ISSN: 0212-8799

180
Volumen 34
July - August 2017

# Archivos de medicina del deporte

Organo de expresión de la Sociedad Española de Medicina del Deporte

## ORIGINALS



## REVIEWS

# Electromyographic (EMG) activity during pedalling, its usefulness in diagnosing fatigue in cyclists

**Alfredo Córdova[1], Iván Nuin[2], Diego Fernández-Lázaro[2], Ibán Latasa[3], Javier Rodríguez-Falces[3]**

*[1]Departamento de Bioquímica, Biología Molecular y Fisiología. Facultad de Fisioterapia. Campus Universitario de Soria. Universidad de Valladolid. Soria. [2]Consulting de Recuperación Global. Bilbao. [3]Departamento de Imaginería Electrónica y Eléctrica. Universidad Pública Navarra, Pamplona.*

**Received:** 25.01.2017
**Accepted:** 16.03.2017

**Summary**

Muscle fatigue has multiple definitions, but with a special mission what is the protective mission, warning the body about weakness or the appearance of a functional disability. In this review, we present the applications of Electromyography (EMG) as a technique to gain insight into the activation patterns during cycling and the onset of fatigue.

A narrative review has been carried out in which analysis of the EMG activity during the different phases of the pedal cycle. The movement of the pedal has been studied exhaustively and has been able to distinguish 4 phases in the pedaling that originate the propulsion and the recovery. By using the EMG it is possible to describe the typical activation patterns in terms of the activity level and activation time of the main muscles of the lower limbs. Muscle activity and coordination can vary between people throughout a single cycle of pedaling and between different cycles of the same person. Moreover, we examine the main factors that can influence these electromyographic patterns during the pedal cycle. We also describe the influence of factors such as output power, cadence or frequency of pedaling, slope and posture, foot pedal interface, training level and muscle fatigue that produce alterations in the time of activation and muscular coordination.

In conclusion, we believe that EMG can detect the occurrence of muscle fatigue, either of central or peripheral origin. The method used to estimate the neuromuscular fatigue threshold from the EMG amplitude during an incremental test on a cycle ergometer is presented. In general there is an increase in amplitude to try to maintain the force and a decrease in the frequency spectrum.

**Key words:**
EMG. Pedaling. Cyclism. Fatigue.

## Actividad electromiográfica (EMG) durante el pedaleo, su utilidad en el diagnóstico de la fatiga en ciclistas

**Resumen**

La fatiga muscular tiene múltiples definiciones, pero con una misión especial cual es la misión protectora, avisando al organismo sobre la debilidad o la aparición de una incapacidad funcional. En esta revisión se hace un análisis de las aplicaciones de la electromiografía (EMG) como técnica para comprender los patrones de activación musculares durante el pedaleo y la aparición de fatiga muscular.

Se ha realizado una revisión en la cual se analizan las variaciones de la actividad EMG durante las fases del pedaleo. El movimiento del pedaleo ha sido estudiado exhaustivamente y se ha legado a distinguir 4 fases en el pedaleo que originan la propulsión y el recobro. Mediante el uso de la EMG se pueden describir los patrones de activación típicos, en cuanto al nivel de actividad y el tiempo de activación de los principales músculos de las extremidades inferiores. La actividad muscular y la coordinación pueden variar entre personas a lo largo de un solo ciclo de pedaleo y entre diferentes ciclos de la misma persona. También se examinan los principales factores que pueden influir en estos patrones EMG durante las fases del pedaleo. Asimismo, se describe la influencia de factores como la potencia de salida, cadencia o frecuencia de pedaleo, pendiente y postura, interfaz calzado pedal, nivel de entrenamiento y fatiga muscular, que producen alteraciones en el tiempo de activación y coordinación muscular.

En conclusión, la EMG permite detectar la aparición de la fatiga muscular, bien de origen central o periférico. También, estimar el umbral de fatiga de neuromuscular a partir de la amplitud EMG durante un test incremental en un cicloergómetro. Al aumentar de la amplitud para intentar mantener la fuerza y una disminución del espectro de frecuencias.

**Palabras clave:**
EMG. Pedaleo. Ciclismo. Fatiga.

**Correspondence:** Alfredo Córdova Martínez
E-mail: a.cordova@bio.uva.es

## Introduction

Muscle fatigue has multiple definitions, though we can assume the following: "the reduction in physical performance associated with a real increase or with the perceived difficulty upon performing a task or exercise"[1-3]. In reality, it has a protective function, warning the body about the weakness in energy reserves, or that functional incapacity is starting to appear[1-3].

This very common sign/syndrome is particularly accentuated among athletes. Within sport it is much clearer and easier to assess and diagnose in individual sports such as cycling. For this reason, the aim of this review is to understand electromyographic behaviour and its usefulness in diagnosing fatigue. We must mention that the electromyography (EMG) is a technique used increasingly in electrophysics as a way of assessing muscle behaviour, and in particular in diagnosing fatigue.

During the muscle contraction process, a series of events occur dominated by brain commands that discharge in the cross-bridge formation of actin-myosin, which is why fatigue can be associated with alterations to the central nervous system (CNS) or to causes associated with contractile activity[1-3]. For this reason, from a practical perspective we can distinguish two types of fatigue that will intervene directly in the muscle contraction process: a) central or regulation fatigue, in which the cause is located above the motor plate, causing a reduction in the voluntary activation of the muscle, which is due to a reduction in the number and rates of discharge of the motor units (MUs), recruited at the start of the generation of muscle strength; b) peripheral or effectuation fatigue, which affects structures below the motor plate, causing a reduction in the muscle contraction strength of the muscle fibres and changes to the mechanisms underlying the transmission of muscle action potentials[1-3].

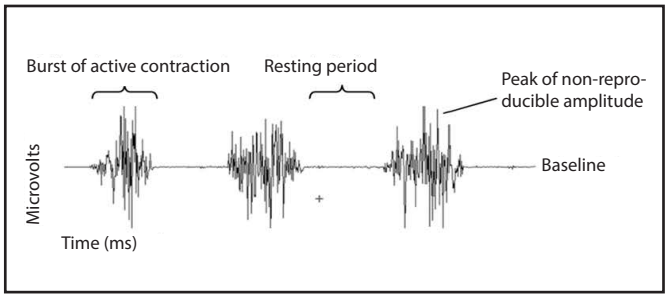
During sustained contractions in maximum efforts, or sub-maximum efforts, central and peripheral fatigue is produced, whilst only central fatigue is produced in intermittent contractions. Fatigue is more evident in maximum efforts; if it is a sub-maximum effort or if there is enough rest between contractions, only peripheral fatigue is produced[2-3].

## Some concepts of the electromyography (EMG)

The EMG is an indirect assessment measurement of muscle activity, as it detects the electrical activity that is generated by the passing of the nervous impulse that causes an action potential in the membrane of the muscle cell. This potential comprises three phases: the de-polarisation of the membrane, the re-polarisation and a period of hyper-polarisation, generating an electrical field that is collected by the EMG electrodes[4,5].

In dynamic studies the motor unit recruitments (MURs) detectable in the place where the electrode is positioned superpose electrically, observed as a bipolar signal with a symmetric distribution of negative and positive amplitudes. The signal that is obtained without filtering is the so-called "Raw signal" which is composed of periods of contraction and relaxation. In the relaxation period we observe the baseline EMG,

**Figure 1. Characteristics of the Raw Signal.**




which depends on many factors (quality of the amplifier, environmental noise and the quality of the detection condition given), and if these factors are within suitable margins, the line should not exceed 3-5 microvolts[6] (Figure 1).

There are many factors that can alter the quality of the signal, such as: the characteristics of the tissue; cross talk, i.e. the possibility of registering signals from other muscles near the one being studied, which is mainly produced with the surface electrodes; changes in the geometry between the stomach and the area of electrodes; and external noises[6].

In the signal quantification process it should be considered that many variables can affect it, such as:

- *Frequency variables*. The spectrum of frequencies reveals the frequency of activity of the MURs during the action analysed. In the EMG this frequency oscillates between 10 and 250 Hz[6]. In the frequency analysis we can extract various values: average frequency, which is the average of all the frequencies; and middle frequency (MF), the frequency at which the spectrum is divided into two regions of equal power[4]. There are also other frequency measurements that are not based on the frequency spectrum, such as the "zero cross" (number of times the raw signal crosses the baseline), which is related to the strength of contraction; the "number of turns" (point where the signal direction changes following a power difference of over 100 mV).
- *Amplitude variables*. The "integration of the signal" is studied, which is the total amount of muscle activity in an interval of time. The "envelope curve" is obtained following the rectification of the signals. In order for it to be valid, the recording must be carried out at high sample frequencies[4,6] (Figure 2).

The electrical power of the electromyographic signal is also assessed, i.e. the "root mean square" (RMS), which is the square root of the area between the square of the signal and the time counted in an interval of time divided by that time. Rectification is not required, it is obtained in variable times depending on the activity studied and it provides more information than the integrated signal[4,6].

Finally, a normalisation of the data is required, i.e. to express it regarding an obtained reference value, as the absolute values do not represent the muscle effort.

Numerous factors influence the signal received in the muscle, such as the variability of neural recruitment, the thickness of the adipose tissue, the length of the resting muscle, the cross-sectional muscle

**Figure 2. Amplitude variables. Register of the rectified signal (mV), the enveloping curve (mV) and the integrated signal (mV/s).**

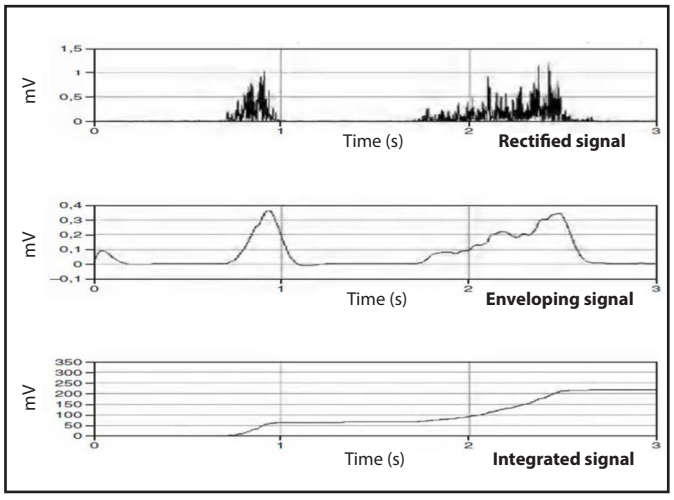
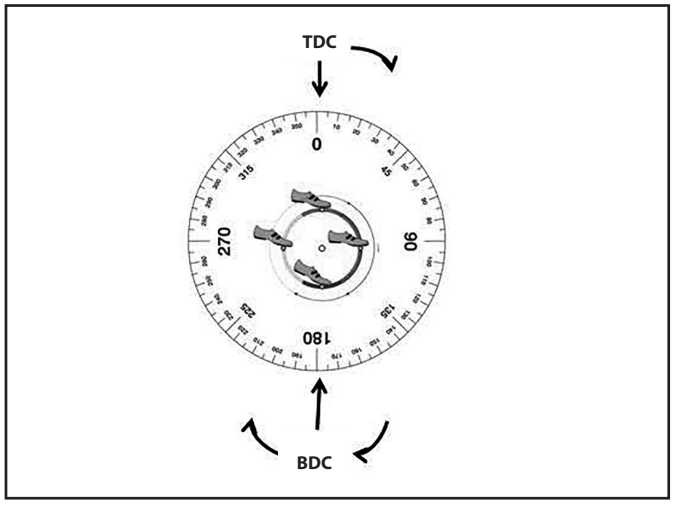



**Figure 3. Phases during the pedalling cycle.**




TDC: top dead centre; BDC: bottom dead centre.

area, the contraction speed, fibre types, distance between electrodes, positioning of electrodes, impedance of the skin, etc.[4,6].

## Muscle activation in pedalling and EMG normalisation

The pedalling movement has been studied exhaustively and 4 phases of pedalling have been classified7, (Figure 3), which generate propulsion and retrieval:

- *Phase I:* This goes from 20º to 145º in relation to the vertical line that passes through the axis of the pedal. During this phase the foot is extended 30º over the leg, the leg is stretched 70º, the muscle is stretched at an extent of 44º. The thigh extension is due to the action of the gluteus maximus muscles, to the tensor of the fascia lata, and to the hamstrings. The extension of the leg is due to the muscle action of the quadriceps by means of the vastus externus and the crural. The foot extension is performed using the sural triceps and also with the collaboration of the internal and external retromalleolar muscle groups. The intrinsic foot muscles do not have an apparent effect;
- *Phase II:* It goes from 145º to 215º. It is an inverse phase in which the lower limb moves from completing the extension to starting its bend. From 180º to 215º, the orientation of the foot remains similar to the previous phase (from 145 to 180º). A bending of the lower limb can be observed: the foot bends from 150º to 135º over the foot, the knee from 150º to 125º over the thigh, and this approaches 5º of the horizontal position;
- *Phase III:* This is the opposite phase to phase I. It goes from 215º to 325º; and
- *Phase IV:* From 325º to 20º, which would begin phase I again. The movements in this phase are complex. At the start of this phase, the foot is extended to 140º, flexing quickly again to 105º, however the extension changes of the knee and hip are minimal[7,8] (Figure 3).

The model of muscle activation can be analysed in terms of level of activity and/or muscle activation time. The level of muscle activity is identified through the simple motor response (SMR) during a complete cycle (0º-360º) or during the muscle activity period in the EMG bursts. An average is made of various consecutive pedalling cycles to obtain the enveloping curve[9-12].

EMG activity is expressed in relation to the record of a short isometric maximum voluntary contraction (IMVC) (<5 seconds)[13,14]. This method has been widely criticised as it cannot be used to represent the maximum neuronal conduction in pedalling[15]. Hautier *et al.*[16] observed an activity level over 100% of the IMVC. They have proposed new normalisation methods that may help improve the interpretation of the signals in future studies, but in general, and to date, there is no agreement regarding the best method to adopt[17]. De Luca[18] and Yang and Winter[19] indicate that it is more appropriate to take sub-maximum contractions rather than maximum ones as a reference, as above 80% of the MVC the signal is unstable and the reference is not reliable.

### Characterisation of the activation of the muscle models of the lower limb during pedalling

Cycling is a repetitive activity that uses coordinated combinations of the leg muscles to apply strength to the pedals. Muscle activity and coordination can vary between people during a single pedal cycle and between different cycles by the same person[20, 21]. On the other hand, coordination or the muscular activation of the leg affects the direction, magnitude and duration of the strength applied to the pedal, which is reflected in the mechanical work and power of the cyclist[22,23].

For the study of the muscle activation model, variables such as the "onset of muscle activity" and "offset of muscle activity" are important in the EMG register in terms of the angles of the bracket or the angle of the bicycle crankset[12].

Blake *et al*[24] indicate that global mechanic efficiency in cycling depends on the levels of activation, synchronisation and coordination in all of the active muscles of the leg, and not of any specific muscle. The time and coordination of muscle activation play an important role in the muscle activity used during the pedalling cycle. Various authors[10,12,13,24-26] have performed an EMG analysis of pedalling. Houtz and Fischer[26] performed it by testing the majority of the superficial musculature of the lower limb (except for the soleus) and affirm that they are activated in an ordered and coordinated way. Ericson[13] revealed that a workload of 120 W (approximately 54% of the maximum aerobic power in their work) induces an EMG activity at 45%, 44% and 43% of the IMVC for vastus medialis (VM) and soleus (SOL) respectively. Whilst for the biarticular muscles, such as the rectus femoris (RF) and gastrocnemius lateralis (GL), it was less, 22% and 18% respectively[13].

During the pedalling phase, the gluteus maximus (GMax) is activated from the top dead centre (TDC) up to approximately 130º, being within the region of the strike of power (25-160º) (Figure 3)[13,25,26]. The vastus lateralis and medialis (VL and VM) are activated from just before the TDC, up to just after 90º. The start of the rectus femoris (RF) activity is previous to that of the vastus (some 270º) and ends at almost 90º. The region of activity of the tibialis anterior (TA) is produced in the second half of the ascending phase (from the bottom dead centre (BDC) to the TDC) of almost 270º. The gastrocneumius (GAS) lateralis and medialis (GL and/or GM), depending on the study, start just after the activation of the TA ends (some 30º) and finish just before the start of the TA activity (approximately 270º). The soleus is activated during the descending phase (from 0º to 180º), specifically from 45º to 135º[13,25,26].

On the other hand, there is much controversy regarding the hamstring muscles, i.e. the femoral biceps (FB), semimembranosus (SM) and semitendinosus (ST). Dorel *et al*.[12] indicate a region of lesser activation, from just after the TDC to the BDC, compared to that shown by Jorge and Hull[25] of around the TDC at almost 270º. In fact, Ryan and Gregor[27] observed these two different models for the FB during pedalling. Dorel *et al*.[12] also observed two different activation models for TA, GL and SOL.

