Troubles cognitifs L-M & interfaces avec le domaine de la psychiatrie: **1 Vignette clinique**

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Disclosure: Last 5 years, personal, related to the topic

- **O Godefroy:**
  - scientific advisory boards (Novartis),
  - funding for meeting support, travel, speaking honoraria and speaker bureau (Astrazeneca, Biogen, Boehringer, BMSquibb, Covidien, Genzyme, GlaxoSmithKline, ISPEN, Lilly, Novartis, Pfizer, TEVA, EISAI)

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Tr. Cognitifs: interfaces avec psychiatrie

- Qq rappels: cognition et ses troubles
- **Critères diagnostiques** MA, (TCVa), DFT, **MCLewy**
- Interfaces avec Psychiatrie

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Cas clinique 1 Sept 2019

- 69a, droitier, Nscol 3
- ancien rugbyman avec 2 épisodes IAmn trauma
- TRANSIPEG, SEROPLEX 5 mg, OMEPRAZOLE 20
- **Motif réduction d'activité, désintérêt**
- **66a**:
  - apathie avec restriction d'activités
  - Tr sommeil: agitation avec cris
- **Ex** :
  - Somatique RAS sf **tres discrete asym rapidité MSG**
  - MMSE **29**/30; 4IADL: **2**/12
  -

Cas clinique 2

- **BNPsycho**
  - **Instrumentales**: Deno=N; copie Fig Rey= déficit
  - **MEpisodique**: Ve=déficit (stockage); Vis=N
  - **FExec**= ralentissemt + diminution flu litt (sem=N)

    Comportt (ISDC)= hypoactivité avec apathie
  - **Humeur-anxiété**: MADRS = 5/60; Goldberg=1/9

- **-> TNCog Léger dysexécutif cptt multidomaine**

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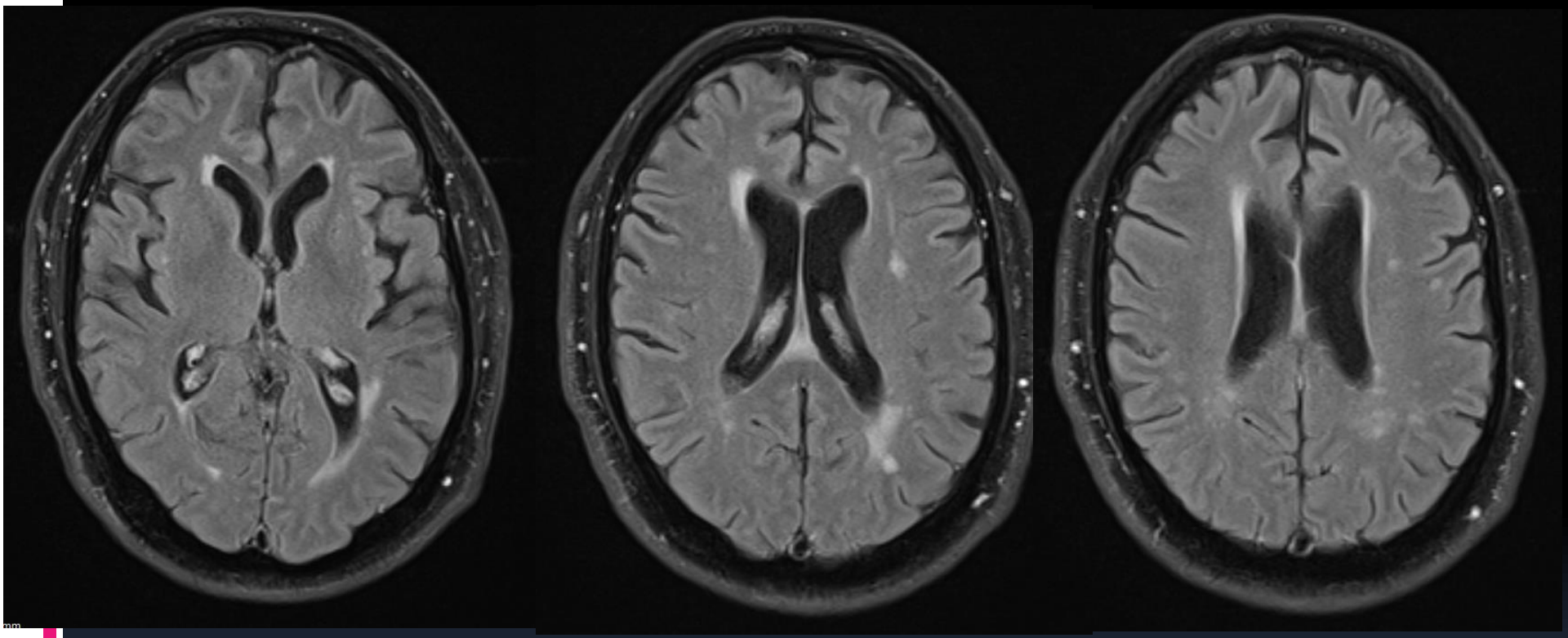
Cas clinique 1 IRM



