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MEDICAL POLICY – 2.02.517
Electrophysiology (EP) studies

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Introduction

Cardiac conduction abnormalities (disruptions in the heart's electrical signaling) can pose serious health risks, including fainting episodes and sudden cardiac death. To evaluate and manage these conditions, a diagnostic procedure known as a cardiac electrophysiology study (EPS) may be performed. An EPS is a minimally invasive test that uses specialized catheters placed inside the heart to monitor and stimulate electrical activity. This allows clinicians to identify abnormal pathways that may be causing irregular heartrhythms, known as arrhythmias. In some cases, arrhythmias are intentionally triggered during the study to better understand their origin and guide treatment decisions. EPS is commonly used to assess patients with known or suspected rhythm disorders. When appropriate, cardiac ablation, a procedure to eliminate the source of arrhythmia, may be performed during the same session. Additionally, EPS can help determine whether a patient with a slow heart rate may benefit from pacemaker therapy. This policy describes when EPS may be considered medically necessary.

Policy Coverage Criteria

Procedure	Medical Necessity
Electrophysiology Studies	**Electrophysiology studies may be considered medically necessary for ANY of the following indications:**

Procedure	Medical Necessity

	• The evaluation of syncope (fainting) in individuals with any of the following:
	  ○ Ischemic cardiomyopathy, non-ischemic cardiomyopathy (NICM), or structural heart disease (e.g. adult congenital heart disease) who do not meet criteria for implantable cardio-defibrillator (ICD) placement.
	  ○ Survivors of myocardial infarction (MI) with left ventricular ejection fraction (LVEF) greater than 35%.
	  ○ Sinus bradycardia (slow heartbeat less than 60 beats per minute).
	  ○ Suspicion of bradyarrhythmias (slow irregular heart rate) or tachyarrhythmias (abnormally fast heart rate) with inconclusive electrocardiogram (ECG) or echocardiogram.
	  ○ Bi-fascicular block (left or right bundle branch block [BBB] and hemi-fascicular block) with inconclusive ECG or echocardiogram.
	  ○ Cardiac sarcoidosis. (clumps of cells form in the heart)
	• Survivors of sudden cardiac arrest (SCA)
	• Recurrent or persistent symptomatic supraventricular tachyarrhythmias (SVT) (e.g. atrial tachycardia, atrial fibrillation, atrial flutter, atrial ventricular node re-entry tachycardia, paroxysmal SVT)
	• Symptomatic recurrent ventricular tachycardia (VT)
	• Pre-excitation syndromes (e.g. Wolf-Parkinson-White (WPW))
	• Frequent symptomatic non-sustained ventricular arrhythmias (e.g. premature ventricular contractions (PVCs) 10,000 per 24 hours)
	
	Electrophysiology studies are considered not medically necessary for any other indication including but not limited to the following conditions:
	• Risk stratification in an individual that meets criteria for an ICD
	• Risk stratification for hypertrophic cardiomyopathy
	• Risk stratification for long QT syndrome (LQTS)
	• Risk stratification for catecholaminergic polymorphic ventricular tachycardia (CPVT) (episodic syncope occurring during exercise or emotional stress)

Procedure	Medical Necessity
	• Risk stratification for short QT syndrome (SQTS) • Risk stratification for early repolarization syndromes • Evaluation of syncope with Brugada syndrome

Length of Approval	
Approval	**Criteria**
Initial authorization	**One electrophysiology session within 12 months.**

Documentation Requirements

The patient's medical records submitted for review for all conditions should document that medical necessity criteria are met. The record should include the following:

- Office visit notes that contain the relevant history and physical and diagnosis being evaluated or treated with detailed cardiac history and suspected or known cardiac arrhythmia to be studied.

