Secure Phylogenetic Attachment: The Antithesis of Trauma?
A Monozygotic Twin Study Assessing the Efficacy of Somatic Experiencing® and Attachment Focused-Somatic Experiencing

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Secure Phylogenetic Attachment: The Antithesis of Trauma?

A Monozygotic Twin Study Assessing the Efficacy of Somatic Experiencing® and Attachment Focused-Somatic Experiencing

Joseph P. Riordan

Abstract

Monozygotic twins offer a unique opportunity to examine the contagious nature of trauma in attachment dyads when one twin experiences trauma, but the other does not. Dyadic trauma is suboptimal and antagonistic to the dynamics of secure phylogenetic attachment (SPA). Attachment perturbations due to trauma may be complicit in widespread psychopathology, loneliness, social isolation, and loss of social cohesion. Somatic experiencing (SE) is an effective treatment for PTSD and attachment focused-somatic experiencing (AF-SE) resolves dyadic trauma. Traumatology may require a paradigm shift to include the interpersonal neurobiological dynamics of trauma as a contagion and the emerging role of SPA as the antithesis of trauma.

Monozygotic twins share the same DNA and may share a unique form of sibling attachment, described here as monozygotic attachment characterized by greater than expected interpersonally attuned face–heart connectedness in uniquely congruent somatic expressions of monozygotic attunement resulting in suboptimal attachment. A quantitative, experimental approach was used to determine whether dyadic trauma is contagious and compromises SPA and also whether SE and AF-SE are effective treatments for posttraumatic stress disorder (PTSD), trauma symptomology, and dyadic trauma to restore SPA. Inferential analysis offered observations into the nature of trauma in attachment relationships and the significance of trauma in relation to widespread psychopathology, loneliness, social isolation, and loss of social cohesion.

Smartphone devices were used to record seven autonomic variables pre-and post-treatment for both twins: heart rate variability (HRV), three HRV index variables, heart rate, sleep duration, and sleep disturbances. Mean and small sample *t* tests were applied to determine statistical significance. The study concludes that trauma is contagious in attachment dyads and contributes to psychopathology, loneliness, social isolation, and loss of social cohesion. SE is an effective treatment for PTSD and trauma symptomology. AF-SE resolves dyadic trauma restoring SPA as the antithesis of trauma.

Keywords: attachment focused-somatic experiencing, dyadic trauma, dyadic completion, monozygotic attachment, monozygotic attunement, secure phylogenetic attachment, somatic experiencing

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Trauma and dyadic trauma in all their manifestations are socially destructive (Riordan et al., 2017, 2019) and may have a role in widespread community psychopathology, loneliness, social isolation, and loss of social cohesion. Twenty-five percent of Australian households are single-person homes (Australian Bureau of Statistics, 2016). One in four people are lonely (Abbott et al., 2018) or experience social isolation (Relationships Australia, 2017). Deaths by suicide in Australia have increased every year in the last decade, numbering 12.9 deaths per 100,000 in 2019 (Australian Bureau of Statistics, 2020). Given these figures, social isolation is now emerging as a major issue for health care professionals (Abbott et al., 2018; Lim, 2018) and governments. A Minister for Loneliness was appointed by the United Kingdom government in 2018 (ABC News, 2018), and in Australia a parliamentary inquiry to address loneliness was set up by the Queensland government in 2020 (Gramenz, 2020).

There are many circumstances that contribute to loneliness, social isolation, and loss of social cohesion. When viewed through the lens of neurophysiology, trauma compromises and hijacks the social networks of the prefrontal cortex (Schore, 2019a). This in turn generates a flight survival response (Porges, 2011) that promotes avoidance, a key symptom of posttraumatic stress disorder (PTSD; American Psychiatric Association, 2013). People with PTSD have a high tendency to experience social isolation and loss of social cohesion because sympathetic arousal devolves in a cascade of trauma responses (loosely defined as the fight/flight/freeze response) that later manifest in relationships as perturbations in the dyad, generating dyadic trauma (Riordan et al., 2019). Serious trauma like PTSD impairs a person's ability to form and sustain relationships in multiple ways—for example, they may face significant challenges in social and attachment relationships (Riordan et al., 2019), have difficulty resolving attachment ruptures (Riordan et al., 2017), and cannot independently complete autonomic survival imperatives to sustain secure phylogenetic attachment relationships (Riordan et al., 2019). Trauma is experienced interpersonally as perturbations in attachment relationships. Severe trauma can initiate a complex web of trauma symptomology that generates widespread psychopathology across communities.

The field of traumatology offers only sparse quantifiable understanding of how trauma impacts relationships and how it is transposed neurogenically in secure attachment dynamics. Measures of trauma responses in pre- and posttreatment studies offer objective platforms to understand the nature of trauma in nervous systems and how it impacts attachment. This study of monozygotic (identical) twins attempts to understand the effect of dyadic trauma on suboptimal attachment styles, and in particular, how *trauma as a contagion* is transposed in dyadic trauma when one twin is traumatized and the other is not.

SE (Levine, 2010) and AF-SE (Riordan et al., 2019) may offer important therapeutic utility regarding trauma in relationships.

This quantifiable experimental research aims to determine:

a. the efficacy of SE and AF-SE as effective treatments for PTSD, trauma symptomology, and dyadic trauma; and
b. whether trauma is manifest in dyadic trauma, is contagious, and compromises secure phylogenetic attachment.

Three secondary aims will be considered inferentially:

i. To elucidate the nature of monozygotic attachment and monozygotic attunement by comparing autonomic physiological variables between T1 (the traumatized twin) and T2 (the non-traumatized twin) pre- and post-treatment;
ii. To examine the construct of secure phylogenetic attachment as the antithesis of trauma; and
iii. To consider the impact of dyadic trauma on loneliness, social isolation, and loss of social cohesion.

Somatic Experiencing

SE is a clinically effective treatment for the neurobiological resolution of trauma within the individual nervous system (Brom et al., 2017; Leitch, 2007; Leitch et al., 2007; Levine, 2010; Parker et al., 2008). A core tenet of SE in the resolution of trauma is "the completion of thwarted, biologically based, self-protective and defensive responses, and the discharge and regulation of excess autonomic arousal" (Payne et al., 2015 p. 1). SE offers a comprehensive understanding of trauma recovery through an active process of therapeutic engagement and as an *empathic witness* (Levine, 2010) of the client's interoceptive, kinesthetic, and proprioceptive neurophysiology during dynamic SE sessions. The SE therapist is engaged and attuned to the client's somatic shifts as they internally regulate and reorganize in homeostasis toward neural synchrony between the cortex and lower brain structures allowing for a flexible state of relaxed readiness, renegotiating trauma triggers to effectively respond to current events. (Figure 1)

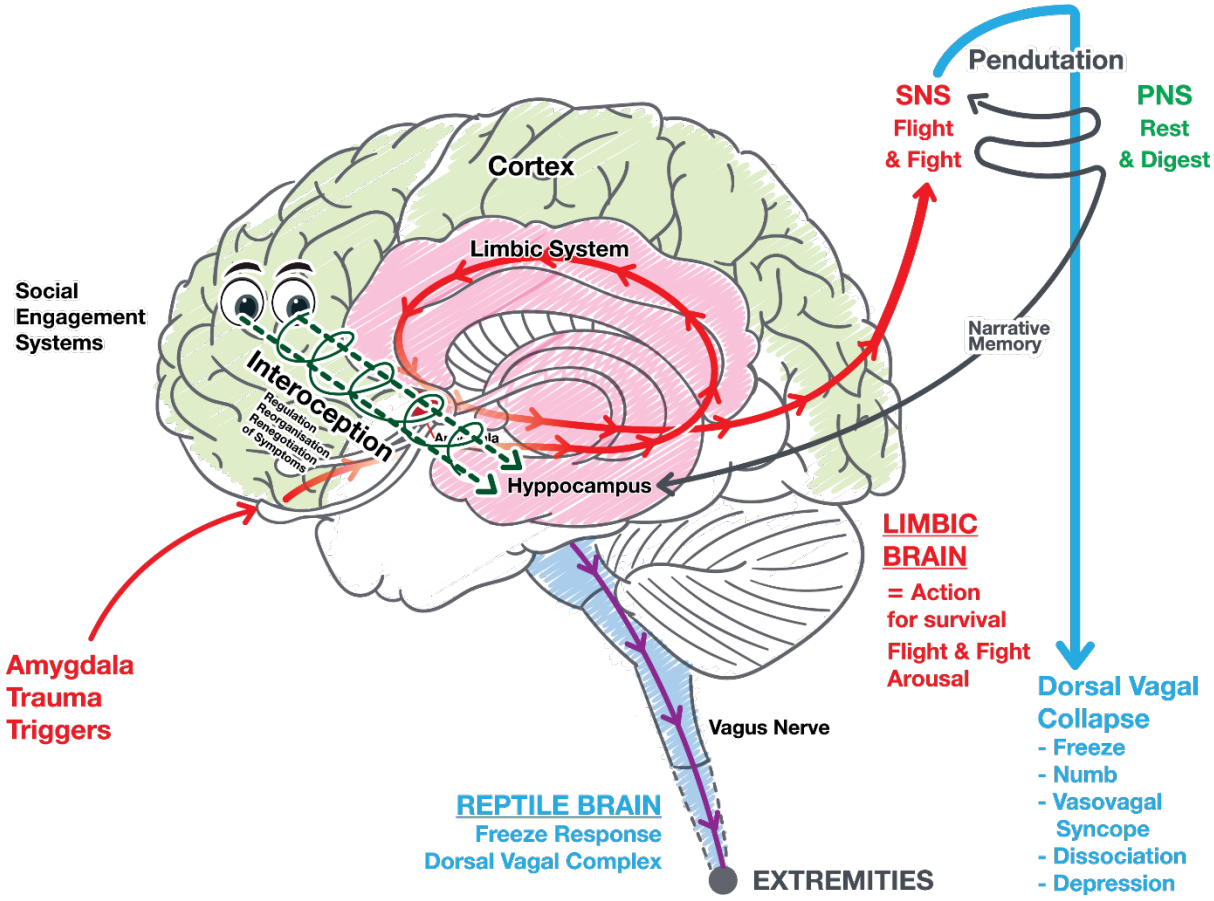

Figure 1 *The Neurogenic Action of SE Interoception on the Traumatized Brain*

Note. The processes of interoception and the impact of SE on the brain are symbolized in Figure 1 showing the major structures of the brain and the general action of SE pendulation during and after interoception, a shift from sympathetic arousal (flight/fight/freeze responses) to parasympathetic (rest and digest) state with transposing of traumatic somatic and image memory into narrative memory in the hippocampus. Adapted and modified from "Attachment Focused-Somatic Experiencing: Secure Phylogenetic Attachment, Dyadic Trauma and Completion Across the Life Cycle," by J. P. Riordan, A. Blakeslee, and P. Levine, 2019, *International Journal of Neuropsychotherapy, 7*(3) p. 73. Copyright by the International Association of Applied Neuroscience

SE works directly with implicit or nonconscious memory and is particularly effective with preverbal infants as well as for accessing preverbal trauma memory in older children and adults (Riordan et al., 2017, 2019; Schore, 2019a, 2019b). SE works inherently to engage nervous systems in secure attachment; however, combining SE with modern attachment theory and emerging psychoneurobiological paradigms of traumatology offers an alternative focus toward the traumatized attachment dyad as a feature of treatment with AF-SE throughout the life cycle. AF-SE is an emerging form of somatic psychotherapy that offers a more nuanced and direct way of treating dyadic trauma in attachment dyads (Riordan et al., 2017, 2019).

Attachment Focused-Somatic Experiencing

As described in Riordan et al. (2017, 2019), AF-SE offers neurobiological coordinates and a therapeutic process to address dyadic trauma (Figure 2) and utilizes interpersonal constructs to understand threat recovery and its role in resolving trauma with *dyadic completion*. AF-SE regulates threat arousal in a similar fashion to SE by targeting somatic coordinates of attachment trauma in the neurobiology of the traumatized individual and the attachment dyad. Interoceptive, kinesthetic, and proprioceptive coordinates known to downregulate autonomic threat responses associated with interpersonal betrayals, neglect, abandonment, and somatic memory of harm from others are utilized to reinstate healthy interpersonal attunement and attachment neuroception in the homeostatic processes to restore SPA.

