

100 per cent  $O_2$  to patients for at least five minutes before discontinuing  $N_2O-O_2$  anesthesia. The results of this study should not be interpreted as making this practice unnecessary or undesirable. Although "diffusion anoxia" does not exist as a clinical entity, hypoxia may occur in the immediate postanesthetic period, either because of respiratory irregularity or because of ventilation-perfusion changes which occur as part of the aging process or for other reasons and which result in low "control"  $SA_{O_2}$  values. In either instance, the breathing of high concentrations of  $O_2$  will eliminate or reduce considerably any hypoxemia which would result from such causes.

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### References

1. Fink, B. R.: Diffusion anoxia, *ANESTHESIOLOGY* 16: 511, 1955.
2. Rackow, H., Salanitre, E., and Frumin, M.: Dilution of alveolar gases during nitrogen oxide excretion in man, *J. Appl. Physiol.* 16: 723, 1961.
3. Severinghaus, J. W.: Blood gas calculator, *Appl. Physiol.* 21: 1108, 1966.
4. Cotes, J. E.: Lung Function. Philadelphia: F. A. Davis, 1965, pp. 423-424.
5. Nunn, J. F.: Influence of age and other factors on hypoxemia in the postoperative period, *Lancet* 2: 466, 1965.

### Drugs

**ISOPROTERENOL** The effects of isoproterenol infusion (.005 to .006 mg per minute) were studied in anesthetized dogs. Blood pressure decreased from 150 to 100 mm Hg. Vertebral artery flow increased from 50 to 150 ml per minute. Left circumflex coronary artery flow increased from 50 to 75 ml per minute. Aortic blood flow increased from 4.0 to 4.8 l/min. Vascular resistance decreased 58 per cent in the left coronary artery, 70 per cent in the vertebral artery, and total resistance decreased 45 per cent. Cardiac work increased 27 per cent; cardiac output increased 58 per cent; and coronary perfusion increased 94 per cent. The changes are considered to be due to beta-adrenergic receptor stimulation and vasodilatation. Similar results were obtained when animals were given norepinephrine and phenoxybenzamine simultaneously. (Dedichen, H., and Schenk, W. G.: *Hemodynamic Effects of Isoproterenol Infusion*, *Arch. Surg.* 97: 934 (Dec.) 1968.)

**PHYSOSTIGMINE** Physostigmine salicylate in doses of one to two mg administered parenterally was found to be an effective antidote to intoxication with centrally-active anticholinergic agents. Confusion, agitation, hallucinations, stupor, ataxia, dysarthria, and other symptoms were reversed promptly in 26 consecutive patients in whom toxic reactions developed after they received scopolamine, atropine or drugs for Parkinson's disease. Physostigmine deserves a place in therapeutics as an antidote to anticholinergic intoxication. (Duvoisin, R. C., and Katz, R.: *Reversal of Central Anticholinergic Syndrome in Man by Physostigmine*, *J.A.M.A.* 206: 1963 (Nov.) 1968.)

**IMIPRAMINE INTOXICATION** A 2½-year-old boy died as a result of cardiovascular complications of imipramine hydrochloride overdosage. Through a direct toxic effect on the myocardium, imipramine lowers myocardial contractility and cardiac output, with resultant hypotension. Cardiac arrhythmias are also a constant feature of imipramine toxicity. Since there is no specific antidote to imipramine, treatment of overdosage with this drug must be directed at its life-threatening circulatory and respiratory manifestations if the patient is to survive. (Sacks, M. H., and others: *Cardiovascular Complications of Imipramine Intoxication*, *J.A.M.A.* 205: 588 (Aug.) 1968.)