GLOBAL VOICES

# Contributions of France to the field of clinical cardiac electrophysiology and pacing

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## Introduction

The contributions of France to the field of clinical cardiac electrophysiology and pacing during the last 7 decades have been considerable. This review will summarize the most important original articles considering only those that were mainly made in France. For simplicity reasons, these contributions will be classified as topics covering chronologic order. Several princeps publications dealing with multiple fields of cardiac electrophysiology and pacing have been reported in French-language journals only, and that explains why they are rarely referenced. One of the goals of this review will be to make them better known. Other publications dealing with miscellaneous topics, antiarrhythmic drugs (AADs), surgical treatment of cardiac arrhythmias, experimental studies, and outstanding case reports are included in the Supplemental Material. For publications dealing with multicenter studies, only those with first and last French authorship have been summarized. The others are cited in the Supplemental Material.

## Historical development of the field of cardiac electrophysiology and pacing in France

The first endocardial electrocardiogram (ECG) recordings were performed by Jean Lenègre[1] in Paris in 1943 but were only published in 1945 for obvious reasons. He was followed by cardiologists from Montpellier (Giraud, Puech, and Latour) who published during years 1952–1962 a great number of articles dealing with endocardial recordings of various supraventricular arrhythmias originating from the right atrium (RA) and left atrium (LA), the latter with coronary sinus (CS) recordings or through a patent foramen ovale.[2–4] The same authors fortuitously recorded in 1957 the first His bundle activity in a patient with Fallot trilogy,[5] and occasionally while recording RA potentials.[6] Puech and colleagues[7] were among the first who mapped the RA and LA (via esophageal recordings) of patients with atrial flutter (AFL) and concluded that the mechanism of flutter was a macro–re-entry involving the RA.

Defibrillation devices, first devised by physiologists, started to be used in the early 1950s in man by cardiac surgeons, as in Lyon by Santy and Marion in 1950.[8] It took 10 years to have commercially available defibrillators. Paris, Marseille, and Nancy were the first cardiologic centers that reported their initial experience on pacemaker implantations in years 1964–1967.[9–11]

French Rhythmology owes a lot to Yves Bouvrain and Robert Slama, who were the first to create, in the early 1960s, at Lariboisière Hospital in Paris, a cardiologic intensive care unit oriented toward the diagnosis and treatment of cardiac arrhythmias. Programmed cardiac pacing combined with endocardial recordings were initiated in that hospital by Philippe Coumel in 1967.[12] In the early 1970s, Guy Fontaine, and his coworkers from Pitié-Salpêtrière Hospital (Paris), promoted computer use in the electrophysiology laboratory and initiated, first in Europe and in close cooperation with a cardiac surgeon Gerard Guiraudon, the ablation of Kent bundles and ventricular tachycardia (VT) guided by cardiac mapping.[13,14] While 2 Parisian cardiologic centers (Lariboisière and Pitié-Salpêtrière) were among the most active electrophysiologic centers between years 1970–1990, Bordeaux started to play a prominent role at the inception of cardiac ablation of arrhythmias at the end of the 1980s, followed by university hospitals in most French regions.

## Junctional and atrial tachycardias

In 1967, Philippe Coumel and colleagues in Paris[12] and Durrer and colleagues in Amsterdam[15] independently reported a new technique, later called programmed electrical stimulation of the heart. The ability to reproducibly initiate and terminate arrhythmias heralded the beginning of invasive clinical cardiac electrophysiology as a medical discipline.

The same year, Robert Slama and colleagues[16] published the first 2 surgical atrioventricular blocks (AVBs) for intractable rapid atrial tachyarrhythmias. Slama and Coumel were among the first to report on inapparent or latent left accessory

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pathways (APs) in 3 patients.[17] Motté[18] and Coumel[19] were among the first to report on the influence of functional bundle branch block (BBB) on the rate of the tachycardia in patients with overt and latent pre-excitation. Slama and colleagues[20] reported a patient with recurrent drug-refractory tachycardias in whom the slowing of the ventricular rate during rate dependent left BBB (LBBB) played a crucial role in the diagnosis of concealed left free wall AP leading to surgical disconnection of the left atrioventricular (AV) groove.

Coumel and colleagues[21] also synthetized the different mechanisms of AV junctional reciprocating tachycardias. They were the first to assess the inferior point of junction of the re-entry circuit in AV junctional tachycardias using His and high right ventricular (RV) septum simulation. Coumel, Krikler and colleagues[22,23] described several unusual modes of initiation of incessant reciprocating tachycardias in patients with Wolff-Parkinson-White (WPW) syndrome. Barold and Coumel[24] reported an excellent review of the role of re-entry and concealed APs in the mechanism of AV junctional tachycardias.

Coumel and colleagues[25,26] were also the first to describe in children the permanent junctional reciprocating tachycardia, a rare form of nearly incessant orthodromic AV reentrant tachycardia (AVRT) mediated by a concealed, retrogradely conducting AP exhibiting slow and decremental conduction properties. This arrhythmia is frequently called in the literature "the Coumel tachycardia."

Coumel and colleagues[27] reported the first largest electrophysiologic series of sustained intra-atrial re-entrant tachycardia in 20 patients.

Brembilla-Perrot and colleagues[28] assessed the delivery of single and double extrastimuli in 46 patients with WPW syndrome (10 concealed) and 36 control patients with normal ECGs. They found that patients with WPW syndrome and anterograde pre-excitation are more likely to have inducible ventricular fibrillation (VF) and nonsustained polymorphic ventricular tachycardia (PVT) than individuals without WPW syndrome.

Pambrun from Toulouse and other French centers[29] reported the first algorithm for localization of manifest APs in adults using maximal pre-excitation obtained with rapid atrial pacing performed at the beginning of electrophysiologic procedure.

## Ventricular tachycardia

The largest contribution of French authors to the field of VT deals with arrhythmogenic right ventricular dysplasia (ARVD) (see the following). French authors reported several other interesting articles dealing with VT.

Motté and colleagues[30] reported the first largest series of bitachycardia including the association of atrial tachyarrhythmias and VT.

Coumel and colleagues[31] compared the QRS morphology of a hundred of tracings of VT belonging to 85 patients with myocardial infarction to 70 cases of incessant, benign, idiopathic VT. They found several differences in terms of QRS axis, sum of QRS amplitude in unipolar limb leads, and QRS morphology.

Coumel and colleagues[32] investigated electrophysiologically 22 patients with repetitive apparently idiopathic monomorphic VT. Their analysis suggested the presence of an unprotected parasystolic focus. The repetition of the extrasystolic activity, which could be triggered and terminated at will, is explained by phenomena of delayed after potentials.

Brembilla-Perrot and colleagues[33] from Nancy followed 73 patients who had ventricular flutter or rapid monomorphic VT (>270/min) induced during electrophysiologic study (EPS) but no spontaneous VT. They found that inducible ventricular flutter was a nonspecific finding in patients with normal Holter monitoring. In contrast, in patients with salvos of extrasystoles, ventricular flutter was associated with a high risk of cardiac mortality and VT.

Andrade and colleagues[34] from the Fontaine group showed that the diagnostic criterion of VT (RS interval >100 ms) established in patients with organic disease is not valuable in patients with idiopathic verapamil-sensitive left posterior fascicular VT. In this latter group of patients, the RS interval is <80 ms in all of them.

Brembilla-Perrot and colleagues[35] from Nancy found that in patients with coronary heart disease, but without documented VT, the long-term reproducibility of programmed ventricular stimulation (PVS) was excellent in those with inducible VT at initial study (94%); the patients remain at risk of VT, and a prophylactic implantable cardioverter-defibrillator (ICD) could be considered. In patients with initially negative study, reproducibility of PVS was lower (61.5%), probably because of the progressive remodeling after myocardial infarction. Delasnerie and colleagues[36] from Toulouse analyzed the factors underlying clinical tolerance and hemodynamic consequences of 114 episodes of sustained monomorphic VT in 58 patients undergoing an ablation procedure. Clinical tolerance during VT was mainly related to VT rate, presence of resynchronization therapy, location of previous myocardial infarction, presence of concordant VT, and QRS duration during VT.

## Torsades de pointes

Torsades de pointes (TdP) are a unique type of ventricular tachyarrhythmia, which comprises a changing QRS configuration and axis that seems to magically twist around the baseline during repetitive episodes. This arrhythmia was first described by François Dessertenne, a French cardiologist from Lariboisière Hospital. His observations were initially made on 8 patients, including 7 with complete or paroxysmal AVB unrelated to myocardial infarction or to antiarrhythmic medications. The presence of QT prolongation in this form of VT was already mentioned in Dessertenne's original reports,[37,38] yet the causal association between the long QT and the arrhythmia was not recognized. The first 2 princeps articles of Dessertenne published in French in 1966 have been translated into English.[37,38]

Motté, Coumel, and Dessertenne[39,40] reported the main clinical and ECG features, etiologies, and treatment of TdP.

Fontaine and colleagues[41] from Pitié-Salpêtrière reported the initiation of TdP following slow ventricular pacing.

In 2019, Fontaine and colleagues,[42] reviewing tracings of TdP after ventricular temporary pacing interruption in 16 patients with spontaneous high-degree AVB, suggested a phase 2 re-entry mechanism due to early afterdepolarization.

Locati and colleagues[43] from the Coumel group were the first to investigate the cycle length changes preceding the spontaneous onset of TdP during Holter recording in 12 patients with acquired prolonged ventricular repolarization. They found that adrenergic- and pause-dependent mechanisms (possibly inducing afterdepolarizations and triggered activity) may have a synergetic role in the genesis of TdP.

## Catecholaminergic PVT

Catecholaminergic PVT (CPVT) is a rare inherited arrhythmogenic disorder characterized by adrenergic-induced bidirectional and PVT. The first case was reported in 1975 by Reid and colleagues[44] in a 6-year-old girl. In 1978, Coumel and colleagues[45] reported the first world series in 4 children, while in 1995, Leenhardt and colleagues[46] from the same medical center reported a 7-year follow-up of 21 children. Key features include PVT reproducibly induced during exercise tests, isoproterenol infusion, or emotion and exercise. CPVT occurs in children and adolescents and causes syncope and sudden cardiac death (SCD) at a young age, in the absence of structural heart disease. In 2009, Hayashi and colleagues[47] from Lariboisière found that cardiac and fatal or near-fatal events were not rare in both CPVT probands and affected family members during the long-term follow-up, even while taking beta-blockers. The same group[48] found that an exercise-stress test can be used as a simple diagnostic tool for predicting mutations and future cardiac events in CPVT family relatives. In 2012, Lenhardt and colleagues[49] published an extensive updated review of CPVT mainly dealing with the clinical presentation, ECG features, genetic results, and management.

## BBBs and AVBs

The first French cases of rate-dependent BBB and concealed retrograde conduction in the blocked branch as well as of bradycardia-dependent BBB were reported by Slama and colleagues[50] and Motté and colleagues,[51] respectively. Coumel and colleagues[52] reported the first case of paroxysmal AVB elicited by either atrial or ventricular induced premature beats, in which normal AV conduction resumed through appropriately timed junctional escape beats. The mechanisms of the block initiation (phase 4 dependent) and recovery were thoroughly discussed. Guérot and colleagues[53] reported the first case of phase 4 dependent intra-His AVB while the first world series including 5 patients were reported by Motté and colleagues.[54] Valère and colleagues[55] specifically assessed the AV nodal conduction during incremental atrial pacing in 40 patients who had >second-degree infranodal block. They found normal or accelerated AV nodal conduction in 60% and 35% of patients, respectively, suggesting that the AV node is not affected by the pathologic process affecting the infranodal conduction system. Guérot and colleagues[56] were the first to report on the use of ajmaline test in the diagnostic of infranodal conduction disturbances. Motté and colleagues[57] and Fournial and colleagues[58] reported the first largest series assessing the sensitivity and specificity of the administration of ajmaline (1 mg/kg/ at a rate of 1 mg/s), in patients with documented or suspected paroxysmal AVB. The test was considered positive when ≥second-degree infranodal block or doubling the baseline HV interval (or reaching 90 ms) were achieved. The sensitivity of the test was 100%.[57] The specificity of the test was 75% when the syncopal episode was typical for Adams-Stokes[58] and 29% when the syncope was atypical.[58] Other French groups confirmed these results.[59–61]

Perrot and colleagues[60] from Nancy, who reported the results of ajmaline test in 1170 patients, showed that the test could also be beneficial for detecting sinus node dysfunction. Saoudi and colleagues[62] from Rouen were the first to show that the sensitivity of ajmaline (1 mg/kg, 1 mg/s) was greater than with procainamide (10 mg/kg, 10 mg/s). More recently, Maille and colleagues[63] from Marseille were the first to use the ajmaline test in patients who developed LBBB after transcatheter aortic valve replacement (TAVR). Importantly, they found that an infusion of 1 mg/kg over 1 minute in 42 patients, when used in patients without drug contraindications, was safe even in patients who received TAVR in the previous few days.

