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ORIGINAL ARTICLE

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Parkinson's disease, temporomandibular disorders and bruxism: A pilot study

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Summary

Background: Even though bruxism and Parkinson's disease (PD) share common characteristics, their relation is still not clear. Both bruxism and PD are movement disorders in addition, patients with bruxism as well as those with PD complain about musculoskeletal pain, including temporomandibular disorders (TMD) pain.

Objectives: Therefore, the aim of this pilot study was to gain more insight into the possible relation between bruxism and TMD on one hand and PD on the other.

Methods: In total, 801 persons gave their written informed consent and agreed to participate in the study filling in a questionnaire. Complete data were collected from 708 persons (368 with PD or Parkinsonism [PR] and 340 controls) and were included in the analysis. The questionnaire included the graded chronic pain scale, the DC/TMD oral behaviour checklist, the DC/TMD symptom questionnaire and the TMD pain screener. In addition, a question about self-reported tooth wear was included. The chi-square test and independent samples *t* test were used for the data analysis.

Results: Patients with PD/PR reported significantly more often bruxism during sleep and wakefulness than controls. Also, patients with PD/PR had more often possible TMD and reported a significantly higher mean pain intensity in the orofacial region than controls. There was no significant difference in complaints of jaw locking between the patient group and the control group. A tendency towards a significant association was found between PD/PR and tooth wear.

Conclusion: There is a relation between PD/PR and bruxism. Furthermore, a relation of PD/PR with TMD pain is suggested to be present.

KEYWORDS

bruxism, facial pain, Parkinson disease, pilot project, questionnaire, temporomandibular joint disorders

1 | INTRODUCTION

In the near future, human's life expectancy is estimated to increase significantly: in 1950 only 8% of the population was above 65 years old, while in 2040 this prevalence is estimated to become triple reaching almost 26%.[1] Additionally, in the Netherlands, the actual number of inhabitants is also expected to increase with more than 7.5% amongst others because of the improvement of the health care system.[1]

This estimated ageing of the population is expected to cause an increase in the prevalence of age-related diseases like the

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neurodegenerative diseases. Neurodegenerative diseases, such as Alzheimer's, Huntington's and Parkinson's disease (PD), affect neurons in the central nervous system and especially in the brain. Currently, there is no treatment to cure them and therefore they can progress in the long term, causing movement disorders and problems affecting the patients' mental state.[2]

Parkinson's disease is a movement disorder that causes serious impairment in patients' life. The prevalence of PD in 2007 in the Netherlands was estimated between 201 and 372 per 100.000 persons. Statistics Netherlands (CBS) predicts an increase in the prevalence of about 40% in 2025.[2] Even though the exact pathophysiological mechanism of PD is not fully understood, it is considered to be caused by a deficiency in the dopamine levels due to degeneration of neurons in the substantia nigra. In PD, as a result of the reduced dopamine levels in the striatum, fluent movements of the human body are disturbed.[3]

The same symptoms as PD can also be present in patients suffering from other diseases in which the dopamine producing cells are affected. These patients are considered to suffer from Parkinsonism (PR), which is a general term that reflects the characteristic symptoms of PD.

Until now, the prevalence of oromandibular movement disorders in patients with PD/PR is not yet studied. One of the most common oromandibular movement disorders in humans is bruxism. Bruxism is defined as a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible and has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism).[4] Its prevalence in adults can vary between 8% and 31.4%, depending on factors like the methodology and the population studied.[5] Bruxism is considered one of the important risk factors for the initiation and perpetuation of temporomandibular disorders (TMD).[6]

Temporomandibular disorders is a collective term embracing disorders of the masticatory muscles, the temporomandibular joint and adjacent structures.[7] Painful TMD is considered the second most common cause of orofacial pain after dental pain.[8] The prevalence of painful TMD is about 10% in adults.[9] Other symptoms of TMD include limitations in the movement of the mandible (either while opening or during closing of the mouth), joint sounds and headache attributed to TMD.[10] In the aetiology of TMD, oral parafunctions like bruxism are considered to play an important role.[6]

