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3   The effect of inspiratory muscle fatigue on acid-base status and performance

4   during race-paced middle-distance swimming

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6   Inspiratory muscle fatigue, swimming and acid-base status

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56   **The effect of inspiratory muscle fatigue on acid-base status and performance**

57   **during race-paced middle-distance swimming**

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83   **Abstract**

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85   The aim of this study was to investigate the effect of pre-induced inspiratory muscle

86   fatigue (IMF) on race-paced swimming and acid-base status. Twenty-one collegiate

87   swimmers performed two discontinuous 400-m race-paced swims on separate days,

88   with (IMF trial) and without (control trial) pre-induced IMF. Swimming

89   characteristics, inspiratory and expiratory mouth pressures, and blood parameters were

90   recorded. IMF and expiratory muscle fatigue ($P < 0.05$) were evident after both trials

91   and swimming time was slower ($P < 0.05$) from 150-m following IMF inducement.

92   Pre-induced IMF increased pH before the swim ($P < 0.01$) and reduced bicarbonate (P

93   < 0.05) and the pressure of carbon dioxide (PCO_2) ($P < 0.05$). pH ($P < 0.05$),

94   bicarbonate ($P < 0.01$) and PCO_2 ($P < 0.05$) were lower during swimming in the IMF

95   trial. Blood lactate was similar before both trials ($P > 0.05$) but was higher ($P < 0.01$)

96   in the IMF trial after swimming. Pre-induced IMF induced respiratory alkalosis,

97   reduced bicarbonate buffering capacity and slowed swimming speed. Pre-induced and

98   propulsion-induced IMF reflected metabolic acidosis arising from dual role breathing

99   and propulsion muscle fatigue.

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Introduction

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).

133

134   Propulsion induced inspiratory muscle fatigue (IMF), which has been documented in

135   all four swimming strokes (Lomax, Iggleden, Tourell, Castle & Honey, 2012) and

136   distances ranging from 100-m to 400-m (Brown & Kilding, 2011; Lomax &

137   McConnell, 2003; Thomaidis et al., 2009), has been shown to increase breathing

138   frequency (fr) and stroke rate (SR) during constant velocity sub-maximal swimming

139   (Lomax & Castle, 2011). Tachypnoea at a given velocity is disadvantageous, even if

140   it reduces dyspnoea, because breathing pattern is constrained by stroke mechanics and

141   increasing f_r disrupts streamlining (Seifert, Chollet & Allard, 2005). However,

142   reducing f_r to avoid such disruption is not without problem. The natural stroke-induced

143   reduction in f_r during front crawl is associated with hypercapnia and hence respiratory

144   acidosis. As well as curtailing swimming distance (Kapus, Ušaj, Kapus & Štrumbelj,

145   2003), hypercapnia is also capable of impairing respiratory muscle function (Jonville

146   et al., 2002; Rafferty et al., 1999). In addition, tidal volume increases to compensate

147   for the lower f_r (Rodriguez, 2000) thereby increasing the elastic load of breathing

148   leading to IMF (Jakovljevic & McConnell, 2009).

149

150   It is currently unknown whether IMF in swimming is partly due to respiratory acidosis

151   arising from a constrained breathing pattern, and whether f_r increases following its

152   occurrence to relieve dyspnoea or maintain normocpania. This is complicated further

153   by the fact that some breathing muscles during swimming are also key in generating

154   forward propulsion and upper body stabilisation during the front crawl arm stroke

155   (Lomax, Tasker & Bostanci, 2014; Lomax, Tasker & Bostanci 2015; Nuber, Jobe,

156   Perry, Moynes & Antonelli, 1986; Pink, Perry, Browne, Scovazzo & Kerrigan, 1991)

157   and could therefore contribute to IMF independently of breathing. Pre-inducing IMF

158 before swimming could therefore be used to distinguish the effects of IMF on acid-

159 base status and stroke mechanics more clearly. It is conceivable that pre-existing

160 fatigue of these dual role muscles could lead to sustained metabolic perturbations

161 during subsequent swimming similar to that occurring following the inducement of

162 the inspiratory muscle metaboreflex (McConnell & Lomax, 2006) thereby curtailing

163 swimming performance.

