Left bundle-branch block is associated with increased 1-year sudden and total mortality rate in 5517 outpatients with congestive heart failure: A report from the Italian Network on Congestive Heart Failure

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Background A deleterious effect of complete left bundle-branch block (LBBB) on left ventricular function has been established. Nevertheless, the independent effect of a widened QRS on mortality rate in congestive heart failure (CHF) is still controversial. Therefore, we carried out this analysis to determine whether LBBB is an independent predictor of mortality in CHF.

Methods and Results We analyzed the large Italian Network on CHF Registry of unselected outpatients with CHF of different causes. The registry was established by the Italian Association of Hospital Cardiologists in 1995. Complete 1-year follow-up data were available for 5517 patients. The main underlying cardiac diagnosis was ischemic heart disease in 2512 patients (45.6%), dilated cardiomyopathy in 1988 patients (36.0%), and hypertensive heart disease in 714 patients (12.9%). Other causes were recorded in the remaining 303 cases (5.5%). LBBB was present in 1391 patients (25.2%) and was associated with an increased 1-year mortality rate from any cause (hazard ratio, 1.70; 95% confidence interval, 1.41 to 2.05) and sudden death (hazard ratio, 1.58; 95% confidence interval, 1.21 to 2.06). Multivariate analysis showed that such an increased risk was still significant after adjusting for age, underlying cardiac disease, indicators of CHF severity, and prescription of angiotensin-converting enzyme inhibitors and β-blockers.

Conclusion LBBB is an unfavorable prognostic marker in patients with CHF. The negative effect is independent of age, CHF severity, and drug prescriptions. These data may support the rationale of randomized trials to verify the effects on mortality rate of ventricular resynchronization with multisite cardiac pacing in patients with CHF and LBBB. (Am Heart J 2002;143:398-405.)
of left bundle-branch block (LBBB) in CHF are more conflicting. In fact, the deleterious effect of LBBB on left ventricular systolic and diastolic function has been established in subjects without overt heart disease and in patients with dilated cardiomyopathy, and whether mortality rate is increased independently with a widening of QRS is still controversial.

Therefore, we carried out this analysis of a database of unselected outpatients with CHF of different causes, who were followed by a large number of cardiology centers in Italy. The purpose was evaluation of the prevalence of complete LBBB and testing of whether complete LBBB is an independent predictor of all-cause mortality and of sudden death in patients with CHF.

Methods
Study design, collected data, and definitions

Data for this analysis were originated from the database of the Italian Network CHF Registry, a survey designed in 1995 by an ad hoc committee of the Italian Association of Hospital Cardiologists (Florence, Italy). One-hundred fifty cardiology centers accepted participation in the study. Centers were distributed across the national territory and were more frequently located in Northern (46%) than in Central (24%) or Southern (30%) Italy, well representing the whole country.

Short training sessions were organized to prepare clinicians to collect and enter data with standardized methods. With an ad hoc designed software, patient data were recorded at each center by trained cardiologists and then were pooled into a single database at the Italian Association of Hospital Cardiologists Research Center. Entry into the database required that the patient had a diagnosis of New York Heart Association (NYHA) classification I to IV CHF on the basis of the guidelines of the European Society of Cardiology. Demographic, clinical, instrumental, and laboratory variables and information on drug therapy were collected for each patient. At baseline, a 12-lead electrocardiogram was recorded and coded by a single cardiologist at each participating center, with a standardized format outlined in the database. In particular, presence of LBBB was recorded and QRS duration was coded as less than 120 ms, 120 to 140 ms, or more than 140 ms. This information was used by the computer program to control for the acceptability of the diagnosis of LBBB and to distinguish between incomplete (QRS duration, 120 to 140 ms) and complete (QRS duration, >140 ms) block. Patients were followed according to the routine clinical practice of the participating centers. In this context, patients underwent standard chest x-ray, 24-hour Holter electrocardiogram monitoring, 2-dimensional echocardiography, and blood sampling for the most common laboratory tests (eg, creatinine level, electrolyte level, etc) when the attending cardiologists deemed them necessary. Cardiologists at the participating centers were responsible for defining the cause of CHF and the NYHA classification, noting whether a 3rd heart sound was audible and computing the cardiothoracic ratio. When an echocardiographic examination was performed, calculation of left ventricular ejection fraction from 4-chamber apical echocardiographic view was also done. Ventricular tachycardia was defined as an episode of tachycardia with widened QRS that lasted longer than 3 beats with a heart rate of more than 100 beats/min, as revealed with 24-hour Holter electrocardiogram monitoring. Renal dysfunction was diagnosed for serum creatinine level of more than 2.5 mg/dL. Previous hospitalizations for CHF in the last year were also recorded. After the baseline visit, the patients were followed. In the case of out-of-hospital death, the event was confirmed with telephone interview of patient’s relatives, with a standard questionnaire aimed at determining the mode of death (sudden versus non sudden).

