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, particularly those living in developed countries. The pathophysiological sequelae of HF progression has a substantial negative effect on physical function. Diminished physical function in the older patient with HF, which is the result of combined disease-related and age-related effects, has important implications on health. A substantial body of research spanning several decades has clearly 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 the clinician and patient. This Review will address the need to improve the 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 afflicts those who are older.[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 consequent, there has been heightened focus on reducing hospital admissions associated with HF. Much of what is known about HF treatments that have been shown to reduce mortality and morbidity have largely been evaluated in younger cohorts of patients, aged up to 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 in HFrEF[2]. Therefore, the majority of research cited in this review was conducted in patients with HFrEF, unless HFpEF is specifically mentioned.

The pathophysiologic sequelae of HF has a substantial negative effect on all domains 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 activities of daily living and work-related activities, and oftentimes underlie a very poor quality of life. The risk of falls is also increased in the older HF patient. 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 clearly demonstrates the safety and benefits of regular physical activity and exercise training in the HF

population[4-6], regardless of age, sex, and 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 older than 65 years.[7] Improving communication between the clinician and the patient, and increasing comprehension of data examined under the health literacy framework, is an important consideration in the search of strategies to improve physical activity patterns and exercise training adherence in this population. This Review will address the need to improve the physical function and exercise habits in patients with HF, focusing on the older population, usually referred to as aged greater than 65 years.

[H1] Physiological effects of ageing and HF

[H2] 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 (that is, musculature driving air exchange), pulmonary (that is, ventilation–perfusion coupling), and peripheral skeletal muscles coordinate and integrate with neural, reflex, and hormonal control systems in response to a physical stimulus.[8] Physical stimuli can be divided into two broad categories: aerobic stimuli and a 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 exertion.[8] 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 culprit.[9] 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.

[H2] Physiological consequences of ageing

Normal ageing is associated with reduced physiologic reserve capacity in a wide range of systems responsible for response 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 registry[12] reported that the 50^{th} percentile peak VO_2 declined from 48.0 to 24.4 ml per kg per min, and from 37.3 to 18.3 ml per kg per 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 properties.[14] 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 performance.[14] Notably, published normative tables and regression equations for age 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, particular caution when extrapolating these values to older individuals, irrespective of health status (that is, from apparent health to confirmed chronic disease diagnosis).

[H2] Physiological consequences of HF

Like in ageing, a core pathophysiological issue associated with HF is diminished cardiac output. The mechanism for this substantial disease-related decline in cardiac output can be broadly classified into systolic or diastolic dysfunction (such as is seen in HFrEF or HFpEF, respectively). Systolic and diastolic dysfunction might also coexist, although the primary diagnosis is based upon 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 compared with

age and sex-predicted normal values.[15] 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 chronic disease population.[16] Moreover, the initial insult in 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 an aerobic and muscle resistance stimuli, resulting in a global impairment in physical function.

Established and long-standing HF is often characterized by a substantial decline in skeletal muscle mass associated with immune activation, neurohormonal over-activity, 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 sympatho-excitation via muscle ergoreceptor overactivity, and this feature might worsen the severity of HF through its effects on persistent neurohormonal over-activity. Physical exercise training of the altered HF skeletal muscle has been shown to at least partially reduce this reflex sympatho-excitation.[19] In addition, 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 cells.[20]

Together, the age-related decline in physiological pathways associated with the ability 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 and 2**. As such, the older patient 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.

[H1] Quantification of impaired 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 intolerance.[21] Exercise tolerance can be measured objectively during a cardiopulmonary exercise test as a decrease in peak VO_2.[21] A study by Kitzman and coworkers 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 HFpEF. [22] The peak VO_2 in older patients with HF (~14 ml per kg per min) was also well below the cutoff value (18 ml per kg per min) often used to distinguish between high versus low physical function.[23-25]. 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.[26,27]

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 walk 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 higher likelihood of compromised exercise function.

