BEDSIDE PHYSIOLOGY

Is exercise tolerance limited by the heart or the lungs?

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Medical subject headings: ergometry; exercise test; exercise tolerance; heart function tests; lung; mitral valve; pulmonary gas exchange; respiratory function tests; spirometry

Clin Invest Med 2001;24(2):110-7.

Glossary of terms used in the text

Ca_{O_2} = arterial oxygen concentration
Cc_{O_2} = capillary oxygen concentration
Cv_{O_2} = venous oxygen concentration
D_{LCO} = diffusion capacity of carbon monoxide
Hb_{CO} = carboxyhemoglobin content of hemoglobin
Pa_{O_2} = partial pressure of arterial oxygen
P_{AO_2} = partial pressure of oxygen in the alveoli
Pa_{CO_2} = partial pressure of carbon dioxide, arterial
P_{ECO_2} = partial pressure of expired carbon dioxide
P_{ETCO_2} = partial end-tidal pressure of carbon dioxide
P_{IO_2} = partial pressure of inspired oxygen
$Pmus_{CO_2}$ = partial pressure of carbon dioxide in muscle
$\dot{Q}_S/\dot{Q}_T$ = cardiac output shunted through the lung relative to total cardiac output
$R\dot{Q}$ = the respiratory exchange ratio ($\dot{V}_{CO_2}/\dot{V}_{O_2}$)
Sa_{O_2} = arterial oxygen saturation
V_D/V_T = ratio of the volume of dead space to tidal volume
$V_I max$ = maximum inspiratory volume
$\dot{V}_{O_{2max}}$ = maximum oxygen consumption
$\dot{V}_{CO_2}$ = carbon dioxide consumption
$\dot{V}_E$ = expired volume

Normal values

Forced expiratory volume in 1 second (FEV_1) = 3.5 L
Vital capacity (VC) = 4.4 L
Ratio of FEV_1/VC = 80%
Maximum power output (MPO) = (1604 + [650 in males]) × height $(m)^{1.86}$ × $age^{-0.44}$
Maximum achievable ventilation (MAV) = (130 + [31 in males]) × height $(m)^{1.86}$ × $age^{-0.30}$
$\dot{V}_{O_{2max}}$ = (3.55 + ([1.55 in males]) × height $(m)^{1.85}$ × $age^{-0.48}$

Equation

The Bohr equation: $V_D/V_T = (Pa_{CO_2} - P_{ECO_2}) / Pa_{CO_2} \times 100$

Introduction

A 55-year-old man presented with progressive dyspnea, exercise intolerance and fatigue for 1 to 2 years. He had had rheumatic fever at 14 years of age and began smoking in his teens, accumulating a 40 pack-year exposure. He had had a chronic cough and sputum production for the past 10 years.

On clinical examination he was in moderate distress. He was 1.72 m tall, weighed 89 kg (139% of his ideal body weight) and had a body mass index of 30. He had an irregular heart rate ranging from 140 to 160 beats/min, his blood pressure was 145/85 mm Hg, peripheral perfusion was normal and all pulses were present. The jugular venous pressure was modestly elevated 1 to 2 cm above the clavicle in the semi-recumbent position. There was no peripheral edema. His apex beat was hyperdynamic but not displaced. There was no appreciable right ventricular lift. Auscultation was consistent with mitral stenosis and incompetence. Respiratory examination disclosed an expiratory wheeze with mild hyperinflation but no end-inspiratory crackles. Clinically, there was no appreciable consolidation, collapse or effusion. There were no other abnormal findings on clinical examination.

Chest radiography showed cardiomegaly but no other abnormality. On pulmonary function testing obstructive ventilatory impairment was found (FEV_1 of 1.7 L/s, VC of 2.2 L/s, and an FEV_1/VC ratio of 57%). After inhalation of a short-acting selective β2-agonist, the subject's FEV_1 improved to 2.2 L/s, the VC to 3.5 L/s and the FEV_1/VC ratio 63% (Table 1). Echocardiography confirmed mitral stenosis and incompetence (mitral valve area >2 cm^2), moderate regurgitation, a large left atrium, normal left ventricular size, good left ventricular function and an estimated pulmonary artery systolic pressure of 30 mm Hg.

