by ablation type: for SVT, 0.8%; for idiopathic VT, 3.4%; for VT ablation in the setting of structural heart disease, 6%; and for AF, 5.2%. Others have reported major complication rates after AF ablation between 4.5%²⁸⁰ and 5.8%.²⁸¹ Patients who undergo complex ablation such as pulmonary vein isolation for AF or ablation for VT are at higher risk of major complications in the postoperative period. These patients often have additional comorbidities and usually require general anesthesia during ablation. Investigators²⁷⁸ identified no thromboembolic events after SVT ablation; the vast majority occurred after AF ablation (1%). Of all thromboembolic events, only 27.3% occurred before the patient left the procedure room.²⁷⁸ For all ablation types, most major complications occurred on the procedure day (54.7%), a third (31.1%) during the procedure. However, postprocedural complications occurred at a mean of 4.4±5.6 days and included 2 pericardial effusions on days 5 and 6.²⁷⁸

The following recommendations are provided. However, the duration of monitoring varies with procedure, vascular access, and patient factors. Expert clinical judgment is needed.

Recommendation

1. Patients with more serious comorbidities (eg, advanced heart failure) or who undergo complex ablations (eg, pulmonary vein isolation for AF) should receive arrhythmia monitoring

for 12 to 24 hours after the procedure (Class I; Level of Evidence C).

Atrioventricular Nodal Ablation

Patients who have experienced prolonged rapid heart rates from incessant tachycardia may be at greater risk for TdP, as demonstrated in case reports,^{282,283} as are patients with chronic AF who undergo atrioventricular nodal ablation and concomitant pacemaker implantation.²⁸⁴

Recommendation

1. Patients who undergo atrioventricular nodal ablation who have experienced prolonged rapid heart rates from incessant tachycardia and patients with chronic AF who undergo atrioventricular nodal ablation with concomitant pacemaker implantation should receive arrhythmia monitoring for 12 to 24 hours after ablation (Class I; Level of Evidence C).

After Pacemaker or ICD Implantation Procedures

Temporary pacing is indicated in situations of acute life-threatening bradyarrhythmias, as well as situations in which permanent pacing is indicated for bradyarrhythmias but temporarily contraindicated by infection or other acute comorbidities. Temporary pacing may be achieved via transcutaneous pacing pads, transvenous pacing wires, or semipermanent transvenous pacing wires.

Transcutaneous Pacing Pads

Transcutaneous pacing can be deployed rapidly, but it is moderately or very uncomfortable for most patients,²⁸⁵ and capture is variable.^{285,286} Thus, transcutaneous pacing is used for urgent situations pending insertion of a transvenous lead or for situations with lower likelihood of pacing necessity. Transcutaneous pacing is subject to the same concerns as 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 electrocardiographic monitoring leads should be tested 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 nonelectrocardiographic method is recommended (eg, arterial pressure, pulse oximetry monitoring, or both).

Recommendation

1. Arrhythmia monitoring is recommended until transcutaneous pacing is no longer necessary and the device is removed or replaced with a permanent device (Class I; Level of Evidence C).

Standard Temporary Transvenous Pacing Wires

Standard temporary transvenous pacing wires are stiff wires that have no fixation mechanisms, thus increasing the likelihood of dislodgment. In addition, loss of 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 because of extraneous electric potentials such as muscle artifact or nearby faulty electric equipment. In a prospective observational study conducted in patients with temporary transvenous pacing wires, dislodgement rates were 16%, with 50% of dislodgements occurring within the first 24 hours and the other half occurring later during the hospital course.²⁸⁷

Recommendation

- 1. All patients with standard temporary transvenous pacing wires should receive arrhythmia monitoring until either pacing is no longer necessary and the device is removed or it is replaced with a permanent device (Class I; Level of Evidence C).**

Semipermanent Transvenous Temporary Pacing

The use of transvenous active fixation leads is increasing, such as those designed for permanent pacing that are inserted transvenously, externalized, and connected to an epicutaneous pulse generator.^{288–291} These temporary systems have been used in place of standard temporary wires in several settings: device explantation because of infection to allow a period of antibiotic treatment, when the need for pacing may be temporary such as Lyme disease, or when permanent pacing is indicated but temporarily precluded by comorbidities such as systemic infection.²⁹² Several single-center studies of 17 to 60 patients have shown no loss of function or dislodgements over mean or median uses of 2 to 19 days. Although 1 of the earlier centers²⁸⁸ described maintaining patients in telemetry units during this period, 2 centers did not maintain patients on telemetry wards,^{290,291} and 1 center²⁸⁹ allowed patients to go home or to a nursing facility with these systems in place. Telemetry may be considered in these patients.

Recommendations

- 1. It is reasonable for all patients with semipermanent transvenous pacing to receive arrhythmia monitoring for 24 hours (Class IIa; Level of Evidence C).**
- 2. After the first 24 hours in low-risk patients, arrhythmia monitoring may be considered if the patient is not discharged to home or a skilled nursing facility (Class IIb; Level of Evidence C).**

Permanent Pacemaker or ICD

The number of pacemaker, ICD, and cardiac resynchronization device implantations continues to increase dramatically as the population ages and indications broaden. The risks of major complications, including cardiac perforation, hemothorax, pneumothorax, stroke, MI, and death, increase with age and complexity of device implantation.^{77,293}

Although many of the major adverse complications occur during the procedure, acute lead failure often manifests in the immediate postprocedural period.²⁹⁴ Acute failure to capture or sense appropriately can be caused by lead fractures, loose set screws, cardiac perforation, sudden increase in pacing threshold, and most commonly, lead dislodgement. Rates of lead dislodgement have been reported in 1% to 2% of cases after pacemaker or ICD insertion and up to 5.7% after cardiac resynchronization device insertion.^{294–296}

The main purpose of electrocardiographic monitoring after device implantation is to detect acute lead failure as evidenced by oversensing, undersensing, or failure to capture. Early confirmation of lead failure with device interrogation or chest x-ray can result in rapid procedural correction. Although less common, electrocardiographic monitoring may also detect arrhythmias related to the major adverse complications such as sinus tachycardia in the setting of cardiac tamponade. Monitor manufacturers have specific electrode configurations to achieve the most reliable recognition of paced rhythms. In addition, the clinician may need to enable pacemaker recognition by the monitor to avoid a potential monitor alarm for VT resulting from the width of the QRS with paced rhythm. Improvement in visibility of pacemaker spikes is still needed by some monitor manufacturers. Because technologies for pacemaker recognition vary, no recommendation for their use can be made at this time.

Recommendations

- 1. Patients who do not have a consistent, intrinsic, hemodynamically stable heart rhythm are considered pacemaker dependent, and arrhythmia monitoring is recommended for 12 to 24 hours after device implantation (Class I; Level of Evidence C).**
- 2. For patients who are not pacemaker dependent, arrhythmia monitoring 12 to 24 hours after implantation may be reasonable because detection of complications could lead to early intervention such as adjustment of settings or lead revision (Class IIb; Level of Evidence C).**

Generator Change

Although the risks of major perioperative complications after nonendovascular procedures such as gen-

erator changes are low, the risk of system malfunction requiring reoperation caused by loose set screws and inadvertent lead problems is $\approx 1.5\%$.²⁹⁷ Patients with uncomplicated generator replacement do not need further monitoring after a short observation period.

Recommendation

- 1. Arrhythmia monitoring may be reasonable for the immediate postprocedure period (Class IIb; Level of Evidence C).**

Preexisting Rhythm Devices

ICD Shocks Requiring Hospital Admission

The evaluation of patients who present after an ICD shock includes determination of the cause, whether the ICD shock was appropriate, and whether hospitalization is required.

Recommendation

- 1. Arrhythmia monitoring is recommended for patients requiring hospitalization after an ICD shock for the duration of the related hospitalization until the precipitating event is treated (Class I; Level of Evidence C).**

ICD or Pacemaker: Admission for Unrelated Indication

Given the significant rise in pacemaker and ICD use in the United States,²⁹⁸ many patients with these implanted devices are admitted to the hospital for noncardiac reasons. In addition, as the cost and availability of external monitors and implantable recorders improve, a growing number of patients with these monitors will present to a hospital setting for noncardiac concerns.

Recommendation

- 1. Arrhythmia monitoring is not recommended for patients with preexisting rhythm devices who do not otherwise meet indications for active inpatient monitoring (Class III: No Benefit; Level of Evidence C).**

Stable With Wearable Defibrillator: Admission for Unrelated Indication

Patients deemed at risk for sudden death who are not candidates for ICD implantation, because of contraindications or because their higher risk is deemed transient, may receive a wearable cardiac defibrillator. These devices have monitoring capabilities and are able to deliver electric energy that is as effective as that of ICDs.²⁹⁹ With help and education from nursing staff and company representatives, patients who demonstrate self-management of the wearable cardiac defibrillator and ability to wear the

device reliably can be discharged to a nonmonitored setting such as home or skilled nursing facility.

Recommendation

- 1. Arrhythmia monitoring is not recommended for patients with a wearable cardiac defibrillator who are admitted for noncardiac reasons (Class III: No Benefit; Level of Evidence C).**

Other Cardiac Conditions

Acute Decompensated Heart Failure

Acute decompensation of chronic heart failure (ADHF) occurs as the result of a number of precipitating events such as volume overload; ischemia; anemia; progressive ventricular, respiratory, or renal failure; hypertension; exacerbation of comorbidities; new-onset AF; and infection. These same pathogenic entities also increase the risk of lethal and hemodynamically unstable arrhythmias during treatment. During the aforementioned clinical situations, the increases in demand for cardiac output exceed the ability of the heart to supply oxygenated blood to meet the metabolic demands of tissues. This is true of heart failure with reduced or preserved left ventricular function.

In new acute heart failure, the problem is usually ischemia from a new MI, inflammation from myocarditis or endocarditis, or mechanical related to ruptured chordae or papillary muscles leading to acutely insufficient valves. The addition of arrhythmic instability resulting from the hemodynamic effects of structural or ischemic failure is rapidly lethal if not identified quickly and treated.

Patients who have a new MI and those who present as hemodynamically unstable are readily admitted to an ICU where continuous electrocardiographic monitoring is the standard of care. However, the majority of patients with ADHF are admitted to non-ICU beds, most often for diuresis to achieve optimal volume status and to relieve congestion. No objective, prospective evaluation of telemetry monitoring in patients with ADHF admitted to non-ICU beds has been reported.

Many, especially those with dilated cardiomyopathy of both ischemic and nonischemic origin, have ICDs. The incidence of arrhythmias such as AF and nonsustained VT is high in patients with ADHF. Fibrosis, dilation, changes in ion currents, and other structural changes in the myocardium are proposed mechanisms of arrhythmias.³⁰⁰ In patients with nonischemic cardiomyopathy, female sex, lack of statin therapy, and elevated creatinine were independent risk factors for malignant ventricular arrhythmias.³⁰¹

Benza et al³⁰² reviewed the OPTIME-CHF (Outcomes of a Prospective Trial of Intravenous Milrinone for Exacerbations of Chronic Heart Failure) registry and concluded that new arrhythmias during an exacerbation of

heart failure were associated with both higher inpatient and 60-day morbidity and mortality. Opasich et al³⁰³ surveyed physicians about their perception of the usefulness of telemetry monitoring among 711 patients with heart failure (199 of whom were monitored) admitted to a heart failure unit. Electrocardiographic monitoring was used primarily in unstable patients, and telemetry was deemed useful in 70% of cases.

Although many patients with heart failure now have ICDs, the increased risk of ventricular arrhythmias with ADHF and the extremely unpleasant experience of defibrillation make reliance on the defibrillator unreasonable if the arrhythmia can be identified and suppressed, thus avoiding defibrillation. Most patients with ICDs have the antitachycardia pacing feature programmed "on" in an effort to minimize ICD shocks, which are not only uncomfortable but may be associated with an increased risk of mortality. In addition, antiarrhythmic drugs carry the risk of proarrhythmia and do not obviate the need for an ICD.

Recommendations

1. **Because of the risk of ventricular arrhythmias and the frequency of new-onset AF, arrhythmia monitoring is recommended for patients with ADHF until the precipitating event (eg, volume overload; ischemia; anemia; progressive ventricular, respiratory, or renal failure; hypertension; exacerbation of comorbidities; new-onset AF; or infection) is successfully treated (Class I; Level of Evidence B).**
2. **Because of the potential for new ischemia, ischemia monitoring may be reasonable, but only if there is a possible ischemic origin and in the setting of evaluable ST segments (Class IIb; Level of Evidence C).**

Endocarditis

Guidelines published in 2005 do not mention electrocardiographic monitoring for patients with endocarditis,³⁰⁴ although conduction abnormalities occur with moderate frequency in the setting of infective endocarditis. Limited data exist on the role of electrocardiographic monitoring of patients with this condition. Meine et al³⁰⁵ reported a series of 137 cases of endocarditis classified as definite or possible by the Duke criteria with an interpretable ECG. Conduction abnormalities of new or unknown duration occurred in 36 of these patients (27%). Among these 36 patients, conduction abnormalities included the following: 18 with intraventricular conduction abnormalities alone, 14 with atrioventricular block alone, and 4 with both intraventricular conduction abnormalities and atrioventricular block. The extent of the infection was significantly associated with the occurrence of conduction change on the ECG:

53% of those with invasive infection, defined as abscess or paravalvular regurgitation, exhibited a conduction change on the ECG compared with 26% of those with isolated valve infections ($P=0.046$). Conduction change on the ECG was associated with a 2-fold higher mortality compared with those without a conduction change on the ECG (31% versus 15%; $P=0.039$). Similarly, 41% of patients with intraventricular blocks that were new or of unknown duration died compared with only 15% of patients without intraventricular blocks ($P=0.003$).³⁰⁵ The occurrence of conduction abnormalities in the setting of infective endocarditis may be a marker of more advanced disease rather than serving simply as an arrhythmic cause of death. Telemetry is appropriate for patients with endocarditis who have signs or symptoms of heart block, heart failure, or high-risk features noted on central nervous system examination or echocardiogram (eg, ring abscess).

Recommendation

1. **Arrhythmia monitoring for patients with infective endocarditis can be beneficial until the patient is clinically stable (Class IIa; Level of Evidence C).**

Noncardiac Conditions

Continuous electrocardiographic monitoring of patients with noncardiac conditions occurs both in the ICU and outside the ICU. No randomized trials or observational studies could be found that describe how and whether differing forms of monitoring influence patient outcomes or care processes in the ICU. Although not explicitly required by The Joint Commission, electrocardiographic monitoring is standard in ICUs in the United States. If a patient is unstable enough to require an ICU, adequate rationale would need to be documented for why electrocardiographic monitoring would not be warranted. In general, it is reasonable that electrocardiographic monitoring should be continued until patients are weaned from mechanical ventilation and are hemodynamically stable. Continuous electrocardiographic monitoring in patients receiving intensive care for noncardiac indications is recommended.

Continuous electrocardiographic monitoring should not be used outside the ICU setting unless a clear clinical indication exists and for only as long as that indication is present. The decision to place a patient on electrocardiographic monitoring should not be based only on an assumption that the patient will be observed more frequently or that more intensive nursing care will be provided. This section examines the current available evidence for the use of continuous electrocardiographic monitoring of patients with noncardiac conditions in the non-ICU setting such as those undergoing procedures with conscious sedation, noncardiac surgical pa-

tients, and medical patients. The final section provides a brief discussion of electrocardiographic monitoring of patients with an order not to resuscitate or intubate.

After Conscious Sedation

Major cardiac events resulting from common procedures with conscious sedation (such as colonoscopy) are exceedingly rare, particularly in the ambulatory setting.^{306,307} The primary cause of these cardiac events is probably respiratory in nature, leading to nearly ubiquitous use of continuous pulse oximetry as a key monitoring modality. However, prospective studies are needed to determine causality.³⁰⁸ As others have pointed out,³⁰⁷ although intraprocedural ST-segment or QT changes appear to be associated with poorer postprocedural outcomes,³⁰⁹ few studies have provided empirical data on which patients benefit from electrocardiographic monitoring during conscious sedation, and such trials are unlikely to be carried out. Although few studies exist in which electrocardiographic monitoring is compared with other types of monitoring such as pulse oximetry and capnography, these may provide additional information or even alternatives to electrocardiographic monitoring for the patient undergoing minor procedures.

Recommendation

- 1. Arrhythmia monitoring may be reasonable in patients undergoing conscious sedation and should be continued until the patient is awake, alert, and hemodynamically stable (Class IIb; Level of Evidence C).**

Noncardiac Surgery

ACC/AHA guidelines for perioperative management of patients after major surgery³¹⁰ do not make recommendations for the use of continuous electrocardiographic monitoring as an approach to detecting major cardiac events (eg, MI, arrhythmias). However, the guidelines do make a specific recommendation against routine postoperative use of the static 12-lead ECG for screening of asymptomatic patients because of a lack of clinical trial data defining an optimal approach to acting on electrocardiographic findings in asymptomatic patients. In contrast, obtaining a postoperative 12-lead ECG would be appropriate if a patient becomes symptomatic after a noncardiac surgical procedure. In this scenario, if a patient becomes symptomatic (eg, has chest pain, symptoms of heart failure, or palpitations) and the ECG and clinical risk profile suggest a cardiac cause, other areas of these practice standards would be applicable as the basis for continuous electrocardiographic monitoring in a patient after noncardiac surgery.

Few studies exist to guide the use of perioperative electrocardiographic monitoring to manage patients who have preexisting arrhythmias (eg, AF) or who are at high risk for arrhythmias. The absolute risk of

new or worsening AF after noncardiac surgery is relatively low, between 1% and 2% in large studies, but is associated with higher costs and worse clinical outcomes.^{311,312} However, data are limited for defining a high-risk subgroup and a clinically effective approach to monitoring these patients. Postoperative patients with angina-equivalent symptoms or rhythm changes should be treated according to the chest pain/coronary artery disease standards above.

Recommendation

- 1. Routine use of arrhythmia monitoring after noncardiac surgery is not indicated for asymptomatic patients (Class III; Level of Evidence C).**

Two particular types of noncardiac surgery deserve special consideration related to the use of continuous electrocardiographic monitoring outside the ICU: major thoracic surgery and major vascular surgery.

Noncardiac: Major Thoracic Surgery. AF is by far the most common arrhythmia after thoracotomy and lung surgery.³¹³ For patients who undergo thoracic surgery even without direct cardiac involvement, the incidence of postoperative AF is dramatically higher (4%–37%) than for those who undergo nonthoracic surgery.^{313–316} Heterogeneity of samples and methods contributes to a lack of understanding of the true incidence. A review spanning 2002 to 2012 noted a 3.2% to 30% incidence of SVT after pulmonary surgery, with return to normal sinus rhythm before discharge for most patients.³¹³ However, challenges for these patients included hemodynamic consequences, potential for systemic embolization, increased length of stay, and potential need for long-term prophylactic medication. In a consecutive sample of patients undergoing pulmonary resection, the overall incidence of postoperative AF was 11.8%, with a quarter of patients experiencing AF in the first 24 hours; the incidence peaked at 2.5 days postoperatively.³¹⁶ Other investigators noted that AF develops most frequently 2 days after noncardiac thoracic surgery.^{315,317} Thus, it is reasonable to recommend electrocardiographic monitoring through postoperative day 2 to 3 for pulmonary resection, with longer periods determined by the clinician for those with multiple risk factors.

In 2 retrospective studies, the incidence of new postoperative AF was 16% to 17% with open thoracotomy and 10% to 12% with a video-assisted thoracic approach to pulmonary lobectomy.^{314,318} Ivanovic et al³¹⁶ reported a similar incidence (11.8%) of a new documented postoperative AF that required pharmacological therapy among patients undergoing pulmonary resection (n=363). Patients with postoperative AF had a significantly longer mean length of stay (10.5 versus 6.9

days). Ivanovic et al³¹⁶ described the AF as mainly transient and uncomplicated; however, one third of cases either were not transient or led to a major intervention. Sohn et al³¹⁵ reported that patients who developed AF had a longer hospitalization and increased in-hospital mortality (although the cause of death was noncardiovascular such as pneumonia or hemorrhage).

Risk factors for postoperative AF after thoracotomy have included older age, coronary artery disease, more extensive surgery or more serious stage of cancer, emergency surgery, male sex, and low body mass index.^{315,316} Although certain risk factors cannot be modified, further attention to pathogenesis may highlight directions for the prevention of postthoracotomy arrhythmias. The factors triggering new AF after cardiac or thoracic surgery may include direct intrathoracic stimulation or atrial irritation; thus, some have examined strategies for prophylaxis (eg, β -blockers or calcium channel blockers, amiodarone) with varying risks and benefits.^{313,319} Autonomic denervation and stress-mediated neurohumoral mechanisms have been recognized as contributing to frequent AF after thoracotomy.³¹⁸ A key intervention to preventing SVT in patients undergoing thoracotomy is aggressive pain management, including thoracic epidural analgesia.^{313,320}

Recommendations

1. **Arrhythmia monitoring is reasonable for patients after noncardiac major thoracic surgery such as pulmonary resection to identify AF through postoperative day 2 to 3 (Class IIa; Level of Evidence B).**
2. **Arrhythmia monitoring for a longer period of time can be useful for patients with multiple risk factors for AF after noncardiac major thoracic surgery (Class IIa; Level of Evidence B).**

Noncardiac: Major Vascular Surgery. Continuous arrhythmia monitoring in patients undergoing major vascular surgery such as invasive aneurysm repair deserves further study to determine benefit/nonbenefit in the postoperative period, given the very high rate of ischemia reported.³²¹ The risk factors for major vascular problems (eg, smoking, hypertension, hyperlipidemia, diabetes mellitus) are analogous to those of cardiac disease. These patients may have undiagnosed coronary lesions, which may be identified on postoperative ST-segment monitoring, triggering a need for evaluation and potential intervention. Because current data are limited, specific recommendations cannot be made for monitoring. For patients who develop new signs or symptoms suggestive of cardiac ischemia, heart failure, or arrhythmias, relevant practice standards apply. Further study is indicated for this at-risk population.

Medical Conditions

Among general medical patients, the limited data available suggest that the incidence of clinically important arrhythmias is quite low (<2%), even in a telemetry setting.^{322–326} Moreover, in noncardiac patients, serious and fatal arrhythmias tend to be a secondary manifestation of serious underlying comorbidities, and signs and symptoms of worsening clinical status do not require telemetry to detect.^{327–329} In fact, the few studies suggest that the rationale for monitoring may be more to detect and manage a broad range of complications, not just arrhythmias or myocardial ischemia.³²⁶ This section includes a discussion of the evidence currently available to guide the use or nonuse of continuous electrocardiographic monitoring among patients with the following medical conditions: stroke; pneumonia; COPD; severe electrolyte abnormalities; drug overdose, including specific types of drugs; and hemodialysis.

Stroke

Guidelines for the early management of patients with acute ischemic stroke (2013) recommended that arrhythmia monitoring should begin in the prehospital setting and continue throughout the initial assessment and management of acute stroke. The guidelines list a COR I, LOE B recommendation for arrhythmia monitoring for at least 24 hours after stroke to identify possible AF and other potentially serious cardiac arrhythmias that would necessitate emergency cardiac interventions.³³⁰ The 2009 AHA/American Stroke Association scientific statement on transient ischemic attack lists a COR IIa, LOE B recommendation for prolonged inpatient or Holter monitor use among patients with an unclear pathogenesis.³³¹

Kallmünzer and colleagues³³² evaluated the Stroke-Arrhythmia-Monitoring-Database to assess the risk and timing of serious arrhythmias in patients admitted with a stroke. Their analysis revealed that 25% of patients had significant arrhythmias (mostly ventricular or SVT) with ventricular rates in excess of 130 bpm. These arrhythmias were associated with older age and more severe neurological deficits measured on the National Institutes of Health Stroke Scale. The incidence of these arrhythmias was highest in the first 24 hours and declined over 3 days.³³²

Ritter and colleagues³³³ conducted a prospective study of 256 patients with ischemic strokes who were continuously monitored for at least 24 hours. In this study, 15% had episodes of a heart rate >120 bpm; this was associated with larger lesion size, higher National Institutes of Health Stroke Scale score, and AF. The authors concluded that continuous electrocardiographic monitoring allowed the detection of arrhythmias to support treatment decisions.

Electrocardiographic monitoring is useful for identifying significant arrhythmias in patients with strokes

that result in a change in therapy, including identification of AF and initiation of anticoagulation. Although arrhythmia monitoring is recommended in the setting of stroke up to 24 to 48 hours, the likelihood of documenting AF is low, and longer-term monitoring has been shown to have an increased yield in identifying patients with AF.³³⁴

QTc prolongation has been documented in patients who have experienced acute neurological events such as stroke or neurological trauma. It is most frequently documented among patients with subarachnoid hemorrhage, who are especially prone to QT prolongation. However, they rarely develop TdP,⁹⁸ and thus, QTc monitoring is not recommended.

The following recommendations are provided, with considerations for shorter monitoring in patients in whom the source of ischemic stroke is identified (eg, AF) and for longer monitoring in older and sicker patients.

Recommendations

- 1. Arrhythmia monitoring is recommended for patients with strokes for up to 24 to 48 hours after admission (Class I; Level of Evidence B).**
- 2. Arrhythmia monitoring can be useful for a longer duration in the setting of cryptogenic stroke to assess for intermittent AF and asymptomatic rapid ventricular response (Class IIa; Level of Evidence B).**

Patients with cardiac disease encompass a high-risk population for occurrence of stroke, whereas patients with acute stroke are similarly at increased risk for cardiovascular complications. Electrocardiographic abnormalities are frequent in the setting of acute stroke, described in 50% to 92% of patients studied,^{335–338} including ST-segment shifts suggestive of myocardial ischemia or injury in up to two thirds of patients with ischemic stroke.^{339–341} When evaluations for both ischemia and arrhythmia are combined, telemetry monitoring has been demonstrated to provide a high yield of abnormalities with important diagnostic and prognostic relevance. In an acute stroke unit study of 692 patients with acute cerebral infarction, 155 patients with intracerebral hemorrhage, and 223 patients with transient ischemic attack, 12 to 24 hours of electrocardiographic monitoring documented electrocardiographic abnormalities in the majority (60%, 50%, and 44%, respectively), including potential ischemic findings such as ST-segment elevation (5%, 5%, and 2%), ST-segment depression (16%, 7%, and 9%), and/or T-wave inversion (18%, 8%, and 11%).³⁴² The occurrence of electrocardiographic abnormalities was associated with impaired 3-month outcome in patients with cerebral infarction or hemorrhage but not those with transient ischemic attack in this study.³⁴²

Although it has been suggested that aggressive monitoring, including electrocardiographic monitoring, of patients in a stroke care unit may lead to improved outcomes,³⁴³ evidence for ST-segment monitoring is lacking. Indeed, the prognostic benefit of ST-segment monitoring in the setting of acute stroke, despite the clear shared association of risk factors and concomitant cerebrovascular and cardiovascular events, is further confounded by the frequency of ST-segment shifts identified in this population³⁴⁴ and the lack of clarity about cause and effect. Concurrent stroke and evidence of MI are not infrequent, with angina, MI, or evidence of cardiac ischemia present in up to 6% of patients with acute stroke, although associations with elevation of biomarkers of myocardial injury and left ventricular wall motion abnormalities vary by stroke type and location, noted particularly in association with subarachnoid hemorrhage.^{335,345,346} It has been postulated that these changes occur via a centrally mediated release of catecholamines resulting in subendocardial ischemia with elevation of cardiac biomarkers, although even this elevation is inconsistent and not clearly associated with remediable ischemia.^{347–349} Given the clear association between risk of stroke and incident cardiac disease, a high index of suspicion for inducible myocardial ischemia must be maintained in patients with acute stroke. However, given the frequent occurrence of evolutionary ST-segment and T-wave changes of unclear origin and significance, limited therapeutic options for hemodynamically stable MI in the setting of acute stroke, and a lack of prospective data to support a prognostic benefit for ischemia monitoring in patients with stroke, ST-segment monitoring should be considered only in patients with acute stroke at increased risk for cardiac events with evaluable ST segments and may require expert consultation.

Recommendation

- 1. ST-segment ischemia monitoring may be considered only in patients with acute stroke at increased risk for cardiac events who have evaluable ST segments (Class IIb; Level of Evidence C).**

Pneumonia

Recent studies and literature reviews have proposed an association between community-acquired pneumonia and cardiovascular-related events during hospitalization.^{350,351} Among patients hospitalized with pneumonia, cardiac arrest occurring without preceding shock or respiratory failure may be related to myocardial ischemia, a maladaptive response to hypoxia, or sepsis-related cardiomyopathy.³⁵² The possibility of proarrhythmic effects of antibiotics should be considered and may be mitigated by appropriate QTc monitoring.

Recent attention has been given to the frequency of arrhythmias among patients with pneumonia. Of 3068 hospitalized patients with pneumonia, 12% developed a cardiovascular event (defined as pulmonary edema, cardiac arrhythmia, or MI). Hyperlipidemia and severity of pneumonia were associated with increased risk.³⁵³ Similar findings were reported from another study of 32 689 patients with pneumonia and no prior diagnosis of a cardiac arrhythmia: 12% had a new diagnosis of cardiac arrhythmia (AF, VT/VF, cardiac arrest, and symptomatic bradycardia) within 90 days of admission.³⁵⁴ Older age, history of heart failure, and need for mechanical ventilation or vasopressors were associated with more events. Perry et al³⁵⁵ studied 50 119 inpatients admitted with pneumonia and identified congestive heart failure (10.2%), arrhythmia (9.5%), MI (1.5%), and stroke (0.2%) occurring primarily within the hospitalization period but also up to 90 days after the initial hospitalization.

Among 3921 patients with community-acquired pneumonia, Viasus et al³⁵⁶ identified 8 risk factors for acute cardiac events and mortality: age >65 years, chronic heart disease, chronic kidney disease, tachycardia, septic shock, multilobar pneumonia, hypoalbuminemia, and pneumococcal pneumonia. Patients with at least 6 of these factors were deemed high risk and had a 21.2% occurrence of cardiac complications ($P < 0.001$). Overall, mortality was higher in patients who had acute cardiac events. Thus, Viasus et al³⁵⁶ recommended electrocardiographic monitoring in patients with pneumonia who also had at least 6 of the identified risk factors. This may be a reasonable recommendation given that many patients with 6 of these risk factors, in addition to acute pneumonia, may already be in the ICU.

The standard practice for electrocardiographic monitoring for patients with pneumonia in the ICU is not being questioned, despite a lack of prospective, interventional studies evaluating outcomes of electrocardiographic monitoring in the ICU. However, the existence of a subset of high-risk patients on telemetry units who directly benefit from electrocardiographic monitoring is unknown. Further prospective study is needed to determine whether telemetry monitoring results in better outcomes in high-risk patients with the use of a valid risk stratification tool or whether these high-risk patients would meet criteria to be in the ICU where electrocardiographic monitoring is already the standard of care. At this time, evidence is lacking to provide recommendations for continuous electrocardiographic monitoring among non-ICU patients with pneumonia.

