JAHRESTAGUNG DER ÖSTERREICHISCHEN PARKINSON GESELLSCHAFT

www.parkinson.at

8.-10.11.2018

KEPLER UNIVERSITÄTSKLINIKUM
AUSBILDUNGSZENTRUM
AM MEDCAMPUS V

LINZ

HAUPTPROGRAMM

ÖSTERREICHISCHE PARKINSONGESELLSCHAFT

The *Movement* Disorder Society


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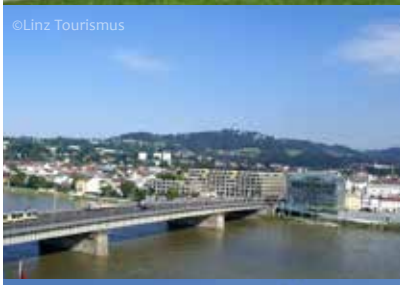
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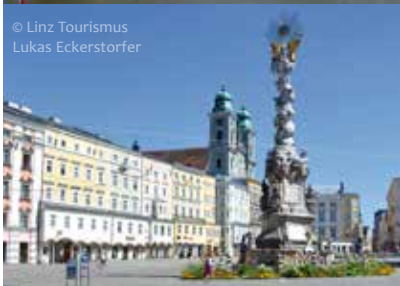
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INHALTSVERZEICHNIS

GRUSSWORT WILLKOMMEN

Sehr geehrte Damen und Herren,
liebe Kolleginnen und Kollegen,

zur Jahrestagung 2018 der Österreichischen Parkinson Gesellschaft (ÖPG) begrüßen wir Sie herzlich, diesmal am Kepler Universitätsklinikum Linz. Wir wollen Ihnen ein Programm anbieten, das aktuelle wissenschaftliche Erkenntnisse und Entwicklungen aus dem Spektrum der Bewegungsstörungen und deren praktische medizinische Anwendung auf den Punkt bringen soll. Dazu konnten wir renommierte nationale und internationale Expertinnen und Experten gewinnen. Österreichische Wissenschaftlerinnen und Wissenschaftler werden über neueste Forschungsergebnisse berichten. Außerdem bieten Satellitensymposien, unterstützt von der forschenden Industrie, Einblicke in die praktische Anwendung neuer Therapien. Das Basal-Ganglien-Seminar (lehrreiche Beispiele mit Videodokumentation), die Fortbildungs-Akademie, der Zertifizierungskurs Botulinumtoxin und der Vortrag des Ehrengastes runden das Programm ab. Wir freuen uns, dass Sie am Kongress teilnehmen und wünschen Ihnen dabei viel Erfolg.

Mit besten Grüßen

G. Ransmayr, Tagungspräsident
E. Auff, Präsident der ÖPG
L. Kellermair, Tagungssekretär

PROGRAMMÜBERSICHT

	Donnerstag, 08.11.2018
08:00 - 08:30	**Eröffnung** 08:15 - 08:30
08:30 - 09:00	**HAUPTTHEMA 1** Neues zu atypischen Parkinson-Syndromen – Klassifikation, Diagnostik, Therapien 08:30 - 10:30
09:00 - 09:30	
09:30 - 10:00	
10:00 - 10:30	
10:30 - 11:00	**Kaffeepause und Besuch der Industrieausstellung** 10:30 - 11:00
11:00 - 11:30	**Themenvortrag** 11:00 - 11:30
11:30 - 12:00	**Freie Vorträge** V1 - V6 11:30 - 13:00
12:00 - 12:30	
12:30 - 13:00	
13:00 - 13:30	**Mittagspause und Besuch der Industrieausstellung** 13:00 - 14:00
13:30 - 14:00	
14:00 - 14:30	**HAUPTTHEMA 2** Interdisziplinäre Aspekte von Parkinson-Syndromen 14:00 - 16:00
14:30 - 15:00	
15:00 - 15:30	
15:30 - 16:00	
16:00 - 16:30	**Kaffeepause und Besuch der Industrieausstellung** 16:00 - 16:30
16:30 - 17:00	**Satellitensymposium** 16:30 - 17:30
17:00 - 17:30	
17:30 - 18:00	**Generalversammlung der ÖPG** 17:30 - 18:00
18:00 - 18:30	**Basal-Ganglien-Seminar** 18:00 - 19:00
18:30 - 19:00	
19:00 - 19:20	

Freitag, 09.11.2018	Samstag, 10.11.2018	
	Hörsaal 1	Hörsaal 3
	Basics in Diagnostik und Therapien von Dystonien 08:00 - 09:00	Grundlagen der Diagnostik und Differenzialdiagnostik von Ataxien 08:00 - 09:00
HAUPTTHEMA 3 Neue Therapien und Therapiekonzepte für Bewegungsstörungen 08:30 - 10:00	Neue klinische Diagnose-kriterien und Biomarker der Parkinson-Krankheit 09:15 - 10:15	Management neuropsych-iatrischer Probleme beim M. Parkinson 09:15 - 10:15
Kaffeepause und Besuch der Industrieausstellung 10:00 - 10:30		
Satellitensymposium 10:30 - 11:45	Therapie nicht-motorischer Symptome der Parkinson-Krankheit 10:30 - 11:30	Differenzialdiagnose von Tremor-Syndromen 10:30 - 11:30
Freie Vorträge V7 - V12 11:45 - 13:00	**Zertifizierungskurs Botulinumtoxin** 12:00 - 15:00	
Mittagspause und Besuch der Industrieausstellung 13:00 - 14:00		
HAUPTTHEMA 4 Was gibt es Neues zu Bewegungsstörungen? 14:00 - 16:00		
Kaffeepause und Besuch der Industrieausstellung 16:00 - 16:30		
Satellitensymposium 16:30 - 18:00		
In memoriam Prof. Gerald Stern 18:00 - 18:10		
Honorary Lecture 18:10 - 19:10		
Wissenschaftspreisverleihung und Verabschiedung 19:10 - 19:20		

WISSENSCHAFTLICHE LEITUNG

Tagungspräsident
Prim. Univ.-Prof. Dr. Gerhard Ransmayr
Tagungssekretär
Dr. Lukas Kellermair

Programmkomitee
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Priv.-Doz. Dr. Sylvia Boesch
Priv.-Doz. Dr. Atbin Djamshidian
Priv.-Doz. Dr. Regina Katzenschlager
Univ.-Prof. Dr. Walter Pirker
o. Univ.-Prof. Dr. Werner Poewe
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Ao. Univ.-Prof. Dr. Christoph Scherfler
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Univ.-Prof. Dr. Klaus Seppi
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Univ.-Prof. DDr. Gregor Wenning
Priv.-Doz. Alexander Zimprich

LOKALE ORGANISATION

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Tagungsort

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Website

www.parkinson.at

ALLGEMEINE INFORMATIONEN

Fortbildungspunkte

Die Teilnahme an der Jahrestagung der Österreichischen Parkinson Gesellschaft wurde für den Erwerb des Fortbildungsdiploms der Österreichischen Ärztekammer approbiert.

