Guidelines for Clinical Cardiac Electrophysiologic Studies (JCS 2011)

– Digest Version –

JCS Joint Working Group

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Introduction of the Revised Guidelines

Treatment strategies for severe arrhythmias have evolved significantly based on the recent development of non-pharmacological procedures such as catheter ablation, implantable cardioverter defibrillator (ICD), and cardiac resynchronization therapy (CRT). Because of the invasiveness of these procedures, their indications should be carefully determined after thorough consideration of their short and long-term effects. Since the results of large-scale clinical studies in Europe and the United States have suggested that ICD/CRT be used for more patients for the primary prevention of sudden death in Japan, appropriate risk stratification is required to determine indications of these procedures.

The history of clinical electrophysiologic studies (EPS) began when Scherlag et al. established His bundle electrogram in 1969. Use of His bundle electrogram dominated in the evaluation of sinus node function and atrioventricular (AV) conduction during that era. In addition to the His bundle electrogram, the premature stimulation technique significantly contributed to the understanding of the pathogenesis of arrhythmias, and was established as an important method to evaluate the efficacy of antiarrhythmic drugs. This method was also used to identify arrhythmic foci, which stimulated the development of ablation therapy.

Under these circumstances, “the Guidelines for Clinical Cardiac Electrophysiologic Studies” was published in 2006 (2004–2005 Joint Working Groups Report, Chair: Iwao Yamaguchi) to provide guidance to physicians in Japan regarding the use of cardiac EPS in the diagnosis, selection of treatment strategies, and prediction of prognosis in patients with arrhythmias.¹ The present edition of the guidelines was prepared as a partial revision that is undertaken once every five years. For the present edition, a new Joint Working Groups was organized and has worked for one year with the following considerations in mind: 1) Contents of this guideline should be consistent with those of “the Guidelines for Non-Pharmacotherapy of Cardiac Arrhythmias” and “the Guidelines for Indications and Procedures of Catheter Ablation” which were under preparation by

the Japanese Circulation Society, and overlap with these guideline documents should be avoided; 2) the authors who wrote chapters for the previous edition revised, and, if not possible, specialists with expertise in the relevant therapeutic areas revised each chapter; 3) substantial modifications were made for the chapters regarding therapeutic areas where new understandings of a disease have changed its diagnosis and treatment during the past five years. For example, regarding “Ventricular Fibrillation” a new section “Idiopathic Ventricular Fibrillation” was added to separate it from “Ventricular Fibrillation Associated with Structural Heart Diseases” because new concepts such as early repolarization syndrome and short QT syndrome have been incorporated in the field. On the other hand, for Brugada syndrome and some other conditions, the indications for EPS remained almost unchanged although new authors were involved since no consensus was achieved regarding the significance of clinical EPS in determining treatment strategies despite large amounts of new data were added. The section for the identification of ablation sites for the treatment of chronic atrial fibrillation (AF) was modified by the author who wrote the section in the previous version to reflect the change in indications for EPS to identify ablation sites in the treatment of chronic AF for which catheter ablation has become increasingly common.

Indications for EPS (indications by class) are listed in the same format as before in this revision. However, for the points where the members of Joint Working Groups could not reach agreement due to the lack of sufficient evidence, the present view was included upon the discussion among the members for which further revision may be necessary.

It is difficult to decide which should be included in a guideline document to be revised every five years regarding those therapeutic areas that are rapidly evolving. The present guidelines are the accumulation of wisdom of the members of the Joint Working Groups, who are top experts actively engaged in the area of arrhythmias and cardiac electrophysiology. Readers should use this guideline accordingly.

I Equipment, Technology and Knowledge Required in Electrophysiologic Studies

1. Equipment, Technology and Knowledge Required in Electrophysiologic Studies²

Although many types of electrode catheters are available for cardiac EPS, all electrode catheters share the same basic function of recording intracardiac electrical potentials and stimulating heart muscles. Physicians should select appropriate electrode catheters according to the purpose of the test. The test requires an X-ray machine, a recorder to record surface electrocardiography (ECG) and intracardiac electrical potentials, and a stimulator to determine refractory periods and induce tachycardia (Table 1).³ A defibrillator and emergency resuscitation equipment should be prepared for use. EPS are performed by a team of physicians, nurses, radiology technicians and/or clinical engineers.

Physicians who perform EPS should have expertise in inserting and manipulating electrode catheters and have extensive knowledge of clinical electrophysiology (especially on cardiac conduction and refractory period) (Table 2). The stimulator is

used to induce arrhythmias, and the results are used to diagnose arrhythmias and develop treatment strategies for each case. EPS must be performed carefully by preparing for rare but serious complications.

2. Radiation Exposures

EPS may cause radiation exposure not only to the patient but also to the staff members in the laboratory. The amount of radiation exposure increases with the time spent during the test. Usually, the total exposure time rarely exceeds 10 minutes, and the amount of radiation exposure during EPS is smaller than that during coronary angiography.⁴ Since a posterior-anterior projection is commonly used in EPS, erythema and necrosis of the skin of the back may develop.⁵ The threshold level of radiation dose that causes radiation-induced skin injuries is 2 Gy, and the absorbed dose in the skin reaches to 2 Gy when the patient received X-rays from a conventional X-ray machine for a total of about 60 minutes.⁶ An EPS requires at most 10

Table 1. Equipment Required for EPS

Equipment	Role
X-ray machines: Common X-ray machines used for cardiac catheterization	Machines for two-dimensional cinefluoroscopy are useful for ablation since they do not have to move around.
Recorders and electrocardiographic monitors	Data should be stored either on magnetic tape or optical discs. The display of electrocardiographic data should be adjusted to ensure visibility.
Stimulators	Programmable stimulators that can produce extra or continuous stimulation and have at least two output terminals are preferable.
Ablation systems	Ablation systems should be able to set the temperature during energy delivery and the amount of energy delivered, and monitor the temperature and change in impedance during ablation.
Defibrillators	Defibrillators are necessary to treat ventricular fibrillation and atrial fibrillation.
External pacing devices	External pacing devices are necessary for temporary pacing.
Emergency resuscitation devices/drugs	Emergency resuscitation devices/drugs include intratracheal tubes, Ambu bags, and drugs for emergency care.

EPS, cardiac electrophysiologic studies.

Adapted from Fujiki A. Electrophysiologic Studies: techniques and devices. In: Inoue H, Okumura K, editors. Clinical Cardiac Electrophysiologic Studies. p24. Igaku-Shion Ltd., Tokyo, 2002.³

Table 2. Technical Skills Needed to Perform EPS

- Operational skills to perform right and left heart catheterization with percutaneous techniques through vascular access (including techniques such as intracardiac pressure recording, coronary angiography, and ventriculography).
- Manual dexterity to safely place and manipulate electrode catheters in the appropriate chambers to access the target location (such as the His bundle area, coronary sinus, outflow tract, and annular area).
- Ability to record intracardiac electrocardiographic signals, determine conduction intervals and refractory periods, induce and terminate tachycardia, and identify reentrant circuits with programmed stimulation.
- Ability to recognize and manage procedural complications.
- Proficiency in the use of external defibrillation.
- Proficiency in the use of antiarrhythmic drugs.
- The use of intravenous anesthesia.
- Emergency procedures.

EPS, cardiac electrophysiologic studies.

minutes of radiation exposure, and the radiation exposure to the skin will not cross the threshold.

Staff members are also exposed to X-rays in the laboratory due to X-ray scattering in every direction. Therefore, staff members should wear protective clothing which also covers their back. The operator who handles electrode catheters should wear a neck guard and goggles as well, and should be careful not to expose his/her hands and arms directly to X-rays. Radiation

exposure to the patient and staff members should be minimized using appropriate X-ray techniques (e.g., narrowing the radiation field, reducing X-ray exposure time, avoiding magnified imaging, and holding the image intensifier close to the patient), appropriate equipment setting (e.g., using pulse radiation, a low image acquisition rate, and non-radiological devices), and appropriate protection measures.⁷

II Sinus Node Function

(Indications by class are listed in [Table 3](#).)

Sinus node function is influenced by three factors, i.e., sinus node automaticity, sinoatrial conductivity, and the activity of the autonomic nerves affecting sinus node. The electrical impulse generated in the sinus node travels through the sinoatrial junction to the atria.

Sick sinus syndrome (sinus node dysfunction) is characterized by bradycardia due to abnormal sinus node automaticity or conductivity that causes central nervous system symptoms such as syncope and blackouts.

Increased sinus rate caused by enhanced sinus node automaticity may be observed in patients with sympathicotonia, fever, hyperthyroidism and inappropriate sinus tachycardia (IST) or other conditions.

Sick sinus syndrome is a clinical term used to describe signs

and symptoms of sinus node dysfunction. In order to diagnose the condition, the relationship between sinus bradycardia and the symptoms must be confirmed. Patients in whom the relationship between bradycardia and symptoms can be confirmed by long-term Holter ECG monitoring or other tests are not indicated for EPS. For patients who are suspected to have sinus node dysfunction but in whom the relationship between ECG findings and symptoms is not clear, noninvasive, long-term monitoring such as frequent use of Holter ECG monitoring, wireless ECG monitoring, and ambulatory ECG is performed to obtain ECG findings during the onset of symptoms. Electrophysiologic evaluation of sinus node function to determine sinus node recovery time (SNRT), sinoatrial conduction time, and intrinsic heart rate is beneficial when no definitive findings have been obtained with noninvasive assessment. During

Table 3. Indications for EPS in Patients With Sinus Node Dysfunction**Class I**

1. Patients with sinus node dysfunction who have symptoms such as syncope, dizziness, and blackouts, and in whom the relationship between sinus node dysfunction and the symptoms has not been confirmed with noninvasive testing such as ECG and Holter ECG monitoring.

