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**Citation for published version:**

Turner, AP, Smith, T & Coleman, SGS 2008, 'Use of an audio-paced incremental swimming test in young national-level swimmers' *International Journal of Sports Physiology and Performance*, vol 3, no. 1, pp. 68-79.

**Link:**

[Link to publication record in Edinburgh Research Explorer](#)

**Document Version:**

Author final version (often known as postprint)

**Published In:**

*International Journal of Sports Physiology and Performance*

**Publisher Rights Statement:**

© Turner, A. P., Smith, T., & Coleman, S. G. S. (2008). Use of an audio-paced incremental swimming test in young national-level swimmers. *International Journal of Sports Physiology and Performance*, 3(1), 68-79.

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## **Use of an audio-paced incremental swimming test in young national-level swimmers**

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Running title: Audio-paced swimming test

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Submission type: Original investigation

Abstract word count: 228

Text-only word count: 4442

Number of Figures: 2

Number of Tables: 1

## **Abstract**

**Purpose:** This investigation evaluated the reliability and sensitivity-to-training of an audio-paced incremental swimming test. **Methods:** Eight young national-level male swimmers (mean (SD); aged 15 (1) year) performed a 7 x 200 m incremental swimming test (velocities 1.19, 1.24, 1.28, 1.33, 1.39, 1.45 m.s<sup>-1</sup> and maximal sprint pace respectively) using an audio-pacing device. The same test was performed 4 times by each participant, 1 week apart to assess reliability (WK1, WK2) and after 9 and 20 weeks of training (WK9, WK20). Blood lactate concentration ([La<sup>-</sup>]) and heart rate (HR) were recorded after each stage. Outcome measures were the velocity ( $v$ ) and HR at lactate markers of 2mM, 4mM and  $\Delta$ 1mM. **Results:** Velocities at the lactate markers proved to be more reliable than the heart rates with typical errors ranging from 0.66 to 2.30 % and 1.28 to 4.50 % respectively (shift in mean ranged -0.91 to 0.73 % and -0.84 to 1.79 % respectively). Across WK1, WK9 and WK20 there were significant improvements in peak velocity ( $P < 0.001$ ) and each of the velocities associated with the lactate markers ( $P < 0.05$ ), whereas only HR at  $\Delta$ 1mM improved ( $P < 0.05$ ). **Conclusions:** This paper has demonstrated that an audio-paced incremental swimming test is reliable for use with junior swimmers and is sensitive to changes observed following training. The post-swimming measurement of HR in the pool was comparatively less reliable.

**Keywords:** lactate threshold; swimming; reliability; training

## **Introduction**

The blood lactate response to exercise has long been recognised as providing useful insight into the metabolic demands of exercise. The lactate threshold (LT), defined as the oxygen uptake, or work rate, above which a sustained accumulation of lactate is observed in arterial blood,<sup>1</sup> is an attractive measure for providing physiological support to athletes and coaches since it is sensitive to training,<sup>2-5</sup> relates to performance<sup>2,6,7</sup> and can be used for prediction of performance or training prescription.<sup>3</sup>

There is little argument that blood lactate concentration ( $[La^-]$ ) is relatively unchanged, or increases very slightly, for exercise intensities below LT and then progressively increases above LT. However, there are a variety of complexities associated with measurement and interpretation of the LT. There are a range of competing theories regarding the mechanisms responsible for the blood lactate profile during incremental exercise<sup>3,8</sup> and this is one contributing reason for the wide-ranging terminology used, often interchangeably, to describe interrelated phenomena. Further assumptions requiring consideration concern the issue that lactate is typically measured in the blood rather than the muscle, and therefore  $[La^-]$  reflects a balance of lactate production, transport from the muscle into the blood and clearance from the blood to other muscles and organs for subsequent oxidation or gluconeogenesis.<sup>9</sup> Additional confounding factors include the site of blood sampling, assays for lactate analysis, diet and the environment.<sup>3</sup>

