Title: Inspiratory muscle fatigue affects latissimus dorsi but not pectoralis major activity during arms only front crawl sprinting

Running head: Inspiratory muscle fatigue and swimming

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ABSTRACT

The purpose of this study was to determine whether inspiratory muscle fatigue affects the muscle activity of the latissimus dorsi and pectoralis major during maximal arms only front crawl swimming. Eight collegiate swimmers were recruited to perform two maximal 20 s arms only front crawl sprints in a swimming flume. Both sprints were performed on the same day and inspiratory muscle fatigue was induced 30 minutes after the first (control) sprint. Maximal inspiratory and expiratory mouth pressures (PImax and PEmax, respectively) were measured pre and post each sprint. The median frequency (MDF) of the electromyographic signal burst was recorded from the latissimus dorsi and pectoralis major during each 20 s sprint along with stroke rate and breathing frequency. MDF was assessed in absolute units (Hz) and then referenced to the start of the control sprint for normalization. After inspiratory muscle fatigue inducement stroke rate increased from 56 ± 4 to 59 ± 5 cycles·min^{-1} and latissimus doris MDF fell from 67 ± 11 Hz at the start of the sprint to 61 ± 9 Hz at the end. No change was observed in the MDF of the latissimus dorsi during the control sprint. Conversely, the MDF of the pectoralis major shifted to lower frequencies during both sprints but was unaffected by inspiratory muscle fatigue. As the latter induced fatigue in the latissimus doris, which was not otherwise apparent during maximal arms only control sprinting, the presence of inspiratory muscle fatigue affects the activity of the latissimus dorsi during front crawl sprinting.

Key words: electromyography, swimming kinematics, fatigue

INTRODUCTION

There is now a substantial body of evidence demonstrating that the global inspiratory musculture, which includes the diaphragm, external intercostals, scalene muscles and sternomastoids (42) amongst others, is susceptible to fatigue during maximal (24,38) and sub-maximal (16,25,26) front crawl swimming. Whilst it has been shown that inspiratory muscle fatigue can increase stroke rate, breathing frequency and reduce stroke length (23), it is not known if inspiratory muscle fatigue impacts the activity of the relevant musculature during the front crawl swimming stroke. Given that over 30 muscles are active during the front crawl stroke (6), identifying the relevant musculature must be done by determining which muscles have a dual function in contributing significantly to the front crawl stroke (i.e. propulsion and stabilization) and supporting inspiration.

Two muscles that meet these criteria are the latissimus dorsi and the pectoralis major (6,19,31,32). Although other muscles such as the serratus anterior and sternocleidomastoid also fulfill the above requirements (6,31,32), the latissimus dorsi and pectoralis major are dominant in producing force during the underwater pull through phase (29) and hence in overcoming the resistance to forward movement. Moreover, electromyography (EMG) recordings have shown that along with the rectus abdominus and gluteus maximus, the latiismus dorsi is one of the three most active front crawl muscles (6) and has been labeled ‘the workhorse’ of the upper body during swimming (29).

As well as identifying muscle activity patterns (6), surface EMG can be used to examine muscular fatigue (10). Specifically, the power spectral density and amplitude of the EMG signal energy can be assessed and inferences made about fatigue (10). Changes in the frequency content of the signal are however, believed to be more sensitive to fatigue than amplitude changes (33). To separate the signal into its frequency components the mean or median frequency (MDF) of the signal is calculated, although the MDF is the preferred method as it is less sensitive to noise and signal aliasing (10).

Terrestrial studies have shown that the MDF shifts to a lower frequency in response to fatiguing dynamic and sustained muscle contractions of the quadriceps, hamstrings and biceps brachii (27,33), while in swimming it has been shown that the mean frequency of the latissimus dorsi, pectoralis major, triceps brachii and biceps brachii decrease with fatigue (37). This decrease is evidenced as a leftward shift in the power spectral density

curve and has been attributed to changes in motor unit synchronization (13), altered sarcolemma characteristics (20) such asa slowing of conduction velocity brought about by the accumulation of metabolic by-products (30), and altered central drive (41).

Given the importance of the latissimus dorsi and pectoralis major to the front crawl stroke and in supporting increased inspiratory activity, the aim of this study was to examine whether or not inspiratory muscle fatigue induced fatigue in the latissimus dorsi and pectoralis major muscles, and if it did, the impact of such fatigue on stroke kinematics during sprint swimming. Such information could potentially aid in the development of appropriate training interventions. We hypothesized that inspiratory muscle fatigue would induce fatigue in the latissimus dorsi and pectoralis major muscles as evidenced by a fall in latissimus dorsi and pectoralis major MDF during maximal arms only front crawl swimming, and would increase stroke rate and breathing frequency.

