Personal paper

Idiopathic Parkinson's disease and depression: a psychosomatic view

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SUMMARY The link between idiopathic Parkinson's disease and depression is examined in the light of psychosomatic theory. A view of the condition is offered as a manifestation of chronic emotional disorder in an organic sense. Predisposition arises from bereavement and/or maternal failure in early emotional development.

James Parkinson,¹ in the original comprehensive description of the disease that bears his name, concluded that "senses and intellect" were left uninjured in the process. However, the literature in recent years, admirably reviewed by Tune *et al*,² reveals that psychiatric disorder, including depressive illness, psychoses and cognitive deficits are a frequent occurrence among patients (see also ref. 3). Mindham⁴ has shown that 90% of Parkinson patients admitted to a psychiatric hospital had some degree of depression, while two-thirds of them were diagnosed as having affective disorders.

The disablement and chronicity of Parkinson's disease understandably produce a depressive reaction, but there exists a significant group of patients where the depression is an endogenous one. Celesia and Wanamaker⁵ in a study of 153 patients found that one third had a form of depression. Warburton⁶ in a study of 140 patients referred for thalamotomy found that 60% of males and 18% of females had contemplated suicide and needed psychiatric treatment. In their review of the literature associating depression and Parkinson's disease Tune *et al* concluded that, although there is a disagreement as to the relationship of the severity of Parkinson's disease and the incidence and severity of depression, all the studies they review had documented a relationship between Parkinson's disease and depression. Some authors have used this relationship to support the catecholamine hypothesis of depression with

the locus coeruleus being implicated in both.⁷

In the preface to his classic monograph of 1817, Parkinson offers an apology that "mere conjecture takes the place of experiment", but he felt it warrantable in order to "excite the attention of those who might be in the best position to obtain more information about the malady". Stimulated by this pragmatic viewpoint, I felt supported in presenting my own personal experience of the illness.⁸ Though only a single case, I thought a study in depth might reveal useful clues about the illness in general. In this present paper, I propose briefly to explore the psychological consequences of bereavement in childhood, and to trace the connection through resultant endogenous depression to the emergence later on of the somatic condition of idiopathic Parkinson's disease.

The concept of significant life events in the pathogenesis of illness, including depression, is one which has been developed and researched by Brown *et al*.⁹ In *Depression and Loss* he underscores the significance of death of the mother, experienced before the age of 11 in the genesis and quality of subsequent depressive disorder. Quality refers to the profundity of an endogenous/psychotic depression in contrast to a reactive, that is, neurotic depression. The latter, less profound, Brown associates with the loss of a mother by separation rather than the finality of death.

My personal experience of such a life event was the relatively sudden death of my mother when I was 7 years old. Retrospective study revealed a failure to mourn, a pattern so frequently found in chronic depressive disorders. An aphorism attributed to Maudsley says: "The sorrow that has no

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vent in tears makes other organs weep."

Mourning and grieving are not readily experienced by children of that age, probably because the expectable psychological internalisation of parental figures is not yet sufficiently complete to allow the real parents to be relinquished, without overwhelming the labile ego. Psychological defences are automatically resorted to, including splitting and projection¹⁰ as the basis of magical thinking to deny the underlying sense of hopelessness. Another outcome of loss referred to by Deutsch¹¹ is complete inhibition of feeling.

A normal child makes use of its fantasy and symbol in order to deal with ordinary absence and separation from important people. The *actual* loss of mother during the critical phase of emotional development can have the effect of so stirring these fantasies, particularly aggressive ones, that they become replaced by inner deadness. In this way the child avoids the primitive fear of inward collapse and disintegrative abandonment. Growing up after such a loss, it may then proceed to restrict and sever affective links with people and to drain relationships of their meaning. It may further produce a super-adaptation to reality with a deadened imagination and destruction of feeling in order to obliterate pain. I subsume these effects under the rubric of "Emotional Flat Earthers", people whose limited imagination forces them not to let go for fear of falling over the edge or destroying the boundary of their circumscribed world. Engel¹² in discussing the mind-body relationship contends that it is a principal function of the mind to buffer the body by testing reality and modifying experience in such a way that a threat can be perceived as symbolic rather than existentially present. The paradigm for this buffering role, is of course the mother. She mediates the total physical and psychological needs of her infant in the pre-verbal symbiotic phase.¹³ This then moves increasingly into psychological mediation with the child differentiating itself from its mother and becoming an individual in its own right.

This developing mother-child interactive process has vicissitudes within itself, producing strains and failures. Failure leads to an activation of anachronistic flight/fight (manic) and conservation/withdrawal (depressive) categories of innate neuro-biological patterns. These in turn provide a necessary, if not a sole condition for the emergence of somatic disorders later on.

Somatisation can be defined as the visceral expression of emotional conflict. It requires no great leap of the imagination to move from depression to somatisation. The area of the psyche and the soma is highly complex, mental and physical, being inexorably bound, yet essentially different. There is not a

causal relationship between the two, but rather an analogical one.¹⁴ The psyche/soma functions as a whole with reverberations in both directions.

McDougal writes about a group of patients in whom there has been a failure in the infant-mother relationship, producing the syndrome, alexithymia (originally described by Nemiah and Sifneos¹⁵), an absence of affect which results from an inability to put feelings into words and to cope with unconscious fears, more akin to psychotic than to neurotic-anxiety.

In severely alexithymic patients, all awareness of affective pain or pleasure is eroded and a survival struggle is maintained against the experience of both pain and pleasure. The underlying disorder is masked and the most frequent presentation is in the psychosomatic realm. Marty, de Muzan, and David¹⁶ highlight the significance of what they call "operational thinking" as characteristic of the psychosomatic pattern. For example a patient when asked: "Were you upset when you ran over this woman and baby?" (killed in an accident) replied, "No, I am insured against third party accident". Unlike psychotic patients they remain closely adhered to facts and realities, but with marked lack of affect.

