Percutaneous Cardiopulmonary Support for Catheter Ablation of Unstable Ventricular Arrhythmias in High-Risk Patients

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Abstract

Background and Purpose: In patients with severe cardiomyopathy, recurrent episodes of nontolerated ventricular tachycardia (VT) or electrical storm (ES) frequently cause acute heart failure and cardiac death; the suppression of the arrhythmia is therefore lifesaving, but feasibility of catheter ablation (CA) is precluded by the adverse hemodynamic conditions together with the characteristics of the arrhythmia that interdicts efficacious mapping. The use of the percutaneous cardiopulmonary support (CPS) for circulatory assistance may allow patient's stabilization and enhance efficacy and safety of CA in this emergency setting.

Patients and Methods: 19 patients (19 males; mean age 61 ± 6 years; chronic ischemic cardiomyopathy, eleven patients; primary dilated cardiomyopathy, six patients; arrhythmogenic right ventricular dysplasia/cardiomyopathy, two patients) with recurrent nontolerated VT episodes undergoing CPS-assisted CA were retrospectively evaluated. Twelve patients had acute hemodynamic failure refractory to inotropic agents and ventilatory assistance, seven patients had undergone a failing nonconventional CA procedure. 14 patients presented with ES, and in twelve the procedure was undertaken under emergency conditions within 24 h from admission. Patients were ventilated under general anesthesia and assisted by a multidisciplinary team. The CPS system consisted in a Medtronic Bio-Medicus centrifugal pump and in a Maxima Plus oxygenator, a 15-F arterial cannula, and a 17-F venous cannula.

Results: Flows between 2 and 3 l/min were activated after induction of 56/62 forms of nontolerated VT, achieving hemodynamic stabilization in all patients. CA was mainly guided by conventional activation mapping and was effective in abolishing 45/56 supported VTs; in 10/19 patients all clinical VTs were suppressed by CA. Mean procedural time was 4 h and 20 min. Complete stabilization was achieved in 13 patients (68%) without VT recurrence during a 7-day in-hospital monitoring. A significant clinical improvement was observed in two patients (11%); one patient (5%) with persistent VT episodes acutely died after heart transplant. At a mean follow-up of 42 months (range 15–60 months), 5/18 patients (28%) were free from VT recurrence, 7/18 (39%) had a clear clinical improvement with reduced implantable cardioverter defibrillator interventions. 5/14 patients (36%) had ES recurrence; among them, three died because of acute heart failure. No serious CPS-related complications were observed.

Conclusion: The CPS warrants acceptable hemodynamic stabilization and efficacious mapping in high-risk patients undergoing CA for unstable VT in the emergency setting. Safety and efficacy of this technique translate into significant clinical improvement in the majority of patients. Even if only relatively invasive, CPS should be reserved to patients with ES or intractable arrhythmia causing acute heart failure; moreover, the need for an experienced team of multidisciplinary operators implies that its use is restricted to selected high-competency institutions.

Key Words:
Ventricular Tachycardia · Catheter ablation · Heart failure · Hemodynamic support

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(Perkutaner kardiopulmonaler Support für die Katheterablation instabiler ventrikulärer Arrhythmien bei Hochrisikopatienten

Zusammenfassung

Hintergrund und Fragestellung: Bei Patienten mit schwerer Kardiomyopathie verursachen rezidivierende Episoden nicht tolerierter ventrikulärer Tachykardie (VT) oder eines elektrischen Sturms (ES) häufig akutes Herzversagen und plötzlichen Herztod; die Suppression der Arrhythmie ist daher lebensrettend, jedoch stehen der Durchführbarkeit der Katheterablation (KA) ungünstige hämodynamische Verhältnisse sowie die Charakteristika der Arrhythmie, die ein effizientes Mapping verhindert, entgegen. Der Einsatz des perkutanen kardiopulmonalen Supports (KPS) zur Kreislaufunterstützung kann zur Stabilisierung des Patienten beitragen und die Effizienz

Schlüsselwörter:
Ventrikuläre Tachykardie · Katheterablation · Herzversagen · Hämodynamische Unterstützung

und Sicherheit der KA in diesem Notfallszenario erhöhen.

Patienten und Methodik: 19 Patienten (19 Männer; Durchschnittsalter 61 ± 6 Jahre; chronische ischämische Kardiomyopathie, elf Patienten; primäre dilatative Kardiomyopathie, sechs Patienten; arrhythmogene rechtsventrikuläre Dysplasie/Kardiomyopathie, zwei Patienten) mit rezidivierenden nicht tolerierten VT-Episoden, die einer KPS-unterstützten KA unterzogen wurden, wurden retrospektiv evaluiert. Zwölf Patienten hatten ein akutes, gegenüber inotropen Agenzien und assistierter Beatmung refraktäres hämodynamisches Versagen. Sieben Patienten hatten sich einem frustranen nichtkonventionellen KA-Verfahren unterzogen. 14 Patienten hatten ES, und bei zwölf wurde das Verfahren innerhalb von 24 h nach der Aufnahme unter Notfallbedingungen durchgeführt. Die Patienten wurden unter Vollnarkose beatmet und von einem multidisziplinären Team unterstützt. Das KPS-System bestand aus einer Medtronic-Bio-Medicus-Zentrifugalpumpe und einem Maxima-Plus-Oxygenator, einer arteriellen Kanüle (15 F) und einer venösen Kanüle (17 F).

