pain thresholds following intravenous doses ranging from 0.6 to 2.0 mg. ($P < 0.05$). It also decreased ischemic pain. (2) Subhypnotic doses of propiomazine neither raised the pain threshold nor disminished ischemic pain. [Supported in part by a grant from Wyeth Laboratories.]

Hypnotic Actiyity of Chloral Hydrate. FRANCES MACKAY, M.D., and JACK R. COOPER, M.D., *Section of Anesthesiology and the Department of Pharmacology, Yale University School of Medicine, New Haven, Connecticut.* When chloral hydrate is administered to man or to experimental animals, it is metabolized in the body to trichloroethanol, a potent hypnotic agent. On the basis of studies of the blood levels of chloral hydrate and trichloroethanol, Butler (J. Pharmacol. Exp. Ther. *95:* 360, 1949) and Marshall and Owens (Johns Hopkins Hosp. Bull. *95:* 1, 1954) have suggested that most, if not all, of the pharmacological effect seen after the administration of chloral hydrate is due to trichloroethanol. In the present study we have tried to correlate the degree of neurological depression with the levels of chloral hydrate and trichloroethanol in the brain. *Method and Results:* In the first series of experiments, chloral hydrate, 0.4 mg./g. body weight, was given intraperitoneally to mice. The animals were sacrificed at different time intervals after the injection and the brains were analysed for chloral hydrate and trichloroethanol. During the first 5 to 10 minutes after the injection of the drug, the neurological state of the animals appeared to be related to the concentration of chloral hydrate rather than trichloroethanol. Intravenous injections of chloral hydrate, 0.4 mg./g. body weight, in 5 mice produced a loss of righting reflex in 7 to 32 seconds. The average concentration of chloral hydrate in the brain at this time was 283 μg./g. brain, while the average concentration of trichloroethanol was only 31.1 μg./g. brain. Another series of 4 mice was given 0.04 mg./g. body weight of trichloroethanol intravenously. This dose is insufficient to produce any discernable neurological effect. These mice were sacrificed 10 seconds after the end of the injection. Although these mice showed no sign of sedation, the average concentration of trichloroethanol was 86.3 μg./g. of brain. *Comment:* These data suggest that the hypnosis observed in the mice given chloral hydrate intravenously must have been produced by the chloral hydrate itself rather than by trichloroethanol. Thus chloral hydrate appears to be a potent hypnotic and may be responsible for the initial neurological effects after its administration. The rapid enzymatic reduction of chloral hydrate to trichloroethanol can account for the fact that previous workers have found a correlation between the blood level of trichloroethanol and the state of central nervous system depression during all but the very early stages of hypnosis after the administration of chloral hydrate.

Effects of Intrathecal Oxygen on Cortical Survival During Cardiac Arrest. WALTER H. MASSION, M.D., JOSEPH M. WHITE, M.D., *Department of Anesthesiology, University of Oklahoma Medical Center, Oklahoma City, Oklahoma.* The central nervous system is most vulnerable to acute oxygen depletion. Histologically, the gray matter of the brain is not uniformly affected by hypoxia. The earliest and most severe lesions are usually found in the pyramidal cell layer of the cortex (Courville, C. B.: Cerebral Anoxia, Los Angeles, San Lucas Press, 1953). Since these cells lie in close proximity to the subarachnoid space, an attempt was made to satisfy part of their oxygen requirement by simple diffusion from that space after the cerebrospinal fluid had been drained and substituted with oxygen. The rate of exchange between a gas pocket and the surrounding tissues is governed by Fick's first law of diffusion and depends on the solubility of the gas, the diffusion coefficient, the area of the gas tissue interface, the thickness of the cortex, and the pressure gradient of oxygen between the pocket and the tissues (Rahn, H.: Fed. Proc. *16:* 685, 1957). From standard values taken from the literature, it can be calculated that between 5 and 24 ml. of oxygen per minute will become available to the cortex depending on whether the highest or the lowest reported value for the diffusion coefficient is used. *Method:* The following experimental approach was chosen: oxygen was introduced through a frontal burrhole in anesthetized dogs at a