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**Translations of:**

1. **“Nature, Clinic and Significance of Spondylogenic Reflex Syndrome”**
  - a. **M. Sutter**
2. **“An Attempt to Define Radicular and Pseudoradicular Syndromes“**
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## **Nature, Clinic and Significance of Spondylogenic Reflex Syndrome**

**M. Sutter**

### **Reasons for definition**

The clinical work with diagnostic-therapeutical problems of the manual medicine on the one hand and at the same time, with certain questions of the primary non inflammatory soft tissue rheumatism on the other hand, has lead the editor on a empiric way to find reproducible clinical relationships between certain vertebral joints and peripheral soft tissue. These relationships must be arranged neural due to their clinical characteristic. Neuroanatomically, the connection is only possible over the spinal cord; according to definition the relationships have to be of reflectory nature. Because the happening of the axial skeleton is attested regularly as primary cause, while the changes of the peripheral soft tissues are presented as pathogenetical dependent consequences, we call these new clinical relations/connections spondylogenic reflex syndrome (SRS).

### **Up to date (current knowledge)**

Till this day only a part of these relationships are known and their attribution to specific vertebral joints as far as confirmed, that they meet diagnostic and therapeutical requirements. Also these assured facts need review, addition and specification, with increasing experience. Progresses are expected faster and more reliable, the more these relationships in diagnostic and therapy are considered. The known and assured relationships represent a fit diagnostic, which allows the exact causal attribution of numerous phenomena, which otherwise would have to be considered isolated and not to be interpreted closer. These relationships are causal, they open the path to causal therapy.

Widely known and affirmed are the aspects of the spondylogenic reflex syndrome in the area of the lower extremities. The relations of torso, head and upper extremities are more complicated due to numerous interferences, that's why their clarification is more difficult and less advanced.

### **Diagnostic findings of the axial organ**

Efferent(?) source of the SRS are the vertebral joints. Probably the complementary parts of the dorsal ligament apparatus/systema of the joint functions with the monosegmental-vertebral musculature belong to that. The spondylogenic-reflectory caused state change of the tissue of this narrow circumscribed area, is displayed through direct, temporal as

quantitative binding on the irritation of the related equilateral intervertebral joint area. The state changes of these circumscribed tissue are objective palpable and painful under palpation pressure. The special temporal and quantitative binding makes it a reliable local indicator of functional diagnostic on the axial organ. We call it after *Caviezel* (spondylogenic) irritation zone. *Sell* introduced therefore the definition segment point. From today's point of view the appellation is misleading due to the confusion with the neurological segment definition. The irritation zone is an integral component and the central phenomenon of a florid spondylogen reflex syndrome. The sacroiliac joints have reflectoric-functional the meaning of the phylogenic intervertebral joints S1-S3.

By far the most frequent causation of a verifiable spondylogenic irritation zone and therefore of a spondylogen reflex syndrome is the *functional segment disorder of the vertebra*. This definition needs detailed explanation for an exact understanding.

The anatomical or radiographic vertebra position describes in contrast to the functional the spacial classification of an element in the system of the spine. Basically nothing changes, although the spine is examined in movement and different positions. Though we are used to name an examination, which considers different aspects of positions, as functional. But we have to realize, that the entirety of the functional is not captured in no way. All the radiographic presented aspects can only refer to a part of that, namely what we call the *external function*.

Under the external function of an organ or an element of that, we understand the activity for the benefit of the whole issue. The external heart function for example is the amount of delivered blood, the one of the spine is the spacial posture and the movement of torso and head.

The external function must be distinguished from the *interior function*. With that we mean the harmony of the organ or structural mechanism, the own interior relation structure, the adjustment of interaction of the detail functions. The external and interior function are distinguished not only in their nature, but also in their characteristics: The external function is quantity, is activity, is measurable. It can be increased, constricted and lost in favor or in account of the whole issue. In contrast the interior function is a method of operation, a quality. It is not measurable, but understandable after the principle and the components, and can be disturbed, changed and is adaptable. The tolerance limits are defined through the whole concept.

The damage of the external function hits the beneficiary, the disturbance of the interior function the bearer. External function is the product of the interior function, the external has the interior one as a condition. The interior function is a coordination of the external function of subordinate units on a higher unit. The longterm and adequate external function bases in general on an intact interior one. Structural integrity usually assumes undisturbed interior function. The disturbance of the interior function doesn't have to mean the damage of the external function. The interior one expires with prohibition of the external one.

