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Finding out if “The ‘me’ will shut down”: Successful cognitive-behavioural therapy of seizure-related panic symptoms following subarachnoid haemorrhage: A single case report

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Successful cognitive and behavioural therapies for anxiety disorders in separate cases of acquired brain injury and seizure disorder have been reported although evidence of efficacy is limited. This paper describes the presentation and cognitive-behavioural therapy (CBT) of seizure-related panic symptoms in the context of subarachnoid haemorrhage and cavernoma. Multidisciplinary clinical assessment was conducted and 12 sessions of CBT according to the model of Clark (1986) were delivered. Outcome was measured in terms of goal attainment, belief ratings of target cognitions and completion of standardised questionnaire measures pre and post-treatment. Process was measured through client's ratings of anxiety-related beliefs through treatment. The client attained all goals, eliminated avoidance and other unhelpful coping behaviour, and rated reduced levels of anxiety on a standardised measure. Changes in identified target cognitions were also evident. It is concluded that a cognitive-behavioural approach may be helpful in understanding and treating anxiety disorders where symptom presentation is complicated by neurological problems. Further investigation of the relationship between development of anxiety disorders, occurrence of neurological events, and processes of CBT following acquired brain injury is suggested.

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INTRODUCTION

Subarachnoid haemorrhage (SAH) is a form of acquired brain injury the consequences of which include emotional, cognitive and physical impairments, increased likelihood of seizure disorders, and restricted social participation consequent to these problems (Lishman, 1998). Cavernous haemangioma or cavernoma is a benign vascular malformation disorder thought to occur in 0.02% (Robinson, Awad, & Little, 1991) to 0.5% (Otten et al., 1989; cited by Moran et al., 1999) of the population. Susceptibility to both partial and generalized seizures is high, and varies depending on the location of the cavernoma (Moran et al., 1999).

Treating emotional consequences of SAH

Application of psychological intervention for emotional sequelae of acquired brain injury has been described and potential efficacy of cognitive-behavioural therapy (CBT) recognised (Williams & Evans, 2003). Williams, Evans, and Fleminger (2003) note that evidence-based CBT protocols for treatment of DSM-IV (Diagnostic and Statistical Manual 4th Edition, American Psychiatric Association, 1994; DSM-IV-TR, American Psychiatric Association, 2000) Axis I anxiety disorders are applicable following brain injury, although atypical presentations and interactions need to be recognised. Hibbard, Grober, Gordon, and Aletta (1990) describe specific adaptations required for CBT following stroke. Alderman (2003) notes that wide individual variability between cases following traumatic brain injury warrants a single case, rather than a randomised control trial approach to gathering evidence for effective interventions. In support of application of CBT, Nicholl, Lincoln, Muncaster, and Thomas (2002) report negative cognitions in post-stroke depressed clients to be comparable to those in non-neurological cases of depression. Lincoln, Flannaghan, Sutcliffe, and Rother (1997) report a successful pilot study of CBT for post-stroke depression. A randomised controlled trial (Lincoln & Flanagan, 2003) did not demonstrate efficacy of CBT compared with a placebo treatment and standard care, although the investigators recognise methodological weaknesses.

Psychosocial and emotional aspects of epilepsy and application of CBT

Psychological consequences of seizure disorders have been described and clinical levels of anxiety identified in 25–33% of cases where both neurological and adjustment or stigma-related aetiology may be present (Baker, 1997). Claassen et al. (2003) found seizures to occur in 7–12% of cases following SAH, also identifying an association with poorer functional outcome and increased anxiety.

Successful psychological treatments for epilepsy (Goldstein, 1997) and related psychosocial consequences (Baker, 1997; Goldstein & Cull, 1997) have been reported. Goldstein (1997) offers a description of cognitive-behavioural interventions for seizures, featuring aura, prodrome, seizure, and thought monitoring, and teaching of coping skills such as relaxation and inhibitory counter-measures. Baker (1997) describes approaches to managing emotional adjustment and other psychosocial consequences of epilepsy. A systematic review by Ramaratnam, Baker, and Goldstein (2003) concluded psychoeducational programmes to have likely effectiveness on emotional outcome, while findings regarding CBT have produced both negative (Tan & Bruni, 1986) and positive (Davis, Armstrong, Donovan, & Temkin, 1984) outcomes. A behavioural model of development of phobic responses to seizure triggers has also been presented (Noecker & Haverkamp, 2001), which describes successful treatment of photophobia that developed secondary to photogenic partial seizures.

