

Scompenso cardiaco

Progressi diagnostico-terapeutici “DOPO IL DILUVIO”

Prof Labriola

**46° CORSO ANNUALE DI AGGIORNAMENTO MEDICO
“FRANCO FERRATINI”**

Organizzato dalla Facoltà di Medicina e Chirurgia
dell'Università degli Studi di Bologna
nell'ambito dell'attività ECM

19 ottobre 2006

Appunti della lezione del

Prof. ERNESTO LABRIOLA
“L'insufficienza cardiaca: diagnosi difficile”



A cura dei Dott.ri Orietta Baiesi e Vincenzo Cavara

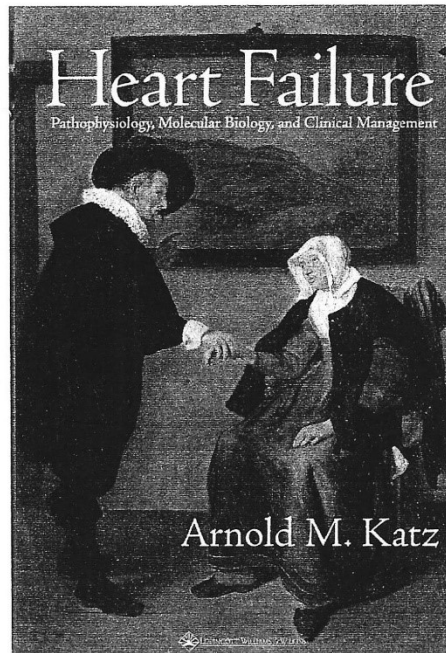
43° CORSO ANNUALE DI AGGIORNAMENTO MEDICO
“FRANCO FERRATINI”

Nell'ambito dell'attività E.C.M. della Facoltà di Medicina e Chirurgia
Ente Organizzatore Università di Bologna
con il Patrocinio dell'Ordine dei Medici Chirurghi della Provincia di Bologna

6 novembre 2003

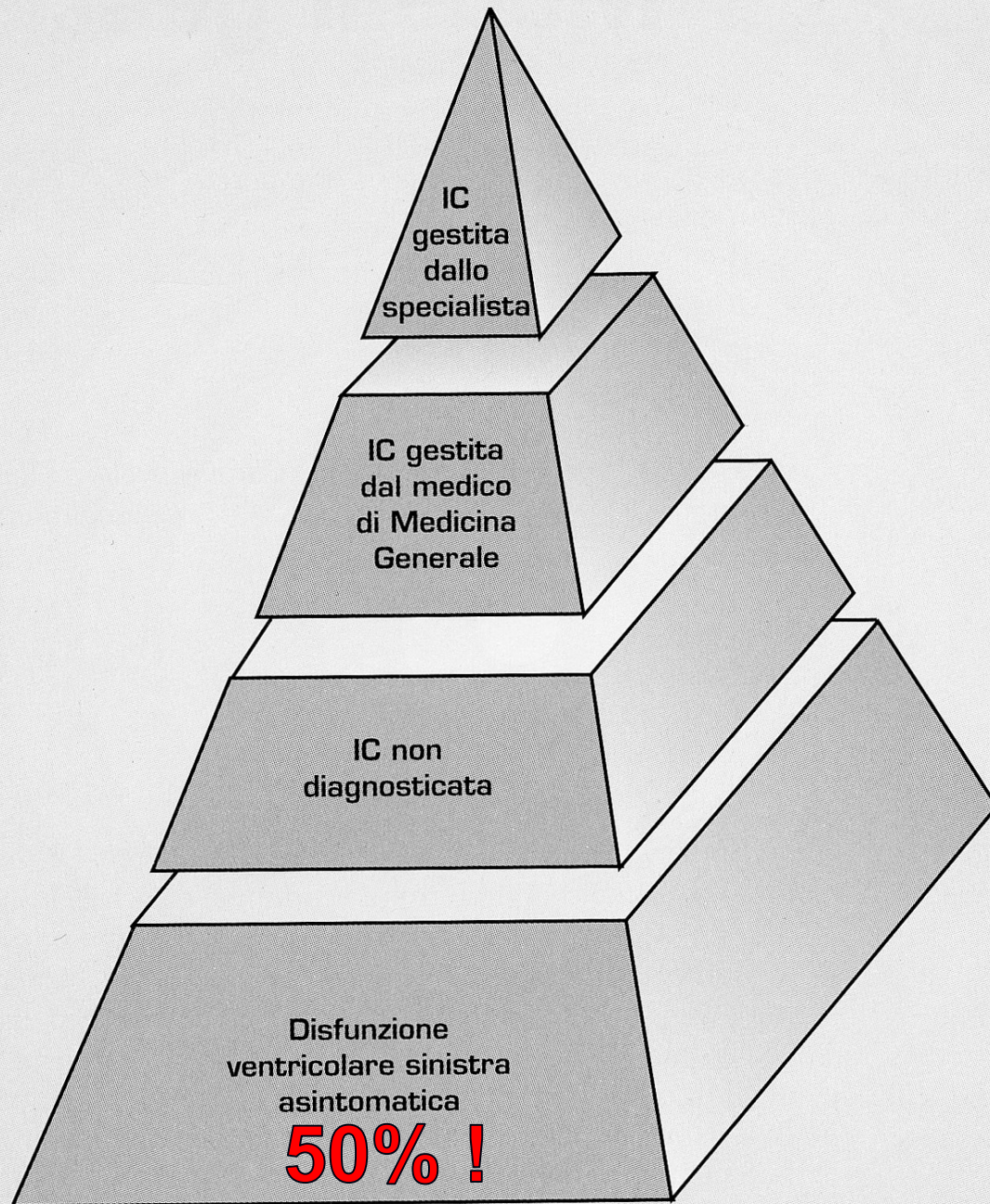
Appunti della lezione del
Prof. BRUNO MAGNANI

“Si può prevenire lo scompenso cardiaco?”



Le medicine non mi soccorrono più, aumenta l'enfiagione delle mie gambe; e sonnacchio seduto più che disteso. Uno dei vantaggi della morte sarà d'essere disteso ancora, in un letto. Mi hanno portato a Baia; con questo caldo di luglio, il tragitto è stato penoso, ma in riva al mare respiro meglio. L'onda manda alla riva il suo mormorio, fruscio di seta e carezza; godo ancora le lunghe serate rosate.

Memorie di Adriano



I.C.: storia non solo “cardiologica”

Terapie antineoplastiche; obesità viscerale (NAFLD!); diabete; malattie autoimmuni; pregresse gravidanze “a rischio”; tunnel carpale; malattie b. polmonari; endocrinopatie; ecc. ecc.

