JAHRESTAGUNG DER ÖSTERREICHISCHEN PARKINSON GESELLSCHAFT

www.parkinson.at

15.-17.10.2015

WIEN AKH Wien

HAUPTPROGRAMM

ÖSTERREICHISCHE PARKINSONGESELLSCHAFT

The *Movement* Disorder Society

INHALT

PROGRAMMÜBERSICHT

	Donnerstag, 15.10.2015	Freitag, 16.10.2015	Samstag, 17.10.2015
09:00 - 09:30		**HAUPTTHEMA 2** Neues zur Therapie der Parkinson-Krankheit und anderer Bewegungsstörungen – Medikamentöse Therapie, Neuromodulation, Rehabilitation 09:00 - 10:30	**HAUPTTHEMA 3** Lokale Therapien von Bewegungsstörungen - Botulinumtoxin 09:00 - 10:30
09:30 - 10:00			
10:00 - 10:30			
10:30 - 11:00		**Kaffeepause und Besuch der Industrieausstellung** 10:30 - 11:00	**Kaffeepause und Besuch der Industrieausstellung** 10:30 - 11:00
11:00 - 11:30		**Themenvortrag 1** (Licher MT) Apomorphin-Pen 11:00 - 11:30	**Themenvortrag 4** (Ipsen Pharma) Abobotulinumtoxin A 11:00 - 11:30
11:30 - 12:00		**Themenvortrag 2** (Novartis Pharma) Parkinson-Demenz 11:30 - 12:00	**Themenvortrag 5** (Merz Pharma) Injektionsintervalle Botulinumtoxin 11:30 - 12:00
12:00 - 12:30		**Generalversammlung** 12:00 - 12:30	**Verleihung der Wissenschaftspreise 2015** **Schlussworte und Verabschiedung** ab 12:00
12:30 - 13:00		**Mittagspause und Besuch der Industrieausstellung** 12:00 - 13:30	
13:00 - 13:30	**Eröffnung** 13:00 - 13:10		
13:30 - 14:00	**HAUPTTHEMA 1** Neue ätiologische und pathogenetische Aspekte bei der Parkinson-Krankheit und anderen Bewegungsstörungen 13:10 - 15:30	**Satellitensymposium 2** (UCB Pharma) Therapie von IPS und RLS 13:30 - 14:30	**Zertifizierungskurs der ÖDBAG** **Botulinumtoxin Zertifizierungskurs 1** 13:30 - 16:30
14:00 - 14:30			
14:30 - 15:00		**Autonome Dysfunktion bei Bewegungsstörungen** 14:30 - 15:00	
15:00 - 15:30		**Kaffeepause und Besuch der Industrieausstellung** 15:00 - 15:30	
15:30 - 16:00	**Kaffeepause und Besuch der Industrieausstellung** 15:30 - 16:00	**Honory member Session/Special Lectures** 15:30 - 17:00	
16:00 - 16:30	**Satellitensymposium 1** (AbbVie) Kontiniuerliche dopaminerge Stimulation 16:00 - 17:00		
16:30 - 17:00			
17:00 - 17:30	**Posterpräsentation und Kurzvorträge 1** 17:00 - 18:00	**Themenvortrag 3** (Medtronic) Chirurgische Therapien 17:00 - 17:30	
17:30 - 18:00		**Posterpräsentation und Kurzvorträge 2** 17:30 - 18:30	
18:00 - 18:30	**Videoforum** 18:00 - 19:00		
18:30 - 19:00			
19:00 - 19:30			
ab 19:30			

KOMITEES

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ALLGEMEINE INFORMATION

Kongressunterlagen

Sie erhalten Ihre gesamten Kongressunterlagen gemeinsam mit Ihrem Namensschild vor Ort an der Registrierung im AKH Wien, direkt vor Hörsaal 3. Ihr Namensschild gilt als Eintrittsausweis zum wissenschaftlichen Programm und ist innerhalb des Veranstaltungsortes gut ersichtlich zu tragen.

Registrierung

Öffnungszeiten des Anmeldeschalters im Hörsaalzentrum (Ebene 7 vor Hörsaal 3):

Donnerstag, 15.10.2015	11:30 – 18:00 Uhr
Freitag, 16.10.2015	08:15 – 18:30 Uhr
Samstag, 17.10.2015	08:15 – 14:00 Uhr

Hauptthemen

- Neue ätiologische und pathogenetische Aspekte der Parkinson-Krankheit
- Neues über die Therapie der Parkinson-Krankheit – Medikamentöse Therapie, Neuromodulation, Rehabilitation
- Lokale Therapien von Bewegungsstörungen inklusive Botulinum-Toxin Praxisseminar
- Honorary-Member-Lectures
- Freie Posteranmeldung mit mündlicher Kurzpräsentation

ANMELDUNG

Bitte melden Sie sich online über www.parkinson.at zur Tagung bzw. zum Zertifizierungskurs an.

Teilnahmegebühren

Kongress Teilnahmegebühren inkl. Rehabilitations- & Pflegesymposium:

Mitglieder	
in Ausbildung *	€ 60,00
Facharzt/Fachärztin	€ 110,00

Nicht-Mitglieder	
in Ausbildung *	€ 110,00
Facharzt/Fachärztin	€ 160,00
Dipl. Pflegepersonal, TherapeutInnen, PsychologInnen	€ 110,00
Karenzierte KollegInnen (aus Kindererziehungsgründen), Studierende *	kostenlos

* Bitte bringen Sie einen entsprechenden Nachweis zum Registrierungsschalter in Wien mit.

Zertifizierungskurs der ÖDBAG (Botulinumtoxin Zertifizierungskurs 1)
Mitglied ÖPG € 25,00
Nichtmitglied € 40,00

Nur Kursbuchungen ohne Kongressteilnahme an: parkinson2015@cmi.at

Fortbildungspunkte

Die Teilnahme an der Jahrestagung der Österreichischen Parkinson Gesellschaft wurde für den Erwerb des Fortbildungsdiploms der Österreichischen Ärztekammer mit 20 DFP-Punkten approbiert. Der Zertifizierungskurs der ÖDBAG wird mit 4 DPF-Punkten approbiert.

Donnerstag 15.10.15

13:00 **Eröffnung**

13:10-15:30 **HAUPTTHEMA 1**

Neue ätiologische und pathogenetische Aspekte bei der Parkinson-Krankheit und anderen Bewegungsstörungen
Vorsitz: W. Poewe, E. Auff

Aktuelle Einblicke in die Pathogenese verschiedener Parkinson-Syndrome
Gabor Kovacs, Wien

Aktuelles zur Pathogenese der MSA
Gregor Wenning, Innsbruck

Essentieller Tremor: Aktuelle Konzepte zur Pathogenese und Klassifikation
Dietrich Haubenberger, Bethesda, USA

Neues zur Pathogenese und Pathophysiologie der Dystonien
Gottfried Kranz, Wien

Genetische Aspekte von Bewegungsstörungen
Alexander Zimprich

15:30-16:00 **Kaffeepause und Besuch der Industrieausstellung**

PROGRAMM - Donnerstag, 15.10.15 - Hörsaal 3

16:00-17:00 **Satellitensymposium 1**

(mit freundlicher Unterstützung der Firma AbbVie)
Kontiniuerliche dopaminerge Stimulation – Erfolgreiche Strategien zur Behandlung des fortgeschrittenen Morbus Parkinson
Vorsitz: G. Ransmayr