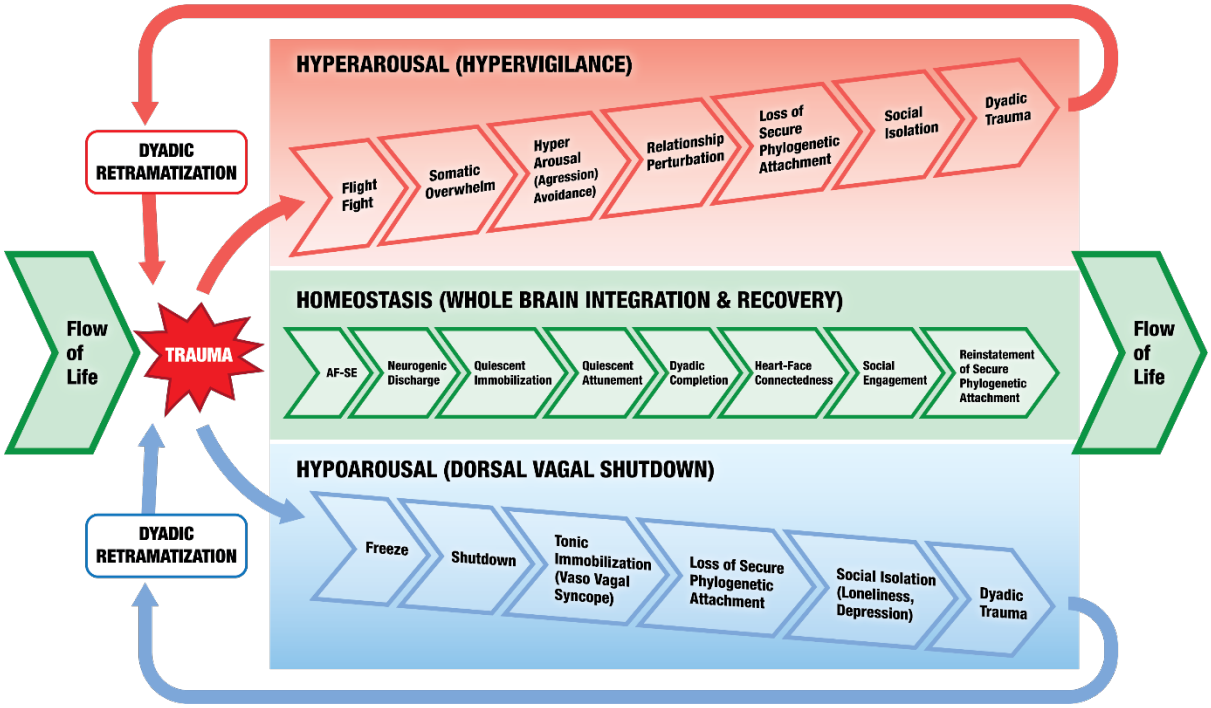

Figure 2 *The Neurobiological Coordinates and Therapeutic Processes of Threat Response and Recovery with AF-SE*

Note. Adapted from "Attachment Focused-Somatic Experiencing: Secure Phylogenetic Attachment, Dyadic Trauma and Completion Across the Life Cycle," by J. P. Riordan, A. Blakeslee, and P. Levine, 2019, *International Journal of Neuropsychotherapy, 7*(3) p. 73. Copyright by the International Association of Applied Neuroscience.

The AF-SE model demonstrates how traumatic hyperarousal and hypoarousal (numbing) can lead to dyadic trauma and social isolation. It also shows the therapeutic pathways to recovery in SPA. SE therapists are trained in close observation of somatic states and how to negotiate hyperarousal and hypoarousal by regulating (titrating) access to internal traumatic states allowing discharge of excessive autonomic arousal during the SE and AF-SE process.

Trauma has many facets and a variety of definitions. In all its permutations, trauma has emerged as a major focus of research for neurotraumatologists and attachment theorists to provide better outcomes in psychotherapy. Peter Levine, developer of SE, has defined trauma as an incomplete survival response after fear/terror immobilization, after which "persistent maladaptive procedural and emotional memories form the core mechanism that underlies all traumas, as well as many problematic social and relationship issues" (Levine, 2015, p. 38). According to Riordan and colleagues (2019), trauma compromised whole brain function by innervating phylogenetic survival imperatives. Flight/fight/freeze responses in the limbic and primitive brain override social and cognitive function in the prefrontal neocortex. Social engagement systems are downregulated and attachment dynamics are compromised. Riordan, Blakeslee, and Levine (2019) further define trauma as "a homeostatic disruption that involves disintegration of neurological communication between the major structures of the brain. Primitive subcortical brain structures hijack and dominate neural functioning to drive survival along flight/fight/freeze trajectories where social engagement is compromised" (p. 3). The authors add to this definition as follows:

> Dyadic trauma occurs when resonance and attuned connectedness in secure attachment alters for both participants in the dyad. Social engagement, governed by mutually integrated whole-brain connection, shifts to disorganized social avoidance. Fear and anger drive survival imperatives dominated by limbic and subcortical brain structures. Dyadic trauma is neurologically dis-integrating and contagious in attachment dyads and may promote psychopathology throughout the life cycle. (Riordan et al., 2019, p. 3)

Modern Attachment Theory and Dyadic Trauma

Modern attachment theory, building on the work of Bowlby (1969), emphasizes neurophysiology and its role in interpreting the nature of attachment to elucidate the earlier observations of attachment theory. Modern attachment theorists agree that attachment is an interpersonal neurobiological process (Siegel, 2012) involving bonding (Scaer, 2014) in secure phylogenetic attachment for survival (Riordan et al., 2019). Attachment is manifest in attuned connectedness (Porges, 2016) for individual homeostatic neurodevelopment (Schore, 2019b) and mutual regulation (Tronick, 2007). This interpersonal

neurobiology is visually represented in the fMRI image of mother and child in Allan Schore's *The Development of the Unconscious Mind* (2019a, p. 6).

Early childhood attachment rupture and sequentially repeated attachment perturbations negatively influence the neurobiological development of the child's right social brain (Schore, 2019a). Secure phylogenetic attachment with the primary attachment figure during infancy and preschool years is an essential feature of right brain social neurodevelopment and critical if children are to develop secure attachment bonds in adult life (Schore, 2019a). Trauma-induced sibling attachment is suboptimal and compromises secure phylogenetic attachment style in later life (Riordan et al., 2019).

Repeated early childhood "dysregulating attachment experiences (abuse and neglect)" (Schore, 2012, p. 90) generate attachment perturbations in the dyad, and repeated threat arousal initiates growth in the dorsal vagal hyperarousal system (Porges, 2011), which is dominant in states of danger (flight/fight/freeze) for survival.

Bowlby (1969) hypothesized that the individual's capacity to cope with stress is correlated with certain maternal behaviors and that the developing emotional and limbic systems are impacted by attachment transactions. This observation, emphasized by Schore (2003), highlights attachment outcomes and the environment of adaptiveness to have consequences that are "vital to the survival of the species" (p. 178).

Trauma in all its forms, particularly postpartum and early childhood traumatic separation from primary attachment figures, can generate dyadic trauma (Riordan et al., 2017). Psychopathology may emerge for participants in the traumatized dyad and acts as a contagion for all future relationships throughout the life cycle (Riordan et al., 2019).

Monozygotic twins are more concordant than dizygotic (nonidentical) twins (Torgersen et al., 2007), and their genetic relatedness has profound impact on their attachment hierarchy and the relative rank of their romantic partner in comparison to their sibling (Schwarz et al., 2015). Monozygotic twins often prefer their twin to their romantic partner and experience more intrusive attachment perturbations and sibling attachment rupture during courtship than non-twin siblings.

Therapeutic attendance to developmental factors and relational issues in the attachment dynamics of traumatized individuals may be a fundamental necessity in the battle against trauma, dyadic trauma, and widespread community psychopathology. SPA and its reinstatement with SE and AF-SE after trauma may be an important theoretical construct for traumatologists supporting these treatment options in psychotherapy to address the psychopathology of loneliness, social isolation, and loss of social cohesion.

SPA refers to evolutionary dyadic survival imperatives of psychoneurotraumatology and the innate capacity to form secure attachments throughout the life cycle. SPA incorporates neurobiological, neurochemical, behavioral, and affective interpersonal dynamics that promote attuned, connected, and engaged attachment between individuals for survival and promotes dyadic synergy that is resonant, attuned, engaged, and mutually regulating. SPA invites synergy in social dynamics that involve nurturing, support, acceptance, regard, tolerance, love, and respect.

SPA refers also to the evolution of attachment dynamics in a dyad from postpartum infant/maternal regulation through to end-of-life caregiving. Behaviorally, SPA involves face-to-face, heart-to-heart connection in mutual somatic-attachment-soothing (Riordan et al., 2017) that promotes interpersonal connectedness and evolutionary survival of the individual, the dyad, the family unit, communities, and the human species. Neurochemically, SPA involves biological processes of trauma recovery manifest in neurochemical shifts from cortisol (stress hormone) to oxytocin (love and bonding hormone). Furthermore, SPA promotes an internal neurophysiological balance between parasympathetic relaxation and sympathetic excitation in the autonomic nervous system (Riordan et al, 2019).

Traumatic perturbations in the individual's neurophysiology may be transposed in attachment dyads leading to dyadic trauma via the same interpersonal neurobiological pathways as secure attachment and intergenerationally may be a significant contributor to increasing community psychopathology.

There is a linear relationship between trauma, social isolation, and loss of social cohesion. Neurological perturbations in the brain of the traumatized person emphasize vigilance in the survival networks of the limbic brain—specifically, innervation of the amygdala (Riordan et al., 2019). This in turn impacts social engagement, which causes loss of trust in relationships due to increased social vigilance and leads to interpersonal conflict. The eventual outcome after long-term unresolved trauma is a tendency toward social isolation, loneliness, and loss of social cohesion.

Dyadic trauma may resolve into secure phylogenetic attachment when completion of survival imperatives is initiated in the processes of SE and AF-SE, thereby validating SPA as the antithesis of trauma. This neurological process is most applicable in preverbal infants and preschool children (Riordan et al., 2017, 2019) but can be applied to adolescent and adult dyads.

Schore (2019a) noted that chronic relational trauma in infancy "leads to the dysregulation of emotions" (p. 240). Relational trauma may emerge as dyadic trauma (Riordan et al., 2017, 2019) in all future attachment dyads due to early childhood adverse experiences and other traumatic experiences that emerge as increasingly traumatizing perturbations in dyads over time, compromising SPA throughout the life cycle.

Schore (2019a) in his dialogue regarding relational trauma stated: "Early relational trauma negatively affects the limbic and autonomic nervous system maturation, producing enduring neurobiological alterations that underlie right brain affective instability, inefficient stress tolerance, memory impairment, and dissociative disturbances" (p. 51).

Postpartum trauma compromises SPA dynamics in the primary attachment dyad, but for monozygotic twins it may also generate suboptimal parental and sibling attachment styles. Monozygotic twins in their face–heart connection often attune in mutual soothing with each other, which in turn

compromises SPA in later life, particularly in their attachment hierarchy dynamics regarding procreative pair bonding.

Porges (2011) defined the face–heart connection as "an anatomical and neurophysiological linkage between neural regulation of the heart via the myelinated vagus and the special visceral efferent pathways that regulate the striated muscles of the face, head, and neck. Together, this linkage between brain stem and motor systems is responsible for cardiovascular functions and those necessary for regulating the face, head, and neck and forms an integrated social engagement network." (p. 125)

Critically, when the attachment dyad is compromised by trauma, dyadic completion (Riordan et al., 2017) with AF-SE may be required to achieve full recovery for both participants in the dyad, particularly in the vulnerable young who are so heavily dependent on SPA for survival.

Trauma is a normal life event that provides experiential learning and strengthens phylogenetic adaptation (stress tolerance) provided trauma is followed by neurogenic completion of survival responses (Levine, 2010). However, Scaer (2014) asserted that "survival of a mortal threat has important implications for treatment of physical and emotional disorders and victims of trauma" (p. 18). Consequently, an incomplete survival, freeze, or dorsal vagal shutdown—characterized by Scaer (2014) as "the truncated freeze discharge" (p. 18)—appears to be "the physiological event that initiates the central nervous system changes leading to posttraumatic stress disorder" (p. 18). Similarly, Kozlowska et al. (2015) described the freeze response as a "flight-or-fight response put on hold" (p. 1).

Trauma in all its forms—episodic, relational, and developmental—impacts key secure phylogenetic attachment relationships. The younger a person is, the more profound the impact of trauma on primary attachment relationships, consequently postpartum separation may have profound effects on SPA throughout the life cycle. Schore (2003) noted that "the infant's environment of adaptiveness has consequences that are vital to the survival of the species" and, further, that "the attachment relationship directly influences the infant's capacity to cope with stress by impacting the maturation of a control system in the infant's brain that comes to regulate attachment functions" (p. 178).

SPA may be the antithesis of trauma in that it directly counteracts the psychoneurosocial dynamics of debilitating attachment perturbations (dyadic trauma), promoting mutual connectedness and social cohesion.

Sibling Dyadic Trauma

Van der Kolk (2014) noted that an increasing number of neglected, traumatized siblings are presenting in clinical settings with developmental trauma or childhood PTSD (American Psychiatric Association, 2013) accompanied by secondary attachment figures, usually grandparents or other foster parents. Due to the changing nature of Western communities, historically secure attachment in child/parent relationships is no longer an assumed variable in child development. Equally, children are being parented by primary attachment figures who have themselves experienced childhood or adult trauma and/or mental health conditions.

