safely depressed to $V = 1$ by infusion of base, since $V = 1$ suffices for normal oxygenation. In principle, the permissible rate of administration can be calculated. [Supported by Grant No. RG-9069 USPHS.]

Cardiopulmonary Resuscitation: A Laboratory Evaluation. LEROY C. HARRIS, JR., M.D., HERBERT G. KUNKEL, M.D., and PETER SAFAR, M.D., *University of Pittsburgh School of Medicine and Presbyterian-University Hospital, Pittsburgh, Pennsylvania.* *Method and Results:* Controversial points of cardiopulmonary resuscitation, *i.e.*, intermittent positive pressure ventilation (IPPV) plus external cardiac compression (ECC) were evaluated in 31 anesthetized dogs with ventricular fibrillation (produced by electric shock), utilizing standardized experimental protocols. Sternal pressures, 60 per minute, were kept regular and constant by the use of a Beck-Rand machine. All lung inflations were kept constant (15 ml./kg.; air), produced by compression of a Ruben bag or a piston respirator synchronized with the Beck-Rand machine. It has been shown that ECC alone can not be relied upon to ventilate the lungs adequately (Dis. Chest *41:* 1, 1962). *Coordination of IPPV and ECC:* (1) One lung inflation interposed after each two sternal compressions was compared with one lung inflation simultaneous with every second sternal compression. Carotid blood flows were higher with simultaneous than with interposed lung inflations in 7/15 observations, the same in 5/15, and lower in 3/15. Arterial oxygen saturations remained normal (85 to 97 per cent) with interposed inflations, but dropped to an average of 65 per cent with simultaneous inflations. The progressive drop in arterial *p*H during ECC was less during interposed inflations. (2) Oxygenation with IPPV/ECC ratios of 3/15 and 6/30: During 3/15 ratios, the arterial O_2 saturation was maintained at control levels. During 6/30 ratios, the arterial O_2 saturation dropped to an average of 74 per cent at the end of 30 seconds without ventilation. Arterial *p*H and P_{CO_2} values remained closer to control levels with the 3/15 than with the 6/30 ratio. These data support our clinical recommendation to use the 3/15 ratio, at least in the nonintubated patient, where frequent interposing is difficult and brief interruptions of ECC for inflation allow recognition of airway patency. *Augmentation of Blood Flows during ECC by Pressure over the Abdomen:* Continuous pressure over the abdomen increased the artificial carotid blood flows during ECC by 25 to 50 per cent in 17/18 comparisons. This was not due to aortic compression, since both the carotid and femoral arterial pressures increased. (3) *Epinephrine and Norepinephrine during ECC:* Intravenous injections of epinephrine (0.25 mg., 0.5 mg.) given during ECC increased the artificial aortic pressures in all observations. Carotid flows did not increase in 9/14 observations and increased only minimally in 5/14. After defibrillation, intravenous epinephrine always significantly increased spontaneous aortic flows and pressures. Intravenous norepinephrine gave similar results. Subcutaneous injections of epinephrine 2 mg. given over the sternum never increased blood flows or pressures significantly in 20 minute observations. *Blood Volume Expanders during ECC:* Dextran, 25 per cent of estimated blood volume, given intravenously within five minutes to normovolemic dogs, increased the artificial carotid blood flows by 15 to 80 per cent in 7/10 and increased the arterial pressures slightly in 8/10 observations. Blood flows were more improved by intravenous dextran than by intravenous epinephrine.

Measurement of Bronchomotor Tone in Man. LAMAR P. JACKSON, M.D., and ARTHUR S. KEATS, M.D., *Division of Anesthesiology, Baylor University College of Medicine and Jefferson Davis Hospital, Houston, Texas.* Our knowledge of the action of many commonly-used anesthetic agents and adjuvants (such as narcotics and barbiturates) upon the bronchial musculature of man is tenuous. The literature revealed that many of our operational concepts regarding these actions are derived solely from *in vitro* or animal studies. These results may not represent the pharmacological actions *in vivo* and, more particularly, in man. The paucity of information seems to result from the lack of a quantitative, yet simple, method of measuring changes in bronchomotor tone in man. We have adapted a method used in dogs by Harasawa and Rodbard (J. Pharma-