


Dyadic trauma and attachment: A monozygotic twin study assessing the efficacy of Somatic Experiencing®

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Orientation: 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 antagonistic to secure phylogenetic attachment (SPA).

Research purpose: Attachment perturbations in trauma may be complicit in psychopathology. Somatic Experiencing (SE) is an effective treatment for post-traumatic stress disorder (PTSD), and attachment focused somatic experiencing (AF-SE) resolves dyadic trauma.

Motivation for the study: Monozygotic twins may share a unique form of sibling attachment, described here as monozygotic attachment, characterised by elevated somatic congruence resulting in suboptimal attachment. The study was conducted to determine whether dyadic trauma is contagious and compromises SPA and whether SE and AF-SE are effective treatments to restore SPA.

Research approach/design and method: A quantitative experimental approach was used to examine the nature of trauma in monozygotic attachment and the significance of trauma in relation to psychopathology. 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.

Main findings: Results conclude that trauma is contagious in attachment dyads and contributes to psychopathology. Somatic Experiencing is an effective treatment for trauma. The AF-SE resolves dyadic trauma restoring SPA as the antithesis of trauma.

Implications for practice: Traumatology and attachment theory may be linked theoretically to resolve trauma.

Contribution/value-add: Secure phylogenetic attachment offers a new category of attachment theory that defines trauma in dyads and its relationship to psychopathology.

Keywords: dyadic trauma; dyadic completion; monozygotic; secure phylogenetic attachment; somatic experiencing.

Introduction

Orientation

Trauma and dyadic trauma in all manifestations are socially destructive (Riordan, Blakeslee, & Levine, 2017, 2019) and may have a role in widespread community psychopathology, loneliness, social isolation, and loss of social cohesion. Twenty-four to twenty-seven percent of Australian households are estimated to be single-person homes (Australian Bureau of Statistics, 2022). Thirty-three percent of Australians report loneliness or social isolation (Australian Institute of Health and Welfare, 2021). One in four people are lonely (Abbott, Lim, Eres, Long, & Matthews, 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 healthcare professionals (Abbott et al., 2018; Lim, 2018).

Trauma compromises and hijacks the social networks of the prefrontal cortex (Schoore, 2019). This in turn generates a flight survival response (Porges, 2011) that promotes avoidance, a key symptom of post-traumatic stress disorder (PTSD) (American Psychiatric Association, 2013). People with PTSD have a high tendency to experience loss of social cohesion and social isolation, because

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sympathetic arousal devolves in a cascade of trauma responses that are later visible in relationships as perturbations in the dyad, generating dyadic trauma (Riordan et al., 2017, 2019). Post-traumatic stress disorder impairs a person's ability to form and sustain relationships – for example, causing difficulty in maintaining social and attachment relationships (Riordan et al., 2019), resolving attachment ruptures (Riordan et al., 2017) and independently completing autonomic survival imperatives to sustain SPA (Riordan et al., 2019). Trauma is experienced interpersonally as perturbations in attachment dyads that can evolve in a complex web of trauma symptomology that generates widespread psychopathology interpersonally and across communities.

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

Somatic Experiencing is a clinically effective treatment for the neurobiological resolution of trauma within the individual nervous system (Brom et al., 2017; Leitch, 2007; Leitch, Vamslyke, & Allen, 2007; Levine, 2010; Parker, Doctor, & Selvam, 2008). A core tenet of SE is 'the completion of thwarted, biologically based, self-protective and defensive responses, and the discharge and regulation of excess autonomic arousal' (Payne, Levine, & Crane-Godreau, 2015, p. 1). Somatic Experiencing offers a comprehensive understanding of trauma recovery through an active process of therapeutic engagement where the therapist is an *empathic witness* (Levine, 2010) of the client's interoceptive, kinaesthetic 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 reorganise in homeostasis towards whole brain neural synchrony to a state of relaxed readiness.

The field of traumatology offers only sparse quantifiable, physiological understanding of how trauma impacts relationships and how it is transposed neurogenically in secure attachment dynamics. Physiological measures of trauma responses in pre- and post-treatment studies offer objective platforms to understand the nature of trauma in nervous systems and how it impacts attachment. This study of one monozygotic twin pair attempts to understand the effect of dyadic trauma on suboptimal attachment styles and specifically, how *trauma as a contagion* is transposed in dyadic trauma when one twin is traumatised and the other is not.