The definition functional shows that the disturbance of health hasn't a morphological change as cause, but an intolerable change of the relation order of the intact components. Model for further discussion is represented through a machine, which though their intact and right placed units doesn't work well, because of the wrong spring tension. With the exceeding of tolerance the disturbance of the interior function is given. Their manifestation are quantitative, unusual, the normal crossing, subordinate, extreme functional situations and functional sequences. As consequence the changes can stimulate local pain receptors and provoke reversible till irreversible structure and form changes. The straight relation disturbance passes in a mixed functional-morphological disturbance.

Form change of organic (?) components has tolerance limits too. Primary morphological change remains insignificant, till it is either contradicting with normal internal function, or prevents external function in insupportable dimension. Degenerative changes can only be ascribed pathological significance, if they meet the criteria. Their representation as illness factors *eo ipso* is not qualified. Both above mentioned cases lead to illness, though depending on the affected function on different levels of organisation. Is the axial organ subject to morphological changes with functional implications, it results – as long as only the external function is damaged – in merely painless constriction of movements of the organism. If instead the internal function is affected, disturbance of the harmonic components co-operation follows. In the case of the axial organ the disharmony is shown in unsound coordinated muscle tension, mechanical overstraining, uncoordinated movement of the vertebra, functional segmental disorder of the vertebra, also on the organ itself. The disturbance of the interior function can cause the loss of the external function. But it's not a necessary consequence. Despite the disturbance of the interior organisation, the external function can be provided, though often through overstraining of subordinated function units (disci, ligaments, cartilage, joints, musculature) and on the cost of structural integrity.

Pathological extension of the external function with adequate disturbance of the internal one is not seldom in the movement apparatus/system. Though mutual conditionality of internal and external function, the disturbance respectively the constriction of the one or the other doesn't assign necessarily. Besides causal proportion, the possibilities of isolated occurrence of one or the other are given, as also the one of temporal and local parallelism (without relation). The argument of the intact interior function through evidence of the obtained external one or otherwise is unacceptable. This rule demands absolute attention, if false conclusions, especially in judgement of vertebra disorders, should be avoided.

The anatomical or radiographic spine position describes a state of the external function. The functional spine position on the other hand is an element of the interior function. In this position it has an essential reference to the functional state of the axial organ as a whole issue. This reference is missing in the radiographic-anatomical spine position, because some deciding factors of the interior function are not shown in the x-ray like the tension-length-correlation of the musculature for example. Though certain radiographic spine combination show with high probability functional position disorders, there's no necessity for consent, just as little radiographically unremarkable spine disposition represents functional right position from the beginning. Both definitions need clear disjunction. In consequence the examination of the external function alone or added through the x-ray finding is not a base for the analysis or judgement of the interior function. Due to it needs necessarily addition through clinical examination of the interior function. The elements of the interior functional disorder need interpretation with pathogenetical knowledge and empiricism.

Under normal circumstances the connection of the interior function remains hidden. They display through their pathology. Only the analysis of a plurality of different disorder pictures let us capture the right combination, understand the character and relation order and their principles and based on that, understand the disturbances and diagnosis. Purpose of the excursion is to prove, that functional segmental disorder of the vertebra is understood wholly only clinically, that means, through palpation of the irritated zone. Functional segmental disorder of the vertebra has multiple possible causes. Wrong tension of muscles, traumas, uncoordinated movement ("faux mouvement"), acute and chronic mechanical overstraining of the vertebra, insufficiency of the osteo-chondro-ligament movement coordinator apparatus/system are supposed to be the most important ones.

Besides functional position disorder – though less common – also other abnormal states are considered as cause for pathological changed afferences from intervertebral joints, for example inflammation, arthritic stimulus, neoplastic infiltration. General examination, laboratory and x-rays are best suitable to show such causation.

Indication and contraindication of therapeutic measures are deviated from the causation. With the last mentioned causation manual therapy is contraindicated, as long as no combination with functional segmental disorder of the vertebra exists. Under circumstances it can be decided only *ex iuvantibus*. Uncomplicated functional segmental disorder of the vertebra is the main indication of manipulation. Physical application and certain pharmaceuticals are valuable adjuvants. In little fixed cases they're adequate to initiate the remission. *Determined manipulation or local antiphlogistic-analgetic infiltration – under certain circumstances in combination – leads normally to the goal, unless an important and inalienable condition has been fulfilled before: The effect of the pathogenetic causation chain, which led to the functional segmental disorder of the spine, has to be reduced effective or wholly eliminated.* Otherwise the relapse menaces, already before the regeneration of the secondary peripheral change can be subjective noticeable. The missing therapy success is wrongly taken as an argument against the spondylogenic reflexory pathogenesis. Chronic or recidivating spondylogenic reflex syndromes can only be eliminated definitely through exact clarification of their etiology and the derivated step-by-step therapy.

