

## Occlusion and Gait: A clinical observation

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The concept of gait is repeatedly explored in PRI and many documents outlining the influence of at least two of the three main polyarticular chains of muscle on gait have been produced as a result (1). These bilateral chains of muscle directly affect body posture and, because of inherent human asymmetry, will become dominant on one side over another. When this dominance occurs, compensation begins as the human body attempts to shift the center of mass from the right side of the body to the left. The AIC (Anterior Interior Chain) and the BC (Brachial Chain) end or begin at the diaphragm. The diaphragm's position of extension on the left and flexion on the right is a result of human asymmetry and L AIC (Left Anterior Interior Chain) and R BC (Right Brachial Chain) dominance is an inevitable outcome of this asymmetry. With increased R BC dominance, there is a tendency to compensate for apparent insufficiency of expansion of the R thoracic cavity during inhalation and likewise contraction of the L thoracic cavity during exhalation. The purpose of this document is to explore the effect upon gait, of the R BC and R TMCC (Right Temporal Mandibular Cervical Chain) and how gait, or compensation therein, occurs in the cervical spine, cranium, maxilla and mandible.

One portion of the BC is comprised of the two hemi-diaphragms (left and right) and the subsequent "other end" of the BC that we will explore is the scalenes, levator scapula and SCM (Sternocleidomastoid). In the R BC pattern of movement we understand that the activity of the two hemi-diaphragms is non-symmetrical, with the right hemi-diaphragm positioned more in a state of exhalation maintaining a more adequately formed Zone Of Apposition (ZOA) and the left more in inhalation with a subsequent loss of ZOA. If diaphragmatic position does not alternate efficiently from side to side then activity of the scalenes, levator scapula and SCM will likewise be non-symmetrical. With orientation of the thoracic spine to the right as a result of asymmetric diaphragmatic function, the human thorax must initiate rotation back to the left. The result of thoracic rotation to the left is spinal rotation to the left along with leftward lateral flexion of the mid-thoracic spine as well. The phenotypic representation of left spinal rotation appears to be right thoracic abduction and depression of the right ribcage when compared to the left.

Depression of the right ribcage can, and frequently does, limit efficiency of ipsilateral inhalation. When inhalation is restricted on the right side then there is usually a concomitant increase in accessory muscle activity to increase apical expansion on that same side. When these accessory muscles are engaged, there can be a resulting increase in TMCC activity.

Rothbart (2) explores vertical facial dimensions as they are related to abnormal foot position and concludes that foot pronation results in ipsilateral loss of vertical facial dimension. One can hypothesize that a portion of this asymmetric facial presentation is linked to, but not limited to, non-alternating scalene, levator scapula and SCM activity and the subsequent asymmetric activation of the TMCC.

As the scalenes on the right increase activity to increase right apical expansion in the R BC pattern, then right lateral flexion of the lower C-spine can occur. Likewise, there can be some ipsilateral orientation of the C-spine as a result of this lateral flexion. As the right scalene activity remains continuous (as a result of increased right lateralization and subsequent accessory respiratory function), an ongoing and unrelenting counter-rotation of the C-spine must occur despite alternation of the center of mass through the lower extremities. Right lateral flexion of the C-spine may require ipsilateral inhibition of right longus capitis while leftward rotation of the C-spine requires inhibition of the right levator scapula.

The levator scapula is the second of the three R BC “muscles-of-interest” that function non-symmetrically during increased effort to respire or fill the R chest cavity with air. However, the R levator scapula is not necessarily functioning as an accessory respiratory muscle on that R side nor is it increasing in activity. It is, in fact, long and inhibited as a result of ipsilateral scalene activity and lack of R apical expansion. In the L AIC/R BC pattern the thorax will demonstrate a right-sided posterior rib hump as the anterior ribs move into internal rotation. The position of the posterior ribs on the right can result in a malpositioned scapula with the scapula positioned in internal rotation (IR) about a vertical axis on the right side. The end result of scapular IR is a natural “untwisting” of the levator scapula as it lengthens distally at the scapula and proximally at the neck as the neck rotates to the left. Leftward rotation of the C-spine is the result of compensation from the right lateral flexion and orientation of the C-spine described in the previous paragraph. The left levator scapula can therefore be positioned in a more mechanically advantaged state and aid in leftward rotation and leftward lateral flexion of the C-spine as a result of external rotation of the left scapula about a vertical axis. The “untwisted” levator scapula on the right side then allows the right SCM to become a primary left rotator of the C-spine.

