Society Guidelines

The 2013 Canadian Cardiovascular Society Heart Failure Management Guidelines Update: Focus on Rehabilitation and Exercise and Surgical Coronary Revascularization

Primary Panel: Gordon W. Moe, MD, MSc, FRCPC (Chair),[a]
Justin A. Ezekowitz, MB, BCh, MSc, FRCPC (Co-Chair),[b] Eileen O'Meara, MD, FRCPC,[c]
Jonathan G. Howlett, MD, FRCPC,[d] Steve E. Fremes, MD, FRCPC,[e]
Abdul Al-Hesayen, MD, FRCPC,[a] George A. Heckman, MD, MSc, FRCPC,[f]
Anique Ducharme, MD, FRCPC,[c] Estrellita Estrella-Holder, RN, BN, MScA, CCN(C),[g]
Adam Grzeslo, MD, CCFP, FCFP,[h,i] Karen Harkness, RN, BScN, CCNC, PhD,[i]
Serge Lepage, MD, FRCPC,[k] Michael McDonald, MD, FRCPC,[l]
Robert S. McKelvie, MD, FRCPC,[i] Anil Nigam, MD, MSc, FRCPC,[c]
Miroslaw Rajda, MD, FRCPC,[m] Vivek Rao, MD, PhD, FRCPS,[l]
Elizabeth Swiggum, MD, FRCPC,[n] Sean Virani, MD, FRCPC,[o] Vy Van Le, MD, FRCPC,[p]
Shelley Zieroth, MD, FRCPC,[g] **Secondary Panel:** J. Malcolm O. Arnold, MD, FRCPC,[q]
Tom Ashton, MD, FRCPC,[r] Michel D'Astous, MD, FRCPC,[s] Paul Dorian, MD, FRCPC,[a]
Nadia Giannetti, MD, FRCPC,[t] Haissam Haddad, MD, FRCPC,[u] Debra L. Isaac, MD, FRCPC,[d]
Simon Kouz, MD, FRCPC, FACC,[j] Marie-Hélène Leblanc, MD, FRCPC,[v]
Peter Liu, MD, FRCPC,[u] Heather J. Ross, MD, FRCPC,[l] Bruce Sussex, MD, FRCPC,[w] and
Michel White, MD, FRCPC[c]

[a] St Michael's Hospital, University of Toronto, Toronto, Ontario, Canada; [b] University of Alberta, Edmonton, Alberta, Canada; [c] Institut de Cardiologie de Montréal, Montreal, Québec, Canada; [d] University of Calgary, Calgary, Alberta, Canada; [e] Sunnybrook Health Science Centre, Toronto, Ontario, Canada; [f] University of Waterloo, Waterloo, Ontario, Canada; [g] Cardiac Sciences Program, St Boniface General Hospital, Winnipeg, Manitoba, Canada; [h] Joseph Brant Memorial Hospital, Burlington, Ontario, Canada; [i] Hamilton Health Sciences, McMaster University, Hamilton, Ontario, Canada; [j] Centre Hospitalier Régional de Lanaudière, Joliette, and Université Laval, Québec, Canada; [k] Centre Hospitalier Universitaire de Sherbrooke, Sherbrooke, Quebec, Canada; [l] University Health Network, University of Toronto, Toronto, Ontario, Canada; [m] QE II Health Sciences Centre, Dalhousie University, Halifax, Nova Scotia, Canada; [n] Royal Jubilee Hospital, Victoria, British Columbia, Canada; [o] University of British Columbia, Vancouver, British Columbia, Canada; [p] Centre Hospitalier Universitaire de l'Université de Montréal, Québec, Canada; [q] University of Western Ontario, London, Ontario, Canada; [r] Penticton, British Columbia, Canada; [s] Université de Moncton, Moncton, New Brunswick, Canada; [t] McGill University, Montreal, Québec, Canada; [u] Ottawa Heart Institute, Ottawa, Ontario, Canada; [v] Hôpital Laval, Sainte-Foy, Québec, Canada; [w] Health Sciences Centre, St John's, Newfoundland, Canada

experts on this topic with a mandate to formulate disease-specific recommendations. These recommendations are aimed to provide a reasonable and practical approach to care for specialists and allied health professionals obliged with the duty of bestowing optimal care to patients and families, and can be subject to change as scientific knowledge and technology advance and as practice patterns evolve. The statement is not intended to be a substitute for physicians using their individual judgement in managing clinical care in consultation with the patient, with appropriate regard to all the individual circumstances of the patient, diagnostic and treatment options available and available resources. Adherence to these recommendations will not necessarily produce successful outcomes in every case.

ABSTRACT

The 2013 Canadian Cardiovascular Society Heart Failure Management Guidelines Update provides focused discussions on the management recommendations on 2 topics: (1) exercise and rehabilitation; and (2) surgical coronary revascularization in patients with heart failure. First, all patients with stable New York Heart Association class I-III symptoms should be considered for enrollment in a tailored exercise training program, to improve exercise tolerance and quality of life. Second, selected patients with suitable coronary anatomy should be considered for bypass graft surgery. As in previous updates, the topics were chosen in response to stakeholder feedback. The 2013 Update also includes recommendations, values and preferences, and practical tips to assist the clinicians and health care workers manage their patients with heart failure.

RÉSUMÉ

La mise à jour 2013 des Lignes directrices sur la prise en charge de l'insuffisance cardiaque de la Société canadienne de cardiologie propose 2 sujets de discussion axés sur les recommandations de prise en charge : 1) l'activité physique et la réadaptation; 2) la revascularisation coronarienne chirurgicale chez les patients ayant une insuffisance cardiaque. Premièrement, tous les patients qui ont des symptômes stables correspondant à la classification I-III de la New York Heart Association devraient être jugés admissibles à la participation au programme d'entraînement physique personnalisé afin d'améliorer leur tolérance à l'activité physique et leur qualité de vie. Deuxièmement, les patients dont l'anatomie coronarienne s'y prête devraient être jugés admissibles au pontage. Comme dans les mises à jour précédentes, les sujets ont été choisis en réponse à la rétroaction des parties prenantes. La mise à jour 2013 inclut des recommandations, des valeurs et des préférences, et des conseils pratiques pour aider les cliniciens et les travailleurs de la santé à prendre en charge leurs patients souffrant d'insuffisance cardiaque.

The Canadian Cardiovascular Society (CCS) has published heart failure (HF) management guidelines since 2006[1] as part of a commitment to a multiyear, closed-loop initiative to provide support for the best practice of HF management. The CCS has also implemented the National Heart Failure Workshop Initiative; a series of case-based workshops initiated to discuss how to implement guidelines and identify challenges facing health care providers in HF management. Feedback from these sessions, together with specific solicited input from key stake holders, led to other important topics covered annually in subsequent years. These annual updates have produced a series of evidence-based reports with recommendations and practical tips outlining suggestions for HF management.[2-7]

The constitution and roles of the primary and secondary panels, systematic review strategy, and methods for formulating the recommendations and practical tips are described in detail on the CCS HF Consensus Web site (www.ccsguidelineprograms.ca).

Since 2011, the recommendations have followed the Grading of Recommendations Assessment, Development and Evaluation (GRADE).[8] The GRADE system classifies the quality of evidence as high (further research very unlikely to change confidence in the estimate of effect), moderate (further research likely to have an important impact on confidence in the estimate of effect and may change the estimate), low (further research very likely to have an important impact on confidence in the estimate of effect and likely to change the estimate), and very low (estimate of the effect very uncertain). The GRADE system offers 2 grades of recommendations: "strong" (desirable effects clearly outweigh undesirable effects or clearly do not) and "weak" or "conditional." When trade-offs are less certain, either because of low-quality evidence or because evidence suggests desirable and undesirable effects are closely balanced, weak recommendations become mandatory. Furthermore, since 2012 the Committee has included Values and Preferences,[8] which complement the GRADE system of recommendations. Recommendations will not be given in areas in which the evidence is believed to be inadequate.

The objectives of the 2013 CCS Heart Failure Consensus Update were to provide a review of HF management and recommendations in 2 areas: (1) exercise and rehabilitation; and (2) surgical coronary revascularization.

Rehabilitation and Exercise in HF

Exercise training in patients with HF

RECOMMENDATION

1. We recommend that all patients with stable New York Heart Association (NYHA) class I-III symptoms be considered for enrollment in a supervised tailored exercise training program, to improve exercise tolerance and quality of life (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. This recommendation places a high value on improvements in nonmorbid outcomes and recognizes that not all patients will be able to participate in a structured exercise training program because of patient preferences or availability of resources.

2. We recommend that an assessment of clinical status by a clinician experienced in the management of HF patients be completed before considering an exercise training program (Strong Recommendation, Low-Quality Evidence).

Values and preferences. This recommendation places a high value on clinician's assessment of the clinical stability of a patient and their appropriateness to start exercise, recognizing that most patients will be eligible to participate.

It is well recognized that exercise-based cardiac rehabilitation programs for patients with HF improve exercise capacity, skeletal and respiratory muscle function, quality of life, autonomic function, and biomarkers, and reduce depressive symptoms and cardiovascular risk factors.[9,10] Based on the results of past studies of exercise training, maintenance of physical activity should be considered for stable patients with systolic dysfunction.[11] Patients are considered stable if in the past 6 weeks there is no change in NYHA functional class, and there are no HF hospitalizations, major cardiovascular events, or procedures.