164

165 The aim of this study was to investigate the effect of pre-induced IMF on acid-base

166 status during middle-distance race-paced swimming and its impact on stroke

167 characteristics. We hypothesised that swimming with pre-induced IMF would

168 exacerbate the metabolic perturbations and acidosis during subsequent swimming,

169 increase SR and f_r, and slow swimming time.

170

171 **Methods**

172 *Experimental approach to the problem*

173 Swimmers completed one pulmonary familiarisation and three swimming trials in an

174 indoor heated (mean ± SD: 27.2 ± 0.3 °C) 25-m swimming pool. The first trial was

175 used to determine maximum-effort 400-m front crawl swimming time. The remaining

176 two trials were administered in a counterbalanced order and consisted of a single 400-

177 m broken (8 × 50-m) front crawl swim. One of the swims was preceded by the

178 inducement of IMF (IMF trial) and the other was not (control trial). Swimming

179 characteristics (SR, f_r, swim time) were recorded during each swim and maximal

180 inspiratory and expiratory mouth pressures (PImax and PEmax, respectively), pH,

181 bicarbonate (HCO$\bar{3}$), partial pressures of oxygen and carbon dioxide (PO$_2$ and PCO$_2$,

182   respectively) and blood lactate (b[lac]) were measured pre, mid (except PEmax and

183   b[lac]) and after each experimental swim from the earlobe.

184

185   *Participants*

186   Twenty-one well-trained club level swimmers volunteered for this study (see Table 1

187   for their descriptive characteristics). All provided written informed consent and

188   testing conformed to the Declaration of Helsinki. Institutional ethical approval was

189   received before the start of the study.

190

191   **Table 1 about here**

192

193   *Pulmonary familiarisation*

194   PImax (measured from residual volume) and PEmax (measured from total lung

195   capacity) were determined with the nose occluded and using a hand-held respiratory

196   pressure meter whilst standing on poolside (RPM, Micro Medical Ltd, Kent, UK).

197   Reliability was deemed present when the three highest manoeuvres from a series of

198   manoeuvres were within 10 cmH$_2$O. Forced vital capacity (FVC) and forced expired

199   volume in the first second of exhalation (FEV$_1$) were recorded for descriptive purposes

200   using a digital spirometer (Micro Spirometer, Micro Medical Ltd, Kent, UK).

201   Participants completed a minimum of three satisfactory manoeuvres with the highest

202   recorded.

203

Swimming trials

205 All swimming trials were initiated from a push start, completed using the front crawl

206 stroke, and occurred on separate days. The time taken to complete the maximum effort

207 (race-paced) 400-m swim was used to determine the pace and hence target times for

208 the two subsequent broken 400-m swimming trials, which consisted of 8 x 50-m at

209 400-m race pace. Each 50-m partial was separated by 10 s of rest, with the exception

210 that a 60 s rest separated the 4th and 5th 50-m partial so that PImax could be measured

211 and a capillary blood sample taken (modified from Aujouannet, Bonifazi, Hintzy,

212 Vuillerme & Rouard, 2006).

213

214 PImax was assessed before the inducement of IMF in the IMF trial (baseline$_1$), before

215 the 1st 50-m partial (baseline$_2$), following the 4th 50-m partial (mid-swim) and after the

216 final 50-m partial (end-swim). PEmax was assessed at baseline$_2$ and end-swim only.

217 Additionally, in a sub-set of 13 participants, b[lac], pH, HCO_3^-, PO_2 and PCO_2 were

218 measured from a capillary blood sample (60-80 μl) taken by micro puncture from a

219 hyperaemic earlobe and collected in heparinized glass capillaries. b[lac] was diluted

220 (LKM41 lactate solution, Dr. Lange, Berlin, Germany) and analysed using a

221 photometer (MINI8, Dr. Lange, Berlin, Germany) at baseline$_2$ and end-swim. pH,

222 HCO_3^-, PCO_2 and PO_2 were analysed using a blood gas analyser (ABL5, Radiometer,

223 Copenhagen, Denmark) at baseline$_1$, baseline$_2$, mid-swim and end-swim.

224 Additionally, dyspnoea (Borg CR10 scale) was assessed before and at end-swim.

225

226 To measure clean swimming SR and *fr*, a 12.5-m zone was identified from the middle

227 of the pool and stroke characteristics were measured in this segment only. SR was

228 determined as the number of strokes divided by the time taken to swim through the

229   12.5-m zone (Hz, measured using a stop watch) and multiplied by 60 to convert to

230   cycles$\cdot$min^{-1} (Lomax et al., 2013). *fr* was calculated as the number of times the head

231   turned during the 12.5-m zone divided by time taken to cover the 12.5-m zone

232   multiplied by 60 (Lomax et al., 2013). The values were then averaged over the first

233   and second 25-m length of each 50-m partial to give a mean value per 50-m partial.

