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Phasic Electrodermal Activity During the Standardized Assessment of Concussion (SAC)

Adam C. Raikes, MS, LAT, ATC*; Sydney Y. Schaefer, PhD†

*Motor Rehabilitation and Learning Laboratory, College of Education and Human Services, Utah State University, Logan; †School of Biological and Health Systems Engineering, Arizona State University, Tempe

Context: The long-term effects of concussion on brain function during cognitive tasks are not fully understood and neuroimaging findings are equivocal. Some images show hyperactivation of prefrontal brain regions in previously concussed individuals relative to controls, suggesting increased cognitive resource allocation. Others show prefrontal hypoactivation and hyperactivation in other regions as a presumed compensatory mechanism. Given the relationship between sympathetic arousal and neural activation, physiologic measures of arousal, such as electrodermal activity, may provide additional insight into the brain's functional changes in those with a history of concussion.

Objective: To quantify differences in electrodermal activity during a commonly used standardized neurocognitive assessment between individuals with or without a history of concussion.

Design: Descriptive laboratory study.

Setting: Research laboratory.

Patients or Other Participants: Seven asymptomatic individuals with a self-reported history of physician-diagnosed, sport-related concussion (number of previous concussions = 1.43 ± 0.53; time since most recent concussion = 0.75 to 6 years, median = 3 years) and 10 individuals without a history of concussion participated in this study.

Main Outcome Measure(s): All participants wore bilateral wrist electrodermal activity sensors during the Standardized Assessment of Concussion. We measured normalized phasic (reactive) electrodermal activity during each test element (orientation, immediate recall, concentration, delayed recall).

Results: A significant group-by-test element interaction was present ($P = .003$). Individuals with a history of concussion had greater phasic activity during delayed recall ($P < .001$). Delayed-recall phasic activity was greater in both groups relative to the other elements.

Conclusions: Delayed recall resulted in greater physiologic arousal in previously concussed individuals relative to healthy control participants, supporting previous neuroimaging findings of increased prefrontal cortex activity during memory tasks after concussion. Given similar task performance and arousal patterns across the test, our results suggest that previously concussed individuals incur additional cognitive demands in a short-delay recall task.

Key Words: traumatic brain injuries, memory, wireless technology

Key Points

- Individuals with a history of diagnosed concussion displayed an increased physiologic arousal response to the delayed-recall section of the Standardized Assessment of Concussion.
- Increases in physiologic arousal may reflect alterations in how individuals with a history of concussion deal with the cognitive demands of short-term memory tasks. Therefore, short-term memory processes may not fully recover by 6 months after a concussion.

Numerous authors[1–5] have identified short-term cognitive impairments resulting from concussion. Affected cognitive processes include attention, memory, and executive function.[1,6] These impairments are generally viewed as transient, and a recent consensus statement[7] specified the (1) resolution of cognitive impairment and (2) successful completion of cognitive tasks as important markers in the return-to-play and return-to-learn decisions. Although clinical guidelines emphasize the recovery of cognitive function, there is little agreement on the nature, timeline, or measurement of such recovery. Some physiologic indices and neuroimaging techniques suggest lingering cognitive deficits,[8–13] whereas others indicate no long-term impairments.[14–16] Therefore, additional methods for probing the presence and extent of long-term postinjury impairment in a clinical setting are warranted.

One possible method of assessing cognitive effort after injury is through electrodermal activity (EDA); this is an established physiologic measure that reflects changes in arousal as changes in the skin's electrical conductance, which is controlled by the sympathetic branch of the autonomic nervous system through sweat secretion.[17–21] Emotional regulation and cognitive processes influence the control of sweating through changes in sympathetic drive.[17–21] For example, increased sympathetic drive due to high stress, cognitive load, or strong emotional responses results in more sweat secretion than low-activation states (ie, boredom, low cognitive load), producing higher or lower levels, respectively, of EDA.[19–22] Furthermore, EDA signals contain a tonic component, a measure of system state or responses over time (measured in minutes or longer) or both, and a phasic component, which offers insight into systemic responses to particular stimuli on the

Table. Demographics and Standardized Assessment of Concussion (SAC) Performance[a]

Variable	Concussion History		Value[b]	*P* Value
	No	Yes		
n (women)	10 (7)	7 (5)	$\chi^2 = 0.00$	1.00
Age, y	23.30 ± 3.30	21.80 ± 1.70	1.27	.22
Number of concussions	NA	1.43 ± 0.53	NA	NA
SAC Scores				
Orientation	4.92 ± 0.29	5.00 ± 0.00	–1.00	.34
Immediate recall	14.83 ± 0.39	14.86 ± 0.38	–0.13	.90
Concentration	3.50 ± 1.00	4.14 ± 0.90	–1.44	.17
Delayed recall	4.42 ± 1.00	4.57 ± 1.13	–0.30	.77
Total score	27.50 ± 1.83	28.57 ± 1.27	–1.50	.15

Abbreviation: NA, not applicable.

