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MYOREFLEX THERAPY: THE DYNAMICS OF KNEE AND GROIN PAIN IN PROFESSIONAL SOCCER PLAYERS

Physics is inscribed within the great book that lies open before our eyes…But we can only begin to read it after we have learned its language and become familiar with the symbols in which it is written. (Galileo Galilei)

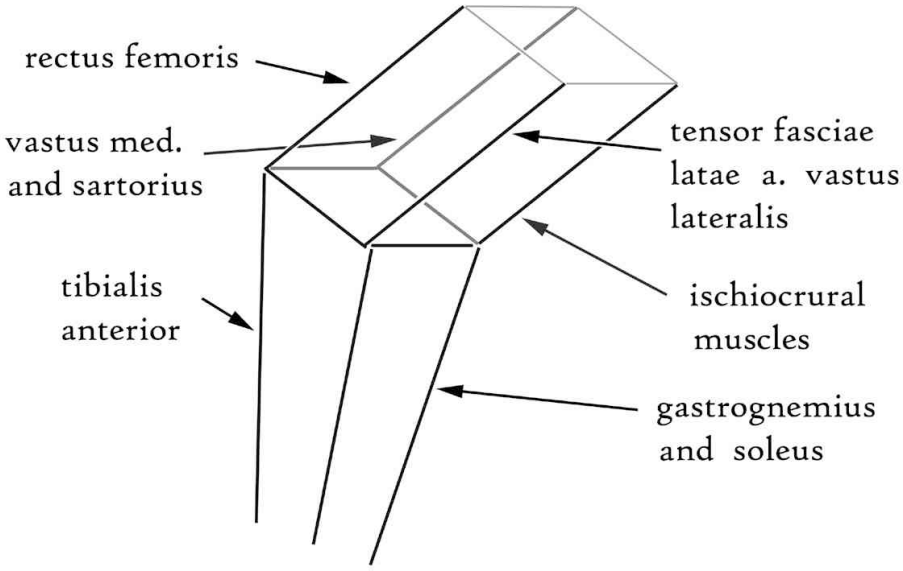
Microtrauma

Improper loading or excessive stress combined with insufficient regenerative intervals can lead to changes in muscles and tendons. Insufficient elasticity, insufficient relaxation capacity, increased tone, non-optimal performance parameters and muscle stiffness thereby initially result in reduced resilience. If the relative improper loading continues unchanged, then degenerative changes are likely to develop. Recurrent excessive stress then establishes manifest microtraumatic changes, with muscle pains, periosteal irritation, and irritated muscle-tendon junctions manifesting as insertional tendinopathies. Aseptic inflammatory changes, improper loading and excessive stress with insertional tendinopathies and microtraumas are all potentially reversible with appropriately precise, targeted and individually tailored treatment.

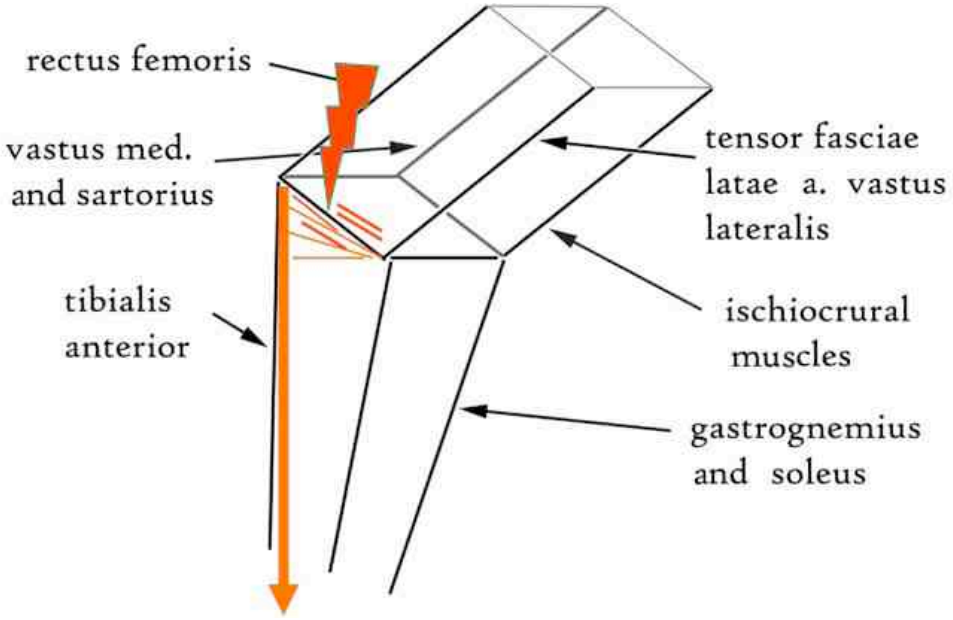
In this process, it is quite difficult to determine the stress capacity and individual properties of individual components such as ligaments, tendons, muscles and joint and bone structures. Great differences may exist within a single individual and between individuals, along with individual asymmetries resulting from previous injuries or stereotyped stress patterns and special stresses dependent upon movement. An individual's capacity to respond to medical treatment is just as wide-ranging as the variability of time factors.

