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The role of Nitric Oxide (NO) in the pathophysiology of the Myofascial Syndrome

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ABSTRACT

This paper analyzes the role of Nitric Oxide (NO) in the pathophysiology of the Myofascial Syndrome, a pathological condition characterized by alterations of the physiological mechanical stress within Fascia and the Musculoskeletal system. In particular, two kinds of alterations are presented: Tendinous Myofascial Units, in which muscles are tetanically contracted and Fascia is relaxed, and Granulomatous Myofascial Units, in which muscles are extended and Fascia is contracted. In the last section examples of Myofascial Syndrome are reported.

Keywords: Nitric oxide, Myofascial syndrome, Pathophysiology, Stress, Myofascial units

INTRODUCTION

The Myofascial System is a network for the managing of the biotensegrity of an organism and whose task is to maintain a tensional homeostasis, related to the concept of chemical homeostasis (Chiera, 2017).

The human body is 'kept in shape' by a network of connective tissues and muscles forming the Myofascial System, arranged in Myofascial Units (MFU) which in turn can be subdivided in many different Myofascial Districts or, in simple terms, different muscles together with the myofascial layers associated to them.

The functions of many tissues and organs (e.g. the lungs, kidneys) are regulated by the internal stress state. The fascia bears structural loads through a special modality of contraction which reveals to be central in maintaining the posture and in regulating the body movements.

The Interstitium is one of the biggest organs in the human body, since it is widespread in the entire organism: under the skin and whithin the tissues that cover the digestive system, the lungs, the blood vessels and the muscles. It is made up by interconnected cavities full of liquid and sustained by collagen fibers (Fratzl et al., 1997 and Guatieri et al. 2012) and elastin, which form a microvacuolar structure acting as a proper damper. Its presence could also explain a lot of biological phenomena such as the diffusion of tumors, the aging of the skin, the inflammatory degenerative diseases and the mechanism of acupuncture.

A recent study published on the journal Scientific Reports by the New York University and by the Mount Sinai Beth Israel Medical Centre supports this statement. By means of a tecnique known as Laser Confocal Endomicroscopy, which enables to observe the living tissues directly inside the body instead of picking them up and then fixing them on a slide the real structure of the Interstitium has been found in all the parts of the body which undergo continuous movements and stresses.

The structural tensegrity is guaranteed by the disposition of the collagen fibers, which form aqueous structures inside the ExtracellularMatrix (ECM) (Tadeo et al., 2014). Such structures, called microvacuoles, are characterized by a high level of disorder and anisotropy. This is associated to a high level of entropy, which diminishes after the alignment along the main direction of stress in the case that stress acts along preferential directions. The energy absorbed by the system causes an increase in the structural order along certain directions and a change in the shape. The Fascial System guarantees maximum efficiency in managing the tensional homeostasis whenever the microvacuoles are disposed in the most entropic configuration.

In the case of an external traumatic cause breaking the tensional equilibrium within the myofascial system, Pathological Myofascial Contractures are generated, i.e. a pathological condition of fascial tissues' shortening which determines the myofascial

syndrome.

In this paper I put forward the hypothesis that the cause of the presence of Pathological Myofascial Contractures is disequilibrium in the physiological concentration of Nitric Oxide within the Myofascial Tissues

THE MYOFASCIAL SYNDROME

The Myofascial Syndrome is characterized by imbalances of the internal stresses of the Myofascial System due to the presence of Myofascial Pathological Contractures.

When a phenomenon of structural imbalance affects the human body, it is continuosly corrected with compensations by the Myofascial System, which modifies the distribution of the internal stresses changing its proper geometry and constitutive nature in terms of chemical, structural and morphological composition of the tissues and the cellular population in order to react to the imbalance of the internal tensional homeostasis. Such an adaptation is caused by variations in the chemical homeostasis following the mechanical mutations which become constitutive for the organism over time.

