rest and during exercise, with and without venous-occluding tourniquets on arms and thighs, and after release of tourniquets. Partially occluding the flow of blood (and humoral substances) from exercising muscles does not alter the overall increase in DL_{CO} produced by exercise. This study supports previous observations that the initial increase (first ten seconds of exercise) and the later increase are caused by different mechanisms. Flow of blood from exercising muscles into the pulmonary circulation is not necessary for an increase in DL_{CO} during exercise and is not the determinant of the increase in DL_{CO} during exercise. (*Hsieh, Y. C., and others: Effect of Diffusing Capacity (DL_{CO}) during Rest and Leg Exercising, Amer. J. Med. Sci. 256: 9 (July) 1968.*)

RESPIRATORY FAILURE Of 91 consecutive cases of acute respiratory failure complicating chronic lung disease, 89 per cent were managed conservatively with an overall mortality of 13 per cent. Patients were admitted to the study if the arterial carbon dioxide level was 55 mm Hg or higher and the arterial oxygen saturation was 85 per cent or less. Treatment with mechanical respirators was avoided. Oxygen, when given, was administered by special masks in 24 per cent or 28 per cent concentration. A specially trained staff provided constant intensive care consisting of respiratory physiotherapy, bronchodilator aerosols delivered by blower or freon propellant, antimicrobials, control of fluid and electrolytes and administration of aminophylline, digitalis, diuretics and, occasionally, corticosteroids. Sedative drugs were not used. Mechanical respirators were used only in patients in coma or in those who failed to improve with conservative care. Provided oxygen therapy is of the type described and general care is unremitting, hypoxemia is usually promptly improved and hypercapnia gradually lessens. (*Smith, J. P., and others: Acute Respiratory Failure in Chronic Lung Disease, Amer. Rev. Resp. Dis. 97: 791 (May) 1968.*)

PULMONARY HEMORRHAGES Pulmonary hemorrhages in the newborn have been difficult to evaluate at autopsy, and few clinicopathologic studies of this entity have been made. This study revealed that: (1) pulmonary hemorrhages are not definitely correlated with any particular clinical situation, except possibly severe intrauterine anoxia; (2) any spontaneous bleeding in the newborn should lead the clinician to suspect significant pulmonary hemorrhages; (3) in the newborn, pulmonary hemorrhages can be evaluated adequently only by microscopic examination of the lungs; (4) of the three types of pulmonary hemorrhages in the newborn, only the extra-alveolar type seems specific and is confined to premature infants with altered respiratory capabilities; and (5) many factors predispose to pulmonary hemorrhages in the newborn, including (a) any condition that can produce pulmonary congestion, (b) immaturity or altered general status of the infant, (c) increased anoxia, (d) altered hemostasis, and (e) infection. (*Parker, J. C., Jr., and others: Pulmonary Hemorrhages in the Newborn, Mayo Clin. Proc. 43: 465 (July) 1968.*)

LACTATE AND O_2 DEFICIT Because changes in P_{CO_2} alone are known to affect blood levels of lactate, experiments were designed to ascertain the effect of the level of arterial P_{CO_2} on the relationships of increases in blood lactate and excess lactate to the oxygen deficit incurred during hypoxia. Twelve anesthetized dogs were made hypoxic for 30 minutes while eucapnic and again while hypercapnic, with appropriate control and recovery periods. Another group of twelve was treated similarly except that the dogs were hypocapnic during one hypoxic period and eucapnic during the other. The net O_2 deficit was estimated from the decrease in V_{O_2} from the baseline value just prior to hypoxia and corrected for changes in O_2 stores. A linear relationship was obtained for changes in blood lactate and net O_2 deficits which were different at each level of P_{CO_2} and which varied inversely with P_{CO_2} level. Similar results were obtained for the relationship of excess lactate to net O_2 deficit. Therefore, the more complicated measure of excess lactate offered no advantage over the simpler measurement of the lactate increase. (*Cain, S. M.: Effect of P_{CO_2} on the Relation of Lactate and Excess Lactate to O_2 Deficit, Amer. J. Physiol. 214: 1322 (June) 1968.*)