Clinical Experience with Fulguration and Antiarrhythmic Therapy for the Treatment of Ventricular Tachycardia*

Long-term Follow-up of 43 Patients

Guy Fontaine, M.D.; J. L. Tonet, M.D.; R. Frank, M.D.; and I. Rougier, M.D.

Forty-three patients (mean age, 45 ± 18 years) with drug-refractory VT of varied etiologies, including 15 cases occurring after chronic myocardial infarction, underwent fulguration procedures. With a mean follow-up of 29 ± 12 months (range, 9 to 55 months), after one to four sessions, VT had been controlled without a need for antiarrhythmic drugs in 22 (56 percent) of the 39 patients surviving the perioperative period and was controlled in 17 patients (44 percent) with the help of drugs. No malignant arrhythmias were observed following fulguration. There were five early deaths, four deaths related to the procedure, and eight late deaths, but no death was thought to be related to the endocardial shock itself. Thus, fulguration appears to be a valuable adjunct to the treatment of drug-resistant VT.

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Fulguration (electrode catheter ablation) is a method used for the radical treatment of VT. Its aim is to alter permanently the arrhythmogenic substrate, preventing arrhythmic relapses. We consider fulguration an aggressive form of therapy. This approach is used when none of the palliative methods of treatment, including drugs, antitachycardiac pacemakers, the cardioverter, and the implantable defibrillator, are appropriate. At the beginning of the surgical approach to VT, we observed that a simple ventriculotomy proved successful in some patients. The concept suggested by this approach was that a surgical procedure directed by epicardial mapping could modify enough myocardium to prevent relapse of life-threatening VT; however, the main limitation of surgery is its restriction to patients with sufficient cardiac function to enable them to withstand the procedure.

Therefore, we investigated other methods to modify conduction in a limited area of myocardium. These techniques, which use physical agents, have been referred to as “ablative techniques.” Many physical agents were considered: cryosurgery, radio frequency waves, microwaves, laser irradiation, and high-energy ultrasound.

Endocardial catheter fulguration uses a strong electrical shock delivered at the tip of an endocardial catheter positioned in the area to be modified. It has also been used to interrupt AV conduction in order to treat supraventricular tachycardia refractory to medical therapy. The same electrical energy can be applied directly to the site of origin of abnormal ventricular activation, as determined by endocardial mapping, for the treatment of chronic VT. This technique was first used in the treatment of VT by Hartzler in 1982 and by Puech et al in a case of arrhythmogenic right ventricular dysplasia. We have evaluated this new form of therapy in 43 cases.

Clinical Series

We report our experience with our first 43 fulgurated cases, with a follow-up period extending from 9 to 35 months, starting in May 1983. This report updates our previously published experience (26 cases; and 31 cases).

There were 35 men and eight women, with an age range of 14 to 74 years (mean age, 45 ± 18 years [± SD]). Thirteen patients had arrhythmogenic right ventricular disease, 15 patients had VT after an old myocardial infarction (range: minimum, three months; maximum, ten years), seven had VT complicating idiopathic dilated cardiomyopathy, and there were eight patients with tachycardia of miscellaneous causes, including four patients with RBBB-left axis VT, two patients with infundibular idiopathic VT, one case of VT occurring seven years after infundibular resection for a congenital anomaly, and one case of myocarditis sequelae. The main clinical features are summarized in Table 1.

This group was selected from 127 consecutive cases which were the total patients with major ventricular arrhythmias observed at Jean Rostand Hospital during the same period. The series consists of six patients who presented with VF and 131 with chronic recurrent sustained VT. Almost all of these patients were referred from other institutions where their condition was considered refractory to antiarrhythmic drug therapy. All of these patients were restudied, and only those patients whose condition was resistant to antiarrhythmic drugs, including amiodarone alone or in combination with class I antiarrhythmic drugs or β-adrenergic blocking agents (or both), were considered candidates for fulguration. The cases are consecutive, and there was no exclusion due to age, cardiac or

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Table 1—Clinical Features of Patients*

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*RV: Right ventricular; FC: functional class (New York Heart Association); EF: ejection fraction (echocardiography, angiography, scintigraphy); TL: time interval since first attack of VT; NM: number of clinical morphologies of VT; NE: number of VT episodes prior to facilitation; Inc: incessant VT in electrophysiologic laboratory; NH: number of facilitation sessions.
†Loc: Location of abnormality: Diaph, diaphragmatic; Infus, infundibulum; LV, left ventricle; FW, free wall, Sept, septum; RV, right ventricle; Ant-sep, anteroseptal; Inf, inferior; Post-p, posterior and posterior; Post-sep, posterosseptal.
‡D and W, longest and shortest intervals between two episodes of VT; M, month; Y, year; W, week; D, day; I, incessant.
§Jjules delivered, i.e., 100j = five discharges of 160j (value concerning last procedure).
| RIF† reasons for interrupting procedure: R, changes in rate; M, changes in morphology; NI, VT no inducible; TL, time limit; NP, programmed pacing not performed; and NC, nonclinical VT.
| AR†, antiaarrhythmic prescription upon discharge from hospital: Amio, amiodarone; A + P, amiodarone and propafenone; A + FL, amiodarone and flecainide; Bd, β-adrenergic blockers; QD, quinidine; A + B, amiodarone and β-adrenergic blockers; A + PM, amiodarone and pimavasir; Fle, flecainide; and Dilt, diltiazem.
| **MT** Mode of treatment: Proc, prophylactic treatment; and Th, therapeutic treatment.
| †+Proactive test performed ten days after facilitation. NP, Proactive test not performed; RM, change in rate and morphology of VT. NI, VT not inducible; and In, inducible by programmed stimulation.
| ‡Fold, follow-up: DC, death, D, days, and M, months.

