ACTA SCIENTIFIC NEUROLOGY

Volume 2 Issue 6 June 2019

Case Study

# Consideration of Growth Profiles of Foetal Mid-Face in Neurological Treatment

**David Zimmerman***

*Department of Neurology, New Zealand*

***Corresponding Author:** David Zimmerman, Department of Neurology, New Zealand.

**Received:** March 26, 2019; **Published:** May 14, 2019

## Abstract

To show changes made by oral appliances (orthotics) that are related to both airway patency and to high levels of nociceptive signaling. These major metabolic and pain-related loads both create and maintain neurological illness.

There is now adequate peer reviewed knowledge that shows the impact of commercialisation of sugar, cigarettes and alcohol has on general health, growth and development [1].

These three items seem to be consistently involved in creating ill health. The mechanism of these intrusions is complex and dietary sugar is in complicit in this as demonstrated by Creuzet LeDouarin., *et al* [1]. The greatest influence of sugar in particular can arguably be during the early hours and days of embryonic development. Exposure to sugar can involve HOX genes to change the [2] growth pattern of the midface. This leads to impairment of the naso-oro-pharyngeal airway [2-5] which results in increased intermittent hypoxia due to easier collapsibility of the pharynx [6-25].

Intermittent hypoxia induces peptides (Hypoxia Induced Factors = HIF's) and thereby links short period drops of blood oxygen to generation of systemic inflammation [26-36] Simplistically the process of red blood cell accommodation to protracted low oxygen is unable to adapt to ultra-short periods of cyclic hypoxia and recovery.

These issues are now reasonably well established and accepted in literature but require transition into clinical applications. Because of the wide-spread of these issues (diabetes to dementia; from atherosclerosis to arthritis and so on, there is no part of health delivery that is exempt, nor is there a single discipline that can and has focused on the whole picture.

These implications have posed clinical issues which remain unresolved. These are a small number of cases where other-wise intractable pain has been resolved and remains resolved when inciting factors were reinstated by withdrawal of oral–splint therapy. These are neither neuropathic pains nor associated with trauma as recovery is too rapid and in similar cases, withdrawal of oral splints has seen recurrence of symptoms and this process continues until the involved tissues had adequate time to heal. In this case and a few similar cases, the rapidity denies either neuropathy or trauma.

**Keywords:** Nociception; Embryonic Growth; Sugar; HOX Genes; Midface; Naso-Oro-Pharynx; Diabetes Dementia; Oral Splint; Intermittent Hypoxia; Blood Oxygen; HIF's; Systemic Inflammation; Pain

## Abbreviations

HOX: Genes; NOP: Naso-Oro-Pharynx; HIF's: Hypoxia-Induced Factors; TMJ: Temporomandibular Joint; HIP: Hamular Notch/Incisive Papilla.

## Methods

Case - based records, demonstrating from records of a single case immediate recovery of significant neurological problems.

Standardized records include palpation of face, jaw head and neck muscles.

## Presentation

Male 50 yo. Presented with history of left-side numbness on waking. This occurred on waking every day for some months. This would resolve within 2 -3 hours. He was unable to function in work or in leisure most mornings including driving in traffic.

## Treatment history

The patient reported attending numerous health professionals. To date there was no treatment offered that made any measureable impact.

The patient felt that removal of old amalgam dental restorations may prove helpful and this was undertaken using IAOMT protocols, results were without issue but as expected proved ineffective.

## Screening process

### Motor reflex testing

A protocol of comparing muscle strength and ability to resist gentle/moderate limb deflection – was used. Where there is a significant source of afferent nociception there is a centrally driven diversion of motor resources into protection of this or these sources. This is at the expense of non-involved muscles. By altering posture and/or anatomical relationships such as sitting when there is low-back pain, there are changes in the diversion pattern that can be tested by reversing the change in posture [37-41]. In this case the MRT showed that the primary source of afferent signaling was in his jaw joint. The vascular bed of the TMJ is highly innervated with type 4 nociceptors. Compression of these is akin to compression of the sclera of the eye. Otsuka [42] showed there were rapid brain-area changes in response to compression of these vascular tissues via the Trigeminal system.

Palpation of face, jaw head and neck muscles; multiple trigger points noted both in head posturing muscles such as SCM.

### Radiographs

Tomogram of tmj, AP skull and Lateral head radiographs.