Ergebnisse: Nach der Induktion von 56/62 Formen nicht tolerierter VT wurden Flussraten zwischen 2 und 3 l/min eingestellt, so dass bei allen Patienten eine hämodynamische Stabilisierung erreicht wurde. Die KA wurde hauptsächlich durch konventionelles Aktivierungsmapping geführt, und sie war effizient bei der Beseitigung von 45/56 VT mit KPS; bei 10/19 Patienten wurden alle klinischen VT durch KA supprimiert. Die mittlere Eingriffszeit betrug 4 h und 20 min. Bei 13 Patienten (68%) wurde eine völlige Stabilisierung ohne rezidivierende VT während einer 7-tägigen stationären Überwachung erreicht. Bei zwei Patienten (11%) wurde eine erhebliche klinische Verbesserung beobachtet; ein Patient (5%) mit persistierenden VT-Episoden verstarb akut nach einer Herztransplantation. Nach einem mittleren Beobachtungszeitraum von 42 Monaten (Range 15–60 Monate) waren 5/18 Patienten (28%) in Bezug auf die VT rezidivfrei und 7/18 (39%) zeigten eine deutliche klinische Verbesserung mit weniger ICD-Interventionen (implantierbarer Kardioverter-Defibrillator). 5/14 Patienten (36%) hatten ES-Rezidive; davon starben drei aufgrund von akutem Herzversagen. Es wurden keine schweren, mit dem KPS im Zusammenhang stehenden Komplikationen beobachtet.

Schlussfolgerung: Der KPS garantiert eine akzeptable hämodynamische Stabilisierung und effizientes Mapping bei Hochrisikopatienten, die sich notfallmäßig aufgrund einer instabilen VT einer KA unterziehen. Die Sicherheit und Effizienz dieser Technik führen bei der Mehrheit der Patienten zu einer erheblichen klinischen Verbesserung. Selbst wenn der KPS nur relativ invasiv ist, sollte er beschränkt sein auf Patienten mit ES oder hartnäckiger Arrhythmie, die akutes Herzversagen verursacht. Ferner impliziert die Notwendigkeit eines erfahrenen multidisziplinären Teams einen limitierten Einsatz in ausgewählten Einrichtungen mit hoher Fachkompetenz.

Introduction

Catheter ablation (CA) has been recognized as a first-line therapy for recurrent ventricular tachycardia (VT) in implantable cardioverter defibrillator (ICD) recipients, in whom a favorable impact on quality of life and survival derives from the reduction of device interventions and from the prevention of electrical storm (ES) [1–4]; as a consequence, new strategies are required aiming at a more effective treatment of hemodynamically nontolerated forms of arrhythmias.

The use of advanced methods of mapping (i.e., electroanatomic mapping, multielectrode noncontact system) has recently modified our approach for the treatment of unstable VT [5–10]; these techniques minimize the time required for mapping during VT, but are of limited value in patients presenting with severe hemodynamic impairment resulting from intractable recurrences of unstable arrhythmias.

In this setting, the use of techniques for circulatory assistance may contribute to patient's stabilization and may enhance efficacy and safety of CA in the emergency setting. The percutaneous cardiopulmonary support (CPS) has been originally described in the heart catheter laboratory for complex interventional revascularization procedures [11–14]; its use has been more recently proposed by our group as a support for emergency CA in patients with nontolerated VT [15]. The aim of our study is to retrospectively evaluate overall feasibility and clinical outcome of CPS in a selected population of high-risk patients undergoing CA for unstable VT between 1999 and 2008.

Table 1. Patients' characteristics. CA: catheter ablation; CPS: cardiopulmonary support; F: female; M: male; SD: standard deviation.

