SPECIAL ARTICLE

Sylvia Plath and the depression continuum

Brian Cooper MD FRCPsych

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Sylvia Plath, a gifted young poet, died by her own hand in London forty years ago. Subsequently a friend and fellow-poet, Al Alvarez, included a personal account of her final illness, as well as of his own unconnected suicidal attempt, in *The Savage God: a Study of Suicide*.[1] A review in the *British Journal of Psychiatry* by Eliot Slater, at that time its editor, concluded that Alvarez had failed to grasp the essential difference between his own depression and hers: 'His own experience', he wrote, 'so similar as he must think, does not help him to understand the act of Sylvia Plath; he is cut off by the gulf that separates the reactive from the endogenous depression, alike in appearance but different in kind. Sylvia Plath was a manic-depressive.'[2]

How well do these comments stand up today? Leave aside the fact that the two suicidal acts were not so different in terms purely of dangerousness, and that in each instance chance played some part in deciding on survival or death. The question remains of interest because it throws light on changing trends in the nosology and classification of psychiatric disorders, as well as on the psychology of a writer who since her untimely death has acquired iconic status both for the feminist movement and for a new literary generation.

Dr Slater's appraisal was based on a binary model of affective illness, in which the terms endogenous and reactive—or alternatively psychotic and neurotic—were used to denote two contrasting syndromes: the one consisting of recurrent severe disorders of mood (depressive and/or manic), apparently spontaneous in onset, the depressive phase being accompanied by psychomotor retardation, feelings of guilt and unworthiness, early-morning waking and somatic changes; the other presenting as milder, often intermittent depression mingled with anxiety, triggered by adverse life events, marked by irritability and self-concern rather than guilt and by subjective complaints rather than objective bodily disorder.[3] If one accepts this model, there is undoubtedly a strong case for allocating Sylvia to the former group. It was given support by John Horder, the respected London general practitioner (GP) who treated her in her last illness and who later wrote: 'I believe, indeed it was repeatedly obvious to me, that she was deeply depressed, ''ill'', ''out of her mind'', and that any explanations of a psychological sort are inadequate . . .'.[4]

In the intervening decades, however, the underlying assumptions of this model have been seriously challenged. To begin with, belief in a natural boundary or discontinuity between the main types of depression has been weakened by repeated failure to confirm a bimodal distribution, or to demonstrate any point of rarity between the subgroups, when the clinical features of unselected case series are submitted to discriminant function analysis.[5,6]

Secondly, there has been a corresponding failure to demonstrate clear-cut differences in the frequency of provoking stresses. Brown and his co-workers,[7] on the basis of detailed standardized assessments, found that depressed patients diagnosed as psychotic and neurotic, respectively, did not differ in experience of adverse life events over the months before illness onset. Indeed, age and experience of 'past loss' were the only personal characteristics that distinguished between them. When patients with and without a history of preceding severe life events were compared, only a slight tendency was found for the latter group to have more psychotic features.

Thirdly, US workers, bringing together evidence from various research fields, concluded that depressive illness represents the final common pathway of several different pathogenetic processes focused on the diencephalon, and is essentially the same whether the processes in question are biochemically, experientially or behaviourally triggered.[8]

These various findings, whose influence on psychiatric thinking became apparent when the standard classifications were last revised (see later), do not imply that individual cases cannot be characterized as typically endogenous or typically reactive. They do, however, suggest that the distribution as a whole corresponds to a dimensional rather than a categorical model, in which such pure-culture cases represent the extremes on a continuum and are likely to be greatly outnumbered by others presenting intermediate or mixed clinical pictures. Sylvia Plath's illness can be placed roughly on this continuum because information now in the public domain, including her posthumously published journals[9] and letters home,[10] a semi-autobiographical novel,[11] the memoirs of family and friends, a well-researched, sensitive biography[12] (one of several) and a life

Section of Old Age Psychiatry, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK

E-mail: spjubco@iop.kcl.ac.uk

of her husband,[13] together provide material for a reconstruction of the case history.

SYLVIA PLATH: A CASE HISTORY

In January 1963 Sylvia consulted her GP complaining of depression, and for the first time told him of a serious suicidal attempt she had made ten years earlier. The current episode had been triggered by a marital crisis, combined with some lesser life events, and had developed over six or seven months, during which time her emotional state had been one first of rage and bitterness, then of a prolonged alternation between hopefulness and despondency. For most of this time she had remained professionally active, writing daily, correcting proofs and recording poetry for the BBC. Now, however, her mood had deepened into a severe depression marked by constant agitation, suicidal thoughts and inability to cope with everyday life. Dr Horder prescribed an antidepressant (monoamine oxidase inhibitor), arranged to keep in daily contact and found a nurse to visit her daily at home. Having first tried to arrange a hospital bed at short notice, he then instead got her a psychiatric outpatient appointment.

