Medicine Matters
2016

Version: 0.0
Contents

NEWS FROM UEFA

FOOTBALL AND EPILEPSY

GROIN PAIN SYNDROME: THE ADOPTION OF COMMON LANGUAGE

PALET-RICH PLASMA
News from UEFA by Michel D’Hooghe, chairman of the UEFA medical committee

The UEFA EURO 2016 has been an extremely successful tournament, with 24 of the world’s strongest footballing nations competing in France. The greatest moment in the history of Portuguese football was written; twelve years after the final defeat at home against Greece, Portugal were proudly lifting that long waited trophy.

It was essential that UEFA’s medical operations were designed to ensure the best possible services for those participating in, working at and attending European football’s highest event. UEFA collaborated with EURO 2016 SAS and the Fédération Française de Football to develop a detailed medical concept for the tournament. The medical committee reviewed and approved the medical concept to ensure that the planned services for all target groups met UEFA’s requirements and regulations. Prof. Pierre Rochongar, vice-chairman of the UEFA medical committee, contributed to the development of the medical concept and recommended high profile doctors around France from his network. During the tournament, all training sessions at team base camps were fully covered by ambulances and emergency doctors, while every participating team had a dedicated liaison doctor, recommended by Prof Rochongar, to assist them with any medical needs they may have had, as well as a local hospital with relevant specialists on standby. At every stadium, UEFA’s detailed medical requirements for players and officials were put in place: pitchside emergency doctors with lifesaving equipment, stretcher teams, ambulances and a fully equipped medical room. Crowd medical services were planned according to French law, with multiple first-aid posts and ambulances at every stadium. UEFA Medical Officers were based in each host city, and inspected stadium medical services before all MD-1 training sessions and all matches. In addition, they provided medical support to UEFA staff in the host cities. Four UEFA Medical Officers were present at the referees’ base camp near Paris to ensure permanent medical coverage for the referees. UEFA’s general medical and anti-doping officer for the tournament, Dr Mogens Kreutzfeldt, was based in Paris to oversee operations and to provide medical support to UEFA staff and officials.
In addition to the medical plan, much focus was turned to the EURO 2016 antidoping programme. In advance of the tournament, 23 collaboration agreements were signed with European National Anti-Doping Organisations (NADOs) in order to share intelligence, whereabouts and testing plans. With the expansion of the EURO to 24 teams, this collaboration was crucial in improving UEFA’s capacity to test players in the pre-tournament phase. The sharing of the whereabouts information enabled all 24 teams to be tested at least once by UEFA or the team’s NADO in a ‘no-notice’ out of competition test. A total of 32 out of competition tests were conducted by UEFA and 1,464 samples were collected on EURO players by UEFA and the NADOs either in the pre-tournament period or between EURO matches. An agreement was also signed in 2015 between UEFA and the World Anti-Doping Agency (WADA) for the use of ADAMS. The use of ADAMS and the collaboration agreements with NADOs enabled UEFA to identify that an additional 336 samples were collected on EURO players from the 1st January 2016. This enabled UEFA to deliver a balanced pre-tournament programme based on intelligence and knowledge sharing. During the EURO, all 51 matches in the tournament were tested with players selected either by random draw or by UEFA target. All samples were analysed in WADA accredited laboratory in Châtenay Malabry, Paris. The laboratory of the “Agence Française de Lutte contre le Dopage” (AFLD), applied an analytical menu designed by Europe’s top Anti-Doping experts and scientists. The EURO samples were analysed within 24 hours of receipt by the laboratory to ensure the initial results were known before the team’s next game. All urine and whole blood samples collected at the EURO were also included in the player’s steroid and blood passports. The laboratory results and biological passports of all EURO players were analysed by an independent expert with no positive samples reported. In an additional deterrent measure, UEFA has introduced a long-term sample storage programme. All the EURO 2016 samples will be stored, meaning that UEFA will be in a position to re-analyse samples if new analytical methods become available or additional intelligence is received.

Jan Ekstrand, former vice-chairman of the UEFA medical committee, ran an extensive injury study for all 24 teams collecting injury data during the pre-tournament and the tournament phase. The results of the study can be found on UEFA.org.

I hope that you enjoyed this short résumé of the medical and anti-doping programme carried out at UEFA’s EURO 2016. It is with great pleasure that I now present to you three wide ranged articles depicting the continuous hard work that goes on within European medical community in football.
FOOTBALL AND EPILEPSY

1.1 WHAT IS EPILEPSY?

1.2 SPORT AND EPILEPSY: WHY THE RETICENCE?
Is sport (and football in particular) a bad idea for epileptic patients? This condition still has faint associations with demonism and alcoholism and is often a source of fear. We will seek to define the condition, as well as ascertaining the impact that playing sport can have on epilepsy – and, conversely, the impact that epilepsy can have on sporting performance.