This brief review suggests that cognitive-behavioural approaches, especially where psychoeducation is provided, may be helpful for addressing anxiety disorders following acquired brain injury, and for psychosocial aspects of seizure disorders. Exposure-based behavioural intervention may be of use where phobic disorder develops. However, evidence is both limited and inconclusive and further well-conducted single case studies will help to develop the evidence base where variability between cases limits the usefulness of group-based outcome studies.

We describe our client Paul, and his presentation following SAH, in the context of cavernoma and post-stroke seizures. We present the successful application of a cognitive model of panic (Clark, 1986) to the treatment of anxiety symptoms relating to the occurrence of neurological symptoms. The case aims to contribute to evidence for interventions targeting the psychosocial consequences of neurological disorders and acquired brain injury, and is augmented with Paul's first person accounts of his symptoms and the intervention.

CASE REPORT: PAUL

Method

Paul experienced a SAH at age 42, resulting in loss of consciousness for approximately 5–10 minutes, and hospitalisation for one week. The initial CT scan identified blood in the third ventricle and pons, no hydrocephalus, and subarachnoid haemorrhage over the vertex. A follow-up MRI (magnetic resonance image) report 4 weeks later confirmed presence of blood products overlying both cerebral convexities, in the occipital and inferior left frontal

regions, as well as on the left side of the pons. The pontine lesion was thought to be consistent with a small cavernoma. Paul reported no history of anxiety or depression prior to the SAH.

Assessment

Paul was assessed at a neuropsychological rehabilitation centre 1 year and 8 months following the haemorrhage. Assessment followed a biopsychosocial approach to emotional consequences of acquired brain injury (see Ponsford, 1995; Williams & Evans, 2003) and seizure disorders (Baker, 1997; Barry, 2003). The aim of this was to provide an account for social participation limitations in terms of deficits and emotional adjustment.

Cognitive functioning

Cognitive impairments relative to premorbid functioning included some subtle visual memory difficulties, and a relative drop in attentional functioning compared with estimated premorbid levels. Functioning was reported to fluctuate according to arousal and fatigue levels.

Neurological symptoms

Post-haemorrhage neurological symptoms included altered scalp sensations, both partial and generalised tonic seizures, altered sensation to noise and light, and migraine. No epigastric sensations, olfactory sensations or any other auras preceding seizures were reported. Paul reported two clear examples of seizure occurrence following the SAH. On each occasion Paul described feeling unwell and dizzy, experiencing some alteration to his thinking (“my brain flooding with thoughts”), symptoms worsening, leading to lability of mood and a brief alteration of consciousness, with recovery lasting hours. On one occasion this was followed by a generalised seizure and hospital admission. Seizures were treated with sodium valproate, and migraines treated with a tryptan.

Paul also reported recurrent symptoms in instances that were similar to the seizures described above but where symptoms remitted on departure from the situation or through use of other strategies. Paul attributed the lack of onset of a seizure on these occasions to successful management of a neurological vulnerability, appraising symptoms as a consequence of the haemorrhage that would not be amenable to treatment. Paul specifically attributed such symptoms to sensitivity to noise and light, and reported triggers included the lighting in a particular supermarket, long car journeys, and busy social events or gatherings. Paul reported using sunglasses to manage lighting, headphones to block out noise during car journeys, and sitting down or leaving situations. He did not describe feeling particularly anxious, adding that “I don’t do panic”,

and initial assessment score for anxiety on the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) was below clinical cut off. Paul also noted symptoms arising in other situations that he would not consider typical trigger situations. A strong tendency to be hypervigilant to potential “triggers” and neurological symptoms was evident.

Diagnosis and initial formulation of emotional adjustment

The failure of ratings of anxiety symptoms to reach clinical cut-off on the HADS was accounted for in terms of Paul’s cognitions regarding the SAH, cavernoma and seizures, and his notion of himself as someone who does not “do panic”. Thus while anxiety symptoms may have been present, they were considered both atypical, and also not perceived or experienced by Paul as anxiety related. The atypical acute symptom presentation and notable hypervigilance was formulated as potentially arising from an interaction between normal attentional changes during anxiety, impaired attentional functioning, and altered physiological arousal.

