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APRILE 2025

4° Congresso Nazionale SIONG (Società Italiana OtoNeuroGeriatria)

Il deficit cognitivo è influenzato dal rapporto cuore/cervello?

3 Aprile 2025

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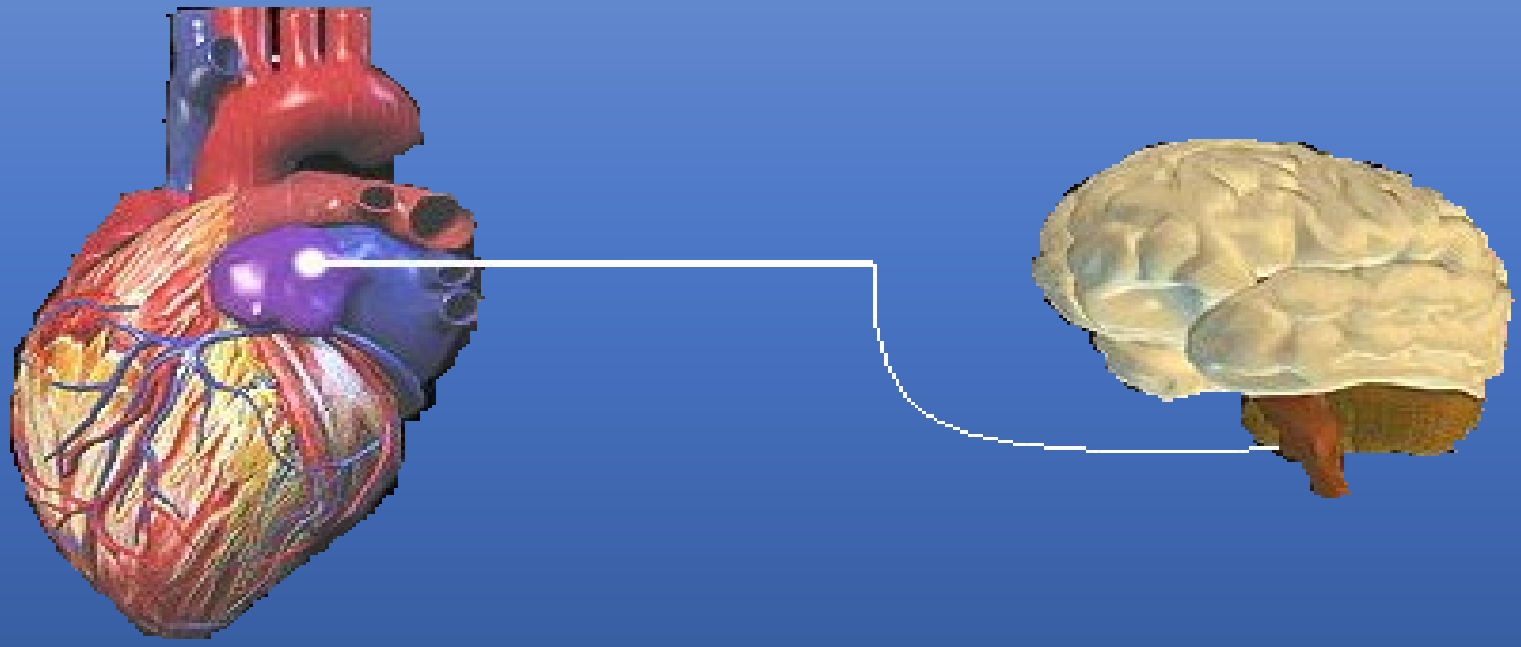
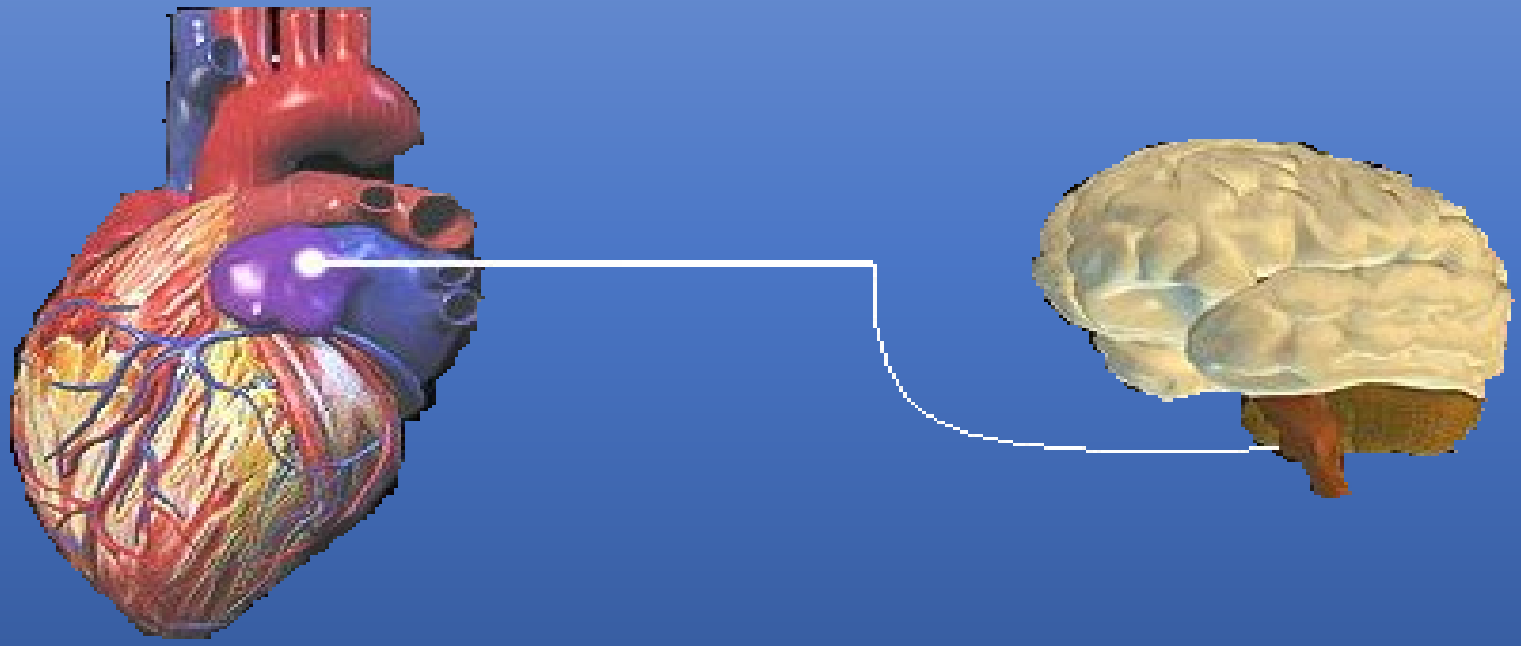
Department of Medical Translation Sciences,
University of Naples, Federico II Italy
Director Saint Joseph Nursing Home

INTERAZIONE CUORE-CERVELLO



CUORE-CERVELLO: RAPPORTO DIALETTICO



1. L'antichità: il cuore come centro delle emozioni e del pensiero

a. Antico Egitto (Gli Egizi credevano che il cuore fosse il centro delle emozioni, della moralità e della memoria.
b. Mesopotamia: I Babilonesi e gli Assiri associavano il cuore alla volontà e all'intelletto.
c. Grecia antica (prima di Aristotele): Omero, nell'Iliade e nell'Odissea, descriveva le emozioni come originate dal cuore o dal petto (es. "parlare dal cuore").

2. La svolta greca: Scuola cardiocentrica (Aristotele) vs Scuola cerebrocentrica (Ippocrate e Galeno)

Il cuore era l'organo più importante perché quando il cuore si ferma l'uomo muore.

Aristotele
Stagira, 384 o 383 a.C.
Calcide, 322 a.C.

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2. La svolta greca: Scuola cardiocentrica (Aristotele) vs Scuola cerebrocentrica (Ippocrate e Galeno)

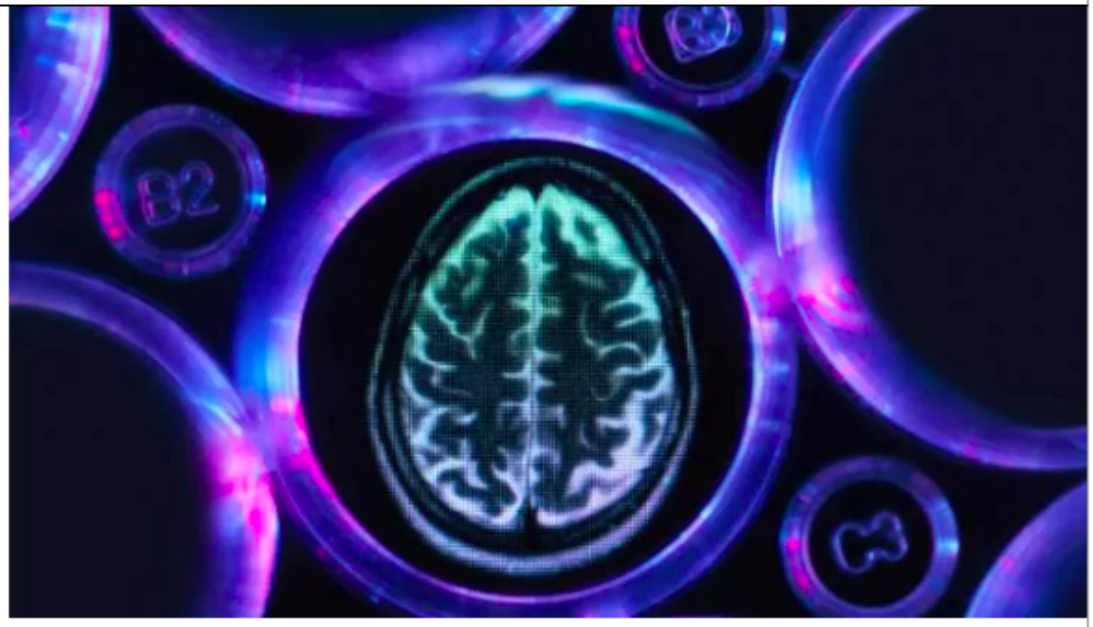
3. Medioevo e Rinascimento: una lenta transizione: Nel mondo islamico, studiosi come Avicenna (980-1037) sostenevano il ruolo del cervello nelle funzioni cognitive. In Europa, però, la visione aristotelica rimase dominante fino al Rinascimento. Leonardo da Vinci e Andrea Vesalio (XVI sec.) contribuirono a una migliore comprensione dell'anatomia cerebrale.

4. Età moderna: il trionfo del cervello. Con l'avvento della scienza moderna (XVII-XIX sec.), il cervello fu definitivamente riconosciuto come sede della mente

“Nel cervello c’è una zona speciale, che potremo chiamare memoria poetica che registra tutto quello che ci affascina o ci commuove, cioè che rende bella la nostra vita”.

Milan Kundera
Brno, Repubblica Ceca, 1 Aprile 1929
Parigi, Francia, 11 Luglio 2023

Sportello Cuore



Infarto e scompenso, perché ci vuole attenzione se il cervello inizia a perdere colpi

di Federico Mereta

Un filo rosso unisce cuore e cervello. L'intelligenza artificiale aiuterà a capire cosa accade e a cogliere i primi deficit cognitivi

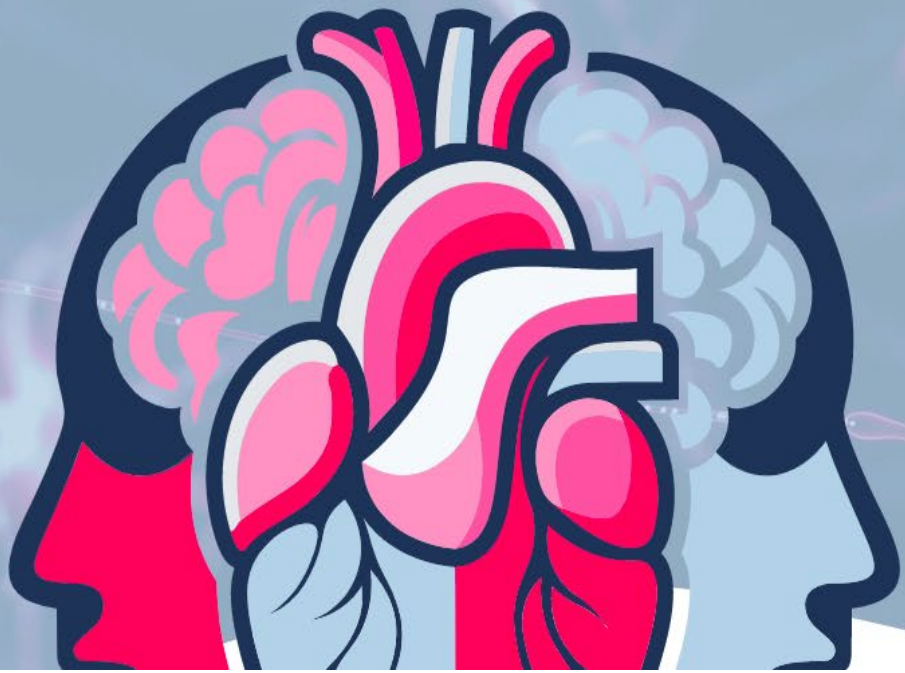
27 FEBBRAIO 2025 ALLE 12:06

2 MINUTI DI LETTURA

DORIAN GRAY

Bridging Cognitive Decline and Cardiovascular Health for Healthy Aging

An AI-driven approach to prevent Mild Cognitive Impairment (MCI) in cardiovascular patients

