La Clinica Neurologica

Matteo Pardini

Person centred coordinated care: a definition

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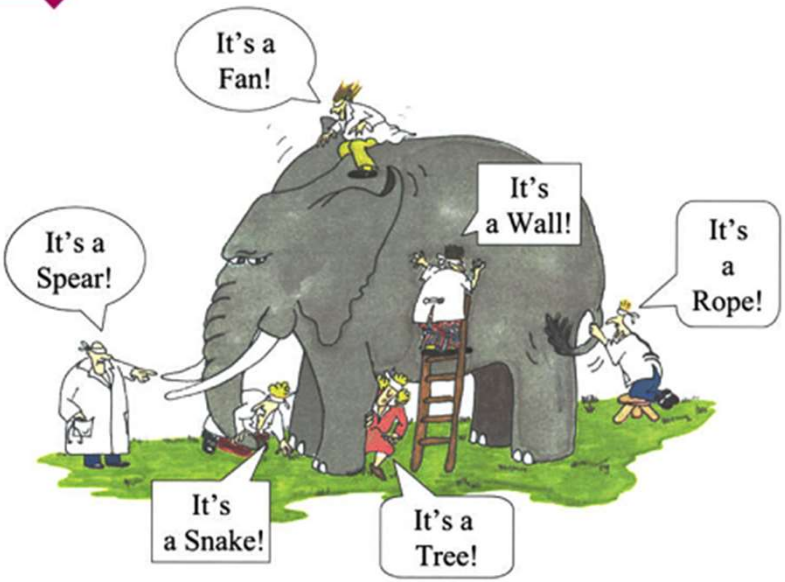


Decadimento cognitivo & neurologi	La Clinica Neurologica: quello che facciamo	La Clinica Neurologica: quello che vorremmo fare in futuro
Esame neurologico	Attenzione al confine tra disturbi movimento e disturbi cognitivi	Integrare le competenze con i *movement disorders specialists*
Biomarcatori fluidi (CSF, sangue) e neurofisiologici	Biomarcatori su CSF. EEG ed EEG nel sonno	Ampliare marcatori su CSF. Attivare marcatori su sangue. Ampliare repertorio neurofisiologico
Co-morbidità neurologica	Collaborazione con altri gruppi sugli aspetti di confine	Affrontare la complessità delle comorbidità in modo sempre più integrato

Tab. 1 Characteristics of different disorders with co-occurring parkinsonism and dementia

	Core syndromes	Supportive features	Primarily affected cognitive domains
Primary neurodegenerative disorders			
Parkinson's disease dementia	Parkinsonism Asymmetric onset of Parkinsonism Levodopa responsiveness Dementia	Slowly progressive cognitive decline Depression Visual hallucinations Change in personality Delusions Excessive daytime sleepiness	Executive function ++ Attention ++ Visuoconstructive abilities ++ Memory ++ Language +
Dementia with Lewy bodies	Dementia Symmetric parkinsonism Fluctuations Visual hallucinations	Cognitive decline prior to or within 1 year of motor worsening REM sleep behavior disorder Neuroleptics sensitivity Non-visual hallucinations Repeated falls and syncopes Autonomic dysfunction	Executive function ++ Visuoconstructive abilities ++ Language ++ Memory +
Progressive supra-nuclear palsy	Early onset of postural instability Bradykinesia Supranuclear gaze palsy Dementia	No levodopa sensitivity Apathy Depression	Executive function +++ Attention ++ Behavioral control ++ Visuoconstructive abilities ++
Corticobasal degeneration	Asymmetric akinetic-rigid syndrome Cortical senory loss Alien limb phenomenon Dementia	Levodopa insensitivity Disinhibition Depression	Limb-kinetic apraxia ++ Ideomotor apraxia ++ Executive function ++ Memory ++ Visuo-constructive abilities ++ Behavioral control +
Secondary parkinsonism and dementia			
Vascular parkinsonism	"Lower body" parkinsonism Apathy, bradyphrenia Positive frontal reflexes	"Atactic" gait Cardiovascular risk factors White matter lesions	Variable
Normal pressure hydrocephalus	Bradyphrenia "Magnetic gait" Urine incontinence	Hydrocephalus on neuroimaging Normal CSF pressure	Executive function ++ Attention ++ Visuoconstructive abilities ++ Language +
Drug-induced parkinsonism/ dementia	Variable	Variable	Variable

++ predominately affected, + affected.