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

In their meta-analysis of HRV index variables as indicators of PTSD, Negpal et al. (2013) concluded that three HRV index variables – high-frequency (HF) power, root mean square of successive R-R interval difference (RMSSD), and low-frequency (LF) to HF ratio – are key indicators of PTSD symptomology that can provide quantifiable evidence of post-treatment recovery.

Together with the HRV index variables described here, heart rate, sleep duration, and sleep disturbances were also identified as objective measures of trauma responses used in this study (Schneider & Schwerdtfeger, 2020).

Smartphone-connected devices that measure autonomic function are a portable and easily accessible option for measuring autonomic variables and can be readily adapted to inform treatment outcomes in clinical settings.

Van Boxtel et al. (2018) advocated for the use of HRV measures in studies of individuals at risk of developing symptoms of PTSD and observed 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 Van Boxtel et al. (2018), Porges (2011) and Negpal et al. (2013), HRV may be a measure of sympathetic and parasympathetic homeostatic states in PTSD. Heart rate variability measured by smartphone devices can offer convenient measures of change over time and objective measures in pre- and post-treatment research.

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 – has two roles: firstly, to foster physiological homeostasis to promote growth and restoration (rest and digest) and secondly, during environmental challenges, to act as a brake (the vagal brake) to rapidly regulate cardiac and metabolic output (flight, fight and freeze). The phylogenetic purpose of the vagal brake 'provides a neurophysiological mechanism that may promote the development of appropriate social behaviour' (Porges, 2011, p. 106).

By this definition, HRV is a measure of the action of the vagus nerve 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 behavioural cascade of escalating fear avoidance or aggression, neither of which are conducive to SPA nor social cohesion. However, consistent low heart rate in PTSD may indicate a sustained state of bradycardia or dorsal vagal collapse manifested as dissociative states of numbness, somatic overwhelm and

shutdown, including vasovagal syncope, which are similarly not conducive to SPA or social cohesion.

A comparison of monozygotic twins who share the same DNA and are more concordant than other siblings offer a unique research opportunity to evaluate the clinical validity of trauma treatment based on measures of autonomic variables of HRV, HRV index variables, heart rate, sleep duration, and sleep disturbances.

Research purpose and objectives

The purpose of this study is to determine whether dyadic trauma is contagious and compromises SPA and whether SE and AF-SE are effective treatments for PTSD, trauma symptomology, and dyadic trauma to restore SPA.

The primary aims of this research are to determine:

1. the efficacy of SE and AF-SE as effective treatments for PTSD, trauma symptomology, and dyadic trauma
2. whether trauma is manifested in dyadic trauma, is contagious and compromises SPA.

The secondary aims will examine:

1. the nature of monozygotic attachment and monozygotic attunement by comparing autonomic physiological variables between T1 and T2 pre- and post-treatment
2. the construct of SPA as the antithesis of trauma.

Research methods and design

Research approach

This study followed a quantitative, experiential approach.

Researcher's role

The researcher's role was to facilitate the therapeutic process, to ensure safety and to gather, analyse and report on data.

Research participants and sampling method

Monozygotic male twins (aged 13 years; T1 and T2) presented for psychological treatment with anger, fear vigilance, and avoidance of medical procedures and routine health examinations. Episodes of vasovagal syncope (fainting) during minor medical procedures were common for both twins.

T1 presented with childhood PTSD. T2 experienced no actual trauma but experienced PTSD symptoms in monozygotic attunement with T1. T2 was diagnosed as experiencing dyadic trauma.

Monozygotic twins offer a unique research opportunity because the dependent variables can be easily isolated. Because one twin was traumatised and the other was not, an opportunity to measure dyadic trauma and dyadic completion pre- and post-treatment emerged. The twins readily agreed to the research proposal.

Intervention

T1 was treated with SE and AF-SE. T2 did not receive treatment.