Dr Arnaud Biraben

Neurologist and neurosurgeon, Rennes University Hospital – president of the French League Against Epilepsy

Pr Pierre Rochcongar

President of the medical committee of the French Football Federation
1.1 What is epilepsy?

This question is impossible to answer, as there are various different forms of epilepsy. The term spans a range of conditions, with a variety of different causes, varying prognoses and equally varied means of treatment, the one thing they have in common being the fact that all patients have epileptic seizures. It is also one of the most common neurological conditions, affecting between 0.8% and 1% of the population, making it ten times more common than Parkinson's disease or multiple sclerosis.

An epileptic seizure is defined as the clinical signs that accompany an abnormal neuronal discharge. Such abnormal discharges can only be observed using an electroencephalogram (EEG). However, an EEG needs to be carried out during a seizure in order to properly confirm the diagnosis. While the presence, between seizures, of acute anomalies (spikes, spike waves, polyspikes, etc.) points towards such a diagnosis, those EEG anomalies, taken in isolation, are not necessarily a sign of epilepsy if no seizure has ever occurred. It is thought that 4% of the population experience spikes or spike waves without ever having a seizure. Conversely, a genuinely epileptic patient can have a normal EEG between seizures, and even a series of EEGs cannot definitively rule out epilepsy. Diagnosis is often difficult and is essentially clinical, based on the questioning of the patient and witnesses.

Epilepsy is defined as the spontaneous repetition of seizures. Thus, someone who only has seizures while in a state of inebriation is not an epileptic; their brain is simply sensitive to alcohol.

There are various different causes of epilepsy. Ultimately, any attack on the brain can cause epileptic seizures. In very simple terms, there is a threshold as regards the excitement of neurons above which a seizure occurs. If the threshold is exceeded, a seizure is triggered; if the person remains below the threshold, there is no seizure.

If that threshold is very high, only an electric shock will trigger a seizure (and that can happen to anyone). However, if the threshold is low, seizures can occur during everyday activities. If it is low throughout the cerebral cortex, the seizure will immediately ignite the entire cortex, resulting in a generalised seizure. Conversely, if the threshold is low in just one small area, it will result in a partial seizure, and the patient will always have the same type of seizure, affecting that one area.

In the first case, there is general brain dysfunction and the patient remembers nothing (referred to as ‘postictal amnesia’). In the second case, it is very common for the patient to feel the seizure coming (termed an ‘epileptic aura’). This allows them to move to a safe place or sound the alarm. A seizure where the patient remains fully conscious throughout is called a ‘simple partial seizure’, while any change in consciousness is referred to as a ‘complex partial seizure’.

A seizure can also spread to the rest of the cortex, which is termed a ‘secondary generalised seizure’. Certain factors reduce the threshold and make epileptic seizures more likely (alcohol, certain types of medication, lack of sleep, forgetting to take medication, etc.). In most cases, the causes of generalised epilepsy are congenital, while any focal brain injury (resulting from a stroke, a head trauma, a tumour, etc.) can give rise to focal seizures.
Between 60% and 70% of all epileptics have partial seizures (so between 30% and 40% have generalised seizures). Around 60–70% of epileptics are stabilised by their treatment if they have a healthy lifestyle, 30% have drug-resistant conditions, and in 5–10% of cases the epilepsy is accompanied by encephalopathy and multiple disabilities.

There are different types of generalised seizure, such as absence seizures (involving a brief loss of consciousness, with no movement), tonic-clonic seizures and myoclonic seizures. The symptoms of partial seizures vary depending on the area of the brain that is initially affected: motor symptoms in the case of the frontal lobe, sensory symptoms in the case of the parietal lobe, visual symptoms in the case of the occipital lobe, déjà vu or other memory-related symptoms in the case of the temporal lobe, and so on.

Patients’ disability stems entirely from the fact that they cannot predict when the next seizure will occur: while out shopping, crossing the road, playing sport, at work, etc.
1.2 Sport and epilepsy: why the reticence?

1. Is there a risk of seizures being caused by exertion, or by hyperpnoea brought on by exertion?
2. Does the condition impair patients’ sporting performance? What effect does their medication have?
3. What is the risk of an accident resulting from a seizure while playing football?
4. What needs to be done in the event of a seizure? Does an ambulance need to be called? Does the patient need to go to hospital? Or do we know enough about the condition to handle the situation ourselves?