(Llpelt-Scarfone et al., 2012)

Neuroimaging findings and clinical trajectories of Lewy-body disease in patients with MCI

Patient Nº	Age (yrs)	Gender	Education (yrs)	MMSE score	GDS-15 score
1	75.7	M	13	26	5
2	69.5	M	8	27	0
3	79.8	F	13	29	9
4	80.7	M	8	27	2
5	78.2	M	12	26	5
6	70.0	F	5	27	3
7	75.4	M	5	29	2
8	75.3	M	5	21	7
9	74.6	M	17	29	4
10	69.6	F	3	29	1
11	78.2	F	10	30	4
12	73.9	M	13	24	0
13	72.0	M	21	28	1
Mean	74.8	9M	10.3	27.1	3.3
SD	3.8	4F	5.3	2.5	2.7

(Massa et al., under revisions NoA)

Front Biosci (Landmark Ed). 2018 Jan 1;23:183-220.

EEG and ERP biomarkers of Alzheimer's disease: a critical review.

Horvath A[1], Szucs A[2], Csukly G[3], Sakovics A[2], Stefanics G[4], Kamondi A[2].

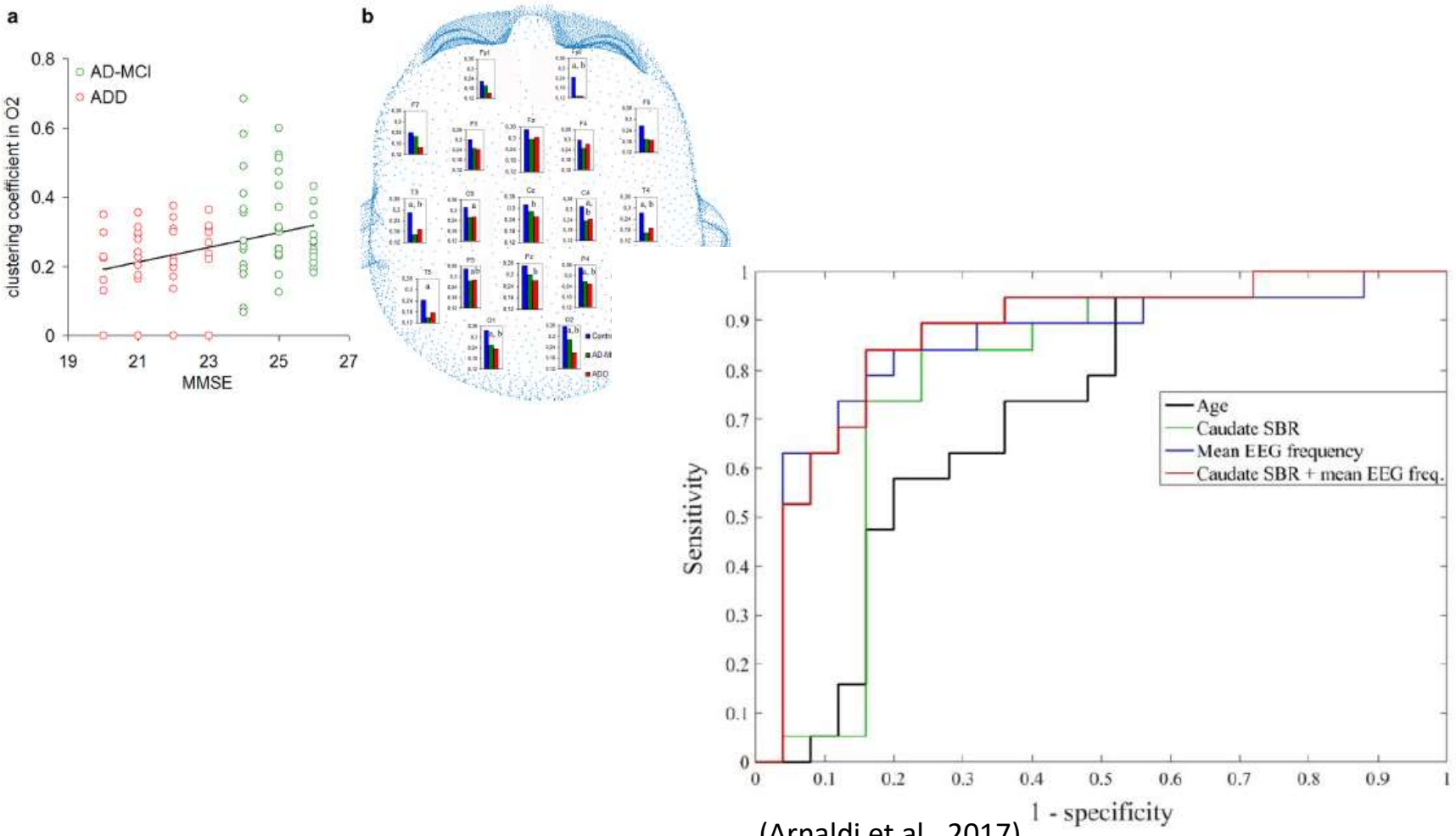
Author information

Abstract

Here we critically review studies that used electroencephalography (EEG) or event-related potential (ERP) indices as a biomarker of Alzheimer's disease. In the first part we overview studies that relied on visual inspection of EEG traces and spectral characteristics of EEG. Second, we survey analysis methods motivated by dynamical systems theory (DST) as well as more recent network connectivity approaches. In the third part we review studies of sleep. Next, we compare the utility of early and late ERP components in dementia research. In the section on mismatch negativity (MMN) studies we summarize their results and limitations and outline the emerging field of computational neurology. In the following we overview the use of EEG in the differential diagnosis of the most common neurocognitive disorders. Finally, we provide a summary of the state of the field and conclude that several promising EEG/ERP indices of synaptic neurotransmission are worth considering as potential biomarkers. Furthermore, we highlight some practical issues and discuss future challenges as well.

Cortical Network Topology in Prodromal and Mild Dementia Due to Alzheimer's Disease: Graph Theory Applied to Resting State EEG

Raffaella Franciotti[1] · Nicola Walter Falasca[1,2] · Dario Arnaldi[3,4] · Francesco Famà[3,5] · Claudio Babiloni[6,7,8] · Marco Onofrj[1] · Flavio Mariano Nobili[3,4] · Laura Bonanni[1]



(Arnaldi et al., 2017)