Chronic Obstructive Pulmonary Disease

Two major hypotheses for arrhythmogenesis in COPD are hypoxemia, hypercapnia, and acid-base disturbances and COPD-associated autonomic neuropathy that

decreases heart rate variability.³⁵⁷ The latter has gained attention as investigations of heart rate variability have increased.³⁵⁸

About one third of patients with stable COPD also have heart failure.³⁵⁹ Ischemic heart disease is likely a missed diagnosis among patients hospitalized with COPD exacerbation.³⁶⁰ Data from a large retrospective review demonstrated that COPD is an independent risk factor for cardiovascular disease.³⁶¹ However, prospective studies of patients with COPD receiving electrocardiographic monitoring are rare. Holter monitoring of a consecutive series of patients ($n = 7441$) demonstrated that the severity of COPD was independently associated with the occurrence of AF.³⁶²

Patients with concurrent COPD and AF may have increased breathlessness and should be treated according to usual AF guidelines; if β -blockers are used, β_1 -selective drugs are preferred.³⁶³ Because these patients are often excluded from clinical trials, more data on COPD medication in patients with AF are needed and could inform future electrocardiographic monitoring studies.

Patients with COPD often have electrocardiographic abnormalities; the degree of abnormality increases with disease severity. Abnormalities include the verticality of the P-wave axis ($> 60^\circ$) and narrowness of the QRS complex (< 75 milliseconds),³⁶⁴ as well as right atrial enlargement, right ventricular hypertrophy, and right BBB.^{365,366} Ischemic electrocardiographic changes are common among patients with COPD.³⁶⁷

The unanswered question is not whether cardiovascular disease and arrhythmias occur in patients with COPD but rather whether any new changes on the ECG will be clinically important, thus justifying electrocardiographic monitoring. Reports of research evaluating the use of telemetry for hospitalized patients with COPD are lacking. For example, patients with COPD demonstrating shortness of breath attributed to COPD exacerbation accompanied by T-wave inversion in V_1 through V_3 on ECG³⁶⁶ are not typically considered candidates for immediate revascularization. Thus, if a patient with COPD demonstrated ischemic abnormalities on telemetry, the clinical response is unclear in the acute setting. At this time, there is insufficient evidence to provide recommendations for electrocardiographic monitoring for patients with COPD.

Electrolyte Abnormalities

Abnormalities in potassium and magnesium levels can cause changes on the ECG.

Potassium. Hypokalemia, the most common electrolyte abnormality, is often the result of diuresis, but it may also result from the administration of potassium-free intravenous fluids, potassium loss from vomiting and diarrhea, and other endocrine and renal mechanisms.³⁶⁸ Hypokalemia has been defined as $K < 3.5$ mEq/L.^{369,370}

Various sources have classified hypokalemia with slight differences but typically as mild (3.0–3.5 mEq/L), moderate (2.5–2.9 mEq/L), or severe (<2.5 mEq/L).³⁶⁹ Although many noncardiac patients are asymptomatic until potassium levels are <3 mEq/L,³⁶⁹ those with rapid losses may be symptomatic sooner. It has been suggested recently that patients with heart failure maintain a level of at least 4 mEq/L.^{370,371} Changes on the ECG associated with hypokalemia are broadening of the T waves, ST-segment depression, and prominent U waves.³⁶⁸ A variety of arrhythmias have been associated with hypokalemia, including first- or second-degree atrioventricular block or AF. Ventricular arrhythmias include PVCs, VT, TdP, VF, and cardiac arrest.³⁷⁰

Hyperkalemia occurs less commonly but is a well-known challenge, primarily among patients with renal dysfunction. Upper limits range from 5 to 5.5 mmol/L, depending on the laboratory and institution.³⁷⁰ Changes on the ECG do not usually manifest until serum potassium levels are >6.5 mmol/L.³⁷² One of the most common findings on ECG for patients with hyperkalemia are nonspecific ST-segment abnormalities.³⁷³ Various sources have classified hyperkalemia with slight differences but typically as mild (5.5–6.4 mmol/L), moderate (6.5–8.0 mmol/L), or severe (>8.0 mmol/L).³⁷⁴ This classification helps illustrate the progressive effect on the ECG. As hyperkalemia progresses, the T wave often (but not always) becomes peaked (5.5–6.5 mmol/L), the PR interval lengthens (6.5–7.5 mmol/L), and the QRS widens (7.0–8.0 mmol/L).³⁷⁵ Bradycardia may occur in severe hyperkalemia as a result of the extremely prolonged PR and QRS.^{373,375,376} Finally, a sine wave pattern, VF, and asystole or pulseless electrical activity may be seen at potassium levels exceeding 10 mmol/L.^{372,374} It is important to note, however, that electrocardiographic manifestations for hyperkalemia vary among individuals and may not be predictable.^{345,373,377}

Magnesium. Magnesium deficiency is common, and magnesium has been associated with benefit in treating TdP. Differing normal values are reported, but <1.3 mEq/L is undisputedly low.³⁷² Keren and Tzivoni³⁷⁸ described a number of incidences in which administration of magnesium bolus or infusion resolved TdP in situations when it was preceded by both normal levels and hypomagnesemia. In an RCT among hospitalized patients with heart failure, hypomagnesemia was associated with more frequent ventricular arrhythmias, likely caused by diuresis; patients randomized to receiving magnesium supplements intravenously demonstrated significantly fewer PVCs.³⁷⁹ A second RCT reported significantly fewer PVCs after magnesium supplements.³⁸⁰

Magnesium toxicity is less common but seen in patients with renal dysfunction and as an iatrogenic

overdose possible in pregnant women receiving magnesium for preterm labor.³⁸¹ Magnesium levels of 2.5 to 5 mmol/L may manifest as prolonged PR, QRS, and QT intervals; severely elevated levels of 6 to 10 mmol/L may result in atrioventricular nodal conduction block, bradycardia, hypotension, and cardiac arrest.³⁷²

In summary, use of electrocardiographic monitoring among hospitalized patients with moderate and severe imbalances of potassium or magnesium facilitates the use of published algorithms to prevent or intervene for lethal cardiac rhythms. In less severe electrolyte abnormalities, if a 12-lead ECG demonstrates electric abnormalities, continuous arrhythmia monitoring should be considered.

Recommendation

1. Arrhythmia monitoring is recommended for moderate and severe imbalance of potassium or magnesium (*Class I; Level of Evidence B*).

Pediatric Considerations. Because the substrate of scarred, hypertrophic myocardium is much less common among children compared with adults, many pediatric clinicians believe that electrolyte abnormalities are less likely to be associated with electrocardiographic abnormalities. However, insufficient published data make specific recommendations for electrocardiographic monitoring for abnormal electrolytes in children difficult.

Drug Overdose

Overdose of drugs can occur purposely as an attempt at suicide or through error, accident, or inexperience. The drugs involved may be prescribed (for pain, anxiolytics, antidepressants), industrial (inhalants), or illegal/recreational (street drugs/club drugs). In addition, children can access and ingest medications of adults in the household. Regardless of the particular drug or the circumstances of the ingestion, all of these drugs have arrhythmogenic properties when consumed in toxic doses. The minimum toxic dose may be individually defined.

Psychotropic Drugs. Buckley et al³⁸² compared 39 patients with serious arrhythmias (VT, SVT, or cardiac arrest) with 117 patients without arrhythmias, all of whom were admitted with overdose of tricyclic antidepressants or thioridazines. Initial evaluation of QRS duration and QTc interval to predict serious arrhythmias was not helpful because they were prolonged in the majority of both groups. Monitoring of prolonged QTc is discussed in Section 1, Overview of QTc Monitoring, and Section 2, Arrhythmias.

Opiates. Heroin, methadone, and oxycodone (in either rapid-release or sustained-release formulations) are used by individuals who may overdose by error or design. Large doses of opiates cause central nervous

system depression that often manifests in respiratory depression.³⁸³ Methadone, in particular, may cause QT prolongation³⁸⁴ that worsens with overdose because of concomitant drug use, including methamphetamine,^{384–386} benzodiazepines, or cannabis.³⁸⁶ In addition, street heroin may be “cut” with cardioactive drugs such as quinidine, diltiazem, cocaine, procaine, lidocaine, phenacetin, methorphan, or caffeine.³⁸⁷

Inhalants. The inhalation (“huffing,” “sniffing,” or “bagging”) of substances such as toluene, butane, propane, fluorocarbons, chlorinated hydrocarbons, or acetone to experience an associated euphoria is commonly done by children and adolescents. However, it is an inexpensive high that is not limited to children. When combined with alcohol or drugs such as benzodiazepines, barbiturates, or sympathetic stimulation, inhalants can result in “sudden sniffing death syndrome.”³⁸⁸ This syndrome is thought to be the cause of at least 50% of the deaths resulting from inhalant abuse and likely is attributable to a combination of anoxia, vagal inhibition, and catecholamine surge.³⁸⁹ Prolonged use of toluene leads to renal dysfunction and a potential acute presentation of metabolic acidosis and potentially Goodpasture syndrome, which causes lung damage and renal failure.³⁸⁹

Cocaine. Ventricular arrhythmias that occur early after cocaine ingestion may be attributable to effects on sodium channels, whereas ventricular arrhythmias occurring later may be related to ischemia.³⁹⁰ Thus, it is possible that electrocardiographic monitoring may facilitate interventions for ischemia. Cocaine prolongs the QT interval for several days after ingestion,³⁹¹ requiring the avoidance of other QT-prolonging medications during this time. In 84 of 107 deaths (81%) associated with cocaine, no definitive cause was noted, leading investigators to hypothesize that MI with VF was the cause of unknown deaths among cocaine users.³⁹²

The most common symptom reported by cocaine users is chest pain³⁹⁰ described as heavy in nature.³⁹³ Unfortunately, correlation of MI with ECGs has been reported to be low in this population, so troponin measurement is essential.³⁹⁰ Risk stratification for observation status versus inpatient admission using established criteria (eg, changes on the ECG and troponin) is critical because although only 0.7% to 6%^{394,395} of patients initially presenting with cocaine-induced chest pain had a subsequent MI, among high-risk patients admitted for evaluation, 24% had an MI and another 24% were diagnosed with unstable angina.³⁹⁵ AHA guidelines recommend 9 to 12 hours of observation in a chest pain unit for those patients with nondiagnostic electrocardiographic findings and negative cardiac markers, whereas they recommend hospital admission with continuous electrocardiographic monitoring for high-risk patients.³⁹⁰

Other Recreational Drugs. Overdose of stimulant drugs such as 3,4 methylenedioxymethamphetamine (ecstasy), methamphetamine, or substituted cathinones (bath salts) commonly found at dance clubs and parties present challenges in the ED and in follow-up care. Risks include tachyarrhythmias, bradyarrhythmias, MI, hypothermia, hyperthermia, hypertension, acute neurological and psychological symptoms, and violent behavior.^{396–399} Ingestion of γ -hydroxybutyrate, originally developed as an anesthetic, may lead to excessive depressant side effects. Patients admitted after taking γ -hydroxybutyrate may have knowingly or unknowingly also ingested other drugs and thus may have additional side effects.⁴⁰⁰ Death is uncommon when appropriate management is instituted.

Summary of Drug Overdose. All of the psychotropic drugs, methadone, and the inhalants prolong the QT interval and predispose patients to ventricular arrhythmias. Stimulants predispose to arrhythmias by activating the sympathetic nervous system.³⁹⁶ The temperature regulation effect of several of the drugs is an added source of arrhythmogenesis.^{397–399} A majority of people who overdose have >1 drug in their system either because they purposely took multiple drugs or because the drug they knowingly took was mixed with other drugs without their knowledge. Patients with drug overdoses of these substances should undergo electrocardiographic monitoring until they are free of the influence of the drug and are ready for discharge from acute care. Specific recommendations for QTc monitoring are described in the QT section of these practice standards and corresponding table (Table 6).

Recommendation

- 1. Arrhythmia monitoring is indicated until the patient is free of the influence of the drug(s) and clinically stable (Class I; Level of Evidence B).**

Hemodialysis

Although experts from the National Kidney Foundation: Kidney Disease Outcomes Quality Initiative Workgroup⁴⁰¹ called for automatic external defibrillators in all outpatient hemodialysis clinics because the common occurrence of fatal arrhythmias among patients, electrocardiographic monitoring is generally not provided in outpatient centers. In contrast, most hospitals provide continuous electrocardiographic monitoring for inpatients undergoing hemodialysis. The benefit of monitoring of inpatients undergoing hemodialysis is not known. Some inpatients receiving hemodialysis demonstrate criteria for electrocardiographic monitoring as listed elsewhere in these practice standards. For example, for inpatients with new acute renal failure with severe electrolyte abnormalities (eg, hyperkalemia)

or acidosis, continuous electrocardiographic monitoring is recommended. Patients undergoing dialysis while in the ICU receive electrocardiographic monitoring. Patients being dialyzed for drug intoxication with proarrhythmic drugs should have QT monitoring in addition to arrhythmia monitoring. However, for stable patients who are hospitalized for a procedure such as repair of a clotted dialysis access or an orthopedic procedure, evidence is lacking on which to base recommendations for electrocardiographic monitoring.

Reports addressing both atrial and ventricular arrhythmias among patients on hemodialysis have been published. However, studies evaluating the use of electrocardiographic monitoring for patients on hemodialysis are limited. A high prevalence and incidence of AF in patients on maintenance hemodialysis have been identified through routine ECGs.⁴⁰² Using the ICD remote monitoring function, investigators demonstrated that AF was more frequent on hemodialysis days and increased during the hemodialysis procedure.⁴⁰³ Abnormal ECGs, including QTc prolongation, are common among patients on hemodialysis,⁴⁰⁴ and QT prolongation has been identified as an independent predictor of mortality for patients on hemodialysis.⁴⁰⁵ As described in a review of 5 studies, SCD has been noted in patients on hemodialysis, most commonly occurring during the 72-hour time frame between dialysis treatments and within the first 12 hours after receiving a treatment.⁴⁰⁶ The Kidney Disease Workgroup⁴⁰¹ provided guidelines for evaluation of heart disease on initiation of dialysis, including baseline and annual ECGs; however, no specific recommendations were provided for continuous electrocardiographic monitoring during inpatient hemodialysis. Studies demonstrating the occurrence of rhythm changes during dialysis that provided real-time data to clinicians to confirm or modify care during hemodialysis were not identified. Further study is needed to determine whether continuous electrocardiographic monitoring is associated with improved outcomes for patients receiving hemodialysis in non-ICU settings.

Patients with severe hyperkalemia or metabolic imbalance (such as in new, acute renal failure) or who are hospitalized for another condition for which a COR I indication exists should have arrhythmia monitoring, including possible QTc monitoring for QT-prolonging medication.

Recommendation

- 1. Efficacy of arrhythmia monitoring for all hospitalized patients receiving chronic hemodialysis is not well established (Class IIb; Level of Evidence B).**

Do Not Resuscitate/Do Not Intubate

There are no RCTs examining the uses or outcomes of continuous electrocardiographic monitoring in patients

who have requested do not resuscitate (DNR) or do not intubate (DNI) status. It is important to note that a request for DNR or DNI (or both) care limitations provides only a very limited narrowing of care, even for severely ill patients, and should be clearly delineated from patients transitioned to comfort-focused end-of-life care for whom monitoring would not be indicated. Monitoring in patients with DNR or DNI status would therefore be indicated if it would guide therapy congruent with the patient's overall care wishes.

DNR or DNI status does not limit the use of other treatments (eg, vasopressors, oxygen support, or electrolyte replacement) and can be suspended when surgery or invasive procedures are planned.⁴⁰⁷ As a result, in patients with DNR or DNI status, clinical judgment and evidence from other aspects of these practice standards should be used to guide whether specific electrocardiographic monitoring is used. Arrhythmia monitoring may be considered if findings would trigger interventions consistent with patient wishes (eg, rate control if symptomatic); practice standards for related conditions could then be applied. Arrhythmia monitoring is not recommended for patients when data will not be acted on and comfort-focused care is the goal. Ischemia monitoring can be useful if the patient is a candidate for angiography with temporary reversal of DNR status, as well as when electrocardiographic monitoring facilitates medication adjustment to promote comfort.

Summary: Other General Medical Patients

There are a variety of patient populations for whom clinician judgment is needed to decide whether they are appropriate for an ICU, telemetry, or nonmonitored unit. The clinician judges whether a patient is stable using parameters such as blood pressure, heart rate, oxygenation, mental status, and signs and symptoms of angina. Certain patient conditions may be appropriate on medical-surgical and telemetry/progressive care/step-down units versus the ICU, depending on hemodynamic stability. For example, Patient A with a gastrointestinal bleed may be very appropriate for a medical unit while receiving a transfusion and awaiting endoscopy. However, Patient B presents with a gastrointestinal bleed and associated SVT to the extent that she has lightheadedness and chest pain. She may benefit from being in a telemetry/progressive care unit with arrhythmia monitoring, as well as potential continuous ST-segment monitoring for demand ischemia if there is interprofessional agreement for this. Finally, Patient C presents with a gastrointestinal bleed and hemodynamic instability to the extent that she needs vasopressors and thus belongs in an ICU until stabilized. A similar approach can be taken by the clinician caring for the patient with sepsis, for example, to decide whether electrocardiographic monitoring is appropriate.

Prescribers, nurses, and hospital administrators must work cooperatively to identify the appropriate and safe level of care for each patient without using electrocardiographic monitoring as a surrogate for better staffing ratios. It is reasonable to use telemetry monitoring for general medical patients who have clinical symptoms or electrocardiographic or laboratory abnormalities that provide an indication for telemetry as outlined elsewhere in these practice standards. However, the utility of continuous electrocardiographic monitoring in detecting life-threatening arrhythmias or ischemia in general medical patients is uncertain, and a cost-effective approach remains to be determined.

SECTION 3: ORGANIZATIONAL ASPECTS: ALARM MANAGEMENT, EDUCATION OF STAFF, AND DOCUMENTATION

In addition to knowing when electrocardiographic monitoring is indicated (and when it is not), other considerations are relevant to ensure the effectiveness and safety of monitoring. This section covers the following as they relate to electrocardiographic monitoring: alarm management, education of staff, and documentation.

Alarm Management

Alarm Hazards

The myriad alarm-enabled medical devices in use today, coupled with the large number of false or nonactionable alarm signals, have created a noisy environment prone to patient safety risks. The sources of a large proportion of alarm signals in hospitals are electrocardiographic monitors. In an observational study, Drew et al⁵⁰ reported a total of >2.5 million unique monitor alarms in 5 ICUs over 31 days.

Since 2007, the ECRI Institute has published a top 10 health technology hazards list that identifies sources of danger involving medical devices and steps to minimize the likelihood of adverse patient events. Alarm hazards has been at or near the top of this list since its inception.^{48,52,408} The Joint Commission made alarm management the focus of a National Patient Safety Goal that was phased in between 2014 and 2016. This should result in major efforts to improve alarm management.⁴⁰⁹

Alarm signals are triggered by either patient or technical issues. Patient alarms are specific to the clinical status of the patient such as arrhythmias or low heart rate. Technical alarms are triggered by equipment-related problems such as impending telemetry battery depletion.⁴⁷ The priority of alarm signals is classified according to the seriousness of the problem causing the alarm-triggered event. High-priority alarm signals

require an immediate response; medium priority indicates that a prompt response is necessary; and low-priority alarm signals indicate that clinician awareness is required.

Alarm hazards include an array of problems that may occur with alarm-equipped medical devices such as electrocardiographic monitors. These interrelated hazards include alarm fatigue, inadequate alarm response, and lack of a reliable alarm notification system.

Alarm Fatigue

Alarm fatigue occurs when clinicians are barraged by so many false or nonactionable alarm signals that they become desensitized. False alarms occur when there is no valid triggering event, whereas nonactionable alarms correctly sound but for an event that has no clinical relevance.

Research indicates that 68% to 99% of alarm signals are false or nonactionable.^{50, 200,410–412} The Joint Commission's Sentinel Event database includes reports of 98 alarm-related events between January 2009 and June 2012. Of the 98 reported events, 80 resulted in death, 13 in permanent loss of function, and 5 in unexpected additional care or extended hospital stay.⁴⁹ From 2005 to 2008, the US Food and Drug Administration reported 566 deaths linked specifically to monitor alarms.^{54,413}

Medical devices generate enough false and nonactionable alarm signals to cause a reduction in response known as the "cry wolf" effect. Alarm signals are designed to interrupt and call attention to problems, but if not clinically significant, they are distracting and interfere with the performance of critical tasks. Excessive false and nonactionable alarm signals have led to desensitization, such that nurses develop mistrust with the alarm system, may take unsafe actions like disabling alarm systems,^{414–416} and are less likely to act on real events.^{414–418}

Inadequate Alarm Response

National surveys of healthcare providers indicate that false and nonactionable alarms occur frequently, disrupt patient care, and reduce trust, thereby causing staff to take inappropriate actions such as disabling or ignoring alarm signals.^{417,418} A delayed response to alarm signals or no response at all compromises patient safety.⁴¹⁹

Alarm response is affected by the rate of exposure to nonactionable alarms, perceived alarm urgency, and workload conditions. In an observational study using video, the response time of nurses to alarm signals increased incrementally as the number of nonactionable alarms in the preceding 120 minutes increased.⁴²⁰ Other research indicated that the probability of responding to an alarm signal is related to the perceived true alarm rate. If an alarm signal is perceived to be reliable 90% of the time, the response rate will be ≈90%. Conversely, if the alarm signal is perceived to be 10% reliable, the response rate will be only ≈10%.⁵⁵ Response

to alarms improves in low-workload conditions and when staff has not developed mistrust for the alarm system.⁴¹⁶ Nurses adjust the order of their activities by evaluating the urgency of the alarm in relation to the condition of the patient and do not rely solely on the alarm sounding.⁴¹⁵

Minimizing nonsignificant alarm signals^{47,421–425} and tailoring acoustic properties of audible alarm signals to the urgency of the triggering situation are critical to improving alarm response.^{426,427}

Alarm Notification: Monitor Watcher Versus Medical Device Data Systems

Various ancillary alarm communication methods have been proposed to ensure that those providing care to patients are notified of true and actionable alarms. These methods include the use of monitor watchers and medical device data systems (MDDSs) that route alarms to the care provider's wireless device using alarm algorithms.^{428–430} The need for human oversight in the interpretation of electrocardiographic monitoring data remains as important today as it was with the advent of continuous electrocardiographic monitoring >50 years ago.¹

Funk et al⁴³¹ demonstrated little to no difference in patient outcomes when dedicated monitor watchers were used, although it is important to note that this study was conducted 20 years ago. It is unknown whether the structural changes in hospital-based healthcare delivery involving consolidation into larger health systems, trends of higher-acuity patients in the non-ICU setting, and an increasing focus on alarm management in the past 5 to 10 years may alter strategies in favor of centralized monitoring with monitor watchers. Despite the lack of studies to support the benefit of human monitor surveillance, national surveys revealed that this alarm notification strategy is used in 47% to 61% of hospitals.⁴³² Monitor watchers may be based on the unit, in a nearby ICU, or in a central remote location where monitors are watched from multiple patient care units. Methods to communicate from a centralized telemetry monitoring area to a bedside caregiver have been studied. Use of a voice badge significantly shortened the time to first contact, time to completion, and rate of closed-loop communication, resulting in more timely bedside care.⁴³³

The number of waveforms a monitor watcher can effectively and safely observe is not known, although a recent study used simulation to compare response time of monitor watchers to VF over 5 different patient loads (16, 24, 32, 40, and 48 patients). As patient loads increased, response times increased significantly. Frequency of failure to meet a response time goal of <20 seconds was significantly higher in the 48-patient condition than in all other conditions.⁴³⁴ The number of waveforms observed is also dependent on the lay-

out and location of the screens, the type of monitoring and alarm notification system in use, alarm burden, and whether the staff is expected to watch more than electrocardiographic waveforms (eg, oxygen saturation measured by pulse oximetry [SpO₂], end-tidal CO₂, blood pressure).

Potential monitor watchers have included nurses, monitor technicians, nursing assistants, and unit secretaries. The minimum educational preparation of monitor watchers, the content and length of orientation, and the frequency of ongoing education and competency evaluations vary widely.⁴³²

An MDDS, also known as middleware, can integrate multiple alarms from various medical devices such as monitors, patient call systems, ventilators, and infusion pumps. An MDDS uses alarm escalation rules that allow closed-loop communication. This alarm notification system sends alarms from a primary to an ancillary notification device such as a telephone, pager, or voice badge. It uses algorithms with slight delays to allow alarm autocorrection before routing the alarm to the designated caregiver. Many false alarms are caused by patient movement or staff manipulation of the patient when positioning or bathing. The slight alarm delay allows caregivers in the room to silence the alarm before it is sent through the ancillary notification device, thus reducing the number of false and nonactionable alarms sent. However, before any ancillary notification system is implemented, alarms must be properly managed to reduce the alarm burden.⁴³⁰

Methods to Reduce Alarm Hazards

Research on methods to reduce alarm hazards is in its infancy. In its 2014 National Patient Safety Goals, The Joint Commission emphasized that hospitals must identify the most important alarms to manage on the basis of their own internal situations.⁴⁰⁹ In response, hospitals are undertaking quality improvement (QI) projects to determine the best ways to reduce alarm hazards in their specific environments. The results of some of these QI projects have been published.^{47,430,435–437} Often in QI projects, hospitals have bundled their alarm interventions, thus making it impossible to identify which intervention influenced the outcome.^{47,430,435,437} The following approaches to reducing alarm hazards should be considered.

Interdisciplinary Committee

Interdisciplinary teams have been identified both in QI projects^{47,435,436,438–440} and by experts⁴⁴¹ as essential for addressing alarm management. Typically, teams are composed of nurses, physicians, clinical engineers, quality and safety experts, information technology professionals, and senior hospital leadership.^{47,440} Some teams also include a representative from the monitoring equipment manufacturer⁴³⁸ and families of patients.⁴³⁶ In an integrative review, an interdisciplinary alarm man-

agement committee was identified as key to selecting alarm management strategies.⁴⁴²

Recommendation

- 1. An interdisciplinary committee should be instituted to address alarm management (Class I; Level of Evidence C).**

Alarm Data Assessment

A monitor alarm assessment includes a collection of alarm signal data to identify the types of alarm conditions that occur within the monitoring system.^{47,440} This can be done through an MDDS with annotation,⁵⁰ by direct observation,⁴²³ or via video recording.^{410,443} The MDDS can produce electronic reports of alarm data in multiple formats such as in tables or graphs. It can handle an unlimited number of alarms, which can be sorted by frequency and duration and by unit, bed, or time. The benefit of direct observation and video recording is the ability to see actions taken by the caregiver in response to alarm signals and to determine whether an alarm signal is false or nonactionable. Assessment of alarm data is essential in the evaluation of the effect of alarm reduction interventions.

Recommendation

- 1. Data should be used to guide decisions about alarm management (Class I; Level of Evidence C).**

Default Alarm Presets

Default settings for monitor alarms are activated by the alarm system without operator action any time the monitor is turned on or a new patient is admitted. Default monitor alarm settings are preset by the manufacturer but with most systems can be changed. Reviewing alarm data is essential to standardizing monitor alarm default settings. Alarm default settings need to be examined carefully to eliminate nonactionable alarms such as some PVC alarms and duplicative alarms, for example, AF and irregular heart rate. Studies have indicated that adding a short delay before the alarm activates can eliminate a significant number of nonactionable alarms, especially related to SpO₂ and ST-segment alarms.^{50,423,440} Standardizing monitor alarm default settings across similar populations (eg, intensive care, telemetry, pediatrics, neonatal) is a reasonable strategy in alarm management and has been successfully used in QI projects.⁴⁴⁰

Recommendation

- 1. Manufacturer alarm default settings should be evaluated and adjusted as needed according to the population being monitored (Class I; Level of Evidence C).**

Alarm Customization

Monitor alarm customization, based on individual patient need, has been shown to decrease the number of nonactionable alarm signals.^{47,422,440} In a QI project, revising default alarm settings, customizing alarms on the basis of patient need, and providing nursing education resulted in a 43% reduction in high-priority alarm signals. This QI project focused on the most frequent, duplicative, and staff-perceived “nuisance” alarms.⁴⁷

Recommendation

- 1. Protocols should be developed that encourage nurses to adjust alarms for individual patients within established parameters (Class I; Level of Evidence C).**

Skin Preparation

Electric signals are transmitted via the epidermis to reach the electrodes that sense their signal. As the epidermal cells mature, they begin to degenerate. The stratum corneum sheds millions of skin cells daily and may be the source of some problems with the quality of electrocardiographic tracings. Removing this outer layer is important because the dead skin cells contain dirt and oil that may increase skin impedance and compromise the cardiac signal.⁴⁴⁴ Conductivity of the signal can be enhanced by proper skin preparation before electrocardiographic electrodes are placed.^{444,445}

The purpose of skin preparation is to cleanse the area to optimize signal transfer. Research,⁴⁴⁶ QI projects,^{435,437} and expert opinion^{445,447} have identified proper skin preparation before electrocardiographic electrode application to optimize conductivity. The skin is prepared by cleansing the area and wiping it with a dry washcloth.^{435,447} Alcohol should not be used because it dries the skin and may diminish electric flow.⁴⁴⁷ Melendez and Pino⁴⁴⁶ showed that interference was reduced with proper skin preparation as one of their interventions. However, they did not specifically identify their mode of skin preparation. In addition, clipping excessive hair before electrode application contributed to optimizing signal acquisition.^{435,447}

Recommendation

- 1. The skin should be prepared before electrode placement (Class I; Level of Evidence B).**

Electrocardiographic Electrodes and Lead Wires

Experts agree that old or dried electrodes, motion artifact, and poor skin-electrode contact trigger monitor alarms and result in disruption in patient monitoring. One published QI project demonstrated that daily electrocardiographic electrode changes decreased the number of alarms per monitored bed by 46% in both

a cardiology care unit and a progressive care unit.⁴³⁵ In another study that included an alarm annotation protocol, only 9% of the false arrhythmia alarms were rated as having poor signal quality.⁵⁰ The integrity of monitoring can be maintained by using fresh electrodes that are opened immediately before use. Results of a recent single-center comparative-effectiveness study revealed that disposable electrocardiographic lead wires were associated with fewer technical alarms than reusable lead wires.⁴⁴⁸

Recommendation

1. Evaluation of the integrity of electrodes and lead wires and change of electrodes should be done at a minimum of every 48 hours (Class I; Level of Evidence C).

Avoiding Unnecessary Electrocardiographic Monitoring

Despite the clearly defined electrocardiographic monitoring criteria in the previous AHA practice standards,¹ telemetry monitoring is overused. Studies that examined appropriate electrocardiographic monitoring, on the basis of the 2004 AHA practice standards criteria, found that 35%^{449,450} to 43%⁵⁶ of patients being monitored in non-ICU areas had no clinical indications for doing so. In addition, baseline data from the PULSE trial (Practical Use of the Latest Standards of Electrocardiography) revealed that 85% of patients in cardiac units with no indication for monitoring were on a monitor.⁴⁵¹ Telemetry discontinuation protocols following specific criteria for monitor discontinuation have been used to minimize false and nonactionable alarms.⁴⁵² Others have integrated the AHA practice standards¹ into their electronic ordering system on the basis of the right indication for the right duration and found no apparent increase in mortality, cardiac arrest, activation of the rapid response team, or life-threatening arrhythmias.^{59,453} After an intervention consisting of education and strategies to implement and sustain change in practice in the PULSE trial, the proportion of appropriate telemetry monitoring increased significantly.⁶

Policies and Procedures

Policies and procedures to guide practice for alarm management are key to reducing alarm hazards and are mandated as part of The Joint Commission's National Patient Safety Goal.⁴⁰⁹ Policies and procedures should include patient issues (eg, alarm limit defaults, criteria for customizing alarm limits according to patient condition) and system issues (eg, timing of telemetry battery replacement, who replaces the batteries).

