Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. Genetic differences have been demonstrated to modulate the risk of developing AF. Nonetheless, the majority of AF cases are sporadic, without evidence of familial disease. Recent work has suggested that genetics may still mediate arrhythmia development among sporadic cases secondary to somatic mutations. A somatic mutation, in contrast to a germline mutation, develops following fertilization and becomes confined to a subgroup of cells within the body. Restriction of mutations to a subgroup of organs or cells within an organism is referred to as mosaicism. Atrial mosaicism has been hypothesized as a potential cause of regional structural and electrical heterogeneity that can serve as an ideal substrate for the initiation and maintenance of AF. Based on promising results from the sequencing of two candidate genes, investigators have hypothesized that somatic mutations may represent a common cause of sporadic AF among younger, otherwise healthy individuals. Our study sought to characterize the burden of somatic mutations within the atria. Using a very sensitive form of next generation sequencing, we sequenced 560 genes (nearly 3 million base pairs) within atrial tissue and blood from 35 individuals (26 with atrial fibrillation and 9 controls). We found somatic mutations to be exceedingly rare, with indistinguishable mutation rates for cases and controls. Our findings suggest that atrial somatic mutations are unlikely to represent a common cause of sporadic AF.