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THE ANATOMY OF SORROW: A SPIRITUAL, PHENOMENOLOGICAL, AND NEUROLOGICAL PERSPECTIVE

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Abstract

There is considerable controversy, both within and outside the field of psychiatry, regarding the boundaries of normal sadness and clinical depression. Furthermore, while there are frequent calls for a "pluralistic", comprehensive approach to understanding depression, few writers have tried to integrate insights from the spiritual, philosophical, and neurobiological literature. The author proposes that such a synthesis is possible, and that our understanding of ordinary sorrow and clinical depression is enriched by drawing from these disparate sources. In particular, a phenomenological analysis of sorrow and depression reveals two overlapping but distinct "lifeworlds". These differ in the relational, temporal, dialectical, and intentional realms. Recent brain imaging studies are also beginning to reveal the neurobiological correlates of sorrow and depression. As we come to understand the neurobiology of these states, we may be able to correlate specific alterations in "neurocircuitry" with their phenomenological expressions.

1. Introduction

The field of psychiatry has always sought to incorporate insights from disciplines outside the realm of biology, notwithstanding the widespread notion that "biological psychiatry" is now the field's dominant paradigm. To be sure, recent advances in neurobiology–particularly in the area of mood disorders–have cast a bright light on the molecular and neurochemical bases of psychiatric illnesses.

To some degree, this has come at the expense of other modes of understanding. Indeed, some have upbraided modern-day psychiatry for ignoring the psychological, social

and spiritual dimensions of emotional disorders. These attacks, in my view, distract us from the overriding task of integrating biological discoveries with a broader philosophy of emotional dysfunction. Insights from both the Western and Eastern spiritual traditions can help illuminate important aspects of ordinary sadness and pathological depression. A phenomenological analysis of these mood states can further enrich our understanding. Ultimately, I believe that a pluralistic view of mood disorders will aim at "mapping" experiential aspects of depression, such as hopelessness or self-deprecation, on to specific areas of brain dysfunction. In this paper, I try to provide a broad outline of such an integrated understanding of mood.

2. A brief spiritual history of sorrow and depression

Psychiatrists and psychologists are hardly the only ones who have recognized the difference between clinical depression and "normal" sadness or sorrow. The distinction seems to be as old as recorded history. Surprisingly, in the Old Testament, the figure of King David presents us with portraits of *both* severe depression *and* normal bereavement. In Psalm 38,conventionally ascribed to David, the psalmist is lamenting his sins. He tells us that "There is no soundness in my flesh...no health in my bones because of my sin...my wounds grow foul and fester because of my foolishness, I am utterly bowed down and prostate; all the day I go about mourning...I groan because of the tumult of my heart." [1]. Modern diagnosticians would see in this description a picture quite consistent with an episode of major depression. In contrast, after the death of his beloved friend, Jonathan, the very same King David is far from "bowed down and prostrate". Rather, after a brief period of weeping and fasting, David is moved to write a passionately stirring dirge, known as "The Lament of the Bow" (2 Samuel 1:17–27), addressed to his lost friend: "How have the mighty fallen...I grieve for you, my brother Jonathan, you were most dear to me..." [1]. There is no trace, in David's lament, of the self-loathing and bodily decay found in Psalm 38. David's period of mourning after Jonathan's death represents roughly what modern-day mental health professionals would call "bereavement"–not clinical depression.

That life brings with it certain unavoidable or at least "expectable" sorrows is a concept found in Eastern religious thought, as well. In Buddhism, for example, we are told there are two roots of unhappiness in human existence: *dukha*and *tanha*. Dukha comprises the "...inevitable occasions of unhappiness" that come with human suffering, frailty, disease, loss of loved ones, and of course, death. Then there is *tanha*, which is translated as "blind

demandingness": that part of our nature "...which leads us to ask of the universe...more than it is ready or even able to give." [2] Very roughly, we can see the precursors of normal and pathological sadness, respectively, in *dukha*and *tanha*.

Similarly, the 14th century monk, Thomas Kempis (1380– 1471) recognized that sorrow is sometimes appropriate. "Levity of heart and neglect of our faults," he wrote, "make us insensible to the proper sorrows of the soul." [3] Thomas asks, "Is there anyone who enjoys everything as he wishes? Neither you, nor I, nor anyone else on earth. There is no one in the world without trouble or anxiety, be he King or Pope."[3] Indeed, like many medieval theologians, Thomas saw this earthly existence as a vale of tears. He believed that, "...we often engage in empty laughter when we should rightly weep." [3]

Four centuries after Thomas Kempis, several Hassidic masters also distinguished between normal and abnormal degrees of sorrow. Rabbi Levi Yitzchak of Berditchev (1740–1810) wrote,

> "There are two kinds of sorrow...When a man broods over the misfortunes that have come upon him... [and] cowers in a corner and despairs of help–that is a bad kind of sorrow..." In contrast, "...the other kind is the honest grief of a man who knows what he lacks." [4]

"For it is a good thing to have a broken heart, and pleasing to God, as it is written: 'The sacrifices of God are a broken spirit...' [Psalm 51:19]...God does not entirely heal those who have broken hearts. He only eases their suffering, lest it torment and deject them. For dejection is not good and not pleasing to God. A broken heart prepares man for the service of God, but dejection corrodes service. *We must distinguish as carefully between the two as between joy and wantonness..."* [[4], p. 115, italics added].