T1 was treated for medical trauma at the age of six years, having the plaster on a broken arm removed, with a single session of SE. The SE session targeted T1's belief ('the technician was going to cut my 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, discharge and reorganisation of flight, fight or freeze responses in the sympathetic nervous system and polyvagal network), T1 pushed (extended from chest to full arm length) with his unbroken arm against a cushion as he interocepted his fear of having his arm cut off. The SE therapist provided resistance to achieve realistic full arm extension to 'push away the danger', somatically enacting T1's survival response for completion, a neurogenetic and phylogenetic survival imperative. This *push work* simulated somatic completion of flight or fight responses for survival. Somatic 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, manifested 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 and somatic shift towards homeostasis.

T1 was then treated with AF-SE to redress the twins' dyadic trauma, a suboptimal sibling attachment style, and the specific attachment ruptures regarding separateness in their monozygotic attachment dynamics (that being their separate attuned attachment to girlfriends). Attachment-focused somatic experiencing consisted of T1 cognitively recalling and intercepting his fear of separation from T2, thereby losing his primary soothing support person (his fears of separation in class, intrusion from others and less monozygotic attunement with T2 associated with romantic bonds).

Data collection methods

Pre- and post-treatment PTSD symptoms were recorded with smartphone devices employed to measure HRV and the HRV index variables of HF power, RMSSD, and HF/LH ratio. Sleep disturbances, sleep duration, and heart rate were also recorded. Pre- and post-treatment changes were compared, and conclusions were drawn on the constructs of dyadic trauma, dyadic completion, monozygotic attachment, monozygotic attunement, and SPA as the antithesis of trauma.

Smartphone devices can record nocturnal behaviour 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. Data were collected from Fitbits and Elite HRV monitoring devices (a chest strap monitor, with measurements taken once daily, after waking). Fitbits were worn 24 h per day to record heart rate and at night to record sleep disturbances.

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

Data analysis, method and process

There were 41 observations per variable for T1 prior to receiving treatment. For T2, there were 47 observations per variable prior to receiving treatment. Values that were three or more standard deviations above or below the mean were removed.

Following the 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.

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

Nocturnal waking and sleep duration were delineated between deep sleep, wakes, and other non-deep sleep wakes. Data were compared pre- and post-treatment for each twin and comparatively between the twins pre- and post-treatment. Deep sleep disturbances indicated frequency of the PTSD symptoms of nightmares, night terrors, night sweats, or startled waking.

T1 and T2's autonomic indicators of PTSD and dyadic trauma were compared pretreatment 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 post-treatment results were compared individually for T1 and T2. Similarly, the impact of AF-SE on dyadic trauma was considered regarding T2's pre- and post-treatment results. Inferential analysis considered the twin pair's somatic congruence, monozygotic attunement, dyadic trauma, dyadic completion, and implications for their attachment style. Inferences were made on the data for global issues of psychopathology, loneliness, social isolation, and loss of social cohesion in communities.

Firstly, 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 were conducted. Pretreatment scores were compared with post-treatment 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).

Secondly, outcomes between T1 and T2 pretreatment 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). In addition, the assumption of independent observations was accounted for. This means that the participants in one group were not also participants in the other group.

Post-treatment HRV, HRV index variable scores and non-HRV PTSD-related indicators (heart rate, sleep duration and sleep disturbances) were compared between T1 and T2. All data were recorded in statistical tables.

Ethical considerations

This study followed the Australian Psychological Society's ethical guidelines for human subject research. Ethical clearance was obtained from the Ergos Institute of Somatic Education, Peter A. Levine, PhD, and Dr Abi Blakeslee, PhD. Informed consent was obtained from both parents and both twins, with an option to decline at any time for all or any of the components 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.

Results

T1 pre- and post-treatment

Pre- and post-treatment scores were compared for T1 (Table 1). It was hypothesised 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.

For HRV scores, there was only one significant difference in T1's pre- and post-test HRV indicator scores. T1 had an increase in HF power from pretest to post-test (i.e. higher HF power at post-test compared with pretest), $t(33) = -2.151$, $p = 0.039$. Regarding the non-HRV PTSD symptomology, there were significant differences from pretreatment to post-treatment for T1. There was a significant reduction in HR from pre- to post-treatment, $t(33) = 3.220$, $p = 0.003$. In addition, there was a significant reduction in wakes from deep sleep, $t(33) = 2.282$, $p = 0.030$. Pre- and post-treatment scores support Research Aim 1. The only statistic that did not conform with Research Aim 1 was an insignificant reduction in sleep hours post treatment.

