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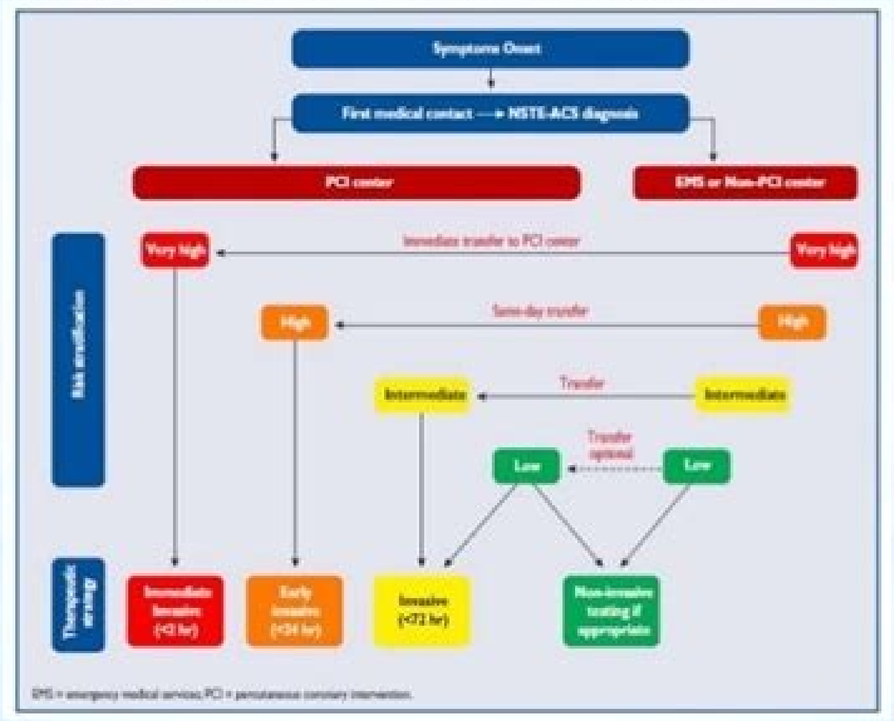
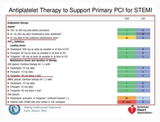
Selection of treatment strategy and timing according to initial risk stratification.



Antiplatelet Therapy to Support Primary PCI for STEMI



Clinical Practice Guideline

Physical Therapist Clinical Practice Guideline for the Management of Individuals With Heart Failure

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Michael J. Shoemaker, Konrad J. Dias, Kristin M. Lefebvre, John Heick and Sean M. Collins

The American Physical Therapy Association (APTA), in conjunction with the Cardiovascular and Pulmonary Section of APTA, have commissioned the development of this clinical practice guideline to assist physical therapists in their clinical decision making when treating patients with heart failure. Physical therapists treat patients with varying degrees of impairments and limitations in activity and participation associated with heart failure pathology across the continuum of care. This document will guide physical therapist practice in the examination and treatment of patients with a known diagnosis of heart failure. The development of this clinical practice guideline followed a structured process and resulted in 9 key action statements to guide physical therapist practice. The level and quality of available evidence were graded based on specific criteria to determine the strength of each action statement. Clinical algorithms were developed to guide the physical therapist in appropriate clinical decision making. Physical therapists are encouraged to work collaboratively with other members of the health care team in implementing these action statements to improve the activity, participation, and quality of life in individuals with heart failure and reduce the incidence of heart failure-related re-admissions.

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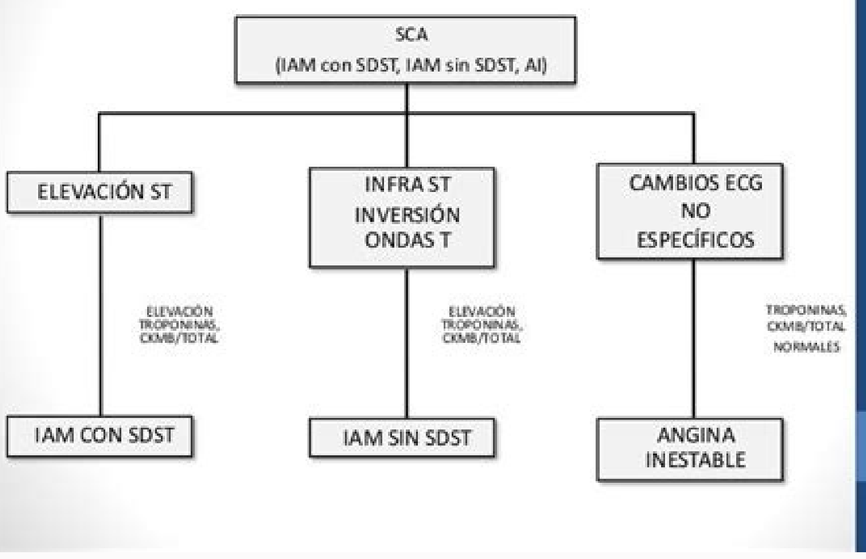
[Shoemaker MJ, Dias KJ, Lefebvre KM, et al. Physical therapist clinical practice guideline for the management of individuals with heart failure. *Phys Ther.* 2019;99:1–28.]

Accepted: August 24, 2019
Submitted: January 13, 2019

Clasificación de los SCA



Indications for appropriate Cath Lab activation	Diagnostic criteria for patients with symptoms <12 h	2004 ACC/AHA guideline recommendation	Proposed update vs. ACC/AHA guidelines	Comment
Classic STEMI				
Anterior	ST-elevation ≥1 mm in 2 contiguous leads V_1-V_4	Class I-A	Agree	ST-elevation ≥2 mm (men) and ≥1.5 mm (women) improves diagnostic specificity.[1] Presence of reciprocal changes (ST-depression in opposite leads) improves diagnostic specificity.
