Predictors of Congestive Heart Failure Mortality in Elderly People From the General Population

The Cardiovascular Study in the Elderly (CASTEL)

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SUMMARY

Congestive heart failure (CHF) is highly prevalent in the elderly. The aim of this study was to identify the predictors of CHF mortality in patients over 65 years of age who were free of CHF at initial screening. A total of 3,282 elderly subjects were recruited in a population-based frame and 12-year events were recorded. Continuous items were divided into tertiles and for each tertile adjusted the relative risk (RR) with 95% confidence intervals (CI) was derived in both genders from multivariate Cox analysis of CHF mortality.

Age ≥ 72 years ([RR]: 2.24; 95% CI 1.56 – 3.24), male gender ([RR]: 1.4; 95% CI 1.02 – 1.76), clinical history of coronary artery disease ([RR]: 1.25; 95% CI 1.02 – 1.76), pulse pressure ≥ 79 mmHg ([RR]: 1.33; 95% CI 1.03 – 1.87), heart rate ≥ 81 bpm ([RR]: 1.32; 95% CI 1.10 – 1.96), atrial fibrillation ([RR]: 1.82; 95% CI 1.18 – 2.81), left ventricular hypertrophy ([RR]: 1.42; 95% CI 1.01 – 2.02), diabetes ([RR]: 1.35; 95% CI 1.02 – 1.78), vital capacity $\leq 81\%$ of the theoretical value ([RR]: 2.50; 95% CI 1.88 – 3.32), forced expiratory volume in 1 second $\leq 72\%$ of the theoretical value ([RR]: 2.02; 95% CI 1.55 – 2.72) and serum sodium level ≤ 139 mmol/L ([RR]: 1.95; 95% CI 1.44 – 2.63) predicted CHF mortality.

This model is able to identify elderly people at increased risk of death from CHF. (Int Heart J 2005; 46: 419-431)

Key words: Congestive heart failure, Mortality, Epidemiology, Relative risk, Population, Elderly

IN recent decades, congestive heart failure (CHF) has gradually become one of the most prevalent cardiovascular disorders in Western societies, particularly in the elderly.¹⁾ More than 75% of patients with CHF are older than 65 years²⁾ and in this age class CHF is one of the most important causes of death and chronic disability.³⁾ This is due to the progressive ageing of the population,⁴⁾ as well as to the decrease in mortality from acute coronary events.⁵⁾ It is, therefore, not surprising

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that in the last two decades many papers have been devoted to the epidemiology, physiopathology, and etiology of CHF.^{3,4,6)}

The most important data concerning the impact of CHF on public health derive from pharmacological trials,⁷⁾ hospital records,⁸⁾ and general practice studies,⁹⁾ and involve mainly subjects aged less than 65 years. On the contrary, only a limited number of studies have included elderly people from the general population.^{6,10-12)} This is particularly true in Europe, where only 1 population-based study on CHF is available.¹³⁾

As a consequence, the independent predictors of CHF in this age class are not well defined.

In this paper, the parameters able to predict CHF mortality have been identified in 3,282 subjects aged over 65 years of age from North-East Italy, taking part in the CARDiovascular STudy in the ELderly (CASTEL), a prospective population-based study aimed at identifying the cardiovascular risk factors and, more in general, the predictors of mortality in elderly people. The 12-year results are described herein.

METHODS

General protocol: The CASTEL enrolled 3,282 subjects (1,281 men and 2,001 women) aged 65 years or over, representing 73% of the elderly subjects from the Northern Italian towns of Castelfranco and Chioggia (Veneto region). An initial screening and a follow-up including mortality over a period of 12 years were scheduled.¹⁴⁾

During the initial screening as well as at the end of follow-up, heart rate and sphygmomanometric supine blood pressure were measured in triplicate at 15-min intervals. This last procedure was repeated 3 times at 1-month intervals and the average of the last 2 of the 9 measurements was taken into consideration. Pulse pressure was the difference between systolic and diastolic blood pressure. Since 72% of the men and 75% of the women were on antihypertensive medication, "antihypertensive therapy" as a dichotomic covariate was included in the multivariate Cox analysis in order to adjust the results. Historical data were recorded by means of a questionnaire.

