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Lower NOAC Doses Used Despite Lack of Data PERTs Improve Response and Management of Massive PE Requiem for IVC Filters: Not so Fast When Should Patients With AF Undergo Ablation? Open-Data Movement: Share and Share Alike? Cardiovascular Innovation: Dying or Thriving? AFib Guidelines New Gui The control of the second of Camm (UK), Scipione Careri (Italy), Claudio Ceconi (Italy), Antonio Coca (Spain), Perry Elliott (UK), Cetin Erol (Turkey), Justin Ezekowitz (Canada), Covadonga Ferna Advectory, Canada), Covadonga Ferna Advectory, Canada, Covadonga Ferna Advectory, Justin Ezekowitz (Canada), Covadonga Ferna Advectory, Justin Ezekowitz (Canada), Covadonga Ferna Advectory, Canada, Covadonga Ferna Advectory, Justin Ezekowitz (Canada), Covadong Inversity, Benchangen, Benchan scientific and medical knowledge and the evidence available at the time of their publication. The ESC is not responsible in the event of any contradiction, discrepancy and/or ambiguity between the ESC Guidelines and any other official recom- mediations or guidelines issued by the relevant public health authorities, in particular in relation to good use of healthcare or therapeutic strategies. Health professionals are encour- aged to take the ESC Guidelines for the event of any contradiction, discrepancy and/or ambiguity between the ESC Guidelines and any other official recom- mediations or guidelines issued by the relevant public health authorities, in particular in relation to good use of healthcare or therapeutic strategies. Health professionals to make appropriate and accurate decisions in consultation with that patient and, where appropriate and accurate decisions in consultation with that patient and, where appropriate and accurate decisions or guidelines exempt health professionals from taking into full and careful consideration of each patient's case in light of the scientifically accepted data pursuant to their respective ethical and professional's responsibility to verify the applicable rules and regulations relating to drugs and medical devices at the time of preserved in respective ethical and European Heart Journal. & European Heart Jou implementation of preventive, diagnostic or therapeutic medical strategies; however, the ESC Guidelines of not override, in any way whatsoever, the maxy event of the expensional or the spensional or the spensio Out optimize interfact int the big back the big back to be been shall be been individual and available with a back term in the big back to be been shall be been individual and available with a back term individual and available with a back term individual and be been shall be been Guidelines. 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Different issue of HFFF [Table 3.1]. Different issue of HFmEF [Table 3.1]. Different issue 117 prognostic models 38 revealed only a moderate accuracy of models predicting mortality, whereas models designed to predict the combined endpoint of death or hospitalization, or only hospitalization, and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs are often non-specific and do not, therefore, help discriminative ability. 4. Diagnosis 4.1 Symptoms and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly with diuretic therapy. Signs, such as elevated jugular venous pressure and signs of HF due to fluid retention may resolve quickly and interpret in bate sets elevated jugular venous pressure and signs of HF due to fluid retention that appropriate is a fluid retention and outcome compares the sets sets of and outcome compares therapy. Signs are important in patients with signs of HF due to fluid retention and outcome compares therapy of sets descered with of death pressures at sets sets (NF and outcom serious development (placing the patient at risk of ur- gent hospital admission and death) and merits prompt medical attention. 4.2 Essential initial investigations: natrituretic peptides (Nes) can be texclussion at inter ecolocardiography (see also Section 12.) Antical exclussion at inter ecolocardiography (see also Section 12.) Antical exclussion at inter ecolocardiography (see also Section 12.) Antical exclussion at inter ecolocardiography (see also Section 12.) Antical exclussion at inter ecolocardiography (see also Section 12.) Antical exclussion at intervient and ecolocardiography (see also Section 12.) Antical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion at intervient and ecolocardiography (see also Section 12.) Astical exclussion astica presenting with symptoms or signs for the first time, non-urgently in primary care or in a hospital outpatient clinic (Table 4.1), the probability of HF should first be evaluated based on the patient's prior clinical history [e.g. coronary artery disease (CAD), arterial hypertension, diuretic use], presenting symptoms (e.g. orthopnoea), physical examination (e.g. bilateral oedema, in- creased jugular venous pressure, displaced apical beat) and resting ECG. If all elements are normal, HF is highly unlikely and other diag-noses need to be considered. If at least one element is abnormal, plasma NPs should first be evaluated based on the patient's prior clinical history [e.g. coronary artery disease (CAD), arterial hypertension, diuretic use], presenting symptoms (e.g. orthopnoea), physical examination (e.g. bilateral oedema, in- creased jugular venous pressure, displaced apical beat) and resting ECG. If all elements are normal, HF is highly unlikely and other diag-noses need to be considered. If at least one element is abnormal, plasma NPs should first be evaluated based on the patient's prior clinical hypertension, diuretic use], presenting