two patient groups also limits interpretation. The recruitment of consecutive patients, with its inherent variability, can be seen as a strength of the study, but it also creates problems due to the range of disease seen—for example, patients' mini mental state examination scores ranged from 4 to 26. It is therefore not clear how confident one can be about the positive findings irrespective of disease duration or severity given the few patients at each data point. Indeed it could be argued that comparing only mildly affected patients may be more useful as it is this group that represent the greatest challenge both diagnostically and therapeutically—although the absence of effective long term therapies may even limit this potential at the present time.

In conclusion, this study highlights the possibility that neurodegenerative disorders targeting the cerebral cortex may be distinguished through their effects on sympathetic nerve endings in the heart. However, perhaps the most important message from this study is to highlight once more that neurodegenerative disorders display widespread pathology and that this needs to be accommodated in any theory of pathogenesis as well as novel therapeutic approaches.

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Videodynamic and sphincter motor unit potential analyses in Parkinson's disease and multiple system atrophy

In the paper by Sakakibara *et al* (this volune, pp 600–606),[1] the differences in findings of investigations of bladder function between patients with Parkinson's disease and multiple system atrophy are reported. The authors' new findings focus on the urethral sphincter mechanism, examining this functionally (using some of the newer urodynamic indices) and fluoroscopically. But the paper also provides a useful summary, reviewing as it does, previous work in this area, which has so far been mainly published in the urological literature. Although urinary symptoms can be troublesome in advanced Parkinson's disease they do not have the same prominence and severity as those seen in early multiple system atrophy, probably due to the multiple defects of neurological control of the bladder and sphincter function that develop in the initial stages of multiple system atrophy. Abnormalities of urethral sphincter innervation, both the external (striated) and intrinsic (bladder neck) are marked as a feature of multiple system atrophy but not Parkinson's disease, as has been shown in this study.

There has been a tendency up to now to include bladder symptoms as part of the "autonomic failure" which characterises multiple system atrophy, but the growing realisation that the entire system of neurological control of the bladder is selectively involved in its early stages, may lead to a new approach in understanding the evolution of this progressive and fatal neurodegenerative disease.

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Sphincter EMG in possible multiple system atrophy: to do or not to do?

In multiple system atrophy (MSA) the motor neurons of sphincter muscles (Onuf's nucleus) degenerate earlier than those of limb muscles (Sung *et al*[1]). Such lower motor neuron loss is, in principle, accompanied by muscle pathology, which may be readily assessed by EMG methods, of which there are several. Although there is no doubt that expertise may be accumulated with any of the various methods, data comparing different techniques are only beginning to appear. Gilad *et al* (this issue, pp 596–99) report on results obtained by concentric needle EMG (CNEMG) single fibre EMG (SFEMG), and interference pattern (IP) analysis in a small group of patients with MSA.[2] Their findings both confirm and contradict the commonly held views on sphincter EMG findings in MSA. As expected, some EMG abnormalities were found in all patients studied, but the reported changes in isolation (reduced