Cardiac catheterization disclosed a left ventricular end-diastolic pressure of 12 mm Hg, a mildly dilated left ventricle, good systolic wall motion, a moderate mitral regurgitation and a large left atrium. His aortic valve and coronary arteries were normal.

Digoxin was used to control his heart rate. Somewhat later, sotalol was prescribed and digoxin discontinued. His prescribed medications were: sotalol, coumadin, furosemide, potassium chloride, triazolam and fluoxetine.

He was referred for incremental and steady state exercise tests with the following question: Is his exercise tolerance limited by the state of his heart or his lungs?

Is this patient disabled?

An incremental exercise test to a symptom-limited maximum capacity was performed on a cycle ergometer. The maximum power output (MPO) achieved was 800 kpm/min, which amounted to 76% of the predicted normal of 1060 kpm/min (Table 2). The $\dot{V}O_{2max}$ achieved by this patient was 1.47 $L \cdot min^{-1}$ (16.5 $mL \cdot kg \cdot min^{-1}$), which amounted to 72% of the

Table 1: Function measurements obtained in the 55-year-old subject at the time of admission

Measurement	Result (and % of predicted)
Muscle strength, kg	
Arm press	85 (115)
Arm flex, kg	58 (98)
Leg press, kg	53 (89)
MIP, cm H_2O	80 (83)
MEP, cm H_2O	90 (75)
Spirometry*	
FEV_1, L/s	2.1 (59)
VC, L/s	3.5 (77)
FEV_1/VC, %	60
Flow volume, L/s	
$\dot{V}E_{max}$	4.3 (56)
$\dot{V}E_{50}$	1.5 (26)
$\dot{V}E_{25}$	0.4 (16)
$\dot{V}I_{max}$	4.3 (81)
$\dot{V}I_{50}$	3.7 (78)
$\dot{V}I_{25}$	4.1 (93)
Gas transfer,	
DLCO, mL/mm Hg · min^{-1}	20.1 (61)
VA, L	5.9 (96)
KCO (DLCO / VA), mL/mm Hg · min^{-1} / L	3.4 (65)
HbCO, %	5.7
Arterial blood gas values	
PCO_2, mm Hg	39
PO_2, mm Hg	66
pH	7.42
SaO_2, %	93
HCO_3^-, mmol/L	24

MIP = maximum inspiratory pressure, MEP = maximum expiratory pressure, DLCO = diffusing capacity of lung for carbon monoxide, VA = communicating lung volume, Kco = DLCO/ VA, HbCO = carboxyhemoglobin content of hemoglobin.

predicted normal of 2.04 L. During cycle ergometry, the relationship between oxygen uptake and power output is very close: $\dot{V}O_2$ = 0.316 + 0.0017 kpm/min (r = 0.96). With treadmill exercise, power output increases with speed and grade but is dependent on the patient's weight and cannot be reliably measured. The common practice of estimating $\dot{V}O_{2max}$ from weight, speed and grade is highly inaccurate. In ideal circumstances the $\dot{V}O_2$ achieved after treadmill ergometry is referenced to the predicted normal values and is on average 10% higher than that achieved with cycle ergometry. For reference purposes disability may be categorized as mild (70%–80%), moderate (50%–70%) and totally disabled (<50%); this patient was mildly disabled, achieving 76% of the predicted normal.

What limited his exercise capacity?

