EAN 2026

A total of 9,503 participants attended, comprising 7,590 onsite delegates and 1,913 joining virtually, representing 121 countries

Congress Review

Review of the European Academy of Neurology (EAN) Congress 2026

Location:	Geneva, Switzerland
Date:	27th–30th June 2026
Citation:	EMJ Neurol. 2026;14[1]:10-21. https://doi.org/10.33590/emjneurol/YH48S4OT

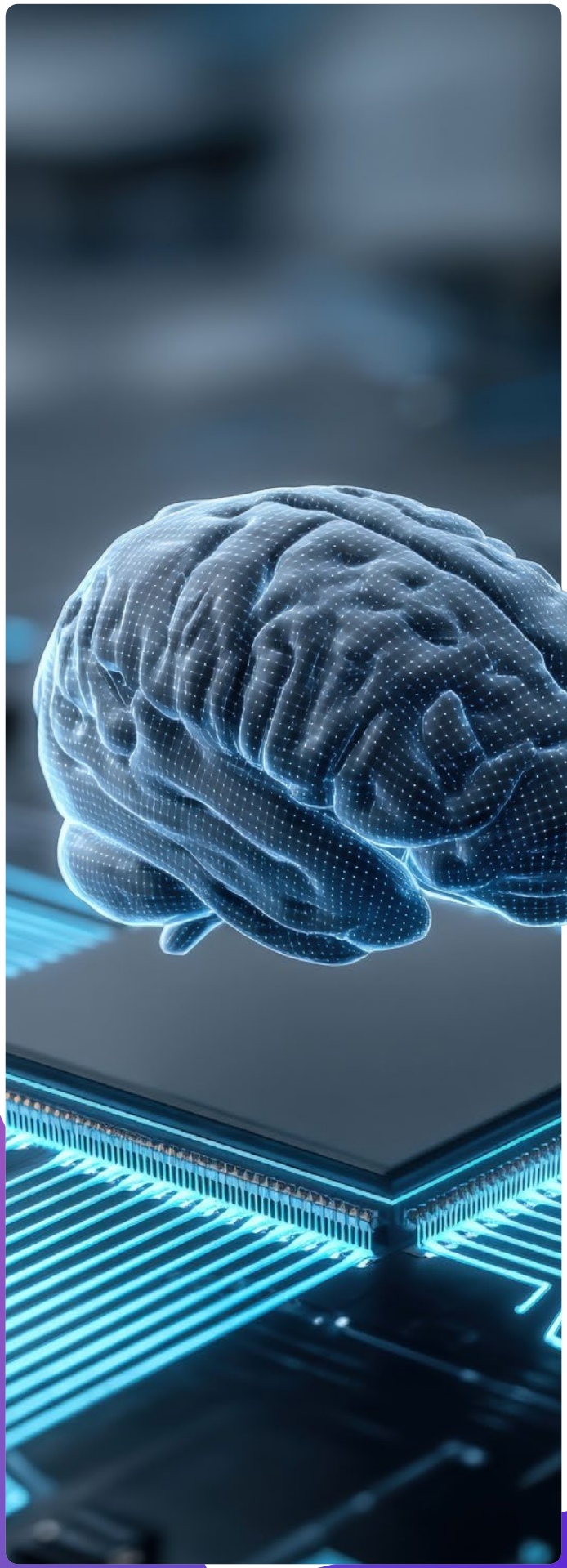
THE EUROPEAN Academy of Neurology (EAN) Congress 2026 opened in Geneva, Switzerland, with EAN President Elena Moro, Department of Psychiatry, Neurology and Neurological Rehabilitation, Grenoble University Hospital Center, France, welcoming participants to "my home, your home, the home of neurology." Geneva's long association with international dialogue suited the message, and Moro reminded the audience that neurology knows no borders.

This year, the community came together in record numbers. A total of 9,503 participants attended, comprising 7,590 onsite delegates and 1,913 joining virtually, representing 121 countries. More than 370 speakers contributed to the scientific programme, underlining the breadth, diversity, and global relevance of the EAN community.

During the Opening Session on Saturday 27th June, Moro presented the EAN's priorities for shaping the future of neurology across Europe, highlighting the Enhancing Neurology in Europe initiative and the associated Brussels Neurology Declaration, signed by presidents and delegates of national neurological societies. Through this, EAN is advancing a common agenda built around brain health for all, equal access to treatment and funding, improved prevention and care, a stronger neurological workforce, interventional neurology, research and innovation, and the responsible integration of new technologies and AI.

Brain health was singled out as one of the strongest examples of cross-border collaboration, with EAN's advocacy now extending from national settings to European and global policy platforms, including the European Parliament and the United Nations, alongside continued work with the WHO to support implementation of the Intersectoral Global Action Plan. Moro described how the Brain Health Mission has grown into an inclusive platform bringing together neurologists, strategic partners, patient organisations, and stakeholders beyond neurology, with initiatives ranging from school-based brain health activities to Public Brain Health Day in Geneva.