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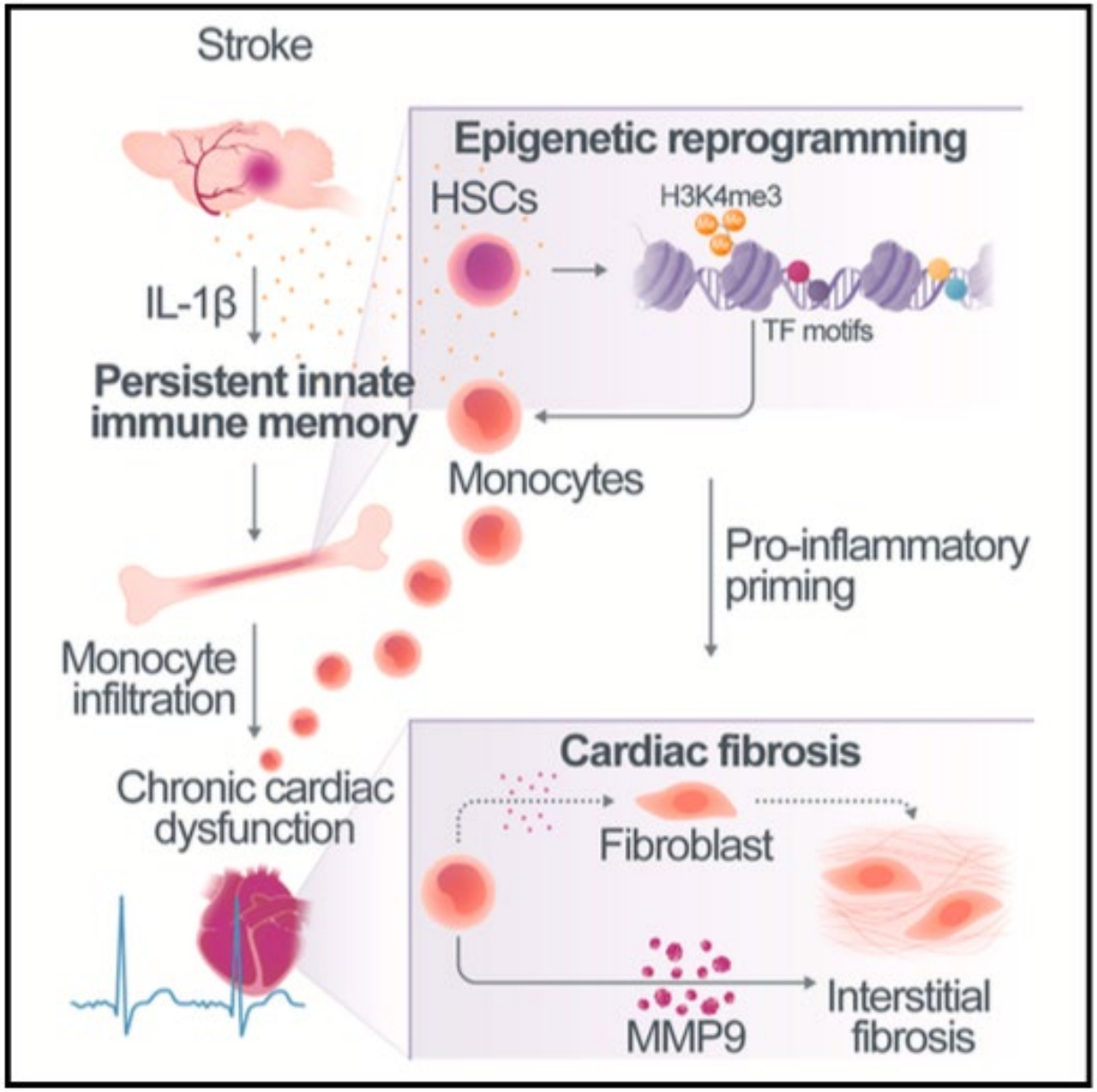
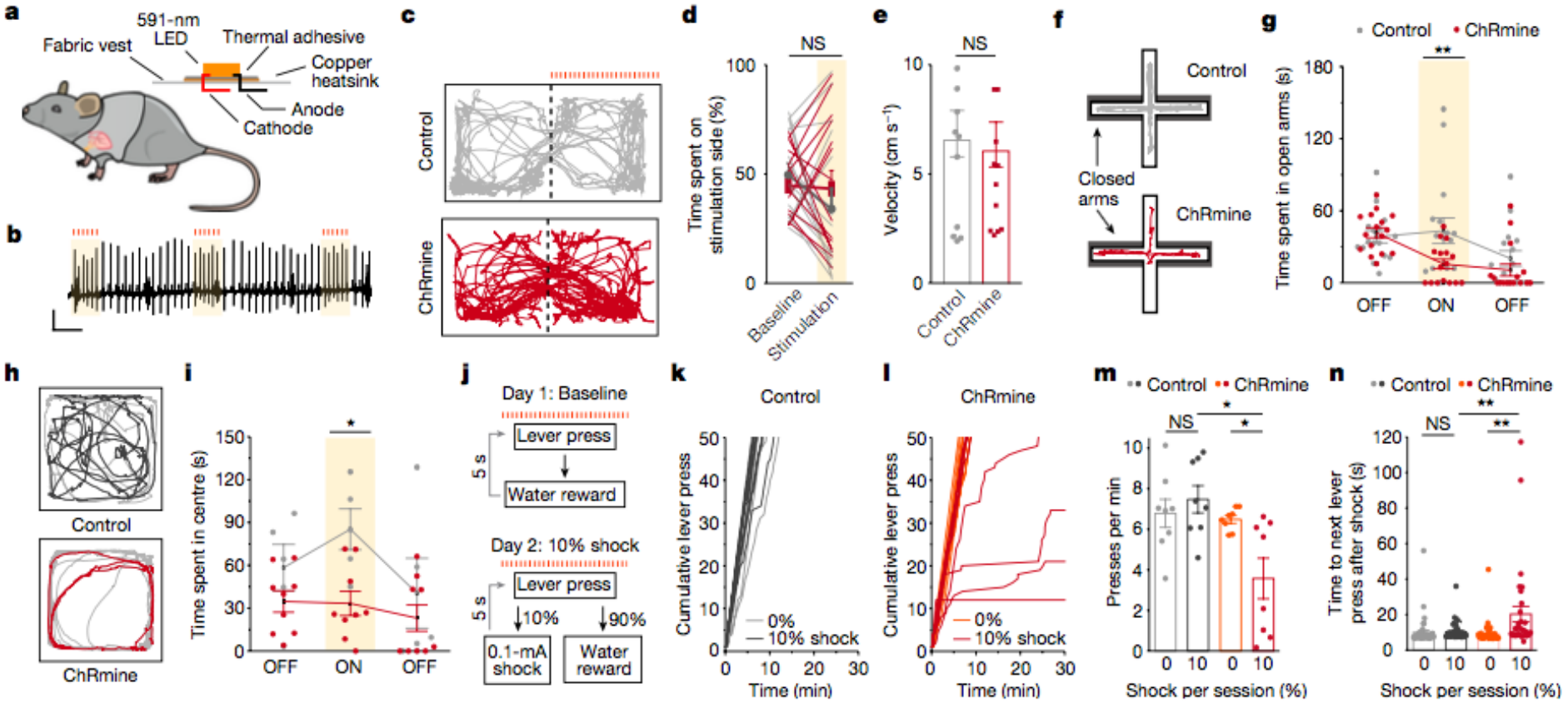
NUROGAMES
RHL
UHK
FNUSA-ICRC
UNIBS (coordinator)
IUBCVB
ROPARDO
FORTH
INFOTREND
MSHS

Innate immune memory after brain injury drives inflammatory cardiac dysfunction

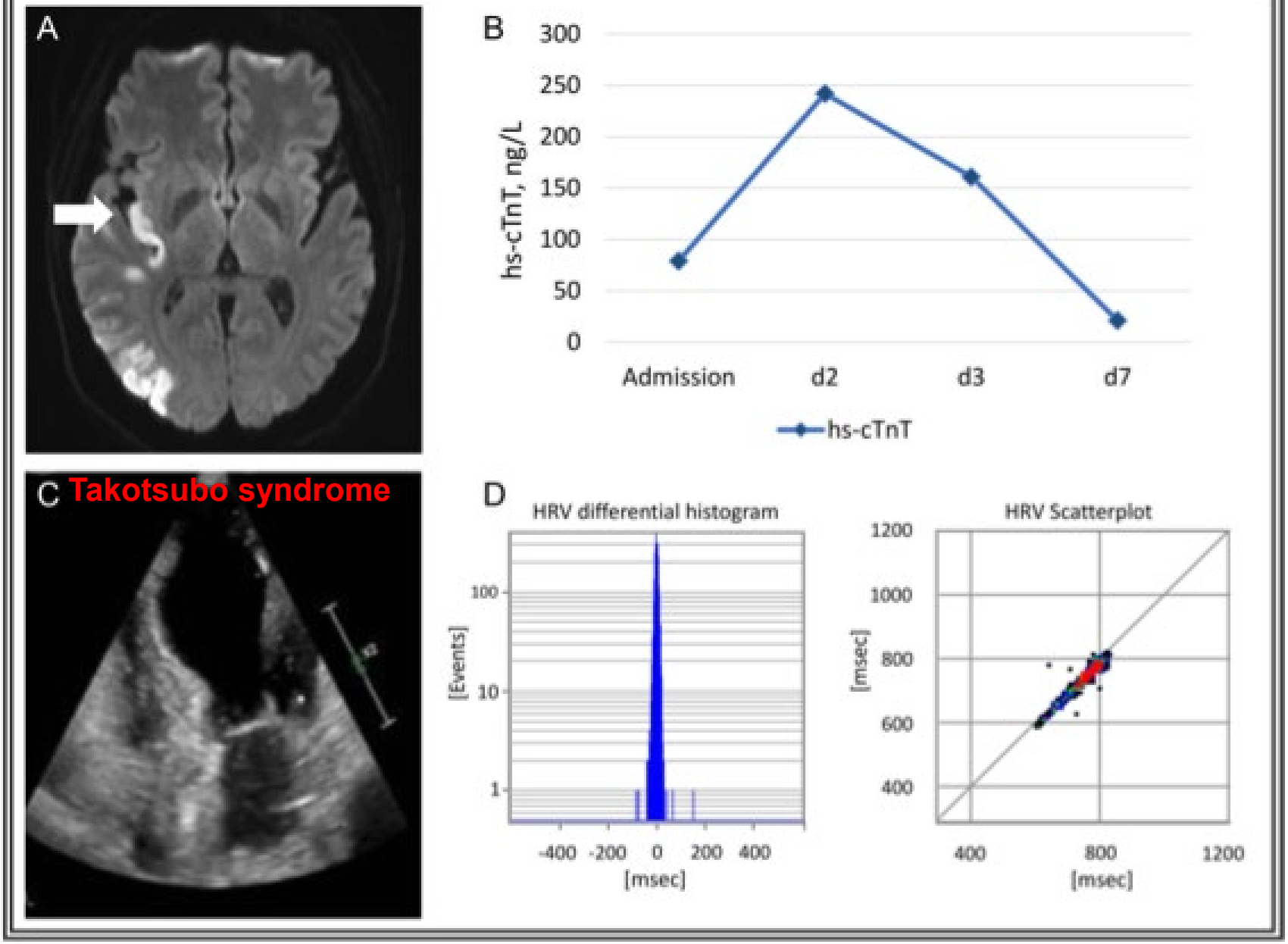


Simats et al., 2024, Cell *187*, 4637–4655
August 22, 2024
https://doi.org/10.1016/j.cell.2024.06.028



Nature
. 2023 Mar;615(7951):292-299.
doi: 10.1038/s41586-023-05748-8. Epub 2023 Mar 1.

Cardiogenic control of affective behavioural state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The Stroke-Heart Syndrome – Recent Advances and challenges

Stroke-Heart Syndrome

Definition:
New evidence of cardiac alterations or documented worsening of premorbid cardiac function after ischemic stroke

Manifestations:
ECG changes, arrhythmia
Myocardial injury, ACS
Heart Failure, Takotsubo
Sudden cardiac death

Frequency:
10-20% cardiac SAE within 30 days

Time-Course:
Peak within 72 hours up to 30 days.

Prognosis:
2-3 fold increased risk of short-term mortality
1.5-2 fold increased risk of MACE during long-term

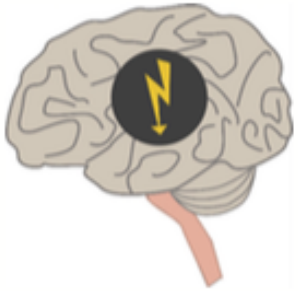
Risk factors:
Age, pre-morbid heart disease, stroke-specific factors (severity, insular cortex)

Pathophysiology:
Autonomic dysbalance and inflammation
→ Stroke-induced ‚cardiac stress test'

Perspectives:
Join forces to identify treatments (basic/ clinical scientists; neurologists/ cardiologists/ immunologists)

STROKE-HEART Syndrome

Stroke



Stroke characteristics

- Lesion size, stroke severity
- Central autonomic network changes

Mediators

Immunological

- Brain-bone marrow-spleen signals
- Systemic Inflammation
- Inflammasome

Neuronal

- Autonomic nervous tone

Humoral

- Catecholamines
- Cortisol
- Vasoactive mediators (e.g. endothelin-1)
- Brain derived death factors
- miRNAs
- Extracellular vesicles

Heart

Downstream Mechanisms

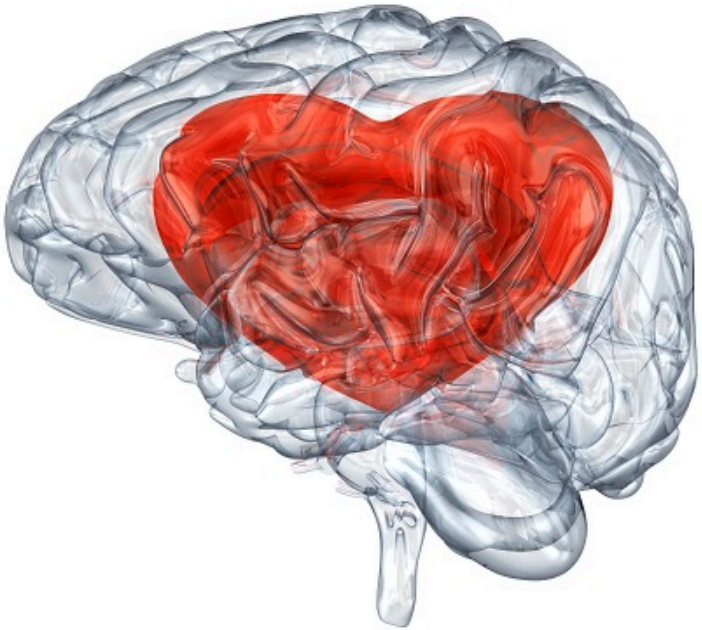
- Impaired coronary (micro-) circulation
- Macrophage dysfunction
- Cardiomyocyte injury

Outcome

High risk of

- Cardiovascular events
- Heart failure
- Cardiac death
- Cognitive dysfunction

(Epi-)Genetic modification



Heart Failure and cognitive impairment

Hypertension and cognitive impairment

Heart Failure and cognitive impairment

Hypertension and cognitive impairment

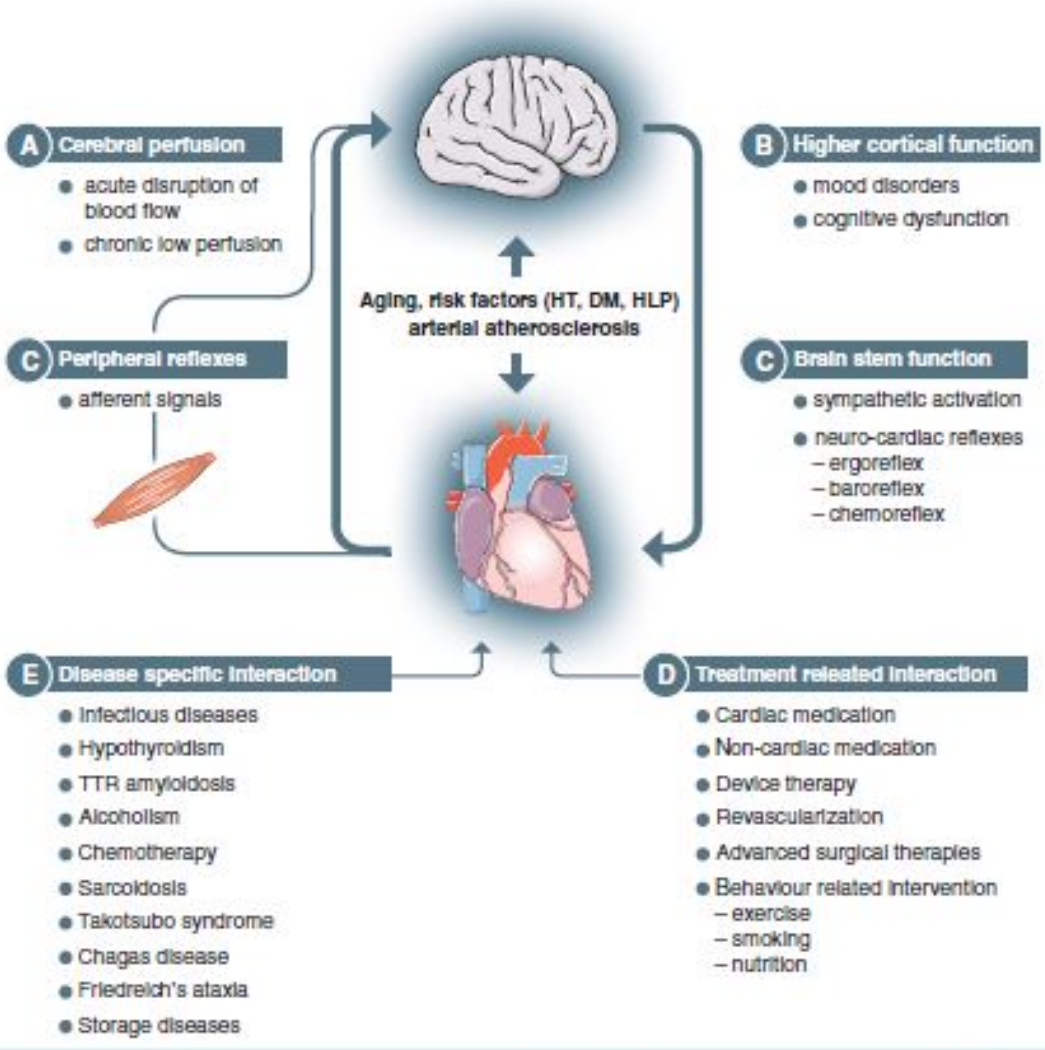
European Journal of Heart Failure (2018) 20, 199–215
doi:10.1002/ejhf.1100

HFA POSITION PAPER

Heart and brain interaction in patients with heart failure: overview and proposal for a taxonomy. A position paper from the Study Group on Heart and Brain Interaction of the Heart Failure Association

Doehner W. et al.