1.2.1 Is there a risk of seizure being caused by exertion, or by hyperpnoea brought on by exertion?

Generally speaking, accidents are no more likely while playing sport than they are at home. It appears that only around 0.5% of epileptic patients have seizures that are triggered by sport (so sport does not trigger seizures in 99.5% of cases), while lack of sleep and stress trigger seizures in 18% and 30% of cases respectively (Frucht et al., 2000). The risk of having a seizure while playing sport is very small (with such seizures being suffered by only 2% of the patients monitored by Nakken et al. in 1999 and Korczyn et al. in 1979).

In certain extreme situations, the epileptic threshold could be lowered by the stress of competition, the resulting fatigue, exertion, hypoxia or hypoglycaemia. Prolonged exertion combined with inappropriate hydration can produce hydroelectrolytic imbalances, but not epileptic seizures! Meanwhile, medical literature shows that exertion has either no impact or a positive impact on the frequency of seizures (Frucht et al., 2000; Heise et al., 2002; Ericksen et al., 1994; and Nakken et al., 1990). In the event of prolonged exertion and/or exertion at altitude, basic precautions should be taken.

The playing of sport also has a positive impact on the psychiatric comorbidities associated with epilepsy (namely depression and chronic anxiety), improving patients’ prognosis, mood and quality of life (Roth et al., 1994). Improvements in EEG anomalies have even been observed (Gotze et al., 1967; Horyd et al., 1981; and Nakken et al., 1994).

There is a scientific reason for these findings: attention and increases in sensory input (which raise attention levels) reduce the risk of a seizure occurring. By raising the patient’s lactate and acidosis levels, exertion increases levels of GABA, the neurotransmitter that inhibits neurons. The adenosine that is excreted during exertion also has an impact on various neuroreceptors, acting as an anticonvulsant.

Finally, hyperpnoea is used to help trigger seizures during EEGs. For a long time now, studies have shown that hyperpnoea that is not accompanied by exertion causes hypocapnia and alkalosis, which make seizures more likely, while physiological hyperpnoea resulting from exertion helps the body to maintain homeostasis and has no epileptogenic effect (Wasserman et al., 1973).

In a recent article, Arida et al. (2013) reviewed literature studying both epileptic animals and human patients. In the case of animals and humans alike, those studies confirmed not only that exertion and sport have a positive impact on epilepsy, but also that epilepsy does not appear to have a negative impact on sporting performance.

What sport should patients choose?
Sports played on the ground would obviously seem to be preferable: team sports (i.e. including football), dance, martial arts, etc. Water sports (swimming, windsurfing, water skiing, etc.), equestrian sports and cycling should not be practised without supervision, and the same goes for gymnastics apparatus. Finally, sports where a seizure could be fatal (mountaineering, scuba-diving, skydiving, hang-gliding, paragliding, motor sports, etc.) should be avoided. The same is true of boxing, given the repeated blows to the head.

1.2.2 Does the condition impair patients' sporting performance?

What effect does their medication have?

None of the various anti-epileptic drugs (AEDs) are regarded as performance-enhancing substances. Certain AEDs may increase patients’ appetite, in which case sport will certainly have a positive impact. All AEDs have the effect of inhibiting the central nervous system, which may cause drowsiness. Doctors will amend the treatment on a case-by-case basis to determine the lowest effective dose for each patient. There are now around 30 different AEDs available, enabling doctors to select the best type of treatment for a given form of epilepsy or a particular epileptic. Taking AEDs is no barrier to performance – even world-class levels of performance.

1.2.3 What is the risk of an accident resulting from a seizure while playing football?

As each form of epilepsy is different, this decision needs to be made on a case-by-case basis. The majority of patients are able to manage their conditions well using medication; the only problem may be the ‘third half’! There is not necessarily any reason why epileptics should not play. If seizures occur too frequently, interfering too much with the game, that could cause difficulties, as could the behavioural problems and (involuntary) aggression that follow certain types of seizure, or psychiatric problems associated with the condition. In those cases, though, it is not the epilepsy itself which is the problem. Finally, there are those exceptional cases (0.2% of epileptics) where it is sport that triggers seizures.

Ultimately, we need to ask ourselves whether a seizure on a football pitch is worse than a seizure at home.

