To the Editor: Tissue perfusion sufficient for adequate cellular oxygenation depends, in part, on optimal erythrocyte (RBC) rheology (i.e., RBC deformability and RBC aggregation). Even a small adverse hemorheological change magnifies blood flow resistance and leads to serious health consequences (1). Both Connes et al. (2) and Le Gallais et al. (5) presumably would agree that the literature contains ample evidence confirming that significant alterations in erythrocyte rheology occur in sickle cell trait (SCT) carriers; some suggest that SCT subjects might be prone to vascular alterations, cardiac ischemia, and arrhythmias leading to sudden death, particularly when exacerbated by heat stress and dehydration. Yet strenuous exertion is not the only activity causing cellular oxygen deprivation in SCT individuals. Sleep poses a risk also. According to Connes et al. (3), autonomic nervous system (ANS) activity impairment (determined by heart rate variability parameters) correlated significantly with abnormal hemorheological values (elevated blood viscosity and RBC rigidity) during sleep in SCT subjects, but not in control subjects (without SCT). This phenomenon, in SCT individuals, could represent the existence of an autoregulatory mechanism to compensate for elevated blood viscosity; such mechanisms have been hypothesized (4, 6). With hemorheopathy (abnormal blood flow mechanics) now identified in SCT individuals during the widest of life’s activities, from sleep to strenuous exercise, the disease-inducing potential of sickle cell trait during physical activity should be self-evident. It should NOT be considered an asymptomatic benign condition during either sleep or physical activity.

REFERENCES


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