Temporal bone hypermobility and MS

Bruxism and temporal bone hypermobility in patients with multiple sclerosis

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Abstract

Objectives: In this preliminary study, we investigated the link between jaw clenching/bruxism and temporal bone movement associated with multiple sclerosis (MS). Methods: 21 subjects participated in this study (10 patients with MS and 11 controls). To quantify the change in intracranial diameter between the endocranial surfaces of the temporal bones during jaw clenching, an ultrasonic pulsed phase locked loop (PPLL) device was used. A sustained jaw clenching force of 100lbs was used to measure the mean change in acoustic wavelength (ΔL).

Results: In the control subjects the mean ΔL was 0.27mm ± 0.24. In subjects with MS the mean ΔL was 1.71mm ± 1.18 (p < 0.001). Conclusions: The increase in magnitude of bi-temporal bone intracranial expansion was nearly six times greater in subjects with MS compared to controls. Therefore, jaw clenching/bruxism is associated with more marked displacement of the temporal bones and expansion of the cranial cavity in patients with MS than control subjects. In future studies, we must determine if clenching/bruxism leads to temporal bone hypermobility in patients with MS or whether it is a latent sign of the disease.

Short Title: Temporal bone hypermobility and MS

Key words: bruxism, intracranial pressure, multiple sclerosis, temporal bone instability
**Introduction**

Multiple sclerosis (MS) is the major cause of non-traumatic neurological disability in young adults in North America (1. Noseworthy 2000). Patients with MS suffer from a progressive loss of normal brain function, leading to disability sometimes with severe pain, dementia and even death. Current medical management offers palliative treatment and some slowing of the disease process, but the etiology of MS remains elusive. One early study suggested that there may be a link between MS and tooth decay (2. Craelius, 1978), and this study lead researchers to investigate other dental factors associated with MS. Recently, three dimensional (3D) radiographic imaging studies have demonstrated the presence of a malpositioned superior border of the temporal bone in patients with MS. Using the live, real-time capture capability of these imaging studies, there is evidence of a shifting of the squamosal suture during sustained, maximal jaw clenching in patients with MS when compared to control subjects (REF req’d?). Studies of jaw clenching have demonstrated pressures of 975psi at the molars, and as high as 175,000psi at the incisors (12. Attanasio, 1991). Although it is assumed that structures that support the insertions of the masticatory muscles are stable and stationary, and that the impact of clenching/bruxism is strictly a dental issue, evidence is beginning to emerge that bruxism/TMD may be associated with a compromised airway (13. Singh and Olmos), and TMJ health may be important in overall cranial health, such as the prevention of concussion (14 Singh et al., 2009). Moreover, modern radiographic techniques have allowed critical evaluations of compliance in the cranial sutures with some very clear results (Oleski et al. 2002). With external manipulation of the cranial vault, temporal bone movement (the mean angle of change at the squamosal suture) is about 1.75°. Other measurements indicate that this amount of movement is common in most sutures. For example, the mastoid process moves by 1.66°, the malar line moves by 1.25° and the sphenoid bone moves by 2.4°.

It is thought that changes in intra-cranial pressure (ICP) lead to corresponding changes in intra-cranial diameter (6. Heifetz MD and Wiess M, 7. Heisey SR and Adams T). These changes can be measured using a pulsed phase locked loop (PPLL) device (8. Ueno T, et al. 2005). The PPLL
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device originally was used to measure pulsatile changes in ICP (Ueno T, Shuer LM, 1998; Ueno T, Ballard RE, 1998). The PPLL device transmits a 500kHz ultrasonic tone burst through the cranium via a transducer placed on the subject’s head. The tone burst passes through the cutaneous tissues, reflects off the ipsilateral intracranial temporal bone (echo 1), passes through the intracranial contents; reflects off the endocranial surface of the temporal bone on the opposite (contralateral) side of the skull, and is received back (as echo 2) by the originating transducer. Any change in cranial diameter produces a phase shift in the ultrasound signal (9. Ueno T, et al. 1998). The PPLL processing software is designed to track changes in the phase of the ultrasonic signal as it strikes the intracranial surfaces of the temporal bones, and converts those changes into an estimated target delay ($\Delta L$, change in acoustic wavelength; Ueno, 2005). The resulting target delay estimates are then converted into a distance measurement using the equation

$$d = \frac{1}{2} \nu t$$

where $t$ is the target delay estimate and $\nu$ is the speed of sound. Thus, the time of the phase shift is converted into millimeters of movement ($d$) of the temporal bones (change in acoustic wavelength, $\Delta L$).

