NCC Pediatrics Continuity Clinic Curriculum:
Sports Physical II: Special Topics
Faculty Guide

Goals & Objectives:

To understand the importance of the pre-participation exam and to gain the skills necessary to perform an adequate exam and recognize common problems.

- Understand presentation and management of concussion, including baseline neuro-psychological testing and a strict return-to-play protocol with cognitive and physical rest.
- Gain a better understanding of the importance of the cardiovascular risk factors elicited in the history and a greater understanding of the importance of the cardiovascular exam.
- Become familiar with classification of sports by contact vs. noncontact and by levels of dynamic components in order to adequately counsel potential athletes.
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Pre-Meeting Preparation:

Please read the following enclosures:

- "Pediatric Sport-related Concussion: Recommendations From the Amsterdam Consensus Statement 2023 *(excerpt, Pediatrics, 2024)*
- "Athlete Screening and Sudden Cardiac Death" (*PIR, 2023)*
- Review Tools
  - 36th Bethesda Conference Sports Classifications
  - AHA 14 and PPE-4 Monograph
  - SCAT6: Sports Concussion Assessment Tool (2017)
  - **Heads Up** (CDC Concussion Program) and ACE Tool

Conference Agenda:

- Review Sports Physical II Quiz
- Complete Sports Physical II Cases
- **Exercise:** Perform SCAT6 w/partner.

Post-Conference: *Board Review Q&A*

Extra-Credit:

- "Demystifying the Pediatric Electrocardiogram: Tools for the Practicing Pediatrician" *(PIR, 2021)*
- "Diagnosis and Management of Mild Traumatic Brain Injury in Children: A Systematic Review" *(JAMA Peds, 2018)*
- "Sudden Death in the Young: Information for the Primary Care Provider" *(AAP Policy Statement, 2021)*
- "Association Between Early Return to School Following Acute Concussion and Symptom Burden at 2Weeks Postinjury" *(JAMA Pediatrics, 2022)*
- "Characteristics and Outcomes of Athletes With Slow Recovery From Sports-Related Concussion" (*Neurology, 2023)*

- **Local Programs:** S.C.O.R.E. @ CNMC; Kennedy Krieger Neurorehab Clinic

Pediatric Sport-related Concussion: Recommendations From the Amsterdam Consensus Statement 2023

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abstract

The 6th International Consensus Conference on Concussion in Sport, Amsterdam 2022, addressed sport-related concussion (SRC) in adults, adolescents, and children. We highlight the updated evidence-base and recommendations regarding SRC in children (5–12 years) and adolescents (13–18 years). Prevention strategies demonstrate lower SRC rates with mouthguard use, policy disallowing bodychecking in ice hockey, and neuromuscular training in adolescent rugby. The Sport Concussion Assessment Tools (SCAT) demonstrate robustness with the parent and child symptom scales, with the best diagnostic discrimination within the first 72 hours postinjury. Subacute evaluation (>72 hours) requires a multimodal tool incorporating symptom scales, balance measures, cognitive, oculomotor and vestibular, mental health, and sleep assessment, to which end the Sport Concussion Office Assessment Tools (SCOAT6 [13+] and Child SCOAT6 [8–12]) were developed. Rather than strict rest, early return to light physical activity and reduced screen time facilitate recovery. Cervicovestibular rehabilitation is recommended for adolescents with dizziness, neck pain, and/or headaches for greater than 10 days. Active rehabilitation and collaborative care for adolescents with persisting symptoms for more than 30 days may decrease symptoms. No tests and measures other than standardized and validated symptom rating scales are valid for diagnosing persisting symptoms after concussion. Fluid and imaging biomarkers currently have limited clinical utility in diagnosing or assessing recovery from SRC. Improved paradigms for return to school were developed. The variable nature of disability and differences in evaluating para athletes and those of diverse ethnicity, sex, and gender are discussed, as are ethical considerations and future directions in pediatric SRC research.

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International sports organizations have worked collaboratively with the Concussion in Sport Group to hold quadrennial consensus conferences and produce a summary or consensus statement[1–6] from each meeting that summarizes the evidence and makes recommendations regarding sport-related concussion (SRC) in adults, adolescents, and children. A core component informing the last 2 conferences and consensus statements has been a series of systematic reviews published with the Consensus Statement. In Berlin 2016, 1 of the 12 systematic reviews was

dedicated to pediatric concussion.[7] At the 6th International Consensus Conference on Concussion in Sport, Amsterdam 2022, the scientific committee incorporated pediatrics into each systematic review, rather than produce a stand-alone pediatric systematic review. In addition, pediatric concussion was included as a separate section in the Consensus Statement. Multiple clinicians and scientists with experience in pediatric concussion were coauthors of each systematic review to ensure that pediatrics was adequately addressed. Each review extracted data specific to children (<13 years) and adolescents (13 to <18 years), resulting in significant pediatric data being acquired. This paper provides a dedicated pediatric publication consolidating each component of the Amsterdam Consensus Statement pertaining to child and adolescent SRC, informed by the systematic reviews.

The scientifically rigorous consensus methodology and details on the systematic reviews and associated processes are described in detail by Schneider et al.[8] The definition of SRC was updated as part of the Amsterdam process and is described in the Appendix 1.[9]

PEDIATRIC FINDINGS FROM THE SYTEMATIC REVIEWS

Prevention Strategies and Modifiable Risk Factors for Sport-related Concussions and Head Impacts: A Systematic Review and Meta-analysis[10]

Adolescents were the target populations in over 50% of the published studies evaluating SRC prevention strategies and/or modifiable risk factors,[10] with few focused on the 5 to 12 year age group alone.[6] Concussion prevention strategies for children and adolescents highlight the role of personal protective equipment, policy and/or rule changes, training strategies, and management strategies targeting recurrent concussion.

Protective Equipment

Studies evaluating headgear (as opposed to helmets) in football (soccer), Australian football, and Rugby Union (rugby) report mixed findings regarding their protective effect against concussion. When data were combined in a meta-analysis across adolescent studies in rugby, soccer, and lacrosse, headgear was not significantly associated with SRC rates (IRR [incidence rate ratio] = 0.74; 95% confidence interval [CI]: 0.5–1.09).[10,11] By sport, headgear use was associated with lower SRC rates in the meta-analysis combining 2 soccer studies (IRR = 0.64; 95% CI: 0.44–0.92)[10,12,13]; however, further evaluation in larger studies evaluating different headgear design and materials is necessary to inform any recommendation. In helmeted adolescent collision sports, evidence indicates that secure helmet fit may reduce concussion rates and severity in ice hockey and American football.[14,15] The protective effect of mouthguards has been demonstrated in adolescent ice hockey (IRR = 0.75; 95% CI: 0.64–0.88).[16] Protective eyewear in adolescent field hockey reduced head and face injuries, but did not reduce SRC rates (IRR = 0.96; 95% CI: 0.57–1.59; IRR = 0.77; 95% CI: 0.58–1.02).[17,18]

Policy or Rule Changes

A combined 58% lower concussion rate was identified where policy disallowed bodychecking in child and adolescent ice hockey leagues (IRR = 0.42; 95% CI: 0.33–0.53).[10] Further, number of years of body checking experience was not protective for concussion,[19] thus disallowing body checking for children and most levels of adolescent ice hockey is recommended. Restricting the frequency and/or duration of collision practices in adolescents in American football reduced head contact (IRR = 0.22; 95% CI: 0.21–0.23)[20] and practice-related concussion rates (IRR = 0.44; 95% CI: 0.25–0.75).[21] The positive effect of limiting body checking in ice hockey and restricting contact practice in American football in reducing SRC rates may be a consideration across a range of adolescent collision sports.

Training Strategies

On-field neuromuscular training (NMT) warm-up strategies (eg, balance, strength, agility) have been demonstrated to be effective in reducing injuries across multiple adolescent team sports.[22] When compared with the standard practice warm-up, NMT inclusive of a neck strengthening component was associated with a 59% lower SRC rate in school-boy (ages 14–18) rugby players (risk ratio = 0.41; 90% CI: 0.17–0.99) when completed ≥3 times per week.[23] The effect of NMT programs for specifically reducing concussion rates has not been assessed in other sports and a focus on evaluating specific NMT components for concussion prevention is necessary in children and adolescents.

Concussion Management

Evidence supports implementation of concussion laws (eg, mandatory removal from play, requirements to receive clearance to return to play from a licensed health care professional (HCP), and education of coaches, parents, and athletes) to reduce recurrent concussions in adolescent sports.[24,25]

Acute Evaluation of Sport-related Concussion and Implications for the Sport Concussion Assessment Tool (SCAT6) for Adults, Adolescents, and Children: A Systematic Review[26]

Examination of pediatric age groups (5–12, 13–18 years) at the acute stage (<72 hours) of concussion assessment revealed a significant disparity in the literature. For children ages 5 to 12 years, only 5 eligible studies[27–31] examined Child SCAT tool utility, with none SRC-focused. Specifically, the cognitive measures of the Child SCAT demonstrated low test-retest stability,[28] implying limited

clinical utility, and there was no comparison of injured versus uninjured children. More robust psychometric characteristics are reported for the parent and child symptom scales,[31] with solid evidence of internal consistency and stability over time,[27,28] as well as strong differentiation of concussed athletes from controls.[31] The modified Balance Error Scoring System balance examination exhibits variability,[28,32] with promise for improved reliability with more systematic training methods. A significantly greater body of literature supports the discriminatory ability of the SCAT tools in adolescents within the first 72 hours of injury, with its utility diminishing by 7 days.

Routine, across-the-board, mandatory baseline testing was not recommended in children because of rapid developmental changes through childhood and adolescence, although it could be considered for older athletes and parasport athletes in competitive sport settings if resources permit.[8] If baseline testing is conducted, it requires (1) appropriate resources (ie, trained personnel) to conduct the testing effectively, and (2) use of measures with robust appropriate psychometrics (ie, reliable change metrics) to demonstrate meaningful clinical change for the individual.

A significant need exists for developmentally appropriate concussion tools spanning the full age range of children and adolescents.[33,34] Additional recommendations include collecting more diversified, global normative data for all ages, including subgroups with developmental and psychiatric diagnoses. Focused efforts are needed to study tools in the under-12 age group in SRC cases and controls and to expand settings to emergency departments and primary care for younger age samples. Modifying cognitive (eg, timed components) and balance measures (eg, dual task) can enhance their diagnostic sensitivity.

Beyond Acute Concussion Assessment to Office Management: A Systematic Review Informing the Development of a Sport Concussion Office Assessment Tool (SCOAT6) for Adults and Children[35]

Several assessment tools have been used to diagnose SRC in children and adolescents, including symptom scales, balance measures, cognitive tests, and oculomotor and vestibular tests. Symptom scales reliably distinguished between concussed and nonconcussed athletes in the acute and subacute (3–30 days) periods post-SRC.[36–42] Balance assessment with the modified Balance Error Scoring System significantly differentiated between concussed adolescent athletes and controls in the subacute period, with more errors in concussed athletes,[43] and complex tandem gait assessments elicited significantly more sway or errors in concussed subjects compared with controls in the subacute period.[43] Concussed athletes performed tandem gait slower than controls for both single-task and dual-task conditions and demonstrated worse dual-task cognitive accuracy.[44–46] Similarly, Vestibular Ocular Motor Screening components were significantly different in concussed adolescents compared with baseline measures and with healthy controls 0 to 14 days postinjury.[36,47–49] The Visio-Vestibular Examination assesses visio-vestibular function, including complex tandem gait, and has been validated for use in the diagnosis of concussion in children.[50]

Two new office assessment tools were developed as part of the Amsterdam consensus to assist in the assessment of children and adolescents with concussion in the subacute period. The Sport Concussion Office Assessment Tool (SCOAT6) was designed for ages 13 years and older and the Child SCOAT6 was developed for children aged 8 to 12 years.[51,52] As with the SCAT6 and Child SCAT6, these tools are designed for use by HCPs.

Rest and Exercise Early After Sport-related Concussion: A Systematic Review and Meta-analysis[53]

This review synthesized the best evidence on the risks and benefits of early physical activity (PA), prescribed aerobic exercise treatment, rest, cognitive activity, and sleep during the first 14 days after SRC. Most papers reviewed included the pediatric age group, and although some included children ≤12 years among the larger cohort, the majority assessed adolescents and young adults. There was no evidence that strict physical and cognitive rest until complete symptom resolution (so called "cocooning") facilitated recovery from SRC. In a meta-analysis, PA and prescribed individualized exercise treatment (based on systematic exercise testing) improved recovery by a mean of 4.64 days (95% CI 6.69–2.59).[53] During the first 2 days after SRC, early return to light PA (eg, walking) and reduced screen time followed by prescribed aerobic exercise treatment (days 2–14) safely facilitated recovery, whereas sleep disturbance was associated with slower recovery.[53] Prescribed aerobic exercise treatment within 14 days of SRC also significantly reduced the incidence of concussive symptoms persisting beyond 30 days and the associated reduced quality of life and learning difficulties in school. Aerobic exercise was found to also benefit those with persisting symptoms beyond 1 month. The data confirmed that brief, mild concussion symptom exacerbation (ie, no more than a 2-point increase on a 0–10 scale when compared with the preactivity level for no more than an hour) during physical or cognitive activity is not harmful and does not delay recovery.[53] Despite current evidence predominantly involving adolescents, evidence suggests that strict rest until symptom resolution may delay recovery in children.[53–57] As such, early PA and subsymptom threshold aerobic exercise in children should align with the paradigm in adolescents until age-specific data become available.

