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The Effect of Sport Concussion on Neurocognitive Function, Self-Report Symptoms and Postural Control

A Meta-Analysis

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Abstract

Sport concussion is commonly assessed using a battery of tests that evaluate neurocognitive functioning, postural control and self-report symptoms. The degree to which concussion affects each of these measures is unclear. Thus, the purpose of this meta-analysis is to systematically review and quantify the effect of sport concussion on each assessment measure when administered immediately post-injury and in the 2 weeks following injury. PubMed and PsychINFO databases were searched from January 1970 to June 2006, from which 39 were

included for review. Studies were selected for review if they included concussed athletes who were evaluated using one of the three assessment measures. One post-morbid assessment must have been completed within 14 days of injury and compared with a baseline measure or control group. Study design, type of neurocognitive assessment, timing of assessment following injury and number of post-concussion assessments were extracted as potential moderators. Sport-related concussion had a large negative effect (mean Δ; 95% confidence interval) on neurocognitive functioning (−0.81; −1.01, −0.60), self-report symptoms (−3.31; −6.35, −0.27) and postural control (−2.56; −6.44, 1.32) in the initial assessment following injury. A reduced, but large effect, was also seen in the 14 days following the initial assessment for neurocognitive functioning (−0.26; −0.46, −0.06), self-report symptoms (−1.09; −2.07, −0.11) and postural control (−1.16; −2.59, 0.27). Our findings demonstrated large effects for each aspect of the assessment battery. These findings support the use of the multifaceted concussion evaluation.

Between 1.6 and 3.8 million sport-related concussions occur in the US annually,[1] with an estimated 250 000 in high-school football alone.[2] The cumulative cost of concussive injuries is nearly $US17 billion (1999 values) in both direct and indirect costs.[3] Injury rates in American football are well documented with investigations of high-school football reporting that 3.6–5.6% of players will sustain a concussion in a single season.[4,5] Collegiate concussion incidence is similar and reported to range from 4.0% to 5.0%,[5,6] although the rate of game day concussions has steadily increased from 1987 through 2003.[6] At the professional level, a 6-year study found the rate of concussion to be 0.41 injuries per game.[7]

Certified athletic trainers working with sports teams are commonly the first to assess a suspected concussive injury. The National Athletic Trainers' Association position statement on concussion[8] and the International Symposium on Concussion in Sport summary and agreement statement[9] recommend a battery of tests that included evaluations of neurocognitive functioning, self-report concussion-related symptoms and postural stability. Baseline testing of individual athletes on all facets of the battery during the preseason is recommended for direct comparison to post-morbid evaluations. The objective test results may be appraised by the team or primary-care physician to support the physical examination when making a diagnosis and return-to-play decision.[10] Each assessment measure adds additional information on the status of the concussed athlete by independently evaluating differing aspects of cerebral functioning.

The neurocognitive evaluation is suggested to provide the greatest amount of information to the clinician following injury and is commonly used as the cornerstone of the assessment.[9,11,12] Traditionally, extensive pencil and paper assessments were administered to evaluate multiple domains (e.g. information processing, planning and memory) and have proven effective in detecting cognitive decrements following concussion.[13-15] With improvements in computer technology and availability, computerized neurocognitive tests are now common in sports medicine settings.[10] At least three computerized concussion assessment programs are commercially available and each has shown sensitivity to the effects of concussion.[16-18] Computerized tests are purported to offer many advantages over pencil and paper tests,[12] but both assessment techniques are limited to the athletic training room or other controlled environments. To remedy this shortcoming, the Standardized Assessment of Concussion (SAC) was developed as a brief concussion screening tool for sideline administration and has proven effective in the assessment of acutely concussed athletes.[19]

Self-report concussion-related symptoms are the most commonly used tool for concussion assessment employed by athletic trainers,[10] but considerable variation exists in the specific content of the

symptom list.[20-22] Recent research showing nine items divided equally into three latent constructs (somatic, cognitive, neuropsychological) may best describe concussion and remove the potential of other symptoms confounding the assessment.[23] Use of self-report symptoms, however, relies on the accurate reporting of symptoms by the concussed athlete. In some instances, athletes may under-report symptoms to accelerate their return to play or may not be aware their symptom is related to concussion.[24,25]

Recently, instrumented and non-instrumented postural control assessments have been used to evaluate concussed athletes. The Balance Error Scoring System (BESS) is a brief, non-instrumented assessment of balance.[26] The test can be completed on the sideline or in the athletic training room. Following injury, concussed athletes commonly commit more errors on the BESS test compared with a baseline evaluation and control subjects, indicating a decrease in postural control.[27] The more sophisticated, instrumented NeuroCom Sensory Organisation Test (SOT) has also been used for concussion assessment.[13,14,17] An evaluation of concussed athletes on the SOT has shown sensory integration decrements that lead to an overall decrease in postural control.[14] This assessment technique, however, is cost prohibitive in most settings.

Assessment of sport concussion is difficult as no two injuries result in the same changes to cerebral functioning. The multifaceted assessment battery was developed to evaluate distinct aspects of cognitive functioning, but post-injury tests may yield conflicting results.[14] This problem is magnified with variability in the test battery, including differing neurocognitive tests or symptom lists. A previous systematic review elucidated the neurocognitive response to concussive injury.[28] This analysis focused on the effect of concussion on various cognitive domains when evaluated by pencil and paper tests, computerized assessments or the SAC. The results suggested the greatest impact of concussion was on memory acquisition and global cognitive ability. To what degree concussion affects postural control and self-report symptoms was not completed. Therefore, the main purpose of this article was to ascertain the degree of change in each distinct aspect of the concussion assessment battery: neurocognitive functioning, self-report symptoms and postural control. We also sought to directly compare effects between different aspects of the assessment battery in studies where multiple techniques were employed. Finally, we evaluated how study design, type of neurocognitive assessment technique, the time of assessment following concussion, and the number of post-concussion assessments may moderate the effects of sport concussion.