Fulgeration and Antiarrhythmic Therapy for Ventricular Tachycardia (Fontaine et al)
clinical condition, or other factors.

We did not use therapy other than fulguration alone or in association with drug treatment, except in one patient who had cardiac surgery.

**Materials and Methods**

A detailed description of the technique developed in our department has been reported elsewhere. 

Prior to the procedure, therapy with class I antiarrhythmic drugs is withheld for a period equivalent to five half-lives. About 50 percent (22/43) of the patients were taking amiodarone, and therapy with this drug was not discontinued. Prolonged general anesthesia is used because of the duration of the procedure and the frequent need to deliver more than one shock in a single procedure.

**Endocardial Mapping**

Programmed stimulation is used to induce VT in patients who do not have incessant VT. The stimulation protocol includes the introduction of one to three progressively more premature stimuli after pacing at a progressively increasing rate. In some cases, isoproterenol is injected to facilitate VT induction. Induction of VT was not possible during the procedure in only two cases. When unstable VT or VF is induced during the pacing protocol, the session is interrupted or postponed, and drugs are prescribed to slow the rate of the arrhythmia.

Endocardial mapping is used to localize the presumed area of the origin of the VT. Confirmation is sought by trying to reproduce the morphology of the VT with ventricular pacing during sinus rhythm or VT. Three methods of ventricular pacemapping are used: (1) continuous pacing in sinus rhythm at a rate identical to the tachycardic rate; (2) slight overdrive of VT; and (3) introduction of a premature stimulus during VT (Fig 1). Comparison of QRS morphologies is performed using the portable 12-lead recorder. In the most recent studies, special attention was paid to identification of the area of slow conduction and investigation of its role as a necessary link of VT. An interesting marker could be the recording of perfect reproduction of VT morphology after either pacing in sinus rhythm or VT, provided that ventricular activation is obtained after a delay which is similar to the interval between presystolic endocardial potential and onset of the ventricular QRS complex during VT.

**Fulguration**

Fulguration is delivered at the conclusion of a checklist which is followed by a countdown. The shock is provided by equipment developed specifically for this application (Fulguor ODAM, Wissembourg, France). The shock is synchronized with the surface QRS complexes during either VT or sinus rhythm. This latter method is now preferred (Fig 2).

The shock is applied between the distal electrode of the fulgurating catheter used as an anode and an indifferent electrode which functions as a cathode and is positioned in the patient's back (Fig 3). The preselected discharge energy varied from 160 to 320 joules and was equal to 3 joules/kg of body weight. This amount of energy was determined from experiments in animals that suggested it was appropriate with our equipment. From one to eight shocks are delivered during each session.

In case of AV block, ventricular pacing is performed. In case of acceleration of VT or its degradation to VF, a defibrillating external shock is immediately delivered through the anterior patch electrode. Within a few minutes after fulguration, the pacing threshold returns to a level compatible with high-level impulses, provided that the catheter has not moved. After completion of the fulguration shock, it is possible to record flattening of the endocardial potential provided stability of the fulgurating electrode is maintained.

After a ten-minute rest period to permit electrical and hemodynamic stabilization, programmed stimulation is resumed.

The main end points of the session are as follows: (1) failure to induce a stable monomorphic VT by a programmed pacing protocol equivalent to or more aggressive than that employed for initiating

**Figure 1.** Advance in waveform recording and processing during fulguration procedure. Use of oscilloscope (Tektronix 7854) with waveform calculator provides recording of both voltage and current curves. In addition, power and impedance curves are calculated from digitized previous waveforms and displayed on screen. Use of specially identified cursor allows precise measurements of all of these parameters. Voltage curve is preceded by artifact indicating voltage charging of capacitor (4 kV), peak voltage on cardiac lead which is reduced to 1.9 kV. In this shock, peak current reaches 52.56 amps, and delivered amount is 250.7 joules. Impedance curve which is also preceded by artifact shows impedance of 39 Ω in middle of pulse (X), and 275 Ω (Y) just before collapse of cavitation bubble, and finally gets back to 98 Ω (Z). Low value of 39 Ω is related to surface of electrode which has been increased by plasma formed within cavitation bubble (see also Fig 5).