Surprisingly, Rabbi Bunam seems to have foreseen not only our distinction between normal grief and clinical depression, but perhaps also that between *normal joy* and *hypomania* or *mania* ("wantonness").

Of course, it is not always easy to tell "proper sorrows" from intense grief, "pathological" grief, or clinical depression. Indeed, it is very doubtful that these are strictly delineated categories. Furthermore, the nature of the putative "cause" or precipitating event is not a reliable predictor of where, on this emotional continuum, a given individual may end

up. The loss of a loved one, for example, ordinarily provokes sorrow and a finite period of grief and mourning. Most mourners do not develop a severe, intractable clinical depression. Indeed, in the Judaic tradition, it is expected that after the seven days of mourning known as *shiva*, the bereaved will generally be ready to resume some "everyday" activities (while refraining, however, from any kind of celebration) [5].

There are, of course, many exceptions to the generally selflimited course of mourning; in principle, there are as many kinds of mourning as there are mourners. The great medieval philosopher, Moses ben Maimon (Maimonides, 1135–1204), appears to have developed a profound and prolonged depression, after the death of his beloved brother, David, in a shipwreck. Maimonides writes, in a letter dated from 1176,

"On the day I received that terrible news [of David's death], I fell ill and remained in bed for about a year, suffering from a sore boil, fever, and depression, and was almost given up. About eight years have since passed, but I am still mourning and unable to accept consolation...all joy has gone...whenever I see his handwriting or one of his letters, my heart turns upside down and my grief awakensagain." [6]

3. The biology of sorrow

If sorrow, bereavement, pathological grief, and major depression are distinguishable clinically and phenomenologically, we might hypothesize that they also differ *biologically*. This might be investigated from two perspectives. On the one hand, we might regard these mood states not as discrete categories, but as conditions along a *continuum* or *spectrum* of dysphoric mood and impaired function. Ghaemi, for example, posits a "unipolar depressive spectrum" that distinguishes *acute* from *chronic* major depression; and *single* from *recurrent* episodes of major depression [27]. (The terms "continuum" and "spectrum" are often used interchangeably in the depression literature. Technically, however, a *continuum* denotes a *progression of values* or elements varying by minute degrees, such as blood pressure readings (120/80, 121/81, 122/82, etc.). A *spectrum* denotes an ordered arrangement by a particular *characteristic*, such as a spectrum of visible color wavelengths. For our purposes, however, this distinction is not critical). Based on the spectrum-continuum model, we might hypothesize that mood states and disorders would yield *subtle gradations* of biological differentiae, rather than black-and-white distinctions.

On the other hand, we might posit a *categorical* separation of mood states. The now outmoded "reactive" (exogenous) versus "endogenous" distinction is one example of a categorical classification of depression (though, in theory, one could envision subtle gradations of "endogenicity"). If we posit such a categorical separation of mood states, we might also hypothesize *binary* or *dichotomous biological differentiae*; e.g., "abnormal" versus "not abnormal" laboratory values. Indeed, Taylor and Fink have proposed that *melancholia* is a biologically distinct subtype of depression, characterized most notably by abnormal elevations of serum cortisol [28].

While it is too early to decide which model – *spectrum-continuum* or *categorical* – more accurately corresponds to "reality", we continue to amass data on the neurobiology of dysphoric mood states.

For example, the "neurocircuitry" of *major depressive disorder* has been investigated by several groups [29]. Although a detailed review of this voluminous literature is beyond the scope of this paper, the most robust and reproducible finding using positron emission tomography (PET) is that of *decreased metabolic activity in the frontal lobes*. Conversely, a return to normal frontal lobe activity is associated with improvement in the patient's depression [29]. Many other brain regions, including various subcortical and limbic areas, also show abnormalities in some studies of major depression.

Unfortunately, the neurocircuitry of *normal and spontaneous* sadness, sorrow, or grief has received considerably less study. Preliminary data suggest both regional similarities and differences between normal sadness and clinical depression. However, since most studies have involved some method of "sadness induction" using visual images or verbal cues [30], inferences regarding *spontaneous*, "everyday" sadness or grief are probably premature.

