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ORIGINAL RESEARCH ARTICLE

Changes in oxygen uptake kinetics after exercise caused by differences in loading pattern and exercise intensity

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Abstract

Aims The kinetics of recovery-period oxygen uptake (VO_2) are affected by the O_2 deficit generated during exercise. However, studies using ramp tests (RTs) and constant work rate tests (CT) have differently characterized VO_2 responses to increased exercise intensity differently. We used these two types of loading patterns to investigate the effects of low-intensity, medium-intensity, and high-intensity exercises on the half time ($T_{1/2}$) of recovery-period VO_2 and the mechanism.

Methods and results Ten healthy men aged 21.2 ± 0.9 years underwent symptom-limited cardiopulmonary exercise tests with the ramp protocol to determine their anaerobic threshold. All subjects subsequently underwent three submaximal RT and CT at low, moderate, and high intensities. In all RTs, subjects began exercise by warming up (20 W). In CT, $T_{1/2}$ was significantly lengthened as exercise intensity increased (CT-low: 34.0 ± 3.9 s, CT-moderate: 39.5 ± 3.5 s, CT-high:44.6 ± 4.2 s; $P < 0.01$, ANOVA), whereas no significant change was observed in RT, which began with the same work rate (RT-low: 46.0 ± 5.7 s, RT-moderate: 45.7 ± 4.8 s, RT-high: 44.6 ± 3.5 s, RT-max: 44.8 ± 3.2 s; $P = 0.868$, ANOVA). Only high-intensity exercise resulted in two components (the fast and slow components) of VO_2 decay, reflecting the increased O_2 deficit by anaerobic metabolism.

Conclusions The exercise intensity at the beginning of an exercise affects early recovery-period VO_2, which is a fast component. The $T_{1/2}$ of recovery-period VO_2 occurs during the fast component, and an increase in O_2 deficit affects both the fast and slow components, lengthening the $T_{1/2}$. The $T_{1/2}$ of recovery-period VO_2 in CT at moderate or high intensities, even if not symptom limited, can be used to evaluate exercise intolerance and early occurrence of anaerobic metabolism. Submaximal exercise tests may be considered as convenient methods for evaluating exercise tolerance in patients with cardiac failure.

Keywords Oxygen uptake; O_2 deficit; Recovery-period; Half time; Exercise intensity

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Introduction

In recent years, it has been acknowledged that the kinetics of post-exercise recovery-period oxygen uptake (VO_2) can be used to evaluate the severity of cardiac failure.[1] The kinetics of recovery-period VO_2 consist of two components: the fast component, which attenuates rapidly after the cessation of exercise, and the slow component, which attenuates gradually thereafter.[2] The time constant determined by exponentially regressing VO_2 is one index that can be used to evaluate recovery-period VO_2 kinetics.[3,4] The half time ($T_{1/2}$) of VO_2 has also garnered attention as an index of recovery-period VO_2 kinetics,[1] and the meaning of these two indices is deemed to be essentially equivalent. It is thought that the kinetics of recovery-period VO_2 reflect the O_2 deficit generated during exercise,[5–7] but the changes observed in these indices as the exercise intensity increases differ; reports using constant work rate tests (CT) do not match with those using ramp tests (RTs). Shimizu *et al.*[8] reported that in CT, the time constant lengthens as the exercise intensity increases. However, Cohen-Solal *et al.*[1] report that in RT, the exercise intensity at the end of the exercise period does not significantly affect $T_{1/2}$. There is no existing research comparing how increases in work rate via these two loading patterns (i.e. CT vs. RT) affect recovery-period VO_2 kinetics.

Thus, in this study, we have used two types of loading patterns—CT and RT—to investigate the effect of low-intensity, medium-intensity, and high-intensity exercises, as indicated by intensity at the end of exercise, on the $T_{1/2}$ of recovery-period VO_2 and to determine the mechanism by which these effects are brought about.

Methods

Subjects

The research subjects consisted of healthy men with no history of smoking or cardiovascular disease. Twelve-lead electrocardiograms revealed no cardiac abnormalities in these subjects (*Table 1*).

Cardiopulmonary exercise test

Expired gas was analysed using a Cpex-1 (Inter Reha Co. Ltd., Tokyo, Japan), and VO_2 (mL/min) were measured on a breath by breath basis.

