Svět guidelines a kritérií, šedá zóna, klinická praxe

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ELSEVIER

Alzheimer's & Dementia 7 (2011) 263–269

Alzheimer's & Dementia

The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease

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ELSEVIER

Alzheimer's & Dementia 7 (2011) 280–292

Alzheimer's & Dementia

Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease

Reisa A. Sperling[a,*], Paul S. Aisen[b], Laurel A. Beckett[c], David A. Bennett[d], Suzanne Craft[e], Anne M. Fagan[f], Takeshi Iwatsubo[g], Clifford R. Jack, Jr.[h], Jeffrey Kaye[i], Thomas J. Montine[j], Denise C. Park[k], Eric M. Reiman[l], Christopher C. Rowe[m], Eric Siemers[n], Yaakov Stern[o], Kristine Yaffe[p], Maria C. Carrillo[q], Bill Thies[q], Marcelle Morrison-Bogorad[r], Molly V. Wagster[r], Creighton H. Phelps[r]

Možnosti léčby ACH

- Normální stárnutí – (žádné příznaky) ***nemá amyloid***
  rizikové faktory
- Preklinická ACH – (žádné příznaky)
- Prodromální ACH (mírná kognitivní porucha)
- Plně rozvinutá ACH (demence)

má amyloid

Ovlivnění klinické praxe

EFNS guidelines 2010

European Journal of Neurology 2010, **17:** 1236–1248 doi:10.1111/j.1468-1331.2010.03040.x

EFNS GUIDELINES/CME ARTICLE

EFNS guidelines for the diagnosis and management of Alzheimer's disease

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Keywords:
Alzheimer's disease, dementia, diagnosis, guideline, management, review, treatment

Received 21 January 2010
Accepted 11 March 2010

Background and objectives: In 2008 a task force was set up to develop a revision of the European Federation of the Neurological Societies (EFNS) guideline for the diagnosis and management of Alzheimer's disease (AD) and other disorders associated with dementia, published in early 2007. The aim of this revised international guideline was to present a peer-reviewed evidence-based statement for the guidance of practice for clinical neurologists, geriatricians, psychiatrists, and other specialist physicians responsible for the care of patients with AD. Mild cognitive impairment and non-Alzheimer dementias are not included in this guideline.

EFNS-ENS guidelines 2012

European Journal of Neurology 2012, **19:** 1159–1179 doi:10.1111/j.1468-1331.2012.03784.x

EFNS-ENS GUIDELINES/CME ARTICLE

EFNS-ENS Guidelines on the diagnosis and management of disorders associated with dementia

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Stále přísnější metodika

- Studie class I-IV
- Síla doporučení A,B,C

- Grade system

Další faktory

Alzheimer's Disease Subtype of Major or Minor Neurocognitive Disorders

Mild

The Neurocognitive Disorders Work Group has posted the draft criteria for the Alzheimer Disease Subtype as an illustration of how they propose to organize other subtypes. They are inviting comment on the general approach. Work on other subtypes is currently in progress, and the Work Group is in consultation with various expert groups that work in those areas (e.g. vascular cognitive impairment and dementia, frontotemporal lobar degeneration, dementia with Lewy bodies, Huntington's disease, Parkinson's disease, traumatic brain injury, etc.).

Alzheimer's Disease Subtype of Major and Minor Neurocognitive Disorders

A. **Major:** Meets criteria for Major Neurocognitive Disorder, with memory being one of the impaired domains.

Minor: Meets criteria for Minor Neurocognitive Disorder with memory impairment AND there is clear supporting evidence for the Alzheimer etiology (e.g., a positive test for a known mutation in an Alzheimer's disease associated gene), or with evolving research, documentation based on biomarkers or imaging.

B. Early and prominent impairment in the Memory domain (rarely, other domains such as visuoconstructive perceptual domain may be prominently affected, but Alzheimer's disease would not be diagnosed without clear supporting imaging, biomarker or genetic evidence).

Major: Deficits are observed in at least one other domain, often Executive Ability, and as the disease progresses, in additional domains.

Minor: Only Memory may be affected, but deficits in Executive Abilities are common.

C. The course is characterized by gradual onset and continuing cognitive decline

D. Evidence from history, examination, and investigations that deficits are not wholly or primarily attributable to other disorders. However, other such disorders may coexist.

Důkazy

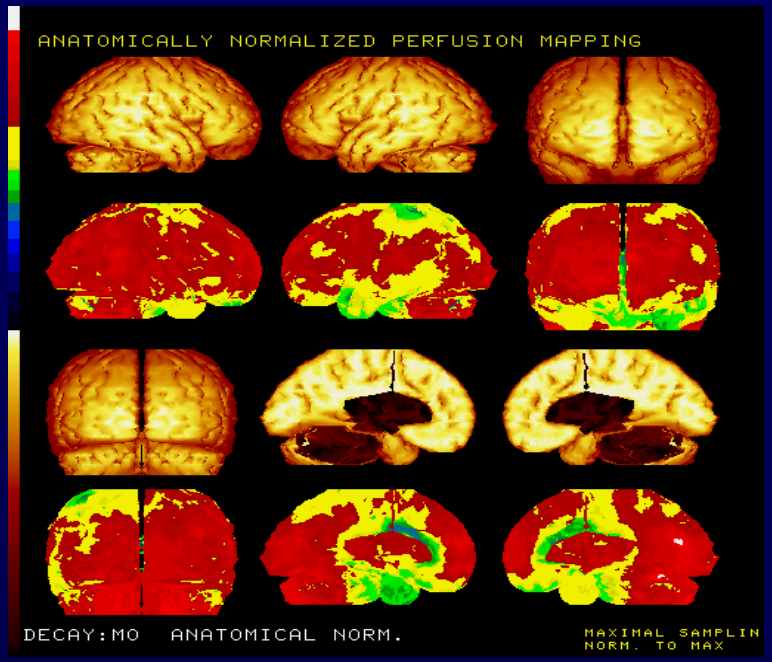
- Funguje
- Nefunguje
- Nedostatek důkazů že funguje