[a] No differences were observed between the groups for age or any measure of SAC performance. Values are mean ± SD.

[b] Unless otherwise noted, *t* test was performed.

order of milliseconds to seconds.[12,13,19,20,23–25] Thus, EDA, and particularly the phasic component, has been used as a proxy for quantifying cognitive stress and load under numerous laboratory and real-world conditions.[23–26] Two previous groups measured EDA in previously concussed, asymptomatic individuals and demonstrated decreased phasic responses compared with healthy, never-concussed control participants in error-detection tasks[12] and decision making.[13] This decrease in phasic activity indicates that individuals with a history of concussion display decreases in emotional or cognitive arousal in response to anticipatory tasks and during error detection.[12,13]

Given these characteristics, EDA is a candidate biomarker for neurologic function after concussion. Importantly, changes in EDA appear to be positively related to patterns of neural activation, particularly within the prefrontal cortex during memory and attentional tasks.[27–29] For this measure to be used in future investigations of concussion, however, typical responses first need to be quantified. To do so, we measured EDA in individuals with or without a history of concussion during administration of the Standardized Assessment of Concussion (SAC)—a valid and reliable clinical test of cognitive status often used as a baseline measure and after concussion.[7,30–33] Because the different test elements of the SAC require different levels of cognitive load in theory and by design,[30] we hypothesized that the phasic (reactive) component of EDA would change as a function of SAC test element in healthy young adults. We also hypothesized that asymptomatic, age-matched adults with a self-reported history of concussion would demonstrate less phasic EDA in response to the test, given the suppressed phasic responses in decision making and error detection in traumatic brain injury.[12,13] Thus, the purpose of our study was to quantify differences in phasic EDA during the SAC in individuals with or without a self-reported history of concussion.

METHODS

Participants

A total of 17 adults (7 asymptomatic individuals with a history of concussion, 10 without a history of concussion) participated in this study. Volunteers were recruited by advertisement to several large classes on a university campus and reported for testing between 11:00 AM and 3:00 PM. They were told that they would be taking part in a study looking at physiologic responses to a brief neurocognitive test. Individuals were separated into history and no-history groups based on self-report at the time of testing. A *history of concussion* was defined as a self-reported history of physician-diagnosed, sport-related concussion no less than 6 months (range = 8 months to 6 years, median = 3 years) before participation. This time frame was used because most individuals present as clinically asymptomatic (according to somatic, cognitive, and postural measures) by this point.[1,7,30] Those in the history group were excluded from the study if they reported any concussion symptoms or a concussion more recently than 6 months before participation or if they were limited in work, school, or sport due to their concussion history. The Utah State University Institutional Review Board approved all procedures. All participants provided written informed consent before the study. The Table contains demographic and test-related comparisons between the groups.

Electrodermal Sensors

Wireless, wrist-worn sensors recorded EDA (model Q Sensors 2.0; Affectiva, Waltham, MA; Figure 1A), sampling at a rate of 16 Hz. These commercially available sensors pass a low electrical current (up to 1100 mAh) between 2 small electrodes (1-cm^2 surface area).[34] The units of EDA are microSiemens (μS), which indicate the amount of electrical conductance. Traditionally, EDA is recorded at the fingertip because of the density of sweat glands there that are sensitive to autonomic responses.[21,34] However, wireless wrist-worn sensors allow for unobtrusive testing while the participant sits and moves freely. Previous researchers[34] using measures of EDA collected from the wrist demonstrated moderate to strong reliability as evidenced by correlations with responses from the fingertips ($0.56 \leq r \leq 0.96$).

We collected bilateral EDA to account for task-related lateralization effects in electrodermal responses.[35] Right-handed individuals tend to demonstrate greater phasic responses to verbal tasks on the left side and to spatial tasks on the right side. Left-handed individuals tend to demonstrate similar patterns of activation (left side > right side) regardless of task type.[35] Therefore, EDA was collected from both wrists during the entire test, and the data were pooled individually for each wrist in each test component.