Earlier studies on cardiac rehabilitation in HF included meta-analyses and systematic reviews were inadequately powered to evaluate mortality and morbidity, and often lacking adequate control groups.[12,13] This lack of data led to the **H**eart **F**ailure and **A** **C**ontrolled **T**rial **I**nvestigating **O**utcomes of Exercise Trai**N**ing (HF-ACTION) trial,[11] a randomized controlled trial (RCT) of 2331 patients powered to assess the effects of long-term exercise training on cardiovascular outcomes and safety in patients with chronic HF and reduced left ventricular (LV) ejection fraction (LVEF). Patients in the trial had a median age of 59 years, 28% were women, 37% had NYHA class III or IV symptoms, 51% had an ischemic etiology and median LVEF was 25%. Patients randomized to the exercise training arm first participated in a structured, group-based, supervised exercise program, with a goal of 3 sessions per week for a total of 36 sessions in 3 months (walking, treadmill, or stationary cycling as their primary training mode). Exercise was initiated at 15 to 30 minutes per session at a heart rate (HR) corresponding to resting HR plus 60% of HR reserve (HRR), defined as maximal HR on cardiopulmonary exercise test minus resting HR. After 6 sessions, the duration of the exercise was increased to 30-35 minutes and intensity was increased to resting HR plus 70% of HRR. Patients then began home-based exercise after completing 18 supervised sessions and were to fully transition to home exercise after 36 supervised sessions. The target training regimen for home exercise was 5 times per week for 40 minutes at a HR of 60%-70% of HRR + resting HR. Subjects in the usual care group were not provided with an exercise prescription. All patients received self-management educational materials, in the form of a booklet at the time of enrollment including a recommendation of activity durations of 30 minutes of moderate intensity activity on most days of the week. Cardiopulmonary exercise testing and/or a 6-minute walk test were performed up to 36 months after randomization.

The HF-ACTION trial demonstrated no significant reduction in the primary end point of combined all-cause mortality or hospitalization (hazard ratio, 0.93; 95% confidence interval [CI], 0.84-1.02; $P = 0.13$) or for cardiovascular mortality or hospitalization (hazard ratio, 0.92; 95% CI, 0.83-1.03; $P = 0.14$), and cardiovascular mortality or HF hospitalization (hazard ratio, 0.87; 95% CI, 0.75-1.00; $P = 0.06$).[11] Importantly, exercise training was found to be safe, with no difference in major adverse events. However, in prespecified analyses, after adjusting for 5 highly prognostic baseline variables (duration of the cardiopulmonary exercise test, LVEF, Beck Depression Inventory II score, history of atrial fibrillation or flutter, and HF etiology), exercise training was found to reduce the incidence of all-cause mortality or all-cause hospitalization (the primary end point) by 11% (hazard ratio, 0.89; 95% CI, 0.81-0.99; $P = 0.03$). Moreover, exercise training conferred modest and statistically significant improvements in self-reported health status

compared with usual care without training. Improvements occurred early and persisted over time.[14]

The findings from the HF-ACTION trial are consistent with a recent Cochrane systematic review of 19 RCTs (3647 patients) with HF and systolic dysfunction, including participants in HF-ACTION.[15] Although the rate of all-cause hospitalization was not reduced, exercise training was associated with a 28% reduction in HF-related hospitalizations and improvement in health-related quality of life.

Adherence to exercise

Regular assessment and ongoing re-enforcement for adherence are required, because the beneficial effects of cardiac rehabilitation are rapidly lost if regular training is not maintained, and is a major limitation in research and clinical implementation.[12,16] During the first 3 months of follow-up in the HF-ACTION trial, median exercise time in the exercise training arm was 76 minutes per week (goal, 90 minutes per week).[9] The time increased to 95 minutes per week at 4-6 months after enrollment, decreased to 74 minutes per week at 10-12 months (training goal of 120 minutes per week), and to 50 minutes per week by the third year of follow-up. At every time point, only 30% of patients in the exercise training group exercised at or above the target weekly exercise time. As in other interventions, long-term adherence to an exercise program is challenging for patients with HF.

Safety of exercise

Cardiac rehabilitation for patients with HF has generally emphasized supervised and centre-based programs. Patients are often unsure about beginning an exercise program alone in an unsupervised setting; supervised programs provide reassurance and increase confidence for patients with HF and highlight that exercise is not dangerous. It is acknowledged, however, that long-term cost-effectiveness of supervised cardiac rehabilitation in HF is uncertain. Furthermore, the benefits of cardiac rehabilitation in this population are rapidly lost when exercise training is stopped.[16]

Practical tip. Frequent reinforcement, including letters, phone calls, and home visits, might enhance adherence to exercise.

Identifying and addressing patient-specific barriers might aid in the uptake of exercise for patients.

When a home-based program is initiated, more frequent follow-up visits and occasional supervised "refresher" sessions to answer questions, review concerns, or modify the training program might give patients the guidance needed to ensure that home-based cardiac rehabilitation is successful.

Cardiac rehabilitation programs for patients with recently decompensated or advanced HF

RECOMMENDATION

1. We recommend that gradual mobilization and/or small muscle group strength/flexibility exercises be considered as soon as possible either alone or in combination for patients with NYHA class IV symptoms or recently decompensated HF. This should be considered only in

---

consultation with an experienced HF team (Strong Recommendation, Low-Quality Evidence).

Values and preferences. This recommendation places high value on initiating mobilization and therapy early (even if only limited exercises are prescribed) to prevent further decline of muscle function, improve function during day-to-day activities, and provide a baseline from which to add further exercise modalities.

There is limited evidence on the effectiveness of exercise in patients with severe HF symptoms.[17,18] Motivated and carefully selected patients with recent decompensated HF or with NYHA class IV might be eligible to participate in training programs, under supervision of a team with experience and expertise. Considering that the ultimate goal of cardiac rehabilitation is to encourage patients to become and remain active over the long-term, transition to a home-based program is a safe, viable, and essential modality toward ensuring this goal.[9,17]

Practical tip. Selected patients might benefit from limited exercise therapy, such as lower extremity or inspiratory muscle strengthening, directed toward alleviating symptoms of muscle fatigue.

Cardiac rehabilitation in HF with preserved ejection fraction

The role of cardiac rehabilitation has not been established in patients with HF and preserved LVEF. Data to date appear to indicate that exercise training improves peak oxygen uptake (VO_2) but has no effect on endothelial function and arterial stiffness.[19,20] A recent systematic review[21] that included 3 RCTs, 1 nonrandomized trial, and 1 pre-post study (total, 228 individuals) suggested benefits with respect to improving exercise capacity (between-group mean difference, 3.0 mL/kg/min; 95% CI, 2.4-2.6) and quality of life. The duration of exercise programs and follow-up ranged from 12 to 24 weeks. Considering the multiplicity of comorbid conditions present in this patient population,[22-24] exercise programs using an approach similar to that for patients with HF and low LVEF might be considered.

Practical tip. Until data specific for patients with HF and preserved ejection fraction are available, exercise programs using an approach similar to that for patients with impaired systolic function might be considered in patients with HF and preserved ejection fraction.

Cardiac rehabilitation in patients with cardiac resynchronization therapy and implantable cardioverter defibrillators

In a recent systematic review, 9 studies were identified (1889 patients) using the terms "exercise training," "implantable cardioverter defibrillator (ICD)," and "cardiac rehabilitation."[25] During exercise training over an average of 9.6 weeks of cardiac rehabilitation, 10 ICD therapies including 7 shocks were reported in the 834 patients. Between exercise sessions and during follow-up, 182 events were recorded including 166 shocks. Three studies (2 RCTs) showed that patients in the control group

(sedentary patients) were more prone to ICD discharge than patients who underwent cardiac rehabilitation. In all studies, the patients with an ICD improved their aerobic fitness.

Programming of cardiac resynchronization therapy (CRT) devices might need to be re-evaluated after the pretraining exercise test. Tracking rate, rate response, and loss of biventricular capture during exercise testing should be noted and programming adapted when necessary before starting cardiac rehabilitation.[26] The maximal target HR should be set at least 20 beats per minute less than the ICD intervention HR to avoid inappropriate ICD shocks.[26,27]

Practical tip. Exercise training is safe and not associated with an increased risk of ICD therapy. The maximal target HR should be at least 20 beats less than the ICD intervention HR to avoid inappropriate ICD shocks.

Exercise in frail seniors with HF

HF affects primarily older persons frequently accompanied by functional decline and frailty.[28] As a result, seniors with HF are less likely to receive recommended therapies, including cardiac rehabilitation.[29] Reasons for the lower rates of cardiac rehabilitation among older persons include a lack of encouragement from physicians and uncertainty about the safety, tolerability, and effectiveness of exercise programs.[30] Proportionally fewer seniors were enrolled in RCTs of cardiac rehabilitation, in which median ages ranged from 43 to 72 years,[15] and the median age of participants in HF-ACTION was 59 years.[11] A recent systematic review identified 47 RCTs of exercise interventions in frail seniors with a mean age ranging from 71 to 90 years.[31] Evidence from this review underlines the safety of multicomponent (endurance, resistance, and balance) exercise and its efficacy in improving quality of life and functional outcomes in such patients.

The Hospitalized Elder Life Program is a multifaceted intervention designed to prevent delirium and functional decline in at-risk hospitalized seniors, including those with HF.[32] In addition to protocols to promote sleep, cognitive stimulation, proper vision and hearing, and adequate hydration and nutrition, it also includes early mobilization and bed-mobility exercise in nonambulatory patients. The intervention reduces the incidence of delirium and functional decline, and is also cost-effective.[33,34]

Practical tip. Frail seniors with HF should be offered multicomponent (endurance, resistance, balance) tailored exercise programs appropriate for their comorbidities.

Exercise prescription and exercise modalities in HF

RECOMMENDATION

1. We recommend moderate-intensity continuous aerobic exercise training (eg, brisk walking, jogging, and cycling) at rate of Borg Rating Perceived Exertion (RPE) scale 3-5, 65%-85% maximum HR, or 50%-75% of peak VO_2 in patients with HF (Strong Recommendation, Moderate-Quality Evidence).

