The effect of inspiratory muscle fatigue on acid-base status and performance
during race-paced middle-distance swimming

Inspiratory muscle fatigue, swimming and acid-base status

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Abstract

The aim of this study was to investigate the effect of pre-induced inspiratory muscle fatigue (IMF) on race-paced swimming and acid-base status. Twenty-one collegiate swimmers performed two discontinuous 400-m race-paced swims on separate days, with (IMF trial) and without (control trial) pre-induced IMF. Swimming characteristics, inspiratory and expiratory mouth pressures, and blood parameters were recorded. IMF and expiratory muscle fatigue (P < 0.05) were evident after both trials and swimming time was slower (P < 0.05) from 150-m following IMF inducement. Pre-induced IMF increased pH before the swim (P < 0.01) and reduced bicarbonate (P < 0.05) and the pressure of carbon dioxide (PCO₂) (P < 0.05). pH (P < 0.05), bicarbonate (P < 0.01) and PCO₂ (P < 0.05) were lower during swimming in the IMF trial. Blood lactate was similar before both trials (P > 0.05) but was higher (P < 0.01) in the IMF trial after swimming. Pre-induced IMF induced respiratory alkalosis, reduced bicarbonate buffering capacity and slowed swimming speed. Pre-induced and propulsion-induced IMF reflected metabolic acidosis arising from dual role breathing and propulsion muscle fatigue.
Reducing the force generating capacity of the inspiratory muscles by 20-31% before exercise decreases the duration of both exhaustive cycle ergometry (Mador & Acevedo, 1991; Wüthrich et al., 2013) and fatiguing calf plantar flexion exercise (McConnell & Lomax, 2006). The curtailment in performance might be the result of evoking the inspiratory muscle metaboreflex, (McConnell & Lomax, 2006; Sheel et al., 2001). Activation of the inspiratory muscle metaboreflex causes a sympathoexcitatory increase in vascular resistance via vasoconstriction of vascular beds in both inactive tissue (Sheel et al., 2001) and active muscle (Rodman, Henderson, Smith & Dempsey, 2003). This could elevate blood lactate and lower pH because of the negative consequences that high breathing loads have on oxygen supply to, and metabolite removal from, working muscle (Harms et al., 1997; Harms, Wetter, St Croix, Pegelow & Dempsey, 2000).

It is also likely that breathlessness will increase in the presence of inspiratory muscle fatigue (IMF) resulting in tachypnoea at rest (Gallagher, Hof & Younes, 1985; Śliwiński, Yan, Gauthier & Macklem, 1996), during cycle (85-90 % of maximal power) ergometry (Mador & Acevedo, 1991; Śliwiński et al., 1996), and during recovery from fatigue (Gallagher et al., 1985). The increase in breathing frequency is aimed at minimizing dyspnoea (Gallagher et al., 1985; Śliwiński et al., 1996) and may (Gallagher et al., 1985; Mador & Acevedo, 1991) or may not (Śliwiński et al., 1996) be associated with a fall in tidal volume. The inducement of IMF can also increase the phasic and tonic activity of the abdominal muscles. As this permits a more efficient force-length relationship of the diaphragm, less inspiratory muscle activation is required for a given degree of muscle shortening (Śliwiński et al., 1996).
Propulsion induced inspiratory muscle fatigue (IMF), which has been documented in all four swimming strokes (Lomax, Iggleden, Tourrell, Castle & Honey, 2012) and distances ranging from 100-m to 400-m (Brown & Kilding, 2011; Lomax & McConnell, 2003; Thomaidis et al., 2009), has been shown to increase breathing frequency (fr) and stroke rate (SR) during constant velocity sub-maximal swimming (Lomax & Castle, 2011). Tachypnoea at a given velocity is disadvantageous, even if it reduces dyspnoea, because breathing pattern is constrained by stroke mechanics and increasing fr disrupts streamlining (Seifert, Chollet & Allard, 2005). However, reducing fr to avoid such disruption is not without problem. The natural stroke-induced reduction in fr during front crawl is associated with hypercapnia and hence respiratory acidosis. As well as curtailing swimming distance (Kapus, Ušaj, Kapus & Strumbelj, 2003), hypercapnia is also capable of impairing respiratory muscle function (Jonville et al., 2002; Rafferty et al., 1999). In addition, tidal volume increases to compensate for the lower fr (Rodriguez, 2000) thereby increasing the elastic load of breathing leading to IMF (Jakovljevic & McConnell, 2009).

