PLACE

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

ROMA
Centro Congressi
di Confindustria
Auditorium
della Tecnica

9ª Edizione

30 Settembre
1 Ottobre
2022

ARIMOLOGIA CLINICA ED INTERVENTISTICA

Fibrillazione atriale ed imbalance simpato-vagale. Dalla scelta della terapia antiaritmica, ai pazienti con sincope e all'ablazione dei gangli

MARCO REBECCHI

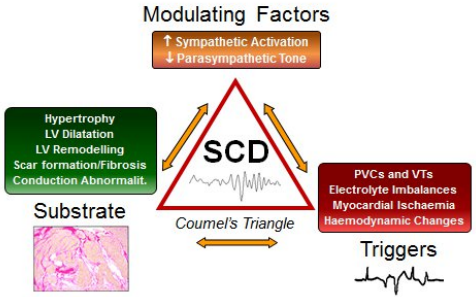
ARITMOLOGIA CLINICA ED INTERVENTISTICA
POLICLINICO CASILINO
ROMA

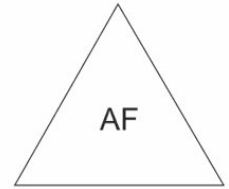
Europace (2004) 6, 464e 465

ELSEVIER

EUROPEAN SOCIETY OF CARDIOLOGY

Philippe Coumel: a founding father of modern arrhythmology*





Extrasystoles
Tachycardias
Atrial flutter

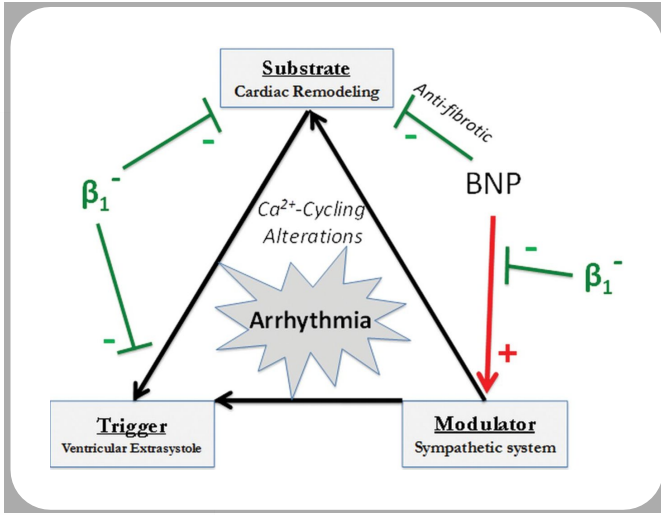
TRIGGERING FACTOR

MODULATING FACTORS

Autonomic nervous system
– sympathetic
– parasympathetic

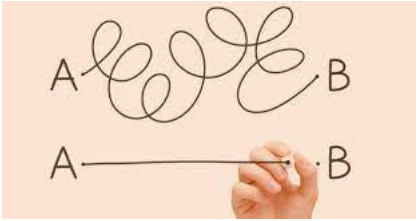
SUBSTRATE

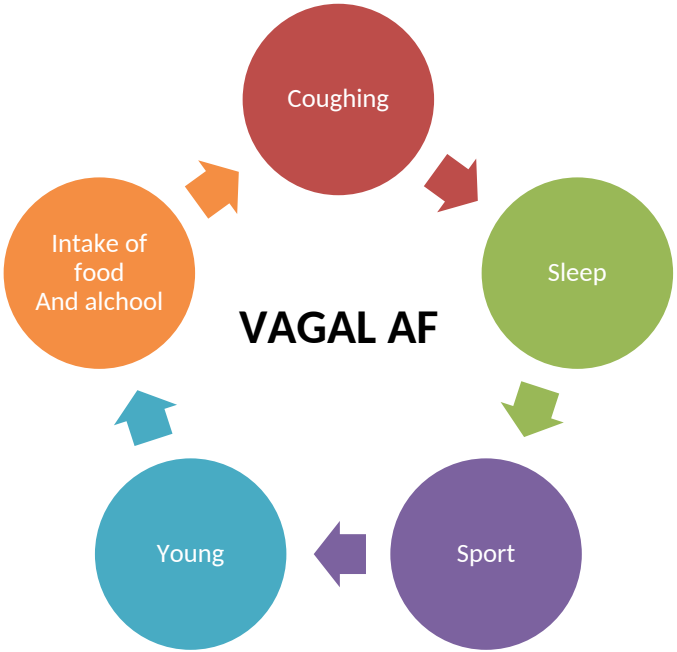
– ischemia
– hypertension
– heart failure
– valvular heart disease
– cardiomyopathy
– none (idiopathic AF)

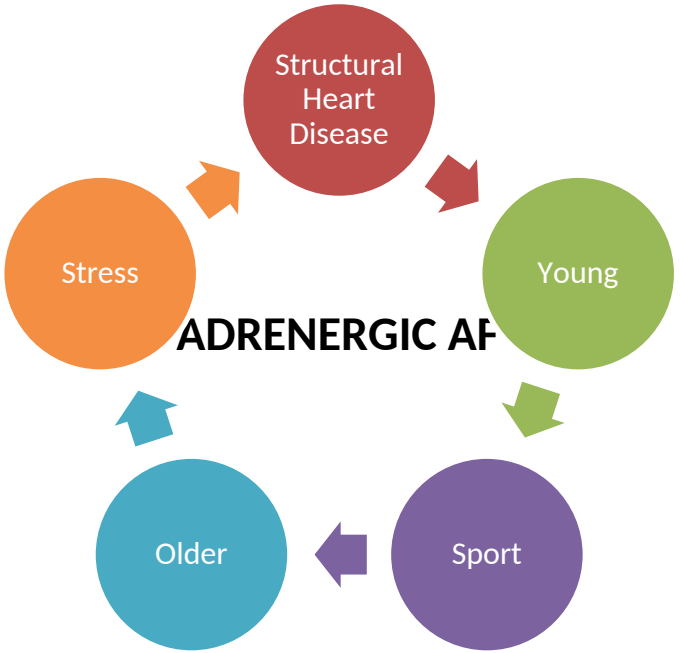


Vagal and adrenergic AF

Too much simple definition for a very complex mechanism







European Heart Journal (2008) 29, 632–639
doi:10.1093/eurheartj/ehn025

Autonomic trigger patterns
treatment of paroxysmal at
from the Euro Heart Surve

Cees B. de Vos[1]*, Robby Nieuwlaat[1], Harry J.G
Jean-Yves LeHeuzey[3], Charles J. Kirchhof[4], Ale
Günter Breithardt[6], Panos E. Vardas[7], Ron Pist

Overall populatio: 495 with p
- in 91 patients (6%) a vaga
- 229 patients (15%) had ar
- 175 patients (12%) a mix

Table 2 Characteristics of patients with an adrenergic, vagal, and mixed trigger pattern

	Adrenergic trigger pattern	Vagal trigger pattern	Mixed trigger pattern	*P*-value
n	229	91	175	
Age (years)	62 ± 13	62 ± 14	62 ± 13	0.609
Female	94 (41%)	38 (42%)	73 (42%)	0.988
Body weight	81 ± 15	80 ± 19	80 ± 16	0.906
BMI	27 ± 5	28 ± 6	27 ± 5	0.484
Heart rate at inclusion (when SR) (BPM)	69 ± 13	66 ± 15	69 ± 17	0.533
Heart rate at inclusion (when AF) (BPM)	110 ± 32	109 ± 40	106 ± 29	0.390
Lone AF	35 (15%)	14 (16%)	37 (21%)	0.268
Underlying heart diseases				
Heart failure	58 (25%)	22 (24%)	34 (20%)	0.368
Coronary artery disease	69 (30%)	26 (29%)	55 (31%)	0.891
Valvular heart disease	43 (19%)	18 (20%)	28 (16%)	0.698
Mitral stenosis	16 (7%)	7 (8%)	8 (5%)	0.508
Hypertension	149 (65%)	65 (71%)	109 (62%)	0.331
Other diseases				
Thyroid disease	12 (6%)	5 (6%)	8 (5%)	0.906
Pulmonary disease	25 (11%)	6 (7%)	22 (13%)	0.322
Sick sinus syndrome	8 (3%)	7 (8%)	10 (6%)	0.261
Peripheral vascular disease	16 (7%)	2 (2%)	12 (7%)	0.220
Renal failure	12 (5%)	5 (6%)	7 (4%)	0.804
Malignancy	7 (3%)	5 (6%)	9 (6%)	0.429
Major bleeding	2 (1%)	1 (1%)	2 (1%)	0.960
Stroke/TIA	25 (11%)	8 (9%)	16 (9%)	0.796
Previous interventions				
Pharmacological cardioversion	125 (55%)	43 (47%)	98 (56%)	0.374
Electrical cardioversion	63 (28%)	22 (24%)	62 (35%)	0.107
Catheter ablation	12 (5%)	3 (3%)	17 (10%)	0.082
Pacemaker	8 (4%)	7 (5%)	0	0.542

BMI, body mass index; SR, sinus rhythm; BPM, beats per minute; TIA, transient ischaemic attack.

- Anatomical Complexity
- Pathophysiological Complexity

Available online at www.sciencedirect.com
ScienceDirect
Journal of Electrocardiology 39 (2006) S180–S183
JOURNAL OF Electrocardiology
www.elsevier.com/locate/jelectrocard

The neural basis of atrial fibrillation

Benjamin J. Scherlag, PhD,* Eugene Patterson, PhD, Sunny S. Po, MD, PhD
Cardiac Arrhythmia Research Institute, at the University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA
Received 3 May 2006; accepted 31 May 2006

➢Già da alcuni anni, diversi studi hanno mostrato l'importante ruolo del Sistema Nervoso Autonomo nella genesi della fibrillazione atriale.

➢Gli impulsi nervosi attraverso il SNA convergono verso strutture superiori localizzate nel grasso epicardio, definite plessi gangliari costituiti da gangli autonomici e nervi. Nel cuore umano sono stati ben identificati **almeno 7 PG, di cui 4 localizzati attorno all'antro delle vene polmonari**

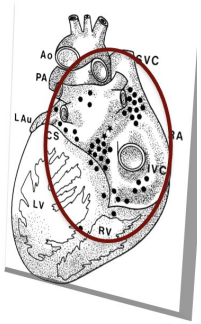
Sherlag BJ Journal of Electrocardiology 2006
Jackmann JCE 2009

Topography of cardiac ganglia in human heart

TOPOGRAPHY OF CARDIAC GANGLIA IN THE ADULT HUMAN HEART

Sanjay Singh, MSc[a]
Patricia I. Johnson, PhD[a]
Robert E. Lee, MD, PhD[a]
Emilo Orfei, MD[b]
Vassyl A. Lonchyna, MD[c]
Henry J. Sullivan, MD[c]
Alvaro Montoya, MD[c]
Hoang Tran, BS[a]
William H. Wehrmacher, MD[a]
Robert D. Wurster, PhD[a]

Published descriptions of the topography of cardiac ganglia in the human heart are limited and present conflicting results. This study was carried out to determine the distribution of cardiac ganglia in adult human hearts and to address these conflicts. Hearts obtained from autopsies and heart transplant procedures were sectioned, stained, and examined. Results indicate that the largest populations of cardiac ganglia are near the sinoatrial and atrioventricular nodes. Smaller collections of ganglia exist on the superior left atrial surface, the interatrial septum, and the atrial appendage–atrial junctions. Ganglia also exist at the base of the great vessels and the base of the ventricles. The right atrial free wall, atrial appendages, trunk of the great vessels, and most of the ventricular myocardium are devoid of cardiac ganglia. These findings suggest modifications to surgical procedures involving incisions through regions concentrated with ganglia to minimize arrhythmias and related complications. Repairs of septal defects, valvular procedures, and congenital reconstructions, such as the Senning and Fontan operations, involve incisions through areas densely populated with cardiac ganglia. The current standard procedure for orthotopic heart transplantation severs cardiac ganglia and their projections to nodal and muscular tissue. One modification of the current heart transplantation procedure, involving bicaval anastomosis, preserves atrial anatomy and the cardiac ganglia. Preservation of cardiac ganglia within the donor heart may provide additional neuronal substrate for intracardiac processing and targets for regenerating nerve fibers to the donor heart. (J Thorac Cardiovasc Surg 1996;112:943-53)



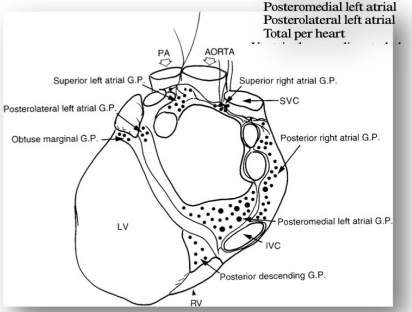
- **Para-SA nodal ganglia** are concentrated primarily **lateral to the right pulmonary veins.**
- **The para-AV nodal ganglia** are on **the epicardial surface superior to the coronary sulcus (CS) and within the interatrial septum.**
- Smaller collections of ganglia are dispersed throughout both atria, including the region superior to coronary sinus, the superior left atrial surface, and lateral to the left pulmonary veins.
- **The right atrial free wall (RA) and the adventitia of the aorta (Ao) and pulmonary artery (PA) do not contain cardiac ganglia**

