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Resumo	Voiding abnormalities are common among the sickle cell disease (SCD) population, among which overactive bladder (OAB) syndrome is observed at rates as high as 39%. Although detrusor overactivity is the most common cause of OAB, its molecular pathophysiology is not well elucidated. The nitric oxide (NO) signaling pathway has been implicated in the regulation of lower genitourinary tract function. In the present study, we evaluated the role of the NO signaling pathway in voiding function of transgenic SCD mice compared with combined endothelial and neuronal NO synthase gene-deficient mice, both serving as models of NO deficiency. Mice underwent void spot assay and cystometry, and bladder and urethral specimens were studied using in vitro tissue myography. Both mouse models exhibited increased void volumes; increased nonvoiding and voiding contraction frequencies; decreased bladder compliance; increased detrusor smooth muscle contraction responses to electrical field stimulation, KCl, and carbachol; and increased urethral smooth muscle relaxation responses to sodium nitroprusside compared with WT mice. In conclusion, our comprehensive behavioral and functional study of the SCD mouse lower genitourinary tract, in correlation with that of the NO-deficient mouse, reveals NO effector actions in voiding function and suggests that NO signaling derangements are associated with an OAB phenotype. These findings may allow further study of molecular targets for the characterization and evaluation of OAB.
Fomento	