

Use of Graded Exercise Testing in Concussion and Return-to-Activity Management

John J. Leddy, MD, FACSM FACP¹ and Barry Willer, PhD²

Abstract

Concussion is a physiologic brain injury that produces systemic and cognitive symptoms. The metabolic and physiologic changes of concussion result in altered autonomic function and control of cerebral blood flow. Evaluation and treatment approaches based upon the physiology of concussion may therefore add a new dimension to concussion care. In this article, we discuss the use of a standard treadmill test, the Buffalo Concussion Treadmill Test (BCTT), in acute concussion and in postconcussion syndrome (PCS). The BCTT has been shown to diagnose physiologic dysfunction in concussion safely and reliably, differentiate it from other diagnoses (*e.g.*, cervical injury), and quantify the clinical severity and exercise capacity of concussed patients. It is used in PCS to establish a safe aerobic exercise treatment program to help speed recovery and return to activity. The use of a provocative exercise test is consistent with world expert consensus opinion on establishing physiologic recovery from concussion.

Introduction

Expert consensus holds that the best treatment during the immediate and early recovery period after concussion is rest from physical and cognitive exertion (41). The concept that rest is best is supported partially by animal and human evidence that excessive activity soon after concussion prolongs recovery (22,36). There is also some evidence of a vulnerable period early after concussion during which the brain is susceptible to repeat injury and/or worsening symptoms with cognitive or physical stress (19). The concept of using rest for recovery from the acute effects of concussion also has been extended generally to apply to those with postconcussion syndrome (PCS), *i.e.*, the persistence of symptoms beyond several weeks or months (25). The efficacy of rest in all phases of concussion recovery recently has been challenged (44). A study by Majerske *et al.* (36) demonstrated that cognitive recovery is best when the

patient is involved in some limited exercise and cognitive activity after concussion but is worse when the activity is either too great or too little.

Concussion has been thought traditionally to represent primarily a disturbance of cognition, and there is a considerable body of research describing and promoting cognitive testing as the optimal approach to establish the degree of functional (read: cognitive) recovery. More recently, concussion is being described as a physiological insult to the brain (32). The metabolic and physiologic changes that accompany concussion result, among other things, in altered autonomic function and control of cerebral blood flow (CBF)

(16,26). As such, evaluation and treatment approaches that are based upon the physiology of concussion may add a new dimension to concussion care. The purpose of this article is to review the use of exercise testing to evaluate physiologic recovery from the acute effects of concussion and to review the theory and evidence behind using individualized aerobic exercise treatment in the return-to-activity (RTA) management of those with concussion and PCS.

Definitions of Concussion and PCS

Concussion is a transient disturbance of neurologic function resulting from traumatic forces imparted to the brain (41). While sport concussion may be differentiated from non-sport-related mild traumatic brain injury (TBI) based upon mechanism of injury (41), the symptoms reported are the same. While there continues to be debate about the finer aspects of concussion diagnosis, there appears to be general consensus on the key elements and a host of measures available to assess symptoms (35), cognitive impairments (8), and balance (23). Most patients recover from the acute effects of concussion within days to weeks, but some take longer, up to several months or more (37). Those that take longer to recover are said to have PCS.

The definition of PCS is much less specific than that of acute concussion. The symptom checklists still apply, but in many instances patients believed to have PCS have no more

¹Department of Orthopaedics, University at Buffalo, Buffalo, NY; and
²Department of Psychiatry, University at Buffalo, Buffalo, NY

Address for correspondence: John J. Leddy, MD, FACSM FACP, University Sports Medicine, 160 Farber Hall - SUNY, Buffalo, NY 14214; E-mail: leddy@buffalo.edu; bswiller@buffalo.edu.

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symptoms than those who have never been concussed (37). Neuropsychological testing of those with PCS does not appear to have much value for confirming the diagnosis (9). The timeframe for recovery is influenced by factors such as athlete status (31), age (34), sex (42), and history of prior concussions (24). Research so far, however, has been unsuccessful at identifying factors that consistently predict PCS, largely because PCS is not one syndrome but is a heterogeneous disorder without reliable diagnostic criteria.