It would seem only sensible to warn the coach and certain administrators at the club of the risk of a seizure, so that there are no unpleasant surprises, and to educate people around the patient, telling them what to do in such a situation (breaking medical confidentiality only with the permission of the patient or their parents, of course). Someone who is ‘in the know’ can then assist the patient, ensure that they take their medication, try to manage risk factors such as alcohol and lack of sleep, etc. That is the approach that we developed at the French Football Federation, informing players, administrators and referees (www.fff.fr/actualites/145124-553661-coup-denvoi-de-la-campagne-foot-et-epilepsie).

1.2.4 What needs to be done in the event of a seizure?

Does an ambulance need to be called?

Does the patient need to go to hospital?

Or do we know enough about the condition to handle the situation ourselves?
We will look at the case of a generalised tonic-clonic seizure. When the seizure occurs, it is necessary to (i) protect the head, (ii) move everything out of the way that could cause injury during the clonic phase, and (iii) place the person in the recovery position as soon as possible. You could also try to prevent the patient from biting their tongue, without using either a metal object (given the risk of breaking a tooth) or your fingers. If you manage to get your wallet in their mouth in time, they will be grateful. It is not possible to ‘swallow your tongue’ during an epileptic seizure; that is a myth.

Try to observe the various symptoms of the seizure, then check that the patient is breathing and their heart is beating. (Is it a convulsive syncope?) Calmly wait for the patient to slowly recover, which will normally take a few minutes. Take note of any postictal deficits (aphasic deficits, motor deficits, etc.).

**When should you call an ambulance or go to hospital?**

- First seizure (as far as you are aware)
- Seizure lasting more than five minutes (so it is important to time it)
- Two seizures in succession
- Injury requiring medical attention (dislocated shoulder, head trauma, etc.)
- Loss of consciousness lasting more than ten minutes
- Known or suspected intoxication
- Fever and/or meningeal signs
- Neurological deficits that are new or do not resolve themselves quickly
- Intense and persistent headaches

**When is an ambulance not required?**

In the case of a known epileptic, an ambulance does not need to be called in the event of a ‘normal’ seizure with a ‘normal’ return to consciousness, which explains why it is useful to warn people.

An epileptic patient will always be grateful for not making them waste a day at the hospital after what for them is a very ordinary seizure.

**Conclusion**

Sport has only beneficial effects for epileptic patients and their mental and social health. It probably even has the effect of reducing the likelihood of seizures by tackling the anxiety and depression that are often associated with – and exacerbate – the condition. It also has an impact on patients’ waistlines, as some AEDs increase their appetite, as well as helping to tackle the osteoporosis that is associated with AEDs. It is thought that sport even has a positive impact on the – thankfully exceptionally small – risk of sudden unexpected death in epileptic patients (SUDEP). It is exceptionally rare for seizures to be triggered by sport (with only 0.2% of epileptics falling into this category).

Epilepsy is still sometimes a source of fear on account of the aura that surrounds it, the fact that there is no sign of the condition prior to a seizure, and the unpredictability of seizures. We have to remember that, while we may not be used to dealing with seizures, the 30% of patients who have drug-resistant epilepsy are.

There is almost no reason for patients not to play football – quite the opposite. However, others should be made aware of the situation, in the interests of honesty and to avoid any panic. There is no shame in being epileptic.

Denying an epileptic the opportunity to play fosters both exclusion and stress.
GROIN PAIN SYNDROME: THE ADOPTION OF COMMON LANGUAGE

2.1 TERMINOLOGY
2.2 ARTICULAR CAUSES
2.3 VISCERAL CAUSES
2.4 BONE-RELATED CAUSES
2.5 MUSCLE/TENDON-RELATED CAUSES
2.6 PUBIC SYMPHYSIS-RELATED CAUSES
2.7 NEUROLOGICAL CAUSES
2.8 DEVELOPMENT-RELATED CAUSES
2.9 CAUSES RELATED TO INFLAMMATORY AND NON-INFLAMMATORY GENITOURINARY DISEASES
2.10 INFLAMMATORY MUSCULO-SKELETAL CAUSES
2.11 NEOPLASTIC CAUSES
2.12 INFECTIOUS CAUSES
2.13 SYSTEMIC CAUSES
2.14 MACRO SUBDIVISION
2.15 CONCLUSIONS
2.16 REFERENCES
Volpi, P.1,2

Bisciotti, G. N.2

1. Istituto Clinico Humanitas, IRCCS, Rozzano, Milan, Italy

2. Medical staff of FC Internazionale Milano, Milan, Italy
2.1 Terminology

It is always important, when tackling a subject as thorny and controversial as groin pain, to remember that this term – as opposed to all the other substantially interchangeable terms, such as ‘pubalgia’, ‘athletic pubalgia’, ‘groin disruption’ or ‘osteitis pubis’, which express the same type of symptomatology – is in fact only a description of a symptom (or rather, a group of symptoms) that the patient complains of at the level of the pubic area. For this reason, we always need to be extremely careful not to equate the term ‘groin pain’ with a diagnosis. In fact, groin pain represents a multifactorial etiopathogenesis, which often involves overlapping clinical frameworks that sometimes make diagnosis a real challenge. In addition, we must objectively admit that the anatomical complexity of the pubic region certainly does not facilitate the adoption of a clear nosological nomenclature.