J Thorac Cardiovasc Surg 1996;112:943-5

Gross and Microscopic Anatomy of the Human Intrinsic Cardiac Nervous System

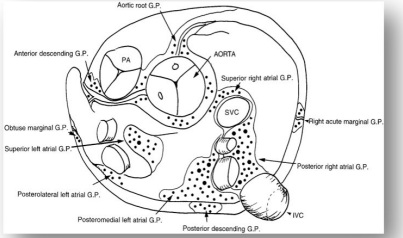
J. ANDREW ARMOUR,[3,*] DAVID A. MURPHY,[1] BING-XIANG YUAN,[3]
SARA MACDONALD,[2] AND DAVID A. HOPKINS[2]
[1]*Departments of Surgery,* [2]*Anatomy and Neurobiology, and* [3]*Physiology and Biophysics,*
Faculty of Medicine, Dalhousie University, Halifax, Nova Scotia, Canada

Ganglionic plexus	5–10 Neurons	11–50 Neurons	50–100 Neurons	100–200 Neurons	>200 Neurons	Total no. ganglia per heart
Atrial ganglionated plexuses						
Superior right atrial	19.2 ± 2.9	9.5 ± 2.8	2.2 ± 0.4	0.3 ± 0.1	0	31 ± 5
Superior left atrial	29.4 ± 5.9	19.7 ± 5.1	5.3 ± 1.9	2.2 ± 0.7	0.5 ± 0.2	56 ± 12
Posterior right atrial	90.1 ± 13.7	66.4 ± 7.6	22.8 ± 1.9	9.7 ± 0.7	4.7 ± 0.7	194 ± 22
Posteromedial left atrial	82.8 ± 13.5	56.4 ± 9.8	18.2 ± 4.1	4.5 ± 0.9	1.8 ± 0.6	161 ± 27
Posterolateral left atrial	8.2 ± 2.2	5.7 ± 1.1	1.7 ± 0.4	0.3 ± 0.1	0	16 ± 2
Total per heart						458 ± 43

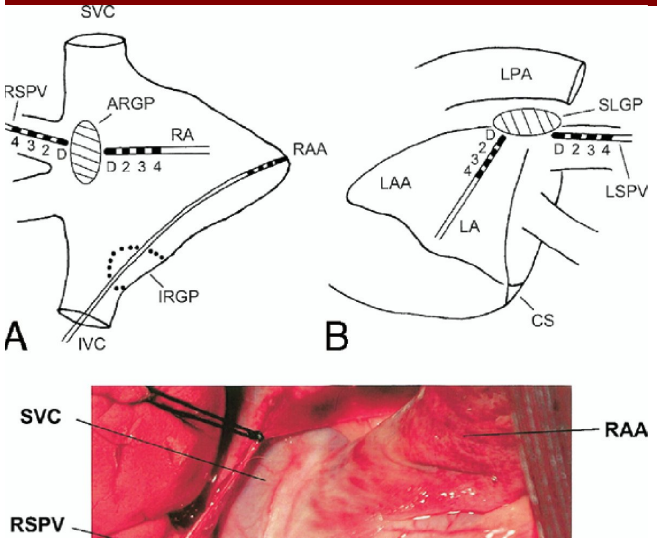


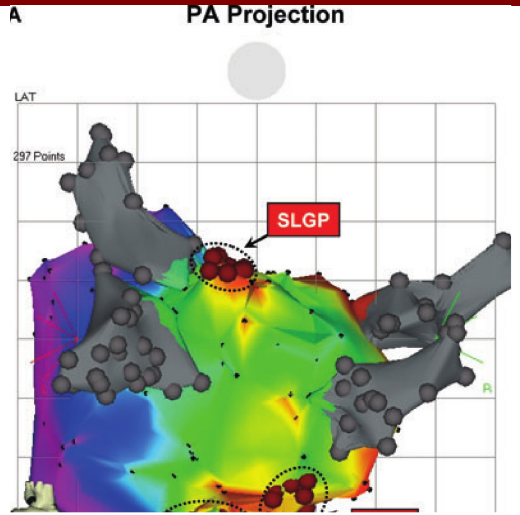
Posterior view

Superior view



The role of ganglia as integration centers in regulating AV node and SA node function

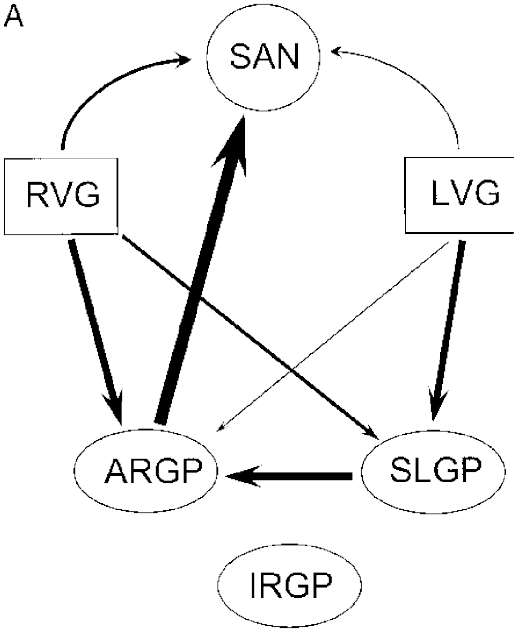




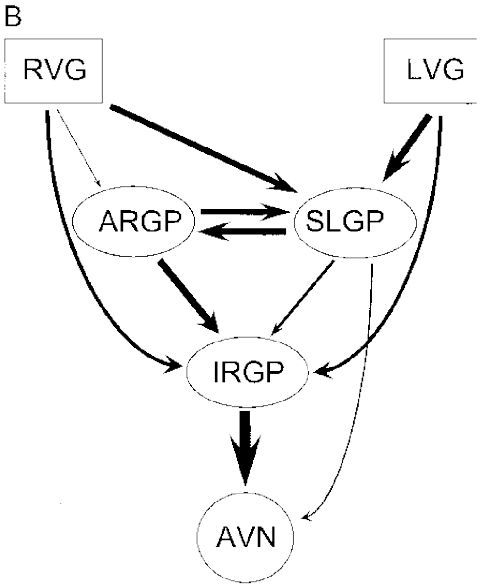
- **IRGP** *seems to be the integration center for the extrinsic ANS to innervate the AV node as ablation of IRGP completely eliminated the VR slowing response induced by vagosympathetic stimulation.*
- **ARGP and IRGP** *play a selective role in regulating SA and AV nodal function, respectively*

The role of ARGP and IRGP

GP function as “integration centers”



Modulation of sinus rate by vagosympathetic stimulation.



Modulation of ventricular rate during atrial fibrillation by vagosympathetic stimulation

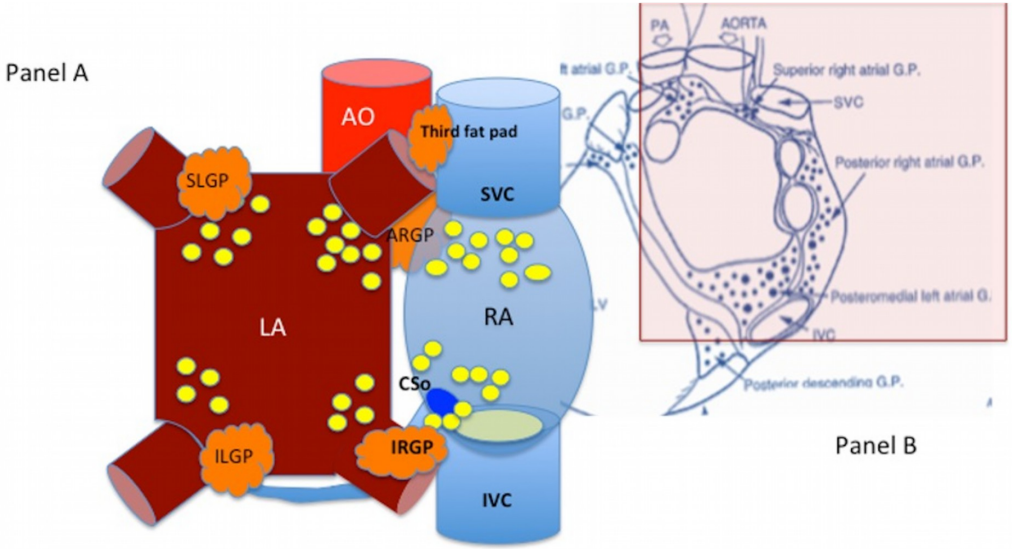
Received: 31 August 2020 | Revised: 21 December 2020 | Accepted: 15 January 2021
DOI: 10.1002/joa3.12512

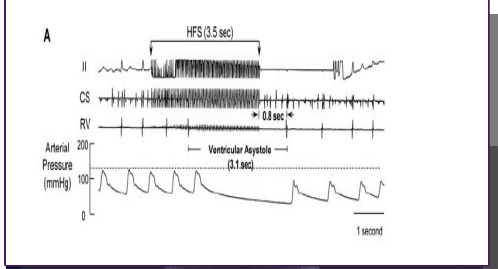
CLINICAL REVIEW

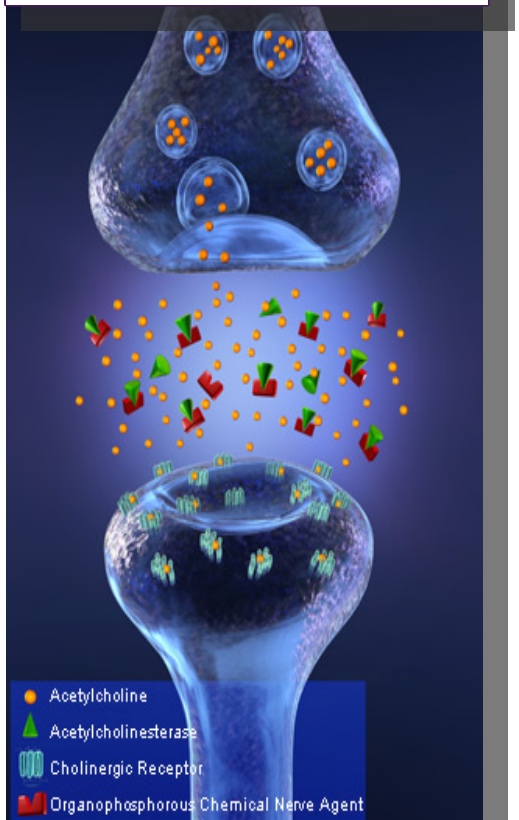
Journal of Arrhythmia WILEY

Atrial fibrillation and autonomic nervous system: A translational approach to guide therapeutic goals

Marco Rebecchi MD | Germana Panattoni MD | Bressi Edoardo MD | Ermenegildo de Ruvo MD | Luigi Sciarra MD | Alessandro Politano MD | Marianna Sgueglia MD | Chiara Ricagni MD | Sara Verbena MD | Cinzia Crescenzi MD | Catia Sangiorgi CACN | Alessio Borrelli MD | Lucia De Luca MD | Antonio Scarà MD | Domenico Grieco MD | Ilaria Jacomelli MD | Annamaria Martino MD | Leonardo Calò MD, FESC







Available online at www.sciencedirect.com
ScienceDirect
Journal of Electrocardiology 39 (2006) S180–S183
JOURNAL OF Electrocardiology
www.elsevier.com/locate/jelectrocard

The neural basis of atrial fibrillation

Benjamin J. Scherlag, PhD,[4] Eugene Patterson, PhD, Sunny S. Po, MD, PhD
Cardiac Arrhythmia Research Institute, at the University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA
Received 3 May 2006; accepted 31 May 2006

HFS
(high frequency stimulation)

↓

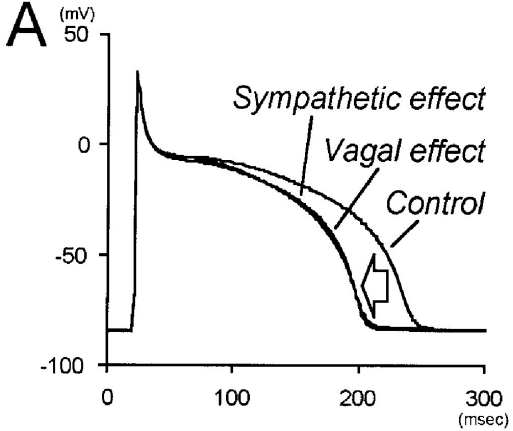
Acetylcholine

↓

- shortening of atrial and PV sleeve refractoriness
- Triggering/Ca-related of PVs firing