Increased activity of the right SCM is heightened as the SCM is also acting to increase right apical expansion. The right SCM functions efficiently to rotate the C-spine to the left via the temporal and occiput bones, as the right levator scapulae continues to inhibit and the left continues to activate. Continuous and unrelenting activity of the right SCM without a left ZOA results in morphological changes in the cranium as the right SCM acts on its proximal attachment site: the mastoid process of the right temporal bone.

The neuro-respiratory drive to maintain a right ZOA limits the ability to alternate the center of mass from one side of the body to the other. As non-alternating gait continues we can potentially see three significant issues occur: increased right-sided Ground Reaction Force (GRF) compared to left, excessive flexion of the right mid-cervical facets or bilateral mid-cervical spine and loss of normal cervical osteokinematics:

1. Effects of Ground Reaction Force at the C-spine: One result of GRF is upward movement of the Atlas (C-1) against the occiput (AO movement). AO

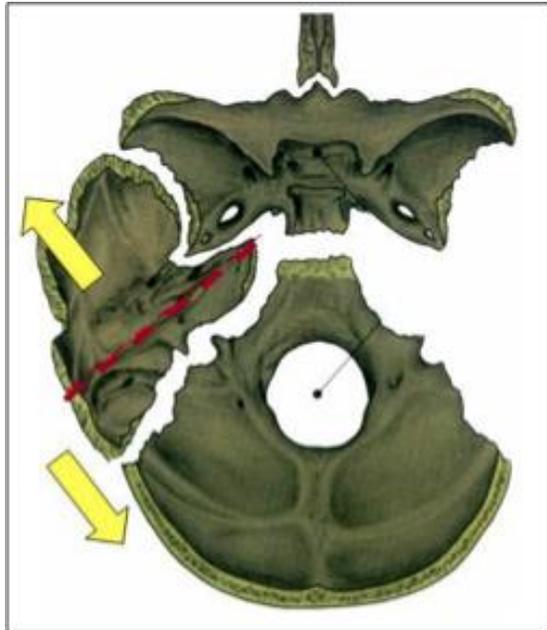
- movement is heightened through neurological activation of right rectus capitis anterior and lateralis, which are both activated on the right in order to maintain gaze and body position during upright activities as well as for completion of left lateral flexion of the C-spine. GRF is heightened by an inability to unload the right lower extremity during upright, bipedal gait and the subsequent need for balance through maintenance of center of mass over the right lower extremity (3, 11,12, 13).
2. Excessive flexion of the C-spine occurs as a result of increased activity of the muscles highlighted above plus a host of others including but not limited to: longus colli, inferior and superior hyoid muscles, masseters and temporalis (4,5,6)
  3. Normal cervical arthro and osteokinematics are altered due to the loss of lordosis in the C-spine. Normal mechanics result in cervical vertebral bodies rotating ipsilaterally with the direction of mid-cervical lateral flexion. This may be compromised as a result of muscle activity and/or loss of normal lordotic position or ability or both. (7)

Osteokinematics of the OA and AO joint in the R TMCC pattern result in an increased need to laterally flex the right occiput on the Atlas during rotation of left O on A. This can lead to pathologic OA osteokinetic positioning and arthrokinematic patterns (7). Unilateral, non-alternating GRF and OA kinematics may produce right-sided OA extension and left-sided OA flexion. Right OA extension can be produced from loss of normal cervical lordosis (A on O movement) or increased O on A movement with O moving into extension on the right side (8). Right OA or AO extension can create a right sphenobasilar synchondrosis (SBS) flexion state and a left SBS extension state (8). The strain at the SBS along with OA leftward rotation results in right sphenoid External Rotation (ER). Right Sphenoid Flexion and ER coupled with right lateral pterygoid activity (along with masseters and temporalis) can produce IR of the right temporal bone. At this point it is important to recall the activity of the right SCM, as it is also acting on the right temporal bone to create posterior rotation as well. Combined, the following actions contributes to a raised right wing of the sphenoid:

1. left mid-cervical lateral flexion
2. right flexion of the SBS
3. right sphenoidal ER
4. posterior rotation of the right temporal bone
5. anterior rotation of the left temporal bone
6. internal rotation of the right temporal bone
7. external rotation of the left temporal bone

While Strokon and James describe cranial strains from the standpoint of an occlusally driven phenomenon, we can also consider that strains may be from respiration and gait-driven phenomena (9,10). Certainly occlusion can alter mechanics of mastication, masticatory muscle activity, position of bones of the cranium and orientation of the neck. Additionally it can be extrapolated that

occlusal causations can continue into the ribcage, pelvis and lower extremity mechanics position and function. But, likewise, the reverse must also be considered; that gait and respiration may actually have an effect on occlusion (2). To this point the Postural Restoration Institute® has shown that the R TMCC pattern results in alteration of positions of the C-spine, occiput, sphenoid and temporal bones. As the occiput tips down on the left it carries the temporal bone with it (as yet another force to ER the temporal bone on the left side). The temporal bone rotates about an oblique axis from the external auditory meatus toward the direction of the sella turcica of the sphenoid see figure 1 (9).



