

SHORT REPORT

Separating depersonalisation and derealisation: the relevance of the "lesion method"

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Objectives: Depersonalisation (DP) and derealisation (DR) are often met with in patients with a wide range of localisable neurological conditions. This suggests that the "lesion method" might be a valid approach to study the neurobiology of DP/DR. However, the fact that anxiety can trigger DP/DR makes it difficult to establish whether the presence of DP/DR in neurological patients is mainly determined by coexisting anxiety or by lesion location. To overcome this difficulty, we suggest the study of neurological phenomena, which although not considered as DP/DR, bear enough phenomenological resemblance with them as to warrant their use as models.

Methods: One patient with "visual hypoemotionality" and another with "hemiasomatognosia" are described in detail together with a selective literature review.

Results: Complaints of patients with visual hypoemotionality are indistinguishable from those of patients with "visual derealisation". There is also a phenomenological overlap between "asomatognosia" and the symptom of "body alienation", which is a central feature of depersonalisation.

Conclusions: Phenomenological similarities between visual hypoemotionality and DR suggest that a disruption of the process by means of which perception becomes emotionally coloured may be an underlying mechanism in both conditions. Likewise, phenomenological overlaps with asomatognosia suggest that DP might result from parietal mechanisms disrupting the experience of body ownership and agency. These findings give validity to the notion that DP and DR may have distinct neurobiological mechanisms.

Depersonalisation (DP) and derealisation (DR) remain poorly understood and underresearched conditions. DP is often described as an "alteration in the perception or experience of the self so that one feels detached from, and as if one is an outside observer of, one's mental processes or body."¹

DR involves "an alteration in the perception or experience of the external world so that it seems strange or unreal."¹ It is not known if the differentiation between DP and DR has any neurobiological validity.

Although DP/DR can occur as a primary condition,² it often accompanies other psychiatric² or neurological diseases.³

Most of the neurological conditions associated with DP/DR, such as epilepsy, migraine, and mild head trauma etc³ have poor localisation value. However, DP/DR have also been described accompanying localised lesions.^{4,5} This raises the question if the study of such cases (the lesion method) might be a valuable approach to study the neurobiology of DP/DR.

THE EXPLANATORY LIMITS OF "ORGANIC" DEPERSONALISATION

One major problem with the "lesion approach" is that DP/DR may be a non-specific response to brain insult,⁶ and the location of brain lesions might play only an indirect role.⁷ Another problem is that intense anxiety has been shown to trigger DP/DR,⁸ and this could confound the association with some neurological diseases.^{9,10}

In view of the above difficulties, an alternative approach might be the study of neurological conditions that although not considered as DP/DR, bear enough phenomenological resemblance to them as to warrant their use as models. In particular, this paper will draw attention to the phenomenological similarities between DR and visual hypoemotionality and between DP and asomatognosia. It is our contention that the study of neurological phenocopies of DP/DR might help tease out those symptoms with more neurobiological grounding from those more psychosocially determined.

Two cases assessed by the authors will be presented to substantiate our ideas.

"VISUAL HYPOEMOTIONALITY" AS A NEUROLOGICAL MODEL OF DEREGALISATION

Emotional hyporeactivity to visual stimuli (visual hypoemotionality) has been described in some patients with prosopagnosia (an inability to recognise familiar faces).^{11,12} Patients with visual hypoemotionality complain that what they see lacks vividness and emotional colouring, and their narratives resemble those of patients with DR. Visual hypoemotionality is thought to be a consequence of a right basal occipitotemporal lesion or bilateral basal occipitotemporal lesions, thought to disconnect visual from temporal-limbic areas.¹¹

CASE 1

The patient was a 58 year old man with a high educational level, who sustained a severe head injury and developed bilateral basal temporo-occipital haematomas (fig 1). From the onset, it became clear that in addition to prosopagnosia, the patient also described an inability to endow visual perception with emotional feelings (for a full report of neuropsychological findings see Lopera and Ardila¹³). The following verbatim excerpts from an early interview convey the intensity and ineffable character of this symptom.

Interviewer "What is it for you to look at flowers, or a landscape?"

Patient "Flowers to me have lost their essence, I fail to see them as part of nature. They have become almost synthetic, artificial, I seem to lack a kind of knowledge, no, it's not really a knowledge, rather a certain clarity to see nature itself. I fail to see the flower in all its authenticity."

Interviewer "What about landscapes?"

Abbreviations: DP, depersonalisation; DR, derealisation

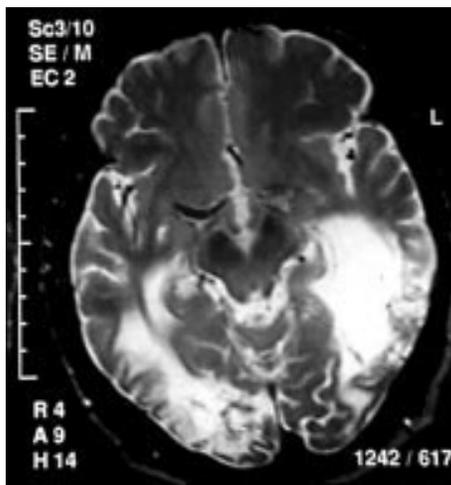


Figure 1 MRI of case 1. Bilateral occipitotemporal lesions are present with a more basal distribution on the right side and a left posterior temporal lesion.

Patient “Just as with flowers, there is also an emptiness to landscapes. I cannot appreciate them, I cannot grasp the beauty of nature. I lack a kind of lucidity, a lucidity in my vision that would normally allow me to appreciate it; its colours, the temperature of its colours so to speak. I cannot think of a right word to explain it. I just cannot enjoy that sense of beauty that nature brings.”

