from the temperature dependence of the narrow line (15 mg) and the independence of the broad line (50 mg). Were exchange occurring between the liquid and the clathrate, the narrow line would diminish in amplitude relative to the broad line rather than broaden to the width characteristic of a solid as was seen in this experiment.

Conclusion

The NMR evidence of the molecular freedom of halothane within the mixed hydrate of halothane and H_2S suggests a clathrate structure, a finding in agreement with previous results.[1, 3]

This study also suggests a technique to establish the existence of clathrate structures *in vivo*, provided sufficient sensitivity may be obtained. The existence of such structures is essential to the theory put forward by Pauling.[2]

References

1. Cornell BA, Troup GJF: A mass spectrographic investigation of the hydrate of H_2S and halothane. ANESTHESIOLOGY 34:183–184, 1971
2. Pauling L: A molecular theory of general anaesthesia. Science 134:15, 1961
3. van der Heem P: Preparation of a solid hydrate of halothane. ANESTHESIOLOGY 27:85, 1966
4. Abraham A: The Principles of Nuclear Magnetism. Oxford, Clarendon Press, 1961
5. Pople JA, Schneider WG, Bernstein HJ: High Resolution Nuclear Magnetic Resonance Spectroscopy. New York, McGraw-Hill, 1959

Drugs

HALOTHANE HEPATITIS Stimulation of lymphocytes, as measured by incorporation of ^{3}H-thymidine into their desoxyribonucleic acid, was observed in the presence of halothane in ten of 15 patients with halothane hepatitis, but not in healthy controls, patients with hepatic disease, or patients exposed to halothane who did not have hepatic damage. Lymphocytes of a patient with hepatic damage attributable to methoxyflurane were stimulated by methoxyflurane. Preliminary data indicate that sensitization is temporary. The plasma of the patients may contain a factor that inhibits lymphocytic stimulation. Australia antigen was not detected in the sera of the patients, but antimitochondrial antibodies seemed to correlate with lymphocytic stimulation. Stimulation of lymphocytes in the presence of halothane is helpful in the differential diagnosis of viral and halothane hepatitis, and indicates that in some patients the anesthetic may be a sensitizing agent with a pathogenetic role in the hepatic damage. (*Paronetto, F., and Pepper, H.: Lymphocyte Stimulation Induced by Halothane in Patients with Hepatitis Following Exposure to Halothane, New Eng. J. Med. 283: 277 (Aug.) 1970.*)

PROPRANOLOL AND PACEMAKER THRESHOLD The increasing use of implanted pacemakers and propranolol for the control of arrhythmias prompted a study of the effects of this drug on the stimulation threshold during electrical pacing. Five patients requiring pacing via transvenous electrodes were studied at various intervals following the intravenous administration of 1 mg, then 4 mg, and finally, 5 mg propranolol. A marked increase in energy threshold (product of mean voltage, mean current, and impulse duration required for appropriate pacing) followed administration of the drug, and the maximum did not appear to be reached within the 45-minute testing period. If propranolol is to be used in a patient with an implanted pacemaker, the initial doses should be small and the patient observed closely for loss of response to pacemaker stimulation. (*Kubler, W., and Sowton, E.: Influence of Beta-blockage on Myocardial Threshold in Patients with Pacemakers, Lancet 2: 67–68, 1970.*)