BARORECEPTORS On the basis of blood pressure responses to stage-by-stage destruction of the baroreceptors (pressure receptors), the baroreceptors reflexes were exaggerated under chloralosane and depressed under barbital. There was no significant difference between ether and pentobarbital. Ether did not show a marked depression of baroreceptor reflexes, as has been reported for the chemoreceptor reflexes. (*Brown, R. V., and Hilton, J. G.: Effectiveness of the Baroreceptor Reflexes Under Different Anesthetics, J. Pharmal. & Exper. Therap. 118: 198 (Oct.) 1956.*)

STRESS The eosinopenia resulting from induction of pentobarbital anesthesia in dogs may be an indication of the degree of stress imposed by the procedure. Intravenous administration of acetylcholine caused a greater eosinopenia in pentobarbital anesthetized dogs than in controls. (*Schopp, R. T.: Effect of Pentobarbital Sodium and Acetylcholine on Eosinophil Count in Dogs, Am. J. Physiol. 186: 488 (Sept.) 1956.*)

ACID-BASE BALANCE The excretion of titratable acid in the urine of anesthetized dogs increased during respiratory acidosis and decreased during respiratory alkalosis. Measurements during recovery periods revealed that the effects of respiratory acidosis were rapidly reversible, while respiratory alkalosis produced a lasting depression of the tubular capacity to secrete hydrogen ions. (*Fuller, G. R., and MacLeod, M. B.: Excretion of Titratable Acid During Acute Respiratory Disturbances of Acid-Base Balance, Am. J. Physiol. 186: 505 (Sept.) 1956.*)

ANOXIA Anoxia *per se* is not the cause of glycogen decrease in the anoxic hearts of rats, but the amount of work performed by the anoxic heart is the critical factor in producing glycogenolysis. (*Bloom, W. L.: Glycogenolysis in Anoxic Heart, Am. J. Physiol. 186: 518 (Sept.) 1956.*)

DEFORMED CHESTS Ventilatory and lung volume measurements were studied in patients with kyphoscoliosis, Marie-Strumpel disease, pectus excavatum, and fusion of thoracic spine, who were free of pulmonary or cardiac symptoms. The abnormalities were characterized by a decreased lung volume due to decreased expansibility of the chest wall and an accompanying restrictive breathing pattern. (*Iticovici, H. N., and Lyons, H. A.: Ventilatory and Lung Volume Determinations in Patients with Chest Deformities, Am. J. Med. Sc. 232: 265 (Sept.) 1956.*)

EMPHYSEMA As determined by ear oximeter, the 90 per cent desaturation time separated patients with diffuse obstructive emphysema, with or without pulmonary fibrosis, from normal subjects and from patients with pulmonary disease other than emphysema. The degree of prolongation of the desaturation time was not directly related to the severity of the emphysema as judged by clinical disability and the maximum breathing capacity. (*Woolf, C. R., and others: Simple Tests of Respiratory Function Using Direct-Writing Ear Oximeter, Am. Rev. Tubere. 74: 511 (Oct.) 1956.*)

CO_2 NARCOSIS Acute ventilatory insufficiency and carbon dioxide narcosis usually develops when ventilation becomes mechanically more difficult or when the respiratory center is depressed by anesthetic or sedative drugs. Therapy includes bronchoscopic drainage, bronchodilator drugs, steroids, antimicrobial drugs, correction of congestive heart failure, and increase in alveolar ventilation. Five cases were successfully treated with a Seeler mechanical resuscitator with careful attention to maintenance of a patent airway. Tracheal intubation could be continued twenty-four hours without severely damaging the larynx or trachea. (*Sieker, H. O., and others: Treatment of Carbon Dioxide Narcosis by Use of Automatic Positive-Negative Resuscitator, Am. Rev. Tubere. 74: 309 (Sept.) 1956.*)

VENTILATION The alveolar-arterial oxygen pressure gradient was more influenced by the alveolar oxygen partial pressure than by the degree of ventilation of the lungs of anesthetized dogs. It would seem that ventilation is important as it affects alveolar oxygen partial pressure and not primarily as it ventilates poorly aerated areas of the lung. (*Atwell, R. J., Tomashefski, J. F., and Ryan, J. M.: Factors Influencing Alveolar-Arterial Oxygen Pressure Gradient Effect of Ventilation and Alveolar Oxygen Tension, Am. J. Physiol. 186: 501 (Sept.) 1956.*)

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