All exercise tests were performed using an electromagnetically braked cycle ergometer (IP-ES50P, Ergoline Co. Ltd., Bitz, Germany). During the test, a 12-lead electrocardiogram was monitored (1200 W, NORAV Medical Co. Ltd., Yoqneam, Israel), and blood pressure was measured every minute using an automatic sphygmomanometer (Tango M2, Suntech Co. Ltd., NC, USA). The expired gas data were converted from breath by breath values to 3 s values and expressed using an 8-point moving average.

The $T_{1/2}$ of recovery-period VO_2 was defined as the time taken (s) to reach 50% of the difference between exercise-final VO_2 and rest VO_2.[9]

Symptom-limited maximal ramp test

First, all subjects underwent a symptom-limited maximal RT (RT-max) (*Figure 1A*). After 6 min of rest on the ergometer, subjects started 20 Watt warming-up for 4 min followed by 30 W/min ramping until their exhaustion. The pedalling speed was set to 60 rpm. After the exercise, the subjects sat on the ergometer without pedalling for 10 min. The anaerobic threshold (AT; VO_2, mL/min) was determined by following criteria[1]: an increase in respiratory exchange ratio as exercise intensity increased,[2] nonlinear increase in VCO_2 vs. VO_2,[3] an increase in VE/VO_2 without a corresponding increase in VE/VCO_2,[4] and an increase in end-tidal O_2 fraction ($FETO_2$) without a corresponding increase in end-tidal CO_2 fraction ($FETCO_2$).[10]

Submaximal ramp test

The protocol for the submaximal RT was carried out similarly to the RT-max, but exercise was terminated when the exercise intensity (VO_2) reached 75% (low intensity of RT: RT-low), 100% (moderate intensity, RT-moderate), and 125% (high intensity, RT-high) of the VO_2 at each subject's predetermined AT (*Figure 1B–1D*).

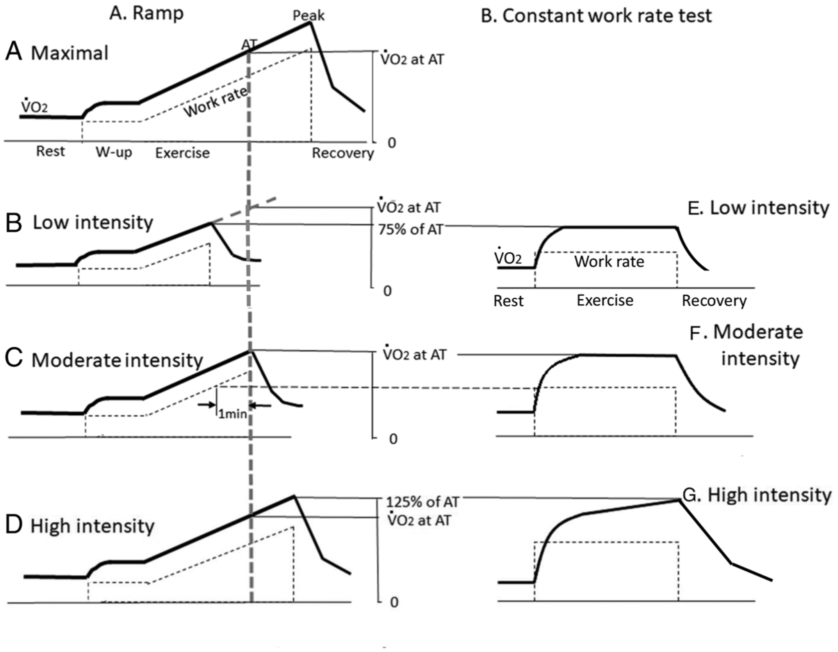
Table 1 Gas analysis data, heart rate, and work rate at rest and at end of each exercise