From the perspective of biomechanical laws, vector calculations based upon deductions from movement geometry lead to a completely new level of understanding – and thereby to new treatment and optimization strategies. The dynamics of the muscular system and neuromuscular synchronization in kinetic synergist and antagonist chains precisely regulated for all functions makes it possible to gain a more profound understanding of the passive elements in the movement apparatus (ligaments, tendons, cartilage and bones). In complex three-dimensional tension band systems there are fixed points, moments of static loading and dynamic movement with pressure, traction and relatively force-free nodal points.

In the context of this *relative movement model*, it is no longer "pain points" or local stress areas that are the focus of our attention, but instead the passive elements that hold back the actors in their impulse to move. This concept will be presented here in simplified form using the example of the knee joint.



The framework, dynamics and every activity within the three-dimensional vector analysis of the thigh and the knee joint are all highly dependent on the position of the pelvis, the iliosacral joint and the lumbar spine. When there are changes in the relative length of the active muscle fibers (raM), relative shortening or elevated resting tone of the iliopsoas muscle system in its over-trained middle working range, then all vectors of the lower extremity and their relative angles are altered. Stereotypically over-trained abdominal muscles together with rectus femoris sections of the quadriceps muscles that are relatively over-trained in their middle working range synergistically lead to an escalation and overt manifestation of these vector and angle alterations in muscle origins and insertions. These functional changes also affect the ligaments and the tendon apparatus and have a structural impact on the meniscus, the cartilage, and the joint. Sections of the iliopsoas muscle that are not optimally trained and restricted thus have a direct impact on the rectus femoris muscle —with nodal points at the anterior inferior iliac spine (groin) or the upper (rectus insertion) or inferior (patellar ligament) pole of the patella.



Even when considered only from the perspective of the direct kinetic chain, the problem of patellar tendonitis (jumper's knee) and weakness in the anterior cruciate ligament can be seen to be located not in the patellar pole but can be calculated back to the anterior inferior iliac spine or the iliopsoas muscle. The passive element of this chain that is responsible for the release of ventral activity is thus the ischiocrural muscles. Relatively restricted ischiocrural muscles functioning as "brake levers" can be sensed as a signal in the active element, that is, in the location where tension should occur.

The stereotypical presentation of problems in the ischiocrural group, which is frequently less adequately trained in professional soccer players than the quadriceps muscles, can be manifested in the control of the medial meniscus as meniscus signs, as medial knee pain or may be expressed antagonistically in the area of the patella and the rectus femoris muscle. Even alterations in a single vector lead to asymmetries, changes in angles, and to high stress points in the entire force parallelogram and in the passive portions of the meniscus system, the anterior and posterior cruciate ligaments and cartilage. Loss of symmetry of tone and disturbances in the geometry of movement then lead to deficits in the economy of motion, dexterity, agility, speed, strength and performance capacity.

The tensor fasciae latae muscle is an important physiological synergist of the rectus femoris muscle. The angular changes in the pelvis described earlier, accompanied by restrictions in the iliopsoas muscle, can lead to a reversal of function in the tensor fasciae latae muscle. Either directly or indirectly by means of shortened portions of the tensor muscle at the lateral anterior superior iliac spine, this vector can lead to lateral pain in the knee joint, patellar movement disorders (lateral displacement) or to pseudo hip pain. Because of the change in angle, many fibers of the tensor fasciae latae muscle now work as flexors in the knee joint. In extreme cases, this alone can lead to extension lags in the knee joint of up to 18°.

