Chapter 14

Neurologic Problems in Sport

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Neurologic conditions are common in sport and the effects of exercise on chronic neurologic conditions represent a relatively underinvestigated form of therapy. An understanding of common neurologic syndromes is therefore core knowledge for the practicing sports physician and all those involved in neurologic care need to be acquainted with the role that exercise can have in the overall management of such patients.

Headache and sport

Headache is one of the most common medical complaints and athletes are no exception to this. Few headaches fail to evoke some anxiety in the sufferer, which may in turn distort the clinical symptoms. Confronted by an athlete with exercise-related headache, the sports physician needs to be accurate in diagnosis, clear in the direction of treatment, and reassure the individual concerned (McCrory 2000).

History

The association between headache and exercise has been known since the time of Ancient Greece. In 450 BC, Hippocrates wrote: "One should be able to recognize those who have headaches from gymnastic exercises or running or walking or hunting or any other unseasonable labour or from immoderate venery" (Hippocrates 1849) By the first century AD, Areteus of Cappadocia (30–90 AD) termed headache as heterocrania and this concept was further developed in the following century by Galen of Pergammon (138–201 AD) who was the first to use the term hemicrania from which the terms "megrim" and migraine were subsequently derived (Arateus 1856).

Epidemiology of sport-related headache

The prevalence of headache in different sports is largely unknown. In a study of collegiate athletes, headaches were reported by 35% of all respondents with no gender effect evident (Williams & Nukada 1994a,b). Their headache prevalence by headache type is set out in Table 14.1. The sports noted to cause headache included running/jogging, weights/gym, aerobics, and rugby football. Post-traumatic headaches were seen almost universally in males resulting from participation in rugby football.

Community studies also note exercise as a potent trigger of migraine and other forms of headache; however, the precise epidemiology of this phenomenon is unknown (Rasmussen 1995).

Table 14.1 Headache prevalence in athletes. After Williams & Nukada 1994a,b.

Headache type	Headache prevalence (%)
Effort migraine	9
Trauma-induced migraine	6
Effort/exertion headache	60
Post-traumatic headache	22
Miscellaneous	3

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There have been anecdotal reports of migraine with aura in particular sports such as soccer (Mathews 1972) and rugby league (Gibbs 1994). In a recent study in Australian football, approximately 50% of players reported regular headaches, with 22% of all players fulfilling the International Headache Society (IHS) criteria for migraine (McCrory *et al*. 2005).

Causes of headache

Intrinsic to the understanding of the causation of headache are the intracranial pain pathways and their interconnections especially the trigemino-cervical pathway. The most important structures that register pain within the skull are the blood vessels. Neurotransmitter control in this pain pathway includes serotonin, peptides, and acetylcholine. These may provide the pharmacologic basis of drug therapy. For example, sumatriptan and methysergide both directly affect the serotonin receptor to modulate migraine. Recent advances in the field of molecular biology have suggested a causative role for other vasoactive agents in the genesis of headache which may have important treatment implications (Olesen 1988; Olesen *et al*. 1995).

Clinical approach to headache

The majority of cases do not require detailed radiologic investigations but rather a thorough history and physical examination. When seeing an athlete complaining of headache for the first time, a sports physician may follow the simple diagnostic clinical algorithm set out below:

1 Exclude possible intracranial causes on history and physical exam. If intracranial pathology is suspected then an urgent work-up is required which usually includes neuroimaging studies and/or laboratory investigations.

2 Exclude headaches associated with viral or other infective illness.

3 Exclude a drug-induced headache (see below) or headache-related to alcohol and/or substance abuse.

4 Consider an exercise (or sex-related) headache syndrome.

5 Differentiate between vascular, tension, cervicogenic, or other cause of headache.

Table 14.2 Commonly used drugs that may cause headache in athletes.

Alcohol	NSAIDs
Anabolic steroids	Nicotine
Analgesics	Nitrazepam
Antibiotics	Oral contraceptives
Anti-hypertensives	Sympathomimetics
Caffeine	Theophylline
Corticosteroids	Vasodilator agents
Dipyridamole	

NSAIDs, non-steroidal anti-inflammatory drugs.

Many commonly used drugs can provoke headaches. Some of these drugs such as non-steroidal anti-inflammatory drugs (NSAIDs) are in widespread use by athletes. If not recognized, this may be the reason for treatment failure. A list of commonly used drugs that can cause headaches in athletes is set out in Table 14.2.

A headful of symptoms

As with many aspects of clinical medicine, the history is the most important component of the assessment of the athlete with headache. Many headache syndromes such as migraine can be diagnosed with a degree of confidence on history alone. The typical qualities of the headache that should be sought on history are set out in Table 14.3. Particular emphasis should be placed on recent

Table 14.3 Clinical history of headache.

Age of onset of the headaches
Frequency and duration
Time of onset of headache
Mode of onset
Site of pain and radiation
Headache quality
Associated symptoms
Precipitating factors
Aggravating and relieving factors
Previous treatments
General health
Past medical history
Family history
Social and occupational history
Drug and medication use

changes in neurologic function such as the development of focal or systemic symptoms.

In all patients presenting with headache, a full neurologic and general physical examination is required. Particular attention should be paid to the cervical spine as a potential source of headache. The examination should consist of some or all of the following components of a focused and thorough neurologic examination, depending on the presence or absence of specific symptoms in the patient's history. The main examination points should include general appearance (including skin lesions), vital signs (pulse, blood pressure, and temperature), mental status and speech, gait, balance and coordination, cranial nerve and long tract examination, visual fields, acuity and ophthalmoscopic fundus examination, and skull palpation.

Key symptoms to flag

The majority of headaches are a result of benign causes. Nevertheless, certain symptoms may indicate the presence of more serious pathology, such as a mass lesion or infective process, and require urgent neurologic assessment. These new symptoms, which should be sought by specific questioning in all cases, are set out in Table 14.4.