The above complaints were specific for the visual modality. In fact, he noticed increased emotional reactivity to auditory information. A thorough psychiatric assessment did not show any underlying psychiatric pathology.

DISCUSSION (Case 1)

The phenomenological overlap between the experiential narratives of patients with DR and with visual hypoemotionality, suggests a common underlying mechanism—namely, a disruption of the process by means of which perception becomes emotionally coloured. It is our contention that the latter may result in a qualitative change in the experiencing of perception, which is then reported by the subject as unreal or detached.

A further parallel with visual hypoemotionality is the fact that in DR the visual modality is more often affected,¹⁴ and in some patients this impairment seems selective.⁶

Likewise, as seems to be the case with visual hypoemotionality,¹¹ patients with DP have been shown to have diminished electrodermal responses to emotional pictures.¹⁵

“Hemidepersonalisation”: asomatognosia as a model for depersonalisation

The term ‘asomatognosia’ refers to an alteration of body awareness in which a patient denies ownership of a limb (is unable to recognise the affected limb as his or her own) contralateral to a brain lesion (usually right parietal).¹⁶ Early neuropsychiatrists drew attention to its phenomenological similarity with DP (loss of feelings of agency—that is, the feeling that one is not in charge of one’s movements; and loss of body ownership feelings are central features of DP¹⁴) and used the term “total asomatognosia” when referring to patients with DP.¹⁷ Alternatively, other authors have referred to asomatognosia as “hemidepersonalisation”.^{18–20}

As is the case with DP, asomatognosia often retains an “as if” quality (for example, the feeling as if no left arm and leg existed),²⁰ and can be intermittent or continuous.^{16, 20}

Patients with asomatognosia can also experience lack of agency feelings (for example, the subjective component of the alien hand syndrome) as a result of the same parietal lesion.²¹

Unlike patients with neglect syndromes or anosognosia, patients with asomatognosia do not show attentional impairments and do not deny any additional deficits (there is commonly a left hemiplegia).²⁰ In addition, neglect syndromes and asomatognosia seem to have different neurological substrates.¹⁶

The following is the case of a patient who developed hemiasomatognosia and DP after a right parietal chronic subdural haematoma.

CASE 2

A 39 year old woman was taken to casualty after having a clonic simple partial seizure in her left hand, lasting several minutes. There was a history of frequent headaches and vomiting during the previous 15 days. She had no history of epilepsy, migraine, or any other neurological or psychiatric condition. Neurological assessment disclosed a left hemiparesis, right oculomotor paresis, and bilateral subhyaloid haemorrhages. A brain CT showed a right subdural haematoma in the right parietal lobe and a carotid angiography showed an aneurysm of the right carotid siphon. The haematoma was drained and the aneurysm clipped. She made a satisfactory recovery except for a residual mild left hemiparesia. However, at a follow up assessment, she complained that the left side of her body felt strange, as if it did not belong to her. Also on occasions she had the feeling as if her fingers on the right hand suddenly “dropped off her hand”, or as if her limbs had disappeared. These episodes would last a day or two and were not accompanied by tactile numbing or clouding of consciousness. She found these experiences distressing and felt an urge to touch her limbs or pour hot water on them to reassure herself of their existence. In addition, the patient also complained about more global DP symptoms in the absence of DR: “Sometimes I do not seem to know who I am, I doubt my own existence, I feel like a piece of furniture. I do not feel either a human being or an animal. I can feel the heat and the cold but at the same time it feels very weird. I walk but I just cannot convince myself that I am walking as I have this feeling as if I could not move, as if I was a piece of furniture.”

She was not depressed or delusional but seemed slightly anxious (Beck’s depression Inventory=10,²² Zung anxiety inventory=20,²³ Dissociative experiences scale=8²⁴). Facial emotional expressions and prosody were preserved.

DISCUSSION (Case 2)

In summary, the patient had a right parietal chronic subdural haematoma which was accompanied by a left hemiparesia and left hemiasomatognosia. She also described more general DP feelings. It is unclear if this more global DP syndrome was related to the hemiasomatognosia but her descriptions suggest a close similarity. Indeed, patients with asomatognosia often experience global (not restricted to one hemibody) distortions in body experiencing.²⁵ Interestingly, an antagonistic relation between “lack of body ownership feelings” and DR, has been described in patients with full blown depersonalisation syndrome in the absence of brain damage.^{6, 26}

SUMMARY AND CONCLUSIONS

Because both visual hypoemotionality and asomatognosia depend on the localisation of lesions and are unlikely to be significantly modified by psychological variables, we suggest that their study as neurological models of DP/DR might advance our understanding of the neurobiological mechanisms which underlie these conditions. Our two cases suggest a double dissociation between DP and DR-like symptoms in patients with brain damage—that is, patients with visual hypoemotionality—do not complain of DP; and patients with hemiasomatognosias do not report DR. These findings, and the fact that temporal and parietal lesions seem to reproduce

different components of the DP syndrome, suggest that different phenomenological components of the DP syndrome might have distinct neurobiological substrata. In keeping with our findings, a recent PET study found that patients with DP differed in relative glucose metabolic rate from comparison subjects in portions of the sensory cortex in the temporal, parietal, and occipital lobes.²⁷

A recent model of depersonalisation proposes that inhibitory mechanisms on emotional processing underlie the experience of unreality, and hence support the view that a functional corticolimbic disconnection is present in DR.²⁸

In summary, the evidence discussed suggests that the phenomenological differentiation between DR and DP has neurobiological validity.

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