PHYSIOLOGICAL AND PERFORMANCE CHANGES OF ELITE ICE HOCKEY PLAYERS IN RESPONSE TO ACUTE EXPOSURE TO MODERATE ALTITUDE.

by

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We accept this thesis as conforming to the required standard

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ABSTRACT

The purpose was to determine if a change in altitude from 850m to 1650m would cause acute physiological and performance changes in elite male ice hockey players and to determine if initial acclimatization to the higher altitude could be accomplished in a three day stay at that altitude. To do this, 17 trained male ice hockey players were studied at rest, during submaximal exercise and during on-ice performance at 850m and for three days at 1650m. Resting blood samples were analyzed for hematocrit (Hct), hemoglobin (Hb), total white blood cell count (TWB) and differentials. Plasma volume decreased 5% ($p<0.05$) during exposure to altitude and Hct and Hb both showed corresponding increases from 44±2% to 46±1% and 15.1g·100mL^{-1} to 15.7g·100mL^{-1} respectively ($p<0.05$). TWB did not change significantly, however, neutrophils showed a 30.2% decrease and lymphocytes a 30.5% increase by day 3 at altitude ($p<0.05$). Upon exposure to altitude resting heart rate (HR) increased from 56±9 beats·min to 60±10 beats·min ($p<0.05$), but had decreased back to pre-altitude levels by day 2 at altitude. Resting percent oxygen saturation (SaO_2) did not change significantly. Submaximal exercise responses, ventilation (V_E), oxygen consumption (VO_2), HR, SaO_2, and blood lactate concentration (BL) were measured using a cycling protocol. V_E and VO_2 increased 5.0% and 7.9% respectively on day 1 at altitude ($p<0.05$). HR was not significantly different at altitude at the same workload compared to the pre-altitude test. SaO_2 measured during the last minute of exercise decreased 3% from the pre-test value of 94% on days 1 and 2 (91%) at altitude ($p<0.05$), but by day 3 had increased back to the pre-altitude level. BL decreased by 1.0 mmol·L^{-1} by day 2 at altitude ($p<0.05$). On-ice performance variables, agility tests, 60 and 120 ft sprint and drop-off tests and HR and SaO_2 were measured at 850m and 1450m

altitude. The pre and post-sprint agility tests were slower (3% and 2% respectively) on day 1 at altitude ($p<0.05$). At altitude, the pre/post-sprint agility test difference was not significantly different ($p>0.05$). The 60 ft and 120 ft sprint times and drop-off times were not significantly altered by altitude ($p>0.05$). Recovery HR following the repeated sprint test was not significantly different at altitude, but SaO_2 at 15, 30 and 60 seconds recovery was significantly lower on days 1 and 2 at altitude. These findings suggest that there are some selected physiological responses in elite ice hockey players with acute exposure to moderate altitude and that some initial acclimatization does occur within 3 days of exposure. Furthermore, there did appear to be a related decrease in some of the aerobic aspects of on-ice performance.

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Dedication

To my Mom and Dad who always encouraged my pursuit of an education and to my husband, Mark, whose support, love and enthusiastic encouragement kept me going.

INTRODUCTION

Upon exposure to altitude, the human body undergoes physiological changes as it acclimatizes to the decreased alveolar (PAO_2) and arterial oxygen pressure (PaO_2) which occur as a result of the decreased partial pressure of oxygen in the air (Welch, 1987; Smith & Sharkey, 1984). The initial stage of altitude acclimatization is characterized by some functional changes which collectively facilitate O_2 transport in the hypoxic environment (Buskirk, Kollias & Picon-Reategui, 1966) and include an increase in ventilation (V_E) to increase alveolar O_2 concentration (Burki, 1984; Hannon & Vogel, 1977) and an increase in erythrocyte production to increase the O_2 carrying capacity of the blood (Birchard & Tenny, 1991; Hannon & Vogel, 1977; Klausen, Mohr, Ghisler & Nielsen, 1991). As a result of dehydration there is also a decrease in plasma concentration (PC) and subsequent increase in Hct (Jung, Dill, Horton & Horvath, 1971).

These initial acute effects of altitude exposure cause other physiological changes such as increased heart rate (HR), increased resting blood lactate and pyruvate concentrations and an initial increase in blood buffering capacity, which, as HCO_3^- is excreted by the kidneys during the first 24 hours of hypoxic exposure, becomes a diminished blood buffering capacity. With acclimatization to altitude, V_E, SaO_2, HR, PV, blood lactate and pyruvate concentrations return back to pre-altitude levels (Hansen, Stelter & Vogel, 1967; Jung et al., 1971; Wenger, 1986).

Hypoxia and the subsequent physiological changes that occur cause a decrement in performance in athletic events that require the aerobic energy system for energy (ATP) production or regeneration. Acclimatization results in an improvement in altitude

performance. The time that it takes to acclimatize to altitude is dependent upon the altitude of ascent. It usually takes 1-2 weeks for the initial effects of hypoxic exposure to subside (Astrand & Rohdal, 1986), thus recommended acclimatization time for sea level natives is 14 days for 2000m and increases as altitude increases (Ratzin-Jackson & Sharkey, 1988; Smith & Sharkey, 1984). However, moderate altitude natives (residence = 800-1200m) may require even less time to acclimatize to higher altitudes. Noble & Maresh (1979) and Maresh, Kraemer, Noble and Robertson (1988) showed that moderate altitude natives (residence = 1830-2220m) experienced a less impaired capacity for O_2 uptake at 4300m altitude and did not display the magnitude of hypoxic response expected of sea level residents. No studies have been conducted to determine if residents of 800-1000m would also acclimatize to moderate altitudes of 1400-2200m more rapidly than sea level residents.

The type of training in which athletes engage is one determinant of their success to acclimatize to altitude and subsequently perform as well as they do at sea level. Athletes that train for and compete in events that depend primarily on the aerobic system for energy production find altitude detrimental to performance. A high maximal oxygen consumption (VO_2 max) does not ensure tolerance to high altitude (Cymerman et al., 1989). Subjects with higher VO_2 max are more affected by altitude than persons with lower maximal aerobic power because their VO_2 max is more fragile (Astrand & Rodahl, 1986, 690). However, even though the effect of altitude is greater on athletes with a higher VO_2 max, they still have higher relative uptakes at altitude than less aerobically fit athletes (Cymerman et al., 1989).

Post-exercise recovery is also affected by altitude because of increased muscle lactate, decreased SaO_2 and decreased blood flow to the tissues (Linnarsson, Karlsson, Fagraeuss & Saltin, 1974; Lorentzen, 1962). Thus, athletes that depend on their

aerobic system for energy such as those who compete in events that last longer than 2 minutes (Haymes & Wells, 1986) or in sports that require fast recovery from repeated anaerobic work such as in hockey (Jette, 1980) or basketball (Noble & Maresh, 1979) find altitude detrimental to performance.

Saltin (1967) suggested that differences in anaerobic capacity influences performance at altitude. At altitude, a larger portion of energy is derived from the anaerobic system for a given workload, thus leading to fatigue earlier than it would occur at sea level (Faulkner et al., 1968). Saltin (1967) suggested that athletes who have larger anaerobic capacities would have an advantage at altitude in that they would be able to maintain a given work intensity for longer and would be better able to tolerate a decreased muscle pH than those athletes with smaller anaerobic capacities.

No studies have been conducted to determine the effects of acute moderate altitude exposure on elite athletes who participate in a sport which relies heavily on both the anaerobic and aerobic energy systems such as ice hockey. Due to the intermittent nature of the game of hockey, both the aerobic and anaerobic systems are involved in the metabolic process of energy production (Green, 1979; Green & Houston, 1975).

About 70% of energy expenditure in a single game by an ice hockey player is anaerobic (Jette, 1980). Thus, a large part of the training program for hockey players is development of anaerobic capacity (Rhodes & Twist, 1989). In view of Saltin's (1967) findings, ice hockey players may be better able to tolerate hypoxic stress than less anaerobically fit athletes because of their well-trained anaerobic systems.

However, ice hockey players require a recovery period during and between their on-ice shifts. The need for a recovery time arises from the anaerobic energy sources being utilized repeatedly (Watson & Hanley, 1986). Recovery between high intensity work bouts is primarily an aerobic event (Green, 1979). At sea level, recovery of the

anaerobic alatic and lactic systems is rapid and a subsequent anaerobic exercise bout can begin after a 30 to 60 second recovery (McKardle, Katch & Katch, 1991). Because of the increased muscle lactate accumulation and impaired oxygen delivery at altitude, a longer recovery time may be required.

Knowledge of performance at altitude is based primarily on research done at high altitude (above 3048m) and has focused mainly on the effect of altitude on aerobic exercise. However, most athletes train and compete at moderate altitude elevations (Ratzin-Jackson & Sharkey, 1988). In addition, many athletes compete in events requiring anaerobic energy production as well as aerobic energy production. The effect of moderate altitude (1400-3000m) on the physiological responses and performance of elite athletes involved in sports utilizing both the aerobic and anaerobic energy systems are still relatively uninvestigated.

The Canadian Olympic Hockey Team competed in the 1992 Winter Olympics in France. The accommadation for the team was located at 1650m, but they played their games at 1500m. Because of their tournament schedule before the Olympics, the team could only arrive at altitude 3 days prior to their first game. This purpose of this study, completed at Lake Louise (1650m) and Banff (1450m), was to simulate the situation the players were to face in France in order to determine if altitude would affect the players performance and to determine if the players could acclimatize to 1650m in three days.

Statement of the Problem

The purpose of this study was to determine if a change in altitude from 850m to 1650m would cause acute physiological and performance changes in elite male ice-hockey players and to determine if initial acclimatization to 1650m could be accomplished in a three day stay at this altitude.

Research Questions

1. What are the selected physiological responses at rest when moving from a low altitude of 850m to a moderate altitude of 1650m?

2. Are there differences in these responses after 2 or 3 days at altitude?

3. What are the selected physiological responses during submaximal exercise when moving from a low altitude of 850m to a moderate altitude of 1650m?

4. Are there differences in these responses after 2 or 3 days of exposure to moderate altitude?

5. Do the physiological changes reflect a difference in performance as measured by maximal on-ice speed, agility and drop off times at 1450m?

6. What are the possible implications on performance at altitude for elite ice hockey players?

METHODS

Subjects

Seventeen healthy trained males from the Canadian Olympic Hockey Team made up the subject population. The physical characteristics of the subjects are displayed in Table 1. All subjects received medical clearance from the team doctor to participate in the study and signed an informed consent (Appendix D).

Experimental Design

The dependent variables were: body weight; resting, exercise and recovery heart rate (HR); hemoglobin (Hb), hematocrit (Hct), plasma concentration (PC), white blood cell total and differential counts and percent arterial O_2 saturation (SaO_2) at rest, during submaximal exercise and after supramaximal exercise; submaximal exercise ventilation (V_E), oxygen consumption (VO_2), exercise blood lactate, time to steady state VO_2 and HR and perceived exertion; and on-ice sprint, agility and drop off tests. The independent variables were altitude elevation and the number of days of exposure to moderate altitude. The time of day for the tests of body weight, resting heart rate, hemoglobin, hematocrit, and plasma volume was between 0700 and 0800h. On-ice agility, sprint and drop-off tests took place from 0930 to 1015h before a morning practice and were measured at 1450m altitude (Banff). The submaximal testing sessions took place between 1230 and 1430h. The resting and submaximal exercise variables were measured at 1650m altitude (Lake Louise). The post-altitude test took place in Calgary 5 weeks after the altitude exposure.

Control variables were the sampling time, the subject's body position prior to and during the sampling of resting HR and SaO_2 and blood measures, and meal times. Sleep and hydration requirements were also recommended.

Table 1. Physical characteristics of subject pool of elite, male hockey players (n=17).

	Mean	SD	Range
Age (years)	22.9	2.25	19-27
Body Mass (kg)	87.5	5.0	76.6-94.2
Height (cm)	183.8	6.2	172.7-193.0
% Body Fat	12.9	1.5	11-16
VO2 max ($L \cdot min^{-1}$)	4.93	0.33	4.55-5.77
VO2 max ($mL \cdot kg^{-1} \cdot min^{-1}$)	56.8	4.02	49.0-62.6

Experimental procedures

VO_2 Analysis

Subjects expired air through a Rudolph valve into the mixing chamber of the Beckman Metabolic Measurement Cart (MMC). HR, VO_2 and V_E were monitored every 30 seconds. The oxygen (OM-11) and carbon dioxide (LB-2) analyzers of the MMCs were calibrated against primary gas standards before and after each submaximal and maximal test. The MMCs were calibrated for volume and barometric pressure before and after each testing session. The maximum error of the MMCs over a 1 hour period after calibrations are: $FEO_2 \pm 0.2\%$, $FECO_2 \pm 0.1\%$, barometric pressure $\pm$ 5mmHg, temperature $\pm$ 0.5°C, and volume $\pm$ 2% (Norton, 1976). If calibration did not remain stable from pre to post-test, calculations were made to adjust for the fluctuations. Each subject was assigned to one of the MMCs and was monitored on that same cart for all of the maximal and submaximal exercise testing sessions.

