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Honnold's Calm at Extreme Heights and the Emotional Storm of Takotsubo Syndrome: The Two Poles of the Amygdala

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Free solo climber Alex Honnold conquers one of the world's tallest buildings in Taiwan on Jan 25, 2026, whose climbing ascent of Taipei 101 was not merely a breathtaking spectacle of extreme sport; it also served as a powerful neuroscientific demonstration centered on the amygdala [1]. A clear "brain–heart axis" emerges from this perspective, with the amygdala standing as a crucial hub.

In 2016, researchers at the Medical University of South Carolina used functional magnetic resonance imaging (fMRI) to scan Honnold's brain [2]. When he was shown highly dangerous or frightening images, the amygdala—the region responsible for fear processing and threat detection—showed almost no significant activation. In most individuals, the amygdala rapidly activates upon detecting danger, stimulating the sympathetic nervous system and leading to increased heart rate, elevated blood pressure, and a surge in adrenaline. In Honnold's case, however, this system appears to have an unusually high threshold of activation, demonstrating a near "hyporeactive" state. This does not mean he lacks awareness of risk; rather, his physiological response to threatening stimuli is markedly reduced. Such neural characteristics enabled him to maintain composure and precision during his 91-minute and 30-second ascent at a height of 508 meters.

In contrast, patients with Takotsubo syndrome—commonly known as "broken heart syndrome"—present a dramatically different neuroimaging pattern [3]. A 68-year-old male patient with a prior history of minor stroke later developed irritability, reduced emotional control, and depressive tendencies. Based on the characteristics and dynamics of (99m)Tc-ethyl cysteinate dimer ((99m)Tc-ECD) as a tracer, (99m)Tc-ECD single-photon

emission computed tomography (SPECT) demonstrated significant hyperperfusion in the bilateral amygdalae, alongside hypoperfusion in the prefrontal cortex and anterior cingulate cortex [4]. Scholars have proposed that this imbalance within the limbic system leads to amygdala hyperactivity, which in turn strengthens sympathetic output via the limbic–hypothalamic–brainstem pathway. As a result, the myocardium becomes exposed to high concentrations of norepinephrine and neuropeptide Y, triggering stress-induced cardiomyopathy. This produces transient myocardial contractile dysfunction and the characteristic "apical ballooning" phenomenon [5]. This is precisely what is referred to as Takotsubo syndrome—an emotional storm ultimately transformed into structural and functional abnormalities of the heart.

Thus, we observe two extremes. The first is the "hyporeactive amygdala" type exemplified by Honnold. When confronted with extreme external threats, his amygdala does not activate intensely; therefore, the sympathetic nervous system does not become excessively aroused, and cardiovascular stability is maintained. This neural trait allows him to preserve precise motor control and clear judgment even at the edge of a skyscraper.