symptoms (e.g. orthopnoea), physical examination (e.g. bilateral oedema, in- creased jugular venous pressure, displaced apical beat) and resting ECG. If all elements are normal, HF is highly unlikely and other diag-noses of the clinical hypertension, diuretic use], presenting symptoms (e.g. orthopnoea), physical examination (e.g. bilateral oedema, in- creased jugular venous pressure, displaced apical beat, 20 agnosis of HF EF expective the exclusion threshold or if crualating NP levels is above the exclusion threshold or if crualating NP levels is above the exclusion threshold or if crualating NP levels is above the exclusion of the clinical conditions. The diagnosis of HFPEF repersented by objective measures of cardiac dysfunction dysfunction dysfunction dysfunction dysfunction dysfunction dysfunction dysfunctions of the presence of symptoms and/or signs of HFF (PEF expecte Support by the big by Here the function of the funsyment of filling pressures [pulmonary capillary wedge pressures [PCWP] > 15 mmHg or left ventricular end diastolic pressures. The diagnosis of HEDEP in patients with AF is difficult. Since AF is associated with higher NP levels, the use of NT-proBNP or levels and cardiac output, can be performed 87. The diagnosis of HEDEP in patients with AF is difficult. ented by other modalities, chosen according to their ability to answer specific clinical questions and taking account of contraindications to and risks of specific tests 71 73 In general imaging quality. Normal values may with age sex and imaging modality 5 v). safety and cost.68.69.72 Echocar- diography may be control of the second s r- ders. In case of poor image quality, contrast agents astolic dysfunction. Therefore, a comprehensive echocardiography examination incorpor sure is derived from an optimal recording of maximal tricuspid regurgitant jet and the tricuspid systolic gradient, together with an estimate of RA pressure on the basis of inferior vena cava (IVC) size and its soesophageal echocardiography Transoesophageal echocardiography (TOE) is not n ded in the routine diagnostic assessment of HF; however, it may be valuable in some clinical scenarios of patients with valve disease, suspected and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease avertic disease and for ruling out intracavitary thrombi in AF patient's symptoms using TTE alone, a TOE examin- ation should be performed avertic disease aver clusive diastolic narameters at rest 85.86.5.5 ce from RCTs has failed to show that viability assessed by CMR or other means identified patients who obtained clinical benefit from revas- cularization.105 -107 Clinical limitations of CMR include local expertise. lower availability and higher costs compared with echocardiography, uncertainty about safety rity, 5.9 Cardiac computed tomography The main use of cardiac CT in patients with HF is as a non-invasive means to visualize the coronary anatomy in patients with HF with low intermediate pre-test probability of CAD or those with equivo- cal non-invasive stress tests in order to exclude the diagnosis of CAD, in the giography should be considered in patients with HF and intermediate to high pre-test probability of CAD and the presence of ischaemia in non-invasive stress tests in order to establish the ischaemic aetiology and CAD sev s LVEF in order to identify patients with HF who would h dial structure and function in subjects to be exposed to treatment which potentially can damage myocardial structure and patients with poor acoustic window and patients with a strain rate), should be considered in a TTE protocol in subjects at risk of developing HF in order to identify myocardial structure and function indices, i.e. strain and strain rate) and the preclinical structure and function at the preclinical structure and strain rate) and the preclinical structure and function at the preclinical structure and function a int of cautions/contra-indications to CMR). I C CMR with LGE should be considered in patients with dilated cardiomyopathy in order to dist al non-invasive stress tests in order to rule out coronary artery stenosis. IIb C Reassessment of myocardial structure and function is recomm nded using non-invasive imaging: - in patients presenting with worsening HF symptoms (including episodes of AHF) or experiencing any other important cardiovascular event; - in patients with HF who have received evidence-based pharmacotherapy in maximal tolerated doses, before the decision on device implantation (ICD,CRT); - in patients exposed to therapies which may damage the myocardium ents). I C AHF ¼ acute heart failure; CAD ¼ coronary artery disease; CMR ¼ cardiac magnetic resonance; CRT ¼ cardiac resynchro ire with preserved ejection fraction; HFmrEF ¼ heart failure with mid-range ejection fraction; HFrEF ¼ heart failure with reduced ejection fraction; ICD ¼ implantable cardioverter-defibrillator; LGE ¼ late gadolinium enhancement; LVEF ¼ left ventricular ejection fraction; PET ¼ positron emi 16 17. 