Lascault and colleagues[64] studied the course of symptoms and the conduction disturbances disorders in 97 patients who had an HV interval >70 ms at EPS, received a pacemaker, and were followed during 26.5 ± 19.5 months. The actuarial incidence of permanent complete AVB was about 5 per 100 per annum until 4 years. The only predictive parameter for such a course was the occurrence of a second- or third-degree AVB prior to pacemaker implantation.

Boulé and colleagues[65] from several centers from the North of France found that, in patients presenting with syncope and BBB, first-degree AVB and left posterior fascicular block increased the likelihood of finding intra-His or infra-His abnormalities. However, no single ECG feature could consistently predict the electrophysiological results.

The respective role of surface ECG and His bundle recordings to assess the risk of AVB after TAVR was studied by Badenco and colleagues[66] from Pitié-Salpêtrière and Bourenane and colleagues[67] from Rennes. Finally, Massoullié from Clermont-Ferrand and colleagues from other French centers reported the first prospective multicenter study dealing with the incidence and management of AV disorders in new onset LBBB after TAVR.[68]

## Autonomic nervous system and arrhythmias

After having studied the substrate of tachycardia and some of its triggers, Coumel and colleagues realized the limitations of programmed electrical stimulation in investigating all the factors in the development of cardiac arrhythmias. They

then used the Holter recording to unravel the role of the autonomic nervous system. Coumel and colleagues[69,70] were the first to describe the entity of "vagal mediated" atrial fibrillation (AF). This entity mostly occurring in relatively young males 30–50 years of age with no obvious heart disease, in whom the AF usually perdures as paroxysmal AF with no progression into permanent AF. The usual history is that of weekly episodes of paroxysmal AF lasting a few hours, occurring predominantly at night with usual arrhythmia conversion at the morning period. Rest, postprandial state, particularly after dinner, and alcohol are also precipitating factors. Patients usually can determine the difference between the wines that preferentially provoke their attacks (red rather than white wines, with the noticeable exception of champagne). The Holter ECG data confirm the role of the autonomic nervous system by showing the progressive sinus rate slowing preceding AF onset. Coumel and colleagues[71] were also the first to show the efficacy of atrial pacing at 90/min (alone or with amiodarone) in the long-term prevention of vagal-mediated AF. Finally, these same authors also showed the efficacy of flecainide alone or in combination with amiodarone in the management of this type of arrhythmias.[72]

They also found that 3 major determinants of spontaneous ectopic activity are present in repetitive monomorphic idiopathic VT[73] and suggested that a mechanism of triggered activity may be responsible for the repetitive activity in this ventricular arrhythmia (VA).

Finally, they also brought original observations on the apparent clinical effect of exercise may be either arrhythmogenic or antiarrhythmic in apparently identical diseases, even in the same patients.[74]

The influence of autonomic nervous system on both atrial arrhythmias (AAs) and VAs were summarized in an outstanding review published in 1993.[75]

The Coumel group[76] also made an important contribution to the field of the mode of initiation of VF in 62 cases recorded during Holter monitoring. The major findings of this analysis were the existence of 2 different terminal events: TdP due to drugs or hypokalemia (n = 13) facilitated by bradycardia and long-short RR sequences, and VF (n = 49) without a preceding pause occurring in the setting of a higher heart rate.

## Arrhythmogenic right ventricular dysplasia

Among the first patients with refractory VTs who underwent epicardial mapping and surgery,[14] Guy Fontaine and colleagues[77] from Pitié-Salpêtrière Hospital discovered that some of them originated from previously undetected abnormal RV. They also identified tiny signals during the epicardial mapping that consistently occurred after the end of each QRS complex on the surface ECG[77] and demonstrated that it was a late activation, with incremental conduction with ventricular pacing rate before VT induction.[78] Myocardial biopsies showed structures of fatty infiltrations with fibrosis and dying myocytes in dilated parts of RVs until then undescribed.[79] Fontaine coined the term ARVD for designing this cardiomyopathy and epsilon wave for designing the small deflection buried in the end of the QRS complex. The name "epsilon wave" was given for 2 main reasons: (1) it is a postexcitation phenomenon that mirrors the pre-excitation delta wave at the beginning of each QRS complex in patients with WPW syndrome; and (2) epsilon is the next Greek letter after delta. The topic of epsilon waves was the subject of an extensive review by the Fontaine group.[80]

ARVD was suggested to explain previous reports of idiopathic right VT and some unexplained cases of sudden death of young people, often during exercise.[81,82] The first large series of ARVD patients was published in 1982 by Frank Marcus,[83] who spent a sabbatical at Pitié-Salpêtrière Hospital with Guy Fontaine. This publication included data on the clinical, ECG, and angiographic characteristics in 24 patients, and also mentioned left-sided locations, which will be later evidenced in another French study.[84] The Marcus publication gave world recognition to the disease prompting the publication of multiple studies around the world and in France by the Lariboisière[85] and the Pitié-Salpêtrière[86] groups.

Although ARVD was initially described as a dominant RV cardiomyopathy, significant progress has been made about its understanding, and a biventricular (BiV) involvement has been recognized. Therefore, the more inclusive term of arrhythmogenic cardiomyopathy has been proposed, although arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D) reflects most of historical work and clinical practice in this field.

The high incidence of AF published by Tonet and colleagues[87] also suggested a more global heart disease. Subsequent studies in the field of ARVD dealt with the evidence of apoptosis,[88] time- and frequency-domain analyses of the signal-averaged ECG,[89] the diagnostic value of isoproterenol testing,[90–92] and the arrhythmogenic response to isoproterenol testing vs exercise testing.[93]

Mijloen and colleagues[94] from Nancy reported an important study using electroanatomic mapping of VT in ARVC/D patients. They found that peritricuspid ventricular re-entry is a frequent mechanism of VT in these patients, which can be identified by detailed 3-dimensional (3D) electroanatomical mapping and guiding ablation.

Another important study by Leclercq and colleagues[95] from Lariboisière assessed the spontaneous occurrence of 43 episodes of sustained monomorphic VT recorded during Holter monitoring in 12 ARVD patients. Increased heart rate and shortening of the coupling intervals of the first cycles was observed before VT occurred and were attributed to an increased sympathetic tone. This increase appears to be the main determinant of VT in ARVD, in contrast to the multifactorial origin of VT due to coronary heart disease.

In 1998, Fontaine and colleagues[86] proposed a new classification of ARVC/D based on his experience of >250 patients and 72 histological samples collected during a period of 23 years in France and 7 other countries. They concluded that the polymorphism and wide clinical spectrum of ARVC in general appears to be the result of 1 and possibly 2 basic

characteristics of the heart musculature of the human species: replacement of myocardium by fat and susceptibility to environmental factors.

In 2004, Hulot and colleagues[96] from the Fontaine group performed a multivariate analysis of the risk factors related to long-term prognosis in 130 ARVD patients recruited between 1977 and 2000. They found that after adjustment for sex, history of syncope, chest pain, inaugural VT, recurrence of VT, and QRS dispersion, clinical signs of RV failure and left ventricular (LV) dysfunction both remained independently associated with cardiovascular mortality. The combined presence of one of these risk factors and VT identifies high-risk subjects for cardiovascular mortality, whereas patients without VT displayed the best prognosis.

More recently, in 2018, Maupain and colleagues[97] from Pitié-Salpêtrière reported a consecutive series of 137 ARVC/D patients without ICD, who were recruited between 2000 and 2010. Of these 137 patients, 95 were symptomatic (42 with sustained VT) and 32 asymptomatic. They found that LV ejection fraction $\leq$50%, positive EPS (induction of sustained VT/VF), and physical activity $>$6 h/wk were independent predictors of sustained VAs. Interestingly, syncope had a 100% sensitivity and 89% specificity to predict SCD/aborted cardiac arrest (ACA). In contrast, asymptomatic patients at diagnosis as well as patients with well-tolerated arrhythmias on drug therapy (at least beta-blockers) were at low risk. EPS predicted all VAs but had limited value to predict SCD/ACA.

Therapeutic approaches in ARVC/D were like those other ventricular arrhythmogenic diseases, including drug therapies,[98] ICD,[99] catheter ablation using fulguration,[100,101] radiofrequency (RF) energy,[102–104] or both.[105]

Finally, in the dawn of the 21st century, new genetic tools allowed to find the link of half of the ARVC/D with desmosome anomalies mainly. An outstanding review of the clinical diagnosis, imaging, and genetics of ARVC/D was published by Gandjbakhch and colleagues[106] from the Pitié-Salpêtrière group.

## Ablation of cardiac arrhythmias

The French contribution to the field of ablation, first using direct current shocks ("fulguration") and later RF energy has been tremendous.

### Historical notes

Catheter ablation (fulguration) was discovered by serendipity by the Guy Fontaine's group at Pitié-Salpêtrière Hospital in Paris.[107] One of their patients had an induced poorly tolerated VT that could not be interrupted by pacing and needed several electrical shocks The last shock was effective and the patient reverted to sinus rhythm, but in complete AVB. The external part of a bipolar electrode placed on the His bundle happened to be in contact with the patient's skin at that moment, and a spark had simultaneously occurred there, demonstrating that some current had reached the His bundle. This case was published by Vedel and colleagues[108] as an unusual accident during an EPS and gave ideas to the arrhythmologic community. His bundle ablation at that time was just an open chest procedure for severe supraventricular tachyarrhythmias, and after animal studies, Frank, Fontaine, and colleagues reported in 1983 the first series of "Therapeutic interruption of the bundle of His by electric shock."[109]

This technique, which was called "fulguration" by Fontaine and colleagues,[110] was the subject of extensive experimental investigation (Supplemental Material), and extended to ablate AFL, APs and VT.

### Fulguration

Multiple French groups have gained experience in the field of fulguration of cardiac arrhythmias, frequently resulting in princeps publications.

#### *Atrial flutter*

Saoudi and colleagues[111] from Rouen reported the first case of fulguration of AFL. Later, Saoudi and colleagues[112] reported a series of 8 patients with drug-refractory AFL referred for AV nodal ablation and achieved satisfactory results with fulguration given at the low septal atrium. Touboul and colleagues[113] identified the low atrial septal area surrounding the CS ostium as being the critical area of slow conduction of the AFL and the optimal ablation site.

#### *Ventricular tachycardia*

Haissaguerre and colleagues[114] from Bordeaux reported the results of fulguration of refractory sustained VT in 31 patients. No serious complications occurred during the procedure. No VT could be inducible in 13 (42%) patients at EPS performed 7–10 days after ablation.

Frank and colleagues[115] reported the results of fulguration performed during an 8-year period in 86 patients with recurrent drug-refractory VT including 21 cases of ARVD, 35 post–myocardial infarction, 11 dilated cardiomyopathy, 10 bundle branch re-entry VT, 5 idiopathic VT, and 3 congenital heart disease. During a mean follow-up of 56 $\pm$ 33 months, 32 (37%) patients were without any antiarrhythmic therapy and had no recurrence of VT

#### *Accessory pathways*

The Bordeaux group, led by Warin and Haissaguerre, reported the first world series of fulguration of APs,[116,117] Mahaim fibers,[118] and the retrograde fast pathway conduction in patients with AV nodal re-entrant tachycardia (AVNRT).[119] The same authors reported the largest series of APs and AVNRT treated with fulguration,[120,121] and the immediate complication of fulguration of APs (64 patients) and VT (21 patients) during the performance of 318 intracardiac shocks applied in 110 sessions.[122] Complete AVB and sinus pauses were the most frequently observed complications but were always transient. Severe complications were rare with no procedure related deaths.

