to 6 to 14 minutes, but the breaking-point alveolar carbon dioxide tensions did not exceed those noted above. A decrease in lung volume occurred, which in 13 minutes was equal to the vital capacity. (*Klocke, F. J., and Rahn, H.: Breath Holding After Breathing of Oxygen, J. Appl. Physiol. 14: 689 (Sept.) 1959.*)

BREATH HOLDING When the breath is held the arterial P_{CO_2} rises rapidly as a result of decreased clearance of carbon dioxide from venous blood, the lung volume shrinkage, and the Haldane effect from oxygenation of hemaglobin. The venous P_{CO_2} rises more slowly because of the uptake of carbon dioxide by the tissues and the Haldane effect from reduction of oxyhemoglobin. By this mechanism the carbon dioxide output into the lungs progressively falls and eventually stops. The cycle then is reversed and the carbon dioxide moves from lungs to arterial blood. (*Mithoefer, J. C.: Mechanism of Pulmonary Gas Exchange and Carbon Dioxide Transport During Breath Holding, J. Appl. Physiol. 14: 706 (Sept.) 1959.*)

ARTIFICIAL RESPIRATION In the collapsed, nonbreathing victim, the direct mouth-to-mouth method is without doubt the most effective way to restore life. The mouth to oral airway or self-inflating bag with mask is the safest and most satisfactory method for those who have had limited instructions. Any other apparatus should be used only by those specially trained in resuscitation. (*Dobkin, A.: Save a Life with a Breath of Air, Canad. M. A. J. 81: 458 (Sept. 15) 1959.*)

ARTIFICIAL RESPIRATION Mouth-to-mouth respiration by laymen often fails to be effective because of the difficulty in maintaining a free airway and insuring a good enough seal. These deficiencies could be overcome by teaching the laymen to open his mouth more widely around the victim's mouth to insure a tight seal and to insist upon maximum hyperextension of the patient's head. This method was tested on more than 300 unconscious patients; there were only three in whom good artificial respiration could not be maintained. (*Ruben, A., Elam, J. O., and Ruben, H.: Rescue Breathing, Lancet 2: 69 (Aug. 1) 1959.*)

AIRWAY OBSTRUCTION Airway patency was studied in 80 anesthetized spontaneously breathing patients who received no muscle relaxants. With the neck flexed (chin towards chest) the airway was obstructed in all patients, both in the supine and prone positions. With extension at the atlanto-occipital joint (chin up) in the supine position, approximately 50 per cent of the patients had an open airway. The other 50 per cent required, in addition to extension of the neck, forward displacement of the mandible or the insertion of an oropharyngeal airway or both. Roentgenograms demonstrated that the tongue is pushed against the posterior pharyngeal wall when the neck is flexed and the mandible is not held forward. (*Safar, P., Escarraga, L. A. and Chang, F.: Upper Airway Obstruction in the Unconscious Patient, J. Appl. Physiol. 14: 760 (Sept.) 1959.*)

AIRWAY RESISTANCE Alveolar-arterial oxygen differences were determined in anesthetized, paralyzed man. Approximately 20 per cent of the determinations showed a difference greater than 20 mm. Hg. The arterial tension was increased an average of 10 mm. Hg when the expiratory pressure was increased from -5 to $+5$ mm. Hg. The insertion of a 3 mm. orifice expiratory resistance increased the arterial oxygen tension an average of 7 mm. Hg. (*Frumin, M. J., and others: Alveolar-arterial Oxygen Differences During Artificial Respiration in Man, J. Appl. Physiol. 14: 694 (Sept.) 1959.*)

TRACHEOTOMY DANGERS Five cases of fatal cardiovascular and respiratory collapse immediately after tracheotomy are presented. It is hypothesized these were due to rapid reversal of pre-existing respiratory acidosis. Patients having elective or semielective tracheotomies for relief of respiratory obstruction should be followed with frequent blood pressure recordings, and hypotension should be treated with Trendelenburg position and intravenous vasopressors. (*Greene, N. M.: Fatal*