	Ramp test				
	Rest	Low intensity	Moderate intensity	High intensity	Maximal intensity
VO_2 (mL/min)	293.1 ± 36.4	1062.2 ± 167.5	1373.1 ± 231.7	1766.4 ± 333.0	2383.1 ± 465.3
VCO_2 (mL/min)	254.2 ± 31.2	891.9 ± 135.0	1243.2 ± 178.4	1788.4 ± 302.9	2860.3 ± 501.5
RER	0.87 ± 0.01	0.84 ± 0.06	0.9 ± 0.04	1.02 ± 0.08	1.21 ± 0.08
VE (L/min)	8.5 ± 1.2	21.9 ± 3.0	28.4 ± 4.7	38.0 ± 5.9	64.0 ± 10.5
HR (b.p.m.)	74.8 ± 8.3	105.0 ± 10.5	119.8 ± 11.5	132.7 ± 13.5	164.6 ± 13.1
Work rate (W)	—	68.7 ± 25.4	99.5 ± 22.9	136.5 ± 20.1	199.4 ± 27.6

	Submaximal constant work test			
	Rest	Low intensity	Moderate intensity	High intensity
VO_2 (mL/min)	303.9 ± 47.4	980.2 ± 165.1	1330 ± 188.4	1746.7 ± 302.4
VCO_2 (mL/min)	258.4 ± 41.9	913.6 ± 149.8	1316.4 ± 189.0	1823.9 ± 289.8
RER	0.85 ± 0.02	0.96 ± 0.05	0.99 ± 0.10	1.05 ± 0.10
VE (L/min)	8.5 ± 1.7	23.4 ± 3.2	31.2 ± 4.2	42.6 ± 6.4
HR (b.p.m)	76.4 ± 8.8	103.7 ± 11.9	121.8 ± 12.9	140.1 ± 17.9
Work rate (W)	—	47.7 ± 13.3	73.9 ± 14.0	106.3 ± 20.0

Rest values are the average of all the tests at rest with different protocols for each loading pattern. HR, heart rate; RER, respiratory exchange ratio; VCO_2, carbon dioxide output; VE, minute ventilation; VO_2, oxygen uptake.

Figure 1 Determining work rates for submaximal ramp tests and submaximal constant work rate tests. VO_2, oxygen uptake; AT, anaerobic threshold; W-up, warming up.



Submaximal constant work rate test

After resting for 6 min, the subjects exercised at one of the three constant work rates for 6 min (low intensity, CT-low; moderate intensity, CT-moderate; high intensity, CT-high). After each bout, they were observed at rest for 10 min.

The work rates used in each CT were 30 W less than the final work rates of each corresponding RT. In order to ensure that exercise-final VO_2 was similar in the submaximal RT and CT, we took into account that the time lag between the increase in work rate and increase in VO_2 (e–g, *Figure 1*). Subjects performed each test randomly with an adequate interval of time in between each test.

Inflection point of two exponential regression curves

Because the inflection points were clear in all cases, we visually determined the inflection point, which divided the fast and slow components on the graphs of recovery-period VO_2 kinetics and measured the time (s) from the end of the exercise to the inflection point (*Figure 2*).

Measurement of O_2 deficit at the beginning of exercise and O_2 debt after exercise

We measured the O_2 deficit at the beginning of exercise and the area under the curve (AUC) during the recovery-period VO_2 (O_2 debt) (*Figure 3*).

As shown in *Figure 3A*, the O_2 deficit at the beginning of the RT (a) was calculated by subtracting the AUC of the VO_2 curve over the 4 min warming-up period (b) from the area of the rectangle whose height was the difference between rest VO_2 and VO_2 at the end of the 4 min warming-up period and whose width was 4 min (a + b). We actually measured only A and D because the O_2 deficit, which was speculated to be generate during the incremental loading of RT, was not measurable (c).

The O_2 deficit during the submaximal CT (e), as shown in *Figure 3B*, was calculated by subtracting the AUC of the VO_2 curve over the 6 min testing period (f) from the area of the rectangle whose height was the difference between rest VO_2 and VO_2 at the end of exercise and whose width was 6 min (e + f).

Post-exercise O_2 debts (d and g in *Figure 3A and 3B*, respectively) were calculated by taking the exercise-final VO_2 as the peak of the curve and integrating VO_2 from there until it decayed to the rest value. VO_2 at measurement was calculated without the use of moving averages. Finally, we calculated the ratio of O_2 debt to O_2 deficit (O_2 debt/O_2 deficit) for each exercise intensity. We calculated the percentage of the O_2 deficit [e/(e + f)] and that of the O_2 debt [g/(e + f)] in O_2 consumption during exercise in CT.