Haissaguerre and colleagues[123] showed that in 135 patients undergoing fulguration of 142 distinct antegradely

conducting APs, the unfiltered unipolar recording mode (PQS pattern) contributed significantly to optimizing the accuracy of AP localization.

Adragao from the Fontaine group[124] reported the factors predicting success in fulguration of APs in 33 patients. The presence of a probable Kent potential was the parameter most strongly associated with success, while the absence of earliest activation recorded on the ablating catheter prior to shock delivery was associated with procedure failure.

*Atrioventricular junction*

Levy and colleagues[125] on behalf the French Cardiac Arrhythmia Working group reported the results of fulguration of the AV junction for refractory supraventricular tachyarrhythmias in 91 patients. Complete AVB was obtained in 83 patients and persisted at hospital discharge in 46 (50.5%) patients. Immediate procedural complications were rare. One patient with additional sustained VT died 3 days after the procedure. At the time of the follow-up, there were 3 additional deaths related to sepsis due to pacemaker pocket infection in 1 patient.

## RF ablation

In the early 1990s, when alternative energies were studied, RF became a source of energy easier and safer to be used everywhere and replaced fulguration.

*Atrial flutter*

Kirkorian and colleagues[126] from Lyon reported good results of RF current directly delivered to the atrial isthmus between the inferior vena cava and the tricuspid ring, regardless of the morphology of local electrograms (anatomically guided approach).

Poty and colleagues[127] from Rouen were the first to report that bidirectional isthmus block may predict the long-term success of AFL RF ablation.

Fisher and colleagues[128] from Bordeaux showed the safety and high efficacy of AFL ablation when RF energy is applied in the isthmus between the inferior vena cava orifice and the tricuspid valve.

In a prospective study involving Lariboisière and Bordeaux, Cauchemez and colleagues[129] found that the mechanism of successful AFL ablation targeting the cavotricuspid isthmus (CTI) is local bidirectional conduction block. This change can be used as a new and complementary electrophysiological endpoint for the procedure. AFL recurrences are associated with failure to achieve a permanent CTI block.

Jais and colleagues[130] from Bordeaux were the first to report the safety and efficacy of irrigated-tip catheter ablation for achieving CTI block when conventional RF energy has failed.

Chen and colleagues[131] from Nancy confirmed that reversal of the atrial depolarization sequence up to the line of block is a definitive and mandatory criteria of successful AFL ablation. Using high-density isthmus mapping during recurrent AFL after a previous catheter ablation in the CTI, Shah and colleagues[132] from Bordeaux found slow conduction through gaps of recovered conduction of varying dimensions in the ablation line followed by a curved front of activation antidromically activating its downstream flank, this detour producing wide double potentials on the line.

Takahashi and colleagues[133] from Bordeaux found that 17% of patients undergoing AFL ablation have pre-existing partial CTI block, indicated by a large contiguous zone of double potentials separated by an isoelectric interval.

Shah and colleagues[134] from Bordeaux reported a simple technique to recognize a persistent gap or complete linear block in the CTI. This technique involves the successive stimulation of the distal and proximal bipoles of a quadripolar catheter placed close to the ablation line.

Kasai and colleagues[135] from Rouen compared the effectiveness of an 8-mm vs a 4-mm tip electrode catheter for ablation of typical AFL and did not find significant differences between the 2.

Jais and colleagues[136] from Bordeaux reported the first world series of left AFL ablation in 22 patients. Complete maps in 17 patients mainly demonstrated macro–re-entrant circuits around the mitral annulus, a zone of block including the pulmonary veins (PVs), or a silent area (n = 15) and a small re-entry circuit with a zone of markedly slow conduction (n = 2). Linear ablation performed across the most accessible part of the circuit cured 16 (73%) patients during a follow-up of 15 ± 7 months.

To assess the best method for diagnosing complete bidirectional CTI block, Anselme and colleagues[137] from Rouen conducted a randomized comparison of 2 established methods (sequential detailed activation mapping during proximal CS and anteroinferior RA pacing vs mapping only the ablation line). Although the first method was better, the second provided additional information in some cases. Andronache and colleagues[138] from Nancy found that morphological changes in bipolar atrial electrogram recorded at sites lateral and adjacent to the target line of block may be used as a unique and robust criterion to validate CTI conduction block during AFL ablation procedures.

In a prospective, randomized clinical study comparing the efficacy of irrigated and large-tip catheters of different designs, Scavée and colleagues[139] from Bordeaux found that the externally irrigated catheter has a higher efficacy for rapid achievement of CTI block.

Laurent and colleagues[140] from Dijon reported a new and simple method for distinguishing complete from incomplete block through the CTI during AFL ablation. This method consists in a combination of lateral vs septal RA pacing sites with the measurement of AV conduction delay on a surface ECG.

Tonet and colleagues[141] from Pitié-Salpêtrière found that the CS musculature is a critical part of some AFL re-entrant circuits in patients with typical and atypical AFL. AFL can be terminated in these patients by targeting CS mid-diastolic fragmented atrial potentials.

*Slow pathway*

Haissaguerre and colleagues[142] were among the first to show the presence of slow potentials at the mid or posterior part of

the septum near the tricuspid annulus. In 64 patients with AVNRT, application of RF energy rendered tachycardia noninducible through the preferential modification of the anterograde slow pathway. No patient had AVNRT over a follow-up period of 1–16 months, and all had preserved AV conduction.

The same group[143] reported their experience in 176 patients with AVNRT who were treated by preferential modification of the fast retrograde (n = 8) or slow anterograde pathway (n = 167) of the re-entry circuit, with a 99% success rate (1 failure) and without significant complications.

### *Accessory pathways*

In 8 patients with left lateral APs that could not be ablated using multiple endocardial attempts with RF energy, Haissaguerre and colleagues[144] showed that successful ablation can be performed safely via the mid or distal CS. The same group[143] reported their experience in 362 patients with 1 or more APs, patent or latent, were treated using the same type of energy. The ablation site was determined based on indirect criteria and/or recording of the specific activity of the AP. The success rate here was 98%, without significant complications.

Haissaguerre and colleagues[145] studied the efficacy and safety of catheter ablation of APs in 79 children (4–16 years of age), using fulguration (n = 25) or RF energy (n = 54). Fulguration was successful in all 25 patients with a single patient developing complete AVB postablation. RF ablation was successful in all 54 patients without significant immediate side effects. After 10 ± 5 months' follow-up, all patients but 1 (98%) were asymptomatic without any drug therapy.

Haissaguerre and colleagues[146] found that parahissian APs have a pre-excitation pattern that is distinctive from that of anteroseptal APs. They could ablate with RF energy these APs in 8 patients using low energy with preservation of normal AV conduction.

Haissaguerre and colleagues[147] studied 21 patients with AVRT or AF associated with antegrade right APs with decremental properties. Long and short atrioventricular APs were observed in 81% and 19% of patients, respectively. Long APs often have a distal arborization and may have either a fascicular or ventricular insertion. RF energy was more efficient when applied to the AP bundle or AP proximal insertion rather than to the distal insertion in patients with long APs.

Yamane and colleagues[148] used irrigated-tip ablation catheters to ablate 18 APs that could not be ablated with conventional RF energy. Six APs were in the left free wall, 5 were in the middle/posterior-septal space, and 7 were inside the CS or its tributaries. Seventeen (94%) of the 18 resistant APs were successfully ablated with a median of 3 applications. No serious complications occurred.

### *Atrial fibrillation*

Unless specifically indicated, the publications on RF ablation of AF originated from the Bordeaux group. Almost all these articles were princeps publications in top cardiology journals.

The first 3 cases of ablation of atrial tachycardia (AT)/AF in the RA were reported in 1994 by Haissaguerre and colleagues.[149] These cases suggested that AF may be linked to "focal" mechanisms that can be treated by catheter ablation.

The same year, Haissaguerre and colleagues[150] reported the case of a 46-year-old man with incessant AF ablated using linear RA lesions created by sequential applications of RF energy.

In 1997, Jais and colleagues[151] reported the successful ablation of a focal source of AF in 9 patients. Three foci were found to be in the RA, 2 near the sinus node, and 1 in the CS ostium. Six others were in the LA at the ostium of the right (n = 5) or the left (n = 1) superior PVs (n = 5). All AAs were successfully treated by use of a mean of 4 ± 4 RF pulses. One year later, Haissaguerre and colleagues[152] reported 45 patients with recurrent AF. A single point of origin of atrial ectopic beats was identified in 29 patients, 2 points of origin were identified in 9 patients, and 3 or 4 points of origin were identified in 7 patients, for a total of 69 ectopic foci. Three foci were in the RA, 1 in the posterior LA, and 65 (94%) in the PVs (31 in the left superior, 17 in the right superior, 11 in the left inferior, and 6 in the right inferior PVs). During a follow-up period of 8 ± 6 months after ablation, 28 (62%) patients had no recurrence of AF.

Shah and colleagues[153] reported the first demonstration of intra-atrial figure-of-8 re-entry in man and its treatment with RF ablation in 5 patients for drug-resistant AT/AFL who had prior surgical closure of an ostium secundum atrial septal defect. A specific type of figure-of-8 re-entry circuit involving simultaneously the atriotomy scar and the tricuspid valve was observed. Ablation of the CTI transected the peritricuspid loop and left a single periatriotomy loop tachycardia, producing an instantaneous change in the ECG pattern, which required ablation at a second isthmus.

Studying 10 necropsied hearts, Chauvin and Brechenmacher from Strasbourg, as well as colleagues from the Bordeaux group[154] sought to determine the histological features of the atrial myocardium connecting the CS and the LA in humans. They found a consistent but morphologically variable LA CS myocardial connection. This emphasizes the need for surgical dissection or catheter ablation in or around the CS to eliminate these connections.

Haissaguerre and colleagues[155] assessed the endpoint for catheter ablation of PV foci initiating AF. In 90 patients who underwent mapping during spontaneous or induced ectopy and/or AF initiation, they found that multiple PV foci are involved in initiation of AF, and elimination of PV muscle conduction is associated with clinical success.

To determine the extent of ostial ablation necessary to electrically disconnect the PV myocardial extensions that initiate AF from the LA, Haissaguerre and colleagues[156] performed PV mapping with a circumferential 10-electrode catheter (called "the Lasso catheter") during sinus rhythm or LA pacing in 70 patients. A total of 162 PVs (excluding right inferior PVs) were ablated. They found that although PV muscle covers a large extent of the PV perimeter, there are specific breakthroughs from the LA that allow ostial PV disconnection by use of partial perimetric ablation.

Yamane and colleagues[157] assessed the value of 12-lead ECG P-wave morphology to recognize the paced PV in 30 patients. P-wave configurations were studied during sinus rhythm and during pacing at 6 sites from the 4 PVs. They found that pacing from the different PVs produced a P-wave with distinctive characteristics that could be used as criteria in an algorithm to identify the PV of origin with an accuracy of 79%.

Yamane and colleagues[158] assessed the anatomical distribution and electrogram characteristics of breakthrough from the LA to the PVs in 157 patients with paroxysmal AF undergoing PV disconnection guided by mapping with a circumferential 10-electrode catheter. They found that bipolar electrogram polarity reversal allows more precise localization of breakthrough compared with the earliest activation, particularly in cases of wide synchronous PV activation.

Shah and colleagues[159] found that far field LA appendage (LAA) activity consistently adds to PV myocardial electrograms in the left superior PV whereas lower, less sharp extra venous potentials in the left inferior PV originate from the inferior LA. These far field activities can be identified by pacing from the LAA and the CS.

To assess the relationship of anatomical dimensions of PVs vs PV arrhythmogenicity, Yamane and colleagues[160] measured the diameters of 4 PVs by selective PV angiography before ablation in 39 patients with only 1 (n = 25 patients) or 2 (n = 14 patients) arrhythmogenic PVs. They found that the diameters of arrhythmogenic PVs were significantly larger than those of nonarrhythmogenic PVs irrespective of the specific PV concerned, which might imply a possible role of PV dilatation in the arrhythmogenesis.