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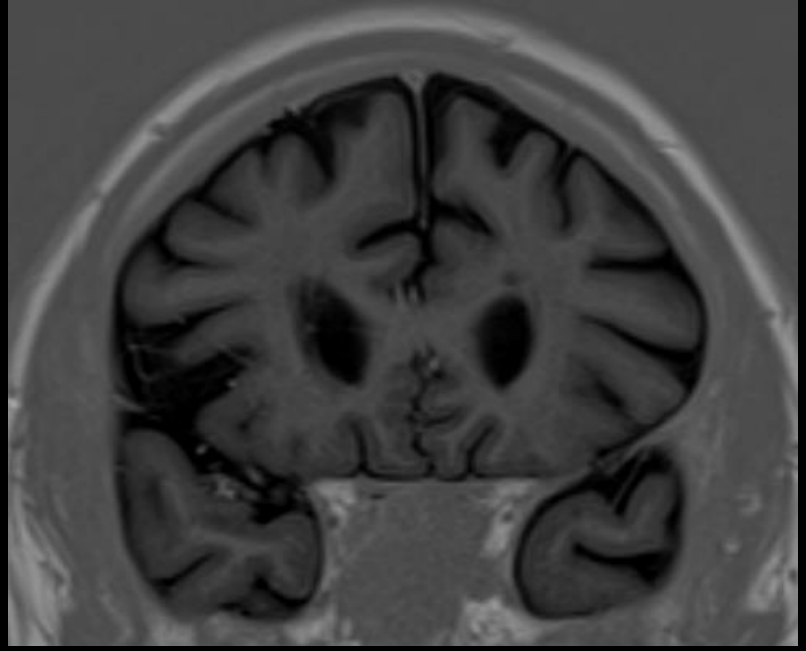

