

## **A Study of Depersonalisation Neurosis**

Sumant Khanna, MBBS, DPM<sup>1</sup>,

R. Ramasubbu, MBBS, DPM<sup>2</sup> &

S. M. Channabasavanna, MBBS, MD, DPM<sup>3</sup>.

### **SUMMARY**

All 12 cases who received a diagnosis of depersonalisation neurosis over a 6½ year period were studied. The illness was found to occur in young males. It usually took a deteriorating course after starting insidiously. Anxiety, depression and derealisation were common concomitants. Other associated symptoms included deaffectualisation, disturbed sleep and appetite, obsessive ruminations and hypochondriacal preoccupations. An attempt was made to classify the phenomenon of depersonalisation and 7 groups were found. In our cases, better response was obtained when tricyclic antidepressants were tried than when amphetamine was tried. The literature on depersonalisation neurosis is reviewed and discussed in light of our findings.

Depersonalisation is an alteration in the perception or experience of the self so that the feeling of one's own reality is temporarily lost (APA, 1980) with an "as if" feeling (Ackner, 1954 a). In depersonalisation neurosis it is the prominent symptom in the absence of other mental disorder (WHO, 1978). Although the existence of the depersonalisation symptom has been known since a long time (Dugas and Moutier 1911). There is scant literature on the primary depersonalisation syndrome (Shorvon et al 1946). More interest has focussed on the pathogenesis of this symptom (Sedman 1970) than on the existence of the syndrome (Davison 1964). However today it finds a place in official nomenclature (APA 1980, WHO 1978).

### **Methodology**

All patients who received a diagnosis of depersonalisation neurosis (WHO, 1978) in the Psychiatry Out Patient Department of the National Institute of Mental Health and Neurosciences, Bangalore in the period January 1978 to July 1984 formed the sample of this study. Relevant information

from the case files was entered in a semi-structured performa and later analysed. Twelve such cases were found.

### **Results**

a) *Sociodemographic data*: The average age at consultation was 25.08 years (range 15 to 38 years). 10 of the patients were male. 9 were single; 4 were reported to have been shy and sensitive before onset of illness; no other abnormal personality traits were recorded. Only one patient gave a history of a similar episode in the past. There was a past history of enteric fever in 3 cases and of peptic ulcer in 2. In 3 cases there was a family history of mental illness—mental retardation, suicide and unspecified psychosis.

b) *Illness data*: The average age of onset was 21.75 years (range 15 to 38 years) and duration of illness on consultation was 40.17 months (3 months to 15 years). In 11 cases the illness was of insidious onset, starting acutely in one. It took a deteriorating course in all patients, no precipitating factor being present in any case.

<sup>1</sup> Junior Resident, Department of Psychiatry, N. I. M. H. A. N. S. Bangalore - 560 029.

<sup>2</sup> Former Senior Resident

<sup>3</sup> Professor and Head, Department of Psychiatry, National Institute of Mental Health and Neurosciences Bangalore - 560 029.

c) *Depersonalisation*: Out of the 12 cases 1 had derealisation without depersonalisation. In the remainder 11, 31 different depersonalisation symptoms were recorded. These can be classified into 7 groups (Table I).

Table I  
Depersonalisation Symptoms

Involving part of body	9
Involving whole body	9
Nihilistic	8
Automaton	4
Giddiness/movement	4
Weightlessness	2
Non specific	2

d) *Other symptoms*: Anxiety was present in all cases (Table II) followed by depression and derealisation. Deaffectualisation—the feeling as if one cannot feel emotions—was present in a fourth of the sample.

Table II  
Associated Symptoms

Anxiety	12	100%
Depression	10	83.33%
Derealisation	7	58.33%
Deaffectualisation	3	25%
Decreased sleep/apetite	5	41.67%
Obsessive reminations	2	16.67%
Hypochondriacal preoccupation	2	16.67%

e) *Investigations*: Routine blood investigations done in 6 cases were normal. Psychometry was done in 7 cases. In only one case was there a psychotic profile. Others had evidence of anxiety and/or depression with evidence of perception of body distortion. In 6 cases EEG was done, being abnormal in only one. The abnormality was in

the form of theta slow waves in the fronto-temporal regions. This was the same patient with a psychotic profile on psychometry.