In turn, this is due to modifications of the tissues, which react either by getting stiffer or contracting depending on the tensional tenso-bending or compressive stresses which they undergo.

If we consider the role of fascia as a structural constituent along with the Myofascial Continuum, we can see that it adapts to the behavior of the muscular fibrocells with which it is in contact. In particular:

- Where the muscle is contracted, fascia is extended, causing a TendinousMFU, i.e. a Myofascial Pathological Contracture;
- Where the muscle is extended, Fascia is contracted (Schleip et al. 2005-2006), resulting in a Granolomatous MFU.

TENDINOUS MFUS:

Following a trauma, an entire MFU enters a state of continuous contraction. When a deformation is imposed, the stress relaxation in those visco-elastic tissues tends to diminish the internal stress and, even if the intensity of stress falls below the threshold perceived by the muscular mechanoreceptors also due to the damping caused by a more and more rigid ECM the deformation over time becomes chronic, so that the gaps between the contracted and circled muscle fibers are covered by the application of new connective tissue. Should the contraction persist over time, it takes the name of Myofascia Pathological Contracture.

The aim of this paper is to explain the role of Nitric Oxide in the creation of such pathological contractures.

The increase of NO concentration in the Myofascial System promotes the transformation of slow-twitch fibers in fast-twitch fibers (Petrogalli, 2019), which tend to contract in a tetanic manner, leading to the formation of ordered fascial structures which disrupt the histological structure and functionality of Fascia and eventually to a Myofascial Pathological Contracture which limits the relative slidings within the fascia itself.

The muscular fibers which belong to Tendinous MFU are contracted because of a malfunctioning in the behavior of the neuromuscular joint and of the biochemical milieu, due to the production of lactate (i.e. Lactic Acid, HLa), acting as a pro-inflammatory agent within healing processes and promoting the deposition of collagen fibers and angiogenesis (Figure 1).

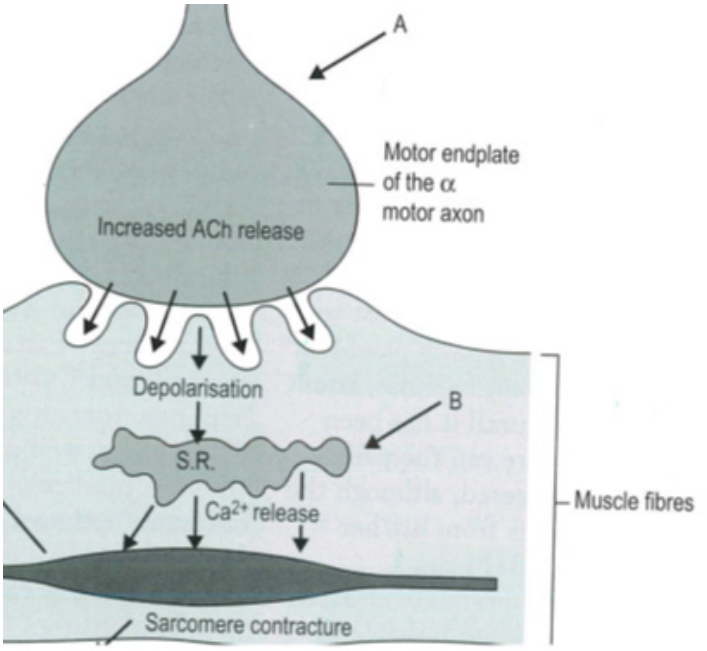

Figure 1: Model for the Myofascial Pathological Contracture.

In particular, capillary blood vessels are dilated due to the presence of Nitric Oxide and the muscular fibrocells work in anaerobic conditions, since this is the preferential way in which fast-twitch fibrocells work, despite the aboundance of Oxygen, due to the dilated blood vessels and the action of NO which tends to promote the oxygen supply to the cells (Doctor and Stamler, 2011).