> **Values and preferences.** This recommendation places a high value on using commonly available measurements to assist in developing the exercise prescription. The priority is safety, hence, if a patient has a history of ICD discharges, exercise should be avoided if a short loss of consciousness is dangerous (ie, during swimming and activities associated with an increased risk of falling).

Strength training

Aerobic training remains the mainstay of exercise training in patients with HF considering the evidence showing the health benefits of this approach. However, strength/resistance training might be a useful adjunct to aerobic training, particularly for individuals with decreased skeletal muscle mass and wasting. Resistance training can improve daily function, quality of life, skeletal muscle mass and strength, has anti-inflammatory effects, and improves insulin resistance.[35-37] Nordic walking adds a small element of resistance training to an aerobic training activity and has recently been shown in standard cardiac rehabilitation care to improve functional capacity in patients with moderate to severe HF.[38]

The use of light (5-10 pounds) free weights during short training sessions (10-20 repetitions) approximately 2 to 3 times per week (Table 1) can be considered to improve muscle tone and strength of small muscle groups. In patients with greater exercise capacity, a variety of resistance exercises should ideally be prescribed to cover most of the muscular groups. The resistance exercise program should be selected and initially supervised by an experienced exercise therapist.

Practical tip. For strength training, the use of light (5-10 pounds) free weights for 10-20 repetitions 2 to 3 times per week might improve muscle tone and strength.

Aerobic training: continuous and interval training

The following common modes of aerobic activities are to be carried out at moderate intensity: walking, jogging, cycling, or swimming (Table 1). In addition to traditional moderate-intensity continuous exercise training, aerobic interval training has also been proposed as a training modality for stable HF patients.[39-44] Patients with HF often perform their activities of daily living in "spurts" or intervals; interval training might be more physiologically appropriate for subjects with HF. Using this training modality, subjects exercise at fixed intervals lasting seconds to minutes, which are interspersed with recovery or rest intervals of similar duration. Interval training was recently shown to be superior to continuous aerobic training for improving the percentage of symptom-limited aerobic capacity (peak VO$_2$), endothelial function, and reversing LV remodelling among elderly subjects with ischemic cardiomyopathy.[44] Short (15-30 seconds) exercise/rest intervals are better tolerated than longer (60-90 seconds) intervals among patients with cardiovascular disease and HF, while still maintaining a high mean VO$_2$.[39,42] Furthermore, interval training with short exercise/rest intervals is better tolerated than continuous training and allows patients to exercise for longer periods with a decreased sensation of breathlessness.[45] Interval training might also

improve adherence to cardiac rehabilitation considering that it is better tolerated and less monotonous that continuous aerobic training.[46] A recent comparison of intermittent and continuous exercise has demonstrated higher exercise adherence in those randomized to intermittent exercise.[47]

Interval training should be considered and initiated in a supervised setting and considered for all stable HF patients who are already undergoing moderate-intensity continuous aerobic training. Interval training sessions should use short (15-30 seconds) exercise intervals (RPE, 3-5) with rest intervals of equal duration and might last for 15-30 minutes. Interval training is best performed on an ergocycle but might be adapted to other activities including brisk walking and aquatic exercises.

Practical tip. Interval training sessions should use 15- to 30-second exercise intervals (RPE, 3-5) with rest intervals of equal duration and might last 15-30 minutes.

Aerobic exercise training intensity

The exercise intensity to be maintained during exercise should ideally be prescribed on the basis of a cardiopulmonary exercise test results (ie, 50%-75% of peak VO$_2$). Training intensity can also be expressed as resting HR + percentage of maximal HR (%HR$_{max}$) recorded during a stress test. Alternatively, exercise intensity can be expressed relative to a percentage of a person's HRR using Karvonen's formula:

$$HRR = \text{age predicted } HR_{maximum} - \text{resting HR;}$$

$$\text{Training HR} = HRR \times 70\% + \text{resting HR.}^{[45,46]}$$

Exercise intensity can be easily monitored using the RPE scale (between 3 and 5). In summary, percentage of peak VO$_2$, %HR$_{max}$, percentage of HRR or the Borg/RPE scale might be used for exercise prescription.

Practical tip. The RPE scale and %HR$_{max}$ are easier to use in practice than equations based on HRR or measurement of peak VO$_2$.

For patients with ICD, the devices can be programmed with sinus tachycardia discriminators turned on and patients should be encouraged to monitor HR with a portable device.

Surgical Coronary Revascularizatioin in HF

> **RECOMMENDATION**
>
> 1. We recommend that noninvasive imaging for patients with HF be considered to determine the presence or absence of coronary artery disease (CAD) (Strong Recommendation, Moderate-Quality Evidence).
>
> **Values and preferences.** This recommendation places value on identification of CAD, which might identify the cause of HF, have prognostic implications, and require treatments aimed toward secondary vascular prevention.
>
> 2. We recommend that coronary angiography be:
>   i. Performed in patients with HF with ischemic symptoms and who are likely to be good candidates

Table 1. Exercise modalities according to clinical scenario

	Discharged with heart failure	NYHA I-III	NYHA IV
Flexibility exercises	Recommended	Recommended	Recommended
Aerobic exercises			
• Suggested modality	• Selected population only • Supervision by an expert team needed (see text)	• Walk • Treadmill • Ergocycle • Swimming	• Selected population only • Supervision by an expert team needed (see text)

• Intensity		Continuous training:
		  Moderate intensity:
		• RPE scale 3-5, or
		• 65%-85% HR_{max}, or
		• 50%-75% peak VO_2
		  Moderate intensity aerobic interval might be incorporated in selected patients
		• Intervals of 15-30 minutes with an RPE scale of 3-5
		• Rest intervals of 15-30 minutes	

• Frequency		• Starting with 2-3 days per week • Goal: 5 days per week	
• Duration		• Starting with 10-15 minutes • Goal: 30 minutes	
Isometric/resistance exercises			
• Intensity		10-20 repetitions of 5- to 10-pound free weights	
• Frequency		2-3 days per week	

HR_{max}, maximal heart rate; NYHA, New York Heart Association; RPE, rating perceived exertion; VO_2, peak oxygen uptake.

for revascularization (Strong Recommendation, Moderate-Quality Evidence).

ii. Considered in patients with systolic HF, LVEF < 35%, at risk of CAD, irrespective of angina, who might be good candidates for revascularization (Strong Recommendation, Low-Quality Evidence).

iii. Considered in patients with systolic HF and in whom noninvasive coronary perfusion testing yields features consistent with high risk (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. These recommendations place value on the need for coronary angiography to identify CAD amenable to revascularization. Available evidence suggests that coronary revascularization might provide quality of life and prognostic benefits to patients with HF and noninvasive imaging delineating high risk. In particular, patients with systolic HF because of ischemic heart disease might derive clinical benefit from coronary revascularization even in the absence of angina or reversible ischemia.

Nearly 60% of patients with chronic HF suffer from CAD, and approximately 15% of acute decompensated HF cases occur in the setting of an acute coronary syndrome (ACS).[48,49] Despite the coexistence of CAD and HF, few clinical trials have been performed that can inform optimal care for patients with these conditions. Several areas exist in which high grade evidence is lacking, such as coronary revascularization in the setting of HF with preserved ejection fraction. Although ample evidence exists to support percutaneous coronary intervention (PCI) in patients with HF due to ACS,[50] there is limited evidence to support its use in the setting of chronic HF to reduce adverse clinical outcomes.[51]

Diagnosis of CAD in patients with HF

The approach to the assessment of CAD in patients with HF is illustrated in Figure 1. Identification of the etiology of HF has significant therapeutic and prognostic implications. CAD is present in more than 60% of patients with HF and the absence of a history of myocardial infarction or angina is insufficient to rule out CAD as the etiology for HF.[52,53] Presence of CAD is associated with a poor prognosis and has implications regarding revascularization or device therapy. An important distinction must be made between the presence of CAD, which might coexist with HF but not be the primary etiology of the syndrome of HF and ischemic cardiomyopathy, when the principal etiology for HF is CAD. Although CAD might contribute as a comorbid factor in HF, it is largely treated for symptoms and prevention only, whereas ischemic cardiomyopathy is a distinct condition defined by the combination of LV systolic dysfunction due to multivessel CAD or clear evidence of a previous ischemic insult to a large portion of the myocardium and its subsequent remodelling.[53] Surgical revascularization for ischemic cardiomyopathy might have prognostic and symptomatic effects.[54] As such, it is important to determine the presence and effect of CAD for all patients with HF.