In our view, the frequent overlapping of different clinical frameworks linked by causal relationships, combined with the complex of symptoms which help to characterise a well-defined groin pain framework, justifies the use of a different, more appropriate term: ‘groin pain syndrome’ (GPS) (Bisciotti et al., 2013a; 2013b; 2015). Indeed, the term ‘syndrome’ represents the most suitable way of describing the set of symptoms and clinical signs that characterise the clinical framework – or, more frequently, clinical frameworks – involved in the onset of a groin pain framework. Obviously, the term ‘GPS’ must be supplemented by a description of the associated clinical framework. Thus, we could, for example, have a GPS caused by adductor tendinopathy or an inguinal hernia, or a combination of the two, or a combination of other factors. In our opinion, therefore, it is only by adopting a descriptive global term such as ‘GPS’ and combining it with a clinical description of the cause of the patient’s symptoms that we can achieve a clear and rational identification of the problem.

Below, therefore, we propose a summary of the various clinical frameworks that cause GPS, based on studies by different authors (Le Blanc & Le Blanc, 2003; Omar et al., 2008; Jansen, 2010; Brophy & Prather, 2014; Gilmore et al., 2014; Lyons & Brunt, 2014), which we have summarised, sorted and divided into different categories. This will be proposed in the forthcoming Italian Groin Pain Consensus in February 2016.
2.2 Articular causes

1. Acetabular labrum lesion
2. Femoro-acetabular impingement (FAI)
3. HALTAR® lesions
4. Hip arthritis
5. Intra-articular snapping hip
6. Legg-Calvè-Perthes syndrome
7. Dysplasia
8. Epiphysiolysis
9. Head femoris avascular necrosis
10. Growth plate fracture
11. Sacroiliac joint dysfunctionality
12. Lumbar column disease
13. Synovitis
2.3 Visceral causes

1. Inguinal hernia
2. Other types of abdominal hernia
3. Weakness of the inguinal wall
4. Testicular torsion
5. Appendicitis
6. Diverticulitis
7. Gastrointestinal disorders
2.4 Bone-related causes

1. Stress fractures
2. Avulsion fractures
2.5 Muscle/tendon-related causes

1. Rectus abdominis injuries
2. Rectus abdominis tendinopathy
3. Adductor muscle injuries
4. Adductor muscle tendinopathy
5. Direct muscle trauma
6. Injuries to the common aponeurosis shared by the rectus femoris and the adductor longus
7. Iliopsoas injuries
8. Iliopsoas tendinopathy
9. Iliopsoas impingement
10. Internal snapping hip
11. External snapping hip
2.6 Pubic symphysis-related causes

1. Osteitis pubis
2. Symphysis instability
2.7 Neurological causes

1. Nerve entrapment syndrome
2.8 Development-related causes

1. Apophysitis
2. Growth plate at pubic level
2.9 Causes related to inflammatory and non-inflammatory genitourinary deseases

1. Prostatitis
2. Epididymitis
3. Orchitis
4. Varicocele
5. Hydrocele
6. Urethritis
7. Urinary tract infections
8. Cystitis
9. Ovarian cysts
10. Endometriosis
11. Ectopic pregnancy
12. Round ligament entrapment
2.10 Inflammatory musculo-skeletal causes

1. Bursitis
2.11 Neoplastic causes

1. Testicular cancer
2. Osteoid osteoma
3. Colorectal cancer
2.12 Infectious causes

1. Osteomyelitis
2. Septic osteoarthritis
2.13 Systemic causes

1. Inguinal lymphadenopathy
2. Inflammatory arthropathies
3. Fibromyalgia

Thus, on the basis of the literature to date, we can divide the most common and probable pathologies capable of causing GPS (i.e. 59 possible clinical pictures) into 12 different nosological categories (see Table 1).