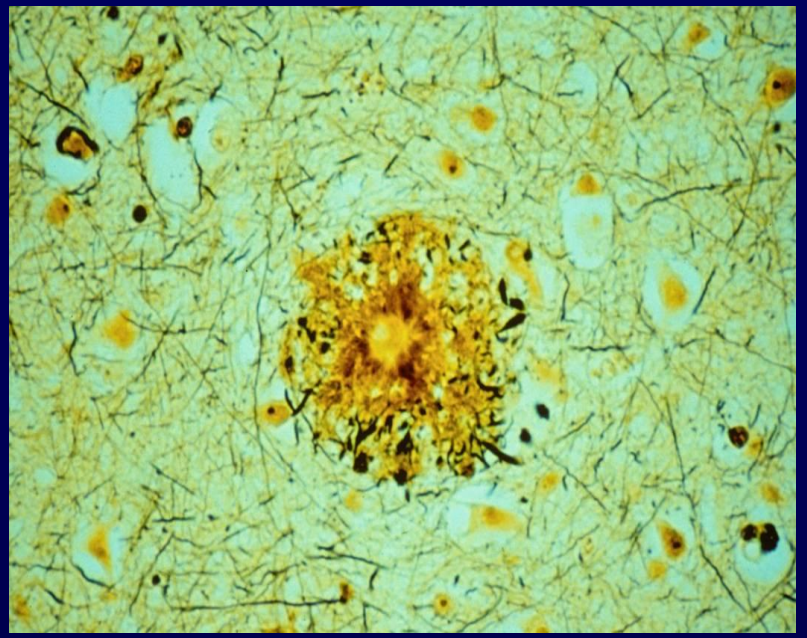
Ovlivnění klinické praxe

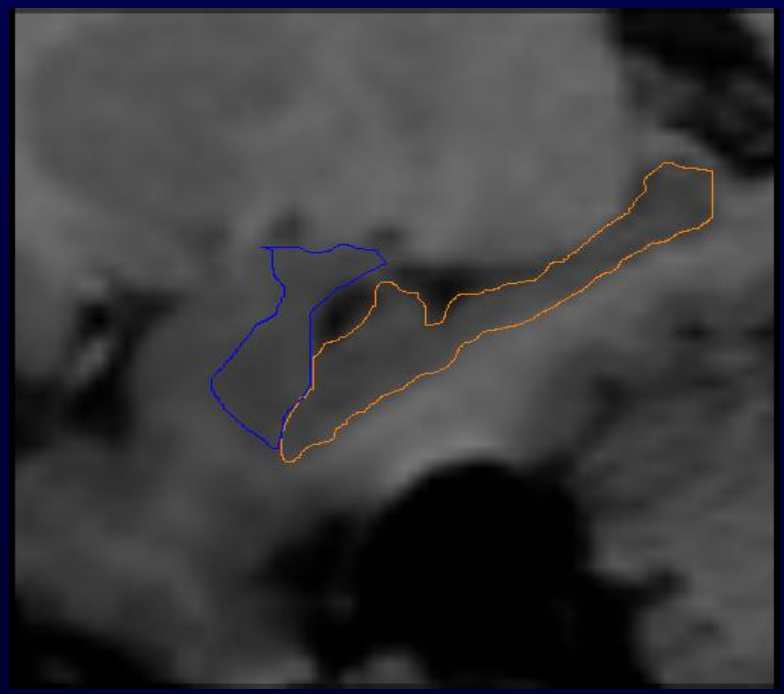
- Diagnostika
- Terapie

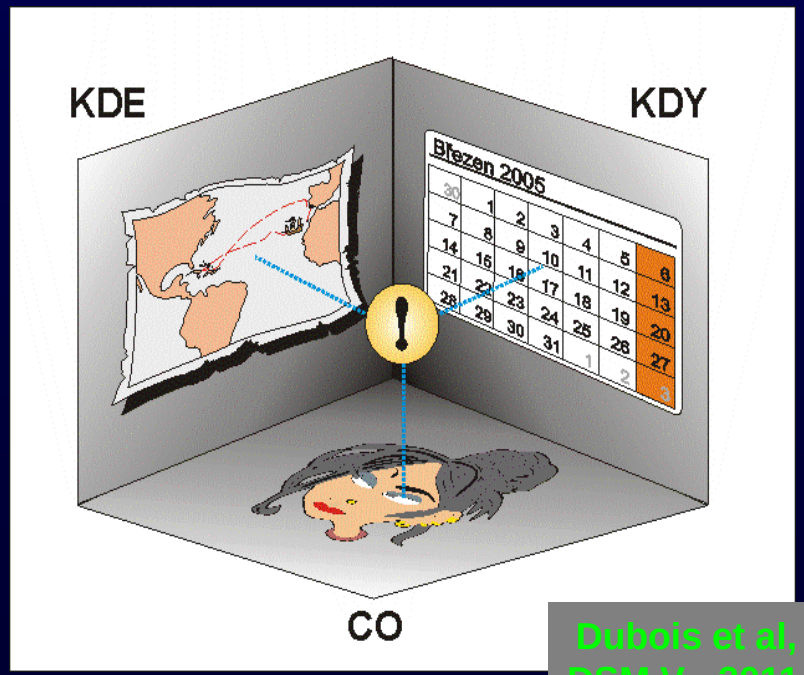
- Důkazy

- Opravdoví pacienti









Dubois et al, 2007
DSM V - 2011

Metabolismus

ACH postupuje velmi pozvolně

Preklinická ACH

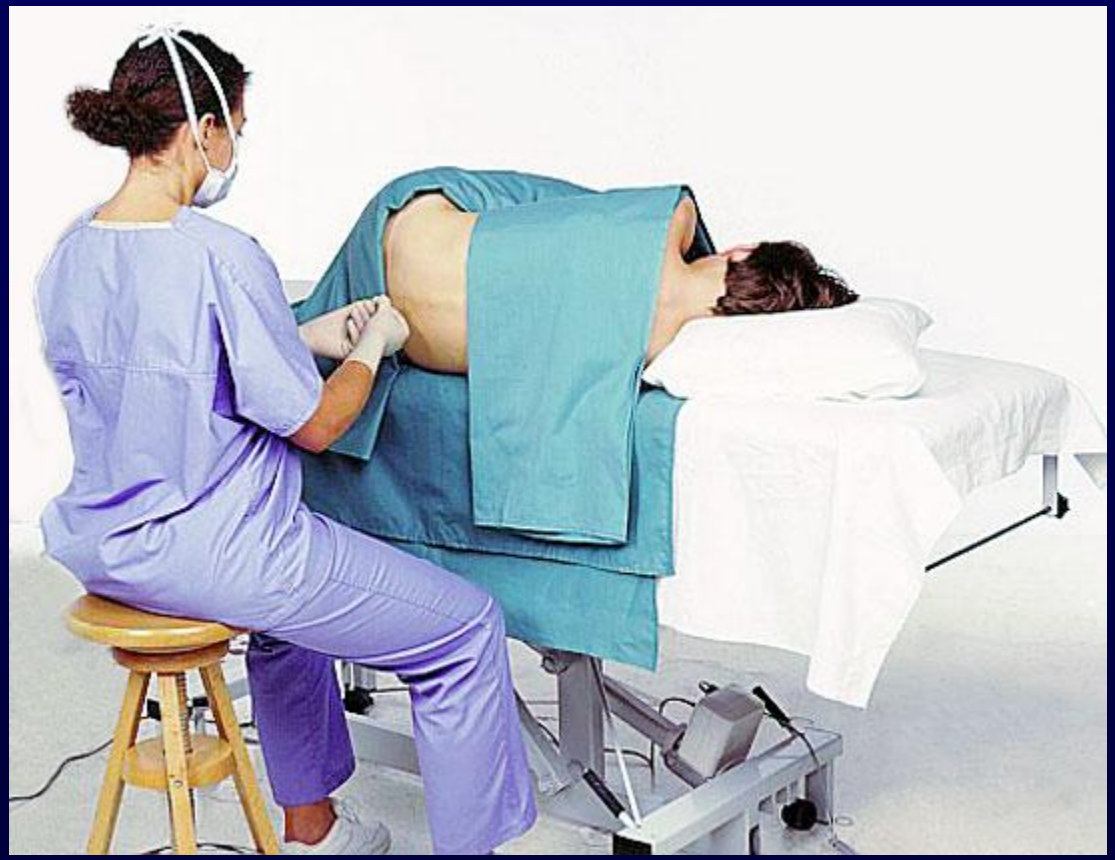
- Syndrom demence u pacientů s ACH předchází depozice amyloidu v mozku (o dekádu dříve)

- 20%-30% kognitivně normálních seniorů má ACH patologické změny v mozku

Prodromální ACH

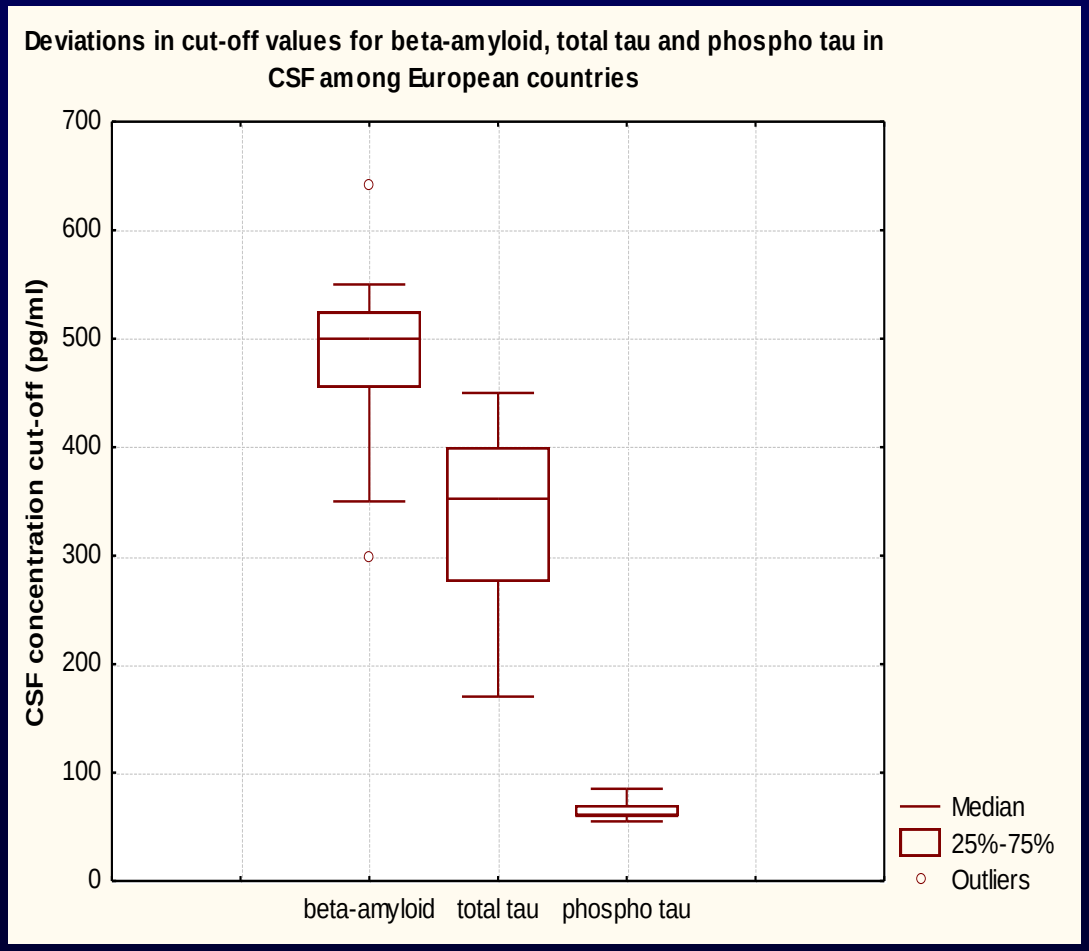
- 50%-70% pacientů s MCI má ACH patologii v mozku (konverze 15% za rok)

