metabolites, and receptors in the striatum and medial frontal cortex. These changes have been related to cognitive disorders and depression in patients with both these disorders.

**Cholinergic system**

Repeatedly reported shrinkage and depletion of cholinergic neurons in the magnocellular part of the basal nucleus of Meynert are accompanied by decreased choline acetyltransferase activity in the neocortex by 86% to 91%. In Parkinson’s disease, cell loss averages 30% to 40% without correlation with age or duration of illness, and is much higher in demented parkinsonian patients in whom it approaches the values in Alzheimer’s disease (50% to 70%) than in non-demented patients (0% to 40%) who show neuronal losses only slightly higher than normal aged controls. Even more severe depletion of the basal nucleus of Meynert with 75% to 80% loss or large cholinergic neurons has been found in Lewy body dementia which correlates well with recent biochemical data. The heterogeneity of degeneration of cholinergic neurons in the basal forebrain and the variability in nucleus basalis cell depletion and loss of cholinergic markers in the neocortex and hippocampus are partly explained by cortical Lewy body or Alzheimer type pathology, suggesting a primary degenerative process of the cholinergic forebrain system in Parkinson’s disease, while secondary retrograde degeneration proposed for Alzheimer’s disease has been confirmed by defective retrograde transport of nerve growth factor to the basal nucleus in the brains of patients with Alzheimer’s disease.

In conclusion, there are still some conflicting data on the neurochemical and pathological basis of behavioural changes in dementia disorders, the elucidation of which will be a major task for modern neurosciences.

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Esiri, in her editorial about behavioural disturbances in dementia makes a common, and I believe important error in her classification scheme of behavioural abnormalities. She lumpa apathy, withdrawal, and listlessness with tearfulness and “other manifestations of unhappiness” as part of “depressive behaviour disturbances”. Actually her depressive category can be further subdivided into two distinct categories: (1) behaviour characterised by diminished activities and interactions often accompanied by slowness. Apathy, increased inertia, and abulia are terms often used to describe diminished spontaneous behaviour, long latency in responding to queries and requests for action, and difficulty persevering with tasks. Many apathetic, abulic patients are not sad or unhappy or discouraged. Many have no associated mood abnormalities and most have little insight into their apathy. Lesions of the caudate nuclei, medial thalamus, rostral brainstem tegumentum, and frontal lobes can cause such inert states. (2) Mood abnormalities that include sadness, crying, discouragement, depression etc.

True enough, many depressed patients have diminished activities but it is a great mistake to attribute all apathy, decreased activity, and inertia to depression. Apathy and diminished activities are a common presentation of caudate and thalamic infarcts, frontal lobe tumours, and progressive supranuclear palsy. The term “psychomotor retardation” is often used by psychiatrists to indicate depression, but the term normalised apathy and depression, should be thought of as different phenomena that are often but not necessarily related.

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Esiri replies to Jellinger and Caplan:

I am grateful to Jellinger for adding supplementary information to that presented in my editorial. I was not necessary to select in such a confined account of a complicated subject and largely limited my comments to findings that related to behavioural disturbances in dementia. I would emphasise the importance of my colleagues and I attach to studying behavioural change prospectively and systematically. It is not clear that this was done in the study by noradrenalin by Zulbenko et al that Jellinger refers to. The very small number of patients that are studied may also lead to unintended bias—for example, when therapies have not been completely documented or are not fully taken into account as possible factors influencing neurochemical findings. The studies of the cholinergic system referred to by Jellinger, while of interest, have not specifically examined the relevance of cholinergic changes for behaviour in dementia, an area that certainly deserves attention.

The comments that Caplan makes about the desirability of subdividing my depressive category of behaviour into apathy and depressed mood, as reflected in evident unhappiness or crying, are well taken. I did not intend to indicate broad categories of behavioural change but agree that it is best to avoid assumptions about which individual types of behaviour go together, particularly in demented subjects who are often unable to give a direct account of subjective feelings. In searching for neurochemical correlates of behavioural change in dementia we have tried to avoid making assumptions about which types of behaviour are related, although analysis of detailed, prospectively acquired, data suggest that there are some constellations of symptoms that cluster together (T Hope, unpublished data).

I would agree that there are some conflicting data on the neurochemistry of behaviour change in dementia. In such a complex area of investigation it would be surprising if there were not. The important point is that prospective studies are being undertaken that are likely to resolve the differences and uncover new findings that have a direct bearing on the optimal way to manage the difficult behavioural problems that people with dementia suffer from.

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