### **Findings in the periphery**

How far peripheral structures and organs are implicated in spondylogenic reflex syndromes, is not clarified by today's point of experience. Multiple clinical observations suggest, that the significance of the spondylogenic reflex syndrom is not limited to the movement apparatus/system and the integument, but furthermore extends on *inner organs and the central nervous system*. If this happens over the direct nervous way, over changes of the local circulation or through interconnection of other control mechanism, has to remain open for the moment. The effect on the inner organs appears less constantly and is clinically less exact comprehensible than the change in the ligament-muscle-apparatus. Therefore their diagnostic significance stands back. The fact, that the tissue reacts on reflexory influence not stimulus specific, but tissue specific, complicates extensively the correct evidence of spondylogenic-reflexory conditionality for disorders of the inner organs. Forehand only argumentations *ex iuvantibus* or *per exclusionem* are reliable enough. Even impressive results of treatment failed the evidence of adequate general validation, because of insufficient comparability and reproducibility.

The relations in the *integument* (*kultis* and *subkultis*) are clearer. Everywhere it is accessible directly for the palpation. Accurate localisation is hindered, because of unsharp local differentiation and missing of clear anatomical limitation.

The spondylogenic conditioned change of the integument corresponds to a non-inflammatory soft tissue rheumatism. The clinical picture is defined through consistency increase of the subcutis with weight gain by missing of a free oedema(?). The free instability between cutis and subcutis becomes lost, pain appears spontaneously or with mechanical exposure. Typically is the massive provocation through comparatively light shear of the tissue. These changes don't allow a differentiation from the florid painful panniculosis, because neither character nor quantity of the change make the spondylogenic causation nameable. It can only be accessible through the special composition of the change in certain patterns.

For the local determined spondylogenic panniculosis we prefer the definition *Zellulalgie*. It formulates direct substratum and clinical appearance, namely skin-subcutis-fat tissue and the pain, and avoids the abused and misleading ending *itis*. *Zellulalgie* in the sense of

spondylogenic reflex syndrom means also systematized local composition of floride pannikulosis. Residual changes after healed spondylogenic reflex syndromes are pain free. They behave like unspecific pannikulosis. With the diagnostic significance the need for differentiation of concept drops.

The Zellulalgie as spondylogenic conditioned change pattern of the integument is located in zone till lamellar composition. The superficialness of the analysis can involve confusion with the segmental dermatomic area. Also light layered and tight integument, as for example on the forehead, over the pretibial and the heel, underlies zellulalgic change. The hindered evidence let them be overlooked easily on this point. Zellulalgie is not constricted on sites with strong developed panniculus adiposus. In contrast to the inner organs and the integument, the anatomical exact and different disposition of the *ligament-muscle-apparatus* allows the precise localisation of tissue changes. The local precision is at the same time the base of the clinical checkability. The circumscribed tissue changes of the ligament-muscle-apparatus become often painful at rest as well as under normal tension and movement exposure.

They become rapidly a field of view of practical medicine. These features makes the ligament-muscle-apparatus to the clinical most important manifestation organ of spondylogenic reflex syndroms. The critical significance becomes only clear through the possibility of reflectoric-mecanical feedback between muscle tension disorder and causing functional segmental disorder of the vertebra and reverse. Therapeutical, the patogenetic vicious circle, which emerges thereby on the axial organ, can only be escaped through simultaneously elimination of the muscle tension disorder and the functional segmental disorder of the vertebra. Also for the muscle-ligament-apparatus the localisation patterns of tissue specific changes remain crucial characteristic of spondylogenic-reflectoric causation. The changes in the tissue are located with the diagnostic method, assigned locally and identified at first as known forms of the inflammatory soft tissue rheumatism, for example as tendonosis, tendovaginitis, bursitis, myositis etc. If these simple and apparent isolated clinical units should be captured and understood in the bigger context, they need first systematisation. The order is the essential precondition of founded interpretation with regard to reasonable diagnosis.

