

A Forty-Year Follow-Up of the Dallas Bed Rest and Training Study: The Effect of Age on the Cardiovascular Response to Exercise in Men

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Background. In 1966, five 20-year-old men underwent a comprehensive physiological evaluation of the capacity for adaptation of the cardiovascular system in response to 3 weeks of bed rest and 8 weeks of heavy endurance training; these same participants were reevaluated before and after training at the age of 50. The aim of the present study was to reexamine these same men 40 years following the original assessments.

Methods and Results. In all three studies, minute ventilation and expired gases were analyzed during exercise testing with Douglas bag collection. Cardiac output (CO) was determined using the acetylene rebreathing technique. Compared with the original 30-year interval, the decline in maximal oxygen uptake (VO_{2max}) (–11% vs –25%), maximal CO (+6% vs –11%), and maximal stroke volume (+10% vs –10%) were greater between 50 and 60 years of age. The annualized decline in VO_{2max} (55 mL/min/y) between ages 50 and 60 was approximately fourfold higher than the decline between 20 and 50 years (12 mL/min/y).

Conclusions. In the original five participants of the Dallas Bed Rest and Training Study, VO_{2max} declined after 40 years of living due to a balanced decrease in central and peripheral determinants of oxygen uptake. The rate of decline in VO_{2max} and its components accelerated after the age of 50 years secondary to age and clinical comorbidities. The net proportional decline in VO_{2max} for a period of 40 years of life was comparable with that experienced after 3 weeks of strict bed rest at the age of 20 (27% vs 26%, respectively).

Key Words: Maximal oxygen uptake—Cardiac output—Arteriovenous oxygen difference—Longitudinal.

IN the 1966 Dallas Bed Rest and Training Study, five 20-year-old men were submitted to 3 weeks of bed rest and 8 weeks of heavy endurance training to test the limits of the cardiovascular system (1). To appreciate the influence of aging on the cardiovascular response to acute exercise before and after an endurance exercise training program, these same five men were reexamined 30 years later (2,3). The primary findings from the 30-year follow-up were that the average decline in maximal oxygen uptake (VO_{2max}) from age 20 to 50 was less than that observed after 3 weeks of bed rest when the participants were 20 years old and was reversible with endurance exercise training (2,3). Capitalizing on the comprehensive information gathered from these previous studies, the primary aim of this investigation was to reexamine these same five men at the age of 60 to assess the longitudinal changes in the cardiovascular response to maximal exercise.

A decline in maximal oxygen uptake is a long appreciated consequence of aging (4–14); however, the nature of this decline and the underlying mechanisms are not completely understood. Previous cross-sectional studies suggest that the decline in VO_{2max} with age is linear, occurring at a rate ranging from 0.2 to 0.5 mL/kg/min/y (4–14). In contrast, longitudinal studies suggest that the decline in VO_{2max} with

age is nonlinear, with values declining rapidly after the age of 50 years (7). As longitudinal serial measurements of maximal cardiac output (CO_{max}) and arteriovenous oxygen difference ($AV-DO_{2max}$) have yet to be performed, the mechanisms underlying the reduced VO_{2max} with age remain unclear. Reevaluating the five original members of the Dallas Bed Rest and Training Study a third time, at the age of 60 years, provided a unique opportunity to study the serial longitudinal changes in VO_{2max} and its determinants.

METHODS

The participants were five men aged 59–60 years who participated in the original Dallas Bed Rest and Training Study in 1966. All participants were screened by medical history, physical examination, routine laboratory tests, and resting electrocardiogram (ECG). Each participant provided written informed consent to the study protocol approved by the University of Texas Southwestern Medical Center Institutional Review Board.