Before baseline data collection, participants performed a 5-minute jog at a self-selected pace to generate sweat on the wrists for initializing the sensors, per the manufacturer's recommendation. Sensors were then placed on the anterior surfaces (Figure 1B) of both wrists and participants sat quietly in front of a laptop (model X501A; Asus, Taipei, Taiwan) displaying a blue circle on a white background. Participants were instructed to "clear their minds as completely as possible" during the baseline period to limit any cognitive activity that could trigger electrodermal responses. The baseline period lasted 5 minutes, which was sufficient for establishing skin-to-electrode contact as evidenced by a stable electrodermal response (indicating a stable autonomic state) and skin temperature (< 0.01 μS

and <1°C change over the final 30 seconds), and was then followed by the administration of the SAC in compliance with published instructions.[33]

Standardized Assessment of Concussion

The SAC (2nd edition) is a paper-and-pencil neurocognitive test for rapidly screening individuals. The maximum score is 30, and higher scores indicate better performance.[30,33] The SAC comprises 5 test elements (orientation, immediate recall, neurological screen, concentration, delayed recall), preceded by a standard set of oral instructions. This assessment was developed particularly for testing the neurocognitive changes most sensitive to impairment when a patient is concussed: immediate and delayed recall and concentration.[1,30] The SAC has 3 forms to allow for baseline testing and serial follow-up while minimizing learning effects[30]; all have demonstrated reliability, sensitivity, and specificity across different age groups and levels of participation.[30–32]

In this study, a single evaluator with clinical athletic training experience (4 years) first read the test instructions identically to each participant. Each participant performed the standardized tasks that made up the 5 test elements related to time orientation, immediate and delayed recall, and concentration, while also undergoing a peripheral neurologic screen.[33] This screen involves sensory and strength testing of the upper and lower extremities as well as finger-to-nose and tandem-walk coordination tasks.[33] Although the multiple test forms of the SAC minimize learning or test-retest effects,[30] all participants in this study received Form A of the test unless they described previous experience with the test. In these cases (2 of 17 participants), Form C was used. The average test duration was 4.95 ± 0.53 minutes.

Data Processing and Analysis

We sampled raw EDA data at 16 Hz and stored them remotely in each sensor with time stamps corresponding to the start of each test element. Immediately after collection, we uploaded the data from the sensor to a desktop computer using the manufacturer's software, which then exported the data from the left and right wrist sensors together as comma-separated value files. All exported data were preprocessed in MATLAB (release 2014b; The MathWorks, Inc, Natick, MA). Each participant's entire raw data signal was filtered and smoothed with a Hanning window (window length = 16 samples) to mitigate fluctuations in the EDA signal due to wrist movement. Data were then standardized per wrist following standard procedures.[17] This standardization permitted interindividual comparisons of electrodermal responses. We deconvolved all data, separating them into tonic and phasic components, via continuous deconvolution analysis in Ledalab (version 3.4.6; The MathWorks, Inc) and then exported the phasic components for further analysis.[22]

We calculated mean values for the phasic components per person, per wrist, and per test element. We ran a mixed-model analysis of variance in SAS (version 9.4; SAS Institute Inc, Cary, NC) with main effects of group (history or no history of concussion), wrist (dominant or nondominant, based on self-report), and test element (orientation, immediate recall, concentration, or delayed recall), as well as all 2- and 3-way interactions for phasic EDA with significance set at $\alpha = .05$. Because the neurologic screen of the SAC requires arm movement and physical exertion that could introduce significant movement artifact, we excluded this test element from our analyses. Where appropriate, Tukey honestly significant difference post hoc comparisons were conducted. To determine whether the groups performed differently on the SAC, we ran separate *t* tests on