234   Additionally, the time taken (s) to complete each 50-m partial was recorded using a

235   stop watch.

236

237   ***Inspiratory muscle fatigue inducement***

238   IMF was induced using a commercially available inspiratory muscle trainer

239   (POWERbreathe, HaB International, Southam, UK). With the nose occluded,

240   participants generated 70% of their PImax using a duty cycle of 0.60 (three seconds

241   for inspiration and two seconds for expiration) and an *fr* of 12. Participants coordinated

242   breathing pattern with a metronome and continued with this pattern until it could no

243   longer be maintained for three consecutive breaths. Participants then continued for a

244   further minute after which PImax was re-assessed and recorded as baseline$_2$. This has

245   been shown to result in a fall in PImax of 17-25% in swimmers (Lomax & Castle,

246   2011; Lomax Tasker & Bostanci, 2014), which is consistent with the magnitude (8-

247   29%) observed following 100-m-400-m front crawl swimming (Brown & Kilding,

248   2011; Jakovljevic & McConnell, 2009; Lomax & McConnell, 2003; Lomax et al.,

249   2013; Thomaidis et al., 2009).

250

251 *Data analysis*

252 Data were assessed for normality using a Shapiro-Wilk test. The impact of pre-induced

253 IMF on PImax, b[lac], pH, $HCO\overline{3}$, PO_2, PCO_2, between basleine$_1$ and basleine$_2$ (IMF

254 trial) were assessed using paired samples t-tests. Two-way (condition x time) repeated

255 measures ANOVA assessed for differences in swim time, SR, *fr*, b[lac], pH, $HCO\overline{3}$,

256 PO_2, PCO_2, PImax and PEmax between IMF and control trials between baseline$_2$, mid-

257 swim and end-swim. Post hoc analyses were undertaken using repeated measures

258 ANOVA's with Bonferroni adjustments and paired samples t-tests. Wilcoxon-Signed

259 rank tests were used to assess post swim dyspnoea between trials expressed as delta

260 change (Δ) from baseline$_1$.

261

262 Effect sizes were calculated using Cohen's *d* for parametric data, whereby 0.2 was

263 deemed small, 0.6 moderate, 1.2 large, 2.0 very large and 4.0 extremely large

264 (Hopkins, Marshall, Batterham & Hanin, 2009). For non-parametric data, effect sizes

265 were calculated as r, whereby r is the z score divided by the square root of the total

266 number of observations. A value of .1 is deemed small, .3 medium and .5 and above

267 large (Field, 2013). Significance was set as $P \leq 0.05$ and statistical analyses were

268 undertaken using IBS SPSS statistics version 24 (Chicago, Illinois, USA). Unless

269 otherwise stated data are presented as mean ± SD.

270

271 **Results**

272 Pre-inducing IMF caused PImax to fall by 19 ± 10 cmH$_2$O from 130 ± 30 cmH$_2$O to

273 111 ± 30 cmH$_2$O (P < 0.001, *d* = 0.67). Time (F = 15.230, P < 0.001) and condition (F

274 = 9.911, P = 0.08) were both significant for PImax and an interaction was observed (F

11

275 = 3.872, P = 0.029). Swimming with pre-induced IMF did not cause PImax to fall

276 further. When IMF was not pre-induced, PImax fell by mid-swim (P < 0.001, $d = 0.87$)

277 but did not fall any further by end-swim. PEmax was unaffected by condition but was

278 lower after swimming in both trials (F = 24.704, P < 0.001: Figure 1).

279

280 **Figure 1 about here**

281

282 Swimming time slowed during the latter part of each trial (F = 18.827, P < 0.001) and

283 there was an interaction between time and condition (F = 3.579, P = 0.001; Table 2).