Statistical analyses

Data were expressed as mean ± SD. Statistical analysis used paired *t*-test and ANOVA where applicable. A *P*-value less

Figure 2 Inflection point of VO_2 decay in recovery phase after exercise. Dotted line a: exponential regression curve of fast component. Dotted line b: exponential regression curve of slow component.

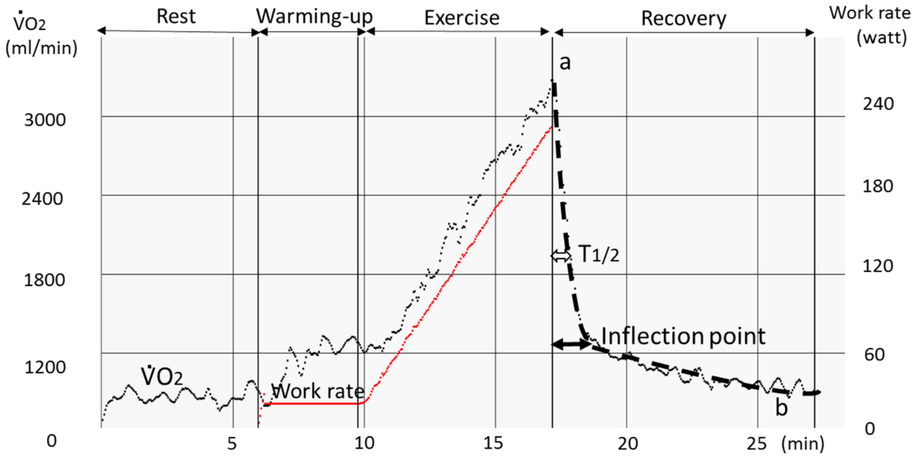
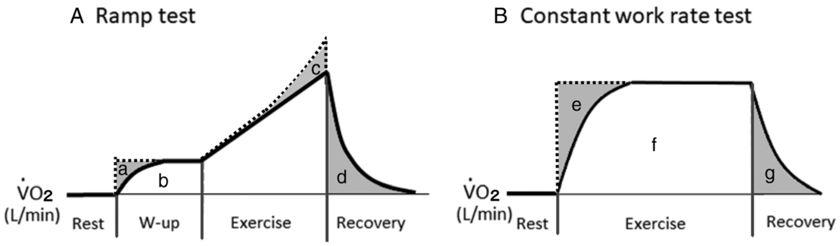


Figure 3 O_2 deficit at the beginning of exercise and O_2 debt after exercise. VO_2, oxygen uptake; W-up, warming up. (a) and (e): O_2 deficit at the beginning of exercise. (b): total warming up VO_2. (f): total exercise VO_2. (d) and (g): O_2 debt after exercise. (c): O_2 deficit presumed to occur during incremental load in RT. (c) was not actually measured as it is not measurable. Dotted line: theoretically speculated ATP requirement for the exercise.



than 0.05 was considered statistically significant. All analyses were carried out using the JMP computer software (Ver. 11.2.0, SAS Institute Inc., NC, USA).

Ethical considerations

This study was approved by the Tokyo University of Technology Ethics Committee (no. E17HS-002) and conformed to the Declaration of Helsinki. Consent was obtained from the subjects after they were thoroughly informed of what study participation entailed.

