electroencephalographic effect. (*Davidson, L. A., and Jefferson, J. M.: Encephalographic Studies in Respiratory Failure, Brit. Med. J. 2: 397 (Sept. 12) 1959.*)

MANAGEMENT OF RESPIRATORY INSUFFICIENCY Using the Mörch respirator, ten patients were treated for pulmonary insufficiency. This followed trauma in one patient, pulmonary surgery in two, and cardiopulmonary bypass in seven patients. The advantages of this apparatus is that a large and certain stroke volume is available which can produce a slight hypocapnia and permits use of a loose tracheotomy tube. Five ml. of sterile saline is instilled into the tracheotomy every thirty minutes. One patient died from a diffuse tracheobronchitis. Pulmonary hypertension was present in five of the above mentioned seven patients, and it is noted that a degree of anoxia easily tolerated by patients with a normal pulmonary artery pressure may be fatal to a patient with severe pulmonary hypertension. (*Spencer, F. C.: Use of a Mechanical Respirator in the Management of Respiratory Insufficiency Following Trauma or Operation for Cardiac or Pulmonary Disease, J. Thoracic & Cardiovasc. Surg. 38: 758 (Dec.) 1959.*)

POSITIVE PRESSURE BREATHING Dogs subjected to 18.5 mm. Hg positive pressure showed a five-to-six fold increase in venous pressure as well as a decrease in mean arterial pressure. There was a progressive decrease in circulating plasma volume as measured by the T-1824 method. A 30 per cent decrease in plasma volume was recorded after 160 minutes of increased intrapulmonary pressure. All circulatory changes returned to pre-pressure breathing levels upon release of pressure breathing. Other changes such as oliguria, periods of apnea and an alkaline urine, accompanied positive pressure breathing. This suggests that the decrease in plasma volume is the result of venostasis caused by the rapid increase in venous pressure. (*Sobel, S., Marotta, S. F., and Marbarger, J. P.: Circulating Plasma Volume Changes in Anesthetized Dogs During Positive Pressure Breathing, J. Appl. Physiol. 14: 937 (Nov.) 1959.*)

GAS DISTRIBUTION The stratification theory suggests that the main factor causing nonuniform composition of alveolar gas is that more deeply situated spaces (alveoli) receive less inspired gas than more superficial spaces (alveolar ducts). The more recent theory, series ventilation, also assumes a superficial well-ventilated space and a deeper less-ventilated space without, however, giving a definite anatomical location of these spaces. The theory of parallel ventilation states that nonuniform gas distribution is due to the occurrence of separately located spaces which are ventilated at different rates independently of one another and in parallel. These theories are discussed at some length, and it is concluded that no explanation is completely satisfactory. A series element may be present at exceptionally high breathing rates, but regional ventilation differences probably account for most of the inequalities observed under physiological circumstances. Regional ventilation differences may be caused by a number of factors, the most commonly accepted ones being unequal volume expansion and differences in time constants of lung regions. Factors changing the extent of nonuniform distribution are: age and sex; breathing frequency; lung volume; hyperventilation; added dead space; posture; and drugs. It seems likely that the distribution of ventilation and blood flow within the lungs are even more dynamic processes than is apparent from the experimental results. The hydrostatic pressure effects in pulmonary circulation appear to be compensated, to some extent, by changes in gas distribution, but this compensation is insufficient to maintain uniform ventilation-to-perfusion ratios in the erect position. (*Bouhuys, A., and Lundin, G.: Distribution of Inspired Gas in Lungs, Physiol. Rev. 39: 731 (Oct.) 1959.*)

PULMONARY SHUNTS In patients without intracardiac shunts a rise in saturation during the Valsalva maneuver and a fall following the release of the maneuver was seen. The change averaged 1.2 per cent in normal subjects and varied from 3.9 to 7.3 per cent in patients with cardiac or pulmonary disease. The rise in saturation probably results from a decrease in blood flow through pulmonary