284 SR and *fr* were affected by condition (SR: F = 29.291, P < 0.001; *fr*: F = 14.93, P =

285 0.001) and time (SR: F = 3.958, P = 0.044; *fr*: F = 5.820, P = 0.001) but no interactions

286 were observed (Table 2).

287

288 **Table 2 about here**

289

290 Mean SR and *fr* were correlated in both control (r = 0.690, P = 0.001) and IMF (r =

291 0.675, P = 0.001) trials, as were mean swimming time and SR (control: r = -0.722, P

292 < 0.001; IMF: r = -0.762, P < 0.001), and mean swimming time and *fr* (control: r = -

293 0.528, P = 0.014; IMF: r = -0.534, P = 0.013). The change in PImax was not correlated

294 with SR, *fr* or swimming time (P > 0.05).

295

296 The inducement of IMF increased pH (t = -4.129, P = 0.001), decreased PCO_2 (t =

297 4.813, P < 0.001) and decreased HCO_3^- (t = 3.498, P = 0.004) from $baseline_1$ to

298 $basleine_2$, but had no impact on PO_2 (P > 0.05). Main effects were observed for b[lac]

299 (time: F = 18.374, P = 0.001; condition: 191.406, P < 0.001), HCO_3^- (time: F =

300   154.314, P < 0.001; condition: F = 19.563, P = 0.001), PCO_2 (time: 6.218, P = 0.007;

301   condition: F = 19.138, P = 0.001) and pH (time: F = 110.016, P < 0.001). Interactions

302   were observed between time and condition for b[lac] (F = 12.659, P = 0.004), pH (F

303   = 19.930, P < 0.001), HCO_3^- (F = 4.432, P = 0.024) and PCO_2 (F = 5.381, P = 0.012).

304   A moderate correlation (r = 0.450, P = 0.024) was observed in *fr* and PCO_2 for Δ

305   between mid- and end- swim (pooled data). PO_2 was unaffected by time or condition

306   (P > 0.05) (Table 3). Dyspnoea was higher (z = -2.910, P = 0.003, r = -0.46) following

307   the IMF trial (IMF trial: 8.1 ± 1.3; control trial: 7.4 ± 2.3).

308

309   **Table 3 here**

310

311   **Discussion**

312   It was our aim to assess the impact of pre-induced IMF on acid-base status during

313   middle-distance race-paced swimming and stroke characteristics. Pre-inducement of

314   IMF caused respiratory alkalosis, which complicates the interpretation of the findings.

315   Nevertheless, the main observations were: 1) pre-induced IMF exacerbated the

316   metabolic perturbations occurring during subsequent swimming; 2) IMF, whether pre-

317   induced or propulsion induced, is unlikely to be attributed to respiratory acidosis; 3)

318   pre-induced IMF slowed swimming time; and 4) *fr* increased in both trials but was not

319   correlated with IMF.

320

321   The pre-inducement of IMF caused PImax to fall by 14%, which is consistent with

322   that reported in response to swimming *per se* (Jakovljevic & McConnell, 2009; Lomax

323   et al., 2012; Lomax et al., 2015; Thomaidis et al., 2009). The IMF protocol

324   significantly increased pH, reduced HCO_3^- and caused a non-significant fall in PCO_2

13

325 (Table 3). This meant that swimmers began the swim in a state of mild hypocapnia

326 and alkalosis. It is likely that this state developed because of hyperventilation arising

327 from the increase in breathing depth associated with the IMF protocol and not IMF

328 *per se* (Chin et al., 2007; Costanzo, 2010; Le Blanc, Parolin, Jones & Heigenhauser,

329 2002). It could therefore be argued that as the method we adopted to induce IMF

330 created a breathing pattern (hyperventilation) antithetical to that in front crawl

331 swimming (hypoventilation), it may not reflect the metabolic milieu accompanying

332 swimming-induced IMF. Indeed, the observation that swimmers began the swim in

333 the IMF trial with lower HCO_3^- levels means that their ability to buffer H^+ during

334 subsequent swimming was likely compromised. This could be exacerbated by a

335 slowing of pulmonary oxygen uptake kinetics and microvascular blood flow, which

336 have been observed in the presence of hyperventilation-induced hypocapnic alkalosis

337 (Chin et al., 2007; Chen, Heigenhauser, Paterson & Kowalchuk, 2013). Contrastingly,

338 when CO_2 is added to an inspirate during hyperventilation to prevent hypocapnic

339 alkalosis, pH and PCO_2 are prevented from falling and pulmonary oxygen uptake

340 kinetics and microvascular blood flow are improved (Chin et al., 2013). Had a

341 hypercapnic gas mixture been administered during IMF inducement in the current

342 study, the resultant respiratory alkalosis would have been prevented. This approach

343 should be adopted in future.