This is supported by the fact that after manual treatment on Myofascial contractures an increase of Nitric Oxide in the blood is observed (Salamon et al. 2004).

In the case of a Myofascial Contracture, a more and more fibrous, extended and more liquid ECM is generated because of a continuous and reiterative remodeling process characterized by an inappropriate and excessive apposition of collagen fibers within the ECM. The excess of water, in particular, is responsible for the inflation of the tissues.

The metabolites produced in this district by the continuous contraction of the fibers accumulate and trigger stasis phenomena of acid substances. This, in turn, determines a condition of Metabolic Acidosis and a proinflammatory milieu, since the TRL-Toll

like Receptors warn the presence of non-self-toxins (e.g. ATP, Uric Acid) within the extracellular milieu.

The presence of an acid milieu also alters the muscular function, since a high concentration of H^+ inhibits the myofibrillar and sarcoplasmatic ATPase and reduces the re-uptake of Ca^{2+} ions.

Myofascial Pathological Contractures are accompanied by an overall increase of the myofascial stiffness of the affected districts; furthermore given the continuous and unceasing activity of gravity on each living organism over the whole life the situation on a biostructural level can only get worse due to the phenomenon of Hyperstatic Call: since the Myofascial System is a hyperstatic structure, the stiffest parts tend to bear most of the structural loads during exercise.

GRANULOMATOUS MFUS:

The Muscular Fibers of a Granulomatous MFU are subject to the opposite traction forces coming from Tendinous MFUs. This determines an increase in the concentration of the following substances (Shah et al. 2008): bradikynin, P Substance, Serotonin, Noradrenalin, Interleukins (e.g. IL-6, IL-8) and Histamine.

In a Granulomatous MFU, Nitric Oxide concentration is lower than the physiological one and this determines the contraction of the fascial tissues and of blood vessels togheter with an increase of the consume of oxygen, which determines a state of Hypoxia, causing the cells to release HIF-1, Hypoxia Induced Factor-1, a protein acting as a transcription factor for pro-inflammatory molecules.

EXAMPLES OF MYOFASCIAL SYNDROME:

Let's suppose to observe a patient who has been operated (osteosynthesis) and then plastered after a multiple fracture of the left radius. Let's suppose the course of the operation turns pathological because of the iron inserted for osteosynthesis damaging the tendon of one or more medial muscles of the

Forearm (e.g. Brachioradialis) and causing an inflammation which is treated by numerous medications in the insertion area of such muscles (medial area of the wrist).

Since the inflammation persists, the surgeon chooses to remove the plaster after twenty days instead of a month and the inflammation goes away within few days thanks to the structural compensation which takes place at the Fascial level. As a consequence, a Myofascial Pathological Contracture of the antagonist MFUs (e.g. Flexor Ulnaris Carpi, Palmaris Longus, and Flexors of the fingers...) occurs.

This may be a case study in which tensional imbalances are generated as a result of surgical operations therefore labeled as 'traumatic' which have not been carried out to perfection. In such cases the Brachioradialis Muscle features a Granulomatous MFU, along with the Synergistic MFUs (e.g. the MFUs in the Extensor Pollicis and Radia and the Brachial Muscle). On the other hand, the Flexor Ulnaris Carpi works as a Tendinous MFU in which the contracted muscular fibrocells remain in a state of continuous contraction.

The following image describes a further example in which the patient keeps his head forward with the chest bending inwards due to the curved back.

The upper muscle fascicles of the Trapezium Muscle undergo a displacement due to an eccentric load caused by the weight of the head, while the single fibrocells are subject to extraphysiological stresses of traction; we can observe, in this district, a clear example of a Granolomatous MFU. The muscular fascicles of the Front Deltoid are contracted due to a concentric load and show a Myofascial Pathological Contracture, causing the district to become a Tendinous MFU (Figure 2).