## Factors that may have an influence on the EMG models during pedalling

– *The output power* (in watts) can be modified by the rhythm of pedalling, the mechanical load (i.e. the resistance imposed by the cycle-ergometer) or both[9]. Ericson[13] observed an increase in the EMG activity of the main muscles of the lower extremities (GMax, VL, RF, VM, FB, ST and GM), in exercises of constant loads performed at different intensities. The output power increased from 120 to 240 W (at a rhythm of 60 rpm) and they proposed that the GMax activity is influenced by the level of workload. These results were confirmed by Sarre *et al*[28] on three knee extensor muscles (VM, VL and RF), at three different output powers, expressed as a percentage of the maximum aerobic power (60%, 80% and 100%). However, another study[25] revealed that at low intensities and with less difference between output powers (from 83 to 125 W), the EMG activity of the GAS does not appear to change. This was confirmed during a progressive pedalling exercise, in which the EMG activity of the GM did not vary until approximately 70% of the maximum aerobic power.

  Farina *et al*.[29], using a new method (eight electrodes in a linear arrangement) on two muscles of the lower extremities (VM and VL), demonstrated that the conduction speed of the muscle fibre increases in accordance with the load, producing a greater and progressive recruitment of the motor units with the conduction speed raised with the increase of strength.

– *Pedalling rhythm or frequency*. Various authors have quantified the level of EMG activity in the muscles of the lower extremity during different pedalling rhythms, accepting that it is an important factor that affects performance in cycling[13,14,29-32].

  In turn, Baum and Li[33] investigated the effects of the frequency and inertia of EMG activity on the muscles of the lower limb during different pedalling rhythms (60, 80 and 100 rpm) at a single output power of 250 W. All the muscles, except for the GAS, displayed significant differences at the onset of the activity in terms of the chain-set axis, and all apart from the SOL revealed a significant linear trend, as the start of the activity occurs earlier with the increase of rhythm. In terms of the offset of activity, GMax, RF, FB and VL displayed significant differences and there were differences in TA, GAS and SOL. With regards to the duration of the FB activity, this reduced, whilst that of the TA and RF increased. Furthermore, on an articular level, changes were observed at the start of the activity (29º GMax, 19º TA, 4º SOL) and at the end (20º GMax, 23º VL, 9º TA and 5º SOL), in that the change of speed of movement and the alteration of the inertia affect the activity and coordination of the lower limb muscles during the pedalling cycle[33,34].

– *The gradient and posture*. Pedalling on a gradient is also important in road cycling, as it will produce changes in the gravitational strengths and it is also accompanied by an alternate sitting and standing posture[9]. Li and Caldwell[30] were the first to study the EMG activity model in the muscles of the lower extremities in terms of gradient (0% to 8%), with no significant changes observed in the activation model. The result was later confirmed by Duc *et al*.[10] on gradients of 4%, 7% and 10%.

  Contrary to the gradient, the change in pedalling posture, from sitting to standing, affects the intensity and time of EMG activity of the main muscles involved[10,30]. Li and Caldwell[30] observed a major activation of the GMax, RF and TA, and a longer duration of GMax, RF and VL. These authors suggest that the greater and more sustained activation of the GMax was due to the fact that the position of the foot forces the pelvis to be stabilised as there is no support from the seat.

  Furthermore, Duc *et al*.[10] observed a greater activation and duration of EMG activity of the lower extremity muscles, with the exception of those that cross the ankle joint (GAS, SOL and TA). The duration

of the EMG activity of the GMax of the foot was greater and they supposed that this could be due to the lateral rolls. This data had already been observed by Li and Caldwell[30] with the exception of three muscles (GMax, RF and FB), which did not occur in the previous study.

In the study by Duc *et al.*[10] the RF revealed a significant increase in EMG activity in the second impulse phase (between 90º and 180º), whilst in the study by Li and Caldwell[30] this increase was less. They put this down to the need to increase the extensor moment in the foot position, in which the weaker single-joint knee extensors (VM and VL) may need the help of the RF to extend the knee with strength. Also, that the RF may act in synergy with the GMax to stabilise the pelvis.

In terms of the greater EMG activity of the FB observed by Duc *et al.*[10] regarding the study by Li and Cadwell[30], it could be due to the muscle coordination used by cyclists in pedalling standing up, associated with a specific pedalling technique. In some cases of FB, it is activated by the extension of the hip and the knee (during the descending phase 0º-180º) and in other cases the activity is associated with the bending of the hip and knee, starting much before 0º and stopping at approximately 130º. It could also be associated with the need to generate greater pushing force during the ascending phase of the pedal[35] or to help the GMax and RF to stabilise the pelvis[10].

Regarding the activity of the SM muscle, a reduction in the EMG activity was observed, whilst being the agonist of the FB, similar results would be expected. The hypothesis would be that this muscle would act more in the bending of the knee than in the extension of the hip[10]. The greater plantar flexor momentum in both studies during standing pedalling led to the hypothesis that by removing the support of the saddle, the gravitational forces on the pedal increase, with more weight falling on the pedal during the descending phase. The use of gravity along with fixing the ankle in a horizontal position enables this greater plantar flexor momentum without producing a change in the EMG activity of the flexors and extensors of the ankle[10].

- *Pedal-footwear interface.* Bicycle pedals have become a study focus, as this is the main energy transfer point between the cyclist and the bicycle. Today, the majority of professional and amateur cyclists use automatic pedals. Standard pedals enable the application of positive effective strength during the descending phase (TDC to the BDC), whilst automatic pedals also enable the application of positive effective strength from the BDC to the TDC[9].

  However, very few studies have focused on the study of the effects of the pedal-shoe interface and the muscle activation models of the lower limbs. Hug and Dorel[9] compared the level of EMG activity of the lower extremity muscles when automatic pedals were used, finding a greater level of activity in RF, FB and TA, and less in VM, VL and SOL. Other muscles were not affected (hamstring and gastrocnemius muscles, GMax)[13].

- *Level of training.* Professional cyclists reach distances of approximately 35,000 km/year, between competitions and training sessions, equivalent to 25 hours a week[31,32]. This has led to the considerations that muscle behaviour could be different in amateur and professional cyclists. In this respect, Hug and Dorel[9] have suggested that there are differences in the muscle recruitment models between professional and amateur cyclists. However, Marsh and Martin[14] did not discover significant differences in the electromyographic models of five muscles from the lower extremities (VL, RF, SOL and GM) between cyclists and non-cyclists with similar aerobic capacities.
- *Muscle fatigue.* The EMG studies carried out to date indicate that muscle fatigue can be studied and diagnosed based on the changes in the frequency spectrum[4,36]. In general, when the muscle is fatigued there is an increase in the low frequency components and a reduction of those of high frequency. The reduction of frequency may be due to a reduction in conduction speed, but also possibly to an increase of the synchronisation of motor units[4,36].

  During fatigue, increases in the width of the signal (RMS) are described, arguing that this is due to the increase of the recruitment of more motor units or to the increase in synchronisation of the already active motor units[4], in an effort to maintain the strength. This increase in width has been observed in the muscles of the lower limbs during strenuous pedalling exercises with a constant load[34].

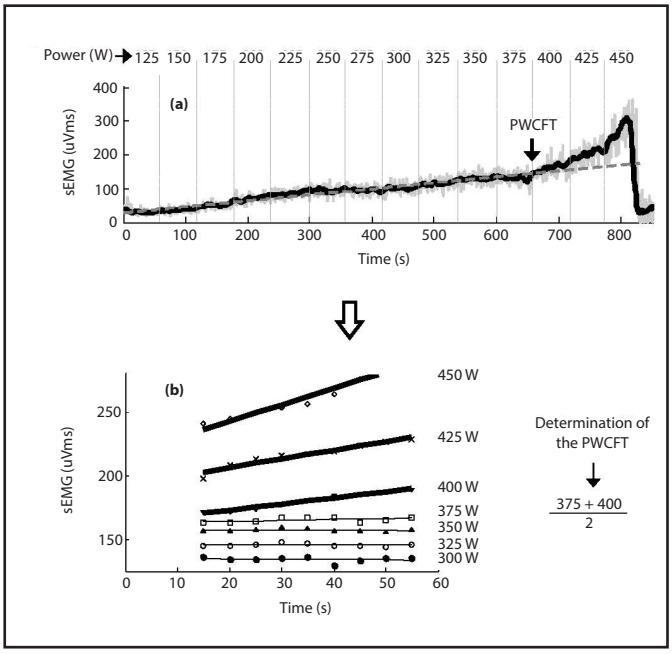
  When it is not possible to hold the contraction for more time, the force reduces and a reduction can be seen in the extent[36], probably caused by a reduction of the excitement of the motor units. If a truly reliable discrimination of fatigue were achieved, it could be used as a diagnostic method.

## Determination of the threshold of neuromuscular fatigue via EMG techniques

Over the past few decades, significant research efforts have been made in identifying the workload thresholds that enable the start of critical fatigue to be distinguished during incremental tests performed on a cycle-ergometer. Depending on the measurement variable used, there are different recognised fatigue thresholds, such as metabolic thresholds (point of accumulation of blood lactate) and ventilatory thresholds (aerobic ventilatory thresholds and point of respiratory compensation)[37,38]. However, as the exercise intensity increases, fatigue is not just related to the cardio-respiratory system, but also to the neuro-muscular system. Neuro-muscular fatigue can be identified based on the superficial electromyography measurement (sEMG) and its variations in time[39-41].

In previously performed research studies, deVries *et al.*[38,39] proposed a neuro-muscular fatigue threshold based on EMG activity defined as the "Physical Working Capacity and Fatigue Threshold" (PWCFT), via a specific test on a cyclo-ergometer. In their original version, the establishment of the PWCFT threshold was performed by examining the EMG-time curves obtained from 4 series of work carried out at 4 different power levels. The authors of this method identified the PWCFT threshold by determining

**Figure 4. Representative model of the method to stimulate the neuro-muscular fatigue threshold based on EMG (PWCFT) during an incremental test on an ergometer. (a) Time record of the sEMG obtained during the incremental test. The solid black line represents the averaged sEMG width for each 15 pedals. (b) Regression lines corresponding to the sEMG width vs time relationship for each power. The highest power with a non-significant gradient is of 375 W (P >0.05), whilst the lowest power with a significant gradient was 400 W (P<0,05). The PWCFT (387.5 W) is calculated as the average of these two powers.**




the greatest power level (load) that the cyclist could maintain over a 2-minute period without the EMG signal increasing significantly. In their original version, the deVries method had the major shortcoming of being a discontinued test that required the cyclist to pay various visits to the laboratory. Later, this group[41] refined its method, which enabled the researchers to extract the PWC threshold by performing a single incremental test.

The EMG signals are obtained using electrodes positioned on the dominant leg over the VL muscle at 1/3 of the distance between the lateral extreme of the kneecap and the projection of the hip bone[42].

During each phase (25w/min) of the incremental test, various consecutive segments of EMG are registered (each segment coinciding with the interval in which the muscle is active in a single pedal cycle). Normally the first 10-15 seconds of each stage of 1 minute are ruled out for analysis, as during this initial period the cyclist performs changes in posture to adapt to the new power. For each power level of the test, the sEMG width of each of the segments is calculated and represented depending on the time. Likewise, the lowest workload is identified (power), which generates a significant positive curve in the sEMG width/time relationship and also the highest workload (power), which generates an insignificant positive curve in the sEMG width/time relationship[37,41]. The PWCFT is established by getting the average of the two aforementioned powers (Figure 4).

However, despite the large amount of information that the EMG provides, we[42] have recently observed heterogeneous and irregular behaviour in the register of the superficial sEMG signal, fundamentally due to the effect that depends on the distance of the conductor of the muscle volume, the diaphony, the cancellation, the length of the muscle, the temperature and the lack of distinction of the fibre diameter via motor units with different recruitment thresholds. We have seen that the sEMG indications did not reduce significantly in the final periods of fatigue in the incremental test, as recently suggested.

## Conclusions

We think that EMG allows the level of muscle activation in any muscle to be detected, apart from deep muscle, where wired electrodes are needed (intra-muscular), and can be used as a diagnostic method to assess muscle fatigue. The clinical applications of EMG as a diagnostic tool can include not only fatigue, but also neuromuscular illnesses, assessment of lumbar pain, kinesiology and motor control disorders. The activation time of the lower limb muscles during pedalling can be discovered, and the influence that specific factors - such as the output power, pedalling rate, posture, pedal-footwear interface, and the level of training - may have on the activation time and muscle coordination of the main muscles in the lower limbs can be seen.

We consider that the EMG enables the detection of the appearance of muscle fatigue, whether of central or peripheral origin. In general an increase occurs in the amplitude to try and maintain strength and a reduction of the frequency spectrum.

## Bibliography


1. Boyas S, Guével A. Neuromuscular fatigue in healthy muscle: underlying factors and adaptation mechanisms. *Ann Phys Rehabil Med.* 2011;54:88-108.
2. Córdova A. Fatiga muscular en el rendimiento deportivo. Madrid. Ed. Síntesis, 1997. p. 193-231.
3. Enoka RM, Duchateau J. Translating Fatigue to Human Performance. *Med Sci Sports Exerc.* 2016;48:2228-38.
4. Villarroya MA. Electromiografía cinesiológica. *Rehab*. 2005;39:255-64.
5. Masso N, Rey F, Romero D, Gual G, Costa L, German A. Aplicaciones de la electromiografía de superficie en el deporte. *Apunts Med Esport.* 2010;45:127-36.
6. Konrad P. *The ABC of EMG: a practical introduction to kinesiological Electromyography.* USA: NORAXON I.N.C; Arizona. 2006. P. 9-15.
7. Haushalter G, Lang G. Biomecanique du pied du cycliste appliqueé au positionnement de la chaussure. *Med Sport (Fr).* 1985;58:136-7.
8. Gregor RJ, Broker JP, Ryan MM. The biomechanics of cycling. *Exerc Sport Sci Rev.* 1991;19:127-69.
9. Hug F, Dorel S. Electromyographic analysis of pedaling: a review. *J Electromyogr Kinesiol.* 2009;19:182-98.
10. Duc S, Bertucci W, Pernin JN, Grappe F. Muscular activity during uphill cycling: effect of slope, posture, hand grip position and constrained bicycle lateral sways. *J Electromyogr Kinesiol.* 2008;18:116-27.
11. Laplaud D, Hug F, Grelot L. Reproducibility of eight lower limb muscles activity level in the course of an incremental pedaling exercise. *J Electromyogr Kinesiol.* 2006;16:158-66.

