

Fasciitis or plantar fasciosis in the sportsmen? Treatment through Intratissue Percutaneous Electrolysis (IPE)

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Summary :

From the new anatomopathological paradigm, the chronic plantar fasciitis may be considered as a degenerative process. Therefore, it would be more logical to accept the new term of "plantar fasciosis" better than "plantar fasciitis". The Intratissue Percutaneous Electrolysis (IPE) will have a direct biological effect on the necrotic and fibrotic tissue. Through the different needle fenestrations and the chemical reaction which is produced, a liquifying and irritating effect will activate the inflammatory response, necessary to guarantee phagocytosis and the plantar fascia regeneration.

Key words : plantar fasciosis,regeneration,electrolysis ,percutaneous.

• Introduction.

This type of fasciopathy by repetitive microtrauma is more common every day in our daily sports clinic practice and many researchers have pointed that the most important thing in the pathology under these conditions is fasciosis or collagen degeneration. If we admit that plantar fasciopathy is due to a fasciosis process, not a fasciitis process, we will undoubtedly have to modify the therapeutic approach.

Plantar fasciopathy is considered as a typical functional overload fasciopathy, located in most cases in the insertion of the plantar aponeurosis in the medial and plantar tuberosity of the calcaneus. These data show us that the most vulnerable region to repetitive microtrauma is the osteo-aponeurotic joint, exactly the most complex histological region with the biggest sensitive receptor concentration. The biomechanical changes in the first radius area as a consequence of the hallux rigidus and/or alteration of the plantar vault are the endogenous mechanisms in the pop-up of this pathology in the sportsman. These changes are associated to the slowing down of the biological factors of scarring recovery.

In this essay, I introduce a new technique, the Intratissue Percutaneous Electrolysis (IPE), which I am practising since the year 2000 for the treatment of plantar fasciopathy and other pathologies in the connective and muscle tissue, obtaining brilliant results and reducing the recovery time of sportsmen. This method is based in the integration of the biological principles of connective and neural tissues, of the base system regulation, of the locomotor apparatus biomechanics and of its involvement in the soft tissue and in the basic principles of molecular pathology.

• Anatomopathology of the chronic plantar fasciopathy

The chronic plantar fasciopathy refers to a series of anatomopathological events scarcely related to an inflammatory process. The plantar aponeurosis is an intrinsic element of the connective tissue family and, consequently, its functions are the support, nutrition and mechanics. Due to its viscoelastic biomechanical properties and its anatomical location, this tissue is exposed to intermittent compression, tension and shear forces. This is the reason why the addition effect of intermittent microtrauma and the lack or recovery response may condition the appearance of the first pathological signs, even though its big resistance to tension forces.

If we observe through an electronic microscope the plantar fascia of a diagnosed patient in "chronic plantar fasciitis", we will observe a disorientation, a lack of organization and the separation of the collagen fibers, with the increase in the mixoide substance (semisolid substance in gel state made up of cell degradation and waste products) with increase of cell prominence and focused necrosis. But in a more detailed analysis, we will

observe hypoxia signs with the existence of lipolytic vacuoles which are characteristic of an anaerobic metabolic process (glycolysis) and as result of an anoxic extracellular environment with a very low oxygen tension.

In the insertion area of the bone-plantar aponeurosis, a fibrocartilagenous metaplasia can be identified, suggesting that the biochemical changes in the plantar fascia are largely produced by the lack of junction and cohesion of the neighbour cells. The ground substance, now in mixoide degradation state, acts as a true "fibrous-adipous glue" between the degenerated collagen fibers and the focused cells, creating a non physiological adhesion system which will alter the biomechanical functions and properties of the plantar fascia and the mechanisms of nutrition, scarring repairing and entropy which are needed for dynamic and normophysiological balance of the cell.

Cells present an anoxia state due to an acid pH as a consequence of the collagen fiber damage and cytotoxic substance release. The result of this process is the use of anaerobic metabolism due to the lack of oxygen and nutrients necessary to carry on the metabolic functions. Therefore, a glicolytic anaerobic metabolism will start at intracellular level with the following release of lactate. This substance is associated to lisosome´s waist products. This negative feedback effect generatives an environment create by the cell itself for self-destruction.

