



Colmare il gap autonomico: un piano d'azione personalizzato contro la tempesta aritmica.

- Fase acuta**
- Protezione a lungo termine**

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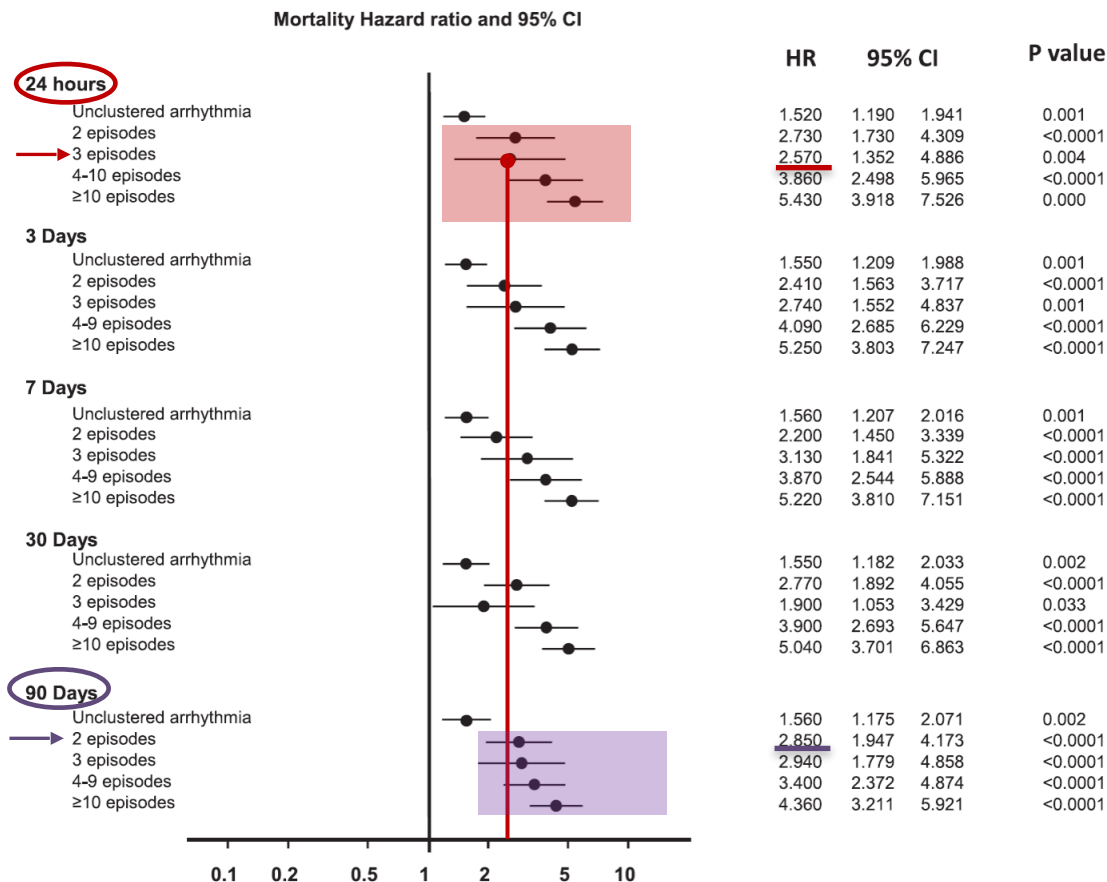


Electrical Storm

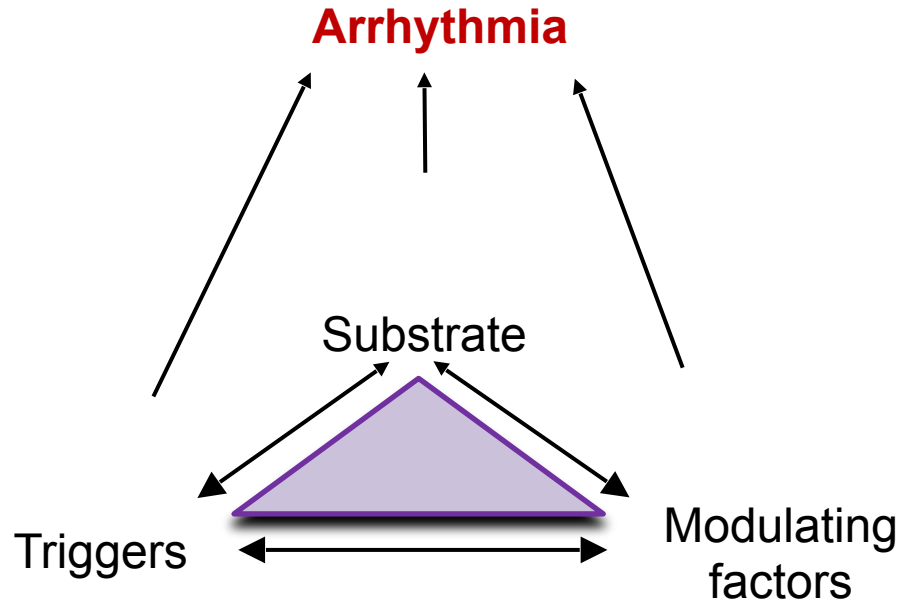


≥ 3 sVT/VF episodes over 24 h (including ICD-treated VT/VF)
or an incessant VT lasting ≥ 12 h

Mortality associated with clustered sVT/VF

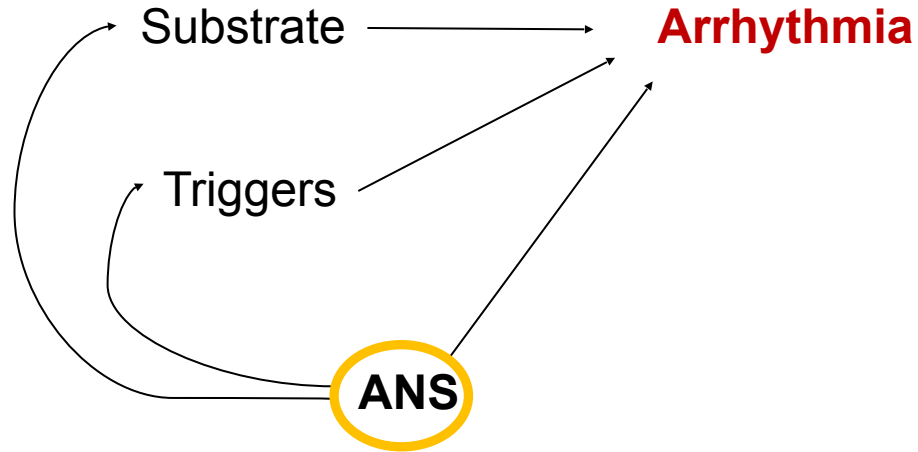


Physiopathology of VA



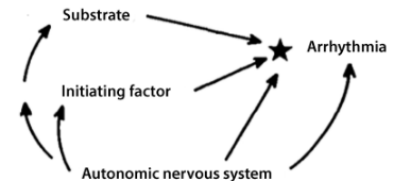
Modified triangle of Coumel

Physiopathology of VA



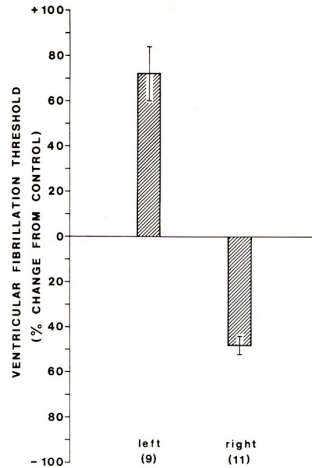
Original concept
Modified triangle of Coumel

(EHJ 1987)



What are the electrophysiological effects of left/bilateral cardiac sympathetic block/denervation?

Stellate ganglion block and ventricular fibrillation threshold

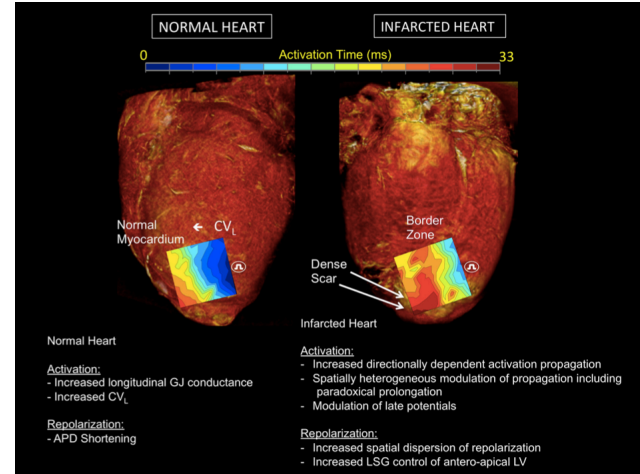


PJ Schwartz et al, Am J Cardiol 1976

Polymorphic VT/VF susceptibility at rest and during acute sympathetic activation

pVT/VF: 15-20% of clinical VT

Sympathetic modulation and electrical activation in the infarcted myocardium



Ajjola et al. Am J Physiol Heart Circ Physiol 2017; 312

Scar-related monomorphic VT susceptibility at rest and during acute sympathetic activation

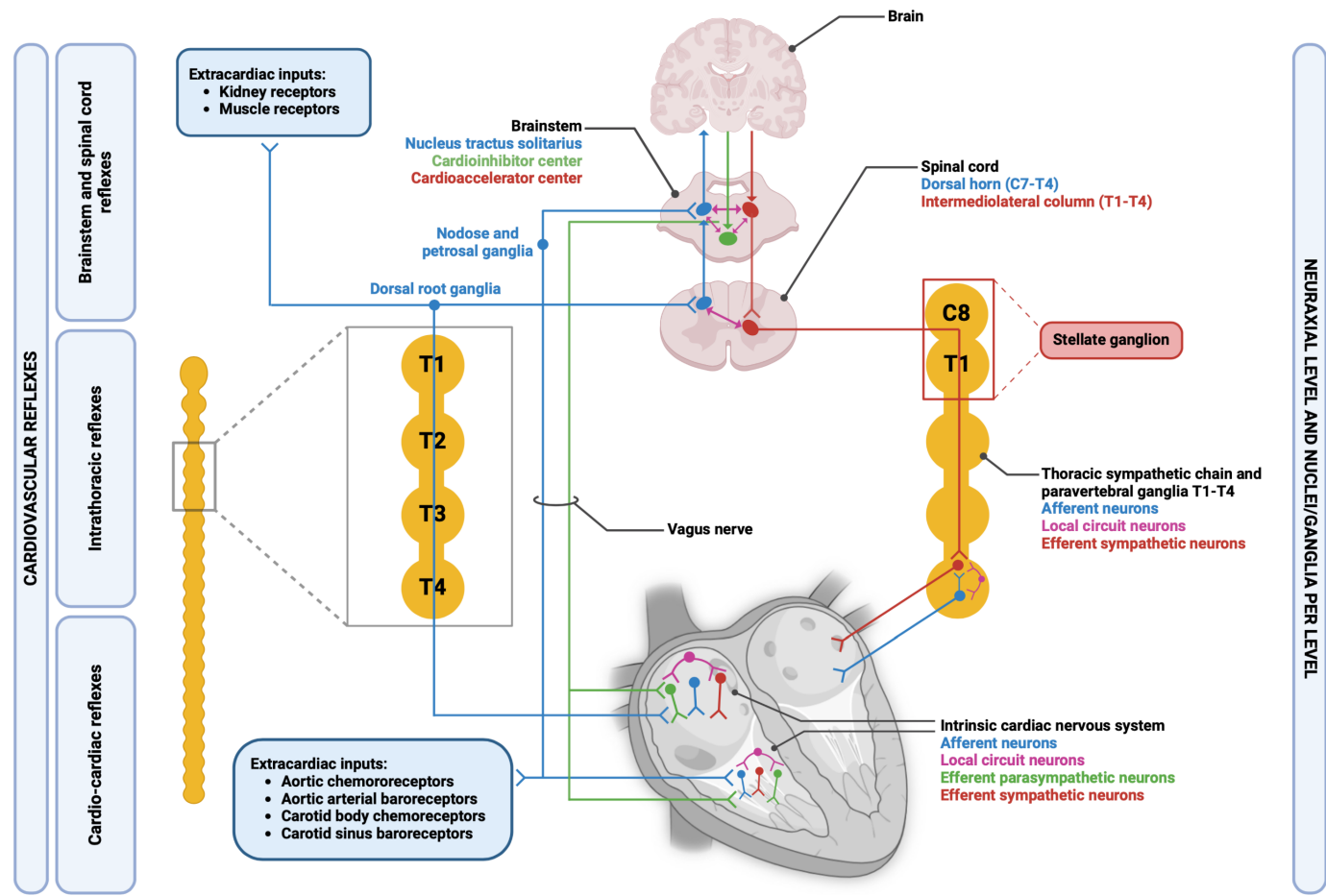
MMVT: 75-80% of clinical VT

Neural control of the heart

Sympathetic Asymmetry

Right SG: SAN, both atria and ventricular anterior surface (mostly right ventricle)