Overview delle interazioni cuore-cervello



Systematic overview of heart and brain interaction in heart failure. DM, diabetes mellitus; HLP, hyperlipoproteinaemia; HT, hypertension; TTR, transthyretin.

European Journal of Heart Failure (2018), W. Doehner et al.

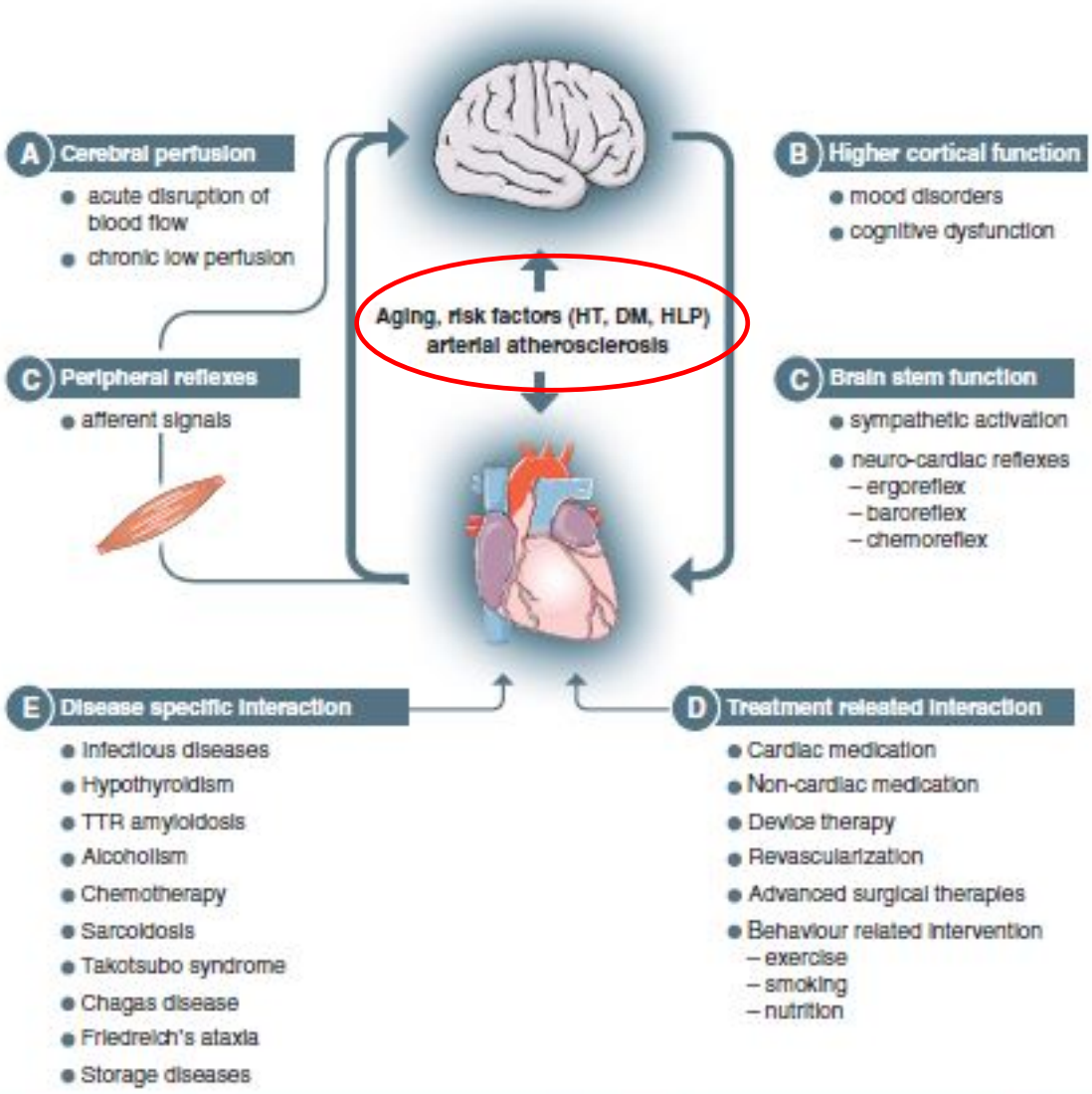


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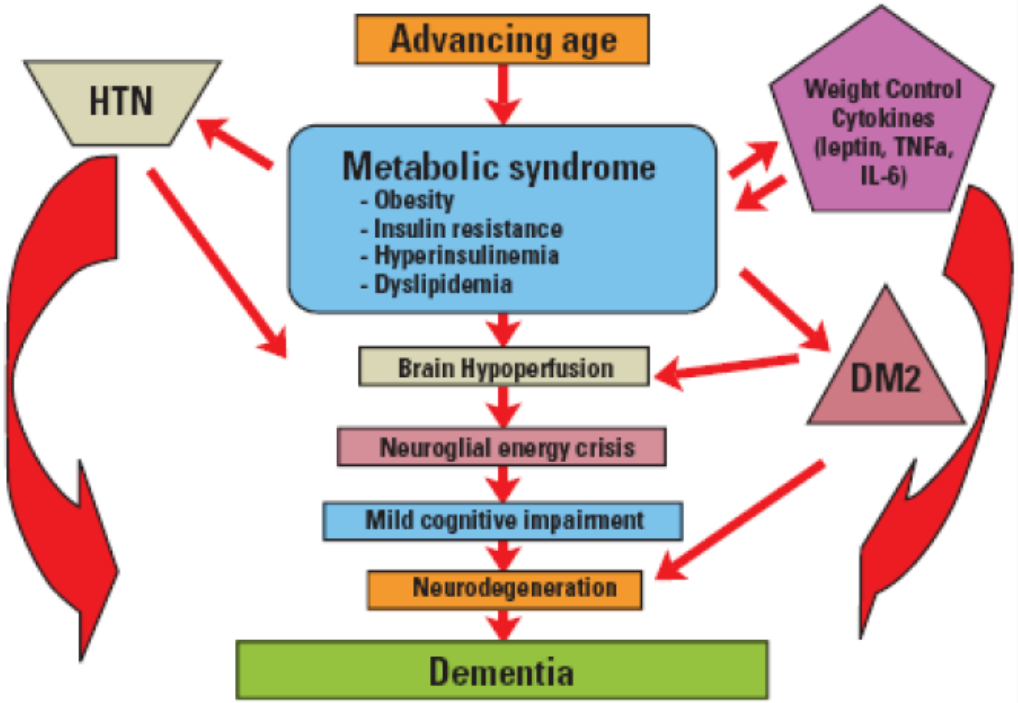
FIGURE.

Potential relationships between components of the metabolic syndrome and the development of Alzheimer's disease: *A link to a vascular etiology?*[61]



HTN=hypertension; TNFa; tumor necrosis factor-alpha; IL=interleukin; DM2=type 2 diabetes mellitus.

de la Torre JC. Is Alzheimer's disease a neurodegenerative or a vascular disorder? Data, dogma, and dialectics. *Lancet Neurol*. 2004;3:184-190. Adapted with permission from Elsevier Limited. Copyright 2004.

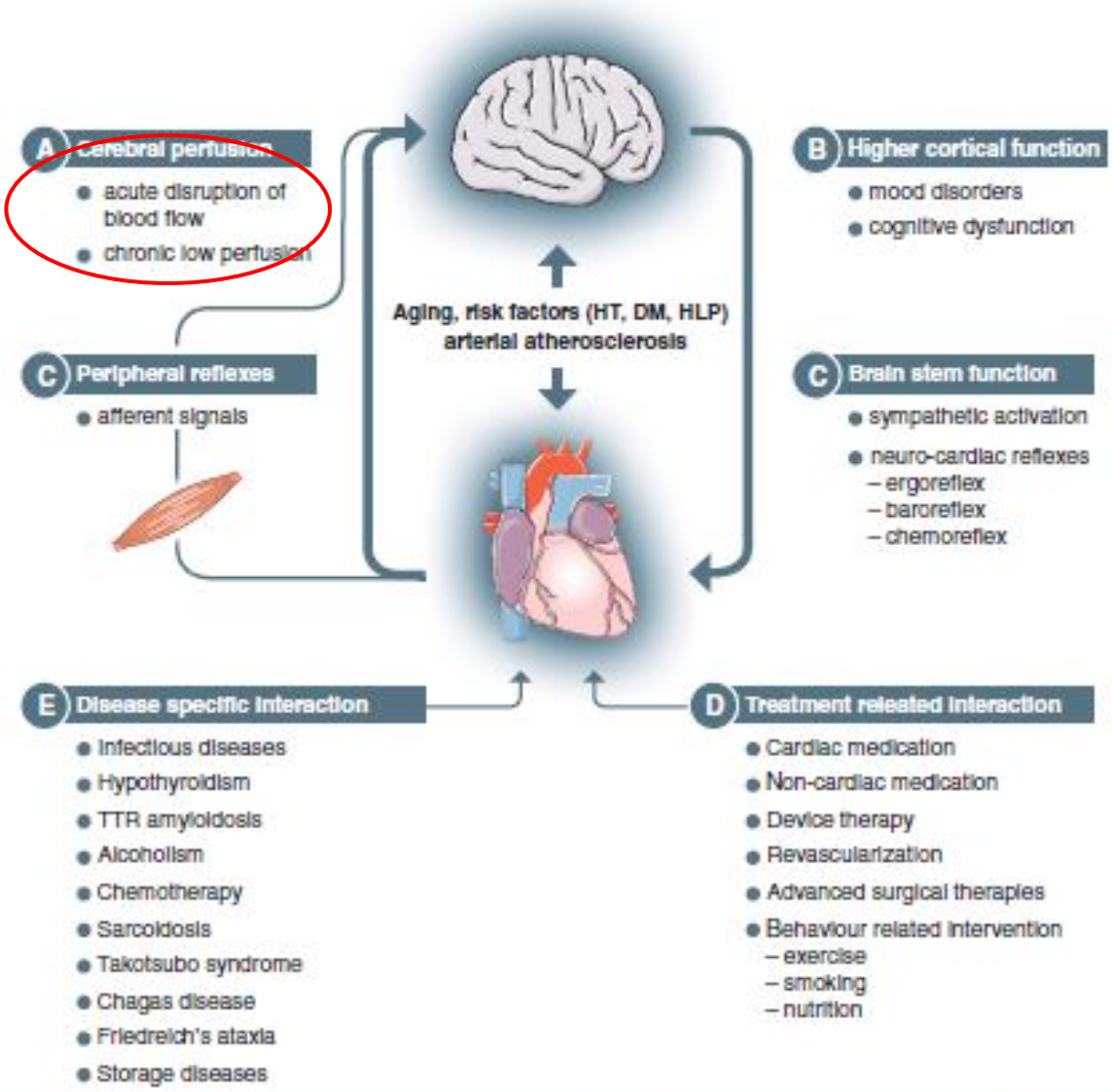
Milionis HJ, Florentin M, Giannopoulos S. *CNS Spectr*. Vol 13, No 7. 2008.

Similarities between heart failure and neurological disorders

	Heart failure	Neurological disorders
Aetiologies		
Aging	Increasing prevalence	Increasing prevalence
Vascular end-organ damage	Acute myocardial infarction	Acute stroke
Atrophy and fibrosis	HFpEF, HFrEF	Cerebral atrophy
Prevention		
Management of risk factors	Effective, especially hypertension, dyslipidaemia and diabetes	Effective, especially hypertension
Aspirin, statins	Effective for secondary prevention in CAD	Effective for prevention of ischaemic stroke
Antithrombotics for AF and patients with prosthetic heart valves	OAC effective for prevention of systemic thromboembolic events	OAC effective for secondary prevention of stroke
Management		
Revascularization	PCI effective to improve outcome	CEA/CAS effective to prevent TIA, stroke
Thrombolysis	Effective in AMI to decrease HF development	Effective in ischaemic stroke to decrease sequelae

AF, atrial fibrillation; AMI, acute myocardial infarction; CAD, coronary artery disease; CAS, carotid artery stenting; CEA, carotid endarterectomy; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; OAC, oral anticoagulant; PCI, percutaneous coronary intervention; TIA, transient ischaemic attack.

European Journal of Heart Failure (2017), W. Doehner et al.