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of pathologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Articular causes</td>
<td>13</td>
</tr>
<tr>
<td>Visceral causes</td>
<td>7</td>
</tr>
<tr>
<td>Bone-related causes</td>
<td>2</td>
</tr>
<tr>
<td>Muscle/tendon-related causes</td>
<td>11</td>
</tr>
<tr>
<td>Pubic symphysis-related causes</td>
<td>2</td>
</tr>
<tr>
<td>Neurological causes</td>
<td>1</td>
</tr>
<tr>
<td>Development-related causes</td>
<td>2</td>
</tr>
<tr>
<td>Causes related to inflammatory and non-inflammatory genitourinary diseases</td>
<td>12</td>
</tr>
<tr>
<td>Inflammatory musculo-skeletal causes</td>
<td>1</td>
</tr>
<tr>
<td>Neoplastic causes</td>
<td>3</td>
</tr>
<tr>
<td>Infectious causes</td>
<td>2</td>
</tr>
<tr>
<td>Systemic causes</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 1: Overview of the 59 most likely causes of GPS, divided into 12 different nosological categories

Furthermore, in accordance with the report presented at the Doha Agreement Meeting on Definitions and Terminology on Groin Pain in Athletes (Weir et al., 2015), we would advise against the use of the following terms in the description of GPS:
a. Athletic groin pain
b. Athletic pubalgia
c. Biomechanical groin overload
d. Gilmore's groin
e. Gracilis syndrome
f. Groin disruption
g. Hockey-goalie syndrome
h. Pectineus syndrome
i. Pseudo-pubalgia
j. Pubalgia (the term 'groin pain syndrome' being preferable)
k. Soft groin
l. Sports hernia (the term 'weakness of the inguinal wall' being preferable)
m. Sports groin
n. Sportsman's groin
o. Sportsman's hernia
p. Adductor tendinitis (the term 'adductor tendinopathy' being preferable)
q. Rectus abdominis tendinitis (the term 'rectus abdominis tendinopathy' being preferable)
r. Iliopsoas tendinitis (the term 'iliopsoas tendinopathy' being preferable)
2.14 Macro subdivision

In addition, we can divide GPS into three broad subcategories:

a. ‘Overuse GPS’, which is characterised by an insidious and progressive onset whereby the patient has no memory of an injury or incident to which the onset of pain can be attributed with any certainty.

b. ‘Traumatic GPS’, whereby the onset of pain is linked to a precise traumatic event and this hypothesis is supported by the patient’s history, a physical examination and clinical imaging.

c. ‘Long-standing GPS’ (LSGPS) or ‘chronic GPS’, whereby the group of symptoms suffered by the patient endures for a long time and is resistant to all conservative therapies. It is important to stress that both overuse GPS and traumatic GPS can result in LSGPS, while traumatic GPS may occur in the context of overuse GPS or LSGPS. LSGPS can be regarded as a clinical framework that persists for more than eight weeks (Hölmich et al., 1999; Jansen et al., 2008).
2.15 Conclusions

The dispute regarding diagnostic classification in this field can only be overcome through the adoption of shared language that satisfies the principles of clarity, accuracy and commonality. With this in mind, we suggest the following:

a. the adoption of the term ‘groin pain syndrome’ as a term describing a group of symptoms that the patient complains of in the pubic area;

b. the division of GPS into three subcategories: overuse GPS, traumatic GPS and LSGPS;

c. the use, for diagnostic purposes, of one or more of the nosological nomenclatures reported in Table 1, depending on the relevant clinical picture.
2.16 References


2.16.1 Footnotes

1. Cam FAI, pincer FAI or subspine impingement
2. Hip anterosuperior labral tear with avulsion of rectus femoris
PALET-RICH PLASMA

3.1 DOES PLATELET-RICH PLASMA ENHANCE MICROFRACTURE TREATMENT FOR CHRONIC SMALL CHONDRAL DEFECTS?
3.2 ABSTRACT
3.3 INTRODUCTION
3.4 METHODOLOGY
3.5 STATISTICAL ANALYSIS
3.6 RESULTS
3.7 DISCUSSION
3.8 CONCLUSIONS
3.9 REFERENCES
3.1 Does platelet-rich plasma enhance microfracture treatment for chronic small chondral defects?

A cohort study performed using footballers
Henrique Jones
UEFA Medical Committee; Clínica Ortopédica do Montijo; Lusófona University, Lisbon;
oretojones@gmail.com
3.2 Abstract

3.2.1 Purpose

The purpose of this study was to compare the effectiveness of (i) platelet-rich plasma (PRP) in combination with microfractures (MF) and (ii) microfractures alone in the treatment of chronic focal chondral defects in athletes, using a new surgical technique – the multi-needle technique.

