

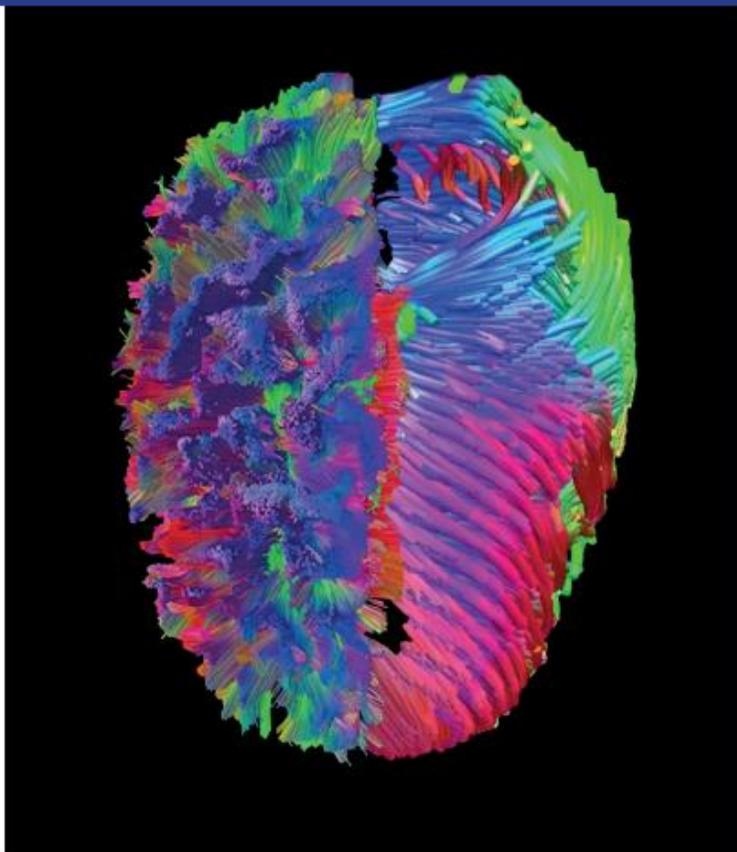


Complicanze
cardiache da eventi
neurologici acuti

LA NEUROCARDIOLOGIA

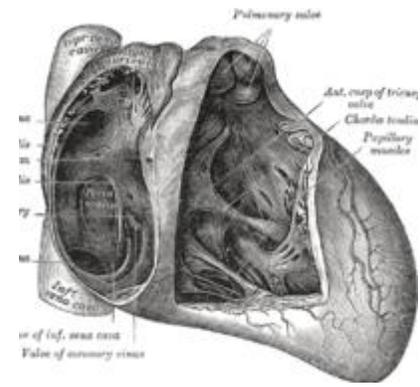
QUANDO CUORE E CERVELLO SI INCONTRANO

Georgette Khoury • Stefano Caproni

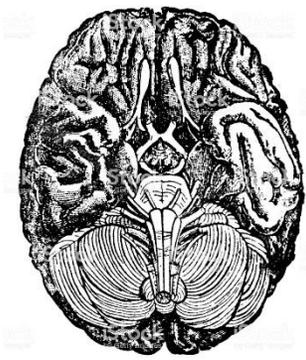


 **CIC** Edizioni Internazionali



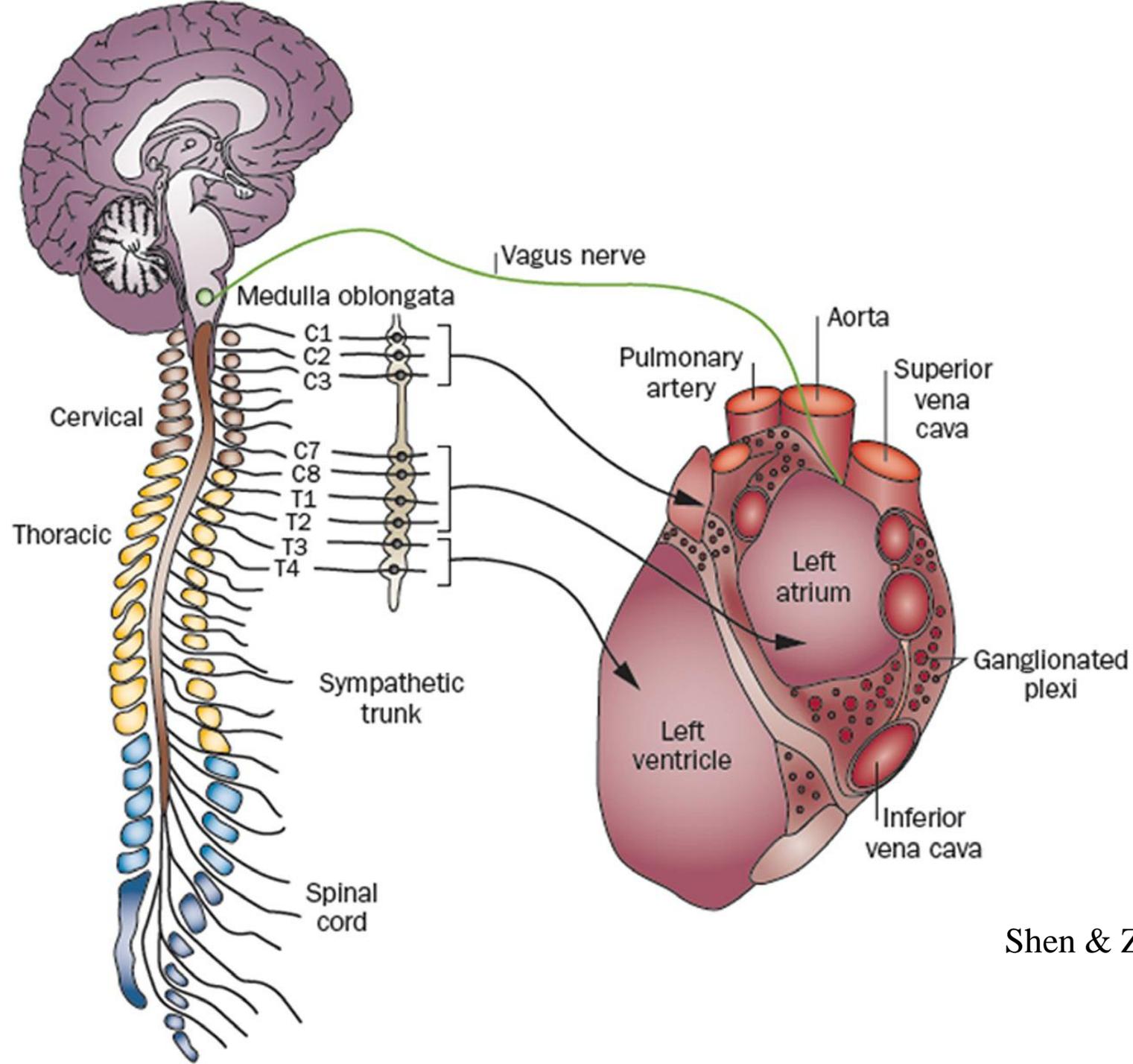


Cenni storici



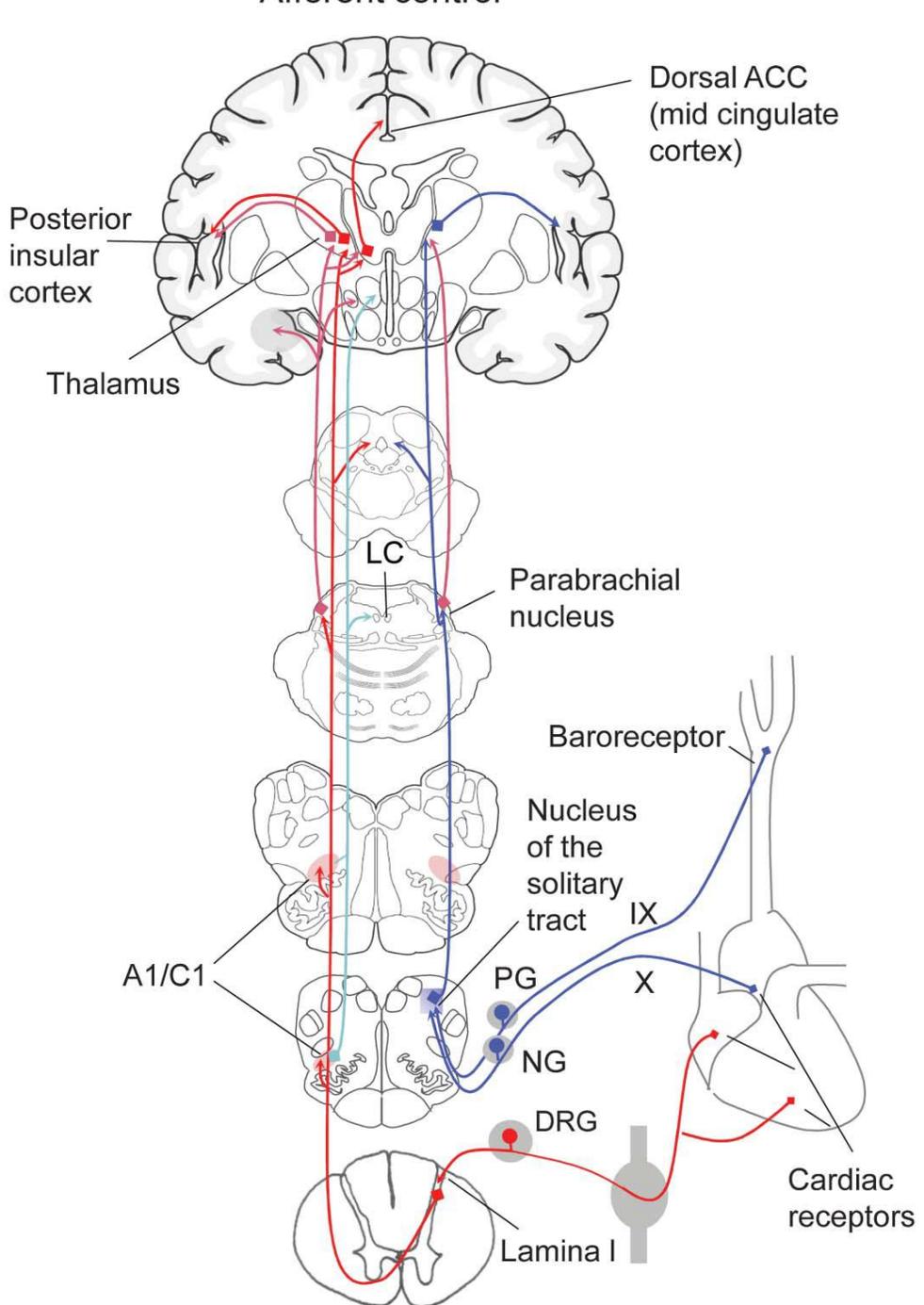
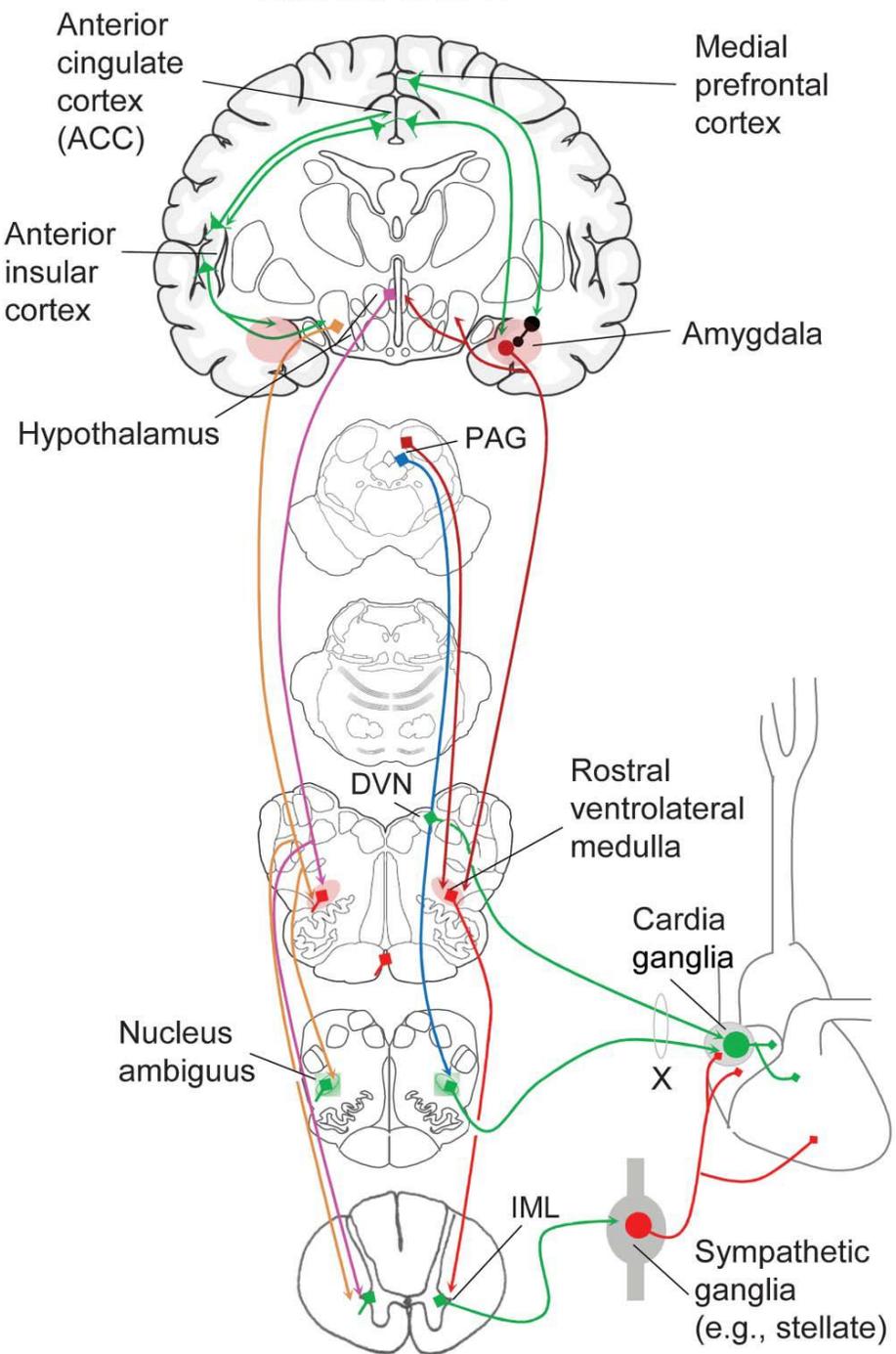
- Già nel 1903 Cushing aveva riportato la presenza di aritmie e disregolazione pressoria in corso di emorragia cerebrale
- L'edema polmonare post-comiziale è stato descritto per la prima volta da Shanahan nel 1908.
- La prima descrizione delle alterazioni elettrocardiografiche in corso di emorragia subaracnoidea risale al 1938.

