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Short Report

Neurobehavioral consequences of repetitive head impacts in Para swimming: A case report

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ABSTRACT

Para swimmers with limb deficiency are faced with the particular situation that they must use their head to finish each competition by a hit to the wall. Repetitive head impacts may impair behavioral and brain functions. We therefore investigated neurobehavioral functions of a Para swimmer with dysmelia before and after repetitive head impacts (T1) and without (T2). Average head impact at T1 constituted 13.6 g with a mean impact force of 6689.9 N. Behavioral and brain functions decreased from pre to post at T1 but not at T2. Para swimmers with limb deficiency are therefore affected from the same consequences onto brain health that are observed after repeated sport-related concussions.

1. Introduction

Repeated head impacts in sports can impair brain health.[1] Most understanding of those effects is grounded in the investigation of sports such as boxing, ice hockey, rugby, American football, and/or soccer. However, relatively little attention has been paid to Para athletes. In fact, the most recent position statement of concussion in Para sport states that diagnosis and management of potential head injuries in Para athletes are challenging with limited data available.[2] In fact, the concussion experience of the Para athlete is unique, due to the interaction of the individual's primary impairment and the pathophysiology of concussion.[2,3] The consensus statement particularly points out that the heterogeneity of Para athletes, by nature of impairment, implies case-by-case decisions in order to develop concussion protocols. We address this issue in a Para swimmer with limb deficiency due to dysmelia. Dysmelia constitutes a congenital disorder resulting from a disturbance in embryonic development.[4] Such swimmers are affected by repetitive head impacts because they finish their race by hitting their head to the wall, instead of touching the finish pad with their hands (see Fig. 1). Thus, the head of a Para swimmer with dysmelia experiences repeated accelerations (/collisions) at the end of each race. High accelerations of the head may not only cause mild traumatic brain injuries (mTBI; concussions)[5] but its repeated occurrence may increase the risk of cognitive and neuropsychiatric impairment, as well as the risk of neurodegenerative disease and chronic traumatic encephalopathy (CTE).[6] The focus of the current paper is therefore to characterize the acute neurobehavioral consequences that follow repetitive head impacts in Para swimming with dysmelia.

2. Methods

2.1. Participant

The subject is a Para swimmer with limb deficiency due to dysmelia (19 years old, male, left-handed, height: 150 cm; weight: 50 kg; currently in the national team, participated in the Paralympics Tokyo 2020, sport classification S3/SB3/SM3). He reported to hit his head against the wall up to 60 times per year "in competition". An unknown number of "training hits" should be added. Informed consent was obtained in advance of the study. The local Ethics Committee approved the study (nr. 182/2020).

2.2. Measurements

We conducted two measurements with a five-week interval (T1, T2). At both times we performed a concussion assessment protocol before (pre) and after (post) each swim. T1 consists of 4 repetitions of swimming of 20 m with the highest velocity possible and finishing as in real competitions: by hitting the head against the microchronometer attached to the wall of the swimming pool (Fig. 1). At T2, the identical intervention protocol was performed but without head collisions against the wall.

