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A cure for PTSD? An outcome achieved through Structured Image Framework Theory, exposure therapy and ketamine

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This case study explores how an Intensive Care Paramedic (ICP) experienced a significant decline in his diagnosed chronic post-traumatic stress disorder (PTSD) and co-morbid depression and anxiety symptoms following a spontaneous episode of Rapid Atrial Fibrillation (AF) treated with ketamine and cardioversion emergency medical intervention. This article explores how by using therapeutic interventions from a newly developed Structured Image Framework Theory (SIFT) and exposure therapy interventions from prolonged exposure therapy (PET) and Eye Movement Desensitisation and Reprocessing (EMDR) a diagnosed chronic PTSD ICP was able to return to full non-operational 'off road' emergency crisis duties. This was owing to developing a more reliable capacity to effectively engage with *in vivo* exposure exercises involving triggering environmental stimuli, gaining a greater sense of control, understanding the volatility of traumatic triggering, and developing emotional tolerance in order to maintain self-regulating control. This continued to be evident during ongoing PTSD triggering and while being exposed to paramedical emergency information repeatedly disseminated throughout his Australian governmental ambulance working environment. Approximately 12 months after being diagnosed with chronic PTSD, this ICP experienced a life-threatening spontaneous episode of rapid AF, which required an emergency ambulance response to hospital. Once Mr T. reached the emergency department (ED) he was initially given a magnesium infusion and then a bolus dose of verapamil. He was in ED for approximately 2 h prior to his cardioversion. He was given an 80 mg injection of ketamine to sedate him before receiving a 100 joules external shock defibrillation via multifunction electrodes (MFE) to restart his heart, which was unsuccessful in achieving an appropriate heart rhythm reset. Mr T. was then administered 200 joules, before his heart rate reset into sinus rhythm. The 80 mg ketamine injection and subsequent 100 then 200 joules heart restart defibrillations were completed within 3 min. As soon as Mr T. received his 80 mgs of ketamine, he subsequently had a successful 200-joule defibrillation cardioversion. He was not aware of his cardioversion as he experienced what he described as shifts in his ability to emotionally connect and process the world around him. While he remained in a resus bed in ED for the next 17–18 min with his eyes open supported initially by fellow paramedic friends, he described feeling like he had experienced a 'factory reset'. Mr T. also reported that, after returning to the reality of the ED recovery room, it took approximately 8 h for the effects of the ketamine to wear off and then he went to sleep. The following day Mr T. felt a significant drop in PTSD, depression and anxiety symptoms. This was assessed in clinical therapy sessions and with self-report scales within 2 days of his rapid AF cardioversion episode and then over the next 18 months, with remarkable results. In conclusion, this paper explores how the combination of therapeutic interventions from down-regulating practices – SIFT, PET, and EMDR – prepared Mr T. for the cardioversion rapid AF/ketamine-induced journey that shifted his capacity to process his internal and external worlds. What could have been affected his brain functioning, which then significantly decreased his PTSD, depression and anxiety symptom levels, for now over 18 months? He has not required any ongoing psychiatric medication support throughout his journey and only received therapeutic monitoring sessions since his significant reduction in PTSD symptom levels. To date, Mr T. continues to maintain full-time non-operational 'off road' duties within his ICP Australian governmental ambulance workplace environment.

Contribution: This remarkable case has significantly shifted an intensive care paramedic's PTSD condition to a predictable long-term stabilised state.

Keywords: chronic PTSD treatment; structured image framework theory; ketamine; rapid atrial fibrillation; trauma exposure therapy; Intensive Care Paramedic (ICP); Structured Image Framework Theory (SIFT); rapid atrial fibrillation (AF).

Introduction

In this case study, Mr T. is a highly experienced intensive care paramedic (ICP) who has been exposed to over 26 years of operational life-threatening incidents and now over 2 years of full-time non-operational 'off road' emergency crisis project duties within an Australian governmental ambulance work context.

The combination of this emergency worker being stretched as well as shifting from one demanding life-threatening crisis to another had the capacity to limit his ability to adjust to ongoing escalating levels of potential traumatisation in the workplace.

Clinical presentation

Mr T. initially presented as a 51-year-old male, with 26 years of ambulance service as well as 4 years of Australian Army Reserve Service (Armoured Corps). He has been married for over 20 years to his wife (a registered nurse), and has 2 adult children (a son and a daughter).

Despite being highly experienced and an effective instructionally advanced ICP, Mr T. progressively started to emotionally fluctuate, discombobulate, break down and deteriorate in his everyday functioning capacity. This alarmed him because of his high personal and professional standards. With encouragement from close friends and family as he struggled to maintain his previously assessed exceptional professional standards and ability to serve the community in crisis, he reluctantly sought psychological support.

As part of improving Mr T's ability to manage, understand and conceptualise his erratic post-traumatic stress disorder (PTSD) symptoms, and to prepare for traumatic memory and/or imaginal targeting within exposure therapeutic protocols with Structured Image Framework Theory (SIFT) (Wilson, 2018, 2019), prolonged exposure therapy (PET) (Foa et al., 2007) and Eye Movement Desensitisation and Reprocessing (EMDR) (Shapiro, 1989), he produced 38 pages of traumatic memories and vivid image descriptions in a comprehensive, emotionally disturbing narrative script.

For illustrative purposes, some of the examples of multiple traumatic events and detailed image intrusions were associated with the following descriptions:

Motor vehicle accidents

Brain matter stuck in tread of his boot; crushed, somewhat liquified body like rag doll; eye on road; tree crushing a car.

Individual accidents and suicides

A female pouring petrol on herself and lighting it; burnt human flesh; train impact body in a ball; major highway jumping body in a ball shape; child push bike truck impact.

Assault and/or Murder

Sliced open abdomen, intestines; assailant coldly admitting to being the murderer at the scene, while Mr T. was attempting to administer medical assistance to the dying and recently deceased victim; rape, blood, abrasions, sobbing, and devastated victim.

Paediatric arrest

Mother not giving over deceased baby, sobbing, highly distressed parent with many questions about how and why.

Clinical diagnosis

During his 26 years of operational ('on-road') emergency crisis ambulance ICP service, Mr T. experienced life-threatening incidents including murders, assaults, motor vehicle accidents (MVAs), suicides, paediatric deaths, life-threatening community aggression and hostility, and the devastation of Australian natural disasters. Unfortunately, owing to his ongoing accumulative exposure to the devastating aspects of repeated fatalities and life-threatening incidents necessitating an emergency response, Mr T. repeatedly experienced the horrors of human tragedy. Over time this reduced the emergency first-responder's capacity to access timely traumatic reprocessing intervention and supports. He repeatedly experienced time-pressured 'on road' emergency crises, received fluctuating levels of distributed information and had shifting resources relevant to the medical interventions. There were also increasing levels of media and community scrutiny surrounding the urgency in saving lives where ambulances were required. This overall situation was not helped by the repeated lack of any clear understanding by his employer of how traumatic exposure can affect the emergency worker acutely and accumulatively in the long term.

The potential ongoingly slow deterioration that can occur in emotional regulation, focus and concentration, and the corresponding increase in avoidant behavioural patterns, anger outbursts, tearfulness, intolerance, dismissiveness, emotional detachment, emotional numbing, random traumatic memory intrusions and sleep disturbances can be hidden as the emergency worker strives to save others within the community. The potential development of traumatic symptoms can further be masked and 'downplayed' within the crisis intervention culture and attributed to extra demands with lack of resources and fatigue caused by shift work hours across the 24-hr timeframe of serving in the emergency field. All these factors contributed to Mr T. being clinically assessed using the Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1995) and the PTSD Checklist (PCL-5) (Bovin et al., 2016) self-report scale and diagnosed to have developed a PTSD (Criteria 309.81; DSM 5, 2013) condition. Mr T. was further assessed with several confirming self-report scales such as the Depression Anxiety Stress Scale (DASS 21) (Lovibond & Lovibond, 1995a, 1995b); Beck Anxiety Inventory (BAI) (Beck et al., 1988); Beck Depression Inventory-II (BDI-II) (Beck et al., 1996) and the Beck Hopelessness Scale (BHS) (Beck et al., 1989).

Treatment interventions

Mr T's initial highly distressed state from his developed PTSD (Criteria 309.81; DSM 5, 2013) symptoms involved deteriorated concentration and persistent ruminating on high over-analysis of potential emergency scenarios that kept intruding into his everyday functioning which made no sense. He reported that it was essential for him to understand how traumatic triggering activated the more powerful primitive parts of the brain during increased emotionally unsafe situations and how that would override his capacity to conceptualise and operate effectively.

Structured image framework theory (SIFT)

Using conceptual visual frameworks from SIFT (Wilson, 2018, 2019) with descriptive mapping diagrams created a strong therapeutic bond and a higher sense of control over the chaos Mr T. was initially experiencing.

The predictable mapping of how traumatic impact develops within Acute Stress Disorder (ASD) and PTSD (DSM 5, 2013) symptom clusters with diagrammatic SIFT descriptions (Wilson, 2019) further enabled Mr T. to feel a greater sense of safety approaching the challenges of further exposure therapy interventions. This included PET (Foa et al., 2007) and EMDR (Shapiro, 2012), and they were repeatedly practiced as his capability to better tolerate the increased arousal levels with down regulating exercises (controlled breathing, relaxation, imagery, hypnosis guided meditation scripts and improved sleep hygiene practices) improved (Toussaint et al., 2021; Vickers & Zollman, 1999).

Structured Image Framework Theory enabled Mr T. to understand the brain functioning aspects (Wilson, 2018) of how his ICP emergency ambulance service traumas have accumulated over his 26 years of experiencing front-line community crises. It also predictably mapped how aspects of PTSD have developed and how his traumatic history is now designed for him to survive in the future through multiple emotionally volatile symptom levels.

As depicted in Figure 1, SIFT enabled a reluctant 'street smart' paramedic to understand how reliving of the trauma with flashbacks, nightmares, intrusive thoughts and memories was occurring in his current suffering state.

Structured Image Framework Theory enabled Mr T. to understand how his behavioural or cognitive attempts to avoid trauma-reminiscent stimuli, the suppression of emotionally upsetting thoughts/memories and emotional numbing developed over the latter years of his ambulance service. He was able to describe his hyperarousal with decreased or restless sleep, muscle tension, irritability, jumpiness, fluctuations in concentration and attention difficulties. He could also understand his dissociative responses, including depersonalisation, feeling 'spaced-out' (cognitive-emotional disengagement), amnesia or 'missing time' and identity alteration or confusion associated with his traumatic history. He understood his increases in substance abuse and somatic disturbance, high sweating episodes, some preoccupation with bodily dysfunction and chronic pain intrusion with increased emotional distress that could not be medically explained (Briere & Scott, 2006; DSM 5, 2013).