Jais and colleagues[161] compared the studied the effective and functional refractory period and conduction time from PV to LA by use of programmed stimulation with a single extrastimulus in the PVs and LA in 2 groups of patients, 1 with paroxysmal AF and 1 without AF. They found that the PVs of patients with AF exhibited distinctive electrophysiological properties, which were strikingly different from those of patients devoid of AF potentially explaining their arrhythmogenicity.

The study by Choi and colleagues[162] evaluated the use of ectopic P-wave morphology to localize PV and non-PV sources of 54 atrial ectopics in 44 patients with paroxysmal AF. Comparison of subtracted ectopic P waves with a pacemap catalogue provides a simple and accurate 12-lead ECG-based method for localization, which can facilitate ablation of arrhythmia triggers irrespective of origin from the PV or elsewhere.

Hocini and colleagues[163] found that prolonged sinus pauses after paroxysms of AF can be eliminated by curative ablation of AF. This is accompanied by improvement in parameters of sinus node function, suggesting reverse remodeling of the sinus node.

The first world series of 5 patients with AF originating from a persistent left superior vena cava was reported by Hsu and colleagues.[164] The vena cava was electrically connected to the lateral LA and through the CS to the RA. Ablation of these connections resulted in electrical isolation in 4 of the 5 patients without complications.

Haissaguerre and colleagues[165] assessed the modification of AF cycle length during AF. They showed that AF ablation results in a decline in AF frequency, with a magnitude correlating with termination of AF and prevention of inducibility that is predictive of subsequent clinical outcome.

In 2004, Jais and colleagues[166] described their technique and results of linear ablation at the mitral isthmus.

Hsu and colleagues[167] reported the first prospective study of AF ablation in 58 patients with congestive heart failure and LV ejection fractions <45% and compared their results with 58 control patients without heart failure who underwent AF ablation. They found that restoration and maintenance of sinus rhythm by catheter ablation without the use of drugs in patients with heart failure and AF significantly improve cardiac function, symptoms, exercise capacity, and quality of life.

Hocini and colleagues[168] found that anatomically guided circumferential PV ablation results in apparently coalescent but electrically incomplete lesions with residual conduction in 45% of PVs. Wide encircling of the PVs was associated with LA macro–re-entry in 20% of patients.

Sanders and colleagues[169] found that spectral analysis and frequency mapping identify localized sites of high-frequency activity during AF in humans with different distributions in paroxysmal and permanent AF. Ablation at these sites results in prolongation of the AF cycle length and termination of paroxysmal AF, indicating their role in the maintenance of AF.

Reant and colleagues[170] performed serial echocardiographic studies at baseline and at 1-, 3-, 6-, 9-, and 12-month intervals after AF ablation in 48 patients with isolated AF (37 paroxysmal and 11 chronic) associated with mild LA enlargement and LV diastolic dysfunction. They found reverse morphological remodeling of the LA and improvement of LV diastolic and systolic functions after restoration of sinus rhythm by ablation.

Sanders and colleagues[171] assessed 20 patients with AT having failed prior ablation or occurring after AF ablation. They found that high-density multielectrode mapping can be used to perform vector mapping to localize complex tachycardias. It provides novel insight in distinguishing focal tachycardia from localized re-entry.

Hocini and colleagues[172] were the first to report the technique, electrophysiological evaluation, and clinical consequences of complete linear block at roofline joining the superior PVs in patients with paroxysmal AF. They studied 90 patients with drug-refractory AF prospectively randomized into 2 ablation strategies: (1) PV isolation (n = 45) or (2) PV isolation in combination with linear ablation joining the 2 superior PVs (roofline; n = 45). They demonstrated the feasibility of achieving complete linear block at the LA roof. Such ablation resulted in the prolongation of the fibrillatory cycle, termination of AF, and subsequent noninducibility and is associated with an improved clinical outcome compared with PV isolation alone.

In 74 patients with paroxysmal AF, Jais and colleagues[173] performed PV isolation during induced or spontaneous AF. If AF was inducible after PV isolation, 1 to 2 additional linear lesions were placed at the mitral isthmus and/or LA roof, with the endpoint of noninducibility of AF or AFL. This study demonstrates that an individual staged approach using PV isolation and linear ablation guided by inducibility of AAs is associated with long-term success in 91% of patients with paroxysmal AF resistant to AADs.

Haissaguerre and colleagues[174] studied the endocardial sources driving AF in 50 patients with AF organized by prior PV and linear ablation. They found that AF emanates mostly from localized sources that can be mapped and ablated. Some sources harbor electrograms suggesting the presence of localized re-entry.

Takahashi and colleagues[175] aimed to assess the feasibility of identifying sites of focal atrial activity by localized high-density endocardial mapping during AF. They found that termination of AF without ablation at the sites of atrial focal activity suggests that this activity may be triggered by impulses originating from other regions, such as the PVs.

To evaluate the contribution of the posterior LA to chronic AF, Sanders and colleagues,[176] after PV isolation, also isolated the posterior LA by ablation joining the right and left PVs. This study demonstrates the feasibility of complete posterior LA exclusion by catheter ablation. This strategy results in maintenance of sinus rhythm in 63% at 2 years' follow-up.

Jais and colleagues[177] conducted a prospective, randomized controlled trial, comparing a strategy of catheter ablation vs continued medical therapy with AADs in patients with paroxysmal AF who had failed at least 1 AAD. This randomized multicenter study demonstrated the superiority of catheter ablation over AADs in AF patients. The substantial improvement in quality of life, symptoms, and physical performance in this series of relatively young and healthy patients constitutes an important benefit that may support earlier use of catheter ablation in this context.

O'Neill and colleagues[178] prospectively performed catheter ablation in 153 patients with persistent AF using a stepwise approach with the desired procedural endpoint being AF termination. They found that procedural termination of long-lasting AF by catheter ablation alone is associated with an improved outcome.

Matsuo and colleagues[179] evaluated the role of baseline clinical variables to predict procedural and clinical outcomes of catheter ablation in patients with long-lasting persistent AF in 90 patients. They found that the surface ECG AF cycle length is a clinically useful preablation tool for predicting patients in whom sinus rhythm can be restored by catheter ablation.

Hocini and colleagues[180] aimed to assess whether additional ablation in the RA improves termination rate in long-lasting persistent AF. They found that a divergent pattern of AF cycle length prolongation after LA ablation resulted in a right-to-left gradient, demonstrating that the RA is driving AF in approximately 20% of persistent APs.

Miyazaki and colleagues[181] compared the outcomes of circular mapping catheter–guided PV isolation performed in 30 patients using a remote magnetic navigation system and in 44 patients using a conventional hand-controlled ablation technique. They found that the magnetic navigation system–guided PV isolation is feasible. However, it requires longer ablation, fluoroscopy, and procedural times than the conventional approach in the early experience stage.

Weerasooriya and colleagues[182] assessed the 5-year follow-up results of catheter ablation for AF (63% paroxysmal) in 100 patients (175 procedures). Arrhythmia-free survival following the last procedure was 87%, 81%, and 63% at 1, 2, and 5 years, respectively. Patients with long-standing persistent AF experienced a higher recurrence rate than those with paroxysmal or persistent forms. Valvular heart disease and nonischemic dilated cardiomyopathy independently predicted recurrences. Cardiac tamponade requiring drainage occurred in 3 (3%) patients.

Hocini and colleagues[183] found that the LAA is an important source of localized re-entrant AT in patients with persistent AF at index and repeat ablation procedures. Ablation targeting the site with long fractionated or mid-diastolic LAA electrogram is highly effective in acute and medium-term elimination of the arrhythmia.

Rivard and colleagues[184] tested the hypothesis that restoration/maintenance of sinus rhythm preablation would facilitate AF termination and improve outcomes in patients with persistent AF. They found that restoration of sinus rhythm prior to catheter ablation for persistent AF whenever possible decreases the extent of ablation with the same high clinical efficacy.

In 103 consecutive patients with persistent AF, Haissaguerre and colleagues[185] showed that the arrhythmia in early months is maintained predominantly by drivers clustered in a few regions, of these, 69% re-entries and 71% foci were in the LA. Driver ablation alone terminated 75% and 15% of persistent and long-lasting AF, respectively.

Scherr and colleagues[186] aimed to determine the 5-year efficacy of catheter ablation for persistent AF using AF termination as a procedural endpoint in 150 patients using a stepwise ablation approach (PV isolation, electrogram-guided ablation, and linear ablation) with the desired procedural endpoint being AF termination. Such ablation strategy aiming at AF termination was found to be associated with freedom from arrhythmia recurrence in most patients over a 5-year follow-up period.

Scherr and colleagues[187] sought to determine the impact of anatomical location of the ablation line on the efficacy of mitral annulus ablation in 40 patients undergoing stepwise AF ablation. They found that decreased length but not anatomical location of the mitral annulus line as assessed with a 3D mapping system predicts success in achieving bidirectional mitral isthmus block.

Lim and colleagues[188] extensively reviewed the results of 20 studies which directly compared the clinical outcome of patients who achieved AF termination during ablation with those who did not. Most studies (17 of 20) demonstrated significantly improved outcomes in the group with procedural AF termination. Termination of the arrhythmia

provides a robust measurable procedural endpoint, and bolstered by newer AF mapping techniques, facilitates a patient-tailored approach to persistent AF ablation. The authors extensively discussed the mechanisms of procedural AF termination.

Lim and colleagues[189] conducted a multicenter study that sought to characterize the clinical characteristics, atrial substrate, and prognosis in a subgroup of patients (n = 129) with persistent AF from the onset (PsAFonset). They compared them with a group of patients with persistent AF that progressed from paroxysmal AF (n = 231). In addition, 90 patients (30 patients with PsAFonset and 60 control subjects) were studied with noninvasive mapping to characterize the AF drivers. They found that patients with PsAFonset represent a distinct subgroup defined by specific demographics, underlying diffuse biatrial substrate disease, and worse clinical outcome. Lim and colleagues[190] sought to investigate the complexity and distribution of AF drivers in persistent AF of varying durations. They found that the complexity of AF drivers increases with prolonged AF duration. Re-entrant and focal drivers are predominantly located in the PV antral and adjacent regions. However, with longer AF duration, multiple drivers are distributed at extra-PV sites. AF termination rate declines as patients progress to long-standing persistent AF, underscoring the importance of early intervention.

Cochet and colleagues[191] assessed the relationship between fibrosis and re-entrant activity in 41 patients with persistent AF using high-resolution ECG imaging performed during AF; phase mapping was applied to locate re-entrant activity. Late gadolinium enhancement (LGE) cardiac magnetic resonance was performed to characterize atrial fibrosis and measure atrial volumes. The number of re-entrant regions during AF relates to the extent of LGE on cardiac magnetic resonance, with the location of these regions clustering to LGE areas. These characteristics affect procedural outcomes of ablation.

Pascale and colleagues[192] sought to identify ECG characteristics to help identifying the mechanism of 196 AT occurring during or after ablation of symptomatic drug-refractory persistent AF in 127 patients (macro–re-entry in 57%, centrifugal AT in 43%). Surface P waves were analyzed during higher (>2:1) grades of AVB. They found that only 2 unique features could help identify perimitral AT (n = 60): (1) the presence of a negative or negative-positive P-wave in any of leads $V_2$ to $V_6$ identified perimitral AT with 97% specificity and 30% sensitivity; (2) a "notched" negative component at the beginning of a positive P-wave in the inferior leads specifically identified clockwise perimitral AT (specificity 98%, sensitivity 25%).

Takigawa and colleagues[193] assessed the mechanisms of AT recurrence (macro–re-entry vs focal) in patients who had undergone AF ablation. They found that (1) perimitral flutter is the most common type of recurrence of the AF ablation–related AT. Redo AF ablation–related AT ablation procedures demonstrated a true failure rate of 57.7%. Roof-dependent macro–re-entrant AT had a true failure at 44.4%; (2) epicardial structures were included in the circuit in 75% of perimitral flutters, 28.6% of roof-dependent macro–re-entrant ATs, and 28.6% of nonanatomic macro–re-entrant ATs. The high true failure rate in perimitral flutter may be explained by involvement of the CS or the vein of Marshall.

