Predicted Values for Clinical Exercise Testing[1-3]

JAMES E. HANSEN, DARRYL Y. SUE, and KARLMAN WASSERMAN

SUMMARY Following thorough evaluation at rest, 265 of 400 current or ex-shipyard workers rode a cycle ergometer with equal work increments each minute to exhaustion while continuous multiple noninvasive cardiorespiratory measures and intermittent intra-arterial blood pressure and blood gas measures were made. Seventy-seven men, with a mean age of 54, including some who were smokers, obese, or hypertensive, were judged to have normal cardiorespiratory systems based on history, physical, electrocardiogram during rest and exercise, chest X-ray, pulmonary function tests, and exercise performance. Their responses to exercise are given. It was unusual to find at maximal exercise a breathing reserve less than 11 L/min, arterial P_{O_2} less than 80 mm Hg, alveolar-arterial P_{O_2} difference greater than 38 mm Hg, arterial-end tidal P_{CO_2} difference greater than 1 mm Hg, respiratory frequency greater than 60, or a dead space/tidal volume ratio greater than 0.28. The normal anaerobic threshold/maximal O_2 uptake ratio exceeded 40%. With maximal exercise, the intra-arterial systolic and diastolic pressures rose an average of 68 and 13 mm Hg, respectively. For predicting maximal oxygen uptake and oxygen pulse in an overweight man, we find it preferable to use age and height rather than age and weight.

Introduction

To evaluate patients with exertional dyspnea, clinicians need to know the range of expected performance during exercise in normal subjects taken from an appropriate population. Because previous studies present normal values during exercise testing for athletes (1, 2, 3), faculty and associates (4, 5), military personnel (2, 6), the nonobese (1, 3, 4, 7, 8), the nonhypertensive (5), students (2, 4), and hard working laborers (7, 8), all invariably volunteers, they are not often representative of the patients requiring study for dyspnea. To define the normal responses to exercise in a more appropriate group, we reviewed exercise studies of former or current male shipyard workers without discernible pulmonary parenchymal or cardiovascular disease who exercised in an incremental fashion to their voluntary limits on a cycle ergometer. In contrast to other series, we included those with obesity, hypertension, or a history of tobacco smoking because these are commonly present in this population. However, we excluded those who appeared to be poorly motivated.

Methods

Subjects

We evaluated 400 currently or formerly employed shipyard workers, all of whom were referred by the U.S. Department of Labor over a 2-yr period because of occupational exposure to asbestos. They were evaluated by history; physical examination; posteroanterior, lateral, and oblique chest roentgenograms; 12 lead resting electrocardiograms; and routine respiratory function tests including spirometry, lung volumes, and diffusing capacity. Two hundred fifty-seven men underwent incremental exercise testing to tolerance on the cycle ergometer with cardiorespiratory measurements and an indwelling brachial artery catheter for blood gas and blood pressure measurements.

From these tests, 77 men were identified as not having abnormal cardiovascular or pulmonary function at rest or exercise. Individuals with the following were excluded: history or evidence of myocardial infarction, valvular heart disease, cardiomegaly, coronary bypass surgery, peripheral vascular disease, treatment with beta blockers, quinidine, or digitalis, or depression of ST segments of 1 mm or more during exercise; interstitial lung disease by auscultation or roentgenograms, obstructive or restrictive lung disease by pulmonary function testing, pulmonary vascular disease, or chest wall disease; neuromuscular, skeletal disease, or poor motivation limiting exercise; malignancy; cirrhosis; resting metabolic acidosis or unregulated diabetes mellitus. However, men were not excluded because of a history of tobacco smoking, exertional dyspnea or cough; findings of obesity, systemic hypertension, pleural thickening or plaques; or hypoxemia at rest or exercise.

Resting Respiratory Function

A 9 L Collins water-filled spirometer was used for spirometry and single breath diffusing capacity for carbon monoxide (D_{LCO}) measurement, the latter by the method of McGrath and Thompson (9). The functional residual capacity (FRC) was measured by nitrogen washout for 7 min, correcting for nitrogen washed out from the tissues (10).

We used predicting equations from the following series: Knudson and associates (11) for vital capacity (VC) and forced expiratory volume in one second (FEV_1); Goldman and Becklake (12) for residual volume (RV) and total lung capacity (TLC); Boren and colleagues (13) for expiratory reserve volume (ERV)/inspiratory capacity (IC); Kory and coworkers (14) for maximal voluntary ventilation (MVV); and Cotes (15) for single breath diffusing capacity for carbon monoxide (D_{LCO}). For Asians and blacks, the predicted volumes, flow rates, and D_{LCO} were reduced by 10%. The $FEV_1 \times 40$ was also used as an indirect estimate of MVV.

Exercise Study Methods

Subjects exercised on a stationary, calibrated, electrically-braked cycle (Monark, Quinton, Seattle, WA, or Godart, Bilthoven, Netherlands). After 2 to 5 min of rest and 2 to 4 min of pedaling at 0 watts, an incremental work rate test was performed during which work rate was increased by a fixed amount between 10 and 30 watts every min until the subject could no longer continue. During exercise, subjects breathed through a low dead space breathing valve (Edward Koegel Co., San Antonio, TX) whose expiratory outlet was connected to a Fleisch-type #3 pneumotachygraph. Expired minute ventilation ($\dot{V}E$), oxygen uptake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$), end tidal P_{O_2} and P_{CO_2} (PET_{O_2}, PET_{CO_2}), and respiratory gas exchange ratio (R) were measured over 15 to 20 s and displayed every 30 s as previously reported (16). Oxygen pulse in ml/beat was calculated from $\dot{V}O_2$/heart rate.