The diagnosis of PCS among nonathletes generally includes persistent symptoms for at least 3 months. Athletes (defined as those individuals competing in sport who sustain a concussion during sport) have delayed recovery when symptoms persist for a time frame longer than normally would be expected for that athlete, *i.e.*, from 3 to 6 wk (25). For example, younger adolescent and children athletes may require 4 to 6 wk to recover from the acute effects of concussion (38) whereas collegiate and professional athletes are outside the typical recovery period after 3 wk or more of persistent symptoms (25,48). These time-to-diagnosis differences and other symptom-based criteria are arbitrary, so one of the objectives of this article is to provide a more scientific definition of PCS based on some of the physiologic changes that are precipitated by concussion.

Pathophysiology of Concussion and PCS

It is intellectually useful (although perhaps not necessarily accurate) to try to differentiate the pathophysiology and clinical presentation of the acute effects of concussion from those of PCS. Linear, rotational, and blast-delivered forces to the brain induce rapid dynamic changes in neurotransmitters, intracellular and extracellular ions, glucose metabolism, and CBF (19). Recent evidence from studies using diffusion tensor imaging shows that this traumatic metabolic cascade also may be accompanied by microstructural injury to neurons, especially in the white matter of the brain (7). The acute pathophysiology of concussion produces a constellation of evolving signs and symptoms that reflect cognitive, emotional, and somatic dysfunction. Animal and human data suggest that this typically resolves, assuming no recurrent insult, within days to weeks after the injury (19,40). The etiology of the persistent symptoms in PCS patients is, however, controversial because until recently, the lack of findings on standard neuroimaging has led some to conclude that PCS, rather than being a direct consequence of brain injury, represents either an unmasking of a subclinical psychological illness, a reactive depression, a form of post traumatic stress disorder, a consequence of pain, or a form of malingering (25).

Recent physiological and advanced neuroimaging studies reveal, however, that some PCS patients have objective evidence of brain dysfunction that may explain their symptoms and limitations. For example, concussed athletes with prolonged depressive symptoms showed reduced functional magnetic resonance imaging (fMRI) activation in the dorsolateral prefrontal cortex and striatum and attenuated activation in medial frontal and temporal regions accompanied by gray matter loss in these areas (11). Some PCS patients have persistent abnormalities of brain blood flow

on single-photon emission computerized tomography scan (1), neurochemical imbalances (*e.g.*, serum S100B) (45), and electrophysiological indices of impairment (5). Postural instability is much more likely to be present when the other signs and symptoms are the result of organic-based PCS (23). In magnetic resonance spectroscopy studies, athletes who reported being symptom free at 3 to 15 d did not demonstrate complete metabolic recovery until a mean of 30 d postinjury, and mitochondrial metabolism took an additional 15 d to recover if a second concussion occurred before full metabolic recovery after a first concussion (47). There is fMRI evidence of abnormal CBF volume and distribution in humans both acutely after concussion (39) and in those with PCS (10) that may explain why concussed patients become fatigued easily with cognitive activity.

Concussion-induced mechanical changes coupled with the neurometabolic alterations described above can affect functional cerebral circulation (19). This, in conjunction with post-TBI autonomic dysfunction, has been proposed as a possible etiology for prolonged symptoms of PCS (32). Cerebral autoregulation — the capacity to maintain CBF at appropriate levels during changes in systemic blood pressure (BP) — and CBF itself are disturbed after concussion (26), which may explain why symptoms reappear or worsen with excessive physical exertion or other stressors. This appears to be a particularly important issue in adolescents, where abnormal CBF has been reported up to 4 wk after injury despite reported resolution of resting symptoms (38). Abnormal regulation of CBF may be due to altered autonomic nervous system (ANS) function and/or altered carbon dioxide (CO₂) regulation after concussion. CO₂ tension in the blood is the primary regulator of CBF (32). Concussed athletes have altered ANS balance (16), which is reflected by higher heart rates (HR) during steady-state exercise versus controls (17). The primary ANS control center, located in the brainstem, may be damaged in concussion, particularly if there is a rotational force applied to the upper cervical spine (18). Altered autonomic regulation after TBI 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. It is proportional to TBI severity and improves during TBI recovery (20).