Exercise is initiated by volitional effort and terminated in response to excessive effort, a neurocognitive experience influenced by highly integrated pathophysiological events. During incremental exercise testing, the subject continues to increase power until the discomfort associated with exercise becomes intolerable. The discomfort is predominantly associated with the increased demands of exercise on the leg muscles (leg effort ≈ exertional discomfort) and on the respiratory muscles (dyspnea ≈ breathing discomfort). These symptoms were rated by matching the intensity of the discomfort to simple descriptive phrases tagged to numbers from 0 to 10 (Borg scale[1]). The magnitude of each symptom is referenced to the normal expected intensity (Fig. 1). Throughout exercise, the intensity of both dyspnea and leg effort were disproportionately high. The most intense symptom was dyspnea ("limiting"). The patient experienced chest pain, which is not normally present.

What pathophysiological constraints contributed to these symptoms?

Dyspnea

Discomfort associated with breathing is not confined to excessive effort; breathlessness is also experienced with breath-holding. When breathing is insufficient, the $PaCO_2$ rises, pH drops and PaO_2 drops, and an unpleasant urge to breathe occurs. Throughout

Table 2: Results obtained from the incremental exercise test

		Exercise workload, kpm							
Measurement	Rest	100	200	300	400	500	600	700	800
Metabolic									
$\dot{V}O_2$, L·min^{-1}	0.25	0.41	0.58	0.68	0.84	0.93	1.14	1.38	1.47
$\dot{V}CO_2$, L/s	0.25	0.34	0.47	0.57	0.77	0.94	1.24	1.54	1.65
RER	1.03	0.83	0.82	0.84	0.92	1.01	1.08	1.11	1.13
Symptoms, Borg scale									
Chest*								2	4
Breath					3	5	6	7	9
Effort				2	3	4	6	7	8
Cardiac response									
Heart rate, beats/min	78	85	93	99	106	110	130	137	139
Systolic blood pressure, mm Hg	110		115		130		140		170
Diastolic blood pressure, mm Hg	60		60		60		60		60
Respiratory response									
$\dot{V}E$, L/s	12.8	15.1	18.3	20.9	27.5	33.9	43.9	54.7	59.9
V_T, L	0.58	0.64	0.8	0.89	1.05	1.27	1.55	1.75	1.82
Fb, breaths/min	22	23	23	23	26	27	28	31	33
$P_{ET}CO_2$, mm Hg	27.4	29.5	31.2	32.4	31.9	31.3	30.5	29.8	29.2
P_ECO_2, mm Hg	17.0	19.3	22.2	23.7	24.2	23.9	24.3	24.2	23.8
SaO_2, %	96	96	96	96	96	96	96	96	96

*Chest measurements made at 1, 5 and 10 min. after exercise were 2, 2 and 0 on the Borg scale respectively.
RER = respiratory exchange ratio, Fb = frequency of breathing.

exercise, the patient's P_{ETCO_2} did not increase and his Sa_{O_2} did not decline, therefore breathlessness was not the cause of discomfort associated with his breathing. The intensity of dyspnea is closely coupled to the intensity of the motor command required by the inspiratory muscles to drive breathing. The effort this patient required to drive his breathing was substantially increased. Effort can be indirectly quantified by measuring ventilation and ventilatory capacity following the logical sequence of motor command / maximal motor command = effort / maximal effort = force / maximal force = ventilation / maximal ventilation.

Maximum ventilatory capacity (MVC) can be measured using maximal volitional ventilation. This is not commonly measured because of difficulties with reproducibility. The ventilatory capacity can be reliably and reproducibly estimated from the maximal flow volume loop. The rationale is conceptually simple. The maximal tidal volume achieved during exercise was 1.82 L; the minimal time for inspiration was 0.42 seconds (V_T / $V_I$max = 1.82/4.3 L/s); the minimal time for expiration was 0.86 seconds (V_T / FEV_1 = 1.82 L / 2.1 L/s). The minimal time for a single breath was thus 1.28 seconds (0.86 + 0.42, allowing 47 breaths/min (60/1.28). His maximal ventilatory capacity (≈ maximal achievable ventilation [MAV]) was 85.5 L/min (47 × 1.82 L). He achieved 59.9 L/min or 70% of his ventilatory capacity. His ventilatory capacity of 85.5 L/min was 64% of his predicted normal of 133 L/min. The magnitude of the ventilatory reserve does not take into account fatigue. The ventilatory capacity measured at rest is not the ventilatory capacity at maximal exercise because muscle fatigues with the intense activity seen as maximal exercise is approached.