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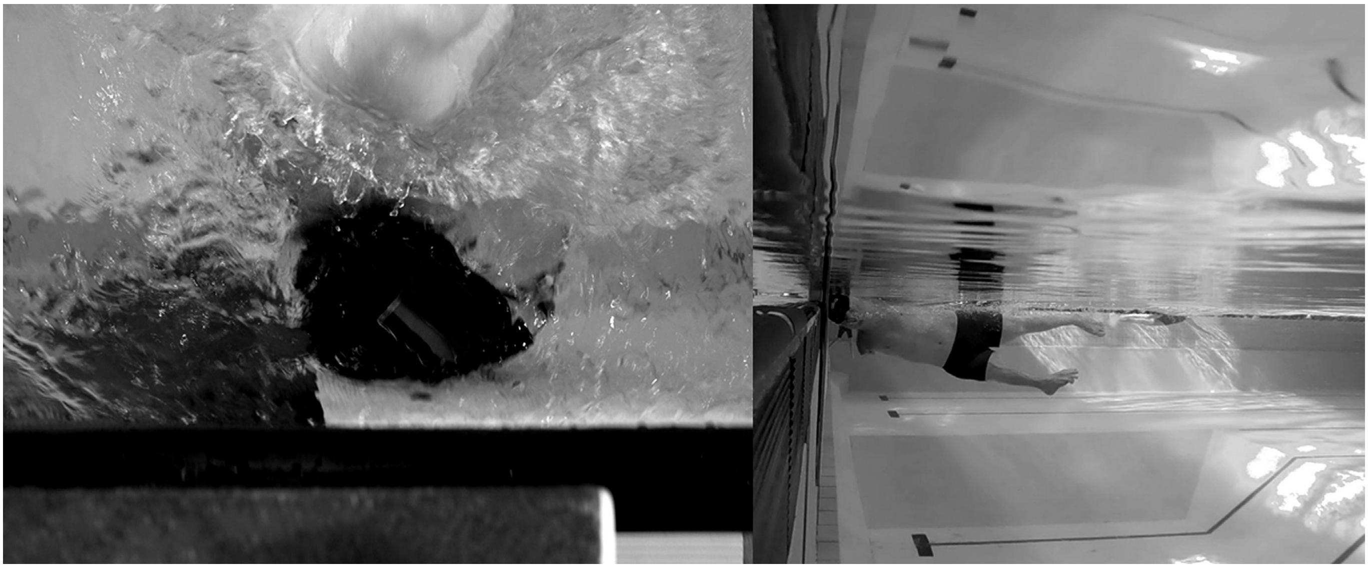

Fig. 1. One (out of four) head collision at T1.

Each head collision was assessed by an accelerometer (Witmotion) and video recordings. To calculate the acceleration of the head, we used the 3D Pythagorean Theorem formula.[7] The force (F) on the head was calculated in Newtons (N), with (m) constituting the mass of the body in kilograms (kg), and (a) the acceleration in m s^{-2}.

2.3. Pre/post measurements

At T1 and at T2, we performed pre/post measurements that have been previously used to assess post-concussion symptoms.[8–13] Post measurements were conducted immediately after the intervention (within 10 min) in a locker room next to the swimming pool. First, we conducted an interview by applying the Sport Concussion Assessment Tool 5 (SCAT5)[14] and the Positive and Negative Affect Schedule (PANAS).[15] The entire interview was video-taped (Go Pro Hero7 black) for the analysis of nonverbal hand movements. The fact that the subject still had two hands allowed for the gestural analysis with the NEUROpsychological GEStUre system.[16] Then, two cognitive tests were performed: (I) a working memory (WM) task[17] and (II) the King–Devick (KD).[18] Cerebral oxygenation changes were recorded by using a portable continuous wave functional Near InfraRed Spectroscopy (fNIRS) system (NIRSport 2, NIRx, Medical Technologies LLC, Berlin, Germany) during a (I) three minute rest period (for connectivity analyses) and during the (II) WM task (functional activation) above frontal cortices of each hemisphere. The fNIRS data (during the WM task) was analyzed using the Satori (v.1.8) toolbox.[19] Betas of the hemodynamic response were estimated by a general linear model, exported and analyzed by a repeated Analysis of Variance (rmANOVA) for the conditions of *time* (T1, T2) and *intervention* (Pre/Post). The fNIRS resting data pre-processing and analysis were carried out in MATLAB R2021a (Mathworks Inc.) as well as with the NIRS-toolbox.[20] Node strength was estimated, which refers to the total weight of all connections that link a particular node with other nodes within the network.

3. Results

3.1. Head impact

Head collisions at T1 resulted in a mean peak linear acceleration of 13.6 g ± 4.5. The mean peak force (F) acting on the head during the four head collisions was 6689.9 N ± 2701.8. The average acceleration during the control measurements at T2 was 3.0 g ± 0.2. The mean maximum velocity during swimming was 1.1 ± 0.1 meters per second (m/s) at T1 and 1.1 ± 0.0 m/s at T2.

3.2. Symptoms

At T1, the individual reported a PCS score of 0 pre and 54 post the head acceleration. At T2, the individual did not report symptoms. The PANAS score (positive affect) decreased from before 47 to 36 at T1 (at T2: pre 45, post 47).