Groups of fibers in this muscle actually end up working against each other as their own antagonists. Very clearly, the causes of this symptom are not located in the spot where it hurts, but in the asymmetrical geometry of movement—in the passive element. Through activities of the passive element, changes in the tensor fasciae latae muscle may sometimes show up in the medial knee joint, in the control of the medial meniscus (the semimembranous muscle) or in the adductors. Thus, pain, muscle strains, irritation, inflammation, and insertional tendinopathies of the adductor system may not be primarily caused by the adductors themselves, but instead by the tensor muscle, its iliotibial tract and the iliopsoas muscle.

Acute and chronic sports injuries, muscle strains, inflammation and insertional tendinopathies most often do not require treatment directly where the symptoms are located. The classical strategy of orthopedics sports medicine, which is to treat the problem at the site of symptoms, to place the site at rest, obtain radiographs, use anti-inflammatory injections and perform operative interventions (discissions, surgical notching) frequently prove inadequate as therapeutic methods. Degeneration and sports injuries such as ruptures, ligament tears, and meniscus lesions are thus first and foremost the consequences of misunderstanding the nature of the problem and using shortsighted local interventions. As a rule, local therapy for

the weakest member of a chain in the presence of an excessively short leverage arm ends up causing excessive stress and overstimulation.

In the best case, signal symptoms may be suppressed, concealed or disguised. The result is the development of asymmetrical restrictions of the musculature, damage to the running economy and strength capacity as well as deficits in all areas of regeneration. Recurrent, degenerative damage and more serious injuries resulting from even small force impacts, sometimes seeming to appear from nowhere, are the logical consequences of these persistent asymmetries.

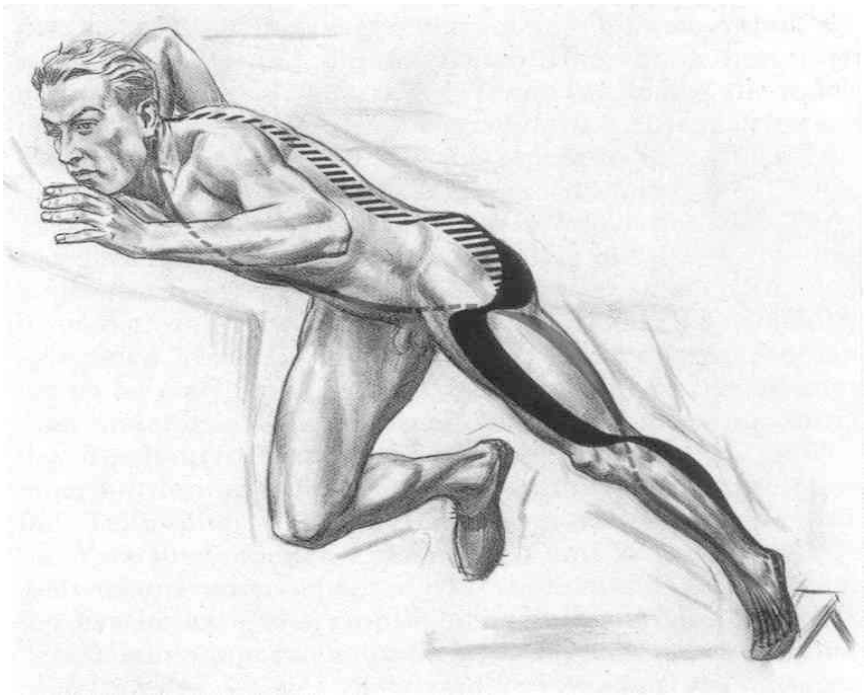
Pulled muscles, tears, and damage in the domain of muscles, tendons, cartilage and bones frequently take place on the basis of non-local alterations. One-dimensional considerations of reduced biomechanics (see Bauman 1989), such as shin-ankle-foot calculations, analyses of individual properties, measurement of the visco-elasticity of a tendon, measurement of local metabolism) are not sufficient to meet the needs of professional soccer players. Theories that focus on local pathological changes to conceptualize etiology, pathogenesis and treatment planning are frequently reductionistic to an extent that is hard to believe. As a result, chronic sports injuries in particular have often remained unexplained until now. Thus, apophyseal tears in the rectus femoris muscle or the ischiocrural muscle in professional soccer players are not instances of simple structural overload, but instead the consequences of dysfunctional biomechanics.