Prior to exercise, a 25-cm 5F polyethylene catheter (Universal Medical Instrument Corp., Ballston Spa, NY) was introduced into the brachial artery with the Seldinger technique for intermittent blood sampling and measurement of blood pressure using a microtransducer (Micron Instrument Inc., Los Angeles, CA) and multichannel recorder (Beckman Instruments, Fullerton, CA). Arterial blood samples were drawn into heparinized syringes over a 20-s period while expired gases were analyzed simultaneously by the computerized system. Blood samples were immediately placed in ice and analyzed within 30 min using a blood gas analyzer (Radiometer ABL-1, Copenhagen) calibrated with tonometered blood. The $P(A\text{-}a)O_2$ and dead space/tidal volume ratio (V_D/V_T) were calculated as previously described (17).

The maximal $\dot{V}O_2$ ($\dot{V}O_2$max), maximal expired ventilation (VEmax), maximal heart rate (HR max), and maximal O_2 pulse (O_2 pulse max) were selected as the highest

[1] From the Division of Respiratory Physiology and Medicine, Department of Medicine, Harbor-UCLA Medical Center, UCLA School of Medicine, Torrance, California.

[2] Supported in part by Department of Labor and NIH Grants HL-11907 and HL-07388.

[3] Requests for reprints should be addressed to James E. Hansen, M.D., Harbor-UCLA Medical Center, Box 24, 1000 West Carson St., Torrance, CA 90509.

TABLE 1

ANTHROPOMETRIC AND RESTING RESPIRATORY FUNCTION DATA OF 77 MALE SHIPYARD WORKERS

	Mean	SD	Range	% Mean Predicted
Age, yr	54.3	9.2	34 to 74	
Weight, kg	86.5	16.3	53 to 124	
Height, cm	172.0	7.7	155 to 189	
Vc, L	4.15	0.69	2.78 to 5.90	103
ERV, L	0.89	0.39	0.18 to 1.90	66*
RV, L	2.09	0.54	0.63 to 3.40	106
TLC, L	6.25	0.99	3.63 to 7.67	104
FEV_1, L	3.28	0.55	2.08 to 5.06	103
MVV, L/min	131.0	23.6	81 to 203	97
D_{LCO}, ml/mm/Hg/min	29.8	4.5	20.7 to 41.6	112*
Hematocrit, %	45.8	3.3	37 to 53	
Asbestos exposure, yr	21.3	10.0	2 to 40	

* Significantly different from predicted at $p < 0.001$ level by paired *t* test; these are due to bias in the mean values because of obesity; all others not significant at $p > 0.05$.

values obtained from any 20-s measurement period. The ventilatory equivalents for O_2 and CO_2 ($\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$), were calculated BTPS/STPD with valve dead space subtracted from each breath. The anaerobic threshold (AT) was chosen at the $\dot{V}O_2$ at which the $\dot{V}E/\dot{V}O_2$, $P_{ET}O_2$, and R increased, while $\dot{V}E/\dot{V}CO_2$ and $P_{ET}CO_2$ decreased or remained constant (18).

Predicted Exercise Values

We used the equation of Bruce and associates (19) to relate normal weight to height: $W = 0.79\ H - 60.7$, where W = normal weight in kg and H = height in cm. For predicted $\dot{V}O_2$max, we used 90% of Bruce's treadmill $\dot{V}O_2$ max values in his sedentary male population: $\dot{V}O_2$ max in ml/min = $W \times (50.75 - 0.372A)$, where A = age in years and W = weight in kg, actual or normalized if overweight (19). We predicted HR max as: HR max (beats/min) = 210 − 0.65 A (20).

Paired *t* tests were used to compare actual resting respiratory function values with predicted values. We used analyses of variance with completely randomized design or repeated measures design and the Tukey test to compare differences between groups (21). Single or multiple regression analyses were also employed (22). We accepted $p < 0.05$ to indicate a significant difference.

Results

Resting Data

As seen in table 1, the subjects ranged in age from 34 to 74 yr, 53 to 145 kg, and 155 to 189 cm, with an average asbestos exposure of 21 yr. Pleural thickening was detected in 47 of the 77. The individual and group resting respiratory function values were normal except for a decrease in ERV and increase in D_{LCO} in the more obese. On the basis of their individual weight to height relationships, the subjects were separated into 5 groups: group A in which weight was lower than that predicted by height; and groups B, C, D, and E in which weight was, respectively, up to 110, 120, 130, and 160% of predicted (table 2 and figure 1). Age, height, smoking history, and flow rates were not significantly different between groups, but the ERV/IC ratio decreased and the D_{LCO} increased significantly with increasing weight. There was a slight difference in average asbestos exposure between the groups.

Oxygen Uptake and Transport

The $\dot{V}O_2$ max was significantly higher in the most obese group (table 3). However, there was a significant decrease in percent of predicted $\dot{V}O_2$ max with increasing weight when predicted $\dot{V}O_2$ max was based on the individual's actual weight. No significant differences in percent predicted $\dot{V}O_2$ max were found when the predicted $\dot{V}O_2$max was based on *height*; the measured $\dot{V}O_2$ max of the obese groups were very close to the predicted $\dot{V}O_2$ max normalized for height. In contrast, the underweight individuals had $\dot{V}O_2$ max values closer to predicted $\dot{V}O_2$ max when each person's actual, rather than predicted, weight was used.

In figure 2, the $\dot{V}O_2$ max values of all of the overweight individuals are compared using the predicted equations based on weight and age (figure 2A) and on height and age (figure 2B). In the comparison based on height, the values are evenly distributed about the line of identity, whereas an obvious difference is evident when actual weight is used.

The absolute values of $\dot{V}O_2$ max and O_2 pulse max (table 4) are higher in the most obese. There is no significant difference between groups when predicting equations for $\dot{V}O_2$ max are based on age and height in those overweight and on age and weight in those underweight.

The HR max was not different between groups. Nevertheless, we found that the HR max declines not only with age but also bears some relationship to weight. By multiple regression analysis: HR max = 226 − 0.78 A − 0.252 W, r = 0.406.

The mean ratio of the $\dot{V}O_2$ at the AT and $\dot{V}O_2$ max was 0.56, with a standard deviation of 0.08 and a range of 0.40 to 0.78 (figure 3).

