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