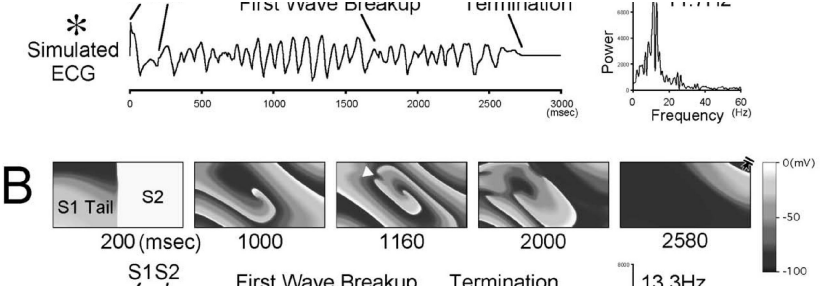
↓

Atrial fibrillation

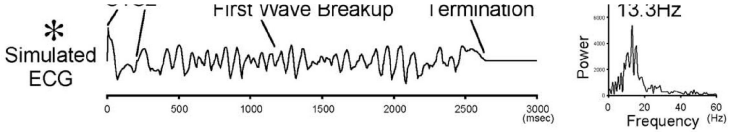


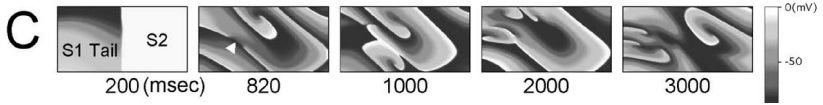
Vagal and Sympathetic effect
Shortened Action Potential (AP)

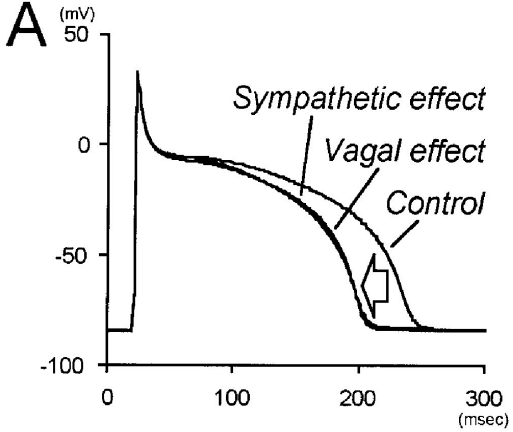
Vagal effect without spatial dispersion of refractoriness,



Sympathetic effect

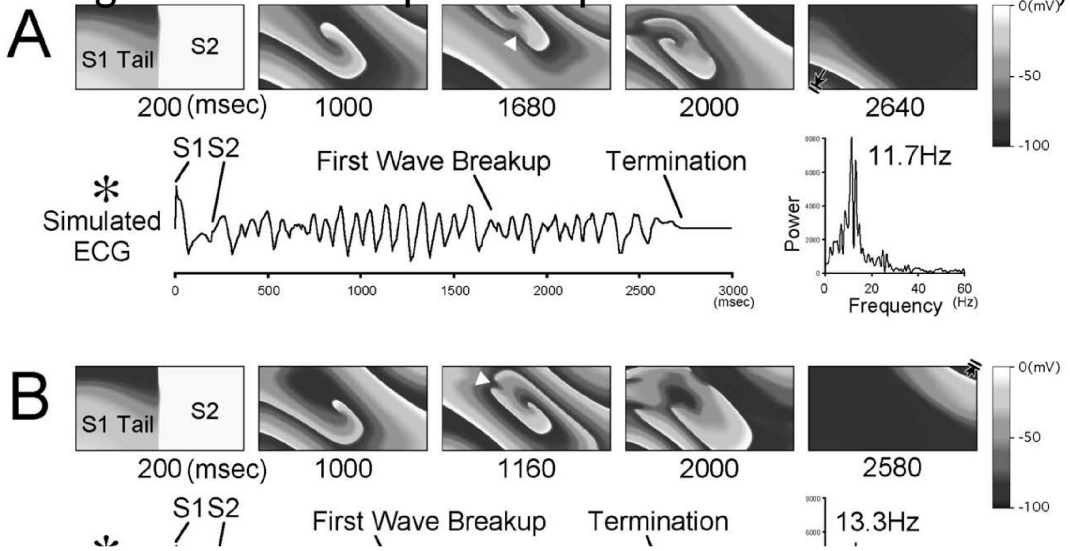


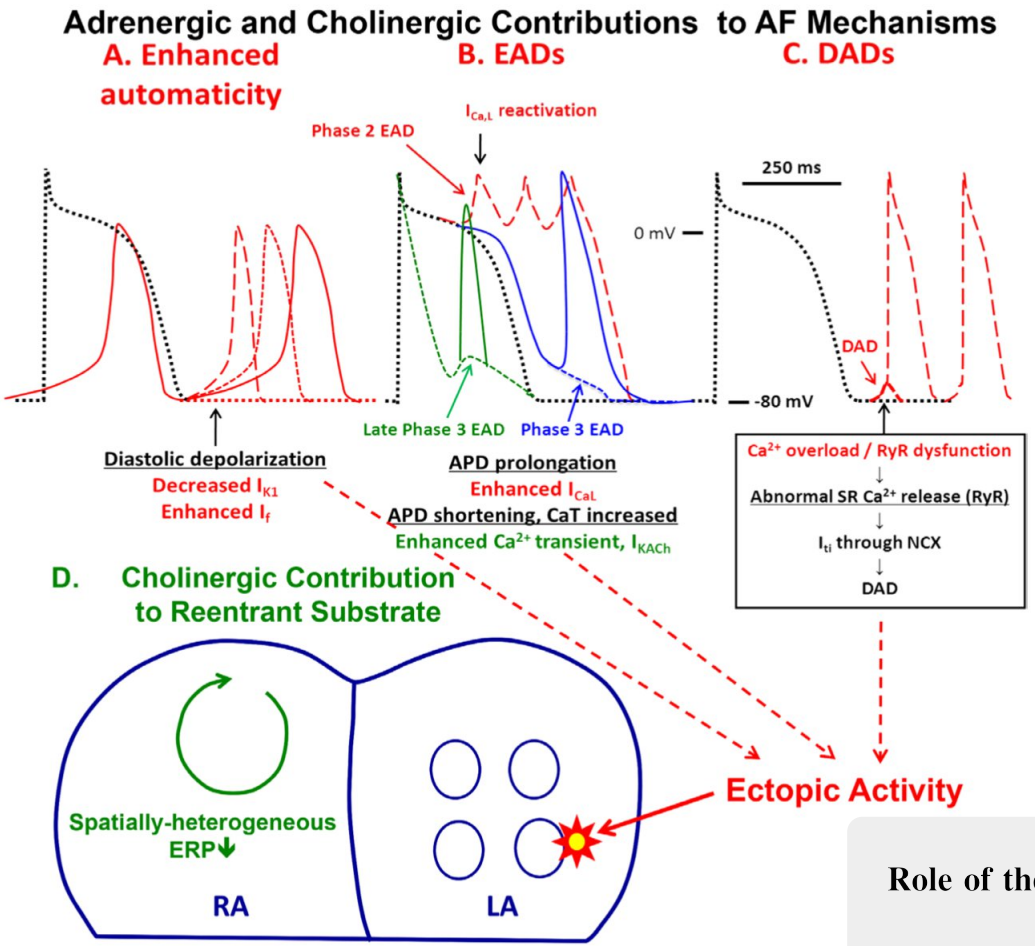




Vagal and Sympathetic effect
Shortened Action Potential (AP)

Vagal effect with spatial dispersion of refractoriness,





(*Circ Res*. 2014;114:1500-1515.)

Role of the Autonomic Nervous System in Atrial Fibrillation

Pathophysiology and Therapy

Peng-Sheng Chen, Lan S. Chen, Michael C. Fishbein, Shien-Fong Lin, Stanley Nattel

ELSEVIER

Biomed Pharmacother 56 (2002) 359s–363s

& BIOMEDICINE PHARMACOTHERAPY

www.elsevier.com/locate/biopha

Original paper

Differences in sympathetic and vagal effects on paroxysmal atrial fibrillation: a simulation study

Takashi Ashihara [a,*], Takenori Yao [a], Tsunetoyo Namba [b], Ayaka Kawase [c], Takanori Ikeda [c], Kazuo Nakazawa [d], Makoto Ito [a]

[a] *Division of Cardiology, Shiga University of Medical Science, Otsu, Japan*
[b] *Department of Medical Technology, Kagawa Prefectural College of Health Sciences, Kita, Japan*
[c] *Third Department of Internal Medicine, Toho University School of Medicine, Tokyo, Japan*
[d] *Department of Epidemiology, National Cardiovascular Center Research Institute, Suita, Japan*

……..the adrenergically-mediated paroxysmal AF terminates spontaneously and the vagally- mediated paroxysmal AF tends to be maintained……

VAGAL and ADRENERGIC AF
Effect of circadian day variations

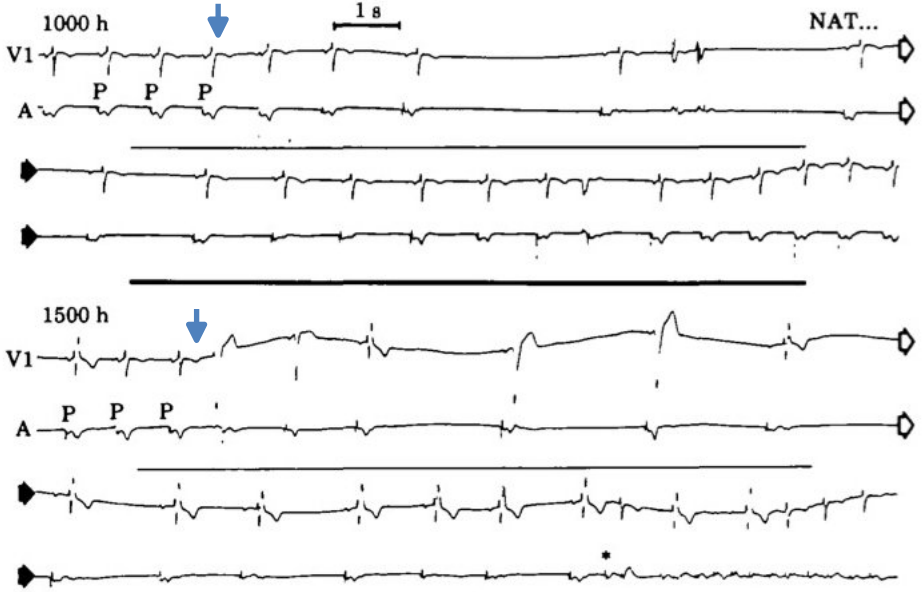
From these considerations, it is tempting to debate whether the role of the ANS in paroxysmal AF is essential or accessory, whether the primary disease lies in the atrial tissue itself or in its innervation, and whether the main target for treatment should be to control the myocardial substrate or its autonomic modulators. A particular sensitivity of the substrate, or any dysfunction of the ANS, or both, may be a cause of AF. Determining which is responsible might seem an easy task given the clinical tools that are currently available, but in fact this is not so.

Clinical aspects

In studying patients with frequent paroxysmal attacks of AF, and the relationships between their arrhythmia and

any structural heart disease. This is most probably due to the fact that any heart disease tends to shift the vago-sympathetic balance toward some degree of sympathetic predominance[3]. The usual history is of weekly episodes, lasting a few hours: they predominantly occur at night, are preceded by a progressive bradycardia, and typically a pattern of common atrial flutter alternates with the ECG aspect of AF. The morning period is the commonest time for reversion to sinus rhythm (Fig. 1). It is exceptional for such attacks to occur between breakfast and lunch, when sympathetic predominance is most marked. Neither physical exertion nor emotional stress triggers the arrhythmia. However, the relaxation that follows effort or emotional stress is frequently associated with the onset of AF. Rest,

Adenosine



10 am

15 am

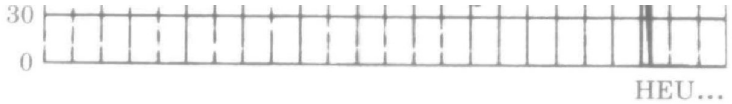


Figure 1 Circadian distribution of paroxysmal AF. The two panels display the typical opposition between the diurnal distribution of adrenergically mediated (upper diagram) and vagally related (lower diagram) forms in two different patients. The periods during which AF is present are clearly apparent from the heart rate trends, and the distribution of the maximal and minimal heart rates.

Adrenergic AF

Vagal AF

or during exercise. During the attacks, no features of common flutter are visible at any time. On the contrary when organized electrical activity alternates with AF, it suggests the presence of an ectopic automatic focus. In our experience, in the absence of heart disease, adrenergically mediated paroxysmal AF is much less frequent than vagally mediated AF. On the other hand, when considering paroxysmal AF in patients with identified heart disease, sympathetic influences at the onset of the arrhythmia are predominant. This probably explains why studying the time of onset of AF without considering the presence or

Experimental aspe

It is difficult to s
studies which hav
and the atrium[10,11
the findings are co
ity of vago-sympatl
doubt.

