ORIGINALIA

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PRM HOMEOMESOTHERAPY AND MYOFASCIAL TRIGGERS: A VICTORIOUS COMBINATION

– DIAGNOSIS, CLINIC, THERAPY –

PART ONE

UPRIGHT POSTURE

The very complex and articulate process of "homination", far from having been completely deciphered and decoded, has run the course of a few million years.

The slow acquisition and conquest of an optimal and habitual upright posture has, by necessity, modified some anatomofunctional weight-bearing structures (vertebral column, pelvic bone complex, coxofemoral and tibio-tarsal articulation, position of occipital condyles, the foot in its entirety, etc.), and consequently, the complete **statural muscular system**, from distinctive involuntary innervations to muscular-tendinous biochemistry.
The liberation of the hand from the ground during deambulation created the premise for the development of a *long opposable thumb*, while cortical (motor and sensitive), axial and somatotopic representation of the hand involves a number of neurons equal to or greater than what innervates all of the voluntary muscular system of the body trunk (FIG. 1).
This "device" has made possible the construction of utensils, a consensual preamble to intellectual and cultural development.
The progressive acquisition of habitual upright posture has, by necessity, brought with it some negative aspects in the form of a series of problems such as vertigo (*Neri-Barrè-Lieu syndrome*), visceral ptosis and herniation, pathologies due to the burden of weight on the feet, lower limb varices and their complications, difficulties in the parturition process, and, last but not least, difficulties in learning to walk are necessary as they are inevitable, and tributes to being "*on one's feet*".

In man, the *foramen magnum*, the grafting zone of the cervical column and point of passage between the spinal cord and brain stem, is always much more anteriorized (advanced) compared to that of all of the other Primates: a nodal element for the comprehension of the physiopathology of upright posture and of its postural, physiological, and pathological adjustments and modifications (FIG. 2).

- The advancement of the *foramen magnum* by **2.5 cm** allows a good comprehension of the functions of the 2 lordoses alternate to the 2 vertebral kyphosis and the many vertebral pathologies in total (rectilinearization or inversion of physiological curvature, scoliosis) or partial (osteoarthritis, spondylolisthesis, bulging or herniated disc), and paravertebral (fibromyosis, tendinous-muscular triggers, etc.) that afflict mankind much more than other Primates and even more compared to other mammals in which the vertebral column is nothing other than a simpler vertebral "beam".

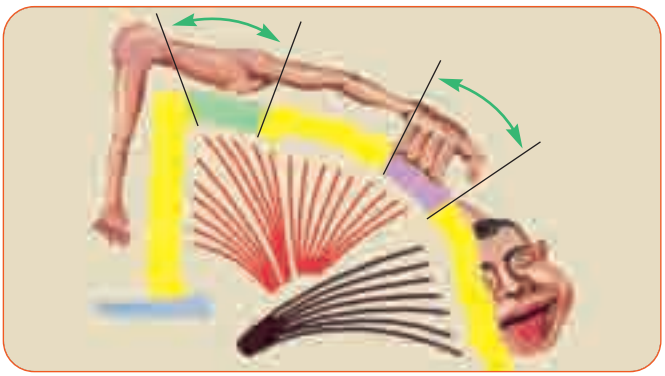


FIG. 1
Cortical motor representation of the somatic scheme: the number of neurons destined for innervation of the hand is equivalent to that for the innervation of the entire body trunk.

THE STATURAL MUSCULAR SYSTEM

Human musculature can be schematically subdivided into **3 large groups**:

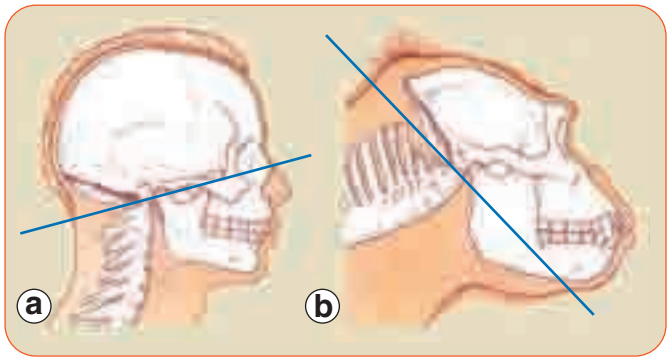
1) **Voluntary kinetic musculature**, innervated from the corticospinal or pyramidal bundle; pathological examples: spastic hemiparesis from cerebral stroke, flaccid paralysis from section of motor nerve: NEOCORTICAL ACTIVITY;

2) **Emotional musculature**, innervated by the efferences of the Limbic System or system of emotive-instinctive activity (Fisher-Coury circuit, Papez circuit); pathological examples: amimia in catatonic schizophrenia or from the intake of major psychiatric medications: PALEOCORTICAL ACTIVITY;

3) **Involuntary statural musculature**, innervated by spinal ganglia bundles and the cerebellospinal tract; pathological examples: rigidity and tremors as in those of Parkinson's disease, lack of motor coordination (from acute alcoholism): ARCHEOCORTICAL ACTIVITY.

The ***Triune Brain***, a neologism created in 1970 by Mac Lean: archeo-, paleo-, and neocortical, is, in effect, the processor of 3 motor identities that are distinct, but cooperative toward one goal: motor expression and modulation.
Normally, in an individual in upright posture with positioning of the head with a gaze straight ahead toward the horizon, a lead wire passing through the most posterior point of the cranium (*opisthocranion*) (1) must simultaneously touch the thoracic interscapular point (2) and the sacrum (scapulo-sacral vertical plane) (3) ((FIG. 3).

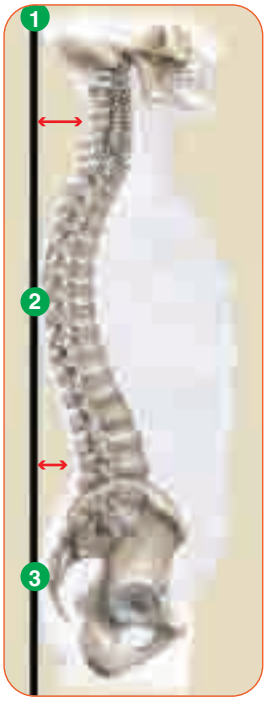
FIG. 2
Anteriorization of the occipital *foramen magnum* of man ⓐ compared to that of other Primates ⓑ.



Since the cervical lordosis is more accentuated than that of the lumbar (respectively, 6 and 4 cm on average in the point of maximum curvature), the paravertebral cervical muscles must accomplish powerful **antigravity action**.

