products growing continuously at about 1 mm/year. This rate is sufficient to produce a molten layer 10 km thick in only 10×10^6 years. Dehydration reactions would yield product columns growing at comparable or even greater rates. Such production rates are, of course, unacceptable; the conclusion remains that a concave-upward geotherm could be of only an extremely transient nature. An effective heat sink caused by a descending diapir is also transient, a priori.

Hence, the lower part of the paleogeotherm inferred from petrological data must relate to a transitory tectonic event, immediately preceding surface emplacement [for example, see (*2*)], and not representative of any steady evolutionary development in southern Africa or any other Precambrian shield. The frequency of such events may be estimated by the frequency of occurrence of kimberlite pipes, that is, a few events per 10^6 km^2 per 10^7 years. It is not surprising that these events should fail to have been reflected in earlier thermal models.

Alternatively, it seems reasonable that the partition of aluminum and calcium between coexisting pyroxenes might depend upon shear strain, as well as upon pressure and temperature, through distortion of the crystal field. If so, then the pressures and temperatures (*1*) for the asthenosphere would be in doubt. However, I am not aware of any work which establishes the existence or importance of such an effect. Further discussion of petrologically determined temperatures is contained in a recent review (*5*) of a special session of the American Geophysical Union.

Finally, a long-lived *downward* inflection of the geotherm at the top of the asthenosphere is easily understood as a sudden increase in F_{conv} (corresponding to the onset of convection) with a corresponding decrease in dT/dz, maintaining a near-constant total flux. Similarly, an upward inflection at the bottom of the convecting zone (*6*) is a reflection of the disappearance of F_{conv} upon passage from "fluid" to solid, with an increase of the thermal gradient necessary for the near-equality of heat flow.

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24 October 1974; revised 28 January 1975

Erythrocytes in Human Muscular Dystrophy

The observations of Miale *et al.* (*1*) and Matheson and Howland (*2*) appear to represent a conflict with respect to the adequacy of scanning electron microscopic analyses of erythrocytes in detection of heterozygote carriers of Duchenne muscular dystrophy. Miale *et al.* stated that they were unable to detect heterozygotes in their study; Matheson and Howland (*3*) replied that an inadequate sample was employed and the methods of cell preparation may be critical. The latter cited contributions from our laboratory, presented in part at the Third International Congress on Muscle Diseases, as evidence in favor of the diagnostic value of scanning electron microscopy (SEM). However, the citation was somewhat misleading.

We agree that the methods of preparation are critical. It is apparent from the work of Miale *et al.* as well as our own that Matheson and Howland's method does not give easily reproducible results. The data we presented at the congress do not exactly confirm their results. With rigidly defined conditions in which the cells are unwashed and fixed immediately in solutions controlled with respect to *p*H, ionic strength, and osmolarity, we were able to demonstrate stomatocytic changes in Duchenne carrier erythrocytes but unable to demonstrate the echinocytic changes described by Matheson and Howland. Furthermore, the stomatocytic shapes were not specific for Duchenne dystrophy or the carrier state, but were noted in other muscular disorders including myotonic muscular dystrophy, limb-girdle dystrophy, and myotonic congenita, as well as in a person with no clinical evidence of muscle disease (*4*).

Thus, despite our findings of altered erythrocyte morphology in Duchenne carrier states, we cannot agree with Matheson and Howland that SEM can be used to detect the carrier state. The changes observed by SEM, which are obviously in vitro artifacts produced by the fixation procedure, are insufficient to establish or confirm the diagnosis, although they do implicate a subtle membrane defect. Other data support the expression of the metabolic defect in membranes of many different tissues, especially in myotonic muscular dystrophy (*5*). When combinations of these metabolic alterations are used in conjunction with the clinical state, the detection of carriers is made more definitive. We agree that the use of erythrocytes as a model is an important approach to the study of various muscular dystrophies that have widespread expression in membranes from different tissues. However, we do not believe that any single parameter is sufficiently specific at the present time to be used as a diagnostic tool.

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10 February 1975