

**INTRAVENOUS LIDOCAINE** In four patients undergoing cardiac catheterization under "light barbiturate anesthesia," infusion of 1 mg lidocaine per minute for one hour caused no significant change in cardiac output, heart rate, stroke volume, mean arterial blood pressure or total systemic resistance. One patient had a transient drop in left ventricular dp/dt from 1,518 to 1,363 mm Hg/sec after intravenous injection of 1 mg/kg of lidocaine over a two-minute period. Injections of 400 mg lidocaine in dogs (almost 20 mg/kg) caused transient severe falls in arterial blood pressure. In patients receiving large doses of lidocaine, arterial blood pressure as well as the electrocardiogram should be monitored. (Binnion, P. T.: *Toxic Effects of Lignocaine on the Circulation*, *Brit. Med. J.* 1: 470 (May) 1968.)

**OCULOCARDIAC REFLEX** Oculocardiac reflexes occur quite frequently during operations on the eye performed with the patients under general anesthesia. Sympathetic effects are far more dangerous than parasympathetic ones. Several patients had serious disturbances in rhythm following traction on eye muscles. In one patient (a 24-year-old man, physical status I) traction on the bulbous caused tachycardia, followed by cardiac arrest which responded to resuscitation. (Benzer, H., and others: *Pulse Rate during Ophthalmic Surgery*, *Der Anaesthetist* 17: 157 (May) 1968.)

**CARDIAC MASSAGE** During closed-chest cardiac massage, left-atrial and systemic arterial blood pressures are nearly equal. The former falls while the latter rises toward pre-arrest values with the resumption of effective cardiac activity. The high left-atrial pressures may explain pulmonary edema after prolonged closed-chest cardiac massage. An extremely critical state exists so long as there is no effective cardiac contraction even if a satisfactory systemic arterial blood pressure is maintained by closed-chest cardiac massage. (Thomsen, J. E., and others: *Intracardiac Pressures during Closed-chest Cardiac Massage*, *J.A.M.A.* 205: 46 (July) 1968.)

## Respiration

### RESPIRATORY NEURONS AND CO<sub>2</sub>

The effects of changes in alveolar CO<sub>2</sub> tension on discharge patterns of medullary and pontine respiratory neurons were studied in cats. Efferent phrenic-nerve discharge served as an indicator of central respiratory periodicity. In all neurons, lowered CO<sub>2</sub> resulted in reduction of respiratory oscillation of discharge frequency. On the basis of direction and degree of change in discharge, the responses to lowered CO<sub>2</sub> levels were classified as three major types. Respiratory periodicity arises from activity of systems of reciprocally discharging neurons: a pair of reciprocal systems consisting of expiratory-facilitatory neurons with type 2 responses and inspiratory-facilitatory neurons with type 1 responses; a pair of reciprocal systems consisting of inspiratory-facilitatory neurons with type 2 responses and expiratory-facilitatory neurons with type 1 responses. (Cohen, M. I.: *Discharge Patterns of Brainstem Respiratory Neurons in Relation to Carbon Dioxide Tension*, *J. Neurophysiol.* 31: 142 (March) 1968.)

### PULMONARY MECHANICS

Lung tissue resistance is not a constant fraction of total pulmonary resistance. It is directly related to size of tidal volume, inversely related to vital capacity and lung compliance and independent of the flow rate at which it is measured. These observations suggest that lung tissue resistance is not an ohmic type of resistance where force required to overcome oppositions to motion originating in lung tissue would be related to flow. Lung tissue resistance probably represents the degree of retarded elastic response of the lung related to non-flow-resistive volume-pressure hysteresis and stress relaxation. (Bachoffen, H.: *Lung Tissue Resistance and Pulmonary Hysteresis*, *J. Appl. Physiol.* 24: 296 (March) 1968.)

### LUNG MECHANICS

Comparison of esophageal and pleural pressures in anesthetized dogs in the prone position indicated that differences were less than 3 cm H<sub>2</sub>O except at extremes of lung volume, and pneumothorax did not systematically alter the differences. When static volume-pressure curves obtained

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