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Posture, etiology of a syndrome

Paolo Platania

Posturology guidelines and interdisciplinary case study

Pathomechanics

The mechanics section highlights Head forwarding postural strategy (HFPS) concurrent with Mandible Retruding Postural Strategy (MRPS), both featuring Cranio-Cervical Extension (CCE) but with independent features, thus a double pathomechanics and a consequently double diagnostic process is required:

- Tongue motor insufficiency (TMI) as first primary pathomechanics for the whole Head Forwarding Postural Strategy (HFPS) + Mandible Retruding Postural Strategy (MRPS) unbalance;
- Dental Conflict (DC) as second primary pathomechanics for the Mandible Retruding Postural Strategy (MRPS) unbalance only;

Cranio-Cervical Extension (CCE) mechanics is discussed prior to expose single pathomechanics in order to describe the common feature of Head Forwarding Postural Strategy (HFPS) and Mandible Retruding Postural Strategy (MRPS).

Cranio-Cervical Extension (CCE) mechanics

Cranio-Cervical Extension (CCE) combines Atlanto-Occipital joint (AOJ) extension with cervical spine forward inclination and is widely observable in the population, moreover its presence is documented to be statistically recurrent in obstructive sleep apnea (OSA) [6].

Whole body Cranio-Cervical Extension (CCE) muscle spasm

Legend:
Blu muscles: physiologic contraction, ***Red muscles:*** hypertonic muscles, ***Cyan muscles:*** inhibited muscles, *Spots*: head center of rotation, hip center of tilt, *markers*: **A** height loss due to exacerbated spine curves, **B** anterior pelvis downward rotation (hypertonic tensor fascia lata and rectus femori), **C** posterior pelvis upward rotation (inhibited gluteus and hamstring), **CCE** augmented Cranio-Cervical angle, ***Blu frame:*** physiologic posture, ***Red frame:*** CCE posture.

The model displays the implementation of the Whole body Cranio-Cervical Extension (CCE) muscle spasm and the shape assumed by the scheletal structure under the effect of the pathologic chronic and asymmetric torques:

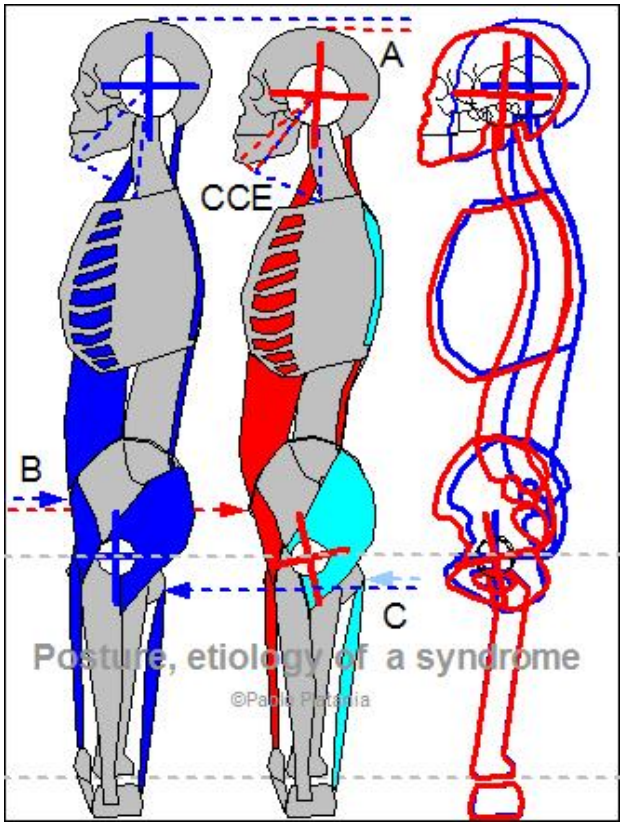
Image 1) Body physiologic posture

Image 2) Implementation of Cranio-Cervical Extension (CCE) by Head Forwarding Postural Strategy (HFPS):

- anterior hypertonic muscles to forward incline the cervical spine and incidentally anteriorly tilting the hip, hence, exacerbating lumbar and cervical lordosis
- posterior suboccipital hypertonic muscles extend Atlanto-Occipital Joint, hence, upward inclining the head,
- inhibited dorsal muscles allow dorsal chyphosis to augment, inhibited gluteus and hamstring allow pelvis to anteriorly tilt.
- hypertonic lumbar muscles increase lumbar lordosis to induce chest and head backward motion to restore the position of forwarded body center of mass.

Image 3) Physiologic/CCE comparative model

REMARK: Although the anatomical displacement induced by Cranio-Cervical Extension (CCE) at first glance may appear symmetric, it is induced by unilateral torque as the pathologic hypertonic muscles are on one side only (Head Forwarding Postural Strategy (HFPS)), this muscle unbalance introduces higly harmful asymmetric torques increasing joint torsion and degeneration.



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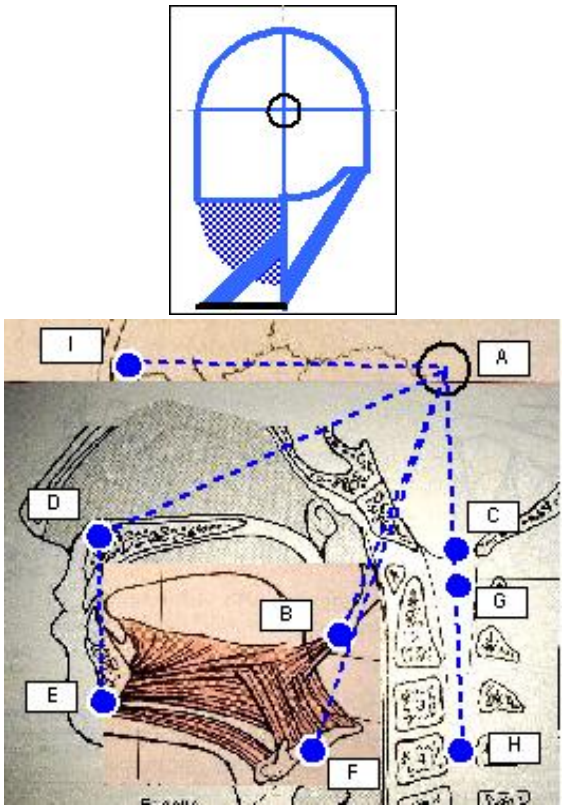
After understanding the Whole body implementation of CCE, to better understand the importance (image on the left) of this apparently unmotivated muscle spasm, the local Cranio-Cervical relationship is below isolated and specifically concerned.