Jahrestagung	08. – 10.11.2018	ID 629239	15 Punkte
Basal-Ganglien-Seminar mit Videos	08.11.2018	ID 630935	1 Punkt
Basics in Diagnostik und Therapien von Dystonien	10.11.2018	ID 629241	1 Punkt
Grundlagen der Diagnostik und Differenzialdiagnose von Ataxien	10.11.2018	ID 629244	1 Punkt
Neue klinische Diagnosekriterien und Biomarker der Parkinsonkrankheit	10.11.2018	ID 629247	1 Punkt
Management neuropsychiatrischer Probleme beim M. Parkinson	10.11.2018	ID 629251	1 Punkt
Therapie nicht-motorischer Symptome der Parkinson-Krankheit	10.11.2018	ID 629255	1 Punkt
Differenzialdiagnose von Tremor-Syndromen	10.11.2018	ID 629258	1 Punkt
Zertifizierungskurs Botulinumtoxin	10.11.2018	ID 624512	4 Punkte

ALLGEMEINE INFORMATIONEN

Die Fortbildungspunkte werden, der Fortbildungssatzung des Bundeslandes entsprechend, auf dem Punktekonto gutgeschrieben.
Bitte denken Sie an Ihren Barcode-Sticker der Ärztekammer (falls vorhanden) und tragen Sie sich während der Veranstaltung in die DFP Punkteliste bei der Registrierung ein.

Teilnahmegebühren

Kongressteilnahme inkl. Crash-Kurse am Samstag, 10.11.2018

Mitglieder	
Facharzt/Fachärztin	€ 120,00
in Ausbildung *	€ 60,00

Nicht-Mitglieder	
Facharzt/Fachärztin	€ 170,00
in Ausbildung *	€ 110,00
Dipl. Pflegepersonal, TherapeutInnen, PsychologInnen	€ 110,00
Karenzierte KollegInnen (aus Kindererziehungsgründen), Studierende *	kostenlos

*Bitte bringen Sie für Vor-Ort-Anmeldungen einen entsprechenden Nachweis zum Registrierungsschalter in Linz mit.

ALLGEMEINE INFORMATIONEN

Kursgebühren

Die Kurse finden am Samstag, 10.11.2018 in den Hörsälen des Ausbildungszentrums am Med Campus V statt und sind in den Teilnahmegebühren zur Jahrestagung inkludiert.

Zertifizierungskurs der ÖDBAG (Botulinumtoxin Zertifizierungskurs)	
Kursgebühr	€ 30,00

Crash-Kurse ohne Kongressteilnahme	
Teilnahme an 3 Kursen	€ 100,00

Nur Kursbuchungen ohne Kongressteilnahme an: **parkinson@cmi.at**

Kongressunterlagen

Sie erhalten das Kongressprogramm gemeinsam mit Ihrem Namensschild vor Ort an der Registrierung im Kepler Universitätsklinikum. Ihr Namensschild gilt als Eintrittsausweis zum wissenschaftlichen Programm und ist innerhalb des Veranstaltungsortes gut sichtbar zu tragen.

WLAN

WLAN steht Ihnen im Ausbildungszentrum am Med Campus V kostenfrei zur Verfügung.
Für die Verbindungen wählen Sie bitte den „KUK-Hotspot“ aus und stimmen anschließend im Browser den Allgemeinen Bedingungen zu.

PROGRAMM

PROGRAMM/*PROGRAM*

DONNERSTAG, 8.11.2018/*THURSDAY NOV. 8, 2018*

Mehrzwecksaal

8:30-10:30 **HAUPTTHEMA 1/*MAIN TOPIC 1***

Neues zu atypischen Parkinson-Syndromen - Klassifikation, Diagnostik, Therapien

Update on atypical Parkinson syndromes - classification, diagnostics, therapies

Vorsitz/*Chair*: E. Auff (Wien), W. Struhal (Tulln)

Neuropathologische Klassifikation
E. Gelpi (Barcelona/Wien)

Progressive Supranuclear Palsy
G. Höglinger (München)

Multisystem Atrophie-Pathophysiologie und typische sowie atypische Präsentationen
G. Wenning (Innsbruck)

Progressives autonomes Versagen: ein Prädiktor für M. Parkinson – Kurzvortrag
A. Fanciulli (Innsbruck)

Autonome Störungen bei Taupathien: ein Update – Kurzvortrag
W. Struhal (Tulln)

10:30-11:00 **Kaffeepause und Besuch der Industrieausstellung/ *Coffee break and visit of the industrial exhibition***

PROGRAMM/*PROGRAM*

11:00-11:30 **THEMENVORTRAG 1,** unterstützt von UCB
Motorische und nicht-motorische Parkinson-Symptome in der zweiten Tageshälfte
G. Ransmayr (Linz)

11:30-13:00 **FREIE VORTRÄGE 1 V1 - V6/FREE LECTURES 1 V1 - V6**
Vorsitz/Chair: K. Seppi (Innsbruck), K. Wenzel (Graz)

V1 Alpha-synuclein propagation in Multiple System Atrophy: a systematic review of literature
N. Campese (Pisa)

V2 Early distinction of Parkinson-variant multiple system atrophy from Parkinson's disease
A. Fanciulli (Innsbruck)

V3 Tilt table testing in MSA-P and PD: preliminary findings
F. Leys (Innsbruck)

V4 Characterization of REM sleep without atonia in MSA and IPD
C. Kaindlstorfer (Innsbruck)

V5 Test-Retest-Reliabilität des Toronto Clinical Scoring Systems bei Patienten mit idiopathischem Parkinson-Syndrom
C. Homann (Graz)

V6 Quantification of tremor severity with a mobile tremor pen
T. Zechmeister (Klagenfurt)

PROGRAMM/*PROGRAM*

13:00-14:00 **Mittagspause und Besuch der Industrieausstellung/**
Lunch break and visit of the industrial exhibition

14:00-16:00 **HAUPTTHEMA 2 /*MAIN TOPIC 2***
Interdisziplinäre Aspekte von Parkinson-Syndromen/
Interdisciplinary aspects of Parkinson syndromes
Vorsitz/Chair: S. Bösch (Innsbruck), T. Sycha (Wien)

Neurourologische Störungen bei Parkinson-Syndromen
G. Kiss (Innsbruck)

Microbiom und Verdauung
F. Scheperjans (Helsinki)

Neuroophthalmologische Störungen bei
Parkinson-Syndromen
M. Kögl (Graz)

Schluckstörungen
T. Warnecke (Münster)

16:00-16:30 **Kaffeepause und Besuch der Industrieausstellung/**
Coffee break and visit of the industrial exhibition

PROGRAMM/*PROGRAM*

16:30-17:30 **SATELLITENSYMPOSIUM 1,** unterstützt von GRÜNENTHAL
Apomorphin in der Therapie des Morbus Parkinson
Vorsitz/Chair: W. Poewe (Innsbruck)

Apomorphin - ein Parkinson-Mittel mit einer langen Geschichte
W. Poewe (Innsbruck)

Apomorphin Infusion bei motorischen Fluktuationen - erste randomisierte, Placebo-kontrollierte Studie
R. Katzenschlager (Wien)

