well controlled by shifting to the lower tourniquet after anesthesia was established, and by alternating tourniquets. Releasing the tourniquets for brief periods also gave prolonged relief. (Narcotics probably would have helped but were not used.) The next most serious problem was untoward drug reactions. Two significant systemic reactions resulted from investigational variations in dose and produced generalized twitching and obtundation without effect on circulation or respiration. Twenty-two patients had mild subjective reactions and 10 showed mild objective signs. Reactions included: sensations of warmth, tingling, dizziness, sleepiness, a sinking feeling, fearfulness, thickness of speech, inappropriate affect, and trembling (not twitching). Reactions, when present, lasted under four minutes and yielded less discomfort than would routinely be expected after general anesthesia. The only postanesthetic complication was thrombophlebitis. This subsided uneventfully in 3 of the cases followed. The follow-up also revealed enthusiasm for this technique by the patients. *Conclusions:* The place of this technique in routine extremity operations cannot be established from this short series. We believe that it is frequently the best technique in special circumstances, such as, the patient with a full stomach, the patient with severe systemic disease, or in the ambulatory patient. The minimal preparation and postoperative care required make the technique extremely valuable in the major catastrophe and military casualty situation.

Arterial Blood Gas and Acid-Base Derangements in Children with Status Asthmaticus. JOHN DOWNES, M.D., DAVID WOOD, M.D., DAVID NIGHTINGALE, M.D., and LEONARD BACHMAN, M.D., *Children's Hospital of Philadelphia and the University of Pennsylvania, Philadelphia.* Status asthmaticus is potentially fatal in infants and children, yet little published data exist describing arterial blood gas and acid-base derangements, and their therapeutic implications. The present study consists of serial arterial Pa_{O_2}, Pa_{CO_2}, *p*H, base excess and organic acid determinations in 39 acute, clinically severe, epinephrine-resistant, asthmatic episodes in 25 patients. The clinical course and arterial acid-base data distinguish two groups of patients. *Material: Group I* consisted of 10 patients (mean age 4 years) who at maximal clinical severity and breathing spontaneously had a Pa_{CO_2} exceeding 65 mm. of mercury with physical signs indicative of impending exhaustion and gross ventilatory insufficiency. All of these patients received assisted ventilation. *Group II* consisted of 15 patients (mean age 4 years) who maintained a Pa_{CO_2} below 65 mm. of mercury with spontaneous ventilation. *Results and Conclusions:* At the peak clinical severity and during $NaHCO_3$ infusion, the mean arterial acid-base status in group I was *p*H 7.16, Pa_{CO_2} 80 mm. of mercury, and base excess 4.8 mEq./liter. In group II at a comparable time, the mean values were *p*H 7.30, Pa_{CO_2} 46 mm. of mercury, and base excess 3.9 mEq./liter. Hypoxemia during air breathing was nearly always present. In 14 patients (group II) the initial mean air breathing Pa_{O_2} was 64 mm. of mercury (range 38–83 mm. of mercury), with the mean calculated $P_{A_{O_2}}$ 104 mm. of mercury, and a P_A-a_{O_2} gradient of 40 mm. of mercury. This large P_A-a_{O_2} gradient was probably related to atelectasis (proved radiographically in 60 per cent of these patients) and to maldistribution of gas within the lung. The actual magnitude of the metabolic acidosis was revealed by the initial mean base excess values prior to $NaHCO_3$ infusion: -9.5 mEq./liter (group I) and -5.2 mEq./liter (group II). Initial blood ketone levels were significantly elevated in each of 11 patients (group II) with a mean of 26 mg./100 ml. (4.5 mEq./liter), a value in the range found for normal children after 24 hours fasting. Initial serum lactate, pyruvate, and L/P ratio were within the upper normal limits (6 patients of group II). As a patient went into an epinephrine-fast state (status asthmaticus) the following sequence appeared to occur: the Pa_{O_2} fell, the initial Pa_{CO_2} was low or normal (28–43 mm. of mercury), a metabolic acidosis developed; subsequently the Pa_{CO_2} rose, the Pa_{O_2} continued to fall, the combined acidosis increased. Unless this sequence reversed with conservative management, including intravenous $NaHCO_3$, endotracheal intubation and mechanically assisted ventilation (Bird Mark VIII ventilator) were necessary to prevent fatal asphyxia. Even patients in the most extreme acidosis (*e.g.*, arterial *p*H 6.89, Pa_{CO_2} 168)

were successfully managed. Upon clinical recovery, a normal acid-base state in both groups I and II was restored within 24 to 72 hours. However, hypoxemia on air persisted up to that time with a mean Pa_{O_2} 72 mm. of mercury possibly due to residual atelectasis. *In summary,* status asthematicus in infants and children is usually associated with hypoxemia and metabolic acidosis. Varying degrees of respiratory acidosis develop subsequently in severe cases. Potentially fatal asphyxia, when it occurs, can be reversed with mechanically assisted ventilation.