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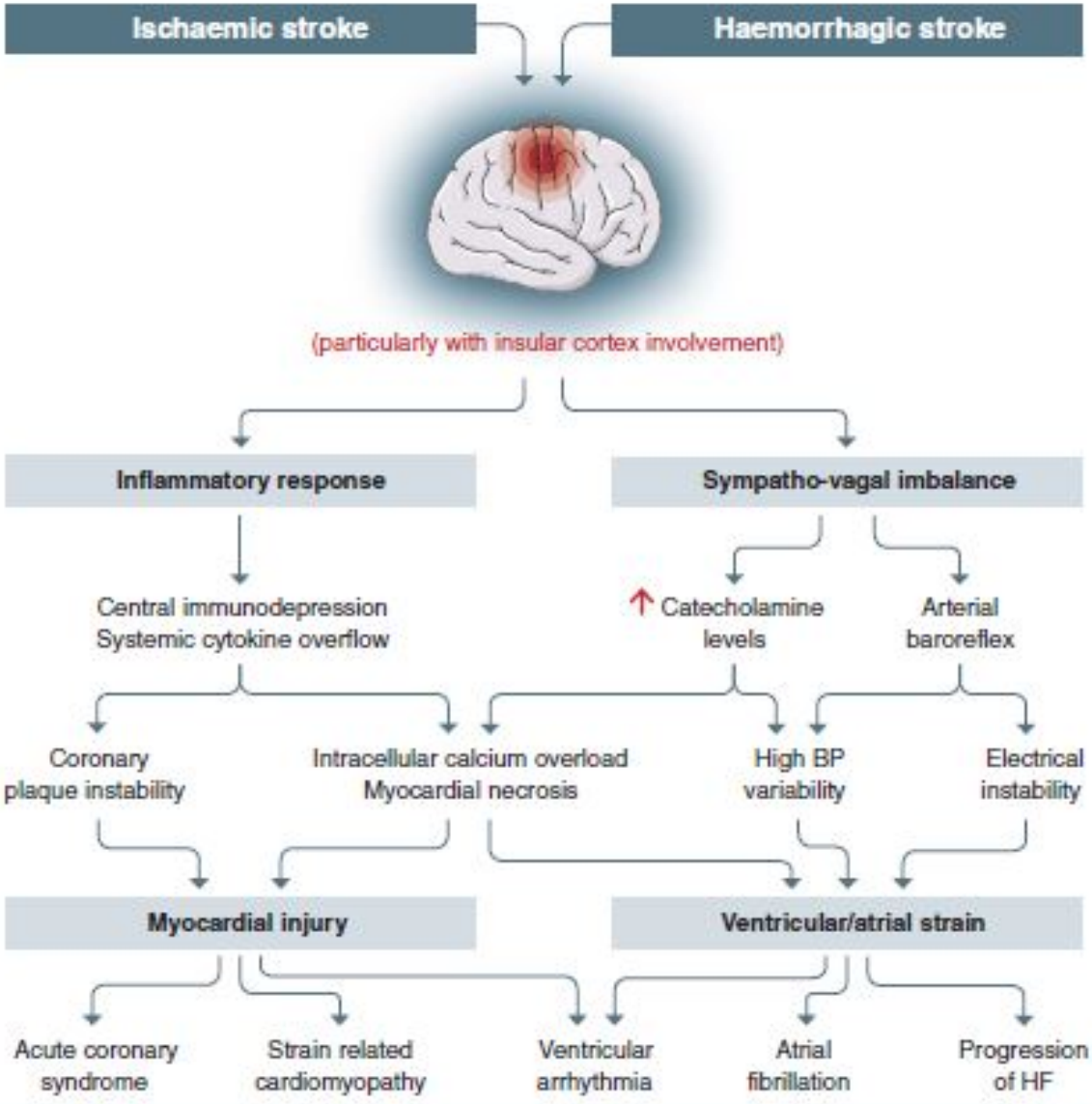
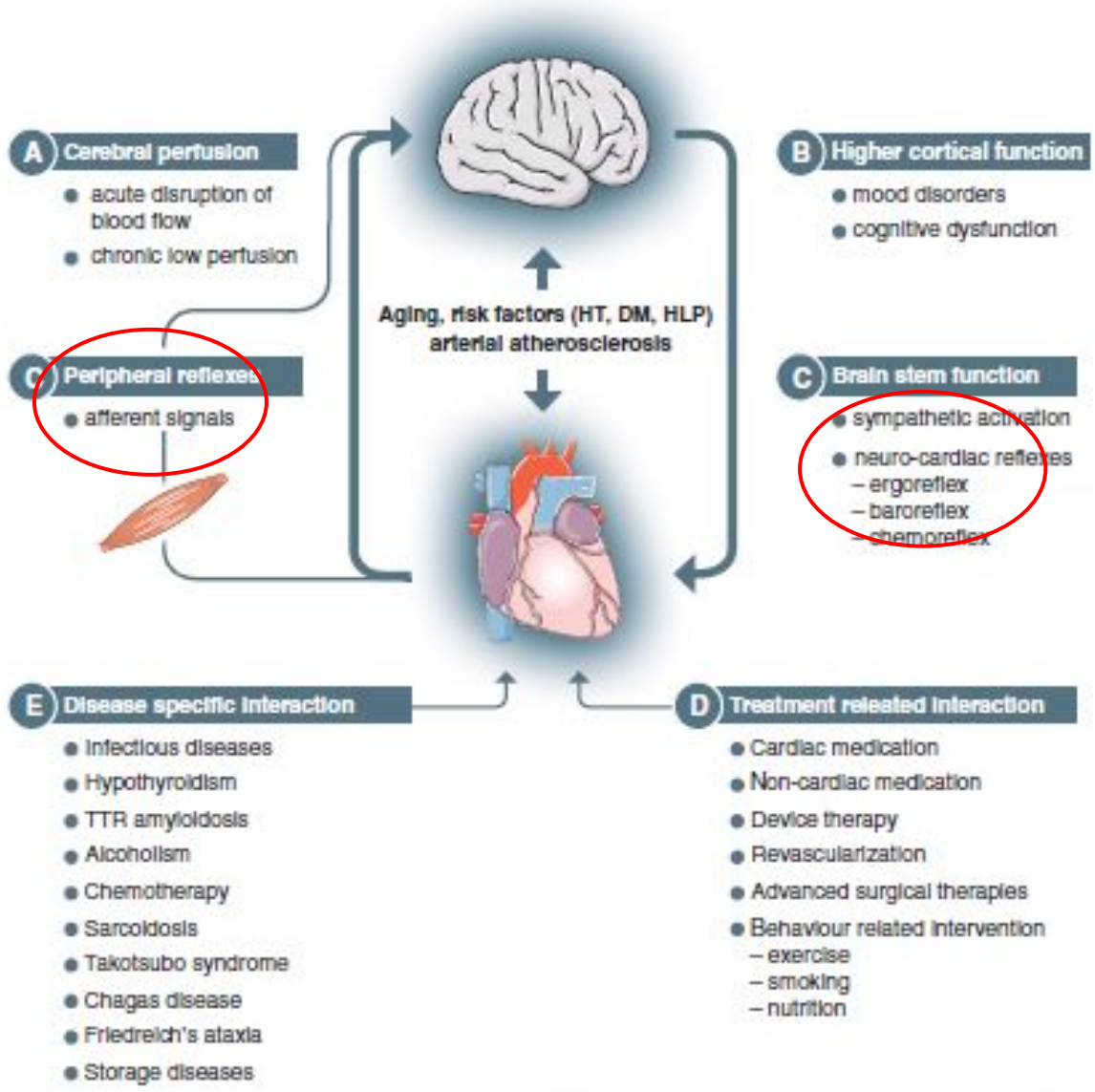


Cerebro-cardiac signalling after stroke. BP, blood pressure; HF, heart failure.

European Journal of Heart Failure (2017), W. Doehner et al.



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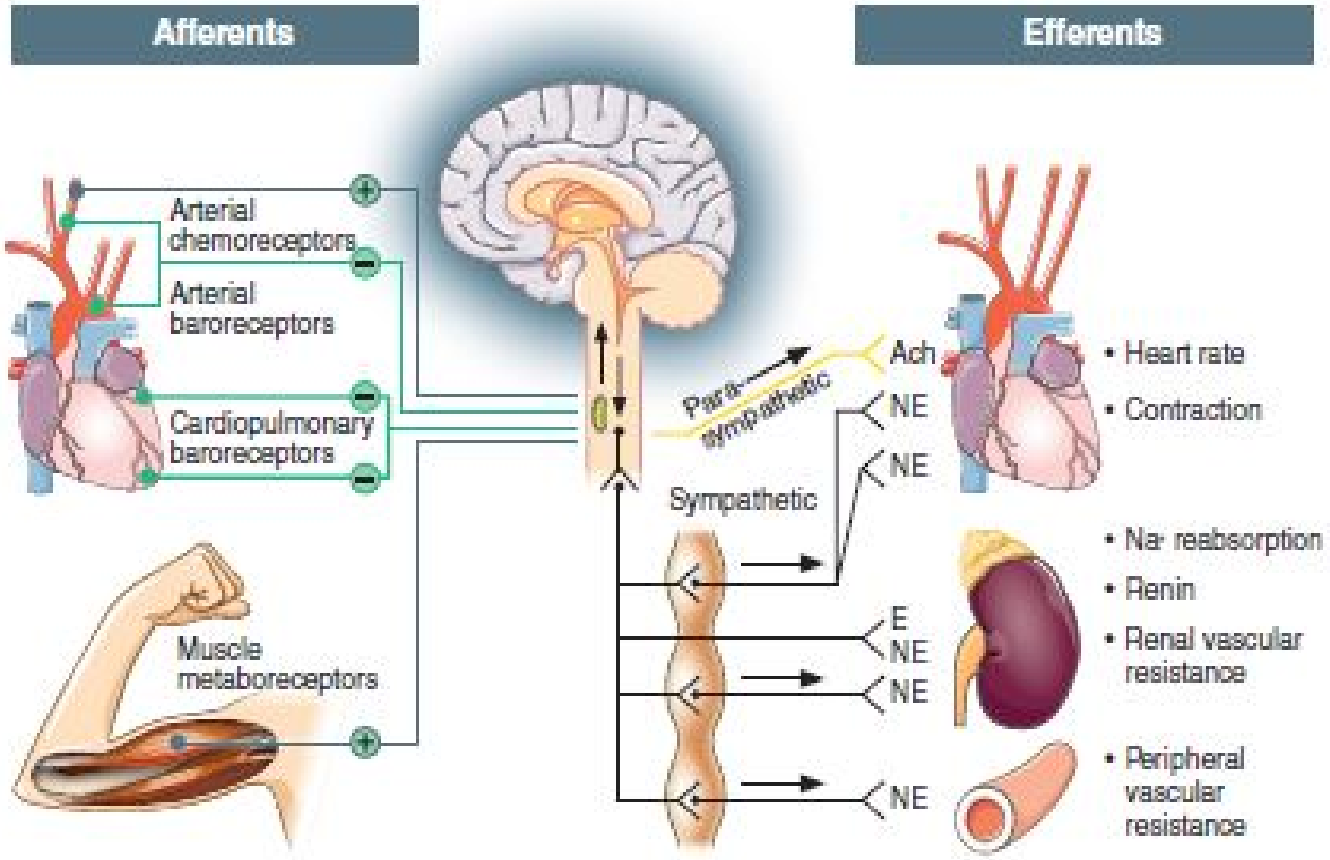
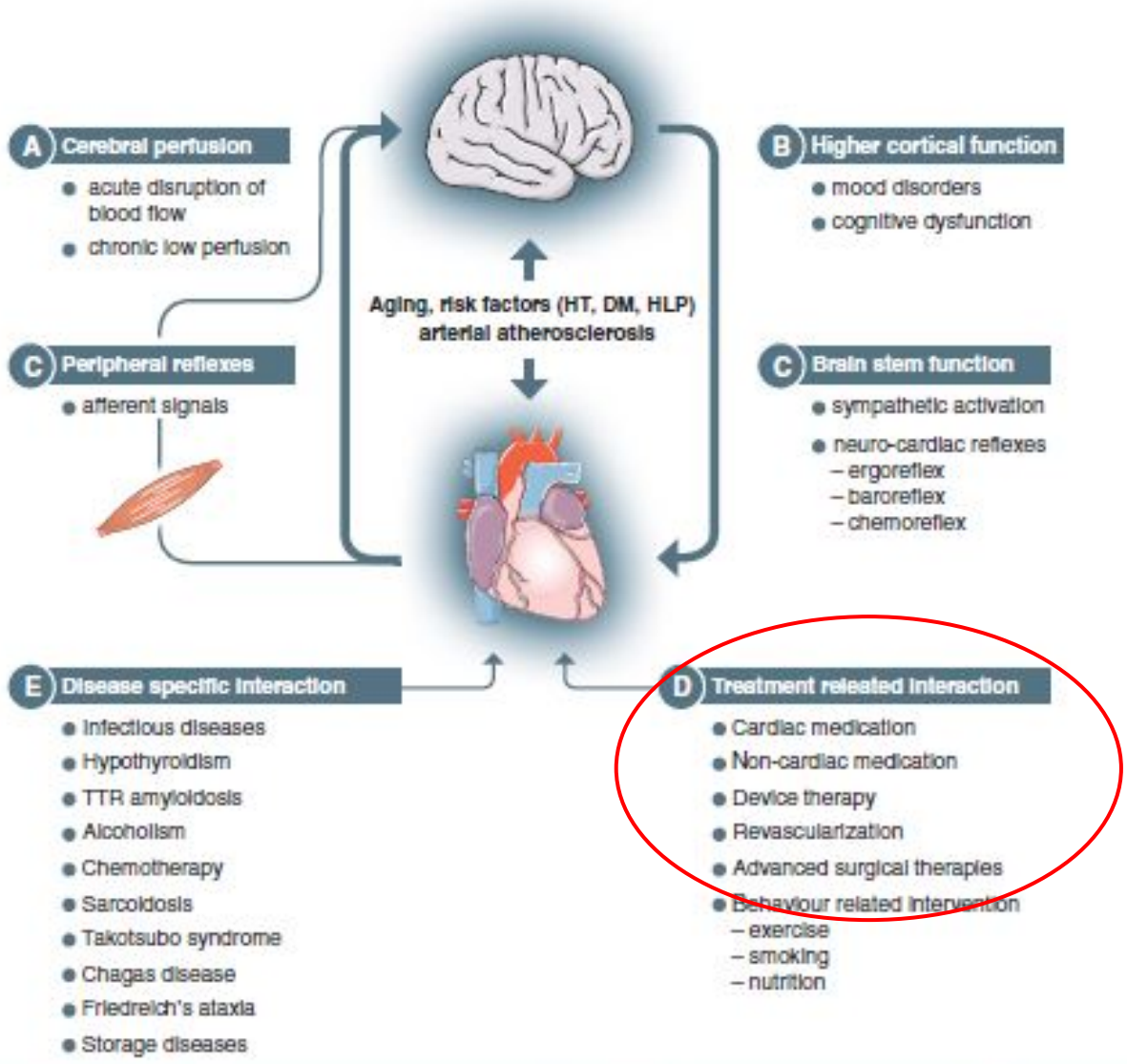


Neuro-cardiac reflexes. Cardiovascular signals from chemo-, baro- and ergo-receptors trigger neuro-vegetative stimulation that feeds back on cardiovascular regulation. Ach, acetylcholine; E, epinephrine; NE, norepinephrine. Modified from Floras.[80]

European Journal of Heart Failure (2018), W. Doehner et al.



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European Journal of Heart Failure (2018), W. Doehner et al.

Interactions and possible cardiovascular side effects of antidepressants and therapies for cognitive impairment

Treatment	Interaction with heart failure
Antidepressants	
SSRIs	Safe in patients with HF. They rarely lead to bradycardia (when used with beta-blockers) or bleeding (when used with antiplatelets or anticoagulants), and may increase serum levels of common cardiac agents due to CYP450 enzyme inhibition. Citalopram causes dose-dependent QT interval prolongation.
SNRIs	Slight increase in blood pressure and heart rate, rarely arrhythmias may be observed.
Mirtazapine	May cause weight gain and hyperlipidaemia, arterial blood pressure elevation, oedema, rarely myocardial infarction, sudden cardiac death and heart failure.
Bupropion	Safe in patients with HF. It rarely causes orthostatic hypotension, hypertension and arrhythmias.
MAOIs	Are usually avoided due to due to cardiovascular side effects, including hypertensive crisis and orthostatic hypotension.
TCAs	Are usually avoided due to their pro-arrhythmic potential and cardiac side effects like hypotension and worsening heart failure.
Pharmacological agents for treatment of cognitive impairment	
Acetylcholinesterase inhibitors	Cardiovascular adverse events occur rarely. The most frequently reported adverse effect is dysrhythmia in the form of bradycardia and conduction disorders. Syncope, oedema and hypertension maybe observed. Co-administration with beta-blockers, digoxin, amiodarone, and calcium channel blockers may increase the risk for syncope or heart block.
Memantine	Favourable side effect profile. Dizziness, hypertension, angina, bradycardia and heart failure may be observed.

MAOI, monoamine oxidase inhibitor; SNRI, serotonin–norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor; TCA, tricyclic antidepressant.