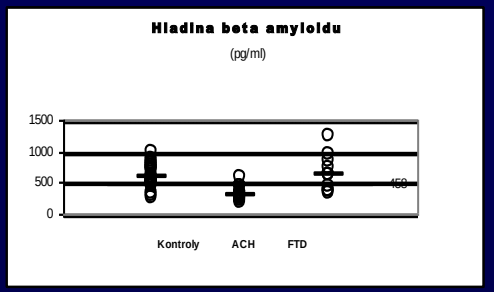
Hansson O, et al. *Lancet Neurol.* 2006;5:228-234; Okello A, et al. *Neurology.* 2009;73:754-760 ; Jack CR Jr, et al. *Lancet Neurol.* 2010;9:119-128

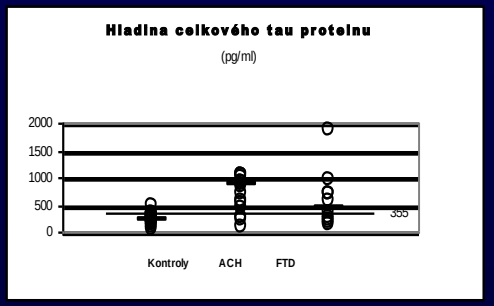


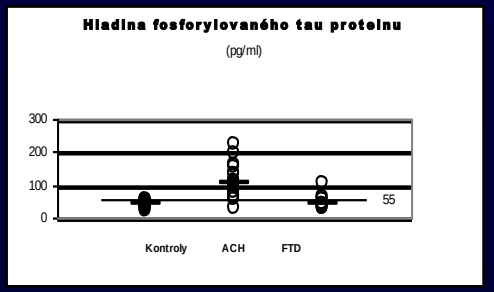
- Aβ42 ↓
- tau ↑
- fosfotau ↑

Likvor – praktické problémy!



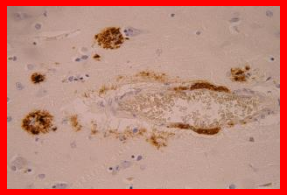
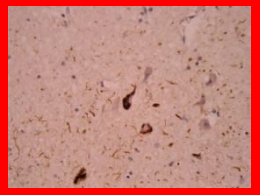
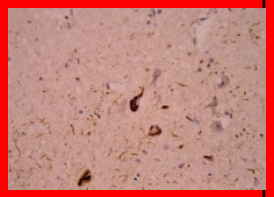






Hort et al., 2007, Hort et al., 2010

Zdraví nemocní

(pg/l)	β-Amyloid	total τ
ACH definitivní	↓ 	↑ 
Subjektivní paměť	→ 650	→ 300
Subjektivní paměť	↓ 	↑ 

	Normy Lab 1	Normy Lab 2	Normy Lab 3
ß-A	665	450 500 /70 let	458
Tau	358	149 275 /60 let	355
P-Tau	48	nemá	55

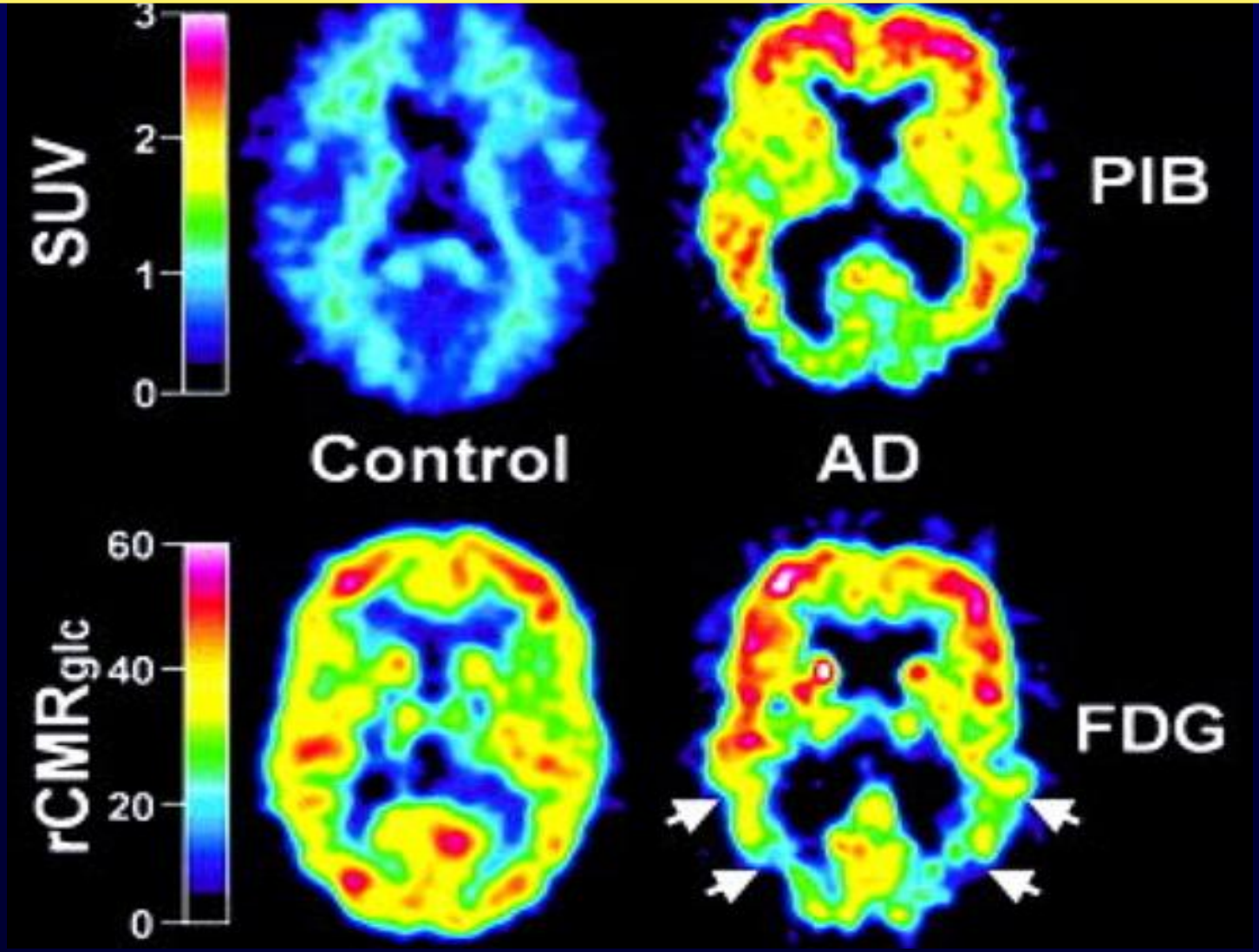
Výsledky Lab 1

		Normy Lab 1	Normy Lab 2	Normy Lab 3
ß-A	575			
Tau	419			
P-Tau	71.5			
		Počáteční ACH	**FTD**	**FTD**