A Monarch cycle ergometer (model #868) was calibrated before each testing session to ensure that the resistance settings were consistent across all trials. The seat height on the cycle ergometer was adjusted for each subject so that there was 5° flexion of the knee when the foot was at the lowest point on the pedal. The subjects cycled at 70 rpm during all tests and used the same ergometer for all tests.

Blood Sampling

Sterile techniques were used with all blood sampling. Resting blood samples were drawn by venapuncture of the anticubital vein. For each sample, 7 mL of blood was drawn into a syringe using a 21 gauage needle and transferred to pre-cooled vacutainers

containing EDTA. All tubes were labelled with subject ID number, the date, and the time of the test. The vacutainers were delivered to the Alberta Medical Laboratories within 30 minutes of the last sample being drawn for analysis of Hb, Hct, red and white blood cell counts and white blood cell differentials. Plasma concentrations were calculated using the Dill and Costill (1974) equations based on Hct and Hb (Appendix E). Total white blood cell counts and white blood cell differentials were corrected for changes in plasma concentration.

Blood lactate samples were obtained via fingerprick and subsequently analyzed in duplicate for lactate using a Yellow Springs Instrument Lactate Analyzer (model #23L).

VO_2 max protocol

VO_2 max tests were administered in August 1991 and November 1991. Monarch cycle ergometers (model # 868) were used for the testing. The protocol was a 2 min warm-up at 2 kp, 2 min at 3 kp, 2 min at 4 kp, 1 min at 5 kp, and a 1 kp increase per minute thereafter until VO_2 max was reached. VO_2 max was considered to have been reached when there was an increase in VO_2 of less than 2 $mL \cdot kg^{-1} \cdot min^{-1}$ with an increase in load.

Resting Conditions

Subjects were awakened daily at their scheduled time and immediately went to the testing room where their resting HR and SaO_2 saturation were measured and recorded using a Criticare Systems Pulse Oximeter (model # 504-US). The error of measurement of the Pulse Oximeter is ±2% for arterial oxygen saturations between 70-100%. A resting blood sample was also taken at this time on day 0 (in Calgary), on

days 1 and 3 at altitude (in Lake Louise) and five weeks after altitude exposure (in Calgary).

Submaximal exercise protocol

A submaximal cycle ergometer test occurred daily before altitude exposure, during altitude exposure and at the post-altitude test 5 weeks after exposure. For all subjects the initial resistance was set at 2.0 kp for 1 minute, increased to 3.0 kp for 1 minute and then 4.0 kp for 6 minutes.

An arterialized blood sample was taken during the final minute of exercise via a finger prick. Percent arterial O_2 saturation (SaO_2) was measured prior to starting the test, during final minute of exercise and 30 and 60 seconds after the cessation of exercise.

Four experienced technicians were required at each testing session. There was one technician on each MMC, another drew blood samples and another technician was used to spot to ensure subject safety, and to prepare subjects for the test.

On-ice Tests

The on-ice testing session consisted of an agility test, a drop-off/sprint test and a final agility test. The agility test required the subject to skate around five pylons placed 15 ft apart without a puck. The start line was located 5 ft behind the first pylon and the finish line was the first pylon (see Appendix F for a diagram of the setup). Timing was done by two testers using manual timing devices. Each subject was allowed two trials without the puck and two trials with the puck. If he fell, the fall was recorded and he was allowed to rest and then attempt the test again. All times were recorded and the fastest times on each day were used in data analysis.

The 120 ft sprint test and the drop-off test were combined in one protocol. Infra Red Electronic Eyes (model #63501-IR) and a stop clock (model #54050) were used to record split times at 60 and at 120 feet . The subjects were allowed to choose the forward or side position to start and had to use that same position to start on all subsequent tests. The drop-off test required the subject to complete 5 repeat sprints at 20 second intervals. A manual timing device was used to time the 20 second intervals. The subjects started 1m behind the first photocell and sprinted 120 ft, circled back to the start and had to be ready to start the next sprint at 20 seconds (see Appendix F for a diagram of the setup). The fastest 60 ft and 120 ft intervals were used as the sprint times. The drop-off was the difference between the fastest and slowest intervals in the 5 repeats. When the subjects finished the drop-off test, they went straight to a designated tester who recorded HR and SaO_2 15, 30, 60, and 90 seconds following the final sprint. At exactly 120 seconds after the completion of the drop-off test the subjects repeated the agility test using the protocol described above.

STATISTICS

A one-way ANOVA with repeated measures was used to determine the physiological responses when moving from 850m altitude to 1650m altitude and after 1, 2 and 3 days of exposure to moderate altitude. A post-hoc Newmans Keuls was used where warranted.

RESULTS

The effects of exposure to altitude on selected physiological and performance variables are described in three sections depending on the conditions under which the dependent variables were measured: at rest, during and after submaximal exercise, and on-ice.

Rest Conditions

Heart rate and Blood Oxygen Saturation

Body weight did not significantly change during the three day exposure to altitude or from the pre to post-altitude test (Table 6, Appendix C). There was a significant increase in resting heart rate on day 1 (60±10 beats·min^{-1}) at altitude compared to the pre-test in Calgary (56±9 beats·min^{-1}), but not on days 2 and 3 (57±9 and 58±11 beats·min^{-1} respectively) (Table 2).

Resting SaO_2 decreased only 1% upon ascent to altitude and remained at this level for the entire exposure (Table 2).

Plasma concentration, hemoglobin and hematocrit

Plasma concentration (PC) decreased 5% ($p<0.05$) during the exposure to altitude from 55.1 mL·100mL^{-1} in Calgary to 52.4 mL·100mL^{-1} on day 3 at Lake Louise (Table 2). Post-altitude PC was not significantly different to pre-altitude PC or PC at altitude. Hematocrit increased significantly during the exposure to altitude from a

pre-altitude value of 44% to 46% on the final day at altitude. From the pre to post-test in Calgary there was a 3% increase in hematocrit (Table 2).

Hemoglobin increased from 15.1 $g \cdot 100mL^{-1}$ pre-altitude to 15.7 $g \cdot 100mL^{-1}$ on day 3 at altitude. In the post-test, hemoglobin increased by 0.7 $g \cdot 100mL^{-1}$ over the pre-test value (Table 2).

White blood cells

Total white cell count was not significantly different on any day at altitude compared to the pre-altitude test when corrected for plasma concentration changes.

Cell differentials changed significantly over the altitude exposure. There was a 23.8% and 30.2% decrease in neutrophils by days 1 and 3 at altitude respectively and an increase in lymphocytes of 24.7% by day 1 and 30.5% by day 3 of altitude exposure when compared to pre-altitude values (Figure 1). Both differentials returned to pre-altitude levels in the post-test.

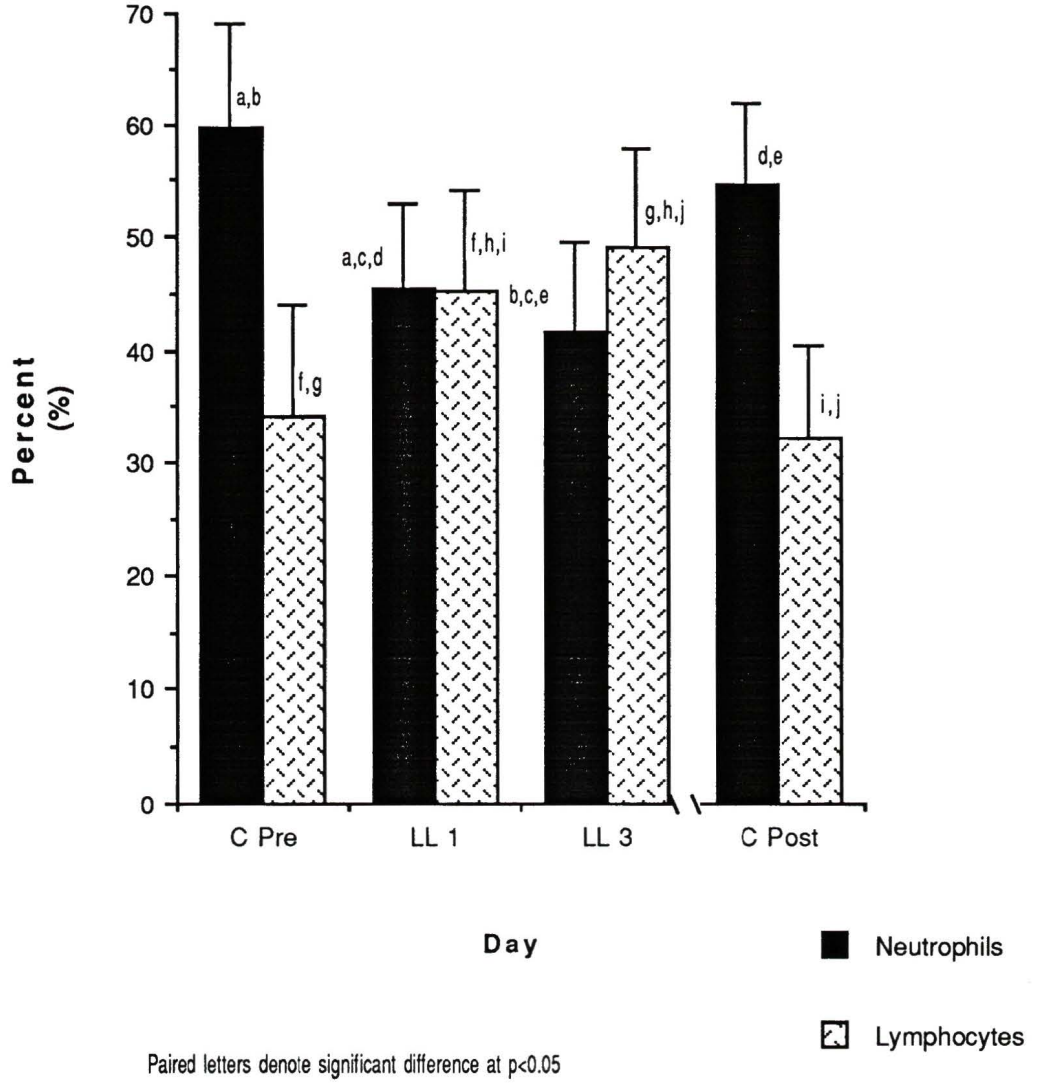
Table 2. Mean (SD) resting heart rate, SaO_2 and selected blood variables of elite male ice hockey players pre-altitude, at altitude and 5 weeks after altitude exposure.

Day	Calgary Pre	LL/Banff 1	LL/Banff 2	LL/Banff 3	Calgary Post 5 wks
Heart Rate (beats·min^{-1})	56 (9)^{a}	60 (10)^{a}	57 (9)	58 (11)	
O_2 Saturation (%)	98 (1)	97 (1)	97 (1)	97 (1)	98 (1)
Plasma Conc (mL·100mL^{-1})	55.1 (1.9)^{a}	54.6 (3.1)^{b}		52.4 (2.5)^{a,b}	53.1 (1.3)
Hemoglobin (g·100mL^{-1})	15.1 (0.66)	15.2 (0.62)		15.7 (0.48)*	15.8 (0.44)*
Hematocrit (%)	44 (2)^{a,b}	45 (2)^{c}		46 (1)^{a}	47 (1)^{b,c}
White Cells ($x10^{9}$·L^{-1})	5.6 (1.2)	5.9 (0.7)		6.0 (0.6)	5.5 (0.2)

* Significantly different to unstarred values at $p<0.05$.

^{a,a} Paired letters denote significant differences at $p<0.05$.

FIGURE 1. Neutrophil and lymphocyte differentials measured as a percent of total white blood cell count before altitude exposure (C Pre), during exposure (LL1 and LL3) and 5 weeks after altitude exposure (C Post)



Paired letters denote significant difference at $p<0.05$

Submaximal Exercise

VO_2 and V_E

During submaximal exercise performed at altitude on Day 1 of exposure, VO_2 was 5.0% and V_E was 7.9% higher than the pre-test in Calgary (Table 3). On the subsequent days of exposure and in the post-test, VO_2 and V_E decreased significantly from the Day 1 test at altitude to values similar to the pre-altitude test (Table 3). Time to reach a steady state VO_2 was not significantly different from pre-altitude or on any day at altitude (Table 6, Appendix C).