Probably the greatest inconsistency in the literature, however, resides in the technique applied to accurately determine the LT from the  $[La^-]$  profile in response to

incremental exercise,<sup>3</sup> usually expressed relative to work rate or velocity. Some common approaches include: use of arbitrary values for  $[La^-]$  at LT, such as 2 mM or 4 mM; an increase in  $[La^-]$  of 1 mM from resting values; intersection of lines best-fit to baseline and post-LT regions or improved logarithmic functions; formal modelling of the  $[La^-]$  curve. Evidence of this is provided by Tokmakidis et al.,<sup>10</sup> who applied a variety of published methods for determining LT to the same data-set and reported LT to occur between 79 and 92% of maximal oxygen uptake, a considerable range for a measure requiring accuracy.

Despite such limitations  $[La^-]$  analysis is routinely conducted in swimming,<sup>6,11</sup> although this is a sport that has been less researched presumably due to the difficulties in effectively controlling work rate in the pool.<sup>12</sup> It is nonetheless a sport with a significant aerobic component, particularly in longer distance events (1500 m and longer), with shorter events also estimated to require a balance of aerobic and anaerobic contributions.<sup>6,13</sup> Therefore, the LT is of functional relevance and a variety of incremental swimming testing protocols have been used,<sup>4,14-16</sup> with a range in the distances covered per increment stage (200 m to 400 m) and no. of stages (2 to 7). Of the above protocols the 7 x 200 m incremental step test proposed by the Australian Sports Commission<sup>11</sup> appears most logical as there is a higher frequency of data points for more accurate profiling of the blood lactate response. However, central to this and all of the other protocols is the accurate implementation of the swimming velocities, requiring that swimmers accurately pace themselves not only within each 200 m split, but also evenly increase this pace between consecutive stages. If this is not carried out the intensity will not actually be increasing incrementally. Whilst this issue is acknowledged,<sup>11,16</sup> there are no data presented in the previous research as to

how accurate the self-pacing is during these tests and Pyne et al.<sup>11</sup> report a common mistake of going too fast on the first split. More recently, Thompson et al.<sup>12</sup> have demonstrated an accurate and very reproducible method for controlling swimming velocity for training and competition purposes in swimmers, as well as proposing its suitability for future use in fitness testing.

Therefore, this study investigated the reliability and trainability of the blood lactate response to an accurately-paced 7 x 200 m incremental swimming test in national level junior male swimmers.

## **Methods**

### *Participants*

Eight male swimmers (mean  $\pm$  SD; age  $15 \pm 1$  year, body mass  $76.0 \pm 5.5$  kg, height  $181 \pm 4$  cm) with at least 6 years experience at National level (one British Youth squad member, three Scottish Youth swimmers, and four Scottish East District Team members) participated in the study. Freestyle personal best times for 200 m ranged from 1:58 – 2:07 (min:s). Participants were deemed to be short-to-middle distance swimmers in competition (100 – 400 m) and all participated in a periodised annual training programme, training between 6 and 9 two-hour sessions per week for 50 weeks each year. Training volume had averaged  $\sim 20,000$  km of pool training in the previous year leading up to the study. Participants, who were all familiar with the testing equipment and protocols to be used, provided written informed consent (with parental consent for minors), approved by the School Ethics Committee.

### *Study design*

This repeated measures research investigation was designed to be mainly observational,<sup>17</sup> the athletes being monitored before and after periods of training that were already planned, rather than a controlled intervention study. This approach was deliberately implemented as it would be impractical (lack of volunteers) and potentially unethical<sup>17</sup> to exclude a number of competitive athletes from training throughout the duration of the study (part of a competitive season) and there would be little scientific value in using a different population as a control group. Swimmers were initially tested one week apart (WK1 and WK2) to establish the reliability of the incremental protocol in this population. The athletes were tested a third time at the end of a 9-week training macrocycle early in the season that was predominantly aerobic in nature (WK9), and again a fourth time following a further 11-weeks of training that was focussed on aerobic and anaerobic endurance (WK20). Training consisted of between seven and eight pool-based sessions per week, volume ranging from 35 – 64 km.week<sup>-1</sup> and two land-based sessions per week. Approximately 60 – 70 % of this training consisted of high-intensity interval training; the remainder mostly lower intensity aerobic work. This training was outside the competitive season and so tapering (aside from pre-testing preparation) did not impact on training volume or intensity during the training phase. Compliance with training was excellent with swimmers attending between 98 and 100% of sessions. However, two swimmers unfortunately became injured immediately before WK20, automatically excluding them from the final testing session such that the training results refer to the remaining subset of swimmers.