The order problem presents the question after the principle, which underlies the system. Classification under consideration of the external function, after vascular, segmental, radicular, neural and arthral principles haven't lead to a clear, satisfying and transparent result. The apparent isolation of the single phenomenon isn't resolved in productive way through the systematisation of these main ideas. In contrast, these ways lead diagnostically into an impasse, instead of a better and deeper understanding.

One step further leads the order under simple anatomical considerations. This shows, that apparent secondary phenomena in reality are strung like pearls on the functional unit of muscle and belonging ligaments. With tight, long stretched muscles this combination can be proofed easy. With wide, flat or fan shaped muscles, it is only apparent through further division. The smaller units, which have to be defined, are not anymore founded anatomically, but clinical. They consist in certain longitudinal stripes of the muscle and their belonging ligament parts, which are extending these stripes to the origine and to the onset. This unit takes off only under clinical-pathological conditions from the remaining muscle and his ligaments, but then with constancy and without any variability neither with the single patient nor between the individuals. We name this clinical functional unit after her components as *myotenen*.

Smaller muscles, partly also big ones, built one single myotenen, while others include a majority of myotenenata. The myotenen is defined clinical-local. It's the clinical unit of the reflectoric-functional muscle-ligament-disease. Basic phenomenon of it's reflectoric-functional disorder is the *myogelosis*. Clinically this situation presents itself as continuing,

involuntary and isometric tonus augmentation in the myotendon. It's pain free and doesn't prevent the external muscle function in clinical verifiable dimension. After short latency – defined through the grade of the tonus augmentation and secondary factors – it displays under the myogelosis posed myotendon with lawfully regularity the development of tissue changes, with the attribute to degrade the stimulus limit of local pain receptors. After a longer phase of rest, the changes are painful to pressure, functional overstraining or just spontaneously painful. The first tight described *tendinosis* is built at the origin and the onset of the ligaments. With little specification they can be extinguished clinically only through the exact located palpation pain of the side of insertion.

Distinctive forms are captured by the palpating finger through the smooth limited local flow and aggregation of the ligament. The insertion tendinosis is not limited to ligament-bone-transitions. It can be found also on the aponeurotic insertion, for example on the onset of the m. pyramidalis on the linea alba.

The tendinosis appears simultaneously at the origin and the onset, so that inversely analogy is given from the beginning. The mirror image is decisive characteristic of the myogelosis depending tendinosis. Resultant is besides the diagnostic control possibility, a practical valuable criterion of the anatomical right classification.

With the insertions of the ligaments, the muscle also underlies often to a change, which is equivalent to the tendinosis. It is clinical-palpatoric represented as constant, to pressure or spontaneous painful circumscribed texture increase of the muscle tissue. Diagnostically we describe this change as *myosis*. Myosis and tendonosis from a myotendon can be concentrate conceptual and also be understood as unit under the description myotendonosis or myotendonose.

The expression myotendonosis is ordinary used in the rheumatology since long time, but more in the meaning of casual contemporaneity and more or less without relation of coexistence of myosis and tendonosis. In clear contrast to other aetiologies, appearance and localisation of these changes of the spondylogenic reflex syndrom are not casual. They emphasize in contrast through regularity and exact local determination. Diagnostically decisive is the evidence of their systematic order on the myotendon. To distinguish from different – for example traumatic – myotendonostic constellations, we name the spondylogenic-reflectoric as systematic myotendonosis.

The myotendon as clinical essence can only appear in clinical characteristic, thus as systematic myotendonosis. This feature allows the formula: The Spondylogenic-reflectoric disease of the muscle-ligaments-apparatus is demonstrated on the myotendon as substrate in form of systematic myotendonosis. Pathologically considered, the systematic myotendonosis presupposes a spondylogenic conflict situation. Local, for example traumatic, circulatoric, local-mechanical or general factors as psychic tension, metabolism and hormonal disorder, can be excluded for this reason as possible, locally effective, triggering cause.

As secondary, modifying, the quantitative development and pain constitutive factors, they're of importance. Probably the myogelosis is the only possible primary cause of systematic myotendonosis, and the regularly appearing tendonosis of the origin and the onset are determined through local factors. This requires, that these factors a priori are given on each insertion.

