be fatal if missed, thrombocytopenia, altered platelet aggregation, and minor gastrointestinal intolerance, but for a patient who is unable to function because of chronic illness the benefits may outweigh the risks.

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Chronic Depersonalisation Neurosis *au* Shorvon – A Successful Intervention

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A patient with chronic primary depersonalisation responded well to a combination of psychotherapy and abreaction.
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In ICD-9 (World Health Organization, 1978), depersonalisation neurosis is described as "a neurotic disorder with an unpleasant state of distorted perception in which external objects or parts of one's body are experienced as changed in their quality, unreal, remote or automised. The patient is aware of the subjective nature of the changes he experiences."

Shorvon (1946) has described what he called primary idiopathic depersonalisation, a disorder of primary depersonalisation commencing in late adolescence and which is continuously present for many years, although with some fluctuation in severity. We report a patient with this disorder who improved substantially with a combination of psychotherapeutic techniques and abreaction.

Case report

A 55-year-old lady was referred to a psychiatric out-patient clinic because of worsening of her feelings of unreality, which had been present for 35 years. She complained that her voice felt distant from her, that she felt distant from things, and that she felt as if she were not there. She found these phenomena difficult to explain but said that she felt herself to be unreal. She was able to feel the full range of emotions, and no parts of her body had a 'woolly' or unreal character. She had no altered perception of time, although she complained of distorted perception of space. Her image seemed real in the mirror and other people and external objects retained their real quality, but she described life as sometimes having the quality of a play.

She described occasional dysphoria, usually when she was inactive and when she had time to ponder on the unpleasantness of her subjective experiences. She had never had any biological features of depressive illness or features suggestive of an agoraphobic, obsessional, or other neurotic disorder, apart from primary depersonalisation. She had never suffered from features suggestive of temporal lobe epilepsy, she never abused alcohol or drugs, and there was no history of head injury or intracranial infection.

The subjective depersonalisation first arose when she was 20, at a time when her husband was called up for national service, leaving her at home to look after two children under 18 months of age. She is unable to recall the initial experience, but feels that its onset was insidious. She was first referred to a psychiatrist in 1954 when she was prescribed electroconvulsive therapy; this gave no improvement. She soon became disillusioned with psychiatric

practices and stopped attending, despite the persistence of her unpleasant symptoms.

She was again referred to psychiatric attention in 1976, when she was prescribed a combination of drug therapy with amphetamines and trifluoperazine. Again there was little improvement and she stopped attending, although continued the drug prescription for six years and describes feeling a little better, but without the depersonalisation improving. She continued to take trifluoperazine until 1989 although the amphetamine was discontinued by her general practitioner in 1982. During the 1976 attendance she was given a full blood screen and electroencephalography, neither of which revealed any underlying abnormalities.

She was referred to psychiatric care once more in 1987, when she attended several times as an out-patient and was prescribed lofepramine (70 mg t.d.s.) on the rationale of a possible underlying masked depression. After several attendances she again stopped attending because of the lack of improvement. Therefore, over 30 years of visiting three different psychiatrists at different hospitals, there was never any evidence of underlying psychiatric morbidity other than the primary depersonalisation and therapeutic interventions proved of little benefit.

Despite the continued persistence of the depersonalisation symptoms, she managed a fairly successful career within the insurance business and managed to continue a stable marriage and bring up two children. Several changes in her social circumstances over the year before her referral in 1989 precipitated a worsening of her condition. These included taking early retirement from her job because of cervical spondylosis, with the result that the majority of her time was not constructively occupied. In addition, her mother suffered from a serious illness which necessitated her spending long periods of time nursing her mother. She found this excessively stressful, particularly as the resentment she felt towards her mother from childhood had been intensified by her mother's lack of gratitude.

Several obsessional characteristics were evident during interview: her precise phraseology, the care taken over her appearance, and the way she described planning daily routines. This was confirmed by a symptom score of 21 on the Leyton Obsessionality Inventory (Cooper, 1970).

After two assessment sessions, a number of psychological conflicts became evident and a trial of dynamic psychotherapy was felt to be appropriate to investigate these issues further. Weekly sessions were undertaken by a male therapist, with regular supervision sessions in a group format.

Session 1. The patient verbally communicated a loathing and hate of her father, although she talked about him in terms suggesting fondness. This was highlighted. She also described the attributes of her husband in a way that strongly resembled her earlier description of her father; this link was made.

Session 2. She described a strong desire to be successful, particularly to be better than men in her professional sphere. It was suggested to her that this might have been because of a desire to impress her father; she denied this strongly and became emotionally aroused.