Bulsara et al, J Neurosurg 2003

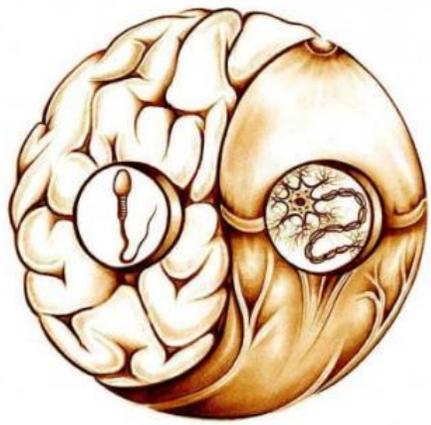


Efferent control

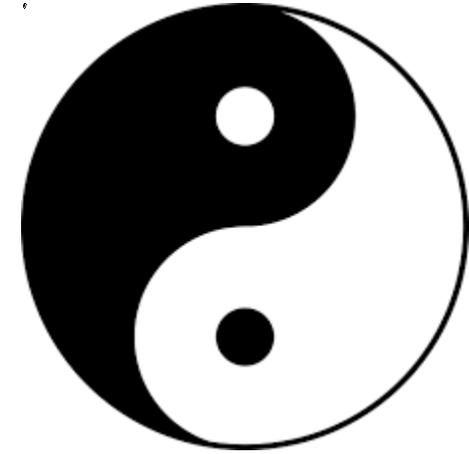
Afferent control



Palma & Benarroch, Neurology 2014



Sistema Nervoso Autonomo



Nella concezione “classica” del sistema nervoso autonomo, si ritiene che, come nella filosofia cinese dello Yin e dello Yang, i sistemi simpatico e parasimpatico agiscano sul cuore come poli opposti

Il controllo autonomico sul cuore può essere caratterizzato dall'interazione tra simpatico e parasimpatico, che in circostanze fisiologiche e fisiopatologiche può essere sincrona e sinergica

Review

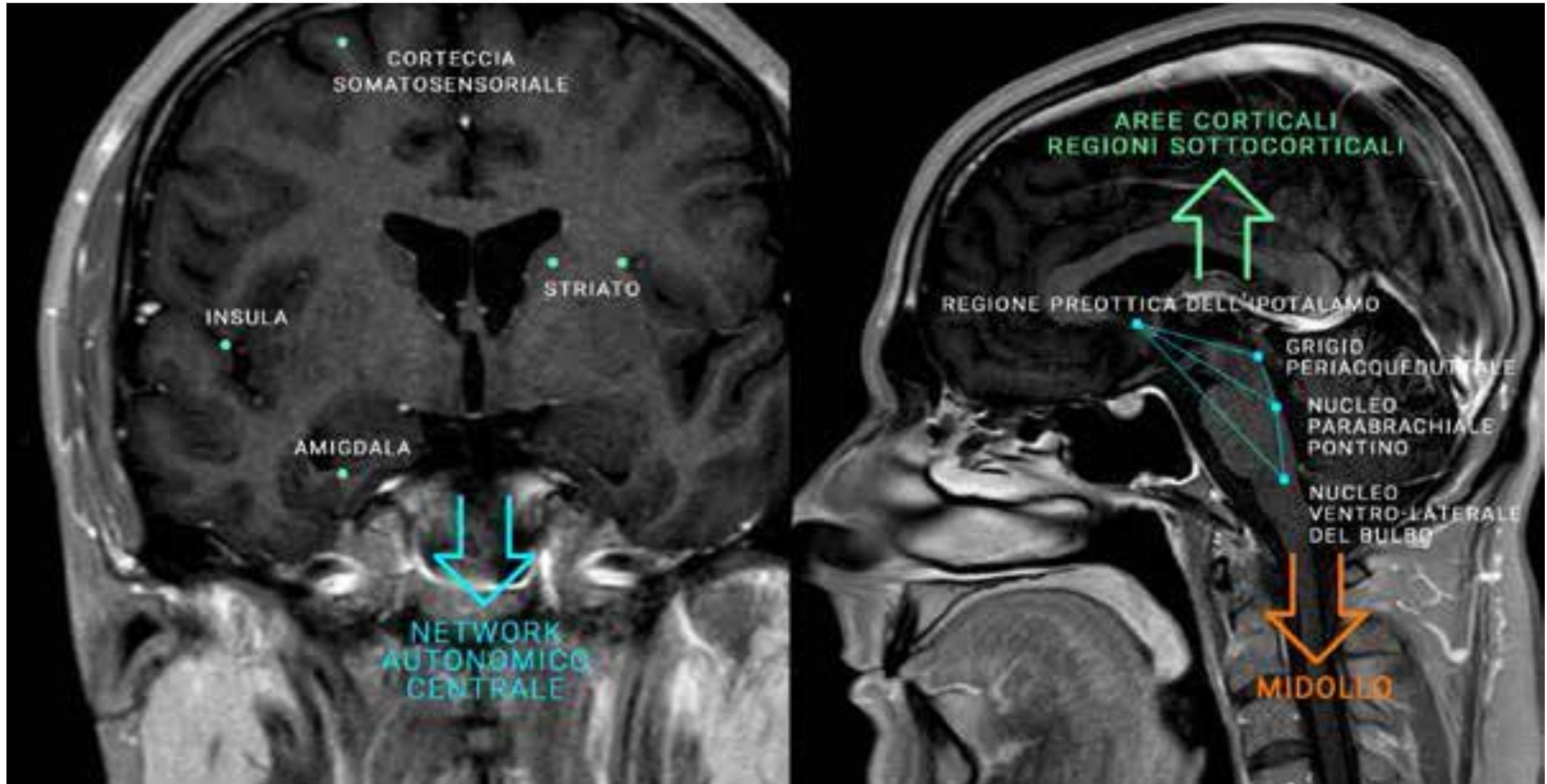
The yin and yang of cardiac autonomic control: Vago-sympathetic interactions revisited

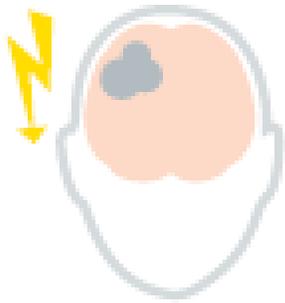
J.F.R. Paton^a, P. Boscan^c, A.E. Pickering^b, E. Nalivaiko^{d,*}



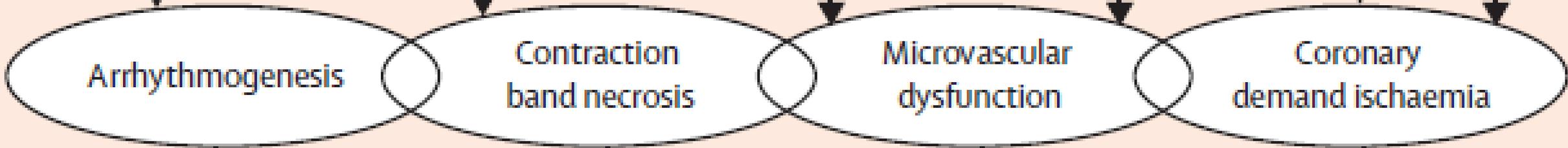
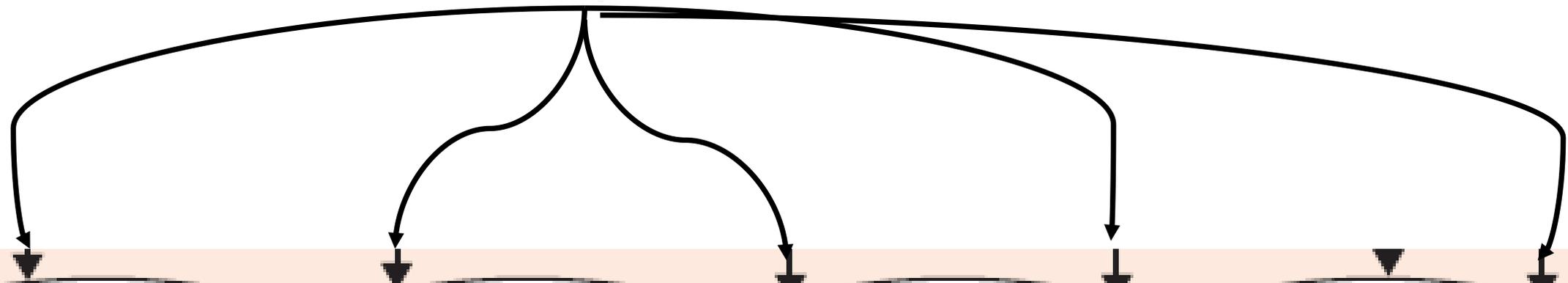
Many reflexes that are apparently protective (peripheral chemoreceptor, startle, noxious, ocular, defensive), but not regulatory or homeostatic (e.g., the baroreceptor), appear to excite both autonomic outflows to the heart simultaneously. As we have discussed, the view of the sympathetic and parasympathetic inputs to the heart as being polar antagonists is no longer tenable, rather they should be viewed as the yin and yang, different but often complimentary. In the same way that co-activation of both autonomic limbs regulates penile erection, micturition and secretory gland function, we can see that the control of cardiac function is often regulated by the sympathetic and parasympathetic systems in tandem. Co-activation of cardiac vagal and sympathetic fibers can produce bradycardia (e.g., peripheral chemoreceptor), tachycardia (somatic nociceptors) or biphasic responses in heart rate (e.g., startle). The possible physiological relevance of co-activation has been discussed, but it is clear that this may also have pathophysiological significance as it is potentially pro-arrhythmic. Interpretation

Network Autonomico Centrale





Ischaemic brain injury



Variable clinical manifestations of the stroke–heart syndrome



“VOODOO” Death



In records of anthropologists and others who have lived with primitive people in widely scattered parts of the world is the testimony that when subjected to spells or sorcery or the use of “black magic” men may be brought to death. Among the natives of South America and Africa, Australia, New Zealand, and the islands of the Pacific, as

nearby Haiti, “voodoo” death has been reported by apparently competent observers. The phenomenon is so extraordinary and so foreign to the experience of civilized people that it seems incredible; certainly if it is authentic it deserves careful consideration. I propose to recite instances of this mode of death, to inquire whether reports of the phenomenon are trustworthy, and to examine a possible explanation of it if it should prove to be real.

Walter
Bradford
Cannon

Pioneer

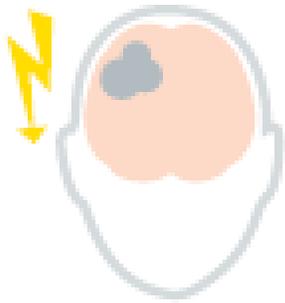
Physiologist of
Human Emotions



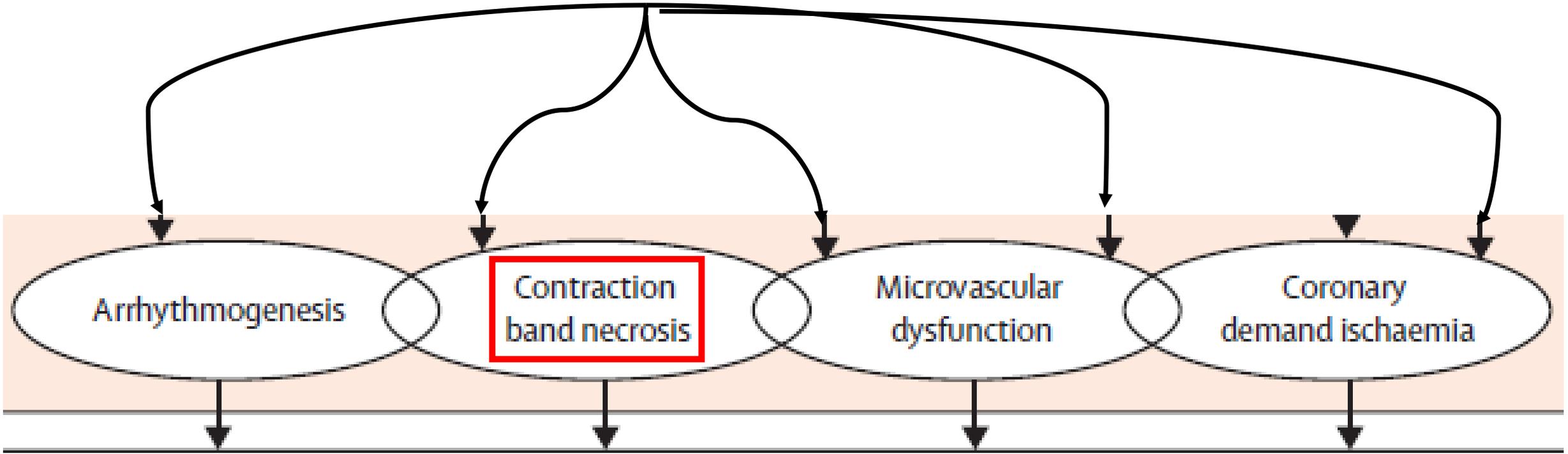
Psychosom Med. 1957



L'ESA, così come il trauma cranico, induce un significativo rilascio di catecolamine nel torrente ematico nei primi minuti dopo l'evento acuto, con immediate conseguenze cardiache, determinanti aritmie (prevalentemente di natura ipercinetica sopraventricolare o ventricolare), rilascio di enzimi miocitolitici e possibile riduzione dell'inotropismo miocardico