We do not find ourselves in a situation of "fasciitis", but of "plantar fasciosis", or more precisely, of an "alteration process of the connective tissue scarring repair mechanisms" (**Fig.1**). This fact could explain why some sportsmen suffer from plantar fasciitis, being submitted to the training rhythm, physical overcharge, and having the same morpho-structural features than others who don´t suffer from this pathology. Maybe the answer could be more related to the repairing mechanisms of the scarring tissue. There will surely be sportsmen who will have a higher threshold of their tissue repair mechanisms activation against repetitive microtrauma, thanks to their biological characteristics.



Fig 1: Plantar fasciosis .The degeneration collagen fiber process in the medial tuberosity of the calcaneus insertion area is observed. The failure in the inflammatory response produces a cascade of degenerative events on the dense connective tissue. Cyclical vascular ischemical processes and the afferent nociceptive hypenervation of the medial branches of the posterior tibial nerve could explain the extension of the plantar fasciopathy symptomes.

• **Clinic of the chronic plantar fasciopathy**

The sportsman with plantar fasciopathy is characterised by the located pain sensation in the posteromedial region of the calcaneus plantar face. Pain is involved with very similar episodes or stages to overload tendinopathy. In the initial stages, pain is relieved with the warm-up, but in advances stages, it becomes more persistent, even at rest.

By the time the problem continues and the chronification time is larger, pain appears in the plantar aponeurosis medial and lateral regions. We must remember that plantar aponeurosis sends medial extensions to the first toe abductor and lateral extensions to the fifth finger abductor. The central region of aponeurosis in its deep portion is upholsted by the short flexor muscles. In an advanced stage of the pathology, these muscle elements will be metabolically affected by the plantar aponeurosis affectation. Histologically, we will observe the appearance of collagen tissue rings surrounding these muscle fibers, and generating the typical muscle stiffness that we observe in the clinic.

This proliferation of collagen tissue in the muscle fibers extends to the base of the plantar aponeurosis, producing an alteration in the homeostasis and producing pain in the adjacent areas to the injury focus. The frequent presence of fibrotic nodules in the medial and central aponeurosis confirms this histopathological fact. Consequently, we will talk about a primmary located injury in the calcaneus insertion and a secondary injury as a result of the homeostasis alteration which is located in the medial and central region of the plantar aponeurosis.

We find again with the dilemma: if there is no inflammatory response and, therefore, there are no inflammatory cells which could justify the pain sensation, which are the non-inflammatory mechanisms that produce pain in the plantar fasciopathy?

Macroscopically, we can observe that patients with plantar fasciopathy are characterised by the presence of a soft consistence fascia of collagen fibers which lack of organization and which have a brownish yellow colour in their proximal portion next to the calcaneus insertion. This macroscopical appearance is described as a mixoide degeneration and through the microscope we can observe how the collagen fibers are disorganized and separated by an increase in the ground substance. Therefore, collagen degeneration, a variable fibrosis and a proximal neovascularization to the insertion are some of the constant discoveries in the up-dated studies. The mechanical model tries to explain the pain appearance in plantar fasciopathy according to two conditions: a collagen fiber injury and those who associate pain to a tissue impingement due to the existence of a calcaneus spur (Lian et al 1996; Cook et al 2000; Khan et al 1997).

Pain in plantar fasciosis could be caused by biochemical factors which activate the nociceptors (Kranshcar et al 1999). In the heels, nociceptors are located in the subcalcaneus bone region, periosteum and in the bone-plantar aponeurosis joint (mostly in the deep fascicle) and all these structures may play an active role in the plantar fasciopathy pain. Also, the P substance and the neuropeptids related to the P substance which are located next to the collagen fibers are involved in nociception. Through microanalysis technique, it has been observed the presence of lactate increase in the degenerated dense connective tissue. This increase in the lactate concentration in the plantar fascia show us the existence of anaerobic conditions in the plantar fasciosis area, a possible cause for pain.

This cyclic ischemical process stimulates the release of neural growth factor (NGF) and, consequently, the release of P substance, providing the nociceptive sensitive hyperinnervation instead of the insertion. On the other hand, periodical ischemical crisis in the plantar fascia may take place as the consequence of force vectors in tension and/or vessel shear of the proximal region to the calcaneus medial tuberosity, producing a vascular neof ormation with no physiological functions and an increase in the free sensitive endings. When there is an injury in the plantar fascia due to degeneration, the damaged cells and the blood vessels release toxic chemical substances which impact on the intact neighbour cells. One of these substances is the negative charge glutamate aminoacid, which produces a known process named excitotoxicity. When this degeneration in the plantar fascia releases large quantities of this neurotransmitter, overexciting the neighbour cells and allowing the big ions in, destructive processes take place. These discoveries reveal that glutamate may be involved in the plantar fasciopathy pain and emphathizes that there is no chemical inflammation, since the PGE2 levels are normal in these chronic conditions.