### *7 x 200 m testing protocol*

All incremental testing was conducted in a temperature-controlled (27 °C) 25 m deck-level swimming pool with anti-wave lane ropes in position. Testing was conducted on the same day of the week and same time of day on each occasion and participants were instructed to arrive for testing in as similar condition as possible for each test. This required swimmers to: abstain from training and alcohol the day before and caffeine 3-hours before, testing; to ensure adequate hydration status; and to follow a similar diet ahead of each testing session. These pre-test instructions are routine for the participants prior to each fitness testing session and so to replicate their normal testing condition as closely as possible no additional monitoring was introduced regarding diet and hydration, as this may have altered their status. Prior to each test swimmers performed a standardised 800 m warm-up of moderate intensity, confirmed by initial low  $[La^-]$  below 2mM.

Swimmers performed each 200 m swim, in progressively shorter time periods (faster velocity) for the first six steps and the final 200 m step was a maximal test to provide an indication of performance.<sup>11</sup> Each 200 m step was begun with a push-start exactly 5 minutes after the previous one had started, recovery between steps being passive on the pool-side. Pacing during the test was controlled by use of an audio-pacing device (Aquapacer, Challenge and Response, UK) that was programmed to not only provide a sound signal at the end of the 200m split, but also provide a sound signal at the end of each 25 m pool length, thus accurately regulating swimming velocity. The pacing device was placed under the swimmer's cap adjacent to the ear. The 200 m split times that were selected were the same for all participants on all four testing occasions (min:s; 2:48, 2:42, 2:36, 2:30, 2:24 and 2:18, equivalent to velocities of 1.19, 1.24,



1.28, 1.33, 1.39 and 1.45 m.s<sup>-1</sup>), reflecting their quite homogenous swimming fitness levels, although all tests were performed individually. The time for each 200 m was checked to ensure even pacing and to measure the performance in the final maximal sprint. In all tests this pacing was extremely accurate, with all swimmers attaining their target split times to within 1 s. It is recognised that the test could also be performed in a 50 m pool by simply adjusting the split times accordingly, although there remains the possibility that some accuracy may be lost due to the lower frequency of audible signals as there are less turns. Similarly, the test would be readily adaptable for both more heterogeneous populations and more experienced swimmers, perhaps by basing the split times on personal best competition times as suggested by Pyne.<sup>11</sup>

On completion of each 200 m stage, swimmers immediately had their heart rate (HR) measured whilst still in the pool using a heart rate monitor (Treffene Coaches Heart Rate Monitor, Australia). Then, after exiting the pool (typically 20 – 30 s after completion of that swim), a 5 µl blood sample was taken from the finger-tip and analysed for [La<sup>-</sup>] using an automated lactate analyser (Lactate Pro, Arkray, Japan), that has been shown to be effective and reliable for such use<sup>18</sup> with a coefficient of variation (CV) between 3.1 and 4.1 % within the 2 – 10 mM range.<sup>19</sup> Before blood sampling the swimmer's finger was dried, cleaned with an alcohol swab and then the area was coated with petroleum jelly, to ensure that no sweat contaminated the sample and to facilitate the blood sampling.

### *Analysis*

For each test the  $[\text{La}^-]$ -velocity relationship was plotted (Figure 1) and three velocities were determined: the velocity at a  $[\text{La}^-]$  of 2 mM ( $v_{2\text{mM}}$ ),<sup>3</sup> the velocity at a  $[\text{La}^-]$  of 4 mM ( $v_{4\text{mM}}$ )<sup>20</sup> and the velocity at which an increase of 1 mM above baseline was observed ( $v_{\Delta 1\text{mM}}$ ).<sup>21</sup> The approaches used were chosen from a wide selection (Introduction) as, excluding the simplistic linear interpolation, they did not rely on derivatives of any complex data models and hence we made no assumptions regarding the kinetics of the response or their underpinning mechanisms. For example, the formula of Pyne et al.<sup>16</sup> was not repeated as this required use of a mathematical model to determine the slope and intercept, the details and physiological rationale for which were not presented.