17:30 -18:00 **Generalversammlung der Österreichischen Parkinson Gesellschaft**

18:00-19:00 **BASAL-GANGLIEN-SEMINAR MIT VIDEOS/**
BASAL-GANGLIA-SEMINAR INCLUDING VIDEOS
Vorsitz/Chair: P. Schwingenschuh (Graz),
A. Djamshidian-Teherani (Innsbruck)

PROGRAMM/*PROGRAM*

FREITAG, 9.11.2018/*FRIDAY NOV. 9, 2018*
Mehrzwecksaal

8:30-10:00 **HAUPTTHEMA 3 /*MAIN TOPIC 3***
Neue Therapien und Therapiekonzepte für Bewegungsstörungen
New therapies and therapeutic concepts in movement disorders
Vorsitz/Chair: R. Katzenschlager (Wien), W. Pirker (Wien)

Neue krankheitsmodifizierende Therapieansätze für die Parkinson-Krankheit
W. Poewe (Innsbruck)

Deep Brain Stimulation
J. Volkmann (Würzburg)

Neurorehabilitation
G. Ebersbach (Beelitz-Berlin)

10:00-10:30 **Kaffeepause und Besuch der Industrieausstellung/**
Coffee break and visit of the industrial exhibition

10:30-11:45 **SATELLITENSYMPOSIUM 2**, unterstützt von ABBVIE
Motorische und nicht-motorische Aspekte der fortgeschrittenen Parkinson Erkrankung
Vorsitz/Chair: G. Ransmayr (Linz)

Motorische Probleme beim fortgeschrittenen Morbus Parkinson und Indikationen für invasive Therapien
W. Pirker (Wien)

Nicht-motorische Symptome der Parkinson-Krankheit
A. Djamshidian-Tehrani (Innsbruck)

Levodopa-Carbidopa intestinale Gel (LCIG) Therapie: Fallbeispiele
L. Kellermair (Linz)

11:45-13:00 **FREIE VORTRÄGE 2 V7 - V12/*FREE LECTURES* 2 V7 - V12**
Vorsitz/Chair: G. Wenning (Innsbruck),
P. Katschnig-Winter (Graz)

V7 Nigral iron load as diagnostic parameter in Parkinson's Disease
S. Franthal (Graz)

V8 Elevated iron load in globus pallidus in patients with cervical dystonia
S. Franthal (Graz)

V9 A clinical and pathological study of small fibers in Friedreich´s Ataxia
E. Indelicato (Innsbruck)

V10 Caregiver Burden in patients with progressive supranuclear palsy and corticobasal syndrome
L. Kellermair (Linz)

V11 Associations of gait disorders and future falls in the elderly: a prospective population-based study
K. Marini (Innsbruck)

V12 Eye tracking in patients with restless legs syndrome with and without augmentation – interim results of an ongoing study
P. Ellmerer (Innsbruck)

PROGRAMM/*PROGRAM*

13:00-14:00 **Mittagspause und Besuch der Industrieausstellung/**
Lunch break and visit of the industrial exhibition

14:00-16:00 **HAUPTTHEMA 4/MAIN TOPIC 4**
Was gibt es Neues zu Bewegungsstörungen?
What's new in movement disorders?
Joint Session between the Austrian Parkinson Society and the International Parkinson and Movement Disorder Society - European Section
Vorsitz/Chair: W. Poewe (Innsbruck), A. Zimprich (Wien)

Parkinsonism and heavy metal exposure
P. Taba (Tartu)

What's new in chorea?
K. Seppi (Innsbruck)

Gait disorders – a clinician's approach
E. Ruzicka (Praha)

What's new in ataxia?
S. Bösch (Innsbruck)

16:00-16:30 **Kaffeepause und Besuch der Industrieausstellung/**
Coffee break and visit of the industrial exhibition

16:30-18:00 **SATELLITENSYMPOSIUM 3,** unterstützt von ZAMBON
Safinamid – Neue Option für die Therapie der Parkinson-Krankheit
Vorsitz/Chair: W. Poewe (Innsbruck)

Erfahrungen mit Safinamid in der Praxis –
aktuelle Real-World-Daten
H. Reichmann (Dresden)

Safinamid als Add-on: Wirkungen auf motorische und
nicht-motorische Symptome
R. Katzenschlager (Wien)

Potenzial von Safinamid beim Morbus Parkinson –
Überblick über die Daten klinischer Studien
W. Poewe (Innsbruck)

18:00-18:10 **IN MEMORIAM PROF. GERALD STERN**
W. Poewe (Innsbruck)

18:10-19:10 **HONORARY LECTURE**
Vorsitz/Chair: E. Auff (Wien)

Concepts and hypotheses about the etio-pathophysiology
of Parkinson's disease
E. Hirsch (Paris)

Überreichung der Ehrenmitgliedschaft der Österreichischen
Parkinson Gesellschaft /
Presentation of the honorary membership
of the Austrian Parkinson Society

19:10-19:20 **ÜBERREICHUNG DES WISSENSCHAFTSPREISES /**
PRESENTATION OF THE SCIENTIFIC AWARD
ENDE DES WISSENSCHAFTLICHEN PROGRAMMES /
END OF THE SCIENTIFIC PROGRAM
VERABSCHIEDUNG / *FAREWELL*

PROGRAMM/*PROGRAM*

SAMSTAG, 10.11.2018/*SATURDAY NOV. 10, 2018*

8:00-11:30 **FORTBILDUNGSAKADEMIE/CONTINUING EDUCATION**

8:00-9:00 Basics in Diagnostik und Therapien von Dystonien
Hörsaal 1
P. Schwingenschuh (Graz)

Grundlagen der Diagnostik und Differenzialdiagnostik von Ataxien
Hörsaal 3
S. Bösch (Innsbruck), A. Eigentler (Innsbruck)

9:15-10:15 Neue klinische Diagnosekriterien und Biomarker der Parkinson-Krankheit
Hörsaal 1
H. Stockner (Innsbruck)

Management neuropsychiatrischer Probleme beim M. Parkinson
Hörsaal 3
R. Katzenschlager (Wien)

10:30-11:30 Therapie nicht-motorischer Symptome der Parkinson-Krankheit - ein Evidence-based medicine-Update der Int. Parkinson und Movement Disorder Society
Hörsaal 1
G. Ransmayr (Linz)

Differenzialdiagnose von Tremor-Syndromen
Hörsaal 3
W. Pirker (Wien)

12:00-15:00 **ZERTIFIZIERUNGSKURS BOTULINUMTOXIN**

Hörsaal 1
Leitung: P. Schnider (Wiener Neustadt, Hochegg),
T. Sycha (Wien)
Modul Autonom

Autonom – 1 und 2

- Botulinumtoxin und die autonome Synapse
- Systemische autonome Nebenwirkungen
- Autonomes Indikationsspektrum
- Formen der fokalen Hyperhidrose
- Pathologische autonome Innervation (Frey Syndrom, Krokodilstränen)
- Behandlungsmöglichkeiten
- Stellenwert der BTX Behandlung
- Klassifikation und Dokumentation
- Behandlung der axillären und palmaren Hyperhidrose mit Botulinumtoxin Typ A
- Dosierungsrichtlinien und praktische Hinweise
- Behandlung anderer fokaler Hyperhidrosen

