

# Oral and Maxillo-Facial Surgeons and Migraine

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## Introduction

I qualified as a Maxillo-Facial and Oral Surgeon in 1972 at the University of the Witwatersrand in Johannesburg, South Africa.

Throughout my professional career, one of my main preoccupations has been the prevention and treatment of pain.

It all started with the pain following impacted wisdom tooth removal. This led me to develop techniques for the removal of impacted wisdom teeth through the smallest possible incisions, which resulted in minimal post-operative swelling[1]. Small incisions went against the current teaching at the time which was 'Big Surgeon, Big Cut'. Despite the reduction in post-operative swelling, however, I was perplexed that patients still experienced the same amount of post-operative pain. What I found was that the post-operative pain was not so much related to the size of the incision nor to the amount of swelling – it was instead coming predominantly from the masseter muscles. I then discovered that the patients most prone to developing post-operative pain were those whose masseter muscles were tender to palpation prior to surgery. These patients concurrently had tenderness to percussion of the lower sevens. To reduce post-operative pain, I discovered that pre-operative occlusal adjustment of the tender sevens was all that was necessary.

I then turned my attention to the treatment of Myofascial Pain Dysfunction (MPD). This led to my inventing a new design of intra-oral appliance to achieve relaxation of the craniomandibular muscles. I called it the 'Posture Modifying Appliance' (PMA). The PMA is NOT a bite plate. It does not affect the occlusion at all, and exerts its effect through the tongue. It is so comfortable that it can be worn day and night and it is adjusted so as not to affect speech at all. This means that it exerts its effect 24/7. As it does not cover the occlusal surfaces of the teeth, the patient cannot grind through it. The results for the treatment of MPD are stunning.

What I did not anticipate though, was that some of my patients subsequently reported that since wearing the PMA, they were no longer experiencing migraine attacks. On studying the specialist headache literature, I found that muscle tension affecting the craniomandibular and craniocervical muscles was an important source of migraine pain [2].

My interest in migraine was piqued, and consequently, the treatment of migraine gradually supplanted other aspects of my Oral and Maxillo-Facial surgery practice. I have restricted my practice since 1992 almost exclusively to the diagnosis, treatment and research of migraine and other primary headaches, particularly tension headache and cluster headache. The consequence of my studying the subject of primary headaches from an Oral and Maxillo-Facial Surgeon's point of view, is that I have had the unique opportunity to approach the subject from a completely different perspective to the traditional approach taken by Neurologists. My success rate speaks for itself, and to date I have patients flying to South Africa from the four corners of the globe for treatment that they are unable to receive in their countries of origin.

## What is migraine?

The main theories in modern times are the 'vascular' theory (the pain comes from certain arteries) and the 'neurogenic' theory (the problem originates in the brain). The dispute between the proponents of the vascular and neurogenic theories has been raging for many years now. Progress in the understanding of migraine has been severely retarded by this

controversy, as researchers spend vast amounts of time and energy trying to prove the superiority of their preferred theory. The tragedy is that there need be no dispute. The proponents of the neurogenic theory claim that migraine starts in the brain. On the other hand, supporters of the vascular theory do not claim that migraine starts in the blood vessels. What they are saying is that the pain comes from the blood vessels, and not that migraine starts there. The two groups are at odds with each other, but in reality they are talking about different things. The proponents of the 'neurogenic' theory are theorizing about what initiates a migraine attack, while the proponents of the 'vascular' theory are describing the end result of an attack.

The fact that most headache experts are Neurologists has no doubt given added impetus to the popularity of the neurogenic theory, simply because Neurologists specialize in diseases of the brain and nervous system. This is their field of expertise. A reasonable consequence of this is that they would be more likely to look at the brain instead of at other structures that do not form part of their knowledge base.

## The Brain and Migraine

There is no dispute that there are indeed brain changes during a migraine attack, but whether they cause the attack or are caused by the attack has yet to be determined. Both may be true. Many medical conditions cause brain changes, even though they do not originate in the brain. In time it may indeed be proven that there is an underlying brain problem in migraine, but until such time it remains pure speculation. One prominent researcher wrote, after reviewing the evidence, 'to presume that migraine is always generated from the brain, based on the evidence, is naïve at best and unscientific at worst [3].

What has been proven though is the opposite - that pain in structures remote from the brain can most certainly lead to the kind of brain changes that have been observed in migraine [4]. This means that the brain changes that occur during migraine may possibly be caused by the migraine attack, instead of the brain changes causing the attack.