During exercise, his ventilatory response was normal at lower power outputs but increased toward maximal exercise (Table 2). He terminated exercise when dyspnea was "very very severe" (Borg 9). Most people stop exercise when the limiting symptom is rated severe to very severe (Borg 5–7) because maximal intensity is rarely tolerated.

Leg effort

The intensity of effort required to drive the leg muscles was disproportionately high throughout exercise.

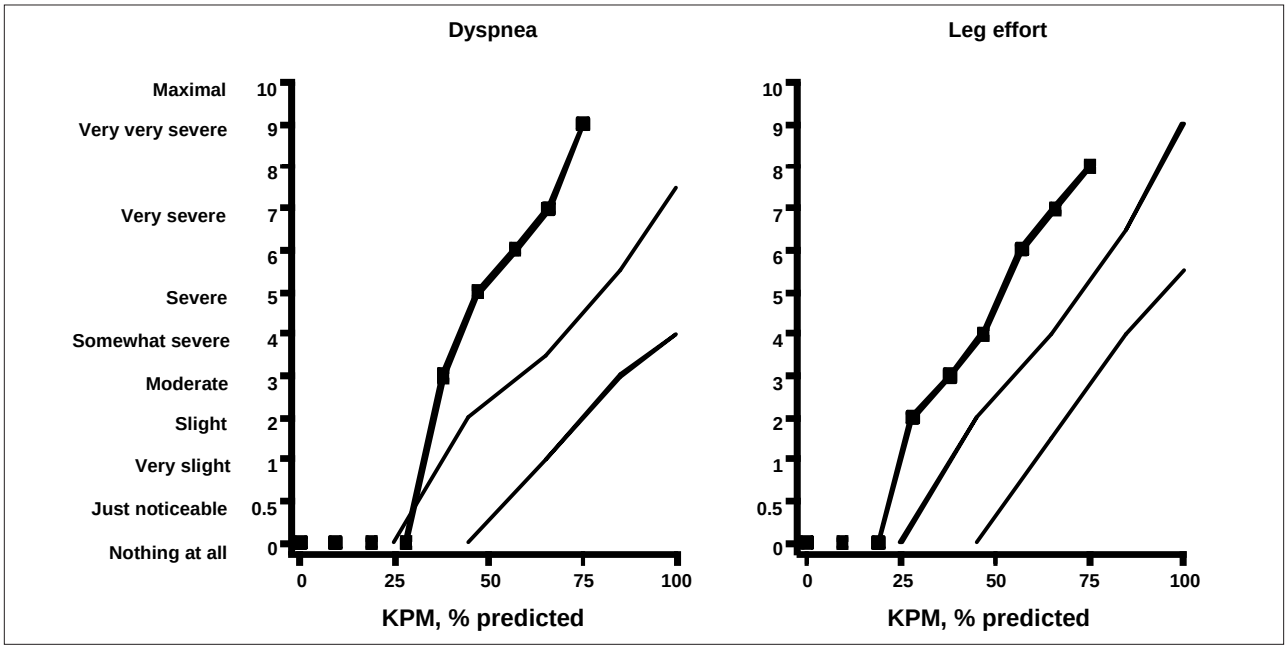

Fig. 1: The intensity of leg effort and dyspnea (closed squares), rated according to the Borg scale,[1] were plotted against the power output. The power output is expressed as a percentage of the normal predicted maximal power output in units of kg-m/min (KPM). The solid lines represent the 25th and 75th percentiles of the normal expected values.