Update klinischer Daten zu Levodopa Carbidopa intestinalem Gel (LCIG)
Gerhard Ransmayr, Linz

Erfahrungen aus der Praxis basierend auf Evidenz
Mariella Kögl-Wallner, Graz

17:00 - 18:00 **Posterpräsentation – Kurzvorträge 1**

Vorsitz: G. Wenning, G. Kranz

Identifying freezing of gait in Parkinson's disease using waist-mounted accelerometry
Heidemarie Zach, Wien

Coin rotation as strongest predictor of buttoning in Parkinson's: A Multicenter Study demonstrating the everyday impact of limb-kinetic apraxia
Thomas Foki, Wien

Sniffing the diagnosis: Olfactory testing in neurodegenerative parkinsonism
Florian Krismer, Innsbruck

Orthostatic intolerance and falls in Parkinson's disease
Alessandra Fanciulli, Innsbruck

Variation of nigral Cytochrome C oxidase IV expression during the progression of Parkinson's diesease-related pathology
Ivan Milenkovic, Wien

Early dysfunctions of fronto-parietal networks in Parkinson's disease
Evi Matt, Wien

Age- and gender effects on brainstem MR-planimetry
Stephanie Mangesius, Innsbruck

Differential diagnosis of neurodegenerative parkinsonism using magnetic resonance imaging at 1.5 and 3.0 Tesla
C. Müller, Innsbruck

18:00 - 19:00 **Videoforum**
R. Katzenschlager, S. Bösch

PROGRAMM - Freitag, 16.10.15 - Hörsaal 3

Freitag 16.10.2015

09:00-10:30 **HAUPTTHEMA 2**
Neues zur Therapie der Parkinson-Krankheit und anderer Bewegungsstörungen – Medikamentöse Therapie, Neuromodulation, Rehabilitation
Vorsitz: G. Ransmayr, R. Katzenschlager

Meilensteine der Parkinson Therapie –
Rückblick und Ausblick
Werner Poewe, Innsbruck

Was können nicht-medikamentöse Therapieformen zum Behandlungserfolg bei der Parkinson-Krankheit beitragen?
Eduard Auff, Wien

Neues zur Therapie der Ataxien
Sylvia Bösch, Innsbruck

10:30-11:00 **Kaffeepause und Besuch der Industrieausstellung**

11:00-11:30 **Themenvortrag 1**
(mit freundlicher Unterstützung der Firma Licher MT)
Vorsitz: P. Schwingenschuh

Apomorphin-Pen: Neue Daten und praktische Anwendung
Regina Katzenschlager, Wien

11:30-12:00 **Themenvortrag 2**
(mit freundlicher Unterstützung der Firma Novartis Pharma)
Vorsitz: E. Auff

Parkinson-Demenz: Risikofaktoren und klinisches Management
Gerhard Ransmayr, Linz

12:00-12:30 **Generalversammlung der ÖPG**

12:00-13:30 Mittagspause, Industrieausstellung & Posterbesichtigung

13:30-14:30 **Satellitensymposium 2**
(mit freundlicher Unterstützung der Firma UCB Pharma)
Therapie von IPS und RLS
Vorsitz: W. Poewe

IPS: Früh erkennen und früh behandeln
Gerhard Ransmayr, Linz

RLS: Therapiemöglichkeiten bei Augmentation
Stefan Seidel, Wien

14:30-15:00 **Autonome Dysfunktion bei Bewegungsstörungen**
Aktuelle Erkenntnisse
Walter Struhal, Linz

15:00-15:30 **Kaffeepause und Besuch der Industrieausstellung**

PROGRAMM - Freitag, 16.10.15 - Hörsaal 3

15:30-17:00 **Honorary Member Session/Special Lectures**
The prodromic phase of genetic Parkinson disease
Eduardo Tolosa, Barcelona, Spanien

Update on Huntington's disease - Three stepping stones for the betterment of HD therapeutics
Cristina Sampaio, New York, USA

17:00-17:30 **Themenvortrag 3**
(mit freundlicher Unterstützung der Firma Medtronic)
Vorsitz: S. Bösch

Chirurgische Therapien für Morbus Parkinson.
State of the art und zukünftige Entwicklungen
Klaus Novak, Wien

17:30 - 18:30 **Posterpräsentation – Kurzvorträge 2**
Vorsitz: R. Katzenschlager, T. Sycha

L-Dopa effect on power and variability of Parkinson's tremor is influenced by the behavioral setting
Heidemarie Zach, Wien

Verkehrsunfallsbedingte Verletzungen bei Patienten mit Morbus Parkinson und Patienten mit Maculadegeneration
Carl Homann, Graz

Problems and potential solutions in using intraduodenal Levodopa/Carbidopa infusion therapy
Volker Tomantschger, Hermagor

The Global Multiple System Atrophy Registry. A prospective multi-centre observational cohort study
Sabine Eschlböck, Innsbruck

DYT1, DYT6 und DYT25 in Österreich – genetische Charakterisierung einer neuen Population mit isolierter Dystonie
Christoph Linder, Wien

Injektionsintervalle für unterschiedliche Indikationen für Botulinumtoxin – eine retrospektive Studie
Christoph Linder, Wien

Nabilone in Huntington's disease: a case series of three patients
Robert de Marzi, Innsbruck

Downbeatnystagmus bei einem Patienten mit Essentiellem Tremor: ein Hinweis auf eine zerebelläre Genese?
Ivan Milenkovic, Wien

Augmentation and Impulse Control Disorders in Restless Legs Syndrome –Coexistence or Association?
Beatrice Heim, Innsbruck

Samstag, 17.10.15

09:00-10:30 **HAUPTTHEMA 3**
Lokale Therapien von Bewegungsstörungen - Botulinumtoxin
Vorsitz: P. Schnider, S. Bösch

Präparate, Indikationen, Dosierungen, Injektionsintervalle - alles neu?
Thomas Sycha, Wien

Die Praxis der Botulinumtoxintherapie in einer großen Spezialambulanz
Christoph Linder, Wien

Botulinumtoxin und Emotion
Thomas Sycha, Wien

Botulinumtoxin und Kognition
Gottfried Kranz, Wien

10:30-11:00 **Kaffeepause und Besuch der Industrieausstellung**

11:00-11:30 **Themenvortrag 4**
(mit freundlicher Unterstützung der Firma Ipsen Pharma)
Vorsitz: T. Sycha

Abobotulinumtoxin A zur Behandlung der oberen Extremität Erwachsener – neue Daten
Peter Schnider, Wiener Neustadt

11:30-12:00 **Themenvortrag 5**
(mit freundlicher Unterstützung der Firma Merz Pharma)
Vorsitz: E. Auff

Injektionsintervalle in der Botulinumtoxin-Therapie – Nutzen für Arzt und Patient
Thomas Sycha, Wien

ab 12:00 **Preisverleihung, Schlussworte und Verabschiedung**

13:30-16:30 **Zertifizierungskurs der ÖDBAG**
Botulinumtoxin Zertifizierungskurs 1

Sylvia Bösch, Innsbruck
Thomas Sycha, Wien
Peter Schnider, Wiener Neustadt

Allgemein – Teil 1:
Vorstellung des Curriculums
Vorteile des Zertifikates für den Anwender, Übersicht über den Einsatz von Botulinumtoxin Typ A in der Neurologie