12. Dorel S, Couturier A, Hug F. Intra-session repeatability of lower limb muscles activation pattern during pedaling. *J Electromyogr Kinesiol.* 2008;18:857-65.
13. Ericson M. On the biomechanics of cycling. A study of joint and muscle load during exercise on the bicycle ergometer. *Scand J Rehabil Med Suppl.* 1986;16:1-43.
14. Marsh AP, Martin PE. The relationship between cadence and lower extremity EMG in cyclists and noncyclists. *Med Sci Sport Exerc.* 1995;27:217-25.
15. Mirka GA. The quantification of EMG normalization error. *Ergonomics.* 1991;34:343-52.
16. Hautier CA, Arsac LM, Deghdegh K, Souquet J, Belli A, Lacour JR. Influence of fatigue on EMG/force ratio and cocontraction in cycling. *Med Sci Sport Exerc.* 2000;32:839-43.
17. Burden A, Bartlett R. Normalisation of EMG amplitude: an evaluation and comparison of old and new methods. *Med Eng Phys.* 1999;21:247-57.
18. De Luca C. The use of surface electromyography in biomechanics. *J Appl Biomech.* 1997;13:135-63.
19. Yang JF, Winter DA. Electromyography reliability in maximal and submaximal contractions. *Arch Phys Med Rehabil.* 1983;64:417-20.
20. Jammes Y, Arbogast S, Faucher M, Montmayeur A, Tagliarini F, Robinet C. Interindividual variability of surface EMG changes during cycling exercise in healthy humans. *Clin Physiol.* 2001;21:556-60.
21. Hug F, Bendahan D, Le Fur Y, Cozzone PJ, Grelot L. Heterogeneity of muscle recruitment pattern during pedaling in professional road cyclists: a magnetic resonance imaging and electromyography study. *Eur J Appl Physiol.* 2004;92:334-42.
22. Bigland-Ritchie B, Woods JJ. Integrated electromyogram and oxygen uptake during positive and negative work. *J Physiol.* 1976;260:267-77.
23. Arnaud S, Zattara-Hartmann MC, Tomei C, Jammes Y. Correlation between muscle metabolism and changes in M-wave and surface electromyogram: dynamic constant load leg exercise in untrained subjects. *Muscle Nerve.* 1997;20:1197-9.
24. Blake OM, Champoux Y, Wakeling JM. Muscle Coordination Patterns for Efficient Cycling. *Med. Sci. Sports Exerc.* 2012;44:926-38.
25. Jorge M, Hull ML. Analysis of EMG measurements during bicycle pedalling. *J Biomech.* 1986;19:683-94.
26. Houtz SJ, Fischer FJ. An analysis of muscle action and joint excursion during exercise on a stationary bicycle. *J Bone Joint Surg Am.* 1959;41:123-31.
27. Ryan MM, Gregor RJ. EMG profiles of lower extremity muscles during cycling at constant workload and cadence. *J Electromyogr Kinesiol.* 1992;2:69-80.
28. Sarre G, Lepers R, Maffiuletti N, Millet G, Martin A. Influence of cycling cadence on neuromuscular activity of the knee extensors in humans. *Eur J Appl Physiol.* 2003;88:476-9.
29. Farina D, Macaluso A, Ferguson RA, De Vito G. Effect of power, pedal rate, and force on average muscle fiber conduction velocity during cycling. *J Appl Physiol.* 2004;97:2035-41.
30. Li L, Caldwell GE. Muscle coordination in cycling: effect of surface incline and posture. *J Appl Physiol.* 1998;85:927-34.
31. Faria EW, Parker DL, Faria IE. The science of cycling: factors affecting performance – part 2. *Sport Med.* 2005a;35:313-37.
32. Faria EW, Parker DL, Faria IE. The science of cycling: physiology and training – part 1. *Sport Med.* 2005b;35:285-312.
33. Baum BS, Li L. Lower extremity muscle activities during cycling are influenced by load and frequency. *J Electromyogr Kinesiol.* 2003;13:181-90.
34. Sarre G, Lepers R. Neuromuscular function during prolonged pedalling exercise at different cadences. *Acta Physiol Scand.* 2005;185:321-8.
35. Neptune RR, Kautz SA, Hull ML. The effect of pedaling rate on coordination in cycling. *J Biomech.* 1997;30:1051-8.
36. Fernández JM, Acevedo RC, Tabering CB. Influencia de la fatiga muscular en la señal electromiográfica de músculos estimulados eléctricamente. *EIA.* 2007;7:111-19.
37. Lucía A, Sánchez O, Carvajal A, Chicharro JL. Analysis of the aerobic-anaerobic transition in elite cyclists during incremental exercise with the use of electromyography. *Br J Sports Med.* 1999;3:178-85.
38. deVries HA, Moritani T, Nagata A, Magnussen K. The relation between critical power and neuromuscular fatigue as estimated from electromyographic data. *Ergonomics.* 1982;25:783-91.
39. deVries HA, Tichy MW, Housh TJ, Smyth KD, Tichy AM, Housh DJ. A method for estimating physical working capacity at the fatigue threshold. *Ergonomics.* 1987;30:1195-204.
40. Camic CL, Housh TJ, Johnson GO, Hendrix CR, Zuniga JM, Mielke M. *et al.* An EMG frequency-based test for estimating the neuromuscular fatigue threshold during cycle ergometry. *Eur J Appl Physiol.* 2010;108:337-45.
41. deVries HA, Housh TJ, Johnson GO, Evans SA, Tharp GD, Housh DJ, *et al.* Factors affecting the estimation of physical working capacity at the fatigue threshold. *Ergonomics.* 1990;33:25-33.
42. Latasa I, Cordova A, Malanda A, Navallas J, Lavilla-Oiz A, Rodriguez-Falces J. Limitations of Spectral Electromyogramic Analysis to Determine the Onset of Neuromuscular Fatigue Threshold during Incremental Ergometer Cycling. *J Sports Sci Med.* 2016;15:148-57.

# Arrhythmogenic right ventricular cardiomyopathy. Prevention of sudden death in athletes

**Franc Peris, José Poveda, Diego Oliver, Luis Franco, Francisco J. Rubio, Alfredo Valero**

*Unidad de Medicina del Deporte. Hospital Universitario Sant Joan de Reus. Facultad de Medicina / Universidad Rovira y Virgili. Reus. Tarragona.*

**Received:** 24.02.2017
**Accepted:** 03.05.2017

**Summary**

**Introduction:** Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited disease characterized by progressive replacement of myocardial tissue and patched by fibrofatty tissue, which can cause ventricular arrhythmias and sudden cardiac death (SCD), even as a first manifestation. The manifestations of the disease are favoured by physical exercise, so it is one of the main causes of SCD in athletes under 35 years old.
**Material and method:** A systematic review in different scientific databases related to ARVC has been made. The initial research on 938 publications was eventually reduced to 36, after applying the different criteria of inclusion and exclusion.
**Results:** In our environment, medical history, physical examination and electrocardiogram (ECG) are the main tools used in the screening of SCD from 12 years of age (basic cardiovascular evaluation). However, in professional or high risk athletes, echocardiography and maximal exercise test are added to the initial screening (advanced cardiovascular assessment). Still, the gold standard test for the diagnosis of ARVC is cardiac magnetic resonance (RMC). The genetic test plays an important role in the study of suspected patients as well as in the evaluation of the relatives of patients who have already been diagnosed. ARVC treatment involves the use of antiarrhythmic drugs, implantation of an implantable cardioverter defibrillator (DAI) based on the risk of SCD and restriction of physical activity.
**Discussion:** The lack of standardized studies on large populations of athletes and the absence of sudden death registries difficult to obtain solid evidence in the interpretation of the results of the reviewed articles.
Conclusions: The preparticipative screening of all athletes should include medical history, complete physical examination and 12-lead ECG; considering running an echocardiography being highly recommended.

**Key words:**
Sudden death. Arrhythmogenic cardiomyopathy (ARVC). Sport. Physical exercise.

## Prevención de la muerte súbita por miocardiopatía arritmogénica del ventrículo derecho en deportistas

**Resumen**

**Introducción:** La miocardiopatía arritmogénica del ventrículo derecho (ARVC) es una enfermedad hereditaria caracterizada por la sustitución progresiva y parcheada del tejido miocárdico por tejido fibroadiposo, lo cual puede originar arritmias ventriculares y muerte súbita (SCD), incluso como primera manifestación. Las manifestaciones de la enfermedad se ven favorecidas por el ejercicio físico, siendo una de las principales causas de SCD en deportistas menores de 35 años.
**Material y método:** Se ha realizado una revisión sistemática en las diferentes bases de datos científicas relacionada con la ARVC. La búsqueda inicial de 938 artículos se redujo finalmente a 36, tras aplicar los diferentes criterios de inclusión y exclusión.
**Resultados:** En nuestro medio, la historia clínica, la exploración física y el electrocardiograma (ECG) son las principales herramientas usadas en la prevención de la SCD a partir de los 12 años de edad (evaluación cardiovascular básica). En deportistas profesionales o con alto riesgo, se añade ecocardiografía y prueba de esfuerzo máxima (evaluación cardiovascular avanzada). Aun así, la prueba de elección para el diagnóstico de ARVC es la resonancia magnética cardiaca (RMC). El test genético juega un papel importante tanto en el estudio de pacientes sospechosos como en la evaluación de los familiares de pacientes ya diagnosticados. El tratamiento de la ARVC consiste en el uso de fármacos antiarrítmicos, la implantación de un desfibrilador automático implantable (DAI) en función del riesgo de SCD y la restricción de la actividad física.
**Discusión:** La falta de estudios estandarizados de grandes poblaciones de deportistas y la ausencia de registros de muerte súbita dificultan la obtención de una evidencia sólida en la interpretación de los resultados de los artículos revisados.
**Conclusiones:** El screening preparticipativo a todos los deportistas debería incluir: historia clínica, exploración física completa y ECG de 12 derivaciones; considerándose altamente recomendable la realización de una ecocardiografía.

**Palabras clave:**
Muerte súbita. Miocardiopatía arritmogénica del ventrículo derecho (ARVC). Deporte. Ejercicio físico.

**Correspondence:** Luis Franco Bonafonte
E-mail: lfranco@grupsagessa.com

## Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a hereditary illness characterised by the progressive loss of myocytes in the right ventricle (RV), causing a segmental or diffuse thinning of the wall[1]. The left ventricle (LV) may also be affected, with bi-ventricular progression being the most common form of the illness[2]. In Spain and Europe[3], ARVC is one of the main causes of SCD in athletes under the age of 35 years, though there is a lack of large-scale studies of incidence rates (Figure 1).

ARVC tends to arise clinically between the third and fifth decade of life, with ventricular arrhythmias that may cause sudden cardiac death (SCD)[4]. Clinical manifestations and the progression of the illness are very variable. In the initial phase, the structural changes may be absent or subtly confined to localised areas of the RV, with the most common being: the front part of the infundibulum, the apex and the infero-basal tricuspid area of the RV, which make up the so-called *triangle of dysplasia*, considered to be the identification mark of ARVC. With the onset of the illness, both ventricles may be affected with cavity dilation and even aneurysms.

In ARVC, a defect occurs in the cellular adhesion proteins such as plakoglobin, plakophilin-2, desmoplakin, desmocollin-2 and desmoglein, and in others responsible for cell stability[3]. This protein alteration causes alterations of the structure and function of the myocardial wall with the replacement of fibrofatty tissue, as shown by Saberniak *et al*[5].

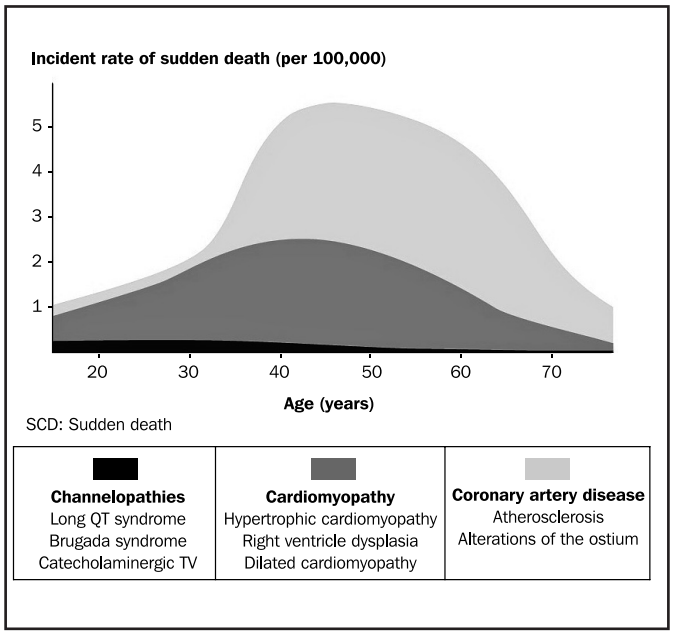
In accordance with the *American Heart Association* (AHA), the diagnosis is made based on the presence of greater and lesser criteria reviewed in the *Task Force Criteria*[6] (Table 1). The criteria are based on findings in six different diagnostic categories: Basal ECG, signal-averaged EGC and Holter; endomyocardial biopsy; family history; and advanced cardiac image tests. Depending on the points scored, the patients are classified into possible, borderline or definite ARVC.

Intense physical exercise may trigger arrhythmic episodes and sudden death, as the first sign of the illness in athletes that have diagnosed ARVC or not. The mechanical stress to which the heart is subjected produces an increase of sympathetic stimulation, which can explain the ventricular arrhythmias that appear in the illness. Furthermore, it has been shown that stress maintained over time causes greater ventricular dysfunction in athletes suffering from ARVC than in non-athletes.

Despite the incidence rate of the illness being low, it is still one of the most frequent causes of SCD in young athletes, on the other hand it has a major social impact as the episodes occur in apparently healthy people, on occasions well-known people, that die unexpectedly whilst performing sport.

It is also worth highlighting its broad clinical spectrum and its difficult diagnosis, for which it is not clear which protocol should be applied routinely for its diagnosis in athletes, thus preventing the SCD. There are authors that defend the need for a greater number of initial studies to rule it out, whilst others give greater importance to the cost-effectiveness relationship. In this review the diagnostic methods and treatment of ARVC described in the current bibliography are analysed, with the aim of clarifying a suitable protocol for the screening of ARVC and finding ways of preventing SDC in athletes with this pathology.

**Figure 1. Changes in the incidence and aetiology of the SCD depending on age[25].**




## Material and method

### Design

A systematic review was performed of ARVC-related articles from scientific societies, as well as clinical practice guides and systematic reviews.

### Search strategy and exclusion criteria

Firstly, a search was performed on Google Scholar and Pubmed for articles and clinical practice guides published by different European and American societies on ARVC, SCD and their relationship with sport. Next a search was performed of clinical practice guides and of systematic reviews of scientific literature in Pubmed using the search terms: *(arvd OR arvc) AND "sudden death"; (arvd OR arvc) AND "exercise"; (arvd OR arvc) AND "screening";* previously obtained from the MeSH Database. The search was restricted to articles published within the last 5 years in English and in Spanish. The studies had to provide significant data about the diagnostic tests of ARVC, or be related to the different methods that are useful in preventing SCD caused by ARVC in athletes < 35 years. The publications that did not refer to the object of our study were excluded.

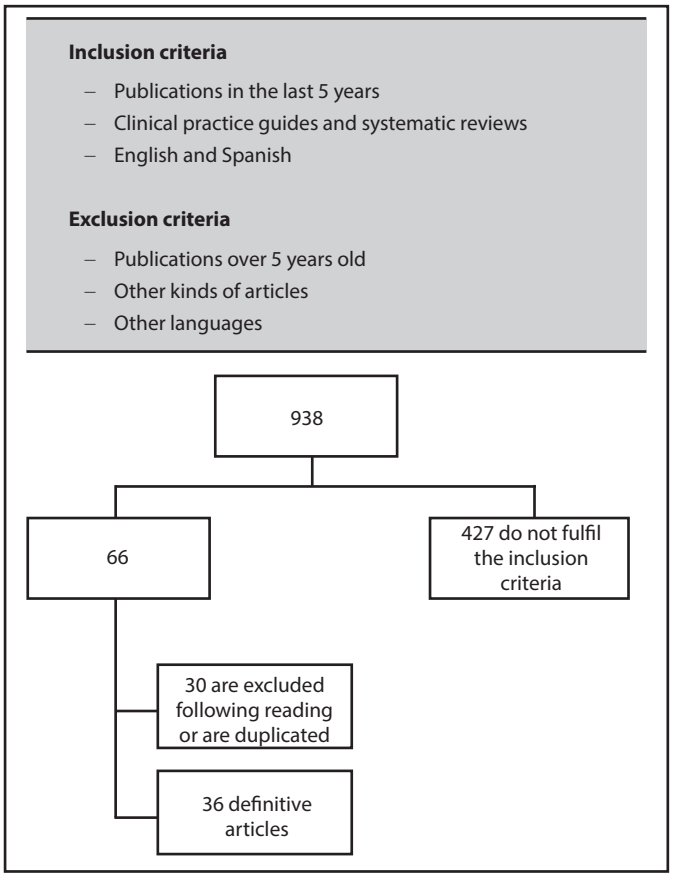
*Data extraction:* After performing the initial search on Pubmed with the term (*arvd* OR *arvc*) AND *"sudden death"*, 593 articles were found, limi-

