Sport and exercise medicine

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Abstract

Sport and exercise medicine is now an established specialty within the UK. As with many new ventures, advances in the classification of injury have improved the categorization of intervention and the study of its effects. Molecular understanding of injury in relation to skeletal muscle, bone and connective tissue has advanced in the past decade, allowing adaptation of the microscopic observation to direct macroscopic intervention. Conditioning of both organ systems and muscle and visceral strength is essential for health and to avoid musculoskeletal injury. Age-grouped physical activity guidelines are now available for the UK population. The use of physical activity quantified by cardiorespiratory fitness is a resource whose role is still to be defined within the NHS.

Keywords Compartment syndrome; concussion; exercise; muscle injury; NSAID; rehabilitation; sport medicine; tendinopathy

The origins of sport and exercise medicine

In 2005, sport and exercise medicine was introduced as a specialty within the NHS to redress the 'physical inactivity epidemic', tackle the prevention and treatment of disease resulting from an increasingly sedentary lifestyle, and address the problems faced by the NHS due to musculoskeletal disorders and consequent sickness and absence from the workplace.[1] Since the previous version of this article, the United Kingdom has seen the unparalleled success of the London 2012 Olympic and Paralympic Games, measured not only by the overall medal successes of Team GB and Paralympics GB,[2] but also financially in terms of gross domestic product (GDP)[3] and in relation to an increase in national moderate-intensity sport participation.[4]

The discipline, now established but still developing its role within the NHS, is involved with the diagnosis and treatment of musculoskeletal injuries, and the use of exercise as an intervention and an adjunct in the treatment of all morbidity. So what are the current issues being addressed by sport and exercise physicians across the country?

Therapeutic approaches to inflammation

Tissue damage of skeletal muscle, bone and connective tissue results in initiation of inflammatory responses potentiated by the arachidonic acid (ArA) pathway, mediated and regulated by prostaglandin release (Figure 1). These responses help to protect the organism from further damage, but can also cause damage if the inflammatory response is hyperactive. Non-steroidal anti-inflammatory drugs (NSAIDs) act upon the cyclooxygenase and lipoxygenase enzymes within the ArA pathway, modulating the response and reducing the production of prostaglandins,[5] thereby reducing pain and swelling. Current research has elucidated the positive and negative roles of these responses for appropriate tissue healing. Within adult muscle tissue, satellite cells are essential for muscle repair within damaged tissue or to replenish the cell pool within the muscle, and use of NSAIDs inhibits this function.[6]

Expression of cyclooxygenase-2 (COX-2) is mediated by lipopolysaccharide and tumour necrosis factor-α and is inhibited by COX-2 inhibitors; it allows formation of cartilagenous callus at healing fracture sites through mesenchymal cell recruitment of chondrocytes, which secrete the angiogenic and osteoclastogenic factors necessary for endochondral ossification. Within tendon–bone interfaces, animal models have provided evidence of inconsistent regrowth of fibrocartilage zones at the enthesis when NSAIDs are used during the healing process.[5] Physicians need to be aware of these factors, as well as patient demographics and health status, if they are to prescribe NSAIDs judiciously to optimize recovery and avoid the harmful effects of treatment. Particular caution is necessary when contemplating extended courses of treatment.

Muscle injury classification

The historical classification systems within sport and exercise medicine resulted in highly variable and inconsistent categorization of injury by medical practitioners, and unclear diagnosis.

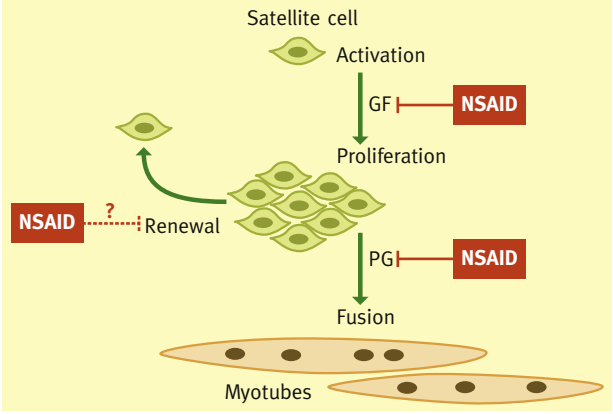

Figure 1 Influence of non-steroidal anti-inflammatory drugs (NSAIDs) on the cycle of satellite cell activity. Satellite cells are activated to proliferate and either undergo fusion or return to quiescence to renew the satellite cell pool. Many growth factors (GF) have been shown to be capable of activating satellite cells following an appropriate stimulus, such as exercise, and are potential targets of NSAID action. The synthesis of prostaglandins (PG), which are important for fusion of myoblasts, is inhibited by NSAIDs and thus represents the main target of NSAID action in skeletal muscle. How NSAIDs could affect satellite cell renewal has not been investigated. (From Mackey A. Does an NSAID a day keep satellite cells at bay? *J Appl Physiol* 2013; **115**: 900–8.)

