

“Connecting The Dots”: A Prevention and Intervention Methodology Targeting Psychiatric Comorbidities in Neurodiversity

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With alarming levels of psychiatric disorder and relational discord preventing, or compromising, functionality, arguably, humanity is ill-equipped to contend with global crisis while we are dysfunctional as inter-relating beings. Dysfunction that breeds dysregulation, a central nervous system dis-integrated and in disarray, thwarting capacity to reach potential, psychosocially, and intellectually and vocationally, renders huge societal cost. Hence, irrespective of psychiatric symptomatology, and of neurotype, looking forward to a more promising future mandates reducing dysregulation and enhancing capacity for connection.

Connecting the Dots (CTD) is an intervention and prevention methodology that utilises a relational framework, emphasising partnered and parental relationships, to foster enhanced self- and co-regulation capacity, thereby enabling improved psychosocial outcomes and minimising psychiatric disorder. This methodology combines well-known evidence-based theories and therapies toward dysregulation reduction.

CTD features Attachment (Bowlby, 1988; Ainsworth, Blehar, Waters, and Wall, 1978), Adult Attachment (Main and Solomon, 1990; Bartholomew, Henderson and Dutton in Clulow, 2001), Regulation (Schore, 2016) and Polyvagal (Porges, 2019) Theories, incorporating aspects of White and Epston’s Narrative Therapy and Bowen’s Family Systems Theory. CTD also represents trauma-informed practice heavily influenced by Levine (2017) and Van der Kolk (2019).

CTD has demonstrated clinical efficacy in reduction of dysregulation and affiliated psychiatric symptomatology across neurotypes. The CTD formula consciously challenges compelling urges toward symptomatic self-regulation, through use of a top-down and bottom-up approach. Incorporating an attachment-based, “making sense of” (Siegel, 2014) approach, CTD utilises unconsciously derived and engaged in interactional patterns to demystify dysregulated behaviour, toward reduction of dysregulation and symptomatology, and enhancement of dopaminergic connection.

Sporting both Lived Experience and a large twice exceptional client base, demonstrating high intellectual potential and neurodivergence, this article refers to the use of CTD in elimination of dysregulated behaviours and associated psychiatric comorbidities in this population. The fundamental purpose of CTD in this client population moves beyond intervention, to achieving potential. And toward active prevention of psychiatric symptomatology in subsequent generations.

With reference to the neurodivergent brain and dysregulation, societal cognitive reframing is required. Whether dysregulation is a direct result of a neurodivergent brain struggling in a world not designed to accommodate, or impacted by trauma, dysregulation is the problem, not the neurotype. Rather than being about solving the problem of Autism or ADHD, or of traits, it is about solving the problem of dysregulation; dysregulation that all neurotypes demonstrate.

In working with neurodivergent clients, therefore, the active psychosocial upskilling of clients using the methodology, toward reduced dysregulation and enhanced connection opportunities, deviates from being an individual-focused, deficits-based, social skills training methodology. CTD is a strengths-based methodology that highlights shared responsibility. Neither shaming/blaming, nor exonerating dysregulation and symptomatology, CTD shapes the person and those around them. Through recognition and support of valid neurodivergent need, and modification of shared dynamics in psychosocial interaction that develops core interactional skillsets synonymous with secure attachment, dysregulation is reduced. Optimal psychosocial and educational/vocational outcomes thrive, actively preventing associated psychiatric symptomatology common to the neurodivergent population.

While there is clear collective benefit for all in reaching individual potential, with humanity on the brink, we can scarcely afford losing the gifts of this extraordinary population. However, largely due to psychosocial difficulty, relational discord, and associated psychiatric disorder development, particularly when neurodivergence intersects with trauma, this

loss is real. Temple Grandin's, if it weren't for Autism we'd still be living in caves (2009), reiterates the collective benefit of protection of great minds so vulnerable to psychiatric comorbidities.

Although, co-occurrence is the identity-first language preferred by the neurodivergent community, co-morbidity will be used throughout this article, referencing the psychiatric conditions typically present for neurodivergent people. While the definition of co-morbidity, is, simply, two conditions occurring at the same time, the Oxford Dictionary's definition of morbidity is the condition of suffering from a disease or medical condition (<https://languages.oup.com>).