METHODS

Experimental approach to the problem

Inspiratory muscle fatigue has been shown to occur in response to maximal (24,38) and sub maximal (25,26) swimming, and to alter stroke characteristics during fixed-velocity swimming (23). Some of the upper body muscles have a dual function during front crawl swimming by supporting both breathing and propulsion. Consequently it is possible that inspiratory muscle fatigue might directly fatigue one or more of these muscles. In turn, fatigue of the dual-function muscles might alter stroke characteristics. To test this we selected two of the most dominant upper body front crawl muscles, the latissimus dorsi and pectoralis major (6,31,32), which are also key in assisting breathing (19), and recorded EMG from these muscles during two maximal 20 s arms only sprints: one following the inducement of inspiratory muscle fatigue and one without pre-induced inspiratory muscle fatigue. To avoid the possibility of an inspiratory muscle fatigue induced compensatory increase in leg kick the legs were immobilized and swimmers used only their upper bodies to exert maximum effort. However, the possibility of an inspiratory muscle fatigue-induced shoulder girdle compensation could not be eliminated. The MDF of the EMG recordings was subsequently determined as this is sensitive to fatigue and is known to fall in the presence of fatigue (27,33). Each sprint was also recorded for the determination of stroke rate and breathing frequency.

Subjects

Eight collegiate swimmers (6 males and 2 females), with an age range of 18-33 years volunteered for this study. Mean ± SD for age, body mass and stature were 22.0 ± 5.5 years, 79.0 ± 7.5 kg, and 176.8 ± 8.0 cm. Barometric pressure, air temperature, water temperature and humidity were 770.8 ± 7.8 mmHg, 25.2 ± 10.8^{o}C, 28.0 ± 0.1^{o}C, and 72.2 ± 9.7%, respectively. All swimmers were well trained colligate swimmers with a seasonal personal best of 139 ± 52.3 s for 200 m front crawl. All were well hydrated prior to, and avoided training or competition for at least 24 hours before, testing. None-had any history of cardio-pulmonary disease. Participants provided written informed consent and local ethical approval was obtained from the Biosciences Research Ethics Committee, University of Portsmouth before the start of the study.

Procedures

Participants attended at least one pulmonary familiarization session and then completed two experimental sprint tests on a separate day to pulmonary familiarization. In the pulmonary familiarization session, standing maximal inspiratory mouth pressure (PImax) and standing maximal expiratory mouth pressure (PEmax) maneuvers were practiced (RPM, Micro Medical, Rochester, UK) and technique perfected. The nose was occluded throughout each maneuver and a 60 s rest period separated each effort. PImax was measured from residual volume and PEmax from total lung capacity. Reliability in this session was deemed present when three technically proficient maneuvers within 5 cmH_2O were obtained (24). The highest PImax and PEmax values in this session and the baseline values of the experimental sprint tests were used to assess the overall reliability of PImax and PEmax. Intraclass correlation coefficients (ICC's) demonstrated excellent reliability for both PImax (ICC=0.994) and PEmax (ICC=0.997).

On a separate day to the pulmonary familiarization session participants completed two experimental 20 s arms only maximal FC sprints in a swimming flume (SwimEx 600-T Therapy Pool, length 4.2 m, width 2.3 m and depth 1.5 m). One sprint occurred in the presence of pre-induced inspiratory muscle fatigue (IMF sprint) and the other without pre-induced inspiratory muscle fatigue (control sprint). EMG was recorded from the right latissimus dorsi and pectoralis major throughout each sprint. To ensure that EMG sampling sites remained identical between sprints participants completed both sprints on the same day. To avoid any potential residual inspiratory muscle fatigue affecting the control sprint, the latter was administered before the inspiratory muscle fatigue sprint. Thus, the control and IMF sprints were neither counterbalanced nor randomized. To perform each 20 s sprint on different days would have necessitated the removal and re-application of the EMG

electrodes, which might reduce the reproducibility of the EMG signal because of slight electrode position differences (27). We felt that this would have been a greater limitation and aimed to eradicate as many confounding variables as possible from masking any true effect capable of detection within the EMG signal.

