Heart failure (HF) is a complex clinical syndrome including several bidirectional feedback interactions between the heart and other organs, including the brain. The impaired cardiac function affects cerebral structure and functional capacity, whereas neuronal signals impact on the heart and the vessels. These bidirectional heart-brain interactions contribute to the development of the HF phenotype and affect many HF comorbidities of HF. The term cardiocerebral syndrome refers to a state of cognitive impairment of undefined cause in HF patients, beyond that anticipated in age-matched controls, and typically accompanied by anatomic brain changes. Improved cardiac performance and systemic hemodynamics have a positive impact on the brain. Conversely, overactivity of the sympathetic limb of the autonomic nervous system is the main process leading to the development of major cardiac pathologies in neurological disorders. A typical example is the Takotsubo syndrome (TTS), an increasingly recognized type of non-ischemic cardiomyopathy. Although the pathogenesis of TTS remains unclear, a complex interaction between catecholamine-mediated stimulation, myocardial stunning, and subsequent stress-related myocardial dysfunction seems to be the main pathophysiological mechanism. Moreover, there is a strong association between pre-existing psychiatric illness, particularly anxiety and mood spectrum disorders, and TTS. Acute exacerbation of psychiatric illness, rapid up-titration or overdose of certain psychotropic agents, and electroconvulsive therapy may trigger TTS. Treatment options of TTS are largely empiric and supportive; however, when hemodynamics permit, beta blockers seem to be helpful.