Suffering is the lived experience of many neurodivergent people and their loved ones. Existing under the cloak of hidden disability, this population is often misunderstood and inappropriately responded to in ill-equipped societal systems. Poor psychosocial and learning outcomes ensue, and with a neurobiology predisposed to major psychiatric comorbidities, severe suffering is likely.

Seen in clinical practice, ratified in research, are common comorbid psychiatric disorders including, but not limited to, Phobias, Obsessive Compulsive Disorder (OCD), and Social Anxiety (<https://autism.org>autism-and-anxiety>), Eating Disorders (Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder, Avoidant Restrictive Food Intake Disorder) and Substance-use Disorder.

Anxiety is at the root of the lived experience of the neurodivergent person and is frequently diagnosed in unidentified neurodivergence. Research suggests that roughly half of ADHD adults have an anxiety disorder, (<https://www.webmd.com>add-adhd>), (2021), while the percentage is around 40-50% for Autistic people (<https://www.austism.org.uk>topics>). This is unsurprising, given masking to avoid adverse psychosocial and educational outcomes, and the neurodivergent brain that struggles with change (transitions), fear of upsetting others, and doing the wrong thing (Rejection Sensitivity Dysphoria) (RSD) or instinctive, fear-based "no" (Pathological Demand Avoidance/Persistent Drive for Autonomy) (PDA). Likewise, experiencing poor psychosocial outcomes, on repeat, feeling unable to change the status quo, generates anxiety.

With anxiety as the cornerstone to severe psychiatric comorbidities, a brain predisposed to these, a non-ideal environment, let alone a context of trauma, typical in the neurodivergent population, is the hall-pass for worse to come. Approximately half the population diagnosed with anorexia nervosa (48-51%), bulimia nervosa (54-81%) and binge eating disorder (55-65%) is also diagnosed with anxiety disorder (<https://www.nationaleatingdisorders.org>). While systems are slow to change, psychosocial outcomes remain poor, and with many neurodivergent clients reporting abuse, psychiatric comorbidity prevails.

CTD has demonstrated strong efficacy with neurodivergent clients in reduction and elimination of dysregulation and psychiatric co-morbidity symptomatology. Perhaps, partly as CTD leans into neurodivergent strengths in the process of demystification of dysregulation and symptomatology.

Of the diagnostic traits outlined in the DSM-5 (2013) for Autism and ADHD, contributing to poor psychosocial and educational/vocational outcomes linked to dysregulation, are socioemotional challenges related to communication and interaction, restricted, repetitive behaviours/activities, and/or executive functioning difficulty. CTD's formula leans into the structure, routine, order, and rules this person enjoys, either because they are good at it, or requires, to feel psychologically safe in the socioemotional realm. While, rules can be challenging, especially for PDAers, they are welcomed when they reduce anxiety linked to socioemotional processes. This formula, therefore, incorporates a neurodivergent lens and a trauma lens, that can be followed when in an activated, dysregulated state, with reliable, eventually predictable, improved outcomes, lessening psychiatric symptomatology.

In its simplest form, the CTD formula is $1+1=2$. Application of CTD, works backwards from identification of symptomatology (2). The CTD formula begins with recognition that a dysregulated response, non- commensurate to the here and now, originates from a trigger that activates a nervous system reactive response, and catapults historical attachment- and trauma-based injury into the present. This past to present experience adds to poor psychosocial outcomes, influencing psychiatric symptomatology in urges toward unproductive self-regulation. Further, CTD application consciously connects the dots between trigger, potentially closely resembling the past trauma, or looking nothing like it, and trauma- and attachment-based themes and narratives synonymous with unconscious attachment-based interactional patterns.

Hence, recognition of triggers and attachment- and trauma-based themes and narratives is fundamental to CTD. As is timely application of a Time Out oriented circuit breaker, once activated, to follow $1+1=2$ in processing the activated response. This Time Out provides space to unpack the part neurobiology (1) and context, past and present (+1) have played in generating the activated response and symptomatology. Disentangling past from present, with safe platform to lean into the feelings attributed to past, releasing the toxicity attributed to present, allows the client to examine the real cues in the current situation, thereby reassessing safety, danger and life threat.