The HR-velocity relationship was also plotted and a linear regression was performed, the resultant equations were solved for each of the three velocities determined using the lactate-velocity relationship to give comparable heart rates ( $\text{HR}_{2\text{mM}}$ ,  $\text{HR}_{4\text{mM}}$  and  $\text{HR}_{\Delta 1\text{mM}}$ ). The velocity, HR and  $[\text{La}^-]$  in the final and maximal 200 m stage were recorded as peak values ( $v_{\text{PEAK}}$ ,  $\text{HR}_{\text{PEAK}}$  and  $[\text{La}^-]_{\text{PEAK}}$  respectively). As velocity in each stage was kept constant each time the test was conducted (excluding the final 200m), improvements would be observed as increases in the velocities at the blood lactate markers described above.

Reliability was tested by calculating values for Typical Error, shifts in the mean and IntraClass Correlations (ICC) for the test-retest data sets (WK1-WK2) using the methods advocated by Hopkins.<sup>22</sup> The training data (WK1, WK9 and WK20) were entered into a One-Way Analysis of Variance with One Repeated Measure (Time).

Sphericity of the data was confirmed with Mauchly's test and statistical significance was set at  $P < 0.05$ , tests carried out using two-tailed hypotheses. A significant F-ratio was followed using pairwise comparisons, making no adjustment. This was considered appropriate as only 3 pairwise comparisons were to be made, and with a low  $n$ , adjustments to reduce Type 1 errors (such as Bonferroni) may have compromised statistical power and led to inflated Type II errors. All statistical analysis was conducted using the Statistical Package for Social Sciences (SPSS version 13, SPSS Inc., USA).

## **Results**

### *Reliability*

Means and standard deviations for the velocities, lactate concentrations and heart rates measured in the test-retest reliability trials (WK1 – WK2) are shown in Table 1, along with reliability measures of Typical Error, the shift in the mean and the IntraClass Correlation.

### *Training-induced changes*

With the repeated identical velocity protocols used, all velocity measures at fixed blood lactate markers showed some significant improvements (increases) over the time period investigated between WK1, WK9 and WK20 (Figure 1a). Firstly,  $v_{2mm}$  significantly increased ( $F_{2,10} = 4.52$ ,  $P < 0.05$ ), with WK1 and WK9 significantly different (mean difference  $0.037 \text{ m}\cdot\text{s}^{-1}$ , 95%CI 0.006 to 0.068,  $P < 0.05$ ), but WK1-WK20 and WK9-WK20 differences were not (although the difference between weeks 1 and 20 was approaching significance - mean difference  $0.047 \text{ m}\cdot\text{s}^{-1}$ , 95%CI -0.001 to 0.094,  $P = 0.054$ ).

Secondly,  $v_{4\text{mm}}$  also increased significantly ( $F_{2,10} = 6.29$ ,  $P < 0.05$ ) between WK1 and WK20 (mean difference  $0.03 \text{ m}\cdot\text{s}^{-1}$ , 95%CI 0.003 to 0.057,  $P < 0.05$ ) and between WK9 and WK20 (mean difference  $0.017 \text{ m}\cdot\text{s}^{-1}$ , 95%CI 0.004 to 0.029,  $P < 0.05$ ), but not between WK1 and WK9. Similarly,  $v_{\Delta 1\text{mm}}$  also increased significantly ( $F_{2,10} = 9.85$ ,  $P < 0.01$ ), between WK1 and WK20 (mean difference  $0.087 \text{ m}\cdot\text{s}^{-1}$ , 95%CI 0.036 to 0.137,  $P < 0.01$ ) and between WK9 and WK20 (mean difference  $0.058 \text{ m}\cdot\text{s}^{-1}$ , 95%CI of 0.004 to 0.112,  $P < 0.05$ ), but not between WK1 and WK9.

