New Model Regarding the Characteristics of Somatic Dysfunction

Subjects: Others

Contributor: Luca Di Pietrantonio , Marco Verzella , Erika Affede , Vincenzo Cozzolino , Luca Cicchitti

Somatic dysfunction (SD) is classified by the ICD 11 as a “Biomechanical lesion, not elsewhere classified”; however, the definitions are not equally shared and codified by osteopathic professionals.

exclusion zone water | interstitial fluid pressure | water | somatic dysfunction

1. Introduction

The main means available to osteopathic medicine is to assess tissues by palpating, in particular tissues of the musculoskeletal system, with the aim of diagnosing a possible somatic dysfunction (SD).

By underscoring some contradictory aspects, several researchers have called into question SD, by defining it as a nosological entity detectable on palpation [1][2][3].

SD is classified by the ICD 11 [4] as a “Biomechanical lesion, not elsewhere classified”; however, the definitions are not equally shared and codified by osteopathic professionals [1][5][6][7].

SD presents the characteristics of impaired or altered function of components related to the somatic system, involving skeletal, arthrodial, and myofascial structures, and osteopathic manipulative treatment (OMT) is aimed at the treatment of SD [8][9][10].

The osteopathic literature describes the relationship between SD and OMT in many studies [10][11][12][13].

OMT is a drug-free manual medicine, a patient-centered, whole-body intervention. OMT has shown positive effects in different fields such as gynecology and obstetrics, neonatology, chronic inflammatory disease management, and musculoskeletal disorders [14][15][16][17][18][19].

There are many aspects to consider regarding the etiology and diagnosis of SD, and the osteopathic literature provides details on the signs that characterize it, including tissue texture changes [8][20][21][22].

Over the last few years, some researchers have proposed a variety of interpretation models in order to clarify the mechanisms of onset and the inherent characteristics of tissue alterations concerning SD. Among such models, there are also clinical reasoning and decision-making procedures suitable to establish a treatment routine [23][24][25][26][27].

Recent knowledge suggests that tissue, and, in particular, connective tissue, may react by modulating the inflammation degree. This issue should also be extended to any response to OMT, and several studies show the efficacy of OMT on inflammatory tissue levels [28][29][30][31][32][33][34][35].

LGI would act on the ECM, and alter its structure, such as in fibrosis, which is defined as a lesion of the connective component in an organ or tissue [36].

These alterations occur through mechanisms mediated by the environment in which the tissues are placed, namely water [37][38][39][40][41].

The water under consideration is water present in living matter. It has particular biophysical characteristics, which could exemplify the functioning of both healthy and injured tissues [39][42][43].

2. New Model Regarding the Characteristics of Somatic Dysfunction

With the acronym TART (tenderness, asymmetry, range of motion abnormality, and tissue texture changes), osteopathic literature accurately provides the characteristic elements of SD, at which OMT is aimed [7][8][21]. However, some researchers disagree on the relevance to be attributed to different clinical signs: some indicate the range of motion abnormality as fundamental for a diagnosis of SD, but there is no univocal evidence on the reproducibility in the evaluation [44]. Other researchers suggest the need for the presence of at least 2 of these 4 signs; still, others do not consider the sign of hypersensitivity or tenderness [1][5][6][7][45]. Regarding the asymmetries of the musculoskeletal structures, these can occur for a variety of causes, and are, therefore, difficult to attribute solely to SD [46][47][48].

In light of the results of this research, researchers believe that among the 4 clinical signs considered, tissue texture changes are the most significant to define an SD, thus proposing the hypothesis that SD can be compared to a condition of LGI.

The mechanisms underlying SD are still widely discussed in the literature, but it is reasonable to think that without first having tissue texture changes, caused by inflammatory phenomena, the presence of the other three clinical signs is not possible.

The researchers suggest that an inflammatory phenomenon could determine an alteration of the tissue as described in the chapters above, and only subsequently tenderness, altered movement, and asymmetry of the musculoskeletal structures can occur (**Figure 1**D).

