Depersonalization Syndromes

Depersonalization is a strange, complex, and essentially private experience, one characteristic of which is the individual's difficulty in communicating a comprehensible account of it.1 2 A prominent feature of the experience is a feeling of change involving either or both the inner and outer worlds and carrying with it a vague but uncomfortable sense of unfamiliarity. The description "unreal" or "detached" is usually accepted,3 but the experience varies greatly between individuals and between attacks.

A variety of terms have been used to categorize the various aspects of the experience. The term "depersonalization" has been restricted to changes in the perception of the entire self, the individual feeling dead, hollow, detached, strange. His actions appear to be automatic or puppet-like. They may be felt as disconnected, so that each normally automatic action has to be thought through in its component parts. "Derealization" is employed to describe the changes the environment seems to have undergone. It may appear like a stage set, two-dimensional or flat, and its colours altered. Things may appear smaller, larger, altered, closer or further away, cloudy, or dreamlike. "Desomatization" describes changes in the experience of the body, individual parts of which may seem enormous, tiny, telescoping, detached, hollow, without sensation, or oddly deformed. "De-affectualization" has been used to describe a sense of the loss of the capacity to feel emotion, so that the person seems unable to cry, worry, love, or hate. The invention of these terms could be extended to cover the experience of such aberrations in perception of space as weightlessness, floating, out-of-body sensations, and the time distortions in which time seems speeded up or slowed down and present actions seem to be occurring in the past or future simultaneously.

But diagnosis is easier than definition in this condition, and for clinical purposes it is sufficient to use the general term depersonalization to cover all of these phenomena which occur intermittently, always have the quality of unfamiliarity and discomfort, and are recognized as changes in experience rather than in reality itself. The patient always uses an "as if" qualification in his often bizarre descriptions of the experience, and one of the serious risks of the condition is that this may be misunderstood and a more malign significance be attributed to it. "I am dead" or "I don't exist" or "My body is not there" may be the delusions of a patient with psychotic depression. Loss of affect or the existence of a feeling that all actions are automatic or controlled like a puppet may suggest schizophrenia. But if the patient says "it feels as if," then depersonalization is the more likely diagnosis.

Attacks of depersonalization may be of any duration from seconds to months but most commonly last minutes or hours. They occur in a wide variety of clinical states, but also occur in normal people, particularly in states of exhaustion, drug intoxication, or when half asleep.4-6 Since the experience is so strange and unfamiliar, it is important for it to be recognized, its significance assessed, and the sufferer reassured.

Transient attacks occurring in isolation, especially when associated with tiredness, require the exclusion of temporal lobe epilepsy, which is usually possible on clinical grounds, and then firm reassurance. Episodic persistent depersonalization can occur, and then requires treatment in a specialist clinic.7 Probably the commonest pathological setting is in association with anxiety syndromes, particularly the calamine syndrome or phobic anxiety depersonalization syndrome,8 and in normal women it is found in association with mild agoraphobia.9 In both neurotic and psychotic depression depersonalization may occur, especially in severely depressed people. During the early stage of schizophrenia true depersonalization may be noted, but it later tends to be incorporated in the delusional system. Acute depersonalization in adolescents is at times associated with extreme panic, resulting in a severe behaviour disturbance which may be mistaken for a major psychosis.

Many patients find relief simply in knowing that this strange and sometimes frightening experience is a common one, familiar to their doctor. His reassurance may be all that is required, but if other symptoms are present it deserves further treatment.

Blood Clotting and the Pill

The possible danger of intravascular clotting resulting from oral contraceptives has been studied in two different ways. The first is by the laboratory measurement of coagulation and platelet changes in necessarily small groups of women taking oestrogen-progestogen compounds. The second is by the epidemiological study of the incidence of thrombosis in women at risk. The latter approach has shown that taking oral contraceptives leads to a considerable increase in the incidence of thromboembolic disorders1 and that preparations with 100 μg or more of oestrogen are more dangerous than others.2

Laboratory studies of clotting may provide an early warning of the dangers of different preparations and may help in the development of less thrombogenic preparations. Though general agreement is lacking on the interpretation of the
laboratory findings, oral contraceptives have been shown to shorten the prothrombin time, the partial thromboplastin time, and the thromboplastin generation test, and assays have shown an increase in specific clotting factors—namely, fibrinogen, factor VII, factor VIII, factor IX, and factor X. Platelet adhesiveness and aggregation are also increased, whereas the possible protective antithrombin III and fibrinolysis are reduced.