Three types of ischemic abnormalities portend differential responses to medical therapy (such as β-blockade) and revascularization.[52] These include: (1) reversible ischemic myocardium; (2) hibernating, or viable myocardium, a state in which segments of the myocardium exhibit abnormalities of contractile function[55]; and (3) nonviable myocardium. In theory, these types of myocardium behave differently. Ischemic myocardium is likely to improve function after revascularization (> 80% likelihood), and hibernating and viable myocardium states are less likely (40%-50% likelihood) to improve measured according to segmental wall motion.[56-59] A body of evidence supports the concept that patients with reversible segments experience the best
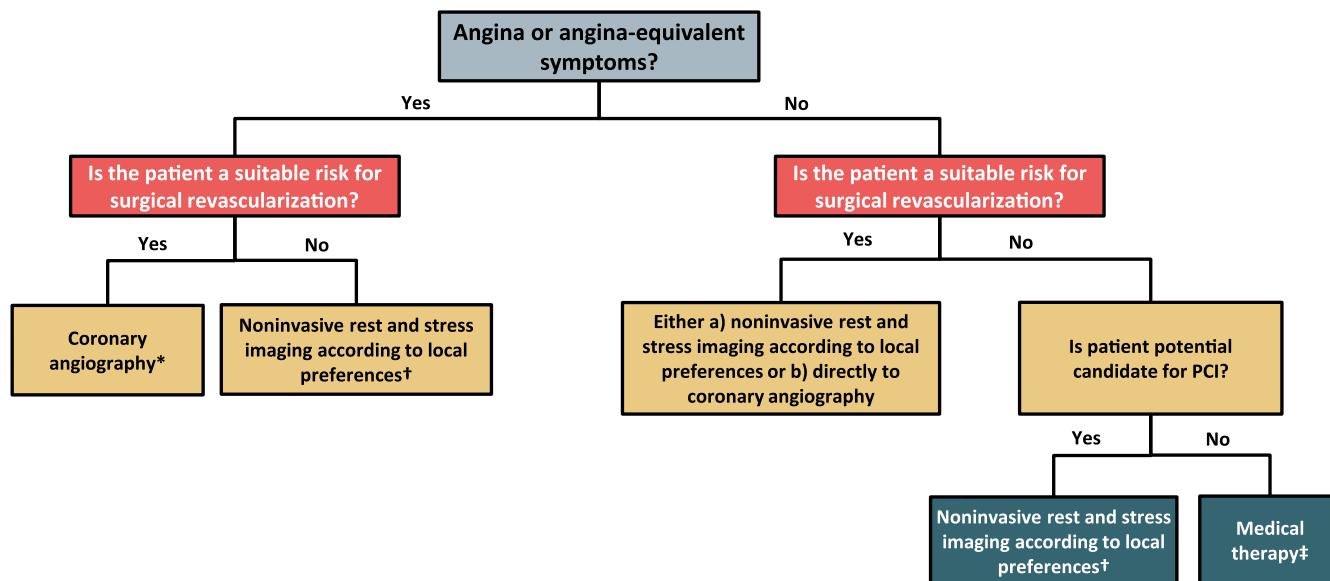
Figure 1. Approach to assessment for coronary artery disease in patients with heart failure. All patients with heart failure are expected to undergo noninvasive measurement of systolic function (not included in this algorithm). *Some centres might additionally perform noninvasive imaging, especially when coronary anatomy is not optimal. †If imaging indicates features of high risk, progression to coronary angiography is expected. ‡Noninvasive imaging might be performed in certain centres for risk stratification or diagnosis. PCI, percutaneous coronary intervention.

clinical and functional outcomes after surgical revascularization, followed by those with hibernating/viable segments.[59-61] As such, the presence of reversible or viable myocardium might affect the decision to proceed to a revascularization procedure, but will not be the sole determining factor.[62]

Imaging for reversible ischemia as a guide to the presence of CAD

Although noninvasive imaging is increasingly used to determine the presence of CAD, coronary angiography is still the gold standard for diagnosis. It has been shown to lead to an etiological reclassification in HF in up to 25% of cases.[48,63]

Several noninvasive tests might allude to the presence or absence of CAD. These include exercise or pharmacologic stress perfusion imaging using cardiac magnetic imaging (CMR), nuclear imaging, positron emission tomography (PET), or echocardiography. The diagnostic accuracy for presence of CAD has been reported from 70% to 90% using nuclear stress imaging.[58,62,64] Sensitivity is typically very high (> 90%) and specificity might be as low as 60%, with PET demonstrating the higher sensitivity and stress echocardiography the highest specificity.[59-62] Stress CMR is an emerging modality that might be used as a screening test to define the need for coronary angiography. However, it is most commonly used for determination of the functional implications of known CAD. Patterns of delayed gadolinium enhancement are usually able to differentiate between ischemic and nonischemic cardiomyopathy.[65] Coronary computed tomography and calcium scoring might accurately identify the presence of CAD, but at present do not provide clinically useful functional data and are of uncertain value.

Imaging for hibernating myocardium

Hibernating myocardium is underperfused, hypocontractile, but viable tissue that has the potential for functional recovery with restoration of normal blood flow.[61,66] Therefore, hibernating myocardium can only be defined with certainty after revascularization. Multiple imaging modalities are used to identify the presence of hibernating myocardium and are grouped into 3 categories. First, nuclear imaging studies test metabolic and cellular integrity and PET scanning is considered to be the gold standard. The PET and Recovery Following Revascularization-2 trial investigators reported improved outcomes after revascularization in patients with > 7% hibernating myocardium.[67] Second, CMR might define scarred and fibrotic myocardium with limited potential for recovery. Kim et al. reported a > 75% potential for functional recovery in the absence of scar, and < 2% recovery in segments with more than 75% scar thickness.[64] Finally, dobutamine stress echocardiography (DSE) might be used to measure contractile reserve. The **V**iability **I**dentification With **D**obutamine **A**dministration (VIDA) study investigators reported lower mortality in patients with viable myocardium treated with revascularization compared with those treated medically using DSE.[68] CE-MARC (**C**linical **E**valuation of **M**agnetic **R**esonance Imaging in **C**oronary Heart Disease) is the largest, prospective, real world evaluation of CMR and has established CMR's high diagnostic accuracy in CAD and CMR's superiority over single-photon emission computed tomography.[69] Comparative studies between these different imaging modalities report higher sensitivity with PET, and higher specificity with CMR and DSE.

Despite the observational evidence base evaluating these therapies, few adequately powered randomized studies exist. The **M**edical **I**maging **T**rial **Ne**twork of **C**anada (MITNEC) is studying imaging applications in clinical research and practice (www.mitnec.org). A Canadian initiative, the IMAGE-HF Project I-A: Cardiac Imaging in Ischemic Heart Failure (AIMI-HF) (ClinicalTrials.gov identifier NCT01288560) has a primary objective to compare the effect of imaging strategies on the clinical outcomes. Although the myocardial perfusion

literature is robust in terms of predicting surgical risk and the likelihood of experiencing clinical improvement after surgical revascularization, data from the only randomized trial of surgery in ischemic cardiomyopathy failed to demonstrate the utility in identification of patients who would benefit from a surgical strategy.[70]

Practical tip. Several noninvasive methods for detection of CAD are in widespread use, including DSE, perfusion CMR, cardiac PET testing, and nuclear stress imaging. Local factors (availability, price, expertise, practice patterns) will determine the optimal strategy for imaging.

Noninvasive imaging modalities might provide critical information such as the amount and degree of ischemic or hibernating myocardium, and might be used to determine the likelihood of regional and global improvement in LV systolic function after revascularization.

Patients with HF and reduced LVEF are more likely to experience significant improvement in LVEF after successful coronary revascularization if they demonstrate:

- Reversible ischemia or a large segment of viable myocardium (> 30% of the left ventricle) in nuclear stress testing/viability study;
- Reversible ischemia or > 7% hibernating myocardium on PET scanning;
- Reversible ischemia or > 20% of the left ventricle shown as viable using DSE;
- < 50% wall thickness scarring shown by late gadolinium enhancement on CMR imaging.

Disease management, referral, and perioperative care

RECOMMENDATION

1. We recommend that the decision to refer patients with HF and ischemic heart disease for coronary revascularization should be made on an individual basis and in consideration of all cardiac and noncardiac factors that affect procedural candidacy (Strong Recommendation, Low-Quality Evidence).
2. We recommend that efforts be made to optimize medical status before coronary revascularization, including optimizing intravascular volume medical therapy (Strong Recommendation, Low-Quality Evidence).
3. We recommend that performance of coronary revascularization procedures in patients with chronic HF and reduced LVEF be undertaken with a medical-surgical team approach with experience and expertise in high-risk interventions (Strong Recommendation, Low-Quality Evidence).

Values and preferences. This recommendation reflects the preference that high-risk revascularization is best preformed in higher volume centres with significant experience, and known, published outcomes.

Care of the patient with HF is best accomplished using an interdisciplinary team approach including a primary care physician, nurse with disease management and HF care skills, and specialist with experience and expertise in HF care. Selection of revascularization will depend on many factors in addition to LVEF and coronary anatomy, such as comorbid conditions (especially frailty and renal dysfunction), access to rehabilitative services, caregiver support, care preferences, and goals of care. Patients with HF are subject to between 2 and 5 times increased procedural risks of elective procedures such as coronary revascularization, whether surgical or percutaneous, compared with similar patients without HF.[2,71,72] The risk is substantially increased in the settings of nonelective surgery or in the presence of decompensated HF.[72] As such, careful consideration and control of all concomitant medical conditions including optimization of HF are essential before revascularization. These strategies are thought to improve a given patient's clinical status, functional status, and LV function thereby reducing perioperative risk and, in some cases, mitigating the need for concomitant surgical procedures.

RCTs that examined device therapy, CRT ICD mandated that all planned revascularization be completed at least 3 months before enrollment into the trial.[73-75] If a decision to refer for revascularization is made, the team accepting care should include medical and surgical members with expertise and experience in revascularization of patients with chronic HF. For patients with advanced HF, this might require referral to a centre that has an established program and expertise in management of advanced HF, mechanical circulatory support, or cardiac transplantation.[6,7] A multidisciplinary team should be involved with the care from the planning and assessment phase before surgery through the course of hospitalization with a coordinated plan for transitioning to the chronic care setting.

Many clinicians believe that patients who present with HF characterized by significant volume retention will benefit from a period of diuresis which might require adjunctive preoperative inotropic support. Some clinicians advocate for preoperative intra-aortic balloon pump support. A recent meta-analysis of intra-aortic balloon pump support suggested a modest clinical benefit but potential harm.[76] Postoperatively, reinstitution of standard HF therapies should be undertaken in a measured manner. In addition, significant volume overload is often present immediately after surgical revascularization, necessitating concomitant diuresis and increased risk of toxicity associated with medication uptitration.

Practical tip. Assessment for advanced HF therapies, by an appropriate team, should be performed before the revascularization procedure in any patient with advanced HF.

Surgical revascularization for patients with CAD and HF

RECOMMENDATION

1. We recommend consideration of coronary artery bypass surgery for patients with chronic ischemic cardiomyopathy, LVEF < 35%, graftable coronary arteries, and who are otherwise suitable candidates for surgery, irrespective of the presence of angina and HF symptoms to improve quality of life, and reduce rates of

---

cardiovascular death and hospitalization (Strong Recommendation, Moderate-Quality Evidence).