It is currently unknown whether IMF in swimming is partly due to respiratory acidosis arising from a constrained breathing pattern, and whether fr increases following its occurrence to relieve dyspnoea or maintain normocpania. This is complicated further by the fact that some breathing muscles during swimming are also key in generating forward propulsion and upper body stabilisation during the front crawl arm stroke (Lomax, Tasker & Bostanci, 2014; Lomax, Tasker & Bostanci 2015; Nuber, Jobe, Perry, Moynes & Antonelli, 1986; Pink, Perry, Browne, Scovazzo & Kerrigan, 1991) and could therefore contribute to IMF independently of breathing. Pre-inducing IMF
before swimming could therefore be used to distinguish the effects of IMF on acid-base status and stroke mechanics more clearly. It is conceivable that pre-existing fatigue of these dual role muscles could lead to sustained metabolic perturbations during subsequent swimming similar to that occurring following the inducement of the inspiratory muscle metaboreflex (McConnell & Lomax, 2006) thereby curtailing swimming performance.

The aim of this study was to investigate the effect of pre-induced IMF on acid-base status during middle-distance race-paced swimming and its impact on stroke characteristics. We hypothesised that swimming with pre-induced IMF would exacerbate the metabolic perturbations and acidosis during subsequent swimming, increase SR and $f_r$, and slow swimming time.

Methods

Experimental approach to the problem

Swimmers completed one pulmonary familiarisation and three swimming trials in an indoor heated (mean ± SD: 27.2 ± 0.3 °C) 25-m swimming pool. The first trial was used to determine maximum-effort 400-m front crawl swimming time. The remaining two trials were administered in a counterbalanced order and consisted of a single 400-m broken (8 × 50-m) front crawl swim. One of the swims was preceded by the inducement of IMF (IMF trial) and the other was not (control trial). Swimming characteristics (SR, $f_r$, swim time) were recorded during each swim and maximal inspiratory and expiratory mouth pressures (PImax and PEmax, respectively), pH, bicarbonate (HCO₃⁻), partial pressures of oxygen and carbon dioxide (PO₂ and PCO₂,
respectively) and blood lactate ([b lac]) were measured pre, mid (except PEmax and [b lac]) and after each experimental swim from the earlobe.

Participants

Twenty-one well-trained club level swimmers volunteered for this study (see Table 1 for their descriptive characteristics). All provided written informed consent and testing conformed to the Declaration of Helsinki. Institutional ethical approval was received before the start of the study.

**Table 1 about here**

Pulmonary familiarisation

PImax (measured from residual volume) and PEmax (measured from total lung capacity) were determined with the nose occluded and using a hand-held respiratory pressure meter whilst standing on poolside (RPM, Micro Medical Ltd, Kent, UK). Reliability was deemed present when the three highest manoeuvres from a series of manoeuvres were within 10 cmH2O. Forced vital capacity (FVC) and forced expired volume in the first second of exhalation (FEV1) were recorded for descriptive purposes using a digital spirometer (Micro Spirometer, Micro Medical Ltd, Kent, UK). Participants completed a minimum of three satisfactory manoeuvres with the highest recorded.
Swimming trials

All swimming trials were initiated from a push start, completed using the front crawl stroke, and occurred on separate days. The time taken to complete the maximum effort (race-paced) 400-m swim was used to determine the pace and hence target times for the two subsequent broken 400-m swimming trials, which consisted of 8 x 50-m at 400-m race pace. Each 50-m partial was separated by 10 s of rest, with the exception that a 60 s rest separated the 4th and 5th 50-m partial so that PImax could be measured and a capillary blood sample taken (modified from Aujouannet, Bonifazi, Hintzy, Vuillerme & Rouard, 2006).