In man, the anterior displacement of the *foramen magnum* conditions the positioning of the "Atlas vertebra – occipital condyles complex" to the center of the cranial base.
From the moment that the 2/3 anterior of the head + facial bones are much heavier in a static or dynamic phase than the 1/3 posterior (notwithstanding the presence of paranasal pneumatic sinuses), the cervical antigravity statural muscles must carry out **constant tonic action** and, furthermore, do so through levers not optimized through lordosis and contractions on inclined planes.
This indicates that functionally disadvantageous conditions are, in part, corrected in a sense of improvement by particular sub-adaptations (slight lateral displacement of the articular facets) even if these corrections may cause eventual pathological development (scoliosis).
Obviously, all of the statural (deep) muscles **are located posteriorly** with respect to the plane of gravity, guaranteeing proper antigravity functions.

FIG. 3
Cranic-scapulo-sacral vertical plane.



In antigravity cervical musculature, the complex of the *splenius muscle of the neck – splenius muscle of the head – semi-spinal muscle of the head* carries out the **true** statural function (FIG. 4), while the 2 *rectus* suboccipital *muscles* (*small* or *medial rectus of the head*, *large* or *lateral rectus of the head*) and the 2 *obliques* (*superior* and *inferior*) (FIG. 5) ensure that the Atlas vertebra and axis are in the nuchal line inferior to the occiput (cervicocranial junction).

FIG. 4
Deep cervical paravertebral muscular complex: *recti muscles* (pink), *oblique muscles* (purple); these 8 (4 x 2) muscles carry out the function of a zipper and cooperate toward the primary antigravity function. Function angles: ⓐ *small rectus* = 15º; ⓑ *large rectus* = 45º; ⓒ *inferior oblique* = 60º.

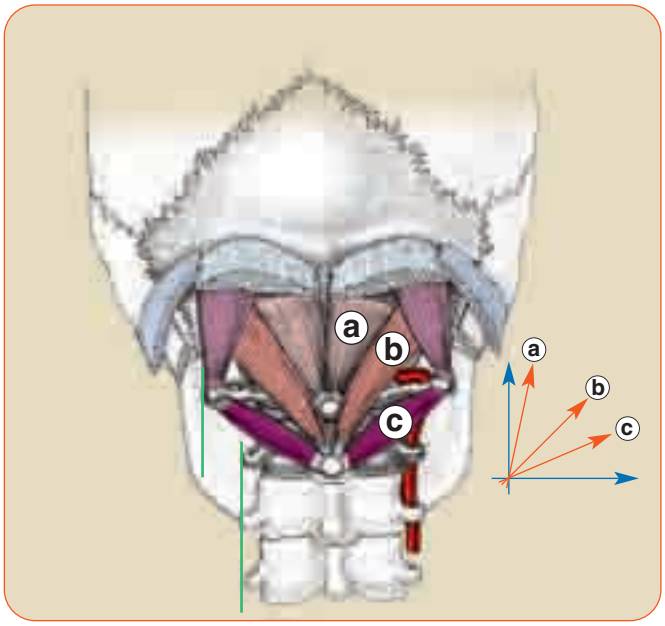
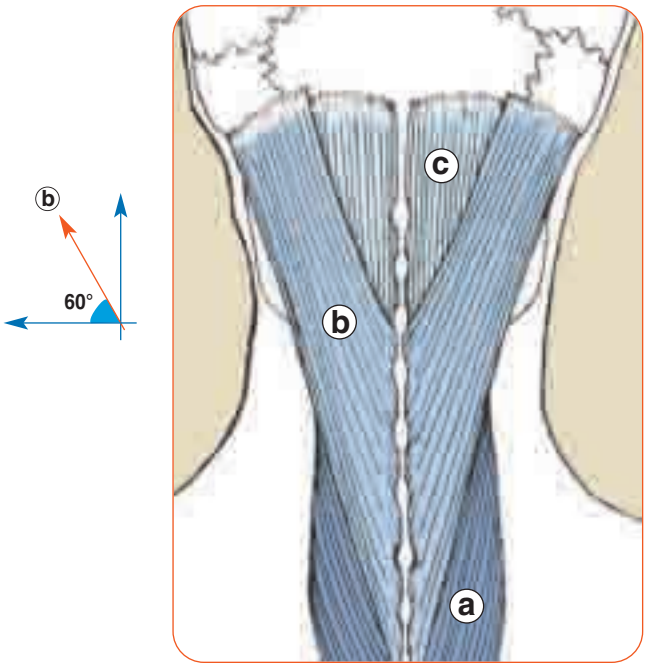


FIG. 5

Superficial cervical paravertebral muscular complex: ⓐ *splenius of the neck*, ⓑ *splenius of the head*, ⓒ *semispinal of the head*.

These 6 (3 x 2) muscles carry out the primary antigravity functions.



- Note how the lines of muscular traction occur on vertical planes and incline at approximately 15°, 45°, and 60°, thereby guaranteeing the maximum "lever" effect with the minimum expenditure of energy.

A kinetic device in cooperation with cervical stabilization is represented by the complex of *3 scalene muscles* that "tie" transverse apophyses from C3 to C7 to the first and second rib: **the scalene complex** is much developed in Primates compared to all other mammals.

Insofar as the paravertebral axial musculature, as for all of it as well as only that which is included in the paravertebral gutter formed from a series of spinose and transverse vertebral apophysis, in other words, in the area included between the Paravertebral Line and the Posterior Median Line or,

FIG. 6

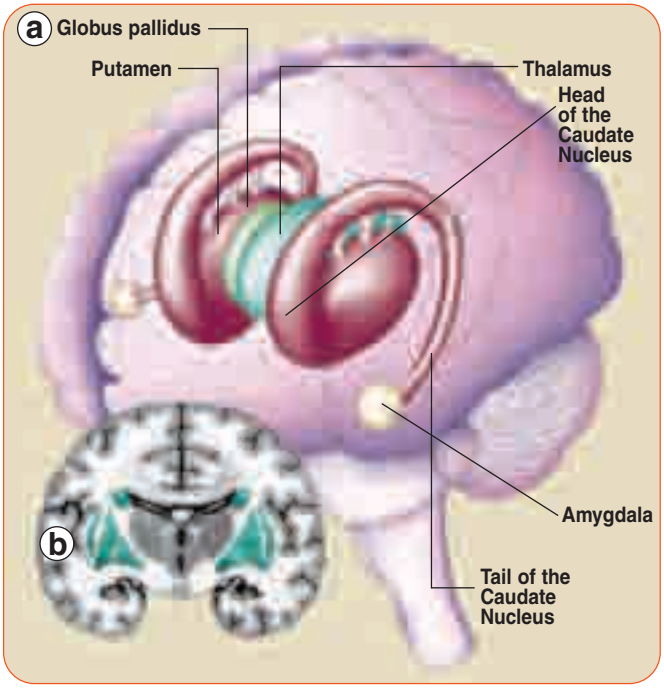


FIG. 7

ⓐ **Globus pallidus.**

ⓑ **Transverse encephalic section: basal ganglia** (green).

