Bruxism and Temporal Bone Hypermobility In Patients with Multiple Sclerosis

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ABSTRACT: In this study, the authors investigated the link between jaw clenching/bruxism and temporal bone movement associated with multiple sclerosis (MS). Twenty-one 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 100 lbs was used to measure the mean change in acoustic wavelength (ΔL) as the measure of intracranial distance. In the control subjects the mean ΔL was 0.27 mm±0.24. In subjects with MS the mean ΔL was 1.71 mm±1.18 ($p<0.001$). The increase in magnitude of bi-temporal bone intracranial expansion was approximately 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 in control subjects.

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Multiple sclerosis (MS) is the major cause of nontraumatic neurological disability in young adults in North America.[1] 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] This study led researchers to investigate other dental factors associated with MS. Unpublished, three dimensional (3D) radiographic imaging studies have demonstrated the presence of a malpositioned superior border of the temporal bone in patients with MS, and evidence is emerging of a shifting of the squamosal suture during sustained, maximal jaw clenching in those patients. Studies of jaw clenching have demonstrated pressures of 975 psi at the molars and as high as 175,000 psi at the incisors.[3] 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,[4] and TMJ health may be important

in overall cranial health. Moreover, modern radiographic techniques have allowed critical evaluations of compliance in the cranial sutures, suggesting that cranial mobility is detectable. With external manipulation of the cranial vault, temporal bone movement (the mean angle of change at the squamosal suture) is about 1.75°. For example, the mastoid process moves by 1.66°, the malar line moves by 1.25° and the sphenoid bone moves by 2.4°. Other measurements indicate that this amount of movement is common in most sutures.[5]

It is thought that changes in intra-cranial pressure (ICP) lead to corresponding changes in intra-cranial diameter.[6-7] These changes can be measured using a pulsed phase locked loop (PPLL) device[8] (**Figure 1**). The PPLL device originally was used to measure pulsatile changes in ICP.[9-10] The PPLL device transmits a 500 kHz 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 (**Figure 2**). Any change in cranial diameter produces a phase shift in the ultrasound signal.[9-10] 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 (ΔL, change in acoustic wavelength).[8] The resulting

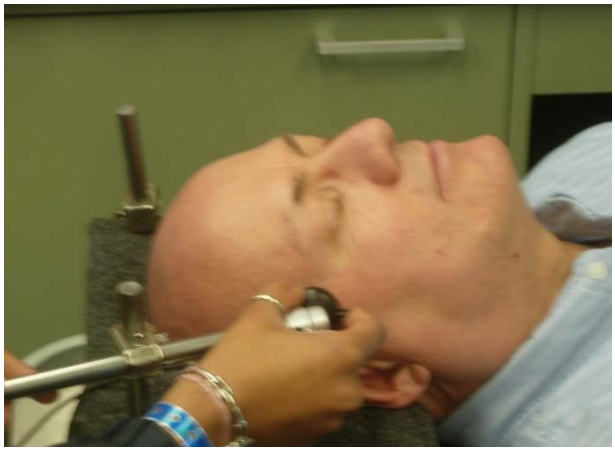

Figure 2
Transducer fixation system for tests conducted with the transducer placed over the temporal lobe.

target delay estimates are then converted into a distance measuring the intracranial distance using the equation:

$$d = 1/2v*t$$

where *t* is the target delay estimate and v 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, ΔL).

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.[11] Thus, an abnormal bite relationship may exert unequal pressure

Figure 1
The pulsed phase locked loop (PPLL) device used to measure changes in intra-cranial pressure.and corresponding changes in intra-cranial diameter.

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 may, under certain circumstances, cause increased cranial suture mobility. This mobility could result in pressure changes inside the cranium, which might in turn alter CSF flow, venous flow, or neurologic tissues directly. Therefore, the aim of this 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

The UCSD Human Research Protections Program approved this study. 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; and 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. The mean age of the control group was 44.9 years. The mean age of the MS group was 48.2 years. There were approximately the same number of males and females in the control and MS groups. All study subjects were of Caucasian ethnicity.

