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Letters to the editor

Unexpectedly severe hypoxia during sprint swimming

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To the editor: The possibility of hypoxia during competitive swimming has been recognized for over a decade [1], yet, because of technical limitations, SpO_2 has not previously been measured during swimming. We have succeeded in confirming hypoxia during strenuous sprint swimming by using a new generation pulse oximeter (Masimo Set; Masimo, Irvine, CA, USA) [2], which is resistant to motion artifacts, to measure SpO_2 from a finger probe. A surgical glove was worn over the

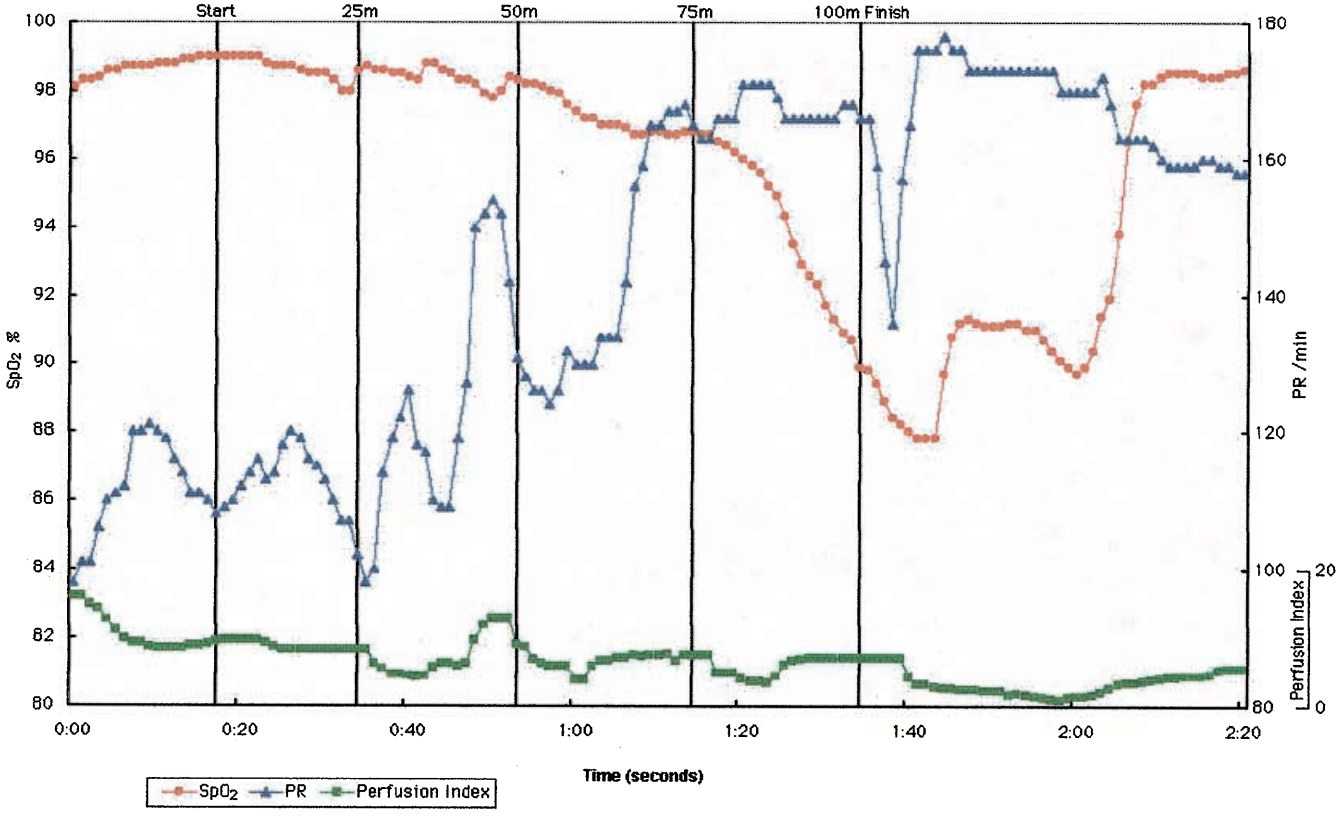

Fig. 1. Sample tracing of SpO_2 (*circles*), pulse rate (*PR*; *triangles*), and perfusion index (*squares*) during 100-m sprint swimming. The perfusion index (AC/DC of the signal) indicates the adequacy of perfusion at the measurement site. A perfusion index of above 0.5 is considered indicative of valid measurement

hand the sensor was on and polyolefin putty was used to make the sensor submersible.

Three well informed fit male athletic swimmers did three 100-m sprints (four 25-m lengths; 28°C) with rests between trials. SpO_2 and pulse rate were recorded digitally every second (Profox PFW; Profox Associates, Escondido, CA, USA). Each swimmer breathed into a standard capnometer without a nose clip for ten breaths immediately after finishing the sprint, and the highest end-tidal CO_2 values were recorded.

A representative graph of the data acquired is presented in Fig. 1. Unexpectedly significant arterial hypoxemia, a 6%–14% fall (median of 12%) in SpO_2 from the baseline, was seen in all the trials, especially at the end of the sprint. Pulse rate at that time reached as high as 182 per min, indicating how strenuous the trial was.

Exercise-induced arterial hypoxemia (EIAH) is recognized to occur in fit subjects [3], but this is the first objective report of its occurrence during sprint swimming. Among possible mechanisms that include ventilation-perfusion inequality and O_2 diffusion limitation, inadequate compensatory hyperventilation to match the increased CO_2 production caused by mechanical limitations during swimming has an important role.

In this study, the highest observed values of end-tidal CO_2 at the end of swimming ranged from 58 to 96 mmHg. This estimated high level of $PaCO_2$ might be characteristic of swimming sprints, as breathing may be sacrificed for speed, especially in the final stretch. We speculate that the combination of an increase in CO_2 production and intentional breath-holding decreased alveolar oxygen, causing significant hypoxia. A similar mechanism was suggested for synchronized swimming, but SpO_2 was not measured [4].

The lowest SpO_2 in our series was 83%, but it could be even lower in real sprint racing. The clinical significance of this hypoxia could not be determined, but further research using this pulse oximeter with a larger number of subjects is warranted. Relative hypoventilation is believed to be the main mechanism for this hypoxemia, as end-tidal CO_2 at the end of the sprint was extremely high. The highest end-tidal CO_2 observed in this series (96 mmHg) may bring about an alveolar oxygen partial pressure that is as low as 30 mmHg. Because swimming is often recommended for medical reasons [5], it is important to recognize the increased stress of restricted breathing in strenuous swimming compared with land-based exercises.

In summary, unexpectedly severe arterial desaturation during strenuous sprint swimming was detected. Relative hypoventilation is believed to be the main mechanism for this hypoxemia, as end-tidal CO_2 at the end of the sprint was extremely high.

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