1. Methods

1.1 Study Selection

PubMed and PsychINFO databases from January 1970 to June 2006 were searched using the following keywords: 'concussion', 'mild traumatic brain injury', 'sport', 'athlete', 'football', 'soccer', 'hockey', 'boxing', 'cognition', 'cognitive impairment', 'balance', 'postural control' and 'symptoms'. The search was limited to English-language publications. Searches were supplemented by reference lists from retrieved articles and related reviews. Colleagues familiar with the topic area also were consulted in a further effort to locate published studies.

Studies were included if they met the following criteria: (i) the sample included athletes concussed during a sporting event; (ii) the dependent variable was a measure of neurocognitive function, self-report concussion-related symptoms, and/or postural control; (iii) post-concussion assessments were compared with pre-season baseline measures or a control group; (iv) at least one post-concussion assessment was completed within 14 days of injury; and (v) enough information was provided by the authors to calculate effect sizes. Review articles, abstracts, case studies and editorials were excluded from the analysis. Studies or data within studies providing post-concussion assessment information beyond 14 days post-injury were excluded from the analysis. The 14-day post-concussion window was selected as the suggested time for neurometabolic recovery in humans following concussion.[29,30] Studies involving imaging techniques were not included in the analysis because sport concussion has been described as a functional change to the brain, rather than a structural change, resulting in normal imaging studies.[31] Included studies were separated

into three categories based on the type of outcome measure used to assess sport concussion, namely neurocognitive function, self-report symptoms and postural control. When possible, these main outcome categories were further broken down based on the time of the assessment of the sport concussion; that is, whether it was the initial or a follow-up assessment.

1.2 Data Extraction

Data were extracted from reports by one of the authors (SPB), checked by the second (TWP) and discrepancies resolved by consensus. Sample sizes as well as baseline and post-concussion means and standard deviations for the concussed and control groups were extracted from the investigations in order to calculate effect sizes. Effect sizes (Hedge's g) were calculated by subtracting the mean change (i.e. follow-up minus baseline) for a control group from the mean change for a concussed group and dividing this difference by the pooled standard deviation of baseline scores.[32] In those studies that did not include a separate control group (i.e. one group baseline-follow-up design), the effect sizes were calculated by subtracting the baseline score from the post-concussion score and dividing the difference by the baseline standard deviation. All effects sizes were adjusted using Hedge's small sample size bias correction before being entered into the analysis.[32] Effect sizes were calculated so that decreases in neurocognitive function and postural control and increases in self-reported symptoms resulted in negative effect sizes. In studies with multiple assessment points, in which multiple effects could be obtained, effects were averaged so that only one value contributed to the analysis.[33] When precise mean data were not reported, effect sizes were estimated[34] from F-tests,[35] p-values[36] or figures.[13,14,23,26,27,37-41]

Moderators extracted from the studies included the study design, type of neurocognitive technique used for assessment, time the post-concussion assessment was administered following injury and number of follow-up assessments post-concussion. The study design was categorized as those studies with control groups and those studies without control groups. The type of neurocognitive assessment technique was categorized as a pencil and paper test, computer test or the SAC. Although other moderator variables were considered, these variables were selected based on theoretical grounds as likely independent moderators and on the distribution of data in that adequate information was available to make meaningful interpretations. Furthermore, with the relatively small number of effects, the inclusion of too many variables could severely reduce the statistical power of the analysis limiting valid interpretations of moderating effects.[42,43] Information concerning subject demographics, the sport played, severity of injury, incidence of loss of consciousness and when post-concussion assessments were made were also extracted for descriptive purposes.

1.3 Assessment of Study Quality

The methodological quality of each study was assessed using a 15-item scale (1 point per item for a maximum of 15) as described elsewhere.[44] The scale addressed the fundamental aspects of the methods and reporting of clinical trials such as randomization, sample selection, outcome measures and statistical analysis. Quality assessment scoring was performed independently by the two authors (SPB and TWP) and showed high concordance between the two raters (intraclass correlation coefficient [3,2] = 0.91, 95% CI 0.83, 0.95).[45] Furthermore, using the Bland and Altman limits-of-agreement procedure, the average disagreement (mean; 95% CI) was close to zero (0.04; −0.14, 0.22) thus suggesting no evidence for a systematic disagreement bias between the two reviewers.[46,47] Quality scores were reported for each study for descriptive purposes (table I). Quality scores were not used as weights or moderators in the analysis because of the potential disparity that can exist in results depending on the specific quality scale employed.[48]

1.4 Statistical Methods

Statistical analyses were performed separately for those investigations assessing sport concussion based on (i) neurocognitive function; (ii) postural control; and (iii) self-reported symptoms. When possible, these categories were further broken down and analysed based on the initial assessment and any follow-up assessments. Analyses were performed

