A Study on the Rehabilitation of Ischemic Heart Disease Patients

The Heart Rate, β -Receptor Blocking Agents and Strength-Duration Relationship of Exercise*

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SUMMARY

Exercise tests with bicycle ergometer were performed in 9 normals and 54 patients with ischemic heart disease (IHD), in order to evaluate the heart rate (HR) as a parameter for determining the physical working capacity and controlling the daily activities, and to observe the circulatory and metabolic effects of β -blockers and moreover to study the strength-duration relationship of exertion. The following results were obtained and discussed:

1. Reliable correlation between heart rate and O_2 consumption during all phases of exercise, including the recovery period ($R=0.60-0.87$, $p<0.001$) and parallelism of heart rate and pressure rate product allows the heart rate to be used as a parameter for myocardial O_2 demand and ST·T changes. It is reasonable to evaluate the exercise effect by the net increment of heart rate during and after exercise, and to utilize the increment of heart rate in the recovery period for the evaluation of physical fitness and adaptability.

2. Similar to other studies made on β -blockers, in this study too, the agents including Kō 1366** suppressed the exercise responses of heart rate ($p<0.01$), the product of heart rate and blood pressure ($p<0.01$), and O_2 consumption ($p<0.01$). But a relatively slow recovery of O_2 consumption from the peak response towards pre-exercise level and a higher O_2 pulse in the early recovery period was observed after β -blocker medication.

3. Capillary (ear lobe) pH decreased and recovered in correlation with the strength of exertion. This corresponded with the response of other factors such as ST·T, heart rate, pressure-rate product, and O_2 consumption, showing that the exercise strength was one of the more dominant factors to determine allowable work load for ischemic heart disease patients.

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** 0-2-Hydroxy-3-(tert-butylamino) propoxybenzonitrile HCl

4. Recovery of pH was delayed after medication with Kō 1366 ($p < 0.01$).

Additional Indexing Words:

Heart rate as a parameter O_2 consumption Pressure-rate product
Myocardial O_2 consumption Oxygen debt Heart rate debt
Recovery period Capillary pH β -blockers

THE observation, control and rehabilitation of the socially active (ambulatory) ischemic heart disease (IHD) patients has been difficult, since it is not so easy to catch the responses of coronary circulation in daily activities. Heart rate (HR), which is well known for its good correlation with whole body and myocardial O_2 consumption (O_2 cons.)^{1),10)} as well as ECG ST-T changes,²⁾ has been chosen in our department to be used as a parameter of cardiac work for observation and control of the daily activities of such patients. During recent years we have invented some ME apparatuses such as the HR alarm, HR memo, ECG memo, ergometer for multiple use³⁾ etc and developed the Check-Alarm-Training System (CATS)³⁾ to deal with cardiac patients. The application of this system depends on the HR to a large extent, based on various studies.^{4),5)} This report presents some further basic informations about the reliability of the HR as a parameter for cardiac performance, not only during exertion, but also during recovery time. Focusing on HR and its relationship with other circulatory and bodily factors, the strength-duration relationship of exercise and the role of medication with β -blockers are studied along with some physiological and metabolic alterations which accompany these specific conditions.

INSTRUMENTS

For exercise, the ergometer for multiple use (already published in this name)³⁾ was used. It is a Monark bicycle which we equipped with some electronic devices and a mini-computer system. Douglas bags were used to collect expired gas. Sampled gas was readily analyzed by Fukuda breath analyzer type R-1000S, the reliability of which was proved by its comparison with the Schollander method. A 4 channel ECG recorder was used to record 2 bipolar chest leads of ECG, the power and cardiogram. Blood pressure (B.P.) was measured manually by ordinary mercury type sphygmomanometer.

MATERIALS

Nine normal subjects with an age range of 40–62 years (average 45.9 years) and 54 IHD patients with an age range of 42–78 years (average 62.1 years) performed a total number of 155 exercise tests (normal 19 tests, IHD 136 tests). All

these IHD patients had ischemic ECG changes at rest and/or by exercise. Among the IHD patients 13 subjects had experienced myocardial infarction, 21 subjects had angina pectoris and out of 54 patients 21 subjects were hypertensive and 27 patients were under medication with β -blockers.