**Figure 2.** Pagemapping during VT. Premature stimulus (Sr) is followed by isoelectric line before activating ventricle with QRS configuration identical to spontaneous VT QRS complexes, as studied in leads 1, 2, 3, aVL, aVF, V₁, and V₆. This suggests that catheter is located on zone of slow conduction or zone connected to zone of slow conduction with same exit site as spontaneous VT. This zone could be appropriate for fulguration provided that it is necessary link for VT perpetuation. RA, Right atrium; and CS, coronary sinus.
the VT required for mapping; (2) spontaneous interruption in less than one minute of a previously sustained VT; (3) induction of repeated episodes of acceleration of VT or VF after fulguration; (4) repeated induction of VT leading to hemodynamic deterioration; or (5) time limitation due to technical considerations (procedure lasting more than eight hours or eight shocks).

Postoperative Surveillance

The radial arterial and venous blood pressures are monitored for 24 hours. A left subclavicular catheter is left at the apex of the right ventricle in order to permit reassessment of bedside VT reinduction, which is done at or within 10 days after fulguration, provided that no recurrence has occurred spontaneously. This reassessment is done using a programmed pacing protocol incorporating up to three extrastimuli on basic pacing cycles of 600 to 400 ms.

Electrocardiographic monitoring is done by computer during the ten-day interval, either by cable or telemetry (Hewlett-Packard 78225 system associated with the NADIA software). All alarm signals are recorded. Graphs indicating trends in cardiac rhythm, extrasystolic frequency, tachycardia, and so forth can be printed out, and the data can be corrected when necessary by using the "recall" function.

When VT comparable to previous attacks either occurs spontaneously or is inducible, antiarrhythmic drug therapy is attempted again. If the latter proves ineffective, fulguration therapy is reconsidered. Amiodarone is generally continued prophylactically (50 percent [22] of the cases; dosage ≤400 mg/day) in cases where the previous attacks of VT were life-threatening. This is called "prophylactic" antiarrhythmic treatment (Table 1). This category also includes patients who are receiving amiodarone for treatment of extrasystoles. When drugs are necessary to prevent spontaneous or programmed pacing-induced VT, the treatment is called "therapeutic" antiarrhythmic treatment. Table 1 lists the antiarrhythmic drugs administered, whether for therapeutic or prophylactic indications.

Effectiveness of fulguration is reassessed after a patient's discharge by use of 24-hour Holter recording, stress testing on a stationary bicycle, and programmed stimulation.

Follow-Up

The follow-up of this series is based on the general computer data bank of our department. A specialized application program has been developed to facilitate the follow-up. Information on patients given by the patient's physician, cardiologist, or family member is permanently updated in the computer system. Direct phone calls to the patient's home or a family member have proven to be the most effective form of follow-up (in case of an absence of information).

No patient has been lost to follow-up, despite the fact that one third of our patients were referred from other countries. Follow-up time was computed from the difference between the last fulguration procedure and the current date. Each death was investigated in order to determine if it met the definition of "sudden death," which is defined as unexpected death occurring within one hour after a new symptom.

RESULTS

The follow-up periods for the 30 survivors range from 9 (case 13) to 55 months (case 14) (mean, 29 ± 12 months). The follow-up of nonsurvivors is calculated from the first fulguration date to the date of death, excluding early deaths. It extends from 1 to 22 months (mean, 9 ± 7 months).

Description of Success Rate

In view of the fact that five (cases 5, 14, 16, 23, and 25) of the candidates for fulguration therapy were moribund, and two (cases 5 and 16) were already unconscious, the results were surprisingly favorable. Five early deaths (within one month of procedure) occurred (cases 1, 5, 10, 19, and 35); however, none of the deaths seemed to be related to arrhythmia or perforation or to have occurred as a direct result of the fulguration itself. For reasons explained later, we chose to assess the success rate at three months after discharge from the hospital.

Of the 43 patients on whom a first fulguration procedure was performed, two (cases 19 and 35) died within a few days following the procedure (Fig 4). The follow-up group is therefore limited to 41 cases. During reevaluation of the rhythm disorder after the first fulguration procedure, including the stay in the hospital and the follow-up period of up to three months, no VT relapse was observed in 13 patients. Therefore a single session without the need for drug therapy was able to prevent arrhythmia in 13 out of 41 survivors or 32 percent (in this percentage, we have included as a success of VT fulguration patient 5, who died after
A third fulguration procedure was attempted, but in no case was the third fulguration procedure effective by itself; however, drug therapy was effective in three patients. Therefore, 37 out of the 39 patients are considered effectively treated, a success rate of 95 percent. Only two patients' conditions were not controlled. A fourth fulguration attempt was performed in these two last cases; however, antiarrhythmic treatment was necessary in both to achieve complete in-hospital prevention of VT. We then reach a success rate for VT of 100 percent; however, recurrences of VT after discharge, although better tolerated and less frequent, were observed in three patients (cases 6, 9, and 18). These cases were originally considered as fulguration failures; however, after three months, these three patients experienced no further episodes of VT despite progressive reduction of their antiarrhythmic treatment, with one now being classified as prophylactic (case 18).

