The fixation element of the articular subluxation:

More than a vertebral dysfunction. Part 1 of a series.

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Abstract: This narrative review examines the limited and varied interpretation of what constitutes evidence in evidence based chiropractic practice. The authors report a bias towards only one of the three evidential The majority of spinal motion studies appear to focus on the anterior vertebral motor unit. This paper discusses the vertebral subluxation (VS) element of segmental dysfunction within a facet's centrode in relation to mechanical articular fixation along the path of instantaneous axes of motion. In effect, there would be three segmental axes of motion, one through the vertebral body, and two within each zygapophyseal joint. Discussion is offered here on the loss of facet joint motion identified as a vertebral fixation as being but one of a complex of factors comprising a vertebral subluxation (VS). It is appreciated that a change in segmental motion such as a vertebral fixation would affect all axes. It is generally accepted that mechanically, a vertebra may be fully fixated (locking), partially fixated, or subject to aberrant (erratic) motion. There is also a classification of a hypermobile subluxation – a condition not addressed in this dissertation. In essence, a VS may comprise varying degrees of dysfunction, displacement and somatosensory activation. It is seen here as a more complex consideration than just a minor mechanical disturbance. Lineal displacement and rotation may vary considerably depending on the spinal region. Somato-autonomic stimulation may be initiated by activated somatosensory sensations such as nociceptors, mechanoreceptors and proprioceptors. As noted by the World Health Organisation (WHO) definition, there are three main elements in the chiropractic model of a vertebral subluxation. This discussion is presented to explore the pathophysiological fixation element of the dysfunction in the segmental subluxation. It may be regarded as a relatively common form of dysfunction attended by chiropractors and others. The fixation has been adopted elsewhere under such synonyms as blockage, dysfunction, locking and blockade. Theories regarding the biological mechanism of a fixation are discussed and our preferred hypothesis of negative pressure (suction or adhesiveness) resulting in contraction of the articulating surfaces.

Indexing terms: Vertebral subluxation, Facet fixation, Segmental fixation, Vertebral subluxation complex (VSC).

Introduction

As outlined by the WHO definition, a chiropractic subluxation includes complex considerations to be differentiated from a purely mechanical disturbance. It could be noted that such a limited interpretation of a mechanical disturbance could only occur on a dry skeleton articulation. Common associated symptoms of pain or tenderness can be apparent through somato-autonomic, somato-somatic and somatovisceral neurological reflex activation which may occur at a particular level following mechanical disturbance of innervated structures. Vertebrogenic sciatica, cervicogenic headache, mechanical back pain, paresthesias and intercostal neuralgia could be regarded as common examples of somatosensory and somato-autonomic activation. (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12)

> *... the application of evidence based principles is far from even let alone equal resulting in papers published by chiropractors being seen as having less*

The term subluxation has been a diagnostic identifier nominated by chiropractors for over 120 years. It encompasses more than the very limited traditional term. Other terms have since been proposed for the physical-mechanical dysfunction element of the lesion although this implies just the mechanical element of the disturbance. As indicated in the WHO definition, the lesion involves more complexity than that, and is also well recognised as such clinically. (11, 12, 13, 14, 15, 16)

The WHO definition of the subluxation explicates the necessity for a more encompassing rationale than that of a narrow mechanical image. The range of factors suggests more of a complex clinical finding in this definition: '*A lesion or dysfunction in a joint or motion segment in which alignment, movement integrity and/or physiological function are altered, although contact between joint surfaces remains intact. It is essentially a functional entity, which may influence biomechanical and neural integrity.*' (1)

The WHO definition of a subluxation also recognises more than just the minor displacement of osseous structures at an articulation. The pathophysiology of sensory disturbances must also be considered as they are known to activate somatosomatic, somatosensory, and somato-autonomic reflexes. In addition, other soft tissue structures may be disturbed and deserve to be considered. These include ligaments, muscles as well as their relationship with neural, vascular and lymphatic structures, and their functions. These factors may combine to constitute more of a clinical condition, especially when considered in association with clinical signs and symptoms. Even if there was only displacement there would be sensory disturbance (somatosensory) of mechanoreceptors - even just proprioceptors.