Heart rate and oxygen saturation

Submaximal heart rate was not significantly different at altitude at the same workload compared to the pre or post-altitude test (Table 3). Time to reach a steady state HR was not significantly different on any of the testing days (Table 6, Appendix C).

Blood oxygen saturation measured during the last minute of exercise decreased 3% from the pre-test value (94%) on days 1 and 2 (91%) at altitude, but on Day 3 had increased back to the pre-altitude level (95%) (Table 3).

Blood lactate

Blood lactate concentration during the final minute of submaximal exercise decreased by 1.0 $mmol \cdot L^{-1}$ on day 2 at altitude compared to both the pre-test value and day 3 at altitude. It was also significantly lower on days 1 (5.1 $mmol \cdot L^{-1}$) and 2 (4.7 $mmol \cdot L^{-1}$) at altitude when compared to the post-test value (6.2 $mmol \cdot L^{-1}$) .

Table 3. Mean (SD) selected submaximal exercise responses of elite male ice hockey players before, during and 5 weeks after altitude exposure.

Day	Calgary Pre	LL/Banff 1	LL/Banff 2	LL/Banff 3	Calgary Post 5 wks
VO_2 ($L \cdot min^{-1}$)	3.62 (0.22)	3.81 (0.28)*	3.71 (0.20)	3.65 (0.24)	3.60 (0.14)
Ventilation ($L \cdot min^{-1}$)	109.1 (16.8)	118.4 (17.1)*	109.9 (15.3)	110.3 (16.0)	109.5 (14.3)
Heart Rate ($beats \cdot min^{-1}$)	162 (11)	162 (9)	160 (10)	159 (10)	162 (8)
O_2 Saturation (%)	94 (3)	91 (3)*	91 (2)*	95 (2)	94 (5)
Blood Lactate ($mmol \cdot L^{-1}$)	5.7 (1.8)[a]	5.1 (1.2)[b]	4.7 (1.5)[a,c,d]	5.8 (2.1)[d]	6.2 (2.1)[b,c]

* Significantly different from unstarred values at $p<0.05$.

[a,a] Paired letters denote significant differences at $p<0.05$.

On-ice performance tests

Agility tests

Both the pre and post-sprint agility tests on day 1 at altitude were significantly slower (3% and 2% respectively) compared to the Calgary tests. The post-sprint agility test on day 3 at altitude was significantly faster than the pre-altitude post-sprint test in Calgary or day 1 at altitude (Figure 2).

In the Calgary pre-altitude test the post-sprint agility test was 0.23 seconds slower than the pre-sprint agility test. In the Calgary post-test, this difference was 0.20 seconds. At altitude the pre/post-sprint agility test difference was not significantly different.

60 ft and 120 ft sprint and drop-off times

No significant difference in times were found for the 60 ft and 120 ft sprints between the pre and post-altitude tests in Calgary and the tests at altitude (Table 4). Drop-off times between the slowest and fastest sprint were also not significantly altered by altitude.

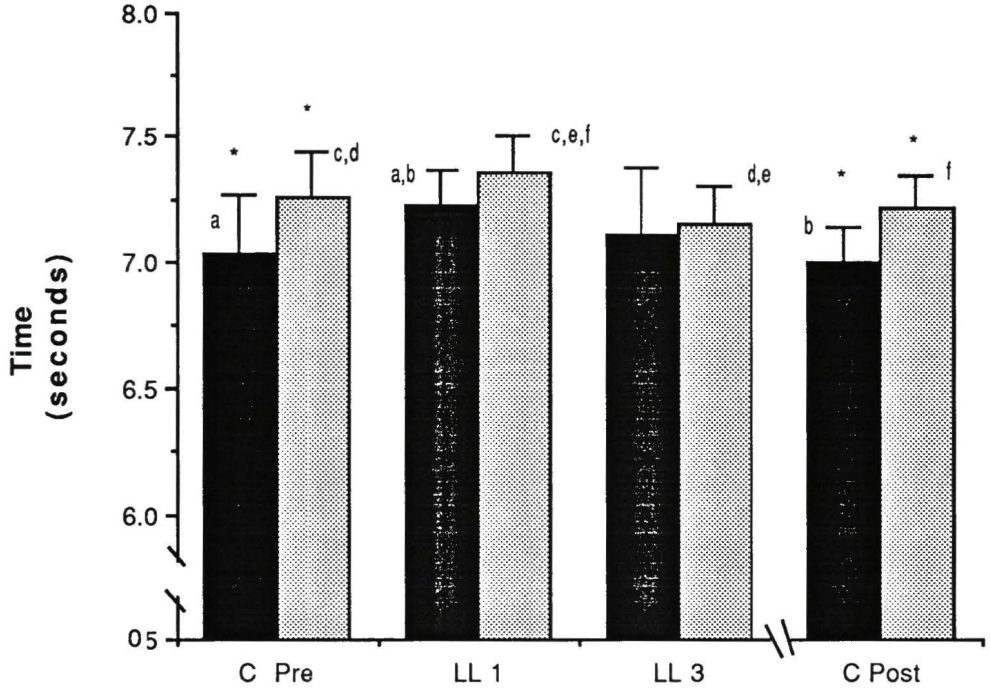
Heart rate and SaO_2

Recovery heart rates at 15, 30, 60 and 90 seconds after the repeated sprint test were not significantly different at altitude compared to the pre and post-altitude tests (Table 5).

SaO_2 15 seconds and 30 seconds after completing the sprint test was significantly lower on day 1 at altitude compared to all other days (Figure 3). SaO_2 60 seconds after

completing the sprint test on days 1 and 3 at altitude was significantly lower than the pre-altitude test but there was no significant difference on any of the days after 90 seconds of recovery (Figure 3).

FIGURE 2. On-ice agility test time in seconds before and after the repeated sprint test before (C Pre), during (LL1 and LL2) and 5 weeks after altitude exposure (C Post).



Paired letters denote significant differences at $p<0.05$.

* Denotes significant differences between Pre and Post-sprint times on the same day at $p<0.05$.

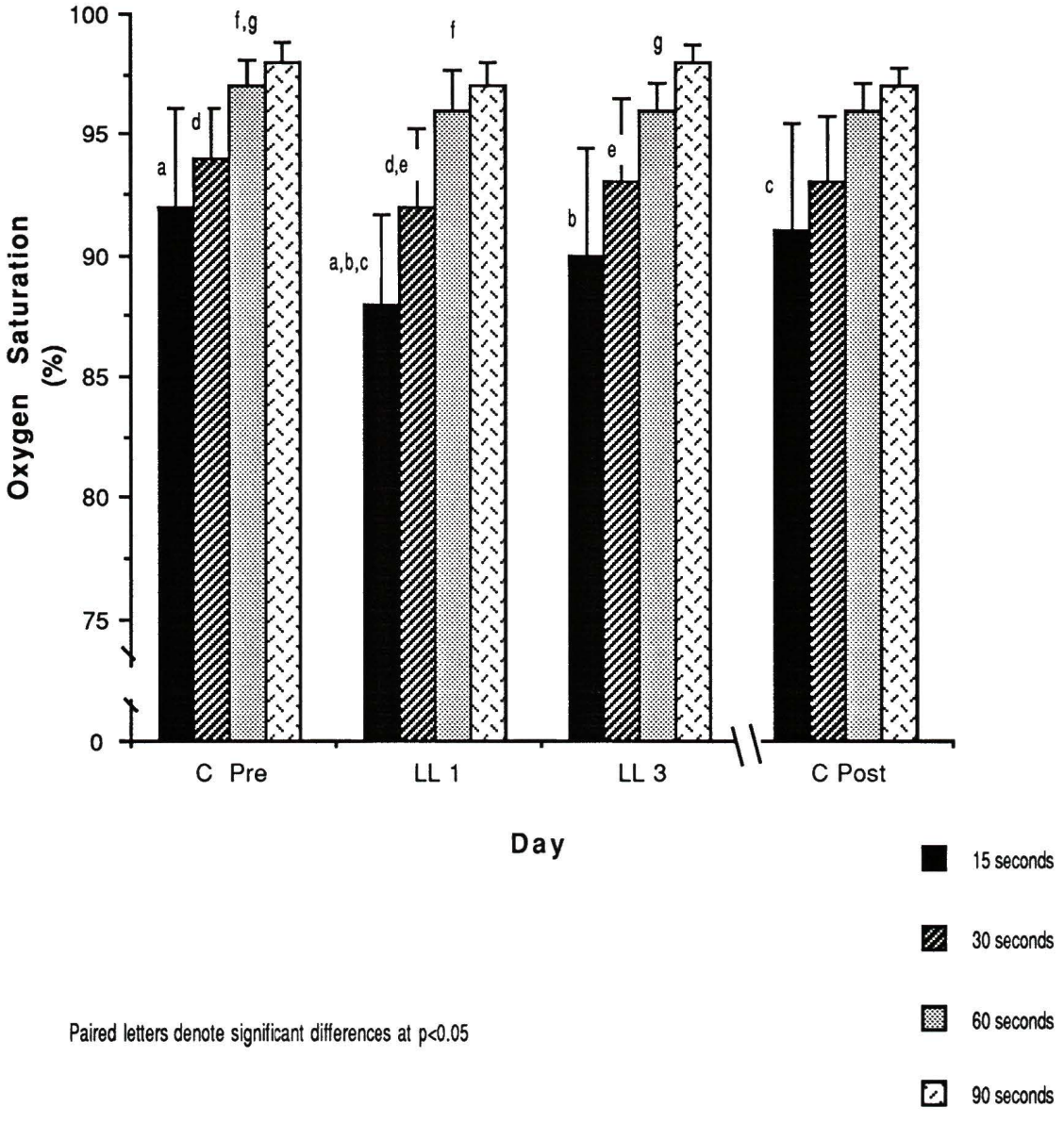
Table 4. Mean (SD) 60 ft and 120 ft Sprint and Drop-off times for elite male ice hockey players pre-altitude, at altitude and 5 weeks after altitude exposure.

Day	Calgary Pre	LL/Banff 1	LL/Banff 3	Calgary Post 5 wks
60 ft sprint (seconds)	2.99 (0.06)	3.04 (0.07)	3.02 (0.07)	2.97 (0.03)
60 ft drop-off (seconds)	0.25 (0.04)	0.22 (0.04)	0.24 (0.02)	0.19 (0.03)
120 ft sprint (seconds)	5.17 (0.43)	4.97 (0.81)	5.17 (0.45)	5.05 (0.03)
120 ft drop-off (seconds)	0.38 (0.06)	0.37 (0.07)	0.40 (0.10)	0.33 (0.04)

Table 5. Mean (SD) recovery heart rates 15, 30, 60 and 90 seconds after the completion of the repeated sprint test before (CalgaryPre), during (LL/Banff 1 and 3) and 5 weeks after (Calgary Post) altitude exposure.

Time	15 seconds	30 seconds	60 seconds	90 seconds
Calgary Pre	171 (2)	161 (3)	142 (4)	132 (4)
LL/Banff 1	170 (3)	159 (3)	140 (3)	125 (3)
LL/Banff 3	169 (2)	161 (2)	139 (3)	125 (4)
Calgary Post 5 weeks	169 (3)	163 (2)	144 (4)	132 (5)

FIGURE 3. Percent oxygen saturation of the blood 15, 30, 60 and 90 seconds after the repeated sprint test before (C Pre), during (LL1 and LL3) and 5 weeks after altitude exposure (C Post).



Paired letters denote significant differences at $p<0.05$

DISCUSSION

RESTING VARIABLES

Heart rate and SaO_2

Heart rate (HR) increased from 56 beats·min^{-1} at Calgary to 60 beats·min^{-1} on day 1 at altitude and decreased to pre-altitude values by day 2 and remained there on day 3 (Table 2). Increased resting HR is a common index of altitude stress and the results of this study concur with other studies conducted at higher altitudes which have found significant increases in resting HR at altitude (Hannon & Vogel, 1977; Huang et al., 1984; Richalet et al., 1988; Vogel, Hartley, Cruz and Hogan, 1974). HR increases at altitude due to increased Q in response to decreased PaO_2 (Brooks & Fahey, 1985, 472; Vogel & Harris, 1967). The decrease in PC shown with the hypoxic exposure (Table 2) would also act to increase HR since Q is the product of HR times stroke volume (SV) and SV is directly dependent on plasma volume (Alexander & Grover, 1983; Grover, Weil & Reeves, 1986). Other factors in this study may have also played a role in increasing resting HR on day one of altitude exposure. HR may have been elevated in anticipation as the morning blood sample was going to be taken right after resting variables were recorded. The new location and fatigue from travelling may also have increased HR.