PImax was assessed before the inducement of IMF in the IMF trial (baseline1), before the 1st 50-m partial (baseline2), following the 4th 50-m partial (mid-swim) and after the final 50-m partial (end-swim). PEmax was assessed at baseline2 and end-swim only. Additionally, in a sub-set of 13 participants, b[lac], pH, HCO3, PO2 and PCO2 were measured from a capillary blood sample (60-80 µl) taken by micro puncture from a hyperaemic earlobe and collected in heparinized glass capillaries. b[lac] was diluted (LKM41 lactate solution, Dr. Lange, Berlin, Germany) and analysed using a photometer (MINI8, Dr. Lange, Berlin, Germany) at baseline2 and end-swim. pH, HCO3, PCO2 and PO2 were analysed using a blood gas analyser (ABL5, Radiometer, Copenhagen, Denmark) at baseline1, baseline2, mid-swim and end-swim. Additionally, dyspnoea (Borg CR10 scale) was assessed before and at end-swim.

To measure clean swimming SR and fr, a 12.5-m zone was identified from the middle of the pool and stroke characteristics were measured in this segment only. SR was determined as the number of strokes divided by the time taken to swim through the
12.5-m zone (Hz, measured using a stop watch) and multiplied by 60 to convert to cycles min\(^{-1}\) (Lomax et al., 2013). \(fr\) was calculated as the number of times the head turned during the 12.5-m zone divided by time taken to cover the 12.5-m zone multiplied by 60 (Lomax et al., 2013). The values were then averaged over the first and second 25-m length of each 50-m partial to give a mean value per 50-m partial. Additionally, the time taken (s) to complete each 50-m partial was recorded using a stop watch.

**Inspiratory muscle fatigue inducement**

IMF was induced using a commercially available inspiratory muscle trainer (POWERbreathe, HaB International, Southam, UK). With the nose occluded, participants generated 70% of their \(P_{I_{\text{max}}}\) using a duty cycle of 0.60 (three seconds for inspiration and two seconds for expiration) and an \(fr\) of 12. Participants coordinated breathing pattern with a metronome and continued with this pattern until it could no longer be maintained for three consecutive breaths. Participants then continued for a further minute after which \(P_{I_{\text{max}}}\) was re-assessed and recorded as baseline\(_2\). This has been shown to result in a fall in \(P_{I_{\text{max}}}\) of 17-25% in swimmers (Lomax & Castle, 2011; Lomax Tasker & Bostanci, 2014), which is consistent with the magnitude (8-29%) observed following 100-m-400-m front crawl swimming (Brown & Kilding, 2011; Jakovljevic & McConnell, 2009; Lomax & McConnell, 2003; Lomax et al., 2013; Thomaidis et al., 2009).
Data analysis

Data were assessed for normality using a Shapiro-Wilk test. The impact of pre-induced IMF on PImax, b[lac], pH, HCO₃, PO₂, PCO₂, between baseline₁ and baseline₂ (IMF trial) were assessed using paired samples t-tests. Two-way (condition x time) repeated measures ANOVA assessed for differences in swim time, SR, fr, b[lac], pH, HCO₃, PO₂, PCO₂, PImax and PEMax between IMF and control trials between baseline₂, mid-swim and end-swim. Post hoc analyses were undertaken using repeated measures ANOVA’s with Bonferroni adjustments and paired samples t-tests. Wilcoxon-Signed rank tests were used to assess post swim dyspnoea between trials expressed as delta change (Δ) from baseline₁.

Effect sizes were calculated using Cohen’s d for parametric data, whereby 0.2 was deemed small, 0.6 moderate, 1.2 large, 2.0 very large and 4.0 extremely large (Hopkins, Marshall, Batterham & Hanin, 2009). For non-parametric data, effect sizes were calculated as r, whereby r is the z score divided by the square root of the total number of observations. A value of .1 is deemed small, .3 medium and .5 and above large (Field, 2013). Significance was set as P ≤ 0.05 and statistical analyses were undertaken using IBS SPSS statistics version 24 (Chicago, Illinois, USA). Unless otherwise stated data are presented as mean ± SD.