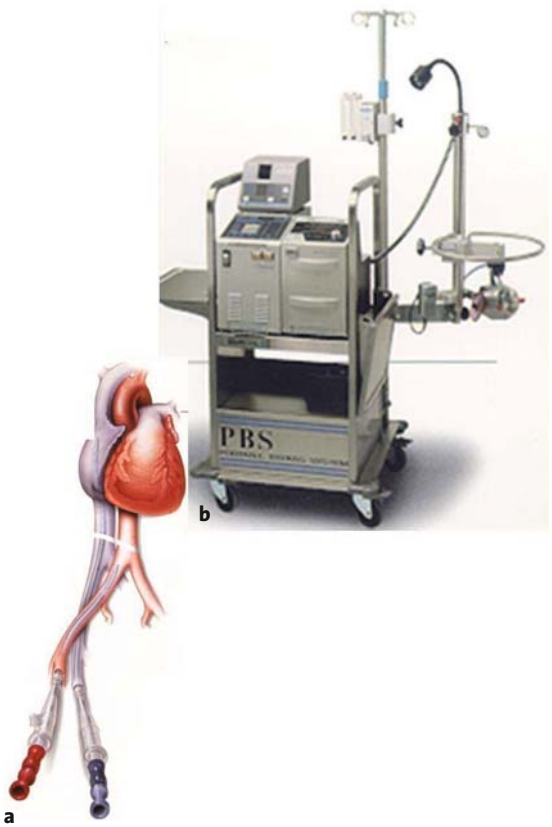
Tabelle 1. Patientenmerkmale. CA: Katheterablation; CPS: Cardiopulmonarer Support; F: weiblich; M: männlich; SD: Standardabweichung.

Age (years, mean ± SD)		61 ± 6
Gender, M/F (n)		19/–
Underlying heart disease [n (%)]	Coronary artery disease	11 (58)
	Idiopathic dilated cardiomyopathy	6 (31)
	Arrhythmogenic right ventricular dysplasia	2 (11)
Indication for CPS [n (%)]	Cardiogenic shock	12 (63)
	Failed CA with hemodynamic impairment	7 (27)

Patients and Methods

19 patients (19 males; mean age 61 ± 6 years) presenting recurrent hemodynamically nontolerated VTs refractory to antiarrhythmic drugs were enrolled in the study population: eleven patients had chronic ischemic cardiomyopathy (mean left ventricular ejection fraction 28%, range 10–40%), six had primary dilated cardiomyopathy (mean left ventricular ejection fraction 32%, range 15–45%), two had arrhythmogenic right ventricular dysplasia/cardiomyopathy (left ventricular ejection fraction > 55%); patients' characteristics are shown in Table 1. Patients represented a minority among a total of 737 patients (2.6%) with structural heart disease undergoing CA for sustained VT in the same period. The indication for the use of the CPS was defined by the evidence of recurrent nontolerated VTs (or of ES) responsible for acute hemodynamic failure/cardiogenic shock developing in the previous 12–24 h, refractory to pharmacological treatment and ventilatory assistance (twelve patients). In a minority of cases (seven patients), CPS was chosen after failure of a previous CA procedure, guided by nonconventional mapping techniques (electroanatomic mapping or multielectrode noncontact system), for the treatment of frequent episodes of nontolerated VTs, requiring prompt termination because of acute severe hemodynamic deterioration (systolic arterial pressure of 60 mmHg or less; hemodynamic collapse within 60 s) despite the infusion of inotropic agents, or because of cardiac arrest. 14 patients presented with ES and in twelve patients the procedure was undertaken under emergency conditions due to the development of cardiogenic shock following intractable VT recurrences; in these cases CA was performed within 24 h from admission at our institution. Patients with significant atherosclerotic disease (aortoiliac – femoral district), valvular aortic regurgitation, evidence of absolute contraindications to anticoagulation, were excluded.

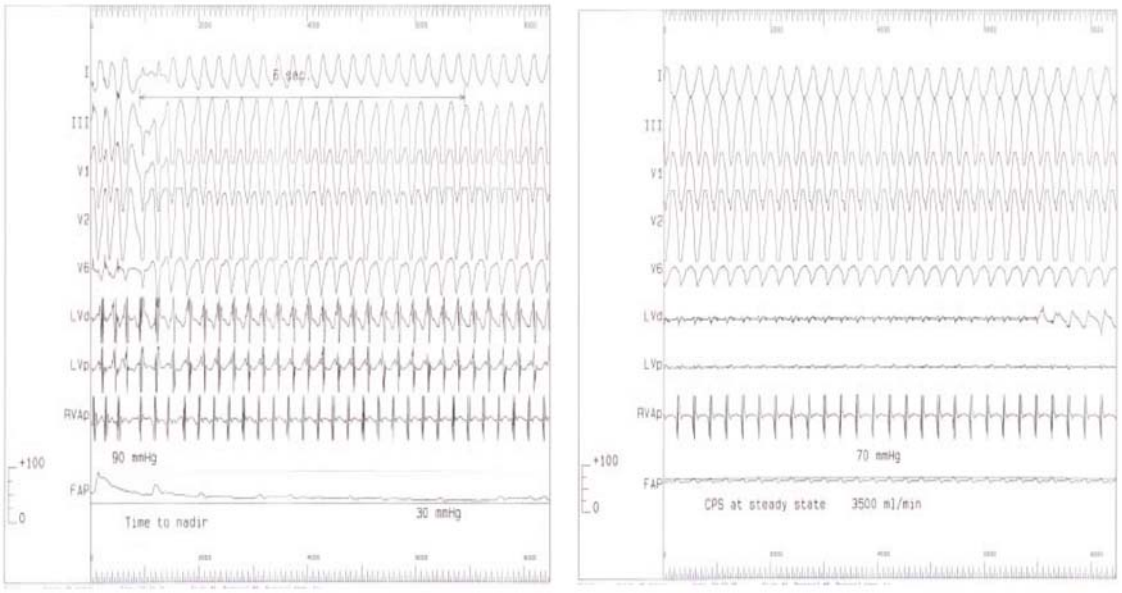
Written informed consent was obtained from all patients. All patients were fully sedated and ventilated. Patient's management was assured by a multidisciplinary team that included three cardiac electrophysiologists, one interventional cardiologist, one cardiac anesthesiologist, one perfusion technician, and two nurses. Femoral cannulation was achieved using the standard Seldinger technique with angiographic control of the cannulation site. A 15-F arterial cannula was placed in the left common femoral artery, and a 17-F venous cannula was placed in the left common femoral vein. The CPS system used a Medtronic Bio-Medicus centrifugal pump and a Medtronic Maxima Plus oxygenator (Medtronic, St Paul, MN, USA; Figure 1). This setup, using relatively small cannulae in combination with pump-driven venous return, allowed CPS flows of 1–5 l/min, ordinarily of 2–3 l/min. The priming solution of the CPS circuit was composed of Ringer's lactate (800 ml). Hemodynamic monitoring was achieved by an arterial line; systemic anticoagulation was achieved by i.v. heparin with a target activated clotting time of 300–350 s and monitored at 15-min intervals together with blood gases.