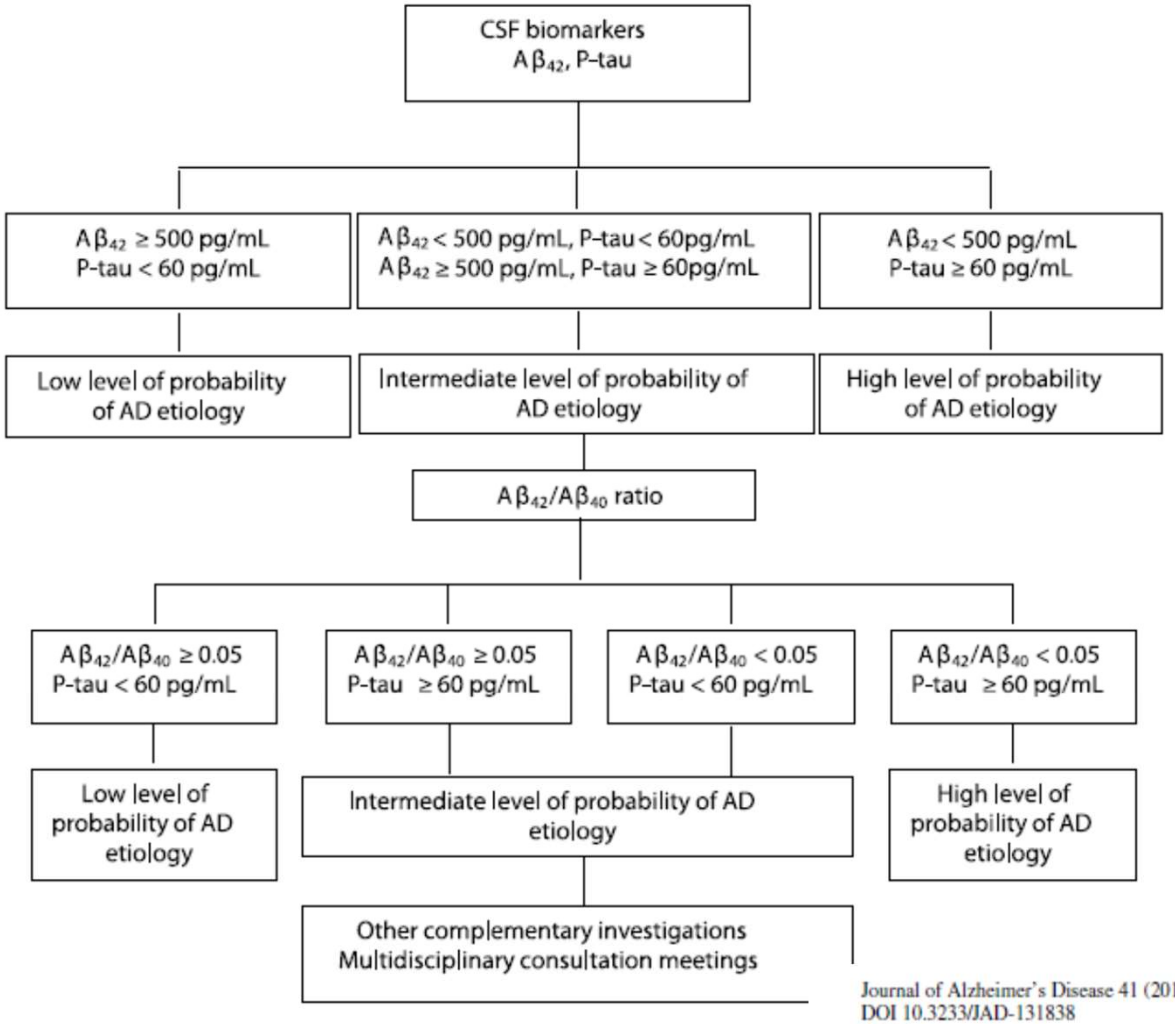


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Fig. 1. Decision tree for patient classification.

Laboratorio di Diagnostica Liquorale
U26L2

BIOMARKERS LIQUORALI PER MALATTIA DI ALZHEIMER

Proteina Amiloide Beta (Aβ 1-42): 888.2 pg/ml	v.n. > 600 pg/ml
Proteina Amiloide Beta (Aβ 1-40): 7212 pg/ml	
Rapporto Amiloide Beta 1-42/1-40 (Aβ 1-42/1-40): 0.12	v.n > 0.1
Proteina Tau totale (h-Tau): 346.5 pg/ml	v.n. < 400 pg/ml
Proteina Tau Fosforilata (P-Tau 181): 38.3 pg/ml	v.n. < 40 pg/ml

INTERPRETAZIONE: Il quadro neurochimico liquorale non è compatibile con AD.

Dr. Federico Massa
Medico in formazione specialistica

D.ssa Elisabetta Capello
Dirigente medico

Ultrasensitive RT-QuIC Seed Amplification Assays for Disease-Associated Tau, α-Synuclein, and Prion Aggregates

Eri Saijo, Bradley R. Groveman, Allison Kraus, Michael Metrick, Christina D. Orrù, Andrew G. Hughson, and Byron Caughey