The following statement may be closest to the truth 'If we swing between the vascular and neurogenic views of migraine, it is probably because both vascular and neurogenic mechanisms for migraine exist and are important [5].

Notwithstanding the hitherto unknown underlying pathological process that initiates a migraine attack, the end result is severe pain, usually accompanied by nausea and vomiting, photophobia, phonophobia, and a plethora of other unpleasant symptoms. Whatever it is that initiates migraine attacks, if one can break any link in the chain of events that takes place during an attack, the end result of pain and the other symptoms can be prevented.

## Why Migraine Treatment so Frequently Fails

The most important factor in the failure of migraine treatment worldwide is that migraine is thought by most doctors to be a Neurological condition. As a consequence, migraineurs are referred to Neurologists for specialist treatment. Neurologists play a vital role in medical science, but they are sorely ill-equipped to diagnose and treat migraine and other primary headaches. Their failure rate is testament to this fact. The reason for this is that Neurologists are not trained to treat conditions affecting the extracranial structures.

I will present published proof that the pain of migraine does not originate from those parts of the body that are studied when one becomes a Neurologist, but from the extracranial structures. World-wide, the Neurology curriculum does not include the study of these structures. This is why the current treatment of migraine is totally inadequate.

I will next explain the origin of the pain in migraine.

I will then explain why migraine should be treated by Oral and Maxillo-Facial Surgeons and not by Neurologists. Although this chapter deals mainly with migraine, the same principles hold true for other varieties of primary headache, such as tension headache and cluster headache.

Migraine is an exceedingly common condition, with a 1-year prevalence of approximately 18% in women, 6% in men, and 4% in children [6]. This means that there are approximately 1 billion migraine sufferers world-wide.

The intensity of the pain, combined with the concomitant neurological, gastrointestinal, and sensory symptoms, reduces the quality of life of migraineurs significantly [7,8]. The quality of life of migraineurs is worse than that associated with other debilitating conditions, such as arthritis and diabetes, and is comparable to the level of dysfunction seen with recent myocardial infarction or congestive heart failure [9].

## Present Day Diagnosis of Migraine

At present specialist treatment of migraine is carried out by Neurologists. There are, however, major problems with the way Neurologists diagnose and treat migraine.

The main problem is that, according to the 3rd edition of International Classification of Headache Disorders (ICHD), published in 2013, [10] and compiled by Neurologists, the diagnosis of migraine is determined solely by the symptoms that the patient experiences during an attack. This classification, based solely on symptoms, is almost universally accepted by Neurologists as the 'gold standard' for migraine diagnosis.

According to the ICHD migraine is diagnosed if the pain is characterised by unilaterality and is of a throbbing nature, is accompanied by nausea and vomiting, photophobia and phonophobia, and is exacerbated by exercise. The illustration below shows that the ICHD is simply an extension of the original description by Hippocrates in 400BC.

	<b>UNILAT</b>	<b>NAUSEA VOMITING</b>	<b>PULSATION PHOTOPHOBIA PHONOPHOBIA</b>	<b>SEVERITY WORSE WITH EXERCISE</b>
<b>HIPPOCRATES 400BC</b>	<b>X</b>	<b>X</b>		
<b>Olesen 1978</b>	<b>X</b>	<b>X</b>	<b>X</b>	
<b>ICHD 2013</b>	<b>X</b>	<b>X</b>	<b>X</b>	<b>X</b>

The fatal flaw in both Hippocrates' and the ICHD diagnosis is that neither provides any information as to the reasons for, or the origin of the pain and associated symptoms. The ICHD has taken migraine science back by 2400 years. As will be explained later, the same symptoms can be generated by different mechanisms, and unless the underlying mechanism has been identified, the clinician simply doesn't know what he or she is treating.

Unbelievable as this may seem, according to the ICHD the clinician is not required to examine the patient when making a diagnosis – the diagnosis is made solely on the history – on the symptoms that the patient describes. This is contrary to one of the most fundamental principles of the practice of medicine. More mistakes are made from want of a proper examination than for any other reason.

In 2012 at the Congress on Controversies in Neurology I debated the validity of the ICHD with the Chairman of the Classification Committee of the International Headache Society, Professor JesOlesen, and was judged the winner of the debate by those present. There were however no changes in the 2013 edition of the ICHD.

As will be explained in greater detail later in this chapter, the symptoms do not reveal any information as to the origin of the pain – and in migraine the pain does not always originate from the same anatomical structures. If the clinician does not examine the patient, it is impossible to determine the origin of the pain. As medical students, we were constantly reminded of the old maxim 'if you do not put your finger in (on) it, you are going to put your foot in it'. Migraine specialists who see no need to examine their patients are constantly 'putting their foot in it'.