Physiologic head position (PHP) model

Cervical spine forward inclination model

Cervical spine forward inclination + Atloanto-Occipital Joint (AOJ) extension = Cranio-Cervical Extension (CCE) model



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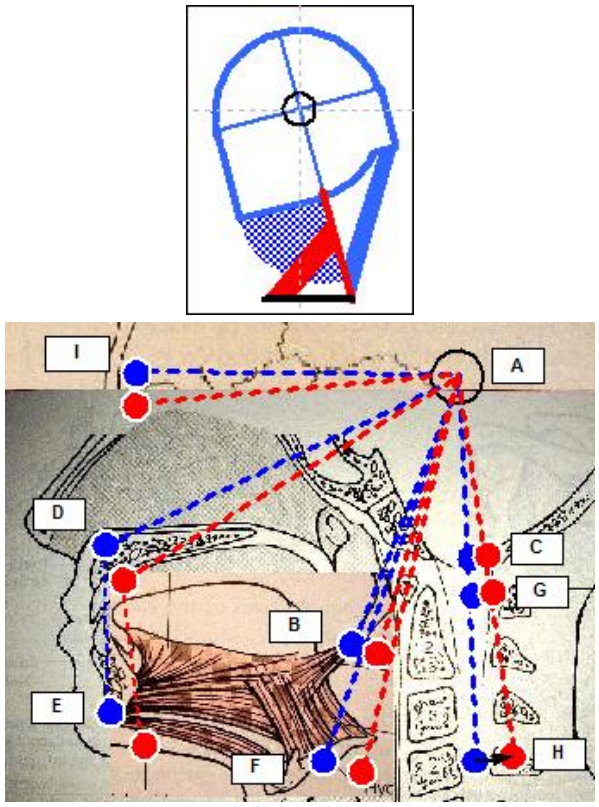

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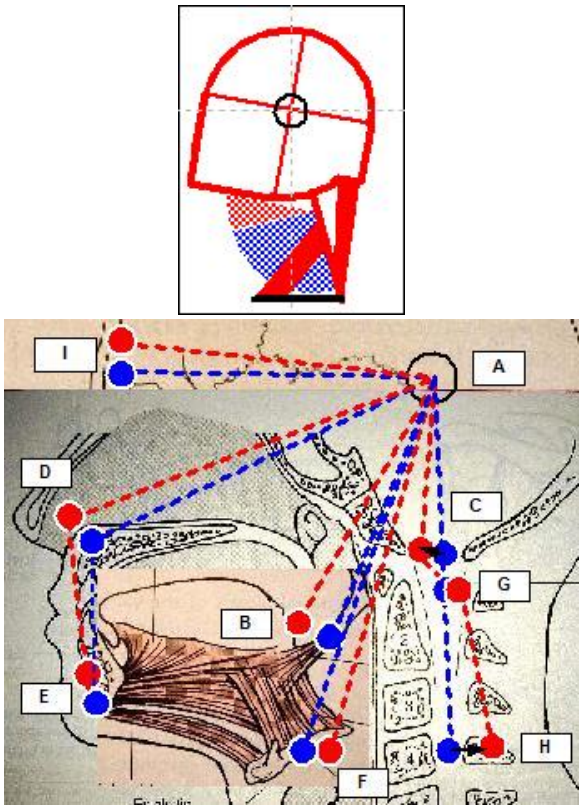

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Legend:
Blu: physiologic head position, ***Red***: Cranio-Cervical Extension (CCE), ***Black***: Cranio-Cervical Extension (CCE) motions, *Spots*: head position markers, *dashed lines*: cephalometric segments, *markers*: **A** (cranial center of rotation), **B** (styloid process centre of force), **C** (occipital spinal chord axis), **D** (maxillary marker), **E** (chin marker), **F** (hyoid centre of force), **G** (atlas spinal cord axis), **H** (4^{th} vertebra spinal cord axis), **I** (ocular bulb).

Physiologic Head Position (PHP) model: positional references marked in blue.

Cervical spine forward inclination model: cranial displacement (red markers) resulting from cervical spine forward inclination which is here represented as fourth vertebra backward motion (**HH black arrow**) instead of cranial forward motion.

Cranio-Cervical Extension (CCE) model: cranial displacement (red markers) resulting from the complex adaptation induced by Cranio-Cervical Extension (CCE) [6]:

- cervical spine forward inclination (**HH black arrow**);
- Atlanto-Occipital Joint (AOJ) extension (**CC black arrow**);
- **FF** hyoid backward motion is secondary to fourth vertebra backward motion;
- **CG** is the PHP AOJ relationship whereas **CG** represent the tortuous spine chord.