As evident in Figure 2, SIFT could also describe to Mr T. any sexual difficulties occurring, cognitive disturbance with low self-esteem, helplessness, hopelessness and excessive or inappropriate guilt or shame in how his functioning had significantly shifted into more unpredictable levels. His developed PTSD could also be mapped using SIFT (Wilson, 2014, 2018, 2019) to describe where tension reducing behaviours such as impulsive aggression and workplace system frustrations have also been linked to PTSD clients' best attempts to adapt using maladaptive behavioural patterns (Briere & Scott, 2006; DSM 5, 2013) to regain control of the accumulated traumatic chaos felt within.

Mr T. found the SIFT descriptive framework concise, simple and dynamic. This enabled him to apply management of his PTSD symptoms within his own individual belief structures as he prepared to commence a graduated return to work (RTW) within non-operational 'off road' ambulance service duties. Structured Image Framework Theory also prepared him to use a visual structure based on how the brain functions when a person is experiencing PTSD-triggering symptoms. Mr T. reported that SIFT assisted him in writing an immersive 38-page emotionally challenging narrative account of his ICP traumatic history and in understanding how exposure therapy techniques would activate him during therapy.

Prolonged exposure therapy and eye movement desensitisation and reprocessing (EMDR)

Mr T. could understand that SIFT (Wilson, 2019) was designed as an inclusive neuro-psychotherapeutic approach capable of forming the foundation for multiple therapeutic techniques such as narrative descriptions, PET (Foa et al., 2007) and EMDR (Shapiro, 2010, 2012), which can be used to further process trauma to an adaptable stabilised level. He reported that SIFT gave him hope by empowering him to understand the multiple dynamics associated with his accumulated traumatic history, his development of PTSD and how exposure therapeutic dynamics operate to process his traumas in the future.

From a psychobiological basis (van der Kolk, 1994; van der Kolk et al., 1996), trauma-processing mechanisms have also been described in which stressful experiences can excessively stimulate the amygdala, interfering with the hippocampal functioning. The hippocampus has in turn been described as inhibiting cognitive evaluation of experience and semantic representation. Shapiro (1989) further indicated in relation to EMDR that images form the basis of, or vehicle for, treatment dynamics that encourage sequenced trauma processing by the client in accordance with their established belief structures.

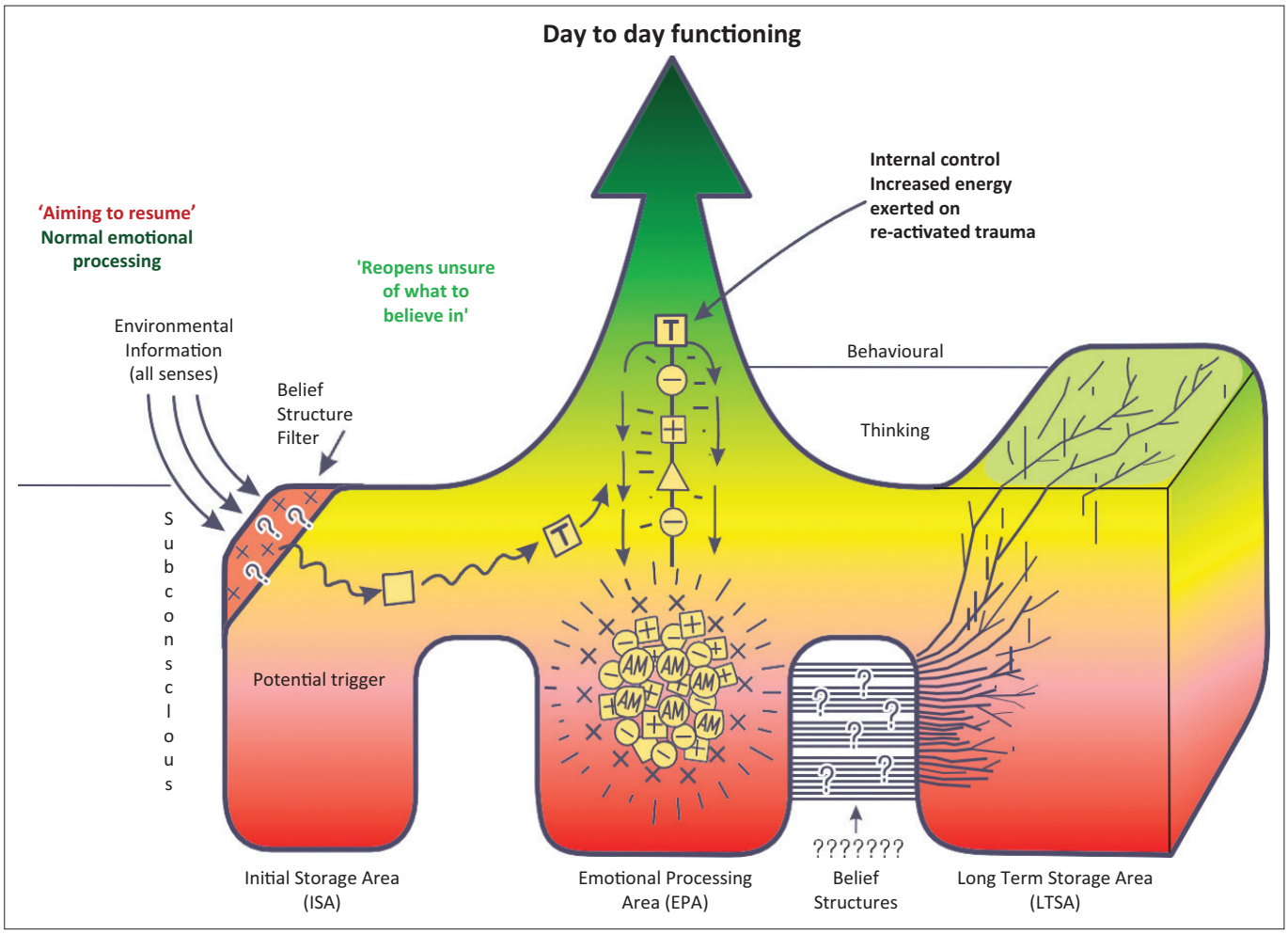


FIGURE 1: Structured Image Framework Theory (SIFT): Aiming to resume normal emotional processing following traumatic impact. Copyright by D J Wilson, Clear View Psychology Services Pty Ltd.

The aim of traumatic exposure is to personally challenge the shattered belief in, or understanding of, a person's world view. This in turn must be personalised and integrated into the person's life history of experiences (Van der Kolk & Ducey, 1989) in a more emotionally stabilised state. Bryant's (1997) review of psychological treatment of PTSD supports the use of behaviourally orientated therapy initiatives that understand specific trauma processes and how interventions may interact with an individual's distorted beliefs as being beneficial in trauma treatments. In summary, traumatic processing is highly individualised because of what a client brings to a shocking life-threatening incident. Trauma has the potential to shatter a client's understanding of their world with more permanent maladaptive changes (Shapiro, 2012; van der Kolk, 1994). Shapiro also acknowledges that therapy should endeavour to combine a variety of techniques in creative collaboration with the client to promote trauma adaptation (Shapiro, 2010). This, when combined with down-regulation exercises and trauma reprocessing approaches, enables more adaptive cognition and behavioural patterns to potentially develop greater PTSD adaptive growth.

The developed SIFT model (Wilson, 2014) enabled both the clinician and Mr T. to understand the processes involved in adapting to complex, overwhelming and disorganised traumatic stimuli that are beyond a person's current capabilities. An overwhelming sense of personal loss of control was a fundamental consideration for Mr T., who was experiencing significantly elevated psychological distress during a forced time of adaptation. This consideration is typically exacerbated by the client lacking a full understanding of how they have arrived at their current highly emotionally distressed state. In this situation, SIFT was invaluable in enabling him to understand how instantaneous raw traumatic stimuli experienced during trauma then activates past memory processes within a framework. These processes revealed to Mr T. how predictable survival-based behavioural changes could occur during and after treatment. As depicted in van der Kolk's (1994) schematic representation of the effects of emotional arousal on declarative memory, explanatory models can give the client and clinician a greater applied understanding of trauma processing. Mr T. openly reported that his developed understanding within an applied SIFT model was concise, simple, flexible and dynamic enough to enable him to review the past, present and future elements of his psychological state. This was while he repeatedly challenged his traumatic ICP accumulated history during a graduated RTW schedule as he secured full-time non-operational 'off road' ambulance duties.

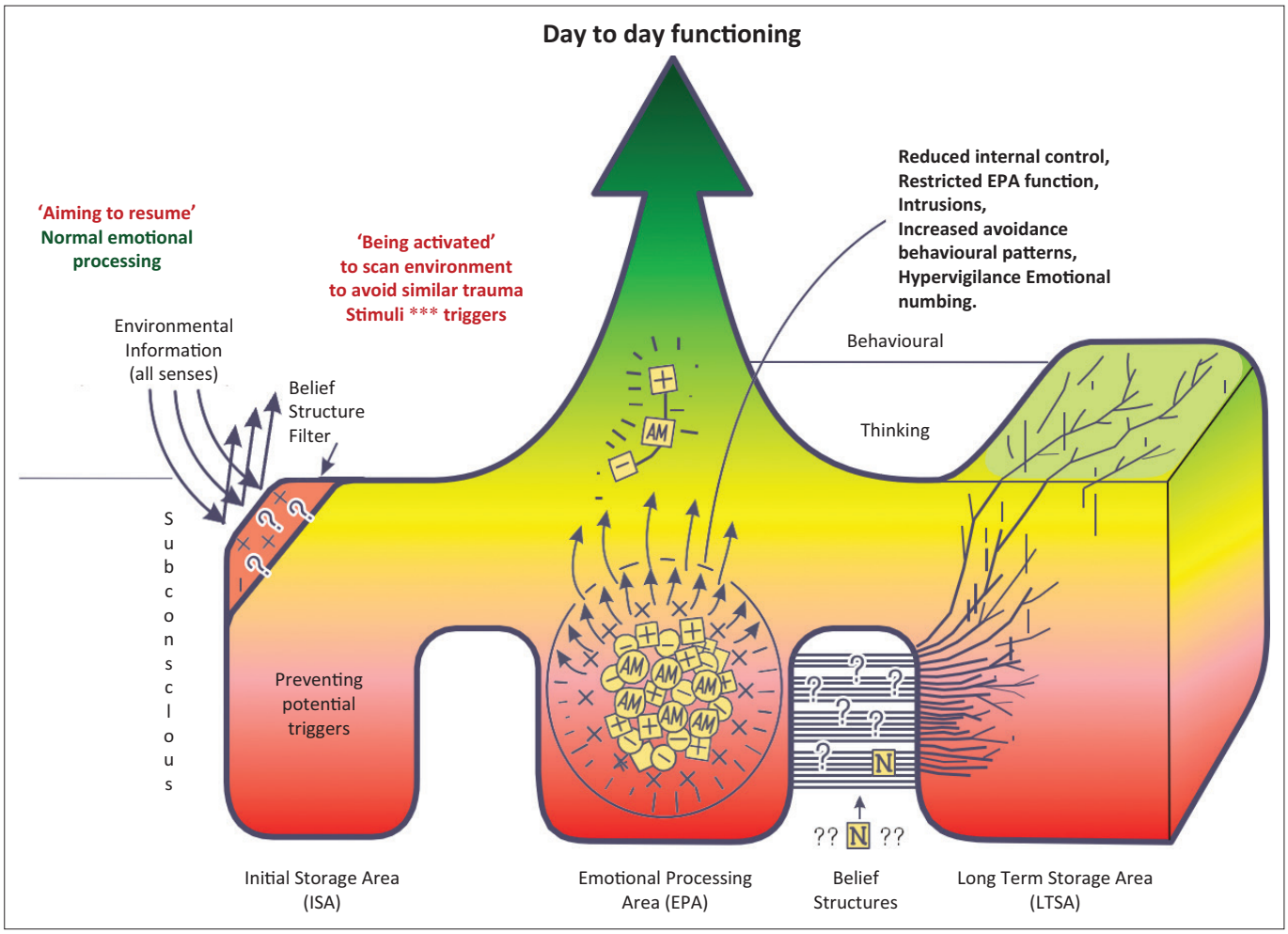


FIGURE 2: Structured Image Framework Theory (SIFT): The potential development of post-traumatic stress disorder. Copyright by D J Wilson, Clear View Psychology Services Pty Ltd.