Paul’s presentation of possibly anxiety-related somatic symptoms arising in response to a set of specific triggers, at times arising “out of the blue” and associated alteration of behaviour in relation to these triggers, if not arising directly from neurological events, was sufficient to satisfy DSM-IV-TR Axis I criteria for panic disorder (American Psychiatric Association, 2000), as an indirect consequence of a medical disorder, DSM-IV-TR Axis III (American Psychiatric Association, 2000).

Paul appeared to be describing two distinct phenomena, seizures and panic attacks, and this formed the basis for the cognitive-behavioural formulation, as described below and shown in Figure 1. As noted above and in contrast to typical panic disorder clients, Paul did not describe marked anticipatory anxiety relating to trigger situations.

Social participation

Functionally, secondary to the impairments and symptoms described above, Paul was unable to work full time. At assessment he was working flexibly for himself allowing more control over his work environment, deadlines, and pattern of work according to his fluctuating cognitive and physical symptoms. Possibly as a result of his perception of vulnerability to neurological symptoms, Paul reported a reduced ability to manage social situations, resulting in reduction of his social network. He reported reduced ability to engage in routine activities such as shopping, and longer car journeys. He made use of an electronic organiser as a memory and planning aid, and maintained a supportive relationship with his long-term partner. The assessment concluded that psychological treatment for coping with the symptoms

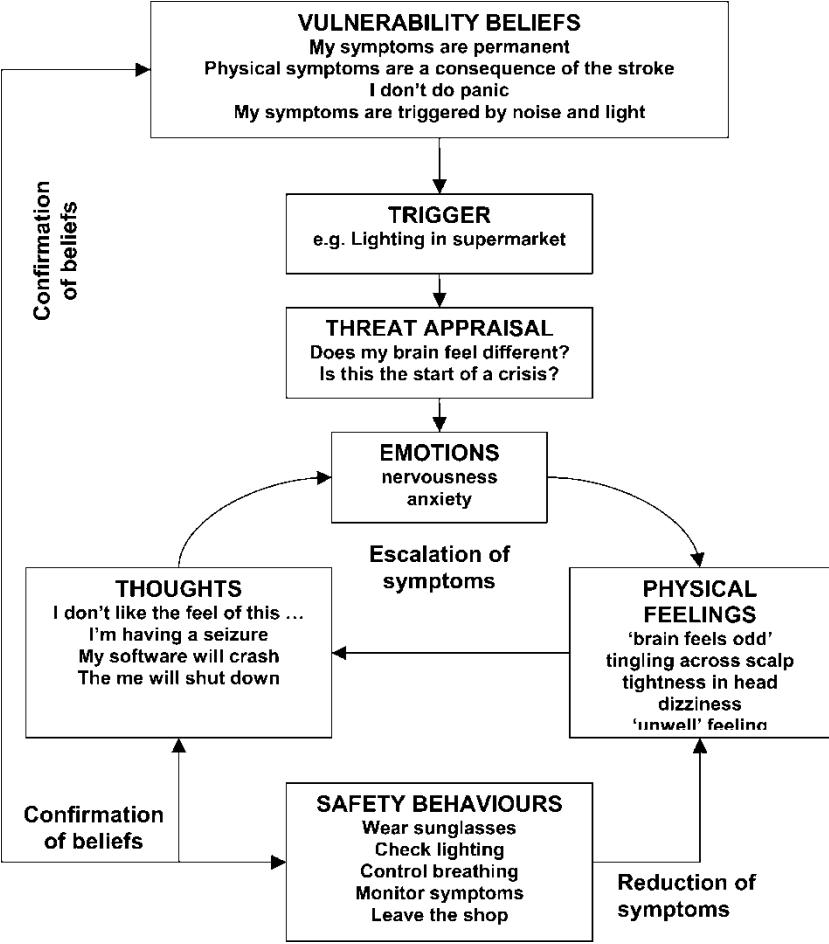


Figure 1. The cognitive panic cycle formulation developed with Paul based on the models of Clark (1986) and Wells (1997).

underpinning restricted social participation using a cognitive-behavioural approach should be tried.