This WHO definition must supersede the traditional one of a basic osseous displacement which overlooks these inherent associated disturbances rendering the traditional version somewhat deficient in relation to articulations.

The New Zealand Inquiry also explains that '*When the chiropractor uses the term "subluxation", however, he is referring principally to a functional defect in a joint...and further that 'the chiropractor's first emphasis has been on function rather than structure.*' The mechanical element of a subluxation may take the form of a hypermobile segment, or aberrant motion, or the most common - a fixation. Haldeman indicated the subluxation was largely dynamic, (and) functional as well as structural. (17)

Haberl et al suggest that aberrant motion may be described as the pathologic motion for specifying conservative treatment concepts. (18)

The articular centrode

We could find little definitive explanation outlining the mechanism of a facet fixation. Consequently we present essentially a discussion based on hypotheses. In addition, we explore the concept of a centrode as being the path of moveable axes of rotation. This raises the interesting research possibility of precisely where in a centrode a particular fixation may occur in a particular Range of Movement (ROM) on an articular surface. (11 p 232-235,270-273; 12 p 121-197)

In 2006, Rousseau et al identified four separate centres for helical axes in lumbar vertebrae. A similar finding by Wachowski et al in 2010 also noted at least four finite helical axes in axial rotation of lumbar vertebrae and that these independent helical axes migrated along distinct centrodes. Wachowski identified a zone of migratory instantaneous helical axes, with Rousseau noting that the centrode as the path of the instantaneous axis of rotation. (19, 20, 21, 22, 23)

We suggest that the mechanical fixation element of a subluxation must therefore reside along its centrode, and well within the physiological limits of a vertebral facet's ROM. In addition the centrode by its very nature must represent a neutral zone – an area within the facet surface. A combined pathophysiological fixation/displacement would therefore lie well within the joint and

as the corrective adjustment is a release only of that fixation, there is no need to take the vertebra beyond its physiological or anatomical limit. Indeed when displacement is also evident, the release impulse should be towards the central neutral zone, not away from it, and towards a physiological limit - but not beyond it.

As the centrode encompasses a zone of instantaneous helical axes, it is not likely to broach the physiological limit. However, in our research, it was noted that studies of vertebral axes of rotation of motion primarily focus on the vertebra as a whole, with little consideration of the facet axes except for loading forces. (24, 25, 26, 27, 28)

Although there are two types of centrodes - a body centrode and a space centrode, it is suggested in the case of vertebral facets, where the points of contact are instantaneous, the classification may alternate from one surface to the other. Suffice to conclude that a distinct path of axes has been demonstrated. (29)

It was concluded here that the fixation element of a subluxation occurs along its centrode which exists within the central zone of instantaneous axes of movement of the facet surfaces. We would suggest that any subluxation displacement does not translate beyond this axis zone but along its centrode. It may also importantly explain why the subluxation is sometimes difficult to demonstrate on x-ray as it is primarily a dysfunctional state. The release of a fixation at this level would not then be conducted beyond the joints physiological range, but well within the joint's limits.

It may also be said that it is not possible to have a displacement without a fixation, otherwise the displacement would not be immobilised. On the other hand, a fixation without displacement would be possible if it occurred in the neutral centre of motion within the centrode. (21, 19, 30)

The actual mechanism of a fixation has yet to be definitively identified. However the symptomatic presentation and its remediation with associated positive symptomatic response being reduced or eliminated has been demonstrated. The loss of clinical signs and symptoms would suggest a clear cause and effect association. (31)

The vertebral facet fixation as proposed here is an articular immobilisation within a zone of its axes called a centrode. Wachowski et al and Rousseau et al have both identified multiple axes of motion. Wachowski tracked the moveable axis of the independent helical axis (IHA)-up to 10–60 mm within small angular intervals (±1 deg). The centrode may be described as the path taken by an instantaneous centre of rotation during a range of motion. (19, 21)

In an example of the remarkable range of facet translation in lumbar facets, Svedmark and colleagues noted 3D physiological facet translations averaging 6.5 mm (L4/5) and 4.65mm (L5/S1) in normal subjects. (32)

The vertebral facet surface contains a smaller functional region as a more centralised zone containing the axes of rotation – the centrode. This neutral zone of helical axes is then surrounded by a secondary contact zone enabling the smooth translational movements. (Fig 1.)