Segni e sintomi di scompenso cardiaco acuto

Sintomi	
Più tipici	Dispnea a riposo, ortopnea, dispnea parossistica notturna, astenia
Meno tipici	Tosse notturna, vertigine, inappetenza, dispepsia, dolore addominale, confusione, perdita di coscienza, palpitazione
Segni	
Più specifici	Rantoli polmonari, turgore delle giugulari, reflusso epatogiugulare, terzo tono, ritmo di galoppo, lateralizzazione dell'itto
Meno specifici	Incremento o decremento polmonare, soffi cardiaci, edemi periferici, ittero, riduzione del fremito vocale tattile, del murmure respiratorio ed ottusità basali, tachicardia, polsi irregolari ed iposfigmici, tachipnea, epatomegalia, ascite, cute fredda alle estremità, diaforesi, oliguria

I.C.: turbe del respiro

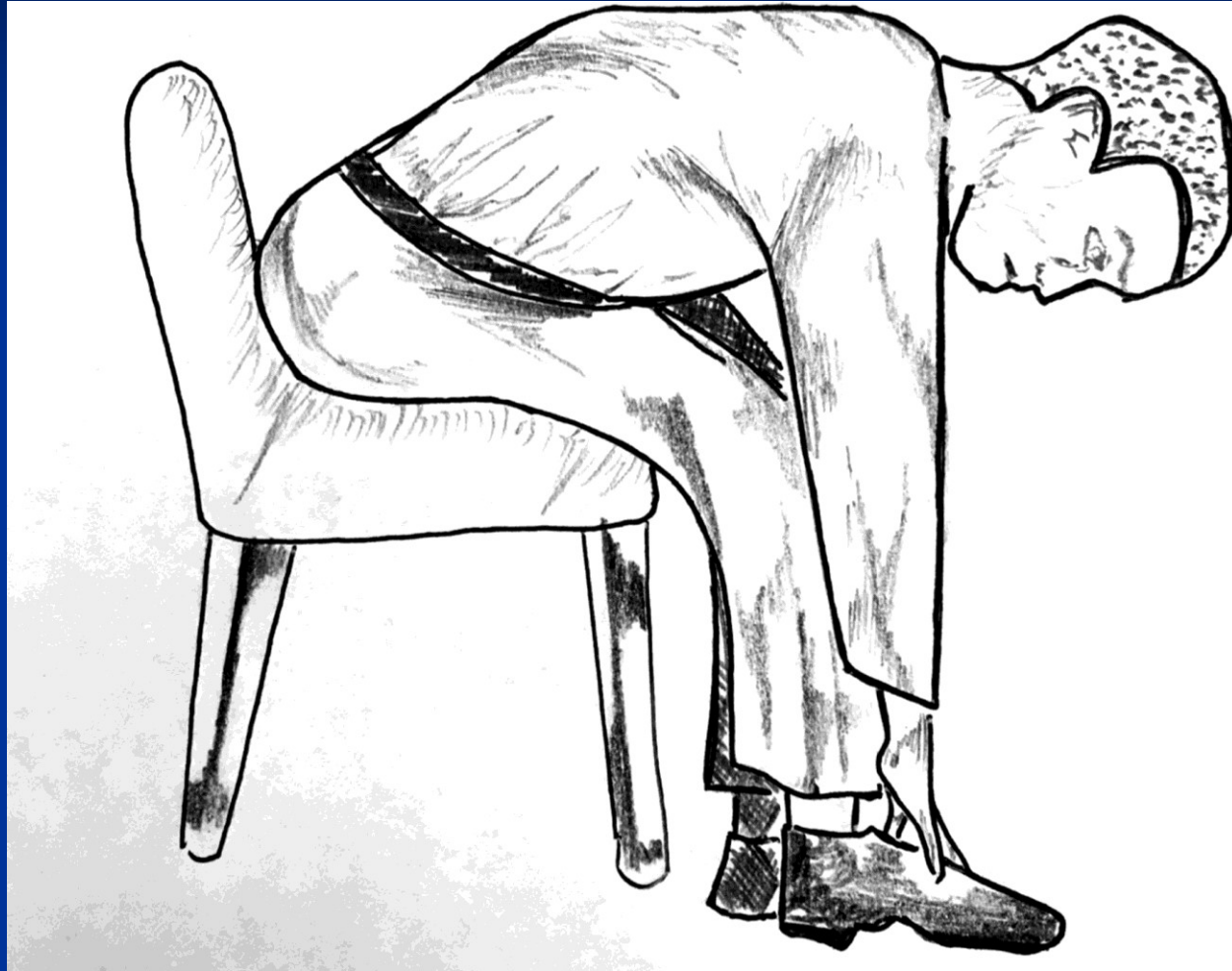
- Polipnea
- Dispnea
- Ortopnea
- Bendopnea
- Platipnea – ortodessia
- Apnea (centrale, ostruttiva)

Apnee “centrali”

Il suo respiro era lento e profondo, come una persona che dimentica per un po' il bisogno di respirare, per poi ricordarsene improvvisamente

Ippocrate

Dispnea entro 30 sec!



Thibodeau JT and Drazner MH, J Am Cardiol HF 2018; 6:543-51

Accuratezza diagnostica dei sintomi

Harlan WR, et al. Ann Int Med, 1977

	Sensibilità	Specificità	Cause non cardiache
Dispnea da sforzo	75%	50%	Malattie Polmon. (BPCO - Fibrosi) TEP
Dispnea parossistica notturna	31%	76%	Sindrom.Nefros. Insuff Ren. Anemia Obesità
Ortopnea	21%	81%	Ansietà Decondiz. Fisico

ALTRI SINTOMI

(bassa sensibilità e specificità, in genere tardivi):

Disturbi addominali (gonfiore, nausea, dolore ipocondrio destro, anoressia)

Stanchezza e facile stancabilità - Pallore ed altri segni di ipoperfusione periferica

Confusione mentale (anziani)

Dyspnea: COPD or Heart Failure?

- Both conditions present with dyspnea
- They are both chronic conditions
- Both conditions present in the elderly
- Incidence
 - 20% of COPD patients will have HF^[a]
 - 20% of HF patients will have COPD^[b]
- Similarities of symptoms at night
 - PND is associated with HF
 - Bronchoconstriction associated with COPD is also worse at night

a. McCullough PA, et al. *Acad Emerg Med*. 2003;10:198-204.

b. De Miguel Diez J, et al. *Int J COPD*. 2013;8:305-312.

