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ORIGINAL ARTICLE

Improved walking speed is associated with lower hospitalisation rates in patients in an exercise-based secondary prevention programme

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ABSTRACT

Objective To determine the relationship between walking speed (WS) maintained during a 1 km test and its improvement on hospitalisation in cardiac outpatients who were referred to an exercise-based secondary prevention programme.

Methods Hospitalisation was assessed in 1791 patients 3 years after enrolment and related to the WS achieved during a 1 km walk at moderate intensity on a treadmill. Hospitalisation was also assessed during the fourth-to-sixth years as function of improvement in WS in 1111 participants who were re-evaluated 3 years after baseline.

Results Three-year hospitalisation rate across tertiles of baseline WS was 50% for the slow walkers (2.7 ±0.6 km/hour), 41% for the moderate (4.1±0.3 km/hour) and 25% for the fast walkers (5.2±0.5 km/hour) (p for trend <0.0001), with adjusted HRs (95% CI) of 0.93 (0.74 to 1.17, p=0.53) for intermediate and 0.58 (0.43 to 0.78, p=0.0003) for fast. Every 1 km/hour increase in WS was associated with a 21% reduction in hospitalisation (p<0.0001). Hospitalisation from the fourth-to-sixth years was lower across tertiles of improved WS, with 44% for the low (0.2±0.4 km/hour), 34% for the intermediate (0.8±0.2 km/hour) and 30% for the high tertile (1.6±0.4 km/hour) (p for trend <0.0001). Adjusted HRs were 0.68 (p=0.002) for the intermediate and 0.58 (p<0.0001) for the high tertile. Every 1 km/hour increase in WS was associated with a 35% reduction in hospitalisation (p<0.0001).

Conclusion Improvement in WS is associated with a significant, dose-dependent lower rate of all-cause hospitalisation in cardiac outpatients. WS is a simple, easily applied and clinically useful tool for cardiac patients undergoing secondary prevention.

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INTRODUCTION

Cardiovascular diseases (CVDs) are the leading cause of mortality in the world and 'produce immense global health and economic burdens'.[1] In the USA, the annual medical costs of CVDs are projected to increase between 2012 and 2030 from $396 billion to $918 billion. Of this, 60.5% is attributable to hospital costs.[1] Prioritisation of health behaviours (including physical activity), in addition to the treatment of established CVD, is a primary goal of numerous health organisations worldwide to improve cardiovascular health and reduce healthcare costs related to CVD.[1]

Cardiorespiratory fitness predicts cardiovascular and total morbidity in adults with and without CVD[2 3] and is strongly related to walking capacity.[4 5] The ability to walk reflects the integrated performance of numerous organ systems. Lower walking speed (WS) has been shown to predict hospitalisation and postoperative morbidity and mortality in patients with CVD,[6 7] as well as incident CVD in pre-frail men and women.[8] Slow WS also has been associated with many health-related factors including physical impairments, disability, morbidity and mortality[9] and an individual's capacity to recover after cardiac surgery.[7] Therefore, the assessment of WS is an indicator of health and function in ageing and disease and has been recommended as a potential additional 'vital sign'.[10]

Evidence for the prognostic value of WS is largely based on a single measure at baseline. However, since physical activity habits can change during a given follow-up period, inferences based on a single measure at baseline could lead to erroneous conclusions.[11] Walking tests of varying distance and times are used to assess exercise tolerance in various clinical conditions and among community-dwelling adults. Even though these tests are considered submaximal, they are largely reasonable surrogates for an individual's functional capabilities since the participant is instructed to 'cover as much ground as you possibly can' during a certain time[12] or 'to walk as fast as possible' for a certain distance.[13 14] Daily physical activities rarely require maximal effort. Thus, a key to health-related fitness assessment is perhaps the ability to perform submaximal exercise.