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, Blacksburg, VA). The transducer's position was adjusted manually, and the head was rigidly fixed in a frame. 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 (**Figure 3**). Once adjusted, a maximum strength test was conducted to measure the maximum clenching strength of the subject, using a dental bite I-scan sensor (Tekscan Inc., South Boston, MA). The subjects bit down on the sensor that was a Mylar sheet with pressure sensitive ink between metal tracings. The sensors have double layers of thin rubber protectors to dissipate the forces and prevent perforation. The subjects clenched as hard as they could for one second 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. Later the subjects were able to judge their strength and maintain a 100 lb clench-force for this test. The signal was determined with the muscles at rest. The signal was checked again and data were acquired for 20 seconds, while the subject underwent the jaw clenching procedure, as described in **Table 1**. The change in intracranial temporal bone diameter was expressed as ΔL (change in acoustic wavelength), which

Figure 3
Echoes tracked for the jaw clenching tests using PPLL software.

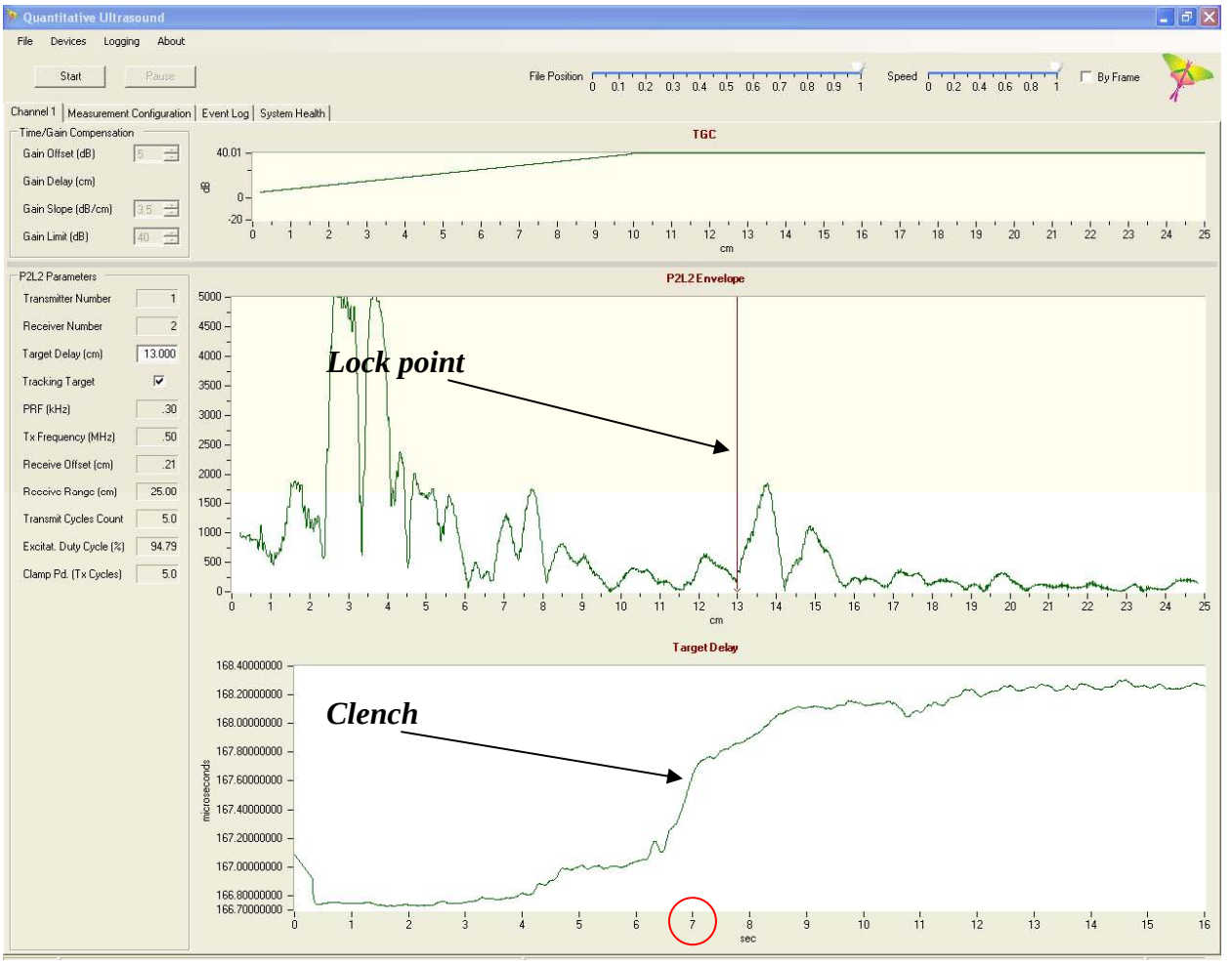

Figure 4
Locking on back surface reflection

was converted into intracranial distance in mm (using the aforementioned equation).

To ensure that the subject was clenching at approximately 100 lbs, the subject watched a video monitor, which showed clenching force. This force was detected by a load cell developed by T-scan (Tekscan Inc., South Boston, MA). 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 was 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 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, it was possible 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 (derived from the change in acoustic wavelength, 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

Table 1
Jaw Clenching Procedure Performed by Each Subject

Time (sec)	Action
0-5	Relaxed
5-10	Clenching begins, gradually increasing the force until reaching 100 lbs
10-15	Maintain clench at 100 lbs
15-20	Clench is released