Table I. Characteristics of the 39 studies included in the meta-analysis

Study	Sample size; study design	Immediate post-injury					14 Days post-injury			Mean study quality score
		assessment	neurocognitive assessment	time from injury (days)[a]	effect size	95% CI	assessment	effect size	95% CI	
Barr and McCrea[49]	118; control	NC	SAC	0.003	−2.52	−2.82, −2.22				8.75
Bruce and Echemendia[50]	38; control	NC	P&P	0.08	−0.69	−1.30, −0.08	NC	−0.52	−1.13, 0.10	9.00
Collie et al.[18]	109; control	NC	P&P	2.2	−0.11	−0.56, 0.33				8.75
Collins et al.[51]	136; baseline–post-concussion	NC Sympt	Computer	2.17	−0.8 −1.34	−1.03, −0.57 −1.56, −1.13				7.75
Collins et al.[52]	78; baseline–post-concussion	NC Sympt	Computer	1.74	−0.86 −0.9	−1.16, −0.57 −1.19, −0.6				8.25
Cremona-Meteyard and Geffen[37]	21; control	NC	Computer	7	−0.23	−1.08, 0.62				7.50
Echemendia et al.[53]	49; control	NC Sympt	P&P	0.08	−1.47 −0.96	−1.98, −0.95 −1.50, −0.43	NC	−1.23	−1.76, −0.71	8.00
Erlanger et al.[17]	26; baseline–post-concussion	NC	Computer	NA	−0.91	−1.42, −0.40				5.25
Field et al.[54]	92; control	NC Sympt	P&P	1	−1.08 −1.48	−1.47, −0.69 −1.86, −1.11	NC Sympt	−0.6 −0.11	−1.00, −0.20 −0.53, 0.30	8.25
Guskiewicz et al.[38]	20; control	Sympt PC		1	−3.99 −1.26	−4.61, −3.37 −2.06, −0.45	PC	−0.52	−1.36, 0.33	6.75
Guskiewicz et al.[14]	22; control	NC PC	P&P	1	−0.44 −0.87	−1.26, 0.37 −1.65, −0.09	PC	−0.57	−1.37, 0.24	7.50
Guskiewicz et al.[27]	72; control	NC PC	P&P	1	−0.43 −1.5	−0.88, 0.02 −1.91, −1.09	NC PC	−0.04 −0.81	−0.50, 0.42 −1.25, −0.38	8.00
Guskiewicz et al.[39]	196; baseline–post-concussion	Sympt		0.003	−12.31	−12.45, −12.16	Sympt	−3.17	−3.32, −3.01	7.25
Hinton-Bayre et al.[55]	20; control	NC	P&P	1.5	−0.34	−1.19, 0.52				7.75
Hinton-Bayre et al.[40]	50; control	NC	P&P	2	−1.57	−2.08, −1.07	NC	−0.81	−1.35, −0.28	8.00
Iverson et al.[56]	41; baseline–post-concussion	NC	Computer	1.3	−0.72	−1.13, −0.30				6.50
Iverson et al.[35]	19; baseline–post-concussion	NC	Computer	1.6	−0.16	−0.79, 0.47				6.50
Iverson et al.[57]	30; baseline–post-concussion	NC Sympt	Computer	1.5	−0.88 −1.45	−1.36, −0.40 −1.91, −0.99	NC Sympt	−0.23 0	−0.73, 0.26 −0.51, 0.51	6.25

Continued next page

Table I. Contd

Study	Sample size; study design	Immediate post-injury					14 Days post-injury			Mean study quality score
		assessment	neurocognitive assessment	time from injury (days)[a]	effect size	95% CI	assessment	effect size	95% CI	
Lovell and Collins[21]	14; control	NC	P&P	1	−0.45	−1.59, 0.68				5.75
Lovell et al.[58]	88; control	NC Sympt	Computer	1.5	−1.04 −1.52	−1.48, −0.60 −1.95, −1.09	NC Sympt	−0.49 −0.2	−0.95, −0.03 −0.66, 0.27	7.25
Lovell et al.[59]	43; baseline–post-concussion	NC	Computer	1.4	−0.24	−0.66, 0.17	NC	0.4	−0.03, 0.83	6.75
Macciocchi et al.[60]	231; control	NC	P&P	1	−0.13	−0.45, 0.18	NC	0	−0.31, 0.32	8.75
Maddocks et al.[36]	56; control	NC	P&P	0.007	−0.95	−1.44, −0.46				7.50
Maddocks and Saling[61]	20; control	NC	P&P	5	−0.85	−1.68, −0.02				8.25
McClincy et al.[62]	104; baseline–post-concussion	NC Sympt	Computer	2.42	−0.86 −1.4	−1.12, −0.61 −1.64, −1.15	NC Sympt	−0.52 −0.4	−0.78, −0.26 −0.67, −0.14	6.25
McCrea et al.[63]	6; baseline–post-concussion	NC	SAC	0.003	−1.15	−1.95, −0.34				8.50
McCrea et al.[64]	601; control	NC	SAC	0.003	−1.1	−1.44, −0.75	NC	0.44	0.09, 0.80	8.50
McCrea[65]	118; control	NC	SAC	0.003	−1.65	−1.98, −1.33	NC	−0.19	−0.55, 0.16	8.50
McCrea et al.[66]	91; baseline–post-concussion	NC	SAC	0.003	−1.02	−1.29, −0.75	NC	−0.17	−0.46, 0.12	7.50
McCrea et al.[26]	150; control	NC Sympt PC	SAC	0.003	−1.35 −0.95 −0.96	−1.65, −1.05 −1.26, −0.64 −1.26, −0.64	NC Sympt PC	−0.23 −1.71 −0.15	−0.55, 0.10 −2.01, −1.42 0.48, 0.18	6.00
Moser and Schatz[67]	35; control	NC	P&P	7	−0.7	−1.35, −0.05				7.00
Moser et al.[68]	122; control	NC	P&P	3.5	−0.45	−0.81, −0.08				8.50
Pellman et al.[69]	95; baseline–post-concussion	NC	P&P	1.5	0.13	−0.15, 0.42				7.50
Pellman et al.[70]	48; baseline–post-concussion	NC Sympt	Computer	1.48	−0.42 −0.97	−0.81, −0.03 −1.35, −0.60	NC Sympt	−0.09 −0.65	−0.49, 0.31 −1.04, −0.27	6.00
Peterson et al.[13]	42; control	Sympt PC	P&P	1	−14.39 −8.83	−14.92, −13.86 −9.01, −8.65	Sympt PC	−2.66 −4.17	−3.16, −2.16 −4.60, −3.74	7.00
Piland et al.[23]	33; control	Sympt		1	−3.58	−4.09, −3.07	Sympt	−0.83	−1.48, −0.18	8.00
Riemann and Guskiewicz[41]	32; control	PC		1	−1.92	−2.53, −1.31	PC	−0.69	−1.36, −0.03	7.25
Schatz et al.[16]	138; control	NC Sympt	Computer	2	−0.88 −1.09	−1.20, −0.57 −1.40, −0.78				8.00
Warden et al.[71]	14; baseline–post-concussion	NC	Computer	4	−0.33	−1.06, 0.39				7.00

