attributed to the excitation of a predominately crossed occipitopretectal tract. Cogan stated that "removal of the pupillo-constrictor zone in one occiput of the cat results in anisocoria with the larger pupil on the opposite side." This finding presumably explains the unilateral pupillary dilatation reported here as a negative ictal phenomenon.

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Giall cytoplasmic inclusions are not exclusive to multiple system atrophy

In 1989 Papp et al reported finding argyrophilic inclusions in the cytoplasm of oligodendrocytes in cases of multiple system atrophy, and their presence in the sporadic form of this condition has since been confirmed. One feature of giall cytoplasmic inclusions as a diagnostic hallmark of multiple system atrophy has been emphasised by one of the authors as well as by others.7 At the UK Parkinson’s Disease Society Brain Bank in London, tissue is donated by patients with principally movement disorders. Giall cytoplasmic inclusions occurred in all brains from patients with multiple system atrophy (total 56); however, in three of seven cases with a pathological diagnosis of corticobasal degeneration and two of 18 cases with Steele-Richardson-Olszewski syndrome similar intracytoplasmic oligodendrocyte inclusions were identified. These were filamental argyrophilic structures (figure) immunoreactive with tau and ubiquitin antisemum. In corticobasal degeneration they were most numerous in white matter underlying the affected cortex, in the corpus callosum, internal capsule, and in one case, the basis pedunculi; occasional similar inclusions were also identified in the affected cerebral cortex and in the brain stem as well as in cerebellar hemispheric white matter, in the absence of any neuronal abnormalities. In the cases of Steele-Richardson-Olszewski syndrome inclusions were most prominent in the cerebellar white matter. We have not counted or mapped the distribution of glial inclusions in our cases, but have the impression that they are less numerous than in multiple system atrophy.

These findings have important implications for histological diagnosis and our understanding of disease pathogenesis. There is increasing awareness of overlap between many neurodegenerative conditions, in particular those associated with parkinsonism; thus the Lewy body, Pick body, neurofibrillary tangle, or the giall cytoplasmic inclusion are not exclusive to any of the conditions in which they abound. One explanation may be that neurons and glia have a limited repertoire of responses to a variety of different stimuli, resulting in morphological similarities between very distinct neurodegenerative diseases. Alternatively, shared pathogenetic pathways may underlie the cytoskeletal abnormalities seen in these conditions, the exact pattern of pathology being dictated by host factors such as age of exposure and genotype.

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