The anatomical
different for vag
latency time and

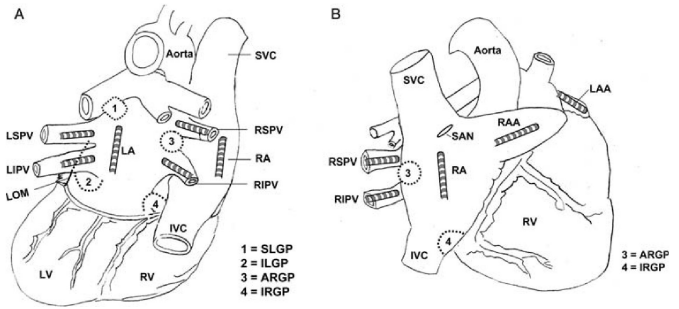
Why a selective approach?

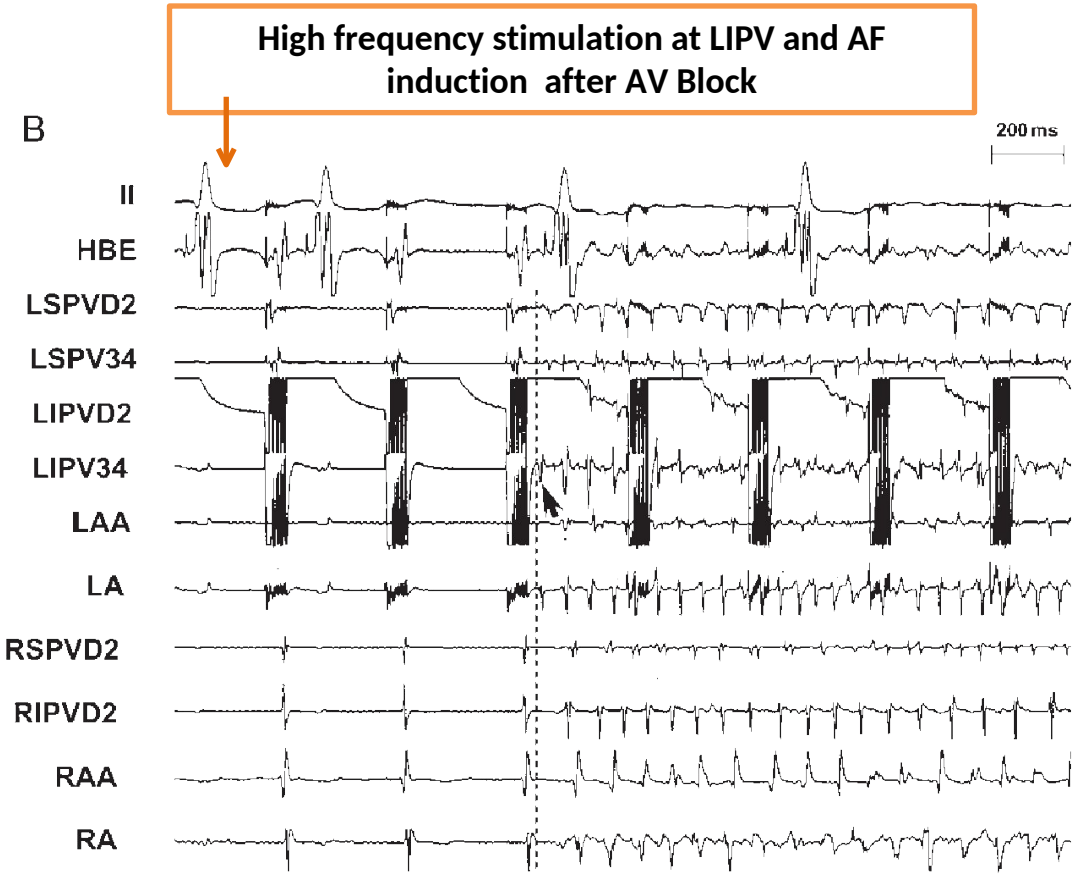
Cardiovascular Research (2009) 84, 245–252
doi:10.1093/ cvr/ cvp194

Experimental data

Autonomic mechanism for initiation of rapid firing from atria and pulmonary veins: evidence by ablation of ganglionated plexi

Zhibing Lu[1], Benjamin J. Scherlag[2], Jiaxiong Lin[3], Lilei Yu[1], Ji-Hong Guo[4], Guodong Niu[2], Warren M. Jackman[2], Ralph Lazzara[2], Hong Jiang[1], and Sunny S. Po[2]*




High frequency stimulation at LIPV and AF induction after AV Block
B
200 ms
II
HBE
LSPVD2
LSPV34
LIPVD2
LIPV34
LAA
LA
RSPVD2
RIPVD2
RAA
RA

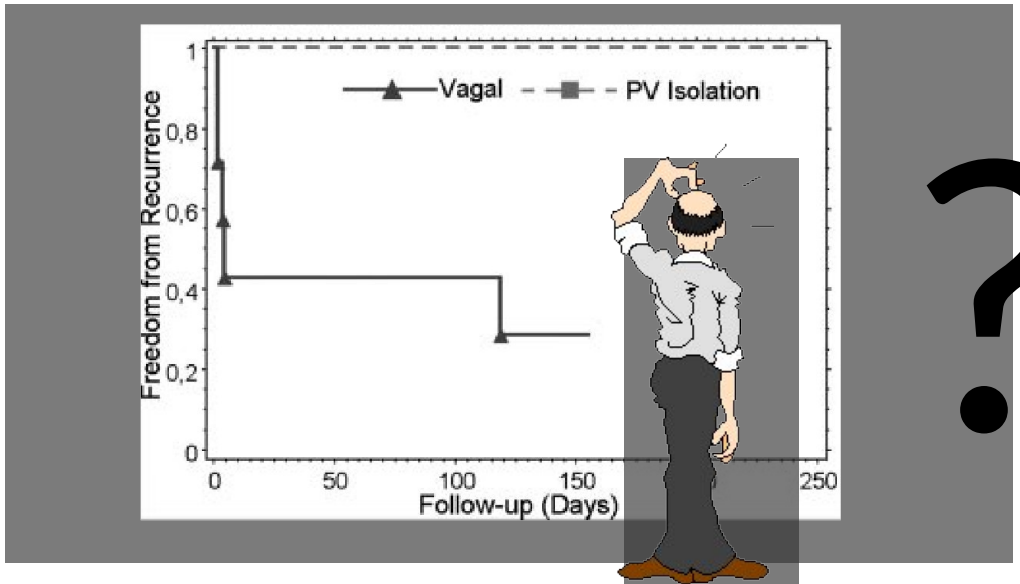
Selective Atrial Vagal Denervation Guided by Evoked Vagal Reflex to Treat Patients With Paroxysmal Atrial Fibrillation

Mauricio Scanavacca, Cristiano F. Pisani, Denise Hachul, Sissy Lara, Carina Hardy, Francisco Darrieux, Ivani Trombetta, Carlos Eduardo Negrão and Eduardo Sosa

Circulation 2006;114;876-885; originally published online Aug 21, 2006;

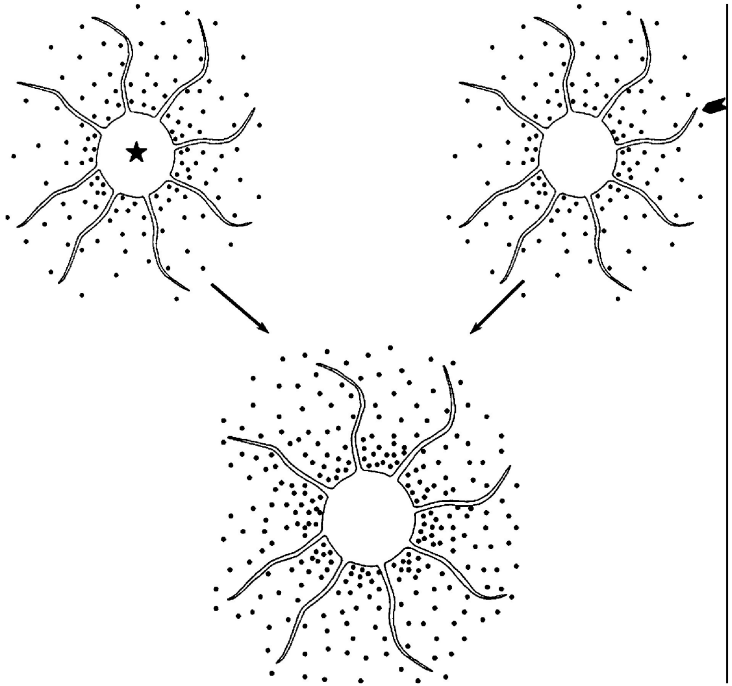
Conclusion

RF catheter ablation of selected atrial sites in which high-frequency stimulation induced vagal reflexes ***may prevent AF recurrences in selected patients*** with apparently vagal-induced paroxysmal AF.



Selective AF ablation (epi-endo) in 10 pts

Why the reduced effectiveness of selective approach
The octopus Hypotesis?



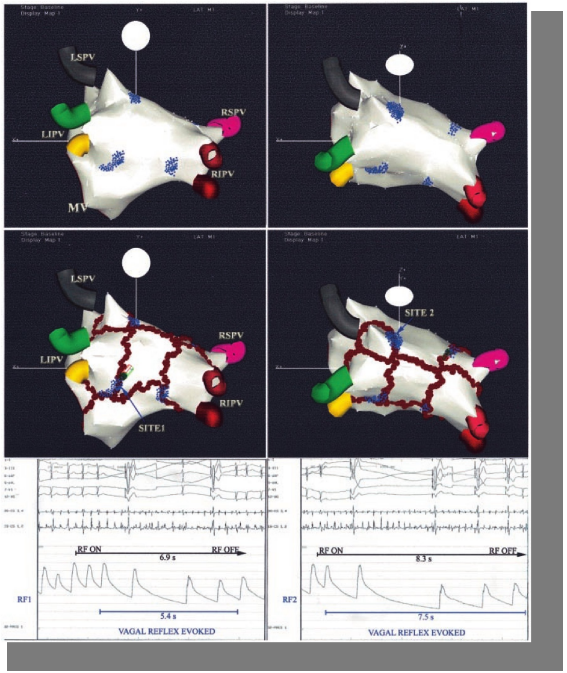
HFS of octopus head may trigger local release of a gradient of excessive amounts of neurotransmitters and subsequently initiate AF

- **HFS assons/tentacles can determines** a retrograde activation of GP at distance, can provide an interesting explanation for **the discrepancy between the sites of vagal response (which are also the sites of radiofrequency ablation) and real location of GP.**

J Cardiovasc Electrophysiol, Vol. 18, pp. 83-90, January 2007

Anatomic Approach
cardioneuroablationis in left atrium is all we need?

Pulmonary Vein Denervation Enhances Long-Term Benefit After Circumferential Ablation for Paroxysmal Atrial Fibrillation



Risposta vagale evocata in 100 pz su 297 sottoposti ad isolamento delle vene polmonari. Il 34% di tali pz presentò una percentuale del 99% di libertà da recidive di FA ad un follow up di 12 mesi

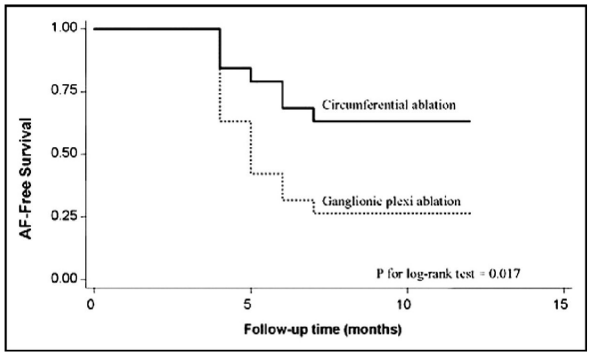
Pappone et al. Circulation 2004; 109:327–334.

Anatomic Approach
cardioneuroablationis in left atrium is all we need?