Left SG: Mostly AV node, both atria and ventricular posterior surface (most of the LV)

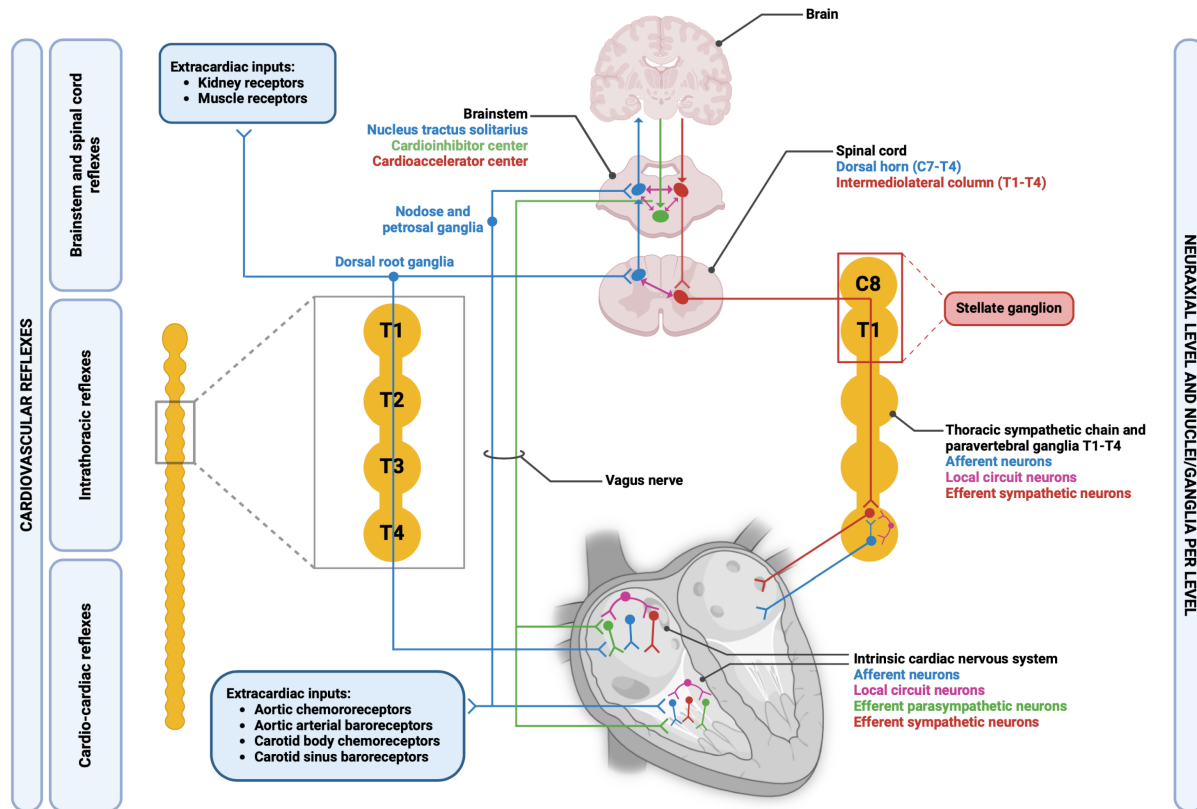


Cardiac neuraxis as a therapeutic target in the ICU

3. Central level Central sympathetic modulation (Sedation, VNS)

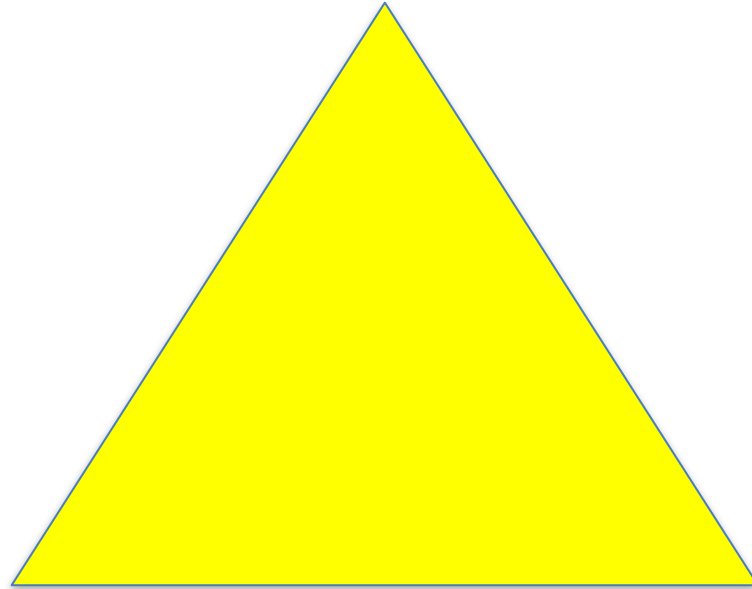
2. Intrathoracic level Percutaneous neuronal sympathetic block

1. Intracardiac level Antiadrenergic drugs



Calming the storm: the pillars of adrenergic blockade in the ICU

**Left stellate
ganglion block**



**Antiadrenergic
drugs (Not
selective- BBs,
amiodarone)**

Sedation

Intervention	Current										Receptors					Peak levels (min)	Inotro-pism	BP	Effects on ECG			
	INa peak			INaL	ICaL	Potassium					α-Adren		β- Adren		NPY				HR	PQ	QRS	JT
	Fast	Med	Slow			IKr	IKs	IK1	IK ACh	Ito	α1	α2	β1	β2								
Oral Amiodarone	●			●	●	●	●			●	●	●	●	●			→	→	↓	↑	→	↑
Amiodarone IV (Bolus)	●			●	●	●	●			●	●	●	●	●		15-20	→	↓	→	↑	→	→
Lidocaine	●			●												1-5	→	→	→	→	→	↓
Procainamide IV		●				●										15-60	↓	→	→	↑	↑	↑
Nadolol												●	●			120-240	↓	↓	↓	↑	→	→
Propranolol Short-acting	●											●	●			60-240	↓	↓	↓	↑	→	↓
Metoprolol Short-acting												3	1			90-120	↓	↓	↓	↑	→	→
Metoprolol IV												3	1			5-15	↓	↓	↓	↑	→	→
Esmolol IV												33	1			1-5	↓	→	↓	↑	→	→
Landiolol IV												255	1			2-5	↓	→	↓	↑	→	→
PLSGB				↓	↓		↓			↓	Ligand release blocked					1-5	→	→	→	→	→	↓

● Low potency Antagonist

○ Low potency Agonist

↑ Increases

● Medium potency Antagonist

○ Medium potency Agonist

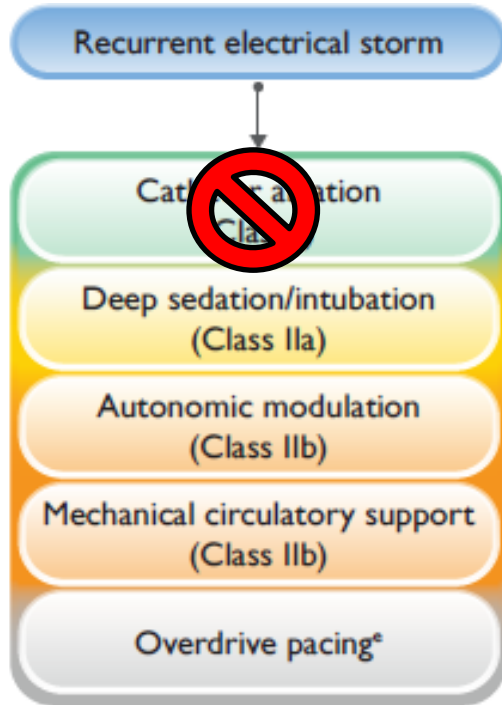
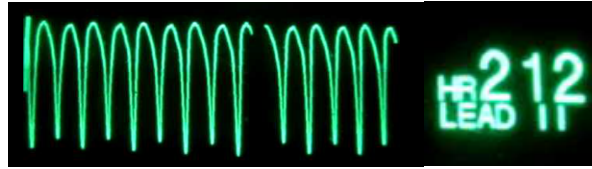
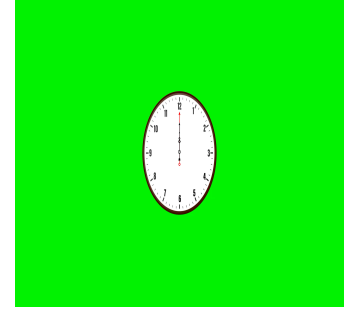
↓ Decreases

● High potency Antagonist

○ High potency Agonist

→ No changes

Acute setting (ICU): what if antiarrhythmic drugs fail?The clock is ticking



Hours to days

Minutes to Hours

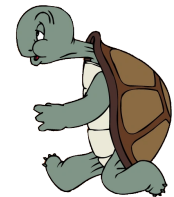
Percutaneous left stellate ganglion block: minutes

Thoracic epidural anesthesia: minutes to hours

Cardiac sympathetic denervation: days (and hemodynamic stability)

Bolus (one shot)

Continuous infusion



Electrical storm management in structural heart disease

Acute phase stabilization

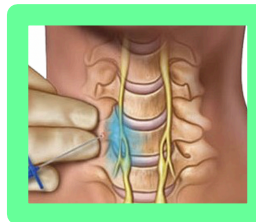
Subacute/Chronic phase

Intra-hospital dedicated protocols

Referral to specialized centers

Initial strategy:

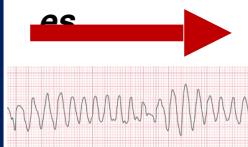
- **Anti-arrhythmic drugs:**
 - **Amiodaron iv:** 300 mg or 5 mg/kg (loading dose) in 5-20 min (max 60'), then 600-1200 mg/24h irrespective of acute efficacy
 - **Lidocaine iv:** 1-2 mg/kg boluses, if effective continue 20-50 y/kg/min
 - **Procainamide iv** (consider only if NYHA I/II and/or SCAI A): 100 mg bolus repeatable (max 50 mg/min), then 2-6 mg/min
 - **Non-selective BB:** e.g. oral propranolol 10-40 mg every 6h; if more rapid effect needed: iv esmolol or landiolol
- **Mild to moderate sedation:** benzodiazepine + opioid, consider dexmedetomidine.
- **Correction of reversible causes:** ischemia, hypoxemia, fever, electrolytes imbalance ($K^+ > 4$ mEq/L, $Mg^{2+} > 2$ mg/dL, corrected Ca^{2+} in range), toxins
- **ICD reprogramming**
- **Pharmacological LV unloading**



PLSGB

independently from SCAI

Recurrent



SCAI D/E

ECMO +
deep
sedation

Inotropes, pVADs,
ECMO

SCAI C

Combination strategy:

- BB ± levosimendan
- BB ± IABP

SCAI A/B

Hemodynamic evaluation

Multidisciplinary heart team discussion
(eligibility for permanent LVAD and/or HTx
assessment)

1. Non-pharmacological treatments implementation:

- **RFCA** (MMVT or PVB-triggered polymorphic VT/VF). Asses need for prophylactic MCS (PAINESD score)
- **CSD** (Polymorphic VT/VF, recurrent MMVT > 150 bpm after RFCA and/or MMVT of multiple different morphologies)
- **Non-invasive stereotactic ablation** (slow MVT, worse NYHA class, frail/older patients)

2. If 1 unfeasible/excessive risk/refused:

- **Propranolol** or **nadolol** +
- **Oral amiodaron** +
- **Mexiletine** (or **ranolazine**)

Timing of Ablation and Prognosis of Patients With Electrical Storm and Scar-Related Left Ventricular Dysfunction



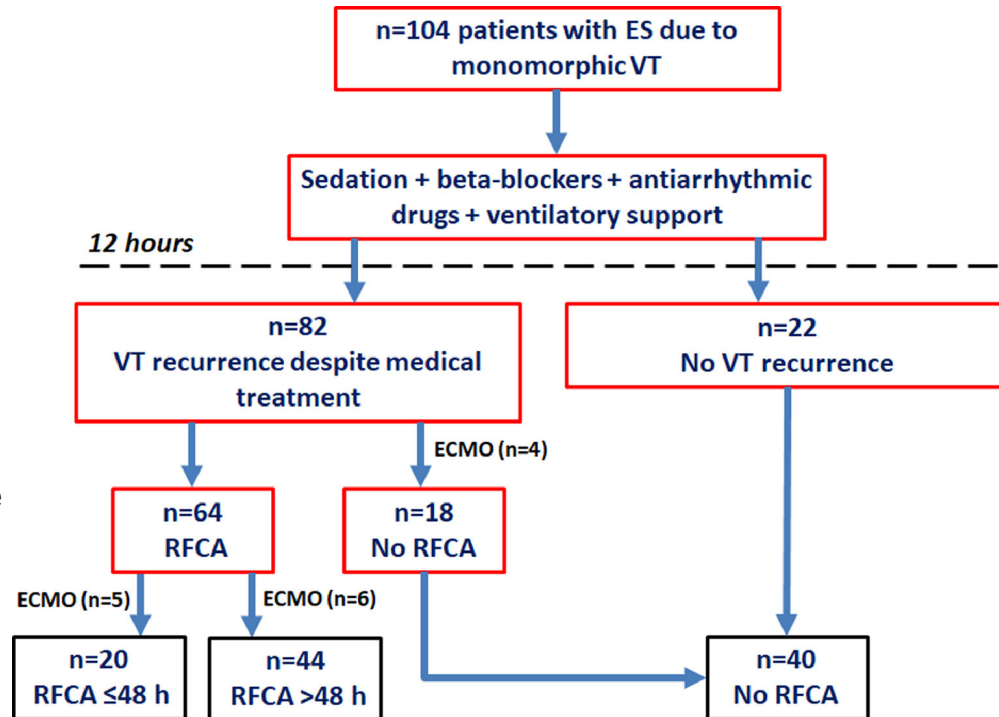
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Javier Fernández-Portales, MD, PhD^b, Manuel Durán, MD^c, Olga Durán, MD^d, Jendri Pérez, MD^a,
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Presentation