As the lower body muscles e.g. gluteus maximus, rectus femoris, semitendinosus and gastrocnemius contribute substantially to the front crawl stroke (2,3,6,15), the legs were immobilized to exclude the possibility of increased leg activity compensating for inspiratory muscle fatigue. The turbine in the swimming flume was switched off throughout and the legs rested on a padded support bar running across the width of the flume. The height of support bar was adjusted per swimmer to ensure that each participant's thighs rested across the bar whilst ensuring that the hips reflected the swimmer's usual self-determined hip position. Each swimmer was tethered so that while maximally sprinting in an unfatigued state the swimmer moved neither forward nor backward but remained as stationary as possible.

Before each 20 s sprint swimmers performed standing PImax and PEmax maneuvers on poolside (baseline). Following the measurement of baseline PImax and PEmax participants entered the flume in preparation for the sprint (approximately 60 to 120 s delay). In the case of the IMF sprint baseline refers to the value immediately after the inducement of inspiratory muscle fatigue. The assessment of post sprint PImax and PEmax was completed on poolside within 60 s of sprint cessation. Furthermore, PImax was always measured before PEmax whether at baseline or post sprint. Each sprint was recorded (digital camera interfaced to ShowBiz software, ArcSoft USA) for subsequent analysis of stroke rate and breathing frequency. Stroke rate was firstly converted to cycles per second by dividing the total number of stroke cycles by 20 (swim time in seconds), and was then multiplied by 60 to convert to cycles per minute ($cycles \cdot min^{-1}$). To calculate breathing frequency the total number of breaths taken was divided by 20 and then multiplied by 60 to convert to breaths per minute ($breaths \cdot min^{-1}$) (26).

A thirty minute rest separated the end of the control sprint and the start of inspiratory muscle fatigue inducement (PImax after this rest period was re-measured and was not significantly different from the baseline control sprint value). A commercially available inspiratory muscle trainer (POWERbreathe, HaB International, Southam, United Kingdom) was used to induce inspiratory muscle fatigue. With the nose occluded, participants sat on a padded bench to the side of the flume. The one-way inspiratory value inside the trainer was set to open

when participants generated 70% of their PImax as determined by the highest PImax value of the experimental swim session (no load was presented to the expiratory muscles). A duty cycle of 0.60 was used (three seconds for inspiration and two seconds for expiration) and a breathing frequency of 12 breaths·min^{-1} adopted. Participants coordinated inspiration and expiration via a bespoke computer metronome and continued this breathing pattern until it could not be maintained for three consecutive breaths despite strong verbal encouragement. Participants then continued for a further minute (1249 ± 596 s) after which PImax was measured to confirm the presence of inspiratory muscle fatigue. We have shown previously that this loading regime produces a reduction in PImax of around 17-19% (23,28), which is consistent with the 11-27% fall in PImax reported following front crawl swimming (16,25,26,38).

Electromyography data collection

Surface EMG was recorded on the right side of the body. The latissimus dorsi and pectoralis major were chosen because of their significant contribution to both the front crawl arm stroke (6,31,32) and to increased inspiratory muscle work (19). The electrode sites were identified and marked in accordance with the methods of Criswell (9). Specifically, the clavicular placement was used for the pectoralis major with the electrode placed at a slight oblique angle two cm below the clavicle and medial to the axillary fold. The latissimus dorsi was placed four cm below the inferior tip of the scapula halfway between the lateral edge of the torso and the spine and at a slight oblique angle (9).

The electrode sites were first shaved and then rubbed with an alcohol wipe to minimize the impedance of the skin (9). Waterproof bipolar electrodes with an interelectrode distance of two cm (Biometrics Ltd, Newport, Wales) were adhered to the prepared site using medical grade adhesive tape (Biometrics Ltd, Newport, Wales). The EMG signals were recorded with a sampling rate of 1000 Hz, preamplified (x 1000) and filtered with a bandwidth of 20-450 Hz. Input impedance was > 10^{15} Ohms and the common mode rejection ratio at 60 Hz dB was greater than 96 dB. Each electrode was connected to a portable data acquisition unit (DataLOG, Biometrics Ltd, Newport, Wales) by five meter waterproof cables. The ground electrode was fixed over the styloid process of the radius and interfaced with the data acquisition unit again via a five meter waterproof cable (Biometrics Ltd, Newport, Wales). All EMG electrode cables were fixed to the skin via medical grade adhesive tape and supported by a guide cable running across the width of the flume above the swimmer. This minimized cable movement and hence interference with the signal. The data acquisition unit was placed away from the flume on

poolside ensuring that it did not come into contact with water: the electrodes and their cables were the only EMG equipment carried by the swimmer. Each right arm stroke was marked on the data acquisition unit in real time, which permitted each right arm stroke cycle to be identified during the signal processing stage.