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Classification of acute muscle disorders and injuries

A. Indirect muscle disorder/injury	Functional muscle disorder	Type 1: overexertion-related muscle disorder	Type 1A: fatigue-induced muscle disorder
			Type 1B: delayed-onset muscle soreness (DOMS)
		Type 2: neuromuscular muscle disorder	Type 2A: spine-related neuromuscular muscle disorder
			Type 2B: muscle-related neuromuscular muscle disorder
	Structural muscle injury	Type 3: partial muscle tear	Type 3A: minor partial muscle tear
			Type 3B: moderate partial muscle tear
		Type 4: (sub)total tear	Subtotal or complete muscle tear
			Tendinous avulsion
B. Direct muscle injury		Contusion	
		Laceration	

Reproduced from Mueller-Wohlfahrt H-W et al. Terminology and classification of muscle injuries in sport. The Munich Consensus Statement. *Br J Sports Med* 2013; **47**: 342–50.

Table 1

Consensus panels have recently collaborated to produce comprehensive classification systems that have led to more effective communication and standardized treatment strategies. One such field is in muscle injury. Within elite football, 31% of all documented injuries are muscle 'strains'; thigh muscle injuries alone account for 10–22% of injuries occurring in athletics, rugby, basketball and American football,[7] resulting in missed participation from training and competition. A proposed four-part system defining athletic muscle injury has been developed (Table 1). Subclassifications within this system have allowed delineation of injury according to aetiology, treatment pathway and prognostic relevance. Muscle injury can thus be classified as indirect or direct, with causes of indirect injury being related to functional or structural compromises.[7]

A functional muscle disorder is now defined as an 'acute indirect muscle disorder without macroscopic evidence (on magnetic resonance or ultrasound scanning) of muscular tear'. It is often associated with a circumscribed increase of muscle tone (muscle firmness) in varying dimensions, and predisposes to tears. There are several subcategories of functional muscle disorder, which relate to its cause.

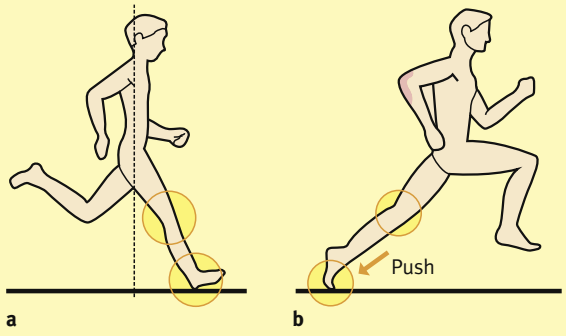
A structural muscle injury is now defined as 'any acute indirect muscle injury with macroscopic evidence (on magnetic resonance or ultrasound scanning) of muscle tear'.

This classification system acknowledges the different anatomical structures within a muscle organ that may be affected by an injury (endomysium, perimysium, epimysium and fascia) and the different roles these may play in remodelling and repair, leading to complete healing without any residual deficit within the muscle. The term 'strain' is no longer recommended.

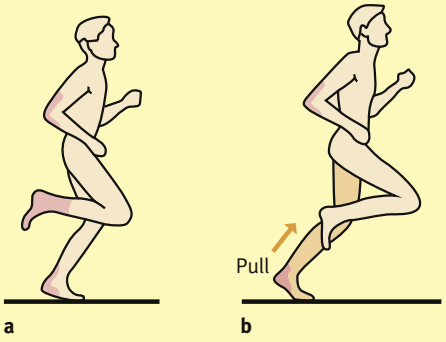
Tendinopathy

The treatment and rehabilitation of chronic tendon conditions in the past have relied upon physical exercise rehabilitation and adaptation of load, or upon reducing or obtunding pain associated with the condition itself. However, over the past decade, the previously held beliefs that tendinopathy was related to

Running gait related to anterior leg pain



1. A hindfoot-striking runner at ground contact (**a**) and toe off (**b**). Notice the extended knee and dorsiflexed ankle at ground contact (muscular activity is increased in the tibialis anterior). The ankle is plantarflexed, and the runner is pushing off from the ground, which increases the activity of the gastrocnemius/soleus muscles.



2. A forefoot-striking runner at ground contact (**a**) and toe off (**b**). The ankle is in a more neutral position at ground contact, therefore decreasing the activity of the tibialis anterior. At toe off, the foot is pulled from the ground by the hamstring muscles, and no push off occurs.

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Figure 2

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