Anatomic Approach for Ganglionic Plexi Ablation in Patients With Paroxysmal Atrial Fibrillation

Demosthenes Katritsis, MD, PhD[a,*], Eleftherios Giazitzoglou, MD[a], Demetrios Sougiannis[a], Nicolaos Goumas[a], George Paxinos, MD[a], and A. John Camm, MD[b]

Variable	Circumferential Ablation (n = 19)	GP Ablation (n = 19)
Age (yrs)	52.2 ± 9.4	51.2 ± 8.8
Men	16 (84%)	16 (84%)
Cause of AF		
Hypertension	13 (68%)	11 (58%)
Coronary artery disease	2 (10%)	2 (10%)
Lone atrial fibrillation	5 (26%)	6 (32%)
Medication		
β Blockers	18 (95%)	19 (100%)
Angiotensin-converting enzyme inhibitors/ angiotensin receptor blockers	13 (68%)	11 (58%)
Diuretics	11 (58%)	10 (53%)

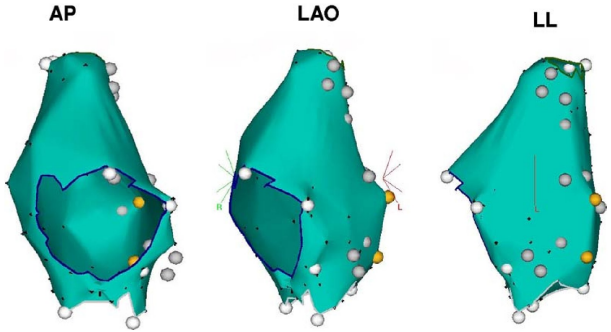


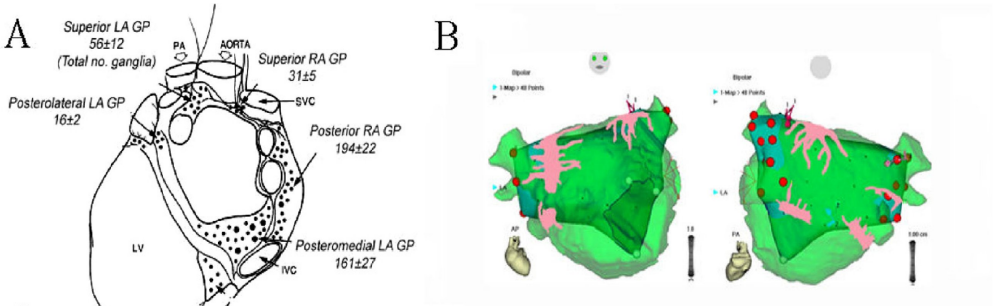
J Am Cardiol
2008;102:330–334

Selective ganglionated plexi ablation for paroxysmal atrial fibrillation

Evgeny Pokushalov, MD, PhD,* Alex Romanov, MD,* Pavel Shugayev, MD,* Sergey Artyomenko, MD,* Natalya Shirokova, MD,* Alex Turov, MD,* Demosthenes G. Katritsis, MD, PhD†

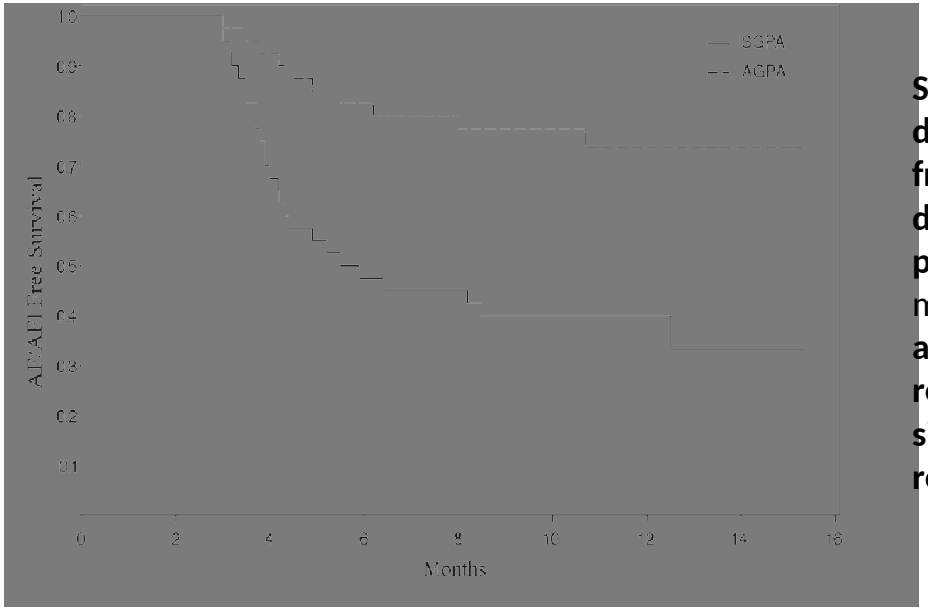
	Selective GP ablation (n = 40)	Anatomic ablation (n = 40)	*P*
Age (years)	53 ± 9	54 ± 11	.4
Sex, male/female, n	32/8	34/6	.3
AF history (years)	6 ± 5	6 ± 4	.9
Number of episodes of AF/mo	12 ± 13	12 ± 14	.9
LVEF, %	58.2 ± 5.3	57.6 ± 5.6	.3
Hypertension, n	9	8	.4
Diabetes mellitus, n	2	3	.6
Left atrial diameter, mm	48 ± 6.1	49 ± 5.2	.6





Selective ganglionated plexi ablation for paroxysmal atrial fibrillation

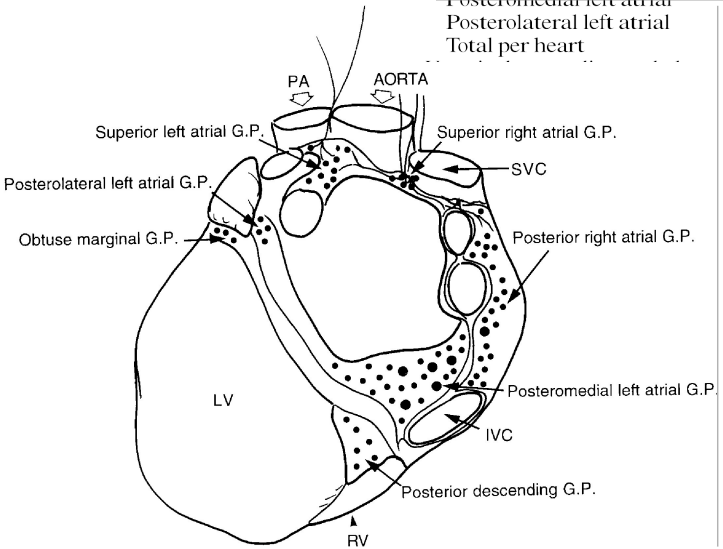
Evgeny Pokushalov, MD, PhD,* Alex Romanov, MD,* Pavel Shugayev, MD,* Sergey Artyomenko, MD,* Natalya Shirokova, MD,* Alex Turov, MD,* Demosthenes G. Katritsis, MD, PhD†



Selective GP ablation directed by high-frequency stimulation does not eliminate paroxysmal AF in the majority of patients. **An anatomic approach for regional ablation at the sites of GP confers better results.**

The rationale of cardioneuroablation in the right atrium
The anatomy

Ganglionic plexus	5–10 Neurons	11–50 Neurons	50–100 Neurons	100–200 Neurons	>200 Neurons	Total no. ganglia per heart
Atrial ganglionated plexuses						
Superior right atrial	19.2 ± 2.9	9.5 ± 2.8	2.2 ± 0.4	0.3 ± 0.1	0	31 ± 5
Superior left atrial	29.4 ± 5.9	19.7 ± 5.1	5.3 ± 1.9	2.2 ± 0.7	0.5 ± 0.2	56 ± 12
Posterior right atrial	90.1 ± 13.7	66.4 ± 7.6	22.8 ± 1.9	9.7 ± 0.7	4.7 ± 0.7	194 ± 22
Posteromedial left atrial	82.8 ± 13.5	56.4 ± 9.8	18.2 ± 4.1	4.5 ± 0.9	1.8 ± 0.6	161 ± 27
Posterolateral left atrial	8.2 ± 2.2	5.7 ± 1.1	1.7 ± 0.4	0.3 ± 0.1	0	16 ± 2
Total per heart						458 ± 43



A large number of ganglia is placed in the posterior wall of RA

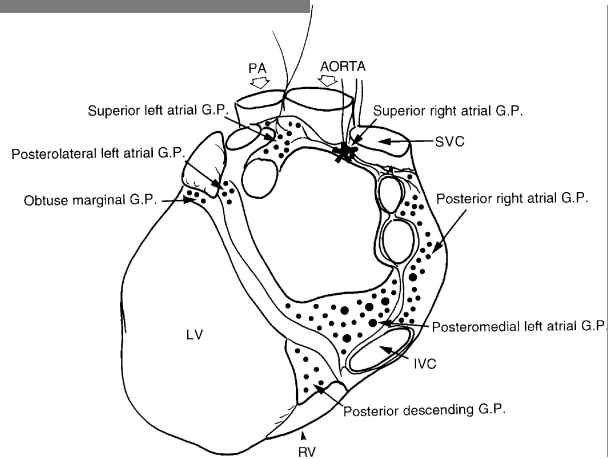
The rationale of cardioneuroablation in the right atrium Eletrophysiology

Autonomic mechanism for initiation of rapid firing from atria and pulmonary veins: evidence by ablation of ganglionated plexi

Zhibing Lu[1], Benjamin J. Scherlag[2], Jiaxiong Lin[3], Lilei Yu[1], Ji-Hong Guo[4], Guodong Niu[2], Warren M. Jackman[2], Ralph Lazzara[2], Hong Jiang[1], and Sunny S. Po[2]*

SVC-aorta-GP in AF initiated by rapid firing from the SVC.

HFS of this anatomical structure slowed sinus rate and/or atrioventricular conduction and determined more significant shortening of ERP and a greater increase in window of vulnerability at the SVC than other sites



Role of ARGP, IRGP Third fat pad

Received: 31 August 2020 | Revised: 21 December 2020 | Accepted: 15 January 2021
DOI: 10.1002/joa3.12512

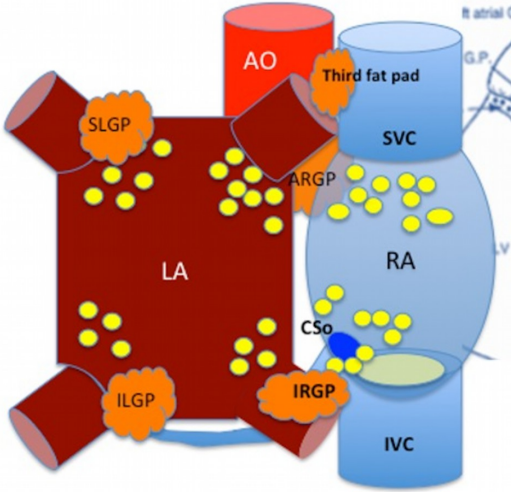
CLINICAL REVIEW

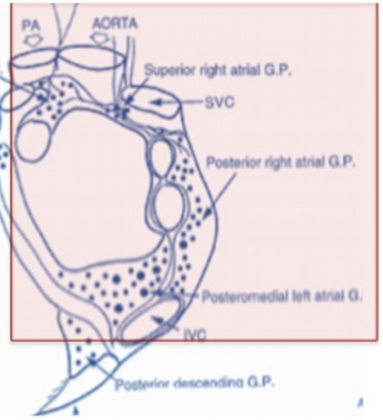
Journal of Arrhythmia WILEY

Atrial fibrillation and autonomic nervous system: A translational approach to guide therapeutic goals

Marco Rebecchi MD | Germana Panattoni MD | Bressi Edoardo MD | Ermenegildo de Ruvo MD | Luigi Sciarra MD | Alessandro Politano MD | Marianna Sgueglia MD | Chiara Ricagni MD | Sara Verbena MD | Cinzia Crescenzi MD | Catia Sangiorgi CACN | Alessio Borrelli MD | Lucia De Luca MD | Antonio Scarà MD | Domenico Grieco MD | Ilaria Jacomelli MD | Annamaria Martino MD | Leonardo Calò MD, FESC

Panel A





Panel B

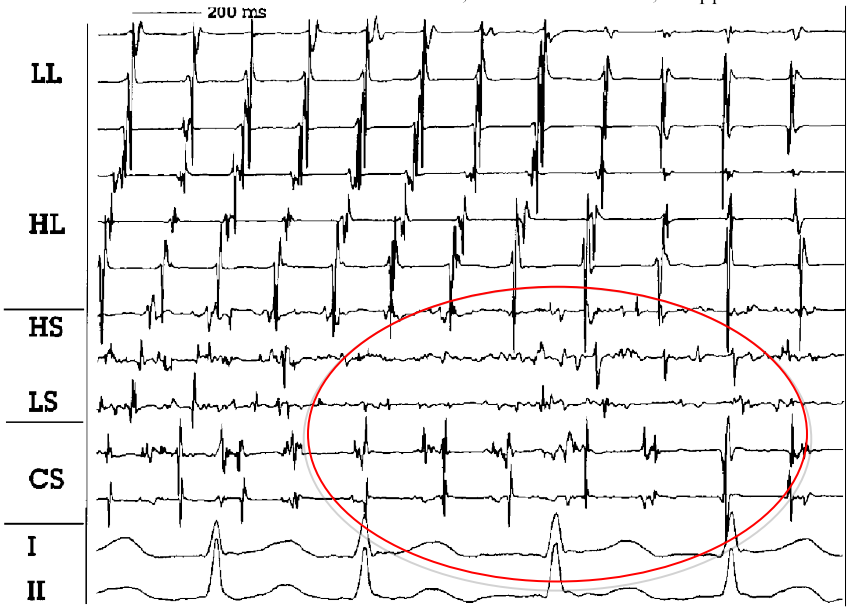
Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association®