In summary, of 39 patients surviving the periope- rative period, single or multiple VT was brought under control in all of them by means of one or more fulguration sessions, with 17 (44 percent) requiring the help of therapeutic antiarrhythmic treatment following the fulguration therapy.

Mortality

During the overall period of this study, which extended up to 55 months, 13 deaths were observed, none of which were attributable to fulguration itself.

Early Death. Five deaths were early (less than one month after the procedure).

Cardiac Deaths. Two technically related deaths, leading to low cardiac output, occurred during the procedure. The first of these (case 1) was a patient with arrhythmogenic right ventricular dysplasia, who had undergone surgery seven years previously but had had recent recurrent episodes of life-threatening VT. Death was probably the consequence of a lack of hemodynamic monitoring during the procedure. This occurred at the beginning of our experience. The second patient (case 10) died from an irreversible low cardiac output associated with a progressive decline of myocardial contractility.

Noncardiac deaths. The first patient (case 5), who was referred after multiple episodes of VT following angiography, was unconscious and in a state of incessant VT upon arrival. A series of low-energy (180-joule) shocks led to a reduction in the rate of VT, which stopped spontaneously a few hours later without the need for antiarrhythmic therapy. The patient nevertheless died eight days later from refractory hypoxemia due to preexisting extensive pulmonary infection. The VT did not recur.

A second patient (case 19) died with a low ejection fraction following an old myocardial infarction. He had
been rejected as a candidate for surgical treatment. Delay in the resuscitation procedure during fulguration resulted in irreversible brain damage and death four days after fulguration.

The third patient (case 35) had poor cardiac function due to idiopathic dilated cardiomyopathy and was in permanent VT for two years. The patient died one day after the procedure due to septic shock complicated by hyperkalemia. Autopsy was not performed.

Anatomic and histologic examinations were performed in cases 1, 5, and 10. The examinations revealed myocardial damage which was similar to histologic lesions found in experimental animals following the application of endocavitary shock therapy.

Late Mortality. Eight late deaths were observed.

Cardiac Death. Three cases (cases 2, 24, and 29) met the criteria for sudden death at 4, 14, and 22 months after the fulguration procedure. These deaths were considered the consequence of VT relapses and will be presented in detail later.

Congestive heart failure caused death in three cases. Patient 20 died one month following discharge from acute pulmonary edema without recurrence of VT. The patient was not a surgical candidate, since he had severe triple-vessel coronary artery disease with poor distal vessels, a low ejection fraction, and left-sided ventricular failure. Case 30 was diagnosed as idiopathic dilated cardiomyopathy; death resulted from pulmonary edema three months following fulguration, with no recurrence of major rhythm disturbance. Patient 23 died of cardiac failure ten months after the fulguration procedure while receiving therapeutic antiarrhythmic drugs.

Noncardiac Death. Two patients (cases 11 and 31) died of noncardiac causes (suicide and cancer). All of the preceding late deaths occurred outside of the hospital, and autopsy was not possible.

Complications

Acute pulmonary edema was observed in three cases (cases 13, 16, and 28) during the first ten minutes following fulguration but resolved after treatment with standard therapy.

In one case (case 6), the patient experienced chest pain associated with modification of the ST segment and transient RBBB during the second fulguration session. The rise in the level of CPK and, particularly, the CPK-MB fraction was 140 IU, greater than that observed in the other patients treated (37 ± 15 IU).

Transient complete AV block was frequently observed (in 17 percent of 167 shocks in the course of the procedure), and occurred immediately after the shocks. It persisted after the session in only two cases (cases 14 and 16) and resolved in a maximum of two hours (case 16). Intraventricular conduction anomalies of short duration were also noted (8 percent LBBB and 5 percent RBBB for 167 shocks).

Ventricular tachycardia acceleration and VF were each observed in 15 percent of the shocks immediately following the initial electrical discharge. They were easily reverted or defibrillated, except in two patients (cases 19 and 35), one of whom was in acute hemodynamic cardiac failure (case 19) and the other of whom was hypoxic; however, no malignant arrhythmia resistant to defibrillation was observed following fulguration.

One patient with a pacemaker (case 20), in whom the fulguration shocks were delivered close to the ventricular pacing electrode, needed generator replacement.

