

### Summary

A method for the determination of mepivacaine hydrochloride after extraction with ethylene dichloride and removal of most of the interfering substances, is described. It can be used satisfactorily for the evaluation of concentrations of 1-9  $\mu\text{g./ml.}$  using one milliliter of blood or biological fluids; suitable dilutions can be made for higher concentrations. The method is not specific and can only be used if similar drugs are absent.

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### References

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2. Way, E. L., Sung, C. Y., and McKelway, W. P.: The absorption, distribution and excretion of *d, l* Methadone, *J. Pharmacol. Exp. Ther.* 97: 222, 1949.
3. Bromage, P. R., and Robson, J. G.: Concentration of lignocaine in the blood after intravenous, intramuscular, epidural and endotracheal administration, *Anaesthesia* 16: 461, 1961.

### Drugs

**GALLAMINE, CURARE AND HALOTHANE** The hemodynamic effects of gallamine or curare administered during halothane anesthesia were investigated before surgery. The administration of gallamine increased cardiac output, heart rate, arterial pressure, and left ventricular work; decreased total peripheral resistance and mean transit time; and produced a variable response in stroke volume. Curare decreased arterial pressure and total peripheral resistance, but produced variable changes in other areas. The action of gallamine can be attributed to a cardiovagal block, myocardial stimulation, and a slight sympathetic ganglionic block. The effects of curare arise from a peripheral ganglionic block, myocardial depression, and possible histamine release. The results do not suggest any significant change in the clinical use of either of these neuromuscular blocking agents with halothane. (Smith, N. T., and Whitcher, C. E.: *Hemodynamic Effects of Gallamine and Tubocurarine Administered during Halothane Anesthesia*, *J.A.M.A.* 199: 704 (March) 1967.)

**QUINIDINE RECURARIZATION** Drug interaction is commonly observed today because of the simultaneous use of many therapeutic agents. The neuromuscular blocking effects of the cinchona alkaloids, of which quinidine is one, are well documented. This blockade seems to be related to a curariform activity at the myoneural junction, as well as a depression of muscle action potential. The failure of neostigmine to antagonize the neuromuscular blockade produced by the combination of quinidine and tubocurarine may be related to a direct effect of quinidine on muscle action potential. A clinical situation was presented in which a nondepolarizing muscle relaxant was used successfully and its action terminated as judged by clinical observation and response to peripheral nerve stimulation. The administration of quinidine at this point resulted in apnea. It is reasonable to expect that this sequence of drug administration may occur again. With awareness of the possible outcome, the physician may prevent harm to his patient. (Way, W. L., and others: *Recurarization with Quinidine*, *J.A.M.A.* 200: 153 (April) 1967.)