Screening Exercise Test

Three participants underwent a screening exercise test with gas exchange analysis to exclude prohibitive coronary

artery disease; two performed the test on a cycle ergometer, whereas another performed the test on a recumbent elliptical machine. One of the participants declined a screening exercise test and another presented with persistent atrial fibrillation and was not tested until normal sinus rhythm was achieved pharmacologically. All screening exams began at a workload of 60 watts for 2 minutes and increased by about 30 watts every 2 minutes until maximal exhaustion. Ventilatory flow was evaluated using an in-line pre-vent pneumotach (MedGraphics, St. Paul, MN), and in-line gas exchange was evaluated using a metabolic cart (MedGraphics CPX/D).

Exercise Testing

Four of five participants performed a familiarization session to determine appropriate workloads for the maximal exercise test. Maximal exercise treadmill testing was performed within 2 weeks of the familiarization session using the same protocols that were used during the 30-year follow-up assessments in 1996. Due to severe low back pain, one of the participants performed the familiarization and maximal exercise testing on a recumbent elliptical machine. During all maximal tests, heart rate (HR) was monitored continuously by ECG, and blood pressure was monitored every 2–3 minutes by recording brachial artery cuff pressure (Suntech Biosystems, Morrisville, NC). Gas exchange analysis was performed by the technique of Douglas, with $[\text{CO}_2]$, $[\text{N}_2]$, and $[\text{O}_2]$ determined by mass spectrometry (Marquette MGA 1100) (3); ventilatory volume was measured from Douglas bags collected for 60-second intervals with a Tissot spirometer. In-line breath-by-breath evaluation by mass spectrometry served as backup to the Douglas bag collections, and ventilatory flow was determined by a turbine flowmeter in a Ventilatory Measurement Module (Interface Associates, Inc., Morrisville, NC). CO was determined with a standard inert gas rebreathing technique, with acetylene as the soluble gas and helium as the insoluble gas (15,16). This technique has previously been validated at rest and exercise against the direct Fick equation by others and within our laboratory (17,18). This technique was also used in these same participants in the 1966 and 1996 evaluations (1,3). Arterialized lactate was determined via fingerstick samples at the end of exercise (YellowSpring International 2300 Stat Plus; YSI, YellowSprings, OH).

Oxygen uptake and CO were determined at rest and at maximal effort. During maximal exercise testing, Douglas bags were collected in the second minute of each of the final three stages (as predicted from screening test data), with consecutive 60-second collections when the participant was nearing maximal effort. CO was determined during the final 10 seconds of maximal exercise, followed within 2 minutes by fingerstick sample collection for lactate determination. Maximal exercise was defined as an inability to continue exercise despite vigorous encouragement and confirmed by respiratory exchange ratio greater than 1.1, (minute ventilation)/ VO_2 greater than 35, arterialized lactate greater than

6.0 mmol/L, HR approximately 100% predicted maximum, and respiratory rate greater than 30. Mean arterial pressure obtained during each CO determination was divided by the respective CO to estimate total peripheral resistance (19). CO was divided by HR during rebreathing to calculate stroke volume (SV). Oxygen uptake was divided by CO to calculate AV- DO_2 according to the Fick equation.

Body Composition

Underwater weighing was performed, with residual volume corrections made by rebreathing an inert mixture of helium in all participants (20). Measurements were made in triplicate and averaged, with body density calculated with the formula of Goldman and Buskirk, and percent body fat calculated according to the Siri equation (21).

Statistical Analysis

Nonparametric statistical analyses were performed on paired data from the 1966 baseline, 1996, and 2006 data. Nonparametric statistical analyses were also used to test for differences in the age-related changes between the two follow-up periods. Pearson's correlational analysis was used for select dependent variables (SPSS system for Windows). We acknowledge the limitations of statistical analyses resulting from the small sample size, with the focus on individual data and group trends.