Friends who at this time helped with the care of her two small children later described her as overwrought, 'hysterical' and intensely preoccupied with the breakdown of her marriage, which she blamed entirely on her husband's infidelity and the hostility to herself of his family and friends. She suffered badly from insomnia and early waking, relying on a hypnotic to get to sleep. Her normally rounded features had become gaunt and by her own account she had lost some 20 lb (9 kg) in weight, though she could still eat a meal with relish when it was cooked for her. Speech and movement were not retarded, apart from a loss of spring in her gait, and she continued to take great care over dress and appearance. She expressed no ideas of guilt or unworthiness, nor is there any evidence that she was deluded or hallucinated. In mid-February, shortly before her psychiatric appointment was due, she was found dead in her own kitchen, with her head in the oven and the gas turned on.

Medical and psychiatric history

Sylvia was born by normal delivery in 1932, a healthy 8½ lb baby, and a brother followed thirty months later. Her only childhood illnesses were measles and recurrent sinus trouble, and her subsequent medical history was uneventful apart from viral pneumonia at 25 and a miscarriage and removal of a grumbling appendix three years later. She had two children, born in 1960 and 1962 following uncomplicated pregnancies.

The previous onset of depression, at the age of 20, was associated with overwork and failure to get into a Harvard writing class on which she had set her heart. After inflicting gashes on her legs and talking of suicide she was referred to a psychiatrist and was started on electroconvulsive therapy (ECT), which seemingly convinced her that she was fated to become insane. Hiding herself in a small space under the house porch, she swallowed all her sleeping pills and lay in a coma for two days, until the family heard her groans. After hospital resuscitation she was transferred to the McLean psychiatric clinic, where she remained for four months, had modified insulin treatment, a second course of ECT and psychotherapy, and made a good recovery. In a further spell in psychotherapy with the same physician four years later, her emotional difficulties were explored in terms of an ambivalent relationship with her mother and the early loss of her father.

Family and social history

Mother and father alike were of Middle European extraction. Her father, Otto, was born in eastern Germany and emigrated to the US at the age of 16, while her maternal grandfather had come from Vienna and settled in Massachusetts. Both parents were intelligent, hard-working and socially aspirant; they shared, indeed, a veneration for work and self-betterment, for which they were willing to sacrifice material comforts and leisure activities. Otto took an MA degree in 1912, supported himself by teaching languages (he spoke five) while studying biology and zoology at different universities, received a doctorate in entomology from Harvard in 1928 and became a faculty member at Boston University. It is known that his German parents, who had followed Otto to the US, intended him to train as a Lutheran pastor and cut him off when he refused to do so. Aurelia, his wife, worked to pay her way through college, became a high school teacher, and two years after her husband's death was appointed to develop and run a college course for medical secretaries, also at Boston.

In Sylvia's eighth year, Otto, who had been ailing for some time, developed gangrene in one foot after minor trauma and was found to have late stage untreated diabetes mellitus. It transpired that he had neglected his condition because of a conviction that it was cancer. The leg was amputated but three weeks later, while still in hospital, he suffered a pulmonary embolism and died. The circumstances of his illness and death are strongly suggestive of depression, though neither he nor his wife had any recorded psychiatric history.

Sylvia was throughout her school and college career a consistently high achiever, conformist, joiner and prize-winner. She finished high school with straight As, won a scholarship to college, where she graduated *summa cum laude*, and at the age of 23 was awarded a Fulbright scholarship to Cambridge, England. There her life course

began to change when in 1956 she met and fell deeply in love with another aspiring poet, Ted Hughes, whom she married in the same year. Hughes, at that time much less conformist than his wife, disliked teaching and was determined to make his living as a freelance author. A year after being appointed to the faculty at her American *alma mater*, Smith College, Sylvia under his influence also decided to give up academic life and devote herself to full-time writing. The couple returned to England at the end of 1959 and their first child was born soon afterwards. At first their new life was cushioned by the award to Hughes of a Guggenheim Fellowship, but before long marital strains began to grow under the constant pressure to earn from writing, compounded for Sylvia by the care of two small children and her increasing jealousy. In 1962 she insisted on a separation after learning about an affair her husband had begun.

The appearance of a posthumous volume of verse, *Ariel*,[14] established Sylvia's reputation, and slowly the recognition grew that this young woman, whilst engaged in a life-and-death struggle with depression, had in the last months of her life achieved a literary breakthrough, producing some forty remarkable poems in an intense burst of creative energy.

Premorbid personality

Three features stand out in everything written by or about Sylvia that throws light on her personality. To begin with, there was a constant dissonance between the bright, buoyant, high-achievement persona whose ideals of success, social status and domesticity are conveyed in the letters to her mother, and the dark sense of isolation and inner emptiness that finds expression in her journals and poems. 'No matter how enthusiastic you are,' she wrote as a young student, '. . . nothing is real, past or future, when you are alone in your room', and later 'I look down into the warm, earthy world . . . and feel apart, enclosed in a wall of glass.'