Under the influence of duration and intensity of the myogelosis, the initial, simple inversely form of the myotendonosis extends through development of other tendonosis and myosis on the myotendon. Obviously mechanical and probably structural factors determine their predilection (?). First of all they're places of direction change of muscles and ligaments. We name them deflecting tendonosis and myosis. Typical place is the direction change of the m. deltoideus over the caput humeri or the m. sartorius on the medial condylus of the femur. Deflecting tendonosis can be found regularly with myotendonosis of the long foot muscles in

the area of the malleoli and the of the ligamentum cruciatum. Other predilection sites are defined through the transition of the muscle into the ligament. The tendonosis of the soleus and the gastrocnemii at the beginning of the achilles tendon, is a common example. Typical myosis develops often on free muscle borders, like they're characteristic for the muscoli trapezius, pectoralis maior, latissimus dorsi, gastrocnemii. Intensity and duration of the myogelosis favor the length of myosis and tendonosis on the myotenon. A confluence of the initial tight located and clear distinguished phenomena is possible. The myotenon becomes painfull in the whole extension. Only rare cases develop the image of a true infection. The spondylogenic-reflectoric myotendonosis is like that of other aetiology, the preliminary stage of irreversible degenerative tissue changements. With the change and degeneration increase, (instead of the pure change of state) the connection to the cause disolves; the peripheric tissue changement becomes pathogenetic independent. With this development the originally clear causative relations get vague. That stage demands therapeutically a new concept, and diagnostically complicates the pathogenetic judgement, which can not be based anymore only on the current finding. The determination of systematic myotendonosis is the first step of a clinical possible systematisation of non inflammatory soft tissue rheumatism phenomena. The second step extends the context, based on the new unity of systematic myotendonosis. Verified empiricism appears in certain patterns with determinated characteristics. The patterns are reproductive, qualitative identically and symmetric, locally different, unitary, can be combined and aren't interpretated through any known clinical order system. The reproducibility is general. Under comparable conditions it affirms in infinite reproducibility with every individual. Qualitative identical means, that every pattern builds itself exclusively from systematic myotendonosis, even if the number of these units diversifies from pattern to pattern. The qualitative symmetry emphazises, that the patterns with possible quantitative differences of every level, are always implemented qualitative practically in both body parts. Obviously the functional segmental disorder of the vertebra with elective onesided irritation of the invertrebral joints, is very seldom. Of special interest is the quality of the symmetry for practical diagnosis. Qualitative and quantitative symmetry in bilateral comparison are not against, but an important argument for spondylogenic cause. The judgement of a finding shouldn't be compared with the opposite side, but with similar structures of the same body part. Local difference is the constitutive and only feature, according to the single pattern distinguish from each other. The local division is certainly incomplete. A row of systematic myotendonosis is a component of different patterns. This results in partial, local overlapping. Unitary means, that the pattern always appears or is missed as a whole unit. Are parts of the pattern verified and is the belonging irritation zone still present - the evidence of quantitative less developed components must succeed empirically with adequate palpation technique. For this reason, the pattern is objectified as clinical whole unit, even if only a small part of that gains subjective relevance for the patient. With all the possible quantitative difference, the "appearance modus" according to the everything-or-nothing-law allows the solution of differential diagnostic problems, which results from the partial local overlapping. Each pattern can be combined with all others. Due to the reciprocal conditionality - it will be discussed further on - the multiplicity of the patterns is a normal case. The causal classification of myotendonosis patterns to certain vertebra is based clearly empirical. While single patterns appear clarified largely, others still are not captured. From the known central, radicular, segmental, neural, vegetativ, vasal, hormonal, arthral and complex functional order principles, non contributes to their understanding from todays knowledge. Still it is not to see through, what the patterns are meaning functionally for the organism. Our study has not overcome the stade of partial finding.

The myotendonosis disorder pattern are not only related to the torso and extremity muscles, but also to the mimic and back musculature. While myotendonosis tension disorder on torso and extremity seldom causes position disorder of peripheric joints with accompanying pathological reflex event, the circumstances on the spine are different. The myogelosis of single myotenata of the erector spinae represents a delicate disturbance of the differentiated and harmonic balanced cooperation of this complex muscle system. The myotendonosis disorder tension is characterized through tonus increase in non physiological, that means through no normal position and movement stereotyp adequate pattern. The normal, gradual gliding tension dispersal is “open worked” abrupt through the myogelosis of single myotenata of the erector. The disharmonic forces reach over the lever of the ribs, the transverse and dorsal process of the selective single vertebra and cause their sheering from the coordinated function system of the spine in functional segmental disorder of the vertebra. The plastic invertebral disks and the interapophyseric ligament apparatus with the limp invertebral joint capsules aren't a handicap for the considered micro movements. The plastic deformation of the invertebral ligament could be also responsible therefore. Over the myogelosis of the spinal myotenata the spondylogenic reflex syndromes become the cause of further spondylogenic reflex syndromes. The duration of this development diversifies in quantitative circumstances. With existent subliminal tension disorder normal movements or enforced position suffice under certain circumstances to cause the functional segmental disorder of the vertebra. Due to immovable local determination of the components, spondylogenic reflex syndromes provoke identical pattern disturbance of the axial organ and with it the periphery. Individual factors as basic shape of the spine, the position and movement stereotyp, the muscle force and muscle length ratio, are playing a role on this *third step* of the systematisation, so that the development and the specification on this level don't extrapolate exactly anymore, but though can be predicted with approximation.