Injury prevention

"I visited Hoffenheim on one of those days when the training area next to the gas station at the entrance was once again transformed into an international pilgrimage destination … Dr. Mosetter from Bodensee happened to be the guest of the physiotherapists. While the Myoreflex Therapist was hanging out around defenseman Andreas Beck, he explained that many professional soccer players lost speed simply on the basis of incorrect body posture and at the same time, became more injury-prone." (Christoph Biermann. Die Fußball-Matrix - Auf der Suche nach dem perfekten Spiel. [The Football Matrix: the quest for perfect play])

The most important preventive measure is optimized biokinematic training. Besides classical sports medicine examinations, this also includes "point of weakness analyses" – not merely with regard to structural functions but also for vector analyses of the geometry of movement. Therefore, based upon the specific nature of the sport as well as attention to the individual's point of weakness, strength training must be directed at the weakest link, the passive element, the direct and indirect antagonist, and must improve its strength.

These measures not only promote injury prevention, but also optimize performance. Besides the running economy, this affects all strength capacities, both aerobic performance capacity and capacity for recovery. On the ground power reaction capacity, maximal velocity of contraction and relaxation half-life can all be improved significantly in this way. Thus, just the extensors and the quadriceps muscle all by themselves are not sufficient for sprints and running speed during the first 10 meters.



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Starting from preparatory activation of all involved joints in a fixed position, connected to a certain degree of pre-stretching of the extensor muscles of the back (for both crouched and running starts), entire groups of muscles are preactivated. The sudden explosive contraction of the extensor system involves the synchronous contraction of back extensors all the way to the occiput. Not only the flexors of the upper thigh and the ischiocrural musculature, but also the calf musculature and the flexors of the trunk, the iliopsoas muscle and the abdominal muscular system must all "unleash the explosion" together during this process.

Constriction of these muscle groups and loops has the effect of limiting the passive element and thus "braking" acceleration and contraction. At the same time, it alters the body's center of gravity (in the area of the sternum) away from its ideal position. With regard to this parameter of the dynamic vector calculation, a number of muscles play important collaborative roles, including the chest muscles (the pectoralis major and minor), the serratus anterior muscle, the insertions of the rectus femoris muscle up to the 5th intercostal space, the fibers of the internal abdominal oblique muscle to the lower costal arch and the diaphragm. Asymmetries and constrictions along with single-sided overtraining of the chest and abdominal muscles thus restrict both overall acceleration, the acceleration of the body's center of gravity, maximal velocity, the targeted velocity of contraction as well as "slackness" due to alterations in the overall vector networks.

Dysfunctional neuromuscular tensions in the area of the atlanto-axial joints and the cervico-thoracic insertions (especially through the scalene muscles) also enter into the vector equation, as if by a secondary neurogenic innervation substrate with inhibition of the phrenic nerve and its controlling function for the diaphragm. Not least, the collateral movements of the arms (in terms of their looseness and capacity for relative take-off movements) also have an impact on aspects of performance optimization with accelera-

tion, speed, and maximal velocity potentiality. Asymmetric hypertonicity and stiffness without elasticity shut have a restraining effect upon all elements of performance. Sprinting speed, the capacity for repetitive sprints, initial acceleration and sprint-to-stop capability thus turn out to be dependent variables upon a multi-dimensional asymmetry of tone in a three-dimensional vector network.

Muscular Dynamics of Goal Shots

Goal shots involve very high tensile loads, pressure loads and twisting loads for the knee joint in the supporting leg. In this position, the entire vector equation of the muscular system is required to provide the supporting leg with stability, support, traction and equilibrium functions, and at the same time to provide the active leg with mobility, acceleration and maximal dynamic activation. In the trunk, there are activities in lateral inclination with simultaneous rotation of the trunk.