Return to work at full-time non-operational 'off road' paramedic duties

As part of standardised requirements and recommended treatment directives within Australian Government psychological counselling support for injured workers, Mr T. was required to undergo regular self-reported psychometric assessments. This was in conjunction with recommended ongoing treatment plan reviews necessary every 6 to 8 sessions. Sessions took place on a 1–2 weekly schedule within 60-90 minute timeframes. This is recommended by approved psychologists to facilitate timely trauma reprocessing, support a safe and durable RTW under workers' compensation provider directives. The following self-report assessments were conducted on 2–4 monthly schedules, according to ongoing insurance approval requests for further treatment sessions to support Mr T's RTW upgrading capacity.

Mr T. was able to return to work after being initially medically unfit on 05 November 2020, then gradually increased his hours towards securing full-time non-operational 'off road' emergency ambulance duties on 05 March 2021. During Mr T's initial 4 months of treatment, he developed a greater predictable understanding of reducing and moderating his PTSD symptoms (PCL-5) (Bovin et al., 2016), which has shown to be consistent with results when using the CAPS associated with the DSM-5 (2013) criteria. The PCL-5 is thus a psychometrically sound instrument to optimally diagnose PTSD (K[0.5] = 0.58). Further improvements in Mr T's stress (within DASS), anxiety (within DASS; BAI) (Beck et al., 1988), depression (within DASS; BDI-II) (Beck et al., 1996) and hopelessness/avoidance (BHI) (Beck et al., 1989) levels enabled him to RTW within supportive, safe and predictable 'off-road' clinical and operational administrative ambulance project roles with ongoing counselling. Research supports multiple self-reports with the DASS anxiety scale correlating at 0.81 with the BAI, and the DASS depression scale at 0.74 with the BDI (Lovibond & Lovibond, 1995a, 1995b).

The RTW of Mr T. to full-time non-operational 'off road' duties successfully occurred over 6 months with timely exposure therapy 60–90 min counselling sessions, prior to his rapid AF life-threatening incident.

The following case study descriptions from Mr T. being rushed to hospital as he suffered a life-threatening rapid AF episode forms an unexpected extra medical treatment dynamic. This additional critical life-threatening incident

came as he was experiencing a clinically observed reduction in PTSD, anxiety and depression symptom levels.

Mr T's cardioversion episode in response to a spontaneous episode of rapid atrial fibrillation involving 80 mg of intravenous ketamine and a 200-joule heart re-start

Mr T. was rushed to hospital emergency department (ED) to be cardioverted for a spontaneous episode of rapid AF. The cardioversion was facilitated by a twilight anaesthesia dose of 80 mg of intravenous ketamine and a heart re-start of 100 then 200 joules within 3 min to re-establish his sinus heart rhythm.

Mr T's first-hand account of his ketamine hospital ED experience over approximately 20 minutes

Mr T. recalled that the induction of the 80 mg dose of intravenous ketamine was *'similar to the sensation of other anaesthetics'* that patients reported during his years of ICP experience.

Mr T.'s induction

He described that he:

> was immediately given a sense of zero gravity, along with hearing a loud oscillating sound, that was like a cross between the sound of heavy machine gearing whirring and a very low vibrating tone. The sound was varying in volume with a rhythmic oscillation.

Visually

Mr T. reported that these:

> sounds were accompanied by a visual cacophony of images, involving an array of squares and rectangles of varying sizes with a slight yellowing background. This absence of colour with a slight yellowing background remained the entire time. The strong geometric shapes appeared to be collapsing mechanically on themselves.

and he was 'aware of a metallic sound accompanying each movement of the shapes as they collapsed'.

The sound of these shapes as they collapsed, made 'a similar sound to a camera shutter as it cycles through multiple shots'. The shapes filled his 'visual field and were all collapsing in somewhat of a sequence, with many more different sequences involved' simultaneously.

Sounds and visual shapes

Mr T. was 'mesmerized at first by the myriad of shapes and their movements', though as he:

> began to focus upon an individual shape, a loud banging noise arose. This noise seemed to fluctuate in volume and cadence of the banging. Sometimes to the point of being uncomfortably loud and appeared to randomly appear from side to side.

While these sounds and visual shapes were happening Mr T. had 'absolutely no awareness of his physical body, nor did he have any usual sensory input'. He could 'not see his body and had a very emotionally neutral sensation'. He 'was not scared, just fascinated with the images and sounds shifting around' him.

He reported that he 'began to focus on the banging sound', and it appeared as if he was 'being accelerated through the shapes, not unlike what imagining a wormhole would be'. 'When that stopped, the shapes would assemble themselves into a structure from the base up. The whole while making a photocopier sound as they assembled or collapsed back down'. He described no sooner was he 'trying to focus on the created structure, it would collapse back down' and he would be 'accelerated again to observe another structure assembling'.

As Mr T. continued:

> the banging sound appeared to come in bursts, some loud and others quiet. Some of the sounds appeared in a noticeable cadence, others appeared to have no cadence at all. The low whirring mechanical sound was still present, though appeared to be outdone by the intermittent banging.

He described that it:

> became apparent that there were two visual structures that were at the centre of the field, seemed to be not moving around or completely collapsing away as the other shapes were.

He reports that he:

> became fascinated with these two shapes and watched them folding mechanically in on themselves with the persistent camera shutter sound. The shapes remained squares and rectangles of varying sizes and orientations.

The closer he 'focussed on these two objects the louder the banging, whirring and photocopying sounds became', as he 'began to lose awareness of the two shapes around them'.

Predominantly two shapes

Mr T. 'had a sense that these two objects were trying to show' him something, but he:

> couldn't work out what it was. The two shapes began to speed up the rate at which they were transforming their shape, with the sounds becoming more and more intense. They were collapsing in on themselves geometrically, like each section became its own square or rectangle and folded into itself and transformed into a typically smaller or different shape.

This 'continued for some time, with the two shapes slowly moving closer together as they continued to mechanically fold in on themselves'. Mr T. became aware 'that each of the two shapes was exhibiting less and less complicated structures each time they collapsed in on themselves', and the:

> banging, whirring and camera shutter noises were also beginning to fade and were starting to be replaced with a constant low tone. As the shapes continued to implode and come closer to merging, the low tone slowly started to increase in pitch. This process steadily became faster and faster as the shapes began to merge.

Again:

> the initial chaos of movement and sound, seemed to be moving toward a singularity and there was a strong sense that something was going to be revealed. The faster the shapes collapsed and merged, the faster the tone increased in pitch. This culminated with the complete merging of the two objects, where suddenly all movement and sound stopped.

Complete silence within a slight yellowing background

Mr T. then described 'in complete silence, in slight yellowing of the background, was a perfect small square in front' of him, 'perfectly still. It became apparent that all of the proceeding cacophony of sound and movement appeared to have been collated to this one square' and he 'had a sensation as if a cursor was going to appear and type out' '**factory reset complete, press restart'**.

He reported further that he then found himself 'staring at the square waiting for something to happen in the silence, yet nothing was changing'. Mr T. recalled 'thinking at that point' '*have I just died?*' He remembers 'looking around the field of view and seeing nothing'. Again thinking '*is this what death is, is that it?*'. That thought 'was not attached to absolutely any emotion or sadness, just very matter of fact'. He recalled that 'it was a most peculiar sensation' and he 'was unsure what to do or what was going to happen, again with absolutely no fear or emotion, just curiosity'.

Vision of Mr T.'s external world returns

As he began to ponder this, 'a blurry, extremely shuddering image of what looked like a word appeared in front' of him and 'the square was gone'. It was 'still silent' and as he 'became fascinated at what this word was and somewhat frustrated' that he 'couldn't stop the shuddering or double/triple vision to be able to read it'. As he 'struggled to focus', he 'became aware of movement in the field at the peripheries'. Again he 'couldn't focus on what it was, just light and dark movement'.

'Slowly the shuddering of vision began to slow and the word in front' of him 'was just able to be determined through the blur and shudder': '*resus 2*'. He further 'pondered that word for a few moments before it dawned on' him that he 'must be still in the resuscitation area of the hospital'. Though he recalled that he 'couldn't make anything else out and was thinking' '*well if I can read that, I can't be dead yet*'.

The 'dark movement' he described he had 'seen, slowly began taking shape'. 'Strangely, [the shapes] became digital like versions of people. Similar to the figures on the game Minecraft. Their body shapes and features were comprised of squares and rectangles, with their movements being robotic'. The things that Mr T. 'could see had appeared to have been digitized'.

Returning to reality

Mr T. 'began to make out structures within the resus bay' and realised he 'was coming out from where' he 'had been'. He interestingly described that he 'too felt quite robotic'. He remembered feeling like he 'was the terminator robot from the movie, rebooting and the waking back up process felt exactly like this'. He described he felt like his 'brain was rerouting circuits and power supplies to bring the visual cortex back online'. He remembered at the time, 'then testing the function' of his 'left hand whilst looking at it'. As he 'opened and closed' his hand, 'the movement made a robotic/machine like sound' as he moved it. Then he tested the 'right hand' and it did the same. Next for some reason it seemed to make sense to test his 'swallowing reflex' and with each test, he 'became more cognisant' of his 'surroundings'.

He describes that 'like throwing a switch', he could 'hear sound and garbled voices. Peoples' shapes became more rounded and their movements more natural'. He realised he 'was back in the real world and trying to shake off the sedation. Slowly from this point the shuddering vision resolved' and he 'became more aware' of his 'body, surroundings and peoples' voices talking' to him in the ED again.