Exercise-related headache syndromes

As in the general population, the common headache syndromes such as migraine, episodic tension-type headache, and cervicogenic headache will occur in athletes. These are discussed briefly. Readers are referred to more general headache textbooks for a greater understanding of these syndromes (Dalessio 1987; Lance & Goadsby 1998). In addition, a group of headache syndromes unique to exercise need to be considered and are described in more detail.

Table 14.4 Key symptoms of intracranial pathology.

Sudden onset of severe headache
Headache increasing over a few days
New or unaccustomed headache
Persistently unilateral headaches
Chronic headache with localized pain
Stiff neck or other signs of meningism
Focal neurologic symptoms or signs
Atypical headache/change in the usual pattern of headache
Headaches that wake the patient during the night or early morning
Local extracranial symptoms (e.g., sinus, ear, or eye disease)
Systemic symptoms (e.g., weight loss, fever, and malaise)

MIGRAINE

Given that the prevalence of migraine is approximately 12–18% in community populations, it follows that migraine will be commonly seen in exercising athletes. Migraine is essentially an episodic headache that is usually accompanied by nausea and photophobia and may be preceded by focal neurologic symptoms. Symptoms vary considerably between individuals. Migraine represents an inherited tendency to headache with a lowered threshold of susceptibility to a variety of headache triggers such as exercise and head trauma. At present, there is no simple "cure" for migraine and an individualized management strategy needs to be developed for each patient. This may incorporate non-pharmacologic as well as pharmacologic strategies.

The accurate diagnosis of headache syndromes in sport has important treatment implications. (McCrory 1997). In elite athletes, there are specific management considerations related to the use of "banned" drugs. Many conventional headache medications (e.g., beta-blockers, caffeine, codeine-containing preparations, dextro-propoxyphene, narcotics, and opioids) are banned agents and their use, if detected, may result in severe penalties for the athlete concerned.

TENSION-TYPE HEADACHE

Tension-type headache results in a constant tight or pressing sensation which may initially be episodic and related to stress but can recur almost daily in its chronic form without regard to any obvious psychologic factors. In general, these headaches are distinguished from migraine by their milder severity and longer duration, although a precise separation may not always be possible. Treatment

is usually multifactorial and by necessity includes psychologic and physical therapy, physiologic intervention, and pharmacologic treatment.

CERVICOGENIC HEADACHE

Abnormalities of the various structures within the neck have been implicated as the cause of cervicogenic headache. These structures include the synovial joints, the intervertebral disks, ligaments, muscles, nerve roots, and the vertebral artery (Bogduk *et al*. 1985; Bogduk & Marsland 1986). Cervicogenic headache shares many of the clinical features of chronic tension-type headache. It is usually occipital in onset and may radiate to the anterior aspect of the skull and face. The headache is usually constant in nature, lasts for days to weeks, and has a definite association with movement of cervical structures. Treatment usually involves physical or manipulative therapy to the cervical spine as well as consideration of anti-inflammatory drug therapy.

BENIGN EXERTIONAL HEADACHE

Benign exertional headache (BEH) has been recognized as a separate entity for over 50 years. In 1932, Tinel first described severe but short-lasting headaches following exercise and subsequent authors have characterized a clear-cut syndrome (Diamond & Medina 1982a,b; Diamond *et al*. 1998; Powell 1982; Tinel 1932).

The criteria for BEH include:

1 The headache is specifically brought on by physical exercise;
2 It is bilateral, throbbing in nature at onset, and may develop migrainous features in those patients susceptible to migraine;
3 It lasts from 5 min to 24 h;
4 It is prevented by avoiding excessive exertion; and
5 It is not associated with any systemic or intracranial disorder.

The onset of the headache is with straining and Valsalva type maneuvres such as seen in weightlifting. Clearly, the major differential diagnosis to be considered in this situation is a subarachnoid hemorrhage which needs to be excluded by the appropriate investigations. It has been postulated that exertional headache is caused by dilatation of pain-sensitive venous sinuses at the base of the brain as a result of increased cerebral arterial pressure caused by the exertion. Studies of weightlifters demonstrate that systolic blood pressure may reach levels above 400 mmHg and diastolic pressures above 300 mmHg with maximal lifts (MacDougall *et al*. 1985).

The implication that these headaches have a vascular basis is supported by the migrainous nature of the headache and one interesting study that utilized intravenous dihydroergotamine to relieve the headache (Hazelrigg 1986). A similar type of vascular headache is described in relation to sexual activity and has been termed benign sex headache or orgasmic cephalgia. It is worth noting, however, that despite their vascular nature, no convincing association with migraine is demonstrable.

Treatment strategies include NSAIDs such as indometacin at a dosage of 25 mg t.i.d. (Diamond & Medina 1982a,b). Other pharmacologic strategies that have anecdotal support include the prophylactic use of ergotamine tartrate, methysergide, or propranolol pre-exercise. In practice, the headaches tend to recur over weeks to months and then slowly resolve although some cases may be lifelong. In the recovery period, a graduated symptom-limited weightlifting program is appropriate.

Effort headache

In clinical practice, these types of headache can be seen as migraine-type headaches triggered by aerobic exercise. Effort headaches have been reported to be the most common type of headache in athletes (Williams & Nukada 1994a,b). These differ from exertional headaches in that they are not necessarily associated with a power or straining type exercise and a variety of sports have been associated with these headaches.

The clinical features of effort headache syndrome include:

1 Onset of mild to severe headache with aerobic type exercise;
2 More frequent in hot weather;

3 Vascular type headache (i.e., throbbing);
4 Short duration of headache (4–6 h);
5 Provoking exercise may be maximal or submaximal;
6 Patient may have prodromal "migrainous" symptoms;
7 Headache tends to recur in individuals with exercise;
8 Athlete may have a past history of migraine;
9 Normal neurologic exam and investigations.