Van der Kolk (2014) has predicted a dramatic surge in developmental disorders due to the changes in attachment styles and child/parent relationships.

Gender differences in neurodevelopmental attachment styles identifies that boys have specific neurodevelopmental needs that require attention (Schore, 2019a). Regarding male infants and children, Tronick (2007) reported:

> Mothers with a history of major depression were more affectivity negative with their sons than their daughters. In addition, male infants, as compared to female infants, were less emotionally positive and less likely to use self-comforting strategies to regulate affectivity states on their own. (p. 345)

Schore (2017) stated:

> The stress-regulating circuits of the male brain mature more slowly than those of the female in the prenatal, perinatal and postnatal critical periods, and . . . this differential structural maturation is reflected in normal gender differences in right brain attachment functions. Due to this maturational delay, developing males also are more vulnerable over a longer period of time to stressors in the social environment (attachment trauma) and toxins in the physical environment (endocrine disruptors) that negatively impact right brain development. (p.1)

He went on to state "that a certain group of 'high risk' male children will, as they enter adolescence, be highly susceptible to a number of psychiatric disorders whose diagnostic symptomatology first appears in that period of human development" (Schore, 2017, p. 17).

In the evolution of a traumatic episode over time, siblings may experience a cumulative process of attachment rupture with their primary attachment figure resulting in *displaced traumatic sibling attachment* that in turn interrupts secure phylogenetic attachment with their caregiver. Displaced traumatic sibling attachment is characterized by siblings in trauma adopting an attachment relationship style in their sibling dyad where one child adopts a parental protective/nurturing role and the other child transfers attachment to their sibling in their phylogenetic drive for survival. This process is most often observed in neglect episodes where there is a transference of the primary attachment bond from child–parent to sibling–sibling (Riordan et al., 2019) but can also occur in safe, loving families in the aftermath of birth or postsurgical trauma, or following periods of maternal separation.

In polyvagal theory (Porges, 2011), episodes of vasovagal syncope or fainting may occur in response to trauma where neurogenic traumatic overwhelm is incomplete. This dorsal vagal survival imperative occurs most often in the very young or preadolescent where the individual has few resources to address traumatic triggers.

When impulses to run or fight for survival are thwarted, they may lead to physiological overwhelm through the action of the

parasympathetic dorsal vagal branch of the polyvagal network to shut the nervous system down for survival (Porges, 2011). For the immature infant brain, when overwhelm occurs, somatic-attachment-soothing by the primary attachment figure is a key feature of regulation. When twin infants are separated from their primary attachment figure and each other after trauma, attachment dynamics may be compromised leading to loss of attachment neuroception and displaced traumatic sibling attachment, a suboptimal attachment style. Maternal somatic-attachment-soothing may be replaced with mutual sibling somatic-attachment-soothing, this being the forerunner to suboptimal attachment hierarchy in adolescent pair bonding.

In his observations regarding right brain-to-right brain interpersonal neurobiological connectedness in early childhood between infant and caregiver, Schore (2019a) reinforced the importance of secure phylogenetic attachment by noting that "an expansion of the child's right brain regulatory coping capacities . . . underlies the developmental principle that secure attachment is the primary defense against trauma-induced psychopathology" (p. 42). He noted further that "caregiver-induced relational trauma is qualitatively and quantitatively more potentially psychopathogenic than any other social or physical stressor (aside from those that directly target the developing brain)" (p. 43).

Postpartum separation is relationally traumatic for both participants in the primary attachment dyad due to the loss of mutual regulation (Schore, 2019a; Tronick, 2007). In his seminal, maternal still-face observations, Tronick, (2007) concluded:

> Boys, compared to girls, are more demanding social partners, have more difficult times regulating their affective states, and may need more of their mother's support to help them regulate affect. This increased demandingness would affect the infant boy's interactive partner. (p. 345)

When a distressed infant brain tries to regulate in isolation from their primary caregiver, attuned connectedness and attachment neuroception are compromised, the child overwhelms into dorsal vagal shutdown, and social development is compromised, leading to dyadic trauma. (See Tronick, 2007, for his maternal still-face behavioral experiments.)

Riordan and his colleagues (2017) in their discussion of toddler trauma described the dynamics of attachment neuroception as follows:

> The primary attachment figure (PAF) acts as the ventral vagal, social engagement, soothing system for an infant's nervous system until it is myelinated and can regulate itself. The PAF relays to the toddler whether situations and people are safe, dangerous, or life threatening. Attachment neuroception is particularly relevant in toddler soothing during medical procedures where the toddler's unmyelinated neurosurvival networks cannot independently neurocept during experiences of pain, immobilization, and fear. (p. 46)

Attachment–neuroception, a coregulating process (Tronick, 2007), may be phylogenetically ordered as a survival behavior compelling a parent, when stimulated by the distress cries of their infant, to seek proximity and initiate protection and soothing. Traumatic arousal of the *fear and defense cascade* described by Kozlowska et al. (2015) may be arrested by attachment neuroception at the point of fear arousal thereby avoiding toddler trauma altogether, an important reason to have emotionally regulated parents involved in postoperative recovery.

Schore (2003) noted that trauma impacts attachment most significantly during life cycle events: "Early failures in dyadic regulation . . . skew the developmental trajectory of the corticolimbic systems that mediate the social and emotional functioning for the individual for the rest of the life span" (p. 33). This regulatory failure was described by Reite and Capitanio (1985, cited in Schore, 2003, p. 33) as "*impaired autonomic homeostasis* disturbances in limbic activity, and hypothalamus dysfunction".

Monozygotic twins, in traumatic circumstances, may share a level of dyadic attunement that goes beyond normal secure sibling attachment dynamics to a platform of attuned connectedness where one twin may experience the sensations, symptoms, and experiences of traumatic overwhelm in a state of sympathetic arousal for the other, who painlessly remains in the state of dorsal vagal numbness. (See Figure 3.)

Monozygotic Attunement

This phenomenon may offer insights into the dynamics of attachment including the phenomena of dyadic trauma, dyadic completion, and attachment neuroception (Porges, 2011; Riordan et al., 2017). Autonomic evidence of neurogenic congruence may validate the constructs of monozygotic attachment, monozygotic attunement, and secure phylogenetic attachment.

In pre- and posttreatment studies, positive changes in PTSD after treatment are deemed self-evident if there are increases in heart rate variability (HRV) and the three HRV index variables, HF power, RMSSD, and LF/HF ratio. HRV is the variation of heart beats in duration and intensity. More variable heart rate is an indication of wellness and wellbeing. RMSSD, or the root mean square of successive differences between heart beats, reflects the beat-to-beat variance in heart rate and is the primary time domain measure used to estimate the vagally mediated changes in reflected HRV.

The HRV spectrum contains two major components—the high frequency (HF) component, which is synchronous with respiration and is identical to respiratory sinus arrhythmia (RSA), and a low frequency (LF) component that appears to be mediated by both the vagus and cardiac sympathetic nerves.

The LF/HF ratio (the ratio of low frequency to high frequency power in HRV) may estimate the ratio between the sympathetic nervous system and the parasympathetic nervous system under controlled conditions.

Reduced heart rate, fewer sleep disturbances, and increased sleep duration may also indicate improvement from PTSD after treatment. Congruence of HRV changes in monozygotic twins

may verify the constructs of secure phylogenetic attachment as the antithesis of trauma, monozygotic attachment, and monozygotic attunement.

Method

Research Participants

Monozygotic male twins (aged 13 years; T1 and T2) presented with anger, fear vigilance and avoidance of medical procedures, and in fear vigilance avoided routine health examinations with their GP. Episodes of vasovagal syncope in connection to minor procedures were common.

T1 presented with childhood PTSD due to several early childhood traumas. T2 experienced no actual trauma but experienced PTSD symptoms in monozygotic attunement with T1, including dorsal vagal collapse when T1 was exposed to trauma triggers, particularly during medical procedures. T2 was diagnosed as experiencing dyadic trauma.

The first-born twin, T1, displayed episodes of childhood PTSD, anxiety, and depression, manifest as hypervigilance, aggression, and oppositional behavioral traits predominately directed at his mother. The twin pair and all family members agreed that T1 had formed a suboptimal attachment with his twin brother in early childhood, evident in his inability to be soothed by anyone but T2. T1 displayed lower tolerance to incongruence than T2, as was evident from his vigilance, oppositional behavior, emotional overwhelm (anger and fear responses), and verbal aggression. The twins' physical appearance was more dizygotic than monozygotic in that they looked different, T1 being leaner and somewhat emaciated compared to T2, who was physically more robust. Neither twin could open the other's phone with facial recognition.

T1 experienced foetal distress (suboptimal development), birth trauma (postpartum separation), and early childhood impact trauma (a broken arm with complications) and postsurgical traumatic episodes at 6 years old. T2 did not experience trauma but experienced T1's trauma symptomology in his monozygotic attunement with his traumatized twin.

Both T1 and T2 were experiencing vasovagal syncope in response to minor medical procedures: T1 was more likely to respond to life stressors with unpredictable shifts in hyperarousal with aggression, or hypoarousal, and T2 reported somatic attunement with sensate experience of T1's physiological states. (See Figure 3 and Table 1.) T1 also described somatic numbing when triggered in PTSD episodes while his brother experienced the physical sensations and sympathetic arousal symptoms of PTSD.

Because T1 suffered foetal, birth, postpartum, and early childhood trauma, the twins experienced suboptimal maternal and monozygotic attachment. T2 had no trauma, a normal birth, and developed secure attachment with his mother. However, T2 experienced T1's trauma vicariously through the interpersonal neurobiological pathways (Siegel, 2012) of somatic attunement and connectedness. These circumstances were the precursors of T2's sibling dyadic trauma with T1. Further, their constant physical proximity and monozygotic attunement strongly influenced their sibling attachment style where throughout their childhood T2 soothed T1. In this suboptimal attachment dynamic, monozygotic attunement may have been reinforced by T1's heightened need for soothing, resulting in suboptimal displaced traumatic sibling attachment.

The twins' mother and family members were agreed that T1 has a primary attachment relationship with T2 rather than his mother and that the twins' monozygotic attachment had been evident from birth. For T1 and T2, their somatic congruence was not just DNA-based concordance but was also grounded in their unusual levels of somatic-attachment-soothing and monozygotic attunement resulting in many specific attachment dynamics. For example, as newborns, neither twin would settle until they were co-sleeping, an arrangement that continued through most of their preschool and middle childhood. The twins are fiercely loyal and supportive of each other above maternal, paternal, non-twin sibling, and friendship relationships. Predictably, early preadolescent pair bonding relationships generated considerable attachment rupture dynamics for the twins, who each opposed the other's romantic choice, compromising this developmental stage of procreative pair-bonding.

Familial Trauma History

In her first trimester, the twins' mother was advised to terminate T1 due to the pregnancy being monochorionic–diamniotic (i.e., having different amniotic sacs but sharing the same placenta). T1 was being undernourished and was therefore in a state of survival stress from conception. This was T1's first survival trauma. Neurostructural growth and the developmental impact of foetal undernourishment on attachment as predeterminants of attachment perturbation and trauma is speculative. Postpartum and childhood trauma and stress response to threat vulnerability in childhood and adult life is well reported in the literature on the other hand. In their meta-analysis of adverse childhood experiences (ACEs), Karen Hughes and colleagues (Hughes et al., 2017) observed:

> Physiological and biomolecular studies are increasingly establishing how childhood exposure to chronic stress leads to changes in development of nervous, endocrine, and immune systems, resulting in impaired cognitive, social, and emotional functioning and increased allostatic load (i.e., chronic physiological damage). . . . Thus, individuals who have ACEs can be more susceptible to disease development through both differences in physiological development and adoption and persistence of health-damaging behaviours. (p. 2)

Delivered by induction at 36 weeks due to maternal hypertension and concerns of preeclampsia, T1 was significantly undersized (at 2,440 g) and underdeveloped by comparison to T2 (normal birth weight). He was placed in an incubator and separated from his mother for the first three weeks of his life. In order to develop a phylogenetic attachment bond individuals must share proximity.

Separated from his mother, T1 did not form a phylogenetic attachment bond with her and instead formed a primary attachment bond with T2. This traumatic separation represents the second ACE, with traumatic attachment perturbation preceding dyadic trauma. Notably, later maternal soothing yielded poor parasympathetic responses in T1, and neither twin could emotionally regulate until they were placed together in the incubator where in the face–heart connection they settled into parasympathetic (rest and digest) state and peaceful sleep.