Mr T's initial interactions and subsequent reflections within emergency department

Mr T. is an extremely science and fact-based person and reflecting on this experience has raised certain questions. Visually, were all the strange shapes he was seeing being generated from his mind? Alternatively, were they like a pixelated view of the information still being received by his eyes from the lights in the resus room? Mr T. confirmed with the staff that his eyes were open the entire time and his pupils were only slightly reactive. Likewise, were the **sounds** of the banging, whirring and shutter noises digital/binary versions of the auditory data coming from his ears, or was he hearing actual peoples' voices and/or equipment sounds coming from the room? Mr T. describes that it seemed as if what he was experiencing was the digital, binary, and electronic messages that were being passed by his sensory organs to his brain for processing, but without the capacity to be able to do the processing part. What Mr T. reflected on is that perhaps he was experiencing the raw data from the sensory organs before processing and formatting by the brain. He further speculated that similarly, the way in which his eyes and hearing didn't recognise the people around him, but just received raw visual and auditory information to the brain – which could not process the information or compare it with memories and other confirming information – meant that he could not then recognise what he was seeing because of sensory processing interference during the 20-min ketamine experience.

On reflection, Mr T. thinks the initial cacophonous nature of the sounds and shapes that slowly, but logically, reduced to a single stable square, did have a strong sense of bringing order to overwhelming visual and auditory chaos. He also reported that, interestingly, when the chaos finally arrived at the 'single perfectly shaped square', this was accompanied by a sense of comfort, wellness and calm. As he stated, when

he questioned had he '*just died*' there was no fear, remorse or sadness, just calm and quiet. Again, there was a sense that he had been '*reset to factory settings*' and '*all corrupting files had been removed*'.

Mr T's firsthand descriptions of feeling like he had experienced 'a factory reset' illustrated several aspects of how his brain functioning was potentially affected during his rapid AF/ketamine cardioversion incident. During the ketamine anaesthesia state, he reports he felt no sense of physical awareness of his body, apprehension, fear or endangerment as he self-generated visual geometric shapes that, when collapsing, sounded like a camera shutter while he heard rhythmic blocks of pulsing tones coming and going. The question of what he experienced and how the brain had been affected have been already highlighted by ketamine studies trying to shift long-term treatment resistant major depression (Lally et al., 2015).

Researchers into treatment resistant depression describe how ketamine can be combined with therapeutic intervention owing to it facilitating changes in the brain's neurochemistry, with naturally occurring chemicals in the brain being rebalanced, neuroplasticity increased, and changes in thought patterns influenced through greater insights into the client's world view (Abdallah et al., 2016). Ketamine has been shown to affect two chemical messengers called glutamate and gamma-aminobutyric acid (GABA). These two neurotransmitters almost have opposing effects on the brain's function. Glutamate is one of the brain's most prominent excitatory neurotransmitters, while GABA has more of an inhibitory role. This combination has been shown to decrease suicidality while improving a person's reported mood state (Stone et al., 2012).

Specifically focussing on glutamate's excitatory function, it can be summarised that when a glutamate receptor is activated, it increases the possibility that the host neuron will fire. To further illustrate with one of many types of glutamate receptors, is the NMDA(N-methyl-D-aspartate) receptor. The NMDA receptor operates as a tiny gate on the neuron's surface that can open and close when glutamate binds to it. This binding and then opening of the gate allow ions to rush in, increasing the probability it will fire (Yao et al., 2018). Ketamine functionally blocks the gate, which prevents signals from passing through the gate. This in turn increases glutamate levels in the brain, which have been correlated with the dissociative states produced in people using ketamine (Hashimoto et al., 2007).

Researchers have also found when these glutamate bursts across the brain occur, this facilitates neuronal growth, re-establishing and strengthening neural connections via dendrites. Dendrites are microscopic spine 'like' structures that send and receive information. When a person is chronically stressed or depressed, these dendrite structures die. Studies have shown that ketamine facilitates the growth of dendrites in mice (Hascup et al., 2010).

Mr T. described what researchers have found (Niesters et al., 2012) in resting state functional magnetic resonance imaging (rsfMRI). Their research discovered that ketamine decreased connectivity in the auditory and somatosensory networks with regions of physical and affective processing associated with pain, such as the amygdala, insula and anterior cingulate cortex (ACC). Additional research has shown ketamine-induced hyperconnectivity in hippocampal networks vulnerable to mood and cognitive disorders (Bonhomme et al., 2016). These researchers studied how ketamine effected brain connectivity with increased levels of sedation correlating significantly with decreased connectivity in the medial prefrontal cortex (mPFC) and connectivity with the default mode network (DMN), as well as between the left and right executive control networks. Another study by Grimm et al. (2015), observed that an acute ketamine challenge created hyperconnectivity between the PFC and the left hippocampus.

Mr T. 2 days after cardioversion for a spontaneous episode of rapid atrial fibrillation and 80 mg of intravenous ketamine incident

It took Mr T. several hours for the full effects of the ketamine to subside. As they did, he 'became increasingly aware of a sense of calm outside the experience'. At first, he 'surmised this must be the residual effects of the ketamine' in his system. Though remarkably with clinical psychological review and self-report assessments after 3 days post the procedure (05 November 2021), the 'sense of calm remained', which was difficult for him to describe. He stated that it had been 'a long time since' he had 'felt this level of calmness' and it made him 'acutely aware of how rampant' he 'had been in thought, feeling and emotion over the past twelve months' with his developed chronic PTSD (DSM 5, 2013).

Mr T. reported he 'noticed a strong sense of quiet in my mind'. Typically, he 'would have a kaleidoscope of thought rampaging through' his 'head 24 hrs a day'. Now his 'thoughts seem to be very ordered and purposeful'. He now had 'a general sense of feeling emotionally stable, which again has been insightful to how emotionally unstable' he had been, something he had 'been resistant to accept'. He 'noticed when doing a range of tasks' his 'mind seemed more capable of staying on track without being distracted by erroneous thoughts'. He had 'tried thinking about thoughts which were previously extremely disturbing', the type he:

> would typically forcefully repress. Interestingly, they seem hard to force into conscious thought. When they do appear, there seems to be much less emotionally attached to them or they don't draw out the same emotional response as before.

He had noted though, 'that although the level of emotion attached to these thoughts was greatly reduced, these thoughts did seem to still trigger the sweating response' he was having previously. In saying that, he 'would rather sweat than feel the emotional avalanche of these thoughts any day'.

After several days post his rapid AF cardioversion and ketamine episode (02 November 2021), it is unclear whether Mr T. has experienced a permanent or temporary state. His clinical presentation and self-reports showed significant reductions in anxiety, depression, avoidance and PTSD symptoms. Mr T. at this time continued to have fortnightly treatment sessions and remained on full-time (non-operational 'off road') ambulance duties within his government position. Mr T. reported he was:

> extremely happy if it was permanent, but as for now, I am just going to enjoy the calm. I had forgotten that it was possible to feel this level of calmness and quietness of thought.

Day 5 post rapid atrial fibrillation cardioversion and ketamine event

After 5 days, Mr T. reported that he was amazed as the 'sensation of a quiet mind' continued and it was 'difficult to describe what has been happening' as there's a sense that if he 'talks about it' or tries to 'push too much' he 'might accidently undo what's going on'. He described he found that:

> each day is dominated by a lack of thoughts going in my mind. *It feels like for the last 12 months there has been constant loud music playing while I'm trying to go about life and suddenly, it's been switched off and I can hear and perceive the world in a much clearer way. At times I find the absence of thoughts going through my head concerning, as it is just so different to what I have been experiencing*.

Reporting on his reflections on day 5, Mr T. found himself 'purposely forcing' himself 'to think about different things just to make sure' he could 'still analyse things in my mind'. The 'absence of spontaneous thoughts' was making him 'question whether' he had 'accidently been chemically lobotomised'. He tested his thinking capacity on maths and physics and was pleased to still be able to process and philosophise about different concepts. Interestingly, when he was testing himself, he 'clearly noticed a sharper ability to focus on the task at hand without distraction', something he had found incredibly difficult since developing his chronic PTSD condition over the past 12 months.

Mr T. also reported that, coinciding with the absence of random and incessant thoughts, was a complete lack of scenario generating. A previous chronic PTSD management symptom described by him, apart from 'thousands of thoughts,' was that he 'would constantly solve scenarios' in his head. As an example, he 'would perhaps see someone crossing a road and automatically generate the scenario of them being struck by a car, what he would do and how he would prioritise the situation' as an ICP ambulance officer. Previously, as part of his chronic PTSD condition, Mr T. would constantly generate scenarios, almost entering a daydream like state, and would become lost in working the scenario through his mind, slightly detached from his immediate surroundings. Scenarios could be elicited by almost anything, even generated by something like what he would do if 'the phone rang, and someone had just died'.

After 5 days, Mr T. was fascinated that his scenario generating hypervigilance and increased physical arousal 'had completely stopped' and it was 'very strange to suddenly become aware that this wasn't occurring' anymore. Overall, Mr T. reported that he now experienced an 'absence of these and random thoughts', which is why he now observes that his 'mind is extremely quiet'.

Further, Mr T. reported noticing that he was no longer 'irritated as easily'. He had been previously spending a significant amount of energy 'controlling the constant avalanche of thoughts happening 24/7' and anything extra that was asked of him 'automatically was irritating' and he consistently revealed he 'would have to fight hard to not show this, with limited success' to his family. He reported that all of his family has commented on him appearing 'less distracted' more in the moment and more responsive'.

Mr T. described that he had started to test the waters and spoken about a few things with his family, things that pre-2nd November 2021 would have 'resulted in a tsunami of emotions'. He was now able to 'speak about these things in surprising detail without any overwhelming emotion'. He described that it wasn't that the emotion was absent, but it was what he would call 'an appropriate emotion to the situation'. Encouraged from treatment counselling sessions, Mr T. was able to describe a traumatic job to his adult daughter, for which he would have endeavoured to shelter the family from pre-cardioversion incident. Previously, Mr T. would have typically experienced overwhelming emotions just thinking about this tragic MVA, but now he was able to talk it through with her and explain the anguish he felt about the traumatic incident. The most surprising aspect was that when he previously thought about this job there were feelings of fear, guilt, horror and emotional turmoil. Yet now, at the end of his description with his daughter, the only thought he had was 'what a horrible position to be put in' as a paramedic:

> I actually felt sorry for myself for having to be there and experience what happened. That is a completely foreign thought! Previously it was just what I should have or could have done that I didn't!.

It should be noted, however, that although the overwhelming emotions for Mr T. appear to have been subdued, the sweating he had experienced previously remained. He described it as 'not as severe, but still occurring with the same triggers'. So the traumatic job description he gave to his daughter where he would normally have become emotionally distressed 'still elicited the sweating response', predominately in the armpits, even though he was 'substantially more emotionally calm'.

Further research aligned to Mr T's experience at the University of British Columbia (Walsh et al., 2022) found that ketamine may reset the system by counteracting synaptic deficits, neural atrophy and loss of connectivity with depression. Ketamine, according to Raichle et al. (2001), disrupts the default mode network (DMN) in which we can develop neurobiological 'ruts', over-analysis and scenario

speculation. These are common maladaptive ways of thinking and behaving with chronic long-term psychopathological conditions. Ketamine seems to improve the inherent natural ability of the brain to heal itself by getting a different perspective on other paths and possible growth opportunities. This is where ketamine-assisted psychotherapy (KAP) enables the client to build more positive and prospective adaptive growth orientated pathways and goals which break clients out of treatment-resistant psychopathology conditions such as depression or chronic PTSD.

