THAM The administration of THAM to six normal adults was followed by a series of changes in ventilation, arterial blood and urine. Alveolar ventilation and oxygen saturation fell significantly, as did alveolar carbon dioxide excretion and tidal volume. Alveolar and blood carbon dioxide tension rose slightly. Urinary *p*H and electrolyte excretion increased strikingly without any change in endogenous creatinine clearances. No toxic effects were observed. The findings suggest that THAM cannot presently be recommended for clinical use in the treatment of respiratory acidosis, unless some means of stimulating respiration is also provided. (*Berman, L. B., and others: Carbon Dioxide Buffering in Man, J. Appl. Physiol. 15: 393 (May) 1960.*)

RESPIRATORY COMPLICATIONS Respiratory complications in the postanesthetic period are usually due to obstruction of major or minor bronchi. Atelectasis should be suspected before gross physical signs are demonstrated or significant shadows become apparent on a roentgenogram. Simultaneous rises in temperature, pulse, and respirations, suggesting early atelectasis, usually responded promptly to tracheal aspiration. Preoperative instruction about pulmonary exercises proved to be of great value. (*Roe, B. B.: Prevention and Treatment of Respiratory Complications in Surgery, N. Engl. J. Med. 263: 547 (Sept.) 1960.*)

PULMONARY CIRCULATION The pulmonary capillary blood volume and the diffusing capacity of the pulmonary membrane were caluculated from measurements of diffusing capacity of the lungs for carbon monoxide in normal subjects. Pulmonary capillary blood volume fell during 45 degree head-up tilt. The infusion of trimethapane in the recumbent position also decreased pulmonary capillary blood volume and accentuated its decrease during the head-up tilt. Diffusing capacity did not change significantly during these procedures. Norepinephrine did not change the pulmonary capillary blood volume in the recumbent position but led to an increase in the diffusing capacity. The decrease in pulmonary blood volume on tilting was abolished by norepinephrine. Pulmonary blood volume changed in the same direction as did the capillary transmural pressure deduced from available hemodynamic data during tilting, trimethapane infusion and exercise. This could be done to passive conformity of the capillary bed to changes in transmural pressure. Active vasomotion cannot be excluded. Pulmonary blood volume did not change although transmural pressure probably was increased during norepinephrine infusion in the recumbent position. This is best explained by active vasomotion in the pulmonary capillaries. (*Lewis, B. M., and others: Effects of Body Position, Ganglionic Blockade and Norepinephrine on Pulmonary Capillary Bed, J. Clin. Invest. 39: 1345 (Sept.) 1960.*)

DYE DILUTION Of the commonly used arterial sampling sites the femoral artery is most likely to yield a curve which approaches the curve as it emerges from the left ventricle. Variable alteration of one or more curve parameters is frequently seen when simultaneous brachial artery or radial artery curves are compared with those from the femoral artery. Distortions encountered may assume proportions which would lead to serious inaccuracy in the estimation of appearance time, mean circulation time and disappearance slope. Radio-opaque injections of femoral and brachial arteries of human subjects without arterial disease show extremely variable slope patterns in the brachial but not in the femoral artery. Whenever possible the femoral artery in humans should be the most distal sampling site used when accurate reproduction of the contour of the curve and accurate estimation of the arterial volume is required. A high degree of aortic coarctation and presumably obstructive disease of the great vessels or aneurysmal dilatation of the thoracic or abdominal aorta render the femoral artery curve invalid. (*Lange, R. L., Smith, C., and Hecht, H. H.: Arterial Blood Flow Patterns in Human Subjects and Their Effect on Indicator Dilution Curves from Various Arterial Sites, J. Clin. Invest. 39: 1413 (Sept.) 1960.*)