*Image from Strokon and James Cranial Strains  
And Malocclusions V Part 1 IJO, 2006*

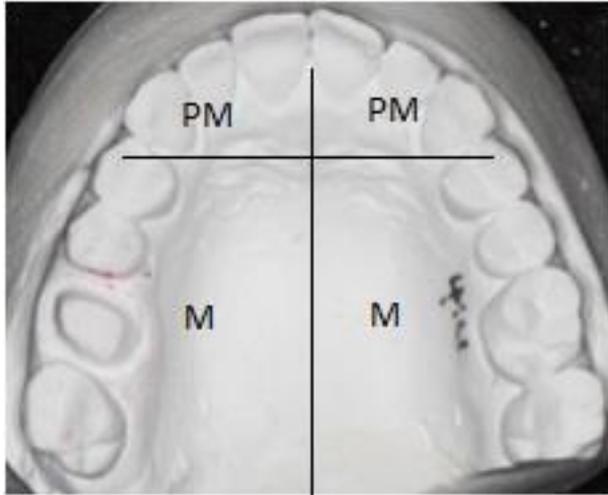
The angular axis rotates the glenoid fossa distally and upward and as the mandible follows, it is carried upward and thus the teeth in the maxilla are prevented from erupting (9). Since the maxilla is carried to the right with movement of the sphenoid, the left molars can be prevented from making solid, referencing contact. Additionally, clockwise rotation of the sphenoid results in a gear-like effect upon the junction between the pterygoid processes and the maxillo-palatine complex. Thus, as the maxilla is drawn upwards, its lateral structures are drawn toward midline resulting in Internal Rotation (IR) of the maxilla and a subsequent Angle Class II molar and cuspid relationship on the left side and Class I on the right (9). At this point it should be noted that ER of the L temporal bone is multi-factorial with several of the causes outlined below:

Etiology of ER of the L temporal bone:

1. Occipital down tipping on the left
2. Anterior rotation of the L temporal bone from:
  - a. IR of the L greater wing of the sphenoid

- b. L SBS extension
- c. L Sidebending of the L greater wing of the sphenoid
- d. Posterior rotation of the R temporal bone as induced by:
  1. Right SCM activity
  2. ER of the right greater wing of the sphenoid

A fascinating clinical observation has led me to believe that there is a consideration of not only the maxilla, but the pre-maxilla as well.



It becomes apparent that there is not only a tendency for the left maxilla to internally rotate but a need for premaxillary external rotation on the left as well. In short, a table can be created that shows positioning of the maxillae as follows:

Left Maxilla IR (Supination)	Right Maxilla ER (Pronation)
Left Premaxilla ER (Pronation)	Right Premaxilla IR (Supination)

The maxilla is actually divisible into four quadrants. As I have looked at the premaxillae and the teeth attached (primarily the canines or cuspids) I have seen a tendency for the left canine to follow the premaxilla. On the left, the lingual aspect of the tooth moves inferiorly and toward midline. Likewise, the labial aspect of the tooth has been noted to move away from midline. This is consistent with the concept of pronation. The left canine and premaxilla pronate, the left ribcage pronates and the left foot tends to pronate in the R TMCC, R BC, L AIC pattern. There is also a great deal of “contact” made between the upper and lower canines and cuspids on the right side premaxillary region and it can be hypothesized that this contact is likely since supination is occurring on the right pre-maxilla. Delivery and subsequent adjustment of MOOO-type appliances frequently requires reduction of two components in many instances. The first component is contact on the right side first molar which appears to be the end result of the previously described and detailed unrelenting GRF and can subsequently become a driving reinforcer of the GRF. The second is the contact of the right cuspids or lateral incisors that appears to

be the direct result of the supination of the right premaxilla. Splint adjustment must take these issues into account in order to minimize the potential “top-down” effect that can potentially limit a Postural Restoration®-based program designed to alter GRF and aid in achievement of a reciprocal and alternating palate.

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