Allgemein – Teil 2:
Pharmakologische Aspekte von Botulinumtoxin (BoNT)
Rechtliche Aspekte (Patientenaufklärung, zugelassene Indikationen, Rückerstattung)
Praktische Hinweise (Verdünnung, Lagerung, Haltbarkeit, Entsorgung)

Dystonie – Teil 1:
Dystonie: allgemeine Grundlagen
Behandlungsmöglichkeiten
Antikörperbildung
Dokumentation

Dystonie – Teil 2:
Zervikale Dystonie: Klinik und Klassifikation Muskelauswahl und BoNT Dosierung - Praktische Fallbeispiele und wichtige Hinweise

A01

Identifying freezing of gait in Parkinson's disease using waist-mounted accelerometry

Heidemarie Zach[1,2], Arno M. Janssen[1], Anke H. Snijders[1], Arnaud Delval[3], Murielle U. Ferraye[1], Eduard Auff[2], Vivian Weerdesteyn[4,5], Bastiaan R. Bloem[1], Jorik Nonnekes[4]

[1] Radboud University Medical Centre, Donders Institute for Brain, Cognition and Behaviour, Department of Neurology, Nijmegen, The Netherlands
[2] Department of Neurology, Medical University of Vienna, Vienna, Austria
[3] Department of Neurology and Movement Disorders, Regional University Hospital, Lille Cedex, France
[4] Radboud University Medical Centre, Donders Institute for Brain, Cognition and Behaviour, Department of Rehabilitation, Nijmegen, The Netherlands
[5] Sint Maartenskliniek Research, Nijmegen, The Netherlands

Background:
Freezing of gait (FOG) is a common and debilitating phenomenon in Parkinson's disease (PD). Wearable accelerometers might help to assess FOG in the research setting. Here, we evaluate whether accelerometry can detect FOG while executing rapid full turns and while walking with rapid short steps (the two most common provoking circumstances for FOG).

Methods:
We included 23 PD patients, who all had objective FOG. Participants performed several walking tasks, including walking rapidly with short steps and rapid full turns in both directions with a triaxial linear waist-mounted accelerometer. Two independent experts identified FOG episodes using off-line video-analysis (gold standard). A validated algorithm [ratio between pathological freezing (3–8 Hz)-and normal locomotor frequencies(0.5–3 Hz)] was applied on the accelerometer data to detect FOG episodes.

Results:
Clinically, FOG was most often observed during full rapid turns (81% of all episodes), followed by walking with short rapid steps (12% of all episodes). During full rapid turns, accelerometry yielded a sensitivity of 78% and specificity of

59%. A sensitivity of 64% and specificity of 69% was observed during walking rapidly with small steps. Combining all tasks rendered a sensitivity of 75% and specificity of 76%.

Conclusion:
Our results suggest that FOG can be detected from a single lumbar accelerometer during several walking tasks, including full rapid turns and walking with short steps rapidly, with reasonable sensitivity and specificity. This approach holds promise for possible implementation as complementary objective outcome in a research setting, but more work remains needed to improve the sensitivity and specificity.
Previously presented: MDS 2015, San Diego

A2

Coin rotation as strongest predictor of buttoning in Parkinson's: A Multicenter Study demonstrating the everyday impact of limb-kinetic apraxia

Thomas Foki[1,5], Tim Vanbellingen[2], Codrin Lungu[3], Walter Pirker[1], Stephan Bohlhalter[2], Thomas Nyffeler[2], Julia Kraemmer[1], Dietrich Haubenberger[1,3], Florian Ph.S Fischmeister[1,5], Eduard Auff[1], Mark Hallett[4], Roland Beisteiner[1,5]

[1] Department of Neurology, Medical University of Vienna, Vienna, Austria
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[3] Office of the Clinical Director, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA
[4] Human Motor Control Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA
[5] MR Center of Excellence, Medical University of Vienna, Vienna, Austria

Parkinson's disease (PD) is associated with impaired dexterity even at early-to-moderate disease stages. Reduced fine motor skills have a detrimental impact

on typical activities of daily living (ADL) such as buttoning. It has been suggested that upper limb bradykinesia represents the most important determinant of dexterity skills. Only recently, limb-kinetic apraxia (LKA) was introduced as a second source of impaired dexterity in PD. It is defined as a loss of hand and finger dexterity resulting from an inability to connect or isolate individual movements. Coin rotation has become its well-acknowledged surrogate task. Nevertheless, the relevance of LKA for typical ADL in PD has not been proven yet. Applying a three site multi-center approach, we therefore aimed to identify the significant predictor of buttoning and unbuttoning in PD. We hypothesized that coin rotation is the most important determinant, but not bradykinesia or overall motor impairment.

64 right-handed, non-demented PD patients were recruited (30 female; age 63±10 years; Hoehn and Yahr stages 1-3). Buttoning, unbuttoning, coin rotation with the right and left hand, respectively, represented the four target tasks, which were performed at maximum speed. Buttoning and unbuttoning consisted of opening or closing five buttons of a cardigan, and 20 flips of a US nickel were defined as coin rotation task (outcome variable: time to complete the particular task). Motor impairment was assessed according to the UPDRS III. All experiments were performed ON medication.

Multiple linear regression analysis showed that coin rotation with the right hand was the only significant predictor of both buttoning ($p<0.001$) and unbuttoning ($p=0.002$) performance. Notably, measures of bradykinesia or overall motor impairment, age or disease duration did not represent significant predictors.
In summary, we demonstrate the relevance of LKA for ADL requiring fine motor skills in PD. The results change the notion of LKA as a pure "academic" phenomenon into a symptom with significant impact on everyday life of PD patients. They also stimulate further research into the pathophysiological basis of LKA and future treatment options, considering the limited response of LKA to dopaminergic therapy. Finally, our results also show that the easy and quick to perform coin rotation test provides valuable information about ADL-relevant dexterity skills in PD.

A3

Sniffing the diagnosis: Olfactory testing in neurodegenerative parkinsonism

F. Krismer*, B. Pinter*, C. Mueller§, P. Mahlknecht, M. Nocker, E. Reiter, A. Djamshidian-Tehrani, S. M. Boesch, G. K. Wenning, C. Scherfler, W. Poewe, K. Seppi§

*, § Authors contributed equally

Department of Neurology, Medical University of Innsbruck, Innsbruck, Austria

Objective:
To determine the diagnostic utility of olfactory testing in patients with neurodegenerative parkinsonism.

Methods:
The Sniffin´ Sticks test battery for assessment of odor identification, odor discrimination, and olfactory threshold was applied to two independent cohorts – a screening and a validation cohort. The screening cohort included Parkinson's disease (PD) patients and healthy controls (HC) and was used to calculate optimal cut-offs for a diagnosis of PD with a sensitivity or specificity exceeding 95%. The validation cohort was used to determine the diagnostic accuracy of the newly established cut-off values in discriminating patients with PD from those with atypical parkinsonian disorders (APD) including multiple system atrophy (MSA) and progressive supranuclear palsy (PSP).

Results:
PD patients (screening cohort n=20, validation cohort n=47) performed significantly worse in olfactory testing than HC (n=41) and patients with MSA (n=23) or PSP (n=23). Diagnostic performance of the identification subscore was similar to the sum score of the Sniffin' Sticks test (AUC identification test 0.94, AUC sum score 0.96). In subjects with neurodegenerative parkinsonism, the specificity cut-off predicted a diagnosis of PD with a sensitivity and specificity of 76.6 and 87.0%, respectively. The discriminative value of this cut-off in separating PD from MSA was 76.7% (sensitivity) and 95.7% (specificity). Optimal cut-offs of the sum score provided a sensitivity and specificity for a diagnosis of PD of 78.7% and 76.1%, respectively. The positive predictive value of olfactory testing adjusted for PD prevalence in the community exceeded 95%.