### Tracing of lateral head xrays

Analysis of lateral head radiographs is by Yosh Jefferson's modified Sassouni method.

### Mounted plaster casts

These are mounted in a non-arcon articulator which prevents the problems of arc where the opening or vertical increases between upper and lower casts are far greater in the anterior teeth than in molars. A rack-pinion system allows a vertical increase in a linear manner. Mounting is typically done using HIP (Hamular Notch – Incisive Papilla) which relates the bite registration to the skull base via the sphenoid bones.

Bite registration is taken using phonetics. The Sibilant Phoneme is a process where use of speech offers appropriate, predictable and demonstrable ideal, or close to ideal placement of internal components of the TMJ and symmetry of the chewing muscles [43]. This is a word with, in this case, an "S" buried in it. This results in ideal mandibular positioning.

### Pharyngometery

In essence this is the use of 'sonar' and uses the reflection of the sound waves coming off the tissues both in terms of time lag and in terms of echo volume to indicate how far from the source the echo was generated and its volume indicates how large the reflecting wall is Ecovision appliances USA.

Figure 1 typical tracing and data associated with juvenile orthodontic case with smaller than ideal airway, in this case no amount of jaw relocation altered this, confirming orthodontic treatment in increase width and vertical dimension would be important and helpful.

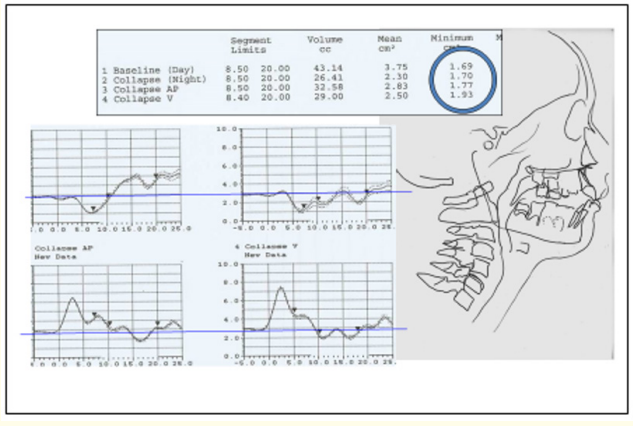


**Figure 1**

## Appliances

Fabrication of a night appliance to both reduce the impact of muscle loads by inhibition of major chewing muscles, to prevent mandibular horizontal swinging and thereby prevent unilateral compression of the TM Joint and to maintain a patent nocturnal oro-pharyngeal airway. Fabrication of a 'day' appliance maintains a 'close to normal' function with a clip-on set of teeth designed to add both vertical and anterior placement of the mandible (Figure 2,3).

Here the mandibular condyle has completely squashed the vascular bed and was noted on the CT scan ordered by neurologist to rule out intracranial masses. This patient had endured a migraine 24/7 for a year and got total pain reduction within a few hours of relocating the condyle with appropriate treatment. This patient had sought relief from many but treatment had been symptomatic. The potency of the TM Joint is underrated. The condyle is seen inside the blue oval.