The main swimming performance measure,  $v_{\text{PEAK}}$ , also increased significantly ( $F_{2,10} = 39.88$ ,  $P < 0.001$ ), with significant improvements evident across all time intervals (WK1-WK9 mean difference  $0.038 \text{ m}\cdot\text{s}^{-1}$ , 95%CI 0.020 to 0.056,  $P < 0.01$ ; WK1-WK20 mean difference  $0.060 \text{ m}\cdot\text{s}^{-1}$ , 95%CI of 0.041 to 0.079,  $P < 0.001$ ; WK9-WK20 mean difference of  $0.022 \text{ m}\cdot\text{s}^{-1}$ , 95%CI of 0.006 to 0.037,  $P < 0.05$ ). Finally,  $[\text{La}^-]_{\text{PEAK}}$  increased significantly ( $F_{2,10} = 6.53$ ,  $P < 0.05$ ), although only between WK1 and WK20 (mean difference of 2.00 mM, 95%CI of 0.895 to 3.105,  $P < 0.01$ ).

The only significant differences in any of the HR data were for those at  $v_{\Delta 1\text{mM}}$  ( $F_{2,10} = 5.327$ ,  $P < 0.05$ ) between WK1-WK20 (mean difference 14  $\text{beats}\cdot\text{min}^{-1}$ , 95%CI 2 to 27,  $P < 0.05$ ) and between WK9-WK20 (mean difference 13  $\text{beats}\cdot\text{min}^{-1}$ , 95%CI 3 to 23,  $P < 0.05$ ) but not between WK1-9. All other changes in HR were not significantly different (Figure 1b).

## Discussion

This investigation has demonstrated that an audio-paced 7 x 200m incremental swimming test is reliable and sensitive to changes in physiological response and performance in national-level young male swimmers following training. The blood lactate response was more reliable and influenced more by training than the heart rate response.

#### *Reliability of protocol*

The reliability measures obtained compared well with those reported by other studies. Gore<sup>23</sup> gave technical error of measurement (equivalent to typical error) values of 2.4 % and 2.2 % for power outputs at 2 mM and 4 mM thresholds in rowing. Similarly, Gore produced HR typical error of measurement values of 1.2% and 1.0% at the aforementioned thresholds – values that compare directly with those in the present study. Grant et al.<sup>24</sup> reported limits of agreement of -1.10 to 1.51 km.h<sup>-1</sup> for the  $v_{4mM}$  in treadmill running, with a shift in the mean of 0.21 km.h<sup>-1</sup>. The corresponding limits of agreement for the current study are considerably lower; from -0.055 to 0.029 m.s<sup>-1</sup> (-0.198 to 0.104 km.h<sup>-1</sup>). This is perhaps unsurprising given the greater velocities achieved in treadmill running compared to swimming, however percentage values could not be calculated from their data for direct comparison with the present study. Also, it has been noted by Hopkins<sup>22</sup> that limits of agreement are too large as a reference range for making a decision about a change in a participant's measurements, a statement supported by Grant et al.<sup>24</sup> Therefore, the current study used the more sensitive measure of typical error as proposed by Hopkins.<sup>22</sup>

Intraclass correlations were diverse for the dependent variables measured. The lowest values were for the  $v_{\Delta 1mM}$  and  $HR_{\Delta 1mM}$  (0.688 and 0.800 respectively), with

correlations for HR being generally higher than those for the velocity measures. These ICC values compare less well with those reported in the literature, such as the 0.96, 0.98 and 0.98 for  $v_{\Delta 1mM}$ ,  $v_{2mM}$  and  $v_{4mM}$  thresholds in treadmill running reported by Pfitzinger and Freedson.<sup>25</sup> The same authors also gave ICC of 0.67, 0.90 and 0.88 for  $HR_{\Delta 1mM}$ ,  $HR_{2mM}$  and  $HR_{4mM}$ . While the present study found values well below these, Hopkins<sup>22</sup> has pointed out that intraclass correlations are not a single suitable measure of reliability for homogenous samples (as studied here), and that shift in the mean and typical error are better indicators of reliability. In these two measures, the present study showed good reliability for all dependent variables when compared to published literature, although those for HR were typically less than velocity.

