radical protecting capacity. It is possible that this loss is due to increased oxygen stress induced by the levodopa therapy. This would be in accordance with the impression that combination therapies with dopamine agonists and thereby reduced amounts of levodopa may postpone the development of fluctuations in therapeutic response and of the "on-off syndrome".²⁵

This study was supported by a grant from "Fonden af 02.07.1984 Til Bekæmpelse af Parkinson's Syge".

- 1 Langston JW. Current theories on the cause of Parkinson's. *J Neurol Neurosurg Psychiatry*. Suppl 1989;13-17.
- 2 Cadet JL. The potential use of vitamin E and selenium in parkinsonism. *Medical Hypotheses* 1986;20:87-94.
- 3 Barbeau A. Etiology of Parkinson's disease: a research strategy. *Can J Neurol Sci* 1984;11:24-8.
- 4 Cohen C. The pathobiology of Parkinson's disease: biochemical aspects of dopamine neuron senescence. *J Neurol Transm Suppl* 1983;19:89-103.
- 5 Cohen G, Heikkilä R. The generation of hydrogen peroxide, superoxide radical, and hydroxyl radical by 6-hydroxydopamine, dialuric acid and related cytotoxic agents. *J Biol Chem* 1974;249:2447-52.
- 6 Graham DG, Tiffany SM, Bell WR, Gutknecht WF. Autooxidation versus covalent binding of quinones as the mechanism of toxicity of dopamine, 6-hydroxydopamine, and related compounds towards C1300 neuroblastoma cells in vitro. *Molecular Pharmacology* 1978;14:644-53.
- 7 Parson PG. Modification of dopa toxicity in human tumor cells. *Biochemical Pharmacology* 1985;34:1801-7.
- 8 Flohe L, Gunzler WA, Schook HH. Glutathione peroxidase: a selenoenzyme. *Febs Letters* 1973;32:132-4.
- 9 Carmagnol F, Sinet PM, Jerome H. Selenium-dependent and non-selenium-dependent glutathione peroxidases in human tissue extracts. *Biochem Biophys Acta* 1983;759:49-57.
- 10 Perry TL, Godin DV, Hansen S. Parkinson's disease: A disorder due to nigral glutathione deficiency? *Neuroscience Letters* 1982;33:305-10.
- 11 Ambani LM, Van Woert MH, Merphy S. Brain peroxidase and catalase in Parkinson's disease. *Arch Neurol* 1975;32:114-8.
- 12 Kish SJ, Morito C, Hornykiewicz D. Glutathione peroxidase activity in Parkinson's disease brain. *Neurosci Lett* 1985;58:343-6.
- 13 Poirier J, Barbeau A. Erythrocyte antioxidant activity in human patients with Parkinson's disease. *Neurosci Lett* 1987;75:345-8.
- 14 Kilic A, Yalcin AS, Yalcin D, Taga Y, Emerk K. Increased erythrocyte susceptibility to lipid peroxidation in human Parkinson's disease. *Neuroscience Letters* 1988;87:307-10.
- 15 Lewitt P, Chase TN. On-off effects the new challenge in parkinsonism. *Trends in Neurosciences* 1983;6:1-3.
- 16 Lees AJ. The on-off phenomenon. *J Neurol Neurosurg Psychiatry*. Suppl 1989:29-37.
- 17 Fahn S, Elton RL, and members of the UPDRS Development Committee. Unified Parkinson's disease Rating Scale. In: Fahn S, Marsden CD, Calne DB, Goldstein M, eds. *Recent developments in Parkinson's disease*, vol 2. Florahne Park, NJ: Macmillian Health Care Information, 1987:153-64.
- 18 Paglia DE, Valentine WN. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J Lab Clin Med* 1967;70:158-69.
- 19 Watkinson IH. Fluorometric determination of selenium in biological material with 2,3 diamino-naphtalene. *Anal Chem* 1966;38:92-7.
- 20 Thorling EB, Overvad K, Heerfordt A, Foldspang A. Serum selenium in Danish blood bank donors. *Biol Trace Element Res* 1985;8:65-73.
- 21 Neve J, Vertogen F, Capel P. Selenium supplementation in healthy Belgian adults: response in platelet glutathione peroxidase activity and other blood indices. *Am J Clin Nutr* 1988;48:139-43.
- 22 Thomson CD, Steven SM, van-Rij AM, Wade CR, Robinson MF. Selenium and vitamin E supplementation: activities of glutathione peroxidase in human tissues. *Am J Clin Nutr* 1988;48:316-23.
- 23 Marttila RJ, Lorentz H, Rinne UK. Oxygen toxicity protecting enzymes in Parkinson's disease. Increase of superoxide dismutase-like activity in the substantia nigra. *Neurol Sci* 1988;86:321-31.
- 24 Marttila RJ, Rinne UK. Oxygen protecting enzymes in aging and Parkinson's disease. In: Calne DB, et al, eds. *Parkinsonism and aging*, New York: Raven Press, 1989: 89-95.
- 25 Rinne UK. Early dopamine agonist therapy in Parkinson's disease. *Movements Disorders* (suppl 1), 1989;4:86-94.

Neurological stamp

Russia

Avicenna or Ibn Sina (980-1036 AD)

He was court physician, vizier to different caliphs and physician in chief to the celebrated hospital in Baghdad, and translator of Galen. Avicenna wrote a gigantic medical tome, and had a great reputation in his time. He gave us the term "vermis" and "tailed nucleus" from which was derived "caudate nucleus".

He described meningitis which he considered to be inflammation or tumour of the envelopes of the brain, and distinguished this from secondary meningism. He knew about the pupil and its movement, of six motor muscles for the globe, and of central and peripheral types of facial weakness. Avicenna defined apoplexy as "loss of sensibility and movement following an occlusion seated within the brain in those places traversed by the nervous influx of sensibility and motricity."

This Russian stamp was issued in 1980 to commemorate one thousand years of his birth. (Stanley Gibbons 5022, Scott 4852).

LF HAS