Body mass index (BMI, in kg/m²) was calculated as the weight/squared height ratio. All subjects were screened for albuminuria with a dipstick using a random morning urine sample. Subjects were classified into current (≥ 1 cigarette daily) smokers, never smoked, or ex-smokers; the latter had given up the smoking habit for at least one year prior to the screening. The subjects were divided into drinkers (≥ 50 mL daily) and nondrinkers according to their consumption of alco-

Table I. Cut-off Values and Diagnostic Criteria of Disease

Condition or disease	Cut-off and criteria
Arterial hypertension	Systolic blood pressure ≥ 140 or diastolic ≥ 90 mmHg or treated
Diabetes	Blood glucose ≥ 126 mg/dL or antidiabetic treatment
Hypercholesterolemia	Total cholesterol > 200 mg/dL
Hypertriglyceridemia	Triglycerides > 150 mg/dL
Left ventricular hypertrophy	Minnesota code 3.1 or 3.3
Atrial fibrillation	Minnesota code 8.3
Historical myocardial infarction	Minnesota code 1.1, 1.2 or 1.3 (being absent 6.4.1) or positive history in hospital records or akinesia/dyskinesia at time of echocardiogram or angiography
Historical angina pectoris	Typical chest pain with Minnesota code 4.1, 4.2, 5.1 or 5.2 (being absent 6.4.1, 7.1.1, 7.2.1, 7.4)
History of stroke	Cerebrovascular episode with permanent outcomes (motor or sensory loss, aphasia, dysarthria)
Overweight	BMI > 25 kg/m ² in both genders
Proteinuria	> 200 mg/L

hol. Forced expiratory volume in 1 second (FEV₁) and vital capacity (VC) were measured by a spirometer, and reference values individually calculated.¹⁵⁾

Electrocardiograms (ECG) were analysed on the basis of the Minnesota code.¹⁶⁾ Coronary artery disease (CAD) at entry was defined as a history of myocardial infarction and/or angina pectoris. Cut-off values for categorical items and diagnostic criteria of disease are summarised in Table I.

Definition of CHF: Subjects with a self-reported diagnosis of CHF underwent a careful clinical examination by a physician. According to the guidelines of the European Society of Cardiology,¹⁷⁾ subjects experiencing breathlessness, ankle swelling fatigue, raised venous pressure, and a third heart sound were considered to have CHF. A diagnosis of CHF by a physician was then followed by review of the participant's medical records. For the present paper, 329 subjects (116 men and 213 women, respectively) having a history of CHF at initial screening, were excluded from the analysis of data.

Mortality: Annual mortality was monitored by examining government records and the causes of death were verified by referring to hospital, retirement home, and/or physician records.

All records were coded according to ICD-9-CM by a specially trained research physician supervised by another more experienced colleague, to accurately determine the cause of death. If necessary, a third physician was contacted. No information about mortality was lost to follow-up. The codes for CHF were 398.91, 402.11, 402.91, 404.11, 404.13, 404.91, 404.93, 428.0-428.9.

Statistical analysis: Cumulative survival curves of CHF mortality were generated with the life-table method and compared with the Mantel-Haenzels approach. Analysis of variance was used for comparing groups, and Pearson's χ^2 test was used to compare the prevalence of categorical variables.

Continuous variables, expressed as mean $\pm$ standard deviation (SD), were divided into tertiles. For each tertile the relative risk (RR) with 95% confidence intervals (95% CI) adjusted for confounders was derived from logistic regression^{18,19} for CHF mortality. Stepwise Cox proportional hazard models with a *P* value of 0.05 were used to identify risk factors for CHF mortality in multivariate analysis excluding subjects having CHF at baseline. The models included as covariates demographic items (age, gender), historical disease (history of CAD, stroke or transient ischaemic attack and intermittent claudication, chronic obstructive pulmonary disease [COPD], hypertension, and diabetes), laboratory (blood lipids and proteinuria) and clinical examination measures (BMI, resting heart rate, murmurs at the neck), ECG abnormalities (atrial fibrillation and left ventricular hypertrophy [LVH]), and spirometry (VC and FEV₁). Statistics were analyzed using a BioMedical Data Processing (BMPD) package (Cork, Ireland) version 6.0.

The study lasted 12 years and was carried out in accordance with the Declaration of Helsinki of the World Medical Association and approved by the local Ethics Committee. The procedures followed were in accordance with institutional guidelines.