Conclusions:
Our data suggest that assessment of olfactory function, particularly odor identification, can be useful to discriminate PD from APDs, particularly MSA patients.

A4

Orthostatic intolerance and falls in Parkinson's disease

Alessandra Fanciulli[1], Christian Dallinger[1], Georg Göbel[2], Roberta Granata[1], Susanne Dürr[1], Fabienne Sprenger[1], Florian Krismer[1], Christoph Müller[1], Sylvia Boesch[1], Michael Nocker[1], Christoph Scherfler[1], Klaus Seppi[1], Werner Poewe[1], Gregor K. Wenning[1]

[1]Department of Neurology, Innsbruck Medical University, Austria
[2]Department of Medical Statistics, Informatics and Health Economics, Innsbruck Medical University, Austria

Background:
Falls are associated with increased morbidity and mortality in the aging population. Falls may occur in up to 60% of patients with Parkinson's disease (PD). Beyond age and disease duration, history of previous falls, dyskinesias and freezing of gait are relevant risk factors for future falls in patients with PD. Previous studies reported an association between falls and orthostatic hypotension (OH) in the general population, but not in PD.

Objective:
To assess the relationship between falls, orthostatic intolerance and OH in PD.

Methods:
180 patients with PD (111 males, 32 patients with dementia) who underwent a tilt table examination in Innsbruck between 2008 and 2013 were retrospectively included. Following information was collected from the clinical recordings of the 6 months preceding and following the tilt table examination: history of falls, L-Dopa fluctuations (Off-Phases, Dyskinesias), deep brain stimulation, cardiovascular comorbidities, use of anti-hypotensive or anti-hypertensive drugs, syncope, orthostatic intolerance (dizziness, nausea, headache or "coat-hanger pain" upon standing), and gait abnormalities (freezing of gait, start or turning hesitation).

Results:
In the present PD cohort [median age: 71 years (65; 76); median disease duration: 5 years (3; 10); median L-Dopa equivalent daily dose: 545 mg/day (258; 1080)], falls were reported in 46% of patients. At univariate analysis, falls were associated with more advanced age (p<0.001), disease duration (p=0.002), Hoehn & Yahr stage (p<0.001), higher L-Dopa daily intake (p=0.002), history of dyskinesias (p=0.02), syncope (p<0.001), orthostatic intolerance (p<0.001) and gait abnormalities (p=0.001). Multivariate analysis confirmed an association between falls and more advanced age (p=0.02; OR: 1.6, 95% c.i.: 1.1-2.5), Hoehn & Yahr stage (p=0.04, OR: 1.8, 95% c.i.: 1.0-3.1), gait abnormalities (p=0.04, OR: 2.2, 95% c.i.: 1.0-4.8) and orthostatic intolerance (p=0.001, OR: 3.4, 95% c.i.: 1.7-7.0).

Conclusions
Here we confirm that advanced age, Hoehn & Yahr stage and history of gait abnormalities are significant risk factors for falls in PD. Orthostatic intolerance, rather than OH itself, conveys a 3.4-fold increased risk of falls in PD. Anti-hypotensive measures may thus prevent falling in PD patients reporting symptomatic OH.

ABSTRACTS

A5

Variation of nigral Cytochrome C oxidase IV expression during the progression of Parkinson's diesease-related pathology

Ivan Milenkovic[1], Eva Dassler[2], Michael Weber[3], Gabor G. Kovacs[2]

[1] Department of Neurology, Medical University of Vienna, Vienna, Austria
[2] Institute of Neurology, Medical University of Vienna, Vienna, Austria
[3] Departement of Biomedical Imaging and Image-guided Therapy, Medical University of Vienna, Vienna, Austria

Background:
Parkinson disease (PD) is a neurodegenerative disorder characterized clinically by rigidity, akinesia, resting tremor and postural instability. It is associated with the degeneration of projectory neurons and in particular of the nigrostriatal system. The key player in the pathogenesis is ◊-synuclein, which deposits in the brain following a hierarchical manner (Braak stages 1-6). Mitochondrial dysfunction is thought be central in the pathogenesis of PD. However, it is still not clear whether the nigral neurons already show cytopathological alterations preceding the deposition of ◊-synuclein. Moreover, it is not clear whether mitochondrial alterations of the nigral neurons differ when so called pre-aggregates or ubiquitinated mature ◊-synuclein inclusions are present in the cell.

Aim:
We aimed to evaluate the expression of the mitochondrial Cytochrome C oxidase IV (COX-IV) in the substantia nigra in PD. In particular, we were interested how 1) COX-IV is expressed in the substantia nigra in pre-symptomatic cases (i.e. Braak stage 2); and 2) whether in symptomatic patients with severe involvement of the substantia nigra COX-IV expression differs in cells with ◊-synuclein pre-aggregates versus those with mature inclusions.

Method:
Using cell-based morphometric immunohistochemistry we evaluated COX-IV volume density in cellular populations with different cytopathologies in brain

sections from formalin-fixed, paraffin-embedded tissue blocks of the mesencephalon (containing the pars compacta of the substantia nigra). We compared cells in two PD stages (Braak-Stage 2 and 6) and control brains. As controls we selected cases lacking neurological symptoms and neuropathological evidence of neurodegenerative disease.

Results and Conclusion:
Our observations suggest 1) that mitochondria respond with increased COX-IV volume density to a general neurodegenerative effect even before pathological α-synuclein is detected in the nigral neurons. Interestingly, COX-IV-immunoreactivity 2) decreases significantly as pathological pre-aggregates of α-synuclein deposits in the neurons appear and then 3) slightly increase in neurons with ubiquitinated Lewy bodies. This suggests that surviving mitochondria are able to compensate the loss as a response to disease-associated α-synuclein deposition.

A6

Early dysfunctions of fronto-parietal praxis networks in Parkinson's disease

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Background:
Apraxia is a common symptom in Parkinson's disease (PD) but is clinically difficult to assess, since praxis deficits are superimposed on elementary motor dysfunctions.
Objective:
The aim of the study was to investigate early functional aberrations between PD patients and healthy elderly controls during a praxis sensitive task and to investigate the impact of dopaminergic therapy on praxis-related brain functions.

Methods:
13 clinically non-apraxic PD patients (Hoehn and Yahr stage 1-3) and 14 healthy controls entered this study. During fMRI participants performed a pantomime task in which they imitated the use of visually presented objects. Patients were measured twice, once ON and once OFF dopaminergic therapy. Clinical standard evaluations of parkinsonian symptoms and praxis abilities were administered before fMRI scans and intrascan pantomime performance was assessed.
Results: Compared to controls, patients in both states exhibited significant overactivations in left fronto-parietal core areas of the praxis network and in bilateral occipital regions. Patients ON showed additional overactivations in several left-hemispheric areas including striatum, thalamus, precentral gyrus, superior parietal areas and frontal orbital cortex while patients OFF displayed additional overactivation in the left inferior frontal gyrus and in the left angular gyrus. Although none of the patients was apraxic according to clinical standard evaluations, patients OFF scored significantly lower on praxis and intrascan performance scores that controls.

Conclusion:
We conclude that even non-apraxic PD patients already show signs of praxis network dysfunctions and rely on specific overactivations to avoid clinically evident apraxic symptoms.