METHODS

Study I: General consideration of the relationship between the heart rate (HR), ECG, blood pressure (B.P.), and O_2 consumption (O_2 cons.):—

Nine normal subjects and 40 IHD patients, out of whom 13 were under β -blocker (propranolol, alprenolol, Kö 1366¹⁹) medication (β -IHD group), took part in this study. In a sitting position on the ergometer, the resting HR, B.P., ECG, and O_2 cons. for 3 min were measured and recorded. Keeping the revolution number constant (50 rpm), each subject had to cycle for 6 min with different loads of 0.5–2.0 kp (in some cases, for correlation study, loads of 2.5–3.0 kp were also tried). Every subject had to undergo 2 to 3 tests in order to get stable values. Between tests, the subject was given enough time (30–40 min) to return to pre-exercise condition. Collection and sampling of expired gas, measurement of HR, B.P., and recording of ECG were done as in Fig. 1. Two chest leads which reflected the most ischemic zone in the resting ECG, were chosen to be recorded. Fig. 2 shows our criteria for evaluation.

METHOD OF MEASUREMENT

Fig. 1. Method of measurement. O_2 cons.=oxygen consumption, ECG=electrocardiogram, B.P.=blood pressure, HR=heart rate.

	remarkable	slight
ST	$\pm$ over 0.1 mV	$\pm$ 0.05 ~ 0.09 mV
T		

Fig. 2. Criteria for ECG ST-T change evaluation.

Study II: Effect of K $\ddot{o}$ 1366:—

Eleven patients, who were previously under neither β -blocker nor digitalis medication, performed 6 min of ergometry with 1.0 kp resistance and then, after being medicated with K $\ddot{o}$ 1366 (15–30 mg/day) for 1 week, repeated the same test. In addition to the measurements as in Study I, capillary (ear lobe) pH was measured before, during and after exercise. A group of 10 other patients performed 21 min cycling with 0.5 kp resistance once, and repeated it for the second time after a week of K $\ddot{o}$ 1366 (15–30 mg/day) administration.

Study III: Study concerning the effect of strength and duration of exercise:—

Six patients who were not under β -blocker or digitalis therapy, were chosen to perform ergometry, first with 1.0 kp resistance for 6 min and then after a reasonable recovery (30–40 min) with 2.0 kp for 3 min. All the other procedures were carried out as in Study I and capillary blood was examined for pH as in Study II.

RESULTS

Study I

The relation between electrocardiographic ST·T changes and maximum percentage of HR increase (Max Δ HR %) during exercise is shown in Table I. The dependency of ECG ischemic change on the HR response and their parallelism during exercise was observed.

HR and pulse pressure $\times$ HR (P.R.) were then studied and plotted in each of the non β -IHD and β -IHD groups, under almost the same average total work load (144 ± 48 kpm for the non β -IHD and 145 ± 48 kpm for the β -IHD group). Comparing the response of the β -IHD group with that of the non β -IHD, both the P.R. (Fig. 3a) and HR (Fig. 3b) were significantly lower in the β -IHD group from resting to exercise and also to recovery period ($p < 0.01$).

HR and O $_2$ cons./body surface area: Correlation and parallelism of these 2 factors were studied under different amounts of work load, ranging from 70 kpm/min to 450 kpm/min for patients and 70–600 kpm/min for normal subjects (Table II). Each group showed a significant positive correlation

Table I. Relation between HR and ST·T Change

Max Δ HR (%)	No. of cases	ST·T Response (%)		
		No resp.	Slight	Remarkable
10–40	25	28	48	24
41–70	42	4.8	40.5	54.7
71–100	16	12.5	25	62.5
101–	6	0	16.7	83.3

$$\text{Max } \Delta\text{HR } \% = \frac{\text{Peak HR} - \text{Rest HR}}{\text{Rest HR}} \times 100$$

Fig. 3. The course which HR and PR took in β -IHD and non β -IHD in response to exercise under almost the same average work load (Kpm).