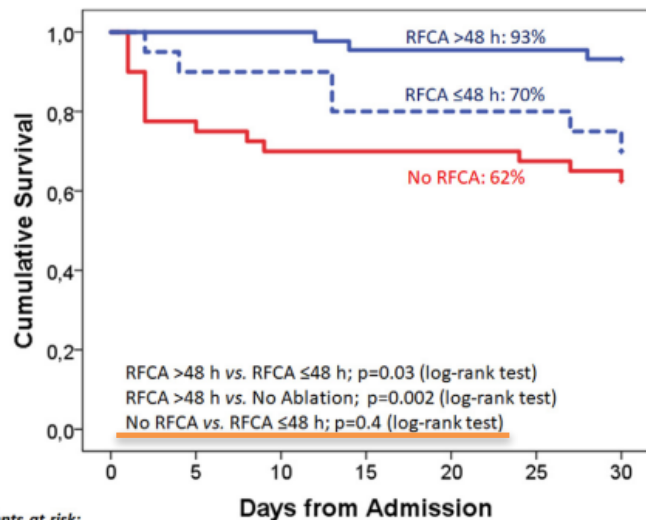
72 ± 10 yrs,
LVEF 30 ± 6%,
34% previous NYHA III/IV
64% with ICD, 33% NICM,
15% on chronic amiodarone
Clinical VT cycle: 352 ± 42 ms
**24% severe HF at admission
(pulmonary oedema or shock)**

Treatment

14% ECMO
62% RFCA (15% endo/epi)
85% on oral amiodarone at discharge



30-day Mortality Predictors



Patients at risk:

	0	5	10	15	20	25	30
RFCA >48 h	44	44	44	42	42	42	41
RFCA ≤48 h	20	18	18	16	16	16	14
No RFCA	40	35	28	28	28	26	25

Table 2

Thirty-day mortality predictors. Cox-regression multivariate analysis. Model 1 (n = 104 patients)

Variable	Hazard ratio	95% confidence interval		p value
		Lower	Upper	
Age (y)	1.03	0.9	1.1	0.09
Female gender	3.1	1	9.6	0.06
Diabetes mellitus	0.9	0.4	2.1	0.8
Functional Class >1 (NYHA)	1.4	0.4	1.7	0.6
LVEF (%)	1	0.9	1.05	0.9
Severe heart failure (pulmonary oedema o shock) at admission	3.3	1.2	9.3	0.02
Creatinine clearance <60 ml/min/1.73 m ² at admission	2.2	0.8	5.4	0.1
At least one documented episode of VF during hospitalization	2.3	0.8	7.1	0.1
Ischaemic cardiomyopathy	0.1	0.02	0.6	0.01
Coronary revascularization	0.4	0.1	1.2	0.1
ECMO	0.8	0.2	3.2	0.7
Catheter ablation	0.42	0.06	0.6	0.008

ECMO = extracorporeal membrane oxygenation; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; VF = ventricular fibrillation.

Table 3

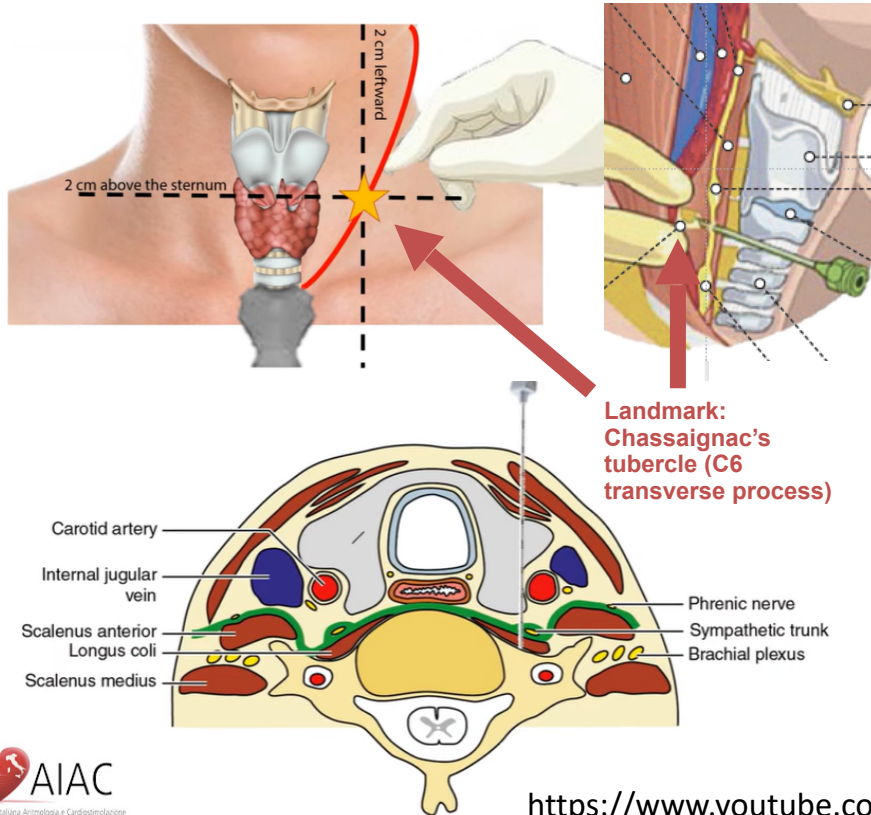
Thirty-day mortality predictors. Cox-regression multivariate analysis. Model 2 (n = 104 patients)

Variable	Hazard ratio	95% confidence interval		p value
		Lower	Upper	
Age (y)	1.03	0.9	1.07	0.1
Female gender	2.5	0.9	7.4	0.09
Diabetes mellitus	0.8	0.3	2.1	0.7
Functional Class >1 (NYHA)	1.2	0.4	3.4	0.7
LVEF (%)	1	0.9	1.07	0.7
Severe heart failure (pulmonary oedema o shock) at admission	3.9	1.4	11.2	0.009
Creatinine clearance <60 ml/min/1.73 m ² at admission	1.9	0.7	2.1	0.2
At least one documented episode of VF during hospitalization	2.1	0.7	6.06	0.2
Ischaemic cardiomyopathy	0.2	0.03	0.8	0.2
Coronary revascularization	0.5	0.2	1.3	0.1
ECMO	0.7	0.2	2.7	0.6
Catheter ablation >48 h from admission	0.33	0.03	0.6	0.007

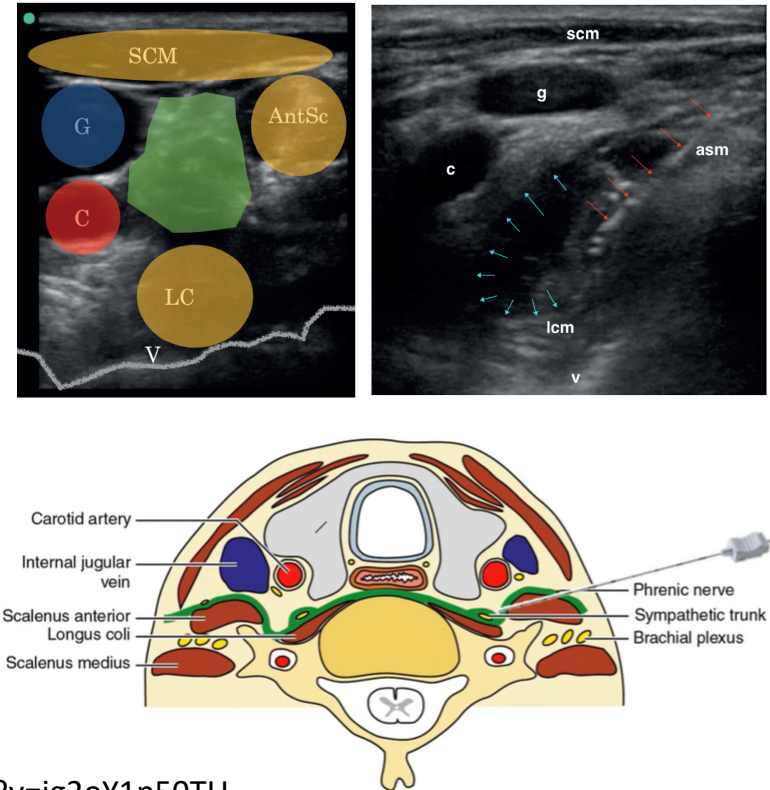
ECMO = extracorporeal membrane oxygenation; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; VF = ventricular fibrillation.

Percutaneous stellate ganglion block (PSGB)

Anatomical approach (anterior)



US-guided approach (lateral)



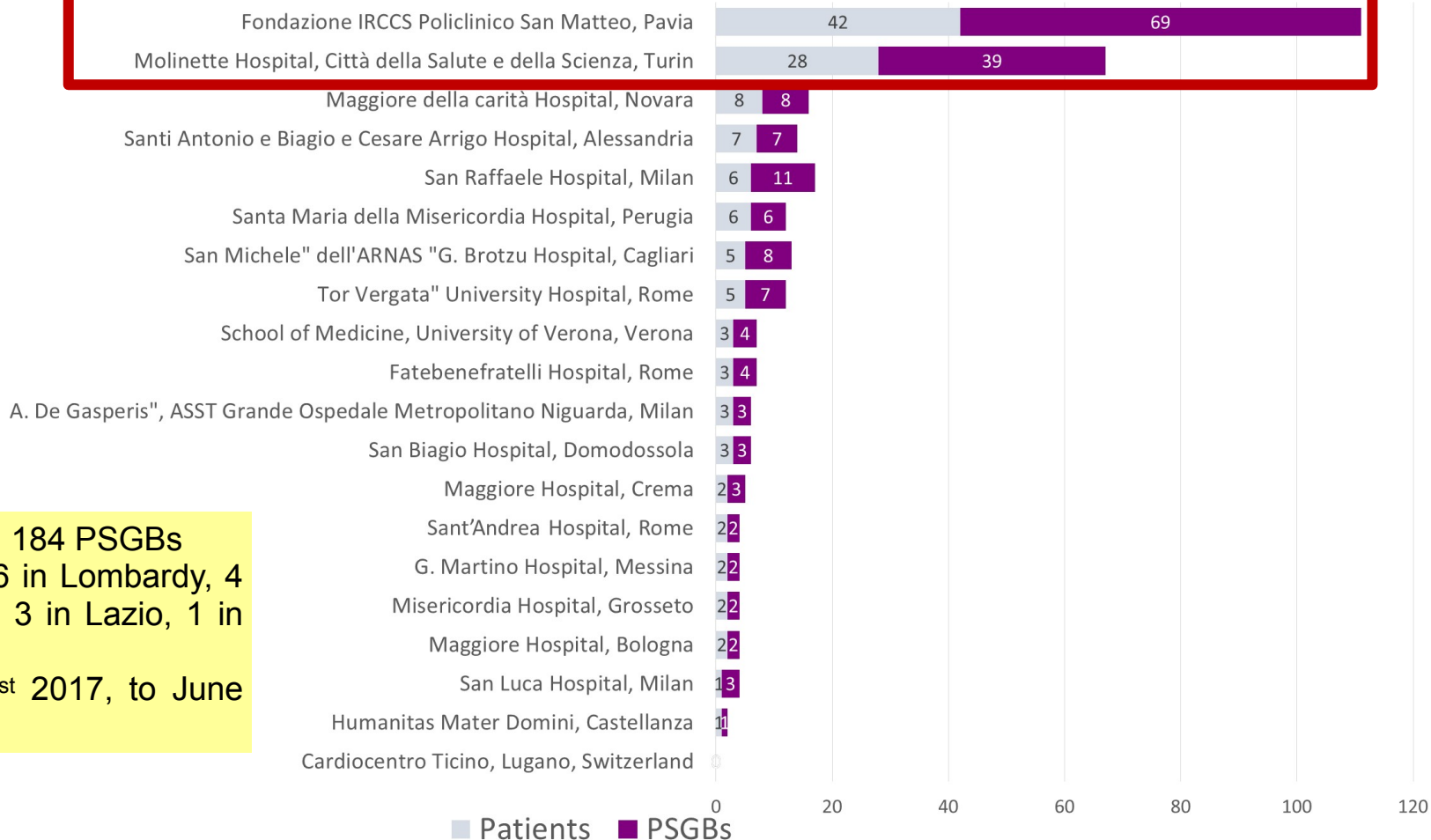
Electrical storm treatment by percutaneous stellate ganglion block: the STAR study

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Active Centres



- 131 patients, 184 PSGBs
- 19 centres, 6 in Lombardy, 4 in Piedmont, 3 in Lazio, 1 in Sardinia
- From July 1st 2017, to June 30th 2023.

