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A case of episodic derealisation in focal cortical dysplasia: First looks at the whole elephant?

Dr (Maj) Ashutosh Ratnam, MBBS[a,*], Lt Col Aditya Gupta, DNB[b], Brig R.C. Das, MD, PDC[a], Maj S.P. Panda, MBBS[a]

[a] Department of Psychiatry, Armed Forces Medical College, Sholapur Road Pune, Maharashtra 411040, India
[b] Department of Neurology, Command Hospital (Southern Command), Pune, India

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ABSTRACT

Derealisation remains a bewildering and obscure phenomenological entity with shifting identities on the dissociation spectrum of disorders. A consensus seems to have emerged that the symptom can be produced by a variety of agents which finally act through a common pathway whose identity remains elusive and whose components have thus far only emerged piecemeal. Temporal lobe dysfunction, a covert epileptogenic process and aberrations within a network comprising the amygdala, the anterior cingulate and the prefrontal cortices have all been implicated, however independently none account holistically for the peculiar phenomenology of derealisation. We present a case of focal cortical dysplasia of the right supramarginal gyrus, who reported with three distinct symptoms – panic episodes, a heightening of baseline anxiety and recurrent derealisation, all of which responded well to antiepileptic drugs. This represents the first case with evidence of dysfunction in all areas of the heteromodal association cortex (HASC), a neural network responsible for integration of sensory input and required to understand the significance of events and objects in the external environment. Derangements in the HASC have produced non-modality specific neuropsychiatric deficits, and the component anatomy can account well for the aberrations in emotion, perception and memory characteristic of derealisation. Collectively, the model provides a credible basis with which to explain the curious symptoms encountered in derealisation and provides a promising line of enquiry.

1. Introduction

> "Reality exists in the human mind, and nowhere else."- George Orwell, 1984

Few phenomenological entities remain as intriguing yet as ill-understood as derealisation. Derealisation has now existed in medical literature for over seven decades, yet little about it can be spoken with any true conviction. Foremost the very origins of the term are uncertain – the name has been attributed to Edward Mapother in a paper by Mayer-Gross (Mayer-Gross, 1935), however locating the printed source where Mapother made the actual suggestion has proved impossible (Berrios, 1996). Derealisation made its first (unnamed) appearance in DSM-II, subsumed in the description of depersonalization as a condition "dominated by a feeling of unreality and of estrangement from the self, body or *surroundings*" (DSM-II, 1968). Since then, where to place and even whether to recognize the label of 'derealisation' has become the focus of a vocal and slow-moving debate. Jacobs and Bovasso have presented derealisation as one of five forms of depersonalization (Jacobs & Bovasso, 1992), while Coons has questioned the very existence of derealisation without depersonalization, citing that a more scrupulous evaluation of 150 references to derealisation, depersonalization and depersonalization-derealisation (Goettman, Greaves, & Coons, 1994) showed that none, not even the two that the review initially endorsed (Krizek, 1989; Rosen, 1955), actually contained derealisation without any depersonalization symptoms (Coons, 1992). DSM-5 seems to have accorded derealisation an equivalency of sorts, aligning with ICD-10 nosology to describe an integrated Depersonalization/Derealisation disorder (Association, 2013). Derealisation continues to be described as "Experiences of unreality or detachment with respect to surroundings". Divergence also prevails in attempts to delineate the anatomy responsible – a dedicated network comprising the amygdala, anterior cingulate and the prefrontal cortices has been hypothesized and tested (Devinsky, Morrell, & Vogt, 1995; LeDoux,

* Corresponding author.
E-mail address: ashratnam@yahoo.com (D.(.A. Ratnam).

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1995; Sierra & Berrios, 1998). Simultaneously phenomenological allegory and anatomical evidence implicating the temporal lobe continues to accumulate. We present a case of recurrent derealisation which while highlighting the intricate relationship between temporal lobe pathology, frontal lobe dysfunction and the role of a possible ongoing epileptogenic process, also contributes material with which to conjecture the role of a novel and complete anatomical network.

2. Case report

2.1. Clinical history

A 33yr old infantry soldier presented with a 3 year history of recurrent episodes of derealisation, lasting several seconds to minutes at a time. The first episode happened when he was doing yoga exercises at approximately 6 a.m. Depersonalization has been experienced in yoga meditation but only in the deepest states of transcendence by the most skilled practitioners (Castillo, 1991). Our patient admitted he did not have the expertise to reach the requisite levels of mental involvement and application for this to take place, and was merely performing the bodily poses ('*asanas*') as a form of physical exercise (Ross & Thomas, 2010). Taking a break, he suddenly felt "detached from the world" around him. Everything around him seemed "unreal", and he claimed he "could see and hear everything around me, but it meant nothing to me". The event had happened in the wake of a significant personal upheaval – a month ago, his wife had eloped with his best friend, and he was having great difficulty keeping up a brave face.