2. We suggest consideration of PCI for patients with HF and limiting symptoms of cardiac ischemia, and for whom coronary artery bypass grafting (CABG) is not considered appropriate (Conditional Recommendation, Low-Quality Evidence).

3. We recommend against routine performance of surgical ventricular restoration for patients with HF undergoing CABG who have akinetic or dyskinetic segments (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. These recommendations are based on data from RCTs on CABG and surgical ventricular restoration in patients with reduced systolic function and CAD. The recommendation on PCI is based on clinical need rather than RCT trial data.

The approach to the decision of coronary revascularization in patients with HF is illustrated in Figure 2. CABG surgery is indicated in adult patients with symptoms of angina, a history of stable HF in association with LV dysfunction (LVEF < 35%), graftable coronary arteries, and who have an otherwise good life expectancy. This recommendation was based on historical data from earlier landmark clinical trials comparing medical and surgical therapy, which identified a survival benefit with CABG in patients with triple vessel CAD along with ventricular dysfunction.[77-80] The Coronary Artery Surgery Study enrolled 780 patients with stable ischemic heart disease between 1975 and 1979. Randomization was stratified initially into 3 groups: patients with angina and LVEF ≥ 50%; patients with angina and LVEF < 50%; and asymptomatic patients within 6 months after myocardial infarction.[77] Randomization was further stratified by the number of diseased vessels for the first 2 groups and by the number of diseased vessels and ejection fraction in the third group. Although survival was similar in the medical and surgical groups overall at 10 years (79% vs 82%; $P = 0.25$), survival was greater in patients with angina and LVEF < 50% (59% vs 80%; $P = 0.01$) and in patients with LVEF < 50% (61% vs 79% 10-year survival) treated with surgery. This conclusion was based on only 160 patients with LVEF < 50% of whom there were few patients with an ejection fraction < 35%. A similar result was seen in the VA Cooperative Study (N = 686; 595 patients without left main stenosis; 55% of patients had LV dysfunction [LVEF < 50% or regional dysfunction of < 25% of the myocardium]).[78] At 7 years, survival was 63% in medically treated patients compared with 74% in CABG-treated patients ($P = 0.049$). This benefit was attenuated by 11 years (49% vs 53%; $P = 0.25$). The other major trial was conducted by the European Coronary Surgery Study group, and enrolled 767 male patients with normal LV function.[79] This study identified an overall survival benefit in patients randomized to CABG (92.4% vs 83.1%; $P = 0.0001$ at 5 years; 70.6% vs 66.7% at 12 years; $P = 0.04$). In a systematic review and meta-analysis, relative survival advantage was similar in patients with normal or reduced LV function (odds ratio, 0.61 vs 0.59), although the absolute

survival advantage was greater in patients with decreased LV function.[80] An important limitation of these earlier studies is the limited representation of patients with significant LV dysfunction, limited medical and device therapy, and very few patients had symptomatic HF and the results might not be applicable to the contemporary HF population.

More recent studies have included PCI and surgical revascularization together to assess the potential benefit of revascularization compared with medical therapy. A meta-analysis of observational studies (3088 patients, average LVEF of 32%) to determine the importance of either early or late perfusion viability, shown using thallium imaging, PET, or DSE testing before revascularization.[62] In patients with viability, annual mortality was 3.2% in the revascularized patients compared with 16.3% in medically treated patients ($P < 0.0001$). In patients without viability, revascularization was associated with similar annual mortality (7.7%) compared with medically treated patients (6.2%). In an observational study from Canada,[81] 4228 patients with a history of HF were treated medically (n = 1690) or with PCI or surgical revascularization (n = 2538). Only 23% of patients had a normal LVEF and 25% had a LVEF < 35%. Revascularization using PCI or surgery was associated with a 48% lower mortality in adjusted analyses. Surgery and PCI provided a similar association of a lower mortality in unadjusted analyses.

A major concern regarding surgical revascularization in patients with LV dysfunction is a greater rate of operative mortality. A meta-analysis of 26 observational studies (3621 patients) with a preoperative LVEF < 35% showed an operative mortality of 5.4%.[82] The 2 risk calculators for surgical mortality have been recently updated: Euroscore II (http://www.euroscore.org/calc.html) and the STS score (http://riskcalc.sts.org/STSWebRiskCalc273/de.aspx). In summary, a 70-year-old man with preserved renal function, history of hypertension, mild chronic lung disease, remote myocardial infarction, and NYHA III symptoms undergoing elective, isolated CABG, the estimated risk of death is 0.83% with LVEF of 50% and 1.28% with an LVEF of 25%, according to the STS risk calculator, and 1.07% and 2.37%, respectively, using the Euroscore II risk calculator.

It is unclear whether off-pump surgery is associated with lower operative mortality than traditional on-pump CABG for patients with HF. A Cochrane review of 86 RCTs (10,716 participants) compared on- and off-pump CABG.[83] Overall, off-pump surgery was associated with a greater risk for 30-day mortality (3.7% vs 3.4%; relative risk, 1.24; 95% CI, 1.01-1.53; $P = 0.04$). The applicability of this review to patients with HF and impaired systolic function is unclear.

The STICH trial

The **S**urgical **T**reatment of **Isc**hemic **H**eart Failure (STICH) trial sought to address 2 hypotheses: (1) does CABG improve survival in addition to optimal medical therapy for patients with HF and CAD (LVEF < 35%) who are acceptable candidates for cardiac surgery; and (2) does the addition of surgical ventricular reconstruction (SVR) of an akinetic/dyskinetic anterior wall provide better outcomes than isolated CABG for eligible individuals.[84] This study evaluated patients with ischemic cardiomyopathy with or without HF symptoms and randomized them into 3 groups, namely,
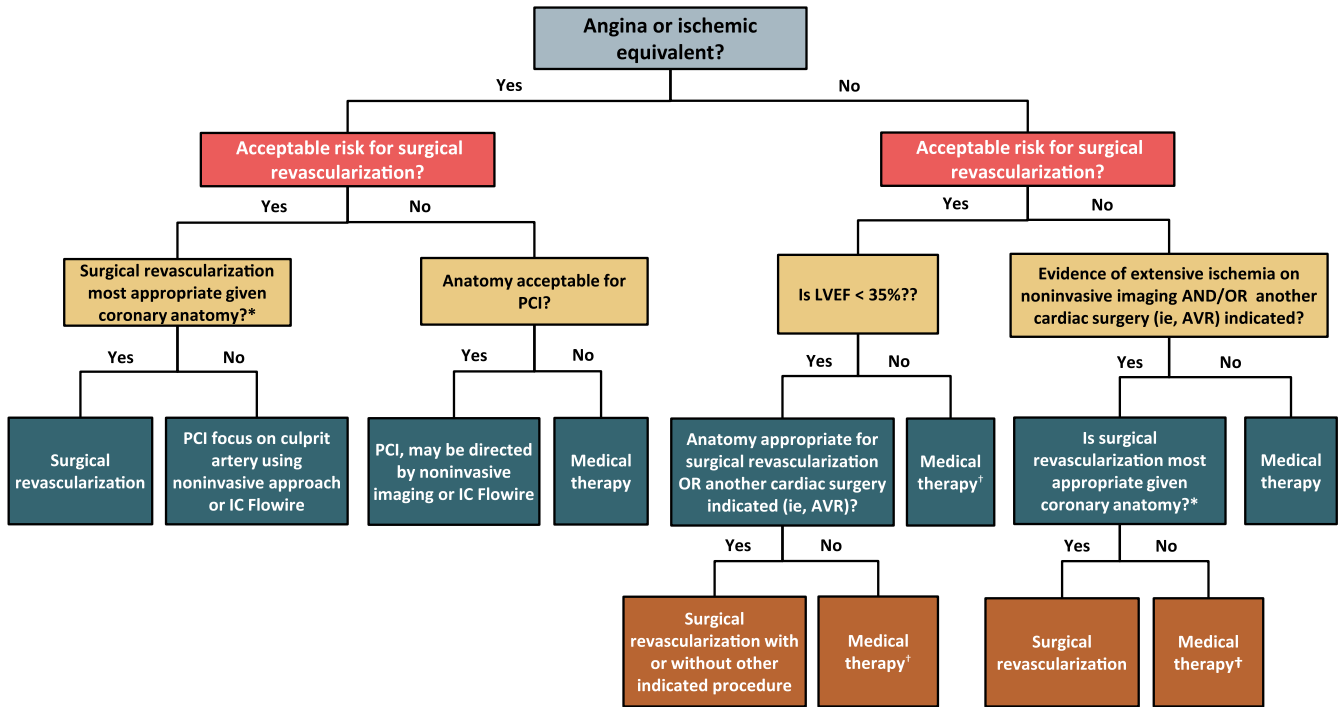
Figure 2. Decision regarding coronary revascularization in heart failure. It is recommended that surgical consultation be performed very early in this process. *Coronary anatomy suitable for CABG includes multivessel disease > 70% stenosis, left main stem stenosis > 50%, or diabetes with left anterior descending artery stenosis > 70%. †In selected cases in which there is noninvasive imaging evidence of extensive cardiac ischemia, PCI might be considered. AVR, aortic valve replacement; CABG, coronary artery bypass grafting; IC, intracoronary; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention.

optimal medial therapy and CABG alone, CABG plus the SVR procedure, or neither procedure.