RESULTS

General characteristics: Mean age was 73.8 ± 5.3 years (range, 65 to 91) overall, 73.0 ± 4.8 in the 1195 men, and 74.0 ± 5.5 in the 1758 women ($P < 0.0001$). Men showed higher mean values of uric acid and serum creatinine, as well as a greater prevalence of proteinuria, than women (Table II). Meanwhile, age, heart rate, total cholesterol, high density lipoprotein cholesterol and low density lipoprotein cholesterol, VC, and FEV₁ as well as the prevalence of arterial hypertension were higher in the women than in the men.

Longitudinal study: Univariate analysis of outcome. A total of 3,282 subjects were enrolled at initial screening, and 329 of those having CHF were excluded from the analysis of data. At the end of the follow-up period, 1,372 subjects had died and 1,581 were alive (overall mortality rate, 46.4%, 56.9% in men and 42.4% in women; $P < 0.0001$). No information about mortality was lost to the follow-up.

In the CASTEL study, CHF mortality was more prevalent in men than in women (Figure 1).

Table II. Baseline Characteristics of the 2953 Elderly Subjects From the CASTEL Without CHF at Initial Screening Adjusted for Age and Gender

Characteristic	Men (n = 1195)	Women (n = 1758)	P-value between groups
Age (years)	73.0 ± 4.8	74.0 ± 5.5	< 0.002
Pulse pressure (mmHg)	69.2 ± 19.0	72.4 ± 19.5	< 0.001
Heart rate (bpm)	73.3 ± 11.4	77.5 ± 11.3	< 0.002
Uric acid (mmol/L)	0.33 ± 0.08	0.29 ± 0.08	< 0.002
Creatinine (mmol/L)	88.4 ± 0.3	79.5 ± 0.4	< 0.002
Total cholesterol (mmol/L)	5.4 ± 1.1	5.9 ± 1.1	< 0.0001
HDL-cholesterol (mmol/L)	1.4 ± 0.4	1.6 ± 0.4	< 0.0001
LDL-cholesterol (mmol/L)	3.3 ± 0.97	3.6 ± 1.0	< 0.002
Triglycerides (mmol/L)	1.6 ± 0.9	1.7 ± 0.9	< 0.0001
VC (% of theoretical)	90.3 ± 21.5	94.2 ± 21.0	< 0.002
FEV ₁ (% of theoretical)	80.5 ± 23.7	85.3 ± 25.9	< 0.0001
Hypertension (%)	51.7	58.2	< 0.0001
Diabetes (%)	6.8	8.8	< 0.0001
Proteinuria (%)	12.1	9.5	

BMI, systolic and diastolic blood pressure, serum sodium, smoking habits and alcohol consumption, historical CAD, stroke or transient ischemic attack, atrial fibrillation, and LVH were also tested but there were no significant differences between the groups.

HDL = high-density lipoprotein; LDL = low-density lipoprotein; VC = vital capacity; FEV₁ = forced expiratory volume in 1 second.

Figure. Kaplan-Meier survival curves for fatal congestive heart failure in 1,758 women (—) and 1,195 men (····) from the CASTEL.

Multivariate analysis for CHF mortality. In the whole population, 12 parameters (age, male gender, historical CAD, atrial fibrillation, FEV₁ and VC, LVH, pulse pressure, diabetes, heart rate, and serum sodium level) turned out to be multivariate independent predictors of CHF mortality (Table III).

Table III. Results of the Multivariate Cox Equation for Cardiac Heart Failure Mortality in the CASTEL

	χ^2 enter	χ^2 remove	Improvement χ^2	P-value
Age	55.45	18.54	45.78	< 0.0001
Historical CAD	50.43	29.32	35.45	< 0.01
Vital capacity	49.01	27.81	31.42	< 0.0001
FEV ₁	46.61	18.22	22.88	< 0.0001
LVH	3.97	3.78	7.69	< 0.01
Male gender	8.85	2.49	6.83	< 0.01
Atrial fibrillation	15.64	6.47	6.33	< 0.001
Diabetes	9.80	3.69	5.67	< 0.01
Pulse pressure	7.10	3.03	5.12	< 0.04
Serum sodium	9.26	3.76	5.02	< 0.0001
Heart rate	9.65	5.27	4.87	< 0.04
Overweight	1.03	-	-	-
Antihypertensive therapy	1.36	-	-	-
Uric acid	7.10	-	-	-
Hypercholesterolemia	6.77	-	-	-
Hypertriglyceridemia	4.67	-	-	-
Historical stroke	8.03	-	-	-
Proteinuria	4.64	-	-	-
Cigarette smoking	1.19	-	-	-

CAD = coronary artery disease; FEV₁ = forced expiratory volume in 1 second;
LVH = left ventricular hypertrophy.