5.10 Other diagnostic tests Comprehensive assessment of patients with HF comprises, besides medical history and physical examination, including adequate imaging techniques, a set of additional diagnostic tests, i.e. laboratory vari- ables, ECG, chest X-ray, exercise testing, invasive hae ce(s) supporting recomm sion computed tomography; TTE ¼ transthoracic echocardiography. a Clas ns for genetic testing in patients with HF are based on the position statement of the European Society Recommendations for diagnostic tests in natients with heart failure Recommendations endations Classa Level b Ref c The following diagnostic tests are recommended/should be considered for initial assessment of a patient with newly diagnosed HF in order to evaluate the patient's suitability for particular therapies, to detect reversible/treatable causes of HF and co-morbidities interfering with HF: - haemoglobin and WBC - sodium de GFR) - liver function tests (bilirubin,AST,ALT, GGTP) - glucose, HbA1c -TSH - ferritin,TSAT =TIBC I C - natriuretic pepi tion is needed to plan and monitor treatment. I C Exercise testing in patients with HF: - is recommended as a part of the ernative pulmonary or other diseases, which may contribute to dyspnoea. It may also identify pulmonary congestion/o amic status is unclear. IIb C EMB should be considered in natients with rapidly progressive HE despite standard therapy when there is a probability of a IIa C 93 IIb C 121 Ultrasound measurements of the standard therapy when there is a probability of a IIa C 93 IIb C 121 Ultrasound measurements of the standard therapy when there is a probability of a IIa C 93 IIb C 121 Ultrasound measurements of the standard therapy when there is a probability of a IIa C 93 IIb C 121 Ultrasound measurements of the standard therapy when there is a probability of a IIa C 93 IIb C 121 Ultrasound measurements of the standard the tural heart disease: IIa C - may be considered in order to adjust therapy in patients with HF who remain severely symptomatic despite initial standard therapies and whose haemodyn irement of inferior vena cava diameter may be considered for the assessment of volaemia status in patients with HF. IIb C AHF ¼ acute heart failure: ALT ¼ alanin ; BNP ¼ B-type natriuretic peptide; ECG ¼ electrocardiogram; eGFR ¼ es mmendation. b Level of evidence. c Reference(s) supporting recommendations. ESC Guidelines Page 17 of 85 byguestonMay22,2016 18. of Cardiology Working Group on Myocardial and Pericardial Dis- eases. 94 In most bati ended in patients with HCM, idiopathic DCM and ARVC. R rigin and should also be considered for genetic testing. HCM is mostly inherited as an autosomal dominant disease with variable expressivity and age-related penetrance. Currently, more than 20 genes and 1400 mutations have been identified, most of which are located in the sarcomere genes encoding cardiac b-myosin binding protein C (MYBPC3).88,122 DCM is idiopathic in 50% of cases, about one-third of which are been identified, most of which are located in the sarcomere genes and 1400 mutations have been identified. eone with sufficient knowledge of the specific psychological, social and medical implica- tions of a diag e related to the cytoskeleton. The most frequent ones are titin (TTN), lamin (LMNA) and desmin (DES).88,123 ARVC is hereditary in most cases and is ca osis. Determination of the genotype is important, since some plamban (PLN)] are related to a poorer prognosis. DNA analysis could also be of help to establish the diagnosis of rare forms, such as mite nt (O), genetic inheritance pattern (G), aetio-logical annotation (E), including genetic defect or underlying disease/ substrate, and the functional status (S) of the disease.125 6. elopment of overt heart failure or preventing death before the onset of symptoms There is considerable evidence that the onset of HF may be delayed or prevented through interventions aimed at modifying risk factors for HF or treating asymptomatic LV s eventing death before the onset of symptoms i here is considerable evidence that the onset of intrinay be delayed of province anotal intervalue and an anotal intervalues in hypertensive non-diabetic subjects, the recent SPRINT study has already second anotal intervalue and the i y the onset of HF and some also show that it will prolong life.126 - 129 Different antihypertensive drugs [diuretics, ACEIs, angio- tensin receptor blockers (ARBs), beta-blockers] have been shown to be effective, especially in older people usive non-diabetic subjects, the recent SPRINT study has already demonstrated that treating hypertension to a lower goal [systol SBP), 120 mmHg vs. 140 mmHg lin older hypertensive subjects (>75 years of age) or high-risk hypertensive patients reduces the risk of cardiovascular disease, death and hospitalization for HF.129 Recently, empaglifozin (an inhibitor of sodium-glucose cotran-sporter 2), has been shown to improve out on of hypoglycaemic therapy to drive down glycated haemoglobin (HbA1c) with agents other than empaglifozin does not reduce the risk of developing HF (for details see Section 11.6 on diabetes). Although smoking cessation has not been shown to reduce the risk of developing HF, the epidemiological iological associations with the de-velopment of cardiovascular disease131 suggest that such advice, if followed, would be beneficial. The association between alcohol intake and the risk of developing de novo HF is U-shaped, with the lowest risk with n (up to 7 drinks/week).132 -134 Greater alcohol intake may trigger the development of toxic cardiomyopathy, and when present, complete abstention from alcohol is recomm mended. An inverse relationship between physical activity and the risk of HF has been reported. A recent meta-analysis found that doses of physical activity in excess of the quideline recommended minimal levels may be required for more substantial reductions in HF risk. 135 It has been shown that among subjects >40 years of age with ei- ther cardiovascula scularization, have been shown to reduce the risk of developing HF or sed plasma concentrations of NPs.136,143 A primary percutaneous coronary intervention (PCI) at the earli- est phase of an ST segment elevation myocardial infarction (STEMI) to reduce infarct size decreases the risk of developing a substantial reduction in LVE ind (LCZ696) that combines the moieties of an