data were reprocessed, this time *locked* on the echo from the proximal wall (Echo 1). By subtracting the second result (Echo 2) from the first, the authors 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**.

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 who participated in this study.

Results

Figures 4 and **5** show the PPLL locked at the back surface and front surface of a patient with MS, and the changes in target delay can be seen at these points. With the PPLL locked on the back surface, the PPLL tracks changes in the distance between the transducer and the distal skull wall. To subtract out soft tissue movement between the transducer and the proximal skull wall, the saved data was reprocessed, this time locked on the echo from the proximal skull wall, as shown in **Figure 5**. By subtracting the second result from the first, the authors are able to monitor changes in the distance between the ipsilateral and contralateral temporal bones during clenching.

As can be seen in **Figure 4**, as the subject clenched, the distance between skull plates increased. **Figure 5** shows that, as the subject was clenching, the soft tissue distance was actually decreasing, so the resulting change in skull was actually greater than that shown in **Figure 4**.

After subtracting soft tissue movement for all subjects, **Figure 4** 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 who participated in this study are summarized in **Table 4**.

Figure 6 shows the results recorded for cranial movements in the temporal region: Distance is the change in intracranial distance (derived from the change in the acoustic wavelength, ΔL) in mm with jaw clenching, i.e., widening of the diameter at the intracranial surfaces of the temporal bones.

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 diame-

Table 2
Reproducibility Tests on Subjects Chosen at Random*

Test number	ΔL (mm)
1	0.385
2	0.077
3	0
4	0.385
5	0.154
6	0.1386
7	0.077
8	0.1078
9	0.0924
10	0.1694
11	0.231
12	0.0462
13	0.077

*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 Subjects

	Value
Mean	0.1493
Standard error	0.0332
Standard deviation	0.1197
Sample variance	0.0143
Confidence level (95%)	0.0723