according to Chinese Traditional Medicine (CTM) terminology, Internal Branch of the Bladder (BL) Channel and the Pilot Vessel [Governor Vessel (GV) Channel], during the embryonic period, the posterior (epiaxial) metameric segments unite through progressive disappearance of the intermuscular septum (*myoseptum*).
In the adult, however, vestigial structures of a connective type still remain where, in the fetus, the *myosepta* (myoblastic-connective zones for the binding together of metameric myomers) were present (Milani, 1983).
Many kinetic muscles (e.g. *abdominal rectus*) also attest to their own polysegmented composition (FIG. 6).
- The 5 segments of the *abdominal rectus muscle* are innervated by 5 different nerves, serially adjoined.

This allows us to consider that the long paravertebral muscles are "*long*" **only in appearance**: actually, they are the myoconnective soldering of short metameric muscles, much like *single pearls united in the same necklace*.

This device guarantees **controlled** antigravity tonic functional performance, metamer by metamer, from **each corresponding posterior spinal nerve** (the long muscles derive from the embryonic fusion of the short muscles, but the innervation still remains metameric: every single paravertebral long muscles is polyinnervated: the entrapment of only one posterior spinal nerve is enough to compromise the functionality of a long muscle of even 40 cm, such as the very long dorsal muscles. This occurrence would produce many trigger points even if, for optimum therapeutic evolution, the nodal element is just one.
- The involuntary statural musculature owns a particular enzymatic arsenal that allows **sustained and prolonged activity**; it is innervated by the so-called reptilian motor

brain (MacLean's R Complex, 1970) consisting of the *corpus striatum*, the *claustrum*, the most rudimentary versions of the *amygdala*, the *colliculi of the lamina quadrigemina* (for the important correlations between the *corpora quadrigemina* and *epiphyses*: *see* Milani, 2002), the *diencephalo-hypothalamic structures*, the *red nucleus*, the Luys *subthalamic nucleus*, and the *substantia nigra*: all nuclei pertaining to the extrapyramidal system **not** of cortical origin (FIG. 7).
The basal ganglia, islets of grey substance encased in the hemispheres, carry out their own function such as relaying for the cortex and the *thalamus*: in mammals, and, above all, in Primates, the basal ganglia + the *cerebellum* are **automatic regulators of postural tone**.
- It is interesting to note how the nuclei of the R complex are selectively evident through a specific dopaminergic coloration (FIG. 8).

Certainly, when it comes to the genesis of trigger points, in addition to peripheral phenomena, an important role is played by the **subcortical reflexes** (important psycho-neuro action).

TRIGGER POINTS

Trigger points (TPs) are irritation points localized in the muscles, muscle fasciae, skin, ligaments, articular capsules, periosteum, and scars.

The statement according to which a TP always causes an **anatomical damage** (Howler, 1989) or *minimal muscular changes* should be revised, at least in the sense intended by the mentioned author. Howler stated that every muscular TP is generated by a microherniation of muscle-related tissue through a discontinuity of the *fascia propria* or, in the more superficial TPs, of the *fascia corporis superficialis*.
This statement has never been confirmed from an anatomopathological standpoint, even if, unfortunately, it has been passed from text to text without a critical revision or further elaboration. Histological studies (2001) have demonstrated, at the level of the TPs, the presence of releases of serum and mucopolysaccharide deposits, due to the inflammatory reaction induced by substances such as **histamine**, **prostaglandins**, and proinflammatory **cytokines**, (e.g. IL-1). All cells, but muscle cells in particular, are covered by a flexible structure referred to as "coating" (cell coat, fuzz, glycocalyx) with spikes having adhesive properties (Bretscher, 1971): under an electronic microscope it presents itself with the appearance of sparse, soft hair [it is clearly visible with red ruthenium, osmium, and ferritin, capable of attaching itself to the structure of the coating (Schmidley, 1990)] (FIG. 9). The portion of the coating most adherent to the cellular membrane is composed of branched glycoproteins (polypeptides to which oligosaccharides are attached) for antigenic or receptorial functions; the intermediate portion is composed primarily of proteoglycans (polypeptides

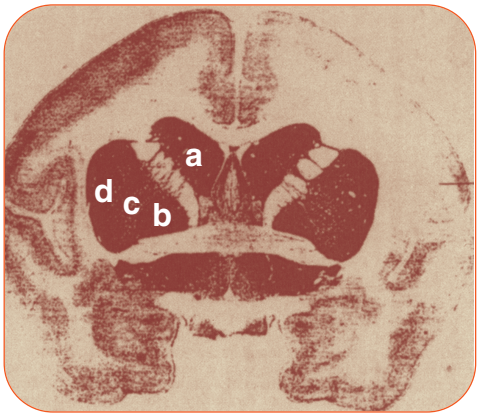
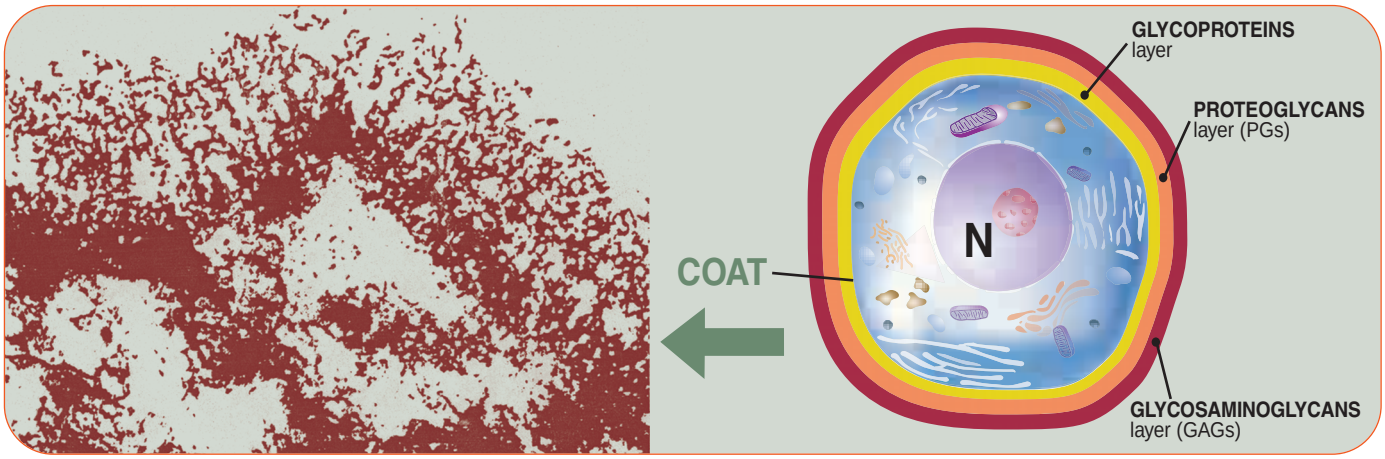