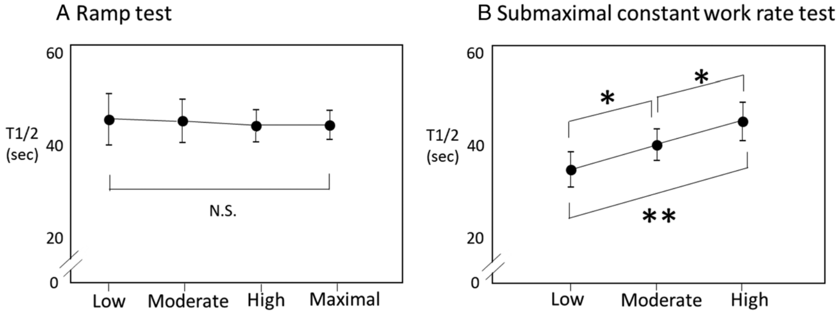
Results

Thirteen subjects underwent the RT-max. Of these subjects, three were excluded because of a vagal reflex after exercise. As such, the data from 10 subjects were included in the study and analysed (age: 21.2 ± 0.9 years, height: 170.6 ± 5.9 cm, weight: 58.6 ± 7.0 kg, AT: 1311 ± 234 mL/min, work rate at AT: 104.9 ± 17.2 W, peak VO_2: 2383 ± 465 mL/min, work rate at peak: 199.4 ± 27.6 W). Each subject performed a total of seven exercise tests: the RT-max, three submaximal RT, and three submaximal CT (*Figure 1*). There were no significant differences in exercise-final VO_2 between the submaximal RT and CT at any of the three intensities (VO_2: mL/min, RT-low: 1062 ± 167 vs. CT-low: 980 ± 165; RT-moderate: 1373 ± 231 vs. CT-moderate: 1330 ± 188; RT-high: 1766 ± 323 vs. CT-high: 1746 ± 302).

$T_{1/2}$ of recovery-period VO_2

We found no significant differences in the $T_{1/2}$ of recovery-period VO_2 between the four RT intensities (RT-low: 46.0 ± 5.7 s, RT-moderate: 45.7 ± 4.8 s, RT-high: 44.6 ± 3.5 s, and RT-max: 44.8 ± 3.2 s; $P = 0.868$) (*Figure 4*). The $T_{1/2}$ for RT-high and RT-max appeared lower than the corresponding values for the RT-low and RT-moderate; however, these differences were not statistically significant. On the other hand, in the submaximal CT, the $T_{1/2}$ of recovery-

Figure 4 Half time of VO_2 in different exercise intensity of ramp tests and constant work rate tests. Half time: $T_{1/2}$. (*A*) In the ramp test, there was no significant difference in $T_{1/2}$ between the exercise intensities. (*B*) In the constant work rate test, as the work rate increased, the $T_{1/2}$ was extended. $^{*}P < 0.05$ and $^{**}P < 0.01$. N.S., not significant.



period VO_2 significantly lengthened as work rate increased (CT-low: 34.0 ± 3.9 s, CT-moderate: 39.5 ± 3.5 s, CT-high:44.6 ± 4.2 s; $P < 0.01$).

$T_{1/2}$ and the inflection point

In each test, as the $T_{1/2}$ occurred before the inflection point, we considered it to be located in the fast component (time to fast component, s: RT-high: 84.0 ± 3.0, RT-max: 100.6 ± 11.9, CT-high: 82.5 ± 10.2). In the low and moderate intensities of the RT and the submaximal CT, no inflection point occurred; as such, we judged there to be no slow component in these cases.

O_2 deficit and O_2 debt

In the RT, there was no significant difference in O_2 deficit as the final-exercise work rate increased, while O_2 debt increased along with final-exercise work rate. In the CT, both of O_2 deficit and O_2 debt increased along with work rate (*Table 2*).

Discussion

$T_{1/2}$ of recovery-period VO_2 in different loading patterns

First, in the RT, even when exercise-final work rates increased, no significant changes were noted in $T_{1/2}$ of recovery-period VO_2. This observation largely corresponds with finding reported by Cohen-Solal *et al.*[1] who were using exercise intensities above the AT.

Next, in the submaximal CT, the $T_{1/2}$ of recovery-period VO_2 significantly lengthened as the work rate increased. We believe that this result corresponds with that of Shimizu *et al.*[8] who reported the increase in the recovery-period time constant.

Table 2 O_2 deficit during exercise and O_2 debt after exercise

	Ramp test				
	Low	Moderate	High	Maximal	*P*-value
O_2 deficit (L)	0.67 ± 0.07	0.66 ± 0.05	0.65 ± 0.09	0.69 ± 0.10	0.743
O_2 debt (L)	1.72 ± 0.03	2.49 ± 0.41	3.37 ± 0.49	7.06 ± 1.55	<0.001
O_2 debt/O_2 deficit	2.75 ± 0.47	3.78 ± 0.43	5.19 ± 0.92	10.18 ± 1.80	<0.001

	Constant work rate test			
	Low	Moderate	High	P-value
O_2 deficit (L)	1.38 ± 0.28	2.00 ± 0.41	3.64 ± 0.71	<0.001
O_2 debt (L)	1.33 ± 0.28	1.98 ± 0.39	3.35 ± 0.64	<0.001
O_2 debt/O_2 deficit	0.96 ± 0.03	0.99 ± 0.02	0.92 ± 0.05	0.002
O_2 deficit/total O_2 consumption (%)	10.9 ± 1.4	10.7 ± 1.4	14.0 ± 2.0	<0.001
O_2 debt/total O_2 consumption (%)	10.5 ± 1.2	10.6 ± 1.3	12.9 ± 2.1	0.006

P-values were calculated by ANOVA. Total O_2 consumption is the sum of the VO_2 and O_2 deficit during exercise in CT cases.

