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Physical function and exercise training in older patients with heart failure

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Abstract

Heart failure (HF) is a common end point for numerous cardiovascular conditions, including coronary artery disease, valvular disease, and hypertension. HF predominantly affects older individuals (aged ≥ 70 years), particularly those living in developed countries. The pathophysiological sequelae of HF progression have a substantial negative effect on physical function. Diminished physical function in older patients with HF, which is the result of combined disease-related and age-related effects, has important implications on health. A large body of research spanning several decades has demonstrated the safety and efficacy of regular physical activity in improving outcomes among the HF population, regardless of age, sex, or ethnicity. However, patients with HF, especially those who are older, are less likely to engage in regular exercise training compared with the general population. To improve initiation of regular exercise training and subsequent long-term compliance, there is a need to rethink the dialogue between

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clinicians and patients. This Review discusses the need to improve physical function and exercise habits in patients with HF, focusing on the older population.

Heart failure (HF) is the common end point for numerous cardiovascular conditions, including coronary artery disease, valvular disease, and hypertension. Although HF can develop at any age, it predominantly affects those who are older (aged ≥ 70 years)¹. Older individuals with HF are more likely to be hospitalized more than once, resulting in substantial economic burden on the health-care system. As a consequence, a heightened focus has been placed on reducing hospital admissions associated with HF. Much of what is known about HF treatments that have been shown to reduce morbidity and mortality have largely been evaluated in younger cohorts of patients (aged <70 years), and almost exclusively in those with HF with reduced ejection fraction (HFrEF). Older patients with HF often have a different clinical pattern with relatively preserved left ventricular (LV) systolic function, known as HF with preserved ejection fraction (HFpEF)². Although symptomatology and adverse outcomes associated with HF are similar between HFrEF and HFpEF, particularly with regard to reduced functional capacity, the evidence base for therapies in HFpEF is often lacking compared with HFrEF². Therefore, the majority of research cited in this Review was conducted in patients with HFrEF, unless HFpEF is specifically mentioned.

The pathophysiological sequelae of HF have a substantial negative effect on all aspects of physical function. Diminished physical function, owing to decreased cardiorespiratory fitness and muscular strength in the older population with HF, is particularly concerning. These changes result in a reduced capacity to perform daily activities, and often underlie a very poor quality of life. The risk of falls is also increased in older patients with HF. Assessing the functional capacity of an older patient with HF is vital from multiple perspectives, such as risk prediction for future adverse events, assessment of disease severity, and guidance of medical management (including recommendations for physical activity and exercise training)³.

Patients with stable HF should remain physically active and participate in a structured exercise programme. A substantial body of knowledge spanning several decades has clearly demonstrated the safety and benefits of regular physical activity and exercise training in the HF population⁴⁻⁶, regardless of age, sex, or ethnicity. These benefits including improvements in physiology, prognosis, physical function, and quality of life.

Unfortunately, physical activity patterns and engagement in regular exercise training are poor in the HF population, particularly in those who are aged >70 years⁷. In this Review, we address the need to improve the physical function and exercise habits in patients with HF, focusing on the older population. Improving communication between clinicians and patients, and increasing understanding of data examined under the health literacy framework, are important considerations in the search for strategies to improve the uptake of and adherence to physical activity.

Physiological effects of ageing and HF

Normal response to physical stimuli.

The body's capacity to respond to a physical stimulus is dependent on the integration of several physiological systems, as well as the type and intensity of the stimulus. The cardiovascular, respiratory (musculature driving air exchange), pulmonary (ventilation–perfusion coupling), and peripheral skeletal muscles coordinate and integrate with neural, reflex, and hormonal control systems in response to a physical stimulus⁸. Physical stimuli can be divided into two broad categories: aerobic stimuli and muscle resistance stimuli. Response to an aerobic stimulus is dependent upon the capacity of the respiratory muscular system to augment air exchange, pulmonary system to augment gas exchange at the alveoli–pulmonary vasculature interface, cardiovascular system to augment cardiac output and delivery of oxygen to working skeletal muscle, and working skeletal muscle to augment oxygen consumption (VO_2) and increase aerobic energy production to meet the demand induced by a given level of exertion⁸. Although any of these systems, or combinations thereof, can be the rate-limiting step in the adequate response to an aerobic stimulus, insufficient augmentation of cardiac output is a common core culprit⁹. The response to a muscle resistance stimulus is dependent primarily on the capacity of working skeletal muscle to generate force for a single contraction (maximal force generation) or over several contractions (muscular endurance). This type of physical stimulus is exclusively or largely anaerobic in nature, and is dependent upon muscle cross-sectional area, neuronal firing pattern, and the contractile properties of muscle fibres.

