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From the Executive Director

The Case Against Depression

The wife of a 42-year old physician pushes him to call for an urgent appointment. He is described as suicidally depressed, has rejected all offers of help, but agrees to see me later that day. Within minutes of beginning our initial session he described anxiety that had increased steadily over a 3-month period. A nearly fanatical platform tennis player, he had been sidelined by persistent, gnawing shoulder pain that his orthopedist claimed would remit were he to agree to the arthroscopic surgery he had been avoiding for 6 months. "I can handle pain easily, but this damn anxiety just won't quit. It eats away at me day and night and I've begun to think about killing myself. Do you think I have to be hospitalized for depression?" The chronic and worsening anxiety responded nicely to fairly low bedtime doses of the benzodiazepine *clonazepam*. Free of the crippling anxiety, he had no further thoughts of suicide, began to deal with his secret fear that he might succumb to the same rheumatoid arthritis that had constrained his mother to a wheelchair since his own childhood, and scheduled the relatively minor operation that eventually returned him to the level of competition he had enjoyed a year earlier. Through the course of our work he took a total of 11 tablets of medication.

The 82-year old father of a friend began to withdraw from all social activity and sat slumped in a chair staring at the wall "I can't remember things people tell me," he complained briskly with some asperity as his wife nodded in assent. "Things get into my ears and don't stay in my head. But I can remember things that happened 30 years ago as if it were yesterday. And I've lost my ability to read." And, in a voice so low it was almost inaudible, "I feel so unworthy. For so many years I've fooled people, but now I can't fool them any more." All at once he began a sordid recital of infidelities committed years earlier, as if in hope that disclosure to his wife of these shameful secrets might reduce his pain. A full workup for dementia demonstrated far better cognitive and memorial function than one might have expected, while a battery of psychological tests suggested that he had a psychotic depression. Hospitalization, a wide range of psychotherapeutic interventions, and a wide variety of biological and somatic treatments were equally ineffective. A year later I still see this now-wizened old man in the neighborhood of my office, walking helplessly in the company of his vigorous wife, who can barely reconcile herself to the idea that some depressions resist all known treatment.

It is hard to avoid information about depression. DSM-IV lists more than a dozen forms of depressive illness and cites depressive complications of many other conditions, while nearly every day one of our journals presents an article about some new aspect of depression. Only the studiously avoidant remain unaware of the degree to which this cluster of discomforts is assigned biological causality; studies of blood flow and cerebral metabolism using brain imaging techniques of the greatest sophistication present more and more data about the neurobiology of depression even as more and more classes of medication are shown to relieve its symptoms. Intense debate swirls about the relative efficacy of therapeutic protocols based on psychopharmacology, cognitive theory, and psychoanalysis. There is even a journal called *Depression*.

And yet, with all this attention to what by now must be one of the best studied disease complexes in the history of psychiatry and psychology, I argue for the removal of the term from our nomenclature and its replacement by language more attentive to the affective experience actually involved. The system I propose would require a statement denoting the specific affects being experienced by the patient on a chronic basis. The first case presented might bear the label "Persistent Dysphoria; fear-terror with secondary distress-anguish; somatic precipitant" while the second might be "Persistent Dysphoria; inhibition of interest-excitement, cause unknown; possible biological shame syndrome." Diagnosis might be based on the affect pattern chart introduced at the 1993 meeting of the Tomkins Institute. In a subsequent editorial I will discuss the use of this chart in clinical work.

If you listen carefully to the complaints offered by patients, it becomes clear that one may be mildly depressed or severely depressed for a few moments or for what seems like a lifetime or intermittently at varying degrees of severity. When depressed, one may shun contact with others or crave it incessantly. Some who suffer depression seek relief in hedonism, while others find pleasure nowhere and still others seem "driven" to suicide. Melanie Klein, who more than any other psychoanalytic pioneer viewed the infant as a miniature adult, found evidence of a "depressive position" from birth. Where once there was a sharp distinction between symptom complexes characterized by the presence of depression or anxiety, now we see chronic anxiety being treated with antidepressants and chronic depression treated with antianxiety agents. Terms like "hysteroid dysphoria," "classical depression," and "atypical depresssion" are used to explain differences in symp-tom patterns as well as response to medication. Some schizophrenics seem to benefit from treatment with antidepressants, while non-schizophrenic patients with "delusional depression" fare better with antianxiety medication. In contrast, the cognitive-behavioral treatments for panic disorder and depression differ little.