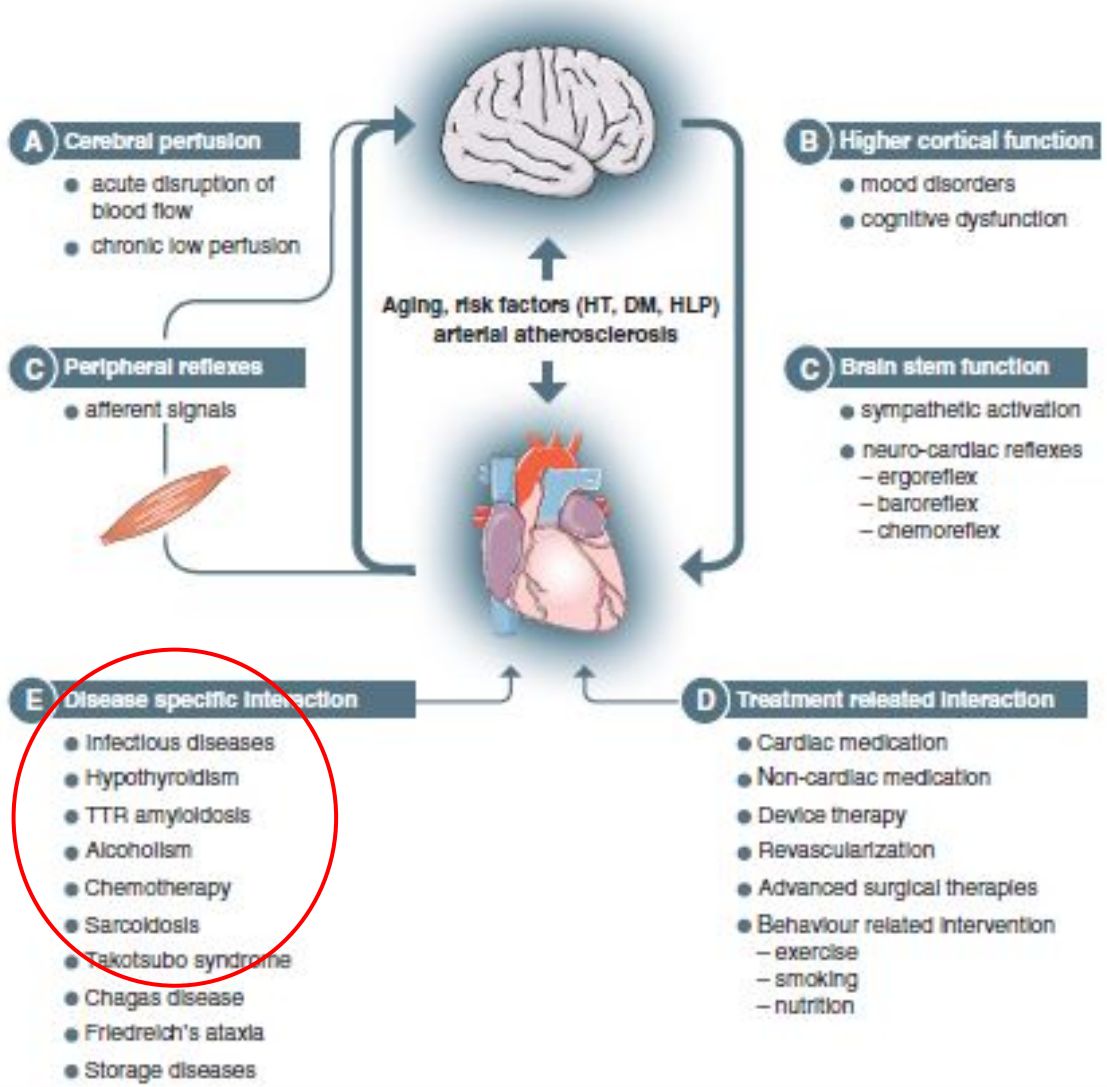
European Journal of Heart Failure (2018), W. Doehner et al.

Interactions and possible neurological side effects related to heart failure therapies

Treatment	Interaction with neuropsychiatric status
Medical therapy for HF	
ACE inhibitors	Excessive hypotension may lead to dizziness and syncope. No association with depression. Possible beneficial effects on cognitive impairment.
Beta-blockers	Hypotension may lead to dizziness. Conflicting evidence for increased depression (care may be taken in patients with positive personal or family history and using lipophilic beta-blockers). May cause loss of sex drive.
MRAs	Conflicting evidence for increased depression (increase in cortisol levels may lead to depression).
Sacubitril/valsartan	Concerns about a possible effect on Alzheimer's disease.
Device therapy	
Pacemaker	Stroke is infrequent, unless associated with PFO.
CRT, ICD	Anxiety, depression and PTSD, particularly in those with inappropriate shocks.
Ablation	Stroke may develop during AF/AFl ablation (6%).
Revascularization, mechanical circulatory support and HTx	
PCI	Stroke is infrequent (1.3%).
CABG	Perioperative stroke (1.6%) is associated with increased mortality.
LVAD	Cerebral infarction, haemorrhagic stroke, cognitive impairment.
VA-ECMO	Cerebral infarction, haemorrhagic stroke, cognitive impairment (especially in patients with ASD or PFO).
HTx	Peripoperative hypoperfusion, improved cerebral blood flow with successful HTx.

ACE, angiotensin-converting enzyme; AF, atrial fibrillation; AFl, atrial flattar; ASD, atrial septal defect; CABG, coronary artery bypass grafting; CRT, cardiac resynchronization therapy; HF, heart failure; HTx, heart transplantation; ICD, implantable cardioverter-defibrillator; LVAD, left ventricular assist device; MRA, mineralocorticoid receptor antagonist; PCI, percutaneous coronary intervention; PFO, patent foramen ovale; PTSD, posttraumatic stress disorder; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

European Journal of Heart Failure (2018), W. Doehner et al.



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European Journal of Heart Failure (2018), W. Doehner et al.

Specific diseases linking development or progression of heart failure with cerebral interaction

Disease	Main cardiac implication	Cerebral interaction
Atherosclerosis, carotid artery disease	Ischaemic HF	Regional hypoperfusion
Infectious diseases	Cardiomyopathy, arrhythmia, pericardial effusion, valvular damage	Sensomotor neuropathy, hypoperfusion, embolism
Alcoholism	Non-ischaemic HF	Atrophy, Wernicke encephalopathy, Korsakow syndrome, epilepsy, dementia
Hypothyroidism	Arrhythmias, heart failure	Agitation, tremor
TTR amyloidosis	Myopathy, arrhythmias, HF	Autonomic neuropathy
Chemotherapy	Cardiotoxic myopathy, HF	Neurotoxic effects, sensomotor injury
Sarcoidosis	Arrhythmias, AV block, HF, pericardial effusion	Diabetes insipidus, hypopituitarism, *nervus facialis* neuropathy
Takotsubo syndrome	Takotsubo cardiomyopathy	Sympathomimetic overflow
Chagas disease	Chagas heart disease	Stroke, autonomic neuropathy
Storage diseases	Cardiomyopathy, HF	Sensomotor neuropathies
Friedreich ataxia	AF, hypertrophic cardiomyopathy	Sensomotor neuropathy, cerebellar impairment
Muscle diseases with cardiac phenotype	Cardiomyopathy, HF	Sensomotor neuropathies

AF, atrial fibrillation; AV, atrioventricular; HF, heart failure.

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European Journal of Heart Failure (2018), W. Doehner et al.

CLINICAL INVESTIGATION

Congestive Heart Failure and Cognitive Impairment in an Older Population

Francesco Cacciatore, MD,[*] Pasquale Abete, MD, PhD,[*] Nicola Ferrara, MD,[†]*
Claudio Calabrese, MD,[*] Claudio Napoli, MD,[*] Stefania Maggi, MD, MPH,[‡]
Michele Varricchio, MD,[§] and Franco Rengo, MD,[†]*
for the Osservatorio Geriatrico Campano Study Group

OBJECTIVE: Congestive heart failure (CHF) is potentially preventable, and the identification of modifiable risk factors for cognitive impairment (CI) for older persons is a very important issue. We examined the cross-sectional relationship between CHF and CI in an older population.

DESIGN: A cross-sectional survey.

SETTING AND PARTICIPANTS: A total of 1339 subjects aged 65 and older were selected from the electoral rolls of Campania, a region of southern Italy.

MEASUREMENTS: Sociodemographic characteristics were recorded, as was the presence of cardiovascular diseases, including CHF classified according to the New York Heart Association (NYHA) guidelines for disease severity; CI evaluated by means of the Mini-Mental State Examination (MMSE), with a score of <24 indicating impairment; geriatric depression scale (GDS) rating; blood pressure (BP); and heart rate (HR).

RESULTS: The final sample numbered 1075; 172 subjects were excluded because of neurological disorders and psychotropic therapy, and 92 were excluded because their BP, HR, or cognitive examination was not recorded. Prevalence of CHF was 8.2%, and 23.0% of subjects scored <24 on the MMSE. The prevalence of CHF in subjects with an MMSE score of <24 and ≥24 was 20.2% and 4.6%, respectively ($P < .001$). Logistic regression analysis showed that CHF was associated independently with CI by sex, age, educational level, GDS, diabetes, hypertension, alcohol consumption, smoking, atrial fibrillation, systolic and diastolic BP, and HR. The risk of CI was 1.96-fold greater in subjects with CHF (odds ratio: 1.96; 95% confidence interval: 1.07–3.58; $P < .028$). Systolic BP decrease was correlated negatively with NYHA classes only in subjects with CI ($r = -0.981$; $P < .020$), whereas HR increase was correlated positively with NYHA classes only in subjects without CI ($r = 0.985$; $P < .015$).

CONCLUSIONS: In our population, CHF is associated with CI in subjects aged 65 years and older. Systolic BP reduction and the lack of HR increase, related to NYHA classes, might characterize cognitively impaired subjects with CHF. **J Am Geriatr Soc 46:1343–1348, 1998.**

A large part of the population aged 65 years and older is cognitively impaired.[1] A call for action against the vascular dementias[2] will reinforce the concept that cardiovascular diseases and vascular atherosclerosis may contribute to cognitive decline.[3,4] Congestive heart failure (CHF), the terminal manifestation of various heart diseases, is a condition common to older people.[5] CHF is not only one of the most important causes of death and disability in older people in Western countries, it is also a major cause of morbidity and contributes greatly to health care costs.[6] The prevalence of CHF is approximately 6% in subjects between 65 and 74 years of age and 10% in those aged 75 years and older.[6]

The effect of CHF on cognitive impairment is uncertain. Depression of cardiac output may alter the oxygen and nutrient supply to the brain, thereby increasing the risk of cognitive deterioration.[7] Decreased cerebral perfusion leads to progressive loss of neurocognitive processing and causes neurobehavioral disorders.[8] Moreover, white matter lesions have been associated with cognitive impairment in subjects with CHF,[9,10] and the TOAST study found that subjects affected by CHF have a 1.88-fold greater risk of silent cerebral infarction.[11] Finally, an improvement in cognitive brain function was observed in patients with symptomatic dilated

Characteristics of population without cerebro- vascular diseases stratified by age

Age range (years)	**65-74** (n=665)	**75-84** (n=334)	**> 85** (n=76)
Females (%)	356 (53.5)	183 (54.8)	56 (73.7)‡§
Educational level ^	3.6±1.4	3.1±1.5*	3.0±1.6‡
MMSE < 24 (%)	**21.0**	**24.5**	**34.4 ‡§**
GDS score	9.7±6.6	12.0±5.9*	13.1±6.4‡
HF (%)	**6.2**	**10.8***	**13.2‡**
SBP (mmHg)	**144.1±18.5**	**147.0±19.4°**	**147.9±19.9**
DBP (mmHg)	**82.4±9.1**	**81.3±9.2**	**82.0±8.7**
HR (bpm)	74.4±9.7	75.3±10.4	77.3±11.8

<0.01, 75-84 vs 65-74; ‡p<0.01, ≥85 vs 65-74; §p<0.01, ≥85 vs 75-84; ° p<0.05, 75-84 vs 65 74;
¶ p < 0.05, ≥85 vs 65-74

Cacciatore F et al., JAGS 1998

Distribution of variables according to the presence or absence of HF

	CHF		
Variables	Present	Absent	p value
Age (years)	75.4±7.2	73.7±6.2	0.01
MMSE < 24 (%)	**56.8**	**20.0**	**0.000**
Educational level^	2.8±1.5	3.5±1.4	0.001
GDS score	15.3±5.8	10.3±6.1	0.000
SBP(mmHg)	148.2±20.5	145.1±18.9	0.142
DBP (mmHg)	82.8±10.5	82.0±9.0	0.404
HR (bpm)	77.1±13.0	74.7±9.7	0.03

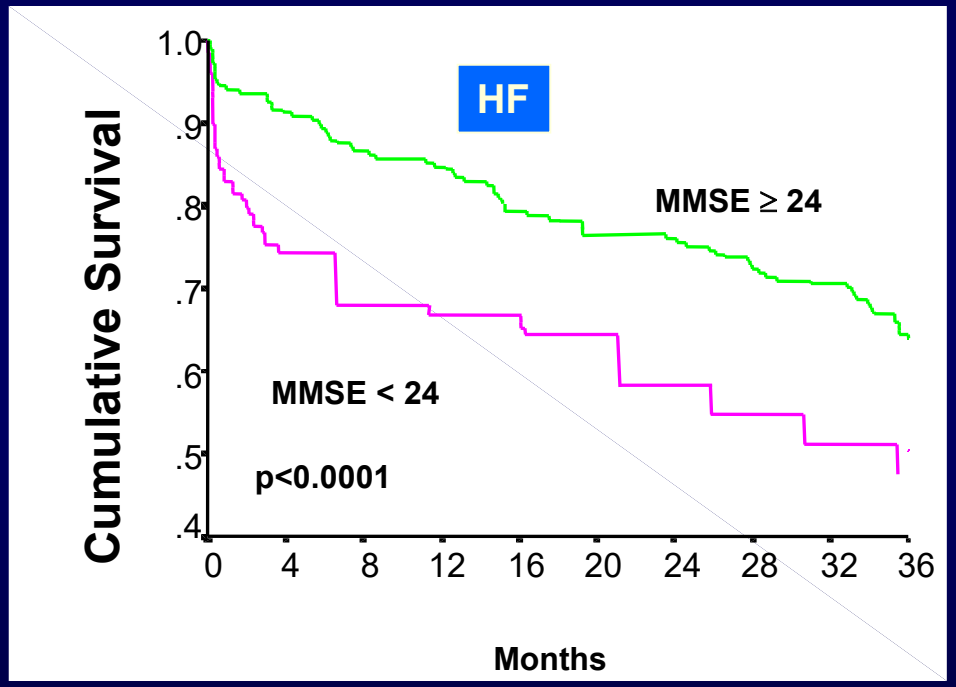
Cacciatore F et al., JAGS 1998

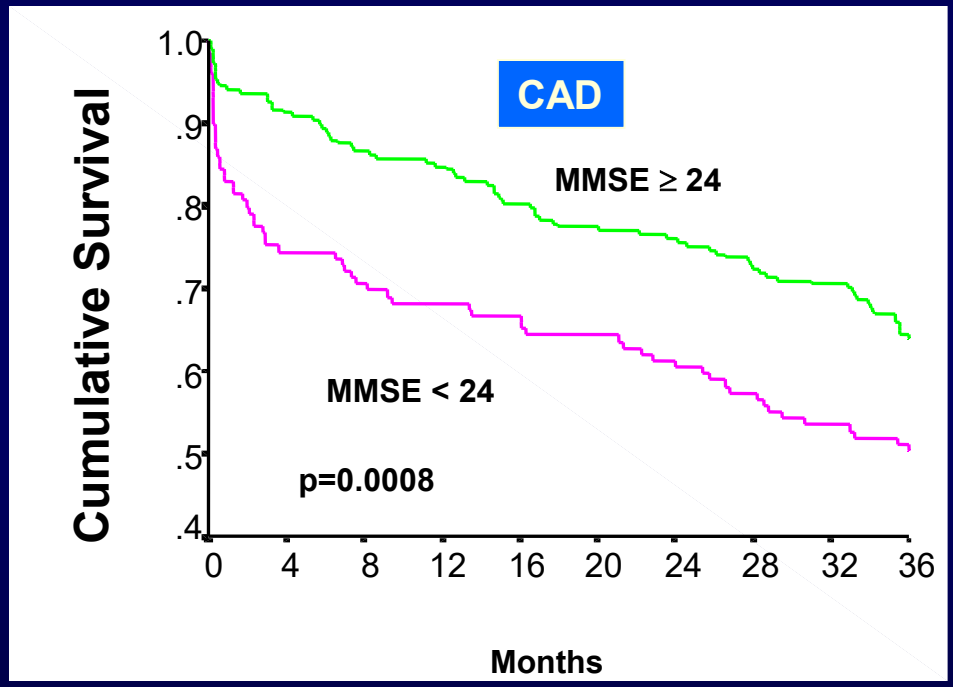
Logistic regression analysis of the effect of sex, age, educational level, GDS score, systolic and diastolic blood pressure, heart rate and congestive heart failure on Mini-Mental State Examination.