3.3. Working memory

At T1, the individual reached 92 % correct answers before and 71 % after head impacts (at T2: pre 77 %, post 85 %). Reaction times at T1 were on average 732 milliseconds (ms) before and 926 ms after (at T2: pre 807 ms, post 730 ms).

3.4. King–Devick

At T1, the mean mistakes were 2.3 before and 1.7 after head impacts (at T2: pre 0.3, post 0.3). Reaction times decreased from before 15.0 seconds (s) to 12.1 s after head impact at T1 (at T2: pre 13.8 s, post 12.5 s).

3.5. Hand movements

The hand movement frequency at T1 showed 6.9 left hand (lh) and 8.1 right hand (rh) movements before and 0.6 lh and 4.4 rh movements after (at T2: pre 8.1 lh and 4.1 rh movements, post 7.2 lh and 7.8 rh movements). The hand movement duration at T1 was 2.6 s for the left hand (lh) and 1.4 s for the rh before and 6.8 s for the lh and 2.5 s for the rh after (at T2: pre 1.6 s lh, 0.8 s rh, post 2.2 s lh, 1.4 s rh).

3.6. Functional brain activation

The rmANOVA of the betas (ΔHbO_2) during the WM task showed a significant effect for the interaction of *intervention* and *time* ($F(1,19) = 11.335$, $p < 0.01$, $\eta^2 = 0.374$). Post-hoc comparisons revealed a significant decrease from pre to post at T1 ($p < 0.01$) (Fig. 2).

3.7. Brain connectivity

The rmANOVA of the node strength showed a significant effect for the factors *intervention* ($F(1,19) = 9.438$, $p < 0.01$, $\eta^2 = 0.332$) and