Table 2 Characteristics of PSGBs (*n* = 184)

Characteristic	<i>n</i> = 184
Approach, <i>n</i> (%)^a	
Anterior anatomical	106 (57.6)
Lateral ultrasound-guided	78 (42.4)
Side, <i>n</i> (%)^a	
Left	181 (98.4)
Right	3 (1.6)
Mode of administration, <i>n</i> (%)^a	
Bolus	152 (82.6)
Bolus and infusion	32 (17.4)
Anaesthetic used for bolus, <i>n</i> (%)^a	
Lidocaine	53 (28.8)
Bupivacaine	8 (4.3)
Ropivacaine	1 (0.5)
Mepivacaine	3 (1.7)
Lidocaine + bupivacaine	44 (23.9)
Lidocaine + ropivacaine	63 (34.2)
Lidocaine + mepivacaine	11 (6)
Lidocaine + levobupivacaine	1 (0.5)
Anaesthetic used for infusion, <i>n</i> (%)^a	
Lidocaine	21 (65.6)
Ropivacaine	11 (34.4)
Anaesthetic infusion duration, min^b	3660 (1440–7203.8)

Type of arrhythmias during electrical storm, *n* (%)^a

VT	118 (64.1)
VF	35 (19)
VT and VF	31 (16.8)
ATPs/shocks in the 12 h before the first PSGB ^a	6 (3–15.8)

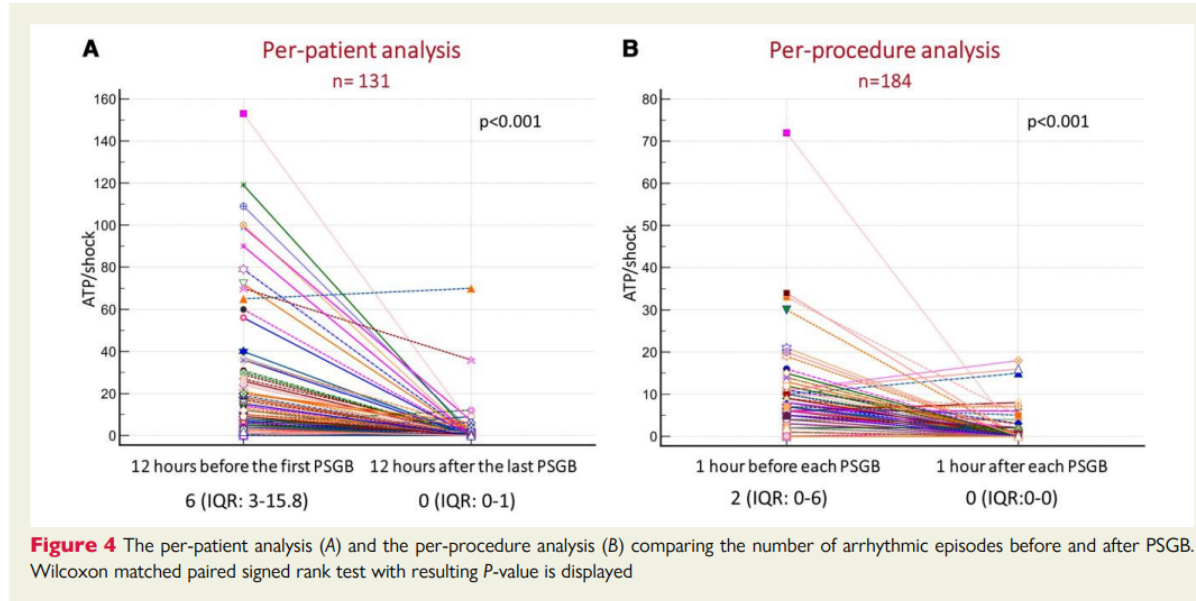
Anticoagulant/antiplatelet therapy, *n* (%)^a

None	26 (14.1)
SAPT only	35 (19)
SAPT + heparin	29 (15.8)
SAPT + VKA/DOAC	6 (3.3)
DAPT only	13 (7.1)
DAPT + heparin	16 (8.7)
DAPT + VKA/DOAC	1 (0.5)
Heparin only	24 (13)
VKA/DOAC only	34 (18.5)

Primary efficacy endpoint:

92% of pts with a reduction of ATP/shock $\geq 50\%$ in the first 12 hours after PSGB

Secondary efficacy endpoints: impact of PSGB on the arrhythmic burden



Primary safety endpoint: major complications

Anticoagulant/antiplatelet therapy, n (%)^a

None	26 (14.1)
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Post-PSGB anisocoria, n (%)^a

Yes	72 (39.1)
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No	103 (56)
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Unknown	9 (4.9)
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PSGB major complications, n (%)^a

Respiratory depression	1 (0.5)
------------------------	---------

PSGB minor complications, n (%)^a

Bradycardia	1 (0.5)
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Hypotension	1 (0.5)
-------------	---------

PSGB described side effects, n (%)^a

Temporary brachial plexus paralysis	3 (1.6)
-------------------------------------	---------

Hoarseness	2 (1.1)
------------	---------

Dysphonia	1 (0.5)
-----------	---------

Neck pain	1 (0.5)
-----------	---------

Vomiting	1 (0.5)
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Beware of LAST (local anesthetic systemic toxicity), potentially lethal but 100% treatable!!



Lipidic emulsion 20%

- >70 kg: Bolus 100 ml (2-3 min), infusion 250 ml in 15-20 min
- <70 kg: Bolus 1.5 ml/kg in 2-3 min, infusion 0.25 ml/kg/min in 15-20 min

A Multicenter Study of Stellate Ganglion Block as a Temporizing Treatment for Refractory Ventricular Arrhythmias

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ABSTRACT

BACKGROUND Ventricular tachycardia (VT) and ventricular fibrillation (VF) are life-threatening conditions and can be refractory to conventional drug and device interventions. Stellate ganglion blockade (SGB) has been described as an adjunct, temporizing intervention in patients with refractory ventricular arrhythmia. We examined the association of SGB with VT/VF in a multicenter registry.

OBJECTIVES This study examined the efficacy of SGB for treatment/temporization of refractory VT/VF.

METHODS The authors present the first analysis from a multicenter registry of patients treated for refractory ventricular arrhythmia at a clinical site in the Czech Republic and the United States. Data were collected between 2016 and 2022. SGB was performed at the bedside by anesthesiologists and/or cardiologists. Outcomes of interest were VT/VF burden and defibrillations at 24 hours before and after SGB.

RESULTS In total, there were 117 patients with refractory ventricular arrhythmias treated with SGB at Duke ($n = 49$) and the Institute for Clinical and Experimental Medicine ($n = 68$). The majority of patients were male (94.0%), were White (87.2%), and had an implantable cardioverter-defibrillator (70.1%). The most common etiology of heart disease was ischemic cardiomyopathy (52.1%), and monomorphic VT was the most common morphology (70.1%). Within 24 hours before SGB (0-24 hours), the median episodes of VT/VF were 7.5 (Q1-Q3: 3.0-27.0), and 24 hours after SGB, the median decreased to 1.0 (Q1-Q3: 0.0-4.5; $P < 0.001$). At 24 hours before SGB, the median defibrillation events were 2.0 (Q1-Q3: 0.0-8.0), and 24 hours after SGB, the median decreased to 0.0 (Q1-Q3: 0.0-1.0; $P < 0.001$).

CONCLUSIONS In the largest cohort of patients with treatment-refractory ventricular arrhythmia, we demonstrate that SGB use was associated with a reduction in the ventricular arrhythmia burden and need for defibrillation therapy. (J Am Coll Cardiol EP 2024;■:■-■) Published by Elsevier on behalf of the American College of Cardiology Foundation.

Bi-centric study: Duke University Medical Center, Durham, North Carolina, USA, and the Institute of Clinical and Experimental Medicine (IKEM), Prague, Czech Republic

Enrollment period: between 2016 and 2022.

Population: 117 patients, bilateral PSGB 18% (39% at Duke, 3% at IKEM)

Patients in active ischemia were not included in the study before revascularization

JACC Clin Electrophysiol. 2024;10:750-758

TABLE 1 Patient Baseline Characteristics and Interventions			
	All SGB (N = 117)	Duke SGB (n = 49)	IKEM SGB (n = 68)
Age, y	63.5 ± 11.0	62.1 ± 11.2	64.4 ± 10.8
Male	110 (94.0)	47 (95.9)	63 (92.6)
Study location			
Duke (United States)	49 (41.9)	—	—
IKEM (Czech Republic)	68 (58.1)	—	—
Race			
White	102 (87.2)	34 (69.4)	68 (100.0)
Black	15 (12.8)	15 (30.6)	0 (0.0)
Atrial fibrillation	56 (47.9)	30 (61.2)	26 (38.2)
ICD	82 (70.1)	40 (81.6)	42 (61.8)
CRT-D	44 (37.6)	20 (40.8)	24 (35.3)
LVEF, %	26.7 ± 9.2	22.6 ± 9.1	26.1 ± 8.2
Beta-blocker (intravenous or oral at time of VT/VF)	16 (13.7)	5 (10.2)	11 (16.2)
Etiology			
NICM	59 (50.4)	26 (54.2)	33 (48.5)
ICM	61 (52.1)	23 (46.9)	38 (55.9)
Post-MI	26 (22.2)	9 (18.4)	17 (25.0)
Vasospasm	3 (2.6)	3 (6.1)	0 (0.0)
Long QT	21 (17.9)	19 (38.8)	2 (2.9)
Arrhythmia type			
Polymorphic VT	48 (41.0)	30 (62.5)	18 (26.5)
Monomorphic VT	82 (70.1)	25 (52.1)	57 (83.8)
VF	37 (31.6)	17 (34.7)	20 (29.4)
Antiarrhythmic drugs			
Amiodarone	99 (84.6)	47 (95.9)	52 (76.5)
Lidocaine/mesocaine	62 (53.0)	39 (79.6)	23 (33.8)
Mexiletine	9 (7.7)	9 (18.4)	0 (0.0)
Procainamide	22 (18.8)	22 (44.9)	0 (0.0)
Quinidine	3 (2.6)	3 (6.1)	0 (0.0)
Interventions			
General sedation/anesthesia	66 (56.4)	26 (55.3)	40 (59.7)
Inotropes	22 (18.8)	12 (24.5)	10 (14.7)
Electrophysiologic ablation	66 (56.4)	26 (53.1)	40 (58.8)
Mechanical circulatory support			
IABP	9 (7.7)	6 (12.2)	3 (4.4)
Impella (Abiomed)	5 (4.3)	4 (8.2)	1 (1.5)
ECMO	9 (7.7)	5 (10.2)	4 (5.9)
LVAD	15 (12.8)	13 (26.5)	2 (2.9)

TABLE 2 SGB Procedure Characteristics and Outcomes				
	All SGB (N = 117)	Duke SGB (n = 49)	IKEM SGB (n = 68)	P Value
Pre-SGB				
VT events 48 h prior	1.0 (0.0-8.8)	1.0 (0.0-3.0)	3.0 (0.0-15.0)	0.266
Shocks 48 h prior	0.0 (0.0-2.0)	0.0 (0.0-2.5)	0.0 (0.0-2.0)	0.268
VT events 24 h prior	7.5 (3.0-27.0)	3.0 (2.0-6.0)	15.0 (7.0-34.0)	<0.001
Shocks 24 h prior	2.0 (0.0-8.0)	3.0 (1.0-6.0)	2.0 (0.0-8.8)	0.600
Post-SGB				
VT events 48 h post	0.0 (0.0-1.0)	0.0 (0.0-1.0)	0.0 (0.0-2.0)	0.742
Shocks 48 h post	0.0 (0.0-0.0)	0.0 (0.0-0.0)	0.0 (0.0-0.0)	0.512
VT events 24 h post	1.0 (0.0-4.5)	0.0 (0.0-2.0)	1.0 (0.0-11.0)	0.022
Shocks 24 h post	0.0 (0.0-1.0)	0.0 (0.0-1.5)	0.0 (0.0-1.0)	0.438
Change in 48-h VT events	0.0 (-8.0 to 0.0)	0.0 (-2.0 to 0.0)	0.0 (-11.0 to 0.0)	0.377
Change in 48-h shocks	0.0 (-2.0 to 0.0)	0.0 (-2.0 to 0.0)	0.0 (-1.8 to 0.0)	0.812
Change in 24-h VT events	-5.0 (-16.0 to 0.0)	-2.0 (-5.8 to -0.25)	-10.0 (-28.0 to 0.0)	0.020
Change in 24-h shocks	-1.5 (-6.0 to 0.0)	-2.0 (-6.5 to 0.0)	-1.0 (-6.0 to 0.0)	0.118
Drug used				<0.001
Bupivacaine	83 (70.9)	15 (30.6)	68 (100.0)	
Ropivacaine	33 (28.2)	33 (67.3)	0 (0.0)	
Lidocaine	1 (0.9)	1 (2.0)	0 (0.0)	
Bilateral block	21 (17.9)	19 (38.8)	2 (2.9)	<0.001
Repeat injection	53 (45.3)	22 (44.9)	31 (45.6)	0.242
Complications				
Brachial plexus block	1 (0.9)	1 (2.0)	0 (0.0)	—
Recurrent laryngeal nerve block	1 (0.9)	1 (2.0)	0 (0.0)	—
In-hospital death	47 (40.1)	27 (55.1)	20 (29.4)	<0.001
Values are median (Q1-Q3) or n (%). Values in bold are statistically significant. Abbreviations as in Table 1.				