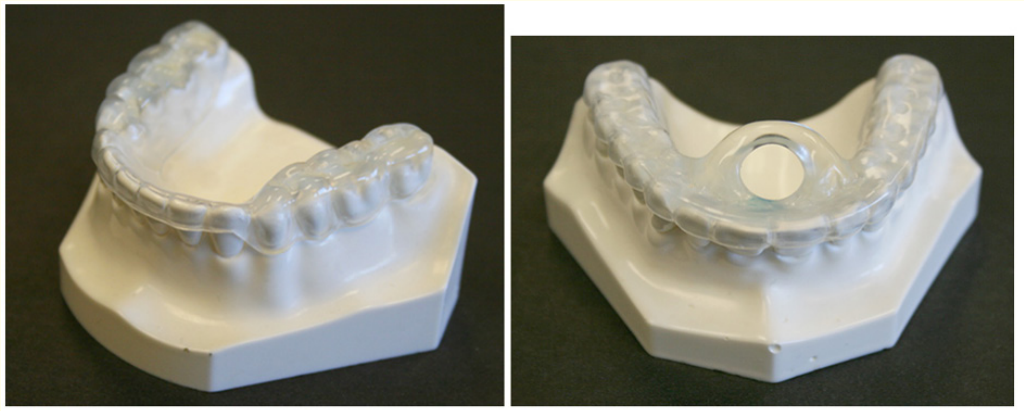


**Figure 2:** Photo of Olmos appliances courtesy John's Lab.

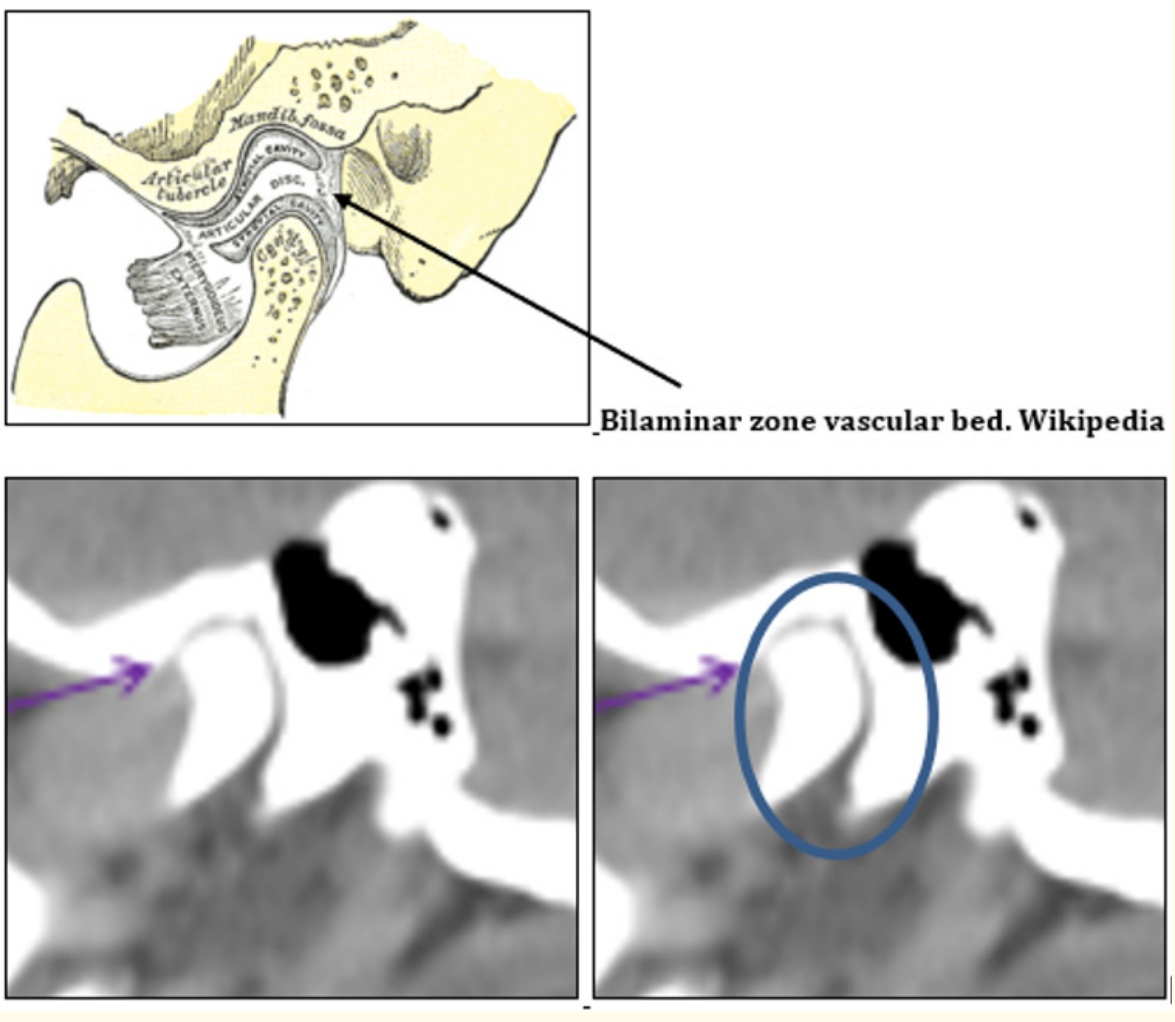


**Figure 3**

## The response

The patient failed to report as requested within 24 hours, but did return for followup 5 days later. He apologised for not contacting, but reported the morning following delivery of appliances he;

1. Could read newspaper without glasses.
2. Had no numbness
3. Had stable posture and gait.
4. Regained mental clarity.

His response was guarded as he did not trust the reality of the result. After 3 days without symptoms he realised the result was genuine. These changes were both rapid and for him unexpected.