A7

Age- and gender effects on brainstem MR-planimetry

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Background:
Brainstem planimetry has been successfully applied to differentiate Progressive Supranuclear Palsy (PSP) from Parkinson's Disease (PD), Multiple- System-Atrophy (MSA) and healthy controls (HC). It has been suggested that ageing might influence the brainstem midbrain-to-pontine-area-ratio (M/PA). (1)

Objectives:
To evaluate age- and gender effects on brainstem MR-planimetric measures.

Methods:
MR planimetry was performed in 39 HC (mean age 55.7 years; SD 13.7 years; female-to-male-ratio = 23:16) on a 1.5T scanner to assess midsagittal midbrain area, midsagittal pontine area, mean MCP diameter, mean SCP diameter, (2) as well as midsagittal midbrain and pontine diameter. (3) From these parameters, the MR-Parkinson-Index (MRPI), M/PA as well as the midbrain-to-pontine-diameter-ratio (M/Pd) were calculated. (1-3). Planimetric measures were performed on an imaging database program (IMPAX EE). Gender-related differences of all MR-planimetric measurements as well as of the MRPI, M/PA and MPd were calculated by using ANCOVAs entering gender as factor and age as covariate. Due to the multiple comparisons, p-values of < 0.01 were considered statistically significant.

Results:
ANCOVA models revealed no significant gender-effects on any variable. However, ANCOVA models revealed significant age-effects on midbrain area ($p < 0.0001$; F 34.8), on midbrain diameter ($p < 0.0001$; F 23.6), on the MRPI ($p=0.005$; F 8.8), the MPd ($p=0.002$; F 10.9) and the M/PA ($p < 0.0001$; F 28.0).

Conclusions:
While there are no significant gender-effects on any brainstem MR planimetric measures, our data suggest significant age-effects on the MRPI, MPd and M/PA. This has clinical implications highlighting the need for age-specific cut-offs for the differential diagnosis of PSP from other forms of parkinsonism.

(1) Morelli M, et al. Mov Disord. 2014;29(4):488-495.
(2) Hussl A, et al., Mov Disord. 2010;25(14):2444-2449.
(3) Massey LA, et al. Neurology. 2013;14;80(20):1856-1861.

A8

Differential diagnosis of neurodegenerative parkinsonism using magnetic resonance imaging at 1.5 and 3.0 Tesla

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Background/Aim:
While magnetic resonance imaging (MRI) at 3.0 Tesla (T) is increasingly used in the diagnostic work-up of parkinsonian disorders, no studies have assessed its diagnostic potential for differential diagnosis of neurodegenerative parkinsonism in comparison to 1.5T MRI. Our aim is to assess the diagnostic value of 3.0T MRI in detecting atrophy and signal changes in basalganglia and infratentorial structures for the differential diagnosis of Parkinson's disease (PD), multiple system atrophy (MSA) and progressive supranuclear palsy (PSP).

Methods:
Patients with early stage PD, MSA, PSP and healthy controls (HC) underwent brain MRI at 1.5 and 3.0T. Blinded to diagnosis and field strength, semiquantitative and -qualitative rating of different brain regions was performed. Abnor-

malities rated as indicative for MSA included putaminal atrophy, T2 putaminal hypointensity, T2 hyperintense putaminal rim, hot cross bun sign, MCP atrophy, T2 MCP hyperintensity and cerebellar atrophy, while midbrain atrophy, hummingbird sign and SCP atrophy were rated as signs for PSP.

Results:
Scans positive for MSA at 1.5T revealed good discriminatory power between groups. Sensitivity at 3.0T was 90.5% in distinguishing MSA from PD, PSP and HC, whereas specificity was poor, but improved by excluding putaminal signal changes. MRI scans indicative for PSP showed similar diagnostic accuracies at 1.5 and 3.0T in distinguishing PSP from MSA, PD and HC.

Comclusion:
Rater-based assessment of structural abnormalities in MRI at 3.0T does not seem to yield greater diagnostic accuracy as compared to 1.5T. Evaluation of putaminal signal abnormalities on 3.0T carries a high rate of false positive rates for MSA.

Grant:
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Table 1:
Demographical characteristics of study participants (mean ± standard deviation).

	PD (H&Y 1-2)	MSA	PSP	HC
Number	21	21	20	21
Sex (m/f)	14/7	12/9	14/6	10/11
Age (years)	62.6 ± 10.2	62.8 ± 8.9	68.1 ± 6.1	66.2 ± 5.9
Disease duration (Years)	4.3 ± 10.2	2.3 ± 1.5	1.9 ± 1.6	NA
Hoehn and Yahr	1.9 ± 0.1	3.2 ± 0.9	3.1 ± 0.8	NA
UPDRS total	30.3 ± 8.2	67.8 ± 12.9	54.6 ± 16.5	NA

Table 2: Sensitivity and specificity of scans indicative for MSA in patients with PD, MSA, PSP and HC.

Field strength	Evaluation	MSA	PD	PSP	HC
		n (sensitivity)		n (sensitivity)	
1.5T	Atrophy patterns° and signal changes*	19 (90.5%)	1 (95.2%)	4 (80%)	0 (100%)
3.0T	Atrophy patterns° and signal changes*	19 (90.5%)	4 (81.0%)	12 (40%)	8 (61.9%)
1.5T	Atrophy patterns°	16 (76.2%)	1 (95.2%)	3 (85%)	0 (100%)
3.0T	Atrophy patterns°	15 (71.4%)	2 (90.5%)	3 (85%)	0 (100%)

* Signal changes: T2 putaminal hypointensity, T2 hyperintense putaminal rim, hot cross bun sign, T2 MCP hyperintensity.
° Atrophy patterns: putaminal atrophy, MCP atrophy, cerebellar atrophy.

Table 3:
Sensitivity and specificity of scans indicative for PSP in patients with PD, MSA, PSP and HC.

Field strength	Evaluation	PSP	PD	MSA	HC
		n (sensitivity)		n (sensitivity)	
1.5T	Atrophy patterns°	19 (95%)	1 (95.2%)	4 (81%)	0 (100%)
3.0T	Atrophy patterns°	16 (80%)	0 (100%)	4 (81%)	1 (95.2%)

° Atrophy patterns: midbrain atrophy, hummingbird sign, SCP atrophy.

A9

L-Dopa effect on power and variability of Parkinson's tremor is influenced by the behavioral setting

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Objective:
To investigate L-Dopa effects on resting tremor (RT) parameters, influenced by behavioral settings.

Background:
Parkinson's disease (PD) RT responds variably to dopaminergic treatment. However,the influencing factors on the L-Dopa effect are unclear. In contrast to many other PD symptoms,RT varies depending on behavioral context like cognitively demanding tasks.

Methods:
In 42 tremordominant PD patients(RT score >2 MDS-UPDRS pts.) clinical scores(UPDRS,tremor rating scale) and accelerometry in the 2 contexts:rest(3x60s) and cognitive coactivation (COCO; counting backwards in steps of three;3x60s)were collected in ON and OFF state. RT power at individual RT frequency was estimated in Fieldtrip by calculating time frequency representations between1-20Hz. For the RT variability we calculated the coefficient of variation of the resulting RT amplitude regressor. Using repeated measures ANOVA,we analyzed the effect of factors TREATMENT(OFF vs. ON),CONTEXT (rest vs. counting) and REPETITION (3 episodes) on average RT power and tremor variability.