Concussion and Exercise

Emerging data suggest that exercise improves brain function via favorable effects on brain neuroplasticity (2) as early as after 6 to 8 wk of exercise (46). The rapidity of the effect of exercise on the brain suggests that the mechanism may not involve exercise influence on cerebrovascular disease risk but rather improved neuronal function. Aerobic exercise has been shown to improve significantly fMRI cortical connectivity and activation (12). Moderate aerobic exercise (60% of maximum HR performed for 150 min·wk⁻¹) is cognitively protective (28) and is associated with greater levels of brain-derived neurotrophic factor (BDNF), which is involved in neuron repair after injury, as well as greater hippocampal volume and improved spatial memory (14). Another salutary effect of regular exercise is improved regulation of CBF (3).

It is reasonable to infer that, if introduced at the right time, exercise might improve neuronal function after TBI. Experimental animal data show that premature voluntary exercise within the first week after concussion delays recovery and is associated with impaired cognitive memory task performance by interfering with the postconcussion rise of neuroplasticity molecules including BDNF (21). Aerobic exercise performed 14 to 21 d after concussion, however, upregulates BDNF in association with improved cognitive performance (22). Nonexperimental human data support that too much activity too soon after concussion is detrimental to recovery, but that too little activity is also detrimental (36). Individuals with TBI who exercise are less depressed and report better health status when compared with those who do not exercise (49). Thus exercise treatment after concussion may be beneficial if administered at the appropriate time and as long as the exercise is of appropriate type (*i.e.*, aerobic), intensity, and duration.

Physical deconditioning of the cardiovascular system due to prolonged rest is common in TBI (48). Deconditioning has been associated with reduced CBF (50) whereas exercise training has beneficial effects on CBF control (3), which may relate to restoration of autonomic balance and/or sensitization of the autoregulatory system to gradual increases in systemic BP with controlled exercise (32). With respect to acute concussion, there is no evidence that complete rest beyond 3 d in adults is beneficial, whereas gradual reintroduction of activity appears to be (44). We have shown recently that it is safe for adult PCS patients to exercise up to 74% of maximum predicted capacity (27), which provides an evidence base for stage 2 (light aerobic exercise) of the Zurich Conference Guidelines' graduated return to play (RTP) protocol (41). We have shown also that aerobic exercise treatment improves symptoms and outcome in PCS subjects in association with improved fitness and autonomic function (*i.e.*, better HR and BP control) during exercise (31). The precise mechanisms for the effect, however, have yet to be elucidated.

The Buffalo Concussion Treadmill Test

We have developed a standard treadmill test, the Buffalo Concussion Treadmill Test (BCTT), that is the only functional test thus far shown to diagnose safely (31) and reliably (29) physiologic dysfunction in concussion, differentiate it from other diagnoses (*e.g.*, cervical injury, depression, migraines) (6), and quantify the clinical severity and exercise capacity of concussed patients (31). The BCTT is based upon the Balke cardiac treadmill test, which requires a very gradual increase in workload that has been shown to be safe in

Table 1.

Absolute and relative contraindications to performing the Buffalo Concussion Treadmill Test.

<i>Absolute Contraindications to Performing the BCTT</i>	
History	Unwilling to exercise. Increased risk for cardiopulmonary disease as defined by the American College of Sports Medicine. ^a
Physical examination	Focal neurologic deficit. Significant balance deficit, visual deficit, or orthopedic injury that would represent a significant risk for walking/running on a treadmill.
<i>Relative Contraindications to Performing the BCTT</i>	
History	Beta-blocker use. Major depression (may not comply with directions or prescription). Does not understand English.
Physical examination	Minor balance deficit, visual deficit, or orthopedic injury that increases risk for walking/running on a treadmill. SBP >140 mm Hg or DBP > 90 mm Hg. Obesity: body mass index $\geq 30 \text{ kg}\cdot\text{m}^{-2}$.