In addition, mechanical tensions placed on the teeth are transmitted to the cranium. In one study, these tensile forces were found to be positively correlated with osteogenic responses in the interparietal sutures (10. Miyawaki S, Forbes DP 87). Thus, an abnormal bite relationship may exert unequal pressure on the cranial bones, which may be a precursor of cranial bone dysfunction. Therefore, the PPLL device offers a non-invasive method for evaluating the physical stresses of bruxism on the cranium and its components. In view of these technical developments and clinical observations, it was hypothesized that periodic episodes of bruxism/clenching increases ICP and cranial bone hypermobility, which may be associated with the development of MS lesions in and around the cranial ventricles. Therefore, the aim of this
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preliminary study is to test the null hypothesis that clenching/bruxism is not associated with hypermobility of the temporal bones in patients with MS.

Materials and methods

Sample
This study was approved by the UCSD Human Research Protections Program. After obtaining IRB approval, 11 control subjects and 10 patients with MS diagnosed by a neurologist participated in this study. Inclusion criteria for the control subjects were: 1) No relevant medical history; 2) Aged 18-60 years old. Exclusion criteria for the control subjects were: 1) History of chronic headaches; 2) History of cranial trauma; 3) History of neurologic symptoms or diseases; 4) Anodontia in one or both dental arches; 5) Advanced periodontitis with dental mobilities over class 1; and 6) Dental or muscular pain upon clenching. Similarly, inclusion criteria for the MS patients were: 1) Medical diagnosis of MS by a neurologist; 2) Aged 18-60 years old. Exclusion criteria for the MS patients were: 1) Anodontia in one or both dental arches; 2) Advanced periodontitis with dental mobilities over class 1; and 3) Dental or muscular pain upon clenching.

Measurement Procedures
All subjects lay supine on a bed with their head resting in a headrest to rigidly position the transducer, which sends and receives ultrasound waves (Luna Innovations, Toano, USA). The transducer’s position was adjusted manually, and the head was rigidly fixed in a frame (Fig. 1)? After the transducer was placed over the right temple of the subject, it was adjusted until strong ultrasonic echoes were obtained from the endocranial surfaces of the temporal bones. Once adjusted, a maximum strength test was conducted to measure the maximum clenching strength of the subject, using a dental bite I-scan sensor (Ref req’d GWE?). The subjects bit down on the sensor that was a Mylar sheet with pressure sensitive ink between metal tracings (Manufacturer req’d?). The sensors have double layers of thin rubber protectors to dissipate the forces and prevent perforation. The subject clenched as hard as they could for one second
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while data were acquired. This procedure was used both to test the subject’s maximum clenching force, and to show them the desired clenching levels. The subjects were able to judge their strength and maintain it at 100lb clench-force for this test. The signal was determined with the muscles at rest. The signal was checked again and data were acquired for 20secs while the subject underwent the jaw clenching procedure, as described in Table 1. The change in intracranial temporal bone diameter was expressed as \( \Delta L \) (change in acoustic wavelength in mm).

\textbf{Table 1} Jaw clenching procedure performed by each subject.

<table>
<thead>
<tr>
<th>Time (sec)</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5</td>
<td>Relaxed</td>
</tr>
<tr>
<td>5-10</td>
<td>Clenching begins, gradually increasing the force until reaching 100lbs</td>
</tr>
<tr>
<td>10-15</td>
<td>Maintain clench at 100lbs</td>
</tr>
<tr>
<td>15-20</td>
<td>Clench is released</td>
</tr>
</tbody>
</table>

To ensure that the subject was clenching around 100lb, the subject watched a video monitor, which showed clenching force. This force was detected by a load cell developed by T-scan (Manufactur req’d. GWE?). With care taken to ensure that the subject did not move the head during this procedure, one member of the research team monitored the clench strength, while a second member coached the subject on proper jaw clenching. To ensure reproducibility, all study subjects were tested several times under this protocol. For each test, the ultrasonic data were saved to a file and processed in real time.