The Presidential Symposium, held on Sunday 28th June, brought together the Congress' Named Lectures, presented to outstanding, active basic and clinical scientists. Frank Winkler, Department of Neurology, Heidelberg University Hospital, Germany, delivered the Brain Prize Lecture on neural influences on brain tumour growth and therapy resistance. Manju Kurian, University College London, UK, gave the Anita Harding Award Lecture on the translational arc for childhood movement disorders. Daniela Berg, University Hospital Tuebingen, Germany,



presented the Moritz Romberg Award Lecture, reflecting on what research and patients can teach us about Parkinson's disease. John Rothwell, University College London, UK, delivered the Charles Édouard Brown-Séquard Award Lecture on non-invasive neuromodulation in neurology, and Riccardo Soffietti, University of Turin, Italy, closed the session with the Camillo Golgi Award Lecture on progress in gliomas, from histology to molecular biology and from surgery to precision therapies.

The Congress was built around the overarching theme 'Brains, Bytes & Beyond: Tech in Neurology', which explored how computing is finding its way into neurological practice. Rapid advances in computing are transforming clinical medicine, and the outsourcing of core cognitive tasks from human agents to AI brings both opportunity and risk. The theme ran through a selection of invited lectures, a dedicated symposium on Innovations in Neurology, and two workshops examining AI applications across hospital and outpatient settings, with key sessions including 'AI in Hospital Neurology', 'Innovations in Neurology: From Brain Machine Interface to AI', and 'AI in Outpatient Neurology'.

As the Congress came to a close, it also marked a change in leadership, with Kailash Bhatia, University College London, UK, formally taking office as EAN President and Moro moving into the role of Past President. EMJ had the pleasure of interviewing both Bhatia and Moro. Find their highlights and thoughts on what lies ahead for EAN within this issue of *EMJ Neurology*.

Read on for key insights into this year's Congress, and don't miss our coverage of the EAN Congress 2027, which will be held in Gothenburg, Sweden, with the overarching theme 'Transforming Neurology: Embracing Every Brain'.

Frailty Associated with Structural and Metabolic Brain Changes in Cognitively Unimpaired Adults

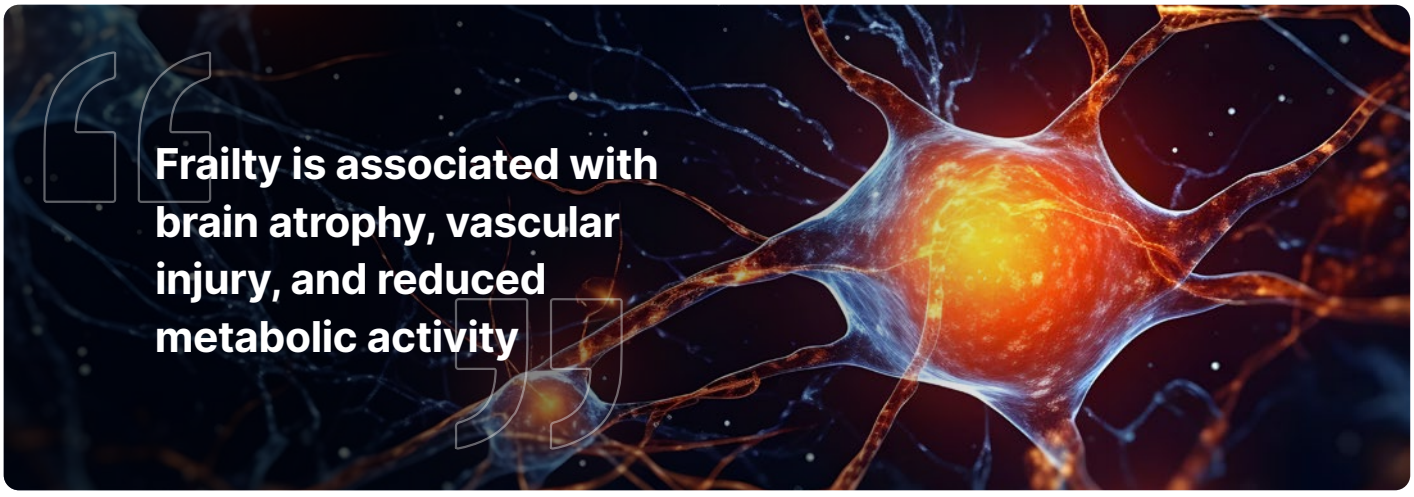
NEW DATA presented at EAN 2026 has linked frailty in middle-aged adults with no cognitive impairment to measurable brain changes, including thinner cortex, smaller hippocampal volume, and lower brain glucose metabolism, with these patterns appearing regardless of amyloid status.[1]

Frailty is increasingly recognised as a multidimensional syndrome that raises dementia risk, yet how it affects the brain before cognitive symptoms emerge is not well understood. Understanding these early changes could help clarify whether frailty contributes to later cognitive decline through pathways distinct from Alzheimer's disease. This presentation examined structural and metabolic brain differences linked to frailty in middle-aged adults with no cognitive impairment, drawn from the ALFA+ cohort, all of whom had cerebrospinal fluid data on Alzheimer's disease biomarkers available.