Study population

As of January 2000, 6593 patients had complete 1-year follow-up information. We excluded from this analysis 1076 patients for any of the following reasons: CHF as the result of primary valvular heart disease (n = 745), inadequate quality of electrocardiogram (n = 270), and cardiac transplantation within the 1st year of follow-up examination (n = 61). Thus, the study population for this analysis consisted of 5517 patients.

Statistical analysis

The data were analyzed with the SAS statistical package and are presented as mean ± standard deviation. The univariate association of complete LBBB with several demographic and clinical characteristics and with 1-year mortality rate was analyzed with the χ² test. Cox proportional hazards multivariate models with calculation of the adjusted hazard ratio and 95% confidence interval were used to identify the independent determinants of all-cause mortality rate and of mortality rate as the result of sudden death. A 2-tailed P value of less than .05 was considered statistically significant.

Results

The study population of 5517 patients had a mean age of 63 ± 12 years (range, 14 to 96 years) and included 1295 women (23.5%) and 1544 cases (28.0%) that were classified in NYHA class III to IV. The 1076 patients with complete 1-year follow-up data who were excluded from this analysis had a similar age (65 ± 12 years; range, 17 to 91 years) but a larger prevalence of women (38.6%; P < .01) and of NYHA classification III to IV (38.8%; P < .01).

Complete left bundle-branch block: prevalence and associated clinical characteristics

Complete LBBB was diagnosed in 1391 of 5517 patients (25.2%), complete right bundle-branch block was diagnosed in 336 of 5517 patients (6.1%), and other forms of intraventricular delay were diagnosed in 339 of 5517 patients (6.1%). Demographic and clinical characteristics of patients with LBBB are reported in Table I. The prevalence of patients older than 70 years was similar in the group with and without LBBB, and female patients were significantly more represented in the group with LBBB. Cause of CHF was different between the 2 groups, with dilated cardiomyopathy...
and ischemic heart disease being the most common diagnosis in patients with and without LBBB, respectively. Although previous hospitalizations for CHF during the last year had a similar prevalence in both groups, there were several indicators of greater severity of clinical status in patients with LBBB as compared with those without LBBB. In particular, LBBB was associated with higher prevalence of NYHA classification III to IV CHF, reduced systolic blood pressure, 3rd heart sound, and abnormally increased cardiothoracic ratio (>0.55), while the prevalence of a heart rate higher than 100 beats/min was similar in the 2 groups. Echocardiographic results were available and of adequate quality to permit a reliable measurement of left ventricular ejection fraction in 3392 of 5517 patients (61.5%), a proportion that was similar in patients with and without LBBB (2523 of 4126 [61.1%] versus 869 of 1391 [62.5%]; P = not significant). Echocardiographic results also indicated a greater disease severity in patients with LBBB, who had a higher prevalence rate of severely reduced left ventricular ejection fraction (<30%). Conversely, the prevalence rate of chronic atrial fibrillation was higher in the group without LBBB, and ventricular tachycardia and renal failure did not differ between the 2 groups. Significantly higher proportions of patients with LBBB were undergoing treatment with diuretics, angiotensin-converting enzyme (ACE) inhibitors, digoxin, and amiodarone, while nitrates, antiplatelet agents, β-blockers, and Ca-antagonists were prescribed more frequently to patients without LBBB.

