provided during periods of respiratory depression. Simultaneous arterial and venous blood samples were withdrawn at predetermined intervals following thiopental administration. *p*H was determined on arterial samples. Plasma thiopental levels were determined on venous samples using the technique of Brodie and associates. In two additional dogs effects of variations in blood *p*H on plasma thiopental levels were studied by simultaneous measurement of these variables during progressive respiratory acidosis induced by rebreathing. *Results:* In the two dogs studied during respiratory acidosis average decrease of plasma thiopental below expected values was 1.5 mg./liter per 0.1 unit decrease in blood *p*H. Plasma thiopental levels obtained from samples withdrawn during significant acidosis in all other animals were corrected to a *p*H in the normal range using this value. *Discussion:* The observed decline of plasma thiopental levels following administration of 25 mg./kg. is best defined by a two-component exponential function. The equation for control animals is: $y = 20.3 \times 10^{-.0373\,t} + 29.0 \times 10^{-.00152\,t}$ (y is concentration of thiopental in mg./liter and t is time in minutes). For dogs subjected to hemorrhage the equation is: $y = 23.9 \times 10^{-.0426\,t} + 19.3 \times 10^{-.00228\,t}$. Although the exponent defining the rapid component of the equations is larger for bled dogs, this difference is not significant. The difference between exponents defining the slow component of the equations is statistically significant ($P < .05$). Thus, under conditions of this experiment, plasma thiopental levels declined more rapidly in dogs subjected to hemorrhage than in control dogs. This is contrary to what is predicted on the basis of a dynamic concept of the distribution of thiopental in the body (Price, H.; ANESTHESIOLOGY *21:* 40, 1960). It is concluded that following hemorrhage other factors which determine plasma thiopental levels in addition to peripheral blood flow are altered. These factors might include increased ability of tissues to take up thiopental from blood and sequestration of thiopental in poorly perfused peripheral vascular beds.

Problems in Resuscitation from Cold Exposure. ANTONIO BOBA, M.D., and HIROKUNI SAKAI, M.D., *Department of Anesthesiology, the Albany Medical Center Hospital and the Albany Medical College of Union University, Albany, New York.* Death from cold exposure is due to the peripheral circulatory failure (Lynch, H. F., and Adolph, E. F.: J. Appl. Physiol. *11:* 192, 1957) at a time when no measurable oxygen deficit has been accumulated (Adolph, E. F.: Publ. 451 Nat. Acad. Sci. 1956, p. 44). On this basis it was predicated that if adequate means were made available for support of the circulation and ventilation and heat transferred from an external source, successful resuscitation could be accomplished even after circulatory and respiratory arrest. *Method:* A modification of the machine proposed by Gollan (Science *111:* 85, 1950) was employed for the purpose of resuscitating 45 splenectomized dogs immersed in ice water and allowed to breathe room air without mechanical assistance. The animals were divided into three groups. The first group of animals were removed when the heart rate had fallen to one half of control rate, the animals still breathing and the blood pressure still present. The second group of animals was removed from immersion at the time of acute circulatory failure. The third group of animals was removed from the tub at the time of "death" as manifested by ventricular fibrillation or arrest. From each group some animals were used as controls (removed from exposure and left in room air), some animals were rewarmed by the external application of heat, and some were resuscitated by means of the pump-oxygenator-heat-exchanger. *Results:* All animals removed from exposure at the time the heart rate had fallen to one-half of the control rate survived. Of the group of animals removed from exposure at the time of acute circulatory failure, the controls and all those rewarmed by external means died. All animals in whom the pump-oxygenator-heat-exchanger was employed were resuscitated and lived from 4 to 24 hours, the cause of death being atelectasis or hemorrhage. Of the group of animals removed from exposure at the time of "death," all controls and all rewarmed by external means died. All animals in whom the pump-oxygenator-heat-exchanger was employed could be resuscitated although survival was limited

to 24 hours. Resuscitation could be successfully completed even if undertaken 65 minutes after onset of apnea and 45 minutes after cardiac arrest. *Conclusion:* Tentatively, it is concluded that, if cold exposure has not progressed to the point of circulatory failure, conservative measures are effective for resuscitation. However, if the syndrome has progressed to the stage of circulatory failure, external measures are expected to fail; while some hope of success might be expected from mechanical assistance of the circulation and respiration and highly efficient means for heat transfer. [Supported by a grant from the Research Council of the American Medical Association.]