In this respect, examination of submaximal exercise capacity can be useful for functionally evaluating patients and for developing appropriate exercise prescriptions, adjusting the medical regimen and identifying the need for further diagnostic interventions.[15] WS is a convenient measure of physical function, consistent with the most routine activity of most adults. The moderate speed maintained during a 1 km treadmill walk has been demonstrated to be a valid and simple tool for cardiorespiratory fitness estimation and is inversely related to survival in an outpatient setting.[16–18] However, little is known regarding the association

between WS and non-fatal events and even less is known about the prognostic utility and clinical meaning of changes in walking performance over time as predictors of health outcomes.

The current study was conducted among 1791 ambulatory outpatients with stable CVD to investigate whether moderate walking performance during a 1 km walk is associated with all-cause hospitalisation and whether serial changes in WS are accompanied by changes in long-term hospitalisation.

METHODS

Study population

A total of 1791 consecutive patients (80.5% with coronary heart disease), aged 25–86 years, were enrolled in the exercise-based secondary prevention programme at the Center for Biomedical Studies Applied to Sport at the University of Ferrara, Italy. The ultimate goal of the programme was long-term promotion and maintenance of a physically active lifestyle in order to improve cardiorespiratory fitness and functional ability. A home programme consisting of 30–60 min of moderate aerobic exercise such as brisk walking, at least 3–4 days and preferably 7 days of the week was recommended. All subjects were also encouraged to improve physical activity habits by increasing daily activities, such as walking breaks at work, gardening or household work. Left ventricular ejection fraction derived from prior echocardiographic evaluations and standard blood chemistry analyses previously performed were registered. Before admission to the programme, participants underwent a comprehensive clinical evaluation, including medical history. Body mass index (BMI) and blood pressure (BP) were measured and hypertension was defined as systolic BP≥140 mm Hg, diastolic BP≥90 mm Hg or use of antihypertensive agents. The study was approved by the Human Studies Committee of the University of Ferrara, no. 22-13 and all subjects gave written informed consent.

WS determination

On admission to the programme and regularly during follow-up, each patient performed a 1 km treadmill walk test as previously described (1k-TWT).[16] Briefly, the test was carried out as follows: the participants were instructed to select a pace that they could maintain for 10–20 min at a moderate perceived exercise intensity using the Borg 6–20 scale. Participants began the test walking on the level at 2.0 km/hour, with subsequent increases of 0.3 km/hour every 30 s up to a WS corresponding to a perceived exertion of 11–13 on the Borg scale. The test was then started and the rate of perceived exertion was acquired every 2 min. WS was adjusted to maintain the selected moderate perceived intensity. Heart rate was monitored continuously during the test using a Polar Accurex Plus heart rate monitor (Polar Electro, Kempele, Finland). BP was monitored before and immediately after the test. No individual was excluded on the basis of his or her performance on the treadmill protocol. Subjects walking at a moderate speed of <3.0 km/hour performed the test over the distance of 500 m. The time to complete either 500 m or 1 km was recorded and average WS was calculated accordingly.

Follow-up and hospitalisation

Subjects were enrolled between October 1997 and January 2013. Re-evaluation 3 years after baseline was between October 2000 and January 2013. The last date of follow-up for hospitalisation was December 2014. Functional assessments included baseline, quarterly during the first 2–3 years and thereafter twice per year. Examinations for guiding prognosis, motivating patients and adjusting exercise prescriptions included clinical examination, history of changes in physical activity or symptoms, response to therapy and adverse events, development of relevant or new conditions or changes in existing conditions and assessment of WS.

Participants were flagged by the regional Health Service Registry of the Emilia-Romagna Region, which provided data on re-hospitalisation. The first end point was the incidence of hospitalisation during the first, second and third year after baseline. The second end point was the incidence of hospitalisation during the fourth, fifth and sixth year after baseline (performed in a subgroup of participants who received an additional examination 3 years after baseline). Any hospital admission was considered an event. Patients who had more than one hospital admission within a 24-hour period, usually due to transferral to a second hospital, were classified as having had a single hospitalisation. For patients experiencing >1 hospitalisation, only the first event was considered in the analysis.