Several important studies dealing with the abolition of Marshall bundle epicardial connections with ethanol infusion have been reported by the Bordeaux group.[194–198] Feasibility, pitfalls, and complications in over 700 patients undergoing vein of Marshall ethanol Infusion were reported by Kamakura and colleagues.[199]

In a research letter, Krisai and colleagues[200] reported the safety and outcome of catheter ablation in 39 hyperthyroid AF patients and found that the procedure was associated with a high acute success rate and no increased risks for periprocedural complications. However, the long-term success rate was lower compared with euthyroid patients, with only 49% of the hyperthyroid patients being arrhythmia free 1 year after ablation (vs 71.8% in the control group).

To examine the relationship between abnormal atrial potentials (AAPs) recorded during sinus rhythm/atrial pacing, which may indicate areas of slow conduction capable of supporting re-entrant AT circuits, Nakatani and colleagues[201] studied 123 re-entrant ATs in 104 patients. They found that AAPs recorded during sinus rhythm/atrial pacing are associated with areas of slow conduction during re-entrant AT. The burden and distribution of AAPs may provide actionable insights into AT circuit features, including in cases in which ATs are difficult to map.

Benali from Rennes and other French centers[202] assessed the optimal ablative strategy for those patients with recurrences of AF after PV isolation despite durable PV isolation (the Impact of Ablation Strategy in Patients With Atrial Fibrillation despite Effective Pulmonary Vein Isolation study). They found that no ablation strategy used alone or in combination during the redo procedure appears to be superior in improving arrhythmia-free survival. LA size is a significant predictor of ablation outcome in this population.

Gandjbakhch from Pitié-Salpêtrière with several other French centers[203] analyzed the rate, incidence, risk factors, and optimal management of atrioesophageal fistula after catheter ablation for AF, performed in 103 French centers between 2006 and 2019. The reported rate of this complication was 0.026% (33 of 129,286 AF ablations) with a stable reported annual incidence over time. Prognosis remains poor with a mortality of 60% and crucially dependent of immediate surgical correction. No clear protective strategy has been proven effective.

### *Ventricular tachycardia*

De Chillou from Nancy with several other French centers[204] performed 3D electroanatomic mapping of 33 episodes of VT in 21 postinfarct patients. They found that such a mapping is helpful in reconstructing postinfarct VT circuits and in defining the characteristics of their related protected isthmi, which are the target for ablation. The wide range of isthmus

width values supports the need of linear RF lesions to eliminate the re-entrant substrate of postinfarct VTs.

Grimard and colleagues[205] from Pitié-Salpêtrière reported the first French series of epicardial RF ablation of VT in 9 years on 32 patients after failure of the conventional endocardial approach. An immediate procedural success on the clinical VT was obtained in 22 (76%) of 29 cases. During a follow-up of 384 ± 405 days, 9 (26%) patients experienced a recurrence of a sustained VT. One patient died from tamponade during the procedure despite surgical drainage.

Jais and colleagues[206] from Bordeaux hypothesized that elimination of local abnormal ventricular activities (LAVAs) during sinus rhythm or ventricular pacing would be a useful and effective endpoint for substrate-based VT ablation. As an adjunct to this strategy, they used a new high-density mapping catheter and frequently used epicardial mapping. Seventy patients with VT and structurally abnormal ventricle(s) were prospectively enrolled. They found that LAVAs can be identified in most patients with scar-related VT. Elimination of LAVAs is feasible and safe and is associated with superior survival free from recurrent VT.

Pauriah and colleagues[207] from Nancy performed a "stepwise" approach to the management of VT with ablation as a first-line treatment in postinfarct patients. An ICD was only implanted if at repeat EPS a poorly tolerated VT could be induced. In a prospective study involving 40 patients with LV ejection fraction between 30% and 50% and a median follow up of 45 months, they found that an ICD did not reduce mortality and that the only prognostic value for mortality was age. The authors concluded that it seems reasonable to leave patients with no re-inducible VT at repeat EPS without ICD.

Maury, de Chillou, and colleagues from several European centers[208] performed primary RF ablation of well-tolerated sustained monomorphic VT (no syncope) without ICD implantation in 136 patients. These patients were compared for all-cause mortality with 378 similar patients who were implanted with an ICD during the same period. After a mean follow-up of 32 ± 27 months, both groups had the same mortality rate of 12%. In the nonimplanted patients, half mortality was due to noncardiological events, and only 4 (2.4%) had sudden deaths (after 1 month in a valvular patient who did not have repeat EPS and after 33, 42, and 75 months in 3 ischemic patients). These data would support a randomized clinical trial comparing this approach with others incorporating implantation of an ICD as a primary strategy.

Komatsu and colleagues[209] from Bordeaux aimed to characterize LAVA in postinfarction VT patients with respect to their anatomic locations. From a total of 18,270 electrograms reviewed in 31 study patients, 1104 LAVAs (endocardium 839, epicardium 265) were identified and analyzed. They found that lateness of LAVAs is affected largely by their locations. The chance of detecting late LAVAs increases when the electrogram onset is later. A substrate-based approach targeting delayed signals relative to the QRS complex may miss critical the arrhythmogenic substrate, particularly in the septum and other early-to-activate regions.

Epicardial RF delivery is limited by fat, bleedings, and the proximity of coronary vessels and phrenic nerve. Komatsu and colleagues[210] from Bordeaux found in 46 patients that once the epicardial site for ablation is identified, an endocardial site guided by the epicardial one may be effective, at least partially, avoiding epicardial risks, in ischemic, nonischemic, or ARVC/D patients.

Using integrated computed tomography scan images with mapping images in 95 consecutive VT patients, Yamashita and colleagues[211] from Bordeaux confirmed that the epicardial electrophysiological VT substrate is often close to coronary arteries the phrenic nerve in patients with nonischemic cardiomyopathy. They concluded that high-resolution image integration is potentially useful to minimize risks of these injuries during epicardial VT ablation.

Laredo and colleagues[212] from Pitié-Salpêtrière reported the 10-year outcomes of monomorphic VT ablation on 34 patients with repaired tetralogy of Fallot (TOF), 71% of them left on no AAD. There were 2 cases of SCD and 4 VT recurrences. Freedom from death and arrhythmia recurrence was 94% at 5 years, 81% at 10 years, and 70% at 20 years. Global survival was 91% at 20 years. Baseline LV ejection fraction <60% was significantly associated with VA recurrence. The authors concluded that ablation can safely address macro–re-entrant monomorphic VT in these patients with an acceptable long-term rate of recurrence in this high-risk population.

Wolf and colleagues[213] from Bordeaux reported long-term outcomes of substrate elimination targeting LAVA for post–myocardial infarction VT in 159 patients undergoing first ablation. They found that substrate modification targeting LAVA for post–myocardial infarction VT resulted in a substantial reduction of VT storm and ICD shocks and up to 49% of patients free from arrhythmia at 5 years after a single procedure. Complete LAVA elimination, multielectrode mapping, and real-time integration were associated with improved VA-free survival.

Walton and colleagues[214] from Bordeaux provided an important study on the potential role of the of the moderator band in the arrhythmogenicity of the RV in humans and sheep wedge preparations. They found that the moderator band presents anatomic and electrical compartmentalization between myocardium and Purkinje fibers, providing a substrate for macro–re-entry. The vulnerability to sustain VT via this mechanism is dependent on moderator band structure and action potential duration gradients between the RV free wall and the moderator band.

Kitamura and colleagues[215] from Bordeaux aimed to assess the effect of substrate-based VT ablation targeting LAVA on recurrent VF events in 21 patients with structural heart disease. They found that the total number of VF events after ablation targeting LAVAs decreased from 80 to 3, from a median of 1.0 (range 1–29) to 0.0 (range 0–1) in the 6 months before and after ablation. These results suggest that VT and VF share overlapping arrhythmogenic substrates in patients with structural heart disease.

Maury and colleagues[216] from Toulouse and Bordeaux sought to study surface ECG waveforms and effect of

ablation in long-lasting VF in patients with left assist devices. They recorded electrical recordings of a median of 24 min of VF in 3 patients. RF ablation was performed during VF in 2 patients. Dominant frequency decreased after RF ablation in both cases and VF terminated spontaneously shortly after ablation in 1 case. The previously incessant VFs in these 2 patients did not recur afterward.

Haissaguerre and colleagues[217] from Bordeaux performed detailed mapping of the activities underlying the onset of VF and targeted ablation in patients with structural cardiac abnormalities using body surface mapping coupled with computed tomography scan, and a specific software. They found that the onset of human VF is sustained by activities originating from Purkinje and structural substrate, before spreading throughout the ventricles to establish disorganized VF. Targeted ablation resulted in effective reduction of VF burden.

## Long QT syndrome

Lupoglazoll and colleagues[218] from Paris and Lille investigated the notched T waves proposed as a phenotypic marker of LQT1 and LQT2. They found that Holter recording analysis is superior to the 12-lead ECG in detecting grade 1 and grade 2 T-wave notches. These repolarization abnormalities are more indicative of LQT2 vs LQT1, with G2 notches being most specific and often reflecting HERG core domain missense mutations.

Haissaguerre and other French coworkers[219] were the first to report on mapping and ablation in 4 patients with long QT syndrome (LQTS) and malignant arrhythmias. In 1 patient, premature beats originated from the right ventricular outflow tract (RVOT) and in 3 from the left Purkinje conducting system. In the latter patients, repetitive beats were also preceded by Purkinje activity with a variable delay ranging from 20 to 110 ms.

Denjoy and colleagues[220] from Lariboisière analyzed data from 186 patients with Jervell and Lange-Nielsen syndrome obtained from the literature (31%) and from individual physicians (69%). They found that JLN syndrome is a most severe variant of LQTS, with a very early onset, triggered by emotions or exercise, with major QTc prolongation, and poor response to beta-blockers. Subgroups at relatively lower risk for arrhythmic events include females and those with mutations on KCNE1. Patients with a QTc $>$550 ms and syncope during the first year of life are at higher risk. Early therapy with ICD must be considered.

In a retrospective multicenter study involving 9 French hospitals, Delannoy from Nantes[221] studied the long arrhythmic outcome of 36 patients with Andersen-Tawil syndrome, a channelopathy linked to mutations in the KCNJ2 gene. They found that despite a severe clinical presentation with a very high rate of VAs, the arrhythmic prognosis of the Andersen-Tawil syndrome patients is relatively good under treatment including beta-blockers.

## Brugada syndrome

Haissaguerre and colleagues[219] reported the first 3 patients with Brugada syndrome (BrS) and VAs who had successful endocardial ablation of RVOT triggers (2 patients) or anterior RV Purkinje network (1 patient). In the first 2 patients, VF inducibility was modified after ablation.

Hermida and colleagues[222] from Amiens and Paris assessed the effects of hydroquinidine on inducibility of PVT/VF at PVS in 31 asymptomatic patients with Brugada ECG and found that the medication was effective in 76% of the patients. The medication also abolished multiple appropriate ICD shocks in 5 patients.

Bordachar and colleagues[223] from Bordeaux prospectively assessed the incidence, therapeutic implications, and prognosis of AAs in 59 BrS patients (24% with a history of syncope or ACA). The AA incidence during 34 $\pm$ 13 months follow-up was 20% in BrS patients vs 0% in control subjects. Ventricular inducibility was significantly related to AAs during follow-up. The incidence of AAs was greater in patients with spontaneous type 1 BrS (26% vs 10% in those with drug-induced BrS). Multivariate analysis identified the implantation of a single-chamber device as an independent predictive factor of inappropriate ICD discharges.

In 2006, Sacher from Bordeaux and other French centers[224] reported the outcome of 220 patients with type 1 Brugada ECG who received an ICD for ACA (8%), syncope (40%), inducible VT/VF (in asymptomatic patients) (45%), or various reasons (7%). During a follow-up of 38 $\pm$ 27 months, no patient died and 18 (8%) patients had appropriate device therapy (annual event rate of 2.6%). There was a significant risk of device-related complications (8.9% per year).