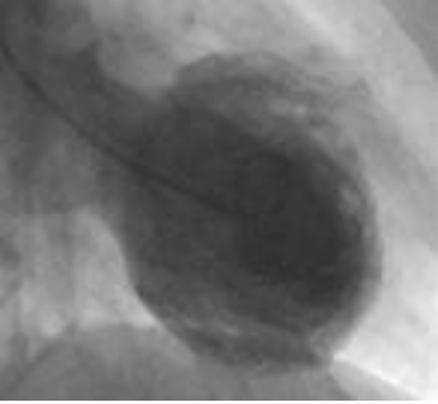


Ischaemic brain injury



Variable clinical manifestations of the stroke–heart syndrome

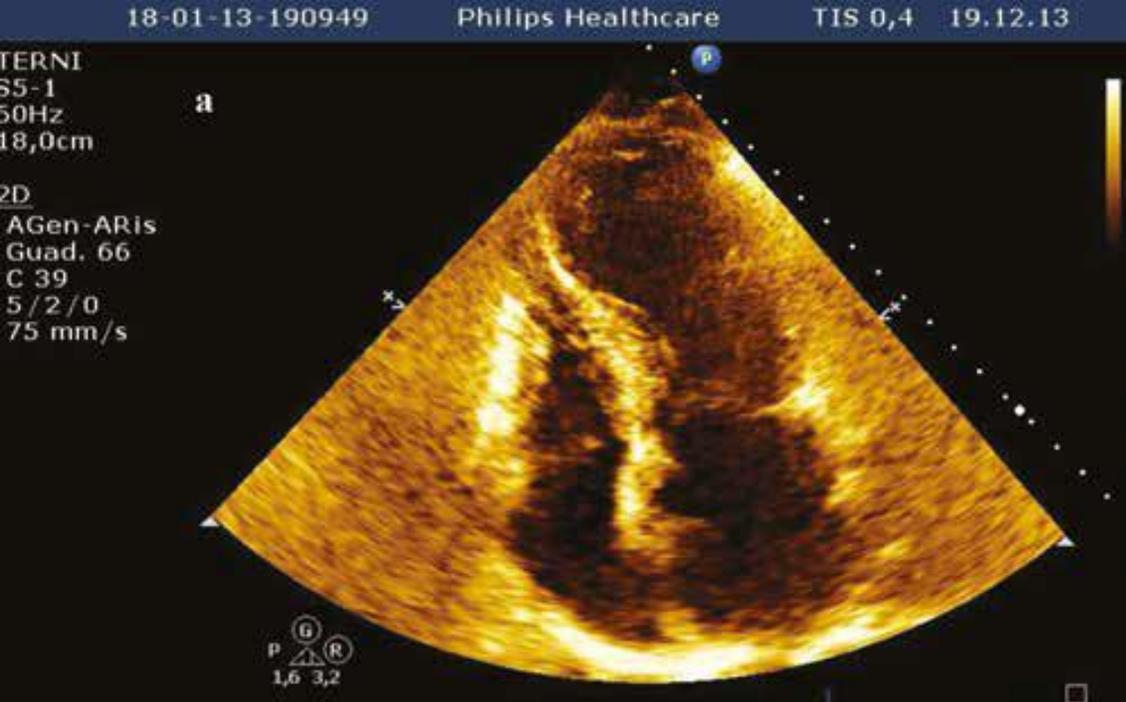




Sindrome di Tako-Tsubo

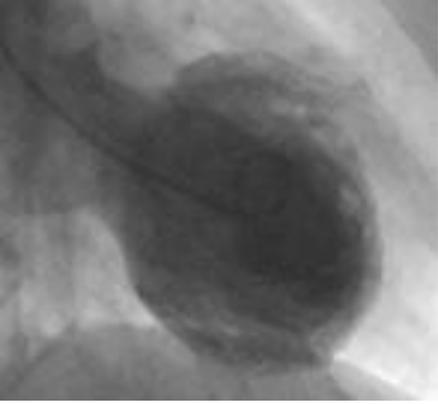


Stunning miocardico neurogeno (SMN): danno cardiaco neurologicamente indotto, reversibile, inducente disfunzione ventricolare sinistra (e conseguenti alterazioni emodinamiche), con aumentato rischio di aritmie



Estesa riduzione della cinesi miocardica in sede medio-apicale, probabilmente in relazione alla distribuzione delle terminazioni nervose miocardiche simpatiche con ipercinesia compensatoria dei segmenti basali

Più raramente i segmenti coinvolti dall'ipo-acinesia contrattile sono quelli medio-basali, identificando la variante "reverse" Tako-Tsubo

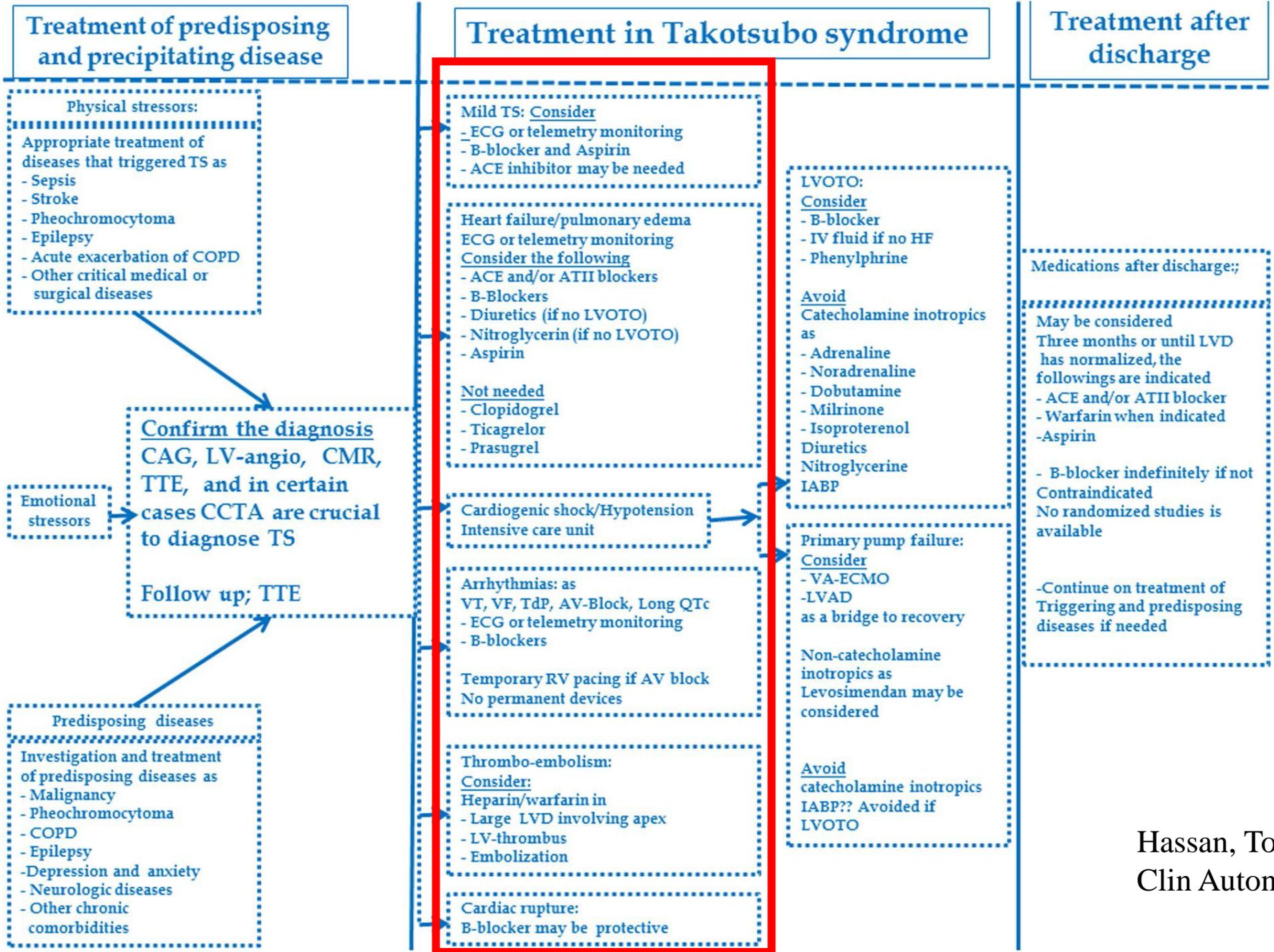


Sindrome di Tako-Tsubo

Criteri diagnostici Mayo Clinic aggiornati:



- Transitoria alterazione della cinesi dei segmenti medi del ventricolo sinistro con o senza coinvolgimento dei distretti apicali, con distribuzione non coincidente al territorio di distribuzione di una singola coronarica epicardica
- presenza di fattore scatenante emotivo o fisico
- assenza di malattia coronarica ostruttiva all'esame angiografico
- alterazioni ECG di nuova insorgenza con lieve incremento della troponina, che nella maggioranza dei casi risulta concomitante all'innalzamento dei livelli ematici di catecolamine (di-idrossi-fenilalanina, adrenalina, noradrenalina e dopamina).



Cardiogenic shock/Hypotension
Intensive care unit

Arrhythmias: as
VT, VF, TdP, AV-Block, Long QTc
- ECG or telemetry monitoring
- B-blockers

Temporary RV pacing if AV block
No permanent devices

Heart failure/pulmonary edema
ECG or telemetry monitoring
Consider the following
- ACE and/or ATII blockers
- B-Blockers
- Diuretics (if no LVOTO)
- Nitroglycerin (if no LVOTO)
- Aspirin

Not needed
- Clopidogrel
- Ticagrelor
- Prasugrel

Thrombo-embolism:

Consider:

Heparin/warfarin in

- Large LVD involving apex
- LV-thrombus
- Embolization

Cardiac rupture:

B-blocker may be protective

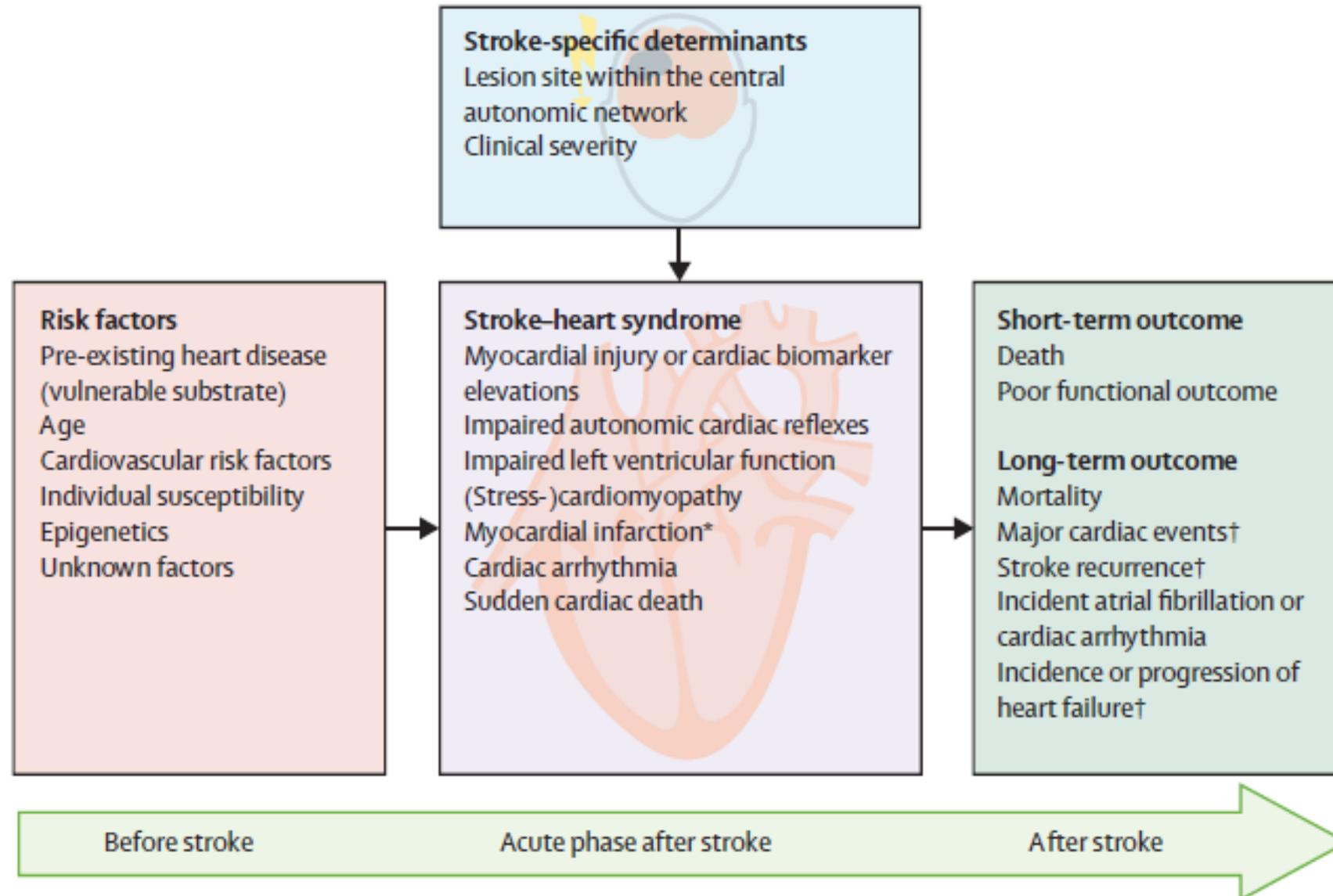
Stroke–heart syndrome: clinical presentation and underlying mechanisms

Jan F Scheitz, Christian H Nolte, Wolfram Doehner, Vladimir Hachinski, Matthias Endres