RESULTS

During the screening visit, three of the five participants reported antihypertensive medications, two of which were new since 1996. Participant A was being treated with a combination of angiotensin-converting enzyme (ACE) inhibitor and diuretic (Lotrel and Hydrochlorothiazide [HCTZ]). Participant D was being treated with a combination of ACE inhibitor, diuretic, and alpha channel blocker (Prinivil, HCTZ, and Cardura). Participant E was being treated with a combination of calcium channel blocker, ACE inhibitor, and diuretic (Norvasc, Lisinopril, and HCTZ). Screening visits revealed persistent atrial fibrillation in one of the participants and paroxysmal atrial fibrillation in another, both of whom were referred to their primary care providers for treatment and exercise testing was not performed until restoration of normal sinus rhythm in both participants. A renal cell carcinoma that had metastasized into the lower lumbar vertebrae was discovered by magnetic resonance imaging (MRI) in a third participant who had reported severe back pain for several months prior to his initial visit. Exercise testing had been completed on this participant prior to this finding using a recumbent elliptical machine.

Group Average Results

Average baseline characteristics are presented in Table 1 with comparative values from 1966 and 1996 included. On

Table 1. Participant Characteristics and Baseline Hemodynamics

Variable	1966		1996	2006
	Baseline	After Bed Rest		
Height (cm)	184	184	185	183
Weight (kg)	77 (15)	76 (14)	100 (37)	91 (31)
Body % fat	13.9 (4.1)	15.7 (4.5)	28.0 (3.0)	28.0 (10.0)
Fat-free mass (kg)	67 (12)	64 (12)	69 (21)	63 (3)
Systolic blood pressure (mm Hg)	131 (23)	134 (10)	140 (4)	128 (17)
Diastolic blood pressure (mm Hg)	75 (14)	74 (10)	88 (7)	83 (9)
HR (b.p.m.)	76 (27)	83 (15)	90 (17)	87 (23)
VO ₂ (mL/min)	0.3 (0.04)	0.3 (0.09)	0.4 (0.13)	0.4 (0.09)
CO (L/min)	5.7 (1.3)	4.9 (0.7)	5.9 (1.1)	4.9 (0.5)
SV (mL/beat)	79 (23)	60 (13)	68 (18)	64 (13)
AV-DO ₂ (vol%)	5.8 (1.2)	7.0 (2.3)	6.1 (1.2)	7.3 (1.7)

Note: HR = heart rate; VO₂ = oxygen uptake; CO = cardiac output; SV = stroke volume; AV-DO₂ = arteriovenous oxygen difference.

average, body weight increased significantly from 1966 to 2006 (77 vs 91 kg), attributable to a significant increase in percent body fat (13.9% vs 28.0%). Fat-free mass (FFM) (63 kg) declined slightly from 1966 (67 kg) and 1996 (69 kg) values. Among all resting hemodynamics, only resting HR was significantly lower in 1966 compared with 2006 values (76 vs 87 beats per minute [b.p.m.]); and none were different compared with 1996 values.

The age-related changes in several cardiopulmonary variables at maximal exertion are presented in Table 2. All participants achieved maximal exertion upon cardiopulmonary exercise testing. Absolute VO_{2max} declined in three of the five men (mean decline=17%) between 1996 and 2006 (Figure 1). The magnitude of the decline in VO_{2max} was proportionally less when indexed to total body mass (8.0%; 31.0 vs 28.5 mL/kg/min) and FFM (9.6%; 42.9 vs 38.8 mL/kg FFM/min). Between 1996 and 2006, CO_{max} declined in the same three of the five participants (mean decline=11%; Figure 2), secondary to a decline in both HR_{max} (181 vs 174 b.p.m.) and SV_{max} (121 vs 109 mL). AV-DO_{2max} also declined in three of the five men (mean decline=8%; Figure 3). Changes in CO_{max} were linearly correlated with the change in VO_{2max} between 1996 and 2006 ($r=0.77$; Figure 4).

Several differences in the changes in cardiopulmonary variables between the two follow-up intervals (1966–1996 and 1996–2006) are of particular interest (Figure 5). The proportional decline in VO_{2max} was greater in the 10 years between 1996 and 2006 than the previous 30-year interval (–17% vs –8%). In the interval between 1966 and 1996, the decline in VO_{2max} was attributed entirely to a 17% decline in AV-DO_{2max}. Although the proportional decline in AV-DO_{2max} was slightly less between 1996 and 2006 (8%), it was accompanied by an 8.5% decline in CO_{max}, whereas no decline in CO_{max} had been observed during the previous 30-year interval.