Prevalence of unrecognized heart failure in older persons with shortness of breath on exertion

Evelien E.S. van Riet^{1*}, Arno W. Hoes², Alexander Limburg³,
Marcel A.J. Landman⁴, Henk van der Hoeven⁵, and Frans H. Rutten⁶

^{1,2,6}Julius Center for Health Sciences and Primary care, University Medical Center Utrecht, PO Box 85500, 3508 AB Utrecht, the Netherlands; ^{3,5}Diakonessenhuis Zeist, Zeist, the Netherlands; and ⁴Meander Medical Center, Amersfoort, the Netherlands

Conclusions

Both GPs and pulmonologists should be aware that patients with shortness of breath on exertion could have unrecognized heart failure, mainly with preserved ejection fraction. To improve early detection, GPs should be alert to heart failure in these persons, also when they are already labelled as having a respiratory disease.

Health Care Use Before First Heart Failure Hospitalization

Identifying Opportunities to Pre-Emptively Diagnose Impending Decompensation



Kim Anderson, MD, MSc,^{a,b} Heather J. Ross, MD, MHSc,^{c,d} Peter C. Austin, PhD,^b Jiming Fang, PhD,^b
Douglas S. Lee, MD, PhD^{b,c,d}

CONCLUSIONS Patients consulted physicians multiple times before their incident acute HF hospitalization. These health care contacts could represent missed opportunities to prevent hospitalizations for HF.

J Am Coll Cardiol HF 2020;8:1024–34

Breathlessness -- Are Lung Disease and HF Coexisting?

- It is important to exclude respiratory disease when breathlessness is present
- With breathlessness, respiratory disease and HF may coexist
- In screening studies of patients with COPD in primary care, 25% of the patients that GPs think have COPD actually do not have it; they have HF
- COPD is a common comorbidity in patients with HF, but it is important to distinguish between the 2

Scompenso cardiaco Segni

Stevenson LW, et al. JAMA 1989 - Chakko CS, et al. Am J Med 1991

EDEMI DECLIVI

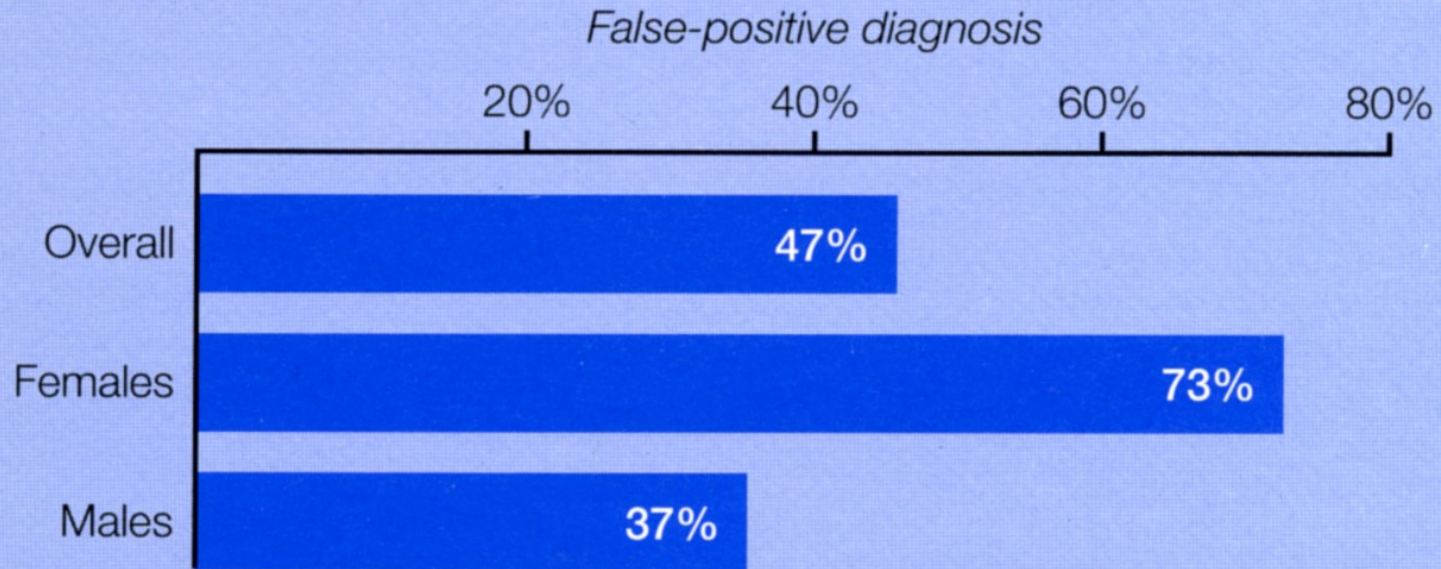
Bassa sensibilità (18% - 23%)

Bassa specificità (se isolati)

Valutare presenza di:

- **Insufficienza venosa**
- **Trombosi venosa profonda**
- **Ipoalbuminemia**
- **Farmaci (calcioantagonisti)**

Misdiagnosis of heart failure in the community



n=78

Patients with presumptive heart failure who underwent echocardiography to determine presence of LV dysfunction.

Wheeldon *et al.* 1993.




Barriers to accurate diagnosis and effective management of heart failure have not changed in the past 10 years: a qualitative study and national survey

Helen C Hancock,¹ Helen Close,¹ Ahmet Fuat,^{2,3} Jerry J Murphy,^{2,3}
A Pali S Hungin,² James M Mason¹

Conclusions: Reported differences in the way heart failure is diagnosed and managed have changed little in the past decade. Variable access to diagnostic tests, modes of care delivery and non-uniform management approaches persist. The current National Health Service (NHS) context may not be conducive to addressing these issues.

Non temo l'I.A. ma la stupidità umana !