Long-Term Relapse of VT

These relapses should be classified into the following categories:

Relapses Better Tolerated. This situation was observed in four patients (cases 22, 24, 29 and 41), of whom one (case 24) was asymptomatic. Patient 29 died suddenly 14 months after fulguration, when he had an exacerbation of heart failure, due to the terminal stage of an idiopathic dilated cardiomyopathy. Patient 24 had two forms of sustained VT elicited during programmed pacing. A fulguration procedure was performed and seemed to be effective for one form. The nonfulgurated VT was still inducible at the time of discharge, but the attacks were slower and better tolerated. Sudden death occurred four months after the procedure, and it is unknown if this was due to recurrence of the fulgurated VT or to degradation of the nonfulgurated VT or to VF. Patient 22 was in incessant VT when the fulguration procedure was performed. Relapse was observed a few hours later, and at that time, VT could be terminated by pacing. It was reproducibly demonstrated that a class 1c antiarrhythmic drug was then able to prevent VT. This drug had not been effective prior to the procedure. One year later, the patient was reevaluated by programmed pacing one week after discontinuation of the class 1c drug, and VT was found to be noninducible. The patient was receiving amiodarone only. Holter monitoring exhibited nonsymptomatic VT at 110 beats per minute. Class 1c drug therapy was resumed without reinvestigation at Jean Rostand Hospital. The patient suffered sudden death four months later. Patient 41 experienced relapses after six months at the same rate and morphology; however, these attacks were less frequent, and for personal reasons the patient did not want to undergo a new attempt.

Relapses Controlled by Drugs. This was obtained in three cases; in one (case 34), a modification in drug therapy led to the control of the arrhythmia. This patient had a first relapse 12 months after fulguration.
during an attempt at reduction of the two drugs given as therapeutic treatment. Two more episodes were later observed after resumption of the original therapy; finally, replacement of class 1c antiarrhythmic therapy by a β-adrenergic blocking agent continues to appear to be preventing relapses.

In the second case (case 27), amiodarone therapy used alone at a dosage of 200 mg/day controlled the arrhythmia. After nine months, therapy with the drug was interrupted. The patient experienced relapse one month later. After a short hospitalization, therapy with amiodarone was resumed and controlled the arrhythmia.

The third patient (case 6) had a relapse three years after the third fulguration procedure, after antiarrhythmic therapy had been discontinued. This patient originally had VTs of five morphologies; two of them were better tolerated and were not ablated. The morphology of the relapses was identical to one of the nonfulgurated VTs. Drug treatment was reinstituted. Some other relapses were again observed; however, a new fulguration was not considered appropriate.

*Relapses Controlled by Refulguration.* This approach was used in five cases, with success in the second procedure, although in case 12, some additional episodes were observed and finally disappeared. This behavior suggests the previously mentioned evolution of some of our early relapses. Patient 39 had relapses identical to previous episodes after four months and was refulgurated successfully. Patient 31 experienced frequent relapses of the same VT morphology five months after the first session. A new fulguration procedure performed in the same area proved successful during a follow-up of nine months. Patient 34 experienced a syncopal relapse of VT 14 months after the fulguration. He denied this event for ten months for professional reasons. Later, relapses became more frequent, and this patient has undergone a second fulguration procedure. Nineteen months of follow-up indicate that the patient’s condition was controlled by the second attempt.

A relapse after 31 months was observed with patient 9. A new fulguration procedure controlled the arrhythmia with a follow-up of four months; however, five short episodes of nonsustained VT were observed transiently after two months. Amiodarone therapy was prescribed and seems successful.

**Discussion**

Our results confirm the initial favorable results reported by Hartzler et al 15,16 in patients with coronary artery disease and by Puech et al 16 in a case of arrhythmogenic right ventricular dysplasia. These authors reported the first cases of fulguration in the treatment of VT, using a technique derived from the closed-chest His bundle fulguration for the indirect treatment of supraventricular tachycardia 14,16-35

Other investigators have reported less successful results in the management of VT with this technique. 36-43 In view of the very poor cardiac condition of most of our patients, we do not think that differences in the populations explain why we observed better results. The favorable results may be due to a difference in the selection of the equipment and methods. During the fulguration of our second patient (case 14), we observed that the vast majority of the regular catheters (USCI) were not able to withstand the high peak of current and voltage necessary to obtain a sufficient effect on the endocardium. As no alternative catheters were available, we developed a technique for selection of tripolar or quadripolar catheters (USCI) by a nondestructive high-voltage test (Fig 5). 44-45 As a result, we were able to demonstrate that a successful outcome could be obtained in some cases using a

![Figure 5. Complex behavior of quadripolar catheter (USCI) during fulguration procedure. Before session, catheter has been tested (upper panel) according to technique previously published. 44-45 (Fontaine G, Cansell A, Lampe L, Baraka M, Tomet JL, Frank B, Grosgeote Y. Endocarditary fulguration [electrode catheter ablation 2 equipment related problems. In: Fontaine G, Scheinman MM, eds. Ablation in cardiac arrhythmias. Mt Kisco, NY: Futura, 1987; 85-100). Nevertheless, shock recorded during clinical session shows notches on voltage curve which indicate partial internal arcing, indicating that something wrong appeared during procedure (oblique arrow, middle panel). Same catheter retested in laboratory demonstrates abnormal behavior (lower panel). This indicates that previously properly selected catheter can loosen its insulation strength during procedure. V, voltage waveform; LC, leakage current; and C, current waveform.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21592/ on 06/19/2017)
relatively low fulgurating energy delivered in a single shock.46

A second difference is based on the fact that we originally decided to use the active electrode as an anode instead of a cathode because in vitro studies of the effect of shocks demonstrated that anodal shocks provided a stronger mechanical effect. We also observed with electron microscopy the rupture of myofibres at a distance of 1 cm from the fulguration site. Since it is well known that the stretch of myocardial fibers modifies cardiac conduction,47 we chose to incorporate this parameter into our protocol. Recently, we have observed that the variation in wave forms generated by different types of defibrillators should also be taken into account. Limited in vitro experiments have shown that anodal shocks with fast rise times lead to stronger mechanical effects and tissue damage.