The second episode took place some 02 months later, while he was on guard duty in the early afternoon – again everything felt "strange and unreal" and his "mind went completely blank, like a cell-phone that gets 'hanged"'. He felt that he could "see himself from the outside" and when he looked at his own hands they seemed "too large to be his own". He also perceived parasthesias rapidly ascending up his body. Acutely confused, he grabbed onto a nearby pole and cried out. He was taken to the infirmary, and when nothing ominous emerged on evaluation he was given anxiolytics and rejoined duty. However the episodes started becoming more frequent, and by the end of the month he was experiencing them on a near daily basis. The episodes were invariably brief, lasting some 20–90 s, but their phenomenology was consistent throughout.

In addition the patient also began having panic attacks, which he could clearly identify as symptomatically distinct entities – they were "more frightening", "less confusing", less frequent and longer, lasting some 2–3 min. These were accompanied by palpitations and piloerection, and he developed severe anticipatory dread of these panic attacks.

Over time as the derealisation episodes and panic attacks continued, a gross heightening of baseline anxiety levels and overt depressive symptoms set in. Attempts to treat him with incremental doses of Sertraline (50 mg to 200 mg) yielded unsatisfactory results. Though there was second-hand history suggesting possible seizure episodes (06 and 03 months prior to admission), no eye-witness accounts were available and descriptive semiology of the events was not consistent with grand-mal attacks – thus when he reported, the patient had never received anti-epileptics.

2.2. Physical and mental state examination

At the time of his admission, the patient's general examination revealed he had 06 toes, exhibited digital tremors and tachycardia (HR = 98/min). Mental state examination revealed an extremely nervous and fidgety patient who at times openly pleaded for help, fearing he would "go mad or kill himself". Hamilton Anxiety Rating Scale Score at intake was 38, indicating severe anxiety (Hamilton, 1959). He spoke rapidly, describing his 'terrible mood', expressed passive death wishes and exhibited cognitive distortions (catastrophisation and depressive schemas). In spite of his ongoing panic and derealisation attacks, he remained in a clear sensorium throughout. His mean Dissociative Experiences Scale (DES) score was 21 (Steinberg, Rounsaville, & Cicchetti, 1991).

2.3. Electroencephalography and neuroimaging

Long Term Continuous EEG Recordings were taken during numerous derealisation attacks. However no significant EEG changes or epileptiform activity was recorded during these depersonalization states.

On MRI brain, axial 5 mm thick T2 and FLAIR slides showed Focal Cortical Dysplasia of the right Supramarginal Gyrus (See Photos, Supplemental Digital Content 1 & 2, Axial 5 mm thick T2 and FLAIR MRI Brain slides with arrows indicating area of Focal Cortical Dysplasia of the right Supramarginal Gyrus.). Neuropsychological Testing demonstrated below average performance in tests for working memory. Wisconsin Card Sorting Test showed below average scores in perseverative errors and perseverative responses, indicating poor set-shifting.

In a still antiepileptic-naïve state, a series of epileptic seizures were documented with simultaneous video/EEG registration. The background showed posteriorly dominant 8–12 Hz, 40–60 μV-alpha activity reactive to eye closure. Sleep markers were seen in the recording. The seizure events happened at approximately 2:30AM when the patient was sleeping – he exhibited a sudden flexion of both his upper limbs, pedaling movements of both legs shortly followed by leftward verse neck movements and ocular deviation, followed by a blank stare and an unresponsiveness to verbal commands, all coinciding with epileptiform recordings (See Video, Supplemental Digital Content 3, recording of the seizure activity). The patient did not awaken after the seizure, drifting back to sleep instead. During the seizure there was continuous spike and wave/polyspike discharges localized to the right frontotemporal region. Semiology and the ictal EEG were consistent with seizures of a frontal origin (See Photo, Supplemental Digital Content 4, arrow indicating the onset of continuous spike and wave/polyspike discharges localized to the right frontotemporal region).

2.4. Treatment and course of illness

Following recording of the seizures, the patient was put on incremental dosages of antiepileptics (target dose Oxcarbazepine 1800 mg/day), Fluoxetine (40 mg/day) was continued and he was provided sessions of Cognitive Behaviour Therapy to deal with the pervasive anticipatory dread. Over a 03 month period, he showed steady remission of all three target symptoms – panic and derealisation attacks effectively stopped, and baseline anxiety levels improved steadily. At the time of discharge, his Hamilton Anxiety Scale Score was 18, indicating mild anxiety

3. Discussion

3.1. Background

Attempts at explaining depersonalization and derealisation have ranged from holding a dearth of breast-feeding responsible (the 'impersonal bottle/parent') (Searl, 1932) to conceiving the activation of an ingrained mechanism to escape physical/psychiatric punishment by 'possessing the immunity of an inanimate object' and then compensating for the inanimation by eroticized thinking (Oberndorf, 1934; Searl, 1932). Mescaline has been