FIG. 8

Transverse section of a *squirrel monkey* brain: selective coloring from the reptilian brain; a) head of the *caudate nucleus;* b) internal *globus pallidus;* c) external *globus pallidus;* d) *putamen.* b) + c) + d) = *lenticular nucleus. Caudate nucleus + lenticular nucleus = corpus striatum.* The pyramidal bundle passes between the *caudate nucleus* and the *lenticular nucleus*, and it is here, in the pyramidal bundle, that the voluntary *(pyramidal)* corticospinal information and involuntary *(extrapyramidal)* *basal ganglia* information find their own points of contact and reciprocal interference.

FIG. 9

Cell coat. Right: placement of 3 pericellular concentric protective layers; Left: the glycocalyx at the E.M. The coat forms palisade structures or more or less enlarged mesh network structures.



to which glycosaminoglycans are attached), and the most external portion is represented by glycosaminoglycans (GAGs) composed of disaccharides. The coat, in its entirety, constitutes an important protective mechanism, defensive and receptorial of the entire cell against external and internal disruptions: it is the cell's true and own pericellular "*matrix*", in tight contact with the extracellular matrix.
In muscular "*adjustments*", through central hypertonus, the statural muscles can show (often at the metameric level also by input triggers with skeletal or visceral beginnings, in addition to the more important subcortical input) areas of acetylcholinic discharge (as a computer in a "loop"), and, therefore, areas of **rigidification**. The contracted muscular fibers exert pressure on the small venous vessels (stasis edema) and arterial structures through which a minor amount of blood, glycides, and O_2 reach the muscle cell; the **aerobic glycometabolism** can not be completely carried out, and only the first part of the process can be executed (anaerobic glycolysis), with production and deposit of *pyruvic acid* ↔ *lactic acid*.
- The absence of neosynthesis of glycoproteins and glycosaminoglycans strips the muscle cell of its own coating, exposing the membrane to mechanical micro-attacks, disarming the muscle cell of every defense; the deficiency of O_2 will produce acid radicals→ **pain**.
- The ischemic zone localized in the muscle area (trigger) induces, by contiguous polysegmentary autonomic reflexes, reactive dermal hyperemia which can be quite extended and evident clinically through dermographism and instrumentally through thermography (FIGS. 10, 11).

The sequentiality of these phenomena provokes a TP in an area confined by muscle tissue: the inevitable reaction will be new formation of collagen and of areas of scar repair. **These understandings are fundamental for a true and definitive TP resolution** (FIG. 12).

Another interesting aspect is that a radiculopathy can activate a muscular TP that mimics root pain and persists even after the root decompression: this trigger activity can be frequently found in the pain syndrome known as postlaminectomy syndrome (many are the cases of pain persistence even after the treatment of a herniated disc).

• A muscle that hosts a TP shows definite semeiologic characteristics: it is contracted, hard (with a cord-like and nodular aspect) with limited agonist movement, painful upon spontaneous and/or provoked contraction and upon stretching; if a TP is pressed with fingers, the patient warns of vivid pain: if the pressure is maintained, the pain can extend to other contiguous metamers, or, paradoxically, can disappear for a short time.

The pain is perceived as dull, deep, constant, and can cause extensive suffering, often with notable emotional impact in the form of depression.
The area does not always correspond to a local distribution, nor to the distribution of cutaneous and muscular peripheral nerves, nor as far as referred pain of visceral origin is concerned. In 90% of cases, the metamers in which the TP is located are involved.

FIG. 10

Thermography: sensitive increase in suboccipital cutaneous temperature due to the presence of bilateral cervical TPs.

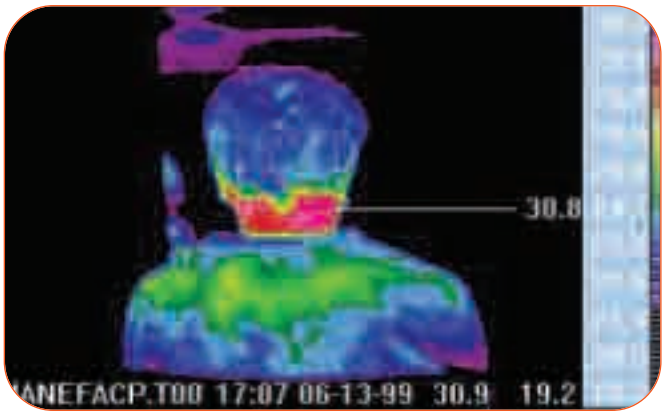


FIG. 11

Thermography: sensitive increase in dorsal cutaneous temperature *(left)* **and lumbar cutaneous temperature** *(right)* **due to the presence of TPs in underlying statural muscles.**

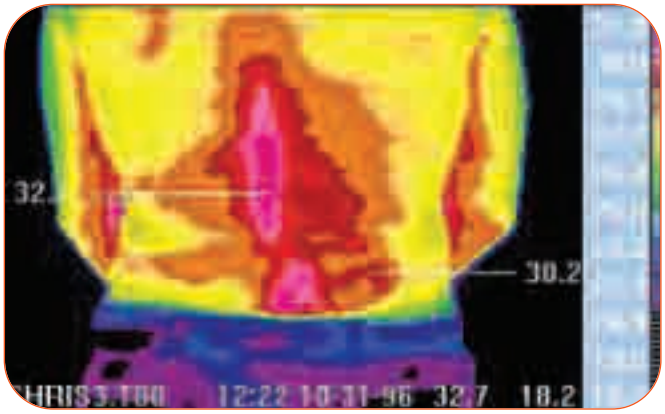
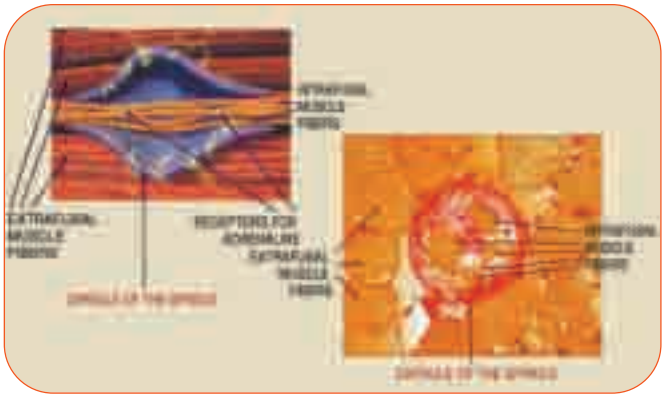