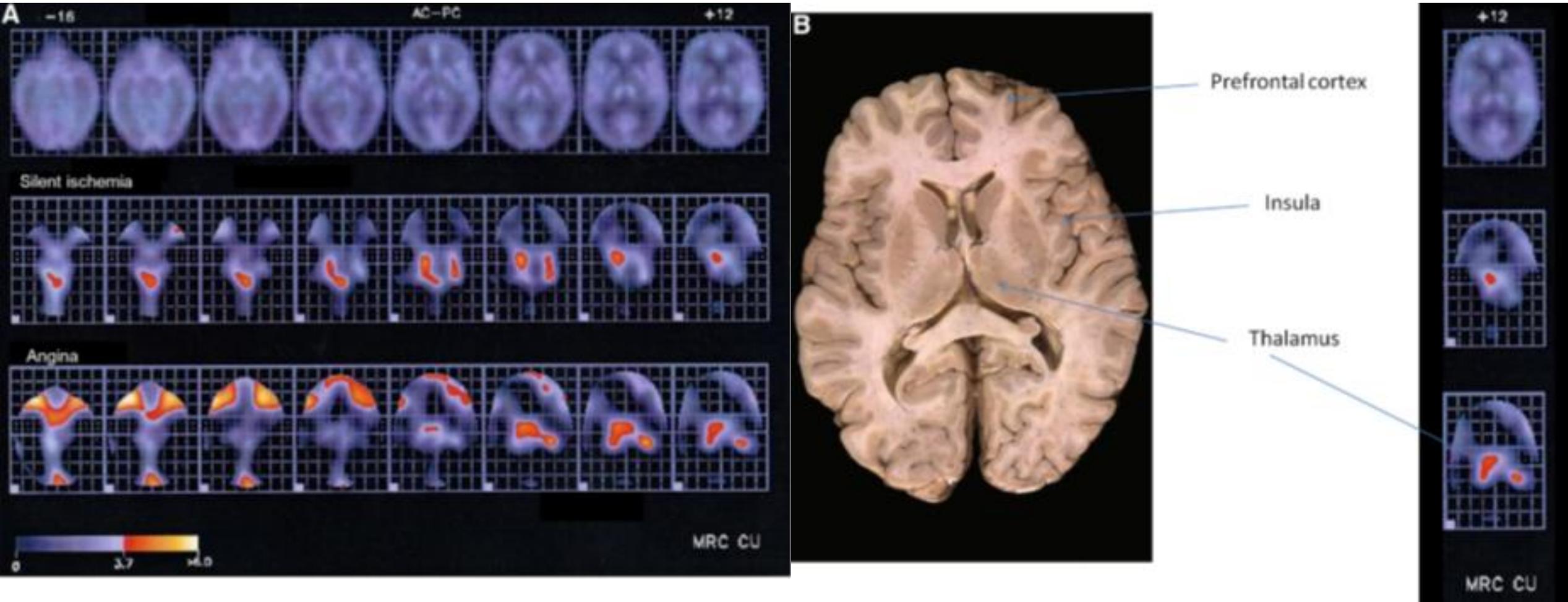
Lancet Neurol 2018



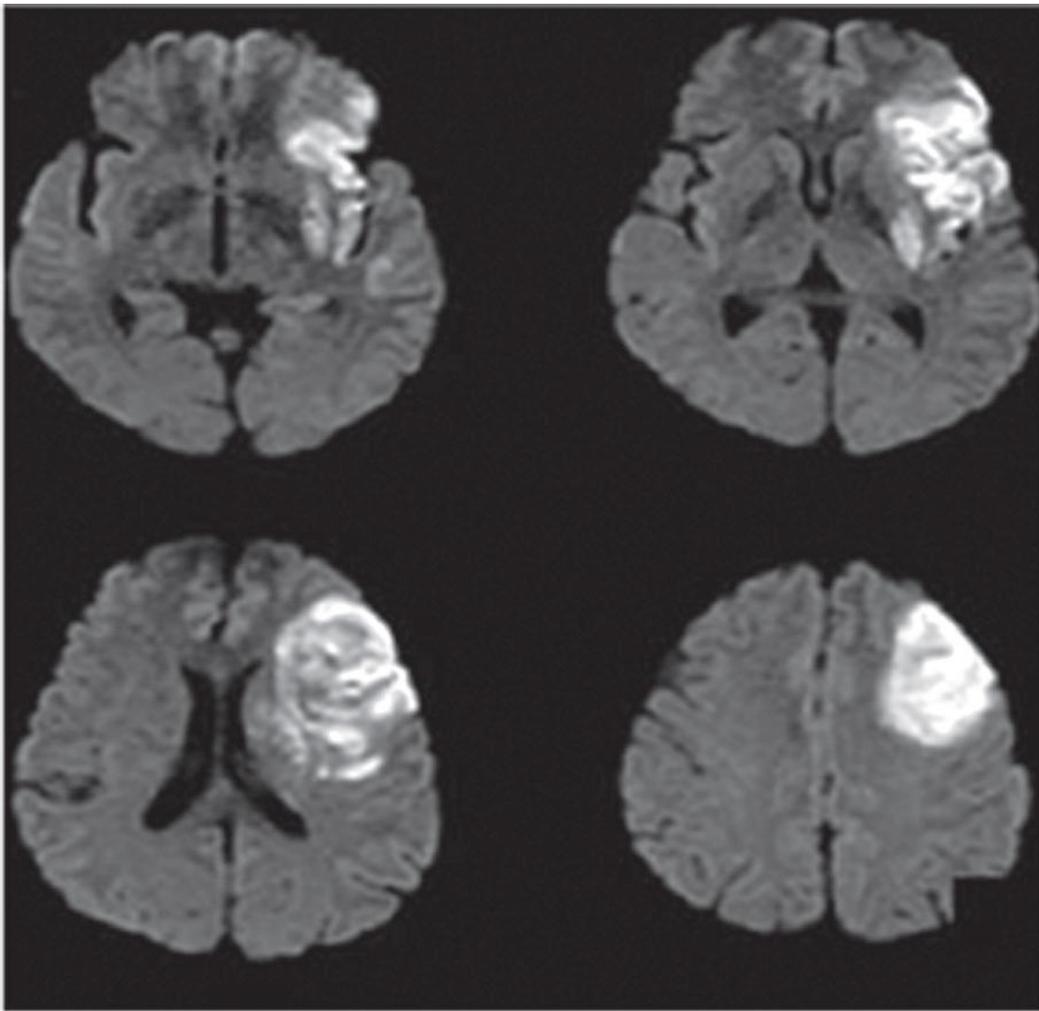
Review

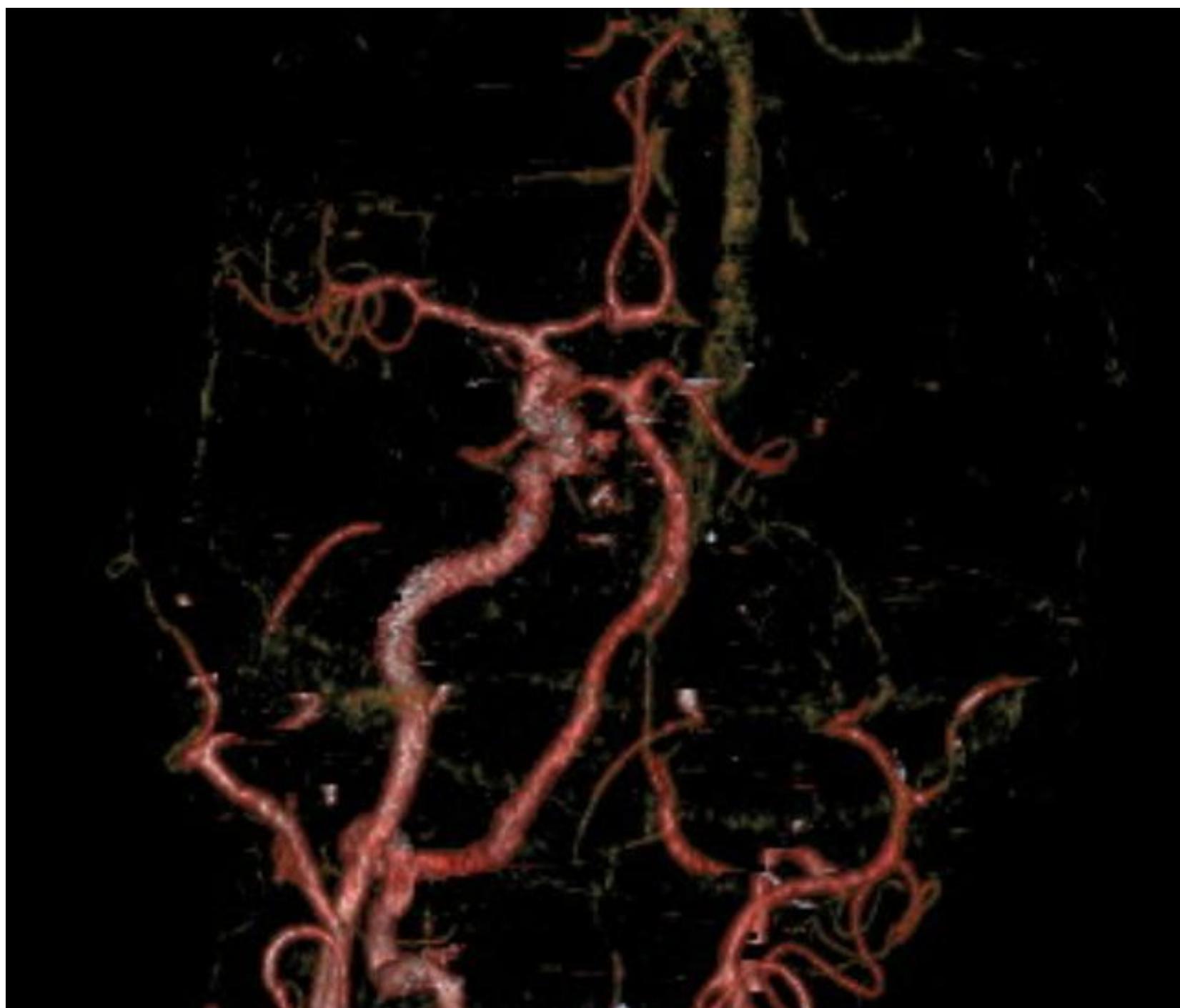


Corteccia insulare e funzione cardiaca



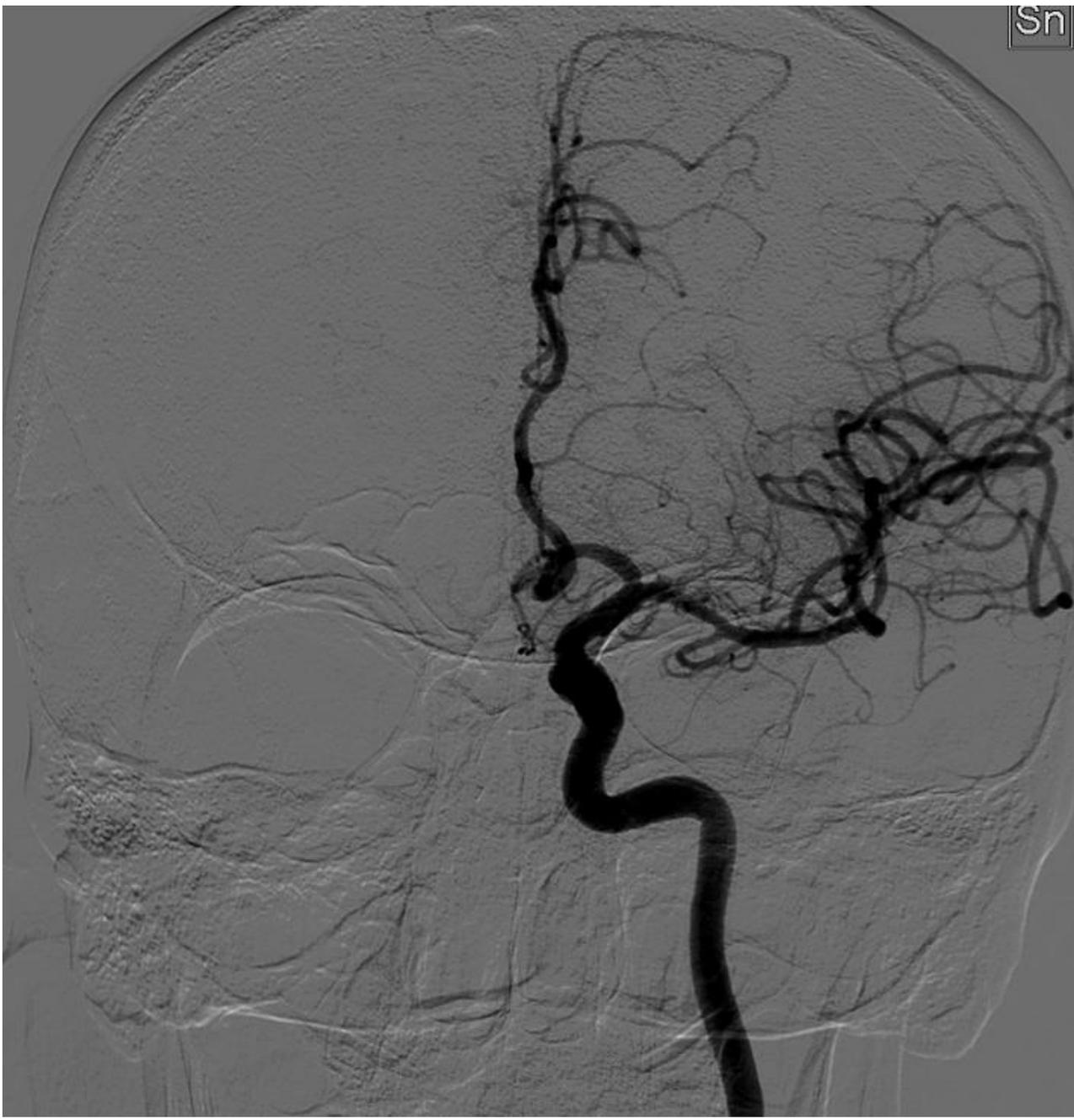
Corteccia insulare e funzione cardiaca

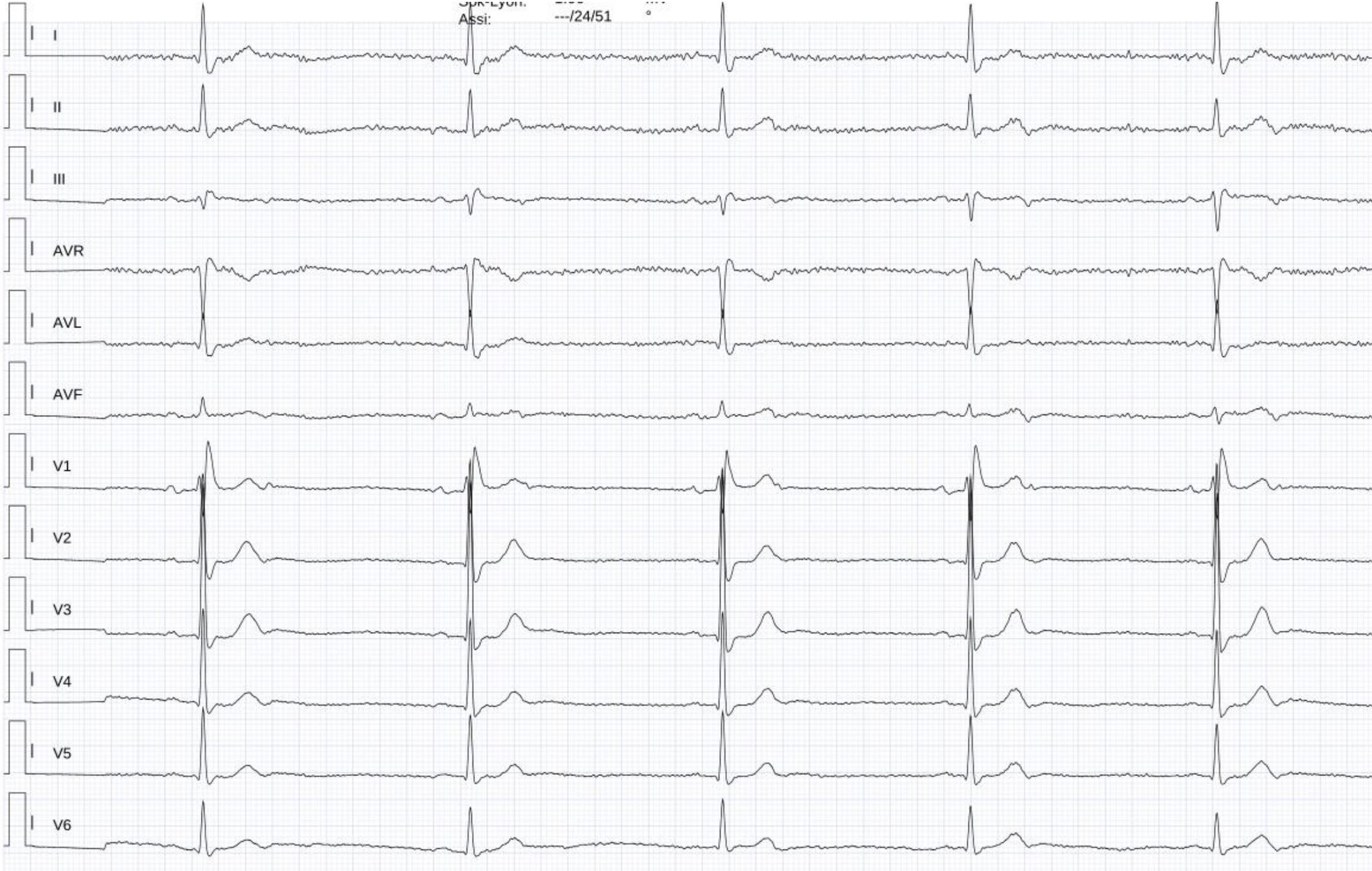


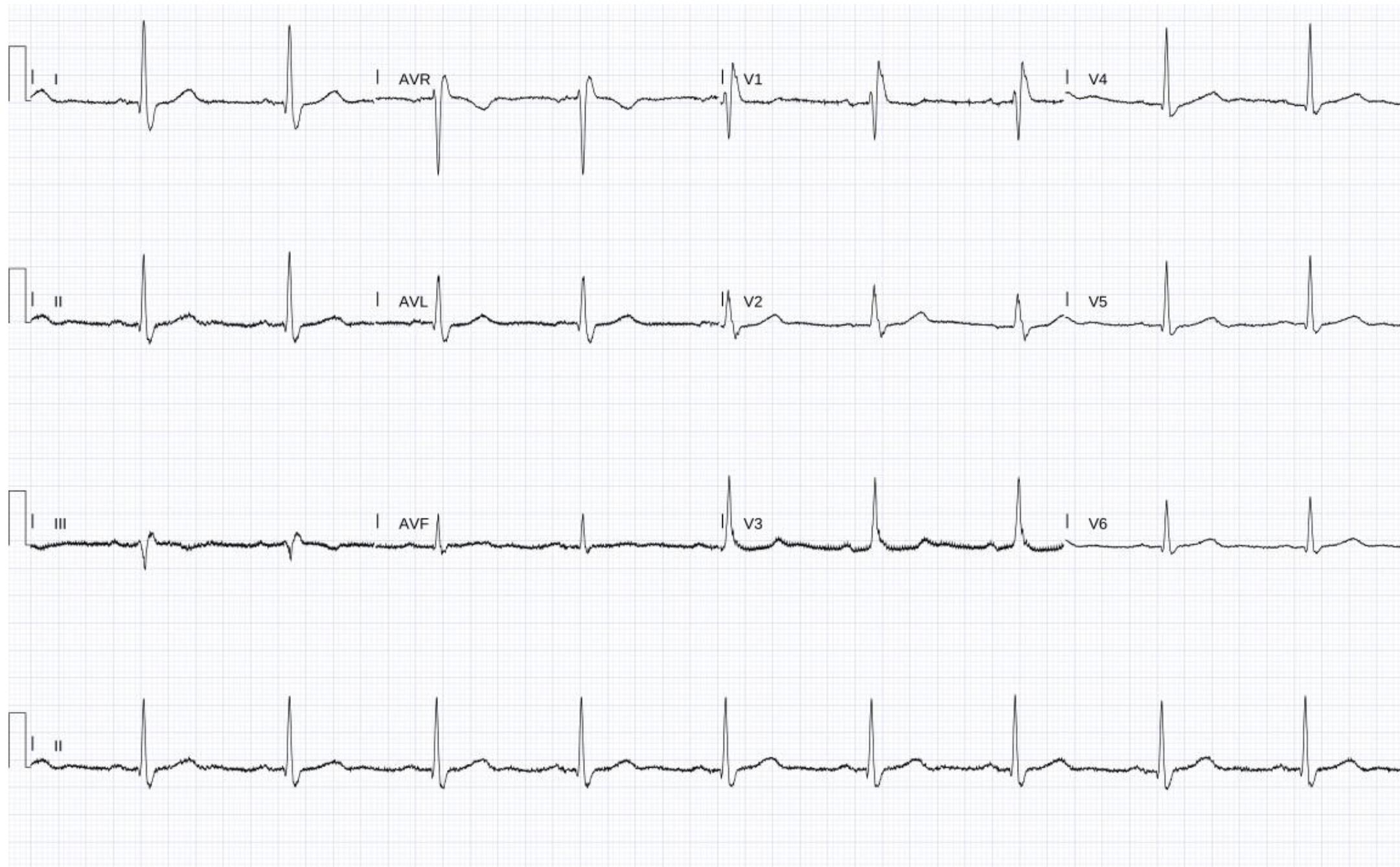


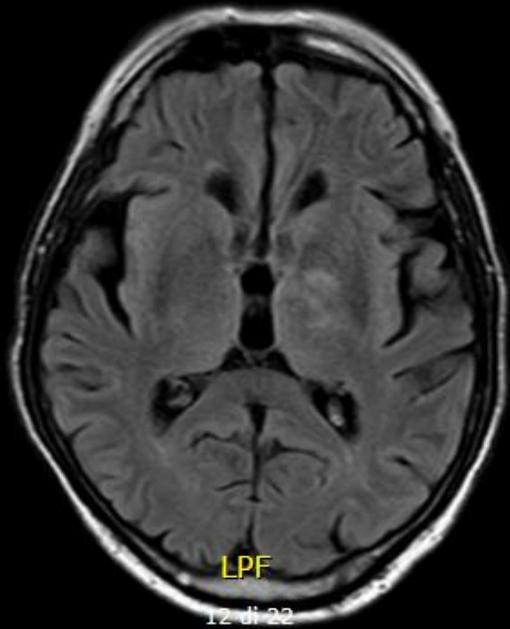


Sn









LPF

12 di 22

RAH

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P
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F

-24.96