This effort cannot be attributed to preexisting muscle weakness because he generated 53 kg of force or 89% of his predicted strength after a maximal volitional contraction of his quadriceps prior to exercise. If muscles fatigue, increasing effort is required to sustain power output. Poorly conditioned or poorly perfused muscles fatigue. Conditioning leads to a reduction in the amount of effort required to generate power (fatigue resistant) whereas deconditioning is associated with increased effort (fatigue prone). Reductions in muscle blood flow due to his mitral valve dysfunction were explored. Cardiac output was measured during steady state exercise and was normal (see below). Activity initiates biologic changes in the muscle, such as increased capillaries, increased mitochondria, higher concentrations of aerobic enzymes and increases in the overall rate of muscle protein synthesis. Reduction in activity leads to decreased capillaries, decreased mitochondria, lower concentrations of aerobic enzymes and a decrease in the overall rate of muscle protein synthesis.

During exercise, the cardiac response is often assessed on the basis of the heart rate, blood pressure and electrocardiographic findings. This be easily be misinterpreted. During exercise, lactate (La^-), K^+, PO_4^{3-}, carbon dioxide and a variety of other mediators leave the muscle and accumulate in the interstitium of the active muscles (peripheral, respiratory and cardiac) where they stimulate intramuscular free nerve endings. The concentrations of all these mediators increase with power output, increase further in poorly conditioned muscle and increase even further if they are not cleared from the interstitium by appropriate blood flow. This sensory input is relayed to motor neurons in the anterior horns of the spinal cord and reduces the responsiveness of α-motor neurons (central fatigue). More effort is required to sustain muscular activity because both the muscles and the α-motor units become less responsive (peripheral and central fatigue). The activation of these free nerve endings is also relayed through the spinothalamic tracts and projects to medullary neurons. Feedback from the active muscles combined with forward projection from the motor cortex determines the sympathetic stimulation seen during exercise. Thus, a higher heart rate response is seen when muscles are weak, poorly conditioned or poorly perfused. All these conditions may coexist. When primary muscle weakness or muscle weakness secondary to fatigue is not recognized, a cardiovascular limitation to exercise is often assumed based on a high heart rate response. At rest and during exercise our subject remained in atrial fibrillation with an appropriate increase in heart rate response. He had been prescribed sotalol, which would attenuate the effects of high sympathetic stimulation; he had a modestly lower blood pressure response than expected.

Chest pain

The chest pain experienced increased with power output and receded after exercise was stopped and was thereby compatible with myocardial ischemia. However, there was no evidence of ST segment depression on the exercise electrocardiogram; the coronary angiographic findings were within normal limits, and the cause of his chest pain remained undetermined.

Steady state exercise

The cardiac and respiratory responses during exercise were analyzed in more detail during steady state exercise. Steady state measurements were made at rest, and at 250 and 500 kpm/min. His hemoglobin was normal at 139 g/L but his HbCO was 5.7%, indicating a modest reduction in oxygen-carrying capacity.

Respiratory responses

The mixed expired P_{ECO_2} values were 20.2, 26.0 and 26.5 mm Hg at rest and at the 2 workloads, indicating that the total ventilation was high relative to the metabolic load ($P_{ECO_2} = \dot{V}_{CO_2}/\dot{V}_E$) (Table 3). The P_{ECO_2} values were 32.8, 32.7 and 31.1 mm Hg, respectively, indicating modest alveolar hyperventilation. The dead space to tidal volume ratios (V_D/V_T), calculated according to Bohr's equation were 35.6%, 19.8% and 14.4% respectively. The Pa_{O_2} values were 103.9, 103.0 and 103.9 mm Hg. respectively, indicating normoxemia. The estimated Pa_{O_2} values were 113.4, 111.9 and 117.9 mm Hg, respectively, according to the alveolar air equation

($Pa_{O_2} = [P_{IO_2} - Pa_{CO_2}] / R\dot{Q}$). The alveolar–arterial oxygen gradients were 9.5, 8.9 and 14.0 mm Hg (normal 5–12 mm Hg). The shunt fractions were estimated using the equation ($\dot{Q}_S/\dot{Q}_T = Cc_{O_2} - Ca_{O_2} / Cc_{O_2} - Cv_{O_2}$): these were 5.0, 2.0, and 3.7% (normal <5%). Using the classic 3-compartment analysis (ideal, dead space and shunt) $\dot{V}/\dot{Q}$ inhomogeneity was not a substantial problem.