In the early weeks of life, therefore, T1 was regularly deprived of maternal soothing, tube-fed with breastmilk, and isolated from his mother and T2 for long periods. In the dynamics of early childhood traumatic attachment, it is contended that T1, in an overwhelmed survival state of dorsal vagal shutdown due to suboptimal face–heart connection with his mother, attuned to his more regulated twin. Consequently, T1 became somatically and emotionally attached to T2 rather than to his mother, which goes toward explaining the unusual somatic attunement between the twins. This dynamic has sponsored coining the terms *monozygotic attunement* and *monozygotic attachment* to describe the interpersonal neurobiological dynamics of monozygotic twin attachment that appears to involve a stronger face–heart connectedness than seems to be the case with dizygotic twins and non-twin siblings. Monozygotic attachment correlates with phenotypic similarity because monozygotic twins share the same DNA.

Sibling caregiving/comfort-seeking attachments are suboptimal in that caregiving is inadequate and dis-regulatory for the comfort seeking sibling and places a significant burden of emotional overwhelm on the caregiving sibling. Riordan and his colleagues (Riordan et al., 2019) observed: "The overwhelm of burdensome caregiving and inadequate neuroception from an underdeveloped older sibling caregiver, who cannot adequately soothe, compromises sibling relationships throughout their life cycle. Mutual sibling caregiving perturbs immature nervous systems leading to traumatic overwhelm and dyadic trauma" (p. 76).

Maternal separation equates to abandonment for the infant's nervous system. T1's life-preserving postpartum separation from his mother and his isolation compromised his attachment to his mother and influenced his monozygotic sibling attachment dynamics from independent/mutually supportive to dependent/exclusive sibling interpersonal attachment styles. Suboptimal sibling attachment severely compromises resilience (Kain &Terrell, 2018) and can impact sibling attachment neuroception throughout the life cycle (Riordan et al., 2019).

A common interpersonal difficulty for monozygotic twin pairs occurs during the period of mid to late adolescence when procreative attachment bonds are formed, as each twin individually seeks their life partner. Because monozygotic twins are more concordant than dizygotic twins (Torgersen et al., 2005) and genetic relatedness has a profound impact on a person's attachment hierarchy and the relative rank of the romantic partner, monozygotic twins have been found to prefer their twin sibling to their romantic partner (Schwarz et al., 2015). Monozygotic twin pairs are often so attached to each other they have difficulty forming external secure procreative attachment bonds, preferring their twin to their procreative partner; conversely, they may be unreceptive to third party involvement in their monozygotic twinship.

T1's Trauma History

At 6 years old, T1 experienced a complex arm break requiring surgery and several follow-up medical procedures. Despite his father being present, T1 experienced postsurgical trauma and overwhelm in isolation after surgery as he was already compromised in his attachment style with his twin as a suboptimal primary attachment figure. T2 was not present to assist in soothing during these procedures. Primary attachment figure somatic-attachment-soothing is imperative preceding and immediately after surgery to regulate emotional and neurological overwhelm (Riordan et al., 2017). T1 experienced several postsurgical traumas, a common occurrence among early childhood postsurgical presentations (Riordan et al., 2017, 2019).

At T1's first procedure to remove and replace his plaster, T1 experienced traumatic overwhelm manifest as vasovagal syncope, believing the technician *was going to cut my arm off*. Outside in the waiting room, T2 simultaneously experienced sympathetic vasovagal syncope corresponding to T1's dorsal vagal collapse. This episode was recorded as a critical incident by hospital medical staff. T1's dorsal vagal episode may have occurred because he was neurogenically primed by foetal, birth, postpartum, and postsurgical trauma (i.e., multiple early childhood adverse experiences) and therefore neurogenically more vigilant and survival–reactive at the time he was having his plaster removed. The same dynamics may have also influenced T2's collapse due to their shared monozygotic attunement. However, T2's collapse synchronous with T1's vasovagal syncope while physically separated cannot be adequately explained.

In summary, T1 presented with a heavy burden of foetal, infant, and early childhood trauma experienced through foetal distress trauma (undernourishment), birth trauma, postpartum separation attachment trauma, early childhood impact injury trauma (complex arm break), and surgical and postsurgical trauma resulting in vasovagal syncope and multiple episodes of re-traumatization from routine medical procedures such as visits to the GP. In T1's most recent episode that precipitated his attendance at therapy he had run away (PTSD-related avoidance manifest as a flight response) when overwhelmed during a routine visit to his doctor.

Diagnosis

Separation from attachment figures during fear vigilance and forced immobility (as in postsurgical procedures) can generate dyadic trauma. T1 was diagnosed with childhood PTSD with emerging mood and anxiety disorders in early adolescence, characterized by sympathetic hypervigilance, avoidance of medical procedures, and parasympathetic

hyperarousal leading to vasovagal syncope (dorsal vagal shutdown). T2's sympathetic vasovagal syncope with his twin implies that trauma in attachment dyads is contagious (Riordan et al., 2019) and can result in compromised interpersonal neurobiological connections beyond normal sibling attachment parameters yet to be fully understood.

Similarly, at presentation, T2 was also experiencing avoidance of medical procedures. Whenever T1 experienced onset of vasovagal syncope at medical procedures, T2 would also faint when in proximity to T1 in these situations. T1 explained that he preferred to have T2 with him at his doctor's visits because *T2 would faint and I would feel numb*. This curious attachment dynamic may be evidence of their monozygotic attunement and the contagious nature of trauma.

During T1's triggered episodes, T2 displayed symptoms of arousal typical of PTSD that culminated in vasovagal syncope while T1 displayed either arousal in the flight/fight response or somatic numbing (freeze) typical of shutdown in the dorsal vagal complex (Porges, 2011). T2's adverse hyperarousal and PTSD symptomology corresponds with the traumatological construct of trauma as a contagion in attachment dyads. T2 was diagnosed with dyadic trauma.

The twins and their mother agreed that T1 had formed a primary attachment bond with T2 from birth that was only now being challenged due to emerging phylogenetic imperatives for preadolescent pair bonding and drive toward independence. T1 had significant difficulty regulating his emotions in preadolescent pair-bonding relationships. T2 appeared to be emotionally better equipped to sustain the complexity and interpersonal challenges of preadolescent pair bonding. Neither twin warmed to the other's romantic partner and both were extremely critical and negatively oriented regarding their sibling's romantic involvement (attachment) with another. This interpersonal dynamic did not emerge between their older sisters when they explored procreative pair bonding. The twin pair was diagnosed as having suboptimal traumatic sibling attachment style.

Pretreatment Behavioral Experiment: Initial Testing of the Constructs

At presentation, the twins were reporting an atypically attuned somatic congruence to trauma triggers. Their assertion was that when T1 was stressed T2 also experienced autonomic symptoms of arousal manifest as somatic congruence. To test the twins' assertion of somatic congruence, a simple behavioral experiment was devised.

T1's sympathetic arousal (heart rate, beats per minute) was simultaneously measured and compared to T2's heart rate at the onset of T1's mild stress response. Each wearing a Fitbit fitness tracker to measure heart rate, the twins were separated into different rooms. They were instructed to simply record their heart rate at 60-second intervals with no preamble or indication of the nature of the experiment. Heart rate for both twins was recorded every minute for 10 minutes. At 4-minute intervals, and unknown to T2, T1 (who remained with the experimenter) was asked to recall his traumatic memories. Predictably, T1's heart rate increased at each 4-minute interval during recall of traumatic events. (Table 1) Surprisingly, T2's heart rate also increased at the exact same time intervals as T1. T2 was unaware of T1's experience in the other room and neither twin had preexperimental knowledge of the purpose of the experiment.

Table 1

Heart Rate Changes: Behavioral Experiment

Description of Trauma	T1. BPM	T2. BPM
	77	81
Arm break injury (6 years)	79	85
	78	80
	76	81
	76	80
Plaster taken off	80	83
	79	79
	77	80
	77	79
Bullying in school	82	80

Note. Heart rate changes in beats per minute (BPM) were recoded on Fitbit devices, over ten minutes, in 1-minute intervals, where T1 was asked to recall his traumatic events every four minutes. T2 was in another room recording his heart rate every minute for the duration of the experiment and did not know the nature of the experiment. At every *description of trauma* recall the heart rate of both twins increased.

Although this experiment is not statistically powerful enough to confirm the twins' monozygotic attunement and somatic congruence, it does validate their claim of somatic congruence in some measure. Given that heart rate is a reactive autonomic function, these results indicate somatic attunement consistent with the family's assertion of the twins' somatic impact on each other. As Table 1 shows, T2's heart rate is consistently higher than T1's heart rate, validating his reported traumatic congruence state of monozygotic attunement with T1. Considering that elevated heart rate is an autonomic symptom of PTSD, this experiment supports the diagnosis of dyadic trauma for T2 and the construct that trauma is contagious in attachment relationships.

Self-Report Expression of Monozygotic Attunement

The twins were then asked to draw their physical sensations while T1 silently recalled the trauma of having his plaster removed. Drawing on paper silhouettes while facing away from each other (i.e., no face-to-face communication), the twins expressed their somatic perturbations. T1 (shown on the left of Figure 3) expressed only minimal somatic awareness while thinking of his trauma, indicating dorsal vagal shutdown (somatic numbing). This is typical of traumatic presentations in a state of shutdown, or numbness, in the parasympathetic dorsal vagal complex. T2, however, in his monozygotic attunement with his twin, had a much broader somatic experience of heightened sensate awareness of somatic perturbation indicating his monozygotic attuned connection with his twin. Interestingly, T2's depiction of arm perturbation is a mirror image of his brother's with heightened awareness. Self-report behavioral experiments do not offer scientific rigor; however, these authentic, unsophisticated expressions of somatic states adequately demonstrate this twin pair's unique monozygotic attachment dynamic.

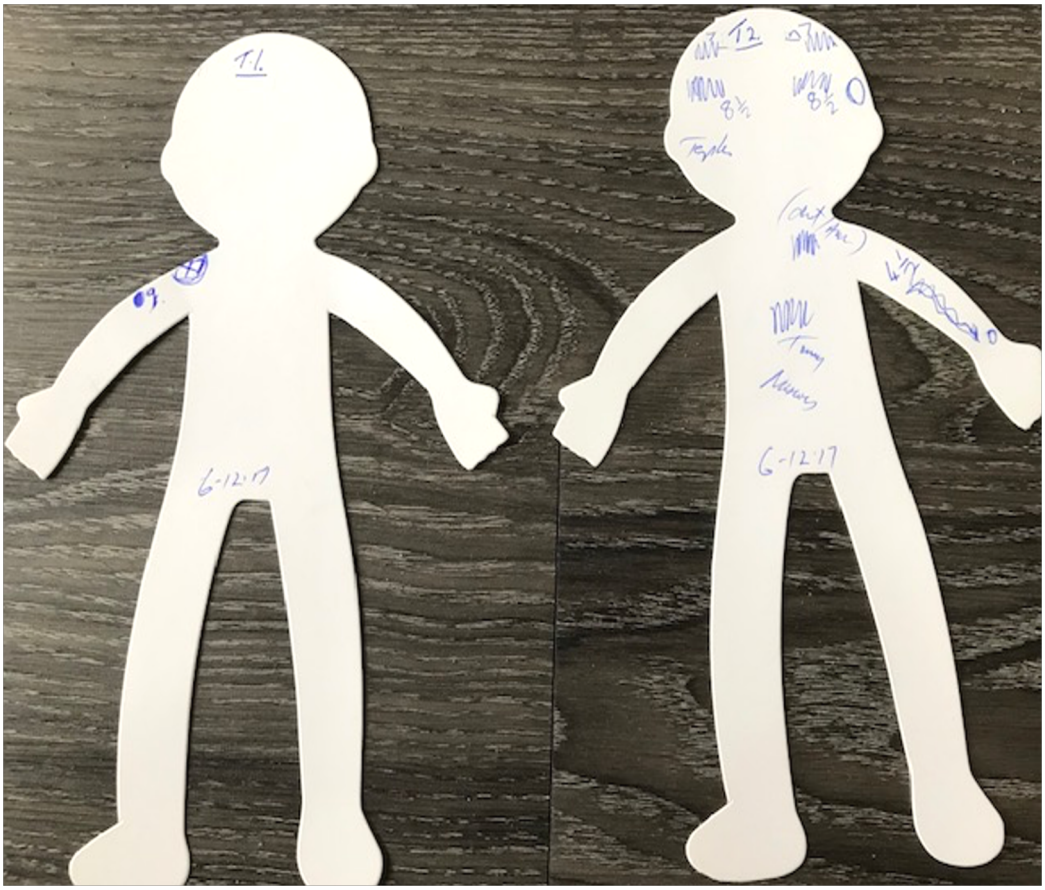

Figure 3 *T2's Monozygotic Attunement in Response to T1's Medical Trauma*

Note. Depictions of dyadic trauma experienced somatically by T2 (right) while T1 (left) silently reflected on his arm injury at age 6, dramatically illustrating the somatic connection and interpersonal neurobiology of dyadic trauma and monozygotic attunement.

Heart Rate Variability and Quantifiable Assessment of Autonomic Indicators of Trauma

Self-report and psychometric assessment of PTSD and trauma symptomology have limitations in that they are intrinsically subjective.

