How do we assign social position to women? Traditionally, the woman's occupation, her husband's occupation (single women being classified by their own occupation), or the household based "dominance" method are used. The last compares the two spouses' occupations and assigns the higher of these to the woman as well as to the man. In Sweden, use of the household dominance method showed greater social differences among women than use of the woman's own occupation, both for cardiovascular disease and for total mortality.[8] British data on self assessed health (but not on longstanding illness) gave the same results; the household based measures of social position showed greater social differences than methods based on individual criteria.[5]

In the paper by Sacker and colleagues, greater social differences among women were found with the Cambridge scale of occupations than with the ONS classification. Was this because the Cambridge scale used a household based method or was it because the principles behind this scale are more suitable for describing the general standing of women in society than those of the ONS classification? It seems unfair to compare the ONS scheme, which here is based on the woman's own occupation, with Cambridge scores based on the highest occupation in the household.

Koskinen and Martelin's study of socioeconomic mortality differences suggested that the smaller differences among women arose entirely from the subpopulation of married women; for single, divorced, or widowed women the differences in mortality were of the same size as in men.[9] Koskinen and Martelin also showed that for specific causes of death the socioeconomic differences in mortality among women were not smaller than those in men. Looking at specific causes of death using indicators of social position based on household criteria could find socioeconomic differences in mortality among women to be as large as or even larger than in men. For a major cause of death such as cardiovascular disease there are already indications that this is the case.[8 9]

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Depression in Parkinson's disease

Must be properly diagnosed and treated to avoid serious morbidity

Psychiatric symptoms frequently coexist with idiopathic Parkinson's disease and are often underdiagnosed and poorly treated.[1] Depression and anxiety are the most common psychiatric conditions that accompany Parkinson's disease. A study by Menza et al found that 12 out of 42 patients with Parkinson's disease met the criteria for an anxiety disorder according to the *Diagnostic and Statistical Manual* and 11 of them had a comorbid depressive disorder.[2] Recent reviews show that depression is a common and potentially debilitating aspect of Parkinson's disease, affecting 40-50% of patients.[3–5] While its aetiology in Parkinson's disease is unclear (biochemical changes, psychosocial factors, and situational stressors have all been implicated), it has an adverse effect on the quality of patients' lives, and doctors should ensure that it is diagnosed and properly treated.

The diagnosis is not easy because clinical symptoms of depression can overlap with or be mistaken for those of Parkinson's disease (such as the flat affect, inability to work, fatigue, preoccupation with ill health, loss of desire, and reduction in libido. Moreover, depression in patients with Parkinson's disease is qualitatively different from primary major depression in that self blame, guilt, delusions, a sense of failure, self destructive thoughts, and suicide are less frequent.[7]

Several studies have failed to find a clear association between the severity of depression and motor disability. Depressive symptoms precede those of motor dysfunction in 12-37% of patients with Parkinson's disease.[7] The severity of depression contributes to the cognitive disorders in Parkinson's disease; in a prospective cohort study of patients with Parkinson's disease who did not have dementia, depression was associated with a significantly increased risk of developing dementia.[9]

Depression in Parkinson's disease is usually linked to a reduction in brain catecholamines, serotonin (a decrease in the concentration of 5-hydroxyindoleacetic acid in cerebrospinal fluid), or dopamine (postmortem studies show dopamine depletion in the ventral tegmental area; glucose positron emission tom-

BMJ 2000;320:1287–8

ography shows hypometabolism in the orbital and prefrontal cortices). Paradoxically, levodopa and dopamine agonists (except selegiline at high doses, 30-40 mg/day) do not consistently alleviate depressive symptoms. In patients with fluctuating motor symptoms depression occurs when motor function is poor; more puzzling, deep brain stimulation, notably of subthalamic nuclei, can induce a delayed depression, although it improves motor function.[10]

Once depression is diagnosed, treatment is complicated by the drugs the patient is already taking. Due to the lack of systematic clinical trials there are still three main questions concerning the prescribing of an antidepressant.[3 4] The first is whether the antidepressant drug can increase or induce parkinsonian symptoms—tricyclic antidepressants such as desipramine, nortriptyline, and imipramine can improve motor symptoms, but selective serotonin reuptake inhibitors are repeatedly reported in case reports as potential inducers of parkinsonism. Fluoxetine is the only one to have been studied in this way, but a retrospective chart review by Caley and Friedman did not find that fluoxetine caused parkinsonian symptoms.[5] There are no data on the more recently launched antidepressants such as venlafaxine (a serotonin noradrenaline recapture inhibitor) and mirtazapine (a noradrenaline serotonin specific antidepressant).

The second question is the safety of antidepressant drugs in patients with Parkinson's disease. Tricyclic antidepressants can cause delusions, cognitive disorders (due to their anticholinergic effect), or orthostatic hypotension (they block adrenergic alpha receptors). The third question concerns interactions between antidepressant and antiparkinson drugs. Only one drug combination seems to be risky for patients: selective serotonin reuptake inhibitors (such as fluoxetine and fluvoxamine) and selegiline are associated with the potential and rare (the incidence is 0.24%) serotonin syndrome.[12] The diagnosis of serotonin syndrome is made on the basis of three of the following symptoms: a change in mental status (such as the onset of delusions, change in level of consciousness), myoclonus, sweating, hyperreflexia, tremor, diarrhoea, shivering, uncoordination, and fever. This syndrome can be fatal.

The depression associated with Parkinson's disease must be treated. The first choice is selective serotonin reuptake inhibitors (sertraline 50-200 mg/day; paroxetine 20-40 mg/day) or, in some countries and on an empirical basis, tianeptine (12.5 mg three times a day), which increases the presynaptic recapture of 5-hydroxy-indoleacetic acid, or moclobemide (300 mg/day), which is a reversible and selective inhibitor of monoamine-oxidase type. Adverse drug interactions are rare, except when selegiline is given at more than 5 mg twice daily. Clinical trials are needed not only to determine the risk-benefit ratio of these drug regimens but also to determine the optimum dose and duration of antidepressant therapy in Parkinson's disease.

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The health hazards of mobile phones

The only established risk is of using one while driving

Despite repeated horror stories about mobile phones in the media, nearly half of the British public now owns one. Some 500 million people worldwide use mobile phones. Clearly, they have decided that the benefits outweigh any risks to their health. The benefits to the Exchequer in the United Kingdom are also substantial—£22bn ($13.75bn) from the recent round of bids for new licences. In this context, the publication of the *Report of the Independent Expert Group on Mobile Phones*, a group organised by the Department of Health, could have political implications.

Mobile phones are low power radio devices that transmit and receive radio frequency radiation (at frequencies in the microwave range of 900-1800 MHz) through an antenna used close to the user's head. Digital systems have recently replaced analogue. There is concern that microwaves might induce or promote cancer, and the symptoms associated with their use include sleep disturbance, memory problems, headaches, nausea, and dizziness.[1] Changes in the permeability of the blood-brain barrier, electroencephalographic activity, and blood pressure have

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