Individual Results

Individual age-related changes in maximal oxygen uptake and its determinants are provided in Table 3. Participant A was one of the sedentary participants from the 1966 study. At the time of his visit in 2006, he was being treated for hypertension and hypercholesterolemia, with a combination of ACE inhibitor or diuretic and statin, respectively. He had not performed regular exercise for more than 20 years prior to the 30-year follow-up but was performing resistance exercise training 3 d/wk for 2 years prior to the 2006 study. Body weight and body fat increased between 1966 and 1996 by

Table 2. Results from Treadmill Maximal Exercise Test: Group Averages

Variable	1966			2006	
	Baseline	After Bed Rest	1996	Participant E included	Without Participant E
VO _{2max} (L/min)	3.3 (1.1)	2.4 (1.0)	2.9 (0.7)	2.4 (0.6)	2.5 (0.6)
VO _{2max} (mL/kg LBM/min)	49.7 (10.9)	37.4 (11.4)	42.9 (9.5)	38.8 (11.0)	42.3 (9.3)
VO _{2max} (mL/kg/min)	43.0 (10.9)	31.8 (11.1)	31.0 (7.6)	28.5 (11.0)	32.4 (7.9)
CO (L/min)	20.0 (4.1)	14.8 (4.8)	21.4 (5.1)	18.9 (2.5)	18.8 (2.8)
HR (b.p.m.)	193 (8)	197 (7)	181 (16)	174 (13)	179 (10)
SV (mL/beat)	104 (22)	75 (22)	121 (39)	109 (19)	106 (20)
AV-DO ₂ (mL/O ₂ /100 mL)	16.2 (2.3)	16.5 (3.2)	13.8 (2.0)	12.7 (3.0)	13.4 (2.9)
Systolic blood pressure (mm Hg)	204 (49)	153 (42)	206 (23)	176 (9)	175 (10)
Diastolic blood pressure (mm Hg)	81 (9)	63 (16)	96 (22)	82 (24)	94 (5)
MAP (mm Hg)	122 (21)	93 (24)	133 (15)	114 (15)	121 (4)
TPR (dyne/s/cm ⁵)	484 (72)	525 (148)	519 (138)	651 (169)	661 (101)

Note: HR = heart rate; VO_{2max} = maximal oxygen uptake; CO = cardiac output; SV = stroke volume; AV-DO₂ = arteriovenous oxygen difference; LBM = lean body mass; MAP = mean arterial pressure; TPW = total peripheral resistance.

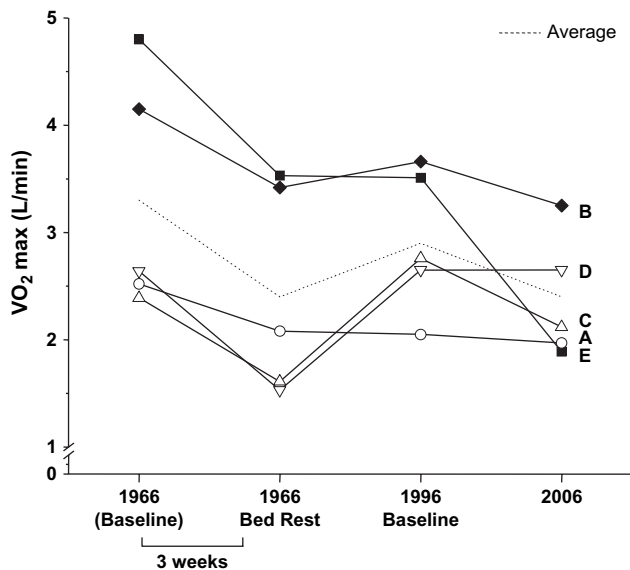


Figure 1. Changes in absolute maximal oxygen uptake.