These three points (catheter selection, anodal shocks, and shock impulse wave form) therefore make difficult any comparison between our results and those of others concerning the results obtained in animal experiments or in the clinical field.

Electrophysiology

Morphologies of VT. The induction of VT by programmed pacing may lead to varied results. It is now well accepted that some forms of nonsustained or polymorphic VT induced by programmed pacing may not have clinical significance.48 Therefore, only sustained monomorphic VT has been considered; however, programmed pacing can also induce episodes of types of VT not previously documented, the so-called "nonclinical" VTs. Since the class 1 antiarrhythmic treatments were interrupted several days before the fulguration procedure, it is unlikely that they would alter the morphology of VT induced during the fulguration procedure.49 We have learned that both clinical and nonclinical VT should be considered for fulguration treatment.50,51 In general, a correlation was observed between the number of VT morphologies and the number of sessions (or the number of shocks per session). On the other hand, we have also observed cases in whom a single shock was able to ablate more than one VT morphology.

In some cases, so-called resistant and almost incessant VT was not the result of lack of efficacy of drug therapy but rather due to the proarrhythmic effect of the drugs. In one patient studied with the arrhythmia monitoring computer system, a direct relationship was observed between the dose of a class 1c antiarrhythmic agent and the number of nonsustained episodes of VT or extrasystoles (or both).

Endocardial Catheter Mapping. Evaluation of the prematurity of endocavitary potential recorded by mapping as a marker for determination of the fulguration site was bleak; however, the absence of presystolic activity invariably led to failure. On the other hand, success was generally greater in cases where high-amplitude presystolic VT potential had a definite prematurity.18

Mortality

Three patients (cases 5, 14, and 16) were moribund when effective fulguration was performed. At least 11 cases (cases 5, 10, 14, 16, 19, 20, 23, 25, 29, 30, and 35) involved prohibitive surgical risks. None died of the immediate effect of the endocardial electrical shocks.

It is possible that two deaths early in the study could have been avoided, the first (case 1) by hemodynamic monitoring, and the second (case 19) was a death due to anesthetic. The anesthetic protocol was subsequently modified. The six deaths (cases 5, 10, 20, 23, 30, and 35) have been interpreted as consequences of the evolution of preexistent pathologic processes. Deaths in cases 5 and 10 were interpreted as inevitable due to the patient's precarious hemodynamic situation. In four cases (cases 20, 23, 30, and 35), clinical evidence of deterioration of myocardial function apart from the rhythm disorder had been observed during the period preceding fulguration.

The three last patients (cases 22, 24, and 29) died suddenly. In patient 24, the nonfulgurated less rapid VT was still inducible at the time of discharge; recurrences occurred but were better tolerated. In retrospect, this patient, who was not in definite cardiac failure, should have been a candidate for other therapy such as an implantable defibrillator or another fulguration procedure. Patient 22, who died suddenly, had an ejection fraction of 46 percent, and the cardiac failure observed at the time of nonsymptomatic VT was probably related more to the arrhythmia than to intrinsic cardiac contractility. In patient 29, sudden death was observed 14 months after the procedure. Despite documented episodes of VT, preterminal deterioration of cardiac function did not prompt us to attempt a repeat fulguration; again, a new fulguration procedure or the implantation of an implantable defibrillator could have been considered.

Recurrences

In some cases, recurrences are easily ascribed to technical difficulties involving the catheter,45 leading to insufficient energy output (case 14), on the one hand, and fulguration situated too far from the zone of origin of the VT on the other (cases 16 and 18). Other recurrences are less easily accounted for and allow only the formulation of hypotheses: inadequate mapping; arrhythmogenic area extending beyond the zone benefiting from fulguration, thus partially inhibiting abnormal activation; and increasing sensitivity to
antiarrhythmic drugs. The implications of a favorable spontaneous evolution of the disease remain to be evaluated. On the other hand, the fact that some patients were able to be cured after a transient period of relapses led us to postulate the possibility of delayed effects of fulguration. Several investigators have seen the late development of AV conduction impairment several weeks or months after a seemingly inadequate fulguration procedure aimed at His bundle ablation.

Experience gained during follow-up prompted us to realize the need for an implantable device both for arrhythmia detection and possible palliative treatment.

