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NCAA Football Off-Season Training: Unanswered Prayers. . . A Prayer Answered

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Off-season training in year-round collegiate football is purported to be performance enhancing. Absent principles of exercise physiology, excesses in sport-training regimens pose risk to the participant athletes. Since 2000, 33 National Collegiate Athletic Association (NCAA) football players have died in sport: 27 nontraumatic deaths and 6 traumatic deaths, a ratio of 4.5 nontraumatic deaths for every traumatic death. On average, 2 NCAA football players die per season. Best practices, consensus guidelines, and precautions are ignored, elevating the risk. However, standards exist that will, if heeded, prevent nontraumatic death in athletes training for sport. Sickle cell trait status knowledge and tailored precautions are preventing deaths from exertional collapse associated with sickle cell trait. Adherence to established principles of exercise physiology and best-practice training standards, which is long overdue, will help to prevent not only deaths from exertional collapse associated with sickle cell trait but also sudden cardiac, exertional heat stroke, and asthma deaths.

Key Words: performance enhancement, year-round training, nontraumatic death

Yet another collegiate football player lay, unresponsive, in an intensive care unit after collapsing in an off-season college football performance-enhancement session. His teammates gathered, privately, in a nearby hospital chapel to pray for his life. Within hours, he was pronounced dead, their petition for his recovery unrequited. Sir Roger Bannister, the first to run a sub–4-minute mile, quite rightly stated, "The notion that courage and esprit de corps can somehow defeat the principles of physiology is not only wrong but dangerously wrong."[1] Contemporary National Collegiate Athletic Association (NCAA) football off-season performance enhancement boasts esprit de corps while, dangerously and wrongly, expecting to cheat physiological principles with predictable loss of life.

Football *is* a traumatic sport. In our practice and play of the game, we anticipate injury. . .traumatic injury. . .and even traumatic death. Collegiate football's dirty little secret is that *we* are killing our players—not in competition, almost never in practice, and rarely because of trauma—but primarily because of nontraumatic causes in off-season sessions alleged to enhance performance.

In the early 1900s, President Theodore Roosevelt challenged university presidents to reduce traumatic injury and death or abolish the game; formation of the NCAA ensued. The 1960s and early 1970s represented the deadliest era of football: on average, 31 players died each year, primarily because of traumatic head or neck injury.[2] Reducing traumatic deaths required a fundamental change in the game: the outlawing of tackling with the head as the point of initial contact, that is, *spearing*. This 1976 rule change lowered the incidence of these traumatic deaths by approximately 50% virtually overnight, and continued enforcement has rendered a progressive decline in the number of traumatic injury fatalities.[3] An untold number of players since 1976 owe their health and welfare to the stewards of the game who had the courage to act. Nontraumatic deaths have outstripped traumatic deaths in every decade since the 1976 spearing rule change.[4] In tragic irony, 1960–1976 was an era of excessive traumatic head/neck deaths at all levels of play; college was the only level of football in which nontraumatic deaths exceeded traumatic deaths.[2] Football awaits resolute collegiate leadership willing to take action to end nontraumatic training deaths.

The years 2000–2016 represent the age of year-round NCAA football and are the era of this review. The summary is 33 dead NCAA football players: 27 nontraumatic deaths and 6 traumatic deaths, a ratio of 4.5 nontraumatic deaths for every traumatic death. On average, 2 NCAA football players die per season.

Terminology used in this article is *traumatic* and *nontraumatic* rather than *direct* and *indirect*. *Indirect* implies "not directly caused". However, for players in the data set, training for sport was the direct, proximate cause of their deaths. The data set is drawn from (1) media accounts, (2) public records including autopsy reports, (3) case reports published by the National Center for Catastrophic Sports Injury Research (NCCSIR), and (4) personal communications with physicians, athletic trainers, coroners, medical examiners, and attorneys who had specific case knowledge.

Nontraumatic death in exercising football players is a known factor. The NCCSIR has been amassing data since 1931 and has shared the grim statistics in annual publications, with specific reporting to their research sponsors: the NCAA, the American Football Coaches Association, the National Athletic Trainers' Association, and the National Federation of State High School Associations. Known, too, are the 4 nontraumatic causes of death in exercising athletes: exertional heat stroke (EHS), sudden cardiac death (SCD), exertional collapse associated with sickle cell trait (ECAST), and asthma.[5] These 4 causes represent all NCAA football nontraumatic training deaths with a common theme in their

final workouts: unphysiological workload lacking sport specificity.