12% and 75%, respectively, but were unchanged between 1996 and 2006. The age-related decline in VO_{2max} was significantly attenuated between 1996 and 2006 (2.1 vs 2.0 L/min), relative to the first 30-year interval (2.6 vs 2.1 L/min). HR_{max} was unchanged during the first 30-year interval but declined 14 b.p.m. between 1996 and 2006 (197 vs 183 b.p.m.). In contrast to the 30-year interval, SV_{max} (80 vs 101 mL/beat) increased significantly between 1996 and 2006, resulting in an increase in CO_{max} (17.3 vs 18.4 L/min).

Participant B ran up to 75 miles weekly before the 1966 study and had continued a regular training program until 1996. He was not taking medications and was otherwise healthy at the 40-year follow-up visit. Between 1996 and 2006, he maintained his training program, replacing running

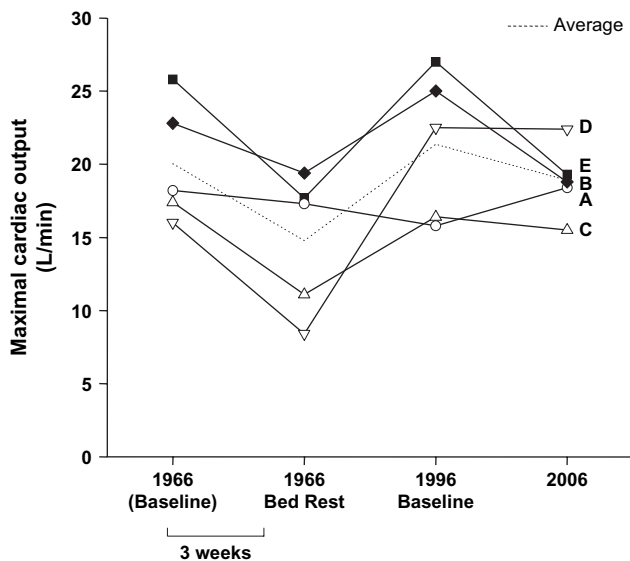


Figure 2. Changes in maximal cardiac output.

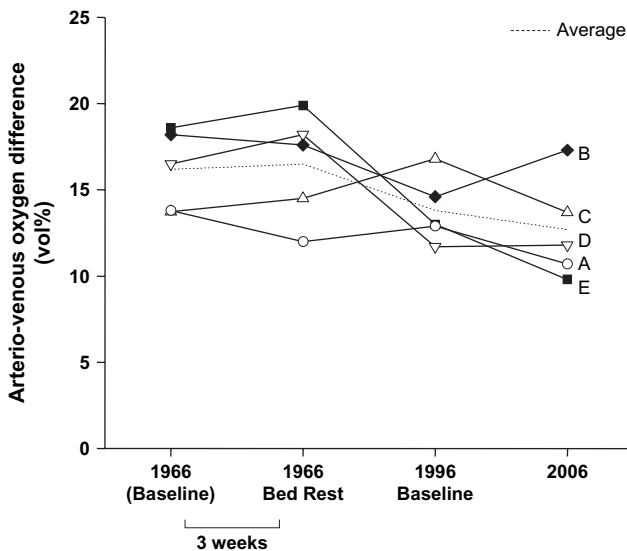


Figure 3. Changes in maximal arteriovenous oxygen difference.

with upright cycling and regularly performed interval training. The significant gains in body weight (15 kg) and percent fat (17%) observed between 1966 and 1996 were annulled (-10 kg and -17%) between 1996 and 2006. VO_{2max} declined to a similar degree (~11%) during the two follow-up periods (4.2 vs 3.7 vs 3.3 L/min). The rise in CO_{max} observed during the first 30-year year follow-up was followed by a 30% decline (25.0 vs 18.8 L/min) in the subsequent 10-year follow-up. The reduction in CO_{max} was attributed almost entirely to a decline in SV_{max} (149 vs 111 mL/beat), as HR_{max} remained unchanged (170 vs 168 b.p.m.). AV- DO_{2max} increased 18% (14.6 vs 17.3 vol%) in the 10-year interval.