For the first hypothesis, 1212 patients were randomized to medical therapy alone or in combination with CABG, of whom 64% showed evidence of inducible ischemia on noninvasive testing.[85] At a median follow-up of 56 months, 17% of patients allocated to medical therapy crossed over to the surgical arm, and 91% of the surgically allocated group actually underwent CABG within 1 year. For the primary outcome, 41% of those allocated to medical therapy died, compared with 36% in the CABG arm (hazard ratio, 0.86; 95% CI, 0.72-1.04; $P = 0.12$). The secondary end point of death from any cause or hospitalization for cardiovascular causes occurred in 411 patients (68%) in the medical therapy group and 351 (58%) in the CABG group (hazard ratio, 0.74; 95% CI, 0.64-0.85; $P < 0.001$). Thus, for every 10 patients who underwent CABG in this study, 1 subsequent death or hospitalization was prevented over the course of 4.5 years. When analyzed on a "per protocol" basis whereby patients in the medical arm who received surgery within a year of randomization were grouped with patients randomized to surgery, the hazard ratio for the primary end point of all-cause mortality in the surgical arm was 0.76 ($P < 0.001$).[86]

For the second hypothesis, 1000 patients who were eligible for SVR in addition to CABG were randomly allocated to receive CABG alone or in combination with SVR surgery. This group (mean age, 62 years; median LVEF 28%; end systolic volume index of 82 mL/m^2) experienced only a 5% 30-day mortality with a 17-mL/m^2 reduction in LV volume. Despite the excellent technical surgical result, there was no

reduction in mortality or the composite end point of mortality plus repeat hospitalization. Subgroup analyses failed to identify any particular group that might benefit from SVR.[87] One limitation of the STICH second hypothesis was that many patients in whom SVR might have derived benefit were not included in the trial and instead might have received SVR as primary therapy. Conversely, many patients who underwent SVR outside of the trial might have been at much lower or higher risk.

Regardless of how STICH is interpreted, several technical considerations were incorporated into patient selection. First, the presence of at least 1 good coronary target with a critical proximal lesion was required. This did not have to be the left anterior descending artery and indeed, some patients might have been subjected to SVR in the presence of viable, but ischemic inferior and lateral walls. Second, the presence of mitral insufficiency increased the risk of surgical intervention, particularly when accompanied by significant pulmonary hypertension and concomitant tricuspid insufficiency.

More recently, there has been increased attention to the repair of functional mitral regurgitation (MR) in patients with ischemic heart disease. These patients tend to present with mild to moderate systolic dysfunction and restricted mitral leaflets.[87] The data on such patients are conflicting in relation to the increased morbidity and mortality when compared with their peers without functional MR. A single blind randomized trial assessed mitral repair in patients with mild to moderate systolic dysfunction and moderate MR.[88] In this study, the degree of MR was significantly reduced in the group allocated to CABG plus mitral repair compared with those with CABG

alone. The primary end point, peak VO$_2$, was significantly increased by more than 2.0 mL/kg/min. However, no significant reduction in clinical events or mortality was noted in this small trial, and the dominant clinical feature of the study population was severe MR rather than HF per se. Additional data are needed before consideration of routine mitral repair can be recommended. Similarly, discussion of percutaneous methods for reduction of MR in the HF population is premature and beyond the scope of this update.

Practical tip. In the setting of HF, angina and single territory CAD, PCI might be the treatment of first choice. However, PCI has not been shown to improve outcomes for patients with chronic stable HF, irrespective of underlying anatomy.

In contrast to the chronic stable patient with HF, urgent directed culprit vessel angioplasty continues to be the revascularization modality of choice for patients with ACS complicated by HF.

In highly selected cases, patients with advanced HF symptoms in association with large areas of dyskinetic and nonviable myocardium might experience clinical improvement with SVR or similar type procedures, when performed by experienced surgeons.

Mitral valve repair, when used concomitantly during CABG, might lead to clinical improvement in symptoms of HF in highly selected cases.

Device considerations in HF patients after cardiac surgery

RECOMMENDATION

1. We recommend that after successful cardiac surgery, patients with HF undergo assessment for implantable cardiac devices within 3-6 months of optimal treatment (Strong Recommendation, High-Quality Evidence).
2. We recommend that patients with implantable cardiac devices in situ should be evaluated for programming changes before surgery and again after surgery, in accordance with existing CCS recommendations (Strong Recommendation, Low-Quality Evidence).
3. We recommend that after successful cardiac surgery, all patients be referred to a local cardiac rehabilitation program (Strong Recommendation, High-Quality Evidence).

Values and preferences. These recommendations reflect our support of and conformity with pre-existing cardiac device and rehabilitation guidelines statements.

The rationale and evidence supporting the use of devices, ICD and CRT, in patients with HF and reduced ejection fraction have been addressed in detail in previous HF and CRT guideline updates.[7,73]

Although no studies to date have directly assessed the optimal timing of ICD implantation in the setting of ischemic cardiomyopathy, evidence from primary prevention trials suggests that ICDs do not confer an overall mortality benefit when implanted during, or immediately after, an acute event or revascularization.[89] The CABG Patch trial was designed to

assess whether an ICD is associated with additional survival benefit in patients at high risk for sudden cardiac death.[90] This study enrolled more than 1000 patients with LVEF ≤ 35% and abnormal signal averaged electrocardiographs undergoing elective CABG surgery. Patients were randomized to ICD implantation at the time of CABG surgery or to CABG alone; after a mean follow-up of 32 months, the overall mortality rate between groups was similar, and no survival advantage was seen in the ICD group. These findings were essentially mirrored in other studies of ICD after acute myocardial infarction and reinforce the role of ICD therapy to be one for chronic LV dysfunction.[89,91,92] Most clinical trials that have shown a survival benefit of ICDs in ischemic cardiomyopathy have included patients more than 1 month after infarct, irrespective of revascularization status,[74,93] or have included very high-risk patients with previous history of sudden cardiac death.[94]

After revascularization, the risk of sudden cardiac death continues to increase over time,[95] while systolic function might not improve substantially,[96] posing a challenge in defining the optimal timing for ICD therapy. In the STICH trial,[87] fewer than 20% of patients underwent device implantation over the course of the study, and the interaction between revascularization, timing of implantation, and survival is therefore difficult to assess from the available data. It is reasonable to consider referring patients with a low LVEF to physicians experienced with the management of HF for medical optimization and appropriate referral for device therapy.

Similarly, the optimal timing for CRT implantation in suitable candidates with ischemic cardiomyopathy has not been well defined. Key clinical trials demonstrating a mortality benefit with CRT excluded patients with a recent (1-6 months) myocardial infarction or revascularization procedure.[75,97,98] However, data from observational studies provide a rationale for considering epicardial LV lead placement at the time of CABG surgery in patients who might otherwise have an indication for CRT. Transvenous LV lead delivery via the coronary sinus is technically not feasible in approximately 10% of cases[99]; surgical lead placement can overcome anatomical limitations imposed by the coronary sinus, with acceptable long-term lead performance and rates of clinical response similar to conventional transvenous implantation.[100] Additionally, surgical revascularization might not have any effect on dyssynchrony, which is associated with a worse prognosis.[101] Data from 1 randomized trial[102] suggest that CRT using an epicardial lead implanted concomitantly with CABG is associated with improved systolic function and survival compared with CABG alone in patients with poor systolic function and evidence of preoperative device candidacy. Therefore, epicardial LV lead placement might be considered in selected patients undergoing surgical revascularization for ischemic cardiomyopathy who are likely to remain candidates for CRT after surgery.

Perioperative management of existing devices remains an important component of care. In keeping with existing guidelines, which state device deactivation is necessary before any procedure in which electrocautery, or potential for electrical interference with the device might occur.[103] Postoperatively, re-establishment of appropriate device threshold determination and programming are recommended. The

Canadian Association of Cardiac Rehabilitation and CCS joint position statement includes routine cardiac rehabilitation for patients with HF who successfully complete CABG surgery.[104]

Practical tip. During surgical revascularization, consideration can be given to implantation of epicardial LV leads to facilitate biventricular pacing in eligible patients who might be candidates for CRT, especially if the coronary sinus anatomy is known to be unfavourable for lead placement.

Patients with HF and who have successful surgical coronary revascularization can be referred to a cardiac rehabilitation program.

Funding Sources

References

1. Arnold JM, Liu P, Demers C, et al. Canadian Cardiovascular Society consensus conference recommendations on heart failure 2006: diagnosis and management. Can J Cardiol 2006;22:23-45.

2. Arnold JM, Howlett JG, Dorian P, et al. Canadian Cardiovascular Society Consensus Conference recommendations on heart failure update 2007: prevention, management during intercurrent illness or acute decompensation, and use of biomarkers. Can J Cardiol 2007;23:21-45.

3. Howlett JG, McKelvie RS, Arnold JM, et al. Canadian Cardiovascular Society Consensus Conference guidelines on heart failure, update 2009: diagnosis and management of right-sided heart failure, myocarditis, device therapy and recent important clinical trials. Can J Cardiol 2009;25:85-105.

4. Howlett JG, McKelvie RS, Costigan J, et al. The 2010 Canadian Cardiovascular Society guidelines for the diagnosis and management of heart failure update: heart failure in ethnic minority populations, heart failure and pregnancy, disease management, and quality improvement/assurance programs. Can J Cardiol 2010;26:185-202.

5. Malcom J, Arnold O, Howlett JG, et al. Canadian Cardiovascular Society Consensus Conference guidelines on heart failure—2008 update: best practices for the transition of care of heart failure patients, and the recognition, investigation and treatment of cardiomyopathies. Can J Cardiol 2008;24:21-40.

6. McKelvie RS, Moe GW, Cheung A, et al. The 2011 Canadian Cardiovascular Society heart failure management guidelines update: focus on sleep apnea, renal dysfunction, mechanical circulatory support, and palliative care. Can J Cardiol 2011;27:319-38.

7. McKelvie RS, Moe GW, Ezekowitz JA, et al. The 2012 Canadian Cardiovascular Society heart failure management guidelines update: focus on acute and chronic heart failure. Can J Cardiol 2013;29:168-81.