Targeted Interventions and Their Effect on Recovery in Children, Adolescents, and Adults Who Have Sustained a Sport-related Concussion - A Systematic Review[58]

Much of the literature evaluating rehabilitation strategies after SRC included adults and adolescents, with few studies including children. Light aerobic activity should be started as soon as 2 days after SRC. The athlete does not need to be "cleared" for subsymptom threshold aerobic exercise. Cervicovestibular rehabilitation is recommended for adolescents with dizziness, neck pain, and/or headaches for greater than 10 days, and may decrease time to medical clearance for return to sport.[59,60] Adolescents experiencing dizziness for more than 5 days may benefit from vestibular rehabilitation.[61] Active rehabilitation and collaborative care for adolescents with persisting symptoms for more than 30 days may decrease symptoms.[62]

There is limited literature evaluating rehabilitation strategies in children aged 5 to 12 years with SRC and those studies that did include children often only included 11- to 12-year-olds. Although more research has evaluated rehabilitation in adolescents, an understanding of differences in response to rehabilitation by age is limited since most studies crossed age groups. Most studies did not consider sex or gender. In addition, other research may be available across all types of mild traumatic brain injury arising from mechanisms of injury other than sport that identifies additional types of rehabilitation that could be of benefit and were not captured in this SRC-focused review.

What Tests and Measures Accurately Diagnose Persisting Postconcussive Symptoms in Children, Adolescents, and Adults Following Sport-related Concussion? A Systematic Review[63]

Up to 30% of children and adolescents experience persisting symptoms after concussion, defined as symptoms lasting for 4 weeks or longer after SRC. Of 26 studies in the systematic review, 8 involved children and adolescents only, whereas 11 bridged both pediatric and adult ages, most often including both adolescents and adults. The studies used a wide variety of measures and tests to investigate persisting symptoms, but none were designed to assess their ability to accurately diagnose persisting symptoms.

Neuroimaging studies reported subtle differences in white matter microstructure, brain activation during memory and balance tasks, and altered cerebral blood flow in children with persisting symptoms. Other measures in a variety of domains may support the diagnosis of persisting symptoms, especially the use of rating scales to demonstrate associated mood problems and lower quality of life.[63]

Overall, the evidence supporting the use of specific tests or measures for the differential diagnosis of persisting symptoms was deemed to be inconsistent, of limited quality (ie, mostly high risk of bias), and insufficient to determine how the differential diagnosis of persisting symptoms might differ among children, adolescents, and adults. Pediatric samples were reasonably balanced for sex, although gender, race, ethnicity, and other social determinants were usually not reported.

Persisting symptoms can be assessed using clinical expertise and standardized and validated symptom rating scales, but evidence-based recommendations regarding the use of other specific tests or measures in the clinical diagnosis of persisting symptoms are not possible currently. Future research is needed to determine which tests or measures differentiate children with and without persisting symptoms after SRC, preferably based on large prospective cohort studies with longitudinal follow-up, limited attrition, and common data elements.

Role of Biomarkers and Emerging Technologies in Defining and Assessing Neurobiological Recovery After Sport-related Concussion: A Systematic Review[64]

Limited pediatric-specific data were available. When compared with adults, the influences of puberty and brain development in children and adolescents may result in differences in the performance and utility of fluid biomarkers and emerging technologies for the purpose of diagnosing SRC and assessing neurobiological recovery.

Findings regarding objective diagnosis of SRC were similar to adults. Although group differences were demonstrated for several objective tools between young athletes with and without SRC,[64] the evidence is insufficient for recommending their use in clinical practice.

For monitoring recovery, studies on fluid biomarkers, advanced neuroimaging, and emerging technologies showed group differences at both symptom resolution and/or medical clearance, demonstrating that underlying physiologic effects of SRC may persist beyond symptom resolution and apparent clinical recovery.[64] However, their role in guiding clinical management at the individual level remains unclear.

Research characterizing the genetic aspects of concussion and recovery remains limited, with no studies including participants <18 years. Study results remain difficult to compare given substantial heterogeneity in study designs, methodologies, and data elements across domains.

Clinical Recovery From Concussion — Return to School and Sport: A Systematic Review and Meta-analysis[65]

The majority of children and adolescents who sustain SRC demonstrate complete resolution of concussion-related symptoms within 1 month.[66–69] The most consistent predictor of a longer recovery is a greater initial symptom burden (number and severity).[70–73] Other factors contributing to longer recovery times included continued play postinjury[74]; delayed presentation to a medical provider[75]; migraine history in females[76]; very high physical and

cognitive activity levels after injury[77,78]; and prolonged cognitive rest.[79–81]

The majority of children and adolescents return to school by 10 days without academic supports.[69,71,77,82] Longer return to school was associated with greater initial symptom severity[70–73] and low activity levels after injury.[77] Students experiencing difficulty with return to school may find the consensus strategy helpful,[65] in addition to receiving symptom-specific academic supports that encompass environmental, physical, curriculum, and testing factors.[83]

Most children and adolescents are able to return to sport following SRC within 1 month.[39,69,84] A higher symptom burden (number of and severity of symptoms) after concussion is associated with a longer return to sport, as well as the need for academic support.[69,83] Children and adolescents can safely follow the consensus return to sport strategy.[65] A qualified HCP should monitor the return to sport process, with medical clearance before return to any activities with risk of contact, collision, or fall.

When Should an Athlete Retire or Discontinue Participating in Contact or Collision Sports After Sport-related Concussion? A Systematic Review[85]

Although focused mostly on career-ending decisions related to SRC in adults, this systematic review also discussed retirement in children and adolescents participating in contact and collision sport. Children and adolescent athletes progressing to the next age group level in contact or collision sports, or to higher levels of competition, including participation in elite pathway programs and/or open-age competitions, may be at greater risk of concussion with increased training loads, exposure to players of a larger size, and higher velocity of impacts.[85]

The cognitively immature child or adolescent athlete may not yet be capable of adequately understanding the relative risks and benefits of participating in contact or collision sports. Parents and guardians may not be unanimous in their recommendations or influenced by multiple factors, including cultural and socioeconomic background, expectations for the child's future professional sports capabilities, vicarious benefits from the child's sporting achievements, and anxieties.

Decisions on when to cease participation in contact or collision sports are typically complex and multifaceted. The systematic review examined the contraindications to children and adolescent athletes entering or continuing with contact or collision sports[85] and, as none of the studies directly examined the issue of retirement and/or discontinuation from contact or collision sports, included studies that assessed factors associated with (1) *prolonged recovery* after SRC and/or (2) *increased risk of concussion*. Results across studies were heterogenous, and although not specific to children, the most consistent factors associated with *prolonged recovery* were longer time to presentation, total number and/or severity of symptoms at initial presentation, sleep disturbance, and symptom provocation with vestibular ocular motor testing and, for *increased risk of concussion*, history of previous concussion was the most consistent risk factor. Only 2 studies specifically examined children and only 14 examined adolescents. Major limitations of the studies included significant heterogeneity in study methodologies, definitions of "prolonged symptoms," age distribution, and selection bias with few high-quality cohort studies.

Limitations Common to All Pediatric Systematic Reviews

Limitations common to all the systematic reviews included a lack of studies in the 5 to 12-year age group, results not being stratified by age in studies with mixed age populations, and potential selection bias in studies with patients presenting to specialty clinic settings. Additionally, many studies of children with concussion were excluded from the systematic reviews because they did not meet the criteria of majority SRC. Many studies included predominantly male athletes and most studies were from North America, limiting generalizability. Definitions of clinical recovery varied across studies, making comparisons difficult. Increased media attention, awareness of concussion, and concurrent concussion education programs may have influenced concussion reporting rates for children, adolescents, and their parents, and may have affected study results when evaluating concussion prevention strategies longitudinally.

Para Sports

Globally, approximately 10% of children and adolescents are estimated to have a disability,[86] and participation in physical activity and sport within this population is on the rise.[87] Several of the more common types of developmental and childhood-onset disability (eg, spina bifida, cerebral palsy) impact functioning of the central nervous system and likely lead to differences in how an individual is impacted by concussion. Given the distinct paucity of research evaluating the concussion experience in the pediatric para athlete, the Concussion in Para Sport Group developed a Position Statement[88] to summarize the available literature, as well as expert opinion, related to the recognition, assessment, and management of concussion in the para athlete across the lifespan, inclusive of the pediatric athlete.

The Concussion in Para Sport Group Statement noted that children and adolescents with disabilities may uniquely benefit from preseason baseline testing given the variable nature of their disability and thus, atypical presenting concussion signs or symptoms. Additionally, individuals with a history of central nervous system injury may require more careful

evaluation and an extended period of initial rest after SRC. Testing for concussion may require modifications, such as use of arm ergometry, as opposed to a treadmill or stationary bike, with return to sport protocols tailored to include use of the individual's personal adaptive equipment. The most commonly used SRC assessment tools (eg, Child SCAT) are not validated in the pediatric para athlete population, who require an even more individualized approach. More research is needed to understand the impact of concussion on children and adolescents with disabilities.

Ethics

The application of the extant child and adolescent research in SRC to clinical management is fraught with conceptual, methodological, and translational challenges that have significant ethical import. The concept of childhood is itself vague and contested and has no unequivocal legal or moral border with adolescence, which in turn has no absolute border with adulthood.[89] The borders are influenced by a range of biopsychosocial factors that are not subject to universal agreement. In most western medical contexts, distinctions between children, adolescent, and adult populations are mostly artificial or arbitrary.[90] More specifically, sport medicine professionals often work with a binary pediatric or adult distinction. However, evidence for brain developmental changes in childhood indicate the important difference between children (≤12 years) and adolescents (≥12 years).[91] Although there is more SRC research for later adolescents that can help inform shared-decision making than for early adolescents, there is greater ambiguity around their competence to consent to research and treatment. Conversely, there is greater clarity on ethical processes with children. Due to children's lack of, or merely emerging, capacity, and their physiologic vulnerability because of developmental considerations, parents or other proxy decision-makers are therefore required on ethical and legal grounds. Paradoxically, less specific research is available on children with SRC to guide informed decisions.[92]

Additionally, there is a general paucity of childhood clinical trials,[93] to the point where they have been described as "therapeutic orphans" in research.[94] The lack of clinical trials is mirrored both in pediatric medical ethics research[95] and in childhood sport medicine research, as highlighted by the Concussion Consensus Statement.[6] Indeed, the empirical research and extant recommendations pertaining to pediatric SRC in the systematic reviews conducted as part of the Amsterdam consensus are dominated by findings from adolescents, in some cases predominantly focused on males (eg, prevention and acute detection of concussion).[10,26] However, a substantial body of relevant research on children, as well as adolescents, is available that did not meet the methodological requirement to focus primarily on SRC, as opposed to concussion from all causes, as reflected in the Amsterdam Consensus methodology.[8,96–105] Moreover, evidence-based clinical practice guidelines for pediatric concussion are available for consultation. These points serve as a general ethical precaution on the interpretation of the empirical research relevant to nonadult populations.

A general ethical foundation is that clinicians must work from what evidence arises in both sport and nonsport contexts to inform clinical care. Thus, in the absence of universally agreed principles to guide application, it is frequent to resort to a focus on good (ie, reasoned, transparent, and accountable) processes. In the medical ethics lexicon, this is referred to as Accountability for Reasonableness.[106] Thus, with concussed pediatric patients, good practice may simply demand a transparent process whereby clinicians, using the information cited in the Consensus Statement, including guidance offered for tools such as the Child SCAT6[107] and Child SCOAT6,[108] and using other relevant research with appropriate inferential justification, can satisfy Accountability for Reasonableness.[106] This would be underpinned by a broadly precautionary approach[109] given children's and adolescent's inherent status as vulnerable populations with protected characteristics (ie, according to the differing global contexts of care, this may include liberal notions such as the right of children and adolescents to an open future).[110,111] This approach must be balanced with careful considerations of the notable health-related benefits of sport and physical activity. In particular, sports that modify training and competition based on age and developmental considerations[112] may be considered as better satisfying the precautionary approach than those that do not. Notwithstanding these accommodations, the importance of brain health and development in children will require greater than normal precautions in relation to sports regulation and practices. In that regard, children and adolescents require consideration of a return-to-learn strategy, as detailed in the Consensus Statement[6] and the systematic review informing it,[65] which should take precedence over return to sport.

Tools

The Amsterdam process included updates of the Concussion Recognition Tool (CRT6)[113,114] and the Sport Concussion Assessment Tools (SCAT6 and Child SCAT6),[107,115–117] and development of a new set of tools, the Sport Concussion Office Assessment Tool (SCOAT6 and Child SCOAT6),[51,52,108,118] (Fig 1), the evidence for which was discussed earlier. The CRT6 is designed for the layperson to recognize concussion symptoms and signs in children, adolescents, and adults and provides advice regarding removal from play, immediate management, and "red flags." Widespread use of CRT6 by parents, coaches, teachers, and referees involved at any level with pediatric athletes across all sports is encouraged.

The SCAT6 (adolescents and adults)[115] and Child SCAT6[117] (ages 8–12 years) tools have been developed for HCPs for use in the acute period postconcussion.

FIGURE 1
The Tools developed for use by the lay person (CRT6), and for health care professionals in the acute period (SCAT6 and Child SCAT6) and subacute period (SCOAT6 and Child SCOAT6). Free downloads of all the tools available at https://bjsm.bmj.com/content/57/11.

The SCOAT6[52] (13+ years) and Child SCOAT6[51] (8–12 years) tools were developed for HCPs for subacute assessment in the office environment and promote a multimodal assessment of the concussed athlete, including detailed clinical history, symptom evaluation, cognitive tests, orthostatic vital signs, cervical spine assessment, neurologic examination, balance assessment, timed tandem gait, complex tandem gait, dual-task tandem gait, visio-vestibular examination, sleep assessment, mental health screen, and graded aerobic exercise tests. The Child SCOAT6 includes age-appropriate versions of each of the test components. Specific additions or changes relative to the Child SCOAT6 include:

- Additional symptoms for child and parent report that capture multiple subacute domains.
- An age-appropriate measure of cognitive reaction time (ie, the Symbol Digit Modalities Test).
- Validated pediatric measures of (i) orthostatic tachycardia, (ii) orthostatic intolerance, (iii) visio-vestibular function, and (iv) child mental health and sleep questionnaires.