Although the possible relation between bruxism and PD has not been studied yet, some suggestions for the existence of such relationship can be derived from the literature as mentioned earlier, PD and bruxism are both movement disorders. Moreover, bruxism is considered to be regulated centrally and not peripherally, with an important role of the dopaminergic nervous network of the brain.[11] PD is also a disease related to a shortage of dopamine. This can cause uncontrolled movements in the orofacial region as well.[3] Furthermore, specific antidepressants (like Selective Serotonin Reuptake Inhibitors [SSRI's]), which have bruxism as a possible side effect, work through inhibition of specific dopaminergic neurons. Because of the reduction of dopamine, the possibility of developing uncontrolled movements like bruxism increases.[12] In addition, in about 9% of the cases, depression already exists in PD patients before the actual diagnosis of PD is established.[2] Stress and depressed feelings are also risk factors for awake bruxism.[6] Furthermore, a case report showed a patient with a basal ganglia infarct who developed bruxism. This report concluded that there could be a connection between bruxism and a dysfunction of the dopaminergic pathway.[13]

Even less information is available about the possible relation between PD and TMD. A possible link is that besides restrictions in movements, also sensory aspects play a role in both PD as TMD. PD is characterised by the regular presence of pain.[14] The prevalence of pain as a symptom of PD is approximately 30%-50%. In 45%-74% of these cases, the pain was classified as musculoskeletal pain.[15] Bruxism itself is not painful, but it can be an important risk factor for developing TMD-pain complaints. It is also known that in TMD, orofacial pain is one of the cardinal symptoms. Besides, pain elsewhere in the body is a risk factor for having orofacial pain.[16] Finally, in another study a significantly reduced D1/D2 ratio and an increased amount of D2 receptors was found in patients with atypical facial pain, which suggests that alterations in the striatal dopaminergic system could influence chronic orofacial pain conditions.[17]

As mentioned above, little is known so far about a possible relation between PD on one hand and bruxism and TMD on the other. Although some theories currently exist in the literature, no definite conclusion has been drawn and a causal relationship has not been proven. Within these premises, the aim of the present pilot study was to gain more insight into a possible relation between bruxism and TMD on one hand and PD/PR on the other.

2 | METHODS

Patients with a diagnosis of PD or PR were asked to participate in the study (patient group). Patients suffering from other movement disorders than PD or PR were excluded. Partners, caregivers, friends and non-related persons with a maximum of 5 years of age difference were asked also to participate (control group). Relatives of the patients and persons with more than 5 years of age difference were excluded from the control group. Written informed consent was obtained from each participant. The study was independently reviewed and approved by the Medical Ethics Committee of the VU Medical Centre, Amsterdam, The Netherlands (file no. 2015.461; approval date November 26th, 2015).

2.1 | Questionnaire

Data were collected with the use of a questionnaire (Appendix 1) that was composed by the authors with questions taken from

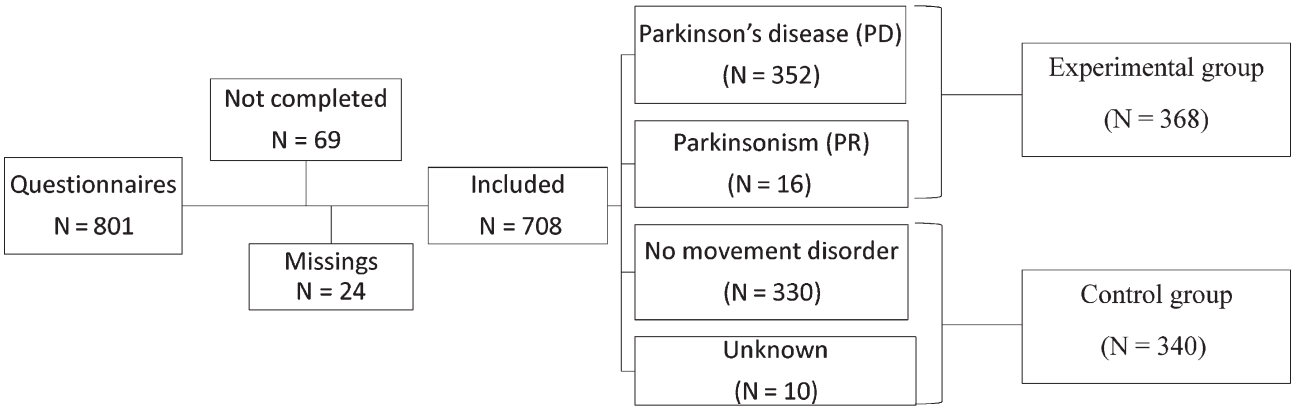

FIGURE 1 Flowchart participants

already existing instruments (Appendix 1). There are 18 questions: about demographics (gender and age; two questions), research location (one question), diagnosis of PD/PR and the possible use of medication (three questions), and bruxism and related problems like TMD/orofacial pain and presence of tooth wear (12 questions). Except for the question about tooth wear, which was taken from the intake questionnaire of the Clinic of Orofacial Pain and Dysfunction of ACTA, the latter questions were selected from the graded chronic pain scale,[18] the DC/TMD oral behaviour checklist,[19,20] the DC/TMD symptom questionnaire[10] and the TMD-pain screener.[21]