Quantifiable measures of autonomic functions as indicators of trauma responses may offer internal validity in controlled pre- and posttreatment measures. As the physiological marker of variation in the time interval between heartbeats, HRV is considered a measure of the autonomic nervous system functioning and reflects an individual's ability to adaptively cope with stress (Negpal et al., 2013).

In their meta-analysis of HRV index variables as indicators of PTSD, Negpal and colleagues (2013) concluded that three HRV index variables—HF power, RMSSD, and LF/HF ratio—are key indicators of PTSD symptomology that can provide quantifiable evidence pre- and posttreatment of recovery. "Reductions in HF power, RMSSD and LF/HF ratio have utility as indicators of autonomic effects of PTSD, which can be associated with impaired vagal activity" (Negpal et al., 2013, p.1), and conversely, increases in HF power, RMSSD and LF/HF ratio indicate positive reduction of PTSD symptoms after treatment.

Together with the HRV index variables described above, heart rate, sleep duration, and sleep disturbances were also identified as objective measures of trauma responses used in this study.

Smartphone connected devices that measure autonomic function, despite limited validation of their experimental utility to date, are a portable and easily accessible option for measuring autonomic measures and can be readily adapted to inform treatment outcomes in clinical settings.

In his discussion of infant development in relation to RSA, a measure which is directly proportional to HRV, Porges (2011) noted:

> As the infant matures, the special visceral efferent pathways are recruited by corticobulbar pathways and expressed through social engagement behaviors. Autonomic support for these muscles is provided by the myelinated vagus, which can be dynamically monitored by quantifying RSA (*respiratory sinus arrhythmia*). This face–heart connection provides the necessary elements for an integrated social engagement system. (pp. 123–124)

Van Boxtel et al. (2018) advocated for the use of HRV measures in studies of individuals at risk of developing symptoms of PTSD, noting the following:

> During awake resting states, PTSD patients are characterized by low parasympathetic tone, relative to healthy controls, resulting in elevated mean heart rates and reduced cardiac reactivity. By contrast, during sleep, PTSD patients appear to be characterized by increased sympathetic activation, mainly observed during REM sleep, again with elevated mean heart rate and reduced reactivity, as a consequence. (p. 1)

Based on the findings of Porges (2011) and Negpal et al. (2013), HRV may be a measure of sympathetic/parasympathetic homeostatic states in PTSD. When HRV is measured with computer applications, smartphone devices can offer convenient measures of change over time and objective measures in pre- and posttreatment research. A comparison of monozygotic twins who share the same DNA and are more concordant than other siblings offers a unique research opportunity to evaluate the clinical validity of treatment based on measures of autonomic variables of HRV, HRV index variables, heart rate, sleep duration, and sleep disturbances.

Porges (2011) proposed that HRV, bradycardia, and RSA are mediated by separate branches of the vagus and therefore are distinct measures of cardiac vagal tone. Vagal tone—the functional relationship between the brainstem and the heart (Porges, 2011)—has two roles, first, to foster physiological homeostasis to promote growth and restoration (rest and digest), and second, during environmental challenges, to act as a brake (the vagal brake) to rapidly regulate cardiac and metabolic output (flight/fight/ freeze). The phylogenetic purpose of the vagal brake "provides a neurophysiological mechanism that may promote the development of appropriate social behavior" (Porges, 2011, p. 106).

By this definition, therefore, HRV is a measure of the action of the vagus nerve and the vagal brake and may indicate whether a subject is in a state of arousal (flight or fight), hypoarousal (freeze, shutdown), or homeostasis (rest and digest). Aroused states of flight, fight or freeze in PTSD are represented in an affective and behavioral cascade of escalating fear avoidance or aggression, neither of which are conducive to secure phylogenetic attachment or social cohesion. However, when someone who has PTSD displays consistent low heart rate, they might be experiencing a sustained state of bradycardia. This may be particularly true of those who report numbness when triggered in their trauma where somatic overwhelm is followed by vasovagal syncope.

HRV Research

Mather and Thayer (2018) observed that "individuals with high HRV tend to have better emotional well-being than those with low heart rate variability" (p. 1). Fear, anger, and overwhelm may correlate with the trauma states of flight, fight, and freeze and might be identified in low HRV measures.

In a recent review and meta-anaylsis of PTSD and HRV studies, Ge et al. (2020) concluded: "Given the relationships among low vagal state, inflammation, and alterations in brain structure and function, including executive function and emotion regulation, reduced HRV may be regarded as an endophenotype in PTSD research" (p. 1).

In a meta-analysis to investigate links between the social brain and HRV, and the implications for psychotherapy, Petrocchi and Cheli (2019) demonstrated that increased HRV

predicts positive psychotherapy outcomes, summarized as follows:

- Social relationships have emotional and self-regulating properties.
- The experience of inter- and intrapersonal safeness is connected to prosocial motives, such as compassion, and the inhibitory function of the prefrontal cortex.
- Social relationships and compassion influence different body systems, such as the vagus nerve.
- Many forms of psychopathology represent the activation of evolved, defensive strategies especially in contexts where there are few stimuli indicating safeness and social support.
- Heart rate variability predicts psychotherapy outcomes.

These insights offer further evidence that increased HRV may reduce community psychopathology, loneliness, and social isolation and increase social cohesion, as well as being a reliable pre- and posttreatment measure of PTSD.

Research Procedure

Treatment

T1 was treated with SE and AF-SE. T2 did not receive treatment but was assessed for autonomic somatic congruence with T1 pre- and posttreatment.

After pretreatment baseline measures were taken, T1 was treated for avoidance of routine medical procedures with a single session of SE. The SE session targeted an activated memory of T1's trauma (a belief that the technician was going to cut his arm off) with interoception—the ability to sense internal states and bodily processes (Craig, 2009; Porges, 2011). During activation (accessing the somatic memory of trauma) and before discharge (the neurogenic release and reorganization of flight/fight/freeze responses in the sympathetic nervous system and polyvagal network), T1 pushed with his non-broken arm on a cushion as he recalled his fear of having his arm cut off as a 6-year-old, the SE therapist providing resistance to achieve realistic full arm extension to "push away the danger", somatically reenacting a survival response for completion (a neurogenetic survival imperative, the functional equivalent of running from danger or running to safety for toddlers). This *push work* simulated somatic completion (of flight/fight responses for survival) of T1's unresolved survival imperative to push away the threat. Somatic interoception and completion was followed by T1 shifting neurogenically from sympathetic arousal to parasympathetic homeostasis during *pendulation*. Pendulation is a key dynamic in SE and AF-SE for both the individual and the dyad, manifest as "the continuous, primary, organismic rhythm of contraction and expansion" (Levine, 2015, p. 55). During and after pendulation, T1 experienced a neurogenic shift from sympathetic arousal (hypervigilance) to parasympathetic (rest and digest) into a state of attuned quiescence (Riordan et al., 2019), an autonomic, phylogenetic, somatic shift toward homeostasis.

Although T2 was not treated, vasovagal syncope ceased immediately for both boys, and vigilance around medical procedures gradually abated until they were able to visit their GP with minimal arousal. This therapeutic outcome is significant behavioral evidence that highly intrusive dorsal vagal symptomology of trauma can be resolved in a single session with SE. Similarly, resolution of dyadic trauma with SE is an effective treatment in attachment dyads because the non-traumatized individual may experience recovery automatically. When SE is targeted at the traumatized attachment dyad, dyadic trauma can reduce automatically, or specific AF-SE sessions may be required.

T1 was then treated with AF-SE to redress the twins' suboptimal sibling attachment style and the specific attachment ruptures regarding third-party intrusions (first romantic relationships) in their monozygotic attachment dynamics. AF-SE was conducted with T1 tracking his somatic responses of the experiences of attachment rupture with T2. This included the developmental changes the twins were experiencing around inclusion of procreative partnerships in their twin–pair relationship. The AF-SE treatment process consisted of T1's interoception of his experience of incongruence, that being the twins' opposition to each other's girlfriends. Similarly, T1 was invited to somatic interoception on issues of separation in class, more individual time with friends and girlfriends, and less time with T2 at home. These *separation activations* challenged the twins' suboptimal monozygotic attachment style and represented a triggered response of fear vigilance around separation. Separation for procreative purposes in monozygotic twin dyads is a secure phylogenetic attachment process. Adolescent monozygotic twin pairs need to be inclusive of procreative partners while maintaining the security of their monozygotic dyad to successfully enter the next developmental stage of life, namely establishing secure phylogenetic partnerships in a procreative pair-bond for child rearing.

Post-treatment, the twins happily separated into different classrooms for the first time and stress over separation ceased altogether. Conflict between the twins over girlfriends remained, however.

Ethical Considerations

This study followed the Australian Psychological Society ethical guidelines for human subject research. Consultation with a senior psychologist and faculty member of Somatic Experiencing International confirmed the degree of risk for the participants was minimal. Informed consent was obtained from both parents and both twins, with an option to decline at any time for all or any component of the research or treatment. The twins were keen to participate in the research and eager to learn how their nervous systems could be used as feedback for health and recovery from their fear of medical procedures and fainting. The parents readily purchased the necessary devices, viewing the process as an opportunity for their sons to recover from PTSD, gain unique insight into their twinship, and resolve

their monozygotic dyadic trauma. Strict confidentiality was observed.

Research Measures and Devices

SE and AF-SE were applied to address PTSD and dyadic trauma. Pretreatment PTSD symptoms and posttreatment outcomes were investigated with smartphone devices employed to measure HRV and the HRV index variables of HF power, RMSSD, and HF/LH ratio.

Smartphone devices can measure changes in the autonomic somatic variables of HRV, HRV index variables, sleep disturbances, sleep duration, and heart rate, overt autonomic measures of neurogenic arousal complicit in PTSD. Similarly, pre- and posttreatment changes can be compared and inferences made on the constructs of dyadic trauma, dyadic completion, monozygotic attachment, monozygotic attunement, and secure phylogenetic attachment as the antithesis of trauma.

Smartphone devices can record nocturnal behavior within the four stages of sleep, including nocturnal waking and disturbances, REM sleep, light sleep, and deep sleep. In this study they were used to provide quantifiable measures of sleep disturbances (e.g., night terrors, night sweats, nocturnal waking) that are common features of PTSD presentations.

A baseline measure was conducted collecting data from both boys on HRV, HRV index variables, heart rate, sleep duration, and nocturnal disturbances. Pre- and posttreatment data were logged remotely into Elite-HRV Team Dashboard on the experimenter's computer for analysis.

Pre- and posttreatment statistical comparisons identified changes in HRV, HRV index variables, heart rate, sleep duration, and nocturnal disturbance.

Nocturnal waking and sleep duration were recorded and delineated between deep sleep, wakes, and other non deep-sleep wakes (Figure 4). Data were compared pre- and post-treatment for each twin and comparatively between the twins pre- and post-treatment. Data were collected from Fitbits and Elite-HRV monitoring devices (a chest-strap monitor, with measures taken once daily, after waking). Fitbits were worn 24 hours per day to record heart rate, and at night to record sleep disturbances. Deep-sleep disturbances indicated frequency of the PTSD symptoms of nightmares, night terrors, night sweats, or startled waking.

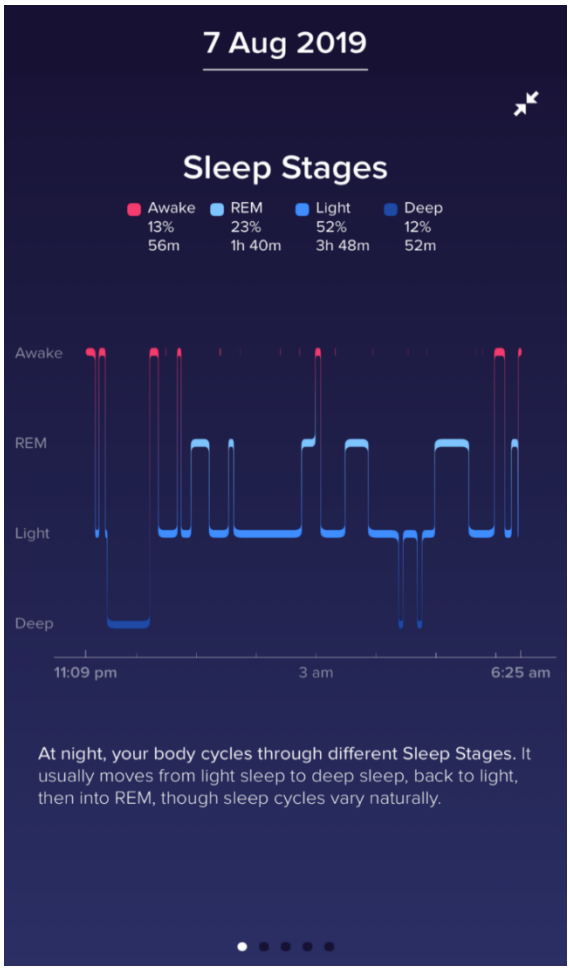

Figure 4 *Fitbit Example of Nightly Sleep Record*

Note. Example of sleep stages as recorded by smartphone device (Fitbit).