During unloaded pedaling, $\dot{V}O_2$, in ml/min was related to body weight (W, kg) by the formula: $\dot{V}O_2 = 5.89\ W + 140$. The increase in $\dot{V}O_2$ per increase in work rate was 9.3 ± 1.35 ml/min/watt. The mean $\Delta\dot{V}O_2$/work rates of groups A through E were not significantly different: 9.1, 9.4, 9.5, 8.9, and 9.6 ml/min/watts, respectively.

Blood Pressure

Sixty-nine of the 77 men had technically

TABLE 2

SHIPYARD WORKERS SEPARATED INTO 5 GROUPS ON BASIS OF WEIGHT AND HEIGHT*

Groups	A	B	C	D	E	Significance†
Predicted wt, %‡	79 to 99	100 to 109	110 to 119	120 to 129	130 to 160	
n	14	19	18	13	13	
Race (A,B,W)§	2,0,12	4,1,14	3,4,11	1,5,7	1,1,11	
Age, yr	53 ± 10	54 ± 10	57 ± 11	56 ± 11	51 ± 10	NS
Weight, kg	68 ± 8	78 ± 7	86 ± 5	96 ± 8	111 ± 14	All but B versus C
Height, cm	170 ± 8	170 ± 8	172 ± 6	173 ± 10	175 ± 6	NS
Asbestos exposure, yr	26 ± 11	22 ± 10	23 ± 11	16 ± 6	17 ± 9	A versus D
Cigarette, pack yr	13 ± 17	13 ± 12	19 ± 17	8 ± 8	15 ± 13	NS
FEV_1/VC, %	78 ± 4	80 ± 5	78 ± 4	79 ± 4	80 ± 4	NS
ERV/IC, %	44 ± 15	32 ± 10	22 ± 9	25 ± 8	17 ± 18	A versus B,C,D,E and B versus E
D_{LCO}, ml/min/mmHg	29 ± 5	28 ± 4	29 ± 3	32 ± 4	33 ± 5	E versus A,B,C
MVV, L/min	138 ± 26	129 ± 20	131 ± 23	124 ± 26	134 ± 25	NS

* Values are mean ± SD.
† Significant difference between groups at $p < 0.05$; NS = not significant.
‡ Predicted weight in kg = 0.79 × height (cm) − 60.7
§ A,B,W = Asian, black, white.

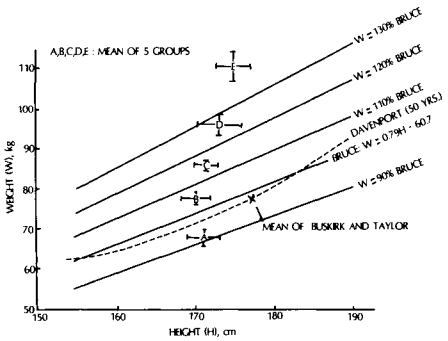

Fig. 1. The relationships between weight and height in several male populations. Values of Bruce and associates (19), Davenport (23), and Buskirk and Taylor (2) are depicted plus the mean ± SEM of the 5 groups of this study (*see* text).

TABLE 3

$\dot{V}O_2$ MAX OF 77 SHIPYARD WORKERS ON CYCLE ERGOMETER*

Group	A	B	C	D	E	Significance†
Predicted weight, %‡	77 to 99	100 to 109	110 to 119	120 to 129	130 to 160	
$\dot{V}O_2$ max, L/min STPD	2.13 ± 0.41	2.17 ± 0.26	2.15 ± 0.43	2.21 ± 0.44	2.63 ± 0.43	E versus A,B,C,D
$\dot{V}O_2$ max, % predicted§ (using actual weight)	101 ± 15	91 ± 8	85 ± 16	77 ± 7	76 ± 13	A versus C,D,E B versus D,E
$\dot{V}O_2$ max, % predicted (using predicted weight)	93 ± 16	96 ± 8	98 ± 18	97 ± 9	107 ± 14	NS

* Values are mean ± SD.
† Significant at $p < 0.05$; NS = not significant.
‡ Predicted weight in kg = 0.79 cm − 60.7.
§ Predicted $\dot{V}O_2$ max in ml/min = actual or predicted weight × (50.75 − 0.372 yr).

satisfactory intra-arterial blood pressure measurements throughout their study. Thirteen subjects had systolic pressure over 140 mm Hg or diastolic pressure over 90 mm Hg when measured during their initial clinical evaluation and were not receiving antihypertensive drug therapy; 13 others were receiving such therapy, while 43 were considered to be normotensive (table 5). Resting intra-arterial systolic and diastolic blood pressures were higher than previously measured cuff pressure in all subjects. From rest to maximal exercise, the directly recorded systolic pressure rose a mean of 68 mm Hg (65 mm Hg in the normal subjects and 72 mm Hg in all hypertensive subjects) and the directly recorded diastolic pressure rose a mean of 13 and 12 mm Hg in the normal subjects and hypertensive subjects, reaching the highest values at maximal exercise.

Ventilatory Pattern

There was an increase in $\dot{V}E$, tidal volume (VT), and respiratory frequency (f) from rest, to 0 watts exercise, to exercise at the anaerobic threshold, and to maximal exercise (table 6). The VT increased more than f as exercise increased up to the AT. The most common pattern thereafter was an increase in f as VT approached 2/3 of the IC. In no case did the VT exceed the IC; in only 3 men did f exceed 60. The maximal VT/VC was 55.0 ± 8.7% (mean ± SD) with a coefficient of variation of 15.8%. The mean maximal VT/IC was 70.0 ± 10.7% with a coefficient of variation of 15.3%.

As work increased, there was a progressive fall in the $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ (table 7). Both values reached their nadir near the AT; thereafter the $\dot{V}E/\dot{V}O_2$ rose strikingly.