Consequently, as the work rate increased, we observed differences in the change in the $T_{1/2}$ of recovery-period VO_2 between the two different loading patterns (RT vs. CT).

Differences in work rate at the beginning of exercise

We speculated that differences in the change in the $T_{1/2}$ along with the work rate between the two loading patterns were caused by differences in the work rate at the beginning of exercise. In the RT, the subjects always began exercising at the same work rate. In the CT, the higher the initial exercise work rate, the longer the $T_{1/2}$ of recovery-period VO_2 became. Gore and Withers[11] reported that O_2 deficit was affected by both exercise intensity and duration, of which intensity was the major determinant of excess post-exercise oxygen consumption.

Fast and slow components of VO_2 after exercise

At the beginning of exercise, the adenosine triphosphate (ATP) stored inside the skeletal muscles, and the ATP regenerated by creatine phosphate (PCr) are used as energy for the exercise (alactic), after that aerobic metabolism ensues and ATP needed was satisfied. The O_2 deficit generated here is reflected in the fast component at post-exercise. However, if the exercise intensity at the beginning of exercise is above one's AT, the energy stored in the muscles as ATP and PCr is metabolized first, and ATP deficiencies that cannot be covered by aerobic metabolism are compensated by anaerobic metabolism (lactic). The sum of these three metabolic systems increases O_2 deficit and prolongs the decay of post-exercise VO_2 (i.e. slow component).[7]

In other words, at exercise intensities below AT, the O_2 deficit caused by energetic metabolism of ATP and PCr stored in the skeletal muscles is reflected only in the fast component post-exercise, whereas at exercise intensities above AT, the kinetics of recovery-period VO_2 is composed of two exponential functions: the fast component and the slow component. If one considers the fact that the time point at which after exercise VO_2 has decayed by half of the difference between it and rest VO_2 (the measurement point of $T_{1/2}$) occurs within the fast-component period, we believed that it was possible for exercise-initial O_2 deficit to affect recovery-period $T_{1/2}$.

Additionally, we surmised that if exercise intensity is below AT, an inflection point will not be observed (i.e. no slow component will exist). In the recovery-period VO_2 kinetics at exercise intensity above AT, after the O_2 deficit from the beginning of exercise to AT compensated, the slow component, which reflects the remaining O_2 deficit caused by anaerobic metabolism, becomes prominent. The border between this component and the fast component appears as an inflection point (*Figure 5C and 5D*).

$T_{1/2}$ of recovery-period VO_2 in the ramp test

The O_2 debt increased along with exercise intensity at the end of exercise (*Table 2*). However, there was no significant difference in the $T_{1/2}$ of recovery-period VO_2 regardless of the increase in exercise intensity. We speculated this to be so because as exercise-final VO_2 increased, and the VO_2 decay curve became steeper (and $T_{1/2}$ shortened). The $T_{1/2}$ for R-max was not smaller than that of R-high. We surmise that this is so because the primary difference between R-high and R-max was an increase in anaerobic metabolism, causing an increase in during-exercise O_2 deficit, which was then added after exercise to the fast component.

In the RT in which the exercise-final intensity was below AT, during-exercise O_2 deficit (alactic) was reflected in the fast component. However, at exercise intensities above AT, while further anaerobic metabolism causes an increase in O_2 deficit (lactic), in practice, the O_2 debt of anaerobic metabolism is added immediately post-exercise, thereby increasing the AUC of the fast component of VO_2. For this reason, we thought that the VO_2 decay steeping by increased in exercise-final VO_2 cancelled the prolongation of $T_{1/2}$ (*Figure 5D*).