The most consistent and reproducible results have been obtained with tests showing acceleration of factors VII and X and of platelet aggregation. Clotting factors are more active from the third month onwards, but changes in platelet aggregation have been noted as early as the first cycle on the pill. All combined preparations, whether of high- or low-dose oestrogen, have produced similar changes.

A recent approach to the prevention of thromboembolism has been to formulate progestogen-only contraceptives, excluding oestrogen. The progestogen chlormadinone acetate when used alone in this way reversed the accelerated clotting patterns in women who had been on combined preparations and caused no change in the clotting factors in women not previously using oral contraceptives. After two years on chlormadinone acetate contraceptive pills some acceleration of platelet aggregation was noted, but it was lower than with oestrogen-containing preparations. Chlormadinone acetate was for a time generally available in Britain but was withdrawn for reasons unconnected with thrombosis.

Since 1969 doctors have been able to prescribe only combined preparations, with an acknowledged thromboembolic risk. The situation is now likely to change dramatically with the imminent release of a number of progestogen-only preparations to be used as oral contraceptives. It is therefore reassuring to learn from a paper by Dr. L. Poller and his colleagues this week (page 391) that one of these, norethisterone, a 19-norsteroid progestogen, resembles chlormadinone acetate in lacking adverse effects on blood clotting. When this progestogen was taken in place of combined preparations, increased levels of clotting factors rapidly returned to normal, and in the first six months of administering it no accelerating effect has been found on a battery of clotting and platelet tests. But long-term studies will be necessary to detect delayed or cumulative effects. As the Manchester workers point out, the absence of clotting and platelet changes is not proof that the 19-norsteroid progestogens will be devoid of thrombotic complications, but the absence of adverse effects on clotting raises this possibility.

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Parents of Psychotic Children

Over the last 30 years considerable attention has been devoted to the interaction between the parents and children suffering from that variety of early childhood psychosis which L. Kanner1 so well characterized and labelled as "early infantile autism." The multitude of terms such as "pseudodefective" and "infantile psychosis" applied to this group of psychoses of early onset tend to reflect the uncertainty about both the pathognomonic features and the aetiological basis.

Kanner also provided the now classical parental stereotype. Alone or with L. Eisenberg,2 he described many of the parents of autistic children as having obsessive and cold personalities, with limited genuine interest in people or an "emotional frigidity." Some degree of detachment and what might be considered introversion have also been ascribed to these parents. Later authors based their psychogenetic theories on the presumed association of the disorder in the children with the personality characteristics of the parents. The putting forward of such theories was understandable because it is a common assumption in child psychiatry that the personality and attitudes of the parents exert a fundamental influence on the developing child.3-6 However, as Kolvin and his colleagues have pointed out, there are two main methodological steps in the examination of such theories—firstly, the proving of a correlation between childhood and parental variables, and secondly experimental or other studies to demonstrate causal relationships between these variables. Unfortunately the early studies did not take this methodological approach, so even without satisfactory objective proof or correlation many doctors formed clinical subjective impressions of coldness and aloofness in parents and attributed the child's autistic condition to the parental personality and attitude. This opened the way to the development of a variety of concepts of autism, explaining it as an extreme variant of the aberrant parental personality or an acute reaction in the infant to such types of parental personality. Ideas of this kind led to psychotherapy being undertaken with families in an attempt at modifying parental attitudes and hence the course and the severity of the autistic illness. These theories and practices produced guilt-conned parents of autistic children.

With such serious implications it is astonishing how little and how scientifically inadequate was the basic research which was undertaken, and even this research can be faulted on many counts. Most of the early clinical studies lacked adequate methods for objective clinical assessment and have been described by Rutter7 as lacking quantification or control groups. The most serious lack was that of standard measuring instruments with adequate norms. Even so the major clinical study of E. Mildred Creak and S. Inni8 did not substantiate Kanner's hypothesis or provide unqualified support for the original personality stereotype. They came to the important conclusion that many of the parents did not differ greatly from those whose children were not psychotic and further offered the view that part of the deviant parental attitudes could be secondary to the strain of living with an autistic child.

A number of researchers have now used more objective instruments such as self-rating questionnaires and projective techniques. Even so, the small size of the series has sometimes induced a lack of confidence in the findings and dis-