^a Individuals with known cardiovascular, pulmonary, or metabolic disease; signs and symptoms suggestive of cardiovascular or pulmonary disease; or individuals aged ≥ 45 years who have more than one risk factor to include: 1) family history of myocardial infarction, coronary revascularization, or sudden death before 55 yr of age; 2) cigarette smoking; 3) hypertension; 4) hypercholesterolemia; 5) impaired fasting glucose; or 6) obesity (body mass index $\geq 30 \text{ kg}\cdot\text{m}^{-2}$).

SBP, systolic blood pressure; DBP, diastolic blood pressure.

patients with cardiac and orthopedic problems. The HR and BP recorded at the threshold of symptom exacerbation become the basis for the individualized exercise prescription for patients with PCS. The contraindications to performing the BCTT are those that typically would contraindicate the performance of a cardiac stress test. The absolute and relative contraindications to performing the BCTT are presented in Table 1.

BCTT protocol (31)

The test is a modification of the cardiac Balke protocol. The starting speed is 3.6 mph at 0% incline but can be altered

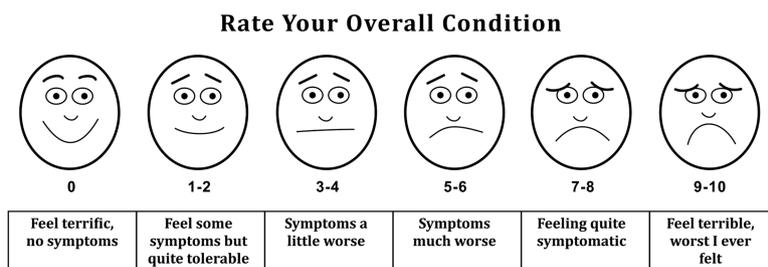


Figure 1: Visual Analog Scale for assessment of overall symptom level before and during the Buffalo Concussion Treadmill Test.

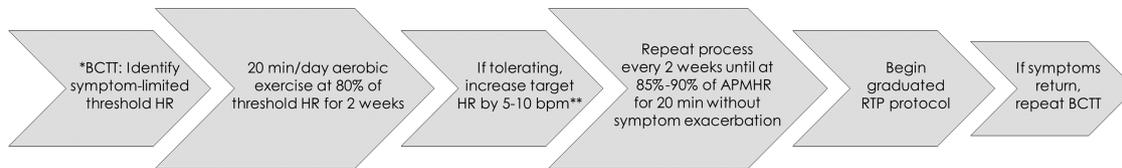


Figure 2: Use of the BCTT and exercise prescription for RTA in physiologic PCD. APMHR, age-predicted maximum HR. *After 3 wk of symptoms. **5 bpm for nonathletes; 10 bpm for athletes. To obtain a more precise target HR, consider repeating the BCTT every 2 wk.

if needed (increase speed a little to comfort for taller or athletic persons and reduce the speed for shorter or sedentary persons). During the first minute, the patient walks at 0% incline. The incline is increased by 1% at minute 2 and by 1% each minute thereafter while maintaining the same speed until the maximum incline is reached or the patient cannot continue. Rating of perceived exertion (RPE, Borg scale) and symptoms are assessed every minute. HR (by HR monitor) and BP (by automated cuff if available; if not, HR alone is sufficient) are measured every 2 min, and the test is stopped at a significant exacerbation of symptoms (defined as ≥ 3 points from that day's pretreadmill test resting overall symptom score on a 1- to 10-point visual analog scale (VAS), Fig. 1) or at exhaustion (RPE of 19 to 20). If the patient reaches maximum incline and can still continue (not yet at RPE of 19 to 20 or exacerbation of symptoms), the speed is increased by 0.4 mph for each subsequent minute until stopping criteria are fulfilled. The test is deferred in those with significant pretest resting symptoms (*i.e.*, ≥ 7 on the pretest VAS).

