7 minutes. The mean of the percentage flow rates for all infusions at a given dose of noradrenalin was not less than 100 per cent of controls in either foot until 0.8 μg./minute was administered, when flow was reduced in both warm and cool feet. Thus, cooled vessels were not found to be more sensitive than warm vessels. Noradrenalin infused at 3.2 μg./minute caused a significantly greater reduction in flow rate in the cool than in the warm feet, but this was not as a greater reactivity of the cooler vessels to the drug. *(Gaskell, P., and Bray, G. M.: Effect of Local Cooling on the Sensitivity to Noradrenalin of Vessels in the Feet, Canad. J. Physiol. Pharmacol. 45: 75 (Jan.) 1967.)*

LIMB BLOOD FLOW Peripheral vascular effects and mechanisms of action of cyclopropane and halothane were investigated in both innervated and denervated dogs' forelimbs perfused under a variety of experimental conditions. Both agents caused vasodilatation in the forelimbs which tended to parallel concentration of inspired gas. This response was not abolished by denervation and hence was not totally dependent on vasomotor nerves. Vasodilatation in the dog forelimb during cyclopropane and halothane administration may be mediated indirectly through a change in blood concentration of some unidentified vasoactive agent. A direct vascular action of halothane could not be conclusively ruled out. *(Emerson, T. E., and Massion, W. H.: Direct and Indirect Vascular Effects of Cyclopropane and Halothane in the Dog Forelimb, J. Appl. Physiol. 22: 217 (Feb.) 1967.)*

CIRCULATORY RESPONSES TO CO_2 In anesthetized dogs, breathing of 10 per cent CO_2 caused peripheral vasoconstriction, increased arterial pressure and decreased cardiac output. Following administration of hexamethonium, peripheral vasodilatation, decreased arterial pressure and increased cardiac output occurred during CO_2 breathing. In dogs treated with both propranolol, a beta adrenergic blocking agent, and hexamethonium, vasodilatation and increased cardiac output still occurred in response to CO_2. These findings suggest that peripheral vasodilatation and increased cardiac output occurring during CO_2 breathing are not mediated through stimulation of beta adrenergic receptors. *(Wendling, M. G., Eckstein, J. W., and Aboud, F. M.: Cardiovascular Responses to Carbon Dioxide Before and After Beta-Adrenergic Blockade, J. Appl. Physiol. 22: 223 (Feb.) 1967.)*

ULTRASONIC FLOWMETER An ultrasonic flowmeter based on the Doppler effect has proved useful in the evaluation of peripheral vascular disease. An ultrasonic beam of 5 megacycles per second generated by a piezoelectric crystal is passed through the intact skin over the vessel being studied. Sound back scattered from stationary objects has the same frequency as the generated sound while sound from moving blood is reflected at a different frequency. A second crystal is used for detection of the reflected sound waves. Frequency changes produced by the moving blood are proportional to the velocity of flow. Arterial flow signals have two components, a first high frequency and high intensity component corresponds to systolic flow while a lower frequency component reflects the normal transient reversal of flow in peripheral arteries during diastole. Venous flow is continuous but varies with respiration; venous flow in the legs being temporarily halted during inspiration while venous arm flow is decreased during expiration. Changes in flow over stenotic or occluded areas of vessels can be readily detected with this flowmeter. This method of flow detection has obvious advantages over other flowmeters in that it is not necessary to expose or catheterize the vessels being studied. Unfortunately, it is not possible to quantitate the changes in flow with this instrument. *(Strandness, D. E., Jr., and others: Ultrasonic Flow Detection, J. Surg. 113: 311 (March) 1967.)*

ABSTRACTOR'S COMMENT: This instrument might prove useful in measuring changes in flow after sympathetic block.

MONITORING A monitoring system is used to acquire, record and display data and should not be considered as a replacement for the physician but as an addition to his armamentarium. Central venous pressure serves as