Mr T. receiving a one-off intravenous 80 mg ketamine dose for his 84 kg frame highlights some differences from the typical KAP model's dose levels and reports substantially variable effects (Auer et al., 2000; Lener et al., 2017). The lower doses recommended for sessions involve more active therapist–client communication, whereas higher-dose intramuscular administrations adhere more closely to psychedelic psychotherapy with more inward focus, eye covering and music dynamics (Stone et al., 2012). Mr T., on the other hand, with a one-off 80 mg dose he simply remained in his resus bed with no interactions in the ED recovery room as supportive fellow paramedic friends stood by initially during the cardioversion as he continued in his open-eyed ketamine-induced state. He could not recall or was not aware of how long his fellow paramedics stayed in support as he remained in his own world.

In early studies of the ketamine dose-response relationship for treating addiction, higher doses of 2.0–3.0 mg/kg were used through intramuscular injection (Chen et al., 2009; Stahl, 2013). More recent research generally uses 0.4 mg/kg–0.5 mg/kg administered intravenously, infused over 40–60 min (Murrough et al., 2015). The reported effects on depression have generally lasted from several days to 2 weeks, which has prompted repeated dosing to extend therapeutic recovery outcomes. The approved FDA dosing model for ketamine has an induction phase of twice weekly dosing levels, which tapers to a maintenance phase of weekly and then a fortnightly dosing, with no maximum period recommended for treatment (Abdallah et al., 2016). Mr T. reported rapid and long-lasting permanent changes in his emotional state and cognitive functioning levels immediately following his 20-min, 80 mg ketamine induction and cardioversion event. Overall, there is no clear empirical research to ascertain an optimal model of ketamine administration during KAP.

One month post incident

Mr T. described that, after more than 4 weeks since the incident:

> the calmness of thought in my mind continues. I have noticed that when confronted by something that prior to the incident would have promoted an avalanche of thought, does now seem to cause a momentary consideration of the topic, rather than no consideration at all. In saying this, the "**thought"** that enters my head might remind me of a job or a situation or feeling, but it is extremely brief. The thought overwhelmingly comes with a sense of forgiveness, and I guess some self-pity that I experienced that situation, which I still find a peculiar way to feel about it.

Mr T. reported that he had 'noticed that because of the absence of random thoughts', what he calls 'noise', he has been 'able to concentrate very attentively to things' that he 'chooses to think about'. This includes him being able to contemplate 'ideas, but also being more attentive, focussed on conversations and listening'. His family and people closer to him have also commented that he appears '*more attentive and in the moment, less vague and distracted and interestingly, that's exactly how I feel*'. He describes that the 'raging thoughts have stopped' and further explains the shift as though 'it's almost like I was a thought schizophrenic and now that the "voices/noise" have stopped, I can fully focus on the moment'. Even as Mr T. reflected on his changes to write his first-hand account of his emotional shifts, 'no other thoughts are entering my head other than what I'm doing right now. When I'm contemplating ideas, it somewhat surprises me just how deep into them I can go without being distracted, something I'm enjoying being able to do again' since being diagnosed with chronic PTSD in late 2020.

Mr T. had continued to be exposed to highly activating and triggering situations at his full-time work through ICP non-operational ambulance interviews, where candidates were raising some intense traumatic scenarios as part of the government selection process. He was surprised that, rather than becoming lost in his 'own world when listening to a variety of these traumatic scenarios', he found himself 'more attentive to the candidate who had to endure the scenario' and having a greater focus on ensuring they are coping after their experience. He recalled this is typically how he previously reacted prior to experiencing the development of PTSD symptoms, which 'felt more inward'. He reported that if he 'would have listened to these scenarios 12 months ago', he 'would have been completely overcome with emotion, probably burst into tears and would not have been able to continue'.

One month post AF cardioversion, Mr T. also reports he has a 'newfound sense of intrigue in the world around' him. He describes how he finds himself examining things 'intently and admiring their form and structure. Further to this is a strange awareness of shape'. He has become 'very aware of something's shape', predominantly meaning 'geometrical type shapes'. He was 'aware of shapes in general, but more specifically aware of their geometry'. Mr T. spoke to his son, a physics student, about this and he was obviously fascinated. Mr T. proposed that as his 'experience on ketamine was very geometric,' his mind now has 'a new reference point to observe the world, along with all my previous memories'. Overall, Mr T. is not sure of the overall significance of this observation, but the reference point is certainly there.

Mr T. reports he only twice experienced noticing becoming emotionally '*choked up*' while explaining something that involved an issue he felt strongly about. However, both these times it was very brief, and he was able to continue the

conversation without long pauses to regather himself, as had been the case before the rapid AF/ketamine incident. Sweating had been more intermittent, with days of almost a complete absence. On other days, some of which had obvious triggers while others did not, he experienced underarm sweating episodes. Overall, Mr T. described his underarm sweating having reduced to approximately *'60% of what it was'* prior to the AF/ketamine incident.

Mr T. further reported a total absence since hospitalisation of what he described as 'triggered and self-generating scenarios being formulated'. Prior to this experience, he reported regularly spending a lot of his day lost in generating these scenarios. These at times were 'all-consuming and could be either triggered by something' with *in vivo* traumatic exposure or could 'just randomly occur without obvious cause'. He previously reported he would use a lot of energy *'spent going from scenario to scenario in my head'*. He would be 'extremely aloof or vague', lost in his own mind. This had completely stopped since Mr T. had been hospitalised and accounted for a significant portion of what he described as '**thought noise**'. For example, Mr T. described receiving a random text message from a friend he had not seen in over 18 months. Strangely, about an hour after receiving the text, he found himself plunging into a scenario/reflection of a significant paramedic job he had done with this co-worker within the ambulance service. He reported it lasted approximately 5 min until he 'realised that he was being fully focussed on the previous emergency ICP scenario'. Apart from feeling disappointed that after 1 month he reactivated the scenario over-analysis again, Mr T did notice that the scenario was not accompanied by 'any significant emotion or sweating'. He reported, 'it felt more like a focussed clinical review of the job,' again with a sense of feeling removed and compassionately feeling '*wow what a difficult position to be in and have to make those decisions*'. He described finding it 'fascinating' that the phone text triggered a scenario generating response with a 'symptom dampened' emotionally connected response.

Although, ketamine infusions have primarily been administrated over extended time frames, participants report experiencing emotional blunting, which could be associated with reduced limbic responses to emotional stimuli (Abel et al., 2003) while activating the reward processing areas connected to reward neurocircuitry (Zhang et al., 2013).

While Mr T. reported feeling 'almost lobotomised of thought and feeling at day 5', there had been a return of some symptoms he had prior to the incident 1 month post rapid AF/ketamine incident. However, all his PTSD, anxiety and depressive symptoms were reduced by what he describes as 'about 90-95%' and, if experienced, the remaining symptoms were 'now extremely short lived'. In conclusion, Mr T. now reflects on more of a '*philosophical approach that presents when these symptoms do occur*' which helps to '*naturally pass them by without great emotional distress and effect*'.

As McFarlane (1994) and Van de Kolk (1994) proposed, the importance of developing valid and reliable measures of how PTSD is studied needs to take into account the degree of generalisation from one culture and victim population to another. They further acknowledge there are limitless adaptations that can occur from trauma, ranging from resilience to the detrimental effects of developing highly debilitating psychopathology. As described by Mr T's clinical case descriptions coupled with the use of standardised research instruments, specific isolative treatment methods without attention to careful clinical observations during treatment can prematurely close us off from the discovery of new information or paradigm shifts in treating multifaceted complex psychopathology such as PTSD.

The following self-report psychometric results open the possibility of how PTSD treated with multifaceted combined exposure therapy dynamics using SIFT, narrative descriptions, PET (Foa et al., 2007) and EMDR (Shapiro, 1989, 2010, 2012) within the parameters of descriptive neuroscience may potentially unlock a remarkable reduction in PTSD symptoms. This approach, coupled with a unique one-off rapid AF/ketamine medical emergency, has produced a remarkable shift in Mr T's PTSD, anxiety, depression, and capacity to function in the long term at a more self-reliant adaptive level for over 18 months.

Results

As depicted in Figure 3, there remained a steady non-significant fluctuation in levels of stress, anxiety and depression within the DASS (Lovibond & Lovibond, 1995a) scores once Mr T. could moderate his PTSD, co-morbid anxiety, and depressive symptom levels within his RTW full-time hours prior to the rapid AF/ketamine incident on 02 November 2021.

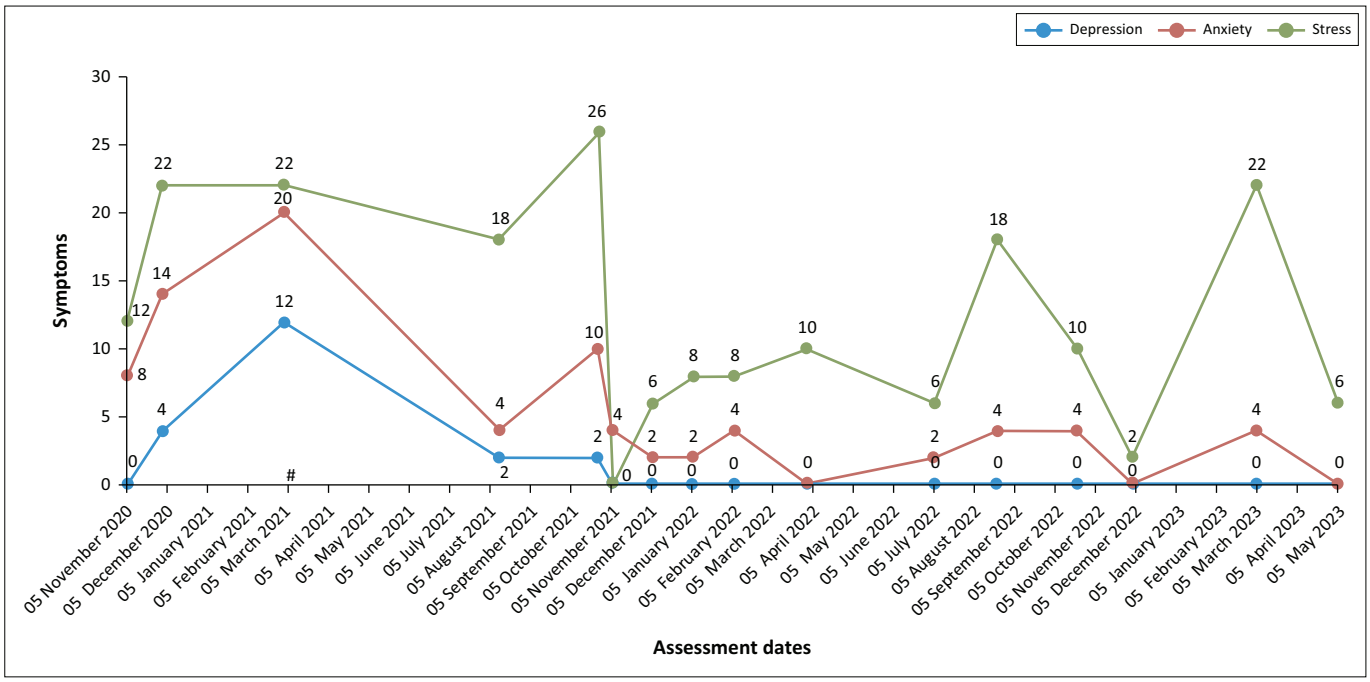
Mr T. was clinically observed and demonstrated significant shifts in his symptom levels on an extended 90-min clinical therapeutic session on 04 November 2021. These changes were further confirmed with a DASS assessment on 05 November 2021 and ongoing subsequent assessments with further requests for treatment support in the following 18 months.