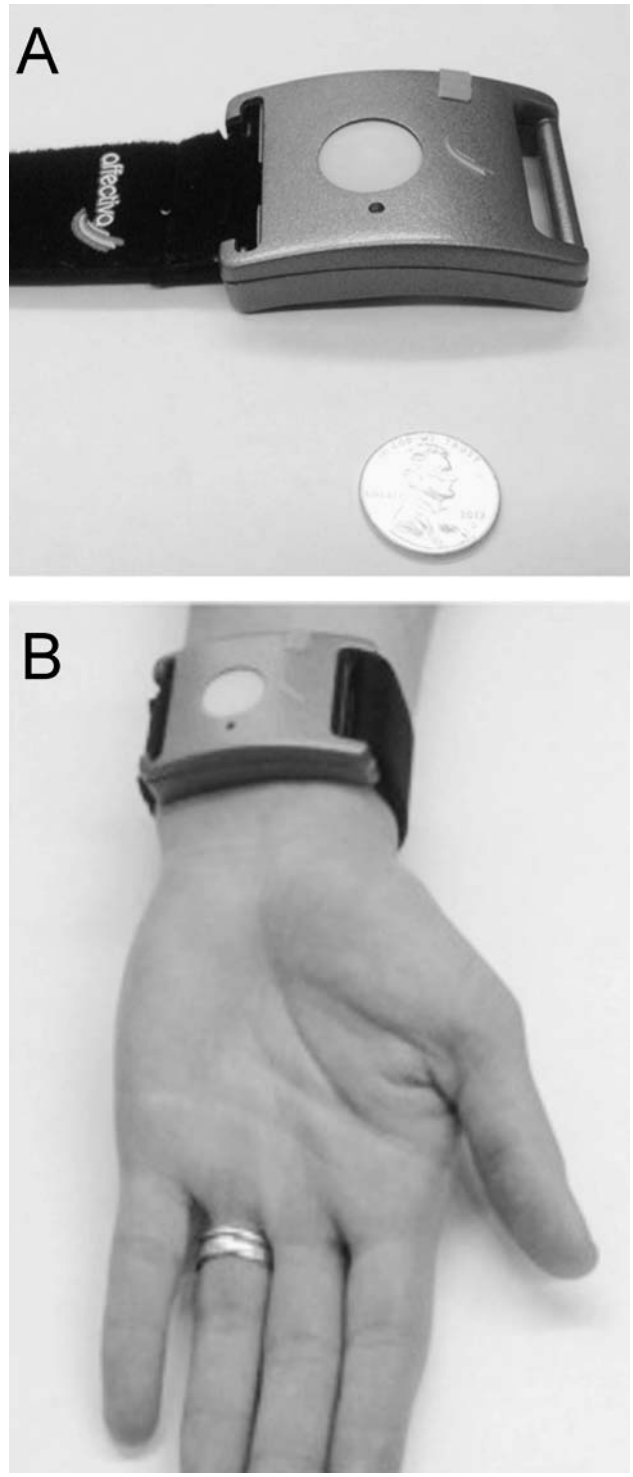

Figure 1. A, Example of electrodermal activity sensor. A penny is shown for scale. B, Position and placement of the sensor on the anterior surface of the wrist.

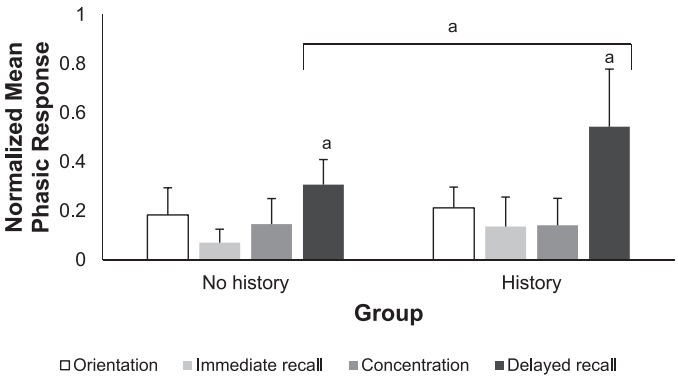

Figure 2. Normalized mean phasic responses per test element by group. Mean phasic electrodermal activity differed between the 2 groups for the delayed-recall element of the Standardized Assessment of Concussion ([a] $P < .05$). No other between-groups differences were observed. Within groups, delayed recall was associated with greater phasic activity than the other test elements. Error bars represent standard deviations.

the SAC item scores as well as the overall test score. Significance for these tests was also set at $\alpha = .05$.

RESULTS

We inspected the phasic data to ensure that they met the assumptions of approximate normality and heteroscedasticity. Amplitudes of individual phasic electrodermal responses are known to have positive skew, and, consistent with standard analyses, our data required transformation [y = log(1 + mean phasic response)] to meet the assumptions.[18,22]

As hypothesized, there was a significant interaction between concussion history and test element ($F_{3,45} = 5.49$; $P = .003$; $\eta^2_{partial} = 0.267$) and a main effect of test element ($F_{3,45} = 39.35$; $P < .001$; $\eta^2_{partial} = 0.724$). Post hoc analyses revealed greater phasic EDA in the delayed-recall element of the test for the history group compared with the no-history group (Figure 2; $P < .001$). Additionally, both groups had greater phasic EDA responses during delayed recall compared with orientation (history: $P < .001$; no history: $P = .023$; Figure 2); immediate recall (history: $P < .001$; no history: $P < .001$); and concentration (history: $P < .001$; no history: $P = .002$). The no-history group also had greater phasic responses during immediate recall than during orientation ($P = .045$). In terms of test performance, the groups did not differ on any test element or in the overall score for the SAC (Table).