Table II

Items	Total	Increment	Debt
Groups			
Normal control N=19	$Y = 5.84X - 480$ $R = 0.79$ $P < 0.001$	$Y = 11.6X + 1,260$ $R = 0.87$ $P < 0.001$	$Y = 9.40X + 33.5$ $R = 0.81$ $P < 0.001$
Non β -IHD N=52	$Y = 3.40X + 1,170$ $R = 0.66$ $P < 0.001$	$Y = 7.25X + 1,970$ $R = 0.68$ $P < 0.001$	$Y = 9.80X + 32.7$ $R = 0.55$ $P < 0.001$
β -IHD N=22	$Y = 4.53X + 560$ $R = 0.63$ $P < 0.001$	$Y = 8.50X + 1,990$ $R = 0.83$ $P < 0.001$	$Y = 9.26X + 13.0$ $R = 0.86$ $P < 0.001$

Non β -IHD=cases which are not under medication of β -blockers, β -IHD=cases which are under medication with β -blockers, BSA=body surface area, X=heart rate, Y= O_2 cons./BSA

between total heart rate (THR) and total O_2 cons./BSA (TOC) ($R=0.63-0.79$, $p<0.001$), between the increments of heart rate and O_2 cons./BSA ($R=0.68-0.87$, $p<0.001$) and between the debts of the two factors* ($R=0.55-0.86$, $p<0.001$). Furthermore, the significant correlation between HR ($R=0.68-$

* In this study, for the purpose of convenience, the heart rate increment during the post-exercise period in its comparison with O_2 debt (Fig. 1) is called the heart rate debt.

Table III. Correlation of Total Heart Rate (THR) and Total O₂ Consumption (TOC) with Total Kpm (TKpm)

Groups	Y vs X	Y	R	P
Non β -IHD N=52	THR vs Tkpm	1.92X + 350	0.78	<0.001
	TOC vs Tkpm	14.2 X + 3,680	0.81	<0.001
Normal N=19	THR vs Tkpm	3.13X + 208	0.68	<0.001
	TOC vs Tkpm	12.3 X + 3,759	0.68	<0.001
β -IHD N=22	THR vs Tkpm	2.55X + 191	0.68	<0.001
	TOC vs Tkpm	17.7 X + 2,720	0.82	<0.001

Table IV

Items Group	O ₂ cons./min (ml)			P-R	P-Rec. 3	O ₂ pulse			P-R	P-Rec. 3
	Rest	Peak	Recov. 3 min	R × 100	Rec. 3 × 100	Rest	Peak	Recov. 3 min	R × 100	Rec. 3 × 100
Non β -IHD N=22	271.6 ± 36.3	999.7 ± 240.0	461.8 ± 97.1	266.7 ± 66.1	112.5 ± 30.9	4.39 ± 0.75	10.27 ± 2.46	6.50 ± 1.47	136.18 ± 41.38	62.5 ± 24.1
	264.3 ± 39.0	828.1 ± 114.5	447.9 ± 82.6	205.8 ± 59.1	86.82 ± 20.8	4.41 ± 1.03	9.71 ± 1.78	6.63 ± 1.42	128.55 ± 31.77	52.2 ± 27.6
P	n.s.	<0.01	n.s.	<0.01	<0.01	n.s.	<0.01	n.s.	n.s.	<0.01

Non β -IHD vs β -IHD

P=Peak, R=Rest, Rec. 3=Recovery 3 min, n.s.=not significant.

Average of total Kpm for non β -IHD was 136.9 ± 30 and that for β -IHD was 133.9 ± 31.6 .

0.78, $p < 0.001$) or O₂ cons. ($R = 0.68-0.82$, $p < 0.001$) and total work load (T kpm) was also observed as in Table III. In most of these correlations, the β -IHD group showed more similarity to the normal group than the non β -IHD group.

