Suicide Explained

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A Neuropsychological Approach

Federico Sanchez

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In loving memory of my son Mitchell Xavier

Nov. 23, 1979-Nov 12, 2002

A Man for All Seasons

Front cover: The image is a detail of the painting *Mexican Mythology* (1.60x1.60 meters) by Jorge Gonzalez Camarena, in the author's collection. The gods of the earth appear from top to bottom: Presiding over these powers is Tlaloc, master of the rains and water, represented by two interlocked serpents, whose heads, as they join sideways, form the mouth or upper lip. Immediately below, is Xipe Totec, the maintenance god, the god of vegetation, of seeds and of sowing. He is covered with the skin of a flayed human being, symbolizing the renovation of plants that cover the earth every year. Lower still, is Mictlantecuhtli, god of the regions of death where the souls of the dead dwell. At the bottom is Coatlicue of the serpents' skirt who gives and takes away life, represented by the breast that gives sustenance, and at the same time, with a handful of dead bodies she is the devourer of men.

Back cover: the author in front of the *Mexican Mythology* showing a different detail, to the right, Tezcatlipoca, the wicked god of the night and of the powers of evil, whose symbol is the moon and his guise is that of the tiger, and Quetzalcoatl, the creator of culture, and generically speaking the god of goodness, whose symbol is Venus. Tezcatlicopa and Quetzalcoatl, unable to agree on the creation of the human species, originate the cyclical battles in which they alternate as winner and loser, representing the eternal struggle between good and evil. Here, Quetzalcoatl is being defeated as he dons the mask of Ehecatl, the god of the wind. To the left of the author, we see Xiuhtecuhtli, the god of fire, also known as the god of ancient times, whose place in the Sun was taken by Huitzilopochtli, the god of war, supreme god of Tenochtitlan and the Aztecs.

Introduction to Aftoktognosis (Knowledge of Suicide)

The road from suicide to life is cold and colder and colder still, but—with steely effort, the grace of God, and an inevitable break in the weather—I could make it."

Kay Redfield Jamison, *Night Falls Fast*

The younger of my two sons, Mitchell Xavier, committed suicide shortly before his twenty-third birthday. He was diagnosed incorrectly a few times, once as obsessive-compulsive, and another time as schizophrenic. Three other doctors diagnosed him, in my opinion, correctly: moderately depressed with occasional panic attacks. The professionals that saw him all concurred that he was not suicidal. He was bright, good looking, a great athlete, loved by his family and friends, recently graduated from college, with a whole array of future opportunities at his feet—his death took me by surprise even though I was aware of a 20 percent incidence of suicide among depressed people.

In the state of Virginia alone, between 1999 and 2004, on average, there were about 950 hospital discharges each year from self-inflicted injuries. There were 1,284 self-inflicted injury hospitalizations for 10-24 year olds in 2004 for a rate of 81.7 per 100,000. The average length of hospital stay was 3 days and the median charge for an episode of care was $5,288. Medical costs resulting from hospitalizations in Virginia due to self-inflicted injuries total over $40 million per year.[1] These figures do not include completed (successful) suicides.

The figures for the whole United States indicate about 500,000 suicide attempts a year. Research suggests that social stigma leads to inaccurate reporting; thus, these figures may not indicate the full extent of suicide attempts and completions. Added to the loss of life from completed suicides and the financial burden that results from suicide attempts is the tremendous emotional toll on the health and functioning of families and communities imposed by the special suffering experienced when young people die.

My experience with psychiatrists, when we seeked help, seemed to show that they couldn't be of much assistance. As a consequence, I didn't stress seeking help from

[1] *Youth Suicide Prevention, Data and Statistics,* Virginia Department of Health. Updated March 21, 2007.

them. In that, I was wrong. Yet, Mitchell and I were not mistaken in intuiting how limited their knowledge was. I had promised him that in time we would find answers to his questions: why panic attacks occur, or why depression happens. Shortly after my son's death I set out to write a book that would answer these questions and also explain why suicide happens, in part to fulfill the promise I had made him, in part to answer my own, overwhelming questions. Almost three years later, a good friend of mine, newly married, just a few years older (29) than my son when he died, attempted suicide by ingesting his recently prescribed antidepressant pills, but luckily was discovered and rushed to the hospital. The book I had written,[2] even though it explains why suicide occurs, was too long and too personal to be of immediate help to him or his wife, at least in the short term. This book is written to address that deficit and hopefully provide some explanations quickly and concisely.

Can we understand suicide?

I feel that we have enough knowledge today to answer, "Yes, in most cases, though perhaps not in all."

Can we accept the self-inflicted death of a young person?

"Definitely not."

Can we prevent suicide?

"In many cases, yes."

Can we predict suicide accurately?

"Mostly not, and this is the area where much improvement could be done."

Can the number of suicide attempts or deaths be reduced?

"Yes, particularly if more focused programs, based on the insights we have learned, educate a broader group of people on the general problems of mental health and suicide in particular."

Can we explain suicide?

"Yes."

This book looks into all these questions in detail. This is a book on aftoktognosis, a word I coined from the Greek aftoktonia = suicide and gnosis = knowledge. Aftoktognosis seeks to answer these questions. It is a journey of exploration, a journey to find answers to the unthinkable. Aftoktognosis is suicidology taken to the next step.

In common language, suicide is defined as, "Death from injury, poisoning or suffocation where there is evidence that the injury is self-inflicted and that the decedent intended to kill himself/herself."

Why does suicide happen?

Many are the theories proposed to answer this question. Most theories I have come across, in my opinion, are extremely superficial, and at best can explain only a small percentage of suicides.

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[2] Federico Sanchez, *A Thousand Moments of Solitude.*

Freud, after allegedly hearing Wilhelm Stekel in Vienna in 1910 state, "No one kills himself unless he had either wanted to kill another person or wished another's death," proposed a theory of self-destruction, "As murder of an introjected love object toward whom the victim felt ambivalent." This was based on his initial conclusions, where he believed that inward directed hostility led to the prominence in aggression in the dynamics of melancholia (as depression was called).

In the late 1600's, legal and religious prescriptions against "self-death" began to decline. This marks the beginning of society's understanding of suicide. In place of "willful badness" or "possession by spirits," "unreasoning passion" and "idiotic incomprehension" were used as possible explanations of suicide. Some began to see suicide as a form of insanity. For the next three centuries, with the exception of a few authors like Hales and Donne who contemplated the rationality of suicide, suicidal persons by definition would be considered insane. The connection between mental disorders and suicide isn't made until the 20th century.

In general, suicide has a low base rate in the general U.S. population, with an annual incidence of 11.2 suicides for every 100,000 persons. This low incidence contributes to the difficulty of developing accurate methods to identify individuals at risk. There is no psychological test, clinical technique, or biological marker sufficiently sensitive to support accurate short-term prediction of suicide at the individual level. However, with an annual incidence of 30,000 suicides per year among the general population of the U.S., perhaps we should not use the word "low base rate." Given that the annual prevalence for affective disorders is approximately 18 million, and assuming that 50 percent of suicides are related to an affective disorder, the annual suicide rate in this population would be approximately 83.3 suicides per 100,000 depressive persons. This hypothetical rate is about eight times that of the general population, and means that about 99.92 percent of persons with affective disorder do not commit suicide *in a given year* (italics added).[3] This explains why most patients in a clinical setting are considered to be a low risk for suicide and why so many suicides come as a surprise.

Attempts have been made to explain suicide as an episode in a long, hard life, occasionally through the analysis of personal documents such as letters, diaries, autobiographies, and especially suicide notes. Others try to explain suicide by looking for statistical significance in many variables. Philosophical explanations abound, some beginning with the question of: What is the purpose of life? Others have tried to explain suicide in terms of an individual's relationship with his culture or society. Others have limited their study to the interaction between two people or within a family nexus. Others have approached the subject from the psycodynamic point of view in terms of unconscious conflicts or unconscious hostility to the father; where suicide is seen as an unconscious murder.

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[3] Douglas G. Jacobs, Editor., *The Harvard Medical School Guide to Suicide Assessment and Intervention,*1999.

Dr. Edwin Shneidman, founder of modern suicidology and one of the first to do a full scientific study of suicide notes, many years after presenting his results, wrote, "Reluctantly, after a decade or so of earnest efforts, I came to recognize that many notes are, in fact, bereft of the profound insights that we had hoped would be there. Now it seems that we have come to rest somewhere in the middle, believing that, as a group, suicide notes are neither always psychodynamically rich nor psychodynamically barren, but rather, on occasion—*when the note can be placed within the context of the known details of a life* (of which the note is a penultimate part)—then words and phrases in the note can take on special meanings, bearing as they do a special freight within that context."[4] (Italics added.)

Explanations of suicide in terms of genetic vulnerabilities have been advanced based on the fact that some mental illnesses run in families. Psychological theories in terms of psychological pain have been proposed.

Perhaps closer to explaining, but still leaving much to desire, is a psychiatric approach, which looks at mental illnesses such as borderline personality disorder, depression, schizophrenia or alcoholism, or biochemical explanations that blame suicide on chemical imbalances in the blood or the brain.[5]

But why does suicide happen? My response (and forgive the pun) is: Ignorance. This is the plain and simple answer as to why we loose so many to suicide: ignorance among the sufferers of mood disorders, ignorance of their families and loved ones, and ultimately ignorance of the doctors that treat them.

Ignorance is why mental disorders are stigmatized. Ignorance is why suicide has been viewed with repulsion, fear, or superstition. Even under the veil of scientific discovery, many strange theories, based on a total misunderstanding of how the brain works, have been proposed. This is why, historically, people that suffer mood disorders, the people most vulnerable to suicide, have been shunned or treated as if possessed by demonic or evil forces, or treated as sinners or witches, or at best as a family embarrassment and kept quiet. Simplistic explanations for suicide abound, and at best are a feeble facsimile of what really happens in the brain.

Many who suffer mental disorders are ignorant of what they are experiencing, and think this is normal or how life is. Kay R. Jamison, an expert on manic depression and suicide, said, referring to her own manic-depression, "I went to college and graduate school. It was a totally tumultuous period in my life, although I had no diagnostic label or understanding for it yet."

The quandary of the mind/brain is a most baffling and difficult one. As Kay Jamison says in her book "*Night Falls Fast*", *"One hundred billion individual nerve cells—each reaching out in turn to as many as 200,000 others—diverge, reverberate, and converge into a webwork of*

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4 Edwin Shneidman, *Autopsy of a Suicidal Mind.* 2004.

5 Ronald W. Maris, Alan L. Berman, John T. Malsberger and Robert T. Yufit, *Assessment and Prediction Of Suicide.* 1992.

staggering complexity. This three pound thicket of gray, with its thousands of distinct cell types and estimated one hundred trillion synapses, somehow pulls out order from chaos, lays down the shivery tracks of memory, gives rise to desire or terror, arranges sleep, propels movement, imagines a symphony or shapes a plan to annihilate itself."

But, returning to the question, "Why does suicide happen?" in the sense of what actually takes place in the brain that makes suicide possible, is the subject of this book. Hopefully this book will eliminate the ignorance by clearly explaining the brain processes involved in producing this behavior so contrary to life. Once these processes are understood, suicide becomes a natural response under certain conditions, as we will see later on. This book will also review present knowledge and the limitations of present therapies or pharmacologies, which, for now, is what people encounter when they seek help; and it seems to be all we've got. Hopefully, the theories presented here can begin to influence the future improvement of what the medical community can offer to many that suffer mental disorders. A scientific framework will be established to explain suicide by providing a comprehensive theory of the human brain based on neurology.

If we look at some of the statistics on suicide they bring us face to face with the relatively simple questions of the how's, where's, and who's of suicide. The statistics are staggering. The question of why is more elusive.

I want to focus on suicide among the young (thirty five years and younger) and otherwise physically healthy. However, many of the causes for suicide among the young apply to older people. First, one thing needs to be understood: Psychiatric disorders are an illness, and like many other diseases they can lead to premature death. It is impossible to understand the type and the intensity of the suffering that people who have these disorders endure when one hasn't experienced the suffocating pain and horror that accompany these conditions. This kind of pain can be more severe and unbearable than physical pain, and certainly is less comprehensible to the sufferer.

The rate of suicide among the young is increasing but there is no consensus as to why this is so. The reasons proposed are varied, going from the threat of nuclear extermination, terrorist attacks, MTV, peer and parental pressure, child abuse, promiscuity, increased affluence, almost universal divorce, home-alone children, excessive freedom, boredom, Watergate, too much discussion or too little discussion on suicide, terrorism, war, excessive choices or too few choices. Others have proposed that today's societal pressures to "grow up" in ways that both appeal to and overwhelm adolescents may be another factor. The fact remains that we don't know.

The increasing numbers of mothers joining the workforce since the 1950's could be a source for the beginnings of emotional problems in young children that eventually could culminate in suicide. In 1955, 18 percent of mothers with children under six years old were in the workforce. A decade later this number had risen to one fourth. By 1975 it had climbed to 39 percent. By 1985, slightly more than half of mothers with children under 6 were working. By the year 2000 the number of mothers of young children in the workforce is 65 percent, with a slight decrease after that. If we look

at women in the workforce with children six to seventeen years old the numbers are higher: in 1955 it is almost double at 38 percent, climbing steadily to 65 percent by 1980, and by 2000 reaching almost 80 percent.[6] Yet, I have not been able to find any studies comparing suicide rates (or risks) for the children of working mothers compared to mothers who stay at home.

Allow me to put suicide statistics in perspective: in 2001 we lost ten times more people to suicide than to terrorism in that year. In some years there are more deaths by suicide in one hour than losses due to terrorist acts in the United States in a whole year.[7]

There are approximately thirty thousand deaths by suicide per year in the United States and almost half-a-million suicide attempts serious enough to require emergency room treatment.

Suicides of people under thirty-five number about ten thousand per year. Suicide is the third leading cause of death among the young. For white males aged 15-19 it ranks second, and for physicians under 40 it ranks first. These figures include all types of suicide.

Suicide rates among blacks, one-third that of whites three decades ago, is now practically equal, especially in urban areas. The highest rates of suicide among American Indians and Alaskan Natives occur among young men ages twenty to twenty-nine, with a decline in the later years of life.

Approximately 70 percent of the people who have committed suicide were successful on their first attempt, and the other 30 percent were successful only after they had made one or more attempts.[8] Of that 30 percent, about one in six were successful on their second attempt.[9] On the other hand, only 10-15 percent of the suicide attempters go on to complete suicide.[10]

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6 U.S. Department of Labor, Bureau of Labor Statistics.

7 The figures and numbers on suicide in this section are taken from the following sources:
Kay Redfield Jamison, *Night Falls Fast.*
John H. Hewett, *After Suicide.*
William Styron, *Darkness Visible.*
Carla Fine, *No Time to Say Good-by.*
Harold I. Kaplan, *Synopsis of Psychiatry.*
Eric Marcus, *Why Suicide?*
Adina Wrobleski, *Suicide Why?*
E. Fuller Torrey & Michael B. Knable, *Surviving Manic Depression.*
Andrew Solomon, *The Noonday Demon*. And:
Armand Nicholi, Jr., M.D., *The Harvard Guide to Psychiatry.*

8 Ronald W. Maris, *Pathways to Suicide: A Survey of Self-destructive Behaviors.* 1981.

9 R.W. Maris, A.L. Berman, J.T. Maltsberger and R.I. Yufit, eds. *Assessment and Prediction of Suicide.* 1992.

10 Thomas White, *How to Identify Suicidal People, A Systematic Approach to Risk Assessment,* 1999.

Underreporting of suicide is estimated at between ten to twenty percent. However, some experts believe that suicide rates could be three to five times higher. The numbers vary enormously. Many drug overdoses and one-car accidents that could be suicides are reported as accidental deaths; some coroners and doctors are reluctant to label a death as a suicide if there isn't enough evidence.

About half of all suicides are committed by people under psychiatric care, and yet most come as a surprise. This seems to indicate that something is quite wrong with our thinking as concerns suicide. Suicidality should not be lumped together with simple symptoms such as disrupted sleep; nor should it be ignored because depression has lifted, because most suicides are related to depression, either unipolar or bipolar. Suicidal behavior can be linked to other causes, such as schizophrenia, personality disorders, panic attacks and anxiety disorders; perhaps suicide should have a diagnosis of its own, because most that suffer these ailments do not commit suicide. Suicidal behavior tends to occur early in the course of unipolar depression *before* diagnosis and treatment and it is generally accompanied by increased agitation. Anxiety increases the risk of suicide in people with affective disorders.

On average, people will experience four lifetime major depressive episodes. However not all are at the same risk. People with at least three prior episodes relapse at rates of 70-80 per cent within three years, while those with no prior depression relapse at rates of 20 % over a comparable period.[11]

Severe anxiety and panic attacks are statistically significantly differentiated among patients who committed suicide within one year of assessment from the majority of depressed patients who survived the one-year follow up period. The frequency of moderate-severe anxiety symptoms in depression is about 65 to 70 percent. In contrast, expressed suicidal ideation or intent and prior suicide attempts were not associated with suicide within one year of assessment, but were associated with suicide between two to five years of follow-up. Not surprisingly, studies have found that treatment outcomes in patients with depression with anxiety attacks, panic attacks or obsessive compulsive disorder are relatively poor and require more intensive pharmacological treatments.

In another study, although 65 percent of patients who subsequently committed suicide denied suicidal intent as their last recorded communication, 86 percent of this group were rated to have severe anxiety/agitation associated with depression in the week prior to their suicide.[12] The combination depression and morbid thoughts with high energy and agitation is dangerously uncomfortable. Impulsive and violent agitations, during which an individual can feel like punching his hand through a wall or jumping from a car are common in mixed states.

Depression among suicide victims has been frequently found to be undiagnosed, untreated, or undertreated.

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[11] Robert Leahy, Editor. *Contemporary Cognitive Therapy*. 2004.

[12] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention*.

Not surprisingly, 94 % of subjects qualified for a psychiatric diagnosis at the time of the suicide. And a major affective disorder, or alcoholism, or both were implicated in 57-86% of all suicides, with affective disorder the more common diagnosis. It is generally considered that this estimate is conservative because most studies permit a maximum of one psychiatric disorder per subject. Other studies show that suicide victims struggling with terminal illness account for maybe 2-6% of suicides.[13]

The severity of psychopathology greatly increases suicide risk, especially when two or more conditions exist, such as depression with anxiety, panic attacks or psychotic episodes. In 70-80 percent of suicides comorbid disorders have been identified. There is a fivefold increase of attempted suicides associated with more than one diagnosis. Mood and addictive disorders, and mood and personality disorders appear to be particularly lethal combinations.

There is evidence that there is a genetic factor contributing to suicide risk because major psychiatric illnesses run in families. Most suicides are associated to mood disorders, schizophrenia, alcoholism and substance abuse, and cluster B personality disorders.

As many as 50 percent of suicide victims were drinking at or near the time of their death, and 89 percent of alcoholics that committed suicide were drinking when they carried out the deed. There is yet no unifying theory that adequately explains the connection between suicide and alcohol. Some have suggested that the predisposition to substance abuse and certain personality disorders derive from a common but as yet unidentified biological substrate. Others have insinuated that it is a form of self-medication or because of the brain neurochemical alterations produced, alcohol in some way produces suicidal behavior.[14] It probably has to do with other, well-known effects of alcohol, such as increased impulsivity and/or reduced self-control. The fact remains that a high incidence of depression exists along side substance abuse.

Completed suicide rates for borderline personality, despite their high frequency of suicidal behavior, are 9 percent, comparable to rates in schizophrenics. Typically, a co-occurrence of another disorder increases suicide risk. Male patients suffering borderline personality disorder and depression completed suicide in 18 percent of cases.

Suicide rates are significantly increased for both sexes and all age groups following severe natural disasters such as, giant earthquakes, huge floods, and/or a category 4 or 5 hurricanes.[15]

There is a range of suicidal thought and behavior. It varies from risk-taking behaviors that could include any activity that is dangerous, like driving fast, parachuting, rock climbing and so on, through different degrees and types of suicidal thinking and ends with suicide attempts and ultimately, completed suicide.

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[13] R.W. Maris, et.al., *Assessment and Prediction of Suicide.* 1992.

[14] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

[15] Ibid.

In some cases, a person might want to die, and manipulate others into murdering him or her without sharing the suicidal intent. According to one study, as many as 25% of all homicides are provoked by their victims.[16]

Even after identifying large numbers of suicidal patients, mainly because they sought professional help, many treatment failures are documented. Part of the reason for this is the lack of understanding of the psychology of suicide, or even the intervention methods, such as antidepressant drugs, lithium carbonate, electroshock or psychotherapies. The present consensus among experts varies greatly and there are many misconceptions, among them that the road to suicide is gradual. The reality is that the divide between suicidal thoughts and action is not clear. A potential deadly impulse might be interrupted before it is ever acted upon, or an attempt with mild intent of death might be carried out with a full expectation of discovery and survival. And a few individuals use suicide threats or attempts to provoke change in the behaviors of others.

Any scientific theory of suicide needs to explain the facts. I try to summarize in the following pages the most accepted models. But current neurological or psychiatric theories cannot explain suicide satisfactorily. Consequently, therapies based on erroneous premises fail in many cases, more often than not.

Even though most suicides are related or concurrent with depression, there is no great correlation between suicide and the severity of depression. Some suicides occur during mild depression while others cling desperately to life under the worst concurrent, imaginable conditions: loss of loved ones, work, freedom, and even home or country. And, on the other hand, people with bright futures and apparently no real problems occasionally resort to suicide. Suicide is not an escape from a difficult life; it comes from the dark recesses of the brain, beyond awareness and rationality.

Different investigations conclude that 10% of the adult population has had suicidal thoughts at some moment in their lives. Of the adult population interviewed, 3% reported having attempted suicide one or more times. Of those who attempted suicide once, there is a 10 to15% chance they will eventually succeed in killing themselves. This group is the one that is at highest risk of suicide. For every completed suicide about eight attempts occur.

Attempted suicide rates in adults vary between 1 and 4 percent, among countries, even regions in countries, or cultures. Adolescents vary more, reporting between 2 and 10% with a significant number reporting more than one attempt.