Cardiopulmonary exercise testing is the gold standard assessment for aerobic exercise capacity and should be performed whenever feasible. The methods for cardiopulmonary exercise testing in the older patient are largely consistent with those employed in younger patients, but specially tailored testing protocols and modes of exercise might be available for the older cohort. The protocol chosen to improvement maximal aerobic tolerance in an older patient should typically be conservative in nature (for example, ramp protocol). In addition, testing using an ergometer rather than treadmill might be more appropriate in older patients who have issues with balance. Cardiopulmonary exercise testing can indeed be highly reproducible in elderly patients with either HFrEF or HFpEF.[28,29] When CPX is not feasible (for example, owing to lack of equipment, appropriate expertise, or appropriate 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 walk test.[30] **Table 3** 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/endurance perspective is of critical value in older patients, particularly with regard to assessing balance integrity, fall risk, and the ability to perform daily activities.[31] The assessment of muscular strength and endurance can be performed in several ways and is again dependent upon available equipment, expertise, and appropriate levels of supervision.[32] 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. Non-computerized 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 to physical function in the HF population, the incorporation of this parameter to 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.

[H2] 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 per kg per min, minute ventilation/carbon dioxide production slope ≥45, presence of exercise oscillatory ventilation, and resting partial pressure of end-tidal carbon dioxide <33mmHg and < 3mmHg increase during exercise) have been shown to have prognostic value in patients with HF.[33] Keteyian and coworkers examined the association among cardiopulmonary exercise testing-derived variables and all-cause mortality in 2,100 patients with HFrEF from the HF-

ACTION trial.[26] 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 duration.[26] Specifically, for each 5 percentage point-lowering of percent-predicted peak VO_2, 1 ml per kg per 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, respectively.[26] 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 transplantation.[27] In particular, patients with HFpEF with a peak VO_2 $\leq$13.9 ml per kg per min and a percent predicted peak VO_2 $\leq$58% had a cumulative 5-year survival rate of 69% and 67%, respectively. Taken together, these studies suggest 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 study[34] involving 3,794 patients with HFrEF reported that a score including Peak VO_2 and VE/VCO_2 slope (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 HF.[35]

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 walk distance $\leq$300m was associated with decreased survival in older (mean age $\geq$75 years) patients with HF.[36,37] Moreover, Boxer and colleagues also reported that each 30m reduction in 6-min walk distance was associated with a 19% increase in mortality in this patient cohort.[36] Impaired lower extremity physical function, as measured by the short physical performance

battery (SPPB, 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 feet, and time to rise from a chair and return to the seated position 5 times) at hospital discharge has also been shown to be an independent predictor of long-term survival in older (mean age 80 years, range 65-101 years) patients with HFrEF.[38]

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 prognosis.[30] Importantly, these impairments should be assessed and targeted with individualized exercise prescriptions when initiating exercise interventions in older patients in order to prevent injuries.[39]

[H1] 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 minutes per week of vigorous physical activity, or a combination thereof.[40] However, it is important to recognize that these recommendations might seem like an insurmountable goal for a number of individuals, particularly those who are leading a sedentary lifestyle, but contemplating 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 guidelines.[41,42] For example, frail older patients who walked for one hour per day had markedly lower mortality compared with those who did not.[43] Older patients with HF who are clinically stable should be prescribed an individualized physical activity and exercise plan that: the patient perceives as 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 balance deficits. In the older patient 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 sub-maximal exercise tolerance in patients with HF, irrespective of age.[44] **Figure 1** illustrates a decision tree for approaching physical activity and exercise assessment, counselling, and prescription 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 fluid nature of the disease process (that is, compensated versus decompensated). A thorough review of contraindications is beyond the scope of this Review, and has been published previously.[46] Once contraindications have been resolved, all patients should be prescribed an individually tailored physical activity and exercise plan.