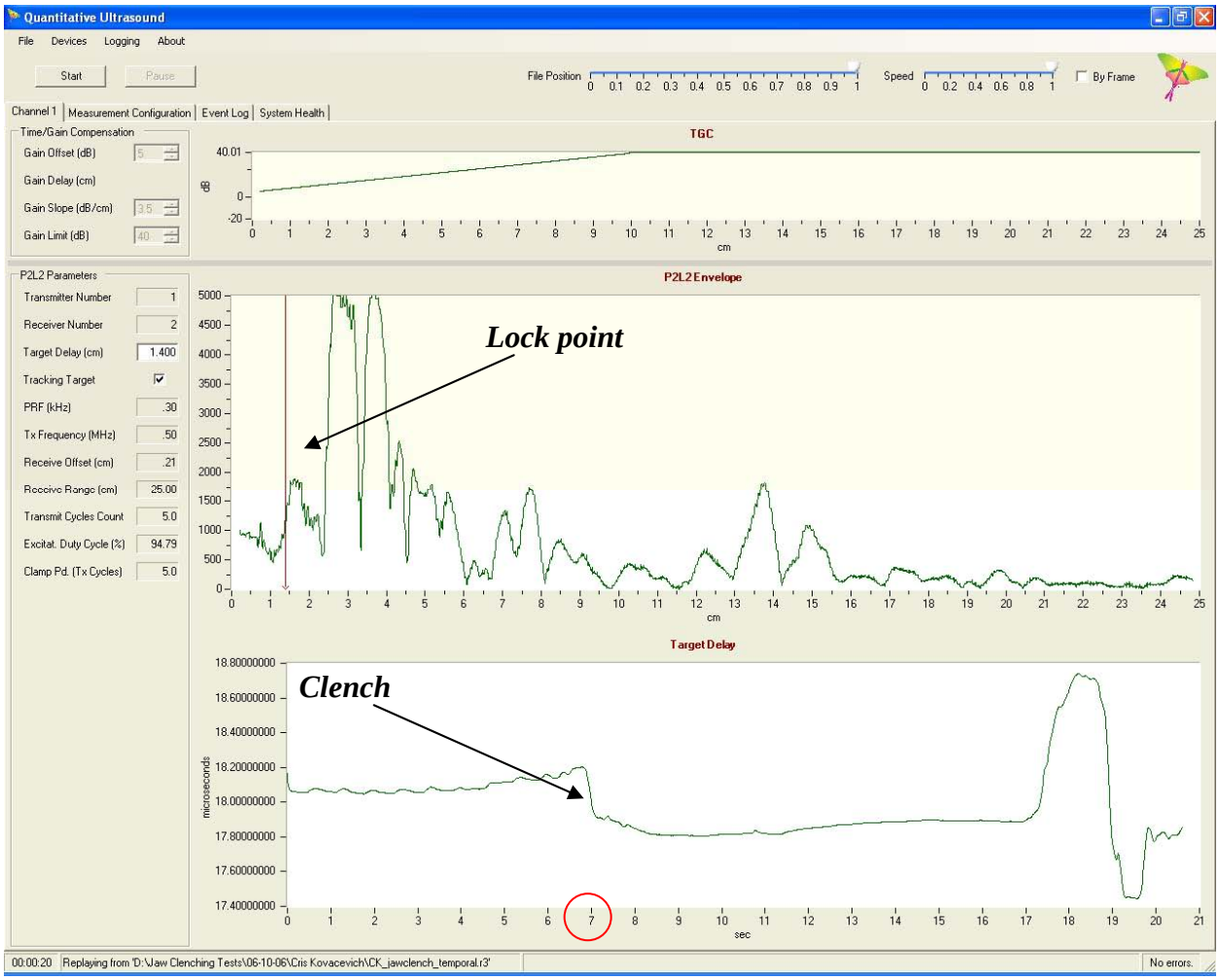

Figure 5
Locking on soft tissue reflection.

ter (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 fibers in the white matter.[12] There are several ideas on the patho-etiology of MS. A significant relationship between decreased vitamin D levels in patients with MS is documented.[13-14] Vitamin D deficiency could lead to reduced bone density in patients with MS, which, in turn, could lead to greater cranial compliance along the layered bone[15] and sutures.[16] Indeed, some investigators believe that trauma may be an instigating factor[17] in its development. Thus, briefly, externally derived forces might cause paroxysmal pressure spikes in the fluids surrounding the brain and spinal cord that could act as a traumatic factor. In addition, it is well documented that changes in intracranial pressure (ICP) lead to corresponding changes in cranial diameter.[6-7] 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.[18]

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[19] showed that the temporo-parietal (squamosal) suture does not begin to

Table 4
ANOVA of Data from Figure 6

Groups	Number	**Mean**	SD	*p* value
Control	11	**0.27**	0.24	0.0008
MS	10	**1.71**	1.18	

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 magnitude of movement is common in most sutures in most crania.[5] Indeed our current, unpublished 3D radiographic imaging studies demonstrate the presence of a malpositioned superior border of the temporal bone in patients with MS. This observation led the authors to hypothesize that periodic episodes of bruxism may be associated with increased intracranial pressure, which in turn, might be associated with demyelination in patients with MS. In this study, using a jaw clenching protocol and the PPLL, the aim was 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, it was found that an 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 compared to control subjects. Nevertheless, it must still be determined whether clenching/bruxism leads to temporal bone hypermobility in patients with MS or whether it is a latent sign of the disease.