EMG signal processing

Version 5.06 DataLog software (Biometrics Ltd, Newport, Wales) was used for signal processing and hence the determination of MDF (10). The first and last right arm strokes were disregarded and the EMG energy of the start (strokes two to six) and end (five strokes preceding the final right arm stroke) of the sprint were identified. The mean MDF of each set of five strokes (i.e. five strokes at the start and five strokes at the end) was calculated and in the case of the control sprint served as the reference value for normalizing the EMG data (2). In addition, strokes, two, three, four and five of the control sprint were used to assess MDF reliability.

To determine the MDF of each stroke the strokes were separated into an active and inactive phase. In accordance with the methods of Stirn et al (37), the active phase was defined as the EMG signal per stroke which was at least 30% of the local (i.e. given stroke) maximum energy. As Stirn et al (37) state, this reflects regions of low and high energy rather than truly active and inactive regions. The local maximum energy was determined using the average rectified value of the EMG signal calculated using a window length of 250 ms for a given stroke. The mean MDF was then obtained by fast fourier transformation per active phase using a window length of 64 ms (based on Stirn et al [37]). This process was repeated per stroke for both the latissimus dorsi and pectoralis major. The reliability of latissimus dorsi MDF (ICC=0.989) and pectoralis major MDF (ICC=0.918) was excellent.

Statistical Analyses

All dependent variables were normally distributed (Shapiro-Wilk test) and exhibited homogeneity of variance (Levene's test). A two-way (time x sprint) repeated measures ANOVA assessed differences in PImax and PEmax values. Where differences were found planned comparisons using paired samples t-tests identified where differences lay. Differences in stroke rate and breathing frequency between trials were assessed using paired samples t-tests. In addition, 95% confidence intervals were calculated for PImax, PEmax, stroke rate, breathing frequency, latissimus dorsi MDF and pectoralis major MDF per sprint. The MDF of the latissimus dorsi and pectoralis major was assessed using two-way (time x sprint) repeated measures ANOVA's and paired

samples t-tests to identify where differences lay. Additionally, control and IMF sprint end MDF values were normalized by expressing them as a percentage of the control sprint start value and analyzed using paired samples t-tests.

Where relevant effect sizes were calculated using Cohen's *d* with an effect size of 0.2 deemed small, 0.5 medium and 0.8 and above large (7). Significance was set at $P \leq 0.05$ as *a priori*, and statistical analyses were conducted using PASW Statistics 18 (Chicago, Il, USA). Unless otherwise stated data are expressed as mean ± SD.

RESULTS

The 20 s sprint *per se* was not sufficient to induce inspiratory muscle fatigue in the control sprint or induce further decrements in PImax in the IMF sprint (F=.865, *P*=0.383). However, the inducement of inspiratory muscle fatigue reduced PImax in the IMF sprint by 25 ± 7% (*P*<0.001, *d*=3.00) confirming that the IMF sprint was undertaken in the presence of inspiratory muscle fatigue. Interestingly PImax showed a non-significant trend towards recovery in response to the IMF sprint (*P*=0.112, *d*=-0.88), although this post IMF sprint value was still lower than PImax after the control sprint (*P*=0.011, *d*=0.86) (table 1).

The inducement of inspiratory muscle fatigue did affect PEmax (F=20.156, *P*=0.003). Specifically, PEmax was 15 ± 11% lower after the inducement of inspiratory muscle fatigue when compared with the baseline value of the control sprint (*P*=0.005, *d*=0.86). This difference was still evident after the sprints (*P*=0.006, *d*=0.98) (table 1), however, the 20 s sprints *per se* caused no expiratory muscle fatigue (F=.511, *P*=0.498).

Table 1 here

The inducement of inspiratory muscle fatigue did affect latissimus dorsi MDF (F=12.686, *P*=0.009). Inspiratory muscle fatigue reduced the start MDF value of the IMF sprint (*P*=0.007, *d*=0.60) but not the end value (*P*=0.139) when compared to the control sprint (table 2). However, when the latissimus dorsi MDF was normalized by expressing as a percentage of the control start value, the end value of the IMF sprint was significantly lower than the end value of the control sprint (*P*=0.003, *d*=0.83) (figure 1). The inducement of inspiratory muscle fatigue did not affect pectoralis major MDF with the start value being the same for both

sprints (F=.378, *P*=0.558). The 20 s sprint itself did induce pectoralis major fatigue (F=8.852, *P*=0.021) (table 2) but only in the IMF sprint (*P*=0.012, *d*=0.47). Importantly, the fall in pectoralis major MDF from start to end only just missed statistical significance in the control sprint (*P*=0.053) despite a larger effect size (*d*=0.96) (table 2). However, when normalized to the control sprint start value the end MDF values were significantly lower for both the control sprint (*P*=0.032, *d*=1.41) and IMF sprint (*P*=0.049, *d*=1.21) (figure 1).