Functional impact: every postural adaptation takes place to promote functionality whenever it becomes undeliverable by physiologic motor control, every compensation induces homeostasis modification and compromises other functional districts, understanding what's improved and what's worsen helps understanding the goal a postural adaptation:

- **Sight**: under eyes point of view, Cranio-Cervical Extension (CCE) restores a sight angle (**AI**) that is upward inclined [6] (see also vagal reset), confirming that sight is not a priority to Cranio-Cervical Extension (CCE);
- **Mastication**: under occlusion point of view, whether the subject is mandible retruded or protruded or normocclusive, Cranio-Cervical Extension (CCE) doesn't seem to promote mastication, suggesting that mastication is not a priority to Cranio-Cervical Extension (CCE);
- **Respiration**: under tongue point of view, Cranio-Cervical Extension (CCE) features pharynx wall backward motion, mandible forward motion, styloid process up-forward motion and hyoid backward motion, in other words, *upper airway patency improvement*, as such, prospecting respiration as possible Cranio-Cervical Extension (CCE) priority;

<table>
<tr><th colspan="2">PATHOMECHANICS: Tongue Motor Insufficiency (TMI) (first primary)</th></tr>
<tr><td colspan="2">The term Tongue Motor Insufficiency (TMI) scientifically describes unilateral tongue weakness.
Head Forwarding Postural Strategy (HFPS) has previously been claimed to be the actuator of unilateral Cranio-Cervical Extension (CCE) which is hereby suspected to compensate highly disabling functional limitations introduced by Tongue Motor Insufficiency (TMI), hence identifying in Tongue Motor Insufficiency (TMI) the first primary pathology.
Universally agreed tongue muscular functionality are airway patency, swallowing, phonation and mastication, whereas, although less agreed, head flexion and maxillary arch expansion are considered primary tongue functions as well. Airway patency, head flexion and maxillary arch expansion are pointed to be Head Forwarding Postural Strategy (HFPS) primary pathomechanics, following, the elements supporting it (Ptd01, Ptd02, Ptd03) and the element excluding the alternatives (Ptd04, Ptd05).</td></tr>
<tr><th>#</th><th>Description</th></tr>
<tr><td>Ptd01</td><td>Upper airway patency mechanics

Cranio-Cervical Extension (CCE) induced by this mechanics is defined Active Unilateral Cranio-Cervical Extension (CCE) (AUCCE):

• Active: Tongue Motor Insufficiency (TMI) provokes an effort to gain Cranio-Cervical Extension (CCE) even when against gravity,
• Unilateral: Cranio-Cervical Extension (CCE) is induced only on the Tongue Motor Insufficiency (TMI) affected side.

AUCCE mechanics

Extrinsic tongue muscles (genioglossus (GG), hyoglossus (HG) and styloglossus (SG)) are responsible for pharyngeal airway patency [1, 2, 3, 4, 6, 8] as their co-contraction optimises tongue shape and position to reduce airflow resistance, this goal is achieved by rhythmic co-contractions [3, 4] reflex, synchronized with the respiratory muscles, and triggered by the same chemo receptors eliciting the respiratory reflex.

Tongue Motor Insufficiency (TMI) induces weak tongue muscle contraction, thus, poor pharyngeal airway patency [18]; this increased airflow resistance may be further enhanced by:

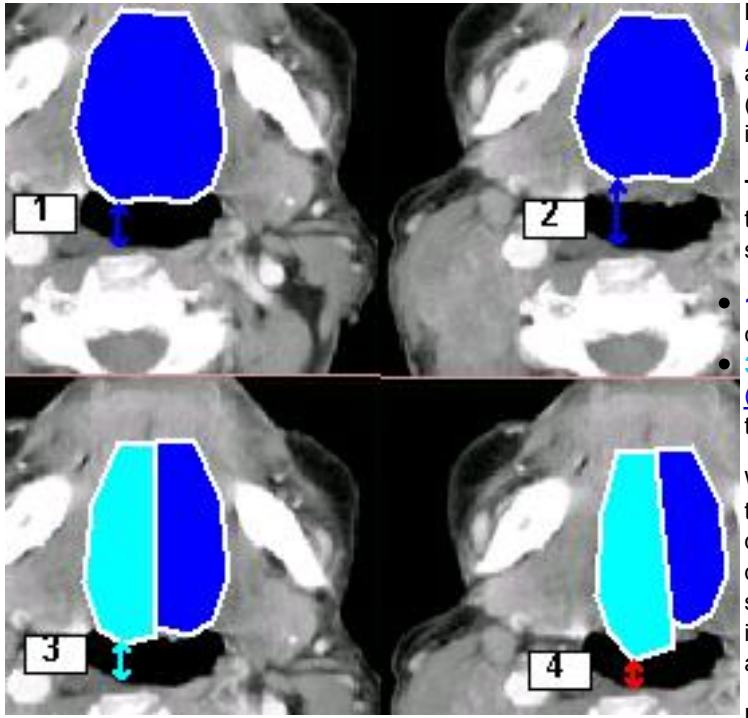
• head position [14, 15]: in the supine position, gravity requires an over effort for weak GG muscle to sustain the tongue weight in addition to provide airway patency, resulting in further limitation due to airway increased collapsibility;
• oxygen demand: in panting breathing during physical efforts, airway cross sectional area requirement is higher then in normal airflow demand, resulting in further limited airflow capability.

Cranio-Cervical Extension (CCE) role in upper airway patency improvement (as to say airflow resistance reduction) is widely known [16] and accepted (employed even in first aid procedures), hence, is not by chance that human physiology has developed air resistance vagal receptors which are there to elicit a permanent pharyngeal reflex</td></tr>
</table>

to improve airway patency as soon as an airflow resistance is detected, engaging Head Forwarding Postural Strategy (HFPS) to implement Cranio-Cervical Extension (CCE).

This hypothesis highlights an **Upper Airway Patency Maintenance Reflex (UAPMR)** elicited by unilateral upper airway patency loss owed to Tongue Motor Insufficiency (TMI) and triggering itself the AUCCE spasm featured in Head Forwarding Postural Strategy (HFPS) fully justifying Head Forwarding Postural Strategy (HFPS) presence in the prone and supine positions, moreover, the contralateral physiologic tongue contraction is suspected to further limit airflow by moving the tongue according to the following model:

Tongue dislocation model



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Legend:
Blu tongue: physiologic tongue contraction, ***Cian tongue***: weak tongue contraction, *Arrow*: airway patency indicator, *imaging*: **1** (tongue motor physiology (TMP) + basal airflow demand), **2** (TMP + augmented airflow demand), **3** and **4** (same as **1**, **2** but Tongue Motor Insufficiency (TMI) instead of TMP).