Autonom – 3

- Ursachen der Hypersalivation
- Behandlungsmöglichkeiten
- Stellenwert der BTX Behandlung
- Durchführung, Dosierung, Behandlungstechniken
- Klassifikation und Dokumentation

Autonom – 4

- Einführung in die Versorgung der Blase
- Idiopathische und neurogene Blasenstörung
- Patientenselektion

ABSTRACTS

DONNERSTAG, 08. NOVEMBER 2018

V1

Alpha-synuclein propagation in Multiple System Atrophy: a systematic review of literature

Campese N.[1], Stefanova N.[2], Wenning G. K.[2]

[1] Neurology Unit, Department of Clinical and Experimental Medicine, University of Pisa – Pisa, Italy
[2] Department of Neurology, Medical University of Innsbruck – Innsbruck, Austria

Background:
Multiple System Atrophy (MSA) is an orphan oligodendroglial alpha-synucleinopathy of unknown etiology. Misfolded α-synuclein aggregates seem to play a central role in promoting neurodegeneration; nevertheless mechanisms leading to α-synuclein misfolding and spreading still remain unclear and no target therapies have been accordingly developed.

Objectives:
We aim to analyze α-synuclein propagation patterns in MSA, whose characterization may help in identifying potential targets for MSA-specific disease-modifying interventions.

Methods:
We performed a Medline-based systematic review of literature. Only papers written in English and published in peer-reviewed journals were included.

Results:
In all α-synucleinopathies misfolded proteins seem to propagate in a "prion-like" fashion. In MSA more complex disease-specific interplays between neurons and glia could explain GCIs accumulation and neurodegeneration. Mechanisms underlying these interplays should be further explored to promote disease-specific therapeutic strategies.

Conclusions:
Multiple System Atrophy (MSA) is a fatal neurodegenerative disorder of unknown etiology, for which no disease-modifying therapies are currently available. Pathological hallmarks of the disease are misfolded α-synuclein aggregates (Glial Cytoplasmatic Inclusion, GCIs) selectively, though not exclusively, accumulating within glial cells.
Recently a "prion-like" α-synuclein seeding mechanism has been postulated for Parkinson's Disease, Dementia with Lewy Bodies and Multiple System Atrophy. Evidence of cell-to-cell spreading has been detected in both mice and human brains. In MSA more complex interplays between neurons and glia seem anyway to be involved

in GCIs' formation. α-synuclein misfolding seems in fact to begin in neurons, where it leads to the organization of monomers, oligomers and fibrils that are subsequently internalized by oligodendrocytes. Under the influence of glial cells' specific intracellular factors, aggregates undergo further modifications, turning into more aggressive strains, able to further propagate. Mechanisms of aggregates' cell-to-cell transfer may include exosomes formation, endocytosis, direct binding to outer membrane, membrane permeabilization.
In conclusion α-synuclein misfolding and propagation in MSA seem to occur according to disease-specific mechanisms that deserve a better characterization as potential key targets for tailored disease-modifying interventions.

References:
Fanciulli A et al., N Engl J Med 2015
Reyes JF et al., Glia 2014
Peng C et al., Nature 2018
Jellinger K et al., J Alzheimers Dis 2018

V2

Early distinction of Parkinson-variant multiple system atrophy from Parkinson's disease

Fanciulli A.[1], Goebel G.[2], Lazzeri G.[3], Scherfler C.[1], Gizewski E. R.[4], Granata R.[1], Kiss G.[5], Strano S.[6], Colosimo C.[7], Pontieri F. E.[8], Kaufmann H.[9], Seppi K.[1], Poewe W.[1], Wenning G. K.[1]

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Background:
Distinguishing the Parkinson variant of multiple system atrophy (MSA-P) from Parkinson's disease (PD) is often difficult at the disease beginning. This is a major drawback for counselling of patients and enrollment in disease-modifying clinical trials. Autopsy-confirmed studies suggest that autonomic failure and postural instability emerge earlier in MSA-P than PD and may thus represent early clinical warning signs.

Objectives:
To determine the diagnostic yield of early-onset autonomic failure and postural instability in differentiating MSA-P from PD.

Methods:
We retrospectively studied 186 parkinsonian patients who had undergone a cardiovascular autonomic function test at early disease (Hoehn & Yahr stage <3 and/or disease duration <2 years). Established PD and MSA-P diagnostic criteria were applied at last visit and served as clinical gold-standard. The clinical diagnosis was further substantiated by means of cerebral MRI-volumetry.
Clinical features associated with a diagnosis of MSA-P at last visit were investigated and a MSA-P diagnostic probability score was generated on the basis of discriminant variables.

Results:
Twenty-seven patients with MSA-P and 159 patients with PD composed the study cohort. At early disease, the presence of orthostatic hypotension (OR: 7.056, p=0.006), symptoms of overactive-bladder (OR: 7.077, p=0.007), urinary retention (OR: 5.585, p=0.019) and postural instability (OR: 21.743, p=0.004) predicted MSA-P at last visit. By assigning 1 point per abovementioned feature, a cumulative score ≥2 (score range: 0-4) showed 78% sensitivity and 86% specificity for a final diagnosis of MSA-P.

Conclusions:
We conclude that: i. not the simple presence, but the early onset of autonomic failure and postural instability distinguishes MSA-P from PD; ii. the combination of multiple warning signs in patients with early parkinsonism points towards a MSA-P diagnosis.
The 4-points MSA-P probability score provides clinicians with a cost- and time-effective screening tool to distinguish patients at no or low risk of having MSA-P (0-1 points) from those at high-risk (2-4 points), who may benefit from further investigations and ultimately recruitment in ongoing disease-modifying studies.

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V3

Tilt table testing in MSA-P and PD: preliminary findings

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Background:
Multiple system atrophy (MSA) and Parkinson's disease (PD) are sporadic neurodegenerative diseases of the adulthood characterized by accumulation of misfolded α synuclein. Especially at disease beginning, it is difficult to differentiate the Parkinson variant of MSA (MSA-P) from PD due to overlapping features, such as neurogenic orthostatic hypotension (nOH). Neuropathological studies showed that the site of lesion of the autonomic nervous systems is central in MSA-P and peripheral in PD. As of today, it is controversial whether MSA-P and PD can be differentiated by the means of tilt table testing (TTT).

Objective:
In this retrospective study we aim to determine whether routine TTT discriminates between MSA-P and PD by means of an expert-based blinded evaluation or a systematical comparison of hemodynamic parameters.

Methods:
We included 22 patients with nOH (11 MSA-P, 11 PD), aged 55 to 80, with either clinically probable MSA-P according to current consensus criteria (n=11, Gilman, 2008) or clinically definite PD according to Queen Square diagnostic criteria (n=11, Lees, 2009). In this preliminary phase, two physicians with expertise in TTT were blinded to the neurological diagnosis and had to try hitting the correct diagnosis (MSA-P or PD) by applying a self-created evaluation scheme to the tilt table recordings under continuous noninvasive blood pressure (BP) monitoring (Task Force™ Monitor, CNSystems).