• **Treatment of the chronic plantar fasciopathy through the Intratissue Percutaneous Electrolysis (IPE)**

I am applying the Intratissue Percutaneous Electrolysis (IPE) since the year 2000 in chronic pathologies of the soft tissues (tendinopathies, neuromas, muscle fibrosis). Results are surprising because of their quick action mechanism on the injured tissue. In oncology, a similar technique is practised (electrochemotherapy) with certain parameter variations in order to improve the penetration of cytotoxic drugs inside the cancerous cells (Okino, M. 1987) .

During a lot of time it has been known that the use of galvanic current in a salty water solution produces a chemical reaction. The electrical current forces the splitting appart of salt (NaCl) and water (H₂O) into its two basic chemical elements. These elements regroup between each other to form completely new substances. This process is known as electrolysis. The new substances which are produced are sodium hydroxide (NaOH), hydrogen gas (H₂) and chlorine gas (Cl₂). In our case, gases produced lack of importance. But sodium hydroxide (or "organic bleach") is an effective destruction instrument when it is used on the region of the injured plantar fascia we are going to treat by degradation (fasciosis) because it is extremely caustic. The IPE is basically a chemical process where there is neither tissue "cooking" nor "electrocution". When we introduce one or several needles in the plantar fascia that we are going to treat, and we connect the electrical current, the salts in the interstitial tissue transform into "biological bleach" when they combine with the humidity of the ground substance itself. This bleach is the one which will produce the destruction in the tissue, and the inflammatory response for its repair.

All the ions produced during IPE look forward regrouping themselves quickly. We observe that chloride ions couple in stable pairs (Cl₂) to form chlorine gas molecules. In the same way, hydrogen ions match to form hydrogen gas (H₂). But the most important fact is that every sodium ion (Na) prones to combine with an hydroxile ion (OH) in order to form sodium hydroxide (NaOH). This will be our therapeutic tool, the "organic bleach" or "galvanic bleach" (**Table 1**).