The tools also include summary tables for return-to-school and return-to-sport strategies, with additional explanatory notes.

All the tools are available as free downloads.[51,52,114,115,117,119]

Future Directions

The systematic reviews highlight the paucity of age-specific research in children and the need for more research in the 5 to 12-year age group, particularly in children <8 years, in all areas of SRC. Future research evaluating concussion and head impact prevention strategies targeting sport-specific equipment, rule changes, training strategies, and management strategies is needed in children, and especially in girls. Other required research includes patient reported outcomes, specific objective outcomes, and operationally defined functional measures of recovery; comparisons of general versus targeted treatments; recording of specific treatment subcomponents, timing, frequency, duration, or intensity and combinations of treatments; and measurement of factors such as sex, gender, ethnicity, socioeconomic status, and para athlete-specific considerations. Additional research evaluating return to school and academic supports after SRC is needed, as well as studies of modifying factors for recovery and for returning to school and sport, and modifying factors in younger age groups, including mechanisms of injury. Accomplishing this goal may necessitate changing how SRC is defined and recognized in younger age groups. Studies that include mixed age populations should stratify results by age and sex whenever possible. Future research must be methodologically sound, including standardized definitions and evidence-based metrics. The Child SCAT6 and Child SCOAT6 require research and validation in different clinical and cultural settings.

CONCLUSIONS

The 6th International Consensus Conference on Concussion in Sport, the associated systematic reviews, and the Consensus Statement have provided significant updates on SRC in athletes of all ages. This paper highlights the key findings pertaining to children and adolescents, including prevention, sideline screening, office assessment, rest and exercise, rehabilitation, persisting symptoms, recovery, return to school and to sport, retirement, the para sport athlete, and ethical considerations, in addition to the important tools for the acute and subacute periods, with versions of each relevant to the adolescent and child. The clinical care of concussion in children and adolescents has improved significantly since the initial Concussion in Sport Group meeting at the turn of this century; however, the paucity of studies in younger children and lack of studies that are stratified by age to better understand the specific modifications to concussion care in pediatric athletes indicates the urgent requirement for more SRC research dedicated to children.

ABBREVIATIONS

CRT6: Concussion Recognition Tool 6
HCP: health care professional
NMT: neuromuscular training
PA: physical activity
SCAT6: Sport Concussion Assessment Tool 6
SCOAT6: Sport Concussion Office Assessment Tool 6
SRC: sport-related concussion

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Athlete Screening and Sudden Cardiac Death

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EDUCATION GAP

Primary care providers play an instrumental role in preventing sudden cardiac deaths in young athletes. Therefore, primary care providers should be familiar with warning signs and risk factors of cardiac conditions that can cause sudden death, preparticipation screening guidelines, and secondary prevention measures.

OBJECTIVES *After completing this article, readers should be able to:*

1. Identify the causes of sudden cardiac death in young athletes.
2. Describe screening guidelines and be aware of controversies in screening.
3. Recognize and promote secondary prevention measures.

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ABBREVIATIONS

AAOCA	anomalous aortic origin of a coronary artery
AAP	American Academy of Pediatrics
AC	arrhythmogenic cardiomyopathy
AED	automated external defibrillator
AHA	American Heart Association
ALCA	anomalous left coronary artery
ARCA	anomalous right coronary artery
CERP	Cardiac Emergency Response Plan
CMR	cardiac magnetic resonance
CPR	cardiopulmonary resuscitation
CPVT	catecholaminergic polymorphic ventricular tachycardia
ECG	electrocardiogram
HCM	hypertrophic cardiomyopathy
ICD	Implantable cardioverter-defibrillator
LQTS	long QT syndrome
LV	left ventricular
PPE	preparticipation physical evaluation
SCA	sudden cardiac arrest
SCD	sudden cardiac death

INTRODUCTION

Sudden cardiac death (SCD) is defined as unexpected and abrupt death caused by a cardiovascular condition, symptoms of which have begun within the past hour. Although SCD is rare, each death has a significant and long-lasting effect on the victim's family and community. Athletes with preexisting cardiac conditions are at increased risk for SCD during training and competition. (1) Conducting preparticipation physical evaluations (PPEs) of athletes is an important step in preventing SCD in susceptible individuals, partly because many of the predisposing conditions would otherwise not be noticeable. To ensure proper screening, primary care providers should be familiar with cardiac conditions associated with SCD.

Because no screening program can eliminate the risk of SCD, secondary preventive measures, such as increasing access to training in cardiopulmonary resuscitation (CPR) and automated external defibrillators (AEDs), as well as establishing emergency response plans at schools, are essential. This review provides an overview of the epidemiology and causes of SCD in young athletes as well as preparticipation screening and secondary prevention.

EPIDEMIOLOGY

SCD has an estimated incidence ranging from 1:917,000 to 1:3,000 athletes younger than 40 years in the United States per year. (2) Most cases occur at the

high school or college level, often during practice or competition. (3) The overall risk is higher in male compared with female athletes, with a 9:1 ratio, and more than 3 times higher in black versus white athletes. (3)

Although some data suggest that SCD is more common in nonathletes than in athletes, it is clear that intense physical activity increases the risk of SCD in individuals with predisposing cardiovascular disease. (4)(5)(6) Certain sports are more strongly associated with SCD, such as football, basketball, and baseball in male athletes. In contrast, female athletes are at greater risk while participating in basketball, cross-country/track, and soccer. (3) Although speculation attributes the increased risk of SCD in these sports to each sport's popularity, intensity of training, and genetic predispositions of athletes, further studies are needed to confirm this possibility.

CAUSES OF SUDDEN DEATH

Based on data from the US National Registry of Sudden Death in Athletes from 1980 to 2011, 40% of sudden deaths in young athletes occur in the absence of a preexisting cardiac condition. Approximately half of these deaths are due to blunt trauma (51%), with commotio cordis composing a much smaller percentage (7%). (3)

In large autopsy-based studies of athletes in the United States, hypertrophic cardiomyopathy (HCM) has consistently been the most common confirmed cardiovascular cause of sudden death, followed by coronary artery anomalies. Table 1 lists the most common cardiac conditions associated with SCD. In this section, we highlight the most prominent causes of SCD. An overview of these conditions and some others is provided in Table 2.

Table 1. Cardiovascular Causes of Sudden Cardiac Death (SCD) in Young Athletes

CONDITION	SCD, %[a]
Hypertrophic cardiomyopathy	36
Anomalous coronary artery	19
Myocarditis	7
Arrhythmogenic cardiomyopathy	5
Coronary artery disease	4
Mitral valve prolapse	4
Aortic rupture	3
Aortic stenosis	2
Dilated cardiomyopathy	2
Other[b]	18

[a]Percentages are based on 842 young athletes in the United States from 1980 to 2011 with confirmed cardiac causes of sudden death. (3)
[b]Other conditions include (in decreasing order of frequency) left ventricular hypertrophy of unresolved etiology, bridged left anterior descending artery, long QT syndrome, congenital heart defect, Wolff-Parkinson-White syndrome, myocardial infarction, sarcoidosis, stroke, conduction system abnormality, cardiac rupture, cardiac tumor, tetralogy of Fallot, electrolyte abnormality, ruptured cerebral arteriovenous aneurysm.

Hypertrophic Cardiomyopathy

HCM is a genetic disorder affecting sarcomere proteins, the basic contractile unit in cardiomyocytes. As previously noted, it is the most frequently identified cause of SCD in young athletes in the United States, with an estimated prevalence of 1:500. (7)(8) However, as of 2019, only 1:1,250 individuals are clinically diagnosed, suggesting that 60% of affected individuals remain undiagnosed. (9)

More than 900 mutations have been identified in patients with HCM, most commonly located in the genes encoding β-myosin heavy chain (*MYH7*), cardiac myosin binding protein C (*MYPBC3*), and cardiac troponin (*TNNT2*). The clinical course of the disease varies considerably, with some patients presenting in childhood and others remaining asymptomatic through adulthood. Chest pain and dyspnea on exertion are commonly reported symptoms. In addition, the physical examination may reveal a systolic murmur that becomes louder with reduced preload (eg, Valsalva maneuver or standing) and quieter with increased preload (eg, squatting) or afterload (eg, handgrip). A family history of sudden unexplained death is another important clue because HCM is usually inherited as an autosomal dominant trait. (10)

The 12-lead electrocardiogram (ECG) is abnormal in most patients with HCM, with findings suggestive of left ventricular (LV) hypertrophy and repolarization abnormalities (eg, T-wave inversion). (10) Ambulatory ECG monitoring also provides valuable information for risk stratification, as nonsustained ventricular tachycardia is a risk factor for SCD. Other risk factors for SCD in patients with HCM include history of syncope or cardiac arrest, family history of SCD, severe LV hypertrophy (in adults, LV maximal wall thickness ≥30 mm), and abnormal blood pressure response to exercise. (11) However, these risk factors are extrapolated from adult data, and pediatric-specific risk factors and calculators are currently under investigation.

Echocardiography is the principal diagnostic imaging modality for HCM (Fig 1). Although diastolic LV wall thickness of 15 mm or greater without a known underlying cause is diagnostic in adults, a body surface–adjusted *z* score of 2 or greater suggests HCM in children. (10)(12) Regardless of age, asymmetrical septal hypertrophy is a common and distinctive feature of HCM. Cardiac magnetic resonance (CMR), which offers enhanced spatial resolution and image quality, can be helpful in patients with diagnostic uncertainty or poor echocardiographic imaging windows. Late gadolinium enhancement,

a marker of replacement fibrosis found in approximately half of patients, is a risk marker for adverse outcomes in HCM. (13)

Screening is recommended for first-degree relatives of patients with HCM, who can manifest at any age and thus warrant surveillance imaging every 1 to 3 years. In addition, genetic testing with counseling is offered to individuals with HCM. If genetic testing reveals a pathogenic variant, cascade genetic testing should be offered to first-degree relatives, with clinical surveillance for those who carry the pathogenic variant. If a pathogenic mutation is not identified in the proband, cascade genetic testing is not recommended for first-degree relatives. (10)

Guidelines, which have historically recommended against participation in most competitive sports for patients with HCM, are complicated and have recently evolved. Although HCM is the most common cause of SCD in young athletes, growing evidence shows that recreational exercise of mild to moderate intensity is safe and beneficial in these patients. (10)(14) As of 2020, class I recommendations permit mild to moderate exercise in most patients with HCM. (10) Athletes with HCM are encouraged to undergo a comprehensive evaluation and shared discussion of the potential risks and benefits of sports participation with an expert. (10)(15) Although participation in low-intensity competitive sports is reasonable in most cases, if certain conditions are met, participation in moderate- to high-intensity sports can also be considered through shared decision-making. (10)

Coronary Artery Anomalies

Coronary artery anomalies are a diverse group of congenital conditions with a wide range of clinical manifestations. Anatomic variations in coronary anatomy are common and range from benign and not causing myocardial ischemia or SCD to life-threatening. (16)(17) In fact, anomalous aortic origin of a coronary artery (AAOCA) is the second most common cause of SCD in young athletes. (3)

Blood flow to the myocardium is derived from 2 main coronary arteries, each arising from a different location of the aorta. In most individuals, the right coronary artery arises from the right aortic sinus and supplies blood to the right side of the heart, whereas the left main coronary artery arises from the left aortic sinus and bifurcates into the left anterior descending and circumflex arteries, together supplying blood to the left side of the heart.

In patients with AAOCA, 1 of the coronary arteries arises from an abnormal location on the aorta, creating an abnormal coronary artery opening (ostium), take-off angle, and course. If the coronary artery becomes stretched, kinked, or compressed, blood flow to the myocardium can be disrupted and result in myocardial ischemia, ventricular arrhythmias, and SCD. Due to the silent nature of coronary anomalies, the true prevalence of AAOCA and absolute risk of SCD are unknown. (18) However, studies suggest that although anomalous left coronary artery (ALCA) is at least 6 times less common than anomalous right coronary artery (ARCA), it has a much higher risk of SCD. (19)

Although half of SCD cases occur in previously asymptomatic individuals, patients may present with chest pain, exertional syncope, or diminished exercise tolerance. (18) Results of physical examination, ECG, and stress testing are often normal. Coronary anomalies are typically diagnosed by transthoracic echocardiography and advanced imaging modalities, which should also be directed at identifying high-risk anatomic features that increase the risk of SCD. These high-risk anatomic features include slitlike ostium, acute take-off angle, intramural course (within the wall of the aorta), and interarterial course (between the aorta and the pulmonary trunk).