Role of RA Posterior Septum

Atrial Mapping and Radiofrequency Catheter Ablation in Patients With Idiopathic Atrial Fibrillation: Electrophysiological Findings and Ablation Results

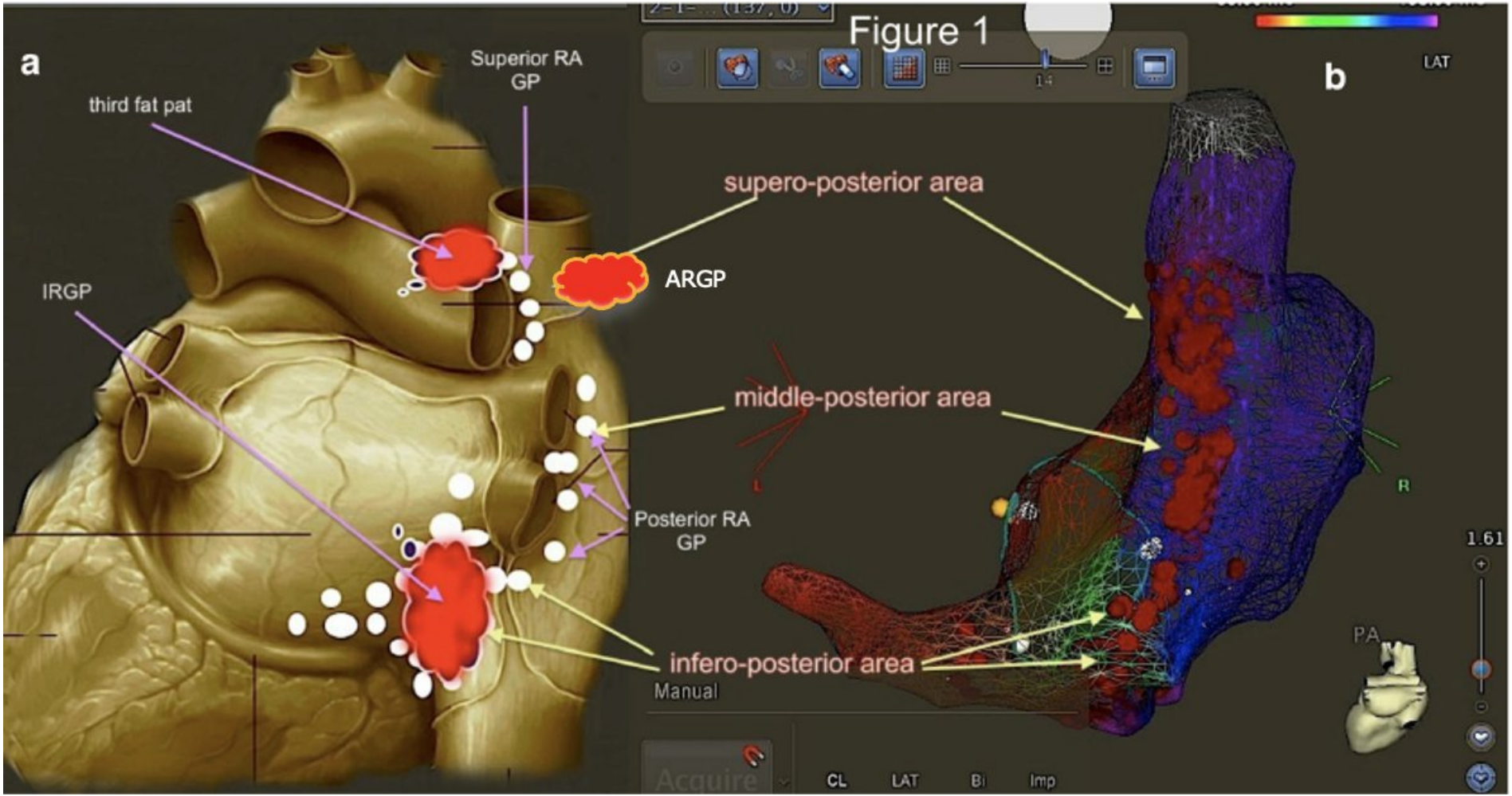
Fiorenzo Gaita, Riccardo Riccardi, Leonardo Calò, Marco Scaglione, Lucia Garberoglio, Renzo Antolini, Michele Kirchner, Filippo Lamberti and Elena Richiardi



The septal line can be effective in patients with vagal paroxysmal AF, particularly when the septum presented "disorganized" electric activity (a normal activity i showed at level of Lateral wall)

Gaita, et al. Circulation. 1998;97:2136-2145.

Figure 1


a
Superior RA GP
third fat pat
IRGP
ARGP
Posterior RA GP
b
LAT
supero-posterior area
middle-posterior area
infero-posterior area
Manual
R
1.61
PA
Acquire
CL
LAT
Bi
Imp

JACC: CASE REPORTS VOL. 3, NO. 4, 2021

RA GPs and LA GPs interconnections

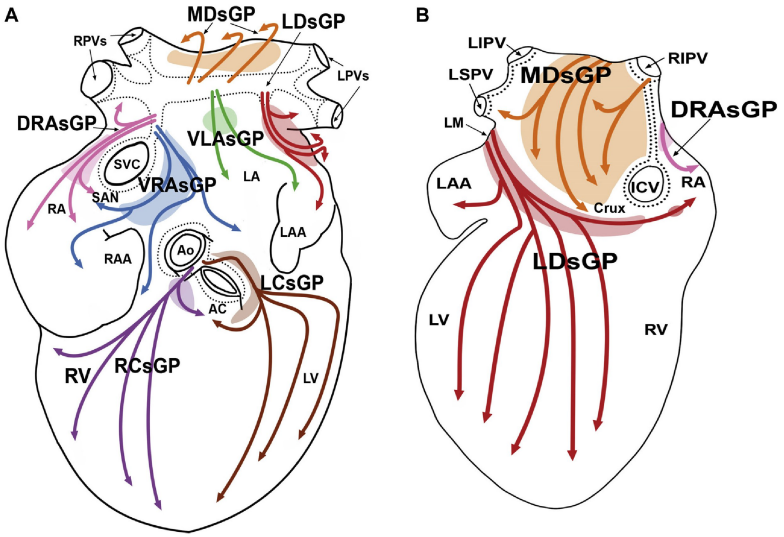
MINI-FOCUS ISSUE: ELECTROPHYSIOLOGY

ANATOMY CARD: DA VINCI CORNER

Anatomy and Physiology of Intrinsic Cardiac Autonomic Nervous System

Da Vinci Anatomy Card #2

Tolga Aksu, MD,[a] Dhiraj Gupta, MD,[b] Dainius H. Pauza, PhD[c]



and the region over the interatrial septum.

Circulation
Arrhythmia and Electrophysiology

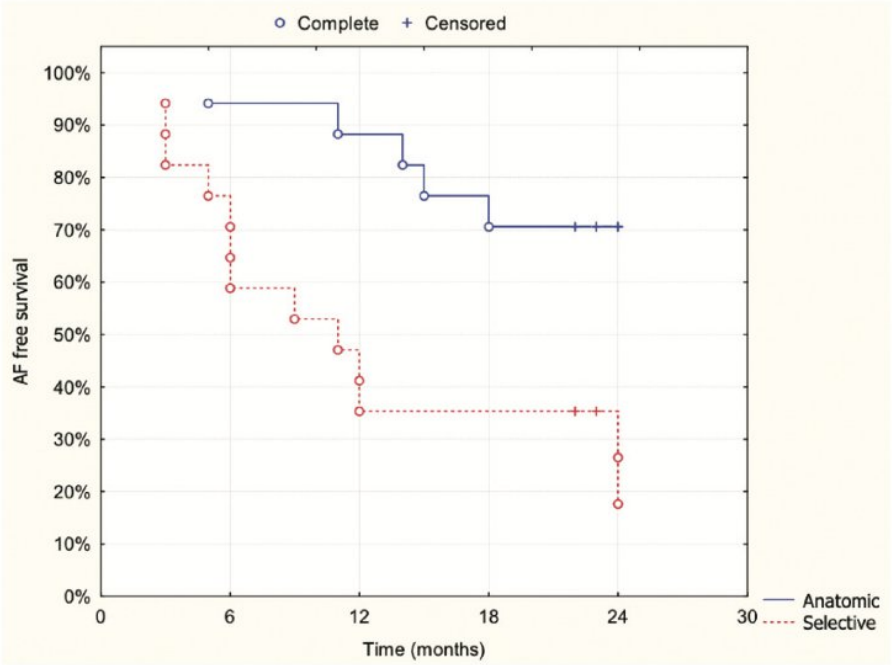
American Heart Association®

Catheter Ablation of Right Atrial Ganglionated Plexi in Patients With Vagal Paroxysmal Atrial Fibrillation

Leonardo Calò, Marco Rebecchi, Luigi Sciarra, Lucia De Luca, Alessandro Fagagnini, Lorenzo Maria Zuccaro, Pietro Pitrone, Serena Dottori, Maurizio Porfirio, Ermenegildo de Ruvo and Ernesto Lioy

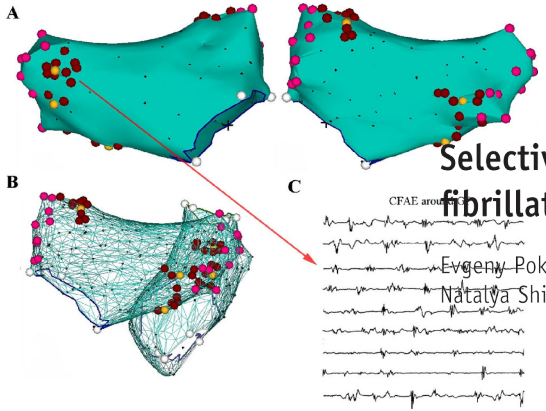
	Overall (n=34)	Anatomic GP Abl (n=17)	Selective GP Abl (n=17)
Age, y*	48.6±4.6	49.5±4.8	47.7±4.4
Sex, males, n (%)	22 (64.7)	10 (58.8)	12 (70.6)
AF vagal triggers, n (%)			
During sleep	23 (67.6)	11 (64.7)	12 (70.6)
After meals	8 (23.6)	5 (29.4)	3 (17.6)
Coughing	3 (8.8)	1 (5.9)	2 (11.8)
AF history, y*	4.9±1.3	4.9±1.4	4.9±1.2
AF episodes/y*	83.6±22.3	85.4±25.6	81.8±23.3
Risk factors for cardiopathy, n (%)			
Hypertension	3 (8.8)	2 (11.8)	1 (5.9)
Dyslipidemia	3 (8.8)	1 (5.9)	2 (11.8)
Echocardiogram*			
Left atrium AP diameter, mm	37.2±0.8	37.1±0.6	37.3±0.9
LVEDD, mm	48.3±3.7	46.4±3.6	47.5±3.8
LVESD, mm	28.4±3.6	27.7±3.4	28.2±3.4
EF, %	63.4±5.3	63.3±5.1	63.5±5.2
Septal thickness, mm	9.7±0.4	9.4±0.6	9.6±0.5
Posterior wall thickness, mm	8.9±0.5	8.7±0.4	8.8±0.6

Circ Arrhythm Electrophysiol. 2012;5:22-31.



Calò, et al. Circ Arrhythm Electrophysiol. 2012;5:22-31.

CFAEs ablation as target of cardioneuroablation?