8. McCormack JP, Loewen P. Adding "value" to clinical practice guidelines. Can Fam Physician 2007;53:1326-7.

9. Vanhees L, Geladas N, Hansen D, et al. Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular risk factors: recommendations from the EACPR. Part II. Eur J Prev Cardiol 2012;19:1005-33.

10. Piepoli MF. Exercise training in heart failure. Curr Heart Fail Rep 2006;3:189-96.

11. O'Connor CM, Whellan DJ, Lee KL, et al. Efficacy and safety of exercise training in patients with chronic heart failure: HF-ACTION randomized controlled trial. JAMA 2009;301:1439-50.

12. Piepoli MF, Davos C, Francis DP, Coats AJ. Exercise training meta-analysis of trials in patients with chronic heart failure (ExTra-MATCH). BMJ 2004;328:189.

13. Smart N, Marwick TH. Exercise training for patients with heart failure: a systematic review of factors that improve mortality and morbidity. Am J Med 2004;116:693-706.

14. Flynn KE, Pina IL, Whellan DJ, et al. Effects of exercise training on health status in patients with chronic heart failure: HF-ACTION randomized controlled trial. JAMA 2009;301:1451-9.

15. Davies EJ, Moxham T, Rees K, et al. Exercise training for systolic heart failure: Cochrane systematic review and meta-analysis. Eur J Heart Fail 2010;12:706-15.

16. Willenheimer R, Rydberg E, Cline C, et al. Effects on quality of life, symptoms and daily activity 6 months after termination of an exercise training programme in heart failure patients. Int J Cardiol 2001;77:25-31.

17. Goodlin SJ. Palliative care in congestive heart failure. J Am Coll Cardiol 2009;54:386-96.

18. McKelvie RS. Exercise training in patients with heart failure: clinical outcomes, safety, and indications. Heart Fail Rev 2008;13:3-11.

19. Fujimoto N, Prasad A, Hastings JL, et al. Cardiovascular effects of 1 year of progressive endurance exercise training in patients with heart failure with preserved ejection fraction. Am Heart J 2012;164:869-77.

20. Kitzman DW, Brubaker PH, Herrington DM, et al. Effect of endurance exercise training on endothelial function and arterial stiffness in older patients with heart failure and preserved ejection fraction: a randomized, controlled, single-blind trial. J Am Coll Cardiol 2013;62:584-92.

21. Taylor RS, Davies EJ, Dalal HM, et al. Effects of exercise training for heart failure with preserved ejection fraction: a systematic review and meta-analysis of comparative studies. Int J Cardiol 2012;162:6-13.

22. Heran BS, Chen JM, Ebrahim S, et al. Exercise-based cardiac rehabilitation for coronary heart disease. Cochrane Database Syst Rev 2011;CD001800.

23. McKelvie RS, Komajda M, McMurray J, et al. Baseline plasma NT-proBNP and clinical characteristics: results from the irbesartan in heart failure with preserved ejection fraction trial. J Card Fail 2010;16:128-34.

24. Yusuf S, Pfeffer MA, Swedberg K, et al. Effects of candesartan in patients with chronic heart failure and preserved left-ventricular ejection fraction: the CHARM-Preserved Trial. Lancet 2003;362:777-81.

25. Isaksen K, Morken IM, Munk PS, Larsen AI. Exercise training and cardiac rehabilitation in patients with implantable cardioverter defibrillators: a review of current literature focusing on safety, effects of exercise training, and the psychological impact of programme participation. Eur J Prev Cardiol 2012;19:804-12.

26. Vanhees L, Rauch B, Piepoli M, et al. Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular disease (part III). Eur J Prev Cardiol 2012;19:1333-56.

27. Vanhees L, Kornaat M, Defoor J, et al. Effect of exercise training in patients with an implantable cardioverter defibrillator. Eur Heart J 2004;25:1120-6.

28. Harkness K, Heckman GA, McKelvie RS. The older patient with heart failure: high risk for frailty and cognitive impairment. Expert Rev Cardiovasc Ther 2012;10:779-95.

29. Dafoe W, Arthur H, Stokes H, Morrin L, Beaton L. Universal access: but when? Treating the right patient at the right time: access to cardiac rehabilitation. Can J Cardiol 2006;22:905-11.

30. Grace SL, Shanmugasegaram S, Gravely-Witte S, et al. Barriers to cardiac rehabilitation: does age make a difference? J Cardiopulm Rehabil Prev 2009;29:183-7.

31. Theou O, Stathokostas L, Roland KP, et al. The effectiveness of exercise interventions for the management of frailty: a systematic review. J Aging Res 2011;2011:569194.

32. Inouye SK, Bogardus ST Jr, Charpentier PA, et al. A multicomponent intervention to prevent delirium in hospitalized older patients. N Engl J Med 1999;340:669-76.

33. Rizzo JA, Bogardus ST Jr, Leo-Summers L, et al. Multicomponent targeted intervention to prevent delirium in hospitalized older patients: what is the economic value? Med Care 2001;39:740-52.

34. Rubin FH, Neal K, Fenlon K, Hassan S, Inouye SK. Sustainability and scalability of the hospital elder life program at a community hospital. J Am Geriatr Soc 2011;59:359-65.

35. Cheetham C, Green D, Collis J, Dembo L, O'Driscoll G. Effect of aerobic and resistance exercise on central hemodynamic responses in severe chronic heart failure. J Appl Physiol 2002;93:175-80.

36. Conraads VM, Beckers P, Bosmans J, et al. Combined endurance/resistance training reduces plasma TNF-alpha receptor levels in patients with chronic heart failure and coronary artery disease. Eur Heart J 2002;23:1854-60.

37. Singh MA, Ding W, Manfredi TJ, et al. Insulin-like growth factor I in skeletal muscle after weight-lifting exercise in frail elders. Am J Physiol 1999;277:E135-43.

38. Keast ML, Slovinec D'Angelo ME, Nelson CR, et al. Randomized trial of Nordic walking in patients with moderate to severe heart. Can J Cardiol 2013;29:1470-6.

39. Guiraud T, Juneau M, Nigam A, et al. Optimization of high intensity interval exercise in coronary heart disease. Eur J Appl Physiol 2010;108:733-40.

40. Guiraud T, Nigam A, Juneau M, et al. Acute responses to high-intensity intermittent exercise in CHD patients. Med Sci Sports Exerc 2011;43:211-7.

41. Meyer P, Normandin E, Gayda M, et al. High-intensity interval exercise in chronic heart failure: protocol optimization. J Card Fail 2012;18:126-33.

42. Meyer P, Gayda M, Juneau M, Nigam A. High-intensity aerobic interval exercise in chronic heart failure. Curr Heart Fail Rep 2013;10:130-8.

43. Moholdt T, Aamot IL, Granoien I, et al. Long-term follow-up after cardiac rehabilitation: a randomized study of usual care exercise training versus aerobic interval training after myocardial infarction. Int J Cardiol 2011;152:388-90.

44. Wisloff U, Stoylen A, Loennechen JP, et al. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. Circulation 2007;115:3086-94.

45. Myers J, Hadley D, Oswald U, et al. Effects of exercise training on heart rate recovery in patients with chronic heart failure. Am Heart J 2007;153:1056-63.

46. Karvonen MJ, Kentala E, Mustala O. The effects of training on heart rate; a longitudinal study. Ann Med Exp Biol Fenn 1957;35:307-15.

47. Normandin E, Nigam A, Meyer P, et al. Acute responses to intermittent and continuous exercise in heart failure patients. Can J Cardiol 2013;29:466-71.

48. Fox KF, Cowie MR, Wood DA, et al. Coronary artery disease as the cause of incident heart failure in the population. Eur Heart J 2001;22:228-36.

49. Loehr LR, Rosamond WD, Chang PP, Folsom AR, Chambless LE. Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). Am J Cardiol 2008;101:1016-22.

50. Wright RS, Anderson JL, Adams CD, et al. 2011 ACCF/AHA focused update incorporated into the ACC/AHA 2007 Guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines developed in collaboration with the American Academy of Family Physicians, Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons. J Am Coll Cardiol 2011;57:e215-367.

51. Shaw LJ, Berman DS, Maron DJ, et al. Optimal medical therapy with or without percutaneous coronary intervention to reduce ischemic burden: results from the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial nuclear substudy. Circulation 2008;117:1283-91.

52. Gheorghiade M, Bonow RO. Chronic heart failure in the United States: a manifestation of coronary artery disease. Circulation 1998;97:282-9.

53. Keogh AM, Baron DW, Hickie JB. Prognostic guides in patients with idiopathic or ischemic dilated cardiomyopathy assessed for cardiac transplantation. Am J Cardiol 1990;65:903-8.

54. Rizzello V, Poldermans D, Biagini E, et al. Prognosis of patients with ischaemic cardiomyopathy after coronary revascularisation: relation to viability and improvement in left ventricular ejection fraction. Heart 2009;95:1273-7.

55. Wijns W, Vatner SF, Camici PG. Hibernating myocardium. N Engl J Med 1998;339:173-81.

56. Abidov A, Hachamovitch R, Hayes SW, et al. Prognostic impact of hemodynamic response to adenosine in patients older than age 55 years undergoing vasodilator stress myocardial perfusion study. Circulation 2003;107:2894-9.

57. Elhendy A, Sozzi F, van Domburg RT, et al. Effect of myocardial ischemia during dobutamine stress echocardiography on cardiac mortality in patients with heart failure secondary to ischemic cardiomyopathy. Am J Cardiol 2005;96:469-73.

58. Hachamovitch R, Hayes SW, Friedman JD, Cohen I, Berman DS. Comparison of the short-term survival benefit associated with revascularization compared with medical therapy in patients with no prior coronary artery disease undergoing stress myocardial perfusion single photon emission computed tomography. Circulation 2003;107:2900-7.