Background to treatment

Efficacy of CBT was measured through assessment of symptoms of anxiety and depression, goal attainment, and changes in ratings of relevant cognitions. The Hospital Anxiety and Depression Scale (HADS: Zigmund & Snaith, 1983) is a standardised measure of mood for use with patients

with physical problems. This was administered pre and post-treatment and at 3 month follow-up. The Panic Rating Scale (PRS; Wells, 1997) assesses frequency of panic attacks, coping behaviours and cognitions, and was used repeatedly through therapy to monitor change. Ratings of cognitions specific to the formulation of Paul's anxiety were also carried out through treatment.

Treatment

The protocol followed in this case was based on the approach described by Wells (1997) based on the model of Clark (1986) and was carried out over 10 weekly sessions, followed by 2 follow-up sessions at 3 and 6 months. The approach was tailored to the specific formulation of Paul's presenting problems. The key issues requiring further attention as compared to CBT for panic disorder in non-neurological clients were: altered self-report of anxiety symptoms, possible interaction between attention, physiological arousal, and affect; and the presence of real, rather than perceived, health problems. McGrath and King's (2004) description of the application of behavioural experiments (BE's) in CBT following ABI, and Silver, Surawy, and Sanders (2004) use of BEs in the context of CBT with physical illness, which highlights the role of illness-related cognitions, were drawn upon. These considerations and approaches were integrated into the model of panic used, forming the basis for changes in emphasis of intervention and individual formulation, rather than radical changes to the approach. Interventions carried out are described below.

Agreement of treatment targets. Paul described his goals for treatment as follows:

- To be able to go to the supermarket and other shops without wearing sunglasses and be symptom-free.
- To stay in busy social situations for longer and without experiencing symptoms.
- To complete a long car journey without using noise cancelling headphones and be symptom-free.

Socialisation to therapy. The cognitive model of panic was discussed with Paul. A possible distinction between past seizure-related events in which symptoms worsened regardless of leaving the situation, and panic symptoms which remit after leaving the situation was proposed. He accepted the rationale of the model as a possible explanation that could be investigated through therapy, and was curious to explore this hypothesis.

Collaborative formulation. The process of formulation in panic described by Wells (1997) was carried out with Paul and repeated a number of times

over sessions 1–4 in relation to specific trigger situations. A “panic cycle” maintenance formulation was developed from this and is presented in Figure 1. The atypical factors relating to physiological arousal, attention and neurological symptoms were not included in the diagrammatic formulation developed with Paul in order to keep the formulation and rationale for intervention clear and simple. Illness-related cognitions were recognised at both vulnerability and maintenance aspects of the formulation. The issues were however discussed with Paul throughout treatment.

Paul reported symptom onset in large stores, in particular one supermarket. He attributed this to the effects of lighting, and reported wearing sunglasses to be mostly effective in reducing symptoms. The wearing of sunglasses was formulated as a “safety behaviour” that temporarily reduced anxiety but prevented disconfirmation of the belief that the lighting would trigger a catastrophic neurological event. Similar safety-seeking processes were used to understand wearing of headphones in the car, and early departure from busy social events.

Specific threat-related appraisals were not initially evident from questioning. However, during further formulation Paul described his memories of seizure onset. He described a distressing progressive deterioration in his cognitive faculties, this “being like death only slightly less so” in that the body stays living, like “the software will crash ... the ‘me’ will shut down”. This was identified as the “hot cognition” linking seizure experiences and symptoms, cognitive appraisals, and escalation of anxiety symptoms.

The main target cognitions thus related to:

- Paul’s belief that he experiences photosensitive and other neurological events that are not amenable to change, “my problems are permanent and won’t change”.
- Paul’s appraisal of physiological symptoms as a sign of a threatening neurological event, “these physical sensations are a sign of something going wrong in my brain”, “the software will crash ... the ‘me’ will shut down”.

Restructuring target cognitions. Cognitive restructuring of specific symptom appraisals through behavioural experiments commenced in session 4. The aim of the experiments was to test the target cognitions identified by Paul through the collaborative formulation against an alternative perspective that his symptoms were anxiety related, and that they would thus initially worsen then improve without use of “safety behaviours”.