Frailty status was determined using a 35-item Frailty Index. Linear regression models were then used to test how frailty scores related to several brain measures: predicted brain age, cortical thickness across the whole brain and in specific regions, overall brain volume, the extent of white matter hyperintensities, and glucose metabolism on PET imaging. All models were also rerun after accounting for cerebrospinal fluid amyloid-β 42/40 ratios, to establish whether any associations held independently of amyloid status. Of the 418 adults included in the analysis, 57 met the criteria for frailty.

Frailty scores correlated with an older-appearing brain on predicted brain-age measures, along with thinning in average, fronto-temporal, and Alzheimer's-signature cortical regions, and a smaller hippocampus. Glucose uptake was also lower in frail participants across average, parietotemporal, and posterior cingulate regions, a pattern consistent with reduced neuronal activity in these areas. White matter hyperintensities, a marker of small vessel damage, were more extensive among frail participants, most notably in the fronto-parietal and basal ganglia regions. None of these relationships weakened once amyloid-β levels were accounted for.

Taken together, the findings suggest that frailty is associated with brain atrophy, vascular injury, and reduced metabolic activity in regions typically affected by Alzheimer's disease and normal ageing, and that this pattern holds regardless of amyloid status. The authors framed frailty as a possible early marker of brain vulnerability that may appear before dementia becomes clinically apparent. As the data were observational, the findings show association rather than a confirmed causal or predictive role.



RFC1 Repeat Expansions May Explain CANVAS Mechanism

NEW RESEARCH, presented at EAN 2026, suggests that AAGGG repeat expansions in the *RFC1* gene may contribute to the development of cerebellar ataxia, neuropathy, and vestibular areflexia syndrome (CANVAS) by reducing *RFC1* expression, with a consequent impairment of the DNA damage response. The findings also indicate that these changes could increase the risk of clinically significant neuropathy following oxaliplatin treatment.[2]

CANVAS is a progressive neurological disorder characterised by impaired balance and coordination, sensory neuropathy, and vestibular dysfunction, and is increasingly recognised as one of the most common causes of ataxia and sensory neuropathy.

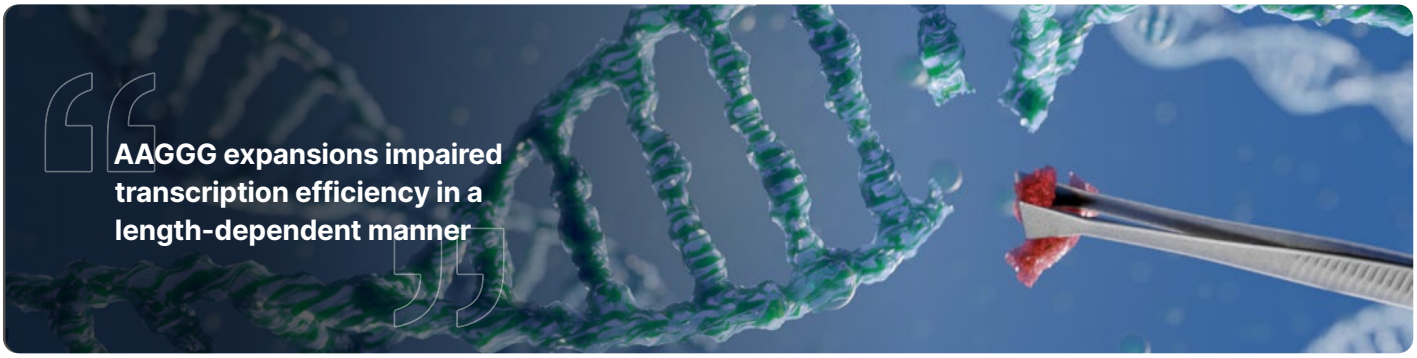
Although previous studies failed to detect reduced *RFC1* transcript or RCF1 protein levels, the recessive inheritance pattern of CANVAS and the identification of patients carrying compound heterozygous null variants have suggested that loss of RFC1 function may underlie the disease. Researchers therefore examined how AAGGG repeat expansions influence *RFC1* expression and function.