Variables	OR	95% CI	p value
Sex (Female/Male)	1.40	1.01-1.95	0.04
Age (65-96)	1.10	1.08-1.13	0.000
Educational level^	0.45	0.39-0.51	0.000
GDS score	1.07	1.04-1.10	0.000
SBP (mmHg)	0.99	0.98-1.00	0.16
DBP (mmHg)	1.02	1.01-1.05	0.02
HR (bpm)	0.98	0.96-0.99	0.03
CHF	**1.92**	**1.10-3.36**	**0.02**

Cacciatore F et al., JAGS 1998

Cumulative Survival in Elderly Patients with HF or CAD stratified for the presence or the absence of Cognitive Impairment (MMSE < 24)





CHF Italian Study, unpublished data

Heart Failure and cognitive impairment

Hypertension and cognitive impairment

Hypertension, have a negative correlation with cognitive functions, especially with memory, attention and executive functions (Hanon 2005, Vicario 2005)

Long-term high blood pressure interferes with brain perfusion leading to chronic ischemic lesions and silent strokes (Staessen 1997, Ciobica 2009)

Hypertension can be also involved in amyloid β deposits or neurofibrillary tangles formation (Lee 2003, Bomboi 2010)

Chronic hypertension lead to ventricular enlargement, silent infarct, white matter lesions and brain atrophy, when compared to normotensive individuals Vermeer 2003, Takeda 2008, Nagai 2010). These brain changes could determine cognitive regression which is often found in patients with chronic hypertension (Takeda 2008, Ciobica 2009)

The reduction of systolic blood pressure may have a protective effect against cognitive impairment (Fogari 2003, 2006).

The role of blood pressure in cognitive impairment in an elderly population

Francesco Cacciatore, Pasquale Abete, Nicola Ferrara*, Giuseppe Paolisso†, Laura Amato†, Silvestro Canonico‡, Stefania Maggi**, Michele Varricchio† and Franco Rengo, for the 'Osservatorio Geriatrico Campano Group'

Objective The aim of this study was to investigate the cross-sectional relationship between arterial blood pressure and cognitive impairment in a group of elderly subjects, controlling for such confounding variables as age, education, depression, drug use and antihypertensive treatment.

Design and setting A cross-sectional survey in Campania, a region in southern Italy.

Subjects and methods A random sample of 1339 elderly subjects aged 65–95 years (mean 73.9 ± 6.2 years) selected from the electoral rolls was interviewed by trained physicians. Sociodemographic characteristics, results of Mini-Mental State Examination (MMSE), Geriatric Depression Scale (GDS), blood pressure and whether antihypertensive treatment was being administered were recorded. When subjects with neurological diseases and those under psychotropic therapy were excluded from the analyses, the population numbered 1106.

Results The MMSE score was less than 24 for 27.8% of the subjects and the mean GDS score was 10.8 ± 6.3. The mean systolic blood pressure (SBP) was 145.3 ± 19.0 mmHg and the mean diastolic blood pressure (DBP) was 82.0 ± 9.2 mmHg. Logistic regression analysis showed that female sex, age, GDS score and DBP but not SBP were predictive of cognitive impairment. Educational level and antihypertensive treatment, on the contrary, play a protective role. DBP was associated with cognitive impairment in subjects aged 75 years (odds ratio 1.62, 95% confidence interval 1.16–2.25) and over (odds ratio 5.16, 95% confidence interval 1.50–17.71) but not in those aged 65–74 years.

Conclusion DBP but not SBP is predictive of cognitive impairment in subjects aged 75 years and over without neurological disorders independently from sex, age, education, GDS and antihypertensive treatment.

Journal of Hypertension 1997, 15:135–142

Keywords: ageing, blood pressure, cognitive impairment

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Sponsorship: This study was supported by a grant from the Regione Campania to the Osservatorio Geriatrico Regione Campani.

Requests for reprints to Dr Franco Rengo, Istituto di Medicina Interna, Cardiologia e Chirurgia Cardiovascolare, Cattedra di Geriatria, Università degli Studi di Napoli 'Federico II', Via S. Pansini 5, 80131 Napoli, Italy.

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Table 3 Distribution of variables according to the Mini-Mental State Examination results < 24 and ⩾ 24

Variables	MMSE < 24	MMSE ⩾ 24	Significance (*P*)
Age (years)	75.3 ± 6.7	72.1 ± 5.l	0.0001
Educational level	2.8 ± 1.3	4.2 ± 1.2	0.0001
GDS score	12.6 ± 6.2	8.6 ± 5.8	0.0001
SBP (mmHg)	146.6 ± 19.3	143.7 ± 18.4	0.01
DBP (mmHg)	82.8 ± 9.l	81.0 ± 9.0	0.001

Values are expresssed as means ± SD. GDS, Geriatric Depression Scale; SBP, systolic blood pressure; DBP, diastolic blood pressure.

F.Cacciatore et al. J Hypertension. 1997

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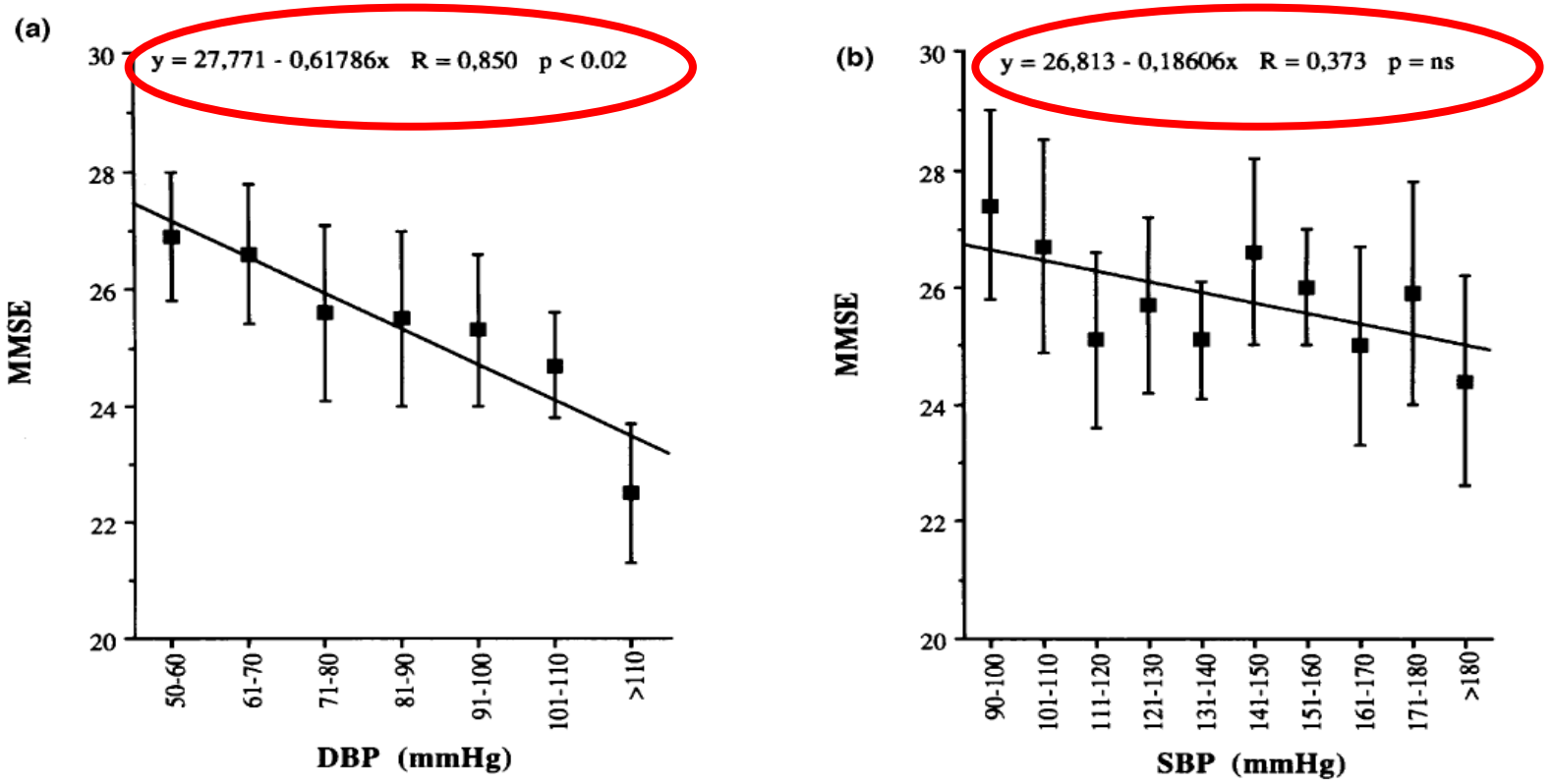


F.Cacciatore et al. J Hypertension. 1997

Role of anti-hypertensive treatment on cognitive impairment

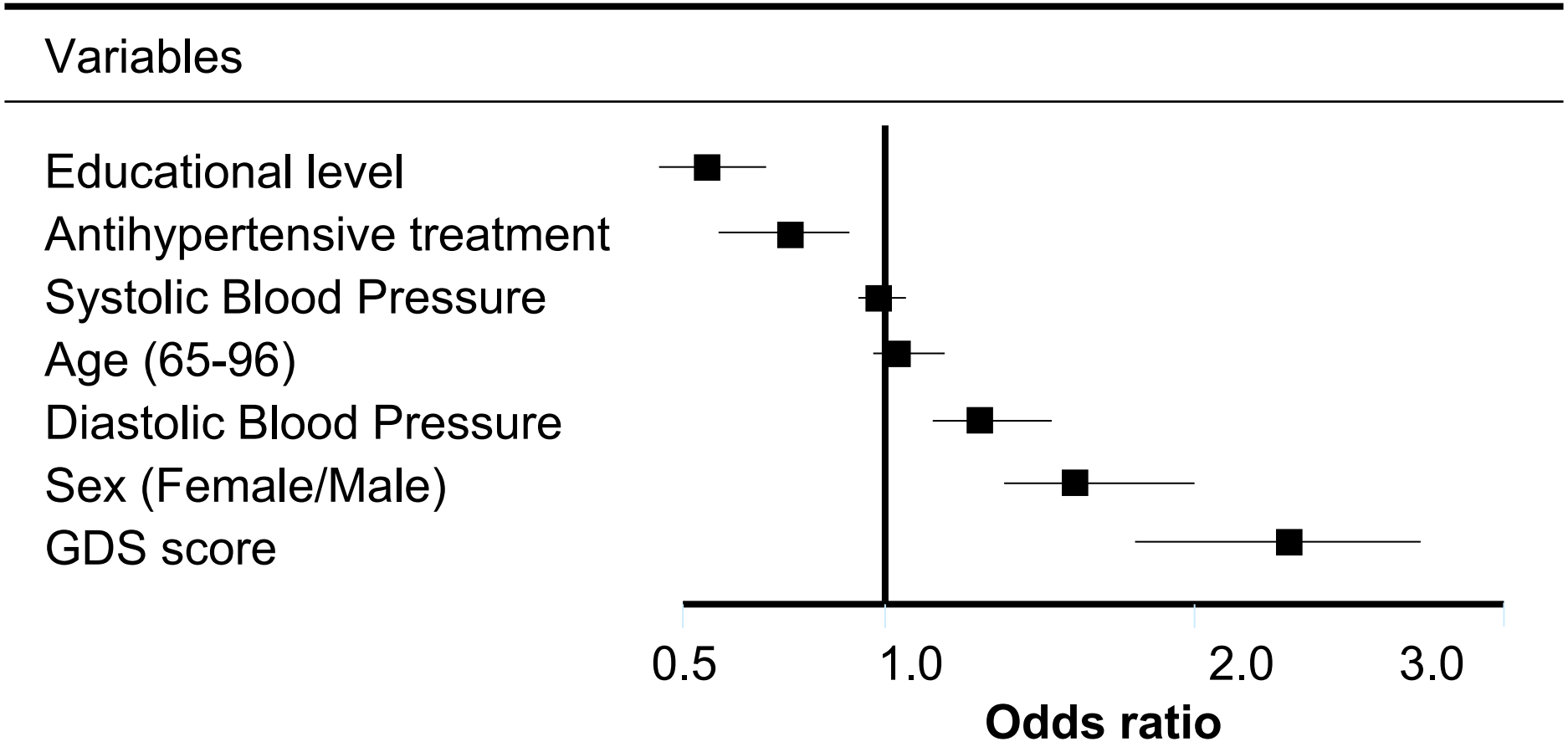
Table 4 Logistic regression analysis considering the role played by various variables in the Mini-Mental State Examination results

Variables	Odds ratio (95% CI)	Significance
Sex (female/male)	1.51 (1.30–1.91)	$P = 0.05$
Age (65–96 years)	1.09 (1.07–1.10)	$P = 0.0001$
Educational level	0.44 (0.23–0.46)	$P = 0.0001$
GDS score	2.22 (1.85–2.66)	$P = 0.0001$
SBP	1.00 (0.99–1.01)	NS
DBP	1.29 (1.19–1.37)	$P = 0.001$
Antihypertensive treatment	0.70 (0.59–0.98)	$P = 0.03$

CI confidence interval; GDS, Geriatric Depression Scale; SBP, systolic blood pressure; DBP, diastolic blood pressure.