a When time from injury to initial assessment was <24 hours, the number of minutes were divided by the number of minutes in a day.

Computer = computer-based neurocognitive assessment; **NA** = data not available; **NC** = neurocognitive; **P&P** = pencil and paper neurocognitive assessment; **PC** = postural control; **SAC** = standardized assessment of concussion; **Sympt** = self-report concussion-related symptoms.

with Meta-analysis with Interactive eXplanations (MIX) version 1.3 (Sagamihara city, Kanagawa, Japan) and SPSS macros (SPSS version 13.0, SPSS Inc., Chicago, IL, USA). MIX version 1.3 was used to calculate the aggregated mean effect size, the associated 95% CI and the sampling error variance using a fixed-effects model. Heterogeneity and consistency were evaluated with the Q statistic and the I^2 statistic (i.e. estimates the percentage of variation across studies that cannot be attributed to chance or sampling error), respectively. Because of the liberal estimate of heterogeneity associated with the Q statistic, heterogeneity was further examined in terms of the percentage of observed variance accounted for by sampling error. Heterogeneity was indicated if the sampling error accounted for <75% of the observed variance.[32] When significant heterogeneity was found, the analysis was redone with a random-effects model. Publication bias was subjectively addressed by inspection of the funnel plot[72] on the outcome measure and quantified with the trim-and-fill method.[73,74]

Moderator variables were entered into weighted least squares multiple linear regression analyses to determine their independent effects ($p < 0.05$) on variation in effect size.[32] Three variables (i.e. study design, the type of neurocognitive assessment technique, time of assessment post-concussion) and two variables (i.e. study design, time of assessment post-concussion) were examined in the analysis of the initial assessment of neurocognitive function and self-reported symptoms post-injury, respectively. Three variables (i.e. study design, neurocognitive assessment technique, number of assessments post-concussion) and two variables (i.e. study design, number of assessments post-concussion) were examined in the analysis of the follow-up assessments for neurocognitive function and self-report symptoms post-injury, respectively. The limited number of studies (6 of 39) examining postural control post-concussion provided a sub-optimal dataset for testing moderators of the effect; thus, such analyses were not conducted. A macro (SPSS version 13.0)[75] was used for the analyses, which employed a random-effects model to account for between-study heterogeneity associated with both study-level sampling error and random effects variance. Each effect was weighted by the inverse of its variance and then recalculated with the random-effects variance component added. Significant moderators in the regression analysis were decomposed using a random-effects model to compute effect sizes and 95% CIs.[75]

Sport-related concussion studies that concurrently measured neurocognitive function and self-reported symptoms were selected from the examined literature in order to directly compare the magnitude of the effects between the two most common types of post-concussion outcome measures.[10] Effect sizes were calculated as previously described. Neurocognitive function and self-reported symptom effect sizes were then dummy coded. A macro (SPSS version 13.0) was used to compute aggregated effect sizes adjusted for sample size, calculated the 95% CI and tested the significance of coded effect-size variables.[75] Differences between the effects for neurocognitive function and self-reported symptoms were determined using the Q_B statistic.[32] The analysis employed a random-effects model in which the effects were weighted by the inverse of their variance and recalculated with the random-effects variance component added. Contrasts were tested at $p < 0.05$.

2. Results

2.1 Search Findings

The search strategy yielded 3364 citations, from which 89 studies examining sport-related concussion were targeted for detailed review. The most common reasons for exclusion were (i) lack of adequate information to calculate effect sizes; (ii) the first post-concussion assessment was completed later than 14 days following injury; and/or (iii) the study design did not include a baseline assessment or control group (figure 1). Of the 89 trials examined in detail, 39 trials met the inclusion criteria for the meta-analysis, with some reporting more than one assessment technique of concussed athletes (table I); 34 used a post-concussion assessment of neurocognitive function, 14 used post-concussion assessment of self-reported symptoms, and six used post-concussion assessment of postural control.

