

Literature Briefs

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Literature briefs were supplied by Drs. P. J. Cohen and J. H. Tinker. Briefs appearing elsewhere in this issue are part of this column.

Neuromuscular Blockers

RENAL DISEASE AND RECURARIZATION Renal excretion is important in the disposition of *d*-tubocurarine (*d*Tc), a finding supported by computer analysis and now by examination of three patients who became "recurarized" after *d*Tc had apparently been antagonized adequately. These patients all had end-stage renal disease, and ranged in age from 38 to 54 years. The operative procedures were either renal transplantation or nephrectomy. The doses of *d*Tc were 48–54 mg. Reversal was accomplished with 2.5–5.5 mg neostigmine; neuromuscular function was not analyzed with indirect electrical stimulation at the time of reversal. Reversal was judged adequate on clinical grounds. However, within 20–90 minutes after reversal, obvious signs of neuromuscular block developed; this was treated with controlled respiration and additional doses of neostigmine and atropine. The authors believe that a single administration of neostigmine may not suffice to provide permanent antagonism of the effects of *d*Tc in patients who have renal disease. The duration of action of neostigmine (approximately 60 minutes) is not altered by impaired renal function. Indeed, the authors point out that neostigmine is "eliminated rapidly and completely in animals with ligated renal pedicles." It is therefore not surprising that "re-curarization" should occur in these patients after administration of large doses of *d*Tc. The use of smaller doses of *d*Tc is suggested. In addition, since elimination of pyridostigmine is extremely slow in the absence of normal renal function, the authors suggest the use of this drug for reversal might be more rational. (Miller RD, Cullen DJ: Renal failure and postoperative respiratory failure: Recurarization? *Br J Anaesth* 48:253–256, 1976.)

Regional Analgesia

CAUDAL ANALGESIA SYMPATHETIC BLOCKADE The presence of high sympathetic block following caudal analgesia was assessed by examining for changes in pupillary size and the presence of ptosis in 20 consecutive patients receiving caudal analgesia during labor. The block was unsuccessful in one patient; in a second patient, observations were not pursued because of previous eye surgery. The drug used was either 0.5 per cent bupivacaine or 1.0 per cent lidocaine, with or without epinephrine. The doses injected ranged from 12 to 22 ml (average 17.4 ml). The upper levels of analgesia ranged from T3 to L1 (average T8). It is concluded that administration of 10 ml of local anesthetic for caudal analgesia may result in spread high enough (probably to T1) to cause myosis and/or ptosis. (Mohan J, Potter JM: Pupillary constriction and ptosis following caudal epidural analgesia. *Anaesthesia* 30:769–773, 1975.)

Blood-Gas Analysis

OXYGEN TENSION AND HALOTHANE The responses of a gold/silver–silver chloride microelectrode to known concentrations of oxygen were examined in the presence and absence of halothane. The addition of halothane produced an apparent increase in oxygen tension, which varied linearly with the concentration of halothane. It appeared that halothane was also reduced polarographically by the cathode in this electrode system. The error was produced by clinically relevant concentrations of halothane. The authors suggest that the measurement of oxygen tension with a gold/silver–silver chloride electrode during administration of halothane would be expected to be inaccurate. (Dent JG, Netter KJ: Errors in oxygen tension caused by halothane. *Br J Anaesth* 48:195–197, 1976.)