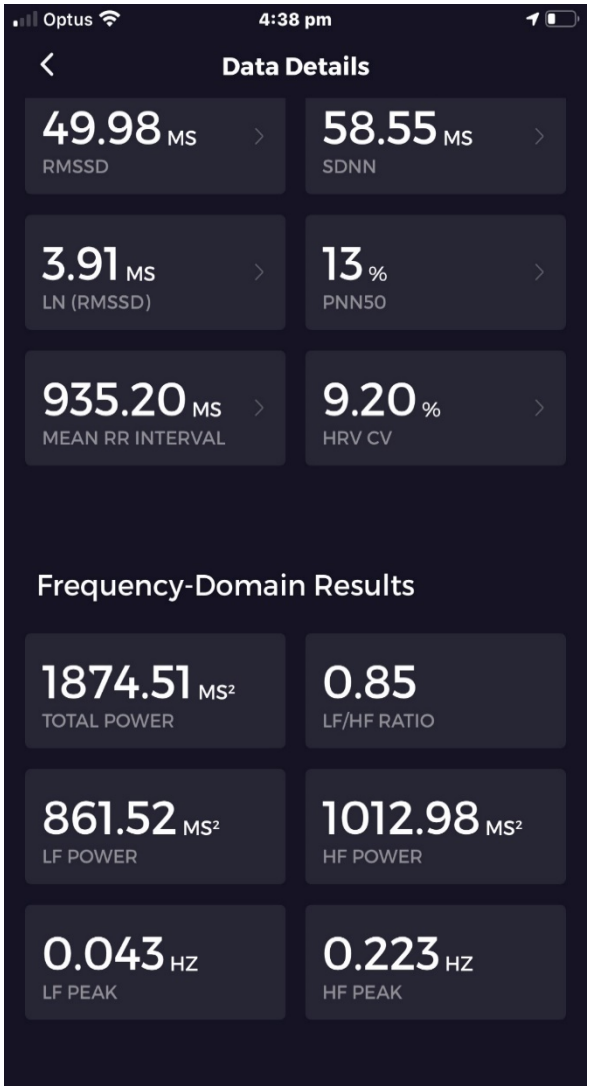

Figure 5 *Elite-HRV Team Dashboard (1)*

Elite-HRV Device

HRV index variable scores were recorded daily and automatically logged online on the researcher's Elite-HRV Team Dashboard—see Figures 5 and 6 for examples. The twins used an earlier software format than the examples below. Online logging of data remined the same.

Figure 6 *Elite-HRV Team Dashboard (2)*

Statistical Method

T1 and T2's autonomic indicators of PTSD and dyadic trauma were compared pre-treatment to determine differences and again post-treatment to determine treatment outcomes. T1 and T2 were each individually assessed pre- and post-treatment.

Increased HRV and HRV index variables are measures of recovery after treatment for PTSD, therefore, the primary dependent variables were HRV and three HRV index variables: HF power, RMSSD and LF/HF ratio. Separate measures of PTSD symptomology—heart rate, sleep duration, and sleep disturbances—were also taken. Differences between T1 (PTSD twin) and T2 (control) were tested to determine whether PTSD symptoms varied prior to T1 receiving treatment.

Comparisons were made between T1 and T2 post-treatment to determine the effect of SE and AF-SE as treatments for PTSD and dyadic trauma. Pre- and posttreatment results were compared individually for T1 and T2. Similarly, the impact of AF-SE on dyadic trauma was considered regarding T2's pre- and posttreatment results. Inferential analysis considered the twin pair's somatic congruence, monozygotic attunement, dyadic trauma, dyadic completion, and implications for their attachment style as they developed into the procreative stage of their life cycle. Inferences were made on the data for global issues of widespread psychopathology, loneliness, social isolation, and loss of social cohesion in communities.

Hypotheses for Statistical Analysis

It was anticipated that after treatment, T1 would have higher HRV scores and higher HRV index scores than before treatment. It was also expected that after treatment T1 would have lower heart rate, more sleep, and fewer nocturnal disturbances, confirming the efficacy of SE and AF-SE as treatment for PTSD and trauma symptomology.

Autonomic measures of recovery are determined primarily with an increase in the three HRV index variables (HF power, RMSSD, and HF/LF ratio). Corresponding increases in HRV scores will confirm recovery.

Corresponding autonomic measures of PTSD symptomology (dyadic trauma) for T2 before treatment will support the construct of trauma as a contagion generating dyadic trauma.

T2's recovery from dyadic trauma after treatment, measured by the same parameters as T1's recovery, will support the hypothesis of dyadic completion. Statistical evidence of somatic congruence in the twinship will support the hypothesis of monozygotic attachment, monozygotic attunement, and the construct of secure phylogenetic attachment as the antithesis of trauma.

Hypotheses

1. Post-treatment, T1 will have higher HRV index scores (HF power, RMSSD, and LF/HF ratio), higher HRV scores, lower heart rate, more sleep, and fewer nocturnal disturbances than his pre-treatment scores.
2. Post-treatment, T1 will have higher HRV scores, higher HRV index scores (HF power, RMSSD, and LF/HF ratio) lower heart rate, more sleep, and fewer nocturnal disturbances than T2 who experienced dyadic trauma but did not receive treatment.
3. Post-treatment, T2 will display somatically congruent autonomic changes with T1 in HRV, HRV index scores (HF power, RMSSD, and LF/HF ratio), heart rate, sleep duration and nocturnal disturbances.
4. Post-treatment, T2 will have higher HRV scores, higher HRV index scores (HF power, RMSSD and LF/HF ratio) lower heart rate, more sleep, and fewer nocturnal disturbances than his pre-treatment scores, confirming the efficacy of AF-SE in the treatment of dyadic trauma and validating the construct of secure phylogenetic attachment as the antithesis of trauma.

Descriptive Statistics

There were 41 observations per variable for T1 prior to receiving treatment. For T2, there were 47 observations per variable prior to receiving treatment. Following treatment, T1 had 34 observations and T2 had 33 observations. There were four outliers identified (from the same observation number). Therefore, one case was removed from the LF power data, the total power data, and the RMSSD data. SPSS Version 24 and two types of *t* tests were used: independent samples *t* test and paired samples *t* test. HRV, HRV index variables, and the non-HRV, PTSD-related indicators (heart rate, sleep duration, and sleep disturbances) were examined before and after treatment for each twin, separately and then comparatively.

First, pretreatment HRV, HRV index variable scores and non-HRV, PTSD-related indicators (heart rate, sleep duration, and sleep disturbances) were compared between T1 and T2, establishing the baseline for the twins' PTSD indicators and the nature of their monozygotic attachment and monozygotic attunement.

To compare the outcomes pre- and post-treatment for each twin individually, a series of paired-samples *t* tests was conducted. Pretreatment scores were compared to posttreatment within the same twin. For this set of analyses, the paired samples *t* test was the most appropriate because the dependent variables were continuous and the independent variables were related groups (i.e., the same subjects were present in both conditions). Prior to the analyses, the data were assessed for outliers. Values that were three or more standard deviations above or below the mean were removed.

Second, outcomes between T1 and T2 pre-treatment were compared by conducting a series of independent samples *t* tests. The independent samples *t* test was the most appropriate for these analyses because the independent variable consisted of two categorically independent groups (i.e., T1 vs. T2). Additionally, the assumption of independent observations was accounted for. This means that the participants in one group were not also participants in the other group.

Next, posttreatment HRV, HRV index variable scores, and non-HRV, PTSD-related indicators (heart rate, sleep duration, and sleep disturbances) were compared between T1 and T2.

Statistical Results
T1 Pre- and Post-Treatment

Pre- and posttreatment scores were compared for T1 (Table 2). It was hypothesized that, post-treatment, T1 would have higher HRV scores, higher HRV index scores (HF power, RMSSD, and LF/HF ratio), lower heart rate, more sleep, and fewer nocturnal disturbances than his pretreatment scores.

Table 2

T1 Pre- and Posttest Scores for all HRV and Non-HRV Indicators

	T1			
	Pre-Treatment	**Post-Treatment**	***t***	***p***
HRV	61.46 (4.51)	63.56 (3.04)	-1.345	.188
RMSSD	56.85 (18.10)	63.47 (13.05)	-0.927	.361
HF power	**926.75 (486.91)**	**1298.82 (605.31)**	**-2.151**	**.039**
LF/HF ratio	1.73 (0.90)	2.02 (1.11)	-1.619	.115
HR	**77.10 (4.98)**	**72.23 (6.61)**	**3.220**	**.003**
Sleep hours	6.86 (0.76)	6.66 (0.79)	1.156	.257
Wakes per night	2.07 (1.44)	1.70 (0.99)	1.302	.203
Wakes from deep sleep	**0.80 (0.85)**	**0.37 (0.56)**	**2.282**	**.030**

Note. Significant differences between pre- and post-treatment shown in bold.

For HRV scores there was only one significant difference in T1's pre- and posttest HRV indicator scores. T1 had an increase in HF power from pretest to posttest (i.e., higher HF power at post-test compared to pre-test), t (33) = -2.151, p = .039. Regarding the non-HRV PTSD symptomology, there were significant difference from pre-treatment to post-treatment for T1. There was a significant reduction in HR from pre- to post-treatment, t (33) = 3.220, p = .003. Additionally, there was a significant reduction in wakes from deep sleep, t (33) = 2.282, p = .030. Pre- and posttreatment scores support Hypothesis 1. The only statistic that did not conform with Hypothesis 1 was an insignificant reduction in sleep hours post-treatment.

Table 3

T2 Pre- and Posttest Scores for all HRV and Non-HRV Indicators

	T2 Pre-Treatment	**Post-Treatment**	***t***	***p***
HRV	59.23 (5.24)	58.70 (6.69)	1.287	.207
RMSSD	49.81 (17.08)	50.16 (28.01)	1.896	.067
HF power	896.67 (629.48)	919.02 (659.13)	0.728	.472
LF/HF ratio	**1.25 (0.79)**	**2.62 (1.75)**	**-4.347**	**<.001**
HR	**82.25 (7.34)**	**84.93 (11.56)**	**-2.012**	**.053**
Sleep hours	6.81 (0.90)	6.69 (0.95)	0.615	.543
Wakes per night	1.17 (0.87)	0.87 (0.57)	1.361	.184
Wakes from deep sleep	0.47 (0.63)	0.17 (0.53)	1.795	.083

Note. Significant differences between pre- and post-treatment shown in bold.

T2 Pre- and Post-Treatment

Pre- and posttreatment scores were compared for T2 (Table 3). It was hypothesized that, post-treatment, T2 would have increased scores in HRV and HRV index variable scores, lower heart rate, more sleep, and less frequent nocturnal disturbances. Somatic congruence with T1 was also predicted.

Against the predicted hypothesis, T2's HRV posttreatment score reduced non-significantly. All other posttreatment scores improved consistent with Hypothesis 2. T2's posttreatment results displayed congruence with T1's posttreatment scores and confirmed Hypothesis 3. There were two significant differences from pre-treatment to post-treatment. The LF/HF ratio significantly increased, t (32) = -4.347, p < .001 and HR increased = -2.012, p .052. Although inconsistent, the data trends to support Hypothesis 4. T2's reduced HRV score demonstrates that HRV scores can be influenced by all 12 HRV index variables described in Table 6 in conflict with the three primary HRV index variables known to indicate improvement after treatment. Despite having a lower posttreatment HRV score, T2 still showed improvement on posttreatment HRV index variables and nocturnal disturbances, indicating an improvement in trauma symptomology. Behaviorally post-treatment, T2 displayed reductions in symptomology (vasovagal syncope and avoidance of trauma triggers), indicating observable validation of the hypotheses.

Comparisons Between T1 and T2: Pre-Treatment

Pre-treatment outcomes were compared between T1 and T2 (Table 4). Differences between T1 (PTSD twin) and T2 (control) were tested to determine whether outcomes varied prior to T1 receiving the SE treatment. Prior to treatment, T1 had higher HRV, HRV indicator scores, and lower heart rate; T1 also woke more often than T2. These data tend to support the hypothesis that before treatment T2 experienced the somatic symptoms of PTSD (dyadic trauma) in his monozygotic attunement with T1.