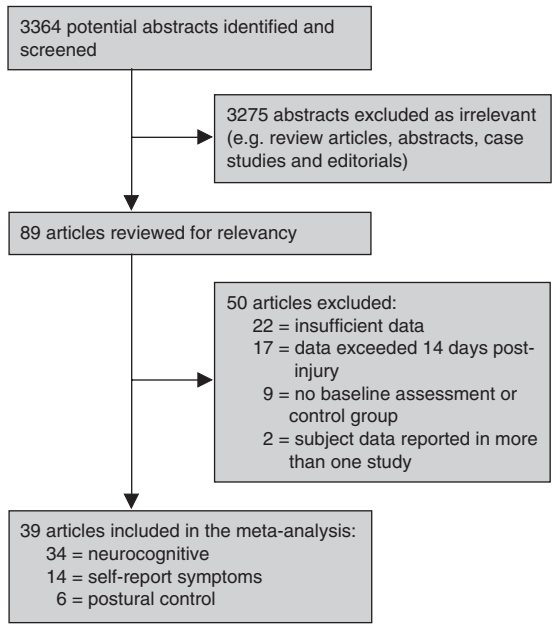

Fig. 1. Selection of sport concussion manuscripts. Some articles included in the analysis reported more than one assessment technique of the concussed athletes.

2.2 Characteristics of Included Studies and Quality Assessment

Participant characteristics of the studies included in the initial and follow-up assessments of neurocognitive function, self-report symptoms, and postural control are presented in table II. A total of 4145 concussed and control participants were evaluated in the 39 studies included for analysis. These individuals were primarily male (92.9%), American football athletes (72.0%) with a mean age of 19.0 ± 0.40 years.

2.3 Neurocognitive Function: Post-Concussion Outcomes and Moderators of the Effect

2.3.1 Initial Assessment

Post-concussion outcomes: the distribution of the 34 effects was negatively skewed (g1 = −0.99) and leptokurtic (g2 = 2.04). Thirty-three of the 34 effects (97.1%) were less than zero. The mean effect size Δ was −0.81 (95% CI −1.01, −0.60). The evidence showed a significant decrease in cognitive function in the initial assessment post-concussion (z = 7.73, p < 0.001). The effect was heterogeneous ($Q_{T(33)}$ = 271.53, p < 0.001). Sampling error accounted for 17.8% of the observed variance. The effect also was not consistent across studies (I^2 = 87.9%; 95% CI 84.1, 90.7).

Moderator analysis: the regression model containing study design, the type of neurocognitive technique used at assessment and time of assessment post-concussion was significantly related to effect size ($Q_{R(3)}$ = 28.71, p < 0.001, R^2 = 0.52 and $Q_{E(29)}$ = 26.36, p = 0.61). Study design (β = 0.53, z = 3.31, p = 0.001) and the neurocognitive assessment technique (β = 0.45, z = 4.02, p < 0.001), but not time of assessment post-concussion (β = 0.03, z = 0.72, p = 0.47), were independently related to the effect. Decomposition of the study-design variable showed that there was a larger effect (Δ; 95% CI) for studies that used a control group (−0.92; −1.18, −0.66) than studies without a control group (−0.63; −0.95, −0.32). Decomposition of the neurocognitive assessment technique variable showed that there was a larger effect for studies that used the SAC (−1.49; −1.85, −1.12) than studies that used either pencil and paper (−0.61; −0.85, −0.36) or computer (−0.70; −0.96, −0.43) tests.

Table II. Demographic information for each analysis based on the number of included studies

Time of assessment	No. of studies[c]	Total sample[d]	Age (y) [mean ± SD]	Male athletes (%)	Sport (%)[a]						Concussion severity (%)[a,b]			LOC (%)	Time to initial assessment (days) [mean ± SD]	No. of follow-up assessments [mean ± SD]
					AF	R	S	IH	BB	other[e]	grade I	grade II	grade III			
Neurocognitive assessment																
Initial	34	3761	18.7 ± 3.0	93.2	62.9	20.0	3.3	2.8	2.5	8.6	63.4	27.2	9.2	17.8	1.7 ± 1.8	
Follow-up	15	2121	19.0 ± 3.0	95.1	74.2	6.7	4.2	4.8	3.7	6.2	62.1	28.9	8.9	12.2		2.9 ± 1.3
Self-report symptom assessment																
Initial	14	1796	18.5 ± 3.0	91.3	79.3		7.8	4.2	5.0	4.1	34.4	58.4	7.0	12.0	1.2 ± 0.8	
Follow-up	9	1161	19.5 ± 3.5	92.9	83.6		9.3	3.6	0.5	3.1	34.4	58.4	7.0	10.6		4.0 ± 1.9
Postural control assessment																
Initial and follow-up	6	338	19.2 ± 1.0	84.3	59.8		5.3	2.5		6.4	38.1	56.5	5.4	9.8	0.8 ± 0.4	4.3 ± 1.4

a Percentages may not add to 100% due to rounding or unreported data.
b Concussion severity is based on reported scales that included the American Academy of Neurology, Colorado Medical Society, and Cantu guidelines.
c Data from multiple assessment techniques were extracted from some studies. See table I for details.
d Total sample = concussed and control.
e Other sports may include wrestling, lacrosse, cheerleading and/or equestrian.
AF = American football; **BB** = basketball; **IH** = ice hockey; **LOC** = loss of consciousness; **R** = rugby; **S** = soccer.

