HAEMOGLOBIN SATURATION DURING INCREMENTAL ARM AND LEG EXERCISE

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ABSTRACT

There are few reports concerning the alterations in the percent of haemoglobin saturated with oxygen (%SO₂) during non-steady state incremental exercise. Further, no data exist to describe the %SO₂ changes during arm exercise. Therefore, the purpose of this study was made to assess the dynamic changes in %SO₂ during incremental arm and leg work. Nine trained subjects (7 males and 2 females) performed incremental arm and leg exercise to exhaustion on an arm crank ergometer and a cycle ergometer, respectively. Ventilation and gas exchange measurements were obtained minute by minute via open circuit spirometry and changes in %SO₂ were recorded via an ear oximeter.

No significant difference (p > 0.05) existed between arm and leg work in end-tidal oxygen (PETO₂), end-tidal carbon dioxide (PETCO₂), or %SO₂ when compared as a function of percent VO₂ max. These results provide evidence that arterial O₂ desaturation occurs in a similar fashion in both incremental arm and leg work with the greatest changes in %SO₂ occurring at work rates greater than 70% VO₂ max.

NB. In this paper "%SO₂" does NOT mean "percentage of sulphur dioxide" — Ed.

Key words: Exercise hypoxia, Haemoglobin saturation, Arm ergometry, Cycle ergometry, Incremental exercise.

Much of the literature describing changes in the percent of haemoglobin saturated with oxygen (%SO₂) during exercise has focused on steady-state work at light to moderate work rates. The traditional textbook version of the effects of exercise on arterial blood gases is that the partial pressure of oxygen in arterial blood (Pao₂) and percent haemoglobin saturated with oxygen (%SO₂) remains unchanged from resting values during submaximal work. However, recently this concept has been challenged by reports that Pao₂ may fall during non-steady state leg exercise (Dempsey et al, 1982; Young and Woolcock, 1978). At present, there are few reports concerning the dynamic behaviour of this variable during heavy incremental exercise. Further, to our knowledge, no data exist to describe changes in haemoglobin saturation during arm work. Therefore, the primary objective of the present study was to report the changes in %SO₂ during non-steady state incremental arm and leg exercise.

METHODS

Subjects

Nine trained healthy subjects (seven men and two women) volunteered to participate in these experiments.

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All subjects were engaged in a regular running exercise programme (3-5 days/week), but none were specifically arm trained. Physical characteristics (mean ± SEM) for the subjects are shown in Table I.

| Physical characteristics of the nine subjects. Mean and ± SEM |
|------------------|--------------|-------------|----------------|
| Age (years)      | Height (cm)  | Weight (kg)| VO₂ max (ml·kg⁻¹·min⁻¹) |
|                  |              |            |                      |
| Men              | 27 ± 1.2     | 172.7 ± 2.7| 66.75 ± 1.4          | 58 ± 3                     |
| Women            | 22.5 ± 0.1   | 161.2 ± 0.9| 52.3 ± 0.9            | 48 ± 1                     |

Test Protocol

On one testing day subjects performed an incremental arm crank test while on another day the subjects completed an incremental cycle ergometer test. One to three days rest separated the two experiments and the treatment order was counterbalanced to prevent an ordering effect. All tests were conducted on the same cycle ergometer (Monark) and arm crank ergometer (Monark Rehab Trainer) and performed under uniform environmental conditions (temperature 18.0-19.6°C; barometric pressure Pb) — 754-760 torr (100.5-101.3kPa); relative humidity 69-72%.

The progressive arm test began with subjects cranking (70 rpm) at a work rate of 70 watts, and every minute...
the load was increased by 15 watts until exhaustion. The incremental leg test began at a work rate of 100 watts (70 rpm), and the power output was increased by 30 watts per minute until the subject could not maintain the desired work rate. Subjects were verbally encouraged in both experiments to exercise as long as possible.

Subjects breathed through a low resistance non-rebreathing valve (Daniel's) with the expired gases passing through a 50 cm length of 34 mm diameter tubing into a 5-litre mixing chamber. Expired \text{O}_2 and \text{CO}_2 gas concentrations were monitored by electronic gas analysers (Beckman OM-11 and LB-2, respectively). The gas analysers were calibrated against standardised gases prior to and following each test. End-tidal \text{O}_2 (\text{PETO}_2) and \text{CO}_2 (\text{PETCO}_2) partial pressures were recorded over the last 10 seconds of each work rate via a sampling tube connected to the expiratory side of the Daniel's valve. Inspired ventilation was measured by a turbine flow meter (Pneumoscan S-300) which was calibrated at various velocity profiles against a 120-litre Tissot spirometer.