One partial exception is an intriguing study by Najib et al [31], which assessed nine women whose "romantic relationship" had ended within the preceding 4 months. Subjects were scanned using functional magnetic resonance imaging (fMRI) while they alternated between recalling a sad, ruminative thought about their loved one (acute grief state) and a neutral thought about another person they knew an equally long time. Acute grief was associated with *increased activity* in *posterior* brain regions, including the cerebellum, posterior brainstem, and posterior temporoparietal and occipital brain regions. In contrast, more *anterior* regions, such as the orbitofrontal cortex, showed *decreased* activity. Strikingly,

the higher the subject's baseline level of grief, the greater the decrease in anterior brain activity. Some of these findings overlap with PET studies of depression [29]. However, in the Najib et al study, *acute grief* was associated with *decreased activity in the amygdala*, whereasmost studies of *depression* have found *hyperactivity* in the amygdala [31]. Replication of this last finding in spontaneous states of sadness or bereavement might point to different neurobiological substrates, compared with depression.

In this regard, it is interesting that–in the author's experience – severely depressed individuals who have recovered or achieved remission with antidepressant therapy consistently report the ability to experience ordinary sorrow or sadness. This might be interpreted as reflecting differing neurobiological substrates for major depression and ordinary sadness. On the other hand, some research finds that, in patients who meet DSM-IV criteria for both major depression *and* bereavement, response to an antidepressant [bupropion] produces a *concomitant reduction in both depressive symptoms and intensity of grief* [32]. This could be consistent with some degree of "biochemical overlap" between depression and bereavement, in those who meet criteria for both conditions. However, this does not rule out neurobiological differences between those with *depression alone* versus those with *bereavement alone*.

Further support for the biological separation of normal sadness and clinical depression comes from very recent research on *deep brain stimulation* (DBS) in extremely refractory cases of major depression. DBS entails the implantation of a tiny device called a "brain pacemaker", which sends electrical impulses to specific parts of the brain. DBS has been approved by the U.S. Food and Drug Administration for use in the treatment of Parkinson's disease and other movement disorders [33]. A small pilot study by Mayberg and colleagues [34] found that chronic deep brain stimulation (DBS) of the *subgenual cingulate* region (Brodmann area 25) resulted in "a striking and sustained remission of depression" in four of six patients with very resistant depression. All patients met DSM-IV criteria for major depression, and all had failed to respond to at least four treatments for depression (medication, psychotherapy, or electroconvulsive therapy). It is noteworthy that, in an attempt to control for placebo effects, the researchers performed a "blinded discontinuation" of the DBS in one patient who had experienced an early and robust response to treatment. After a period of about a month–and despite sustained euthymia (normal mood) on the Hamilton Depression Rating Scale–the patient began to exhibit a progressive decrease in

energy, initiative, and concentration. When the correct stimulation frequency was restored (with the patient still "blinded" to the procedure), the patient's energy, initiative and concentration returned to pre-discontinuation levels within a week.

Dr. Helen Mayberg, considering the available neurobiological data, has opined that, "...we don't know how sadness/grief fits into the continuum of major depression... [however] the notion of normal circuits in a state of dysequilibrium is at least tenable and the imaging data supports it." (H. Mayberg MD, personal communication, 5/ 08/08). Moreover– notwithstanding the preliminary state of the evidence–the hypotheses developed in this paper allow us to generate a number of empirically testable predictions. For example, I would predict that among depressed individuals who experience *severe distortions in the relational, temporal, dialectical, and intentional realms*, we are likely to find (a) a higher frequency of *treatment-resistant depression*; and (b) a higher frequency of markedly *abnormal findings on fMRI and PET imaging*. If such predictions are borne out, this may have important treatment implications. For example, severedistortions in the phenomenological realm may someday point us toward especially effective neurobiological or psychosocial interventions. In the mean time, the hypotheses developed here might encourage researchers to develop semistructured interviews or rating scales, aimed at quantifying pathology in the phenomenological realm.

4. Conclusion

Though our mythic and literary heritage depicts ordinary grief and clinical depression as more or less discrete existential categories, it seems more likely that these conditions lie along a complex spectrum or continuum of dysphoric states. Moving from less to more severe, we may distinguish *normal sadness or sorrow; normal grief; complicated (pathological) grief*; and *major depression* as gradations along this continuum. Though this continuum may be characterized by very subtle gradations, both clinical and phenomenological features can help us distinguish normal sadness from severe, clinical depression. The syndrome of "complicated grief" (pathological mourning) may serve as a conceptual and phenomenological bridge between ordinary sorrow or grief, and major depression.

That said, both the *components* and *boundaries* of such a proposed continuum may be subject to debate. For example, should we exclude states of "normal sadness" and simply

consider more incapacitating dysphoric states? And can we ever express, in objective terms, the subtle gradations and almost endless range of human emotional states? Certainly, the continuum proposed here should not be reified or made into a rigid instrument of classification; it is, at best, a heuristic tool in service of understanding the patient.

However we answer these questions, I believe that an understanding of the phenomenological "lifeworld" of the patient [36-38] must be incorporated into pluralistic models of depression. In time, we may come to understand how the phenomenology of depression and "proper sorrows" relates to their neurobiological substrates. Indeed, I believe that a full understanding of sorrow and depression will synthesize insights from spiritual, phenomenological and neurobiological perspectives.

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