The team used reporter assays, human cerebellar tissue, patient-derived cell lines, induced pluripotent stem-cell-derived neurones, and a *Drosophila* model to investigate the effects of the repeat expansion. They found that AAGGG expansions impaired transcription efficiency in a length-dependent manner, reduced *RFC1* expression in the human cerebellum, and induced pluripotent stem-cell-derived neurones. The expansions also impaired the DNA damage response. Patient-derived cell lines also showed an increased apoptotic response after exposure to platinum compounds.

CRISPR/Cas9-mediated removal of the AAGGG repeat restored *RFC1* expression and the DNA damage response in patient-derived cells, reversing key cellular changes observed in the disease model. In addition, neuronal-specific *RFC1* knockdown in flies resulted in reduced survival, motor impairment, and increased DNA damage.

The researchers also found that people carrying *RFC1* repeat expansions who received oxaliplatin were at higher risk of developing clinically significant neuropathy than non-carriers.

The findings support a cell- and tissue-specific reduction in *RFC1* expression as a potential mechanism underlying CANVAS and provide a foundation for exploring strategies that restore *RFC1* expression and DNA repair. Further research will be needed to determine how these findings translate into clinical practice, but they could support the development of therapies aimed at restoring *RFC1* expression and help guide future studies into chemotherapy-induced neuropathy in people carrying *RFC1* repeat expansions.



Women with Parkinson's Disease Show Greater Alzheimer's Pathology Burden

WOMEN with Parkinson's disease (PD) may be more likely than men to develop co-existing Alzheimer's disease (AD) pathology, according to research presented at EAN 2026.[3]

Women had more than double the odds of having a high amyloid plaque burden compared with men

AD and PD frequently occur together in older adults, but little is known about whether biological sex influences the development of Alzheimer's-related pathology in people with PD.

In this study, researchers examined 230 autopsy-confirmed cases of PD enrolled in the Arizona Study of Aging and Neurodegenerative Disorders (AZSAND) and the Brain and Body Donation Program (BBDP). Participants underwent annual standardised clinical assessments conducted by neuropsychologists and specialist behavioural and movement disorder neurologists, followed by comprehensive neuropathological examinations after death.

The investigators compared measures of AD pathology between male and female patients with PD, focusing particularly on amyloid plaque burden within the brain.

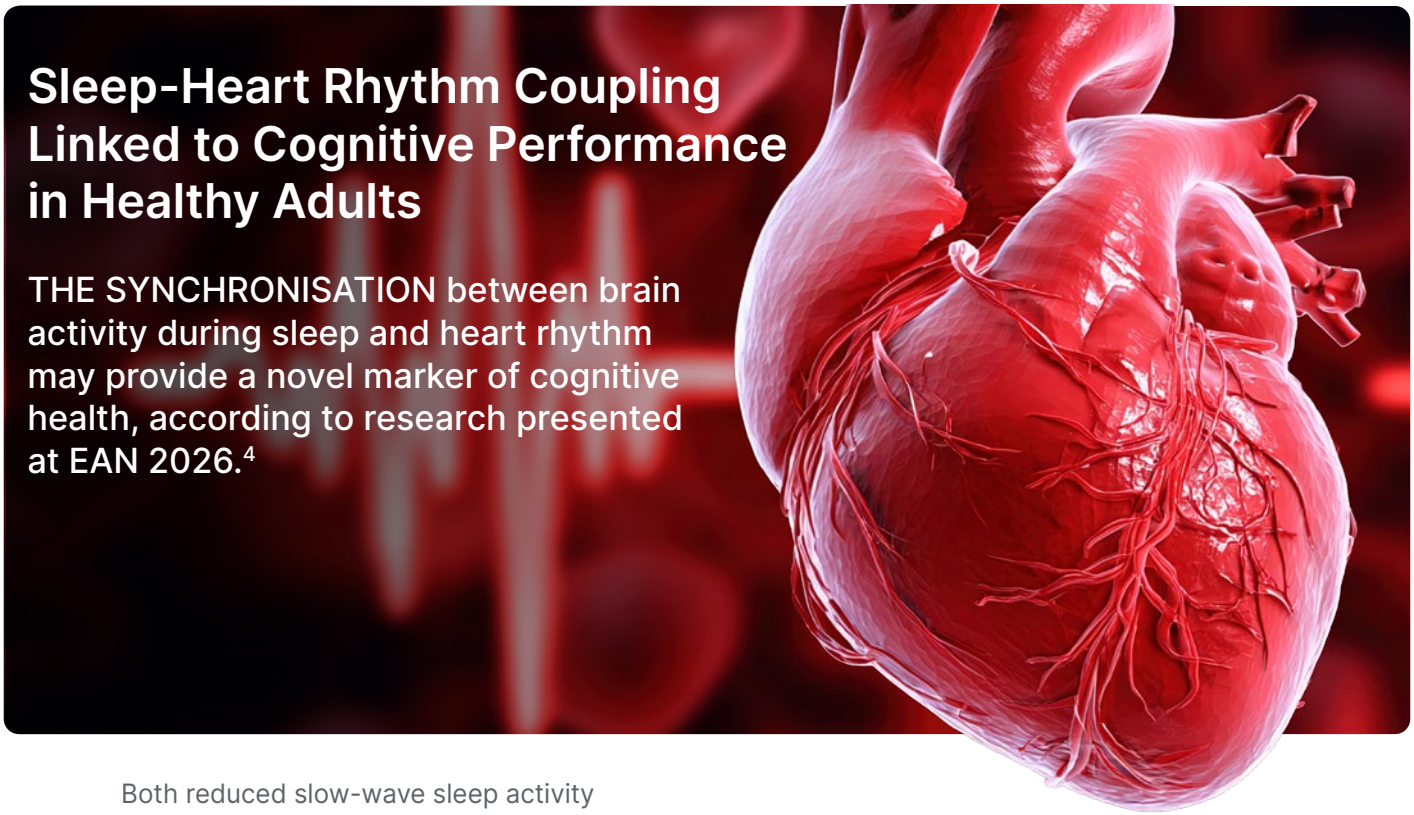
Women with PD demonstrated significantly greater amyloid plaque pathology than men, regardless of whether they also met diagnostic criteria for AD. Female patients had higher mean cortical total plaque scores than male patients (6.5/15 versus 4.9/15; p=0.045) and greater Consortium to Establish a Registry for Alzheimer's Disease (CERAD) neuritic plaque density (1.7/3 versus 1.3/3; p=0.035).

