Journal of Neurology, Neurosurgery, and Psychiatry 1997;**62**:436–446

Olfactory dysfunction in Parkinson's disease

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Abstract

***Objective*—To evaluate olfactory function in Parkinson's disease.**

Methods—A standardised odour identification test was used, together with an evoked potential assessment with hydrogen sulphide. In addition, histological analysis was performed on the olfactory bulbs of cadavers who died from Parkinson's disease.

***Results*—Over 70% of patients studied (71 of 96) were outside the 95% limit of normal on the identification test in an age matched sample and there was an unusual pattern of selective loss to certain odours, not hitherto described. The evoked potentials were significantly delayed but of comparable amplitude to a control matched population. Of the 73 patients studied only 37 had a technically satisfactory record containing a clear response to both gases and of these, 12 were delayed. For H_2S there was more delay on stimulating the right nostril than the left. Some patients with normal smell identification test scores had delayed evoked potentials. In the pathological examination of olfactory bulbs from eight brains, changes characteristic of Parkinson's disease (Lewy bodies) were seen in every olfactory bulb, particularly in the anterior olfactory nucleus, and were sufficiently distinct to allow a presumptive diagnosis of Parkinson's disease.**

***Conclusions*—Olfactory damage in Parkinson's disease is consistent and severe and may provide an important clue to the aetiology of the disease.**

(*J Neurol Neurosurg Psychiatry* 1997;**62**:436–446)

applied to patients with Parkinson's disease. Such techniques were pioneered by Kobal and Plattig[2] and adopted by us.[3]

The basal ganglia have been the subject of intense pathological study in Parkinson's disease, but the rhinencephalon has not been investigated systematically. Chui *et al*[4] examined four patients with Parkinson's disease with dementia and in one brain found an Alzheimer type change in the amygdala, adjacent anterior temporal cortex, and CA2 sector of the hippocampus. The hippocampus was normal in the remaining three cases. It is uncertain whether all the central olfactory areas were examined. Furthermore, the cases were complicated by the presence of dementia.

We have undertaken a multidisciplinary study of olfactory identification, olfactory evoked potential, and pathological examination of the olfactory bulb in Parkinson's disease.

Methods

After local ethics committee approval and informed consent of patients and controls we undertook the procedures described below.

SMELL IDENTIFICATION TEST

These measurements were carried out using the University of Pennsylvania smell identification test (UPSIT).[5] This test uses strips of paper impregnated with microencapsulated odours which are released on scratching the strip with a pencil. There are 40 different odours and a forced choice is made from four possible answers.

Controls

Normative UPSIT data are available for Americans but because some odours are unfa-