tion occurring when cerebral venous oxygen tension falls to about 28 mm. Hg (as in arterial hypoxia). With the exception of skin and pulmonary vessels, most other vascular beds are likewise dilated, the vasodilatation occurring when venous oxygen tension falls below a critical level for that organ—and regardless of arterial oxygen tension. Thus, local vasodilatation seems to be due to some alteration in cellular metabolism. Analysis of the various factors operative during arterial and primary tissue hypoxia (cardiac performance, humoral mechanics, chemoreceptors and other reflex mechanisms) gives but little positive information about the mechanisms of circulatory control during hypoxia. The cardiac response is nonspecific and closely related to the pattern of peripheral vasomotor tone. Regulation by adrenaline is not an essential part of the response to hypoxia. The chemoreceptors, of dominating importance in the respiratory response to arterial hypoxia, have slight circulatory effects. Most of the circulatory changes in hypoxia can be related to changes in tissue oxygen tension. (*Korner, P. I.: Circulatory Adaptations in Hypoxia, Physiol. Rev. 39: 687 (Oct.) 1959.*)

MYOCARDIAL ELECTROPHYSIOLOGY A normal recording from the sinoatrial node reveals a progressive loss of the transmembrane potential until the firing or threshold level is reached. This diastolic loss of potential is due to the natural incapacity of pacemaker cells to maintain intact impedence during diastole. Clinical tachycardia or premature systoles may be due to a cell or group of cells somewhere in the heart which, either normally or as a result of disease, displays a similar dissipation of the transmembrane potential during diastole. Block may be caused by a slowing or failure of depolarization in ventricular cells (*e.g.*, potassium slows rise time of depolarization, and a high serum concentration can prevent the action potential completely). The effects of digitalis are opposed by potassium and augmented by calcium. It is suspected that on some occasions where cardiac arrest has occurred in the operating room and cardiac massage instituted along with the injection of calcium, the result was unfavorable because it was forgotten that the patient was digitalized. The known additive effects of calcium and digitalis have led to the development of two clinical tests. The first is the intravenous infusion of a chelating agent (disodium ethylenediamine tetracetate, 15 mg./min.) to a patient who has the cardiac manifestations of digitalis toxicity. Disappearance of the arrhythmia serves both to demonstrate that it was indeed due to digitalis and to terminate a potentially dangerous rhythm. The second is the use of calcium as a test of degree of digitalization (10 per cent calcium chloride given over a period of 20 minutes or until evidence of toxicity appears). Potassium also is used clinically in eliminating or decreasing the toxic effects of digitalis. The effects on the electrocardiogram of high and low potassium and serum calcium are discussed. Molar sodium lactate has not been studied at the membrane level, but it would seem that the change in *p*H of the perfusion milieu must have effects on the impedance of the myocardial membrane and the gradient of ions that exists across it. (*Kossmann, C. E.: Some Clinical Aspects of the Bioelectrics and the Electrochemistry of Myocardium, Bull. New York Acad. Med. 36: 3 (Jan.) 1960.*)

Reviewer's note: This article may perhaps be coupled with that by Hoffman, B.: Electrophysiology of Single Cardiac Cells, loc. cit. 689 (Nov.) 1959. It was presented at the same scientific session and really is a clinical companion piece to it.

MYOCARDIAL PHYSIOLOGY By catheterizing the coronary sinus, chemical comparisons can be made between the venous and the arterial blood in the coronary vessels of the human heart in situ. Metabolic studies by this technique have demonstrated the importance of fatty acids in the nutrition of the myocardium. By the introduction of microelectrodes into the interior of single cells of the myocardium, the atrioventricular node, and the ventricular conducting system, it is possible to follow electric changes and ionic fluxes in small units. Such studies indicate that resting potential probably results from the difference in potassium concentration within and outside the heart muscle cell; the different phases of the action potential, on the other hand, are due to inward movement of sodium

during the phase of depolarization, while repolarization may result from an outward flux of potassium. Regarding pharmacology, these studies further indicate that digitalis preparations have no effect on myocardial oxygen consumption. However, since they increase the work of the failing heart, they elevate myocardial efficiency. Norepinephrine and epinephrine, on the other hand, lower myocardial efficiency by increasing myocardial oxygen consumption. (*Bing, R. J., and others: Physiology of the Myocardium, J. A. M. A. 172: 438 (Jan. 30) 1960.*)