When the participants answered positive to the question 7 (Appendix 1), they were considered having "Orofacial Pain." When the score of the TMD-pain screener was equal or above 4, participants were considered having "Possible TMD-pain." Moreover, characteristic pain intensity (CPI) was calculated based on the analysis of the data of the graded chronic pain scale. When a person reported clenching, grinding or bracing the mandible during sleep or awakening at least "1-3 nights a month" or "Some of the time," this was interpreted as presence of sleep or awake bruxism.[4,22]

2.2 | Data collection

The data were collected from February 8th, 2016, until February 8th, 2017 through visiting unofficial gatherings of PD/PR patients organised by the Dutch Association of PD patients (Parkinson cafés), hospitals, or individual nurses. In addition, advertisement took place through social media, viz., through Facebook and on the website of the Dutch association of PD patients. The advertisement was placed on March 1st, 2016. In order to obtain the same amount of responses regarding the control group, people from several public places, like Schiphol Airport, were also asked to fill in the questionnaire.

2.3 | Data analysis and statistics

Descriptive data were calculated for all variables. Subsequently, Chi-square test was used to test possible association between the presence of PD/PR and the following variables: orofacial pain, possible TMD-pain, possible sleep or awake bruxism, jaw locking, and tooth wear. Differences between the patient group and the control group

TABLE 1 Descriptive statistics of all included participants (PD/PR and control group)

Total participants (N = 708)			**Controls (N = 340)**		
Mean ± SD age (y)		67 ± 9.25	Mean ± SD age (y)		65 ± 9.25
Gender	Men	344 (49%)	Diagnosis	No PD/PR	330 (97%)
	Woman	364 (51%)		Unknown	10 (3%)
Questionnaire	Paper	526 (26%)	Gender	Men	125 (37%)
	Internet	182 (74%)		Women	215 (63%)
Data source	Parkinson Café	420 (59%)	**Patients (N = 368)**		
	Regional hospital	12 (2%)	Mean ± SD age (y)		68 ± 8.54
	The Dutch association of PD patients	65 (9%)	Diagnosis	PD	352 (96%)
	Social media	21 (3%)		PR	16 (4%)
	Otherwise	190 (27%)	Gender	Men	219 (60%)
				Woman	149 (40%)

PD, Parkinson's disease; PR, Parkinsonism.

TABLE 2 Statistical results of the chi-square tests (level of significance: $P < 0.005$)

	Parkinson's disease (PD)/ Parkinsonism (PR)	
	X^2	*P*-value
Orofacial pain	6.304	0.012
TMD pain	17.988	<0.001
Sleep bruxism	10.296	0.001
Awake bruxism	14.864	<0.001
Jaw locking	0.544	0.461
Tooth wear	14.864	0.056

TMD, temporomandibular disorders.

TABLE 3 Statistical results regarding the characteristic pain intensity (CPI; independent sample *t* test; level of significance: $P < 0.005$)

	Characteristic pain intensity		
	Mean	SD	SE
Controls	35.51	53.40	6.43
PD/PR	80.00	76.32	7.59
	$t(167.89) = -4.472$, $P < 0.001$		

PD, Parkinson's disease; PR, Parkinsonism.

were tested with independent sample *t* test for the CPI. Statistical analyses were performed with IBM SPSS statistics, version 24. The level of significance was set at $\alpha < 0.05$.

3 | RESULTS

Eight hundred and one questionnaires were filled in. Data from 93 persons were excluded from the analysis, because the questionnaires were either not completed (n = 69) or contained missing values (n = 24; Figure 1). The demographics and distribution of the patient and control group are shown in Table 1. There was no significant difference between the excluded and included group on the basis of gender and diagnosis. A significant association between PD/PR and orofacial pain (Table 2) was found. PD/PR patients reported pain more frequently than the control group. Further detailed analysis among the patients who reported orofacial pain (N = 178) showed a significant association between PD/PR and possible TMD-pain (Table 2). Regarding the CPI, significantly higher values were found for the patient group in comparison with the control group (Table 3). A significant association was also found between PD/PR and bruxism and at the same time for possible sleep bruxism and possible wake bruxism separately (Table 2).