Class IIa

1. Patients with sinus node dysfunction who have symptoms such as syncope, dizziness, and blackouts, in whom the relationship between sinus node dysfunction and the symptoms has been confirmed with noninvasive testing such as ECG and Holter ECG monitoring, and who are complicated with other disorders such as AV conduction disturbances and tachycardia.
2. Patients with bradycardia-tachycardia syndrome in whom administration of antiarrhythmic drugs exacerbates bradycardia although the use of the drugs is absolutely imperative.
3. Patients with asymptomatic sinus node dysfunction who need treatment with drugs that may exacerbate sinus node dysfunction.

Class IIb

1. Patients with sinus node dysfunction who have symptoms such as syncope, dizziness, and blackouts, in whom the relationship between sinus node dysfunction and the symptoms has been confirmed with noninvasive testing such as ECG and Holter ECG monitoring, and in whom sinus node dysfunction is suspected to be caused by drugs used for the treatment of other underlying diseases.
2. Patients with suspected sinus node dysfunction in whom antiarrhythmic drugs may indicate sinus node dysfunction.

Class III

1. Patients with sinus node dysfunction who have symptoms such as syncope, dizziness, and blackouts, in whom the relationship between sinus node dysfunction and the symptoms has been confirmed with noninvasive testing such as ECG and Holter ECG monitoring, and who are not complicated with other disorders such as AV conduction disturbances and tachycardia.
2. Patients with asymptomatic sinus bradycardia.

AV, atrioventricular; ECG, electrocardiography; EPS, cardiac electrophysiologic studies.

Table 4. Indications for EPS in Patients With AV Block**Class I**

1. Patients with suspected AV block as the cause of symptoms such as syncope, dizziness, and blackouts in whom the relationship between AV block and these symptoms is unclear.
2. Patients with second or third degree AV block who are implanted with a pacemaker and still have symptoms such as syncope, dizziness, and blackouts and in whom other arrhythmias are suspected as the cause of the symptoms.

Class IIa

1. Patients with AV block who are indicated for a pacemaker and who need to be evaluated for sinus node function.
2. Patients with Mobitz type II second or third degree AV block and bifascicular or trifascicular block in whom the sites of block must be identified and assessment of sinus node function is necessary.

Class IIb

1. Patients with asymptomatic AV block who need to receive treatment with drugs that may exacerbate conduction disturbance.

Class III

1. Patients in whom ECG findings demonstrated the relationship between AV block and symptoms such as syncope, dizziness, and blackouts.
2. Asymptomatic patients with first degree AV block or Wenckebach type second degree AV block.

AV, atrioventricular; ECG, electrocardiography; EPS, cardiac electrophysiologic studies.

EPS patients should also be assessed for the presence/absence of supraventricular tachyarrhythmia, AV conduction disturbance, and retrograde ventriculoatrial conduction to obtain information useful for the determination of treatment strategies, drug regimens and pacing modes. EPS are generally not necessary for patients in whom the relationship between bradycardia and symptoms has been confirmed with noninvasive methods such as ECG and Holter ECG monitoring and who

are not complicated with AV conduction disturbance or tachycardia, or have only asymptomatic sinus bradycardia.

A new minimally-invasive ECG monitoring method using an implantable loop recorder implanted under the skin in the left anterior chest has been introduced to obtain detailed ECG data during syncope in patients with syncope of unknown etiology.

III Atrioventricular Block

(Indications by class are listed in [Table 4](#).)

AV block is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles through the AV node, His bundle and His-Purkinje system. The severity of AV block is determined not only by ECG pattern but also by the sites of block. The severity is high especially among patients where the conduction is blocked within or below the His

bundle since these blocks may induce more severe conduction block or represent unstable lower pacemakers that generate escape rhythms. It is therefore important to locate the sites of block in predicting prognosis and determining treatment strategies. Since there is a limit to locate the sites of block by using the standard 12-lead ECG, Holter ECG monitoring or exercise ECG, EPS is necessary for patients with AV block.

IV Bundle Branch Block and Intraventricular Conduction Disturbance

(Indications by class are listed in [Table 5](#).)

The intraventricular conduction system consists of the His bundle, right bundle branch, left bundle branch and Purkinje fibers. Although the geometry of the left bundle branch can vary greatly among individuals, it generally subdivides into the anterior and posterior fascicles in the interpretation of ECG. [Table 6](#) lists the criteria for ECG-based diagnosis of intraventricular conduction disturbance.⁸ A blockage in either the right or left bundle of the conducting pathway is called bundle branch

block. A blockage in either the left anterior or posterior fascicle is called fascicular block. Bifascicular (bilateral bundle branch) block shows up on the ECG as a combination of characteristic wave patterns of right bundle branch block in conjunction with either left anterior or posterior fascicular block. A conduction disturbance in all three fascicles is called trifascicular block, and shows up on the ECG as a mixture of waveforms of bifascicular block and either first or second degree AV block. EPS using intracardiac electrogram recording and

Table 5. Indications for EPS in Patients With Bundle Branch Block and Intraventricular Conduction Disturbance

Class I
1. Patients with bundle branch block or intraventricular conduction delay who have cerebral ischemic symptoms of unknown etiology such as syncope, convulsion, dizziness and light-headed feeling.
2. Patients with wide QRS tachycardia who require differentiation of supraventricular tachycardia associated with bundle branch block or intraventricular conduction disturbance from ventricular tachycardia.
Class IIa
None.
Class IIb
1. Asymptomatic patients with bundle branch block who are expected to receive drugs that may exacerbate conduction disturbance or induce AV block.
2. Asymptomatic patients with intraventricular conduction disturbance.
Class III
1. Symptomatic patients in whom the relationship between their symptoms and intraventricular conduction disturbance has been ruled out by ECG or other available data.

AV, atrioventricular; ECG, electrocardiography; EPS, cardiac electrophysiologic studies.

Table 6. Criteria for ECG-Based Diagnosis of Intraventricular Conduction Disturbance

1. Complete right bundle branch block
1) A QRS interval of more than 0.12 sec in the lead with the greatest QRS width
2) The presence of rSR' pattern (or sometimes Rsr' or Rr' pattern) in lead V ₁ and negative T waves
3) The presence of a wide S wave at the end of the QRS complex in lead V ₅ , V ₆ or I, and late R waves in lead aVR
2. Incomplete right bundle branch block
1) A QRS interval of less than 0.12 sec in the lead with the greatest QRS width
2) The presence of rsr' pattern (or sometimes rsR pattern) in lead V ₁ and negative T waves
3. Complete left bundle branch block
1) A QRS interval of more than 0.12 sec in the lead with the greatest QRS width
2) The presence of wide notched or slurred R waves in lead V ₅ , V ₆ or I
3) The absence of Q waves in lead I, V ₅ or V ₆
4) A wide S wave at the end of the QRS complex in lead V ₁ or V ₂
4. Incomplete left bundle branch block
1) A QRS interval of less than 0.12 sec in the lead with the greatest QRS width
2) The presence of wide notched or slurred R waves in lead V ₅ , V ₆ or I
3) The absence of Q waves in lead I, V ₅ or V ₆
5. Left anterior fascicular block
1) A QRS interval of less than 0.12 sec in the lead with the greatest QRS width
2) A leftward deviation of the QRS axis by – 45 degrees or more
3) The presence of qR pattern (or R pattern) in lead I or aVL
4) The presence of rS pattern in lead II, III, or aVF
6. Left posterior fascicular block
1) A QRS interval of less than 0.12 sec in the lead with the greatest QRS width
2) The absence of right ventricular hypertrophy, pulmonary emphysema, extensive lateral wall infarction, or vertical heart, and a rightward deviation of the QRS axis by +110 degrees or more
3) The presence of rS pattern in lead I or aVL
4) The presence of qR pattern in lead III or aVF
7. Right bundle branch block with left anterior fascicular block
The presence of findings 1) and 2) of complete right bundle branch block, and findings 2) and 3) of left anterior fascicular block
8. Right bundle branch block with left posterior fascicular block
The presence of findings 1) and 2) of complete right bundle branch block, and findings 2) and 4) of left posterior fascicular block
9. Nonspecific intraventricular conduction disturbance
A QRS interval of more than 0.12 sec with no findings suggestive of right or left bundle branch block

ECG, electrocardiography.

Table 7. Indications for EPS in Patients With WPW Syndrome

Class I
1. Patients being evaluated for catheter ablation or surgical ablation of an accessory pathway.
2. Patients who have survived cardiac arrest or who have syncopal attacks of unknown etiology.
3. Symptomatic patients in whom determination of the mechanism of arrhythmia or knowledge of the electrophysiologic properties of the accessory pathway and normal conduction system would help in determining appropriate therapy.
Class IIa
1. Asymptomatic patients who have a family history of sudden cardiac death or engage in high-risk occupations or activities that affect public safety when a serious attack develop and in whom knowledge of the electrophysiologic properties of the accessory pathway or presence/absence of inducible tachycardia may help determine recommendations for further activities or appropriate therapy.
Class IIb
1. Patients who are undergoing cardiac surgery for other reasons.
2. Asymptomatic patients who are willing to undergo EPS.
Class III
1. Asymptomatic patients except those in Class IIa and IIb above.