Table 3: Cardiac and respiratory responses of the subject* at rest and at 2 workloads during steady state exercise†

		Exercise workload, kpm	
Measurement	Rest	250	500
Ventilation			
$\dot{V}$, L/min	14.1	25.9	47.0
Fb, breaths/min	26.0	23.0	28.0
V_T, L	0.542	1.126	1.679
V_D, mL	193.0	222.5	242.4
V_D/V_T, %	35.6	19.8	14.4
V_A, l/min	9.1	20.8	40.2
Carbon dioxide			
CO_2 output, mL/min	304	731	1331
O_2 intake, mL/min	327	833	1290
RER	0.930	0.878	1.032
P_{ECO_2}, mm Hg	20.2	26.0	26.5
P_{ETCO_2}, mm Hg	28.9	31.4	29.9
Pa_{CO_2}, measured, mm Hg	32.8	32.7	31.1
Pa_{CO_2}, estimated, mm Hg	30.4	31.4	28.9
P_{VCO_2}, mm Hg	45.0	49.2	54.9
$P_{V-PE\,CO_2}$, mm Hg	24.8	23.2	28.4
Venous–arterial CO_2 content difference, mL/L	5.85	7.66	10.66
Oxygenation			
PA_{O_2}, mm Hg	113.4	111.9	117.9
Pa_{O_2}, mm Hg	103.9	103.0	103.9
Alveolar–arterial O_2 gradient, mm Hg	9.5	8.9	14.0
Sa_{O_2}, %	98.2	99.0	98.0
Pc_{O_2}, mm Hg	121.3	119.7	126.4
Sc_{O_2}, %	98.3	98.3	98.5
Cc_{O_2}, mL/L	18.6	18.6	18.7
Cv_{O_2}, mL/L	12.0	9.7	7.9
Ca_{O_2}, mL/L	18.3	18.5	18.3
Cardiac response			
Heart rate, beats/min	87	104	129
Cardiac output, L/min	5.2	9.5	12.5
Stroke volume, mL	59.7	91.7	96.8
$\dot{Q}_S$, %	5.0	2.0	3.7
pH	7.46	7.46	7.46
Plasma HCO_3^-, mmol/L	22.4	22.7	21.4

*Hemoglobin level 139 g/L
†Barometric pressure 755 mm Hg
D_S = dead space, Sc_{O_2} = capillary oxygen saturation.

The capacity of the lungs to exchange oxygen was estimated based on the D_{LCO}. The molecular weight of oxygen is 32, similar to that of carbon monoxide (28); the solubility of oxygen is 0.023 mL gas/760 mm Hg/mL blood, whereas the solubility of carbon monoxide is 0.018 mL gas/760 mm Hg/mL blood. The diffusion properties of both gases are similar ($D_{O_2} \approx 1.2 \times D_{CO}$). We measured the transfer of carbon monoxide across the lung at 20.1 mL/mm Hg·min^{-1} (predicted normal 33.5 mL/mm Hg·min^{-1}) and from this can estimate the oxygen transfer capacity across the lung simply by multiplying the D_{LCO} by 1.2. The maximum pressure gradient across the alveolar capillary membrane can also be estimated using the alveolar gas equation and knowing that the mixed venous P_{O_2} drops systematically to a value approximating a P_{O_2} of 20 mm Hg at maximal exercise. The maximal gradient has to be averaged and amounts to 60 to 80 mm Hg. The diffusion properties of the lung are thus $D_{LCO} \times 1.2 \times 60$ mL/min (1447 mLO_2/min). During exercise, the D_{LCO} approximately doubles as more alveolar capillaries are recruited; this action is absent or attenuated in the presence of emphysema. This man was not limited by the gas transfer capacity of his lungs because his capacity was not exceeded and no arterial oxygen desaturation was found.