Data analysis

At baseline, patient's CVD diagnosis was determined from the hospital discharge record. If >1 cardiac diagnosis was recorded during that admission, we defined the diagnosis as follows: coronary artery bypass graft (CABG) superseded other reasons for hospitalisation such as myocardial infarction (MI) or valve replacement or repair. If the admitting diagnosis was MI and a subsequent percutaneous transluminal coronary angioplasty (PTCA) was or was not performed, it was coded as an MI. If a PTCA was performed in the absence of MI, it was coded as PTCA without MI. If valvular replacement was performed in the absence of MI, it was coded as valvular replacement. Admitting diagnosis of heart transplantation, cardiac tumours or coronary artery anomalies were coded as others.

The participants were grouped into tertiles on the basis of (1) the WS during the 1k-TWT at baseline and (2) the change in WS during the test performed 3 years later. One way analysis of variance was used to determine differences between tertiles in terms of age, BMI, left ventricular ejection fraction, total cholesterol and high-density lipoprotein (HDL) cholesterol, triglycerides, fasting blood glucose and WS. Differences in categorical variables across tertiles were assessed using the χ^2 test for trend.

The covariates considered as potential confounders were age, gender, BMI, left ventricular ejection fraction, current smoking status, hypertension, family history, fasting glucose, total cholesterol, HDL cholesterol, serum triglycerides, serum creatinine, personal medical history and use of ACE inhibitors, angiotensin receptor blockers (ARBs), aspirin, β-blockers, calcium antagonists, diuretics, statins and number of medications.

To assess the association between WS and incidence of hospitalisation over time, we constructed Kaplan-Meier curves. Significantly correlated variables were entered for the fully adjusted multivariable regression model. The risk of hospitalisation was considered independently for each variable, including WS (using increments of 1 km/hour); adjustments were made for age and gender. In addition, formal tests of interaction were performed between WS and WS change and all the covariates included in the multivariable models. Competing-risk regression analysis was used to determine the risk of hospitalisation adjusted for confounders. Individuals in the tertile with the lowest WS at baseline and individuals in the tertiles with the lowest improvement in WS 3 years later were considered the reference groups. The assumption of proportionality for all variables introduced in the models was assessed by analysis of Schoenfeld residuals. The proportional hazards assumption was

met for all models. The level of statistical significance was set at p<0.05. Statistical analyses were performed using MedCalc 14.12.0 (Mariakerke, Belgium) and Stata (V.13.0; StataCorp, College Station, Texas, USA).

RESULTS

Baseline characteristics

The average WS of the 1791 subjects who completed the test without complications was 4.0±1.1 km/hour. Table 1 illustrates the baseline clinical characteristics of the study population grouped into tertiles of WS.

Comparison between categories revealed significant differences for the following variables: age, gender, BMI, left ventricular ejection fraction, hypertension, family history, fasting glucose, total and HDL cholesterol, serum creatinine, history of CABG, acute MI (AMI), PTCA, other medical conditions and use of ACE inhibitors or ARB, aspirin, statins, β-blockers and diuretics. There were no significant differences in current smoking status, serum triglycerides and use of calcium antagonists or number of medications.

Baseline WS and 3-year hospitalisation

During the 3 years following the baseline examination (median 36 months), 699 subjects (39.1% of the sample) were hospitalised for all causes.

The age-adjusted and gender-adjusted HRs for hospitalisation relative to WS and the other clinical variables considered are presented in table 2.

The best predictor of all-cause hospitalisation was the WS determined at baseline, with lower values associated with a higher likelihood of admission. Family history of CVD, use of diuretics, serum creatinine and left ventricular ejection fraction were also significantly associated with hospitalisation. The cumulative risk of hospitalisation by tertiles of WS is presented in figure 1 (log rank, p<0.0001). Hospitalisation increased across decreasing tertiles of WS (fastest, n=145, 25.4%; middle, n=252, 40.7%; slowest, n=302, 50.3%). None of the tested interactions between WS with covariates were statistically significant.