Artificial intelligence for the diagnosis of heart failure

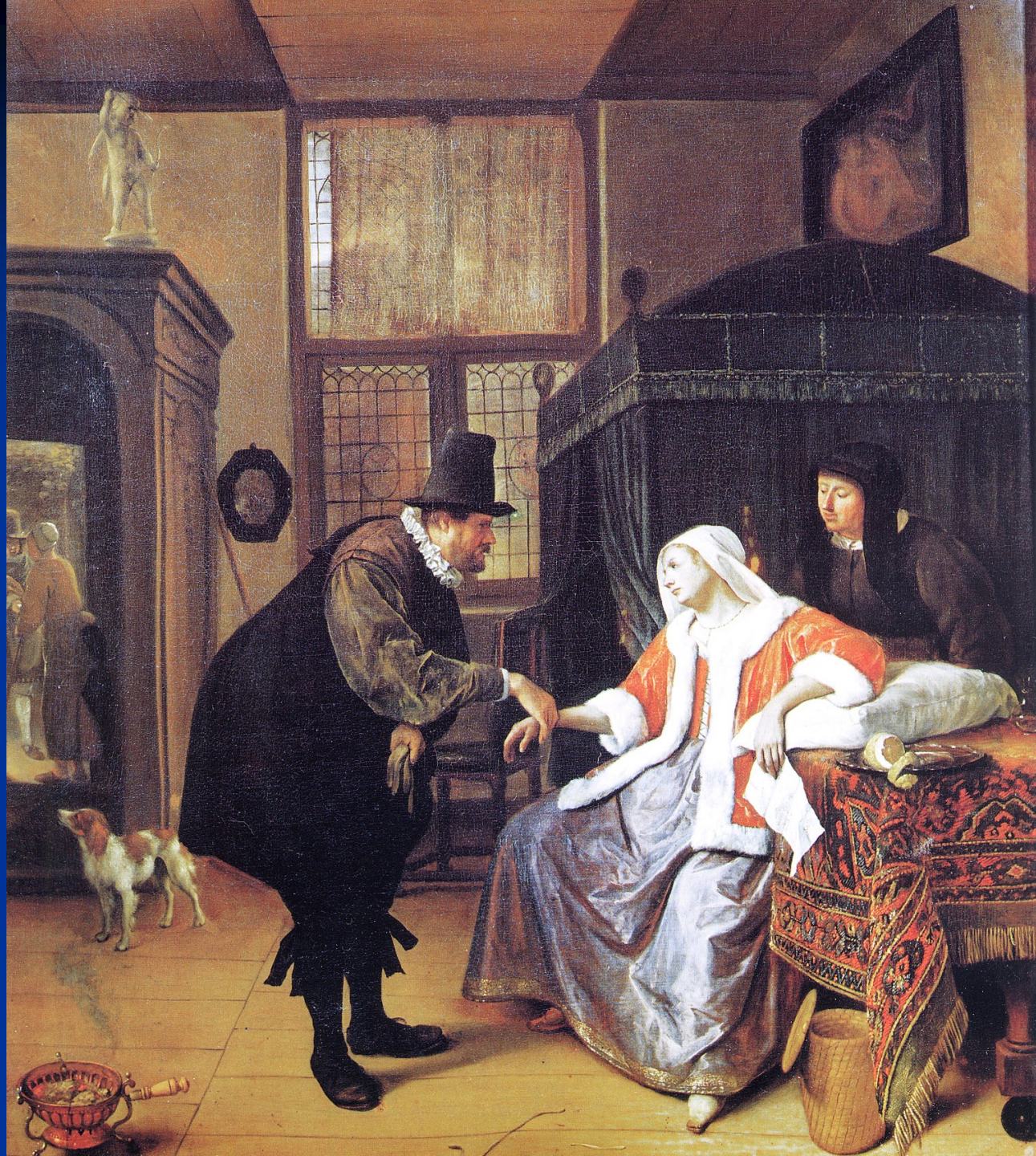
Dong-Ju Choi ^{1,3}✉, Jin Joo Park ^{1,3}, Taqdir Ali² and Sungyoung Lee ²✉

The diagnosis of heart failure can be difficult, even for heart failure specialists. Artificial Intelligence-Clinical Decision Support System (AI-CDSS) has the potential to assist physicians in heart failure diagnosis. The aim of this work was to evaluate the diagnostic accuracy of an AI-CDSS for heart failure. AI-CDSS for cardiology was developed with a hybrid (expert-driven and machine-learning-driven) approach of knowledge acquisition to evolve the knowledge base with heart failure diagnosis. A retrospective cohort of 1198 patients with and without heart failure was used for the development of AI-CDSS (training dataset, $n = 600$) and to test the performance (test dataset, $n = 598$). A prospective clinical pilot study of 97 patients with dyspnea was used to assess the diagnostic accuracy of AI-CDSS compared with that of non-heart failure specialists. The concordance rate between AI-CDSS and heart failure specialists was evaluated. In retrospective cohort, the concordance rate was 98.3% in the test dataset. The concordance rate for patients with heart failure with reduced ejection fraction, heart failure with mid-range ejection fraction, heart failure with preserved ejection fraction, and no heart failure was 100%, 100%, 99.6%, and 91.7%, respectively. In a prospective pilot study of 97 patients presenting with dyspnea to the outpatient clinic, 44% had heart failure. The concordance rate between AI-CDSS and heart failure specialists was 98%, whereas that between non-heart failure specialists and heart failure specialists was 76%. In conclusion, AI-CDSS showed a high diagnostic accuracy for heart failure. Therefore, AI-CDSS may be useful for the diagnosis of heart failure, especially when heart failure specialists are not available.

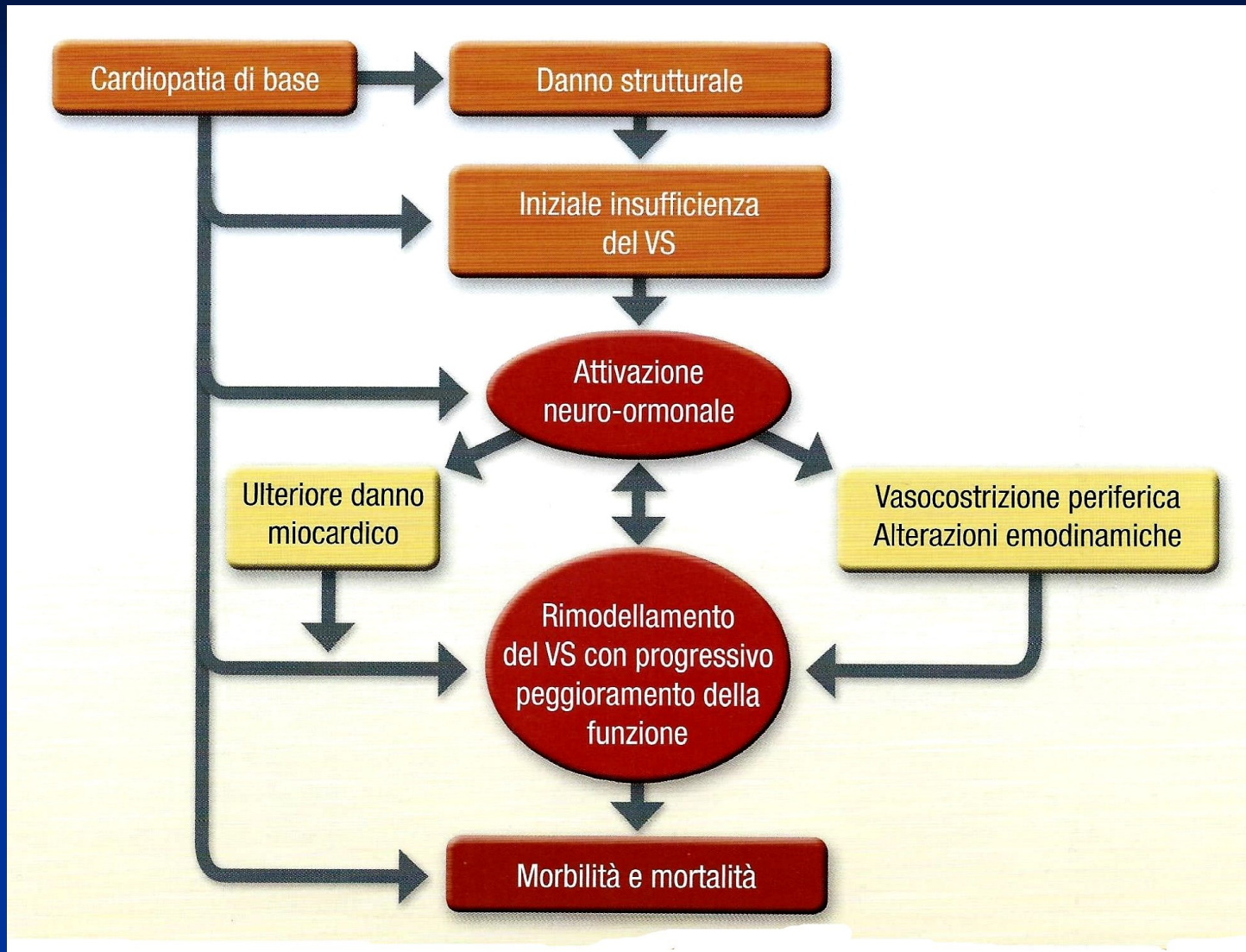
npj Digital Medicine (2020)3:54; <https://doi.org/10.1038/s41746-020-0261-3>