**Table 1. Diagnostic criteria of ARVC/D reviewed Task Force 2010 AHA (Modified Ref.17).**

| I. Regional or global dysfunction and structural alterations | IV. Repolarisation / circulation abnormalities |
|---|---|
| **Greater**<br>**2D ECHO**<br>• Akinesia, dyskinesia or regional aneurysm of RV<br>• and 1 of the following (end-diastole):<br>- PLA: Flow of RV outflow tract ≥32 mm (corrected by BA ≥19 mm/m²)<br>- PLA: Flow of RV outflow tract ≥36 mm (corrected by BA ≥21 mm/m²)<br>- or Ejection Fraction ≤33%<br>**CRM**<br>• Akinesia, dyskinesia or regional disynchrony of RV<br>• and 1 of the following:<br>- End diastolic volume ratio RV/BA ≥110 ml/m² (Men) o ≥100 (Women)<br>- or Ejection Fraction of RV ≤40%<br>**RV angiography**<br>• Akinesia, dyskinesia or regional aneurysm of RV | **Greater**<br>• Y-wave in right precordial leads (V1-V3). Replicable in low-amplitude signals between the end of QRS and the start of the T-wave |
| **Lesser**<br>**2D ECHO**<br>• Akinesia, dyskinesia or regional dyskinesia of RV<br>• and 1 of the following (end-diastole):<br>- PLA: Flow of RV outflow tract ≥29 mm (corrected by BA ≥ 16 to <19 mm/m²)<br>- PLA: Flow of RV outflow tract ≥36 mm (corrected by BA ≥21 mm/m²)<br>- or Ejection Fraction >33% to ≤40%<br>**CMR**<br>• Akinesia, dyskinesia or regional disynchrony of RV<br>• and 1 of the following:<br>- Ratio of end diastolic volume of RV/ BA ≥ 100 ml/m² (Men) or 90 to < 100 (Women)<br>- or Ejection Fraction of RV >40% to ≤45% | **Lesser**<br>• Potential delays in SAECG in ≥1 parameter in the absence of QRS ≥110 ms in standard ECG<br>• Duration of filtered QRS (fQRS) ≥114 ms<br>• Duration of terminal QRS <40 µV (low-amplitude signal) >38 ms<br>• Square root of the terminal voltage 40 µV ≤20 ms<br>• Duration of the terminal activation of QRS ≥55ms, measured from the nadir of the S-wave to the end of the QRS including R' in V1, V2 or V3, in the absence of complete RBBB (Figure 3) |
| **II. Tissue characteristics of the wall** | **V. Arrhythmias** |
| **Greater**<br>• Residual myocytes >60% in morphometric analysis (or >50% if it is estimated) with fibrous replacement of the free wall of the RV in ≥1 sample or with or without replacement of endomyocardial fatty tissue in biopsy | **Greater**<br>• Sustained or non-sustained tachycardias with BBB morphology with superior axis (negative or undetermined QRS in II, III and in AVF, and positive in AVL) |
| **Lesser**<br>• Residual myocytes 60-75% in morphometric analysis (or 50-60% if it is estimated) with fibrous replacement of the free wall of the RV in ≥1 sample or with or without replacement of endomyocardial fatty tissue in biopsy | **Lesser**<br>• Sustained or non-sustained tachycardias with origin in the RV outflow tract, with BBB morphology with inferior axis (positive QRS in II, III and in AVF, and negative in AVL)<br>• >500 ventricular extrasystoles in 24 hours (Holter) |
| **III. Anomalies in repolarisation** | **VI. Family history** |
| **Greater**<br>• Inverted T-waves in right precordial leads (V1-V2) in individuals of >14 years in absence of complete RBBB (QRS ≥120 ms) | **Greater**<br>• ARVC confirmed in first-degree relative that fulfils the TF criteria<br>• AVRC confirmed by biopsy in 1st degree family member<br>• Identification of mutation categorised as associated or probably associated with ARVC in assessed patients |
| **Lesser**<br>• Inverted T-waves in right precordial leads (V1-V2) in individuals of >14 years in absence of complete RBBB (QRS ≥120 ms), or in V4, V5 or V6<br>• Inverted T-waves in V1, V2, V3 y V4 in individuals of >14 years in presence of complete RBBB. | **Lesser**<br>• ARVC confirmed in first-degree relative in which it is not possible or practical to establish if the relative fulfils the TF criteria<br>• Premature sudden death (<35 years) due to suspected ARVC, of a first-degree family member<br>• ARVC confirmed in pathological analysis or that fulfils the criteria of the TF in 2nd degree family member |

PLA: Parasternal Long Axis; BA: Body Surface Area; SAECG: Signal of Averaged ECG.
Definitive Diagnosis: 2 greater criteria or 1 greater and 2 lesser or 4 lesser from different categories; Borderline: 1 greater and 1 lesser or 3 lesser from different categories; Possible: 1 greater and 2 lesser from different categories. Pathogenic mutation: alteration of the DNA associated with ARVC that alters or is expected to alter the codified protein. It is not observed or it is unusual to observe it in a no-ARVC control, and it alters or is expected to alter the structure or function of the protein, or its link to the phenotype of the illness in a conclusive pedigree has been proven

**Figure 2. Inclusion and exclusion criteria and diagram of article selection.**




ted to 184 within the past 5 years. By only selecting the clinical practice guides and systematic reviews, a total of 48 publications were obtained, with 40 remaining upon selecting English and Spanish languages. With the search term *(arvd OR arvc) AND "exercise"*, the initial number of 188 articles was reduced to 15 after applying the previously mentioned filters. Finally, with the formula of *(arvd OR arvc) AND "screening"* 157 were obtained, of which 11 remained after restricting the search.

Once 3 initial search equations were performed with the corresponding filters, a total of 66 articles remained, of which, after eliminating duplicates and excluding those that did not refer to the subject of our review, a total of 36 remained (Figure 2). To these, 5 general references were added about sudden death in sport.

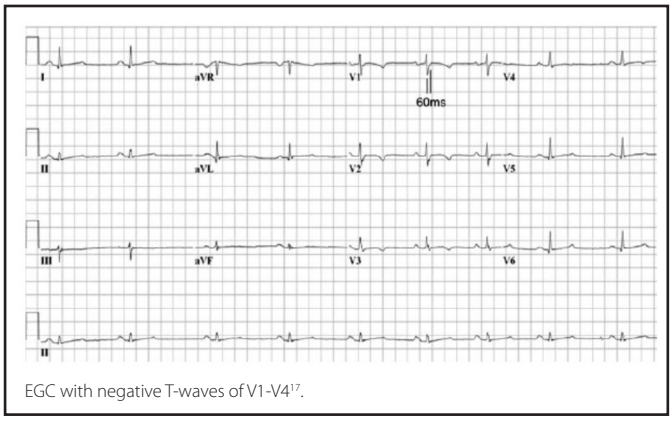
# Results

## Diagnostic tests

### *Basal ECG*

The ECG is the initial ARVC diagnostic method. The most common sign is T-wave inversion in the precordial leads $V_1$-$V_3$.[7-11] (Figure 3) and it forms part of the early diagnosis of the illness[7]. In the absence of a right-bundle branch block, it is considered a greater diagnostic criteria in those over the age of 14 years. In minors below this age, it may be a variety of normality. The presence of T-wave inversion in more than three precordial leads is a predictor of arrhythmic events. However, in 12% of patients the ECG is normal[12]. If ARVC is suspected with a normal ECG, a series of ECG must be carried out[13].

**Figure 3. ECG of patient with ARVC.**




EGC with negative T-waves of V1-V4[17].

In severe phases, low amplitude signals may appear, defined as epsilon waves in $V_1$-$V_3$, which are also considered a greater diagnostic criteria[3,6-8]. The appearance of TV with morphology of the left bundle branch block is also characteristic6. The duration of the QRS of more than 120 ms is associated with ARVC[6-8,11,14]. These findings are highly sensitive (88-100%) but they have low specificity (46%), with the PPV being 61% and the NPV being 91-100%[6]. The application of the Seattle criteria gives over 97% sensitivity to the screening of athletes via ECG[15].

### *Signal-averaged ECG*

It contains the average of multiple QRS to filter noise and late potentials. The QRS duration of more than 114 ms, the terminal QRS duration of more than 38 ms, and a terminal voltage of less than 20 microV are considered abnormal. If any of these values are present, it is considered a lesser diagnostic criteria.

### *Holter monitoring (24 hours)*

It is used in the diagnosis of ARVD, but especially, in its follow-up. A lesser criteria is considered to be less than 500 premature ventricular contractions in 24 hours or more than 30% of ventricular extrasystoles of the total heartbeats on the electrocardiographic record[3].

### *Echocardiography*

Echocardiography is the most used image modality in the diagnosis and follow-up of ARVC[7]. The most common finding is the lengthening of the RV outflow tract[3,6,16]. A tract >30 mm has a diagnostic sensitivity of 89% and a specificity of 86%[16]. Ventricular dysfunction is also habitual, defined as an EF <32% and the abnormalities in the mobility of the wall[3,7,16]. The presence of akinesia, dyskinesia or aneurysm are also

considered greater criteria[12]. The extension of the hypokinesia, along with the measurement of the end diastolic volume, can give us an idea of the evolution of the illness[16].

### *Cardiac magnetic resonance*

CMR is the test of choice as it provides information about the degree of fibrofatty replacement of the structure and of the ventricular function[3,7,17,18]. In comparison with the echocardiograph, it enables a better assessment of the previously mentioned characteristics[15,17-19]. The most common alterations are ventricular dilation (S77% and E95%), systolic dysfunction, dilation of the outflow tract, regional abnormalities in the wall movement, ventricular aneurysms, the presence of intramyocardial fat and late gadolinium enhancement[7,16,18].

The characterisation of the tissues is performed by assessing the images weighted in T2, indicative of recent myocardial injury (oedema); and by the characterisation of the late gadolinium enhancement, characteristic of irreversible tissue injury (scar). The percentage of the latter has been shown to be a powerful factor in the prediction of TV and of SCD caused by ARVC[20]. The CMR was positive in 100% of the patients diagnosed with ARVC, but it obtained a specificity of 29% due to the over-interpretation of the regional abnormalities of the movement[16]. The addition of quantitative parameters manages to raise specificity to up to 90-98%[12].

It is the ideal follow-up method in asymptomatic patients and is highly valuable for assessing symptomatic athletes[9,15]. However, there are inconveniences in terms of availability, cost, the need for experts to interpret the images and the tendency to over-diagnose[3,7,13,18]. This is why, despite its numerous advantages, it cannot be applied to the screening of large groups of athletes[15].

### *Computed tomography*

The CT is an alternative for patients for whom the CMR cannot be used. In comparison to the CMR, the CT does not have resolution in the characterisation of the tissues and tends to over-estimate the final systolic and diastolic volume.

### *Electrophysiology*

3D electro-anatomical mapping is a recent technique and is very sensitive when detecting areas of low voltage, which would indicate the loss of electrically active myocardium and their substitution with fibrofatty tissue, characteristic of this pathology[7,18,21]. If the signs of low voltage are present in the RV, this may help locate the area to perform a biopsy[6]. This technique has allowed for a greater understanding of the illness and for the assessment of the distribution of lesions, with the discovery that they are more frequent in the epicardium[21].

As it is an invasive test, it will only be performed in patients with suspected ARVC and ventricular arrhythmias of the RV, as well as for the differential diagnosis with idiopathic RV tachycardia[21]. In fact, some authors still consider it to be an experimental technique[3,7,13]. It is more sensitive than the CMR in cases in which the heart wall is thin, but it can cause confusion with other pathologies such as sarcoidosis[7,18].

### *Heart biopsy*

In the typical ARVC biopsy, the myocytes are replaced by fibrofatty tissue, especially in the heart apex and the ventricular outflow tract, whether or not associated with atrophy of the ventricular myocardium[13,17]. The diagnosis can be made without the presence of fibrous tissue, as long as the infiltrated adipose is accompanied by inflammation and necrosis typical of myocytes[22]. Routine biopsies are taken from the interventricular septum, though the sample may provide a false negative as the process of the illness is frequently patchy[3,9,19,22]. Furthermore, the progression of the lesions is from sub-epicardium to endocardium, which is why the endocardial biopsy may not be positive in early stages. On the other hand, there is a major risk of perforation if it is performed on the free wall of the ventricle[3,7].

The diagnostic performance of the heart biopsy increases if it is carried out alongside electro-anatomical mapping; the sample should be collected from the affected area (low voltage)[7,12,13]. The immunohistochemical analysis reveals a reduction of the plakoglobin signal in the affected myocytes. It is considered to be a highly sensitive (91%) and specific (82%) diagnosis technique, but it is a tool that is still being researched due to its high rate of false positives and negatives (sarcoidosis and myocarditis of giant cells), which is why it is not usually performed today[7,9,21].

### *Clinical and genetic diagnostic criteria*

The diagnosis of ARVC is based on the application of greater and lesser diagnostic criteria. Depending on the points scored, the patients are classified into possible, borderline or definite ARVC. Applying the diagnostic criteria updated by the AHA in the 2010 TFC, different publications have revealed an increase in sensitivity from 57 to 71% ($p = 0.001$), identifying 25 patients with defined ARVC and 64 with probable ARVC[14]. However, these criteria have limitations as they do not include cases with dominant left affection of the illness[7,19]. Infradiagnosis is probable due to the complexity of the cases and the existence of unknown variants[23].

The inclusion of genetic criteria was one of the changes made in the TFC. All patients with suspected or diagnosed ARVC must be genetically analysed, and in the event that they are positive, their family members must also be analysed[21,24,25]. The PKP-2 genes (plakophilin-2) located in the chromosome 12p11.21, and the DSG2 and DSP genes are responsible for over half of the cases of SCD due to ARVC. The mutation of the PKP2 gene is the most prevalent[26]. It follows an autosomal dominant inheritance model in the majority of cases[19]. Models of recessive autosomal inheritance have been described, linked to proteins such as plakoglobin[2]. In patients without a PKP2 mutation, additional tests in DSC2, DSG2, and DSP can identify a genetic cause in 5% to 10% of cases. When the participation of the LV is predominant in a family line, genetic tests for DSP, DSG2 and mutations of the transmembrane protein TMEM43 should be included in the first round of tests[27].

Of all the cases diagnosed using the TFC criteria, only 30-50% presented desmosomal anomalies, and of these, around 10% had more than one known mutation, a fact that has been associated to its greater severity[6,7,14,21]. Approximately 50% of mutations are unknown[2,8,14,19,28] and 16% of healthy controls revealed mutations characteristic of ARVC[19,21].

The interpretation of genetic tests is difficult due to their heterozygosity. Furthermore, the prognosis of ARVC according to the mutation is limited due to a lack of data[29]. Genetic diagnosis is considered a greater criteria, though the absence of identifiable mutations does not exclude the illness. It is most useful in family screening, in which asymptomatic carriers will require life-long monitoring, whilst non-carriers will not require serialised checks[9]. On the other hand, the genetic study of patients with diagnosed ARVC may be useful in establishing the importance and behaviour of the genes in the illness. The test should be considered for patients with possible ARVC (Recommendation level IIb) and is not recommendable for individuals with just one lesser criteria (Recommendation level III)[13].

First degree family members are considered to have a 50% risk of carrying the illness causing mutation with variable penetrance. Genetic testing is recommended for these patients (Level of evidence I)[3,13]. The follow-up of asymptomatic patients carrying a mutation is recommended from 10 to 50 years of age. This should be carried out with a complete screening every 1-2 years (history, ECG, echocardiograph, Holter, CMR)[19]. The clinical screening of family members of unexplained sudden death victims can identify ARVC in 7-9% of cases[19]. In second degree family members, the study is usually limited to an ECG, an echocardiograph and a Holter every 5 years[19].

## Treatment

The handling of these patients must be orientated towards avoiding the appearance of arrhythmias that trigger syncopes or cardiac arrest. Patients with ventricular failure or ventricular tachycardias will be considered high risk and will receive a more aggressive treatment[13]. Currently, the majority of patients are recommended the implantation of an ICD as primary prevention, and the consumption of anti-arrhythmic drugs, or the excision via catheter of accessory circulation pathways to reduce the arrhythmic impact. Heart transplant could be recommended in terminal stages of heart failure or maintained ventricular tachycardias[10, 30].

### Drugs

Beta blockers constitute the general treatment of these patients, despite their use being merely empirical[8,31,32]. As highlighted by Kantor *et al*[31], they are mainly used in the initial treatment of moderate or severe systolic failure of the LV. Within beta blockers, the most used in these patients is sotalol[6,7,12,13,31,33]. This pharmaceutical drug is also recommended to prevent electric shocks in patients fitted with an ICD[6,33], and is considered to be the only drug that reduces the risk, especially risk induced by exercise. The prescription of this drug is a prior requirement to the implantation of an ICD or to the excision via catheter in patients with ARVC[9,34]. However, some studies[13,34] did not show that beta blockers reduce the rate of TV in different series assessed. Another area of discrepancy radiates in the greater effectiveness of amiodarone compared to sotalol[7,13,24], relegating the latter to second place[34].

### Implantable cardioverter defibrillator

The ICD is considered the most effective treatment in preventing sudden death, as it converts the VF into a sinus rhythm. It is therefore the fundamental pillar in preventing SCD in patients with ARVC[7,33,35,36]. As indicated by Schinkel *et al.*[35] and Saguner *et al.*[7], multiple studies have revealed that the ICD is effective in both primary and secondary prevention, despite not being exempt of risks. The complications associated with its implantation are a source of fatality in these patients. It is considered that complications appear in 58% of those implanted after 7 years, mainly related to the catheter[14]. Another problem with the ICD is that of inappropriate electrical shocks, though these shocks are appropriate in 94% of cases of TV[33].

The AHA recommends the implantation of an ICD (recommendation level IIA) to prevent SCD in patients with extensive illness, TV or sustained ventricular fibrillations, LV affection and patients with more than one family member with reanimated sudden death or unexplained syncope, as long as they are receiving optimum medical therapy and have a reasonable life expectancy[7,12,35].

The stratification of the risk continues to be the main challenge for the indication of ICD[21]. In accordance with international guidelines and expert consensus, the indications of the ICD in ARVC patients are well-established for high-risk patients with a history of SCD or episodes of sustained ventricular tachycardia (recommendation level IB), whilst the presence of unknown origin syncope, non-sustained ventricular tachycardia, a family history of sudden death, extensive illness including LV affection, are considered possible indications of ICD with an intermediate risk of SCD (Recommendation Level IIA, evidence level C)[31,32].

### Excision via catheter

The excision of nerve channels via catheterisation has been considered a useful treatment for patients with poorly controlled ARVC. Excision via catheter cannot substitute ICD, but it can be useful in patients that already have one, in people resistant to medical treatment, in recurring monomorphic TV or in final stages with incessant ventricular tachycardias with palliative purposes[8,21,34]. It has been proven that better results are obtained if it is combined with electro-anatomical mapping to visualise the circulation pathways[24]. However, there is a high re-occurrence rate in the form of TV in the first 5 years of follow-up[14]. The application of current epicardium techniques along with endocardium techniques, has improved the success rate of eliminating TV, by not limiting the excision solely to the endocardiac lesion, less frequent in these patients[6,7,12]. This way, recurrence rates reduce by up to 30%[12].