344

345 The control trial did not suffer from this same limitation as swimmers began the

346 control trial swim with normal pH, HCO_3^- and PCO_2 values (Table 3). By mid-swim

347 (i.e. 200-m) however, PImax had fallen by 24% indicating the presence of IMF (Figure

348 1). At this same time point, pH had become mildly acidic (i.e. < 7.35), HCO_3^- levels

349 had fallen (Table 3), but there was no evidence of hypercapnia or respiratory acidosis.

14

350   This pattern of change in pH, HCO_3^- and PCO_2 during swimming in the control trial

351   was similar to the pattern observed in response to swimming in the IMF trial, but the

352   absolute values differed (Table 3).

353

354   Pre-inducing IMF did exacerbate the metabolic perturbations (e.g. pH, HCO_3^-, PCO_2

355   and b[lac]) occurring during the swim. Despite reducing the buffering capacity of H^+

356   in this trial it did not translate into a greater magnitude of IMF (Figure 1). This does

357   not mean that a lowered buffering capacity was without consequence. As the

358   progressive fall in pH and HCO_3^- (Table 4) was greater in the IMF trial, the slower

359   swimming times (Table 2) in this trial might be indicative of a reduced ability to buffer

360   H^+ generated during swimming.

361

362   Acidosis can be respiratory or metabolic in origin, or can be a combination of both

363   (Brooks et al., 2000). The progressive fall in pH without concomitant hypercapnia

364   makes it likely we detected its metabolic origin. Moreover, the development of IMF,

365   whether pre-induced or propulsion-induced, cannot be attributed to ventilatory

366   impairment causing respiratory acidosis; at least not when the breathing pattern is

367   optional and fr is ≥ 25 breaths·min^{-1}. However, there are a number of caveats to this

368   interpretation.

369

370   It is known that exercise can increase carbon dioxide storage in the body and this

371   storage capacity is diminished at higher exercise intensities leading to an increase in

372   carbon dioxide excretion (Jones & Jurkowski, 1979). The relatively short duration of

373   the 50-m repeat swims and the fact that they were not maximal effort swims but were

374   instead based on 400-m race pace, could be incompatible with the detection of

375 hypercapnia. Additionally, it took approximately 20-30 s to obtain blood samples for

376 analysis during which time breathing was very intense. If a small swimming-induced

377 increase in PCO_2 did occur, it could be reversed during this period resulting in the

378 measured PCO_2 failing to change. Lastly, failure to observe hypercapnia does not

379 necessarily mean that ventilation poses no limitation during front crawl swimming, or

380 that hypercapnia does not occur. It may be that a ventilatory limitation was not

381 sufficient to cause respiratory acidosis. In support of this, Ušaj (1999) found

382 ventilation to be less effective at compensating for metabolic acidosis during 400-m

383 race-paced front crawl swimming (pH of 7.09 ± 0.09, PCO_2 of 4.0 ± 0.5 kPa) compared

384 to maximal effort kayak paddling (pH of 7.17 ± 0.05, PCO_2 of 3.6 ± 0.2 kPa) but this

385 failed to result in hypercapnia in the former. We observed a similar pattern and also

386 found the Δ (increase) in f_r between mid- and end- swim (mid-swim being the first

387 time point where IMF was evident in both trials) was correlated with Δ (fall) in PCO_2

388 (r = 0.450), suggesting that the increase in f_r, which occurred in both trials, partly

389 contributed to the prevention of hypercapnia. However, the small coefficient of

390 determination (20%) indicates that prevention of hypercapnia was not the primary

391 cause for the increase in f_r.