2.3.2 Follow-Up Assessment

Post-concussion outcomes: the distribution of the 15 effects was negatively skewed (g1 = −0.31) and leptokurtic (g2 = 0.60). Twelve of the 15 effects (80.0%) were less than zero. The mean effect size Δ was −0.26 (95% CI −0.46, −0.06). The evidence showed a significant decrease in cognitive function in the follow-up assessments post-concussion (z = 2.59, p = 0.001). The effect was heterogeneous ($Q_{T(14)} = 54.68$, $p < 0.001$). Sampling error accounted for 28.7% of the observed variance. The effect also was not consistent across studies ($I^2 = 74.4\%$; 95% CI 57.5, 84.6).

Moderator analysis: the regression model containing study design, the type of neurocognitive technique used at assessment and the number of assessments taken post-concussion was not significantly related to effect size ($Q_{R(3)} = 3.83$, $p = 0.22$, $R^2 = 0.25$ and $Q_{E(11)} = 11.12$, $p = 0.43$). Neither the study design ($\beta = 0.03$, $z = 0.13$, $p = 0.90$) nor the number of assessments taken post-concussion ($\beta = 0.06$, $z = 0.67$, $p = 0.50$) were independently related to the effect; however, the type of neurocognitive assessment technique neared significance ($\beta = 0.24$, $z = 1.84$, $p = 0.08$). Decomposition of the type of neurocognitive assessment technique showed that there was a larger effect (Δ; 95% CI) for studies that used a pencil and paper test (−0.50; −0.83, −0.16) than studies that used either computer (−0.19; −0.55, 0.14) or the SAC (−0.04, −0.41, 0.33) tests.

2.4 Self-Reported Symptoms: Post-Concussion Outcomes and Moderators of the Effect

2.4.1 Initial Assessment

Post-concussion outcomes: the distribution of the 14 effects was negatively skewed (g1 = −2.13) and leptokurtic (g2 = 3.44). All 14 effects (100.0%) were less than zero. The mean effect size Δ was −3.31 (95% CI −6.35, −0.27). The evidence showed a significant increase in self-reported symptoms in the initial assessment post-concussion (z = 2.13, p = 0.03). The effect was heterogeneous ($Q_{T(13)} = 17\,463.68$, $p < 0.001$). Sampling error accounted for 0.1% of the observed variance. The effect also was not consistent across studies ($I^2 = 99.9\%$; 95% CI 99.8, 99.9).

Moderator analysis: the regression model containing study design and the time of assessment post-concussion was not significantly related to effect size ($Q_{R(2)}$ = 2.03, p = 0.36, R^2 = 0.17 and $Q_{E(11)}$ = 10.17, p = 0.52). Neither study design nor the time of assessment post-concussion were independently related to the effect.

2.4.2 Follow-Up Assessment

Post-concussion outcomes: the distribution of the nine effects was negatively skewed (g1 = −1.02) and platykurtic (g2 = −0.43). Eight of the nine effects (88.9%) were less than zero. The mean effect size Δ was −1.09 (95% CI −2.07, −0.11). The evidence showed a significant increase in self-reported symptoms in the follow-up assessments post-concussion (z = 2.17, p = 0.03). The effect was heterogeneous ($Q_{T(8)}$ = 600.66, p < 0.001). Sampling error accounted for 2.1% of the observed variance. The effect also was not consistent across studies (I^2 = 98.7%; 95% CI 98.2, 99.0).

Moderator analysis: the regression model containing study design and the number of assessments taken post-concussion was significantly related to effect size ($Q_{R(2)}$ = 10.34, p = 0.01, R^2 = 0.57 and $Q_{E(6)}$ = 7.74, p = 0.26). The number of assessments taken post-concussion (β = −0.43, z = 2.92, p = 0.004) was independently related to the effect with an increased number of post-concussion assessments being associated with a decreased number of self-reported symptoms. The study design (β = 0.39, z = 1.02, p = 0.31) was not independently related to the effect.

2.5 Postural Control: Post-Concussion Outcomes and Moderators of the Effect

2.5.1 Initial Assessment

Post-concussion outcomes: the distribution of the six effects was negatively skewed (g1 = −2.37) and leptokurtic (g2 = 5.68). All six of the effects (100%) were less than zero. The mean effect size Δ was −2.56 (95% CI −6.44, 1.32). The evidence showed a non-significant decrease in postural control in the initial assessment post-concussion (z = 1.29, p = 0.19). The effect was heterogeneous ($Q_{T(5)}$ = 2902.17, p < 0.001). Sampling error accounted for 0.4% of the observed variance. The effect also was not consistent across studies (I^2 = 99.8%; 95% CI 99.7, 99.9).

2.5.2 Follow-Up Assessment

Post-concussion outcomes: the distribution of the effects was negatively skewed (g1 = −2.35) and leptokurtic (g2 = 5.60). All six effects (100.0%) were less than zero. The mean effect size Δ was −1.16 (95% CI −2.59, 0.27). The evidence showed a non-significant decrease in postural control in the follow-up assessments post-concussion (z = 1.59, p = 0.11). The effect was heterogeneous ($Q_{T(5)}$ = 230.31, p < 0.001). Sampling error accounted for 1.6% of the observed variance. The effect also was not consistent across studies (I^2 = 97.8%; 95% CI 96.7, 98.6).

