500 ml daily for three days, and 500 ml every other day until ambulation or discharge. Positive diagnosis of pulmonary embolism was made only if confirmed at autopsy or if supported by positive lung scans. There were eight confirmed and four unconfirmed but clinically suspected instances of pulmonary embolism in the control group within a month of operation. Only one documented pulmonary embolism, seven weeks after operation and five weeks after discharge, occurred in the treated group. Total platelet count increased by 35 per cent in the control group and remained unchanged in the treated group. Platelet adhesiveness increased 13 per cent in the control group and decreased 31 per cent in the treated group, while plasma fibrinogen increased 13 per cent in the control group and changed little in the treated group. Although dextran was not ideal for prevention of pulmonary embolism, it was the best available agent. (*Atik, M., Harkess, J. W., and Wichman, H.: Prevention of Fatal Pulmonary Embolism, Surg. Gynec. Obstet. 130: 403 (March) 1970.*)

PULMONARY EDEMA To investigate possible mechanisms leading to high-altitude pulmonary edema, venous distensibility and forearm blood flow were measured in healthy human subjects at sea level and at an altitude of 11,800 feet. Venous distensibility decreased significantly upon going to altitude and increased toward normal when the subjects were given supplemental oxygen. Exercise at altitude accentuated the venoconstriction and simultaneously decreased forearm blood flow. The venoconstriction in subjects who developed pulmonary edema at altitude was significantly greater than that in those who did not. There is a shift of blood from peripheral veins to the central circulation in man exposed to high altitude, reaching a maximum three to four days following ascent. The delay in development of the maximum changes suggests a process of acclimatization rather than a simple and immediate response to hypoxia. The peripheral arteriolar constriction with exercise at altitude is opposite to the dilation which occurs with exercise at sea level. If the pulmonary vascular bed acts in a similarly paradoxical manner upon ascent, then the high resistance of the arterioles may decrease, thereby increasing capillary pressure. An alternative possibility is an increase in pulmonary venular resistance with exercise, causing capillary pressure to rise. These changes, increased pulmonary blood volume, and increased capillary permeability probably all contribute to the development of pulmonary edema. (*Wood, J. E., and Roy, S. B.: The Relationship of Peripheral Venomotor Responses to High Altitude Pulmonary Edema in Man, Amer. J. Med. Sci. 259: 56 (Jan.) 1970.*)

INTERNAL JUGULAR CATHETER The internal jugular vein was cannulated for central venous pressure measurements and transfusions in approximately 1,000 patients. With the patient in the Trendelenburg position and the head turned to one side, a no. 14 Venocath needle (with a 2-ml syringe attached) was inserted into the skin two fingerbreadths above the clavicle at the outer border of the sternomastoid muscle. The needle was directed toward the suprasternal notch and puncture of the vein wall verified by easy aspiration of dark blood. The syringe was then removed and the Venocath threaded into the vein and fixed securely to the skin. Only three complications, all nonfatal, occurred in this series. These were air embolism, thrombophlebitis due to staphylococcal cellulitis at the site of the puncture, and perforation of the vein with mediastinal infusion of electrolytes. It was felt that these complications were avoidable. (*Jernigan, W. R., and others: Use of Internal Jugular Vein for Placement of Central Venous Catheter, Surg. Gynec. Obstet. 130: 520 (March) 1970.*)

POSTOPERATIVE HYPERTENSION Hypertension after operative correction of aortic coarctation may occur immediately after surgery and persist for about 36 hours. A delayed unexpected ("paradoxical") unexpected hypertension may appear two to three days later and last about two weeks. Much less frequently, severe abdominal pain occurs, which on occasion has led to laparotomy or has caused death from necrotizing arteritis. Eighty patients underwent operations for correction of aortic coarctation. They ranged in age from

3 to 40 years. Blood pressures during the operations were regulated with trimetaphan. Seventeen patients incurred "paradoxical" hypertension in the early postoperative period. Twenty-three patients developed the delayed type of hypertension, which lasted several weeks. Abdominal symptoms were noted in 12 patients, nine of whom had associated hypertension. Reactive hypertension and abdominal symptoms occurred mostly in patients with severe stenosis and poor collateral circulation. There was no early postoperative mortality in the 80 patients. Five patients died five to seven months after surgery; four of these had significant postoperative hypertension. Several mechanisms may explain the pathogenesis of these syndromes. The early hypertension may be secondary to the decrease in pressure which occurs in the aortic pressure-receptor area after the stenosis has been corrected. Another mechanism may be postulated on the basis of development of a Goldblatt kidney secondary to ischemia during operation or following injury of the renal arterioles by too-rapid release of the aortic clamp. Indeed, in the cases in which the patients had the abdominal pain syndrome necropsies showed tears of the intima of many arterioles of the splanchnic circulation. It is recommended that the aortic clamp be released slowly, and that the blood pressure in the lower extremities be monitored. If a rapid rise in blood pressure occurs, re-clamping may be mandatory. The use of ganglionic blocking drugs is advised until the tendency toward increased total peripheral resistance has regressed. *(Seidel, W., Borst, H. G., and Martin, C.: Paradoxical Hypertension and Abdominal Pain Syndrome as Possible Sequelae of Surgical Correction of Aortic Coarctation, Thoraxchirurgie, 18: 84 (Feb.) 1970.)*

Respiration

LUNG IRRITANT RECEPTORS Lung irritant receptors were studied in rabbits by recording action potentials from single vagal nerve fibers. Some animals were bilaterally vagotomized and some paralyzed and artificially ventilated. The receptors produced rapidly-adapting irregular discharges on inflation and deflation of the lungs. Many were stimulated by insufflation of ammonia vapor into the lungs, and some by passage of a fine catheter into the right bronchial tree. The receptors were strongly stimulated by intravenous injections of histamine. The response to histamine was reduced by the prior injection of isoproterenol, which also reduced the bronchoconstriction due to histamine. The receptors were stimulated by intravenous injections of isoproterenol and microemboli and by anaphylaxis induced in rabbits previously sensitized to egg albumin. Receptor responses could not be closely correlated in size with simultaneous changes in total lung resistance, lung compliance, tidal volume or breathing frequency. In rabbits with intact vagi, lung irritant receptors contributed to the reflex hyperpnea and bronchoconstriction seen in the conditions studied. *(Mills, J. E., Sellick, H., and Widdicombe, J. G.: Activity of Lung Irritant Receptors in Pulmonary Micro-embolism, Anaphylaxis and Drug-induced Bronchoconstriction, J. Physiol. (London) 203: 337 (Aug.) 1969.)*

IRRITANT RECEPTORS The activity of lung irritant receptors was studied in the rabbit by recording from single vagal nerve fibers. Receptors were stimulated during induction and removal of a pneumothorax. Pneumothorax caused greater depressions of minute volume in bilaterally vagotomized rabbits than in those with intact vagi. Hyperpnea due to breathing through an added deadspace increased receptor discharge. Experiments on paralyzed and artificially ventilated rabbits showed that this was not a direct action of the asphyxial changes in blood gas tensions. Pulmonary congestion, induced by inflating a balloon in the left atrium, stimulated the receptors in paralyzed, artificially ventilated rabbits. The evidence that receptors cause vagal reflex hyperpnea and bronchoconstriction and that they are responsible for the reflex ventilatory and bronchomotor changes in the conditions studied is reasonable. *(Sellick, H., and Widdicombe, J. G.: The Activity of Lung Irritant Receptors during Pneumothorax, Hyperpnoea and Pulmonary Vascular Congestion, J. Physiol. (London) 203: 359 (Aug.) 1969.)*