The oxygen uptake and carbon dioxide output at the mouth were normal for the work performed. The carbon dioxide output exceeded the oxygen uptake at 500 kpm/min which is less than 50% of his predicted normal exercise capacity. The respiratory exchange ratio ($\dot{V}_{CO_2}/\dot{V}_{O_2}$) was higher than expected. This is commonly attributed to anaerobic metabolism alone. However, there are several factors that contributed to the respiratory exchange ratio and the disproportionate increase in ventilation during exercise. The carbon dioxide appearing at the mouth is influenced by the substrate oxidized during exercise. As exercise becomes more intense there is greater dependence on the oxidation of carbohydrate because the rates at which fat can be oxidized are inadequate to meet the demands of intense exercise. The ratio of oxygen consumption to carbon dioxide production is 0.7 for fatty acid metabolism, 0.84 for

protein and 1.0 for carbohydrate metabolism. The capacity to oxidize free fatty acids is lower in poorly conditioned muscle. The carbon dioxide appearing at the mouth must transiently increase when the Pa_{CO_2} drops because carbon dioxide is washed out of body stores and appears in the expired gas until a new steady state is established. An increase in alveolar ventilation helps in the removal of carbon dioxide from the exercising muscle by widening the $Pmus_{CO_2} - Pa_{CO_2}$ such that more carbon dioxide is loaded into the blood. Alveolar hyperventilation minimizes the drop in intramuscular pH, thereby optimizing the responsiveness of the muscle to motor stimulation, reducing the intensity of leg effort. Hyperventilation is commonly seen in the presence of decreased cardiac output. This may be a behavioural adaptation in an attempt to preserve carbon dioxide output in the face of reduced blood flow. Hyperventilation may become prominent toward maximal exercise. Each of these factors play a role in the disproportionate increase in ventilation toward maximal exercise.

Cardiac response

The cardiac output was 5.2 L/min at rest ($\dot{V}_{O_2}$ = 327 mL/min), 9.5 L/min at 250 kpm/min ($\dot{V}_{O_2}$ = 833 mL/min) and 12.5 L/min at 500 kpm/min ($\dot{V}_{O_2}$ = 1290 mL/min). The cardiac output was normal relative to the metabolic demand (cardiac output = 5 + 5 × $\dot{V}_{O_2}$ [L/min]). He was not limited by his cardiac output despite the mitral valve disease and the mechanical constraints imposed by the increased work required by the left ventricle.

During incremental exercise, it should be noted that the oxygen uptake and carbon dioxide were lower than during steady state. There is a time lag in the response of both the heart and lungs to the metabolic demands as exercise increases. A slow response does contribute to actual reduction in blood flow at the time of force development contributing to the excessive effort. There was no evidence of excess lactate appearing in the circulation (HCO_3^- 22.4, 22.7 and 21.4 mmol/L) during steady state exercise where the appearance of lactate is accompanied by a decline