Due cose sono infinite, l'universo e la stupidità umana, ma sull'universo ho ancora dei dubbi.

Albert Einstein



Cuore: organo endocrino!



Natriuretic peptides in the diagnosis of acute HF

Biomarker	Sensitivity (%)	Specificity (%)	Accuracy (%)
BNP* (100 ng/l)	90	76	83
<i>NT-proBNP</i>			
Patient age <50 years (450 pg/ml)	97	93	94
Patient age 50-75 years (900 pg/ml)	90	82	85
Patient age >75 years (1,800 pg/ml)	85	73	83

*BNP does not vary significantly with age. Abbreviations: BNP, B-type natriuretic peptide; HF, heart failure; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Masel AS and Choudhary R., Nat Rev Cardiol 2012; 9:478-490

**Livelli di peptide natriuretico di tipo B:
un possibile analogo della “conta dei
bianchi” nello scompenso cardiaco**

Alan Maisel

I peptidi natriuretici per la gestione dei pazienti con scompenso cardiaco: un ponte fra Medicina Generale e Cardiologia?

Gaetano D'Ambrosio¹, Damiano Parretti¹, Renata De Maria², Iacopo Cricelli¹,
con il contributo di Giuseppe Di Tano³ e Nadia Aspromonte⁴

¹*Società Italiana di Medicina Generale, Firenze*

²*Istituto di Fisiologia Clinica del CNR, Dipartimento Cardiotoracovascolare "A. De Gasperis", A.O. Ospedale Niguarda Ca' Granda, Milano*

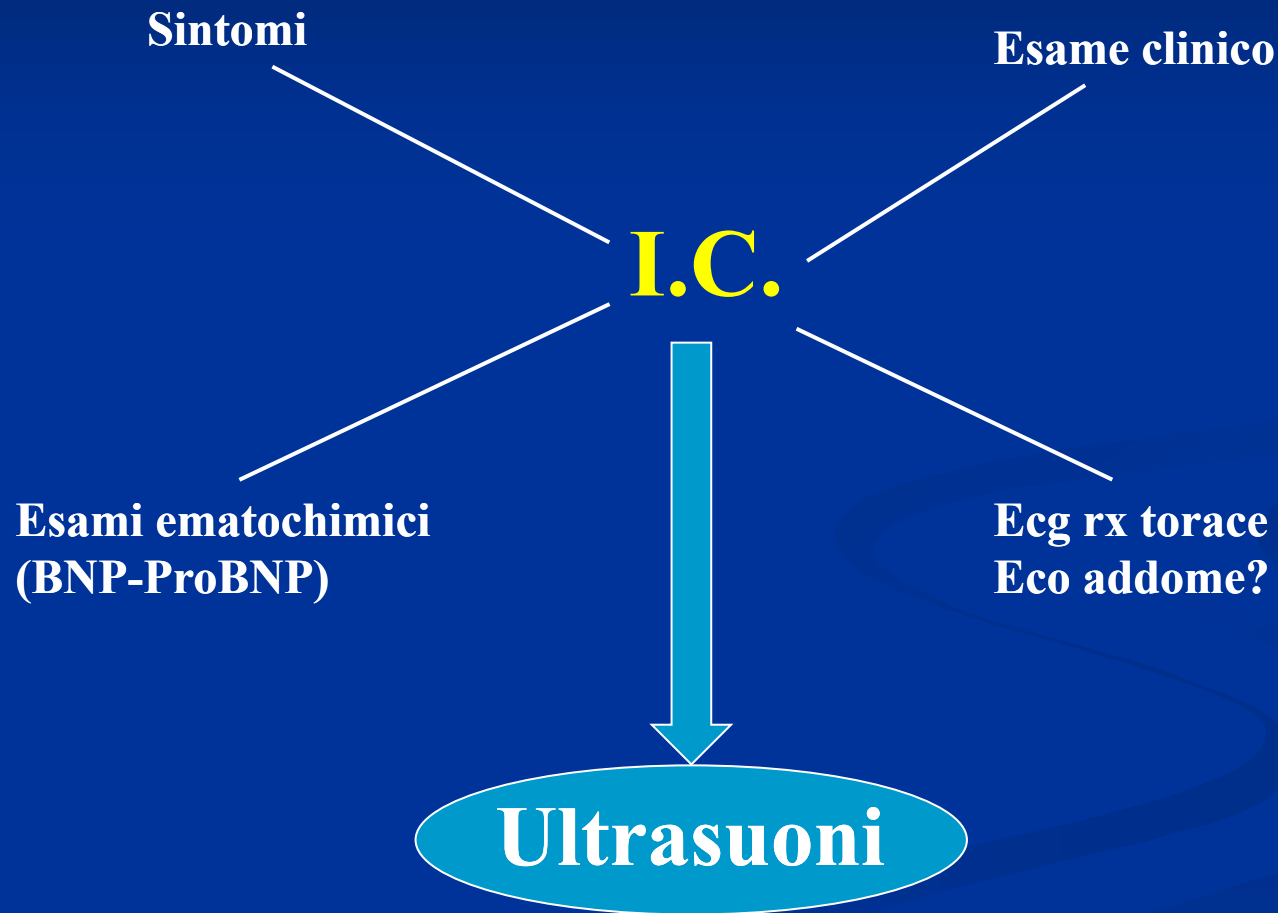
³*U.O. Cardiologia, Azienda Istituti Ospitalieri, Cremona*

⁴*U.O. Cardiologia, Ospedale San Filippo Neri, Roma*

G Ital Cardiol 2016; 17:41-47



I.C.: “approccio” diagnostico



Frazione di eiezione (F.E.)