FIG. 12

Intrafusal muscle fibers: graphic *(left)* ***and microscopic*** *(right)* ***image.***



The TPs are activated **directly** by acute and chronic traumatic stimuli, by fatigue and cooling, and **indirectly** by visceral pathologies, emotional disturbances and articular suffering caused by arthritic osteophytes or by capsular-ligamentary stretching.

The pain degree of the TPs varies over the course of time, even within a few hours.
The "**active**" TP, after an initial acute phase, often becomes "**latent**", inhibiting the contractile activity of the muscle and its motor capacity. In such a case, a true recovery with the absence of pain is not vouched for, and acute relapses can occur after minimal exertions or minor trauma.

As indicated, the TP corresponds to a zone of **increased metabolic demand** or to **decreased energetic metabolism**. An ischemic alteration of the circle in the location of the TP is documented; this can be represented as an area enclosed by "*hot*" irritation in the center of a "*cold*" zone by minor blood flow. This area, which is actually "*hot*" (measured with a thermocouple), is the site of tissue ailments, with irritation of neural receptors and arousal of afferent fibers, and with them lies the responsibility of local and referred autonomous reflex phenomena.

PART TWO

MYOFASCIAL TRIGGER PAIN SYNDROME

Myofascial pain is one of the major and most frequent causes of compromised life quality. It also results in common diagnostic errors (and is, therefore, prognostic).
Frequently, it is incorrectly attributed to arthritic signs and symptoms, though arthritis can trigger the activation of active hyperalgic confined muscle areas, whether latent or silent (trigger). Fibromyalgia, Myofascial Pain Syndrome and Chronic Benign Pain Syndrome have important and damaging somatic, psychosocial and economic implications, as detailed in Popelianskii *et al.* (1978). The nosologic picture of these diseases has created, and still creates, much diagnostic confusion, both terminological and therapeutic.
Here I will briefly analyze the major findings of 3 clinical forms (and their diagnosis):

The term, "trigger" stands for: command lever, lever for unhooking, or lever to discharge a firearm.

1) Fibromyalgia

Fibromyalgia is a form of non-articular rheumatism characterized by muscle pain generally associated with numerous tender points. It is frequently associated with asthenia, morning rigidity and non-recuperative sleep. Moldafsky *et al.* (1975) described disturbances in sleep patterns in fibromyalgic patients that are predominantly characterized by the overlapping of the 7-11 Hz rhythm on the slower 0.5-2 Hz rhythm. Frequently patients have also anxiety, major depressive disorders, affective spectrum disorders, altered sympatic nervous system response; endocrine disorders (hypothyroidim, headache, dysmenorrhea), irritable bowel syndrome and paresthesia.
Hudson *et al.* (1985) and Kirmayer *et al.* (1988) noted an increase in cases of depression.
Tricyclic antidepressants block the presynaptic reuptake of serotonin and catecholamines and promote analgesic action independent of the antidepressant action. However, they generate several side effects and are not without contraindications; non-tricyclic antidepressants have the same antidepressant efficacy with fewer adverse effects, yet can not be considered equally as effective as tricyclics as to analgesic efficacy.

2) Chronic Benign Pain Syndrome (C.B.P.S.)

C.B.P.S. or persistent pain is characterized by widespread muscular – fascial pain with confined hyperalgic points (knotted or algic spots) as well as by anxiety, depression, and hypochondria. Patognomic symptoms may be characterized by: widespread migrating muscle pain, depression, and sleep disturbances.

3) Myofascial Pain Syndrome (M.P.S.)

M.P.S. is characterized by localized muscle pain associated with stiffening and general weakness. The muscle pain is located in well-defined TPs.
M.P.S. may affect a single muscle or may be multiregional or generalized. This disease is associated with depression, fear of undertaking motor activity, and anxiety, as well as sleep disturbances as previously indicated. In generalized forms, it can be associated to hypothyroidism (also hypothyroid "traits"), vitamin deficiencies (mainly of the B Group in the elderly), endocrine dysfunctions, postural anomalies, and multiarticular pain.

FIG. 13
Dr. Janet Travell (1902-1997) in the 1950's.

Other diagnoses, such as "*myofascial pain dysfunction syndrome*" (Butler, 1975), seem to overlap with that which is described.

▶ The three syndromes described (already under the generic term "*fibrositis*" (Abel, 1939), an obsolete term, yet still used in some recent publications) are nothing more than clinical or chronological expressions of the **same disease** that, for uniformity, may be correctly defined as **"Myofascial Trigger Pain Syndrome"**, a term that is being put forward here for the first time and will be referred to in the text.

- The strictly somatic elements (active TPs, latent or silent) intensify the psychological symptoms (the cortex and the reptilian

basal ganglia synapse directly with the Limbic System) and psychosomatic symptoms (*diencephalon*– epiphisary circuits) that reverberate on the soma, reactivating and amplifying the vicious circle.
Many myofascial TPs overlap with acupuncture points (Melzack, 1981) and Weihe points (Milani, 2004).
The reason for this overlap was researched in a common anatomical location: the muscular-tendinous junctions, areas of increased receptor density with structural polymorphism (Bistevins, 1981). Furthermore, both the TPs and the acupuncture points are characterized by an abrupt drop in electrical resistance; a term reported in ancient Chinese texts to designate the "point" of acupuncture is "*hi-shueh*", which literally means "*well's bottom*".

DOES MYOFASCIAL REFERRED PAIN EXIST?