Physiological consequences of ageing.

Normal ageing is associated with reduced physiological reserve capacity in a wide range of systems responsible for responses to aerobic and muscle resistance stimuli. The normal ageing process reduces cardiac function through several pathways, with a final common pathway being diminished diastolic function associated also with mild reductions in maximal systolic function^{10,11}. An age-related decline in maximal heart rate has also been observed. The decline in maximal stroke volume and decrease in heart rate work synergistically to reduce cardiac output, which is the primary reason for the fall in peak VO_2 in apparently healthy individuals^{12,13}. The FRIEND registry¹² reported that the 50th percentile peak VO_2 declined from 48.0 to 24.4 ml/kg/min, and from 37.3 to 18.3 ml/kg/min from the second decade to the seventh decade of life in apparently healthy men and women, respectively (see original publication for full decade-related declines across percentiles of peak VO_2). Although these declines in cardiorespiratory fitness substantially reduce physical function across the lifespan, the slope of this decline can be attenuated by lifelong participation in appropriate levels of physical activity and structured exercise training. Ageing is also associated with changes in skeletal muscle characteristics, namely a decline in capacity for aerobic energy production, cross-sectional area, and fibre contractile properties¹⁴. As such, age-related changes to skeletal muscle further contribute to the decline in capacity to respond to both an aerobic stimulus and a muscle resistance stimulus. Whereas other age-related changes to the respiratory, pulmonary, and cardiovascular systems exist, the changes associated with declined cardiac and skeletal muscle function are the primary drivers of reduced physical performance¹⁴. Notably, published normative tables and

regression equations for age-specific and sex-specific peak VO_2 provide an estimate that is influenced by many factors and, therefore, is subject to considerable prediction error. In particular, the data used to develop peak VO_2 normative values and prediction equations have historically not included a large number of older patients. As such, care needs to be taken when extrapolating these values to older individuals, irrespective of health status (that is, from apparent health to confirmed chronic disease diagnosis).

Physiological consequences of HF.

As with ageing, a core pathophysiological issue associated with HF is diminished cardiac output. This substantial disease-related decline in cardiac output is attributable to systolic or diastolic dysfunction, as is seen in HFrEF or HFpEF, respectively. Systolic and diastolic dysfunction might also coexist, although the primary diagnosis is based on resting LV ejection fraction in the presence of a confirmed diagnosis of HF. Regardless of the mechanism of HF, a considerable fall in cardiac output during physical exertion is a final common outcome that results in significantly lower peak VO_2 levels compared with age-predicted and sex-predicted normal values¹⁵. Although this drop in cardiac output associated with HF is commonly thought to originate from the left ventricle, researchers are gaining an increased appreciation of the effect of HF on right-sided ventricular function and ventriculo-arterial coupling in the presence of secondary pulmonary hypertension, a common occurrence in this population¹⁶. Moreover, the initial insult on cardiac function is accompanied by a near-simultaneous cascade of pathophysiological events that compromise multiple systems, all related to the capacity to perform activities that require physical exertion. Specifically, HF is associated with diminished respiratory muscle strength and endurance, pulmonary ventilation-perfusion matching, and skeletal muscle function. As such, HF commonly impairs the body's capacity to respond to both aerobic and muscle resistance stimuli, resulting in global impairment of physical function.

Established and long-standing HF is often characterized by a substantial decline in skeletal muscle mass associated with immune activation, neurohormonal overactivity, impaired anabolic hormone actions, and possibly cardiac cachexia^{17,18}. In these patients, the accompanying changes in the peripheral musculature, peripheral vascular and endothelial function, and neurohormonal and cardiopulmonary reflex control systems might even overtake haemodynamic disturbance as the most important factor leading to impaired physical activity and functional capacity.

The changes in skeletal muscle function in older patients with HF have been shown to exaggerate reflex sympathoexcitation via muscle ergoreceptor overactivity, and this feature might worsen the severity of HF through its effects on persistent neurohormonal overactivity. Physical exercise training of the altered HF skeletal muscle has been shown to reduce this reflex sympathoexcitation at least partially¹⁹. Another feature of the elderly HF phenotype associated with exercise intolerance is impaired endothelial function; exercise training has been shown to correct this dysfunction more effectively in older than in younger patients with HF, through an increase in endothelial progenitor cells²⁰.