It is more accurate to classify patients experiencing prolonged symptoms after concussion as having one of several "postconcussion disorders" (PCD) rather than a single "PCS" because there is more than one cause of prolonged symptoms after concussion (6). The patient's performance and symptom pattern during the BCTT, combined with a pretest physical examination, can help with the differential diagnosis of PCD (Table 2). Concussion symptoms, for example, typically are exacerbated by exercise, whereas exercise usually rapidly improves symptoms of depression (13). If patients can exercise to exhaustion without reproduction or exacerbation of headache or other concussion symptoms, and they demonstrate a normal physiological response to exercise, then we conclude that the symptoms are not due to the physiologic concussion but to another problem, most commonly, a cervical injury, vestibular/ocular dysfunction, or a posttraumatic headache syndrome such as migraine (6). A careful physical examination of the cervical spine and a neurologic examination focusing on the vestibular system and oculomotor responses (Table 2) can help identify sources other than concussion that produce similar symptoms such as dizziness, headache, trouble concentrating, and blurred vision (33). In general, an isolated posttraumatic vestibular injury presents with symptoms of true positional vertigo (*i.e.*, a sensation of motion and nausea precipitated by head motion) with a positive Dix-Hallpike maneuver (sustained nystagmus and vertigo with sudden head rotation) but typically without other associated postconcussion symptoms. Vestibular symptoms caused primarily by concussion/PCS, while they may be exacerbated temporarily by head position changes, are typically not

as position dependent, do not demonstrate nystagmus with the Dix-Hallpike maneuver, are almost always present at rest (with or without head motion), and are associated usually with multiple other PCS complaints such as lightheadedness and cognitive problems. Furthermore it must be recognized that exercise itself can produce symptoms such as fatigue, headache, and dizziness as the patient approaches voluntary exhaustion. Exercise symptoms, however, can be differentiated from those of physiologic concussion since they typically occur at peak exertion near exhaustion or immediately following intense exercise, whereas concussed patients develop symptoms earlier on in exercise that prevents them from continuing (31). The other common response observed during exercise is a cervical headache, which often improves as the muscles warm up only to return at the end of the test when patients may be straining to finish. The key differentiating point is that those with a cervicogenic headache or dizziness are able to exercise to exhaustion, despite some symptoms, whereas patients who have not recovered from the physiologic disturbance of concussion stop at a submaximal level because of significant symptom exacerbation. It is important to realize that many patients with prolonged symptoms after concussion no longer have concussion as the source of their symptoms; rather, they have symptoms from other sources, most commonly a cervical injury, a vestibular injury, or a posttraumatic migraine (6), which can benefit from therapy specifically tailored to these etiologies (4). Thus the BCTT combined with a careful physical examination can help the practitioner narrow the differential diagnosis of persistent postconcussion symptoms and so direct therapy to the specific cause, enhancing the RTA process.

BCTT versus Computerized Neurocognitive Testing in RTP

Since the BCTT has good reliability for identifying patients with symptom exacerbation from concussion, it can help to establish physiologic recovery from concussion and readiness for RTA (29). In a sample of 117 high school athletes, the BCTT, when used in combination with the Zurich Guidelines' recommendations for graduated RTP, was 100% successful for returning concussed athletes to sport (Darling *et al.*, accepted for publication). All athletes exercised to exhaustion without exacerbation of concussion symptoms on the BCTT, and all returned to sport without recurrent symptoms. Meanwhile almost half (48%) of the athletes had one or more computerized neuropsychological (cNP) subtest or composite scores (on ANAM or ImPACT) "below average" on the same day that they successfully completed the BCTT. Thus the ability of concussed athletes to exercise to exhaustion on the BCTT without

Table 2.
Differential diagnosis of PCD using the BCTT and the physical examination.