The 2014 National Patient Safety Goal⁴⁰⁹ requires policies and procedures on the following:

- Clinically appropriate settings for alarm signals
- When alarm signals can be disabled

- When alarm parameters can be changed
- Who in the organization has the authority to set alarm parameters
- Who in the organization has the authority to change alarm parameters
- Who in the organization has the authority to set alarm parameters to "off"
- Monitoring and responding to alarm signals
- Checking individual alarm signals for accurate settings, proper operation, and detectability

Education

Education about the purpose and proper operation of alarm systems is part of the National Patient Safety Goal.⁴⁰⁹ Education has also been identified in QI projects^{47,439,440,454} and by experts as a critical part of the process of alarm management. Education about alarm management is beneficial on both an initial and ongoing basis for nurses and physicians.^{417,440,441} Ongoing education should be budgeted when monitoring systems are purchased.

Device Improvements

Although clinicians bear significant responsibility for reducing alarm hazards, monitor manufacturers are responsible for making monitors more intuitive, using principles of human factors engineering. In discussing the results of their large observational study, Drew et al⁵⁰ suggested the use of all available electrocardiographic leads to identify leads without artifact and those with adequate QRS amplitude, prompts to help in customizing alarm settings, and delays for certain parameters before alarms are triggered. Some newer monitors consider other parameters before alarming such as considering blood pressure before alarming for asystole.⁴²¹ By incorporating information from other parameters, a multiparameter alarm system can determine a more reasonable hypothesis of the cause of an alarm and suppress false alarms.

The ability to perform continuous ST-segment monitoring is limited by current electrocardiographic monitoring equipment. False and nonactionable alarms from ST-segment monitoring could be reduced by monitor enhancements such as adding customizable delays for ST-segment alarms to allow autocorrection of momentary threshold breaches caused by movement or position changes. Other potential improvements include redesigning ST-segment alarms so that the monitor alarms only when ST-segment changes occur in 2 contiguous leads and adding an option for ST-segment alarms to be visual (low priority, flashing) but not audible.

Although upgrading to the latest monitoring systems with enhanced features has significant financial implications, it may need to be a priority if it can reduce alarm fatigue and enhance patient safety. However, to avoid further alarm fatigue, caution needs

to be taken before alarm notifications are added to electrocardiographic monitors (eg, specific respiratory alarms that duplicate those on ventilators) without the consensus of clinicians. It is wise to pilot test upgraded monitors before fully incorporating them into the entire hospital system.

Pediatric Considerations

As is the case in units serving adult patients, alarm signals from electrocardiographic monitors are also ubiquitous in pediatric units, and most are false or nonactionable.⁴⁵⁵ A number of issues unique to the monitoring of pediatric patients may lead to excessive false or nonactionable alarms. Pediatric units care for patients whose ages range from birth to young adulthood, resulting in a heterogeneous population physically, intellectually, and emotionally. Further confounding the situation, patients may have an underlying condition in which their developmental age is disparate from their chronological age. An infant or young child may have a developmentally appropriate substantial elevation in heart rate when approached by an unfamiliar person or confronted by an unfamiliar situation. In addition, as the child attempts to avoid an uncomfortable situation, monitor electrode dislodgement or movement artifact may occur. These developmentally appropriate responses may trigger a false alarm that then has to be evaluated.

Alarm systems have preset default settings that are broadly based on age.^{455,456} These default settings have to be customized to the patient and evaluated frequently to minimize false alarms and to maximize the detection of clinically relevant arrhythmias.⁴³⁶ Dandoy et al⁴³⁶ described a QI project to test a team-based intervention to reduce alarms on a pediatric unit. They reported that their intervention reduced the median number of alarms per patient-day from 180 to 40. In addition to customizing alarm settings, they recommend that electrodes be replaced daily with a pain-free approach and monitors be discontinued when no longer clinically indicated.

Recommendations for Further Research

More research focusing on signal quality, networking of medical devices at the bedside, diagnostic alarms and predictive warnings, usability of alarm systems, creation of annotated clinical databases for testing, standardization efforts, patient monitoring in non-ICU and non-step-down unit settings, ancillary alarm notification, and education of healthcare providers is recommended.⁴⁵⁴ Research also needs to focus on which interventions will reduce false or nonactionable alarm conditions and the best approach to increasing the specificity of an alarm condition without an unacceptable loss of sensitivity.⁴⁵⁴

Arrhythmia algorithms are considered proprietary by monitor manufacturers. However, biomedical engineers and clinicians need to have input into the condi-

tions that will elicit alarm signals. Manufacturers should make available the sensitivity and specificity of their arrhythmia algorithms. Future research and development by manufacturers should include multiparameter alarms (eg, consider blood pressure or Spo_2 before alarming for asystole), use of short delays to allow alarm signal autocorrection, and autocustomization of alarm limits according to patient status.

Consideration of the design of future research is critical. RCTs are needed. Comparative-effectiveness trials would be the next step after RCTs. Studies must be interdisciplinary, emphasizing collaboration across industry, engineers, and clinicians. The focus needs to be on meaningful patient outcomes rather than just on the reduction of the number of alarms. Statistical power may be lacking to detect the effect of interventions on mortality and sentinel events. Large multicenter studies and appropriate surrogates for outcomes are necessary.

Education of Staff

Education is a critical aspect of the process in electrocardiographic monitoring.^{1,39,447,457,458} Adequate education is crucial for correct interpretation of electrocardiographic waveforms and data and proper care of patients undergoing continuous electrocardiographic monitoring. Incorrect interpretation can result in unnecessary diagnostic or surgical interventions.^{5,38}

Published reports note that both physicians and nurses have performed poorly in the assessment of rhythms from 12-lead ECGs or from continuous electrocardiographic monitoring.^{458–461} Sobering findings were reported by Viskin et al,⁴⁶² who asked 902 physicians from 12 countries to measure QT and to calculate QTc in 4 sample ECGs. For the 2 electrocardiographic samples with prolonged QTc, >80% of arrhythmia experts were able to calculate the QTc correctly compared with <50% of general cardiologists and <40% of noncardiologists. Fewer than 25% of general cardiologists and noncardiologists were able to classify all QT intervals correctly as either long or normal. In an examination of cardiology versus noncardiology fellows in interpreting 12-lead ECGs, Novotny et al⁴⁶³ found that although the rate of correct diagnosis was more frequent by cardiology fellows (70.1%) than noncardiology fellows (55.0%), it was still inadequate.

Among nurses in a quasi-experimental study to improve QT-interval monitoring, Pickham et al⁴⁵⁸ found that at baseline 94% of nurses were unable to calculate the QTc interval. After education, this skill improved, but still only half were able to calculate QTc correctly. In a later multisite, quasi-experimental study to improve QT-interval monitoring, Sandau et al⁴⁶⁴ augmented on-line education with computerized enhancements to electronic health records to notify nurses of patients on QT-prolonging medications and computerized calcula-

tion of QTc on the nurse entering QT and heart rate, resulting in significantly improved QTc documentation. In the PULSE trial, accuracy of documented arrhythmia interpretation by nurses improved from 82% correct to 97% after an interactive online electrocardiographic monitoring education program in the experimental group.⁶ Both nurses^{465,466} and physicians^{467,468} report a significant increase in knowledge with an educational intervention for electrocardiographic monitoring.

Variable results are seen with pediatricians interpreting 12-lead ECGs. Wathen et al⁴⁶⁹ found an 87% concordance rate between emergency room pediatricians and pediatric cardiologists. However, in examining accuracy rates with pediatric residents, Snyder et al⁴⁷⁰ demonstrated no difference between senior pediatric residents and interns, with complex arrhythmias being correctly interpreted only 28% (interns) to 45% (pediatric residents) of the time. Crocetti and Thompson⁴⁷¹ found that pediatric residents who completed a pediatric cardiology rotation were better able to correctly interpret ECGs than those who had not ($P=0.001$).

The ACC established standards for training fellows in clinical cardiology for ECG and ambulatory ECG.⁴⁷² The standards are based on the 6 general competencies endorsed by the Accreditation Council for Graduate Medical Education and the American Board of Medical Specialties and endorsed by the American Board of Internal Medicine with recommended time points within the fellowship training for the competency to be achieved, including 3000 to 3500 ECGs read within 36 months.⁴⁷²

Although such competencies do not currently exist for nurses, patient care needs on each unit should guide the clinical leadership team to identify the electrocardiographic monitoring priorities and to establish content and processes for baseline and ongoing education.⁴⁴⁷ For example, a unit in which patients include those undergoing ICD placement should have required initial education and ongoing updates as technology changes to support knowledge to assess both patient and device appropriately (ie, including recognition of antitachycardia pacing, overpacing, or underpacing).

Continuous electrocardiographic monitoring is performed in a wide variety of inpatient clinical areas and managed by nurses, with the assistance of monitor watchers in some facilities. Although it is recommended that minimal electrocardiographic monitoring competencies be established for healthcare providers who monitor patients, published competencies or educational standards for nurses or monitor watchers are lacking.^{447,459}

Elements to include in education related to electrocardiographic monitoring include the following:

1. Goals of monitoring, that is, arrhythmia, ischemia, and prolonged QTc. For example, for patients in the early phase of ACS who are on continuous

ST-segment ischemia monitoring, the goal is to enable the clinician to identify myocardial ischemia as soon as possible to promote rapid implementation of reperfusion strategies and to optimize patient outcomes. If the detection of ischemia is the primary goal of monitoring, then nurses should know to select the monitor lead(s) most likely to reveal ischemic changes, in addition to, or instead of, leads likely to be diagnostic of arrhythmias.

2. Electrode placement and skin preparation. Although placement of electrodes is a critical aspect of education because misplaced electrodes can result in misdiagnosis and inappropriate treatment,^{39,447,473–478} it is commonly done incorrectly. Studies have demonstrated that misplaced electrode placement of hospitalized patients ranged between 20% and 80%.^{6,479} Among patients in the PULSE trial,⁶ correct placement of the precordial electrode (defined as being in any of the correct V_1 – V_6 sites) was only 20% at baseline but improved to 59% immediately after the interactive online educational intervention and to 65% 15 months later. Proper preparation of the skin before placement of electrodes and regular replacement of electrodes should also be part of education to improve electrocardiographic monitoring and to minimize false alarms resulting from artifact.
3. Interpreting electrocardiographic waveforms and data. Interpreting waveforms and data includes understanding normal and abnormal rhythms (Table 8), general electrophysiological concepts (Table 9), and specific monitoring skills (Table 10).^{1,447,460,480} The content of electrocardiographic monitoring education needs to match the nature and complexity of the patient population served. Unit nursing leaders and educators are responsible for annually assessing the content of ongoing education on the basis of the electrocardiographic monitoring needs of patients in their care.
4. Appropriate response to an electrocardiographic abnormality.^{39,447,457,481} The appropriate response to an observed electrocardiographic abnormality will differ depending the team members, that is, physician, advanced practice nurse, physician assistant, staff nurse, or a monitor watcher, and their scope of practice.^{447,457} For example, for a patient with new AF, a monitor watcher would inform the staff nurse, who would assess the patient's hemodynamic status and laboratory values. The nurse would then notify the physician, advanced practice nurse, or physician assistant, who would take appropriate action. Education also should include when to call a rapid response team or a "code blue." Case studies appropriate for the type of clinician may be helpful in

Table 8. Education* Related to Specific Abnormalities on ECG

| |
|--|
| Normal rhythms |
| Sinus rhythm |
| Sinus bradycardia |
| Sinus arrhythmia |
| Sinus tachycardia |
| Intraventricular conduction defects |
| Right and left BBB |
| Aberrant ventricular conduction |
| Bradyarrhythmias |
| Inappropriate sinus bradycardia |
| Sinus node pause or arrest |
| Nonconducted atrial premature beats |
| Junctional rhythm |
| Atrioventricular blocks |
| First degree |
| Second degree |
| Mobitz I (Wenckebach) |
| Mobitz II |
| Advanced (≥2:1) |
| Third degree (complete heart block) |
| Asystole, pulseless electric activity |
| Sino-ventricular rhythm (eg, in severe hyperkalemia) |
| Tachyarrhythmias |
| Supraventricular |
| Atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia |
| AF |
| Atrial flutter |
| Multifocal atrial tachycardia |
| Atrial tachycardia |
| Accelerated junctional rhythm |
| Ventricular |
| Accelerated ventricular rhythm |
| Nonsustained/sustained monomorphic VT |
| Nonsustained/sustained polymorphic VT |
| Prolonged QT interval–associated ventricular ectopy, TdP |
| VF |
| Premature complexes |
| Supraventricular (atrial, junctional) |
| Ventricular |
| Pacemaker electrocardiography |
| Failure to capture |
| Failure to pace (no pacemaker output) |
| Failure to sense |
| Failure to capture both ventricles in biventricular pacing |

(Continued)

Table 8. Continued

| |
|---|
| Electrocardiographic abnormalities of acute myocardial ischemia |
| ST-segment elevation/depression |
| T-wave inversion |
| Muscle or other artifacts simulating arrhythmias |

AF indicates atrial fibrillation; BBB, bundle-branch block; TdP, torsade de pointes; VF, ventricular fibrillation; and VT, ventricular tachycardia.

*The content of electrocardiographic monitoring education needs to match the nature and complexity of the patient population served. Unit nursing leaders and educators are responsible for annually assessing the content of ongoing education on the basis of the electrocardiographic monitoring needs of patients in their care.

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teaching the appropriate response to an observed abnormality.^{39,481}

Education about electrocardiographic monitoring should be included in orientation and on an ongoing basis, with both didactic content and clinically based hands-on practice. Education should be matched to the complexity of the monitoring needs of the patient population served.^{39,447} However, no established standards related to content breadth or depth or duration of a course exist. Formal tests to determine knowledge and skill readiness are usually institution specific; they are rarely published with reports of validity or reliability. Goodridge et al⁴⁵⁹ described their pilot study that resulted in cessation of medical-surgical nurses providing electrocardiographic monitoring because of the low level of accuracy of interpretation of cardiac rhythms. Although some hospitals use unlicensed technicians to watch monitors, evidence related to the appropriate education and evaluation of their knowledge and skill is lacking.

Because education has an impact on cost and time, e-learning has been investigated in addition to face-to-face learning. Ongoing education was evaluated in the PULSE trial in which experienced nurses working on cardiac units completed an interactive online electrocardiographic monitoring education program that covered the essentials of electrocardiographic monitoring and arrhythmia, ischemia, and QT-interval monitoring. Mean scores on a 20-item validated online test were low at baseline (49.2% correct) but improved significantly to 70.2% correct after the intervention.⁶ Alternatively, investigators studying the best method to teach second-year medical students in their introductory class on electrocardiographic material randomized “near-peer” teaching (physicians in their second postgraduate year using a 1-hour PowerPoint presentation) and e-learning (including short multiple-choice assessments).⁴⁸² Although both groups increased knowledge, the near-peer group scored significantly higher (84%) than the e-learning group (74.5%). A face-to-face component may be important for students without prior knowledge of ECG, whereas e-learning may be appropriate for build-

Table 9. Education* Related to General Electrophysiological Concepts

| |
|---|
| Automaticity |
| Physiological 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 |
| Atrioventricular 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 |
| Sex 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 atrioventricular synchrony |
| Influence of left ventricular synchrony |
| Implantable devices |
| Function of electronic pacemakers, including biventricular pacemakers |
| Function of automatic defibrillators |
| Acute myocardial ischemia |
| ST-segment elevation (anterior, septal, inferior, lateral, right ventricular; ST recovery indicative of successful reperfusion; reperfusion arrhythmias; intermittent reperfusion; leads on the ECG related to occlusion of 3 main coronary arteries) |

(Continued)

Table 9. Continued

| |
|--|
| NSTEMI |
| Transient myocardial ischemia (including coronary spasm and effects of body position changes mimicking ischemia) |
| Syncope |
| Effects of common antiarrhythmic drugs, rate control vs rhythm control |

NSTEMI indicates non–ST-segment–elevation myocardial infarction.

*The content of electrocardiographic monitoring education needs to match the nature and complexity of the patient population served. Unit nursing leaders and educators are responsible for annually assessing the content of ongoing education on the basis of the electrocardiographic monitoring needs of patients in their care.

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ing on existing knowledge; however, further study is needed. Knowledge can be enhanced by collaborative activities such as preceptorships, skill validations, and case studies,^{16,481–486} and studies have demonstrated no differences in knowledge or confidence with face-to-face classes versus a hybrid approach (ie, online course combined with face-to-face classes).^{482,487,488}

Hospital, service line, or unit-based nurse educators or clinical nurse specialists can provide this education.^{485,487,488} In addition, the manufacturer of the monitors used should provide education related to the equipment. With the purchase of new monitoring equipment, the contract with the manufacturer should include both initial and ongoing education.

Recommendation

- 1. Initial and ongoing education for correct interpretation of electrocardiographic waveforms and data, congruent with type of patients being cared for, is recommended (Class I; Level of Evidence A).**

Documentation

Very little research has been published on documentation related to electrocardiographic monitoring, so the following section is based primarily on expert opinion and contemporary practice. Documentation of the ECG is critical for diagnosis and to guide subsequent treatment. All rhythms that require immediate attention should be preserved as actual tracings; written diagnostic statements alone are not adequate.¹ Documentation of actual electrocardiographic tracings so that they are immediately available is particularly important as hospitals have transitioned to electronic health records.

Appropriate and timely documentation of electrocardiographic waveforms is needed as a record of the rhythm and the clinical context. For example, as discussed in the syncope section, occurrence of symptoms with a documented arrhythmia on monitoring is considered a gold standard for the diagnosis of arrhythmia.

Table 10. Education* Related to Specific Monitoring Skills

| |
|--|
| Operation of monitoring system used in hospital unit (arrhythmia, ST-segment, and QTc monitoring) |
| Recognition of limitations of computer algorithms |
| Proper skin preparation before applying electrodes |
| Landmarks for and importance of accurate electrode placement |
| Setting heart rate, ST-segment alarm parameters appropriately |
| Importance and procedure for customizing alarm parameters to unit and patient needs |
| Measurement of heart rate |
| Measurement of intervals (use of manual and electronic electrocardiographic calipers) |
| Diagnosis of specific rhythms, recognition of atrial activity, evaluation of pauses |
| Recording of standard 12-lead ECG from monitor, including moving limb lead wires from electrodes on torso to electrodes on limbs |
| Recording of atrial electrograms from postoperative epicardial wires (including electrical safety) |
| Ability to intervene (unit protocols for responding to, reporting, and documenting) for: |
| Defibrillation/cardioversion |
| Patient with bradycardia |
| Patient with tachycardia |
| Patient with syncope |
| Patient with cardiac arrest |
| Patient with implanted device (new or chronic) |
| Patient with temporary transvenous pacemaker |
| Patient with transcutaneous pacemaker |

*The content of electrocardiographic monitoring education needs to match the nature and complexity of the patient population served. Unit nursing leaders and educators are responsible for annually assessing the content of ongoing education on the basis of the electrocardiographic monitoring needs of patients in their care.

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However, asymptomatic findings on monitoring, including asystole >3 seconds, Mobitz type 2 atrioventricular block, ventricular arrhythmias, or rapid supraventricular arrhythmias, when associated with syncope at other times, have also been considered diagnostic.²⁷¹ For some patients, an arrhythmia may occur only within the first few minutes of presenting to the ED. Furthermore, this information may provide vital documentation for insurance reimbursement if an implantable device is indicated. The patient's activity, symptoms, and blood pressure should be documented with the waveform strip, which will provide valuable information about a patient's tolerance of the arrhythmia and activity or will be used to guide medication adjustment or other intervention. If the documentation of electrocardiographic waveforms is done by a monitoring technician, it should be confirmed by a qualified nurse. Care must also be taken to verify that the lead identified on the waveform strip accurately reflects the true electrode configuration on

the patient's chest. This may facilitate differentiation of aberrant conduction from PVCs.⁷ If electrocardiographic monitoring is performed remotely, a system should be in place for routine verification of electrode placement.

Some monitors are equipped to monitor only 1 precordial lead at a time but allow the nurse to manually change the position of the precordial electrode on the chest to any of the 6 precordial leads. If this change is made, the nurse must document the new lead selected on the waveforms printed. Otherwise, it may appear that the patient had a sudden change from a negative (ie, V₁) to a positive (ie, V₆) QRS deflection. Correct documentation of the lead used in monitoring is vital to correct interpretation.

Typical expectations at hospitals include the following to be documented: rhythm, rate, PR interval, QRS duration, and QT, as well as whether they are within established parameters. Some hospital protocols expect QTc documentation for all patients. The writing group recommends that select patients receive QTc monitoring (Table 6). Documentation of a patient's waveform strip is typically expected on admission, on transfer to a monitored unit, and every 8 hours. Additional documentation of a waveform strip is typically expected for any significant change in the patient's rhythm or hemodynamic status, including before and after cardioversion.¹ The waveform strip should be accessible to all healthcare providers.

Thorough documentation for a significant arrhythmia includes events before and during the arrhythmia (eg, defibrillation, insertion of subclavian central line), signs and symptoms that may be related to the arrhythmia, vital signs, and interventions with patient response. This documentation must be available to all healthcare providers in a timely fashion so that a consulting cardiologist, for example, is able to access the electrocardiographic waveform as a piece of comprehensive assessment to guide the patient's care.

Additional documentation is important for patients with pacemakers and ICDs, including documentation of any improper sensing or failure to capture, as well as an estimated percentage of patient's reliance on atrial or ventricular pacing. It is important to have easy access to the patient's brand and type of device (pacemaker, ICD) and settings to be able to discern whether the device is functioning appropriately (eg, according to lower rate limits, antitachycardia pacing capability). Temporary pacemakers require more detailed documentation because adjustments to settings may be made from shift to shift. Documentation should include patient's skin at site of transcutaneous patches or wire exit sites.

As electronic health record systems evolve, methods to save waveforms will vary. Some will seamlessly integrate with electrocardiographic monitors. For these, the nurse will select an electronic waveform to be electronically transferred into the electronic health record. As

with any computer-generated diagnostic interpretation of an ECG, computer-generated labels of arrhythmias must be verified by the nurse. However, with many current electronic health record systems, personnel must print out waveform strips from the monitors and tape a paper copy into a paper chart in a format that can then be electronically scanned into patient medical records on a shift-to-shift basis. Similarly, portable monitor/defibrillator machines used in electric cardioversion or emergency situations print out paper electrocardiographic strips; it is critical that these strips be quickly and efficiently transferred onto a paper chart and scanned per protocol into an electronic health record. When a waveform strip is obtained for documentation, care should be taken to verify that voltage standardization and paper speed are visible on the strip.

Other considerations for documentation are dependent on the type of electrocardiographic monitor used. Many monitor systems have waveforms stored as full disclosure, meaning the clinician can view waveforms from several leads over the past several hours, allowing identification of when conversion from AF to sinus rhythm occurred, for example. The documentation of this event from full disclosure is helpful for evaluating the effects of an antiarrhythmic medication over time. Alternatively, documentation from full disclosure memory may be helpful for evaluating for ST-segment changes during a specific time period at night when a patient experienced chest pain but did not tell the nurse until morning. Finally, a 12-lead ECG obtained through a continuous electrocardiographic monitoring system should be clearly documented as such to avoid being mistaken for a standard 12-lead ECG obtained from a free-standing electrocardiographic machine.

Specific guidelines for frequency of ST-segment and QTc documentation have already been provided in this update to practice standards (Tables 3 and 6).

SECTION 4: IMPLEMENTATION OF PRACTICE STANDARDS

The discussion of implementation of practice standards into routine clinical care has 2 major aspects: the findings from past studies evaluating implementation of electrocardiographic monitoring practice standards and practical considerations for clinicians as they work to implement practice standards. Designs of future research studies that implement practice standards require thoughtful interprofessional collaboration to facilitate rapid translation of research into clinical practice.

Findings From Past Studies of Electrocardiographic Monitoring

The number of published evaluations of the original 1991 ACC/Emergency Cardiac Care Committee⁴⁸⁹ and

AHA¹ electrocardiographic monitoring standards has increased and has included QI projects,⁴⁹⁰ interventional research of electrocardiographic monitoring guidelines,^{56–59,449,491} and more recently, an RCT.⁶ These studies have addressed electrocardiographic monitoring for chest pain, association of practice standards with patient outcomes, practice standard use, and outcomes evaluated in studies of electrocardiographic monitoring.

Electrocardiographic Monitoring for Chest Pain

Early reports included overuse of electrocardiographic monitoring in the setting of chest pain in low-risk patients, resulting in a shortage of monitored beds and financial resources.⁴⁹² Several investigators reported that the use of validated risk stratification tools could identify which patients admitted for possible ACS could be considered low or very low risk; they advised that electrocardiographic monitoring was not necessary for these patients.^{106,492,493}

Association of Practice Standards With Patient Outcomes

Fåln et al⁵⁸ evaluated the appropriateness and outcomes of telemetry use according to the 2004 practice standards¹ by evaluating the frequency and type of arrhythmias in patients in each of the 3 CORs for monitoring. They found an overall arrhythmia rate of 33% (COR I, 43%; COR II, 28%; COR III, 47%). Change in management occurred in 25% of COR I patients, 14% of COR II patients, and 29% of COR III patients. These rates of change in care management were higher than the 7% to 8% reported in other studies.^{491,494} Fåln et al⁵⁸ recommended revision of the 2004 practice standards¹ on the basis of their finding that nearly half of COR III patients experienced arrhythmia events and one third of these events resulted in changes in care management. This may be related in part to the classification of patients with syncope in COR III per 2004 standards. The current writing group gives a COR I recommendation for arrhythmia monitoring for at least 24 hours for patients who, on the basis of presentation characteristics, are admitted for syncope with suspected cardiac origin. For those patients admitted for treatment of syncope with an identified cause, such pathogenesis should guide the use of arrhythmia monitoring.

Studies of the Use of Practice Standards for Electrocardiographic Monitoring

Among published reports (Table 11) of studies evaluating the use of practice standards for electrocardiographic monitoring,¹ the largest was the multisite PULSE trial.⁶ The PULSE trial was the only RCT and the only trial that evaluated continuous ST-segment and QTc monitoring. The PULSE trial revealed that appropriate use of electrocardiographic monitoring improved after the intervention of an interactive online education program and

Table 11. Published Reports of Use of Electrocardiographic Practice Standards

| Source | Design/Findings | Outcomes Measured |
|---|--|--|
| Kanwar et al ⁵⁶ (2008, United States) | Retrospective study to evaluate educational interventions to improve compliance with AHA monitoring standards. Charts of patients admitted to telemetry units were reviewed for 3 mo: before (n=972) and after (n=856) intervention. Education included lectures to ED and internal medicine residents and faculty, NPs, PAs, and unit clerks; included reminders via email, laminated cards, and posting. Unit clerks were encouraged to call to confirm telemetry indication. Appropriate monitoring percentage: A significant increase was noted for percent of patients with COR I/II indications for monitoring from before (57%) to after (71%) intervention, with a trend for shorter LOS (4.3±4 to 3.8±3 d). | Evaluated for indications for QTc or ST-segment monitoring: No. Safety: Not reported. |
| Dhillon et al ⁵⁷ (2009, United States) | Retrospective study of patients (n=562) admitted to the telemetry unit. Guidelines (based on the 1991 ACC/ECCC guidelines) were developed and implemented for telemetry admissions. Orders for telemetry expired after 48 h unless renewed. Nurses were encouraged to review need for telemetry daily and to confer with treating team. Significantly higher arrhythmia events were noted in the "telemetry indicated" group compared with the "telemetry not indicated" group. Appropriate monitoring percentage: Not reported. | Evaluated for indications for QTc or ST-segment monitoring: No. Safety: No patients in the telemetry not indicated group had a clinically significant arrhythmia requiring a change in management or further study. |
| Fåln et al ⁵⁸ (2013, Norway) | Prospective observational study of a 3-mo consecutive sample of all telemetry patient admissions with follow-up measurements at discharge from the hospital (n=1194). Arrhythmias that might result in a change in management were recorded; monitor watchers at a central monitoring station abstracted 64 variables that were reviewed along with medical records. The overall arrhythmia rate was 33%; of all events, 54% resulted in changes in care management. Patients were identified as COR I (18%), COR II (71%), and COR III (11%). These patients experienced arrhythmias at the following rates: COR I, 43%; COR II, 28%; and COR III, 47%. Changes in care management occurred as follows: COR I, 25%; COR II, 14%; and COR III, 29%. Nearly half of COR III patients experienced arrhythmia events, and one third of them resulted in changes to care management. Appropriate monitoring percentage: 89%. | Evaluated for indications for QTc or ST-segment monitoring: No. Safety: A large proportion of patients with chest pain were confirmed as having ACS at discharge, so although they were originally assigned as COR II, they would be upgraded to COR I. |
| Leighton et al ⁴⁹⁰ (2013, United States) | Prospective QI project of patients admitted to telemetry on non-ICU or cardiac ward units. Telemetry bed use was followed up 4 wk before and 4 wk after implementation of electronic order sets on the basis of AHA 2004 practice standards. Appropriate monitoring percentage (on admission to telemetry): Before (n=196): 65% of patients met guidelines. After (n=156): 81% of patients met guidelines; however, at 48 h after admission: 13% of patients met guidelines. | Evaluated for indications for QTc or ST-segment monitoring: No. Safety: No clinically significant arrhythmia events occurred among those without indications for monitoring. |
| Benjamin et al ⁴⁴⁹ (2013, United States) | Retrospective, descriptive, multisite study of 4 hospitals with non-ICU patients receiving electrocardiographic monitoring in which telemetry bed use was examined for 1 wk. In 35% of telemetry days, electrocardiographic monitoring was not supported by clinical indication for monitoring. Appropriate monitoring percentage: 65% of telemetry-indicated days. | Evaluated for indications for QTc or ST-segment monitoring: No. Safety: 3.1 arrhythmias per 100 d. Cost: Estimated \$53–\$88.4 per patient per day or \$250 000/y savings for 400-bed hospital (based on estimated 15–30 min of nursing time per patient per shift=45–90 min/d). |
| Dressler et al ⁵⁹ (2014, United States) | Prospective study of revised telemetry order sets based on AHA practice standards (2004) in which prescribers were required to select from a list of clinical indications, each with a predetermined duration of monitoring. Order sets included nurse assessment guidelines; nurses could request reorder of telemetry if patient was believed to be unsafe (eg, unstable blood pressure). Mean daily number of patients with telemetry ordered decreased by 70%. | Evaluated for indications for QTc or ST-segment monitoring: None reported. |

(Continued)

Table 11. Continued

| Source | Design/Findings | Outcomes Measured |
|--|---|--|
| Dressler et al ⁵⁹ (2014, United States) (Continued) | Appropriate monitoring percentage: not reported. | Safety: Code blue, mortality, and rapid response team activation rates remained stable. Cost: Estimated mean daily cost to deliver telemetry was \$53.44 per patient; mean daily cost for non-ICU cardiac telemetry decreased from \$18 971 to \$5772 on the basis of estimated 19.75 min per patient on telemetry-related tasks daily. |
| Funk et al ⁶ (2017, United States) | Prospective, multisite RCT in 17 hospitals with all patients in cardiac ICUs and telemetry units (n=4587 patients; n=3013 nurses). Medical records of current patients were reviewed to determine whether they had a COR I or II indication for arrhythmia, ST-segment ischemia, and QTc interval monitoring and noted if the patient was being monitored. Patient outcomes were assessed in 95 884 admissions. An online interactive education intervention for nurses was supplemented by strategies to change practice led by nurse champions, including initiation by some hospitals of protocols for QTc and ST-segment monitoring according to the 2004 practice standards. Appropriate monitoring percentage: Appropriate monitoring increased significantly immediately after the intervention and was sustained for 15 mo (66% at baseline to 70% immediately after the intervention and 70% 15 mo later). Appropriate ST-segment monitoring went from 56% at baseline to 67% immediately after the intervention to 75% 15 mo later. Appropriate QTc monitoring was 13% at baseline and 51% immediately after the intervention but dropped to 45% 15 mo later. | Evaluated for indications for QTc or ST-segment monitoring: Yes. Safety: In-hospital MI declined significantly immediately after the intervention and was sustained 15 mo later (2.4% to 1.9% to 1.5%). Mortality ranged from 1.9%–3.1% and was not associated with the intervention. |

ACC/ECCC indicates American College of Cardiology/Emergency Cardiac Care Committee; ACS, acute coronary syndrome; AHA, American Heart Association; COR, Class of Recommendation; ED, emergency department; ICU, intensive care unit; LOS, length of stay; NP, nurse practitioner; PA, physician's assistant; QI, quality improvement; and RCT, randomized clinical trial.

strategies to implement and sustain change in practice, and this improvement was sustained over time.⁶

Outcomes Evaluated in Studies of Electrocardiographic Monitoring

Researchers evaluating preimplementation to postimplementation of practice standards have some variability in their selection of outcome measures. Investigators have used the number of rapid response calls as an outcome variable, but the number of calls alone is difficult to interpret without contextual data. Reasons for the call may be unrelated to electrocardiographic monitoring (eg, dyspnea, decreased cognition). Alternatively, a rapid response call may be made because of a syncope episode that could potentially have been averted if a patient had been monitored. A patient undergoing ischemia monitoring may have a nurse who calls rapid response more quickly as a result of identification of ST-segment changes with silent ischemia. Thus, the number of rapid response calls could be unrelated to electrocardiographic monitoring or could increase or decrease as a result of appropriate monitoring.