Tongue dislocation model: the model describes the suspected increased airflow resistance due to increased oxygen demand, the background MRI image is a general (not patient's image) cranial section just above the mandibular arch:

- **1 blue arrow / 2 blue arrow** ratio is the airflow resistance variation by TMP bilateral co-contraction inducing bilateral protrusion and depression,
- **3 cyan arrow / 4 red arrow** ratio is the airflow resistance variation by Tongue Motor Insufficiency (TMI) muscular co-contraction inducing unilateral protrusion and depression along with dislocation toward the unaffected side.

With Tongue Motor Insufficiency (TMI), a unilateral tongue contraction, is neither able to provide the required airway patency with basal respiration (**3** vs **1**) nor in case of augmented airflow demand (**4** vs **2**), moreover, unilateral extrinsic muscles contraction induces tongue displacement decreasing the distance between tongue and posterior pharynx wall (**red arrow**), thus, further stimulating air resistance pharyngeal receptors of the vagus nerve, enforcing the afferent signals inducing a stronger Upper Airway Patency Maintenance Reflex (UAPMR), augmenting AUCCE and worsening Head Forwarding Postural Strategy (HFPS).

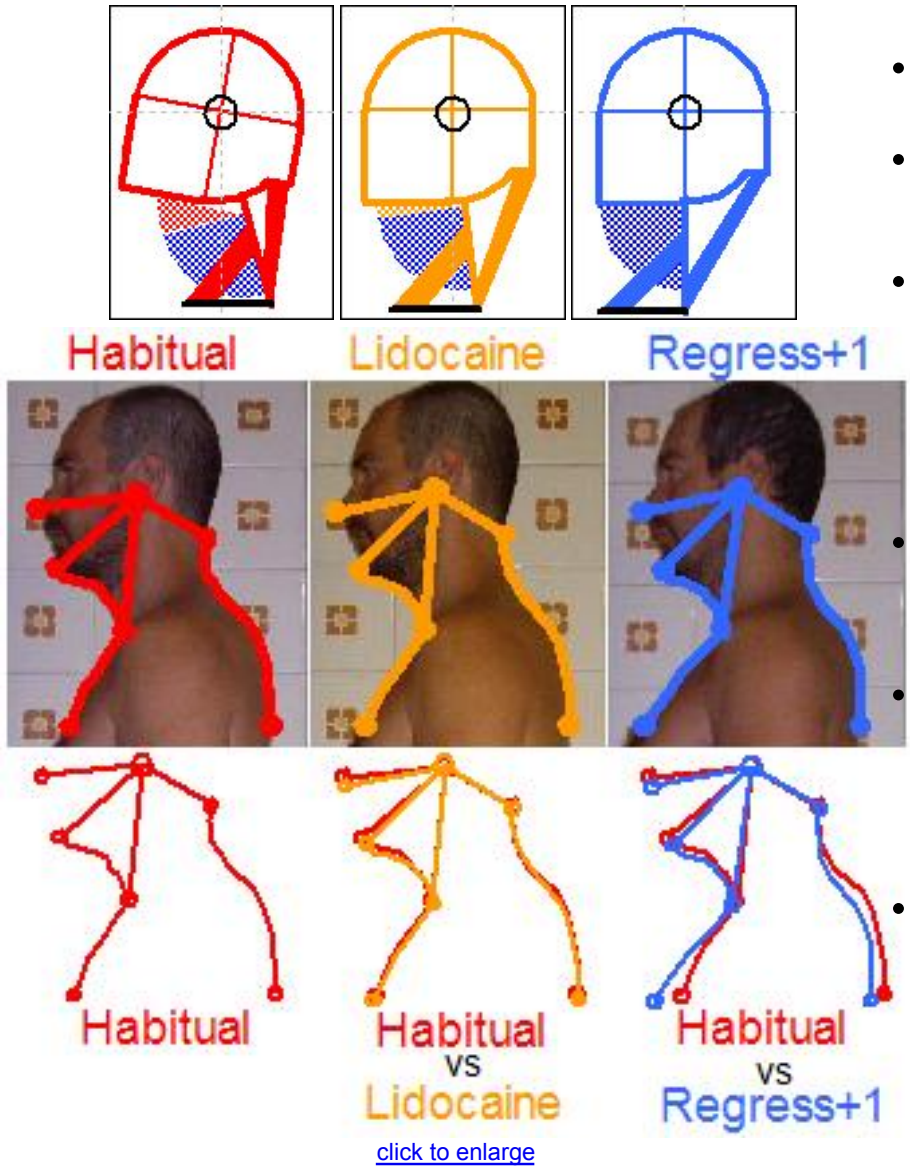
Please note that extrinsic tongue muscles not only provide tongue intraoral volume regulation (Genioglossus) and vertical elevation (Styloglossus and Hyoglossus), but also cuncur in giving shape to pharynx as Styloglossus lies behind pharynx mucosa and superior pharyngeal constrictor muscle, as such, unilateral tongue weakness may either increase emitongue volume, displace tongue within pharynx and give asymmetric shape to oro-pharyinx, resulting in pharyngeal airflow irregular and asymmetric resistance.

Upper Airway Patency Maintenance Reflex (UAPMR) is implemented by a firing pattern that mimics other well known vagal ejective reflexes: the cough reflex and the gag reflex. It is not by chance that these reflexes share the same base firing pattern, given Cranio-Cervical Extension (CCE) is the key condition to provide upper airway patency to maximise either ejection and intake. Since Upper Airway Patency Maintenance Reflex (UAPMR) goal is not to maximize emergency peak ejection but to maximise regular intake, it is implemented as a light Cranio-Cervical Extension (CCE) chronic spasm instead of an abrupt acute Cranio-Cervical Extension (CCE) spasm with ejective feature.