The phenomenon of **cutaneous visceral referred pain** ("referred pain" is an expression that was popular at the beginning of the last century, often poorly interpreted) described by H. Head and continued by Pancot (1929), Wetterwald (1940), Hausen and Schliack (1962), Bolke (1965), and Jarricot (1932, 1971, 1980), and actually well-recognized, even if generally undervalued in practice.
As to **muscular** pain, paradoxically, things are different.
The first quality and exhaustive text on the subject was published in 1983 by Travell and Simons. In 1983, Travell (FIG. 13) had already gained a valuable experience on the study and therapy of myofascial pain (Travell, 1949 *et Seq*)

The text by the 2 authors is well-written and detailed, meticulously illustrated, with a rich bibliography with a vast proportion of references, and in keeping with better American scientific traditions of the time, it is dogmatic, leaves no open doubts, and is entertaining in its illustrations.
In a word, it is a "ready to use" text despite being very articulate and complex.

One of the "key" concepts in the theory set forward by Travell and Simons and, later, by Travell and Daltz, is that **every** single muscular TP produces, at rest or in movement, **referred pain** in a **specific extrametameric** area for **each muscle**. According to the authors mentioned, the patient refers pain in areas not corresponding to those that host the TP/TPs that has/have provoked the pain; each muscle may be the location of more than one TP and, consequently cause more than one kind of pain after a while, with consequentially outstanding complications for topographic diagnoses since much of the referred types of pain juxtapose or overlap.

▶ Personally, I believe that TP form where the myoblastic–connective zones of the metameric myomeres welding-type (*myoseptum*) were present in the embryo.

According to the authors cited, the projection or "referring" of pain presents topographic characteristics that are constant and distinct for each muscle: they develop the concepts for their entire "manual" according to this dogma, in some ways they readdress the concept relative to visceral-cutaneous pain, however...

HOWEVER

…While anteriorly and posteriorly referred visceral pain **always has metameric characteristics**, in continuation with a rational neurophysiological and embryological interpretation (there is high clinical and therapeutical support for this statement), the referred muscular pain of Travell and Simons is **very frequently extrametameric**.

▶ More than thirty years of personal clinical experience in benign Pain Therapy and, in particular, in that of Myofascial Trigger Pain Syndrome brings forth a few considerations:

1) Myofascial TPs form in **every** portion of the muscular tissue and **not** necessarily in predefined areas.
- Consequently, they can not be indicated "*a priori*" and, therefore, can not be specified in anatomic tables as they are variable and must be considered on a case by case basis.

2) In 90% of cases, TPs **do not refer** pain at a distance, nor on the same metameric level in the horizontal sense, nor on distant metamers in the craniocaudal sense:
a) the patient *perceives* and *indicates* the pain **where** the algogenic TP is present, or, at least, in the *nearby* zone (a few cm) (70%);
b) the patient indicates pain referred at the most at 1-2 contiguous metamers above or below with respect to that of the location of TP (30%), in line with the theory of the Sherrington's Law of the 3 Roots.

- It is not understood why muscle pain must be **extrametamerically** referred if every muscle is constituted by many anatomically distinct **metameric units**.

3) The "*stretch-and-spray*" technique, proposed by Travell and Simon while occasionally effective for the short term, is basically ineffective for the long term (if the TP is not treated effectively, it will present itself again).
- It is impossible to manually stretch the deep paravertebral muscles. Their "*control*" is automatic (tonus), and their voluntary "*relaxation*" is impossible (they are involuntary), especially if they host TPs. The formation of a TP and its maintenance are mediated by spinal and reticular reflexes. In addition, the stretching of a muscle that hosts a TP is very painful.

4) Thermographic studies [in Milani, 2003] have thoroughly evidenced how each trigger is not a *point*, but, rather, an ischemic "*cold*" *area* surrounded by a "*hot*" cutaneous zone (see FIGS. 10, 11)involving wide somatic portions.
The onset of muscular pain is tied to the stimulation of relatively vast areas, leading to widespread hyperalgia (Cambier, 1979; Arcangeli, 1980).

Reflex vasoconstriction may cause ischemic suffering of the nerve, and in this case, the pain will be **irradiated** (not "*referred*") along the "downstream" neural course of the TP: this phenomenon, in fact, is frequently observed, but it deals with secondary neuropathy (e.g. Arnold's grand occipital nerve syndrome causes TPs of the homolateral *rectus muscles of the head*, and abdominal–genital nerve syndrome causes triggers of the homolateral *psoas muscle*).

In these cases, the irradiated nerve pain is **delimited, acute, burning** and not **widespread, silent, and constricting as with myofascial pain**. The entrapment of a single nerve may compromise the functionality at a distance of some muscles, but it does not induce referred muscular pain, and, instead, induces irradiated nerve pain: this would deal with 2 distinct diagnostic and therapeutic events.

- From all of these and other considerations and from daily experience, it is clearly demonstrated that myofascial TPs therapy is **simpler**, **more practical** and **direct** than the one proposed by the abowe mentioned authors.

In practice, the patient *indicates* the hyperalgic area and the operator, after having detected the presence of a TP, inactivates it.

All of this is neither simplistic nor banal: experience has evidenced that "*simply*", this is the way it is, and this is an observation reinforced by therapeutic pragmatism in addition to neurophysiology.
The theories may be argued, but the facts may not.

LOCALIZATION OF MYOFASCIAL TPs

The localization of TPs is a simple and quick procedure:

1) Have the patient calmly and precisely **indicate** the painful zones beginning from the cervical musculature, followed by the thoracic and lumbar musculature: first the involuntary *(statural)* paravertebral musculature and then those muscles which are voluntary *(kinetic)*.
The inactivation of the paravertebral TPs resolves at least 70-80% of **all somatic pain pathology complaints**.
2) Note with a dermic pencil (make up pencil) **all** the pain areas indicated by the patient.

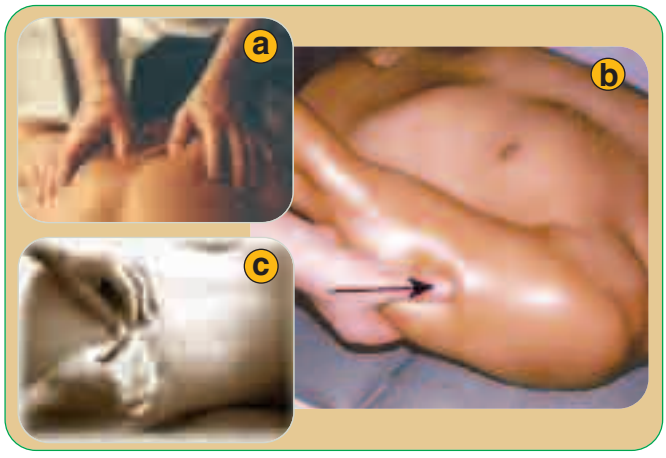


FIG. 14

a) Placement of the *"exploratory"* **finger (in this case, the thumb) for the comparative exam of the musculature of the 2 antimeres. b) medium-deep digital pressure on the painful muscular portion indicated by the patient: if it is a** *"true"* **TP, the operator will feel the presence of a hard or tense elastic band underneath the exploratory finger. c) position of the operator's fingers in the** *"inverted fold"* **maneuver in order to evidence the visceral-cutaneous dermalgia reflex** *(palper roulé technique)*.