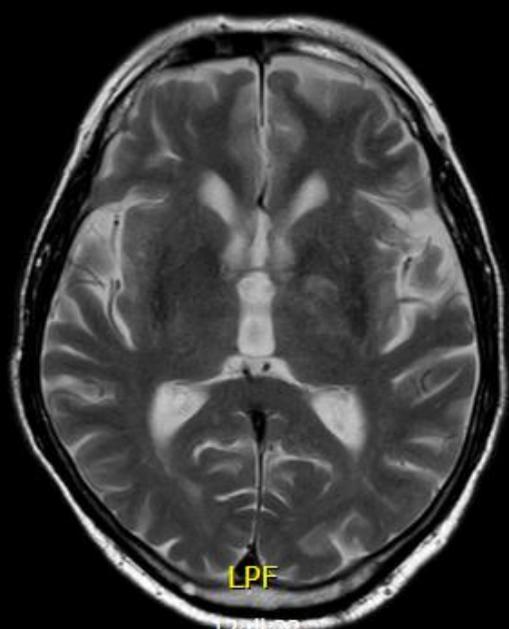


LPF

12 di 22

RAH

4: DWI_og (Serie 301)



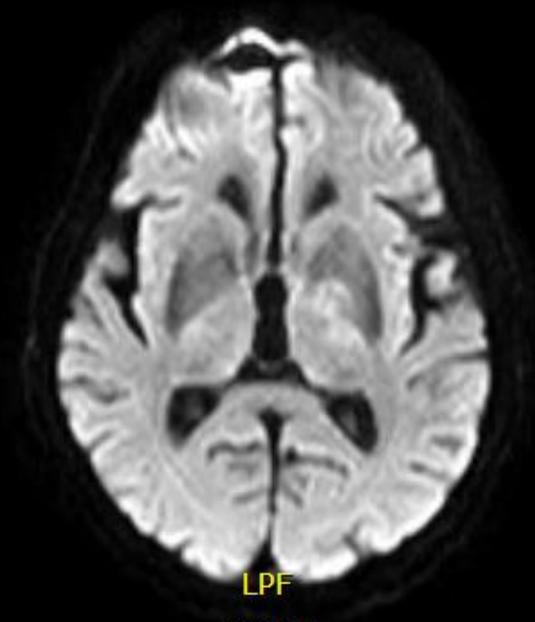
LPF

12 di 22

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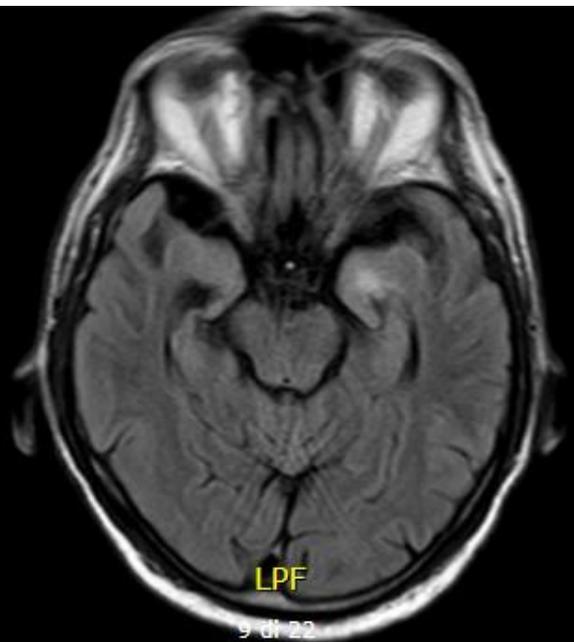
P
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-24.96



LPF

24 di 44



LPH

9 di 22

RAH

-6.96

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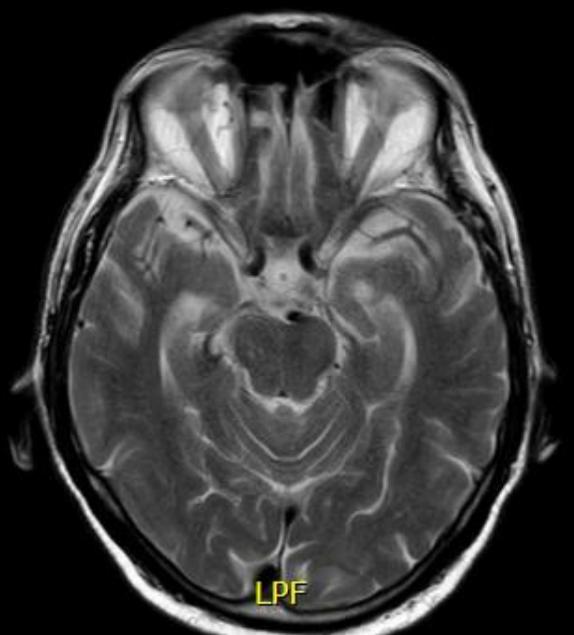


LPH

9 di 22

RAH

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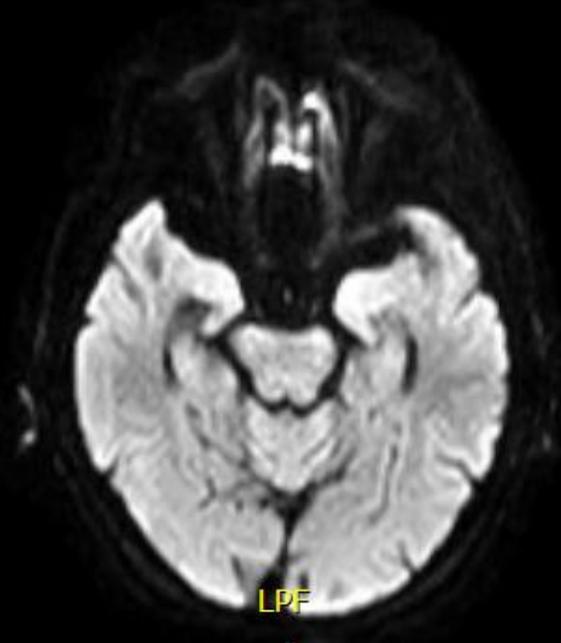