Financial outcomes require thoughtful consideration. Dressler et al⁵⁹ reported a 70% decrease in inappropriate monitoring, with a resultant estimated cost savings of \$4.8 million annually. Although a cost reduction of this magnitude was impressive for this site, it may not be appropriate to generalize to other hospitals. The amount of caregiver time for the management of patients receiving electrocardiographic monitoring has

never been rigorously quantified, so it is challenging to provide accurate financial data. Leighton et al⁴⁹⁰ asked nurse managers to estimate the amount of time and supplies for monitoring, resulting in an estimated 15 to 30 minutes per patient per 8-hour shift. It may be helpful to avoid reporting only aggregate data for the calculation of financial outcomes to permit more accurate comparison across institutions. Although cost savings have been reported, one must consider that a reduction in monitored beds could potentially reduce revenue for monitored beds while simultaneously increasing admission to nonmonitored beds.

Finally, associations of the implementation of electrocardiographic monitoring practice standards with patient outcomes such as mortality, in-hospital MI, and outcomes of cardiac arrest have been reported (Table 11). Thoughtful examination of published studies reporting implementation of electrocardiographic monitoring standards allows investigators to design robust experimental studies. These studies must be adequately powered to enable detection of rare patient outcomes such as mortality and TdP.

Practical Considerations to Implementing Practice Standards

When implementing electrocardiographic monitoring practice standards, the clinician needs to consider a number of issues related to arrhythmia, ST-segment,

and QTc monitoring when developing new protocols as electronic health records become more common.

Arrhythmia: Practice Standards Embedded Into Electronic Health Record Order Sets

Dressler et al⁵⁹ reported that education on practice standards alone was not enough to bring about increased adherence. Instead, interprofessional education needed to be augmented with new electronic order sets for prescribers and institution-wide guidelines for electrocardiographic monitoring⁵⁹ based on the practice standards.¹ This practice is increasingly common.⁴⁹⁰

ST-Segment Monitoring: In-Hospital Protocols to Promote Awareness, Education, and Use of Standards

Through a national survey of cardiologists published in 2010, Sandau et al⁴⁹⁵ found that only 45% of practicing cardiologists (n=200) were aware of ST-segment monitoring practice standards, despite publications in 1999¹⁴ and 2004.¹ The cardiologists surveyed selected top barriers to use of this technology as concerns about false-positive alarms, lack of understanding by nurses, concern for extra telephone calls resulting from inaccurate monitoring, lack of understanding by other physicians, and concern for extra cost or treatment caused by false-positive alarms. In a single-site study, Sangkachand et al⁴⁸¹ identified top barriers for nurses' use of continuous ST-segment monitoring as similar to those of cardiologists, with an additional barrier that prescribers did not order it. Clinicians should share experience with implementation of protocols³⁹; identifying effective strategies can enhance successful implementation at other sites.

QTc Monitoring: Electronic Alerts, Education, Protocol, and Computerized Calculation

Investigators have recently used both alerts by pharmacists and computerized alerts to identify hospitalized patients receiving QT-prolonging medications. Ng et al⁴⁹⁶ evaluated a pharmacist-led intervention (n=149 patients), resulting in patients randomized to the intervention of pharmacist involvement and a specific QTc algorithm having less frequent QTc prolongation. Haugaa et al⁴⁹⁷ evaluated an institution-wide (n=1145) QT alert system that identified ECGs with QTc ≥ 0.50 seconds, with a "semi-urgent" e-mail alert to the prescriber. Tisdale et al⁴⁹⁸ evaluated a computerized clinical decision support system incorporating a validated risk score for QTc prolongation to alert pharmacists when a QT-prolonging cardiac medication was prescribed for cardiac unit inpatients (n=2400) and found a significantly reduced risk of QTc prolongation and decreased prescribing of noncardiac medications known to cause TdP, including fluoroquinolones and intravenous haloperidol.⁴⁹⁸

In a quasi-experimental study of 4011 patients receiving electrocardiographic monitoring in 10 hospitals, Sandau and colleagues⁴⁶⁴ tested a 3-part intervention

of online education for nurses, electronic notifications to alert nurses when a patient received QT-prolonging medication, and computerized calculation of QTc in the electronic health record. They found that appropriate QTc documentation increased significantly from baseline (17.3%) to 3 months after intervention (58.2%), with a further increase by 6 months after intervention (62.1%), demonstrating that improvements persisted over time.

Conclusions

Electrocardiographic monitoring offers a number of clinical benefits that are rarely evaluated in implementation studies. First, electrocardiographic monitoring is used to assess the response to medications for arrhythmias. Titration of the dose of medications such as diltiazem, β -blockers, and dofetilide based on electrocardiographic monitoring provides data for communication among care providers (eg, prescriber, nurse, pharmacist, exercise rehabilitation professional), allowing evaluation of heart rate changes with exercise or sleep. Individualized dosing of QT-prolonging medications relies on monitoring QTc, which is usually not captured in implementation studies.

The absence of abnormal findings on telemetry has clinical relevance. For example, telemetry may be helpful for the patient admitted with syncope with a suspected cardiac cause. It is typical that if no electrocardiographic abnormalities are found, the patient may be transferred to a neurological unit without electrocardiographic monitoring. Electrocardiographic monitoring in the immediate post-pacemaker/ICD implantation period is used to confirm the absence of undersensing or oversensing of a device. Finally, the absence of ST-segment changes may be helpful to verify reperfusion after PCI and can be used to help distinguish between a chest ache that is sometimes reported after stent manipulation and signs of reocclusion.¹ Although clinicians must be mindful of the appropriate duration of monitoring, the purpose and benefit of a brief prescribed period of monitoring to confirm the absence of abnormal findings should not be overlooked. It is important that careful consideration be given to the type of data that may be useful to assess for relevant changes⁵⁸ and for interprofessional collaboration in the design of any implementation protocols for ECG.⁵⁶

SECTION 5: CALL FOR RESEARCH

The majority of recent studies reporting adherence to practice standards have 2 main limitations: lack of evaluation for appropriate QTc monitoring and lack of evaluation for appropriate continuous ST-segment monitoring. Researchers who evaluate only the indication for arrhythmia monitoring miss the opportunity to examine whether patients are also receiving QTc monitoring while on QT-

Table 12. Future Research Directions for Electrocardiographic Monitoring

| Patient Population | Future Research Directions |
|--|---|
| Chest pain and ACS | What type of monitoring system and alarm management approaches result in accurate identification of ischemia with the fewest false and nonactionable alarms? |
| | Does continuous ST-segment monitoring result in earlier identification of ischemia for patients deemed at high risk in the ACS continuum? |
| | Does continuous ST-segment monitoring result in earlier identification of ischemia for patients with impaired ability to sense or communicate ischemia (eg, those with diabetes mellitus or communication or cognitive barriers)? |
| | What is the effect of targeted temperature management after cardiac arrest on the ST-segment and QTc? |
| Major cardiac interventions | What are the expected ST-segment changes after open heart surgery (eg, restoration of normal ST segment postoperatively)? |
| | What is the optimal duration of monitoring for adult and pediatric patients after cardiac surgery? |
| | Does continuous ST-segment monitoring result in earlier identification of ischemia for patients after heart transplantation because of impaired sensory discrimination? |
| Arrhythmias | Does prospective QTc monitoring for patients at risk result in decreased TdP or mortality? |
| Syncope of unknown origin | What risk stratification strategies are helpful in identifying which patients with syncope benefit from continuous electrocardiographic monitoring? |
| After electrophysiology procedures and pacemaker/ICD implantation | What is the timing of minor and major complications after an electrophysiology procedure and pacemaker/ICD implantation that require arrhythmia monitoring? |
| | How can monitor manufacturers enhance the visibility of pacemaker spikes on telemetry? |
| ADHF | What risk stratification strategies are helpful in identifying which patients with ADHF benefit from continuous electrocardiographic monitoring? |
| Stroke | Does the risk of serious arrhythmia vary by type of stroke (ie, embolic, hemorrhagic)? |
| | Is the risk window for rapid intervention (ie, 24 h) the same for patients who receive thrombolytics and patients who do not? |
| Noncardiac medical conditions (eg, hemodialysis, sepsis, gastrointestinal bleed; postconscious sedation) | In what subgroup of noncardiac patients is electrocardiographic monitoring associated with improved outcomes? |
| | What alternative surveillance monitoring methods (oximetry, capnography) provide clinical benefit for noncardiac patients outside the ICU? |
| Electrolyte abnormalities | What electrolyte abnormalities are serious enough to warrant electrocardiographic monitoring for adults and children? |
| Pediatric | What changes in alarm criteria can be used to safely limit false-positive alarms? |
| | How should automated algorithms be validated across age groups? |
| | Can wireless leads provide cost-effective, quality monitoring (eg, patches as transmitters) for pediatric and adult patients? |

(Continued)

Table 12. Continued

| Patient Population | Future Research Directions |
|---|--|
| Alarm management | What is the best way to increase the specificity of alarms without a significant loss of sensitivity? |
| | What is the best geographic setup for a unit with no dedicated monitor watchers? |
| | What is the effect of the use of dedicated monitor watchers on quality of care, patient outcomes, and cost? |
| | What are the best systems to notify direct caregivers of alarms? |
| | Should patients in noncardiac units (eg, orthopedic) be monitored from a centralized base for electrocardiographic monitoring for staff nurses on noncardiac units who are providing direct care? |
| | What is the effect of altering alarm parameters on adverse patient outcomes? |
| | What are the most effective alarm sounds to improve alarm response? |
| | What is the best method to ensure alarm audibility/secondary alarm notification? |
| Is the use of multiparameter alarm algorithms effective in reducing false alarms? | |
| | |
| Education of physicians, NPs, PAs, and staff nurses | What education is necessary for initial competency and subsequent maintenance of skills for electrocardiographic monitor interpretation? |
| Implementation of practice standards | What are the most collaborative, efficient, and cost-effective methods for incorporating practice standards for electrocardiographic monitoring into electronic order sets? Who should review for discontinuation of telemetry? How often should orders be reviewed, and what criteria should be used? |
| | What is the impact of implementation of these practice standards for electrocardiographic monitoring on patient outcomes, alarms, cost, and bed use? |

ACS indicates acute coronary syndrome; ADHF, acute decompensated heart failure; ICD, implantable cardioverter-defibrillator; ICU, intensive care unit; NP, nurse practitioner; PA, physician's assistant; and TdP, torsade de pointes.

prolonging medications. Researchers wanting to assess for appropriate ST-segment monitoring face a particular challenge in study design for 2 main reasons. First, some hospitals have not universally incorporated this practice and related interprofessional education. Second, retrospective designs are generally unable to capture whether continuous ST-segment monitoring was used because use of continuous ST-segment monitoring is not typically documented on either paper waveform strips or any drop-down menus in an electronic health record.

Throughout this statement, an attempt has been made to highlight areas where there is limited evidence on which to base clinical practice guidelines. Table 12 is provided to highlight areas in particular need of targeted studies. The considerable time, education, and cost associated with continuous electrocardiographic monitoring is a call to action for the research needed to ensure that we are providing the best-quality monitoring for those who truly benefit from this diagnostic intervention.

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FOOTNOTES

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*Modest.

†Significant.

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*Modest.

†Significant.

REFERENCES

- Drew BJ, Califf RM, Funk M, Kaufman ES, Krucoff MW, Laks MM, Macfarlane PW, Sommargren C, Swiryn S, Van Hare GF, American Heart Association, Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young. 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 [published correction appears in *Circulation*. 2005;111:378]. *Circulation*. 2004;110:2721–2746.
- ACCF/AHA Task Force on Practice Guidelines. Methodologies and policies in the ACCF/AHA Task Force on Practice Guidelines. https://my.americanheart.org/idc/groups/ahamah-public/@wcm/@sop/documents/downloadable/ucm_319826.pdf. Accessed September 3, 2015.
- Chan PS, Krumholz HM, Nichol G, Nallamothu BK; American Heart Association National Registry of Cardiopulmonary Resuscitation Investigators. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med*. 2008;358:9–17. doi: 10.1056/NEJMoa0706467.
- Chen LM, Nallamothu BK, Spertus JA, Li Y, Chan PS; American Heart Association's Get With the Guidelines-Resuscitation (formerly the National Registry of Cardiopulmonary Resuscitation) Investigators. Association between a hospital's rate of cardiac arrest incidence and cardiac arrest survival. *JAMA Intern Med*. 2013;173:1186–1195. doi: 10.1001/jamainternmed.2013.1026.
- Knight BP, Pelosi F, Michaud GF, Strickberger SA, Morady F. Clinical consequences of electrocardiographic artifact mimicking ventricular tachycardia. *N Engl J Med*. 1999;341:1270–1274. doi: 10.1056/NEJM199910213411704.
- Funk M, Fennie KP, Stephens KE, May JL, Winkler CG, Drew BJ, The PULSE Site Investigators. Association of implementation of practice standards for electrocardiographic monitoring with nurses' knowledge, quality of care, and patient outcomes: findings from the Practical Use of the Latest Standards of Electrocardiography (PULSE) Trial. *Circ Cardiovasc Qual Outcomes*. 2017;10:e003132.
- Alzand BS, Crijns HJ. Diagnostic criteria of broad QRS complex tachycardia: decades of evolution. *Europace*. 2011;13:465–472. doi: 10.1093/europace/euq430.
- Geggel RL. Conditions leading to pediatric cardiology consultation in a tertiary academic hospital. *Pediatrics*. 2004;114:e409–e417.
- Rijnbeek PR, Witsenburg M, Schrama E, Hess J, Kors JA. New normal limits for the paediatric electrocardiogram. *Eur Heart J*. 2001;22:702–711. doi: 10.1053/euhj.2000.2399.
- Nadkarni VM, Larkin GL, Peberdy MA, Carey SM, Kaye W, Mancini ME, Nichol G, Lane-Truitt T, Potts J, Ornato JP, Berg RA; National Registry of Cardiopulmonary Resuscitation Investigators. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. *JAMA*. 2006;295:50–57. doi: 10.1001/jama.295.1.50.
- Thygesen K, Alpert JS, White HD; on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction, Jaffe AS, Apple FS, Galvani M, Katus HA, Newby LK, Ravkilde J, Chaitman B, Clemmensen PM, Dellborg M, Hod H, Porela P, Underwood R, Bax JJ, Beller GA, Bonow R, Van der Wall EE, Bassand JP, Wijns W, Ferguson TB, Steg PG, Uretsky BF, Williams DO, Armstrong PW, Antman EM, Fox KA, Hamm CW, Ohman EM, Simoons ML, Poole-Wilson PA, Gurfinkel EP, Lopez-Sendon JL, Pais P, Mendis S, Zhu JR, Wallentin LC, Fernández-Avilés F, Fox KM, Parkhomenko AN, Puri SG, Tendera M, Voipio-Pulkki LM, Vahanian A, Camm AJ, De Caterina R, Dean V, Dickstein K, Filippatos G, Funck-Brentano C, Hellemans I, Kristensen SD, McGregor K, Sechtem U, Silber S, Tendera M, Widimsky P, Zamorano JL, Morais J, Brener S, Harrington R, Morrow D, Lim M, Martinez-Rios MA, Steinhubl S, Levine GN, Gibler WB, Goff D, Tubaro M, Dudek D, Al-Attar N. Universal definition of myocardial infarction. *Circulation*. 2007;116:2634–2653. doi: 10.1161/CIRCULATIONAHA.107.187397.
- Cohn PF, Fox KM, Daly C. Silent myocardial ischemia. *Circulation*. 2003;108:1263–1277. doi: 10.1161/01.CIR.0000088001.59265.EE.
- Conti CR, Bavry AA, Petersen JW. Silent ischemia: clinical relevance. *J Am Coll Cardiol*. 2012;59:435–441. doi: 10.1016/j.jacc.2011.07.050.
- Drew BJ, Krucoff MW. Multilead ST-segment monitoring in patients with acute coronary syndromes: a consensus statement for healthcare professionals: ST-Segment Monitoring Practice Guideline International Working Group. *Am J Crit Care*. 1999;8:372–386.
- Drew BJ, Tisdale LA. ST segment monitoring for coronary artery reocclusion following thrombolytic therapy and coronary angioplasty: identification of optimal bedside monitoring leads. *Am J Crit Care*. 1993;2:280–292.
- Sandau KE, Smith M. Continuous ST-segment monitoring: 3 case studies in progressive care. *Crit Care Nurse*. 2009;29:18–27; quiz 1 p following 27. doi: 10.4037/ccn2009953.
- Sangkachand P, Cluff M, Funk M. Detecting myocardial ischemia with continuous ST-segment monitoring: two case studies. *Heart Lung*. 2012;41:284–289. doi: 10.1016/j.hrtng.2011.10.007.
- Drew BJ, Pelter MM, Adams MG. Frequency, characteristics, and clinical significance of transient ST segment elevation in patients with acute coronary syndromes. *Eur Heart J*. 2002;23:941–947. doi: 10.1053/euhj.2001.2987.
- Booker KJ, Holm K, Drew BJ, Lanuza DM, Hicks FD, Carrigan T, Wright M, Moran J. Frequency and outcomes of transient myocardial ischemia in critically ill adults admitted for noncardiac conditions. *Am J Crit Care*. 2003;12:508–516.
- Krucoff MW, Parente AR, Bottner RK, Renzi RH, Stark KS, Shugoll RA, Ahmed SW, DeMichele J, Stroming SL, Green CE, Rackley CE, Kent KM. Stability of multilead ST-segment “fingerprints” over time after percutaneous transluminal coronary angioplasty and its usefulness in detecting reocclusion. *Am J Cardiol*. 1988;61:1232–1237.
- Langer A, Krucoff MW, Klootwijk P, Simoons ML, Granger CB, Barr A, Califf RM, Armstrong PW. Prognostic significance of ST segment shift early after resolution of ST elevation in patients with myocardial infarction treated with thrombolytic therapy: the GUSTO-I ST Segment Monitoring Substudy. *J Am Coll Cardiol*. 1998;31:783–789.
- Maas AC, Wyatt CM, Green CL, Wagner GS, Trollinger KM, Pope JE, Langer A, Armstrong PW, Califf RM, Simoons ML, Krucoff MW. Combining baseline clinical descriptors and real-time response to therapy: the incremental prognostic value of continuous ST-segment monitoring in acute myocardial infarction. *Am Heart J*. 2004;147:698–704. doi: 10.1016/j.ahj.2003.08.014.
- Cruden NL, Graham C, Harding SA, Ludlam CA, Fox KA, Newby DE. Plasma TAFI and soluble CD40 ligand do not predict reperfusion following thrombolysis for acute myocardial infarction. *Thromb Res*. 2006;118:189–197. doi: 10.1016/j.thromres.2005.06.014.
- Terkelsen CJ, Nørgaard BL, Lassen JF, Poulsen SH, Gerdes JC, Sloth E, Gøtzsche LB, Rømer FK, Thuesen L, Nielsen TT, Andersen HR. Potential significance of spontaneous and interventional ST-changes in patients transferred for primary percutaneous coronary intervention: observations from the ST-MONITORING in Acute Myocardial Infarction study (the MONAMI study). *Eur Heart J*. 2006;27:267–275. doi: 10.1093/eurheartj/ehi606.
- Jernberg T, Abrahamsson P, Lindahl B, Johanson P, Wallentin L, Dellborg M. Continuous multilead ST-monitoring identifies patients with unstable coronary artery disease who benefit from extended antithrombotic treatment. *Eur Heart J*. 2002;23:1093–1101.
- Klootwijk P, Meij S, Melkert R, Lenderink T, Simoons ML. Reduction of recurrent ischemia with abciximab during continuous ECG-ischemia monitoring in patients with unstable angina refractory to standard treatment (CAPTURE). *Circulation*. 1998;98:1358–1364.
- Stefanidis A, Melidonis A, Tournis S, Zairis M, Handanis S, Olympios C, Asimacopoulos P, Foussas S. Intensive insulin treatment reduces transient ischaemic episodes during acute coronary events in diabetic patients. *Acta Cardiol*. 2002;57:357–364. doi: 10.2143/AC.57.5.2005453.
- Jernberg T, Lindahl B, Wallentin L. ST-segment monitoring with continuous 12-lead ECG improves early risk stratification in patients with chest pain and ECG nondiagnostic of acute myocardial infarction. *J Am Coll Cardiol*. 1999;34:1413–1419.
- Herren KR, Mackway-Jones K, Richards CR, Seneviratne CJ, France MW, Cotter L. Is it possible to exclude a diagnosis of myocardial damage within six hours of admission to an emergency department? Diagnostic cohort study. *BMJ*. 2001;323:372.
- Pelter MM, Adams MG, Drew BJ. Transient myocardial ischemia is an independent predictor of adverse in-hospital outcomes in patients with acute coronary syndromes treated in the telemetry unit. *Heart Lung*. 2003;32:71–78. doi: 10.1067/mhl.2003.11.
- Landesberg G, Vesselov Y, Einav S, Goodman S, Sprung CL, Weissman C. Myocardial ischemia, cardiac troponin, and long-term survival of high-cardiac risk critically ill intensive care unit patients. *Crit Care Med*. 2005;33:1281–1287.
- Yan AT, Yan RT, Tan M, Senaratne M, Fitchett DH, Langer A, Goodman SG; INTERACT Investigators. Long-term prognostic value and therapeutic implications of continuous ST-segment monitoring in acute coronary syndrome. *Am Heart J*. 2007;153:500–506. doi: 10.1016/j.ahj.2007.02.003.
- Ottander P, Nilsson JB, Jensen SM, Näslund U. Ischemic ST-segment episodes during the initial 24 hours of ST elevation myocardial infarction pre-

- dict prognosis at 1 and 5 years. *J Electrocardiol.* 2010;43:224–229. doi: 10.1016/j.jelectrocard.2009.12.010.
34. Akkerhuis KM, Klootwijk PA, Lindeboom W, Umans VA, Meij S, Kint PP, Simoons ML. Recurrent ischaemia during continuous multilead ST-segment monitoring identifies patients with acute coronary syndromes at high risk of adverse cardiac events; meta-analysis of three studies involving 995 patients. *Eur Heart J.* 2001;22:1997–2006. doi: 10.1053/euhj.2001.2750.
 35. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD; on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction. Third universal definition of myocardial infarction. *Circulation.* 2012;126:2020–2035. doi: 10.1161/CIR.0b013e31826e1058.
 36. Jernberg T, Cronblad J, Lindahl B, Wallentin L. Observer variability and optimal criteria of transient ischemia during ST monitoring with continuous 12-lead ECG. *Ann Noninvasive Electrocardiol.* 2002;7:181–190.
 37. Adams MG, Drew BJ. Body position effects on the ECG: implication for ischemia monitoring. *J Electrocardiol.* 1997;30:285–291.
 38. Drew BJ, Adams MG. Clinical consequences of ST-segment changes caused by body position mimicking transient myocardial ischemia: hazards of ST-segment monitoring? *J Electrocardiol.* 2001;34:261–264.
 39. Sandau KE, Smith M. Continuous ST-segment monitoring: protocol for practice. *Crit Care Nurse.* 2009;29:39–49; quiz following 50. doi: 10.4037/ccn2009703.
 40. Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, Kors JA, Macfarlane P, Mason JW, Mirvis DM, Okin P, Pahlm O, van Herpen G, Wagner GS, Wellens H. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram, part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. *Circulation.* 2009;119:e241–e250. doi: 10.1161/CIRCULATIONAHA.108.191096.
 41. Goldberger AL, ed. *Clinical Electrocardiography: A Simplified Approach.* 7th ed. Philadelphia, PA: Mosby Elsevier; 2006.
 42. Haerberlin A, Studer E, Niederhauser T, Stoller M, Marisa T, Goette J, Jacommet M, Traupe T, Seiler C, Vogel R. Electrocardiographic ST-segment monitoring during controlled occlusion of coronary arteries. *J Electrocardiol.* 2014;47:29–37. doi: 10.1016/j.jelectrocard.2013.10.003.
 43. Pollak P, Brady W. Electrocardiographic patterns mimicking ST segment elevation myocardial infarction. *Cardiol Clin.* 2012;30:601–615. doi: 10.1016/j.ccl.2012.07.012.
 44. Antzelevitch C, Yan GX. J wave syndromes. *Heart Rhythm.* 2010;7:549–558. doi: 10.1016/j.hrthm.2009.12.006.
 45. Noda T, Shimizu W, Tanaka K, Chayama K. Prominent J wave and ST segment elevation: serial electrocardiographic changes in accidental hypothermia. *J Cardiovasc Electrophysiol.* 2003;14:223.
 46. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome: a multicenter report. *J Am Coll Cardiol.* 1992;20:1391–1396.
 47. Graham KC, Cvach M. Monitor alarm fatigue: standardizing use of physiologic monitors and decreasing nuisance alarms. *Am J Crit Care.* 2010;19:28–34; quiz 35. doi: 10.4037/ajcc2010651.
 48. Physiologic monitoring systems. *Health Devices.* 2013;42:310–340.
 49. The Joint Commission. The Joint Commission Sentinel Event Alert Issue 50: Medical device alarm safety in hospitals. http://www.jointcommission.org/sea_issue_50/. Accessed July 18, 2016.
 50. Drew BJ, Harris P, Zègre-Hemsey JK, Mammone T, Schindler D, Salas-Boni R, Bai Y, Tinoco A, Ding Q, Hu X. Insights into the problem of alarm fatigue with physiologic monitor devices: a comprehensive observational study of consecutive intensive care unit patients. *PLoS One.* 2014;9:e110274. doi: 10.1371/journal.pone.0110274.
 51. Top 10 health technology hazards for 2015. *OR Manager.* 2015;31:15–17.
 52. ECRI Institute. 2016 Top 10 health technology hazards: What technology hazards are lurking in your hospital? <http://www.ecri.org/2016hazards>. Accessed December 10, 2015.
 53. Honan L, Funk M, Maynard M, Fahs D, Clark JT, David Y. Nurses' perspectives on clinical alarms. *Am J Crit Care.* 2015;24:387–395. doi: 10.4037/ajcc2015552.
 54. Weil KM. Alarming monitor problems. *Nursing.* 2009;39:58. doi: 10.1097/01.NURSE.0000360252.10823.b8.
 55. Bliss JP, Gilson RD, Deaton JE. Human probability matching behaviour in response to alarms of varying reliability. *Ergonomics.* 1995;38:2300–2312. doi: 10.1080/00140139508925269.
 56. Kanwar M, Fares R, Minnick S, Rosman HS, Saravolatz L. Inpatient cardiac telemetry monitoring: are we overdoing it? *JCOM.* 2008;15:16–20.
 57. Dhillon SK, Rachko M, Hanon S, Schweitzer P, Bergmann SR. Telemetry monitoring guidelines for efficient and safe delivery of cardiac rhythm monitoring to noncritical hospital inpatients. *Crit Pathw Cardiol.* 2009;8:125–126. doi: 10.1097/HPC.0b013e3181b6ef41.
 58. Fållun N, Nordrehaug JE, Hoff PI, Langørgen J, Moons P, Norekvål TM. Evaluation of the appropriateness and outcome of in-hospital telemetry monitoring. *Am J Cardiol.* 2013;112:1219–1223. doi: 10.1016/j.amjcard.2013.05.069.
 59. Dressler R, Dryer MM, Coletti C, Mahoney D, Doorey AJ. Altering overuse of cardiac telemetry in non-intensive care unit settings by hardwiring the use of American Heart Association guidelines. *JAMA Intern Med.* 2014;174:1852–1854. doi: 10.1001/jamainternmed.2014.4491.
 60. Loreto L, Andrea T, Lucia D, Carla L, Cristina P, Silvio R. Accuracy of EASI 12-lead ECGs in monitoring ST-segment and J-point by nurses in the coronary care units. *J Clin Nurs.* 2016;25:1282–1291. doi: 10.1111/jocn.13168.
 61. Hillis LD, Smith PK, Anderson JL, Bittl JA, Bridges CR, Byrne JG, Cigarroa JE, Disesa VJ, Hiratzka LF, Hutter AM Jr, Jessen ME, Keeley EC, Lahey SJ, Lange RA, London MJ, Mack MJ, Patel MR, Puskas JD, Sabik JF, Selnes O, Shahian DM, Trost JC, Winniford MD. 2011 ACCF/AHA guideline for coronary artery bypass graft surgery: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines [published correction appears in *Circulation.* 2011;124:e652–e735]. doi: 10.1161/CIR.0b013e31823c074e.
 62. Schwartz PJ, Garson A Jr, Paul T, Stramba-Badiale M, Vetter VL, Wren C; European Society of Cardiology. Guidelines for the interpretation of the neonatal electrocardiogram: a task force of the European Society of Cardiology. *Eur Heart J.* 2002;23:1329–1344.
 63. McCulley ME, Bennett RL. ST segment monitoring in the pediatric ICU: detecting myocardial ischemia in children. *Crit Care Nurse.* 1997;17:81–86, 88–92.
 64. Rydberg A, Rask P, Teien DE, Hörnsten R. Electrocardiographic ST segment depression and clinical function in children with Fontan circulation. *Pediatr Cardiol.* 2003;24:468–472. doi: 10.1007/s00246-002-0374-2.
 65. Newburger JW, Takahashi M, Gerber MA, Gewitz MH, Tani LY, Burns JC, Shulman ST, Bolger AF, Ferrieri P, Baltimore RS, Wilson WR, Baddour LM, Levison ME, Pallasch TJ, Falace DA, Taubert KA. Diagnosis, treatment, and long-term management of Kawasaki disease: a statement for health professionals from the Committee on Rheumatic Fever, Endocarditis and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation.* 2004;110:2747–2771. doi: 10.1161/01.CIR.0000145143.19711.78.
 66. Uehara R, Belay ED. Epidemiology of Kawasaki disease in Asia, Europe, and the United States. *J Epidemiol.* 2012;22:79–85.
 67. Tatara K, Kusakawa S, Itoh K, Kazuma N, Lee K, Hashimoto K, Shinohara T, Kondoh C, Hiroe M. Long-term prognosis of Kawasaki disease patients with coronary artery obstruction. *Heart Vessels.* 1989;5:47–51.
 68. Mehta SK, Finkelhor RS, Anderson RL, Harcar-Sevcik RA, Wasser TE, Bahler RC. Transient myocardial ischemia in infants prenatally exposed to cocaine. *J Pediatr.* 1993;122:945–949.
 69. Maguire JF, O'Rourke PP, Colan SD, Geha RS, Crone R. Cardiotoxicity during treatment of severe childhood asthma. *Pediatrics.* 1991;88:1180–1186.
 70. Yang P, Kanki H, Drolet B, Yang T, Wei J, Viswanathan PC, Hohnloser SH, Shimizu W, Schwartz PJ, Stanton M, Murray KT, Norris K, George AL Jr, Roden DM. Allelic variants in long-QT disease genes in patients with drug-associated torsades de pointes. *Circulation.* 2002;105:1943–1948.
 71. Lehtonen A, Fodstad H, Laitinen-Forsblom P, Toivonen L, Kontula K, Swan H. Further evidence of inherited long QT syndrome gene mutations in antiarrhythmic drug-associated torsades de pointes. *Heart Rhythm.* 2007;4:603–607. doi: 10.1016/j.hrthm.2007.01.019.
 72. Paulussen AD, Gilissen RA, Armstrong M, Doevendans PA, Verhasselt P, Smeets HJ, Schulze-Bahr E, Haverkamp W, Breithardt G, Cohen N, Aerssens J. Genetic variations of KCNQ1, KCNH2, SCN5A, KCNE1, and KCNE2 in drug-induced long QT syndrome patients. *J Mol Med (Berl).* 2004;82:182–188. doi: 10.1007/s00109-003-0522-z.
 73. Pickham D, Helfenbein E, Shinn JA, Chan G, Funk M, Weinacker A, Liu JN, Drew BJ. High prevalence of corrected QT interval prolongation in acutely ill patients is associated with mortality: results of the QT in Practice (QTIP) Study. *Crit Care Med.* 2012;40:394–399. doi: 10.1097/CCM.0b013e318232db4a.

