

free plasma, no evidence of circulating anti-A, B or AB antibodies has been found. (McDonald, W., and others: *A Hidden Danger of Pooled Plasma*, *Amer. J. Surg.* 114: 629 (Nov.) 1967.)

Kidney

ANESTHETIC OLIGURIA The kidney is placed in functional jeopardy (1) when systemic arterial pressure is reduced to levels below 80 mm. Hg or (2) when renal vascular resistance is increased markedly. Emotional stress, pain, anesthesia, narcotics, sympathomimetic amines and trauma may cause increases in renal vascular resistance. General anesthesia by any agent is probably always attended by decreases in renal plasma flow (RPF) and glomerular filtration rate (GFR). A decrease in RPF of 32 to 53 per cent has been shown to occur during ether anesthesia; 32 to 63 per cent during cyclopropane anesthesia. Thiopental is associated with a 31 per cent decrease in RPF. Meperidine, 100 mg., can reduce RPF by 30 per cent. Spinal anesthesia alone causes no change in RPF. Postoperatively, reduced renal blood flow may not return to normal for two or three hours. Systemic arterial pressure curves during major surgery do not reflect the adequacy of renal blood flow. (Coln, H. E., and Capelli, J. P.: *Diagnosis and Management of Oliguria in the Postoperative Period*, *Surg. Clin. N. Amer.* 47: 1187 (Oct.) 1967.)

DEXTRAN RENAL FAILURE Three patients with advanced atherosclerotic vascular disease developed acute renal failure following infusions of low-molecular-weight dextran. These complications prompted an experimental evaluation of the effect of rapid dextran infusion on the kidney function of dogs. In each animal, the function of the normal right kidney was determined for comparison with the left which had been subjected to partial constriction of its renal artery. In five of eight experiments, dextran produced anuria in the constricted kidney. Histologically, more dextran was demonstrated in the anuric than in the control kidneys. In three of four studies, the anuria persisted after release of the renal artery constriction. A reduction in filtration

pressure combined with a marked increase in urinary viscosity produced by the dextran may lead to tubular stasis and subsequent blockade. (Mailloux, L., and others: *Acute Renal Failure after Administration of Low-molecular-weight Dextran*, *New Eng. J. Med.* 277: 1113 (Nov.) 1967.)

Respiration

AIRWAY STABILITY Distensibility of airways was measured during reflex bronchoconstriction produced by laryngeal irritation in cats. Ratio of anatomic deadspace to transpulmonary pressure was used as an index of airway compliance, and lung resistance was used to estimate the degree of bronchoconstriction. During reflex bronchoconstriction the airways became less distensible. This was interpreted as indicating increased airway stability during bronchoconstriction. This is a possible mechanism to minimize compressive narrowing of airways by increased intrapleural pressure during performance of maneuvers to increase gas flow. (Olsen, C. R., DeKock, M. A., and Colebatch, H. J. H.: *Stability of Airways during Reflex Bronchoconstriction*, *J. Appl. Physiol.* 23: 23 (July) 1967.)

PULMONARY MECHANICS During forced exhalation, most young individuals expire vital capacity quickly (2 to 4 sec.), and no further volume change occurs in spite of continuing expiratory effort. Older subjects approach residual volume more slowly (10 to 15 sec.); some continue to expire with decreasing flow as long as expiratory effort is maintained. When external pressure assisting exhalation was applied at or near residual volume, spiograms of young individuals indicated that a small additional increment of gas was exhaled, whereas in most elderly subjects, prevailing expiratory flow rate was not affected. These results suggest that in young individuals static balance of muscle and recoil forces, mainly in the chest wall, sets the limit to forced exhalation, and external forces can alter this balance. In older subjects, with diminished lung recoil and perhaps diminished airway conductance, the limiting mechanism may be a dynamic one operating in the lung rather than in the chest wall. (Leith, D. E.,

and Mead, J.: *Mechanisms Determining Residual Volume of the Lungs in Normal Subjects*, *J. Appl. Physiol.* 23: 221 (Aug.) 1967.)

RESPIRATORY MECHANICS Hysteresis is that property whereby the pressures exerted by the elastic recoil of the respiratory system are greater on inflation than on deflation at the same volume. Stress adaptation refers to the relatively slow fall in transrespiratory pressure following the sudden pressure rise accompanying abrupt inflation, or the relatively slow rise in transrespiratory pressure following an abrupt steep deflation. These phenomena were investigated in anesthetized volunteers during neuromuscular blockade produced by succinylcholine. In normal subjects, hysteresis and stress adaptation were present in lungs, and to a slightly lesser extent in the thorax. Stress adaptation increased with lung volume in a nonlinear fashion and was disproportionately high at large lung volumes. Stress adaptation probably resides in the properties of respiratory system tissue components in both lung and thorax; some characteristics of stress adaptation may be explained by a simple mechanical analogue consisting of viscoelastic units arranged in series and parallel combinations. (Sharp, J. T., and others: *Hysteresis and Stress Adaptation in the Human Respiratory System*, *J. Appl. Physiol.* 23: 487 (Oct.) 1967.)

PULMONARY DYNAMICS IN ASTHMA

Hyperinflation of the lungs and increased functional residual capacity are known to persist in asthmatics after relief of airway obstruction. The present study demonstrated that this pulmonary hyperinflation was due to decreased elastic recoil of the lungs and was reversible with continued treatment. Acute reduction in elastic recoil of the lungs could not be duplicated in healthy volunteers or treated asthmatics by induced bronchoconstriction with histamine aerosol or by sustained mechanical hyperinflation. Diminished elastic recoil of the lungs may be partly responsible for the decreased maximal airflow rates in asthmatic patients. (Gold, W. M., Kaufman, H. S., and Nadel, J. A.: *Elastic Recoil of the Lungs in Chronic Asthmatic Patients before and after*

Therapy, *J. Appl. Physiol.* 23: 433 (Oct.) 1967.)

PULMONARY MECHANICS Elastance (reciprocal of compliance) of the human lung was compared during the conscious relaxed state and during anesthesia with neuromuscular blockade produced by succinylcholine. Subjects were normal volunteers and individuals with obstructive lung disease, ankylosing spondylitis, and extreme obesity. Each subject served as his own control. Thoracic and total respiratory elastance were consistently greater (compliance was lower) in the conscious, voluntarily-relaxed state than in the anesthetized, paralyzed state; elastances in the two states correlated poorly. The most reasonable explanation for the higher elastances measured in the conscious state is the presence of expiratory muscle activity at the resting midposition. This muscle activity probably is not under voluntary control and may be activated by muscle spindle reflexes. It was concluded that elastances measured in the conscious, relaxed state are probably of questionable validity, particularly in poorly-trained subjects. Results of this study are at variance with most previously-reported measurements of pressure-volume relationships during anesthesia, in which compliance generally has been lower during anesthesia than in the conscious state. (Van Lith, P., Johnson, F. N., and Sharp, J. T.: *Respiratory Elastances in Relaxed and Paralyzed States in Normal and Abnormal Men*, *J. Appl. Physiol.* 23: 475 (Oct.) 1967.)

HYPERCARBIA In most patients with respiratory failure, neurologic effects are due to hypoxia rather than to CO₂ retention because arterial CO₂ partial pressure cannot rise above 80 mm. Hg during breathing of air. The patient dies of anoxia before CO₂ rises to narcotic levels. CO₂ narcosis can occur only during breathing of oxygen. The neurologic manifestations of CO₂ retention are entirely nonspecific, with the possible exception of papilledema, which is by no means common. The CO₂ tension is important for six main reasons: (1) A very high CO₂ tension causes CNS depression and convulsions.