Participant C had participated in high school athletics but was otherwise sedentary prior to the 1966 study. During the

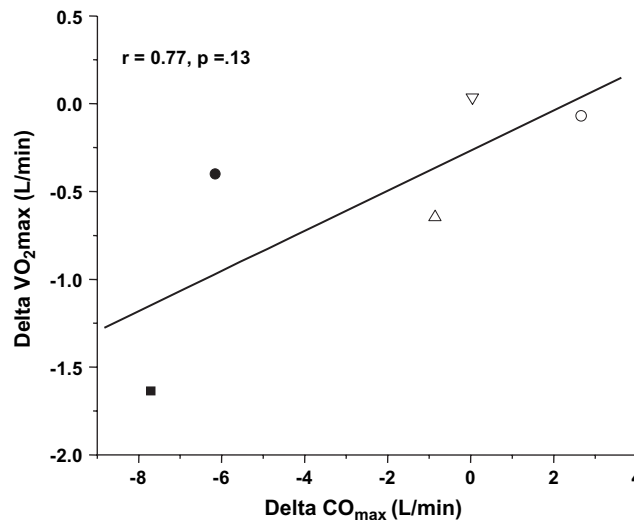


Figure 4. Association between changes in maximal cardiac output and oxygen uptake during the 10-year follow-up.

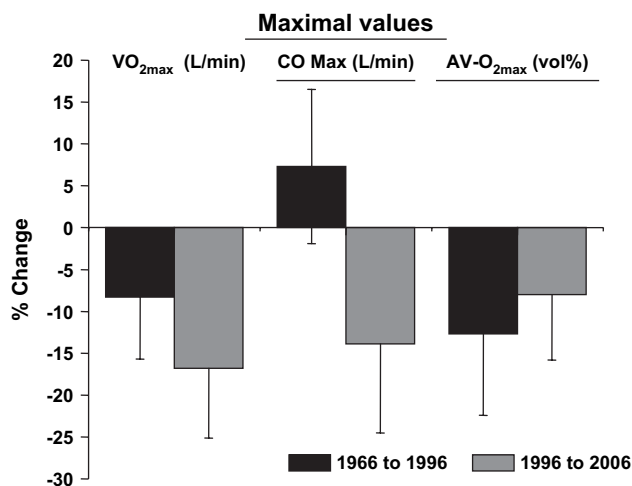


Figure 5. Comparison of changes in selected maximal variables between the 30-year and 10-year follow-up.

30-year follow-up, he had intermittently jogged and pedaled a stationary bicycle. Prior to the 2006 follow-up, participant C was not taking any medications and was cycling 3 d/wk for 30–45 minutes on a recumbent cycle ergometer. Prior to the screening visit, participant C had been diagnosed with paroxysmal atrial fibrillation and was in atrial fibrillation during his screening visit. He was treated with a calcium channel blocker and low-dose beta blocker for 6 weeks prior to performing maximal exercise testing, at which time he was in normal sinus rhythm. In the 10 years following the 1996 visit, he had lost 10 kg and 7% body fat. VO_{2max} had increased between 1966 and 1996 by 15% but decreased by 23% (2.8 vs 2.1 L/min) during the last 10-year interval, secondary to a decline in both CO_{max} (16.4 vs 15.4 L/min) and AV-DO_{2max} (16.8 vs 13.7 vol%). The decline in CO_{max} could be attributed to a decline in SV_{max} (86 vs 81 mL/beat), whereas HR_{max} remained unchanged (190 vs 190 b.p.m.).

Participant D was sedentary prior to the 1966 study. He played soccer in a semiprofessional league intermittently between 1996 and 2006 and had recently taken up cycling 1–2 d/wk. He was diagnosed with hypertension 2 years before the 1996 evaluation and was being treated with a combination of diuretic, alpha blocker, and ACE inhibitor. Following maximal exercise testing in 2006, participant D developed atrial fibrillation that was resolved within

1 month with beta-blocker therapy. During the 10-year interval, weight and body fat remained unchanged, whereas VO_{2max} was unchanged from the 1966 and 1996 visits. Accordingly, there was no change in CO_{max} (22.6 vs 22.5 L/min) or AV-DO_{2max} (11.8 vs 11.7 vol%). The decline in HR_{max} from 1996 (189 vs 171 b.p.m.) was matched by an increase in SV_{max} (120 vs 131 mL/beat).