Neurobiology (1). Crucial in psychiatric intervention and prevention in subsequent generations, is recognition of neurobiological predisposition to psychiatric symptomatology in the neurodivergent population. The fictitious, very bright, high performing client, "Millie", presenting with Generalised Anxiety Disorder (GAD), and joking about a touch of OCD, may be bringing more to the table.

While inaccurate, over-diagnosis of neurodivergence is a concern, missed neurodivergence can be life-ending. Without neurodivergence identification, caution is inadvertently thrown to the wind, the family unwittingly engaging in psychiatric-disorder-Russian roulette, with pre-disposition to psychiatric comorbidities waiting to be set off by adverse psychosocial/educational circumstances. Accurate identification of neurodivergence, leading to shared-responsibility oriented psychosocial intervention, plays a pivotal role in best outcomes. Accurate identification of neurodivergence requires robust understanding of the traits of neurodivergence, specifically the traits of the different presentations (female/internalised presentation versus male/externalised presentation). Also of import, is recognition of neurodivergent relevant issues of interoception, alexithymia, masking, difficulty with transitions, sensory issues (E.g., food/clothing/noise issues), Autistic overwhelm and burnout, RSD and PDA.

Without this knowledge base, assessment is compromised, hence, to work collaboratively with the neurodivergent person and those closest to them, managing the presentations and co-occurrences, without demonising the less charming, more challenging “quirks” synonymous with traits. Thereby, missing opportunity to minimise dysregulation and achieve enhanced psychosocial outcomes that can prevent psychiatric symptomatology. Opportunity is missed to harness neurodivergent capacity.

Of note, failure to work with these presentations, clinically, can result in further traumatising in the therapeutic context. For instance, without knowledge of PDA, intervention in Anorexia Nervosa can make the symptomatology exponentially worse.

Invaluable in accurate identification of neurodivergence, is exploration of the broader family network. Identification of developmental co-occurrences, including learning disorders such as visual and auditory processing issues, and neurological co-occurrences, including Tic Disorders, such as Tourette’s, along with psychiatric comorbidities in the client’s children and extended family system, uncover potential neurodivergence, otherwise missed.

“Millie” attracts greater attention from the neuro-affirming clinician, from reports that her daughter was diagnosed with dyslexia, demonstrates sleep issues and separation anxiety, unusual eating habits (sensory and routine-related) and hates change, alongside a niece with Anorexia Nervosa during COVID-19 Lockdowns, and a brother with Substance-use disorder in his teens. Potential neurodivergence throughout the family system, if missed, places “Millie” and her children in the psychiatric-disorder-Russian roulette queue.

The second category in the CTD framework (+1) develops cognitive understanding of dysregulation by actively working with the parts of the dysregulation puzzle that can be modified. Recognition of context, past and present, generating dysregulation and psychiatric symptomatology, utilising related neuroscience, has been shown to foster sustainable change across neurotypes.

Therapeutically relevant for the neurodivergent brain, is the tendency to struggle with nebulous, and feelings-oriented work. Plus, interoception issues can make body-based work harder.

While the externalised presentation struggles with empathy, the internalised presentation tends to become overwhelmed by the emotions of all. And neither the ADHD nor Autistic brain appreciates nebulous. The ADHDer gets lost. The Autistic client becomes frustrated by lack of structure. Hence, CTD, featuring clear structure, along with neuroscience and pattern recognition that neurodivergence favours, providing psychological holding for all, has shown clinical efficacy with this population.

The second category of CTD, therefore, crucial to demystification of dysregulation and intervention, and instrumental in teaching clients how to apply this intervention themselves, outside of therapy, pertains to pattern identification. Attachment patterns make sense of socioemotional processes unconsciously engaged in; the area many neurodivergent brains struggle most with.

Attachment-based interactional patterns in most adult clients, regardless of neurotype, originate from insecure anxious and avoidant, and disorganised attachment, representing inconsistent caregiver attunement, consistent non-attunement and, for some, alarming responses in the caregiver respectively, that correspond to specific, compromised interactions with important others (Bowlby, 1988; Ainsworth et al, 1978; Main and Solomon, 1990; Bartholomew, Henderson and Dutton, in Clulow 2001, Siegel and Hartzell, 2014; Schore 2016) In combination with the associated emotional climate in the family of origin, chaotic or cold for insecure anxious and avoidant (Siegel 2008, 2012, 2015), let alone dangerous, these experiences wire neurobiology to guide us, unconsciously, like a magnet, to what is familiar (Schore, 2016, Siegel, 2008, 2012, 2015), good or bad.