Rapporto tra gittata sistolica e volume telediastolico: non misura la “contrattilità” ma la funzione di pompa del cuore

HFpEF: Incidence, Prevalence, and Outcomes

- Overall, incidence rates for HF in general are decreasing but not specifically for HFpEF^[a]
- Prevalence of HFpEF approaches 1% to 2% of the overall population^[b]
- HFpEF represents approximately 50% of all HF^[a,c]
- Prevalence is increased in^[a]
 - Patients > 70 years of age
 - Women
 - Obese patients
- Outcomes are variable, depending on severity and comorbidity, but approach those of HFrEF^[d,e]



a. Savarese G, et al. *Cardiac Fail Rev.* 2017;3:7-11; b. Oktay AA, et al. *Curr Heart Fail Rep.* 2013;10:1-17; c. Lam CSP, et al. *Eur J Heart Fail.* 2011;13:18-28; d. Borlaug BA, *Nat Rev Cardiol.* 2014;11:505-515; e. Bello NA, et al. *Circ Heart Fail.* 2014;7:590-595.

Perché è così insidioso lo scompenso cardiaco con frazione di eiezione conservata?

Antonello Gavazzi

FROM Fondazione per la Ricerca, A.O. Papa Giovanni XXIII, Bergamo



Is Cardiac Diastolic Dysfunction a Part of Post-Menopausal Syndrome?

Petra Zubin Maslov, MD, PhD,^a Jin Kyung Kim, MD, PhD,^b Edgar Argulian, MD,^c Amir Ahmadi, MD,^c Nupoor Narula, MD,^d Mandeep Singh, MD,^e Jeroen Bax, MD, PhD,^f Jagat Narula, MD, PhD^c

Post-menopausal women exhibit an exponential increase in the incidence of heart failure with preserved ejection fraction compared with men of the same age, which indicates a potential role of hormonal changes in subclinical and clinical diastolic dysfunction. This paper reviews the preclinical evidence that demonstrates the involvement of estrogen in many regulatory molecular pathways of cardiac diastolic function and the clinical data that investigates the effect of estrogen on diastolic function in post-menopausal women. Published reports show that estrogen deficiency influences both early diastolic relaxation via calcium homeostasis and the late diastolic compliance associated with cardiac hypertrophy and fibrosis. Because of the high risk of diastolic dysfunction and heart failure with preserved ejection fraction in post-menopausal women and the positive effects of estrogen on preserving cardiac function, further clinical studies are needed to clarify the role of endogenous estrogen or hormone replacement in mitigating the onset and progression of heart failure with preserved ejection fraction in women. (J Am Coll Cardiol HF 2019;7:192-203)

Risk of Heart Failure in Patients With Nonalcoholic Fatty Liver Disease



JACC Review Topic of the Week

Alessandro Mantovani, MD,^{a,*} Christopher D. Byrne, MB, BCH,^{b,*} Giovanni Benfari, MD,^c Stefano Bonapace, MD,^d Tracey G. Simon, MD,^{e,f} Giovanni Targher, MD^a

ABSTRACT

Heart failure (HF) and nonalcoholic fatty liver disease (NAFLD) are 2 conditions that have become important global public health problems. Emerging evidence supports a strong and independent association between NAFLD and the risk of new-onset HF, and there are multiple potential pathophysiological mechanisms by which NAFLD may increase risk of new-onset HF. The magnitude of this risk parallels the underlying severity of NAFLD, especially the level of liver fibrosis. Patients with NAFLD develop accelerated coronary atherosclerosis, myocardial alterations (mainly cardiac remodeling and hypertrophy), and certain arrhythmias (mainly atrial fibrillation), which may precede and promote the development of new-onset HF. This brief narrative review aims to provide an overview of the association between NAFLD and increased risk of new-onset HF, discuss the underlying mechanisms that link these 2 diseases, and summarize targeted pharmacological treatments for NAFLD that might also reduce the risk of HF. (J Am Coll Cardiol 2022;79:180-191)

BPCO e cuore sinistro: l'epidemiologia

COPD and left heart disease: epidemiology

*C. Scardurelli, R. Frizzelli;
Rassegna di patologia dell'apparato respiratorio 2014*