## Molecular therapy

A promising treatment for the future is the reprogramming of somatic cells in pluripotent cells. Skin fibroblasts can generate autologous cardiomyocytes, which can be implanted in a heart with ARVC[36].

## Heart transplant

This is mainly used in the final stages, when the ARVC has progressed to extensive heart failure[13], or when faced with refractory disease to the medical treatment or other techniques[7,12]. It is not a frequent treatment, with an average age of 40 years and an 88% survival rate at 4.5 years[9].

## Restriction of physical activity

The restriction of intense exercise is considered to be a hugely important treatment, as it has been demonstrated that exercise, especially when intense, increases the possibility of ARVC appearing, and is associated with a lower survival rate[9]. Competing athletes aged under 35 years with ARVC face a 5.4 times greater risk of SCD than non-athletes[2,30,34]. Recommendations from Lavine *et al.*[1] indicate that any exercise should be avoided apart from those of IA class, and in general any activity greater or equal to 4 METS. Although there are no studies on the progression rate of the illness, restricting exercise is considered the most important factor[9,19].

The limitation of physical activity is also recommended for family members of those affected by ARVC that have the mutation, whilst there is discrepancy in family members in which the genetic screening is not possible or in which no mutation has been identified. In this situation, an individualised series of assessments is recommended, mainly if they are high-level athletes[37]. Exercise and lifestyle recommendations should be given with the consensus of the patient and the family. The psychological impact of the diagnosis and the treatment, along with recommendations, poses a challenge for patients[32].

## Prognostic factors

The possibility of SCD in patients with ARVC can be favoured by the presence of the following factors: Family history of SCD; Early onset of the illness[7]; Malignant arrhythmias or spontaneous or inducible unstable TV[34]; Heart attack or syncope, especially in stress[7], as it reflects the existence of malignant arrhythmias; Serious dysfunction of the RV (EF < 50% or thinning or aneurysm of the wall); QRS dispersion >50 ms (Increase of the QRS dispersion is considered to be from 40 ms and is the strongest independent predictor, with a sensitivity of 90% and a specificity of 77% according to Cadrin-Tourigny *et al*[34]); and the failure of anti-arrhythmic pharmaceuticals[2,34]. Furthermore, patients with the combination of spontaneous sustained TV, signs of right heart insufficiency and/or LV dysfunction are classified as being at the greatest risk of sudden death and have the worst long-term outlook[14].

On the other hand, for patients with an implanted ICD, the main risk factors related to secondary prevention are an aborted SCD and unstable TV, the presence of which implies a 10% annual risk of fatal TV[34].

### *Screening*

Along with the antecedents, the resting ECG is the foundation for prevention of SCD in sports. When a basal ECG detects negative T-waves of $V_1$ to $V_3$, the study should be completed with an echocardiograph and a maximum stress test. Depending on the result of these tests, the study can be continued with CMR, with late gadolinium enhancement (LGE) and 24-hour Holter. Also a 12-lead ECG and echocardiograph of first-degree family members under the age of 10 years[38].

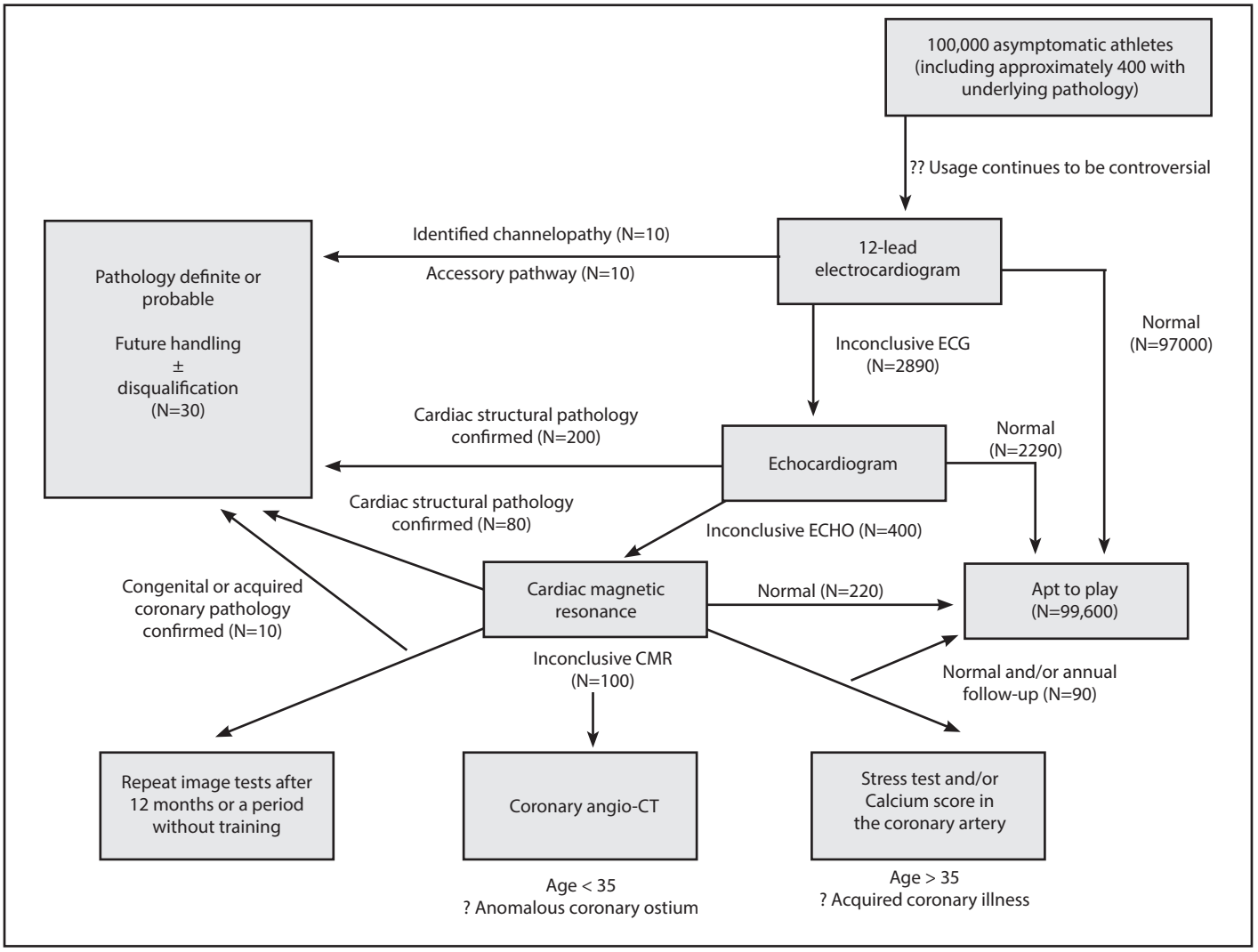
The European Sports Medicine Society, the European Society of Cardiology (ESC), the International Olympic Committee and FIFA recommend the carrying out of screening tests on athletes to prevent SCD, orientated at discovering the most frequent pathologies in athletes under 35 years. In the case of the ARVC, it is complex and difficult to perform, due to the low prevalence of the illness, the high cost of implementation and possible false positives. However, the success of screening and preventive measures in reducing SCD in athletes with ARVC has been proven. In the region of Venetto (Italy), screening with clinical history, physical examination and annual ECGs, and in high-risk cases, the limitation of sport, are factors that have led to an 89% reduction in ARVC-based SCD[16].

The ESC recommends screening of all athletes before starting intensive sporting activity to prevent cardiovascular-origin SCD. Basic cardiovascular assessment includes all federated athletes, is based on anamnesis, a physical examination and a 12-lead ECG, and should be carried out every two years. In turn, advanced cardiovascular assessment, within that found in high-risk athletes and professionals, requires the addition of the echocardiograph and the maximum stress test to the previous tests, and should be performed annually[39]. However, the AHA only recommends a clinical-family history and a physical examination. This discrepancy could be due to the greater weight of cost-efficiency issues in the United States in terms of healthcare. Despite this, and in contrary to most algorithms, different sporting bodies such as FIFA or the NBA place image tests in the initial screening, with the aim of ruling out heart anomalies, aortopathies or some cardiomyopathies with normal ECG readings[15] (Figure 4).

In terms of the age for starting the tests, the screening of patients with ARVC should be performed at around 12 years, given that if it is later, some cases of SCD may appear or the illness may progress[16,19]. On the other hand, initial assessment is recommended for all patients with suspected ARVC with a physical examination, clinical history, family antecedents with clinically compatible cases, ECG, signal-averaged ECG, Holter and echocardiograph[2].

Finally, the study by La Gerche *et al*[40] questions the evidence of screening programmes, indicating the researchers' concern for the long-term repercussion of the unnecessary exclusion from sport with

**Figure 4. Algorithm of screening of 100,000 asymptomatic athletes[25].**




the aim of avoiding SCD, stating that it is a very uncommon pathology and is difficult to diagnose. The study also confirms that if generalised screening is to be carried out, it should be developed in parallel with the creation of centres of excellence in sporting cardiology so as to facilitate the task, with the creation of a record of SCD considered necessary.

## Discussion

ARVC is one of the most frequent causes of SCD in athletes under 35 years of age, and therefore it is necessary to discover their natural history and the different diagnostic criteria with the aim of preventing SCD via screening and treating it appropriately. Unfavourable prognostic factors of the illness are considered to be: a previous heart attack, a history of exertional syncope or family SCD, the early onset of symptoms, physical exercise, the presence of TV, RV dysfunction or affection of the LV and resistance to pharmaceutical drug[s2,7,34].

Its diagnosis is based on the TFC criteria of the AHA, with this being difficult as those affected may remain asymptomatic and may suffer SCD as the first symptom. Genetic tests are indicated in patients with suspected ARVC or with a definitive diagnosis. In the case that any of the mutations are present, genetic tests should also be carried out on patients' family members as a screening method[21,24].

Following the anamnesis and the performance of a complete clinical history, ECG is the first diagnostic tool, with T-wave inversion in right precordial leads being the most common symptom[7-11]. Its presence in the ECG requires the study to be completed with a stress test and an echocardiograph[15]. Echocardiogram is considered to be the first and most used image test in ARVC[7], with the lengthening of the RV outflow tract being the most common anomaly[3,6,16].

CMR is the test of choice as it provides detailed information about the detail of the structure, ventricular function and the degree of fibrofatty replacement, characteristic of ARVC[3,7,16-18]. It is the most useful

follow-up method in asymptomatic patients[9]. In the event it cannot be performed, CT is considered an alternative. 3D electro-anatomical mapping is a useful diagnostic technique as it detects areas with a loss of electrically active myocardium, though it is used individually in patients with suspected ARVC with ventricular arrhythmias[7,18,21].

ARVC treatment is complex, as there is no clear evidence about the effectiveness of the different therapies. Current recommendations indicate the implantation of an ICD and the consumption of anti-arrhythmic drugs (sotalol or amiodarone) and/or excision via catheter of accessory pathways[3,10]. ICD is the most effective preventive treatment for SCD[7,33,35,36], which is indicated in patients with a history of SCD or episodes of sustained TV (high risk). The presence of unknown-origin syncope, non-sustained TV, a family history of SCD and extensive illness are considered to be intermediate risk indications[31,32].

According to the current bibliography, physical exercise should be restricted in patients with a possible, borderline, or definite diagnosis of ARVC, only permitting those of class I-A[1]. This prohibition should be made with the security that the patient has the illness, as this restriction entails a significant limitation on the lifestyle of these athletes. To assess the degree of progression, an ECG and an echocardiograph should be performed each year, along with a Holter and a CMR, the regularity of which depends on the patient[25].

The main sporting organisations and medical societies recommend the carrying out of biannual screening tests to prevent SCD in all athletes from the age of 12 years, before initiating sporting practice[16,19]. The basic cardiovascular assessment is performed systematically on all federated athletes and includes an anamnesis, a physical examination and a 12-lead ECG. On the other hand, advanced testing is only carried out on professional athletes and those with a high risk of SCD. To this latter group, a stress test and echocardiograph are added. If there is suspected ARVC, an initial assessment is recommended via a physical examination, clinical history and compatible family antecedents, ECG, signal-averaged ECG, Holter and echocardiograph[2].

For first-degree family members of patients with ARVC, the necessary tests are considered to be an ECG, an ergometry, an signal-averaged ECG, a Holter and a CMR; performed every 2-3 years if the results are normal. In second degree family members, the study is usually limited to an ECG, an echocardiograph and a Holter every 5 years[19]. The genetic test should be considered in patients with possible ARVC and first-degree family members[3,13], but it is not recommended on those with a single lesser-diagnostic criteria[13]. In patients carrying asymptomatic mutation, an annual or biannual follow-up should be carried out with a clinical history, ECG, echocardiograph, Holter and CMR[19]. In the case of asymptomatic patients without mutation or without a genetic study, follow-up will be every 3-5 years[13].

This systematic review is limited by heterogeneity and the reduced size of the populations studied, as currently no large-scale studies have been carried out in our media, and those that do exist do not include the echocardiograph as an initial screening test for all athletes, with this being highly recommendable for the detection of structural heart disease causing SCD. As well as this, there are no established records of sudden death in athletes; which is why there is no solid evidence of the prevalence of the different illnesses responsible for SCD.

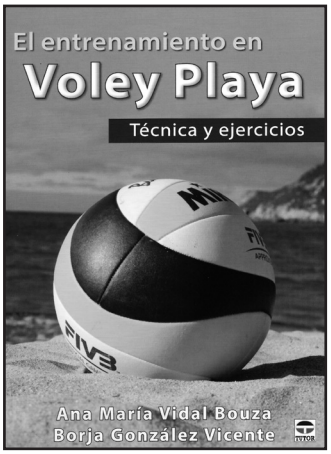
## Conclusions

- Carry out screening on all athletes using: anamnesis, family history, thorough physical examination and 12-lead ECG with the aim of detecting possible causes of SCD. In the case of ARVC, adding the echocardiogram is highly recommendable.
- In cases with anomalies detected via pre-participative screening, it is recommendable to continue the study with an echocardiogram, though this test has diagnostic limitations[41].
- The CMR will be reserved for patients with suspected ARVC without a firm diagnosis using the previously mentioned tests.
- Restrict physical exercise over 4METS in patients with a possible, borderline, and definitive ARVC diagnosis, as well as practising sports, apart from those in I-A class.
- Use sotalol and amiodarone as medical treatment, along with the implantation of an ICD in high-risk patients.
- Perform a periodic follow-up of patients with suspected or diagnosed ARVC, as well as of their first degree family members.
- Create a SCD register with the aim of quantifying the main causes with our means, so as to create a suitable prevention programmes and facilitate the research of these pathologies.

## Bibliography


1. Maron BJ, Levine BD, Reginald L, Baggish AL, Kovacs RJ, Maron MS. A Scientific Statement from the American Heart Association and American College of Cardiology. Eligibility and Disqualification Recommendations for Competitive Athletes with Cardiovascular Abnormalities: Task Force 2: Preparticipation Screening for Cardiovascular Disease in Competitive Athletes. *Circulation.* 2015;132:e267–e272.
2. Pinamonti B, Brun F, Mestroni L, Sinagra G. Arrhythmogenic right ventricular cardiomyopathy: From genetics to diagnostic and therapeutic challenges. *World J Cardiol.* 2014; 6(12):1234–44.
3. Haugaa KH, Haland TF, Leren IS, Saberniak J, Edvardsen T. Arrhythmogenic right ventricular cardiomyopathy, clinical manifestations, and diagnosis. *Europace.* 2016;18(7): 965-72.
4. Philips B, Cheng A. Update on the diagnosis and management of arrhythmogenic right ventricular cardiomyopathy. *Curr Opin Cardiol.* 2015;31(1):6-56.
5. Saberniak J, Hasselberg NE, Borgquist R, Platonov PG, Sarvari SI, Smith HJ, *et al.* Vigorous physical activity impairs myocardial function in patients with arrhythmogenic right ventricular cardiomyopathy and in mutation positive family members. *Eur J Heart Fail.* 2014;16(12):1337–44.
6. Marcus FI, Abidov A. Arrhythmogenic right ventricular cardiomyopathy 2012: diagnostic challenges and treatment. *J Cardiovasc Electrophysiol.* 2012;23(10):1149–53.
7. Saguner AM, Brunckhorst C, Duru F. Arrhythmogenic ventricular cardiomyopathy: A paradigm shift from right to biventricular disease. *World J Cardiol.* 2014;6(4):154–74.
8. O'Hanlon R, Mohiaddin RA. Inherited cardiomyopathies. En: Mushabbar AS, Mohiaddin RA. Magnetic Resonance of Congenital Heart Disease. Berlin: Spinger Science and Business Media; 2012. p.211.
9. Calkins H. Arrhythmogenic right ventricular dysplasia/cardiomyopathy-three decades of progress. *Circ J.* 2015;79(5):901–13.
10. John RM, Tedrow UB, Koplan BA, Albert CM, Epstein LM, Sweeney MO, *et al.* Ventricular arrhythmias and sudden cardiac death. *Lancet.* 2012;380(9852):1520–9.
11. Sniderman JD, Sado DM, Sniderman AD, McKenna WJ.Evaluation of Suspected Right Ventricular Pathology in the Athlete. *Prog Cardiovasc Dis.* 2012;54(5):397–406.