Dr. Al Fonder DDS in his text The Dental Physician 1970 [Dental Arts pub] reports this was common finding in those who had repeated failed treatment.

## The problem

Neurologically, removal of the primary stimulus is in keeping with reduction of signs and symptoms. He enjoyed this regained benefit and it unlike most cases, he reported weaning off the appliances within a few weeks did not see return of symptoms. Other similar cases showed often immediate (less than 24 hours) total and durable relief. The question is why the pattern of signs and symptoms does not return with re-establishment of the original cause?.

## Findings

The rapid relief offered by simple mechanisms that are predictable in their likely outcome, proven efficacious and non-invasive/reversible opens a valid and practical pathway for the treatment of pain. The lack of a valid triage protocol that is able to use spinal level motor response removes cognitive involvement of the patient; removes the prospect of relying on patient to tell you where subclinical pain (or more accurately afferent nociceptive signalling) stems from; shows the immediate neurological response and reversal of motor diversion based on an anatomical change in the inciting source such as sitting down (LBP) and mandibular repositioning in migraine, TMD neck pain. These simple processes have good test/retest results [37,41,44,45]. Appliances are tolerable and removable, can be easily made by a dental laboratory –with minimal training and reasonable in terms of costs.

## Summary: Treat the cause.

There is a pattern of symptomatic treatment across health spectra. The problem has been the physically small but physiologically large changes wrought by lifestyle and dietary change nearly two centuries ago. A smaller mid-face and airway has created in as Dr. James Mold noted a population where most of those attending a primary care clinician have a sleep-related problem [4]. Mold and five colleagues screened the records of 180 PCP's across USA in five large facilities and using a list of comorbidities to sleep-linked pathology, they arrived at this. They also noted that more than 90% of the adults visiting a PCP on any given day were experiencing sleep-related problems. Based on Berlin Questionnaires more than one third were at high risk of having sleep apnoea…. Most have not discussed their sleep-related questions with their PCP. Very few PCP's screen for OSA…. Very few of them are being diagnosed or treated.

Given the spectra of signs and symptoms, from early infancy [Bonuck and Freeman] who show the early years tainted as to behavioural profiles, Kheirandish-Gozal, who showed with imaging snoring juveniles are clearly indicating major white matter degradation and the rise of hard evidence that links most of the major health issues to OSA-related systemic chemical changes including inflammation is given context when it is recognised. Jaw pain is now rivalling tooth-ache as the most frequent presenting dental problem at A & E across USA. Dementia is reported to be the second most frequent cause of non-accidental death in Australia, and despite the resources applied remains a puzzle.

There is equally a very high percentage of sufferers who inhabit prisons and as Dr. Aldred Fonder reported in the 1970's these issues are pervasive. Fonder treated a man in his 30's who had been placed in his third 'mental institution' and about to under yet another electric shock treatment. Prompted by his psychologist brother-in-law his wife called a halt and had Fonder treat. Fonder added height to the worn teeth and while unaware of the mechanism, changed this family's life. The man was discharged 6 days later. On the first followup his wife drove the hours to visit, after then he drove himself. Fonder's text (the dental physician) has over 100 such cases logged.

When the patient is simply unaware of the fact that their brain is dealing with what arguably should be considered pain, then exposure of its presence and of its effects is difficult. While the general demeanour may offer many clues as will behaviour, the source of this may elude practitioners.

The use of MRT and an awareness of the potency of small joint afferent nociception makes a deeper understanding of how 'pain' manifests, however pain may actually be defined. Arguably how clinicians view or describe pain will be at odds with the brain's interpretation. It seems the issue revolves around whether or not the primary nociceptive source is both identified and isolated and allowed to settle and this is accompanied by a drop in afferent input both from this source and in body-total afferent loading, thereby reducing relative load to one below cognitive/awareness thresholds and that this threshold which has probably been reset higher during prolonged exposure to high afferent levels, does not fall. The gap between triggering threshold and experienced levels seems to be a critical factor.