Competing-risk regression analysis showed that, compared with the slowest group, the HR for hospitalisation was lower in the moderate group (HR 0.93, 95% CI 0.74 to 1.17, p=0.53) and further lower in the fastest group (HR 0.58, 95% CI 0.43 to 0.78, p=0.0003). After adjustments for confounders, every 1 km/hour increase in gait speed was associated with a 21% reduction in risk of hospitalisation (HR 0.79, 95% CI 0.71 to 0.89, p<0.001) (see online supplementary table S1).

WS improvement and 4–6 years' hospitalisation

Of the 1791 participants, 1111 (62% of the sample) were re-evaluated 3 years (median 36 months) after baseline; 680

Table 1 Baseline characteristics of the subjects by tertiles of walking speed

	All subjects (n=1791)	T1 (n=600)	T2 (n=621)	T3 (n=570)	p Value
Walking speed (km/hour)					
Mean (SD)	4.0 (1.1)	2.7 (0.6)	4.1 (0.3)	5.2 (0.5)	<0.001
Range	1.20–7.09	1.20–3.50	3.51–4.50	4.51–7.09	
General					
Age (year)	63 (10)	68 (9)	62 (9)	58 (9)	<0.001
Gender (n, M/F)	1499/292	410/190	544/77	545/25	<0.0001
BMI	27.5 (3.7)	27.8 (4.3)	27.7 (3.3)	27.1 (3.4)	0.002
LV ejection fraction (%)	56 (10)	55 (11)	56 (10)	58 (10)	<0.001
Risk factor					
Current smoking (%)	6	6	6	7	0.3
Hypertension (%)	60.1	69.0	60.4	50.4	<0.0001
Family history (%)	49.9	42.2	49.4	58.5	<0.0001
Fasting glucose (mg/dL)	108 (28)	112 (33)	109 (25)	105 (23)	0.001
Total cholesterol (mg/dL)	191 (39)	196 (41)	191 (38)	188 (37)	0.004
HDL cholesterol (mg/dL)	51 (14)	52 (14)	51 (15)	50 (13)	0.04
Serum triglycerides (mg/dL)	141 (76)	143 (82)	141 (73)	138 (74)	0.60
Serum creatinine (mg/dL)	1.1 (0.5)	1.2 (0.7)	1.1 (0.3)	1.1 (0.4)	<0.001
Medical history					
CABG (%)	50.9	60.8	50.4	41.0	<0.0001
Myocardial infarction (%)	22.2	13.7	24.4	28.9	<0.0001
PTCA (%)	7.4	4.5	6.6	11.4	<0.0001
Valvular replacement (%)	12.7	18.2	11.0	8.9	<0.0001
Other (%)	3.5	1.3	3.2	6.1	<0.0001
Medications					
ACE inhibitor or ARB (%)	54.0	59.3	52.5	50.4	0.002
Aspirin (%)	71.8	67.0	74.0	74.6	0.04
β-blocker (%)	56.4	49.0	61.6	58.7	0.007
Calcium antagonist (%)	13.2	14.5	13.1	12.1	0.22
Diuretic (%)	17.0	31.8	13.4	5.4	<0.0001
Statin (%)	50.2	43.5	50.4	57.1	<0.0001
No. of medications (n)	2.7 (1.3)	2.7 (1.3)	2.7 (1.3)	2.6 (1.3)	0.48

Values are presented as mean (SD) or percentage.
ARB, angiotensin receptor blocker; BMI, body mass index; CABG, coronary artery bypass graft; F, female; HDL, high-density lipoprotein; LV, left ventricular; M, male; PTCA, percutaneous transluminal coronary angioplasty, stenting or both.