Effect of β -blockers on exercise O₂ consumption:—

Twenty-two tests of the β -IHD group with a range of 70–150 kpm (average 136.9 ± 29.9) were compared to 22 tests of the non β -IHD group with almost the same range of work load (average 133.9 ± 31.6) (Table IV). The Oxygen consumption per minute was significantly lower in the β -IHD group ($p < 0.01$) during exercise, while it was approximately the same at rest and 3 min after the exercise in both groups. Furthermore, similar results were obtained when the ratios e.g.,

$$\frac{\text{Peak O}_2 \text{ cons./min} - \text{Rest. O}_2 \text{ cons./min}}{\text{Rest. O}_2 \text{ cons./min}}$$

and

$$\frac{\text{Peak O}_2 \text{ cons./min} - \text{Recov. 3 min O}_2 \text{ cons./min}}{\text{Recov. 3 min O}_2 \text{ cons./min}}$$

were calculated and compared (Table IV). The same tendency was also observed in the following study II with Kö 1366 (Table VI).

Oxygen pulse was determined by dividing the amount of oxygen used during each 3 min period by the number of pulse beats or heart rate during the same period.²⁰⁾ It was almost the same at rest and early recovery period in both groups but had a lower peak during exercise in β -IHD group ($p < 0.01$).

Study II

ECG:—Alteration of resting ECG was not observed by using Kö 1366. In the first test (before Kö 1366) of $1\text{kp} \times 6\text{ min}$ exercise, 9 out of 11 cases (81.8%) showed remarkable, 1 case slight, and 1 case showed no ST-T change. On the contrary, after 1 week of medication with Kö 1366, 3 cases (27.3%)

Table V. Effect of Kö 1366 on HR and O₂ Consumption

Group	Item
		Resting		Total		Increment		Net debt		Recov. 3 min debt	
		Before	After	Before	After	Before	After	Before	After	Before	After
H.R.		195.9 ± 34.7	198.8 ± 25.4	682.1 ± 104.3	529.6 ± 57.6	309.4 ± 105.3	132.5 ± 69.1	93.2 ± 63.1	48.7 ± 34.5	42.3 ± 30.6	21.4 ± 15.7
P		n.s.		<0.001		<0.001		<0.001		<0.001	
O ₂ cons. (ml)		809.3 ± 118	805 ± 120.5	6940.8 ± 1792.7	5434.3 ± 364.3	5298.8 ± 1746.9	3816.4 ± 279.7	986.7 ± 686.4	721.1 ± 226.5	731.9 ± 332.3	663.8 ± 140.5
P		n.s.		<0.01		<0.01		<0.01		n.s.	

Table VI. Before Kö 1366 vs After Kö 1366

Items	O ₂ cons./min (ml)			P-R	P-Rec. 3	O ₂ pulse			P-R	P-Rec. 3
	Rest	Peak	Recov. 3 min	R ×100	Rec. 3 ×100	Rest	Peak	Recov. 3 min	R ×100	Rec. 3 ×100
Group	Rest	Peak	Recov. 3 min	R ×100	Rec. 3 ×100	Rest	Peak	Recov. 3 min	R ×100	Rec. 3 ×100
Before Kö N=11	272.1 ± 40.3	1085.1 ± 257.8	518.6 ± 127.8	298.7 ± 64.3	118.3 ± 40.1	4.4 ± 0.93	10.83 ± 2.86	6.36 ± 2.55	152.8 ± 47.6	65.3 ± 29.1
After Kö N=11	268.3 ± 40.3	879.5 ± 80.0	488.6 ± 64.3	230.9 ± 32.3	80.1 ± 15.4	4.3 ± 0.57	10.29 ± 1.75	7.34 ± 1.13	141.3 ± 19.1	38.6 ± 18.1
S	n.s.	<0.01	n.s.	<0.01	<0.01	n.s.	n.s.	<0.01	n.s.	<0.01

P=Peak, R=Rest, Rec. 3=Recovery 3 min.