392

393 It has been proposed that stimulation of thin fibre afferents within fatigued inspiratory

394 muscles create an additional breathing stimulus. This increase in central respiratory

395 drive to the weakened inspiratory muscles is discernible as dyspnoea (Gallagher et al.,

396 1985; Mador & Acevedo, 1991; Sliwinski et al., 1996) and the resultant tachypnoea is

397 thought to be directed at minimising this sensation (Gallagher et al., 1985). Post swim

398 dyspnoea was higher than baseline[1] in both trials and was higher still in the IMF trial,

399 but no interaction was observed between condition and time for f_r. This lack of

400 interaction is not surprising given that no interaction was observed between time and

401 condition for f_r.

402

403 In previous studies we have shown that 20 s front crawl sprinting following the

404 inducement of IMF leads to an increase in SR but not f_r (Lomax et al., 2014) and that

405 IMF magnitude is not correlated with f_r during the four swimming strokes (Lomax et

406 al., 2012). Although fr and SR were correlated in the current study (P < 0.001), the

407 coefficient of determination was only 46-48%. Thus, the cause of the increase in f_r that

408 may or may not accompany IMF in swimming is multifactorial and is likely impacted

409 by the ventilatory drive to maintain normocapnia, a need to alleviate dyspnoea, and an

410 increase in SR if SR does increase: SR did not increase in the present study but has

411 been shown to previously (Lomax & Castle, 2011).

412

413 It is important to recognise here that the breathing muscles in swimming are also key

414 propulsion muscles. For example, the pectoralis major, latissimus dorsi, upper

415 trapezius, and serratus anterior, are activated during deep inspirations (Kendall et al.,

416 2005) and front crawl swimming (Nuber et al., 1986; Pink et al., 1991). We have

417 shown that 20 s arms only front crawl sprinting is sufficient to induce pectoralis major

418 fatigue but not latissimus dorsi fatigue (Lomax et al., 2014). In contrast, latissimus

419 dorsi and pectoralis major fatigue have been reported following 100-m (63 ± 2 s) full

420 stroke front crawl swimming (Stirn et al., 2011). Given that the measurement of PImax

421 is holistic in nature, the pressure recorded at the mouth will reflect the collective

422 activity of all the muscles recruited (Gibson, 1995). This will include contributions

423 from muscles vital in creating forward propulsion and trunk stability (Pink et al., 1991)

424 and fatigue of these muscles is evident in the PImax manoeuvre (Lomax et al., 2015).

425

426   It is therefore possible that the propulsion and stabilisation requirements of the dual

427   role muscles during swimming caused fatigue of these muscles and this in-turn was

428   evident in the holistic assessment of PImax and PEmax. The presence of IMF and

429   expiratory muscle fatigue therefore does not automatically indicate a breathing

430   induced origin for fatigue. Rather, it could be propulsion induced. If this is the case,

431   the aetiology of IMF in the present study differed between the two trials: breathing

432   induced in the IMF trial and propulsion induced in the control trial. However, the net

433   effect was the same: the dual role breathing and propulsion/trunk stabilisation muscles

434   experienced fatigue. As we did not measure electromyography of the dual role muscles

435   or their force output while swimming, we are unable to identify exactly which muscles

436   were affected.

437

438   **Conclusion**

439   Swimming with pre-induced IMF caused transient respiratory alkalosis, which was

440   reversed by mid-swim. Pre-induced IMF was associated with a greater level of

441   metabolic perturbation including reduced H^+ buffering capacity, increased acidosis

442   during subsequent swimming and slowed swimming times. However, the overall

443   magnitude of IMF experienced was similar between IMF and control trials and the

444   increase in f_r accompanying both trials was not correlated with IMF but was correlated

445   with PCO_2.

446

447   The cause of acidosis and IMF, whether pre-induced or propulsion induced, could not

448   be attributed to ventilatory impairment and therefore must have been metabolic in

449   origin. Given the dual role function of the affected muscles we think it likely that IMF

450   occurring in response to swimming reflects propulsion induced fatigue during 400-m

451   crawl swimming.

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623   Figure 1. PImax (A) and PEmax (B) immediately before the swim (baseline$_2$), mid-

624   swim and end-swim in control (filled bars) and IMF (open bars) trials.