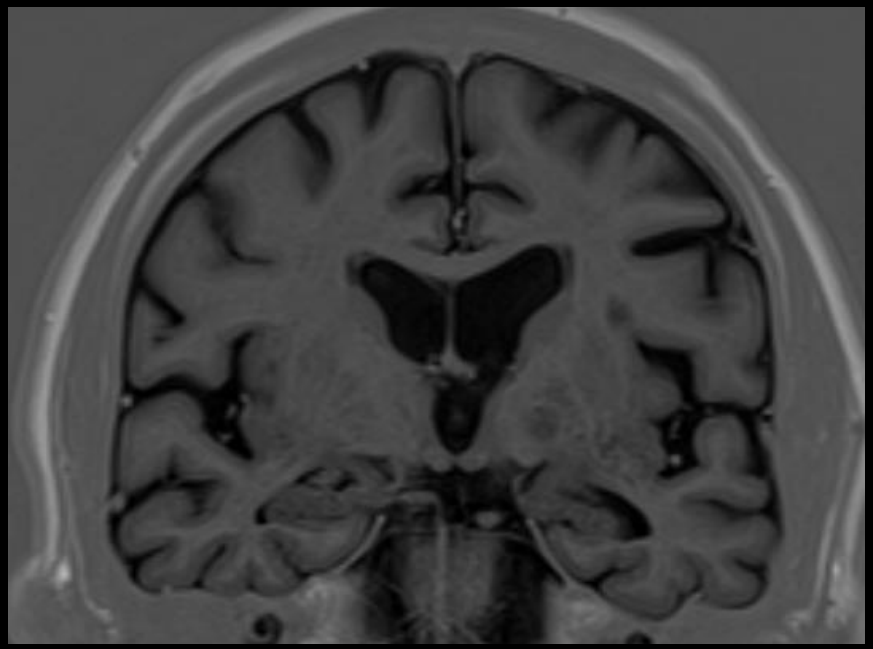

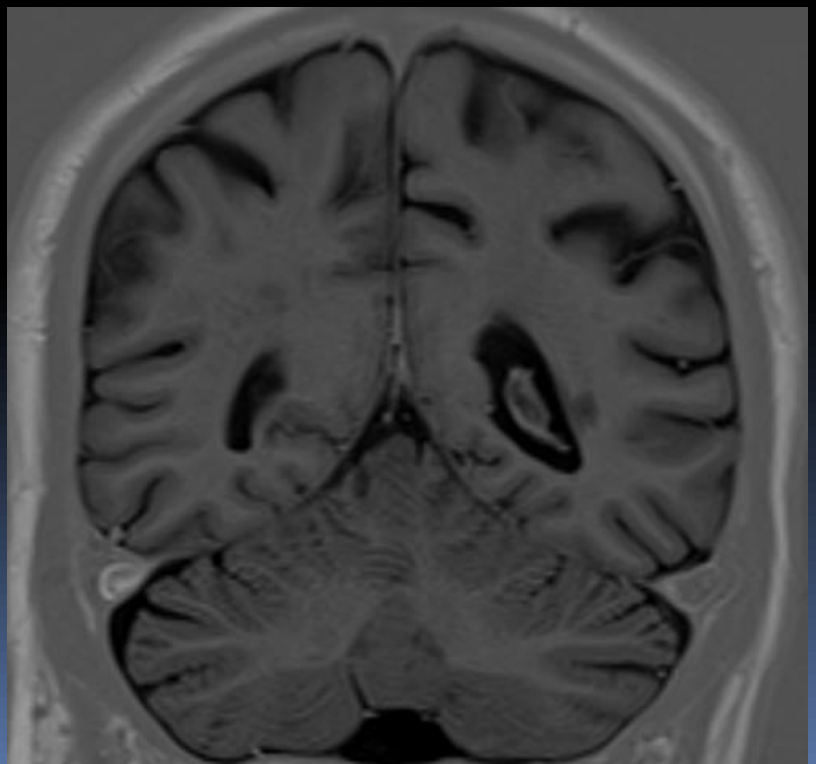

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Cas clinique 3:DIAG ?

- TNCog Léger dysexécutif cptt multidomaine
- Progressif sur 3 ans
- Avec tr sommeil particulier
- Allure NDégénérative
- Diag ?

- Quelles explorations de 2° ligne ?

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Cas clinique 3:DIAG ?

- TNCog Léger dysexécutif cptt multidomaine
- Progressif sur 3 ans
- Avec tr sommeil particulier
- Allure NDégénérative
- -> MCLewy prodromale ?

- Quelles explorations de 2° ligne ?

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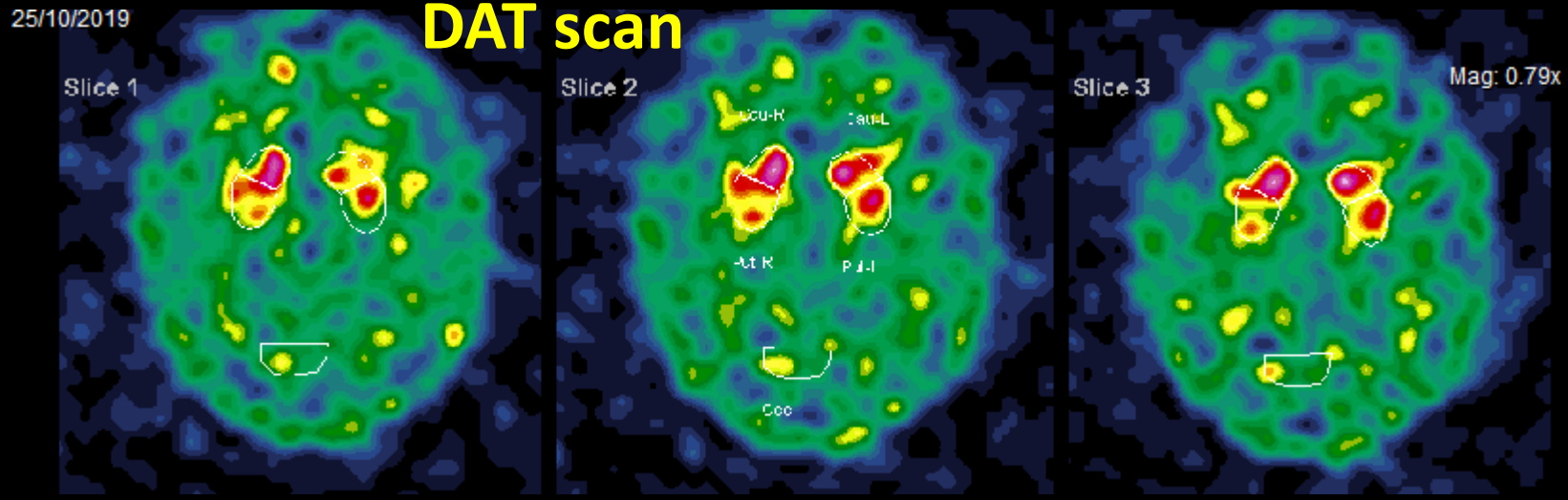
Paraclinique