Bartholomew, Henderson and Dutton’s (in Clulow, 2001) two-dimensional, four-category model of adult attachment highlights the ongoing deleterious interactional effects of childhood insecure attachment, with development of a positive or negative model of self, and positive or negative model of other, that determines compromised interactional patterns in a partnered relationship. Reflected in practice over more than two decades, the consistency, and predictability of the specific interactional patterns of Preoccupied, Fearful or Dismissing attachment showcase these as being of profound therapeutic utility. Enabling shifting of interactional patterns from unconscious to conscious allows for modification of interactions in the real world, outside of therapy.

Using Fearful attachment, cognition of a lifetime of having been unconsciously locked into deferring, accommodating and second guessing is a powerful change agent. When clients fully recognise how their history informs their current interactional outcomes, there is platform to challenging these. For Fearful attachment, this means recognition of childhood

formation of negative model of self, representing fear of rejection and abandonment in close relationships, and negative model of other, avoiding close contact, not turning to others for security, and distancing to maintain some indirect support through not upsetting the other, having adaptively learnt to anticipate unresponsive or rejecting attachment figures (Bartholomew, Henderson and Dutton, in Clulow, 2001) Demystifying this client's fear of intimacy and associated, unproductive interactional behaviours that set the scene for repetition of poor psychosocial outcomes, gives rise to true behaviour modification, including core interactional skillset development.

To expand, all insecure attachment-based interactional patterns demonstrate compromised core interactional skillsets that prohibit healthy relating. Healthy and appropriate self-expression, self-assertion, limits and boundaries, and power and control, enabling capacity to negotiate difference and resolve conflict, and showcasing optimal self- and co-regulation and self-management capacity, are absent or compromised in insecure attachment. Development of the core interactional skillsets, required for improved psychosocial outcomes, falls flat without awareness of the unconscious interactional patterns from childhood.

To return to "Millie", reporting people pleasing, gives vital clue helpful in intervention, with minimal other information. Enquiry as to her tendency to defer and accommodate with everyone, met with a resounding yes, clarifies. As does reports of heavy self-reliance and independence, that her employer enjoys, but her partner dislikes. Fearful attachment is identified. And corroborated with background story of "Millie's" mother as sole parent, working two jobs, consistently unavailable, despite love and best intentions, who yelled, scaring her at times.

Effective intervention with "Millie", therefore, necessitates development of core interactional skillsets, including capacity to self-assert appropriately. This is difficult without addressing the unconscious attachment-related content that drives this deleterious interactional pattern.

Of note, because of the reliability of patterns, minimal information gathering provides maximal therapeutically relevant content without the minutiae of a client's attachment injury, or trauma, reducing risk of re-traumatizing reactivation, particularly before the environment of safety is fully established. Additionally, accurate assessment can be formed rapidly. Simply understanding the family of origin interactional climate and attachment-related interactional issues, gives vital clues to the contextual challenges in relating in current relationships, influencing dysregulation and psychiatric symptomatology.

Caregiver capacity to provide affect regulation, right brain to right brain, upregulating in boredom and down regulating in stress and distress (Schore, 2016), to provide the safe and secure base from which to come and go (Bowlby, 1988), establishes the caregiver as a valid source, someone to seek

out for coregulation in times of stress and distress. For insecure attachment, lack of secure base means the child must find other means to re-regulate in stress and distress, such as turning to food or substances to regulate, that can ignite a lifetime struggle. "Millie's" crippling anxiety and binge eating episode after a performance review with her historically supportive boss, exemplifies dysregulation and psychiatric symptomatology.

While neurodivergence is not the result of poor parenting, many neurodivergent clients report insecure attachment with parents struggling to manage their own dysregulation in the face of parenting the neurodivergent child, without neuro affirming resources, particularly when the parent is unidentified and unmanaged neurodivergent themselves. Prevention of the known psychiatric comorbidities in neurodivergence for the subsequent generation, therefore, heavily relies upon provision of the attuned caregiving of Secure attachment. This necessitates development of self-attunement, to self-regulate and self-manage toward optimal parenting, in the form of earned security, for the parent-client who does not present with Secure attachment.