Participant E had played football at the college and semi-professional level in 1966. He had been lifting weights 2 d/wk prior to the 1996 study but was completely sedentary prior to the 2006 visit, due to severe low back pain. Since the 1996 visit, participant E had developed hypertension and was being treated with a combination of calcium channel blocker, ACE inhibitor, and diuretic. In 2003, participant E had lap-band surgery and therefore experienced significant declines in body weight from 1996 to 2006 (163 vs 146 kg) and percent fat (30% vs 27%). Because of his persistent back pain, an exploratory MRI revealed destructive changes of his lumbar spine and he was ultimately diagnosed with metastatic renal cell carcinoma. Since the 1996 visit, VO_{2max} declined by 46% (3.5 vs 1.9 L/min). This was explained by a 28.5% decline in CO_{max} (27.0 vs 19.3 L/min) and a 25% decline in AV-DO_{2max} (13.0 vs 9.8 vol%). Although HR_{max} remained unchanged (160 vs 157 b.p.m.), SV_{max} decreased 52% (169 vs 123 mL/beat).

Participant E experienced the largest declines in maximal oxygen uptake and its determinants, due in large part to the extreme sedentary lifestyle invoked by low back associated with the renal cell carcinoma. To more accurately describe the age-related trends on cardiovascular function data at maximal exercise, we have provided a column of mean data where his values were excluded from the analysis (Table 2).

DISCUSSION

The present study is a 10-year extension of a previous 30-year follow-up and represents one of the longest follow-up studies of the determinants of VO_{2max} in nonathletes. With repeated measurements performed after the fifth decade of life, it is also one of the few studies to demonstrate changes in the age-specific decrements to the cardiovascular system during exercise.

The major finding of the previous follow-up study was that 3 weeks of bed rest resulted in a greater deterioration in cardiovascular and physical work capacity than 30 years

Table 3. Individual Changes in Maximal Oxygen Uptake and Its Determinants

Participants	VO _{2max} (L/min)			CO _{max} (L/min)			AV-DO _{2max} (vol%)		
	1966	1996	2006	1966	1996	2006	1966	1996	2006
A	2.5	2.1	2.0	18.2	15.8	18.4	13.8	12.9	10.7
B	4.2	3.7	3.3	22.8	25.0	18.8	18.2	14.6	17.3
C	2.4	2.8	2.1	17.4	16.4	15.5	13.7	16.8	13.7
D	2.6	2.7	2.7	16.0	22.5	22.4	16.5	11.7	11.8
E	4.8	3.5	1.9	25.8	27.0	22.4	18.6	13.0	9.8

Note: VO_{2max} = maximal oxygen uptake; CO_{max} = maximal cardiac output; AV-DO_{2max} = maximal arteriovenous oxygen difference.

of aging (3). Within that context, the average $\text{VO}_{2\text{max}}$ after 40 years of aging was nearly identical to the value observed after 3 weeks of bed rest (2.37 vs 2.43 L/min) in these five men.