Inferior	ST-elevation ≥1 mm in 2 contiguous leads (II, III, or AVF)	Class I-A	Agree	Presence of reciprocal changes improves diagnostic specificity.
Lateral	ST-elevation ≥1 mm in 2 contiguous leads (I, AVL, V_5, or V_6)	Class I-A	Agree	As above.

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AbstractThis clinical practice guideline for the evaluation and diagnosis of chest pain provides recommendations and algorithms for clinicians to assess and diagnose chest pain in adult patients.Methods:A comprehensive literature search was conducted from November 11, 2017, to May 1, 2020, encompassing randomized and nonrandomized trials, observational studies, registries, reviews, and other evidence conducted on human subjects that were published in English from PubMed, EMBASE, the Cochrane Collaboration, Agency for Healthcare Research and Quality reports, and other relevant databases. Additional relevant studies, published through April 2021, were also considered.Structure:Chest pain is a frequent cause for emergency department visits in the United States. The “2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain” provides recommendations based on contemporary evidence on the assessment and evaluation of chest pain. This guideline presents an evidence-based approach to risk stratification and the diagnostic workup for the evaluation of chest pain. Cost-value considerations in diagnostic testing have been incorporated, and shared decision-making with patients is recommended.Abstract e368Top 10 Take-Home Messages for the Evaluation and Diagnosis of Chest Pain e370Preamble e3701. Introduction e3721.1. Methodology and Evidence Review e3721.2. Organization of the Writing Committee e3731.3. Document Review and Approval e3741.4. Scope of the Guideline e3741.4.1. Scope of the Problem e3741.4.2. Defining Chest Pain e3751.5. Abbreviations e3762. Initial Evaluation e3772.1. History e3772.1.1. A Focus on the Uniqueness of Chest Pain in Women e3782.1.2. Considerations for Older Patients With Chest Pain e3792.1.3. Considerations for Diverse Patient Populations With Chest Pain e3792.1.4. Patient-Centric Considerations e3802.2. Physical Examination e3802.3. Diagnostic Testing e3812.3.1. 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Intermediate-Risk Patients With Acute Chest Pain and No Known CAD e3944.1.2.1.1. Cost-Value Considerations e3954.1.2.2. Intermediate-Risk Patients With Acute Chest Pain and Known CAD e3954.1.3. High-Risk Patients With Acute Chest Pain e3984.1.4. Acute Chest Pain in Patients With Prior Coronary Artery Bypass Graft (CABG) Surgery e3984.1.5. Evaluation of Patients With Acute Chest Pain Receiving Dialysis e3994.1.6. Evaluation of Acute Chest Pain in Patients With Cocaine and Methamphetamine Use e4004.1.7. Shared Decision-Making in Patients With Acute Chest Pain e4004.2. Evaluation of Acute Chest Pain With Nonischemic Cardiac Pathologies e4014.2.1. Acute Chest Pain With Suspected Acute Aortic Syndrome e4014.2.2. Acute Chest Pain With Suspected PE e4024.2.3. Acute Chest Pain With Suspected Myopericarditis e4024.2.4. Acute Chest Pain With Valvular Heart Disease e4034.3. Evaluation of Acute Chest Pain With Suspected Noncardiac Causes e4044.3.1. 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Patients With Suspected Ischemia and No Obstructive CAD (INOCA) e4165.3. Cost-Value Considerations in Diagnostic Testing e4185.3.1. CCTA and CAC Scanning Cost-Value Considerations e4185.3.2. Exercise Electrocardiographic Cost-Value Considerations e4185.3.3. Stress Echocardiographic Cost-Value Considerations e4185.3.4. Stress Nuclear MPI Cost-Value Considerations e4195.3.5. Stress CMR Cost-Value Considerations e4196. Evidence Gaps and Future Research e419References e421Appendix 1 Author Relationships With Industry and Other Entities (Relevant) e445Appendix 2 Reviewer Relationships With Industry and Other Entities (Comprehensive) e448Chest Pain Means More Than Pain in the Chest. Pain, pressure, tightness, or discomfort in the chest, shoulders, arms, neck, back, upper abdomen, or jaw, as well as shortness of breath and fatigue should all be considered anginal equivalents.High-Sensitivity Troponins Preferred. High-sensitivity cardiac troponins are the preferred standard for establishing a biomarker diagnosis of acute myocardial infarction, allowing for more accurate detection and exclusion of myocardial injury.Early Care for Acute Symptoms. Patients with acute chest pain or chest pain equivalent symptoms should seek medical care immediately by calling 9-1-1. Although most patients will not have a cardiac cause, the evaluation of all patients should focus on the early identification or exclusion of life-threatening causes.Share the Decision-Making. Clinically stable patients presenting with chest pain should be included in decision-making; information about risk of adverse events, radiation exposure, costs, and alternative options should be provided to facilitate the discussion.Testing Not Needed Routinely for Low-Risk Patients. For patients with acute or stable chest pain determined to be low risk, urgent diagnostic testing for suspected coronary artery disease is not needed.Pathways. Clinical decision pathways for chest pain in the emergency department and outpatient settings should be used routinely.Accompanying Symptoms. Chest pain is the dominant and most frequent symptom for both men and women ultimately diagnosed with acute coronary syndrome. Women may be more likely to present with accompanying symptoms such as nausea and shortness of breath.Identify Patients Most Likely to Benefit From Further Testing. Patients with acute or stable chest pain who are at intermediate risk or intermediate to high pre-test risk of obstructive coronary artery disease, respectively, will benefit the most from cardiac imaging and testing.Noncardiac Is In. Atypical Is Out. “Noncardiac” should be used if heart disease is not suspected. “Atypical” is a misleading descriptor of chest pain, and its use is discouraged.Structured Risk Assessment Should Be Used. For patients presenting with acute or stable chest pain, risk for coronary artery disease and adverse events should be estimated using evidence-based diagnostic protocols.Figure 1 illustrates the take-home messages.Figure 1. Take-Home Messages for the Evaluation and Diagnosis of Chest PainPreambleSince 1980, the American College of Cardiology (ACC) and American Heart Association (AHA) have translated scientific evidence into clinical practice guidelines with recommendations to improve cardiovascular health. These guidelines, which are based on systematic methods to evaluate and classify evidence, provide a foundation for the delivery of quality cardiovascular care. The ACC and AHA sponsor the development and publication of clinical practice guidelines without commercial support, and members volunteer their time to the writing