Within the axial sceleton the disturbance doesn't has to remain tied to the primary causes. A primary spondylogenic reflex syndrome can heal, the secondary induceted instead can persist and may become prima for others. In this way the disturbance develops from the primary cause – for example a local trauma – and affects somewhere else, similar to summer storms, which don't have to unload necessarily on an agglomeration place of thunderclouds. In praxi we found often a complex clinical image, which comprise a plurality of spondylogenic reflex syndromes of different development level and specification, which are definying each other partly, their basic triggering cause may disappeared already from the context. Such a clinical image, such disturbance development, which underlies the interdependence of different spondylogenic reflex syndromes, demands a summary definition. We suppose therefore the definition *rachiosis*.

## **Appendix**

Musculature of the lower extremities in spondylogenic-reflectoric correlation

P= only a myotenon of the muscle

For further particulars we refer to planed publications.

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# **An Attempt to Define Radicular and Pseudoradicular Syndromes**

**M. Sutter**

Since the description of a herniated disk as cause of a radix irritation, beltlike or lamellar radiating pain raises suspicion to a radicular syndrome or to a so-called neuritis. In the majority of cases with radiating pain as cardinal symptom, the diagnosis of a radicular syndrome can not be confirmed, though the pain symptomatology seems to exclude other interpretations. Beside the definition of the radicular, therefore also the definition of the pseudoradicular syndrome has taken place. But the prefix “pseudo” doesn’t create an affirmative definition. It denies merely the radicular genesis of clinical substrate, which is defined at first only through similarity with the radicular phenomenon. For this reason the question raises to define the character of the pseudoradicular and its exact boundary of the radicular. These questions can only be answered through exact understanding of the diagnostical and clinical definitions of the radicular and the pseudoradicular.

According to its nomination the radicular refers to an anatomical defined section of the peripheral neural system, to the radix. The clinical praxis defines the radix not only as the ventral motor-driven and sensitive dorsal radix, but also the short spinal nerve till its ramification into the thick ramus ventralis, the thin ramus dorsalis and in the vertebral canal self-contained fine ramus meningicus. From the functional view this line of the segmental neural system represents all somatic-nerval benefits of the segment. In contrast to the somatic ones, the vegetative fibers pass the radix without subordination under the segmental principle. Resultant, that the diagnosis of a radicular syndrome – if it wants to bear against strict criteria – only can consider the nerve functions, which can be located on a clinical checkable area of a determined spinal nerve. Motor activity and sensibility plus certain reflexes with motor effect belong to that.

In contrast the clinical most common cardinal symptom, the lamellar radiating pain, has only conditional conclusiveness for following reasons: Radicular pain follows the course of the dermatom, which can show extensive individual differences. There are different, partial contradictorily schema of the dermatom limits. Pain is something subjective, it can often only be located approximately by the patient. The pain source is always smaller than the accordant zone of subjective sensation of pain. This diversifies with the intensity of pain and with the significance of the pain source in the body scheme (emission). Pains of the extremities are sensed subjectively lamellar, even if the cause is constricted to narrow described places. The most common misinterpretation bases on the confusion of peripheric caused pain with the ones of radicular genesis. The radicular pain emerges through the irritation of pain transmitting fibers in the radix. The subordination of their function through compression leads to analgesia of the related periphery. Only the irritation by obtained function activates pain. According to its nature the radicular pain is a projection pain. Radicular caused projection pain is activated in the periphery, according to its body schematic competence of the irritated radix. Pain fibers transmit centripetal. They don’t have any direct influence to the functional state of their peripheral main tissue. The pain receives evidentiary value for a radicular syndrome only through a clear verification of its central genesis and the dermatom suitable projection. On the other hand the pseudo radicular syndrome is a pain situation (defined in first approximation), which imitates a radicular syndrome, but its genesis is not from radicular nature. The critical differences to the radicular are the absence of the radicular paresis and sensibility disfunction on the one hand, as well as the existence of peripheral pathological tissue changements as pain causation on the other hand. But also in this conception the pseudo radicular has still a negativ notion. It misses the representational autonomy, because

peripheral tissue change as pain causation only can not constitute a “nosological”(?) unit.