Table 2 Age-adjusted and gender-adjusted risk of hospitalisation according to clinical variables

	HR	95% CI	p Value
General			
Walking speed (km/hour)	0.76	0.70 to 0.83	<0.0001
BMI (kg/m^2)	1.02	1.00 to 1.04	0.10
LVEF (%)	0.98	0.98 to 0.99	0.001
Risk factor			
Current smoking	1.33	0.95 to 1.87	0.10
Hypertension	1.14	0.97 to 1.33	0.11
Fasting glucose (mg/dL)	1.00	0.99 to 1.01	0.82
Total cholesterol (mg/dL)	1.00	0.99 to 1.00	0.65
HDL cholesterol (mg/dL)	1.00	0.99 to 1.01	0.86
Serum triglycerides (mg/dL)	0.99	0.99 to 1.00	0.99
Family history	0.84	0.72 to 0.98	0.02
Serum creatinine (mg/dL)	1.20	1.10 to 1.30	0.002
Medical history			
CABG	1.00	–	–
Myocardial infarction	0.99	0.81 to 1.20	0.89
PTCA	0.86	0.62 to 1.19	0.36
Valve	1.12	0.89 to 1.40	0.33
Other	1.28	0.86 to 1.89	0.23
Medications			
Calcium antagonist	1.08	0.88 to 1.34	0.46
Aspirin	1.00	0.85 to 1.19	0.96
Statin	1.00	0.86 to 1.16	0.97
β-blocker	0.90	0.78 to 1.05	0.19
Diuretic	1.37	1.14 to 1.64	<0.001
ACE inhibitor or ARB	1.02	0.88 to 1.18	0.82

Walking speed, LVEF, fasting glucose, total and HDL cholesterol, serum triglycerides and creatinine were analysed as continuous variables.
ARB, angiotensin receptor blocker; BMI, body mass index; CABG, coronary artery bypass graft; HDL, high-density lipoprotein; LVEF, left ventricular ejection fraction; PTCA, percutaneous transluminal coronary angioplasty, stenting or both. Valve, valve replacement or repair.

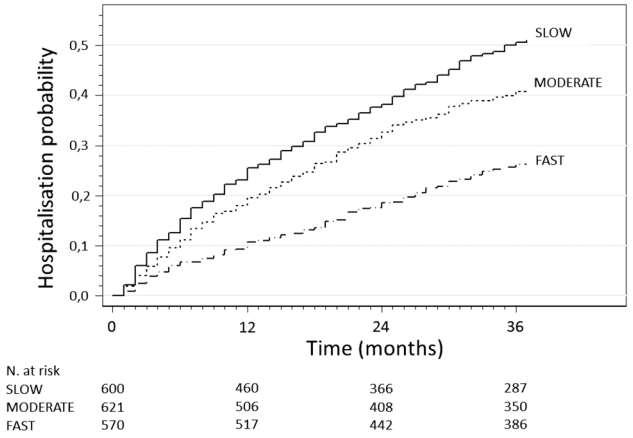

Figure 1 Kaplan-Meier curve showing the rate of hospitalisation during 36 months after enrolment as function of walking speed at baseline.

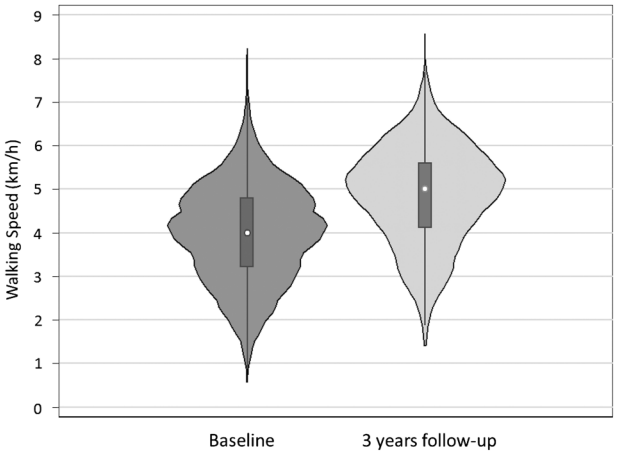

Figure 2 Violin plot illustrating walking speed distribution at baseline and after 3 years of follow-up.