It is worth highlighting that although the homogenous fitness levels of this group of swimmers permitted the use of the same velocities for these tests, it is relatively straight-forward to individualise the protocol according to initial swimming performance. A useful approach in this regard is the calculation of Pyne et al.<sup>11</sup> which bases the velocities as percentages of the personal best (PB) swimming time for that swimmer. The current study elected to use 6 s increments between the 200 m splits to ensure that these sub-elite junior swimmers demonstrated a clear baseline lactate response below LT, however Pyne et al.<sup>11</sup> recommend 5 s increments based on a percentage of PB. Aside from the aforementioned differences in increments to be used with more senior elite swimmers the test should certainly prove at least as reliable in this population, indeed these more experienced swimmers may be more efficient at self-pacing. Presumably the heart rate measurement in this population would also prove more reliable with poolside measurement due to more experience, although the use of heart rate monitors during swimming would introduce less error than post-

swim measurement. Additionally, despite only freestyle being examined here, the approach of Pyne et al. is suggested to apply equally to the other three swimming strokes.

### *Responses to training*

The right-shift in the lactate-velocity relationship observed in this study is the classic training response observed for a range of sports following aerobic training.<sup>3</sup> However, since no control group could be used in the current investigation it is not possible to conclusively attribute the observed responses completely to training. Nonetheless, it is highly likely that training was the main factor responsible for the significant improvements in physiological and performance measures. Similar studies with young,<sup>27</sup> trained<sup>4</sup> and elite<sup>16</sup> athletes have drawn such conclusions. Contributing mechanisms for the shift in lactate curve are likely to include a combination of both central and peripheral adaptations resulting in a reduced rate of lactate production and/or an improvement in the ability to clear lactate from the blood.<sup>3,9</sup> Experimental design issues encountered in the current research, such as the lack of a control group and low participant numbers, are not uncommon in applied sport science when investigating trained competitive athletes in individual sports.<sup>16,26</sup> For example, top-level athletes will not volunteer to abstain from training during the competitive season and different athletes in the same event will have differing strength & conditioning programmes..

Although there were many significant improvements in the velocity data, reflecting participants swimming at faster velocities for the same  $[La^-]$ , there were fewer differences in HR. However, Hopkins and co-workers<sup>20,28,29</sup> have pointed out that

meaningful 'worthwhile' performance increases may be very small, and might not show in established significance tests. Stewart and Hopkins<sup>28</sup> used a similar sample to the present study (junior swimmers) with comparable events (swimming distances which included 200m) and found that 'meaningful' improvements (defined as approximately half the intra-subject variability) might be as low as 0.5 %. In the current study, improvements of 0.9 to 2.5% and 2.1 to 6.5 % were found in the velocity measures between WK1-9 and WK1-20 respectively. These changes are greater than those of Stewart and Hopkins<sup>28</sup> and also larger than the typical error values for the same variables (0.66 to 2.30 %) reported in the current study, suggesting real improvements were made over the period of investigation. Heart rate data were more equivocal, with changes of -2.8 to 1.7 % and -0.5 to 9.3 % indicating greater intra- and inter-subject variability.

Given the population investigated it is worth highlighting that the improvements may have been slightly larger if more mature participants had been used. Whilst conclusive answers are not always available regarding the effects of growth and maturation on sport and physiological performance, the majority of evidence suggests that, if anything, children have a higher lactate threshold than adults relative to their maximal oxygen uptake and that as they mature the relationship tends to shift to the left.<sup>30</sup> Therefore, the current results tend to imply that the training responses are very likely to be real, especially given the relatively short time period studied. Although other minor factors such as diet, environment and hydration status cannot be excluded, these were controlled to an extent that was routine for the swimmers investigated and that is both realistic and achievable for swimmers without physiological measurement. It is acknowledged that tighter control of these factors by future



investigators may further improve the reliability of the protocol. The observed improvements in  $v_{PEAK}$  make it less likely that glycogen depletion was a factor causing the improvements observed in the current study.<sup>15</sup>