For the entire group, both the mean directly measured MVV and the mean indirectly calculated MVV were 131 L/min. The $\dot{V}E$ max/MVV averaged 71.5%, whereas when the $\dot{V}E$ max of each individual was compared to the larger of MVV, either directly or indirectly, the mean ± SD percentage was 66.6% ± 13.8%. The exercise breathing reserve (MVV − $\dot{V}E$ max) averaged 38 L/min, whereas when the larger of direct or indirect MVV was used the mean ± SD breathing reserve was 47 ± 23 L/min. The breathing reserve was less than 11 L/min in only 3 men, one each in groups C, D, and E.

Pulmonary Gas Exchange

There was a significant increase in $P(A\text{-}a)O_2$ and Pa_{O_2} with increasing exercise with the highest mean Pa_{O_2} (101 mm Hg) and largest mean $P(A\text{-}a)O_2$ (19 mm Hg) near maximal exercise (table 8). The Pa_{O_2} at the AT was 75 to 79 mm Hg in 6 men and at maximal exercise was below 80 mm Hg in only 1 man: a thin 55-yr-old heavy smoker (figure 4). In 3 subjects, the $P(A\text{-}a)O_2$ at the AT exceeded 28 mm Hg and at maximal exercise exceeded 35 mm Hg (figure 5).

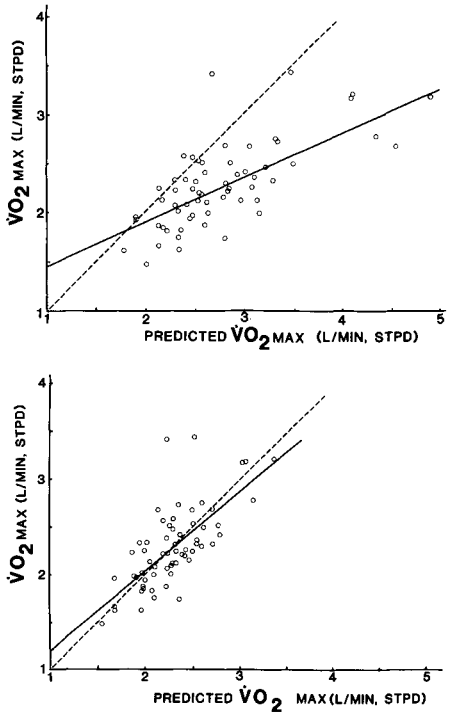

Fig. 2. Comparison of predicted and actual $\dot{V}O_2$ max on the cycle ergometer in 63 overweight men (groups B,C,D, and E). A. The dotted line of identity is based on age in yr (A) and weight in kg (W), where predicted $\dot{V}O_2$ max in L/min = 0.001 W (50.75 − 0.372 A) (19). The solid line equation of actual (Y) to predicted (X) $\dot{V}O_2$ max is Y = 0.459X + 0.997. Note that actual values are less than predicted in 56 men. B. The dotted line of identity is based on age in yr (A) and height in cm (H), where predicted $\dot{V}O_2$ max in L/min = 0.001 (0.79H − 60.7) (50.75 − 0.372A), or 0.0401H + 0.0226A − 0.000294AH − 3.08. Solid line equation is Y = 0.827X + 0.366. Note better distribution of actual and predicted values near the line of identity when height is used rather than weight.

The Pa_{O_2} was not different between weight-determined groups at rest or at maximal exercise (table 9). However, the $P(A\text{-}a)O_2$ at rest was significantly higher

TABLE 4

$\dot{V}O_2$ MAX, HR MAX, AND O_2 PULSE MAX DURING CYCLE ERGOMETER EXERCISE*

	A	B	C	D	E	Significance†
Predicted weight, %	77 to 99	100 to 109	110 to 119	120 to 129	130 to 160	
$\dot{V}O_2$ max, ml/min	2,130 ± 410	2,170 ± 260	2,150 ± 430	2,210 ± 440	2,630 ± 430	E versus A,B,C,D
HR max, beats/min	167 ± 20	165 ± 14	156 ± 20	150 ± 22	163 ± 12	NS
HR max, % predicted‡	95 ± 1	94 ± 7	90 ± 11	86 ± 11	92 ± 7	NS
O_2 pulse max, ml/beat	13.1 ± 2.6	13.5 ± 1.7	14.0 ± 2.2	14.9 ± 2.4	16.5 ± 2.8	E versus A,B,C
Compared to $\dot{V}O_2$ Max Predicted from Actual Weight and Age						
$\dot{V}O_2$ max, % predicted	101 ± 15	91 ± 8	85 ± 16	77 ± 7	76 ± 13	A versus C,D,E B versus D,E
O_2 pulse max, % predicted	109 ± 14	99 ± 8	96 ± 16	91 ± 15	84 ± 11	A versus D,E B versus E
Compared to $\dot{V}O_2$ Max Predicted from Predicted Weight and Age in Obese§						
$\dot{V}O_2$ max, % predicted		96 ± 8	98 ± 18	97 ± 9	107 ± 14	NS
O_2 pulse max, % predicted		104 ± 8	110 ± 18	115 ± 20	118 ± 13	NS

* Values are mean ± SD.
† Significant difference between groups at $p < 0.05$; NS = not significant.
‡ Predicted HR max = 210 − 0.65 yr.
§ By weight in group A and height in groups B,C,D,E.

TABLE 5

INTRA-ARTERIAL SYSTOLIC AND DIASTOLIC PRESSURES AT REST AND EXERCISE*

Group	n	Systolic/ Diastolic	Prior Cuff (C)	Rest (R)	0 Watts (O)	Anaerobic Threshold (A)	Maximal Exercise (M)	Significance†
Normal	43	S	124 ± 11	142 ± 18	159 ± 20	182 ± 23	207 ± 27	All
		D	79 ± 7	86 ± 10	89 ± 11	92 ± 11	99 ± 12	All but O versus R,A
Treated Hypertensive	13	S	142 ± 16	149 ± 14	174 ± 23	196 ± 17	220 ± 20	All but C versus R
		D	91 ± 14	97 ± 14	98 ± 15	102 ± 11	107 ± 12	C versus A,M R versus M
Untreated Hypertension	13	S	150 ± 8	161 ± 12	184 ± 18	203 ± 25	235 ± 20	All but C versus R
		D	90 ± 8	96 ± 11	98 ± 8	103 ± 10	110 ± 10	C versus O,A,M M versus R,O,A

* Values are mean ± SD.
† Significant difference between groups at $p < 0.05$.

with obesity, 18 mm Hg in the most obese and 10 mm Hg in the 2 least obese groups. At maximal exercise the difference in P(A-a)O_2 between groups was not significant.