The phenomenon of the pseudo radicular demands an exact version on the conceptual and clinical-diagnostical level.

*Brügger* has dedicated an important part of his scientific work to the problem of the pseudo radicular syndromes. It's his credit to discover and describe the causal correlation, which exists between the stimulus situations of the joints and determined localizations of the so-called soft tissue rheumatism. These correlations are of a vegetative neural nature. They are transmitted through reflexes. The effect of a reflex depends on the functional possibilities of the “success organ”(?). If the success organ is a muscle, the effect will be a change of the tension. The connective tissue is the success organ of the pseudo radicular reflexes.

Therefore they're of vegetative nature. Their effect is a physico-chemical state change.

This conclusion is probably a neuro-physiological speculation. But it is based on a fundamental clinical ground, because the state change of the tissue is accessible to direct palpated cognition.

The pathogenetic factor of the change of the soft tissue is according to that a pathological reflex event. Other arguments for these thesis are first of all the exact described local ligation of the changes and second their healing up under the unconditional supposition of previous rehabilitation of the causal stimulus state of the joint. The change of the soft tissue is tissue- and not stimulus specific. Influences of other nature can cause the same tissue changes as the pathological-reflectoric interferences. *Not the nature of the peripheral change is the essential of the pseudo radicular, but the ordered location. That means, that besides the radicular also the pseudo radicular represents a clinical order principle, which diagnostical reliability can reach absolutely the radicular one.*

The causation for the pathological-vegetative stimulus event is a functional disorder in the for the afferenz(?) responsible receptor area; we call it *afferenz source*. It is assumed, that the mentioned reflexes already fulfill a function under normal circumstances. They're changed with disturbances in the area of the afferenz source, for example in the sense of pathological augmentation or pathological oneness. The qualitative or quantitative different influence of the success organs releases their abnormal changes. The conception, which sees the normal mechanical joint movement of a coordinated reflex act for mediation of vegetative connections between joints and soft tissue, has to be proofed by future research on the physiological accuracy. Legitimated through solid clinical basement, we enter with these hypothesis interim the field of neurophysiological speculation. The sense is to order the empirical clinical observations and consolidated findings in a interim and conceivable system. *Brügger* comes to the fore with the reflectoric connections between joints and functional related musculature. *Gutzeit* has summarized this arthro-muscular function alliance under the definition *Arthron* respectively *Vertebron* to a conceptual unit. The pseudoradicular syndrome L1 – to name an example – doesn't summarize the clinical phenomena, which are imitating a radicular syndrome of the first lumbar radix. In contrast the changes of the soft tissue are characterized, which are caused through the irritation of the movement segment between 1. and 2. lumbar vertebra. The different localisations of these soft tissue changes are summarized under the pseudoradicular territory L1. The changes of the soft tissues are amongst others clinical known as tendomyosis, as far as it concerns sinews and musculature, and as panniculosis (respectively zellulitis, zellulalgie), as far as it affects the subcutaneous connective tissue. To the territory L1 belong parts of the iliopsoas, the caput breve of the biceps femoris, the rectus femoris, the tibialis posterior and the flexor hallucis brevis. Besides the muscles also cartilage tissue in the area of the medial partellar border and the capsular tissue in the dorsal part of the ankle joint plus in the fibular part of the lower ankle joint belong to that. Part of this pseudoradicular territory are also lamellar zones of the

subcutaneous connective tissue in the area of the dorsal and lateral lumbar region. The example shows, that pseudoradicular territories don't present topographical consistency and that only partly an accordance with the correspondent radicular syndrome exists.

The ostensible similarity of the clinical appearance with real radicular syndrome is to be ascribed to the chain-like composition of the painful soft tissue. This disposition pattern is only valid for a part of the pseudoradicular syndrome. The pseudoradicular reflex is neither segmental nor radicular connected, its *afferenzis are polyradicular*.