T2 pre- and post-treatment

Pre- and post-treatment scores were compared for T2 (Table 2). It was hypothesised 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 post-treatment score reduced non-significantly. All other post-treatment scores improved consistently with Research Aims 1 and 2, because T2's post-treatment results displayed congruence with T1's post-treatment scores. There were two significant differences from pretreatment to post-treatment. The LF-HF ratio significantly increased, $t(32) = -4.347$, $p < 0.001$ and HR increased = $t = -2.012$, $p = 0.053$ Although inconsistent, the data tends to support Research Aim 1. T2's reduced HRV score demonstrates that HRV scores can be influenced by all 12 HRV index variables described in Table 5 in conflict with the three primary HRV index variables known to indicate improvement after treatment. Despite having a lower post-treatment HRV score, T2 still showed improvement on post-treatment HRV index variables and nocturnal disturbances, indicating an improvement in trauma symptomology. Behaviourally, post treatment, T2 displayed reductions in symptomology (vasovagal syncope and avoidance of trauma triggers), indicating observable behavioural validation of AF-SE a treatment for dyadic trauma.

TABLE 1: T1 pre- and post-test scores for all heart rate variability and non-heart rate variability indicators.

	T1		<i>T</i>	<i>P</i>
	Pretreatment	Post-treatment		
HRV	61.46 (4.51)	63.56 (3.04)	-1.345	0.188
RMSSD	56.85 (18.10)	63.47 (13.05)	-0.927	0.361
HF power	926.75 (486.91)	1298.82 (605.31)	-2.151	0.039
LF-HF ratio	1.73 (0.90)	2.02 (1.11)	-1.619	0.115
HR	77.10 (4.98)	72.23 (6.61)	3.220	0.003
Sleep hours	6.86 (0.76)	6.66 (0.79)	1.156	0.257
Wakes per night	2.07 (1.44)	1.70 (0.99)	1.302	0.203
Wakes from deep sleep	0.80 (0.85)	0.37 (0.56)	2.282	0.030

HRV, heart rate variability; RMSSD, root mean square of successive R-R interval difference; LF, low frequency; HF, high frequency; HR, heart rate.

Significant differences between pre- and post-treatment shown in bold. Significance levels were set as ($p < 0.001$).

TABLE 2: T2 pre- and post-test scores for all heart rate variability and non-heart rate variability indicators.

	T2		<i>t</i>	<i>p</i>
	Pretreatment	Post-treatment		
HRV	59.23 (5.24)	58.70 (6.69)	1.287	0.207
RMSSD	49.81 (17.08)	50.16 (28.01)	1.896	0.067
HF power	896.67 (629.48)	919.02 (659.13)	0.728	0.472
LF-HF ratio	1.25 (0.79)	2.62 (1.75)	-4.347	< 0.001
HR	82.25 (7.34)	84.93 (11.56)	-2.012	0.053
Sleep hours	6.81 (0.90)	6.69 (0.95)	0.615	0.543
Wakes per night	1.17 (0.87)	0.87 (0.57)	1.361	0.184
Wakes from deep sleep	0.47 (0.63)	0.17 (0.53)	1.795	0.083

HRV, heart rate variability; RMSSD, root mean square of successive R-R interval difference; LF, low frequency; HF, high frequency; HR, heart rate.

Significant differences between pre- and post-treatment shown in bold. Significance levels were set as ($p < 0.001$).

Comparisons between T1 and T2: Pretreatment

Pretreatment outcomes were compared between T1 and T2 (Table 3). 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 construct of trauma as a contagion in that before treatment, T2 experienced the somatic symptoms of PTSD (dyadic trauma) in his monozygotic attunement with T1.