Restriction from competitive sports is recommended for patients who have ARCA with high-risk features, symptoms, arrhythmias, or evidence of ischemia. Athletes can generally resume competitive sports 3 months after reparative surgery if they are asymptomatic and if testing reveals no evidence of ischemia. For patients with ARCA without symptoms or concerns for arrhythmias or ischemia, participation in competitive sports is acceptable with adequate counseling and a discussion about the potential risk of cardiac events. In athletes with ALCA, restriction from competitive sports is generally recommended. As with ARCA, athletes with ALCA may be cleared for competitive sports 3 months after surgery if they are asymptomatic and have no evidence of ischemia. (20)

Myocarditis

Myocarditis, the third most common cause of SCD in young athletes, is an inflammatory disease of the myocardium due to infections, systemic diseases, drugs, and toxins. (3) Viral infections, particularly due to enteroviruses (eg, coxsackieviruses) and adenoviruses, are the most common causes of myocarditis in the Unites States. Clinical manifestations vary widely, ranging from asymptomatic cases to heart failure and potentially fatal arrhythmias. Common symptoms associated with myocarditis are fatigue, shortness of breath, nausea (and/or abdominal pain), and chest pain. (21)(22) The diagnosis is usually suspected due to the presence of cardiac symptoms, elevated plasma troponin level, nonspecific ECG abnormalities (ST/T-wave changes), and echocardiographic findings, such as cardiac chamber enlargement and/or

Table 2. Cardiovascular Conditions Associated with Sudden Cardiac Death

CONDITION	CAUSE	PHYSICAL EXAMINATION	ECG/HOLTER	ECHOCARDIOGRAM	SPORTS GUIDELINES (USA)[a]	MANAGEMENT	NOTES
HCM	Sarcomere mutations (mostly AD)	Systolic murmur	LV hypertrophy and/or RV hypertrophy, large R or S waves; left axis deviation; ventricular arrhythmias	LV wall thickness ≥15 mm or 2 SD for weight; small LV cavity size; abnormal mitral valve +/− mitral regurgitation; LV outflow tract obstruction	Restrict from high-intensity sports, encourage mild-intensity activity; shared decision-making[b]	Consider ICD if high risk β-Blockers may improve symptoms but do not reduce mortality	High risk: previous cardiac arrest or sustained VT; family history of SCD; syncope; nonsustained VT; abnormal blood pressure response to exercise; severe LV hypertrophy
AAOCA	Abnormal coronary embryogenesis	Normal	Usually normal; ischemic changes; arrhythmias	Abnormal coronary artery	*Anomalous left coronary artery* Restrict from competitive sports *Anomalous right coronary artery* Restrict from competitive sports if symptoms, arrhythmias, or evidence of ischemia	*Anomalous left coronary artery* Surgical repair *Anomalous right coronary artery* Surgical repair if high-risk features, symptoms, arrhythmias, or evidence of ischemia	Coronary angiography is recommended Stress tests may yield false-negatives Consider sports participation 3 mo after reparative surgery if asymptomatic and no evidence of ischemia
Myocarditis	Acquired; usually viral infection	Evidence of cardiogenic shock, sinus tachycardia or tachyarrhythmias, gallop or hepatomegaly	Nonspecific ST-segment and T-wave abnormalities; ectopy/arrhythmias	Wall motion abnormalities; ventricular dysfunction; mitral regurgitation; pericardial effusion	Restrict from sports for at least 3–6 mo	Supportive	Clearance depends on normalization of serum markers of cardiac injury, normalization of systolic function, and resolution of any arrhythmias on exercise ECG
AC	Mutations in desmosomal proteins, eg, *PKP2* (mostly AD) Fatty fibrous tissue replaces heart muscle	Normal	Epsilon wave; localized QRS widening; prolonged S-wave upstroke; T-wave inversion in leads V_1 through V_3; LBBB, PVCs, or VT	Enlarged RV, LV, or both; ventricular dysfunction	Restrict to low-intensity sports[c]	ICD if high risk	Cardiac MRI is recommended High risk: aborted SCD, sustained VT, severe ventricular dysfunction
Channelopathies							
LQTS	*Congenital:* mutations in potassium or sodium channels (mostly AD) *Acquired:* medications, electrolyte abnormalities, etc	Normal	QTc prolongation Abnormal T-wave morphology	Normal	Restrict to low-intensity sports if symptomatic or prolonged QTc[c] LQTS type 1 genotype should avoid competitive swimming	β-blockers ICD if high risk	Often incidental detection on ECG; consider in patients with syncope or atypical seizures Risk of SCD is related to QTc duration, genotype, history of symptoms, sex of individual

Continued

Table 2. Cardiovascular Conditions Associated with Sudden Cardiac Death (Continued)

CONDITION	CAUSE	PHYSICAL EXAMINATION	ECG/HOLTER	ECHOCARDIOGRAM	SPORTS GUIDELINES (USA)[a]	MANAGEMENT	NOTES
Brugada syndrome	Mutations in sodium channels, eg, *SCN5A* (mostly AD)	Normal	Coved ST-segment elevation in leads V_1 and V_2; abnormal T-wave morphology	Normal	Restrict to low-intensity sports[bc] If asymptomatic, participation can be considered with precautionary measures	Quinidine Ablation ICD if high risk	Highest risk of SCD with type 1 Brugada pattern on ECG Fevers increase risk; treat with antipyretics
CPVT	Calcium dysregulation Mutation in ryanodine receptor 2 (*RyR2*) (mostly AD)	Normal	Normal	Normal	Restrict to low-intensity sports[c]	Antiarrhythmic medications (eg, nadolol, flecainide) Left cardiac sympathetic denervation ICD if high risk	Exercise stress test may reveal ectopy, polymorphic VT
WPW syndrome	Accessory pathway; unknown cause	Normal	Slurred QRS upstroke ("delta wave"), short PR interval	Normal	Asymptomatic: No restriction Symptomatic: EPS recommended	β-Blockers or ablation useful to prevent SVT	Return to competitive sports 4 wk after radiofrequency ablation

AAOCA=anomalous aortic origin of a coronary artery, AC=arrhythmogenic cardiomyopathy, AD=autosomal dominant, CPVT=catecholaminergic polymorphic ventricular tachycardia, ECG=electrocardiogram, EPS=electrophysiology study, HCM=hypertrophic cardiomyopathy, ICD=implantable cardioverter-defibrillator; LBBB=left bundle branch block, LQTS=long QT syndrome, LV=left ventricle, MRI=magnetic resonance imaging, PVC=premature ventricular contraction, RV, right ventricle, SCD=sudden cardiac death, SVT=supraventricular tachycardia, VT=ventricular tachycardia, WPW=Wolff-Parkinson-White.

[a]Sports restriction guidelines are from Maron BJ, Zipes DP, Kovacs RJ; on behalf of the American Heart Association Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, Council on Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and Translational Biology, and the American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: preamble, principles, and general considerations: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132:e256–e261.

[b]Individuals who are genotype positive but phenotype negative can continue to participate in all competitive sports.

[c]Low-intensity class 1A sports have low static (<20% of maximum voluntary contraction) and low dynamic (<40% of maximum oxygen uptake) components. These sports include billiards, bowling, cricket, curling, golf, and riflery.

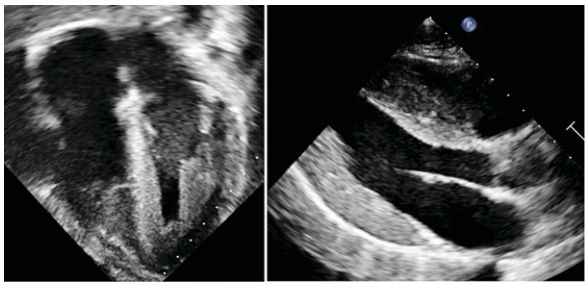

Figure 1. Echocardiograms showing left ventricular hypertrophy in a teenager with hypertrophic cardiomyopathy.

impaired LV systolic or diastolic function. Although endomyocardial biopsy remains the gold standard, CMR has emerged as a more sensitive and less invasive test to confirm the diagnosis.

To promote the resolution of inflammation, athletes diagnosed as having myocarditis should be restricted from exercise for 3 to 6 months, depending on the extent of cardiac injury and inflammation on CMR. (15) Because SCD in myocarditis is most likely due to development of ventricular tachyarrhythmias resulting from myocardial scarring, ambulatory ECG monitoring and/or exercise stress testing are used in addition to CMR to evaluate for arrhythmias before clearing patients for return to competitive sports. (23)

Arrhythmogenic Cardiomyopathy

Arrhythmogenic cardiomyopathy (AC), previously known as arrhythmogenic right ventricular cardiomyopathy, is an inherited cardiomyopathy characterized by progressive replacement of myocardium with fibrofatty tissue and a predisposition for ventricular arrhythmias and SCD. Although structural abnormalities of the right ventricle predominate, LV involvement is also possible. (24)

AC is usually inherited in an autosomal dominant manner with incomplete penetrance and variable expressivity. Most cases involve mutations in genes encoding proteins in desmosomes, the membrane structures that regulate intercellular adhesion and maintain structural integrity of tissues during mechanical stress. Mutations in the plakophilin-2 (*PKP2*) gene are the most frequent cause. (25) AC predominantly affects men, whereas women with an associated gene mutation have a lower chance of expressing the disease and are more likely to be asymptomatic carriers. (25)

The prevalence of AC is challenging to estimate because it is often discovered postmortem. Its association with the Mediterranean region explains why it is the most common cause of SCD in young athletes in Italy while accounting for less than 5% in the United States. (24)(26)(27)

Clinical signs or symptoms of AC such as dizziness, syncope, or palpitations are rarely recognized before puberty, with onset typically ranging from the late 20s to early 30s. (27) As a result, the condition is not likely to be detected based on history and physical examination findings unless there is a family history notable for SCD or relatives diagnosed as having AC. Although ECG has low sensitivity for this condition, it may show important abnormalities, including a widened QRS, epsilon wave (Fig 2A), and inverted T waves in the right precordial leads (V_1 through V_3), the latter of which is abnormal after 14 years of age. (25)(28) Although echocardiography is generally normal, especially in the early stages of the disease course, a normal study does not exclude the diagnosis. If clinical suspicion is high, CMR should be performed to evaluate for myocardial changes consistent with the disease.

There is no cure for AC. When the diagnosis is made, the individual should be restricted from competitive sports with the possible exception of low-intensity class 1A sports, which include billiards, bowling, cricket, curling, golf, and riflery. (15) Patients who meet specific high-risk criteria usually undergo insertion of an implantable cardioverter-defibrillator (ICD). (29)

Ion Channelopathies

Ion channelopathies are a group of hereditary defects in the membrane channel proteins that can cause lethal arrhythmias and SCD in individuals with structurally normal hearts. These disorders are suspected to account for a significant proportion of cases in which the autopsy reveals a structurally normal heart with no identifiable cause of SCD. (30) Long QT syndrome (LQTS), Brugada syndrome, and

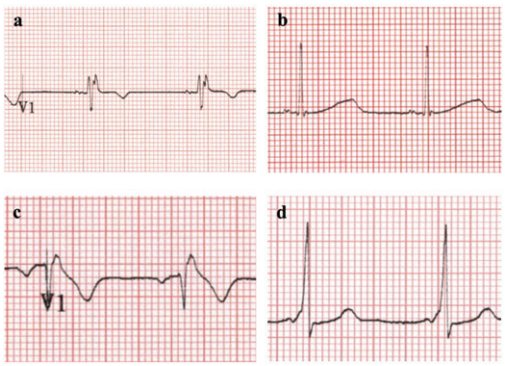

Figure 2. Electrocardiographic findings associated with cardiac diseases. A. Epsilon wave in arrhythmogenic right ventricular cardiomyopathy. B. Prolonged QT interval in long QT syndrome. C. Coved-type ST-segment elevation in Brugada syndrome. D. Delta wave in Wolff-Parkinson-White syndrome.

catecholaminergic polymorphic ventricular tachycardia (CPVT) are the most common channelopathies associated with SCD. Patients with these disorders may report palpitations and syncope (particularly exertional or with auditory stimulation) and a family history notable for early and/or unexplained death. Although findings from physical examination and echocardiography are generally normal, each channelopathy has a unique ECG fingerprint.

LQTS, which can be inherited or acquired (eg, QT-prolonging drugs), is characterized by prolonged ventricular repolarization leading to ventricular arrhythmias, classically *torsade de Pointes* ("twisting of peaks" in French). The risk of arrhythmias and SCD is directly related to the duration of the QTc interval; each 10-millisecond increase in QTc has been shown to increase the risk of malignant arrhythmias. (31) QTc intervals are considered prolonged when they are greater than 460 milliseconds in males or greater than 470 milliseconds in females and highly abnormal regardless of sex when greater than or equal to 500 milliseconds (Fig 2B). Although QTc prolongation is a hallmark of LQTS, 40% of patients with genetically confirmed LQTS have a normal QTc duration on baseline ECG, with only subtle T-wave abnormalities. (32)(33) In such cases of concealed LQTS, exercise stress testing can unmask ECG abnormalities. On the opposite end of the spectrum, LQTS is also frequently overdiagnosed, especially in patients with prolonged QTc secondary to vasovagal syncope. (34)

At least 17 different genes are associated with LQTS, of which 90% are accounted for by 1 of 3 major genes: *KCNQ1* (LQTS1), *KCNH2* (LQTS2), and *SCN5A* (LQTS3). (35) SCD associated with mutations in each have signature triggers: exercise, especially swimming, for LQTS1; arousal, especially a sudden loud noise (along with one-third of SCDs during exercise) for LQTS2; and during sleep (and <5% of SCDs during exercise) with LQTS3. (35)

Current guidelines recommend restriction of individuals with LQTS to class 1A sports if they have symptoms, ICDs, or significant QTc prolongation (males: >470 milliseconds; females: >480 milliseconds), although participation may be considered after initiation of treatment and appropriate precautionary measures. (36) Genetically positive individuals who have normal QT intervals can compete in sports without restriction, except for individuals with symptomatic LQTS1, who should be restricted from competitive swimming. β-Blockers, particularly nadolol and propranolol, are the first-line therapy for patients with LQTS and are most effective in LQTS1. Other therapeutic options reserved for high-risk patients include ICD placement and left cardiac sympathetic denervation to reduce adrenergic stimulation of the heart. (37)

Brugada syndrome commonly involves autosomal dominant mutations in the cardiac sodium channel *SCN5A* gene, although pathogenic variants in other genes have been identified. (38) Patients with this condition may report a history of palpitations or syncope triggered by fever and a family history of Brugada syndrome or sudden death. The cardiac examination is usually normal; however, the diagnosis is based on ECG abnormalities that may occur spontaneously or be unmasked by a sodium channel blocker (ie, provocative drug testing). ECG may demonstrate pathognomonic abnormalities, including a "coved-type" ST-segment elevation (Fig 2C) or "saddleback" ST-segment elevation in the right precordial leads, V_1 through V_3, whereas Holter monitoring is useful to evaluate for asymptomatic arrhythmias. Lethal arrhythmias may be triggered by increased vagal tone (eg, during exercise recovery or sleep) as opposed to during exercise. (39) Despite the absence of a clear association between exercise and SCD in Brugada syndrome, previous US guidelines recommended restriction from competitive sports with the potential exception of class 1A sports. (39)(40) Current guidelines specify that participation in competitive sports may be considered once appropriate precautionary measures and treatments are in place provided that the athlete has been asymptomatic for at least 3 months. (36) Treatment has historically been limited to drugs and ICDs for high-risk patients, although catheter ablation—a minimally invasive procedure used to destroy abnormal tissue—has also been suggested as a therapeutic option. (38)