MYOCARDIAL INFARCTION Fifty individuals underwent 64 major surgical procedures at least four weeks after their infarction. There were 43 men and 7 women with ages of 46 to 87 years. Six men died within 48 hours after operation. Myocardial infarction after four weeks of treatment need not prohibit major surgery. Such a patient must be guarded against fear, hypoxia, and falls in blood pressure. (*Weiss, M. M., Sr., and Weiss, M. M., Jr.: Risk of Major Surgery in Patients with Old Myocardial Infarction, Surgery 46: 1094 (Dec.) 1959.*)

CARDIAC WORK The dog heart arrested with potassium citrate after digitalization, is able to perform more than 19.5 gram-meters stroke work in response to loading by transfusion. The heart of a nondigitalized dog of comparable size under similar loading conditions, in contrast, is unable to exceed 9.1 gram-meters stroke work in the period after arrest. Predigitalization is beneficial to the canine myocardium subjected to 30 minutes of arrest with potassium citrate. (*Cooper, T., and others: Effect of Prophylactic Digitalization on Myocardial Function After Elective Cardiac Arrest, Ann. Surg. 151: 17 (Jan.) 1960.*)

CARDIAC OUTPUT A new method for the determination of cardiac output every 5–8 seconds was developed in the dog. Injection of 0.5 mg. of a tricarbocyamine dye into the left atrium of anesthetized dogs every 5–8 seconds (phased with respiration) results in reproducible dye-dilution curves during cardiovascular equilibrium. Variations in determinations do not exceed ∓ 5 per cent. (*Opdyke, D. F., and Sniffen, R. E.: Estimation of Cardiac Output by Rapidly Repeated Dye-Dilution Technic, Proc. Soc. Exp. Biol. & Med. 102: 725 (Dec.) 1959.*)

PULMONARY EDEMA When pulmonary edema is due to low cardiac output, intravenous morphine, oxygen by mask, rotating tourniquets, phlebotomy, mercurial diuretics and intravenous Cedilanid are indicated. If, however, pulmonary edema is associated with high output failure, as in thyrotoxicosis, then the treatment of the primary disease is most important, and digitalis might even be harmful. (*Tiffany, F. B.: Diagnosis and Treatment of Cardiac Emergencies, J. Lancet 80: 17 (Jan.) 1960.*)

BLOOD FLOW Circulation through extremities varies morphologically and functionally so that it may be erroneous to classify drugs and surgical procedures as general dilators or general constrictors. Careful choice of procedures or agents is necessary to treat circulatory insufficiency. Vasodilation therapy may actually diminish blood flow to compromised parts if it also has a specific effect on segments of tissue that have normal blood flow by diverting blood away from areas of insufficient circulation. Increased blood flow through tissue is not necessarily associated with increased nutrition of tissue, as in the case of increased blood flow to the skin via non nutritive arteriovenous channels. Non-nutritive arteriovenous channels may also exist in skeletal muscle. Clinical examples are shown that demonstrate diversion of blood flow through muscle at the expense of skin, diversion from one skin area to another, and diversion from diseased tissues to vasodilated normal tissues. In addition to mechanical factors such blood flow diversions may also be created on basis of differential neural responses of blood vessels of various tissues to the usual methods of producing vasodilatation or constriction. The evidence shown here clearly demonstrates that attempts to relieve circulatory insufficiency may in fact aggravate it to the point of gangrene if procedures and agents are not rationally chosen. (*Hyman, C., and Winsor, T.: Blood Flow Redistribution in the*