These remaining questions, which when clarified may be a value for treatment planning in the future.

## Conclusions

There is by definition and by clinical experience a triggering threshold that in cases of protracted and severe pain appears to be reset at high levels.

When these sources and specifically the primary source of afferent nociception can be identified, they can be reversed, often non-invasively and safely. Where this is possible it seems that there develops a large gap between pre and post treatment afferent levels. It is plausible that this gap allows people to endure significant but not threshold afferent levels essentially unaware of their experiencing 'pain'. This in a few cases offers either a real or perhaps a false sense of freedom from pain and regaining their quality of life.

The cloudiness of understanding regarding the sudden reversal of signs and symptoms would normally indicate elimination of pain signalling. But as this occurs in a small fraction of similarly treated patients, who will reach freedom from pain, but only after physical healing of tissues, the question must be asked WHY?

### Take home message

Where there is ill health, there are two primary factors recognised as cause and triggers. These are the ability to breathe and freedom from pain. Discovery of both can be simple. The author often simplifies 'is your bed tidy or trashed in the morning?' And 'how many times per night do you wake to go to the toilet?'

Both are closely related to impaired and fragmented sleep via well recognised pathways.

Use of MRT in its simplest form can rule in/out if there is a significant volume of afferent nociception – even if this does not trigger awareness in the patient.

Given the epidemiology of these factors they deserve to be included in all health disciplines.

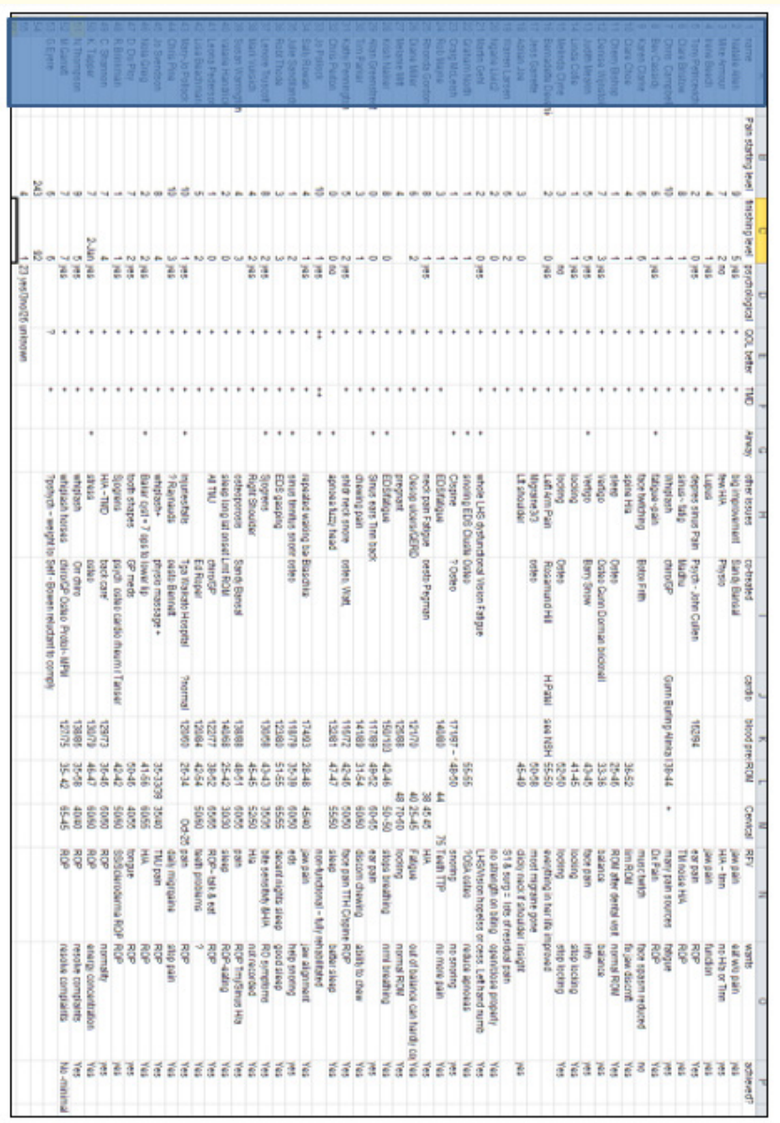

**Figure 4**

This forms the control group.