The acute acclimatization process during which resting HR is elevated, usually lasts up to 2 weeks, after which it decreases back to pre-altitude values (McKardle, Katch & Katch, 1991, 543; Ratzin Jackson & Sharkey, 1988). The decrease in HR following acclimatization is due to a shift to increased O_2 extraction at the tissues which

allows the initial increase in Q via increased HR to subside (Grover, Weil & Reeves, 1986; Lenfant et al., 1968). A return of resting HR to pre-altitude levels can be used as an indicex of acclimatization state (Wenger, 1986). Resting HR returned to pre-altitude levels on day 2 at altitude suggesting that acclimatization to 1650m does not take as long as acclimatization to higher altitudes. The reduction in acclimatization time in this study compared to the studies of Asmussen and Consolazio (1941) and Reeves, Grover and Cohn (1967) which occurred at altitudes of 4300m and 3400m respectively may be because of the higher starting altitude (850m) and the smaller ascent (1650m).

Resting percent O_2 saturation (SaO_2) at altitude did not change significantly from pre-altitude values (Table 2). Terrados, Mizuno and Anderson (1985) tested 8 trained and 8 untrained subjects at 900m, 1200m and 1500m and found no differences in resting SaO_2 at the three altitudes. However, other studies conducted at higher altitudes by Vogel, Hartley, Cruz and Hogan (1974) and Vogel and Harris (1967) found that resting SaO_2 decreased from sea levels values of 97-99% to values of 92% at 3400m and 79% at 4600m thus showing that resting SaO_2 does decrease with decreasing PAO_2. These results suggest that the PO_2 at moderate altitude of 1650m was high enough to maintain resting SaO_2 at pre-altitude values.

Plasma Concentration, Hemoglobin and Hematocrit

During exposure to altitude, plasma concentration (PC) decreased continuously from a pre-altitude value of 55.1ml·100mL^{-1} to a low of 52.4 ml·100mL^{-1} on day 3 at altitude (Table 2.). This agrees with previous studies that showed that during acute exposure to altitude, PC decreases leading to hemoconcentration (Birchard & Tenneys, 1991; Hannon & Vogel, 1977; Klausen, Mohr, Ghisler & Nielsen, 1991; Stokke, Rootwelt, Wergeland & Vale, 1986; Wolfel, Groves & Brooks, 1991). The decreased

PC during acute altitude exposure increases the work done by the heart to maintain Q but does help to increase oxygen delivery to the tissues for a given volume of blood by increasing hematocrit (McKardle, Katch & Katch, 1991, 535).

Hemodilution has been proposed as a means for improving the aerobic performance of acclimatized subjects (Cerretelli, 1976). Winslow et al. (1985) found that hemodilution increased arterial and mixed venous PO_2, increased Q, improved V_E/flow match and subsequently increased anaerobic threshold. Theoretically, by decreasing the viscosity of the blood through maintainance of plasma volume, the heart would not have to work as hard and thus O_2 transport at altitude would be improved. However, other studies have shown that, while hemodilution decreased Hct and increased Q, VO_2 max and endurance time decreased as a result of the procedure (Boutellier, Deriaz, diPrampero and Cerretelli, 1990; Horstman, Weiskopf & Jackson, 1980).

Hemoglobin levels changed significantly from the pre-test values during the three day exposure to altitude (15.1g·100mL^{-1} pre-altitude to 15.7 g.·100ml^{-1} on day 3), and was still significantly elevated in the post-test five weeks later (15.8 g·100mL^{-1}). The immediate increase in Hb seen during the altitude exposure was most probably due to the decrease in PC as hemoglobin concentration is susceptible to different levels of hydration (Dill, Horvath, Dahms, Parker & Lynch, 1969). An accelerated release of erythrocytes from the bone marrow may also have helped to increase Hb (Klausen, Mohr, Ghisler & Nielsen, 1991).

The prolonged increase in Hb was expected as increases in Hb are usually seen after exposure to altitude in response to increased erythropoietin (EPO) production by the kidneys during hypoxic stress (Klausen et al., 1991). Erythropoietic stimulation of red cell production and consequently increased Hb levels occurs after approximately 4 to 7 days of altitude exposure. Klausen et al. (1991) studied erythropoietic responses in 7

elite skiers after training at moderate altitude (2700m). They found that EPO concentration increased on days 1-3 at altitude and that Hb values on days 2,4 and 7 post-altitude were all higher than pre-altitude levels.

The subjects in this study were exposed to moderate altitude for five days in Europe following the altitude testing session in Lake Louise and two weeks prior to the post-test session in Calgary in which the hemoglobin levels were significantly higher than pre-altitude test. This suggests that these two intermittent exposures to moderate altitude may have stimulated EPO production and thus helped the subjects maintain a higher hemoglobin concentration. Previous studies by Atland and Highman (1951) and Asahina, Ikai, Agawas and Kuroda (1966) have already demonstrated that intermittent exposure to high altitude is capable of increasing hemoglobin.

An increase in hematocrit (Hct) has been shown to be associated with the concurrent decrease in PC (Jung, Dill, Horton & Horvath, 1971). In this study Hct increased from 44% at the pre-test to 46% on day 3 at altitude to 47% at the post-test. This increase occurred concomitantly with the increase in Hb (Table 2). The immediate increase of both Hct and Hb concentration with acute altitude exposure increases the O_2 carrying capacity of a given volume of blood (Birchard & Tennys, 1991; Klausen et al., 1991).

The final post-test Hct reading of 47% was lower than expected considering the maintenance of hemoglobin concentration following altitude exposure. Cerretelli (1976) found that subjects acclimatized by repeated exposures to altitude up to 7000m over a period of two months had a lower Hct than subjects acclimatized by a continuous exposure to the same altitudes. This may explain the lower than expected value obtained in this study since the subjects did complete two short, intermittent exposures to altitude.

The lower overall increase in Hct with intermittent exposure may be advantageous. Stokke et al. (1986) stated that no evidence exists that a Hct above 45% provides any benefit in normoxic or hypoxic conditions. The increased viscosity of the blood leads to increased resistance to blood flow, increased work by the heart and increased fatigue (Haymes & Wells, 1986, 75) and thus eliminates any benefits of higher Hct. Therefore, intermittent exposures resulting in increased Hb and moderate increases in Hct may be the most beneficial way to acclimatize athletes to altitude prior to performance at that altitude.

White Blood Cells

In this study, total white cell count did not change significantly with an increase in elevation, but cell differentials did show fluctuating values (Figure 1.). The percent of the total white blood cell count made up by neutrophils decreased with altitude exposure and the percentage of lymphocytes increased with exposure. There is indirect evidence that hypoxia may impair the function of the immune system (Biselli et al, 1991; Chohan, Singh, Balkrishan, and Talwar, 1975; Meehan et al., 1988), but data has been limited to lymphocyte response. T cells were found to have impaired function at extreme altitude, but B cell function was normal (Meehan et al., 1988).

The increase in the percentage of lymphocytes with acute hypoxic exposure may be due to increased production of B cells. Increased rates of immunolglobulin synthesis have been shown with acute altitude exposure by increased levels of IgG and IgM (Chohan et al., 1975; Meehan et al., 1988). Increased levels in serum IgM have been found during acute submaximal exercise as well. The suggested mechanism for increased antigen stimulation during exercise was through greater-than-normal quantities of microorganisms entering the body through both increased ventilation rates and

breakdown of natural mucosal immunity by drying of airway secretions (Nieman & Nehlsen-Cannarella, 1991). Since altitude exposure is also associated with increased V_E at rest and during exercise and with decreased atmospheric humidity, this mechanism may also explain why lymphocytes increased at altitude.

SUBMAXIMAL EXERCISE

Heart rate and SaO_2

Most studies have found that during submaximal exercise in acute hypoxic conditions HR increases for a given absolute workload. This was due to an increase in sympathetic activity in response to the decrease in PaO_2 (Schibye, Klausen, Trap-Jensen, Lund & Hartling, 1988; Vogel & Harris, 1967; Wolfel et al., 1991). However, in this study, submaximal HR at altitude was the same as the pre-altitude value (Table 3). Several other studies have also found that ascent altitude did not cause an increase submaximal HR (Alexander, Hartley, Modelski and Grover, 1967; Hartley, Vogel and Cruz,1974; Noble & Maresh, 1979; Squires & Buskirk, 1982). One explanation for this finding is the effect of individual variability of response to hypoxia on the group mean (Alexander et al., 1967; Hartley et al., 1974). The altitude ascended to in this study may not have been high enough to influence submaximal HR. Noble and Maresh (1979) found no differences in submaximal HR in college basketball players exercising at 1000m and 2200m. Squires and Buskirk (1982) also found that submaximal HR did not change at altitudes ranging from 914m to 2286m. Thus, the maintainance submaximal HR in this study may be due to individual variability of

hypoxic response or because the hypoxic stress was not great enough to increase submaximal HR for a given load compared to low altitude submaximal HR.

SaO_2 during submaximal exercise decreased significantly from 94±3% pre-altitude to 91±2% on days 1 and 2 at altitude and then increased to 95±2% on day 3 at altitude (Table 3). During exercise, a ventilation-perfusion mismatch and diffusion limitation occur and are worsened at altitude (Houston, Sutton, Cymerman & Reeves, 1987; Welch, 1987). A decrease in atmospheric PO_2 acts to decrease PAO_2 and PaO_2. The reduction in driving pressure of oxygen into the blood results in a lower SaO_2 during exercise.

SaO_2 may have also been reduced at altitude due to a shift in the oxyhemoglobin dissociation curve down and to the right as a result of decreased blood and tissue pH. The shift of the curve down and to the right would decrease the affinity of hemoglobin for oxygen resulting in a decreased oxygen loading at a given PO_2 and a decrease in the percent oxygen saturation measured.

The decrease in SaO_2 with exercise at altitude is a common phenomenon, but the values obtained were slightly lower than were expected for this altitude. This could be explained by the fitness level of the subjects used in the study. The average VO_2 max of the subject population was 56.8 $mL \cdot kg^{-1} \cdot min^{-1}$, which indicates a high level of aerobic fitness. Terrados, Mizuno and Anderson (1985) found that during submaximal and maximal exercise, their more highly trained group of subjects had lower SaO_2 values than their untrained counterparts. This suggests that even at submaximal efforts, hypoxia may be more detrimental for highly fit athletes than for less fit athletes.

SaO_2 is determined by the oxygen binding characteristics of Hb (as defined by the Hb-O_2 dissociation curve), the PAO_2-PaO_2 diffusion gradient and the O_2 tension of arterial blood (PaO_2). With ventilatory acclimatization to hypoxia, PaO_2 and SaO_2 also

increase (Grover, Weil & Reeves, 1986). In both the submaximal test and after the on-ice tests, SaO_2 increased back to pre-altitude levels on day 3, lagging slightly behind the decrease in V_E (Table 3). Again, these results also suggest that some early acclimatization occurred by the second and third day of exposure to 1650m.

V_E and VO_2

On day 1 at altitude, V_E was significantly higher than pre-test values and subsequent altitude values. V_E increased from a pre-altitude value of 109.1±16.8 $L \cdot min^{-1}$ to 118.4±17.1 $L \cdot min^{-1}$ on day 1 and then decreased on days 2 and 3 to 109.9±15.3 and 110.3±16.0 $L \cdot min^{-1}$ respectively (Table 3). An increase in resting and exercise V_E is the most fundamental functional change that characterizes the initial stages of acute altitude acclimitization (Hannon & Vogel, 1977; McKardle, Katch & Katch, 1991, 535). Previous studies have found that submaximal V_E is elevated for several days upon ascent to altitude (Burki, 1984; Huang et al., 1984)), whereas in this study, on day 2 at altitude, V_E values had decreased to pre-altitude levels indicating that some acclimatization had already occurred. The most probable reason for the difference in the duration of the elevated V_E are the differences in elevations at which the different studies were conducted. Because the subjects of this study only increased 800m in elevation and they already live 850m above sea level, acclimatization to the new altitude may not have taken as long because the athletes may have been 'pre-acclimitized' to moderate altitude by their place of residence (Noble & Maresh, 1979). A decrease in V_E back to pre-altitude levels is one of the first indicators of altitude acclimatization (Wenger, 1986). The decrease in V_E back to pre-altitude levels on day 2 corresponds with the decrease in resting HR back to to pre-altitude levels on day 2 and

thus provides further evidence that some acclimatization may have occurred within 2 days at 1650m.