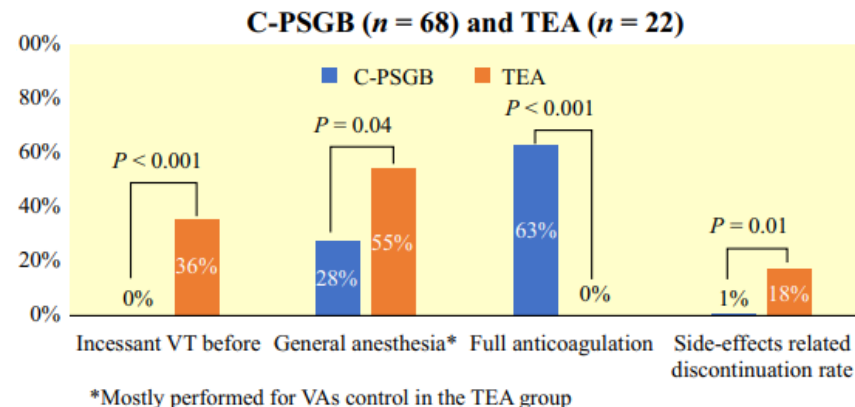
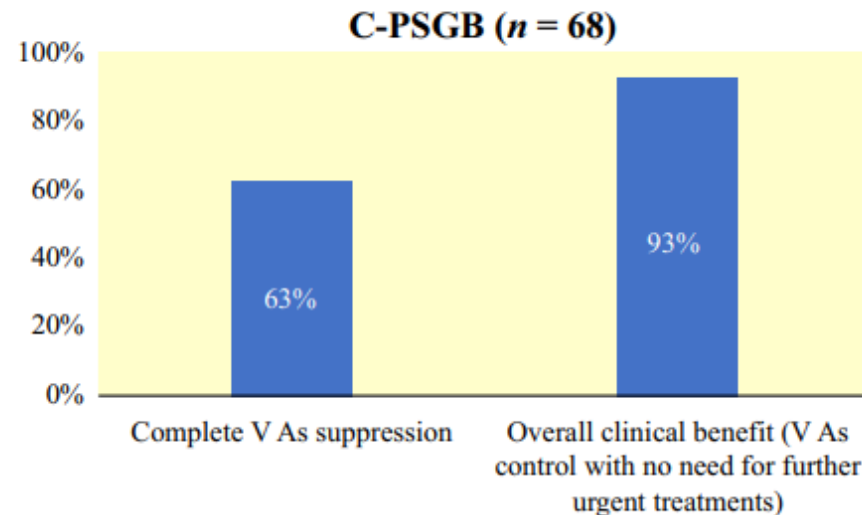
Continuous stellate ganglion block for ventricular arrhythmias: case series, systematic review, and differences from thoracic epidural anaesthesia

Veronica Dusi ^{1,2,*†}, Filippo Angelini ^{2†}, Enrico Baldi ^{3†}, Antonio Toscano ⁴, Carol Gravinese ², Simone Frea ², Sara Compagnoni ⁵, Arianna Morena ^{1,2}, Andrea Saglietto ², Eleonora Balzani ⁶, Matteo Giunta ⁴, Andrea Costamagna ⁴, Mauro Rinaldi^{6,7}, Anna Chiara Trompeo⁴, Roberto Rordorf ³, Matteo Anselmino ^{1,2}, Simone Savastano ^{3‡}, and Gaetano Maria De Ferrari ^{1,2‡}

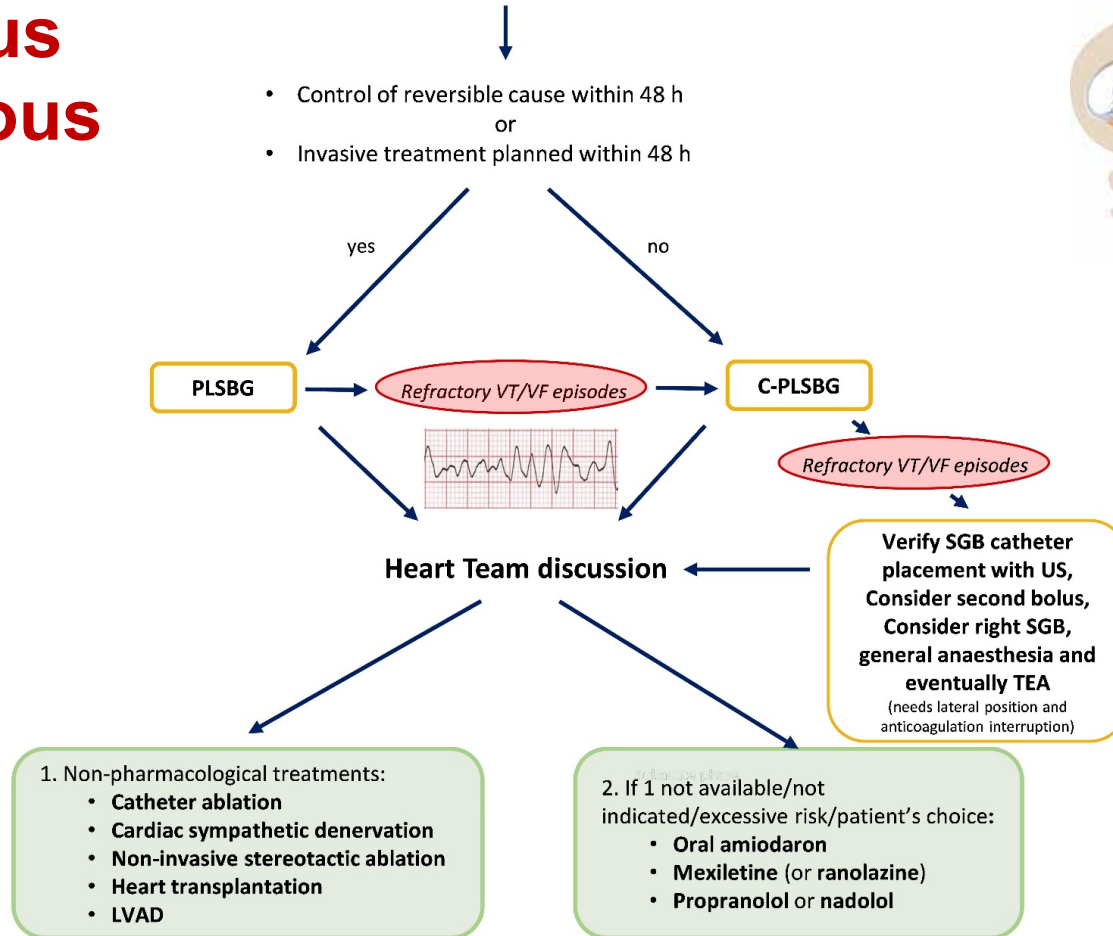
Largest case series ever described (26 patients, 88% male, 60±16 years, all with advanced structural heart disease, LVEF 23±11 %: 32 C-PSGBs performed, median duration 3 days, IQR 1–7)

All previously published data plus ours were analyzed: 61 patients received 68 C-PSGBs and 22 TEA for refractory VAs. No major complications occurred. efficacy was not compared due to patients' heterogeneity.

Ropivacaine and lidocaine were the most used drugs for C-PSGB, and the available data support a starting dose of 12 mg/h and 100 mg/h, respectively



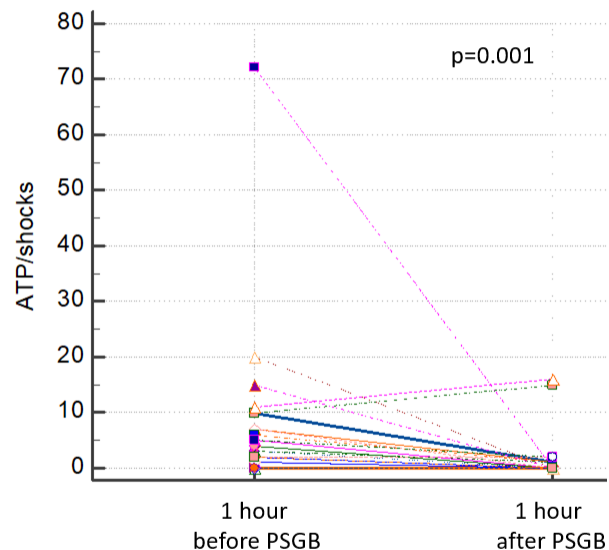
PSGB: bolus or continuous infusion??





Comparison of the number of treated arrhythmic episodes in the hour before and after PSGBs in the three groups

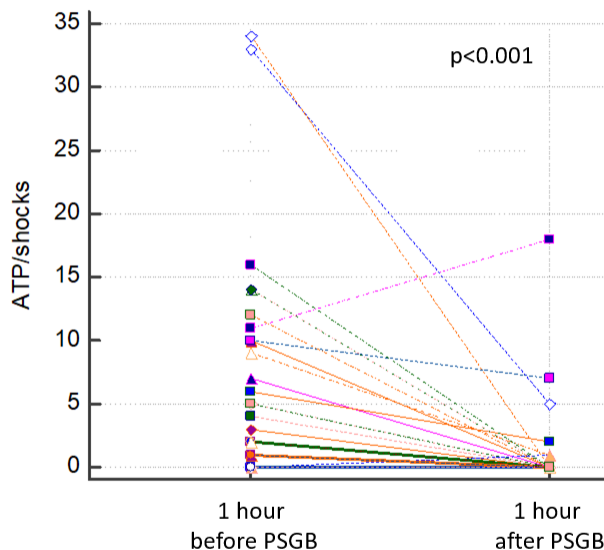
A Slow-VT ($F_c \leq 160$ bpm)



1 (IQR:0-4.5) vs 0 (IQR:0-0)

82% free from recurrences

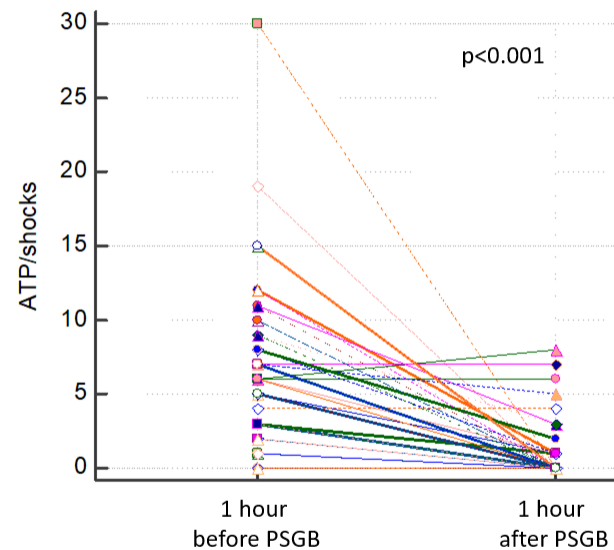
B Fast-VT ($F_c > 160$ bpm)



5 (IQR:0-6.5) vs 0 (IQR: 0-0)

86% free from recurrences

C Ventricular fibrillation (VF)



5 (IQR:2-8) vs 0 (IQR:0-1)