Jaumard et al present a diagrammatic outline of an articular surface which depicts an elliptically-shaped inter-articular contact region of the cartilage on the inferior facet surface, the synovium, and meniscoids of a lumbar vertebra. (Figure 1) The author (PR) estimated that the articular surface covers approximately 75% of the total cartilage surface in this instance. (33)

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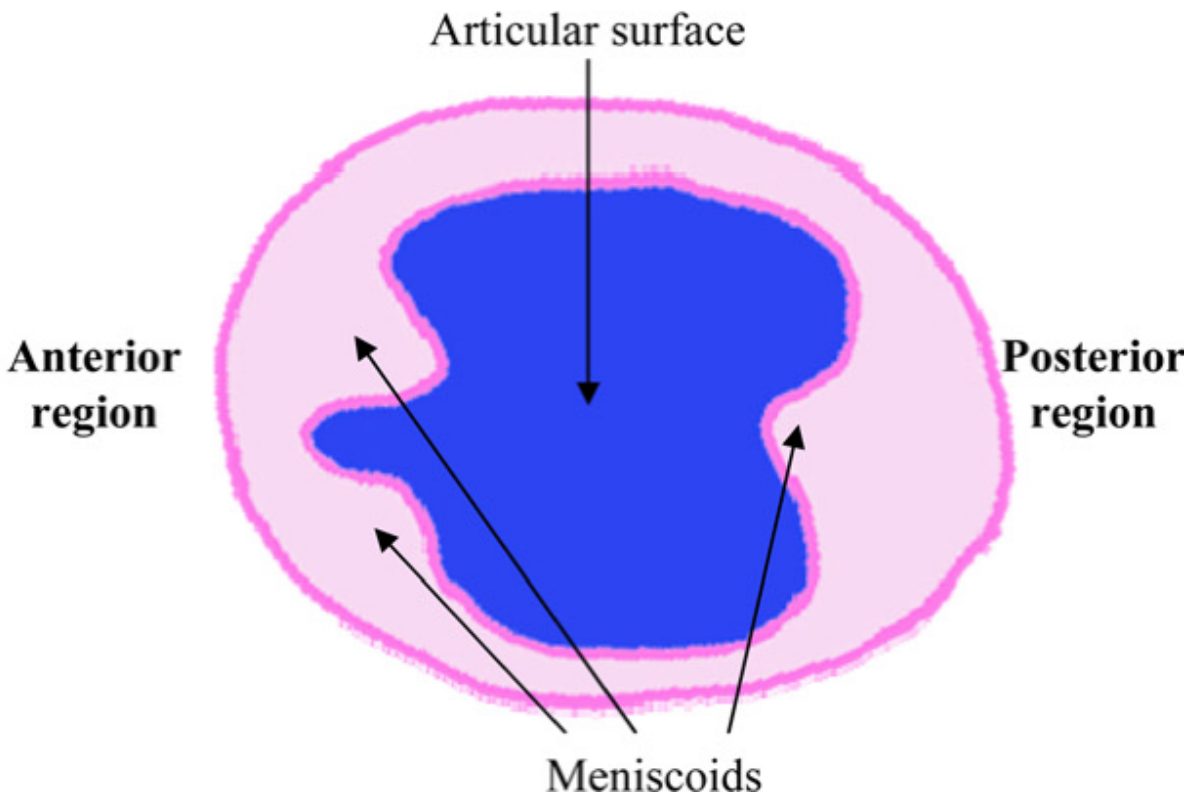
Figure 1: Adapted collectively from Jaumard et al.,2011 (33), Martin et al., 1998, (34) Pierce et al.,2009, (35) and Bogduk and Engel, 1984. (36) [From: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3705911/figure/F2/]