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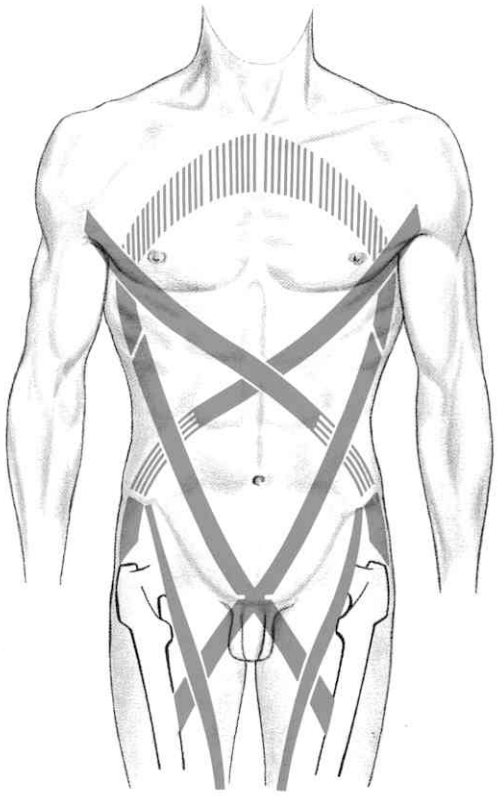
Some of the important kinetic chains and their muscle loops should be specified at this point:

1. Levator scapulae muscle – serratus anterior muscle– internal oblique muscle, pubic intersection (with the rectus abdominis) – supporting leg adductors– peroneus longus muscle, biceps brachii muscle on the side of the supporting leg.

2. Pectoralis major and minor muscles – internal abdominal oblique muscle– tensor fasciae latae muscles – gluteus medius muscle in the active leg–anterior tibialis muscle–levator scapulae muscle.
3. Triceps muscle – Serratus anterior on the supporting leg side–external abdominal oblique muscle – junction of the pubic bone with the rectus abdominis muscle– adductors in the active leg– peroneus longus muscle
4. Extensor hallucis longus muscle – extensor digitorum longus muscle – tibialis anterior muscle – rectus femoris muscle – rectus abdominis muscle –sternocleidomastoid muscle
5. Trunk rotation in the direction of the internal oblique muscle – internal abdominal oblique muscle – rectus sheath
6. Passive element of the active leg: plantaris muscle – Achilles tendon – gastrocnemius muscle – biceps femoris and semimebranous muscles – gluteus maximus muscle – iliocostal muscle – erector spinae muscle (contralateral) – semispinalis muscle – splenius cervicis muscle
7. Passive element of the supporting leg: Tibialis posterior and soleus muscles – tensor fasciae latae muscle – internal abdominal oblique muscle – quadratus lumborum muscle –latissimus dorsi muscle (contralateral) – biceps brachii muscle – flexor carpi ulnaris and radialis muscles



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The contractile power of each individual component along with the synergistic activity of individual parts of the kinetic chain as well as the stretch capacity and relaxation qualities of the immediate antagonists and the antagonist chain are all critical for smooth coordination in dynamically synchronized movement sequences. Super-fast anticipatory synchronization is made possible in this way, and can be summoned at will; it guarantees exceptional performance. Besides the principal vectors that have been described, this beautifully exemplifies the individual anatomical vector chains, organized in such an amazing way in the abdominal muscle system and its connections with the serratus anterior muscle.