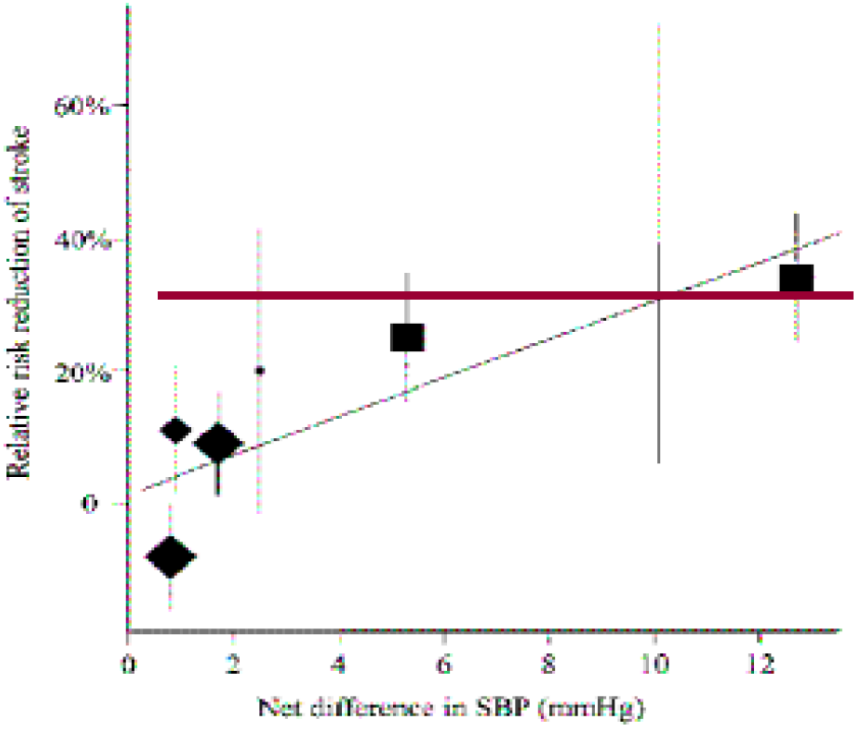
Cacciatore F et al. J Hypertens 1997

Logistic regression analysis considering the role of sex, age, educational level, GDS score, SBP, DBP and antihypertensive treatment on MMSE.



Cacciatore F et al., J Hypertens 199

Riduzione del rischio di ictus e trattamento antipertensivo



La riduzione netta dei valori di PAS e la riduzione del rischio relativo di stroke ottenuta dai trial clinici randomizzati sono valutatati mediante una meta-regressione, ovvero una relazione diretta tra la riduzione dei valori di PAS plottati verso la riduzione del rischio di stroke per ognuna delle 7 meta-analisi. Lo slope della curva indica che per una riduzione di 10 mmHg si osserva una riduzione del rischio di stroke del 31%. R^2=71%

Lawes, Stroke 2004

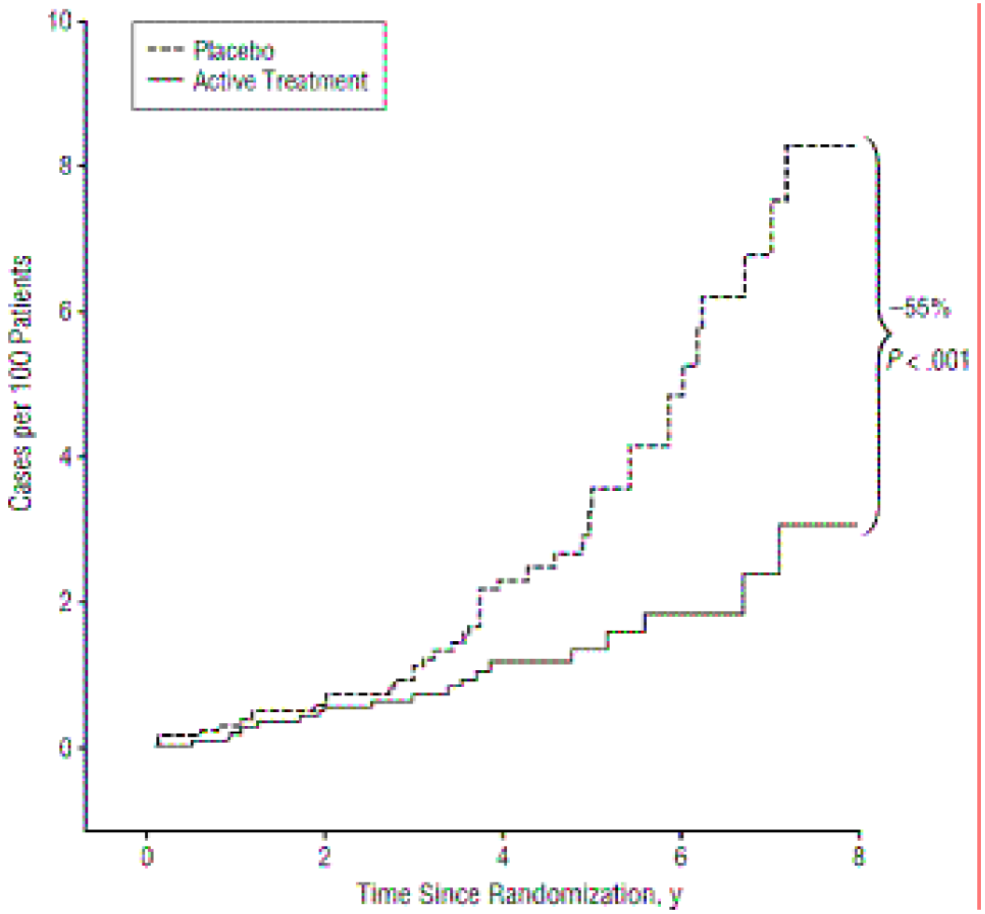
The Prevention of Dementia With Antihypertensive Treatment

New Evidence From the Systolic Hypertension in Europe (Syst-Eur) Study

Arch Intern Med. 2002;162:2046-2052

Treatment consisted of nitrendipine (10-40 mg/d) (70.2%), with the possible addition of enalapril maleate (5-20 mg/d) (35.4%), hydrochlorothiazide (12.5-25 mg/d) (18.4%), or both add-on drugs.

"Nitrendipine" calcium channel Blocker dihydropyridines cross the blood-brain barrier.



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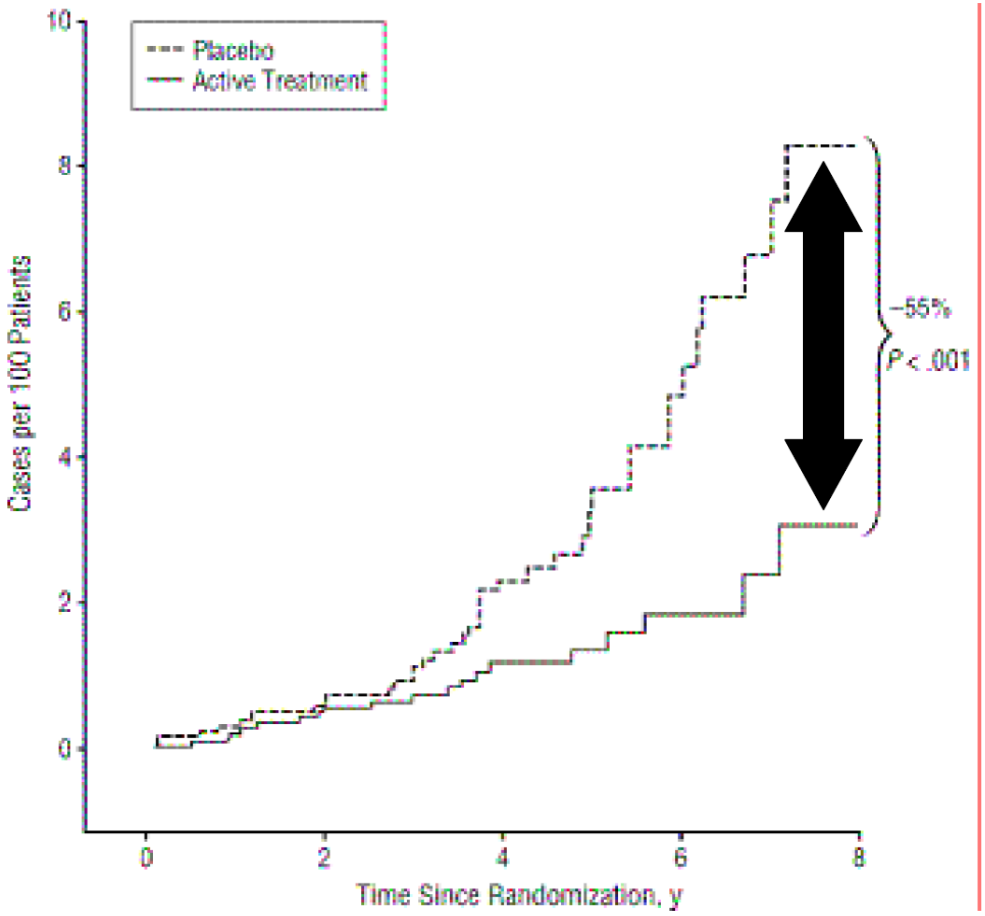
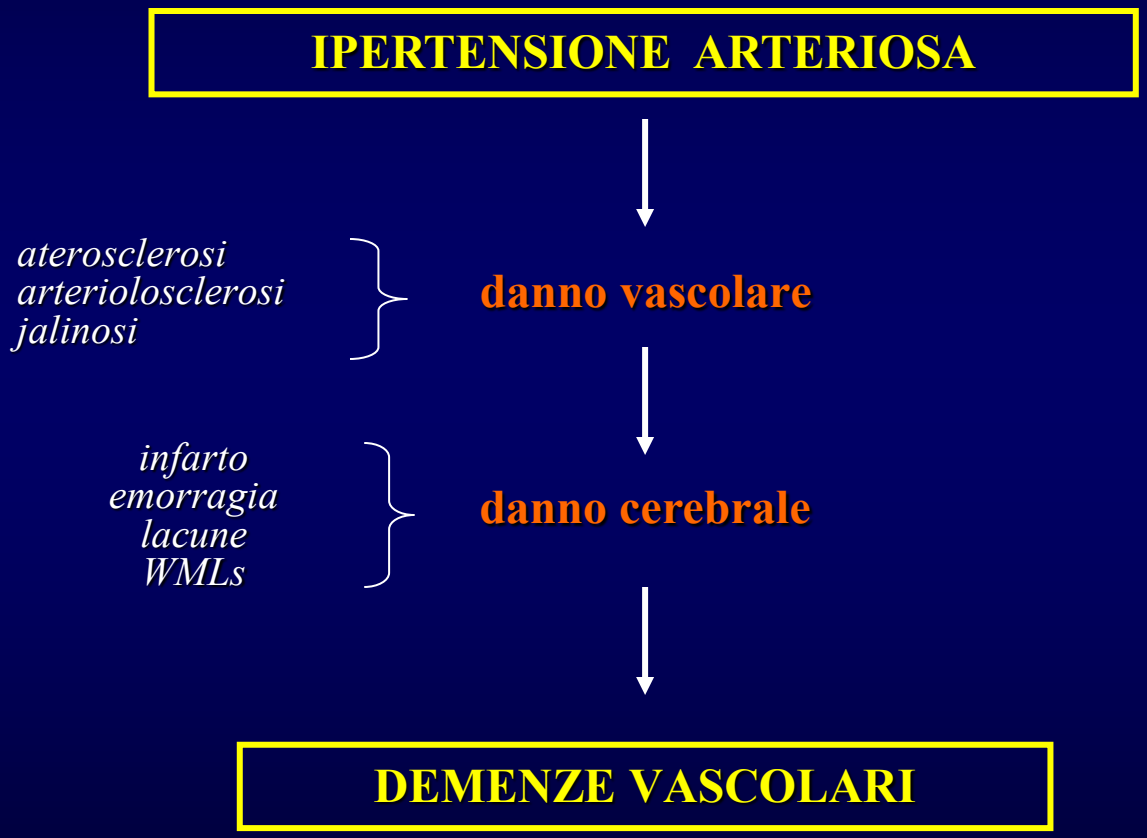


IPERTENSIONE ARTERIOSA E FUNZIONI COGNITIVE NELL'ANZIANO



Hypertension

Aging

Progressive obliteration of cerebral arterioral bed

Ischemic lesions

VASCULAR DEMENTIA

Low blood pressure

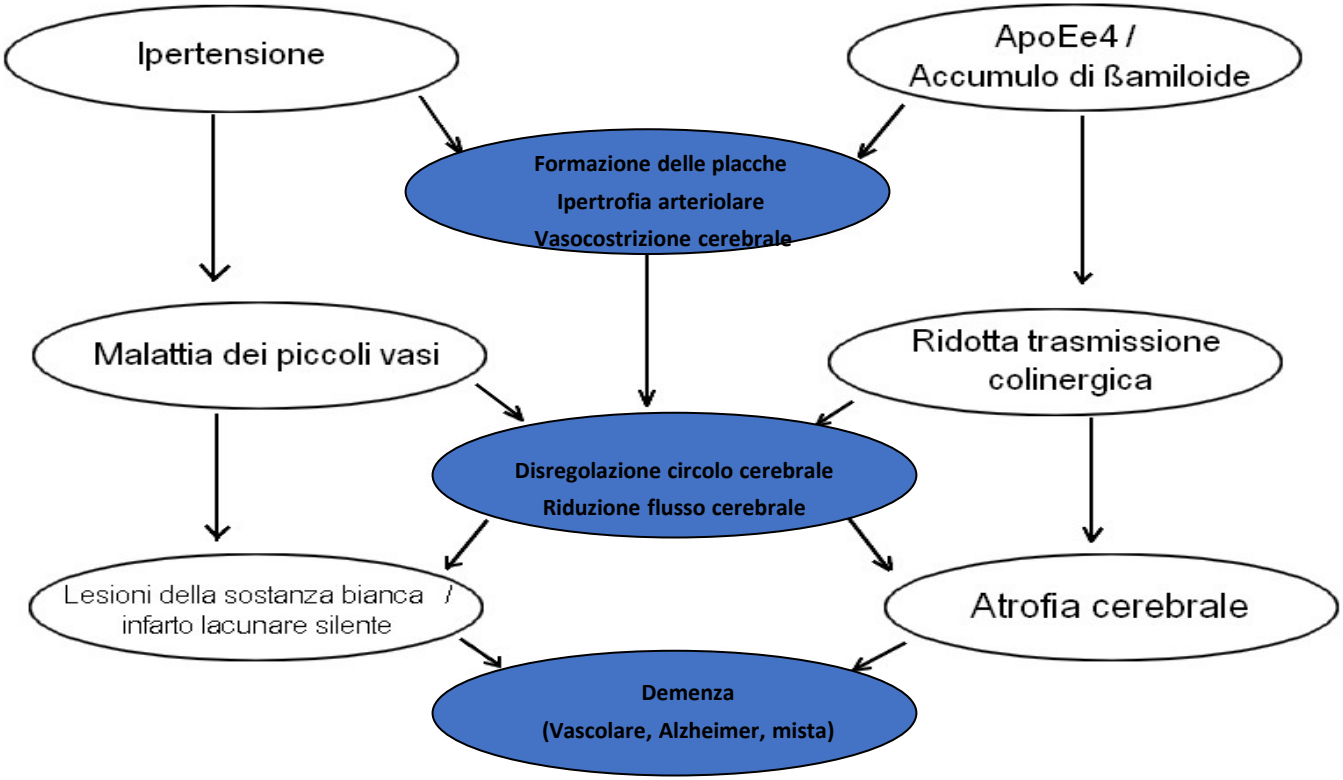
(Iatrogenic effects?)

(CHF?)

