TABLE I Changes in Doppler Measurements During Dobutamine Infusion

	E Velocity (cm/s)	A Velocity (cm/s)	E/A Ratio	Time from Baseline to E Velocity (ms)	Time from E Velocity to Baseline (ms)	Systolic Blood Pressure (mm Hg)	Heart Rate (beats/min)
Baseline	76 ± 9	40 ± 10	1.96 ± 0.43	90 ± 15	125 ± 7	112 ± 9	66 ± 10
2.5 μg/kg/min	88 ± 10*	43 ± 9	2.17 ± 0.48	94 ± 15	129 ± 25	115 ± 9	63 ± 13
5.0 μg/kg/min	100 ± 7*†	43 ± 6	2.41 ± 0.44	89 ± 12	123 ± 14	117 ± 11	66 ± 13
7.5 μg/kg/min	101 ± 10*†	51 ± 12	2.09 ± 0.44	91 ± 13	128 ± 13	128 ± 14*†	69 ± 13
10.0 μg/kg/min	104 ± 11*†	50 ± 9	2.16 ± 0.46	90 ± 11	128 ± 16	132 ± 8*†‡	77 ± 10

* p <0 01 vs baseline, † p <0 01 vs 2.5 μg/kg/min; ‡ p <0 01 vs 5 0 μg/kg/min

The effect of dobutamine on filling velocities in older subjects or in those with heart disease may differ from its effect in normal subjects. However, the addition of inotropic agents between serial Doppler examinations is a factor that must be considered in the evaluation of changes in early filling velocities.

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Relation of Potassium Flux During Incremental Exercise to Exercise Intensity

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Serum potassium and catecholamine levels, the latter an important regulator of potassium homeostasis,[1] both begin to increase during exercise at an intensity associated with a rise in serum lactate (lactate threshold).[2–4] This study was performed to evaluate the effect of exercise intensity on postexercise potassium flux.

Fifteen male subjects performed symptom-limited treadmill exercise (Bruce protocol) with an indwelling venous catheter in an antecubital vein. Subjects with renal disease, diabetes mellitus and those taking diuretics or medications affecting the sympathetic nervous system were excluded from evaluation to eliminate confounding variables capable of affecting potassium kinetics. Using a 3-way stopcock, venous blood samples for potassium determination were drawn through the catheter at baseline, peak exercise, 2-minute recovery and 5-minute recovery. Venous serum lactate was determined at baseline and peak exercise. Samples were drawn while the subject was standing.

Exercise-related potassium flux in subjects with peak lactate >4 mmol/liter (group 1, n = 9) was compared with subjects with peak lactate ≤4 mmol/liter (group 2, n = 6). Peak lactate >4 mmol/liter, a parameter commonly used to denote exercise intensity exceeding lactate threshold,[2,5] was used as the standard for comparison because lactate threshold has been shown to be a more useful measure of exercise intensity than external workload for studying exercise-related changes in both catecholamines[4] and potassium.[3]

Nonpaired t tests were used to detect significant differences between the 2 groups. Paired t tests were used to detect variations within group changes. Values are expressed as mean ± standard error of the mean.

The 2 groups had similar mean age, baseline lactate, peak heart rate on the treadmill and duration of exercise

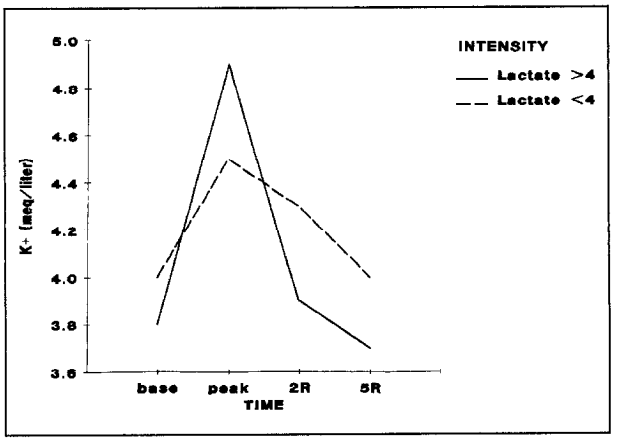

FIGURE 1. Plasma potassium as a function of time and exercise intensity. Base = baseline; K+ = plasma potassium; peak = peak exercise; 2R = 2-minute recovery; 5R = 5-minute recovery.

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(Table I). Group 1 had significantly higher peak lactate (7.9 vs 2.9 mmol, p <0.01).

The mean baseline venous plasma potassium was similar in both groups (3.8 ± 0.1 vs 4.0 ± 0.1 mEq/liter, difference not significant). The results of potassium determinations during exercise and recovery, however, demonstrated significant group differences (Figures 1 and 2).