ARB (valsartan) and a neprilysin (NEP) inhibitor (sacubitril) has recently been shown to be superior to an ACEI (enalapril) in reducing the risk of death and of hospitalization for HF in a single trial with strict inclusion/ex- clusion criteria. ARBs have not been consistently proven to reduce mortality in patients with HFrEF patients with HFrEF patients with HFrEF uld be restricted to patients intolerant of an ACEI or those who consume to restricted to prevent or delay the onset of HF and the onset of HF and the onset of HF. I A 126, 129, 150, 151 Tree and prolong life. I A 137-140, 152 Courselling and treatment for smoking cessation and alcohol intervent or delay the onset of HF. I C 131-134 Treating of HF. I C 131-134 Treating of HF. I C 131-134 Treating of HF. I C 130, 141, 153-155 ment with statins is recommended in patients with or at high-risk of CAD whether or not they have LV systolic dysfunction, in order to prevent or de IIa A 142 Beta-blocker is recommended in patients with asymptomatic LV systolic dysfunction and a history of myocardial infarction, in order to prevent or delay the onset of HF or prolong life. I The second process of rated dose. In patients admitted due to acute HF (AHF) beta-blockers should be cautiously initiated in hospital, once the patient is stabilized. An individual patient data meta-analysis of all the major beta-blockers should be cautiously initiated in hospital admis- sions and mortality in the subgroup of patient stabilized. An individual patient with HFrEF who are in AF.177 However, since this is a retrospective subgroup of patients with HFrEF who are in AF.177 However, since this is a retrospective subgroup analysis, and because beta-blockers should be cautiously initiated in hospital, once the patient is stabilized. An individual patient data meta-analysis of all the major beta-blockers should be cautiously initiated in hospital admis- sions and mortality in the subgroup analysis. Beta-blockers should be considered for rate control in patients with HFrEF and AF, es- pecially in those with high heart rate (see Section 10.1 for details). Beta-blockers are reco in Web Table 7.5. 7.2.3 Mineralocorticoid/aldosterone receptor antagonists MRAs (spironolactone and eplered mmended in patients with a history of myocardial infarction and asymptomatic LV systolic dysfunction to reduce the risk of death (see Section 6). Practical guidance on how to use beta-blockers is give [] soft affinity, other ster- oid hormone (e.g. corticosteroids, and rogens) receptors. Spirono- lactone or eplerented according to clinical symptomatic patients (in patients with an ACEI and a beta-blocker) with HFrEF and LVEF \leq 35%, to reduce mortality and renal function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with an ACEI and a beta-blocker) with HFrEF and LVEF \leq 35%, to reduce mortality and renal function should be performed according to clinical function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with impaired renal function should be exercised when MRAs are used in patients with impaired renal function function for a patient with reduced ejection fraction f dion. ACE1 ¼ angiotensin-converting enzyme inhibitor; BNP ¼ Betype natriuretic peptide; CRT ¼ cardiac resynchronization therapy; HF ¼ heart failure with reduced ejection fraction; H-ISDN ¼ hydralazine and isosorbide dinitrate; HFrEF ¼ heart failure with reduced ejection fraction; HFrEF ¼ heart failure with reduced ejection fraction; NF ½ Betype natriuretic peptide; CRT ¼ cardiac resynchronization therapy; HF ¼ heart failure with reduced ejection fraction; HFrEF ¼ heart failure with reduced ejection fraction; HFrEF ¼ heart failure with reduced ejection fraction; HFrEF ¼ heart failure with reduced ejection fraction; NF ½ rentricular eje dividua-lized decision). For further details, see Sections 7 and 8 and corresponding web pages. ESC Guidelines Page 21 of 85 byguestonMay22,2016 22. mortality and morbidity have not been studied in RCTs. A Co- chrane meta-analysis has shown that in patients with chronic HF, loop and thiazide diuretics appear to reduce the risk of death and worsening HF compared with an active control, diuretics appear to improve exercise capacity. 178,179 Loop diuretics produce a more intense and shorter diuresis than thiazides stically and the combin- ation may be used to treat resistant oedema. However, adverse effects are more likely and these combinations should only be used with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the individual needs over time. In selected asymptomatic entry is to achieve and main- tain euvolaemia with the lowest achievable dose. The dose of the diuretic must be adjusted according to the adjusted acc ureticsd +ACE-I/ ARB -ACE-I/ ARB +ACE-I/ ARB -ACE-I/ ARB Spironolactone/ eplerenone 12.5-25 50 50 100- 200 Amiloride 2.5 5 5-10 10-20 Triamterene 25 50 100 200 ACE-I ¼ angior ous; dose might need to be adjusted according to volume status/ weight; excessive doses may cause renal impairment and ototoxicity. b Do not use thiazides if estimated glomerular filtration rate ,30 mL/min/1.73 m2 , except when prescribed synergistically with loop diu inder is a non-thiazide sulfonamide. d A mineralocorticoid antagonist (MRA) i.e. spironolactone/eplerenone is always preferred. Amiloride and triamterene should not be combined with an MRA. Table 7.2 Evidence-based doses of disease-modifying drugs in key randomized trials in heart failure with reduced ejection fraction (or after myocardial infarction) Starting dose (mg) ACE-I Captoprila 6.25 t.i.d. 50 t.i.d. 20 b.i.d. Enalapril 2.5 b.i.d. 20 b.i.d. Lisinoprilb 2.5-5.0 o.d. 20-35 o.d. Ramipril 2.5 b.i.d. 20 b.i.d. 10 o.d. Ramipril 2.5 b.i.d. 20 b.i.d. Enalapril 2.5 b.i.d. 20 b.i.d. 25 b.i.d. 25 b.i.d. 20 b.i.d. 25 b.i.d. 20 b.i.d. 10 o.d. Ramipril 2.5 b.i.d. 20 b.i.d. 20 o.d. 10 o.d. Ramipril 2.5 b.i.d. 20 b.i.