Results:
Rater 1 evaluated 22 out of 22 cases (11 MSA-p, 11 PD) by applying a 6-item evaluation scheme of MSA-P typical aspects (age <65, resting heart rate >70 bpm, moderate to severe neurogenic supine hypertension, orthostatic BP fall >30/15 mmHg, missing BP overshoot at late phase II and phase IV of Valsalva maneuver (VM), pathological VM and deep breathing (DB)). If the patient scored >3 points, the diagnosis MSA-P was assigned. Rater 1 correctly predicted MSA-P with 45.45% sensitivity, 72.73% specificity, 62.50% positive predictive value (PPV) and 57.14% negative predictive value (NPV). Diagnostic accuracy was 59.09%.
Rater 2 evaluated 20 out of 22 cases (10 MSA-P, 10 PD) by applying a 4-item evaluation scheme of MSA-P typical aspects (orthostatic BP fall >30/15 mmHg, rise of heart rate

>10 within 3 minutes after tilt, missing BP overshoot at late phase II and phase IV of VM, pathological VM and DB). Rater 2 correctly predicted MSA-P with a sensitivity, specificity, PPV and NPV of 30%. Diagnostic accuracy was 30%.

Conclusions:
Our preliminary results suggest that tilt table testing is useful in assessing the presence and severity of cardiovascular autonomic failure but is of limited value in the differential diagnosis between MSA-P and PD once nOH is present. Combining multiple parameters into a probability score seems to achieve a higher diagnostic accuracy.

V4

Characterization of REM sleep without atonia in MSA and IPD

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Background and Study Objectives:
Rapid eye movement (REM) without atonia (RWA) and REM sleep behavior disorder (RBD) are commonly associated with multiple system atrophy (MSA) and idiopathic Parkinson´s disease (IPD). The aim of the current investigation was to determine the characteristics of RWA in MSA and IPD patients with RBD and to evaluate associations between RWA and demographic or clinical characteristics.

Methods:
We performed a cross sectional study of 20 patients with a diagnosis of MSA (n=7) or IPD (n=13) and a history of sleep disturbances. All patients underwent a clinical evaluation and 2 nights of video-polysomnography followed by a multiple sleep latency test. Demographic characteristics, medical history, course and severity of disease, as well as data from RBD questionnaires, sleep quality and daytime sleepiness were collected. Phasic, tonic and "any" EMG muscle activities were determined in chin and limb muscles with an integrated software algorithm after artifact correction and were compared between patients with (IPD and MSA) and without RBD (IPD). Receiver operating characteristic curves were applied to evaluate the best cutoff thresholds for a diagnosis of RBD in the current cohort and regression was used to explore associations with clinical variables.

Results:
All mean RWA measures were higher in patients with than without RBD ($p<0.01$), except for phasic chin and tibial EMG measures. The highest discriminative power was achieved by the phasic EMG activity in the flexor digitorum superficial muscle (FDS) and the sternocleidomastoid muscle (SCM) (sensitivity and specificity 100%; $p<0.001$) and the combination of "any" EMG activity in the mental muscle and phasic EMG activity in bilateral FDS/SCM/chin channels (sensitivity and specificity 92-100%; $p\leq0.001$). Mental, submental and tibial channels alone were insufficient in differentiating RBD patients from patients without RBD. No differences in RWA measures were observed between IPD and MSA patients with RBD. Patients with RBD demonstrated significantly higher PLMS indices during total sleep time and during REM as compared to IPD patients without RBD ($p<0.05$).

Conclusions:
Phasic mental/submental EMG activity alone is insufficient to identify RBD in patients with IPD and MSA, whereas the combination of "any" mental and phasic FDS/SCM or chin EMG activity reliably detects RBD in these patients. This suggests that upper limb (FDS) and SCM EMG channels should be implemented in the polysomnographic assessment of patients with neurodegenerative diseases.

V5

Test-Retest-Reliabilität des Toronto Clinical Scoring Systems bei Patienten mit idiopathischem Parkinson-Syndrom

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Hintergrund:
Polyneuropathie (PNP) wird zunehmend als typisches Syndrom im Zusammenhang mit dem Idiopathischem Parkinsonsyndrom (IPS) anerkannt. Obwohl die Nervenleitgeschwindigkeitsmessung (NLG) als Goldstandard gilt, wird aufgrund von Praktikabilität die PNP häufig mit Hilfe von PNP-Skalen diagnostiziert und bewertet. Im Gegensatz zu anderen IPS-Symptomen gibt es jedoch für PNP bei IPS keine gründlich validierten Skalen.

Fragestellung:
Diese Studie dient der Untersuchung der Test-Retest-Reliabilität des Toronto Clinical Scoring-Systems (TCSS), eine der am häufigsten verwendeten PNP-Skalen.
Methode:

14 konsekutive IPS-Patienten, die innerhalb eines Monats an unserer Klinik ambulant oder stationär vorgestellt wurden, wurden mit dem TCSS getestet und einer NLG unterzogen. Jeder Patient wurde für die Inter-Rater- Reliabilitätsprüfung am selben Tag von 2 Untersuchern (CNH und PM) getestet und dann für die Intra-Rater-Reliabilitätsprüfung innerhalb von 24 Stunden von einem Untersucher (PM) erneut getestet. Die Untersucher waren verblindet in Bezug auf die Skalenwerte des jeweils anderen, und das Ergebnis der NLG.

Ergebnisse:
Ein Patient konnte aufgrund ausgeprägter kognitiver Defizite nicht zuverlässig getestet werden und wurde ausgeschlossen. Von den verbleibenden 13 IPS Patienten, 6 Männern und 7 Frauen, im Alter von 70,69 Jahren, mit mittlerem Krankheitsschweregrad (UPDRS-ME: 21,69), wurden 10 mittels NCS als PNP diagnostiziert. Neun davon wurden mittels TCSS richtig erkannt. Das TCSS zeigte gute Praktikabilität, ausgezeichnete Sensitivität (100%) moderate Spezifizität (69,2%), mittlere Intra-Rater- Reliabilität (Cronbachs Kappa = 0,494, $p < 0,001$), aber nur eine bescheidene Inter-Rater- Reliabilität (Cronbachs Kappa = 0,262, $p = 0,002$).

Zusammenfassung:
Unsere Ergebnisse legen nahe, dass sich das TCSS für Studien zur Erfassung der PNP bei IPS Patienten eignet. Allerdings sollte die Testung durch ein und denselben Tester durchgeführt werden. Für eine definitive Aussage wären größere Studien sinnvoll und begrüßenswert.

V6

Quantification of tremor severity with a mobile tremor pen

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Background:
Quantifying tremor severity is a challenging procedure. Standardized questionnaires for assessing tremor are subjective, available objective devices have a high complexity or are immobile. The evaluation of tremor intensity is however necessary to document the general course of disease and to adjust individual therapy.