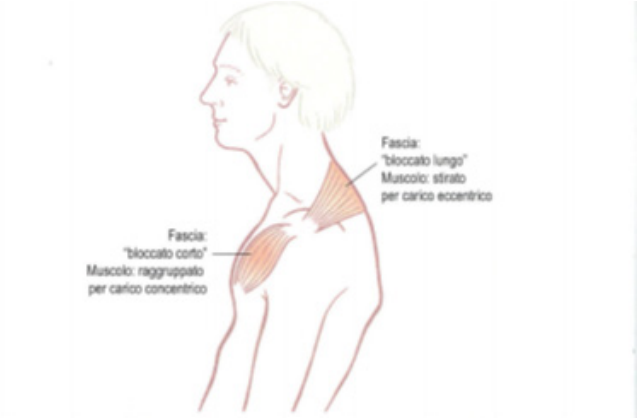

Figure 2: Example of Myofascial Syndrome in which a muscle is stretched due to eccentric load, while another one is short due to concentric load.

CONCLUSION

Myofascial diseases, also called Somatic Disfunctions (Tozzi, 2015), are characterized by pain, reduced articular mobility and damage to neighboring tissues (e.g. skin, muscles and tendons). Such discomforts, especially in case they are particularly serious, can lead to modifications in the constitutive morphology of tissues and in the shape of the organism and eventually in its physiology. In other words, because of the imbalance of the tensional homeostasis which characterize the somatic dysfunctions the neuroendocrine activity may undergo modifications which affect the functioning of the internal organs controlled by a retroaction feedback regulated by chemical mediators such as hormones and neurotransmitters.

To sum up, in a state of complete relaxation the Tendinous MFUs (where a Pathological Fascial Contracture occurs) are short, while the Granulomatous MFUs are extended. The Tendinous MFUs are in a condition of local metabolic acidosis, while the Granulomatous MFUs experience alkalosis; the two biochemical thrusts in opposite direction with respect to pH are counterbalanced according to the concept of Allostasis, without giving rise to systemic alteration of pH values. The cronical interstitial inflammation characterizing the Generalized Myofascial Syndrome allows the pathology to feed itself: as a matter of

fact Nitric Oxide has a crucial role in triggering and maintaining a proinflammatory status.

The compression of the neurovascular tract in fascial disfunctions contributes to the imbalance of the chemical homeostasis, not only from a functional point of view that is, the biochemical milieu altered by chemical stress alters in turn the behavior of certain organs but also mechanically, with circlings (following extraphysiological stresses of traction) or bulkings (following extraphysiological stresses of compression) of the biological structures in the Extracellular Matrix.

In the presence of Myofascial Pathological Contractures, the abdominal and lumbar areas are weak because of the weakness of the Diaphragm which determines a smaller toracic expansion and an altered central control of breathing. Fibrocontractive diseases can also cause adhesions of the structure which make up the abdominal wall.

From a biomechanical perspective, the diaphragmatic disfunctions determine a decreased articular ROM of the cervical tract and a more marked tensional state in Crural Fascia. This condition affects the whole Fascial System with a chronic state where an increase of Nociceptive Fibers and a loss of the motor coordination without apparent causes of neurological type occur. From the endocrinological point of view, on the other hand, the altered mobility of the Diaphragm within a Generalized Myofascial Syndrome can generate a hormonal disorder, given the close relationship of the muscle with the pituary-adrenal axis (HPA) which controls the whole endocrine production of the organism. As a result of imbalances and structural compensations, the behavior of the paravertebral muscles and, consequently, of the Fascia Profonda of the trunk, are altered. This alters the proprioception and nociception, determining the disfunction of the Autonomous Nervous System and an impairment of the functioning of the afferent autonomous post gangliar fibers, thus resulting in alteration of such phenomena as the blood vessels vasoconstriction and the hyperhidrosis of sweat glands.

To conclude, I claim that the molecule responsible for the arisen of extraphysiological mechanical tensions which caracterize the Myofascial Syndrome is Nitric Oxide (NO).

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