### Table I. Main demographic and clinical characteristics of 5517 patients with congestive heart failure and presence or absence of associated complete left bundle-branch block

<table>
<thead>
<tr>
<th>Variable</th>
<th>Present (n = 1391)</th>
<th>Absent (n = 4126)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 70 years</td>
<td>30.0%</td>
<td>31.7%</td>
<td>NS</td>
</tr>
<tr>
<td>Female gender</td>
<td>29.3%</td>
<td>21.5%</td>
<td>.001</td>
</tr>
<tr>
<td>CHF cause</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>49.3%</td>
<td>31.6%</td>
<td></td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>33.7%</td>
<td>49.5%</td>
<td>.001</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>12.7%</td>
<td>13.0%</td>
<td></td>
</tr>
<tr>
<td>Other causes</td>
<td>4.2%</td>
<td>5.9%</td>
<td></td>
</tr>
<tr>
<td>Previous hospitalization for CHF</td>
<td>56.4%</td>
<td>54.3%</td>
<td>NS</td>
</tr>
<tr>
<td>NYHA classification III to IV</td>
<td>32.8%</td>
<td>26.4%</td>
<td>.001</td>
</tr>
<tr>
<td>Heart rate ≥ 100 beats/min</td>
<td>10.9%</td>
<td>10.7%</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;100 mm Hg</td>
<td>3.9%</td>
<td>2.7%</td>
<td></td>
</tr>
<tr>
<td>100 to 130 mm Hg</td>
<td>61.2%</td>
<td>54.2%</td>
<td>.001</td>
</tr>
<tr>
<td>&gt;130 mm Hg</td>
<td>34.9%</td>
<td>43.1%</td>
<td></td>
</tr>
<tr>
<td>3rd heart sound</td>
<td>34.2%</td>
<td>22.2%</td>
<td>.001</td>
</tr>
<tr>
<td>CT ratio &gt; 0.55</td>
<td>63.2%</td>
<td>55.0%</td>
<td>.040</td>
</tr>
<tr>
<td>LVEF</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.30</td>
<td>49.2%</td>
<td>30.4%</td>
<td></td>
</tr>
<tr>
<td>0.30 to 0.40</td>
<td>40.3%</td>
<td>44.5%</td>
<td>.001</td>
</tr>
<tr>
<td>&gt;0.40</td>
<td>10.5%</td>
<td>25.1%</td>
<td></td>
</tr>
<tr>
<td>Chronic AF</td>
<td>13.3%</td>
<td>19.3%</td>
<td>.001</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>28.5%</td>
<td>28.8%</td>
<td>NS</td>
</tr>
<tr>
<td>Renal failure</td>
<td>2.1%</td>
<td>2.5%</td>
<td></td>
</tr>
<tr>
<td>Drug prescriptions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretics</td>
<td>88.6%</td>
<td>82.0%</td>
<td>.001</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>87.4%</td>
<td>82.8%</td>
<td>.001</td>
</tr>
<tr>
<td>Digoxin</td>
<td>71.8%</td>
<td>62.8%</td>
<td>.001</td>
</tr>
<tr>
<td>Nitrates</td>
<td>38.7%</td>
<td>43.2%</td>
<td>.003</td>
</tr>
<tr>
<td>Antiplatelet agents</td>
<td>31.4%</td>
<td>38.5%</td>
<td>.001</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>24.7%</td>
<td>20.0%</td>
<td>.001</td>
</tr>
<tr>
<td>β-blockers</td>
<td>16.2%</td>
<td>19.0%</td>
<td>.017</td>
</tr>
<tr>
<td>Ca-antagonists</td>
<td>9.7%</td>
<td>14.3%</td>
<td>.001</td>
</tr>
<tr>
<td>Other antiarrhythmic agents</td>
<td>1.8%</td>
<td>2.2%</td>
<td>NS</td>
</tr>
</tbody>
</table>

LBBB, left bundle-branch block; CHF, congestive heart failure; NYHA, New York Heart Association; CT, cardiothoracic; LVEF, left ventricular ejection fraction; AF, atrial fibrillation; ACE, angiotensin-converting enzyme; NS, not significant.