Dysfunction element

The cessation of segmental movement as discussed here is not a surgical fixation but is suggested to be a type of *dysfunctional adhesiveness with negative pressure at the synovial surfaces.* It is theorised here that a vertebral fixation is a combination of facet surface negative pressure (suction) compounded by hypertensive muscles (particularly intrinsic spinal musculature), either spontaneously reactive due to mechanical trauma or strain – being probably the most common finding, or predisposed from chronic tonicity from stress, posture, or muscular response to biological irritants. (37) In the same vein, Kawchuk and colleagues note that joint distraction in synovial fluid leads to *tribonucleation*, '*a known process where opposing surfaces resist separation until a critical point where they then separate rapidly creating sustained gas cavities*.' (38)

A paper by Gongal'skii and Moroz in 2002 reasoned that *wedged* meniscoids could be responsible for fixations *functional blocking* and rendering it immobile. Although mentioned elsewhere (33,36), this theory would be different to our concepts. However, they also indicate that mechanical disorders such as joint hypomobility may need to be addressed to obviate capsule and cartilage degeneration, and for prophylaxis.

We hypothesise that meniscoids might be early effects of joint degeneration rather than a cause. (39,40)

Dysfunction

A superficial but ready analogy of a fixation may be similar to that of a metacarpophalangeal (MCP) or other finger articulation with or without a sensory sensation of stiffness. This typically registers as an awareness of a change in the joint's biomechanics. Metacarpophalangeal joints would typically exhibit less neurological signs and symptoms than those found in the vertebral fixation. The fixation may also accompany an instinctive urge by the owner to manually distract the finger to release a sensation of restrictive pressure. This action is usually accompanied by a familiar sonic click called a cavitation. This release may be considered a similar principle to the audible adjustment in the release of a vertebral fixation. The vertebral fixation would generally exhibit accentuated sensory activation due to the greater proliferation of sensory mechanoreceptors and nociceptors in the spinal articulations. It also has the potential for greater neurological influence due to these rich neural associations.

We suggest that the traditional conservative diagnosis of back or neck pain seems to primarily focus on the nociceptive aspect. The somato-autonomic and somatovisceral activated reflexes seem to attract limited attention compared with their potential source of the disturbed function and neural activation of vertebral segments – subluxations.

There are two basic *biomechanical* considerations in a subluxation. These primary elements comprise the loss of normal, free-flowing joint motion - a loss of joint physiology, and this dysfunction may be complete, partial, or aberrant. The second factor is the position of the vertebral facet at the time of it becoming fixated. It is suggested here that the fixation must occur within the centrode – the path of instantaneous axes of a joint's normal range of motion. This infers that fixations may occur at different sites within the centrode of the same joint. This dynamic zone would include the physiological neutral resting state.

As if endorsing the subluxation concept, Alapan and colleagues indicated the importance of dysfunction by stating that *'any dysfunction in this 3-joint complex can cause abnormality in the translational motion of the segment. In the present study this change in the length of the centrode, and thus the translational motion, was evident in all directions of motion.'* (41)

A facet fixation may be identified as one form of *dysfunction*. This designation could be seen as a somewhat generic term which has been applied to a number of disrupted biological functions. If applied to vertebrae as *somatic dysfunction,* the term could be regarded as a somewhat limiting term implying just the mechanical element of the more complex subluxation. (42, 43, 44)

In reference to a functional spinal unit's instantaneous center of rotation or disturbance of only somatic structure such as muscles, Schmidt et al state that The *loss of mobility within its range of motion is an indicator for mechanical disorders and is relevant for the development of motion preserving techniques.'* (25)

Jaumard et al analysed vertebral facet motion in computational and clinical studies, they stated the '*mechanical behaviour ensures the normal health and function of the spine during physiologic loading but can also lead to its dysfunction when the tissues of the facet joint are altered either by injury, degeneration or as a result of surgical modification of the spine.*' Further, they recognise a '*mechanotransduction processes by which mechanical loading to the specific tissues of the joint translate into signals that drive physiologic responses in health, injury and trauma, and spinal degeneration.*' (33)

While exercises may be prescribed to complement adjustments and manipulations, they would be of limited benefit while restricted joints continue to exhibit limitations. It would appear reasonable, and clinical experience would confirm that mobility and muscle strengthening exercises are more efficacious from the start when articular motion is physiological rather than pathophysiological. (45, 46, 47, 48)

We also note that undisturbed vertebral segments (those normally functioning) do not appear to exhibit associated signs or symptoms, but subluxated ones do. Addressing those subluxated segments has been documented and clinically demonstrated to alleviate many of these associated signs and symptoms.