Toward optimal psychosocial outcomes for self and other, CTD identifies attachment-based, interactional pattern-related themes and narratives that link to likely triggers and subsequent activation responses. Narratives of "I am not (consistently) seen/heard", "I am not good enough", "I have no power and control" are common in Preoccupied and Fearful attachment, while "I am fabulous, everyone else is an idiot", and "I need and want to take control" are common in Dismissing.

Clinically evident, is the dangerous combination of Fearful and Dismissing in partnership, generating an environment that lends itself to coercive control. This combination is even more problematic when combined with the rigidity of some neurodivergent presentations.

Individual-specific experiences, including trauma, add to themes and narratives relevant to triggers and subsequent responses. Relational examples include, "People are unsafe/abandoning", "Everyone transgresses my boundaries", "Being seen is unsafe". Similarly, trauma-specific experiences add triggers to environments. For instance, sexual assault on holiday translates, obviously, to trauma activation with the trigger of travelling. Less obviously, in any environment change, such as moving house or workplace. This distress will be exacerbated for the neurodivergent person who struggles with transitions. Awareness of attachment- and trauma-based themes and narratives, arms and forewarns.

With CTD's therapeutic arsenal, "Millie's" dysregulated response with her supportive boss, when put into perspective with historical context, makes sense of otherwise baffling circumstances. Experience with an intelligent mother with unmet career aspirations, reactively angry and withdrawing for long periods when "Millie" "failed to do her best", translates to the themes and narratives, "I'm not seen" combined with "being seen/performance is dangerous", "I have no control",

“I’m not good enough”, and “People are unsafe/abandoning”. These themes are further emphasised with reports of being the “weird little kid, left alone to read in the corner”, when she wasn’t being bullied at school. Unsurprisingly, she has a reactive, activated response in the current safe context. Which leads to the neuroscience, crucial to this second category of CTD, and inextricably linked to attachment- and trauma-based themes and narratives that impact triggers and subsequent responses.

Schore (2016), Seigel (2008, 2012, 2015), Levine (2017), Van der Kolk (2019) and Porges (2019) provide eloquent explanations of the central nervous system and peripheral nervous system, and the deleterious impact of trauma, complex and relational, on the neurobiology, notably the Right Brain Limbic Autonomic Network (RBLAN) (Schore, 2016). That trauma, including attachment injury, shapes the structure and function of the brain (Schore, 2016), consequently, all aspects of living in the world, is well known.

Hence, fundamental to symptom reduction in CTD, is client recognition of the neurobiology beneath the non-commensurate-to-current-context, dysregulated response to a trigger, bringing toxic past into, at times, safe present. Recognition of the triggered bodily response has shown strong utility in intervention, especially in the neurodivergent population that favours cognitive understanding. This includes identification of clues from the viscera in an activated state and understanding of the Autonomic Nervous System (ANS).

CTD utilises Porges’ Polyvagal Theory (2019) to demystify compelling, reactive urges to a trigger in an activated state. Firstly, among other neurological impacts of trauma, is nervous system capacity to accurately read safety, danger and life-threat (Porges, 2019; Levine, 2017), leaving the individual vulnerable to poor decisions that lead to poor psychosocial outcomes, often requiring regulation through unproductive means.

Neuroception detecting safety enables social engagement and co-regulation opportunities, whereas neuroception of danger generates the mobilised responses of fight, flight and fawn of the Sympathetic Nervous System (SNS), or the immobilised response, Freeze, of the Parasympathetic Nervous System (PNS), incorporating the Vagus Nerve (Porges, 2019). Awareness of corresponding unhelpful interactional and behavioural patterns of reactive withdrawal (flight), reactive anger/anxious/chaotic response (fight), people pleasing (fawn) and shutdown (freeze) at a trigger, can shift the reported bewildering, compelling response to one that “makes sense”.