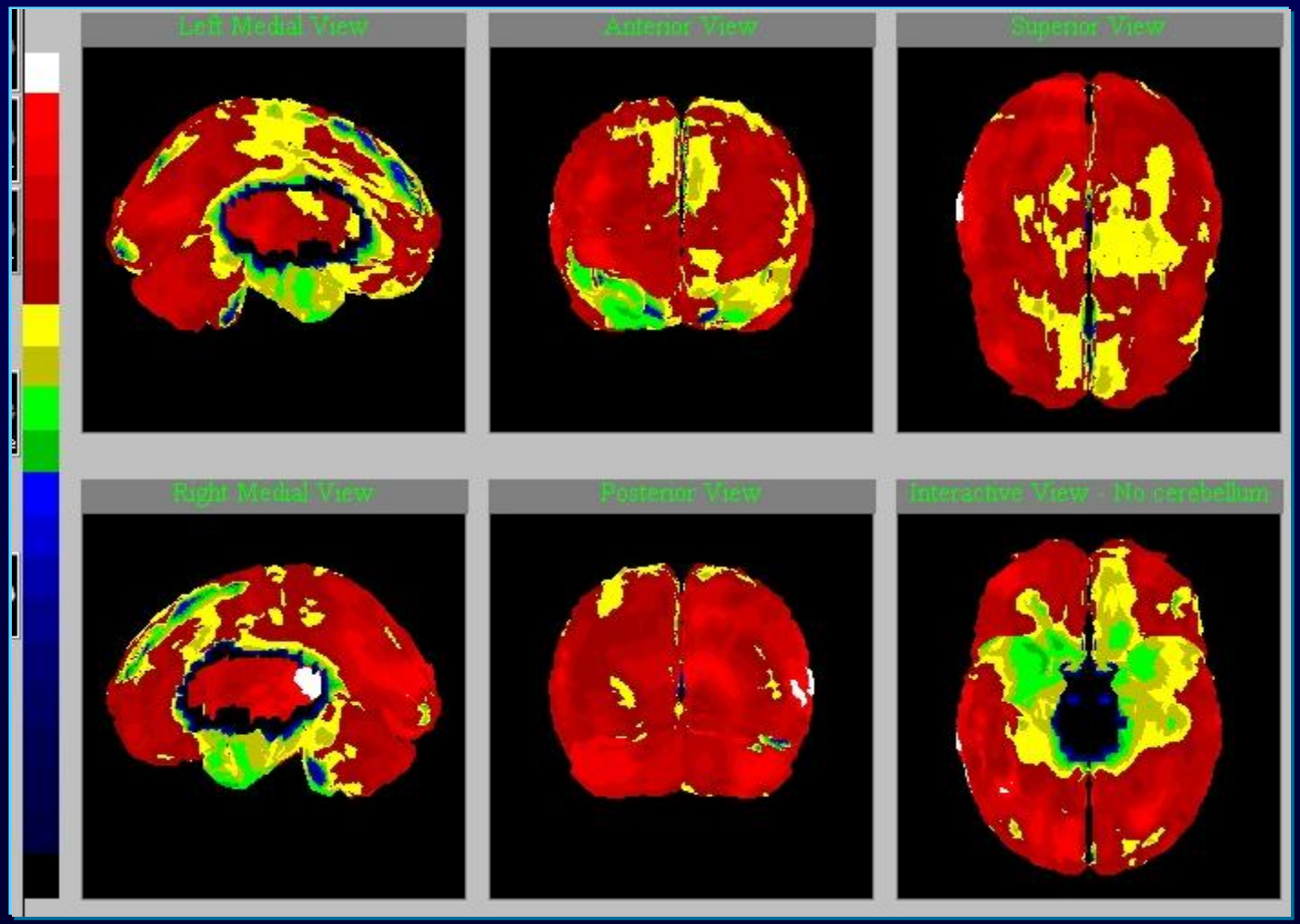
Výsledky Lab 2

ß-A	293			
Tau	931			
P-Tau	82			
		Pokročilá ACH	**Počáteční ACH**	**Pokročil ACH**