No association between jaw locking and PD/PR was present (Table 2).

Finally, a tendency towards a significant association between PD/PR and tooth wear was found (Table 2). Patients with PD/PR reported significantly more often "much tooth wear" compared to the control group (Table 4).

4 | DISCUSSION

The aim of this pilot study was to gain more insight into a possible relation between bruxism and orofacial pain or TMD-pain on the one hand and PD/PR on the other. The results showed a significant relation between possible sleep bruxism and PD/PR, and also between possible awake bruxism and PD/PR. Moreover, a significant association was found between orofacial pain, possible TMD-pain and PD/PR.

In general, establishing the diagnosis of PD is a difficult procedure. Only post-mortem examination can provide 100% certainty. In this study, the clinical diagnosis of PD/PR was used, as it was set by the specialised medical practitioner and was self-reported by the patients. The specificity and sensitivity of the diagnostic accuracy of PD from a general neurologist is 57.8% and 89.2%, respectively.[23] It was not realistic to follow all patients over time in order to confirm the diagnosis through post-mortem examination. There is therefore a slight possibility that people were included in the patient group in whom this diagnosis would not be verified after death. In addition, a number of researchers have reported that there is a preclinical phase of PD in which a diagnosis cannot yet be made, and that PD has several different stages during the progression of the disease.[24] A classification for this progression is described in the literature.[25] Consequently, this would mean that

TABLE 4 Statistical results regarding tooth wear (adjusted residuals; chi-square test; level of significance: $P < 0.005$)

	Tooth wear (adjusted residuals)						
	Not	Little	Some	Much	A lot	Don't know	Total
No (controls)	162	98	29	8	9	34	340
Adjusted residuals	0.5	1.5	1	**−2.2**	−1	−1.6	
Yes (PD/PR)	169	88	24	21	15	51	368
Adjusted residuals	−0.5	−1.5	−1	**2.2**	1	1.6	
Total	331	186	53	29	24	85	708

PD, Parkinson's disease; PR, Parkinsonism.
Wherein PD/PR patients compared to controls showed a tendency towards a significant association in 'much' tooth wear signifies bold.

in the control group some people could have had one of the preliminary PD stadia that could not be easily diagnosed. Regardless of this possibility, significant associations with bruxism and TMD-pain were found for the PD/PR group. In future research, it would possibly be more accurate to collaborate directly with a neurologist in order to analyse in which stage the patient is, and if bruxism is associated with PD at specific stages.

In this pilot study, a questionnaire was used for the data collection. This questionnaire was distributed mainly in Parkinson Cafés. Therefore, not the researchers themselves but the individuals organising the meetings were responsible for introducing the research to the participants, thereby potentially introducing bias or confounders. Moreover, nine of the Parkinson Cafés did not respond and 10 Parkinson Cafés did not want to cooperate, which may have reduced the generalisability of the findings of this study. In addition, the questionnaire was designed in a way to gather as much information as possible with the least possible burden for the participants. Nevertheless, some patients experienced difficulties to complete the questionnaire: 69 people did not fill in the questionnaire at all, 19 of whom filled only the first two questions and 15 of whom stopped at the question about medication. These missing data may also have led to a reduction of the generalisability of the present findings.

Unfortunately, there was no question about dentures in the questionnaire. Many patients pointed out that they were wearing a denture. This information could have had an influence on the answers to the question about tooth wear. Most people do not know that also dentures can show wear, and that bruxism is also possible in the absence of teeth. Therefore, it is possible that the answers underestimated the presence of bruxism and wear in the group of people with dentures.

The authors expected that people with PD had higher amounts of tooth wear than the control subjects. In practice, the validity of the self-reported question about wear is a matter of debate. This could possibly explain that only a tendency towards a significant association could be found between PD/PR and tooth wear. In further research, it is therefore more valuable to examine patients with the use of the dental wear screener[26] in order to have clinical data regarding tooth wear.

In an experimental model in monkeys, an association was found between PD and jaw movements.[27] The reported changes in range, velocity and pattern of jaw movements were confirmed in another study.[28] Because of the study design of the present research, only information about jaw locking was present; not about other deviant jaw movements. For future research, the authors suggest to use this determinant as well. Finally, bruxism and TMD was only based on self-report data. Although there is a possibility that TMD is related to bruxism, the relation between both conditions is not linear.[6] It is therefore not possible to assume a direct relation between bruxism, TMD, and PD/PR.