Figures 1a and 1b. CPS equipment. a) Arterial (15-F) and venous (17-F) multiple side-holed cannulas for femoral catheterization. b) Centrifugal cone-type pump (Medtronic Bio-Medicus) and membrane oxygenator coupled with a heat exchanger (Medtronic Maxima Plus). (By courtesy of Medtronic, St Paul, MN, USA.)

Abbildungen 1a und 1b. KPS-System. a) Arterielle (15 F) und venöse (17 F) Kanülen mit mehreren Seitenlöchern zur femoralen Katheterisierung. b) Konusartige Zentrifugalpumpe (Medtronic Bio Medicus) und Membranoxygenator, gekoppelt mit einem Wärmetauscher (Medtronic Maxima Plus). (Mit freundlicher Genehmigung von Medtronic, St Paul, MN, USA.)

After priming of the circuit, 1-l/min flow was maintained during the standby. When VT induction was followed by hypotension (systolic arterial pressure < 60 mmHg), CPS flow was increased targeting mean arterial pressure values of 60–70 mmHg (Figure 2). Despite acceptable systemic pressure values, each VT episode was terminated – either by overdrive or by external cardioversion/defibrillation – after a maximum period of 15 min because of possible myocardial ischemia and due to the "vane effect", which could compromise left ventricular emptying and further impair pulmonary congestion. VT was induced again after a stabilization period of 5–10 min, switching off the CPS, until ablation was achieved. At the end of the procedure, discontinuation of CPS was carried out after echocardiographic evaluation. After heparin neutralization with protamine, CPS cannulae were pulled out and hemostasis was achieved by compression, ordinarily within 6–8 h.



a b

Figures 2a and 2b. Effect of CPS activation after induction of syncopal VT.
a) VT induction (cycle length about 230 ms) immediately causes dramatic systemic hypotension (from 90 mmHg to 30mmHg of the pressure curve). b) Improvement and maintenance of systemic pressure (mean arterial values about 70 mmHg) are achieved after CPS activation (3,500 ml/min) while the VT is ongoing. Five-lead surface ECG, right (RVAp) and left (LVp, LVd) ventricular electrograms, and mean systemic pressure values (FAP) are shown.