The workout before the lone asthma death called for 2160 yards of serial sprinting with a 1 : 1 work : rest ratio in approximately 12 minutes.[6] The work : rest ratio in a football game is typically about 1 : 8 or 1 : 10. In a "hurry-up" offense, the ratio can drop to 1 : 4, but this pace is typically not sustained. Physiologically unsound collegiate football workouts create undue risk, lack sport specificity, and are ineffective for performance enhancement.[7]

The classic picture of fatal EHS is of a highly motivated individual who participates in badly organized training and exerts himself beyond his capability. The onus is on those conducting the training to manage the organizational factors of work-rest cycles, exercise intensities that match physical fitness, and training schedules that avoid the hottest hours of the day.[8] Exertional heat stroke is *exertional* heat stroke, not *environmental* heat stroke. It is the result mainly of exercise rather than climate.[9] Exertional heat stroke "fatality factors" primarily concern organizational training regulations and not individual factors, thus emphasizing the importance of a proper organizational training climate in preventing EHS fatalities.[8]

Sudden cardiac death is excused as foreordained. This rationale ignores cause: "...cardiovascular collapse [in young athletes] was clearly associated with intense physical activity...."[10] Statistical evidence is crystal clear that collegiate football game stress does not ever result in SCD. All SCDs, save 1 November practice death in a noncontact drill, occurred during off-season workouts. Undoubtedly, collegiate football players with mild or covert cardiac anomalies are surviving, if not thriving, in football, and for decades thereafter, but some cannot survive the irrational intensity of an off-season workout.

Exertional collapse associated with sickle cell trait (SCT), like EHS, is a death due to too much, too fast, for too long. All 12 of the NCAA football deaths from ECAST were in athletes in "conditioning", 11 in Division I (DI) and 1 in Division II. The insult that creates the injury is sustained intense exercise: exertion that ignores risk and abandons precautions.

Statistical evidence of excess death in collegiate football dates back 30 years. Van Camp et al[5] mined the NCCSIR database to study deaths of high school and collegiate athletes from 1983 to 1993. They reported that a male collegiate athlete was twice as likely to die a nontraumatic death as compared with a male high school athlete. A defiant defender of the culture might, naively, dismiss these data as passé and not reflective of "contemporary" training techniques under the guidance of a credentialed professional strength and conditioning coach.

Boden et al[11] narrowed the target to nontraumatic deaths in football only, also using the NCCSIR database and overlapping the Van Camp years, in a retrospective review of fatalities from 1990 to 2010, which encompassed year-round football training. The risk of nontraumatic death rose to 3.6 for a collegiate versus a high school football player. The risks of SCD, EHS, and ECAST increased over time, being greater from 2000 to 2010 than in the 1990s. Evidence basis of the inherent danger collegiate football players face—"...preseason practices and intense conditioning sessions were vulnerable periods..."[11(p1108)]—is reflected.

Institutional investigation into the dangers of collegiate off-season training seems designed to seek, not find. The University of Central Florida's president ordered "...a review of the...football, strength and conditioning and sports medicine...as they relate to the health and well-being of the University's football student-athletes." The presidential directive carried a specification to not "investigate or to reach conclusions about specific past events" despite those specific past collapses yielding emergent hospitalizations with 1 player dying and 1 surviving.[12]

The University of Central Florida's hired investigator was dutiful in providing the institution with an alibi by blaming the culture without indicting it. "The conditioning activities and requirements for football student-athletes are rigorous, but within the range normal to other Division I programs."[12]

Killing DI football players in required rigorous conditioning activities is "normal." Since 2000, the number of DI football players who have died during February conditioning alone (5 nontraumatic deaths) exceeds the 2 DI football players who died from direct trauma in 16 seasons of games, in-season practices, preseason practices, and spring practices. In conditioning, no other sport kills as does football...no level kills at the rate of NCAA football...no division kills more players than DI. The nontraumatic toll is 8 dead in winter workouts, 6 in summer, 10 in August, 2 in September conditioning, and 1 in a November practice; 12 ECAST deaths, 9 SCDs, 5 EHS deaths, and 1 asthma death. "Conditioning as practiced in many high schools and colleges, including elite college programs is antiquated, scientifically unstudied, and can be, obviously, dangerous" (J. Moriarity, written communication, March 24, 2009).

Dead men tell no tales...but, occasionally, the victims live and lessons can be learned. Such an occasion emanates from University of Iowa Hawkeyes case reports. On day 2 of January winter workouts, the players were required to perform barbell snatches, pull-ups, dumbbell rows, and a weighted sled-pushing exercise before completing 100 back squats at 50% of their 1-repetition maximum. Subsequently 13 players were hospitalized with rhabdomyolysis.[13]