## Bibliography

1. Basu S., *et al*. "The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data". *PLoS One* 8.2 (2013): e57873.

2. Creuzet S., *et al*. "Negative effect of Hox gene expression on the development of the neural crest-derived facial skeleton". *Development* 129.18 (2002): 4301-4313.

3. Helms JA., *et al*. "Sonic hedgehog participates in craniofacial morphogenesis and is down-regulated by teratogenic doses of retinoic acid". *Developmental Biology* 187.1 (1997): 25-35.

4. Marshall H., *et al*. "Retinoic acid alters hindbrain Hox code and induces transformation of rhombomeres 2/3 into a 4/5 identity". *Nature* 360.6406 (1992): 737-741.

5. Sperber GH. "Current concepts in embryonic craniofacial development". *Critical Reviews in Oral Biology and Medicine* 4.1 (1992): 67-72.

6. Ahuja S., *et al*. "Role of normal sleep and sleep apnea in human memory processing". *Nature Science Sleep* 10 (2018): 255-269.

7. Almendros I., *et al*. "Intermittent Hypoxia Is Associated with High Hypoxia Inducible Factor-1α but Not High Vascular Endothelial Growth Factor Cell Expression in Tumors of Cutaneous Melanoma Patients". *Front Neurology* 9 (2018): 272.

8. Arnaud C., *et al*. "The inflammatory preatherosclerotic remodeling induced by intermittent hypoxia is attenuated by RANTES/CCL5 inhibition". *American Journal of Respiratory and Critical Care Medicine* 184.6 (2011): 724-731.

9. Badran M., *et al*. "Uncoupling of Vascular Nitric Oxide Synthase Caused by Intermittent Hypoxia". *Oxidative Medicine and Cellular Longevity* (2016): 2354870.

10. Baessler A., *et al*. "Treatment for sleep apnea by continuous positive airway pressure improves levels of inflammatory markers - a meta-analysis". *Journal of Inflammation* (Lond) 10 (2013): 13.

11. Berger S., *et al*. "Endothelial progenitor cells in acute myocardial infarction and sleep-disordered breathing". *American Journal of Respiratory and Critical Care Medicine* 187 (2013): 90-98.

12. Cai XH., *et al*. "Chronic intermittent hypoxia exposure induces memory impairment in growing rats". *Acta Neurobiologiae Experimentalis* (Wars) 70.3 (2010): 279-287.

13. Chen HL., *et al*. "Systemic inflammation and alterations to cerebral blood flow in obstructive sleep apnea". *Journal of Sleep Research* 26.6 (2017): 789-798.

14. Daulatzai MA. "Death by a thousand cuts in Alzheimer's disease: hypoxia--the prodrome". *Neurotoxicity Research* 24.2 (2013): 216-243.

15. de Lima FF., *et al*. "The role inflammatory response genes in obstructive sleep apnea syndrome: a review". *Sleep Breath* 20.1 (2016): 331-338.

16. Decker MJ., *et al*. "Episodic neonatal hypoxia evokes executive dysfunction and regionally specific alterations in markers of dopamine signaling". *Neuroscience* 117.2 (2003): 417-425.

17. Dewan NA., *et al*. "Intermittent hypoxemia and OSA: implications for comorbidities". *Chest* 147.1 (2015): 266-74.

18. Doumit J and Prasad B. "Sleep Apnea in Type 2 Diabetes". *Diabetes Spectr* 29.1 (2016): 14-19.

19. Drager LF., *et al*. "Translational approaches to understanding metabolic dysfunction and cardiovascular consequences of obstructive sleep apnea". *American Journal of Physiology-Heart and Circulatory Physiology* 309.7 (2015): H1101-111.

20. Feldstein CA. "Blood pressure effects of CPAP in nonresistant and resistant hypertension associated with OSA: A systematic review of randomized clinical trials". *Clinical and Experimental Hypertension* 38.4 (2016): 337-346.

21. Gaines J., *et al*. "Increased inflammation from childhood to adolescence predicts sleep apnea in boys: A preliminary study". *Brain, Behavior, and Immunity* 64 (2017): 259-265.

22. DG., *et al*. "Putative links between Sleep apnoea and cancer: From hypothesis to evolving evidence". *Chest* 148 (2015).