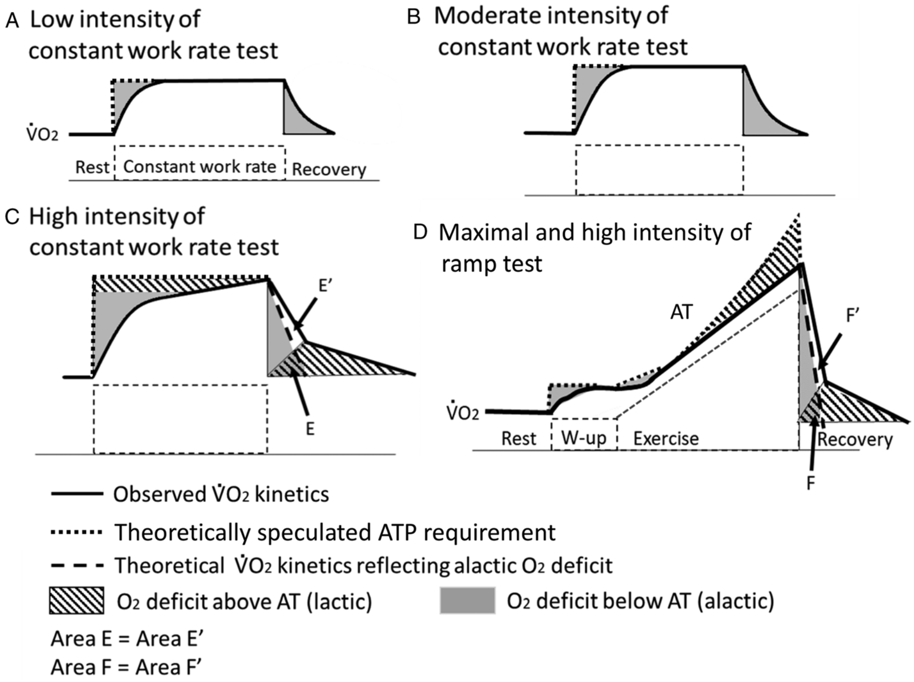
Cohen-Solal *et al.*[1] reported no significant differences in the $T_{1/2}$ of after exercise VO_2 in RT. We presumed that this was because the exercise endpoint in their RT were at an exercise intensity above AT, and according to the previously mentioned reasoning, the increase in O_2 deficit caused by during-exercise anaerobic metabolism was cancelled out by the shortening of $T_{1/2}$ caused by a higher peak VO_2.

Reports suggest that there is an unmeasurable O_2 deficit during exercise in RT.[12] In this study, we could not measure the O_2 deficit either directly or during exercise in RT. However, we conceive that the O_2 deficit during the incremental loading should represent the difference of O_2 deficit at the beginning of exercise (warming up, 20 W) and O_2 debt after exercise, that is, $d - a = c$ (*Figure 3*).

Although we speculated that O_2 debt is mainly increased by lactic acid, the elevated temperature and secreted catecholamine may increase the O_2 debt even in RT-low and moderate cases. Therefore, the O_2 debt always exceeds the O_2 deficit at the beginning of warming up in RTs, and the difference increases with the peak work rate. This phenomenon was also seen below AT.

There may be several reasons for this as follows[1]: when the subjects are young and healthy as in this study, they may not use up the stored PCr during warming up and may use it for producing ATP during incremental loading.[2] Small amounts of lactic acid may be produced during exercise although the exercise intensity is below AT.[3] There are effects

Figure 5 Schematic diagram of O_2 deficit and O_2 debt of ramp tests and constant work rate tests. (*A*) and (*B*) The during-exercise O_2 deficit corresponds to the post-exercise O_2 debt (grey area). (*C*) and (*D*) Adding the O_2 deficit (grey area) generated at an exercise intensity below AT (inside dashed line) to the additional O_2 deficit (area with slanted lines) generated at an exercise intensity above AT results in the creation of the fast component (solid line). After the O_2 deficit generated below AT (dashed line, area E, F) is compensated, the O_2 deficit generated above AT (area with slanted lines) remains, and an inflection point occurs (slow component). VO_2, oxygen uptake; AT, anaerobic threshold; W-up, warming up.



of increased body temperature, and catecholamine are observed during exercise.[7] Additionally, we thought that an increased lactic acid accumulation markedly enhances the O_2 debt above AT.