These episodes of effort migraine are not necessarily benign, with at least one case of hemispheric cerebral infarction associated with an episode of effort headache (Seelinger *et al*. 1975). Treatment strategies for effort headaches are anecdotal and include the use of indometacin or various antimigraine preparations. In the author's experience, prophylactic NSAIDs are effective if given prior to exercise, although in the headaches that occur in hot weather these drugs seem to have reduced efficacy. Graduated exercise programs have also been studied as a means of preventing such headaches with limited success (Lambert & Burnett 1985).

Conclusions

The treatment of exercise-related headaches in athletes can be potentially rewarding for the sports physician as well as the athlete. While the common headache syndromes seen in the general population must be considered, recognition of the diversity of these sport-related headache syndromes provides the basis for good clinical care.

Stroke and sport

The occurrence of stroke in sport is a rare phenomenon. Most sports participants tend to be young and unlike older age groups, where stroke represents one of the most common neurologic diseases, stroke in young people is an uncommon event. Of course, both young and older athletes, whether performing at a recreational or elite level, may develop the same stroke syndromes as non-athletes. In some cases, the athlete may have unsuspected risk factors that increase the risk of stroke during exercise, whereas in other cases the sporting activity itself may confer an intrinsic stroke mechanism. A sports physician therefore needs a background knowledge of stroke risk factors at all ages as well as specific knowledge of stroke syndromes associated with sport.

Epidemiology of stroke

Stroke is the third leading cause of death the Western world. In the USA alone, at least 500,000 people experience a new stroke each year and, of these, 150,000 die. The prevalence of stroke according to the US National Health Interview Survey is 720 per 100,000 in the white population and 910 per 100,000 in the non-white population (McCrory 1999). Community studies demonstrate that the annual incidence rate of stroke is 102 per 100,000 population. In the 15–44 year age group, the overall incidence of stroke is nine cases per 100,000 population (Stern & Wityk 1994, 1998).

Etiology of stroke in young patients

Ischemic strokes can be caused by intrinsic vascular occlusions (thrombus) or an occlusion of a vessel from material that originates elsewhere in the vascular system (embolism). An overview of published studies of ischemic stroke in young people suggests that the common causes of stroke are large artery atherosclerosis, migraine, lacunae (small vessel, deep infarctions), and cardiac embolism. Less commonly, hematologic disease and illicit drug abuse is recognized. Finally, in many patients the cause of the stroke remains undefined (van den Berg & Limburg 1993). There are a number of rarer cause of stroke in young populations that also may need to be considered (Table 14.5).

When considering stroke in sport, there are approximately 70 published case reports in the medical literature where stroke occurs in this setting (McCrory 1999). These reports demonstrate that arterial dissection is the predominant pathophysiologic mechanism of sport-related stroke occurring in approximately 80% of cases.

Pre-participation physical examination

Recognition that a variety of underlying conditions may predispose to stroke means that the team or

Table 14.5 Rare causes of stroke in the young. After van den Berg & Limburg 1993.

System	Disease
Cardiac emboli	Endocarditis
	Atrial fibrillation or flutter
	Recent myocardial infarction
	Dilated cardiomyopathy
	Intracardiac thrombus
	Valvular vegetations
	Prosthetic valve
	Mitral valve prolapse
	Atrial septal defect
Hematologic disorders	Sickle cell disease
	Hemoglobin SC disease
	Polycythemia
	Thrombocytosis
	TTP
	DIC
	Antiphospholipid antibodies
	Protein C & S, AT III deficiency
	Disorders of fibrinolysis
Hereditary diseases	Neurofibromatosis
	MELAS syndrome
	Homocysteinemia
	Sneddon's syndrome
	Williams' syndrome
Medication related	Proximal myotonic myopathy
	L-asparaginase
	i.v. immunoglobulin
	Methotrexate
	Interferon
Inflammatory diseases	Rheumatoid arthritis
	Systemic lupus erythematosus
	Scleroderma
	Polymyositis
	Polyarteritis nodosa
	Wegener's granulomatosis
	Sarcoidosis
Infectious diseases	Neuroborreliosis
	HIV infection
	Neurocysticercosis
	Herpes zoster
	Chlamydia pneumoniae
	Hepatitis C virus
	Hydatid cyst embolism
Malignant disease	Tumor emboli
	Malignant angioendotheliomatosis
	Illicit drugs
	Cocaine
	Methamphetamine
	MDMA (ecstasy)
	Ephedrine
	Phenylpropanolamine
	Methylphenidate
	Heroin
	Anabolic steroids

AT III, Anti-thrombin III; DIC, disseminated intravascular coagulation; MDMA, 3,4-methylene-dioxymethamfetamine; MELAS, mitochondrial myopathy, encephalopathy, lactoacidosis, and stroke; SC, sickle cell; TTP, thrombotic thrombocytopenic purpura.

family physician is ideally placed to perform routine pre-participation physical examination (PPPE) and look specifically for these risk factors. In sporting populations, the causes of stroke fall into a relatively small group of risk factors which should be specifically sought on the PPPE:

1 Trauma to the extracranial cerebral arterial tree;
2 Exercise-induced hypertension;
3 Pre-existing cardiac or vascular disease;
4 Environmental injuries (e.g., hyperthermia, decompression illness);
5 Drug and stimulant abuse.

A detailed history, physical examination, and the judicious use of investigations should assist the physician in determining the risk profile of the athlete being examined.

HISTORY

A history of recurrent syncope or loss of consciousness associated with exercise raises the possibility of occult cardiac disease. Approximately 15% of cases of sudden death in sport are associated with prior syncopal or pre-syncopal episodes (Maron *et al*. 1980). The traditional cardiac ischemic symptoms of chest pain or dyspnea with exercise should also be sought as well as a history of congenital heart disease or cardiac valve infection (Driscoll 1985).