subjects were not included in this analysis because they were not re-evaluated; 200 subjects were not included because they were enrolled for less than 3 years, 423 subjects missed the second test and 57 subjects died. Participants who died had lower baseline WS, were older, had lower BMI, were more likely to be a current smoker, more likely to have high blood pressure, higher serum creatinine, history of CABG, valvular repair or replacement and had more common use of aspirin and diuretics. They were less likely to have a family history of CVD, history of AMI and PTCA and had lower serum triglycerides and were less likely to use ACE inhibitors. Among the subjects who missed the second test, 184, 127 and 112 were in tertiles 1, 2 and 3 at baseline, respectively. Compared with the subjects re-evaluated, participants who missed the second test were more likely to be a current smoker (9% vs 4%), had a lower history of CABG (49% vs 58%), had a higher history of PTCA (10% vs 6%) and had more common use of ACE/ARB inhibitors (55% vs 51%). Figure 2 shows the WS distribution at baseline and after 3 years of follow-up. The characteristics of the subjects re-evaluated 3 years after baseline are presented in table 3.

The improvements in WS ranged from 4.0 (1.0) to 4.9 (1.1) km/hour for the total population, from 4.2±1.1 to 4.4 ±1.2 km/hour for the low tertile (n=372, p<0.0001), from 4.1 ±1.0 to 4.8±1.0 km/hour for the moderate tertile (n=369, p<0.001) and from 3.8±0.9 to 5.4±0.9 km/hour for the high tertile (n=370, p<0.001).

During 4–6 years after baseline, 404 subjects were hospitalised for all causes. Figure 3 shows the Kaplan-Meier survival curves for hospitalisation by tertiles of WS improvement. Rates of hospitalisation was progressively lower across tertiles of WS improvement and reached 44%, 35% and 30% in low, moderate and high groups, respectively (p<0.0001). None of the tested interactions between WS change with covariates were statistically significant. The lower hospitalisation for WS improvement persisted after adjustment for age, gender, BMI, baseline WS, hypertension, family history and use of diuretics or calcium antagonists at baseline; compared with patients in the low group, the HRs for those in the moderate and high groups were 0.68 (95% CI 0.54 to 0.86, p=0.002) and 0.58 (95% CI 0.45 to 0.75, p<0.0001), respectively. After adjustments for confounders and baseline WS, every 1 km/hour increase in WS was associated with a 34% reduction in risk of hospitalisation (HR 0.66, 95% CI 0.56 to 0.78, p<0.0001) (see online supplementary table S2).

Hospitalisation rates was lower across tertiles of baseline WS and across tertiles of WS improvements. The improvements in WS were more strongly associated with the risk of hospitalisation in the subjects with lower and moderate WS at baseline (figure 4). The association between change in WS and hospitalisations persisted after adjusting for the WS achieved 3 years

Table 3 Characteristics of the subjects re-evaluated 3 years after baseline subgrouped in tertiles of improvement of walking speed

	All (n=1111)	low improvement (n=372)	moderate improvement (n=369)	high improvement (n=370)	p Value
Walking speed improvement from baseline (km/hour)					
Mean (SD)	0.9 (0.7)	0.2 (0.4)	0.8 (0.2)	1.6 (0.4)	<0.0001
General					
Age (year)	62 (9)	63 (10)	63 (9)	60 (9)	0.001
Gender (M/F)	950/161	308/64	316/53	326/44	0.04
BMI (kg/m^2)	27.9 (3.9)	28.4 (4.0)	27.9 (4.0)	27.5 (3.5)	0.06
LV ejection fraction (%)	56 (10)	57 (10)	56 (10)	56 (10)	0.7
Risk factor					
Current smoking (%)	4	5	4	3	0.5
Hypertension (%)	59.6	61	66	52	0.01
Family history (%)	52.5	47	54	56	0.01
Fasting glucose (mg/dL)	110 (29)	110 (30)	111 (29)	108 (29)	0.5
Total cholesterol (mg/dL)	193 (38)	195 (39)	192 (40)	191 (37)	0.3
HDL cholesterol (mg/dL)	51 (14)	51 (14)	50 (13)	52 (14)	0.3
Serum triglycerides (mg/dL)	139 (75)	142 (76)	137 (62)	137 (84)	0.7
Serum creatinine (mg/dL)	1.10 (0.5)	1.13 (0.6)	1.10 (0.3)	1.10 (0.5)	0.9
Medical history					
CABG (%)	59.2	60	55	58	0.4
Myocardial infarction (%)	21.5	20	21	22	0.8
PTCA (%)	5.7	5	7	5	0.9
Valvular replacement (%)	11.7	11	13	10	0.6
Other (%)	2.0	4	4	5	0.9
Medications					
ACE inhibitor or ARB (%)	53.1	53	55	51	0.7
Aspirin (%)	72.3	71	73	73	0.7
β-blockers (%)	55.6	54	55	58	0.5
Calcium antagonists (%)	14.3	18	12	13	0.06
Diuretics (%)	14.9	18	14	12	0.06
Statins (%)	50.0	49	52	49	0.7
No. of medications (n)	2.7 (1.2)	2.7 (1.3)	2.7 (1.3)	2.6 (1.2)	0.7