Displacement element

This second element of displacement still defers to the traditional definition of a subluxation, but we suggest there cannot be displacement without dysfunction – fixation. Displacement relates to articular positioning of the articular fixation at the time fixation occurs. We conclude that displacement of an articular facet is a state within the range of the axes zone. and could not occur without becoming fixated otherwise it would return to its normal neutral state, or it may become a partial fixation exhibiting aberrant motion. We also maintain that at times, even slight vertebral displacements have the potential to create signs and symptoms – a common state for patients seeking manual care. Many if not most of these signs and symptoms can be related to the disturbance of neural sensory beds in the vertebral facets, or other articulations. (11, 12)

The theory behind the finding of clinical subluxation (and the osteopathic somatic dysfunction) was further supported more recently by Winter and colleagues. In 2018 they recruited 20 adult subjects. Using ultrasound they correlated palpatory diagnoses with landmark measurements. (49)

Taylor and Romano opine that the traditional conservative definition of a subluxation is a narrow structural model, while the broader chiropractic model incorporates functional and autonomic elements as well. (50)

Further recognition of vertebral displacement was noted by Ross and Moore in 2015. They stated that subluxated vertebrae as being in articular *apposition* - the positioning of things side by side or close together. A separation of vertebrae greater than 2mm, they regarded as *diastatic* - an abnormal separation of parts normally joined together. (51, 52)

Harris et al base occipitovertebral displacement on excursion as normal so long as the separation does not exceed 12mm within a normal flexion-extension range of 10mm. Clinically however, it is suggested that any displacement would be significant depending on the correlation of a detailed examination with associated signs and symptoms. If they are indicated, dependence on radiology must be seen as only one part of an examination. For a spinal segment to be displaced, involvement of more than just an osseous shift should be considered as other structures and functions are affected. (53)

Fielding (54) categorised a rotary fixation-type subluxation at the C1/C2 level as follows:

Type I classification is fixation without anterior displacement.

Type II fixation-subluxation is with anterior displacement of 3-5 mm

Type III is anterior displacement of greater than 5 mm.

Type IV is a rotary fixation with posterior displacement.

We note that for an audible cavitation to take place in a MCP or interphalangeal joint there is no apparent articulation subluxation. This may signify that while a cavitation may be a part of an adjustment the articular release itself would be a separate factor.

Neural element of the subluxation

Perolat et al state that '*The capsule, of the facet joints, subchondral bone and synovium are richly innervated with nociceptive and autonomic nerve fibres*.' Once biomechanically disturbed establishing such subluxation elements as neuronal sensory firing, the lesion thereby has the potential to provide a pathway to noxious somato-autonomic reflex stimulation. (55)

Recognition of the pathoneurophysiological reflex phenomenon associated with vertebral subluxation syndrome, plain mechanical disturbance would be a key difference between a conservative physical, segmental displacement, and the noxious neural autonomic reflexes that can be activated by that pathoanatomical state. Apart from the activation of mechanoreceptors, nociceptive pain or tenderness can be common neural symptoms of the somatosensory sequelae triggered by the mechanical disruption. Sato et al also noted other somato-autonomic reflexes that may be stimulated. Consequently the more complex chiropractic model of a subluxation encompasses mechanical disturbance as just one of the elements in the complex as a precursor to neural involvement. (56, 57, 58, 58, 60, 61, 62, 63, 64)

The articular fixation may be seen as the etiological factor in the sensory neural activation component within the subluxation. A structural facet displacement without fixation would not tend to hold the displacement which would then allow the displacement to resume normal function or perhaps aberrant function. In support of this concept, in 2017, Ita and colleagues stated that cervical facet injury can produce a *'complicated and multifaceted cascade of electrophysiological, inflammatory and nociceptive activation of pathophysiology.*' (65)

The capsule factor

The articular capsule must allow facet translation in all planes including dilation (gapping), and rotation. It therefore resists tensile forces while possessing a degree of laxity. (66) It is noted that the capsule can elongate significantly when it is loaded. (33) Capsules also possess a contractile property through their myofibroblasts although their contribution to an articular fixation could not be determined. (67)

The muscular factor

An articular fixation may be a secondary reaction in response to muscular hypertonicity. This would be reflected in the segmental intrinsic spinal muscles in response to a particular stress, strain, or trauma. The hypomobility may also be reactive to physical (postural, or activity related), psychological (tension, stress) or chemical (neural irritant) stimuli with muscle contractions at the innervated level of the activation.