2.6 Direct Comparison of Effects

A total of ten sport-related concussion studies (subjects = 913) concurrently measured neurocognitive function and self-reported concussion symptoms at the initial assessment post-concussion.[16,26,51-54,57,58,62,70] The ten studies had an average sample size of 71 (range = 30–156). There was a significant difference in effect sizes between the measures ($Q_{B(1)}$ = 5.28, p = 0.02). The effect (mean Δ; 95% CI) associated with increases in self-reported symptoms at the initial assessment post-concussion (−1.21; −1.36, −1.05) was significantly greater than the effect associated with decrements in neurocognitive function at the initial assessment post-concussion (−0.95; −1.10, −0.79).

2.7 Assessment of Publication Bias

The funnel plots for all six meta-analyses (i.e. initial and follow-up assessment analyses for neurocognitive function, self-reported symptoms and postural control) were inspected and found to be approximately symmetric, suggesting absence of publication bias. The trim-and-fill analyses also suggested that all six meta-analyses were free of publication bias in that zero studies were imputed in order to reach symmetry. Thus, the resulting effect outcomes were not changed in any of the analyses.

3. Discussion

This meta-analysis sought to elucidate the effects of sport concussion as measured by three common

clinical assessment measures. Studies investigating changes to neurocognitive functioning, self-report symptoms and postural control both immediately following and within the first 14 days of injury were included in the analyses. In addition, we directly compared the magnitude of the effect between neurocognitive and self-report symptom measures and evaluated the moderating effects of study design, type of neurocognitive assessment technique, the time from concussion to the first assessment, and the number of post-concussion assessments. Our analyses indicated sport concussion had a large negative effect on neurocognitive functioning ($\Delta = -0.81$) when evaluated immediately following injury. In the 14 days following injury, the effect was reduced ($\Delta = -0.26$). Large negative effects for self-report symptoms also were seen in both the immediate post-concussion assessment ($\Delta = -3.31$) and those administered during the 14 days post-injury ($\Delta = -1.09$). Finally, the effect on postural control was large immediately following injury ($\Delta = -2.56$) and in the 14 days following the initial assessment ($\Delta = -1.16$).

A detailed description of each component of the assessment battery follows. The neurocognitive, self-report symptoms and postural control assessments were included for analysis as clinical tools that are easily administered and interpreted by sports medicine clinicians. Each evaluative technique has been recommended by the National Athletic Trainers' Association[8] and a summary agreement from the International Conference on Concussion in Sport.[9] The clinician's understanding of the advantages and disadvantages of each component of the post-concussion assessment battery can lead to a more accurate diagnosis and safe return to play.

3.1 Neurocognitve Assessment

A neurocognitive assessment following sport concussion is suggested to provide the greatest amount of information to the clinician[11] and is recommended as the cornerstone of the concussion evaluation.[31] The effects of sport concussion we witnessed were large, but slightly smaller than those previously reported both immediately following concussion and within the first 10 days of injury.[28] Discrepancies in effect sizes may have been related to differences in the number of post-injury days included in the analysis. We included more post-injury days (i.e. 14 days) in our analysis to account for complete neurometabolic recovery following concussion.[29,30] Similar to Belander and Vanderploeg,[28] our analysis found the addition of a control group had a moderating effect on outcomes. This finding would suggest that future investigations should include control participants within the study design to better clarify the effects of concussion. Control subjects evaluated at identical timepoints to the concussed athletes allows investigators to account for practice effects reported to occur in some tests when administered multiple times.[13,56,76]

Significant independent moderating effects were found for time from the injury to the initial assessment and the neurocognitive assessment technique used (i.e. pencil and paper, computer or SAC tests). This would suggest that comparing individuals or groups that have not been evaluated at identical timepoints post-injury may influence outcomes. Previous works have shown a rapid recovery from sport concussion in the first 10–14 days following injury,[26,60,77] and comparing groups at different timepoints may make the valid interpretation of results within and across studies more difficult. In addition, decomposition of the assessment technique found the largest effect associated with the SAC in the immediate post-concussion assessment. This finding is supported by a previous work in which the SAC demonstrated high sensitivity to concussion immediately following injury when used as part of a brief battery of concussion assessment tests.[19] When contrasted with the other assessment techniques, the effects demonstrated by the SAC may be related to administration time subsequent to injury. In many instances, pencil and paper tests and computerized assessments were administered in the following days and not on the sideline after injury (table I). In studies where several days passed before the initial assessment of more traditional neurocognitive assessment techniques occurred, injury recovery may have reduced effect sizes when compared with SAC administration immediately following injury.

When evaluating the effects seen in the 14 days following injury, the neurocognitive assessment showed the smallest effects compared with the other evaluation techniques. Assessments taken at this

timepoint were not influenced by any moderating variable. The neurocognitive assessment technique approached significance ($p = 0.08$), with the pencil and paper test battery showing the largest effect. Such a trend has been supported by a previous work that found an increasing sensitivity of a pencil and paper assessment battery during the first 7 days post-injury.[19]

These results would suggest that the SAC may provide the sports medicine clinician with the greatest amount of information pertaining to neurocognitive status immediately following injury. In the days following concussion, as the sensitivity of the SAC declines, a pencil and paper assessment battery should be considered. Since each of these tests require a separate baseline assessment for proper clinical interpretation, the clinician must balance time spent versus the information gained by using both assessment techniques. In addition, one's clinical practice may not necessitate the use of a sideline test or permit access to an individual trained to interpret pencil and paper tests. Further research is needed to examine the sensitivity of post-concussion assessment techniques over time and the associated cost-benefit analysis of each of these techniques.