The first edition of the ICHD was published in 1985. It supplanted the previous classification, which was compiled by the "Ad Hoc Committee", a group of Neurologists with a special interest in primary headaches. The Ad Hoc Committee classification was based upon the anatomical structures from which the pain emanated [11]. The main groups were:

- 1 Vascular headache of the migraine kind
- 2 Muscle contraction headache, and
- 3 Mixed vascular/muscular headache

The replacement of this classification with the ICHD, based on symptoms alone, was a fundamental error, which has severely retarded the development of migraine science [12,13]. The reason for this is that vascular pain and muscular pain can cause precisely the same symptoms! The consequences of this are:

- 1 Patients diagnosed with migraine according to the ICHD receive the same treatment whether their pain is vascular,

muscular, or mixed vascular/muscular. This results in the treatment being hit-and-miss, so that patients frequently get the wrong treatment.

2 Research based on the symptomatic classification does not distinguish which type of migraine is being researched –muscular, vascular, or mixed. No migraine research articles are accepted by the specialist headache journals unless the cohorts have been diagnosed according to the diagnostic criteria laid down in the ICHD. The consequence of this is that all research into migraine medications and other treatment modalities that is based on the ICHD is scientifically invalid. As the ICHD has been used by researchers since 1985, all migraine research since then is fatally flawed.

## Migraine triggers

The diagnosis of migraine is further confused by the presence of triggers. The list of possible triggers is endless, but the most common ones are stress, hormonal changes, certain foods and food additives, alcoholic drinks, dehydration, a rapid drop in blood sugar, changes in the weather, and sensory stimuli. Although some migraineurs are able to identify their particular trigger, in most cases there is no identifiable trigger. Some of these triggers can be avoided, but others cannot.

Triggers confuse the diagnosis as most people blame the trigger for their migraines. What in fact really occurs is that the patient has an underlying vascular or muscular dysfunction, and that their particular trigger exacerbates this dysfunction causing pain. If the vascular or muscular dysfunction is successfully treated, the triggers are no longer active in bringing on attacks.

The hormone fluctuations in the menstrual cycle often trigger migraine attacks. What is interesting though, is that most women with menstrual migraine have a regular 28 day cycle, which indicates that their hormone levels are normal. If the origin of the pain in the arteries or muscles is identified and treated, hormonal migraine no longer occurs. The same is true for all the other triggers.

## Present Day Migraine Treatment

Present day Neurological migraine treatment is with medication. There are however major problems with:

- 1 When these medications are prescribed,
- 2 Their efficacy, and
- 3 Their side-effects.

The medications are either abortive or preventive.

### Abortive medications

The abortive medications are either non-specific analgesics or vasoconstrictors such as the triptans or the ergots. These medications are intended to reduce or eliminate the pain, but although they are effective in some cases, there are frequently major problems associated with their use.

The first problem is that when patients are diagnosed with migraine according to the ICHD, the most commonly prescribed drugs are the triptans, vasoconstrictors that were specifically developed to treat migraine. A comprehensive meta-analysis of 53 trials showed that the triptans, once touted as the ‘miracle migraine drugs’, were effective in only approximately 25% of patients when used in three consecutive attacks [14]. The reason for this is that in many of these patients their migraine symptoms are muscle-related and not arterial. The ICHD does not distinguish between muscle-related migraine and vascular migraine.

The second major problem encountered with the abortive medications is Medication Overuse Headache (MOH). MOH occurs when patients need to medicate on a regular basis. Using analgesics more than two or three times a week exposes the patient to the possibility of developing MOH. In MOH, although the medication may be palliative in the short term, in the long term the attacks become more frequent and more severe. A vicious circle is set up and the patient is sucked into a downward spiral of taking more and more drugs and getting worse and worse headaches. MOH is more likely to occur if the analgesics are combined with caffeine and/or codeine, but the most frequently prescribed migraine abortive drugs, the triptans and the ergots, are the most potent causes of MOH.

## Preventive medications

The preventive drugs, either antidepressants, anticonvulsants, or beta-blockers are often so ineffective and may have such severe side-effects that they are only prescribed for the very worst cases – and even then most people stop taking them because either they don't help, or the side effects are worse than the pain. Among patients prescribed migraine preventive medication, only 12% were still using the medication 12 months later [15].