The converse may also occur with the articular disturbance leading to reactive muscular hypertonicity or even splinting (muscular guarding, or adaptation) of the adjacent muscles.

It is suggested that in instances of intersegmental facet hypomobility, hydrostatic changes in synovial fluids may enhance a negative pressure (suction) effect within the articulation with potential to further contribute to its '*fixation'* or '*blocking*'. Cramer et al stated that *'Increased resistance may be related to decreased Z joint synovial fluid, increased paraspinal muscle tension, and/or increased stiffness of connective tissues associated with the Z joints.'* (37 p 619)

We theorise that in association with a vertebral fixation there may be a partial approximation or closing of the facet surfaces. This would then have the tendency to force the synovial fluid out of the joint space and into the comparatively lax capsular surrounds. We suspect this would enhance the negative pressure and exaggerate the suction effect on facet surfaces thereby contributing to the fixation. This could be compounded by hypertensive muscles, especially the localised intrinsic spinal muscles.

In clarification, the term *locked joint* should be dismissed in the manual therapies as the appellation is already used to described a '*jumped joint*' where one articular pillar actually 'leaps' over its adjacent articular process and lodges totally outside its usual residence. It is then actually a luxated or dislocated joint. (68)

A further factor would depend on an inflammatory response in or around the disturbed facet. This may also add to the complex with tissue swelling and increased synovial fluid in reactive compensation for the irritant. In the longer term however, degenerative changes of joint surfaces and loss of synovial fluid may occur. (69, 70, 71)

*Hyper*mobility of a segment may result in a compensatory *hypo*mobility and fixation of adjacent segments. Clinically, this appears to exaggerate the hypermobile segment and could provide a rationale for evening out the mobility by specifically releasing the fixations.

A fixation hypothesis

negative pressure within facet articulation

The following theory is discussed by way of explaining both sudden and gradual onset of a subluxation. It is suggested that the release of synovial fluid in the joint may be forced into a '*pouching*' formed by the lax capsular surrounds, the capsules being somewhat flaccid particularly after trauma when the capsular laxity is increased. (33) It is further suggested as a compressive mechanical response on the surface of the facet resulting in a negative hydrostatic pressure or '*suction cup*' effect. The negative pressure of this is sufficient to pathophysiologically fixate the articulations within its axes zone. Depending on duration, there is also the potential for a degree of absorption of synovial fluid by the cartilage layer. Which may then exaggerate the negative pressure further. (72, 73)

We consider the possibility that in the event of an accompanying inflammatory reaction of the joint surface, a further degree of oedema of the cartilage and synovium may lead to an enhanced suction effect. It may also be possible for the immobility to lead to further irritation and inflammation which could justify early intervention to mobilise the articulation.

The chiropractic adjustment is directed at releasing a fixated or hypomobile articulation especially of the spine, but in a corrective direction when that is indicated. It would be expected that an articulation may become fixated as one subluxation factor within its central zones of axes often with a degree of displacement. Beyond that zone we hypothesise that translation then becomes a strain or perhaps even a sprain. That is to say, the segment may become fixated within its central zone of axes within the facet surface and that is where the release takes place with minimal movement or thrust by the clinician. To move outside that ***central zone of a joint's axis (centrode) of movement*** would constitute a severe displacement with ligamentous strain or sprain. For instance, Harrison et al found that although complex, physiological sacroiliac joint motion involved '*simultaneous rotations of 3 degrees or less and translations of 2 mm or less in three dimensions.'* (74)

If motion around the central axis zone is similarly limited to 2mm and 3 degrees it could represent a zone occupying an estimated 20% of an average vertebral facet area.