and review efforts. Guidelines are official policy of the ACC and AHA. For some guidelines, the ACC and AHA partner with other organizations.Intended UseClinical practice guidelines provide recommendations applicable to patients with or at risk of developing cardiovascular disease. The focus is on medical practice in the United States, but these guidelines are relevant to patients throughout the world. Although guidelines may be used to inform regulatory or payer decisions, the intent is to improve quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances and should not replace clinical judgment.Clinical ImplementationManagement, in accordance with guideline recommendations, is effective only when followed by both practitioners and patients. Adherence to recommendations can be enhanced by shared decision-making between clinicians and patients, with patient engagement in selecting interventions on the basis of individual values, preferences, and associated conditions and comorbidities.Methodology and ModernizationThe ACC/AHA Joint Committee on Clinical Practice Guidelines (Joint Committee) continuously reviews, updates, and modifies guideline methodology on the basis of published standards from organizations, including the Institute of Medicine,1,2 and on the basis of internal reevaluation. Similarly, presentation and delivery of guidelines are reevaluated and modified in response to evolving technologies and other factors to optimally facilitate dissemination of information to healthcare professionals at the point of care.Numerous modifications to the guidelines have been implemented to make them shorter and enhance "user friendliness." Guidelines are written and presented in a modular, "knowledge chunk" format, in which each chunk includes a table of recommendations, a brief synopsis, recommendation-specific supportive text and, when appropriate, flow diagrams or additional tables. Hyperlinked references are provided for each modular knowledge chunk to facilitate quick access and review.In recognition of the importance of cost–value considerations, in certain guidelines, when appropriate and feasible, an analysis of value for a drug, device, or intervention may be performed in accordance with the ACC/AHA methodology.3To ensure that guideline recommendations remain current, new data will be reviewed on an ongoing basis by the writing committee and staff. Going forward, targeted sections/knowledge chunks will be revised dynamically after publication and timely peer review of potentially practice-changing science. The previous designations of "full revision" and "focused update" will be phased out. For additional information and policies on guideline development, readers may consult the ACC/AHA guideline methodology manual4 and other methodology articles.5-7 The Class of Recommendation (COR) indicates the strength of recommendation, encompassing the estimated magnitude and certainty of benefit in proportion to risk. The Level of Evidence (LOE) rates the quality of scientific evidence supporting the intervention on the basis of the type, quantity, and consistency of data from clinical trials and other sources (Table 1).4Table 1. Applying ACC/AHA Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care (Updated May 2019)*Selection of Writing Committee MembersThe Joint Committee strives to ensure that the guideline writing committee contains requisite content expertise and is representative of the broader cardiovascular community by selection of experts across a spectrum of backgrounds, representing different geographic regions, sexes, races, ethnicities, intellectual perspectives/biases, and clinical practice settings. Organizations and professional societies with related interests and expertise are invited to participate as partners or collaborators.Relationships With Industry and Other EntitiesThe ACC and AHA have rigorous policies and methods to ensure that documents are developed without bias or improper influence. The complete policy on relationships with industry and other entities (RWI) can be found online. Appendix 1 of the guideline lists writing committee members' relevant RWI; for the purposes of full transparency, their comprehensive disclosure information is available in the Supplemental Appendix. Comprehensive disclosure information for the Joint Committee is also available online.Evidence Review and Evidence Review CommitteesIn developing recommendations, the writing committee uses evidence-based methodologies that are based on all available data.4,5 Literature searches focus on randomized controlled trials (RCTs) but also include registries, nonrandomized comparative and descriptive studies, case series, cohort studies, systematic reviews, and expert opinions. Only key references are cited.An independent evidence review committee is commissioned when there are ≥1 questions deemed of utmost clinical importance and merit formal systematic review to determine which patients are most likely to benefit from a drug, device, or treatment strategy, and to what degree. Criteria for commissioning an evidence review committee and formal systematic review include absence of a current authoritative systematic review, feasibility of defining the benefit and risk in a time frame consistent with the writing of a guideline, relevance to a substantial number of patients, and likelihood that the findings can be translated into actionable recommendations. Evidence review committee members may include methodologists, epidemiologists, clinicians, and biostatisticians. Recommendations developed by the writing committee on the basis of the systematic review are marked "SR."Guideline-Directed Management and TherapyThe term guideline-directed medical therapy (GDMT) encompasses