Values are presented as mean (SD) or percentage.
The values of the variables considered (except walking speed) are baseline values.
ARB, angiotensin receptor blocker; BMI, body mass index; CABG, coronary artery bypass graft; F, female; HDL, high-density lipoprotein; LV, left ventricular; M, male; PTCA, percutaneous transluminal coronary angioplasty, stenting or both.

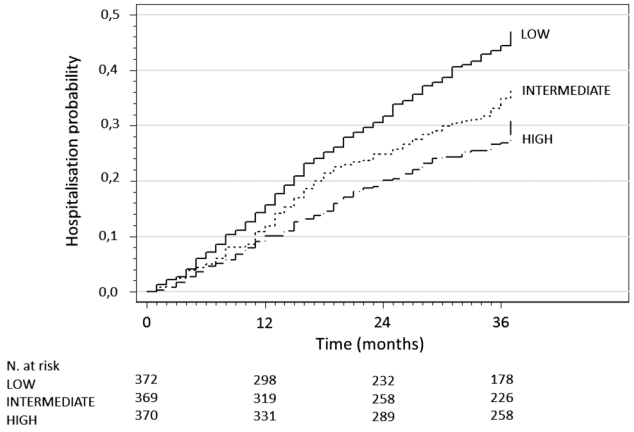

Figure 3 Kaplan-Meier curve showing the rate of hospitalisation 36–72 months after enrolment as function of walking speed improvement.

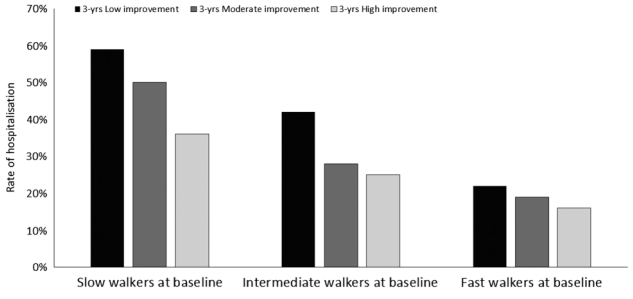

Figure 4 Crude rates of hospitalisation by combined categories of walking speed at baseline and walking speed improvement 3 years after are shown.

after baseline. Finally, WS at year 3 was also significantly associated with the risk of hospitalisation in the following 3 years. Every 1 km/hour increase in WS was associated with an adjusted 18% reduction in the risk of hospitalisation (HR 0.82, 95% CI 0.69 to 0.97, p=0.02).

DISCUSSION

In our cohort of 1791 patients with stable CVD, slower WS observed at baseline was associated with a higher risk of hospitalisation, independent of age, clinical history and traditional risk factors. After grouping the sample into tertiles of WS and adjusting for confounders, a 42% (95% CI 22% to 57%) lower risk of hospitalisation over the following 3 years was documented among the fastest group compared with the slowest. Each 1.0 km/hour increase in WS was associated with a 21% lower rate of all-cause hospitalisation.