A select panel of Iowa faculty, commissioned by the university president, failed to find anyone who, in his or her duty, was negligent or reckless in the planning, conduct, or supervision of the workouts.[14] Iowa's team physician undertook independent, comparative studies. Ten of the 13 hospitalized players authorized release of their hospital records, and their creatine kinase (CK) levels were compared with those from a cohort of 32 Hawkeye football players in subsequent August preseason football practices.[13,15] On day 1 of August football practice, the mean CK was 285 U/L (highest CK = 4659 U/L). On day 3, the mean CK was 1300 U/L (highest CK = 12 067 U/L), and the level peaked on day 7 at 1562 U/L (highest CK = 7453 U/L).[16] These CK levels are consistent with those noted in prior studies[16,17] of preseason collegiate football players and decidedly inconsistent with those of the hospitalized Hawkeyes. From the afflicted in the "winter performance-enhancement workout," the lowest CK level was 97 000 U/L and was the only one below 100 000 U/L. Four athletes' CK levels were greater than 200 000 U/L; the highest was 330 000 U/L.[13] The mean peak CK level was about 120 times higher for the players in the winter workout versus those practicing football, evidencing the unphysiological nature of rigorous off-season football training.

The mantra of collegiate off-season football training is that it yields mental and physical toughness, discipline, and accountability, but the metrics are subjective. The method is

pushing players' limits in a belief that the only limits are self-imposed by the untough, the undisciplined, and the unaccountable. Coach Bill Mallory is about as old school as any and has the dubious distinction of having been the head football coach when 2 ECAST deaths occurred and finally has it figured out: "...the way we're conducting conditioning at this time is putting some people at risk."[18(p1180)] Though deeply embedded in the football culture, these high-risk, physically punishing workouts are no longer justifiable.

The Iowa faculty report mirrors factors that create risk for the participant players: (1) an absence of sports science in the design of the workout; (2) the peril of too much, too fast, for too long in a workout regimen; (3) the "too soon" that disallows acclimation to workload, if not acclimatization; (4) restricted recovery that compromises the work : rest ratio, rehydration, etc; (5) ad lib workouts that abandon scripted workouts consistent with the principles of exercise physiology—if indeed a script exists; and (6) zero evidence of players' behavioral actions as causing or contributing to their malady.[14]

Exertional collapse associated with sickle cell trait in football players with SCT is the leading cause of death in exercising NCAA football players since 2000. That ECAST can hold grave consequences for collegiate football players has, for decades, been detailed,[19–23] not least in the 2007 "Consensus Statement: Sickle Cell Trait and the Athlete."[24] As evidence of the limits of education, in the 3 years before the statement, there were 4 ECAST athlete deaths, and in the 3 years that followed, there were another 4 ECAST deaths in athletes, the last being that of Bennie Abram in a February 2010 DI football winter workout. In August 2010, the NCAA DI inserted SCT status knowledge, with allowance for athletes' written declinations, into the mandatory medical examination.

In the 10 years before the August 2010 mandated SCT screening, there were 10 ECAST deaths in DI football players participating in performance-enhancement sessions. Since August 2010, only 1 NCAA DI football player has died an ECAST death. Ten ECAST deaths in 10 years versus 1 ECAST death in 6 years renders statistical significance to a fact: ECAST deaths are being prevented in NCAA DI football with status knowledge and tailored precautions.[25,26]

There is no record, ever, of an exercising athlete with SCT dying when SCT status was known, athlete and pertinent athletic staff (athletic trainers, sport coaches, strength coaches, etc) were educated, and precautions were heeded. For the lone NCAA DI ECAST death since 2010, media reports reflect a failure to abide by established NCAA guidelines for the athlete with SCT, and the institution admitted its liability.[27,28]

National Collegiate Athletic Association DI football deaths continue apace, as 2014 was one of the deadliest off-seasons on record...and the clock is ticking on another football off-season of "performance" training, with 1 2017 sudden nontraumatic death only 1 to 2 hours after a morning football offseason conditioning workout. Adherence to "The Inter-Association Task Force for Preventing Sudden Death in Collegiate Conditioning Sessions: Best-Practices Recommendations"[29] document is long overdue. These standards have been endorsed by, among others, the Collegiate Strength and Conditioning Coaches Association and the National Strength and Conditioning Association. Ten best-practice points offer application to athletics health care providers, sport coaches, strength and conditioning coaches, and athletics administrators. Just like the 1976 rule change eliminating intentional spearing and the 2010 NCAA bylaw regarding preparticipation SCT status knowledge with tailored training precautions, adherence to established principles of exercise physiology and best-practice training standards will help to prevent not only ECAST deaths but also SCD, EHS, and asthma deaths.

Bridgette Lloyd, mother of Rice University football player Dale Lloyd—whose ECAST death precipitated the NCAA SCT screening bylaw—shared her prayer with me: "As a parent, I pray daily that no other family has to endure the pain that my family has endured since Dale's death. But most importantly I pray that no other young athlete's life is cut short by this very preventable situation." A grieving mother's prayer to the Almighty has been answered and is in our hands. No other young man, or woman, should die a preventable nontraumatic training death.

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