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 b.i.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 20 b.i.d. 10 o.d. Ramipril 2.5 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 10 b.i.d. 150 b.i.d. 10 b.i.d. 150 b.i.d. 10 c.d. Ramipril 2.5 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 25 b.i.d. 20 c.d. 10 o.d. Ramipril 2.5 b.i.d. 10 c.d. Ra inferior to a treatment that does). d A maximum dose of 50 mg twice daily can be administered to patients weighing over 85 kg. ESC GuidelinesPage 22 of 85 byguestonMay22,2016 23. 7.3.2 Angiotensin receptor neprilysin inhibitor (ARNI)]. The first in class is LCZ696, which is a molecule that combines the moieties of valsartan and sacubitril (neprilysin inhibitor) in a single substance. By inhibitor of a treatment that does). d A maximum dose of 50 mg twice daily can be administered to patients weighing over 85 kg. ESC GuidelinesPage 22 of 85 byguestonMay22,2016 23. 7.3.2 Angiotensin receptor neprilysin inhibitor (ARNI)]. The first in class is LCZ696, which is a molecule that combines the moieties of valsartan and sacubitril (neprilysin inhibitor) in a single substance. By inhibitor (ARNI)]. gradation of NPs, bradykinin and other peptides is slowed. High circulating A-type natriuretic peptide (ANP) and BNP exert physiologic effects thr tan compared with an ACEI (enalapril) on morbidity Other pharmacological treatments recommended in selected patients with symptomatic (NYH) rone secretion. Selective AT1-receptor blockade reduces vasoconstriction, sodium and water retention and myocardial hypertrophy.187,188 A recent trial investiga signs and/or symptoms of congestion. I B 178, 179 Diuretics should be considered to reduce the risk of HF hospitalization in patients with signs and/or symptoms of ptor neprilysin inhibitor Sacubitril/valsartan is recommended as a replacement for anACE-I to further reduce the risk of HF hospitalization and cardiovascular death in symptomatic petients with LVEF <35%, in sinus rhythm and a resting heart rate >70 bpm despite treatment with an evidence-based dos The construction of the probability of the provided the probability of Tarte an ACE-I (patients should also receive a beta-blocker and an MRA). I B 182 AnARB may be considered to reduce the risk of HF hospitalization and de ≥ 3 months of OMT, provided they are expected to survive substantially longer than one year with good functional status, and they have: • IHD (unless the survive substantially longer than one year with good functional status, and they have: • IHD (unless the survive substantially longer than one year with good functional status, and they have: • IHD (unless the survive) are supported to survive substantially longer than one year with good functional status, and they have: • IHD (unless the survive) are supported to survive substantially longer than one year with good functional status, and they have: • IHD (unless the survive) supported to survive) substantially longer than one year with good functional status, and they have: • IHD (unless the survive) supported to survive) suppo sist device, or cardiac transplantation. III C 229-233 Patients should be carefully evaluated by an experienced cardiologist before generator replacement, because management goals and the patient's needs and clinical status may have changed. IIB C 239-241 CAD ½ coronary artery disease; CRT ¼ cardiac resynchronization therapy; DCM ¼ dilated cardiomyopathy; HF ½ estonMay22,2016 27. 8.1.2 Prinary prevention of sudden cardiac death Although amiodarone may have reduced mortality in older trials of HF,242,243 contemporary studies conducted since the s with CRT (CRT-Ps), reduce the risk of sudden death (see Section 7). An ICD reduces the rate of sudden arrhythmic death in patients with HFrEF.249,250 In patients with moderate or severe HF, beta-blockers suggest that it does not reduce mor- tality in patients with HFrEF.227,244,245 Dronedarone246,247 and class I antiarrhythmic agents/246,248 should not be used for prevention of arrhythmias in this population. Some guideline-recommended therapies, including beta- blockers, MRAs, sacubitril/valsartan and pacemakers with CRT (CRT-Ps), reduce the risk of sudden death (see Section 7). An ICD reduces the risk of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the risk of sudden death (see Section 7). An ICD reduces the rate of sudden arrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,247 and class I antiarrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,247 and class I antiarrhythmic agents/246,248 should not be used for prevention of arrhythmic agents/246,248 should not be used for prevent about two deaths (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sudden death (see Section 7). An ICD reduces the rate of sud wed no benefit in patients who had an ICD im- planted within 40 days after a myocardial infarction. 