Paul carried out this experiment, and experienced escalating then diminishing symptoms as predicted. Paul documented the experience in his personal journal as follows:

"It was going into the store which was where the 'fun' really began. It's hard to describe but at the time it seemed like my brain was 'buzzing' (in retrospect I think 'roaring' might be a better description) and drowning out everything else. I mean that literally. I could almost hear it! It was quite unlike anything I'd experienced before and seemed to be occupying my whole brain, filling all my senses. It was scary but unlike before when I've had problems, at one level my brain was going 'this is just a panic attack, nothing really bad is going to happen' and after a minute or so it faded away. Superb. I remember grinning inanely."

This experience was interpreted as supportive of the "panic" hypothesis, that symptoms were amenable to change, and that symptoms were not necessarily a sign of imminent neurological threat. The experiment was repeated two further times with reduction of symptoms and increased belief in the alternative explanation. The principles of this experiment were then generalised to work on longer car journeys and busy social situations.

Relapse prevention. A plan was drawn up with Paul to help him maintain awareness of the potential development of new "safety behaviours" in relation to apparent neurological symptoms. Review sessions at 3 and 6 months involved reflection on maintenance of gains and identification of any problems arising.

Results

Behavioural measures and functional outcome

Paul reported full engagement in previously limited or avoided activities, other than when limited by fatigue or other post-SAH symptoms. The functional goals of going to the supermarket, social gatherings and long car journeys remaining symptom-free were all achieved. At 6 month follow up Paul reported continued engagement in these activities with minimum symptoms unless affected by headache or fatigue.

Questionnaire measures

Reduction in HADS Anxiety and Depression scores across treatment can be seen (Figure 2). The initial HADS Anxiety rating fell below the clinical level, although at this point Paul attributed most of his symptoms to neurological problems, thus biasing his pattern of response on this questionnaire. In session 1 the questionnaire was completed "as if" reported symptoms were anxiety symptoms. The HADS Depression subscale score fell below clinical cut-off following initial assessment, dropping further by treatment

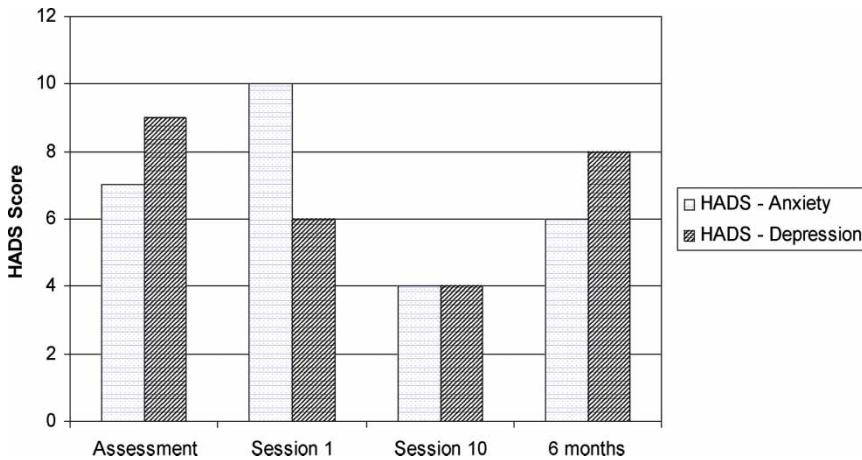


Figure 2. Changes in HADS Anxiety and Depression scores across treatment.

end. HADS ratings increased slightly by the 6 month follow-up, although remained below clinical cut-off.

Ratings of frequency of use of avoidance or safety seeking behaviours, and in beliefs relating to fear of a catastrophic neurological event occurring when panicking as measured by the Panic Rating Scale (Wells, 1997) also showed changes in parallel with the intervention. Specific safety behaviours (wearing sunglasses or headphones) and beliefs (“the me will shut down”, “these symptoms are permanent”) relevant to Paul’s formulation reduced. Ratings of use of safety behaviours fell to zero for all except the items “control my mind” and “control my breathing” which both fell to 4/8 (use half the time) at treatment end, rising at 6 month follow-up. Ratings of degree of belief in core anxiety or symptom-related cognitions “the me will shut down” and “these symptoms are permanent” fell to zero by session 10, maintained at follow-up. At the end of treatment Paul was able to distinguish the symptoms targeted in therapy from other symptoms (fluctuating fatigue and attention levels, attentional difficulties, headaches) directly attributable to neurological factors. He reported a changed perspective and understanding of the nature of his symptoms.