- **Polysomnographie:** très nombreux épisodes d'activité motrice durant le sommeil paradoxal avec mouvements brusques, vocalisations, bagarres.

- **PET amyloïde**: **négatif**

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Cas clinique 4: suivi

- **Sept 2020**
  - MMSE 23
  - **perception d'ombres évoquant une présence humaine**
  - **télébradykinésie** mineure MSG
- **Sept 2021**
  - **hallucinations visuelles: MT** ->TERCIAN 8 mg le soir
  - MMSE: **15** / 4IADL=**9**/12
  - -> accepte intro donepezil et de réduire TERCIAN
- Avril 2022
- Juillet 2022

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Cas clinique 4: suivi

- **Avril 2022**: donepezil 10 mg EFFEXOR LP75 TERCIAN 5mg
  - **Diminution des hallu, MMSE 17**
  - hypomimie, lenteur, discrète hypertonie parkinsonienne symétrique, dysarthrie
    ->arret progressif TERCIAN
- **Juillet 2022** donepezil 10 mg et EFFEXOR LP75
  - 1 épisode **confusionnel** lors Sd grippal CoVid+
  - **Tr orient topo** (y compris domicile, nottt nuit)
  - Aide pour l'ensemble des activités: toilette, alimentation, déplacements …
  - **MMSE 12**

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Table 1 Revised[1,2] criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies (DLB)

Essential for a diagnosis of DLB is dementia, defined as a progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational functions, or with usual daily activities. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention, executive function, and visuoperceptual ability may be especially prominent and occur early.

Core clinical features (*The first 3 typically occur early and may persist throughout the course.*)

Fluctuating cognition with pronounced variations in attention and alertness.
Recurrent visual hallucinations that are typically well formed and detailed.
REM sleep behavior disorder, *which may precede cognitive decline.*
One or more spontaneous cardinal features of parkinsonism: these are bradykinesia (defined as slowness of movement and decrement in amplitude or speed), rest tremor, or rigidity.

Supportive clinical features

Severe sensitivity to antipsychotic agents; postural instability; repeated falls; syncope or other transient episodes of unresponsiveness; severe autonomic dysfunction, e.g., constipation, orthostatic hypotension, urinary incontinence; hypersomnia; hyposmia; hallucinations in other modalities; systematized delusions; apathy, anxiety, and depression.