Table 2 here

Figure 1 here

Breathing frequency was unaffected by the inducement of inspiratory muscle fatigue (t=-1.263, *P*=0.247; *d*=-0.27), however, stroke rate was higher in the IMF sprint (t=-2.393, *P*=0.048, *d*=0.71) (table 1). No correlations were observed between: stroke rate and breathing frequency; the absolute or normalized latissimus dorsi/pectoralis major MDF between control and IMF sprints; the change in stroke rate and absolute or percentage change in PImax between IMF and control sprints (*P*>0.05).

DISCUSSION

The aim of the present study was to evaluate the effects of inspiratory muscle fatigue on the muscle activity of the latissimus dorsi and pectoralis major muscles during maximal arms only front crawl sprinting and the subsequent effect on stroke kinematics. Our main findings were that latissimus dorsi fatigue occurred in response to inspiratory muscle fatigue but that the 20 s sprint was insufficient to induce latissimus dorsi fatigue *per se* or exacerbate the magnitude of fatigue already present in the IMF sprint (since absolute latissimus dorsi MDF at the end of the two sprints were similar). In contrast, the 20 s sprint did induce fatigue in the pectoralis major but inspiratory muscle fatigue had no impact on the magnitude of fatigue observed. Lastly, stroke rate did increase in response to inspiratory muscle fatigue but breathing frequency did not.

The inspiratory muscle fatigue protocol adopted in the current study caused a 25% fall in PImax. Thus, the IMF sprint began in the presence of inspiratory muscle fatigue. This is similar to the magnitude of inspiratory muscle fatigue observed following high intensity 200-m front crawl swimming in Masters (27%) and Age group (22%) swimmers (25, 26). Interestingly, although PImax after the IMF sprint remained lower than pre and post control

sprint values we did observe a non-significant trend (*d*=-0.88) towards recovery during the IMF sprint (table 1). As the horizontal body position unloads the breathing muscles during front crawl (11) they operate at a more mechanically efficient length and require a smaller respiratory motor output for the desired respiratory activity (12). However such an advantage will to some extent be counteracted by pulmonary engorgement and the effects of hydrostatic pressure which compromises the force generating ability of the inspiratory muscles and reduces lung compliance (34). At first glance our data suggest that this mechanical advantage exceeded the negative effects of increased hydrostatic pressure and a horizontal body position, but it is important not to overlook breathing frequency.

It has been suggested that a reduction in breathing frequency during front crawl swimming will favor respiratory acidosis (17) and exacerbate inspiratory muscle fatigue (16). Restricting breathing frequency from 24-30 breaths·min^{-1}, which is a pattern previously observed during 100-m (5) and 200-m (26) front crawl swimming, to a frequency (10-16 breaths·min^{-1}) comparable with that observed in the current study (table 1) can increase PCO_2 (17,40) and significantly shorten the swimming distance achieved prior to volitional exhaustion (17). Moreover, while swimmers must balance breathing frequency with the oxygen requirements of the working muscles (40), a lower frequency is mechanically advantageous because breathing disrupts stroke efficiency and propulsion (22). As a result more skillful swimmers will typically utilize a lower breathing frequency than less skillful swimmers (35) with the reduction in frequency largely compensated for by a higher tidal volume (40). However, once breathing frequency falls to 15 breaths·min^{-1} or less tidal volume can no longer increase to compensate and minute ventilation declines (40). Additionally, more skilled swimmers are better at adapting breathing frequency to reflect appropriate breathing dependent blood gases (18).

As a 20 s sprint relies predominantly on the ATP-PC system and anaerobic glycolysis (4), swimmers did not need to be overly concerned with balancing breathing frequency and oxygen intake in the present study. Moreover, despite the potential disruption to the metabolic milieu with a lower breathing frequency, such a pattern increases the recovery time of the inspiratory muscles as they are less frequently required to generate high tidal volumes. As an increase in tidal volume will require greater inspiratory muscle activity (14), the natural increase in tidal volume occurring during exercise will elevate the work of breathing. In the case of front crawl swimming this effect will be exaggerated as hydrostatic compression increases the elastic and dynamic work of breathing (34). Our control and inspiratory muscle fatigue PImax sprint data (table 1) suggest

that the work of breathing was insufficient to attenuate inspiratory muscle force generating capacity during the 20 s sprints, and in the case of IMF sprint permitted some recovery. Furthermore, the trend towards PImax recovery is probably responsible for the lack of relationship between stroke rate and inspiratory muscle fatigue in the current study, which contrasts with previous research (26).