Women were also more likely to have a high cortical plaque burden, defined as a total plaque score of at least 5 (56.8% versus 39.7%; p=0.015).

After adjusting for age at death and *ApoE* ε4 carrier status, female sex remained independently associated with greater amyloid pathology. Women had more than double the odds of having a high amyloid plaque burden compared with men (odds ratio: 2.18; 95% CI: 1.17–4.06; p=0.014).

The authors concluded that female sex is associated with increased amyloid plaque pathology in Parkinson's disease, independent of *ApoE* ε4 status. These findings suggest a sex-specific vulnerability to Alzheimer's pathology among patients with PD and highlight the need for sex-informed approaches to the diagnosis and treatment of mixed neurodegenerative disease.

Sleep-Heart Rhythm Coupling Linked to Cognitive Performance in Healthy Adults



THE SYNCHRONISATION between brain activity during sleep and heart rhythm may provide a novel marker of cognitive health, according to research presented at EAN 2026.[4]

Both reduced slow-wave sleep activity and cardiac autonomic dysfunction have previously been associated with cognitive impairment. However, little is known about how interactions between these two physiological processes influence cognitive function.

Researchers analysed data from 63 healthy volunteers who underwent cognitive testing and overnight sleep studies. A subset of 31 participants also received high-density EEG using 256 electrodes to map the cortical distribution of slow-wave–heart rhythm (SW-HR) coupling during sleep.

The team assessed several characteristics of SW-HR coupling, including temporal coherence, slow-wave amplitude, and globality, and examined their relationship with performance across multiple cognitive domains.

The findings showed that SW-HR coupling was bidirectional and predominantly involved fronto-central brain regions. Stronger temporal and amplitude coupling was associated with better performance in several cognitive functions, including alertness, response inhibition, verbal memory, and visual memory. Increased slow-wave globality was also linked to improved cognitive outcomes, particularly visuospatial memory recall. No significant associations were observed for measures of density coupling.

According to the authors, this is the first study to describe the cortical topography of SW-HR coupling and its relationship with cognition across multiple domains. The findings suggest that the interaction between sleep-related brain activity and autonomic function may represent a promising biomarker of brain health and cognitive performance.

Apathy Predicts Cognitive Decline in Early Parkinson's Disease

A STUDY presented at EAN 2026 investigated whether apathy and impulse-control behaviours (ICB) were associated with longitudinal cognitive decline, alterations in brain connectivity and structure, and genetic susceptibility in early Parkinson's disease (PD).[5]

PD is frequently accompanied by non-motor symptoms, including motivational disturbances such as apathy and ICBs, which may influence long-term cognitive outcomes. While both are common in early PD, their relative contribution to cognitive decline and the biological mechanisms underpinning these associations remain unclear.

In this study, a key finding was that baseline apathy predicted significantly faster cognitive decline over up to 15 years of follow-up, whereas ICBs showed little consistent association.

Data were analysed from the Parkinson's Progression Markers Initiative (PPMI), including 1,502 participants with early PD at baseline. Participants underwent longitudinal cognitive assessment using latent global cognition and Montreal Cognitive Assessment (MoCA) scores. Resting-state functional MRI data were available for 310 participants, with structural imaging used to assess hippocampal volume and atrophy. The study also examined genetic susceptibility, including glucocerebrosidase (GBA) mutation status, while accounting for demographic factors, motor severity, depressive symptoms, and dopaminergic treatment.