Heart rates (HR) were determined from electrocardiographic tracings recorded over the last 10 seconds of each stage during exercise. Changes in %SO\textsubscript{2} were measured and recorded over the last 10 seconds of each work rate via an ear oximeter (Biox). Prior to each experiment the ear was cleaned with alcohol and massaged to increase perfusion. The heated oximeter was securely fastened to the pinna and the connecting cable was attached to a support headgear on the subject to prevent movement during exercise.

**Statistical Analysis**

Appropriate regression models using a least squares technique were developed via computer to describe the changes in arterial saturation during progressive arm and leg work as a function of the relative oxygen uptake (i.e. expressed as a percent of VO\textsubscript{2} max during arm and leg work. Additionally, analysis of variance for repeated measures was used to test for differences in all measured variables. A Duncan's multiple range test was employed to determine where differences occurred. Significance was established at \( p < 0.05 \).

**RESULTS**

Data for changes in expired ventilation \( (V_E) \), PETCO\textsubscript{2}, PETO\textsubscript{2}, and HR are presented as a function of the relative work rate in Figure 1. No significant difference existed in PETO\textsubscript{2} or PETCO\textsubscript{2} between arm and leg exercise at any work rate when compared as a function of percent VO\textsubscript{2} max. Expired ventilation differed significantly at several points during the tests with the greatest difference occurring at high work rates. Heart rate differed significantly only at VO\textsubscript{2} max. The reasons for this difference in HR and \( V_E \) between arm and leg work has been discussed elsewhere and will not be addressed within this communication (Åstrand and Rodahl, 1977).

Figure 2 shows the changes in arterial saturation plotted as a function of the relative work rate (i.e. %VO\textsubscript{2} max). Comparison of the slopes of the mean regression lines between arm and leg exercise in Figure 3 revealed that no significant differences existed. Additionally, the lowest %SO\textsubscript{2} occurred at VO\textsubscript{2} max in both experimental conditions with no significant difference between arm and leg work. The results are summarised in Table II.

**DISCUSSION**

The results of this study suggest that arterial O\textsubscript{2} desaturation occurs in both arm and leg exercise at high relative work rates. Figure 3 clearly demonstrates that the trends toward desaturation when graphed as a function of the
relative work rate are similar in both types of work. It is of interest to note that little change in %SO₂ occurred until subjects reached work rates requiring greater than 70 percent VO₂ max with the most acute changes resulting at work rates above 90 percent VO₂ max. The reason(s) for this desaturation during heavy exercise are not clear. However, several possibilities exist: (1) hypoxaemia, (2) a shift to the right of the oxygen-haemoglobin dissociation curve and/or (3) a combination of 1 and 2. The present data do not provide information to support or reject any of the above hypotheses. However, based on data in the literature, it appears likely that decreases in arterial O₂ content during heavy exercise could result from a combination of a reduced PaO₂ and a shift to the right of the oxygen-haemoglobin dissociation curve. Early reports suggested that the PaO₂ does not decrease > 68 during work at sea level (Asmussen and Nielsen, 1960; Hesser and Matell, 1964; Wasserman et al, 1967). However, Dempsey (Personal Communication, 1983) has recently shown that PaO₂ may decrease as low as 54 torr (7.2 KPa) in man during work at high relative work rates. Additionally, Young and Woolcock (1978) have demonstrated large drops in PaO₂ during unsteady-state exercise. The possible reason(s) for this hypoxaemia during heavy exercise remain to be elucidated.

Klein et al (1980) have demonstrated short term high intensity exercise induces a reduction in haemoglobin affinity for oxygen by elevating the standard P₅₀. Further, these authors suggested the magnitude of the change in standard P₅₀ induced by exercise is related to

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**Fig. 2:** Changes in the percent of haemoglobin saturated with oxygen (%SO₂) for each subject during incremental arm and leg exercise. Each subject number on the graph represents a measurement point for that individual.

**Fig. 3:** Mean values for the changes in the percent of haemoglobin saturated with oxygen (%SO₂) during incremental arm and leg exercise.
The end-tidal partial pressure of oxygen (P<sub>ET</sub>O<sub>2</sub>), the end-tidal partial pressure of carbon dioxide (P<sub>ET</sub>CO<sub>2</sub>) and the percentage saturation of oxygen (%SO<sub>2</sub>) during exercise of increasing intensities (%VO<sub>2</sub> max).