In 2013, the same authors[225] reported long-term outcomes in a larger series of 378 patients with type 1 Brugada-ECG who received an ICD for ACA (8.2%), syncope (47.9%), or inducible VT/VF (in asymptomatic patients) (43.9%). Appropriate device therapy rates at 10 years were 48% for ACA patients, 19% for those with syncope, and 12% for the asymptomatic patients. At 10 years, rates of inappropriate shock and lead failure were 37% and 29%, respectively.

In a multicenter French study involving 58 patients (8 women) with severely symptomatic BrS (ACA or appropriate ICD shock), Sacher and colleagues[226] made the following 2 observations: (1) in contrast to men, most women do not have a spontaneous type 1 Brugada ECG pattern; and (2) the degree of ST-segment elevation is less pronounced in women. Therefore, risk factors established from a predominantly male population may not be helpful in identifying high-risk women.

Bonny and colleagues[227] from Pitié-Salpêtrière reported the first series of Black Africans exhibiting a Brugada ECG pattern. There were 4 men and 1 women. Convulsive syncope was noticed in 1 patient with a family history of sudden death. Sustained VT/VF was inducible during PVS in 3 patients. An ICD was implanted in 2 patients. *SCN5A* mutation was not found in any of the patients.

Hayashi and colleagues[228] from Lariboisière performed PVS in 21 patients with a type 1 Brugada ECG. They found that the repolarization restitution property is a contributing factor to the propensity for VF in BrS, and regarding this

property, the RV apex plays a more important role than the RVOT.

Roten and colleagues[229] from Bordeaux compared the effects of isoproterenol on J waves: 20 patients with Brugada ECG and 38 patients with early repolarization (ER). They found important differences of J-wave behavior during beta-adrenergic stimulation: right precordial and inferior J waves may persist in a subset of patients, whereas lateral J waves always normalize. The reason for this distinctive regional sensitivity of J waves remains speculative.

Studying 57 patients with BrS and syncope, Sacher and colleagues[230] from Bordeaux found that clinical features suggested an arrhythmic cause in 40% of patients, a nonarrhythmic cause in 30%, and a doubtful cause in the remaining 30%. The VA rate was 5.5% per year in the first group but nil in the other 2 groups.

Rollin from Toulouse and other French centers[231] assessed the prevalence, characteristics, and prognostic value of type 1 ST-segment elevation and ST-segment depression in the peripheral ECG leads in a large cohort of patients with BrS. ECGs from 323 BrS patients with spontaneous (n = 141) or drug-induced (n = 182) type 1 BrS ECG were retrospectively reviewed. They found that type 1 ST-segment elevation in the peripheral ECG leads can be seen in 10% of the patients with BrS and is an independent predictor for a malignant arrhythmic event.

Maury from Toulouse and colleagues[232] from Montpellier prospectively assessed the prevalence of type 1 ST-segment elevation as elicited during pharmacologic challenge with class 1C drugs in a subgroup of patients with myotonic dystrophy. They found that type 1 Brugada ECG is elicited by a class 1C drug in 18% of type 1 myotonic dystrophy patients presenting with minor depolarization/repolarization abnormalities at baseline, but the finding seems to be devoid of a prognostic role.

Bouzeman and colleagues[233] from Lariboisière evaluated the long-term efficacy and safety of an electrophysiological-guided therapy with hydroquinidine among 44 asymptomatic BrS patients with inducible VF. Their long-term follow-up results emphasize that the rate of arrhythmic events among asymptomatic Brugada patients with inducible VF remains low over time. Their results also suggest that residual inducibility under hydroquinidine of limited value to predict events during follow-up.

Gandjbakhch from Pitié-Salpêtrière and other French centers[234] provided the first detailed description of a familial malignant proarrhythmic response to ajmaline associated with a novel *SCN5A* mutation. They concluded that one should be cautious with sodium-channel blocker challenge (SCBC) in suspected *SCN5A* mutation carriers, especially in the presence of conduction abnormalities on baseline ECG.

Maury from Toulouse and other French centers[235] studied the correlation of the time interval between the peak and the end of the T-wave (a marker of transmural dispersion of repolarization that has been linked to malignant VAs in various settings) with arrhythmic events in a large cohort of BrS patients. They found that the time interval between the peak and the end of the T wave in the precordial leads is highly related to malignant VAs in this large cohort of patients with BrS.

The QUIDAM study was a prospective French multicenter randomized (hydroquinidine vs placebo) double-blind study with two 18-month crossover phases in patients with BrS and implanted with an ICD. Andorin and colleagues from Nantes[236] found that although hydroquinidine seems to be effective in preventing life-threatening VA, it could not be an alternative for ICD implantation. Its frequent side effects greatly reduce its probable compliance and therefore do not reveal a significant effect.

Therasse from Nantes and other French centers[237] performed 672 SCBC tests in 137 families with at least 2 subjects affected by BrS who were enrolled and followed prospectively. Of the 672 SCBC tests performed, 337 (50%) were positive. The authors found that the risk of complication during the test is considerably increased in the case of familial history of complicated SCBC, in young patients, and in the presence of decreased ECG conduction parameters. However, QRS enlargement during the test is not directly related to complication.

The same authors[238] demonstrated that ajmaline challenge presents an excellent sensitivity that may rule out the diagnosis of BrS when negative. Conversely, a negative flecainide challenge may not prevent from BrS inheritance and risk of SCD. This may lead to suggest systematic use of ajmaline during the SCBC test.

Berthome from Nantes and other French centers[239] described the clinical characteristics and arrhythmic risk factors in the largest series of BrS women ever reported (n = 493). They found that women represent a lower-risk group than men among individuals with BrS and exhibited their first arrhythmic event 5 years later than men. They also less frequently had a spontaneous type 1 BrS ECG pattern. In asymptomatic women, fragmented QRS and QRS >120 ms seem to be the only event predictors.

Delinière from Lyon and other centers[240] described various ECG markers of depolarization and repolarization that may indicate an increased arrhythmic risk in BrS patients.

In a study involving 15 French tertiary centers, Minier from Nantes[241] described the clinical characteristics and variations in rhythmic risk according to age in 1613 BrS patients followed prospectively. During median follow-up of 5.5 (2.1, 10.0) years, 91 patients experienced an arrhythmic event, including 7 (13%) in the youngest group (<17 years of age), 80 (6%) in middle-age group (17–59 years of age), and 4 (1%) in the oldest (>60 years of age) patients. Annual event rates were 2.1%, 1%, and 0.3%, respectively ($P < .01$). These results suggest the need for early and extensive familial screening.

Probst from Nantes and other French centers[242] investigated the accuracy of the BrS risk scores in the largest cohort of BrS patients ever prospectively enrolled (n = 1613). They concluded that the 2 well-known Shangai and Sieira risk scores identify very high- and low-risk patients but do not allow stratifying the risk of arrhythmic events in intermediate-risk patients.

Kamakura from Bordeaux[243] evaluated the anatomic obstacles that prevent ablation of epicardial abnormal potentials (EAPs) in 16 BrS patients with previous VF and to investigate the feasibility of the elimination of these potentials by endocardial RV ablation. They found that EAPs are near the coronary arteries in most BrS patients, thereby requiring caution during epicardial ablation, whereas epicardial fat is less of an issue. Endocardial ablation is feasible to eliminate some EAPs and may be combined with epicardial ablation.

In 2023, Cheniti from Bordeaux and the group of Nantes[244] provided the first world demonstration that a subset of patients with BrS present an abnormal substrate extending onto the LV epicardium and inferior RV. This LV involvement is associated with *SCN5A* mutations and multigenic variants.

## Idiopathic VF

Leenhardt and colleagues[245] from Lariboisière were the first to describe a new arrhythmia entity in 14 patients (50% men, 34.6 ± 10 years of age) with syncope, no structural heart disease, and normal QT intervals. This entity consisted of PVT in which the first beat of the arrhythmia has a very short coupling interval (245 ± 28 ms). In 10 patients, the PVT deteriorated into VF. Four patients had a family history of SCD. During a mean follow-up of 7 years, there were 5 deaths (4 SCD). In these patients, verapamil was the only drug apparently active on the arrhythmias; however, it did not prevent sudden death (quinidine was not tried in any of these patients).

The first articles on mapping and ablation of idiopathic VF were published in 2002 by Haïssaguerre and his coworkers from Bordeaux.[246,247] The first publication dealt with a series of 16 patients,[246] while the second dealt with 27 patients.[247] In both articles, the triggers of VF were mainly found in various locations with the distal Purkinje system (mostly the LV septum) and less commonly from the ordinary myocardial muscle. The coupling intervals of the first ventricular beat initiating VF were short. The accuracy of mapping was confirmed by acute elimination of premature beats during focal RF ablation.

In 2009, Knecht and colleagues[248] gathered the Bordeaux results with those of 5 other French and foreign centers and reported the long-term follow-up of 38 patients who underwent ablation of idiopathic VF initiated by short-coupled PVCs (SC-PVCs). Triggering PVCs originated from the right (n = 16), the left (n = 14), or both (n = 3) Purkinje systems and from the myocardium (n = 5). During a median postprocedural follow-up of 63 months, 7 (18%) of 38 patients experienced VF recurrence at a median of 4 months. Five of these 7 patients underwent repeat ablation without VF recurrence. The median number of significant malignant arrhythmic events was reduced from 4 (interquartile range 3–9) before to 0 (interquartile range 0–4) after ablation ($P = .01$).

Haïssaguerre and colleagues[249] performed detailed electrophysiological endocardial and epicardial mapping in 24 patients who survived idiopathic VF. During sinus rhythm, areas of abnormal electrograms were identified in 15 (62.5%) of 24 patients revealing localized structural alterations: in the RV in 11, the LV in 1, and both in 3. In the 9 patients without structural alteration, they observed a high incidence of Purkinje triggers (7 of 9 vs 4 of 15; $P = .033$). Catheter ablation resulted in an arrhythmia-free outcome in 15 of 18 patients at 17 ± 11 months' follow-up.

In 2020, Haïssaguerre and colleagues[250] provided an extensive review of the diagnostic value of systematic investigations and the new insights provided by detailed electrophysiological mapping in patients with idiopathic VF.

Surget from Bordeaux and other French centers[251] studied 83 patients who had Purkinje extrasystole–initiated idiopathic VF. They found that these extrasystoles originate dominantly from the RV in men and from the LV or both ventricles in women, adding to other sex-related arrhythmias such as BrS or LQTS.

Escande from Bordeaux along with other French and foreign centers[252] assessed the prevalence of SC-PVCs (induced by ajmaline or flecainide) in 335 patients with suspected or documented severe VAs. They found 16 (4.8%) patients in whom SC-PVCs were induced during drug testing, originating from the Purkinje system in 12 patients. Repetitive PVCs were induced in 15 patients (94%), including PVT in 9 (56%). This inducibility was highly reproducible. Catheter ablation was performed in 9 patients, resulting in arrhythmia elimination in 8 with a follow-up of 6 (interquartile range 2–9) years.

Haissaguerre and other French centers[253] assessed the prevalence of SC-PVCs during SCBC tests, adrenaline, or isoproterenol in 52 patients who had previously documented SC-PVC–related idiopathic VF. Twenty-nine (56%) had an arrhythmic response especially using SCBC tests. SCBC test, isoproterenol, and adrenaline induced an arrhythmic response in 48%, 22%, and 12% patients, respectively. A second SCBC test showed reproducible results in 5 of 5 patients. An arrhythmic response was seen more often after ajmaline than after flecainide. An arrhythmic response to SCBC testing unexpectedly correlated with an incomplete therapeutic effect of quinidine.

## ER syndrome

Haïssaguerre and colleagues[254] reviewed data from 206 case subjects at 22 French and foreign centers who had ACA due to idiopathic VF and assessed the prevalence of ER. They compared their results with a control matched group comprising 412 subjects. They found that ER was more frequent in case subjects with idiopathic VF than in control subjects (31% vs 5%; $P < .001$). Among case subjects, those with ER were more likely to be male and to have a history of syncope or cardiac arrest during sleep than those without ER. During a follow-up of 61 ± 50 months, defibrillator monitoring showed a higher incidence of recurrent VF in case subjects with a repolarization abnormality than in those without such an abnormality.