158,228 Al- though sudden arrhythmic deaths were reduced, this was balanced by an increase in non-arrhythmic deaths. Accordingly, an ICD is contraindicated in this time period. A wearable defibril- lator may be considered if the patient is deemed to be at high risk of ventricular fibrillation, although evidence from randomized trials is lacking.239 - 241 ICD implantation is recommended only after a sufficient trial (minin (eaths. Accordingly, an ICD is contraindicated in this time period. A wearable defibril- lator may be considered if the patient is deemed to be at high risk of ventricular fibrillation, although evidence from randomized trials is lacking.239 - 241 ICD implantation is recommended only after a sufficient trial (minin (eaths were reduced, this was balanced by an increase in non-arrhythmic deaths. Accordingly, an ICD is contraindicated in this time period. A wearable defibril- lator may be considered if the patient is deemed to be at high risk of ventricular fibrillation, although evidence from randomized trials is lacking.239 - 241 ICD implantation is recommended only after a sufficient trial (minin (eaths were reduced, this was balanced by an increase in non-arrhythmic deaths. Accordingly, an ICD is contrained with a LVEF of 30-35%. Were than 400 patients with an LVEF of 30-35% were included in the landmark studies, and although there was no statistical interaction between treatment effect and LVEF, the evidence of benefit is less robust in this group of patients. Conservative programming with long delays252 between detec- tion and the ICD delivering therapy dramatic s for CRT, a ventricular assist device or cardia l life ex- pectancy and are likely to die from pump failure. Patients with serious co-morbidities who are unlikely to survive substantially more than 1 year are unlikely to obtain substantial benefit from an ICD. 229 - 233 Patients should be counselled as to the purpose of an ICD, com- plications related to implantation and device activation (predomin- antly inappropriate shocks) and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deactivated (terminal disease) or explanted (infection, recovery of LV function). 255 If HF deteriorates, and under what circumstances it might be deteriorates. idered for a lim- ited period of time in selected patients with HF who are at high risk for sudden death but otherwise are not suitable for ICD implant- ation (e.g. those with poor LVEF after acute myocardial damage until LV function recovers, patients scheduled for heart transplant- ation).239 -241,260 However, no prospective RCTs ce have been reported. For detailed recommendations on the use/indications of ICD we refer the reader to the ESC/European Heart Rhythm Association (EHRA) guide lines on ventricular tachyarrhythmias and sudden car- diac death. 260 ESC Guidelines Page 27 of 85 byguestonMay22,2016 28. 8.2 Cardiac resynchronization therapy CRT improves symptoms286 and well-being286 and reduces morbidity and mortality. 266 Of the improvement in guality-adjusted life-yea nd favourably to CRT.286 Several character-istics predict improvement in morbidity and mortality, and the ex-tent of reverse remodelling is one of the most important mechanisms of action of CRT. Patients with ischaemic aetiology will have less improvement in LV function due to myocardial scar tis- sue, which is less improvement in allows the inclusion criterion in all nized trials. But QRS morphology has also been related to a beneficial re- sponse to CRT. Several studies have shown that patients with left bundle branch block (LBBB) morphology are more likely to respond favourably to CRT, we eas there is less certainty about patients with non-LBBB morphology. However, patients with LBBB morph- ology often have wider ORS duration, and there is a current debate about whether ORS duration or ORS morphology is the main pre-dictor of a beneficial response to CRT. Evidence from two IPD meta-analyses RS duration, there is little evidence to suggest that QRS morphology or aetiology of action of CRT is not recommended if QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 266 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible harm from CRT when QRS duration is , 130 ms, thus im- logical and an IPD meta-analysis 265 suggest possible es. 288 If the primary reason for implantation in patients with heart failure Recommendations for cardiac resynchronization therapy implantation in patients with heart failure Recommended for symptomatic patients with HF in sinus rhythm with a QRS duration > 150 msec and LBBB QRS morphology and with LVEF <35% despite OMT in order to improve symptomatic patients with herapy implantation in patients with herapy impla ommended for symptomatic patients with HF in sinus rhythm with a QRS duration of 130-149 msec and LBBB QRS morphology and with LVEF <35% despite OMT in order to improve symptoms and reduce morbidity and mortality. I B 266, 273 CRT may be considered for symptomatic patients with HF in sinus rhythm with a QRS duration of 130-149 msec and non-LBBI vith HFEF regardless of NYHA class who have an indication for ventricular pacing and high degree AV block in order to reduce morbidity. This includes patients with AF (see Section 10.1), I A 274-277 CRT should be considered for patients with LVEF <35% in NYHA class III-IVd despite OMT in order to improve symptoms and reduce morbidity and mortality. If hey are 73 CRT rather than RV pacing is recommended for patients with HFrEF regardless rder to improve symptoms and reduce morbidity and mortalit QRS duration ≥130 msec provided a strategy to ensure bi-ventricular capture is in place or the patient is expected to return to sinus rhythm. IIa B 275, 278-281 Patients with HFrEF who have received a conventional pacemaker or an ICD and subsequently develop wors ming HF despite OMT and who have a high proportion of RV pacing may be considered for upgrade to CRT. This does not apply to patients with stable HF. IIb B 282 CRT is contra-indicated in patients with a QRS duration < 130 msec. III A 266, 283-285 AF ¼ atrial fibrillation · ORS 4 O. R and S waves (combination of three of the graphical deflections): RV 4 right ventricular, a Class of recommendation. b Level of evidence, c Reference(s) supporting recommendations, d Use judgement for