Fig. 4. Effect of Kö 1366 on the peak change and recovery of the exercise.

showed no ST·T change to the same exercise, 6 others (54.5 %) showed slight, and only 2 cases (18.2 %) still remarkable ST·T change.

HR and O₂ cons.:—As shown in Tables V and VI, Kö 1366 had almost the same effect on the HR and O₂ cons. as the other β -blocking agents observed in Study I. But, after Kö 1366, the recovery of increased O₂ cons. from the peak response was somewhat slower in the early post-exercise period. The difference in O₂ cons., which was significant during exercise between 2 tests, became insignificant in this period (Fig. 4), while the HR in this period was still significantly lower in the second test (Table V). Oxygen-pulse had almost the same value in both tests during exercise, and it stood even higher ($p < 0.01$) in the early post-exercise period in the second test (Table V, Fig. 4). From the above mentioned results, it can be stated that recovery of O₂ consumption becomes slower in the early post-exercise period after Kö 1366 medication and suppression of HR by Kö 1366 is stronger than that of the O₂ consumption.

Syst. Blood Pressure $\times$ Heart Rate (syst. B.P. $\times$ HR):—In this study, syst. B.P. $\times$ HR was calculated before and after Kö 1366 administration and compared. The mean values during exercise and recovery fell significantly after medication ($p < 0.05$) (Fig. 5).

pH and its relation to exertion and β -blocker:—In the (1.0 kp $\times$ 6 min) exercise, pH decreased during exercise irrespective of Kö 1366. After the exercise the value of pH recovered to pre-exercise level before Kö medication, while it remained unrecovered after administration of this agent ($p < 0.02$)

Fig. 5. Effect of Kö 1366 on syst. B.P. $\times$ HR. Fig. 6. Comparison of pH and Δ pH before and after administration of Kö 1366.

(Fig. 6a). The difference of pH (Δ pH) of this group was compared with that of (0.5 kp $\times$ 21 min) exercise, before and after administration of Kö 1366 (Fig. 6b, 6c). Δ pH during exercise was significantly greater in the (1 kp $\times$ 6 min) exercise than in the (0.5 kp $\times$ 21 min) exercise, both before ($p < 0.01$) and after Kö 1366 ($p < 0.02$) (Fig. 6b, 6c). But, before Kö 1366, Δ pH in the recovery period showed no significant difference between (1 kp $\times$ 6 min) and (0.5 kp $\times$ 21 min) exercise, while there was significant difference between the 2 tests after Kö 1366 ($p < 0.01$) (Fig. 6b, 6c). Therefore it can be stated that pH decreases in correlation with the grade of load during exercise, and recovery of pH is delayed under β -blocker medication, but when the exercise load is small (adequate) this delay doesn't occur.

Study III

In this study, the mean total work load (kpm) of 6 patients was almost the same (147.6 kpm for the first test and 150.8 kpm for the second). The changes of ST-T and peak responses of HR, P.R., and O_2 cons. and the debts

Table VII. (1 Kp×6 min) vs (2 Kp×3 min)

Phase→	Rest/min		Peak/min		Rec. 3 min/min		Total		Increment		Net Debt	
Test→ Item ↓	1 Kp × 6 min	2 Kp × 3 min	1 Kp × 6 min	2 Kp × 3 min	1 Kp × 6 min	2 Kp × 3 min	1 Kp × 6 min	2 Kp × 3 min	1 Kp × 6 min	2 Kp × 3 min	1 Kp × 6 min	2 Kp × 3 min
HR n=6	66.7 ± 8.3	66.7 ± 8.3	95.0 ± 9.5	121.7 ± 6.0	67.7 ± 8.0	69.0 ± 9.2	614 ± 89	594 ± 71	240 ± 79	233 ± 45	69 ± 41	93 ± 46
P	n.s.		<0.01		n.s.		n.s.		n.s.		n.s.	
PR n=6	3011 ± 793	3011 ± 793	7494 ± 1584	11677 ± 2711	3346 ± 1150	4131 ± 1233						
P	n.s.		<0.01		n.s.							
O ₂ con. (ml) n=6	287 ± 32.9	287 ± 32.9	982.8 ± 84.2	1135.2 ± 78.3	462 ± 52.8	632.5 ± 100.7	5987 ± 593	5247 ± 628	4266 ± 598	3527 ± 669	570 ± 335	982 ± 400
P	n.s.		<0.01		<0.01		<0.05		<0.05		<0.02	