Selective ganglionated plexi ablation for paroxysmal atrial fibrillation

Evgeny Pokushalov, MD, PhD,* Alex Romanov, MD,* Pavel Shugayev, MD,* Sergey Artyomenko, MD,* Natalya Shirokova, MD,* Alex Turov, MD,* Demosthenes G. Katritsis, MD, PhD†

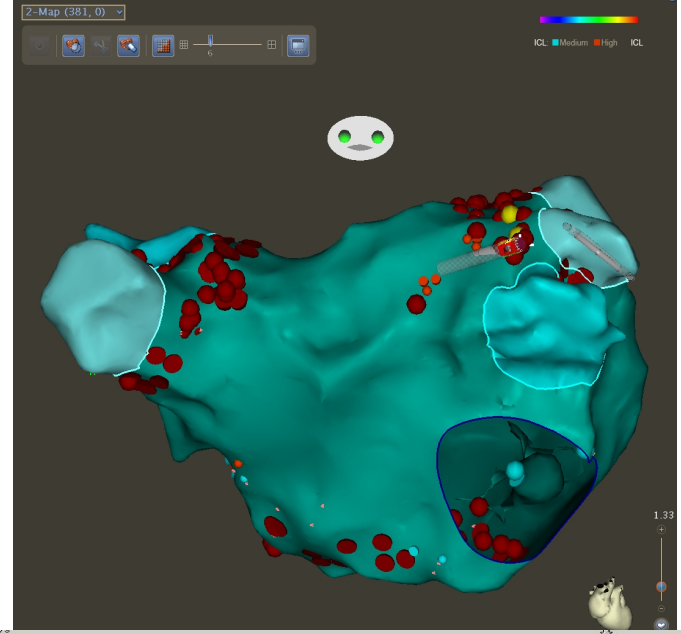
Circulation
Arrhythmia and Electrophysiology

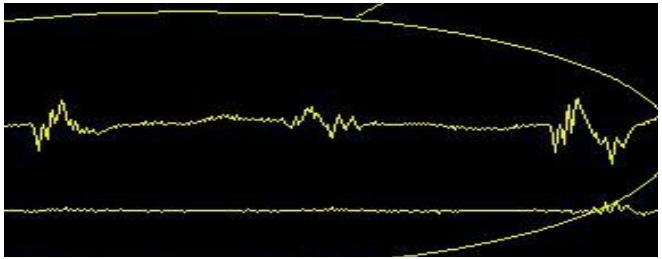
Catheter Ablation of Right Atrial Ganglionated Plexi in Patients With Vagal Paroxysmal Atrial Fibrillation

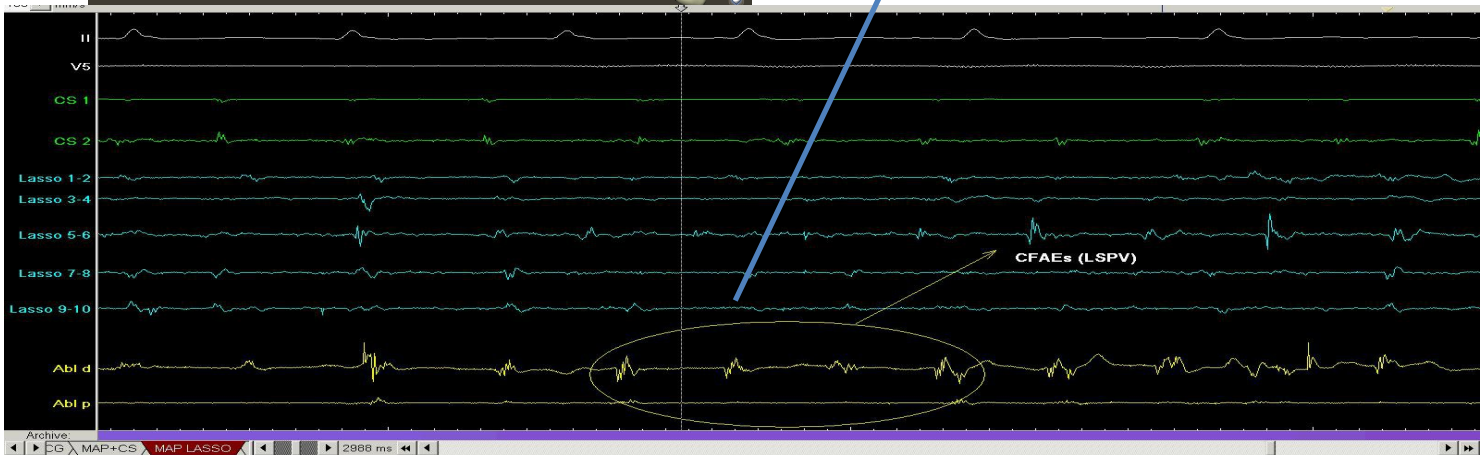
Leonardo Calò, Marco Rebecchi, Luigi Sciarra, Lucia De Luca, Alessandro Fagagnini, Lorenzo Maria Zuccaro, Pietro Pitrone, Serena Dottori, Maurizio Porfirio, Ermenegildo de Ruvo and Ernesto Lioy

American Heart Association®

.....we observed **CFAEs** around ive vagal site the ptal region, particularly the posteroseptal space, showed the greatest prevalence of such electrograms. **GP ablation determined in 33 of the 34 patients studied the disappearance or the significant reduction of CFAEs.**

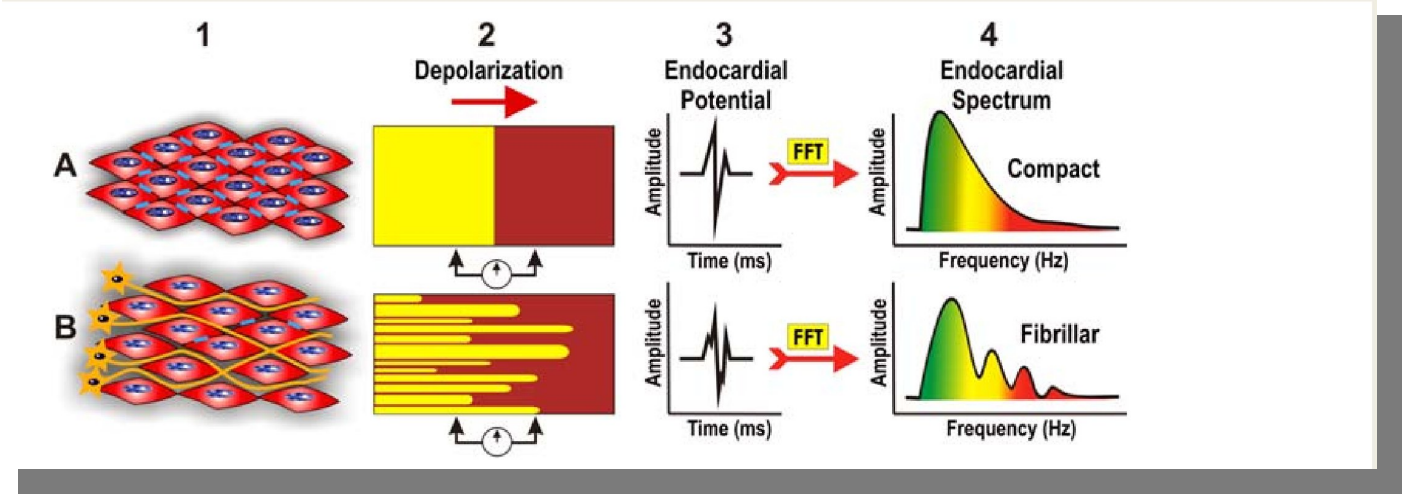

2-Map (381, 0)
ICL Medium High ICL
1.33




II
V5
CS 1
CS 2
Lasso 1-2
Lasso 3-4
Lasso 5-6
Lasso 7-8
Lasso 9-10
Abl d
Abl p
CFAEs (LSPV)
Archive:
CG
MAP+CS
MAP LASSO
2988 ms

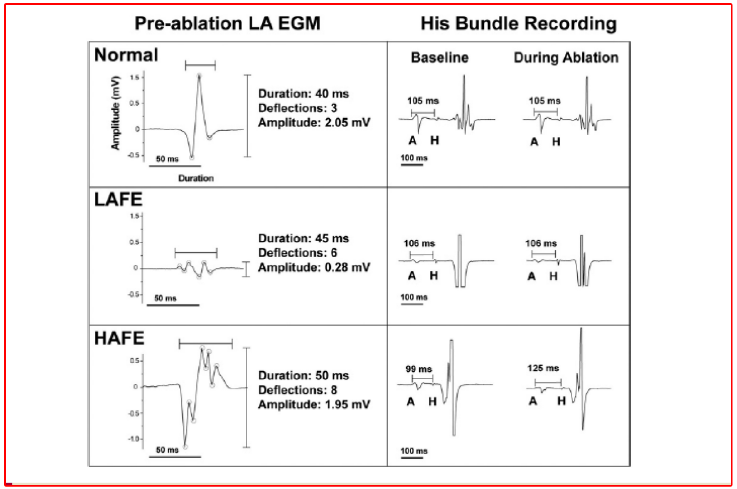
Catheter ablation of severe neurally meditated reflex (neurocardiogenic or vasovagal) syncope: cardioneuroablation long-term results

Jose Carlos Pachon M[1,2,3*], Enrique Indalecio Pachon M[1], Maria Zelia Cunha Pachon[1], Tasso Julio Lobo[1], Juan Carlos Pachon M[1,2], and Tomas Guilhermo Santillana P[1]

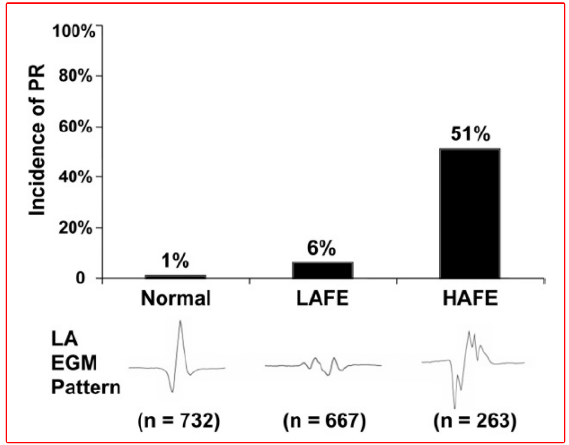


Functional Characterization of Atrial Electrograms in Sinus Rhythm Delineates Sites of Parasympathetic Innervation in Patients With Paroxysmal Atrial Fibrillation

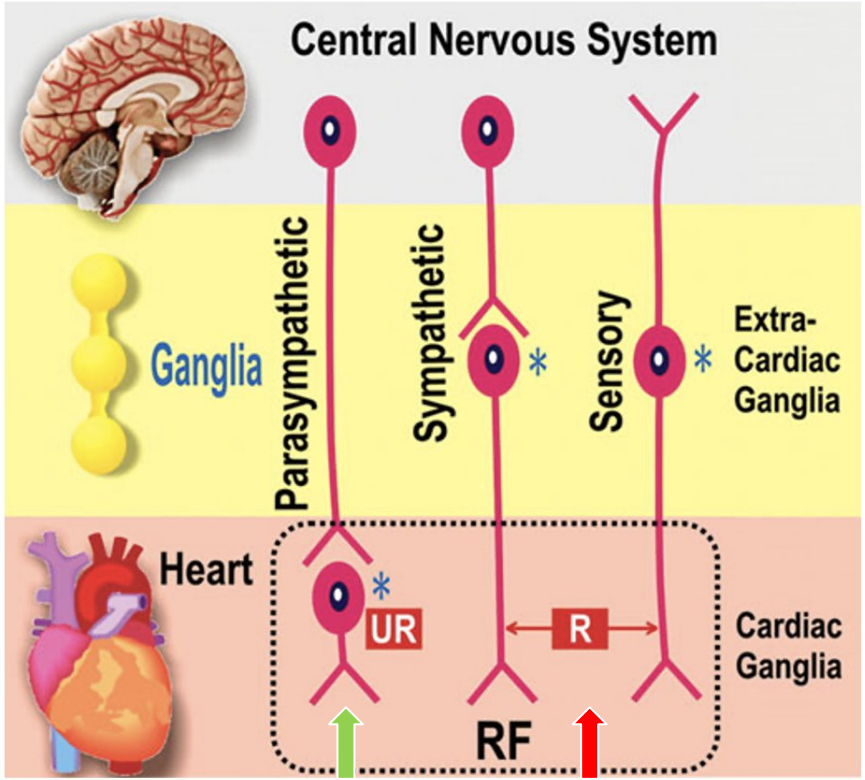
Nicolas Lellouche, MD, Eric Buch, MD, Andrew Celigoj, BS, Carin Siegerman, PHD,
David Cesario, MD, PHD, Carlos De Diego, MD, Aman Mahajan, MD, PHD,
Noel G. Boyle, MD, PHD, Isaac Wiener, MD, Alan Garfinkel, PHD, Kalyanam Shivkumar, MD, PHD



Incidence of vagal response during AF ablation

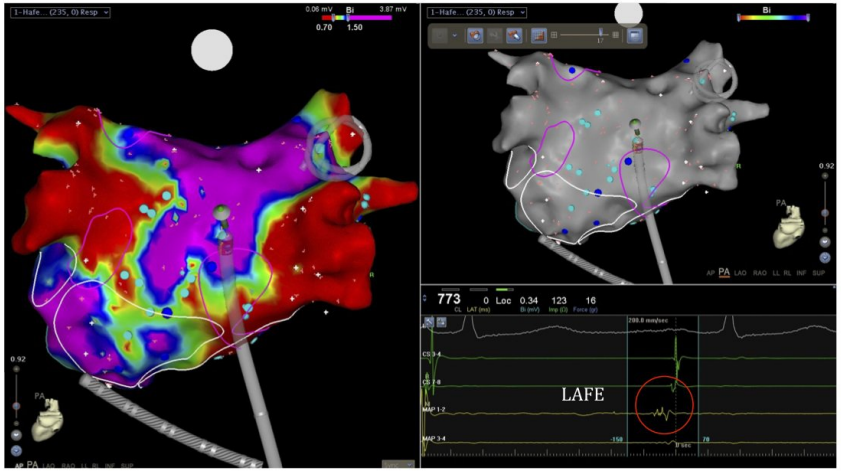


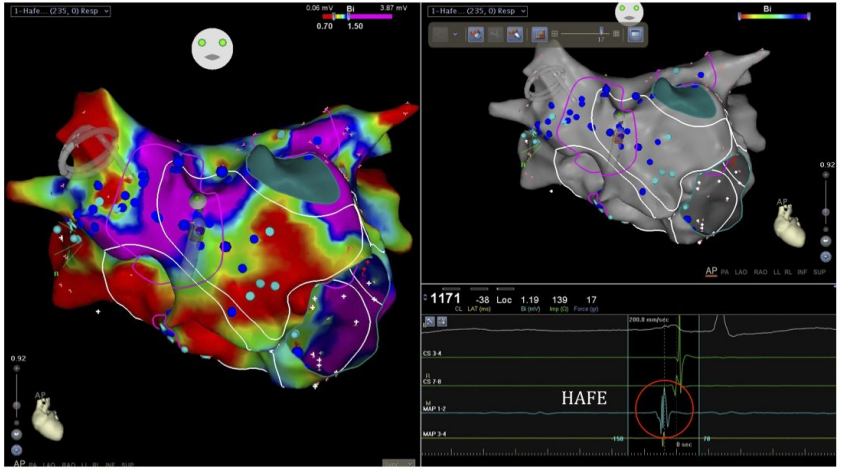
Effectiveness RF cardiac GP ablation on the basis of reinnervation