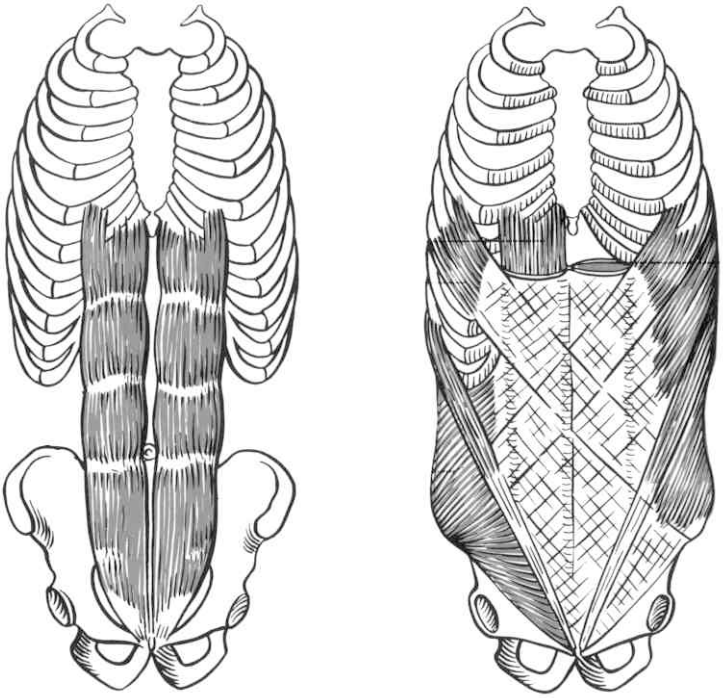
Groin pain

Chronic groin problems are among the most common of soccer injuries. The symptom of groin pain can be caused by a number of different pathologies in this setting. Besides insertional tendinopathies caused by strains in the musculature that inserts in the pubic bone (the pectineus, adductor longus, gracilis, and the rectus abdominis muscles), the most commonly occurring factors in the genesis of groin pain are hernias, urological abnormalities, neurological causes, ailments affecting the pelvic ring and the hips, and more rarely, tumors or lymph node abnormalities. These must be elucidated by means of a process of differential diagnosis.

The groin region is the site of insertion for the powerful muscle groups of the iliopsoas, the rectus abdominis, and the abdominal oblique muscles (both internal and external), the adductor muscles, etc., which are all placed under great strain in soccer and transmit high moments of force to the hips. At the same time, the area of the groin represents a soft, sensitive structure, which serves as a passageway through its fascial openings for blood vessels, nerves and the spermatic cord in men. Since this complicated structure so often goes out of whack in intense athletic activity, the groin region is often described as an anatomical point of vulnerability.

Once complex pain syndromes are considered from the perspective of biomechanical vector equations and one undertakes a precise analysis of the synergistic and antagonistic force vectors, causally based rational therapy for persistent pain syndromes becomes feasible. Very often, *earlier injuries discovered from the biographical history provide guidance* for understanding current symptom presentations. Hidden compensatory postures and injury-specific forms of compensation may thereby result in individual points of vulnerability dependent upon a person's personal history; and these points may well be located in a *totally different place.* New injuries very frequently must be understood as the result of decompensation in a vector model that was already slightly constrained in its flexibility.

Groin pain, muscle cramps in the muscular systems of the groin, insertional tendinopathies, elements of "athletic pubalgia," (also known as "sports hernia") inflammation in the groin area and functionally relevant points of weakness in the vector network along with hernias may thus be clearly and fundamentally understood according to the laws of clinical anatomy and through the biokinematics of vector equations. From an understanding of the clinical anatomy, it is possible to directly infer an entire range of vector candidates for each particular problem.



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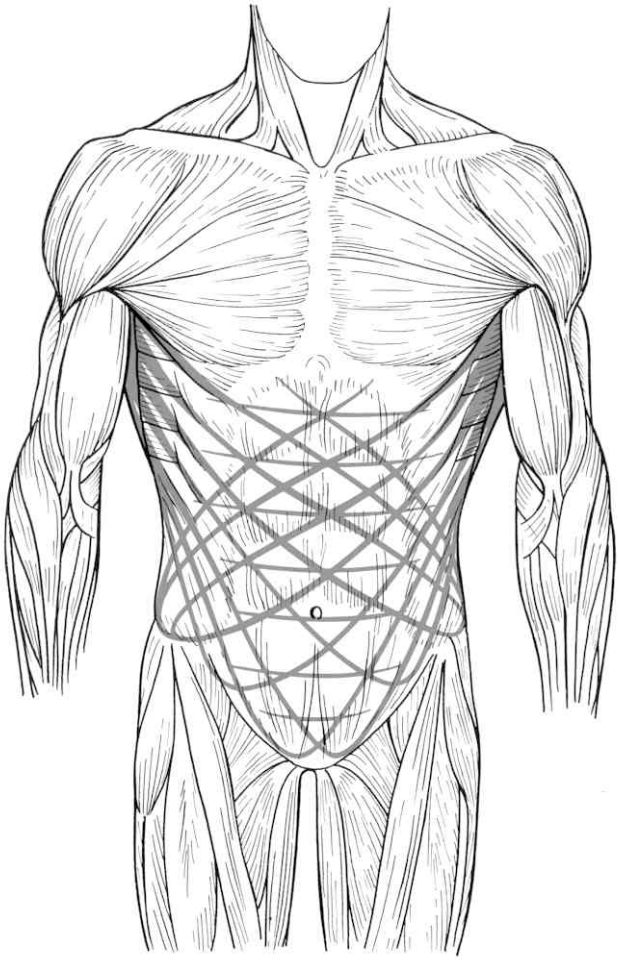

