laxants by circulating halogen concentrations in susceptible patients.

REFERENCES

1. Hogg, S., and Renwick, W.: Hyperpyrexia during anaesthesia, Canad. Anaesth. Soc. J. 13: 429, 1966.
2. Cullen, W. G.: Malignant hyperpyrexia during general anaesthesia: A case report, Canad. Anaesth. Soc. J. 13: 444, 1966.
3. Davies, L. E., and Graves, H. B.: Hyperpyrexia and death associated with general anaesthesia, Canad. Anaesth. Soc. J. 13: 447, 1966.
4. Denborough, M. A., Forster, J. E., Lowell, R. R. H., Mapleston, P. A., and Villiers, J. D.: Anesthesia deaths in a family, Brit. J. Anaesth. 34: 394, 1962.
5. Purkis, I. E., Horrelt, O., de Young, C. C., Fleming, R. A. P., and Langley, G. R.: Hyperpyrexia during anaesthesia in a second member of a family, with associated coagulation defect due to increased intravascular coagulation, Canad. Anaesth. Soc. J. 14: 183, 1967.
6. Wilson, R. D., Dent, T. E., Traber, D. L., McCoy, N. R., and Allen, C. R.: Malignant hyperpyrexia with anesthesia, J.A.M.A. 202: 183, Oct. 1967.
7. Wilson, R. D., Nichols, R. J., Dent, T. E., and Allen, C. R.: Disturbances of oxidative phosphorylation mechanism as a possible etiological factor in sudden unexplained hyperthermia occurring during anesthesia, ANESTHESIOLOGY 27: 231, 1966.
8. Thut, W., and Davenport, H. T.: Hyperpyrexia associated with succinylcholine-induced muscle rigidity: A case report, Canad. Anaesth. Soc. J. 13: 425, 1966.
9. Lavoie, C.: Hyperpyrexia during general anaesthesia: A case report, Canad. Anaesth. Soc. J. 13: 444, 1966.
10. Relton, J. E. S., Creighton, R. E., Johston, A. E., Pelton, D. A., and Conn, A. W.: Hyperpyrexia in association with general anaesthesia in children, Canad. Anaesth. Soc. J. 13: 419, 1966.
11. Tammiston, T., and Airaksinen, M.: Increase of creatine kinase activity in serum as a sign of muscular injury caused by intermittently administered Suxamethonium during halothane anesthesia, Brit. J. Anaesth. 38: 510 1966.

Surgery

CARBON MONOXIDE Severe carbon monoxide poisoning can produce several types of lesions of the skin. The lesions vary in degree from areas of erythema and edema to marked blister and bulla formation. These lesions can easily be mistaken for burns or trauma. The bullous lesions heal by eschar formation. The scalp lesions of edema and erythema evolve into areas of alopecia. (*Long, P. I.: Dermal Changes Associated with Carbon Monoxide Intoxication, J.A.M.A. 205: 50 (July) 1968.*)

HEPATIC DISEASE Metabolic deficiencies in hepatic failure are reflected in hypoalbuminemia, clotting aberrations, vitamin depletion, inability to oxidase glucose and fat, intolerance to opiates and anesthetics, antidiuresis, sodium retention, abnormal immune responses, and abnormal metabolism of proteins, endocrines, bile pigments and ammonia. Prognosis for survival in cirrhotics cannot be predicted from available clinical and laboratory data. Most anesthetic drugs are protoplasmic poisons and either affect hepatic function or must be excreted by the liver. Narcotics, especially morphine, thiopental, and succinylcholine, should be avoided or used in minimal doses. Pre-existing hepatic disease increases the chances of postoperative hepatic complications more than 500-fold. Choice of anesthesia is minimal doses of thiopental and meperidine with nitrous oxide and tubocurarine or gallamine for muscle relaxation. (*Jackson, F. C., and others: Preoperative Management of Patients with Liver Disease, Surg. Clin. N. Amer. 48: 907 (Aug.) 1968.*)