

ing the highest incidence of intellectual impairment (69.9%) used psychological assessment techniques, whereas studies identifying the lowest prevalence of dementia (30.2%) depended on non-standardized clinical examinations. The same year (1988), Mayeux *et al* [6] in a retrospective study of records, using the DSM-III-R, found a poor 10.9%, Girotti *et al* [7] found a little more, 14.28%. Hietanen and Teravainen [8] found that age at onset was quite an important factor as only 2% of patients with onset under 60 years were demented, in contrast to a 25% of patients with onset over 60 years.

At the beginning of the 90s Mayeux *et al* [9] reconsidered their results, and they also found a striking relation with age: 0% prevalence of dementia before 50 years and 69% in patients above age 80 years (which gives a sum prevalence of 41%) [10]. But, at this time this result was an exception. In 1995, Marder *et al* [11] found a less than two-fold risk (1.7) for PD patients to develop dementia compared to controls. In other studies of this period as well, prevalence numbers remained quite low, though rising: 18% for Pillon *et al* [12], 17.6% for Tison *et al* [13], 27.7% for Aarsland *et al* [14]. Again, Reid *et al* in 1996 [15], compared with age and made a follow-up 5 years later: they found an initial prevalence of 9% under 70 years, that was 17% at the 5-year follow-up, and 37% in patients older than 70 years, that increased to 62% after 5 years.

But, in 1999, Hobson and Meara [16] used the CAMCOG to assess intellectual impairment and found a 41% prevalence of dementia in PD patients. The less than two-fold relative risk of Marder *et al* in 1995 [11], increased to a six-fold risk (5.9) in 2001 by Aarsland *et al* [17]. And, in 2003, prevalence and incidence were found to be above 75% [18] (See Table 1).

Evolution of ideas about cognitive dysfunction and dementia in PD

Cognitive deficits

Mindham judiciously called the history of dementia in PD a methodological saga [4]. Brown and Marsden, in 1984, had claimed that the reason for inflated numbers of dementia in PD were either errors in separating idiopathic Parkinson's disease from other causes of the akinetic-rigid syndrome, or errors in differentiating dementia from confusional states, depression and even ageing, or in defining and assessing dementia itself [3].

Indeed, the first reports of deterioration in intellect in PD patients appeared not long after the disease was first described [4]. However, there seemed to be a large consensus that PD patients performed significantly poorer than controls in all tests but those for language, praxis and gnosis (the "instrumental" functions), frequently showing retrieval deficits, cognitive slowing, impaired abstract thinking, and reasoning difficulties [4]. These cognitive symptoms are generally subtle and do not interfere significantly with everyday activities. However, patients and their families usually cite forgetfulness or decreased ability to follow conversations involving several persons, difficulties that are regularly attributed to a depressive state that may coexist with the disease.

If one uses appropriate neuropsychological tests, it appears that these deficits: a) are frequent, affecting up to 93% of patients according to the study by Pirozzolo *et al* [19]; b) they mainly affect visuospatial functioning, memory, and executive functions; and, c) they are observed even at the early stages of the disease, strongly suggesting that they are related to the subcortical pathology of the disease [20].

These selective cognitive deficits are, both phenomenologically and etiologically, somehow related to the motor syndrome or to impaired sensory-motor interaction [21].

Table 1: Evolution of numbers of dementia in Parkinson's disease

Authors	Year	Frequency of dementia	Criteria – Comments
Mayeux <i>et al</i>	1988	10.9%	Retrospective, DSM-III
Girotti <i>et al</i>	1988	14.28%	Examination, Npsy
Hietanen, Terävaäinen	1988	2% < 60 yrs, 25% > 60 yrs	Examination, DSM-III
Pillon <i>et al</i>	1991	18%	Examination, NPsy 2SD
Mayeux <i>et al</i>	1992	41%, 0% < 50 yrs, 61% > 80 yrs	Examination, DSM-III
Tison <i>et al</i>	1995	17.6%	Examination, DSM-III-R
Aarsland <i>et al</i>	1996	27.7%	Examination, DSM-III-R
Reid <i>et al</i>	1996	9% < 70 yrs → 17% 37% > 70 yrs → 62%	Follow-up 5 ys
Hobson, Meara	1999	41%	Examination, CAMCOG
Marder <i>et al</i>	1995	1.7 Relative Risk	Follow up-controls
Aarsland <i>et al</i>	2001	5.9 relative risk	Follow up 4.2 ys-controls
Aarsland <i>et al</i>	2003	78% incidence in 8 years	Follow up 4 – 8 yrs