CPVT results from genetic mutations—most commonly autosomal dominant in the *RyR2* gene encoding a ryanodine receptor in the sarcoplasmic reticulum—that cause abnormal calcium release in cardiomyocytes. The disease is characterized by adrenergic-induced ventricular tachyarrhythmias, including bidirectional ventricular tachycardia, a rare tachyarrhythmia in which dual QRS morphologies alternate on a beat-to-beat basis. In this condition, syncope or sudden death is usually triggered by acute emotional stress or exercise. History and physical examination findings are often normal, with a positive family history of exercise/emotion syncope in approximately one-third of cases. (41) Although resting ECG is usually normal, ventricular ectopy and arrhythmias can be provoked by exercise (ie, exercise stress testing) or epinephrine. With mortality in this condition high if untreated (30%–50% by age 40 years), those diagnosed as having CPVT are generally restricted from competitive sports and treated with antiarrhythmic medications,

including β-blockers (ie, nadolol) and sodium channel blockers (ie, flecainide), left cardiac sympathetic denervation, and occasionally ICD implantation. (36)(41)(42)

Commotio Cordis

Commotio cordis (Latin etymology, "agitation of the heart") refers to a disruption of the normal heart rhythm due to a direct blow to the chest and is particularly distressing because commotio cordis usually occurs in an otherwise healthy athlete with no identifiable heart problem. Each year in the United States, there are approximately 10 to 20 cases of this condition, which is primarily an electrical phenomenon that does not result from structural damage to the myocardium. (43) For ventricular fibrillation to be induced, the blunt impact must involve a minimum pressure of 250 mm Hg and must occur during a narrow window of vulnerability during the cardiac cycle (ie, during the T-wave upslope). (43)

Approximately 95% of cases occur in males, with a mean age of 15 years. (43) Baseball has the highest incidence of commotio cordis. Although commercial protective equipment may be helpful in preventing commotio cordis, it can still occur when the victim is wearing a chest guard. (44)(45)

During the past 2 decades, survival rates of patients with commotio cordis have increased from 15% to 60%, largely due to improved recognition of sudden cardiac arrest (SCA), bystander-initiated CPR, and the widespread availability of AEDs at sporting events. (46) In fact, the survival rate drops to 3% when resuscitation is delayed beyond 3 minutes. (44) If no underlying cardiac abnormality is identified, survivors of commotio cordis are allowed to resume training and competition on full recovery. (46)

Other Causes

Several other conditions are associated with SCD, including severe obstructive lesions such as aortic stenosis or pulmonary stenosis, aortopathies such as Marfan syndrome (ie, due to aortic rupture or dissection), primary pulmonary hypertension, sarcoidosis, and sickle cell trait. Patients with complex congenital heart defects, including those status post repair or palliative procedures, are at risk for arrhythmias and SCD due to surgical scarring, hemodynamic abnormalities, residual lesions, or ventricular dysfunction. (47) Wolff-Parkinson-White syndrome (Fig 2D) is considered a rare cause of SCD, which likely occurs due to the rapid conduction of atrial fibrillation to the ventricles via the accessory pathway, resulting in ventricular fibrillation. (48) Although mitral valve prolapse is typically considered a benign condition, it may pose an increased risk of SCD. (49) Finally, atherosclerotic cardiovascular disease is a major cause of SCD in older athletes but rarely causes SCD in young athletes, although the likelihood is higher if there are risk factors such as a history of Kawasaki disease. (50) Although performance-enhancing drugs can increase cardiac risk, evidence has been inconclusive about their involvement in SCD. (51)

NORMAL FINDINGS IN ATHLETES

The physiological and benign profile of an athlete's heart (so-called athlete's heart) can be difficult to differentiate from cardiovascular abnormalities. Because ECG changes are observed in approximately 40% of trained athletes, international consensus standards for normal, abnormal, and borderline ECG findings in this population have been established. (52)(53) Examples of findings that are considered normal for trained athletes include high QRS amplitude (meeting voltage criteria for LV hypertrophy), early repolarization, sinus bradycardia, sinus arrhythmia, and first-degree atrioventricular block. Such findings are attributed to intense athletic conditioning resulting in structural cardiac remodeling and increased vagal tone and do not warrant further evaluation.

Morphologic remodeling associated with intense athletic training, such as increased LV chamber dimensions and wall thickness, can mimic features of genetic and acquired heart disease such as cardiomyopathies. Certain echocardiographic features are helpful in distinguishing between benign adaptive remodeling and heart disease, and a complete description is beyond the scope of this review. However, a thorough evaluation of LV wall thickness and morphology, diastolic LV cavity size, atrial size, systolic function, and diastolic function is required. For cases in which echocardiographic findings are equivocal, repeated evaluation after a period of deconditioning (ie, avoiding exercise) can be helpful because ECG and echocardiographic changes associated with an athlete's heart should normalize during this time. (54) The distinction between physiological and pathological changes in athletes is important because an incorrect diagnosis can have significant consequences, such as exclusion from competitive sports or inappropriate reassurance and a missed opportunity for therapeutic intervention.

SCREENING GUIDELINES

The primary goal of screening is to identify conditions that predispose individuals to SCA or SCD. Personal history, family history, and physical examination findings are

the core components of screening. The American Academy of Pediatrics (AAP) recommends that all children undergo screening for the risk of SCD regardless of athletic status. (55) Screening should occur during the PPE, a minimum of every 3 years, or on entry into middle or junior high school and into high school. Four main screening questions are recommended:

1. Have you ever fainted, passed out, or had an unexplained seizure suddenly and without warning, especially during exercise or in response to sudden loud noises, such as doorbells, alarm clocks, and ringing telephones?
2. Have you ever had exercise-related chest pain or shortness of breath?
3. Has anyone in your immediate family (parents, grandparents, siblings) or other more distant relatives (aunts, uncles, cousins) died of heart problems or had an unexpected sudden death before age 50 years? This would include unexpected drownings, unexplained car accidents in which the relative was driving, or sudden infant death syndrome.
4. Are you related to anyone with HCM or hypertrophic obstructive cardiomyopathy, Marfan syndrome, AC, LQTS, short QT syndrome, Brugada syndrome or CPVT, or a condition requiring implantation of a pacemaker or ICD at younger than 50 years?

The AAP's recommended screening questions are based on expert consensus and have not been scientifically validated in a prospective study. These questions were designed to be simple and easy to incorporate into a family questionnaire. (55) The first question focuses on personal history of sudden loss of consciousness, particularly events triggered by exertion or sudden loud noises, which tend to occur in patients with channelopathies such as LQTS. The second question focuses on symptoms of chest pain or shortness of breath on exertion, which can signal myocardial ischemia, congestive heart failure, arrhythmias, and valvar disease. The other 2 questions inquire about family members with heart conditions, potentially pointing to a familial inheritance pattern. Steinberg et al (56) previously showed that cardiac abnormalities can be identified in nearly one-third of first-degree relatives of SCA survivors or SCD victims.

Meanwhile, the American Heart Association (AHA) has published guidelines for preparticipation screening of competitive athletes, which consists of a 14-element (previously 12-point) history and physical examination (Table 3). Use of this 14-element checklist for preparticipation screening is a class I recommendation based on a scientific statement jointly published by the AHA and American College of Cardiology. (57)(58) As with the AAP's screening questions, the AHA's 14-element screening tool was developed based on expert opinion and over time has become the most commonly accepted screening method for young athletes. (55)(57)(58) In addition, it has also been incorporated into other screening guidelines, such as a revised version with changes in language and wording that has been incorporated into the *PPE: Preparticipation*

Table 3. The 14-Element American Heart Association Recommendations for Preparticipation Screening of Competitive Athletes (57)

Personal history:
1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope[a]
3. Excessive exertional and unexplained dyspnea/fatigue or palpitations, associated with exercise
4. Previous recognition of a heart murmur
5. Elevated systemic blood pressure
6. Previous restriction from participation in sports
7. Previous testing for the heart, ordered by a physician
Family history:
8. Premature death (sudden and unexpected, or otherwise) before age 50 y attributable to heart disease in ≥1 relative
9. Disability from heart disease in a close relative aged <50 y
10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of certain cardiac conditions in family members
Physical examination:
11. Heart murmur[b]
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting position)[c]

[a]Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring during or after physical exertion.
[b]Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed with the patient in both the supine and standing positions (or with the Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.
[c]Preferably taken in both arms.

Physical Evaluation, 5th Edition, a monograph published by the AAP in collaboration with 5 other medical societies. (59)

However, despite being endorsed by medical societies, the 14-element screening tool remains heavily underused. A recent study in *Pediatrics* found that a minority (27%) of the 48 states providing PPE forms incorporate all 14 elements in their screening forms. (60) Moreover, a quality review by Miliaresis et al (61) found that only one-third of pediatricians are familiar with the AHA's recommended screening form and that, on average, only 3.5 of 14 elements are documented in visit notes. Barriers to PPE use include lack of awareness of the 14-element screening form, use of a different form, and time constraints. Of note, the authors did increase use of the PPE through standard quality improvement methods. (61)

Although the need for preparticipation screening of young athletes is widely supported, the optimal approach has long been debated. Studies have shown that the 14-element screening has sensitivity and specificity of 20% and 68%, respectively, for cardiac risk factors among high school athletes. (62) The incorporation of ECG screening would certainly increase screening sensitivity, as evidenced by a meta-analysis published in 2015, based on screening of 47,137 athletes from several regions, that revealed a 5-fold and 10-fold increase in sensitivity of ECG in detecting cardiac conditions relative to history and physical examination, respectively. (63) However, incorporating ECG screening would also involve a substantial cost that medical societies such as the AHA view as prohibitive. With nearly 10 million student athletes in the United States annually, the widespread implementation of ECG screening would cost billions of dollars. One must also consider the additional monetary, legal, and emotional cost of dealing with false-positive and false-negative test results. Therefore, ECG is usually reserved for patients at increased risk for SCD based on routine screening with the PPE.

SECONDARY PREVENTION

Preparticipation screening is useful for identifying athletes with high-risk cardiac conditions, but no screening strategy can single-handedly prevent SCD. In addition, despite improvements in survival rates after SCA, disparities in outcomes based on race and socioeconomic status continue to exist, with studies showing worse cardiac emergency preparedness and lower survival rates in low-income neighborhoods versus high-income neighborhoods. (64) To address these disparities, it is essential to target these demographics in the implementation of secondary prevention measures.

CPR and AEDs

SCD can be prevented through prompt recognition, administration of high-quality CPR, and early defibrillation. This "Chain of Survival" is highly dependent on public engagement. Administration of bystander CPR is associated with increased survival and improved neurologic outcome. (65) Although previous CPR training increases bystanders' willingness to perform CPR, only a small percentage (<3%) of the US population receives training each year. (66)(67)(68) In recent years, self-directed online courses have emerged as an acceptable alternative to in-person courses. Furthermore, statewide laws requiring CPR training in high schools have also translated to more laypersons learning this lifesaving skill, although the quality of such training is inconsistent and has striking geographic disparities. (69) Currently, the AHA recommends training students as early as middle school on how to perform high-quality CPR.

Early defibrillation is another important intervention affecting outcomes after SCA, and arguably the most important determinant of survival. Despite the fact that most cases of SCA involve a shockable rhythm (ie, ventricular fibrillation), the probability of surviving SCA caused by ventricular fibrillation diminishes rapidly over time, declining approximately 10% each minute defibrillation is delayed. (70)(71) From 2000 to 2006, exercise-related SCA in the young had an overall survival rate of 11%. (72) A more recent study analyzing events from 2014 to 2018 reported a higher overall survival rate of 68% among student athletes, with a survival rate of 85% if an on-site AED is used. (73)

Emergency Preparedness

Schools have a responsibility to prepare for cardiac emergencies that goes beyond the mere presence of an AED or bystander with CPR training. The AHA recommends that schools and/or school districts implement Cardiac Emergency Response Plans (CERPs), which are associated with a lower incidence of SCD. (74)(75) Core components of a CERP include establishing a Cardiac Emergency Response Team (a team of athletic trainers, teachers, and other staff members with CRP/AED training), creating an emergency activation system, ensuring AED equipment is easily accessible and properly maintained, offering frequent CPR/AED training, performing practice drills, and reviewing the plan on an ongoing and annual basis. (76) Documents needed to implement a CERP are available on the AHA's website. (77)

The AHA also recommends that states enact legislation that requires and funds the creation and maintenance of CERPs in schools. (76) State legislation requiring schools to have an AED has been shown to significantly increase

AED availability across high schools, with public schools being most affected by such legislation. (78) As of 2022, according to data collected by the Sudden Cardiac Arrest Foundation, only 20 states require AEDs. (79)

FUTURE DIRECTIONS

The absolute risk of SCD is low, even among athletes with high-risk conditions, and the known detriments of lack of physical activity have led to a recent focus on shared decision-making. Moreover, machine learning algorithms can potentially help clinicians diagnose the conditions that cause SCD and can already detect HCM and LQTS from standard ECGs with excellent sensitivities and specificities, and it is possible that artificial intelligence can similarly be trained to facilitate more accurate risk stratification, resulting in a lower burden of unnecessary sports disqualification. (80)(81) Finally, gene therapy is an emerging area of interest in SCD prevention, as investigators recently published the first hybrid gene therapy for LQTS1, which is now advancing to animal model studies. (82)

CONCLUSION

SCD is a rare but devastating event that is often preventable. When SCD occurs in a young athlete, it is frequently due to an underlying cardiac condition. Due to lack of demonstrated efficacy as well as prohibitive costs and feasibility of large-scale ECG and echocardiography screening, routine PPE is limited to history and physical examination. Primary care providers and pediatric cardiologists can prevent SCD by identifying red flags associated with the most common causes of SCD. Because the PPE is imperfect at identifying athletes with high-risk conditions, secondary measures are also essential to preventing SCD.