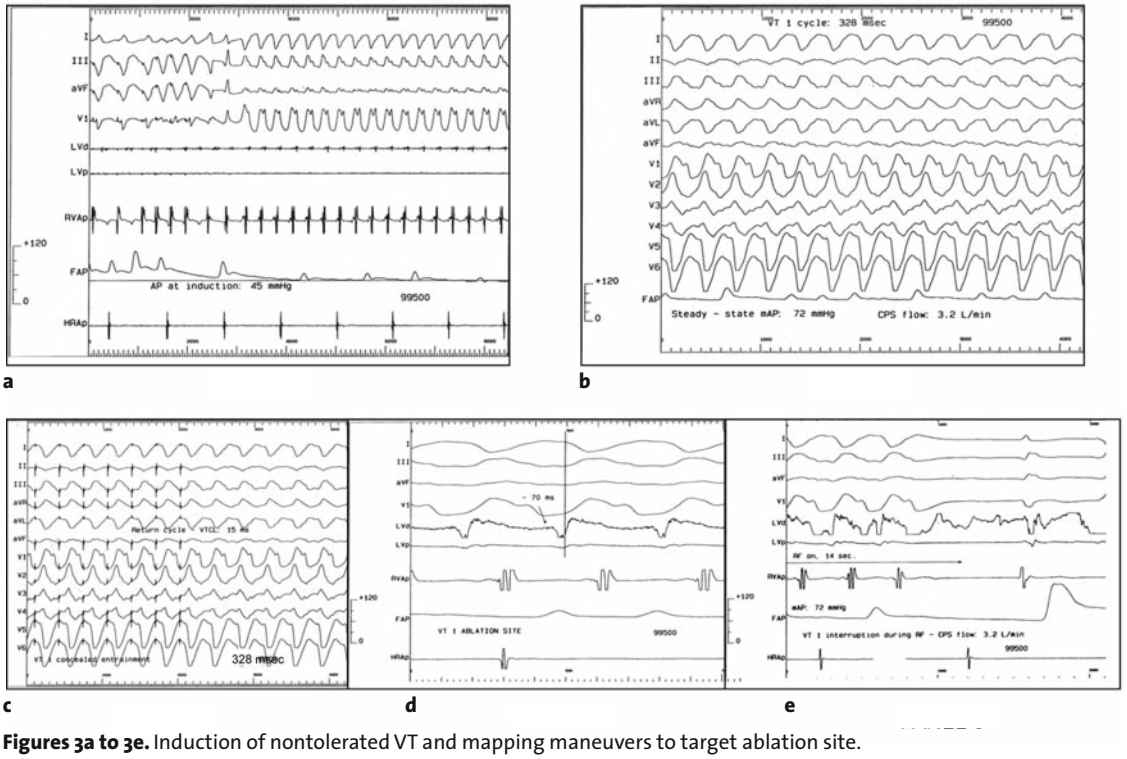
Abbildungen 2a und 2b. Effekt der KPS-Aktivierung nach Induktion synkopaler VT.
a) Die VT-Induktion (Zykluslänge etwa 230 ms) verursacht unmittelbar eine dramatische systemische Hypotonie (von 90 mmHg auf 30 mmHg der Blutdruckkurve). b) Die Erhöhung und Aufrechterhaltung des systemischen Blutdrucks (mittlere arterielle Werte etwa 70 mmHg) werden nach der KPS-Aktivierung (3 500 ml/min) erreicht, während die VT anhält. Fünf-Kanal-Oberflächen-EKG, rechts- (RVAp) und linksventrikuläre (LVp, LVd) Elektrogramme und mittlere systemische Blutdruckwerte (FAP) sind dargestellt.

The electrophysiological procedure was carried out by placing quadripolar catheters in the right atrium and at the right ventricular apex; mapping and ablation were performed by an open-circuit irrigated-tip catheter (ThermoCool, Biosense Webster, Diamond Bar, CA, USA) ntroduced in the left ventricle by a retrograde transaortic (16 patients) or transseptal approach (three patients). Data were recorded on a multichannel electrophysiological system (Prucka Engineering Inc., Houston, TX, USA). Bipolar electrograms were filtered at 30–500 Hz and recorded on a digital system (Prucka Engineering Inc.).

Activation mapping was undertaken in all VTs and CA was guided by the evidence of continuous diastolic activity or of mid-diastolic or presystolic electrograms; validation of ablation site by entrainment maneuvers was undertaken for all VTs with cycle length > 260 ms (Figure 3).

Ablation lesions were obtained as usual by the delivery of radiofrequency current (Stockert, Webster Cordis, ET Technologies Inc, San Jose, CA, USA) between the distal electrode of the mapping catheter and a cutaneous adhesive electrode (30–50 W, maximum temperature 43 °C, irrigation rate 20–30 ml/min). Power output was decreased if any impedance drop of > 10 W was observed. Pulse delivery was interrupted after 30 s whenever ineffective in VT termination; in case of successful termination, sequential point lesions were created by pulses lasting 90–180 s each; loss of capture (10 mA at 2 ms) and electrograms abatement were considered expression of local lesion.

Following CA, a complete stimulation protocol at the 600-, 500- and 400-ms drive cycle through triple extrastimuli from multiple right and left ventricular sites was applied in all patients. Any induced sustained morphology of VT, regardless of its prior documented occurrence, was the target for further ablation and the entire stimulation protocol was repeated subsequently. Endpoint was the noninducibility of any sustained VT, but the procedure was suspended if requested by progressive deterioration of the hemodynamic conditions or by a maximum duration of 5 h.



Figures 3a to 3e. Induction of nontolerated VT and mapping maneuvers to target ablation site.
Drop of systemic arterial pressure after VT onset and its prompt resumption after CPS activation are observed (a, b). During VT, stable pressure values are maintained by CPS; effective concealed entrainment with 12/12-lead match and favorable return cycle is observed at the site (c) where an early presystolic electrogram (70 ms before QRS onset) is recorded (d). Radiofrequency delivery at this site causes termination of the arrhythmia after 14 s (e).