DISCUSSION

The purpose of our study was to quantify phasic EDA responses during a commonly used standardized neurocognitive assessment in asymptomatic individuals with or healthy individuals without a history of concussion. Our results showed a difference between the history and no-history groups on the delayed-recall section of the SAC, with the history group having much larger phasic responses. This indicates that individuals with a history of concussion had more physiologic arousal to the delayed-recall section than did healthy individuals. These results were inconsistent with our hypothesis that individuals with a history of concussion would have a lower phasic EDA response to the test. Partially consistent with our hypothesis, however, was the finding that the different cognitive demands among the test elements of the SAC resulted in different phasic responses. This, again, was evident in the delayed-recall portion of the SAC: in both groups, arousal was higher than in the other test elements.

In addition to its many regulatory functions, the autonomic nervous system is known to respond to the cognitive demands to and emotional responses of an individual.[12,13,24–26,36] Strong functional connections between cognitive and emotional centers–frontal and prefrontal cortical areas, the anterior cingulate cortex, hypothalamus, and limbic system–and visceral centers integrate cognitive and emotional processes into the autonomic system.[19–21] Electrodermal responses, controlled by the sympathetic branch of the autonomic nervous system, have previously been shown to scale with cognitive tasks,[25] such as mental arithmetic task,[36] reading,[24] and driving,[23] even without an emotional component in healthy individuals. Taken collectively, these findings suggest that healthy individuals demonstrate an increase in physiologic arousal in response to increasing levels of cognitive demand.

However, similar conditions may yield less or attenuated arousal after concussion.[12,13] For example, van Noordt and Good[13] observed hypoarousal in those with a history of concussion relative to healthy control participants in the anticipatory stages of a decision-making task, despite similar decision-making performances. Similar effects of hypoarousal in previously concussed individuals have been observed during executive function tests.[12] Both groups hypothesized that hypoarousal, as measured by EDA, was related to functional deficits in the midline[12] and ventromedial prefrontal cortex (PFC).[13] As such, these deficits could manifest as an inability to make appropriate decisions in behavioral regulation[13] and an inability to appropriately respond to such errors.[12] The EDA responses in our study demonstrated the opposite response, hyperactivation in the concussed group, as working-memory demands increased. Yet working-memory task activation is generally associated with the dorsolateral PFC.[8,10,37,38] Thus, task-specific demands and their associated areas in the PFC may explain these differences in task-related activation patterns.

The EDA data from this study may provide clarification regarding mixed postconcussion findings in previous functional magnetic resonance imaging studies. Some investigations have shown increased PFC activity during working-memory tasks performed in the acute phase of concussion recovery, an effect persisting up to 2 months after injury.[8–11] Notably, hyperactivation was evident despite comparable task performance, thereby suggesting increased demand on the PFC to yield similar behavioral results.[8–11] In contrast, other authors[14–16] have reported decreased postconcussive activity in similar brain regions during similar tasks relative to control participants, instead showing increased activity in temporal and parietal areas that the investigators interpreted as compensatory in nature. These mixed results are not necessarily at odds with each other, given that PFC activity in a postconcussive state may increase to a point, but if a task's cognitive demands become too great, then other regions with functional connections to the PFC may be recruited.

Our EDA results support the previous findings of hyperactivation in the PFC, given that (1) the SAC is not designed to be exceedingly challenging (see the additional rationale that follows) and (2) both groups of participants (with or without a concussion history) performed equally well, with comparable EDA patterns on all but 1 test element. For the test element that is theoretically the most challenging (ie, delayed recall), both groups showed increases in phasic EDA, thereby indicating an increase in resources allocated to completing the delayed recall. The history group showed a larger increase in arousal, as reflected by a greater EDA response relative to the no-history group, without a decrement in task performance. Our results are similar to those of researchers[4–7] who noted that participants with PFC hyperactivation achieved similar behavioral outcomes on a working-memory task.