12. Chung FP, Lin YJ, Chang SL, Lo LW, Hu YF, Tuan TC, *et al.* Current and state of the art on the electrophysiologic characteristics and catheter ablation of arrhythmogenic right ventricular dysplasia/cardiomyopathy. *J Cardiol.* 2015;65(6):441–50.
13. McNally E, MacLeod H, Dellefave-Castillo L. Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy. GeneReviews® [revista electrónica] 2005 Abril (consultado 02/04/2016). Disponibe en: http://www.ncbi.nlm.nih.gov/books/NBK1131/
14. Paul M, Wichter T, Fabritz L, Waltenberger J, Schulze-Bahr E, Kirchhof P. Arrhythmogenic right ventricular cardiomyopathy. An update on pathophysiology, genetics, diagnosis, and risk stratification. *Herzschrittmachertherapie und Elektrophysiologie.* 2012; 23(3):186–95.
15. La Gerche A, Baggish AL, Knuuti J, Prior DL, Sharma S, Heidbuchel H, *et al.* Cardiac imaging and stress testing asymptomatic athletes to identify those at risk of sudden cardiac death. *JACC Cardiovasc Imaging.* 2013;6(9):993–1007.
16. Arrigan MT, Killeen RP, Dodd JD, Torreggiani WC.Imaging spectrum of sudden athlete cardiac death. *Clin Radiol.* 2011;66(3):203–23.
17. Perazzolo Marra M, Rizzo S, Bauce B, De Lazzari M, Pilichou K, Corrado D, *et al.* Arrhythmogenic right ventricular cardiomyopathy. *Herz.* 2015;40(4):600–6.
18. Tavano A, Maurel B, Gaubert JY, Varoquaux A, Cassagneau P, Vidal V, *et al*. MR imaging of arrhythmogenic right ventricular dysplasia: What the radiologist needs to know. *Diagn Interv Imaging.* 2015;96(5):449–60.
19. Murray B. Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C): A review of molecular and clinical literature. *J Genet Couns*. 2012;21(4):494–504.
20. Mavrogeni S, Petrou E, Kolovou G, Theodorakis G, Iliodromitis E. Prediction of ventricular arrhythmias using cardiovascular magnetic resonance. *Eur Heart J Cardiovasc Imaging*. 2013;14(6):518–25.
21. Basso C, Bauce B, Corrado D, Thiene G. Pathophysiology of arrhythmogenic cardiomyopathy. *Nat Rev Cardiol.* 2012;9(4):223–33.
22. McGregor SM, Husain AN. A brief review and update of the clinicopathologic diagnosis of arrhythmogenic cardiomyopathy. *Arch Pathol Lab Med.* 2015;139(9):1181–6.
23. Rastegar N, Burt JR, Corona-Villalobos CP, Te Riele AS, James CA., Murray B, *et al.* Cardiac MR Findings and Potential Diagnostic Pitfalls in Patients Evaluated for Arrhythmogenic Right Ventricular Cardiomyopathy. *RadioGraphics.* 2014;34(6):1553–70.
24. Alcalde M, Campuzano O, Sarquella-Brugada G, Arbelo E, Allegue C, Partemi S, *et al.* Clinical interpretation of genetic variants in arrhythmogenic right ventricular cardiomyopathy. *Clin Res Cardiol*. 2015;104(4):288–303.
25. Hertz CL, Ferrero-Miliani L, Frank-Hansen R, Morling N, Bundgaard H. A comparison of genetic findings in sudden cardiac death victims and cardiac patients: the importance of phenotypic classification. *Europace*. 2015;17(3):350–7.
26. Ackerman MJ, Priori SG, Willems S, Berul C, Brugada R, Calkins H, *et al.* HRS/EHRA expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies: This document was developed as a partnership between the Heart Rhythm Society (HRS) and the European Heart Rhythm Association (EHRA). *Hear Rhythm.* 2011;8(8):1308–39.
27. Perrin MJ, Gollob MH. The genetics of cardiac disease associated with sudden cardiac death. *J Mol Diagnostics.* 2012;14(5):424–36.
28. Patel H, Shah P, Rampal U, Shamoon F, Tiyyagura S. Arrythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) and cathecholaminergic polymorphic ventricular tachycardia (CPVT): A phenotypic spectrum seen in same patient. *J Electrocardiol*. 2015; 48(5):874–8.
29. Barsheshet A, Brenyo A, Moss AJ, Goldenberg I. Genetics of sudden cardiac death. *Curr Cardiol Rep.* 2011;13(5):364–76.
30. Asimaki A, Kleber AG, Saffitz JE. Pathogenesis of Arrhythmogenic Cardiomyopathy. *Can J Cardiol.* 2015;31(11):1313–24.
31. Kantor PF, Lougheed J, Dancea A, McGillion M, Barbosa N, Chan C, *et al.* Presentation, diagnosis, and medical management of heart failure in children; Canadian Cardiovascular Society guidelines. *Can J Cardiol.* 2013;29(12):1535–52.
32. Cheung CC, Laksman ZW, Mellor G, Sanatani S, Krahn AD. Exercise and Inherited Arrhythmias. *Can J Cardiol.* 2016;32(4):452-8.
33. Dohadwala M, Link MS. Implantable defibrillators in long QT syndrome, Brugada syndrome, hypertrophic cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy. *Cardiol Clin.* 2014;32(2):305–18.
34. Cadrin-Tourigny J, Tadros R, Talajic M, Rivard L, Abadir S, Khairy P. Risk stratification for sudden death in arrhythmogenic right ventricular cardiomyopathy. *Expert Rev Cardiovasc Ther.* 2015;13(6):653–64.
35. Schinkel AF. Implantable cardioverter defibrillators in arrhythmogenic right ventricular dysplasia/cardiomyopathy: patient outcomes, incidence of appropriate and inappropriate interventions, and complications. *Circ Arrhythm Electrophysiol.* 2013;6(3):562–8.
36. Thiene G. The research venture in arrhythmogenic right ventricular cardiomyopathy: a paradigm of translational medicine. *Eur Heart J.* 2015;36(14):837–46.
37. Rojas A, Calkins H. Present understanding of the relationship between exercise and arrhythmogenic right ventricular dysplasia/cardiomyopathy. *Trends Cardiovasc Med.* 2015;25(3):181–8.
38. Wilson MG, Sharma S, Carre F, Charron P, Richard P, O'Hanlon R, *et al*. Significance of deep T-wave inversions in asymptomatic athletes with normal cardiovascular examinations: practical solutions for managing the diagnostic conundrum. *Br J Sports Med.* 2012;46(Suppl_1):i51–i58.
39. Sitges M, Gutiérrez JA, Brugada J, Balius R, Bellver M, Brotons D, *et al.* Consenso para prevenir la muerte súbita cardíaca de los deportistas. *Apunts Med Esport.* 2013;48 (177):35-41.
40. La Gerche A, Macisaac AI, Prior DL. Should pre-participation cardiovascular screening for competitive athletes be introduced in Australia? A timely debate in a sport-loving nation. *Heart Lung Circ.* 2011;20(10):629–33.
41. Luengo E, Manonelles P. *Muerte súbita en el deporte. Métodos de cribado.* Sociedad Española de Medicina del Deporte (SEMED-FEMEDE). Barcelona. Editorial Esmon Publicidad, S.A.; 2016.p.22.

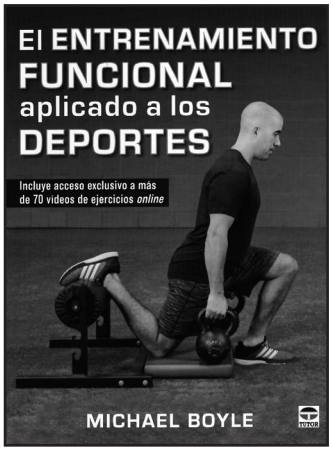



## EL ENTRENAMIENTO DE VOLEY PLAYA. TÉCNICA Y EJERCICIOS

**Por: Ana María Vidal Bouza y Borja González Vicente**
**Edita: Ediciones Tutor-Editorial El Drac.**
**Impresores 20. P.E. Prado del Espino. 28660 Boadilla del Monte. Madrid.**
**Telf: 915 599 832 - Fax: 915 410 235**
**E-mail: info@edicionestutor.com Web: www.edicionestutor.com**
**Madrid 2017, 176 páginas, P.V.P: 25 euros**

Este libro es un completo y actualizado manual de aprendizaje y enseñanza del vóley playa para deportistas de todos los niveles. En un deporte joven como el vóley playa, que ha experimentado una notable evolución desde sus inicios, hace tiempo que era necesaria una guía renovada para dar respuesta a las nuevas tendencias de juego y la nueva reglamentación, reemplazando así a los escasos y ya desfasados textos existentes.

Este manual práctico ofrece una completa descripción de las técnicas básicas del vóley playa actual, con detalladas ilustraciones y fotografías, así como claras explicaciones de la aplicación de esas técnicas en las situaciones de juego. Además, con sus casi 100 ejercicios de entrenamiento para diferentes niveles de preparación, este libro se convierte en un material de trabajo esencial para todo entrenador de vóley playa que desee enriquecer sus conocimientos.



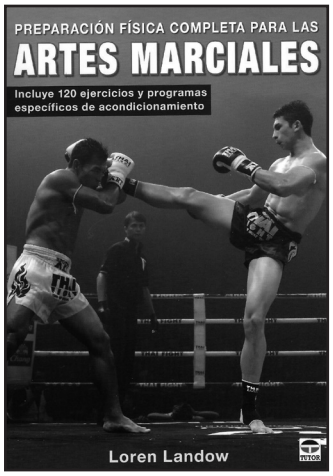

## ENTRENAMIENTO FUNCIONAL APLICADO A LOS DEPORTES

**Por: Michael Boyle**
**Colección: En Forma**
**Edita: Ediciones Tutor-Editorial El Drac.**
**Impresores 20. P.E. Prado del Espino. 28660 Boadilla del Monte. Madrid.**
**Telf: 915 599 832 - Fax: 915 410 235**
**E-mail: info@edicionestutor.com Web: www.edicionestutor.com**
**Madrid 2017, 256 páginas, P.V.P: 29,95 euros**

Entrenar para rendir al máximo con el menor riesgo de lesión. Este libro propicia unos resultados inmejorables en la pista, el terreno de juego, la cancha, el tatami, etc., no solo en la sala de musculación. El autor, uno de los mejores entrenadores de rendimiento deportivo del mundo, presenta en este libro los conceptos, métodos, ejercicios y programas que maximizan los movimientos de los deportistas en competición.

Incluye una serie de evaluaciones funcionales que ayudan a determinar el diseño de un plan específico para cada deportista. Además, las progresiones de autorrefuerzo con ejercicios para el tren inferior, la zona media (Core), el tren superior y, por último, el cuerpo entero, proporcionan a los deportistas el equilibrio, la propiocepción, la estabilidad y la fuerza que necesitan para sobresalir en sus deportes. Se incluyen programas modelo que ayudan a elaborar el programa personalizado con el que trabajar cada aspecto del entrenamiento y garantizar un buen rendimiento físico.

El contenido del libro se completa con el acceso exclusivo a vídeos online con demostraciones, comentarios y análisis de más de 70 ejercicios clave.




## PREPARACIÓN FÍSICA COMPLETA PARA LAS ARTES MARCIALES

**Por: Loren Landow**
**Edita: Ediciones Tutor-Editorial El Drac.**
**Impresores 20. P.E. Prado del Espino. 28660 Boadilla del Monte. Madrid.**
**Telf: 915 599 832 - Fax: 915 410 235**
**E-mail: info@edicionestutor.com Web: www.edicionestutor.com**
**Madrid 2017, 272 páginas, P.V.P: 19,95 euros**

Golpear de mano y pierna con potencia explosiva. Derribar y pelear cuerpo a cuerpo con fuerza máxima. Contraatacar y esquivar con velocidad y agilidades inigualables. Ser el mejor. Dar el máximo de uno. Es lo que el lector puede lograr con este exhaustivo manual para alcanzar el dominio mental y físico.

El libro ofrece 120 ejercicios, cada uno de ellos adaptado para desarrollar una habilidad o atributo clave en artes marciales, a fin de mejorar el rendimiento en la disciplina que practique el lector y los objetivos que tenga establecido. Incluye un capítulo dedicado a la importancia de la recuperación y la nutrición. Aprenderá después a estructurar un programa de acondicionamiento para lograr mejoras a corto plazo y el éxito a largo plazo: triunfo al que asistirá y que sus adversarios sentirán.

## ¿A cuántos estímulos responde tu corazón?

**Vichy Catalán** se preocupa por tu salud e investiga sobre el metabolismo del colesterol.

# Te quiere


www.vichycatalan.es

# Vichy Catalán y el colesterol

**Dra. Míriam Torres Moreno**

Dietista y Licenciada en Ciencia y Tecnología de los Alimentos.
Doctora por la URV en Nutrición y Metabolismo.

El agua es un nutriente esencial para el hombre siendo su consumo indispensable para el mantenimiento del estado de hidratación del organismo y garantizar un buen estado de salud.

A nivel de composición nutricional, el agua aporta como únicos nutrientes los elementos minerales, presentes de forma natural en la misma. El tipo de minerales y el contenido de cada uno de ellos resultan característicos de las distintas aguas y por ello pueden caracterizarse. Vichy Catalán es un agua mineral natural carbónica que contiene 1.097 miligramos de sodio por litro, en cuya composición destacan además otros oligominerales como: bicarbonatos, sulfatos, cloruros y potasio. La biodisponibilidad de los electrolitos en esta agua es muy alta y por ello se considera que por un lado contribuye a la ingesta total diaria de estos nutrientes (FNB, 2004) y por otro que puede desempeñar un papel en la prevención de las enfermedades cardiovasculares.

En esta línea de evidencia, recientes investigaciones han demostrado que el consumo de 1 litro al día de agua mineral bicarbonatada como Vichy Catalán durante 8 semanas reduce el riesgo cardiovascular en mujeres postmenopáusicas y en adultos jóvenes hipercolesterolémicos, reduciendo tanto las cifras de colesterol-LDL como el ratio de colesterol total/colesterol HDL. A nivel de cifras tensionales, aún siendo el contenido en sodio del agua administrada superior a 1 gramo por litro, no se observa afectación en las cifras tensionales entre las mujeres postmenopáusicas e incluso se reducen las cifras de tensión arterial sistólica entre los adultos jóvenes (Schoppen S, 2004 y Perez-Granados, 2010).

Por otro lado, también se ha establecido la relación entre el consumo de 0,5 L/día de agua Vichy Catalán (agua mineral bicarbonatada) con una comida estándar y la reducción de la lipemia postpandrial en mujeres postmenopáusicas también sanas, respecto el consumo de agua mineral con menor contenido en minerales (Schoppen, 2005). Hallazgo de gran interés, ya que se sabe que el metabolismo lipídico postprandial juega un papel muy importante en la salud, ya puede ser un factor de riesgo en el desarrollo de aterogénesis y de las enfermedades cardiovasculares.

Ambos resultados obtenidos en estos estudios demuestran por tanto la influencia que los hábitos alimentarios y, en concreto, la hidratación y el tipo de agua de bebida, pueden tener en la prevención de las enfermedades cardiovasculares.

El efecto preventivo demostrado en estos estudios que ejerce el consumo de *Vichy Catalán,* tanto en el metabolismo del colesterol como en el de las lipoproteínas, parece ser debido a la composición característica de esta agua carbónica que la diferencia del resto de aguas comerciales, por su alto contenido en sodio, potasio, bicarbonato, sílice e incluso litio.

Además de los efectos preventivos a nivel cardiovascular anteriormente descritos, otras investigaciones recientes como la realizada por Toxqui (2012) estudian otros posibles efectos del consumo de agua bicarbonatada y la salud cardiovascular. En este caso, se estudian los efectos postpandriales de la ingesta de agua bicarbonatada sódica consumida con una comida estándar sobre los niveles séricos de triglicéridos (TG), de colecistoquinina y a nivel de la contracción y el vaciado de la vesícula biliar. Así se demuestra que en adultos jóvenes de 18 a 40 años de ambos sexos el consumo de 0,5 L/día de agua bicarbonatada sódica junto con una comida estándar (rica en grasas: 62% de lípidos, 30% de hidratos de carbono y 8% de proteínas) induce a menores niveles de triglicéridos y colecistoquinina postpandriales, elementos con claro impacto sobre la salud cardiovascular.