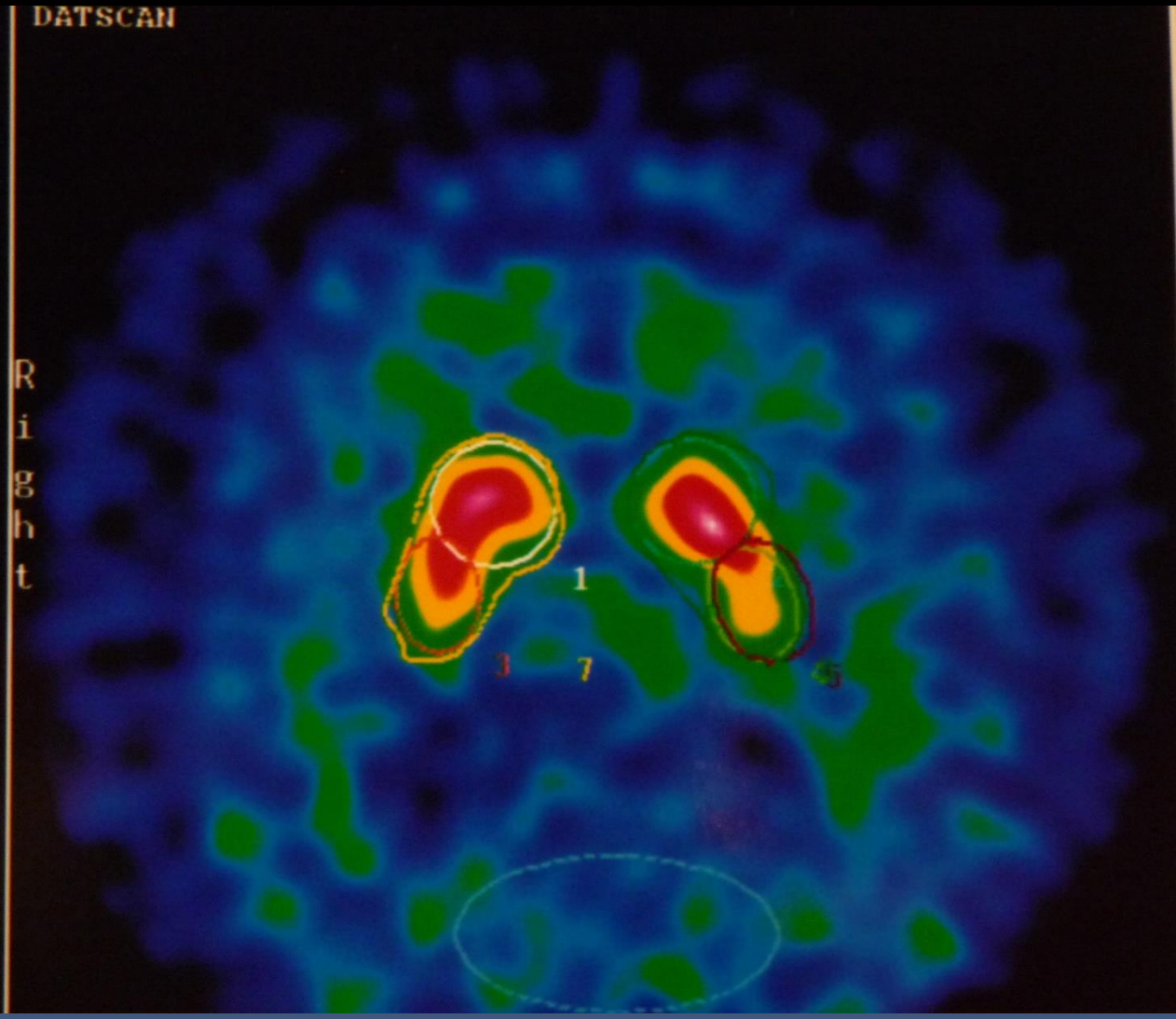
Indicative biomarkers

Reduced dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET.
Abnormal (low uptake) ^{123}iodine-MIBG myocardial scintigraphy.
Polysomnographic confirmation of REM sleep without atonia.

Supportive biomarkers

Relative preservation of medial temporal lobe structures on CT/MRI scan.
Generalized low uptake on SPECT/PET perfusion/metabolism scan with reduced occipital activity ± the cingulate island sign on FDG-PET imaging.
Prominent posterior slow-wave activity on EEG with periodic fluctuations in the pre-alpha/theta range.

REGION CTS/PIX	REGION MAX	REGION MIN
256.0	378	103
233.6	386	85
220.0	331	101
203.8	354	110
193.9	363	83
99.6	139	76
220.4	378	92
204.8	386	81



Probable DLB can be diagnosed if:

a. Two or more core clinical features of DLB are present, with or without the presence of indicative biomarkers, or

b. Only one core clinical feature is present, but with one or more indicative biomarkers.

Probable DLB should not be diagnosed on the basis of biomarkers alone.

Possible DLB can be diagnosed if:

a. Only one core clinical feature of DLB is present, with no indicative biomarker evidence, or

b. One or more indicative biomarkers is present but there are no core clinical features.

DLB is less likely:

a. In the presence of any other physical illness or brain disorder including cerebrovascular disease, sufficient to account in part or in total for the clinical picture, although these do not exclude a DLB diagnosis and may serve to indicate mixed or multiple pathologies contributing to the clinical presentation, or

b. If parkinsonian features are the only core clinical feature and appear for the first time at a stage of severe dementia.