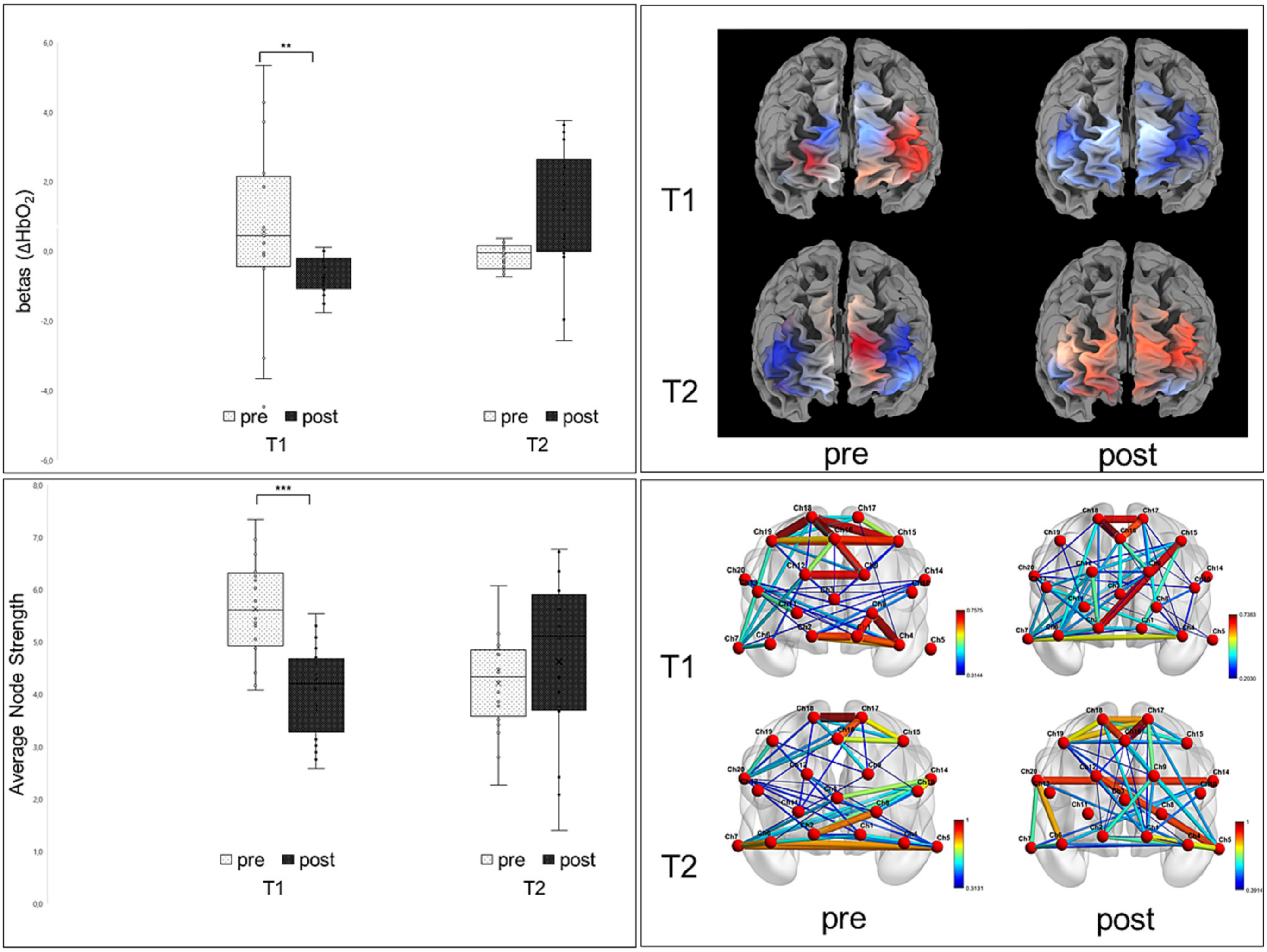

Fig. 2. Top (left/right): Brain oxygenation (betas; oxygenated hemoglobin (ΔHbO_2)) during the working memory (WM) task (overall channels). Bottom (left/right): Node strength (oxygenated hemoglobin (ΔHbO_2)) at resting state (overall channels).

for the interaction of *intervention* and *time* ($F(1,19) = 27.237$, $p < 0.001$, $\eta^2 = 0.589$). Post-hoc comparisons of the factor *intervention* showed significantly decreased node strength from pre to post ($p < 0.01$). The interaction effect revealed a significant decrease of node strength from pre to post at T1 ($p < 0.001$; Fig. 2).

4. Discussion

The present findings show that repeated head impacts in Para swimming with limb deficiency result in high impacts on the head, which is accompanied by reduced neurobehavioral functions.

When a Para swimmer with limb deficiency must stop a competition by using her/his head the average peak acceleration not only reach high values but is accompanied by significant impact forces. Thus, this situation results in high symptoms that are similarly observed after sport-related concussions.

The data further revealed that whereas working memory (WM) scores decreased from pre to post at T1 reaction times increased. The results of nonverbal hand movements also showed altered hand movement frequencies and durations. Furthermore, the athlete was characterized by decreased brain oxygenation in frontal cortices during the WM task as well as reduced node strength after the head impacts as when compared to before. No such differences were found at T2. Thus, the present findings indicate that behavioral and brain functions are affected from repetitive head impacts in Para swimming with dysmelia similarly to what is observed after sport-related concussions.

This investigation therefore provides for the first time objective data about impaired neurobehavioral functions in a Para athlete after repeated head impacts. Because there is growing concern that repetitive head impacts may cause not only short-term concussive like symptoms but also increase brain pathology,[5,6] the present case shows that this is also the case in Para swimming with dysmelia. We therefore conclude that Para swimming with limb deficiency is affected from an increased risk of behavioral and brain dysfunction similar to the consequences of repeated sport-related concussions. This issue should be closely monitored in swimmers with dysmelia, and ought to be considered in future Para sport events and its regulations.

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Confirmation of ethical compliance

The local Ethics Committee of the German Sport University approved the study (nr. 182/2020).

CRediT authorship contribution statement

Helmich I planned the study, collected/analyzed the data and wrote the manuscript. Chang YY, Gemmerich R, and Rodrigo L assisted in the data collection and data analysis. Funken J and Arun KM analyzed the data. Van de Vliet P helped writing the manuscript. All authors discussed the results and contributed to the final manuscript.

Data sharing statement

The data will be shared upon request.

Declaration of Interest Statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. There was no external financial support other than reported (by the Federal Institute for Sports Science (ZMI4-070401/21-23)).

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