### Figure 1

One-year mortality rate for all-cause death and sudden death in study population of 5517 patients with congestive heart failure (gray bars) and in subgroups with complete left bundle-branch block (black bars) or without complete left bundle-branch block (white bars). Unadjusted hazard ratio (HR) is also reported with 95% confidence interval.
sudden death at 1 year. In these models, we entered age, heart rate, and systolic blood pressure as continuous variables and all the other variables presented in Table I, as dichotomous variables, with inclusion of LBBB as a covariate. Of the variables pertaining to drug prescription, we included only those referring to agents with proven effects on mortality rate in randomized clinical trials. Thus, only prescriptions of ACE inhibitors, antiplatelet agents, and β-blockers were entered as further dichotomous covariates in these models, whose results are summarized in Tables II and III.

The risk of all-cause death at 1 year was significantly increased with increasing age and in the presence of ischemic heart disease, previous hospitalization for CHF, and several indicators of greater disease severity or comorbidity, such as NYHA classification III to IV, increased heart rate or reduced systolic blood pressure, 3rd heart sound, chronic atrial fibrillation, sustained ventricular tachycardia, and renal failure (Table II). Prescription of ACE inhibitors and β-blockers significantly and independently protective effects against the risk of all-cause death, and sex, the cardiothoracic ratio, left ventricular ejection fraction, and prescription of antiplatelet agents were not confirmed to be significant predictors of that risk (Table II). After adjustment for all these covariates, complete LBBB still maintained its unfavorable prognostic effect and, on average, it was estimated to increase the risk of all-cause death at 1 year by 36% (Table II). The same variables, with the further exclusion of heart rate, renal failure, and prescription of ACE inhibitors, were significantly associated with the risk of sudden death at 1 year (Table III). Also in this multivariate model, LBBB was independently associated with the risk of sudden death, which was increased by almost 35% in the presence of this intraventricular conduction defect.

### Discussion

The association of a wide QRS with increased mortality rate in CHF has been repeatedly investigated, but results have been conflicting. Although some studies showed that a wide QRS has an independent, unfavorable prognostic significance and increases the mortality rate of patients with CHF during periods of follow-up examination extended to 5 years,11,12,14 other studies adopting similar multivariate approaches did not confirm this finding.10,13,16 Such discrepancies may arise from the variable cutoffs adopted to define the conduction defect, ranging from a mild widening of the QRS complex above 120 ms12 to complete LBBB10,11,13,14,16 and from large differences in the covariates included in multivariate analyses. A further cause of conflicting results may be represented by the variable cause of CHF in the different studies that, in most cases, included only patients with dilated cardiomyopathy.12,14,16 With only 2 studies including a few patients with ischemic heart disease,10,11 however, all these studies included limited numbers of patients, ranging from 6216 to 441,13 and only recently a preliminary report of a large survey of 3654 patients confirmed a direct association of QRS duration with 1-year mortality rate in CHF from dilated cardiomyopathy.18

Thus, we decided to limit our analysis only to the prognostic significance of complete LBBB, with a data-
base that was produced by an Italian epidemiologic survey of outpatients referred to cardiology centers for evaluation and treatment of CHF. The registry was large enough to warrant adequate numbers for analysis.\textsuperscript{19} Our results from 5517 patients showed that complete LBBB develops in as many as 25% of patients with CHF of any origin and is associated with a 70% increase in the univariate risk of all-cause mortality rate at 1 year. At univariate analysis, complete LBBB was also associated with a series of CHF-specific indicators of disease severity, and the relevance of these findings was further supported by the observation that drugs that are commonly used in the routine control of CHF symptoms were prescribed more frequently to patients with LBBB than to those without LBBB. Conversely, patients without LBBB more frequently underwent treatment with anti-ischemic or antithrombotic agents, a finding consistent with the observation that ischemic heart disease was the most frequent cause of CHF in this subgroup.