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I. Active Element

1. Iliopsoas muscle – in the course of its passage under the inguinal ligament to the lesser trochanter (see figure above)
2. Pectineus and adductor brevis muscles (pubic ridge) – pubic tubercle – lesser trochanter
3. Internal oblique abdominal muscle (intermediate zone of the iliac crest and lateral inguinal ligament) – anterior superior iliac spine– inguinal ligament – cremaster muscle
4. External oblique abdominal muscle– linea alba of the aponeurosis – inguinal ligament – pubic tubercle
5. Transverse abdominal muscle– aponeurosis – cremaster muscle – tuberculum
   ↓
   Internal abdominal oblique muscle
6. Rectus femoris muscle (anterior inferior iliac spine)

(Mosetter and Mosetter 2006)

II. Passive Element

1. Tensor fasciae latae muscle
2. Gluteus medius muscle
3. Gluteus maximus muscle
4. Quadratus lumborum muscle
5. Piriformis muscle and internal obdurator muscle
6. Ischiocrural musculature
7. Contralateral abdominal muscle group
8. Contralateral shoulder girdle

Differential analysis

1. The specific muscle vectors responsible for the symptoms in each individual case must be analyzed precisely.
2. Optical determination of postural patterns
3. Manual functional orthopedic examination
4. Palpation
5. Precise historical review regarding the initial occurrence of the symptoms (associated with which function, with which movement sequence)
6. Analysis of the principal individual passive element
7. Association of pain intensification with specific functions
8. Vector analysis of the prior history, pain, relieving posture — compensation

Specific Biokinematics in "athletic pubalgia"

Groin pain and "athletic pubalgia" are very common symptom complexes diagnosed in professional soccer players whose causes are difficult to pinpoint. From the viewpoint of classical therapeutic strategies, one must ask how it could be that apparently well-trained young athletes should have such a tendency to develop this condition. In the tension band model of biokinematics, all "weaknesses" or "gaps"

are at the same time associated with contracted, tense fiber lines. This understanding leads to new solution strategies based upon clinical anatomic pathways.

From the perspective of physics and biokinematics, there are three-dimensional vector systems throughout the body. The anatomical and functional-kinematic architecture of the groin and its vector model are the basis for understanding a broad range of symptomatology, including groin pain, "athletic pubalgia," and groin hernias. Wall structures composed of tension band systems along with physiological openings play an important part in the conception of potential new ideas for treatment.

Together with the fiber insertions of the transverse abdominal muscles, the internal abdominal oblique muscle forms the roof of the inguinal canal. In this process, the abdominal oblique muscle releases fibers for the formation of the cremaster muscle in the spermatic cord. The anterior wall is formed by the aponeurosis of the external abdominal oblique muscle and the superficial abdominal fascia. At the same time, these belt components also form the inguinal ligament with their cranial portion and delimit the external (superficial) inguinal ring. Besides a portion of the external aponeurosis, the tight interweaving with the fascia latea of the upper thigh helps to create the structure of the caudal floor. The dorsal wall structures are formed by the tight interweaving of the transversus abdominis muscle, the fascia transversalis with its invagination in the inguinal canal, the interfoveolar ligament, and the peritoneum.