Summary

- Preparticipation screening for young athletes is important because it can potentially reduce the risk of sudden cardiac death (SCD). (Based on research evidence and consensus) (55)(58)(59)(60)(83)
- Several cardiac conditions are associated with SCD. Hypertrophic cardiomyopathy, coronary artery anomalies, and myocarditis are the 3 most common identifiable causes. Ion channelopathies may account for a significant number of autopsy-negative SCDs. (Based on research evidence and consensus) (3)(30)
- A focused history and physical examination is the recommended preparticipation screening method, although the American Heart Association's 14-element screening tool has low sensitivity. (Based on research evidence and consensus) (58)(62)
- Universal electrocardiographic screening is not recommended in the United States. This is partly due to prohibitive cost and the potential for false-positives resulting in additional unnecessary testing. (Based on research evidence and consensus) (57)(58)(84)
- Providers should follow US guidelines for determining the appropriate sports restriction for individuals with high-risk cardiac conditions. However, it is also important to recognize that these conditions have a low risk of SCD and that shared decision-making has emerged as an important framework for the contemporary sports eligibility discussion. (Based on research evidence and consensus) (10)(15)(36)
- High-quality cardiopulmonary resuscitation and prompt defibrillation are integral to preventing SCD once sudden cardiac arrest (SCA) has occurred. The probability of surviving SCA caused by ventricular fibrillation is reduced by 10% each minute defibrillation is delayed. (Based on research evidence and consensus) (71)(75)
- Schools and/or school districts should implement Cardiac Emergency Response Plans, which can help schools prepare for SCAs and prevent SCDs. (Based on some research evidence and consensus) (74)(75)(77)
- Machine learning could play an important role in SCD prevention in the future. In addition, new gene therapies may be available for individuals diagnosed as having predisposing conditions. (Based on some research evidence) (80)(81)(82)

ACKNOWLEDGMENTS

We dedicate this article to Frank J. Reali III, who lost his life to sudden cardiac death, and we thank his family, who, through the Protecting One Young Heart at a Time Foundation, has generously supported local screenings for high school athletes on Staten Island. We also thank Dr Philip Roth for his insightful suggestions and comments.

References and teaching slides for this article can be found at https://doi.org/10.1542/pir.2023-005975.

JACC Journals

From: Task Force 8: Classification of sports

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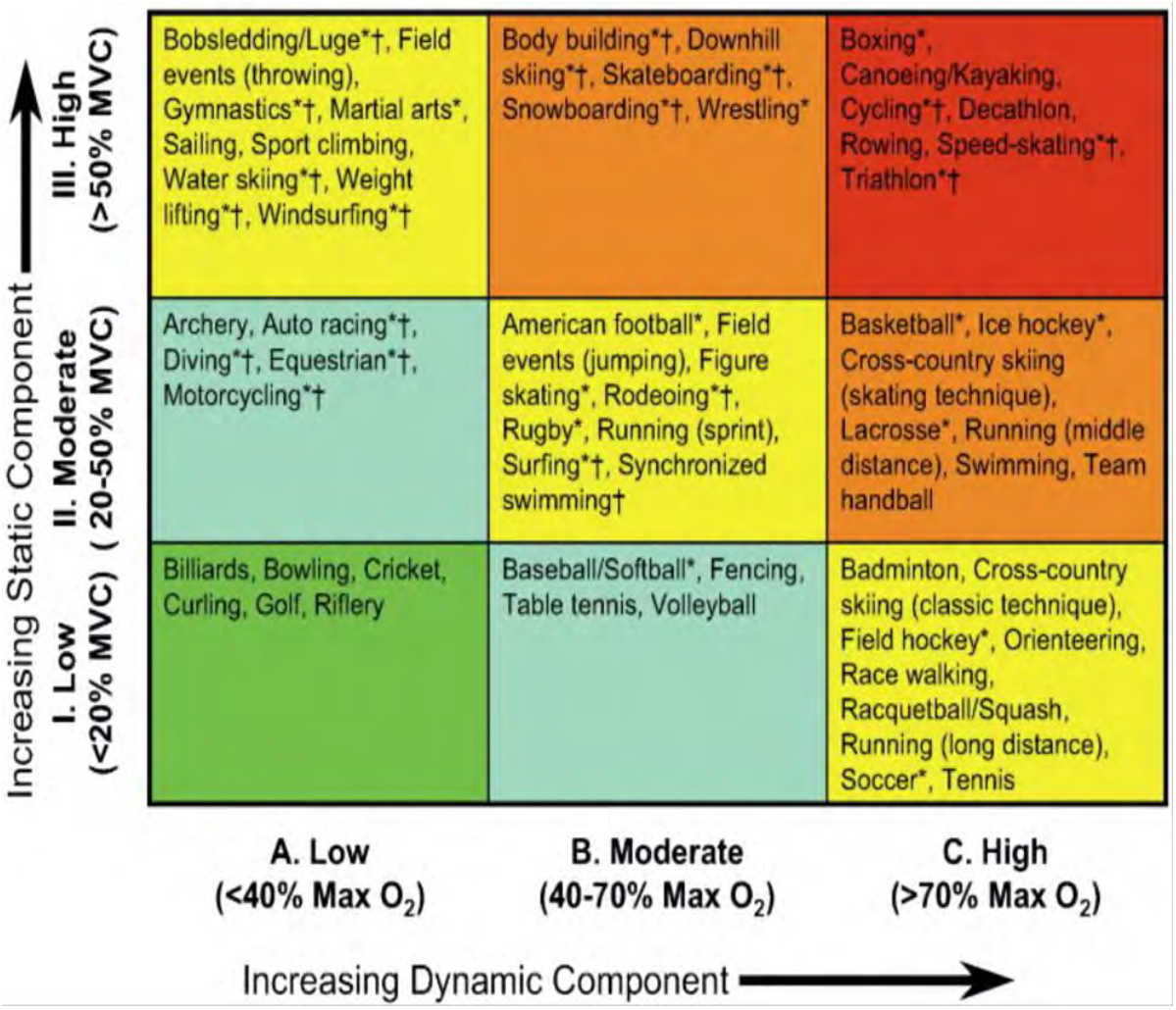

Figure Legend:

Classification of sports. This classification is based on peak static and dynamic components achieved during competition. It should be noted, however, that higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percent of maximal oxygen uptake (MaxO_2) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percent of maximal voluntary contraction (MVC) reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in green and the highest in red. Blue, yellow, and orange depict low moderate, moderate, and high moderate total cardiovascular demands. *Danger of bodily collision. †Increased risk if syncope occurs.

CRT6™

Concussion Recognition Tool

To Help Identify Concussion in Children, Adolescents and Adults

What is the Concussion Recognition Tool?

A concussion is a brain injury. The Concussion Recognition Tool 6 (CRT6) is to be used by non-medically trained individuals for the identification and immediate management of suspected concussion. It is not designed to diagnose concussion.

Recognise and Remove

Red Flags: CALL AN AMBULANCE

If **ANY** of the following signs are observed or complaints are reported after an impact to the head or body the athlete should be immediately removed from play/game/activity and transported for urgent medical care by a healthcare professional (HCP):

- **Neck pain or tenderness**
- **Seizure, 'fits', or convulsion**
- **Loss of vision or double vision**
- **Loss of consciousness**
- **Increased confusion or deteriorating conscious state (becoming less responsive, drowsy)**
- **Weakness or numbness/tingling in more than one arm or leg**
- **Repeated Vomiting**
- **Severe or increasing headache**
- **Increasingly restless, agitated or combative**
- **Visible deformity of the skull**

Remember

- In all cases, the basic principles of first aid should be followed: assess danger at the scene, check airway, breathing, circulation; look for reduced awareness of surroundings or slowness or difficulty answering questions.
- Do not attempt to move the athlete (other than required for airway support) unless trained to do so.
- Do not remove helmet (if present) or other equipment.
- Assume a possible spinal cord injury in all cases of head injury.
- Athletes with known physical or developmental disabilities should have a lower threshold for removal from play.

If there are no Red Flags, identification of possible concussion should proceed as follows:

Concussion should be suspected after an impact to the head or body when the athlete seems different than usual. Such changes include the presence of **any one or more** of the following: visible clues of concussion, signs and symptoms (such as headache or unsteadiness), impaired brain function (e.g. confusion), or unusual behaviour.

CRT6™

Developed by: The Concussion in Sport Group (CISG)

Supported by:

International Olympic Committee · FEI · FIA · FIFA · IIHF · World Rugby

Child SCAT6™

Sport Concussion Assessment Tool

For Children Ages 8 to 12 Years

What is the SCAT6?

The Child SCAT6 is a standardised tool for evaluating concussions in children ages 8-12 years, and designed for use by Health Care Professionals (HCP). The Child SCAT6 cannot be performed correctly in less than 10-15 minutes. The Child SCAT6 is intended to be used in the acute phase, ideally within 72 hours (3 days), and up to 7 days, following injury. If greater than 7 days post-injury consider using the Child Sport Concussion Office Assessment Tool 6 (Child SCOAT6).[1]

The Child SCAT6 is used for evaluating children aged 8-12 years. For athletes aged 13 years or older, please use the SCAT6.[2]

If you are not an HCP, please use the Concussion Recognition Tool 6 (CRT6).[3]

Detailed instructions for use of the Child SCAT6 are provided as a supplement. Please read through these instructions carefully before using the Child SCAT6. Brief verbal instructions for each test are given in ***blue italics***. The only equipment required for the examiner is athletic tape and a watch or timer.

Recognise and Remove

A head impact by either a direct blow or indirect transmission of force to the head can be associated with serious and potentially fatal consequences. If there are significant concerns, including any of the RED FLAGS listed in Box 1 indicating signs that require urgent medical attention, and if a qualified medical practitioner is not present for immediate sideline assessment, then activation of emergency procedures and urgent transport to the nearest hospital should be arranged.

Key Points

- Any child with suspected concussion should be IMMEDIATELY REMOVED FROM PLAY, medically assessed, and monitored for injury-related signs, including deterioration of clinical condition.
- No child with a suspected concussion should be returned to play on the day of injury.
- If a child is suspected of having a concussion, and medical personnel are not immediately available, the child should be referred (or transported if needed) to a medical facility for assessment.
- Children with suspected or diagnosed concussion should not be given medications such as aspirin, anti-inflammatories, sedatives or opiates.
- Concussion signs and symptoms may evolve over time and it is important to monitor the child for ongoing, worsening, or development of concussion-related symptoms.
- The Child SCAT6 should not be used in isolation in making post-acute return to play decisions.
- The diagnosis of a concussion is a clinical determination made by a HCP. The Child SCAT6 should NOT be used by itself to make, or exclude, the diagnosis of concussion. It is important to note that a child may have a concussion even if their Child SCAT6 assessment is within normal limits.

Remember

- The basic principles of first aid should be followed: assess danger at the scene, child responsiveness, airway, breathing, and circulation.
- Do not attempt to move an unconscious/unresponsive child (other than that required for airway management) unless trained to do so.
- Assessment for a spinal and/or spinal cord injury is a critical part of the initial on-field assessment. Do not attempt to assess the spine unless trained to do so.
- Do not remove a helmet or any other equipment unless trained to do so safely.

Completion Guide

Blue: Required part of assessment

Orange: Optional part of assessment

For use by Health Care Professionals Only — **Child SCAT6™**

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SCAT6™

Sport Concussion Assessment Tool

For Adolescents (13 years +) & Adults

What is the SCAT6?

The SCAT6 is a standardised tool for evaluating concussions designed for use by Health Care Professionals (HCPs). The SCAT6 cannot be performed correctly in less than 10-15 minutes. Except for the symptoms scale, the SCAT6 is intended to be used in the acute phase, ideally within 72 hours (3 days), and up to 7 days, following injury. If greater than 7 days post-injury, consider using the SCOAT6/Child SCOAT6.

The SCAT6 is used for evaluating athletes aged 13 years and older. For children aged 12 years or younger, please use the Child SCAT6.

If you are not an HCP, please use the Concussion Recognition Tool 6 (CRT6).

Preseason baseline testing with the SCAT6 can be helpful for interpreting post-injury test scores but is not required for that purpose. Detailed instructions for use of the SCAT6 are provided as a supplement. Please read through these instructions carefully before testing the athlete. Brief verbal instructions for each test are given in ***blue italics***. The only equipment required for the examiner is athletic tape and a watch or timer.

Recognise and Remove

A head impact by either a direct blow or indirect transmission of force to the head can be associated with serious and potentially fatal consequences. If there are significant concerns, which may include any of the Red Flags listed in Box 1, the athlete requires urgent medical attention, and if a qualified medical practitioner is not available for immediate assessment, then activation of emergency procedures and urgent transport to the nearest hospital or medical facility should be arranged.

Completion Guide

Orange: Optional part of assessment

Key Points

- Any athlete with suspected concussion should be REMOVED FROM PLAY, medically assessed, and monitored for injury-related signs and symptoms, including deterioration of their clinical condition.
- No athlete diagnosed with concussion should return to play on the day of injury.
- If an athlete is suspected of having a concussion and medical personnel are not immediately available, the athlete should be referred (or transported if needed) to a medical facility for assessment.
- Athletes with suspected or diagnosed concussion should not take medications such as aspirin or other anti-inflammatories, sedatives or opiates, drink alcohol or use recreational drugs and should not drive a motor vehicle until cleared to do so by a medical professional.
- Concussion signs and symptoms may evolve over time; it is important to monitor the athlete for ongoing, worsening, or the development of additional concussion-related symptoms.
- The diagnosis of concussion is a clinical determination made by an HCP.
- The SCAT6 should NOT be used by itself to make, or exclude, the diagnosis of concussion. It is important to note that an athlete may have a concussion even if their SCAT6 assessment is within normal limits.