## The nightmare journey

Migraine sufferers the world over are taken on a nightmare journey from doctor to doctor and from ineffective drug to ineffective drug. First they try over-the-counter drugs, then they visit the general practitioner who prescribes analgesics, and when these don't help they are referred to a Neurologist. The Neurologist prescribes a variety of drugs, abortive and preventive, and when they are ineffective, the patient is told that unfortunately they have to learn to live with the pain.

In order to develop more effective abortive and preventive treatment modalities for migraine, it is essential to have clear understanding of the mechanisms involved and where the pain originates.

## The Oral and Maxillo-Facial Surgery connection

As Thomas Kuhn, the author of 'The Structure of Scientific Revolutions' wrote 'Individuals who break through by inventing a new paradigm are almost always very young or very new to the field. Those are the people who, because they are not committed to the traditional theories, are more likely to see the flaws in those theories, and conceive others to replace them!'

I have been one of those fortunate enough to be able to invent a new paradigm because of the fact that I, as an Oral and Maxillo-Facial Surgeon, was not schooled in the traditional theories on the diagnosis and treatment of migraine and other primary headaches.

What I found is that the pain of migraine and other primary headaches originates exclusively from the extracranial structures -those parts of the anatomy that we, as Oral and Maxillo-Facial Surgeons study during our training, and that we are more qualified to diagnose and treat than any other discipline.

## The origin of the pain in migraine

The pain in the vast majority of primary headache sufferers originates in either the terminal branches of the external carotid artery, or from the pericranial muscles, or from a combination of these structures.

A search of the literature confirms that:

- a) There is no evidence that the pain of migraine originates from any intracranial structure [16-18].
- b) The involvement of the extracranial terminal branches of the external carotid artery in migraine pain has been extensively documented [19-30]. This evidence has however been ignored and even actively suppressed by some in the Neurological headache community [31,32]. There is no mention of arterial pain in the ICHD [10].
- c) The involvement of the pericranial muscles in migraine pain has also been extensively documented [2, 33-40]. This evidence has also been totally ignored, and there is no mention of muscle pain in the ICHD [10].

Neither vascular pain nor muscular pain, the presence of both of which can easily be determined by clinical examination, forms part of the diagnostic criteria in the ICHD [10]. Specialist training for Neurology does not encompass the extracranial vasculature or musculature, so Neurologists, unlike Oral and Maxillo-Facial Surgeons, do not have the necessary training to enable them to accurately diagnose the origin of migraine pain.

There are approximately 1 billion people world-wide who would benefit and whose lives would be changed if the treatment of migraine and other primary headaches was carried out by Oral and Maxillo-Facial Surgeons instead of by Neurologists.

## The Correct Diagnosis and Treatment of Migraine Pain

### Vascular pain

**Vascular pain is diagnosed by:**

1. Digital compression of the terminal branches of the external carotid arteries. The arteries most commonly involved are the main trunk of the superficial temporal artery, its frontal branch, and the occipital, maxillary, posterior auricular, and angular arteries [41]. Although these are the arteries most commonly involved in migraine, others may be involved. It must be borne in mind that there are significant variations in the anatomy of the terminal branches of the external carotid artery. There are not only variations from patient to patient, but frequently from left to right in the same patient [42].

2. Application of an inflatable head band to reduce the blood flow to the painful distended arteries.

3. Sometimes the patient will volunteer that pressing on certain parts of the scalp reduces the pain level. The areas commonly indicated by patients are the temples (frontal branch of the superficial temporal artery) and the mesial part of the orbital rim (the angular arteries).

4 Some patients experience relief when they tie a tight band round the head [43].

Methods 1) and 2) above can only be employed if the patient is examined during an attack.

In order to ensure the optimum benefit from migraine vasoconstrictive drugs, they should only be prescribed for patients who have had a positive diagnosis of vascular pain.

### Treatment of vascular pain

**Abortive:** The treatment of vascular pain varies from patient to patient, depending upon the frequency and severity of the migraine attacks. Usually mild or infrequent attacks are adequately treated by abortive medication. The most frequently prescribed abortive medications are the triptans. In patients with more severe or frequent pain, the triptans may not be effective in controlling attacks. There is also the danger of their frequent use leading to MOH.

Treatment in the Emergency Room is usually either with triptans, ergots, or with systemic medication

### Abortive Arterial Pain Relief at The Headache Clinic

At The Headache Clinic in Johannesburg, emergency pain relief is carried out only after the source of the pain has been identified. For vascular pain we use a number of innovative techniques.