HR=heart rate, PR=pulse pressure, O₂ cons.=oxygen consumption, Rec. 3=recovery 3 min, Total, Increment, Net O₂ debt see Fig. 1.

Fig. 7. Change of pH value by (1 Kp×6 min) and (2 Kp×3 min) exercises. Recovery does not occur after (2 Kp×3 min) exercise.

of HR and O₂ cons. corresponded well with the strength of exercise (Table VII). The ECG which showed no change in 3 subjects and changed slightly in 3 others in the first test, showed 3 slight and 3 remarkable changes in the second test. Maximal values of heart rate, pressure-rate product and O₂

cons./min naturally were significantly higher in the second test ($p < 0.01$). Total and increment amounts of HR and O_2 cons. were either the same, or larger in the first test (Table VII). Oxygen debt²⁰⁾ was greater in the second test ($p < 0.02$). The mean pH value decreased during exercise in both tests (Fig. 7). Exercise acidosis (decrease of pH) was more prominent in the second test. After the exercise, in the first test pH recovered, while in the second test it remained acidotic ($p < 0.01$) even beneath the exercise level (Fig. 7).

DISCUSSION

I. Heart rate as a parameter of cardiac work

The problems of how to observe and control ambulatory IHD patients in their daily lives, are not satisfactorily elucidated. In order to answer these problems to a certain extent, we developed check-alarm-training system (CATS)³⁾ which mainly utilizes the HR, to evaluate the IHD patient's functional capacity, physical fitness and adaptability.

HR and its response to exertion differ from person to person, according to age,⁶⁾⁻⁸⁾ weight,^{2),8)} habits, training⁷⁾ etc and also according to the physical and physiological conditions of the heart. But generally the magnitude of HR response during exercise (excepting abnormalities such as complete A-V block, fixed idioventricular rhythm, etc) is known to be proportionate to the cardiac output¹⁰⁾ and body O_2 consumption.¹⁰⁾ Many authors have tried to demonstrate the changes of myocardial O_2 demand or cardiac work, which influences the ECG ST-T changes, by the changes of HR during exercise. Using the blood pressure factor, Sarnoff, Katz, and Starr have proposed their indices for the estimation of myocardial O_2 demand. Sheffield¹²⁾ states that it is reasonable to use the heart rate for finding the changes of the coronary circulation during exercise. Lau, Stafford et al¹³⁾ propose atrial pacing¹¹⁾ as a diagnostic procedure in angina pectoris, believing that the myocardial O_2 consumption increases with the HR and referring to Sjostrand,²⁾ they suggest that the HR alone may play a significant role in uncovering inadequacies in coronary reserve. Supporting these studies in general, this paper evaluates the HR as an important parameter which is able to demonstrate myocardial O_2 demand or circulatory load, not only during exercise, but also in the recovery period and not only for the normal subjects, but also for the IHD patients, regardless of their medication. Results given in Table I show the dependency of ischemic reaction of ECG on the HR response. The correlation and parallelism of HR with the O_2 consumption (Tables II and III) and pressure rate product (Fig. 3) can assert this role of the HR as a parameter. A better correlation between the increment of HR and O_2 cons. by

exercise (Table II) suggest that the evaluation of exercise effect can be preferably done by the evaluation of HR increment. The maintenance of good correlation between the debts of the 2 factors can suggest that it is reasonable to utilize the HR recovery or debt, e.g. the increment of heart rate which remained through the recovery period, for the evaluation of physical fitness. Therefore, it can be stated in short that: 1. Since the HR is so reliably proportionate to the whole body O_2 cons. and pressure rate product (circulatory load), the changes in cardiac work or myocardial O_2 demand during daily life activities can be inferred by the changes of HR. 2. In any specific case, if the relation between the magnitude of ST-T change of ECG and HR response is determined, the HR can then be used in substitution of ST-T changes in controlling the activity of that case for a certain period of time, until the patient's condition has changed and the HR-ST-T relation has been re-established.