In psychiatric literature, the term psychosomatic is employed in three different ways: processes, reactions and diseases. Psychosomatic medicine may first deal with the psychological concomitant of physical disease, for example depression accompanying myxoedema. Secondly, the psychological reaction subsequent to organic disease may be seen for example in Parkinson's disease where the response follows the habitual way of coping with threatened debility. Perhaps the term somato-psychic is more appropriate. Finally "psychosomatic" is increasingly used in understanding the production of a disease by the interaction of psychological and somatic factors. This has barely been documented or explored for Parkinson's disease which is generally regarded in text books as a pure organic disorder.

Possibly because Parkinson's disease is predominantly an old persons' disease and because patients experience the illness in their sixties and on, not much thought is given to the pre-morbid personality and life-events. This is further compounded by the assumption that if the organic explanation exists, then no psychological factors need be sought. It is my view, based on personal experience, that there is a group of patients in whom the psychosomatic element in the origins of their idiopathic Parkinson's disease may be established. They will have had long-standing endogenous depression emanating from bereavement at an early age and/or failure of emotional development. That this view may or may not have helpful consequences in their treatment is

at present unclear. It should not impede exploration of this dimension in the same way as epidemiology and pathology are scrutinised and used as comparative studies. Barbeau has initiated a study of Parkinsonians in Canada whose illness commenced under the age of 40. Such a study should be carried out wherever the illness exists.

A helpful conceptualisation of psychosomatics that could be applied to further study of Parkinson's disease exists in the work of Ursin.¹⁷ The theory, built upon psychology, physiology, endocrinology and epidemiology addresses itself to the general standardised somatic response to psychosocial events. The profound changes resulting affect all endocrine and autonomic processes, brain function and muscle tension. This activation is the final common path for all phenomena that lead to higher activity in the central nervous system and persists until the problem is solved.

Activation in turn relates to pathology in two ways: normal short-acting activation may be too great a load for a diseased organ. The other aspect, more relevant here, is that sustained, long-lasting activation may produce somatic change. Activation depends on individual perception of the stimulus situation, the available response and previous experience with stimuli and responses. Processes that he identifies as defence and coping are decisive for resulting activation and the internal state of the organism. Activation is multi-varied and should be studied as such.

Individual variance is related to personality traits affecting defence and coping. Risk groups can be identified based on personality, somatic response and life situations. The theory explains why life changes are related to somatic disease, but only with low correlation. This is due to coping potential of Man. When this is exceeded, sustained activation occurs, which may result in pathology. Sustained activation is a good model for distressful events and chronic insoluble conflicts. Ursin quotes Weiner¹⁸ that disturbed circadian rhythms may have a role in producing psychosomatic disease. In his view, bereavement and distress are important for the development of psychosomatic disease. Finally, Henry and Stephens¹⁹ suggest that cortisol pathology, frequently seen in depression and distress may be related to defective immune response mechanisms.

The psychosomatic concept needs to be preserved against some misunderstanding and abuse. The psyche in the link is not conscious, volitional psyche, and as such is not directly under the control of the conscious mind. Therefore guilt and feelings of personal responsibilities for psychosomatic illness having its roots in early development are misplaced.

One hardly wants to drag idiopathic Parkinson's disease from an unambiguous organic basis to one within the psychosomatic fold in order that patients justify blaming themselves for the illness. This is a hazard to do with the wish for control in the face of growing helplessness that all patients experience.

In the prevalent multifactorial view of the illness, predisposition needs further clarification. Duvoisin's²⁰ twin studies offer evidence against the hereditary element in Parkinson's disease. Twin studies, however, cannot be used in the area of predisposition referred to earlier because, even if similar, the experience of twins *after* birth cannot be identical and must be personal. And it is precisely within the personal experience that the somatic has its roots.

The actual mechanism producing cell degeneration remains to be explored and understood. It has been found that striatal dopamine is already depleted by 50% or more before the clinical features of Parkinson's disease emerge,²¹ which points to a lengthy "incubation" of the disease. One might speculate on there being an upset in the autoimmune response as in the case of young diabetics and as has been suggested, as a link between some cases of cancer and depression.²² The clue as to why the striatum is affected in Parkinson's disease may lie in the recently-discovered association of peptide neurotransmitters of pain with basal ganglial cells.²³ Elliott, Jenner, and Marsden²⁴ postulate that the association enables a protective motor response to be made to painful stimuli. This pathway could well be utilised in the depressive withdrawal from psychosomatic pain of loss involving the sort of threat to survival described earlier in the paper.

The personality of the patient plays a vital part in the response to the established condition and its therapy. It has been said that we experience illness in the style that we live our lives. Failure to develop subtlety of emotion and modulation through experience will involve the individual in gross reaction patterns or sustained activation in Ursin's terms of fight/flight variety, with accumulation of anxiety and constant immersion in depression. The rigidity of personality and poverty of imagination which are frequently the hallmark of the group described is amenable to moderation in skilled psychotherapeutic hands, even though it is so assailed by the continuing illness in the form of a pathological somatic process.

Finally, this interactive view of psyche and soma is presented as a contemporary attempt to resolve the ever-present problem of the interaction of personality and the environment. It reminds us of the personal experience accompanying and influencing all behaviour and seeks to redress the balance in the

direction of understanding the person by the introduction of a developmental view of behaviour. It is offered as a real enrichment of our understanding the person, in this case the Parkinsonian.

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