Together, the age-related decline in physiological pathways associated with the capacity to respond to an exertional stimulus compounded by a HF diagnosis and its associated

physiological derangements create a ‘perfect storm’ with respect to the potential for decline in physical function. These characteristics are summarized in TABLE 1. As such, older patients with HF should be considered to have a vulnerable phenotype requiring thorough assessment and aggressive interventional strategies when physical function is found to be compromised. The causes and the available strategies to address these problems and the effects they have on older individuals with HF are discussed below.

Quantification of physical function

The primary chronic symptom in older patients with either HFpEF or HFrEF, even in those who are clinically stable and nonoedematous, is severe exercise intolerance²¹. Exercise tolerance can be measured objectively during a cardiopulmonary exercise test as a decrease in peak VO_2 (REF. 21). A study by Kitzman and co-workers found that, in older patients with HF (mean age 70 years), peak VO_2 during upright treadmill or cycling training was ~30% lower than in age-matched healthy individuals, and the magnitude of the decline was similar in patients with HFrEF or HFpEF²². The peak VO_2 in older patients with HF (~14 ml/kg/min) was also well below the cut-off value (18 ml/kg/min) often used to distinguish between high versus low physical function^{22–24}. HF-mediated decline in peak VO_2 is associated with decreased physical functional performance and has been consistently shown to be an independent predictor of poor outcomes^{25,26}.

Physical function parameters provide a wealth of clinically valuable data and should be a cornerstone evaluation in most HF patient populations. A wealth of evidence is available demonstrating the value of assessing physical function in patients with HF, with a particular focus on assessment during activities that are aerobic in nature (for example, traditional or cardiopulmonary exercise testing or the 6-min walking test). The majority of the literature reporting on the utility of assessing physical function in HF, however, has focused on younger patient cohorts. Importantly, advanced age is not a contraindication for assessing physical function, and perhaps it is of even higher relevance and importance in older patients given the increased likelihood of compromised exercise function.

Cardiopulmonary exercise testing is the gold-standard assessment method for aerobic exercise capacity and should be performed whenever feasible. The methods for cardiopulmonary exercise testing in older patients are largely consistent with those used in younger patients, but tailored testing protocols and modes of exercise might be available for the older cohort. The protocol chosen to test improvement in maximal aerobic tolerance in an older patient should typically be conservative in nature (for example, the ramp protocol). In addition, testing using an ergometer rather than treadmill might be more appropriate in older patients who have problems with balance. Cardiopulmonary exercise testing can be highly reproducible in elderly patients with either HFrEF or HFpEF^{27,28}. When cardiopulmonary exercise testing is not feasible (for example, owing to lack of equipment, appropriate expertise, or suitable level of supervision), other approaches to assessing aerobic performance should be employed, such as traditional exercise testing without ventilatory expired gas analysis or the 6-min walking test²⁹. TABLE 2 provides a list of approaches to aerobic exercise testing and important considerations.

In addition to aerobic exercise testing, assessment of physical function from a muscular strength and endurance perspective is of critical value in older patients, particularly with regard to assessing balance integrity, fall risk, and the ability to perform daily activities³⁰. The assessment of muscular strength and endurance can be performed in several ways and is again dependent on available equipment, expertise, and appropriate levels of supervision³¹. Advanced physical function laboratories might have computerized isokinetic devices that can accurately quantify one-repetition maximum (maximum amount of force generated in one maximal contraction) and number of repetitions to fatigue. Noncomputerized standard resistance equipment can also be used. When resources are limited, use of field equipment (such as hand-held dynamometers) and tests (for example, timed up-and-go tests) are warranted. Finally, given the importance of respiratory muscle strength and endurance physical function in the HF population, the incorporation of this parameter into exercise testing is also strongly recommended. Hand-held computerized devices that allow for an accurate quantification of respiratory muscle strength and endurance are now readily available.

Prognostic value of physical function.