Figure 3 shows an illustrative shift with an initial reduction in stress symptoms levels following the 02 November 2021 rapid AF/ketamine cardioversion incident. These stress symptom levels, which returned owing to apprehension about Mr T's long-term career, have been questioned since August 2022, where the prospect of not being able to RTW at full-time operational 'on road' emergency ambulance levels remains foreseeably questionable.

Figure 4 shows another rapid illustrative reduction in PTSD [PCL-5] symptom levels, particularly following the unexpected rapid AF/ketamine cardioversion incident on 02 November 2021. It is shown that on 05 November 2021,

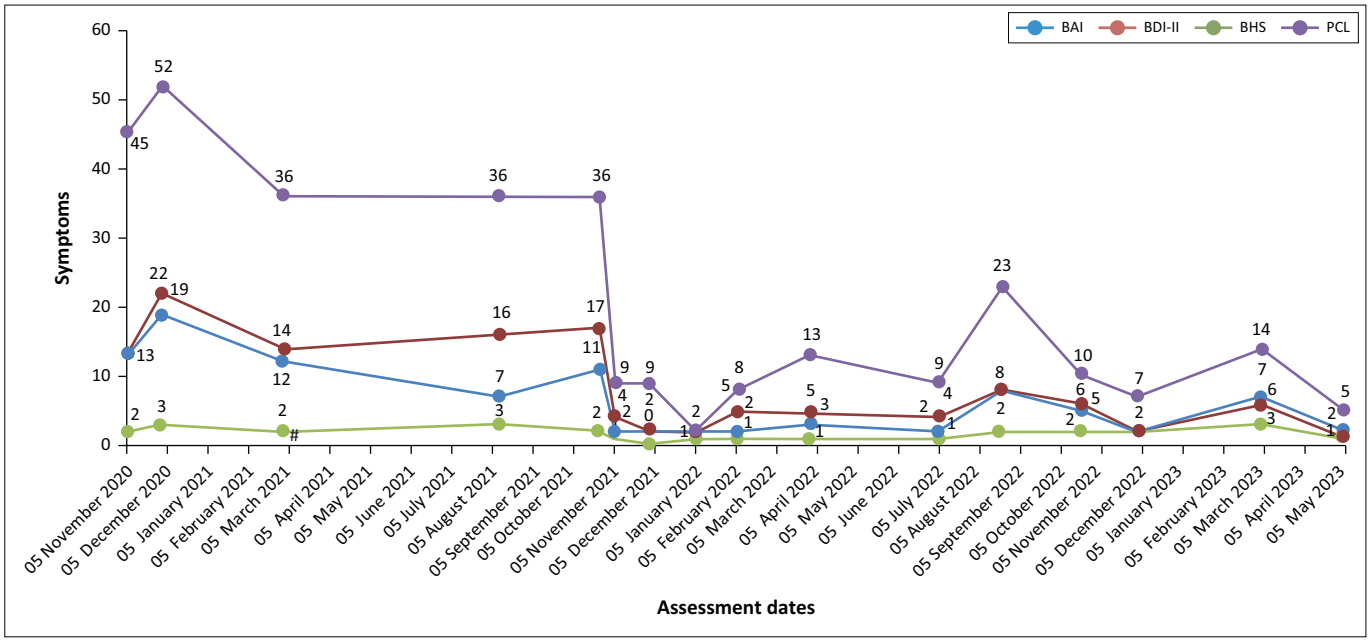
Mr T had three raw scores of a consistent 36 PCL level during full-time, non-operation, suitable RTW duties for over 8 months [mean score of 41; SD range of 7.28011] to shifting in 2 days to an immediate reduction of a raw score of 9 PCL level. This rapid reduction was on average replicated at reduced levels for the next 9 months until July 2022. Mr T. continued to achieve similar PCL-5 scores with ongoing monitoring and SIFT/PET protocols on average (mean score of 9.91; SD range of 4.94975) from 05 November 2021 until 06 May 2023. The reduction in Mr T's PCL-5 mean score of 41 (pre-rapid AF/ketamine cardioversion) to 9.91 (post-rapid AF/ketamine cardioversion) within 2 days, was a 75.85% reduction, which continued for the following 18 months without any ongoing psychiatric medication support.

However, through Chi-Square Testing, Table 1 shows Mr T's overall PCL-5 scores have not been assessed as significant (Ref., 4; Sig. 0.931), although the initial scores of his PCL-5 (PCL1 – pre–rapid AF/ketamine cardioversion) did show significant (Ref., 9; Sig., 0.040) distribution shifts, with a small sample size. Table 1 also shows with Chi-Square Testing that



#, F/T (Non operational).

FIGURE 3: Depression, Anxiety & Stress Scale (DASS) Scores from 05 Nov 2020 to 06 May 2023.



#, F/T (Non operational).

BAI, Beck anxiety inventory; BDI-II, Beck depression inventory-II; BHS, Beck hopelessness scale; PCL, PTSD checklist.

FIGURE 4: Anxiety (BAI), Depression (BDI-II), Hopeless/Avoidance (BHS), PTSD (PCL-5) scores from 05 Nov 2020 to 06 May 2023.

TABLE 1: Null Hypothesis significance using One-sample Chi-Square & Kolmogorov-Smirnov.

Hypothesis Test Summary				
No	**Null Hypothesis**	**Test**	**Sig***	**Decision**
1	The categories of BAI occur with equal probabilities.	One-Sample Chi-Square Test	.023	Reject the null hypothesis.
2	The categories of BDI occur with equal probabilities.	One-Sample Chi-Square Test	.815	Retain the null hypothesis.
3	The categories of BHS occur with equal probabilities.	One-Sample Chi-Square Test	.165	Retain the null hypothesis.
4	The categories of PCL occur with equal probabilities.	One-Sample Chi-Square Test	.931	Retain the null hypothesis.
5	The categories of Depression occur with equal probabilities.	One-Sample Chi-Square Test	<.001	Reject the null hypothesis.
6	The categories of Anxiety occur with equal probabilities.	One-Sample Chi-Square Test	.154	Retain the null hypothesis.
7	The categories of Stress occur with equal probabilities.	One-Sample Chi-Square Test	.926	Retain the null hypothesis.
8	The categories of PCL2 occur with equal probabilities	One-Sample Chi-Square Test	.940	Retain the null hypothesis.
9	The distribution of PCL1 is normal with mean 41 .00 and standard deviation 7.28011.	One-Sample Kolmogorov-Smirnov Test	040**	Reject the null hypothesis

Source: IBM Corp. (2020). *IBM SPSS Statistics for Windows (version 27) Computer software*. Pub. IBM Corp

*, The significance level is .050.

**, Lilliefors corrected. Asymptotic significance is displayed.

Mr T. did have significantly assessed shifts with his anxiety levels (BAI) (Ref., 1; Sig., 0.023) and depression (within the DASS) (Ref., 5; Sig., 0.001) against the significance level of 0.050.

In summary, Mr T. has shown illustrative mean reductions in his PCL-5 of 75.85% and sensitivity to fluctuations in his stress (within the DASS) scores when facing uncertainty with his ambulance ICP career prospects in the future. Mr T. with Chi-Square Testing has been assessed as significant in distribution and against equal probabilities with his PCL-5 (pre-rapid AF/ketamine cardioversion), depression (within the DASS) and anxiety (BAI) scores over the course of his treatment from 05 November 2020 to 06 May 2023.

Discussion

The firsthand descriptions and self-report score results pose several discussion points about what could have happened to Mr T. adapting to his diagnosed chronic PTSD condition over 2 ½ years of psychological and psychiatric monitoring treatment (in the absence of any psychiatric medication). Initially stabilising his diagnosed chronic PTSD condition to more predictable levels enabled him to RTW for non-operational full-time hours involving *in vivo* traumatic information and being exposed to clinical and operational administrative ambulance project roles for approximately 8 months before his rapid AF/ketamine cardioversion incident on 02 November 2021.

It is now apparent from the results that the initial upgrading of Mr T. with exposure therapy, SIFT mapping descriptions (Wilson, 2018, 2019), narrative analysis, PET (Foa et al., 2007) and EMDR (Shapiro, 1989, 2010, 2012) significantly reduced his PTSD symptom levels. This was further confirmed with his depression sub-scale scores (within the DASS) and his anxiety (BAI) (Beck et al., 1988) scores showing significant reductions over his entire treatment intervention from 05 November 2020 through to 06 May 2023.

As shown in Figure 3, Mr T. continues to conceptually find it difficult to further upgrade to full operational 'on road' ICP ambulance duties owing to the inherent time pressurised fluctuating consistency on relayed clinical information, community emotional volatility during crisis, and available resources shifting significantly during emergency call-outs. Further research may find that the stress category of the DASS can indicate high sensitivity to shifting apprehension over job security. Mr T. has shown that the risk of losing his long-term ambulance career and the ongoing reminders of his limits in the past due to his chronic PTSD condition, has elevated his stress category scores. This primarily due him still not being able to fully upgrade to 'on-road' duties in the past 18 months without a significant perception he will risk PTSD relapse. The ongoing governmental case discussions within workers compensation protocols can be shown to have illustratively shifted and fluctuated Mr T's stress (within DASS) scores, as his ICP career capability is ongoingly questioned. However, while his current capacity to have operational administrative ambulance officer roles where he is repeatedly exposed to 'arm's length' emergency ambulance call-out reviews remains exceptional, he cannot RTW to pre-injury capacity without heightened apprehension.

Ketamine Neuroimaging research

Magnetic resonance spectroscopy reviews (Kraguljac et al., 2016) have also shown ketamine's role in stimulating brain connectivity and additional hippocampal function. Overall, ketamine increased hippocampal Glx (glutamate + glutamine), which can indicate enhanced excitatory neurotransmission, while decreasing fronto-temporal and temporo-parietal functional connectivity. This research may suggest that NMDA receptor hypofunction may elevate hippocampal glutamatergic transmission and alter one's resting-state network. Comparing functional magnetic resonance spectroscopy (fMRI) images during a ketamine and placebo infusion (Hoflich et al., 2016) found blood oxygen level dependent (BOLD) activation increases using ketamine in the bilateral middle cingulate cortex, anterior cingulate cortex (ACC), insula and right thalamus.