"The aponeuroses of the lateral abdominal muscles surround the anterior straight abdominal muscles like a quiver on both sides and, together with the fascia of the abdominal wall, form the rectus sheath. The muscle compartment constituted in this manner includes an anterior and a posterior leaf (lamina anterior and posterior). While above the navel, the aponeuroses of the lateral abdominal muscles take part equally in the formation of the lamina anterior and posterior, both leaves combine together about 3-5 cm below the navel (at the level of the linea arcuata) into a single (and thus stable) leaf, which pulls in front of and along the rectus abdominis muscle. Below the linea arcuata, the posterior leaf of the rectus sheath is thus formed only by the fascia transversalis and the peritoneum." (Schünke et al. 2005)

The Differential Diagnosis of Groin Hernias

a.) The anatomical point of weakness is Hesselbach's triangle–the hernial gap for direct inguinal hernias. It is situated under the epigastric vessels and medial to the inguinal ligament.

b.) The inguinal canal represents a second point of weakness for "ruptures" within the abdominal wall.

c.) The internal inguinal ring above the inguinal ligament and lateral to the epigastric vessels represents the weakest anatomic point for indirect, lateral inguinal hernias.

d.) Femoral hernias run along their internal hernial path below the inguinal ligament through the lacuna vasorum. The description of more specific details will be omitted in this kinematic and sports-related dynamic vector consideration (details in Schünke et al. 2005, p. 182-191.).

e.) Extremely rarely, dorsal inguinal hernias penetrate through the muscular transitional zones between the quadratus lumborum muscle and the dorsal portion of the internal oblique muscle.

f.) Occasionally, and especially in women after pregnancy or surgery, residual scarring situations may also develop into umbilical hernias.

Managing points of weakness

For Myoreflex Therapy, the relevant function-dependent and training-dependent points of weakness along with their treatment are defined as disturbed or asymmetrical vector equations with angular deviations within a three-dimensional parallelogram. Therefore, every point of weakness or relatively unstable gap is coupled with horizontal tensed connecting bands within the muscular vector model. Muscles in the middle working range of the ventral abdominal musculature that are relatively over-trained on a chronic basis become shortened, leading to excessively high power peaks at specific levels and ventral restriction. At other levels of possible strain, rotational levels and muscle vectors crossing horizontally at a 90° angle thereby experience relative weaknesses in their function.

For treatment of the groin, the external oblique muscle represents the first priority. Laterally, at the insertion of the aponeurosis of this muscular system into the pubic bone and above it, the fibers diverge and thus contribute to the formation of the gap in the external inguinal canal, the superficial inguinal ring. The internal abdominal oblique muscles create both the rectus sheath and are interwoven with the aponeurosis of the external abdominal oblique muscle, and likewise with the transversus abdominis muscle. The latter radiates at the same time with the upper portions in the posterior wall of the rectus sheath, with the lower portions in the internal abdominal oblique muscle and with the anterior leaf in the anterior portion of the internal abdominal oblique muscle. The outer groin, the inguinal ligament and the inner groin are thus interwoven into an ingenious elastic dynamic tensed belt system. The many layers of this vector network guarantee its stability, elasticity and seamlessly close all gaps. Stability and dynamism are not maintained by the individual parameters of the individual properties themselves, but instead achieved by means of the multiple layers of the tensed belt system. Soft, hard, strong or weak all depend directly upon the physically balanced conditions of the geometry of movement with its muscle vectors.

Under asymmetrical conditions, with relative shortening and corresponding angular changes, points of weakness arise along with "soft gaps." In particular, the shortening and stiffening of a portion of the muscle vector system inevitably leads to corresponding soft points of weakness. In particular, stereotypical abdominal muscle training in the middle working range thus leads to relative weakness outside the middle movement range in interfaces and transitional zones in contact with the muscles, tendons, aponeuroses and the periosteum. Similar to the way that elastic-dynamic sealing rings securely seal, while hardened, inelastic materials lead to leakage, over-trained, stiff muscular systems inevitably lead to points of weakness.

Under the great stresses that occur in professional soccer, it is precisely at the points of particular weakness and in the nodal points that pain, strains, tears and ultimately recurrent symptom and injury complexes arise. It is possible to very clearly conclude at this point that it is frequently not the "structural point of weakness" itself that is responsible for the problems. Much more often, in its biokinematic "retrospective calculations," the clinical anatomical perspective leads back to the therapy-relevant key points derived from each individual athlete's biography of movement.