Noble and Maresh (1979) stated that submaximal VO_2 has not been observed to change in sea level residents even up to 6700m. However, in this study, submaximal VO_2 values followed the same trend as V_E, increasing on day 1 at altitude to 3.81±0.28 $L \cdot min^{-1}$ and then decreasing back to near pre-altitude values on days 2 (3.71±0.2 $L \cdot min^{-1}$) and 3 (3.65±0.24 $L \cdot min^{-1}$) (Table 3). The oxygen cost of the submaximal exercise at altitude should have been virtually identical to pre-altitude cost (McKardle, Katch & Katch, 1991, 533; Squires & Buskirk, 1982). The increase in VO_2 on day 1 at altitude in this study may have been due to decreased efficiency on the bike because of fatigue from travelling the day before or the strenuous on-ice workouts. An increase in muscle lactate accumulation may have decreased the rate of glycolysis during exercise and thus may have increased the amount of energy that had to be supplied by the aerobic system, resulting in an increase in VO_2. The O_2 dissociation from hemoglobin at the tissues may have been increased because of decreased muscle pH and its effect on the oxyhemoglobin dissociation curve.

Other physiological responses such as decreased V_E, decreased plasma volume, increased Hct and increased submaximal lactate values on day 2 and 3 suggest that the acute acclimatization process was just about complete. The change back to pre-altitude levels in these variables may have reduced submaximal VO_2 back to the pre-altitude value due to the re-establishment of acid-base balance in the blood and muscles.

Lactate

The changes in blood lactate concentration observed with acute hypoxic exposure are influenced by the elevation and metabolic rate (Young et al., 1982). There are

conflicting results on the effect of hypoxia on blood lactate during exercise. Some studies have shown blood lactate levels to be higher in acute hypoxia for a given workload (Gonzales, Sokari & Claney, 1991; Lee, Cordain, Sockler & Tucker, 1990) and other studies have found a decreased lactate accumulation with submaximal and maximal exercise at altitude (Dill & Adams, 1971; Klausen, 1966; Yamamoto, Takei, Mutch & Myashita, 1988). In this study, blood lactate concentrations decreased from pre-altitude levels of 5.7 $mmol \cdot L^{-1}$ to 4.7 $mmol \cdot L^{-1}$ on day 2 at altitude and then increased back to pre-altitude levels on day 3 (Table 3). These results provide a good example of the lactate paradox.

Lactate production at altitude is increased for a given work intensity because the body is more dependent on glycolysis because of the reduced aerobic capacity. Mazzeo et al. (1991) studied arterial catecholamine responses during exercise with acute and chronic exposure to 4300m and found increased epinephrine concentrations on acute exposure that decreased with acclimatization. They related the epinephrine levels to circulating lactate concentrations ($r=0.95$) and demonstrated a strong association between exercise blood lactate concentrations and lactate turnover with the epinephrine response. Altitude increases epinephrine concentration and therefore increases lactate concentration. With improved oxygenation, which accompanies ventilatory acclimatization and erythropoeisis, epinephrine secretion subsides. A blunted response in blood lactate may indicate acclimatization and is closely coupled with the decreased epinephrine (Mazzeo et al, 1991). Young et al. (1982) also found that blood lactate levels decreased after acclimatization.

Blood lactate concentration is dependent upon the relationship between lactate production and release and lactate removal and catabolism. Changes in extracellular acid-base states during exercise influence the relationship between lactate turnover and

clearance to altered blood lactate concentrations (Davies, Iber, Keene, McArthur & Path, 1986). The hydrogen ion from lactic acid is almost entirely buffered by HCO_3^- (Beaver, Wasserman & Whipp, 1986). Decreased blood lactate concentration at altitude during submaximal exercise may be due to a loss of buffering capacity since HCO_3^- is excreted during the first 2 days at altitude, which decreases lactate and hydrogen ion efflux (Haymes & Wells, 1986, 77). Thus, the loss of buffering capacity in the blood caused by a hypercapnia-induced extracellular acidosis tends to prevent or slow down the efflux of intracellular lactate into the extracellular fluids (Shepard, Bouhlel, Vandewalle & Monod, 1988; Yamamoto et al., 1987).

At altitude, the subjects cycled at the same workload as the pre-altitude and post-altitude tests, therefore the energy cost of the exercise should have been the same. Thus, lactate production by the muscles must have been the same, therefore, it must have been a reduction in the amount of lactate released by the muscles caused by decreased blood bicarbonate concentration that led to the decreased blood lactate concentrations.

On day 3 at altitude, blood lactate values had returned to the pre-altitude value of 5.7 $mmol \cdot L^{-1}$. This suggests that some acclimatization had occurred and acid-base balances in the blood had been restored so that the blood buffering system was able to clear the lactate produced in the muscles during submaximal work.

ON-ICE PERFORMANCE

Agility Tests

The pre and post-sprint agility tests were significantly slower on day 1 at altitude compared to the Calgary tests (Figure 2). At higher altitudes (2700-3700m) it has

been found that both mental efficiency and the performance of discrete motor movements may decrease as a result of altitude effects such as drowsiness and fatigue (Ward, 1987). Thus, the increase in time to complete the agility test on day 1 may have been due to increased fatigue and decreased motor co-ordination.

The post-sprint test on day 3 was significantly faster than the pre-sprint tests in Calgary and on day 1 at altitude (Figure 2). The effect of acclimatization as shown by the submaximal test variables previously discussed also appear to be reflected in the decreased sprint times. Although not significantly different, the pre-sprint test on day 3 was slightly faster than day 1, but still slower than the Calgary test. These results suggest that hypoxia and subsequent acclimatization effects were more apparent after fatigue and during recovery. A learning effect may also have played a role in the decrease in the times for the agility test on day 3 at altitude and may have been more apparent in the fatigued condition.

Sprint and Drop-off Tests

The increase in altitude and exposure to altitude over three days had no significant effect on 60 ft and 120 ft sprint times (Table 4). These results concur with studies which have shown that altitude does not affect anaerobic sprint performance (Haymes & Wells, 1986, 69). Since oxygen supply is decreased in hypoxic conditions, more energy may have been produced by the catabolism of glycogen to lactate which may have offset the reduced O_2 availability and thus maintained performance comparable to the pre-altitude performance (McLellan, Kavangh & Jacobs, 1990)

No significant differences were found in the drop-off test performed at 1450m altitude. Recovery between high intensity work bouts is primarily an aerobic event (Green, 1979) and given that hypoxia affects aerobic metabolism and increases fatigue,

it was expected that the drop-off times would have increased with altitude exposure. The type of training that the subjects do may explain why the expected increased drop-off time was not seen with the altitude exposure. Saltin (1967) suggested that differences in anaerobic capacity influence performance at altitude. Because VO_2 max is reduced at altitude, a person working at the same intensity at altitude as at sea level would have to use more anaerobic energy, thus the athletes with the largest anaerobic capacities have an advantage at altitude. Jette (1980) indicated that anaerobic power is of primary importance to hockey players and thus anaerobic power and capacity training is an important part of their training. Because the subjects are all elite hockey players, their anaerobic capacities were well developed and thus they were more capable of handling the sprint work at moderate altitude without a decrement in their repeated sprint times. Longer sprints and/or more repetitions may have exceeded their anaerobic capacity and reflected a decrement due to the altitude exposure.

Heart Rate and SaO_2

No significant difference in recovery heart rate 15, 30, 60 or 90 seconds after the repeated-sprints was found between the pre-test session in Calgary and any of the altitude sessions in Banff (Table 5). It was expected that the recovery heart rates would have been higher at altitude, especially in light of the decrease in SaO_2 during recovery at altitude. However, during the sprints there was the same amount of work being done at altitude as during the pre-altitude test so the same mechanical feedforward mechanisms would have been regulating HR and may have overridden the sympathetic activity that would have been trying to increase HR in response to the increased stress of hypoxia. Ahn et al. (1989) studied HR response to breath holding during supramaximal exercise and found that the breath holding resulted in hypoxia and

hypertension. They found a depressed HR response during recovery after the supramaximal exercise and attributed it mainly to the baroreceptor activity due to elevated blood pressure.

SaO_2 was 2-3% lower at 15, 30 and 60 seconds after the repeated-sprint test on day 1 at altitude compared to the pre-altitude test (Figure 3). SaO_2 after the repeated-sprints on ice was lower than after the submaximal bike test even though the on-ice testing took place at a lower elevation. SaO_2 dropped from 91% in the pre altitude test to 88% on day 1 at altitude and then increased to 90 and 91% on days 2 and 3 respectively (Figure 3). In spite of the on-ice tests being conducted at an altitude 200m less than the submaximal tests there was a greater reduction in SaO_2 after the on-ice sprints. The greater decrease in SaO_2 after the on-ice tests was probably due to the higher intensity exercise in which the subjects were engaged (HR= 171 beats·min^{-1} after on-ice exercise versus 163 beats·min^{-1} after submaximal exercise). Higher intensity exercise decreases transit time of the blood through the lungs and thus decreases the time for oxygen to bind to hemoglobin (Dempsey, 1986; Wenger, 1986). The decreased PO_2 driving pressure and the decreased transit time of the blood through the lungs resulting in a decreased loading of oxygen and a subsequent drop in SaO_2 (Wenger, 1986).

In spite of the other acclimatization effects, the 60 second SaO_2 value remained significantly lower on day 3 at altitude and did not follow the same pattern of returning to pre-altitude levels as the other variables such as V_E, VO_2 and lactate (Figure 3). There were no significant changes in the 90 second SaO_2 on any of the days. These results, along with the increased Hct and decreased PV values and submaximal test results, suggest that from day 1 to 3, there was an improvement in the oxygen carrying capacity of the blood and pulmonary diffusion gradient that should have allowed an

improvement in the initial stages of recovery from high intensity exercise. However, even with the enhanced circulatory adaptations, the total recovery of SaO_2 at altitude took between 60 to 90 seconds compared to 30 to 60 seconds in Calgary.

IMPLICATIONS FOR ICE-HOCKEY PERFORMANCE AT ALTITUDE

Hockey is a sport characterized by a series of short, high intensity bursts of activity that last from a few seconds to a maximum of 10-12 seconds, followed by periods of moderate activity during on-ice shifts of 60-88 seconds (Green et al., 1976; Jette, 1980). Due to the intermittent nature of the game, both the aerobic and anaerobic systems are involved in the metabolic process of energy production (Green, 1979; Green & Houston, 1975). On a regular shift, a player spends approximately 20% of the time in high intensity (anaerobic) work and the rest at moderate aerobic work intensities (Rhodes & Twist), however, approximately 70% of energy expenditure by an ice hockey player during a game is anaerobic (Jette, 1980). Opportunities for recovery from the anaerobic work occur when a player has less direct involvement in a play, during play stoppage, between shifts and between periods (Watson & Hanley, 1986).

Events such as hockey that are a combination of aerobic and anaerobic exercise have received little research attention (Ratzin Jackson & Sharkey, 1988). Certain anaerobic performances are not affected by altitude according to this study and the research by Saltin (1967). Thus, most of the effects of altitude on an intermittent type of sport such as hockey appear to be primarily on the aerobic system which is vital to recovery from the high intensity anaerobic phases of the sport.

With repeated anaerobic sprint-type activity, such as would occur during a hockey game, there is an increase in recovery time required at altitude to remove lactate and replenish ATP-CP stores because acute altitude exposure decreases SaO_2, HCO_3^- and PC (Haymes & Wells, 1988, 75). The increased excretion of HCO_3^- as a result of acute exposure to altitude decreases the total capacity of the blood to buffer H^+ and thus impairs the efflux of lactate from the working muscle into the blood (Shepard et al., 1988; Yamamoto et al., 1987). McLellan, Jacobs and Lewis (1988) showed that increasing blood HCO_3^- through soda loading increased blood lactate at a given workload. Since the soda loading increased the efflux of muscle lactate into the blood, it thus seems logical that a decrease in HCO_3^- will decrease lactate efflux from the muscle into the blood. Due to this decreased efflux, a longer recovery time would be necessary to remove the lactate produced in the muscles during high intensity exercise.

This study showed that a longer time was required to bring SaO_2 back to pre-altitude post-sprint levels at 1450m altitude compared to 850m even after only 5 repeated sprints of high intensity and moderate intensity work. An even longer recovery time would be required to get SaO_2 back to resting levels. Thus, the results again imply that when playing a complete hockey game at altitude, longer recovery times or decreased work times than at low altitude are required by the players in order to perform at the same intensity as low altitude.