Evidence of the existence of Upper Airway Patency Maintenance Reflex (UAPMR) and of its complex firing pattern is provided by the postural reaction of a Head Forwarding Postural Strategy (HFPS) patient to pharyngeal vagal receptors reset done by pharyngeal local appliance of lidocaine 2%, as shown below.

Pharyngeal vagal receptors reset

This experiment describes cranio-cervical-thorax relationship in normal (Habitual) Head Forwarding Postural Strategy (HFPS) context and under a "faked" (Lidocaine) and a "true" (Regress+1) context of Head Forwarding Postural Strategy (HFPS) regress.



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Contexts:

- **Habitual**:ordinary posture, this context is reproducible, Cranio-Cervical Extension (CCE) degree is constant, duration, except regression event, is chronic,
- **Lidocaine**: posture under the effect of lidocaine 2%, this context has demonstrated to be reproducible, Cranio-Cervical Extension (CCE) regress is light but systematic, duration is matter of minutes, after that, Habitual context is restored,
- **Regress+1**: posture under the effect of an index 1 regression event, this context is not yet reproducible, this particular event has been triggered by atlas manipulation technique, which appliance has unfortunately revealed not to provide reproducible effects, Cranio-Cervical Extension (CCE) regress is similar to other index 1 regression event, duration has been 1 day, after that, Habitual context is restored.

Context transitions:

- **Habitual vs Lidocaine:** immediate postural reorganization after abrupt and short term pathologic stimulus interruption, evidence of chin-ear-sterno angle reduction is visually confirmed, Head Forwarding Postural Strategy (HFPS) index 0 regression event has been reported by the patient. This is a "faked" context as there is no Tongue Motor Insufficiency (TMI) improvement.
- **Habitual vs Regress+1:** longer term adaptation after 1 day absence of pathologic stimulus, evidence of chin-ear-sterno angle reduction, cervical spine forward inclination decrease, increased chest expansion and decreased shoulder girdle forwarding are visually confirmed, Head Forwarding Postural Strategy (HFPS) index 1 regression event has been reported by the patient. This is a "true" context as the patient reports Tongue Motor Insufficiency (TMI) improvement as well.
- **Lidocaine vs Regress+1:** significant analysis, same cranial-horizon inclination, clue of scalenus contraction release in regress+1 context.

Further evidence: in Lidocaine as well as Regress+1 context the patient is 1,5 cm taller than Habitual context.

Pharyngeal vagal receptor reset procedure has already been successfully employed [21, 22, 23] in demonstrating the relationship between upper airway receptors and airflow resistance but has never concerned any motor reaction (eg. postural) other than upper airway dimension variation, whereas, the author hereby indeed does.

It is author's opinion that airflow resistance increase after vagal receptor reset, occurs as main consequence of the subsidence of the Cranio-Cervical Extension (CCE) spasm in ongoing Head Forwarding Postural Strategy (HFPS) subject, as such, it should NOT occur (or should be much lighter) in healthy posture subjects who fully entrust airway patency maintenance to tongue physiologic rhythmic respiratory contractions [3, 4].

Although apparently denying literature conclusions, the author's claim is not contradictable by existing studies as none of them has been carried out with adequate protocol: "healthy" control subjects have not been sampled according to Head Forwarding Postural Strategy (HFPS) features. According to the undeniably high incidence of Head Forwarding Postural Strategy (HFPS) clues in the population and Head Forwarding Postural Strategy (HFPS) hereby claimed relationship with upper airway patency (TMI), healthy control subjects, without explicit specification, are likely to be HFPS ongoing subjects, as such, the result of these studies on healthy posture subjects is unpredictable and requires further research to be described.

Bibliographic support is provided by cross-correlation analysis of the following interdisciplinary literature:

- obstructive sleep apnoea (OSA) studies disclose augmented pharyngeal collapsibility (snoring) during REM sleep as well as recursive presence of Cranio-Cervical Extension (CCE) in OSA patients [24, 6],
- neurological bibliography describe postural muscle tone drop during REM sleep [25],
- otorhinolaryngology focus on airflow resistance increase after vagal receptor reset [21, 22, 23],
- present study describes Head Forwarding Postural Strategy (HFPS) subsidence after vagal receptor reset,

The above findings are relatable by the hypothesis of Upper Airway Patency Maintenance Reflex (UAPMR) triggering Head Forwarding Postural Strategy (HFPS) to implement AUCCE in response to increased airflow resistance introduced by Tongue Motor Insufficiency (TMI) (hence, preventing snoring during non REM sleep), confirming the consequences of Head Forwarding Postural Strategy (HFPS) loss on airflow (snoring when Cranio-Cervical Extension (CCE) muscular tone drops during REM sleep).

Further supporting evidences:

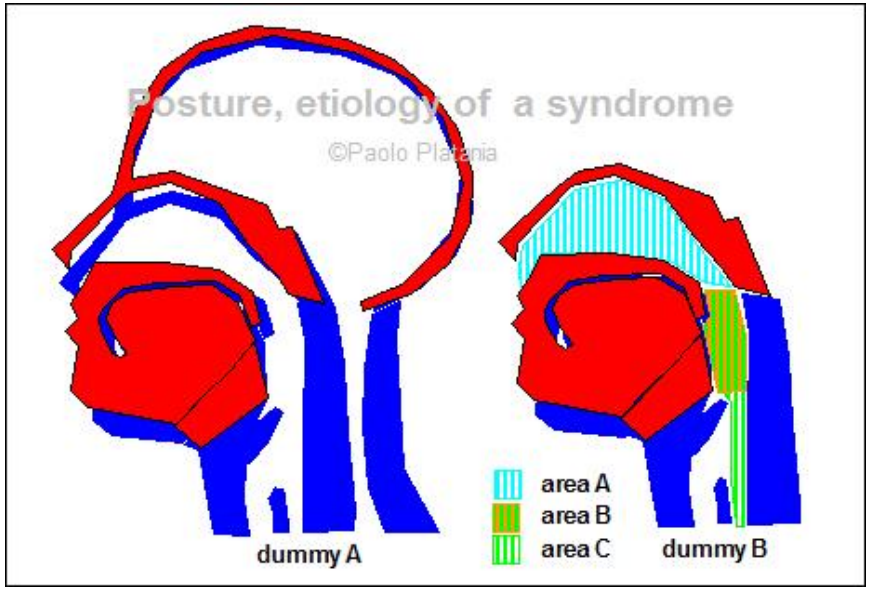
- From self evaluation: Head Forwarding Postural Strategy (HFPS) is present in the prone as well as the supine position (not interested by cranial weight bearing unbalance), evidence is provided by anterior scalenus (AS) and tensor fascia lata (TFL) pathologic contraction undeniably present at night before sleep and since morning arousal confirming active cervical spine forward inclination, furthermore, left sub occipital muscles are overactive in the prone and supine positions providing additional Atlanto-Occipital Joint (AOJ) extension;
- From Self evaluation: oxygen demand dependence of some unilateral motor unbalance (Ese05, Ese07) supports airway adaptation insufficiency;
- From Regression events: oxygen demand dependence of some unilateral symptoms (Ere02) supports airway adaptation insufficiency.

Airflow asymmetric afferences localization

Legend:
Blu: Upper airway in physiologic head position,
Red : Upper airway in Cranio-Cervical Extension (CCE),
dummy A:comparative PHP/CCE upper airway,
dummy B: Upper airway areas classification according to airflow receptor features,
cyan: Receptors sensitive to only-nasal airflow,
orange: Receptors sensitive to CCE airway widening and to tongue weakness airway narrowing,
green: Receptors sensitive to vagal reset,
area A: nasopharynx (only **cyan** receptors),
area B: oropharynx (**orange** and **green** receptors),
area C: laryngopharynx (only **green** receptors).

The proposed pharingeal areas classification provides a helpful model to reliably identify areas that do not detect asymmetric airflow or, to be more precise, that do not elicit Upper Airway Patency Maintenance Reflex (UAPMR). The following evidences suggests that the **asymmetric airflow afferent signal is generated by receptor in the oropharynx:**

- Only-mouth breathing (with held nose) does not improve Cranio-Cervical Extension (CCE) confirming that Upper Airway Patency Maintenance Reflex (UAPMR) is constantly elicited, even in the absence of nose airflow afferences and, therefore, excludes nasopharynx;
- Although laryngopharynx is sensitive to vagal reset it is unlikely responsible of the improvement of CCE induced by it. If the hypothesis that tongue weakness induces an airflow window reduction is true, then only the receptors detecting this asymmetric restriction are capable of elicting asymmetric afferentces (hence, only oropharynx), moreover, even if laryngopharynx would be capable of detecting airway restriction it would be senseless to elicit a CCE reflex to widen another area (the oropharynx). This analysis suggests to exlude laryngopharynx;
- Oropharynx is subject to either mouth and nose airflow, is exposed to airway narrowing in case of tongue weakness, benefits from CCE airflow window widening and is sensitive to vagal reset, hence, oropharynx receptors are eligible as the most likely to produce asymmetric afferent signal that triggers Upper Airway Patency Maintenance Reflex (UAPMR).



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All these findings allow to state that GG, HG and SG actively participate in respiration and a that Tongue Motor Insufficiency (TMI) might result in respiration insufficiency and consequent permanent Upper Airway Patency Maintenance Reflex (UAPMR) to induce unilateral muscular spasm (Head Forwarding Postural Strategy (HFPS)).

Ptd02

Head flexion mechanics

Cranio-Cervical Extension (CCE) induced by this mechanics is defined **Passive Unilateral** Cranio-Cervical Extension (CCE) (PUCCE):

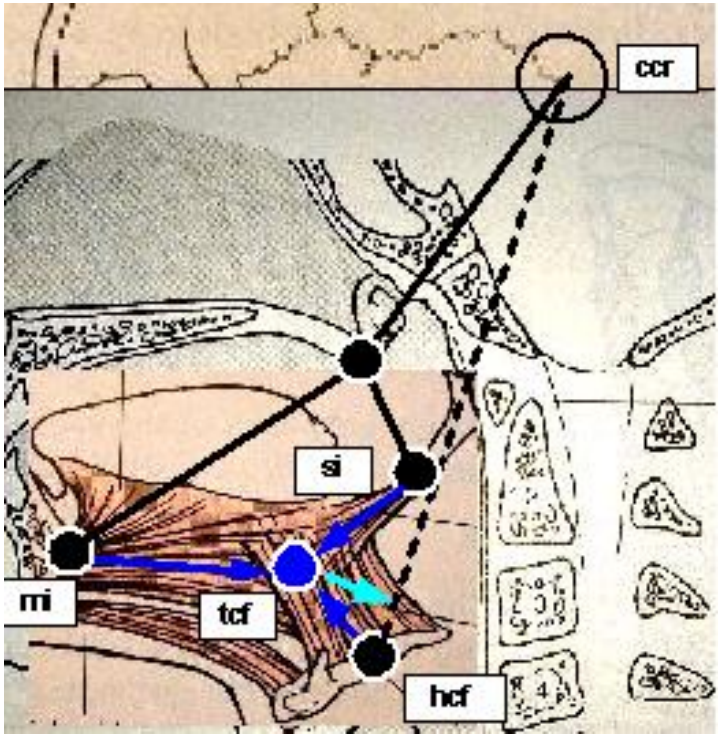
- **Passive:**Tongue Motor Insufficiency (TMI) requires gravity (upright position) to induce cranial extensors-flexors unbalance,
- **Unilateral:** Cranio-Cervical Extension (CCE) is induced only on the patient left side.