DLB should be diagnosed when dementia occurs before or concurrently with parkinsonism. The term Parkinson disease dementia (PDD) should be used to describe dementia that occurs in the context of well-established Parkinson disease. In a practice setting the term that is most appropriate to the clinical situation should be used and generic terms such as Lewy body disease are often helpful. In research studies in which distinction needs to be made between DLB and PDD, the existing 1-year rule between the onset of dementia and parkinsonism continues to be recommended.

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Research criteria for the diagnosis of prodromal dementia with Lewy bodies

Ian G. McKeith, F Med Sci, MD, Tanis J. Ferman, PhD, Alan J. Thomas, PhD, Frédéric Blanc, MD *Neurology*® 2020;

Table 1 Research criteria for the clinical diagnosis of probable and possible MCI-LB

Essential for a diagnosis of MCI-LB is MCI defined by the presence of each of the following:
Concern by the patient, informant, or clinician regarding cognitive decline.
Objective evidence of impairment in 1 or more cognitive domains. The cognitive impairment may include any domain, but is more likely to be associated with attention-executive and/or visual processing deficits.
Preserved or minimally affected performance of previously attained independence in functional abilities, which do not meet the criteria for dementia.
Core clinical features
Fluctuating cognition with variations in attention and alertness.
Recurrent visual hallucinations.
RBD.
One or more spontaneous cardinal features of parkinsonism: these are bradykinesia (defined as slowness of movement and decrement in amplitude or speed), rest tremor, or rigidity.
Proposed biomarkers
Reduced dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET.
Polysomnographic confirmation of REM sleep without atonia.
Reduced meta-iodobenzylguanidine (MIBG) uptake on myocardial scintigraphy.

Research criteria for the diagnosis of prodromal dementia with Lewy bodies

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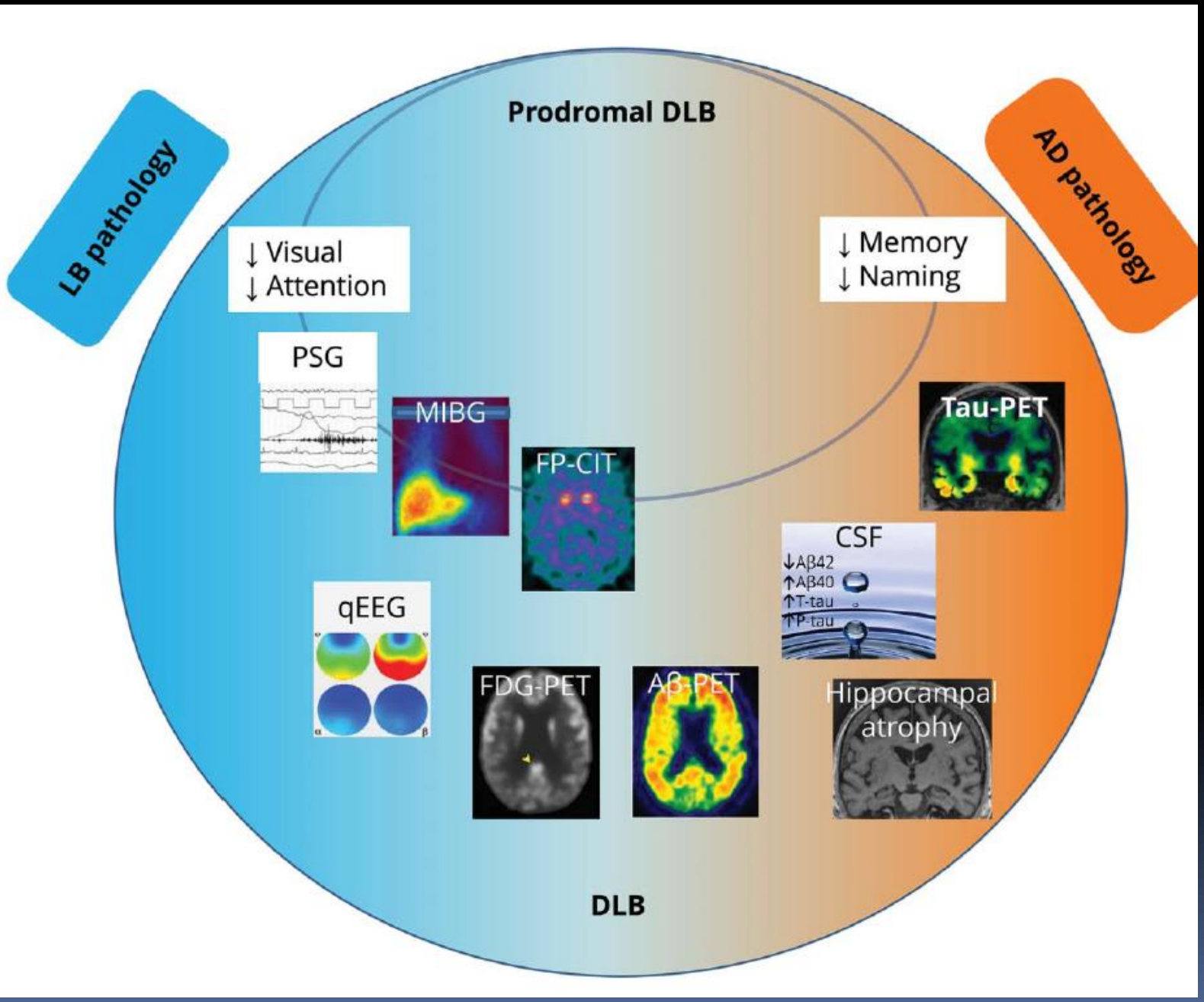
Probable MCI-LB can be diagnosed if:
Two or more core clinical features of DLB are present, with or without the presence of a proposed biomarker, or
Only 1 core clinical feature is present, but with 1 or more proposed biomarkers.
Probable MCI-LB should not be diagnosed based on biomarkers alone.
Possible MCI-LB can be diagnosed if:
Only 1 core clinical feature of DLB is present, with no proposed biomarkers, or
One or more of the proposed biomarkers is present, but there are no core clinical features.
Supportive clinical features
Severe sensitivity to antipsychotic agents; postural instability; repeated falls; syncope or other transient episodes of unresponsiveness; prolonged or recurrent delirium; autonomic dysfunction, e.g., constipation, orthostatic hypotension, urinary incontinence; hypersomnia; hyposmia; hallucinations in other modalities including passage, and sense of presence phenomena; systematized delusions; apathy, anxiety, and depression.