3.2.2 Study design

The study used 46 professional footballers with small (i.e. <3cm²) chronic chondral symptomatic knee lesions (and no other relevant pathology) who were submitted for surgery between 2011 and 2012.

3.2.3 Methodology

The players were divided into two groups. The first group was treated with microfractures in combination with platelet-rich plasma, and the second group was treated with microfractures alone. The lesions were classified on the basis of the Outerbridge classification, and the results were validated using the Chondral Defect Scoring Scale (CDSS) and radiological evaluation.

3.2.4 Results

The PRP-augmented microfracture group had a significantly better mean CDSS score at 18 months (p<0.01) than the group treated with microfractures alone.

3.2.5 Conclusion

The addition of platelet-rich plasma to microfracture treatment appears to result in an improved CDSS score in footballers relative to microfractures alone.

3.2.6 Key terms

Chondral defects; microfractures; platelet-rich plasma; autologous growth factors; multi-needle technique.
3.3 Introduction

Sports-related chondral knee injuries are a common diagnosis in footballers with symptomatic knees. The microtrauma associated with such lesions acts as a persistent condition in football players, just like a cumulative trauma disorder. Many surgical techniques have been proposed to remedy chondral lesions (1, 2, 3, 4), and microfracturing is one of the best-known techniques (5, 6, 7, 8, 9).

Platelet-rich plasma is a highly concentrated formula derived from the patient’s own blood (10). Platelets contain growth factors that may enhance current cartilage repair techniques (11) through multiple mechanisms, including the recruitment of chondrogenic cells (chemotaxis), the stimulation of chondrogenic cell proliferation (mitogenesis) and the enhancement of cartilage matrix synthesis (10). Many studies suggest a potential role for these potent biological regulators of chondrocytes in cartilage repair (12).
3.4 Methodology

Between 2010 and 2012, 46 professional footballers (40 men and 6 women) with small (i.e. <3cm²) chronic chondral symptomatic knee lesions (and no other relevant pathology) were submitted for surgery. They ranged between 16 and 39 years of age, with a mean age of 26.0. The same surgeon conducted all surgeries, and patients were allocated to one of two treatment groups. The major symptoms were pain, swelling and functional disability (FD). The internal condyle (IC) was the most affected area of the knee in both groups (see table below).

<table>
<thead>
<tr>
<th>BASELINE CHARACTERISTICS</th>
<th>MF + PRP group n=23</th>
<th>MF group n=23</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years): Mean; standard deviation</td>
<td>25.2; 4.3</td>
<td>28.6; 5.0</td>
<td>0.018 (*)</td>
</tr>
<tr>
<td>% male</td>
<td>91.3%</td>
<td>82.6%</td>
<td>0.665 (**)</td>
</tr>
<tr>
<td>Location</td>
<td>IC 19; EC 3; Pat. 1</td>
<td>IC 19; EC 4</td>
<td></td>
</tr>
<tr>
<td>Major symptoms</td>
<td>Pain; swelling; FD</td>
<td>Pain; swelling; FD</td>
<td></td>
</tr>
</tbody>
</table>

(*) Independent samples t-test (after verifying normality using Shapiro-Wilk test and equality of variances using Levene’s test); not statistically significant at 1%.

(**) Fisher’s exact test; not statistically significant at 5%.

The Outerbridge classification (13) was used to classify the severity of each injury. The first group of 23 players were treated with microfractures combined with PRP (MF + PRP group), using the GPS III System (Gravitational Platelet Separation System, Biomet® Biologics LLC, Warsaw, Indiana, United States) for platelet separation. This system concentrates both platelets and white blood cells. The second group (MF group) were treated with microfractures alone. The injuries were mostly grade III, with some grade IV. The objective of the study was to demonstrate the validity of arthroscopic microfracture treatment in combination with PRP for small chondral knee lesions in footballers, using an adequate/validated scoring scale.

All the footballers had an X-ray and an MRI (14, 15) before surgery, and the Chondral Defect Scoring Scale was used to analyse the subjective and objective results (8). The Mini GPS III System used needs 27ml of blood combined with 3ml of ACD-A (antiocoagulant) to produce 3ml of PRP in 15 minutes (10).