O_2 supplementation during recovery has been used as an ergogenic aid during intermittent breaks in sports such as soccer and football. Some athletes may believe that supplementation may enhance their recovery from exercise at altitude. However, several studies have shown, using O_2 concentrations of 40-100% that use of supplemental O_2 during recovery does not offer an advantage during recovery from exhaustive exercise or on subsequent performance at sea level (Titlow, 1982; Winter,

Snell, & Stray-Gundersen, 1989). D'urzo, Liu and Rebuck (1986) found that supplemental oxygen sufficient to maintain SaO_2 during progressive exercise to exhaustion in moderate hypoxic conditions did not maintain VO_2 or HR at sea level values. Thus, according to these studies O_2 supplementation may not, physiologically, enhance recovery or performance during aerobic exercise at sea level or at altitude. However, the effectiveness of O_2 supplementation during intermittent activity has not been adequately researched and requires further investigation. O_2 supplementation during any type of activity may have psychological benefits if the athlete believes O_2 supplementation will help recovery and subsequent performance (Titlow, 1982).

When athletes train at moderate altitude, their ability to perform at this altitude is enhanced (Ratzin-Jackson & Sharkey, 1988). Recommended acclimatization time is 14 days for 2000m and 28 days for 2500m (Ratzin and Sharkey, 1988). However, a decrement in low altitude performance has been shown after altitude training because the intensity of training cannot be maintained at altitude (Buskirk, E.R., Kollias, J., Akers, R.F., Prokop, E.K., & Picon-Reatigue, E.P., 1967; Grover & Reeves, 1967). An intermittent exposure schedule may provide the benefits of altitude acclimatization without the decrements in performance. Daniels and Oldridge (1970) found that when 2 week training periods at 2,300m were separated by a few days of training at sea level, post-altitude VO_2 max improved by 5%. Furthermore, they suggested that the intermittent protocol did not interfere with altitude acclimatization. The post-test results of this study showed that the intermittent exposures to altitude may have helped the subjects maintain an increased Hct and Hb, an index of altitude acclimatization. However, the subjects were not tested again at altitude, so it could not be determined for certain if the intermittent altitude exposures helped the subjects maintain their

acclimatized state at altitude. Further research needs to be done to determine if intermittent protocols will allow acclimatization to occur at various altitudes.

CONCLUSIONS

What are the selected physiological responses at rest at a low altitude of 850m and upon ascent to a moderate altitude of 1650m

Selected physiological responses at rest when changing from low altitude to high altitude are increased resting HR and a shift in white cell differentials. PC decreased slightly and Hct showed a corresponding increase. Hb, SaO_2 and total white blood cell counts did not change.

Are there differences in these response after 2 or 3 days at altitude?

After 2 days of altitude exposure, acclimatization had begun as shown by a decrease in resting HR back to pre-altitude levels. There was a further significant decrease in PC and increase in Hct and Hb by day 3 which would increase the O_2 carrying capacity of a given volume of blood. White cell differentials were significantly different on day 3 compared to pre-altitude. Resting SaO_2 and total white blood cell counts did not change over the second and third day of exposure to altitude.

What are the selected physiological responses during submaximal exercise at a low altitude of 850m and upon ascent to a moderate altitude of 1650m?

There was a significant change in the submaximal exercise response of elite hockey players to a change in altitude as shown by an increase in V_E and VO_2 and a decrease in SaO_2. These changes are functional characteristics of the first stage of acute altitude acclimatization. Submaximal HR and blood lactate were not significantly different from pre-altitude values.

Are there differences in these responses after 2 or 3 days of exposure to moderate altitude?

Physiological responses during submaximal exercise on day 2 such as the decrease in V_E and VO_2 back to pre-altitude values provide further evidence that significatant acclimatization to 1650m occurred after 2 days of altitude exposure. SaO_2 remained significantly lower on day 2 than pre-altitude levels and blood lactate concentration was significantly less than pre-altitude concentrations. SaO_2 and blood lactate did not increase back to pre-altitude values until day 3 suggesting that blood acid-base balances lagged behind ventilatory acclimatization.

Do the physiological changes reflect a difference in performance as measured by on-ice speed, agility and drop-off times at 1450m?

There did appear to be a related decrease in on-ice performance at 1450m as shown by an increase in pre and post-sprint agility times on day 1 at altitude and by the increase in recovery time for SaO_2 after the repeated sprint test. There was no performance decrement in the pre and post-sprint agility tests on day 3 at altitude, nor in the 60 and 120 ft sprint and drop-off times. The results suggest that the primary decrement to on-ice performance was aerobic in nature and the anaerobic performance of the repeated sprint test was not affected except in recovery.

What are the possible implications in performance at moderate altitude for elite ice hockey players?

Possible implications for performance at 1450m altitude 1 day after arrival are decreased anaerobic and aerobic performance over repeated shifts if recovery time is not extended to remove lactate and increase SaO_2 back to resting levels. After 2 days of altitude exposure, the athletes seemed to have acclimatized to the increase in elevation, but extended recovery time between shifts should still be incorporated to allow full recovery of the aerobic and anaerobic systems. O_2 supplementation may aid in recovery from intermittent exercise at altitude and may help enhance subsequent performance. Intermittent exposures to altitude in preparation for performance at altitude may allow for successful acclimatization without causing the decrements in performance associated with longer stays at altitude.

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APPENDIX A

Definitions, Limitations and Assumptions

DEFINITION OF TERMS

Low altitude: an altitude of 0-1200m where there is very limited physiological response to an increase in elevation.

Moderate altitude: an altitude of 1200-3040m where there are physiological responses to the decrease in PO_2.

Submaximal exercise: an exercise load of 85-90% VO_2 max. Highly trained althletes appear to avoid lactate accumulation until they reach higher intensities of exercise (MacDougall, Wenger & Green, 1991, 110).

Trained subjects: subjects with a VO_2 of greater than $50mL \cdot kg^{-1} \cdot min^{-1}$ and are used to maximal exertion.

Acute hypoxia: a state in which the oxygen pressure and/or concentration in arterial blood is lower than its normal value at sea level and will refer only to that form of hypoxia resulting from low environmental PO_2 (Welch, 1987).

Acclimatization: the physiological adjustments which occur in response to stimuli which occur naturally in the environment (Hoar, 1975).

Delimitations

1. The results will describe physiological changes in response to acute moderate altitude for trained males between the ages of 18-30 years. No generalization will be made to younger or older individuals or to females.

2. The results will describe physiological changes during three days of exposure to moderate altitude. The conclusions will not be generalized to more days of exposure.

Limitations

1. A small sample size (n=17) of a specific population.

Assumptions

1. Level of intensity of on-ice workouts while at altitude cannot be stringently controlled. It is assumed that subjects will be adequately rested for each testing session.

2. Subjects will be given instructions for rest, dietary and hydration guidelines while at altitude and before each testing session. It is assumed that subjects will be adequately hydrated and rested for each testing session.

3. Any changes in the variables measured will represent an effect of altitude versus an error in measurement or a change in protocol.

APPENDIX B

Review of the Literature

I. INTRODUCTION

Many major sporting events, including the 1968 Summer Olympics at Mexico City (2200m) and the 1992 Winter Olympics at Albertville, France (1500m), are at altitudes where hypoxia can affect performance. Knowledge of performance at altitude is largely based on research done at high (above 3048m) altitude. However, it is the moderate altitude elevations (1400m - 3000m) at which most athletes train or compete (Ratzin Jackson & Sharkey, 1988). The impact of this altitude range on physiological and performance variables has not been well investigated.

It is accepted that a threshold altitude must be reached before the effects of hypoxia are evident, but this threshold is equivocal (Banchero, 1987). Studies done at moderate altitude, where the threshold altitude most probably exists, have produced conflicting results. Therefore, the purpose of this section is to review the literature on the effect of hypoxia on human physiology, compare the effects of different altitudes on selected physiological responses and to discuss how the physiological responses to altitude affect performance.

II. ACUTE HYPOXIA: selected physiological responses at rest

As altitude increases barometric pressure decreases resulting in a decrease in the partial pressure of oxygen (PO_2) which causes the alveolar oxygen tension and arterial oxygen saturation to fall (Smith & Sharkey, 1984). This results in less oxygen (O_2) transfer from the environment to the blood as the diffusion of oxygen across the alveolar

sacs is directly dependent on the PO_2 gradient between the lungs and the blood. Thus, at altitude, less oxygen will be delivered to the tissues of the body. In response to this, physiological changes occur within the first 4 hours of exposure to hypoxia (Welch, 1987) and will continue for many months if the exposure persists. The initial stage of altitude acclimatization is characterized by a variety of functional changes which collectively facilitate O_2 transport to maintain tissue PO_2 and oxygen consumption (VO_2) (Buskirk, Kollias & Picon-Reategui, 1966).

Ventilatory effects

An increase in ventilation (V_E) with hypoxic exposures has been observed by several researchers (Burki, 1984; Hannon & Vogel, 1977; Huang et al., 1984) and is considered the most fundamental change that characterizes the initial stages of acute acclimatization (Hannon & Vogel, 1977; McKardle, Katch & Katch, 1991, 535). The purpose of a hypoxic V_E response (HVR) is to increase alveolar O_2 concentration (Schoene et al., 1984) and has been attributed to an increase in respiratory frequency (15.6 $\pm$ 3.5 breaths/min at sea level to 23.8 $\pm$ 6.2 breaths/min at 3940m) due to a significant decrease in both inspiratory and expiratory time per breath (Burki, 1984). The HVR is mediated by peripheral chemosensory input and is dependent on the individual hypoxic chemosensitivity (Schoene et al., 1984). A high ventilatory response may be advantageous for performance because it tends to minimize the drop in arterial oxygenation. Hyperventilation would produce a left shift in the O_2-hemoglobin dissociation curve, an increase in arterial O_2 partial pressure and a subsequently higher % SaO_2 (Schoene et al., 1984).

When V_E increases upon ascent to altitude, PCO_2 of the blood decreases resulting in a decrease in H^+ and a corresponding increase in pH (Hansen, Stelter & Vogel, 1967).

This initial respiratory alkalosis causes the kidneys to increase renal bicarbonate excretion (Brooks & Fahey, 1985, 474). Bicarbonate (HCO_3^-) acts to stabilize pH, since the acid form (carbonic acid) continually disappears due to the ventilatory expiration of CO_2 (Beaver, Wasserman & Whipp, 1986). HCO_3^- is the primary buffer of lactic acid (Beaver et al., 1986), thus during initial acute exposure to hypoxia, the efflux of muscle lactate into the blood is decreased (Haymes & Well, 1986, 77). With acclimatization, acid-base balance of the blood is restored and V_E decreases. The return to pre-altitude levels of V_E could be used as an index of the state of acclimatization (Wenger, 1986). With V_E acclimatization, arterial oxygen pressure (PaO_2) and arterial oxygen saturation (SaO_2) also increase (Jung, Dill, Horton & Horvath, 1971).

Plasma Concentration, Hematocrit and Hemoglobin

The next stage of acclimatization includes a decrease in plasma concentration (PC) and an increase in erythrocyte production which both increase the O_2 carrying capacity of the blood (Birchard & Tennys, 1991; Hannon & Vogel, 1977; Klausen, Mohr, Ghisler & Nielsen, 1991). An increase in Hct is associated with the concurrent decrease in PC (Jung, Dill, Horton & Horvath, 1971) and begins on the day of arrival at altitude and rises progressively during initial days of acclimatization (Jung et al., 1971). A decrease in PC has a positive effect on increasing the O_2 content of a given volume of blood, however, it has a negative effect on blood flow due to an increase in viscosity. This leads to an increased resistance to blood flow and increased work by the heart (Haymes & Well, 1986, 75).

Little is known regarding the mechanism of the initial hemoconcentration at high altitude. Potential mechanisms include gross dehydration, negative nitrogen balance or a shift of fluid from the intravascular to the extravascular space (Weil, Battock, Grover

& Chidsey, 1969). Increased capillary pressure might also be responsible for the initial fall in plasma concentration(Weil et al., 1969).

The level to which plasma concentration decreases and hematocrit (Hct) increases appears to be affected by the severity of the hypoxia. Boutellier, Deriaz, diPrampero and Cerretelli (1990) studied six subjects on a mountaineering expedition and found that after 5 weeks at 5200m altitude, Hct was 23% and hemoglobin (Hb) was 20.5% higher than at sea level. At lower altitudes of 2300-2700m, the magnitude of increase in Hct and Hb levels is less (Dill, Braithwaite, Adams & Bernauer, 1974; Klausen, Mohr, Ghisler & Nielsen, 1991). Twelve well conditioned men trained for 3 weeks at 2300m and their Hct increased only 3.7% and plasma concentration decreased 6.6% (Dill et al., 1974).