LB pathology
Prodromal DLB
AD pathology
↓ Visual
↓ Attention
↓ Memory
↓ Naming
PSG
MIBG
FP-CIT
Tau-PET
CSF
↓Aβ42
↑Aβ40
↑T-tau
↑P-tau
qEEG
FDG-PET
Aβ-PET
Hippocampal atrophy
DLB

Tr. Cognitifs: interfaces avec psychiatrie

- Qq rappels: cognition et ses troubles
- Critères diagnostiques MA, TCVa, DFT, MCLewy
- **Interfaces avec Psychiatrie**
  - **F. 'psychotiques' MCLewy**,
  - Démences 'vésaniques'
    - M. bipolaire et TOC 'vieillis'
    - Formes déficitaires tardives de schizo et MA
  - Encéphalites Ac AR NMDA
- Qq mots d'actualités: Ttt MA

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[redacted]

Table 3 Summary of key features of psychiatric-onset DLB

Is characterized by predominant psychiatric symptoms that typically correspond to late-onset major depressive disorder or late-onset psychosis, which may feature hallucinations in visual and in other modalities, and systematized delusions including Capgras syndrome
• may also present with apathy, anxiety, and depression
• may be sufficiently severe to require hospitalization
• the frequency of LB disease as a cause of late-onset psychiatric disorder is not known
When assessing for core clinical features of DLB in a patient with a primary psychiatric presentation:
• bradykinesia may be mimicked by psychomotor retardation, which is commonly seen in depressive disorders
• parkinsonism may be induced by antipsychotic medications used to treat psychiatric disorder
• RBD (and REM sleep without atonia) may be induced by antidepressant medications
• mild cognitive disturbance may be present but is not predominant and may fluctuate
• formal neuropsychological testing may be confounded by the psychiatric mental state
• the frequency and character of cognitive fluctuations is unknown
Identification of psychiatric-onset DLB
• may be assisted by use of MCI-LB biomarkers, but further research evidence of this is required
• is important to inform the management plan including the avoidance or minimization of antipsychotic and anticholinergic agents