EPS, electrophysiologic studies; WPW, Wolff-Parkinson-White.

cardiac electrical stimulation are useful to predict the risk of AV block among patients with bundle branch block and/or intraventricular conduction disturbance.⁹ The presence of conduction disturbance may be determined when at least one of the following criteria are met:

1. An HV interval of 55 msec or more for cases of bifascicular or trifascicular block.
2. The presence of HV block induced by continuous atrial stimulation at 150 bpm or less.¹⁰
3. An effective refractory period of the His-Purkinje system of 450 msec or more.
4. At least a two-fold prolongation of the HV interval from baseline or a prolongation of the HV interval by 100 msec or more (the HV interval may prolong by 20% or less under normal condition), or induction of second or third degree block within or below the His bundle following a procainamide loading (300 to 1,000 mg intravenous injection [I.V.]); or induction of second or third degree block by continuous ventricular stimulation at baseline or following administration of procainamide or lidocaine (1 to 2 mg/kg I.V.).
5. Induction of ventricular tachycardia (VT) or ventricular fibrillation (VF) by programmed ventricular stimulation.

V Preexcitation Syndrome

(Indications by class are listed in [Table 7](#).)

American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for Clinical Intracardiac Electrophysiological and Catheter Ablation Procedures were published in 1995, and have been widely used as a guide for clinical EPS in patients with Wolff-Parkinson-White (WPW) syndrome.¹¹ However, indications for EPS in this patient population may be changed when the safety of EPS is improved, the diagnostic criteria to specify high-risk patients are established, and the positive predictive value for sudden death is increased.¹² Although the prevalence of sudden death is low among patients with WPW syndrome,^{13–15} it has been reported that 10% of young patients who resuscitated from cardiac arrest have WPW

syndrome.¹⁶ A broad consensus has been reached on the usefulness of EPS in patients with WPW syndrome who have experienced cardiac arrest, syncopal attacks of unknown etiology, and/or tachycardia attacks.

EPS may be indicated for symptomatic patients with Lown-Ganong-Levine (LGL) syndrome, a condition characterized by tachycardia, but are generally not indicated for asymptomatic patients with LGL syndrome who only show a short PR interval without tachycardia.¹⁷

Indications for EPS in patients with atypical WPW syndrome where the involvement of the Mahaim fiber is suspected should be in accordance with those in overt WPW syndrome.

VI Supraventricular Tachycardia Other Than Atrioventricular Reciprocating Tachycardia

(Indications by class are listed in [Table 8](#).)

Paroxysmal supraventricular tachycardia not involving AV accessory pathways include tachycardias originated from atrial muscle (including the sinoatrial node) other than atrial flutter/AF, and tachycardias originated from the AV junction.¹⁸ Since the safety and efficacy of radiofrequency catheter ablation in the treatment of AV nodal reentrant tachycardia (AVNRT), the most common form of this type of tachycardias, have been established, patients with this type of tachycardia are generally indicated for EPS to make a definitive diagnosis prior to cath-

eter ablation. EPS is especially useful for patients with uncommon-type AVNRT in assessing the mechanism and making differential diagnosis. Even patients in whom ablation is not planned often undergo EPS to determine the optimal drug regimen and evaluate the efficacy of the treatment regimen. EPS is indicated for patients with atrial tachycardia (AT) when they are symptomatic and should be assessed in detail to clarify the mechanism and origin of tachycardia to select appropriate drugs.

Table 8. Indications for EPS in Patients With Supraventricular Tachycardia Other Than AV Reciprocating Tachycardia

Class I
1. Patients with symptomatic tachycardia for whom information such as the origin, mechanism and electrophysiologic properties of tachycardia is necessary to determine treatment strategies.
2. Patients who prefer catheter ablation to pharmacotherapy.
3. Patients with tachycardia and symptoms of cerebral ischemia.
Class IIa
1. Patients with episodes of palpitations in whom supraventricular tachycardia is suspected to be the cause on the basis of heart rate or other findings but has not been confirmed on ECG or other testing.
2. Patients with supraventricular tachycardia who require treatment with antiarrhythmic drugs which may impair sinus node function and AV conductivity or other conditions.
3. Diagnosis of asymptomatic tachycardia.
Class IIb
1. Efficacy evaluation of drugs in patients with paroxysmal supraventricular tachycardia.
2. Long-term follow-up after catheter ablation.
Class III
1. Patients with tachycardia that respond favorably to vagal maneuvers and pharmacotherapy, and who are not indicated for ablation.

AV, atrioventricular; ECG, electrocardiography; EPS, electrophysiologic studies.

Table 9. Indications for EPS in Patients With Atrial Flutter

Class I
1. Diagnostic EPS for patients who are suspected to have episodes of tachycardia including atrial flutter that are not documented by ECG.
2. EPS for patients with atrial flutter for the purpose of catheter ablation.
3. EPS for the purpose of rapid atrial pacing to terminate atrial flutter.
Class IIa
1. EPS for patients with uncommon type atrial flutter which is expected to be treated successfully with catheter ablation.
2. EPS for the assessment of persistent atrial flutter that occurred during EPS in patients with other types of supraventricular tachycardias.
Class IIb
1. EPS for patients with atrial flutter in whom catheter ablation or pacing is not planned.
Class III
1. EPS for patients with atrial flutter who do not wish to undergo catheter ablation or pacing.

ECG, electrocardiography; EPS, electrophysiologic studies.

VII Atrial Flutter

(Indications by class are listed in [Table 9](#).)

Atrial flutter is defined as a regular supraventricular tachycardia at an atrial rate of 240 to 440 bpm. Typical atrial flutter, which is called common type atrial flutter, has an atrial rate of around 300 bpm and negative sawtooth-like flutter waves in inferior wall leads. Many cases of common type atrial flutter are cavotricuspid isthmus-dependent atrial flutter. EPS is indicated for patients who have symptoms suggestive of tachycardia attacks but have not been confirmed to have tachycardia on the ECG, and are performed to make diagnosis and assess

the mechanism of tachycardia. When reentrant circuits leading to arrhythmias are identified during EPS, patients are indicated for curative catheter ablation. Patients with ECG documented atrial flutter are indicated for EPS to assess the feasibility of catheter ablation. Patients with atrial flutter who have not responded to or are contraindicated for antiarrhythmic drugs or can't receive direct current shocks are indicated for EPS for the purpose of the termination of atrial flutter by rapid atrial pacing. This procedure may be more effective when class I antiarrhythmic drugs are used concurrently.¹⁹

VIII Atrial Fibrillation

Since the publication of a report indicating that rapid ectopic beats originated in atrial muscle or in large vessels adjacent to the heart (the pulmonary veins in many cases) trigger AF,²⁰ EPS became common procedures for patients with AF. Currently, pulmonary vein mapping is the most frequently used EPS for patients with AF because ablation of pulmonary vein foci has been proven to be the most effective curative treatment of AF. As ablation techniques for AF become more advanced, catheter ablation is being used not only for patients with paroxysmal AF but also for patients with persistent or chronic AF. Since the efficacy of pulmonary vein isolation is insufficient for patients with persistent or chronic AF, new techniques to

target abnormal atrial substrates that play a role in the maintenance of AF, as well as atrial linear ablation and ablation of the ganglionated plexi (GP) have been developed, and the benefits of these techniques have been reported.

It has been reported that electrical disconnection of the pulmonary veins can be confirmed by placing a circular catheter in the pulmonary vein. The entrance block from the left atrium to the pulmonary vein can be confirmed with the elimination of pulmonary vein potentials, and the exit block from the pulmonary vein to the left atrium can be confirmed with pulmonary vein pacing.

Recently, a new ablation technique targeting complex frac-

Table 10. Indications for EPS in Patients With PVC

Class I
None.
Class IIa
1. High-risk patients such as those with severe symptoms of PVC, those with left ventricular dysfunction due to frequent PVCs, those who do not respond to drugs or cannot use drugs due to adverse reactions, and those with R on T PVC who are considered for catheter ablation.
2. Patients with repeated episodes of VF due to PVC originating from a single ectopic focus who are considered for catheter ablation for the treatment of PVC.
3. Patients with underlying heart disease such as myocardial infarction in whom signal-averaged ECG show a marked positive late potential.
Class IIb
1. Evaluation of drug efficacy in patients in whom sustained VT is induced.
2. Patients with no underlying heart disease who have frequent PVCs and in whom ventricular late potentials are present on signal-averaged ECG.
Class III
1. Patients who have no underlying heart disease or have underlying heart disease with relatively preserved left ventricular function (EF more than 40%), who are asymptomatic or have only mild symptoms, who have no couplets or R on T PVC, and in whom PVC is not considered to induce sustained VT or VF.

ECG, electrocardiography; EF, ejection fraction; EPS, electrophysiologic studies; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

tionated atrial electrograms (CFAE) has been developed.²¹ It has been reported that CFAE correlate with areas of slow conduction, pivot points of reentrant circuits, and evidence for localized drivers.