Coding

Code	Description
CPT	
93609	Intraventricular and/or intra-arterial mapping of tachycardia site(s) with catheter manipulation to record from multiple sites to identify origin of tachycardia
93613	Intracardiac electrophysiologic 3-dimensional mapping
93619	Comprehensive electrophysiologic evaluation with right atrial pacing and recording, right ventricular pacing and recording, His bundle recording, including insertion and repositioning of multiple electrode catheters, without induction or attempted induction of arrhythmia
93620	Comprehensive electrophysiologic evaluation including insertion and repositioning of multiple electrode catheters with induction or attempted induction of arrhythmia; with right atrial pacing and recording, right ventricular pacing and recording, His bundle recording
93621	Comprehensive electrophysiologic evaluation including insertion and repositioning of multiple electrode catheters with induction or attempted induction of arrhythmia; with left atrial pacing and recording from coronary sinus or left atrium

Code	Description
93622	Comprehensive electrophysiologic evaluation including insertion and repositioning of multiple electrode catheters with induction or attempted induction of arrhythmia; with left ventricular pacing and recording
93624	Electrophysiologic follow-up study with pacing and recording to test effectiveness of therapy, including induction or attempted induction of arrhythmia Catheter ablation procedures:
93653	Comprehensive electrophysiologic evaluation with insertion and repositioning of multiple electrode catheters, induction or attempted induction of an arrhythmia with right atrial pacing and recording and catheter ablation of arrhythmogenic focus, including intracardiac electrophysiologic 3-dimensional mapping, right ventricular pacing and recording, left atrial pacing and recording from coronary sinus or left atrium, and His bundle recording, when performed; with treatment of supraventricular tachycardia by ablation of fast or slow atrioventricular pathway, accessory atrioventricular connection, cavo-tricuspid isthmus or other single atrial focus or source of atrial re-entry
93654	Comprehensive electrophysiologic evaluation with insertion and repositioning of multiple electrode catheters, induction or attempted induction of an arrhythmia with right atrial pacing and recording and catheter ablation of arrhythmogenic focus, including intracardiac electrophysiologic 3-dimensional mapping, right ventricular pacing and recording, left atrial pacing and recording from coronary sinus or left atrium, and His bundle recording, when performed; with treatment of ventricular tachycardia or focus of ventricular

Related Information

Definition of Terms

Sudden cardiac arrest/Sudden cardiac death (SCA/SCD): Sudden cardiac death is caused by a sudden cardiac arrest. Sudden cardiac arrest is the result of abnormal function of the heart's electrical system, causing the heart to suddenly beat too fast or without synchrony, impairing the heart's ability to sufficiently pump blood. Thus, sudden altered electrical activity in the heart results in a lack of blood flow throughout the body, causing brain and other organ damage, and can result in death.

Non-Ischemic cardiomyopathy (NICM): Problems with heart muscle function not related to restricted blood flow in the coronary arteries.

Hypertrophic cardiomyopathy (HCM): A genetic condition characterized by the thickening of the heart muscle, particularly affecting the ventricles. This thickening can lead to complications such as heart failure, arrhythmias, and even sudden cardiac death. One type of NICM.

Left Ventricular Ejection Fraction (LVEF): A measure of the effectiveness of the pumping action of the heart.

Implantable Cardioverter-Defibrillator (ICD): A battery powered device connected to the heart that is implanted in the body to detect and counteract arrhythmias if they occur.

Early repolarization syndrome: Individuals with imbalances between epi- and endo-cardial layers that result in dispersion of de- and repolarization. In these individuals, J waves or ST segment elevations can be observed on surface ECGs as manifestations of those current imbalances. Although an early repolarization pattern is relatively frequently found on surface ECGs in the overall population, the majority of individuals presenting with an early repolarization pattern will remain asymptomatic and the isolated presence of an early repolarization pattern does not require further intervention.

Catecholaminergic polymorphic ventricular tachycardia: An inherited genetic disorder that predisposes those affected to potentially life-threatening abnormal heart rhythms or arrhythmias. The arrhythmias typically occur during exercise or at times of emotional stress and classically take the form of bidirectional ventricular tachycardia or ventricular fibrillation.