74. Pasquier M, Pantet O, Hugli O, Pruvot E, Buclin T, Waeber G, Aujesky D. Prevalence and determinants of QT interval prolongation in medical inpatients. *Intern Med J*. 2012;42:933–940. doi: 10.1111/j.1445-5994.2011.02447.x.
75. Tisdale JE, Wroblewski HA, Overholser BR, Kingery JR, Trujillo TN, Kovacs RJ. Prevalence of QT interval prolongation in patients admitted to cardiac care units and frequency of subsequent administration of QT interval-prolonging drugs: a prospective, observational study in a large urban academic medical center in the US. *Drug Saf*. 2012;35:459–470. doi: 10.2165/11598160-000000000-00000.
76. CredibleMeds website. <http://www.crediblemeds.org>. Accessed April 25, 2017.
77. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, Gregoratos G, Klein G, Moss AJ, Myerburg RJ, Priori SG, Quinones MA, Roden DM, Silka MJ, Tracy C. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). *Circulation*. 2006;114:e385–e484. doi: 10.1161/CIRCULATIONAHA.106.178233.
78. Malik M, Camm AJ. Evaluation of drug-induced QT interval prolongation: implications for drug approval and labelling. *Drug Saf*. 2001;24:323–351.
79. Carlsson L, Abrahamsson C, Andersson B, Duker G, Schiller-Linhardt G. Proarrhythmic effects of the class III agent almokalant: importance of infusion rate, QT dispersion, and early afterdepolarisations. *Cardiovasc Res*. 1993;27:2186–2193.
80. Drew BJ, Ackerman MJ, Funk M, Gibler WB, Kligfield P, Menon V, Philippides GJ, Roden DM, Zareba W; on behalf of the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology; Council on Cardiovascular Nursing; American College of Cardiology Foundation. Prevention of torsade de pointes in hospital settings: a scientific statement from the American Heart Association and the American College of Cardiology Foundation. *Circulation*. 2010;121:1047–1060.
81. Storm C, Hasper D, Nee J, Joerres A, Schefold JC, Kaufmann J, Roser M. Severe QTc prolongation under mild hypothermia treatment and incidence of arrhythmias after cardiac arrest—a prospective study in 34 survivors with continuous Holter ECG. *Resuscitation*. 2011;82:859–862. doi: 10.1016/j.resuscitation.2011.02.043.
82. Riaz A, Hieb H, Foley B, Mulvihill N, Crean P, Murphy RT, Daly C, Boyle N. Safety of therapeutic hypothermia in post VF/VT cardiac arrest patients. *Ir Med J*. 2013;106:55–56.
83. Al-Khatib SM, LaPointe NM, Kramer JM, Califf RM. What clinicians should know about the QT interval [published correction appears in *JAMA*. 2003;290:1318]. *JAMA*. 2003;289:2120–2127. doi: 10.1001/jama.289.16.2120.
84. Helfenbein ED, Zhou SH, Lindauer JM, Feild DQ, Gregg RE, Wang JJ, Kresge SS, Michaud FP. An algorithm for continuous real-time QT interval monitoring. *J Electrocardiol*. 2006;39(suppl):S123–S127.
85. Zhou SH, Helfenbein ED, Lindauer JM, Gregg RE, Feild DQ. Philips QT interval measurement algorithms for diagnostic, ambulatory, and patient monitoring ECG applications. *Ann Noninvasive Electrocardiol*. 2009;14(suppl):S3–S8.
86. Kligfield P, Badilini F, Rowlandson I, Xue J, Clark E, Devine B, Macfarlane P, de Bie J, Mortara D, Babaeizadeh S, Gregg R, Helfenbein ED, Green CL. Comparison of automated measurements of electrocardiographic intervals and durations by computer-based algorithms of digital electrocardiographs. *Am Heart J*. 2014;167:150–159.e1.
87. Sommargren CE, Drew BJ. Preventing torsades de pointes by careful cardiac monitoring in hospital settings. *AACN Adv Crit Care*. 2007;18:285–293.
88. Hasanien AA, Drew BJ, Howie-Esquivel J, Fung G, Harris P. Prevalence and prognostic significance of long QT interval among patients with chest pain: selecting an optimum QT rate correction formula. *J Electrocardiol*. 2013;46:336–342. doi: 10.1016/j.jelectrocard.2013.03.007.
89. Chiladakis J, Kalogeropoulos A, Zagkli F, Koutsogiannis N, Chouchoulis K, Alexopoulos D. Predicting torsade de pointes in acquired long QT syndrome: optimal identification of critical QT interval prolongation. *Cardiology*. 2012;122:3–11. doi: 10.1159/000338345.
90. Indik JH, Pearson EC, Fried K, Woosley RL. Bazett and Fridericia QT correction formulas interfere with measurement of drug-induced changes in QT interval. *Heart Rhythm*. 2006;3:1003–1007. doi: 10.1016/j.hrthm.2006.05.023.
91. Luo S, Michler K, Johnston P, Macfarlane PW. A comparison of commonly used QT correction formulae: the effect of heart rate on the QTc of normal ECGs. *J Electrocardiol*. 2004;37(suppl):81–90.
92. Bednar MM, Harrigan EP, Anziano RJ, Camm AJ, Ruskin JN. The QT interval. *Prog Cardiovasc Dis*. 2001;43(suppl):1–45.
93. Beach SR, Celano CM, Noseworthy PA, Januzzi JL, Huffman JC. QTc prolongation, torsades de pointes, and psychotropic medications. *Psychosomatics*. 2013;54:1–13. doi: 10.1016/j.psych.2012.11.001.
94. Moss AJ, Zareba W, Benhorin J, Couderc J, Kennedy H, Locati-Heilbron E, Maison-Blanche P. ISHNE guidelines for electrocardiographic evaluation of drug-related QT prolongation and other alterations in ventricular repolarization: task force summary. *Ann Noninvasive Electrocardiol*. 2001;6:333–341.
95. Anderson ME, Al-Khatib SM, Roden DM, Califf RM; Duke Clinical Research Institute/American Heart Journal Expert Meeting on Repolarization Changes. Cardiac repolarization: current knowledge, critical gaps, and new approaches to drug development and patient management. *Am Heart J*. 2002;144:769–781.
96. Michels G, Kochanek M, Pfister R. Life-threatening cardiac arrhythmias due to drug-induced QT prolongation: a retrospective study over 6 years from a medical intensive care unit. *Med Klin Intensivmed Notfmed*. 2016;111:302–309. doi: 10.1007/s00063-015-0071-6.
97. Digby GC, Pérez Riera AR, Barbosa Barros R, Simpson CS, Redfearn DP, Methot M, Femenía F, Baranchuk A. Acquired long QT interval: a case series of multifactorial QT prolongation. *Clin Cardiol*. 2011;34:577–582. doi: 10.1002/clc.20945.
98. Machado C, Baga JJ, Kawasaki R, Reinhold J, Steinman RT, Lehmann MH. Torsade de pointes as a complication of subarachnoid hemorrhage: a critical reappraisal. *J Electrocardiol*. 1997;30:31–37.
99. Zipes DP, Ackerman MJ, Estes NA 3rd, Grant AO, Myerburg RJ, Van Hare G. Task Force 7: arrhythmias. *J Am Coll Cardiol*. 2005;45:1354–1363. doi: 10.1016/j.jacc.2005.02.014.
100. Helfenbein ED, Ackerman MJ, Rautaharju PM, Zhou SH, Gregg RE, Lindauer JM, Miller D, Wang JJ, Kresge SS, Babaeizadeh S, Feild DQ, Michaud FP. An algorithm for QT interval monitoring in neonatal intensive care units. *J Electrocardiol*. 2007;40(suppl):S103–S110.
101. Zaman S, Kovoor P. Sudden cardiac death early after myocardial infarction: pathogenesis, risk stratification, and primary prevention. *Circulation*. 2014;129:2426–2435. doi: 10.1161/CIRCULATIONAHA.113.007497.
102. Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Judd SE, Kissela BM, Lackland DT, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Matchar DB, McGuire DK, Mohler ER 3rd, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Willey JZ, Woo D, Yeh RW, Turner MB; on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2015 update: a report from the American Heart Association [published corrections appear in *Circulation*. 2015;131:e535 and *Circulation*. 2016;133:e417]. *Circulation*. 2015;131:e29–e322. doi: 10.1161/CIR.0000000000000152.
103. Adabag AS, Therneau TM, Gersh BJ, Weston SA, Roger VL. Sudden death after myocardial infarction. *JAMA*. 2008;300:2022–2029. doi: 10.1001/jama.2008.553.
104. Solomon SD, Zelenkofske S, McMurray JJ, Finn PV, Velazquez E, Ertl G, Harsanyi A, Rouleau JL, Maggioni A, Kober L, White H, Van de Werf F, Pieper K, Califf RM, Pfeffer MA; Valsartan in Acute Myocardial Infarction Trial (VALIANT) Investigators. Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure, or both [published correction appears in *N Engl J Med*. 2005;353:744]. *N Engl J Med*. 2005;352:2581–2588. doi: 10.1056/NEJMoa043938.
105. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, Chavey WE 2nd, Fesmire FM, Hochman JS, Levin TN, Lincoff AM, Peterson ED, Theroux P, Wenger NK, Wright RS. ACCF/AHA focused update incorporated into the ACCF/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines [published correction appears in *Circulation*. 2013;127:e863–864]. *Circulation*. 2013;127:e663–e828. doi: 10.1161/CIR.0b013e31828478ac.
106. Hollander JE, Sites FD, Pollack CV Jr, Shofer FS. Lack of utility of telemetry monitoring for identification of cardiac death and life-threatening ventricular dysrhythmias in low-risk patients with chest pain. *Ann Emerg Med*. 2004;43:71–76. doi: 10.1016/S0196064403007194.

107. Chen EH, Mills AM. Is it necessary to admit low-risk patients with suspected acute coronary syndrome to inpatient telemetry beds? *Ann Emerg Med*. 2005;46:440–444.
108. Morrison LJ, Neumar RW, Zimmerman JL, Link MS, Newby LK, McMullan PW Jr, Hoek TV, Halverson CC, Doering L, Peberdy MA, Edelson DP; on behalf of the American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Peripheral Vascular Disease. Strategies for improving survival after in-hospital cardiac arrest in the United States: 2013 consensus recommendations: a consensus statement from the American Heart Association. *Circulation*. 2013;127:1538–1563. doi: 10.1161/CIR.0b013e31828b2770.
109. Larson TS, Brady WJ. Electrocardiographic monitoring in the hospitalized patient: a diagnostic intervention of uncertain clinical impact. *Am J Emerg Med*. 2008;26:1047–1055. doi: 10.1016/j.ajem.2007.12.003.
110. Brady WJ, Gurka KK, Mehning B, Peberdy MA, O'Connor RE; American Heart Association's Get With the Guidelines (formerly, NRCPR) Investigators. In-hospital cardiac arrest: impact of monitoring and witnessed event on patient survival and neurologic status at hospital discharge. *Resuscitation*. 2011;82:845–852. doi: 10.1016/j.resuscitation.2011.02.028.
111. Chopra V, McMahon LF Jr. Redesigning hospital alarms for patient safety: alarmed and potentially dangerous. *JAMA*. 2014;311:1199–1200. doi: 10.1001/jama.2014.710.
112. Farkouh ME, Smars PA, Reeder GS, Zinsmeister AR, Evans RW, Meloy TD, Kopecky SL, Allen M, Allison TG, Gibbons RJ, Gabriel SE. A clinical trial of a chest-pain observation unit for patients with unstable angina: Chest Pain Evaluation in the Emergency Room (CHEER) Investigators. *N Engl J Med*. 1998;339:1882–1888. doi: 10.1056/NEJM199812243392603.
113. Farkouh ME, Aneja A, Reeder GS, Smars PA, Bansilal S, Lennon RJ, Wiste HJ, Razzouk L, Traverse K, Holmes DR Jr, Mathew V. Clinical risk stratification in the emergency department predicts long-term cardiovascular outcomes in a population-based cohort presenting with acute chest pain: primary results of the Olmsted County Chest Pain Study. *Medicine (Baltimore)*. 2009;88:307–313. doi: 10.1097/MD.0b013e3181b98782.
114. Amsterdam EA, Wenger NK, Brindis RG, Casey DE Jr, Ganiats TG, Holmes DR Jr, Jaffe AS, Jneid H, Kelly RF, Kontos MC, Levine GN, Liebson PR, Mukherjee D, Peterson ED, Sabatine MS, Smalling RW, Zieman SJ. 2014 ACC/AHA guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [published correction appears in *Circulation*. 2014;130:e431–e432]. *Circulation*. 2014;130:2354–2394. doi: 10.1161/CIR.0000000000000133.
115. Goldman L, Cook EF, Johnson PA, Brand DA, Rouan GW, Lee TH. Prediction of the need for intensive care in patients who come to emergency departments with acute chest pain. *N Engl J Med*. 1996;334:1498–1504. doi: 10.1056/NEJM19960603342303.
116. Sanchis J, Bodí V, Núñez J, Bertomeu-González V, Gómez C, Bosch MJ, Consuegra L, Bosch X, Chorro FJ, Llacer A. New risk score for patients with acute chest pain, non-ST-segment deviation, and normal troponin concentrations: a comparison with the TIMI risk score. *J Am Coll Cardiol*. 2005;46:443–449. doi: 10.1016/j.jacc.2005.04.037.
117. Christenson J, Innes G, McKnight D, Thompson CR, Wong H, Yu E, Boychuk B, Grafstein E, Rosenberg F, Gin K, Anis A, Singer J. A clinical prediction rule for early discharge of patients with chest pain. *Ann Emerg Med*. 2006;47:1–10. doi: 10.1016/j.annemergmed.2005.08.007.
118. Backus BE, Six AJ, Kelder JC, Bosschaert MA, Mast EG, Mosterd A, Veldkamp RF, Wardeh AJ, Tio R, Braam R, Monnick SH, van Tooren R, Mast TP, van den Akker F, Cramer MJ, Poldervaart JM, Hoes AW, Doevendans PA. A prospective validation of the HEART score for chest pain patients at the emergency department. *Int J Cardiol*. 2013;168:2153–2158. doi: 10.1016/j.ijcard.2013.01.255.
119. Fesmire FM, Martin EJ, Cao Y, Heath GW. Improving risk stratification in patients with chest pain: the Erlanger HEARTS3 score. *Am J Emerg Med*. 2012;30:1829–1837. doi: 10.1016/j.ajem.2012.03.017.
120. Hess EP, Brison RJ, Perry JJ, Calder LA, Thiruganasambandamoorthy V, Agarwal D, Sadosty AT, Silvilotti ML, Jaffe AS, Montori VM, Wells GA, Stiell IG. Development of a clinical prediction rule for 30-day cardiac events in emergency department patients with chest pain and possible acute coronary syndrome. *Ann Emerg Med*. 2012;59:115–125.e1. doi: 10.1016/j.annemergmed.2011.07.026.
121. Antman EM, Cohen M, Bernink PJ, McCabe CH, Horacek T, Papuchis G, Mautner B, Corbalan R, Radley D, Braunwald E. The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision making. *JAMA*. 2000;284:835–842.
122. Boersma E, Pieper KS, Steyerberg EW, Wilcox RG, Chang WC, Lee KL, Akkerhuis KM, Harrington RA, Deckers JW, Armstrong PW, Lincoff AM, Califf RM, Topol EJ, Simoons ML. Predictors of outcome in patients with acute coronary syndromes without persistent ST-segment elevation. Results from an international trial of 9461 patients: the PURSUIT Investigators. *Circulation*. 2000;101:2557–2567.
123. Eagle KA, Lim MJ, Dabbous OH, Pieper KS, Goldberg RJ, Van de Werf F, Goodman SG, Granger CB, Steg PG, Gore JM, Budaj A, Avezum A, Flather MD, Fox KA; GRACE Investigators. A validated prediction model for all forms of acute coronary syndrome: estimating the risk of 6-month postdischarge death in an international registry. *JAMA*. 2004;291:2727–2733. doi: 10.1001/jama.291.22.2727.
124. NCDR: National Cardiovascular Data Registry. Registries. <https://www.ncdr.com/webncdr/action/>. Accessed August 14, 2017.
125. Newby KH, Thompson T, Stebbins A, Topol EJ, Califf RM, Natale A. Sustained ventricular arrhythmias in patients receiving thrombolytic therapy: incidence and outcomes: the GUSTO Investigators. *Circulation*. 1998;98:2567–2573.
126. Chiriboga D, Yarzebski J, Goldberg RJ, Gore JM, Alpert JS. Temporal trends (1975 through 1990) in the incidence and case-fatality rates of primary ventricular fibrillation complicating acute myocardial infarction: a communitywide perspective. *Circulation*. 1994;89:998–1003.
127. Mehta RH, Harjai KJ, Grines L, Stone GW, Boura J, Cox D, O'Neill W, Grines CL; Primary Angioplasty in Myocardial Infarction (PAMI) Investigators. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. *J Am Coll Cardiol*. 2004;43:1765–1772. doi: 10.1016/j.jacc.2003.09.072.
128. Mehta RH, Starr AZ, Lopes RD, Hochman JS, Widimsky P, Pieper KS, Armstrong PW, Granger CB; APEX AMI Investigators. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. *JAMA*. 2009;301:1779–1789. doi: 10.1001/jama.2009.600.
129. Piccini JP, White JA, Mehta RH, Lokhygina Y, Al-Khatib SM, Tricoci P, Pollack CV Jr, Montalescot G, Van de Werf F, Gibson CM, Giugliano RP, Califf RM, Harrington RA, Newby LK. Sustained ventricular tachycardia and ventricular fibrillation complicating non-ST-segment-elevation acute coronary syndromes. *Circulation*. 2012;126:41–49. doi: 10.1161/CIRCULATIONAHA.111.071860.
130. Winkler C, Funk M, Schindler DM, Hemsey JZ, Lampert R, Drew BJ. Arrhythmias in patients with acute coronary syndrome in the first 24 hours of hospitalization. *Heart Lung*. 2013;42:422–427. doi: 10.1016/j.hrtlung.2013.07.010.
131. Patel DJ, Holdright DR, Knight CJ, Mulcahy D, Thakrar B, Wright C, Sparrow J, Wicks M, Hubbard W, Thomas R, Sutton GC, Hendry G, Purcell H, Fox K. Early continuous ST segment monitoring in unstable angina: prognostic value additional to the clinical characteristics and the admission electrocardiogram. *Heart*. 1996;75:222–228.
132. Patel D, Knight C, Holdright D, Mulcahy D, Clarke D, Wright C, Purcell H, Fox K. Long-term prognosis in unstable angina. *Eur Heart J*. 1998;19:240–249.
133. Fesmire FM, Hughes AD, Fody EP, Jackson AP, Fesmire CE, Gilbert MA, Stout PK, Wojcik JF, Wharton DR, Creel JH. The Erlanger chest pain evaluation protocol: a one-year experience with serial 12-lead ECG monitoring, two-hour delta serum marker measurements, and selective nuclear stress testing to identify and exclude acute coronary syndromes. *Ann Emerg Med*. 2002;40:584–594.
134. Gomez MA, Anderson JL, Karagounis LA, Muhlestein JB, Mooers FB. An emergency department-based protocol for rapidly ruling out myocardial ischemia reduces hospital time and expense: results of a randomized study (ROMIO). *J Am Coll Cardiol*. 1996;28:25–33.
135. Amsterdam EA, Lewis WR, Kirk JD, Diercks DB, Turnipseed S. Acute ischemic syndromes: chest pain center concept. *Cardiol Clin*. 2002;20:117–136.
136. Fesmire FM, Wharton DR, Calhoun FB. Instability of ST segments in the early stages of acute myocardial infarction in patients undergoing continuous 12-lead ECG monitoring. *Am J Emerg Med*. 1995;13:158–163. doi: 10.1016/0735-6757(95)90084-5.
137. Selker HP, Zaleski RJ, Antman EM, Aufderheide TP, Bernard SA, Bonow RO, Gibler WB, Hagen MD, Johnson P, Lau J. Continuous 12-lead ECG. *Ann Emerg Med*. 1997;29:25–28.

138. Fesmire FM, Percy RF, Bardoner JB, Wharton DR, Calhoun FB. Usefulness of automated serial 12-lead ECG monitoring during the initial emergency department evaluation of patients with chest pain. *Ann Emerg Med.* 1998;31:3–11.
139. Carmo P, Ferreira J, Aguiar C, Ferreira A, Raposo L, Gonçalves P, Brito J, Silva A. Does continuous ST-segment monitoring add prognostic information to the TIMI, PURSUIT, and GRACE risk scores? *Ann Noninvasive Electrocardiol.* 2011;16:239–249. doi: 10.1111/j.1542-474X.2011.00438.x.
140. Drew BJ, Pelter MM, Lee E, Zegre J, Schindler D, Fleischmann KE. Designing prehospital ECG systems for acute coronary syndromes: lessons learned from clinical trials involving 12-lead ST-segment monitoring. *J Electrocardiol.* 2005;38(suppl):180–185. doi: 10.1016/j.jelectrocard.2005.06.031.
141. Drew BJ, Sommargren CE, Schindler DM, Benedict K, Zegre-Hemsey J, Glancy JP. A simple strategy improves prehospital electrocardiogram utilization and hospital treatment for patients with acute coronary syndrome (from the ST SMART Study). *Am J Cardiol.* 2011;107:347–352. doi: 10.1016/j.amjcard.2010.09.027.
142. Fesmire FM. Which chest pain patients potentially benefit from continuous 12-lead ST-segment monitoring with automated serial ECG? *Am J Emerg Med.* 2000;18:773–778. doi: 10.1053/ajem.2000.18036.
143. Gottlieb SO, Weisfeldt ML, Ouyang P, Mellits ED, Gerstenblith G. Silent ischemia as a marker for early unfavorable outcomes in patients with unstable angina. *N Engl J Med.* 1986;314:1214–1219.
144. Gottlieb SO, Weisfeldt ML, Ouyang P, Mellits ED, Gerstenblith G. Silent ischemia predicts infarction and death during 2 year follow-up of unstable angina. *J Am Coll Cardiol.* 1987;10:756–760.
145. Nademanee K, Intarachot V, Josephson MA, Rieders D, Vaghaiwalla Mody F, Singh BN. Prognostic significance of silent myocardial ischemia in patients with unstable angina. *J Am Coll Cardiol.* 1987;10:1–9.
146. Langer A, Freeman MR, Armstrong PW. ST segment shift in unstable angina: pathophysiology and association with coronary anatomy and hospital outcome. *J Am Coll Cardiol.* 1989;13:1495–1502.
147. Larsson H, Jonasson T, Ringqvist I, Fellenius C, Wallentin L. Diagnostic and prognostic importance of ST recording after an episode of unstable angina or non-Q-wave myocardial infarction. *Eur Heart J.* 1992;13:207–212.
148. Krucoff MW, Croll MA, Pope JE, Pieper KS, Kanani PM, Granger CB, Veldkamp RF, Wagner BL, Sawchak ST, Califf RM. Continuously updated 12-lead ST-segment recovery analysis for myocardial infarct artery patency assessment and its correlation with multiple simultaneous early angiographic observations. *Am J Cardiol.* 1993;71:145–151.
149. Veldkamp RF, Green CL, Wilkins ML, Pope JE, Sawchak ST, Ryan JA, Califf RM, Wagner BS, Krucoff MW. Comparison of continuous ST-segment recovery analysis with methods using static electrocardiograms for noninvasive patency assessment during acute myocardial infarction. Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) 7 Study Group. *Am J Cardiol.* 1994;73:1069–1074.
150. Langer A, Krucoff MW, Klootwijk P, Veldkamp R, Simoons ML, Granger C, Califf RM, Armstrong PW. Noninvasive assessment of speed and stability of infarct-related artery reperfusion: results of the GUSTO ST segment monitoring study: Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries. *J Am Coll Cardiol.* 1995;25:1552–1557.
151. Santoro GM, Buonamici P. Reperfusion therapy in cardiogenic shock complicating acute myocardial infarction. *Am Heart J.* 1999;138(pt 2):S126–S131.
152. Albertal M, Cura F, Thierer J, Trivi M, Escudero AG, Padilla LT, Belardi JA. Time to stable ST-segment recovery following primary angioplasty is predictive of regional wall motion score and clinical outcome. *Angiology.* 2010;61:638–642. doi: 10.1177/0003319710369099.
153. Bogaty P, Dumont S, O'Hara GE, Boyer L, Auclair L, Jobin J, Boudreault JR. Randomized trial of a noninvasive strategy to reduce hospital stay for patients with low-risk myocardial infarction. *J Am Coll Cardiol.* 2001;37:1289–1296.
154. Kandzari DE, Tchong JE, Cohen DJ, Bakhai A, Grines CL, Cox DA, Effron M, Stuckey T, Griffin JJ, Turco M, Carroll JD, Fahy M, Mehran R, Stone GW; CADILLAC Investigators. Feasibility and implications of an early discharge strategy after percutaneous intervention with abximab in acute myocardial infarction (the CADILLAC Trial). *Am J Cardiol.* 2003;92:779–784.
155. De Luca G, van 't Hof AW, de Boer MJ, Ottervanger JP, Hoorntje JC, Gosselink AT, Dambrink JH, Zijlstra F, Suryapranata H. Time-to-treatment significantly affects the extent of ST-segment resolution and myocardial blush in patients with acute myocardial infarction treated by primary angioplasty. *Eur Heart J.* 2004;25:1009–1013. doi: 10.1016/j.ehj.2004.03.021.
156. Addala S, Kahn JK, Moccia TF, Harjai K, Pellizon G, Ochoa A, O'Neill WW. Outcome of ventricular fibrillation developing during percutaneous coronary interventions in 19,497 patients without cardiogenic shock. *Am J Cardiol.* 2005;96:764–765. doi: 10.1016/j.amjcard.2005.04.057.
157. Piccini JP, Berger JS, Brown DL. Early sustained ventricular arrhythmias complicating acute myocardial infarction. *Am J Med.* 2008;121:797–804. doi: 10.1016/j.amjmed.2008.04.024.
158. Czarnecki A, Welsh RC, Yan RT, DeYoung JP, Gallo R, Rose B, Grondin FR, Kornder JM, Wong GC, Fox KA, Gore JM, Goodman SG, Yan AT; Global Registry of Acute Coronary Events (GRACE/GRACE2); Canadian Registry of Coronary Events (CANRACE) Investigators. Reperfusion strategies and outcomes of ST-segment elevation myocardial infarction patients in Canada: observations from the Global Registry of Acute Coronary Events (GRACE) and the Canadian Registry of Acute Coronary Events (CANRACE). *Can J Cardiol.* 2012;28:40–47. doi: 10.1016/j.cjca.2011.09.011.
159. Ohlow MA, Geller JC, Richter S, Farah A, Müller S, Fuhrmann JT, Lauer B. Incidence and predictors of ventricular arrhythmias after ST-segment elevation myocardial infarction. *Am J Emerg Med.* 2012;30:580–586. doi: 10.1016/j.ajem.2011.02.029.
160. Rahimi K, Watzlawek S, Thiele H, Secknus MA, Hayerizadeh BF, Niebauer J, Schuler G. Incidence, time course, and predictors of early malignant ventricular arrhythmias after non-ST-segment elevation myocardial infarction in patients with early invasive treatment. *Eur Heart J.* 2006;27:1706–1711. doi: 10.1093/eurheartj/ehl100.
161. Harskamp RE, Wang TY, Bhatt DL, Wiviott SD, Amsterdam EA, Li S, Thomas L, de Winter RJ, Roe MT. Hospital patterns of medical management strategy use for patients with non-ST-elevation myocardial infarction and 3-vessel or left main coronary artery disease. *Am Heart J.* 2014;167:355–362. e3.
162. Scirica BM, Braunwald E, Belardinelli L, Hedgepeth CM, Spinar J, Wang W, Qin J, Karwatowska-Prokopczuk E, Verheugt FW, Morrow DA. Relationship between nonsustained ventricular tachycardia after non-ST-elevation acute coronary syndrome and sudden cardiac death: observations from the Metabolic Efficiency With Ranolazine for Less Ischemia in Non-ST-Elevation Acute Coronary Syndrome-Thrombolysis in Myocardial Infarction 36 (MERLIN-TIMI 36) randomized controlled trial. *Circulation.* 2010;122:455–462. doi: 10.1161/CIRCULATIONAHA.110.937136.
163. Harkness JR, Morrow DA, Braunwald E, Ren F, Lopez-Sendon J, Bode C, Budaj A, Scirica BM. Myocardial ischemia and ventricular tachycardia on continuous electrocardiographic monitoring and risk of cardiovascular outcomes after non-ST-segment elevation acute coronary syndrome (from the MERLIN-TIMI 36 Trial). *Am J Cardiol.* 2011;108:1373–1381. doi: 10.1016/j.amjcard.2011.06.058.
164. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med.* 2002;346:557–563. doi: 10.1056/NEJMoa003289.
165. Hypothermia After Cardiac Arrest Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest [published correction appears in *N Engl J Med.* 2002;346:1756]. *N Engl J Med.* 2002;346:549–556.
166. Peberdy MA, Callaway CW, Neumar RW, Geocadin RG, Zimmerman JL, Donnino M, Gabrielli A, Silvers SM, Zaritsky AL, Merchant R, Vanden Hoek TL, Kronick SL. Part 9: post-cardiac arrest care: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation.* 2011;123:e237]. *Circulation.* 2010;122(suppl 3):S768–S786. doi: 10.1161/CIRCULATIONAHA.110.971002.
167. Vassallo SU, Delaney KA, Hoffman RS, Slater W, Goldfrank LR. A prospective evaluation of the electrocardiographic manifestations of hypothermia. *Acad Emerg Med.* 1999;6:1121–1126.
168. Mattu A, Brady WJ, Perron AD. Electrocardiographic manifestations of hypothermia. *Am J Emerg Med.* 2002;20:314–326.
169. Aslam AF, Aslam AK, Vasavada BC, Khan IA. Hypothermia: evaluation, electrocardiographic manifestations, and management. *Am J Med.* 2006;119:297–301. doi: 10.1016/j.amjmed.2005.09.062.
170. Salinski EP, Worrlow CC. ST-segment elevation myocardial infarction vs. hypothermia-induced electrocardiographic changes: a case report and brief review of the literature. *J Emerg Med.* 2014;46:e107–e111. doi: 10.1016/j.jemermed.2013.08.122.
171. Wang H, Hollingsworth J, Mahler S, Arnold T. Diffuse ST segment depression from hypothermia. *Int J Emerg Med.* 2010;3:451–454. doi: 10.1007/s12245-010-0211-y.