Distortion of the chondrocyte membrane and nucleus, changes in membrane potential, electric stimulation from streaming potentials and changes in matrix water content, ion concentrations and pH are all likely to be involved in the metabolic changes of compressed cartilage. Mobasheri et al who state '*Changes in hydrostatic pressure, ionic and osmotic composition, interstitial fluid and streaming potentials are sensed by chondrocytes. Responses to these stimuli alter gene expression, matrix composition and biomechanical competence.'* It is suggested here that the term *Biomechanical competence* could be interpreted as an appropriate *normal* in contrast to the *dysfunction* element of a subluxation complex. (75)

Additionally, Mobasheri and colleagues also note distinct sensitivity features of articular cartilage which is subject to biomechanical stimuli. One of these is a change in *hydrostatic pressure, ionic and osmotic composition, as well as interstitial fluid and streaming potentials* which

may affect *biomechanical competence*. It is suggested that such changes may contribute to the suction effect under the stated circumstances.

Jaumard also identified a loss of synovial fluid on joint loading but also deformation of the cartilage layer. They apply the term *physiologic dysfunction*. (33, 76, 77) They recognise the possibility of activating mechanoreceptors, proprioceptors and nociceptors although they limit this to somatosomatic reflexes.

It is suggested that to our knowledge, this is the first time such theories have been offered as to the state of a functionally fixated zygapophyseal joint which may be released by a vertebral adjustment - be it manual or via instrument. This negative pressure-suction concept may also apply to the 'cracking' sound of a metacarpophalangeal joint. However, it is noted that the release of a MCP is more of a distraction, while release of a vertebral facet can involve different lines of release, more often along the plane of the facet rather than perpendicular to it. This would suggest consideration of different factors involved in the release of these articulations.

It is hypothesised here that an impulse instrument is able to release an articulation though its focal impulse thrust *through the plane of the facet* in order to *defuse* the adhesive suction '*adhesiveness*' without the need of a gapping separation of the joint.

It is suggested further that the release of negative pressure (suction) may explain why some manipulative manoeuvres result in the audible cavitation of vertebral segments, while at other times the release of the joint is soundless. However, there does not seem to be a category of aberrant movement or partial fixation in relation to this digital cavitation.

It is possible that multiple components including negative pressure, muscular and capsular contraction, contribute to the actual fixation. Release of a fixation is often accompanied by a cavitation.It has been reported by Fryer and Kawchuk that the bursting of a gas bubble formed with the adjustment of the articulation occurs some (> .31 ms) seconds *after* the release of the joint. This space is a most interesting observation and contrary to long existing theories. (78, 79, 80)

Neural

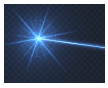
As noted by Sato et al, the pathomechanics and subsequent sensory activation with possible nociception and inflammatory sequelae together with activated somatosensory and somato-autonomic reflexes, may well explain a number of associated clinical signs and symptoms. They stated that '*The elucidation of neural mechanisms of somatically induced autonomic reflex responses, usually called somato-autonomic reflexes, is, however, essential to developing a truly scientific understanding underlying most forms of physical therapy, including spinal manipulation.'* (8)

Conclusion

It is suggested here that a primary contributory factor of the vertebral fixation is the restriction of the zygapophyseal joints and that this may be due to negative pressure (suction) associated with synovial fluid sealant. Further, that the breaking of this synovial 'seal' with the release of the fixation contributes to the sonic cavitation heard during an adjustment. It is also suggested that the suction release is largely responsible for the formation for the gas bubble during the disruption of the facet surfaces due to the input of the adjusting impulse.

The current subluxation hypotheses draw together the science behind certain clinical findings, neuropathophysiology, and structural aberrations in explaining the range of clinical findings and positive clinical outcomes. It provides a rational and reasonable understanding of the complexities of the *pathofunctional* vertebral subluxation. It is submitted that the segmental adjustment of a vertebral subluxation are central distinct components which identify chiropractic.

Further, that the release of a fixated joint is not necessary through distraction gapping, but may occur with adjustive or manipulative forces parallel to the plane of the facet surface in order to disrupt the adhesive nature of the fixation.



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