haps droplets about 3 mm in diameter of molten metal (that thus yield low emissivity and only at radio wavelengths $\lesssim$ 3 mm) develop in the surface layers on the hot side, and then on the cold side freeze into a material with normal emissivity; if so, observations 1 and 2 mm also should give an anomalous phase curve. It is difficult to imagine an atmosphere that would strongly absorb the 3-mm emission from the surface, but not the infrared emission, and also remain cold when the surface was hot. Independent confirmation of these results at 3 mm and at nearby wavelengths is obviously desirable.

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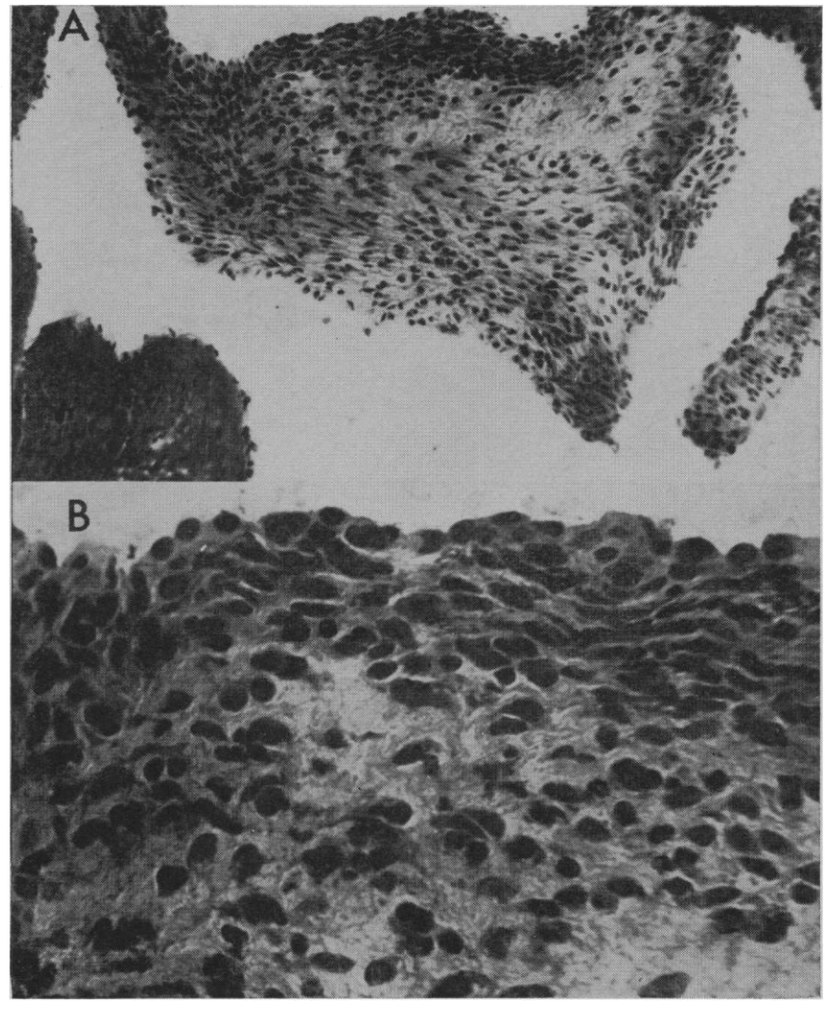

Fig. 1. (*A*) Verrucous lesion on the ventricular surface of the mitral valve of a mouse infected with Coxsackie virus B_4; hematoxylin and eosin staining (about $\times$ 110). (*B*) Portion of the lesion (*A*), showing round-cell infiltration, edema, and endothelial proliferation; hematoxylin and eosin staining (about $\times$ 440).

Endocarditis in Mice Infected with Coxsackie Virus B_4

Abstract. *Endocarditis has not been generally considered to be a complication of viral infection. We show that mural and valvular endocarditis can be produced in mice infected with Coxsackie virus B_4. Because this virus commonly infects man and is highly cardiotropic, it is important to know whether it produces valvular lesions in man similar to those we describe in mice.*

Despite occasional references to viral endocarditis in the medical literature (*1*), cardiologists and pathologists have not considered endocarditis to be a complication of viral disease. Nevertheless, it is well known that B-group Coxsackie viruses produce myocarditis and pericarditis in man (*2*). Detailed studies of the endocardia of patients dying of Coxsackie myocarditis have not been reported. The endocardium has been described as normal in mice having experimental Coxsackie-virus-B_1 myocarditis (*3*). However, endocardial lesions (including valvulitis) have been produced in experimental animals with two other cardiotropic viruses, namely encephalomyocarditis virus (*4*) and virus III (*5*). Recently Lou, Wenner, and Kamitsuka (*6*) found mitral valvulitis in two of nine cynomolgus monkeys infected with Coxsackie virus B_4. Autopsy of two cynomolgus monkeys infected with Coxsackie virus B_4 in our laboratory demonstrated mitral valvulitis in one and aortic valvulitis in the other. These studies prompted reinvestigation of the endocardia of mice infected with Coxsackie virus.

Forty HaM/ICR mice were inoculated intraperitoneally with 0.1 ml of monkey-kidney culture fluid containing Coxsackie virus B_4 (*7*). The mice, varying in age from 2 to 21 days, were killed 2 to 60 days after inoculation. Thirty-six mice, 12 to 20 days old, were inoculated intraperitoneally with 0.1 ml of virus-free monkey-kidney culture fluid; these controls were killed 5 to 20 days after inoculation.

Histologic evidence of valvular endocarditis was found in 55 percent and mural endocarditis in 50 percent of the animals inoculated with the Coxsackie virus B_4. Valves implicated were the tricuspid in 43 percent of the mice, the mitral in 23 percent, the aortic in 10 percent, and the pulmonic in 5 percent. The mural endocarditis affected the right ventricle in 20 percent of the animals, the right atrium in 18 percent, the left ventricle in 3 percent, and the left atrium in 10 percent.