Such complex relationships needed to be accounted for in multivariate models that included several clinical variables together with drug prescriptions. In fact, ACE inhibitors, which were more frequently prescribed to patients with LBBB, are well shown to be able to reduce mortality rate in CHF,\textsuperscript{21,22} and a similar result also can be obtained with antiplatelet agents\textsuperscript{23} and $\beta$-blockers,\textsuperscript{24,25} which were used more frequently in patients without LBBB. In this perspective, the strength of our results is increased by the observation that, even after adjusting for a large number of covariates that included the most relevant variables that describe CHF severity and prescriptions of these pharmacologic agents, LBBB retained its unfavorable, independent prognostic value, consisting of a 36% adjusted increase in all-cause mortality rate at 1 year. In the multivariate model predicting the risk of sudden death, the presence of LBBB maintained a quantitatively similar effect, with an increase in the risk of almost 35%. Consistent with previous findings from large randomized, controlled trials, our observational data showed a protective effect of ACE inhibitors\textsuperscript{21,22} and $\beta$-blockers\textsuperscript{24,25} against all-cause death. Prescription of antiplatelet agents was not associated with such a significant protective effect that, however, had been previously proven only in a subanalysis of the Studies of Left Ventricular Dysfunction trial.\textsuperscript{23}

In the largest of the previous studies that showed the unfavorable prognostic significance of intraventricular conduction defects in CHF, age, creatinine level, heart rate, and left ventricular ejection fraction were the other independent predictors of 1-year total mortality rate,\textsuperscript{18} and in our study, left ventricular ejection fraction was not confirmed to be an independent predictor of death after multivariate analysis. This difference might derive from several other variables that we took into account which might have overridden the variance attributable to left ventricular ejection fraction. Among these we included NYHA classification, which, even though in gross terms, may well reflect exercise capacity, which has been proven as an important prognostic factor in CHF.\textsuperscript{3,5,7}

Asynchrony of left ventricular contraction\textsuperscript{26} and impairment in the systolic and diastolic left ventricular function\textsuperscript{8,9} induced by complete LBBB have been clearly proven. In 1990, Hochleitner et al\textsuperscript{27} showed that optimizing the atrioventricular delay with dual-chamber cardiac pacing can improve the hemodynamic profile of patients with CHF. These results were not confirmed by other authors,\textsuperscript{28} and, even more importantly, because only right ventricular pacing is involved, this proposed strategy cannot be relevant to treatment of LBBB, for which prognosis is actually worse. Multisite stimulation with the purpose of restoring ventricular relaxation and contraction sequences to as homogeneous as possible appears to be more promising, particularly for patients with LBBB. Indeed, in patients with CHF, multisite cardiac pacing can increase the abnormally shortened left ventricular filling time while simultaneously decreasing the interventricular septal dyskinesia—thereby improving left ventricular contractility (dP/dt)—and mitral valve regurgitation.\textsuperscript{29} Several studies have recently shown that left\textsuperscript{29,30} and biventricular cardiac pacing\textsuperscript{31-33} of patients with CHF and complete bundle-branch block can improve exercise tolerance, clinical status, health-related quality of life, and neurohormonal profile and can reduce the hospitalization rate. Uncertainties remain regarding the role of QRS duration or QRS shortening as predictors of the response\textsuperscript{34} and the population of patients more likely to receive a benefit from resynchronization therapy.\textsuperscript{35} A recent review of the available literature suggests that patients with QRS duration of more than 150 ms associated with significant mitral regurgitation and P-R prolongation possibly could receive the most relevant benefit from resynchronization strategies.\textsuperscript{30,55,56} However, it has not yet been shown that this therapeutic approach can also improve the survival rate of patients with CHF. Our results provide a support to warrant controlled studies, adequately powered to specifically investigate this unsolved issue.

There are some limitations in our study that must be acknowledged. Because of the original purpose of the database that we used for this analysis, examination of electrocardiogram and measurement of QRS duration were not carried out in a single, core laboratory with standardized, blinded methods and quality control techniques. For this reason, we decided to limit the analysis only to the effect of a definite diagnosis of complete LBBB that, beyond morphologic criteria, was further confirmed with a QRS duration of more than 140 ms and, most importantly, was made in any case by quali-
fied cardiologists. Another limitation is represented by the fact that patients with complete 1-year follow-up data who were excluded from the analysis were more frequently women and presented a higher prevalence rate of NYHA classification III to IV CHF. However, this difference reflects predominantly the higher frequency of valvular heart disease among women, a cause of CHF that was not included in this analysis because its natural history and management are completely different from that of CHF as the result of ischemic heart disease or dilated cardiomyopathy. Another limitation, common to all observational and randomized control trials for CHF, was represented by the fact that cause of CHF was diagnosed without requiring a systematic use of coronary angiographic data determining a possible underestimation of the rate of ischemic cause. However, the relative prevalence of dilated cardiomyopathy and ischemic heart disease that we found in such a large population was consistent with data from other studies of patients with CHF in which coronary angiographic data were not systematically obtained. We also did not make any attempt at simultaneous adjustment for significant comorbidities other than renal failure, a limitation that becomes particularly important if we consider that CHF is becoming a sort of geriatric epidemic and that older patients commonly have an increased burden of concomitant diseases that may affect their overall prognosis. In this sense, our registry of outpatients followed by cardiologists included a CHF population with a mean age lower than that generally observed in surveys conducted by internal medicine physicians, general practitioners, or geriatricians. Finally, the definition of sudden death was not standardized but, rather, was on the basis of the individual judgment expressed by the responsible cardiologist at each center. However, it must be pointed out that the almost 50% prevalence rate of sudden death that we observed in our study is in keeping with results from previous studies of patients with CHF.

