creasing sharply when the maximal breathing capacity exceeded 50 per cent of predicted. (*Leiner, G. C., and others: Prognostic Significance of the Maximal Breathing Capacity in Cardiopulmonary Diseases, Amer. Rev. Resp. Dis. 87: 545 (Apr.) 1963.*)

ACID–BASE BALANCE A new method is described for monitoring acid-base data during anesthesia. The infrared analyzer is used for measuring alveolar and arterial carbon dioxide concentrations. The total carbon dioxide content of arterial blood is determined by adding lactic acid and measuring the expelled carbon dioxide. Standard bicarbonate is then calculated as in the van Slyke procedure. (*Oehmig, H., and Sommerkamp, H.: A New and Simple Method of Analysis and Control of the Acid Base Balance, Der Anaesthesist 12: 179 (June) 1963.*)

NORADRENALINE STORES On the basis of the effects of guanethidine and reserpine on the dog heart-lung preparation, there are two distinct stores of norepinephrine in the heart. One is situated adjacent to the effector cells and the other at a distance requiring transport by the blood stream to reach its target. The first (smaller) store is probably depleted by guanethedine, the second by reserpine, although both may be depleted by either drug if sufficient time is given. (*Fawaz, G., and Simaan, J.: Cardiac Noradrenaline Stores, Brit. J. Pharmacol. 20: 569 (June) 1963.*)

DICHLOROISOPROTERENOL Uncontrolled cardiac arrhythmia is one of the most important operative complications in the surgical treatment of pheochromocytoma. Dichloroisoproterenol hydrochloride protects the myocardium to some extent when excessive amounts of epinephrine and norepinephrine are released. (*Riddell, D. H., and others: Experience with Pheochromocytoma in 21 Patients —Use of Dichloroisoproterenol Hydrochloride for Cardiac Arrhythmia, Ann. Surg. 157: 980 (June) 1963.*)

PRESSOR RESPONSIVENESS At least four mechanisms contribute to the increased responsiveness to pressor drugs caused by ganglioplegic agents: (1) Elimination of parasympathetic reflexes. (2) Elimination of sympathetic compensatory reflexes. (3) Direct effect on blood vessels by the ganglioplegic agent resulting in sensitization similar to that produced by surgical denervation. (4) Blocking action of the ganglioplegic agent on adrenergic vasodilator receptors. Because of the participation of these different mechanisms, ganglioplegics and surgical denervation do not necessarily affect pressor responsiveness in the same manner. Also, the different mechanisms involved account for the various degrees of augmentation of response to different pressor drugs, since the latter have their major actions on different portions of the cardiovascular system. (*Page, I. H., and McCubbin, J. W.: Mechanisms by which Ganglioplegics and Atropine Enhance Cardiovascular Responsiveness, Amer. J. Physiol. 205: 1 (July) 1963.*)

POSTOPERATIVE DIURESIS Following surgery under general anesthesia, patients who do not excrete hypotonic urine after a water load respond to acute expansion of extracellular fluid volume with saline by excreting solute-free water. Acute expansion of extracellular fluid volume in postoperative patients leads to suppression of high anti-diuretic hormone activity normally seen during this period. (*Wright, H. K., and Gann, D. S.: Correction of Defect in Free Water Excretion in Postoperative Patients by Extracellular Fluid Volume Expansion, Ann. Surg. 158: 70 (July) 1963.*)

NETHALIDE Nethalide, an adrenotropic beta receptor antagonist, greatly decreases the sensitivity of the myocardium to epinephrine-induced arrhythmias in unpremedicated dogs anesthetized with cyclopropane, trichlorethylene, or halothane. Multiple ventricular ectopic beats were produced only after 4 to 15 times the minimum control dose of epinephrine was administered. Ventricular fibrillation did not occur with even large doses of epinephrine after nethalide—except in two of three dogs under halothane anesthesia. (*Murray, W. J., and others: Antagonism of Hydrocarbon Anesthetic-Epinephrine Arrhythmias in Dogs by Nethalide, Proc. Soc. Exp. Biol. Med. 113: 439 (June) 1963.*)