Remember

- The basic principles of first aid should be followed: assess danger at the scene, athlete responsiveness, airway, breathing, and circulation.
- Do not attempt to move an unconscious/unresponsive athlete (other than what is required for airway management) unless trained to do so.
- Assessment for a spinal and/or spinal cord injury is a critical part of the initial on-field evaluation. Do not attempt to assess the spine unless trained to do so.
- Do not remove a helmet or any other equipment unless trained to do so safely.

For use by Health Care Professionals Only **SCAT6™**

Developed by: The Concussion in Sport Group (CISG)

Supported by:

International Olympic Committee

Child SCOAT6™

Sport Concussion Office Assessment Tool

For Children Ages 8 to 12 Years

What is the Child SCOAT6?*

The Child SCOAT6 is a tool for evaluating concussions in a controlled office environment by Health Care Professionals (HCP) typically from 72 hours (3 days) following a sport-related concussion.

Brief verbal instructions for some components of the Child SCOAT6 are included. Detailed instructions for use of the Child SCOAT6 are provided in an accompanying document. Please read through these instructions carefully before using the Child SCOAT6.

The diagnosis of concussion is a clinical determination made by an HCP. The various components of the Child SCOAT6 may assist with the clinical assessment and help guide individualised management.

The Child SCOAT6 is used for evaluating athletes aged 8 - 12 years. For athletes aged 13 years and older, please use the SCOAT6.

Completion Guide

Blue: Complete only at first assessment | **Green: Recommended part of assessment** | **Orange: Optional part of assessment**

Athlete's Name:

Date of Birth: **Sex:** Male ☐ Female ☐ Prefer Not To Say ☐

Sport:

Age First Played Contact Sport: **School Class/Grade/Level:**

Handedness (Writing): L ☐ R ☐ Ambidextrous ☐ **Handedness (Sport):** L ☐ R ☐ Ambidextrous ☐

Dominant Leg (Sport): L ☐ R ☐ Ambidextrous ☐

Name of Accompanying Parent/Carer:

Examiner: **Date of Examination:**

Referring Physician's Name:

Referring Physician's Contact Details:

* In reviewing studies informing the SCOAT6 and Child SCOAT6, the period defined for the included papers was 3–30 days. HCPs may choose to use the Child SCOAT6 beyond this timeframe but should be aware of the parameters of the review.

For use by Health Care Professionals Only **Child SCOAT6™**

Developed by: The Concussion in Sport Group (CISG)

Supported by:

International Olympic Committee · FEI · FIA · FIFA · IIHF · World Rugby

SCOAT6™

Sport Concussion Office Assessment Tool

For Adults & Adolescents (13 years +)

What is the SCOAT6?*

The SCOAT6 is a tool for evaluating concussion in a controlled office environment by Health Care Professionals (HCP) typically from 72 hours (3 days) following a sport-related concussion.

The diagnosis of concussion is a clinical determination made by an HCP. The various components of the SCOAT6 may assist with the clinical assessment and help guide individualised management.

The SCOAT6 is used for evaluating athletes aged 13 years and older. For children aged 12 years or younger, please use the Child SCOAT6.

Brief verbal instructions for some components of the SCOAT6 are included. Detailed instructions for use of the SCOAT6 are provided in an accompanying document. Please read through these instructions carefully before using the SCOAT6.

Completion Guide

Blue: Complete only at first assessment | **Green: Recommended part of assessment** | **Orange: Optional part of assessment**

Athlete's Name:

Date of Birth: **Sex:** Male ☐ Female ☐ Prefer Not To Say ☐ Other

Sport:

Occupational or Educational Status:

Current or Highest Educational Level or Qualification Achieved:

Examiner: **Date of Examination:**

Referring Physician's Name:

Referring Physician's Contact Details:

* In reviewing studies informing the SCOAT6 and Child SCOAT6, the period defined for the included papers was 3–30 days. HCPs may choose to use the SCOAT6 beyond this timeframe but should be aware of the parameters of the review.

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Sports Physical II Quiz

1. What are the "Red Flags" of cardiovascular history/physical that should prompt further evaluation prior to clearance? *Go around the table and list one "Red Flag":*

- Syncope or near-syncope on exertion
- Chest Pain/Discomfort on exertion
- Palpitations at rest
- Excessive SOB or fatigue with activities
- FHx of Marfan, cardiomyopathy, Long QT, or clinically significant arrhythmias
- FHx of premature, sudden death
- Irregular heart rhythm
- Weak or delayed femoral pulses
- Fixed, split S2
- Systolic murmur louder than 3/6
- Any diastolic murmur
- Stigmata of Marfan's
- Chest pain in Turners syndrome

2. The incidence of sudden cariac death ranges from 1:917,000 to 1:3,000
   Complete the following table:

Condition	Mechanism of sudden death
Hypertrophic cardiomyopathy	Myofibrillary disarray → re-entrant ventricular arrythmias
Congenital CA anomalies	? Ischemia when myocardial demands ↑ w/exercise ?
Marfan Syndrome	Aortic root dilation → Dissection and rupture of the aorta
Long QT Syndrome	LQTS → Ventricular fibrillation
Commotio Cordis	Enhanced energy transfer to heart → arrythmias

3. Based on Bethesda Conference Sports Classifications, what sports are young athletes cleared for, who have the following heart conditions? (See Table 3 and Bethesda Classification and have residents list specific sports in each category)
   a. Marfan syndrome with normal aortic root diameter; currently cleared by cardiology for **class IA and IIA** competitive sports play.
   b. Recent dx of SVT with episodes causing breathlessness and dizziness, recently started on medication, currently cleared by cardiology for **class IA** sports.
   c. Moderate Mitral Regurgitation with mild LVH, currently cleared by cardiology for **IA-IB-IC, IIA-IIB-IIC** competitive sports play.

4. **CONCUSSION True or False:**
   A. Concussions result only from a direct blow to the head, face, or neck. False *(also from acceleration/deceleration or rotational injury)*
   B. Concussions result in structural injury to the brain. False *(usually no structural injury and standard neuroimaging is negative)*
   C. Loss of consciousness is a critical historical clue that determines concussion management. False *(LOC seen in <10% of concussions, management is symptom based)*
   D. Concussion results in an impairment in neurologic function that usually resolves spontaneously. True
   E. The developing brain is more vulnerable to reinjury & may take longer to heal from TBI. True *(Second-impact syndrome has never been seen in >20yo athletes; high school students average 10-14 days to recover from concussion, vs 5-7 days for college athletes, 3-5 days for adults, based on computerized neuropsych testing).*

5. What are the "Red Flags" of concussion history/physical that suggest prolonged recovery or caution for return-to-play? What clinical features would *also* indicate neuro-imaging?

Red Flags for Prolonged Recovery	Indications for Neuroimaging
≥ 3 sxs at presentation	LOC or amnesia + HA, vomiting, short term memory deficits, seizures, GCS < 15, coagulopathy, focal neuro deficits
Specific symptoms (fatigue, fogginess)	
Amnesia	No LOC or amnesia, but focal neuro deficits, vomiting, severe headache, signs of basilar skull fracture, GCS <15, coagulopathy, significant mech of injury
HA > 60hrs	
LOC > 60sec	
h/o prior concussion	LOC > 60sec (AAP)
Age < 18 years	Focal neurologic findings/deficits (AAP)
Comorbid conditions; med use	Evidence of a skull fracture (AAP)
High-risk sport (contact, collision)	

Sports Physical II Cases

Case 1:
Michael is a 17 year-old male who comes to see you with his mom. He is on the varsity football team and the regional championship series is about to start this weekend. The first game is against his school's biggest rival, the Honey Badgers. He forgot to come in before the season started, but because he is one of his team's star players, his coach let him slide. Now the school administration has become aware and he cannot play in this weekend's big game until you sign his forms. He thinks a scout will be there. "Help me, Doc!"

What do you need to know in order to complete his sports clearance paperwork?
See Sports Physical I Case . . . It will be more important to pay extra special attention to prior injuries in this patient as he plays a high contact sport, especially joint and head injuries.

His past medical history is benign, no major illness, no hospitalizations. He takes no medications or supplements except a protein powder after practice. Family history unremarkable: no cardiac history or sudden death. When you ask specifically about injuries, Mom does report that he broke his ankle as a freshman and had it pinned by orthopedics, but "they said he was ok to play now." He denies any pain or instability when playing football. You ask him if he has ever had a concussion. He says, "No way, doc. I'm pretty tough. I've never passed out from a hit."

Do you want to know anything else about his injury history?
Many adolescents/athletes do not consider an injury where they do not lose consciousness to be a concussion. You may need to do directed and specific questioning about any type of hit to the head, feeling "dazed" or "foggy," memory loss, headaches following a hit to the head, difficulty playing or practicing following a hit to the head, and any type of injury that resulted in LOC.

On further questioning, with mom's prompting, Michael admits that he has had a few hits where he felt dazed for a few minutes afterwards, but he says he felt fine after a few minutes and was able to still play, so he never told his coach. The most recent of these was less than 1 month ago. He denies any memory loss before or after any of his hits. He does say that he occasionally gets headaches "just like everybody" but they are "no big deal" and respond to Motrin.

What are you concerned about? Will you clear Michael to participate in his big game against the Honey Badgers this weekend? "Com'on Doc!"
There are two potential concerns: Second-Impact Syndrome or Post-concussion Syndrome.

1. Second-Impact Syndrome: Athlete who has sustained an initial head injury sustains a second before the symptoms associated with the first have fully cleared. Results in cerebral vascular congestion, which can progress to diffuse cerebral swelling and death.

2. Post-concussion Syndrome: Presence of cognitive, physical, or emotional symptoms of a concussion lasting longer than expected, with a threshold of 1 to 6 weeks of persistent symptoms after a concussion to make the diagnosis *(from Extra-Credit CPG).*

You discuss some of these concerns with Michael and his mom, and she is surprised that none of his football coaches have ever called his "head dings" concussions and educated them about potential consequences. "What should we do if he gets another concussion? Are there any precautions we can take?"

What sort of anticipatory guidance will you give Michael and his mother?

- No evidence that protective gear prevents concussions, but helmets and mouth guards reduce risk of skull and dental fractures, so should continue to be worn.
- Encourage concussion education for Michael & coaches through CDC's "Heads Up".
  - Symptoms of concussion: *See Table 2—physical, cognitive, emotional, sleep*
  - Initial evaluation: *See Figure 1 & Table 3—e.g. sideline evaluation w/SCAT2*
  - Management: *See Figure 1 & Table 6—rest, then graded return-to-play*

TABLE 5 Concussion Rehabilitation/Stepwise Return to Play

Rehabilitation Stage	Functional Exercise
1. No activity	Complete physical and cognitive rest
2. Light aerobic activity	Walking, swimming, stationary cycling at 70% maximum heart rate; no resistance exercises
3. Sport-specific exercise	Specific sport-related drills but no head impact
4. Noncontact training drills	More complex drills, may start light resistance training
5. Full-contact practice	After medical clearance, participate in normal training
6. Return to play	Normal game play

Each stage in concussion rehabilitation should last no less than 24 hours with a minimum of 5 days required to consider a full return to competition. If symptoms recur during the rehabilitation program, the athlete should stop immediately. Once asymptomatic after at least another 24 hours, the athlete should resume at the previous asymptomatic level and try to progress again. Athletes should contact their health care provider if symptoms recur. Any athlete with multiple concussions or prolonged symptoms may require a longer concussion-rehabilitation program, which is ideally created by a physician who is experienced in concussion management.

- Those with multiple concussions, like Michael, have increased risk of recurrent injury and have longer recovery periods. However, there are no guidelines regarding athletic disqualification or retirement.

Bonus: What is the law which requires concussion education for coaches, athletes, and parents? Has it been enacted in Maryland, D.C., and Virginia?

The Zackery Lystedt Law has been enacted in all 3 states (in 34 states total). The law also mandates removal of athletes from activity if there is any suspicion of concussion, and return to play must be cleared by a licensed health-care professional.

Case 2:

Jay is a 15 year-old male who presents to clinic on "Sports Physical Day". You have churned through 4 physicals so far. The corpsmen bring you his vitals sheet and you note the following:

HR 90 BP 145/95 (>95th %ile) Weight 52 kg (25th %ile) Height 184cm (95th %ile)

What history is most important to obtain when Jay is brought back to your room?

See Sports Physical I Case . . . Cardiac history is particularly important in this hypertensive patient. See 12-Element AHA Recommendations for Preparticipation Screening (*Module 1):*

***Personal History*:**

1. Exertional chest pain/discomfort,
2. Unexplained syncope/near-syncope,
3. Excessive exertional dyspnea
4. Prior recognition of a heart murmur
5. **Elevated systemic blood pressure**.

Family History:

6. Premature death (<50) d/t heart disease
7. Disability from heart disease in <50y rel.
8. Cardiac conditions in family: HCM, DCM, LQTS, Marfan, arrhythmias.

Jay tells you that he is center for his school's basketball team, but also wants to start weight-lifting to "bulk up". He denies exertional chest pain or dyspnea, syncope, or history of heart murmur. PMHx is positive for history of "shoulder dislocation" after a collision with another player during a game. Jay's athletic trainer relocated his shoulder, and he has had no other joint issues. His HEADSS exam is unremarkable, and he denies use of alcohol, tobacco, or other recreational drugs, including supplements. His father reports history of HTN in multiple family members, but no other cardiac disease or premature deaths.

What will you focus on during your physical exam?

Include standard health supervision evaluation, with focused orthopedic exam (e.g. 2min Orthopedic Exam). Cardiovascular exam should include 12-Element AHA recommendations:

Physical Examination:

9. Heart mumur (auscultate supine and standing, to pick up murmurs of dynamic LVOT obstruction)
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan (esp. considering Jay's height and history of joint dislocation)
12. Brachial artery blood pressure (sitting position)

Because of HTN noted on VS, would also obtain 4-extremity blood pressures.