Cardiopulmonary exercise testing is the gold-standard measure of maximal aerobic power, and a number of physiological variables derived from cardiopulmonary testing (peak VO_2 <10 ml/kg/min, minute ventilation/carbon dioxide production slope >45 , presence of exercise oscillatory ventilation, and resting partial pressure of end-tidal carbon dioxide <33 mmHg and <3 mmHg increase during exercise) have been shown to have prognostic value in patients with HF³². Keteyian and co-workers examined the association between cardiopulmonary exercise testing-derived variables and all-cause mortality in 2,100 patients with HFrEF from the HF-ACTION trial²⁵. In the multivariate analysis, adjusted for age and sex, the strongest predictors of all-cause mortality were peak VO_2 , percent-predicted peak VO_2 , and exercise duration²⁵. Specifically, for each 5 percentage point-lowering of percent-predicted peak VO_2 , 1 ml/kg/min-lowering of peak VO_2 , and 1-min-lowering of exercise duration, there was a 19%, 16%, and 16% higher risk of all-cause mortality, respectively²⁵. Shafiq and colleagues extended these findings to patients with HFpEF by demonstrating that peak VO_2 and percent predicted peak VO_2 were significantly associated with all-cause mortality or cardiac transplantation²⁶. In particular, patients with HFpEF with a peak VO_2 <13.9 ml/kg/min and a percent-predicted peak VO_2 $<58\%$ had a cumulative 5-year survival rate of 69% and 67%, respectively. Taken together, these studies suggest that the marked impairment in peak VO_2 and percent-predicted peak VO_2 found in older patients with HF might be associated with a poor prognosis. Similarly, the MECKI Score study³³ involving 3,794 patients with HFrEF reported that a score including peak VO_2 and the slope of the relationship between minute ventilation and the rate of carbon dioxide production preserved its prognostic importance in older patients with their increased background mortality. A further study showed that the combination of impaired diastolic function and an abnormal exercise ventilatory response is predictive of a high-risk phenotype in the elderly patient with HF³⁴.

Physical performance-based tests that are self-paced and simple to perform have also been shown to have important prognostic value in older patients with HF. For example, a 6-min

walking distance 300 m was associated with decreased survival in older (mean age 75 years) patients with HF^{35,36}. Moreover, Boxer and colleagues also reported that each 30 m reduction in 6-min walking distance was associated with a 19% increase in mortality in this patient cohort³⁵. Impaired lower extremity physical function, as measured by the short physical performance battery (a series of physical tests measuring balance, gait, strength, and endurance by examining ability to stand with the feet together in the side-by-side, semi-tandem, and tandem positions, time to walk 8 ft, and time to rise from a chair and return to the seated position five times) at hospital discharge, has also been shown to be an independent predictor of long-term survival in older patients with HFrEF (mean age 80 years, range 65–101 years)³⁷.

Older patients with cardiovascular disease, especially HF, are often frail and have unrecognized physical functional impairments in balance and mobility that are independent predictors of prognosis²⁹. Importantly, these impairments should be assessed and targeted with individualized exercise prescriptions when initiating exercise interventions in order to prevent injuries³⁸.

Physical activity and exercise guidelines

Numerous evidence-based sources are available for guidance on physical activity recommendations and for establishing an exercise regime in older patients with HF. Current physical activity guidelines state that all medically stable adults should accumulate 150 min per week of moderate or 75 min per week of vigorous physical activity, or a combination thereof³⁹. However, clinicians should recognize that these recommendations might seem like an insurmountable goal for many individuals, particularly those who have a sedentary lifestyle, but contemplate becoming more physically active. The issue of perceived ability to meet currently recommended physical activity goals is often more daunting for older individuals and is even further compounded by a diagnosis of HF. In addition, the current body of evidence, reflecting years of data and thousands of participants across the age, sex, and health spectrum, convincingly demonstrates the substantial improvement in health status and prognosis when an individual transitions from a sedentary lifestyle to increased physical activity that still falls far below current guideline recommendations^{40,41}. For example, frail older patients who walked for 1 h per day had markedly lower mortality compared with those who did not⁴². Older patients with HF who are clinically stable should be prescribed with an individualized physical activity and exercise plan that the patient perceives to be achievable and is prepared to perform; aligns patient activity preferences and resources with physical activity and exercise recommendations; and aligns physical capabilities (such as good balance versus increased fall risk), real-time medical status (for example, physiologically stable versus exercise-induced arrhythmias), and level of supervision (that is, independent versus supervised). A physical activity and exercise plan should include both aerobic (for example, walking, cycling, or dancing) and muscular strength and endurance activities (for example, circuit weight training systems, elastic bands, or use of one's own body weight as resistance). Balance-focused activities, such as yoga or Tai Chi might also be advantageous, particularly for those who have problems with balance. In older patients with HF and respiratory muscle weakness, an inspiratory training programme should also be included. The combination of resistance and aerobic training has

been shown to improve muscular strength and submaximal exercise tolerance in patients with HF, irrespective of age⁴³. FIGURE 1 illustrates a decision tree for prescribing physical activity and exercise assessment, and counselling in the clinical setting. All contraindications to physical activity should be identified and screened, and exercise should be prescribed on a continual basis in patients with HF given the changing nature of the disease process (that is, compensated versus decompensated HF). A thorough review of contraindications is beyond the scope of this Review, and has been discussed previously⁴⁴. When contraindications have been resolved, all patients should be prescribed an individually tailored physical activity and exercise plan.