Results

Pre-inducing IMF caused PImax to fall by 19 ± 10 cmH₂O from 130 ± 30 cmH₂O to 111 ± 30 cmH₂O (P < 0.001, d = 0.67). Time (F = 15.230, P < 0.001) and condition (F = 9.911, P = 0.08) were both significant for PImax and an interaction was observed (F
Swimming with pre-induced IMF did not cause PImax to fall further. When IMF was not pre-induced, PImax fell by mid-swim (P < 0.001, d = 0.87) but did not fall any further by end-swim. PEmax was unaffected by condition but was lower after swimming in both trials (F = 24.704, P < 0.001: Figure 1).

Swimming time slowed during the latter part of each trial (F = 18.827, P < 0.001) and there was an interaction between time and condition (F = 3.579, P = 0.001; Table 2). SR and fr were affected by condition (SR: F = 29.291, P < 0.001; fr: F = 14.93, P = 0.001) and time (SR: F = 3.958, P = 0.044; fr: F = 5.820, P = 0.001) but no interactions were observed (Table 2).

Mean SR and fr were correlated in both control (r = 0.690, P = 0.001) and IMF (r = 0.675, P = 0.001) trials, as were mean swimming time and SR (control: r = -0.722, P < 0.001; IMF: r = -0.762, P < 0.001), and mean swimming time and fr (control: r = -0.528, P = 0.014; IMF: r = -0.534, P = 0.013). The change in PImax was not correlated with SR, fr or swimming time (P > 0.05).

The inducement of IMF increased pH (t = -4.129, P = 0.001), decreased PCO₂ (t = 4.813, P < 0.001) and decreased HCO₃⁻ (t = 3.498, P = 0.004 ) from baseline₁ to baseline₂, but had no impact on PO₂ (P > 0.05). Main effects were observed for b[lac] (time: F = 18.374, P = 0.001; condition: 191.406, P < 0.001), HCO₃⁻ (time: F =
154.314, \( P < 0.001 \); condition: \( F = 19.563, P = 0.001 \), \( \text{PCO}_2 \) (time: 6.218, \( P = 0.007 \); condition: \( F = 19.138, P = 0.001 \)) and pH (time: \( F = 110.016, P < 0.001 \)). Interactions were observed between time and condition for b[lac] (\( F = 12.659, P = 0.004 \)), pH (\( F = 19.930, P < 0.001 \)), \( \text{HCO}_3^- \) (\( F = 4.432, P = 0.024 \)) and \( \text{PCO}_2 \) (\( F = 5.381, P = 0.012 \)). A moderate correlation (\( r = 0.450, P = 0.024 \)) was observed in \( fr \) and \( \text{PCO}_2 \) for \( \Delta \) between mid- and end- swim (pooled data). \( \text{PO}_2 \) was unaffected by time or condition (\( P > 0.05 \)) (Table 3). Dyspnoea was higher (\( z = -2.910, P = 0.003, r = -0.46 \)) following the IMF trial (IMF trial: 8.1 ± 1.3; control trial: 7.4 ± 2.3).

**Table 3 here**

Discussion

It was our aim to assess the impact of pre-induced IMF on acid-base status during middle-distance race-paced swimming and stroke characteristics. Pre-inducement of IMF caused respiratory alkalosis, which complicates the interpretation of the findings. Nevertheless, the main observations were: 1) pre-induced IMF exacerbated the metabolic perturbations occurring during subsequent swimming; 2) IMF, whether pre-induced or propulsion induced, is unlikely to be attributed to respiratory acidosis; 3) pre-induced IMF slowed swimming time; and 4) \( fr \) increased in both trials but was not correlated with IMF.

