diastolic blood pressures did not change significantly before intubation. Heart rate decreased in both groups before intubation. The difference was statistically significant ($P < 0.05$) at third minute only. In the group without CO_2 absorption the maximum decrease was 29 beats/minute (third minute) compared to 17 beats/minute (fourth minute) in the other group. The incidence of cardiac arrhythmias in the group without an absorber was 72.5 per cent compared to 55.5 per cent in the group with CO_2 absorption. The difference between the two groups was not highly significant ($0.10 < P < 0.20$). Ninety-four per cent of the arrhythmias were supraventricular and 6 per cent were premature ventricular contractions. *Conclusion:* A significant rise in the end-expired CO_2 concentration occurred when no soda lime canister was used in the to-fro system described above. Blood pressure changes were minimal. It is difficult to determine the role of CO_2 retention in producing bradycardia before intubation since the difference between the two groups was significant at third minute only. CO_2 accumulation may have contributed to the higher incidence of cardiac arrhythmias observed in the group without CO_2 absorption.

Sodium Bicarbonate in Cardiac Resuscitation. JOHN W. PEARSON, B.M., B.Ch., and JOSEPH S. REDDING, M.D., *Baltimore City Hospitals, Baltimore, Maryland.* It is well recognized that severe metabolic acidosis occurs during cardiac arrest. This suggests that sodium bicarbonate is of value in resuscitation. Three reasons have been given in the literautre: (1) Sodium bicarbonate solution helps restore circulation early in the resuscitation period (Ledingham, I. McA., and Norman, J. N.: Lancet 2: 967, 1962, and Bulanova, O. N., and Kiseleva, K. S.: Patol. Fiziol. i Eksptl. Terapiya 3: 59, 1959). (2) Sodium bicarbonate solution helps minimize brain damage (Ledingham and Norman, *op. cit.*). (3) There is a demonstrable decrease of myocardial contractility, and a decreased response to epinephrine, with acidosis (Thrower, W. B., and others: Arch. Surg. 82: 56, 1961). *Method:* In order to simulate clinical conditions we produced cardiac arrest by acute obstructive asphyxia. Treatment was started after the insult had occurred. We used dogs anesthetized with pentobarbital sodium (25 mg./kg.). Resuscitation consisted of artificial ventilation with air at a rate of 20 breaths per minute, tidal volume 25 ml./kg. The sternum was compressed five times during each exhalation in order to produce artificial circulation. This produced a systolic pressure of 50 to 100 mm. of mercury. Vasopressor drugs were given by intracardiac injection. Sodium bicarbonate, as a 7.5 per cent solution, was injected into the femoral vein. External cardiac massage was stopped when spontaneous circulation returned. Dogs which resumed spontaneous respiration after being taken off the respirator were considered to be immediate survivors. Three experiments were performed. *Results:* (1) In four groups of 10 dogs resuscitation was started after five minutes of arrest. In the first group no drug was used: there were 3 survivors. The second group received 50 ml. of bicarbonate (3.75 g.), and 5 survived. The third group received 50 ml. isotonic saline and 3 survived. The last group was given 75 mg./kg. bicarbonate with 6 survivors. Results are not significantly different at the 5 per cent level. There were no differences in severity of brain damage. (2) Two groups of 10 dogs were asphyxiated to circulatory arrest, and resuscitation started only ten minutes later. In the first group no drug was used: 5 dogs survived. In the second group 50 ml. of sodium bicarbonate was given; again 5 dogs survived. (3) This experiment was performed to compare the value of phenylephrine, of bicarbonate, and of a combination, when resuscitation was begun five minutes after circulatory arrest. Phenylephrine was given as a single intracardiac injection of 10 mg. Sodium bicarbonate, 150 m./kg., was given intravenously over a five minute period. There were four groups of 10 dogs each. In the first group no drug was given; 2 dogs survived. In the second group bicarbonate was used; 1 dog survived. Dogs of the third group received phenylephrine; 8 dogs survived. In the fourth group phenylephrine and bicarbonate were given; again there were 8 survivors out of 10. Thus phenylephrine is shown to be of benefit; bicarbonate alone seemed ineffective. Measurements of *p*H, P_{CO_2} and P_{O_2} in aortic blood were made in 5 dogs which received bicarbonate and phenylephrine. Determination of these values 15

minutes after ventilation was stopped showed that some degree of respiratory acidosis occurred. This may be a factor in delayed death and in brain damage. (This investigation was supported by Research Grant H-5439 from the National Heart Institute, P.H.S.)