Age was the main risk factor of CHF mortality; the risk of dying of CHF progressively increased from the first to the second ([RR] 2.24; 95% CI, 1.56 to 3.24) to the third tertile of age ([RR] 4.63; 95% CI, 3.27 to 6.56).

Men were at a disadvantage compared to women ([RR]: 1.4; 95% CI, 1.02 to 1.76) until 75 years of age. In the last age class (≥ 76 years), CHF mortality did not differ between the two genders (14.1 versus 14.5%, NS).

Historical CAD ([RR]: 1.25; 95% CI, 1.02 to 1.76) resulted to be an independent risk factors for CHF mortality both in men and women (insignificant difference between genders).

CHF mortality was comparable in normotensive and hypertensive subjects (10.2 versus 9.2%, respectively, NS). Nevertheless, CHF mortality progressively increased from the first to the last tertile of pulse pressure (8.0, 8.6, and 10.6%, respectively; $P < 0.04$ versus the last versus first). Based on multivariate analysis, pulse pressure was found to be an independent predictor of mortality, with the RR sharply increasing when pulse pressure was ≥ 79 mmHg (Table IV). CHF mortality showed no relation with either systolic or diastolic blood pressure.

Table IV. Relative Risk (RR) of CHF Mortality in the CASTEL

Parameter	Mortality	RR	95% CI
Age (years)			
1 st tertile (65-71)	4.2 (%)	1	<i>Reference</i>
2 nd tertile (72-75)	8.6 (%)	2.24	1.56 – 3.24†
3 rd tertile (≥ 76)	14.4 (%)	4.63	3.27 – 6.56*
Gender			
Male	9.6 (%)	1.4	1.09 – 1.80‡
Female	8.5 (%)	1	<i>Reference</i>
History of coronary artery disease			
No	8.5 (%)	1	<i>Reference</i>
Yes	13.1 (%)	1.25	1.02 – 1.76‡
Pulse pressure (mmHg)			
1 st tertile (≤ 60)	8 (%)	1	<i>Reference</i>
2 nd tertile (61-78)	8.6 (%)	1.03	0.76 – 1.40
3 rd tertile (≥ 79)	10.6 (%)	1.33	1.03 – 1.87‡
Heart rate (bpm)			
1 st tertile (≤ 71)	8 (%)	1	<i>Reference</i>
2 nd tertile (72-80)	8.1 (%)	1.02	0.75 – 1.39
3 rd tertile (≥ 81)	11.2 (%)	1.32	1.10 – 1.96‡
Vital capacity (% of theoretical value)			
1 st tertile (≤ 81)	15.7 (%)	2.50	1.88 – 3.32*
2 nd tertile (82-100)	8.4 (%)	1.45	0.88 – 1.63
3 rd tertile (> 100)	5.5 (%)	1	<i>Reference</i>
FEV ₁ (% of theoretical value)			
1 st tertile (≤ 72)	14.6 (%)	2.02	1.55 – 2.72*
2 nd tertile (73-98)	7.9 (%)	1.33	0.92 – 1.57
3 rd tertile (> 98)	6.8 (%)	1	<i>Reference</i>
Atrial fibrillation			
No	8.7 (%)	1	<i>Reference</i>
Yes	16.6 (%)	1.82	1.18 – 2.81†
Left ventricular hypertrophy			
No	8.7 (%)	1	<i>Reference</i>
Yes	11.7 (%)	1.42	1.01 – 2.02‡
Diabetes			
No	8.4 (%)	1	<i>Reference</i>
Yes	11.4 (%)	1.35	1.02 – 1.78†
Sodium (mmol/L)			
1 st tertile (130-139)	11.4 (%)	1.95	1.44 – 2.63*
2 nd tertile (140-144)	8.4 (%)	1.34	0.98 – 1.83
3 rd tertile (≥ 145)	7 (%)	1	<i>Reference</i>

FEV₁ = forced expiratory volume in 1 second; CI = confidence interval. * $P < 0.00001$; † $P < 0.005$; ‡ $P < 0.04$.