It should also be noted that the inspiratory muscle fatigue inducement regime reduced PEmax by approximately 15% (table 1). Although no load was presented to the expiratory muscles during inspiratory loading, the expiratory muscles would still be recruited to support the increased ventilatory demand (21). Our data indicate that this effort was sufficient to induce expiratory muscle fatigue. However, as expiratory muscle fatigue was neither exacerbated during the IMF sprint, nor present following the control sprint, we can conclude that 20 s of maximal arms only front crawl sprinting does not induce expiratory muscle fatigue. Furthermore, and in contrast with PImax, the expiratory muscles showed no signs of recovery from such fatigue during the IMF sprint. This probably reflects the vital contribution made by key expiratory muscles, primarily the abdominals (6) which are core trunk stabilizers and essential in supporting body roll during front crawl (29), and/or might be reflective of an inspiratory muscle fatigue induced compensatory increase in expiratory muscle activity which can persist for several hours (21).

Irrespective of the partial recovery of PImax during the IMF sprint, inspiratory muscle fatigue did shift the MDF of the latissimus dorsi to a lower frequency domain (P=0.007) at the start of the IMF sprint compared with the control sprint (table 2) and did so with a sizeable effect size (d=0.60). The negative impact of inspiratory muscle fatigue on the latissimus dorsi is supported by the observation that the normalized end MDF value was lower in the IMF sprint than the control sprint (Figure 1). As such an MDF shift is indicative of fatigue (20,27) we can conclude that inspiratory muscle fatigue does cause fatigue of the latissimus dorsi. However, we are unable to determine whether this is due to fatigue of a particular fiber type and specifically fast twitch muscle fibers. Indeed, the fatigue induced fall in MDF is greater in these muscle fibers than slow twitch muscle fibers (20). Importantly however, as the absolute end latissimus dorsi MDF value in the IMF sprint was the same as the start value in the IMF sprint (table 2), the magnitude of latissimus dorsi fatigue was not exacerbated during the sprint. In contrast, the 20 s sprint was associated with a fall in pectoralis major MDF (figure 1) and hence fatigue, but inspiratory muscle fatigue had no impact on pectoralis major MDF at any time point. Given that the latissimus dorsi is the workhorse of upper body swimming (29), it could be that any fatigue as a result of

inspiratory muscle fatigue may be more apparent in the latissimus dorsi during the sprint compared with the pectoralis major, especially as only the clavicular fibres of the pectoralis major were examined. This in-turn may have influenced our findings.

Although a reduction in MDF has been associated with a slowed conduction velocity (30) this is not necessarily the case when dynamic contractions rather than sustained contractions are employed (27,33). In support of this Masuda et al. (27) reported that vastus lateralis MDF fell during both static and dynamic leg extension exercise but conduction velocity declined in the static condition only. A reduction in conduction velocity has also been attributed to a build-up of metabolites (30). During sustained muscle contractions blood flow will be impeded and hence so too will the removal of metabolic byproducts, but this is not the case during dynamic contractions as muscular relaxation permits blood flow (27). As front crawl swimming requires dynamic contractions the reduced latissimus dorsi and pectoralis major MDF observed in the current study are therefore more likely to indicate better motor unit synchronization (13) and/or altered membrane characteristics and metabolic capacity of these muscles (20) than a slowed conduction velocity.

Interestingly the changes in latissimus dorsi and pectoralis major MDF were not correlated with stroke rate. Stroke rate is an integral part of arm coordination (1) and swimmers will adapt their stroke rate to reflect the propulsive force required, velocity, and the available power output and energetic demands of a given swimming situation (36,39). Consequently, there is a balance between stroke rate, stroke length and velocity with the stroke rate and stroke length combination varying between swimmers (8). As fatigue develops stroke rate has been reported to decrease (37,39) and increase (1). A fall in stroke rate has the advantage of prolonging the non-propulsive phase of the stroke, which permits a more efficient force production pattern, increased recovery time and stable stroke length (3,37,39). However, by increasing stroke rate the relative duration of the propulsive stroke phase is increased while the non-propulsive phase i.e. glide, catch and recovery, fall (1). This pattern compensates for a reduced ability to generate enough force to overcome the resistance to forward movement per stroke (1). Assuming that the entry phase and catch still remain effective (36), speed (or mean force production) will nevertheless be maintained even though stroke length is compromised (1).