The arterial-end tidal P_{CO_2} (P[a-ET]CO_2) became increasingly negative with exercise (table 8 and figure 6). The P(a-ET)CO_2 was negative at the AT in all but 2 men and at maximal exercise in all but 1 man; in these cases P(a-ET)CO_2 was 1 mm Hg. The mean V_D/V_T declined from 0.30 at rest to 0.19 at the AT and maximal exercise. In 4 studies, the V_D/V_T exceeded 0.30 at the AT; in only one person, 72 yr of age, did the V_D/V_T exceed 0.28 at maximal exercise (figure 7). There was no difference based on obesity between the group values of Pa_{CO_2}, pH, V_D/V_T, and bicarbonate at rest or maximal exercise.

Cessation of Exercise

The subjects were questioned in a nonleading fashion about the reason for stopping exercise as soon as they came off the mouthpiece following exercise. Forty-six gave a single reason for cessation as follows: general fatigue or exhaustion in 25, shortness of breath in 9, leg fatigue in 8, and leg pain in 4. (Of those citing shortness of breath, 2 subjects, 69 and 74 yr of age, in groups C and D, had HR max of 132 and 117, a decrease in bicarbonate from rest of only 3–4 meq/L, and a large breathing reserve of 40 L.) Of the 25 individuals who gave multiple reasons, the reasons were as follows: shortness of breath and leg fatigue in 13; shortness of breath and general exhaustion in 6; shortness of breath, general and leg fatigue in 3; leg and general fatigue in 2; and shortness of breath and leg pain in 1. The reasons for stopping exercise were not obtained in 6 individuals.

We examined the decrease in bicarbonate from rest to recovery and the R at $\dot{V}o_2$ max as estimates of maximal effort. The difference between resting bicarbonate and bicarbonate after 2 min of recovery (mean ± SD) was 8.5 ± 2.9 meq/L. The R at the time of $\dot{V}o_2$ max was 1.21 ± 0.12, whereas the highest R during 2 min of recovery was 1.59 ± 0.19. We found that R at $\dot{V}o_2$ max was related to the difference between resting and recovery bicarbonate in meq/L (B) by $B = 14.41R - 8.97$, $r = 0.615$ (figure 8). Seven of 77 men (3 each in groups B and C and 1 in group D) had a change in bicarbonate of 4 meq/L or less. We found no statistically significant difference in change in bicarbonate or R at Vo_2 max between groups divided by weight.

Discussion

Our population of 77 current or former shipyard workers without apparent heart or lung disease differs from other populations studied at maximal exercise (1–8, 24–28). They were not volunteers affiliated with universities or athletic programs (3–5, 27) although they did give informed consent. In addition, our incidence of obesity and hypertension was higher than in other series of older individuals (1,4,5,19,24,27,28).

Forty-seven of the 77 men had evidence of pleural thickening without parenchymal disease on oblique roentgenograms, but the normal VC and TLC, and the absence of rales, rhonchi, decreased FEV_1, or low D_{LCO} should eliminate significant parenchymal disease. The higher D_{LCO} and lower ERV in the obese groups are typical of obesity (29), not parenchymal lung disease.

Twenty-five years ago, Buskirk and Taylor demonstrated "that the presence of excess fat per se does not have any important influence on the capacity of the cardiovascular-respiratory system to deliver oxygen to muscles under maximal performance conditions" (2). An adult who gains 5 to 40 kg of fat should not be expected to improve his respiratory or cardio-

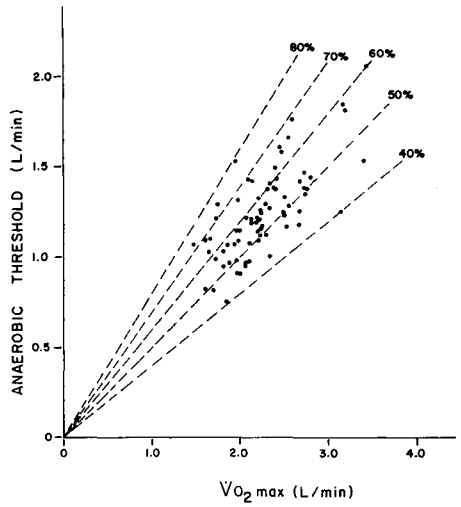

Fig. 3. Comparison between $\dot{V}o_2$ at maximal exercise and at the anaerobic threshold in 75 men.

TABLE 6

VENTILATORY PATTERN IN 77 SHIPYARD WORKERS*

	Rest	0 Watts	Anaerobic Threshold	Maximal Exercise
$\dot{V}_E$, L/min	11.8 ± 4.5	20.0 ± 5.5	33.7 ± 8.9	93.3 ± 23.0
V_T, L	0.71 ± 0.26	0.97 ± 0.26	1.44 ± 0.43	2.28 ± 0.43
f, breaths/min	17.6 ± 5.9	22.1 ± 8.4	25.4 ± 11.2	41.6 ± 9.6

* Values are mean ± SD.

TABLE 7

VENTILATORY EQUIVALENTS FOR O_2 AND CO_2 AT REST AND EXERCISE*

	Rest (R)	0 Watts (O)	Anaerobic Threshold (A)	Maximal Exercise (M)	Significance†
$\dot{V}_E$ (BTPS)/$\dot{V}O_2$ (STPD)	32.8 ± 12.1	28.5 ± 8.1	26.5 ± 4.4	37.7 ± 6.9	All but O versus A
$\dot{V}_E$ (BTPS)/$\dot{V}CO_2$ (STPD)	39.3 ± 9.7	33.4 ± 7.4	29.1 ± 4.3	31.9 ± 4.4	All but O versus M

* Values are mean ± SD.
† Significant difference between groups at $p < 0.05$.