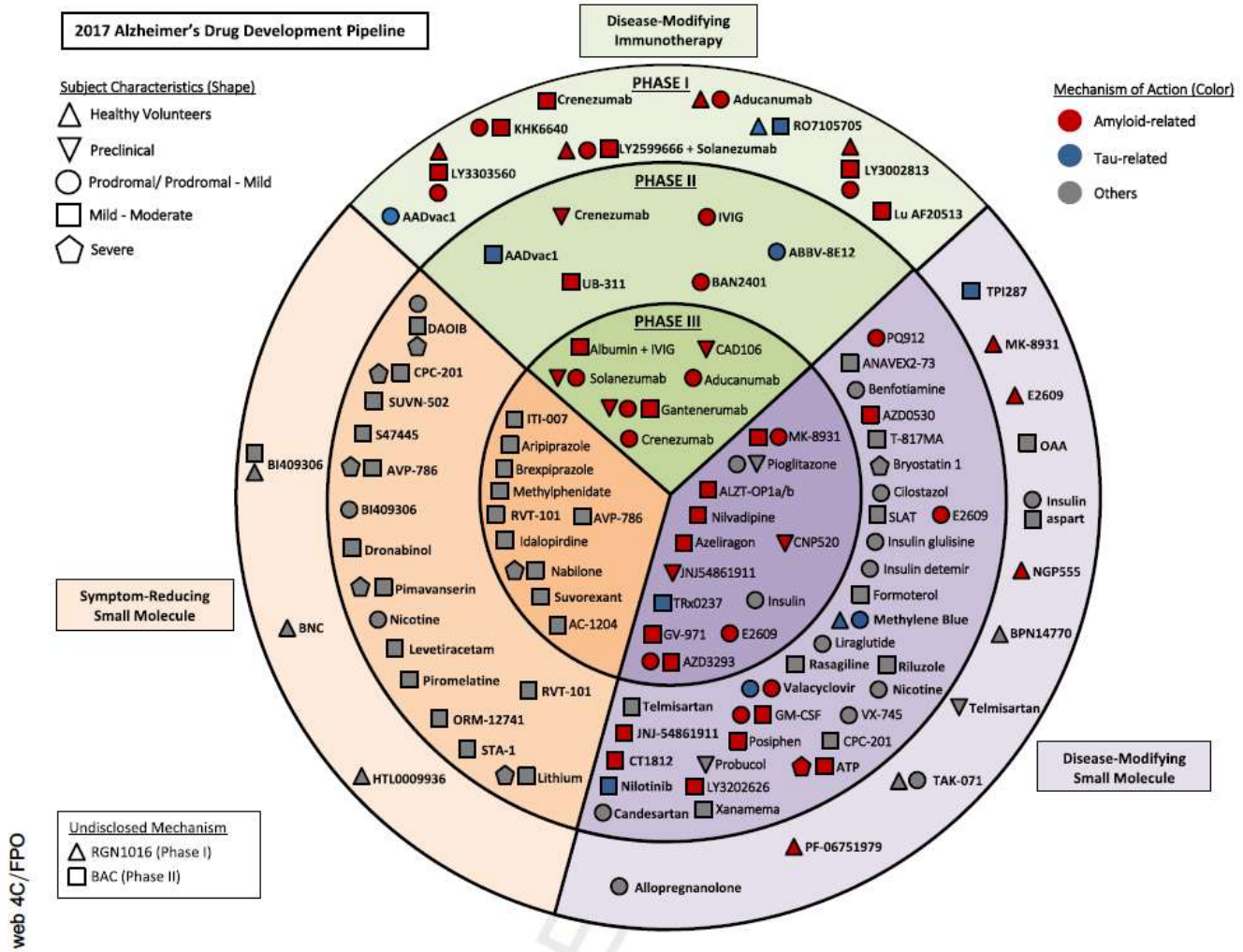
well plates with fluorescent readouts, facilitating efficient throughput. Prion RT-QuIC assays on cerebrospinal fluid (CSF) samples are being widely used for *antemortem* diagnosis of human prion diseases. Recently, we have also described a tau RT-QuIC prototype that has been optimized for Pick disease (with predominant 3R tau pathology) that detects 3R tau seeds in *postmortem* CSF, and brain tissue dilutions as extreme as a billion-fold. αSyn RT-QuIC prototypes have also been developed, providing ~92% diagnostic sensitivity and 100% specificity for Parkinson's disease and dementia with Lewy bodies using *antemortem* CSF. Here we provide detailed protocols for our 3R tau and αSyn RT-QuIC assays and refer the reader to published up-to-date protocols for prion RT-QuIC assays (Orru et al. Methods Mol Biol 1658:185–203, 2017; Schmitz et al. Nat Protoc 11:2233–2242, 2016).

Il sig. Bianchi CSF è
Abeta 42+
ptau -
tau 3R +
… che fare?

IL CERVELLO CHE CAMBIA 8

Sabato 10 novembre 2018

Genova, Aula Magna Clinica Neurologica