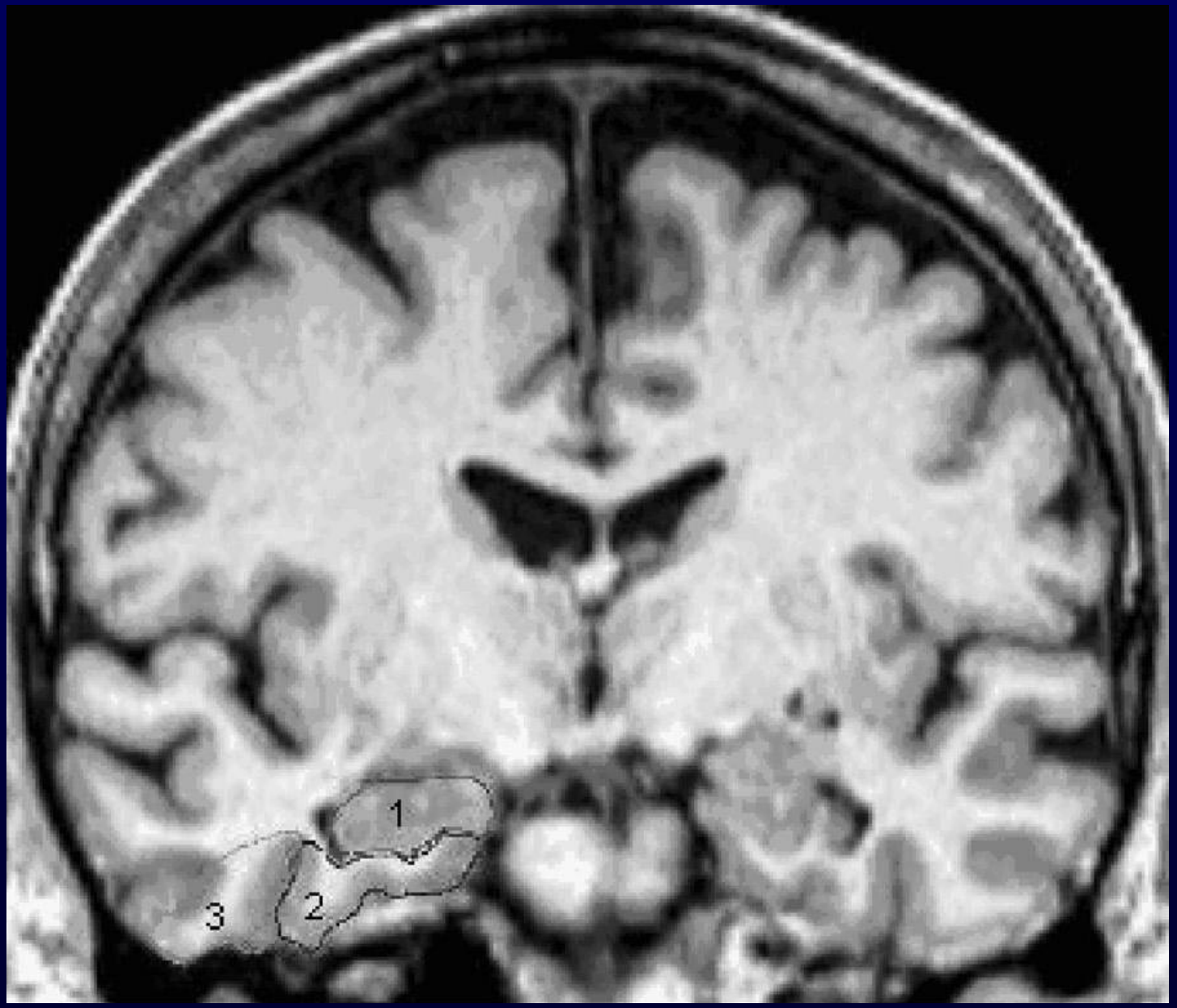
PET



SPECT - společný pacient



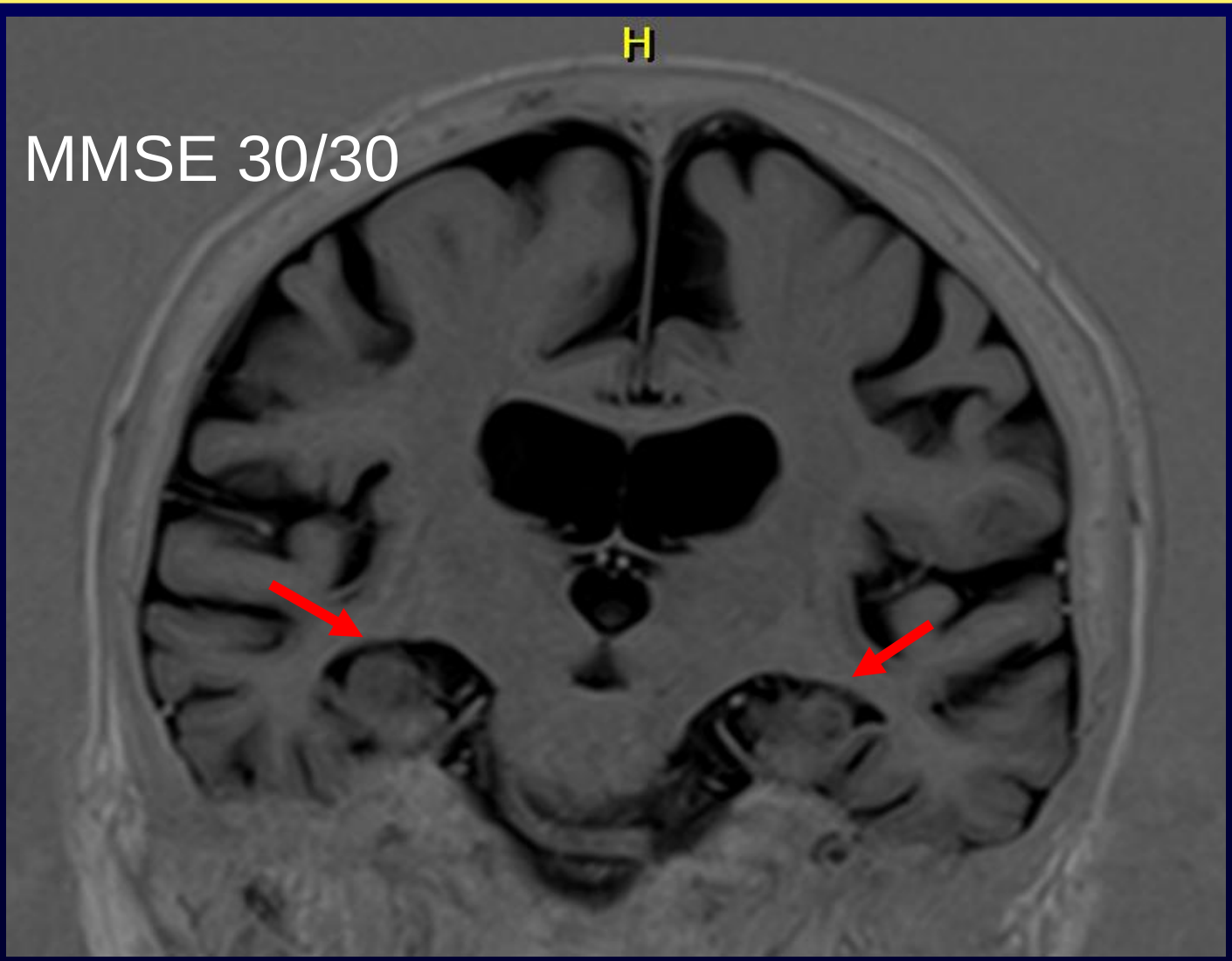
Strukturální


1
2
3

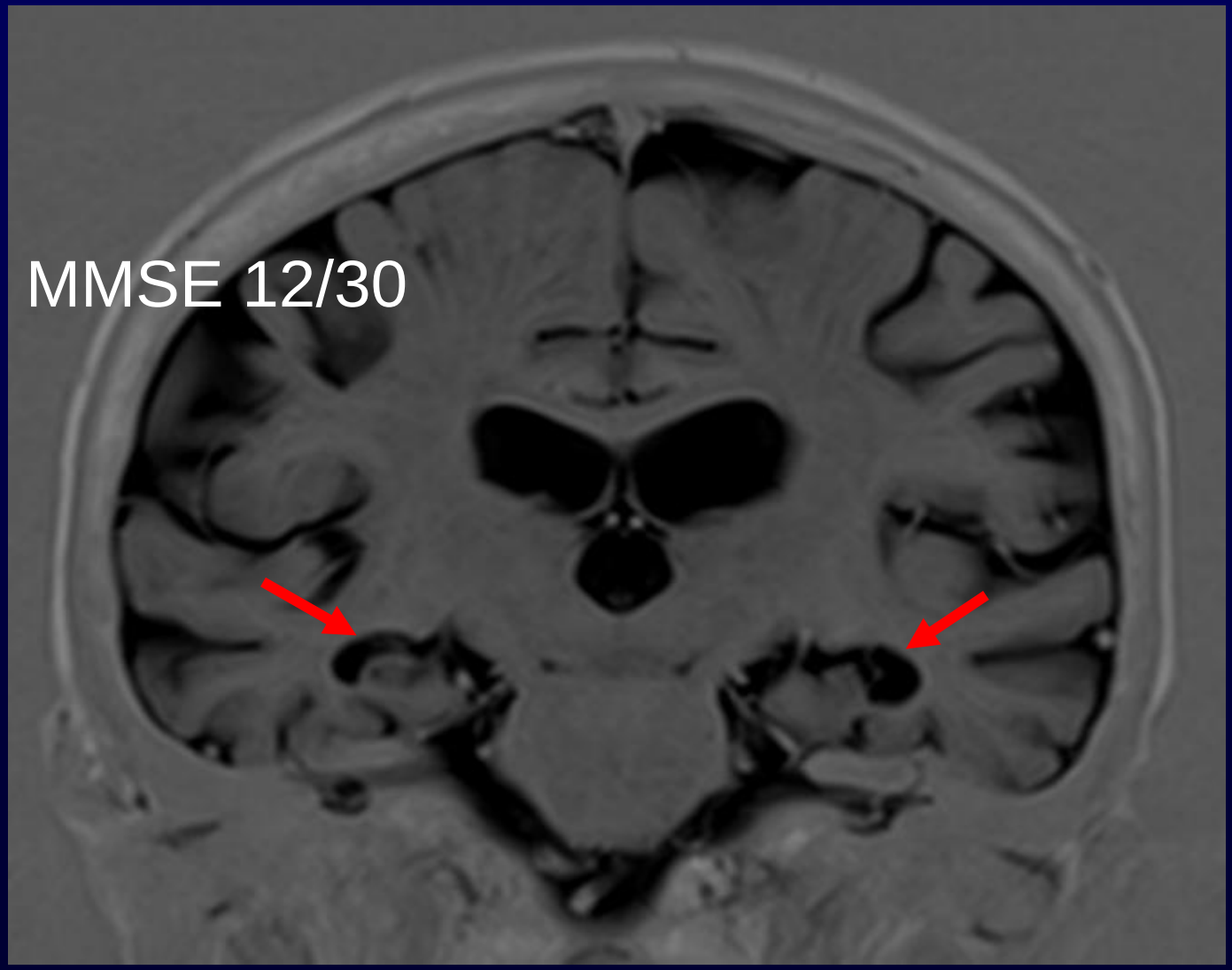
MRI volumometrie

- Windows
- Mac
- Vizuální

Normální nález nebo patologie?



Normální nález nebo patologie?



Interrater variabilita v hodnocení míry atrofie hipokampů pomocí Scheltensovy škály

XX

XX

Autoři deklarují, že v souvislosti s předmětem studie nemají žádné komerční zájmy.
The authors declare they have no potential conflicts of interest concerning drugs, products, or services used in the study.
Redakční rada potvrzuje, že rukopis práce splnil ICMJE kritéria pro publikace zasílané do biomedicínských časopisů.
The Editorial Board declares that the manuscript met the ICMJE "uniform requirements" for biomedical papers.

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Souhrn

Úvod: V poslední době se klade velký důraz na časnou diagnostiku Alzheimerovy choroby. Do nových diagnostických kritérií byly zařazeny nové diagnostické metody, které pomocí stanovení strukturálních a metabolických změn mozku tuto časnou diagnostiku umožňují. Patří mezi ně i vyšetření objemu hipokampů. Scheltens et al zavedli vizuální škálu na hodnocení atrofie hipokampů. Cílem studie bylo zjistit interrater variabilitu a ověřit praktické použití této škály. *Metoda:* Osmi hodnotiteli s různým stupněm erudice v užívání Scheltensovy vizuální škály bylo vyhodnoceno 70 magnetických rezonancí pacientů s kognitivní poruchou a zdravých kontrol vyšetřených v Kognitivním centru při Neurologické klinice FN v Motole. Po krátkém zaškolení vyšetřující vyhodnotili tréninkový vzorek 20 pacientů s následným rozborem nálezů u každého z hodnotitelů a poté provedli vlastní zaslepené hodnocení atrofie hipokampů Scheltensovou škálou za použití snímků MR v koronární rovině. Variabilita byla posuzována pro všech osm hodnotitelů dohromady a pak ještě po jejich rozdělení na čtyři zkušené a čtyři nezkušené (před účastí v této studii neměli se škálou žádnou zkušenost). *Výsledky:* Nalezli jsme velmi vysoký koeficient shody pro všechny hodnotitele dohromady pro pravý (K = 0,87) i levý (K = 0,88) hipokampus. Velmi vysoký

Neuroimaging correlates of pathologically defined subtypes of Alzheimer's disease: a case-control study

Jennifer L Whitwell, Dennis W Dickson, Melissa E Murray, Stephen D Weigand, Nirubol Tosakulwong, Matthew L Senjem, David S Knopman, Bradley F Boeve, Joseph E Parisi, Ronald C Petersen, Clifford R Jack Jr, Keith A Josephs

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Summary

Background Three subtypes of Alzheimer's disease (AD) have been pathologically defined on the basis of the distribution of neurofibrillary tangles: typical AD, hippocampal-sparing AD, and limbic-predominant AD. Compared with typical AD, hippocampal-sparing AD has more neurofibrillary tangles in the cortex and fewer in the hippocampus, whereas the opposite pattern is seen in limbic-predominant AD. We aimed to determine whether MRI patterns of atrophy differ between these subtypes and whether structural neuroimaging could be a useful predictor of pathological subtype at autopsy.

Methods We identified patients who had been followed up in the Mayo Clinic Alzheimer's Disease Research Center (Rochester, MN, USA) or in the Alzheimer's Disease Patient Registry (Rochester, MN, USA) between 1992 and 2005. To be eligible for inclusion, participants had to have had dementia, AD pathology at autopsy (Braak stage ≥IV and intermediate to high probability of AD), and an ante-mortem MRI. Cases were assigned to one of three pathological subtypes—hippocampal-sparing, limbic-predominant, and typical AD—on the basis of neurofibrillary tangle counts in hippocampus and cortex and ratio of hippocampal to cortical burden, without reference to neuronal loss. Voxel-based morphometry and atlas-based parcellation were used to compare patterns of grey matter loss between groups and with age-matched control individuals. Neuroimaging was obtained at the time of first presentation. To summarise pair-wise group differences, we report the area under the receiver operator characteristic curve (AUROC).