Abbildungen 3a bis 3e. Induktion der nicht tolerierten VT und Mapping-Manöver zum Anvisieren der Ablationsstelle.
Ein Abfall des systemischen arteriellen Blutdrucks nach VT-Beginn und dessen sofortiger Wiederanstieg nach der KPS-Aktivierung werden beobachtet (a, b). Während der VT werden stabile Blutdruckwerte durch den KPS aufrechterhalten; ein effizientes „concealed entrainment" mit 12/12-Übereinstimmung und ein günstiger „return cycle" werden an der Stelle (c) beobachtet, an der ein frühes präsystolisches Elektrogramm (70 ms vor QRS-Beginn) aufgezeichnet wird (d). Die Radiofrequenz-Katheterablation an dieser Stelle verursacht die Terminierung der Arrhythmie nach 14 s (e).

Clinical long-term follow-up was obtained by monitoring for the recurrence of arrhythmias by ICD interrogation at regular follow-up visits (at 4-month intervals) or whenever any symptom occurred. At 6-month intervals patients were asked via phone about symptoms, documented VT recurrences, hospitalization episodes, and ongoing medical treatment.

Statistics

Descriptive statistics are reported as mean and standard deviaton (SD, or range for skewed distributions) for continuous variables and as absolute frequencies and percentages for categorical variables.

Results

Mean duration of the procedures was 4 h and 20 min (range 3 h and 12 min to 5 h and 40 min). CPS was employed with a mean duration of 125 min (range 105–160 min). During the 19 procedures, 62 forms of sustained VT were mapped (mean cycle 340 ms), and 56 (90%; mean cycle 313 ms) required CPS activation due to hemodynamic instability. Systemic arterial pressure before VT induction (CPS standby) was 84 ± 16 mmHg (mean ± SD) with a sudden drop after VT induction (measured 20–60 s after VT induction): estimated mean

Table 2. Procedural data. CPS: cardiopulmonary support; SD: standard deviation; VT: ventricular tachycardia.

Tabelle 2. Verfahrensdaten. CPS: Cardiopulmonarer Support; SD: Standardabweichung; VT: ventrikuläre Tachykardie.

Duration of CPS [min, mean (range)]	125 (105–160)
Systemic arterial pressure before VT induction (mmHg, mean ± SD)	84 ± 16
Systemic arterial pressure after VT induction [mmHg, mean (range)]	45 (20–50)
Systemic arterial pressure after stabilization [mmHg, mean (range)]	68 (55–80)
Target flow of CPS [l/min, mean (range)]	2.6 (2.2–2.9)
Time to hemodynamic stabilization [s, mean (range)]	33 (15–48)

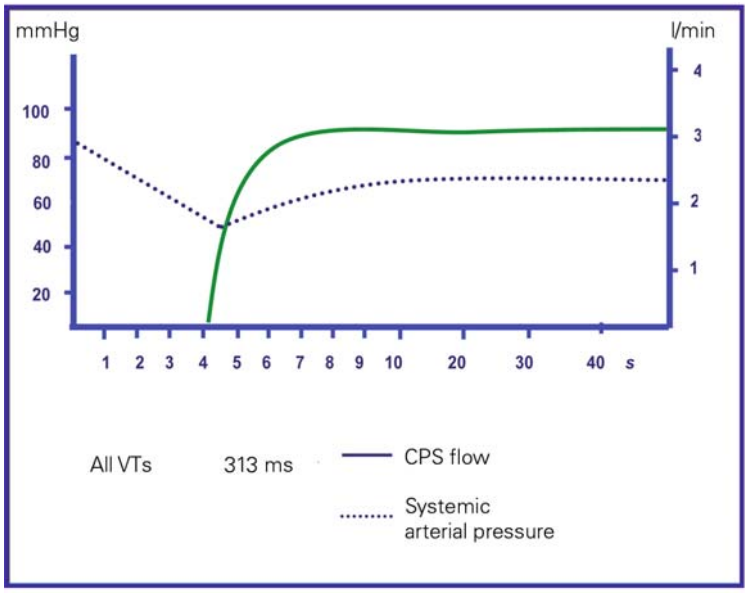


Figure 4. Mean systemic arterial pressure and CPS flow values during circulatory assistance in the course of time in patients' population.
Mean VT cycle length is 313 ms. Systemic arterial pressure before VT induction (CPS standby) is 84 ± 16 mmHg (mean ± SD). Mean arterial pressure after VT induction (measured 20–60 s after onset) is 45 mmHg (range 20–50 mmHg) and increases to 68 mmHg (range 55–80 mmHg) after hemodynamic stabilization (mean time 33 s; range 15–48 s). Target flow of CPS ranges between 2.2 and 2.9 l/min (mean 2.6 l/min).