From a multicenter cohort of 122 patients (90 male subjects, 37 ± 12 years of age) with idiopathic VF and ER in

the inferolateral leads, Haïssaguerre and colleagues[255] found that multiple recurrences of VF occurred in 27% of patients. Isoproterenol in acute cases and quinidine in chronic cases were effective AADs.

Roten and colleagues[256] from Bordeaux compared the effects of the administration of intravenous ajmaline (1 mg/kg) on the inferolateral repolarization in 31 patients with ER, 21 patients with BrS, and 22 control subjects. They found that ajmaline significantly decreases the J-wave amplitude in inferolateral ER and prolongs the QRS width significantly less than in patients with BrS or control subjects. This indicates a different pathogenesis for both disorders.

Roten and colleagues[229] from Bordeaux analyzed the impact of isoproterenol on J waves in 20 patients with Brugada-type ECG and 38 patients with ER and found that J-wave syndromes have distinct regional sensitivity to beta-adrenergic stimulation. J waves may persist in a subset of patients with right precordial and inferior J waves but never in a lateral location.

Gourraud and colleagues[257] from Nantes screened relatives of 4 families affected by ER syndrome and performed Valsalva maneuver in affected and unaffected family members to decrease heart rate and thus increase or reveal an ER pattern. They found that the test resulted in increased J-wave amplitude for 17 of 20 affected patients, revealing an ER pattern in 17 relatives in whom 5 are obligate transmitters of an ER pattern.

In a multicenter study conducted by the Bordeaux group, Mahida and colleagues[258] assessed the VF inducibility at PVS in 81 patients. A standard PVS protocol was used. They found that despite a recent history of ACA, VF was inducible in only 18 (22%) of 81 patients. During follow-up of 7.0 ± 4.9 years, the proportion of patients who experienced VF recurrence was identical (33%) in the 2 groups with or without inducible VF at EPS.

Considering the great prevalence of inferolateral ER, Roten from Bordeaux[259] conducted a multicenter study for evaluating the potential role of T-wave parameters to differentiate between malignant and benign ER. They found that patients with malignant ER have a higher prevalence of low-amplitude T waves, lower T/R ratio (lead II or V5), and longer QTc interval. The combination of these parameters with J-wave amplitude and distribution of J waves may improve identification of malignant ER.

Maury and colleagues[260] from Toulouse assessed the prevalence of ER in a large unselected children populations comprising 1000 successive healthy children from birth to 17 years of age. They found that ER is present in a quarter of children of various ages and is related to ethnic origin, an older age, a slower heart rate, and a higher Sokolow index.

Haissaguerre and colleagues[261] provided an extensive review of the topic of ER, an entity considered not only a benign ECG variant, but also responsible for malignant VAs in a small subset. They demonstrated that distinct substrates, delayed depolarization, and abnormal ER underlie inferolateral J-wave syndromes, with significant implications. They proposed a new simplified mechanistic classification of SCD without apparent structural heart disease.

The outcome of patients with an ER pattern and syncope was assessed by Kamakura from Bordeaux[262] in a multicenter French study involving 97 patients who were implanted with a device allowing ECG monitoring, including 84 with an implantable loop recorder. During a follow-up period of 68 ± 34 months, 16 arrhythmias presumably responsible for syncope were documented (3 VF, 2 VT, 10 bradycardias, and 1 supraventricular tachycardia). Additionally, recurrent syncope not associated with ECG documentation occurred in 16 (11.2%) patients. The cause of syncope was identified in 23 (23.8%) of 97 patients with a monitoring device. The 5-year incidence of VAs and arrhythmic events presumably responsible for syncope was 4.9% and 11.0%, respectively.

## Implantable cardioverter-defibrillators

Martins and colleagues[263] from Nancy showed that a programmed high number of antitachycardia pacing (n = 10) attempted before shocks for patients experiencing fast VT could reduce the occurrence of shocks delivered by the ICD.

Sadoul from Nancy and other French centers[264] showed that arrhythmic mortality was comparable in patients with or without defibrillation testing during ICD implantation.

In this randomized controlled prospective study, Guédon-Moreau from Lille and other French centers[265] found that remote monitoring of ICDs was associated with less shocks in general and inappropriate in the group undergoing remote monitoring. This induced a longer battery longevity in this group. No effect was found on major adverse events including death from any cause, cardiovascular, and procedure- or device-related major adverse events.

Ascoeta from Saint Denis and other French centers[266] a described the characteristics of complications following ICD implantation for primary prevention. The complication rate was 13.5%, mainly related to lead dislodgment or pocket hematoma. Severe renal failure, cardiac resynchronization therapy (CRT) and anticoagulation therapy were independently associated with these complications. Early complications were associated with late complications and overall mortality.

Hamon from Créteil and other French centers[267] found that patients with cardiac amyloidosis and ICD implantation (84% for primary prevention) had at least 1 appropriate ICD therapy in 27% of the population after a mean follow-up of 5 months. No specific factors associated with ICD therapy were identified. However, higher N-terminal pro–B-type natriuretic peptide level and light chain cardiac amyloidosis (AL type) were associated with worse prognosis.

Boulé and colleagues[268] from Lille evaluated the possibility to avoid ICD shocks with remote monitoring. In 90 patients consulting in emergency for ICD alert, 24% were potentially avoidable. Antitachycardia therapy detected by remote monitoring preceding the occurrence of shocks could help the physician to reduce ICD shocks.

Escande from Lille and other French centers[269] found that electrical storms are rare events (1% per year) in patients implanted with ICD for primary prevention and are more likely to occur among men, in patients with renal failure, and in those patients with a low LV ejection fraction <30%.

Clementy from Tours and other French centers[270] evaluated the impact of very high programming therapeutic zones in ICD on appropriate and inappropriate therapy. They found that this programming strategy (very high-rate group: zone 170–219 beats/min; VF zone ≥220 beats/min with 13 ± 4 detection intervals) was associated with a significant 60% reduction of appropriate, inappropriate therapy, and SCD.

Zakine from Paris and other French centers and colleagues[271] compared retrospectively the outcome of ICD implanted in primary prevention in patients older than 80 years of age with a younger population (mean 62 years of age). They found that three-quarters of this old population had no more than 1 associated comorbidity and the rates of appropriate therapies and device-related complications were similar in these two populations.

Galand from Rennes and other French centers[272] evaluated retrospectively the electromagnetic interference (EMI) between LV assist device (LVAD) and ICD. The majority (60.8%) of LVAD patients (n = 659) were implanted with an ICD before LVAD implantation. The rate of EMI was 4.1%. Regarding LVAD devices, the HeartMate 2 (Abbott, Abbott Park, IL) was more frequently involved, representing 18 of 20 of the LVAD devices experiencing interference. Finally, 17% and 10% of patients with the associations of MicroPort/HeartMate 2 and Abbott/HeartMate had interference, respectively. Half of patients with EMI required ICD replacement.

Waldmann from Georges-Pompidou Hospital and other French centers[273] evaluated the outcome of patients with TOF and ICD in a retrospective Nationwide French Registry. They found, on 165 patients, that the annual rate of appropriate therapy was high (10.5% per year), with 7.1% and 12.5% in primary and secondary prevention, respectively. The rate of ICD complication was also high (8.7% per year) including inappropriate therapy. This high rate of complication underlines the need for improving candidate selection for ICD implantation.

Garcia from Poitiers and other French centers[274] evaluated the use of a wearable cardioverter-defibrillator. They found, on 1157 patients, that the rate of appropriate shock was 1.6% over a mean time of 62 days that is equivalent to 7.2% per year. A patient-response button allowed the shock to be aborted in 35.7% of well-tolerated sustained VAs and in 95.4% of inappropriate VA detection, finally resulting in an inappropriate therapy in 8 (0.7%) patients. Finally, the tolerance of this device was correct with a mean daily wearing time of 23.4 hours.

Alonso from Ambroise-Paré Hospital (Paris) and colleagues[275] evaluated in a French national registry different strategies in case of ICD lead fracture: ICD lead replacement, extraction, or abandonment and the outcome of patients according to the strategy used. The decision of the strategy was related to operator experience in lead extraction, patients age and comorbidities, and lead dwelling time. No difference was observed in outcomes in both groups.

Waldmann from Georges-Pompidou Hospital and other French centers[276] evaluated the impact of sex-related differences outcome in patients with TOF and ICD in a retrospective nationwide French registry. They found, on 165 patients, that women represented 30% of the population and that the outcome in terms of appropriate therapy and ICD complications were similar in men and women.

Waldmann from Georges-Pompidou Hospital and other French centers[277] compared the outcome of patients with TOF and ICD in a retrospective nationwide French registry, according to the implantation of a subcutaneous ICD (S-ICD) vs endocardial/epicardial ICD. The results on 238 patients showed that an S-ICD was implanted in 14.3% of TOF patients. The use of S-ICD was associated with a similar incidence of appropriate ICD therapies but with a higher incidence of appropriate ICD shocks. Also, after a median duration of follow-up of 2.5 years for patients with S-ICDs, the overall burden of ICD-related complications was similar in both groups.

Waldmann from Georges-Pompidou Hospital and other French centers[278] studied specifically the population of patients with congenital heart disease in a population implanted with an S-ICD. This population represented 2.1% of the total population (n = 4924). The congenital heart disease population was younger, more frequently female, more likely to receive an S-ICD for secondary prevention, and less likely to have severe systemic ventricular systolic dysfunction. The annual rates of appropriate ICD therapies and complications were similar in both groups.

Laredo from Pitié-Salpêtrière and other French centers[279] evaluated in patients with TOF and ICD the relationship between the VT cycle length of the first VT episode and the development of future rapid VT (cycle length ≤250 ms) or VF. They found that in this population none of the patients with a first documented nonrapid VT episode had rapid VT/VF during follow-up.

## Cardiac resynchronization therapy

In a landmark case report, Cazeau and colleagues[280] from Saint-Cloud presented the first case report of a heart failure patient that was improved with an atrial and ventricular resynchronization. Interestingly, the technique to perform atrial and ventricular resynchronization was a Y connector connected to a dual-chamber pacemaker (DDD). The LA lead was placed in the CS and the LV pacing was obtained surgically with an epicardial lead.

In a small-sample-size study (n = 8), the same group[281] found that those patients with severe systolic heart failure and wide QRS experienced a significant acute hemodynamic and clinical improvement with BiV pacing.

Blanc and colleagues from Brest[282] compared the invasive hemodynamic modification induced by 3 types of ventricular

pacing, lone RV, lone LV, and BiV, in patients (n = 27) with systolic heart failure and first-degree AV block and/or an intraventricular conduction defect. They found that lone LV pacing and BiV pacing induced similar acute hemodynamic improvement compared with lone RV pacing.

Daubert and colleagues from Rennes[283] described the technique of LV lead implantation inside the CS. In this first experience, they demonstrated on 47 patients that LV lead positioning inside ventricular epicardial veins was possible with CS cannulation and that LV lead pacing parameters were acceptable acutely and during midterm follow-up.

Leclercq and colleagues from Rennes[284] demonstrated that AV optimization and BiV pacing induced acute hemodynamic cardiac improvement measured with a Swan-Ganz catheter in 18 patients presenting systolic heart failure and wide QRS duration.

In an editorial, Barold and Cazeau from Rennes[285] reminded the first 4 original publications indicating that AV synchrony and cardiac resynchronization obtained with cardiac pacing could improve cardiac function in patients with systolic heart failure and AV node and intraventricular conduction disease.

Jais and colleagues from Bordeaux[286] found that endocardial LV pacing was feasible to perform cardiac resynchronization. They evaluated 11 patients presenting systolic heart failure and wide QRS. LV lead implantation was performed via a transseptal puncture. This technique was associated with hemodynamic cardiac improvement and necessitated continuous anticoagulation therapy.

Garrigue and colleagues from Bordeaux[287] evaluated specifically the effect of cardiac resynchronization in 12 patients presenting systolic heart failure and wide QRS (≥140 ms) secondary to right BBB. They found a significant improvement in acute cardiac hemodynamic parameters and midterm follow-up at 12 months.