TABLE 8

ARTERIAL BLOOD AND LUNG GAS EXCHANGE DURING EXERCISE*

	Rest (R)	0 Watts (O)	Anaerobic Threshold (A)	Maximal Exercise (M)	Significance†
Pa_{O_2}, mm Hg	94.8 ± 10.5	95.2 ± 10.9	92.1 ± 8.4	100.6 ± 9.9	M versus R,O,A
$P(A\text{-}a)_{O_2}$, mm Hg	12.8 ± 7.4	13.1 ± 7.2	16.5 ± 7.3	19.0 ± 8.8	All but R versus O
Pa_{CO_2}, mm Hg	35.5 ± 5.3	36.5 ± 5.3	38.3 ± 4.4	34.3 ± 4.4	All but R versus O
$P(a\text{-}ET)_{CO_2}$, mm Hg	−0.3 ± 2.9	−1.9 ± 2.7	−3.7 ± 3.3	−4.1 ± 3.2	All but A versus M
V_D/V_T	0.30 ± 0.08	0.24 ± 0.07	0.20 ± 0.07	0.19 ± 0.07	All but A versus M
V_D, ml	202 ± 67	236 ± 95	300 ± 114	404 ± 158	All but R versus O
pH, units	7.43 ± 0.04	7.42 ± 0.04	7.40 ± 0.03	7.37 ± 0.04	All
Hco_3^-, meq/L	23.3 ± 2.4	23.1 ± 2.9	23.2 ± 2.3	19.3 ± 2.5	M versus R,O,A

* All values are mean ± SD.
† At $p < 0.05$.

vascular function. Also, during a physical conditioning program, an adult is as likely to lose weight as gain weight. Our older patients are far more obese than volunteers evaluated by most exercise physiologists. Although muscle weight correlates with lean body weight, estimation of the latter by densitometry requires a very accurate measurement of lung volume, an assumed quantity and density of bone, and gives relative rather than absolute values of muscle weight (30).

Therefore, we theorized that height, rather than weight or complicated estimates of obesity, should be used with age and sex as predictors of expected values of cardiorespiratory performance in the mildly to severely obese. For respiratory physiologists this concept should not be startling. For example, the VC varies widely in the adult population, but it has been apparent since the work of Hutchinson that it is predominantly determined by sex, age, and height (31). Although race and altitude of origin are also important, the addition of body weight to predicting equations for VC has rarely been found to reduce variability (32). Even in cardiovascular physiology, the heart and thoracic cage dimensions are often compared; the latter does not change appreciably with changes in body weight. Nevertheless, we are unaware of any series in adults that directly uses height as a predictor of $\dot{V}o_2$ max or maximal O_2 pulse.

Bruce and coworkers (19) showed the relationship between height and weight in their middle-age population and allowed us to test our theory. Two years ago, we began to use his equations based on a sedentary male population. As $\dot{V}o_2$ max on the cycle varies between 89 and 95% of treadmill values (3, 25–27, 33–36), we used 90% of his treadmill exercise values for cycle exercise. We used height to estimate normal weight and used the normalized weight in all those above actual weight. We used actual weight if it were less than the normalized weight. As shown in figure 2, $\dot{V}o_2$ max in only 2 of the 77 individuals differed widely from the predicted $\dot{V}o_2$ max calculated in this way. Both subjects were very active; one claimed to walk an average of 50 miles per wk, and the other bicycled 70 miles per wk. In contrast, $\dot{V}o_2$ max was poorly predicted from age and weight in obese subjects. Thus, we conclude that expected cardiovascular performance in the usual sedentary and obese patient should be based, not only on the exercise instrument and his age, but on his height rather than weight.

The amount of $\dot{V}o_2$ required to perform external work is necessarily dependent on the $\dot{V}o_2$ required for movement of the body. In cycle ergometry pedaling at 0 watts, the $\dot{V}o_2$ for a 120 kg man is likely to be 847 ml/min, whereas that for a 60 kg man is only 493 ml/min. For increasing loads, the increase in $\dot{V}o_2$ is similar regardless of body weight. The measured O_2 requirements for cycle ergometry are very similar in this population to those previously reported by Wasserman and Whipp (18). The $\dot{V}o_2$ is higher at low work rates and lower at high work rates than those recently suggested by an ATS committee for evaluation of impairment and disability (37).

The mean HR max of 160/min was lower than those of most series and might be due to low motivation, heart disease, age, unfitness, or obesity. We felt that lactate during recovery, as estimated from plasma bicarbonate levels, could assess motivation. Astrand (24) reports that maximal recovery lactate values decline with age: from 11 to 12 meq/L in subjects 30 to 39 yr of age to 7 to 8 meq/L in subjects 50 to 65 yr of age. Although we did not measure lactate directly, the mean fall in HCO_3^- of 8.4 meq/L is comparable for the ages of our subjects and suggests that our subjects were generally well motivated. Powles and associates (38) have described a low HR max in some patients following myocardial infarction, but our evaluees had no angina or abnormal EKG at rest or exercise, making this diagnosis unlikely. The HR max declines with age in all series; the predicted HR max at age 54 ranges from 182 (39) to 174 (20) to 168 (40) to 166 (41). Cooper (42) found at age 54 a mean HR max of 178 in the fit and 163 in the unfit. Finally, we found that our obese men tended to have a lower HR max. Thus, we believe that our lower mean HR max is most likely due to obesity.

The R at $\dot{V}o_2$ max correlates well with the change in bicarbonate accompanying exercise, suggesting that it may be noninvasive measure of motivation. Specifically, an R over 1.05 at the end of exercise suggests satisfactory motivation during the exercise test. The converse is not necessarily true. For example, patients who are ventilatory limited may be well motivated but unable to work hard enough to develop a metabolic acidosis or unable to eliminate the excess CO_2 associated with the metabolic acidosis.