In 1988, in the United States, men 75 years of age and older were at the highest risk for suicide, and for women the highest risk group was those aged 45 to 65. The next high-risk age group is adolescents.

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[16] R.W. Maris, et.al., *Assessment and Prediction of Suicide.* 1992.

A 1994 Gallup poll found that 12 percent of adolescents between the ages 13 and 19 said they had "come close" to taking their own lives, 5 percent stated that they had actually attempted to kill themselves and 59 percent reported knowing a peer that had attempted suicide.[17]

Divorce and out-of-wedlock pregnancies contribute to over half the children in the United States growing up in homes with one or both parents missing. A statistically significant number of adolescents that commit suicide come from fragmented homes. Fifty percent of children and adolescents that attempt suicide come from broken homes with the father absent (not very surprising since half of all the children come from broken homes). Another study showed that 49% of teenage suicides came from homes with one parent missing. And adolescents that attempted suicide, compared to control groups, have a statistically significant incidence of separation and divorce.[18]

Gay people (15-20 percent) attempt suicide much more than heterosexuals (3.5-4 per cent). Gay people probably suffer more stress than heterosexuals and this could be a factor leading to higher depression rates.

There is evidence in the industrialized nations that the rate of suicide among the young has doubled or tripled over the last few decades, with the greater increase happening between the 1950's and the 1970's and subsequently remaining more or less steady, and during the same period the rate of suicide of older people went down. This overall increase in young people might be due to better reporting. There is strong evidence that mood disorders tend to run in families. At the very least there is a genetic disposition to mood disorders.

In the United States suicide rates are about 10-12 per 100,000. In other industrialized nations such as Japan, Austria, Denmark, Sweden, West Germany and Hungary suicide rates are 20 per 100,000. In countries, where perhaps the family ties are much more important, like Italy, Spain, New Zealand and Ireland, the rate is less than 6 per 100,000.

Suicide rates increase with age. Among men, suicides peak and continue to rise after age 45; among women, the greatest number of suicides occurs after age 55. The elderly attempt suicide less often than do younger people but are successful more often. For males between 15 and 24 years old there was an increase of 40% in the suicide rate between 1970 and 1980, and is still rising slightly. The suicide rate for women in the same age group showed only a slight increase. Among men 25 to 34 years old, the suicide rate increased almost 30 per cent.

Suicide rates among 15-24 year-olds in 1950 per 100,000 was 2.7; by 1960 it increased to 5.2, in 1979 it moved up to 12.9 and in ages 5-14 it was 0.4. In 1992 the suicide rate per 100,000 in 5-14 year-olds was 0.9 and in 15-24 year-olds it was 13.0.

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17 Thomas White, *How to Identify Suicidal People,* 1999.

18 Edited by Armand M. Nicholi, Jr., M.D. *The Harvard Guide to Psychiatry.*

Many suicides happen after a severe loss and high levels of stress with a low and/or lack of support system.[19] This tends to stereotype suicidal behavior and rationalize suicide as a reaction to severe loss. Severe loss is just one more link in a chain that leads to self-destruction.

For example: it is undeniable that the cumulative effect of recent losses of spouses, family and friends in rapid succession, without proper time to grieve each one, is a great cause of stress to the elderly. When you add the loss of physical and mental abilities, and are facing retirement, inadequate income, social isolation and loneliness, the loss of self-esteem is inevitable. Yet, this is not the ultimate reason or explanation for suicide among the elderly; these are just links in a chain.

There is strong evidence that serotonin inhibits violent, aggressive and impulsive behavior. Postmortem studies of suicides show low levels of serotonin in certain locations in the brain, especially in areas associated with inhibition, which in turn might produce a strong influence to act impulsively on emotion. More than half of suicide attempts are impulsive; they occur within the context of a premeditation period of less than five minutes with disregard for the consequences to others. Even when many suicidal patients have a well-formulated plan, the final decision to commit suicide is often determined by impulse.

Lower levels of noradrenaline and norepinephrine, though less consistently than the serotonin studies, appear to be reduced in postmortem suicidal brains. This suggests, and this is only a suggestion, that low levels of some neurotransmitters might play a role in suicide, perhaps in indirect ways.

The method of suicide varies from place to place and from time to time. Yet only a few methods account for all suicides: gunshot, jumping, poisons, gas, hanging and drowning. I feel it is obvious that if guns are easily available, there will be more suicides by the use of guns. In the United States, guns are the leading choice of death by suicide. In England where the use of guns is highly restricted, guns rank fifth as the method of choice. Hanging, strangulation and suffocation are lumped together in the United States and are the second leading cause of death involving suicide. If chemicals are easily available, then this will be a more frequently used method, as seems to be the case in China, where pesticides and fertilizers are easily obtainable. In India, one particular method used in high proportion is jumping in front of trains. If high buildings are handy, then more suicides will be recorded by jumping from high places. Economic status does not reflect anything on suicide rates, even though people in the upper class suffer more from mood disorders.

External factors can lower suicide rates. When guns or medications are hard to come by, suicide rates are lower than elsewhere. In England, when they switched from lethal coke gas to less toxic natural gas, annual gas related suicides dropped from 2,368 to 11. Reducing the means to kill one self impulsively will increase the chances that the impulse will pass.

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[19] Thomas White, *How to Identify Suicidal People*. 1999.

In 1992 the suicide rate for people sixty-five and over group is 61.2 per 100,000, compared to about 44 per 100,000 in people under sixty five. The typical explanation is that it shouldn't be surprising that the elderly have a higher suicide rate; after all, they face greater loss of health, possible loss of longtime spouse or greater loss of status than their younger counterparts, with lesser possibilities of re-attaining their accustomed station in life.

People suffering from the following seven diseases of the central nervous system have a higher risk of suicide: epilepsy, multiple sclerosis, head injury, cardiovascular disease, Huntington's chorea, dementia, and acquired immune deficiency syndrome (AIDS). All are diseases in which an associated mood disorder is known to occur. Four endocrine conditions are associated with increased suicide risk: Cushing's disease, anorexia nervosa, Klinefelter's syndrome, and porphyria. Mood disorders also attend these diseases. Peptic ulcer and cirrhosis, both found among alcoholics are two gastrointestinal disorders with an increased risk of suicide. People with prostatic hypertrophy and renal disease treated with hemodialysis, both problems with changes in mood, are also at a higher risk for suicide.

Jails are generally short-term holding facilities, whereas prisons are long-term detention facilities. Suicide is the leading cause of death among jail inmates, and the suicide rate is estimated to be nine to ten times higher than that of the general population. The majority of suicide victims are white, male, young, single or divorced; in many cases they were arrested for nonviolent offenses (drug- or alcohol-related) and were intoxicated at their time of arrest. Most of the suicides took place by hanging within the first twenty-four hours of incarceration and most (90%) were housed in isolation or segregation at the time. The jail administrators interviewed suggested that they were placed in isolation because of abnormal behavior. Psychiatric disorders (including major depression, bipolar disorder and schizophrenia) and previous attempts have been linked with certain jail suicides.

In prisons, suicide victims are generally older, are serving long-term sentences for major offenses, and have histories of psychiatric illness and suicidal behavior. They generally commit suicide after several years of incarceration, and their actions are frequently precipitated by severe problems with the institution. As in jails, most prison suicides occur while the victims are held in isolation.

Ultimately, with present knowledge, the process by which certain inmates become suicidal within the context of a life condition behind bars has not been addressed.[20]

Most suicidal people do not openly announce their intentions. However, about eighty per cent of people that commit suicide leave clues to their therapists or doctors, or their loved ones and friends as to their intentions. Naturally the elderlies' clues are taken less seriously. Men commit suicide three times as often as do women, a rate that is stable for all ages. Women, however, are four times more likely to attempt suicide, as are men. About two thirds communicated their suicidal intentions somehow over a period of weeks prior to their deaths, and 40% communicated it in very clear terms.

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[20] R.W. Maris, et. Al., *Assessment and Prediction of Suicide.*

Explanations of suicide motives vary tremendously and cover a very wide spectrum. On one end, we have people with a terrible chronic disease like cancer or multiple sclerosis, where they face a continuously worsening quality of life and/or pain. Rational suicide is a frightening idea, but this type of decision is generally understood. Yet, most people that are faced with these terrible illnesses *do not* commit suicide. In cancer patients the increased risk for suicide is only twice of the normal population.

Most non-psychiatric medical illnesses, even such serious disorders as Huntington's disease, multiple sclerosis, or cancer, so often tied to pain, disfigurement, diminished dignity and independence, and death, are in proportion to psychiatric disorders, one sixth to one twentieth less likely to end in suicide.

The only group of people that is completely resistant to suicide is pregnant women. The cause is not known, but it is straightforward to imagine that it is related to producing new life.

In contrast, pregnancy has no clear-cut effect on women that suffer panic attacks, but there seems to be some light improvement with pregnancy, especially when the panic attacks are lighter.[21]

There seem to be people who commit suicide for reasons of honor: the General facing military defeat, or the banker caught stealing. But these are a very small percentage of suicides. The most common cases of suicide involve people suffering from mood disorders like depression, manic depression and schizophrenia. Almost 90 per cent of those who commit or attempt suicide have a diagnosed mental illness. Roughly 75 per cent involve depression and manic depression, with schizophrenia accounting for about 10 per cent, and dementia, delirium and personality disorders about 5 per cent. Panic attacks and anxiety disorders are generally diagnosed alongside these other conditions and greatly increase the chances for suicide. Severe anxiety and severe agitation are portent predictors of suicide. One in five people suffering from major depression and nearly half of people suffering from manic depression (bipolar) will attempt suicide. This is the group that is at highest risk.

One explanation of the increased risk of suicide among bipolar II patients is that they might present other problems as well: alcoholism and/or personality disorders. Undoubtedly, they lead more chaotic lives, and have inadequate support systems and more often than not, they don't comply with medication. Although their mood swings are less severe and less dramatically disruptive than patients with bipolar I illness, the overall treatment outcome may be limited by these associated features. Women with manic-depressive illness appear to be at greater risk than men for suicide attempts. The depressive phase of bipolar illness is associated with a great disproportionate rate of suicide.[22] Suicidal ideation did occur in 7 percent of manic episodes, however no suicides. Interestingly, almost 30 percent of manic depressive suicide victims were

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[21] Helen Saul, *Phobias, Fighting the Fear.* 2001.

[22] K. R. Jamison, *An Unquiet Mind.* 1995.

classified as being in a "depressive state, *recovering"* at the time of death. The recovery period and the period following hospitalization carry increased risk of suicide. The beginning of medication response is often cited as a dangerous period, presumably because the patient is experiencing some return of energy and motivation, but is still feeling hopeless and depressed. This has been questioned by others.

Some have proposed that the apparent improvement may simply reflect the resolution of ambivalence once a decision to die has been made; that it may represent a genuine calm before the storm brought about by biological changes, or a transition from one phase of the illness into another; or even, that the improvement is a deliberate attempt to deceive physicians, hospital staff, and family in order to carry out a suicide plan.[23]

The improvement might be temporary and a perceived recurrence of symptoms might trigger despair and a disproportionate hopelessness. It certainly can be demoralizing to think one is getting better only to plunge back into depression.

More depressive disorder patients commit suicide early in their illness; more males than females; and the chance of depressed people killing themselves is increased by their being single, separated, divorced, widowed or recently bereaved. Suicide is more likely at the onset or the end of a depressive episode. One of the highest-risk periods for suicide is, ironically, when patients are recovering from depression. The transition from their low state to a normal mood is a terrifying and hazardous experience. There might also be huge disappointments when after feeling well again one feels ill all over again. The resurgence of will and vitality, signs of returning health, makes possible the acting out of suicidal thoughts. In all these cases there is also a wide range or spectrum of going from people who have been suffering and have suicidal thoughts for many years to cases where there are no previous warning signs that are visible on the surface.

Suicide, after cardiovascular events, is the most likely cause of death for individuals with bipolar disorder. The lifetime rates of suicide attempts in persons with bipolar disorder are 29.2% and with unipolar disorder, 15.9%.[24]

The age of onset of schizophrenia is typically in adolescence or early adulthood. Most schizophrenics who commit suicide will do so during the first few years of their illness. People with schizophrenia who are better educated and more intelligent, perhaps, because of their greater insight into their illness are more likely to kill themselves.

One study found a greater risk of suicide among people with paranoid schizophrenia with a threefold risk compared to non-paranoid schizophrenics and an eightfold increase compared to negative subtypes. The lowest risk for suicide among schizophrenics was found among patients with negative or deficit subtypes of schizophrenia.[25]

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[23] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

[24] Chen, Y.W. and Dilsaver, S.C., *Lifetime rates of suicide attempts among subjects with bipolar disorder and unipolar disorder relative to subject with other Axis I Disorders.* 1996.

[25] W.S. Fenton & T.H. McGlashan, *Natural history of schizophrenic subtypes: II. Positive and negative symptoms and long-term course.* Arch Gen. Psychiatry. 1991.

In the case of schizophrenia, males are at greater risk than females. In general, they are affected at an earlier age and tend to be single. On top of that, each exacerbation often leaves the patient more emotionally handicapped. Most schizophrenic patients that commit suicide are depressed, rather than intensely psychotic. And lastly, some medications used in the treatment of schizophrenia carry risk for side effects that at times can incapacitate the patient and thus contribute to the patient's suicidality. Often schizophrenics are rendered jobless without prospect for employment, inadequately understood, unaccepted in their family and community, and hopeless about their future.[26]

Nearly 3 in 4 people with borderline personality disorder attempt suicide and 5 to 10 per cent do kill themselves. In these cases, they are exquisitely sensitive to actual or perceived rejection. About 40 per cent of these suicides are committed in the presence of other people.

Substance abuse and alcohol increase the chances of suicide by disinhibiting certain behaviors; risk taking, violence and impulsivity are increased. For those who are potentially suicidal, this may be lethal. Many times, mental illness and drug abuse go hand in hand as a form of self-medication.

Among people without psychiatric illnesses the suicide rate is about 8 per 100,000. For people with moderate depression the rate escalates to 220 per 100,000, and to 3,900 per 100,000 among people with severe depression. Depression and manic depression are associated not only with completed suicides but also with serious attempts at suicide. The risk of someone making a second suicide attempt is highest within three months of the first attempt.

The highest predictor of suicide is a previous attempt; almost one third have attempted suicide before. One percent of those that attempted suicide will end their lives within a year and ten percent will do so in ten years. There are about sixteen attempts per every completed suicide.

Eight out of ten people who eventually kill themselves give warnings of their intent (primarily to their doctor or therapist). Fifty percent say openly that they want to die. It is a particularly dangerous sign if a person admits to a plan of action. It can also be an ominous sign when a person that has been threatening to commit suicide becomes quiet and less agitated.

The risk of suicide for psychiatric patients within a month after their discharge, compared to the general population, is estimated at two hundred times greater for men and one hundred times greater for women.

There are many guidelines to evaluate suicide risk, mostly having to do with answering the question, "Do you have suicidal thoughts or plans? Do you feel like dying?" However, these are not reliable. Many people, during severe bouts of mental disorders have suicidal thoughts and never act on them. Family, friends or anyone in a support capacity dealing with anyone that has a mood disorder, especially depression

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[26] Douglas Jacobs, *The Harvard medical School Guide to Suicide Assessment and Intervention.*

or manic depression, should be communicating with each other all signs that can be indicative of "abnormal" behavior.

We lost our son, Mitch to suicide a few days before his 23rd birthday. We were devastated. Ignorance and lack of communication certainly played important roles. If my wife had told me about some things she saw in our son Mitch's behavior the last few months of his life, and I had told her what I saw, it is probable that we would have acted in a more concerted fashion, as the combined information would have alerted us both to a more serious situation than either of us interpreted. Also, I must insist that communication with the person that is suffering the mood disorder be opened and maintained at all costs; even when the person doesn't want to talk "about it." When a person is depressed, engagement, especially emotional engagement with other people, should be maintained or forced at all costs. Yet, depressed people are the last ones to seek engagement. It is up to the family and friends to maintain emotional communications open.

Only about one in five people who commit suicide leave a note. These notes generally don't even start to reflect the dark interior motives that could drive these desperate acts. Even when the perpetrator is a skilled writer, it is difficult to envision that a depressed, confused, hopeless state of mind might lead to great eloquence.

About four fifths (81%) of suicide victims have been under the care of a physician within 6 months prior to their deaths. More soberingly, 91% of victims of suicide by overdose had been under care of a physician, and in over half the cases, the physician had supplied by prescription the complete means to their self-destruction.

Unfortunately, the pharmacological arsenal to relieve the misery, hopelessness and the cumulative dark despair with all its pains and chronic distress is today limited, though it is much greater than in the last decade and increasing.

It is very tempting to look at the life of someone who has committed suicide and attach to it a huge complex tangle of issues. No one illness or event causes suicide, but psychiatric illness is almost always present. Perhaps each case of suicide is ultimately unknowable. It is a very private and terrible act. The privacy of the mind is inaccessible. Any clue we might infer is indirect. Suicide is not necessarily a very awful way to die, but the path that leads to it generally is; the mental suffering is prolonged, intense and relentless. The suffering is inexpressible. Love, friendship and understanding are hardly ever enough to counteract the pain and destructiveness of mental illness. But love and support should be at all times unfailingly provided if there is hope for success.

It is important to dispel some erroneous notions about suicide. Many depressed people think about suicide often, but it is an unreal thought, an abstraction, even when depression is worsening and the methods of suicide contemplated become more violent. Occasionally, the thought of suicide allows the depressed person to exert some control and help them continue on. The knowledge that they can kill himself or herself one minute later might allow them to survive the next few moments.

I cannot emphasize this enough: *most young people that commit suicide do no want to die; it is not an escapist way out.* In some cases suicide is an act of extreme courage, perhaps misguided at the moment, but definitely not an act of weakness or cowardice.

The brain creates a visual map of the outside world and integrates it with the map of the body and thus achieves the trick of moving through space. Similarly, the brain tries to create a continuum between all outside events and the internal emotional landscape, in a way that produces precisely the correct emotions to guide the best responses according to the circumstances. Under certain conditions, emotions go awry. Understanding how this happens is necessary if we want to understand suicide.

Some of the details are ultimately unknowable. No one is there to record the last few moments of a life snuffed out in an act of suicide. But even if one were present as a silent witness, in all probability the suicidal individual could not convey or communicate properly his emotions or rationalizations.

We will see shortly how suicide can be part of a disease, generally depression, manic-depression in the depressive phase, or schizophrenia and personality disorders. These mental disorders produce a diminished sense of self, or even a total loss of self, and it is only under such conditions that suicide becomes possible. With a loss of self, the possibility of self-harm becomes a logical consequence of distorted thinking.

Just as the AIDS/HIV campaigns to generate more awareness of the dangers and forms of transmission of the disease helps lower the spread of this deadly killer, awareness of the problems relating to suicide should help us prevent some of these tragic deaths.

Most of the SSRIs, the more popular antidepressants, have not been studied to determine their capacity to prevent suicide. In manic-depressive patients using lithium, which has been more rigorously tested, the rate of suicide among patients who discontinued lithium treatment increased sixteen fold. Most people suffering from severe depression find it hard to feed themselves, and under such conditions the energy needed to end one's life is simply non-existent. It is possible that some drugs that assuage depression increase the possibility of suicide by increasing motivation in general. It is important to distinguish between an enabling and actual cause.

It has not been proven conclusively, but at the time of this writing, some think that many antidepressants contribute to suicidal behavior, especially in children and teenagers. The F.D.A. is seeking a suicide caution label for ten antidepressants: Prozac, Paxil, Zoloft, Effexor, Celexa, Remeron, Lexapro, Luvox, Serzone and Wellbutrin. All of these medications affect the levels of serotonin. Some side effects might be agitation, anxiety and hostility.[27] My son Mitch was definitely suffering agitation and anxiety, and these symptoms, as best I can determine, were coincidental with his use of Paxil. But, it must also be said that he took the Paxil to alleviate his anxious feelings. This is the argument presented by the drug companies: depressed people have the highest suicide risk, and depressed people take antidepressants; consequently, the depression leads to suicide, not the medicine.

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[27] The Associated Press, *F.D.A. Seeks Suicide Caution for Ten Antidepressants,* NYTimes.com, March 22, 2004.

In 2004, concerns for increased risk for suicidal behaviors caused by SSRIs for childhood depression resurfaced and intensified. Reviews that examine both published and unpublished clinical trial data show increased suicidal ideation and behavior in depressed children on antidepressants compared to those on placebos, but this is true only for certain antidepressants like Paxil and Effexor.

Oddly it is not true regarding Prozac. Medicines like Paxil and Effexor have much shorter half-lives than does Prozac. In this context, half-life means the amount of time it takes for half the medicine to clear the body. Short half-life medicines clear quickly and thus can shock the system if not taken very regularly, causing reactions like anxiety, insomnia, and agitation, which in turn have been linked to increased suicidality. Thus, some antidepressants need to be very carefully managed, particularly with young children and teenagers.[28]

Suicide rates among the young have been increasing in the last fifty years. The use of antidepressants has been increasing, perhaps indiscriminately. The pressure on the F.D.A. to put warning labels on antidepressants is coming from concerned parents, who even though they can't prove it, feel that their children's deaths might have been caused by the antidepressants. A warning label is a minimum precautionary action. Yet, if the warning symptoms are the reason for taking the medication, once you read the warning label, how do you attribute suicidal behavior to the medication? If agitation, anxiety and hostility are symptoms that were not present originally, and appear coincidentally with the ingestion of the antidepressant, then, these might be considered warning suicidal signs, and the medication should be discontinued under medical supervision.

In a recent study sponsored by the National Mental Health Institute comparing cognitive behavioral therapy and drug treatment (Prozac) for depressed adolescents the following was found: 71 percent who received Prozac and therapy responded well to treatment compared with 61 percent who receive Prozac alone, 43 percent who received talk therapy alone and 35 percent of those who received a placebo treatment.

The study also found that patients became significantly less suicidal, no matter which treatment they were given. No patient committed suicide during the trial. But the risk of a suicide attempt among patients given Prozac was twice that of those who did not. There were five suicide attempts among those given Prozac and just one among other participants.