First, we apply a dental cotton wool roll or a small ball of cotton wool, soaked in ethyl chloride, to the skin over the painful artery. The most common area is just anterior to the tragus where the superficial temporal artery crosses the root of the zygomatic arch. One can usually quite easily palpate the throbbing superficial temporal artery so that the cotton wool can be accurately applied. This simple, unique technique, is often sufficient to achieve complete pain relief, as not only the superficial temporal, but all the terminal branches of the external carotid reflexly constrict because of the cold. This is non-invasive and is quite safe as the boiling point of ethyl chloride is 12°C, well above the temperature that may cause frostbite.

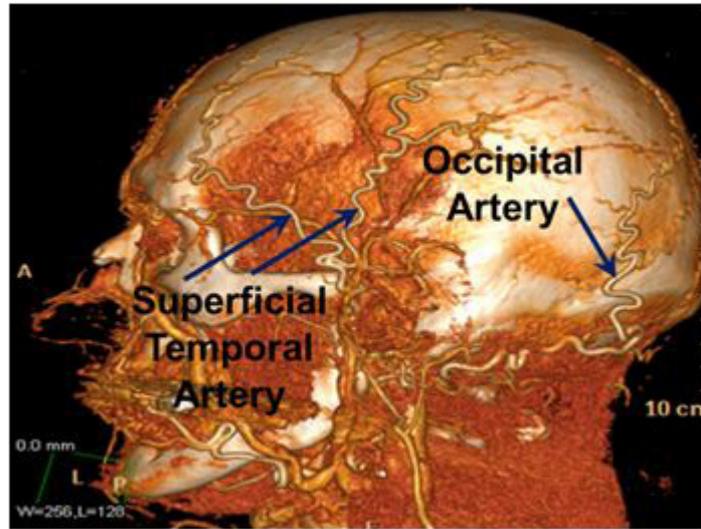
In patients that do not respond to ethyl chloride, a saline pomphus is injected periarterially, with excellent pain relief. This may have to be repeated at a number of sites on the scalp, but as with the ethyl chloride, the pain relief is almost instantaneous [44].

**Preventive - medication:** For the prevention of migraine, the most commonly used drugs are the anti-epileptics, the anti-depressants, and the beta-blockers. Among patients prescribed migraine preventive medication, only 12% were still using the medication 12 months later [15]. The poor persistence with migraine preventive drugs is because a) they are not always effective in preventing migraine, and b) the side-effects are frequently more unpleasant than the migraines.

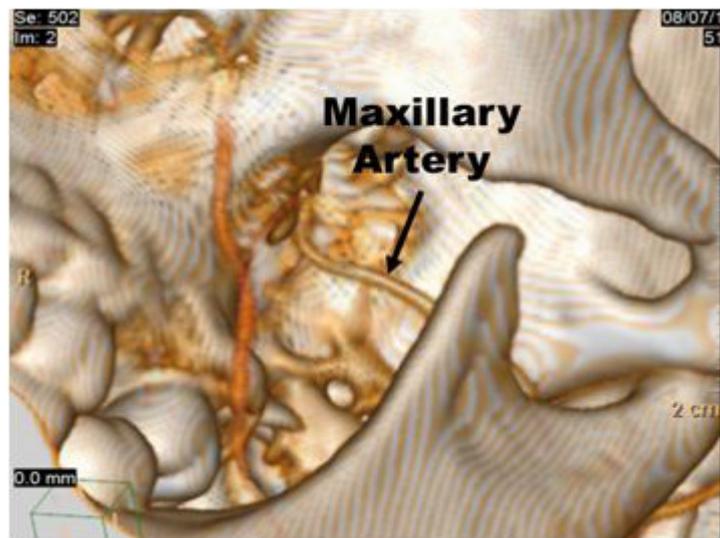
**Preventive – surgical:** Permanent prevention of arterial pain in patients who are not adequately controlled by medication can be achieved by surgical closure of the relevant arteries. Oral and Maxillo-Facial Surgeons are ideally positioned to carry out these procedures.

As detailed before, the vessels most commonly involved in migraine pain are the superficial temporal main trunk, the frontal branch of the superficial temporal, the occipital the angular, the maxillary, and the posterior auricular.

Before the surgery is undertaken, a 3 Dimensional CT Angiogram is done, to enable pinpoint location of the involved arteries. Accurate location of the arteries enables the surgeon to make the smallest possible incisions, so that the surgery is minimally invasive.



**Figure 1:** Dimensional CT Angiogram showing the accurate location of the arteries.



**Figure 2:** 3 Dimensional CT Angiogram showing the position and orientation of the maxillary artery. This varies from patient to patient and from left to right in the same patient.