Differences between resting brachial in-

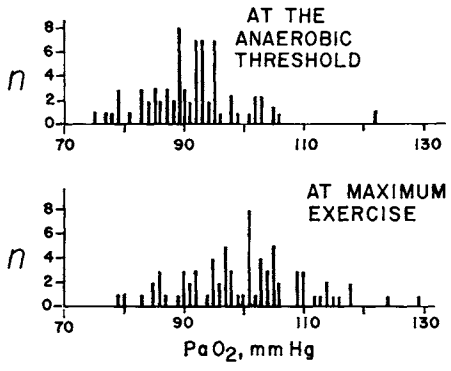

Fig. 4. Pa_{O_2}, in mm Hg, at the anaerobic threshold and at maximal exercise in 74 men.

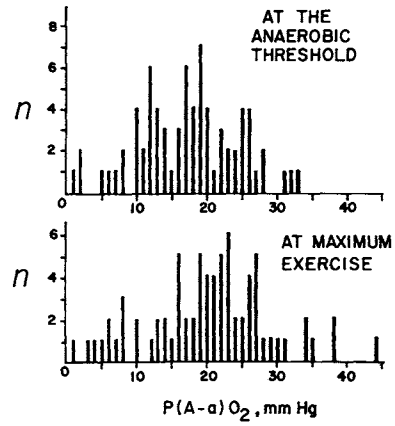

Fig. 5. $P(A\text{-}a)_{O_2}$, in mm Hg, at the anaerobic threshold and at maximal exercise in 74 men.

TABLE 9

INFLUENCE OF OBESITY ON OXYGENATION*

Group	A	B	C	D	E	Significance†
Predicted weight, %	77 to 79	100 to 109	110 to 119	120 to 129	130 to 160	
Pa_{O_2}, mm Hg						
Rest	95 ± 8	95 ± 12	98 ± 10	96 ± 13	90 ± 7	NS
Maximal exercise	105 ± 10	105 ± 10	99 ± 7	97 ± 12	98 ± 12	NS
$P(A\text{-}a)_{O_2}$, mm Hg						
Rest	10 ± 8	10 ± 5	12 ± 6	14 ± 6	18 ± 8	E versus A and B
Maximal exercise	17 ± 9	17 ± 10	19 ± 8	21 ± 7	23 ± 9	NS

Values are mean ± SD.
† Significant difference between groups at $p < 0.05$; NS = not significant.

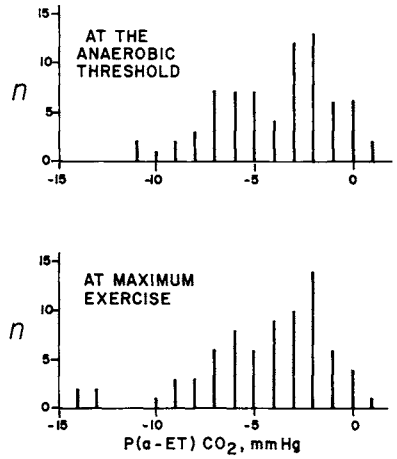

Fig. 6. $P(a\text{-}ET)_{CO_2}$, in mm Hg, at the anaerobic threshold and at maximal exercise in 73 men.

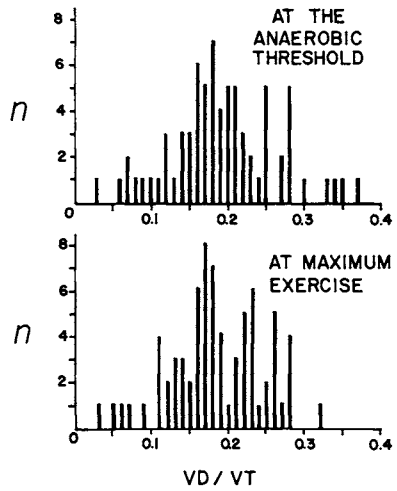

Fig. 7. VD/VT at the anaerobic threshold and at maximal exercise in 72 men.

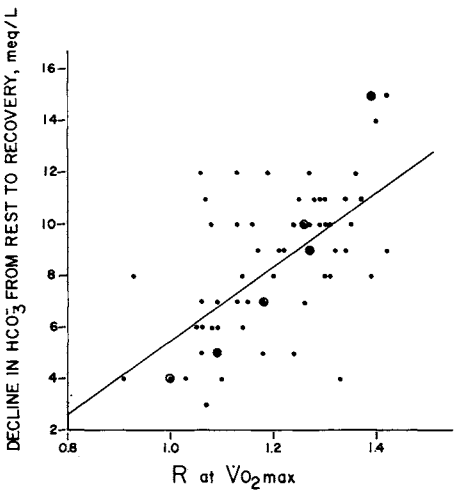

Fig. 8. The relationship between R at $\dot{V}O_2$ max and decline in HCO_3^- (B) in meq/L from rest to 2 min of recovery in 72 men. B = 14.41R − 8.97, r = 0.615.

tra-arterial and arm cuff pressures has been noted previously (43). During incremental treadmill exercise, Bruce and coworkers (44) found a rise in cuff systolic pressure of 62 ± 19 mm Hg in normal men, with significantly lesser rise in hypertensive men without demonstrable coronary artery disease (56 ± 24 mm Hg), men with previous myocardial infarction (38 ± 23 mm Hg), and men with angina (40 ± 26 mm Hg). They also found a fall in cuff diastolic pressure of 6 and 2 mm Hg in normal and hypertensive groups and a mean rise of 2 to 5 mm Hg in groups with detectable coronary artery disease. We found equivalent rises in systolic pressure in nonhypertensives (65 mm Hg) and hypertensives (72 mm Hg) suggesting that there may be a higher incidence of subclinical coronary artery disease in Bruce's series than ours. The universal and significant rise in diastolic intra-arterial pressure in our series and the decline in diastolic cuff pressure in Bruce's normal and hypertensive subjects may relate to artifacts in auscultory or intra-arterial measurement or to a difference between treadmill and cycle exercise (45).