Improvement in exercise performance.

Several investigators have examined the acute haemodynamic responses and chronic adaptations to endurance exercise training in patients with HFrEF aged <65 years^{45–47}. Increased peak VO₂ was found to be associated with a combination of favourable cardiac adaptations (increased maximal exercise cardiac output^{45–48}), peripheral vascular changes (improved conduit artery endothelial function^{45,49}, and decreased systemic and leg vascular resistance^{45,47}), and skeletal muscle adaptations (increased oxidative muscle fibres⁵⁰, elevated oxidative enzyme activity and capacity⁴⁶, and reduced muscle wasting⁵¹) that resulted in increased muscle blood flow and extraction of oxygen by the active muscles^{46,52}.

At present, there is a relative paucity of studies that have examined the central and peripheral adaptations to physical conditioning in patients with HF aged >60 years. Sandri and colleagues assessed the effects of 4 weeks of supervised cycle exercise training on peak VO₂ and resting LV systolic and diastolic function in older (<65 years, mean age 72 ± 5 years) patients with HFrEF⁵³. The increased peak VO₂ after training was associated with an improvement in LV ejection fraction, early (E) to late transmitral filling ratio, septal and lateral systolic and diastolic (E') myocardial annular tissue velocities and decreased mitral E-wave deceleration time, isovolumetric relaxation time, and septal and lateral E/E' (REF. 53).

Kitzman and co-workers have also shown that, in older patients with HFpEF, endurance exercise training does not seem to change resting LV systolic or diastolic function significantly^{54–57}. These findings were extended in a further study demonstrating that the improvement in peak VO₂ after 16 weeks of endurance exercise training in older patients with HFpEF was attributable to noncardiac peripheral factors, as measured by a significant increase in estimated peak arterial–venous oxygen difference with no significant overall change in cardiac output⁵⁸. Two subsequent follow-up studies reported that continuous endurance exercise training or high-intensity interval training did not improve carotid artery distensibility, carotid–arterial pulse wave velocity, or brachial artery endothelial function in older patients with HFpEF^{54,56}. Taken together, these data suggest that, in patients aged >65 years with HFrEF, the increased peak VO₂ after endurance training is a result of favourable skeletal muscle and/or microvascular adaptations that lead to increased oxygen transport and utilization by the active muscles.

Improvement in physical function.

Numerous investigators have shown that endurance exercise training performed alone or with supplemental resistance training increases peak VO_2 (REFS 53,55,58–60), 6-min walking distance^{55,60–62}, and quality of life^{59,60,62} in the older HF population. The magnitude of the improvement in exercise capacity might be related to the intensity of exercise prescribed, as Wisloff and colleagues reported that high-intensity interval training (90–95% peak heart rate for 4 min, interspersed by 3 min of exercise performed at 50–70% peak heart rate) was superior to moderate-intensity continuous exercise (70–75% peak heart rate) for improvement in brachial artery endothelial function, vastus lateralis mitochondrial function, and peak VO_2 in older (mean age 75 years) male patients with HFrEF⁵⁹. However, the larger SMARTEX study⁶³ found that high-intensity interval training and moderate-intensity continuous training resulted in similar improvements in peak VO_2 and LV remodelling in a group of patients with HFrEF. As such, the optimal aerobic training intensity for patients with HF has yet to be elucidated.

The improvement in functional outcomes might also be related to the mode of training performed. For example, high-intensity resistance training (80% one repetition maximum) significantly increased maximal muscular strength^{24,64}, muscular endurance⁶⁴, 6-min walking distance^{24,64}, and physical functional performance²⁴ without a change in peak VO_2 in older patients with HFrEF.