Diagnosis	Physiologic PCD	Cervicogenic PCD	Migraine PCD	Affective PCD	Vestibular/Ocular PCD
BCTT response	Distinct submaximal symptom-limited threshold characterized by complaints of sudden increase in lightheadedness, headache, head pressure, or "fullness" of the head.	No distinct symptom-limited threshold. Able to exercise to exhaustion. Posterior headache that improves early in exercise but often returns near exhaustion.	BCTT not performed if migraine present. If migraine not present, there is no distinct symptom-limited threshold. Able to exercise to exhaustion.	No distinct symptom-limited threshold. Able to exercise to exhaustion. Mood usually improves with exercise testing.	No distinct symptom-limited threshold. Able to exercise to exhaustion. Symptoms are usually visual (blurred vision, difficulty with focusing) or mild lightheadedness. Vertigo typically is not reported during the test.
Physical exam	May have orthostatic drop in BP and or rise in HR.	Cervical muscle tenderness and/or spasm, reduced motion, altered cervical proprioception, suboccipital tenderness	Exam usually normal when not symptomatic. May have photosensitivity.	May have flat or depressed affect.	Discomfort (and sometimes nystagmus) with ocular smooth pursuits and saccades, abnormal ocular convergence (>6 cm), abnormal VOR, ^a positive Romberg and abnormal tandem gait.

^a VOR, vestibulo-ocular reflex; BCTT, Buffalo Concussion Treadmill Test; PCD, post concussion disorders.

symptom exacerbation better predicted readiness to begin the RTP process than did same-day cNP test performance. cNP test performance did not relate to RTP and was not associated statistically with symptoms reported upon return to school. These data support expert consensus opinion that the ability of concussed athletes to perform provocative exercise without symptom exacerbation establishes readiness to RTP (41).

Exercise Treatment of Concussion and PCS

The primary forms of concussion and PCS treatment traditionally have included rest, education, coping techniques, support and reassurance, neurocognitive rehabilitation, and antidepressants (48). There is some evidence that education, coping techniques, and neurocognitive rehabilitation have modest benefit in PCS patients (33). With respect to acute concussion, while it is accepted generally that children and adolescents require more cognitive and physical rest in the beginning to avoid delaying recovery, there is no evidence that complete rest beyond 3 d in adults is beneficial, whereas gradual reintroduction of activity appears to be most effective (44). Prolonged rest, especially in athletes, can lead to physical deconditioning and secondary symptoms such as fatigue and reactive depression (48).

The Zurich Consensus Statement recognizes the important physiologic component of testing and recovery after concussion. It advises that when asymptomatic at rest, concussed patients should progress stepwise from light aerobic activity such as walking or stationary cycling up to sport or work-specific activities (41). Recurrence of symptoms mandates return to the previously tolerated level of activity before advancing again. We have applied this principle to those with persistent symptoms. Our non-randomized pilot study showed that individualized aerobic exercise treatment improved symptoms in PCS subjects in association with improved fitness and autonomic function (*i.e.*, better HR and BP control) during exercise (31) and, when compared with a period of no intervention, safely sped rate of recovery and restored function (sport and work) (31). A similar rehabilitation program has been established for children with PCS after sport-related concussion that combines gradual, closely monitored physical conditioning, general coordination exercises, visualization, education, and motivation activities (15). The efficacy of exercise treatment in patients with persistent concussion symptoms awaits confirmation in larger randomized trials, and the mechanisms for the effect of exercise on concussion have yet to be elucidated, but in a recent small controlled study in PCS we showed that exercise treatment restored normal local CBF regulation, as indicated by fMRI activation, versus a placebo stretching intervention in association with improved aerobic capacity and resolution of symptoms (30). Some concussion symptoms may therefore be related to abnormal local CBF regulation that is amenable to individualized aerobic exercise treatment.

RTP and RTA

Symptom reports and, despite its widespread use, cNP testing may not be the most reliable indicators of concussion recovery (43). The following is an illustration of how the BCTT can be used to gauge recovery after the

symptoms seen acutely after concussion are reported to have resolved at rest. Using the BCTT, the athlete is able to exercise to exhaustion without symptom exacerbation. You conclude that s/he is recovered physiologically and can begin the graduated RTP process safely. Conversely if the athlete developed symptoms that stopped the test before peak exertion, you have objective information that s/he is not physiologically ready and will need more recovery time. The most commonly reported symptoms indicating that the concussion is not resolved are worsening headache, dizziness (*i.e.*, lightheadedness), and/or a sensation that the head feels “full.” A comparison of the HR at the point of symptom exacerbation to the athlete’s theoretical maximum HR gives you a good indication of how close the athlete is to full physiologic recovery. If close to full recovery, the test can be repeated in a few days to a week. The test can be performed in a physician’s office, an athletic training or physical therapy facility, hospital clinic, or a health club, provided that the staff has been trained in treadmill test administration and that there is medical supervision in proximity.