[H2] 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.[47-49] Increased peak VO_2 was found to be associated with a combination of favourable cardiac adaptations (increased maximal exercise cardiac output[47-50]), peripheral vascular changes (improved conduit artery endothelial function[47,51], and decreased systemic and leg vascular resistance[47,49]), and skeletal muscle adaptations (increased oxidative muscle fibres[52], elevated oxidative enzyme activity and capacity[48], and reduced muscle wasting[53]) that resulted in increased muscle blood flow and extraction of oxygen by the active muscles.[48,54]

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 VO_2 and resting LV systolic and diastolic function in older (<65 years, mean age 72 ± 5 years) patients with HFrEF.[55] The increased peak VO_2 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'.[55]

Kitzman and coworkers have also shown that in older patients with HFpEF, endurance exercise training does not appear to significantly change resting LV systolic or diastolic function.[56-59] These findings were extended in a further study demonstrating that the improvement in peak VO_2 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 output.[60] 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 the older patient with HFpEF[56, 58]. Taken together, these data suggest that in patients with HFrEF aged >65 years, the increased peak VO_2 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.

[H2] Improvement in physical function

Numerous investigators have shown that endurance exercise training performed alone or with supplemental resistance training increases peak VO_2 [60, 57, 61,55,62], 6-min walking distance[57, 62-64], and quality of life[61, 62, 64] in 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 the improvement in brachial artery endothelial function, vastus lateralis mitochondrial function, and peak VO_2, and attenuation of LV in older (mean age 75 years) male patients with HFrEF.[61] However, the larger SMARTEX study[65] found that high-intensity interval training and moderate-intensity continuous training resulted in similar improvements in peak VO_2 and left ventricular remodeling 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,[25, 66] muscular endurance,[66] 6-min walking

distance,[25, 66] and physical functional performance[25] 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 responsible for 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).[60, 56] Furthermore, this improvement in peak VO_2 has been associated with favourable adaptations in both cardiovascular and peripheral systems.[61 55] Improvements have also been described in left ventricular diastolic function in both older and younger patients with HFrEF after 4 weeks of supervised endurance training.[63] 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).[48, 54 67] This finding is not surprising given the skeletal muscle's capacity for rapid rejuvenation and remodelling, particularly compared with the myocardium.[21] Researchers have suggested that substantial evidence exists for improved peripheral O_2 utilization through both O_2 delivery and extraction via peripheral effects in patients with HFrEF or HFpEF.[68]

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 if

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 adherence.[45] 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.[69, 57, 70] However, the role of caloric restriction in treatment of HF has been controversial owing to the 'HF paradox'.[71] This paradox is that although increased body weight is a risk factor for the development of heart failure, once heart failure is established increased body weight is associated with increased survival, possibly by the protective effect of improved energy stores. 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 training.[57] 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 VO_2, indicating that patients need to be at an optimal before exercise training to achieve optimal physical fitness. [72]

[H1] Approaches to improving health

[H2] 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".[73] An estimated 12% of adults in the USA are thought to be proficient in health literacy; elderly individuals are at particularly high risk for low health literacy.[74] A consensus statement from the Heart Failure Society of America recognized low health literacy as an important issue among the HF population.[75] Lower health literacy has been associated with poorer quality of life[76], as well as an increased risk of adverse events[75-77] in patients with chronic disease. Furthermore, low health literacy is related to lower self-care behaviour[78], as well as lower physical function cross-sectionally and a faster health decline longitudinally.[79]

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".[80] 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 deposit of information.[81] Moving forward, the approach 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.

[H2] 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.[82-85] Indeed, most HF hospital readmissions are associated with failed self-management behaviours, including nonadherence to an exercise regime.[86, 87] 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, it is generally found to be insufficient to motivate long-term health behaviour change.[88] 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.[89-91] These techniques might include a combination of cognitive-behavioural strategies[92, 93], motivational interviewing, and interventions based on well-established theories of motivation (for example, self-determination theory[94], social cognitive theory[95], theory of planned behaviour[96], and transtheoretical model[97]). 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. [91,98]

Although nonadherence is often viewed as a patient-related issue, it is in fact a multidimensional problem that also includes system 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 management of HF[99], 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 elsewhere.[100]

[H1] 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 routinely engage 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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Table 1. Central determinants of functional capacity: effects of age and disease

