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Advanced Sports Medicine Concepts and Controversies

The Role of Controlled Exercise in Concussion Management

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Abstract

Concussion affects the autonomic nervous system and its control of cerebral blood flow, which may be why uncontrolled activity can exacerbate symptoms after concussion. Traditionally, patients have been advised to restrict physical and cognitive activity until all symptoms resolve. However, recent research suggests that prolonged rest beyond the first couple of days after a concussion might hinder rather than aid recovery. Humans do not respond well to removal from their social and physical environments, and sustained rest adversely affects the physiology of concussion and can lead to physical deconditioning and reactive depression. Some animal data show that early forced exercise is detrimental to recovery after concussion, but other animal data show that voluntary exercise is not detrimental to recovery. We developed the Buffalo Concussion Treadmill Test to systematically evaluate exercise tolerance in persons with prolonged symptoms after concussion (ie, more than 4-6 weeks, which is called postconcussion syndrome [PCS]). Using a predetermined stopping criterion (symptom-exacerbation threshold), akin to voluntary exercise in animals, the Buffalo Concussion Treadmill Test is the only functional test known to safely and reliably reveal exercise intolerance in humans with PCS. The test data are used to develop individualized subthreshold exercise treatment programs to restore the physiology to normal and enhance recovery. Return of normal exercise tolerance can then be used to establish physiological recovery from concussion. New research suggests that absolute rest beyond the first few days after concussion may be detrimental to concussion recovery. However, further research is required to determine the appropriate mode, duration, intensity, and frequency of exercise during the acute recovery phase of a concussion prior to making specific exercise recommendations. For patients with PCS, subsymptom threshold exercise improves activity tolerance and is an appropriate treatment option for this patient population.

Introduction

The International Concussion in Sport Group currently supports the concept that concussion management should promote physical and cognitive rest until acute symptoms resolve [1]. The rationale for rest as treatment hinges primarily on data from elegant animal experiments that show a cascade of increased metabolic demand acutely after simulated concussion that occurs—paradoxically, it would seem—during a state of reduced cerebral blood flow (CBF) at rest [2]. The clinical inference has generally been that nonessential physical or cognitive activity diverts essential oxygen and glucose away from injured neurons and delays recovery from concussion. A consequence of this inference is that many clinicians advise concussed patients to restrict most if not all physical and cognitive activity, including schoolwork, until all symptoms have resolved because of a theoretical risk that activity-induced exacerbation of symptoms damages the brain and delays recovery. This standard of care has been implemented for many years despite a lack of empirical evidence that such "radical rest" is therapeutic [3,4] and without consideration that symptoms after concussion are protean and not specific to brain injury [5].

Recent studies have begun to challenge the utility of prolonged rest as treatment for concussion. Citing the risk for prolonged and exacerbated symptoms that may not be directly related to the concussive injury, some medical organizations have recommended that athletes be permitted to engage in limited physical and cognitive activity as long as it does not worsen symptoms [6]. In support of this approach, recent studies predominantly focusing on patients with postconcussion syndrome (PCS) have shown that more liberal noncontact activity recommendations [7,8] and controlled, subsymptom threshold aerobic exercise [9-11] may enhance recovery

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after concussion, particularly in persons with PCS. The purpose of this article is to discuss the scientific basis for exercise in the treatment of concussion.

Literature Review Methodology

Inclusion Criteria

For the human studies, we searched PubMed and MEDLINE for articles with key words that included "concussion," "postconcussion syndrome," "mild traumatic brain injury" (mTBI), "exercise," "physical," "activity," "therapy," "treatment," "cerebral blood flow," "carbon dioxide," "cerebrovascular reactivity," "autonomic," and "physiology." For the concept of treatment, we included only the articles that focused on aerobic exercise treatment of concussion or PCS. The search terms for the animal studies included "exercise," "traumatic," "brain," "injury," and "rodent." We included only the studies that focused on concussion/mTBI effects. We included articles that looked at treatment both in the acute and chronic phases after concussion. The bibliographies of relevant articles also provided citations.

Exclusion Criteria

Exclusion criteria were non—English language articles, articles before the year 2000, case series, and review articles (except for review articles that relate to the discussion of more general aspects of concussion and PCS). For the human studies, we read each abstract and excluded any that did not feature aerobic exercise for assessment or as a form of treatment for concussion or PCS or that did not discuss the physiology of concussion or mTBI. For example, articles on moderate or severe TBI or that focused on vestibular or physical therapy modalities were excluded. For the animal studies, we excluded those that did not focus on concussion/mTBI (eg, seizures and moderate to severe TBI effects) or were review articles.

The Physiology of Concussion

Concussion has been described as a metabolic [2], physiological [12], and microstructural [13] injury to the brain. The metabolic cascade of events immediately after TBI involves excitatory neurotransmitter release, abnormal ion fluxes, increased glucose metabolism, lactic acid accumulation, and inflammation. The macrophysiological insult involves the autonomic nervous system (ANS) and its control of both CBF and cardiac rhythm. The primary ANS control center, located in the brainstem, may be damaged in concussion, particularly if a rotational force was applied to the upper cervical spine [14]. This mechanism was confirmed in a recent diffusion tensor imaging study showing changes in brainstem white matter neurons after concussion [15]. Animal and human data suggest that this physiological dysregulation typically resolves, assuming no recurrent insult, within days to weeks after the injury is sustained [2,16]. However, there is evidence of reduced resting CBF for up to a month after concussion both in adolescents [17] and in college football players [18]. Evidence has shown that a vulnerable period of brain metabolic imbalance occurs after concussion, the resolution of which does not necessarily coincide with resolution of clinical symptoms. Measuring N-acetylaspartate using proton magnetic resonance spectroscopy, Vagnozzi et al [19] showed that metabolic dysfunction can last up to 30 days after sport-related concussion (SRC) and up to 45 days in persons sustaining a second injury before resolution of the first injury. Thus, a second concussive event prior to metabolic resolution of the first concussion can significantly delay recovery.

Post-TBI autonomic dysfunction has been proposed as a possible cause for prolonged symptoms of PCS [20]. In studies of moderate to severe TBI, altered ANS regulation is believed to be due to changes in the autonomic centers in the brain and/or an uncoupling of the connections between the central ANS, the arterial baroreceptors, and the heart [21], is proportional to TBI severity, and improves with TBI recovery [21]. In concussion/mTBI, autonomic dysfunction has been shown to affect cardiac function during exercise. For example, concussed athletes have altered heart rate variability during exercise [22] that is interpreted to reflect altered balance of the sympathetic and parasympathetic input from the ANS to the heart. This interpretation was supported by a study showing elevated heart rates (HRs) during steady-state exercise in concussed patients versus control subjects, suggesting excessive activity of the sympathetic branch of the ANS [23]. Newer evidence, however, suggests that concussed patients may not have the ability to switch appropriately from one branch of the ANS to the other (ie, from the parasympathetic to the sympathetic) at the appropriate time. Hilz et al [24] showed orthostatic intolerance in concussed subjects when moving from supine to standing (ie, blood pressure did not appropriately rise upon standing), consistent with concussed subjects not withdrawing parasympathetic influence or augmenting sympathetic modulation at the right time. In a recent prospective study of adolescents 13-18 years of age who had PCS, 70% of patients had abnormal tilt table results [25].

The physiological effects of concussion during exercise are especially important for athletes and soldiers. Athletes with PCS have been shown to have exercise intolerance, as well as altered control of CBF [26]. The CBF response during progressive exercise appears to be opposite to the reduced CBF measured at rest. CBF in persons with concussion increased out of proportion to exercise intensity compared with when they were

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