



# Happy Heart Syndrome: A Review of Takotsubo Cardiomyopathy Triggered by Positive Emotional Stressors

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## ABSTRACT

Takotsubo cardiomyopathy, or “broken heart syndrome,” is a condition characterized by transient left ventricular dysfunction, typically triggered by intense emotional or physical stress. While initially thought to follow primarily negative life events, such as grief or fear, a subset of cases now recognized as “happy heart syndrome” occur instead after positive emotional triggers, such as celebrations, romantic moments, or good news. This review summarizes the current understanding of takotsubo syndrome (TTS) precipitated by joyful events, focusing on novel insights from the largest registry analysis to date of 37 “happy heart” cases from the international, multicenter GEIST registry. Compared with the more common negative emotional trigger group, these patients had a higher prevalence of men and atypical midventricular ballooning patterns. While event rates trended lower, likely due to the small sample size, acute complications, such as pulmonary edema, and long-term mortality did not definitively differ. The pathophysiology of “happy heart syndrome” remains unclear but implicates differences in central autonomic processing and peripheral catecholamine responses to positive vs negative emotional stimuli. Several key outstanding questions are highlighted, including understanding susceptibility factors, confirming prognostic differences, and leveraging insights into underlying brain–heart circuitry. Ultimately, dedicated research into this rare but fascinating condition could provide broader mechanistic insights and therapeutic opportunities for the prevention and management of all takotsubo phenotypes at the nexus of mind, brain, and heart.

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## INTRODUCTION

Takotsubo cardiomyopathy, also known as stress cardiomyopathy or broken heart syndrome, is a fascinating condition characterized by transient left ventricular dysfunction that mimics an acute myocardial infarction. It was first described in 1990 in Japan, with the name “takotsubo” referring to the distinct apical ballooning appearance of the left ventricle that resembles a Japanese octopus trap.<sup>1</sup> While initially thought to occur primarily following negative emotional stressors, such as grief, fear, or anger, more recent evidence shows takotsubo can also be triggered by positive emotional events, dubbed “happy heart syndrome.”<sup>2,3</sup>

This review summarizes the current understanding of takotsubo syndrome (TTS) precipitated by joyful life events, with a focus on novel insights from a large international registry study.<sup>4</sup> The pathophysiology, clinical characteristics, prognosis, and outstanding knowledge gaps of this intriguing condition are discussed.

## BACKGROUND

Takotsubo syndrome accounts for an estimated 1–3% of suspected acute coronary syndrome cases.<sup>5</sup> While the exact mechanisms are unclear, the leading hypothesis proposes

that an exaggerated release of catecholamines (adrenaline and noradrenaline) in response to intense emotional or physical stress triggers negative inotropic, metabolic, and vasomotor effects that impair myocardial function.<sup>6</sup> This “brain–heart” interaction, likely mediated by the sympathetic nervous system, appears to induce the characteristic regional wall motion abnormalities seen in TTS, most commonly apical ballooning.<sup>7</sup>

Emotional stressors have long been recognized as common precipitants of TTS, accounting for roughly one-third of cases in registry studies.<sup>8,9</sup> Early reports focused on negative emotional triggers, like grief, fear, anxiety, or interpersonal conflicts, coining the term “broken heart syndrome.”<sup>10</sup> However, in 2005, a seminal case series from the International Takotsubo Registry identified a subset of patients in whom TTS was precipitated instead by positive life events, like celebrations, romantic moments, or good news—so-called “happy heart syndrome.”<sup>2</sup>

## EPIDEMIOLOGY AND TRIGGERS

In the largest registry study to date from the international, multicenter GEIST registry published in 2023, just 37 of 2,482 TTS cases (1.5%) were attributed to positive emotional triggers, while 873 cases (35%) had negative emotional triggers.<sup>4</sup> Thus, while “broken

heart syndrome” is 20–30 times more common, “happy heart syndrome” does appear to represent a distinct clinical entity.

The specific positive emotional triggers identified included birthday and family celebrations, romantic moments, weddings, receiving good news, public performances, family reunions, vacations, and other joyous occasions.<sup>4</sup> This diversity underscores that a variety of pleasant life events have the potential to provoke the catecholamine surge believed to induce TTS in susceptible individuals.

While the overwhelming majority of TTS cases occur in postmenopausal women, a notable 18.9% of “happy heart” cases were men, compared to only 5% of “broken heart” cases.<sup>4</sup> The reasons for this gender disparity are uncertain, but could relate to differences in physical vs emotional trigger prevalence, or biological susceptibility between men and women.

## CLINICAL CHARACTERISTICS

Aside from a higher proportion of male patients, the baseline demographic and clinical characteristics, such as cardiovascular risk factors, clinical presentation, and ejection fraction, were similar between the “happy heart” and “broken heart” groups in the GEIST study.<sup>4</sup> However, atypical nonapical ballooning patterns, especially midventricular, were significantly more common with positive emotional triggers (27 vs 12.5% in the negative group).