The athlete should be asked about a history of headache. Migraine is extremely common, with a prevalence of 15–20% in community populations. The incidence of migrainous cerebral infarction symptoms is extremely low; however, this mechanism represents one of the causes of stroke in young people occurring in 2–18% of cases (van den Berg & Limburg 1993). Congenital intracranial aneurysms occur with an incidence of 2000 per 100,000 population and have a rate of rupture of 12 per 100,000 per year (Phillips *et al*. 1980). As many as 50% of patients describe a severe "sentinel" warning headache prior to rupture (Gillingham 1967). Differentiating these from other headache syndromes may be extremely difficult. The presence of an unexplained severe headache, particularly in patients with no headache history or with a family history of aneurysmal rupture, should prompt further investigation.

A history of drug, alcohol, and stimulant abuse must be specifically sought. A variety of medications and illicit drugs have been associated with stroke in this age group (Table 14.5). Similarly, a history of brain trauma, environmental exposure to altitude, extremes of heat, or scuba diving would raise suspicion of these mechanisms of cerebral injury.

EXAMINATION

On general appearance, a marfinoid body habitus should raise the possibility of cardiac valvular disease. The skin should be examined for lesions suggestive of an underlying vascular disease, such as Osler–Rendu–Weber syndrome. A full cardiovascular examination should be performed in all patients. Detailed guidelines for pre-participation examination in young athletes have been published elsewhere (Brukner & Khan 2006; Driscoll 1985).

INVESTIGATIONS

The role of investigations should be limited to those where the history or examination findings are suggestive of an underlying disease. If cardiac disease is suspected then chest X-ray, electrocardiogram (ECG), and echocardiogram are the usual initial screening investigations. If ischemic heart disease is suspected then an exercise ECG test or thallium scan may be performed. If an intracerebral aneurysm is suspected, then a cerebral computed tomography (CT) or magnetic resonance (MR) scan should be performed with MR angiography. This is considerably less invasive and carries less morbidity than carotid and vertebral angiography. Coagulopathic disease, bleeding diathesis, hyperviscosity syndromes, or auto-immune disease can be detected on appropriate hematologic and serologic work-up. A drug screen may be necessary in some situations.

Specific stroke syndromes in sporting populations

Stroke in athletes is commonly associated with dissection of the extracranial vessels, presumably secondary to arterial trauma either caused by neck movement or through a blow to the region. Far less commonly, other pathophysiologic mechanisms such as hyperviscosity states, hemodynamic compromise, exercise-induced hypertension, drug-induced stroke, or atherosclerotic small vessel disease has been noted.

VERTEBRAL ARTERIAL DISSECTION

Vertebral arterial dissection (VAD) is an uncommon and incompletely understood condition. The precise incidence is unknown (Hart 1988). Approximately 25 cases have been reported to occur in sport (McCrory 1999). Most patients are healthy without predisposing risk factors for stroke (Pryse-Phillips 1989). Failure to make the diagnosis and institute appropriate therapy may result in long-term neurologic sequelae or death, although spontaneous resolution of the condition does occur (Sturznegger 1994). The initiating factor for this condition is thought to be a tear in the arterial intima with subsequent formation of *in situ* thrombosis. The pathologic injury may only involve the arterial intima or may extend to involve the tunica media. More rarely, the adventitial layer is breached with bleeding outside the vessel wall. Spontaneous dissections are rare and reported cases are often related to associated disease states such as cystic medial necrosis, Behçets syndrome, rheumatoid arthritis, giant cell arteritis, osteogenesis imperfecta, and fibromuscular dysplasia (Showalter *et al*. 1997).

A great deal of discussion revolves around the relationship of cervical movement to vertebral artery compression. Anecdotally, the presence of cervical abnormalities such as zygapophyseal joint osteophytes, fractures, dislocations, and rheumatoid arthritis may potentially cause symptomatic vertebrobasilar ischemia with head turning. From a pathophysiologic standpoint, in order for vertebrobasilar ischemia to occur there must be contralateral vertebrobasilar artery hemodynamic restriction either from stenosis, atherosclerotic disease, or hypoplasia (Biousse *et al*. 1995; Mas *et al*. 1987, 1989).

Cases of vertebral artery dissection related to trauma are more commonly reported, often in the presence of major cervical spinal trauma such as vertebral fracture or subluxation (Willis *et al*. 1994).

However, most recognized cases occur following a seemingly trivial traumatic event such as sneezing, coughing, riding a roller-coaster, horse riding, visiting a hairdresser, or performing yoga exercises. Interestingly, in all of these cases the arteriographic appearance of the contralateral vertebrobasilar arteries were normal.

A wide variety of clinical neurologic deficits are found in relation to VAD, perhaps related to the variable pathologic mechanisms (thrombosis or embolism), the complexity of neural structures supplied by the posterior circulation, and the variability of the vascular architecture. The most common initial symptoms in VAD are neck pain and occipital headache which may precede the onset of neurologic symptoms from seconds to weeks. It was noted that headache symptoms in the majority of cases were ipsilateral to the vascular injury and that the pain usually radiated to the temporal region, frontal area, eye, or ear. None of the reported cases had cervical tenderness or objective restriction of neck movement although a subjective exacerbation of pain did occur with neck movement (Sturznegger 1994).

The typical course of untreated VAD is progression in a stuttering fashion over hours to days. This progression is thought to result either from propagation of the thrombus or from distal embolization. This is the rationale for the use of anticoagulation in the treatment of extracranial VAD.