Secondly, there was the emotional instability which Sylvia herself called 'ricochets' and others thought of as her mood swings. Academic or professional success stimulated her to spiralling activity; even small failures plunged her into dejection. The same mercurial quality affected all her personal relationships. Men who responded to her warmth were hurt and puzzled when they were dropped abruptly. With women friends she could change as swiftly from over-reliance to antagonism if once they disappointed her. She who adored her husband, writing with insight that 'He is better than any teacher, even fills somehow that huge, sad hole I feel in having no father', as soon as she suspected him of infidelity tore to pieces in jealous rage all his working papers and his precious edition of Shakespeare. Desperate as she was to save the marriage, the same burning anger undermined all her efforts at reconciliation.

Thirdly, though as a rule overcontrolled and unspontaneous, at times she would impulsively expose herself to physical harm, gashing her legs 'to see if I had the guts', skiing recklessly and breaking a leg, driving her car off the road—actions unexplained by a hypomanic state or by use of alcohol or drugs. She seemed, in the words of an old college friend, '. . . driven periodically to stage a symbolic salvation . . . almost as though only by being snatched from the brink of death could she confirm her worth'.[12]

THE DIAGNOSTIC PROBLEM

This potted case-history corresponds to the traditional stereotype neither of 'endogenous' nor of 'reactive' depression, but presents a mix of clinical features associated with both syndromes—the agitation, weight loss, insomnia with early waking and suicidal ideation, combined with the final deeply intractable mood change, are typical of an illness of the first type; whereas the obvious provoking cause, the early mood shifts through anger, anxiety and hopefulness, the demonstrative behaviour and blaming of others, all point to the second. Whether in the case in question any laboratory test would have helped to discriminate more clearly we cannot know, but in respect of depression generally such techniques are no more sensitive than symptom scales.[5]

The clinical profile can be quantified in dimensional terms by applying one or other of the, admittedly crude, rating systems which in those years were developed as aids to differential diagnosis. Thus on the Newcastle 18-item scale,[15] only four 'endogenous' and two 'neurotic' items can be scored (distinct quality of depression +1; weight loss of over 7 lb +2; early morning waking +1; previous similar episode +1; anxiety −1; blaming others −1), giving a total of 3 points, which is in the 'neurotic' range.

Scores on another system, the diagnostic index constructed by Kendell[5] (who himself subscribed to the unitary hypothesis) are set out in Table 1. In the large inpatient study on which this index was based, a total of 6 points would have lain below the median for cases of psychotic depression (mean +9.5), above the median for those with neurotic depression (mean −1.9), and almost on the median for the unimodal curve obtained for unselected depression cases. Sylvia's illness, in short, can be placed near the centre of a hypothetical depression continuum.

Categorical diagnosis in this field remains contentious. On the one hand, both ICD[16] and DSM[17] systems in their latest revisions have tacitly adopted a unitary model of depression. The term endogenous has been dropped, together with the psychosis–neurosis dichotomy, and reactive and reaction are retained only as alternatives to

Table 1 **Case of depressive illness (SP): known clinical features rated as psychotic or neurotic, in accordance with the diagnostic index score of Kendell (Ref. 5)**

Clinical features	*'Psychosis' weighting (positive score)*	*'Neurosis' weighting (negative score)*
Family history		
No firm evidence	0	0
Personal history		
Previous similar illness	+3	0
Previous serious suicidal attempt	+1	0
Previous subjective tension symptoms	0	−2
Aetiology and history of present illness		
Important precipitating causes (marital and social)	0	−5
Phenomenology of present illness		
Agitated	+5	0
Anxious	+1	0
Rapid mood changes	0	−1
Severe insomnia	+4	0
Subtotals	+14	−8

the preferred nomenclature. In consequence the question 'endogenous or reactive?' might seem no longer directly relevant, yet many psychiatrists remain unconvinced of this and continue to argue in favour of a binary model.[18]

For the mood disorders as a whole, on the other hand, both standard classifications still observe a basic division between bipolar (formerly manic-depressive) and unipolar conditions, assignment of the former label requiring evidence of both kinds of affective disturbance at different times. Some critics, however, now argue for a wider use of the bipolar rubric in connection with the use of so-called 'mood stabilizers',[19] and in DSM–IV the diagnostic criteria have already been loosened by introduction of a subcategory, bipolar II disorder, for cases with no florid manic episodes.