Results:
We found a clear L-Dopa effect: total UPDRS 43vs.26, clinical RT scores 3.2vs.2.4 pts. ($p<0.001$); tremor constancy 3.2 vs.1.8 pts. ($p<0.001$). L-Dopa reduced RT power ($p<0.001$), while COCO increased it ($p<0.001$). The L-Dopa effect was sign.smaller during COCO than during rest (TREATMENT x CONTEXT interaction,$p=0.036$). In fact, RT power during COCO ON L-Dopa was similar to RT power at rest OFF L-Dopa($p>0.1$). RT power correlated significantly with clinical RT scores in both conditions (correlation coefficient>0.5;Spearman's $\rho<0.001$). RT variability increased after L-Dopa ($p<0.001$) and was smaller during COCO than rest($p<0.001$); there were no sign. interactions. RT variability was inverse related to clinical tremor constancy(correlation coefficient<-0.6;Spearman's $\rho<0.001$).

Conclusions:
A cognitively demanding task reduces the L-Dopa effect on RT (inconsistent with a previous study (Sturman et al.,2007)).This emphasizes the importance of a simple counting task in clinical practice, as measurements at rest will overestimate the treatment effect. L-Dopa increased RT variability, indicating that patients experience more alternations between rest and tremor episodes. Both factors may contribute to the subjective experience of many patients that L-Dopa is ineffective for RT. Future research is aimed to identify clinical, electrophysiological and cerebral factors influencing the L-Dopa effect on RT.
Previously Presented: MDS 2015, San Diego

A10

Verkehrsunfallsbedingte Verletzungen bei Patienten mit Morbus Parkinson und Patienten mit Maculadegeneration

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Hintergrund:
Es ist bekannt dass sowohl Parkinsonpatienten (IPS-P) als auch Makuladegenerationspatienten (MD-P) körperliche Defizite haben die sie unfallsanfälliger machen. Bei Parkinsonpatienten kommt darüber hinaus noch dazu, dass aufgrund des Rigors und der gestörten Stellreflexe Stürze tendenziell schwerer ausfallen und dass etliche pathophysiologische Parameter dafür sprechen

dass die Erholung nach Traumata verzögert bzw. schlechter abläuft.

Fragestellung:
Wie stark sich diese zusätzlichen Faktoren bei Parkinsonpatienten im Vergleich zu einer Kontrollgruppe mit ebenso verkürzter aufs Gesamtleben berechneter Gesamtverkehrsteilnahme bemerkbar macht, ist bisher unerforscht und war Zweck dieser Untersuchung.

Methode:
Bei allen 20 IPS-P und 45 MD-P über 65 Jahre die von 01.01.2003 bis zum 31.12.2013 auf Grund eines Verkehrsunfalls an einer chirurgischen Abteilung in Graz (LKH, UKH) aufgenommen wurden, wurden Parameter der Verletzungsschwere (Injury Severity Score, Notwendigkeit operativer bzw. intensivmedizinischer Behandlung) und des Outcomes (Heilungsdauer, Mortalität, Braden-Score) erhoben und statistisch verglichen.
Ergebnis: IPS-P verunfallen relativ sehr viel häufiger als MD-P. Die Verletzungsschwere nach ISS war bei IPS-P leicht erhöht gegenüber der Kontrolle, allerdings nicht in signifikantem Ausmaß (ISS IPS-P: 10.3 vs.MD-P: 9.38, p=0.478). und müssen mit durchschnittlich 16.44 Tagen tendenziell länger stationär behandelt werden (p=0.07); die Mortalität allerdings war nicht erhöht.

Conclusio:
Erfreulicherweise ist die Inzidenz von schweren Verletzungen in beiden Gruppen insgesamt gering, was auf ein verantwortungsvolles Verhalten im Straßenverkehr schließen lässt. Das Outcome bezogen auf Aufenthaltsdauer ist bei nicht wesentlich gesteigerter Verletzungsschwere tendenziell schlechter, sodass größere Studien unter Einbeziehung einer Ursachenerhebung sinnvoll erscheinen.

A11

Problems and potential solutions in using intraduodenal Levodopa/Carbidopa infusion therapy

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Background:
LCIG is one of the recognised therapy options in managing motor and increasingly also non-motor symptoms of APD.

Objective:
To describe our experiences with Levodopa/Carbidopa intraduodenal gel therapy (LCIG) in advanced Parkinson Disease (APD) focussing on problems encountered and solutions used.

Methods:
Between 10/2006 and 12/2014 we switched 55 PD patients (64% male, 36% female) from oral and/or deep brain stimulation to LCIG therapy. Average age: 71.6 yrs (range 49-83); average time since diagnosis: 13,7 yrs (range 3-40). We conducted two surveys (2009 and 2014) on problems encountered by patients and their carers. Inclusion criteria were a minimum of 6 months with LCIG. We identified 10 technical, organisational or health-related problems. In addition, some clients felt disappointed with the effects of LCIG and some found the daily routine too elaborate.

Results:
Our solutions for the 12 problems are summarised in table 1.

Table 1:

Problems	Solutions
Peritonitis	Antibiotics (*)
Painful inflammation at stoma	Investigate and treat cause of problem (*); consider removal of tube
Secretion and formation of hypertrophic granulation tissue	Tighten internal retention plate (don't twist tube); Eosin 0,15 solution; Silver nitrate pen
Dislocation of tube	Setting new tube (*)
Broken tube	Setting new tube (*)
Pump failure	Oral emergency medication until replacement of pump (*)
Broken connectors	Specialist nurse changes connectors during home visit
Unexplainable alarm	Checking tube; switching off and on, telephone help; training
Faulty cassette	Replacement
Problems with order or delivery of medication	Guidelines on: medical prescription, approval of prescription, ordering medication through chemist
Disappointment with effects	Increased information, goals and expectations agreed in writing
Daily routine too elaborate	Increased information and training, ensuring social network is functioning

(*)These items resulted in an interdisciplinary workshop to improve quality of services.

Summary:
LCIG is a method of delivering therapy to patients with APD which is not without problems. However, if managed interdisciplinary and if appropriate support is given to patients and their carers, these problems can be overcome and, moreover, lead to client satisfaction. Nevertheless, 20% of clients decided to discontinue with LCIG therapy, mainly due to effects not living up to expectations or to challenging routine care.

A12

The Global Multiple System Atrophy Registry. A prospective multicentre observational cohort study.

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Background:
Multiple system atrophy (MSA) is a rare progressive neurodegenerative disorder characterized by parkinsonian, cerebellar, pyramidal, and autonomic features in any combination. The focus of attack is mainly on oligodendrocytes of affected brain areas and misfolded alpha-synuclein protein deposits represent the pathological hallmark of the disease. Currently, there exists no effective treatment that can delay or slow down disease progression. The limited natural history data, the insufficient knowledge about the disease progression, and the absence of tests to diagnose presymptomatic subjects or patients at very early stage of disease restrict the development of effective therapies for MSA.

Objective:
In the present study, we aim to create the first global online database patient registry for multiple system atrophy to overcome key gaps in the knowledge of the natural history. We further expect to improve early diagnosis by red flags screen and to further define the fate of the disease. We aim to create a database that will enable us to facilitate multi-centre biomarker studies and clinical trial activitiy by recruiting a sufficient number of patients that provide an adequate statistical power to detect the efficacy of target molecules.