Apathy-containing phenotypes remained comparatively stable over time, whereas isolated ICBs were more transient. Higher baseline apathy independently predicted steeper decline in both latent global cognition and MoCA scores across follow-up, while ICB-related effects were weak and not robust after adjustment.

Functional MRI identified an apathy-associated somato-motor–dorsal-attention connectivity signature that was independent of disease severity and dopaminergic medication. Lower connectivity within this network predicted faster cognitive decline, mediated the relationship between apathy and cognitive deterioration in an age-dependent manner, and was associated with smaller hippocampal volume and more rapid subsequent atrophy.

In contrast, ICB-related connectivity changes were attenuated after adjustment for levodopa-equivalent dose. Among participants with GBA-associated PD, apathy markedly increased vulnerability, with apathetic GBA carriers declining approximately five times faster than non-apathetic sporadic cases.

> **Baseline apathy predicted significantly faster cognitive decline over up to 15 years of follow-up**

These findings suggest that apathy is a stable clinical marker of future cognitive decline in early PD and may help identify individuals at increased risk for accelerated progression. Routine assessment of apathy could therefore support earlier risk stratification, monitoring, and targeted intervention in clinical practice, particularly in patients with GBA-associated PD. Limitations include the observational design, reliance on a single research cohort, and the smaller imaging subgroup, which may limit causal inference and generalisability.

Seizure Improvement After Vorasidenib May Correlate with F-DOPA PET and D2HG

SEIZURE improvement after vorasidenib in post-surgery patients with Grade 2 isocitrate dehydrogenase (IDH)-mutant glioma may be correlated with fluorodopa (F-DOPA) PET and D-2-hydroxyglutarate (D2HG) plasma levels, according to a new prospective study presented at EAN 2026.[6]

The utilisation of IDH inhibitors to improve seizure control is appealing because of the involvement of IDH mutations and D2HG in epileptogenesis. Researchers assessed patterns and timing of seizure response in patients with Grade 2 IDH-mutant glioma receiving vorasidenib after having surgery.

The cohort comprised 56 patients, with seizures recorded at the end of each 28-day cycle. MRI, [18F] F-DOPA PET, and D2HG plasma levels were performed at baseline and every three cycles.

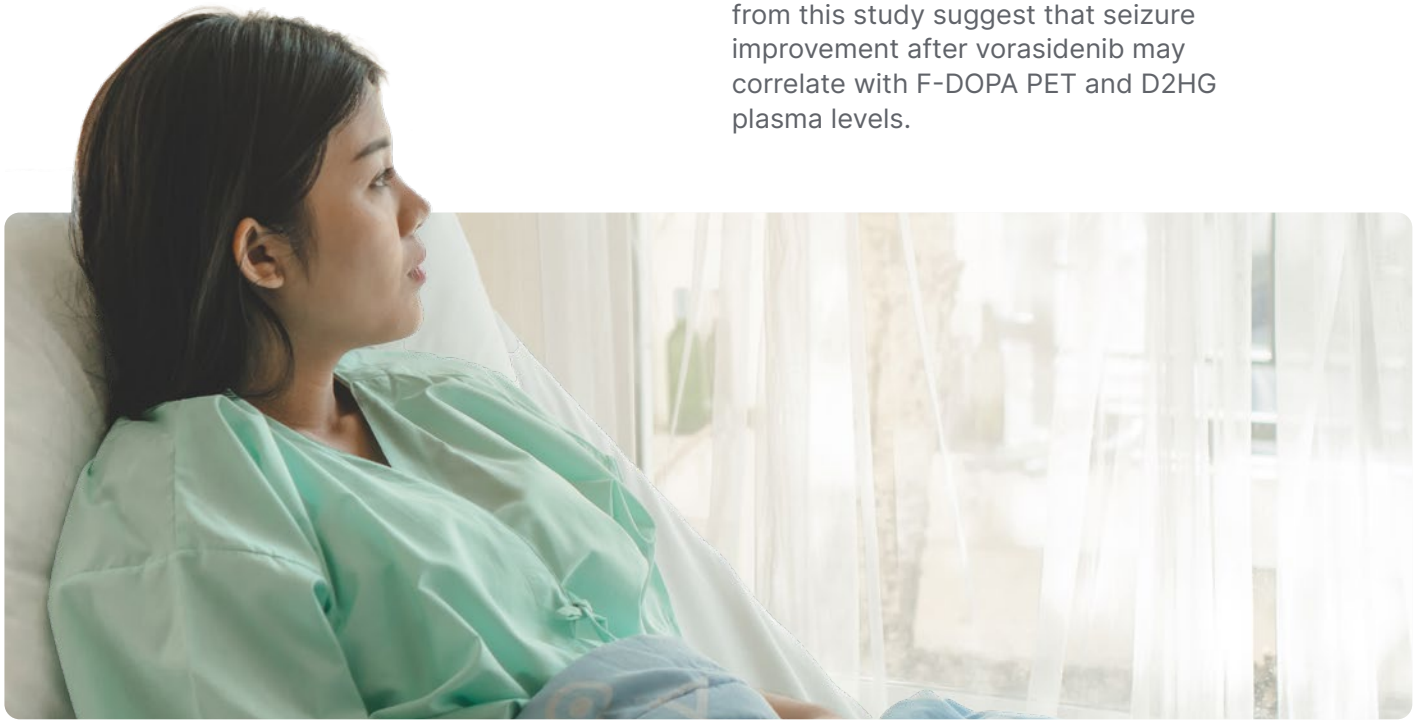
Twelve patients in the cohort had persistent seizures prior to vorasidenib, with a mean frequency of four per month. Nine of these 12 had measurable F-DOPA PET uptake.