3) Wait a few minutes and repeat **1)** beginning from the lumbar musculature, followed by the thoracic and cervical musculature, and then check if all the areas have been re-indicated. In this phase it is possible that not all areas will be confirmed or that other areas may be found.

4) Note any additional areas with a dermic pencil.

5) Press with the fingertip of the 1st finger or with a small plastic or hard rubber tube (not metallic) with a diameter of 2 cm each area signaled (and therefore indicated) making the fingertip or plastic tube run across the skin and on the cleavage plains underneath. The palpation should not be too deep and must be precise and safe (FIG. 14b).

6) Note with a dermic pencil the most painful TPs (for the first sessions, only these will be treated; others will be considered in following sessions).

7) The pain zone indicated by the patient corresponds to a "true" TP if:

a) **by inspection**: the muscle "*in toto*" that hosts the TP/TPs is more contracted than the contralateral same muscle counterpart and appears shorter and "swollen";
b) **by palpation** it occurs to be:
1) a small band with a tense - elastic consistency that is confined and of variable volume (generally not more than ø 2 cm) that "jumps" when pressed by the fingertip or plastic tube, or
2) a slight dipping of the muscular tissue (in any case, a "functional" trigger determines muscular hypo-functionality "*in toto*" with fibral subtrophy and substitution of myotissue with loose connective fibrils or subfascial adipose).
In this case, we would be dealing with a fascial TP.

MYOFASCIAL TPs THERAPEUTIC OPTIONS

For a small number of diseases, such as for Myofascial Trigger Pain Syndrome, quite numerous and diverse therapies have been proposed. Here, we will specify only those commonly used:
1) systemic allopathic pharmacological therapy: corticosteroids, NSAIDs, ASA, myorelaxants, antidepressants, anxiolitics, B-complex vitamins, etc.;
2) local allopathic pharmacological therapy (local anesthetics, myorelaxants, NSAIDs, corticosteroids) by **local** injection (conventional mesotherapy);
3) external allopathic pharmacological therapy: patches containing NSAIDs, pomades or anti-inflammatory gel, vasoactive therapy, revulsive therapy;
4) instrumental physical therapy: soft laser therapy, electrotherapy, magnetotherapy, ultrasonotherapy, ionophoresis, etc.;
5) manual physical therapy: manipulations, massophysiokinesitherapy, muscular kneading, deep connective-tissue massage, stretch-and-spray, and shiatsu;
6) non-conventional therapy: acupuncture according to CTM, neural acupuncture (neuroreflexotherapy), local homeopathic/homotoxycological biopuncture, homeopathy, Bach flower therapy, phytotherapy, etc.;

7) innovative non conventional therapy: Physiological Regulating Medicine (PRM) homeomesotherapy (Milani, 2006).

PRM HOMEOMESOTHERAPY

This therapeutic technique consists of intramuscular, intradermic, and subcutaneous injections of PRM homeopathic medicines directly in the TPs and/or in locoregional and distal points. It is a combined technique of very delicate biological mesotherapy where, in addition to therapeutical possibilities of PRM remedies, there is also the associated consequential action to the stimulation of the reflexogenic zones that, as noted, are very rich in receptors with synergetic and amplified therapeutic effects. The homeomesotherapy points are metameric points: the mechanism of action can be understood in neurophysiologic and neuroreflexologic terms by keeping in mind, at least in their summarized form, Sherrington's Laws of the 3 Roots and by referring back to the Dermato-neuro-viscero-myomeric Theory (Milani, 1978, 1980, 2000, 2004). Among the effects of homeomesotherapy, the most studied is the analgesic one.
It is necessary to research a unique pluripotent mechanism that would explain the various actions of PRM homeomesotherapy (TABLE 1).

PRM homeomesotherapy guarentees a much **safer** and **less troubled** therapeutic treatment and, in its therapeutic effects, it allows **greater operational continuity** and **repeatability**.
In addition, for at least the last 10-15 years, patients seem to be accepting and choosing a more natural, organic and non-toxic approach for the resolution of their health problems, contributing to the establishment of effective compliance, in addition to a further and more thorough diffusion of the method.

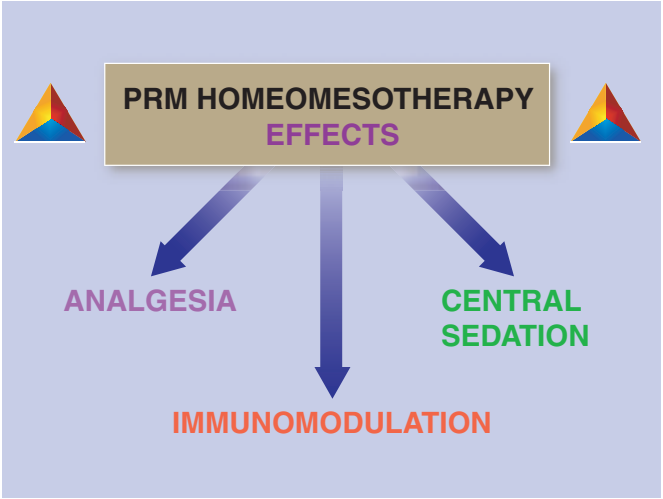
OUTPATIENT PRM HOMEOMESOTHERAPY

- Therapy for Myofascial Trigger Pain Syndrome (acute, subacute, and chronic) through the local infiltration of injectable PRM ampoules is described in detail (TABLE 2 - p. 28).

The therapy is differentiated into **acute** and **subacute-chronic** divisions, as different clinical situations are the translation of different histopathological circumstances: consequently, the different injectable ampoules that are most suitable must be used: short-term and long-term results testify to the consistency and validity of the therapeutic strategy setup.
- In addition, the specific injectable ampoules for local therapy of the cervical, thoracic and lumbar segments of the spinal column are indicated.

