Focus on multiple system atrophy

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Published online: 4 April 2015
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Multiple system atrophy (MSA) is the most aggressive of the synucleinopathies. Its relentless course results in a rapid deterioration in movement—that is unresponsive to

Bennarroch et al., 2015 In this issue *Clin Auton Res*

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medications. Patients also suffer from progressive loss of autonomic control with orthostatic hypotension and bladder, bowel, and sexual dysfunction.

We devote this issue of *Clinical Autonomic Research* to multiple system atrophy, an idea that began at the American Autonomic Society's symposium on MSA sponsored by the MSA Coalition. It is a perfect opportunity to welcome Gregor Wenning as an Associate Editor for *Clinical Autonomic Research*. Gregor has made MSA his lifetime research focus. By bringing him on board, we hope to make CAR the journal that covers MSA and provides regular updates on MSA research.

This special issue begins with *Niall Quinn*, who takes us back to Paris in 1900, where the first MSA cases were described. He shares a handwritten letter from Oppenheimer, the neuropathologist who named the disease multiple system atrophy.

Andrew Singleton and collaborators describe emerging data to answer whether predisposition to the disease could lie within our genes. *Stefanova and Wenning* describe how animal work is helping us to understand alpha-synuclein accumulation, nerve death, and progressive loss of function.

Kaufmann and Palma describe the past clinical trials in MSA and the experience so far, as the search for a neuroprotective agent continues. *Biaggioni, Shibao, and Jordan* describe our successes in the management of the cardiovascular autonomic dysfunction that occurs in many patients with MSA. They review how pharmacological tools have helped us to understand why the autonomic nervous system fails in MSA.

Singer and Low provide their insights into the difficulties in performing clinical trials of disease modifying drugs in MSA, and the lessons learned from the recent multicenter trial of rifampicin. They describe the steps that we should take to improve future trials of new therapies.

David Goldstein writes about progress in finding biomarkers that can help distinguish MSA from Parkinson disease with orthostatic hypotension—a differential diagnosis that can be tricky. Several autonomic centers collaborate to describe how acting out dreams is common in MSA and may provide a clue to spotting MSA in the pre-motor phase. *Benarroch and colleagues* describe the rostral ventrolateral medulla and the interplay between the death of cells in the central autonomic network, oligodendroglia, and alpha-synuclein accumulation in a case of MSA. The issue closes with the argument that, due to the rarity of the

disease, an international effort is needed to develop a natural history study of MSA.

Over a century after MSA was first described, we are closer to understanding the neurodegenerative process that corrodes the brain, but we've made no progress in stopping the disorder. It is time to bridge the gap.

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