59. Schuster A, Morton G, Chiribiri A, et al. Imaging in the management of ischemic cardiomyopathy: special focus on magnetic resonance. J Am Coll Cardiol 2012;59:359-70.

60. Feola M, Biggi A, Chauvie S, et al. Myocardial scar and insulin resistance predict cardiovascular events in severe ischaemic myocardial

dysfunction: a perfusion-metabolism positron emission tomography study. Nucl Med Commun 2008;29:448-54.

61. Rahimtoola SH, La CG, Ferrari R. Hibernating myocardium: another piece of the puzzle falls into place. J Am Coll Cardiol 2006;47:978-80.

62. Allman KC, Shaw LJ, Hachamovitch R, Udelson JE. Myocardial viability testing and impact of revascularization on prognosis in patients with coronary artery disease and left ventricular dysfunction: a meta-analysis. J Am Coll Cardiol 2002;39:1151-8.

63. Cornily JC, Gilard M, Le GG, et al. Accuracy of 16-detector multislice spiral computed tomography in the initial evaluation of dilated cardiomyopathy. Eur J Radiol 2007;61:84-90.

64. Kim WY, Danias PG, Stuber M, et al. Coronary magnetic resonance angiography for the detection of coronary stenoses. N Engl J Med 2001;345:1863-9.

65. Assomull RG, Shakespeare C, Kalra PR, et al. Role of cardiovascular magnetic resonance as a gatekeeper to invasive coronary angiography in patients presenting with heart failure of unknown etiology. Circulation 2011;124:1351-60.

66. Rahimtoola SH. A perspective on the three large multicenter randomized clinical trials of coronary bypass surgery for chronic stable angina. Circulation 1985;72:V123-35.

67. Beanlands RS, Nichol G, Huszti E, et al. F-18-fluorodeoxyglucose positron emission tomography imaging-assisted management of patients with severe left ventricular dysfunction and suspected coronary disease: a randomized, controlled trial (PARR-2). J Am Coll Cardiol 2007;50:2002-12.

68. Cortigiani L, Sicari R, Desideri A, et al. Dobutamine stress echocardiography and the effect of revascularization on outcome in diabetic and non-diabetic patients with chronic ischaemic left ventricular dysfunction. Eur J Heart Fail 2007;9:1038-43.

69. Greenwood JP, Maredia N, Younger JF, et al. Cardiovascular magnetic resonance and single-photon emission computed tomography for diagnosis of coronary heart disease (CE-MARC): a prospective trial. Lancet 2012;379:453-60.

70. Bonow RO, Maurer G, Lee KL, et al. Myocardial viability and survival in ischemic left ventricular dysfunction. N Engl J Med 2011;364:1617-25.

71. Hernandez AF, Newby LK, O'Connor CM. Preoperative evaluation for major noncardiac surgery: focusing on heart failure. Arch Intern Med 2004;164:1729-36.

72. Hernandez AF, Whellan DJ, Stroud S, et al. Outcomes in heart failure patients after major noncardiac surgery. J Am Coll Cardiol 2004;44:1446-53.

73. Exner DV, Birnie DH, Moe G, et al. Canadian Cardiovascular Society guidelines on the use of cardiac resynchronization therapy: evidence and patient selection. Can J Cardiol 2013;29:182-95.

74. Moss AJ, Zareba W, Hall WJ, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877-83.

75. Bristow MR, Saxon LA, Boehmer J, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. N Engl J Med 2004;350:2140-50.

76. Dyub AM, Whitlock RP, Abouzahr LL, Cina CS. Preoperative intra-aortic balloon pump in patients undergoing coronary bypass surgery: a systematic review and meta-analysis. J Card Surg 2008;23:79-86.

77. Alderman EL, Bourassa MG, Cohen LS, et al. Ten-year follow-up of survival and myocardial infarction in the randomized Coronary Artery Surgery Study. Circulation 1990;82:1629-46.

78. Eleven-year survival in the Veterans Administration randomized trial of coronary bypass surgery for stable angina. The Veterans Administration Coronary Artery Bypass Surgery Cooperative Study Group. N Engl J Med 1984;311:1333-9.

79. Varnauskas E. Twelve-year follow-up of survival in the randomized European Coronary Surgery Study. N Engl J Med 1988;319:332-7.

80. Yusuf S, Zucker D, Peduzzi P, et al. Effect of coronary artery bypass graft surgery on survival: overview of 10-year results from randomised trials by the Coronary Artery Bypass Graft Surgery Trialists Collaboration. Lancet 1994;344:563-70.

81. Tsuyuki RT, Shrive FM, Galbraith PD, Knudtson ML, Graham MM. Revascularization in patients with heart failure. CMAJ 2006;175:361-5.

82. Kunadian V, Zaman A, Qiu W. Revascularization among patients with severe left ventricular dysfunction: a meta-analysis of observational studies. Eur J Heart Fail 2011;13:773-84.

83. Moller CH, Penninga L, Wetterslev J, Steinbruchel DA, Gluud C. Off-pump versus on-pump coronary artery bypass grafting for ischaemic heart disease. Cochrane Database Syst Rev 2012;CD007224.

84. Velazquez EJ, Lee KL, O'Connor CM, et al. The rationale and design of the Surgical Treatment for Ischemic Heart Failure (STICH) trial. J Thorac Cardiovasc Surg 2007;134:1540-7.

85. Velazquez EJ, Lee KL, Deja MA, et al. Coronary-artery bypass surgery in patients with left ventricular dysfunction. N Engl J Med 2011;364:1607-16.

86. Doenst T, Cleland JG, Rouleau JL, et al. Influence of crossover on mortality in a randomized study of revascularization in patients with systolic heart failure and coronary artery disease. Circ Heart Fail 2013;6:443-50.

87. Jones RH, White H, Velazquez EJ, et al. STICH (Surgical Treatment for Ischemic Heart Failure) trial enrollment. J Am Coll Cardiol 2010;56:490-8.

88. Chan KM, Punjabi PP, Flather M, et al. Coronary artery bypass surgery with or without mitral valve annuloplasty in moderate functional ischemic mitral regurgitation: final results of the Randomized Ischemic Mitral Evaluation (RIME) trial. Circulation 2012;126:2502-10.

89. Hohnloser SH, Kuck KH, Dorian P, et al. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med 2004;351:2481-8.

90. Bigger JT Jr, Whang W, Rottman JN, et al. Mechanisms of death in the CABG Patch trial: a randomized trial of implantable cardiac defibrillator prophylaxis in patients at high risk of death after coronary artery bypass graft surgery. Circulation 1999;99:1416-21.

91. Goldenberg I, Moss AJ, McNitt S, et al. Time dependence of defibrillator benefit after coronary revascularization in the Multicenter Automatic Defibrillator Implantation Trial (MADIT)-II. J Am Coll Cardiol 2006;47:1811-7.

92. Steinbeck G, Andresen D, Seidl K, et al. Defibrillator implantation early after myocardial infarction. N Engl J Med 2009;361:1427-36.

93. Bardy GH, Lee KL, Mark DB, et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med 2005;352:225-37.

94. Connolly SJ, Hallstrom AP, Cappato R, et al. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trials. AVID,

CASH and CIDS studies. Antiarrhythmics vs Implantable Defibrillator study. Cardiac Arrest Study Hamburg. Canadian Implantable Defibrillator Study. Eur Heart J 2000;21:2071-8.

95. Barsheshet A, Goldenberg I, Moss AJ, et al. Effect of elapsed time from coronary revascularization to implantation of a cardioverter defibrillator on long-term survival in the MADIT-II trial. J Cardiovasc Electrophysiol 2011;22:1237-42.

96. Bax JJ, Schinkel AF, Boersma E, et al. Extensive left ventricular remodeling does not allow viable myocardium to improve in left ventricular ejection fraction after revascularization and is associated with worse long-term prognosis. Circulation 2004;110:II18-22.

97. Cleland JG, Daubert JC, Erdmann E, et al. The effect of cardiac resynchronization on morbidity and mortality in heart failure. N Engl J Med 2005;352:1539-49.

98. Tang AS, Wells GA, Talajic M, et al. Cardiac-resynchronization therapy for mild-to-moderate heart failure. N Engl J Med 2010;363:2385-95.

99. Leon AR, Abraham WT, Curtis AB, et al. Safety of transvenous cardiac resynchronization system implantation in patients with chronic heart failure: combined results of over 2,000 patients from a multicenter study program. J Am Coll Cardiol 2005;46:2348-56.

100. Ailawadi G, Lapar DJ, Swenson BR, et al. Surgically placed left ventricular leads provide similar outcomes to percutaneous leads in patients with failed coronary sinus lead placement. Heart Rhythm 2010;7: 619-25.

101. Penicka M, Bartunek J, Lang O, et al. Severe left ventricular dyssynchrony is associated with poor prognosis in patients with moderate systolic heart failure undergoing coronary artery bypass grafting. J Am Coll Cardiol 2007;50:1315-23.

102. Pokushalov E, Romanov A, Prohorova D, et al. Coronary artery bypass grafting with concomitant cardiac resynchronisation therapy in patients with ischaemic heart failure and left ventricular dyssynchrony. Eur J Cardiothorac Surg 2010;38:773-80.

103. Healey JS, Merchant R, Simpson C, et al. Canadian Cardiovascular Society/Canadian Anesthesiologists' Society/Canadian Heart Rhythm Society joint position statement on the perioperative management of patients with implanted pacemakers, defibrillators, and neurostimulating devices. Can J Cardiol 2012;28:141-51.

104. Grace SL, Chessex C, Arthur H, et al. Systematizing inpatient referral to cardiac rehabilitation 2010: Canadian Association of Cardiac Rehabilitation and Canadian Cardiovascular Society joint position paper endorsed by the Cardiac Care Network of Ontario. Can J Cardiol 2011;27:192-9.