Methods:
This is a prospective multi-centre observational cohort study on a global level that has started on 1st January 2015 and will end on 31st December 2019. In

this time we anticipate to enroll a total of 1000 clinical possible or probable MSA patients. In Innsbruck we expect to recruit up to 150 patients in those 5 years. A minimal mandatory 2-page dataset comprising of contact information (voluntary), basic demographic information and a clinical symptom questionnaire, which will be repeated annually, is used. Data are stored centrally in a de-identified manner for further subject protection.

Conclusions:
We hope to create a database that provides valuable epidemiological and clinical data concerning MSA and which enables us to accelerate clinical trial activity.

Acknowledement: US MSA Coalition

A13

DYT1, DYT6 und DYT25 in Österreich – genetische Charakterisierung einer neuen Population mit isolierter Dystonie

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Poster präsentiert von Christoph Linder[4]

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Einleitung:
Es konnten bislang drei Gene bei Patienten mit einer isolierten Dystonie beschrieben werden, diese sind TOR1A, THAP1 und GNAL.1 Das Ziel dieser Studie war es die Häufigkeit dieser genetischen Veränderungen in einer österreichischen Population mit isolierter Dystonie zu bestimmen.

Methodik:
Es wurden 217 Patienten mit isolierter Dystonie von der Medizinischen Universität Innsbruck rekrutiert. Die kodierenden Regionen der entsprechenden Gene wurden mittels DNA- Sequenzierung und High- Resolution Melting analysiert.

Resultate:
Bei einem Patienten konnte eine bisher einmalig beschriebene Missense- Variante im Exon 5 des TOR1A-Gens detektiert werden. Im THAP1-Gen konnten zwei neue Mutationen gefunden werden, diese waren eine 4-Nukleotid Duplikation sowie eine Substitution (Exon 3). Zwei Patienten präsentierten sich mit Mutationen im GNAL-Gen, es konnte eine neue Missense-Variante sowie eine bereits bekannte Variante in der Splice-Region identifiziert werden.

Diskussion:
In dieser österreichischen Population konnten fünf Mutationen in den drei bisher beschriebenen Genen bei insgesamt 217 Patienten mit isolierter Dystonie gefunden werden. Diese Prävalenz entspricht in etwa den bisher publizierten Daten.1 Eine genauere Untersuchung ist jedoch notwendig, um eventuell weitere Gene zu identifizieren.

1 Klein C. Genetics in dystonia. Parkinsonism Relat Disord 2014;20 Suppl 1:S137-142

A14

Injektionsintervalle für unterschiedliche Indikationen für Botulinumtoxin – eine retrospektive Studie

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Einleitung:
Botulinumtoxin Typ A und B wird zur Therapie unterschiedlicher neurologischer Erkrankungen eingesetzt und führt zu einer Verbesserung der Symptome für durchschnittlich drei Monate. Bei den meisten Patienten sind Re-Injek-

tionen in regelmäßigen Intervallen notwendig. Das Ziel dieser retrospektiven Studie ist Indikationen mit längeren Injektionsintervallen zu finden.

Methodik:
In dieser retrospektiven Studie analysierten wir die Datenbank der Ambulanz für Botulinumtoxinbehandlungen im AKH Wien zwischen Januar 2011 und November 2013. Folgende Indikationen wurden in die Analyse eingeschlossen: zervikale Dystonie, Blepharospasmus, Fokale Spastik, Spasmus hemifacialis, Schreibkrampf, Musikerkrampf, Meige- Syndrom, Kieferöffnungs- und Kieferschlussdystonie sowie fokale Hand – und Fußdystonie.

Resultate:
Wir analysierten 2116 Injektionsintervalle bei 395 Patienten/innen. 344 dieser Patienten wurden aufgrund der folgenden vier Indikationen behandelt: Zervikale Dystonie, Blepharospasmus, Fokale Spastik, Spasmus hemifacialis. Bei diesen Patienten lag das durchschnittliche Injektionsintervall zwischen 3,59 Monaten für die Diagnose Zervikale Dystonie und 3,74 Monaten für die Diagnose Fokale Spastik. 21 Patienten wurden aufgrund eines Kopftremors behandelt mit einem durchschnittlichen Injektionsintervall von 3,65 Monaten. Fünf Patienten wurden aufgrund eines Meige- Syndroms behandelt mit einem Injektionsintervall von 3,91 Monaten. 25 Patienten wurden aufgrund einer der restlichen Indikationen (Schreibkrampf, Musikerkrampf, Kieferöffnungs- und Kieferschlussdystonie, fokale Hand- und Fußdystonie) behandelt. Diese Patienten hatten ein durchschnittliches Injektionsintervall von 4,5 Monaten. Es gab keine statistisch signifikanten Assoziationen zwischen Indikation und Injektionsintervall.

Diskussion:
Aufgrund unserer Analyse konnten wir einen Trend für längere Injektionsintervalle bei 25 Patienten finden, die aufgrund eines Schreibkrampfs, eines Musikerkrampfs, einer Kieferöffnungsdystonie, einer Kieferschlussdystonie oder einer fokalen Hand- oder fokalen Fußdystonie behandelt wurden. Neben dem retrospektiven Studiendesign konnten auch nur wenige Patienten mit den selteneren Indikationen eingeschlossen werden.

ABSTRACTS

A15

Nabilone in Huntington's disease: a case series of three patients

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Background and Objective:
There is limited evidence about the efficacy and safety of cannabinoids in the treatment of patients with Huntington's disease (HD). Three patients with HD were treated with nabilone, a synthetic cannabinoid, in order to alleviate therapy-resistant symptoms.

Methods:
All patients and caregivers were informed about the off-label use of nabilone and gave written informed consent. A Clinical Global Impression Scale (CGI) was applied for routine purposes after one and four weeks to decide on the continuation of nabilone treatment. Furthermore, the Unified Huntington Disease Rating Scale (UHDRS) was completed prior to, one and four weeks after treatment with nabilone. A visual analogue pain scale was performed in patient 2 at these time points.

Case reports:
:: Patient 1 is a 20 year old male who presented with disabling tics, generalized chorea and increased irritability. He developed severe parkinsonism during therapy with amisulpiride and olanzapine without improvement of the psychiatric symptoms and chorea. Amisulpiride was stopped and nabilone 1 mg/day was introduced.

:: Patient 2 is a 48 year old female with a long history of HD and chronic pain. Multiple attempts to alleviate her symptoms with treatments such as olanzapine, tiapride, amisulpiride, amitryptiline were ineffective. She was treated with nabilone 2 mg/day.

:: Patient 3 is a 62 year old female with disabling chorea and increased irritability. She developed severe akathisia as a side effect of different therapies including olanzapine, tetrabenazine, and tiapride. Treatment with nabilone 1.5 mg/day was commenced.

Results:
Transient mild sedation during titration and mild non-disturbing xerostomia occurred in patient 3. No further side effects were observed. All three patients reported a significant improvement of their symptoms as assessed by the CGI. Moreover, chorea and other motor symptoms assessed by the UHDRS improved in all three patients. Furthermore, a reduction of irritability and tics without worsening of parkinsonism was seen in patient 1. Patient 2 reported that her pain completely subsided and irritability substantially improved in patient 3. Moreover, tetrabenazine could be stopped in patient 3 resulting in a remission of akathisia.

Conclusions:
This case series suggests that nabilone may be effective and well tolerated adjunct to the drug treatment of HD. However, larger controlled trials are needed to confirm these preliminary open-label observations.

A16

Downbeatnystagmus bei einem Patienten mit Essentiellem Tremor: ein Hinweis auf eine zerebelläre Genese?