clinical evaluation, diagnostic testing, and both pharmacological and procedural treatments. For these and all recommended drug treatment regimens, the reader should confirm dosage with product insert material and evaluate for contraindications and interactions. Recommendations are limited to drugs, devices, and treatments approved for clinical use in the United States.Patrick T. O'Gara, MD, MACC, FAHA Chair, ACC/AHA Joint Committee on Clinical Practice Guidelines1. Introduction1.1. Methodology and Evidence ReviewThe recommendations listed in this guideline are, whenever possible, evidence based. An initial extensive evidence review, which included literature derived from research involving human subjects, published in English, and indexed in MEDLINE (through PubMed), EMBASE, the Cochrane Library, the Agency for Healthcare Research and Quality, and other selected databases relevant to this guideline, was conducted from November 11, 2017, to May 1, 2020. Key search words included but were not limited to the following: acute coronary syndrome, angina, angina pectoris, aortic valve stenosis, biomarker, biomarkers, brain natriuretic peptide, cardiac-gated single photon emission computer-assisted tomography, cardiovascular magnetic resonance, chest pain, CKMB, coronary angiography, coronary arteriosclerosis, coronary artery disease, creatine kinase, creatine kinase MB, echocardiography, electrocardiography, heart valve disease, hypertrophic cardiomyopathy, magnetic resonance imaging, mitral valve stenosis, multidetector computed tomography, myocardial infarction, myocardial ischemia, myocardium, NT-proBNP, perfusion imaging, positron-emission tomography, pulmonary hypertension, stable angina, troponin I, troponin T, unstable angina, x-ray computed tomography. Additional relevant studies, published through November 2020 during the guideline writing process, were also considered by the writing committee and added to the evidence tables when appropriate. The final evidence tables are included in the Online Data Supplement and summarize the evidence used by the writing committee to formulate recommendations. References selected and published in the present document are representative and not all-inclusive.1.2. Organization of the Writing CommitteeThe writing committee consisted of cardiac intensivists, cardiac interventionalists, cardiac surgeons, cardiologists, emergency physicians, epidemiologists, and a lay/patient representative. The writing committee included representatives from the ACC, AHA, American Society of Echocardiography (ASE), American College of Chest Physicians (CHEST), Society for Academic Emergency Medicine (SAEM), Society of Cardiovascular Computed Tomography (SCCT), and Society for Cardiovascular Magnetic Resonance (SCMR). Appendix 1 lists writing committee members' relevant RWI. For the purposes of full transparency, the writing committee members' comprehensive disclosure information is available in the Supplemental Appendix.1.3. Document Review and ApprovalThis document was reviewed by 16 official reviewers nominated by the ACC, the American College of Emergency Physicians, AHA, ASE, American Society of Nuclear Cardiology, CHEST, SAEM, SCCT, and SCMR, and 39 individual content reviewers. Reviewers' RWI information was distributed to the writing committee and is published in this document (Appendix 2).This document was approved for publication by the governing bodies of the ACC and the AHA and was endorsed by the ASE, CHEST, SAEM, SCCT, and SCMR.1.4. Scope of the GuidelineThe charge of the writing committee was to develop a guideline for the evaluation of acute or stable chest pain or other anginal equivalents, in various clinical settings, with an emphasis on the diagnosis on ischemic causes. This guideline will not provide recommendations on whether revascularization is appropriate or what modality is indicated. Such recommendations can be found in the forthcoming ACC/AHA coronary artery revascularization guideline. In developing the "2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain," the writing committee first reviewed previous published guidelines and related statements. Table 2 contains a list of these publications deemed pertinent to this writing effort and is intended for use as a resource, thus obviating the need to repeat existing guideline recommendations.Table 2. Associated Guidelines and StatementsTitleOrganizationPublication Year (Reference)GuidelinesStable ischemic heart diseaseACC/AHA/AATS/PCNA/SCAI/STS20141*20122Atrial fibrillationAHA/ACC/HRS20143*20194Non-ST elevation ACSAHA/ACC20145Blood cholesterolAHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA20186Heart failureACC/AHA20137*20178Primary prevention of cardiovascular diseaseACC/AHA20199Management of overweight and obesity in adultsAHA/ACC/TOS201410ST-elevation myocardial infarctionACC/AHA201311Ventricular arrhythmias and the prevention of sudden cardiac deathAHA/ACC/HRS201712Coronary artery bypass graft surgeryACC/AHA201113Hypertrophic cardiomyopathyACC/AHA202014Percutaneous coronary interventionACC/AHA/SCAI201115*201516Secondary prevention and risk reduction therapy for patients with coronary and other atherosclerotic vascular diseaseAHA/ACC201117Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care—part 9: postcardiac arrest careAHA201018*201919Prevention, detection, evaluation, and management of high blood pressure in adultsACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA201720StatementsTesting of low-risk patients presenting to the emergency department with chest painAHA201021Prevention of cardiovascular disease in adults with type 2 diabetes mellitusAHA/ADA201522Prevention and control of seasonal influenza with vaccinesCDC2018231.4.1. Scope of the ProblemSynopsisAfter injuries, chest pain is the second most common reason for adults to present to the emergency department (ED) in the United States and accounts for >6.5 million visits, which is 4.7% of all ED visits.1 Chest pain also leads to nearly 4 million outpatient visits annually in the United States.2 Chest pain remains a diagnostic challenge in the ED and outpatient setting and requires thorough clinical evaluation. Although the cause of chest pain is often noncardiac, coronary artery disease (CAD) affects >18.2 million adults in the United