In another study comparing Zoloft, an antidepressant similar to Prozac, with cognitive behavioral theory, in teenagers suffering from obsessive compulsive disorder, it was determined that those that received talk therapy improved more than those with the drug.[29]

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[28] Thomas Joiner, *Why people Die By Suicide*. 2005.

[29] Gardiner Harris, *Antidepressant Seen as Effective in Treatment of Adolescent,*. New York Times, June 2, 2004.

On a more general level, the increasing rates of depression, especially among the young, have been attributed to a number of causes, most pertaining to our modern life styles, but have not been proven conclusively: the quickened pace of life, the technological changes that accompany this, Nintendos, computer games and the internet, the isolation of people from each other, the breakdown of the family, the endemic loneliness, the failure of our belief systems (religious, political, social or moral),[30] the splintering of society into ever more finer special minorities. All of these are big stressors, especially among the young. Alvin Toffler would call it Future Shock. He discusses in the book with the same title, the increasing rate of change of everything around us as technology leads us into a manic environment with a frenetic pace.

There is confirmation between adverse life events and either an exacerbation of mood symptoms or relapse into a mood episode.[31]

The increasing reliance on medications to solve emotional problems (that in all probability are healthy, normal and need to be worked through) can also be a factor, because as soon as the medication is taken, the emotional maturity that is supposed to be developed is warped.

People suffering psychotic episodes and schizophrenia, though less frequent in the general population, and with lower rates of suicide, is the other important group contributing to the suicide statistics. Most of what has been mentioned that applies to depression and manic depression applies to schizophrenia.

One consistent finding, reported both in post mortem studies of suicide completers and in clinical studies of suicide attempters, has been evidence of decreased levels of serotonin (5-HT) or its principal cerebral spinal fluid metabolite (CSF 5-HIAA). This has been proven to be the case independent of whether the suicide was related to impulsive and violent suicides, or linked to a planned, non-impulsive suicide attempt. Alterations in the noradrenergic system have also been observed. Postmortem studies of suicide victims show fewer noradrenergic neurons in the locus ceruleus in the midbrain, the chief source of norepinephrine. And similarly to the serotonergic neurons of the raphe nucleus, the noradrenergic neurons of the locus ceruleus have widespread innervation throughout the brain.

Murder-suicide falls outside the scope of this book, yet a few statistics are in order to complete the numbers on suicide. At least 30 percent of violent persons have a history of self-destructive behavior and nearly 20 percent of suicidal persons have a history of violence directed at others. Nowhere is the link between these two behaviors more evident than in the case of murder-suicide. Although murder-suicide occurs much less frequently than either homicide or suicide, it can affect a large number of people, sometimes whole families, their friends and entire communities. Murder-suicides often

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[30] Andrew Solomon, *The Noonday Demon.*

[31] Johnson, S., & Miller, I. *Negative life events and time to recovery from episodes of bipolar disorder.* Journal of Abnormal Psychology, 106, 449-457. 1995.

occur suddenly; all parties directly involved are dead, generally with little or no prior clinical documentation. In most cases the suicide occurs within seconds or minutes of the homicide (for it to be considered a murder-suicide, the suicide has to occur less than a week after the homicide).

Although countries have very different homicide and suicide rates, the murder-suicide rate is relatively stable, varying from 0.05 in Scotland to 0.55 in Miami. For example, Philadelphia had an extremely high homicide rate from 1948 to 1952, and Denmark had a remarkably low homicide rate between 1958 and 1960; however, both had virtually identical murder-suicide rates (0.21 and 0.22 per 100,000, respectively). In 1995, the overall homicide rate in the U.S. was 8.6 per 100,000, accounting for 22,552 deaths; for suicide, it was 11.0 per 100,000, resulting in 31,284 deaths. Thus it is likely that roughly 1.5 percent of all suicides and 5.0 percent of all homicides in the U.S. occur in the context of murder-suicide.[32]

Kay Redfield Jamison, close to the end of the epilogue of her book on suicide, *Night Falls Fast* states, "I have been impressed by how little value our society puts on saving the lives of those who are in such despair as to want to end them. It is a social illusion that suicide is rare. It is not. Certainly the mental illnesses most closely tied to suicide are not rare. They are common conditions, and, unlike cancer and heart disease, they disproportionately affect and kill the young."

One undisputed fact remains: we—doctors, patients, loved ones—are all very ignorant of the causes and reality of suicide. I discussed suicide briefly, on a couple of occasions, with my son Mitch and he quickly dismissed the idea, especially as it pertained to him. Although he was a smart young man and I am much more educated than the average person, I couldn't even begin to help guide my son through his problems. At one point, I did promise him that a solution for his problems, or at least an explanation, would be found, even if he were fine, as he insisted most of the time, and all this became an experience in his past. This book comes too late for him, but it fulfills the promise I made him.

As a survivor of suicide I cannot even begin to express the devastation that is left in the wake of a young death. Unfortunately, I have seen that premature deaths, irrelevant of the cause, always have a much greater impact on us. One is always left with a sense of being cheated unfairly. I hope this book will prevent more than a few from being cheated at all.

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[32] Douglas Jacobs, Ed., *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

Present Knowledge & Terminology

When you (or a loved one) are faced with a mental problem or mood disorder severe enough to seek assistance, it is extremely important to know the language and the terms that the psychologists, psychiatrists or therapists use, the better to help yourself (or a loved one). In the quest to master aftoktognosis, given that 90 percent or more of suicides are related to mental disorders or personality disorders, this becomes doubly important. It is essential, because the terms they use aid in communicating more clearly to others our confused internal states. It is also important, because this will shed light on some of the limitations of the professionals' knowledge.

A note of caution is in order here: There are many well-intentioned doctors, yet in many cases they fail to explain their limitations, or what they don't know. It seems to be a part of human nature. Unfortunately, the limitations of medical knowledge or psychological theory seem to reflect the shortcomings of doctors. This doesn't mean they don't have good intentions or empathy for their patients. They are, for the most part, trying their best under extremely difficult circumstances.

Even though this section can be boring, I strongly recommend getting through it to get the gist and a general understanding. Also, it is very important to recognize the slight differences between one mental state and another; between one diagnosis and another; treatment and medical attention depend on it, like any other physical illness. Proper diagnosis is of the utmost importance, but at present it is difficult and can be more art than science. Pharmacological or therapeutic follow up will be determined by the initial diagnosis. Wrong diagnosis can have terrible consequences.

Published by The American Psychiatric Association, *The Diagnostic and Statistical Manual of Mental Disorders* is now in its fourth edition. It is known as DSM-IV-TR for short. I will simply call it the Manual. The Manual is occasionally revised to incorporate advances in knowledge and reflect changes in attitudes. It classifies mental disorders for clinical, educational and research settings. Its main purpose is to provide clear descriptions of diagnostic categories so that clinicians and researchers can communicate in a standard language.

The Manual is organized into sixteen major diagnostic classes. My focus will be primarily with the second, fifth, sixth and seventh classes, relating to deliriums, amnesia and dementia, schizophrenia and psychotics, depression and manic depression, and anxiety and panic attacks, respectively. I do this, because approximately ninety percent of people who commit suicide are suffering from these mental disorders.

I will discuss the criteria for some diagnosis following the classes that the American Psychiatric Association has established. Acquiring a basic understanding of delirium,

amnesia and dementia makes it easier to understand schizophrenia, depression and manic depression. Anxiety and panic attacks are often present simultaneously with the schizophrenia or depression, and increase the risk of suicide. It is important to review these conditions to better comprehend suicide. The American Psychiatric Association doesn't consider suicide as a class by itself, a separate disorder, but as part of other mental disorders.

The manual defines criteria for each diagnosis. When these criteria are met, specifiers can be added to the diagnosis. Examples of specifiers used are: Mild, Moderate, Severe, In Partial Remission, In Full Remission and Prior History.

There can be more than one diagnosis for a mental disorder. The principal diagnosis refers to a condition established to be chiefly responsible for admission to an institution. Sometimes it is difficult (and somewhat arbitrary) to determine which is the principal diagnosis when there are dual or multiple diagnoses.

Each diagnostic class has at least one, Not Otherwise Specified (NOS) category as well as a category when a medical (as opposed to mental) condition is suspected of causing the mental disorder. A medical condition will be determined to be responsible by the history, physical examination or laboratory findings relating to a medical problem or a chemical substance. In most cases, the mental disturbance has to be severe enough to disrupt normal social or occupational functioning.

The manual uses terms like "fluctuate," "impairment," "failure," "persist a few months," "superimposed," all of which can be subjectively interpreted. So, diagnosing any mental disorder can be imprecise and problematic. The manual only describes "abnormal" conditions; it does not present a theoretical framework to explain why these "abnormalities" occur.

Delirium, Dementia and Amnesia

The essential feature of a delirium is a disturbance of consciousness manifested as reduced clarity of awareness. The ability to focus, sustain or shift attention is impaired. This change in cognition may include memory impairment, disorientation, language disturbance or development of a perceptual disturbance. The disturbance can develop over a short time and can fluctuate during the day.

Delirium is often associated with a disturbance of the sleep-wake cycle, either excessive sleepiness in the day or wakefulness throughout the night. Many individuals might exhibit emotional disturbances such as anxiety, fear, depression, irritability, anger, euphoria and apathy.

Dementia is characterized by the absence of one or more of the normal cognitive functions (including memory impairment) that are due to direct physiological effects of a general medical condition, to the persisting effect of a substance, or combinations of the above. There are many different types: Dementia of the Alzheimer's Type, Dementia Due to Head Trauma, Dementia Due to Parkinson's or Huntington's or Picks, Substance Induced Dementia, NOS, etc.

The essential feature of dementia is the development of multiple cognitive deficits that include memory impairment and at least one of the following disturbances: aphasia (impairment of speech), apraxia (impaired ability to execute motor activities), agnosia (failure to identify objects) or a disturbance in executive functioning.

Yet, memory impairment occurs in both delirium and dementia, sometimes making the distinction difficult. Typically, symptoms in delirium fluctuate and symptoms in dementia are relatively stable.

Amnestic disorders are characterized by a disturbance in memory. This includes impaired ability to learn new information, or inability to recall previously learned information or past events. The ability to learn and recall new information is always affected in an amnestic disorder. Remembering previously learned or past events depends on the location and severity of the brain damage. Depending on the area of the brain affected, deficits might be predominantly verbal or visual. In some cases an individual might recall the distant past very clearly and recent events not at all.

Schizophrenia

The Manual does not attempt to explain why a mental disorder happens; it just describes it and defines its symptoms.

Included in the Fifth class we have Schizophrenia, Schizophreniform Disorder, Schizoaffective disorder, Delusional Disorder, Brief Psychotic Disorder, Shared Psychotic Disorder, Psychotic Disorder Due to a General Medical Condition, Substance-Induced Psychotic Disorder and Psychotic Disorder NOS (Not Otherwise Specified).

The narrowest definition of psychotic is restricted to delusions or prominent hallucinations, with the hallucinations occurring without the individual's understanding of their pathological nature. A less restrictive definition includes prominent hallucinations that the individual knows are hallucinatory experiences.

Delusions are erroneous beliefs and they may include a variety of themes; persecutory (can include being tormented, tricked, spied on or ridiculed), referential, somatic, religious, or grandiose. Delusions that express a loss of control over mind or body are considered bizarre; these include a person's belief that their thoughts have been taken away by some outside force or aliens—in the past it might have been devils—have been introduced into their minds, or that their body or actions are controlled by some outside force

The diagnosis of Schizophrenia involves a constellation of signs and symptoms associated with impaired occupational or social functioning, such as cognitive and emotional dysfunctions that include perception, inferential thinking, language and communication, behavioral monitoring, affect, fluency and productivity of thought and speech, hedonic capacity, volition and drive, and attention.

Schizophrenia is a disorder that lasts at least 6 months and includes at least one month of active-phase symptoms of at least two or more of the following: delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior,

and negative symptoms such as restrictions in the range and intensity of emotional expression (affective flattening), in the fluency and productivity of thought (alogia) and in the initiation of goal directed behavior (avolition).

An individual with Schizophrenia may display other features: inappropriate affect (smiling, laughing, silly facial expressions in the absence of an appropriate stimulus), anhedonia (manifested by loss of interest or pleasure), a Dysphoric mood (in the form of depression, anxiety or anger), and disturbances in sleep patterns. There can also be a lack of interest in eating, and there can be abnormalities of psychomotor activity (pacing, rocking or immobility). Many have poor insight into the fact that they suffer a psychotic illness, and this predisposes the individual to noncompliance of treatment. Depersonalization, derealization and somatic concerns might reach delusional proportions. Anxiety and phobias are also common.

In Schizoaffective Disorder, a mood episode must be concurrent with the active-phase symptoms of Schizophrenia, mood symptoms must be present for a substantial portion of the total duration of the disturbance, and delusions or hallucinations must be present for at least 2 weeks in the absence of prominent mood symptoms.

By definition, Schizophrenia differs from Schizophreniform Disorder on the basis of duration; otherwise the features are almost identical.

Depression & Manic Depression

It is very important to understand mood disorders because roughly eighty per cent of suicides are related to depression, either bipolar or unipolar. According to the Manual, Mood Disorders are characterized as having a disturbance in mood as the predominant feature. The DSM-IV-TR manual divides this section into three parts. The first part describes mood episodes, which do not have their own diagnostic codes, but are useful as they serve as the building blocks for disorder diagnoses. The second part describes the Mood Disorders. The criteria sets for most Mood Disorders require the presence or absence of the mood episodes described in the first part. The third part includes the specifiers that describe either the most recent mood episode or the course of recurrent episodes.

Major Depressive Episode: its essential feature is a period of at least 2 weeks where there is either a depressed mood or the loss of interest or pleasure in nearly all activities. The symptoms must persist for most of the day, nearly every day. The person describes the mood as depressed, sad, hopeless, or discouraged. Many individuals exhibit increased irritability. In children and adolescents, an irritable or cranky mood may develop rather than a sad or dejected mood. Loss of interest or pleasure is nearly always present. In some cases there is a significant reduction in sexual interest or desire. Appetite is usually reduced, but in a few cases it might increase and crave specific foods. The most common sleep disturbance is insomnia (not being able to fall asleep). But it can be middle insomnia (waking up in the middle of the night), or terminal insomnia (waking up too early). Less frequently there is oversleeping (hypersomnia).

Psychomotor changes include agitation (not being able to be still, pacing, rubbing skin, etc.) or retardation (slowed speech, thinking or body movements, pauses, muteness, etc.). Decreased energy, tiredness and fatigue are common. A sense of worthlessness or guilt can be present and many report impaired ability to think, concentrate or make decisions.

Frequently there might be thoughts of death, suicidal ideation, or suicide attempts. The range of thoughts can go from a belief that others would be better off if the person was dead, to transient but recurrent thoughts of committing suicide, to actual specific plans of how to commit suicide. The frequency, intensity and lethality of these thoughts can be quite variable. Although these behaviors are associated with suicide attempts and may be helpful in identifying a high-risk group, it is not possible to predict whether or when an individual with depression will attempt suicide.

A Major Depression Episode is especially difficult to diagnose when an individual also has a general medical condition (cancer, stroke, diabetes, etc.). Also, present frequently are tearfulness, irritability, brooding, obsessive rumination, anxiety, phobias, and excessive worry over physical health and complaints of pain. During a Major Depressive Episode, panic attacks that occur in a pattern that meets criteria for Panic Disorder are common in some cases.

Manic Episode: defined as a period during which there is an abnormally and persistently elevated, expansive, or irritable mood. This period of abnormal mood must last at least a week. This mood disturbance must be accompanied by at least three additional symptoms: inflated self esteem (ranging from uncritical self-confidence to marked grandiosity), decreased need for sleep, pressure of speech (speech is loud, rapid and difficult to interrupt), flight of ideas (much faster than can be articulated), distractibility, increased involvement in goal directed activities (excessive planning or participation in multiple activities), or psychomotor agitation, or excessive involvement in pleasurable activities with a high potential for painful consequences. If the mood is irritable, then at least four of the above symptoms must be present. Some individuals report having a much sharper sense of smell, hearing or vision. Mood may shift rapidly to anger or depression. Depressive symptoms may last moments, hours, or, more rarely, days.

The elevated mood may be described as euphoric, cheerful or high. Although the person's mood may initially have an infectious quality, those that know the person well recognize it as excessive.

Some individuals with a Manic Episode don't recognize that they are ill. They may engage in activities that have a disorganized or bizarre quality. When the Manic Episode passes, most individuals are regretful for their behavior.

Mixed Episode: is characterized by a period of at least one week in which criteria for both a Manic Episode and a Major Depressive Episode are present nearly every day. The individual experiences rapidly alternating moods accompanied by symptoms of Manic Episode and Major Depressive Episode.

Hypomanic Episode: is defined almost the same as a Manic Episode, except that the duration of the mood lasts at least four days, and in the list of additional symptoms,

delusions and hallucinations are not present. In contrast to a Manic Episode, a Hypomanic Episode is not severe enough to cause marked impairment in social or occupational functioning. The change in some might take the form of increased efficiency, accomplishments or creativity. Flight of ideas is uncommon and, if present, lasts for brief periods of time.

The essential feature of Dysthymic Disorder is a chronically depressed mood that occurs for most of the day more days than not for at least two years. Because these symptoms have become so much a part of the individual's day-to-day existence, they are often not reported (i.e., "I've always been this way").

Bipolar I Disorder: the essential feature is a clinical course that is characterized by one or more Manic Episodes or Mixed Episodes. Often individuals have also had one or more Major Depressive Episodes. Manic Episodes generally occur immediately before or after a Major Depressive Episode in a characteristic pattern for a particular individual. Recurrence is indicated by a shift in the polarity of the episode (when Depressive changes to Manic or Mixed or vice versa) or an interval between episodes of at least two months without symptoms.

Major Depressive Disorder: is a clinical course that is characterized by one or more Major Depressive Episodes without a Manic, Mixed, or Hypomanic Episode. There is the distinction between a Major Depressive Disorder, Single Episode, and a Major Depressive Disorder, Recurrent. For this manual an episode is considered to have ended when the full criteria for the Major Depressive Episode have not been met for at least 2 consecutive months (Full Remission.).

Bipolar II Disorder is the same as Bipolar I, except that instead of Manic Episodes the individual suffers Hypomanic Episodes.

Completed suicide (usually during Major Depressive Episodes) is a significant risk, occurring in 10%-15% of people with Bipolar I Disorder or Bipolar II Disorder, and 20% of people suffering Major Depressive Disorder.

Cyclothymic Disorder: The essential feature is a chronic, fluctuating mood disturbance involving numerous periods of Hypomanic symptoms and numerous periods of depressive symptoms. The hypomanic and the depressive symptoms are of insufficient number, severity, pervasiveness or duration to meet full criteria for Manic Episode or Major Depressive Episode respectively. However, it is not necessary that all hypomanic symptoms meet the criteria for a Hypomanic Episode. In the first two years any symptom-free intervals last no longer than 2 months, and this two-year period is free of Major Depressive, Manic or Mixed Episodes. After the initial two years of the Cyclothymic Disorder, Manic or Mixed Episodes may be superimposed, in which case both are diagnosed.

When other conditions are present, we speak of comorbidity. Comorbid conditions have been found to be associated with a poorer course over time, including longer time for recovery, faster time to relapse, poor medication adherence, and increased suicidality. The most common comorbid anxiety conditions with bipolar disorder are obsessive-compulsive disorder, phobias and panic disorder. The most common

personality include those from Cluster B (antisocial, borderline, histrionic, and narcissistic personality disorders) and Cluster C (avoidant, dependant and obsessive-compulsive personality disorders).[33]

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I share this I read to give a better feeling of the tremendous emotional roller coaster that some people suffer. Andrew Solomon, in his book, *The Noonday Demon, An Atlas of Depression,* chronicles the following from a bi-polar friend, "She was in Austin, Texas because that was where her boyfriend lived, but she was somewhat isolated and bored there. She had been too depressed to work, though she was interested in government service and hoped to get a job in the Texas statehouse. She told me she had taken Prozac, Paxil, Zoloft, Wellbutrin, Klonopin, BuSpar, Valium, Librium, Ativan, and of course, Xanax and was now on several of these as well as Depakote and Ambien. She was having trouble with her supervising psychiatrist, "so—guess what—off to doctor number forty-nine," she wrote in her letter.

Solomon answered her back, and next heard from her a month later. "The Depakote is not proving itself," she wrote. "I am frustrated by memory loss and shaky hands and stuttering and forgetting the lighter when it has taken me forty minutes to compile the cigarettes and the ashtray anyway. I am frustrated because these diseases seem to me so blatantly *multi*polar in many instances—it makes me wish Levi-Strauss had never brought our attention to binary opposition. *Bicycle* is about as far as I'll go with the prefix. I am convinced there are forty different shades of black and I don't like looking at this in a linear scale—I see it more as a circle and a cycle where the wheel is spinning too quickly and a desire for death can enter through any spoke. I thought of checking myself into the hospital this week, but I have been in there enough to know that I would not be allowed a stereo even with headphones, or scissors to make Valentine's Day cards, and that I would miss my dogs, and that I would be terrified without and would miss terribly, Peter, my boyfriend, who loves me through all the vomit and anger and unrest and no sex and that I would have to sleep in the hall by the nurses station or be locked in a room on suicide watch and so on—well, no thank you. I'm fairly confident that with the meds keeping me equatorial—between the two poles—I'll be okay."[34]

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Diagnosing a particular Mood Disorder can be difficult at times for various reasons, especially when the border between mild and normal can be blurry. And ultimately,

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[33] Sheri L. Johnson and Robert L. Leahy, Editors. *Psychological Treatment of Bipolar Disorder.* 2004.

[34] Andrew Solomon, *The Noonday Demon*.

since the mechanisms causing Mood Disorders are poorly understood, the solutions proposed, work poorly.

Depression, especially the first episode, can be triggered as a reaction to life events. However, today's prevailing view is that it is more a mixture of reactive and internal factors. How much is reactive and how much internal, probably varies widely among individuals.

Depression can be recurring, and can be, more likely than not, triggered by outside factors. Over time, the cycling will take a life of its own, and depression will be triggered internally. The sooner the depression is attacked, the more likely the cycle can be broken. The best attack is a combination of medication and therapy.