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Hintergrund:
Obwohl der essentielle Tremor (ET) zu den am häufigsten vorkommenden neurodegenerativen Erkrankungen zählt, ist seine Pathophysiologie noch weitgehend unklar. Jüngste Studien haben auf eine mögliche Assoziation zwischen ET und zerebellären Störungen der Okulomotorik hingewiesen.

Fragestellung:
Die Beschreibung der klinischen und apparativen Untersuchungsergebnisse bei einem Patienten mit ET und assoziiertem downbeat Nystagmus. Ein gleichzeitiges Vorkommen von ET und eines zerebellären Syndroms unterstützt die Theorie einer zerebellären Genese des ET.

Methoden:
Wir berichten über einen 51-jährigen männlichen Patienten, der in der Spezialambulanz für Gleichgewichtsstörungen der Universitätsklinik für Neurologie, MUW, aufgrund einer langsam progredienten Gangunsicherheit vorstellig wurde. Bei der neurologischen Untersuchung zeigte sich ein asymmetrischer Ruhe- und Haltetremor. Dieser sei seit ungefähr 10 Jahren langsam progredient gewesen. Zudem konnte eine positive Familienanamnese für den Tremor erhoben werden. Es ließen sich weder weitere zerebelläre Zeichen noch extrapyramidale Symptome feststellen. In der MRT ließ sich keine strukturelle Läsion nachweisen, insbesondere war keine zerebelläre Atrophie fassbar. Mittels Elektroenzephalographie konnte eine Epilepsia partialis continua ausgeschlossen werden. In der Video-Nystagmographie mit Drehstuhltest ließ sich ein spontaner Downbeatnystagmus objektivieren, ohne zusätzlichen vestibulären Zeichen oder Okulomotorikstörungen.

Ergebnisse und Zusammenfassung:
Wir beschreiben eine Koinzidenz zwischen ET und spontanem Downbeatnystagmus bei einem Patienten mit langsam progredientem Tremor und Gangunsicherheit. Jüngste Studien legen nahe, dass im Vergleich zur Allgemeinpopulation ein positional Downbeatnystagmus häufiger bei Patienten mit ET vorkommt. Somit könnte sowohl dem idiopathischen Downbeatnystagmus, als auch dem ET eine zerebelläre Dysfunktion zugrunde liegen. Weitere systematische Studien sind allerdings erforderlich, um die genaue Pathophysiologie des ET zu klären.

A17

Augmentation and Impulse Control Disorders in Restless Legs Syndrome –Coexistence or Association?

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Background:
Augmentation and impulse control disorders (ICDs) are both serious complications of dopaminergic treatment of restless legs syndrome (RLS) but little is known about possible associations between these drug-induced disorders.

Objectives:
To compare the frequency and severity of ICDs in patients with RLS with and without augmentation under dopaminergic therapy.

Methods:
In total, 50 RLS patients were recruited. Of these, 27 patients had augmentation. The frequency of ICDs was assessed using semi-structural interviews.

Results:
Demographic variables did not differ between RLS patients with and without augmentation. RLS patients with augmentation took significant higher dopaminergic medication than patients without augmentation. Eighteen RLS patients (36%) had ICD symptoms, with half of these patients (n=9) having definitive ICDs. Furthermore, patients with augmentation had an increased risk of having in addition ICDs (p=0.02, OR=5.12, 95% CI [1.37, 19.08].
Conclusions: RLS patients with augmentation have a five-fold risk of exhibiting in addition ICD symptoms, which implies that these two behaviours are related and may share a common pathophysiology. Further studies in a larger sample size are needed to confirm our preliminary results.

ABSTRACTS

ANFAHRT

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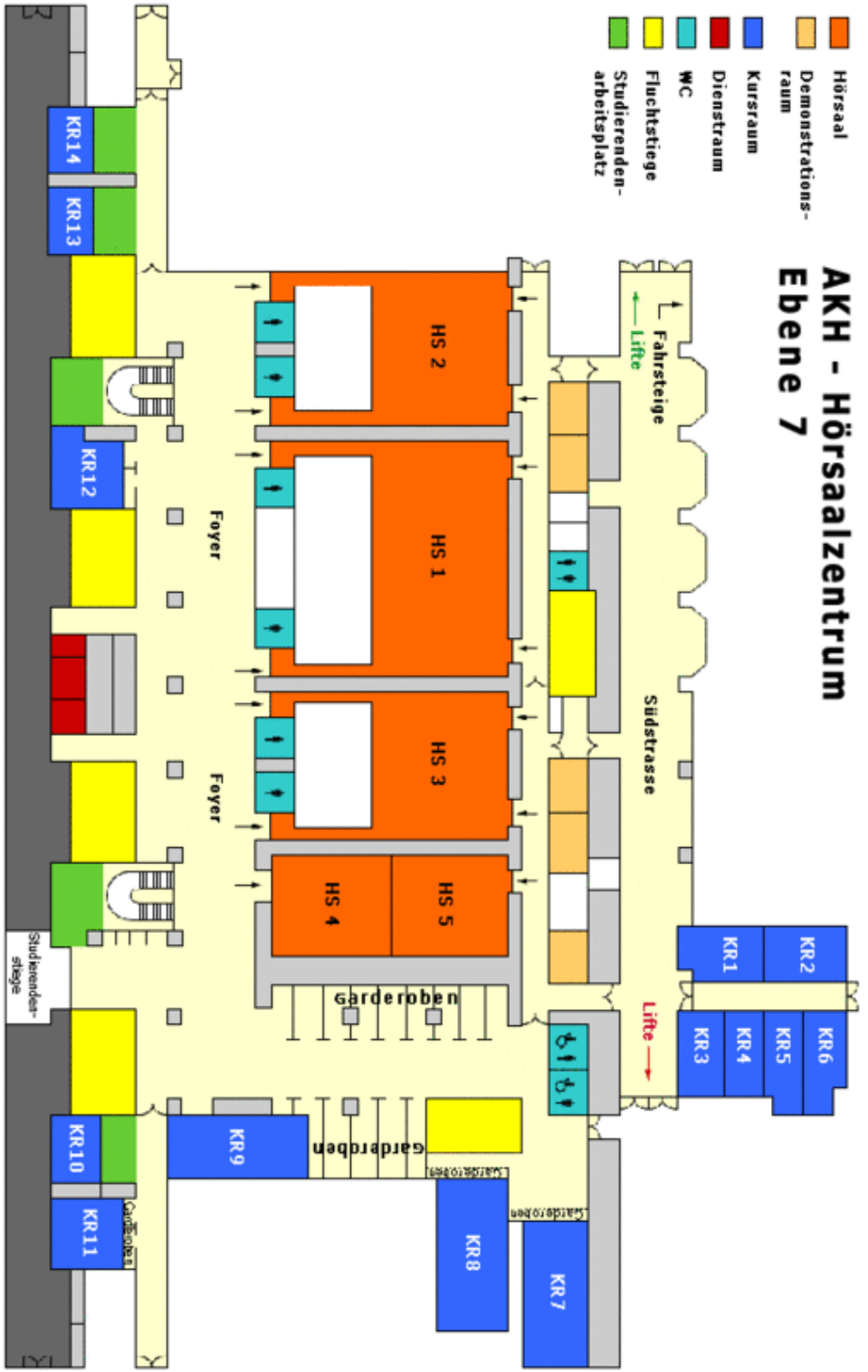

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NOTIZEN

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