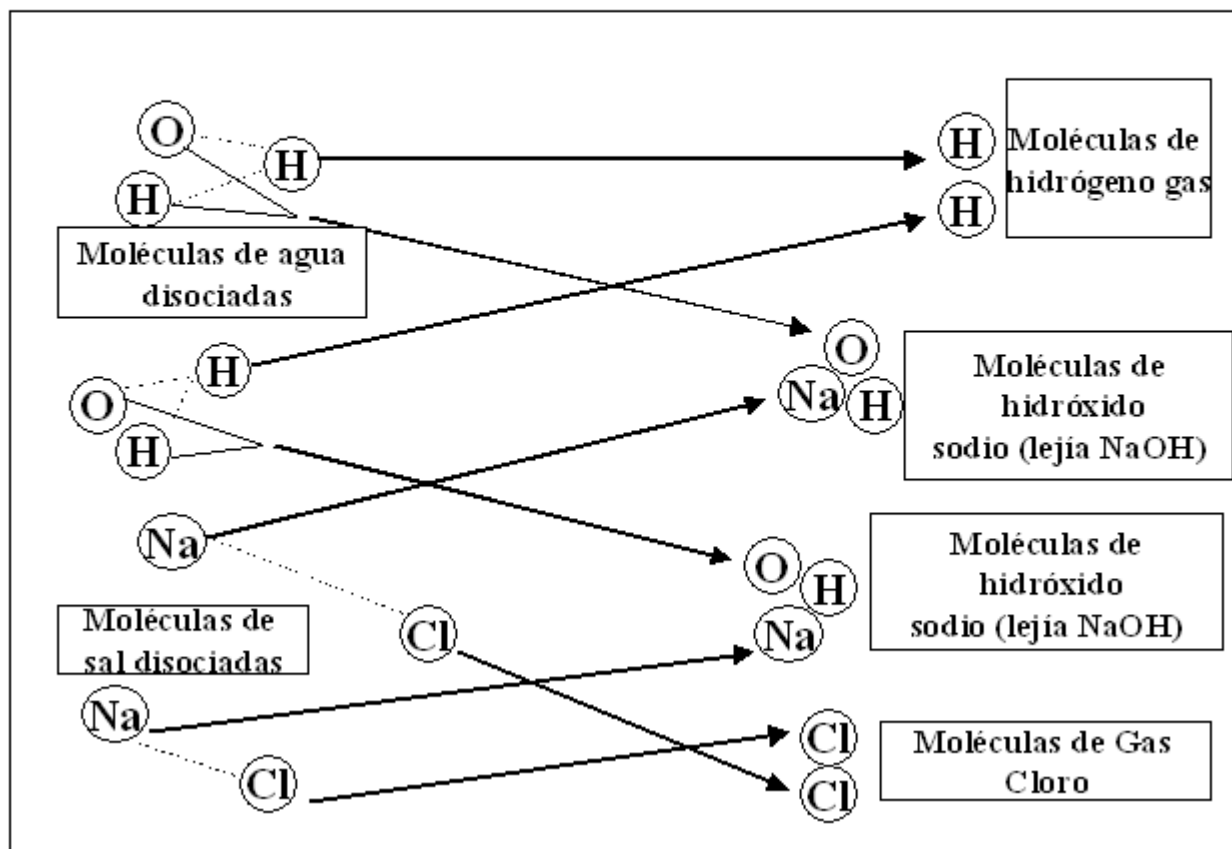


Table 1 : Biological action of of Intratissue Percutaneous Electrolysis (IPE). During IPE, two salt molecules and two water molecules regroup in one molecule of hydrogen gas (H_2), one molecule of chlorine gas (Cl_2) and two molecules of sodium hydroxide($NaOH$).

According to Faraday´s law, we can deduce that the amount of sodium hydroxide produced by IPE is the result of the current applied multiplied by the time along which the current flows (current x time = quantity of NaOH). The longer the time is and the bigger the intensity of the current, the more quantity of NaOH will be produced on the plantar

Effects of Intratissue Percutaneous Electrolysis (IPE) in the tissue regeneration

If we accept that patella tendinopathies with more than 3 weeks of evolution correspond to a degenerative process with fibrotic tissue formation, mixoide substance degradation and fundamental system hypoxia, the therapeutic approach must not be the same than within the inflammatory process. Scientific bibliography already highlights that NSAID treatment or corticoids infiltrations inhibitates the inflammatory cells migrating process (neutrophils, macrophages) which are necessary to activate the phagocytosis process. The depressed response of these cells will avoid fibroblast activation in order to generate new collagen and ground substance self-products (PGs,GAGs). These elements are necessary to reestablish the biological properties of the extracellular matrix surroundings. In this case of plantar fasciosis, we must consider as our main objeive to encourage the inflammatory response, so that the plantar fascia regenerative mechanisms are activated. IPE produces a chemical reaction in the symptomatic focus of plantar fasciosis, and starts a destruction process of the necrossed and fibrotic tissue. In the mean time, the direct contact of the needles with the fascia tendon produces the liquifying of the mixoide substance and the breaking down of the free protein bondings. This fact allows them to flow freely in order to be reabsorbed by the neocapillaries.

When we refer to the localised hyperinnervation in the medial tuberosity of calcaneus and insertion of the plantar fascia, the cathode polarity effect will produce a ionokinesis of the excitatory neurotransmitters freed by the cells during their destruction process. Ionokinesis stimulates the release of the nervous system extended despolarization with no harm for its rest threshold. We will force the destruction of presynaptic buttons produced by hyperinnervation through the caustic effect caused by the direct contact of the neddles to the free sensitive ending and interstitial tissue.

The biological effect produced by IPE is to guarantee the inflammatory response, which is the necessary response to reactivate the plantar fascia's regenerating mechanisms. The IPE dose is selected according to the application time and the intensity. Intensity must always be greater than time in order to guarantee a bigger generation of NaOH and, consequently, more caustic effect. By the time the tissue destruction is produced, an inflammatory response takes place. This reaction produces a neoangiogenesis of the near and sane capillaries which have not been affected by the electricity and which will start the invasion of the injury area.

The chemical mediators which are released by the injured cells (histamine, bradycine) play an important role to show the creation of the capillary network in the area affected. Neutrophils have a short life and they will be the first ones to arrive to the area where the iatrogenic injury is found. They will phagocyte the waste products released by the electrochemical destruction.

