change in cardiac output. *(Kilcoyne, M.M., Schmidt, D.H., and Cannon, P.J.: Intrarenal Blood Flow in Congestive Heart Failure. Circulation 47: 786–797, 1973.)*

Respiration

MARIHUANA AND BRONCHOMOTOR TONE Seventeen volunteers with previous histories of marihuana smoking inhaled known concentrations of marihuana. Nine (high-dose group) received 84 μg/kg and eight (low-dose group) received 32 μg/kg body weight of delta-9-tetrahydrocannabinol. Pulmonary function tests of all the individuals were within normal range prior to the study. The high-dose group readily recognized the "good dope," while the members of the low-dose group were certain that they received "practically nothing." Following marihuana smoking, there was a 25 per cent increase in heart rate among the high-dose group and no significant change in the low-dose group. Functional residual capacity did not change significantly in either group. Average airway resistance in the high-dose group decreased from 1.90 ± 0.067 to 1.38 ± 0.42 cm H_2O/l/sec at 20 minutes and in the low-dose group from 2.06 to 1.67 cm H_2O/l/sec. Mean expiratory flow rate at 25 per cent of vital capacity increased from 1.79 to 2.59 l/sec for the high dose group and from 1.88 to 2.20 l/sec for the low-dose group. The ventilatory response to increasing inspired CO_2 concentrations remained unchanged after inhalation of marihuana regardless of dose. *(Vachon, L., FitzGerald, M.X., Solliday, N.H., and others: Single Dose Effect of Marihuana Smoke. N Engl J Med 288: 985, 1973.)*

ETHANOL AND RESPIRATION Six healthy adult male volunteers received ethanol, 0.35, 0.70, and 1.05 mg/kg, iv, during one hour on separate days, resulting in average blood ethanol concentrations of 40, 99 and 121 mg/l, respectively. Sixty minutes after infusion, the blood concentrations had decreased to 18, 62, and 91 mg/l, and two hours later, to 9, 51, and 80 mg/l, respectively.

Ventilatory response ($\dot{V}_E$) to carbon dioxide immediately after ethanol administration did not differ from control. Sixty minutes post-ethanol, $\dot{V}_E$ at 20 l/min was maximal, and had shifted 1.6, 2.7, and 3.9 torr.

Ethanol did not change minute volumes consistently, and there was no significant change of V_D, V_T, or Pa_{CO_2}. Following 0.7 and 1.05 ml/kg ethanol, expiratory reserve volume and R.Q. decreased significantly, while vital capacity decreased significantly only after infusion of 1.05 ml/kg ethanol. There was no alteration of these values following infusion of 0.35 mg/kg of alcohol. *(Johnstone, R.E., and Reic, C.E.: Acute Respiratory Effects of Ethanol in Man. Clin Pharmacol Ther 14: 501, 1973.)*

RESUSCITATION AND PULMONARY FUNCTION Awake, intact baboons were subjected to a standard modified hemorrhagic-shock protocol. The lungs were fixed *in vivo* and examined by electron microscope during the control period, after hemorrhagic shock, or after saline solution resuscitation. The principal changes after hemorrhagic shock were the development of interstitial edema and a striking increase in interstitial sodium concentration, as judged by staining with potassium pyroantimonate. No change in the endothelial or epithelial lining areas was found. After saline solution resuscitation, these changes had largely disappeared, and the lung specimens were virtually indistinguishable from those of the control period.

The results of this study suggest that changes in interstitial pulmonary sodium concentration must be considered in the list of forces governing the movement of water across pulmonary capillaries during hemorrhagic shock and resuscitation. Saline solution resuscitation cannot be implicated as a principal cause of post-resuscitative pulmonary insufficiency. *(Moss, G.S., and others: The Effect of Saline Solution Resuscitation on Pulmonary Sodium and Water Distribution. Surg Gynecol Obstet 136: 934–940, 1973.)* **EDITOR'S COMMENT:** This is an important piece of work. Validity of the technique needs further documentation. If the changes in osmotically active sodium can be substantiated, then present concepts of post-resuscitation pulmonary edema may have to undergo revision. The authors' finding that capillary endothelium was intact after hemorrhagic shock and prior therapy implies that the "capillary lead" syndrome is iatrogenically induced, which challenges the established tradition of blood and blood product usage for resuscitation purposes.