It has been reported that linear ablation may prolong AF cycle length presumably by cutting the random reentrant circuits involved in the maintenance of AF, and promoting them to form a larger reentrant circuit. After the linear ablation, the AF cycle length may be prolonged over time to eliminate AF

and return to sinus rhythm, or may lead to macroreentrant AT.²²

On the basis of findings suggesting that GP on the epicardium of the left atrium play an important role in the development of AF originated from the pulmonary veins and the pathogenesis of CFAE, a catheter ablation technique to target GP was developed.²³ In this technique, vagal response produced by high frequency stimuli is used to identify and ablate GP sites initiating ectopic firing.

IX Premature Ventricular Contraction

(Indications by class are listed in [Table 10](#).)

In patients with premature ventricular contraction (PVC), ventricular activation (activation of myocardial cells below the His bundle) occurs at a rate higher than the basic rate. Although not all PVCs are dangerous, some PVCs especially those with short coupling intervals may trigger VT or fatal VF.

PVC may also develop in patients who have no apparent underlying heart disease, and in such patients PVC is believed to be induced by reentry, ectopic automaticity, or triggered activity. Patients with PVC originated from the right ventricular outflow tract are generally considered to have a favorable prognosis, but may experience idiopathic VF or polymorphic VT.²⁴ These patients often undergo EPS as a procedure prior to catheter ablation.²⁵

The severity of PVCs has been long categorized by Lown's classification ([Table 11](#)).²⁶

Table 11. Classification of Severity of PVCs	
Grade	Observed
0	No PVCs
1	30/hr or less
2	More than 30/hr
3	Multiform PVCs
4a	Couplets
4b	Salvos
5	Early R on T

PVC, premature ventricular contraction.

Frequent PVCs may result in hemodynamic compromise, and cause heart failure. EPS for the purpose of catheter ablation are indicated for patients with severe symptoms, patients with heart failure or syncope due to frequent PVCs, high-risk patients such as those with R on T PVC, and patients in whom frequent VF induced by PVC ([Table 10](#)).^{1,25,27–32}

1. Methods of Electrophysiologic Studies

1. Intraventricular Mapping

Pace mapping and activation mapping are used to identify the site of origin of PVC.³³

1) Pace Mapping

In pace mapping, the number of leads out of 12 in which waveform during pacing by electrode catheter and during PVC have same configuration (the pace mapping score) is obtained. A pace mapping score of 10 or more indicates the proximity of the pacing site to the focus.^{34–37}

2) Activation Mapping

In activation mapping, an electrode catheter is used to record local ventricular electrogram to locate the site of earliest local activation. A site where the local electrogram preceded the onset of QRS by at least 10 to 15 msec and the unipolar electrogram showing a steep QS pattern indicates the proximity to the focus.

2. Estimation the Site of Origin

PVC has been reported to be originated from the right ventricular outflow tract as well as from the left ventricular outflow tract, mitral annulus, tricuspid annulus, and left ventricular papillary muscle, among other sites. Algorithms based on 12-lead ECG findings have been proposed to estimate the site of origin of PVC.^{38–42}

3. Efficacy of Drugs

Physicians may prescribe most effective drugs for the treatment of PVC when mechanisms of PVC are clarified with EPS.⁴³ The effects of antiarrhythmic drugs may be assessed during EPS by administering antiarrhythmic drugs or adenosine triphosphate (ATP) intravenously.^{44–46} When PVC does not appear during EPS, catecholamine (isoproterenol [ISP]) or other drugs are used to induce PVC.

2. Clinical Significance

Techniques of mapping PVC in detail have been established as methods to find the earliest activation site at which catheter ablation should be performed. The extrastimulus technique may induce monomorphic or polymorphic VT. The prognosis

of patients with PVC not associated with underlying heart disease is generally considered favorable,⁴⁷ while it has been reported that patients with frequent or multifocal PVCs have poor prognosis.⁴⁸

Since PVC may induce VT or VF in patients with coronary artery disease, it was hypothesized that the incidence of sudden cardiac death may be decreased by preventing PVC with antiarrhythmic drugs (PVC hypothesis that PVC suppression would prevent sudden death). However, the hypothesis was denied in CAST (Cardiac Arrhythmia Suppression Trial), a large-scale randomized placebo-controlled study of antiarrhythmic drugs mainly including class Ic drugs in patients with old myocardial infarction, which resulted in a significant increase in mortality in patients receiving antiarrhythmic drugs. Class I antiarrhythmic drugs are not expected to improve prognosis.⁴⁹

PVC is clinically significant as a factor inducing VT or VF. With the recent advances of ICD and technology to analyze intracardiac ECG immediately prior to the onset of VF, the role of PVC as a trigger of VT/VF has been recognized. Cardiac EPS for preventing recurrent VF followed by catheter ablation is being established as a treatment strategy to avoid frequent ICD discharges.

X Nonsustained Ventricular Tachycardia

Nonsustained VT is defined by three or more consecutive PVCs at a cycle length of 600 msec or less with a heart rate of 100 bpm or more which terminate spontaneously within 30 seconds.⁵⁰ EPS for patients with nonsustained VT are performed to confirm whether or not electrical stimulation induces sustained VT, VF or other types of arrhythmia. Therefore, EPS is indicated for patients who have symptoms suggestive of sustained VT, those who should be assessed for whether sustained VT may develop or not, those who should be assessed

for whether pharmacotherapy may terminate nonsustained VT or rather exacerbate the condition to lead to sustained VT, and those with symptoms such as syncope, dizziness and chest discomfort who have ECG-documented nonsustained VT but may have other arrhythmias causing the symptoms. EPS is not indicated for asymptomatic patients with nonsustained VT who have no underlying heart disease nor ECG-documented sustained VT.

XI Sustained Ventricular Tachycardia

(Indications by class are listed in Table 12.)

1. Definition and the Mechanism

VT is defined as a tachycardia at 100 bpm or more originating from the ventricle (including the His bundle and the conducting system below the His bundle). Sustained VT is defined as

VT persisting more than 30 seconds or requiring prompt termination prior to that due to hemodynamic compromise. The mechanism of sustained VT is reentry in many patients, and enhanced automaticity, abnormal automaticity, and triggered activity in other patients.

VT can be classified into monomorphic VT with uniform QRS, and polymorphic VT with beat-to-beat variation in the QRS morphology. Multiple monomorphic VT is called pleo-

Table 12. Indications for EPS in Patients With Sustained VT

Class I

1. Patients with documented sustained monomorphic VT, with or without underlying heart disease.
2. Patients with symptomatic sustained monomorphic VT who are expected to undergo catheter ablation or surgery for the treatment of VT.
3. Patients with wide QRS tachycardia.
4. Patients with syncope and/or dizziness suggestive of VT.
5. Evaluation of the efficacy and proarrhythmic effects of drugs in patients with sustained VT.

Class IIa

1. Follow-up of patients after catheter ablation.
2. Patients after myocardial infarction with left ventricular dysfunction (LVEF less than 35%) who are considered for ICD therapy to terminate sustained VT.

EPS, electrophysiologic studies; ICD, implantable cardioverter defibrillator; VT, ventricular tachycardia; LVEF, left ventricular ejection fraction.

Table 13. Indications for EPS in Patients With Brugada Syndrome**Class I**

1. Patients with a coved type (Type 1) Brugada ECG (including those with a drug-induced coved type) without documented VF or polymorphic VT who have syncope, dizziness, palpitations and other symptoms suggestive of arrhythmia.
2. Patients with a coved type (Type 1) Brugada ECG (including those with a drug-induced coved type) without documented VF or polymorphic VT who do not have syncope, dizziness, palpitations or other symptoms suggestive of arrhythmia but have a family history of sudden death in young adulthood and middle age.

Class IIa

1. Patients with a saddle back type (Types 2 and 3) Brugada ECG without documented VF or polymorphic VT who have syncope, dizziness, palpitations and other symptoms suggestive of arrhythmia.
2. Patients with a saddle back type (Types 2 and 3) Brugada ECG without documented VF or polymorphic VT who have no syncope, dizziness, palpitations or other symptoms suggestive of arrhythmia but have a family history of sudden death in young adulthood and middle age.
3. EPS for the purpose of drug efficacy evaluation in patients with Brugada ECG (coved or saddle back type) with documented VF or polymorphic VT for whom ICD implantation is difficult.

Class IIb

1. Patients with Brugada ECG (coved or saddle back type) who do not have neither documented VF or polymorphic VT, symptoms suggestive of arrhythmia, or a family history of sudden death in young adulthood and middle age.

ECG, electrocardiography; EPS, electrophysiologic studies; ICD, implantable cardioverter defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

morphic VT. Polymorphic VT often leads to VF.

Patients with sustained monomorphic VT often have underlying heart disease, while some patients have idiopathic VT not complicated with underlying heart disease such as verapamil-sensitive left VT and right ventricular outflow tract VT.

EPS for patients with sustained VT are performed to diagnose VT, identify the mechanism, map the circuit, and assess the efficacy of treatment.

A general consensus has been reached on stimulation protocols used to induce VT during EPS. Extrastimulus testing and rapid pacing are commonly used. In extrastimulus testing, three extrastimuli or less are given up to the refractory period after an eight-beat basic drive at two or more different sinus cycle lengths (600 and 400 msec).

