probably caused by anoxia and increased cerebral blood flow. The increased blood flow causes further rise in intracranial pressure, additional anoxia, and eventual vasomotor paralysis. Hyperbaric oxygenation serves to break this cycle, presumably by combating the anoxia while at the same time decreasing cerebral blood flow by vasoconstriction. (*Sukoff, M. H., and others: The Protective Effect of Hyperbaric Oxygenation in Experimentally Produced Cerebral Edema and Compression, Surgery 62: 40 (July) 1967.*)

NOREPINEPHRINE The effects of neuronally-transmitted and blood-borne norepinephrine (NE) on the isolated perfused hind limb and on an isolated segment of the splanchnic circulation were studied in dogs. A comparison was made between intra-arterial injection of NE in the isolated segments and reflex stimulation of these sites produced by carotid body hypotension before and after alpha and beta adrenergic blockade. Results in the two circulations were similar. Intra-arterial NE produced vasoconstriction in the untreated animals, as did reflex release of NE. However, after alpha blockade intra-arterial NE produced vasodilation, whereas reflex stimulation still produced some vasoconstriction although of a much smaller degree than in the controls. In other words, the beta adrenergic effects were unmasked by alpha blockade with injection of NE but not by reflex stimulation. The administration of beta blockers re-reversed the response to NE in the intra-arterial injection, causing vasoconstriction. There was, however, no augmentation of the vasoconstrictor effect on neuronal stimulation after beta blockade. It is postulated that NE released from nerve terminals gains access to alpha receptors but not to beta receptors, whereas blood-borne NE is effective at the alpha receptors reached by nerve stimulation and possibly at other alpha receptor sites not reached by nerve stimulation, and is also effective at beta receptor sites. (*Glick, G., and others: Physiological Differences Between the Effects of Neuronally Released and Bloodborne Norepinephrine on Beta Adrenergic Receptors in the Arterial Bed of the Dog, Circ. Res. 21: 219 (Aug.) 1967.*)

CAVAL OBSTRUCTION Twelve dogs were anesthetized and pressure recordings were made from catheters placed in the peritoneal cavity, in the vena cava just below and just above the diaphragm. Glycerine, saline or air was introduced intraperitoneally in stepwise fashion. When intraperitoneal volume reached 200 ml./kg., intra-abdominal pressure was greater than 30 mm. Hg. Thoracic vena caval pressure rose to more than 30 mm. Hg. There was narrowing and angulation of the vena cava at the level of the diaphragm. Blood flow through the vena cava diminished to less than half the control value. All values returned to normal when intra-abdominal pressure was reduced. (*Rubinson, R. M., and others: Inferior Vena Caval Obstruction From Increased Intra-abdominal Pressure, Arch. Surg. 94: 766 (June) 1967.*)

IPPB AND CIRCULATION Ventilation and cardiac output were measured simultaneously in human volunteers during breathing against graded increments of positive pressure. Cardiac output decreased as positive pressure increased, and when ventilation was maintained constant at 10 l./min., magnitude of the decrease in cardiac output was 0.2 l./min. per additional centimeter of positive pressure imposed. At any level of positive pressure, increases in ventilation counteracted the circulatory effects of positive pressure in the airway; the data suggest that magnitude of tidal volume is an important factor. (*Cruz, J. C., Cerretelli, P., and Farhi, L. E.: Role of Ventilation in Maintaining Cardiac Output Under Positive Pressure Breathing, J. Appl. Physiol. 22: 990 (May) 1967.*)

ARTERIOVENOUS SHUNTS In 44 healthy dogs, continuous infusion of epinephrine in amounts capable of being produced endogenously in shock caused highly significant increases in portal flow and oxygen pressure and femoral venous oxygen pressure. These findings, unexplained by changes in cardiac output or metabolic rate, were thought to be caused by the opening of multiple arteriovenous shunts in these vascular systems. It is suggested that A-V shunts, instead of excessive vasoconstriction due to epinephrine,

may cause the detrimental effects of late shock. (*Berk, J. L., and others: The Effect of Epinephrine on Arteriovenous Shunts in the Pathogenesis of Shock, Surg. Gynec. Obstet. 124: 347 (Feb.) 1967.*)