In spite of these limitations, we believe that our analysis provides important information to support the view that complete LBBB is unequivocally associated with greater disease severity and mortality in patients with CHF. The cause-and-effect relationship of this association, and whether correction of left ventricular asynchrony caused by the intraventricular conduction defect may reduce such an increased risk of mortality, should be investigated with adequately powered and properly designed studies.

References

23. Al-Khadra AS, Salem DN, Rand WM, et al. Antiplatelet agents and...


nario (F. Amaddeo, G. Barbato); Roma Osp Santo Spirito (N. Aspromonte); Roma Osp Cristo Re (V. Baldo, E. Baldo); Abruzzo Popoli (C. Frattaroli, A. Mariani); Vasto (G. Di Marco, G. Levantesi); Molise Larino (A.P. Potena), Termoli (N. Colonna, A. Montano); Campania Napoli Osp Monaldi Medicina (P. Sensale, V. Rullo); Napoli Osp S. Gennaro (A. Somelli); Nola (F. Napolitano, P. Provvisiero), Oliveto Citra (P. Bottiglieri); Puglia Bari Policlinico (N. Ciriello); Brindisi (E. Angelini, C. Andriulo); Casarano (F. De Santis); Francavilla Fontana (F. Cocco); Galatina Medicina (A. Zecca); Gallipoli (A. Pennetta, F. Mariello); Lecce Osp Fazzi (F. Magliari, A. De Giorgi, M. Callerame); Mesagne (V. Santoro); San Pietro Vernotico (S. Pede, A. Renna); Scorrano (O. De Donno, E. De Lorenzi); Taranto Osp SS. Annunziata (G. Polimeni, V.A. Russo); Tricase (R. Mangia); Basilicata Policoro (L. Trun celibito); Calabria Belvedere Marittimeo (F.P. Cariello); Catanzaro Policlinico Servizio (M. Affinita); Catanzaro Policlinico Divisione (F. Peticone, C. Cloro, D. Borelli); Cetraro (M. Matta, D. Lopresti); Cosenza Osp dell’Annunziata (G. Misuraca, R. Caporale); Cosenza Osp dell’Annunziata Medicina (P. Chiap petta); Reggio Calabria Osp Morelli (E. Tripodi, F. Tassone); Rossano (S. Salituri); Siderno (C. Errigo); Trebisacce (G. Meringolo, L. Donnangelo); Sicilia Avola (G. Canonico); Catania Osp Cannizzaro (R. Coco, M. Franco); Messina Osp Papardo (A. Coglitore, A. Donato); Messina Osp Piemonte (G. Di Tano); Messina Policlinico (D. Cento, C. De Gregorio); Palermo Casa Del Sole (M. Mongiovì); Palermo Osp Buccheri La Ferla FBF (A.M. Schillaci); Palermo Osp Civico (U. Mirto); Palermo Osp Ingrassia (F. Clemenza); Palermo Villa Sofia (F. Ingrillì); Piazza Armerina (A Cavallaro, B. Aloisi); Trapani (G. Ledda, C. Rizzo); Sardegna Cagliari Brotzu (M. Porcu, S. Salis, L. Pistis); Cagliari Osp SS. Trinità (G. Pili, S. Piras); Nuoro (I. Maoddi); Sassari (F. Uras).

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