J. Cummings et al. / Alzheimer's & Dementia: Translational Research & Clinical Interventions ■ (2017) 1-18

Strategic project promoted
by the Italian Medicines Agency (AIFA)

INTERCEPTOR PROJECT

ON EARLY DIAGNOSIS OF THE PRODROMAL STAGE OF ALZHEIMER DISEASE. THE PROGRESSION FROM MILD COGNITIVE IMPAIRMENT (MCI) TO DEMENTIA: THE ROLE OF BIOMARKERS IN EARLY INTERCEPTION OF PATIENTS CANDIDATE FOR PRESCRIPTION OF FUTURE DISEASE-MODIFYING DRUG

	M1-6	M7-12 (T0)	M13-18 (T1)	M19-24 (T2)	M25-30 (T3)	M31-36 (T4)	M37-42 (T5)	M42-48 (T6)	M49-54
Identification of the Hubs of the network, harmonization meetings from ECs, preparation of technological platform	■								
Subjects enrollment		■							
Neuropsychological testing		■							
Neurological and Medical exam		■							
Biomarkers collection (MMSE; FCSRT; CSF p-tau e CSF tau/ABeta ;(^{18}F)FDG-PET;APOE4;EEG, volumetric MRI)		■							
Neuropsychological tests and functional scales (follow-up)			■	■	■	■	■	■	
Neurological exam (follow-up)			■	■	■	■	■	■	
Results evaluation									■

Person centred coordinated care: a definition

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