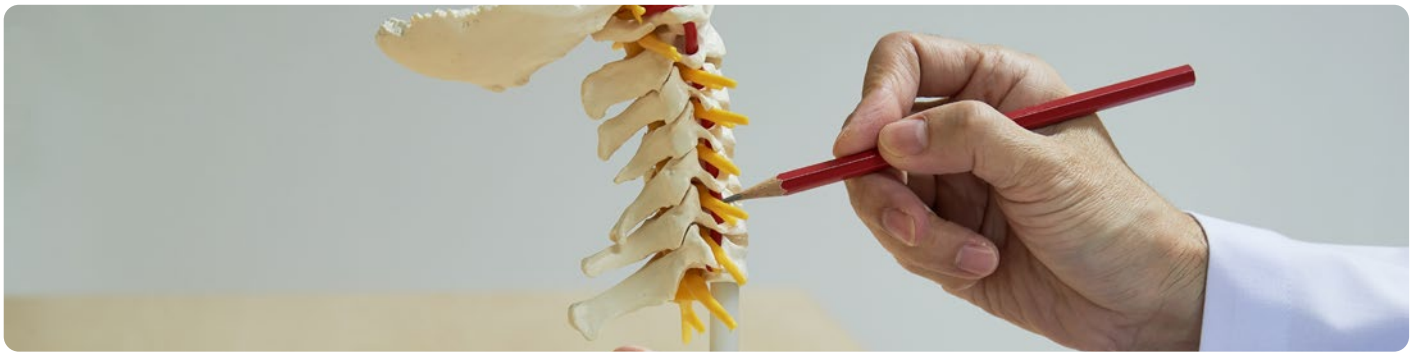
Over a median treatment duration of 11.6 months, six out of 12 patients achieved seizure freedom within one treatment cycle, while five out of 12 experienced gradual seizure improvement. One patient experienced an initial improvement but subsequently worsened by Cycle 4.

Among patients with seizure reduction, eight out of nine showed reduced F-DOPA PET uptake by Cycle 3. Additionally, all 11 patients with durable seizure responses demonstrated reductions in plasma D2HG levels at Cycles 3 and 6 (mean change: −51% and −60%, respectively). MRI assessments showed stable disease in all patients according to response assessment in neuro-oncology criteria.

These results contrasted with the patient who experienced seizure worsening over time. D2HG reduction was −53% at Cycle 3, but the patient demonstrated no further reduction or PET progression disease at Cycle 6.

Researchers concluded that the findings from this study suggest that seizure improvement after vorasidenib may correlate with F-DOPA PET and D2HG plasma levels.





Cervical Cord Atrophy in MS Exceeds Normal Ageing Effects

UPPER cervical cord atrophy in patients with multiple sclerosis (MS) exceeds what would be expected from ageing alone, according to research presented at EAN 2026. This disease-specific effect is most pronounced in early adulthood and midlife, and is independent of sex or age at disease onset.[7]

Brain MRI and clinical records were drawn from 1,295 people with MS

Loss of spinal cord tissue is a recognised driver of disability in MS, and mean upper cervical cord cross-sectional area (MUCCA) is a validated way to track it in both newly diagnosed and long-standing disease. The relative contribution of disease-specific neurodegeneration, as distinct from ageing that would occur independently of diagnosis, has proven more difficult to quantify. To address this, the study built a reference curve for how MUCCA normally changes across life, then measured how far people with MS deviated from it.