Los niveles de triglicéridos postpandriales son un reflejo del metabolismo lipídico postpandrial que tiene un papel fundamental en el desarrollo de las enfermedades cardiovasculares, ya que un anormal transporte y metabolismo de las lipoproteínas LDL (ricas en TG) en el periodo postpandrial se ha relacionado con la aterogénesis. Y, por lo tanto, como se demuestra en este estudio, una reducción en las lipoproteínas ricas en TG podría limitar la progresión de la arteriosclerosis.

La colecistoquinina por su parte es una hormona que estimula la contracción de la vesícula biliar que segrega las sales biliares encargadas de la solubilización y absorción de las grasas. Por tanto, una reducción en los niveles de colecistoquinina postpandriales supone a su vez la reducción de la contracción y vaciamiento de la vesícula biliar y en consecuencia una menor absorción intestinal de lípidos.

Por lo tanto, y a modo de conclusión, demostrado el efecto positivo que el consumo de agua bicarbonatada carbónica tiene sobre la salud, Vichy Catalán podría ser utilizado como un elemento a incluir en la alimentación habitual de la población sana para conseguir reducir el riesgo cardiovascular.

## Bibliografía

Food and Nutrition Board of he Institute of Medicine. *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*. Washington, DC: National Academies Press, 2004.

Schoppen S, Pérez-Granados AM, Carbajal A, Sarria B, Sánchez-Muniz FJ, Gomez-Gerique JA, Vaquero MP. Sodium bicarbonated mineral water decreases postpandrial lipaemia in postmenopausal women compared to a low mineral water. *Br J Nutr* 2005;94(4):582-7.

Schoppen S, Pérez-Granados AM, Carbajal A, Oubina P, Sánchez-Muniz FJ, Gomez-Gerique JA, Vaquero MP. A sodium rich carbonated mineral water reduces cardiovascular risk in postmenopausal women. *J Nutr* 2004;134(5):1058-63.

Pérez-Granados AM, Navas-Carretero S, Schoppen S, Vaquero MP. Reduction in cardiovascular risk by sodium-bicarbonated mineral water in moderately hypercholesterolemic young adults. *J Nutr Biochem* 2010;21(10):948-53.

Toxqui L, Pérez-Granados AM, Blanco-Rojo R, Vaquero MP. A sodium-bicarbonated mineralwater reduces gallbladder emptying and postpandrial lipaemia: A randomised postpandrial four-way crossover study. *Eur J Nutr 2012*;51(5):607-14.

# VII JORNADAS NACIONALES DE MEDICINA DEL DEPORTE

## EL EJERCICIO FÍSICO: DE LA PREVENCIÓN AL TRATAMIENTO

**24-25 de noviembre de 2017**

Zaragoza

Aula Luis Giménez - Pedro Asirón

**ASOCIACIÓN ARAGONESA DE MEDICINA DEL DEPORTE (ARAMEDE)**

**SOCIEDAD ESPAÑOLA DE MEDICINA DEL DEPORTE (SEMED)**

# PROGRAMA PRELIMINAR

## PONENCIAS

### DÍA 24 DE NOVIEMBRE, VIERNES

09.00-09.30 **Recogida documentación**

09.30-11.00 **PONENCIA: Reconocimientos médicos de despistaje**
Moderador: ***José Manuel González de Suso Janariz***

El programa de reconocimientos médicos en populares
***Juan Miguel Morillas Martínez***

El papel del electrocardiograma de reposo

El papel de la prueba de esfuerzo
***Gonzalo María Correa González***

11.00-11.30 **Café/Descanso**

11.30-13.00 **PONENCIA: Componentes de las bebidas para el deporte**
Moderador: ***Juan José Lacleta Almolda***

El agua
***Nieves Palacios Gil de Antuñano***

El sodio
***José Antonio Villegas García***

Los carbohidratos
***Raquel Blasco Redondo***

13.00-13.45 **CONFERENCIA INAUGURAL**
Presentador
***Pedro Manonelles Marqueta***
***José Luis Terreros Blanco***

13.45-14.15 **Inauguración oficial**

16.00-17.30 **PONENCIA: El ejercicio en poblaciones específicas**
Moderador

Personas con discapacidad física
***Josep Oriol Martínez Ferrer***

Personas con discapacidad psíquica
***Francisco Javier Ordóñez Muñoz***

Personas de edad avanzada
***Jovanka Manojlovic Rakocevic***

17.30-18.00 **Café/Descanso**

18.00-19.30 **PONENCIA: El deporte en la mujer**
Moderador: ***Carlos Moreno Pascual***

Embarazo y puerperio
***Juan Carlos De la Cruz Márquez***

La triada de la mujer deportista
***Mª. Josep Martí Utset***

Trastornos de la conducta alimentaria
***Helena Palacín Fornons***

### DÍA 25 DE NOVIEMBRE, SÁBADO

09.00-10.00 **Comunicaciones libres**

10.00-11.30 **PONENCIA: Prescripción y programación de ejercicio**
Moderador: ***Ángel Durántez Prados***

Evidencia científica
***Zigor Montalvo Zenarruzabeitia***

La prescripción de ejercicio
***Carmen Porcar Rivero***

La programación del ejercicio
***Javier Álvarez Medina***

11.30-12.00 **Café/Descanso**

12.00-13.30 **PONENCIA: La prescripción en la enfermedad**
Moderador: ***Carlos Melero Romero***

Enfermedad cardiovascular
***Luis Serratosa Fernández***

Enfermedades respiratorias
***Franchek Drobnic Martínez***

Enfermedades metabólicas
***Fernando Salom Portella***

13.30 **Clausura de las Jornadas**

## COMUNICACIONES CIENTÍFICAS

El Comité Científico invita a todos los participantes a remitir comunicaciones científicas a las **VII Jornadas Nacionales de Medicina del Deporte**.

Los temas de las Jornadas Nacionales para presentación de Comunicaciones Científicas se describen en el siguiente listado:

- Actividad física y salud
- Programación de ejercicio para la salud
- Lesiones deportivas: diagnóstico, prevención y tratamiento
- Medicina del deporte
- Cardiología del deporte
- Nutrición y ayudas ergogénicas
- Cineantropometría
- Fisiología del esfuerzo
- Entrenamiento y mejora del rendimiento

Las Comunicaciones Orales se distribuirán en sesiones de los temas de las Jornadas Nacionales. Por favor, escoja uno de los temas del listado como propuesta para realizar su presentación. El Comité Científico podrá reasignar el abstract en otro tema de las Jornadas.

Los trabajos deberán ser originales y no se habrán presentado en congresos anteriores o reuniones similares.

Los abstracts de las comunicaciones científicas admitidas, comunicaciones orales y pósters (presentación interactiva), serán publicados en la revista Archivos de Medicina del Deporte.

Información complementaria en: **www.femede.es**

## PREMIOS

Los inscritos en las VII Jornadas Nacionales de Medicina del Deporte que presenten comunicaciones **podrán optar al Premio a la Mejor Comunicación oral de las Jornadas.**

Para optar al premio **SE DEBE HACER CONSTAR EXPLÍCITAMENTE QUE SE OPTA A PREMIO** en carta dirigida al presidente del Comité Científico y adjuntar al Resumen remitido. En este caso, además de enviar el Formato del Resumen de Comunicación Científica, se debe de mandar el **trabajo completo en el plazo de presentación de las Comunicaciones Científicas.**

Los trabajos que se presentan en formato de póster (presentación interactiva) no optan a premio.

El trabajo que obtenga la segunda mejor puntuación, y supere en nivel de calidad exigible, será dotado con un accésit a la Mejor Comunicación de las Jornadas.

### Dotación de los premios

**Premio a la Mejor Comunicación Oral** de las Jornadas:

- Dotación económica: **1.000 euros.**
- Certificado acreditativo.
- Publicación en la revista Archivos de Medicina del Deporte con indicación del premio obtenido.

**Accésit a la Mejor Comunicación Oral** de las Jornadas:

- Dotación económica: **500 euros.**
- Certificado acreditativo.
- Publicación en la revista Archivos de Medicina del Deporte con indicación del premio obtenido.

El premio será entregado en la clausura de las Jornadas.

Los trabajos premiados serán publicados en la revista Archivos de Medicina del Deporte (para lo que deberán ser adaptados a las normas de publicación) y se aceptará la revisión efectuada por el Comité Científico.

Los premios podrán ser declarados desiertos si no alcanzan el nivel de calidad exigible.

Información complementaria en **www.femede.es**

## INFORMACIÓN GENERAL

**Sede de las Jornadas**

**Hotel Silken Reino de Aragón**
Vía Ibérica, 1
Calle Coso nº 80. 50001 Zaragoza
Teléfono: +34 976 468 200

**Secretaría Técnica**

**Asociación Aragonesa de Medicina del Deporte**
Paseo de Teruel nº 23, 3º-3ª. 50004 Zaragoza
Teléfono: 976 237 129
Correo electrónico: aramede@telefonica.net
Página web: http://www.femede.es

**Agencia de viajes**

**Viajes El Corte Inglés S.A.**
IATA 78211733. División Eventos Deportivos
Teniente Borges, nº 5. 41002 Sevilla
Teléfono: 954 506 603/04
Fax: 954 222 479
Correo electrónico: areaeventos@viajeseci.es
Horario: Lunes a viernes de 9.00-14.00 y de 16.00-19.00

| Derechos de inscripción | Antes del 9-7-17 | 10-7-17 al 15-9-17 | Desde el 16-9-17 y en sede Jornadas |
|---|---|---|---|
| Cuota general | 125 euros | 150 euros | 200 euros |
| Miembros ARAMEDE/ FEMEDE | 100 euros | 125 euros | 175 euros |
| Médicos MIR* | 60 euros | 75 euros | 125 euros |
| Estudiantes** | 30 euros | 40 euros | 50 euros |

*Es necesaria acreditación
**Grados, Licenciaturas y Diplomaturas: Medicina, CC Actividad Física y Deporte, CC de la Salud...). Es necesaria acreditación. No se considera estudiantes los profesionales que cursen estudios, ni a graduados, licenciados y/o diplomados.

**Forma de pago**

**Efectivo**
Asociación Aragonesa de Medicina del Deporte.
Paseo de Teruel nº 23, 4º-3ª. 50004 Zaragoza (17-20 horas).

**Transferencia**
IBERCAJA-CAJA DE AHORROS DE LA INMACULADA:
C.C. ES78 2085 5261 6703 3007 8328
**En ambos casos hay que aportar el boletín de inscripción adecuadamente cumplimentado.**

| 2017 | | |
|---|---|---|
| **11th International Symposium on Computer Science in Sport** | 6-9 Septiembre<br>Konstanz (Alemania) | web: www.mmsp.uni-konstanz.de/iacss2017/home/ |
| **27º Congress European Society for surgery of the shoulder and the elbow (SECEC-ESSSE)** | 13-16 Septiembre<br>Berlín (Alemania) | web: www.secec2017.com |
| **La dosis correcta para el tratamiento del dolor y la recuperación del atleta** | 15 Septiembre<br>Bari (Italia) | web: http://www.fmsi.it/ |
| **12th European Congress Fédération Internationale d'Education Physique** | 13-16 Septiembre<br>Luxemburgo (Luxemburgo) | web: https://fiep2017luxembourg.uni.lu/ |
| **6th Annual Meeting "European Initiative for Exercise in Medicine" (EIEIM)** | 15-16 Septiembre<br>Lisboa (Portugal) | web: http://exerciseismedicine.eu |
| **Medical and Training Aspects in Handball** | 16 Septiembre<br>Differdange (Luxemburgo) | web: www.handball-congress.lu |
| **VISTA Conference** | 20-23 Septiembre<br>Toronto-Ontario (Canadá) | web: http://sirc.ca/www.vista2017.com |
| **Congreso Internacional de Ciencias de la Actividad Física, el Deporte y la Salud** | 21-23 Septiembre<br>San Miguel de Tucumán (Argentina) | E-mail: guillermorubeno@blanquerna.url.edu |
| **4th Saúde Atlântica & ISAKOS & ESSKA International Meeting** | 22-23 Septiembre<br>Porto (Portugal) | web: http://jornadassaudeatlantica.com |
| **I Congreso Internacional en Prevención y Readaptación Física Interdisciplinar** | 22-24 Septiembre<br>Granada | web: http://en-forma.es/inscripcion-congreso-en-forma/ |
| **Developments in Doping - Keeping Sport Clean** | 25 Septiembre<br>Londres (Reino Unido) | E-mail: events@rsm.ac.uk |
| **Fortius International Sports Injury Conference (FISIC'17)** | 27-28 Septiembre<br>Londres (Reino Unido) | web: www.fortiusclinic.com/ |
| **54º Congreso Nacional de la Sociedad Española de Cirugía Ortopédica y Traumatología (SECOT)** | 27-29 Septiembre<br>Barcelona | web: www.secot.es |
| **VII Congreso Iberoamericano de Nutrición** | 28-30 Septiembre<br>Cuzco (Perú) | web: http://www.iberonutricion2017.com/ |
| **VIII Congreso de la World Federation of Athletic Trainer and Therapy: Patologías de las fascia en el deporte y su readaptación** | 29 Septiembre-1 Octubre<br>Villanueva de la Cañada (Madrid) | web: http://www.ucjc.edu/congresowfattspain/ |
| **USOC's International Altitude Training Symposium (IATS)** | 4-6 Octubre<br>Colorado Springs (EE.UU.) | web: http://www.teamusa.org/ |

| | | |
|---|---|---|
| **4th International Symposium on Intra-Articular Treatment** | 5-7 Octubre<br>Praga (Rep. Checa) | web: www.isiat2017.com |
| **II Congreso de la Asociación Española contra la Muerte Súbita José Durán: "Mitos y realidades en la muerte súbita del deportista"** | 6 Octubre<br>Madrid | web: www.fundacionquaes.com |
| **II World Conference of Sports Physiotherapy** | 6-7 Octubre<br>Belfast (Irlanda del Norte) | web: www.physiosinsport.org |
| **International Scientific Conference on Applied Sports Science (ISCASS)** | 12-14 Octubre<br>Alexandria (Egipto) | web: www.ierek.com/events/applied-sports-science-conference |
| **Congreso Peruano de Ortopedia y Traumatología** | 12-14 Octubre<br>Lima (Perú) | web: http://spotrauma.org |
| **Congreso Internacional sobre la Enseñanza de la Educación Física y el Deporte Escolar** | 12-15 Octubre<br>Villena (Alicante) | E-mail: info@profesport.org<br>web: http://retos.org/feadef/congreso/index.html |
| **XXI Congreso Internacional de Nutrición** | 15-20 Octubre<br>Buenos Aires (Argetina) | web: www.icn2017.com |
| **International Dead Sea Symposium (IDSS) of Sport and Arrhythmia** | 16-18 Octubre<br>Tel Aviv (Israel) | web: www.idss-sport.com |
| **European Medical Fitness Congress** | 20-22 Octubre<br>Barcelona | web: www.simphmedicalfitness.es<br>www.medicalfitnesscongress.com |
| **48 Congreso Nacional de Podología** | 20-22 Octubre<br>Salamanca | web: www.aepode.org / http://www.cgcop.es/ |
| **37º Congresso Nacional de Ortopedia e Traumatologia - SPOT 2017** | 26-28 Octubre<br>Coimbra (Portugal) | web: http://beta.jointogethergroup.com/spot2017 |
| **2nd International Conference of Sport and Health Science** | 1-3 Noviembre<br>Dead Sea (Jordania) | web: http://conferences.ju.edu.jo/sites/icsscc2017 |
| **¿Qué hay de nuevo en la Traumatología de los deportes de nieve?** | 3-4 Noviembre<br>Madrid | web: www.qhdn2017.com |
| **I ESMA Open Meeting: "Stop sports injuries – back to sports"** | 3-4 Noviembre<br>Munich (Alemania) | web: www.esma-conferencia.org |
| **6º Congreso Mundial del Deporte Escolar, Educación Física y Psicomotricidad** | 9-11 Noviembre<br>La Coruña | web: https://www.sportis.es |
| **53º Congreso Chileno de Ortopedia y Traumatología - SCHOT 2017** | 15-18 Noviembre<br>Villa del Mar (Chile) | web: www.schot.cl/congreso-chileno-de-ortopedia-y-traumatologia-2017/ |
| **10th EFSMA (European Federation of Sports Medicine Associations) Congress** | 16-18 Noviembre<br>Cascais (Portugal) | Email: secretariat@efsma2017.org<br>web: www.efsma2017.org |