The valvular lesions consisted of endothelial proliferation, round-cell infiltration, and edema of the valve (Fig. 1). The histologic characteristics of the valvular lesions were quite uniform, without variations that could be attributed either to age at the time of inoculation or to the time between inoculation and death. No histologic evidence of mural or valvular endocarditis was found in any of the control animals.

Coxsackie virus B_4 was recovered from the hearts of the infected animals as late as 8 days after inoculation. Viral antigen was identified in the valves and mural endocardium by immunofluorescent techniques.

We have shown that Coxsackie virus B_4 produces acute valvulitis in mice. Apparently such lesions have been overlooked previously because of lack of interest in the endocardium (*8*). It remains to be seen whether Coxsackie virus can produce chronic valvulitis, with scarring and calcification.

Endocarditis is not considered a complication of viral disease in man (*9*). Nevertheless, clinicians are well aware that many patients with acute and chronic valvulitis have no history of rheumatic fever, bacterial endocarditis, or syphilis. Because group-B Coxsackie viruses commonly infect man, are highly cardiotropic, and produce acute valvulitis in other mammals, it is important to determine whether they produce lesions in man similar to those we describe in mice.

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13 October 1965

Atlantic Deep-Sea Stratigraphy: Extension of Absolute Chronology to 320,000 Years

Abstract. *Thorium-230 measurements on a core of globigerina ooze from the Caribbean Sea substantiate the prediction of Ericson* et al. *that the paleontological boundary U-V (Sangamon-Illinoian boundary in their scheme) in the Atlantic sediments has an age of close to 320,000 years. As the ages derived by Ericson* et al. *were based on extrapolations of mean sedimentation rates established by carbon-14 and protactinium-231 dating of the upper sections of this and other cores, this result confirms the assumption that sedimentation rates in the Caribbean Sea have not changed significantly during the past several hundred thousand years. The uranium content of the ocean as indicated by the deposition rate of thorium-230 was no more than 30 percent higher during glacial than during interglacial periods.*

Study of the planktonic foraminiferans in the Atlantic deep-sea sediment has provided stratigraphic markers as well as evidence of climatic changes in the past (*1–5*). Two methods of study have been extensively explored: Ericson *et al.* (*3, 4*) use the absolute abundance of *Globorotalia menardii* as an indicator of the surface temperature of the ocean, and Emiliani (*2, 5*) uses primarily data on oxygen isotopes and relative species abundances. Although the climatic curves constructed by the two approaches agree only down to the bottom of the W zone of Ericson *et al.* (*5, 6*), and their interpretations in terms of continental glacial sequences await clarification (*6, 7*), the specific and isotopic changes observed in a sizable number of cores taken from the Atlantic Ocean and connected seas are internally consistent and correlatable from core to core. Hence, the establishment of radiometric ages for any one core provides an absolute chronology which can be extended throughout the Atlantic Ocean. Paleontological zones have been designated in

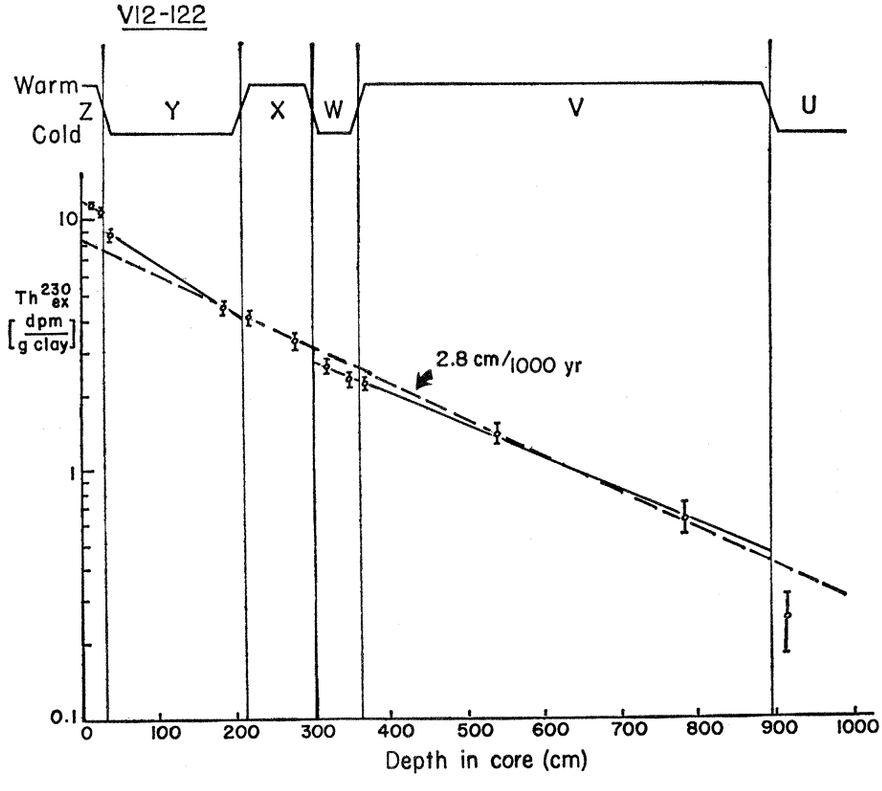

Fig. 1. Activities of unsupported Th^{230} on a basis of unit weight of noncarbonate material as a function of depth. Analytical errors are represented by vertical bars. Zonal divisions are the work of Ericson *et al.* (*4*). In the calculation of the rate, a half-life of 75,200 years for Th^{230} is used (*19*).