States and remains the leading cause of death for men and women, accounting for >365 000 deaths annually.3 Distinguishing between serious and benign causes of chest pain is imperative. The lifetime prevalence of chest pain in the United States is 20% to 40%,4 and women experience this symptom more often than men.5 Of all ED patients with chest pain, only 5.1% will have an acute coronary syndrome (ACS), and more than half will ultimately be found to have a noncardiac cause.6 Nonetheless, chest pain is the most common symptom of CAD in both men and women.1.4.2. Defining Chest PainSynopsisChest pain is one of the most common reasons that people seek medical care. The term "chest pain" is used by patients and applied by clinicians to describe the many unpleasant or uncomfortable sensations in the anterior chest that prompt concern for a cardiac problem. Chest pain should be considered acute when it is new onset or involves a change in pattern, intensity, or duration compared with previous episodes in a patient with recurrent symptoms. Chest pain should be considered stable when symptoms are chronic and associated with consistent precipitants such as exertion or emotional stress.Although the term chest pain is used in clinical practice, patients often report pressure, tightness, squeezing, heaviness, or burning. In this regard, a more appropriate term is "chest discomfort," because patients may not use the descriptor "pain." They may also report a location other than the chest, including the shoulder, arm, neck, back, upper abdomen, or jaw. Despite individual variability, the discomfort induced by myocardial ischemia is often characteristic and therefore central to the diagnosis. For this reason, features more likely to be associated with ischemia have been described as typical versus atypical; however, the latter can be confusing because it is frequently used to describe symptoms considered nonischemic as well as noncardiac. Although other nonclassic symptoms of ischemia, such as shortness of breath, nausea, radiating discomfort, or numbness, may be present, chest pain or chest discomfort remains the predominant symptom reported in men and women who are ultimately diagnosed with myocardial ischemia.3-7 Pain—described as sharp, fleeting, related to inspiration (pleuritic) or position, or shifting locations—suggests a lower likelihood of ischemia.Recommendation-Specific Supportive Text1.Like most visceral discomfort, the sensation produced by myocardial ischemia is characteristically deep, difficult to localize, and usually diffuse. Point tenderness renders ischemia less likely. Reported symptoms lie somewhere on a continuum of higher or lower probability of ischemia based on the presence or absence of specific characteristics (Figure 2). Other clinical elements (eg, duration, provoking and relieving factors, patient age, cardiac risk factors) provide further focus toward or away from ischemia in the diagnostic process. It is essential to ascertain the characteristics of the chest pain directly from the patient for optimal interpretation.1-7 A patient's history is the most important basis for considering presence or absence of myocardial ischemia, but the source of cardiac symptoms is complex, and their expression is variable. The diagnosis of ischemia may require data beyond history alone. In some patients, what appears to be noncardiac chest pain may be ischemic in origin.2.Chest pain has been traditionally stratified into "typical" and "atypical" types. Chest pain that is more likely associated with ischemia consists of substernal chest discomfort provoked by exertion or emotional stress and relieved by rest or nitroglycerin. The more classic the chest discomfort is based on quality, location, radiation, and provoking and relieving factors, the more likely it is to be of cardiac ischemic origin. Atypical chest pain is a problematic term. Although it was intended to indicate angina without typical chest symptoms, it is more often used to state that the symptom is noncardiac in origin. As such, we discourage the use of atypical chest pain. Emphasis is more constructively placed on specific aspects of symptoms that suggest their origin in terms of probable ischemia. Of note, chest pain is broadly defined to also include referred pain in the shoulders, arms, jaw, neck, and upper abdomen. To diminish ambiguity, use "cardiac," "possible cardiac," and "noncardiac" to describe the suspected cause of chest pain is encouraged.Figure 2. Index of Suspicion That Chest "Pain" Is Ischemic in Origin on the Basis of Commonly Used Descriptors1.5. Abbreviations2. Initial Evaluation2.1. HistorySynopsisChest pain or chest pain equivalent will be referred to in these guidelines as "chest pain." Patients presenting to the ED with nontraumatic chest pain are a frequent diagnostic challenge.1 The priorities are: 1) rapid initiation of optimal management in patients with life-threatening conditions such as ACS, aortic dissection, and pulmonary embolism (PE), as well as nonvascular syndromes (eg, esophageal rupture, tension pneumothorax); and 2) deliberate therapy for those with less critical illness. Although there are several life-threatening causes, chest pain usually reflects a more benign condition (Figure 3).2-4 The initial ECG is important to the evaluation, but history, examination, biomarkers, and other aids remain essential. There is frequently a lack of correlation between intensity of symptoms and seriousness of disease and general similarity of symptoms among different causes of chest pain. A comprehensive history that captures all the characteristics of chest pain (Table 3), including but not limited to its: 1) nature; 2) onset and duration; 3) location and radiation; 4) precipitating factors; 5) relieving factors; and 6) associated symptoms can help better identify potential cardiac causes and should be obtained from all patients.Table 3. Chest Pain Characteristics and Corresponding CausesNatureAnginal symptoms are perceived as retrosternal chest discomfort (eg, pain, discomfort, heaviness, tightness, pressure, constriction, squeezing) (Section 1.4.2, Defining Chest Pain).Sharp chest pain that increases with inspiration and lying supine is unlikely related to ischemic heart disease (eg, these symptoms usually occur with acute