The maxillary artery may also be involved in the generation of migraine pain. In cluster headache, the maxillary artery is always involved. The technique for cauterizing the maxillary artery via the intra-oral approach has been described elsewhere [45].



**Figure 3:** The exact location of each artery is confirmed with a Doppler flow meter.

The smallest possible incision is made over each of the involved arteries. Using the Doppler Flowmeter as a guide to locating the artery, the tissues overlying the artery are dissected away, the artery is exposed, and closed either with cautery or ligature.

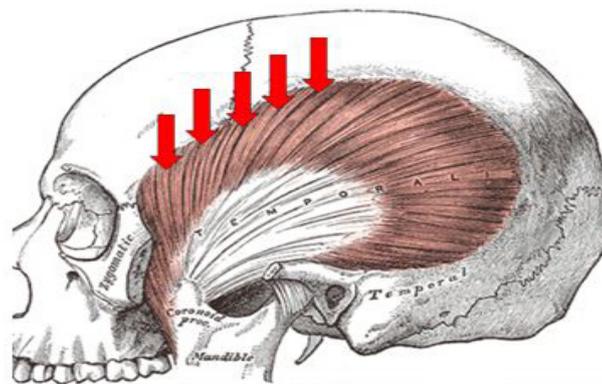


It is safe to cauterize or ligate the responsible vessels, as there are no vital structures nearby, and consequently the procedure has a low morbidity [42, 46-50].

## Diagnosis and Treatment of Muscle Pain

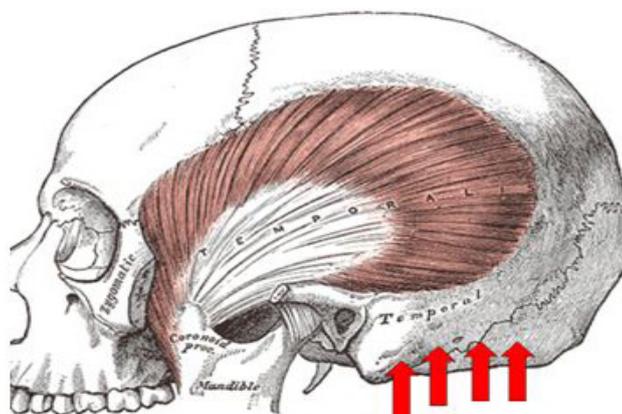
The muscles most frequently involved in migraine pain are the temporalis and cervical muscles. The result of long-standing increased tone in these muscles is that their tendinous attachments to the skull develop tender trigger points.

Any part of the muscle may be tender to palpation, but the most frequent location of tender temporalis muscle trigger points is at the attachment to the skull, as shown in the diagram below.



**Figure 5:** Location of common tender trigger points in the temporalis muscle.

The most common trigger point areas in the occipital region are found just suboccipitally in the two to three centimeters mesial to the tip of the mastoid process, where the cervical muscles attach to the skull, as in the diagram below.



**Figure 6:** Location of common suboccipital trigger points.

## Abortive treatment of Muscle Pain

The current Emergency Room treatment of migraine is undertaken, as I have explained above, without the clinician diagnosing where the pain is coming from. For this reason, Emergency Room treatment of muscle-tension related migraine is exactly the same as Emergency Room treatment of vascular pain as outlined above.

The most blatant example of how the iniquitous it is that the ICHD makes no mention of muscle pain is contained in a paper co-authored by the Chairman of the Classification Committee responsible for compiling the ICHD, Professor Jes Olesen. In this paper, they reported that 100% of migraineurs had pericranial muscle tenderness during an attack. In the same paper they reported that after injecting lidocaine or saline into tender muscle areas, 60% were discharged in excellent condition after 90 minutes. They went on to state 'This is a significantly better result than with medical therapy'. The most inexplicable part is that they concluded 'We do not suggest that common migraine should be treated with pericranial injections' [2].

## Abortive Treatment of Muscle Pain at The Headache Clinic

At The Headache Clinic in Johannesburg, we regularly use local anaesthetic injections into painful muscle trigger point to achieve almost immediate pain relief. An added advantage is that in patients where we achieve significant pain relief using this method, the diagnosis of muscle tension migraine is confirmed.

## Preventive Treatment of Muscle-related Migraine

As they are unable to diagnose where the pain originates, Neurologists use the same preventives for muscle-related migraine as they do for arterial migraine, with the same disastrous results.

