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.*)