

duced an additional decrease in sympathetic activity, which was followed also by a decline in arterial pressure. This last observation indicates that the vascular smooth muscle is reactive to sympathetic impulses under barbiturate anesthesia, and that sympathetic activation could have affected peripheral resistance, had it occurred.

The clinical implication of our findings is that an extraordinary degree of hypotension is to be expected in individuals relying on high sympathetic tone to maintain sufficient cardiovascular function (e.g., hypovolemic shock and heart failure), as suggested in 1943 by Halford.⁷ This should also be true for individuals with weak barostatic reflexes, as in orthostatic hypotension. Our findings fail to provide a basis for the warning of Marc-Aurele *et al.*,⁸ who cautioned against using short-acting barbiturates where reflexly induced hypersecretion of catecholamines could occur, as in patients with pheochromocytomata. Presumably, the effect of thiopental in such cases is not reflex but is exerted upon the adrenal medullae by some direct action of the barbiturate.

References

1. Bendixen, H. H., and Laver, M. B.: Circulatory effects of thiopental sodium in dogs, *Anesth. Analg.* 41: 674, 1962.
2. Price, H. L., Conner, E. H., Elder, J. D., and Dripps, R. D.: Effect of sodium thiopental on circulatory responses to positive pressure inflation of lung, *J. Appl. Physiol.* 4: 629, 1952.
3. Redgate, E. S., and Cellhorn, E.: The tonic effects of the posterior hypothalamus on blood pressure and pulse rate as disclosed by the action of intrahypothalamically injected drugs, *Arch. Int. Pharmacodyn. Ther.* 1: 193, 1956.
4. Egbert, L. D., Dumas, P. A., Cinter, C. C., and Eckenhoff, J. E.: Modification of the circulatory response to electroshock therapy by thiopental, *ANESTHESIOLOGY* 20: 309, 1959.
5. Skovsted, P., and Price, H. L.: The effect of halothane on arterial pressure, preganglionic sympathetic activity and barostatic reflexes, *ANESTHESIOLOGY* 31: 507, 1969.
6. Alexander, R. S.: Tonic and reflex functions of medullary sympathetic cardiovascular centers, *J. Neurophysiol.* 9: 205, 1946.
7. Halford, F. J.: A critique of intravenous anesthesia in war surgery, *ANESTHESIOLOGY* 4: 67, 1943.
8. Marc-Aurele, J., Brouillet, J., LeBoenf, G., Birge, B., Barbeau, A., and Genest, J.: A peculiar form of clinical shock, *Canad. Med. Ass. J.* 78: 589, 1958.

Drugs

LIVER DAMAGE An increase in the serum level of the enzyme ornithine carbamoyl transferase (S-OCT) is a specific and sensitive sign of hepatic damage. Increased protein catabolism leads to increased deamination, which requires greater production of this enzyme by the liver. When the catabolic process subsides, the surplus enzymes are discharged into the serum, where the level increases. S-OCT has been shown to increase following administration of chloroform, carbon tetrachloride, and alcohol. Patient studies demonstrated that: 1) halothane anesthesia for angiography produced no change in enzyme level; 2) halothane anesthesia for surgical operation was followed by an increase in S-OCT for several days; 3) surgical operation with the patient under spinal anesthesia, without a decrease in blood pressure, was followed by a slight increase in S-OCT, but no change in SGOT or SGPT; 4) spinal anesthesia with a decrease in blood pressure was followed by a marked rise in S-OCT. It is concluded that halothane does not affect hepatic function. When surgical operation is carried out with the patient under halothane or spinal anesthesia, S-OCT increases, probably because of increased breakdown of protein. A reduction in blood pressure, leading to reduced perfusion of the liver, also causes a rise in S-OCT, particularly in patients more than 50 years of age. (Brohult, J., and Gillquist, J.: *Serum Ornithine Carbamoyl Transferase Activity in Man After Halothane Anaesthesia and Spinal Anaesthesia with and without Systolic Blood Pressure Fall*, *Acta Chir. Scand.* 135: 113 (No. 2) 1969.)