Table 4

Significant Differences Between T1 and T2 Pre-Treatment

	Pre-Treatment T1	T2	*t*	*p*
HRV	**61.46 (4.51)**	**59.23 (5.24)**	**2.124**	**.037**
RMSSD	56.85 (18.10)	49.81 (17.08)	1.877	.064
HF power	926.75 (486.91)	896.67 (629.48)	0.248	.805
LF/HF ratio	**1.73 (0.90)**	**1.25 (0.79)**	**2.665**	**.009**
HR	**77.10 (4.98)**	**82.25 (7.34)**	**-3.795**	**<.001**
Sleep hours	6.86 (0.76)	6.81 (0.90)	0.223	.825
Wakes per night	**2.07 (1.44)**	**1.17 (0.87)**	**2.931**	**.005**
Wakes from deep sleep	0.80 (0.85)	0.47 (0.63)	1.731	.089

Note. Significant differences between pre- and posttreatment shown in bold.

Pre-treatment, T1 and T2 had significantly different scores on HRV and LF/HF power. HRV was significantly higher for T1 than T2, t (86) = 2.124, p = .037. Additionally, T1 had significantly higher LF/HR ratio than T2 pre-treatment, t (86) = 2.665, p = .009. There were no significant differences in pretreatment RMSSD or HF power between T1 and T2. T2's HR was considerable higher, which was consistent with T2's reported pretreatment sympathetic arousal states (see Table 1 above), which supports the construct of trauma as a contagion. Note that from a population of 72,000 Elite-HRV users, the average HRV score for males aged 18 to 25 years is 68.68 (https://elitehrv.com/) Pretreatment scores for both twins were significantly lower that the best known healthy average HRV scores, indicating both twins were experiencing trauma symptomology. These data support the research aim that trauma is manifest in dyadic trauma and is contagious in attachment relationships compromising secure phylogenetic attachment.

T1 and T2 Analyses: Post-Treatment

Posttreatment outcomes were compared between T1 and T2 (Table 5). In this way, differences between T1 (PTSD twin) and T2 (control) were tested to determine whether outcomes varied after T1 received the SE and AF-SE treatments. Table 5 displays the results from the independent samples t test comparison between T1 and T2 posttreatment scores.

Table 5

Differences Between T1 and T2: Post-Treatment

	Post-Treatment T1	T2	*t*	*p*
HRV	**63.56 (3.04)**	**58.70 (6.69)**	**3.848**	**< .001**
RMSSD	**63.47 (13.05)**	**50.16 (28.01)**	**4.957**	**< .001**
HF power	**1298.82 (605.31)**	**919.02 (659.13)**	**2.458**	**.017**
LF/HF ratio	2.02 (1.11)	2.62 (1.75)	-1.708	.092
HR	**72.23 (6.61)**	**84.93 (11.56)**	**-5.539**	**< .001**
Sleep hours	6.66 (0.79)	6.69 (0.95)	-0.111	.912
Wakes per night	1.70 (0.99)	0.87 (0.57)	**4.00**	**< .001**
Wakes from deep sleep	0.37 (0.56)	0.17 (0.53)	1.425	.159

Note. Significant differences between pre- and post-treatment shown in bold.

Following treatment, there were significant differences between T1 and T2 for HRV and two HRV indicator variables. T1 had significantly higher HRV scores than T2, t (65) = 3.848, $p < .001$. Additionally, T1 had higher RMSSD, t (64) = 4.957, $p < .001$, and HF power, t (65) = 2.458, p = .017, than T2 post-treatment. With the exception of T2's reduced HRV score, the data confirm overall improvement for both twins post-treatment and somatic congruence between the twins, albeit with a lesser treatment effect for T2. There was no significant difference between T1 and T2 on LF/HF ratio. The general trend of the data supports Hypothesis 1 and the construct of somatic congruence, monozygotic attunement, and monozygotic attachment between the twins. These data also support Hypothesis 3, confirming somatic congruence between T1 and T2 posttreatment, confirming that AF-SE is an effective treatment for dyadic trauma, and supporting the construct of dyadic completion.

The purpose of paired sample t tests is to determine whether there is statistical evidence that the mean difference between paired observations on a particular outcome is significantly different from zero. Table 6 compares statistical differences between T1's pre-and posttreatment scores and T2's pre- and posttreatment scores. Table 6 also highlights significant difference between T1 and T2.

Pre-treatment and posttreatment outcomes were compared between T1 and T2. In this way, differences between T1 (PTSD twin) and T2 (control) were tested to determine whether outcomes varied prior to T1 receiving the SE treatment, and if outcomes varied after T1 received the treatment.

Table 6

Paired Samples t Test Within Each Twin: Pre- and Post-Treatment

	T1		T2	
	t	***p***	***t***	***p***
HRV	-1.345	.188	1.287	.207
Morning readiness [a]	0.847	.404	-0.363	.719
HRV CV [b]	-0.530	.599	**-4.914**	**< .001**
HR	**3.220**	**.003**	-2.012	.053
In RMSSD [c]	-1.327	.194	1.329	.193
RMSSD	-0.927	.361	1.896	.067
Nn50 [d]	**-2.075**	**.046**	1.598	.120
Pnn50 [e]	**-1.385**	**.175**	**2.299**	**.028***
SDNN	**-3.558**	**.001**	-1.833	.076
LF power [f]	**-3.135**	**.004**	**-3.469**	**.002**
HF power	**-2.151**	**.039**	0.728	.472
LF/HF ratio	**-1.619**	**.115**	**-4.347**	**< .001**
Total power	**-3.111**	**.004**	-1.809	.080
Sleep hours	1.156	.257	0.615	.543
Wakes per night	1.302	.203	1.361	.184
Wakes from deep sleep	**2.282**	**.030**	1.795	.083

Note. Significant differences between pre- and post-treatment shown in bold.

[a] Morning readiness refers to baseline patterns of HRV measures.
[b] HRV CV is the coefficient of variation in HRV measures over weeks rather than days.
[c] In RMSSD refers to when a natural log is applied to the RMSSD in order to distribute the numbers in an easier to understand range.
[d] Nn50 is the mean number of times per hour in which the change in successive normal sinus NN intervals exceeds 50 ms.
[e] PNN50 is the proportion of Nn50 divided by the total number of NN (R-R) intervals exceeding 50 ms.
[f] LF power refers to heart rate frequency activity in the 0.04–0.15 Hz range.

Prior to receiving treatment, there were significant differences between T1 and T2 on several HRV and HRV index variables. T1 had significantly higher HRV in RMSSD, LF/HF ratio, total power, and wakes per night than T2. Additionally, T1 had a lower average heart rate compared to T2 prior to treatment. See Table 4 for more detailed comparisons between T1 and T2 prior to treatment.

For T1 prior to and following treatment there were statistically significant differences in HRV and HRV index variables ($p < .05$). There were reductions in heart rate and the

number of wakes from deep sleep. However, there were increases in Nn50, SDNN, LF power, HF power, and total power. There was no significant difference from pre-treatment to post-treatment for T1 on morning readiness, HRV CV, In RMSSD, Pnn50, LF/HF ratio, sleep hours, and wakes per night.

For T2, there were significant differences between pre- and post-treatment for HRV CV (an increase from pre- to post-treatment), Pnn50 (a decrease from pre- to post-treatment), and LF/HF ratio (an increase from pre- to post-treatment). These results may account for T2's slightly reduced HRV score post-treatment despite key HRV index variables increasing after treatment as predicted. This result may reflect the stressful circumstances of his first procreative pair bonding and the monozygotic attachment rupture with T1 during the posttreatment measurement phase.

Inferential Analysis

The research aims and hypotheses were considered inferentially in response to the statistical results.

Behavioral Experiments: Table 1 and Figure 3

In two behavioral experiments, heart rate measures of somatic congruence (Table 1) and self-report of somatic congruence (Figure 3), the twins demonstrated their reported autonomic somatic congruence and monozygotic attunement transposed through the interpersonal neurobiology (Siegel, 2012) of their dyadic trauma. Both twins were experiencing vasovagal syncope in response to T1's medical trauma triggers. Although unsupported by statistical evidence these results indicate that trauma may be contagious in attachment dyads. Historically, it was previously established through familial report, observed behavior between the twins and their self-report that the twins had established a suboptimal sibling attachment style in that T1 had a primary attachment to T2.

Compromised SPA has significant negative impact on all future attachment dyads (Riordan et al., 2019) and can impede social development in that suboptimal attachment compromises SPA where the immature caregiver is overwhelmed in the care role and the care receiver experiences inadequate care (Riordan et al., 2019), particularly when there is inadequate somatic-attachment-soothing and suboptimal attuned regulation (Riordan et al., 2017). Suboptimal monozygotic sibling attachment style is antagonistic to third party procreative attachments.

T1 Pre- and Posttreatment Analysis: Table 2

Pre-treatment, T1 met criteria for childhood PTSD as evidenced by his episodes of vasovagal syncope, avoidance of medical procedures, sleep disturbances, elevated heart rate, intolerance to incongruence manifest in anger and oppositional behavior. These outcomes were validated by T1's pretreatment measures compared to posttreatment results. Post-treatment, T1 did not meet criteria for childhood PTSD in that his trauma symptomology had abated. After T1's first treatment with SE, vasovagal episodes ceased for both twins and avoidance of medical trauma triggers abated altogether. Treatment with AF-SE followed shortly after.

The data support the assertion that SE and AF-SE are effective treatments for PTSD and dyadic trauma. Pre-treatment the twins experienced dyadic trauma and post-treatment they experienced dyadic completion, supporting the hypothesis that trauma is contagious and compromises SPA. By implication monozygotic attachment and its component state monozygotic attunement between the twins supports the construct of autonomic somatic congruence pre- and post-treatment. Posttreatment results confirmed statistical Hypothesis 1, in that T1 had higher HRV scores, higher HRV index variables scores, lower heart rate, and fewer nocturnal disturbances than pre-treatment. Time asleep reduced non-significantly and can be attributed to lifestyle changes. Only two measures, HF power and heart rate, fell within the statistically significant range.

T2 Pre- and Posttreatment Analysis: Table 3

T2 did not experience trauma, yet before T1's treatment he experienced the symptomology of trauma, confirming the constructs of dyadic trauma and trauma as a contagion. Statistical evidence supports the construct of dyadic trauma because T2 also recovered after treatment in congruence with T1's recovery, which confirms the twins' somatic congruence as evidenced by T2's dyadic completion after T1's treatment with AF-SE. Functionally, both twins recovered from vasovagal syncope and avoidance of medical trauma triggers after T1's first treatment with SE. However, although measures and statistical analysis of autonomic variables follow the general trend of reduced symptomology after treatment, statistical evidence for dyadic completion is mixed due to T2's slight posttreatment reduction in HRV which was explained in table 6 with his HRV coefficient of variation score that is most likely a result of T2's perturbations due to his first procreative pair bonding negative experience**.**

These data support research aim (b.), demonstrating that trauma is manifest in dyadic trauma, is contagious, and compromises secure phylogenetic attachment, and by inference, that dyadic trauma may compromise SPA. Compromised SPA contributes to psychopathology, loneliness, social isolation, and loss of social cohesion.

T2's posttreatment outcomes for all three HRV index variable and hours slept support statistical Hypothesis 3 in that T2 displayed congruent autonomic changes consistent with T1's posttreatment changes and also support the constructs of monozygotic attunement, monozygotic attachment, dyadic

trauma, and dyadic completion. However, T2's HRV and HR scores went against the trend of data. This discrepancy may be explained by the elevated HRV CV and HF/LF ratio scores shown in Table 6 comprising paired samples *t* tests for each twin comparing pre- and posttreatment scores.

Comparison of Individual Pre-and Posttreatment Results: Table 2 and Table 3

T1 was first treated with SE for PTSD and then with AF-SE to address the twins' dyadic trauma and separation issues. After T1's treatment with SE, episodes of vasovagal syncope and avoidance of medical procedures ceased immediately for both twins. The corresponding research data, measured autonomically, showed consistent improvement of PTSD and trauma symptomology for both twins (Table 2 and Table 3), supporting research aim (a.) to determine that SE and AF-SE are effective treatments for PTSD, trauma symptomology, and dyadic trauma. After T1's treatment with AF-SE, the twins were more readily able to separate and their monozygotic attachment style changed to a more traditional sibling attachment style.

Significant Differences Between T1 and T2 Pre-Treatment: Table 4

Counterintuitively, T1's pretreatment measures of autonomic trauma symptoms were less intrusive than T2's (except for nocturnal disturbances), supporting the twins' assertion of somatic congruence and the construct of dyadic trauma, that being, T2 experienced T1's trauma symptoms. The twins' self-report is supported by the behavioral experiments (see Table 1 and Figure 3). T2's pretreatment results confirm the construct of dyadic trauma in that his HRV and HRV index variables were all consistently lower than T1's. Similarly, T2's heart rate, an indicator of elevated sympathetic arousal, was higher than T1's. Statistically significant pretreatment differences in HRV and LF/HF ratio strongly support the construct of dyadic trauma and support the twins' claim of somatic congruence and monozygotic attunement at presentation and after treatment.

Differences Between T1 and T2 Posttreatment: Table 5

Comparative posttreatment data between T1 and T2 (Table 5) display somatic congruence between the twins consistent with their pretreatment data (Table 4), confirming constructs of trauma as a contagion, monozygotic attunement, dyadic trauma, and dyadic completion.