Although relatively few studies assessing the influence of training protocol on HF outcomes have been performed to date, the available data suggest that the mechanisms underlying increased peak VO_2 might depend partly on HF phenotype. In older patients with HFpEF, the increased peak VO_2 after moderate-intensity continuous endurance exercise training was secondary to peripheral adaptations that result in increased peak exercise arterial–venous oxygen difference without a change in haemodynamic cardiovascular function (that is, no change in peak exercise cardiac output, central artery stiffness, or peripheral arterial endothelial function)^{54,58}. Furthermore, this improvement in peak VO_2 has been associated with favourable adaptations in both cardiovascular and peripheral systems^{53,59}. Improvements have also been described in LV diastolic function in both older and younger patients with HFrEF after 4 weeks of supervised endurance training⁶¹. In both HFpEF and HFrEF, the peripheral contributions to improved peak VO_2 with training seem to be largely attributable to improved skeletal muscle function (that is, increased percentage skeletal muscle oxidative fibres, mitochondrial volume density, oxidative capacity, capillarity, and muscle blood flow)^{46,52,65}. This finding is not surprising given the skeletal muscle's capacity for rapid rejuvenation and remodelling, particularly compared with the myocardium²¹. Researchers have suggested that substantial evidence exists for improved peripheral oxygen utilization through both oxygen delivery and extraction via peripheral effects in patients with HFrEF or HFpEF⁶⁶.

Together, these data indicate that older patients with either HFrEF or HFpEF have severely reduced physical function that is associated with reduced quality of life and poor outcomes that can be improved with exercise training. Future studies are required to determine whether exercise training-mediated improvements in physical function are associated with decreased hospitalization and improved survival in this patient cohort. Additional research is

also needed to determine the optimal exercise intensity, the underlying mechanisms of exercise training improvement, and the interventions that can improve exercise adherence⁶⁷. Furthermore, >80% of older patients with HFpEF are overweight or obese, and multiple sources suggest that increased adipose tissue contributes to exercise intolerance in this setting^{55,68,69}. However, the role of caloric restriction in treatment of HF has been controversial owing to the 'HF paradox'; although increased body weight is a risk factor for the development of HF, after HF is established, increased body weight is associated with increased survival, possibly by the protective effect of improved energy stores⁷⁰. Dietary weight loss via caloric restriction in older patients with HFpEF was shown to improve exercise capacity and quality of life, and these benefits were additive to aerobic exercise training⁵⁵. Accordingly, future studies are required to examine exercise with other interventions (such as diet or medications) on health outcomes in older patients with HF. In this regard, data from a large observational study involving 4,623 patients with HFrEF suggest that the protective effect of obesity is lost if the patient has a low peak VO₂, indicating that patients need to be at an optimal state before exercise training to achieve optimal physical fitness⁷¹.

Approaches to improving health

Health literacy and communication.

The importance of clinician–patient interaction and communication has gained increasing recognition in the past few years. Health literacy is defined as “the degree to which an individual has the capacity to obtain, communicate, process, and understand basic health information and services to make appropriate health decisions” (REF. 72). An estimated 12% of adults in the USA are thought to be proficient in health literacy; elderly individuals are at particularly high likelihood of having low health literacy⁷³. A consensus statement from the Heart Failure Society of America recognized low health literacy as an important problem among the HF population⁷⁴. Lower health literacy has been associated with poorer quality of life⁷⁵, as well as an increased risk of adverse events^{74–76} in patients with chronic disease. Furthermore, low health literacy is related to lower self-care behaviour⁷⁷, as well as lower physical function cross-sectionally and a faster health decline longitudinally⁷⁸.

Health literacy, within its broader framework, includes physical literacy, defined as “the motivation, confidence, physical competence, knowledge, and understanding to maintain physical activity throughout the life course” (REF. 79). Elderly patients with HF are likely to lack most or all of the attributes of physical literacy. Given the potential to improve adherence with exercise interventions in elderly patients with HF, assessment of physical literacy should be a high priority in this population.

Although unintentional, health literacy can be misconstrued as unidirectional; a patient might be viewed as either literate or illiterate, which then affects adherence with health-care interventions and outcomes. In this setting, the patient is viewed as a passive recipient of information that the clinician provides. In 2017, McNeil and Arena proposed a concept termed Health Harmonics, defined as the two-way transaction of health information between a clinician and a patient that allows for an authentic, positive construction of meaning, rather than the passive deposition of information⁸⁰. Future approaches to health literacy and

communication should encourage patients to be their own advocate in the decision-making process and plan for improving their health, including adherence to an exercise programme.

Use of behavioural coaching strategies.