Group 1 had significantly higher potassium at peak exercise than group 2 (4.9 ± 0.1 vs 4.5 ± 0.1 mEq/liter, p <0.01). Both groups showed a significant increase in mean potassium between baseline and peak exercise, but the rise in group 1 was significantly greater than that seen in group 2 (1.1 ± 0.1 vs 0.5 ± 0.05 mEq/liter, p <0.01).

Group 1 had significantly lower potassium than group 2 at the 2-minute recovery sampling point (3.9 ± 0.1 vs 4.3 ± 0.1 mEq/liter, p <0.04). Further, group 1 showed a significant decrease in mean plasma potassium in the peak through 2-minute recovery interval (−1.0 ± 0.1 mEq/liter, p <0.001) not observed in group 2 (−0.2 ± 0.1 mEq/liter, difference not significant).

Both groups had a statistically significant decline in potassium in the 2-minute through 5-minute recovery interval, but there was no significant difference between the groups with respect to change in potassium level in this interval.

Thus, assessment of lactate threshold enabled us to identify 2 groups with markedly different magnitude for postexercise potassium shift. Although there was no significant difference in treadmill time, subjects with peak lactate <4 mmol/liter had no significant decrease in potassium immediately after exercise, whereas subjects with peak lactate >4 mmol/liter had a mean decrease of 1 mEq/liter in the first 2 minutes of recovery.

Lactate threshold refers to the exercise intensity above which a significant rise in plasma lactate occurs.[2] This parameter was used to divide subjects into groups because both catecholamine levels[4] and potassium levels[3] have been shown to have a nonlinear increase during progressive incremental exercise, paralleling the curve of exercise-related elevation of plasma lactate. The lactate threshold reflects a physiologic response to exercise and does not occur at the same percentage of maximal heart rate in all patients, nor can it be related to the same percentage of maximal oxygen uptake. Accurate detection requires direct assessment by venous sampling of lactate or indirect assessment through measurement of ventilation and gas exchange during exercise.[2]

The data from this study provide a framework for additional investigations, including determining the effect of a postexercise "cooling down" period and the effect of medications such as diuretics on exercise-related potassium flux.[6] Of note, the clinical significance of potassium flux immediately following exercise remains to be determined. Exercise-related arrhythmia frequently occurs during recovery[7,8] and may in part be related to a rapid decrease in potassium from the level reached at peak exercise. Evaluating patients with known exercise-related arrhythmia in terms of lactate threshold and associated exercise-related changes in potassium and catecholamines may provide important diagnostic and therapeutic information.

TABLE I Comparison of Group 1 (Peak Lactate >4 mmol/liter) with Group 2 (Peak Lactate ≤4 mmol/liter)

	Group 1* (n = 9)	Group 2* (n = 6)	p Value
Age (yrs)	57 ± 3	63 ± 5	NS
Duration (min)	9.9 ± 0.8	8 8 ± 1.0	NS
Peak heart rate (beats/min)	155 ± 6	137 ± 14	NS
Lactate-base	1.2 ± 0.1	1.2 ± 0.1	NS
Lactate-peak	7.5 ± 0.6	2.9 ± 0.3	<0.001

* Mean ± standard error of the mean
No subject in either group had significant exercise-related arrhythmia
NS = not significant.

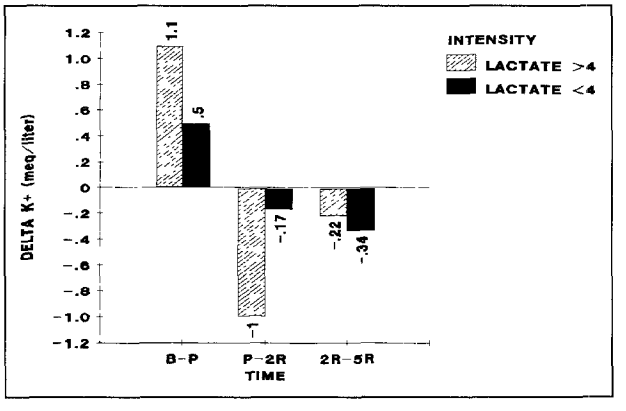

FIGURE 2. Interval change in plasma potassium during exercise and recovery as a function of exercise intensity. B-P = interval between baseline and peak exercise; DELTA K+ = change in potassium over sampling interval (postive and negative values denote increases and decreases in potassium levels, respectively); P-2R = interval between peak exercise and 2-minute recovery; 2R-5R = interval between 2- and 5-minute recovery.

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