patients with yular; CRT ¼ cardiac resynchronization therapy; HF ¼ heart failure: HFrEF ¼ heart failure with reduced ejection fraction; ICD ¼ implantable cardioverter-defibrillator; LBBB ¼ left bundle branch block; LVEF ¼ left v ence in outcome was not observed between CRT and RV pacing in a subgroup analysis of RAFT267 or in patients with HFrEF regardless of NYHA class who have an indication for ventricular pa- cing in order to reduce morbidity, although no clear effect on mor- tality was observed. Patients with HFrEF who acing may exacerbate cardiac dyssyn- chrony. This can be prevented by CRT, which might improve patient outcomes.274,275,277,290 However, a diffe nal pacemaker or an ICD and subsequently develop wor-sening HF with a high proportion of RV pacing, despite OMT, should be considered for upgrading to CRT. Only two small trials have con red pharmacological therapy alone vs. CRT in patients with AF, with conflicting results. Several studies have indicated that CRT is superior to RV pacing in patients undergoing atrio-ventricular (AV) node ablation.275,277,290 How- ever, CRT is not an indication to carry out AV node ablation except in rare cases when ventricular rate (.110 bpm) despite attempts at pharmacological rate control. A subgroup analysis of patients with AF from the RAFT study found no benefit from CRT-D con ifficulty in pacing severely diseased myocardium (which might not be amenable to the above) is uncertain. This observation has not been confirmed in a rand ricular capture.276 Observational studies report that when biventricular capture is ,98%, the prognosis of patients with CRT declines.277 Whether this association reflects a loss of re-synchronization (which might be remedied by device programming), poor placing of the LV lead (which might be avoided at with cRT declines.277 Whether this association reflects a loss of re-synchronization (which might be remedied by device programming), poor placing of the LV lead (which might be avoided at with cRT, but this is true of any treatment for HFrEF and does not reliably predict less clinical benefit.293 Pacing thresholds are higher in scarred myo- cardium and, if poss d avoid such re-gions. 294.295 Although patients with extensive scarring have an intrinsically worse prognosis, there is little evidence that they obtain less prognosic benefit from CRT.266 The reader is directed to guidelines on pacing and CRT for re-commendations on device implantation procedures. The value of trying to optimize AV or VV intervals after implantation using echo- or electrocardiographic criteria or blood pressure response is uncertain, but may be considered for patients who have had a dis-appointing response to tion. Cardiac contractility modulation (CCM) is similar in its mode of insertion to CRT, but it involves non-excitatory electrical stimula- tion of the ventricle during the absolute refractory period to enhance contractile performance without activati tion.298,299 These include vagal nerve stimulation, spinal cord stimulation, carotid body ablation and renal denervation, but so far none of the devices has improved symptoms or outcomes in RCTs. Devices for remote monitoring are discussed in Section 14. 9 is of HFpEF requires an LVEF between 40 and 49% are considered to have HFmrEF (for de- tails, please refer to Section 3). Patients with both HFmrEF and HFpEF. Accordingly, the quidance in this section applies to make re- commendations for each phenotype separately. In clinical practice and clinical trials, compared with HFmrEF patients, only slightly fewere available, it might be possible to make re- commendations for each phenotype separately. In clinical practice and clinical trials, compared with HFmrEF patients, only slightly fewere available, it might be possible to make re- commendations for each phenotype separately. In clinical practice and clinical trials, compared with HFmrEF patients, only slightly fewere available, it might be possible to make re- commendations for each phenotype separately. In clinical practice and clinical trials, compared with HFmrEF patients, only slightly fewere available, it might be possible to make re- commendations for each phenotype separately. ing a reduction in new-onset HF,127 or failure to distinguish between guideline recom-ertension) and non-cardiovascular diseases [diabetes, chronic kidney disease (CKD), a mendations for HFrFF and HFmrEF/HFnFF or a belief that existing clinical trials provide some evidence of benefit with these agents. A summary of phase II and III clinical trials et been shown, convincingly, to reduce mor-bidity or mortality in patients with HFpEF or HFmrEF. However, since these patients are often elderly and highly symptomatic, and often have a poor quality of life,307 an importa oxin beta-blockers or rate-limiting CCBs, or a combin-ation of these, should be preferred is unkn own. Verapamil or diltia- zem should not be combined with a beta-blocker. There are insufficient dat n and HEDEF or HEMTEF should not receive an ARB (olmesartan) if they are receiv- ing ACEIs and beta-blockers.318 The first-line oral hypoglycaemi uld be considered when assessing patients. I lation AF is the most complications (particularly stroke) and may impair cardiac function. 323 Patients with a worse outcome HF 316 Incident HF precipitated by AF is associated with a morse outcome than honore in probably because it is body precipitated by AF is associated with a worse outcome to myticular cardiac function. 