This migration and phagocytosis process is done within few hours the trauma takes place. This phagocytary cell migration is stimulated by the release of chemotaxic chemical substances in the injured area, in such a way that they act as informers of the injury location. This tracking establishes where the neovascularization must develop with its regular flow of nutrients and oxygen.

One or two days later, macrophages appear. These cells help to guarantee the continuity of the phagocytosis process. Macrophages play a basic role in the tissue healing: besides phagocytizing, macrophages stimulate fibroblast migration and they release growth factors. They also improve collagen synthesis and their proliferation.

The IPE application will have immediate direct effects on the tissue depending on the active electrode (**Table 2**). **When we use the cathode as active electrode, an irritation and destruction of the tissue will take place together with the liquifying of the mixoid tissue. This effect will produce a pH environment modification, stimulating the invasion of the injury focus, stimulating the oxygen and nutrients flow and, in short, normalizing PO₂.**

Aguja Anódica	Aguja Catódica
-Produce Ácido Clorhídrico -Acidifica el Ph	-Produce Hidróxido Sodio (Na OH) o sosa caústica ("lejía"). -Alcaliza el Ph
Destruye el Tejido	Destruye el Tejido
Desintegra las agujas de Acero	No tiene efecto sobre las agujas
Forma un tejido cicatrizal duro	Forma tejido cicatrizal sutil
Endurece y solidifica el tejido	Ablanda y relaja el tejido
Alivia el tejido	Irrita el tejido (respuesta inflamatoria)
Reduce el enrojecimiento	Promueve el enrojecimiento como vasodilatador
Germicida (destruye gérmenes)	Germicida (destruye gérmenes)

Tabla 2 : *Physiological effects produced in the body, depending on the cathodic or anodic needle.*
The needle number, size and diameter depends on the area we have chosen to treat. When we deal with

plantar fasciopathy, it is important to identify the entrance points in order to avoid any iatrogenic effect, as the medial branch of the posterior tibial nerve injury could be.

Before the intervention, we divided the plantar fascia into quadrants, introducing the needle in the deep portion of the plantar fascia insertion contiguous to the area of the calcaneus bone periosteum. The EPI treatment in plantar fasciopathy doesn't use to be longer than 60 seconds. It is very important to educate the patient to keep the homeostasis load sector or functionality limit. This is where a discharge orthosis will help us to achieve our aim (**Fig.2**). On the other hand, we must remember that the maximum peak of inflammatory response corresponds to the fifth day post-intervention. Within fifteen days, we can assure it will not exist inflammatory cell infiltrate in the injured region. This is reason why the patient may need a second or third IPE intervention, depending on the healing degree and the treated area. Once we introduce the needle/s in the point/s and select the correct angulation, we will verify the destruction of the tissue when the needle doesn't found elastic resistance on the cauterized tissue. The vasodilatation produced by the cathodic needle favours the diapedesis and, consequently, the neutrophil migration to the microtraumatized region.

In the area touching the needle, a lyophil effect is produced. The mixoide substance matter passes from its gel state to its sol state, much more fluid, to allow the uptake of the catabolits and to facilitate the surrounding pH and PO2 normalisation. Due to the intensity gradient, a contrairritation in the nociceptive endings is produced, associated to the destruction of the synaptic endings, normalizing the resting potential and inhibitating the accessory mechanism of extended depolarization. The polarity effect of the cathodic needle stimulates the removal and drainage of glutamate excitatory neurotransmitters, reestablishing the resting potential.



(click on the image to enlarge)

Fig 2 : Intratissue Percutaneous Electrolysis (IPE). Patient with plantar fasciopathy who is being applied IPE with multi-needles. The cathodic needles are introduced in the insertion of the plantar fascia in the calcaneus medial tuberosity.

En conclusión, los efectos bioterapéuticos de la Electrólisis Percutánea Intratisular (EPI) en el tratamiento de la fasciopatía plantar son excelentes, permitiendo una respuesta inflamatoria y reduciendo notablemente el tiempo de recuperación del deportista.

In conclusion, we can affirm that the Intratissue Percutaneous Electrolysis (IPE) biotherapeutic effects in the plantar fasciopathy are excellent, allowing an inflammatory response and dramatically reducing the recovery time of the sportsman.

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