from an overactivity of the dopaminergic pathway. Antidepressants such as amitriptyline and perphenazine may alleviate levodopa-induced depression (Van Wieringen, 1972; Van Wieringen and Wright, 1972). There are no apparent interactions between levodopa and chlordiazepoxide, oxazepam, or diazepam in parkinsonian patients.

9. Conclusion

The importance of individualising therapy in Parkinson’s disease has been well established (Bianchine and Sunyapridakul, 1974). The large number of variables which affect final dopamine concentration at receptor sites in the striatum necessitate a personalised therapeutic regimen for each patient. Patients who are initially unresponsive to levodopa treatment may often be improved merely by empirical adjustment of dosage size and/or scheduling. If such manoeuvres fail to achieve desired therapeutic responses, estimations of plasma levodopa may be required. Reports by Tolosa et al. (1973), Marsden and Parkes (1976), and Rivera-Calimlim et al. (1970b) indicate that important information can be obtained from such studies. In these reported cases, patients who previously were not well controlled by levodopa therapy, showed marked improvement following treatment modifications resulting from evaluation of levodopa plasma tolerance curves. These are but three instances where suboptimal clinical responses to levodopa may be elucidated by pharmacokinetic investigation.

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