172. Bonnemeier H, Mäuser W, Schunkert H. Images in cardiovascular medicine: Brugada-like ECG pattern in severe hypothermia. *Circulation*. 2008;118:977–978. doi: 10.1161/CIRCULATIONAHA.108.771329.
173. Rolfast CL, Lust EJ, de Cock CC. Electrocardiographic changes in therapeutic hypothermia. *Crit Care*. 2012;16:R100. doi: 10.1186/cc11369.
174. JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (Coronary Spastic Angina) (JCS 2008). *Circ J*. 2010;74:1745–1762.
175. Prinzmetal M, Kenamer R, Merliss R. A variant form of angina pectoris. *Am J Med*. 1959;27:376–388.
176. Kusama Y, Kodani E, Nakagomi A, Otsuka T, Atarashi H, Kishida H, Mizuno K. Variant angina and coronary artery spasm: the clinical spectrum, pathophysiology, and management. *J Nippon Med Sch*. 2011;78:4–12.
177. Figueras J, Domingo E, Ferreira I, Lidón RS, Garcia-Dorado D. Persistent angina pectoris, cardiac mortality and myocardial infarction during a 12 year follow-up in 273 variant angina patients without significant fixed coronary stenosis. *Am J Cardiol*. 2012;110:1249–1255.
178. Holt DB, Singh GK, Rhee EK, Billadello J, Ludomirsky A. Images in cardiovascular medicine: Prinzmetal angina in an adolescent: adjunctive role of tissue synchronization imaging. *Circulation*. 2005;112:e91–e92.
179. Jefferies JL, Treisman B. Prinzmetal angina in a teenage girl. *Pediatr Cardiol*. 2005;26:486–487. doi: 10.1007/s00246-004-0811-5.
180. Kounis NG. Coronary hypersensitivity disorder: the Kounis syndrome. *Clin Ther*. 2013;35:563–571. doi: 10.1016/j.clinthera.2013.02.022.
181. Ong P, Athanasiadis A, Hill S, Vogelsberg H, Voehringer M, Sechtem U. Coronary artery spasm as a frequent cause of acute coronary syndrome: the CASPAR (Coronary Artery Spasm in Patients With Acute Coronary Syndrome) Study. *J Am Coll Cardiol*. 2008;52:523–527. doi: 10.1016/j.jacc.2008.04.050.
182. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*. 2005;352:539–548. doi: 10.1056/NEJMoa043046.
183. Williams DS. Prinzmetal angina. *J Insur Med*. 2009;41:77–79.
184. Pilgrim TM, Wyss TR. Takotsubo cardiomyopathy or transient left ventricular apical ballooning syndrome: a systematic review. *Int J Cardiol*. 2008;124:283–292. doi: 10.1016/j.ijcard.2007.07.002.
185. Kurisu S, Inoue I, Kawagoe T, Ishihara M, Shimatani Y, Hata T, Nakama Y, Kijima Y, Kagawa E. Documentation of dynamic electrocardiographic changes shortly after the onset of tako-tsubo cardiomyopathy. *Int J Cardiol*. 2007;119:258–260. doi: 10.1016/j.ijcard.2006.07.141.
186. Virani SS, Mendoza CE, Ferreira AC, de Marchena E. Left main coronary artery stenosis: factors predicting cardiac events in patients awaiting coronary surgery. *Tex Heart Inst J*. 2006;33:23–26.
187. Li JM, Camenzind E, Meier B, Urban P, Giroud D, Rutishauser W. In-hospital monitoring after coronary angioplasty. *J Interv Cardiol*. 1994;7:229–235.
188. Black AJ, Namay DL, Niederman AL, Lembo NJ, Roubin GS, Douglas JS Jr, King SB 3rd. Tear or dissection after coronary angioplasty: morphologic correlates of an ischemic complication. *Circulation*. 1989;79:1035–1042.
189. Cheneau E, Leborgne L, Mintz GS, Kotani J, Pichard AD, Satler LF, Canos D, Castagna M, Weissman NJ, Waksman R. Predictors of subacute stent thrombosis: results of a systematic intravascular ultrasound study. *Circulation*. 2003;108:43–47. doi: 10.1161/01.CIR.0000078636.71728.40.
190. Spoon DB, Psaltis PJ, Singh M, Holmes DR Jr, Gersh BJ, Rihal CS, Lennon RJ, Moussa ID, Simari RD, Gulati R. Trends in cause of death after percutaneous coronary intervention. *Circulation*. 2014;129:1286–1294. doi: 10.1161/CIRCULATIONAHA.113.006518.
191. Thel MC, Califf RM, Tardiff BE, Gardner LH, Sigmon KN, Lincoff AM, Topol EJ, Kitt MM, Blankenship JC, Tchong JE. Timing of and risk factors for myocardial ischemic events after percutaneous coronary intervention (IMPACT-II): Integrilin to Minimize Platelet Aggregation and Coronary Thrombosis. *Am J Cardiol*. 2000;85:427–434.
192. Jozic J, Orford J, Steinhilb S, Berger P, Hsu A, Topol E. Timing and correlates of very early major adverse clinical events following percutaneous coronary intervention. *J Invasive Cardiol*. 2008;20:113–118.
193. Brayton KM, Patel VG, Stave C, de Lemos JA, Kumbhani DJ. Same-day discharge after percutaneous coronary intervention: a meta-analysis. *J Am Coll Cardiol*. 2013;62:275–285. doi: 10.1016/j.jacc.2013.03.051.
194. Scanlon PJ, Faxon DP, Audet AM, Carabello B, Dehmer GJ, Eagle KA, Legako RD, Leon DF, Murray JA, Nissen SE, Pepine CJ, Watson RM. ACC/AHA guidelines for coronary angiography: executive summary and recommendations: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Coronary Angiography). *Circulation*. 1999;99:2345–2357.
195. Tavakol M, Ashraf S, Brenner SJ. Risks and complications of coronary angiography: a comprehensive review. *Glob J Health Sci*. 2012;4:65–93. doi: 10.5539/gjhs.v4n1p65.
196. Chen J, Gao L, Yao M, Chen J. Ventricular arrhythmia onset during diagnostic coronary angiography with a 5F or 4F universal catheter. *Rev Esp Cardiol*. 2008;61:1092–1095.
197. Johnson LW, Lozner EC, Johnson S, Krone R, Pichard AD, Vetrovec GW, Noto TJ. Coronary arteriography 1984-1987: a report of the Registry of the Society for Cardiac Angiography and Interventions, I: results and complications. *Cathet Cardiovasc Diagn*. 1989;17:5–10.
198. Saleem MA, McClung JA, Aronow WS, Kannam H. Inpatient telemetry does not need to be used in the management of older patients hospitalized with chest pain at low risk for in-hospital coronary events and mortality. *J Gerontol A Biol Sci Med Sci*. 2005;60:605–606.
199. Singer AJ, Visram F, Shembekar A, Khwaja M, Viccellio A. Telemetry monitoring during transport of low-risk chest pain patients from the emergency department: is it necessary? *Acad Emerg Med*. 2005;12:965–969. doi: 10.1197/j.aem.2005.06.007.
200. Atzema C, Schull MJ, Borgundvaag B, Slaughter GR, Lee CK. ALARMED: adverse events in low-risk patients with chest pain receiving continuous electrocardiographic monitoring in the emergency department: a pilot study. *Am J Emerg Med*. 2006;24:62–67. doi: 10.1016/j.ajem.2005.05.015.
201. Chen EH, Hollander JE. When do patients need admission to a telemetry bed? *J Emerg Med*. 2007;33:53–60. doi: 10.1016/j.jemermed.2007.01.017.
202. Six AJ, Backus BE, Kelder JC. Chest pain in the emergency room: value of the HEART score. *Neth Heart J*. 2008;16:191–196.
203. Bovino LR, Funk M, Pelter MM, Desai MM, Jefferson V, Andrews LK, Forte K. The value of continuous ST-segment monitoring in the emergency department. *Adv Emerg Nurs J*. 2015;37:290–300. doi: 10.1097/TME.000000000000080.
204. Siebert J, Rogowski J, Jagielak D, Anisimowicz L, Lango R, Narkiewicz M. Atrial fibrillation after coronary artery bypass grafting without cardiopulmonary bypass. *Eur J Cardiothorac Surg*. 2000;17:520–523.
205. Funk M, Richards SB, Desjardins J, Bebon C, Wilcox H. Incidence, timing, symptoms, and risk factors for atrial fibrillation after cardiac surgery. *Am J Crit Care*. 2003;12:424–433; quiz 434–435.
206. Nisanoglu V, Erdil N, Aldemir M, Ozgur B, Berat Cihan H, Yoluglu S, Battaloglu B. Atrial fibrillation after coronary artery bypass grafting in elderly patients: incidence and risk factor analysis. *Thorac Cardiovasc Surg*. 2007;55:32–38. doi: 10.1055/s-2006-924711.
207. Koletsis EN, Prokakis C, Crockett JR, Dedeilias P, Panagiotou M, Panagopoulos N, Anastasiou N, Dougenis D, Apostolakis E. Prognostic factors of atrial fibrillation following elective coronary artery bypass grafting: the impact of quantified intraoperative myocardial ischemia. *J Cardiothorac Surg*. 2011;6:127. doi: 10.1186/1749-8090-6-127.
208. Maesen B, Nijs J, Maessen J, Allesie M, Schotten U. Post-operative atrial fibrillation: a maze of mechanisms. *Europace*. 2012;14:159–174. doi: 10.1093/europace/eur208.
209. Amat-Santos IJ, Rodés-Cabau J, Urena M, DeLarochelière R, Doyle D, Bagur R, Villeneuve J, Côté M, Nombela-Franco L, Philippon F, Pibarot P, Dumont E. Incidence, predictive factors, and prognostic value of new-onset atrial fibrillation following transcatheter aortic valve implantation. *J Am Coll Cardiol*. 2012;59:178–188. doi: 10.1016/j.jacc.2011.09.061.
210. Maisel WH, Rawn JD, Stevenson WG. Atrial fibrillation after cardiac surgery. *Ann Intern Med*. 2001;135:1061–1073.
211. Brembilla-Perrot B, Villemot J, Carteau J, Haouzi A, Amrein D, Schöth B, Houriez P, Beurrier D, Djaballah K, Vancon AC, Nippert M. Postoperative ventricular arrhythmias after cardiac surgery: immediate- and long-term significance. *Pacing Clin Electrophysiol*. 2003;26(pt 1):619–625.
212. Merin O, Ilan M, Oren A, Fink D, Deeb M, Bitran D, Silberman S. Permanent pacemaker implantation following cardiac surgery: indications and long-term follow-up. *Pacing Clin Electrophysiol*. 2009;32:7–12. doi: 10.1111/j.1540-8159.2009.02170.x.
213. Huynh H, Dalloul G, Ghanbari H, Burke P, David M, Daccarett M, Machado C, David S. Permanent pacemaker implantation following aortic valve replacement: current prevalence and clinical predictors. *Pacing Clin Electrophysiol*. 2009;32:1520–1525. doi: 10.1111/j.1540-8159.2009.02552.x.

214. Gordon MA, Urban MK, O'Connor T, Barash PG. Is the pressure rate quotient a predictor or indicator of myocardial ischemia as measured by ST-segment changes in patients undergoing coronary artery bypass surgery? *Anesthesiology*. 1991;74:848–853.
215. Jain U, Laflamme CJ, Aggarwal A, Ramsay JG, Comunale ME, Ghoshal S, Ngo L, Ziola K, Hollenberg M, Mangano DT. Electrocardiographic and hemodynamic changes and their association with myocardial infarction during coronary artery bypass surgery: a multicenter study: Multicenter Study of Perioperative Ischemia (MCSPI) Research Group. *Anesthesiology*. 1997;86:576–591.
216. Knight AA, Hollenberg M, London MJ, Tubau J, Verrier E, Browner W, Mangano DT. Perioperative myocardial ischemia: importance of the pre-operative ischemic pattern. *Anesthesiology*. 1988;68:681–688.
217. Berry PD, Thomas SD, Mahon SP, Jackson M, Fox MA, Fabri B, Weir WI, Russell GN. Myocardial ischaemia after coronary artery bypass grafting: early vs late extubation [published corrections appear in *Br J Anaesth*. 1998;80:572 and *Br J Anaesth*. 1998;81: 111]. *Br J Anaesth*. 1998;80:20–25.
218. Mangano DT, Siliciano D, Hollenberg M, Leung JM, Browner WS, Goehner P, Merrick S, Verrier E. Postoperative myocardial ischemia: therapeutic trials using intensive analgesia following surgery: the Study of Perioperative Ischemia (SPI) Research Group. *Anesthesiology*. 1992;76:342–353.
219. Cheng DC, Karski J, Peniston C, Asokumar B, Raveendran G, Carroll J, Nierenberg H, Roger S, Mickle D, Tong J, Zelovitsky J, David T, Sandler A. Morbidity outcome in early versus conventional tracheal extubation after coronary artery bypass grafting: a prospective randomized controlled trial. *J Thorac Cardiovasc Surg*. 1996;112:755–764. doi: 10.1016/S0022-5223(96)70062-4.
220. Thielmann M, Massoudy P, Jaeger BR, Neuhäuser M, Marggraf G, Sack S, Erbel R, Jakob H. Emergency re-vascularization with percutaneous coronary intervention, reoperation, or conservative treatment in patients with acute perioperative graft failure following coronary artery bypass surgery. *Eur J Cardiothorac Surg*. 2006;30:117–125. doi: 10.1016/j.ejcts.2006.03.062.
221. Cockburn J, Blows L, Cohen A, Holmberg S, Hyde J, Lewis M, Trivedi U, Hildick-Smith D, de Belder A. Acute ischemic complications of PCI and CABG: who should cover whom for coronary revascularization? *J Interv Cardiol*. 2013;26:372–377. doi: 10.1111/joic.12045.
222. Payne L, Zeigler VL, Gillette PC. Acute cardiac arrhythmias following surgery for congenital heart disease: mechanisms, diagnostic tools, and management. *Crit Care Nurs Clin North Am*. 2011;23:255–272. doi: 10.1016/j.ccell.2011.04.001.
223. Skippen PW, Sanatani S, Gow RM, Froese N. Diagnosis of postoperative arrhythmias following paediatric cardiac surgery. *Anaesth Intensive Care*. 2009;37:705–719.
224. Makhoul M, Oster M, Fischbach P, Das S, Deshpande S. Junctional ectopic tachycardia after congenital heart surgery in the current surgical era. *Pediatr Cardiol*. 2013;34:370–374. doi: 10.1007/s00246-012-0465-7.
225. Batra AS, Chun DS, Johnson TR, Maldonado EM, Kashyap BA, Maiers J, Lindblade CL, Rodefeld M, Brown JW, Hubbard JE. A prospective analysis of the incidence and risk factors associated with junctional ectopic tachycardia following surgery for congenital heart disease. *Pediatr Cardiol*. 2006;27:51–55. doi: 10.1007/s00246-005-0992-6.
226. Backer CL, Tsao S, Deal BJ, Mavroudis C. Maze procedure in single ventricle patients. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu*. 2008;11:44–48.
227. Deal BJ. Late arrhythmias following Fontan surgery. *World J Pediatr Congenit Heart Surg*. 2012;3:194–200. doi: 10.1177/2150135111436314.
228. Smith AH, Owen J, Borgman KY, Fish FA, Kannankeril PJ. Relation of milrinone after surgery for congenital heart disease to significant post-operative tachyarrhythmias. *Am J Cardiol*. 2011;108:1620–1624. doi: 10.1016/j.amjcard.2011.07.023.
229. Epstein A, DiMarco J, Ellenbogen K, Estes N 3rd, Freedman R, Gettes L, Gillinov A, Gregoratos G, Hammill S, Hayes D, Hlatky MA, Newby LK, Page RL, Schoenfeld MH, Silka MJ, Stevenson LW, Sweeney MO. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) [published correction appears in *Circulation*. 2009;120:e34–e35]. *Circulation*. 2008;117:e350–e408. doi: 10.1161/CIRCULATIONAHA.108.189742.
230. McCafferty M, Sorbellini D, Cianci P. Telemetry to home: successful discharge of patients with ventricular assist devices. *Crit Care Nurse*. 2002;22:43–51.
231. Shinn JA, Abou-Awadi N, Ley SJ, Reedy JE, Rountree WD. Nursing care of the patient on mechanical circulatory support. *Ann Thorac Surg*. 1993;55:288–294.
232. Brisco MA, Sundareswaran KS, Milano CA, Feldman D, Testani JM, Ewald GA, Slaughter MS, Farrar DJ, Goldberg LR; HeartMate II Clinical Investigators. Incidence, risk, and consequences of atrial arrhythmias in patients with continuous-flow left ventricular assist devices. *J Card Surg*. 2014;29:572–580. doi: 10.1111/jocs.12336.
233. Enriquez AD, Calenda B, Miller MA, Anyanwu AC, Pinney SP. The role of implantable cardioverter-defibrillators in patients with continuous flow left ventricular assist devices [published correction appears in *Circ Arrhythm Electrophysiol*. 2014;7:185]. *Circ Arrhythm Electrophysiol*. 2013;6:668–674. doi: 10.1161/CIRCEP.113.000457.
234. Garan AR, Yuzefpolskaya M, Colombo PC, Morrow JP, Te-Frey R, Dano D, Takayama H, Naka Y, Garan H, Jorde UP, Uriel N. Ventricular arrhythmias and implantable cardioverter-defibrillator therapy in patients with continuous-flow left ventricular assist devices: need for primary prevention? *J Am Coll Cardiol*. 2013;61:2542–2550. doi: 10.1016/j.jacc.2013.04.020.
235. Feldman D, Pamboukian SV, Teuteberg JJ, Birks E, Lietz K, Moore SA, Morgan JA, Arabia F, Bauman ME, Buchholz HW, Deng M, Dickstein ML, El-Banayosy A, Elliot T, Goldstein DJ, Grady KL, Jones K, Hryniewicz K, John R, Kaan A, Kusne S, Loebe M, Massicotte MP, Moazami N, Mohacs P, Mooney M, Nelson T, Pagani F, Perry W, Potapov EV, Eduardo Rame J, Russell SD, Sorensen EN, Sun B, Strueber M, Mangi AA, Petty MG, Rogers J. International Society for Heart and Lung Transplantation. The 2013 International Society for Heart and Lung Transplantation guidelines for mechanical circulatory support: executive summary. *J Heart Lung Transplant*. 2013;32:157–187. doi: 10.1016/j.healun.2012.09.013.
236. Holmes DR Jr, Mack MJ, Kaul S, Agnihotri A, Alexander KP, Bailey SR, Calhoun JH, Carabello BA, Desai MY, Edwards FH, Francis GS, Gardner TJ, Kappetein AP, Linderbaum JA, Mukherjee C, Mukherjee D, Otto CM, Ruiz CE, Sacco RL, Smith D, Thomas JD. 2012 ACCF/AATS/SCAI/STS expert consensus document on transcatheter aortic valve replacement. *J Am Coll Cardiol*. 2012;59:1200–1254. doi: 10.1016/j.jacc.2012.01.001.
237. Urena M, Webb JG, Tamburino C, Muñoz-García AJ, Cheema A, Dager AE, Serra V, Amat-Santos I, Barbanti M, Imme S, Briaies JH, Benitez LM, Al Lawati H, Ucalon AM, García Del Blanco B, López J, Dumont E, Delarochellière R, Ribeiro HB, Nombela-Franco L, Philippon F, Rodés-Cabau J. Permanent pacemaker implantation after transcatheter aortic valve implantation: impact on late clinical outcomes and left ventricular function. *Circulation*. 2014;129:1233–1243. doi: 10.1161/CIRCULATIONAHA.113.005479.
238. Nazif TM, Dizon JM, Hahn RT, Xu K, Babaliaros V, Douglas PS, El-Chami MF, Herrmann HC, Mack M, Makkar RR, Miller DC, Pichard A, Tuzcu EM, Szeto WY, Webb JG, Moses JW, Smith CR, Williams MR, Leon MB, Kodali SK; PARTNER Publications Office. Predictors and clinical outcomes of permanent pacemaker implantation after transcatheter aortic valve replacement: the PARTNER (Placement of Aortic TRaNs catheter Valves) trial and registry. *JACC Cardiovasc Interv*. 2015;8(pt A):60–69. doi: 10.1016/j.jcin.2014.07.022.
239. Siontis GC, Jüni P, Pilgrim T, Stortecky S, Büllensfeld L, Meier B, Wenaweser P, Windecker S. Predictors of permanent pacemaker implantation in patients with severe aortic stenosis undergoing TAVR: a meta-analysis. *J Am Coll Cardiol*. 2014;64:129–140. doi: 10.1016/j.jacc.2014.04.033.
240. Piazza N, Onuma Y, Jesserun E, Kint PP, Maugenest AM, Anderson RH, de Jaegere PP, Serruys PW. Early and persistent intraventricular conduction abnormalities and requirements for pacemaking after percutaneous replacement of the aortic valve. *JACC Cardiovasc Interv*. 2008;1:310–316. doi: 10.1016/j.jcin.2008.04.007.
241. Bleiziffer S, Ruge H, Hörer J, Hutter A, Geisbüsch S, Brockmann G, Mazzitelli D, Bauernschmitt R, Lange R. Predictors for new-onset complete heart block after transcatheter aortic valve implantation. *JACC Cardiovasc Interv*. 2010;3:524–530. doi: 10.1016/j.jcin.2010.01.017.
242. Nuis RJ, Van Mieghem NM, Schultz CJ, Tzikas A, Van der Boon RM, Maugenest AM, Cheng J, Piazza N, van Domburg RT, Serruys PW, de Jaegere PP. Timing and potential mechanisms of new conduction abnormalities during the implantation of the Medtronic CoreValve System in patients with aortic stenosis. *Eur Heart J*. 2011;32:2067–2074. doi: 10.1093/eurheartj/ehr110.
243. Maan A, Heist EK, Passeri J, Inglessis I, Baker J, Ptaszek L, Vlahakes G, Ruskin JN, Palacios I, Sundt T, Mansour M. Impact of atrial fibrillation on outcomes in patients who underwent transcatheter aortic valve replacement. *Am J Cardiol*. 2015;115:220–226. doi: 10.1016/j.amjcard.2014.10.027.

244. Urena M, Hayek S, Cheema AN, Serra V, Amat-Santos IJ, Nombela-Franco L, Ribeiro HB, Allende R, Paradis JM, Dumont E, Thourani VH, Babaliaros V, Francisco Pascual J, Cortés C, Del Blanco BG, Philippon F, Lerakis S, Rodés-Cabau J. Arrhythmia burden in elderly patients with severe aortic stenosis as determined by continuous electrocardiographic recording: toward a better understanding of arrhythmic events after transcatheter aortic valve replacement. *Circulation*. 2015;131:469–477. doi: 10.1161/CIRCULATIONAHA.114.011929.
245. Erkapic D, De Rosa S, Kelava A, Lehmann R, Fichtlscherer S, Hohnloser SH. Risk for permanent pacemaker after transcatheter aortic valve implantation: a comprehensive analysis of the literature. *J Cardiovasc Electrophysiol*. 2012;23:391–397. doi: 10.1111/j.1540-8167.2011.02211.x.
246. Testa L, Latib A, De Marco F, De Carlo M, Agnifili M, Latini RA, Petronio AS, Etori F, Poli A, De Servi S, Ramondo A, Napodano M, Klugmann S, Ussia GP, Tamburino C, Brambilla N, Colombo A, Bedogni F. Clinical impact of persistent left bundle branch block after transcatheter aortic valve implantation with CoreValve Revalving System. *Circulation*. 2013;127:1300–1307. doi: 10.1161/CIRCULATIONAHA.113.001099.
247. lung B, Himbert D, Vahanian A. Atrial fibrillation following transcatheter aortic valve implantation: do we underestimate its frequency and impact? *J Am Coll Cardiol*. 2012;59:189–190. doi: 10.1016/j.jacc.2011.11.005.
248. Butera G, Carminati M, Chessa M, Piazza L, Micheletti A, Negura DG, Abella R, Giamberti A, Frigiola A. Transcatheter closure of perimembranous ventricular septal defects: early and long-term results. *J Am Coll Cardiol*. 2007;50:1189–1195. doi: 10.1016/j.jacc.2007.03.068.
249. Yang R, Kong XQ, Sheng YH, Zhou L, Xu D, Yong YH, Sun W, Zhang H, Cao KJ. Risk factors and outcomes of post-procedure heart blocks after transcatheter device closure of perimembranous ventricular septal defect. *JACC Cardiovasc Interv*. 2012;5:422–427. doi: 10.1016/j.jcin.2012.01.015.
250. Chessa M, Carminati M, Butera G, Bini RM, Drago M, Rosti L, Giamberti A, Pomè G, Bossone E, Frigiola A. Early and late complications associated with transcatheter occlusion of secundum atrial septal defect. *J Am Coll Cardiol*. 2002;39:1061–1065.
251. Abaci A, Unlu S, Alsancak Y, Kaya U, Sezenoz B. Short and long term complications of device closure of atrial septal defect and patent foramen ovale: meta-analysis of 28,142 patients from 203 studies. *Catheter Cardiovasc Interv*. 2013;82:1123–1138. doi: 10.1002/ccd.24875.
252. Wang Y, Hua Y, Li L, Wang X, Qiao L, Shi X, Hua J, Qu Y, Mu D. Risk factors and prognosis of atrioventricular block after atrial septum defect closure using the Amplatzer device. *Pediatr Cardiol*. 2014;35:550–555. doi: 10.1007/s00246-013-0822-1.
253. Lee V, Hemingway H, Harb R, Crake T, Lambiase P. The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review. *Heart*. 2012;98:1290–1298. doi: 10.1136/heartjnl-2012-302005.
254. Cantillon DJ. Evaluation and management of premature ventricular complexes. *Cleve Clin J Med*. 2013;80:377–387. doi: 10.3949/ccjm.80a.12168.
255. Page RL, Joglar JA, Caldwell MA, Calkins H, Conti JB, Deal BJ, Estes NA3, Field ME, Goldberger ZD, Hammill SC, Indik JH, Lindsay BD, Olshansky B, Russo AM, Shen WK, Tracy CM, Al-Khatib SM. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society [published correction appears in *Circulation*. 2016;134:e232–e233]. *Circulation*. 2016;133:e471–e505. doi: 10.1161/CIR.0000000000000310.
256. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC Jr, Conti JB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT, Sacco RL, Stevenson WG, Tchou PJ, Tracy CM, Yancy CW. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the Heart Rhythm Society [published correction appears in *Circulation*. 2014;130:e272–e274]. *Circulation*. 2014;130:e199–e267. doi: 10.1161/CIR.0000000000000041.
257. Lafuente-Lafuente C, Mouly S, Longás-Tejero MA, Mahé I, Bergmann JF. Antiarrhythmic drugs for maintaining sinus rhythm after cardioversion of atrial fibrillation: a systematic review of randomized controlled trials. *Arch Intern Med*. 2006;166:719–728. doi: 10.1001/archinte.166.7.719.
258. You JJ, Singer DE, Howard PA, Lane DA, Eckman MH, Fang MC, Hylek EM, Schulman S, Go AS, Hughes M, Spencer FA, Manning WJ, Halperin JL, Lip GYH. Antithrombotic therapy for atrial fibrillation: antithrombotic therapy and prevention of thrombosis: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(suppl):e531S–e575S.
259. Hauser TH, Pinto DS, Josephson ME, Zimetbaum P. Safety and feasibility of a clinical pathway for the outpatient initiation of antiarrhythmic medications in patients with atrial fibrillation or atrial flutter. *Am J Cardiol*. 2003;91:1437–1441.
260. Menozzi C, Brignole M, Alboni P, Boni L, Paparella N, Gaggioli G, Lolli G. The natural course of untreated sick sinus syndrome and identification of the variables predictive of unfavorable outcome. *Am J Cardiol*. 1998;82:1205–1209.
261. Dhingra RC, Denes P, Wu D, Chuquimia R, Rosen KM. The significance of second degree atrioventricular block and bundle branch block: observations regarding site and type of block. *Circulation*. 1974;49:638–646.
262. Strasberg B, Amat-Y-Leon F, Dhingra RC, Palileo E, Swiryn S, Bauernfeind R, Wyndham C, Rosen KM. Natural history of chronic second-degree atrioventricular nodal block. *Circulation*. 1981;63:1043–1049.
263. Connelly DT, Steinhaus DM. Mobitz type I atrioventricular block: an indication for permanent pacing? *Pacing Clin Electrophysiol*. 1996;19:261–264.
264. Priori SG, Wilde AA, Horie M, Cho Y, Behr ER, Berul C, Blom N, Brugada J, Chiang CE, Huikuri H, Kannankeril P, Krahn A, Leenhardt A, Moss A, Schwartz PJ, Shimizu W, Tomaselli G, Tracy C. HRS/EHRA/APHR expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm*. 2013;10:1932–1963. doi: 10.1016/j.hrthm.2013.05.014.
265. Timmermans C, Smeets JL, Rodriguez LM, Vrochou G, van den Dool A, Wellens HJ. Aborted sudden death in the Wolff-Parkinson-White syndrome. *Am J Cardiol*. 1995;76:492–494.
266. Benditt DG. Syncope risk assessment in the emergency department and clinic. *Prog Cardiovasc Dis*. 2013;55:376–381. doi: 10.1016/j.pcad.2012.10.010.
267. Reed MJ, Newby DE, Coull AJ, Prescott RJ, Jacques KG, Gray AJ. The ROSE (Risk Stratification of Syncope in the Emergency Department) study. *J Am Coll Cardiol*. 2010;55:713–721. doi: 10.1016/j.jacc.2009.09.049.
268. Thiruganasambandamoorthy V, Stiell IG, Sivilotti ML, Murray H, Rowe BH, Lang E, McRae A, Sheldon R, Wells GA. Risk stratification of adult emergency department syncope patients to predict short-term serious outcomes after discharge (RISEDS) study. *BMC Emerg Med*. 2014;14:8. doi: 10.1186/1471-227X-14-8.
269. Thiruganasambandamoorthy V, Hess EP, Turko E, Tran ML, Wells GA, Stiell IG. Defining abnormal electrocardiography in adult emergency department syncope patients: the Ottawa Electrocardiographic Criteria. *CJEM*. 2012;14:248–258.
270. Shen W, Sheldon RS, Benditt DG, Cohen MI, Forman DE, Goldberger ZD, Grubb BP, Hamdan MH, Krahn AD, Link MS, Olshansky B, Raj SR, Sandhu RK, Sorajja D, Sun BC, Yancy CW. 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society. *Circulation*. 2017;136:e60–e122. doi: 10.1161/CIR.0000000000000499.
271. Task Force for the Diagnosis and Management of Syncope; European Society of Cardiology (ESC); European Heart Rhythm Association (EHRA); Heart Failure Association (HFA); Heart Rhythm Society (HRS), Moya A, Sutton R, Ammirati F, Blanc J, Brignole M, Dahm JB, Deharo J, Gajek J, Gjesdal K, Krahn A, Massin M, Pepi M, Pezawas T, Ruiz Granell R, Sarasin F, Ungar A, van Dijk JG, Walma EP, Wieling W. Guidelines for the diagnosis and management of syncope (version 2009). *Eur Heart J*. 2009;30:2631–2671.
272. Mendu ML, McAvay G, Lampert R, Stoehr J, Tinetti ME. Yield of diagnostic tests in evaluating syncope episodes in older patients. *Arch Intern Med*. 2009;169:1299–1305. doi: 10.1001/archinternmed.2009.204.
273. Benezet-Mazuecos J, Ibanez B, Rubio JM, Navarro F, Martín E, Romero J, Farre J. Utility of in-hospital cardiac remote telemetry in patients with unexplained syncope. *Europace*. 2007;9:1196–1201. doi: 10.1093/europace/eum239.
274. Croci F, Brignole M, Alboni P, Menozzi C, Raviele A, Del Rosso A, Dinelli M, Solano A, Bottoni N, Donato P. The application of a standardized strategy of evaluation in patients with syncope referred to three syncope units. *Europace*. 2002;4:351–355.
275. Anderson JB, Czosek RJ, Knilians TK, Marino BS. The effect of paediatric syncope on health-related quality of life. *Cardiol Young*. 2012;22:583–588. doi: 10.1017/S1047951112000133.