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626   Notes: \\(P < 0.01) different to control trial at given time point; **(P < 0.01) different

627   to baseline$_2$ within trial. See text for abbreviations.

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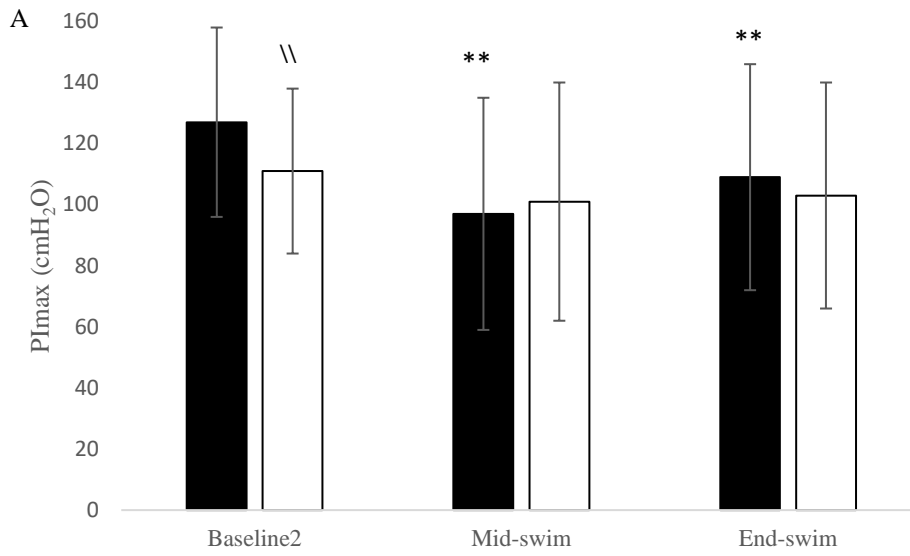643

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PImax (cmH$_2$O)

100

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Baseline2   Mid-swim   End-swim

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B

200

180

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PEmax (cmH$_2$O)

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100

80

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**   **

Baseline2   End-swim
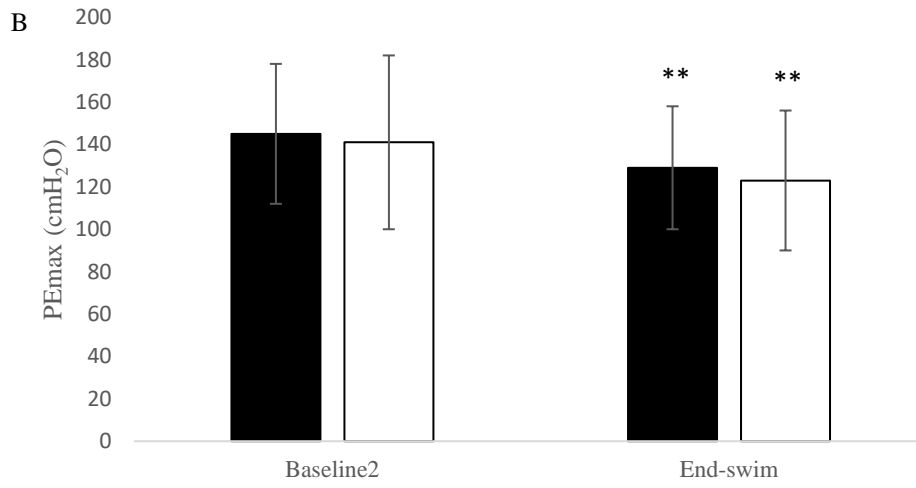
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671   Table 1. Descriptive characteristic of swimmers at the start of the study: mean $\pm$ SD

	Measure	Group	Males	Females
673	n	21	12	9
674	Age (years)	22 ± 3	22 ± 3	22 ± 3
675	Body mass (kg)	71.2 ± 12.7	77.6 ± 13.1	62.3 ± 4.9**
676	Stature (m)	1.75 ± 0.10	1.81 ± 0.09	1.65 ± 0.07**
677	FVC (l)	5.26 ± 1.11	6.04 ± 0.74	3.69 ± 0.39**
678	FEV_1 ($l \cdot s^{-1}$)	4.42 ± 0.91	4.98 ± 0.77	4.21 ± 0.42**
679	FEV_1/FVC (%)	85 ± 7	82 ± 6	88 ± 7
680	PImax (cmH_2O)	134 ± 29	136 ± 32	132 ± 27
681	PEmax (cmH_2O)	154 ± 36	164 ± 42	138 ± 24