LPH

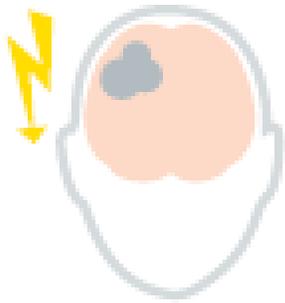
-6.96

A
L
H

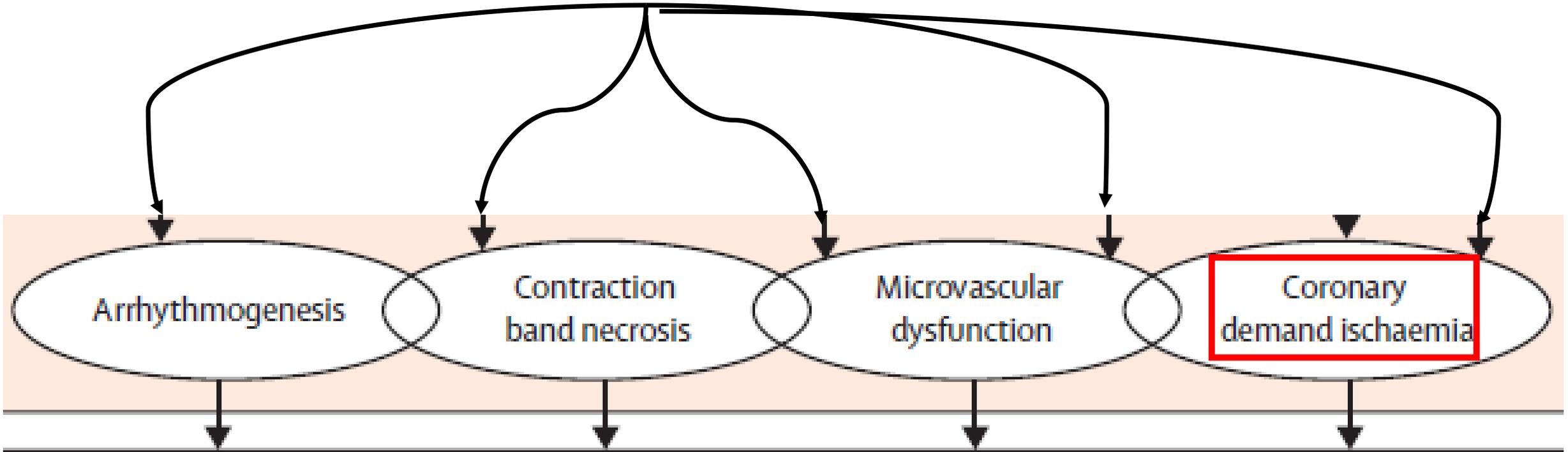
P
R
F



LPH



Ischaemic brain injury



Arrhythmogenesis

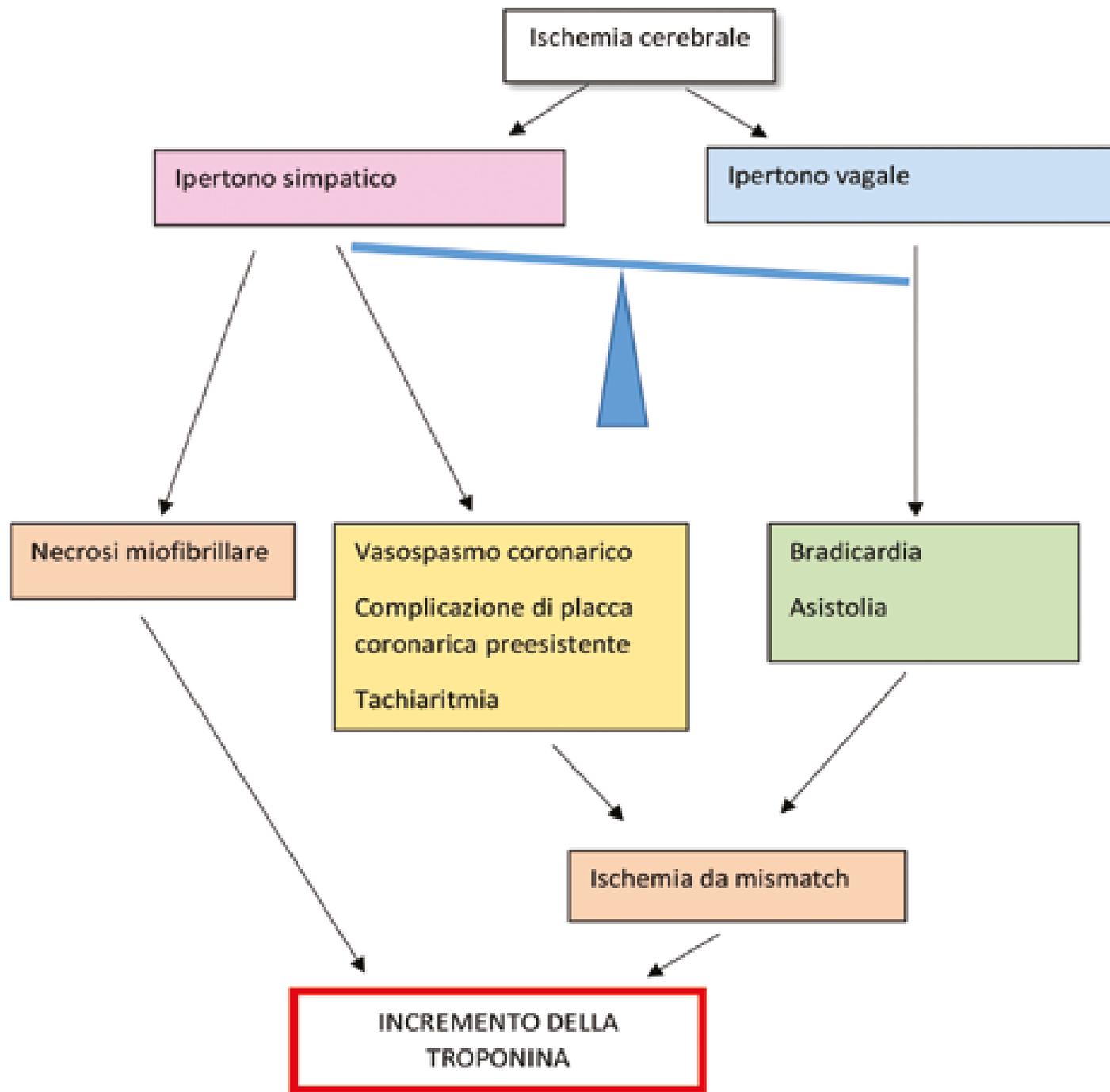
Contraction
band necrosis

Microvascular
dysfunction

Coronary
demand ischaemia

Variable clinical manifestations
of the stroke-heart syndrome



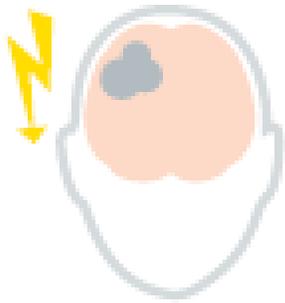


"Troponinosi"

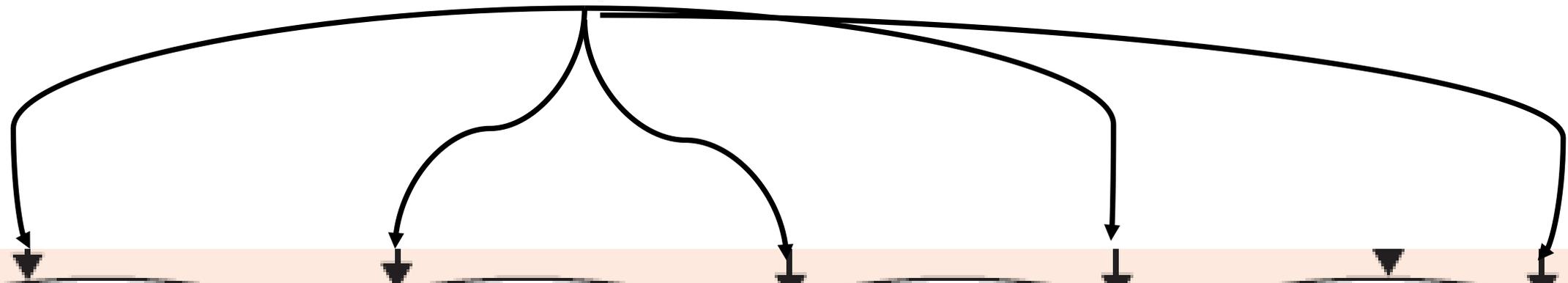
La troponina I si incrementa nel 20-40% dei casi di emorragia cerebrale e nel 15% dei casi di ictus ischemico

L'incremento correla con la gravità della lesione cerebrale e quindi della prognosi

La curva enzimatica presenta un tipico picco entro le prime 48 ore, per poi ridursi gradualmente nei giorni successivi.



Ischaemic brain injury

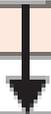
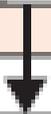


Arrhythmogenesis

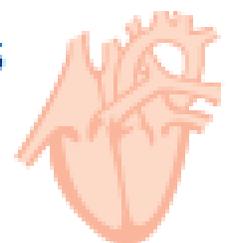
Contraction band necrosis

Microvascular dysfunction

Coronary demand ischaemia



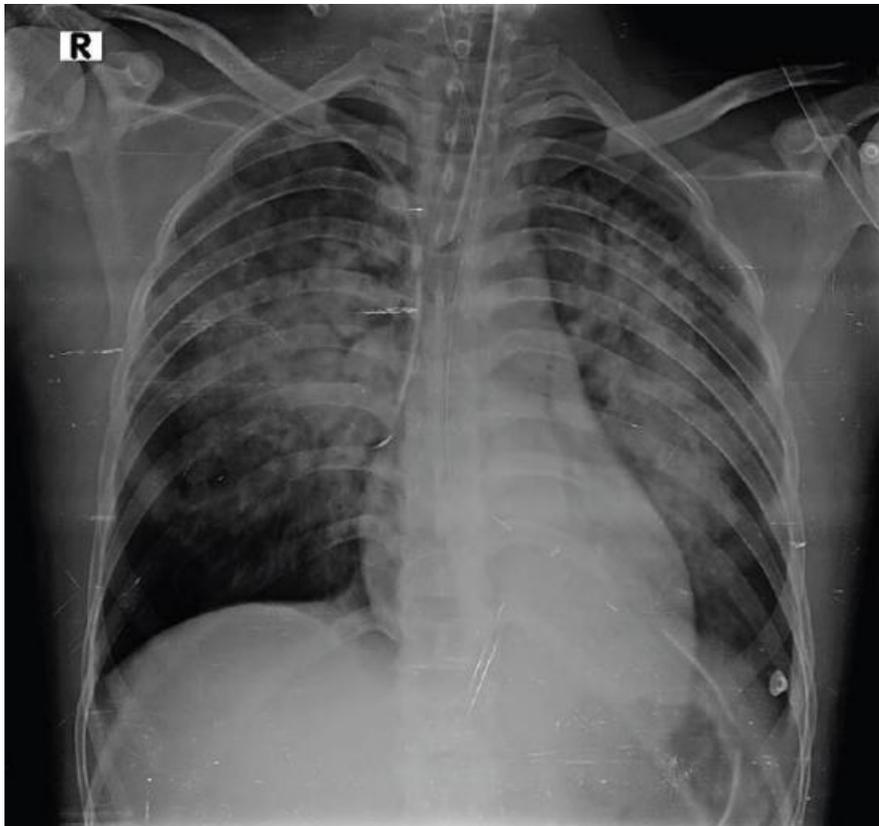
Variable clinical manifestations of the stroke-heart syndrome