From the standpoint of early recognition of this condition, the combination of unilateral neck pain or headache of abrupt onset following a history of neck trauma should lead to the suspicion of VAD even in the face of seemingly trivial trauma. The clinical suspicion of VAD should be investigated by either transcranial Doppler ultrasound, vertebral artery angiography, or magnetic resonance angiography (MRA) after neurologic opinion is sought. The history or detection of major neurologic findings such as vertigo, diplopia, ataxia, dysarthria, cranial nerve palsies, or altered mental function should be an absolute indication for urgent neurologic consultation and hospital admission. It is important to emphasize that the neurologic findings may be delayed days or weeks after the onset of neck pain and optimal management of this condition should involve early recognition, appropriate investigation, and institution of anticoagulation where indicated. A clinical algorithm for the emergency department management of VAD has been recently published (Showalter *et al*. 1997).

CAROTID ARTERIAL DISSECTION

Direct trauma to the head or neck during sport can potentially injure the carotid arteries. In addition, forcible hyperextension or lateral rotation may similarly cause arterial injury (Fields 1981a,b; Noelle & Clavier 1994). Hypertrophy of the posterior belly of the digastric muscle leading to compression of the internal carotid artery and causing transient ischemic episodes with neck rotation has been reported in a retired professional football player (Etheridge *et al*. 1984). Non-penetrating neck trauma has been reported to cause thrombosis of both the external carotid and more importantly the internal carotid artery with associated ischemic hemispheric injury. Fields (1981a,b) distinguishes four types of traumatic carotid arterial injury:

1 A blow to the head causing forceful compression of the ipsilateral carotid artery (ICA) with intimal rupture;

2 A direct blow to the anteromedial neck leading to atherosclerotic plaque rupture and thrombosis;

3 Skull fracture leading to thrombosis of the intrapetrous ICA; and

4 Blunt intra-oral trauma with ICA thrombosis.

Clinically, apart from the history of trauma and ipsilateral hemispheric stroke symptoms, tenderness of the carotid vessels, oculomotor palsies, and an ipsilateral Horner's syndrome may alert the examiner to the carotid injury. In addition to stroke episodes, trauma to the carotid arteries may induce a severe unilateral headache associated with profound autonomic symptoms. This entity is known as traumatic dysautonomic cephalgia which may be successfully treated with propranolol.

Anecdotally, about half of all carotid dissections result in stroke which is in keeping with the age profile of most cases and presumably an intact collateral circulation. Recurrence is unusual and does not typically affect the same segment or artery as the initial presentation. Most authors recommend

anticoagulation therapy for carotid dissection although there are no prospective trials demonstrating the efficacy of such treatment.

SUBCLAVIAN ARTERY STENOSIS

One case of subclavian artery stenosis has been reported in a baseball pitcher who developed an acute hemispheric stroke as a result of propagated thrombus into the carotid artery. The putative lesion was thought to be caused by compression of the subclavian artery at the level of the thoracic outlet and first rib (Fields 1981a,b). More typically, subclavian vessel disease causes acute upper limb ischemia (as may be seen with dissections of the aortic arch), subclavian vein thrombosis (reported in racket sports and in throwing athletes), and subclavian artery stenosis causing the so-called subclavian "steal" phenomenon.

MIGRAINE

Migraine is often an etiologic consideration in young stroke patients. Because there is no diagnostic marker for migraine, the clinician must rely on the patient's history. Because exercise-related headache is common to many sports, differentiating migrainous events from other causes of headache can be clinically difficult.

Migraine without aura is associated with a threefold increase in stroke in young women and a sixfold increase for migraine with aura. In addition, the risk of stroke in migrainous women is markedly increased in those using oral contraceptives (odds ratio 14) or those who are heavy smokers (odds ratio 10). Oral contraceptives have been implicated in some studies as a cause of stroke although this remains extremely controversial (World Health Organization 1996).

A conservative approach is to limit the diagnosis of migrainous stroke to patients with a history of migraine who have a stroke during a typical episode of headache and have no other identifiable cause for stroke. In some patients, angiography at the time of the event can show segmental narrowing of intracranial arteries suggestive of vasospasm. Subsequent angiography is typically normal, confirming the reversible nature of the lesion. It is important not to assign a diagnosis of migrainous stroke until after a thorough evaluation because many conditions may have a history of migraine-like headaches.

CARDIOGENIC EMBOLISM

Cardiac diseases that predispose to stroke syndromes are listed in Table 14.1. In most cases, these are extremely rare phenomena. Cardiogenic emboli leading to focal cerebral ischemia may occur during activity-induced cardiac dysrhythmias. Sinus bradyarrhythmias in athletes have been associated with atrial fibrillation and thrombus formation with secondary embolization to the brain (Abdon *et al*. 1984). Athletes who experience stroke episodes secondary to cardiogenic embolism or arrhythmias are at risk from repeated episodes unless the underlying problem can be corrected. In many cases this is not possible and long-term anticoagulation may be considered as a preventative therapy; however, this form of treatment may preclude athletes from participation in contact and collision sports.

STIMULANT AND DRUG ABUSE

Stroke can occur during the first minutes of acute intoxication with a drug, in the hours following ingestion, or weeks following intoxication (Kaku & Lowenstein 1990). Recognition of the role of drug use in the pathogenesis of stroke requires familiarity with the acute effects of commonly used recreational drugs and a high index of suspicion. The signs and symptoms of a stroke are not different in the user of recreational drugs unless the user is acutely intoxicated. The physician must question for a history of substance abuse from the patient, friends, and family. Suspicion of drug use can be confirmed through prompt urine toxicology screening.

Stroke can occur as both a direct and an indirect medical complication of the recreational use of several drugs. The direct complications related to cerebrovascular disease are either ischemic stroke, intracerebral hemorrhage, or subarachnoid hemorrhage. The drugs typically associated with these

complications include cocaine, methamphetamine, 3,4-methylene-dioxymethamfetamine (MDMA or 'ecstasy'), ephedrine, phenylpropanolamine, methylphenidate, and heroin (Bendixen 1998).