PRM homeomesotherapy in TPs pain control is not, nor is it desired to be, solely a therapy for pain.
Some indicated injectable, in addition to developing **analgesic**, **visceral** (vascular), **somatic anti-spastic**, and **anti-inflammatory** effects, have an **anti-neuralgic** effect, **sedative** effect, **anti-rheumatic** effect, **microcirculatory** effect, **histo-reparative** effect, and **biostimulating** effect.

TAB. 1



- PRM homeomesotherapy promotes not only symptomatic effects but also **deep tissue** and **endocellular effects**, with strong imprinting on the **cell's energy** (holistic effect).

CONCLUSIONS

It is my hope that this publication establishes the elements for a clear and optimal comprehension of the complexity of structural, pathological, and therapeutical problems related to myofascial TPs.
A certain classical and historical "affirmation" regarding referred pain from myofascial TPs was put into question and so were formed the objective elements to support this antithesis.
Furthermore, I tried to put nosological terminology in order.
For completeness and accuracy, many therapeutic options, both conventional and, specially unconventional, were indicated.
Personal and many others experience indicates that the **PRM homeomesotherapy-myofascial trigger combination** is the victorious one: it is on par in terms of therapeutic effectiveness and it is safer, guaranteeing the *primum non nocere* saying of old times, and, consequently, it is a therapeutic practice that is no risky or toxic.

At least 2-3 sessions must be performed to be able to appreciate the initial results so that an active TP would not present itself again after having passed into a latent state. ■

TAB. 2

Myofascial Trigger Pain Syndrome Therapy
ACUTE PHASE (1 to 14 days from clinical onset)

OUTPATIENT THERAPY

GUNA®-MUSCLE 2-3 ampoules

In particular if there are:

- Cervical TPs – add **GUNA®-NECK**, 1 ampoule
- Thoracic TPs – add **GUNA®-THORACIC**, 1 ampoule
- Lumbar TPs – **add GUNA®-LUMBAR**, 1 ampoule

If more than 3 different anatomical parts:
add **GUNA®-POLYARTHRITIS**, 2 ampoules
(each in *cocktail* with **GUNA®-MUSCLE**)

The outpatient therapy involves, on average, 6-8 sessions:
1st week: 2 sessions at least 3 days apart (e.g. Tuesday-Friday)
2nd week: the same
From the 3rd week: 1 session per week.

Subcutaneous injection with a 4mm, 27G needle
Each TP must be injected with 1,5ml of cocktail
If in acupuncture points*: 0.5 ml of cocktail in each point.

AT-HOME THERAPY

GUNA®-LYMPHO (10 drops 3 times a day); 9 a.m.; 3 p.m.; 9 p.m. +
GUNA®-MATRIX (20 drops 2 times a day); 9 a.m.; 3 p.m.
TAMANU ARNICA™: 3-4 applications a day according to individual needs

Myofascial Trigger Pain SyndromeTherapy
SUBACUTE PHASE (3-6 weeks from clinical onset)
CHRONIC PHASE (more than 6 weeks from clinical onset)

OUTPATIENT THERAPY

GUNA®-MUSCLE (1-2 ampoules) + **GUNA®-NEURAL** (1-2 ampoules)
In particular, if there are:
- Cervical TPs – add **GUNA®-NECK**, 1 ampoule
- ThoracicTPs – add **GUNA®-THORACIC**, 1 ampoule
- Lumbar TPs – add **GUNA®-LUMBAR**, 1 ampoule
- Other anatomical parts – add, according to the affected areas:

GUNA®-SHOULDER (1 ampoule); **GUNA®-HIP** (1 ampoule); etc.
if more than 3 different anatomical parts:
add **GUNA®-POLYARTHRITIS** (2 ampoules)
(each in *cocktail* with **GUNA®-MUSCLE + GUNA®-NEURAL**)

The outpatient therapy involves, on average, 8-10 sessions:
first 4 weeks: 2 sessions per week at least 3 days apart (e.g. Tuesday-Friday)
From the 5th week: 1 session per week

- Subcutaneous injection with a 4mm 27G needle
- Each TP must be injected with 1,5ml of cocktail
- If in acupuncture points*: 0.5ml of cocktail in each point.

AT-HOME THERAPY

GUNA®-CELL (10 drops 3 times a day); 9 a.m.; 3 p.m.; 9 p.m. +
GUNA®-MATRIX (20 drops 2 times a day); 9 a.m.; 9 p.m.
TAMANU ARNICA™ 3-4 applications a day according to individual needs

** Recent anatomic research has shown that therapeutic effects of acupuncture may well be explained scientifically:*

According to Heine (2008), Acupuncture Points (APs) are characterized by a nerve-vessel bundle wrapped in a sheath of loose connective tissue (mesenchyme). 82% of all classical APs are circumscript perforations of the superficial body fascia piereced by a nerve-vessel bundle. In body areas not covered by this fascia (i.e. face, skull, fingers and toes) the principle of the mesenchym-covered nerve-vessel bundle can also be demonstrated for APs.
The spinal nerve of a nerve-vessel bundle contains among others substance P reactive axons which are able to antagonize the sympathetic nerve plexus in the adventitia of the wall of the arterie(s) and vein(s) of an AP bundle. Each change within in an AP can generate local axon reflexes which could spread along a Channel. Morphologically Channels (Meridians) are fascia - myo - tendinous kinetic chains.
All events affecting such a chain are registered by corresponding APs causing a regulatory answer.

In case of pathophysiological events the APs involved react painfully (microtrigger points).
Injections of PRM injectable ampoules into these points can have a remarkable effect restoring segmental-regulatory reflexes.

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Pictures without number:
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Fig. 1 VALZELLI L. - Profili di psicofisiologia e neurochimica. Manfredi Editore, Milano, 1971. (modified)
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Fig. 6 VON HAGEN G. - Körperwelten: Fascination beneath the surface. Institute of Plastination. Rathausstrasse, 18 - D-69126 Heidelberg. 2002. (modified)
Fig. 7 a) http://www.nature.com/nrn/journal/v2/n5/slideshow/nrn0501_352a_F1.html
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Fig. 8 MAC LEAN P.D., GUYOT R. - Le trois cerveaux de l'homme. Roland Laffont Ed., Paris, 1990. (modified)
Figs. 10 e11 http://www.thermology.com/infrared-back.htm
Fig. 12 www.myopoint.com/network/physicians/myalgia.html
Fig. 13 http: //www.gwu.edu/gelman/archives/collections/image002.jpg (Dr. Travell photo)
Figs.14a-14b-14c
http: //www.massagesolutions.com/Table5.gif
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