Using a variety of walking tests, similar findings have been observed in 472 patients with MI followed for 5.5 years, with increased cardiovascular events across decreasing tertiles of WS[19] and in 556 outpatients with stable coronary heart disease followed for 8.0 years,[12] as well as 440 patients with chronic heart failure followed for 1 year.[20] An association between impaired WS and increased risk of morbidity and mortality has also been demonstrated in patients with stable CVD[17] and in patients undergoing cardiac surgery.[7]

The second salient observation of our study was the inverse association between improvement in WS and hospitalisation documented in 1111 subjects. Compared with 372 subjects with a minimal change in WS, among 369 and 370 subjects who increased WS by 20% and 42%, respectively, lower 32% and 42% rates of hospitalisation, respectively, were observed between the fourth and the sixth year of follow-up. After adjustment for confounders, the association between WS improvement and all-cause hospitalisation remained robust. To our knowledge, no other study has examined the effect of improvement in WS on hospitalisation in cardiac outpatients during a long-term follow-up.

Why does walking speed and its improvement affect hospitalisation?

Walking reflects the integrated performance of the cardiorespiratory, nervous and musculoskeletal systems, and WS is strongly related to cardiorespiratory fitness.[4 5] In addition, regular physical activity positively affects many CVD risk factors[21–23] including fibrinolysis and coagulability, inflammation, autonomic function, coronary artery disease progression and age-related decline in myocardial blood flow and endothelium-dependent vasodilatation. All these factors likely contribute to WS to some extent and help explain the inverse association between improvement in WS and all-cause hospitalisation.

Strengths of the study

This study has several strengths. First, there was a comparatively large sample size of patients across a wide range in age and WS. Second, the simplicity of the WS makes it easy to apply in clinical practice. Third, compared with other walking tests in use (often carried out at an intensity close to maximum), the 1k-TWT is performed at a moderate intensity and is therefore easier to perform, more palatable to patients, is likely safer and has a minimal 'learning effect' regarding the intensity to be used in unsupervised conditions in a secondary prevention programme. Finally, the non-restrictive inclusion criteria employed is likely to reflect real-world clinical practice.

Limitations of the study

This study was conducted in cardiac outpatients enrolled in a secondary prevention programme, who survived to follow-up and chose to volunteer for an examination; thus, they may not be representative of the general population. The adherence to the home programme recommended was not determined. Thus, a causal relationship between the amount of physical activity and the WS improvement was not established.

Cognitive impairment, cerebrovascular or peripheral arterial disease, as well as social, behavioural or psychological factors that could independently modify WS[24–27] were not considered. Given the relatively small number of women, the findings are mainly applicable to men. Although exercise capacity is an independent predictor of mortality in women, differences in exercise testing responses between men and women have been debated.[28–30] However, after adjusting for gender, the risk estimation was similar.

Finally, since year 3 models were adjusted for baseline covariates, we also cannot account for residual confounding due to changes in baseline variables that could have occurred over 3 years.

Conclusion

The present study adds to the growing body of literature supporting the concept that subjects with slow WS have a higher rate of hospitalisation, which represents a critical and commonly used end point in CVD clinical trials. This study also provides evidence that improvement in WS is associated with a lower hospitalisation, which is proportional to the magnitude of the improvement in WS. These results suggest that the WS maintained in a 1 km test at moderate intensity is a simple metric that can be used by health professionals to promote and maintain physically active lifestyles in cardiac outpatients.

Key messages

What is already known on this subject?
Walking speed is inversely associated with morbidity and mortality. However, the walking speed improvement as predictor of re-hospitalisation among cardiac patients undergoing secondary prevention is not defined.

What might this study add?
This study shows that improvement in walking speed is associated with a significant, dose-dependent lower all-cause hospitalisation in cardiac outpatients.

How might this impact on clinical practice?
The 1 km walking test is a simple and clinically useful tool to follow outcomes in cardiac outpatients.

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Competing interests None declared.

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Improved walking speed is associated with lower hospitalisation rates in patients in an exercise-based secondary prevention programme

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