Panic Attacks

According to the Manual, the essential feature of a Panic Attack is a discrete period of intense fear or discomfort in the absence of real danger. At least 4 of 13 somatic or cognitive symptoms accompany the fear. The attack has a sudden onset and builds to a peak rapidly (generally in ten minutes or less) and is often accompanied by a sense of imminent danger or impending doom and an urge to escape. The anxiety that is characteristic of a Panic Attack can be differentiated from generalized anxiety by its discrete, paroxysmal nature and its typically greater severity.

There are three characteristic types of Panic Attacks: unexpected (uncued—there is no internal or external trigger), situationally bound (cued—almost invariably occurs immediately on exposure or anticipation of a trigger), and situationally predisposed (similar to situationally bound but not invariably associated with the cue and not necessarily occurring immediately after exposure, e.g., attacks sometimes happen while driving and sometimes they don't). Each type is defined by the relationship between the onset of the attack and the presence or absence of situational triggers that can include cues that are internal or external.

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I am asleep. I dream I am in my house. It is dark and late at night. I hear a movement or noise emanating from the kitchen. I consciously decide to move in darkness because the intimate knowledge of the layout of my house would give me an advantage over the intruder, and go investigate. My wife is sleeping soundly in our bed and my two sons are sleeping in their bedroom next to ours as I approach the kitchen. I enter the kitchen very quietly, with all my senses trying to perceive what or who is in there. Suddenly, from behind, I feel two steel hands grab me by the upper arms, close to the shoulders. The hands are cold and hard like metal. The pressure is so great that I can barely move my hands below the elbows pressed to my side. As the pressure increases, I am lifted from the floor. I realize that the strength of this grip pressing into my arms and squeezing my arms into my body can easily kill.

Then, the pressure is slowly, inexorably increased. At this point I am not concerned with my own fate anymore; I just want to yell out a warning, so hopefully my wife and sons will wake up and escape, and run away from whatever is compressing me. I open my mouth to yell but no sound comes out! I am so terrified that I can't even emit a sound.

If I try to describe how I felt in the dream, I would definitely have to say I was having palpitations, sweating, trembling and shaking; I was short of breath; there was definitely discomfort and fear of dying, even if I felt no chest pains, dizziness or lightheadedness (at least four of thirteen of these symptoms have to be present to diagnose a panic attack). Isn't this what extreme fear or panic is like?

The onset was very quick, almost instantaneous. I grant you I didn't feel nausea or abdominal distress, there was no derealization or depersonalization, no paresthesias, I didn't feel like I was going crazy and I didn't have hot flashes or chills. I was simply as scared as I have ever been. Then I woke up.

I experienced nine (five more than required) of the symptoms that are required, so unless the fear was caused by something real, I guess I could say that, at least in a dream, I have experienced a Panic Attack (of course it could be argued that I simply felt panic).

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The occurrence of an unexpected Panic Attack is required for the diagnosis of Panic Disorder (With or Without Agoraphobia). Situationally bound and situationally predisposed attacks are frequent in Panic Disorder but also occur in other anxiety and mood disorders.

Agoraphobia: because agoraphobia occurs in the context of Panic Disorder With Agoraphobia and Agoraphobia Without Panic Disorder, its criteria are provided separately.

The essential feature of Agoraphobia is anxiety about being in places or situations from which escape might be difficult (or embarrassing) or in which help might not be available in case of a Panic Attack. The anxiety typically leads to the avoidance of many situations and may include being alone in or out of the home; being in a crowd; traveling in an airplane, being in a closet or elevator. In some cases the individual can face the feared situation, but with considerable dread, or can face it if accompanied by a friend.

Panic Disorder: the essential feature is the presence of recurrent, unexpected Panic Attacks followed by at least one month of persistent concern about having another Panic Attack, worry about the consequences of the Panic Attack, or there is a significant behavioral change related to the attacks. Panic Disorder should be distinguished from other mental disorders (Anxiety, Mood and Psychotic Disorders) that have Panic Attacks as an associated feature.

Specific Phobia: the essential feature is a marked and persistent fear of clearly discernible, circumscribed objects or situations. Exposure to the phobic stimulus invariably produces an anxiety response. The response may be a situationally bound or situationally predisposed Panic Attack.

OCD, Posttraumatic Stress Disorder & Anxiety

According to the Manual the essential features of Obsessive-Compulsive Disorder are recurrent obsessions and compulsions that are severe enough to occupy more than one hour a day or cause marked distress or significant impairment. At some point the person recognizes that the obsessions or compulsions are excessive or unreasonable.

Obsessions are persistent ideas, thoughts, impulses, or images that are experienced as intrusive and inappropriate and that cause marked anxiety or distress. The most common obsessions are about contamination (e.g., through shaking hands), repeated doubts ("Did I turn the stove off?"), the need to have things in a particular order, aggressive or horrific impulses (e.g., to hurt one's child or yell obscenities), and sexual images (a recurrent pornographic image).

Compulsions are repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (counting, repeating words silently), the goal of which is to prevent or reduce the anxiety or distress, not to provide pleasure or gratification.

Most adults recognize the excessiveness of their obsessions and compulsions. However, there is a broad range of insight into the reasonableness of the obsessions or compulsions. When attempting to resist a compulsion, the individual may feel a mounting sense of anxiety or tension that is relieved by yielding to the compulsion.

Generalized Anxiety Disorder: The essential feature is excessive anxiety and worry, occurring more days than not for a period of at least six months, about a number of events or activities. The individual finds it difficult to control a worry. The anxiety and worry are accompanied by at least three of these additional symptoms: restlessness, easily fatigued, difficulty concentrating, irritability, muscle tension and disturbed sleep. The focus of the worry must not be another disorder.

Acute Stress Disorder: the essential feature is the development of characteristic anxiety, dissociative, and other symptoms that occur within a month after exposure to an extreme traumatic stressor. While experiencing the traumatic event or after the event, the individual has at least three of the following Dissociative symptoms: a sense of numbing, detachment, or absence of emotional responsiveness; a reduction in awareness of their surroundings; derealization; depersonalization; or Dissociative amnesia. After the trauma, the event is persistently re-experienced, and the individual displays marked avoidance of stimuli that might arouse recollection and has marked symptoms of anxiety and increased arousal. If the symptoms last more than four weeks, the diagnosis of Posttraumatic Stress Disorder may be applied.

Posttraumatic Stress Disorder: The essential feature is the development of characteristic symptoms following exposure to an extreme traumatic stressor involving direct personal experience of an event that involves actual or threatened death or serious injury to oneself or others; or learning about unexpected or violent death, serious harm, or threat of death experienced by a family member or a close associate. The person's response must involve intense fear, hopelessness, or horror. The characteristic symptoms include persistent re-experiencing of the traumatic event,

persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, and persistent symptoms of increased arousal. The full symptom picture must be present for more than one month, and the disturbance must cause significant distress or impairment in social or occupational functioning.

The traumatic events include, but are not limited to, combat, personal assault (e.g., sexual assault, robbery, etc.), loss of a loved one, being kidnapped, torture, incarceration, natural or man-made disasters, etc. Commonly the person has recurrent and intrusive recollections of the event or recurring distressing dreams during which the event is replayed. In rare instances the person experiences dissociative states during which the event or parts of the event are relived (flashbacks).

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Five months after my son Mitch took his life, I believe, that for the previous four months I had been tottering on the brink of Posttraumatic Stress Disorder. I continuously experienced a feeling of anxiety. The anxiety woke me up at night and I had a hard time falling back asleep. When I woke up in the morning, I immediately became anxious. I had a recurrent vision, wide-awake, where I see Mitch starting the act. What is so strange is that, even though intellectually I know I wasn't there, the vision has a quality to it that feels like a real past, witnessed event. This is what makes it posttraumatic stress disorder—imagined events take on the quality of real events, because of the intensity of the emotion

I can still see him; he is in the landing of the stairwell in our apartment in New York. He is unafraid, even smirking, with an expression of concentration, such as he would show when preparing to do a complicated skateboard trick. He leaps, is in mid-air . . .

Then, the vision changes. The light is different, perhaps a few hours later. The sun has set. It is dark. Mitch is there, hanging immobile in the dark, at the bottom of the stairwell. The vision kept repeating itself as if it had been a real past event; I kept seeing him as he does it, as if I had been there watching, as if I am there watching.

A month later, I was in our apartment in New York, still suffering from these visions. I had the opportunity to check with a friend who discovered my son's body. Eerily, except for a small element in his clothing, every detail was like I saw it. After inspecting the stairwell, what is strange was that my angle of vision was such that I would have to have been inside a wall of the stairwell to have the proper perspective.

Personality Disorders

A personality disorder is an enduring pattern of inner experience and behavior that deviates from the expectations of the individual's culture (i.e., considered not normal), is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time and leads to stress or impairment. There are several types of personality disorders. They are grouped in three clusters based on descriptive similarities. Cluster A includes

Paranoid, Schizoid, and Schizotypal Personality Disorders. Individuals with these disorders often appear odd or eccentric. Cluster B includes the Antisocial, Borderline, Histrionic and Narcissistic personality Disorders. People with these disorders often appear dramatic, emotional, or erratic. Cluster C includes the Avoidant, Dependent, and Obsessive-Compulsive Personality Disorders.

The manual attempts to discern personality traits, which are enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts. Only when these traits are maladaptive or cause significant impairment or stress are they considered Personality Disorders.

The diagnostic criteria for Borderline Personality Disorder is a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood in one of several of the following contexts: (1) frantic effort to avoid real or imagined abandonment; (2) unstable and intense interpersonal relationships; (3) identity disturbance; (4) impulsivity that is self-damaging; (5) recurrent suicidal behavior or gestures; (6) affective instability due to a markedly reactive mood; (7) chronic feelings of emptiness; (8) inappropriate, intense anger or difficulty controlling anger; and (9) transient stress-related paranoid ideation or severe dissociative symptoms.[35]

Borderline Personality disorder is the only diagnosis in the Manual for which suicidality is a criterion. A continuous struggle with suicide is a fundamental part of their illness, even though their suicide rate might not be much higher than that of other mental disorders. Suicide or suicidality are not a diagnostic category, which in my opinion it should be because suicide happens across many mental disorders, and in most of these, it is only in a minority of cases. Suicide should be treated separately and differently. The wording in the manual differs suggesting a qualitative difference between suicidality of people with borderline personality disorder and those with depressive disorder. In the case of a major depressive disorder, patients may have "recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide. For borderline personality disorder, patients present recurrent suicidal behavior, gestures or threats, or self mutilatory behavior. This suggests that suicidality in depressed patients is more directed inwards psychologically, whereas in borderline personality disorder it is more typically directed outwards as "in-your-face" behavior. This is true not only of the more familiar suicidal gestures of borderline patients, but also of completed suicides.

A study in 1996 noted that 44 percent of completed suicides in patients with borderline personality disorder were witnessed, compared to 17 percent of suicides with other diagnoses.[36]

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[35] *Diagnostic and Statistical Manual of Mental Disorders.* DSM-IV-TR.

[36] B.S. Runeson, J. Beskow, M. Waern, *The suicidal process in suicides among young people.* Acta Psychiatr Scand 1996;93:35-42

Current Approaches to Suicide & Mental Disorders

Leston Havens at the 1997 Cambridge Hospital Harvard Medical School Suicide Conference stated, "Our psychiatry remains the way surgery was before anesthesia. You got four big people to hold a person down. You gave them the biggest slug of whiskey you could get into them without killing them. For an appendectomy, the masters were measured in fifty to sixty seconds, a hernia repair was a little quicker. That's all the time you had." Havens went on to state that even though we need our patients to stay awake, there are at least three psychological analgesics that should be used [in therapy]: protection of self-esteem (one thing that works wonders is the willingness to admire the patient), understanding (seeing the patient's experience from their point of view), and, perhaps most vital is providing a future (in the sense that the world and life can be made better).[37]

The comparison between open surgery without anesthesia and therapy is perhaps not immediately obvious. But psychological pain and distress can be even more painful and intolerable than physical pain, and definitely more confusing.

As we have seen, about 90% of suicide cases present a mood disorder. At present most therapeutic methods used to aid patients suffering from depression, manic depression or schizophrenia, are only partially successful some of the time. These poor results are rooted in the fact that most therapies are based on wrong premises, or at best are based on premises that are tangentially correct; they do not address the root causes, only some of the side issues. I need to emphasize that at the time of writing, the limitations of our understanding of suicide limit what can be done. Present suicide theories are hopelessly inadequate. This book presents a newer and better approach, and I hope, this theory will initiate changes which lead to better therapies.

When it comes to suicide, what do the professionals really know? It is accepted knowledge that there are some suicidal patients who simply will not be effectively treated over the short term, those who present chronic and severe psychopathology. We need to address the question of why not? If we understand the why of suicide, then perhaps we can find how to treat this condition.

There have been many reasons or theories proposed to explain suicide. Fantasies of reunion with a deceased loved-one after death have been frequently reported.

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[37] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

Others imagine themselves infested with a persecutor that they identify with their bodies and attempt suicide in order to escape it. Still, others blame their bodies for life's disappointments and carry grudges against them for years. Other's report that all would be better off if they died, or that loved one's wish them dead. Yet, these are simplistic explanations. The fact of the matter is that people don't commit suicide because their girl-friend left, their wives died, or because they lost money in the stock market. Suicide is much more than the avoidance of a bad feeling or situation.

Emile Durkheim, the renowned French Sociologist published, late in the nineteenth century, *Le Suicide.* It is among the first modern attempts, through the use of statistical methods and bringing several disciplines to bear, to present a rigorous study on suicide. In the first chapter Durkheim argues inconclusively that "suicide itself is either a disease in itself, *sui generis,* a special form of insanity; or it is regarded, not as a distinct species, but simply an event involved in one or several varieties of insanity, and not to be found in sane persons. If suicide can be shown to be a mental disease with its own characteristics and distinct evolution, the question is settled: every suicide is a madman."

Durkheim tries to fit suicide into the accepted psychological ideas of his time, where in traditional terminology of mental pathology certain restricted deliria are called monomanias. Thus he describes a monomaniac as a sick person whose mentality is perfectly healthy in all respect but one; he has a flaw, clearly localized. It follows that if there is a suicidal mania it can only be a monomania. From this point of view the basis of mental life is the same in the monomaniac and the sane person; only, in the former, a specific psychic state is predominantly detached from this common basis.

Durkheim tries to use the statistical method to discern the relation between suicide and race, latitude, time of day (or night). This reflects how deep the lack of understanding of suicide was. He even looks into imitation as reason for suicide, as if suicide was contagious. At one point he says, "a homicidal impulse may be transferred from one person to another even though there is only a chance and temporary contact between them. They need not have any intellectual or moral community between them nor exchange services nor even speak the same language, nor are they any more related after the transfer than before." Not surprisingly, many of his findings are not cited today.

He goes on to borrow a suicide classification by Jousset and Moreau de Tours. The four following types are described: 1) Maniacal suicide: The patient kills himself [or herself] to escape from an imaginary danger or disgrace, or to obey a mysterious order from on high. 2) Melancholy suicide: related to a general state of extreme depression, causing the patient no longer to realize sanely the bonds which connect him with people and things about him. 3) Obsessive suicide: Suicide is caused solely by the fixed idea of death which, without clear reason, has taken complete possession of the patient's mind. It is an instinctive need beyond the control of reflection and reasoning, like the needs to steal, to kill, to commit arson, considered as other varieties of monomania. And 4) Impulsive or automatic suicide: It is equally unmotivated as obsessive suicide; it has no cause either in reality or the patient's imagination. It results from an abrupt and immediately irresistibly impulse.

Durkheim goes on to argue in detail the social causes of suicide concluding from this perspective that there are three types of suicide.

The egoistic suicide, where the individual feels (more or) less a part of society (excessive individuation); where essentially the bonds to family and society are indirectly proportional to suicide rates.

The altruistic suicide, where the individual becomes detached from society (insufficient individuation). In this case, suicides fall into three categories: suicides of old men or stricken with sickness, of women on their husband's death, of servants or followers on the death of their chiefs. The altruistic suicide is seen as a duty.

The anomic suicide, where economic crises increase the suicide rates. Among the many statistics Durkheim gives, he mentions the famous crash of the Paris Bourse in the winter of 1882. In France, from 1874 to 1886 the annual increase in suicide rate was 2 per cent. In 1882 it increased 7 per cent.[38]

Others have viewed suicide as guilt and aggression. K.A. Menninger in 1936 believed that masturbation had an important connection to suicide. He stated, "It has been observed that suicidal attempts sometimes follow the interruption of an individual's habitual auto-erotic activities . . . the mechanism by which the suicide is precipitated are the same: the masturbation occasions a heavy burden of guilt, because of the unconscious mind it always represents an aggression against someone. This guilt demands punishment and as long as the auto-erotic practices are continued, the punishment is bound up in the satisfaction, since masturbation is imagined by many to be a great danger to health, and to one's life both in this life and the hereafter." Menninger postulates that when masturbation is interrupted, the needed punishment is no longer "bound up in the satisfaction," and suicide results as a form of self-punishment.[39]

Others have tried to categorize suicides depending on which unsatisfied needs are seen as the cause, as if suicides were really a rational, even though highly, distorted behavior. Following this logic, first, we have suicides related to thwarted love, acceptance, and/or belonging; second, suicides related to frustrated needs for achievement, autonomy, order, and understanding; third, suicides related to assaulted self-image and avoidance of shame, defeat, humiliation, and disgrace; fourth, suicides related to loss of important relationships, grief and bereftness; and fifth, suicides related to excessive anger, rage and hostility.

Gardner and Cowdry (1985) conceptualized suicidal behaviors in four categories, each having specific affective states, motivations and outcomes: melancholia, despair, and true suicidal acts; impulsive, nihilistic, or retributive rage; communicative parasuicidal gestures; and self-mutilation or overdose to relieve dysphoria.[40]

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38 Durkheim, Emile, *Suicide, A Study in Sociology.* 1951.

39 Thomas Joiner, *Why People Die By Suicide.* 2005.

40 D.L. Gardner and C.W. Cowdry, *Suicidal and Parasuicidal behavior in borderline personality disorder.* Psyciatr Clin North Am 1985;8:389-403.

Many studies formulate an explanation of suicide in terms of factors or behaviors that have been typically found to be associated with suicide.[41] These factors or behaviors then become causes of suicide, when in reality they are symptoms of other more profound mental states that lead to suicide. Some of the factors used this way are: hostility, the withdrawal of social support, or sado-masochistic tendencies. Some of the behaviors mentioned as causes of suicide, primarily from a traditional psychoanalytic point of view, include most self-directed aggression that stops short of suicide, such as martyrdom, neurotic invalidism, antisocial behavior, psychosis and substance abuse; also, mutilation, malingering and even accidental proneness. Many times, what is really an aspect of grieving and mourning, such as, anniversaries of losses and holidays, which re-awaken keen feelings of abandonment, loneliness, fury, guilt, defectiveness, isolation and hopelessness, are also used as superficial explanations of suicide.

Many people bear up under initial insults to their adaptive behaviors, which leads to the theory that suicidal coping breaks down gradually, in some cases after about 40-50 years of certain kinds of lives. This leads to the point of view that certain patterns of behavior leading to suicide are "suicidal careers."

Some, because suicide is a violent act, insist it usually requires some minimal aggressive energy.[42] We will explore how and under what conditions this is possible, but suicide is definitely not an unconscious murderous desire, as Freud argued.

Aaron Beck, as way back as 1967, was *not* able to confirm the presence of internalized aggression in depressed patients. Nevertheless, hostility seems to continue to permeate most suicide models. There have been attempts at creating suicide prediction scales. One of the Best is the Hopelessness Scale, which consists of 20 true-false self-report statements. The scale differentiates among suicide threateners, attempters and controls. The scale has a consistency (when the tests are repeated) of 93 percent. A 10-year follow-up for eventual suicide showed 91 percent sensitivity for inpatients, and a three and a half year follow-up showed 94 percent sensitivity for outpatients

Hopelessness is correctly identified as a component of suicide. Hopelessness intertwined to depression and only in certain situations, as we will see, is one of the true components of the roots of suicide.

Many models or theories of suicide are outmoded, and even though they have been proven wrong on occasion, continue to be used for lack of something better. It is interesting to understand the simple philosophical underpinnings on which they are based, as they will give us insight into how today's approach to suicide has been formulated.

Buie and Maltsberger summarized the state of mind of a person most likely to complete a suicide as having two types of imperative impulses: murderous hate and an urgent need to escape suffering.[43]

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41 Armand M. Nicholi, Jr., M.D., Editor, *The Harvard Guide to Psychiatry.*

42 R.W. Maris., *Assessment and Prediction of Suicide.*

43 D.H. Buie, and J.T. Maltsberger. *The practical formulation of suicide risk.* Cambridge, Mass.: Firefly Press, 1983.

In psychoanalytic theories, self-destruction is viewed as an effort to restore the balance of wounded self-esteem. From the psychoanalysis view point, self-esteem or self-respect is the most basic condition to be guarded if life is to continue. The main job of a psychoanalyst confronted with a suicidal person is the restoration and maintenance of the individual's narcissistic equilibrium.[44] These ideas go back to the seventies and are as outmoded as much as the struggle for supremacy of the ego, the id and the superego. The fact that these ideas are presented in the revised *The Harvard Guide to Psychiatry* (1999), seem to show that they are still in vogue in many psychiatric circles.

In the preface of the Harvard Medical School Guide to Suicide Assessment and Intervention (1999) it mentions, "Given that suicide is a multidimensional, complex clinical problem, there is no correct method of assessment and treatment." If this means that every individual is unique, I would agree. If this means that suicide is different in every individual, then, I disagree. All suicides share some mental conditions or states, and this is where the challenges and opportunities exist to not only combat suicide, but many other mental disorders as well. However, at present, most experts on suicidology agree that reliable prediction of individual suicide is impossible.