pericarditis).Onset and durationAnginal symptoms gradually build in intensity over a few minutes.Sudden onset of ripping chest pain (with radiation to the upper or lower back) is unlikely to be anginal and is suspicious of an acute aortic syndrome.Fleeting chest pain—of few seconds' duration—is unlikely to be related to ischemic heart disease.Location and radiationPain that can be localized to a very limited area and pain radiating to below the umbilicus or hip are unlikely related to myocardial ischemia.SeverityRipping chest pain ("worse chest pain of my life"), especially when sudden in onset and occurring in a hypertensive patient, or with a known bicuspid aortic valve or aortic dilation, is suspicious of an acute aortic syndrome (eg, aortic dissection).Precipitating factorsPhysical exercise or emotional stress are common triggers of anginal symptoms.Occurrence at rest or with minimal exertion associated with anginal symptoms usually indicates ACS.Positional chest pain is usually nonischemic (eg, musculoskeletal).Relieving factorsRelief with nitroglycerin is not necessarily diagnostic of myocardial ischemia and should not be used as a diagnostic criterion.Associated symptomsCommon symptoms associated with myocardial ischemia include, but are not limited to, dyspnea, palpitations, diaphoresis, lightheadedness, presyncope or syncope, upper abdominal pain, or heartburn unrelated to meals and nausea or vomiting.Symptoms on the left or right side of the chest, stabbing, sharp pain, or discomfort in the throat or abdomen may occur in patients with diabetes, women, and elderly patients.Figure 3. Top 10 Causes of Chest Pain in the ED Based on Age (Weighted Percentage) Created using data from Hsia RY et al.3 ED indicates emergency department.Recommendation-Specific Supportive Text1.Angina pectoris is perceived as a retrosternal chest discomfort that builds gradually in intensity (over several minutes), is usually precipitated by stress (physical or emotional) or occurring at rest (as in the case of an ACS) with characteristic radiation (eg, left arm, neck, jaw) and its associated symptoms (eg, dyspnea, nausea, lightheadedness). When actively treated or spontaneously resolving, it dissipates over a few minutes. Relief with nitroglycerin is not necessarily diagnostic of myocardial ischemia and should not be used as a diagnostic criterion, especially because other entities demonstrate comparable response (eg, esophageal spasm).1,5 Associated symptoms such as shortness of breath, nausea or vomiting, lightheadedness, confusion, presyncope or syncope, or vague abdominal symptoms are more frequent among patients with diabetes, women, and the elderly. A detailed assessment of cardiovascular risk factors, review of systems, past medical history, and family and social history should complement the assessment of presenting symptoms.2.1.1. A Focus on the Uniqueness of Chest Pain in WomenSynopsisMost patients who present to the ED with chest pain are women, particularly among those ≥65 years of age.8 The ISCHEMIA (International Study of Comparative Health Effectiveness With Medical and Invasive Approaches) trial demonstrated that women with moderate-to-severe ischemia are more symptomatic than men.9 Women are less likely to have timely and appropriate care.10 This could be explained by the fact that women are more likely to experience prodromal symptoms when they seek medical care.11 Women may also present with accompanying symptoms (eg, nausea, fatigue, and shortness of breath) more often than men.12-14 However, chest pain remains the predominant symptom reported by women among those ultimately diagnosed with ACS, occurring with a frequency equal to men.3,5-7,15,16Recommendation-Specific Supportive Text1.Traditional risk score tools and physician assessments often underestimate risk in women and misclassify them as having nonischemic chest pain.1,2 The PROMISE (Prospective Multicenter Imaging Study for Evaluation of Chest Pain) trial looked at sex differences in the presentation, risk factors, demographics, noninvasive test referrals, and results of 10 003 stable outpatients with suspected CAD.1 Women commonly presented with chest pain symptoms similar to men but also had a greater prevalence of other symptoms such as palpitations, jaw and neck pain, as well as back pain. Women also had more cardiovascular risk factors, including hypertension (66.6% versus 63.2%; P80% probability of dissection9Frequency of syncope >10%8, AR 40%–75% (type A)10Esophageal ruptureEmesis, subcutaneous emphysema, pneumothorax (20% patients), unilateral decreased or absent breath soundsOtherNoncoronary cardiac: AS, AR, HCMAS: Characteristic systolic murmur, tardus or parvus carotid pulseAR: Diastolic murmur at right of sternum, rapid carotid upstrokeHCM: Increased or displaced left ventricular impulse, prominent a wave in jugular venous pressure, systolic murmurPericarditisFever, pleuritic chest pain, increased in supine position, friction rubMyocarditisFever, chest pain, heart failure, S3Esophagitis, peptic ulcer disease, gall bladder diseaseEpigastric tendernessRight upper quadrant tenderness, Murphy signPneumoniaFever, localized chest pain, may be pleuritic, friction rub may be present, regional dullness to percussion, egophonyPneumothoraxDyspnea and pain on inspiration, unilateral absence of breath soundsCostochondritis, Tietze syndromeTenderness of costochondral jointsHerpes zosterPain in dermatomal distribution, triggered by touch; characteristic rash (unilateral and dermatomal distribution)2.3. Diagnostic Testing2.3.1. Setting ConsiderationsSynopsisThe goals in patients presenting to the ED or office with acute chest pain are: 1) identify life-threatening causes; 2) determine clinical stability; and 3) assess need for hospitalization versus safety of outpatient evaluation and management. These concerns entail consideration of the full extent of clinical data. The ACC/AHA STEMI and NSTE-ACS guidelines categorize chest pain cause into 4 types: STEMI, NSTE-ACS, stable angina, and noncardiac.6,7 The 12-lead ECG, which should be acquired and interpreted within 10 minutes of arrival to a medical facility1-7,11 (Section 2.3.2, ECG), is pivotal in the evaluation because of its capacity to identify and triage patients with STEMI to urgent coronary reperfusion. Other