Researchers (Nagels et al., 2011) have found that ketamine induces a general impairment of verbal fluency. It activated several brain regions (left temporal gyrus, superior frontal gyrus to middle frontal gyrus, medial frontal gyrus and left inferior parietal lobe) during a phonic verbal fluency task. The right frontal and left supramarginal regions were activated significantly more during a lexical fluency task.

Primarily through neuroimaging literature, ketamine has been shown to modulate brain regions associated with major depressive disorder (MDD) and with a more specific emphasis on the subgenual anterior cingulate cortex (sgACC) region. Ketamine associated with emotional and cognitive functioning appears to reduce brain activation in regions connected to self-monitoring, while increasing activation in neural regions linked to emotional blunting and reward processing. Immediate blood flow reductions in the sgACC and focal reductions in the orbitofrontal cortex (OFC) have strongly predicted dissociation (Deakin et al., 2008; Kraguljac et al., 2016). Overall, ketamine has shown that its most prominent effects are found in the sgACC, PCC, PFC and hippocampus (Phillips et al., 2015; Price & Drevets, 2012).

In the main, ketamine neuroimaging research shows that ketamine affects different areas of the brain in various ways, which contributes to a global mood improvement in research participants. At a base level of understanding Abdallah et al. (2016) showed ketamine responders having increased connectivity in the lateral PFC, caudate and insula, while Nugent et al. (2016) found decreased connectivity between the amygdala and insulo-temporal regions. At a base level of understanding, it appears that Mr T. may have reclaimed his ability to exert frontal control of his deeper limbic structures and have greater cognitive control over his emotions. This has decreased his hypervigilant traumatic ruminations and generating 'worst scenario' over-analysis associated with his PTSD condition.

Research is hard to find on the combination of an accelerated AF cardioversion episode and ketamine (80 mg) injection prior to a 100, then 200 joules to restart Mr T's heart with expected normal rhythms within 2–3 min before he experienced a hallucinogenic 'factory reset'. Ketamine infusion studies on the parts of the brain that are activated or reduced are consistent with Mr T's shifting ketamine experience. The question remains: how have his significant reductions in reported PTSD, depression and anxiety symptomology, from a 20-min intervention been monitored and maintained at normal population levels for over the past 18 months without any psychiatric medication throughout his treatment history pre- and post-AF cardioversion?

Conclusion

The developed understanding of down-regulating practices (Toussaint et al., 2021; Vickers & Zollman, 1999), the SIFT traumatic processing mapping model (Wilson, 2014, 2019) and creating a narrative script about the developing accumulation of ambulance traumas enabled Mr T. to actively challenge his 26 years of traumatic history serving the community in crisis. The platform of being able to therapeutically discuss and relate within 60-to-90-min sessions coupled with PET (Foa et al., 2007) and EMDR (Shapiro, 2010, 2012) protocols enabled him to actively challenge his traumatic ICP ambulance history. This enabled him to navigate his PTSD with predictable understanding of how it would be activated during *in vivo* exposure triggering environmental stimuli and allowed him to gain greater insight into what he once believed in as a motivated care giver to community patients suffering medical crises.

Mr T. reported that during his hallucinogenic 'factory reset' experience he would not have been prepared to calmly 'be curious' and 'inquisitive' as he experienced a perception of a 'stillness of death' if he did not have his thorough understanding of how traumatic experiences impact one's established belief structures or potentially shatter one's world view. Although Mr T. openly admits that he was not triggered being rushed to hospital within an ambulance, but expressed he was more like annoyed and reacted almost with a 'mild tantrum' towards the rapid AF episode. However, typically, you would expect him to be rushed to hospital in an ambulance while he has not been able to operationally RTW 'on road', would have relapsed his PTSD condition. The AF and ambulance journey are essentially massively traumatic and life-threatening for anyone, regardless of having been diagnosed with a long-term mental illness such as Mr T's chronic PTSD condition. This is why this case study is seemingly unique because Mr T. reports being prepared to almost challenge and be inquisitive about how his traumatic history could be activated by his life-threatening AF/cardioversion incident. Furthermore, as an ICP, Mr T's extensive knowledge of how a rapid AF episode and ketamine effect patients aided him as an ambulance officer in his adjustment levels. Despite facing a potentially life-threatening AF physical condition, he successfully adjusted to these two elements and the effect in the so-called 'K-hole'. Although Mr T. felt annoyed while he was being rushed to hospital ED, undoubtedly it could be argued that an inexperienced and uneducated patient (non ICP) would have experienced a significant *in vivo* highly triggering 'on road-operational' scenario.

Most observers would think that this experience would have significantly relapsed Mr T's PTSD condition, but instead we have seen the opposite, with substantial reductions in PTSD, anxiety and depression the following day of his rapid AF/ketamine cardioversion incident.

As research shows, ketamine working in KAP settings through ongoing lower doses mainly with treatment resistant major depressive disorder is a repeated and ongoing process of intervention. The question then remains: did Mr T. gain symptom reductions because he experienced an accelerated AF heart rate? Was it the 80 mgs of ketamine to his 84 kg body mass? Or did the application 2–3 min after the ketamine of the initial 100 then 200 joules in the cardioversion promote an opportunity for Mr T. to work with his PTSD, anxiety and depressive symptomology and produce his significant rapid and long-term symptom reductions because of the increased glutamate levels from the ketamine facilitating new dendrite growth (Yao et al., 2018)?

Has Mr T's account shown that a combination of down regulation, hypnosis practices, SIFT mapping, PET *in vivo* exposure, homework tasks, knowledge, and EMDR processing as well as the significant (*in vivo*) traumatic experience of being rushed to hospital in an ambulance while having knowledge of the effects of ketamine as an ICP before his 100 then 200 joules cardioversion procedure could be the final piece of the puzzle as to what shifted his symptom levels with such a remarkable rapid drop? The further remarkable finding is that Mr T's armpit sweating has also reduced to an estimated 60% of what it was before. He still experiences emotional moments and occasional scenario generation, but nowhere near the magnitude and duration he previously had prior to his rapid AF ketamine/cardioversion experience.

The significant reductions in Mr T's PTSD, anxiety and depressive symptomology remain remarkable. Did the previous treatment outlined above of this educated ICP with SIFT, PET and EMDR among others, that had already allowed him to return to work in a less volatile emergency services setting, enable a predictable platform on which to explore his ketamine-induced state without fear? Did the 100, then 200 joules cardioversion 2–3 min into Mr T's ketamine induced state consolidate, fuse or reset maladaptive PTSD brain circuitry to a more permanent, long-term, stabilised emotional 'reset'?

Mr T's diagnosis is recommended to be independently re-assessed by several PTSD assessors. Further statistical exploration of his self-reports is encouraged in the future. However, several questions remain. Could future heart surgery protocols reassess how ketamine could be implemented during recovery periods to allow 'factory reset' and reduce depressive and traumatic memories associated with emergency procedures, thus preventing potential ASD or PTSD development?

Could future rapid AF/cardioversion intervention implement standard ketamine induced states with neuroimaging scans during recovery, to reduce traumatic impact levels?

In conclusion, it is proposed in addition to further research that AF/cardioversion protocols for patients who are diagnosed with major depression and/or PTSD have a one-off ketamine 80 mg (approx. 1 mg/kg according to weight) intravenous injection and receive their 100/200 joules as necessary. Immediately after successful cardioversion they are then monitored with neuroimaging scans as they recover and are interviewed immediately following their experiences to further ascertain any hallucinogenic insights. These protocols could further ascertain understanding of how this remarkable case has significantly shifted an intensive care paramedic's PTSD condition to a predictable long-term stabilised state.

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Competing interests

The author declared that they have no financial or personal relationship(s) that may have inappropriately influenced them in writing this article.

Author's contribution

D.J.W. is the sole author of this research article.

Ethical considerations

An application for full ethical approval was made to the IAAN Ethical clearance committee. The application was reviewed according to the international best practices and ethical standards informed by the Declaration of Helsinki (2008) and the International Ethical Guidelines for Health-related Research Involving Humans (2016). Ethics consent was received on 23 August 2023. The ethics approval number is IAAN/ERC0010.

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Data availability

Access to raw data is restricted in line with the consent provided by the participant of this case study.

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References

Abdallah, C.G., Averill, L.A., Collins, K.A., Geha, P., Schwartz, J., Averill, C., DeWilde, K.E., Wong, E., Anticevic, A., Tang, C.Y., Iosifescu, D.V., Charney, D.S., & Murrough, J.W. (2016). Ketamine treatment and global brain connectivity in major depression. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology, 42*, 1210–1219. https://doi.org/10.1038/npp.2016.186

Abel, K.M., Allin, M.P., Kucharska-Pietura, K., Andrew, C., Williams, S., David, A.S., Philips, M.L. (2003). Ketamine and fMRI BOLD signal: Distinguishing between effects mediated by change in blood flow versus change in cognitive state. *Human Brain Mapping, 18*(2), 135–145. https://doi.org/10.1002/hbm.10064

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Pub.

Auer, D.P., Putz, B., Kraft, E., Lipinski, B., Schill, J., & Holsboer, F. (2000). Reduced glutamate cingulate cortex in depression: An in vivo proton magnetic resonance spectroscopy study. *Biological Psychiatry, 47*(4), 305–313. https://doi.org/10.1016/S0006-3223(99)00159-6

Beck, A.T., Brown, G., & Steer, R. (1989). Prediction of eventual suicide in psychiatric inpatients by clinical ratings of hopelessness. *Journal of Consulting and Clinical Psychology, 57*(2), 309–310. https://doi.org/10.1037/0022-006X.57.2.309

Beck, A.T., Epstein, N., & Brown, G. (1996). *Manual for Beck depression inventory-II.* Psychological Corporation.

Beck, A.T., Epstein, N., Brown, G., & Steer, R. (1998). *Beck anxiety inventory*. APA Psych Tests.

Blake, D.D., Weathers, F.W., Nagy, L.M., Kaloupek, D.G., Gusman, F.D., Charney, D.S., & Kearne, T.M. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress, 8*(1), 75–90. https://doi.org/10.1002/jts.2490080106

Bonhomme, V., Vanhaudenhuyse, A., Demertzi, A., Bruno, M.-A., Jaquet, O., Bahri, M.A., Plenevaux, A., Boly, M., Boveroux, P., Soddu, A., Brichant, J.F., Maquet, P., & Laureys, S. (2016). Resting-state network-specific breakdown of functional connectivity during ketamine alteration of consciousness in volunteers. *Anaesthesiology, 125*, 873–88. https://doi.org/10.1097/ALN.0000000000001275

Bovin, M.J., Marx, B.P., Weathers, F.W., Gallagher, M.W., Rodriguez, P., Schnurr, P.P., & Keane, T.M. (2016). Psychometric properties of the PTSD checklist for diagnostic and statistical manual of mental disorders-fifth edition (PCL-5) in veterans. *Psychological Assessment, 28*(11), 1379–1391. https://doi.org/10.1037/pas0000254

Briere, J., & Scott, C. (2006). *Principles of trauma therapy: A guide to symptoms, evaluation and treatment*. Sage.