The results of the current study support observations that stroke rate increases in the presence of fatigue (1), and in response to inspiratory muscle fatigue specifically (23). We observed a 5% increase in stroke rate (P<0.05)

when swimming with pre-induced inspiratory muscle fatigue (table 1), which was associated with a substantial effect size (d=0.71) but as already stated not latissimus dorsi nor pectoralis major MDF. It should be noted that stroke rate is the product of the coordinated action of multiple muscle groups on both sides of the body. EMG was only examined on the right side of the body in this study and only from one site per muscle group. This may limit the ability to detect direct correlations with stroke rate. Nevertheless, the increase in stroke rate most likely reflected a reduced ability to generate force per stroke. As the legs were immobilized and therefore could not compensate for inspiratory muscle fatigue, the options available to swimmers for maintaining a stationary tethered position were; 1) to increase the relative propulsive phase of each stroke by reducing the non-propulsive phase and thus increasing the frequency of stroke cycles; 2) to increase the index of coordination overlapping the propulsive phases of the left and right arm strokes; 3) a combination of both (1). As we did not assess the index of coordination we cannot confirm whether or not swimmers did this in addition to increasing stroke rate in the presence of inspiratory muscle fatigue. Furthermore, as we did not measure force from the upper body whilst sprinting we are unable to confirm whether or not force generation was actually affected by inspiratory muscle fatigue. Clearly this requires examination as it has implications for swimming performance.

It must also be noted that although we examined the muscle activity of two of the most dominant upper body muscles active during front crawl swimming (6,15), there are in excess of 25 such muscles activated during this stroke (6,15,32). Other muscles which contribute to both stroke cycle and breathing during front crawl, such as the serratus anterior and sternocleidomastoid, are worthy of investigation (6, 31,32). Furthermore, our method of inducing inspiratory muscle fatigue and in assessing inspiratory muscle force generating capacity was holistic and therefore it was not possible to target a specific inspiratory muscle over any other. Consequently, while we can confirm that inspiratory muscle fatigue was induced and did impact the starting MDF of the latissimus dorsi, we cannot state whether other muscles were fatigued and their potential role in modifying stroke rate. This lack of specificity might also explain why no relationship was observed between the increase in stroke rate and the change in latissimus dorsi or pectoralis major MDF. Indeed, the 5% increase in stroke rate in response to inspiratory muscle fatigue was not because of the magnitude of fatigue experienced by the latissimus dorsi (IMF induced) or pectoralis major (swim induced) muscles.

In conclusion, inspiratory muscle fatigue did induce fatigue in the latissimus dorsi but not the pectoralis major and did increase stroke rate but not breathing frequency. Importantly however, the increase in stroke rate

following the inducement of inspiratory muscle fatigue was not correlated with either inspiratory muscle fatigue-induced latissimus dorsi fatigue, or the swimming induced pectoralis major fatigue. This indicates that further studies are warranted to examine the reason for inspiratory muscle fatigue-induced alterations in arm stroke kinematics observed during 20 s maximal arms only front crawl swimming.

PRACTICAL APPLICATIONS

This study demonstrates that inspiratory muscle fatigue increases stroke rate during maximal arms only sprinting in well trained colligate swimmers and fatigues the latissimus dorsi but not the pectoralis major. However, the increase in stroke rate was not caused by latissimus dorsi fatigue suggesting that other dual role breathing and propulsion muscles are likely responsible for such a change. Given the potential for inspiratory muscle fatigue to disrupt stroke characteristics coaches and swimmers should include specific training designed to reduce the occurrence and/or magnitude of inspiratory muscle fatigue.