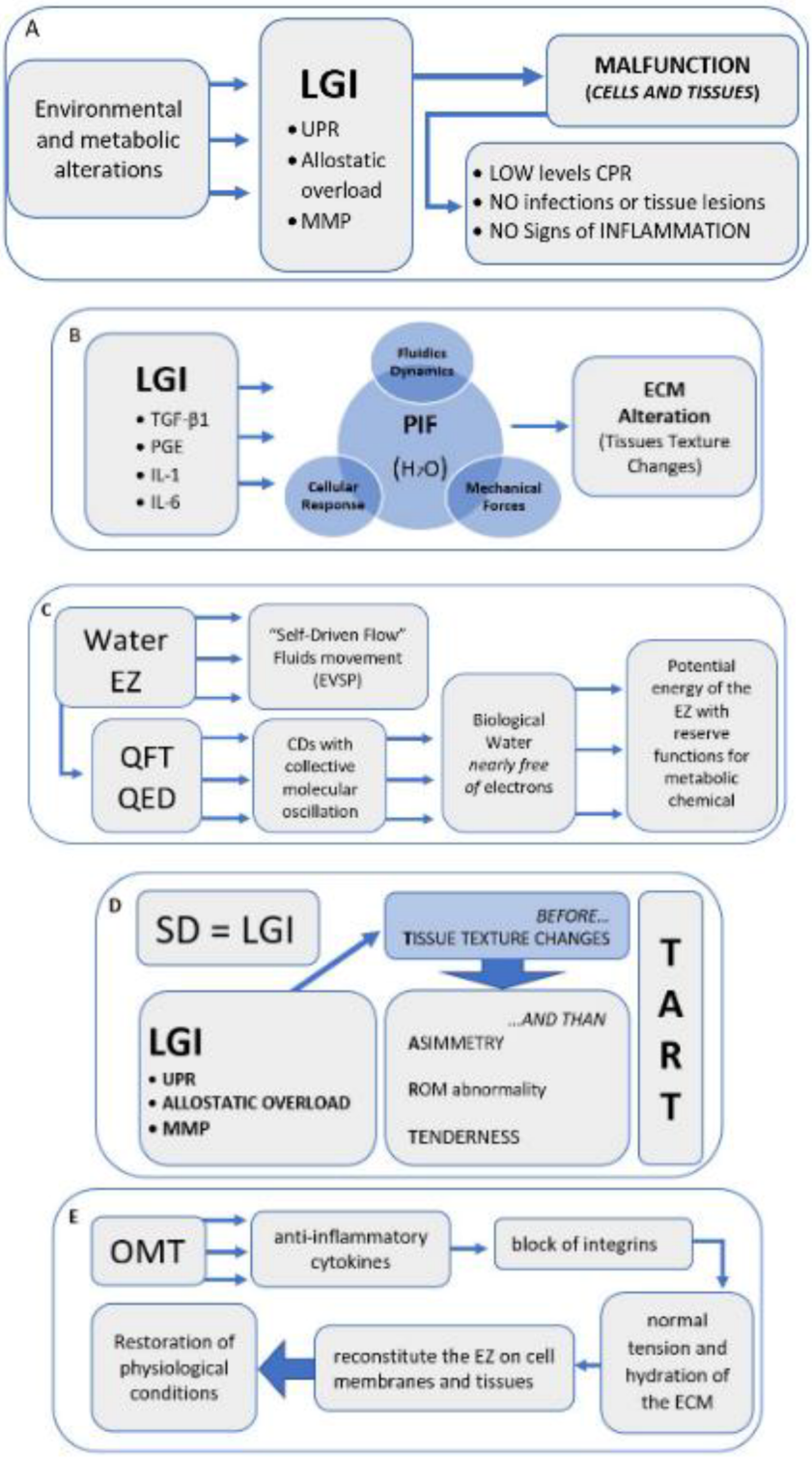

Figure 1. Narrative flow charts.

The timing just described could be explained by one of the most accredited mechanisms of the onset and maintenance of SD: the neurogenic inflammation [1][49][50], in which the primary afferent nociceptors (PAN) determine the release in the periphery of neuropeptides, such as substance P and calcitonin gene-related peptide (CGRP). The neurotransmitters mentioned above are released into the peripheral peri-vascular and extracellular space through an antidromic signal, causing a local inflammatory response with alterations of the surrounding tissue. It should be noted that this area, by means of the axonal branch, can be very large [51].

These neuropeptides have vasoactive functions, recalling immune cells, activating mast cells, and releasing histamine, thus acting on the trophic state of the innervated organ [21][49][51][52]. Together, they contribute to the possible genesis of tissue alterations, also influencing the recovery of tissue lesions and their repair [53].

The nerve fibers involved would be predominantly the poorly myelinated C or A-delta, fibers of the interoceptive component which, therefore, represent the afferent portion of the sympathetic efference [54].

It has been demonstrated that sympathetic efference plays a decisive role in the onset of inflammatory phenomena [55]. These findings agree with what Denslow and Korr underscored regarding SD, as it pertains to expressiveness of phenomena related to neurogenic inflammation [56] and autonomic sympathetic innervation [57].

There may be mechanisms capable of leading to tissue alteration, which are associated with the dynamics of neurogenic inflammation. These dynamics are all probably linked to inflammatory phenomena, such as the unfolded protein response (UPR) [58][59][60][61], as well as the alteration of the functions of the MMP [62][63], which would determine changes to the functions of the ECM [64][65]. Last but not least, the allostatic overload would cause tissue alteration [66].

SD does not represent a real pathological condition [4]. In fact, as for LGI, it would not have a direct cause, such as trauma or tissue injury. Rather, SD appears as an alteration in tissue function, a sign of altered homeostasis, often lasting over time, and, like LGI, it can be placed between a homeostatic basal state and the actual inflammatory response [37][67].

There are studies on the efficacy of OMT in healthy people diagnosed with non-symptomatic SD [45][68][69][70]; the clinical conditions of these subjects could be associated with LGI, in which the blood inflammation markers are modest.

However, the signs of DS are not associated with the classic signs of inflammation. SD represents a sign of metabolic alteration that manifests itself with the alteration of the tissue texture, leading to tissue fibrosis and possible sclerosis and, therefore, is diagnosable through palpation [20][21][36].

The existence of a restriction barrier within the range of motion, a characteristic sign of SD [20][21][22], implies the alteration, both quantitative and qualitative, of a tissue or a joint region in a given district. This alteration is generated on an inflammatory substrate, without necessarily showing signs of classic inflammation [22].

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