PUCCE mechanics

Tongue muscles pulling on the styloid process and on the mandible (behind chin) feature a cranial flexion moment arm and, given the continuous and strong tonic contraction for pharyngeal airways patency [8], exert direct hyoid-cranial flexion force. These literature overlooked features is hardly escaped to physiology which may rely on GG, HG, SG, stylohyoid (SH) and infrahyoid muscular chain for AOJ flexion, undeniable healthy posture indicator. The following models provide explanation of the theory.

Physiologic Head Posture (PHP) model

Physiologic Head Posture (PHP) vs Cranio-Cervical Extension (CCE) model



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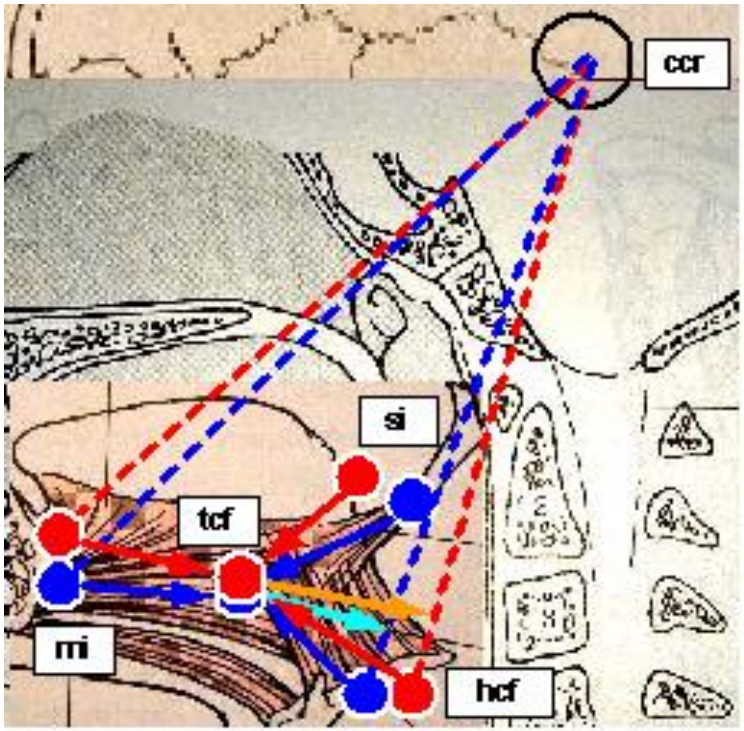

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Legend:
Blu: PHP, ***Red***: Cranio-Cervical Extension (CCE), ***Black***: Fixed relationship, ***Cian***: PHP cranial flexion moment arm, ***Orange***: Cranio-Cervical Extension (CCE) cranial flexion moment arm, *Spots*: head position markers, *dashed lines*: cephalometric segments, *solid arrows*: muscular forces and resultant vectors (color), bony forces (black), *markers*: **ccr** (cranial centre of rotation), **si** (styloid insertion), **mi** (mandible insertion), **hcf** (hyoid centre of force), **tcf** (tongue centre of force).

Conventions:

- temporomandibular joint (TMJ) is considered "locked" (teeth clenching) resulting in non deformable **mi-ccr, mi-si, si-ccr** relationship,
- cranial centre of rotation (**ccr**) is simplified to be a single point, thus, considering occipital condyles to move on a round atlas track concentric with **ccr**,
- cranial centre of rotation (**ccr**) is the fixed point of observation.

Physiologic Head Position (PHP) model

demonstrates the Atlanto Occipital Joint (AOJ) flexing moment arm (**Cian arrow**) exerted by co-contraction of extrinsic tongue muscles during tongue activity, moreover it highlights the fact that whatever tongue muscle contraction induces a cranial flexion, the model assumes the following:

- **hcf** is a fixed pulley system,
- **tcf** represent the resultant point of application of extrinsic tongue muscles force exerted by co-contraction of GG (**mi-tcf**), HG (**hcf-tcf**) and SG (**si-tcf**).

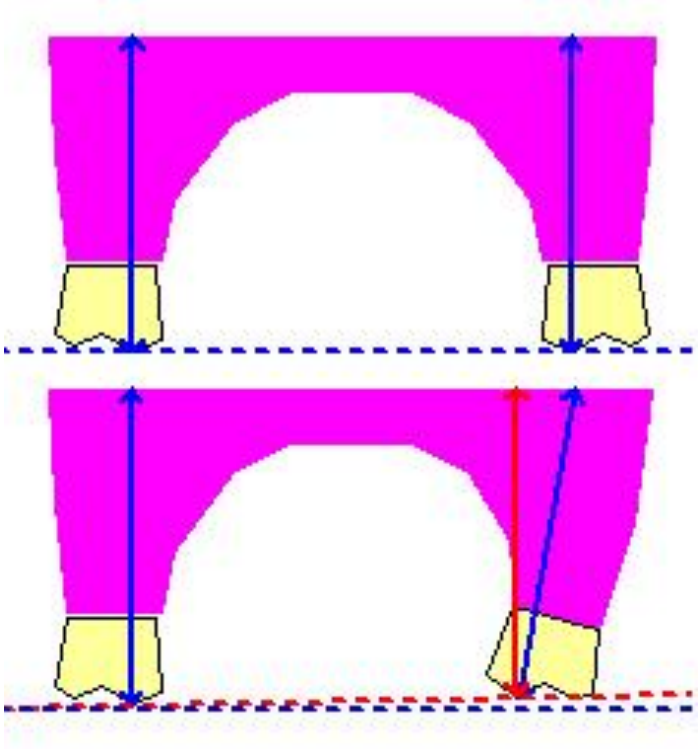
Physiologic Head Posture (PHP) vs Cranio-Cervical Extension (CCE) model

displays a good attitude of the tongue to induce AOJ flexion in the PHP (**Cian arrow**) whereas, with increasing Cranio-Cervical Extension (CCE), GG-HG-SG force (**tcf**) produces a growing cranial flexion moment arm (**orange arrow**) to facilitate tongue muscles restoring the AOJ angle, thus, in case of Tongue Motor Insufficiency (TMI), Atlanto Occipital Joint (AOJ) extension may not be corrected by GG-HG-SG and has to be compensated by forward inclination of the cervical spine generating Cranio-Cervical Extension (CCE) hence Head Forwarding Postural Strategy (HFPS).