323 Patients with precipitated by AF is associated with a worse outcome to HF 316 Incident HF precipitated by AF is associated with a worse outcome to HF 316 Incident HF precipitated by AF is associated with a worse outcome to HF. The cardian provide and the precipitation of a patient with least 150 by Bmytoric cardiac graphical by a start of a patient with least 150 by Bmytoric and the precipitated by AF is associated with a worse outcome to HF. 316 Incident HF precipitated by AF is associated with a worse outcome to PF. The cardian provides and the precipitation of a patient with least 150 by Bmytoric and the precipitation of a patient with least 150 by Bmytoric and the precipitation of a patient with least 150 by Bmytoric and the precipitation of a patient with least 150 by Bmytoric and the precipitation of a patient with least 150 by Bmytoric and the precipitation of a patient with part of the precipitation of a patient with part of precipitation and patient with least 150 by Bmytoric and the precipitation of a patient with part of precipitation of a patient with part of precipitation and precipitation of a patient with part of precipitation and precipitation an rticularly stroke) and may impair cardiac function leading to worsening sy to the stabilished HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established HF is associated with a more benign prognosis.331 but new-onset AF in a pa- tient with established H The first control Assessed during rate control assessed during rate control interview and control assessed during rate control asses reduce symptomatic paroxysms of AF and will help maintain patients in sinus rhythm after spontaneous or electrical cardioversion.343-346 When used, the need for continued administra- tion of amiodarone should be regularly reviewed and justified. The safety and efficacy of catheter ablation in the atria and pul- monary veins (PV) as a rhythm control strategy in HF is at present uncertain except for tachycardia induced cardioversion.343-346 When used, the need for continued administra- tion of amiodarone should be regularly reviewed and justified. 1203 patients with per-sistent AF, HF and an ICD or CRT devices in terms of the output of the analysis that includes and because of a cuted with mixed suggests an encour- aging symptoms and or signs of the output of the analysis that includes and because of a cuted with and and analysis that includes and because of a cuted analysis that includes and because of a non-aging symptoms and/or signs of the analysis that includes and because of a non-aging symptom atic status. IIb B 344 AF ablation may be considered in order to restore sinus rhythm. Ib B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone may be considered prior to (and following) successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone is not recommended because of an increased risk of the following successful electrical cardioversion to maintain sinus rhythm. IIb B 342, 360 Dronedarone is not recommended because of an increased risk of the following successful electrical cardioversion to maintain sinus rhythm. II hospital admissions for cardiovascular causes and an increased risk of premature death in NYHA Class III-IV patients. III A 247, 347 Class I antiarrhythmic agents are not recommended because of an increased risk of premature death. III A 248, 364, 365 ÅF ¼ atrial fibrillation; HF ¼ heart failure; NYHA ¼ New York Heart Association, OMT ¼ optimal medical therapy. Patients should generally be anticoagulated for 6 weeks prior to electrical cardiovascular causes and an increased risk of premature death. III A 248, 364, 365 ÅF ¼ atrial fibrillation; HF ¼ heart failure; NYHA ¼ New York Heart Association, OMT ¼ optimal medical therapy. Patients should generally be anticoagulated for 6 weeks prior to electrical cardioversion. a Class of recommended in the 248, 364, 365 ÅF ¼ atrial fibrillation; HF ¼ heart failure; NYHA ¼ New York Heart Association, OMT ¼ optimal medical therapy. Patients with HF and Association, OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical therapy. Patients with HF and Association; OMT ¼ optimal medical th The function of the reaction of the rest or left ventricular dysfunction, Hypertension, Age 2 75 (doubled), Diabetes, Stroke (doubled)-Vascular disease, Age 65-74, Sex category (female); HAS-BLED ¼ Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile international normalized ratio, Elderly (.65 years), Drugs/alcohol concomitantly (1 point each); HF ¼ heart failure; LMWH ¼ low molecular weight heparin; NOAC ¼ non-vitamin K antagonist oral anticoagulant; NYHA ¼ New York Heart Association; TOE ¼ transoesophageal echocardiography. a Class of recommendations. b Level of evidence. c Reference(s) supporting recommendations. ESC Guidelines Page 33 of 85 byguestonMay22,2016 34. HF and frequent, recurrent ventricular arrhythmias and therefore should be considered in such patients. Seeking the advice of the members of the HF Team with expertise in electro-physiology is recommendations. ESC Function, Category (female); hereina and corrected in patients with ventricular arrhythmias in heart failure Recommendations. Category (e.g. low serum potassium/ magnesium, ongoing ischaemia) should be sought and corrected in patients with HFrEF and ventricular arrhythmias. Ila C Treatment with beta-blocker, MRA and sacubitril/valsartan reduces the risk of sudden death and is recommended for patients with HFrEF and ventricular arrhythmias (as for other patients) (see Section 7). I A 162, 170-175 Implantation of an ICD or CRT-D device is the risk of sudden death and is recommended for patients with HFrEF and ventricular arrhythmias (as for other patients) (see Section 7). I A 162, 170-175 Implantation of an ICD or CRT-D device is the rest or the rest orest or the rest or the rest orest or the rest orest or the rest or