Objectives:
The aim of the study was to analyze the following questions: (1) What differences of clinical significance emerge if tremor is quantified in a wearable constellation (M2) and a hand-held constellation (M1) (2) Which clinical significance does the Total Power of a tremor measurement provide in contrast to the Power of Main Peak (3) Is an accelerometer-based or gyroscope-based tremor measurement more reliable?

Methods:
Tremor measurements were made with the Tremitas system in 14 patients with Parkinson's disease tremor (PD) (assessment of rest tremor) and 16 patients with essential tremor (ET) (assessment of postural tremor). The Tremitas system is a pen-shaped sensor that can capture tremor and calculate relevant tremor parameters: The Power of Main Peak (PMP), the Peak Frequency (PF) and the Total Power (TP).

Results:
The study showed that tremor severity assessed by the Tremitas System significantly correlated with tremor severity in the relevant subitems of the MDS-UPDRS (Part III Subscores 3.17 + 3.18 (sum)) and TETRAS (Performance Part –Point 4a right and left (sum)) ($r = 0.644 - 0.797$). Ad (1): There was no significant difference between M1 and M2 constellations in PD and ET ($p>0.05$). (2) TP is slightly less robust as a tremor indicator (Difference of $R = 0.07$ on average) than PMP, but is still above the $r= 0.6$ threshold. (3) The gyroscope sensor was superior within a wearable constellation, while the accelerometer was superior in a handheld constellation.

Conclusions:
We showed that a handheld tremor quantification tool with an accelerometer is a robust method to quantify rest tremor in PD and postural tremor in ET. PMP and TP were found useful as surrogate markers for tremor amplitude. Further investigations in a home-monitoring environment are being prepared.

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V7

Nigral Iron Load as Diagnostic Parameter in Parkinson's Disease

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Background:
Nigral iron deposition is considered as an important trigger for oxidative stress and neuro-degeneration. Elevated nigral iron load has been described in Parkinson's disease (PD) patient's in histological and imaging analyses.

Objectives:
To determine the diagnostic value of nigral iron depositions in PD, measured by iron specific MRI-techniques.

Methods:
We included 47 patients with clinically diagnosed PD (33 M/ 14 F; age 63. 3±9.7 years; mean disease duration 3.5±0.5 years) and 32 age-matched healthy controls (HC). All subjects underwent a 3T cerebral MRI protocol, as well as detailed clinical examinations. To measure nigral iron load we performed R2*-relaxometry and Quantitative Susceptibility Mapping (QSM) in total substantia nigra (SN) as well as in pars compacta (SNc) and pars reticulata (SNr) separately. Regions of interest were drawn manually on an iron independent contrast. Group differences were calculated by t-test (for normally distributed variables), otherwise by Mann-Witney-test. For determination of diagnostic value, we performed ROC-analyses and for clinical correlations Pearson correlations.

Results:
Groupe differences and ROC analyses are summarised in in the following table:

	QSM		R2*	
	T-test/ MWU p-value	ROC - area under the curve	T-test/ MWU p-value	ROC - area under the curve
Left SNc	< 0.001	0.734	0.058	0.647
Left SNr	< 0.001	0.763	0.005	0.688
Left total SN	< 0.001	0.802	0.005	0.686
Right SNc	< 0.001	0.774	0.003	0.696
Right SNr	< 0.001	0.828	0.001	0.713
Right total SN	< 0.001	0.856	<0.001	0.735
Bilateral SNc	< 0.001	0.772	0.004	0.691
Bilateral SNr	< 0.001	0.828	0.001	0.719
Bilateral total SN	< 0.001	0.852	0.001	0.721

Bilateral total SN showed the best group discrimination with a sensitivity of 78.7% and a specificity of 78.1% for QSM and 68.1% and 65.6% for R2*.
For QSM and R2* we found moderate positive correlations between SN and Nonmotor Symptoms Scale and MDS-UPDRS.

Conclusions:
We found significantly higher iron load in SN in PD compared to HC and positive correlations with parkinsonian motor and nonmotor symptoms. QSM was superior to R2* considering diagnostic sensitivity and showed very good group discrimination. There were no relevant side-differences in nigral iron load and total SN showed better discrimination compared to the sub-regions. QSM values for bilateral total SN showed the highest sensitivity and specificity combination and might be used as diagnostic marker.

V8

Elevated Iron Load in Globus Pallidus in Patients with Cervical Dystonia

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Background:
The pathophysiology of idiopathic cervical dystonia (CD) is still poorly understood. Isolated focal dystonia is considered a primary disorder without any underlying structural brain changes. However, advanced MRI techniques have pointed to the existence of subtle brain abnormalities in CD, but reports are scarce and the results are partly inconsistent. Increased brain iron levels have been described in several movement disorders and a pilot study in12 female patients suggested higher R2* values in the globus pallidus (GP) in CD(1).

Objectives:
The objective of this study was to determine the presence of basal ganglia iron abnormalities in CD compared to healthy controls (HC).

Methods:
We included 37 patients with clinically diagnosed CD (15 M/ 22 F; age 57.6±14 years; mean disease duration 16.6±10. 8 years) and 37 age- and sex-matched HC. All subjects underwent a 3T cerebral MRI protocol, as well as detailed clinical examinations. For quantitative iron measurement we performed R2*-relaxometry and Quantitative Susceptibility Mapping (QSM). As regions of interest we defined caudate nucleus, thalamus, putamen, globus pallidus and substantia nigra. Group differences were calculated by t-test (for normally distributed variables), otherwise by Mann-Witney-test and results were corrected with Bonferroni correction for multiple comparisons. For clinical correlations we performed Pearson correlations.

Results:
CD patients showed significantly higher R2* ($p=0.025$) and QSM ($p=0.015$) values in bilateral GP. There were no significant group differences in other basal ganglia regions. There was no significant correlation between R2* or QSM in GP and disease duration, Burke-Fahn-Marsden scale and TSUI score.

Conclusions:
We found elevated R2* levels and for the first time susceptibility changes in the GP using QSM, which is regarded as more sensitive to iron. Future (longitudinal) studies need to clarify if the pallidal iron accumulation represents a pivotal event or an epiphenomenon in the pathophysiology of CD.

(1) Aschermann Z, Perlaki G, Orsi G, Nagy SA, Horvath A, Bone B, et al. Quantitative assessment of brain iron by R2* relaxometry in patients with cervical dystonia. Mov Disord 2015 Sep;30(10):1422-1426.

V9

A clinical and pathological study of small fibers in Friedreich´s Ataxia

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Background:
Friedreich Ataxia (FRDA) is a caused by homozygous GAA expansions in the FXN gene and typically affects large myelinated fibers in sensory nerves, dorsal root ganglia and posterior column in the spinal cords. Recent evidence suggests that also unmyelinated nerve fibers are affected. Herein, we aimed at investigating the small fiber pathology in FRDA.

Methods:
Genetically confirmed FRDA patients were consecutively enrolled at our ataxia outpatient clinic. The study protocol included: intraepidermal nerve fibers density (IENFD) evaluation, quantitative sensory testing (QST) and cardiovascular autonomic function testing. Symptoms of small fiber pathology were investigated through the SCOPA-aut and the Pain-DETECT questionnaire. Findings at clinical testing were compared with those of control healthy subjects.