The emergence of pharmacological agents capable of relieving the symptoms of some depressions but not others, or treating the depressive symptomatology of some patients but not the apparently identical complaints of others, should have suggested that more was wrong with our language than our medications. Of equal interest is the common observation that successful treatment with antidepressants relieves symptoms about which the patient had not complained, as well as symptoms rarely considered

part of the depressive spectrum. Kramer, whose *Listening to Prozac* opened this debate to a mass audience, adapted a paranoid style for hortatory purposes and suggested that the use of medication to improve the lives of people who did not have a recognized "disease" could be considered "cosmetic psychopharmacology" and perhaps improper.

At issue here is the search for simplicity rather than the careful analysis of complexity. If, in the early days of psychoanalysis, all emotionality was to be explained on the basis of drive forces, then some simple, hydraulic mechanism had to account for depression. A depressed person had no "energy" to challenge life because the energy was being shunted somewhere else. Thus, anger thwarted or turned inward seemed an adequate schematic diagram for the energies of depression, and the search for ways of releasing that anger a reasonable therapeutic technique. By analogy, the medical approach to depressive illness has fostered a search for the ultimate antidepressant that will relieve all symptoms in all sufferers. The success of each new drug has led to its use in ever-widening segments of the population, followed first by steady decreases in the fraction of patients who achieve benefit (thus throwing that approach into disfavor) and then by renewal of the sturdy hope that the next drug will cure everybody. So thoroughly has psychoanalytic logic infused our culture that the concept of depression has remained unitary despite the mounting evidence that the term itself is a collective noun, a wastebasket into which are thrown a wide range of dysphorias.

The classification I have proposed would take note of the fact that six of the nine innate affects defined by Tomkins cause decidedly unpleasant experiences. By definition, all of the affects are brief, lasting a few hundredths of a second in the case of surprise-startle, and a couple of seconds in the case of distress-anguish and anger-rage. Our awareness that an affect has been triggered is called by Basch a feeling (this is the moment that the biology of affect turns into psychology), and the association of an affect with previous experiences of that affect is called an emotion (this is the interface between biology and biography). We use the term mood to speak of the internal loops through which affect and memory reinforce each other to produce the relatively continuous experience of any emotion. Through this mechanism, any negative affect may be experienced over a considerable period of time. If the relatively constant overload of work triggers distress-anguish that reminds me of what made me sob last year, and the rekindling of that remembered distress acts as an additional stimulus load making me feel even more like crying, and the accumulated memories pile up to the extent that I begin to sob at television commercials simply because they trigger enough affect to make the protagonists matter to me, then I may be said to be in a mood characterized by fairly constant distress-anguish.

Persistent distress-anguish is often called sadness, persistent fear-terror is known as steady anxiety, persistent mild anger-rage is irritability or annoyance, persistent anger-rage at a higher density is thought of as being in a bad temper. Persistence of the affect shame-humiliation, when experienced as the *withdrawal* pole of the compass of shame, is a state of loneliness, hurt feelings, and bad thoughts about the self; when admixed with fear-terror it may be called guilt. (When angry, parents adjust our behavior by producing guilt and shame in us; any psychosocial or biological disorder that produces enduring guilt or shame will be experienced as "anger against the self" despite that these emotions have been produced by distinctly different mechanisms.) The bad moods associated with dissmell and disgust make us keep others at a distance and promote a wide range of interpersonal styles when paired with other negative affects.

There are, then, six families of bad mood, each of which when dense is likely to be described by its subject as depression. To those trained in affect theory, the persistent experience of shame is treated quite differently from the persistent experience of distress; for shame we investigate impediments to the positive affects of interest-excitement and enjoyment-joy, while for distress we try to reduce steady-state stimulus load. Both treatments are "antidepressant." Just as in a previous era we tended to use the term "psychosis" only when we were humiliated by our inability to get the patient to accept our arguments and renounce one or another group of scripts, we now think of a patient as "depressed" when conventional therapy does not remediate persistent negative affect. Despite the severity of the symptoms involved, the case presented in the opening paragraph of this essay is an example of normal mood in that it really did involve an internal loop initiated by a discrete stimulus triggering normal affect that became involved with an internal script from which the subject could not extricate himself.

I do not mean to imply that treatment illuminated by affect and script theory is intrinsically easy. For example, every once in a while we see a patient whose chronic distress-anguish is a reaction to the steady experience of fear-terror; here it is the steadiness of the primary affect that then triggers a secondary affective reaction so that chronic anxiety is thus fused with distress. Despite the adequacy of their vocabulary for other matters, most people have few words with which to describe their feelings and lump such fusion products under the rubric of depression. (Often we are required to teach people the language with which they can become accessible to therapy.) In the example just cited, it seems only logical to expect that such a patient might experience relief from an antianxiety agent and feel much worse when given an antidepressant; the verbal psychotherapies might also work better were this situation recognized at its affective roots. I suspect that a cognitive therapist, by training less committed to the search for sources of affect stemming from infantile experience than her psychoanalytic colleagues, might trace distress backwards to anxiety and straightaway develop a systematic treatment for the underlying anxiety which therefore relieved the distress. This, naturally, would be considered successful therapeutic management of depression. If I am correct that the steady experience of any negative affect will be described as depression by anyone naive to the nomenclature of innate affect, then attention to the nature and source of any negative affect is antidepressant.