Rapid pacing is a method to induce VT by delivering stimuli at a rate higher than the sinus rate. The stimuli are given up to the ventricular refractory period or by a pacing cycle length of 210 msec for 5 to 10 seconds to two sites in the right ventricle and one in the left ventricle. ISP is administered to patients in whom VT is not induced by rapid pacing. Reentrant VT is frequently induced by programmed stimulation. VT due to abnormal automaticity is induced more frequently by rapid pacing, but the reproducibility is low.

The presence of a reentry circuit is supported when VT can be induced and terminated by programmed stimulation, or when there is an inverse correlation between the coupling interval of extrastimuli at the time of VF and the interval from the last paced beat to the first subsequent VT beat. The most reliable finding is the presence of the phenomenon of transient entrainment.^{51,52}

Transient entrainment of a tachycardia is demonstrated by the presence of constant fusion beats or progressive fusion beats when sustained monomorphic VT is induced and pacing at a rate that is slower than the rate of tachycardia is performed; the return cycle achieved after the discontinuation of pacing is consistent with pacing rate; or the presence of antidromic capture of the ventricular potential due to localized

conduction block and pacing associated with interruption of the tachycardia.

Mapping is performed using electrode catheters with an interelectrode distance of 5 to 10 mm and a filter setting of 30 to 500 Hz. Normal electrographic characteristics include an amplitude of 3 mV or more, a duration of 70 msec or less, while abnormal electrographic characteristics include a duration of 60 to 70 msec or more, an amplitude of 0.5 mV or less, the presence of frequency-rich, multiphasic waveforms (fractionated or fragmented electrograms).^{53,54} Split potentials are potentials separated by an isoelectric interval of 30 msec or more.

The QRS potential during tachycardia are called diastolic potential, which is divided into the early, middle and late diastolic potentials.

These abnormal potentials are often caused by a reentrant circuit. The presence of a slow conduction is confirmed based on the presence of the phenomenon of transient entrainment. When pacing is performed within the area of slow conduction during VT, the rate is increased to the pacing rate with no change in QRS morphology, and the diastolic potential to QRS interval becomes equal to the stimulus to QRS interval. When VT recurs after discontinuation of pacing, the return cycle is consistent with the rate of tachycardia.

In verapamil-sensitive idiopathic left VT, bundle branch potentials form a part of the reentrant circuit.

Pace mapping is another method to identify the site of origin of tachycardia by using 12-lead ECG to specify sites in which waveform during pacing and during VT have same configuration.⁵⁵ Also, the site of earliest activation during VT is used to identify the site of origin. The site of earliest activation is specified by demonstrating delays in activation of the surrounding muscles. Identification of the site of origin is an essential step to specify the target of catheter ablation.⁵⁶

EPS is performed to evaluate the efficacy of antiarrhythmic drugs and catheter ablation, the significance of EPS in evaluation of drug efficacy is diminishing.

XII Brugada Syndrome

(Indications by class are listed in [Table 13](#).)

EPS for patients with Brugada syndrome are performed mainly to assess the risk of induction of polymorphic VT and/or VF

(i.e., assess whether ICD therapy is indicated or not). The present guidelines therefore do not describe indications for EPS in patients with Brugada syndrome to clarify the patho-

Table 14. Indications for EPS in Patients With Idiopathic VF

Class I
1. EPS in patients in whom right ventricular outflow tract VT has lead to VF for the purpose of ablation.
Class IIa
1. EPS in patients without documented VF who have syncope and/or near syncope and right ventricular outflow tract PVC/VT for the purpose of ablation.
Class IIb
1. Induction of VF after successful ablation of PVC/VT.

EPS, electrophysiologic studies; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

Table 15. Indications for EPS in Patients With Early Repolarization Syndrome

Class IIa
1. Patients with early repolarization and J wave without documented VF or polymorphic VT who have syncope, dizziness, palpitation and other symptoms suggestive of arrhythmia.
2. Patients with early repolarization and J wave who have neither documented VF or polymorphic VT nor symptoms suggestive of arrhythmia such as syncope, dizziness and palpitation, and who have a family history of sudden death in young adulthood and middle age.
Class IIb
1. Patients with early repolarization without J wave who do not have documented VF or polymorphic VT, and who have syncope, dizziness, palpitation and/or other symptoms suggestive of arrhythmia.
2. Clinical EPS to identify and ablate the site of origin of PVC that trigger VF.
3. EPS in patients with early repolarization and documented VF or polymorphic VT to assess the efficacy of pharmacotherapy.

EPS, electrophysiologic studies; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

physiology of this disease. Since pharmacotherapy of VF is limited in efficacy, the significance of EPS as a method to evaluate the efficacy of pharmacotherapy is limited.

Since EPS is a **Class I** indication for patients with Brugada syndrome with a documented history of VT/VF or cardiopulmonary arrest, EPS is not used to assess the risk of VT/VF in this patient population. The present guidelines list the indications for EPS in asymptomatic patients without a history of VT/VF or cardiopulmonary arrest and in patients with no ECG-documented arrhythmias who have syncope and other symptoms suggestive of a high risk of fatal arrhythmia.

Since Brugada syndrome is characterized by temporal variability of ECG waveforms and the autonomic nervous system is believed to be a precipitating factor, it is apparent that EPS, which usually last 30 to 60 minutes, are insufficient to assess the risk of arrhythmias in this patient population. However,

EPS provides important information to physicians and patients to determine treatment strategies in patients with Brugada syndrome in whom available data necessary for appropriate risk assessment are inevitably limited.

1. Indications for Electrophysiologic Studies in Patients With Brugada Syndrome

In the present guidelines, patients with Brugada syndrome are classified by ECG waveforms to a coved type (Type 1), and a saddle back type (Types 2 and 3), and then by the presence/absence of a history of VF or polymorphic VT, symptoms suggestive of arrhythmias (e.g., syncope), a family history of sudden death (death in young adulthood and middle age).

XIII Idiopathic Ventricular Fibrillation

Idiopathic VF is defined as most serious VF occurring in individuals with no apparent underlying heart disease or abnormal ECG.⁵⁷ VF that occurs patients with abnormal ECG such as those with Brugada syndrome and congenital long QT syndrome is not categorized in this type of VF. Recent studies have demonstrated that patients with idiopathic VF may be classified into several pathophysiological groups. The present guidelines describe indications for EPS in each group of patients.

1. Idiopathic Ventricular Fibrillation Triggered by Right Ventricular Outflow Tract Premature Ventricular Contraction/Ventricular Tachycardia

(Indications by class are listed in [Table 14](#).)

Right ventricular outflow tract PVC/VT may lead to VF in patients with no underlying heart disease.⁵⁸ The risk of induc-

tion of VF is high among patients with high-rate VT (240 to 250bpm), those with a history of syncope or near syncope, and those with polymorphic QRS configuration during VT.⁵⁸ Since this type of idiopathic VF may be completely cured by ablating the site of origin of VT, the significance of EPS as a method to specify the origin is high.^{34,37,58–60}

2. Early Repolarization Syndrome

(Indications by class are listed in [Table 15](#).)

Early repolarization is defined as J-point elevation of 0.1 mV or more above baseline in at least two consecutive leads in the absence of other causes of ST elevation such as ischemia and pericarditis.^{61,62} Since early repolarization is relatively common (3 to 13%) even in healthy individuals especially young males,^{61–65} its specificity is very low as a factor to predict the

Table 16. Indications for EPS in Patients With Short-Coupled Variant of Torsade de Pointes

Class I
1. EPS to identify and ablate the site of origin of PVC triggering VT/VF in patients in whom short-coupled variant of torsade de pointes has been demonstrated.
Class IIa
1. Patients with syncope of unknown etiology who have R on T PVC but have not been demonstrated to have torsade de pointes.
Class IIb
1. Asymptomatic patients with R on T PVC who have not been demonstrated to have torsade de pointes.

EPS, electrophysiologic studies; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

Table 17. Indications for EPS in Patients With Short QT Syndrome

Class IIa
1. Patients who meet the criteria for diagnosis of short QT syndrome, in whom neither VF nor polymorphic VT has been documented, and who have symptoms suggestive of arrhythmias such as syncope, dizziness and palpitations.
2. Patients who meet the criteria for diagnosis of short QT syndrome, in whom neither VF nor polymorphic VT has been documented, who have no symptoms suggestive of arrhythmias such as syncope, dizziness and palpitations, and who have a family history of sudden death in young adulthood and middle age.
3. EPS as a method of evaluation of drugs in patients who meet the criteria for diagnosis of short QT syndrome, in whom VF or polymorphic VT has been documented, and in whom ICD implantation is difficult.

EPS, electrophysiologic studies; ICD, implantable cardioverter defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

risk of sudden death. Many reports have described that the significance of EPS as a method of risk assessment is low, and the importance of EPS for this purpose is not clear.

ECG findings suggestive high risk of sudden death include 1) the presence of early repolarization and J wave (notch),^{61,64} especially 2 mV or more, in inferior leads, and 2) the presence of ST elevation in inferior and lateral leads. EPS as a risk assessment method is meaningful for patients with one of these two conditions who have syncope, dizziness, palpitation and/or other symptoms suggestive of severe arrhythmia or have a family history of sudden death in young adulthood and middle age. However, the significance of EPS is limited as a method to identify and ablate the site of origin of PVC triggering VF. The importance of EPS is accepted as a method to assess the efficacy of antiarrhythmic drug such as quinidine in patients with documented VF or polymorphic VT in whom ICD implantation is difficult.