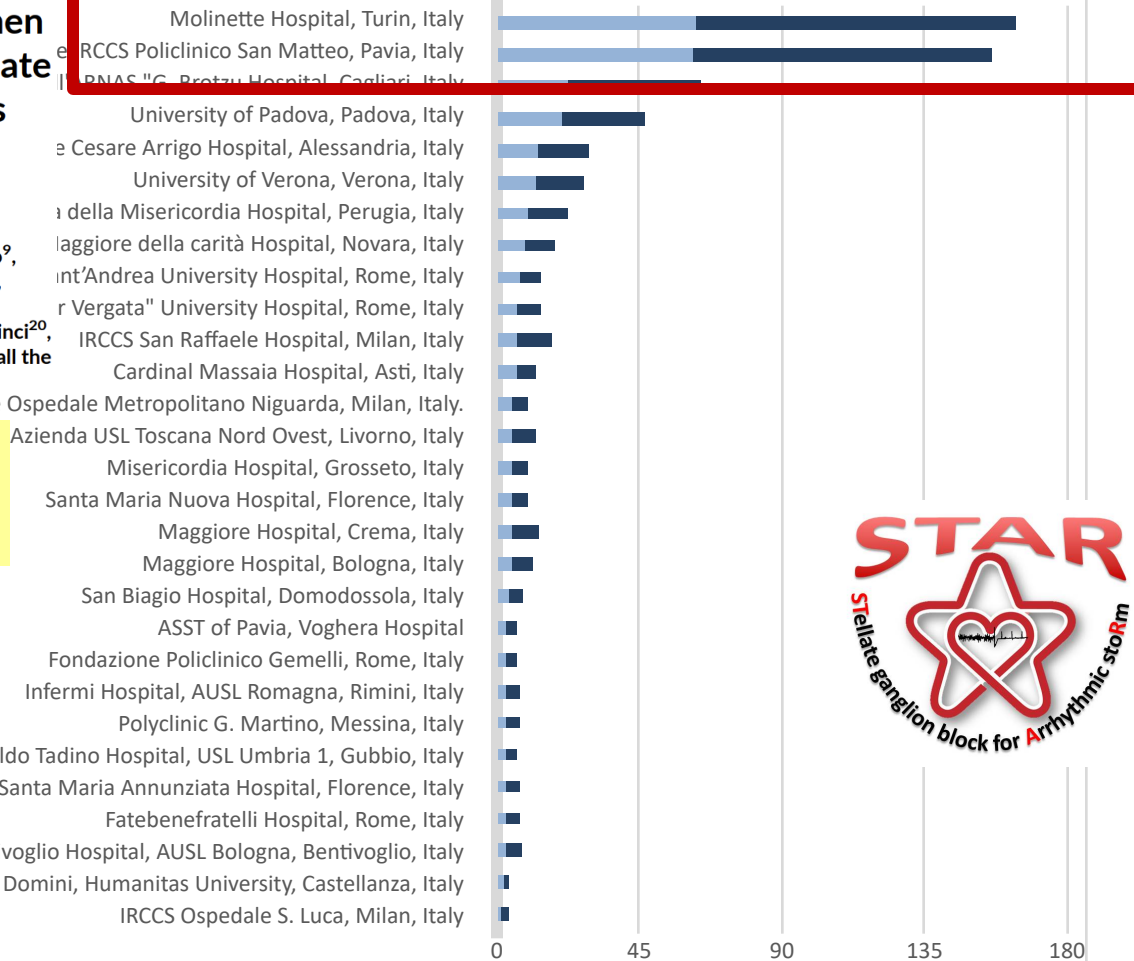
73% free from recurrences

Impact of single vs. dual local anaesthetic regimen on the efficacy and safety of percutaneous stellate ganglion block for electrical storm: new insights from the STAR study

Alessia Currao ^{1,2}, Veronica Dusi^{3,4}, Matteo Casula ⁵, Raimondo Pittorru ⁶, Enrico Baldi ¹, Filippo Angelini^{3,4}, Giulio Binaghi⁵, Federico Migliore ⁶, Edoardo Gandolfi⁷, Giacomo Mugnai⁸, Domenico Tavella⁸, Francesco Notaristefano⁹, Matteo D'Ammando⁹, Gabriele Dell'Era¹⁰, Roberta Falcetti¹¹, Giuseppe Sangiorgi¹², Giuseppe D'Angelo¹³, Marco Scaglione¹⁴, Claudia Colombo¹⁵, Enrica Talini¹⁶, Francesco De Sensi ¹⁷, Alessandro Paoletti Perini¹⁸, Enrico Chieffo¹⁹, Valeria Carinci²⁰, Simone Persampieri²¹, Gaetano Maria De Ferrari ^{3,4} and Simone Savastano ^{1,*}; all the STAR study group

- 298 patients, 422 total PSGBs
- 29 centres
- From July 1st 2017, to October 35th 2025.

The combination of two LAs—typically pairing a fast-acting with a long-acting—resulted in a significantly higher rate of complete VA suppression at 1 h (85% vs. 70%, $P < 0.01$) and 3 h (78% vs. 67%, $P = 0.02$) compared with the single LA regimen.





Our experience: PSGB Molinette Hospital, 06 2026



Bolus: 5 mL 2% lidocaine (100 mg)+ 10 mL 1% ropivacaine (100 mg)

Continuous infusion (epidural catheter)

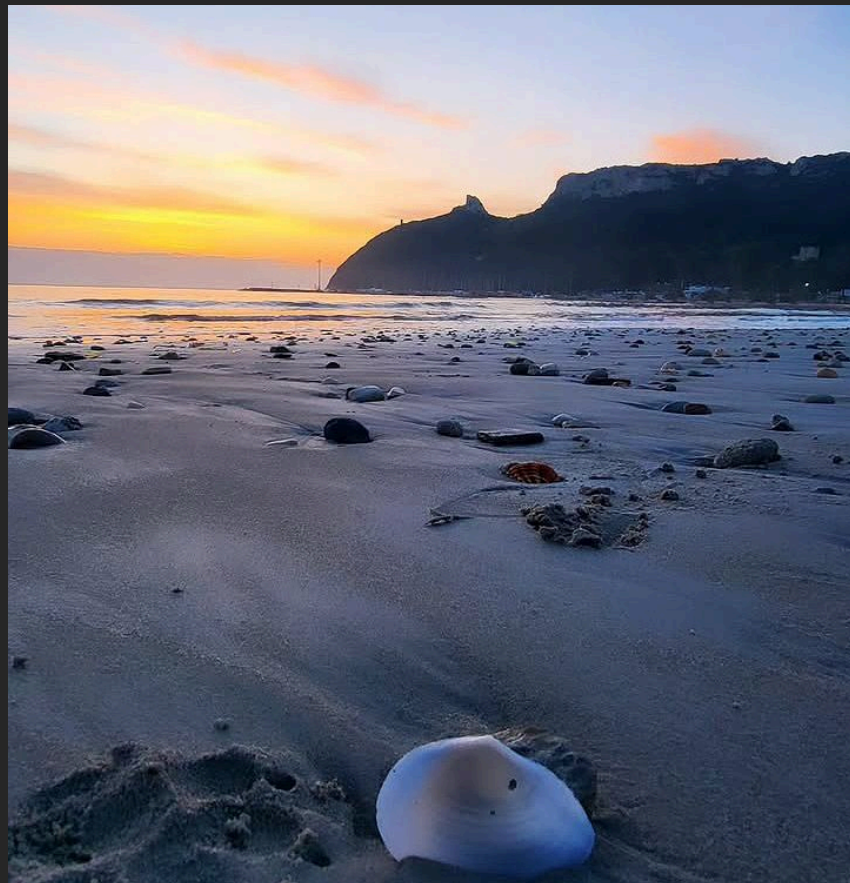
- Ropivacaine 0.2% starting at 6 ml/h=12 mg/h, and up to 12 ml/h=24 mg/h (if needed).
- Lidocaine 1.5%, starting at 75-100 mg/h (if planned VT ablation)

Need for additional urgent antiarrhythmic strategies: 4/193 cases, 2% (IABP, urgent pacing, urgent PCI post CABG, intubation and ECMO)

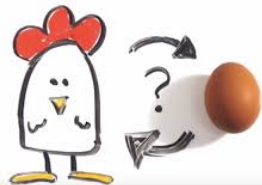
Major Complications: 2/193 (1%), LAST (respiratory and neurological depression)

Minor complications: 36/193 (19%), mostly transient weakness of the left arm and transient hoarseness/dysphagia.

Patient characteristics, N = 125 pts	N, %
Male	110, 88%
Mean age	65 ± 13
- Acute myocarditis	2, 2%
- Takotsubo syndrome	3, 3%
- STEMI/NSTEMI	28, 22%
- ICM/NICM	44/48, 35/38%
Mean LVEF	26 ± 13%
Procedural characteristics, N=193	N, %
Indication electrical storm/refractory VT	134, 69%
Indication high risk of major VT/VF	31, 16%
Indication SV arrhythmias	28, 14%
Lateral (US-guided) technique	192, 99%
Continuous Infusion (CI)	46, 24%
Median duration of the CI (days)	4 (2-7)
Intra-operative block (cardiac surgery)	3, 2%
Left-side only block	191, 99%



How to manage patients with drugs and/or VT recurrences after VT ablation in the setting of SHD ?



- 1) Do I REALLY need to treat primarily VT (vs heart failure vs both)?
- 2) When is an invasive VT ablation redo not the best option (at least temporarily)?
- 3) What's the mechanism of VT recurrences and what's the expected substrate evolution rate?



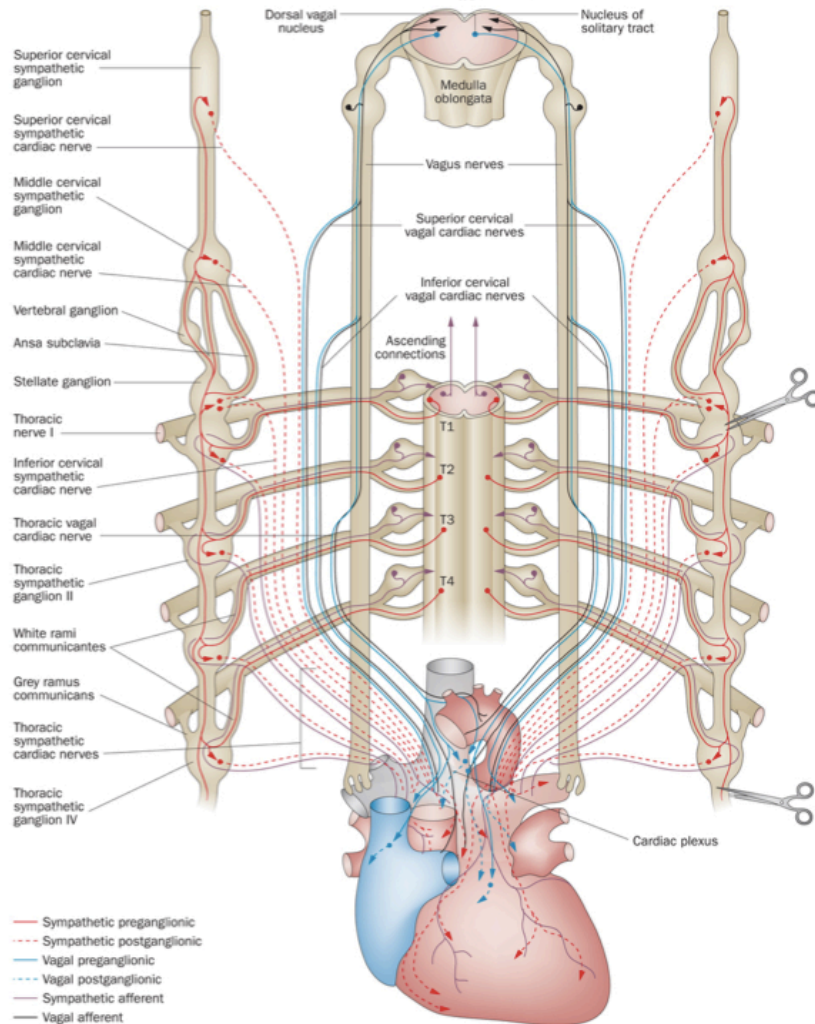
Multidisciplinary heart team discussion

(eligibility for permanent LVAD and/or

HTx assessment)



Left Cardiac Sympathetic Denervation (LCSD)



- Resection and removal of the lower third to half of the stellate ganglion (T1) and T2-T4 thoracic ganglia
- Pre-ganglionic denervation (no reinnervation)
- Right side innervation is preserved (no post-denervation supersensitivity, no impact on chronotropism)
- Affects the heart and the skin

LCSD in channelopathies

LQTS

Left Cardiac Sympathetic Denervation in the Management of High-Risk Patients Affected by the Long-QT Syndrome

Peter J. Schwartz, MD; Silvia G. Priori, MD, PhD; Marina Cerrone, MD; Carla Spazzolini, PhD; Attilio Odero, MD; Carlo Napolitano, MD, PhD; Raffaella Bloise, MD; Gaetano M. De Ferrari, MD; Catherine Klersy, MD, MS; Arthur J. Moss, MD; Wojciech Zareba, MD; Jennifer L. Robinson, MS; W. Jackson Hall, PhD; Paul A. Brink, MD; Lauri Toivonen, MD; Andrew E. Epstein, MD; Cuilan Li, MD; Dayi Hu, MD

(*Circulation*. 2004;109:1826-1833.)