Results:
Twenty-four FRDA patients were recruited (Age= 40±13 years). The IENFD in FRDA (15 patients, age=36±9 years) was significantly reduced compared to controls (5,8±4,68 vs 9,3±1,41 fibers/mm; p=0.01). In a multiple step regression analysis, age at examination and disease duration had no influence on IENFD while a correlation was found with the shorter GAA repeat (R^2=0.58, p=0.03). At QST, mechanical detection threshold and vibration perception were, as expected, severely impaired in FRDA. Among the temperature and pain perception parameters only the cold detection threshold was significantly delayed in FRDA compared to controls. Five patients reported neuropathic complaints, but scores at pain-DETECT remained under the threshold for likely neuropathic pain. Findings at cardiovascular autonomic testing (13 FRDA, Age=44±15) did not differ from that of controls, except for higher rest heart rate in FRDA (70±9 vs 61±8 beats/minute, p= 0,01). Though, several symptoms referred to other autonomic domains, particularly bladder function, thermoregulation and sweating, were reported. In a multiple regression analysis, SCOPA-aut scores showed a good correlation with

disease severity expressed by the ataxia scale SARA, while there was only a modest correlation with IENFD (data not shown).

Conclusions:
FRDA patients showed a heterogeneous small fibers loss. Clinically, subtle signs of impairment in protopathic sensory modalities and autonomic function were observed. The severity of epidermal denervation correlated with GAA repeat length in our cohort, thus supporting a genetically determined small fiber loss in FRDA.

V10

Caregiver Burden in patients with progressive supranuclear palsy and corticobasal syndrome

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Background:
Progressive supranuclear palsy (PSP) and corticobasal syndrome (CBS) are tauopathy spectrum disorders. Because of much worse prognosis of PSP and CBS compared to Parkinson's disease (PD) early caregiving and palliative care are important, which may cause financial, health and psychological burden and strain including depression and other health problems. In contrast to PD little is known about the burden and strain of caregiving in PSP and CBS.

Objectives:
Analysis of caregiving in PSP and CBS patients, caregiver burden, and known and hypothesized underlying factors related to the patients and the caregivers (CGs)

Methods:
We diagnosed 46 patients with probable PSP (45 PSP-Richardson, one Primary Gate Freezing Syndrome; Höglinger et al. 2017) and 13 patients with probable CBS (Armstrong et al. 2013) and their CGs. Besides demographic, educational, occupational and family-related parameters, subscale III of the Unified PD Rating Scale (UPDRS-III), Rivermead-Activities of Daily Living Scale (RADL), Barthel Index (BI), Frontal Behavioral Inventory (FBI; Kertesz et al. 1997), CERAD-plus and Mini Mental State Examination (MMSE), the Zarit Caregiver Burden Interview (CBI; Zarit et al. 1980) and the Caregiver Strain Index (CSI; Robinson 1983) were administered.

Results:
Results are presented as median; mean+standard deviations. Patient age was 72; 71.76+7.33, male:female ratio 32:27, disease duration 30; 36.3+25.7 months, UPDRS III sum-score (OFF) 36.5; 36.28+14.04, MMSE Z-score -1.55; 1.98+1.91, FBI sum-score 11; 14.92+13.38. CG age was 68; 64.56+11.37, CG male:female ratio 17:42. 37 CGs were partners, 14 first, and three second degree relatives. CBI sum-score (assessed in 41 CGs) was 23; 22.29+14.71, range 0-62. 17(41%) CGs reported a CB suggesting significant risk to mental health ("moderate to severe CB"; CBI sum-score >/= 26; Schreiner et al. 2006). CSI was 4; 2.93+2.74, range 0-10. CB correlated positively with duration of the disease ($p<0.05$), behavioral abnormalities (FBI) ($p<0.01$), motor impairment (UPDRS-III; $p<0.05$), and negatively with daily activity scores (RADL, BI; $p<0.01$, Pearson rank correl.). Female CB correlated positively with patient age ($p<0.05$) and disease duration ($p<0.05$), in contrast to male CGs, in whom CB correlated negatively with patient age ($p>0.05$) and cognitive impairment (CERAD; $p<0.05$). Comparison of CB to that of Alzheimer (from the PRODEM data bank) and PD patients as well as social determinants of CB will be reported.

Conclusions:
Mainly informal, female CGs, partners and first degree relatives (sons, daughters) are involved in caregiving for PSP and CBS patients in the catchment area (mainly Upper Austria). CB was found in early to moderately advanced PSP and CBS. CB correlates with behavioral abnormalities, motor impairment and cognitive decline, but was also related to demographic factors of patients and CGs. A risk score of CB should be developed to take measures against CB and to prevent CGs from mental, somatic and financial stress.

V11

Associations of gait disorders and future falls in the elderly: a prospective population-based study

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Background:
Recurrent falls are common in the general elderly population and represent a major source of serious adverse health outcomes such as fractures, institutionalization and death. Previous cross-sectional and longitudinal studies have linked the occurrence of gait disorders (GDs) to recurrent falls. However, there is only limited prospective data

with long-term follow-up on this association.
Objectives:
The aim of this prospective study was to investigate the association of GDs and quantitative gait measures with future falls in an existing longitudinal population-based cohort.

Methods:
The study included 342 subjects aged 59–93 years of the Bruneck Study cohort in a baseline and a 6-year follow-up assessment. At baseline, participants underwent a clinical qualitative gait evaluation (to determine neurological and non-neurological GDs) and an assessment of simple quantitative gait measures (Hauser Index, Tinetti gait and balance scale, and gait speed). Participants with recurrent falls at baseline were excluded from analysis (n=14). A logistic regression analysis adjusted for age and sex was performed to calculate odds ratios (OR) of baseline variables for future falls.
Results: At follow-up 22 subjects (6.7%) reported recurrent falls. GDs in general and neurological GDs in particular were predictive for future falls (OR 4.1; 95% confidence interval [CI] 1.6-10.9; p=0.005 for GDs in general and OR 4.85; 95%CI 1.6-15.0; p=0.006 for neurological GDs), while there was no association for non-neurological GDs with falls. All three simple gait tests were predictive for future falls (Hauser Index, p=0.002; Tinetti gait and balance scale, p=0.006; gait speed, p<0.001).

Conclusions:
Both, a neurological gait evaluation and simple gait tests have significant predictive value for falls assessed as long as 6 years after gait evaluation.

V12

Eye tracking in patients with restless legs syndrome with and without augmentation – interim results of an ongoing study

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Objective:
To assess cognitive flexibility in patients with restless legs syndrome (RLS) and healthy controls (HC) using an eye tracker.

Background:
RLS is a common neurological disease, affecting up to 10% of the population. Dopaminergic drugs, especially dopamine agonists, are commonly used to alleviate

symptoms. However, these drugs may induce unwanted side effects such as augmentation or impulse control disorders in a subgroup of patients (1). These side effects evolve gradually and may be initially difficult to diagnose.