The Fulguration Process

The technique of VT fulguration is still in its early developmental phase, and many of its most basic aspects are only partially understood. This very complex physical phenomenon (Fig 6) involves different sciences: fluid dynamics; thermodynamics; electricity; electrochemistry; and biology. At the risk of oversimplification, fulguration can be reduced to two principal mechanisms: (a) a flow of electric current crossing the myocardium in the area between the active electrode and the indifferent one; and (b) the formation of at least two shock waves. The first shock wave is produced by the abrupt surge of vapor generated by the creation of ionized plasma around the active electrode tip within the liquid environment, and the second is produced when the vapor globe collapses after the end of the delivery of current. This mechanical result of the electrical discharge is propagated in all directions; however, collapse of the vapor globe against the wall of the myocardium could cause a particularly violent impact. While the effect on the myocardium has been extensively discussed with respect to external defibrillation, the same is not true for its endocavitory application. The respective parts played by both electrical and mechanical agents in the process of fulguration remain to be clarified (Fig 7).

Secondary Effects

Cardiac Output. An assessment of cardiac function was performed before and seven days after the procedure by 2D echocardiography on a limited series; as no significant changes were observed, this study was later abandoned; however, the results of hemodynamic studies demonstrate that there is a temporary drop in cardiac output after the procedure, which reverts to its control values within 10 to 15 minutes after the shocks. In addition, the small amount of CPK-MB fraction obtained after the shocks made this concern less important. This was also in agreement with some experimental data from our laboratory which indicate that following endocavitary fulguration delivered to a healthy endocardium (pig weighing 80 kg), the left ventricular cardiac output decreases by 10 to 15 percent for approximately ten minutes; however, one episode of acute pulmonary edema (case 16) in our series was probably related to the fulguration of nearly normal myocardium distant to the area of infarction.

Arrhythmias. Episodes of VT acceleration or VF brought on by fulguration were observed in 15 percent of the shocks. All of these arrhythmias were immediately successfully treated by instant defibrillation. Such episodes further demonstrate the necessity of prepositioning the defibrillating electrode in an anterior position in order to avoid emergency removal of sterile surgical fields and fluoroscopic equipment.

No malignant ventricular arrhythmia was observed after the procedure; however, extrasystoles were frequent, and new forms of sustained VT were observed, particularly in patients without coronary artery disease. These arrhythmias disappeared after a few days and did not represent the creation of a new arrhythmogenic substrate.

Myocardial Infarction. The case of myocardial infarction (case 6) observed during the fulguration procedure was the consequence of difficulty encoun-
tered during manipulation of the catheter in the root of the aorta in an attempt to reenter the ventricle after inadvertent withdrawal. Subsequent modification of the technique employed eliminated this risk.

**Effect on a Permanent Pacemaker and Implantable Defibrillator.** In one patient (case 20), a defect in the functioning of the pacemaker at the time of fulguration required replacement of the generator. This risk is higher in case of a unipolar device, and malfunctioning might not be obvious after the shock. Thorough evaluation of the pacemaker’s pacing safety margin and sensing and programming functions are mandatory after, as compared to before, the fulguration procedure.65-66

Despite the fact that no systematic study has been made, it is probable that an implantable defibrillator could be rendered completely ineffective by fulguration. Since any patient who has an implantable defibrillator could present with episodes needing a more radical approach because of repeated episodes of VT/VF, it may be necessary to temporarily disconnect the implantable defibrillator before delivering the fulgurating shocks.

**Alternative Therapy and Selection of Patients**

To put our results into perspective, we compared our results in terms of overall mortality and relapse to the original cohort of 137 consecutive patients with chronic stable VTs. This study has already been presented in part66 and was done from a review of the outcome of the nonfulgurated patients available in April 1986. Therefore, this comparison is valid vis-à-vis the results of 31 cases of fulguration in the same time period.

The overall mortality in the group of the nonfulgurated patients was 15 cases (21 percent), with six cases of sudden death (8 percent). Relapses were observed in five patients who survived and in six patients who died, yielding a relapse rate of 15 percent.

In the fulgurated population a suitable comparison should omit the patients who died because of inappropriate protocols (three cases; cases 1, 19, and 35). The global mortality was seven (20 percent) calculated from 35 survivors, and sudden death was observed in three cases (9 percent). Long-term relapses were observed in four cases (11 percent) out of 35 survivors.

Finally, despite the small number of patients in each series and the relatively short follow-up, it was possible to conclude in April 1986 that the fulguration procedure associated with drug therapy yields results comparable to those from drug treatment alone; however, as fulguration was only performed in patients resistant to drug therapy and in particularly poor condition, it is suggested that this form of treatment provides a definite adjunct in the management of ventricular arrhythmias. It is also concluded that refulguration should be considered when a previously fulgurated patient presents with relapses.

**Effectiveness of the Technique**

Fulguration appears to be a valuable adjunct to the treatment of chronic stable VT in humans.