Left Cardiac Sympathetic Denervation for Long QT Syndrome

50 Years' Experience Provides Guidance for Management

Veronica Dusi, MD, PhD,^{1,2} Luigi Pugliese, MD,³ Gaetano M. De Ferrari, MD,¹ Attilio Odero, MD,¹ Lia Crotti, MD, PhD,^{1,4,5,6} Federica Dagradi, MD,³ Silvia Castelletti, MD,⁴ Alessandro Vicentini, MD,¹ Roberto Rordorf, MD,¹ Cuilan Li, PhD,¹ Maria Shkolnikova, MD,¹ Carla Spazzolini, DVM, MS,¹ Peter J. Schwartz, MD^{1,2}

J Am Coll Cardiol EP 2022;8:281-294



- 2015 ESC GL: Class IIa for symptoms (after ICD)
- 2017 AHA/ACC/HRS GL: Class I for symptoms (± ICD)
- 2017 AHA/ACC/HRS GL: Class IIb for high-risk asymptomatic
- 2022 ESC GL: Class I for symptoms (after ICD)

CPVT

Clinical Management of Catecholaminergic Polymorphic Ventricular Tachycardia

The Role of Left Cardiac Sympathetic Denervation

Gaetano M. De Ferrari, MD*; Veronica Dusi, MD*; Carla Spazzolini, DVM, MS*; J. Martijn Bos, MD, PhD*; Dominic J. Abrams, MD, MRCP; Charles I. Berul, MD; Lia Crotti, MD, PhD; Andrew M. Davis, MB, BS, MD; Michael Eldar, MD; Maria Kharlap, MD; Asaad Khoury, MD; Andrew D. Krahn, MD; Antoine Leenhardt, MD; Christopher R. Moir, MD; Attilio Odero, MD; Louise Olde Nordkamp, MD; Thomas Paul, MD; Ferran Rosés i Noguera, MD; Maria Shkolnikova, MD; Jan Till, MD; Arthur A.M. Wilde, MD; Michael J. Ackerman, MD, PhD†; Peter J. Schwartz, MD†

Circulation. 2015;131:2185-2193

How Many CPVT Patients Need an ICD?

The Impact of Left Cardiac Sympathetic Denervation

J. Martijn Bos, MD, PhD,^{1,2} Veronica Dusi, MD, PhD,^{1,2} Dana Viskin, MD,³ Thomas Paul, MD,⁴ Ferran Rosés i Noguera, MD, PhD,^{1,2} Jan Till, MD,⁵ Asaad Khoury, MD,⁶ Zahra Emkanjoo, MD,¹ Mohammad Dalili, MD,¹ Sergey A. Termosov, MD,¹ Vera V. Berezinskaya, MD, PhD,¹ Roberto Rordorf, MD,¹ Giulia Musu, MSc,¹ Lia Crotti, MD, PhD,^{1,2} Stephanie F. Polites, MD,³ Gaetano M. De Ferrari, MD, PhD,¹ Luigi Pugliese, MD,¹ Michael J. Ackerman, MD, PhD,² Peter J. Schwartz, MD¹

J Am Coll Cardiol EP 2026, in press



- 2015 ESC GL: Class IIb for symptoms (after ICD)
- 2017 AHA/ACC/HRS: Class I for symptoms (± ICD)
- 2022 ESC GL: Class IIa for symptoms (after ICD)

Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: Intermediate and long-term follow-up

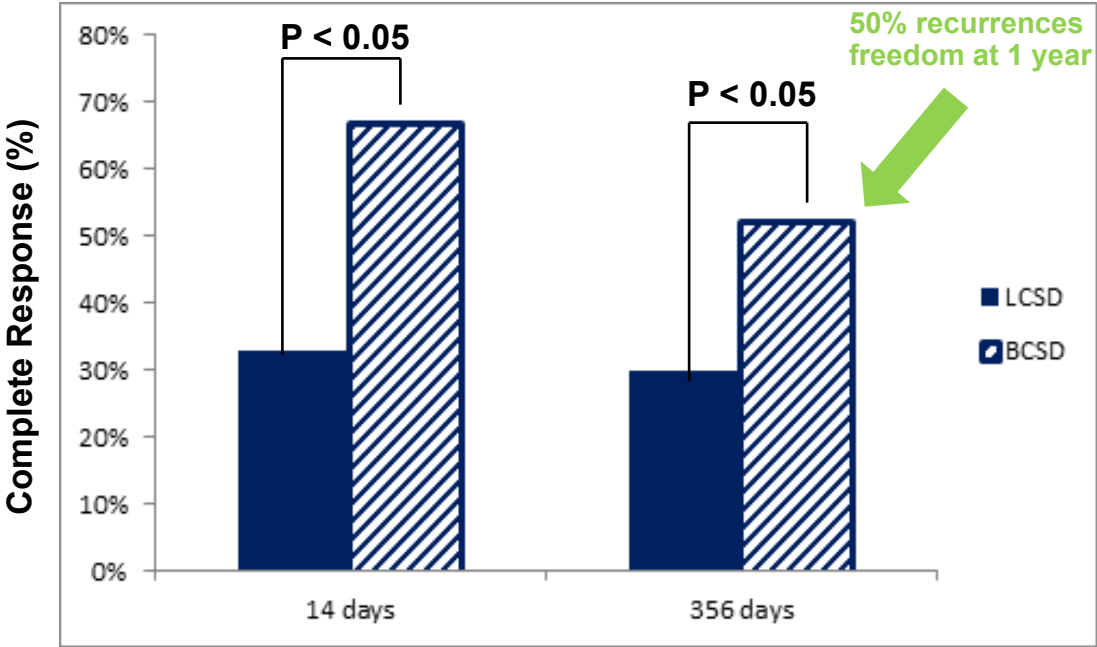
Marmar Vaseghi, MD, MS,* Jean Gima, RN, MSN, NP,* Christopher Kanaan, BS,* Olujimi A. Ajijola, MD, PhD,* Alexander Marmureanu, MD,*† Aman Mahajan, MD, PhD,*‡ Kalyanam Shivkumar, MD, PhD, FHRS*

From the *UCLA Cardiac Arrhythmia Center, †Division of Cardiothoracic Surgery, and ‡Department of Anesthesiology, UCLA Health System, Los Angeles, California.

Patient Characteristics	Bilateral CTS N=26	Left CTS N=15
Male	22(85%)	13 (87%)
Cardiomyopathy		
ICM	4 (15%)	5 (33.3%)
NICM	14 (54%)	8(53.3%)
HCM	1(3.8%)	2(13.3%)
Sarcoid	2(7.6%)	
Chagasic	1 (3.8%)	
ARVC	1 (3.8%)	
Valvular	1(3.8%)	
TGA (+ infarct)	1(3.8%)	
None (idopathic VF)	1(3.8%)	
Type of VT:		
PMVT/VF	6 (23%)	2(13.3%)
MMVTS	20(77%)	13 (86.7%)
Ejection Fraction (%)	32±13	29±13
Pulmonary artery pressure	38±12.5 mmHg	39±9.8 mmHg
Median # of VT ablations	2 (0-5)	2 (0-4)

n=41 ps, 40 with SHD, 66% BCSD, 34% LCSD

CSD For VT & VF: Dose-response



Predictors of recurrences after CSD

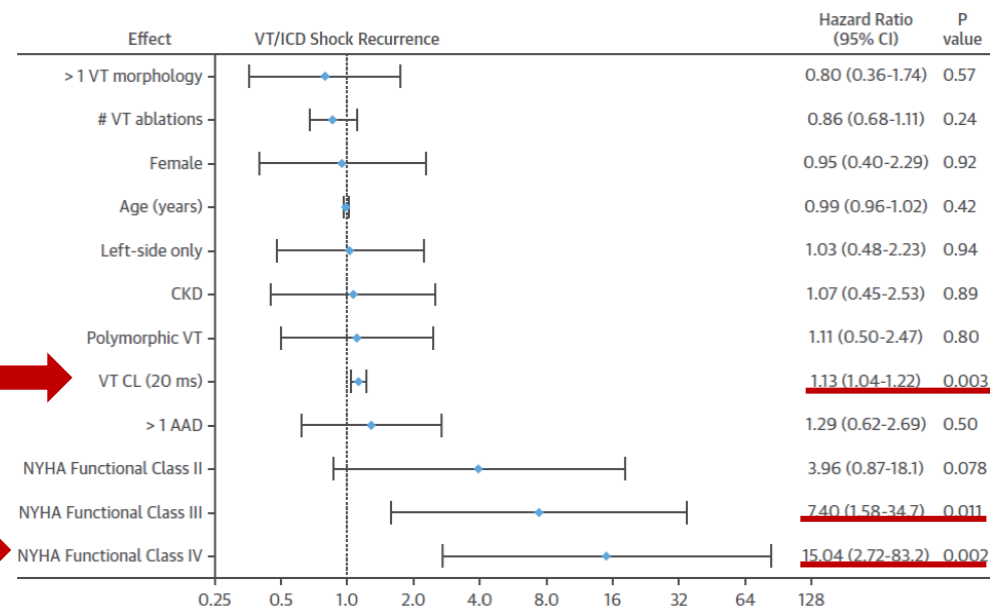
-Retrospective study (5 non European centers)

-121 pts with SHD (mean 55 yrs, mean LVEF 30%), 71% NICM

-19% had LCSD, 81% BCSD

-CSD reduced the median burden of ICD shocks from 10 in the year before 0 in the year after ($p < 0.01$).

FIGURE 5 Pre-Procedural Characteristics Associated With VT/ICD Shock Recurrence After CSD

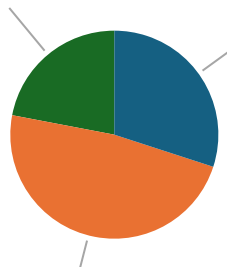


Advanced NYHA functional class and longer VT cycle length (CL) predicted recurrence of sustained VT and ICD shock. AAD = antiarrhythmic drug; CI = confidence interval; CKD = chronic kidney disease; other abbreviations as in [Figures 1, 3, and 4](#).

Cardiac sympathetic denervation for refractory ventricular arrhythmias in patients with inherited cardiomyopathies ^e

Abdullah Sarkar, MD, MSc,¹ Stephany L. Pisciotti, MD,² Carlos Tapias, MD,² Fabrizio Assis, MD,³ Harikrishna Tandri, MD, FHRS,⁴ Jessica Wang, MD, PhD,⁵ Jonathan Chrispin, MD,⁶ Faisal Syed, MBChB,⁷ Jane Yanagawa, MD,⁸ Jay Lee, MD,⁸ Jason S. Bradfield, MD, FHRS,¹ Kalyanam Shivkumar, MD, PhD, FHRS,¹ Luis C. Saenz, MD, FHRS,² Olujimi A. Ajijola, MD, PhD, FHRS¹