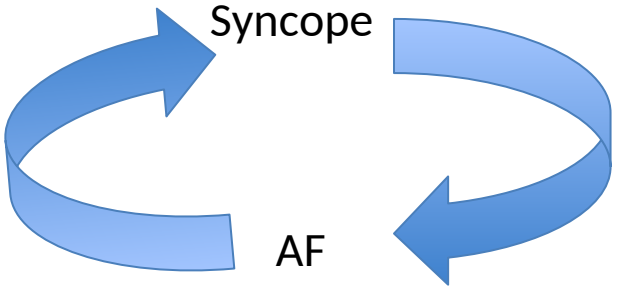
Not Reinnevation

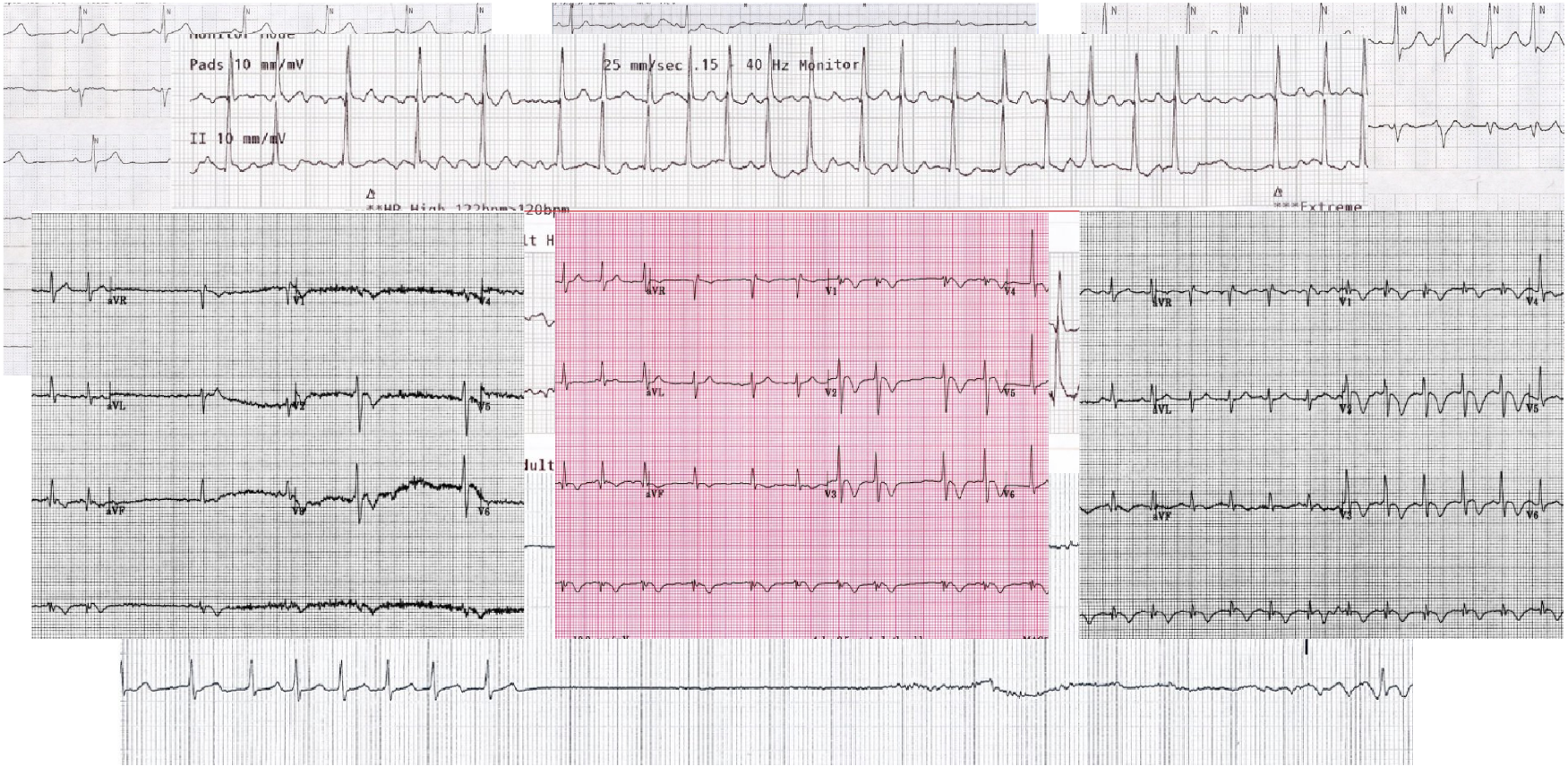
Reinnevation


LAFE


HAFE

Syncope and AF




Pads 10 mm/mV
25 mm/sec .15 - 40 Hz Monitor
II 10 mm/mV
aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

Seizure 21 (2012) 643–645

Contents lists available at SciVerse ScienceDirect

Seizure

journal homepage: www.elsevier.com/locate/yseiz

Case report

Prolonged atrial fibrillation following generalized tonic–clonic seizures

Rainer Surges [a,*], Susanna Moskau [a], Bettina Viebahn [a], Jan-Christoph Schoene-Bake [a], Joerg O. Schwab [b], Christian E. Elger [a]

[a] Department of Epileptology, University of Bonn Medical Center, Sigmund-Freud-Str. 25, 53105 Bonn, Germany

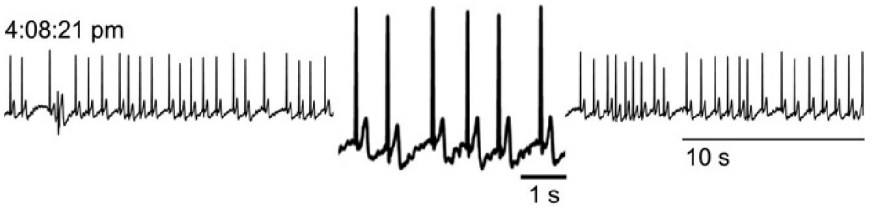
- ❑ Adenergic AF
- ❑ Thromboembolic risk
- ❑ Role of AF of SUDEP

Table 1
Review of

Case
1
2
3
4
5
6
7
8
9

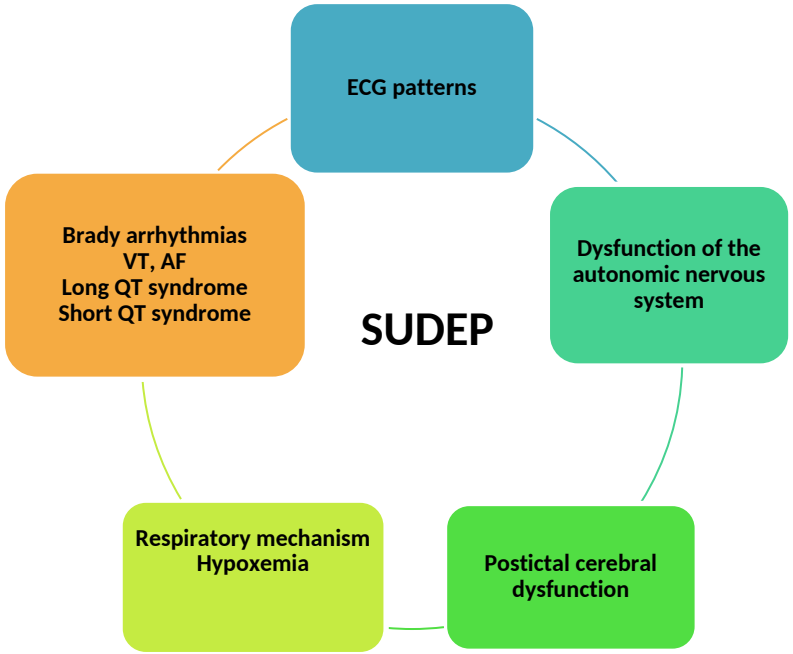
n.r., not reported.

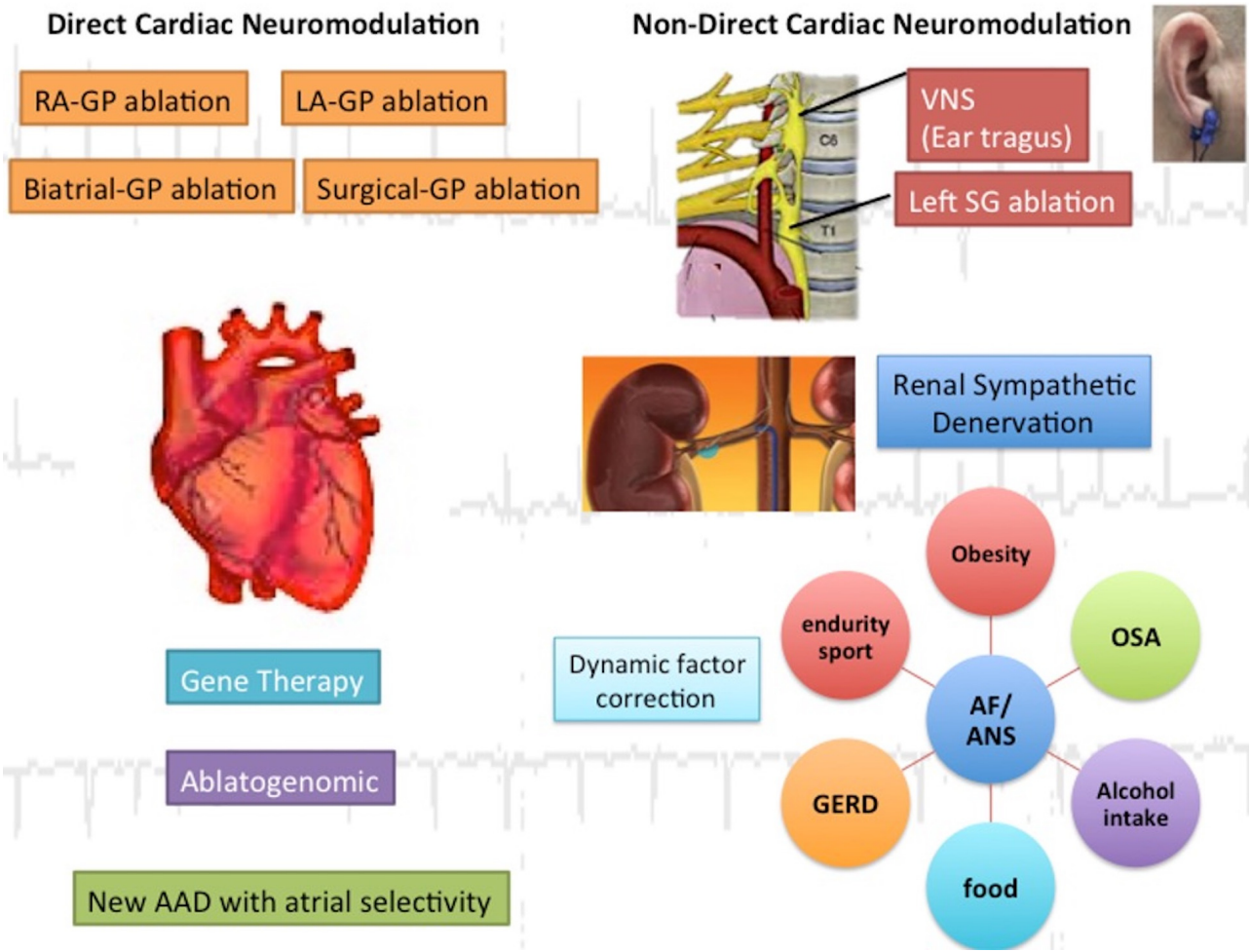
Interval of AF duration
10 s-25 h



Sudden unexpected death in epilepsy: mechanisms, prevalence, and prevention

Rainer Surges[a] *and Josemir W. Sander*[b,c,d]





CENTRAL IMAGE

Rebecchi at al. Journal of Arrhtymia. 2021