the possibility of hypotension and oxygen apnea, artificial respiration with oxygen through the tracheostomy is recommended. (*Glas, W. W., King, Jr., O. J., and Lui, A.: Complications of Tracheostomy, A. M. A. Arch. Surg. 85: 56 (July) 1962.*)

BRONCHIAL ASTHMA Unilateral bronchial asthma was provoked in two asthmatic subjects employing an allergen inhaled as an aerosol into one lung during bronchospirometry. Presence of asthma was demonstrated by a reduction of ventilation and oxygen uptake and a prolonged nitrogen elimination time during oxygen breathing. Unevenness of distribution of the inspired gas did not increase significantly, but ventilatory efficiency decreased, probably as a result of increased dead space/tidal volume ratio in the provoked lung. The asthmatic reaction was abolished within 15 seconds by epinephrine, suggesting that bronchospasm was the main causative factor for this kind of asthma since it is unlikely that edema or mucous secretion could be so rapidly abolished. The effect of theophylline was more gradual but lasted longer, indicating the value of a combination of epinephrine and theophylline in the treatment of asthma. The effect of the preoperative medication (an opiate and scopolamine) or the topical anesthesia (4 per cent lidocaine) was difficult to judge. (*Arborelius, M., and others: Unilateral Provoked Bronchial Asthma in Man, J. Clin. Invest. 41: 1236 (June) 1962.*)

FLAIL-CHEST Seventeen patients with flail-chest injuries treated with a Mörch piston respirator were compared with 16 similar patients treated without the respirator, utilizing towel-clip stabilization, tracheostomy, occasionally IPPB with other types of ventilators, and similar overall supportive care. "Wet lung syndrome" was considered a major cause of mortality in such cases and was prevented or reversed with a significant reduction in mortality in the piston-respirator group as compared with the other group. (*Ransdell, H. T., Jr., and others: Treatment of Flail Chest Injuries with the Piston Respirator, Amer. J. Surg. 104: 22, 1962.*) [*Abstractor's comment*—Analysis of the data presented indicates a major bias favoring recovery in the piston-respiratory group in that the incidence of associated injuries in the nonpiston respirator group is twice that of the piston respirator group. Associated injuries were defined as major fractures, severe head trauma, shock, ruptured viscera, and major hemothorax, usually associated with pneumothorax. Thus, the significance of the difference between these two groups is questionable.]

CORTICOSTEROID THERAPY Seventeen patients who had received long periods of corticosteroid therapy have been investigated to determine the integrity of the pituitary-adrenal axis. In 16 of these no evidence of damage to this axis was obtained, while severe adrenal inertia due to damage to this corticotrophin-releasing mechanism had developed in one. A number of patients will respond in a gratifying way to an infusion of hydrocortone, but this has been shown to occur in individuals with demonstrably normal cortisol levels and functioning mechanisms for its release, and such beneficial results are not evidence of adrenal cortical failure. (*Robison, B. H. B., Mattingly, D., and Cope, C. L.: Adrenal Function After Prolonged Corticosteroid Therapy, Brit. Med. J. 1: 1579 (June 9) 1962.*)

STRESS Effect of complete lesions of the spinal cord above the fifth thoracic level upon adrenocortical response to stress was evaluated in three patients. Plasma and urinary steroid levels during and after surgical procedures were not significantly different from those found in patients without spinal cord lesions. An intact suprasegmental innervation of the adrenal medulla does not seem to be essential for the normal adrenocortical response to stress. (*Osborn W., and others: Adrenal Function in Patients with Lesions High in Spinal Cord, Urology 88: 1 (July) 1962.*)

ACTH RELEASE Pituitary tumor graft in a hypophysectomized rat has been employed to test the ACTH-releasing activity of neurohumoral substances. Vasopressin may be the ACTH-releasing neurohumor. (*Grindeland, R. E., Wherry, F. E., and Anderson, E.: Vasopressin and ACTH Release, Proc. Soc. Exp. Biol. Med. 110: 377 (June) 1962.*)