Relationship between ventilation and arterial potassium concentration during incremental exercise and recovery

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Summary. The purpose of this study was to compare the relationship of ventilation (\(V_E\)) with pH, arterial concentrations of potassium ([K⁺]ₐ), bicarbonate ([HCO₃⁻]ₐ), lactate ([lact]ₐ), and acid-base parameters which would affect hyperpnoea during exercise and recovery. To assess this relationship, ten healthy male subjects exercised with intensity increasing as a ramp function of 20 W-min⁻¹ until voluntary exhaustion and they were then allowed a 5-min recovery period. Breath-by-breath gas exchange data, [HCO₃⁻]ₐ, pH, [lact]ₐ, [K⁺]ₐ, and blood gases were determined during both exercise and recovery. Using a linear regression method, the \(V_E/\text{[K⁺]}ₐ\) relationship was analysed during both exercise and recovery. Several interesting results were obtained: a significant relationship between [K⁺]ₐ and \(V_E\) was observed during recovery as well as during exercise; the \(V_E\) at any given values of [K⁺]ₐ was significantly higher during recovery than during exercise and out of those factors affecting exercise hyperpnoea, only [K⁺]ₐ had a similar time-course to \(V_E\) during recovery. Changes in [K⁺]ₐ during recovery were shown to occur significantly faster than \(V_E\) with an [K⁺] time constant of 70.0 s, SD 16.2 as opposed to 105.5 s, SD 10.0 for \(V_E\) (P<0.01). These results provided further evidence that enhanced [K⁺] might derive from muscle depolarization during exercise in humans as well, stimulating carotid bodies and enhancing sinus nerve discharge. Exercise hyperpnoea might be ascribed to this process.

However, immediately after maximal exercise, severe metabolic acidosis occur and acid-base regulation is disturbed, while \(V_E\) and [K⁺] increase rapidly in parallel. We hypothesize that [K⁺]ₐ may play an important role in controlling \(V_E\) even during recovery.

The purpose of the present study was to assess the effect on hyperpnoea during exercise and recovery as it relates to levels of [K⁺]ₐ, pH, bicarbonate concentration ([HCO₃⁻]ₐ), and related acid-base parameters.

Methods

Subjects. Ten healthy male university students volunteered as subjects for this study. The subjects average age, height and body mass was 19.1 years, SD 1.0, 170.7 cm, SD 1.3, and 59.6 kg, SD 8.6, respectively, while maximal oxygen uptake (\(\dot{V}O_2\max\)) and the peak power output averaged 44.4 ml·kg⁻¹·min⁻¹, SD 7.0 and 245 W, SD 30 W, respectively. All subjects were familiar with the laboratory from previous experimentation, and after being informed of the purpose and possible risks of the present study, they gave written consent to serve as subjects.

Exercise protocol. Exercise was performed in an upright position on an electrically-braked cycle ergometer (Siemens Elema 780, Solna, Sweden). The protocol consisted of the subject sitting quietly at rest while \(V_E\) and gas exchange were monitored breath-by-breath. After the breathing pattern became stable, resting arter-
ial blood sampling was performed. The subject was instructed to warm-up by pedalling at 20 W for 4 min at a rate of 60 rpm. The exercise intensity was then increased by 20 W every min as a ramp function, which was generated by an analog integrating circuit with a linear output in the 0-500 W range. The increments continued until the subject could no longer maintain his pedalling frequency. The highest oxygen uptake (VO₂) measured was judged to be the subject's VO₂max. The subject was then allowed to recover while continuing to cycle at 60 rpm on the unloaded cycle. Analyses of breath-by-breath gas exchange and arterial blood samples were again obtained during recovery.

Data collection. The VO₂ and VO₂ were measured with a computerized on-line breath-by-breath system (RM-300, Minato Medical Science, Osaka, Japan). Inspired and expired gas volumes were measured by using a hot-wire respiratory flow system. The concentrations of O₂ and CO₂ were analysed with a zirconium solid electrolyte oxygen analyser and infra-red carbon dioxide analyser, respectively. To calculate breath-by-breath gas exchange parameters, a time delay (transport and dynamic response delay) of gas concentrations was used against which the gas flow was compensated (Noguchi et al. 1982). The data were stored on disk for further analysis.

Arterial blood sampling and analyses. To obtain the arterial blood samples, a Teflon catheter was inserted into the radial artery after local anaesthesia with 0.5% xylocaine. After the catheterization, each subject sat for at least 15 min on the ergometer and the resting measurements were then made. During the incremental exercise test and the succeeding recovery period, arterial blood samples were taken every minute over several respiratory cycles to avoid the fluctuation of blood gases due to breathing.

To prevent artificially high [K⁺]a, blood was drawn without the use of a tourniquet and fist-clenching, and any sample showing signs of haemolysis was discarded. Samples for [K⁺]a were analysed in duplicate by flame photometry.

Arterial pH, arterial partial pressure of oxygen (PaO₂), and arterial partial pressure of carbon dioxide (PaCO₂) were analysed by glass electrodes (Instrumentation Laboratory model 813/blood gas analyser, Mass, USA). The [HCO₃⁻]a was calculated by the Henderson-Hasselbalch equation. Arterial blood lactate concentration [lact]a was determined enzymatically from blood supernatant (Boehringer, Mannheim, FRG).

Statistical analyses. Linear regression analysis and correlation coefficients were determined by an appropriate procedure. Paired t-tests were applied for the analyses of the differences obtained.

Results

Figure 1 gives an example of the time-course of breath-by-breath VO₂ (A), VE (B) and [K⁺]a (C) during incremental exercise and recovery for one subject. In Fig. 1C, [K⁺]a rose from 3.68 mM gradually increasing to 6.71 mM at exhaustion. Immediately after exercise, [K⁺]a decreased abruptly. The time-course of changes in arterial [K⁺]a was similar to that for VE (Fig. 1B) during both exercise and recovery, but it deviated from that for VO₂ (Fig. 1A).

The relationships between [K⁺]a and VE during incremental exercise and recovery are plotted for all subjects (Fig. 2). There are significant correlations between [K⁺]a and VE during both exercise and recovery (P<0.001). As shown in Fig. 2, the values of VE at any given [K⁺]a during recovery are significantly higher than those during exercise (P<0.01).

Figure 3 indicates the time-courses of [HCO₃⁻]a (A), pH (B), [lact]a (C), [K⁺]a (D), PaO₂ and PaCO₂ (E) and VE (F) during exercise and recovery. During the incremental exercise, [HCO₃⁻]a (A) and pH (B) decreased while [lact]a (C) and [K⁺]a (D) increased in accordance with the exercise intensity. The trends of change in [HCO₃⁻]a (A), pH (B) and [lact]a (C) continued during recovery, the first two decreasing, the latter increasing. Conversely, [K⁺]a (D) started to decrease immediately after exercise. Only [K⁺]a had a similar time-course to VE during recovery. During recovery the time constants were 70.0 s, SD 16.2 for [K⁺]a and 105.5 s, SD 12.0 for VE; the difference being significant (P<0.01).