CHF mortality progressively increased from the first to last tertile of heart rate (8%, 8.1%, 11.2%, respectively), becoming significant ($P < 0.04$) only at the 3rd tertile versus the 1st tertile. In multivariate analysis, heart rate independently predicted CHF mortality; the RR sharply increased to 1.32 (95% CI, 1.10 to 1.96) when it was ≥ 81 bpm.

Atrial fibrillation was diagnosed in 102 subjects at the initial screening (3.5%). CHF mortality was higher in subjects with atrial fibrillation than in those without (16.6% versus 8.7%, $P < 0.001$). In multivariate analysis, atrial fibrillation strongly predicted CHF mortality with a χ^2 of 6.4 ($P < 0.01$) and the RR of having atrial fibrillation was 1.82 (95% CI, 1.18 to 2.81).

Subjects with LVH had higher CHF mortality than those without (11.7% versus 8.7%, [RR]: 1.42; 95% CI, 1.01 to 2.02, $P < 0.04$).

CHF mortality was significantly ($P < 0.05$) higher in diabetic patients (11.4%) than in normoglycaemic subjects (8.4%, [RR]: 1.35; 95% CI, 1.02 to 1.78).

A VC $\leq 81\%$ and FEV₁ $\leq 72\%$ significantly increased CHF mortality ([RR]: 2.50; 95% CI, 1.88 to 3.32, and 2.02; 95% CI, 1.55 to 2.72, respectively).

Finally, the risk of CHF mortality after adjustment for confounding factors was significantly higher when the serum sodium level was ≤ 139 mmol/L ([RR]: 1.95; 95% CI, 1.44 to 2.63).

DISCUSSION

Although CHF is highly prevalent in the elderly, few studies have evaluated the predictors of CHF mortality in elderly subjects at a population level. Indeed, the main findings concerning CHF in this age class derive from studies conducted in the United States; the Framingham study,⁶ the Cardiovascular Health study,¹² the New Haven Connecticut cohort of the Established Population for Epidemiological Studies of the Elderly program,¹³ and the experiences of Cowie, *et al*¹⁰ and Senni, *et al*²⁰ have made the greatest contribution to this topic.

In Europe, the data on CHF in the elderly derive from the Rotterdam Study¹³ and in part from the non-population-based experience that Cowie, *et al*²¹ reported among general practitioners of West London. As a consequence, in Europe the independent predictors of CHF in this age class are not well defined.

In the CASTEL, apart from age, 10 main predictors of CHF mortality were identified, namely male gender, historical CAD, atrial fibrillation, reduced VC or FEV₁, LVH, pulse pressure, diabetes, heart rate, and serum sodium.

The CASTEL supports the belief by Gottdiener¹² and Chen¹¹ that the incidence of CHF is associated with advanced age and male gender. Nevertheless, in our experience elderly men had a CHF mortality significantly greater than women

only until the age of 75 years, after which there was no gender-related difference in CHF mortality. This confirms the observation that with advancing age the cardiovascular risk pattern of women becomes similar to that of men.²²⁾

The role of historical CAD was another important predictor of CHF mortality. There is convincing evidence that prolonged episodes of myocardial ischemia lead to impairment of left ventricular function and ultimately to CHF.²³⁾ On the other hand, it has recently been demonstrated that myocardial blood flow reserve is impaired in subjects with CHF.²⁴⁾ Preventing the progression of CHF can be difficult as multiple risk factors contribute to the decline of cardiac function. However, as CAD is the most common risk factor for CHF,²⁵⁾ preventing CAD may lead to the prevention of CHF progression.

In the last two decades, interest in the prognostic role of pulse pressure (PP) has increased tremendously. Many epidemiological studies have demonstrated that PP is a major predictor of cardiovascular risk in the general population,^{26,27)} particularly in the elderly.^{28,29)} The increase in PP is an age-related phenomenon²⁸⁾ and an indicator of large artery stiffness²⁹⁾; this last condition increases afterload³⁰⁾ and myocardial oxygen demand, impairs ventricular relaxation, and causes subendocardial ischemia even in the absence of CAD.³¹⁾ These effects make elderly people more prone to myocardial ischemia and ventricular dysfunction (ie. to CHF).³²⁾ Nevertheless, in our experience and according to Chen, *et al*,¹¹⁾ the association between high PP and CHF was independent of the presence of CAD, suggesting that the pressor effects on ventricular dysfunction are mediated by other mechanisms. Further investigations will be necessary to clarify this hypothesis. It is worthy of note that CHF mortality did not depend on the level of hypertension. The categorisation into normotensive and hypertensive, certainly appropriate for middle-aged adults, is therefore not appropriate for the elderly.