The increase in erythrocyte production observed upon ascent to altitude is due to an increased concentration of erythropoetin (EPO) which stimulates the bone marrow (Brooks & Fahey, 1985, 480). Increased erythropoiesis starts almost immediately upon arrival at altitude and also appears to be influenced by the degree of severity of hypoxia. Eckardt et al. (1989) exposed six subjects to 3000m and 4000m simulated altitude for 5.5 hours. The EPO levels were significantly raised after 114 minutes at 3000m (16.0 to 22.5 μU/ml) and this response was even greater after 84 minutes at 4000m (16.7 to 28.0 μU/ml). Increases in Hb are dependent on increased erythrocyte production and lags behind the acute changes in PV and Hct. Erythropoietic stimulation of erythrocyte production and consequently increased Hb levels occur after approximately 4-7 days of exposure to hypoxia (Klausen et al., 1991).

Heart Rate and SaO_2

Upon ascent to altitude there is an increase in cardiac output (Q) due to an increase in heart rate (Brooks & Fahey, 1985, 472; Vogel & Harris, 1967). Studies performed at high altitudes (Huang et al.,1984; Richalet et al., 1988; Vogel, Hartley, Cruz and Hogan, 1974) have found significant increases in resting HR at altitude. The acute acclimatization process during which resting HR is elevated usually lasts about 2 weeks (McKardle et al., 1991, 543; Ratzin-Jackson & Sharkey, 1988). However, a sustained increase in HR in response to hypoxic stress would be costly in terms of cardiac work and energy expenditure. Therefore, rather than increasing blood flow indefinitely, there is a shift to increasing O_2 extraction at the tissues for any given capillary PO_2, thus less blood flow is required to maintain the same VO_2. The increase in O_2 extraction is accomplished by the decrease in the affinity of hemoglobin for oxygen as a result of the gradual increase in the concentration of 2,3-diphosphoglycerate in the red blood cells (Brooks & Fahey, 1985, 472; Lenfant et al., 1968). Thus, the initial increase in Q via increased HR subsides (Grover, Weil and Reeves, 1986). The return of HR to resting sea level values may also be used as an index of acclimatization state (Wenger, 1986).

As altitude increases, SaO_2 decreases proportionally to PaO_2 (Lenfant et al., 1968). This is because at altitude the affinity of hemoglobin for oxygen is reduced to make hemoglobin-bound oxygen more available to body tissues. In response to hypoxia, the shift of the O_2-Hb dissociation curve occurs within 24 hours thus making O_2 available to the tissues at any given O_2 tension. Resting SaO_2 is dependent on the degree of hypoxic exposure. Studies done at higher altitude found that resting SaO_2 decreased from sea-level values of 97%-99% to values of 92% and 79% at 3400m and 4600m respectively (Vogel et al., 1974; Vogel & Harris, 1967). However, Terrados, Mizuno

and Anderson (1985) found no differences in resting SaO_2 in 8 trained and 8 untrained subjects at 900m, 1200m and 1500m when compared to sea level SaO_2.

Immune Response

There has been few data published on the effects of hypoxic exposure on the the immune system response. Epidemological data from several studies provide indirect evidence that hypoxia may impair the human immune response (Bisell et al., 1991; Meehan et al., 1988). Chohan, Singh, Balakrishnan and Talwar (1975) found a quantitative change in immunoglobulin synthesis. With acute exposure, when the hypoxic stress was greatest, immunoglobulin synthesis was also at its peak.

Research that looks at the effect of hypoxia on the immune system has focused primarily on the changes in specific immune response systems, the lymphocyte T and B cells. Meehan et al. (1988) and Chohan et al. (1975) found increases in levels of IgG, IgM with acute hypoxic exposure, but impaired T-cell function. It is the B-cells that produce the immunoglobulins IgM, IgG, IgA so the increase in the level of these immunoglobins suggests that B-cell production and activity is increased in hypoxia.

II. EXERCISE AT ALTITUDE

Maximal Exercise

VO_2 max and SaO_2

Exercise by unacclimatized humans exposed to hypoxia reveals impaired aerobic performance as characterized by reduced aerobic power (VO_2 max)(Bouissou, Peronnet, Brisson, Helie & Ledoux, 1986; Boutellier et al., 1990; Dill & Adams, 1971; Fulco,

Rock, Trad, Forte and Cymerman, 1988; Squires & Buskirk, 1982; Torre-Beuno, 1985). Arterial hypoxemia and desaturation during heavy exercise is primarily due to diffusion limitations caused by a decrease in pulmonary transit time (PTT) in athletes possessing an extremely high Q max (Willimas, Powers & Stuart, 1986). Thus, the major limiting factor of maximal performance at altitude is the decrease in the partial pressure of O_2 in the air and the subsequent limitation on pulmonary diffusion. The decrease in exercise SaO_2 has been attributed by Houston et al. (1987) to a ventilation-perfusion mismatch and also to a decrease in PTT (Dempsey et al, 1984).

At rest, blood takes approximately 0.75 seconds to travel the length of a pulmonary capillary. Oxygen from the alveoli can equilibriate with blood in about 0.25 seconds. During heavy exercise, blood travels through the capillaries more rapidly (0.25 -0.30 seconds), but still has time to accept O_2 from the alveoli. During exercise at altitude the speed of the blood through the capillaries is still the same, but the diffusion pressure is less than at sea level, thus there is less force driving the O_2 into the blood. This causes a reduction in hemoglobin saturation and thus a decrease in maximal O_2 consumption (VO_2 max)(Lawler, Powers & Thompson, 1988).

Fulco et al. (1988) found that during exposure to altitudes greater than about 2200m, VO_2 max is immediately diminished in proportion to the reduction in the partial pressure of oxygen in the inspired air. Other researchers have found that altitude starts to affect performance at lower elevations. Squires and Buskirk (1982) studied 12 male subjects who performed VO_2 max tests at sea level and at 914, 1219, 1524 and 2286m. They found that VO_2 max was significantly lower by 4.8, 6.9 and 11.9% at 1219, 1524 and 2286m respectively and concluded that VO_2 max in physically well-conditioned persons is reduced during acute exposure to 1219m and above. A study using rats found that moderate altitude (1350m) lead to manifestations of altitude

hypoxia in the CV system such as an increase in absolute and relative heart weight, right ventricular hypertrophy and to increased resistance of the myocardium to acute anoxia (Barta, Ostadal, Pelouch, Prochazke and Strec, 1985). Other researchers have suggested that there is a loss of 3.2% in VO_2 max for every 305m of ascent above 1500-1600m (Brooks & Fahey, 1985, 471; Buskirk, Kollias & Picon-Reategui, 1966).

Lactate

The increase in blood lactate concentration observed with acute hypoxia is influenced by at least two factors: the altitude ascended and metabolic rate (Young et al., 1982). Some researchers have found that lactate concentration following maximal exercise is the same for hypoxia and normoxia, but because of the reduction in maximal work capacity, maximal lactate concentrations are achieved at lower power outputs at altitude (Adams & Welch, 1980; Bouissou et al., 1986; Hogan et al, 1983; McLellan, Jacobs & Lewis, 1988). However, Dill and Adams (1967), Green, Sutton, Young, Cymerman and Houston (1989) and Klausen, Dill and Horvath (1970) all have found a pronounced reduction in lactate after maximal exhaustive exercise. Blood lacate concentration is dependent upon the relationship of lactate production and release times removal and catabolism. Changes in extracellular acid-base state during exercise or because of environmental changes, influences the relationship between lactate turnover and clearance to altered blood lactate concentrations (Davies, Iber, Keene, McArthur & Path, 1986). Decreased HCO_3^- in the blood as a result of hypoxic exposure may cause a decrease in lactate efflux from the muscle into the blood thus accounting for lower maximal lactate concentrations seen at altitude.

Green et al. (1989) suggested that the lower lactate concentration was due to a decrease in anaerobic glycolysis and this was depressed due to an inability to fully

activate the contractile units of skeletal muscle. Some of the discrepancy may be the result of different protocols. When the control (sea level) experiments are set up so that VO_2 max is attained in 4-6 minutes, the hypoxic test will be shorter. This means that performance times, especially in extreme hypoxia, will be very brief (less than 2 minutes). Under these conditions, it may be that exhaustion is reached before maximum lactate levels are attained (Welch, 1987).

Heart Rate

As with maximum lactate values, maximum heart rate occurs at lower workloads at altitude (Cymerman et al., 1989; Dill & Adams, 1971; Squires & Buskirk, 1982). Several authors have reported a decrease in maximal heart rate (Young et al., 1982), while others have shown no significant difference in max HR (Hogan et al., 1983; Squires and Buskirk, 1982) and some authors, who used multiple levels of hypoxia, have found no effect of milder levels, but significant decreases at higher altitudes (Dill et al., 1966).

Submaximal Exercise

VO_2

The oxygen cost of submaximal exercise at altitude is virtually identical to pre-altitude cost because of the increased submaximal blood flow due to increased submaximal HR (McKardle et al., 1991, 533). Submaximal VO_2 values have not been observed to change in sea level residents even up to 6700m. Hemoconcentration limits SV during exercise, but has no effect on VO_2 during submaximal exercise (Young et al., 1982).

Heart Rate and SaO_2

Most studies have found that heart rate at submaximal workloads is higher in hypoxic conditions for a given workload when compared to normoxic heart rate (Ekblom, 1975; McManus, Horvath, Bolduan & Miller, 1974; Schibye, Klausen, Trap-Jensen, Lund & Hartling, 1988; Wolfel et al., 1991). In the early stages of high altitude exposure, submaximal heart rate may increase 50% above sea level values (Klausen, 1969). The increase in submaximal HR is due to an increase in sympathetic activity which is in response to a decrease in PaO_2 (Schibye et al., 1988; Vogel & Harris, 1967). The increased viscosity of the blood, due to decreased PV which increases peripheral resistance, would also act to increase HR to maintain Q.

Some studies have found identical submaximal HR's at altitude and sea level for given workloads (Noble & Maresh, 1979; Squires and Buskirk, 1982). These two studies were performed at moderate altitudes of 914-2300m and thus, the hypoxic stress may not have been great enough to increase submaximal HR above those values found at sea level.

Arterial oxygen tension and $\%SaO_2$ are maintained near resting levels during moderate exercise at sea level (Willimas, Powers & Stuart, 1986). However, at altitude, the ventilation-perfusion mismatch and diffusion limitation that occur during exercise are worsened resulting in a decrease in PAO_2, PaO_2 and subsequently SaO_2 (Welch, 1987).

Lactate

Past research on the effects of hypoxia on submaximal lactate values have given conflicting results. Some studies have shown blood lactate levels to be higher in acute

hypoxia (Bouissou, Peronnet, Brisson, Helie & Ledous, 1986; Gonzales, Sokari & Claney, 1991; Lee, Cordain, Sockler & Tucker, 1990) while other studies have found a decreased blood lactate accumulation during submaximal exercise at altitude (Dill & Adams, 1971; Klausen, 1966; Yamamoto, Takei, Mutch & Myashita, 1988).

The rationale for the theory of increased lactate production at altitude is that for a given work intensity, the working tissues are more dependent on glycolysis because of the reduced aerobic capacity, hence the higher lactate production for a given workload at altitude. Mazzeo et al. (1991) studied arterial catecholamine responses during exercise with acute and chronic high altitude exposure and found increased epinephrine and decreased arterial norepinephrine levels upon acute exposure to 4300m. With improved oxygenation, which accompanies ventilatory acclimatization and erythropoesis, epinephrine secretion subsides. Mazzeo et al. (1991) found a strong correlation between circulating blood lactate and epinephrine levels ($r=0.95$) that increased with exposure to altitude. Because of this strong relationship between blood lactate and epinephrine, a blunted response in blood lactate may indicate acclimatization (Mazzeo et al, 1991).

Researchers who have found a decreased blood lactate concentration at submaximal workloads in acute hypoxia don't dispute that more lactate may be being produced within the muscle, but rationalize that the decrease in blood lactate seen at altitude is caused by an extracellular acidosis which tends to prevent or slow down the efflux of the muscle lactate into the blood (Shepard, Bouhlel, Vandewalle & Monod, 1988; Yamamoto et al., 1987).

Knuttgen and Saltin (1973) performed several submaximal tests at different percentages of each subjects VO_2 max and found that at lower work intensities (up to

90% VO_2 max) muscle and blood lactate concentrations at altitude were the same or less than sea level.