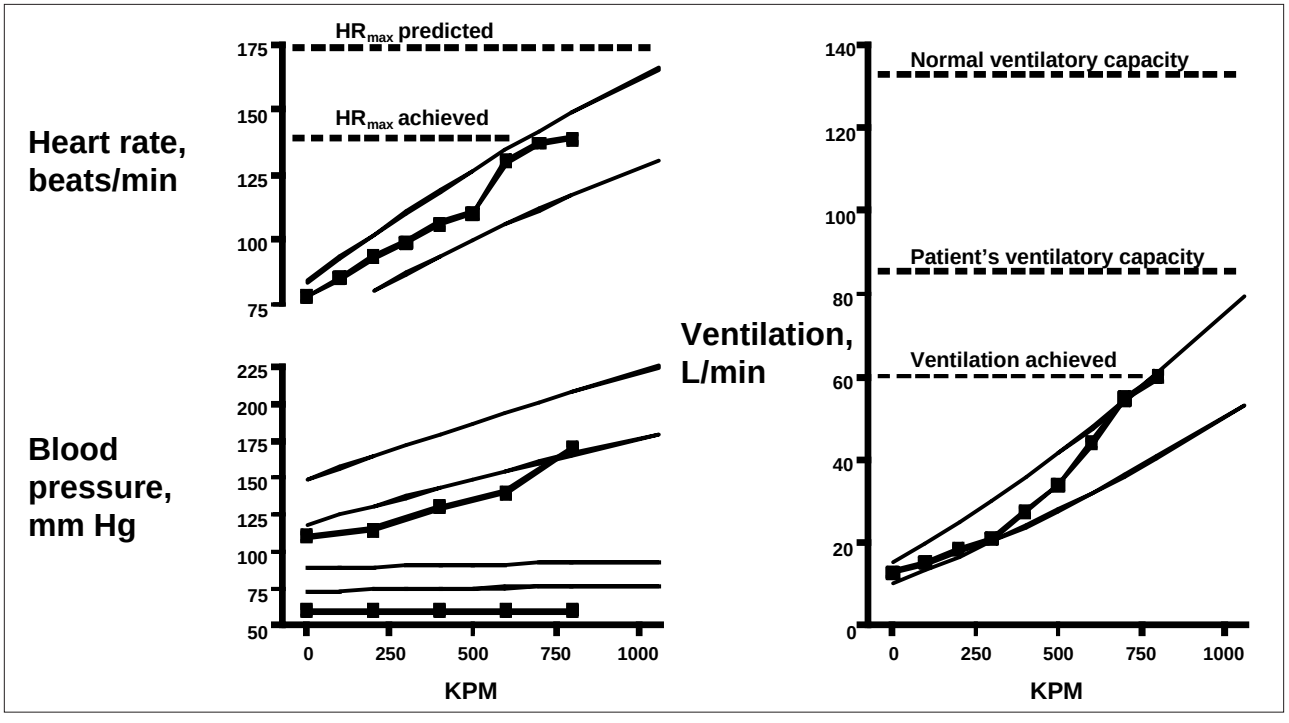

Fig. 2: The heart rate (HR), systolic and diastolic pressure, and ventilation (closed squares) were plotted against the power output (KPM). The solid lines represent ± 1 standard deviation around the normal expected values.

in HCO_3^-. It should be known that lactate leaves muscle slowly and the intracellular lactate level is substantially higher than that seen in the venous effluent from the active muscle. The lactate in muscle is physiologically more important than the lactate in the venous effluent from the muscle. Accumulation of intramuscular lactate reduces the responsiveness of the muscle to motor stimulation leading to peripheral fatigue. The lactate leaving fast twitch fibres moves into slow twitch fibres with greater aerobic potential.

During incremental exercise, the heart rate response was normal and the blood pressure response was reduced (Fig. 2). There is no question that the mitral valve disease has contributed to impair the mechanical function of the heart but it is not tenable to infer that the mechanical impairment contributed to reduced muscular blood flow during exercise. The relationship between maximal exercise performance and the severity of mechanical impairment in the heart is highly variable.

Summary

This 55-year-old man with known rheumatic mitral valve disease is modestly disabled achieving a $\dot{V}O_{2max}$ of 72% and a maximal power output of 76% of the predicted normal. His capacity to exercise is limited by dyspnea due to a reduction in his capacity to breathe. Ipratropium bromide was initiated to maximize his expiratory flow and improve his ventilatory capacity. A trial of inhaled steroids produced no improvement. He was referred for rehabilitation and smoking cessation. A decision was made to continue surveillance, postponing mitral valve replacement.

Reference

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