3.2 Self-Reported Symptoms

Sport concussion showed the largest effect on self-report symptoms at the immediate post-concussion assessment. This effect was significantly greater than the effect on neurocognitive performance in the ten studies that concurrently measured self-report symptoms and neurocognitive performance immediately following injury. In addition, the effect on self-report symptoms occurred without study design or time of initial assessment post-injury significantly moderating the effect. While it would appear self-report symptoms would clearly indicate the presence of concussion, caution is warranted in its use. Unlike other assessment techniques in the assessment battery, concussion-related symptoms are subjectively reported by the athlete. One study suggested that over one-third of unreported concussions may result from the athlete not being aware of the injury's signs and symptoms.[25] In addition, it has been proposed that some athletes may deliberately under-report concussion-related symptoms in an effort to return to play sooner.[24]

The effect of concussion on self-report symptoms during the first 14 days following injury found the number of assessments following injury to be a significant moderator. This would indicate that as more post-concussion assessments were administered, the number of symptoms decreased. This phenomenon has been reported previously and was not unexpected.[39] Neurocognitive functioning, however, was not moderated by the number of assessments, suggesting a disconnect between symptom resolution and neurocognitive recovery. One group has suggested that self-report concussion-related symptom resolution can be used as the sole return-to-play decision in some instances.[9] Based on our findings, this clinical protocol is not warranted. The tracking of self-report symptoms following concussion should continue as it is inexpensive, easy to administer, and can be used in all clinical settings. However, symptom findings should be used in conjunction with a concussion assessment battery that evaluates multiple aspects of cerebral functioning.

3.3 Postural Control

The postural control measures also garnered large effects immediately following injury and during the 14 days following the initial assessment. Clinical inferences from this analysis, however, are limited by an insufficient number of published studies in this area. Nevertheless, the large effects seen in this analysis suggest further research is warranted. Postural control assessments such as the SOT or the BESS are now supported by the National Athletic Trainers' Association[8] as part of the concussion assessment battery. Postural control decrements following concussion are documented and are reported to result from a sensory integration deficit in the balance mechanism.[14,27,78] When used soon after injury, one author reports a postural control test to be more sensitive to concussion than some pencil and paper tests.[14] Balance decrements typically resolve 3–5 days post-injury,[26,27] but resolution time may be dependent on the measurement device. Instrumented measures of postural control, such as the NeuroCom SOT, are likely more sensitive than uninstrumented measures; however, the cost of the device makes its use prohibitive and it cannot be used for sideline assessments. A less expensive postural control assessment (BESS) has also been re-

ported.[26,27] Use of either postural control assessment technique is warranted as demonstrated by large post-concussion effects and the additional clinical information may provide a better understanding of the injury.

3.4 Strengths and Limitations

Despite a large increase in the number of sport concussion publications in the previous decade, evaluation of the injury remains difficult. This meta-analysis sought to clarify the effects concussion has on various aspects of the concussion battery and is the first to evaluate all aspects of a standard assessment battery both immediately following concussion and in the subsequent days. Some limitations were present in this study that restricted the thoroughness of the analyses. Inconsistent reporting of demographic information such as concussion severity was common and the majority of the studies we included used high-school and collegiate American football athletes. Future research should focus on the adolescent athlete, as well as females and other sports. Youth athletes have been shown to take longer to recover from injury than their older counterparts[54] and one author has suggested that female athletes experience greater declines in neurocognitive performance and report more concussion-related symptoms following injury than their male counterparts.[79] Too few studies were available to investigate the effects of either of these variables. Finally, we were only able to include six studies evaluating postural control following sport concussion. Based on our findings, the use of postural control as an assessment measure is warranted, but additional studies are needed to better clarify these changes following injury.

4. Conclusion

This meta-analysis clearly supports the use of an assessment battery in the evaluation of sport concussion. The clinical examination remains the gold standard for concussion evaluation[11] with each aspect of the battery providing supporting information. The battery components may vary, but an assessment of neurocognitive functioning, self-report symptoms, and postural control all warrant inclusion and no single test should be used or interpreted in exclusion of the others. When evaluating concussion immediately post-injury, the largest effects were seen using the SAC assessment for cognitive status. These effects lessened as more assessments were administered within the first 14 days of the injury. During the 2-week follow-up period, the largest neurocognitive effects were seen using the pencil and paper assessment battery. While computer-based assessments are now more commonly utilized for concussion assessment,[10] they did not yield the same effect as their pencil and paper counterparts. Computer-based evaluations are a novel addition to the concussion assessment protocol and are still undergoing development. The evolution of these instruments will require a thorough examination of their validity and reliability before they become an equivalent assessment tool.[80]

The largest effects in the immediate post-concussion assessment were demonstrated with self-report symptoms. While this would appear to be the obvious choice for concussion assessment, their use presupposes accurate reporting by the concussed athlete.[24,25] Symptom reporting is also influenced by the number of post-concussion assessments, suggesting that this tool is best suited only as a guide for recovery and not a definitive tool. Although not investigated here, the potential for practice/learning effects exists with multiple administrations of neurocognitive[56,76] and postural control tests.[13,81] Sports medicine personnel can reduce the number of test administrations by using a daily assessment of symptoms until the athlete reports symptom free. Once asymptomatic, the postural control and neurocognitive assessment can be administered to evaluate for complete recovery from injury.[8] Only once the athlete performs at or above the baseline level of functioning and is symptom-free at rest and during exertion should a full return to play be considered.[82]

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