Halothane Versus Cyclopropane in Shock. P. P. Bosomworth, M.D., Z. Nikolowski, M.D., J. E. Tetirick, M.D., C. B. Edwards, Ph.D., and W. Hamelberg, M.D., *University of Kentucky Medical Center, Lexington, Kentucky, and Ohio State University Hospital, Columbus, Ohio.* General anesthesia during progressive degrees of hypovolemia is usually associated with varying changes in metabolism, cardiovascular compensation, and organ function. These changes are dependent on many factors including the anesthetic agent itself. This study investigated responses of 16 conditioned mongrel dogs to two anesthetic agents, halothane and cyclopropane, in terms of renal blood flow and resistance and femoral blood flow and resistance during progressive hemorrhage through time. The controversy between using cyclopropane and halothane anesthesia for hypovolemic states continues. Fabian (Fabian, L. W., and others: Anesth. Analg. *41:* 272, 962) reported the comparable effects of halothane and cyclopropane on renal blood flow at a 30 per cent reduction in blood volume, concluding that no significant difference in flow existed despite a difference in mean arterial pressure. *Method:* In this study anesthesia was maintained and stabilized for one hour with 0.6 per cent halothane or 10 per cent cyclopropane during exposure of the right renal and left femoral arteries. Square-wave flowmeter probes were placed around the arteries and systemic and central venous pressures were recorded. Arterial blood, 30 ml. per kilogram body weight, was withdrawn in one minute and four minutes allowed for stabilization. Arterial P_{CO_2} values were determined frequently to insure no change during measurement of flow. *Results:* With 100 per cent of the blood volume remaining, during halothane anesthesia the mean renal flow was 150.5 ml. per minute with a mean arterial pressure of 105 mm. of mercury; during cyclopropane anesthesia the mean flow was 91.7 ml. per minute with a mean arterial pressure of 125 mm. of mercury. A significant difference existed in renal flow, resistance, and arterial pressure for the two anesthetic agents ($P > 0.05$). With 70 per cent of the blood volume remaining, there was no significant difference in the renal flow and resistance or in the femoral flow and resistance under halothane or cyclopropane anesthesia. There was no significant difference in the percentage blood volume remaining at the termination of renal flow under halothane (54.7 per cent) or cyclopropane (58.0 per cent) anesthesia or of femoral flow for halothane (60.1 per cent) or cyclopropane (56.1 per cent) anesthesia. When dogs were bled (510 ml.) to termination of renal or femoral flow and administered 250, 500, and finally 1000 gamma of metaraminol, blood pressure was restored, to 125 mm. of mercury, but not renal or femoral blood flow. A reinfusion of 180 ml. of whole blood, after femoral and renal flow ceased, restored both renal (65 ml./minute) and femoral (25 ml./minute) flow to approximately one-half of the control values. *Conclusions:* There is a significant difference in the blood flow of the renal artery as influenced by cyclopropane and halothane anesthesia with normal blood volume. There is not a significant difference in the renal or femoral flow as influenced by either agent when the blood volume is reduced by 30 per cent. There is no difference in the percentage blood volume remaining when renal flow shuts off with either agent. A small volume of blood re-infused after cessation of renal and femoral flow is more effective in the restoration of flow than is the administration of metaraminol.

Potassium Superoxide as an Oxygen Source During Resuscitation. Verne L. Brechner, M.D., and Robert F. Wolff, M.D., *Division of Anesthesia, University of Cal-*