Respiration

HYPERPNEA OF EXERCISE Previous work has suggested that in some stressful situations chemoreceptor function may be influenced by activity of the sympathetic nervous system. In the present study, the magnitude of hyperpnea caused by exercise as well as associated changes in Pa_{O_2}, Pa_{CO_2} and *p*H were evaluated before and after blockade of the sympathetic innervation of the carotid body by bilateral stellate ganglionic block. Results of the study indicated that during exercise, the central nervous system does not operate through the sympathetic innervation of the carotid chemoreceptors or sensitize them to their usual stimuli of hypoxia and hydrogen ion concentration. (*Eisele, J. H., Ritchie, B. C., and Severinghaus, J. W.: Effect of Stellate Ganglion Blockade on the Hyperpnea of Exercise, J. Appl. Physiol. 33: 966 (May) 1967.*)

RESPIRATORY DISTRESS Idiopathic respiratory distress syndrome (IRDS) mothers have significantly lower levels of gamma-globulins than control groups, although they do not differ significantly with respect to the concentrations of total serum protein and serum albumin. The possible explanations are: (1) pregnancy-induced defect in synthesis of gamma-globulins, resulting in lowered maternal serum concentrations and diminished placental transfer; (2) fetal catabolism of maternal globulins; and (3) an immune reaction, with maternal gamma-globulins as either an antibody or an antigen. (*Hardie, G., and Kench, J. E.: Maternal Serum Proteins in Idiopathic Respiratory Distress Syndrome of the Newborn, Lancet 1: 809 (April) 1966.*)

SMOKING Of 133 college seniors, those who smoked had a significantly greater incidence of cough, phlegm, breathlessness, wheezing and colds. All five students with hemoptysis and four with histories of peptic ulcer were smokers. Though exceptions occurred, a dose-response trend was demonstrated between lifetime packs smoked and decreased expiratory flow rates. Surprisingly little smoking was necessary to produce early functional changes of a type compatible with early nonspecific respiratory disease. The degree to which students inhaled was not determined. (*Peters, J. M., and others: Smoking, Pulmonary Function, and Respiratory Symptoms in a College-age Group, Amer. Rev. Resp. Dis. 95: 774 (May) 1967.*)

SMOKING From a retrospective study of 1,623 male and 404 female college graduates based on their visits to the student health service, it was found that the increase in incidence and severity of respiratory disease was highly significant in smokers as compared with nonsmokers. This relationship held for years smoked and lifetime packs smoked. (*Peters, J. M., and others: Smoking and Morbidity in a College-age Group, Amer. Rev. Resp. Dis. 95: 783 (May) 1967.*)

SMOKE INHALATION As many as 1,200 deaths per year in the United States are attributed to smoke inhalation, yet the subject has received little attention in the medical literature. Of particular importance is recognition of the six- to 48-hour latent period which may ensue before complications of acute bronchial obstruction, pneumonia, pulmonary edema, and eventual cardiopulmonary failure develop. Management may require tracheostomy, prolonged intermittent positive-pressure breathing with appropriate concentrations of oxygen and high humidity, and, when indicated, administration of systemic antibiotics and steroids. (*Webster, J. R., and others: Recognition and Management of Smoke Inhalation, J.A.M.A. 201: 287 (July) 1967.*)

EXPERIMENTAL EMPHYSEMA Rabbits continuously exposed to an atmosphere of 8 to 12 parts per million of nitrogen dioxide for three to four months were found to have lung changes which did not regress and were compatible with a diagnosis of emphysema. (*Haydon, G. B., and others: Nitrogen Dioxide-Induced Emphysema in Rabbits, Amer. Rev. Resp. Dis. 95: 797 (May) 1967.*)