The technique provided high-resolution measurements of the change in the position of the echo from the initial estimate. For these tests, the position of three echoes were tracked with the PPLL: An echo from the transducer’s surface at the skin; an echo from the endocranial surface of the right temporal bone just as the signal entered the cranium (echo 1); and an echo from the endocranial surface of the left temporal bone after the signal had passed through the
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cranial cavity (echo 2). After tracking changes in the position of these echoes with time, the data were saved to a file. By subtracting the difference in the position between echo 2 and echo 1, we were able to measure changes in the width of the intracranial distance between the two temporal bones, eliminating dimensional changes due to the motion of the temporal muscle during clenching. This value is the intracranial length or distance between the inner tables of the temporal bones (ΔL). In essence, the PPLL tracks changes in the distance between the transducer, the proximal (right) and the distal (left) intracranial wall. To subtract out soft tissue movement between the transducer and the proximal wall, the saved data were re-processed, this time “locked” on the echo from the proximal wall (echo 1). By subtracting the second result (echo 2) from the first, we were able to monitor changes in the distance between the proximal and distal temporal bones during clenching.

For tests of reproducibility, one subject was chosen at random. Thirteen tests and data points were obtained. The data are displayed in Table 2 with statistical analysis in Table 3.

Table 2 Reproducibility tests on subject chosen at random.

<table>
<thead>
<tr>
<th>Test Number</th>
<th>ΔL (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.385</td>
</tr>
<tr>
<td>2</td>
<td>0.077</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0.385</td>
</tr>
<tr>
<td>5</td>
<td>0.154</td>
</tr>
<tr>
<td>6</td>
<td>0.1386</td>
</tr>
<tr>
<td>7</td>
<td>0.077</td>
</tr>
<tr>
<td>8</td>
<td>0.1078</td>
</tr>
<tr>
<td>9</td>
<td>0.0924</td>
</tr>
<tr>
<td>10</td>
<td>0.1694</td>
</tr>
<tr>
<td>11</td>
<td>0.231</td>
</tr>
</tbody>
</table>
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<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>0.0462</td>
</tr>
<tr>
<td>13</td>
<td>0.077</td>
</tr>
</tbody>
</table>

These data show a 0.0332 standard error and a small sample variation compared to the standard deviation.

**Table 3** Statistics on reproducibility of measurements of study subject.

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.1493</td>
</tr>
<tr>
<td>Standard Error</td>
<td>0.0332</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>0.1197</td>
</tr>
<tr>
<td>Sample Variance</td>
<td>0.0143</td>
</tr>
<tr>
<td>Confidence Level (95%)</td>
<td>0.0723</td>
</tr>
</tbody>
</table>

The results of the reproducibility test showed there was no statistical difference in the measurement procedure. Therefore, analysis of variance (ANOVA) was used on the data obtained from the control subjects and MS patients that participated in this study.

**Results**

The mean age of the control group was \( ? \) years ± S.D.? The mean age of the MS group was 48.2 years ± S.D.? There were \( n \) males and \( m \) females? in the control group. Similarly, there were \( n \) males and \( m \)? females in the MS group. All study subjects were of Caucasian ethnicity.
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After subtracting soft tissue movement for all subjects, Fig. 2 shows the results that were recorded for the cranial movements in the temporal region. The results of ANOVA of the data obtained from the subjects that participated in this study are summarized in Table 4.

**Figure 2** Results recorded for cranial movements in the temporal region. Distance is the change in the acoustic wavelength in millimeters with jaw clenching (ΔL) i.e. widening of the diameter at the intracranial surfaces of the temporal bones.

![Graph showing cranial movements](image)

**Table 4** ANOVA of data from Figure 2.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Count</th>
<th>Mean</th>
<th>SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>11</td>
<td>0.27</td>
<td>0.24</td>
<td>0.0008</td>
</tr>
<tr>
<td>MS</td>
<td>10</td>
<td>1.71</td>
<td>1.18</td>
<td></td>
</tr>
</tbody>
</table>
The results in Table 4 demonstrate a statistically significant difference in temporal bone movement, as measured by the PPLL, between the two groups (p < 0.001). In other words, as the subject clenched, the distance between the endocranial surfaces of the temporal bones increased. As the subject was clenching, the soft tissue distance was decreasing, so the resulting change in intracranial diameter (Echo 2 minus Echo 1) was greater, especially in the group with MS. The increase in magnitude of bi-temporal bone intracranial expansion was nearly six times greater in subjects with MS compared to controls.

Discussion

The pathogenesis of MS involves autoimmune driven breakdown of the myelin sheath surrounding the nerve fibres in the white matter. There are several ideas on the pathogenesis of MS. Some investigators believe that trauma may be an instigating factor in its development. Thus, brief, externally-derived forces might cause paroxysmal pressure spikes in the fluids surrounding the brain and spinal cord that could be acting as a traumatic factor. In addition, it is well documented that changes in intracranial pressure (ICP) lead to corresponding changes in cranial diameter (Heifetz and Wiess 1981, Heisey and Adams 1993). Furthermore, ICP changes in many neurodegenerative diseases manifest idiosyncratic phenomena, and are often accompanied by cellular disruptions that resemble elevated ICP conditions. For example, studies have shown that hydrocephalus may produce significant periventricular demyelination, probably as the result of mechanical stretching (reference required).