An earlier study[29] reported a higher prevalence of bruxism in patients with PD, but no significant difference with a control group was observed. Furthermore, another study[30]concluded that awake bruxism is rare, but when it is present the association with neurological diseases is more frequently made. In the present study, we did find a relation between possible sleep and awake bruxism and the diagnosis of PD/PR. These results are in agreement with the study of Tae Kwak et al.[30] Also, in the present study, we did find a higher prevalence of bruxism in patients with PD. However, we did find a significant relation between the two groups. This can be due to the large power of our study.

In the current literature, there were also some contradictions present. The highest prevalence of bruxism is described around the age of 45 years.[5] In the present study though, the average age was much higher, viz., 67 years. It is therefore possible that because of the retrospective design of this study, people did not remember experiencing bruxism-related problems before. Therefore, it could be true that patients and controls noted less parafunctions and pain than they actually suffered from. However, this effect most likely occurs equally in controls and patients with PD/PR. Moreover, earlier studies described that a short-term use of L-dopa can result in a reduction of sleep bruxism.[31] Although the effect of the long-term use of L-dopa is not yet clear, the researchers suggested that there may be a relation between the increase in bruxism a long-term use of L-dopa. A case study describing a patient who developed bruxism as a result of levodopa therapy supports these suggestions.[32] In this study, there is the possibility that because 99.4% of the people with PD/PR used levodopa, with or without combination therapy, the results could be influenced. Also, a side effect from dopaminergic medication is dyskinesia (a condition that is, amongst others, characterised by choreatic movements) and it is therefore possible that if present in the orofacial region, dyskinesia was identified as possible bruxism. Because the population in this study was not always very accurate at filling in the questions about medication, it was not possible to verify these results and to assess if the use of levodopa or other dopaminergic medication influenced the bruxism behaviour. Additional clinical research with more objective lists of medication would increase our insight in this possible association. Finally, another study concluded that self-reported bruxism has a low sensitivity (62%) and specificity (50.8%)[33] because of the fact that people are often not aware of their behaviour. Also clenching does not produce sounds and report of muscle fatigue at awakening is not a good indicator for sleep bruxism. This could explain why only few people reported sleep bruxism.

Regarding pain intensity, a number of studies, mainly in animals, have reported higher values in PD patients.[15,34] In the present study, a relation was found between the CPI of orofacial pain and PD/PR, where patients with PD/PR noted a higher mean of pain intensity than the control group. In the literature, no clear relation was found between oral parafunctions like bruxism and orofacial pain. In this study, a significant difference in the mean value of pain intensity was found: people who did not report bruxism behaviour had a significantly lower mean in pain intensity than people who reported bruxism behaviour, which is in accordance with other reports.[20]

Clinical history, physical examination and the reaction to medication is nowadays still the main approach in order to set the diagnosis of PD. With biomarkers and Magnetic Resonance Imaging (MRI), it is possible to distinguish between PD and other neurodegenerative

diseases. However, it is still not possible to see whether PD is the correct diagnosis with these additional tests. When a relation with PD and bruxism is proven, bruxism could be of diagnostic value. The main reason is that dentists see their patients more frequently than the General medical practitioner (GP). So, it is possible that an earlier referral from the dentist to the GP or neurologist could be made.

For future research, a more clinical approach is recommended, where a practitioner could examine patients for a more definite diagnosis of bruxism, TMD, or for example if factors like tooth wear are present. Specifically, a diagnosis according to the Diagnostic Criteria for TMDs[10] would yield more possibilities to compare different research projects at an international level. Also, collaboration with a specialised neurologist is an advantage to determine the progression of the disease and the medication used. Finally, a longitudinal study would be suggested in order to give a definite answer to the question whether in selected cases, bruxism and PD have a causal relation. Eventually, our goal is to see whether bruxism could be a prodrome for developing PD.

5 | CONCLUSIONS

The current findings suggest that there is a relation between PD/PR and bruxism behaviour. Furthermore, in the population studied, a relation of PD/PR and TMD-pain is also present.

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DISCLOSURE

The study protocol was reviewed from the medical ethics committee of the Free University Medical Centre (Vrij Universiteit Medisch Centrum-VUmc) and was considered not being covered by the Act Medical Scientific Research in Humans according to the Dutch legislation for medical research in humans (Wet medisch-wetenschapelijk onderzoek-WMO). This decision was made because of the cross-sectional study design and the use of the short questionnaire. As such, the study protocol was accepted by the medical ethics committee of the VUmc, who gave a non-WMO approval-statement (file no. 2015.461, approval date November 26th, 2015). An extra permission was given for an amendment (file no. A2016.459, approval date December 14th, 2016), in order to further expand our control group with participants recruited in public locations. Dr. Lobbezoo reports Grants and Other from Sunstar Suisse, grants from Somnomed, outside the submitted work. Dr. Aarab reports grants from SomnoMed, grants from Braebon Inc, grants from KNAW (The Royal Netherlands Academy of Arts and Sciences), grants from The Dutch Society of Gnathology and Prosthetic Dentistry, grants from American Equilibration Society, outside the submitted work. The other authors have stated explicitly that there are no conflicts of interest in connection with this article.