3. Short-Coupled Variant of Torsade de Pointes

(Indications by class are listed in [Table 16](#).)

Short-coupled variant of torsade de pointes is a polymorphic VT induced by R on T PVC with a very short coupling interval,⁶⁶ and is diagnosed by 1) the presence of polymorphic VT or VF induced by R on T PVC with a short coupling interval; 2) the absence of abnormally long or short QT interval and Brugada syndrome; and 3) the absence of structural heart disease. A potential role of Purkinje fiber network in the initiation of short-coupled variant of torsade de pointes has been suggested, since during EPS Purkinje potentials in the left or right ventricle are recorded before a specific type of PVC induces VF.⁶⁷ Since if this type of PVC can be eliminated with catheter ablation, the resultant VT/VF can be cured completely, the importance of EPS to identify the site of origin of PVC is high.

4. Short QT Syndrome

(Indications by class are listed in [Table 17](#).)

Short QT syndrome⁶⁸ is diagnosed on the basis of the three criteria of 1) a QTc of 330 msec or less; 2) the absence of structural heart disease; and 3) a family history of sudden cardiac death.⁶⁹ Since the prevalence of short QT syndrome is significantly lower than those of congenital long QT syndrome and Brugada syndrome, sufficient evidence has not been accumulated, and evidence-based decision-making is difficult for this syndrome. EPS in patients who meet the above criteria for diagnosis of short QT syndrome and have neither VF nor polymorphic VT are considered significant 1) when symptoms suggesting severe arrhythmias such as syncope, dizziness and palpitations are present; or 2) when a family history of sudden death in young adulthood and middle age is present. On the other hand, the significance of EPS as a method of drug efficacy evaluation is accepted in patients in whom VF or polymorphic VT has been documented but ICD implantation is difficult.

5. Idiopathic Ventricular Fibrillation With No History of ECG Abnormalities

In patients with idiopathic VF who have not had abnormal ECG findings prior to the onset of VF, it is impossible to predict the risk of VF in advance. Since VF or cardiopulmonary arrest is the first manifestation in these patients, and treatment is given to survivors, ICD therapy is a **Class I** indication for patients in whom the cause of VF is unknown. Accordingly, the clinical significance of EPS for the purpose of risk assessment of this patient population is minimal. Since patients who had experienced VF which might have been terminated spontaneously within a short period of time are categorized clinically into those with syncope of unknown etiology, physicians should refer to the section "XVI. Syncope of Unknown Etiology".

Indications by Class

None.

Table 18. Indications for EPS in Patients With Long QT Syndrome

Class I
None.
Class IIa
1. Patients who have a history of syncope of unknown etiology and a long QT interval.
Class IIb
1. Survivors of cardiac arrest, and patients with documented VF or torsades de pointes.
2. Patients with a family history of sudden death or syncope due to torsades de pointes who have a long QT interval on ECG.
Class III
1. Patients in whom the cause of long QT intervals has been identified and QT intervals are normalized after eliminating or correcting the cause, and who have no family history of sudden death or syncope due to torsades de pointes.

ECG, electrocardiography; EPS, electrophysiologic studies; VF, ventricular fibrillation.

Table 19. Indications for EPS in Patients With VF Associated With Structural Heart Disease

Class I
None.
Class IIa
1. Patients with repeated VF induced by PVC originating from a single ectopic focus who are considered for catheter ablation for the treatment of PVC.
2. Patients who do not respond well to pharmacotherapy and have frequent ICD discharges, and who are considered for catheter ablation for the treatment of electrical storms.
3. Patients with VF who rely on ventricular assist devices (e.g., PCPS, LVAD) for whom catheter ablation is considered for the treatment of VF.

EPS, electrophysiologic studies; ICD, implantable cardioverter defibrillator; LVAD, left ventricular assist device; PCPS, percutaneous cardiopulmonary support; PVC, premature ventricular contraction; VF, ventricular fibrillation.

XIV Long QT Syndrome

(Indications by class are listed in [Table 18](#).)

The clinical significance of EPS in patients with long QT syndrome is lower than other arrhythmias.⁷⁰ Patients with congenital long QT syndrome typically have a slow resting heart rate and a longer SNRT as compared with healthy individuals,^{71,72} but generally have normal AV nodal conduction. Since the inducibility of ventricular arrhythmias by programmed stimulation is low in patients with long QT syndrome,⁷³ EPS as a method to estimate the prognosis and evaluate drug efficacy in this patient population is limited. In studies of the repolar-

ization process using recording of monophasic action potentials, patients with congenital and acquired long QT syndrome had a longer duration of action potential and early afterdepolarization-like hump.^{74,75} Although the significance of EPS is limited in patients who have explained syncope and documented long QT interval, EPS is useful for patients with a long QT interval and syncope in whom the etiology of syncope remained unknown after history taking and various testing such as neurological examination, and head-up tilt test as a method to rule out ventricular arrhythmia as an etiology of syncope.

XV Ventricular Fibrillation Associated With Structural Heart Diseases

(Indications by class are listed in [Table 19](#).)

1. Introduction

The most common treatment strategies for VF are ICD therapy and pharmacotherapy. Catheter ablation may also be performed in patients who do not respond well to pharmacotherapy and have frequent ICD discharges, and patients with a history of VF induced by electrical storm or certain types of PVCs.^{23,24}

2. Indications for Electrophysiologic Studies

EPS is not usually indicated for patients with VF, and performed only in specific cases.⁸

1. Electrophysiologic Studies for Risk Assessment

In the treatment of patients with a history of VF (secondary

prevention), the most common practice is to implant an ICD without conducting EPS.^{76–78} It has been demonstrated that in the primary prevention among patients with cardiac dysfunction, ICD therapy may yield a better prognosis than pharmacotherapy in patients with and without the inducibility of VT/VF.^{79,80}

2. Electrophysiologic Studies for Treatment

It has been reported that, although rare, certain types of PVCs may trigger VF.⁸¹ In patients with such conditions, ablation therapy to eliminate certain types of PVCs causing VF may prevent the recurrence of VF, and thereby decrease the frequency of ICD discharges and prevent electrical storms. The significance of EPS as an examination performed prior to ablation therapy is high especially in patients with electrical storms who may need assisted circulation such as percutaneous cardiopulmonary support (PCPS) and left ventricular assist device (LVAD) to maintain their life and need ablation therapy to be weaned off the assisted circulation.

Table 20. Indications of EPS in Patients With Syncope

Class I
1. Patients with syncope which is suspected due to arrhythmias according to the type of symptoms, and who have no documented arrhythmias.
2. Patients with structural heart disease who have syncope of unknown etiology after noninvasive examination.
Class II
1. EPS for the purpose of clarifying the mechanism of arrhythmia that has been documented as the etiology of syncope.
2. Patients with repeated syncope of unknown etiology who have no structural heart disease and have negative head-up tilt tests.
3. Patients with a history of syncope who have nor structural heart disease either symptoms of palpitations, and have normal ECG findings.
Class III
1. Patients with explained syncope in whom EPS do not contribute to the determination of treatment strategies.

ECG, electrocardiography; EPS, electrophysiologic studies.

3. Methods of Electrophysiologic Studies

No consensus has been achieved concerning the protocol to induce VF.

4. Clinical Significance

The role of EPS is limited in the risk assessment of VF. However, EPS is essential and meaningful in patients with VF who can be treated with catheter ablation, as a method to locate the site of origin of PVC triggering VF, although patients with such condition are very rare.^{24,81}

5. Criteria for Implementation of Electrophysiologic Studies

It is rare to perform EPS as a method to induce VF. There is no consensus regarding the criteria for implementation of EPS.

6. Indications for Treatment Using Electrophysiologic Studies

ICD therapy is recommended for the secondary prevention of VF. No criteria applicable to patients in Japan have been established for the use of EPS in the primary prevention in patients with cardiac dysfunction.

XVI Syncope of Unknown Etiology

(Indications by class are listed in Table 20.)

In this guideline, syncope is defined as a transient loss of consciousness due to decreased cerebral blood flow with an inability to maintain postural tone that is followed by spontaneous and complete recovery of consciousness without sequelae. Since the severity and life prognosis depend on the etiology of syncope, accurate differential diagnosis is essential. Especially cardiac syncope may be fatal, and appropriate diagnosis and treatment are important.

1. Indications for Electrophysiologic Studies

Patients in whom arrhythmias are suspected as the etiology of syncope are indicated for EPS. Arrhythmias are more likely the etiology of syncope in patients with structural heart disease as compared with patients without it.⁸² EPS is not indicated for patients with neither findings suggestive of arrhythmias nor structural heart diseases.^{10,82,83}

2. Methods of Electrophysiologic Studies

The following procedures are recommended for the diagnosis of syncope with EPS.⁸⁴

1. Determination of SNRT and corrected SNRT (CSNRT) using atrial pacing.
2. Assessment of the baseline HV interval and the properties of the His-Purkinje system during atrial pacing.

3. Induction of VF with the extrastimulus technique using two basic cycle lengths and two right ventricular sites.
4. Induction of supraventricular tachycardia by atrial pacing.