PATHOMECHANICS: Dental Conflict (DC) (second primary)	
Mandible Retruding Postural Strategy (MRPS) is been supposed to be the actuator of bilateral Cranio-Cervical Extension (CCE), the reasonable relationship between Dental Conflict (DC) and TMJ disorder (mandible retrusion) lead to speculate that Dental Conflict (DC) is the second primary pathomechanics. Following, the elements supporting this hypothesis (Pdc01) and the element excluding the alternatives (Pdc02).	
#	**Description**
Pdc01	**Dental Conflict (DC) mechanics** Cranio-Cervical Extension (CCE) induced by this mechanics is defined **Structural Bilateral** Cranio-Cervical Extension (CCE) (SBCCE): • **Structural**: Dental Conflict (DC) is a consequence of a structural (maxillary arch) disorder, • **Bilateral**: Cranio-Cervical Extension (CCE) is induced symmetrically. **SBCCE mechanics** Dental Conflict (DC) mechanics over TMJ disorder eliciting condition is as follows: neurological afferences (trigeminal nerve) produced by pathologic teeth contacts generate inhibitory reflexes directed to the responsible muscles aimed to avoiding every type of harmful motion, these reflexes successfully grant teeth safety but on the other side generate pathologic torques on mandible provoking the TMJ disorder. TMJ disorders are widely suspected to induce, among several other symptoms, postural unbalances. Although Mandible Retruding Postural Strategy (MRPS) seems reasonably related to TMJ disorder, the pathomechanics of its impairing capabilities over posture is unclear. Although several symptoms are ascribable to TMJ disorder (condile dislocation, mastication disorder and temporal muscles painful contractures), postural disorder doesn't appear to be mediated by these conditions, the author in spite proposes the following hypothesis to justify Cranio-Cervical relationship modification (CCE) consequent to TMJ disorder: • **Vagal mediated**: Cranio-Cervical Extension (CCE) compensates airway patency loss (tongue backward motion) due to mandible retrusion; • **Neck internal proprioception mediated**: Cranio-Cervical Extension (CCE) is induced by internal neck proprioceptive afferences triggered by organs (arteries, nerves, glands) compressed by the retruded mandible; • **Trigeminal mediated**: Cranio-Cervical Extension (CCE) is meant to optimise Cranio-Cervical-Mandibular relationship, in response to pathologic mandible retrusion, to grant functionality (mastication, swallowing); Physiologic maxillary and mandibular arches get together (centric occlusion) when molar and pre molar teeth on one arch contact the opposite molar and pre molar teeth on the opposite arch in normocclusion TMJ position. Normocclusion TMJ is not functional to the patient's bite as it would cause upper and lower incisors to contact (due to upper incisors crowding, Ecl19) preventing molar and pre molar contact. Molar occlusion, in context of incisor teeth crowding, is achievable with mandible retrusion.
Pdc02	*Why not other TMJ displacements at Mandible Retruding Postural Strategy (MRPS) origin ?* Mandible retrusion is not the only TMJ displacement, nevertheless is considered the main responsible for Mandible Retruding Postural Strategy (MRPS) for the following reasons: • left condile further backward motion is suspected (Esy11) but is thought to be derived by unilateral Cranio-Cervical Extension (CCE) component, thus, the consequent postural disorders pertain to Head Forwarding Postural Strategy (HFPS) • slight unilateral lack of vertical dimension is present (also kinesiologically diagnosed) as consequence of maxillary alveolar process asymmetric development (Ecl22,Eot05) but its extent is supposed to be negligible (better say tolerable) according to quite symmetric facial mimic muscles, reliable TMJ displacement

indicators.

Lack of vertical dimension



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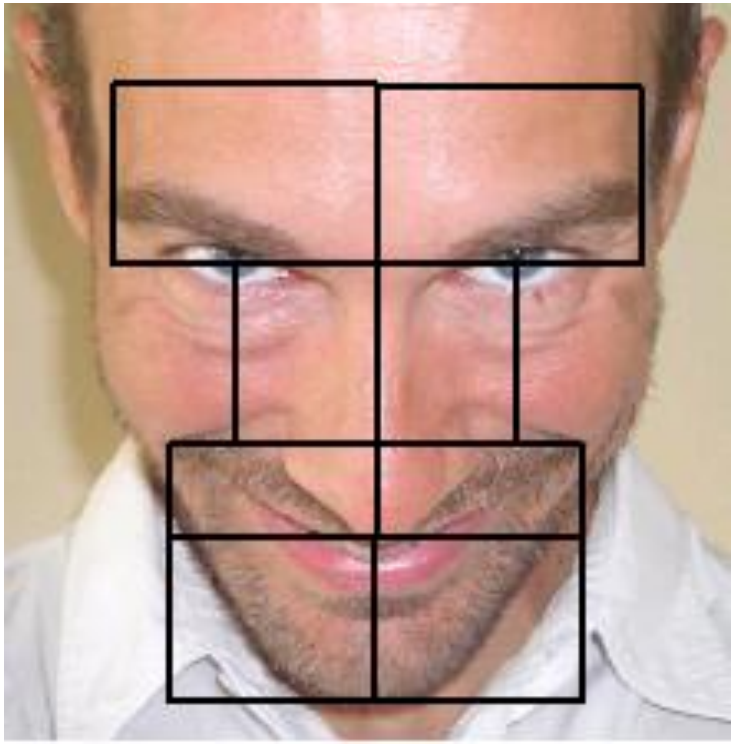
Facial muscles symmetry



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