Pretreatment, T1 and T2 had significantly different scores on HRV and LF-HF power. Heart rate variability was significantly higher for T1 than T2, $t(86) = 2.124$, $p = 0.037$. In addition, T1 had significantly higher LF-HF ratio than T2 pretreatment, $t(86) = 2.665$, $p = 0.009$. There were no significant differences in pretreatment RMSSD or HF power between T1 and T2. T2's HR was considerably higher, which was consistent with T2's reported pretreatment sympathetic arousal states, 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 68.68.¹ Pretreatment scores for both twins were significantly lower than the best-known healthy average HRV scores, indicating that both twins were experiencing trauma symptomology. These data support the research

1. See <https://elitehrv.com/>.

TABLE 3: Significant differences between T1 and T2 pretreatment.

	Pretreatment		<i>t</i>	<i>p</i>
	T1	T2		
HRV	61.46 (4.51)	59.23 (5.24)	2.124	0.037
RMSSD	56.85 (18.10)	49.81 (17.08)	1.877	0.064
HF power	926.75 (486.91)	896.67 (629.48)	0.248	0.805
LF-HF ratio	1.73 (0.90)	1.25 (0.79)	2.665	0.009
HR	77.10 (4.98)	82.25 (7.34)	-3.795	< 0.001
Sleep hours	6.86 (0.76)	6.81 (0.90)	0.223	0.825
Wakes per night	2.07 (1.44)	1.17 (0.87)	2.931	0.005
Wakes from deep sleep	0.80 (0.85)	0.47 (0.63)	1.731	0.089

HRV, heart rate variability; RMSSD, root mean square of successive R-R interval difference; LF, low frequency; HF, high frequency; HR, heart rate.

Significant differences between pre- and post-treatment shown in bold. Significance levels were set as ($p < 0.001$).

TABLE 4: Differences between T1 and T2: Post-treatment.

HRV	Post-treatment		<i>t</i>	<i>p</i>
	T1	T2		
HRV	63.56 (3.04)	58.70 (6.69)	3.848	< 0.001
RMSSD	63.47 (13.05)	50.16 (28.01)	4.957	< 0.001
HF power	1298.82 (605.31)	919.02 (659.13)	2.458	0.017
LF-HF ratio	2.02 (1.11)	2.62 (1.75)	-1.708	0.092
HR	72.23 (6.61)	84.93 (11.56)	-5.539	< 0.001
Sleep hours	6.66 (0.79)	6.69 (0.95)	-0.111	0.912
Wakes per night	1.70 (0.99)	0.87 (0.57)	4.00	< 0.001
Wakes from deep sleep	0.37 (0.56)	0.17 (0.53)	1.425	0.159

HRV, heart rate variability; RMSSD, root mean square of successive R-R interval difference; LF, low frequency; HF, high frequency; HR, heart rate.

Significant differences between pre- and post-treatment shown in bold. Significance levels were set as ($p < 0.001$).

aim that trauma is manifested in dyadic trauma and is contagious in attachment relationships, compromising SPA.

T1 and T2 analyses: Post treatment

Post-treatment outcomes were compared between T1 and T2 (Table 4). In this way, differences between T1 and T2 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 post-treatment scores.

Post-treatment, there were significant differences between T1 and T2 for HRV and two HRV indicator variables. T1 had significantly higher HRV scores than T2, $t(65) = 3.848$, $p < 0.001$. Additionally, T1 had higher RMSSD, $t(64) = 4.957$, $p < 0.001$, and HF power, $t(65) = 2.458$, $p = 0.017$, 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 Research Aim 1 and the construct of somatic congruence, monozygotic attunement and monozygotic attachment between the twins. These data also support Research Aim 2, confirming somatic congruence between T1 and T2 post treatment and that AF-SE is an effective treatment for dyadic trauma, supporting the construct of dyadic completion.

TABLE 5: Paired samples *t*-test within each twin: Pre- and post-treatment.