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REFERENCES

1. Ritsema van Eck J, van Dam F, de Groot C, de Jong A. *Demografische Ontwikkelingen 2010-2040. Ruimtelijke Effecten en Regionale Diversiteit*. Den Haag, The Netherlands: Planbureau voor de Leefomgeving; 2013.
2. Bloem BR, Van Laar T, Keus SHJ, et al. *Multidisciplinaire Richtlijn 'Ziekte van Parkinson'*. Alphen a/d Rijn, The Netherlands: Van Zuiden Communications; 2010.
3. Wolters EC, Van Laar T, Visser DM. *Bewegingsstoornissen*. Amsterdam, The Netherlands: VU Uitgeverij; 2002.
4. Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil*. 2013;40(1):2-4.
5. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain*. 2013;27(2):99-110.
6. Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2010;109(6):e26-e50.
7. de Leeuw R, Klasser GD. *Orofacial Pain. Guidelines for Assessment, Diagnosis, and Management*. Chicago, IL: Quintessence Publishing Co, Inc; 2013.
8. Durham J, Newton-John TR, Zakrzewska JM. Temporomandibular disorders. *BMJ*. 2015;350:h1154.
9. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. *Crit Rev Oral Biol Med*. 1997;8(3):291-305.
10. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Groupdagger. *J Oral Facial Pain Headache*. 2014;28(1):6-27.
11. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil*. 2001;28(12):1085-1091.
12. Beers E, Van Grootheest AC. Bruxisme als bijwerking van serotonineheropnameremmers. *Ned Tijdschr Tandheelkd*. 2007;114:388-390.
13. Tan EK, Chan LL, Chang HM. Severe bruxism following basal ganglia infarcts: insights into pathophysiology. *J Neurol Sci*. 2004;217(2):229-232.
14. Khoo TK, Yarnall AJ, Duncan GW, et al. The spectrum of nonmotor symptoms in early Parkinson disease. *Neurology*. 2013;80(3):276-281.
15. Fil A, Cano-de-la-Cuerda R, Munoz-Hellin E, Vela L, Ramiro-Gonzalez M, Fernandez-de-Las-Penas C. Pain in Parkinson disease: a review of the literature. *Parkinsonism Relat Disord*. 2013;19(3):285-294; discussion
16. Svensson P, Kumar A. Assessment of risk factors for oro-facial pain and recent developments in classification: implications for management. *J Oral Rehabil*. 2016;43(12):977-989.
17. Hagelberg N, Forssell H, Aalto S, et al. Altered dopamine D2 receptor binding in atypical facial pain. *Pain*. 2003;106(1–2):43-48.