The pre-inducement of IMF caused \( \text{PImax} \) to fall by 14%, which is consistent with that reported in response to swimming per se (Jakovljevic & McConnell, 2009; Lomax et al., 2012; Lomax et al., 2015; Thomaidis et al., 2009). The IMF protocol significantly increased pH, reduced \( \text{HCO}_3^- \) and caused a non-significant fall in \( \text{PCO}_2 \).
This meant that swimmers began the swim in a state of mild hypocapnia and alkalosis. It is likely that this state developed because of hyperventilation arising from the increase in breathing depth associated with the IMF protocol and not IMF per se (Chin et al., 2007; Costanzo, 2010; Le Blanc, Parolin, Jones & Heigenhauser, 2002). It could therefore be argued that as the method we adopted to induce IMF created a breathing pattern (hyperventilation) antithetical to that in front crawl swimming (hypoventilation), it may not reflect the metabolic milieu accompanying swimming-induced IMF. Indeed, the observation that swimmers began the swim in the IMF trial with lower HCO₃ levels means that their ability to buffer H⁺ during subsequent swimming was likely compromised. This could be exacerbated by a slowing of pulmonary oxygen uptake kinetics and microvascular blood flow, which have been observed in the presence of hyperventilation-induced hypocapnic alkalosis (Chin et al., 2007; Chen, Heigenhauser, Paterson & Kowalchuk, 2013). Contrastingly, when CO₂ is added to an inspirate during hyperventilation to prevent hypocapnic alkalosis, pH and PCO₂ are prevented from falling and pulmonary oxygen uptake kinetics and microvascular blood flow are improved (Chin et al., 2013). Had a hypercapnic gas mixture been administered during IMF inducement in the current study, the resultant respiratory alkalosis would have been prevented. This approach should be adopted in future.

The control trial did not suffer from this same limitation as swimmers began the control trial swim with normal pH, HCO₃ and PCO₂ values (Table 3). By mid-swim (i.e. 200-m) however, PImax had fallen by 24% indicating the presence of IMF (Figure 1). At this same time point, pH had become mildly acidic (i.e. < 7.35), HCO₃ levels had fallen (Table 3), but there was no evidence of hypercapnia or respiratory acidosis.
This pattern of change in pH, HCO₃ and PCO₂ during swimming in the control trial was similar to the pattern observed in response to swimming in the IMF trial, but the absolute values differed (Table 3).

Pre-inducing IMF did exacerbate the metabolic perturbations (e.g. pH, HCO₃, PCO₂ and b[lac]) occurring during the swim. Despite reducing the buffering capacity of H⁺ in this trial it did not translate into a greater magnitude of IMF (Figure 1). This does not mean that a lowered buffering capacity was without consequence. As the progressive fall in pH and HCO₃ (Table 4) was greater in the IMF trial, the slower swimming times (Table 2) in this trial might be indicative of a reduced ability to buffer H⁺ generated during swimming.

Acidosis can be respiratory or metabolic in origin, or can be a combination of both (Brooks et al., 2000). The progressive fall in pH without concomitant hypercapnia makes it likely we detected its metabolic origin. Moreover, the development of IMF, whether pre-induced or propulsion-induced, cannot be attributed to ventilatory impairment causing respiratory acidosis; at least not when the breathing pattern is optional and fr is ≥ 25 breaths min⁻¹. However, there are a number of caveats to this interpretation.

It is known that exercise can increase carbon dioxide storage in the body and this storage capacity is diminished at higher exercise intensities leading to an increase in carbon dioxide excretion (Jones & Jurkowski, 1979). The relatively short duration of the 50-m repeat swims and the fact that they were not maximal effort swims but were instead based on 400-m race pace, could be incompatible with the detection of
hypercapnia. Additionally, it took approximately 20-30 s to obtain blood samples for analysis during which time breathing was very intense. If a small swimming-induced increase in PCO$_2$ did occur, it could be reversed during this period resulting in the measured PCO$_2$ failing to change. Lastly, failure to observe hypercapnia does not necessarily mean that ventilation poses no limitation during front crawl swimming, or that hypercapnia does not occur. It may be that a ventilatory limitation was not sufficient to cause respiratory acidosis. In support of this, Ušaj (1999) found ventilation to be less effective at compensating for metabolic acidosis during 400-m race-paced front crawl swimming (pH of 7.09 ± 0.09, PCO$_2$ of 4.0 ± 0.5 kPa) compared to maximal effort kayak paddling (pH of 7.17 ± 0.05, PCO$_2$ of 3.6 ± 0.2 kPa) but this failed to result in hypercapnia in the former. We observed a similar pattern and also found the Δ (increase) in $f_r$ between mid- and end- swim (mid-swim being the first time point where IMF was evident in both trials) was correlated with Δ (fall) in PCO$_2$ ($r = 0.450$), suggesting that the increase in $f_r$, which occurred in both trials, partly contributed to the prevention of hypercapnia. However, the small coefficient of determination (20%) indicates that prevention of hypercapnia was not the primary cause for the increase in $f_r$.