Short QT syndrome (SQT): A very rare genetic disease of the electrical system of the heart and is associated with an increased risk of abnormal heart rhythms and sudden cardiac death.

Long QT syndrome (LQT): A condition affecting repolarization of the heart after a heartbeat, giving rise to an abnormally lengthy QT interval. It results in an increased risk of an irregular heartbeat which can result in fainting, drowning, seizures, or sudden death.

Brugada syndrome: An autosomal-dominant inherited arrhythmic disorder characterized by ST elevations with successive negative T waves in the right precordial leads without structural cardiac abnormalities. These individuals are at risk for sudden cardiac death (SCD) due to ventricular fibrillation (VF). Mutations in the SCN5A gene represent the most common genotype but mutations in additional genes have also been associated with Brugada syndrome.

Congenital heart disease (CHD): A general term describing abnormalities in the structure of the heart that are present at birth. The abnormalities can include abnormal heart valves or abnormal communications between the different chambers of the heart.

Congestive heart failure (CHF): A condition in which the pumping action of the heart no longer works properly. This results in congestion of blood in the lungs and other organs.

Supraventricular tachycardia (SVT): An umbrella term used to describe tachycardias (atrial and/or ventricular rates in excess of 100 bpm at rest), the mechanism of which involves tissue from the His bundle or above. These SVTs include inappropriate sinus tachycardia, AT (including focal and multifocal AT), macro-reentrant AT (including typical atrial flutter), junctional tachycardia, AVNRT, and various forms of accessory pathway-mediated reentrant tachycardias. In this policy, the term does not include AF.

Paroxysmal supraventricular tachycardia (PSVT): A clinical syndrome characterized by the presence of a regular and rapid tachycardia of abrupt onset and termination. These features are characteristic of AVNRT or AVRT, and, less frequently, AT. PSVT represents a subset of SVT.

Atrial fibrillation (AF): A supraventricular arrhythmia with uncoordinated atrial activation and, consequently, ineffective atrial contraction. ECG characteristics include: 1) irregular atrial activity, 2) absence of distinct P waves, and 3) irregular R-R intervals (when atrioventricular conduction is present).

Sinus tachycardia: Rhythm arising from the sinus node in which the rate of impulses exceeds 100 bpm.

Physiologic sinus tachycardia: Appropriate increased sinus rate in response to exercise and other situations that increase sympathetic tone.

Inappropriate sinus tachycardia: Sinus heart rate greater than 100 bpm at rest, with a mean 24-h heart rate greater than 90 bpm not due to appropriate physiological responses or primary causes such as hyperthyroidism or anemia.

Atrial tachycardia (AT)

Focal AT: An SVT arising from a localized atrial site, characterized by regular, organized atrial activity with discrete P waves and typically an isoelectric segment between P waves. At times, irregularity is seen, especially at onset (“warm-up”) and termination (“warm-down”). Atrial mapping reveals a focal point of origin.

Sinus node reentry tachycardia: A specific type of focal AT that is due to microreentry arising from the sinus node complex, characterized by abrupt onset and termination, resulting in a P-wave morphology that is indistinguishable from sinus rhythm.

Multifocal atrial tachycardia (MAT): An irregular SVT characterized by 3 or more distinct P-wave morphologies and/or patterns of atrial activation at different rates. The rhythm is always irregular.

Atrial flutter

Cavo-tricuspid isthmus–dependent atrial flutter: typical: Macro-reentrant AT propagating around the tricuspid annulus, proceeding superiorly along the atrial septum, inferiorly along the right atrial wall, and through the cavo-tricuspid isthmus between the tricuspid valve annulus and the Eustachian valve and ridge. This activation sequence produces predominantly negative "sawtooth" flutter waves on the ECG in leads 2, 3, and aVF and a late positive deflection in V1. The atrial rate can be slower than the typical 300 bpm (cycle length 200 ms) in the presence of antiarrhythmic drugs or scarring. It is also known as "typical atrial flutter" or "cavo-tricuspid isthmus–dependent atrial flutter" or "counterclockwise atrial flutter."

