Mutations in spastin are the most common cause of hereditary spastic paraplegia (HSP), so knowledge of the functions of the spastin protein is critical to understand the pathogenesis of HSP. In this talk I discuss the endosomal roles of spastin, showing unpublished data that spastin is required for the correct morphology of an endosomal compartment, and for the correct trafficking of receptors through this compartment. I present results on the role of different ESCRT-III proteins in recruiting spastin to endosomes. I also discuss how these endosomal functions of spastin could be involved in causing axonopathy.