Physiological system	Physiological effects	Age-related effects*	HF-related effects
Cardiovascular	↑ Cardiac output • ↑ Heart rate • ↑ Stroke volume	↓ Peak cardiac output • ↓ Diastolic performance • ↓ Chronotropic reserve	↓↓ Cardiac output (rest and peak) owing to added pathophysiological impairments of systole and/or diastole, and added reduction in chronotropic reserve
	Enhanced ventricular-vascular coupling	Diminished ventricular-vascular coupling • 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	↑↑ Vascular stiffness owing to disease neurohormonal effects (↑ angiotensin II,↓ nitric oxide, ↑ noradrenaline), as well as physiological vasoconstriction to maintain haemodynamics as cardiac output falls
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	↓ Alveoli, especially in context of lifelong tobacco exposure and/or air pollution	↑ Interstitial edema ↓ Impaired alveolar gas diffusion
		↑ Pulmonary pressures owing to typical left heart stiffening	↑↑ Pulmonary pressures continue owing to left heart failure, and often exacerbated by mitral regurgitation, atrial fibrillation, and/or aortic stenosis
*variable, depending on habitual exercise, genetic constitution, and/or other idiosyncratic factors. HF, heart failure			

Table 2. Peripheral determinants of functional capacity: effects of age and disease

Physiological system	Physiological effects	Age-related effects*	HF-related effects
Skeletal muscle	↑Aerobic type I fibres to achieve muscle endurance	Atrophy of type II glycolytic fibres	↓ Percent and ↑ atrophy of type I oxidative fibers
	↑Mitochondrial energetics, cellular metabolism, and gene expression to support physical capacity	↓ Mitochondrial function	↓↓ Mitochondrial volume density and oxidative enzyme activity owing to both deconditioning and direct effects of disease
	↑ Local redirection to augment microvascular perfusion	↓ Local perfusion (deconditioning often determinant)	↓↓ Local perfusion • ↓ Capillary density • ↓ Muscle O_2 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 O_2-uptake kinetics are slowed
	Endothelial responses	↓ Endothelial responses (owing to deconditioning)	↓↓ Endothelial dependent vasodilation
	Force generation	↓ Force generation (owing to deconditioning)	↓ Force generation

*variable, depending on habitual exercise, genetic constitution, and/or other idiosyncratic factors.
HF, heart failure

Table 3

	Exercise Test without ventilatory expired gases	**Exercise Test with ventilatory expired gases**	**Walk Tests**
Key Variables	• ECG • Blood Pressure • Heart Rate during and post exercise • Estimated aerobic capacity • Self-Reported Symptoms	• ECG • Blood Pressure • Heart Rate during and post exercise • Self-Reported Symptoms • Peak VO_2 • Ventilatory Threshold • Peak RER • Ventilatory Efficiency*	• ECG • Blood Pressure • Heart Rate • Self-Reported Symptoms • Distance Walked
Applications and Utility	• Good assessment of cardiorespiratory fitness • Prognostic utility • Some diagnostic utility • Good information related to prescription of a physical activity and exercise plan	• Gold standard assessment of cardiorespiratory fitness • Prognostic utility • Diagnostic utility • Refined information related to prescription of a physical activity and exercise plan	• General information regarding cardiorespira • Some prognostic utility • Some ability to prescribe a physical activity
Advantages	• Lower costs to administer • Acceptable reliability and validity • Good prognostic information	• Excellent reliability and validity • Excellent multivariable prognostic information • Excellent multivariable prognostic information	• Low costs • Easy to administer • Well tolerated by patients
Limitations	• Handrail use increases cardiorespiratory fitness estimation error • Increased risk of patient apprehension or discomfort	• Higher cost and personnel resources • Increased risk of patient apprehension or discomfort	• Significant estimation error for cardiorespira

Legend: ECG, electrocardiogram; VO_2, oxygen consumption; RER, respiratory exchange ratio

* Ventilatory efficiency measures: Minute ventilation/carbon dioxide production (VE/VCO_2) slope, Oxygen uptake efficiency slope (OUES); Partial pressure of end-tidal carbon dioxide ($P_{ET}CO_2$)

Figure 1: Decision tree for integrating the physical activity and exercise assessment, discussion, and prescription in clinical practice. HF, heart failure