3. Criteria for Electrophysiologic Diagnosis of Syncope

In patients with the following conditions, syncope is strongly suspected to be caused by arrhythmias.

1. Sinus bradycardia and markedly prolonged CSNRT (more than 525 msec).
2. The presence of bundle branch block and either a baseline HV interval of more than 100 msec or HV block induced during atrial pacing or with pharmacological stress.
3. The induction of complete AV block with pharmacological stress by antiarrhythmic drugs.
4. The induction of sustained monomorphic VT.
5. The induction of rapid supraventricular tachycardia which reproduces hypotensive symptoms.

In patients with the following conditions, it is controversial whether the etiology of syncope is arrhythmias or not.

1. A HV interval of 70 to 100 msec.
2. The presence of structural heart disease and the induction of polymorphic VT or VF.
3. The presence of Brugada type ECG and the induction of polymorphic VT or VF.

Table 21. Indications for EPS in Patients After Cardiopulmonary Resuscitation

Class I
1. Patients after cardiopulmonary resuscitation in whom cardiac arrest was believed due to ventricular arrhythmias.
2. Patients after cardiopulmonary resuscitation who are suspected to have sick sinus syndrome or AV block.
3. Patients with ECG evidence of WPW syndrome after cardiopulmonary resuscitation who have a history of syncope or have episodes of palpitations.
Class IIa
None.
Class IIb
1. Assessment of pathophysiology of long QT syndrome in survivors after cardiopulmonary resuscitation.
2. Induction of VF in survivors after cardiopulmonary resuscitation who have Brugada syndrome.

AV, atrioventricular; ECG, electrocardiography; EPS, electrophysiologic studies; VF, ventricular fibrillation; WPW, Wolff-Parkinson-White.

Table 22. Indications for EPS for the Purpose of Evaluating Antiarrhythmic Drug Efficacy in Patients With Bradyarrhythmias

Class I
None.
Class IIa
1. Patients with asymptomatic AV block or intraventricular conduction disturbance who need treatment with drugs that may exacerbate conduction disturbance.
2. Patients with asymptomatic sinus node dysfunction who need treatment with drugs that may exacerbate sinus node dysfunction.
Class IIb
1. Patients with sinus node dysfunction or AV block in whom pharmacological stress with antiarrhythmic drugs may reveal abnormalities.

AV, atrioventricular; EPS, electrophysiologic studies.

XVII Patients After Cardiopulmonary Resuscitation

(Indications by class are listed in [Table 21](#).)

In the present guidelines, “patients after cardiopulmonary resuscitation” are defined as patients who have recovered from cardiac arrest through cardiopulmonary resuscitation in whom hemodynamics have been normalized. VT or VF account for more than 80% of all causes of cardiac arrest, while bradycardia may lead to cardiac arrest in some cases. In patients who have been resuscitated from cardiac arrest, sustained monomorphic VT, which is indicative of the presence of abnormal myocardium, is often induced during EPS. EPS in patients after cardiopulmonary resuscitation are performed to induce ventricular arrhythmias (VT or VF), detect abnormal myocar-

dium using mapping techniques, and clarify the mechanism of arrhythmias. EPS should be performed in accordance with the methods of EPS in patients with sustained VT. Patients with WPW syndrome who had VF (or who are at risk for VF) have a short refractory period of the accessory pathway. In patients with a history of cardiac arrest due to bradycardia, the presence/absence and severity of sick sinus syndrome and AV block should be evaluated. Sick sinus syndrome may be effectively assessed with the overdrive suppression test, while techniques to identify the site of block and those to induce block through pharmacological stress tests or pacing are useful in the assessment of AV block.

XVIII Evaluation of Antiarrhythmic Drug Efficacy

(Indications by class are listed in [Table 22](#) and [Table 23](#).)

In patients with occult bradyarrhythmias in whom treatment with antiarrhythmic drugs may exacerbate the condition, EPS are performed to examine whether bradyarrhythmia is inducible with pharmacological stress using class I and IV drugs.^{43,85} Patients with tachyarrhythmias may undergo EPS to evaluate the drug efficacy. Since supraventricular arrhythmia may be successfully treated with catheter ablation, EPS for the purpose of evaluating drug efficacy in patients with tachyarrhythmia are rarely performed. However, patients with intractable ventricular arrhythmias may undergo EPS to evaluate the drug efficacy. The use of EPS in patients with ventricular arrhythmias for the purpose of evaluating drug efficacy is controversial since some reports have concluded that EPS-guided anti-

arrhythmic drug therapy may improve the prognosis of patients with ventricular arrhythmia, while others reported that EPS-guided therapy may not improve the prognosis despite arrhythmia is suppressed during EPS, and have described that EPS did not differ from Holter ECG monitoring in terms of the drug efficacy selected according to the test results.⁸⁶ In a study to compare the efficacy of EPS-guided drug regimens and amiodarone in the prevention of fatal ventricular arrhythmia, patients receiving amiodarone showed better prognosis as compared with patient receiving EPS-guided regimens. This finding suggests that amiodarone is the first-line agent, but EPS-guided regimens may be considered in patients who are contraindicated for amiodarone.⁸⁷

Table 23. Indications for EPS for the Purpose of Evaluating Antiarrhythmic Drug Efficacy in Patients With Tachyarrhythmias

Class I
None.
Class IIa
1. Evaluation of the efficacy and proarrhythmic effects of drugs in patients with sustained monomorphic ventricular tachycardia.
Class IIb
1. Drug efficacy evaluation in patients with WPW syndrome, AV nodal reentrant tachycardia, sinus nodal reentrant tachycardia, atrial tachycardia, and atrial flutter.

AV, atrioventricular; EPS, electrophysiologic studies; WPW, Wolff-Parkinson-White.

Table 24. Indications for EPS in Candidates of CRT

Class I
None.
Class IIa
None.
Class IIb
1. Patients in whom CRT is expected to be effective on the basis of the finding of noninvasive examinations.

CRT, cardiac resynchronization therapy; EPS, electrophysiologic studies.

XIX Surgical Treatment of Arrhythmias

In the cardiology setting, physicians perform EPS to examine whether surgery is indicated, while in the surgical setting, EPS are performed during surgery. This section describes EPS performed during surgery. During surgery, EPS is performed to locate the arrhythmic foci using mapping techniques, and confirm the successful ablation and inactivation of the foci. As to surgical treatment of WPW syndrome, AVNRT, PVC, VT, premature atrial contractions, AT, and atrial flutter, epicardial

mapping is performed to locate the arrhythmic foci, which is destroyed by cryoablation, thermal ablation, surgical ablation or other appropriate techniques. Surgical treatments of VF and AF are performed without EPS since there are no established methods of implementing electrophysiologic data of epicardial mapping. Successful ablation and inactivation of the arrhythmic foci is confirmed by the absence of arrhythmias induced by programmed stimulation.

XX Arrhythmias in Children

EPS in children have special requirements. The number and size of catheters are limited because of their small body size, and physicians must be aware of the type and nature of arrhythmias specific to children, such as congenital complete AV block, junctional ectopic tachycardia, catecholamine-induced polymorphic VT, and arrhythmias that occur commonly after surgical correction of congenital heart diseases. Indications for EPS in children with common pediatric arrhythmias are as follows:

- EPS is indicated in children with junctional ectopic tachy-

cardia when differentiation between it with other types of supraventricular tachycardias is difficult (**Class IIb**).

- EPS, mainly as a procedure to be followed by radiofrequency catheter ablation,⁸⁸⁻¹¹⁰ are indicated for children with AT that developed after the Mustard, Senning or Fontan operation, and children with VT that developed after surgical correction for tetralogy of Fallot (**Class IIa**).
- EPS is absolutely indicated for only a small number of patients with catecholamine-induced polymorphic VT (**Class IIb**).

XXI Cardiac Resynchronization Therapy

(Indications by class are listed in [Table 24](#).)

1. Introduction

In patients with cardiac dysfunction, intraventricular conduction disturbance may cause abnormal left ventricular contraction (dyssynchrony), which further impairs cardiac function.¹¹¹ CRT is a therapy that delivers electrical pulses to the area of delayed contraction in the left ventricle (which is often observed

in the left ventricular free wall) to synchronize the movement of the left ventricle to that of the ventricular septum to correct dyssynchrony. Recently, this procedure is drawing special attention as a new non-pharmacotherapy for heart failure.¹¹²⁻¹¹⁴

2. Indications for Electrophysiologic Studies

EPS for patients who are candidates for CRT are performed to predict the efficacy of CRT in advance. However, researchers

Table 25. Indications for EPS in Candidates for Pacemaker Therapy**Class I**

1. EPS is required to select and set a pacemaker device.
2. Patients with bradyarrhythmia who also have tachyarrhythmia for which EPS is useful in the diagnosis and treatment.
3. Patients who need temporal pacing to evaluate the efficacy of pacemaker therapy for the treatment of bradycardia, neurally mediated syncope or hypertrophic obstructive cardiomyopathy.

Class IIa

1. Patients with dizziness, blackouts, syncope and/or other symptoms which are strongly suspected but not confirmed due to bradycardia.
2. Patients with progressive disease who are highly likely to require pacemaker therapy in the future.
3. Patients with heart failure who undergo temporal pacing to evaluate the efficacy of CRT.