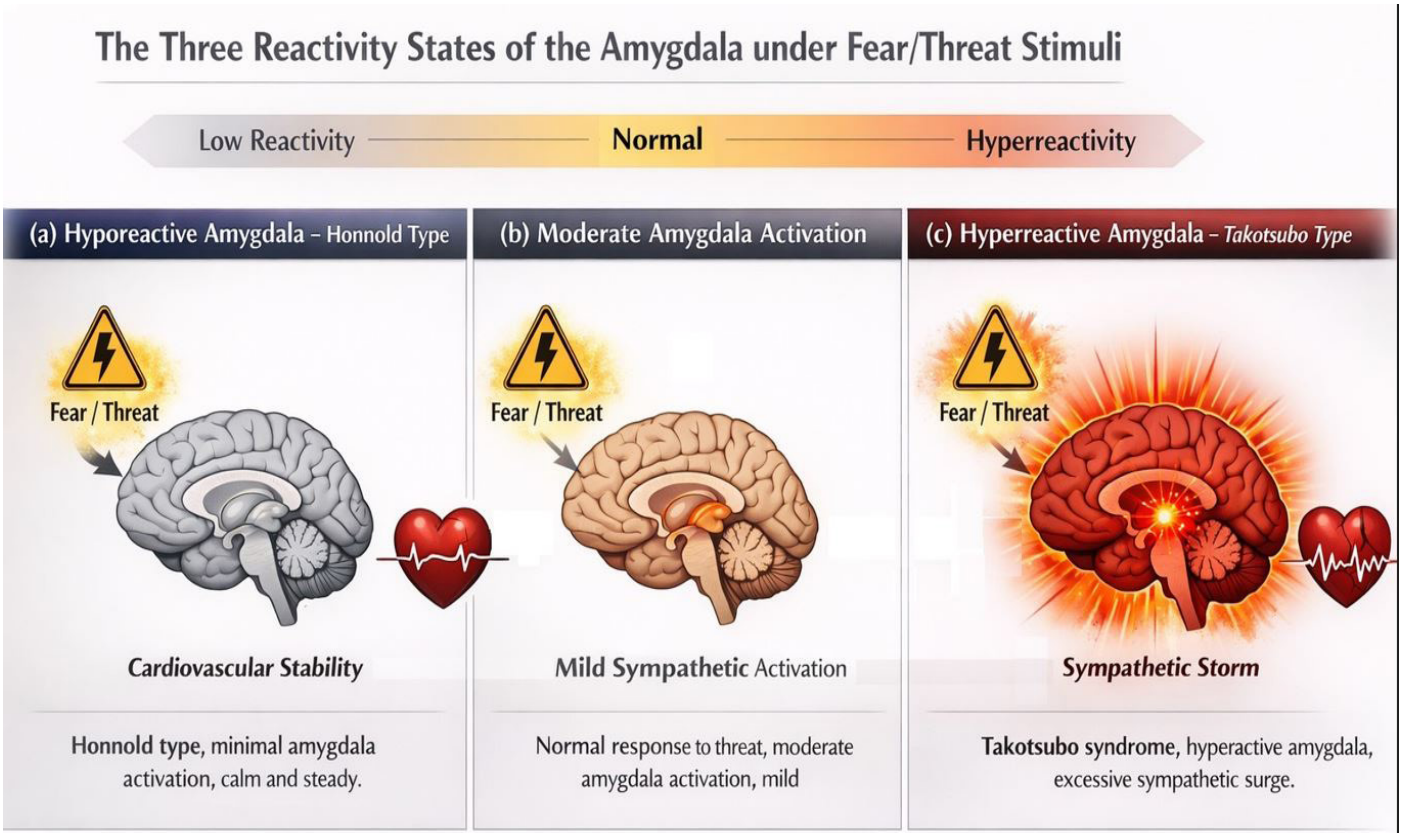
The second is the "hyperreactive amygdala" observed in Takotsubo syndrome. Following stroke-induced limbic imbalance, the amygdala exhibits hyperperfusion and hyperactivity. If accompanied by hypoperfusion in the prefrontal cortex and anterior cingulate cortex, emotional inhibition and rational regulatory control decline, creating a state in which "the emotional accelerator is pressed while the rational brake fails." Emotional fluctuations are then transmitted through the autonomic nervous system directly to the heart, resulting in transient myocardial dysfunction.

From a neuroscientific standpoint, when the amygdala is overly active and the prefrontal cortex insufficiently regulating, emotions may spiral out of control and even affect cardiac function through the brain–heart axis. Therefore, Honnold's ascent of Taipei 101 symbolizes not only courage and adventure, but also an extreme example of low amygdala reactivity. Conversely, Takotsubo syndrome reminds us that heightened amygdala reactivity may lead to real and potentially life-threatening physiological consequences. One is calm and controlled; the other, turbulent and overwhelming. Yet both point to the same conclusion: the amygdala plays a pivotal role in regulating emotion, autonomic function, and ultimately cardiac health.

When we encounter patients who are irritable, anxious, or emotionally dysregulated, we should not interpret their condition solely from a psychological perspective. We must also consider whether there is an imbalance within the limbic system. As suggested in the literature, in post-stroke patients who present emotional instability and cardiac abnormalities, arranging brain perfusion imaging in scintigraphic rehabilitation to evaluate amygdala activity carries significant clinical importance [6].

In summary, from Honnold's serenity at extreme altitude to the emotional storm of Takotsubo syndrome, what we observe is not merely a difference in personality, but two physiological poles of amygdala activation. the two poles of the amygdala facing fear and threat is illustrated in figure. The amygdala can render a person fearless at the edge of a precipice or cause the heart to collapse under stress. To understand the amygdala is to understand how emotion shapes our destiny—and even alters the rhythm of the human heart.

Figure



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