	T1		T2	
	<i>t</i>	<i>p</i>	<i>t</i>	<i>p</i>
HRV	-1.345	0.188	1.287	0.207
Morning readiness [†]	0.847	0.404	-0.363	0.719
HRV CV [‡]	-0.530	0.599	-4.914	< 0.001
HR	3.220	0.003	-2.012	0.053
ln RMSSD [§]	-1.327	0.194	1.329	0.193
RMSSD	-0.927	0.361	1.896	0.067
Nn50 [¶]	-2.075	0.046	1.598	0.120
Pnn50 ^{††}	-1.385	0.175	2.299	0.028*
SDNN	-3.558	0.001	-1.833	0.076
LF power ^{‡‡}	-3.135	0.004	-3.469	0.002
HF power	-2.151	0.039	0.728	0.472
LF-HF ratio	-1.619	0.115	-4.347	< 0.001
Total power	-3.111	0.004	-1.809	0.080
Sleep hours	1.156	0.257	0.615	0.543
Wakes per night	1.302	0.203	1.361	0.184
Wakes from deep sleep	2.282	0.030	1.795	0.083

HRV, heart rate variability; CV, coefficient of variation; RMSSD, root mean square of successive R-R interval difference; LF, low frequency; HF, high frequency; HR, heart rate.

Significant differences between pre- and post-treatment shown in bold. Significance levels were set as ($p < 0.001$).

[†], Morning readiness refers to baseline patterns of HRV measures.

[‡], HRV CV is the coefficient of variation in HRV measures over weeks rather than days.

[§], ln RMSSD refers to when a natural log is applied to the RMSSD in order to distribute the numbers in an easier to understand range.

[¶], Nn50 is the mean number of times per hour in which the change in successive normal sinus N-N intervals exceeds 50 ms.

^{††}, Pnn50 is the proportion of Nn50 divided by the total number of NN (R-R) intervals exceeding 50 ms.

^{‡‡}, LF power refers to heart rate frequency activity in the 0.04 Hz – 0.15 Hz range.

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 5 compares statistical differences between T1's pre- and post-treatment scores and T2's pre- and post-treatment scores. Table 5 also highlights significant differences between T1 and T2.

Discussion

T1 pre- and post-treatment analysis: Table 1

Pretreatment, T1 met criteria for childhood PTSD, as evidenced by his episodes of vasovagal syncope, avoidance of medical procedures, sleep disturbances, elevated heart rate, anger, and oppositional behaviour. These outcomes were validated by T1's pretreatment measures compared with post-treatment results. Post-treatment, T1 did not meet criteria for childhood PTSD because his trauma symptomology had abated. After T1's first treatment with SE and AF-SE, vasovagal episodes ceased for both twins and avoidance of medical trauma triggers abated altogether.

The data support the assertion that SE and AF-SE are effective treatments for PTSD and dyadic trauma. Before 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 support the construct of autonomic somatic congruence pre- and

post-treatment. T1's post-treatment results confirmed Research Aim 1 because T1 had higher HRV scores, higher HRV index variables scores, lower heart rate, and fewer nocturnal disturbances than pretreatment. Time asleep reduced nonsignificantly and can be attributed to lifestyle changes. Only two measures, HF power and heart rate, fell within the statistically significant range.

T2 pre- and post-treatment analysis: Table 2

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, despite having no 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 SE and AF-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 because of T2's slight post-treatment reduction in HRV, which was explained in Table 5 with his HRV coefficient of variation score because of a romantic pair-bonding perturbation episode.

These data support Research Aim 2, demonstrating that trauma manifested as dyadic trauma is contagious and compromises SPA. Inferentially, dyadic trauma appears to compromise SPA.

T2's post-treatment outcomes for all three HRV index variables and hours slept support Research Aim 2 and Secondary Research Aim 1 because T2 displayed congruent autonomic changes consistent with T1's post-treatment changes, supporting 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 5, comprising paired samples *t*-tests for each twin comparing pre- and post-treatment scores.

Comparison of individual pre- and post-treatment results: Tables 1 and 2

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, supporting Research Aim 1 to determine that SE and AF-SE are effective treatments for PTSD, trauma symptomology, and dyadic trauma.

After T1's treatment with AF-SE, somatic activation and discharge challenged T1's suboptimal monozygotic attachment style. Quiescent attunement was achieved with AF-SE, promoting homeostasis in T1's nervous system and dyadic completion for the twin pair. The twins were then

more readily able to separate, and their monozygotic attachment style changed to a more functional sibling attachment style.