$T_{1/2}$ of recovery-period VO_2 in the submaximal constant work rate test

In the submaximal CT, the $T_{1/2}$ lengthened as work rate and O_2 debt increased (*Table 2*). We believe this to be because as work rate increases, the ATP necessary to perform work increases, as did the O_2 deficit, causing the lengthening of $T_{1/2}$.

Isaacs *et al*.[13] reported that the O_2 deficit from the beginning of exercise to the steady state phase affects to the fast component, whereas the O_2 deficit engendered above AT affects the slow component. Similarly, in this study, we observed that at sub-AT exercise intensities, VO_2 reaches a steady state phase, and during-exercise O_2 deficit and post-exercise O_2 debt become essentially equivalent (*Table 2*). For this reason, we thought that O_2 deficit (alactic) corresponds to the post-exercise fast component (*Figure 5A and 5B*). In the CT-high condition, we believe that as the anaerobic metabolism becomes a larger proportion, the slow component increases, leading to an extended $T_{1/2}$ (*Figure 5C*). As shown in *Table 2*, the ratio of the O_2 deficit and O_2 debt increased from CT-moderate to CT-high but not from CT-low to CT-moderate. The extension of $T_{1/2}$ from CT-low to CT-moderate simply represents the effect of an increase in O_2 deficit at the beginning of exercise. However, the increase in O_2 debt from CT-moderate to CT-high was believed to be due to the addition of the O_2 deficit caused by anaerobic metabolism; a slow component appeared, and the $T_{1/2}$ was further extended at CT-high.

Limitations

This study had a limited number of subjects. However, data variance was small, and as far as the physiological interpretation of the data is concerned, our results were meaningful. We measured only VO_2 for energy metabolism and did not measure body temperature, blood catecholamine concentration, or blood lactic acid concentration. Thus, our results regarding the realities of energy metabolism are primarily educated guesses.

Clinical implications

In cardiac failure patients, the $T_{1/2}$ of recovery-period VO_2 is lengthened,[1] and recovery-period VO_2 kinetics are useful in determining the severity of cardiac failure.[14,15] In other words, anaerobic metabolism occurs earlier in those patients,[16,17] causing an enlargement of the slow component, prolonging recovery-period VO_2 kinetics when exercise-final VO_2 is not higher than healthy individuals, and ultimately resulting in a lengthened $T_{1/2}$ of recovery-period VO_2 in comparison to healthy individuals. In RT, even if intensity does not reach maximal levels, if it is high enough, no effect is seen on the $T_{1/2}$ of recovery-period VO_2. Consequently, we can say that as long as it is conducted above AT, the $T_{1/2}$ of a RT can be useful in the evaluation of cardiac failure in comparison to healthy individuals even if it does not reach symptom limits.

The $T_{1/2}$ of recovery-period VO_2 in CT at moderate or higher intensities, even if they are not symptom limits, can be used to evaluate exercise intolerance and the early occurrence of anaerobic metabolism. As such, submaximal exercise tests may be considered a convenient method for evaluating exercise tolerance in cardiac failure patients.

Conclusions

The intensity at the beginning of exercise affected recovery-period VO_2. If the intensity at the end of exercise was below AT, recovery-period VO_2 kinetics was characterized only by the fast component, whereas if it was above AT, the addition of anaerobic metabolism gave rise to a slow component, and the border these curves were characterized by an inflection point. While the $T_{1/2}$ of recovery-period VO_2 occurred within the fast component, the enlargement of the slow component affected the fast component and lengthened $T_{1/2}$.

In RT, where the work rate at the end of exercise was always the same, a lengthening in the $T_{1/2}$ of recovery-period VO_2 was cancelled out by an increase in the intensity at the end of exercise. Thus, even though the exercise intensity increased, $T_{1/2}$ of recovery-period VO_2 did not change. On the other hand, in CT, the $T_{1/2}$ of recovery-period VO_2 lengthened as exercise intensity increased.

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Conflict of interest

None declared.

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Authors' contributions

Y. I., T. M., T. T., and H. I. contributed to the conception or design of the work. Y. I., T. M., and H. I. contributed to the acquisition, analysis, or interpretation of data for the work. All authors drafted the manuscript. All authors gave final approval and agree to be accountable for ensuring integrity and accuracy.

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