Indirect complications are related to the means of its administration or to contaminants mixed in with the drug. For example, cardioembolic stroke can be secondary to bacterial endocarditis in an intravenous drug user who employs unsterile needles. Additionally, cocaine has been reported to cause myocardial infarctions and cardiomyopathies, creating the potential for cardioembolic stroke. Similarly, several drugs intended for intravenous use are mixed with talc or cornstarch. These substances have been found to occlude arteries, leading to a stroke (Brust & Richter 1977).

Strokes have also been reported several bodybuilders who were consuming anabolic steroids (Akhter & Hyder 1994; Jaillard & Hommel 1994). The duration of anabolic use in these cases ranged from 6 weeks to 4 years. Clinical presentations included hemispheric stroke and sagittal sinus thrombosis. Stroke has also been reported in patients using the same agents for medical indications, suggesting that this risk is not wholly unexpected (Ferenchick 1990).

Drug use should be suspected in any young person who presents with a stroke. It should be remembered that drug use encompasses not only illegal substances such as cocaine, methamphetamine, and heroin, but also diet pills, over-the-counter decongestants, methylphenidate (Ritalin), and asthma medications. A detailed history should be sought from the patient, friends, and family and urine toxicology screens used where necessary.

FAT EMBOLISM

Fat embolism is a common complication of severe bone trauma, particularly to the extremities, occurring in up to 15% of such cases (Lepisto & Alho 1975). Given that long bone trauma is common in many contact and collision sports, this entity needs to be considered in this setting. Plugging of small intracerebral blood vessels by lipid particles and fibrin clots leads to brain infarction. The neurologic presentation may be delayed by hours to days after trauma. The onset of neurologic symptoms such as hemiparesis, altered mental status, seizures, and coma is usually acute. Associated hypoxia secondary to pulmonary infarction may complicate the presentation. The treatment of fat embolism is improvement of the oxygen-carrying capacity by correction of hypoxia and, if necessary, red blood cell (RBC) transfusion. Even in cases of coma, the patient may still recover completely.

INTRACRANIAL HEMORRHAGE

Systolic blood pressure increases during exercise and the peak pressure rise correlates with the intensity of the exercise, the age of the patient, and inversely with their fitness (Carlstein & Grimby 1966). Where a vascular malformation or arterial wall defect such as an aneurysm exists, then the risk of rupture is increased during exercise. Given the high peak arterial pressures that occur with sports such as weightlifting, with systolic pressures exceeding 400 mmHg and diastolic pressures above 300 mmHg, it is surprising that intracranial hemorrhage (ICH) does not occur more frequently (MacDougall *et al*. 1985). In cases of stroke, approximately 50% of patients with aneurysms and 25% of those with vascular malformation present with bleeding during physical exertion or emotional strain (Department of Health and Human Services Task Force 1992; van den Berg & Limburg 1993; van Gijn & van Dongen 1980). Alcohol intoxication has been reported as an additional risk factor for aneurysm rupture (Vijayan 1977; Vijayan & Dreyfus 1975). In addition, two cases have been reported of bleeding into occult intracerebral tumours while jogging (Welch & Levine 1990).

Conclusions

Immediate recognition of a CNS injury, institution of cardiopulmonary support where required, and immobilization of the head and neck are the cornerstones of initial management of sport-related neurologic injury. More definitive investigation and treatment of specific stroke syndromes requires an understanding of the etiology, underlying pathogenesis, and temporal profile of the various causes

of stroke in sport. The understanding of stroke risk factors in an athletic population may assist the clinician in instituting an appropriate pre-participation screening program to minimize the occurrence of such conditions.

Epilepsy and sport

Convulsive episodes when they occur in sport are rare but dramatic events. Traditionally, these episodes have been assumed to represent a form of epilepsy; however, recent studies have demonstrated that most, if not all, post-traumatic convulsive episodes seen acutely in a sporting situation are non-epileptic in nature (McCrory & Berkovic 1998).

In addition, individuals with epilepsy are encouraged to participate in sport and sports physicians need to understand the medical issues in relation to epilepsy, particularly drug therapy, in order to advise these individuals correctly. Details of epilepsy management are best sought in any of the published reference books available (Duncan *et al*. 1995; Engel & Pedley 1998).

Epidemiology and nomenclature

Epilepsy affects approximately 2% of the population. In three-quarters of these cases, the diagnosis is made before the age of 21 years. Thus, epilepsy is a relatively common condition that may affect individuals during the years of sport participation.

Epilepsy is a neurologic disorder of the brain characterized by recurrent (more than two) seizures. It has been estimated that approximately 10–30% of the population will have a seizure at some time in their lives (Sander & O'Donoghue 1997; Sander *et al*. 1990). However, neither single episodes of seizures during adolescence or adult life nor febrile convulsions in infancy constitute a diagnosis of epilepsy.

The terms "seizure," "epilepsy," "convulsion," and "fit" are often used interchangeably. For the purpose of this chapter, the term seizure will refer to an epileptic seizure and the term convulsion will be used to describe the movements during an episode without implying a specific etiology.

Pathology

A seizure usually occurs suddenly and is the result of an abnormal electrical discharge within the brain. In the vast majority of cases, the cause of the electrical disturbance in the brain is unknown. In a small percentage, either specific genetic inheritance or structural anatomic abnormalities can induce seizures. Cortical scars related to head injuries, stroke, and other intracranial injuries may also cause seizures. During the seizure there may be an initial prodromal stage ("aura"), followed rapidly by disturbances in movement and alterations in consciousness.

Epilepsy can be classified by criteria developed by the International League Against Epilepsy (ILAE 1989). This classification utilizes the electroclinical features of the seizure to make a syndromal or etiologic diagnosis, which then has important implications for management. In the broadest sense, the ILAE classification breaks down seizures into generalized or focal (depending on the origin of the seizure), and complex or partial (depending upon whether consciousness is preserved during the episode). Outside of neurologic practice, the specific epilepsy subtype may be difficult to quantify and subjects are often simply reported to have a generalized seizure. This type of seizure was previously known as "grand mal" but this term has fallen out of favor and should be avoided.