ST-T abnormalities consistent with possible ischemia also mandate prompt evaluation in a hospital setting. In both cases, transfer should be by EMS; personal automobile for this purpose is associated with increased risk and should be avoided.3-5 Patients with stable angina or noncardiac chest pain that is not life-threatening should be managed as outpatients.Recommendation-Specific Supportive Text1.The ECG is central to the evaluation of stable angina in the office setting to ensure that ACS is not missed.1,2,6,7 If an ECG cannot be obtained, transfer to the ED should be initiated.2.Transfer by EMS from the office setting for acute chest pain with suspected ACS or other life-threating conditions is recommended because of the important advantages provided by EMS including: 1) acquisition of a prehospital ECG, which can facilitate reperfusion if ST elevation is present; 2) presence of trained personnel who can provide treatment for chest pain, arrhythmias, and implement defibrillation en route; and 3) shorter travel time to the ED.1-7,103.Early recognition of STEMI improves outcomes.1-3,6,7 Therefore, regardless of the setting, an ECG should be obtained and interpreted within 10 minutes of arrival. If this cannot be achieved in the office setting, immediate transfer to the ED by EMS is recommended. A substantial proportion of patients with chest pain are transferred to the ED without a prehospital ECG.1-3,6,7 This results in an important and avoidable delay in readiness of the ED and reperfusion teams to implement optimally timed reperfusion therapy.1-7,104.cTn is the most sensitive test for diagnosing acute myocardial injury and, in conjunction with other essential clinical data (eg, history, examination, ECG), its measurement is necessary to implement appropriate therapy.8,95.Delayed transfer to the hospital for determination of cTn or other diagnostic testing beyond the ECG in the office setting can be detrimental and should be avoided.1-7,102.3.2. ElectrocardiogramSynopsisPatients with chest pain and new ST-elevation, ST depression, or new left bundle branch block on ECG should be treated according to STEMI and NSTE-ACS guidelines.1,2,6 An initial normal ECG does not exclude ACS. Patients with an initial normal ECG should have a repeat ECG, if symptoms are ongoing, until other diagnostic testing rules out ACS. An ECG may identify other nonischemic causes of chest pain (eg, pericarditis, myocarditis, arrhythmia, electrolyte abnormalities, paced rhythm, hypertrophic cardiomyopathy, pulmonary hypertension, congenital long QT, or normal variant). Figure 4 depicts an algorithm for the role of the ECG to help direct care for individuals presenting with chest pain or chest pain equivalents.Figure 4. Electrocardiographic-Directed Management of Chest Pain ECG indicates electrocardiogram; MI, myocardial infarction; NSTE-ACS, non–ST-segment–elevation acute coronary syndrome; and STEMI, ST-segment–elevation myocardial infarction.Recommendation-Specific Supportive Text1.When an ECG is nondiagnostic, it should be compared with previous ECGs, if available.7 A normal or unchanged ECG is reasonably useful but not sufficient at ruling out ACS.8-10 Thus, decision-making should not be based solely on a single normal or nondiagnostic ECG. Left ventricular hypertrophy, bundle branch blocks, and ventricular pacing may mask signs of ischemia or injury.11 Up to 6% of patients with evolving ACS are discharged from the ED with a normal ECG.12-17 In patients where the initial ECG is normal or is without ST elevation, hyperacute T waves, left bundle branch block, or ST depression, serial ECGs should be performed and management should be guided by new electrocardiographic changes or other diagnostic testing (see Section 2.3.4 on Biomarkers, Section 3.1 on Anatomic Testing, or Section 3.2 on Stress Testing).7,18-20 The timing for repeat ECG should also be guided by symptoms, especially if chest pain recurs or a change in clinical condition develops.212.When ST-elevation is present on the initial ECG, management should follow the prescribed STEMI treatment algorithms in associated guidelines.2,22 Furthermore, if ST depression is identified on the initial ECG, management should follow the NSTE-ACS guidelines.13.A normal ECG may be associated with left circumflex or right coronary artery occlusions and posterior wall ischemia, which is often "electrically silent"; therefore, right-sided ECG leads should be considered when such lesions are suspected.2-52.3.3. Chest RadiographySynopsisChest radiographs are rapid, noninvasive tests that can be used to screen for several disorders that may present with chest pain. The yield of chest radiography depends on the pretest probability and will thus be higher when history or physical examination point to a greater likelihood of a given diagnosis. However, chest radiographs often do not lead to a diagnosis that requires intervention,1 and their use should be guided by clinical suspicion.Recommendation-Specific Supportive Text1.The AHA/ACC guidelines for NSTE-ACS and heart failure all recommend chest radiographs on presentation, although this should not delay urgent revascularization if it is indicated.2,3 In patients with acute chest pain and heart failure, chest radiographs are useful to assess heart size and pulmonary congestion, as well as identifying potential pulmonary causes that may have contributed to symptoms. Chest radiographs may demonstrate a widened mediastinum in patients with aortic dissection, although they are not sensitive enough in this setting to rule out the diagnosis.4 Chest radiographs may be most useful in the evaluation of patients with acute chest pain to detect alternative cardiac, pulmonary, or other conditions that may cause symptoms, including pneumonia, pneumothorax, or rib fractures. Pleural effusions, pulmonary artery enlargement, and infiltrates may suggest PE, which would need to be confirmed by further testing.2.3.4. BiomarkersSynopsisCardiovascular biomarkers can be useful for the diagnostic and prognostic assessment of patients with chest pain. Their most important application in clinical practice is for the rapid identification or exclusion of myocardial injury. The preferred biomarker to detect or exclude myocardial injury is cTn (I or T) because of its high