That there was no significant training effect of the HR response during this protocol is somewhat surprising, as Foster et al.<sup>31</sup> showed that the  $[La^-]$ -HR relationship is largely unaffected by aerobic training. One would expect to see a downward/right shift in the HR response to an incremental test in concert with the shift in LT. Therefore, aside from the HR analysis at the  $[La^-]$  markers selected, we also performed repeated measures ANOVA on the submaximal HR responses to the first six stages of the test (identical fixed velocities used on each occasion) across WK1, WK9 and WK20 and there was no significant effect of time. This finding is in direct contrast to much of the literature and is probably explained by the lower reproducibility of the HR responses, as discussed previously, and also illustrated by the large standard deviations (Figure 2b). The most likely source of this variation is the measurement of HR in the pool immediately after the completion of each 200 m split, rather than actually during the swim. This observation may have important implications for coaches and swimmers using this technique, that is frequently used and considered reliable, in similar populations.

That the improvements observed in  $v_{2mM}$  and  $v_{4mM}$  did not necessarily occur at the same times (e.g. WK1-WK9 vs. WK9-WK20) may be related to slight differences in training during these periods or the large variations in the individual responses to training observed here. Additionally, a Type I error caused by the relatively low participant number may mean that with a larger group all of the improvements may

have been significant. Previous research has often focused on hypothesis testing within groups of swimmers, whereas it may be more appropriate to examine individual responses to training that can be large enough to be of practical importance, yet not statistically significant within a group.<sup>7,26,32</sup>

There is certainly good confidence in the reported improvement of  $v_{PEAK}$ , or rather the duration of the final 200 m split. In addition to those physiological factors discussed, many other factors (e.g. biomechanical) may have contributed to this improvement. Of course the question remains whether the observed improvements would actually have translated to improved competition performance, had there been any at this time of year. Under such circumstances, where even more factors (e.g. psychological) are likely to play a role, it is possible that performance may not be improved despite improvements in LT and  $v_{PEAK}$ , as found by Pyne et al.<sup>16</sup> However, their subjects were truly elite with an average world ranking of 11 and at that level it is certainly harder to achieve significant improvements in performance.

### *Practical Applications*

It is important during monitoring of performance and physiological fitness that every effort is made to standardise testing and minimise error. The velocity during conventional incremental swimming tests has often been controlled voluntarily by the participant, but this study proposes that an audio-pacing device can be used during such testing to ensure that velocity is controlled effectively not only between, but also within, splits. The protocol can easily be adapted for use in 50 m pools as well as with more senior experienced swimmers using personal best times as a frame of reference. It is recommended that heart rate monitoring during swimming be used in future in

place of immediate post-exercise heart rate measurement as this proved to be less reliable than other measures. Large variations in the individual responses to training were observed and as such group analysis for individual sports such as swimming may not be optimal for detecting meaningful differences in physiological fitness or swimming performance.

### *Conclusion*

An audio-paced incremental step test is reliable for use in young competitive swimmers and is sensitive to training-induced changes, especially in lactate response.

### **Acknowledgements**

The authors thank the swimmers and coaches of Warrender Swimming Club (Edinburgh, UK) for their participation in this study.

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Table 1 - Test-retest reliability data. Means (SD) are shown for velocities ( $v$ ,  $\text{m}\cdot\text{s}^{-1}$ ) and heart rates (HR,  $\text{beats}\cdot\text{min}^{-1}$ ) at lactate concentrations of 2 mM ( $_{2\text{mM}}$ ), 4 mM ( $_{4\text{mM}}$ ) and a change of 1 mM from baseline ( $_{\Delta 1\text{mM}}$ ). Peak values ( $_{\text{PEAK}}$ ) for  $v$ , HR and lactate concentration ( $[\text{La}^-]$ , mM) are also shown alongside statistical output (see text for details).