Generalized tonic–clonic seizure

In the generalized tonic–clonic seizure, the patient usually falls to the ground and goes through a "tonic" phase of muscle stiffness followed by a "clonic" phase of muscle twitches prior to resolution of the attack. After the attack the patient is usually sleepy, confused, and may have a headache. The average length of the seizure is usually no more than 30 s, although most people who have witnessed someone having a seizure feel that the attack seems to last much longer.

Convulsions that are not caused by epilepsy

In addition to the true epilepsy seizures described above, there are other situations where convulsions

may occur. These may superficially resemble epilepsy although the etiology of such syndromes is distinctly different. These have the potential to cause confusion for non-neurologists and the eyewitness history usually provides the basis of the diagnosis. The two most common situations are:

1 Concussive convulsions, where a convulsion may be a manifestation of the concussive impact. Although usually brief and limited to tonic posturing, they may occasionally result in a prolonged convulsion over several minutes. These are benign phenomena and require no specific management beyond that of the underlying concussion (McCrory & Berkovic 1998, 2000);

2 Convulsive syncope, where convulsive movements (including generalized movements, tongue biting, and incontinence) occur in the setting of a syncopal faint.

In both situations, the convulsive movements result from reflex phenomena, not epileptic discharge.

Diagnosis of epilepsy

The diagnosis of epilepsy relies primarily upon the clinical history and on the nature of the electroencephalogram (EEG) changes. The most important and useful diagnostic consideration is history from an eyewitness who has seen and can describe the attack, particularly the onset and offset of the seizure. Any patient observed to have a seizure should be referred to a neurologist for assessment.

Investigations

In most cases of a seizure, a neurologist would order an EEG as well as neuroimaging studies (usually MRI). If the EEG is performed within 24 h of a seizure, its diagnostic sensitivity is increased from 30% to 50% and this may be further improved by performing a "sleep-deprived" EEG study. MRI is the investigation of choice to image the brain in this circumstance. Specific MRI protocols are necessary to obtain diagnostic information in these cases. Where necessary, these investigations would be supplemented by blood tests to rule out other causes of seizures, such as hypoglycemia, hypernatremia or hyponatremia, and hypercalcemia.

Treatment

The role of specific treatment in patients with a single seizure or recurrent seizures (epilepsy) requires an understanding of the nature of the seizure disorder and its natural history as well as individual patient consideration. In some situations, drug treatment should begin after a single seizure. Consideration of lifestyle factors in the overall management is paramount. Specific factors that may lower seizure threshold include sleep deprivation, alcohol, and use of recreational drugs. Patients must be specifically counseled about such lifestyle issues when they begin pharmacologic therapy.

More than half of the individuals taking antiepileptic medication for idiopathic generalized epilepsy can expect to be seizure-free with minimal restriction on their lifestyle. Approximately one-third may have only an occasional seizure, which usually does not greatly limit their lifestyle. The other 20% will have seizures frequently enough to restrict their lifestyle to some extent.

The medications used in the treatment of epilepsy can cause a number of side effects, including tiredness, poor concentration, impairment of coordination, and cognitive impairment. In some cases, medication (e.g. phenytoin) toxicity may result in permanent neurologic symptoms.

Exercise prescription in epilepsy

Regular physical activity is advocated for individuals with epilepsy (Nakken *et al*. 1990). In general, people with epilepsy report better seizure control when exercising regularly. Occasionally, some individuals will have more seizures with exercise and hence every case must be treated individually. Persons with epilepsy have no higher injury rate in sport than those without epilepsy and sport participation does not affect serum drug levels (Nakken *et al*. 1990).

In a sample of over 200 patients with epilepsy in Norway, exercise patterns were similar to that of the average population (Nakken *et al*. 1990). In the majority of the patients, physical exercise had no adverse effects, and over one-third of patients claimed that regular exercise contributed to better

seizure control. In 10% of patients, exercise appeared to be a seizure precipitant and this applied particularly to those with symptomatic partial epilepsy (i.e. underlying structural brain lesion).

There are a number of important considerations when counseling the individual who has epilepsy and wishes to exercise. Patients having frequent seizures must be discouraged from activities such as scuba diving, cycling, horseback riding, or rock climbing. Sports where any impairment in split-second neuromuscular timing is dangerous (e.g. motor-racing or downhill ski racing) should also be avoided. Patients with epilepsy will not be affected adversely by indulging in contact sport provided the normal safeguards for participation are followed.

The frequency of seizures is important when considering activities such as swimming, where the potential for serious injury exists if a seizure were to occur. Generally, swimming is allowed under supervision (e.g. with a "buddy"). Swimming with a companion is a sensible rule for all swimmers, not just those with epilepsy.

The physical and psychologic well-being of the individual also requires attention. In children, particularly adolescents, participation in activities is important in establishing a good self-image and gaining peer group acceptance. Therefore, it is important to allow the child with epilepsy to pursue many activities. Absolute and relative contraindications to sporting activities are shown in Table 14.6.

Management of a seizure

Observing a seizure can be a frightening experience. For an observer, there is often an overwhelming feeling of helplessness and concern that the patient may die during the seizure. It is important to remember that seizures always terminate spontaneously and that rarely is a seizure life-threatening. Furthermore, the patient experiencing the seizure usually does not feel pain or remember the event.

Any individual observing or supervising an epileptic patient should remember two things when confronted by a seizure. First, the individual must be protected from injury. Second, the seizure must be closely observed in order to give an accurate description to the patient's physician. The long-standing convention of trying to put a knotted sheet or spoon in the patient's mouth should be discouraged and the patient should not be physically restrained under most circumstances.