Bryant, R.A. (1997). Psychological treatments of post-traumatic stress disorder. *Psychotherapy in Australia, 3*(2), 58–62.

Chen, X., Shu, S., & Bayliss, D.A. (2009). HCN1 channel subunits are a molecular substrate for hypnotic actions of ketamine. *Journal of Neuroscience, 29*, 600–609. https://doi.org/10.1523/JNEUROSCI.3481-08.2009

Deakin, J.F., Lees, J., McKie, S., Hallak, J.E., Williams, S.R., & Dursun, S.M. (2008). Glutamate and the neural basis of the subjective effects of ketamine: A pharmaco-magnetic resonance imaging study. *Archives of General Psychiatry, 65*(2), 154–164. https://doi.org/10.1001/archgenpsychiatry.2007.37

Foa, E.B., Hembree, E.A., & Rothbaum, B.O. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences, therapist guide*. Oxford University Press

Grimm, O., Gass, N., Weber-Fahr, W., Sartorius, A., Schenker, E., Spedding, M., Risterucci, C., Schweiger, J.I., Böhringer, A., Zang, Z., Tost, H., Schwarz, A.J., & Meyer-Lindenberg, A. (2015). Acute ketamine challenge increases resting state prefrontal-hippocampal connectivity in both humans and rats. *Psychopharmacology, 232*, 4231–4241. https://doi.org/10.1007/s00213-015-4022-y

Hascup, E.R., Hascup, K.N., Stephens, M., Pomerleau, F., Huettl, P., Gratton, A., & Gerhardt, G.A. (2010). Rapid microelectrode measurements and the origin and regulation of extracellular glutamate in rat prefrontal cortex. *Journal of Neurochemistry, 115*(6), 1608–1620. https://doi.org/10.1111/j.1471-4159.2010.07066.x

Hashimoto, K., Sawa, A., & Iyo, M. (2007). Increased levels of glutamate in brains from patients with mood disorders. *Biological Psychiatry, 62*(11), 1310–1316. https://doi.org/10.1016/j.biopsych.2007.03.017

Hjelle, L.A., & Ziegler, D.J. (1992). *Personality theories: Basic assumptions, research and applications* (3rd ed.). McGraw Hill.

Hoflich, A., Hahn, A., Kublbock, M., Kranz, G.S., Vanicek, T., Ganger, S., Spies, M., Windischberger, C., Kasper, S., Winkler, D., & Lanzenberger, R. (2016). Ketamine-dependent neuronal activation in healthy volunteers. *Brain Structure Function, 222*, 1533–1542. https://doi.org/10.1007/s00429-016-1291-0

IBM Corp. (2020). *IBM SPSS Statistics for Windows (version 27) Computer software.* Pub. IBM Corp.

Kraguljac, N.V., Frolich, M.A., Tran, S., White, D.M., Nichols, N., Barton-McArdle, A., Reid, M.A., Bolding, M.S., & Lahti, C. (2016). Ketamine modulates hippocampal neurochemistry and functional connectivity: A combined magnetic resonance spectroscopy and resting-state fMRI study in healthy volunteers. *Molecular Psychiatry, 22*, 562–569. https://doi.org/10.1038/mp.2016.122

Lally, N., Nugent, A.C., Luckenbaugh, D.A., Niciu, M.J., Roiser, J.P., & Zarate, C.A. Jnr. (2015). Neural correlates of change in major depressive disorder anhedonia following open-label ketamine. *Journal of Psychopharmacology, 29*(5), 596–607. https://doi.org/10.1177/0269881114568041

Lener, M.S., Niciu, M.J., Ballard, E.D., Park, M., Park, L.T., Nugent, A.C., & Zarate, C.A. Jr. (2017). Glutamate and gamma-aminobutyric acid systems in the pathophysiology of major depression and antidepressant response to ketamine. *Biological Psychiatry, 81*(10), 886–897. https://doi.org/10.1016/j.biopsych.2016.05.005

Lovibond, P.F., & Lovibond, S.H. (1995a). *Manual for the depression anxiety stress scales* (2nd ed.). Psychology Foundation of Australia.

Lovibond, P.F., & Lovibond, S.H. (1995b). The structure of negative emotional states: Comparison of the Depression Anxiety Scales (DASS) with beck depression and anxiety inventories. *Behaviour Research and Therapy, 33*(3), 335–342. https://doi.org/10.1016/0005-7967(94)00075-U

McFarlane, A.S. (1994). Individual psychotherapy for post-traumatic stress disorder. *Psychiatric Clinics of North America, 17*(2), 393–408.

Murrough, J.W., Collins, K.A., Fields, J., Dewilde, K.E., Philips, M.L., Mathew, S.J., Wong, E., Tang, C.Y., Charney, D.S., & Iosifescu, D.V. (2015). Regulation of neural responses to emotion perception by ketamine in individuals with treatment-resistant major depressive disorder. *Translational Psychiatry, 5*, e509. https://doi.org/10.1038/tp.2015.10

Nagels, A., Kirner-Veselinovic, A., Krach, S., & Kircher, T. (2011). Neural correlates of S-ketamine induced psychosis during overt continuous verbal fluency. *Neuroimage, 54*(2), 1307–1314. https://doi.org/10.1016/j.neuroimage.2010.08.021

Niesters, M., Khalili-Mahani, C., Martini, C., Aarts, L., van Gerven, J., van Buchem, M.A., Dahan, A., & Rombouts, S. (2012). Effect of subanesthetic ketamine on intrinsic functional brain connectivity: A placebo-controlled functional magnetic resonance imaging study in healthy male volunteers. *Anaesthesiology, 117*(4), 868–877. https://doi.org/10.1097/ALN.0b013e31826a0db3

Nugent, A.C., Robinson, S.E., Coppola, R., & Zarate, C.A. Jnr. (2016). Preliminary differences in resting state MEG functional connectivity pre- and post-ketamine in major depressive disorder. *Psychiatry Research, 254*, 56–66. https://doi.org/10.1016/j.pscychresns.2016.06.006

Phillips, M.L., Chase, H.W., Sheline, Y.I., Etkin, A., Almeida, J.R.C., Deckersbach, T., & Trivedi, M.H. (2015). Identifying predictors, moderators, and mediators of antidepressant response in major depressive disorder: Neuroimaging approaches. *The American Journal of Psychiatry, 172*(2), 124–138. https://doi.org/10.1176/appi.ajp.2014.14010076

Price, J.L., & Drevets, W.C. (2012). Neural circuits underlying the pathophysiology of mood disorders. *Trends Cognitive Science, 16*(1), 61–71. https://doi.org/10.1016/j.tics.2011.12.011

Raichle, M.E., MacLeod, A.M., Synder, A.Z., Powers, W.J., Gusnard, D.A., & Shulman, G.L. (2001). A default mode of brain function. *Biological Sciences, 98*(2), 676–682. https://doi.org/10.1073/pnas.98.2.676

Rossouw, P.J. (Ed.). (2014). *Neuropsychotherapy: Theoretical underpinnings and clinical applications*. Mediros.

Shapiro, F. (1989). Eye movement desensitization: A new treatment for post-traumatic stress disorder. *Journal of Behaviour Therapy and Experimental Psychiatry, 20*(3), 211–217. https://doi.org/10.1016/0005-7916(89)90025-6

Shapiro, F. (2010). *The trauma treatment handbook: Protocols across the spectrum*. Norton & Company Inc.

Shapiro, F. (2012). *Getting past the past: Take control of your life with self-help techniques from EMDR therapy*. Rodale Inc.

Stahl, S.M. (2013). Mechanism of action of ketamine. *CNS Spectrum, 18*(4), 171–174. https://doi.org/10.1017/S109285291300045X

Stone, J.M., Dietrich, C., Edden, R., Mehta, M.A., De Simoni, S., Reed, L.J, Krystal, J.H., Nutt, D., Barker, G.J. (2012). Ketamine effects on brain GABA and glutamate levels with 1H-MRS: Relationship to ketamine-induced psychopathology. *Molecular Psychiatry, 17*, 664–665. https://doi.org/10.1038/mp.2011.171

Toussaint, L., Nguyen, Q.A., Roettger, C., Dixon, K., Offenbacher, M., Kohls, N., Hirsch, J., & Sirois, F. (2021). Effectiveness of progressive muscle relaxation, deep breathing and guided imagery in promoting psychological and physiological states of relaxation. *Evidence Based Complementary Alternative Medicine, 2021*, 5924040. https://doi.org/10.1155/2021/5924040

Van de Kolk B A. (1994). The body keeps the score: Memory and the evolving psychobiology od post-traumatic stress. *Harvard Review of Psychiatry, 1*(5), 253–265.

Van der Kolk, B.A., & Ducey, C.P. (1989). The psychological processing of traumatic experience: Rorschach patterns in PTSD. *Journal of Traumatic Stress, 2*, 259–274. https://doi.org/10.1002/jts.2490020303

Van der Kolk, B.A., McFarlane, A.C., & Weisaeth, L. (1996). *Traumatic stress*. The Guildford Press.

Vickers, A., & Zollman, C. (1999). Hypnosis and relaxation therapies. *British Medical Journal, 319*, 1346. https://doi.org/10.1136/bmj.319.7221.1346

Walsh, Z., Mollaahmetoglu, M.O., Rootman, J., Golsof, S., Keeler, J., Marsh, B., Nutt, D.J., & Morgan, C.J.A. (2022). Ketamine for the treatment of mental health and substance use disorders: Comprehensive systematic review. *British Journal of Psychiatry Open, 8*, e19. https://doi.org/10.1192/bjo.2021.1061

Wilson, D.J. (2014). A new treatment for PTSD: Ross, a 65-year-old male survivor of the December 26, 2004, Indian Ocean Tsunami. In P.J. Rossouw (Ed.), *Neuropsychotherapy: Theoretical underpinnings and clinical applications* (pp. 291–308). Mediros.

Wilson, D.J. (2018). Structured Image Framework Theory (SIFT): A neurologically based diagrammatic structure enabling the therapist to describe emotionally distressing situations within known brain functions. *International Journal of Neuropsychotherapy, 6*(2), 93–104. https://doi.org/10.12744/ijnpt.2018.0093-0104

Wilson, D.J. (2019). Structured Image Framework Theory (SIFT): A neurologically based technique designed to understand and process the traumatic experience. *International Journal of Neuropsychotherapy, 7*(2), 43–55. https://doi.org/10.12744/Ijnpt.2019.043-055

Yao, N., Skiteva, O., Zhang, X., Svenningsson, P. & Chergui, K. (2018). Ketamine and its metabolite (2R,6R)-hydroxynorketamine induce lasting alterations in glutamatergic synaptic plasticity in the mesolimbic circuit. *Molecular Psychiatry, 23*, 2066–2077. https://doi.org/10.1038/mp.2017.239

Zhang, W.N., Chang, S.H., Guo, L.Y., Zhang, K.L., & Wang, J. (2013). The neural correlates of reward-related processing in major depressive disorder: A meta-analysis of functional magnetic resonance imaging studies. *Journal of affective disorders, 151*, 531–539. https://doi.org/10.1016/j.jad.2013.06.039