Predicted values are most useful when they discriminate between the normal and abnormal or among disease states. We were especially interested in the normal ranges of the AT, breathing reserve, ventilatory pattern, $P(A\text{-}a)O_2$, and VD/VT.. The AT is often low in patients with circulatory disorders. We report the AT in units of $\dot{V}O_2$ rather than watts or external work because, during incremental exercise, the $\dot{V}O_2$ at a given work rate is dependent on the rate of increase in work rate. All of our normal 77 men had an AT of 40% or more of their actual or predicted $\dot{V}O_2$ max, suggesting that this is a reasonably clear minimal AT in healthy older men.

Dyspnea was a common symptom in the 400 shipyard workers evaluated. As dyspnea is subjective, the value of an objective measurement of ventilation during exercise is obvious. Despite the frequent symptom of shortness of breath in this population as a cause of exercise cessation, we rarely found a breathing reserve less than 15 L/min. This suggests that only the normal subject with unusual motivation or the patient with true ventilatory impairment approaches or exceeds his MVV. We believe that the MVV will be reached or exceeded by the $\dot{V}E$ max only when: (*a*) the MVV was improperly performed, (*b*) the person is exceptionally well motivated despite considerable metabolic acidosis, or (*c*) the patient has true ventilatory impairment. Calculating the indirect MVV as 40 times the FEV_1 is a good check on the reliability of the directly measured MVV, but should not be a substitute, as inspiratory obstruction and myasthenia gravis or other neuromuscular diseases may reduce the directly measured MVV more than predicted from the expiratory spirogram or FEV_1 alone.

In terms of ventilatory pattern, we found that the normal individual relies predominantly on increasing his frequency to increase his $\dot{V}E$ as he approaches exhaustion, but rarely exceeds a frequency of 60 and never has a VT exceeding his IC. This pattern minimizes ventilatory work (46) and maintains a relatively high alveolar ventilation fraction (low VD/VT). By contrast, in patients with parenchymal restrictive disease we have often seen frequency exceed 100. As metabolic requirements increase from rest to low level work, we found an increasing efficiency in oxygen uptake/total ventilation (decline in $\dot{V}E/\dot{V}O_2$). Thereafter, as exercise increases and metabolic acidosis develops, there is a striking rise in $\dot{V}E/\dot{V}O_2$ from 26.5 to 37.7 as $\dot{V}E$ rises approximately 42% more than $\dot{V}O_2$. This pattern of low $\dot{V}E/\dot{V}O_2$ until the AT is surpassed and then increasing $\dot{V}E/\dot{V}O_2$ may not occur in patients with pulmonary disease who cannot reach higher work levels.

The resting Pa_{O_2} and Pa_{CO_2} are obviously affected by voluntary or anxiety provoked hyperventilation or breath holding. From a study of healthy rural older supine Italians, Sorbini and associates (47) predict a Pa_{O_2} of 86 mm Hg in a 54-yr-old man at sea level. With a Pa_{CO_2} of 40 mm Hg and an assumed R of 0.8, the expected $P(A\text{-}a)o_2$ is 14 mm Hg. We found, as expected, a lower $P(A\text{-}a)o_2$ in the less obese group, 10 mm Hg, compared to 18 mm Hg in the most obese group at rest. In the latter group, this is presumably due to basilar microatelectasis and shunting. As the Pa_{O_2} is normally higher in the sitting position, the expected decrease in $P(A\text{-}a)o_2$ with sitting must have been counterbalanced by an increase in $P(A\text{-}a)o_2$ in the obese subjects, resulting in a mean resting level of 13 mm Hg in our entire population. The rise in Pa_{O_2} with exercise can be attributed to improved ventilation-perfusion matching and to increased P_{AO_2} (decreased Pa_{CO_2} and increasing R). The widening $P(A\text{-}a)o_2$ with exercise may be due to lower mixed venous O_2 content or to rapid pulmonary capillary transit. A Pa_{O_2} below 75 or a $P(A\text{-}a)o_2$ above 33 during moderate exercise should be considered as due to disease.

We find that VD/VT decreases and $P(a\text{-}ET)_{CO_2}$ becomes negative during exercise, again suggesting improved matching of perfusion to ventilation and decreasing wasted ventilation. Therefore, a positive $P(a\text{-}ET)_{CO_2}$ or a VD/VT above 0.30 during heavy exercise should be regarded as suspicious for pulmonary vascular disease. Abnormalities of these measures can be attributed to overventilation of poorly perfused air spaces. Although unusual, we have seen anxiety induced tachypnea with shallow VT persist during exercise and cause an elevated VD/VT without pulmonary disease.

In summary, we suggest that, in an older population of overweight men, the predicted values for cardiorespiratory function during exercise should be based on height rather than weight. If this is not done, the

expected values for maximal oxygen uptake and oxygen pulse will be unrealistically high. The anaerobic threshold was invariably above 40% of the predicted and actual maximal oxygen uptake in these older men; a lower value indicates significant cardiovascular impairment. The distribution and limits of lung gas exchange values in this population should be useful reference values with which to compare the responses seen in patients suspected of having interstitial or pulmonary vascular disease.

Acknowledgment

We gratefully acknowledge the medical assistance of Drs. John Andrews, Mark Buchfuhrer, Gregory Hirsch, Jeffrey Kupperman, Kenneth Landis, Michael Lieber, Gregory Mason, Aileen Nosal, Ami Oren, Thomas Pong, Carolyn S. Ray, Richard Van Meter, and Daniel Weiler-Ravell in evaluation and exercise testing; the roentgenographic interpretations of Dr. Daniel Torrance; the technical assistance of Terry Robinson, Greg Marjama, and Don Hellard in exercise testing and data collection; the administrative and nursing support of Grace Mizumoto, R.N.; and the secretarial work of Norma Harris and Barbara Young.

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