The cranium was once thought to be a rigid configuration of bone and ossified sutures. However, modern techniques have allowed critical evaluation of the compliance in the sutures. For example, Kokich (1986?) showed that the temporo-parietal (squamosal) suture does not begin to synostose until the 3rd – 4th decade in humans. Indeed, temporal bone movement (mean angle of change at the suture) is about 1.75°. Other measurements indicate that this
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magnitude of movement is common in most sutures in most crania (Oleski et al, 2002). Recent 3D radiographic imaging studies have demonstrated the presence of a malpositioned superior border of the temporal bone in patients with MS. (REFERENCE req’d). This finding lead us to hypothesize that periodic episodes of bruxism may be related to the development of lesions in and around the cranial ventricles as an inciting event in the development of MS. In this study, using a jaw clenching protocol and the PPLL, we aimed to establish a link between hypermobility of the temporal bones and jaw clenching/bruxism, reflecting increased intracranial pressure in the development of MS. In fact, we found an increase in magnitude of bi-temporal bone intracranial expansion nearly six times greater in subjects with MS compared to controls. Therefore, jaw clenching/bruxism is associated with more marked displacement of the temporal bones and expansion of the cranial cavity in patients with MS compared to control subjects.

It has been postulated that patients with MS exhibit the parafunctional abnormality of nighttime clenching/bruxism, which may cause increased ICP waves. These waves could lead to significant periventricular demyelination (3. Schulz M, 4. Poser CM, 5. Bunge R.P. et al.). Our original hypothesis was that bruxism caused an increased ICP, but we now suspect that marked expansion and contraction of the intracranial cavity by the hypermobile temporal bones might induce CSF pressure waves, as the intracranial distance between the temporal bones expands and contracts during bruxism, and precipitates MS in subjects with a genetic predisposition for the disease. There are several reports of elevated CSF PRESSURE IN patients with MS (20. Chebel and Reikik; 21 Newman and Slezer; 22 O’Brien and Paine; 23 Talbert; 24 Williams and Skinner). However, there are several limitations with this present preliminary study. First, the sample is small and not matched as thoroughly as we would like. Second, for this investigation, a constant speed of sound of 1540 ms⁻¹ in tissue was estimated, due to the non-homogeneity of tissues in the intracranial space. However, based on previous studies (9), the conversion error could be as large as 5-6%, as variations in intracranial distance are more significant than temperature- or pressure-dependent variations in the speed of sound that may also affect the target delay. Despite these constraints, we found much greater temporal bone displacement
with jaw clenching in patients with MS compared to control subjects. Such changes in intracranial diameter could generate high pressure intracranial waves, which could cause periventricular alterations in the blood-brain barrier (BBB) and promote demyelination, in subjects with a genetic susceptibility to the condition. Such alterations in the BBB could lead to lymphocytes entering the periventricular tissues to form ectopic lymph nodes (15. Serafini). Therefore, there is considerable interest in regulation of ICP, venous outflow, and the venous system (16. Gard).

If bruxism compromises normal outflow of blood from the brain in at-risk individuals, chronic cerebrospinal venous insufficiency might be exacerbated, leading to intracerebral iron deposition and inflammatory lesions (17. Zamboni, 18 Singh references). However, an alternative process might be a sudden reduction in the intracerebral pressure when the jaw clenching ceases followed by a sudden wave of increased pressure as the cranial bones deflect back to their original position. Moreover, the marked expansion of the cranial cavity could cause a drop in ICP until blood enters the cranial cavity acutely. In view of these preliminary findings, dental professionals must prevent, diagnose and treat bruxism/clenching as a preventive measure in the putative development of MS. In future studies should endeavor to clearly establish a temporal relationship between the onset of MS and bruxism, and seek to identify a causal relation between the presence of MS and bruxism. In addition, as patients with MS frequently suffer marked fatigability, studies with polysomnography to investigate nocturnal bruxism in patients with MS are warranted.

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Luna Innovations

Disclosure
Luna Innovations
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See Author Guidelines, References, for format


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20. “chebel s AND rekik o”;

21. “newman nj AND slezer ka”;

22. “obrien t AND paine m”;
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23. “talbert dg AND multiple sclerosis;

24. “williams bj AND skinner hj AND multiple sclerosis”