Abbildung 4. Mittlerer systemischer arterieller Blutdruck und KPS-Flussraten während der Kreislaufunterstützung im Zeitverlauf bei der Patientenpopulation. Die mittlere VT-Zykluslänge beträgt 313 ms. Der systemische arterielle Blutdruck vor der VT-Induktion (KPS-Stand-by) beträgt 84 ± 16 mmHg (Mittelwert ± SD). Der mittlere arterielle Blutdruck nach der VT-Induktion (gemessen 20–60 s nach Beginn) beträgt 45 mmHg (Bereich 20–50 mmHg) und steigt auf 68 mmHg (Bereich 55–80 mmHg) nach hämodynamischer Stabilisierung (mittlere Dauer 33 s; Bereich 15–48 s). Die Soll-Flussrate des KPS liegt im Bereich zwischen 2,2 und 2,9 l/min (Mittelwert 2,6 l/min).

Table 3. Acute outcome; 19 patients. VT: ventricular tachycardia.
Tabelle 3. Akute Ergebnisse; 19 Patienten. VT: ventrikuläre Tachykardie.

Class A patients [n (%)]	10 (52)
Class B patients [n (%)]	5 (27)
Class C patients [n (%)]	4 (21)
In-hospital "VT-free" patients [n (%)]	13 (66)
In-hospital "VT recurrence" patients [n (%)]	6 (34)
(Emergency cardiac transplant – death) [n (%)]	1 (5)

arterial pressure value – based on the evidence of frequent "flattening" of systemic pressure curve – was 45 mmHg (range 20–50 mmHg), with a target mean flow of CPS ranging between 2.2 and 2.9 l/min (mean 2.6 l/min). Stable hemodynamic improvement was achieved after a mean time of 33 s (range 15–48 s) reaching mean arterial pressure values of 68 mmHg (range 55–80 mmHg; Table 2, Figure 4). Mean activated coagulation time was 310 ± 65 s. No significant blood gases or arterial oxygen saturation abnormalities were observed. After CPS discontinuation, a normal hemodynamic condition was immediately restored in 15/18 patients.

Mean number of VTs was 3.1/patient in patients with chronic ischemic cardiomyopathy, 4.4/patient in patients with primary dilated cardiomyopathy, 2.5/patient in patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy. CA was acutely effective in 48/62 VTs (77%) and in 45/56 (80%) among those assisted by CPS. Target for CA was isolated diastolic electrogram in 22 VTs, presystolic electrogram in 14, continuous fragmented activity in nine; evidence of concealed entrainment at efficacious ablation site was achieved in ten VTs.

In 10/19 patients (52%) all forms of spontaneous VT and all VT morphologies induced by programmed electrical stimulation were successfully ablated and were no more inducible after CA. In nine patients (47%) one or more forms of VT could be successfully ablated, but one or more were still inducible (in four patients clinical VT, in five only nonclinical forms of VT; Table 3). Mean fluoroscopy time was 49 min (range 22–94 min).

Three patients with severe left ventricular function impairment (ejection fraction 10–20%) required prolonged infusion of inotropic agents before successful weaning from CPS; one patient had transient left ventricular stunning at CPS interruption; one patient had a transient ischemic attack, and two a mild form of anemia. No severe complication related to extracorporeal circulation (thromboembolic disease, renal failure) or to femoral cannulation occurred.

Complete clinical success was observed in 13 patients (68%) in whom no significant recurrence of sustained VT occurred during the subsequent 7-day monitoring period; six patients (32%) had recurrences of VT (in two patients without ICD shocks) that were afterwards suppressed by antiarrhythmic drugs. Among them, one patient with persistent ES after failing CA acutely underwent cardiac transplant and died 2 weeks later because of postsurgical hemothorax (Table 3).

At a mean follow-up of 42 months (range 15–60 months), 5/18 surviving patients (28%) were free from VT recurrences; 7/18 patients (39%) had fewer VT recurrences with ICD interventions and a correspondent clinical improvement. Among 14 patients presenting ES, five had ES recurrences: among them, three died because of acute heart failure (Table 4).

Discussion

Clinical Significance and Rationale for the Treatment of Unstable Arrhythmias

The incidence of hemodynamically nontolerated forms of VT is constantly increasing in the ICD popu-

lation and the occurrence of unstable arrhythmias is the main responsible for multiple shocks and acute heart failure in patients with advanced structural heart disease [16–18]. Pacifico et al. [19] have shown that the occurrence of shocks is a potent negative predictor of survival independent of several risk factors, including ejection fraction. Moreover, based on our previous study [4], spontaneous nontolerated VTs are consistent with the clinical presentation in 71% of patients undergoing CA for drug-refractory ES: in this population the suppression of all clinical VTs is required to control ES and predicts a long-term favorable outcome in terms of prevention of arrhythmia recurrences and of improved survival.