For instance, once “Millie” is aware that her ANS is simply doing its job, trying to keep her safe, but is misdirecting, due to trauma, she has scope to interrupt this. Therapeutic intervention with “Millie”, therefore, teaches her to unravel what occurred around the trigger. Already primed in an environment representing danger (“performance equals danger”), triggered by unfamiliar content and a question she

did not know the answer to, led to the reported SNS flight and mobilisation attempt of withdrawal, presenting as quiet voice, staccato speech, less engagement, smaller physical presence, and agitation. This shift led to a series of questions from her (still supportive) boss endeavouring to re-engage “Millie”, further activating her. In her struggle to respond impressively, possibly now actually failing to perform in a heavily activated state, she stews in trauma soup that reinforces narratives she is indeed “not good enough” and being “seen/performing is dangerous”. “Millie’s” lid is well and truly flipped, referencing Siegel. She cannot perform at her best during prefrontal cortical disengagement and loss of executive functioning. Without psychosocial self-protective skillsets yet, “Millie” deteriorates into immobilisation and freeze, with an incapacitating panic attack ensuing, and later, re-regulation attempts through binge-eating, filled with shame.

Along with ANS understanding, the second category of CTD incorporates recognition of body-based responses to a trigger, where possible, and takes note of chronic physical conditions comorbid with psychiatric symptomatology, especially with trauma history, such as persistent Urinary Tract Infections (UTI’s), Endometriosis, and chronic fatigue. As Van der Kolk coined, “The body keeps the score” (2019). Noting body responses, such as rapid heart rate, head and neck pain, or constricted throat, can assist the client to, at the time, or working back, identify when, therefore, what, they were triggered by, crucial in application of CTD intervention, including the development of psychosocial self-protective skillsets. Identification of constricted throat, for example, indicating early activation, can assist “Millie” to note that she is in an activated state and needs to excuse herself momentarily from the review, to re-regulate. This awareness, after-the-fact, enables recognition of point of activation, crucial in processing and eventual self-intervention.

Likewise, CTD looks for complex trauma activated body-responses, both below the diaphragm, including Gastrointestinal issues, and legs collapsing (Porges, 2019), and above, such as losing one’s words with Wernicke’s Area and Broca’s Area compromised (Van der Kolk, 2019). Heavily activated, now failing to perform at her best, unsurprisingly, “Millie” loses her words, and feels like throwing up, indicative of life-threat and trauma activation.

CTD follows the notion of the Chinese Proverb; there is greater benefit when you “Teach a (person) to fish”. For “Millie”, following the 1+1=2 formula demystifies dysregulation at a trigger, and the resulting anxiety and eating disorder symptomatology. Understanding she has experienced a historical attachment- and trauma-based nervous system activated response in the current context, generating compelling and insistent fear of being in trouble or abandoned in poor performance, or of being directly targeted, potentially exacerbated by neurodivergent-related traits, such as rejection sensitivity, normalises the response, and develops self-compassion.

With a mindset of “No wonder...” (the dysregulation and symptomatology), removing shame, equilibrium can be re-established faster. Through reflection validating the compelling feelings, but with direct reference to past, space is created to explore, and challenge, safety versus danger of the current context; “Millie” can truly recognise, then, that she was in no danger. This provides opportunity for newfound capacity to thwart unproductive self-regulation. Combined with shared-responsibility core interactional skillset development, “Millie” has burgeoning capacity to make early attempts at co-regulation with a close other when activated, rather than through food, crucial with neurodivergent predisposition to eating disorder evidenced in the family system.

As “Millie” becomes more proficient at 1+1=2, identification of triggers and working with attachment- and trauma-based themes and narratives at the root of nervous system dysregulation and psychiatric symptomatology, becomes faster and more reliably successful in symptom reduction. This motivates further use of the 1+1=2 processing at activation, gradually reducing time taken to re-regulate, and of psychiatric symptomatology episode frequency, intensity and duration.

CTD’s 1+1=2 structure, featuring awareness, and appropriate identification of, and working with, neurobiology pertaining to neurodivergence, and neuroscience related to attachment and trauma to solve the problem of dysregulation, leaves “Millie” psychosocially upskilled and free to reach her potential. Creating sustainable change, reducing cost of psychiatric symptomatology across neurotypes, but highlighting the brilliance of neurodivergence, 1+1=2 can teach a client how to fish; and save the planet.

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