DISCUSSION

This case highlights the potential development of anxiety-related problems secondary to seizures or other neurological events, in this case in the context of SAH and cavernoma and related seizures. This case demonstrates successful cognitive-behavioural treatment for a post-ABI seizure-related

anxiety disorder through which the client attained his goals for treatment with a significant positive impact on social participation. Improvement in anxiety and depression symptoms was recorded at end of treatment. An increase in symptom ratings and ratings of frequency of use of two specific safety behaviours 6 months after the end of treatment suggests that further relapse prevention work may have been helpful, although Paul did maintain his functional gains. As with Noecker and Haverkamp's (2001) case, exposure-based treatment was effective in the context of anti-epileptic medication. The cognitive-behavioural intervention in the case presented here, did not, however, focus on control of anxiety and then exposure, but exposure to increased levels of arousal to allow reappraisal of physical symptoms as non-threatening and changeable.

The case raises important issues relating to assessment and outcome measurement. The client's reporting of specific symptoms of anxiety as neurological symptoms, possible interaction between neurological and anxiety symptoms, and non-reporting of subjective anxiety or panic presented a challenge to diagnosis, formulation, and identification of intervention. The scores on the standardised measure of anxiety (HADS) alone would have been misleading. While this measure may often yield valid information in seizure disorders (Jackson & Turkington, 2005), this case suggests that cautious interpretation is warranted. Assessment of outcome at least in part by measurement of goal attainment is supported.

This pre-post design single case study, while suggestive of efficacy of CBT, is not without its limitations. Generalisation of conclusions about efficacy of CBT in other cases of anxiety disorder in the context of neurological problems is not possible from this single case. From the initial HADS Anxiety score, it is evident that symptom levels were not high prior to treatment. Possible relationships between seizure onset and anxiety arousal were not explored in this case, and it is not possible to draw firm conclusions about the causal role of alteration of target cognitions with this design in which ratings of cognitions only occurred at treatment start, middle and end. However, it is clear that Paul held specific cognitions about symptoms and that the altered interpretation of his symptoms, change in experience of symptoms and behaviour change occurred together, consistent with the progress of therapy. Furthermore following the intervention Paul maintained behaviour change and improved levels of social participation compared with initial assessment. The potentially important role of cognitions about alteration or loss of consciousness, "threat to self" (here implicit in the belief "the me will shut down"), and about the nature and controllability of symptoms in anxiety disorders in the context of neurological problems is highlighted.

It is possible that anxiety-related representations of onset of neurological symptoms, specific neurological pathology and cognitive deficits of attention interacted, in the context of idiosyncratic cognitions about anxiety and

neurological symptoms, to account for the altered presentation here. Attention to such interacting processes may be of particular value in formulating interventions for managing altered affective presentations following SAH. However, further research into the nature of such interactions, and development of appropriate measures, are required. The integration of psychoeducational and cognitive-behavioural approaches to addressing beliefs about neurological symptoms may be worthy of further investigation. Future research may usefully target identification of common cognitions about seizures and SAH in clients, as well as further dissemination of treatment outcomes in such cases.

CONCLUSION

In the context of anxiety-provoking neurological disorders, clients may present with apparent neurological symptoms that interact with anxiety symptoms. Loss or alteration of consciousness at the time of a critical neurological incident, neurological and cognitive impairment, and individual cognitions about symptoms may contribute to altered presentation or atypical self-report of symptoms by the client, and development of specific self or consciousness-related threat beliefs. Interventions based on a cognitive-behavioural conceptualisation may offer an efficient and effective approach to treatment. Adaptations for CBT following ABI suggested in the literature appear applicable in this case.

Further research is needed to clarify the interacting processes that may account for altered affective presentations following SAH. Such research would contribute to an understanding of complex presentations and provide a means of identifying those clients whose symptoms may be amenable to psychological intervention, help build a cognitive-behavioural model that takes into account neurological and cognitive problems and processes, and provide a rationale for choice of intervention.

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