Findings Of 177 eligible patients, 125 (71%) were classified as having typical AD, 33 (19%) as having limbic-predominant AD, and 19 (11%) as having hippocampal-sparing AD. Most patients with typical (98 [78%]) and limbic-predominant AD (31 [94%]) initially presented with an amnestic syndrome, but fewer patients with hippocampal-sparing AD (eight [42%]) did. The most severe medial temporal atrophy was recorded in patients with limbic-predominant AD, followed by those with typical disease, and then those with hippocampal-sparing AD. Conversely, the most severe cortical atrophy was noted in patients with hippocampal-sparing AD, followed by those with typical disease, and then limbic-predominant AD. The ratio of hippocampal to cortical volumes allowed the best discrimination between subtypes ($p<0·0001$; three-way AUROC 0·52 [95% CI 0·47–0·52]; ratio of AUROC to chance classification 3·1 [2·8–3·1]). Patients with typical AD and non-amnesic initial presentation had a significantly higher ratio of hippocampal to cortical volumes (median 0·045 [IQR 0·035–0·056]) than did those with an amnesic presentation (0·041 [0·031–0·057]; $p=0·001$).

Interpretation Patterns of atrophy on MRI differ across the pathological subtypes of AD. MRI regional volumetric analysis can reliably track the distribution of neurofibrillary tangle pathology and can predict pathological subtype of AD at autopsy.

doi:10.1093/brain/aws076 Brain 2012: 135; 1554–1565

BRAIN
A JOURNAL OF NEUROLOGY

Clinically concordant variations of Alzheimer pathology in aphasic versus amnestic dementia

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Neuropathologically defined subtypes of Alzheimer's disease with distinct clinical characteristics: a retrospective study

Melissa E Murray, Neill R Graff-Radford, Owen A Ross, Ronald C Petersen, Ranjan Duara, Dennis W Dickson

Summary

Background Neurofibrillary pathology has a stereotypical progression in Alzheimer's disease (AD) that is encapsulated in the Braak staging scheme; however, some AD cases are atypical and do not fit into this scheme. We aimed to compare clinical and neuropathological features between typical and atypical AD cases.

Methods AD cases with a Braak neurofibrillary tangle stage of more than IV were identified from a brain bank database. By use of thioflavin-S fluorescence microscopy, we assessed the density and the distribution of neurofibrillary tangles in three cortical regions and two hippocampal sectors. These data were used to construct an algorithm to classify AD cases into typical, hippocampal sparing, or limbic predominant. Classified cases were then compared for clinical, demographic, pathological, and genetic characteristics. An independent cohort of AD cases was assessed to validate findings from the initial cohort.

Findings 889 cases of AD, 398 men and 491 women with age at death of 37–103 years, were classified with the algorithm as hippocampal sparing (97 cases [11%]), typical (665 [75%]), or limbic predominant (127 [14%]). By comparison with typical AD, neurofibrillary tangle counts per 0·125 mm² in hippocampal sparing cases were higher in cortical areas (median 13, IQR 11–16) and lower in the hippocampus (7·5, 5·2–9·5), whereas counts in limbic-predominant cases were lower in cortical areas (4·3, 3·0–5·7) and higher in the hippocampus (27, 22–35). Hippocampal sparing cases had less hippocampal atrophy than did typical and limbic-predominant cases. Patients with hippocampal sparing AD were younger at death (mean 72 years [SD 10]) and a higher proportion of them were men (61 [63%]), whereas those with limbic-predominant AD were older (mean 86 years [SD 6]) and a higher proportion of them were women (87 [69%]). Microtubule-associated protein tau (*MAPT*) H1H1 genotype was more common in limbic-predominant AD (54 [70%]) than in hippocampal sparing AD (24 [46%]; p=0·011), but did not differ significantly between limbic-predominant and typical AD (204 [59%]; p=0·11). Apolipoprotein E (*APOE*) ε4 allele status differed between AD subtypes only when data were stratified by age at onset. Clinical presentation, age at onset, disease duration, and rate of cognitive decline differed between the AD subtypes. These findings were confirmed in a validation cohort of 113 patients with AD.

Interpretation These data support the hypothesis that AD has distinct clinicopathological subtypes. Hippocampal sparing and limbic-predominant AD subtypes might account for about 25% of cases, and hence should be considered when designing clinical, genetic, biomarker, and treatment studies in patients with AD.

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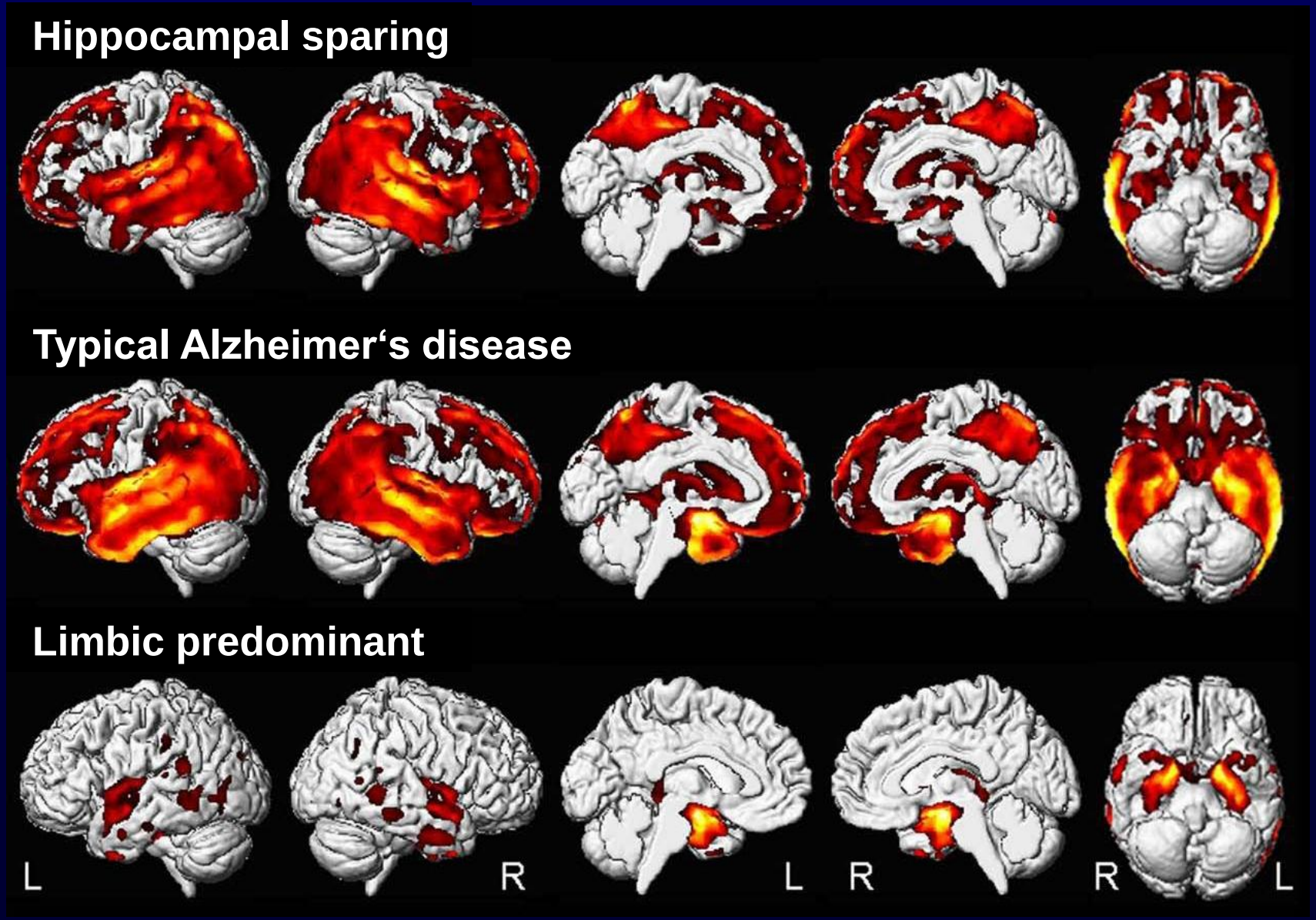
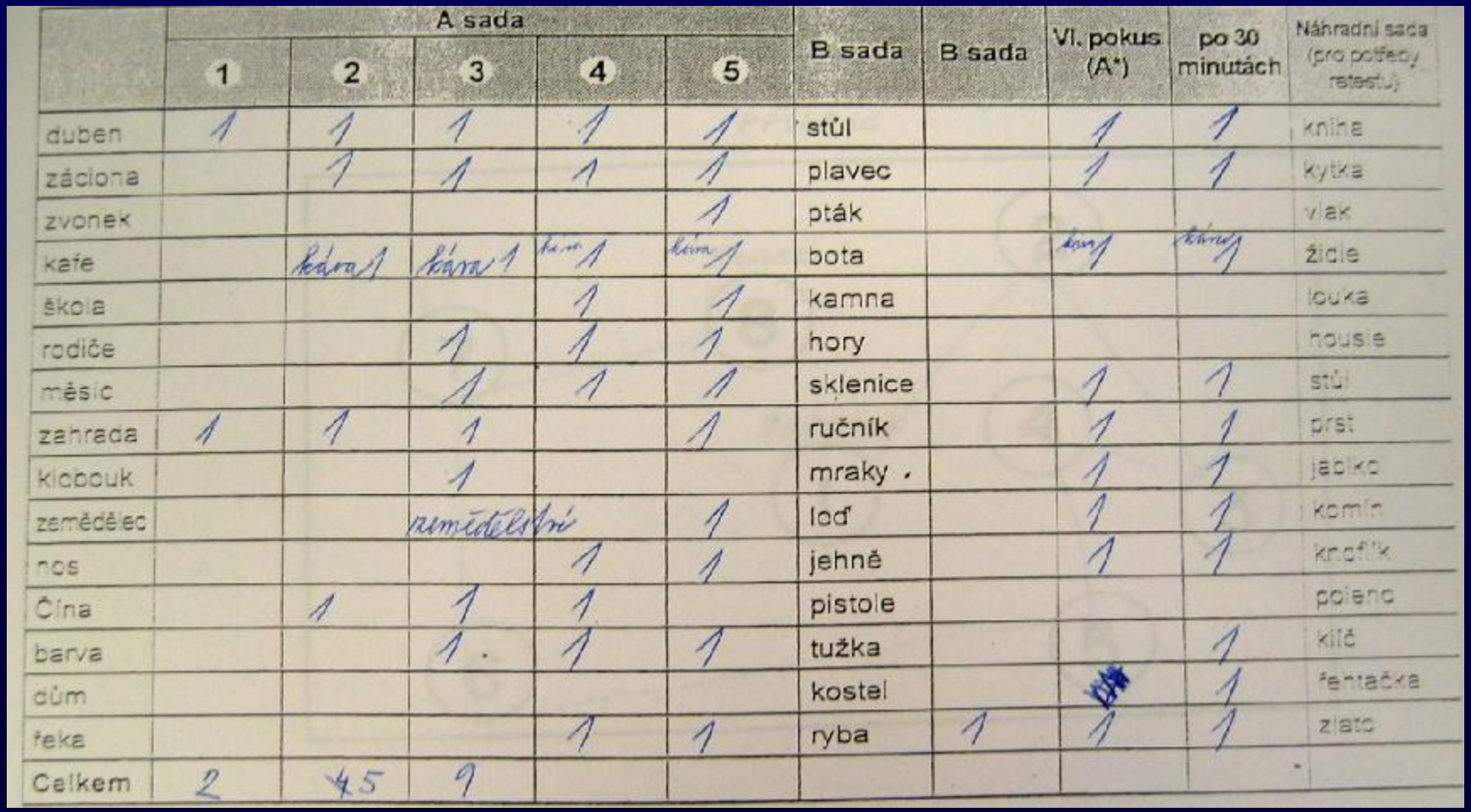
Tři podtypy Alzheimerovy choroby