On your exam, you note that he has a thin body habitus. HR is regular. There are no murmurs or extra heart sounds, and femoral pulses are 2+ bilaterally. Lungs are clear. There is no organomegaly. 2-min orthopedic exam is normal, and there is no kyphoscoliosis, pectus deformity, joint hypermobility, or arachnodactyly. He is Tanner 5 and has no hernias.

What is your assessment of Jay?

- Based on single reading, Jay would meet criteria for Stage I Hypertension (>95th%ile but < 99th%ile + 5mmHg for age, gender, height). ***Confirm with appropriately-sized cuff and 2 additional BP measurements at separate visits*** (see Hypertension Module)

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
15	50th	109	110	112	113	115	117	117	61	62	63	64	65	66	66
	90th	122	124	125	127	129	130	131	76	77	78	79	80	80	81
	95th	126	127	129	131	133	134	135	81	81	82	83	84	85	85
	99th	134	135	136	138	140	142	142	88	89	90	91	92	93	93

- Does not appear to meet criteria for Marfan's Syndrome (see Ghent Nosology).

What will you write on his Pre-participation Evaluation Form *(Cleared, Cleared with further evaluation, Not cleared)*? Can Jay still participate in basketball & weight-lifting?
Answers may vary; points to discuss include the following:

- According to "Medical Conditions & Sports Participation" (*see Module I*), patients with sustained HTN (>95th%ile) need evaluation, but should continue to participate in sports.
- Patients with severe HTN (>5mmHg + 99th%ile) should avoid high-static sports (*see Table 3—includes weight-lifting).*
- Consider EKG, if/when HTN is confirmed over 3 separate readings (e.g. LVH results from chronic HTN; one cause of HTN is aortic coarctation).
- *Differential diagnosis of HTN is beyond the scope of this module, but may need lab work-up (CBC, RFP, U/A, TFTs, lipids) and referral to Renal and/or Cardiology.*

What are the absolute contraindications to sports participation? Is HTN included?
See Table 2: pulmonary vascular disease with cyanosis; severe Pm HTN; severe AS, AR, MS, MR; cardiomyopathies; vascular EDS; coronary anomalies; acute pericarditis, myocarditis, Kawasaki disease; CPVT. (These Bethesda eligibility criteria do NOT apply to non-competitive, recreational activities. The AHA has published separate guidelines).

Before Jay leaves with his signed forms, his father asked whether you will do a "screening EKG" for Jay, as he has read in the news that this can prevent sudden death. *Imagine that Jay's exam was completely normal*, how will you respond?
Answers may vary; points to discuss include the following:

- The cost-to-benefit ratio of obtaining a screening EKG or echo continues to be debated in the US. The European Society of Cardiology, largely based on the Italian experience which showed an 89% relative risk reduction in sudden cardiac death, recommends mandatory EKG screening of all competitive athletes. The AHA does not. One analysis by the JACC found that EKG screenings of all young competitive athletes in the US would cost $69 billion over 20 years and save about 4,183 lives, making the cost per life saved over $10 million.
- In arguing against mandatory EKG screening, the AHA often notes the "human costs" of false positives, which can result in additional potentially unnecessary tests and removal from play of athletes who are not actually at risk.
- Screening considerations differ between Italy and the US, as 25% of Italian cases of sports related SCD are due to the same heritable arrhythmia (ARVD), and the total athlete population much smaller. This impacts the pre-test probability of an EKG screen and the feasibility of universal screening.

Sports Physical II Board Review

1. A 14-year-old boy loses consciousness while playing basketball. He regains consciousness in 30 seconds and is transported to a pediatric emergency department. Results of head computed tomography scan, electroencephalography, and echocardiography are within normal limits. Electrocardiography results are interpreted as abnormal, with a heart rate of 90 beats/min, PR interval of 150 msec, and QTc interval of 550.

Of the following, the MOST likely explanation for this patient's syncopal episode is

A. complete atrioventricular block
B. first-degree atrioventricular block
C. hypertrophic cardiomyopathy
D. long QT syndrome
E. supraventricular tachycardia due to Wolff-Parkinson-White syndrome

The young child or adolescent who experiences an episode of syncope must undergo 12-lead electrocardiography (ECG) as a part of his or her evaluation. Findings on ECG may indicate the possibility of a rhythm disturbance or conduction disorder. However, the corrected QT interval must be measured to assess for the possible diagnosis of long QT syndrome. Findings on ECG almost always are abnormal in the patient who has symptomatic long QT syndrome. In addition to a prolonged corrected QT interval, there may be bizarre or notched T waves and prominent U waves. Exercise testing may elicit abnormalities not seen on resting ECG.

Patients who have long QT syndrome are at risk for life-threatening ventricular tachycardia, torsades de pointes, and ventricular fibrillation. The syndrome may have an autosomal dominant or autosomal recessive inheritance pattern or may be a new mutation. Many of the mutations causing long QT syndrome demonstrate ion channel abnormalities. Clinical laboratory testing is available. Pharmacologic therapy and implantation of automatic cardiovertor-defibrillators are the currently employed treatment modalities. Affected patients may present with cardiac arrest, syncope, seizures, or palpitations. Any patient who presents with suspicious symptoms in whom ECG identifies a corrected QT interval greater than 450 msec warrants specialty evaluation.

Patients who have complete atrioventricular block also may present with syncope. However, the teenager who has complete atrioventricular block would have a resting heart rate dramatically lower than 70 beats/min (typically in the range of 40 to 60 beats/min), and the ECG would demonstrate a profound conduction disturbance characterized by a lack of relationship between the atrial and ventricular rates. It is atypical for first-degree atrioventricular block to result in syncope. In addition, first-degree block is identified easily on baseline ECG by the PR interval exceeding approximately 180 msec.

Hypertrophic cardiomyopathy can present with syncope due to either obstruction of left ventricular outflow and resultant hypotension or ventricular arrhythmias caused by the disturbance to repolarization. However, the corrected QT interval is not markedly prolonged. In addition, the findings of hypertrophic cardiomyopathy are readily discerned by echocardiography. Supraventricular tachycardia due to Wolff-Parkinson-White syndrome can result in syncope because patients are at risk for degeneration of their arrhythmia to atrial and subsequently ventricular fibrillation. However, baseline ECG should demonstrate the classic features of a short PR interval and a delta wave.

2. A 16-year-old girl who is new to your practice comes to the clinic for a physical examination prior to enrollment in a summer volleyball camp. She is generally healthy, and she does well academically. On physical examination, you note that she is unusually tall and slender, and she appears to have long fingers and toes. You are concerned that she could have Marfan syndrome, and you refer her for a clinical genetics evaluation.

Of the following, the additional finding that would MOST strongly suggest the diagnosis of Marfan syndrome for this girl is

A. high myopia
B. long, narrow face
C. mitral valve prolapsed
D. narrow palatal contour
E. spontaneous pneumothorax

Marfan syndrome (MS) is an autosomal dominant connective tissue disorder that has a prevalence of 1 in 10,000 and usually is caused by alterations in the fibrillin 1 (FBN1) gene. MS primarily involves the skeletal, cardiovascular, and ocular systems. Skeletal features include pectus carinatum, pectus excavatum, reduced upper-to-lower segment ratio, scoliosis of greater than 20 degrees or spondylotlisthesis, reduced elbow extension, joint hypermobility, and others. Major cardiovascular features are dilation or dissection of the ascending aorta, and minor features include mitral valve prolapse, dilation of the main pulmonary artery, and calcification of the mitral annulus. Ocular features include ectopia lentis, flat cornea, hypoplastic iris, and increased axial length of the globe. Although high myopia; long, narrow face; mitral valve prolapse; and narrow palatal contour all are associated with MS, they are also relatively common findings in other syndromes and in the general population. Spontaneous pneumothorax occurring in a teenager, however, is unusual and is one of the minor criteria for the diagnosis of MS. Therefore, the history of spontaneous pneumothorax associated with this girl's physical features should increase the pediatrician's suspicion for MS.

Due to the risks for aortic root dilatation and dissection associated with MS, affected individuals are asked not to participate in contact or competitive sports or isometric exercise. In addition, they should avoid activities placing them at increased risk for joint injury or pain.

3. An 18-year-old girl presents with a history of occasional mild chest pain of 1 week's duration. The episodes occur at rest and have not affected her performance as a competitive long-distance swimmer. On physical examination, her heart rate is 48 beats/min and blood pressure is 105/65 mm Hg. Electrocardiography demonstrates left ventricular hypertrophy, which is confirmed by echocardiography.

Of the following, the MOST likely cause of these findings is

A. aortic stenosis
B. athlete's heart
C. cardiac conduction disturbance
D. coronary artery anomaly
E. hypertrophic cardiomyopathy

Chest pain is a common complaint in the general pediatric practice, and no significant cardiac abnormality is found in most cases. Musculoskeletal pain is the most common cause of chest pain in the child and adolescent and is the most likely source of the mild pain reported for the girl in the vignette.

Rarely, however, chest pain is a sign of cardiac pathology such as pericarditis, myocarditis, or myocardial infarction. The lack of pain during exercise (when the individual would be expected to be in a high myocardial demand state) for this girl is reassuring, suggesting that the pain is of a benign, noncardiac origin. In addition, the lack of associated symptoms such as radiation of the pain to the face, arms, or back; associated dizziness or syncope; and PE findings suggestive of CVD are all reassuring.

The low blood pressure and heart rate reported for this girl are not of particular concern and most likely reflect athlete's heart. The well-trained athlete exhibits findings on physical examination and laboratory assessment that differ from the general population. Specifically, the resting heart rate typically is 20% to 30% lower than the average for age in the population, and blood pressure is at the lower range of normal. Often, a physiologic flow murmur can be detected, which is caused by a high cardiac output state. Findings on electrocardiography can include left ventricular hypertrophy. Echocardiographic findings can include left ventricular wall thickening/hypertrophy associated with normal or hyperdynamic left ventricular contractility. Such physiologic left ventricular hypertrophy can be distinguished from pathologic hypertrophic cardiomyopathy based upon features of diastolic function, mitral valve disease, and assessment of left ventricular outflow tract obstruction. The patient who has hypertrophic cardiomyopathy exhibits mitral regurgitation, obstruction to blood flow due to the narrowed left ventricular outflow tract, and diastolic dysfunction.

The individual who has athlete's heart manifests none of these pathologic echocardiographic features. Novel imaging methods (including cardiac magnetic resonance imaging) can differentiate hypertrophic cardiomyopathy from left ventricular hypertrophy associated with athlete's heart. In addition, when the well-trained individual who has athlete's heart is deconditioned for several months, echocardiography demonstrates resolution of the hypertrophy.

Aortic valve stenosis of such severity to cause chest pain always is associated with a pronounced systolic ejection-type murmur that is heard most commonly at the left mid-sternal border, with radiation to the right infra-clavicular region. Cardiac conduction abnormalities can manifest as chest pain in the young child who has difficulty distinguishing pain from other unusual chest symptoms such as palpitations. However, the teenager is unlikely to describe palpitations or other rhythm disturbances as evoking chest pain. Coronary artery anomalies are rare and include acquired conditions such as Kawasaki disease and congenital lesions such as anomalous left coronary arising from the pulmonary artery (ALCAPA) and coronary fistulae. Coronary anomalies do not cause left ventricular hypertrophy and should be detected by detailed echocardiography. *As noted previously, hypertrophic cardiomyopathy can be distinguished from the left ventricular hypertrophy associated with athlete's heart by the presence of a pathologic murmur caused by either mitral regurgitation or left ventricular outflow tract obstruction, findings on echocardiography, and chest pain that is more likely to occur during exercise and have associated dizziness, syncope, and radiation of the pain.*

4. A family has just relocated to your community, and you are evaluating their 12-year-old son for the first time this afternoon. Family history reveals that the boy's father and grandmother had premature cardiovascular disease. The boy's parents are concerned about risk of heart disease.

Of the following, the MOST important next step in this child's evaluation is

A. echocardiography
B. electrocardiography
C. fasting lipoprotein analysis
D. random cholesterol measurement
E. referral to the cardiology clinic

In recent years, an increasing body of literature has indicated that atherosclerotic disease and its effect on the cardiovascular system are progressive processes that begin during early childhood. Research has demonstrated that the complex process of acquired cardiovascular disease is the result of genetic predisposition, along with factors such as diet, physical activity, and other comorbidities.

In adults, the strongest risk factors for the development of cardiovascular disease include a high concentration of low-density lipoprotein, a low concentration of high-density lipoprotein, elevated blood pressure, type 1 or 2 diabetes mellitus, cigarette smoking, and obesity. Research in children and adolescents has shown that some of these risk factors may be present in early childhood. It is imperative, therefore, for pediatricians to take proactive roles in stressing the importance of healthy cardiovascular lifestyles and identifying children at risk for cardiovascular disease.

The importance of the history, especially the family history, cannot be overemphasized because the clinical manifestations of hypercholesterolemia are variable and may not be physically present until later in childhood, adolescence, or even adulthood. Some children who have homozygous familial hypercholesterolemia may demonstrate cutaneous or tendinous xanthomas, but often these findings are not apparent until early adulthood. As a result, some children who have significant hyper-cholesterolemia may have normal findings on physical examination.

The American Academy of Pediatrics has adopted the recommendation that all children undergo cholesterol screening between 9 and 11 years old. Accordingly, the boy in the vignette should undergo a screening test for lipoproteins that includes cholesterol, high-density lipoproteins, and low-density lipoproteins in the fasting state.

Random cholesterol screening may provide important information, but taken in isolation, will not offer as much information as a fasting lipoprotein panel. Referral to a specialized clinic such as cardiology or endocrinology may be indicated, but this should be considered only after more complete information is obtained from the diagnostic evaluation. Neither echocardiography nor electrocardiography is indicated for this patient, and neither is used as a screening test for cardiovascular risk factors in children.