Session 3. She expressed a great deal of anger and hostility towards her mother, saying that her mother had never loved her and had never thanked her for caring for her through her illness. She strongly denied any other possible basis for her anger, and again was emotionally aroused.

Session 4. Much of the ground from the previous session was covered again. In this session she demonstrated a much stronger denial of any other reasons for her anger or hate.

Sessions 5 and 6. The patient wanted to discuss her extramarital affairs and was very keen to portray herself as someone who had a strong sexual drive. She made sexually suggestive remarks during the sessions. This was interpreted to her as a transference response of emotions directed towards her father. She again denied this strongly but in doing so demonstrated considerable distress.

Session 7. She discussed her desire to be loved by her mother but felt that this was not possible. She initially denied any destructive feelings towards her mother but later in the session admitted to these feelings and felt that perhaps it was her fault that her mother did not love her.

It was evident at this stage that the patient had an extremely immature pattern of ego defence mechanisms which included projection and splitting. It was hypothesised that she harboured destructive feelings towards her mother, in fantasy as well as reality, and that her description of her mother's animosity towards her were her projection of her own destructive wishes. It is possible that subconsciously she felt that the omnipotent destructive wishes which she had projected onto her mother were responsible for the death of her father. This could tie in with an 'Electra complex', which however would have to exist at the level of a part object. The depersonalisation is more difficult to explain, but may be seen as a repudiation of a self-image coloured by primitive aggressive drive, with resultant withdrawal of self-directed libidinal cathexis.

The commencement of the depersonalisation symptom at a time when her husband had left to do his national service would tie in with this hypothesis, as it is likely that she harboured destructive wishes towards him which she was unable to project successfully onto her mother on this occasion. It was felt that she was unable to relinquish her immature defences with the use of standard psychotherapeutic techniques and in view of the literature on abreaction and depersonalisation (Sargant & Shorvon, 1945) a trial of abreaction was deemed appropriate.

Session 8. An abreaction technique was used with intravenous diazepam. The patient lay on a couch throughout this session. After intravenously receiving 10 mg of diazepam in 20 ml water she initially appeared relaxed, but suddenly became angry and hostile when her mother was introduced into the conversation. She vehemently protested her hatred for her mother and expressed a wish for her mother to die. This phase lasted about two to three minutes before she started becoming drowsy and eventually fell asleep.

Session 9. The patient was anxious and described symptoms of panic attacks, but she no longer had feelings of depersonalisation. She had gained no further insight into her feelings towards her parents and continued to maintain strong defences, and it was feared that further explorative psychotherapy could increase anxiety and lead to re-adoption of the depersonalisation defence. In view of this, termination of explorative psychotherapy was discussed and

in its place a series of sessions concentrating on anxiety management training was arranged.

Over the subsequent three months Mrs A felt subjectively that her anxiety symptoms had improved about 70%. None of the depersonalisation symptoms had returned. Her level of functioning had improved considerably with a return to active voluntary work.

She continues to be reviewed in the out-patient clinic, continues to have good social functioning, and has suffered no return of her depersonalisation symptoms.

Discussion

The main features of the primary depersonalisation syndrome described by Shorvon (1946), including onset in late adolescence, a long, fluctuating course, obsessional personality traits, a cluster of depersonalisation phenomena, and discordance of a parent–child relationship, were all seen in Mrs A. The most dramatic feature of the case was that symptoms were continuously present for 35 years. Importantly, there can be no doubt about the existence of primary depersonalisation neurosis, nor of the primary idiopathic depersonalisation variety proposed by Shorvon.

Shorvon (1946) reviewed 66 patients with primary depersonalisation neurosis. Of these, 12% fulfilled his criteria for the primary idiopathic variety – a severe and persistent form of the disorder.

There have been few outcome studies of primary depersonalisation disorders (Sedman, 1970) and those which have been undertaken have generally shown that patients do not improve with treatment (Noyes, 1987). The only treatment to have demonstrated efficacy was the ether abreaction technique used by Shorvon & Sargant (1945). Eight out of 14 of their patients improved with the use of this method. Interestingly, patients who improved tended to be the ones who were angry and emotionally aroused during abreaction, whether or not specific conflicts were tackled.

This leads to the obvious speculation that it was the use of abreaction that resulted in the resolution of our patient's depersonalisation symptoms, a hypothesis which is supported by the temporal link between abreaction and the patient's improvement, her emotional arousal during abreaction, and the failure to improve her insight into her subconscious conflicts with the use of psychotherapeutic interventions.

A single case report cannot make elaborate claims for treatment successes. However, in conjunction with Shorvon & Sargant's outcome study there is enough evidence to merit further investigation of abreaction as a treatment for primary depersonalisation disorders.

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