Cavo-tricuspid isthmus–dependent atrial flutter: reverse typical: Macroreentrant AT that propagates around in the direction reverse that of typical atrial flutter. Flutter waves typically appear positive in the inferior leads and negative in V1. This type of atrial flutter is also referred to as "reverse typical" atrial flutter or "clockwise typical atrial flutter."

Atypical or non–cavo-tricuspid isthmus–dependent atrial flutter: Macro-reentrant ATs that do not involve the cavo-tricuspid isthmus. A variety of reentrant circuits may include reentry around the mitral valve annulus or scar tissue within the left or right atrium. A variety of terms have been applied to these arrhythmias according to the re-entry circuit location, including forms, such as "LA flutter" and "LA macro-reentrant tachycardia" or incisional atrial re-entrant tachycardia due to re-entry around surgical scars.

Junctional tachycardia: A non-reentrant SVT that arises from the AV junction (including the His bundle).

Atrioventricular nodal reentrant tachycardia (AVNRT): A reentrant tachycardia involving 2 functionally distinct pathways, generally referred to as "fast" and "slow" pathways. Most commonly, the fast pathway is located near the apex of Koch's triangle, and the slow pathway inferoposterior to the compact AV node tissue. Variant pathways have been described, allowing for "slow-slow" AVNRT.

Typical AVNRT: AVNRT in which a slow pathway serves as the anterograde limb of the circuit and the fast pathway serves as the retrograde limb (also called "slow-fast AVNRT")

Atypical AVNRT: AVNRT in which the fast pathway serves as the anterograde limb of the circuit and a slow pathway serves as the retrograde limb (also called "fast-slow AV node reentry") or a

slow pathway serves as the anterograde limb and a second slow pathway serves as the retrograde limb (also called "slow-slow AVNRT").

Accessory pathway: An accessory pathway is defined as an extranodal AV pathway that connects the myocardium of the atrium to the ventricle across the AV groove. Accessory pathways can be classified by their location, type of conduction (decremental or non-decremental), and whether they can conduct anterogradely, retrogradely, or in both directions.

Manifest accessory pathways: A pathway that conducts anterogradely to cause ventricular pre-excitation pattern on the ECG.

Concealed accessory pathway: A pathway that conducts only retrogradely and does not affect the ECG pattern during sinus rhythm.

Pre-excitation pattern: An ECG pattern reflecting the presence of a manifest accessory pathway connecting the atrium to the ventricle. Pre-excited ventricular activation over the accessory pathway competes with the anterograde conduction over the AV node and spreads from the accessory pathway insertion point in the ventricular myocardium. Depending on the relative contribution from ventricular activation by the normal AV nodal/His Purkinje system versus the manifest accessory pathway, a variable degree of pre-excitation, with its characteristic pattern of a short P-R interval with slurring of the initial upstroke of the QRS complex (delta wave), is observed. Pre-excitation can be intermittent or not easily appreciated for some pathways capable of anterograde conduction; this is usually associated with a low-risk pathway, but exceptions occur.

Asymptomatic pre-excitation (isolated pre-excitation): The abnormal pre-excitation ECG pattern in the absence of documented SVT or symptoms consistent with SVT. Wolff-Parkinson-White syndrome. Syndrome characterized by documented SVT or symptoms consistent with SVT in a patient with ventricular preexcitation during sinus rhythm. Atrioventricular reentrant tachycardia (AVRT) A reentrant tachycardia, the electrical pathway of which requires an accessory pathway, the atrium, atrioventricular node (or second accessory pathway), and ventricle.

Orthodromic AVRT: An AVRT in which the reentrant impulse uses the accessory pathway in the retrograde direction from the ventricle to the atrium, and the AV node in the anterograde direction. The QRS complex is generally narrow or may be wide because of pre-existing bundle-branch block or aberrant conduction.