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Table 1 Group PImax, PEmax, stroke rate and breathing frequency pre and post each 20 s maximal arms only front crawl sprint

	Mean ± SD	SEM	95% CI
Control sprint			
Breathing frequency (breaths·min^{-1})	11 ± 5	1.768	8-14
Stroke rate (cycles·min^{-1})	56 ± 4	1.414	53-59
PImax pre sprint (cmH_2O)	161 ± 17	6.010	149-173
PImax post sprint (cmH_2O)	154 ± 19	6.718	141-167
PEmax pre sprint (cmH_2O)	167 ± 22	7.779	152-182
PEmax post sprint (cmH_2O)	164 ± 25	8.840	147-181
IMF sprint			
Breathing frequency (breaths·min^{-1})	13 ± 9	3.182	8-20
Stroke rate (cycles·min^{-1})	59 ± 5$^{\neg}$	1.768	56-62
PImax pre sprint (cmH_2O)	118 ± 11**§§	3.889	110-126
PImax post sprint (cmH_2O)	135 ± 25*§	8.840	118-152
PEmax pre sprint (cmH_2O)	143 ± 33**	11.669	120-166
PEmax post sprint (cmH_2O)	139 ± 26*§	9.193	121-157

$^{*}P<0.05$ $^{**}P<0.01$ different to control sprint. $^{\S}P<0.05$, $^{\S\S}P<0.01$ different to control sprint post value

$^{\neg}P<0.05$ different to control sprint.

CI = confidence interval; PImax = maximal inspiratory mouth pressure; PEmax = maximal expiratory mouth pressure; SEM = standard error of the mean

Table 2 Group latissimus dorsi and pectoralis major MDF at the start and end of each 20 s maximal arms only front crawl sprint

Condition	Start of sprint			End of sprint		
	Mean ± SD	SEM	95% CI	Mean ± SD	SEM	95% CI
Latissimus dorsi (Hz)						
control sprint	67 ± 11	3.889	59-75	64 ± 11	3.889	56-72
IMF sprint	61 ± 9**	3.182	55-67	61 ± 10	3.536	54-68
Pectoralis major (Hz)						
control sprint	71 ± 9	3.182	68-77	64 ± 5	1.768	61-67
IMF sprint	71 ± 12	4.243	63-77	66 ± 9§	3.182	60-72

** $P<0.01$ different to control sprint. § $P<0.05$ different to start of sprint

CI = confidence interval; SEM = standard error of the mean

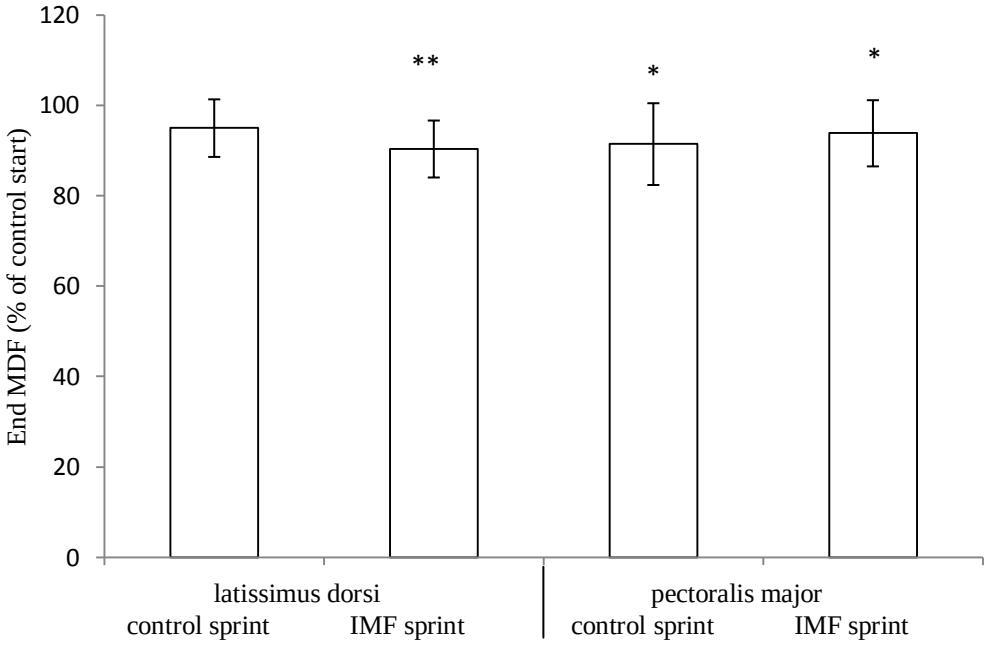
Difference between start and end pectoralis major MDF just missed statistical significance ($P=0.053$)

Legends

Fig 1 Normalized MDF of the latissimus dorsi and pectoralis major at end of each sprint. Group mean ± SD

**P<0.01, *P<0.05 different to control sprint start value

MDF = median frequency;


120
100
80
60
40
20
0
End MDF (% of control start)
**
*
*
latissimus dorsi
control sprint
IMF sprint
pectoralis major
control sprint
IMF sprint