23. Gras E., *et al*. "Endothelin-1 mediates intermittent hypoxia-induced inflammatory vascular remodeling through HIF-1 activation". *Journal of Applied Physiology* 120.4 (2016): 437-443.

24. Hopf HW., *et al*. "Hyperoxia and angiogenesis". *Wound Repair Regen* 13.6 (2005): 558-564.

25. Iturriaga R., *et al*. "Role of Carotid Body in Intermittent Hypoxia-Related Hypertension". *Current Hypertension Reports* 19.5 (2017): 38.

26. Aviles-Reyes., *et al*. "Intermittent hypoxia during sleep induces reactive gliosis and limited neuronal death in rats: implications for sleep apnea". *Journal of Neurochemistry* 112.4 (2010): 854-869.

27. Caramelo C., *et al*. "Response to hypoxia. A systemic mechanism based on the control of gene expression". *Medicina (B Aires)* 66.2 (2006): 155-164.

28. Coleman ML and Ratcliffe PJ. "Oxygen sensing and hypoxia-induced responses". *Essays Biochemistry* 43 (2007): 1-15.

29. Gilkes DM and Semenza GL. "Role of hypoxia-inducible factors in breast cancer metastasis". *Future Oncology* 9.11 (2013): 1623-1636.

30. Grek CL., *et al*. "Hypoxia up-regulates expression of hemoglobin in alveolar epithelial cells". *American Journal of Respiratory Cell and Molecular Biology* 44.4 (2011): 439-447.

31. Haase VH. "Regulation of erythropoiesis by hypoxia-inducible factors". *Blood Review* 27.1 (2013): 41-53.

32. Ichiki T and Sunagawa K. "Novel roles of hypoxia response system in glucose metabolism and obesity". *Trends in Cardiovascular Medicine* 24.5 (2014): 197-201.

33. Imtiyaz HZ and Simon MC. "Hypoxia-inducible factors as essential regulators of inflammation". *Current Topics in Microbiology and Immunology* 345 (2010): 105-120.

34. Trayhurn P. "Oxygen - the forgotten nutrient". *Journal of Nutritional Science* 6 (2017): e47.

35. Triner D and Shah YM. "Hypoxia-inducible factors: a central link between inflammation and cancer". *Journal of Clinical Investigation* 126.10 (2016): 3689-3698.

36. Wiesener MS and Maxwell PH. "HIF and oxygen sensing as important to life as the air we breathe?" *Annals of Medicine* 35.3 (2003): 183-190.

37. Campbell SK. "Neural control of oral somatic motor function". *Physical Therapy* 61.1 (1981): 16-22.

38. Deriu F., *et al*. "Non-nociceptive upper limb afferents modulate masseter muscle EMG activity in man". *Experimental Brain Research* 143.3 (2002): 286-294.

39. Fryer G., *et al*. "Paraspinal muscles and intervertebral dysfunction: part two". *Journal of Manipulative and Physiological Therapeutics* 27.5 (2004): 348-357.

40. Takata M., *et al*. "Excitation and inhibition of trigeminal motoneurons by palatal stimulation". *Experimental Brain Research* 87.4 (1991): 497-504.

41. D Z. "Using motor reflex challenge to identify and appropriately nociceptive sources offers more tailored treatment with prompt and durable outcomes". *Journal of neurorehabilitation* 5.6 (2018).

42. Otsuka T., *et al*. "Effects of mandibular deviation on brain activation during clenching: an fMRI preliminary study". *Cranio* 2009 27 (2): 88-93.

43. Singh GD and Olmos S. "Use of a sibilant phoneme registration protocol to prevent upper airway collapse in patients with TMD". *Sleep Breath* 11.4 (2007): 209-216.

44. Ali A and Sabbahi MA. "Test-retest reliability of the soleus H-reflex in three different positions". *Electroencephalography and Clinical Neurophysiology* 41.1 (2001): 209-214.

45. Sato T and Harada Y. "Depression of the H-reflex during tooth grinding in sleep". *Physiology Behavior* 9 (1972): 893-894.

46. Mold JW., *et al*. "Identification by primary care clinicians of patients with obstructive sleep apnea: a practice-based research network (PBRN) study". *Journal of the American Board of Family Medicine* 24.2 (2011): 138-145.

**Volume 2 Issue 6 June 2019**