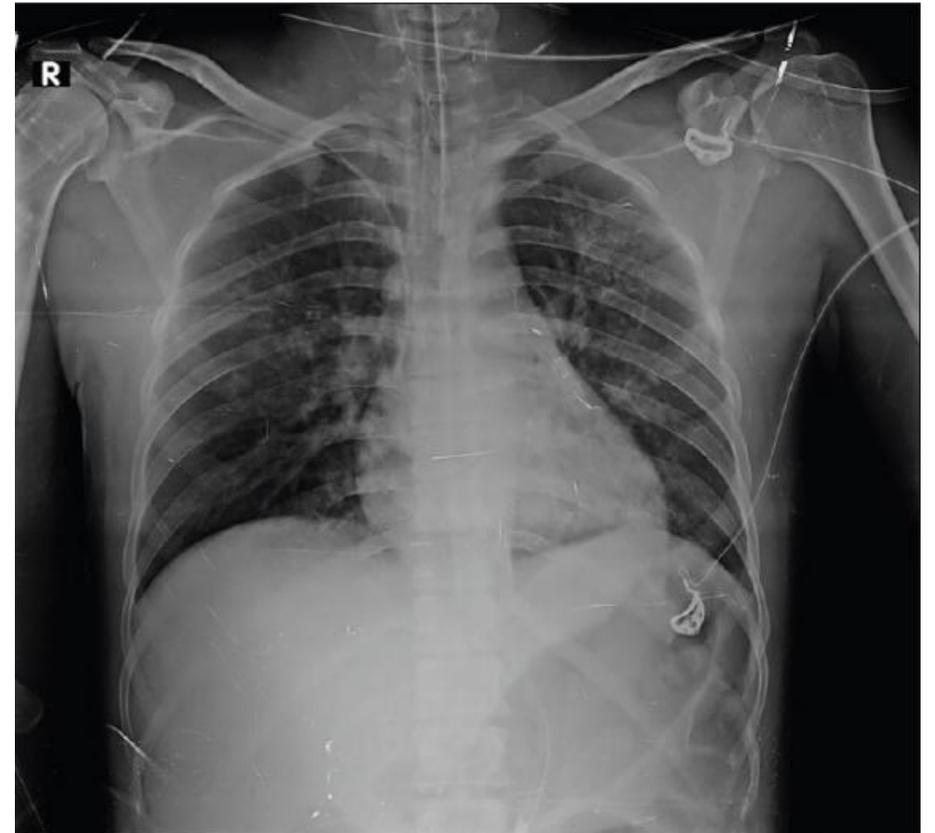


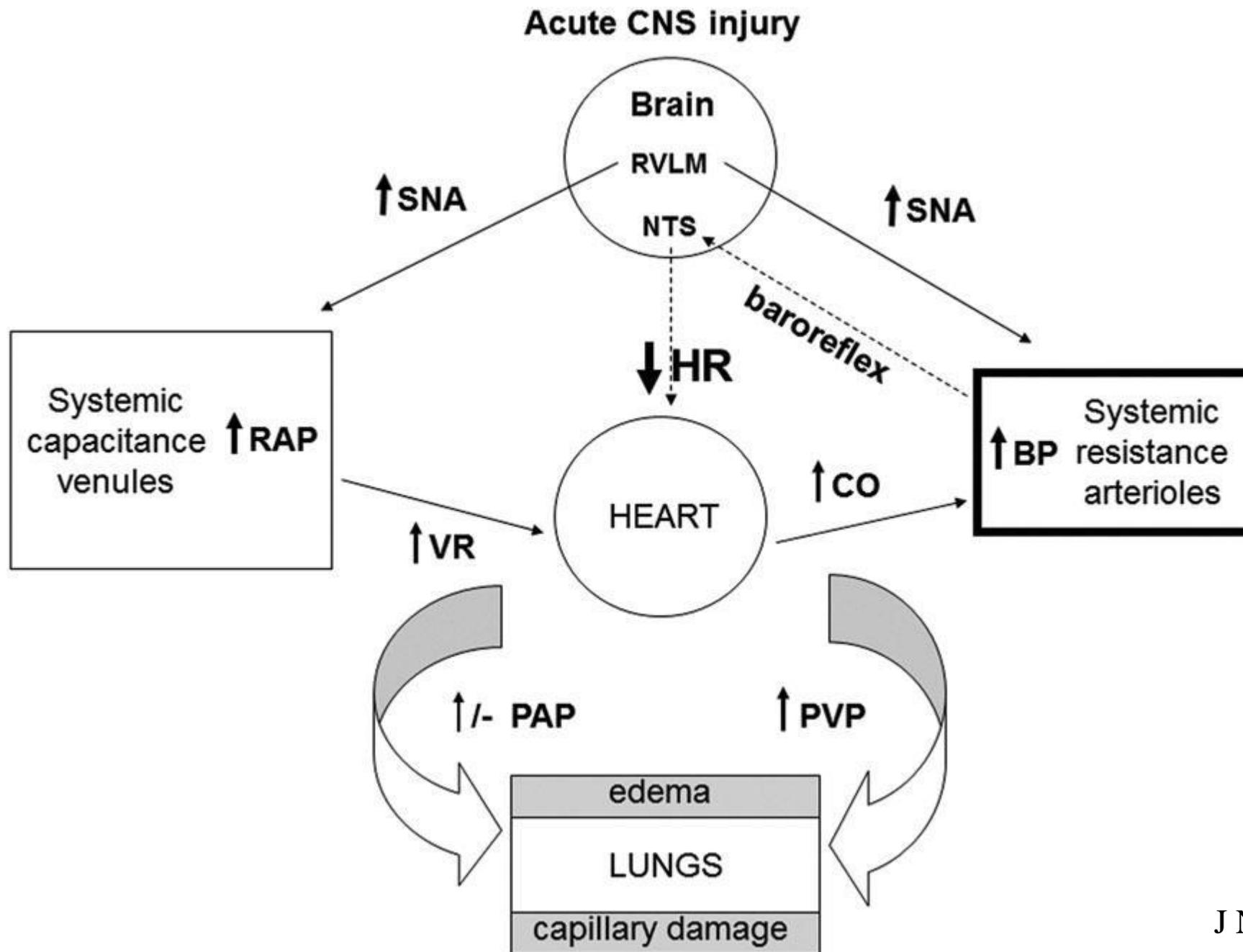
Edema Polmonare Neurogenico

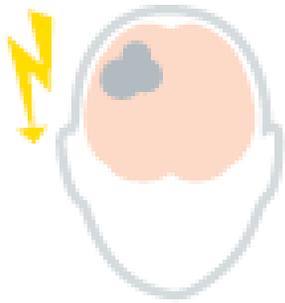
Rajagopal et al,
Indian J Crit Care Med. 2017



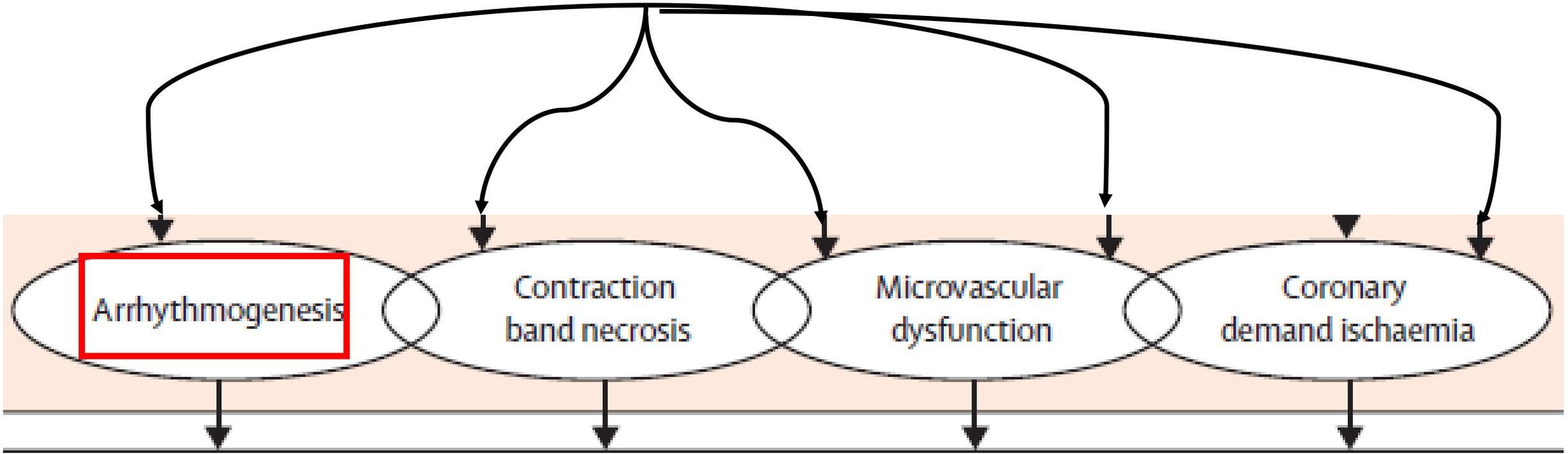
48 h







Ischaemic brain injury



Variable clinical manifestations of the stroke–heart syndrome





Aritmie

Bradicardia o Tachicardia sinusale

Fibrillazione atriale

Battiti atriali prematuri

Wondering pace-maker

Blocchi atrio-ventricolari

Aritmie ventricolari premature

Tachicaria e Fibrillazione ventricolare

Torsione di punta

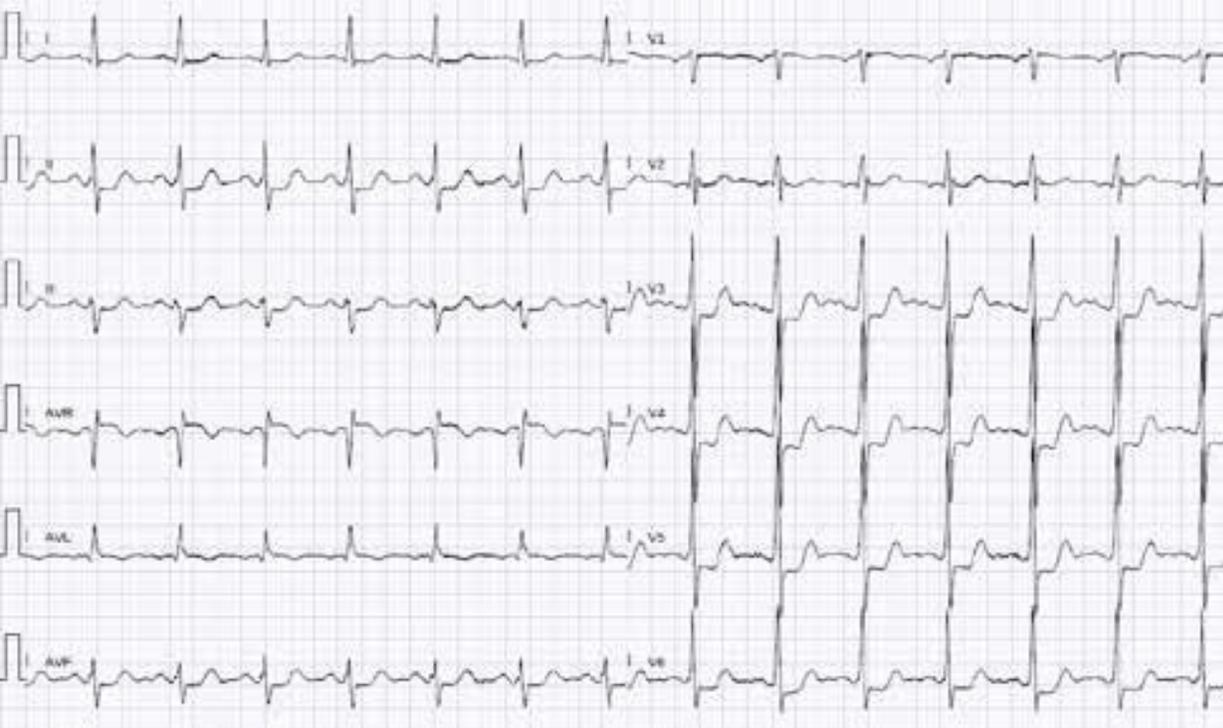


Aritmie

Emorragia capsulo-lenticolare sinistra

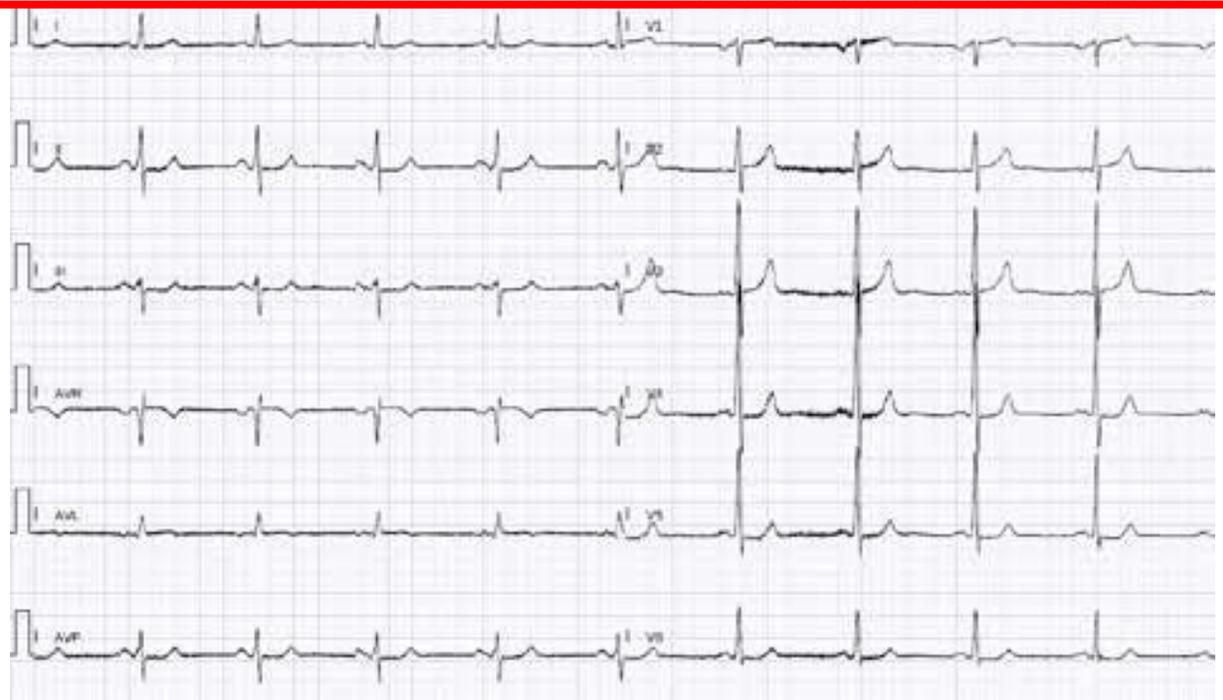


ECG: FA a risposta ventricolare controllata, scarsa progressione dell'onda R nelle precordiali, onde T negative in sede infero-laterale e intervallo QTc ai limiti superiori della norma