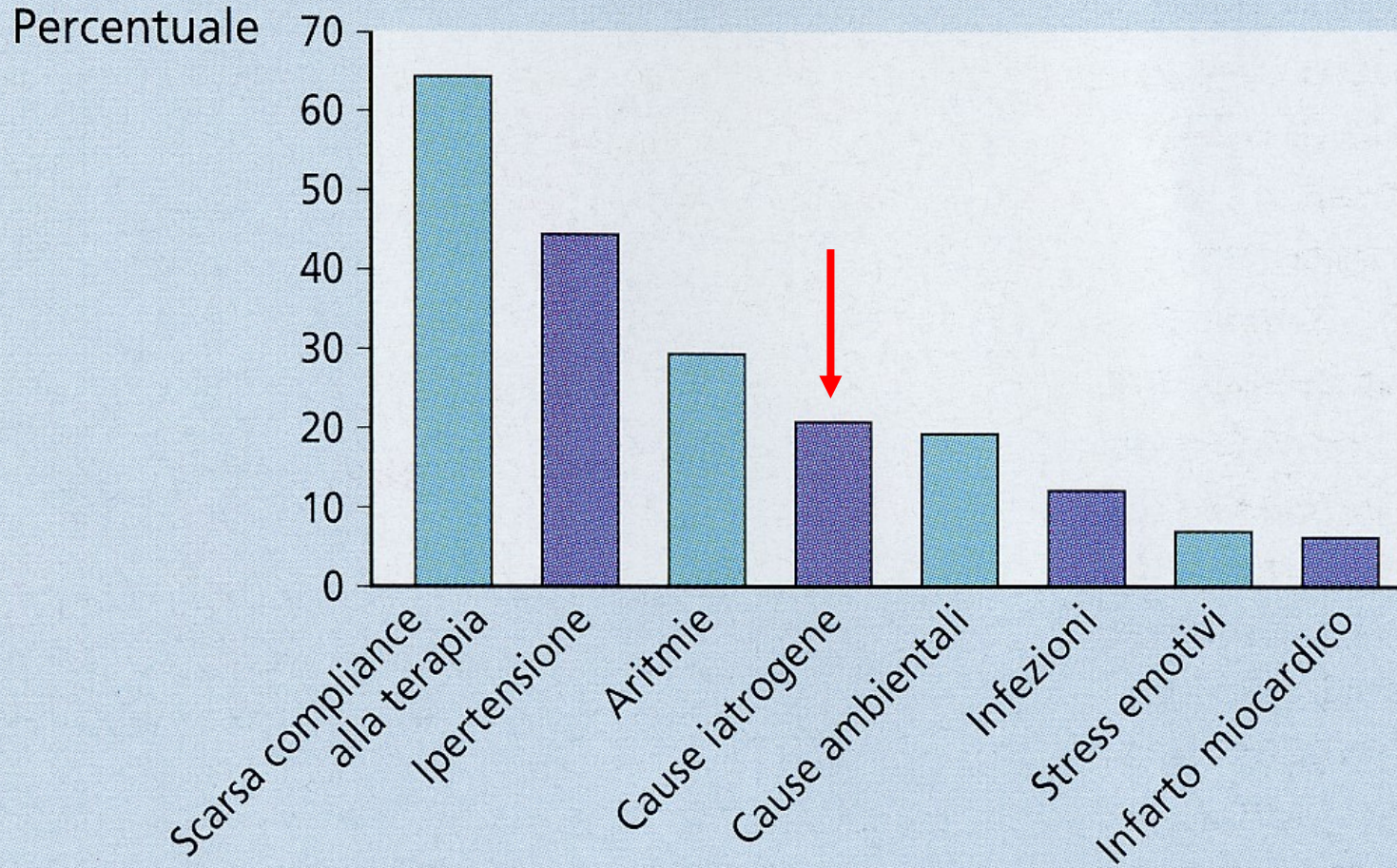
Da una “lettura“ di Stefano Urbinati

Tutti i pazienti con scompenso cardiaco a F.E. ridotta si assomigliano.


Ogni paziente con scompenso cardiaco a F.E. conservata è scompensato a modo suo.

L. Tolstoj: Anna Karenina

FATTORI CHE PROVOCANO UN'ESACERBAZIONE ACUTA DELL'INSUFFICIENZA CARDIACA



Associations of Benzodiazepine With Adverse Prognosis in Heart Failure Patients With Insomnia

Yu Sato, MD; Akiomi Yoshihisa , MD, PhD; Yu Hotsuki, MD; Koichiro Watanabe, MD; Yusuke Kimishima, MD; Takatoyo Kiko, MD; Yuki Kanno, MD, PhD; Tetsuro Yokokawa, MD, PhD; Satoshi Abe, MD; Tomofumi Misaka, MD, PhD; Takamasa Sato, MD, PhD; Masayoshi Oikawa, MD, PhD; Atsushi Kobayashi, MD, PhD; Takayoshi Yamaki, MD, PhD; Hiroyuki Kuni, MD, PhD; Kazuhiko Nakazato, MD, PhD; Takafumi Ishida, MD, PhD; Yasuchika Takeishi, MD, PhD

BACKGROUND: The prognostic impact of benzodiazepines has been unclear in patients with heart failure (HF).

METHODS AND RESULTS: This was a historical observational cohort study. A total of 826 patients who had been hospitalized for HF and were being treated for insomnia with either benzodiazepines or Z-drugs (zolpidem, zopiclone, or eszopiclone), were enrolled and divided on the basis of their hypnotics: benzodiazepine group ($n=488$ [59.1%]) and Z group ($n=338$ [40.9%]). We compared the patient characteristics and postdischarge prognosis between the groups. The primary end points were rehospitalization for HF and cardiac death. The benzodiazepine group was older (age, 72.0 versus 69.0 years; $P=0.010$), had a higher prevalence of depression (17.4% versus 8.9%; $P<0.001$), and showed a higher use of loop diuretics (77.9% versus 67.8%; $P=0.001$). In the laboratory data, the benzodiazepine group demonstrated lower levels of hemoglobin (12.3 versus 13.0 g/dL; $P=0.001$), sodium (139.0 versus 140.0 mEq/L; $P=0.018$), and albumin (3.7 versus 3.9 g/dL; $P=0.003$). Kaplan-Meier analysis showed that both end points were higher in the benzodiazepine group (rehospitalization for HF, log-rank $P=0.001$; cardiac death, log-rank $P=0.043$). Multiple Cox proportional hazard analysis revealed that the use of benzodiazepines was an independent predictor of rehospitalization for HF (hazard ratio, 1.530; 95% CI, 1.025–2.284; $P=0.038$). Furthermore, rehospitalization for HF was higher in the benzodiazepine group after propensity score matching (log-rank $P=0.036$).

CONCLUSIONS: Benzodiazepine is associated with higher risk of rehospitalization for HF compared with Z-drugs in patients with HF.

Le diverse ere fisiopatologiche dello scompenso cardiaco

Era emodinamica	Era idro-sodio retentiva	Era del postcarico	Era neuroumorale o neuroendocrina
<i>Farmaco: digitale</i>	<i>Farmaci: diuretici</i>	<i>Farmaci: vasodilatatori</i>	<i>Farmaci: ACE-inibitori e β-bloccanti</i>
<p>Da un primitivo modello centrale o monocratico dello scompenso si è successivamente passati a un modello periferico o polidistrettuale. La visione centralista è stata messa in crisi da osservazioni sul frequente squilibrio tra danno miocardico e quadro clinico. Come spiegare una capacità di esercizio normale in presenza di una disfunzione severa del VS. (esempio frazione di eiezione bassa) ?</p>	<p>La nuova teoria ha posto al centro la necessità di mantenere una pressione arteriosa adeguata. In prospettiva filogenetica nelle epoche ancestrali la natura non considerava l'insufficienza cardiaca come un evento nocivo per la sopravvivenza della specie, mentre privilegiava la capacità dell'organismo di reagire alle emorragie, alla fatica fisica violenta e improvvisa, alla mancanza di acqua e Sali. In queste circostanze per ripristinare rapidamente una portata sufficiente, veniva attivata una risposta neuroumorale pronta, con una aumentata secrezione di catecolamine e con l'attivazione del sistema renina-angiotensina, con possibilità di agire sulla pressione e sulla volemia.</p>	<p>Attivazione simpato-adrenergica:</p> <ul style="list-style-type: none"> • Resetting dei riflessi barocettivi • Iperattivazione simpatica • Modificazione del sistema β-recettoriale - Attivazione del sistema Renina-Angiotensina-Aldosterone - Aumento delle concentrazioni di ADH - Attivazione dei 4 peptidi natriuretici (ANP, BNP, CNP e DNP) - Meccanismi locali: EDRF (fattore endoteliale di rilascio); prostaglandine; endotelina 	