Significant differences between T1 and T2 pretreatment: Table 3

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 more acutely than T1. T2's pretreatment results confirm the construct of dyadic trauma because 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 post-treatment: Table 4

Comparative post-treatment data between T1 and T2 confirms somatic congruence between the twins consistent with their pretreatment data, 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 tend to support the constructs of monozygotic attachment and monozygotic attunement and T2's somatic congruence with T1 pre- and post-treatment. Post-treatment results of somatic congruence between T1 and T2 also support 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 SPA is the antithesis of trauma, because 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 5

Table 5 shows paired samples *t*-tests comparing pre- and post-treatment results for a broader range of HRV and PTSD index variables. Heart rate variability is influenced by several index variables other than those identified as having post-treatment significance for PTSD. 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 post-treatment HRV score.

The inconsistent outcome of T2's reduced HRV scores post-treatment (Table 2) may be explained by the fact that HRV is calculated through a variety of time-domain, frequency-domain and non-linear metrics, only three of which, according to Negpal et al. (2013), have scientific utility to determine treatment outcomes. The other HRV index variables not shown in Table 2 influenced T2's overall HRV post-treatment score slightly without altering the general trend of the data (see Table 5). 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. Heart rate variability CV measures perturbations such as stress that would decrease post-treatment HRV scores because of stressful lifestyle events. Similarly, T2's lower HF-LF ratio score (Table 5) may have implications for his post-treatment cardiac sympathovagal balance, because 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, Pagani, Lombardi, & Cerutti, 1991), although this theory has been recently challenged by Billman (2013). At the time of measuring post-treatment outcomes, T2 was engaged in his first procreative romantic relationship. This preadolescent relationship was tumultuous, with many shifts in polyvagal and sympathetic and parasympathetic arousal states because of 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 for monozygotic twins.

Before 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. In addition, T1 had a lower average heart rate compared with T2 prior to treatment.

For T1, pre- and post-treatment, there were statistically significant differences in HRV and HRV index variables ($p < 0.05$). 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 pretreatment to post-treatment for T1 on morning readiness, HRV CV, in RMSSD, Pnn50, LF/HF ratio, sleep hours, and wakes per night.

For T2, HRV CV and LF-HF ratio increased after T1's treatment and Pnn50 decreased. 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 post-treatment measurement phase.

Implications for practice

In a five-year follow-up, vasovagal episodes did not recur for either twin following T1's first treatment with SE. 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 behavioural resistance. Both boys are in stable romantic relationships, tolerated by their twin.

These results offer 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, manifested in dyadic trauma, is contagious and compromises SPA.
- AF-SE is an effective treatment for dyadic trauma by resolving traumatic perturbations in attachment dyads and by reinstating SPA.
- Trauma negatively impacts secure attachment relationships and is transposed neurogenically, promoting 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.

Limitations of the study

The efficacy of smartphone devices is not fully established despite the internal validity and objectivity of pre- and post-treatment comparisons of autonomic variables. Future studies might compare the use of smartphone devices in community clinical settings to those of 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 pretreatment and post-treatment measuring. Stressful life events such as those seen in the post-treatment data collection phase for T2 can influence autonomic measures of recovery. More rigorous research design and controlled environments may address unanticipated life events in future HRV trauma research.

A single subject case study, this 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-traumatic presentations. In monozygotic twins, trauma may be more intrusive because

of the highly attuned nature of monozygotic attachment. Therefore, inferences made from monozygotic twin studies might not be fully generalisable to the 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.

Conclusion

Trauma generates dyadic trauma that compromises SPA and contributes to widespread psychopathology, loneliness, social isolation, and loss of social cohesion in our communities. SPA 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.

These results indicate that treatment outcomes can be measured quantifiably in clinical settings using portable measures of autonomic variables. Objective measures of change over time can offer immediate feedback to clinicians, their clients and the therapeutic community about the direction and veracity of treatment.

Further investigation into the constructs of somatic congruence, dyadic trauma, dyadic completion, monozygotic attunement, monozygotic attachment, and trauma as a contagion in attachment dyads has profound implications for treatment of PTSD and the promotion of SPA as a separate attachment category in attachment theory.

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Data availability

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