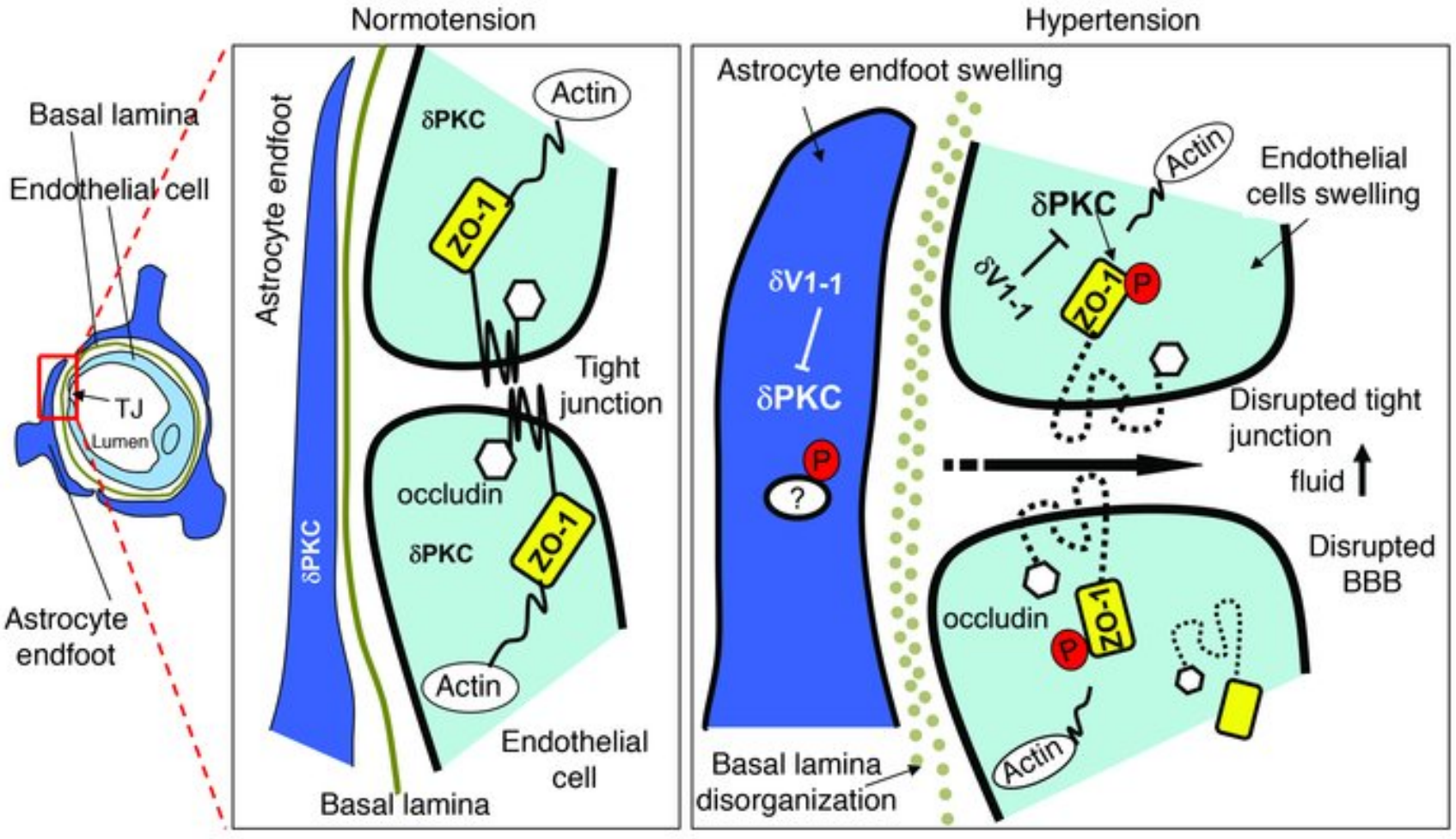
(Frailty?)

COGNITIVE IMPAIRMENT

Momenti fisiopatologici comuni per Demenza Vascolare e Malattia di Alzheimer ed ipertensione



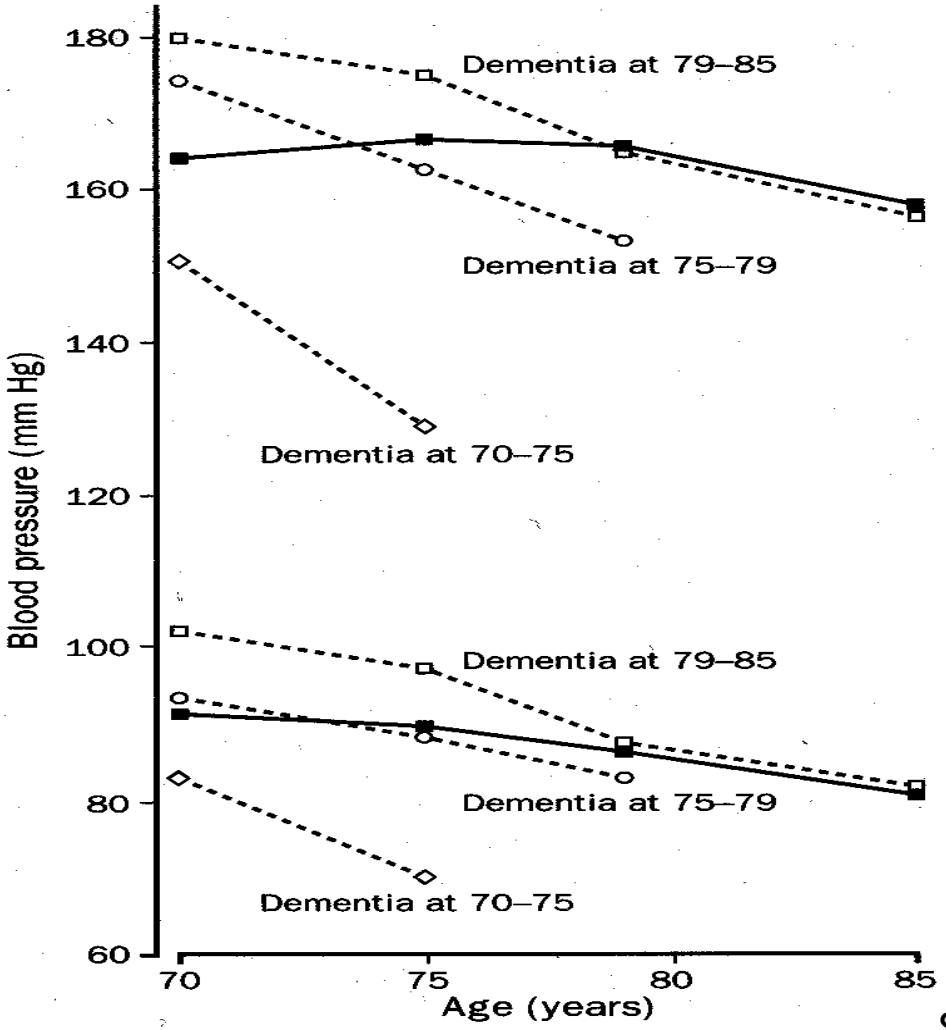
Blood brain barrier dysfunction



Scheme of a cerebral microvessel. Hypertension causes swelling of endothelial and endfeet of astrocytes surrounding the small vessels of brain and increases immunoreactivity of δPKC (right versus middle). Consequently, BBB permeability increased.

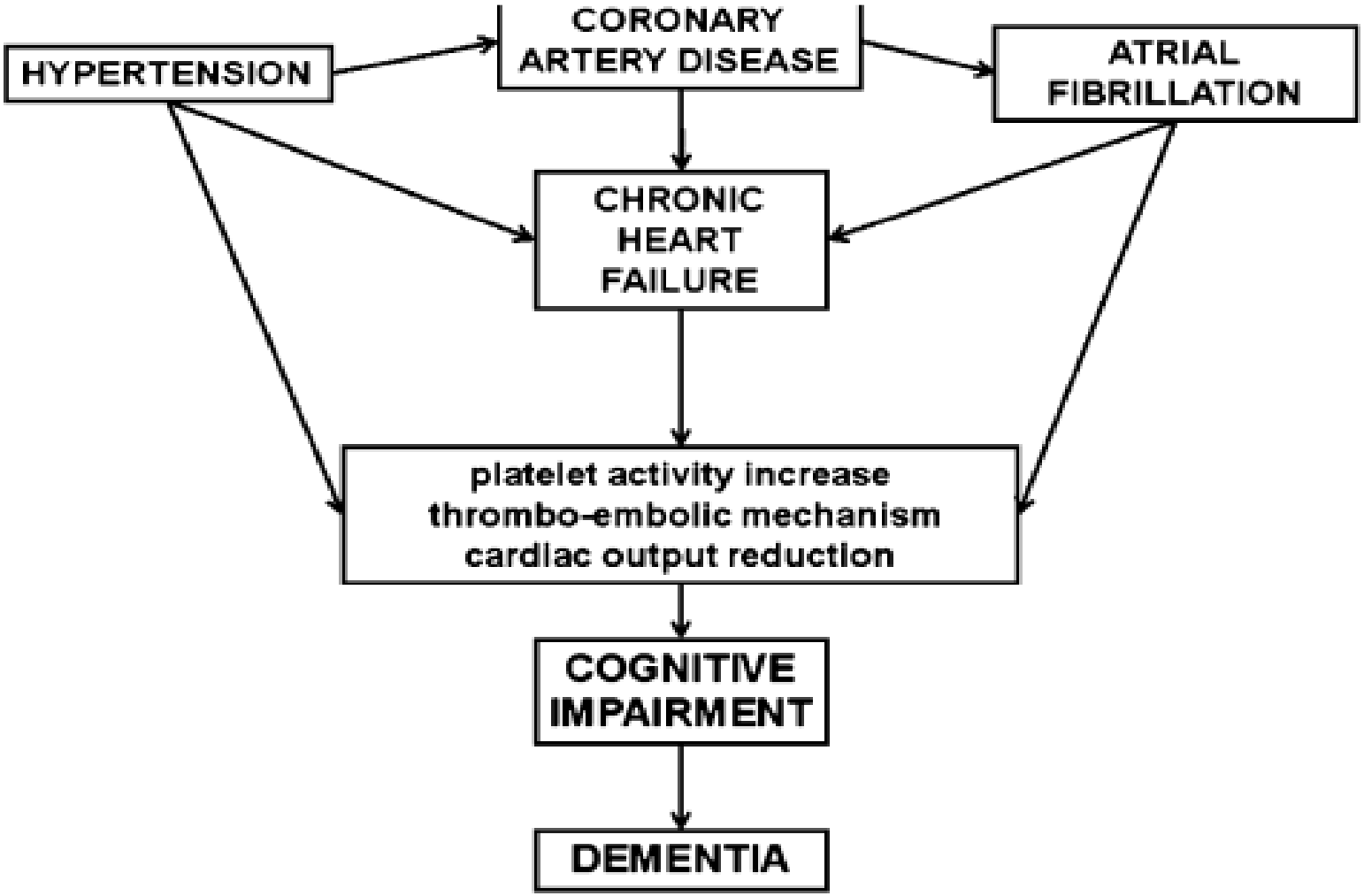
X Qi et al J Clin Invest, 2008

A 15-year follow-up of blood pressure and dementia



Skoog et al. Lancet 1996

Cognitive impairment and cardiovascular diseases in the elderly. A heart–brain continuum hypothesis



Abete P et al. Aging Res Rev, 2014

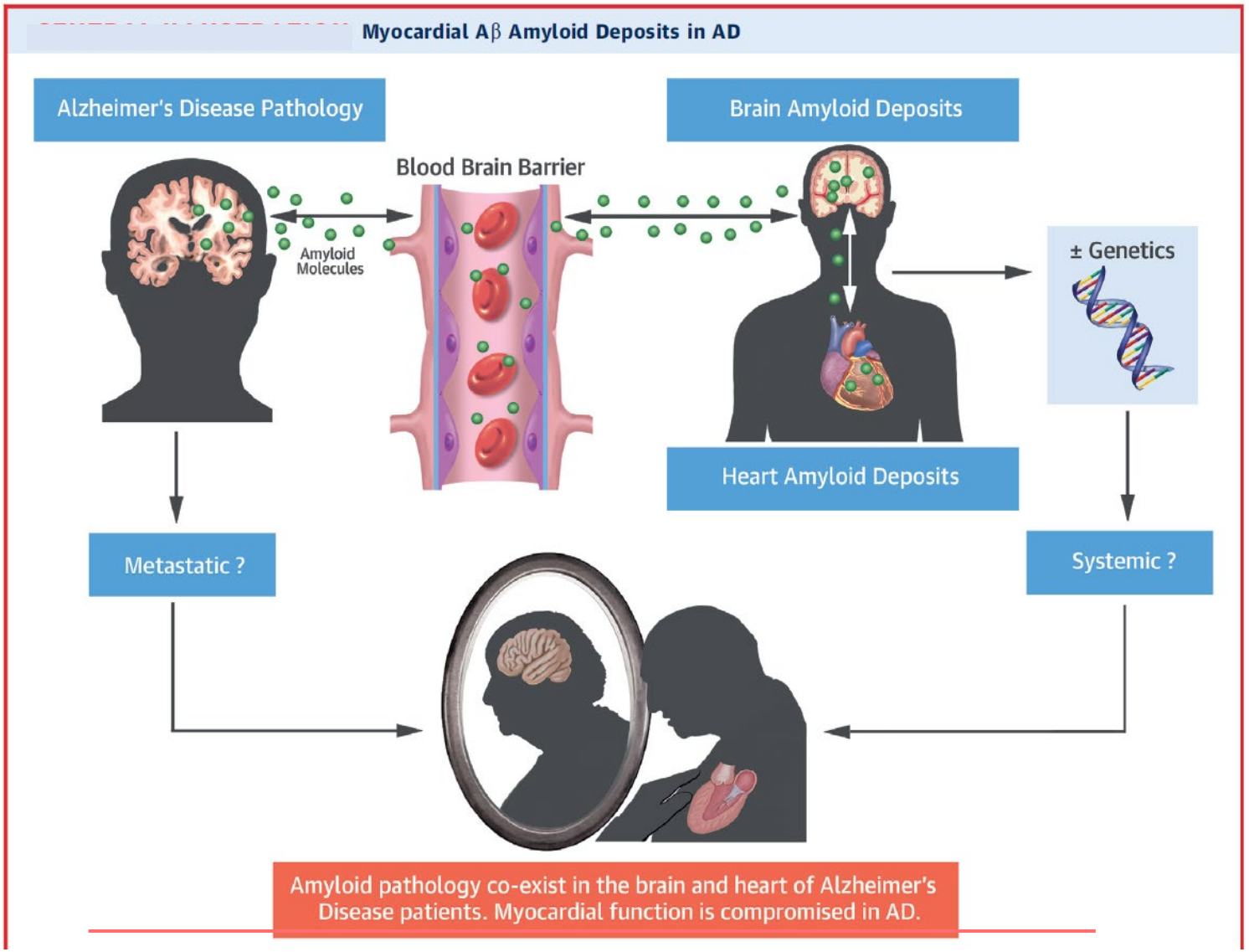
ORIGINAL INVESTIGATIONS

Aβ Amyloid Pathology Affects the Hearts of Patients With Alzheimer's Disease

Mind the Heart

Luca Troncone, PhD,[a] Marco Luciani, MD,[a] Matthew Coggins, MD,[a] Elissa H. Wilker, ScD,[a,b] Cheng-Ying Ho, MD, PhD,[c] Kari Elise Codispoti, MD,[c] Matthew P. Frosch, MD, PhD,[d] Rakez Kayed, PhD,[e] Federica del Monte, MD, PhD[a,f]

J Am Coll Card, 2016



Troncone L, et al. J Am Coll Card, 2016

From Stroke-Heart Syndrome to Alzheimer Heart Disease

TOWERS HOTEL
STABIAE SORRENTO COAST
★★★★

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4° Congresso Nazionale SIONG (Società Italiana OtoNeuroGeriatria)

Thank you for your attention

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Director Saint Joseph Nursing Home