II. Effects of β -blocking agents on exercise response and recovery

These agents, which are recently in wide use for the therapy of angina pectoris, block the adrenergic receptors of the cardiac sympathetic system, during rest and effort,^{14),17)} decrease HR, the force of ventricular contraction, cardiac output and myocardial O_2 consumption. These effects are desirable when myocardial blood flow is restricted. Favorable ECG response to exertion and improved exercise tolerance are brought about by these characteristics of β -blockers. Significant suppression of the whole body O_2 consumption during exercise (Fig. 4, Tables IV, V, and VI) indicates that the changes produced by these agents are not localized only at the cardiac level. But the high O_2 pulse in the early recovery period after administration of Kö 1366 (Table VI) suggests that the blocking effect of these drugs is more prominent on the cardiac nerves than on the rest of body. A relatively slower return of O_2 consumption and O_2 pulse from the peak exercise value toward pre-exercise level in the β -blocker administered group (Fig. 4, Tables IV and V) can be interpreted as the cardiac function has been depressed to such an extent that it couldn't send a cardiac output sufficient to supply the body O_2 demand during exercise, and therefore a compensation by a larger oxygen debt was brought about.²⁰⁾ This phenomenon was further investigated by the change of pH value (Fig. 6). The significant delay of pH recovery in the same patients after administration of Kö 1366 in the post-exercise period (1 kp 6 min) might be interpreted as a consequence of a relatively deficient O_2 supply to body tissue during and after exercise. However, further investigation is needed on the exact metabolic and/or respiratory means by which this delay of pH recovery occurs in the case of β -blocker administration.

Usually an increase in blood lactate and decrease in pH are connected with exercise¹⁵⁾ and with a high HR¹⁶⁾ (though a high HR may not be dependent on the rise of blood lactic acid) and therefore, the ECG change is more apt to happen in a low pH. But in the case of β -blocker medication, because of a depressed cardiac performance, the body O₂ demand can not be met sufficiently, therefore, a larger O₂ debt is required and probably thus results in a prolonged fall of pH. On the other hand, cardiac O₂ demand decreases because of suppressed cardiac work, which results in favorable ST-T change. Therefore, a relatively favorable ECG change can be seen in association with a low pH.

III. The effects of exercise duration and strength

Although the total amounts of O₂ cons. and HR spent for the total work were almost the same in both tests of Study III, or even less in the (2 kp $\times$ 3 min) exercise (Table VII), the instantaneous stronger cardiac work has surpassed the permissible limits of the coronary reserve, thus resulting in undesirable ECG changes. The peak values of HR, P.R., O₂ cons. during exercise and early post-exercise periods are naturally significantly higher in the (2 kp $\times$ 3 min) exercise (Table VII), accompanied by a higher O₂ debt ($p < 0.02$), a stronger exercise acidosis,²²⁾ late pH recovery (Fig. 7), and remarkable ST-T changes. This can be further realized by observing the results of the (0.5 kp $\times$ 21 min) exercise which had an even milder change of pH and a fast recovery (Fig. 6b). From these results, it can be stated that the force or strength of exercise is a more dominant factor in determining the permissible activity. Therefore, for a cardiac patient, the amount of daily activity divided by the unit of time should be strictly adequate to his physical working capacity. But this doesn't mean that the time factor of exercise, i.e. duration of exercise, can be unlimited or underestimated.

In addition, the pH in the recovery period might usefully be measured as an indicator in evaluating the adequacy of the exercise and physical adaptability of the patients.

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