18. Von Korff M, Ormel J, Keefe FJ, Dworkin SF. Grading the severity of chronic pain. *Pain*. 1992;50(2):133-149.
19. Markiewicz MR, Ohrbach R, McCall Jr WD. Oral behaviors checklist: reliability of performance in targeted waking-state behaviors. *J Orofac Pain*. 2006;20(4):306-316.
20. van der Meulen MJ, Lobbezoo F, Aartman IH, Naeije M. Validity of the Oral Behaviours Checklist: correlations between OBC scores and intensity of facial pain. *J Oral Rehabil*. 2014;41(2):115-121.
21. Gonzalez YM, Schiffman E, Gordon SM, et al. Development of a brief and effective temporomandibular disorder pain screening questionnaire: reliability and validity. *J Am Dent Assoc*. 2011;142(10):1183-1191.
22. Klasser GD, Rei N, Lavigne GJ. Sleep bruxism etiology: the evolution of a changing paradigm. *J Can Dent Assoc*. 2015;81:f2.
23. Joutsa J, Gardberg M, Roytta M, Kaasinen V. Diagnostic accuracy of parkinsonism syndromes by general neurologists. *Parkinsonism Relat Disord*. 2014;20(8):840-844.
24. Boeve BF. Idiopathic REM sleep behaviour disorder in the development of Parkinson's disease. *Lancet Neurol*. 2013;12(5):469-482.
25. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology*. 1967;17(5):427-442.
26. Wetselaar P, Lobbezoo F. The tooth wear evaluation system: a modular clinical guideline for the diagnosis and management planning of worn dentitions. *J Oral Rehabil*. 2016;43(1):69-80.
27. Adachi K, Kobayashi M, Kawasaki T, et al. Disruption of programmed masticatory movements in unilateral MPTP-treated monkeys as a model of jaw movement abnormality in Parkinson's disease. *J Neural Transm*. 2012;119(8):933-941.
28. Carvalho Aragao Albuquerque L, Justino da Silva H. Jaw movements in people with Parkinson's disease. *Codas*. 2016;28(2):193-196.
29. Watts MW, Tan EK, Jankovic J. Bruxism and cranial-cervical dystonia: is there a relationship? *Cranio*. 1999;17(3):196-201.
30. Kwak YT, Han IW, Lee PH, Yoon JK, Suk SH. Associated conditions and clinical significance of awake bruxism. *Geriatr Gerontol Int*. 2009;9(4):382-390.
31. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. *Mov Disord*. 1997;12(1):73-78.
32. Magee KR. Bruxisma related to levodopa therapy. *JAMA*. 1970;214(1):147.
33. Yachida W, Arima T, Castrillon EE, Baad-Hansen L, Ohata N, Svensson P. Diagnostic validity of self-reported measures of sleep bruxism using an ambulatory single-channel EMG device. *J Prosthodont Res*. 2016;60(4):250-257.
34. Tassorelli C, Armentero MT, Greco R, et al. Behavioral responses and Fos activation following painful stimuli in a rodent model of Parkinson's disease. *Brain Res*. 2007;1176:53-61.

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APPENDIX 1

Inventarisatiestudie: Ziekte van Parkinson & Bruxisme

Datum van invullen: .....

Geeft u toestemming om de ingevulde gegevens te gebruiken voor wetenschappelijk onderzoek? ☐ Ja

☐ Nee *(Zou u zo vriendelijk willen zijn de vragen 1 t/m 3 toch in te willen vullen?)*

1. Wat is uw geslacht? ☐ Man ☐ Vrouw

2. Wat is uw leeftijd? ... jaar

3. Hoe bent u met dit onderzoek in aanraking gekomen?
   - ☐ Parkinson Café
   - ☐ Regionaal ziekenhuis
   - ☐ Social Media
   - ☐ Parkinson(isme) vereniging
   - ☐ Anders, namelijk .....

4. Heeft u een diagnose gekregen betreffende bewegingsstoornissen?
   - ☐ Nee, ik ben:
     - ☐ Partner/mantelverzorger van iemand met Parkinson/Parkinsonisme
     - ☐ Kennis van iemand met Parkinson/Parkinsonisme
     - ☐ Anders, namelijk .....
     - *(ga door met vraag 6)*
   - ☐ Nog niet/Weet ik niet *(ga door met vraag 6)*
   - ☐ Ja

5. Zo ja, welke diagnose(s) heeft u gekregen? En in welk jaar was dat?
   - ☐ de ziekte van Parkinson — jaar: ...
   - ☐ Parkinsonisme:
     - ☐ MSA — jaar: ...
     - ☐ PSP — jaar: ...
     - ☐ Vasculair parkinsonisme — jaar: ...
   - ☐ Anders, namelijk ..... — jaar: ...
   - ☐ Weet ik niet

6. Gebruikt u op dit moment medicatie? Zo ja, welke? *Let op: Dit gaat om alle medicatie die u gebruikt, niet alleen specifiek voor uw eventuele bewegingsstoornis! Heeft u te weinig ruimte? Schrijf dan verder op de achterkant van deze vragenlijst.*

Medicatie	Dosering	
	Aantal Mg	Hoe vaak per dag?
1.		
2.		
3.		
4.		
5.		
6.		
7.		

7. Heeft u <u>ooit</u> pijn gehad in uw kaak, slaapstreek, in het oor of vóór het oor?
   - ☐ Nee (Ga door met vraag 13)
   - ☐ Ja

(*Continues*)

APPENDIX 1 (Continued)

8. Welke van de volgende omschrijvingen past het best bij deze pijn gedurende de afgelopen 30 dagen? Kies ÉÉN antwoord.
   - ☐ Geen pijn
   - ☐ De pijn komt en gaat
   - ☐ De pijn is altijd aanwezig

9. Welk cijfer zou u geven aan de pijn in uw gezicht die u **OP DIT MOMENT** voelt? Geef een cijfer tussen 0 en 10, waarbij 0 "geen pijn" betekent en 10 "ergst mogelijke pijn".