Reductions in T2's trauma symptoms were synchronous with T1's reduction in autonomic measures of trauma symptomology. Statistical analyses for the twins individually, pre- and post-treatment, demonstrate somatic congruence for each twin and comparatively before and after treatment. These data trend to support the constructs of monozygotic attachment and monozygotic attunement and T2's somatic congruence with T1 pre- and post-treatment. Posttreatment somatic congruence between T1 and T2 also supports the concept of trauma as a contagion because T1's somatic completion was transposed via the twins' monozygotic attunement to T2, resulting in his dyadic completion. These results also support the construct that secure phylogenetic attachment is the antithesis of trauma in that T2 experienced positive changes after T1's treatment despite having received no treatment.

Paired Samples t Test Within Each Twin Comparing Pre- and Post-treatment Scores: Table 6

Table 6 shows paired samples *t* tests comparing pre- and posttreatment results for a broader range of HRV and PTSD index variables. HRV is influenced by several index variables other than those identified as having posttreatment significance. A particularly skewed outcome can influence HRV results. T2's elevated HRV CV and HF/LF ratio scores may account of the slight drop in his posttreatment HRV score.

The inconsistent outcome of T2's reduced HRV scores post-treatment (Table 3) may be explained by the fact that HRV is calculated through a variety of time–domain, frequency–domain, and nonlinear metrics, only three of which, according to Negpal and colleagues (2013), have scientific utility to determine treatment outcomes. The other HRV index variables not shown in Table 3 influenced T2's overall HRV posttreatment score slightly without altering the general trend of the data (Table 6). The HRV CV and LF/HF ratio outcomes for T2 are particularly significant and complicit in T2's reduced HRV score post-treatment, which trends against the flow of the data. HRV CV measures perturbations such as stress that would decrease posttreatment HRV scores due to stressful lifestyle events. Similarly, T2's lower HF/LF ratio score (Table 6) may have implications for his posttreatment cardiac sympathovagal balance in that increases in HF/LF ratio may reflect a shift to sympathetic dominance (stress, vigilance) and decreases in LF/HF ratio may correspond to parasympathetic dominance in health and disease (Malliani et al., 1991), although this theory has been recently challenged by Billman (2013). At the time of measuring posttreatment outcomes, T2 was engaged in his first serious, procreative romantic bond. This preadolescent relationship was tumultuous, with many highs and lows (shifts in polyvagal and sympathetic/parasympathetic arousal states due to an unpredictable, external, perturbing source). T2 endured systemic family opposition to the relationship, particularly from T1 who was overtly opposed to it. The twins' monozygotic attachment rupture, in the context of T2's procreative pair bonding, sufficiently highlights the issue of compromised pair bonding for monozygotic twins, a key developmental and life cycle issue.

Discussion

The research aims of the study were to determine whether SE and AF-SE were effective treatments for PTSD, trauma symptomology, and dyadic trauma. The observable behavioral change in recovery of this twin pair after treatment, supported by statistical evidence of objective autonomic change, offers evidence of the efficacy of SE and AF-SE.

The research also aimed to elucidate through inferential analysis of autonomic measures the veracity of the constructs of dyadic trauma, dyadic completion, monozygotic attachment, monozygotic attunement, and SPA as the antithesis of trauma, and their impact on community psychopathology, loneliness, social isolation, and loss of social cohesion.

The data support the assertion that SE and AF-SE are effective treatments for PTSD, trauma symptomology, and dyadic trauma. Pre-treatment, the twins experienced PTSD and dyadic trauma; post-treatment they experienced resolution of PTSD symptoms with dyadic completion.

Statistical analyses for the twins individually and comparatively pre- and posttreatment demonstrate somatic congruence between the twins. These data support the constructs of monozygotic attachment and monozygotic attunement. Pre- and posttreatment somatic congruence between T1 and T2 supports the concept of trauma as a contagion. These results also support the construct that secure phylogenetic attachment is the antithesis of trauma in that T2 experienced positive changes after T1's treatment despite having received no treatment.

Statistical evidence of somatic congruence in trauma symptomology transposed neurogenically in dyads (dyadic trauma) indicates that treating trauma alone is insufficient to avoid future psychopathological and social consequences for the person suffering trauma, their attachment relationships, and the community in general. It is plausible that those treated for PTSD may recover, but dyadic trauma might remain in their attachment relationships to generate ongoing perturbations in the dyad, with itinerant loss of social cohesion. More emphasis needs to be made on attachment dynamics in trauma treatment. The successful utilization of AF-SE techniques to ensure that trauma is not contagious in relationships and does not compromise secure phylogenetic attachment in all future dyads has been demonstrated here. Had the twins not recovered from their trauma and suboptimal sibling attachment style they may have continued to avoid medical procedures, compromising their health, and remained socially and emotionally dependent on each other. Developmentally, SPA requires monozygotic twins to seek secure phylogenetic attachments outside family bonds in the procreative stage of the life cycle.

In neuroscience, trauma has been largely conceptualized as existing in the neurophysiology of the individual experiencing the trauma (Porges, 2011; Scaer, 2014). Attachment theorists (Schore, 2019a; Siegel, 2012) and traumatologists (Riordan et al., 2019; van der Kolk, 2014) have emphasized the importance of attachment relationships and the empathic witness (Levine, 2010) as healing mechanisms. The construct that trauma exists only in the neurophysiology of the individual can no longer be held true and invites a paradigm shift for traumatologists to address trauma from the perspective of modern attachment theory (Schore, 2019). Trauma treatment should therefore include attachment dyads in treatment and incorporate interpersonal neurobiological processes and the face–heart connection in attachment dyads as essential components of trauma recovery.

Demonstration of pre- and posttreatment somatic congruence between T1 and T2 supports the construct that trauma is contagious in dyads, requiring changes in the nomenclature of modern attachment theory to include secure phylogenetic attachment as a more nuanced and appropriate description of secure attachment. The importance of individual neurophysiology and interpersonal neurophysiology in the dynamics of recovery from trauma and dyadic trauma supports a paradigm shift to include the language of attachment neurogenetics as important components of trauma treatment. Trauma research in relation to attachment requires a more nuanced nomenclature (Riordan et al., 2019) to understand, categorize, and codify trauma and its complex attachment-based components.

These results indicate that recovery from PTSD can be measured quantifiably in clinical settings using objective and portable measures of HRV and secure computing software to record autonomic measures of PTSD symptomology. Treatment that targets objective autonomic variables via the neurophysiology of trauma symptomology may better inform researchers and clinicians of day-to-day changes in their clients' neurophysiology and treatment outcomes. As demonstrated by this study, portable devices that measure autonomic function can now inform clinicians of pre-and posttreatment outcomes. Changes over time in treatment outcomes can be adjusted to address ongoing shifts in somatic, neurological, and behavioral components of recovery. Measures such as self-report, clinical observation, and psychometrics all have an element of subjectivity that do not rely on objective data. Objective measures of change over time can an offer immediate feedback to clinicians, their clients, and the therapeutic community about the direction and veracity of treatment.

Secure phylogenetic attachment is an essential component of individual and community wellness (Riordan et al., 2019) and loss of SPA is a precursor to individual and community psychopathology that may have a lifelong impact on relationships. There are many issues that might contribute to psychopathology, loneliness, social isolation, and loss of social cohesion. However, given the impact of trauma on neurological events in the brain (Porges, 2011) and the behavior changes associated with fear vigilance and avoidance ideation of those with PTSD (Schore, 2019a), it is reasonable to consider that trauma generates dyadic trauma and compromises secure phylogenetic attachment and contributes to widespread psychopathology, loneliness, social isolation, and loss of social cohesion in our communities.

Further investigation into the constructs of somatic congruence, dyadic trauma, dyadic completion, monozygotic

attunement, monozygotic attachment, and trauma as a contagion in attachment dyads, based on measures of autonomic variables, has profound implications for assessment and treatment of PTSD and the issues of community psychopathology, loneliness, social isolation, and loss of social cohesion.

Research Outcomes and Follow-Up Observations

In a 36-month follow-up, vasovagal episodes did not reoccur for either twin following T1's first treatment with somatic experiencing. Emotional overwhelm leading to avoidance of minor medical procedures also abated so that both twins were able to tolerate injections and minor medical procedures where previously they would faint or display avoidance and or behavioral resistance (e.g., running away).

T1 had historically been the more oppositional, aggressive, and verbally confronting twin, and T2 had been the calmer and more passive twin, always allowing T1 to speak for them in family and peer group disputes.

Tolerance to incongruence (manifest in anger) was assessed with researcher observation, family and school records, and self-report, pre- and post-treatment. All parties, including the twins, agree that after treatment there was a shift in pretreatment roles between the twin pair in that T2 became the more outspoken and forthright. Interestingly, T2's posttreatment intolerance to incongruence increased in direct response to T1's reduction of intolerance to incongruence after treatment. (This phenomenon emerged before T2 commenced his first perturbing, procreative, pair-bonding relationship.) T2's posttreatment higher heart rate and lower HRV (Table 3) tend to support this outcome, which may be a remnant of dyadic trauma, the suboptimal monozygotic attachment style between the twins, or T2's first perturbing romantic involvement.

Fiercely loyal to each other, T1 had always been the more anxious twin, worried about consequences for T2 and constantly inviting T2 into more responsible behavior around family commitments, chores, homework, parental restrictions on access to technology, online communications, and other lifestyle issues that impacted the twins. After treatment, this trait ceased altogether, indicating T1's shift into a more typical sibling attachment style characterized now as a secure phylogenetic sibling attachment style rather than his previous suboptimal monozygotic attachment style where he was overly concerned for and dependent on T2's wellbeing as a source of his own sense of security. In effect, the twins had become more independent of their monozygotic twinship and were able to tolerate separation and differences more readily.

Monozygotic twins share the same DNA and in normal circumstances share life experiences that are parallel and similar. In this case the only experiential difference between the twins was T1's sequence of traumatic events. PTSD can influence physical development in that continual sympathetic arousal can influence appetite and energy expended through constant hypervigilance and elevated sympathetic arousal with fight/flight responses, consequently the twins' physical appearance differed in body form, face, and weight, T1 being noticeably leaner at presentation than T2. After treatment both twins were more regulated, and their physiology aligned to better represent their monozygotic twinship in their emerging secure phylogenetic attachment style.[1]

Limitations of the Study

The efficacy of smartphone devices is not fully established and despite the internal validity and objectivity of pre-and posttreatment comparisons of autonomic variables, the accuracy and scientific utility of smartphone devices is yet to be firmly established. Future studies might compare the use of smartphone devices in community clinical settings to those of hospital grade in gold-standard experimental research conditions to establish the veracity and extent of the statistical validity of data obtained via smartphone devices as portable measures of autonomic variables.

The data were collected by the volunteer subjects, and there may be individual differences in control measures across the two phases of pre-treatment and posttreatment measuring. Stressful life events such as those seen in the posttreatment data collection phase for T2 can influence autonomic measures of recovery. More rigorous research design and controlled environments may account for this eventuality. Community-based experimental design where research parameters are less controlled may need to address unanticipated life events in future HRV trauma research.

A single-subject case study, the twin pair, does not offer sample–size statistical validation. Replicating this study with several twin–pair subjects may offer deeper insights into the constructs.

Further monozygotic twin study research is required into the nature of monozygotic attachment and monozygotic attunement given that shared DNA is a control variable that isolates traumatic experience as the target variable for treatment interventions in post-trauma presentations. In monozygotic twins, trauma may be more intrusive due to the highly attuned nature of monozygotic attachment. Therefore, inferences made from monozygotic twin studies might not be fully generalizable to a wider population.

Early childhood trauma is life-altering and generates substantial change in sibling attachment dynamics (Riordan et al., 2017, 2019). Further research on childhood trauma as a contagion in sibling attachment dyads throughout the life cycle is required.

[1] Interestingly, pre-treatment the twins could not open each other's phones with face recognition, post-treatment they could.

Summary of Research Findings

In consideration of the outcomes of this study the researcher offers the following for consideration by the community of traumatologists:

- SE and AF-SE are effective treatments for PTSD, trauma symptomology, and dyadic trauma.
- Secure phylogenetic attachment is the antithesis of trauma.
- Trauma, manifest in dyadic trauma, is contagious and compromises secure phylogenetic attachment.
- AF-SE is an effective treatment for dyadic trauma by resolving traumatic perturbations in attachment dyads and by reinstating secure phylogenetic attachment.
- Trauma negatively impacts secure attachment relationships and is transposed neurogenically to generate dyadic trauma.
- Monozygotic twins share a unique somatic attunement, identified here as monozygotic attunement, which is poorly understood by attachment neuroscientists and requires closer examination.
- Trauma is complicit in dysfunctional attachment styles and may contribute to widespread psychopathology, loneliness, social isolation, and loss of social cohesion.

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