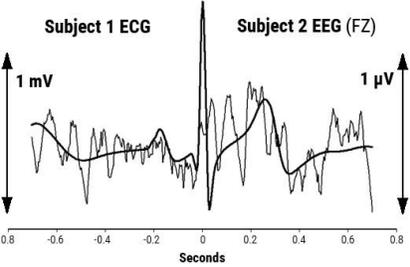


Stato epilettico generalizzato convulsivo

ECG in fase iperacuta: estese alterazioni del tratto ST-T



ECG normalizzato all'estinzione dello stato epilettico

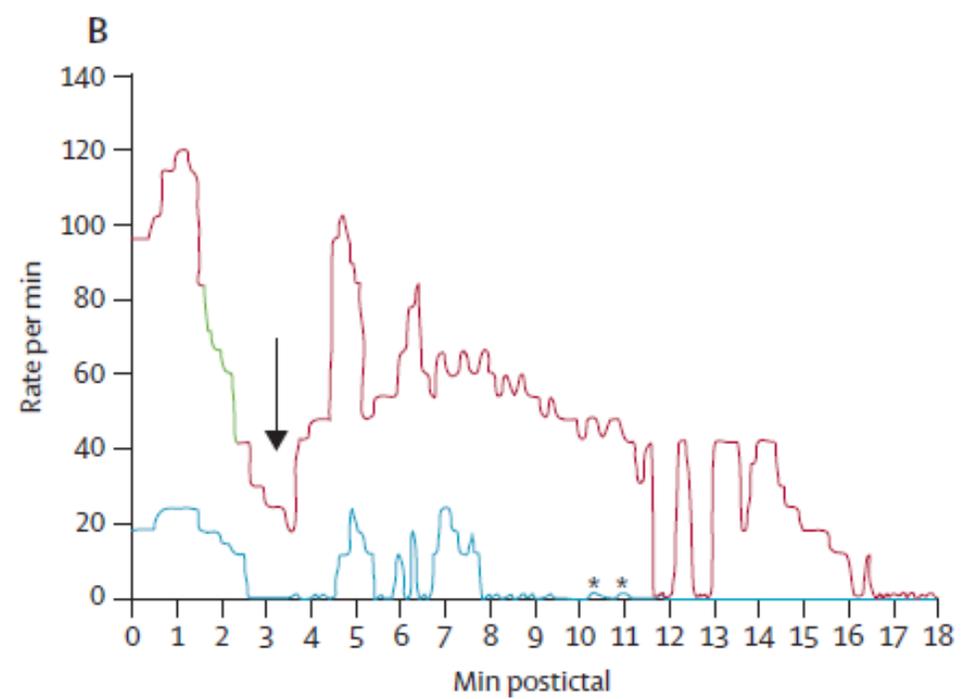
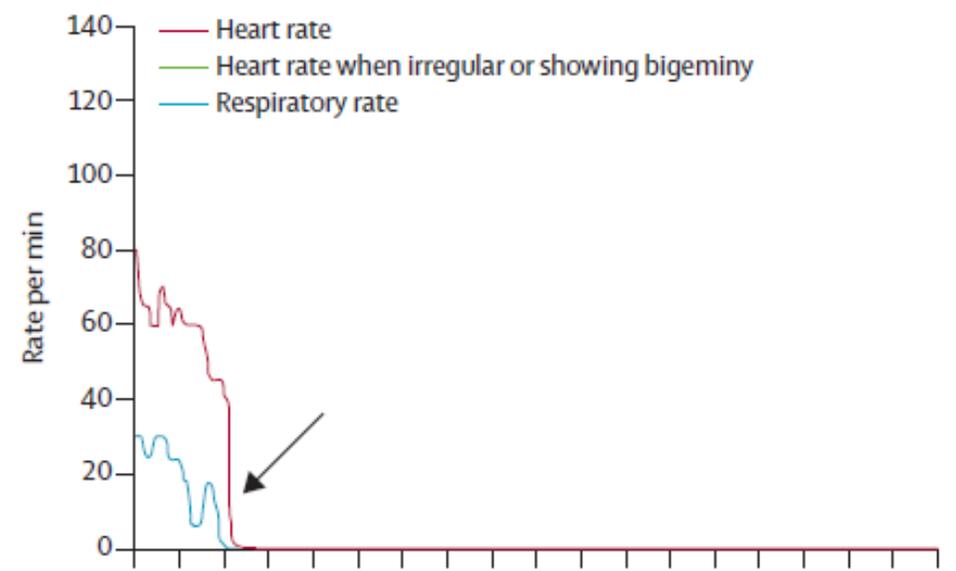
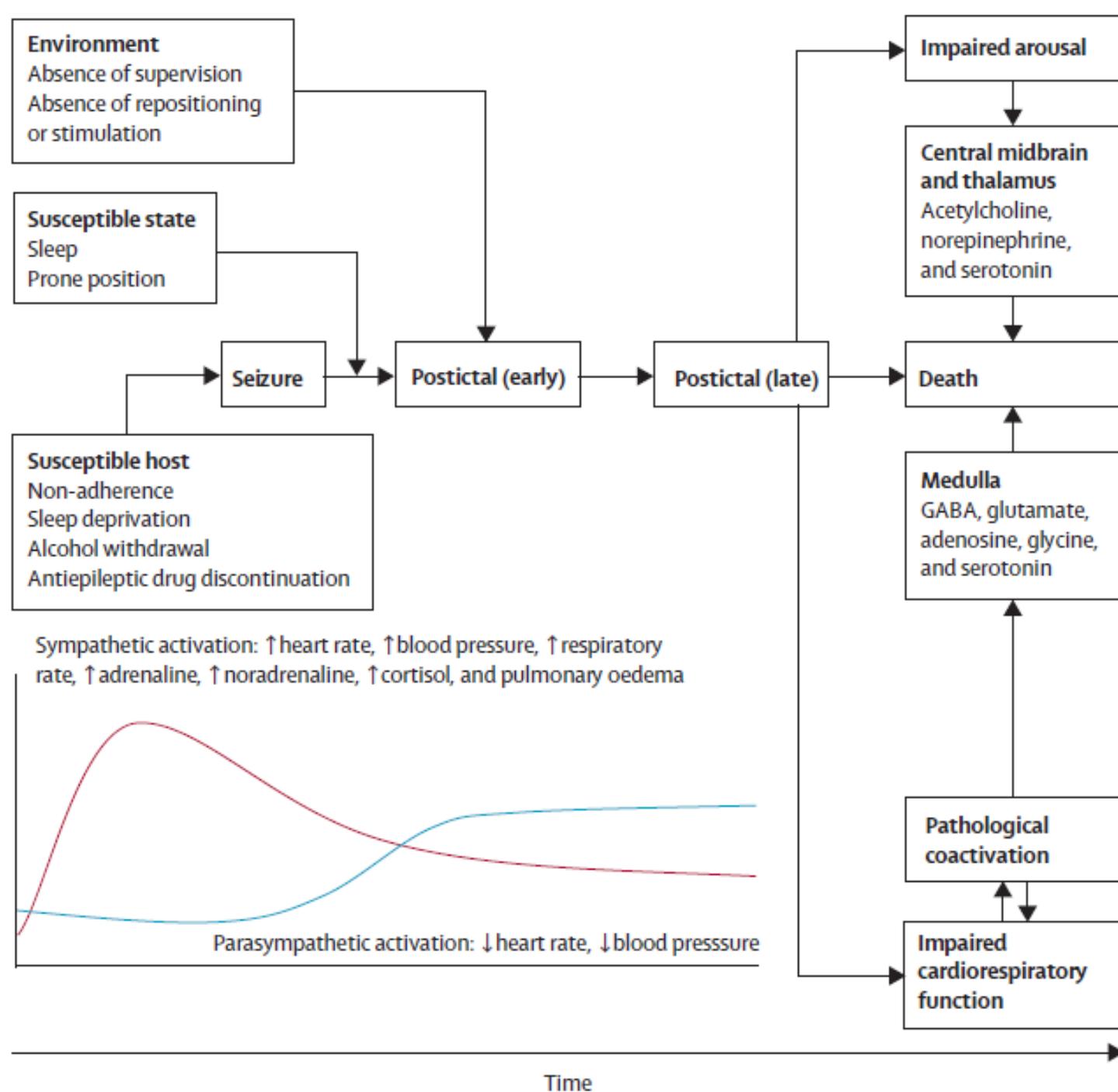


SUDEP

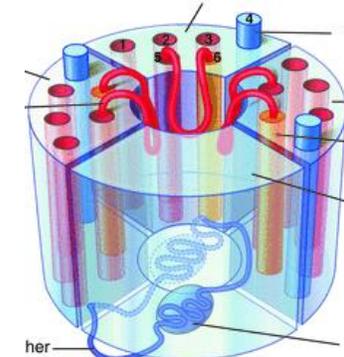
Sudden Unexpected Death in Epilepsy

Disfunzione autonoma:

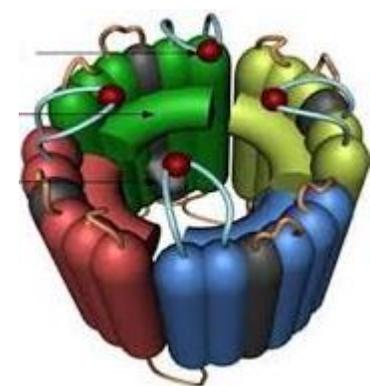
- *Heart Rate Variability*
- Tachicardia e tachiaritmia
- Bradicardia ed asistolia
- Ischemica miocardica
- Aritmie iatrogene (antiepilettici)

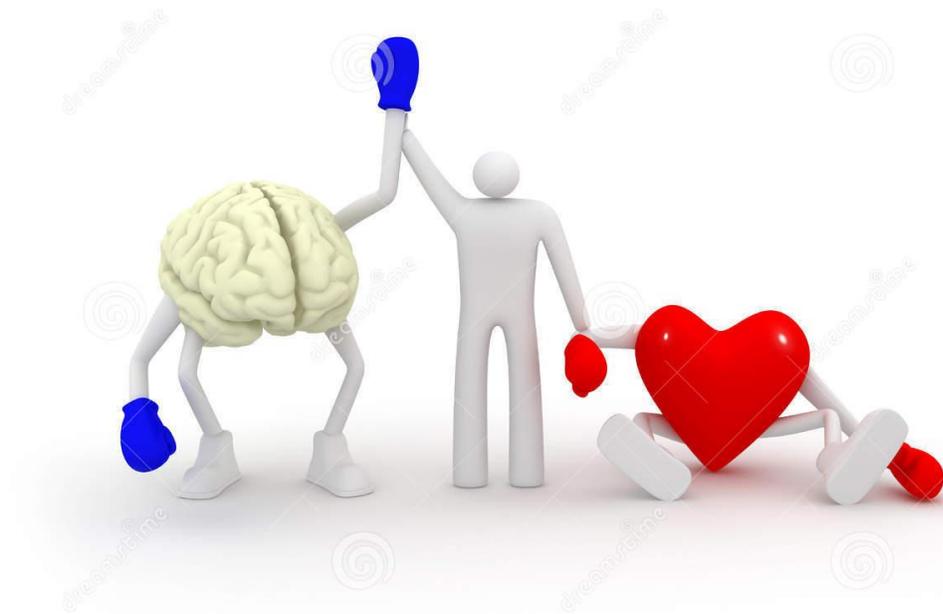
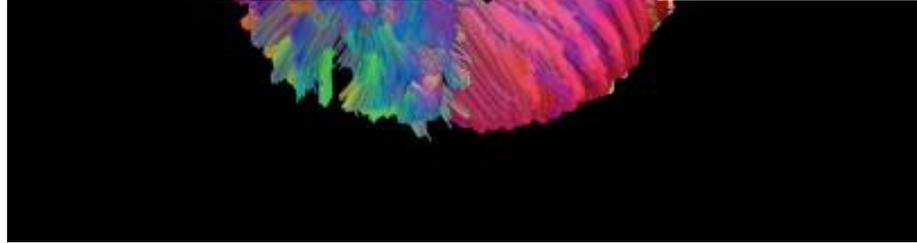
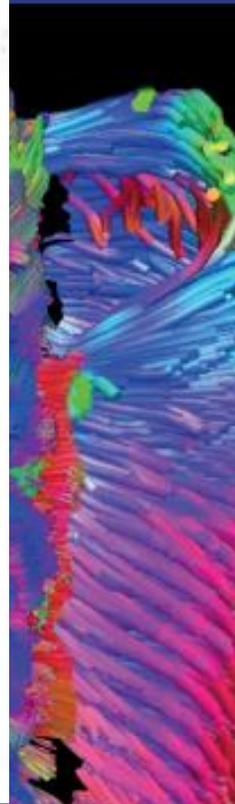
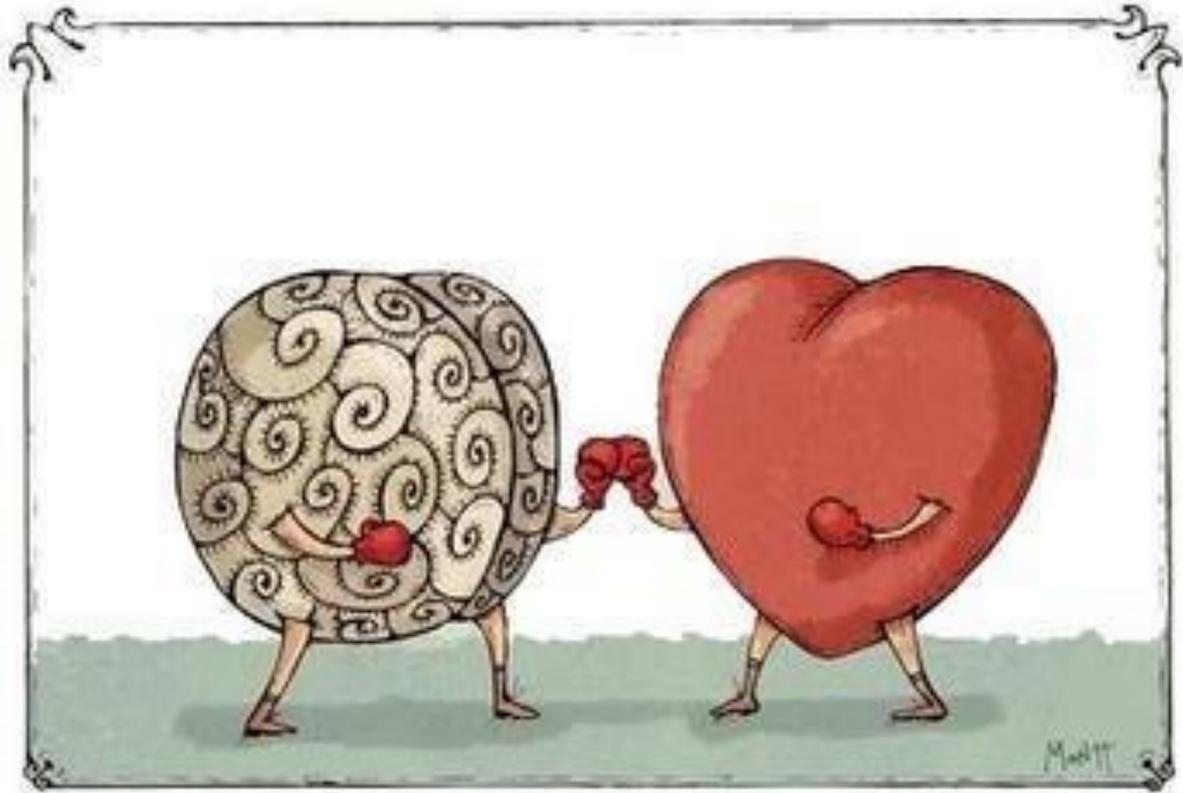


PATOLOGIA	GENE COINVOLTO	CANALE VOLTAGGIO-DIPENDENTE
Epilessia autosomica dominante notturna	CHRNA4	
Epilessia mioclonica giovanile	CHRNA2	
Epilessia generalizzata con convulsioni benigne in plus	GABRA1 GABRG2	Canali calcio
Epilessia mioclonica severa dell'infanzia	SCN1A	Canali potassio
Convulsioni neonatali familiari benigne	SCN2A	Canali sodio
Crisi febbrili senza febbre	SCN1B SCN1A KCNQ2 KCNQ2 KCNQ3 SCN2A	
Eemicrania familiare emiplegica	CACNA1A	Canali calcio
Atassia episodica	CACNA1A KCN1A	Canali calcio Canali potassio
Atassia spinocerebellare	CACNA1A KCNC3	Canali calcio Canali potassio
Iperekplexia	GLRA1	Canali cloro
Paralisi periodica	SCN4A CACNA1S	Canali sodio Canali calcio
Miotonia	KCNJ2 SCN4A	Canali potassio Canali sodio
Sindrome miastenica congenita	CIC1 CHRNA1 CHRNA1 CHRNE CHRND	Canali calcio
Ipertermia maligna	CACNA1S	Canali calcio



Canale coinvolto	Patologia	Ereditarietà	Locus genetico	Gene	Proteina
Potassio	SQT1	Autosomica dominante	7 q35	KCNH2	hERG Kv11.1
	SQT2	Autosomica dominante	11p15.5	KCNQ1	Kv7.1
	SQT3	Autosomica dominante	17q23	KCNJ2	Kv2.1 Kir2.1
Calcio	S. Brugada e SQT4	Autosomica dominante	2p13.3	CACNA1C	Cav 1.2
	S. Brugada e SQT5	Autosomica dominante	10 p12.33	CACMB2b	Volt-dip b-2
	SQT 6	Autosomica dominante	7q21-q22	CACNA2D1	Volt-dip a2/d1





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