## Preventive Treatment of Muscle-Related Migraine at The Headache Clinic

The treatment of MPD involving the craniomandibular muscles is predominantly by means of intra-oral appliances. A number of different designs are used. The types of splints commonly used in occlusal splint therapy include permissive, non-permissive, hydrostatic, and soft rubber (silicone) splints.

Headache and migraine are comorbid in many MPD patients [51,52]. The effectiveness of splint therapy in reducing head and neck pain and muscle hyperactivity is well documented. Occlusal splints promote muscle relaxation by providing a platform for the teeth that allows for equal distribution of tooth contacts, immediate posterior tooth disclusion in all movements (with anterior guidance), and reduced stress on the joint. Neuromuscular harmony that follows provides for optimal function and comfort. There is a plethora of evidence to show that splint therapy is effective in migraine [53-59].

The appliance that I have found to give the best results though, is what I have termed the Posture Modifying Appliance (PMA) [59]. The advantages of the PMA over other intra-oral devices are a) It is so comfortable that the patient is unaware of having anything in the mouth, so it can be worn day and night. This means that it provides round-the clock benefit, b) because of the comfort, it does not affect speech in any way, and c) as it does not cover the occlusal surfaces of the teeth, the patient cannot grind through it.



**Figure 7:** Fully adjusted Posture Modifying Appliance.

In patients where the intra-oral device is not effective, other methods, such as physiotherapy, trigger point therapy, and Botox injections are utilised.

## Discussion

According to the ICHD, primary headaches are divided into two main categories, migraine and tension-type headache. These two categories are further sub-divided according to the symptoms, which vary substantially from patient to patient. A great deal of importance is placed on categorizing which pigeon-hole the patient's headache fits into. Is it migraine or is it tension-type headache? It seems ludicrous to differentiate between the two when the drugs most commonly used for the preventive treatment of migraine and the preventive treatment of tension-type headache are exactly the same – antidepressants and anticonvulsants. This is the most damning indictment of the ICHD – it makes no difference whether the patient has this diagnosis or that diagnosis – the preventive treatment is the same. The diagnostic criteria in the ICHD are meaningless. What is a further indictment is that the diagnostic criteria used in the ICHD were not based on scientific data. The Chairman of the Classification Committee of the International Headache Society, Professor Jes Olesen, admitted as much when he wrote 'The IHS criteria were developed without the collection of data' [60]. As one commentator wrote, the criteria were arrived at by 'GOBSAT' – 'Good Old Boys Sitting And Talking' [13].

This situation cannot be allowed to continue. The amount of unnecessary suffering, world-wide, is totally unacceptable. Countless numbers of people are condemned to living miserable lives because they are being treated by people who have no idea what they are treating.

As the French writer and philosopher Voltaire (1694-1770) wrote 'Doctors pour drugs of which they know little, to cure diseases of which they know less, into human beings of whom they know nothing'. This accurately describes the present day Neurological treatment of primary headaches.

At present, patients suffering not only with intractable migraine, but with cluster headache and hemiplegic headache are forced to come to South Africa for effective treatment, so that they can resume normal, happy, productive lives [45,61].

This article is a plea to Oral and Maxillo-Facial Surgeons, and in particular those in academia, to become involved in the diagnosis and treatment of primary headaches. It will be a win – win – win situation. The patients whose quality of life will be restored will be the biggest winners. The Oral and Maxillo-Facial Surgeons will have a substantial and exceedingly satisfying addition to their scope of work – and even the drug companies will benefit once their drugs are being prescribed for the correct patients, and once their research is focussed on developing the correct medications.