Brain MRI and clinical records were drawn from 1,295 people with MS and 480 people without the condition, aged 18–70 years. MUCCA was measured at the C1 to C2/3 level and normalised for head size (nMUCCA). A regression model incorporating age, the square of age, sex, scanner type, and their interactions was fitted to the healthy group to define an expected nMUCCA trajectory across the lifespan; each person with MS was then scored against this curve to generate a Z-score quantifying how far their nMUCCA fell outside the age-typical range. The analysis also tested whether these Z-scores varied by sex or by whether the disease had started in childhood, adulthood, or after age 50 years.

nMUCCA in the healthy group grew through the third and into the fourth decade of life, peaking around the late 30s ($p \leq 0.033$), then declined from roughly age 50 years onwards ($p \leq 0.008$). Among people with MS, Z-scores worsened steadily from early adulthood through to the late 40s ($p<0.001$), with the pace of decline levelling off in older age groups. Neither the healthy group nor the MS group showed a meaningful difference by sex ($p=0.256$ and $p=0.422$, respectively). Those diagnosed in childhood had worse Z-scores than those diagnosed as adults or after 50 years of age ($p<0.001$), though once diagnosed, the pace of further decline was similar regardless of age of disease onset. Worse Z-scores also tracked with higher disability levels and other markers of brain damage on MRI (rho: −0.355–-0.372; all $p<0.001$).

The findings indicate that cervical cord atrophy in MS exceeds that attributable to ageing alone, an effect most pronounced in early and mid-adulthood, with the relative contribution of ageing increasing in later life. The authors attributed differences between onset groups mainly to how long people had lived with the disease rather than to the age of onset. Because nMUCCA tracked so closely with disability, they suggested it could serve as a marker of disease-driven damage distinct from ageing, though the cross-sectional design means the study shows a strong association rather than proof that it predicts future decline.

Pupil Response May Help Predict Recovery of Consciousness After Acute Brain Injury

A SPECIFIC feature of the pupil's response to light may help predict whether patients with acute brain injury will regain consciousness, according to research presented at EAN 2026.[8]

Predicting neurological recovery in patients who are critically ill remains a significant clinical challenge, particularly during the early stages of intensive care. While automated pupillometry is increasingly used to assess neurological function, its ability to predict longer-term recovery has not been fully established.

In this prospective longitudinal study, researchers followed 250 patients with impaired consciousness after traumatic and non-traumatic brain injury admitted to the ICU. Patients underwent daily pupillometry alongside serial neurological assessments for up to 20 days.

The investigators focused on the late light-off response (LOR), which measures how quickly the pupil begins to dilate after a light stimulus is removed. Specifically, they assessed late LOR latency and examined whether it was associated with changes in patients' level of consciousness over the following week.

Late LOR latency independently predicted improvement in neurological status 7 days later, even after accounting for baseline neurological function, time since injury, and sedation status. Notably, the predictive value of late LOR latency was most apparent in patients who were not sedated and appeared strongest among those with anoxic-ischaemic brain injury.

By contrast, conventional pupillometry measures, including the Neurological Pupil Index and pupillary light reflex latency, did not predict subsequent improvements in consciousness despite often remaining within normal ranges early after injury.

The authors concluded that late LOR latency captures subtle recovery-related changes in pupillary function that may precede clinically meaningful neurological improvement. If validated in future studies, this non-invasive measure could provide clinicians with an additional tool to identify patients with the potential for recovery following acute brain injury.

References

1. Toccaceli Blasi M et al. Frailty is associated with structural and metabolic brain changes in cognitively unimpaired adults. Abstract OPR-077. EAN Congress, 27-30 June, 2026.
2. Curro R et al. CANVAS-associated AAGGG repeat expansions cause cell-specific reduction in RFC1 expression and impaired DNA damage response. Abstract OPR-031. EAN Congress, 27-30 June, 2026.
3. Driver-Dunckley E. Greater burden of Alzheimer's copathology in women with Parkinson's disease. Abstract EPO-0330. EAN Congress, 27-30 June, 2026.
4. Filchenko I et al. Brain and heart cross-talk: cortical topography of slow-wave–heart rhythm coupling is associated with cognition. Abstract OPR-142. EAN Congress, 27-30 June, 2026.
5. Attaallah B. Apathy marks brain network and genetic vulnerability to cognitive decline in Parkinson's disease. Abstract LB_02. EAN Congress, 27-30 June, 2026.
6. Bruno F et al. IDH-mutant gliomas treated with vorasidenib: early seizure control correlates with [18F]- DOPA PET response and D-2-hydroxyglutarate plasma reduction. Abstract OPR-012. EAN Congress, 27-30 June, 2026.
7. Jain K et al. Disentangling age-related and disease-specific upper cervical cord atrophy in multiple sclerosis. Abstract OPR-020. EAN Congress, 27-30 June, 2026.
8. Laigaard P et al. Pupillary light-off latency predicts 7-day improvement in consciousness in patients with acute disorders of consciousness. Abstract OPR-061. EAN Congress, 27-30 June, 2026.