Methods:
A total of 10 HC and 30 patients with idiopathic RLS diagnosed according to the International Restless Legs Syndrome Study Group criteria from our out-patient clinic and sleep laboratory were consecutively and prospectively enrolled. Data acquisition was done by using a Tobii eye tracking system and the Pro Lab software.

Results:
We found a significant group difference in the reaction times of the Go/NoGo task. HCs (299.96 ms ±109.33) and RLS+Aug (308.21 ms ±93.08) had significantly shorter reaction times than RLS (394.73 ms ±92.71)($p = .038$). When we performed a post-hoc analysis, there was no significant group difference. The reaction times in the other tasks did not differ significantly within the groups. Then, we analysed error rates and found a significant group difference in the antisaccade task (HC: .239 ±.167; RLS: .268 ±.249; RLS+Aug .503 ±.284; $p = .013$). Post-hoc analysis showed a significant difference between HC and RLS+Aug ($p = .033$) and RLS and RLS+Aug ($p = .048$). The main analysis of other tasks did not show a significant group difference. When we computed prosaccades and antisaccades separately in the task switching paradigm, RLS+Aug (.207 ±.187) made more errors than HCs (.151 ±.120) and RLS (.116 ±.221) in the prosaccades ($p = .049$). In the second trial, RLS+Aug (.433 ±.280) had a higher error rate than HCs (.204 ±.164) and RLS (.326 ±.181) in the antisaccades ($p = .05$).

Conclusions:
Our preliminary data show that patients with augmentation have difficulties suppressing overt attention towards the target in the antisaccade task. These frequent antisaccade errors in the augmentation group suggest a dysfunction of the frontostriatal network and have been observed in patients with Parkinson's disease as well as in patients with lesions of the dorsolateral prefrontal cortex. Kockler et al. were able to associate verbal learning and memory, executive function and impulsivity. The higher error rates in the task switching paradigm suggest an impairment of mental flexibility in RLS+Aug and are in line with these previous findings. Our results are also consistent with previous reports linking augmentation with impulse control disorders.

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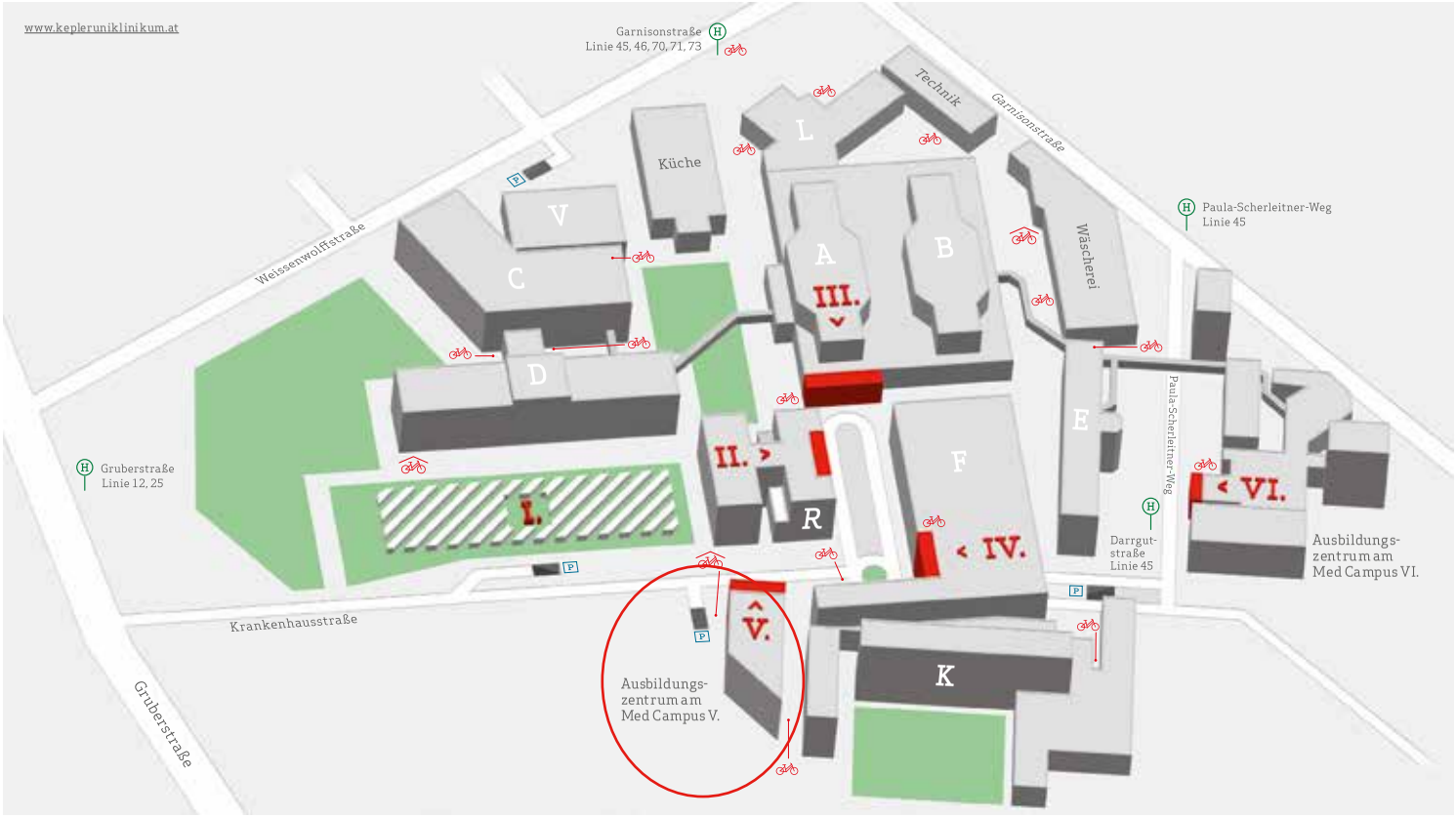
Verbindung zum Neuromed Campus:
Linz AG Buslinien 45 und 46 bis Haltestelle Mozartkreuzung > Straßenbahnlinien 1 oder 2 bis Haltestelle Unionkreuzung > Linz AG Buslinien 41 oder 43 bis Haltestelle Wagner-Jauregg-Weg



Übersichtsplan Med Campus

- I. In Planung: Lehr- und Forschungsgebäude der JKU
- II. Haupteingang MED CAMPUS II.
- III. Haupteingang MED CAMPUS III.
- IV. Haupteingang MED CAMPUS IV.
- V. Haupteingang MED CAMPUS V.
- VI. Haupteingang MED CAMPUS VI.
- Blutzentrale Rotes Kreuz
- Tiefgarage
- Haltestelle
- Fahrradabstellplatz
- Fahrradabstellplatz (versperrt, Zutritt nur mit MA-Karte, kameraüberwacht)

Kepler Universitäts Klinikum

NOTIZEN

NOTIZEN

JAHRESTAGUNG DER ÖSTERREICHISCHEN PARKINSON GESELLSCHAFT

www.parkinson.at

21.-23.11.2019

PARKHOTEL SCHÖNBRUNN
WIEN

SAVE THE DATE

ÖSTERREICHISCHE PARKINSONGESELLSCHAFT

The *Movement* Disorder Society