The arthroscopic surgery involved exploration of the entire knee looking for associated injuries. The cartilage defect was then identified and cleaned using a surgical power tool. The microfracture technique began with multiple perforations 3–4mm in diameter and 6–8mm deep using traditional awls. The difficulty of applying PRP in microfracture holes during an arthroscopic procedure had led us to develop a new application technique, which we named the ‘multi-needle technique’, in which multiple 14mm or 16mm catheters were inserted into the microfracture holes, allowing individual (depending on the number of holes) injections of PRP into each microfracture hole (see Fig. 1).
During PRP application the joint was dry, and the tourniquet was released after five to ten minutes, allowing the main growth factors to be released from the platelets (16). Compression dressing and an ice pack were applied immediately after surgery.
Rehabilitation began with the use of an arthromotor the day after surgery, allowing a tolerated range of motion, with no weight bearing for four weeks. During this period, muscle strength exercises, range-of-motion exercises, electrical stimulation and massage were used. In week 4, partial weight bearing began, gradually progressing to full weight bearing in week 6. Muscular and articular recovery and neuromuscular training were continued between week 6 and week 16, followed by plyometrics, running exercises and individual ball training on the pitch in week 16.

A return to competition was allowed between weeks 20 and 24. The rehabilitation protocol was the same for both groups in the study.
3.5 Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics 19 software. The Type I error level used was 5%, unless otherwise indicated. Exploratory analysis was first conducted. Means and standard deviations were calculated for players' ages and CDSS scores, separately for each time period and for each of the two groups. The assumed normality of the distributions of ages and CDSS scores in the observed groups was assessed using Shapiro-Wilk tests. An independent samples t-test was used to test for a difference in mean ages between groups (after verifying normality using a Shapiro-Wilk test and verifying the equality of variances using Levene's test), and non-parametric Mann-Whitney tests were applied to test for differences in the mean CDSS scores of the two groups for each period of time (15, 17).
3.6 Results

The clinical results were based on CDSS evaluation (both subjective and objective) before surgery and at 12 and 18 months after surgery.

The groups had similar male/female ratios and comparable mean ages, and the observed difference in ages between the groups was not clinically relevant.

Non-parametric Mann-Whitney tests were used for the statistical analysis of the data, since the distributions of the groups’ CDSS scores and the changes relative to the baseline CDSS scores were shown to be non-normal at 18 months using the Shapiro-Wilk test for normality. The difference between the CDSS score at 18 months and the CDSS score before treatment (i.e. the change relative to the baseline) was calculated for each patient, and the mean change relative to the baseline was calculated for both groups. The MF + PRP group had a significantly larger mean change than the MF group (p=0.000) and a significantly higher mean CDSS score 18 months after surgery (p=0.003; see table below).

<table>
<thead>
<tr>
<th></th>
<th>MF + PRP group</th>
<th>MF group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean CDSS score ± standard deviation (95% confidence interval)</strong></td>
<td>number</td>
<td>number</td>
<td></td>
</tr>
<tr>
<td>Before surgery</td>
<td>55.0 ± 6.9 (52.0–58.0) n=23</td>
<td>65.2 ± 6.7 (62.3–68.1) n=23</td>
<td>0.000</td>
</tr>
<tr>
<td>18 months after surgery</td>
<td>85.0 ± 9.4 (80.3–89.7) n=18</td>
<td>80.0 ± 4.8 (77.9–82.1) n=23</td>
<td>0.003*</td>
</tr>
<tr>
<td>Difference between before and 18 months after surgery</td>
<td>29.7 ± 7.4 (26.1–33.4) n=18</td>
<td>14.8 ± 5.9 (12.2–17.3) n=23</td>
<td>0.000</td>
</tr>
</tbody>
</table>

* Although statistically significant, the 95% confidence intervals overlap.

The results of the X-rays and MRIs show a better defect image, meaning a better defect fill (14), in line with the superior clinical improvement in the MF + PRP group relative to the MF group (see Fig. 2).

![Fig. 2a](image1)

![Fig. 2b](image2)
3.7 Discussion

The overall results of the treatments were good or very good with regard to both clinical outcomes and the return to competition (with CDSS scores of between 70 and 90). The improvement in the CDSS score of the MF + PRP group was, on average, significantly larger than that of the MF group.

On the basis of the statistical methodologies applied, the MF + PRP group clearly distinguished itself from the other group. The MF + PRP group had a mean CDSS score that was significantly lower than that of the MF group before treatment. After 18 months of follow-up, the MF + PRP group's mean CDSS score was superior to that of the MF group. The mean CDSS scores of both groups increased between the pre-surgery assessment and the 18-month assessment, but that of the MF + PRP group increased significantly more.
3.8 Conclusions

The addition of platelet-rich plasma to microfracture treatment appears to lead to improved results in footballers (as measured by CDSS scores and MRIs) relative to microfractures alone after 18 months of follow-up.
3.9 References