276. Friedman KG, Alexander ME. Chest pain and syncope in children: a practical approach to the diagnosis of cardiac disease. *J Pediatr*. 2013;163:896–901.e1–e3. doi: 10.1016/j.jpeds.2013.05.001.
277. Tretter JT, Kavey RE. Distinguishing cardiac syncope from vasovagal syncope in a referral population. *J Pediatr*. 2013;163:1618–1623.e1. doi: 10.1016/j.jpeds.2013.07.023.
278. Bohnen M, Stevenson WG, Tedrow UB, Michaud GF, John RM, Epstein LM, Albert CM, Koplan BA. Incidence and predictors of major complications from contemporary catheter ablation to treat cardiac arrhythmias. *Heart Rhythm*. 2011;8:1661–1666. doi: 10.1016/j.hrthm.2011.05.017.
279. Pelargonio G, Fogel RI, Knilians TK, Prystowsky EN. Late occurrence of heart block after radiofrequency catheter ablation of the septal region: clinical follow-up and outcome. *J Cardiovasc Electrophysiol*. 2001;12:56–60.
280. Cappato R, Calkins H, Chen SA, Davies W, Iesaka Y, Kalman J, Kim YH, Klein G, Natale A, Packer D, Skanes A, Ambrogi F, Biganzoli E. Updated worldwide survey on the methods, efficacy, and safety of catheter ablation for human atrial fibrillation. *Circ Arrhythm Electrophysiol*. 2010;3:32–38. doi: 10.1161/CIRCEP.109.859116.
281. Metzner I, Wissner E, Tilz RR, Rillig A, Mathew S, Schmidt B, Chun J, Wohlmuth P, Deiss S, Lemes C, Maurer T, Fink T, Heeger C, Ouyang F, Kuck KH, Metzner A. Ablation of atrial fibrillation in patients ≥ 75 years: long-term clinical outcome and safety. *Europace*. 2016;18:543–549. doi: 10.1093/europace/euv229.
282. Grimm W, Hoffmann J, Menz V, Maisch B. Transient QT prolongation with torsades de pointes tachycardia after ablation of permanent junctional reciprocating tachycardia. *J Cardiovasc Electrophysiol*. 1999;10:1631–1635.
283. Reithmann C, Fiek M, Beckmann BM, Kääh S. Recurrent torsades de pointes after catheter ablation of incessant ventricular bigeminy in combination with QT prolongation. *Europace*. 2012;14:299–300. doi: 10.1093/europace/eur278.
284. Peters RH, Wever EF, Hauer RN, Wittkampf FH, Robles de Medina EO. Bradycardia dependent QT prolongation and ventricular fibrillation following catheter ablation of the atrioventricular junction with radiofrequency energy. *Pacing Clin Electrophysiol*. 1994;17:108–112.
285. Falk RH, Battinelli NJ. External cardiac pacing using low impedance electrodes suitable for defibrillation: a comparative blinded study. *J Am Coll Cardiol*. 1993;22:1354–1358.
286. Zoll PM, Zoll RH, Falk RH, Clinton JE, Eitel DR, Antman EM. External noninvasive temporary cardiac pacing: clinical trials. *Circulation*. 1985;71:937–944.
287. Betts TR. Regional survey of temporary transvenous pacing procedures and complications. *Postgrad Med J*. 2003;79:463–465.
288. Chihrin SM, Mohammed U, Yee R, Gula LJ, Klein GJ, Skanes AC, Krahn AD. Utility and cost effectiveness of temporary pacing using active fixation leads and an externally placed reusable permanent pacemaker. *Am J Cardiol*. 2006;98:1613–1615. doi: 10.1016/j.amjcard.2006.07.041.
289. Kawata H, Pretorius V, Phan H, Mulpuru S, Gadiyaram V, Patel J, Steltzner D, Krummen D, Feld G, Birgersdotter-Green U. Utility and safety of temporary pacing using active fixation leads and externalized re-usable permanent pacemakers after lead extraction. *Europace*. 2013;15:1287–1291. doi: 10.1093/europace/eut045.
290. Kornberger A, Schmid E, Kalender G, Stock UA, Doernberger V, Khalil M, Lisy M. Bridge to recovery or permanent system implantation: an eight-year single-center experience in transvenous semipermanent pacing. *Pacing Clin Electrophysiol*. 2013;36:1096–1103. doi: 10.1111/pace.12175.
291. Pecha S, Aydin MA, Yildirim Y, Sill B, Reiter B, Wilke I, Reichensperner H, Treede H. Transcutaneous lead implantation connected to an externalized pacemaker in patients with implantable cardiac defibrillator/pacemaker infection and pacemaker dependency. *Europace*. 2013;15:1205–1209. doi: 10.1093/europace/eut031.
292. Rosenfeld LE. Temporary permanent or permanent temporary pacing? *Pacing Clin Electrophysiol*. 2011;34:670–671. doi: 10.1111/j.1540-8159.2011.03086.x.
293. Tsai V, Goldstein MK, Hsia HH, Wang Y, Curtis J, Heidenreich PA; on behalf of the National Cardiovascular Data's ICD Registry. Influence of age on perioperative complications among patients undergoing implantable cardioverter-defibrillators for primary prevention in the United States. *Circ Cardiovasc Qual Outcomes*. 2011;4:549–556. doi: 10.1161/CIRCOUTCOMES.110.959205.
294. van Rees JB, de Bie MK, Thijssen J, Borleffs CJ, Schalij MJ, van Erven L. Implantation-related complications of implantable cardioverter-defibrillators and cardiac resynchronization therapy devices: a systematic review of randomized clinical trials. *J Am Coll Cardiol*. 2011;58:995–1000. doi: 10.1016/j.jacc.2011.06.007.
295. Chauhan A, Grace AA, Newell SA, Stone DL, Shapiro LM, Schofield PM, Petch MC. Early complications after dual chamber versus single chamber pacemaker implantation. *Pacing Clin Electrophysiol*. 1994;17(pt 2):2012–2015.
296. Cheng A, Wang Y, Curtis JP, Varosy PD. Acute lead dislodgements and in-hospital mortality in patients enrolled in the national cardiovascular data registry implantable cardioverter defibrillator registry. *J Am Coll Cardiol*. 2010;56:1651–1656. doi: 10.1016/j.jacc.2010.06.037.
297. Gould PA, Krahn AD; Canadian Heart Rhythm Society Working Group on Device Advisories. Complications associated with implantable cardioverter-defibrillator replacement in response to device advisories. *JAMA*. 2006;295:1907–1911. doi: 10.1001/jama.295.16.1907.
298. Kurtz SM, Ochoa JA, Lau E, Shkolnikov Y, Pavri BB, Frisch D, Greenspon AJ. Implantation trends and patient profiles for pacemakers and implantable cardioverter defibrillators in the United States: 1993–2006. *Pacing Clin Electrophysiol*. 2010;33:705–711. doi: 10.1111/j.1540-8159.2009.02670.x.
299. Chung MK, Szymkiewicz SJ, Shao M, Zishiri E, Niebauer MJ, Lindsay BD, Tchou PJ. Aggregate national experience with the wearable cardioverter-defibrillator: event rates, compliance, and survival. *J Am Coll Cardiol*. 2010;56:194–203. doi: 10.1016/j.jacc.2010.04.016.
300. Coronel R, Wilders R, Verkerk AO, Wiegerinck RF, Benoist D, Bernus O. Electrophysiological changes in heart failure and their implications for arrhythmogenesis. *Biochim Biophys Acta*. 2013;1832:2432–2441. doi: 10.1016/j.bbdis.2013.04.002.
301. Kreuz J, Horlbeck F, Hoyer F, Mellert F, Fimmers R, Lickfett L, Nickenig G, Schwab JO. An impaired renal function: a predictor of ventricular arrhythmias and mortality in patients with nonischemic cardiomyopathy and heart failure. *Pacing Clin Electrophysiol*. 2011;34:894–899. doi: 10.1111/j.1540-8159.2011.03059.x.
302. Benza RL, Tallaj JA, Felker GM, Zabel KM, Kao W, Bourge RC, Pearce D, Leimberger JD, Borzak S, O'Connor CM, Gheorghiadu M; OPTIME-CHF Investigators. The impact of arrhythmias in acute heart failure. *J Card Fail*. 2004;10:279–284.
303. Opasich C, Capomolla S, Riccardi PG, Febo O, Forni G, Cobelli F, Tavazzi L. Does in-patient ECG monitoring have an impact on medical care in chronic heart failure patients? *Eur J Heart Fail*. 2000;2:281–285.
304. Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Bolger AF, Levison ME, Ferrieri P, Gerber MA, Tani LY, Gewitz MH, Tong DC, Steckelberg JM, Baltimore RS, Shulman ST, Burns JC, Falace DA, Newburger JW, Pallasch TJ, Takahashi M, Taubert KA. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for health-care professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association [published corrections appear in *Circulation*. 2005;112:2373, *Circulation*. 2007;115:e408, *Circulation*. 2007;116:e547, *Circulation*. 2008;118:e497]. *Circulation*. 2005;111:e394–e434. doi: 10.1161/CIRCULATIONAHA.105.165564.
305. Meine TJ, Nettles RE, Anderson DJ, Cabell CH, Corey GR, Sexton DJ, Wang A. Cardiac conduction abnormalities in endocarditis defined by the Duke criteria. *Am Heart J*. 2001;142:280–285. doi: 10.1067/mhj.2001.116964.
306. Khalid-de Bakker C, Jonkers D, Hameeteman W, de Ridder R, Masclee A, Stockbrügger R. Cardiopulmonary events during primary colonoscopy screening in an average risk population. *Neth J Med*. 2011;69:186–191.
307. Castro G, Azrak MF, Seeff LC, Royalty J. Outpatient colonoscopy complications in the CDC's Colorectal Cancer Screening Demonstration Program: a prospective analysis. *Cancer*. 2013;119(suppl):2849–2854. doi: 10.1002/cncr.28159.
308. ASGE Technology Committee, Gottlieb KT, Banerjee S, Barth BA, Bhat YM, Desilets DJ, Maple JT, Pfau PR, Pleskow DK, Siddiqui UD, Tokar JL, Wang A, Song LM, Rodriguez SA. Monitoring equipment for endoscopy. *Gastrointest Endosc*. 2013;77:175–180.
309. Roberts-Thomson IC, Teo E, Roberts-Thomson KC. Myocardial ischemia, pancreatitis and ERCP. *J Gastroenterol Hepatol*. 2010;25:1469–1470.
310. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, Davila-Roman VG, Gerhard-Herman MD, Holly TA, Kane GC, Marine JE, Nelson MT, Spencer CC, Thompson A, Ting HH, Uretsky BF, Wijeyesundera DN. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing

- noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130:e278–e333. doi: 10.1161/CIR.000000000000106.
311. Bhave PD, Goldman LE, Vittinghoff E, Maselli J, Auerbach A. Incidence, predictors, and outcomes associated with postoperative atrial fibrillation after major noncardiac surgery. *Am Heart J*. 2012;164:918–924. doi: 10.1016/j.ahj.2012.09.004.
 312. Gialdini G, Nearing K, Bhave PD, Bonuccelli U, Iadecola C, Healey JS, Kamel H. Perioperative atrial fibrillation and the long-term risk of ischemic stroke. *JAMA*. 2014;312:616–622. doi: 10.1001/jama.2014.9143.
 313. Vretzakis G, Simeoforidou M, Stamouli K, Bareka M. Supraventricular arrhythmias after thoracotomy: is there a role for autonomic imbalance? *Anesthesiol Res Pract*. 2013;2013:413985. doi: 10.1155/2013/413985.
 314. Neustein SM, Kahn P, Krellenstein DJ, Cohen E. Incidence of arrhythmias after thoracic surgery: thoracotomy versus video-assisted thoracoscopy. *J Cardiothorac Vasc Anesth*. 1998;12:659–661.
 315. Sohn GH, Shin DH, Byun KM, Han HJ, Cho SJ, Song YB, Kim JH, On YK, Kim JS. The incidence and predictors of postoperative atrial fibrillation after noncardiothoracic surgery. *Korean Circ J*. 2009;39:100–104. doi: 10.4070/kcj.2009.39.3.100.
 316. Ivanovic J, Maziak DE, Ramzan S, McGuire AL, Villeneuve PJ, Gilbert S, Sundaresan RS, Shamji FM, Seely AJ. Incidence, severity and perioperative risk factors for atrial fibrillation following pulmonary resection. *Interact Cardiovasc Thorac Surg*. 2014;18:340–346. doi: 10.1093/icvts/ivt520.
 317. Roselli EE, Murthy SC, Rice TW, Houghtaling PL, Pierce CD, Karchmer DP, Blackstone EH. Atrial fibrillation complicating lung cancer resection. *J Thorac Cardiovasc Surg*. 2005;130:438–444. doi: 10.1016/j.jtcvs.2005.02.010.
 318. Park BJ, Zhang H, Rusch VW, Amar D. Video-assisted thoracic surgery does not reduce the incidence of postoperative atrial fibrillation after pulmonary lobectomy. *J Thorac Cardiovasc Surg*. 2007;133:775–779. doi: 10.1016/j.jtcvs.2006.09.022.
 319. Ryu JK. Postoperative atrial fibrillation after noncardiothoracic surgery: is it different from after cardiothoracic surgery? *Korean Circ J*. 2009;39:93–94. doi: 10.4070/kcj.2009.39.3.93.
 320. Oka T, Ozawa Y, Ohkubo Y. Thoracic epidural bupivacaine attenuates supraventricular tachyarrhythmias after pulmonary resection. *Anesth Analg*. 2001;93:253–259.
 321. Landesberg G, Einav S, Christopherson R, Beattie C, Berlatzky Y, Rosenfeld B, Eidelman LA, Norris E, Anner H, Mosseri M, Cotev S, Luria MH. Perioperative ischemia and cardiac complications in major vascular surgery: importance of the preoperative twelve-lead electrocardiogram. *J Vasc Surg*. 1997;26:570–578.
 322. Estrada CA, Prasad NK, Rosman HS, Young MJ. Outcomes of patients hospitalized to a telemetry unit. *Am J Cardiol*. 1994;74:357–362.
 323. Estrada CA, Rosman HS, Prasad NK, Battilana G, Alexander M, Held AC, Young MJ. Role of telemetry monitoring in the non-intensive care unit. *Am J Cardiol*. 1995;76:960–965.
 324. Schull MJ, Redelmeier DA. Continuous electrocardiographic monitoring and cardiac arrest outcomes in 8,932 telemetry ward patients. *Acad Emerg Med*. 2000;7:647–652.
 325. Peterson MC, Whetten DK, Renlund DG, Coletti A. Sensitivity of rhythm disturbance detection by community hospital telemetry. *Ann Noninvasive Electrocardiol*. 2002;7:219–221.
 326. Najafi N, Auerbach A. Use and outcomes of telemetry monitoring on a medicine service. *Arch Intern Med*. 2012;172:1349–1350. doi: 10.1001/archinternmed.2012.3163.
 327. Hillman KM, Bristow PJ, Chey T, Daffurn K, Jacques T, Norman SL, Bishop GF, Simmons G. Antecedents to hospital deaths. *Intern Med J*. 2001;31:343–348.
 328. Vetro J, Natarajan DK, Mercer I, Buckmaster JN, Heland M, Hart GK, Bellomo R, Jones DA. Antecedents to cardiac arrests in a hospital equipped with a medical emergency team. *Crit Care Resusc*. 2011;13:162–166.
 329. Stevenson JE, Israels J, Nilsson GC, Petersson GI, Bath PA. Recording signs of deterioration in acute patients: the documentation of vital signs within electronic health records in patients who suffered in-hospital cardiac arrest. *Health Informatics J*. 2016;22:21–33. doi: 10.1177/1460458214530136.
 330. Jauch EC, Saver JL, Adams HP Jr, Bruno A, Connors JJ, Demaerschalk BM, Khatri P, McMullan PW Jr, Qureshi AI, Rosenfield K, Scott PA, Summers DR, Wang DZ, Wintermark M, Yonas H; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013;44:870–947. doi: 10.1161/STR.0b013e318284056a.
 331. Easton JD, Saver JL, Albers GW, Albers MJ, Chaturvedi S, Feldmann E, Hatsukami TS, Higashida RT, Johnston SC, Kidwell CS, Lutsep HL, Miller E, Sacco RL. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. *Stroke*. 2009;40:2276–2293. doi: 10.1161/STROKEAHA.108.192218.
 332. Kallmünzer B, Breuer L, Kahl N, Bobinger T, Raaz-Schrauder D, Huttner HB, Schwab S, Köhrmann M. Serious cardiac arrhythmias after stroke: incidence, time course, and predictors—a systematic, prospective analysis. *Stroke*. 2012;43:2892–2897. doi: 10.1161/STROKEAHA.112.664318.
 333. Ritter MA, Rohde A, Heuschmann PU, Dziewas R, Stypmann J, Nabavi DG, Ringelstein BE. Heart rate monitoring on the stroke unit: what does heart beat tell about prognosis? An observational study. *BMC Neurol*. 2011;11:47. doi: 10.1186/1471-2377-11-47.
 334. Miller DJ, Khan MA, Schultz LR, Simpson JR, Katramados AM, Russman AN, Mitsias PD. Outpatient cardiac telemetry detects a high rate of atrial fibrillation in cryptogenic stroke. *J Neurol Sci*. 2013;324:57–61. doi: 10.1016/j.jns.2012.10.001.
 335. Dimant J, Grob D. Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents. *Stroke*. 1977;8:448–455.
 336. Norris JW, Froggatt GM, Hachinski VC. Cardiac arrhythmias in acute stroke. *Stroke*. 1978;9:392–396.
 337. Goldstein DS. The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings. *Stroke*. 1979;10:253–259.
 338. Myers MG, Norris JW, Hachinski VC, Weingert ME, Sole MJ. Cardiac sequelae of acute stroke. *Stroke*. 1982;13:838–842.
 339. Hachinski VC. The clinical problem of brain and heart. *Stroke*. 1993;24(suppl):11–12.
 340. Khechinashvili G, Asplund K. Electrocardiographic changes in patients with acute stroke: a systematic review. *Cerebrovasc Dis*. 2002;14:67–76.
 341. Jensen JK, Bak S, Flemming Høiland-Carlsen P, Mickley H. Prevalence of electrocardiographic ST-T changes during acute ischemic stroke in patients without known ischemic heart disease. *Int J Cardiol*. 2008;128:137–138. doi: 10.1016/j.ijcard.2007.05.055.
 342. Christensen H, Fogh Christensen A, Boysen G. Abnormalities on ECG and telemetry predict stroke outcome at 3 months. *J Neurol Sci*. 2005;234:99–103. doi: 10.1016/j.jns.2005.03.039.
 343. Sulter G, Elting JW, Langedijk M, Maurits NM, De Keyser J. Admitting acute ischemic stroke patients to a stroke care monitoring unit versus a conventional stroke unit: a randomized pilot study. *Stroke*. 2003;34:101–104.
 344. McDermott MM, Lefevre F, Arron M, Martin GJ, Biller J. ST segment depression detected by continuous electrocardiography in patients with acute ischemic stroke or transient ischemic attack. *Stroke*. 1994;25:1820–1824.
 345. Yamour BJ, Sridharan MR, Rice JF, Flowers NC. Electrocardiographic changes in cerebrovascular hemorrhage. *Am Heart J*. 1980;99:294–300.
 346. Ramani A, Shetty U, Kundaje GN. Electrocardiographic abnormalities in cerebrovascular accidents. *Angiology*. 1990;41(pt 1):681–686. doi: 10.1177/000331979004100902.
 347. Chua HC, Sen S, Cosgriff RF, Gerstenblith G, Beauchamp NJ Jr, Oppenheimer SM. Neurogenic ST depression in stroke. *Clin Neurophysiol*. 1999;101:44–48.
 348. Ay H, Arsava EM, Saribaş O. Creatine kinase-MB elevation after stroke is not cardiac in origin: comparison with troponin T levels. *Stroke*. 2002;33:286–289.
 349. Chalela JA, Ezzeddine MA, Davis L, Warach S. Myocardial injury in acute stroke: a troponin I study. *Neurocrit Care*. 2004;1:343–346. doi: 10.1385/NCC:1.3.343.
 350. Corrales-Medina VF, Suh KN, Rose G, Chirinos JA, Doucette S, Cameron DW, Ferguson DA. Cardiac complications in patients with community-acquired pneumonia: a systematic review and meta-analysis of observational studies. *PLoS Med*. 2011;8:e1001048. doi: 10.1371/journal.pmed.1001048.

351. Aliberti S, Ramirez JA. Cardiac diseases complicating community-acquired pneumonia. *Curr Opin Infect Dis*. 2014;27:295–301. doi: 10.1097/QCO.0000000000000055.
352. Carr GE, Yuen TC, McConville JF, Kress JP, VandenHoek TL, Hall JB, Edelson DP; American Heart Association's Get With the Guidelines-Resuscitation (National Registry of CPR) Investigators. Early cardiac arrest in patients hospitalized with pneumonia: a report from the American Heart Association's Get With The Guidelines-Resuscitation Program. *Chest*. 2012;141:1528–1536. doi: 10.1378/chest.11-1547.
353. Griffin AT, Wiemken TL, Arnold FW. Risk factors for cardiovascular events in hospitalized patients with community-acquired pneumonia. *Int J Infect Dis*. 2013;17:e1125–e1129. doi: 10.1016/j.ijid.2013.07.005.
354. Soto-Gomez N, Anzueto A, Waterer GW, Restrepo MI, Mortensen EM. Pneumonia: an arrhythmogenic disease? *Am J Med*. 2013;126:43–48. doi: 10.1016/j.amjmed.2012.08.005.
355. Pery TW, Pugh MJ, Waterer GW, Nakashima B, Orihuela CJ, Copeland LA, Restrepo MI, Anzueto A, Mortensen EM. Incidence of cardiovascular events after hospital admission for pneumonia. *Am J Med*. 2011;124:244–251. doi: 10.1016/j.amjmed.2010.11.014.
356. Viasus D, Garcia-Vidal C, Manresa F, Dorca J, Gudiol F, Carratalà J. Risk stratification and prognosis of acute cardiac events in hospitalized adults with community-acquired pneumonia. *J Infect*. 2013;66:27–33. doi: 10.1016/j.jinf.2012.09.003.
357. Theofilogiannakos EK, Anogeianaki A, Tsekoura P, Glouftsiou P, Ilonidis G, Hatzitolios A, Anogianakis G. Arrhythmogenesis in patients with stable chronic obstructive pulmonary disease. *J Cardiovasc Med (Hagerstown)*. 2008;9:89–93. doi: 10.2459/JCM.0b013e328028fe73.
358. Lacasse M, Maltais F, Poirier P, Lacasse Y, Marquis K, Jobin J, LeBlanc P. Post-exercise heart rate recovery and mortality in chronic obstructive pulmonary disease. *Respir Med*. 2005;99:877–886. doi: 10.1016/j.rmed.2004.11.012.
359. Rutten FH, Cramer MJ, Grobbee DE, Sachs AP, Kirkels JH, Lammers JW, Hoes AW. Unrecognized heart failure in elderly patients with stable chronic obstructive pulmonary disease. *Eur Heart J*. 2005;26:1887–1894. doi: 10.1093/eurheartj/ehi291.
360. Brekke PH, Omland T, Smith P, Søyseth V. Underdiagnosis of myocardial infarction in COPD - Cardiac Infarction Injury Score (CIIS) in patients hospitalised for COPD exacerbation. *Respir Med*. 2008;102:1243–1247. doi: 10.1016/j.rmed.2008.04.010.
361. Finkelstein J, Cha E, Scharf SM. Chronic obstructive pulmonary disease as an independent risk factor for cardiovascular morbidity. *Int J Chron Obstruct Pulmon Dis*. 2009;4:337–349.
362. Konecny T, Park JY, Somers KR, Konecny D, Orban M, Soucek F, Parker KO, Scanlon PD, Asirvatham SJ, Brady PA, Rihal CS. Relation of chronic obstructive pulmonary disease to atrial and ventricular arrhythmias. *Am J Cardiol*. 2014;114:272–277. doi: 10.1016/j.amjcard.2014.04.030.
363. Vestbo J, Hurd SS, Agustí AG, Jones PW, Vogelmeier C, Anzueto A, Barnes PJ, Fabbri LM, Martinez FJ, Nishimura M, Stockley RA, Sin DD, Rodriguez-Roisin R. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *Am J Respir Crit Care Med*. 2013;187:347–365. doi: 10.1164/rccm.201204-0596PP.
364. Thomas AJ, Apiyasawat S, Spodick DH. Electrocardiographic detection of emphysema. *Am J Cardiol*. 2011;107:1090–1092. doi: 10.1016/j.amjcard.2010.11.039.
365. Holtzman D, Aronow WS, Mellana WM, Sharma M, Mehta N, Lim J, Chandy D. Electrocardiographic abnormalities in patients with severe versus mild or moderate chronic obstructive pulmonary disease followed in an academic outpatient pulmonary clinic. *Ann Noninvasive Electrocardiol*. 2011;16:30–32. doi: 10.1111/j.1542-474X.2010.00404.x.
366. Lazović B, Svenda MZ, Mazić S, Stajić Z, Delić M. Analysis of electrocardiogram in chronic obstructive pulmonary disease patients. *Med Pregl*. 2013;66:126–129.
367. Vanfleteren LE, Franssen FM, Uszko-Lencer NH, Spruit MA, Celis M, Gorgels AP, Wouters EF. Frequency and relevance of ischemic electrocardiographic findings in patients with chronic obstructive pulmonary disease. *Am J Cardiol*. 2011;108:1669–1674. doi: 10.1016/j.amjcard.2011.07.027.
368. El-Sherif N, Turitto G. Electrolyte disorders and arrhythmogenesis. *Cardiol J*. 2011;18:233–245.
369. Pepin J, Shields C. Advances in diagnosis and management of hypokalemic and hyperkalemic emergencies. *Emerg Med Pract*. 2012;14:1–17; quiz 17–18.
370. Medford-Davis L, Rafique Z. Derangements of potassium. *Emerg Med Clin North Am*. 2014;32:329–347. doi: 10.1016/j.emc.2013.12.005.
371. Alfonso AV, Isles C, Geddes C, Deighan C. Potassium disorders: clinical spectrum and emergency management. *Resuscitation*. 2006;70:10–25. doi: 10.1016/j.resuscitation.2005.11.002.
372. Vanden Hoek TL, Morrison LJ, Shuster M, Donnino M, Sinz E, Lavonas EJ, Jeejeebhoy FM, Gabrielli A. Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [published corrections appear in *Circulation*. 2011;124:e405 and *Circulation*. 2011;123:e239]. *Circulation*. 2010;122(suppl3):S829–S861. doi:10.1161/CIRCULATIONAHA.110.971069.
373. Freeman K, Feldman JA, Mitchell P, Donovan J, Dyer KS, Eliseo L, White LF, Temin ES. Effects of presentation and electrocardiogram on time to treatment of hyperkalemia. *Acad Emerg Med*. 2008;15:239–249. doi: 10.1111/j.1553-2712.2008.00058.x.
374. Webster A, Brady W, Morris F. Recognising signs of danger: ECG changes resulting from an abnormal serum potassium concentration. *Emerg Med J*. 2002;19:74–77.
375. Chon SB, Kwak YH, Hwang SS, Oh WS, Bae JH. Severe hyperkalemia can be detected immediately by quantitative electrocardiography and clinical history in patients with symptomatic or extreme bradycardia: a retrospective cross-sectional study [published correction appears in *J Crit Care*. 2014;29:310]. *J Crit Care*. 2013;28:1112.e7–1112.e13. doi: 10.1016/j.jccr.2013.08.013.
376. Mohanlal V, Haririan A, Weinman EJ. Bradycardia without “classical” EKG changes in hyperkalemic hemodialysis patients. *Clin Nephrol*. 2013;80:464–468. doi: 10.5414/CN107529.
377. Martinez-Vea A, Bardají A, Garcia C, Oliver JA. Severe hyperkalemia with minimal electrocardiographic manifestations: a report of seven cases. *J Electrocardiol*. 1999;32:45–49.
378. Keren A, Tzivoni D. Torsades de pointes: prevention and therapy. *Cardiovasc Drugs Ther*. 1991;5:509–513.
379. Ceremuzyński L, Gebalska J, Wolk R, Makowska E. Hypomagnesemia in heart failure with ventricular arrhythmias: beneficial effects of magnesium supplementation. *J Intern Med*. 2000;247:78–86.
380. Falco CN, Grupi C, Sosa E, Scanavacca M, Hachul D, Lara S, Sacilotto L, Pisani CF, Ramirez JA, Darrieux F. Successful improvement of frequency and symptoms of premature complexes after oral magnesium administration [published correction appears in *Arq Bras Cardiol*. 2013;100:488]. *Arq Bras Cardiol*. 2012;98:480–487.
381. Smith JM, Lowe RF, Fullerton J, Currie SM, Harris L, Felker-Kantor E. An integrative review of the side effects related to the use of magnesium sulfate for pre-eclampsia and eclampsia management. *BMC Pregnancy Childbirth*. 2013;13:34.
382. Buckley NA, Chevalier S, Leditschke IA, O'Connell DL, Leitch J, Pond SM. The limited utility of electrocardiography variables used to predict arrhythmia in psychotropic drug overdose. *Crit Care*. 2003;7:R101–R107. doi: 10.1186/cc2345.
383. Berling I, Whyte IM, Isbister GK. Oxycodone overdose causes naloxone responsive coma and QT prolongation. *QJM*. 2013;106:35–41. doi: 10.1093/qjmed/hcs176.
384. Butler B, Rubin G, Lawrence A, Batey R, Bell J. Estimating the risk of fatal arrhythmia in patients in methadone maintenance treatment for heroin addiction. *Drug Alcohol Rev*. 2011;30:173–180. doi: 10.1111/j.1465-3362.2010.00213.x.
385. Haning W, Goebert D. Electrocardiographic abnormalities in methamphetamine abusers. *Addiction*. 2007;102(suppl):70–75. doi: 10.1111/j.1360-0443.2006.01776.x.
386. Wallner C, Stöllberger C, Hlavín A, Finsterer J, Hager I, Hermann P. Electrocardiographic abnormalities in opiate addicts. *Addiction*. 2008;103:1987–1993. doi: 10.1111/j.1360-0443.2008.02333.x.
387. Phillips KA, Hirsch GA, Epstein DH, Preston KL. Cardiac complications of unwitting co-injection of quinine/quinidine with heroin in an intravenous drug user. *J Gen Intern Med*. 2012;27:1722–1725. doi: 10.1007/s11606-012-2089-2.
388. Bass M. Sudden sniffing death. *JAMA*. 1970;212:2075–2079.
389. Anderson CE, Loomis GA. Recognition and prevention of inhalant abuse. *Am Fam Physician*. 2003;68:869–874.
390. McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, Gibler WB, Ohman EM, Drew B, Philippides G, Newby LK. Management of cocaine-associated chest pain and myocardial infarction: a scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. *Circulation*. 2008;117:1897–1907. doi: 10.1161/CIRCULATIONAHA.107.188950.
391. Levin KH, Copersino ML, Epstein D, Boyd SJ, Gorelick DA. Longitudinal ECG changes in cocaine users during extended abstinence. *Drug Alcohol Depend*. 2008;95:160–163. doi: 10.1016/j.drugalcdep.2007.12.001.