Je rozdíl časná a pozdní?

- Časná – atypické formy, E4 negativní
- Metabolické biomarkery - přínosnější a specifičtější
- Strukturální biomarkery– méně přínosné
- Diferenciální diagnostika ostatní demence
- Osobní zkušenost – mnoho FTLD, 7 JCD

Na čem záleží?



	A sada					B sada	B sada	VI. pokus (A*)	po 30 minutách	Náhradní sada (pro potřeby retestu)
	1	2	3	4	5					
duben	1	1	1	1	1	stůl		1	1	kniha
záclona		1	1	1	1	plavec		1	1	kytka
zvonek					1	pták				vlak
kafe		kávra 1	kávra 1	kávra 1	kávra 1	bota		kávra 1	kávra 1	židle
škola				1	1	kamna				louka
rodiče			1	1	1	hory				housle
měsíc			1	1	1	sklenice		1	1	stůl
zahrada	1	1	1		1	ručník		1	1	prst
klobouk			1			mraky		1	1	jablko
zemědělec			zemědělství		1	loď		1	1	komín
nos				1	1	jehně		1	1	knoflík
Čína		1	1	1		pistole				poleno
barva			1	1	1	tužka			1	klíč
dům						kostel			1	pentačka
řeka				1	1	ryba	1	1	1	zlato
Celkem	2	45	9							

Na výsledku v testu?

Log. paměť:	**Jednotky:**	14	19	**Jedn_20:**	9	14
	Témata:	5	18-21 nadprůměr, ≥+1sd	**Témata_20:**	5	9-16 průměr
AVLT:	**I:**	7	**VI:**	4	podprůměr, <-1,5sd	
	II:	7	**po 30min:**	2	podprůměr, <-1,5sd	
	III:	9	**I – V:**	41	průměr, <-0,5sd	
	IV:	9	**rekognice:**	9	**FP:**	1
	V:	9	**konfabulace:**	8	**opaková.:**	6
	B:	5	**křivka učení:**	plochá		
16 slov:	**Pojmenov.:**	15			**Spont. vyb.:**	7
	Vybaveno:	16			**S nápovědou vyb.:**	8
	Konfabulace:	1		nosorožec		
	Celkem	15	14-16 průměr		nápověda normalizuje výkon	

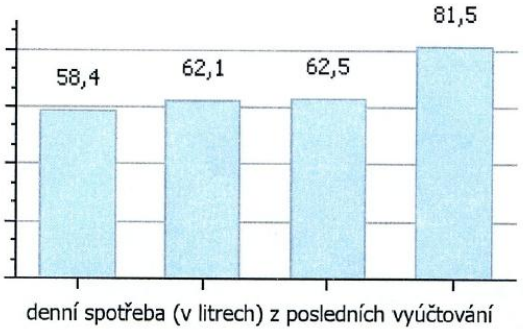
Nebo na spotřebě vody?

INFORMACE O SPOTŘEBĚ

Informace o Vaší běžné spotřebě vody

Vážený zákazníku,

uvedený graf zobrazuje průměrnou spotřebu. Porovnáním posledního odečtu, který je uveden na této faktuře, nebyla zjištěna výraznější odchylka oproti minulému období. Pokud si chcete ověřit, zda na Vašich vnitřních rozvodech nedochází k úniku vody, doporučujeme postupovat následujícím způsobem (pozor, nezavírejte hlavní uzávěr vody před vodoměrem).



denní spotřeba (v litrech) z posledních vyúčtování

Jak ověřit Vaše vnitřní rozvody

1. Večer si zapište hodnoty na vodoměru
2. Ráno si opět zapište hodnoty na vodoměru
3. Pokud jste si jisti, že nebyl v provozu žádný spotřebič vody, obě hodnoty porovnejte
4. V případě, že se hodnoty spotřeby přes noc změnily, doporučujeme Vám, obrátit se na instalatéra a nechat prověřit vnitřní rozvod.

Vaše dotazy rádi zodpovíme na telefonních číslech uvedených na této faktuře.

TELEFONNÍ CENTRUM: 840 111 112

Nepřetržitý provoz

Pro osobní styk:

ZÁKAZNICKÉ CENTRUM

Dykova 3
Praha 10 - Vinohrady

European Journal of Neurology 2011, **18:** 279–285 doi:10.1111/j.1468-1331.2010.03134.x

The use of neuropsychological tests across Europe: the need for a consensus in the use of assessment tools for dementia

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Keywords:
dementia diagnosis, neuropsychological tests