These and other findings highlight how results from behavioral tests alone may not necessarily capture all of the relevant information about postconcussion cognitive status and recovery. For example, postconcussive cognitive impairment measured by the SAC has been shown to resolve within 7 to 10 days[1,30]; thus, we expected our groups to perform similarly on the test itself. In light of previous neuroimaging findings, our results provide further evidence of increased cognitive resource allocation in response to a short-delay (~2 minutes) verbal recall task in asymptomatic, previously concussed individuals who might otherwise be considered fully recovered.

Furthermore, our phasic EDA data are consistent with previous SAC item analyses, which have demonstrated that for individuals without an acute concussion, the orientation and immediate-recall elements are not challenging (more than 92% of healthy respondents accurately answer these questions), whereas the concentration and delayed-recall elements are "acceptably challenging" (between 10% and 92% of healthy respondents answer these questions accurately).[39] For both groups in this study, phasic EDA responses during the delayed-recall element were greater than for all other test elements: that is, an increase in arousal in response to increasing task difficulty, which we suggest indicates an increase in the cognitive demand for that task. This is in comparison with the phasic responses to the other test elements, which were all comparatively small and may therefore provide additional evidence that the different sections of the SAC elicit different physiologic responses in relation to task demands. Also, though a difference was observed in the phasic responses between the orientation and immediate-recall portions, the lack of between-groups differences on the orientation, immediate-recall, and concentration sections suggests that these sections do not discriminate those with a concussion history from those without a concussion history.

We acknowledge, however, that this study is not without limitations. The first is the relatively small sample size, yet on the basis of the effects observed by O'Keeffe et al[12] and van Noordt and Good,[13] a total sample size between 6 and 18 participants was needed to demonstrate power at the 0.8 level.[12,13] Second, in this preliminary examination of EDA and the SAC, numerous personal factors (eg, circadian rhythm, diet, medications) and environmental factors (eg, ambient temperature, lighting, humidity) may have had unintended effects on autonomic outflow. The effects of these individual factors merit further investigation in future studies. Additionally, the self-selected jog before the baseline measurement may have affected the results by increasing autonomic outflow. Given that all of our participants reached a stable level of EDA at baseline and that our previously concussed participants were asymptomatic and had returned to activity, including sport and exercise, this risk is low but may still be a factor to consider. Third, we were unable to fully disentangle cognitive-load effects from potentially emotional effects, as EDA responds to both emotional and cognitive demands. Although it is possible that the individuals with a history of concussion experienced an SAC-related emotional response, perhaps remembering their previous concussion incident, we do not see this as a plausible explanation for the outcomes observed here. This examination was not used in any baseline or follow-up capacity and therefore imparted no clinical effect on any of the participants that could have resulted in anxiety. In addition, only 2 individuals indicated previous experience with the SAC, making it unlikely that the group differences in phasic EDA responses resulted from remembering a previous concussion examination or return-to-play decision. Furthermore, if this had been the case, we would expect to have seen an elevated phasic response across the entirety of the test, which we did not. Indeed, we saw comparable patterns of arousal between groups throughout the test, with the only observed group difference being an elevated phasic response during delayed recall in the history group. Additionally, the words in the delayed-recall element were the same as those in the immediate-recall element, which in and of themselves did not yield a group-specific emotional response. Given these considerations and the previous neuroimaging literature described earlier, we interpret our findings as indicating an increase in arousal related to the need for additional cognitive resources rather than an emotional response to the test.

CONCLUSIONS

Asymptomatic individuals with a history of concussion demonstrated greater increases in physiologic arousal in response to increased cognitive demands during delayed recall compared with those who had no history of concussion. The long-term consequences of concussion demand a comprehensive and objective assessment of its effects on the nervous system. Wireless sensors that record EDA may enable clinicians to assess underlying cognitive function during an entire battery of tests, both before and after concussion or during other conditions resulting in neural trauma. Because of the differences we observed between the history and no-history groups in the present study, EDA in the context of cognitive testing may be valuable in distinguishing those with and those without a previous concussion, and this merits further investigation. Future authors who simultaneously collect EDA measures during baseline neurocognitive testing could better quantify a normative, preconcussive state to which postconcussion tests could be compared for more informed decisions about clearance for returning to work, school, or activity.

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Address correspondence to Adam C. Raikes, MS, LAT, ATC, Motor Rehabilitation and Learning Laboratory, College of Education and Human Services, Utah State University, 7000 Old Main Hill, Logan, UT 84322. Address e-mail to adam.raikes@usu.edu.