<table>
<thead>
<tr>
<th>Work Rate</th>
<th>%VO&lt;sub&gt;2&lt;/sub&gt;</th>
<th>Arm Work</th>
<th>Leg Work</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P&lt;sub&gt;ET&lt;/sub&gt;O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>P&lt;sub&gt;ET&lt;/sub&gt;CO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>%SO&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
<tr>
<td>Rest</td>
<td>108</td>
<td>40</td>
<td>97.5</td>
</tr>
<tr>
<td>50%</td>
<td>112</td>
<td>39</td>
<td>96.5</td>
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<tr>
<td>70%</td>
<td>113</td>
<td>39</td>
<td>95.8</td>
</tr>
<tr>
<td>90%</td>
<td>118</td>
<td>34</td>
<td>93.8</td>
</tr>
<tr>
<td>100%</td>
<td>119</td>
<td>30</td>
<td>88.6</td>
</tr>
</tbody>
</table>

Although the trend toward a reduced %SO<sub>2</sub> was similar in all subjects, the difference between resting %SO<sub>2</sub> and the %SO<sub>2</sub> at 100% VO<sub>2</sub> max ranged from 4 percent in one subject to 16 percent in another. This anomaly in %SO<sub>2</sub> desaturation between healthy subjects during heavy exercise has also been reported by Dempsey et al (1982). The reason(s) for this difference between individuals are unclear. However, it is interesting to note that the three subjects that exhibited the greatest amount of desaturation (i.e. ~ 84%SO<sub>2</sub> at VO<sub>2</sub> max) had the lowest ventilatory equivalents for oxygen (i.e. V<sub>E</sub>/VO<sub>2</sub>) and consequently the lowest P<sub>ET</sub>O<sub>2</sub> when compared to all other subjects at the same workrate. It is tempting to speculate that this finding might explain, at least in part, the differences in %SO<sub>2</sub> between subjects. Additional research seems warranted to clarify this issue.

It is important to consider the possibility that subject apprehension may often alter the normal respiratory rhythm and thus influences the behaviour of arterial blood gases. Special precaution was taken during the course of these experiments to avoid such influences. Subjects were familiarised with the laboratory and testing protocol several times prior to data collection. In addition, the use of the ear oximeter offers many advantages over an arterial catheter in terms of reducing subject anxiety to the experimental protocol. Blood sampling via a catheter in the radial artery would be difficult, if not impossible, during heavy arm exercise; thus, the ear oximeter appears to be the instrument of choice to measure %SO<sub>2</sub> during arm work.

Another methodological concern with exercise studies involving %SO<sub>2</sub> measurement is the possibility that subjects breathing through a mouthpiece might modify their typical ventilatory pattern. To determine if mouthpiece breathing altered the %SO<sub>2</sub> during heavy exercise we repeated the incremental work tests in four subjects without measurement of gas exchange. No significant difference in the pattern of %SO<sub>2</sub> change resulted when these tests were compared to the results obtained during the experiments using the mouthpiece. This finding suggests that the use of the non-rebreathing valve and mouthpiece in this study did not contribute to the observed %SO<sub>2</sub> alterations.

Finally, it is worth noting that the ear oximeter has been shown to be a valid and reliable tool to measure %SO<sub>2</sub> in both steady state and progressive hypoxia during rest and exercise (Scoggan et al, 1977; Saunders et al, 1976; Reuck et al, 1983; Flick and Block, 1977; Poppius and Viljanen, 1977). Further, measurements do not vary significantly with skin pigment, ear thickness, the presence of other light absorbers, or earprobe motion (Scoggan et al, 1977).

In conclusion, these data suggest that the trends toward arterial desaturation with increasing work rate are similar in arm and leg exercise when graphed as a function of the relative workrate. It appears that little change in %SO<sub>2</sub> occurs until subjects reach work rates requiring greater than 70 percent VO<sub>2</sub> max. Further research seems warranted concerning the possibility that maximal O<sub>2</sub> transport may be limited by the respiratory system.

**REFERENCES**


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**BOOK REVIEW**

**Title:** THE MULTIPLY INJURED PATIENT WITH COMPLEX FRACTURES  
**Author:** Marvin H. Meyers  
**Publisher:** Lea & Febiger, Philadelphia (UK Agents — Quest)  
**Price:** $71.50

This book is a multi author book concentrating on all aspects of the acutely traumatised patient with special emphasis placed on the treatment of the fractures involved in such patients. As is so rightly pointed out, often the management of the fracture takes on a completely different picture when occurring within the context of multiple injuries than if it occurred in isolation. Each chapter is well written and the diagrams and radiographic plates of high quality.

The initial part of the book deals with the reception and immediate resuscitation of the injured person. It would have been helpful to have seen greater reference to the fluid management of such cases than is given.

Following this the book turns to the diagnosis and management of the various associated injuries that may occur in such patients. It is gratifying to see that chapters have been set aside to include the management of vascular injuries and the recognition and management of compartment syndromes. Often these are forgotten about but in these days of high velocity trauma are becoming more common and if missed can lead to disastrous consequences.

In order to be complete, a little more general surgery should have been included, a notable omission being abdominal visceral trauma.

The second half of the book ends by dealing in detail on an anatomical basis on the various fractures encountered in such patients and their management. Generally speaking these chapters were of a high quality and well illustrated.

In summary an excellent book with a few notable exceptions. It needs to be read and is not a book that can be quickly picked up and used for solving a particular problem.

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Haemoglobin saturation during incremental arm and leg exercise.

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