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Drug-induced changes in cardiac function is a common cause of compound attrition. e level of contractility of the cardiac muscle is also referred to as its inotropic state, while the rate of contraction can be referred to as its chronotropic state. Furthermore, the ability of a cardiac muscle to relax following peak contraction is referred to as its lusitropic state. e level of cardiac contractile state directly contributes to cardiac output and therefore the maintenance of blood perfusion throughout the body. A decrease in cardiac contractility reduces perfusion to vital organs, while drug-induced increases may lead to increased workload and oxygen demand, both of which can lead to increased mortality. e signi cance of adverse drug e ects on cardiac contractility is o en dependent on the safety margin. Available in vitro assays are poorly predictive of the concentrations that may a ect contractility in humans and furthermore, current in vivo contractility measurements are o en invasive, resource/time consuming, low throughput and indirect. erefore, there is a need for more predictive in vitro assays that have a higher throughput than available assays. We have demonstrated that the Work Loop cardiac contractility assay is more predictive of human ndings than existing assays. We have also recently demonstrated that the Work Loop cardiac contractility assay is highly predictive of inotropy risk to man. Recently we have expanded this investigation to determine whether the cardiomyocyte Work Loop assay had