Avery Weisman points out that death itself may not be the wish of the suicide victim, but that the wish to die signals his conviction that "his potential for being someone who matters has been exhausted." Gregory Rochlin, in a creative transformation of the "death instinct" theory, points out that suicide itself, like other forms of aggressive action, serves to restore bruised or shattered self-esteem. Self-inflicted death is seen as an honorable way out of a person's crisis of self esteem, in which hopelessness and helplessness are not only the key subjective features for the individual, but also the most telling clues that suicide is possible.[45]

From a biosocial perspective, suicidal behaviors are viewed as problem solving behaviors that fix negative emotional arousal and distress directly (i.e., by ending life, and presumably pain) or indirectly (i.e., by expectations of collecting insurance money for one's children), or are viewed as inevitable outcomes of unregulated and uncontrollable negative emotions. Marsha Lineham has further hypothesized, among those who are chronically suicidal, that this pattern of dysregulated emotion and behavior is a result of an initial temperamental or biological disposition to emotionality, combined with an invalidating rearing environment.[46]

Shneidman, among others, has stated that suicidal individuals are prone to fallacies in their ideational process; that the wish to kill, the wish to be killed and the wish to die are evident; and that the suicidal person departs with hate and self-blame.[47] This is a gross simplification, where anger is explained away as self-hate, instead of a natural reaction to a particular threat. We will see later how anger can be part of a process that

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44 Armand M. Nicholi, Jr., M.D., Editor, *The Harvard Guide to Psychiatry.*

45 Ibid.

46 Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

47 R.W. Maris, *Assessment and Prediction of Suicide.*

ends in suicide. And self-blame is seen as some form of guilty reaction to the suicidal ideations, as if guilt could be part of self-destruction.

I mention some of these suicide theories because they continue to guide, for lack of a better explanation, the approaches of most practitioners. It is important to understand that present therapies are based some way or another in these theories. A better model of human behavior, and more specifically the human brain is needed in order to gain better insight in to how suicide, a drive so contrary to life becomes possible. My intimate knowledge of my son allowed me to see how most of these theories or models of suicide didn't apply to his case. A more general theory of suicide, one that can explain most all suicides, based on a new neurological model, is presented later. Hopefully, it will aid in producing better therapies and pharmacologies.

I review what is done currently, which leaves much to be desired, because that is what we can do today. The good news is that there is great room for improvement just by gaining a better understanding.

There is strong evidence that many depressed and suicidal patients seek care from their primary care physician. Perhaps half of all depressed patients receive treatment from an internist or family practitioner. Since 75 percent of suicides have had contact with a physician within six months prior to their deaths, and that 50 percent of suicides have never had any psychiatric care, it becomes logical, if not imperative, that primary care physicians should be concerned and be familiar with suicide. The lack of a comprehensive model or theory of suicide is why most physicians cannot be concerned: Why bother if all is so uncertain when assessing, predicting or even understanding suicide?

The different health organizations should take responsibility to educate medical colleagues about key disorders that increase suicide risk, and pass on frequent updates on new medications, possible drug interactions, the role of stress and other symptoms. This is of the utmost importance if primary care providers are to play an active role in suicide prevention.

A comprehensive review of suicide intervention shows that three studies had positive findings, but each has identifiable limitations, some which are considerable. Termansen and Bywater,[48] not surprisingly, found that what essentially was described as intensive care management by volunteer workers reduced subsequent suicide attempts during a three-month follow up period, relative to those receiving no follow-up care. This should not be surprising. Common sense dictates that if you tie yourself to a suicidal person, the chances of them committing suicide would diminish greatly, if not disappear completely. And of course, when all else fails, what else can you do? It is common sense that the more care, of any kind, a suicidal person receives, the less likely they will commit suicide.

Similarly, a study by Van Heerignen et al,[49] explores the use of home visits by a community nurse in enhancing treatment compliance and reducing subsequent

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[48] P. Termasen &C. Bywater (1975) *S.A.F.E.R.: A follow up service for attempted suicide in Vancouver.* Canadian Psychiatric Association Journal, 20, 29-34.

[49] M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior.*2001.

attempts in comparison to usual outpatient care (i.e., without home visits targeting treatment compliance). The study, however, excluded the highest-risk cases, limiting the usefulness of the study. Again, common sense dictates this should be expected. If a suicidal person following a pharmacological treatment, or therapy, is unsupervised, naturally, the possibility of a relapse increases.

In another study, Morgan et al. found that improved access to 24-hour emergency services over the period of a year following a first suicide attempt significantly reduced subsequent attempts among those in the group relative to those receiving managements as usual after an attempt (i.e., ranging from inpatient psychiatric admission to referral back to the primary care physician).

According to Motto (1976) simple follow-up letters and telephone calls to those refusing treatment after presenting in crisis did not reduce suicide rates over a 4-year follow-up period, although a favorable trend was observed. This is not surprising. People that have been suicidal are more likely to become suicidal again. However, investigators noted an encouraging trend after four years, with fewer suicides among those receiving the simple follow up contacts. This leads us to consider that the ones that survived four years after their first attempt understand better the seriousness of their condition and respond to the calls or letters, as opposed to those that never received anything.

All studies on suicide focus on patients that, forgive the oxymoron, survived a failed suicide attempt. Many people didn't intend to kill themselves, but engage in suicidal gestures, with the intention not of dying, but of drawing attention to their need for help. In practice, of course, the intentions of people that engage in suicidal behavior may be so ambivalent and murky that even to that person there will never be a clear answer as to whether he or she intended suicide, or was doing it for other reasons.[50]

In a study of suicide intervention looking into, "Who was primarily involved in trying to save the suicide attempters life?" in the case of suicide completers compared to suicide attempters, 59 percent of the interveners and 78 percent of suicide interveners, respectively, were family members, friends, police/fireman or physician. It is of interest to note that psychotherapists, crisis workers, or suicide prevention center workers reported no interventions for either attempters or completers.[51]

Hardly anyone is investigating first-successful-suicide-attempts, for the simple reason that they are not here to ask them anything. The mind is impervious and we can only speculate as to what went on in the minds of these individuals during those last moments or even hours before their death. There is a procedure called a suicide autopsy, where through interviews a reconstruction of the suicide victim's last weeks or days is done in an attempt to understand what happened.

A survivor of a failed attempt is clearly tagged as a potential suicidal case. He or she is obviously a high risk: if they have been there once, they can go there again. It is at

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[50] Ibid.

[51] R.W. Maris, *Assessment and Prediction of Suicide.*

this point that many safeguards are implemented. In general, very few safeguards are put in place in most cases when "not suicidal" is the label used, for the simple reason that there is no understanding of who is potentially suicidal or at risk. At present there is no theoretical scientific framework to help us determine this with precision.

Once a potential suicidal patient is identified, (because he or she attempted suicide, or he or she openly admits to having suicidal thoughts) the obvious is implemented: intensive follow-up, case management, telephone contacts, letters, home visits and more to assure treatment compliance, family warnings and education. Access to increased emergency services and clearly stated crisis plans are outlined, which reduce subsequent attempts and service demand by first-time suicide attempters.

It is a well-established fact that in most instances prediction of individual suicidal behavior is nearly impossible to achieve by the existing knowledge. Also, the use of an actual suicide event as a criterion of antecedent suicide risk is questionable, because very high-risk individuals sometimes survive suicide attempts and never try it again, and "low-risk" persons end up committing suicide. Furthermore, those who eventually attempt suicide may do so many years later, in a mental state and under conditions that may have little or no relationship to the situation at the time suicide risk was assessed.[52] The world is a dynamic place with internal and external conditions changing quickly.

In spite of all this, three important suicide identifiers are continuously used: previous suicide attempts, the diagnosis of depression or substance abuse, and a history of suicide in first-degree relatives. Present screening tests for suicide are neither sensitive nor specific. Use of risk factors to identify high-risk patients always yields numerous false positives, and those criteria used to define low-risk always yield some false negatives.[53]

The available suicide-treatment studies are basically broken into two categories: short-term treatment (less than six months) and longer-term therapy (more than six months). *Only therapies offering a variant of the cognitive behavioral therapy showed some positive findings. All other treatments showed a negative efficacy.* Even studies that addressed an additive component to short term treatment, combination of home-visits, telephone contact, flexibility in treatment scheduling, found no appreciable impact on subsequent suicide attempts over periods ranging from six to twelve months.[54]

Home visits improve patients' treatment compliance compared to those receiving weekly outpatient care. Improved compliance, however, does not translate into a significant reduction in subsequent attempts over the 12-month follow-up period (i.e., 10% with subsequent attempts for those receiving home visits vs. 15% for traditional outpatients). Another study by Chowdry et al. (1973) found that home visits, more frequent outpatient

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[52] Ibid.

[53] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

[54] M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior.*2001.

appointments, and improved access to emergency services did not reduce subsequent attempts among multiple attempters in contrast to treatments as usual.

However, a study of cases considered the most high-risk suggest that intensive outpatient treatment, irrespective of approach, is effective.

If a group of people got together and only knew the statistics provided in the first section of this book, it is highly likely that they would reach the expert conclusions presented in the last few paragraphs—you just need some common sense.

Most of the cognitive-behavioral therapies (at present the most effective in dealing with mood disorders) assume that there is a core problem that needs to be solved. Therefore, this results, not in less suicide attempts, but in less suicidal ideation and related symptomatology, such as depression, hopelessness and loneliness over follow-up periods ranging from three months to one year. Given the assumption (by CBT) that a core problem causes suicidal behavior, it should not be surprising that the integration of problem-solving as core intervention is obviously responsible for the reduction of suicidal ideation, depression, hopelessness and loneliness. Yet, suicide attempts do not decrease. This seeming paradox is resolved if we understand that suicide is not caused by a core problem. People, in general, simply do not want to kill themselves.

It is accepted knowledge that reducing suicide attempts requires longer-term treatment targeting the specific skill deficits [assumed to be] related to suicidality, such as emotion regulation, poor distress tolerance, anger management, interpersonal assertiveness, as well as other enduring problems such as interpersonal relations and self-image disturbance (personality disorders).[55] I say, "Assumed to be (in parenthesis) related to suicidality," because I will show that these peripheral issues are manifestations and consequences of certain mental states that might or might not lead to suicide, rather than the cause of suicidal behavior.

Practical training in suicide assessment and management in most graduate programs is limited, and what little there is is terribly inadequate. In 1989, less than 35 percent of graduate programs in clinical psychology offered any formal training in the study of suicide. Not only is graduate training limited, but it can also be hard to get additional training in suicide assessment and management after graduation, forcing many to pick and choose from and array of conflicting sources of information and various schools of thought.[56] With the rise of suicide rates, especially among the young, this has been changing. One study of 300 clinical counseling students noted that 99.2% reported contact with a suicidal client (Dexter-Mazza and Freeman, 2003). Studies by McAdams and Foster at Ohio State University in 2000 showed that graduate students who have the severe misfortune of losing a client or student to suicide experience significant emotional stress; and this is another reason why counselor education programs

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[55] Ibid.

[56] Thomas H. White, *How to Identify Suicidal People,* 1999.

should be invested in proper suicide prevention training. Professional counselors who experienced a client loss from suicide suffered significant levels of stress similar to the loss of a parent. In many of these cases, the counselors themselves needed treatment to process the grief. In comparison, graduate students in counseling who experienced the loss of a client suffered even more severe levels of grief than professionals, resulting in melancholy, atonement, and narcissistic avoidance. Therefore, because of the high probability of contact and potential consequences of treating suicidal clients and students, it behooves counselor education programs to incorporate adequate training, both in quality and quantity, to prepare students for the experiences that are likely to occur during training and professional experience (McAdams and Foster, 2000).

In a five-year analysis of lawsuits brought against psychiatrists, there were more claims resulting from suicide of a patient than from any other cause.[57] Sadly, skyrocketing malpractice suits against mental health professionals over the past twenty years, with the failure to prevent suicide as the most common cause of litigation, are forcing a new attitude to how we view, assess and manage suicide.

I will show why, at present, the most important, and almost the only element of success for suicide intervention is based on the therapeutic relationship. This relationship needs to be one of prominent attachment, at least initially. The quality of this attachment determines subsequent treatment success. This attachment during therapy goes to one of the roots of suicide. It is the one thing the patient can count on and gives them hope. The loss of hope is, currently, the one element correctly recognized as an integral part of the chain of conditions necessary for suicide to occur.

Amazingly, the most common treatment intervention for suicidality—hospitalization—continues without a single controlled study regarding its efficacy, differential effect, or actual need based on severity or complexity of diagnostic presentation.

Few studies have shown any suicide reduction through psychiatric treatment of any kind. To quote D. Lester in *Understanding and Preventing Suicide: New Perspectives* (1990), "The effectiveness of individual psychiatric and psychotherapeutic treatment remains unproven for suicide prevention." However, the problems in documenting that psychiatric treatment can reduce suicide risk should not discourage efforts to prevent suicide but instead call forth redoubling our hard work to do so. It is probable that if studies were conducted on whole treatment systems—medication with therapy with close supervision, for example—significant effects on the outcomes for patients would be shown.

A wide range of theories has been offered, each emphasizing a distinctive feature or aspect, or characteristic of suicide or suicidal behavior, frequently to the purposeful exclusion of others. We find various approaches to suicide: epidemiological, philosophical, sociocultural, sociological, psychodynamic, psychological and biological.

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[57] Ibid.

The scientific study of suicide began with the work of the French sociologist Emile Durkheim, In his 1987 treatise, *Le Suicide,* he attributed suicide to the failed relationship of the individual to society. The suicidal person either feels he must sacrifice himself for society (the "altruistic" suicide), has poor social supports (the "egoistic" suicide), or feels cut off from society (the "anomic" suicide).

Later Freud, who understood melancholia or depression as hostility aimed at the self, tried to explain suicide as a lack of development of psychological tools to be able to tolerate the vicissitudes of life.

A variation on this idea, is the simplistic explanation of lack of self-esteem, which make individuals vulnerable to shallower interpersonal relationships, and harder to find meaning in life, which in turn lead to suicidal thoughts.

In cases of suicide in schizophrenia some have hypothesized that "the progressive loss of social drive, the diminished capacity to experience affect, and the indifference toward the future associated with deficit symptoms, often markedly debilitating, may preclude the painful self-awareness assumed to be associated with suicide.

In many cases, a lack of common descriptive terms, or a lack of clarity because of undefined or vague terms, leads to confusion, and perhaps some of the insights gained by some are lost in translation, or a lack of a common language or base. We will see how important the "preclusion of self-awareness" is to suicide once it is correctly defined and identified.

The majority of the latest models proposed that are specific to suicide and suicidal behavior are, essentially, variations of the diathesis-stress-hopelessness paradigm, well articulated by Schotte and Clum. Diathesis-stress models hold that people may have either genetically based or environmentally acquired trait-like, individual-level vulnerabilities to the onset of a disorder. These vulnerabilities constitute the diathesis part of the model and set the individual's threshold of susceptibility to stressful conditions. Given the genetic vulnerability, the stressors act as precipitants to the onset of more serious dysfunction. The diathesis-stress model proposes (Schotte & Clum, 1982, 1987) that deficits in interpersonal problem-solving skills predispose individuals under chronic stress to depression, hopelessness, and suicide ideation. However, it is now generally concluded that interpersonal problem-solving deficits may be a concomitant, rather than a cause, of depression, hopelessness, and suicide intent.[58]

A range of variables has been proposed as underlying diatheses or vulnerabilities triggered by stress, both acute and chronic. Among the most frequently cited diatheses are dysfunctional assumptions, cognitive distortions and cognitive rigidity.[59] Once

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[58] Schotte DE, Cools J, Payvar S, *Problem-Solving Deficits in Suicidal Patients: trait vulnerability or state phenomenon?* J Consult Clin Psychol. 1990. Oct; 58(5):562-4.

[59] M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior.*2001. Page 16.

individuals "acquire" vulnerabilities, the specific manifestation of a disorder that emerges will depend on or be triggered by identifiable, quite specific "risk" conditions that describe the person's recent life experiences. These late-stage conditions are often mistaken for specific causal factors.[60] Most all these factors are tangential, even though contributory, issues in the model I propose. This will be presented in detail shortly.

Some think that suicide is caused by unhappiness. If this is true, since unhappiness is impossible to prevent, kids should be told that the happiest people are not those with the fewest problems, but those with the best coping skills.

Suicide prevention depends on the accurate identification of who is most likely to kill himself or herself. Criticism of suicide intervention programs, Litman and associates suggest, may in part reflect the failure to differentiate between the acutely suicidal, who is perhaps being rescued, and the chronically suicidal person, who, because of a somewhat lower profile, eludes detection and becomes the program's failure.[61] But the situation is even worse. At present, there is no method to evaluate with any accuracy who can become suicidal, especially when they have never had suicidal thoughts. Failure to identify first-successful suicide-attempters reflects the complete lack of understanding of the true nature of suicide. This group, the ones that committed suicide with perhaps some previous warnings, but with no previous suicidal behavior or ideation, is still the most elusive, and the biggest challenge. I call them firsters, to separate them from others, because their first attempt is their last, because in the time elapsed between the first time they thought about suicide and their deaths, they almost never shared their intentions with anypne. Firsters surprise all around them and present the most baffling cases, as they seem to have shown little (seemingly) outward signs of mental instability.

The survivors of a first (or more) suicide attempt(s) are naturally labeled suicidal; however, they should be separated from those that have suicidal ideas but have never attempted suicide, whom are also labeled suicidal, as if they were identical. Thinking about suicide, and acting it out are different. The mechanism that leads to action is different than the one that contemplates possible actions.

The lack of a comprehensive scientific model of human behavior in general and suicide in particular makes prediction of suicidal behavior at best an art or an educated guess. There are a number of variables that are presently considered in assessing suicide risk, and some are just statistics. These can be divided into static and dynamic variables, meaning that they are enduring or simply descriptive or they are variable through time in either their intensity or frequency of occurrence, respectively.

Among the static variables considered are the following: age (escalation of risk above 45), sex (greater for males than females, males use more violent means of suicide), previous psychiatric diagnosis (bipolar, unipolar, schizophrenia and

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60 R.W. Maris, et.al., *Assessment and Prevention of Suicide.*

61 Armand M. Nicholi, Jr., M.D. *The Harvard Guide to Psychiatry.*

personality disorders are all associated with higher suicide rates), a previous history of suicide attempts or suicidal behavior in the family (there is a genetic tendency), and a history of sexual, physical or emotional abuse, family violence or punitive parenting (presumably part of the core problem, according to cognitive-behavioral theory, which perhaps creates a personality disturbance).

However, it is common knowledge that all these variables are also associated with normal behavior. For example, some people that were abused, are male, are fifty years old, have an uncle who committed suicide, and are bipolar do not ever attempt suicide.

Among the dynamic variables, predominantly acute, recurrent or chronic, varying over time in intensity or meaning to the patient and in the degree to which they are potentially debilitating, we find the following: stressors, particularly related to loss (loss of job, status, relationships, sense of identity, and physical or cognitive abilities), loss of health, psychiatric disorders focusing on mood disorders, substance abuse, schizophrenia or personality disorders; current markers of emotional dysphoria or symptom severity including anger, depression, hopelessness, helplessness, guilt, anxiety, panic, anhedonia, insomnia and diminished attention or concentration; cognitive rigidity and indications of poor problem solving or impaired coping; social isolation and limited support; impulse control problems such as substance abuse, aggressive behavior, risk taking or sexual acting out; and, active suicidal thinking and associated behaviors.[62]

Good practitioners consider different variables as part of an existing circle, and not as a step-by-step escalating process.[63] Severe, stressful life events, in addition to a mental disorder, can precipitate a suicide in vulnerable individuals. Three of the most recognized groups of life stressors that have been identified in completed suicides are conflict-separation-rejection, economic difficulties, and physical illness. These vary in impact according to age and gender. Associated with suicide in young people (15-35 years old) they report interpersonal conflict, separation or rejection and legal problems, especially incarceration. Particularly in men, later in life, job loss or financial strain are important stressor leading to suicide. Simplistically, in older people, medical problems are the predominant stressor.[64]

If friends were presented with an individual that fit several of the descriptions mentioned in the paragraphs above, I am certain they would all be worried that something very wrong is happening to that person—the intervention of a professional would hardly be necessary to reach that conclusion.

A study of 384 suicide attempters, employing scales to measure hopelessness, suicide intention, and depression, demonstrated that hopelessness is the key variable linking depression to suicide.[65]

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62 Ibid.

63 Thomas H. White, *How to Identify Suicidal People, A Systematic Approach to Risk Assessment.* 1999

64 Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

65 Armand M. Nicholi, Jr., M.D. *The Harvard Guide to Psychiatry.*

There is, in general, a simplistic approach concerning suicide. Some consider that some truly wish to end their lives, but that the majority of those who attempt and/or complete suicide want to end a devastatingly painful situation, more than they want to live, which is different than truly wanting to die. The logical corollary to this approach is that if they could find a way to solve or end their problems other than through suicide, they would do so. I think suicide is a more complex, and deeper situation than that.

One thing is certain, and it agrees with my own findings, regardless of approach or theoretical orientation, hopelessness plays a crucial role in depression and suicide. Hopelessness plays a role in ideation, attempts and suicidal completions in clinical samples.[66] Obviously, firsters (first-time completers), the ones that could provide the best information on suicide, are dead, and are not included in most studies. Therefore, most of what is known about them is based, at best on retrospective data from interviews with people who knew or interacted with the deceased.

Many researchers obtain information on suicide by studying the group that most resembles firsters-suicide attempters. However, many recognize that attempters may be very different from completers, especially firsters.

There are considerable differences of opinion by diverse practitioners. But the reality is that most models have very little if any significance or relevance in a clinical setting. Most clinicians, because of their lack of understanding, agree that suicidal patients are the most diagnostically complex and therapeutically challenging patients they see. This, they explain, is consistent with the complexity of suicidality itself, a problem inarguably the result of a complex web of factors with precise interrelationships varying from individual to individual.[67] But choosing a spouse, a career, or any life-affecting decision, is based on a complex web of interrelationships unique to each one of us. What else is new?

The lack of a scientific model results in considerable difficulty communicating coherently or consistently with patients. As hinted, many models lack specificity in terms of treatment according to therapeutic approaches. Some therapeutical methods demand treatment targets across many areas of human functioning, including cognitive, emotional, behavioral, biological, situational and even interpersonal. Because of this, some methods have focused on narrow approaches to target isolated although critical variables. All of this combines to produce a lack of comprehensive treatment-outcome monitoring and a corresponding lack of clarity for patients (as if they needed clarity to understand whether they feel better or not) as to what actually denotes treatment success or progress.