It has been proposed that stimulation of thin fibre afferents within fatigued inspiratory muscles create an additional breathing stimulus. This increase in central respiratory drive to the weakened inspiratory muscles is discernible as dyspnoea (Gallagher et al., 1985; Mador & Acevedo, 1991; Sliwinski et al., 1996) and the resultant tachypnoea is thought to be directed at minimising this sensation (Gallagher et al., 1985). Post swim dyspnoea was higher than baseline in both trials and was higher still in the IMF trial, but no interaction was observed between condition and time for $f_r$. This lack of
interaction is not surprising given that no interaction was observed between time and condition for $f_r$.

In previous studies we have shown that 20 s front crawl sprinting following the inducement of IMF leads to an increase in SR but not $f_r$ (Lomax et al., 2014) and that IMF magnitude is not correlated with $f_r$ during the four swimming strokes (Lomax et al., 2012). Although $f_r$ and SR were correlated in the current study ($P < 0.001$), the coefficient of determination was only 46-48%. Thus, the cause of the increase in $f_r$ that may or may not accompany IMF in swimming is multifactorial and is likely impacted by the ventilatory drive to maintain normocapnia, a need to alleviate dyspnoea, and an increase in SR if SR does increase: SR did not increase in the present study but has been shown to previously (Lomax & Castle, 2011).

It is important to recognise here that the breathing muscles in swimming are also key propulsion muscles. For example, the pectoralis major, latissimus dorsi, upper trapezius, and serratus anterior, are activated during deep inspirations (Kendall et al., 2005) and front crawl swimming (Nuber et al., 1986; Pink et al., 1991). We have shown that 20 s arms only front crawl sprinting is sufficient to induce pectoralis major fatigue but not latissimus dorsi fatigue (Lomax et al., 2014). In contrast, latissimus dorsi and pectoralis major fatigue have been reported following 100-m (63 ± 2 s) full stroke front crawl swimming (Stirn et al., 2011). Given that the measurement of PImax is holistic in nature, the pressure recorded at the mouth will reflect the collective activity of all the muscles recruited (Gibson, 1995). This will include contributions from muscles vital in creating forward propulsion and trunk stability (Pink et al., 1991) and fatigue of these muscles is evident in the PImax manoeuvre (Lomax et al., 2015).
It is therefore possible that the propulsion and stabilisation requirements of the dual role muscles during swimming caused fatigue of these muscles and this in-turn was evident in the holistic assessment of PImax and PEmax. The presence of IMF and expiratory muscle fatigue therefore does not automatically indicate a breathing induced origin for fatigue. Rather, it could be propulsion induced. If this is the case, the aetiology of IMF in the present study differed between the two trials: breathing induced in the IMF trial and propulsion induced in the control trial. However, the net effect was the same: the dual role breathing and propulsion/trunk stabilisation muscles experienced fatigue. As we did not measure electromyography of the dual role muscles or their force output while swimming, we are unable to identify exactly which muscles were affected.

**Conclusion**

Swimming with pre-induced IMF caused transient respiratory alkalosis, which was reversed by mid-swim. Pre-induced IMF was associated with a greater level of metabolic perturbation including reduced H\(^+\) buffering capacity, increased acidosis during subsequent swimming and slowed swimming times. However, the overall magnitude of IMF experienced was similar between IMF and control trials and the increase in \(f_s\), accompanying both trials was not correlated with IMF but was correlated with PCO\(_2\).