It is important to remember that the shaking will cease spontaneously after a period of time. At the end of this time, the patient breathes normally and appears sleepy. The patient should then be managed as for an unconscious patient (e.g. Chapter 18).

Table 14.6 Solo sporting activities contraindicated in people with epilepsy.

Absolute	Relative (with supervision)
Rock climbing	Swimming
Flying	Cross-country skiing
Hang-gliding	Backpacking
Pistol shooting	Cycling
Scuba diving	
Archery	
Sky diving, parachuting	
Motor racing	

Conclusions

Overall, people with epilepsy are able to participate in sport with few limitations. Occasionally, it is appropriate to restrict certain physical activities. A person with epilepsy must meet certain legal obligations when driving a car. The individual with epilepsy must take his/her medication correctly and ensure a well-balanced eating and sleeping schedule. Family, friends, team mates, and coaches must be aware of the epilepsy and understand what to do in the event of a seizure. All these factors will contribute to removing unnecessary barriers to a normal active lifestyle in those with epilepsy.

Other neurolologic conditions and exercise

Multiple sclerosis

The clinical presentation of multiple sclerosis (MS) is characterized by a plethora of neurologic signs and symptoms such as fatigue, motor weakness, and poor balance. These episodes may be "relapsing and remitting" or "progressive" in nature. Specific

episodes, particularly spinal cord lesions, often result in persistent problems such as lower limb spasticity, muscle spasms, or sphincteric disturbance.

In addition, MS symptoms may lead to physical inactivity associated with the development of secondary diseases and/or muscular deconditioning (Slawta *et al*. 2002). Although exercise prescription is gaining favor as a therapeutic strategy to minimize the loss of functional capacity in chronic diseases, it remains underutilized as an intervention strategy in the MS population. However, a growing number of studies indicate that exercise in patients with mild to moderate MS provides similar fitness and psychologic benefits as it does in healthy controls with minimal adverse effects (White & Dressendorfer 2004).

Despite the often unpredictable clinical course of MS, exercise programs designed to increase cardiorespiratory fitness, muscle strength, and mobility provide benefits that enhance lifestyle activity and quality of life while reducing risk of secondary disorders.

Parkinson's disease

Parkinson's disease is characterized by a progressive failure of dopaminergic function in the midbrain and manifest by the clinical triad of resting tremor, muscular rigidity, and bradykinesia. In addition, postural instability is a characteristic finding. Interestingly, higher levels of physical activity in early adulthood seems to be protective against the development of Parkinson's disease (Chen *et al*. 2005). The mechanism for this is unclear.

As a consequence, the physical deconditioning that accompanies advancing disease in combination with the clinical signs makes the individual particularly prone to falls and injury. Exercise programs and balance training improves the functional status and independence of patients with Parkinson's disease (Hirsch *et al*. 2003) as well as improving overall quality of life (Baatile *et al*. 2000).

Alzheimer's disease

The role of exercise and other non-pharmacologic therapy in aging and dementia has received considerable interest in recent times. Although epidemiologic studies are conflicting, it would seem that physical activity may help preserve (and possibly improve) cognitive function and decrease overall dementia risk. These benefits are seen even with relatively low intensity exercise programs (Bragin *et al*. 2005; Larson & Wang 2004; Petrovitch & White 2005; Rovio *et al*. 2005). The role of exercise in the overall management of dementia needs to be studied further.

By contrast, concern has been raised that either repeated sport-related concussions or repeated head impact can predispose to chronic brain injury and conditions such as Alzheimer's dementia (McCrory 2003), although the evidence for this is far from compelling at present. In boxing, where repetitive brain trauma does occur, the so-called "punch drunk syndrome" or chronic traumatic encephalopathy is seen at extremes of trauma or in conjunction with specific genotypes suggesting a multifactorial basis (Jordan 2000).

Motoneuron disease (amyotrophic lateral sclerosis)

Concern has been raised that either repeated concussions or repeated head impact can predispose to chronic brain injury and conditions such as Alzheimer's dementia (McCrory 2003) and motoneuron disease (McCrory 2005).

Chio *et al*. (2005) reported from Italy that there was an increased risk of developing motoneuron disease (MND) amongst Italian soccer players. In this retrospective cohort study, there were five diagnosed MND cases in a subpopulation of 7435 soccer players of the top two Italian divisions who played in the period 1970–2001. Although only small numbers of MND patients were identified, this exceeded the statistical likelihood of developing MND in this population.

This paper adds to the growing body of concern in regard to the risk of developing this condition from sport. Previously, a judicial report from the Italian soccer leagues raised similar concerns. A 4-year study commissioned by a local magistrate looked at every player in Serie A and B between 1960 and 1997. Of the total of 24,000 calciatori, eight were found to have died from MND. A further follow-up of those who were dead or who had fallen

ill since 1997 found a further 32 cases (McCrory 2005).

The *Guardian* newspaper in England has reported that MND has claimed a number of former players in England in recent years including Don Revie, Rob Hindmarch of Derby and Sunderland, Middlesbrough's Willie Maddren, and the former Celtic winger Jimmy Johnstone (Fotheringham 2003).

Compared to individuals with other neurologic disease, patients with amyotrophic lateral sclerosis (ALS) are more likely to have a history of being athletic and slim, according to Scarmeas *et al*. (2002). Such a somatotypic linkage has been suggested by the development of MND in athletes. In the USA, boxer Ezzard Charles, baseball player Catfish Hunter, and baseball icon Lou Gehrig died of MND. Three players from the San Francisco 49ers were diagnosed with MND in the 1980s, and Glenn Montgomery of the Seattle Seahawks lost his life to MND in 1998. It is likely that the pathogenesis of MND reflects a complex interaction between environmental factors, exercise, and specific susceptibility genes (Majoor-Krakauer *et al*. 2003).

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