At present, in cognitive behavioral models, the most widely believed model of suicide defines a suicidal mode. This is done by establishing the idea of core beliefs that permeate the cognitive triad: 1) the self; 2) others; and 3) the future. These core beliefs, the model asserts, fall primarily into three domains: helplessness, unlovability

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66 M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior*.2001. Page 18.

67 Ibid.

and poor distress tolerance. As we will see later, these are symptoms that can lead to suicide, not the root of the problem.

To this they add another dimension where several systems are treated as separate entities, perhaps because of the limitations of the therapeutic approach. There is recognized a cognitive, an affective, a behavioral or motivational and a physiological system. Obviously all these are interrelated and do not exist in isolation from each other. In cognitive behavioral treatment the assumption is that there is an aberrant cognitive problem that needs to be corrected. Within the cognitive system, there would be a suicidal belief system made up of suicidal thoughts and ideation. The components of the cognitive triad are grouped into core belief categories: the self is seen as inadequate, worthless, incompetent, helpless, imperfect, unlovable and defective; others are seen as rejecting, abusing, abandoning and judgmental; and the future is seen as hopeless.

The affective system is dominated by dysphoria (mixed negative emotions): for example, sadness, anger, anxiety, guilt, depression, hurt, suspiciousness, fearfulness, tense, loneliness, embarrassment, humiliation and shame. The cognitivists attempt to separate these states from depression. As we will see later, this confluence of negative emotions could actually be a definition of depression. In simple, plain English, we should say the patient is depressed, not dysphoric. In the old days, we would say he or she is melancholic.

Following the principles of cognitive behavioral theory, the behavioral or motivational system is governed by death-related behaviors (intent to suicide): preparatory behaviors, planning, rehearsal behaviors and attempts. It is accepted that intent to die might have various motivations, for example the desire for revenge, punishment of a loved one, or the relief of tension, agitation, or pain without the desire to die, such as self-destructive behavior, like drug use or self-mutilatory behavior.

Individuals are seen as shifting in and out of the suicidal mode with great frequency and for variable periods. It is believed to be easier to trigger the suicidal mode for those evidencing chronic suicidality.

It is speculated that the duration of time for an active suicidal mode varies in accordance with the chronicity of the behavior. In other words, multiple attempters are likely to experience longer periods of activation of the suicide mode compared to single attempters. By definition, suicidal crises are acute and time limited in nature.

We must keep in mind that the suicidal mode is a conceptual model used to communicate with patients, incorporating empirical findings into the identified systems to translate them into a framework to guide treatment. And it is recognized that there exist a reciprocal interaction and interdependence of the various systems.

Aside from the cognitive, behavioral, affective and physiological systems, predisposing vulnerabilities and various triggers are also recognized. The predisposing vulnerabilities are also called facilitating modes that are associated with the suicidal mode. These include previous psychiatric diagnosis (Axis I and II), prior suicidal behavior, developmental trauma (abuse and neglect) and parental modeling. Depression or panic attacks might be lumped into facilitating modes. The triggers are seen as stressors and are generally

categorized as internal thoughts (images, feelings and physicals sensations) and external stressors (situations, circumstances, places or people).[68]

In many cases, too much time is spent on trying to find out what the predisposing vulnerabilities and triggers are. The therapist will attempt to determine what part of the patient's history facilitates suicide. What earlier experiences are significant? What self-defeating behaviors or patterns are exhibited? Did the parents lock you in a closet or burn you with cigarettes? Does the patient struggle to identify the suicidal precipitant? Are there identifiable themes to the thoughts (i.e., abandonment or rejection)? Is there a pattern for those with repeated attempts (i.e., after a particular type of loss)? It is important to recognize that in many cases there is no developmental trauma, and that the triggers might be only incidentally related to suicidal behavior because they coincidentally happened during the onset of depression, a panic attack or a psychotic episode. The depression, panic attack or psychotic episode is (are) truly at the root of suicidal behavior, not the other way around. I will show later why this is so.

When individuals that attempted suicide (and luckily failed), try to rationalize or verbalize what happened, it becomes easy for the therapist to put this into his or her own suicidal model. This provides a conceptual role for stressors during suicidal behavior. Therapists fail to realize that there always is a tendency to rationalize our behavior. Therefore, when clinicians report that, "Their cognitive systems are consumed with thoughts of death by suicide, with hopelessness pervading every component of the cognitive triad," we need to recognize that this is the therapist translating what the patient attempted to verbalize. When therapists report that the behavioral systems are characterized by an impulse to die, with related behaviors such as planning or rehearsing suicide and that the affective system is characterized by dysphoria, an aversive mixture of negative emotions such as anger, sadness, anxiety and guilt among others, they are failing to see the deep suffering and torment for which the patient has no words. The distress and pain is probably worse than any physical suffering. Any verbalizations or rationalizations of the feelings or thoughts experienced during a suicide attempt are shallow reflections of the true nature of such a mental state. I will attempt to show that this state is, perhaps, incomprehensible to us that haven't been there, at least in a subjective way. However, at least in an abstract way, understanding of this mental state is imperative to comprehend suicide.

Many models and various ideas have been put forth to explain suicide. Another concept is the suicide zone, which essentially represents the convergence of multiple factors (e.g., situational stress, acute dysphoria, psychiatric disturbance, impaired cognitive functioning, deficient problem solving and limited social support systems). The concept is that when an individual enters the suicide zone the risk of suicide raises significantly.[69]

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[68] M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior.*2001. Page 31

[69] David Rudd, Thomas Joiner & Hasan Rajab, *Treating Suicidal Behavior.*

Another model holds, in the case of young people, that exposure to environmental demands beyond those that are developmentally appropriate, or perhaps an insufficiency of necessary support from family and friends, leads to suicide through lack of coping skills and alcohol abuse. Following these ideas researchers have identified risk factors associated with increased risk of suicide. These have been divided into sociodemographic and clinical measures, such as males, older than sixty, single or widowed, living alone and with health or financial problems at are a higher risk, or previous suicide attempts and mood disorders, especially unipolar and bipolar depression. It has been shown that methods that predict suicide based on recognized risk factors will not only lead to a huge overidentification of those at risk, but also to a high number of missed or undetected cases.

Others have separated risk factors into distal and proximal. Distal risk factors may be thought of as the foundation (?) for attempted and completed suicides. Distal factor might not occur immediately before the suicide event. Proximal risk factors, on the other hand, are closely associated with the suicide event, and can be thought of as triggers for suicidal behavior. It is recognized that many people may have risk factors and are not suicidal. These approaches propose that the co-occurrence of distal and proximal factors lead to suicide.[70]

The best therapeutic approach today views treatment in three phases: crisis, skill building and personality development, each with a specific treatment agenda. However, in cognitive behavioral therapy, permanent cognitive restructuring cannot occur without activation of the suicidal mode and each component system. That is affective experience and mobilization of the mode are essential to treatment progress incorporating meaningful skill development and lasting personality change. Enduring change is structural change, and structural change depends on affective and behavioral impulse, both signifying sincere activation of the suicide mode. In accordance with this model, suicidal crises are a necessary and expected part of treatment. That is, if the suicidal mode is to be permanently altered both in content and threshold for activation, as well as in subsequent restriction of the range of potential triggers, the patient needs to undergo and survive a suicide crisis. This approach, implies to some degree, that the therapist be present at the time of crisis, in some cases, a highly unlikely possibility. It is obvious that this approach hopes first to diffuse suicidality and then the bulk of the treatment will address the facilitating modes (i.e., the enduring personality psychopathology) and the development and refinement of competing and more adaptive modes for living.

This is another example of a naive approach where a traumatic event is believed to be at the root of suicidal behavior.

Another view is that suicide is a process by which maladaptive responses transpiring at any or all system levels of biological, psychological and sociocultural vulnerabilities,

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[70] Douglas Jacobs, *The Harvard Medical School Guide to Assessment and Intervention of Suicide.*

interacting with particular demands of environmental life conditions (i.e., stress) over time, result in varying degrees of suicidal intention (i.e., suicidal thoughts, plans, attempts or completion), which ultimately might lead to suicide. Accordingly, certain individuals, as a result of intrinsic vulnerabilities are ill equipped to handle stressful events, and over time, reach a psychological breaking point. At this point, vulnerable individuals are unable to see, generate, and/or effect coping responses and therefore see suicide as perhaps the only viable option. From this point of view, there are individual variations as far as vulnerabilities or stressful life conditions that lead to suicide, except for the common psychological state of hopelessness.[71]

In efforts to understand suicide, researchers studying many characteristics and variables common to individuals who have attempted and completed suicide, have called these risk factors, which serve to increase the potential for suicide, either directly or indirectly. Some risk factors are potentiating (that is, they are direct triggers for the suicide act); others are predisposing (that is, long range, indirect influences on the suicide act, such as schizophrenia).[72]

What they call the facilitating modes, as I mentioned before, are a part of other mental disorders (i.e., anxiety and panic attacks, manic-depression, depression, schizophrenia and personality disorders), which could be caused by neural chemical imbalances in many cases, and not some traumatic event. It is true that huge stressors could precipitate some of these conditions when some genetic vulnerability exists. Attacking these mental disorders in the most aggressive manner possible is the prelude to attacking suicide, as these are unquestionably, the tortuous roads that lead to suicide. Aggressive therapy in the face of depression, schizophrenia or personality disorders is the preemptive strike at suicide. Waiting for a suicide attempt to occur or be seriously contemplated, is not the best way to treat anyone. The road to suicide is a terrible and painful one, much more so than death by suicide. Death, after all, is only painful for the ones that are left grieving.

Edwin Shneidmann has proposed a conceptual model of suicide noting the convergence of pain, perturbation and stress. Suicide and suicidal behavior are viewed as psychological phenomena, a function of individual pain and tolerance; both of which are determined, influenced, and modified by a multitude of factors. He noted that the clinician can best understand, assess, manage, and treat suicidal behavior by attending specifically to these two variables, the patient's experienced (and expressed) pain and demonstrated pain tolerance. Psychological pain, in his view is the result of frustrated psychological needs. He distinguished between day-to-day needs and vital needs—those that when frustrated produce intolerable psychological pain and, if unchecked and under the right circumstances, can lead to suicidal behavior and suicide. He focuses more on the affective needs, emotional pain and pain tolerance than do cognitive theories, but his ideas can be placed within a cognitive therapy.

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71 R.W. Maris, *Assesment and Prediction of Suicide.*

72 Thomas White, *How to Identify Suicidal People.*

His proposed model in a three dimensional Cartesian coordinate system would have pain in the X axis, perturbation in the Y axis and stress on the Z axis. Each variable would be on a scale of 1 to 5. Pain with one for little pain, 5 for intolerable pain. Perturbation, a general term meaning the state of being upset, would have a 1 for low, and a 5 for high. Stress, likewise would have 1 for low and 5 for high. Patients would be rated on these three scales. According to this model, not every individual who is a 5,5,5 commits suicide—he or she might commit homicide, or go crazy, or become amnesic, or destroy a career—but in this conceptual model only patients in the 5,5,5 (maximum pain, maximum perturbation and maximum stress) commit suicide. The implications for therapy and life-saving redress would seem obvious: Reduce any of these three dimensions from a 5 to a 4 (or less); preferably reduce all three. Individuals could then lead long unhappy lives as 4's and 3's, instead of killing themselves.

Part of the stresses—the losses, the humiliations or physical ills—is how the person perceives or misperceives them. From this point of view, character strength [almost impossible to define] becomes an important variable, and at present is beyond us.[73] Here is another case of using concepts that are part of our sociocultural milieu, but for which we have no adequate scientific grasp of. Along these lines, some argue that in the face of low-esteem one cannot soothe oneself sufficiently under stress, and crises of contempt, panic or rage may be expected. These may be intense enough, they argue, to invite suicide. Terms such as narcissistic reserves, defined as applying inner sources, psychic structures, functions, relationships and exterior resources used to preserve the integrity of the self, is what the patient brings to bear to avoid suicide. In this view, the immediate risk of suicide is the degree of narcissistic shock from which the patient suffers.[74]

Thomas Joiner, whose father committed suicide when he was a psychology student, went on to propose a model of suicide building on Aaron Beck's and Shneidman's theories. Despite the fact that he admits that 95 percent of suicides are related to mental illness, he proposes that the main causes for suicide are perceived burdensomeness and failed belongingness. To this he adds an element of hopelessness in relation to these two components coupled with acquired capability for self-harm. He also considers impulsivity as a possible part of the suicidal model, but he gives it a different twist. Of all personality dimensions, impulsivity has the most clearly documented association with suicidal behavior. He believes this has provided the misleading suggestion that the act of suicide itself is an impulsive decision. He doesn't think so. Rather, there is a real and important association between impulsivity and suicidality, and it exists because impulsivity leads people to habituate to pain and provocation. Thus, they acquire the ability to enact lethal self-injury, and are thereby at increased risk for suicide, if the

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73 R.W. Maris, et.al., *Assessment and Prediction of Suicide.*

74 Ibid.

desire for death is in place. Impulsivity could also relate to suicidality through increasing the desire for death as well.

An emphasis on hopelessness places negative thoughts and styles of thinking front and center in explaining risk for suicide. From a similar perspective, Beck has also argued that previous suicidal experience sensitizes suicide-related thoughts and behaviors such that they later become more accessible and active. The more accessible and active the thoughts and behaviors become, the more easily they are triggered, and the more severe are the subsequent suicidal episodes. In this sense, Beck's views share Joiner's in that both perspectives propose psychological mechanisms underlying an escalating course of suicidal behavior over time. In Beck's view there is a cognitive sensitization—with repetition, suicide-related thoughts and behaviors become favored. In Joiner's view the mechanism is one of habituation, or getting used to the fear and pain involved in self-injury, which leads to an acquired ability for serious suicidality, and combined with burdensomeness and disconnection lead to high risk of suicide.

Joiner proposes that the acquired capability to engage in serious self-injury is but one precursor to attempted suicide or death by suicide. Many people, through an array of provocative experiences, become fearless, pain-tolerant, and knowledgeable about dangerous behaviors, and yet have no desire to hurt themselves. Those who have the desire, coupled with the ability are viewed as high risk for suicide. When the need to belong or connect with others, and the need to feel effective with or influence others is frustrated, suicide becomes attractive but not accessible without the ability for self harm.

Joiner states that habituation to pain not only includes previous suicidal behavior but includes any activity that allows people to get used to pain and reduces fear of injury in general and self-injury in particular. In relation to habituation to pain, Joiner includes such topics as tattooing, self-injected drug abuse, even strenuous or violent sports.

Joiner's model proposes that the acquired ability to enact lethal self-injury is a necessary precursor to serious suicidality, especially to completed suicide. This acquired ability involves fearlessness about confronting pain, injury, and indeed death; the reinforcing qualities of repeated self-injury may also be involved. Serious suicidal behavior requires both the desire for suicide and the acquired ability to carry it through.

Joiner bolsters his claims using many studies. One of them compared 134 current suicide ideators, 128 people who had recently made their first suicide attempt, and 68 people who had recently attempted suicide for at least the second time. They compared the three groups on an array of symptom and personality indices. As compared to those with suicidal ideas and those with one attempt, multiple attempters experienced more intense suicidal symptoms, such as desire to die, plans to attempt, resolve to die, intensity and duration of suicidal ideation, and so forth. This was the case on both self-report and clinician-rated scales of suicidality. There were also differences on some personality variables, such as hostility. Even though all participants were in a suicidal crisis, multiple attempters stood out from others in terms of the severity

of their suicidality as well as some features of their personality, (i.e., they had more past practice at suicide, and thus they had moved further along the trajectory toward serious suicidal behavior).

His view is that the need to belong is so powerful that, when satisfied, it can prevent suicide even when perceived burdensomeness and the acquired ability to enact lethal self-injury are in place. By the same token, when the need is thwarted, risk for suicide is increased.

Past suicidal behavior confers risk for later suicidality, including death by suicide, and does take into account other variables like mood disorders. One study compared those who died by suicide to living controls. Suicides and controls were matched for presence and severity of mental disorders (also for gender and age), so any differences between the groups were not likely to be attributable to one group having more psychopathology than the other group. One of the main variables that distinguished those who died by suicide from living controls was a significant past history of deliberate self-harm. Similarly, past attempts comprised a significant predictor of later death by suicide, even taking into account several other *powerful* (Italics added) predictors, such as presence of mood disorders. In these studies multiple attempt status conferred risk to death by suicide, even beyond the effects of other powerful predictors, a finding quite consistent with the current conceptualization that people may "work up" to death by suicide through repeated episodes of deliberate self-harm (as well as through other means).

According to Joiner, multiple suicide attempts are vigorously related to past and future suicidality because people that habituate to self-injury gain the ability to enact increasingly severe suicidal behaviors. He goes as far as insinuate that people with Body Dysmorphic Disorder (a condition characterized by obsessions with one's imagined ugliness) have both high rates of surgery and high rates of suicidality, as if the high rate of surgery predisposes one to suicide. In this vein, people that have witnessed, or engaged in more violence than others are more prone to suicide. He also mentions prison inmates at increased risk for suicide, particularly ones incarcerated for manslaughter or murder. He cites studies where suicidal people can tolerate more pain, extreme temperatures or higher pressures applied to the skin better than other patients. Accordingly, decreased pain sensitivity leads the way to serious suicidal behavior.

Physicians have a relatively higher suicide risk than many other professions. This is true for male and female doctors. Women doctors have quite elevated suicide rates compared to other women, on the order of 3 to 5 times higher. Joiner explains this because they frequently observe the consequences of pain, violence, and injury, as well as gaining specialized knowledge about lethal agents, dosing, and methods of death.[75] Joiner does not even address stress as a major factor, other than to mention that there might be gender-specific pressures at work, burnout and some job stress.

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[75] Thomas Joiner, *Why people Die By Suicide.* 2005.

Joiner seems to think that some symptoms of depression such as poor eye contact, isolation, a lack of response to other's facial expressions or gestures are really something different from failed belongingness and not symptoms of a depressed state. He uses the fact that many who have committed suicide have experienced isolation and withdrawal (which is typical of depression) before their deaths to bolster his claim that a lack of belongingness is what leads to suicide.

Again, as we will see later, I find that the symptoms of a much deeper psychological state, such as burdensomeness, and a sense of belonging (both somewhat philosophical concepts) are mentioned as causes (instead of symptoms) of suicide. The tremendous emotional pain (what Shneidman calls psychache) caused by mood disorders is not even considered as a factor that increases tolerance to physical discomforts. However, Joiner does acknowledge that there is a lack of longitudinal studies showing that pain tolerance is related to later suicidality.

Not surprisingly, Joiner suggests a broadening of the focus in suicide risk assessment from multiple attempters to anyone who has, through various means, acquired the capability for lethal self-injury.

Learning emotional skills and control, or improving personality perception can decrease or even eliminate a suicidal condition. But it must be understood that if this is so, it is only because peripheral issues, ones that inevitably are affected when suicide is possible, can influence the whole brain. It is not because these are necessarily causing suicidal behavior.[76]

It is important to keep in mind that most of these studies do not include first-time-successful suicide attempts (firsters). We need to understand clearly that the cognitive behavioral approach treats symptoms, not the root causes. However, like the flu, alleviating symptoms is, sometimes, the only thing that can be done while we wait for the sickness to pass. I cannot emphasize enough, because of practitioners' limited knowledge, that in suicidal therapy, at best, only symptoms are being alleviated. The good news is that some times the symptoms disappear to never return, with or without therapy. However, when we are faced with suicide, until we have better therapies, it is imperative we remain vigilant of the symptoms.

The only studies on firsters who gave little or no warnings to clinicians are based on a psychological autopsy, which attempts to reconstruct an individual's life after the fact, particularly the person's lifestyle and those thoughts, feelings, and behaviors manifested during their last weeks in order to attempt to understand the circumstances contributing to a suicide. Still, it is accepted that no one yet knows the best way to plumb any specific case given the limitations on current knowledge, and that no one can deliver an authoritative conclusion about why a given suicide occurred.[77]

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[76] Thomas White, *How to Identify Suicidal People.*

[77] R.W. Maris, Alan Berman, John Maltsberger and Robert Yufit, *Assessment and Prediction of Suicide.*

The treatment of suicidal patients has been characterized by relatively high withdrawal rates, with those abruptly stopping treatment often continuing to experience marked symptomatology and continued high risk.[78] This probably reflects the limitations and the lack of success of most treatments (or practitioners) to alleviate the pain and suffering of this condition. It would be interesting to determine how many of those who stopped treatment were survivors of a previous attempt or went on to became firsters. However, I suspect that most firsters sought out treatment for mood disorders and not for suicide, and were probably considered non-suicidal.

Suicide therapy can only be administered when the patient becomes stable enough to engage emotionally with others.

It is important to follow a few guidelines when dealing with a suicidal person. As summarized by Schneidman,[79] and most other practitioners agree:

1.) The therapist must defend the life of the patient in a calm and gentle manner, but firmly make clear that his role is direct and active, including any form of intervention necessary to prevent suicidal behavior. However, the therapist's reach and scope of what he can do to control another person's life is very limited when treating an outside patient.
2.) In suicidal cases, the therapeutical relation requires a violation of the usual confidences, since disclosure of self-inflicted-death plans to others might prevent the outcome. It is imperative to bring in family, friends and loved ones.
3.) Extreme measures of monitoring should be implemented, even when the risk is considered low.
4.) The therapist must be able to listen carefully and through their emotional involvement, be able to tolerate the feelings of hopelessness, despair, desperation, anguish, rage, loneliness, emptiness, and meaninglessness articulated by the suicidal patient. The patient needs to feel that the therapist understands.
5.) Every effort to reduce social isolation and withdrawal is imperative.

Various suicide models approach this in different ways, but whatever the approach, when it works, it is because indirectly it forces the patient to interact with others. As we will see later, it is extremely important to be emotionally engaged with the suicidal patient. And this goes for all involved: family, friends and loved ones.