The cause of acidosis and IMF, whether pre-induced or propulsion induced, could not be attributed to ventilatory impairment and therefore must have been metabolic in
origin. Given the dual role function of the affected muscles we think it likely that IMF occurring in response to swimming reflects propulsion induced fatigue during 400-m crawl swimming.
References


Figure 1. PImax (A) and PEmax (B) immediately before the swim (baseline), mid-swim and end-swim in control (filled bars) and IMF (open bars) trials.

Notes: * (P < 0.01) different to control trial at given time point; ** (P < 0.01) different to baseline within trial. See text for abbreviations.
**Figure 2: Changes in **PImax** during exercise.**

A. **PImax (cmH2O)**

B. **PEmax (cmH2O)**

Legend:

- Baseline2
- Mid-swim
- End-swim

Significance:

- **P < 0.01**
Table 1. Descriptive characteristic of swimmers at the start of the study: mean ± SD

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>Age (years)</td>
<td>22 ± 3</td>
<td>22 ± 3</td>
<td>22 ± 3</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>71.2 ± 12.7</td>
<td>77.6 ± 13.1</td>
<td>62.3 ± 4.9**</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.75 ± 0.10</td>
<td>1.81 ± 0.09</td>
<td>1.65 ± 0.07**</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>5.26 ± 1.11</td>
<td>6.04 ± 0.74</td>
<td>3.69 ± 0.39**</td>
</tr>
<tr>
<td>FEV$_1$ (l s$^{-1}$)</td>
<td>4.42 ± 0.91</td>
<td>4.98 ± 0.77</td>
<td>4.21 ± 0.42**</td>
</tr>
<tr>
<td>FEV$_1$/FVC (%)</td>
<td>85 ± 7</td>
<td>82 ± 6</td>
<td>88 ± 7</td>
</tr>
<tr>
<td>PImax (cmH$_2$O)</td>
<td>134 ± 29</td>
<td>136 ± 32</td>
<td>132 ± 27</td>
</tr>
<tr>
<td>PEmax (cmH$_2$O)</td>
<td>154 ± 36</td>
<td>164 ± 42</td>
<td>138 ± 24</td>
</tr>
</tbody>
</table>

Notes. **(P < 0.01) different to males. See text for abbreviations.
Table 2. Swimming time, stroke rate (SR) and breathing frequency ($f_r$) per 50-m distance and partial number per trial: group mean ± SD (n = 21)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Trial</th>
<th>50-m</th>
<th>100-m</th>
<th>150-m</th>
<th>200-m</th>
<th>250-m</th>
<th>300-m</th>
<th>350-m</th>
<th>400-m</th>
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</thead>
<tbody>
<tr>
<td>Time (s)</td>
<td>control</td>
<td>37.1 ± 8.1</td>
<td>39.0 ± 7.3**</td>
<td>39.7 ± 6.7**</td>
<td>40.3 ± 7.3**</td>
<td>38.3 ± 7.2</td>
<td>39.8 ± 7.1*</td>
<td>41.0 ± 7.2**†</td>
<td>40.9 ± 7.3**§§</td>
</tr>
<tr>
<td>IMF</td>
<td>36.4 ± 6.7</td>
<td>39.0 ± 6.7**</td>
<td>40.1 ± 6.5**</td>
<td>40.7 ± 7.3**</td>
<td>38.7 ± 7.0</td>
<td>40.7 ± 7.3*</td>
<td>41.6 ± 7.3**†</td>
<td>41.5 ± 7.6**§§</td>
<td></td>
</tr>
<tr>
<td>SR</td>
<td>control</td>
<td>37 ± 9</td>
<td>35 ± 7</td>
<td>34 ± 6§</td>
<td>34 ± 6§</td>
<td>36 ± 6</td>
<td>35 ± 5</td>
<td>35 ± 5</td>
<td>35 ± 5</td>
</tr>
<tr>
<td>IMF</td>
<td>39 ± 8†</td>
<td>37 ± 7</td>
<td>35 ± 5§</td>
<td>35 ± 5§</td>
<td>38 ± 6</td>
<td>36 ± 5</td>
<td>36 ± 6</td>
<td>37 ± 6</td>
<td></td>
</tr>
<tr>
<td>$f_r$</td>
<td>control</td>
<td>25 ± 9</td>
<td>26 ± 7§</td>
<td>28 ± 9*</td>
<td>27 ± 7§§</td>
<td>28 ± 7*</td>
<td>29 ± 6**</td>
<td>28 ± 5*</td>
<td>29 ± 6*</td>
</tr>
<tr>
<td>IMF</td>
<td>26 ± 9</td>
<td>29 ± 8**</td>
<td>30 ± 8**</td>
<td>30 ± 7**</td>
<td>30 ± 7*</td>
<td>31 ± 7**</td>
<td>31 ± 6**</td>
<td>32 ± 7**</td>
<td></td>
</tr>
</tbody>
</table>