Studies of the Splanchnic Circulation During Halothane Anesthesia in Man. R. M. Epstein, M.D., S. Deutsch, M.D., Ph.D., L. H. Cooperman, M.D., A. J. Clement, M.B., and H. L. Price, M.D., *Department of Anesthesiology, College of Physicians and Surgeons, Columbia University, New York City and the Department of Anesthesiology, University of Pennsylvania, School of Medicine, Philadelphia. Method:* Nine healthy male volunteers were studied in the supine position following an overnight fast. The right lobe of the liver was catheterized via an antecubital vein using a Lehman catheter and a femoral artery entered with a Cournand needle. Blood flow was estimated by measuring the hepatic extraction of infused indocyanine green. Splanchnic vascular resistance was calculated as net perfusion pressure divided by blood flow and splanchnic oxygen consumption as A-V oxygen difference multiplied by blood flow. Splanchnic blood volume was estimated using RISA. At the end of the control period, anesthesia was induced with halothane nitrous oxide-oxygen and the trachea intubated following an intravenous dose of succinyldicholine (40–60 mg.). Anesthesia was continued with 1.5 per cent halothane in oxygen. Spontaneous respirations were permitted provided that the Pa_{CO_2} remained below 45 mm. of mercury; otherwise they were assisted. The principal findings were that splanchnic blood flow was reduced during anesthesia while oxygen consumption remained nearly normal. There was no evidence for splanchnic vasoconstriction, in contrast to findings during cyclopropane administration. When hypercapnia was produced by added CO_2, vasodilatation and increased hepatic blood flow ensued. (Supported by USPHS Grants GM 09069 and GM 09070.)

Halothane and Hepatic Venous Oxygen Saturation. Ruth I. Gattiker, M.D., Alan D. Sessler, M.D., Richard O. Lundborg, M.D., and H. J. C. Swan, M.D., Ph.D., *Mayo Clinic and Mayo Graduate School of Medicine, Rochester, Minnesota.* The levels of blood oxygen saturation in the hepatic vein during halothane anesthesia in man have been studied with the intention of providing indications of relative changes in splanchnic blood flow and of defining the oxygen tension to which hepatic tissue is exposed during clinical anesthesia. *Method:* Nine patients scheduled for percutaneous renal arteriography were studied. After premedication with pentobarbital and meperidine, 2 catheters were passed under fluoroscopic control: one into the pulmonary artery, the other into a right hepatic vein. Samples for the determination of oxygen saturation were drawn from these sites. A radial artery needle was inserted and samples were drawn for oxygen tension of the systemic arterial blood. These measurements together with determinations of cardiac output were made with patients breathing air, 100 per cent oxygen, halothane in oxygen or halothane in 40 per cent oxygen and 60 per cent nitrous oxide. *Results:* The arterial oxygen tension averaged 87 mm. of mercury in patients breathing air, approximately 400 mm. of mercury with halothane in oxygen, and 170 mm. of mercury with halothane in 40 per cent oxygen and 60 per cent nitrous oxide. The mixed venous saturation averaged 76 per cent with air, 85 per cent with halothane in oxygen, and 83 per cent with halothane in 40 per cent oxygen and 60 per cent nitrous oxide. The cardiac index averaged 3.9 liters per minute per square meter of body surface in patients breathing air; it fell in 2 of 3 patients breathing 100 per cent oxygen and in all patients under anesthesia. Oxygen saturation in the hepatic vein averaged 73 per cent in air, but fell in all patients under anesthesia: to 69 per cent with halothane in oxygen, and to 50 per cent with halothane in 40 per cent oxygen and 60 per cent nitrous oxide. *Conclusions:* Our study showed that the cardiac indexes decreased in all patients given