The mechanisms underlying these distinct regional ballooning patterns are unknown but could involve differences in the temporal dynamics, magnitude, or regional distribution of catecholamine release in response to positive vs negative emotional stressors.<sup>4</sup> Variable regional myocardial beta-

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adrenoreceptor expression and downstream signal transduction effects may also play a role.<sup>11</sup>

## PROGNOSIS

Due to the relatively small number of “happy heart” cases, the GEIST study was likely underpowered to definitively assess differences in outcomes compared to the “broken heart” group. Nevertheless, there was a clear trend toward lower rates of in-hospital complications, such as pulmonary edema, cardiogenic shock, and death, in the positive emotional trigger group, albeit not reaching statistical significance (8.1 vs 12.3%).<sup>4</sup> Long-term mortality over a mean 3.4 year follow-up was also numerically lower at 2.7% compared to 8.8% in “broken hearts,” again a nonsignificant difference.<sup>4</sup>

Potential explanations for a more benign clinical course with positive emotional triggers could include lower stroke volumes or rates of catecholamine release, more frequent atypical ballooning patterns, which have been associated with better outcomes in some studies,<sup>12</sup> or an impact of the emotional milieu itself on neurohormonal recovery pathways. However, larger prospective studies are needed to confirm any prognostic implications of the emotional trigger in TTS.

## PATHOPHYSIOLOGY

While the exact pathophysiology of TTS remains poorly understood, the leading hypothesis centers on exaggerated catecholamine toxicity in a susceptible myocardium, likely triggered by brain–heart neural signaling pathways.<sup>6,7</sup> Proposed mechanisms of catecholamine-mediated myocardial stunning include direct toxic effects, switching of beta-adrenoreceptor signal transduction from positive to negative inotropic pathways, microvascular spasm, and metabolic disturbances.<sup>13</sup>

Why positive emotional events might trigger a similar process is unclear but could relate to differential activation of central autonomic control centers or divergent peripheral effector pathways compared to negative emotional stress. Interestingly, some neuroimaging studies have revealed distinct patterns of brain activity in response to pleasant vs unpleasant stimuli.<sup>14</sup>

The observed differences in regional ballooning patterns between “happy” and “broken” hearts also raise intriguing questions about potential heterogeneity in sympathetic innervation, beta-receptor distribution, or signal transduction effects

across different myocardial segments.<sup>4,11</sup> Ultimately, dedicated mechanistic studies will be needed to dissect these pathways and explain the clinical diversity of TTS phenotypes.

## UNANSWERED QUESTIONS

While the GEIST study represents the most comprehensive analysis of “happy heart syndrome” to date, several key questions remain:

- Why do only a small minority of intense positive emotions trigger TTS, while many more cases follow negative emotional stress? Are there distinct central nervous system processes that modulate the peripheral catecholaminergic response?
- What are the pathophysiologic bases for the observed clinical differences, like gender distribution and ballooning patterns, based on the emotional trigger?
- Do “happy heart” cases truly have more benign acute complications and long-term prognosis, as suggested by GEIST? Larger prospective cohort studies are needed.
- Can improved understanding of “happy heart” cases provide broader insights into the general pathophysiology and therapeutic prevention or management of TTS?
- Are there reliable ways to predict which emotional milieu (positive or negative) might trigger TTS in a given susceptible patient? Can biomarker or neuroimaging approaches help elucidate individual risk?

## CONCLUSION

In summary, while takotsubo cardiomyopathy or “broken heart syndrome” classically follows negative emotional stressors, a subset of around 1–2% of cases appears to be precipitated by positive emotional triggers, termed “happy heart syndrome.”<sup>2–4</sup> The largest analysis to date from the GEIST International Registry confirms several distinct clinical characteristics of this rare condition, including a higher prevalence in men and atypical midventricular ballooning patterns compared to the more common negative emotion–triggered cases.<sup>4</sup>

“Happy heart syndrome” highlights the complex brain–heart interactions that likely underlie all cases of takotsubo cardiomyopathy, with differences in autonomic processing of positive vs negative emotional stimuli potentially translating into divergent patterns of regional myocardial injury.<sup>4,6,7</sup> While preliminary GEIST data

suggest “happy hearts” may have a more benign clinical course, larger prospective studies are still needed.<sup>4</sup>

Ultimately, dedicated research into this unique subgroup could provide broader insights into TTS pathophysiology and heterogeneity. A deeper understanding of how the heart processes and responds to various emotional stressors, whether positive or negative, could uncover novel prevention and management strategies for this fascinating cardiomyopathy at the intersection of mind, brain, and heart.

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