should be watched for and treated. (*Tyler, F. H., Hyperosmolar Coma, Amer. J. Med. 45: 485 (Oct.) 1968.*)

Respiration

CSF OXYGEN TENSION Oxygen and carbon dioxide tensions were determined in 320 CSF samples. In a few patients, cisternal fluid P_{O_2}, P_{CO_2} and *p*H values were compared with simultaneously-obtained arterial and jugular venous blood values for calculation of the mean P_{O_2} in cerebral capillaries. Normal cisternal fluid was found to have values between those of arterial and jugular venous blood. P_{O_2} in lumbar CSF (31 mm Hg) is lower than that in cisternal fluid (47 mm Hg), and the P_{CO_2} is higher (44 *vs.* 37 mm Hg). There was significant *correlation* of the gas tensions in cisternal fluid with the mean capillary P_{O_2} and P_{CO_2} of the brain. There was, however, no evidence that cisternal fluid P_{O_2} is *representative* of mean P_{O_2} of cerebral tissue. In fluid samples with increased cell counts, P_{O_2} decreased with the degree of pleocytosis. An increase in protein was not associated with a decreased oxygen tension in CSF. AV malformations produced high P_{O_2} values in the jugular vein, provided the vein drained the shunt. In these cases, jugular P_{O_2} was occasionally found to be higher than cisternal fluid P_{O_2}. A clinical application of these findings may be the measurement of CSF oxygen tension in lieu of the carotid–jugular AV difference, thus avoiding puncture of two vessels and potential heparinization when continuous monitoring of intravascular gas tensions is indicated. In drainage of ventricles (which may extend over many hours or several days), continued monitoring of CSF gas tensions is simple, without hazard, and can provide information about perfusion and oxygen consumption of the brain. Monitoring CSF gas tensions may also be useful in the study of the effects of vasopressors or anesthetic drugs on the brain. (*Gaenshirt, H.: Oxygen Tension in Cerebrospinal Fluid of Man. Physiological and Clinical Significance, Klin. Wschr. 46: 771 (July) 1968.*)

RESPIRATORY RESISTANCE Total respiratory, lung and chest wall flow resistances were measured in spontaneously-breathing patients with obstructive lung disease by imposing flow oscillations at the airway. Total respiratory and lung resistance decreased with increasing breathing frequency. Compliance was also frequency-dependent. Such frequency dependence was interpreted as a function of uneven distribution of mechanical properties of the lungs. (*Grimby, G., and others: Frequency Dependence of Flow Resistance in Patients with Obstructive Lung Disease, J. Clin. Invest. 47: 1455 (June) 1968.*) ABSTRACTER'S COMMENT: This paper is vital not only for its conclusions but because of the thoroughness with which the technique of forced oscillations was investigated. Although more than a decade has elapsed since the oscillator technique for investigating airflow resistance was proposed, the application of this technique is apparently coming into fashion. We have witnessed cumbersome techniques such as the plethysmographic, esophageal balloon and interrupter methods. It will remain for time to determine whether the oscillator technique is better. Simultaneous determinations of lung volume should be made but, unfortunately, this remains extremely difficult in a supine, anesthetized patient.

DIFFUSING CAPACITY Changes in pulmonary capillary blood volume have been studied by various techniques. The carbon monoxide diffusing capacity (DL_{CO}) is of special interest. Since exercise causes greater increases in breath-holding pulmonary diffusing capacity than can be produced by other means, the mechanism by which this change occurs is important. The increase in DL_{CO} implies an enlargement of the effective pulmonary capillary bed. Previous studies have shown that increases in pulmonary blood flow alone do not increase DL_{CO}. Procedures which transfer blood from the peripheral circulation to the lung or increase pulmonary vascular pressure do increase DL_{CO} somewhat, primarily by increasing pulmonary capillary blood volume. Breath-holding DL_{CO} was determined in 12 normal subjects, seated and supine, at

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. (*Hsich, 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.*)