

drial disorders deserve consideration as part of the differential diagnosis, especially if there is suspected involvement of other organ groups or positive family history of MD. There is no specific consensus approach for treating MELAS syndrome. Management is largely symptomatic and should involve a multidisciplinary team.

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#### EV716

### Serine racemase in inhibitory neurons at striatum and it might be involved in schizophrenia's pathophysiology with D1 and D2 receptors

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**Introduction** There is substantial evidence that hypofunction of the N-methyl-D-aspartate receptor (NMDAR) is a core pathophysiological mechanism underlying schizophrenia. D-serine and serine racemase (SR) (NMDAR co-agonist and its producer) are thought to be involved in schizophrenia's pathophysiology as NMDAR function moderators. Our laboratory showed that excitatory neuron specific SR knock out (SRKO) mice still have just 50% reduction of SR whereas full SRKO mice had no SR. Furthermore D-serine and SR are found in inhibitory neurons not only in excitatory neurons with immunohistochemistry methods. Because NMDAR has excitatory functions, the existence of D-serine and SR in inhibitory neurons and their functions are of interest.

**Aims** To elucidate the existence and roles of D-serine and SR in inhibitory neurons.

**Methods** Inhibitory neuron marker, GAD65, specific conditional SRKO (GAD65 SRKO) mice were made by Cre-lox recombination method. The GAD65 SRKO mice were analyzed by HPLC for D-serine concentration, western blotting for SR expression, immunohistochemistry for SR positive cell's character identification and behavioral testing.

**Results** GAD65 SRKO had about 50% reduction of SR in striatum but no reduction in hippocampus and frontal cortex. D-serine of GAD65 SRKO mice was not different from WT mice. Immunohistochemistry works revealed SR is in medium spiny neuron of striatum and has colocalization with DARRP-32, D1 receptor, and D2 receptor.

**Conclusions** SR is expressed in inhibitory neurons at least in striatum. It might be involved in schizophrenia's pathophysiology because it colocalizes with D1 and D2 receptors.

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## Geriatric psychiatry

#### EV717

### Catatonia and dementia

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**Introduction** Catatonia, described by Kahlbaum in 1874, is usually seen as a type of schizophrenia, but it can also occur in a wide

range of other psychiatric/organic disturbances. There is a documented association between dementia and catatonia, in all phases of cognitive impairment.

**Aims** Literature review and discussion about Catatonia, regarding a case report.

**Methods** Clinical interviews and literature review in PUBMED database.

**Results (case report)** Female patient, 89 years old, without psychiatric history, was diagnosed with dementia 5 months prior to episode. On admission, she presents with prostration, mutism and refusal to eat/drink. Laboratory studies were normal and TC-CE shows signs of an old stroke in left temporo-parietal region and diffuse signs of ischemic leucoencephalopathy. At psychiatric evaluation, she was stuporous, unreactive to pain, mute, not following verbal commands, keeping her eyes closed and resisting attempts to open her eyelids. She had global rigidity, axial and limbs, and maintains the postures the examiner puts her into for long periods. She was already given chlorpromazine, without improvement. Then she takes diazepam 10 mg iv, with remission of the state.

**Conclusions** Although catatonia usually presents with drama, clinicians often forget to consider it in differential diagnosis, probably because of its traditional association with schizophrenia. A promptly diagnostic is crucial to provide adequate treatment, avoiding drugs that can worsen/perpetuate the clinical state. Some authors even support the idea that motor features associated with end-line dementias may correspond to lorazepam-responsive catatonia, in which treatment may have a tremendous impact worldwide.

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### Mini-Mental State (MMS) evaluation of dementia in psychiatric patients admitted to a long stay ward

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MMS scores for 41 psychiatric patients were analyzed at admission and regularly throughout their stay.

**Results** Their average age at admission was 65.7. Thirty-six patients had a diagnosis of chronic psychosis, two with bipolar disorders, one with frontotemporal dementia, two with Korsakoff syndrome.

At admission, 21 (51%) patients showed mild cognitive deterioration (score = 18–26), 12 (29%) moderate deterioration (12–17), 6 severe deterioration (0–11), 2 had normal scores (27–30). Over the following years, 28 patients were reassessed:

– 12 (42%) were stable, 7 (25%) had a fluctuating score, 5 (18%) improved;

– 4 (14%) deteriorated over their successive MMS evaluations;

– age, socio-cultural level and psychiatric diagnosis were not associated with change in MMS scores;

– average change between initial and final assessment was +6.0 points for patients with improved score, –7.75 for those showing deterioration;

– 1.28 for those with fluctuating scores, –1.0 for stable patients.

**Analysis** Unstable psychiatric disorders associated with somatic pathologies influenced MMS scores for all patients, particularly for those with MMS deterioration or fluctuation even if this phenomenon could also be observed to a lesser extent in stable patients. By contrast, patients whose MMS scores improved over