Aging and Cardiovascular Capacity

The influence of age on maximal fitness is a controversial topic. Cross-sectional studies suggest that maximal oxygen uptake declines linearly with age at a rate of approximately 0.45 mL/kg/min (4, 6, 22–27). Short-term longitudinal studies (5–15 years) of athletes and sedentary individuals suggest that maximal oxygen uptake declines nonlinearly (7, 12) and accelerates significantly after the age of 50 or 60 years (7). The longitudinal data presented here support this concept as the annualized decline in $\text{VO}_{2\text{max}}$ doubled after the age of 50 years. This accelerated decline in fitness was evident whether it was expressed in absolute units (55 vs 10 mL/y) or relative to FFM (0.41 vs 0.23 mL/kg FFM/min/y), suggesting that the decrement in $\text{VO}_{2\text{max}}$ is related to changes in cardiovascular function and not changes in muscle mass. The age-specific differences in the rate of decline in $\text{VO}_{2\text{max}}$ observed here is remarkably similar to that reported in the segmented longitudinal follow-up of participants in the Baltimore Longitudinal Study of Aging (12 vs 42 mL/y and 0.55 vs 0.27 mL/kg FFM/min/y at 20 and 50 years of age) (7). As such it appears as though there may be an age-specific threshold at which cardiovascular and physical function begins to deteriorate exponentially. It should be noted though that if the participant who developed metastatic cancer is excluded from this analysis, the reduction in $\text{VO}_{2\text{max}}$ normalized to FFM is greatly attenuated with no difference compared with 1996 values. This finding underscores the importance of the development of comorbidities to the reduction in functional capacity with age.

Mechanistic Considerations

The relative contribution of central (CO) and peripheral (i.e., blood flow distribution and oxygen extraction) components to $\text{VO}_{2\text{max}}$ is a widely debated topic. A decline in HR_{max} is frequently cited as the predominant contributor to the decline in maximal oxygen uptake with age (7, 22, 23). Our previous report demonstrated that SV_{max} increased over time to compensate for the declining HR_{max} , thereby maintaining CO_{max} (3). As such, the decline in $\text{VO}_{2\text{max}}$ was explained entirely by a reduction in $\text{AV-DO}_{2\text{max}}$ (3). In contrast to the previous follow-up, in the present evaluations, reductions in both CO_{max} and $\text{AV-DO}_{2\text{max}}$ contributed to the reduction in $\text{VO}_{2\text{max}}$. The reduced capacity for increasing SV_{max} and CO_{max} (i.e., central cardiovascular reserve), in the face of declining peripheral oxygen extraction, may explain the accelerated decline in $\text{VO}_{2\text{max}}$ occurring after the age of 50. Indeed, the change in CO_{max} during the 10-year follow-up was closely associated with the change in $\text{VO}_{2\text{max}}$ within this small cohort ($r=0.77$; Figure 4).

Study Limitations

The present study has several limitations. Similar to the previous follow-up, this study was uncontrolled with regard to many confounding variables, including activity level, lifestyle, diet and nutrition, biology, and most importantly the development of coexistent pathology. The study design did not permit the exclusion of any of the original participants due to underlying cardiovascular disease. The superimposition of chronic disease does enhance the external validity of the data; however, it limits our ability to assess the influence of age alone on $\text{VO}_{2\text{max}}$. The significant deterioration of maximal oxygen uptake and its determinants in the one individual who effectively became bed ridden secondary to severe low back pain exemplifies this limitation. When his data are excluded from the analysis, the 10-year changes in cardiovascular function are substantially less profound. The small number of participants limits our interpretation of the independent effects of age, activity, and chronic illness on maximal cardiovascular function and precludes meaningful statistical evaluation. Finally, the study examines the changes in $\text{VO}_{2\text{max}}$ in men only, therefore limiting the generalizability of the findings.

CONCLUSIONS

In summary, maximal oxygen uptake declined at a more rapid rate in these five individuals after the age of 50 years, secondary to a decline in both CO_{max} and $\text{AV-DO}_{2\text{max}}$. The rate of decline in maximal fitness was accelerated in the 10-year interval after the age of 50 when expressed in absolute terms or relative to body weight or FFM. The decline in maximal oxygen uptake after the age of 50 years was highly individualized and dependant upon collective changes in muscle mass, habitual physical activity, and development of chronic illness. These variables working in concert likely explain the disproportionate decline in maximal fitness observed in these men after the age of 50. Impressively, the decline in $\text{VO}_{2\text{max}}$ after 40 years of life was comparable to that experienced after 3 weeks of bed rest at the age of 20 years.

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