sensitivity and specificity for myocardial tissue.1-21,33 hs-cTn is preferred and can detect circulating cTn in the blood of most "healthy" individuals, with different sex-specific thresholds.17,21,34 cTn is organ-specific but not disease-specific. Numerous ischemic, noncoronary cardiac, and noncardiac causes of cardiomyocyte injury can result in elevated cTn concentrations.17,21,24,25 Therefore, interpretation of cTn results requires integration with all clinical information.17,21Although multiple other cardiovascular biomarkers, including some in common clinical use such as natriuretic peptides, have been shown to be associated with the risk of adverse cardiovascular outcomes in patients with chest pain, none have sufficient diagnostic accuracy for myocardial injury to be recommended for that purpose. The use of D-dimer for diagnosis of PE is discussed in Section 4.2.2.Recommendation-Specific Supportive Text1.The preferred biomarker to detect or exclude cardiac injury is cTn (I or T) because of its high sensitivity and specificity for myocardial tissue.1-21 Detection of myocardial cell injury, possibly indicative of AMI, is predicated on a rise or fall of this biomarker in blood.1,3,4,10-21 A cTn concentration >99th percentile upper reference limit, which is assay-dependent, is an indicator of myocardial injury.1,9,21 The coefficient of variation at the 99th percentile upper reference limit for each assay should be ≤10%.8,212.There is ample evidence for the superiority of hs-cTn assays over conventional cTn assays in multiple aspects of evaluation for patients presenting with chest pain with and without AMI.17,21,24,25,33 The sensitivity and negative predictive values are greater with hs-cTn compared with previous generation assays.17,21,24,25 In addition, the time interval from onset of chest pain to a detectable concentration at patient presentation is shorter with hs-cTn, affording more rapid rule-in and rule-out algorithms.22 Although it is sometimes challenging to diagnostically discriminate among these causes of myocardial injury, irrespective of the final diagnosis, the presence of myocardial injury is associated with a higher risk of adverse outcomes among patients with chest pain.353.The level of detection, 99th percentile upper reference limit, analytical precision, and criteria for a significant delta are assay-specific, including among the many different manufacturers of the same analyte (eg, hs-cTnI). To appropriately apply a cTn assay, clinicians must be familiar with these analytical performance properties for the assay(s) that they use in their practice.214.Comparative studies have confirmed the superiority of cTn over CK-MB and myoglobin for diagnosis and prognosis of AMI.27-32 The addition of CK-MB or myoglobin to cTn for evaluation of patients presenting with chest pain is not beneficial.3. Cardiac Testing General ConsiderationsFor acute and stable chest pain, noninvasive and invasive diagnostic testing is a core component of the evaluation underpinning its importance. Over the past decade, the quality of evidence supporting clinical indications for noninvasive testing has grown dramatically. The approach outlined in this guideline focuses on selective use of testing, optimization of lower cost evaluations, reducing layered testing, and deferring or eliminating testing when the diagnostic yield is low (Figure 5). Reducing unnecessary testing can provide a means to exert cost savings within the diagnostic evaluation of populations.1 In the same manner, elimination of testing where evidence is lacking and the reduction in testing among low-risk patients for whom deferred testing is appropriate are emphasized in this guideline.Figure 5. Chest Pain and Cardiac Testing Considerations The choice of imaging depends on the clinical question of importance, to either a) ascertain the diagnosis of CAD and define coronary anatomy or b) assess ischemia severity among patients with an expected higher likelihood of ischemia with an abnormal resting ECG or those incapable of performing maximal exercise. ACS indicates acute coronary syndrome; CAC, coronary artery calcium; CAD, coronary artery disease; and ECG, electrocardiogram. Please refer to Section 4.1. For risk assessment in acute chest pain, see Figure 9. For risk assessment in stable chest pain, see Figure 11.Testing choice will be influenced by site expertise and availability, but knowledge regarding which test may be preferable is useful when selecting between different modalities. Cost should also be considered, when known by the ordering clinician and there is equipoise between available modalities.2 The exercise ECG is the lowest cost procedure used in the diagnostic evaluation when compared with stress imaging or anatomic procedures, with the exception of coronary artery calcium (CAC) scoring (Figure 6). For all imaging procedures, costs vary by payer and site of services.Figure 6. Choosing the Right Diagnostic Test ASCVD indicates atherosclerotic cardiovascular disease; CAD, coronary artery disease; CAC, coronary artery calcium; CCTA, coronary computed tomography angiography; CMR, cardiovascular magnetic resonance; ETT, exercise tolerance test; LV, left ventricular; MPI, myocardial perfusion imaging; PET, positron emission tomography and SPECT, single-photon emission computed tomography.The following sections provide a brief overview of the various noninvasive tests available for use in the evaluation of symptomatic patients. Previously, the term known as CAD had been used to define those with a significant obstructive stenosis (ie, ≥50%). In this guideline, we revise the term known CAD to include patients with prior anatomic testing (invasive angiography or coronary computed tomographic angiography [CCTA]) with identified nonobstructive atherosclerotic plaque and obstructive CAD. We recognize this is a departure from convention, but our intent was to ensure that those with lesser degrees of stenosis who do not require coronary intervention but would benefit from optimized preventive therapy do not get overlooked. However, throughout the document, the term "obstructive," consistent with convention, will be used to indicate CAD with ≥50% stenosis and nonobstructive CAD will be used to indicate CAD

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