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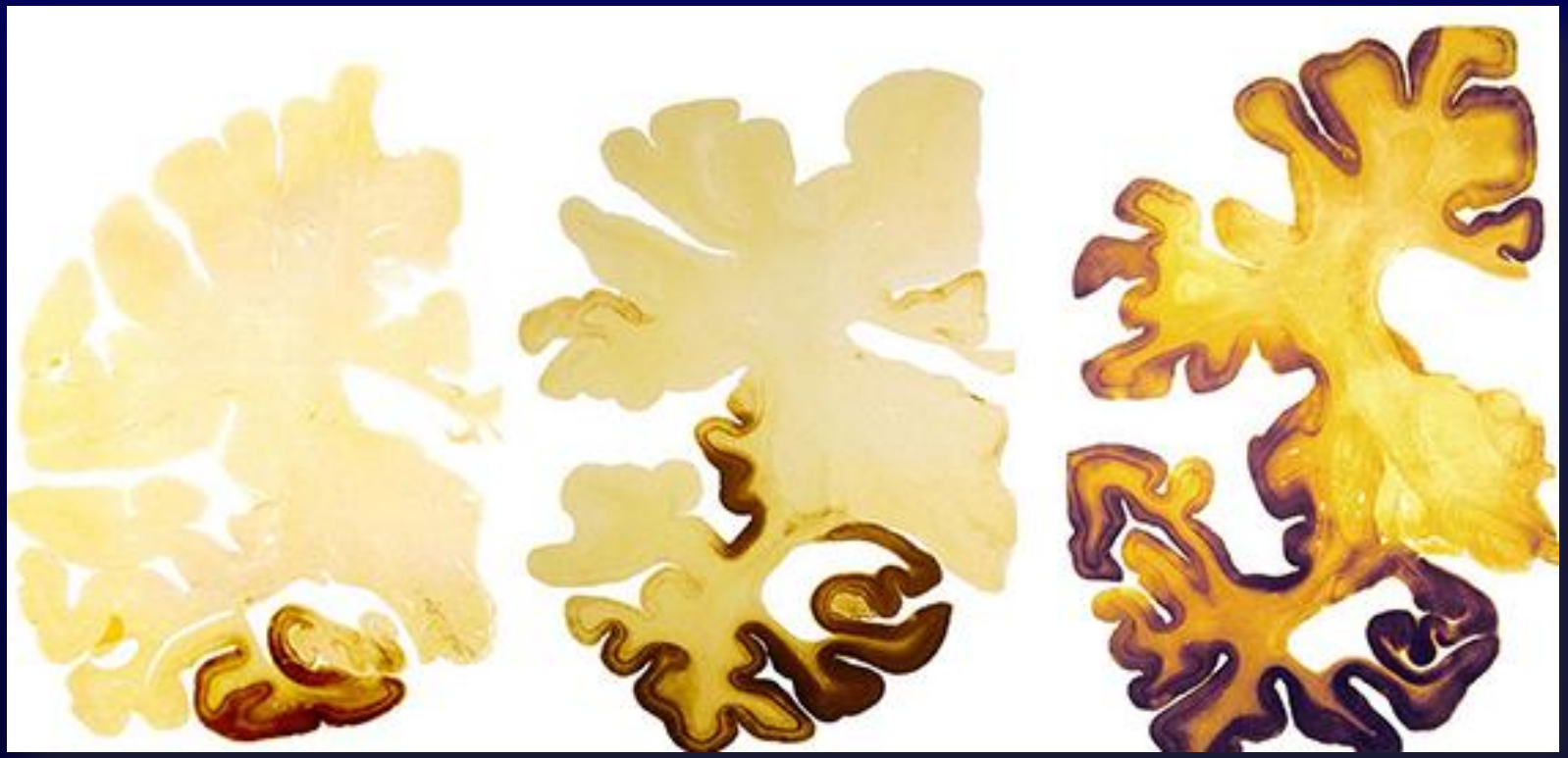
Background and purpose: The centres dedicated to dementia throughout Europe use different neuropsychological tests in clinical practice. The European Federation of Neurological Societies task force on neuropsychological tests produced this survey on neuropsychological tests currently being used in different European countries to gather knowledge on the practice of dementia centres and to promote the harmonization of such instruments and future multicentre collaborations.

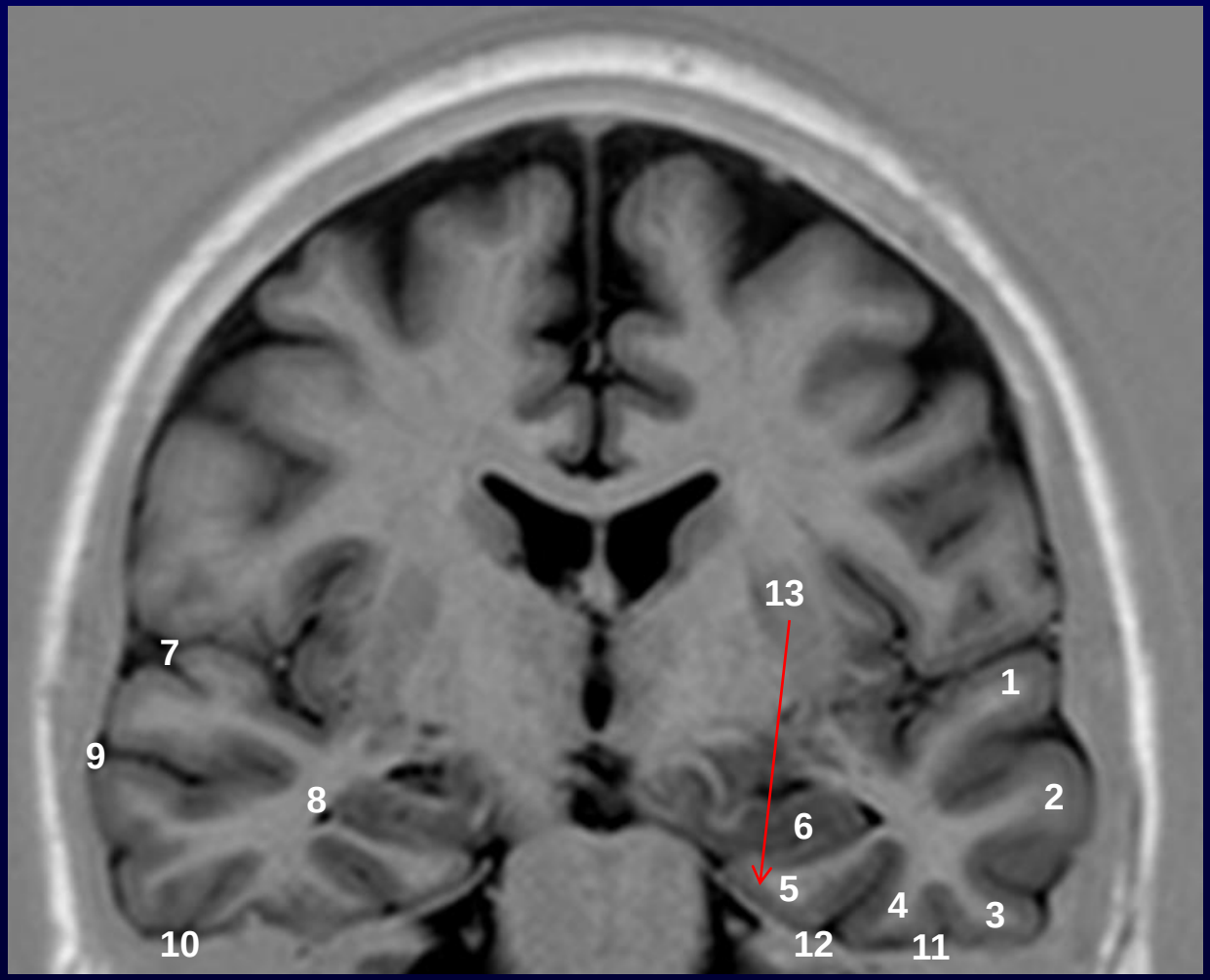
Methods: National representatives of 34 countries received a questionnaire and 25 (73.5%) sent it back.

Results: A few instruments, Mini-Mental State Examination (MMSE), Trail Making Test (TMT), Verbal Fluency and Clock Drawing Test, were available in all countries. Wechsler Adult Intelligence Scales and MMSE were reported to be valid, respectively, in 20 (80%) and 19 (76%) countries, whereas Verbal Fluency and Stroop Test are valid in 18 (72%) of them. Of the 25 countries, 17 have validation norms for Clock Drawing Test and TMT (68%), and Neuropsychiatric Inventory, Alzheimer's Disease Assessment Scale – Cognitive Subscale, Rey Complex Figure Test, Digit Symbol and Beck Depression Inventory were standardized in 16 countries (64%). The remaining tests were validated, at most, in about half of them. Not all countries certificate neuropsychology.

Kognitivní pokles koreluje s tau



Braak H, Braak E. Neuropathological stageing of Alzheimer-related changes. *Acta Neuropathologica, 1991*



1 – gyrus temporalis superior, 2 – medius, 3- inferior, 4 – fusiformis (occipitotemporalis lateralis), 5 – parahipokampalis (occipitotemporalis medialis), 6 – hipokampus, 7 - fisura Sylvii (cerebri lateralis), 8 – temporální roh postranní komory, 9 – sulcus temporalis superior, 10 – inferior, 11 – sulcus occipitotemporalis lateralis, 12 – sulcus collateralis, 13 – transentorhinální kortex


Tower Bridge
Unknown
Charles Bridge
Unknown


Happiness
Surprise
Sadness
Fear
Anger
Disgust


Unknown
Famous
Famous
Unknown
Unknown
Famous
3. Test of famous landmarks identification

Anamnesa

- Nemohu si vybavit jméno toho herce
- Několikrát jsem zapomněl přijít na domluvenou schůzku
- Často hledám své brýle
- Dvakrát jsem se ztratil.
- Když jdu na nákup, musím si vše napsat.
- Opakovaně zapomínám vypnout plyn.

Genetika

- E2
- E4/E2, E4/E3
- E4/E4
- E3/E3 – TOM 40?

Léčba

- Důkazy – inhibitory a memantine, hranice MMSE umělé
- Nedostatek důkazů pro kombinaci
- Důkazy, že nefungují nootropika
- Nedostatek důkazů že funguje EGb761, nitrendipin, nefarmakologické postupy

- Jak byste léčili svou babičku?

- Dožijeme se důkazů kauzální léčby?