With respect to RTA in patients with physiologic PCD, the BCTT can be performed safely in those who remain symptomatic for more than 3 wk (Fig. 2). If a submaximal symptom exacerbation threshold is identified, patients are given a prescription to perform aerobic exercise (on a stationary cycle, treadmill or elliptical) for 20 min per day at a subthreshold intensity, *i.e.*, at 80% of the threshold HR achieved on the BCTT, once per day for 5 to 6 d·wk⁻¹ using an HR monitor. They are required to have someone present during exercise for safety monitoring and should terminate exercise at the first sign of symptom exacerbation or after 20 min, whichever comes first. The BCTT can be repeated every 2 to 3 wk to establish a new symptom-limited threshold HR until symptoms are no longer exacerbated on the treadmill (Fig. 2). A more reasonable and cost-effective approach that avoids repeated testing, however, is to establish the threshold HR on the initial test and increase the exercise HR target by 5 to 10 bpm every 2 wk (via phone call or email), provided the patient is responding favorably (6). More fit patients and athletes generally respond faster (31) and can increase by 10 bpm every 2 wk, whereas nonathletes typically respond better to 5 bpm increments every 2 to 3 wk. Rate of exercise intensity progression varies, and some patients may have to stay at a particular HR for more than 2 wk, which is fine as the idea is to give patients specific goals to achieve without focusing on how fast it takes to realize full physiologic recovery. Physiologic resolution of PCD is defined as the ability to exercise to voluntary exhaustion at 85% to 90% of age-predicted maximum HR for 20 min without exacerbation of symptoms (31). Patients can then begin the Zurich graduated RTP program. Exercise testing should be considered only for patients without orthopedic or vestibular problems that increase the risk of falling off the treadmill and only in those patients who are at low risk for cardiac disease (31). In those patients who have a nonphysiologic cause of persistent symptoms (*e.g.*, cervicogenic PCD or vestibular PCD), or a combination of disorders (patients with physiologic PCD also can have a neck injury), we have found that including regular aerobic exercise at a subthreshold level (80% of

the maximum HR achieved on the BCTT) along with specific treatment for the nonphysiologic disorder is not only not detrimental but in fact appears to enhance recovery (6).

Conclusion

Growing literature suggests that concussion is a physiologic brain injury that produces systemic symptoms and cognitive dysfunction. Most patients who rest acutely recover from concussion within days to weeks but an important minority does not. Treadmill testing as a method to establish physiologic recovery from concussion makes sense since it better replicates what athletes and active persons (such as soldiers) do. For those with PCD, rest beyond 3 wk appears to be detrimental to recovery whereas regular aerobic exercise appears to have beneficial effects on symptoms and cognition, perhaps because aerobic exercise promotes neuroplasticity and improves control of CBF. Treadmill testing in those with PCD can help with the differential diagnosis of persistent symptoms to guide appropriate therapy. Treadmill test performance can establish the degree of physiologic recovery and give patients information on their prognosis. The symptom threshold HR can be used to prescribe an *individualized* subthreshold progressive aerobic exercise program that can improve symptoms safely, speed RTA, and restore function in many patients with PCD. Individualized exercise is a nonpharmaceutical intervention that challenges the current paradigm of prolonged rest, has minimal adverse effects, can be implemented with standard equipment, and could be used at many physician offices and health facilities, including military facilities and in the field, with relative ease. Further study of exercise testing and treatment in concussion and PCD should include randomized trials to evaluate its potential for safely and more rapidly returning acutely concussed patients and those with PCD to activity and to elucidate the underlying mechanisms of the beneficial effects of exercise evaluation and treatment in those experiencing concussion and PCD.

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