Note. *(P < 0.05) **(P < 0.01) different to 1st 50-m; †(P < 0.05) different to 2nd 50-m; §§(P < 0.01) different to 5th 50-m; ¶(P < 0.05) §§(P < 0.01) different to 6th 50-m; ¶¶(P < 0.01) different to control at time point. See text for abbreviations.
Table 3. Blood parameters measured before, mid-swim (200-m) and end-swim (400-m) per trial: group mean ± SD (n = 13)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Trial</th>
<th>Baseline₁</th>
<th>Baseline₂</th>
<th>Mid-swim</th>
<th>End-swim</th>
</tr>
</thead>
<tbody>
<tr>
<td>b[lac] (mmol·l⁻¹)</td>
<td>control</td>
<td>/</td>
<td>1.4 ± 0.2</td>
<td>/</td>
<td>8.2 ± 2.4**</td>
</tr>
<tr>
<td></td>
<td>IMF</td>
<td>/</td>
<td>1.6 ± 0.5</td>
<td>/</td>
<td>10.1 ± 1.9**</td>
</tr>
<tr>
<td>pH</td>
<td>control</td>
<td>/</td>
<td>7.44 ± 0.03††</td>
<td>7.32 ± 0.04**</td>
<td>7.27 ± 0.04**††</td>
</tr>
<tr>
<td></td>
<td>IMF</td>
<td>7.43 ± 0.01</td>
<td>7.48 ± 0.04$§§$</td>
<td></td>
<td>7.30 ± 0.04$§§$</td>
</tr>
<tr>
<td>HCO₃ (mmol·l⁻¹)</td>
<td>control</td>
<td>/</td>
<td>24.1 ± 1.4††</td>
<td>18.9 ± 2.7**</td>
<td>16.4 ± 2.7**††</td>
</tr>
<tr>
<td></td>
<td>IMF</td>
<td>24.2 ± 1.2</td>
<td>22.8 ± 1.5$§§$ ‖ ‖</td>
<td>16.8 ± 2.6$§§$ ‖ ‖</td>
<td>13.8 ± 2.3$§§$ ‖ ‖</td>
</tr>
<tr>
<td>PCO₂ (kPa)</td>
<td>control</td>
<td>/</td>
<td>4.8 ± 0.4</td>
<td>5.0 ± 0.5</td>
<td>4.9 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>IMF</td>
<td>4.9 ± 0.2</td>
<td>4.1 ± 0.7$§§$ ‖</td>
<td>4.7 ± 0.5* ‖</td>
<td>4.5 ± 0.5 ‖</td>
</tr>
<tr>
<td>PO₂ (kPa)</td>
<td>control</td>
<td>/</td>
<td>10.9 ± 1.2</td>
<td>11.8 ± 2.4</td>
<td>11.6 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>IMF</td>
<td>11.9 ± 2.5</td>
<td>11.5 ± 3.7</td>
<td>12.0 ± 1.9</td>
<td>11.7 ± 1.2</td>
</tr>
</tbody>
</table>

Note. $§(P < 0.05)$ $§§(P < 0.01)$ different to baseline₁ (within-trial comparison with baseline₂); *(P < 0.05) **(P < 0.01) different to baseline₂; †(P < 0.05) ††(P < 0.01) different to mid-swim; ‖(P < 0.05) ‖‖(P < 0.01) different to control trial at given time point. See text for abbreviations.