The Effects of an Atmosphere of Nitrous Oxide and Oxygen on the Incubating Chick. GEORGE H. M. RECTOR, M.D., and DOUGLAS W. EASTWOOD, M.D., *University of Virginia School of Medicine, Charlottesville, Virginia.* Nitrous oxide has been shown to inhibit cell growth or cause cell death in the bone marrow of intact animals (Green, C. D.: ANESTHESIOLOGY *24:* 341, 1963) and to reduce the rate of mitosis in the myocardial myoblasts of the mouse embryo (Kieler, J.: Acta Pharmacol. Toxicol. *31:* 301, 1957). Because of these effects, an experiment was designed to determine the effects of an atmosphere of nitrous oxide and oxygen on the incubating chick. *Method:* Two identical electric incubators enclosed separately in plastic containers were used. The same conditions of temperature, humidity, rate of gas flow and egg movements were maintained as far as possible. Eighty per cent nitrous oxide and 20 per cent oxygen was passed through one, and air through the other. Each atmosphere was checked frequently for concentrations of oxygen, carbon dioxide and nitrogen. Blood counts and gross pathologic examinations were performed on a portion of the hatched chicks. Some were allowed to mature. Eggs which did not hatch were opened, and the embryos examined. *Results:* Our experiment showed that 10 to 20 per cent of the incubating experimental eggs will hatch, although this occurs about 36 hours later than the controls. Sixty per cent or over of the controls will hatch. Fifty to 60 per cent of the experimental eggs will progress beyond 14 days but not to hatch, as compared to over 75 per cent of the control. Spastic paralysis, present in three of the 15 live experimental chicks, was not noted in the controls. The experimental chicks that appeared grossly normal ate, drank and developed normally. Some appeared sickly though having no obvious malformations and usually died in less than 24 hours. Both red blood cell counts and smears for leucocytes were normal in all specimens examined. An attempt was made to design the experiment so that any adverse effect noted could be considered due to the absence of nitrogen or the presence of nitrous oxide. The oxygen was maintained at normal tensions and the carbon dioxide below 0.1 per cent to eliminate teratogenic and lethal effects of either a low oxygen tension or a high carbon dioxide tension. (Gallera, J.: Acta Anat. *11:* 549, 1951; Romanoff, A.: J. Morph. Physiol. *50:* 517, 1930). There is conflicting evidence on the effects of an atmosphere free of nitrogen on the chicken embryo. Volskii reported no development after five days (Dokl. Akad. Nauk. SSSR. *128:* 895, 1960). Boriskin reported a 25 per cent hatchability in helium-oxygen mixtures (Dokl. Akad. Nauk. SSSR. *143:* 457, 1962). (Supported by the American Medical Association-Education and Research Foundation Grants-in-Aid for Research Project, No. 170.)

Comparison of Effect of Norepinephrine and Angiotensin on Blood Volume and Viscosity. J. SHIBUYA, M.D., W. E. BAGEANT, M.D., P. GONZALEZ, M.D., F. H. SMALL, III, M.D., and S. N. ALBERT, M.D., *Anesthesiology Research Laboratory, Department of Anesthesiology, Washington Hospital Center, Washington, D. C.* Ample evidence supports the view that an independent volume receptor mechanism exists in the kidney, as well as a stretch mechanism, that functions by activation of the angiotensin system and production of angiotensin II (val angiotensin). Angiotensin II (Hypertensin–Ciba) is an octapeptide. It differs from sympathomimetic amines both chemically and pharmacologically and acts directly on smooth muscle and raises blood pressure by causing constriction of the precapillary sphincters (DePasquale, N. P., and Burch, G. E.: Ann. Intern. Med. *58:* 278, 1963). Norepinephrine on the other hand, produces marked constriction of the capacitance vessels, and of both pre- and postcapillary sphincters (Burch, G. E., and DePasquale, N. P.: Amer. Heart J. *60:* 915, 1960). The authors have conducted a comparative study on the effect of norepinephrine and angiotensin II on blood volume, blood viscosity, and rate of equilibration of labeled red cells with the circulating blood. *Method:* Fourteen unpremedicated patients undergoing minor surgery were studied. Five patients