Despite the clinical and prognostic significance of recurrent unstable VTs in ICD recipients, their effective treatment is a main challenge for CA, as conventional point-by-point activation mapping is precluded by the characteristics of the arrhythmia.

Limits of Alternative Strategies of Mapping for the Treatment of Nontolerated Arrhythmias

Unstable VTs are approached by modern three-dimensional methods in the majority of patients. The electroanatomic mapping system allows the accurate characterization of the endo- and epicardial ventricular aspect and may guide the so-called substrate-based approach [20–22]. In this setting, the detection of conducting channel within the area of dense scar and the use of pacing maneuvers enhance specificity and allow a more precise and effective treatment of the substrate responsible for the arrhythmia [23].

On the other hand, the multielectrode noncontact mapping system allows the reconstruction of the activation pattern of the arrhythmia based on the recording of a single cycle of the arrhythmia, that can be induced and promptly terminated [24]. However, the use of these methods of mapping does not fulfill the need of treatment for patients with unstable arrhythmias, when the absence of at least transient phases of sinus rhythm precludes their application and the ongoing arrhythmia is the cause for hemodynamic failure.

Therefore, it frequently happens that, in patients with severely depressed cardiac function, the recurrence of frequent nontolerated VTs is the cause of cardiogenic shock; in this setting, the administration of antiarrhythmic drugs or inotropic agents is rarely effective [25]. In the majority of patients, the termination of each single arrhythmia episode achieved by multiple consecutive internal cardioversions further impairs clinical and hemodynamic conditions; thus, only potentially lifesaving treatment is represented by CA, but its feasibility is significantly limited by the clinical presentation by itself.

Table 4. Long-term outcome (mean follow-up of 42 months; range 15–60 months); 18 patients. ES: electrical storm; VT: ventricular tachycardia.

Tabelle 4. Langfristige Ergebnisse (mittlerer Nachbeobachtungszeitraum von 42 Monaten; Range 15–60 Monate); 18 Patienten. ES: elektrischer Sturm; VT: ventrikuläre Tachykardie.

"VT-free" patients [n/population (%)]	5/18 (28)
"VT recurrence" patients [n/population (%)]	9/18 (50)
Overall clinical improvement [n/population (%)]	12/18 (66)
"ES-free" patients [n/group (%)]	9/14 (64)
"ES recurrence" patients [n/group (%)]	5/14 (36)
Cardiac death after ES recurrence [n/group (%)]	3/5 (60)

How to Achieve Hemodynamic Stabilization During CA

Inotropic agents may support hemodynamic conditions for brief periods of time in patients with nontolerated arrhythmias during CA; however, they provide no additional benefit for patients presenting with severe refractory cardiac decompensation. Mischke et al. [26] proposed a specific pacing algorithm of coupled ventricular pacing as an alternative "electrical" method to stabilize relatively slow VT in patients with moderate left ventricular impairment, for very short periods; main limitation of this strategy relates, however, to the impossibility to map VT while pacing. The percutaneous left ventricular assist device to support CA of nontolerated VTs has been described in a single patient by Friedman et al. [27]; based on a single case experience, this device represents a very effective alternative to CPS in providing adequate indices of perfusion, but major complications related to the more invasive technique of implant must be taken into account. On the other hand, the effectiveness of the intraaortic balloon pump [28, 29] is conditioned by cardiac rhythm synchronization for circulatory assistance: therefore, it cannot be used for hemodynamic support during ongoing VT.

A relevant experience has been acquired about the use of CPS to protect high-risk patients undergoing coronary angioplasty from myocardial ischemia [11–14]. Our exclusive experience refers to the use of CPS as assistant device to enable CA in patients with unstable VTs; it indicates that CPS may assure stability for a reasonable time to perform activation mapping, thus improving CA safety and success. In addition, the opportunity to make "tolerated" those arrhythmias that were originally "nontolerated", offers the unique opportunity to approach them conventionally by a point-by-point mapping once other approaches have failed. CPS candidates represent about

2% of the total amount of patients with structural heart disease undergoing CA for any type of VT.

Conclusion

CPS-assisted CA aims at the treatment of selected patients in whom unstable VTs cause acute hemodynamic failure; based on our experience, CPS assures acceptable hemodynamic stabilization and protects the patient throughout the procedure allowing VT tolerance and efficacious mapping. Its use is particularly meaningful in patients presenting with intractable arrhythmias in whom the control of the arrhythmia is lifesaving, as patients with ES are. Clinical success in our high-risk population indicates the relative efficacy in VT prevention that translates into an improved short-term survival, without significant procedure-related complications. The need for experienced cardiac electrophysiologists, interventional cardiologists, cardiac anesthesiologists and for extensively trained nurses and technicians, operating in a dedicated milieu, implies that the use of CPS-assisted CA must be restricted to very selected high-competency institutions.

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