Antidromic AVRT: An AVRT in which the reentrant impulse uses the accessory pathway in the anterograde direction from the atrium to the ventricle, and the AV node for the retrograde direction. Occasionally, instead of the AV node, another accessory pathway can be used in the

retrograde direction, which is referred to as pre-excited AVRT. The QRS complex is wide (maximally pre-excited).

Permanent form of junctional reciprocating tachycardia (PJRT): A rare form of nearly incessant orthodromic AVRT involving a slowly conducting, concealed, usually posteroseptal accessory pathway.

Pre-excited AF: AF with ventricular pre-excitation caused by conduction over 1 or more accessory pathway(s).

Ventricular Tachycardia: A rapid arrhythmia arising from the ventricles of the heart that is potentially life-threatening if prolonged.

Evidence Review

Background

Electrophysiology (EP) studies are invasive procedures generally performed in a dedicated EP laboratory. In addition to the cardiac electrophysiologist, several other staff members are required. Intravenous conscious sedation is typically used, although in some situations such as with prolonged catheter ablation procedures, general anesthesia may be used.

EP studies can be used to induce sustained ventricular arrhythmias (VA) in patients with known or suspected VA. With the advent of the ICD and its proven benefit in the primary and secondary prevention of sudden cardiac death (SCD), there are fewer indications for programmed stimulation to provoke VA. Patients with heart failure and LVEF less than 35% generally will have an indication for an ICD and specific induction of ventricular tachycardia/ventricular fibrillation (VT/VF) before implantation is not necessary. Patients with LVEF greater than 35% and unexplained syncope or near-syncope may benefit from an electrophysiological study to determine if VT/VF is the cause of symptoms and to guide further therapy. Induction of VT/VF is often attempted before catheter ablation of the arrhythmia substrate to guide the procedure and to determine the success of the intervention after ablation is performed. An EP study can be used to determine the mechanism of a wide complex tachycardia.

EP studies can also be used to risk stratify patients with conditions that have a high risk for ventricular arrhythmias

Summary of Evidence

Syncope

Diagnostic results detected during EPS occur predominantly in patients who have cardiac disease (e.g., conduction system delay, coronary artery disease, cardiomyopathy, and valvular heart disease). Most of the literature evaluating EPS as a means to diagnose syncope is relatively old, and the data were obtained in referral centers where there was a high pretest probability of an arrhythmia. Eight of these small retrospective studies (total n=625) found that, of the 406 patients with cardiac disease or an abnormal ECG, 41% had a positive result (of these, 21% had VT and 34% had a bradycardia). Of 219 patients without evidence of heart disease, only 5% had a positive result (1% with VT and 10% with evidence of substrate for symptomatic bradycardia). Overall, the diagnostic yield of EPS was approximately 50% and 10% in patients with and without structural heart disease, respectively[2]. The evidence is sufficient to determine that the technology results in an improvement in the net health outcome.

In patients with cardiac sarcoidosis, programmed electrical stimulation may help identify patients at risk of having VA. According to a study of 76 patients with cardiac sarcoidosis and no cardiac symptoms, 8 (11%) had inducible sustained VA. During a median follow-up of 5 years, 6 of 8 had VA or died, versus 1 of 68 in the non-inducible group[2]. The evidence is sufficient to determine that the technology results in an improvement in the net health outcome.

The value of EPS in assessing the mechanism of syncope in patients with Brugada is unknown. In large registries of patients with Brugada, inducibility of VA was higher among patients with a prior history of syncope or SCA. However, the value of EPS in predicting prognosis in patients with Brugada is essentially unknown in patients with syncope. The role of inducibility of VA in identifying high-risk patients remains controversial[2]. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

Risk Stratification

A study of EP testing in patients with symptomatic NICM found inducible VT/VF in 28% of patients which was associated with a higher rate of ICD events during follow-up[5]. In a prospective cohort of 180 patients with ischemic or NICM and syncope, induction of VT or VF at electrophysiological study correlated with cardiac mortality only in patients with ischemic heart

disease. In patients with NICM, cardiac mortality correlated with LVEF but not with inducibility on electrophysiological study[6].