CRT, cardiac resynchronization therapy; EPS, electrophysiologic studies.

are trying to use noninvasive examinations such as echocardiography and MRI to predict the efficacy of CRT in advance,^{115–118} and several prospective clinical studies have demonstrated characteristics of patients in whom CRT is effective.¹¹⁹

Since there is insufficient evidence to support the hypothesis that acute improvement in hemodynamics during EPS may predict the long-term efficacy of CRT, the significance of EPS in candidates for CRT is low.

3. Methods of Electrophysiologic Studies

Pacing catheter electrodes are placed in the right ventricle and a coronary vein. When a catheter electrode for left ventricular pacing is placed in a coronary vein, coronary vein imaging should be performed to understand the anatomy of coronary veins and the positional relationship between coronary veins and pacing sites.

During EPS, blood pressure, pulmonary arterial pressure, cardiac output, arterial blood pressure, and left ventricular dP/dt should be obtained during spontaneous rhythm, right and left ventricular pacing and biventricular pacing. Also, echocardiography is performed to assess the presence and severity of mitral regurgitation, left ventricular dyssynchrony, and other abnormal findings to discuss what is the best pacing mode to ensure stable hemodynamics.

4. Clinical Significance

The clinical significance of EPS in patients receiving CRT is

gradually decreasing.

5. Criteria for Indication of Cardiac Resynchronization Therapy Based on Electrophysiologic Studies Findings

There are no established criteria for indication of CRT based on EPS findings, i.e., a given improvement of hemodynamics by temporal biventricular cardiac pacing during EPS is indicative of the efficacy of CRT using permanent biventricular cardiac pacing. It is also unclear whether acute improvement observed during cardiac catheterization may continue after implantation of CRT device.

6. Indications for Cardiac Resynchronization Therapy

As described above, CRT with biventricular pacing is currently indicated for patients who have moderate to severe heart failure symptoms (New York Heart Association [NYHA] Class III or IV), a QRS interval of 130 msec or more, and a left ventricular ejection fraction (LVEF) of 35% or less, and who have not responded to pharmacotherapy, but the indications of this technique may be extended in the future.

XXII Cardiac Pacemaker Implantation

(Indications by class are listed in [Table 25](#).)

Although EPS is not always necessary to determine whether pacemaker therapy is indicated in patients with AV block,^{120,121} AV block within or below the His bundle is poor in prognosis and thus requires pacemaker therapy.^{122–126} EPS is useful in patients with progressive diseases who are highly likely to require pacemaker therapy in the future. Although the role of EPS is not significant in patients with asymptomatic sick sinus syndrome who are not indicated for pacemaker therapy in principle,^{120,121} assessment of AV conductivity and retrograde AV conduction helps physicians to select appropriate devices for

their patients and control the implanted devices. EPS is also useful in patients who are strongly suspected but not confirmed to have bradyarrhythmia. Assessment using temporal pacing is useful in patients with bradycardia, neurally mediated syncope,^{127–133} hypertrophic obstructive cardiomyopathy^{134–136} and heart failure as a method to determine whether pacemaker therapy is indicated for them.^{137–141} EPS is useful in some patients with tachyarrhythmia and concomitant bradyarrhythmia. When patients show findings indicative of the necessity of pacemaker therapy and have been diagnosed definitely, EPS only for the purpose of diagnosis should not be performed.

XXIII Catheter Ablation

EPS in candidates for catheter ablation and patients after catheter ablation are performed mainly to induce tachycardia, locate the source of the tachycardia, and evaluate the efficacy of catheter ablation. ISP and intravenous atropine sulfate are used to induce tachycardia in patients with abnormal automaticity. Tachycardia is induced by rapid pacing at a relatively long interval in patients with triggered activity and by extrastimuli and rapid pacing at a short interval in patients with reentry. The source of the tachycardia is located as the site of earliest activation in patients with tachycardia due to abnormal automaticity or local reentry, and as the zone of slow isthmus conduction within the reentry circuit in patients with macroreentrant tachycardia. When tachycardias are no longer induced by pharmacological stress or programmed stimulation after catheter ablation in which the source of the tachycardia is ablated or isolated or the tachycardia circuit is interrupted, the treatment may be confirmed successful.

In patients with WPW syndrome, it is important to specify the location of the accessory pathway, and differentiate from conditions due to more than one accessory pathway or atriofascicular Mahaim fibers prior to catheter ablation. After catheter ablation, EPS is useful to assess whether re-conduction is inducible with pharmacological stress using ISP and rapid I.V. of ATP.

In patients with AVNRT, EPS is performed to differentiate slow-fast AVNRT from fast-slow AVNRT with a lower com-

mon pathway; fast-slow AVNRT from permanent junctional reciprocating tachycardia (PJRT); and AVNRT from ATP-sensitive AT originating from the Koch triangle.¹⁴² Catheter ablation targets the antegrade slow pathway (SP) in patients with common slow-fast AVNRT, and the retrograde SP in patients with uncommon fast-slow AVNRT. The endpoint of ablation may not be set to the complete disappearance of SP conduction. Ablation should be performed to ensure that AVNRT is no longer induced under pharmacological stress with ISP. Induction of single AV nodal echo is acceptable.

In patients with AT, EPS is performed to differentiate between ectopic AT, local reentrant AT, and macroreentrant AT based on responsiveness to induction methods and ATP, and locate the optimal site of ablation using the most effective mapping method.

EPS in patients with paroxysmal AF are performed to trigger AF and locate the source. In patients with chronic AF, it is necessary to identify the substrate for AF by targeting CFAE.

In patients with VT, appropriate methods of induction and mapping should be selected for right ventricular outflow tract VT, verapamil-sensitive VT, and macroreentrant VT due to structural heart disease. The optimal target of ablation should be specified using appropriate mapping methods such as the activation mapping, pace mapping, entrainment mapping,¹⁴³ and substrate mapping.^{96,144}

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Appendix

Chair:

- Satoshi Ogawa, International University of Health and Welfare, Mita Hospital

Members:

- Yoshifusa Aizawa, Department of Research and Development, Tachikawa Medical Center
- Kazutaka Aonuma, Cardiovascular Division, Institute of Clinical Medicine, Graduate School of Comprehensive Human Sciences, University of Tsukuba
- Makoto Hirai, Department of Nursing, Nagoya University School of Health Sciences
- Yoshito Iesaka, Tsuchiura Kyodo General Hospital
- Hiroshi Inoue, Second Department of Internal Medicine, Graduate School of Medicine, University of Toyama
- Toshiyuki Ishikawa, Department of Medical Science and Cardiorenal Medicine, Yokohama City University Graduate School of Medicine
- Shiro Kamakura, Division of Arrhythmia and Electrophysiology, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center
- Takao Kato, Nippon Medical School
- Youichi Kobayashi, Division of Cardiology, Department of Medicine, Showa University School of Medicine

- Yoshio Kosakai, Senri Central Hospital
- Koichiro Kumagai, Heart Rhythm Center, Fukuoka Sanno Hospital
- Takashi Kurita, Division of Cardiology, Department of Medicine, Faculty of Medicine, Kinki University
- Yuji Nakazato, Department of Cardiology, Juntendo University Urayasu Hospital
- Ken Okumura, Department of Cardiology, Respiratory Medicine and Nephrology, Hirosaki University Graduate School of Medicine
- Morio Shoda, Department of Cardiology, Tokyo Women's Medical University
- Kaoru Sugi, Department of Cardiology, Toho University School of Medicine Ohashi Hospital
- Naokata Sumitomo, Department of Pediatrics and Child Health, Nihon University School of Medicine
- Seiji Takatsuki, Department of Cardiology, Keio University School of Medicine
- Kan Takayanagi, Department of Cardiology, Dokkyo Medical University Koshigaya Hospital
- Ichiro Watanabe, Division of Cardiology, Department of Medicine, Nihon University School of Medicine

Collaborators:

- Masaomi Chinushi, Department of Medical Technology, School of Health Sciences Faculty of Medicine, Niigata University
- Akira Fujiki, Division of Cardiology, Shizuoka Red Cross Hospital
- Atsushi Iwasa, Division of Cardiovascular Disease, New Tokyo Hospital
- Yoshinori Kobayashi, Division of Cardiology, Department of Internal Medicine, Tokai University Hachioji Hospital
- Keisuke Kuga, Cardiovascular Division, Graduate School of Comprehensive Human Sciences, University of Tsukuba
- Satoshi Nagase, Department of Cardiovascular Medicine, Okayama University Graduate School of Medicine
- Satoshi Ohnishi, Department of Cardiovascular Medicine, Kanto Medical Center Nippon Telegraph and Telephone East Corporation
- Kazuhiro Satomi, Division of Arrhythmia and Electrophysiology, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center
- Kaoru Tanno, Division of Cardiology, Department of Medicine, Showa University School of Medicine
- Masayuki Yasuda, Yasuda Iin

Independent Assessment Committee:

- Hiroyuki Daida, Department of Cardiovascular Medicine, Juntendo University Graduate School of Medicine
- Kazumasa Hiejima, Kudanzaka Hospital
- Hiroshi Kasanuki, Integrated Bioscience and Biomedical Engineering, Graduate School of Advanced Science and Engineering, Faculty of Science and Engineering, Waseda University
- Takuro Misaki, Saiseikai Toyama Hospital
- Tohru Ohe, The Sakakibara Heart Institute of Okayama

(The affiliations of the members are as of July 2012)