682   Notes. **($P \leq 0.01$) different to males. See text for abbreviations.

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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)

		50-m distance and partial number							
		50-m	100-m	150-m	200-m	250-m	300-m	350-m	400-m
Parameter	Trial	1	2	3	4	5	6	7	8
Time (s)	control	37.1 ± 8.1	39.0 ± 7.3**	39.7 ± 6.7**∫∫	40.3 ± 7.3**∫∫	38.3 ± 7.2	39.8±7.1*∫∫	41.0 ± 7.2**†∫∫§§	40.9 ± 7.3**∫∫§§
	IMF	36.4 ± 6.7	39.0 ± 6.7**	40.1 ± 6.5**∫∫⌋	40.7 ± 7.3**∫∫	38.7±7.0	40.7±7.3*∫∫⌋	41.6 ± 7.3**†∫∫§§⌋	41.5 ± 7.6**∫∫§§⌋
SR	control	37 ± 9	35 ± 7	34 ± 6∫∫	34 ± 6∫	36 ± 6	35 ± 5	35 ± 5	35 ± 5
(cycles·min⁻¹)	IMF	39 ± 8†	37 ± 7	35 ± 5∫∫	35 ± 5∫∫	38 ± 6	36 ± 5	36 ± 6	37 ± 6
f_r	control	25 ± 9	26 ± 7§∫	28 ± 9*	27 ± 7§§	28 ± 7*	29 ± 6**	28 ± 5*	29 ± 6*
(breaths·min⁻¹)	IMF	26 ± 9	29 ± 8**	30 ± 8**	30 ± 7**	30 ± 7*	31 ± 7**	31 ± 6**	32 ± 7**

Note. *($P < 0.05$) **($P < 0.01$) different to 1st 50-m; †($P < 0.05$) different to 2nd 50-m; ∫($P < 0.05$) ∫∫($P < 0.01$) different to 5th 50-m; §($P < 0.05$) §§($P < 0.01$) different to 6th 50-m; ⌋($P < 0.05$) ⌋⌋($P < 0.01$) different to control at time point. See text for abbreviations.

715 Table 3. Blood parameters measured before, mid-swim (200-m) and end-swim (400-m) per trial: group mean $\pm$ SD (n = 13)

Parameter	Trial	Baseline$_1$	Baseline$_2$	Mid-swim	End-swim
b[lac] (mmol·l^{-1})	control	/	1.4 ± 0.2	/	8.2 ± 2.4**
	IMF	/	1.6 ± 0.5	/	10.1 ± 1.9**⌋⌋
pH	control	/	7.44 ± 0.03††	7.32 ± 0.04**	7.27 ± 0.04**††
	IMF	7.43 ± 0.01	7.48 ± 0.04§§⌋††	7.30 ± 0.04§§**⌋	7.23 ± 0.05**§§⌋⌋††
HCO$_3^-$ (mmol·l^{-1})	control	/	24.1 ± 1.4††	18.9 ± 2.7**	16.4 ± 2.7**††
	IMF	24.2 ± 1.2	22.8 ± 1.5§§††⌋⌋	16.8 ± 2.6**§§⌋⌋	13.8 ± 2.3**§§††⌋⌋
PCO$_2$ (kPa)	control	/	4.8 ± 0.4	5.0 ± 0.5	4.9 ± 0.5
	IMF	4.9 ± 0.2	4.1 ± 0.7§§†⌋⌋	4.7 ± 0.5*⌋	4.5 ± 0.5⌋⌋
PO$_2$ (kPa)	control	/	10.9 ± 1.2	11.8 ± 2.4	11.6 ± 1.9
	IMF	11.9 ± 2.5	11.5 ± 3.7	12.0 ± 1.9	11.7 ± 1.2

727 Note. §($P < 0.05$) §§($P < 0.01$) different to baseline$_1$ (within-trial comparison with baseline$_2$); *($P < 0.05$) **($P < 0.01$) different to baseline$_2$; †($P <$

728 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.