● HCN ● AR/LVC ● DCM

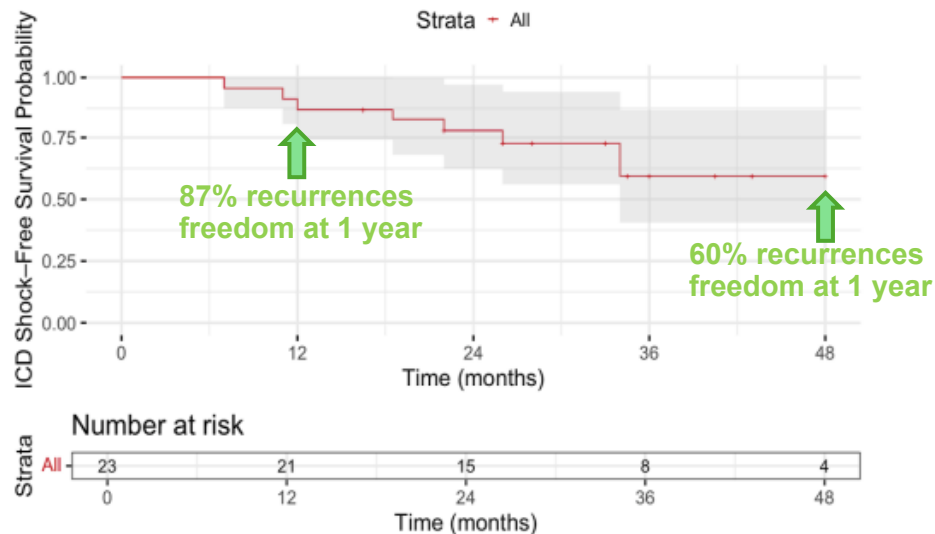


Among 11 AR/LVC: 4 PKP2, 3 LMNA, 4 no C3/C4

Table 1 Baseline patient demographics and clinical characteristics

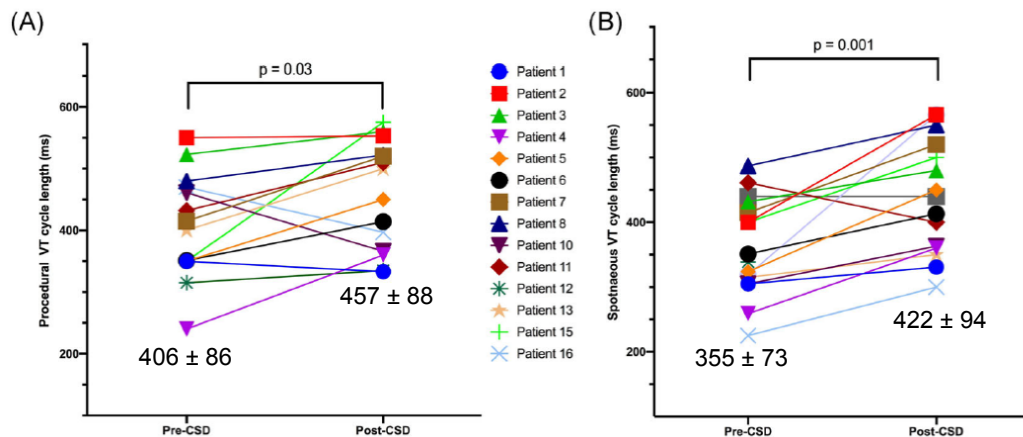
Variable	Overall (N = 23)	Success (n = 11)	VA recurrence (n = 12)	P value
Age	45.1 ± 15.3	44.0 ± 15.0	46.1 ± 16.2	.753
Sex (male)	69.60	81.80	58.30	.442
LVEF	41.9 ± 14.9	40.9 ± 16.1	42.9 ± 14.4	.75
RVEF	39.6 ± 11.4	39.6 ± 11.9	39.6 ± 11.5	.991
NYHA (class 3)	34.80	36.40	33.30	1
HTN	30.40	27.30	33.30	.951
DM	4.30	9.10	0.00	.56
AF	39.10	36.40	41.70	1
CKD	13.00	9.10	16.70	.51
COPD	4.30	0.00	8.30	.544
Follow-up (mo)	44.8 ± 33.4	33.1 ± 11.1	55.5 ± 43.0	.11

B ICD Shock-Free Survival Curve at 4 years



Impact of CSD on VT cycle length

- 16 pts (94% males, 54 ± 13 years, 88% with NICM, LVEF $39 \pm 10\%$) who underwent VT ablation (mean 2 procedure/pt before the index one) both before and after CSD (94% BCSD) were studied.
- The clinical VT cycle length significantly increased after CSD both for spontaneous and induced VT
- **The proportion of mappable, hemodynamically stable VTs increased from 35% during pre-CSD RFA to 58% during post-CSD RFA ($p = 0.038$).**



Cardiac sympathetic denervation Usage To prevent malignant Ventricular Tachyarrhythmias: CUT-VT Study

- ✓ Type of study: multicentric observational prospective and retrospective study
- ✓ Primary aim: standardized assessment of CSD antiarrhythmic efficacy and safety in cardiomyopathies and structural heart disease.
- ✓ Inclusion criteria: patients with clinically and/or genetically diagnosed cardiomyopathy or structural heart disease, with refractory VT/VF (≥ 1 episodes) despite optimized HF and antiarrhythmic therapy (not necessarily including amiodarone, to be justified) and at least one VT ablation attempt, unless not indicated, unfeasible or refused.
- ✓ Coordinating Center: Citta della Salute e della Scienza Hospital, Molinette, University of Turin.
- ✓ 62 pts enrolled so far, 33 Molinette, **LVEF $33 \pm 12\%$, 30% NYHA ≥ 3 , 86% NICM, 76% with ES, 60% previous VT ablation**



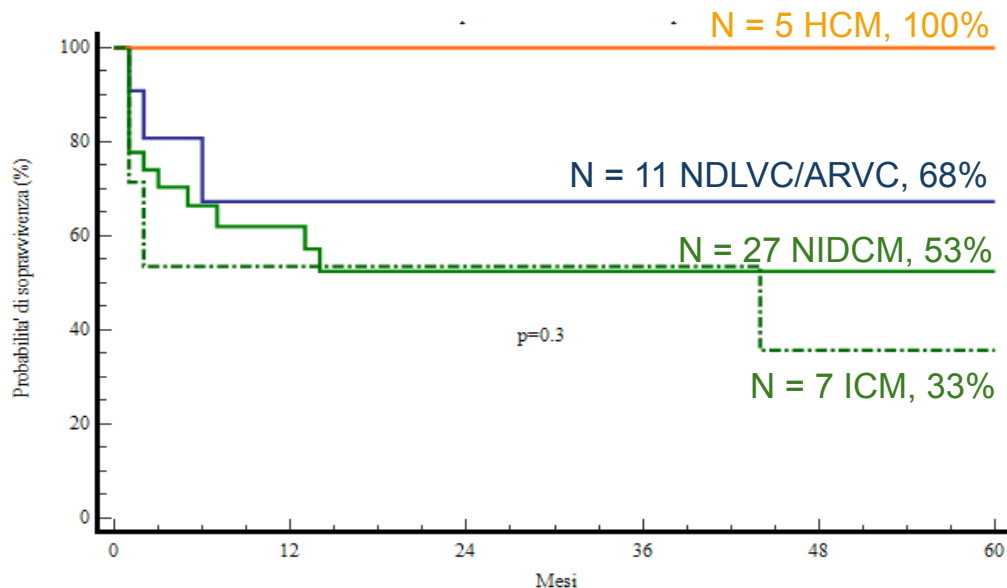
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CUT-VT, ES free survival according to the type of CMP, n=50



Multivariate Cox analysis: predictors of ES after CSD

Variabile	HR	p value	IC 95%
NYHA $\geq$ 3	4.67	0.006	1.57-13.93
LCSD	3.04	0.088	0.85-10.91
VT $\geq$ 200 bpm	0.33	0.033	0.11-0.91

N=50 (20 events). c-index: 0.816, 95% CI: 0.75 - 0.89.

**No ICD end of treatment conditions occurred after CSD
(vs 22% before, p<0.01)**

Median FU 21 months (IQR 9-41)

VT in SHD on GDMT for HF

First line

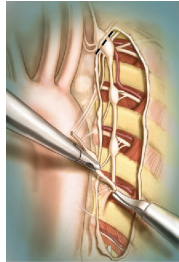
Amiodarone ±
Mexiletine

Sotalol or nadolol +
flecainide

Invasive VT ablation (including epicardial
access when indicated)

“Interventional
Second lines”

CSD, GL approved



Favor in case of:

- Polymorphic VT/VF
 - MMVT >150 bpm
 - Multiple VT morphology
 - Young patients
 - Low NYHA functional Class
-
- Can slow down the VT and improve the response to a subsequent invasive/non-invasive VT ablation
-
- Ongoing Italian Registry (CUT-VT: PI: GM De Ferrari)

**Multidisciplinary
VT team and
Heart team
discussion**

**Non-invasive stereotactic
VT ablation (STAR), still investigational**

Favor in case of:

- Single, very stable, slow MMVT, particularly in fragile pts and/or with no indication for HTx
 - High NYHA functional Class/procedural risk
 - Only within registries/prospective studies
-
- Ongoing Italian Registry within the STOPSTORM European Consortium, (POPSTORM, PI: GM De Ferrari)



WELCOMED TO JOIN!!

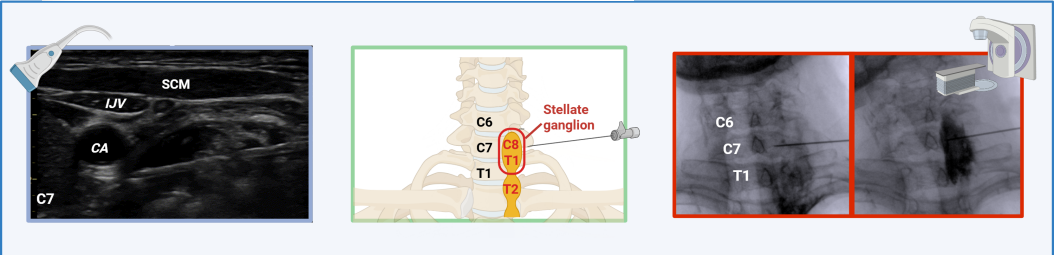
Email: veronica.dusi@unito.it
Twitter: [@veronica_dusi](https://twitter.com/veronica_dusi)



Percutaneous Stellate Ganglion Ablation as a Novel Bail-Out Strategy for Refractory Ventricular Arrhythmias

A Clinical Case Series

Filippo Angelini, MD,^a Paolo Cotogni, MD,^{b,c} Marco Minniti, MD,^b Benedetta De Guidi, MD,^{a,d} Arianna Morena, MD,^{a,d} Simone Frea, MD,^a Antonio Toscano, MD,^e Maurizio Berardino, MD,^c Gaetano Maria De Ferrari, MD,^{a,d} Veronica Dusi, MD, PhD^{a,d}



Three sequential 90-second lesions were created (50 °C, 55 °C, and 60 °C). The IonicRF Generator (Abbott) provided real-time temperature, impedance, voltage, and treatment duration feedback.

Who? • Frail, CMP, refractory VTs • Prohibitive anesthesiological risk for BCSD • Not good candidate at present for VT ablation	Why and what to expect? To favourably modify the clinical expression of refractory VTs aiming at: • Safety: low/null risk of serious complication • ↑ VT cycle lenght • ↑ ATP response • ↑ VT ablation candidacy/ response • ↑ Freedom from EOT • ↓ of ES and ICD shocks • ↑ QoL	What's next? • Unravel uncertainty on target hit, lesion completeness and durability • Assess long term pro arrhythmic risk (reinnervation from the other ganglia?) • Long-term studies needed
What is it? • Minimally invasive bedside neuromodulation strategy • Continuous RF to achieve long-lasting T1-block		

Why performing neuronal sympathetic block in patients with or at high risk of life-threatening VT/VF?

Acute phase stabilization

Acute effects (PLSGB):

- ✓ Acute stabilization (eg ADHF) or bridge therapy (to LVAD, transplant, VT ablation, CSD, etc.); consider continuous infusion if the trigger/precipitating factor lasts longer than 48 hours
- ✓ May dramatically reduce the need for general anesthesia and mechanical circulatory support
- ✓ Good risk/benefit ratio (no impact on BP, chronotropism, or

Subacute/Chronic phase

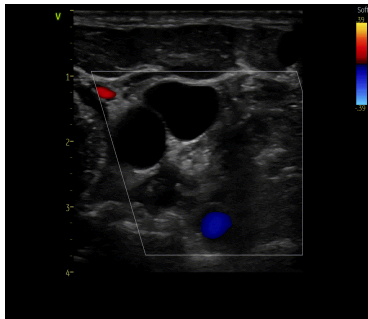
Chronic Effects (CSD)

- ✓ Reduces the risk of EOT conditions and of ES occurrence/recurrence
- ✓ Reduces the risk of ICD shocks (appropriate and even inappropriate)
- ✓ Reduces the risk of MMVT/pVT/VF recurrences and potentially even the PVC burden
- ✓ May slow down the VT cycle length, improve ATP response and the effectiveness of a subsequent VT ablation

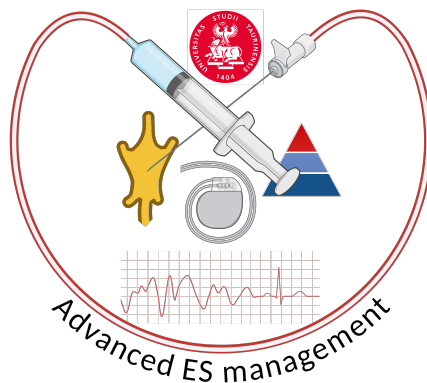
Molinette: programma avanzato di neuromodulazione a scopo antiaritmico:

- **PLSGB farmacologico: 125 pazienti, 193 procedure, circa 70% per storm aritmico**
- **PLSGB con RF: 3 pazienti, 5 procedure, non candidabili a BCSD tradizionale**
- **CSD in SHD e angina refrattaria (CUT-VT Registry Torino-Pavia: 62 pazienti (57 BCSD, 5 LCSD).**
- **LCSD per canalopatie (LQTS, CPVT, FV refrattaria)**

Gestione avanzata dello storm aritmico in terapia intensiva e blocco percutaneo del ganglio stellato



8.9 ECM



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Segreteria UniTo Formazione ECM

Tel. 011 6709549

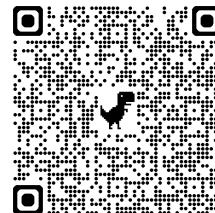
Email: eventiecm.dam@unito.it

SAVE THE DATE:

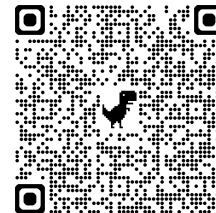
- **5 ottobre 2026**
- **16 novembre 2026**

Per iscriversi

05/10/2026



16/11/2026



Responsabili Scientifici: Veronica Dusi, Filippo Angelini, Gaetano Maria De Ferrari



THANK YOU FOR YOUR ATTENTION



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