f) *Drug response*: In all cases pharmacological intervention was attempted; in some more than one drug was used. Out of the 6 patients who received D-amphetamine, one showed improvement, one deteriorated and there was no effect on the others. Out of the 6 patients who received tricyclic antidepressants, 2 improved. All 3 of the patients on benzodiazepines and 2 on phenothiazines failed to show improvement. One patient underwent behaviour therapy without any beneficial effect.

g) *Follow up*: In one case the diagnosis was changed to obsessive compulsive neurosis, in another to narcolepsy. The diagnosis was stable in the other 10 cases.

## Discussion

The symptom of depersonalisation has been of considerable psychopathological interest (Ackner 1954 a, Sedman 1970, Nuller 1982, Torch 1982) but there have been few studies of the syndrome. Many authors (Mayer Gross 1935, Ackner 1954 b, Roth 1959) feel that it is a common symptom of many psychiatric illnesses. Investigations into its nosological status were heralded by the work of Shorvon et al (1946). Subsequent studies have been few and far apart (Davison 1964, Meyer 1961). Reports in the literature will be discussed in the light of our findings.

Earlier reports stressed that the illness started in young females (Slater and Roth 1977, Meyer 1961, Shorvon et al 1946). We found a preponderance of males in our study. This may be due to the fact that Indian women are more inhibited and most

probably deal with their depersonalisation symptoms more secretly, an argument advanced in another context for Oriental women by Lo (1967). In 11 cases the onset was before 35 years. An earlier report had indicated an association with migraine (Davison (1964); we found an association with peptic ulcer and enteric fever. The family histories did not indicate any genetic factor. Obsessive traits found in earlier reports (Shorvon et al 1946, Slater and Roth 1977) were absent in our study perhaps due to the retrospective methodology.

Most of the cases have been reported to have an acute onset (Shorvon et al 1946) but the opposite was found in our study. While Shorvon et al (1946) refers to a fluctuating course with eventual recovery and Davison (1964) to an episodic course, we found a deteriorating course in all cases, with only 3 showing eventual response even to treatment.

Derealisation rarely occurs alone (Shorvon et al 1946, Editorial 1972) and we found this in only one case. Some authors have tried to club depersonalisation and derealisation (Meyer 1961) while an attempt has also been made to split the depersonalisation syndromes into depersonalisation, derealisation, deaffectualisation and desomatization (Editorial 1972). We have preferred to regard all symptoms as separate.

Many symptoms have been reported in association with depersonalisation neurosis (Shorvon et al 1946, APA 1980). We found anxiety, depression and derealisation to be common concomitants. Disturbed sleep and appetite, with obsessive ruminations, an inability to feel and hypochondriacal preoccupations were also present (Slater and Roth 1977, Nemiah 1980). In attempting to classify the symptoms of depersonalisation,

we found that the predominant group involved change of part of the body, most often the head or the face, and in 2 cases the extremities. The nihilistic group included reports of as if the circulation has stopped, there is no taste and nerves have been destroyed. The feeling of being an automaton (Slater and Roth, 1977) was present in 4 cases.

Earlier reports have supported the use of amphetamine (Davison 1964) but we found that tricyclic antidepressants were still the first line of treatment (Gelder 1983). On follow up only 2 of the cases had a change in diagnosis, suggesting that this is a valid clinical entity.

Differentiation of depersonalisation occurring in normals (Sedman 1966) and the mentally ill is important. Further differentiation between the symptom and the syndrome is essential (Gelder 1983, Nemiah 1980, Lehman 1974). This study provides tentative support for the existence of a primary depersonalisation syndrome. Since this is a relatively rare syndrome (APA, 1980) while depersonalisation as a symptom is far more common, the delineation of this syndrome becomes all the more important. Some of our findings, such as on course and onset and therapeutic response have been at variance with earlier reports. Since this is a basically clinical study, various reasons could be attributed to this. Earlier studies did not differentiate adequately between the symptom and syndrome. Most samples perforce have to be small, and hence individual reports only give an indication of a trend rather than absolute statements. Finally prospective studies, with adequate follow-ups are essential to delineate and refine criteria for this syndrome.

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