Cazeau from Saint-Cloud as well as other French and foreign centers[288] evaluated in this phase 3 prospective crossover (2 months in each group) randomized study, the effect of BiV pacing in patients with systolic heart failure and wide QRS (≥150 ms). They included 67 patients in sinus rhythm. The primary endpoint was the distance walked in 6 minutes. The authors found a significant increase in the primary endpoint and an improvement of cardiac function and less hospitalization for heart failure during the period of cardiac resynchronization.

Touiza and colleagues from Brest[289] compared the effect of lone LV pacing and BiV pacing at 6 months in 33 patients (n = 18 for LV pacing and 15 for BiV pacing) with severe chronic systolic heart failure and wide QRS and LBBB (>140 ms). They found no statistical difference in these 2 groups on clinical and echocardiographic outcome but a trend toward a higher effect of BiV pacing on LV ejection fraction and QRS duration reduction.

Garrigue and colleagues from Bordeaux[290] compared prospectively lone LV pacing and BiV pacing in 13 patients with severe systolic heart failure, and wide QRS (≥140 ms) chronic AF, undergoing AV node ablation. This study had a crossover design with 2 months in each pacing mode. They found that BiV was superior to LV pacing to improve acute cardiac hemodynamics, assessed with a peak endocardial acceleration sensor and better cardiopulmonary exercise test parameters.

Fatemi and colleagues from Brest[291] evaluated the electrical parameters of a new specific unipolar lead for LV pacing with a specific S shape to potentially reduce LV dislodgement. This lead was named AESCULA 1055K lead from St. Jude Medical (St. Paul, MN). They found that, on 43 patients, lead electrical parameters including threshold were stable and acceptable at 6 months. They observed 6 lead dislodgements.

Blanc and colleagues from Brest[292] found that, on 22 patients with systolic heart failure, LBBB and in sinus rhythm, pure LV pacing was associated with clinical and echocardiographic improvement at 12 months' follow-up.

Ennezat and colleagues from Lille[293] found that CRT was associated with mitral regurgitation improvement in 21 patients at baseline and during exercise.

Bordachar and colleagues from Bordeaux[294] provided a comprehensive review that discussed the advantages and disadvantages of LV endocardial stimulation, examined the various techniques of LV endocardial stimulation, and projected their future applications considering these highly promising recent results.

Reant and colleagues from Bordeaux[295] identified characteristics associated with complete LV function normalization (LV ejection fraction ≥50%) after CRT. These patients named, "super-responders," had less ischemic cardiomyopathy, lower LV and LA dimensions, and greater global longitudinal strain.

Thambo and colleagues from Bordeaux[296] evaluated the effect of CRT on 8 patients with TOF and on a TOF pig model (n = 15). They found that CRT improved significantly LV and RV function assessed electrically, echocardiographically, and invasively, by alleviating electromechanical dyssynchrony.

Bordachar and colleagues from Bordeaux[297] evaluated the hemodynamic effects of BiV pacing and triventricular pacing including an endocardial LV lead on an animal dog model (n = 8) with LBBB and LV dysfunction. They found that BiV and triventricular pacing were comparable to improve hemodynamic cardiac function. However, LV endocardial pacing was superior to epicardial pacing to improve LV function.

Marijon from Georges-Pompidou Hospital and other French centers[298] compared the mortality of 1705 patients implanted with CRT defibrillator vs CRT pacemaker. They found that the total mortality was double in patients implanted with CRT pacemaker compared with CRT defibrillator, and 95% of this mortality was related to nonsudden death, suggesting that CRT pacemaker patients, as currently selected in routine clinical practice, would not potentially benefit from the addition of a defibrillator.

Takahashi from Créteil and other French centers[299] evaluated the impact of pacing mode in patients (n = 269)

implanted with pacemaker after TAVR, as around 50% of these patients are supposed to have a recovery of AV conduction at midterm follow-up after implantation. They found that the DDD programming was associated with a worse outcome. Thus, the pacemaker mode should be systematically set to promote spontaneous AV conduction in patients with pacemaker implantation after TAVR.

Bordachar and colleagues from Bordeaux[300] found that the addition of a second LV lead in patients (n = 84) with CRT nonresponse was associated with a significant rate of severe adverse events and did not provide significant long-term clinical benefits at 24 months' follow-up.

Donal and colleagues from Rennes[301] evaluated retrospectively 154 patients with CRT. They found CRT response in 71% of cases at 6 months follow-up. They applied machine learning with ECG and transthoracic echocardiogram parameters to predict CRT response. With the Monte Carlo cross-validation method with 30 runs, using in each run different random sets of 70% of patients for training and 30% for testing, the area under the receiver-operating characteristic curve was 0.80 to predict CRT.

Fischer from Dijon and other French centers[302] found that the CRT rate response was divided by 2 in patients with cardiac amyloidosis compared with patients without cardiac amyloidosis, implanted according to international guidelines. However, patients with cardiac amyloidosis and CRT response had significant better clinical cardiac outcome.

In a prospective registry including 227 patients, Moulin and colleagues from Créteil[303] found that systematic echocardiographic optimization after CRT was not associated with better CRT response. However, CRT patients benefited from this consultation that permitted the optimization of global patients' heart failure care.

## Cardiac pacing

Daubert and colleagues from Rennes[304] evaluated in 10 healthy patients the modification of AV interval according to heart rate at rest and during exercise. They showed that AV variation is occurring instantly after heart rate variation and is inversely proportioned to heart rate increase. These modifications should be applied during AV pacing.

Ritter and colleagues from Rennes[305] evaluated the effect of 3 different AV delays (AVDs) (fixed 200 ms, fixed 150 ms, and rate adapted) on cardiac hemodynamics during exercise with DDD mode pacing in 10 patients. They showed that a rate-adapted AVD (shortening) was associated with better cardiac hemodynamic performance during exercise.

Lascault and colleagues from Ivry[306] found that in patients (n = 23) with implanted DDD pacemaker, the DDD mode was associated with a mean increase of 27% of ventricular stroke volume measured with transthoracic echocardiogram compared with VVI mode. They also showed that AVD optimization increased the stroke volume by a mean of 23.7%.

Eugene and colleagues from Pitié-Salpêtrière[307] evaluated the modification of thoracic impedance measured with an external plethysmography technique in patients implanted with a DDD pacemaker according to pacing mode (DDD vs VVI) and AVD optimization with different pacing heart rates. They also measured the cardiac stroke volume using a $CO_2$ rebreathing method. They found that the cardiac output was higher in DDD mode with AVD optimization compared with VVI pacing. They also showed thoracic impedance modification according to AVD modification for hemodynamic optimization.

Cazeau and colleagues from Rennes[308] studied the ventriculoatrial conduction at baseline and during exercise in 17 patients. They showed that 1:1 ventriculoatrial conduction was observed in 41% of cases at rest and in 76% at the end of exercise. Ventriculoatrial conduction had nodal behavior but was not correlated with AV node conduction. Ventriculoatrial conduction could have an impact in patients implanted with DDD pacemaker and PRAPV programming.

Girodo and colleagues from Montrouge[309] described the preliminary results of a DDD mode programming alternating with AAI mode in patients with paroxysmal AV block. This mode of programming could reduce unnecessary RV pacing that could be potentially deleterious for cardiac function.

Lascault and colleagues from Ivry[310] compared cardiac hemodynamics echocardiographically at baseline and during exercise in DDD mode and VVT mode. They found that DDD mode was associated with better cardiac hemodynamics compared with VVT mode. They also showed that the hemodynamic superiority of DDD was not due to a higher level of contractility but to a higher level of preload secondary to the preserved atrial systole during exercise.

Garrigue and colleagues from Bordeaux[311] postulated that high-rate atrial pacing could prevent AAs. They compared AA occurrence in patients with heart rate programming at 55 beats/min vs a heart rate 10 beats/min higher than the mean heart rate of patients measured during 24 hours before the programming. They found that the group with higher heart rate had significantly less AA compared with heart rate programmed at 55 beats/min.

Garrigue and colleagues from Bordeaux[312] evaluated randomly the effect of atrial pacing in 15 patients with central or obstructive sleep apnea who had received permanent atrial-synchronous ventricular pacemakers for symptomatic sinus bradycardia. They found that the number of hypopnea index was significantly reduced during atrial pacing phase compared with spontaneous cardiac rhythm phase.

Thambo and colleagues from Bordeaux[313] found that chronic RV pacing was deleterious for cardiac hemodynamic–assessed echocardiographically in 23 patients with complete congenital AVB over a follow-up period of 10 years.

Klug from Lille and other French centers[314] prospectively studied clinical risk factors of cardiac implantable electronic device infection in a large patient population. Among 5866 cardiac implantable electronic device implantations, device-related infections were reported in 42 (0.68%) patients. The occurrence of infection was positively correlated with fever within 24 hours before the implantation procedure, the use of temporary pacing before the implantation procedure, and early reinterventions. Implantation of a new system and

antibiotic prophylaxis were negatively correlated with risk of infection.

Laborderie and colleagues from Bordeaux[315] described the management of subacute and delayed RV perforation with a pacing or an ICD lead. On 11 patients presenting this complication, 10 were treated with simple lead traction under fluoroscopic guidance in the operating room, without need for tamponade drainage. One patient had tamponade requiring percutaneous pericardiocentesis and urgent surgical revision.

Wahbi and colleagues from Pitié-Salpêtrière[316] evaluated specifically the impact of EPS in patients with myotonic dystrophy type 1 and a PR interval >200 ms, a QRS duration greater than 100 ms, or both. Patients undergoing EPS compared with patients without EPS had a higher rate of 9-year survival. This could be due to higher rate of pacemaker implantation in this specific population.

Mouillet and colleagues from Créteil[317] found that the pacemaker implantation rate after TAVR due to high-degree AVB was 26%. Post-TAVR QRS duration was the only independent predictor of post-TAVR permanent pacemaker implantation. All patients with post-TAVR QRS ≤128 ms were free of high-grade AVB.

Pospiech and colleagues[318] from Bordeaux described a new technique for epicardial defibrillator implantation in 13 patients with congenital heart disease. They used an epicardial lead pacing sewn on the RV and 2 defibrillator coils (1 on the RV and 1 on the LV) placed without sutures. Median follow-up was 13 months. All patients underwent surgery without acute complication. One patient needed a second surgery because of defibrillation coils fracture. Neither infectious complication nor inappropriate shock was noted.

Takahashi from Créteil and other French centers[319] evaluated the impact of early temporary external pacing removal after TAVR according to post-TAVR QRS duration (<120 ms). They showed no patients with post-TAVR QRS <120 ms necessitating pacemaker implantation. They had lower intensive care unit stay length, 30-day mortality, and incidence of complications during hospitalization.

Significant sleep apnea is associated with numerous cardiac diseases and is frequently underdiagnosed. Defaye and colleagues from Grenoble[320] evaluated the correlation of sleep apnea diagnosis through an ICD using transthoracic impedance modification and polysomnography (the gold standard). They showed on 25 patients a significant correlation between these 2 methods. Particularly, an optimal cutoff value for the apnea-hypopnea index at 30 events/h yielded a sensitivity of 100% and a specificity of 80%.

Mechulan and colleagues from Marseille[321] implanted 20 patients with the Micra AV leadless pacemaker (Medtronic, Minneapolis, MN) following TAVR when needed. No procedural complications occurred during implantation. At 1-month follow-up, 2 patients displayed atrial under-sensing.

Mirolo and colleagues from Rouen and Montivilliers[322] studied electrical and hemodynamic modification following left bundle branch (LBB) pacing on 134 patients with pacemaker indication without CRT indication. In narrow QRS population, no significant difference was reported regarding the interventricular and intra LV synchrony during LBB pacing as compared with baseline rhythm. Electrical and hemodynamic improvement was similar in patients with LBBB and RBBB at baseline. It was concluded that LBB pacing in routine practice preserved intra-ventricular mechanical synchrony in patients with narrow and RBBB QRS and improved asynchrony parameters in patients with LBBB.

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