Variable	Test (WK1)	Retest (WK2)	Shift in Mean (%)	Typical Error (%)	Intraclass Correlation Coefficient
$v_{2\text{mM}}$	1.36 (0.04)	1.37 (0.05)	0.73	0.66	0.962
$v_{4\text{mM}}$	1.44 (0.03)	1.43 (0.04)	-0.91	1.05	0.847
$v_{\Delta 1\text{mM}}$	1.35 (0.06)	1.35 (0.05)	-0.15	2.30	0.688
$v_{\text{PEAK}}$	1.53 (0.04)	1.57 (0.04)	2.39	0.90	0.888
$[\text{La}^-]_{\text{PEAK}}$	9.71 (2.32)	9.83 (2.75)	1.15	11.33	0.811
$\text{HR}_{2\text{mM}}$	158 (14)	161 (17)	1.79	1.28	0.982
$\text{HR}_{4\text{mM}}$	177 (13)	175 (16)	-0.84	1.76	0.954
$\text{HR}_{\Delta 1\text{mM}}$	156 (15)	156 (16)	-0.08	4.50	0.800
$\text{HR}_{\text{PEAK}}$	194 (8)	194 (8)	0.13	0.70	0.973

## Legends

Figure 1 – Changes in the blood lactate concentration ( $[La^-]$ ) to velocity relationship for one participant between weeks 1, 9 and 20 (WK1, WK9 and WK20). Horizontal dashed lines show velocities at  $[La^-]$  of 2 and 4 mM.

Figure 2 – Swimming velocities (a) and heart rates (b) at weeks 1, 9 and 20 (WK1, WK9 and WK20).  $v_{2mM}$ ,  $v_{4mM}$ ,  $v_{\Delta 1mM}$  and  $v_{PEAK}$  represent velocities ( $v$ ) and heart rates (HR) at lactate concentrations of 2 mM and 4 mM, a change in blood lactate concentration of 1 mM from baseline and peak respectively. Values are Mean (+ SD).  
\*  $P < 0.05$ , +  $P < 0.01$ .



Figure 1

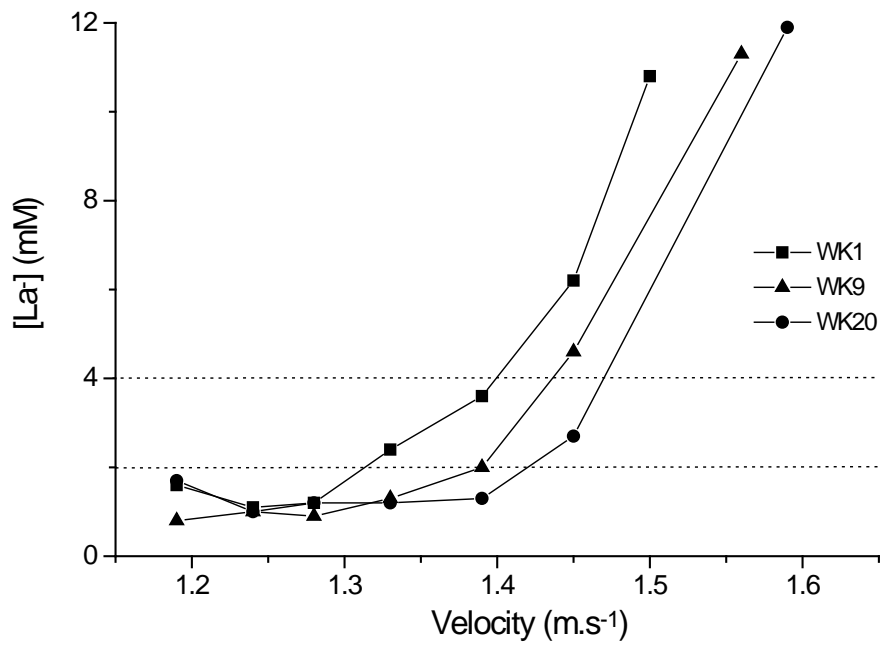


Figure 2