In addition, in eight cases (cases 3, 7, 14, 16, 32, 34, 37, and 40), a single fulguration shock of an amplitude varying between 160 and 240 joules was sufficient to prevent recurrence of VT without the need for therapeutic antiarrhythmic treatment. This suggests that in some cases the injury produced by a single shock is able to modify the pathologic substrate which was the basis of a life-threatening arrhythmia. This observation confirms our previously reported data that simple ventriculotomy was able to prevent recurrence of resistant VT.4,6 However, due to the limited endocardial surface modified by the shock, we want to stress that precise endocardial mapping is probably a crucial prerequisite for the success of the procedure. From a theoretical standpoint, it should be more appropriate to deliver the fulgurating shock to the area of slow conduction, provided that this area is indeed a necessary link for the perpetuation of the arrhythmia, rather than the “site of origin” of VT, which may be located near normal myocardium. Recent data suggest that this goal could be achieved successfully at least in some cases.

Nevertheless, the facts that three patients died suddenly during the follow-up, that 22 patients (56 percent) of the 39 patients surviving the perioperative period required two or more sessions, and that 22 patients (51 percent) needed antiarrhythmic therapy suggest that fulguration is not yet an “ideal” form of therapy. Two points should be mentioned: (1) the current technique of mapping is probably not sufficiently precise; this is suggested by the need for several shock sessions and antiarrhythmic therapy in most of the cases; and (2) the amount of modified tissue is probably too small; the behavior of myocardial contractility after the shock and the small amount of the CPK-MB fraction suggest that a more aggressive physical agent could be employed.

From a practical standpoint, the present techniques require general anesthesia and a large number of personnel and are long and difficult; therefore this technique should be limited to centers dedicated to performing this procedure.

**Limitations of the Study**

We conclude that the fulguration procedure is an effective form of therapy for VT. Nevertheless, many points could be criticized. A limited number will be mentioned in the present report:

There is a bias in the studied population. Since our population consisted of patients whose condition was
resistant to antiarrhythmic drugs, it is a skewed sample of patients with VT who are highly likely to have recurrent VT after almost any therapy. The technique was used as a last resort and was restricted to the most resistant and difficult cases which were beyond surgery; some patients were even moribund when the fulguration procedure was performed.46

Our referral of patients corresponds to a particular category of patients with VT in whom other indications for cardiac surgery were not present.

The experience accumulated over years with surgical treatment of VT tends to bring to our center patients with VT related to noncoronary diseases like arrhythmogenic right ventricular disease and RBBB-left axis VT in young adults.47 In other series, VT is most commonly associated with coronary disease, especially with myocardial infarction. This could also reflect the prevalence of the basic disease, which is not the same in different countries.

Our experience with fulguration has resulted in changes both in the protocols and the equipment.

A limitation in the evaluation of the control of arrhythmia is due also to the fact that after treatment a previously symptomatic patient could become asymptomatic due to a slower rate of tachycardia, the negatively inotropic effect of antiarrhythmic treatment, or spontaneous degradation of cardiac function.

When a patient enters the electrophysiologic laboratory with incessant VT, complete disappearance of the arrhythmia is a clear-cut effect of the treatment. This situation is in fact quite rare. An algorithm to determine when VT should be considered ablated is not available at the present time; for example, in case 9, the patient had only two episodes of VT tolerated badly, with a time interval between attacks of six months. One documented relapse of VT with the same morphology occurred within three months after discharge and then disappeared. Recently, a new relapse was observed 25 months after the last episode. In the same episode, two morphologies were observed. One was similar to the previously documented episode; the other happened to exhibit a new morphology. Was this a new VT due to the evolution of an unstable myocardium (arrhythmogenic right ventricular disease) or the relapse of the fulgurated VT?

The fact that antiarrhythmic treatment is frequently needed is not in our view a sign of failure but a sign of partial effectiveness of the procedure.

The predictive value of VT reinduction during sessions is relatively unreliable. From a general standpoint, it seemed at first view that 50 percent of recurrences took place in cases where it was not possible to reinduce VT at the end of the session; however, a more thorough analysis of VT morphology demonstrated that the recurrences were mainly observed in the nonfulgurated VTs48 and suggested that in all of the patients who remained inducible a spontaneous recurrence was observed. Better results were obtained ten days following fulguration, although this indication completely failed in one case (case 18). Nor can the protocol of reevaluation on the tenth day account for modifications apt to develop over a longer period. It might be too early for long-term evaluation, since stabilization of a lesion on the myocardium generally needs a longer time. Also, the possible modifications induced inside the fibrous tissue created by the procedure may need an even longer time, which could be extended to several months before the stage of stable retractile fibrosis is reached. As usual, it is difficult to extrapolate to humans results obtained in animals. In this latter situation, shocks have generally been delivered to normal myocardium.

Measurement of the myocardial isoenzyme of the creatine kinases revealed low values (37±15 IU), confirming what we noted in experimental studies; the myocardial area altered by the endocavitary shock is limited.49 Consequently, fulguration may be performed without danger one or more times in the absence of an adequate result on the first attempt, before the procedure is determined to be ineffective. In our experience, another attempt at fulguration was preferred by the patients over its cardiovascular surgical counterpart.

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