La “serendipity” degli inibitori del co-trasportatore sodio-glucosio di tipo 2: un nuovo paradigma nello scompenso cardiaco con frazione di eiezione ridotta

Giuseppe Rosano¹, Massimo Iacoviello², Pasquale Perrone Filardi³

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²*Dipartimento di Scienze Mediche e Chirurgiche, Università degli Studi, Foggia*

³*Dipartimento di Scienze Biomediche Avanzate, Università degli Studi di Napoli “Federico II”, Napoli*

Type 2 sodium-glucose co-transporter inhibitors (SGLT2i) are a new drug class with extremely relevant benefits in the prevention and treatment of heart failure (HF). In type 2 diabetic patients with both high cardiovascular risk and known cardiovascular disease, SGLT2i proved effective in reducing the risk of HF hospitalizations as well as the progression of renal disease. New evidence in patients with chronic HF and reduced ejection fraction (HFrEF) has also demonstrated their prognostic beneficial effects both in patients with and without type 2 diabetes mellitus. Based on these data, the use of this class of drugs in daily clinical practice is of primary importance to prevent HF hospitalization in diabetic patients and to improve the prognosis of HFrEF regardless of the presence of diabetes. In these patients, SGLT2i act synergistically with drugs capable of modulating the neurohormonal systems, thus allowing a further prognostic benefit.

S.C.: aspetti “concomitanti”



La carenza di ferro nello scompenso cardiaco: se la cerchi... la trovi

Giovambattista Desideri¹, Raffaella Bocale²

¹ Dipartimento di Medicina Clinica, Sanità Pubblica, Scienze della Vita e dell'Ambiente, Università degli Studi dell'Aquila

² Chirurgia Endocrina, Fondazione Policlinico Universitario "A. Gemelli" IRCCS, Roma

La presenza di un variabile grado di deficit di ferro, assai frequente nello scompenso cardiaco, può influenzare in modo rilevante la prognosi oltre che la qualità di vita dei pazienti. Il problema della carenza marziale resta spesso misconosciuto, soprattutto nel paziente anziano con polipatologie.

Beneficial effects of long-term intravenous iron therapy with ferric carboxymaltose in patients with symptomatic heart failure and iron deficiency.

Ponikowski P¹, van Veldhuisen DJ², Comin-Colet J³, Ertl G⁴, Komajda M⁵, Mareev V⁶, McDonagh T⁷, Parkhomenko A⁸, Tavazzi L⁹, Levesque V¹⁰, Mori C¹⁰, Roubert B¹⁰, Filippatos G¹¹, Ruschitzka F¹², Anker SD¹³; CONFIRM-HF Investigators.

CONCLUSION:

Treatment of symptomatic, iron-deficient HF patients with FCM over a 1-year period resulted in sustainable improvement in functional capacity, symptoms, and QoL and may be associated with risk reduction of hospitalization for worsening HF (ClinicalTrials.gov number NCT01453608).

Eu Heart J 2015; 36:657-668

Hyperkalemia in heart failure

Kiran Sidhu, Rohan Sanjanwala, and Shelley Zieroth

Purpose of review

Hyperkalemia is increasingly prevalent in the heart failure population as more people live with heart failure and comorbid conditions such as diabetes and chronic kidney disease. Furthermore, renin–angiotensin–aldosterone (RAAS) inhibitors are a key component of clinical therapy in these populations. Until now, we have not had any reliable or tolerable therapies for treatment of hyperkalemia resulting in inability to implement or achieve target doses of RAAS inhibition. This review will focus on two new therapies for hyperkalemia: patiromer and sodium zirconium cyclosilicate (SZC).

Recent findings

Patiromer and SZC have been studied in heart failure and both agents have demonstrated the ability to maintain normokalemia for extended periods of time with improved side effect profiles than existing potassium binders such as sodium polystyrene sulfate, though no direct comparisons have occurred. SZC has also shown promise in the treatment of acute hyperkalemia with its quick onset of action.

Summary

Patiromer and SZC will be useful adjuncts in the clinical care of heart failure patients with hyperkalemia. These agents will allow clinicians to maintain patients on RAAS inhibitors and uptitrate their guideline directed medical therapy to target doses without the additional concern for recurrent hyperkalemia and its untoward effects.

Compiti del medico di medicina generale nei confronti del paziente con scompenso cardiaco.

Paziente dimesso dal PS/DEA

- Acquisizione delle informazioni fornite alla dimissione
- Rivalutazione della terapia prescritta in dimissione e delle terapie concomitanti
- Intervento educativo rivolto al paziente, al caregiver e alla famiglia

Paziente in follow-up

- Rinforzo periodico dell'intervento educativo
- Monitoraggio e prevenzione delle riacutizzazioni
- Monitoraggio della terapia di fondo dello scompenso con attenzione costante alle comorbidità
- Integrazione con le altre figure professionali (cardiologo territoriale, Ambulatorio Scompenso, infermiere delle cure primarie)
- Gestione del paziente complesso e/o fragile
- Partecipazione alla gestione delle cure palliative

PS/DEA, Pronto Soccorso/Dipartimento di Emergenza-Accettazione.

Quando inviare in Pronto Soccorso/Dipartimento di Emergenza-Accettazione.

Riacutizzazione severa

- Dispnea non responsiva alla terapia diuretica
- Edema polmonare acuto
- Segni di congestione grave (ortopnea, epatomegalia, ascite, edemi)
- Oligo-anuria
- Ipotensione severa (pressione arteriosa sistolica <80-90 mmHg)

Cause non gestibili a domicilio

- Aritmie (tachicardia >120 b/min, polso aritmico, bradicardia <40 b/min)
- Ischemia miocardica
- Sospetto di embolia polmonare
- Gravi infezioni respiratorie

Inadeguata assistenza domiciliare

Heart failure treatment and the art of medical decision making

Finlay A. McAlister^{1,2*}, Justin A. Ezekowitz^{2,3}, and Paul W. Armstrong²

¹Division of General Internal Medicine, University of Alberta, Edmonton, Alberta, Canada; ²Faculty of Medicine and Dentistry, Canadian VIGOUR Centre, University of Alberta, Edmonton, Alberta, Canada; and ³Division of Cardiology, Mazankowski Alberta Heart Institute, University of Alberta, Edmonton, Alberta, Canada

‘If it were not for the great variability among individuals, medicine might as well be a science, not an art’.

William Osler

**Ho fatto tanti errori
nella mia vita.
Questo ognuno di noi lo dice.
Quello che non sappiamo dire
è questo:
ho fatto tanti errori
nella vita degli altri.**

Franco Arminio