In the case of Sylvia Plath, a diagnostic formulation has to take account both of the clinical features of her second, fatal, depressive illness and of the nature of the underlying predisposition which rendered her so dangerously susceptible to depression. Paradoxically, the latter aspect has to be dealt with first, since if her upswings of mood are regarded as evidence of hypomania, the appropriate DSM–IV diagnosis for her major illness then becomes bipolar II disorder. Closer study of her life pattern reveals, however, not the spontaneously alternating mood-states of an underlying cyclothymia, but rather an overresponsiveness to daily experiences—a heightened reactivity to life's ups-and-downs, and above all to interpersonal tensions—that was the hallmark of her personality.

This character trait, along with her impulsive streak, the history of suicidal behaviour and physical self-harm, the episodes of rage and the deep-rooted ontological insecurity her journals reveal, taken together indicate a personality disturbance. Indeed, the whole profile suggests a variant of the heterogeneous group called 'emotionally unstable personality disorder, borderline type'[16] or simply 'borderline personality disorder',[17] which typically is characterized by over-reactivity, intense unstable personal relationships, identity disturbance, feelings of emptiness, impulsivity and risk-taking, frantic efforts to avoid abandonment, and recurrent suicidal or self-mutilating behaviour. Ill-defined though this category may be,[20] it serves to demarcate a group whose clinical features, prognosis and treatment needs are distinct from those of bipolar disorder.

In summary, the appropriate case-formulation would appear to be: recurrent depressive disorder, severe (without psychotic symptoms);[16] or alternatively major depressive disorder, recurrent,[17] in the setting of a borderline personality disorder.

DISCUSSION

The quality in a depressive illness that clinicians recognize as 'endogenous' often denotes an underlying susceptibility, but one not necessarily rooted in cyclothymia. In point of fact, the prevalence of bipolar disorder as currently diagnosed appears to be generally low and to account for only a small proportion of the major and recurrent depressions that are identified in field surveys.[21]

These conclusions matter for three reasons. First, accurate diagnosis is growing in importance with the emergence of new methods of treatment and strategies for

longer-term management, including serotonergic drugs, atypical neuroleptics and cognitive behavioural therapy. Secondly, there are implications for causal investigation. In respect of bipolar disorder, the evidence points to a strong inherited component, the lifetime risk being estimated at 40–70% in monozygous twins and 5–10% in first-degree relatives, compared with 0.5–1.5% in the general population.[22] In unipolar depression the evidence for a genetic effect is much less compelling and, whatever the root causes, wide national disparities in reported prevalence suggest that there must be important sociocultural factors.[21] For borderline personality disorder the most promising aetiological clues to date are of another order again, referring as they do to failures of parent–child attachment in early life.[23] Each line of inquiry presents its own challenges for future research.

Thirdly, there is, or should be, no gulf in understanding that cuts us off from the 'endogenously' as opposed to the 'reactively' depressed patient. The difficulty in empathizing is one of degree, not of kind, and probably has to do with the relative importance of provoking and predisposing risk factors—how far these have been identified by research, and the extent to which their presence can be recognized in the individual.

Sylvia Plath's case history conforms clearly to a psychosocial model, according to which a severe life-event in the presence of vulnerability factors (negative close relationships and low self-esteem) is highly predictive for depression in women with children, and the risk will be increased if there is a history of previous depressive illness, or of early loss or deprivation in childhood (though in Sylvia's case there is no evidence to suggest early abuse or neglect).[24] In epidemiological studies, moreover, the prevalence of depression will tend to vary with population levels of adverse life events, even if heritability coefficients for depression are substantial.[25]

Sylvia's illness and suicide have generated many articles in specialist journals, but almost all have been focused on issues of psychodynamic interpretation and have failed to deal squarely with the clinical history and diagnosis. Premature death being a strict censor, one can of course speculate that at some point, had she lived longer, she might have developed a manic psychosis. Indeed the view has been widely propagated that hers was a typical manic-depressive illness, and her picture has been featured in a popular science magazine alongside those of other such questionable candidates as Walt Whitman, Gauguin and Mahler.[26] On the evidence summarized above, however, that diagnosis does not seem warranted.

Careful diagnostic validation is important in this as in other cases, not least to evaluate the large claims made in recent years concerning the links between 'manic-depression' (loosely defined), the artistic temperament[27] and creativity,[28] and the extent to which these can be explained purely in terms of familial inheritance. In point of fact, creative artists have been the subjects of a wide range of psychiatric diagnoses, including depression, schizophrenia, alcoholism and drug dependency, and any excess risk they may have for such conditions seems at least as likely to be due to psychosocial as to genetic factors. So characterized have been the lives of poets, novelists, composers and graphic artists historically by ill-health, loneliness, penury, insecurity and lack of the normal social ties provided by a communal working environment, the wonder must be that many more have not become mentally deranged or committed suicide. Whatever hopes may be built on advances in genomic science, it is more likely to be through the study of early developmental influences, and the social milieu in which these operate, that we shall reach a better understanding of the relationships between psychiatric disorder and creativity.

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