Post Exercise Recovery

In general, at altitude, as exercise intensity increases, there is a larger increase in post-exercise blood lactate concentration. In addition, the higher the elevation, the greater the increase in post-exercise muscle lactate concentration (Linnarsson, Karlsson, Fagraeuss & Saltin, 1974; Lorentzen, 1962). McLellan, Kavangh & Jacobs (1990) found that muscle lactate increased more in hypoxia, but throughout recovery, blood lactate concentrations were lower. This suggests that in hypoxic conditions the absence of HCO_3^- in the blood will decrease efflux of muscle lactate into the blood thus causing an increase in post-exercise recovery time to clear muscle lactate. The increase in post-exercise muscle lactate levels, the impaired lactate clearance mechanisms, the decreased SaO_2 and the decrease in blood flow to the tissues results in a longer recovery time from both aerobic and anaerobic exercise.

Cardiovascular fitness is very important during recovery from anaerobic work (Jette, 1980; Rhodes & Twist, 1989). In sports requiring repeated anaerobic exertions, such as hockey, the recovery time in between shifts that is normally sufficient at sea level may not be long enough at altitude to allow for adequate recovery of the anaerobic energy systems. As a result, the players may become increasingly fatigued throughout a game and their performance may suffer.

III. PERFORMANCE AT ALTITUDE

Aerobic Performance

At high altitude (2700-3700m) mental deficiency may increase and the performance of discreet motor movements may decrease and individuals may experience other symptoms of altitude effects such as drowsiness, fatigue, headache, nausea or euphoria (Ward, 1987). A physical effort at moderate altitudes can produce higher pulmonary ventilation, a higher heart rate and other symptoms of fatigue (Astrand & Rodahl, 1986, 689). In order to perform aerobic exercise at the same relative intensity at altitude as at sea level, the pace must be slowed. If not, a larger portion of the energy for exercise is provided by anaerobic metabolism and fatigue develops (Faulkner et al., 1968).

Persons with higher maximal aerobic power are better able to tolerate altitude than less fit individuals because they have a higher initial level of fitness. However, they are more affected than persons with lower maximal aerobic power because their maximal aerobic power is more fragile (Astrand & Rodahl, 1986, 690). Highly trained endurance athletes suffer more severe gas exchange impairments during acute exposure to hypoxia than untrained individuals (Lawler, Powers & Thompson, 1988). The possible mechansims for this hypoxemia are: veno-arterial shunt (Astrand, Cuddy, Saltin & Stenberg, 1964), ventilation perfusion mismatch (Gale, Torre-Bueno, Moon, Saltzman & Wagner, 1985; Houston et al., 1987), hypoventilation (Dempsey et al., 1984), and diffusion limitation (Dempsey et al., 1984). Because of the impairment of gas exchange, elite athletes competing at altitude may find that even moderate increases in elevation have a negative effect on their performance.

Saltin (1967) also suggested that differences in anaerobic capacity influence performance at altitude. As previously mentioned, at altitude, a larger portion of energy is derived from the anaerobic system for a given workload, thus leading to fatigue earlier than it would occur at sea level. Athletes who have a larger anaerobic capacity would have an advantage at altitude in that they would be able to maintain a given work intensity for longer than those athletes with a smaller anaerobic capacity (Saltin, 1967). In addition, if efflux of lactate into the blood from the muscle is decreased, then muscle lactate accumulation will occur resulting in a decrease in muscle pH. If anaerobic capacity is higher then so should be the ability to tolerate the decreased muscle pH and thus hypoxia should not be as detrimental to performance.

Anaerobic Sprint Performance and Strength

Altitude causes marked improvements in most events of short duration and high intensity (Brooks & Fahey, 1985, 475) such as running sprints (<400m). Hypoxia does not impair anaerobic sprint performance as measured by 30s or 45s Wingate tests (McLellan, Kavanagh & Jacobs, 1990). Since the O_2 supply is reduced in hypoxic conditions, catabolism of glycogen to lactate may offset the reduced O_2 availability and performance will be maintained at a level comparable to sea level (McLellan et al., 1990).

Young et al. (1980) found there was no effect of hypoxia (4600m) on strength measurements and concluded that muscular endurance does not decrease at altitudes up to 4600m. Bowie and Cumming (1971) reported that sustained handgrip strength was unaffected at a simulated altitude of 3700m. However, a study that focused on the dynamic and sustained static exercise of the quadriceps found that hypoxia (11%O_2)

impaired performance (Eiken & Tesch, 1984). No clear conclusions can be drawn from the literature on the effect of hypoxia on muscle strength.

Acclimatization to Altitude

When athletes condition at moderate altitude their ability to perform at this altitude is enhanced (Guyton, 1986). Smith and Sharkey (1984) recommend that athletes arrive at altitude far enough ahead of their event to allow time for acclimatization. Recommended times for acclimatization for competitive events is 14 days for 2000m and 28 days for 2500m (Astrand & Rodahl, 1986, 703; Ratzin-Jackson & Sharkey, 1988; Smith & Sharkey, 1984). Events that take place below 2000m should require less time for acclimatization as the hypoxic effects are not as severe.

Moderate altitude natives (residence = 800-1200m) may require even less time to acclimatize to higher altitudes. A study done on college basketball players whose residence was at 1000m found that their Hct and Hb were not significantly changed when they were tested 8 hours after arrival at 2200m and they did not display the magnitude of hypoxic response expected of sea level residents (Noble & Maresh, 1979). Similarly, Maresh, Kraemer, Noble and Robertson (1988) showed that moderate altitude natives (1830-2220m) experienced a less impaired capacity for O_2 uptake than sea level natives following 2 days of exposure to 4270m.

However, for many athletes and teams, the logistics of being able to arrive at the place of the event 2-3 weeks ahead of time make it impossible to employ a continuous altitude exposure acclimatization strategy. In addition, arrival at altitude 2 weeks ahead of the event may prove detrimental to performance because training intensity at altitude must be reduced due to a decreased VO_2 max (Haymes & Wells, 1986, 88). However,

intermittent exposure to altitude may successfully acclimatize athletes to altitude without jeopardizing their VO_2 max. Daniels and Oldridge (1970) studied six champion runners who trained over a 10 week period alternating 11 days at sea level and 5 days at 2300m. The acute exposure resulted in a 14% drop in VO_2 max and an 8% slower 1-mile run time at altitude. By the 5th week altitude VO_2max had risen to within 10% of sea-level control values. They concluded that intermittent sea-level stays of as long as 11 days did not interfere with the process of ventilatory-respiratory acclimatization to altitude.

IV. SUMMARY

The literature suggests that prolonged exposure to altitude will result in a rapid decrease in VO_2 max in highly trained athletes (Haymes & Wells, 1991). However, in order to compete at altitude effectively one must acclimatize, which may take from 10 days to several weeks depending on the altitude where competition will occur. This may be a long enough time for significant detraining to occur, especially in athletes with a high VO_2 max. Levine and Houston (1991) stated that the best strategy is to live high (at altitude) and to train low (at sea level) in order to obtain the advantages of living at altitude without sacrificing intensity of training due to decreases in VO_2 max and increases in recovery time. However, financial and practical considerations usually prevent athletes from being able to utilize a high/low strategy. Using an intermittent acclimatization procedure would allow athletes to have the advantage of altitude training and acclimation without sacrificing their VO_2 max. Based on the study done by Daniels and Oldridge (1970) it would appear that this acclimatization procedure should work

but no studies have been conducted to see if this protocol would successfully acclimatize elite athletes to altitude.

The hockey players on the Canadian Olympic Hockey Team (residence = 850m) have reported that an increase in elevation results in fatigue, headaches and lethargy and that this is detrimental to their performance on the ice. No research has been conducted to determine if there is a physiological basis for this complaint.

For the Albertville Olympics, the hockey team will be staying and practising at 1630m elevation and competing at 1500m elevation. These moderate altitudes may lie within the threshold range where the effects of hypoxia may be significant on the athletes performance. The research on the effects of hypoxia have not provided conclusive evidence on the effects of moderate altitude on the physiological status and performance of elite athletes.

Therefore, the purpose of this study was to determine if moving from a low altitude of 850m to a moderate altitude of 1650m would produce acute physiological changes in elite athletes and to determine if acclimatization could be accomplished using intermittent exposures to altitude and if acclimatization results in increased performance.

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APPENDIX C

Table 6

Table 6. Body weight and time to reach steady state VO2 and heart rate before altitude exposure, at altitude and 5 weeks after altitude exposure.

Day	Calgary Pre	LL/Banff 1	LL/Banff 2	LL/Banff 3	Calgary Post 5 wks
Weight (kg)	87.5 (5.0)	87.5 (4.9)	87.3 (4.7)	87.8 (4.7)	86.8 (5.5)
TTSSVO2 (seconds)	228 (43)	234 (45)	245 (43)	226 (43)	231 (44)
TTSSHR (seconds)	238 (39)	261 (38)	261 (42)	251 (38)	246 (35)

APPENDIX D

Informed Consent

INFORMED CONSENT - CANADIAN OLYMPIC HOCKEY TEAM ALTITUDE STUDY

I,_____,do hereby acknowledge:

*I consent to perform four submaximal cycle ergometer tests, one test each day for four days during specified times. I understand that a mouthpiece will be worn and expired gases will be analyzed by a Beckman metabolic measurement cart every 30s for respiratory and metabolic factors.

*I understand that a heart rate monitor will be attached across my mid-sternum region to record my heart rate at rest, every 30s during the submaximal cycle ergometer test and during on-ice testing.

*I understand that 5 mls of blood will be drawn via venapuncture before each morning practise. I understand that I may also experience some discomfort upon insertion of the needle and a small amount of bruising may result at the puncture site.

*Even though, I will be undergoing exercise to a point of temporary exhaustion, I understand there is very little risk involved if I am a healthy, active individual and that emergency equipment and trained personnel are available to deal with unusual situations that may arise.

*I understand that I may temporarily experience local muscle fatigue and discomfort in the legs, nausea, and light headedness, when performing the on-ice repeated sprint test.

*I understand I may ask any questions or request further explanations or information about the procedures at any time before, during or after testing.

*I understand that I may withdraw from, reduce or modify my involvement in the study at any time and that the test may be terminated by the investigators upon observation of any symptoms of distress or abnormal responses.

*I understand that all my results are strictly confidential, that they will be cited only by an ID label or used to calculate a group mean.

*that I do hereby release, _____, and its employees
Name of the institution
from any liability with respect to injury or damage that I may suffer during participation in this study except where the damage or injury is caused by negligence of

_____.
Name of the institution

I acknowledge that I have read, understood and agree to the contents of this informed consent agreement in its entirety.

_____. _____.
Signature Date

APPENDIX E

Dill and Costill Equation for Determination of Plasma Volume

$$BV_a = BV_b\ (Hb_b/Hb_a)$$

$$CV_a = BV_a(Hct_a)$$
$$PV_a = BV_a - CV_a$$

$$BV\% = 100\ (BV_a - BV_b)/BV_b$$
$$CV\% = 100\ (CV_a - CV_b)/CV_b$$
$$PV\% = 100\ (PV_a - PV_b)/PV_b$$

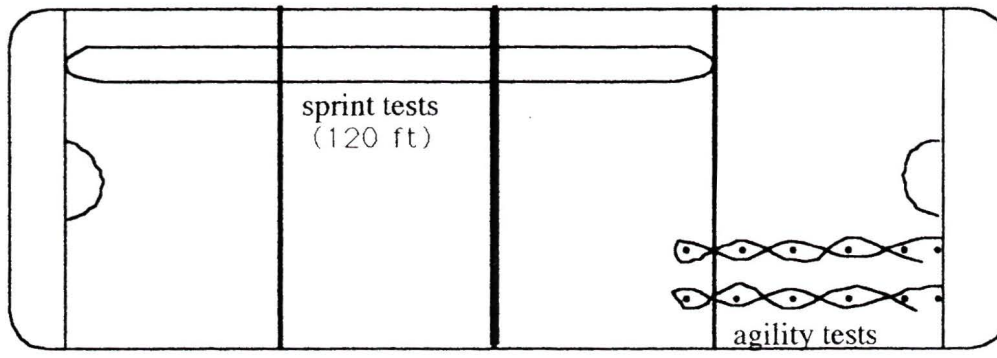
where, BV = blood volume
CV = red cell volume
PV = plasma volume

and b = before treatment
a = after treatment

From: Dill, D.B. & Costill, D.L. (1974). Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. Journal of Applied Physiology, 37(2), 247-248.

APPENDIX F

Diagram of the On-ice Test Set-up


sprint tests
(120 ft)
agility tests

VITA

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Title of Thesis: **PHYSIOLOGICAL AND PERFORMANCE CHANGES OF ELITE ICE HOCKEY PLAYERS IN RESPONSE TO ACUTE EXPOSURE TO MODERATE ALTITUDE.**

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LYNNETH ANN WOLSKI

APRIL 27, 1992