Despite strong evidence supporting the benefits of physical activity in patients with HF, including increased survival rates and decreased hospitalization rates, there is often poor uptake of prescribed exercise programmes, with many studies reporting nonadherence in the majority of patients^{81–84}. Indeed, most HF hospital readmissions are associated with failed self-management behaviours, including nonadherence to an exercise regime^{85,86}. These findings are not surprising, given that successful disease self-management in HF requires the adoption of several concurrent, challenging, and often novel health behaviour changes in addition to physical activity and exercise, including changes to diet and fluid intake, weight loss, complex medication regimen, and frequent medical appointments. In order to promote chronic disease self-management, health-care providers have traditionally employed a communication style that involves the unsolicited provision of expert opinion and directive advice. Whereas knowledge of the benefits of physical activity and exercise and prescription of lifestyle changes might be necessary, these factors are generally found to be insufficient to motivate long-term health behaviour change⁸⁷. Motivational communication is an alternative set of evidence-based and patient-centred strategies and interventions that have been demonstrated to improve adherence to a wide range of health behaviours^{88–90}. These techniques might include a combination of cognitive-behavioural strategies^{91,92}, motivational interviewing, and interventions based on well-established theories of motivation (for example, self-determination theory⁹³, social cognitive theory⁹⁴, theory of planned behaviour⁹⁵, and transtheoretical model⁹⁶). A defining feature of motivational communication is the use of a counselling style based on motivational interviewing that respects patient autonomy and decisions about health behaviours, while attempting to resolve ambivalence around behaviour change by eliciting personal reasons for the change. A rapidly expanding body of evidence demonstrating the efficacy of various motivational communication approaches across a wide variety of health-care settings support increased physical activity duration and/or intensity, reduced activity limitation, and increased exercise self-efficacy (an important predictor of the adoption and maintenance of exercise behaviour) for improved health outcomes^{90,97}.

Although nonadherence is often viewed as a patient-related issue, it is actually a multidimensional problem that also includes system-related and provider-related factors. Whereas motivational communication strategies that promote uptake of positive physical activity and exercise behaviours are recommended in ACC/AHA clinical practice guidelines for the management of HF⁹⁸, the type of skills and preparation required to develop competency in delivery of these motivational strategies are unclear. Preliminary training recommendations, including considerations on duration, ongoing individualized supervision, and clinical decision-making have been reported elsewhere⁹⁹.

Conclusions

Physical activity and exercise training are important for the prevention of disease across the health spectrum. A wealth of evidence supports the adoption of an exercise programme in younger patients with HF. Although there is clear scientific justification for exercise training in the older HF population given its benefits on physiological, functional, and clinical outcomes, there seems to be a low rate of adoption of exercise training in older patients with HF. All stable patients with HF, irrespective of age, should be encouraged to engage routinely in physical activity and a structured exercise training programme. Novel strategies are available with the aim of improving adherence to prescribed exercise regimens in patients.

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Key points

- Older patients (aged ≥ 70 years) with heart failure (HF) have substantially impaired physical function and poor clinical outcomes
- A comprehensive assessment of physical function is strongly recommended in this cohort, and should ideally include assessment of aerobic performance as well as peripheral and respiratory muscle strength and endurance
- Physical activity and exercise training are important for improving outcomes in patients with HF, especially elderly patients, but uptake remains poor
- To improve long-term compliance with exercise training, there is a need to improve two-way communication between clinicians and patients, including comprehension of health information such as exercise training
- Improving health literacy, defined as ‘the motivation, confidence, physical competence, knowledge, and understanding to maintain physical activity throughout the life course’, is essential for improving exercise adherence in elderly patients with HF

Figure 1|. Decision tree integrating the assessment, discussion, and prescription of physical activity and exercise in clinical practice.

This figure outlines the decision-making steps when advising an elderly patient with heart failure on an exercise training regimen. The figure includes a checklist of relative contraindications to exercise and an approach to how these could be managed or addressed. HF, heart failure.