It has been postulated that patients with MS exhibit the parafunctional abnormality of night-time clenching/bruxism, which may cause increased ICP waves. These waves could lead to significant periventricular demyelination.[12,20-21] 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 pressure waves. As the intracranial distance between the temporal bones expands and contracts during bruxism, the compression may decrease cranial cavity volume with a corresponding increase in ICP pressure, at least in principle, and may precipitate MS in subjects with a genetic predisposition for the disease. There are several reports of elevated CSF pressure in patients with MS.[17,22-25] However, there are several limitations with this present preliminary

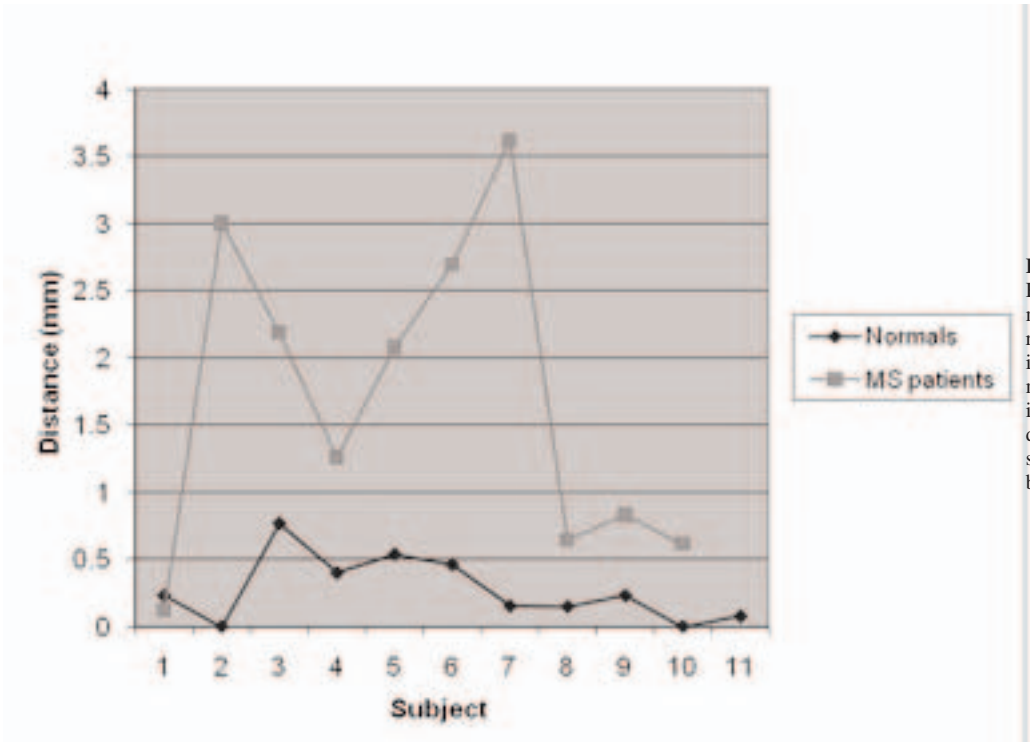

Figure 6
Results recorded for cranial movements in the temporal region. Distance is the change in the acoustic wavelength in millimeters with jaw clenching (ΔL), i.e., widening of the diameter at the intracranial surfaces of the temporal bones.

study. First, the sample is small and not matched as thoroughly as is optimal. Second, for this investigation, a constant speed of sound of 1540 ms-1 in tissue was estimated, due to the nonhomogeneity of tissues in the intracranial space. However, based on previous studies,[10] 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, much greater temporal bone displacement was found 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.[26] Therefore, there is considerable interest in regulation of ICP, venous outflow, and the venous system.[27]

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.[28-29] However, an alternative process might be a sudden reduction in the ICP 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, it is suggested that dental professionals evaluate the need to prevent, diagnose and treat bruxism/clenching as a preventive measure in the putative development of MS. Future studies should endeavor to investigate the existence of 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 and evaluate compromised airway space in patients with MS are warranted.

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