## References

1. Shevell E, Koeppe WG, Butow KW. A subjective assessment of pain and swelling following the surgical removal of impacted third molar teeth using different surgical techniques. *SADJ*. 2001;56:238-241.
2. Tfelt-Hansen P, Lous I, Olesen J. Prevalence and significance of muscle tenderness during common migraine attacks. *Headache*. 1981;21:49-54.
3. Dodick DW. Examining the essence of migraine--is it the blood vessel or the brain? A debate. *Headache*. 2008;48:661-667.
4. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *J Pain*. 2009;10:895-926.
5. Edmeads J. Migraine--resuscitation of the vascular theory. *Headache*. 1989;29:55-56.
6. Silberstein SD, Lipton RB, Goadsby P. *Headache in Clinical Practice*. Oxford: Isis Medical Media; 1998.
7. Dahlof CG, Solomon GD. The burden of migraine to the individual sufferer: a review. *Eur J Neurol*. 1998;5:525-533.
8. Osterhaus JT, Townsend RJ, Gandek B, Ware JE, Jr. Measuring the functional status and well-being of patients with migraine headache. *Headache*. 1994;34:337-343.
9. Solomon GD, Skobieranda FG, Gragg LA. Quality of life and well-being of headache patients: measurement by the medical outcomes study instrument. *Headache*. 1993;33:351-358.
10. Classification, Committee, IHS. The International Classification of Headache Disorders III. *Cephalalgia*. 2013;33:629-808.
11. AdHoc, Committee. Classification of Headache. *JAMA*. 1962;179:717-718.
12. Spierings EL. Migraine, big and small. *Headache*. 2001;41:918-922.
13. de Ru JA. In theory, theory and practice are the same; in practice they are not. *Cephalalgia*. 2016.
14. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT<sub>1B/1D</sub> agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet*. 2001;358:1668-1675.
15. Hepp Z, Dodick DW, Varon SF, et al. Persistence and switching patterns of oral migraine prophylactic medications among patients with chronic migraine: A retrospective claims analysis. *Cephalalgia*. 2017;37:470-485.
16. Lauritzen M, Henriksen L, Lassen NA. Regional cerebral blood flow during rest and skilled hand movements by xenon-133 inhalation and emission computerized

tomography. *J Cereb Blood Flow Metab.* 1981;1:385-387.

17. Bednarczyk EM, Remler B, Weikart C, Nelson AD, Reed RC. Global cerebral blood flow, blood volume, and oxygen metabolism in patients with migraine headache. *Neurology.* 1998;50:1736-1740.

18. Cutrer FM, O'Donnell A, Sanchez del Rio M. Functional neuroimaging: enhanced understanding of migraine pathophysiology. *Neurology.* 2000;55:S36-45.

19. Hare F. Mechanism of the pain in migraine. *Med Pr.* 1905;1:583.

20. Graham JR, Wolff HG. Mechanism of migraine headache and action of ergotamine tartrate. *A Research Nerv & Ment Dis, Proc.* 1937;18:638.

21. Pickering GW. Experimental Observations on Headache. *Br Med J.* 1939;1:907-912.

22. Sutherland AM, Wolff HG. Experimental studies on headache: further analysis of the mechanism of headache in migraine, hypertension and fever. *Archives of Neurology and Psychiatry.* 1940;44:929-949.

23. Wolff HG, Tunis MM. Analysis of cranial artery pressure pulse waves in patients with vascular headache of the migraine type. *Trans Assoc Am Physicians.* 1952;65:240-244.

24. Wolff HG, Tunis MM, Goodell H. Studies on headache: evidence of tissue damage and changes in pain sensitivity in subjects with vascular headaches of the migraine type. *Trans Assoc Am Physicians.* 1953;66:332-341.

25. Tunis MM, Wolff HG. Studies on headache; long-term observation of alterations in function of cranial arteries in subjects with vascular headache of the migraine type. *Trans Am Neurol Assoc.* 1953;3:121-123.

26. Blau JN, Dexter SL. The site of pain origin during migraine attacks. *Cephalalgia.* 1981;1:143-147.

27. Andersen AR, Tfelt-Hansen P, Lassen NA. The effect of ergotamine and dihydroergotamine on cerebral blood flow in man. *Stroke.* 1987;18:120-123.

28. Hachinski V, Norris JW, Edmeads J, Cooper PW. Ergotamine and cerebral blood flow. *Stroke.* 1978;9:594-596.

29. Schumacher G, Wolff H. Experimental studies on headache: A. Contrast of histamine headache with the headache of migraine and that associated with hypertension. B. Contrast of vascular mechanisms in pre-headache and in headache phenomena of migraine. *Arch Neurol Psychiat* 1941;45:199-214.

30. Iversen HK, Nielsen TH, Olesen J, Tfelt-Hansen P. Arterial responses during migraine headache. *Lancet.* 1990;336:837-839.

31. Goadsby PJ. The vascular theory of migraine--a great story wrecked by the facts. *Brain.* 2009;132:6-7.

32. Shevel E. The extracranial vascular theory of migraine--a great story confirmed by the facts. *Headache.* 2011;51:409-417.

33. Bakal DA, Kaganov JA. Muscle contraction and migraine headache: psychophysiologic comparison. *Headache.* 1977;17:208-215.

34. Kidd RF, Nelson R. Musculoskeletal dysfunction of the neck in migraine and tension headache. *Headache.* 1993;33:566-569.

35. Anttila P, Metsahonkala L, Mikkelsen M, et al. Muscle tenderness in pericranial and neck-shoulder region in children with headache. A controlled study. *Cephalalgia.* 2002;22:340-344.