The inner organs are doubtless influenced through a pseudoradicular reflex event. The evidence of these relations and their systematic recording presents bigger difficulties, so that the soft tissue changes of the musculoskeletal system are diagnostically far in the front. A disk radix compression syndrome without coexistent pseudoradicular syndrome isn't nearly imaginable for pathological genetic reasons, because a herniated disk must lead nearly inevitable – besides the optional radix stimulus – to an irritation of the affected musculoskeletal system. In the praxis we see radicular syndromes always accompanied from pseudoradicular ones. Disk radicular syndromes can be evaluated as rare complications of the frequent spondylogenic pseudoradicular syndromes. As well as diagnostically and therapeutically of importance is the rule of thumb, which says that the radicular always appears only in the pseudoradicular.

The irritation of the afferenz source is mostly of mechanical functional nature. But it can be also caused and maintained through inflamed, traumatic and neoplastic processes. The inflammatory rheumatism is therefore besides primary-inflammatory always also accompanied from pseudoradicular caused soft tissue changes. Acute and massive pseudoradicular syndrome activates instantly heavy pain, which often leads to confusion with other acute diseases. But with low intensity spontaneous pain sensation can be completely missing or can only be experienced in form of tension feelings, pain through effort or as early exhaustion. Often pain appears primary with other factors, like pressure, draft, weather, mental tension or trauma. The functional state change of the tissue can pass with longer persistence into histological comprehensible changes, which can be very painful under circumstances. Initially they are still reversible, but later at least partly irreversible. The pathologist describes them as degenerative. Naturally expansive changes of the tissue appear especially with subclinical chronic course.

The tissue change can be explained with the example of the panniculosis. At first a consistency and weight increase of the tissue without oedema formation can be observed. Pressure and 'pincered' exposure cause partly heavy pains. The clinic refers to cellulitis or fibrositis. If the irritation of the afferenz source disappears in short time, it comes to the restitutio ad integrum. But if longer time passes till that, the heightened consistency of the tissue persists despite weight and pain decrease. From the clinical point of view we talk now about fibrosis or panniculosis. New pain episode and new weight increase are possible with the new irritation of the afferenz source. Comparable tissue changes appear also in sinews and muscles, in firm and loose connective tissue.

Due to clinical observation and their resulting pathogenetic hypothesis the pseudoradicular syndrome is defined as: reflectoric conditioned vegetative disorder of a determined (discontinental) body zone due to continuing pathological afferenz from a joint or movement segment with possible secondary tissue specific changes in the sense of the soft tissue rheumatism.

Though primary pseudoradicular genesis the soft tissue changes can become secondary independent. They become new elements of the pathogenetic chain. The chronic changed soft parts affect often the function of the peripheral nerves. The zellulalgic episode of the subcutis can be accompanied from different dysästhesis(?). The continuing hartspann of the musculature can lead to compression syndromes of the peripheral nerves. Examples for that

are the supinator syndrome with the irritation of the ramus profundus of the N. radialis by pseudoradicular syndrome Th2, the piriformis syndrome with partial compression of the N. ischiadicus by pseudoradicular syndrome L5. Parästhesia of the hands don't need to have their causation in the cervical plexus. Their primary cause can be a pseudoradicular syndrome of 'thoracic' origin.

Probably a part of the carpal tunnel syndrome is the consequence of pseudoradicular caused swell and increase of the loose connective tissue in the carpal canal. Long-lasting muscular hantspann can lead - with change of the joint mechanic and in combination with other factors - to arthrosis. A row of observations let this pathogenesis interim assume for certain forms of the Heberdenarthrosis and the Koxarthrosis. In both cases thoracic pseudoradicular syndromes are up for debate. Through little trauma caused periostitis without spontaneous healing tendency are mostly supported through pseudoradicular syndromes. Examples therefore are certain epikondylitis and periarthritis. In these cases the trauma was only pain causing factor. The actual causation is a pseudoradicular syndrome, which deserves therapeutically special attention.

Through the muscular relations between the joints and between the movement segments of the spine, pseudoradicular disturbances can provoke other pseudoradicular syndromes, so that it can lead to an actual generalized pseudoradicular disease. It can be found in the literature under different names, for example as fibrositis syndrome or as "psychogenic" rheumatism.

Although their nearly everywhere presence in the daily praxis, the pseudoradicular syndromes have found little clinical importance till today. Through their accurate study we can gain insight into the very fine functional complexity between the single organs of the musculoskeletal system and the musculoskeletal system with the other organism. Thereby we can understand the clinical phenomena perhaps better, which were classified as vegetative and therefore as not closer interpretable as far. Perhaps we can find the actual character of rheumatic disease in the clinical pseudoradicular complexity of the organism more applicable realized than in the famous "Aschoffsche papule" (?) and in the even more famous rheumatism factor.