The activation of the adrenergic system represents the main regulatory mechanism occurring during the development of left ventricular dysfunction. Furthermore, increased production of catecholamines in patients with CHF determines in the long run both direct damage to cardiomyocytes³³⁾ and a negative feedback on the cardiovascular system, due to activation of the renin-angiotensin system. In the CASTEL, male subjects with a heart rate ≥ 81 bpm were found to be at increased risk of CHF mortality, while this was not the case for women.

Atrial fibrillation (AF) is the most common arrhythmia observed in clinical practice. Although it can occur at any age, its frequency increases with increasing age.³⁴⁾ CHF and AF are often present in the same patient, and interact with each other leading to a vicious circle. In fact, CHF may cause AF via electromechanical feedback and adrenergic activation, while AF may promote CHF by increasing heart rate.³⁵⁾ Our data demonstrate the role of AF as an independent risk factor

for CHF mortality, and underline the clinical value of the simple procedure represented by taking radial pulse in elderly subjects.

LVH was found to be independently associated with an increased risk of CHF mortality even after adjustment for confounders. This is in keeping with the results of other studies.^{36,37)} Myocardial hypertrophy provides an effective short-term compensation in subjects with CHF. However, in the long term, changes occur in the geometry of the myocardium, leading to left ventricular remodelling. This is accompanied by myocyte abnormalities³⁸⁾ leading to ventricular arrhythmias and sudden death.

There is clear evidence that diabetes mellitus increases the prevalence and severity of CHF and worsens the prognosis of patients with CHF, especially of those with coexisting CAD.³⁹⁾ This is particularly true for elderly patients.⁴⁰⁾ When CHF mortality was analysed in the CASTEL, the adjusted RR of diabetes was 1.35. On the other hand, CHF is accompanied by insulin-resistance, which in turn represents the main risk factor for the development of diabetes.⁴¹⁾ In the CASTEL, the prevalence of diabetes was in fact higher among subjects with a history of CHF than in those without.

Chronic obstructive pulmonary disease (COPD) is very common in the elderly, and represents the fifth most common cause of death in the world.⁴²⁾ FEV₁ and VC are the tests most widely used for the diagnosis of impaired pulmonary function, and are considered independent risk indicators for cardiovascular diseases.⁴³⁾ In our study the inverse relationship between FEV₁ or VC and CHF mortality persisted after adjustment for age; according to Kannel, *et al*⁴⁴⁾ reduced pulmonary function therefore independently predicts CHF death. The mechanism of this association is not well established. Conversely, it has been found that pulmonary congestion produced by left ventricular failure causes both obstructive and restrictive alterations of pulmonary function leading to COPD.⁴⁵⁾ Whatever the nature of the association between COPD and CHF mortality may be, ventilatory tests should always be taken into consideration when dealing with elderly people, to allow early identification of the subjects at greatest risk of CHF mortality.

Hyponatremia (serum sodium ≤ 139 mmol/L) was one of the strongest predictors of CHF mortality. Madsen, *et al*⁴⁶⁾ established a threshold of 137 mmol/L for increased risk of sudden death in subjects with CHF. Hyponatremia occurs as a consequence of the activation of the renin-angiotensin-aldosterone system.⁴⁷⁾ This does not explain why hyponatremia independently predicted CHF mortality at a population level; one can only speculate that it is the expression of a subclinical form of CHF (water retention).

In conclusion, the results of this epidemiological survey - the first one in Italy to have provided data about CHF mortality in elderly people from the gen-

eral population - indicate that CHF represents an important risk factor, or better, the most frequent CAD-associated cause of death. In summary, we found that CHF mortality in elderly people from the general population is mainly attributed to classic risk factors, but additional parameters (e.g. PP, heart rate, VC, FEV₁, and serum sodium) have been identified in the CASTEL as independent risk factors of CHF death.

Outlining the *identikit* of elderly patients at increased risk for CHF will provide a reference model for the early detection of elderly subjects prone to die due to this syndrome.

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