392. Bauman JL, Grawe JJ, Winecoff AP, Hariman RJ. Cocaine-related sudden cardiac death: a hypothesis correlating basic science and clinical observations. *J Clin Pharmacol*. 1994;34:902–911.
393. Hollander JE, Hoffman RS, Gennis P, Fairweather P, DiSano MJ, Schumb DA, Feldman JA, Fish SS, Dyer S, Wax P, Whelan C, Schwarzwald E. Prospective multicenter evaluation of cocaine-associated chest pain: Cocaine Associated Chest Pain (COCHPA) Study Group. *Acad Emerg Med*. 1994;1:330–339.
394. Feldman JA, Fish SS, Beshansky JR, Griffith JL, Woolard RH, Selker HP. Acute cardiac ischemia in patients with cocaine-associated complaints: results of a multicenter trial. *Ann Emerg Med*. 2000;36:469–476. doi: 10.1067/mem.2000.110994.
395. Weber JE, Shofer FS, Larkin GL, Kalaria AS, Hollander JE. Validation of a brief observation period for patients with cocaine-associated chest pain. *N Engl J Med*. 2003;348:510–517. doi: 10.1056/NEJMoa022206.
396. Greene SL, Dargan PI, O'Connor N, Jones AL, Kerins M. Multiple toxicity from 3,4-methylenedioxymethamphetamine (“ecstasy”). *Am J Emerg Med*. 2003;21:121–124. doi: 10.1053/ajem.2003.50028.
397. Maraj S, Figueredo VM, Lynn Morris D. Cocaine and the heart. *Clin Cardiol*. 2010;33:264–269. doi: 10.1002/clc.20746.
398. Lenz J, Brown J, Flagg S, Oh R, Batts K, Ditzler T, Johnson J. Cristalius: a case in designer drugs. *Mil Med*. 2013;178:e893–e895. doi: 10.7205/MILMED-D-12-00393.
399. Paillet-Loilier M, Cesbron A, Le Boisselier R, Bourguine J, Debruyne D. Emerging drugs of abuse: current perspectives on substituted cathinones. *Subst Abuse Rehabil*. 2014;5:37–52. doi: 10.2147/SAR.S37257.
400. Munir VL, Hutton JE, Harney JP, Buykx P, Weiland TJ, Dent AW. Gamma-hydroxybutyrate: a 30 month emergency department review. *Emerg Med Australas*. 2008;20:521–530. doi: 10.1111/j.1742-6723.2008.01140.x.
401. K/DOQI Workgroup. K/DOQI clinical practice guidelines for cardiovascular disease in dialysis patients. *Am J Kidney Dis*. 2005;45(suppl 3):S7–S153.
402. Nishi K, Fujimoto S, Hisanaga S, Ogawa O, Kitamura K. Electrocardiographic assessment of incident atrial fibrillation in hemodialysis patients. *Ther Apher Dial*. 2013;17:16–23. doi: 10.1111/j.1744-9987.2012.01137.x.
403. Buiten MS, de Bie MK, Rotmans JJ, Gabreëls BA, van Dorp W, Wolterbeek R, Trines SA, Schaliij MJ, Jukema JW, Rabelink TJ, van Erven L. The dialysis procedure as a trigger for atrial fibrillation: new insights in the development of atrial fibrillation in dialysis patients. *Heart*. 2014;100:685–690. doi: 10.1136/heartjnl-2013-305417.
404. Bignotto LH, Kállás ME, Djouki RJ, Sasaki MM, Voss GO, Soto CL, Frattini F, Medeiros FS. Electrocardiographic findings in chronic hemodialysis patients. *J Bras Nefrol*. 2012;34:235–242.
405. Hage FG, de Mattos AM, Khamash H, Mehta S, Warnock D, Iskandrian AE. QT prolongation is an independent predictor of mortality in end-stage renal disease. *Clin Cardiol*. 2010;33:361–366. doi: 10.1002/clc.20768.
406. Flythe JE, Lacson E Jr. Outcomes after the long interdialytic break: implications for the dialytic prescription. *Semin Dial*. 2012;25:1–8. doi: 10.1111/j.1525-139X.2011.01015.x.
407. Midwest Bioethics Center, Kansas City Area Ethics Committee Consortium. Honoring do-not-resuscitate (DNR) orders during invasive procedures. *Bioethics Forum*. 1998;14:23–26.
408. ECRI Institute. 2015 Top 10 health technology hazards for 2015. <https://www.ecri.org/Pages/2015-Hazards.aspx>. Accessed August 11, 2017.
409. Joint Commission on Accreditation of Healthcare Organizations. The Joint Commission Announces 2014 national patient safety goal. *Jt Comm Perspect*. 2013;33:1, 3–4.
410. Siebig S, Kuhls S, Imhoff M, Gather U, Schölmerich J, Wrede CE. Intensive care unit alarms: how many do we need? *Crit Care Med*. 2010;38:451–456. doi: 10.1097/CCM.0b013e3181cb0888.
411. Schmid F, Goepfert MS, Kuhnt D, Eichhorn V, Diedrichs S, Reichenspurner H, Goetz AE, Reuter DA. The wolf is crying in the operating room: patient monitor and anesthesia workstation alarming patterns during cardiac surgery. *Anesth Analg*. 2011;112:78–83. doi: 10.1213/ANE.0b013e3181fcc504.
412. Inokuchi R, Sato H, Nanjo Y, Echigo M, Tanaka A, Ishii T, Matsubara T, Doi K, Gunshin M, Hiruma T, Nakamura K, Shinohara K, Kitsuta Y, Nakajima S, Umezu M, Yahagi N. The proportion of clinically relevant alarms decreases as patient clinical severity decreases in intensive care units: a pilot study. *BMJ Open*. 2013;3:e003354. doi: 10.1136/bmjopen-2013-003354.
413. Deleted in proof.
414. Bliss JP, Dunn MC. Behavioural implications of alarm mistrust as a function of task workload. *Ergonomics*. 2000;43:1283–1300. doi: 10.1080/001401300421743.
415. Bitan Y, Meyer J, Shinar D, Zmora E. Nurses' reaction to alarms in a neonatal intensive care unit. *Cogn Tech Work*. 2004;6:239–246.
416. Bustamante EA, Bliss JP, Anderson BL. Effects of varying the threshold of alarm systems and workload on human performance. *Ergonomics*. 2007;50:1127–1147. doi: 10.1080/00140130701237345.
417. Korniewicz DM, Clark T, David Y. A national online survey on the effectiveness of clinical alarms. *Am J Crit Care*. 2008;17:36–41.
418. Funk M, Clark JT, Bauld TJ, Ott JC, Coss P. Attitudes and practices related to clinical alarms. *Am J Crit Care*. 2014;23:e9–e18. doi: 10.4037/ajcc2014315.
419. Bridi AC, da Silva RC, de Farias CC, Franco AS, dos Santos Vde L. Reaction time of a health care team to monitoring alarms in the intensive care unit: implications for the safety of seriously ill patients [in Portuguese]. *Rev Bras Ter Intensiva*. 2014;26:28–35.
420. Bonafide CP, Lin R, Zander M, Graham CS, Paine CW, Rock W, Rich A, Roberts KE, Fortino M, Nadkarni VM, Localio AR, Keren R. Association between exposure to nonactionable physiologic monitor alarms and response time in a children's hospital. *J Hosp Med*. 2015;10:345–351. doi: 10.1002/jhm.2331.
421. Aboukhalil A, Nielsen L, Saeed M, Mark RG, Clifford GD. Reducing false alarm rates for critical arrhythmias using the arterial blood pressure waveform. *J Biomed Inform*. 2008;41:442–451. doi: 10.1016/j.jbi.2008.03.003.
422. Gross B, Dahl D, Nielsen L. Physiologic monitoring alarm load on medical/surgical floors of a community hospital. *Biomed Instrum Technol*. 2011(suppl):29–36. doi: 10.2345/0899-8205-45.s1.29.
423. Görges M, Markewitz BA, Westenskow DR. Improving alarm performance in the medical intensive care unit using delays and clinical context. *Anesth Analg*. 2009;108:1546–1552. doi: 10.1213/ane.0b013e31819bdfbb.
424. Imhoff M, Kuhls S. Alarm algorithms in critical care monitoring. *Anesth Analg*. 2006;102:1525–1537. doi: 10.1213/01.ane.0000204385.01983.61.
425. Nouria K, Trabelsi A. Intelligent monitoring system for intensive care units. *J Med Syst*. 2012;36:2309–2318. doi: 10.1007/s10916-011-9698-x.
426. Wee AN, Sanderson PM. Are melodic medical equipment alarms easily learned? *Anesth Analg*. 2008;106:501–508, table of contents. doi: 10.1213/01.ane.0000286148.58823.6c.
427. Edworthy J, Page R, Hibbard A, Kyle S, Ratnage P, Claydon S. Learning three sets of alarms for the same medical functions: a perspective on the difficulty of learning alarms specified in an international standard. *Appl Ergon*. 2014;45:1291–1296. doi: 10.1016/j.apergo.2013.10.003.
428. Hoglund D, Elms J. The use of mobile devices to improve alarm systems. *Biomed Instrum Technol*. 2011;45:381–384. doi: 10.2345/0899-8205-45.5.381.
429. Moorman BA, Gee T. Functional basics of third-party alarm notification systems. *Biomed Instrum Technol*. 2011(suppl):76–82. doi: 10.2345/0899-8205-45.s1.76.
430. Cvach MM, Frank RJ, Doyle P, Stevens ZK. Use of pagers with an alarm escalation system to reduce cardiac monitor alarm signals. *J Nurs Care Qual*. 2014;29:9–18. doi: 10.1097/NCQ.0b013e3182a61887.
431. Funk M, Parkosewich JA, Johnson CR, Stukshis I. Effect of dedicated monitor watchers on patients' outcomes. *Am J Crit Care*. 1997;6:318–323.
432. Funk M, Ruppel H, Blake N, Phillips J. Research: use of monitor watchers in hospitals: characteristics, training, and practices. *Biomed Instrum Technol*. 2016;50:428–438. doi: 10.2345/0899-8205-50.6.428.
433. Bonzheim KA, Gebara RI, O'Hare BM, Ellis RD, Brand MA, Balar SD, Stockman R, Sciberras AM, Haines DE. Communication strategies and timeliness of response to life critical telemetry alarms. *Telemed J E Health*. 2011;17:241–246. doi: 10.1089/tmj.2010.0139.
434. Segall N, Hobbs G, Granger CB, Anderson AE, Bonifacio AS, Taekman JM, Wright MC. Patient load effects on response time to critical arrhythmias in cardiac telemetry: a randomized trial. *Crit Care Med*. 2015;43:1036–1042. doi: 10.1097/CCM.0000000000000923.
435. Cvach MM, Biggs M, Rothwell KJ, Charles-Hudson C. Daily electrode change and effect on cardiac monitor alarms: an evidence-based practice approach. *J Nurs Care Qual*. 2013;28:265–271. doi: 10.1097/NCQ.0b013e31827993bc.
436. Dandoy CE, Davies SM, Flesch L, Hayward M, Koons C, Coleman K, Jacobs J, McKenna LA, Olomajeye A, Olson C, Powers J, Shoemaker K, Jodele S, Alessandrini E, Weiss B. A team-based approach to reducing cardiac monitor alarms. *Pediatrics*. 2014;134:e1686–e1694. doi: 10.1542/peds.2014-1162.

437. Sendelbach S, Wahl S, Anthony A, Shotts P. Stop the noise: a quality improvement project to decrease electrocardiographic nuisance alarms. *Crit Care Nurse*. 2015;35:15–22; quiz 1p following 22. doi: 10.4037/ccn2015858.
438. AAMI Foundation HTSI. Safety innovations: using data to drive alarm system improvement. http://s3.amazonaws.com/rdcms-aami/files/production/public/FileDownloads/HTSI/Johns_Hopkins_White_Paper.pdf. Accessed June 10, 2014.
439. Deleted in proof.
440. Whalen DA, Covelle PM, Piepenbrink JC, Villanova KL, Cuneo CL, Awtry EH. Novel approach to cardiac alarm management on telemetry units. *J Cardiovasc Nurs*. 2014;29:E13–E22. doi: 10.1097/JCN.0000000000000114.
441. Phillips J. Clinical alarms: complexity and common sense. *Crit Care Nurs Clin North Am*. 2006;18:145–156, ix. doi: 10.1016/j.ccell.2006.01.002.
442. Edworthy J. Medical audible alarms: a review. *J Am Med Inform Assoc*. 2013;20:584–589. doi: 10.1136/amiajnl-2012-001061.
443. Bonafide CP, Localio AR, Holmes JH, Nadkarni VM, Stemler S, MacMurchy M, Zander M, Roberts KE, Lin R, Keren R. Video analysis of factors associated with response time to physiologic monitor alarms in a children's hospital. *JAMA Pediatr*. 2017;171:524–531. doi: 10.1001/jamapediatrics.2016.5123.
444. Adams-Hamoda MG, Caldwell MA, Stotts NA, Drew BJ. Factors to consider when analyzing 12-lead electrocardiograms for evidence of acute myocardial ischemia. *Am J Crit Care*. 2003;12:9–16; quiz 17–18.
445. Patel SI, Souter MJ. Equipment-related electrocardiographic artifacts: causes, characteristics, consequences, and correction. *Anesthesiology*. 2008;108:138–148. doi: 10.1097/01.anes.0000296537.62905.25.
446. Melendez LA, Pino RM. Electrocardiogram interference: a thing of the past? *Biomed Instrum Technol*. 2012;46:470–477. doi: 10.2345/0899-8205-46.6.470.
447. Drew BJ, Funk M. Practice standards for ECG monitoring in hospital settings: executive summary and guide for implementation. *Crit Care Nurs Clin North Am*. 2006;18:157–168, ix. doi: 10.1016/j.ccell.2006.01.007.
448. Albert NM, Murray T, Bena JF, Slicak E, Roach JD, Spence J, Burkle A. Differences in alarm events between disposable and reusable electrocardiography lead wires. *Am J Crit Care*. 2015;24:67–73; quiz 74. doi: 10.4037/ajcc2015663.
449. Benjamin EM, Klugman RA, Luckmann R, Fairchild DG, Abookire SA. Impact of cardiac telemetry on patient safety and cost. *Am J Manag Care*. 2013;19:e225–e232.
450. Feder S, Funk M. Over-monitoring and alarm fatigue: for whom do the bells toll? *Heart Lung*. 2013;42:395–396. doi: 10.1016/j.hrtlng.2013.09.001.
451. Funk M, Winkler CG, May JL, Stephens K, Fennie KP, Rose LL, Turkman YE, Drew BJ. Unnecessary arrhythmia monitoring and underutilization of ischemia and QT interval monitoring in current clinical practice: baseline results of the Practical Use of the Latest Standards for Electrocardiography trial. *J Electrocardiol*. 2010;43:542–547. doi: 10.1016/j.jelectrocard.2010.07.018.
452. Bubb C. A timely practice; a nurse-driven telemetry discontinuation protocol. *Pa Nurse*. 2011;66:6–10; quiz 10.
453. Kansara P, Jackson K, Dressler R, Weiner H, Kerzner R, Weintraub WS, Doorey A. Potential of missing life-threatening arrhythmias after limiting the use of cardiac telemetry. *JAMA Intern Med*. 2015;175:1416–1418. doi: 10.1001/jamainternmed.2015.2387.
454. Cvach M. Monitor alarm fatigue: an integrative review. *Biomed Instrum Technol*. 2012;46:268–277. doi: 10.2345/0899-8205-46.4.268.
455. Rosman EC, Blaufox AD, Menco A, Trope R, Seiden HS. What are we missing? Arrhythmia detection in the pediatric intensive care unit. *J Pediatr*. 2013;163:511–514. doi: 10.1016/j.jpeds.2013.01.053.
456. Ahlborn V, Bohnhorst B, Peter CS, Poets CF. False alarms in very low birthweight infants: comparison between three intensive care monitoring systems. *Acta Paediatr*. 2000;89:571–576.
457. Pelter MM. Electrocardiographic monitoring in the medical-surgical setting: clinical implications, basis, lead configurations, and nursing implications. *Medsurg Nurs*. 2008;17:421–428.
458. Pickham D, Shinn JA, Chan GK, Funk M, Drew BJ. Quasi-experimental study to improve nurses' QT-interval monitoring: results of QTIP study. *Am J Crit Care*. 2012;21:195–200; quiz 201. doi: 10.4037/ajcc2012245.
459. Goodridge E, Furst C, Herrick J, Song J, Tipton PH. Accuracy of cardiac rhythm interpretation by medical-surgical nurses: a pilot study. *J Nurses Prof Dev*. 2013;29:35–40. doi: 10.1097/NND.0b013e31827d0c4f.
460. Salerno SM, Alguire PC, Waxman HS. Competency in interpretation of 12-lead electrocardiograms: a summary and appraisal of published evidence. *Ann Intern Med*. 2003;138:751–760.
461. Sur DK, Kaye L, Mikus M, Goad J, Morena A. Accuracy of electrocardiogram reading by family practice residents. *Fam Med*. 2000;32:315–319.
462. Viskin S, Rosovski U, Sands AJ, Chen E, Kistler PM, Kalman JM, Rodriguez Chavez L, Iturralde Torres P, Cruz FFE, Centurion OA, Fujiki A, Maury P, Chen X, Krahn AD, Roithinger F, Zhang L, Vincent GM, Zeltser D. Inaccurate electrocardiographic interpretation of long QT: the majority of physicians cannot recognize a long QT when they see one. *Heart Rhythm*. 2005;2:569–574. doi: 10.1016/j.hrthm.2005.02.011.
463. Novotny T, Bond RR, Andrsova I, Koc L, Sisakova M, Finlay DD, Guldenring D, Spinar J, Malik M. Data analysis of diagnostic accuracies in 12-lead electrocardiogram interpretation by junior medical fellows. *J Electrocardiol*. 2015;48:988–994. doi: 10.1016/j.jelectrocard.2015.08.023.
464. Sandau KE, Sendelbach S, Fletcher L, Frederickson J, Drew BJ, Funk M. Computer-assisted interventions to improve QTc documentation in patients receiving QT-prolonging drugs. *Am J Crit Care*. 2015;24:e6–e15. doi: 10.4037/ajcc2015240.
465. Chronister C. Improving nurses' knowledge of continuous ST-segment monitoring. *AACN Adv Crit Care*. 2014;25:104–113. doi: 10.1097/NCI.0000000000000029.
466. Blakeman JR, Sarsfield K, Booker KJ. Nurses' practices and lead selection in monitoring for myocardial ischemia: an evidence-based quality improvement project. *Dimens Crit Care Nurs*. 2015;34:189–195. doi: 10.1097/DCC.0000000000000118.
467. Rolskov Bojsen S, Räder SB, Holst AG, Kayser L, Ringsted C, Hastrup Svendsen J, Konge L. The acquisition and retention of ECG interpretation skills after a standardized web-based ECG tutorial—a randomised study. *BMC Med Educ*. 2015;15:36. doi: 10.1186/s12909-015-0319-0.
468. Jheeta JS, Narayan O, Krasemann T. Accuracy in interpreting the paediatric ECG: a UK-wide study and the need for improvement. *Postgrad Med J*. 2015;91:436–438. doi: 10.1136/postgradmedj-2013-305788rep.
469. Wathen JE, Rewers AB, Yetman AT, Schaffer MS. Accuracy of ECG interpretation in the pediatric emergency department. *Ann Emerg Med*. 2005;46:507–511. doi: 10.1016/j.annemergmed.2005.03.013.
470. Snyder CS, Bricker JT, Fenrich AL, Friedman RA, Rosenthal GL, Johnsrude CL, Kertesz C, Kertesz NJ. Can pediatric residents interpret electrocardiograms? *Pediatr Cardiol*. 2005;26:396–399. doi: 10.1007/s00246-004-0759-5.
471. Crocetti M, Thompson R. Electrocardiogram interpretation skills in pediatric residents. *Ann Pediatr Cardiol*. 2010;3:3–7. doi: 10.4103/0974-2069.64356.
472. Balady GJ, Bufalino VJ, Gulati M, Kuvin JT, Mendes LA, Schuller JL. COCATS 4 Task Force 3: training in electrocardiography, ambulatory electrocardiography, and exercise testing. *J Am Coll Cardiol*. 2015;65:1763–1777. doi: 10.1016/j.jacc.2015.03.021.
473. Badri M, Goldstein MA, Burke JF, Kowey PR. Reversal of fortunes: recognizing misplacement of ECG electrodes. *Am J Med*. 2013;126:868–870. doi: 10.1016/j.amjmed.2013.06.017.
474. Drew B. Standardization of electrode placement for continuous patient monitoring: introduction of an assessment tool to compare proposed electrocardiogram lead configurations. *J Electrocardiol*. 2011;44:115–118. doi: 10.1016/j.jelectrocard.2010.10.031.
475. García-Niebla J, Llontop-García P, Valle-Racero JI, Serra-Autonell G, Batchvarov VN, de Luna AB. Technical mistakes during the acquisition of the electrocardiogram. *Ann Noninvasive Electrocardiol*. 2009;14:389–403. doi: 10.1111/j.1542-474X.2009.00328.x.
476. Greenfield JC Jr, Rembert JC. ECG acquisition errors due to arm leads placed on the chest. *J Electrocardiol*. 2009;42:235–237. doi: 10.1016/j.jelectrocard.2008.10.009.
477. Harrigan RA, Chan TC, Brady WJ. Electrocardiographic electrode misplacement, misconnection, and artifact. *J Emerg Med*. 2012;43:1038–1044. doi: 10.1016/j.jemermed.2012.02.024.
478. Toosi MS, Sochanski MT. False ST elevation in a modified 12-lead surface electrocardiogram. *J Electrocardiol*. 2008;41:197–201. doi: 10.1016/j.jelectrocard.2007.11.004.
479. Pettersen TR, Fålnun N, Norekvål TM. Improvement of in-hospital telemetry monitoring in coronary care units: an intervention study for achieving optimal electrode placement and attachment, hygiene and delivery of critical information to patients. *Eur J Cardiovasc Nurs*. 2014;13:515–523. doi: 10.1177/1474515113515585.
480. Kadish AH, Buxton AE, Kennedy HL, Knight BP, Mason JW, Schuger CD, Tracy CM. ACC/AHA clinical competence statement on electrocardiography and ambulatory electrocardiography: a report of the American College of Cardiology/American Heart Association/American College of

- Physicians-American Society of Internal Medicine Task Force on Clinical Competence (ACC/AHA Committee to Develop a Clinical Competence Statement on Electrocardiography and Ambulatory Electrocardiography). *Circulation*. 2001;104:3169–3178.
481. Sangkachand P, Sarosario B, Funk M. Continuous ST-segment monitoring: nurses' attitudes, practices, and quality of patient care. *Am J Crit Care*. 2011;20:226–237; quiz 238. doi: 10.4037/ajcc2011129.
 482. Davies A, Macleod R, Bennett-Britton I, McElnay P, Bakhbaki D, Sansom J. E-learning and near-peer teaching in electrocardiogram education: a randomized trial. *Clin Teach*. 2015;12:1–4. doi: 10.1111/tct.12421.
 483. Jeffries PR. Development and testing of a Hyperlearning Model for design of an online critical care course. *J Nurs Educ*. 2005;44:366–372.
 484. Morris LL, Pfeifer PB, Catalano R, Fortney R, Hilton EL, McLaughlin J, Nelson G, Palamone J, Rabito R, Wetzel R, Goldstein L. Designing a comprehensive model for critical care orientation. *Crit Care Nurse*. 2007;27:37–40, 42, 46–48 passim; quiz 61.
 485. Drew BJ. Pulling it all together: case studies on ECG monitoring. *AACN Adv Crit Care*. 2007;18:305–317.
 486. Hatala R, Norman GR, Brooks LR. Impact of a clinical scenario on accuracy of electrocardiogram interpretation. *J Gen Intern Med*. 1999;14:126–129.
 487. Ray K, Berger B. Challenges in healthcare education: a correlational study of outcomes using two learning techniques. *J Nurses Staff Dev*. 2010;26:49–53; quiz 54–55. doi: 10.1097/NND.0b013e3181d4782c.
 488. Spiva L, Johnson K, Robertson B, Barrett DT, Jarrell NM, Hunter D, Mendoza I. The effectiveness of nurses' ability to interpret basic electrocardiogram strips accurately using different learning modalities. *J Contin Educ Nurs*. 2012;43:81–89. doi: 10.3928/00220124-20111011-02.
 489. Recommended guidelines for in-hospital cardiac monitoring of adults for detection of arrhythmia: Emergency Cardiac Care Committee Members. *J Am Coll Cardiol*. 1991;18:1431–1433.
 490. Leighton H, Kianfar H, Serynek S, Kerwin T. Effect of an electronic ordering system on adherence to the American College of Cardiology/American Heart Association guidelines for cardiac monitoring. *Crit Pathw Cardiol*. 2013;12:6–8. doi: 10.1097/HPC.0b013e318270787c.
 491. Tionson J, Robin J, Chana A, Shin DD, Gheorghiadu M. Are the American College of Cardiology/Emergency Cardiac Care (ACC/ECC) guidelines useful in triaging patients to telemetry units? *Acute Card Care*. 2006;8:155–160. doi: 10.1080/17482940600934192.
 492. Durairaj L, Reilly B, Das K, Smith C, Acob C, Husain S, Saquib M, Ganschow P, Evans A, McNutt R. Emergency department admissions to inpatient cardiac telemetry beds: a prospective cohort study of risk stratification and outcomes. *Am J Med*. 2001;110:7–11.
 493. Sultana RV, Kerr D, Kelly AM, Cameron P. Validation of a tool to safely triage selected patients with chest pain to unmonitored beds. *Emerg Med (Fremantle)*. 2002;14:393–399.
 494. Estrada CA, Rosman HS, Prasad NK, Battilana G, Alexander M, Held AC, Young MJ. Evaluation of guidelines for the use of telemetry in the non-intensive-care setting. *J Gen Intern Med*. 2000;15:51–55.
 495. Sandau KE, Sendelbach S, Frederickson J, Doran K. National survey of cardiologists' standard of practice for continuous ST-segment monitoring. *Am J Crit Care*. 2010;19:112–123. doi: 10.4037/ajcc2010264.
 496. Ng TM, Bell AM, Hong C, Hara JM, Touchette DR, Danskey KN, Lindsay TT, Puumala SE. Pharmacist monitoring of QTc interval-prolonging medications in critically ill medical patients: a pilot study. *Ann Pharmacother*. 2008;42:475–482. doi: 10.1345/aph.1K458.
 497. Haugaa KH, Bos JM, Tarrell RF, Morlan BW, Caraballo PJ, Ackerman MJ. Institution-wide QT alert system identifies patients with a high risk of mortality. *Mayo Clin Proc*. 2013;88:315–325. doi: 10.1016/j.mayocp.2013.01.013.
 498. Tisdale JE, Jaynes HA, Kingery JR, Overholser BR, Mourad NA, Trujillo TN, Kovacs RJ. Effectiveness of a clinical decision support system for reducing the risk of QT interval prolongation in hospitalized patients [published correction appears in *Circ Cardiovasc Qual Outcomes*. 2014;7:981]. *Circ Cardiovasc Qual Outcomes*. 2014;7:381–390. doi: 10.1161/CIRCOUTCOMES.113.000651.