Sometimes, of course, the steady experience of negative affect is the result of aberrant neurochemistry; emotionality, no matter where placed on the yardstick of normality, must involve hardware as well as firmware and software. Tomkins suggested that the six basic innate affects evolved as amplifiers of simple qualities—increases and decreases in the levels or gradients of stimulation. Each affect amplifies a quite different stimulus condition, and amplifies it in a highly specific, discrete, and entirely memorable way. To the three basic negative affects (fear-terror, distress-

anguish, and anger-rage) he added two discomforts that evolved as modulators of the drive hunger (the drive auxiliaries dissmell and disgust) and a final mechanism (the affect auxiliary shame-humil-iation) that amplifies any impediment to the two positive affects of interest-excitement and enjoyment-joy. The firmware, of course, is the operating programs for each of these six negative affects, stored as subcortical action scripts dependent on intricate networks of neural pathways leading to activity at specific sites of action. All of these pathways comprise what I have defined as structural effecttors and are dependent on chemical mediators that transmit messages along their course toward the actions we come to know as our affects. The system is so complex, its subunits so intertwined, its mechanisms involving so many layers, that there are lots of places for something to go wrong.

Here, I believe, is the reason there are so many different biological treatments for "depression," all of which work in some despite how inadequate they may be for other patients whose complaints and history seem identical. Even though we have come to understand the family of medications called antidepressants in terms of their effects on the neurotransmitters noradrenalin, serotonin, and dopamine, the action of some therapeutic agents seems unrelated to these compounds. I suspect that antidepressants work when they repair some part of the circuitry for the triggering, maintenance, and cessation of any affect. No one has as yet investigated the biology of any innate affect, for such is the mind set of the contemporary neurobiologist that modern science searches for general solutions to the problem of disturbed emotionality rather than specific solutions to the problems caused by malfunctions in individual affect circuits. I have suggested that the serotonin reuptake inhibitors repair certain defects in shame biology, that the phenothiazines remediate some aspect of fear-terror, and that most tricyclic antidepressants are effective when symptomatology is canted in favor of the peculiar fusion of shame and fear known as guilt. *Buspirone* is highly specific for one form of the serotonin receptor. A much-advertised but largely ineffective remedy for anxiety when used in people who have never tasted a benzodiazepine, it is now touted for its ability to reduce "anxiety" in people whose "depression" is reduced by *fluoxetine*, *sertraline*, or *paroxetine*. If this therapeutic approach turns out to be valid, it suggests that certain aspects of the complex affective patterns presented by these patients may be traced to specific variations of the serotonin-related neurotransmitters. But there is no hope of explaining such effects in the absence of a clearly defined language for normal affect and a consequent redefinition of the broad and confused language for emotion based on the description of illness.

This is the problem presented by a language of science that confuses the effects of endorphin (endogenous morphine-like compounds) with the affect enjoyment-joy. The innate affect is triggered any time stimulus density is reduced, and is associated with highly specific facial displays. Gradual reduction in stimulus density is posited to trigger a smile, rapid reduction a laugh. Tomkins viewed endorphin as another, quite separate system of amplification also capable of creating the good feelings associated with enjoyment-joy but for different reasons under different circumstances. The amplification systems we know as our affects are formed in layers that have accreted through the long process of evolution. Some of these layers involve the innate affects described by Tomkins, and others do not. Disorders of mood can occur when there is something wrong in any layer, even though we humans are likely to describe any emotional experience as if it had been an affective response to a known and discernible trigger for ordinary innate affect simply because that is how we know our affects. The case sketched in the second paragraph of this essay involved a disorder of mood in which the affect interest-excitement was shut down by biological mechanisms we still do not understand and for which we have no treatment. The symptom complexes presented by patients with dementia of the Alzheimer's type stem from primary lesions of memory storage and retrieval that make one poorly able to live in the world; yet these cognitive losses also involve the scripts for the modulation of affect, often making the patient "irrationally" emotional.

Only when we begin to investigate the psychopathology of emotion from the standpoint of normal affect mechanisms can we integrate the texts of neurobiology into the encyclopedia of psychotherapy. It is the affect and script theories of Silvan Tomkins that facilitate this new approach, and the best place to start would be with the replacement of the term "depression" by language both more precise and more useful.

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