In patients who meet criteria for ICD implantation, data do not support the routine use of electrophysiological study solely for risk stratification, as such patients have been shown to derive survival benefit from the ICD[7,8]. An EP study may be helpful, however, in selected patients suspected to have preexcitation or supraventricular arrhythmias as the cause of symptoms or wide complex tachycardias that warrant definitive diagnosis and management. SVT leading to VT/VF or aberrantly conducted SVT may also be suspected in younger patients or those with a preserved LVEF. Induction of SVT and ablation may then be curative, with no need for an ICD. In such cases, failure to induce VT/VF after elimination of the substrate for SVT would be expected. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

Risk stratification for channelopathies is generally made on the basis of symptoms, the ECG[9-16], exercise treadmill testing[17-19], and the results of genetic testing[20-24]. EP studies do not have prognostic value for risk stratification in patients with these cardiac channelopathies[25-28]. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

In individuals with pre-excitation who are asymptomatic, a clinical priority is identifying accessory pathways at increased risk of arrhythmic events, including rapid conduction during AF and development of life-threatening ventricular arrhythmias, with the most useful findings being the following: an R-R interval <250 ms between 2 pre-excited complexes during induced AF; the presence of multiple accessory pathways; the ability to induce sustained AVRT; the finding of AVRT precipitating pre-excited AF; and an accessory pathway refractory period <240ms. Malignant arrhythmias correlate more with the EP properties of the accessory pathway than with the presence or absence of symptoms. This approach is supported by the low risk of complications observed in an EP study in which complication rates among 2,169 patients ranged from 0.09% to 1% and included pneumothorax and access site complications[3]. The evidence is sufficient to determine that the technology results in an improvement in the net health outcome.

Practice Guidelines and Position Statements

The purpose of the following information is to provide reference material. Inclusion does not imply endorsement or alignment with the policy conclusions.

Table 1. 2018 European Society of Cardiology Guidelines[4]

Recommendations	Class[a]	Level of evidence[b]
In patients with syncope and previous myocardial infarction, or other scar-related conditions, EPS is indicated when syncope remains unexplained after non-invasive evaluation	I	B
In patients with syncope and bifascicular BBB, EPS should be considered when syncope remains unexplained after non-invasive evaluation	IIa	B
In patients with syncope and asymptomatic sinus bradycardia, EPS may be considered in a few instances when non-invasive tests (e.g. ECG monitoring) have failed to show a correlation between syncope and bradycardia	IIb	B
In patients with syncope preceded by sudden and brief palpitations, EPS may be considered when syncope remains unexplained after non-invasive evaluation.	IIb	C

[a] I: is recommended/indicated, IIa: should be considered, IIb: may be considered, III: is not recommended.

[b] A: Data derived from multiple randomized clinical trials or meta-analyses, B: Data derived from a single randomized trial or large non-randomized studies, C: Consensus opinion of experts and/or small studies, retrospective studies, registries.

Medicare National Coverage

There is no national coverage determination.

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History

Date	Comments
12/01/25	New policy, approved November 11, 2025, effective for dates of service on or after March 4, 2026, following 90-day provider notification. Add to Cardiology section.

Date	Comments
	Electrophysiology studies may be considered medically necessary when criteria are met.

Disclaimer: This medical policy is a guide in evaluating the medical necessity of a particular service or treatment. The Company adopts policies after careful review of published peer-reviewed scientific literature, national guidelines and local standards of practice. Since medical technology is constantly changing, the Company reserves the right to review and update policies as appropriate. Member contracts differ in their benefits. Always consult the member benefit booklet or contact a member service representative to determine coverage for a specific medical service or supply. CPT codes, descriptions and materials are copyrighted by the American Medical Association (AMA).

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