Table 1|

Determinants of functional capacity: effects of age and disease

Physiological system	Physiological effects	Age-related effects*	Heart failure-related effects
<i>Central</i>			
Cardiovascular	<ul style="list-style-type: none"> • ↑ Cardiac output • ↑ Heart rate • ↑ Stroke volume <p>Enhanced ventricular–vascular coupling</p>	<ul style="list-style-type: none"> • ↓ Peak cardiac output • ↓ Diastolic performance • ↓ Chronotropic reserve <p>Diminished ventricular–vascular coupling</p> <ul style="list-style-type: none"> • Ventricular: ↑ collagen, fibrosis, cardiac amyloid, and lipofuscin; ↑ myocardial stiffness and spherical remodelling • Vascular: ↑ intimal–medial thickness with ↓ nitric oxide and ↑ angiotensin II, with ↓ vasodilatory capacity 	<p>↓ ↓ Cardiac output (rest and peak) owing to added pathophysiological impairments of systole and/or diastole, and added reduction in chronotropic reserve</p> <p>↑ ↑ Vascular stiffness owing to disease neurohormonal effects (↑ angiotensin II, ↓ nitric oxide, ↑ noradrenaline), as well as physiological vasoconstriction to maintain haemodynamics as cardiac output falls</p>
Respiratory	↑ Inspiratory muscle pressure	Changes in thoracic cavity and elasticity of chest impair peak ventilation	Diaphragmatic weakening owing to intrinsic myocyte changes that further limit peak ventilation and increase energy needed to supply oxygen, and ↑ ergoreflex signalling with induced breathlessness
Pulmonary	Alveoli–pulmonary air gas exchange	<p>↓ Alveoli, especially in context of lifelong tobacco exposure and/or air pollution</p> <p>↑ Pulmonary pressures owing to typical left heart stiffening</p>	<p>↑ Interstitial oedema, and impaired alveolar gas diffusion</p> <p>↑ ↑ Pulmonary pressures continue owing to left heart failure, and often exacerbated by mitral regurgitation, atrial fibrillation, and/or aortic stenosis</p>
<i>Peripheral</i>			
Skeletal muscle	<p>↑ Aerobic type I fibres to achieve muscle endurance</p> <p>↑ Mitochondrial energetics, cellular metabolism, and gene expression to support physical capacity</p> <p>↑ Local redirection to augment microvascular perfusion</p>	<p>Atrophy of type II glycolytic fibres</p> <p>↓ Mitochondrial function</p> <p>↓ Local perfusion (deconditioning often determinant)</p>	<p>↓ Percent and ↑ atrophy of type I oxidative fibres</p> <p>↓ ↓ Mitochondrial volume density and oxidative enzyme activity owing to both deconditioning and direct effects of disease</p> <ul style="list-style-type: none"> • ↓ ↓ Local perfusion • ↓ Capillary density • ↓ Muscle oxygen diffusive conductance • Abnormal metaboreflex: ↑ muscle sympathetic nerve activity and ↓ muscle blood flow leading to ↑ substrate level phosphorylation and ↑ anaerobic glycolysis, such that fatigue occurs earlier and oxygen-uptake kinetics are slowed
	Endothelial responses	↓ Endothelial responses (owing to deconditioning)	↓ ↓ Endothelial-dependent vasodilatation
	Force generation	↓ Force generation (owing to deconditioning)	↓ Force generation

*Variable, depending on habitual exercise, genetic constitution, and/or other idiosyncratic factors.

Table 2|

Types of exercise tests

Feature	Exercise test without ventilatory expired gases	Exercise test with ventilatory expired gases	Walk tests
Key variables	<ul style="list-style-type: none"> • Electrocardiogram • Blood pressure • Heart rate during and after exercise • Estimated aerobic capacity • Self-reported symptoms 	<ul style="list-style-type: none"> • Electrocardiogram • Blood pressure • Heart rate during and after exercise • Self-reported symptoms • Peak oxygen consumption • Ventilatory threshold • Peak respiratory exchange ratio • Ventilatory efficiency* 	<ul style="list-style-type: none"> • Electrocardiogram • Blood pressure • Heart rate • Self-reported symptoms • Distance walked
Applications and utility	<ul style="list-style-type: none"> • Good assessment of cardiorespiratory fitness • Prognostic utility • Some diagnostic utility • Good information related to prescription of a physical activity and exercise plan 	<ul style="list-style-type: none"> • Gold-standard assessment of cardiorespiratory fitness • Prognostic utility • Diagnostic utility • Refined information related to prescription of a physical activity and exercise plan 	<ul style="list-style-type: none"> • General information regarding cardiorespiratory fitness • Some prognostic utility • Some ability to prescribe a physical activity and exercise plan
Advantages	<ul style="list-style-type: none"> • Lower costs to administer • Acceptable reliability and validity • Good prognostic information 	<ul style="list-style-type: none"> • Excellent reliability and validity • Excellent multivariable prognostic information • Excellent multivariable prognostic information 	<ul style="list-style-type: none"> • Low costs • Easy to administer • Well tolerated by patients
Limitations	<ul style="list-style-type: none"> • Handrail use increases cardiorespiratory fitness estimation error • Increased risk of patient apprehension or discomfort 	<ul style="list-style-type: none"> • Higher cost and personnel resources • Increased risk of patient apprehension or discomfort 	<ul style="list-style-type: none"> • Substantial estimation error for cardiorespiratory fitness

* Ventilatory efficiency measures: minute ventilation/carbon dioxide production (VE/VCO₂) slope, oxygen uptake efficiency slope (OUES), and partial pressure of end-tidal carbon dioxide (PETCO₂).