6.) All other coexisting psychiatric disorders must be treated. Treatment should address these specific differences. With a suicidal schizophrenic, reality testing,

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78 Thomas White, *How to Identify Suicidal People.* Page 73.

79 E.S. Shneidman. *Suicide.* In *Comprehensive textbook of Psychiatry.* 1975.

increased medication and even electroconvulsive therapy may be essential to prevent death. Similar considerations apply when dealing with borderline personality disorders, depression, anxiety or other conditions.

7.) When treating a suicidal patient, the therapist himself must seek support. It is so emotionally exhausting to engage a suicidal person that the therapist's own reactions as well as the patient's feelings can make treatment seem like an ordeal. *For this reason the therapist should never engage in treating more than one suicidal person at a time.*

During treatment of suicidal patients emotional transference and countertransference problems arise easily. Dysthymic patients form stable, positive dependency transferences and, therefore, persist in treatment. Countertransference problems arise when a therapist becomes weary with a treatment's lack of success. In the case of suicidal schizophrenic patients, the transference problem is that they become totally dependent on and then paranoid toward their therapist. The countertransference problem is that the therapist becomes terrified by the psychotic projections. Clinicians treating suicidal borderline personality patients find themselves feeling responsible for the lives of the patients as suicidal threats and attempts escalate. The solution might be a consultation with another clinician, a case review, and sometimes transfer of a patient is recommended. Occasionally a brief hospitalization is recommended during a transference-countertransference crisis.

Severely suicidal patients should not be treated, if possible, alone. The extreme stress and anxiety inherent in such treatments can interfere with the most finely honed therapeutic skills. Therapists should receive therapy from others.

Hospitalization is much less effective as a form of suicide prevention for persons who suffer chronic suicidal psychiatric disorders such as dysthymia, with recurrent major depression, schizophrenia with suicidal ideation, alcoholism or substance abuse or personality disorder.[80]

Psychiatric Emergency

When a person suffers a sudden mental disturbance a psychiatric emergency should be declared. A psychiatric emergency is a life-threatening event, just like a cut artery, a smashed limb or a bullet wound.

The problem, of course, becomes one of knowing or being able to identify a potential suicidal situation. If it is you suffering through this, the problem is whether you remain rational enough to know this and ask for help.[81]

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[80] R.W. Maris, et., al., *Assessment and Prediction of Suicide.*

[81] Author's note: Susan Rose Blauner, *How I stayed Alive When My Brain Was Trying to Kill Me,* has all sorts of advice on how to help one avoid suicide.

Edwin Schneidman has defined suicide as "the conscious act of self-induced annihilation, best understood as a multidimensional malaise in a needful individual who defines an issue for which the act is perceived as the best solution."[82] This implies that suicide is not a random and pointless act. In that I agree; but it is not a rational act either, at least in most individuals. For some individuals, suicide is not a preplanned action; it is the first and, because they act on it, their last suicidal impulse, with no time to ask for help.

Schneidman tried to distill theory and research to one simple statement, "Suicide is caused by psychache."

If you ever find yourself suicidal and if (and this is a huge if) you can pull back from the brink, as William Styron recounts so lucidly in his book, *Darkness Visible,* when nothing much else can be done, seek emergency assistance immediately. Or if you interrupt someone in the course of committing suicide, emergency support should be sought, making sure the suicidal person is accompanied at all times.

However, it is important to keep certain things in mind. Hospitals are not always safe. There are some completed suicides every year in psychiatric wards, and several times more in the immediate postdischarge period. Psychiatric hospitals can be frightening, especially in the beginning. They can also be stigmatizing or seen as a blow to self-esteem. Hospitalizations might remove patients from their main psychological supports, such as family or work. Sometimes, a patient's life-problems need to be solved, and being in the hospital just makes them worse. Hospitalization can be very expensive, and surprisingly, there are few studies showing evidence that hospitalization is a necessary or even a helpful therapeutic intervention for acutely suicidal patients.

On the other hand, for some suicidal people who are delusional, confused, or extremely agitated, the benefits are obvious. For some suicide attempters, commitment delivers a message that they are being taken seriously, and this in itself may be therapeutically beneficial.

Once in the emergency room of a hospital, one will probably end up at the hands of a competent physician. *Care should be given to specifically request a psychiatric consultation.* Keep in mind that in emergency rooms psychiatric records are generally not available to check any previous history. If another physical condition exists, like a broken bone for example, or cut wrists, the standard approach in the emergency room is to not deal with psychiatric aspects of somatic illness. In general, most staff at the emergency room is not equipped to deal with an added psychiatric complication.

With mental disorders there is a bigger risk that something can go wrong because of the uncertainty of the physician's own knowledge of the subject. The most probable action is a quick assessment of your degree of suicidal risk to determine the appropriate level of response. ER staff will ask you your age, sex, marital status, employment, educational and social background and so on. They will ask you to verbalize your

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[82] Harold I Kaplan & Benjamin J. Sadock, *Synopsis of Psychiatry.*

suicidal intentions and they will ask you about life crises and previous mental disorders. They will try to resolve the immediate crisis. They will probably sedate you and try to stabilize you to the point that you can't act on your impulses, even if you wanted to. They might put you under surveillance and in special rooms to prevent further possible acting out of suicidal tendencies.

A psychiatrist will be called. He or she will try to do a complete psychiatric history; try to talk to friends and relatives to have a better assessment. You will probably be moved to a psychiatric ward. The initial efforts should be focused on reducing suicidality. Nurses will try to reduce psychological pain by modifying a stressful environment; if possible by enlisting the help of a spouse or friends. The psychiatrist will attempt to build a realistic support by recognizing that the patient may have a legitimate complaint and offer alternatives to suicide.

Once a diagnosis is reached, medications will be given as needed, such as sedatives, antidepressants, antipsychotics or mood stabilizers. You will be interned in the psychiatric ward until considered stable enough. Individual, group and family therapies will be provided; you will receive the hospital's social support and sense of security. If they discover other underlying problems like alcoholism or some organic sickness, they will try to address those, too. They will repeatedly search your belongings and person to eliminate exposure to dangerous or potential injurious self-inflicting objects. The treating team must decide how much to restrain you and how often should you be checked or be under continuous observation. It is important to instill a sense of hopefulness regarding the immediate future and the treatment process.

Virtually the effect of all antidepressants take, typically, at least a week and sometimes up to three weeks. However, the failure of objective improvement in activity, sleep, appetite, mood or social interest within 1-2 weeks is an unfavorable prognostic sign, suggesting that the final result will be unsatisfactory. The word "objective" is important because the patient is generally the last to report improvement. Changing doses or medications should be done with great care.

I add a word of caution, and I quote from *The Harvard Guide of Psychiatry, "*Owing to the potentially severe toxicity and limited margin of safety of TCAs (tricyclics), MAO inhibitors, and bupropion, dispensing more than a week's supply to depressed and possibly suicidal patients is unwise. The risk of suicide rises with *inadequate* (italics in original) antidepressant treatment and may increase with initial improvement, since activity usually increases before mood elevation.[83] Prudence suggests that increases in agitation, restlessness, disorientation or insomnia should be taken very seriously to help avoid aggressive behavior including self-injury or suicide.

In many instances, antidepressants can have adverse side effects, and these should be addressed as quickly as possible. To name a few: SRIs carry a high risk of inducing nausea and vomiting or sexual dysfunction; high doses of bupropion, clomipramine,

[83] Armand M. Nicholi, Jr., M.D. *The Harvard Guide to Psychiatry*

and maprotiline can induce epileptic seizures; trazodone can induce confusion; bupropion can induce agitation and insomnia and is best not given at night.[84]

In the case of manic-depressives, antidepressants can induce a manic episode.

In extreme cases of severely depressed patients, especially when there has been no response to medication, doctors might determine that Electroconvulsive therapy is necessary. This might require several treatments, and can be quite disorienting, affecting memory, sometimes permanently.

Once the patient is considered stable enough, a medical and therapeutical program should be initiated and followed aggressively. The hospital can be a sanctuary, where the familiar surroundings of everyday are removed and an oddly gratifying sense of stability and isolation can be found. Even the continuous piercing of sirens and the sound of commotion can be stimulating, as a constant reminder that one is in a cathedral of sickness trying to join the healthy. All efforts should be directed at reducing symptomatology.

After noticeable improvement of depression is achieved with antidepressants, it is usual to continue the treatment with a well-tolerated dose similar to that found to be effective in the acute phase of treatment for several months and perhaps up to a year.

Sometimes, however, the best efforts to recognize suicidal tendencies in one self or others, fail.

Patients recovering from a suicidal depression are at particular risk. As the depression lifts, patients become more energized before the mood improves significantly, and are thus able to put their suicidal plans into action.

In emergency settings, hardly ever is an assessment of murder-suicide carried out. Most patients in a psychiatric emergency room are assessed for suicidal and homicidal ideation separately. Few clinicians link the two behaviors and realize there can be overlap. It should be recommended that patients who present a recent suicide attempt, have a plan for suicide, or suicidal ideation should be evaluated for violent or homicidal behavior. Similarly, those who've had recent violent behavior or voice homicidal ideation should be assessed for suicidal behavior. Although assessment of violence and suicidality is done separately, the clinician should consider the simultaneous occurrence of both in a single individual. Perhaps the most important consideration in the assessment of murder-suicide is the consideration of murder-suicide as a possibility.

It is common with patients with borderline personality disorder to present discrete episodes of acute suicidal in contrast with depressed patients where the suicidality appears in conjunction with the depression. As a consequence, unsolicited hospitalization, aggressive pharmacology or electroconvulsive therapy, which are effective with depressed patients, may not be effective, and in some cases may be counterproductive. In many ways, the effective management of suicidality with borderline patients is contained in the thoughtful day-to-day attention to roles,

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[84] Ibid.

boundaries, and relationships that promote psychological growth. Borderline patients frequently engage in "manipulative" suicidal behavior.[85]

A patient may commit suicide even when in the hospital. According to a study, 1% of suicides occurred in general medical-surgical or psychiatric hospitals; however, the annual suicide rate in psychiatric hospitals is only 3 per 100,000.[86]

And last, as a note of caution: between 0.2-0.5% of all patients discharged from psychiatric hospitals commit suicide within a year, with a decreasing but noticeable suicide rate thereafter.[87]

Suicide Risk Assessment

The average psychologist who works directly with patients has a more than 1 in 5 chance of suffering the blow of patient suicide. For psychiatrists, there is a 50 percent chance that a patient will commit suicide, and even for psychiatric residents, patient suicide has been described as a "common, if not universal part" of their residency.[88] This should come as no surprise given the statistics of mental illness and suicide.

In this section I will try to present the most common or accepted ways used in suicide risk assessment.

The broad social influences known by epidemiologists to favor suicide are, for the most part, so remote from the individual case that they are clinically disregarded. It is beyond the skill of any clinician to discern who is touched by these larger forces, which are in some way at work on everyone. Many things are known, such as that the suicide rate of each age group varies by age and by gender. Suicide rates decrease in wartime and increase during economic depressions. Natives of some countries are at greater risk of suicide than those of others; Icelanders are at greater risk than Italians. Similarly, those living in Nevada are at greater risk than those living in Massachusetts. Considerations such as these rarely enter into the calculations of clinicians.[89]

Prediction of any rare event is a very difficult task. The prediction of suicide or even suicidal behaviors or suicidal ideation, is complicated by many factors and by a lack of complete knowledge and understanding of these factors.

Suicide prediction is mostly a case of predicting correctly or not. At present, suicides are mispredicted more often than not. Given the state of the art, suicide prediction is not very precise or useful. Efforts to improve the sensitivity of available risk-prediction measures result only in more false positives (individuals identified as potential cases

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85 Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

86 Armand M. Nicholi, Jr., M.D. *The Harvard Guide to Psychiatry.*

87 R. W. Maris, et. al., *Assessment and Prediction of Suicide.*

88 Thomas White, *How to Identify Suicidal People.*

89 Ronald W. Maris, Alan L. Berman, John T. Maltsberger and Robert I. Yufit, Editors, *Assessment and Prediction of Suicide.* 1992.

who do not in the near future attempt suicide). Many practitioners prefer to speak of assessment, in this context meaning to set an estimated value on something, and imply less prediction than that of suicide prediction. Shneidman defines suicide as a conscious act of self-induced annihilation, best understood as a multidimensional malaise in a needful individual who defines an issue for which the suicide is perceived as the best solution.[90]

In many cases, the psychiatric staff will attempt to evaluate degree of suicide risk by looking at the method used, the degree of impairment or consciousness when rescued, extent of injury, time required in the hospital to reverse the effects, the intensity of the treatment required, as well as other details of the rescue circumstances, such as remoteness from help, probability of discovery, accessibility to rescue and delay between attempt and rescue.[91] Typically, we find ourselves, again, studying suicide survivors, or at least patients with expressed suicidal ideations. It is a common mistake to equate completed suicides with nonfatal suicide attempts. What about firsters?

While no one can predict with absolute certainty, or even probabilistically, whether a person will commit suicide—an effort should be made to determine where a person lies on the continuum of risk at a given time. Despite this need, there have been few serious efforts by professional organizations or institutions of higher education to develop techniques that would improve our ability to identify and help those who are at risk of suicide. Arguments have been made that suicide prediction scales, psychological tests, statistics, suicide checklists and other quantitative procedures only take us so far. Such procedures are usually not flexible, individualized, specific, or thorough enough. The argument goes that the skilled clinician can read between the lines, and they "know."

Some practitioners are more interested in limiting their legal exposure by exploring if a patient will use the support that is offered, whether it is institutional support, time-limited, person-limited and whether it is available when needed. This presupposes that a suicidal person will actually seek help and utilize any existing resources and that the patient knows the requirements or availability of specific services, when there is actually very little evidence that suicidal people will call for help. In this same vein, many practitioners are concerned about their legal responsibilities, especially when they treat a potential suicidal patient. In regards to this aspect, they are careful to observe "standard" reporting and risk assessment practices, even though what is standard is open to debate.

Courts expect practitioners to perform clinical services in a reasonable and prudent manner in accordance with accepted principles and the customary practices of their profession. It is here, with regards to accepted principles and customary practices, that there is huge room for improvement.

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90 Ibid.

91 Armand M. Nicholi, Jr., *The Harvard Guide to Psychiatry.*

The legal problems could be greater when dealing with high-functioning people. Typically, a suicidal episode would be characterized as a transient aberration, perhaps under the influence of alcohol or some other substance. They usually would be able to convince a judge that they should not be detained against their wills. The Harvard Medical School Guide to Suicide Assessment and Intervention offers explicit instructions on the measures that should be followed when discharging persons to their own care after serious suicide attempts and to carefully document it: keep a chart to show that serious inquiry was made into past history, stressors, mental status, suicidal ideas, impulses, plans and intentions; a family member or spouse should be interviewed; bring in a consultant to assess suicide risk; arrange outpatient follow-up, and inform the family of the limitations, especially if it is refused; if all has failed ask for an extended legal hold and let the court procedure mandate the discharge or continued evaluation and treatment. It is obvious that a legal court has no knowledge of what makes (or doesn't make) a person suicidal, but at least the legal issues are resolved: the doctor is not responsible, he or she did everything they could according to standard procedures.

Aaron Beck identified hopelessness as a key cognitive predictor of suicide as far back as 1973. He developed and validated a series of scales to help measure suicide risk such as the Beck Hopelessness Scale, the Beck Suicide Intent Scale and the Beck Scale for Suicidal Ideation.[92] However, much still remains to be done to understand suicide and design clinical interventions to prevent suicide.

There is a tendency to simplify suicide. This results in the naive notion that all the practitioner needs to do is to determine whether the patient wants to die, and if so, why.

Along these simplified views, practitioners try to determine the probability that death will occur if a suicide attempt is made. This is referred to as lethality. Lethality increases when a person has a good *plan*, uses a *deadly method* or has the skill to carry out the act and the *knowledge* to determine the effectiveness of the chosen method. It is generally understood that rarely will these three components come together to cause self-inflicted death without intent to die. Logic seems to indicate that people with a serious intent to die, will eventually find a way. If he or she fails on their first attempt, chances are good that they will succeed in their second attempt. Of people who have attempted suicide once, and actually end up committing suicide, 84 percent are successful on their second attempt.

According to some lethality can be evaluated by an empirical formula that involves the interaction of various components:

$$\text{LETHALITY} = \text{Intent to die}(\text{Plan} + \text{Means} + \text{Knowledge})$$

In most cases, for a completed suicide to occur, the four components must come together in a particular way and in a particular moment in time. Therefore, determining

[92] Robert Leahy, Editor. *Contemporary Cognitive Therapy*. 2004.

an individual's current suicide risk potential requires an examination of the current relative strength of each of the elements and their current relationship to one another. Intent is represented as a multiplicative and is the factor that most affects the likelihood that death occurs. Attempters who eventually completed suicide increased the lethality with each subsequent attempt.[93]

In my experience, determining intent to die is a simplistic approach to suicide. Many firsters present no intent to die or even suicidal ideation, until (perhaps) a few moments before they kill themselves. However, it is important to understand that many practitioners do have a naïve approach when it comes to suicide. They might explore with a patient reasons for living and determine that these outweigh the desire to die, as if truly suicidal people could rationalize such ideas.

At present, suicide risk is based on some naïve ideas about suicide. In the next paragraphs I present a brief outline of how various levels of risk for suicide are determined according to established practice.

In general, patients with no risk to minimal risk show no apparent risk factors and there is no reason to assume they are going to kill themselves. People who are at minimal risk may have vague suicidal ideations. Yet, occasionally, people in this group sometimes commit suicide.

People with a low risk potential for suicide, might have engaged in self-destructive behavior but without suicidal intent and minimal lethality. This group would generally include self-mutilators or people who engage in self-harm for manipulative reasons.

People with a moderate risk include people with self-destructive behavior without suicidal intent, but with a moderate to high level of lethality, or people with previous attempts with a low level of lethality.

A high risk of suicide is assigned to individuals engaged in self-destructive behavior with serious suicidal intent and moderate to high lethality or have engaged in less lethal behavior but have ongoing suicidal intent.

Many therapists often overlook, or chose not to consult and get the opinions of their colleagues. As a result, they frequently make very difficult, extremely complex and potentially life-threatening decisions in a vacuum.[94]

Generally, suicidal risk is evaluated by asking about behaviors in preparation for suicide (planning, financial and insurance arrangements or letter writing), or related self-destructive behaviors and impulsivity (substance abuse, risk taking, sexual acting out). This is quite naïve, as these behaviors might be symptomatic of other mental disorders, which could precede suicidal behavior, but not always; in most cases not.

Clinicians look for direct suicidal markers that include suicidal ideation (frequency, intensity, duration, and specificity) and suicidal behaviors (discovery avoidance

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93 Thomas White, *How to Identify Suicidal People.*

94 Ibid.

behavior, attempts or preparation and choice of method). They consider indirect markers like hopelessness, depression, anxiety, impulsivity and anger along with individual characteristics (cognitive rigidity and problem-solving ability). In spite of all this, risk assessment is still an art, not a science.[95]

H.E.L.P.E.R. is an acronym that stands for the six steps that should be (according to Thomas White) part of every suicide assessment: (H) historical factors; (E) environmental factors; (L) lethality of the suicidal thinking and behavior; (P) psychological factors; (E) evaluation of suicide risk potential, and; (R) reporting your findings. Here we see another simplification of the conditions that lead to suicide, even though there is special emphasis that each component of the H.E.L.P.E.R. assessment system is important to the determination of suicide risk potential, and that each step cannot be used by itself.[96]

Care should be taken analyzing personal historical events such as erratic and failed employment, legal problems, poor social interaction or individual isolation, as they can be simplifications to explain loneliness, hopelessness or suicidal ideations. In most cases involving potential suicide loneliness and hopelessness are produced by a mood disorder.

Another approach is to look at suicide predictors such as mental illness, isolation, genetic factors, white-older male, stress, anger, aggression, irritability or any and all combinations of the above.

Alcoholism, in one study, found that an on average 18% of alcoholics eventually commit suicide. Another study showed that 72% of completed suicides were either depressed (47%) or alcoholic (25%). No other single predictor was present in more than 5% of the suicides.

Others show that hopelessness is a better predictor of suicide, suicidal ideation, or the wish to die than depression.[97]

Studies on five high-suicide-risk groups showed that social isolation and perceived burdensomeness stood out as correlates of suicidal ideation. Similarly in a study of psychiatrists' reports on their patients' suicides, three variables were seen as frequently present in the month preceding suicide: feeling a burden on others; social withdrawal; and help negation.[98]

Past suicidal behavior is considered one of the greatest indicators of suicide risk. Yet, care should be given to distinguish between suicide attempters and suicide completers, and potential firsters, especially those who have not presented suicidal behavior. Even though the behaviors are closely related, there are some major differences. Hardly anyone who attempts suicide wants to die, they just think so at the time. The lethality of prior attempts is important to determine the probability of completing a suicide. The

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95 M. David Rudd, Thomas Joiner & M. Hasan Rahab, *Treating Suicidal Behavior.*2001. Page 18.

96 Thomas White, *How to Identify Suicidal People.*

97 R.W. Maris, et al., *Assessment and Prediction of Suicide.*

98 Thomas Joiner, *Why People Die By Suicide.* 2005.

vast majority of attempters (90 percent) never end up committing suicide, therefore using a previous attempt as a suicide predictor results in overestimating the risk for this group of people.

Most of the information we have on firsters is derived from psychological autopsies. This term refers to a procedure fro reconstructing an individual's psychological life after the fact, particularly the person's lifestyle and thoughts, feelings, and behaviors manifested during the weeks preceding death. Suicide experts agree that no one yet knows the best way to plumb the depth and breadth of any specific case of suicide, given the limitations of current knowledge, and that no one can give an authoritative conclusion explaining why a particular suicide occurred.

People who have suicidal delusions are at an extremely high risk for suicide. These delusions, generally defined as fixed false beliefs, may not have anything to do with a death wish, but instead may be the by-products of irrational thought processes

Most risk factors have been identified after the suicide using large groups of people and even though they represent epidemiological data, when applied to a single person, they may have little importance, or interestingly, the opposite of the anticipated effect.