| | | |
|---|---|---|
| **World Congress in Sports and Exercise Medicine** | 17-19 Noviembre<br>Kuala Lumpur (Malasia) | E-mail: info@wcsem2017.org<br>web: http://www.wcsem2017.org |
| **VII Convención Internacional de Actividad Física y Deporte AFIDE 2017** | 20-24 Noviembre<br>La Habana (Cuba) | E-mail: afide@inder.cu |
| **VII Jornadas Nacionales de Medicina del Deporte** | 24-25 Noviembre<br>Zaragoza | Información: femede@femede.es |
| **BRICSCESS 2017: Exercise and Sports Science Conference** | 29 Noviembre- 2 Diciembre<br>Santos (Brasil) | web: http://bricscess2017.com/index.html |
| **Congress of Applied Sports Sciences** | 1-2 Diciembre<br>Sofía (Bulgaria) | web: http://icass2017.com/ |
| **54° Congreso Argentino de Ortopedia y Traumatología** | 2-5 Diciembre<br>Buenos Aires (Argentina) | web: http://www.congresoaaot.org.ar |
| **5th Congress of ECOSEP** | 9-10 Diciembre<br>Dubái (Dubái) | web: http://ecosepjdc.eu/ |
| **2018** | | |
| **Congrès francophone de médecine de montagne** | 17-21 Enero<br>Champéry, (Suiza) | web: www.grimm-vs.ch |
| **World Congress on Osteoporosis, Osteoarthritis and Musculoskeletal Diseases** | 19-22 Abril<br>Cracovia (Polonia) | web: www.wco-iof-esceo.org/ |
| **18th ESSKA Congress** | 9-12 Mayo<br>Glasgow (Reino Unido) | web: http://esska-congress.org/ |
| **7thWorld Conference on Women and Sport** | 17-20 Mayo<br>Gaborone (Bostwana) | web: www.icsspe.org/sites/default/files/e8_7TH%20IWG%20Conference%20docx.pdf |
| **XXVII Conferencia Internacional Sports Rehabilitation and Traumatology** | Mayo<br>Barcelona | www.isokinetic.com |
| **European Congress of Adapted Physical Activity (EUCAPA)** | 3-5 Julio<br>Worcester (Reino Unido) | Andrea Faull. E-mail: a.faull@worc.ac.uk<br>Ken Black. E-mail: k.black@worc.ac.uk |
| **23rd Annual Congress of the European College of Sport Science** | 4-7 Julio<br>Dublín (Irlanda) | web: www.ecss-congress.eu/2018/ |
| **World Congress of Biomechanics** | 8-12 Julio<br>Dublín (Irlanda) | web: http://wcb2018.com/ |
| **12th World Congress of the International Society of Physical and Rehabilitation Medicine (ISPRM)** | 8-12 Julio<br>París (Francia) | web: http://isprm2018.com/ |

| | | |
|---|---|---|
| **XXXV Congreso Mundial de Medicina del Deporte** | 12-15 Septiembre<br>Rio de Janeiro (Brasil) | web: www.fims.org |
| **5th International Scientific Tendinopathy Symposium (ISTS)** | 27-29 Septiembre<br>Groningen (Países Bajos) | web: http://ists2018.com/ |
| **28º Congress European Society for surgery of the shoulder and the elbow (SECEC-ESSSE)** | Ginebra (Suiza) | web: www.secec.org |
| **2019** | | |
| **12th Biennial ISAKOS** | 12-16 Mayo<br>Cancún (México) | web: www.isakos.com |
| **24th Annual Congress of the European College of Sport Science** | 3-6 Julio<br>Praga (Rep. Checa) | E-mail: office@sport-science.org |
| **14th International Congress of shoulder and elbow surgery (ICSES)** | 17-20 Septiembre<br>Buenos Aires (Argentina) | web: www.icses2019.org |
| **XV Congreso Nacional de Psicología de la Act. Física y del Deporte** | Zaragoza | web: www.psicologiadeporte.org |
| **2020** | | |
| **25th Annual Congress of the European College of Sport Science** | 1-4 Julio<br>Sevilla | E-mail: office@sport-science.org |
| **XXXVI Congreso Mundial de Medicina del Deporte** | 24-27 Septiembre<br>Atenas (Grecia) | web: www.globalevents.gr |
| **2021** | | |
| **26th Annual Congress of the European College of Sport Science** | 7-10 Julio<br>Glasgow (Reino Unido) | E-mail: office@sport-science.org |

# Cursos *on-line* SEMED-FEMEDE

## Curso "ENTRENAMIENTO, RENDIMIENTO, PREVENCIÓN Y PATOLOGÍA DEL CICLISMO"

Curso dirigido a los titulados de las diferentes profesiones sanitarias y a los titulados en ciencias de la actividad física y el deporte, destinado al conocimiento de las prestaciones y rendimiento del deportista, para que cumpla con sus expectativas competitivas y de prolongación de su práctica deportiva, y para que la práctica deportiva minimice las consecuencias que puede tener para su salud, tanto desde el punto de vista médico como lesional.

## Curso "ELECTROCARDIOGRAFÍA PARA MEDICINA DEL DEPORTE"

ACREDITADO POR LA COMISIÓN DE FORMACIÓN CONTINUADA (ON-LINE 15/10/2015 A 15/10/2016) CON 4,81 CRÉDITOS

Curso dirigido a médicos destinado a proporcionar los conocimientos específicos para el estudio del sistema cardiocirculatorio desde el punto de vista del electrocardiograma (ECG).

## Curso "FISIOLOGÍA Y VALORACIÓN FUNCIONAL EN EL CICLISMO"

Curso dirigido a los titulados de las diferentes profesiones sanitarias y a los titulados en ciencias de la actividad física y el deporte, destinado al conocimiento profundo de los aspectos fisiológicos y de valoración funcional del ciclismo.

## Curso "AYUDAS ERGOGÉNICAS"

Curso abierto a todos los interesados en el tema que quieren conocer las ayudas ergogénicas y su utilización en el deporte.

## Curso "CARDIOLOGÍA DEL DEPORTE"

ACREDITADO POR LA COMISIÓN DE FORMACIÓN CONTINUADA (VÁLIDA DEL 15/10/2016 AL 15/10/2017) CON 8,78 CRÉDITOS

Fecha límite de inscripción: 15/06/2017

Curso dirigido a médicos destinado a proporcionar los conocimientos específicos para el estudio del sistema cardiocirculatorio desde el punto de vista de la actividad física y deportiva, para diagnosticar los problemas cardiovasculares que pueden afectar al deportista, conocer la aptitud cardiológica para la práctica deportiva, realizar la prescripción de ejercicio y conocer y diagnosticar las enfermedades cardiovasculares susceptibles de provocar la muerte súbita del deportista y prevenir su aparición.

## Curso "ALIMENTACIÓN, NUTRICIÓN E HIDRATACIÓN EN EL DEPORTE"

Curso dirigido a médicos destinado a facilitar al médico relacionado con la actividad física y el deporte la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para prescribir una adecuada alimentación del deportista.

## Curso "ALIMENTACIÓN Y NUTRICIÓN EN EL DEPORTE"

Curso dirigido a los titulados de las diferentes profesiones sanitarias (existe un curso específico para médicos) y para los titulados en ciencias de la actividad física y el deporte, dirigido a facilitar a los profesionales relacionados con la actividad física y el deporte la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para conocer la adecuada alimentación del deportista.

## Curso "ALIMENTACIÓN Y NUTRICIÓN EN EL DEPORTE" Para Diplomados y Graduados en Enfermería

ACREDITADO POR LA COMISIÓN DE FORMACIÓN CONTINUADA (NO PRESENCIAL 15/12/2015 A 15/12/2016) CON 10,18 CRÉDITOS

Curso dirigido a facilitar a los Diplomados y Graduados en Enfermería la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para conocer la adecuada alimentación del deportista.

## Curso "CINEANTROPOMETRÍA PARA SANITARIOS"

Curso dirigido a sanitarios destinado a adquirir los conocimientos necesarios para conocer los fundamentos de la cineantropometría (puntos anatómicos de referencia, material antropométrico, protocolo de medición, error de medición, composición corporal, somatotipo, proporcionalidad) y la relación entre la antropometría y el rendimiento deportivo.

## Curso "CINEANTROPOMETRÍA"

Curso dirigido a todas aquellas personas interesadas en este campo en las Ciencias del Deporte y alumnos de último año de grado, destinado a adquirir los conocimientos necesarios para conocer los fundamentos de la cineantropometría (puntos anatómicos de referencia, material antropométrico, protocolo de medición, error de medición, composición corporal, somatotipo, proporcionalidad) y la relación entre la antropometría y el rendimiento deportivo.

**Más información:**
**www.femede.es**

# Guidelines of publication Archives of Sports Medicine

The ARCHIVES OF SPORTS MEDICINE Journal (Arch Med Deporte) with ISSN 0212-8799 it is the official publication of the Spanish Federation of Sports Medicine. It publishes original works on all of the aspects related to Medicine and Sports Sciences from 1984. It has been working uninterruptedly with a frequency of three months until 1995 and two months after then. It's a Journal that uses fundamentally the system of external review by two experts (peerreview). It includes regularly articles about clinical or basic investigation, reviews, articles or publishing commentaries, brief communications and letters to the publisher. The works may be published in SPANISH or in ENGLISH. The submission of papers in English will be particularly valued.

Occasionally communications accepted for presentation will be published in the Federation's Congresses.

The Editorials will only be published after request by the Editor.

The manuscripts admitted for publication will become property of FEMEDE and their total or partial reproduction shall be properly authorized. All the authors of the works will have to send a written letter conceding these rights as soon as the article has been accepted.

## Submit of manuscripts

1. The papers must be submitted, on the Editor Chief's attention, written in double space in a DIN A4 sheet and numbered in the top right corner. It is recommended to use Word format, Times New Roman font size 12. They shall be sent by e-mail to FEMEDE's e-mail address: femede@femede.es.
2. On the first page exclusively and by this order the following data will figure: work's title (Spanish and English), authors' name and surname by this order: first name, initial of the second name (in case there is), followed by the first surname and optionally by the second one; Main official and academic qualifications, workplace, full address and responsible for the work or first author's e-mail address for the correspondence. Also supports received for the accomplishment of the study -by scholarships, equipments, medicaments, etc- will be included.

   A letter in which the first author on behalf of all signatories to the study, the assignment of the rights of total or partial reproduction of the article, if accepted for publication shall be attached.

   Furthermore, attachment, the consignor will propose up to four reviewers to the editor may be used if necessary. In the proposed, one at least shall be responsible for the different nationality work. Reviewers signatory institutions work will not be accepted.
3. On the second page the summary of the work will appear both in Spanish and English, and will have an extension of 250-300 words. It will include the intention of the work (motive and aims of the research), used methodology, the most out-standing results and the principal conclusions. It must be written in such a way that it allows understanding the essence of the article without reading it completely or partially. At the bottom of every summary from three to ten key words will be specified in Spanish and English (keyword), derived from the Medical Subject Headings (MeSH) of the National Library of Medicine (available in: http://www.nlm.nih.gov/mesh/MBrowser.html).
4. The extension of the text will change according to the section to which it is destined:
   a. Original report: maximum 5.000 words, 6 figures and 6 tables.
   b. Reviews articles: maximum 5.000 words, 5 figures and 4 tables. In case of needing a wider extension it is recommended contact the journal Editor.
   c. Editorials: they will be written by order of the Editorial Board.
   d. Letters to the Editor: maximum 1.000 words.
5. Structure of the text: it will change according to the section to which it is destined:
   a. **ORIGINALS REPORTS**: It will contain an introduction, which will be brief and will contain the intention of the work, written in such a way that the reader can understand the following text.

      **Material and method:** the material used in the work, human or of experimentation, will be exposed, as well as its characteristics, criteria of selection and used techniques, facilitating the necessary data, bibliographical or direct, in order to allow the reader to repeat the experience shown. The statistical methods will be described in detail.

      **Results:** They report, not interpret, the observations made with the material and method used. This information can be published in detail in the text or by tables and figures. Information given in the tables or figures must not be repeated in the text.

      **Discussion:** The authors will expose their opinions about the results, their possible interpretation, relating the observations to the results obtained by other authors in similar publications, suggestions for future works on the topic, etc. Connect the conclusions with the aims of the study, avoiding free affirmations and conclusions not supported by the information of the work. The acknowledgments will appear at the end of the text.

b. **REVIEWS ARTICLES:** The text will be divided in as much paragraphs as the author considers necessary for a perfect comprehension of the treated topic.
c. **LETTERS TO THE EDITOR:** Discussion of published papers in the last two issues, with the contribution of opinions and experiences briefed in a 3 DIN A4 size sheets, will have preference in this Section.
d. **OTHERS:** Specific sections commissioned by the Journal's Editorial Board.

6. **Bibliography: i**t will be presented on sheets apart and will be shown by order of appearance in the text, with a correlative numeration. In the article text the quote's number will always figure between parentheses, followed or not by the authors' name; if they are mentioned, in case the work was made by two authors both of them will figure, and if there are more than two authors only the first will figure, followed by "et al".
There will not be included in the bibliographical appointments personal communications, manuscripts or any not published information.
The official citation for the journal Archives of Sports Medicine is Arch Med Sport.
References will be exposed in the following way:
   - **Journal: order number**; surnames and name's initial of the article authors with no punctuation and separated between them with a comma (if the number of authors is higher than six, only the six first will figure, followed by "et al"); work's title in its original language; abbreviated magazine name, segun the World Medical Periodical; year of publication; volume number; first and last page of the quoted extract. Example: Calbet JA, Radegran G, Boushel R and Saltin B. On the mechanisms that limit oxygen uptake during exercise in acute and chronic hypoxia: role of muscle mass. *J Physiol.* 2009;587:477-90.
   - **Book chapter:** Authors, chapter title, editors, book title, city, publishing house, year and number of pages. Example: Iselin E. Maladie de Kienbock et Syndrome du canal carpien. En : Simon L, Alieu Y. Poignet et Medecine de Reeducation. Londres : Collection de Pathologie Locomotrice Masson; 1981. p162-6.
   - **Book**. Authors, title, city, publishing house, year of publication, page of the quote. Example: Balius R. Ecografía muscular de la extremidad inferior. Sistemática de exploración y lesiones en el deporte. Barcelona. Editorial Masson; 2005. p 34.
   - **World Wide Web**, online journal. Example: Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis (revista electrónica) 1995 JanMar (consultado 0501/2004). Available in: http://www.cdc.gov/ncidod/EID/ eid.htm
7. **Tables and figures.** Tables and figures will be sent on separate files in JPEG format. Tables will also be sent in word format.
Tables shall be numbered according to the order of appearance in the text, with the title on the top and the abbreviations described on the bottom. All nonstandard abbreviations which may be used in the tables shall be explained in footnotes.
Any kind of graphics, pictures and photographies will be denominated figures. They must be numbered correlatively by order of appearance in the text and will be sent in black and white (except in those works in which colour is justified). Color printing is an economic cost that has to be consulted with the editor.
All tables as well as figures will be numbered with Arabic numbers by its order of appearance in the text.
At the end of the text document the tables and figures captions will be included on sheets apart.
8. The Archives of Sports Medicine Editorial Staff will communicate the reception of submitted works and will inform about its acceptance and possible date of publication.
9. Archives of Sports Medicine, after hearing the reviewers' suggestions (journal uses peer correction system), may reject the works which doesn't find suitable, or indicate the author the modifications which are thought to be necessary for its acceptance.
10. The Archives of Sports Medicine Editorial Boad is not responsible for the concepts, opinions or affirmations supported by the works authors.
11. Submissions of the papers: Archives of Sports Medicine. By e-mail to FEMEDE'S e-mail address: femede@femede.es. The submit will come with a presentation letter on which the work's exam for its publication in the Journal will be requested, the sent article type will be specified, and it will be certified by all authors that the work is original and it has not been partially or totally published before.

## Conflicts of interests

If there should be any relation between the work's authors and any public or private entity, from which a conflict of interests could appear, it must be communicated to the Editor. Authors shall fulfil a specific document.

## Ethics

All authors that sign the articles accept the responsibility defined by the World Association of Medical Editors.

The papers sent to the Archives of Sports Medicine Magazine for evaluation must have been elaborated respecting the international recommendations about clinical and laboratory animals' researches, ratified in Helsinki and updated in 2008 by the American Physiology.

For the performance of controlled clinic essays the CONSORT normative shall be followed, available at http://www.consort-statement.org/

# Campaña de aptitud física, deporte y salud


La **Sociedad Española de Medicina del Deporte**, en su incesante labor de expansión y consolidación de la Medicina del Deporte y, consciente de su vocación médica de preservar la salud de todas las personas, viene realizando diversas actuaciones en este ámbito desde los últimos años.

Se ha considerado el momento oportuno de lanzar la campaña de gran alcance, denominada **CAMPAÑA DE APTITUD FÍSICA, DEPORTE Y SALUD** relacionada con la promoción de la actividad física y deportiva para toda la población y que tendrá como lema **SALUD – DEPORTE – DISFRÚTALOS**, que aúna de la forma más clara y directa los tres pilares que se promueven desde la Medicina del Deporte que son el practicar deporte, con objetivos de salud y para la mejora de la aptitud física y de tal forma que se incorpore como un hábito permanente, y disfrutando, es la mejor manera de conseguirlo.

**UCAM Universidad Católica San Antonio de Murcia**

Campus de los Jerónimos,
Nº 135 Guadalupe 30107

(Murcia) - España

Tlf: (+34)968 27 88 01 · info@ucam.edu

UCAM
UNIVERSIDAD
CATÓLICA DE MURCIA