proximately 10,000 anesthesiologists, this represents an incidence of about one in 588. It is obviously impractical to examine 588 practicing anesthesiologists for their immunologic reactivity, so this study does not preclude the possibility that a rare individual may suffer suppression of his immune response as a result of chronic inhalation of traces of anesthetic. Even in patients deliberately immunosuppressed following organ transplants, the estimated incidence of cancer is 6 to 8 per cent.[9] It is doubtful, therefore, that any correlation between immunosuppression and propensity to lymphoid malignancy can be found by practicable studies of this sort.

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References

1. Bruce DL, Eide KA, Linde HW, et al: Causes of death among anesthesiologists: A 20-year survey. ANESTHESIOLOGY 29:565–569, 1968
2. Hersh EM, Oppenheim JJ: Impaired *in vitro* lymphocyte transformation in Hodgkin's disease. New Engl J Med 273:1006–1012, 1965
3. Oppenheim JJ, Whang J, Frei E III: Immunologic and cytogenetic studies of chronic lymphocytic leukemic cells. Blood 26:121–132, 1965
4. Bruce DL, Wingard DW: Anesthesia and the immune response. ANESTHESIOLOGY 34:271–282, 1971
5. Linde HW, Bruce DL: Occupational exposure of anesthetists to halothane, nitrous oxide and radiation. ANESTHESIOLOGY 30:363–368, 1969
6. Bruce DL: Halothane inhibition of phytohemagglutinin-induced lymphocyte transformation. ANESTHESIOLOGY 36:201–205, 1972
7. Hersh EM, Oppenheim JJ: Inhibition of *in vitro* lymphocyte transformation during chemotherapy in man. Cancer Res 27:98–105, 1967
8. Pachman LM, Esterly NB, Peterson RDA: The effect of salicylate on the metabolism of normal and stimulated human lymphocytes *in vitro*. J Clin Invest 50:226–230, 1971
9. Hume DW: Organ transplants and immunity, Immunobiology. Edited by RA Good, DW Fisher. Stamford, Conn., Sinauer Associates, Inc., 1971, p 193

Drugs and Their Actions

ENDOCRINE FUNCTION AFTER L-DOPA IN PARKINSONISM Nine patients with Parkinson's disease were studied before and during therapy with L-dopa, 6 g/day. The drug caused no significant change in: 1) thyroid function, as indicated by serum thyroxine levels and uptake of T_3 resin or radioiodine; 2) adrenal function, as indicated by 24-hour urinary excretion of 17-hydroxycorticosteroids and 17-ketosteroids; 3) pituitary ACTH function, as indicated by increased steroid secretion in response to metapyrone orally (750 mg q 4th × 6); or 4) urinary excretion of gonadotropins. These last findings in man are at variance with those found in animals by others, who were able to demonstrate dopamine effects on gonadotropins. Plasma growth hormone levels were elevated following L-dopa; this response was not altered by glucose given either orally or intravenously. Curiously, L-dopa modified the tolerance to glucose administered orally (but not iv), *i.e.*, the plasma glucose levels, which were unchanged in the first 120 minutes, and plasma insulin response, which was markedly diminished during this time, were both significantly elevated three, four, and five hours after orally-administered glucose. In contrast, after glucose iv, L-dopa did not significantly change glucose decay or plasma insulin levels, nor did tolbutamide influence this lack of response. Data from the control studies suggest that impaired growth-hormone release in response to insulin-induced hypoglycemia and abnormal tolerance to intravenous glucose may be present in Parkinson's disease. (*Boyd, A. E., III, Lebovitz, H. E., and Feldman, J. M.: Endocrine Function and Glucose Metabolism in Patients with Parkinson's Disease and Their Alteration by L-Dopa, J. Clin. Endocrinol. Metab. 33: 829–837, 1971.*)