Geen pijn									Ergst mogelijke pijn
0	1	2	3	4	5	6	7	8	9	10

10. Welk cijfer zou u geven aan de **ERGSTE** pijn in uw gezicht die u gedurende de AFGELOPEN 30 DAGEN heeft gevoeld? Geef een cijfer tussen 0 en 10, waarbij 0 "geen pijn" betekent en 10 "ergst mogelijke pijn".

Geen pijn									Ergst mogelijke pijn
0	1	2	3	4	5	6	7	8	9	10

11. Hoe beoordeelt u de **GEMIDDELDE** pijn in uw gezicht die u gedurende de AFGELOPEN 30 DAGEN ondervond? Gebruik dezelfde schaal, waarbij 0 "geen pijn" betekent en 10 "ergst mogelijke pijn". (Dat wil zeggen, uw gebruikelijke pijn op de momenten dat u pijn ondervond.

Geen pijn									Ergst mogelijke pijn
0	1	2	3	4	5	6	7	8	9	10

12. Waren de volgende activiteiten, gedurende de afgelopen 30 dagen, van invloed op uw pijn (dat wil zeggen: het beter of erger gemaakt) in uw kaak, slaapstreek, in het oor of voor het oor aan één of beide kanten?

	Nee	Ja
Kauwen van hard of taai voedsel		
Uw mond openen of uw onderkaak naar voren of opzij bewegen		
Mondgewoonten zoals uw tanden en kiezen op elkaar houden, klemmen of knarsen met uw tanden en kiezen, of kauwgom kauwen		
Andere mondactiviteiten zoals praten, zoenen of geeuwen		

13. Hoe vaak voert u ieder van de onderstaande activiteiten uit, uitgaande van de afgelopen maand. Zet achter elke vraag een vinkje in het juiste vakje en sla geen vragen over. Als het wisselt, kies dan de hogere keuzemogelijkheid.

Activiteiten als u slaapt	**Nooit**	**< 1 nacht/ maand**	**1-3 nachten/ maand**	**1-3 nachten/ week**	**4-7 nachten/ week**
Tanden of kiezen op elkaar klemmen of knarsen als u slaapt, gebaseerd op welke informatie dan ook.					
Activiteiten als u wakker bent	**Nooit**	**Zelden**	**Soms**	**Vaak**	**Altijd**
Tandenknarsen als u wakker bent.					
Tanden of kiezen op elkaar klemmen als u wakker bent.					
Uw tanden of kiezen op elkaar duwen, elkaar laten raken, of op elkaar houden, terwijl u niet aan het eten bent (dus contact tussen boven- en ondergebit).					
Uw kaakspieren aanspannen of strak houden zonder uw tanden of kiezen op elkaar te klemmen of te houden.					
De kaak naar voren of naar opzij houden of duwen.					

(*Continues*)

APPENDIX 1 (Continued)

14. Heeft u de afgelopen 30 dagen pijn of stijfheid in uw kaak gehad bij het wakker worden?
- ☐ Nee
- ☐ Ja

15. Heeft u slijtage aan uw tanden of kiezen?
- ☐ Niet
- ☐ Enigszins
- ☐ Nogal
- ☐ Veel
- ☐ Erg veel
- ☐ Weet ik niet

16. Heeft uw onderkaak ooit "op slot" of vastgezeten, zelfs al was het maar heel even, waardoor uw mond niet VOLLEDIG open kon?
- ☐ Nee (Einde vragenlijst)
- ☐ Ja (Ga door naar vraag 17)

17. Heeft uw onderkaak gedurende de afgelopen 30 dagen "op slot" gezeten waardoor uw mond niet VOLLEDIG kon openen, al was het maar heel even, en vervolgens los te komen zodat u wel VOLLEDIG kon openen?
- ☐ Nee
- ☐ Ja

18. Heeft uw onderkaak gedurende de afgelopen 30 dagen "op slot" of vastgezeten, al was het maar heel even, als u uw mond wijd opende, waardoor u uw mond niet kon sluiten vanuit deze wijd geopende positie?
- ☐ Nee
- ☐ Ja

Heeft u nog opmerkingen?

.....

.....

.....

Hartelijk dank voor het invullen van deze vragenlijst!

U kunt de ingevulde papieren vragenlijst opsturen naar:

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