J. Fawcett and his colleagues have separated some factors that predispose individuals to suicide over a shorter period of time as opposed to a longer one. They have shown that six clinical features are associated with suicide occurring within the first year of posthospital follow-up: panic attacks, severe anxiety, diminished concentration, global insomnia, moderate alcohol abuse and anhedonia.

Verbal communications about suicide occur in 94 percent of suicide completers and 92 percent of suicide attempters within one year of suicide. However, a note of caution should be used, as these verbal communications ranged from direct statements to very indirect references. Many people make references to death or dying without actually making a direct connection with taking their own life.[99] I know of a case where the only potential message was akin to, "I feel like jumping off a building," and this was said in New York City, where this is probably a common saying.

Suicide attempters, as a group, are probably quite different than completers and especially firsters. Yet, many practitioners tend to think that all "suicidal" patients are the same as attempters. They look at several indicators, typical of attempters, such as, a suicide note written or behaviors in preparation for death, such as getting a will or straightening ownership or given away cherished possessions, to determine intent to die, as if this would be representative of all potential suicide cases. They tend to focus on questions such as why does a patient want to die? What are his or her suicidal thoughts? Is there a meaning to death? How long has the patient contemplated suicide? How frequent, persistent or severe are the suicidal thoughts? Sometimes, they want to explore motivation, questioning why he or she has harmed himself or herself. Completers, especially firsters might not even have any such ideas until the very moment when they commit suicide.

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[99] Ibid.

Suicide attempters or self-destructive behavior aimed at manipulating or punishing others should be separated from true suicidal behavior, but it is important to err on the side of caution. Many who talk about suicide or attempt suicide are more interested in finding solutions to their problems than actually dying. They may simply want to let others know they need help. Even though a person didn't intend to kill him or herself, they might die accidentally on occasion. For this reason alone, they should be taken very seriously.

The difference between self-mutilation and suicide is simple, it is the difference between life and death. The definition of self-mutilation is the direct and deliberate destruction or alteration of body tissue without suicidal intent. Self-mutilation, such as cutting, burning the skin, repetitive banging of the head, eye gouging, finger biting, or ingesting strange objects, often ends in serious elf-injury, but the reason for this behavior is not usually to cause death. In some cases, people who self-mutilate consider this as a means of help such as ridding themselves of troubling thoughts and feelings, relieving tension, to obtain a sense of self-control or venting anger and frustration. Even though there is no suicidal intent, serious injury or accidental death might occur and sometimes self-mutilation is used as a means to commit suicide. Almost 7 percent of suicides involved one form or another of self-mutilation.

Prediction of individual suicidal behavior is nearly impossible to achieve with the existing methods. The use of an actual suicide event as a criterion of antecedent risk is questionable, because very high-risk individuals sometimes survive suicide attempts and low-risk persons end up committing suicide. A prediction based on the individual characteristics of a suicidal person is made more difficult because of lack of knowledge or control over such variables as precipitating events, opportunity to act out a suicidal impulse or the availability of the chosen method.

Many suicide prediction scales exist, but the fact remains that the information gained from them or the confidence of the results is unknown. Hardly any existing scale permits an interaction effect among the risk factors. Conflicting results and contradictions exist among the various scales, which indicate that further work in this area is sorely needed. One study, after surveying 19 scales and applying them to five clinical cases found considerable variation in the risk estimates of the subjects.

Most experts advocate a subjective, global suicide risk assessment based on all available information, in addition to rating scales. Clinical judgment is viewed as more important than scores derived from the best empirically constructed tests.[100]

Given the difficulty in suicide assessment, perhaps, at best, in a clinical setting the problem is not suicide prediction, but the identification of a developing suicide crisis. To improve risk assessment, better understanding of the true nature of suicide is necessary.

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[100] Ibid.

Other Considerations

It is outside the scope of this book to look into certain behaviors like pilots who stay with their planes and guide them out to unpopulated areas rather than crashing and killing others, or soldiers who leap on grenades to protect their fellow soldiers; or people that refuse medical treatment because they would rather die than continue to suffer or be a burden on others. It could be argued that these people didn't want to die, therefore their actions, by definition, are not suicide. Other types of behavior considered suicidal such as that of a Japanese kamikaze pilot of World War II, a suicide terrorist bomber, or the 16th century sea captain Sir Richard Grenville and his crew, who blew up their vessel, the *Revenge,* rather than fall into the grip of the Spanish Armada, probably fall into "acts of war" rather than suicide.

There are some suicide attempts that are manipulative without intent to die. Occasionally, these can be distinguished because they have been carried out, usually repetitively, under conditions where rescue is likely and that seem to exact some saving response from a specific person. However, in such cases it is good to err on the side of caution. Manipulative suicide attempts typically involve low lethality or low suicidal intent, yet unintentional deaths can occur, or the attempt might be an effort to inform loved ones or a therapist of escalating suicidality.

The best approach to chronic depression is a combination of medication and psychotherapy. In a recent study sponsored by the National Mental Health Institute comparing cognitive behavioral therapy and drug treatment (Prozac) for depressed adolescents the following was found: 71 percent who received Prozac and therapy responded well to treatment compared with 61 percent who receive Prozac alone, 43 percent who received talk therapy alone and 35 percent of those who received a placebo treatment. The study also found that patients became significantly less suicidal, no matter which treatment they were given. No patient committed suicide during the trial. But the risk of a suicide attempt among patients given Prozac was twice that of those who did not. There were five suicide attempts among those given Prozac and just one among other participants.

In another study comparing Zoloft, an antidepressant similar to Prozac, with cognitive behavioral therapy, in teenagers suffering from obsessive compulsive disorder, it was determined that those that received talk therapy improved more than those with the drug.[101]

The increasing reliance on medications to solve emotional problems (that are probably healthy and need to be worked through) can also be a factor, because as soon as the medication is taken, the emotional maturity that is supposed to develop is warped.

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[101] Gardiner Harris, *Antidepressant Seen as Effective in Treatment of Adolescent.* New York Times, June 2, 2004.

The increase of suicide among the young, starting in the 1950s through the 70s and continuing on till the present, concurred with the appearance of television and could be indirectly related to it in that young children and adolescents spend more time interacting with the tube rather than socially.

Mood disorders are generally accompanied by sleep disturbances. Since the invention of television, the average time people spend sleeping has gone down by as much as two hours. Perhaps, the decreased patterns of sleeping, and not the increased television viewing, are contributing to increases in mood disorders, particularly among the young.

Coinciding with the advent of television, we have the introduction of the pill, and reduction of the average family size. Are fewer siblings, or loneliness a part of the puzzle?

There is probably a deep connection between stressful events and the triggering or worsening of psychiatric illnesses. And under the right conditions, this might well play a role in suicide. Stress has a profound effect not only on the immune system and production of stress hormones but also on the sleep-wake cycle, which is normally affected in mania and depression. It has been demonstrated that psychological stress, certain medications and illnesses, and significant changes in light and temperature can interfere with circadian rhythms.

Stress is also directly related to time of recovery, and the longer the period of recovery, the more extended is the period of vulnerability for suicide.

Prejudice has also been suspected as causing higher rates of depression, as this increases self doubts, undervaluing of people's lives, and ultimately, despair in the face of hatred.

High levels of stress, particularly as those faced by small groups of people living in ancient traditions under the realities of the modern world, such as African tribal communities, Amazonian tribes, subsistence farmers that are being forced to join larger nations, nomadic cultures that are being urbanized generally present high levels of depression. Even in traditional settings, depression has always been high among such people as the Inuit of Greenland, and suicide rates, in some areas, can be as high as thirty-five per ten thousand.

Children of depressed people, though not only because of possible genetic susceptibility, suffer higher depression rates. The earliest signs of childhood depression in infants as young as three months, primarily occur to the offspring of depressed mothers. It is also a well-known fact that depressed mothers are usually not the best mothers to begin with.[102]

Depressed and manic depressive people function quite well between bouts, but the repeated assaults slowly undermine the mind's flexibility and ability to adapt.[103]

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[102] Andrew Solomon, *The Noonday Demon.*

[103] Kay Redfield Jamison, *Night Falls Fast.*

Talking therapies have been proven to be good at keeping depression at bay, and recurrences at a minimum. Good, close, intimate friendships are increasingly more difficult to come by, especially during the formative years of high school and college. Intimate friendships might be part of the solution to avoid depression. Friendships, through sharing of dreams, fears and joys can be more beneficial than therapy—depression can be caused by loneliness. Friendships and family, through love, can be a most wonderful thing and the best medicine. A very old man, when I asked him to summarize a two-day conversation we had, mostly about history and economics, squeezed his hands hard, shook them as if he was going to roll dice, looked me in the eye, and said to me, "When you find love, hang on to it. You never know how long it will last."

I think most would agree that our present world and way of life is increasingly stressful. The lesson is simple; the more stress, the more depression; the more depression, the more suicide.

Suicide prevention groups provide the following advice concerning warning signals: mention of dying, disappearing, jumping, or committing other types of self harm; changes in personality, such as becoming withdrawn, sad, anxious, tired, indecisive or apathetic; change in behavior such as lack of concentration in routine tasks, or changes in eating habits; change in sleep patterns, like insomnia, nightmares or oversleeping; diminished interest in sex or impotence or even missed periods; fear of losing control, feelings of going crazy, or harming others or self. All of this is greatly aggravated with recent loss (death of kin, divorce, job, money, status, self-confidence, self-esteem) and hopelessness (no hope for the future). Of course, any one of these signs might be readily apparent and it doesn't necessarily conduce to suicide.

And last, there are some people that under certain conditions can become suicidal in a very short time, and with brief or no exterior warning signs.

In some cases prevention efforts are targeted at modifying processes and factors that are thought to contribute to suicide at the population level rather than the individual level. However, those charged with the design of such programs should carefully stipulate their assumptions in relation to the "vulnerabilities" predisposing young people to suicide and/or mood disorders. For any chance to succeed, these "processes" should be truly related to suicide, and shifted in the positive direction of unwanted outcomes. There are too many assuming that the problem is one of adaptive developmental functioning or hereditary in predisposing vulnerabilities, instead of the other way around: once the onset of mental disorders are present, adaptive developmental functioning is a symptom and, as a result, suicide is a potential course of action (among others).

These theories postulate that if you reduce a number of things at the general-population level, such as single mothers, single men, abused children, poor self-esteem, and so on, then suicide rates should decline. Following these ideas, assessment of personal predisposition and vulnerabilities and assessment of stress are both critical for predicting, not only, the onset, but also, the course of the disorder. In today's world of

increasing economic well-being for most, all of these factors are being improved. Yet, suicide rates, especially at the youth level, continue to rise. In the U.S. more people die of suicide than of violent crimes every year.

We spend inordinate amounts of money trying to protect society from criminals and bringing them to justice. There are less murders than victims of suicide. It is time to begin to protect, especially our young, against suicide, and help alleviate the pain and suffering that accompanies mental disorders.

It is important to mention people that tend to self-injury behaviors. There is evidence that a primary motive for self-injury in these cases is relief and that they find self-injury rewarding. As they continue to hurt themselves, the primary process of pain fades, and the opponent process amplifies. The opponent process is relief because it distracts from even deeper emotional pain, or because it makes them feel alive, or because it brings their inner world back into harmony with the world at large. Studies shows significant mood elevation as a consequence of self injury. Many patients report that although negative reinforcement (i.e., relief) is a primary motive for self-injury, other motives exist, including positive reinforces such as fascination with the wound and reaffirming the ability to feel. As people proceed to hurt themselves, the perception of pain and fear is diminished and may even begin to feel rewarding.

Many people who show this pattern of behavior eventually kill themselves without intending to do so and can be considered suicidal. By definition, if there was no intention to kill themselves, then there is no suicide. This type of behavior is more similar to Obsessive Compulsive Disorder and even smoking. It should not be lumped together with suicide, it is quite different. Yet, there are probably a few cases where self-injurious behavior might escalate to suicidal behavior. Keep in mind that a certain degree of anxiety is felt for no apparent reason in all three cases, and in each of the three cases, a particular behavior is engaged to reduce the anxiety: cutting in the case of self-injury, carrying out the compulsion in the case of Obsessive Compulsive Disorder, or smoking in the case of nicotine addicts.

A Neurological Model of the Brain

The brain is the organ of the nervous system that controls body functions, movement and initiates actions and reactions as responses to external opportunities and threats. It contains billions of nerve cells called neurons. Neurons communicate constantly with each other and with all parts of the body. The senses are continuously sending information to the brain.

The brain is divided into two hemispheres. Each hemisphere is covered with a thin layer of gray matter known as the cortex. Various areas of the cortex specialize in various functions and ultimately are responsible for many of the higher functions of the brain. Evolutionarily, the cortex developed a series of folds that allows an expanded surface area of cortex to fit in the skull. The cortex communicates with various brain structures, which perform various functions, such as the hippocampus, the thalamus, and the basal ganglia among others. Inside the brain are a series of interconnected hollow spaces called ventricles. They are filled with a clear liquid called the cerebrospinal fluid. This fluid circulates between layers of the protective membranes, the meninges, that surround the brain. The cerebrospinal fluid contains all the chemicals necessary for proper brain functioning. In addition, it helps move wastes produced by the brain and spinal cord into the bloodstream. The brain requires a continuous blood supply. If this blood supply is interrupted for even a few minutes, brain tissue will be damaged. Strokes are caused by blockage or rupture in one or more of the brain's blood vessels.

I am deeply indebted to the coherent and logical presentations and insights of others that have come before me. Without the tireless research of countless scientists, without the brilliant ideas of brain theorists and philosophers, this work would not be possible.

Any model of the brain will have to contend with causes operating at several levels simultaneously, with signals propagated upward and downward (from the center out and vice versa, as well as hierarchically), and, in a chaotic sense, of emerging properties at some levels, combined with random and deterministic processes. Thus, the minute, exact neural details that lead to predictable responses in the brain might prove too complex to unravel in the near future. However, I feel confident that the basic neuronal architecture—the mechanisms, and circuits with their corresponding chemistries—which the brain uses to achieve wondrous results is within our grasp. The brain model presented here is intended to serve as base-starting point to guide future neuropsychological research. Hopefully it will lead to a better understanding of the cause of psychological disorders and consequently to a more rational pharmacology and the development of more efficient therapeutic methods.

For a detailed and complete account I refer the reader to my book, *The Master Illusionist, A Neurological Theory Of Psychology.* Here, I try to give a simplified explanation, hopefully understandable to the lay person as well, of how normal behavior arises in order to understand mental disorders.

Neurons

In the last few decades brain research has reached new heights. It is overdue to synthesize the information coming in from many varied fields and to propose a coherent and logical brain theory. I present a different interpretation of facts discovered by others in an effort to construct a more comprehensive model of the brain. In a court of law, much of what I introduce here would be called circumstantial evidence. But the model presented is based on facts, and the facts seem to fit the evidence.

To begin to understand the mysteries of the brain and the central nervous system, it is necessary to start with a discussion of neurons and their connections. Neurons are the basic unit of the brain and the nervous system. They essentially send electrochemical signals to one another. In large groups, or coordinating between large groups, they store memories, interpret information from the senses, learn, think, deduct, love, govern reproductive behavior, control body movement, and perform all the homeostatic functions necessary for the functioning of the body in an optimal state. Somehow, neurons, with their multiple and complicated interactions, in an emergent sense, are responsible for all the magnificent things the brain performs.

The neuron is a cell specializing in sending and processing information. There are many different types of neurons, but they all have four components: the cell body or soma, where all the proteins and chemicals are manufactured, the axon, the dendrite and the synapse. Neurons communicate with each other though electrical and chemical signals. Nerve impulses are sent down the axon to the synapse. When the impulse reaches the synapse, chemicals are released. Axons can vary greatly in length.

Dendrites, in contrast to axons, are multiple tubular extensions of the cell body. They are the receivers of the chemicals from other neurons. Some are branched with multiple receptor sights that enable them to make contact with many neurons.

The passage of chemicals, called neurotransmitters, through the cell's membranes, changes the electrical potential of the neurons. The various neurotransmitters can have an excitatory or inhibitory effect, meaning that they make it more or less probable that a neuron will fire an impulse.

Neurons are the main component of the brain and the nervous system. In the last twenty years, there have been major breakthroughs in understanding how neurons communicate with each other. We still don't know exactly how neurons can represent or remember things like, my son Mitch, much less. my son Mitch skateboarding in Carmel Valley with his red jacket. But the foundation is laid.

Neurons come in several types, with different receptors. In lay terms, neurons essentially have branching tails that can send an electrical impulse from the main

body down the tail (axon). When the electrical impulse reaches the end of the axon, various chemicals (neurotransmitters) are released into the extracellular space, which is filled with a liquid with many chemicals in solution.

Neurons also have many small branches (compared to the axon) called dendrites. There are numerous and different kinds of receptors on the dendrites. It is to these receptors that the neurotransmitters attach themselves. Think of the neurotransmitter as a key, and the receptor as a lock. Only when the match is right do they attach. When they do so they open a gate and allows ions (electrically charged chemicals) to enter the neuron. When enough ions are present, an electric impulse is sent down the axon.

The space between the dendrites and the axon is called the synapse. The four most important ions are sodium, potassium, calcium and chloride. The first three are positive and chloride is negative in charge. In this way, neurons send messages to one another. Some neurotransmitters are inhibitory in nature, others are excitatory, and others can be both, depending on the receptor they bind to.

Each neuron can be receiving signals from many different neurons, some adding, and some subtracting. A neuron may have as little as five hundred and as many as twenty thousand dendrites. The result is that messages sent from one neuron can affect many neurons. At the same time each neuron can receive electrical impulses from many other neurons.

To complicate things more, there are other chemicals—hormones and neuropeptides—which influence how the neurotransmitters work. These chemicals change, ever so slightly, the way the brain operates.

What is evident is that neurons can fire at different rates and, to some extent, in different styles. At any one moment a neuron can send a limited amount of information, yet the potential information coming into it is very large. We cannot look at a single neuron in isolation; we have to consider the combined effects of many. A neuron simply tells others how excited it is, and there is some information in the pattern of firing as well as the average rate of firing. These signals do not inform where the sender neuron is. In the sense that neurons don't know which neurons stimulated them, it means that there is not one neuron that symbolizes my son Mitch; many neurons throughout the brain acting together are a representation of him.

Neuromodulation is the action of many neurons working together and does not occur only at the cellular level, it occurs at every level of brain functioning. Even at the level of the whole brain. Think of alcohol as an example of a substance that can affect one or more neurotransmitters or receptors, and in turn affects human behavior, as big an order of organization as we can get.

We now know that certain synaptic connections are strengthened with use. When that happens, neurons link to each other and form a circuit of synaptic connections. A system is a complex circuit that performs a specific function, like seeing or remembering. The brain uses clusters of neurons to perform very specialized tasks. Some, such as the visual system, extend about ten centimeters, and within such system there are many maps, some as small as one centimeter square. A map is a cluster of

neurons, generally adjacent to each other, that work together when presented with a particular type of stimulus. Within each map there can be many circuits, each contained in about one cubic millimeter. The neurons can stretch over various distances, though many are contained within one tenth of a cubic millimeter.

Synaptic interactions between two types of neurons are particularly important in understanding the functioning of the brain. Projection neurons have relatively long axons that extend out of the area where their cell bodies are located. Their main job is to reach out to other circuits. Their main function is excitatory. Projection neurons are responsible for sending echo and handshake signals.

The echo signals go to the center of the brain (primarily the thalamus) to help coordinate the relay of incoming signals (stimulus) from the body and senses. An echo signal, as the name implies, is a ghostly representation, an echo of the past, of some event or stimulus. The echoes help serve as traffic directors so incoming signals can be relayed quicker to the appropriate areas.

The handshake signals go from one area in the cortex to another cortical area. A handshake signal, as the name implies, is a signal that is sent out looking for friendly signals or that might have similar characteristics, and when found, a connection is established—a handshake takes place. Handshakes alert, or activate, related bundles of neurons that can have more information that can be used to interpret a stimulus. The handshakes are an automatic process that activates the relevant areas in case they might be needed to help figure out what is going on by producing a particular pattern of signals.

Both handshakes and echoes can carry tags. Tags carry the information to relay a signal to other certain areas.

The second type of neuron is called the interneuron. As the name implies, these neurons connect their short axons to nearby neurons, often projection neurons. Their action is mainly inhibitory. These neurons play an important role in counter balancing the excitatory activity of projection cells. If there were only excitatory impulses, all neurons would slowly be excited more and more; the excitatory activity would escalate like a runaway freight train down a hill, eventually exhausting and even damaging the neurons.

Projection cells tend to be idle in the absence of impulses from other projection cells. They do, however, maintain a minimum background firing, what I called the echo and handshake signals. Interneurons are often tonically active, which means they are firing all the time. This means that the projection neurons have to overcome a degree of initial inhibition in order to fire. Rapidly changing states of excitation and inhibition direct the flow of traffic through the brain. It doesn't take much imagination to see that a minor change in this balance could lead to a breakdown in the flow of impulses and cause havoc.[104]

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[104] For a more detailed description of how the brain works see *The Master Illusionist, A Neurological Theory of Psychology* by Federico Sanchez.

The Body

Movement and sensory perception are associated to certain areas of the cortex. These areas have been studied the most because it is easiest to observe the problems that arise through trauma or stroke to specific sites.

The brain is intimately connected to the body and is a part of the body. The brain is at the service of the body, ourselves, and evolved to aid in maintaining us at an optimum level of adaptation. The brain detects the body and controls movements in different ways: emotional displays, automatic reflexes, and voluntary control.

Emotions are such an integral part of our brains that some body movements are emotionally triggered, like smiling when we see a loved one or an automatic expression of disgust in the presence of putrefied food. A spontaneous smile is produced by the basal ganglia, clusters of cells located next to the thalamus. When you see a friend, the visual message of his or her face reaches the emotional center or limbic system and is relayed to the basal ganglia, which in turn orchestrates the sequences of facial muscle activity needed to produce a smile. When this loop is activated by the correct stimulus, your smile is genuine. Everything happens in a fraction of a second without the thinking parts of the cortex ever being involved. A pleasant echo matched a pleasant stimulus, and a smile was activated automatically.

What happens when someone asks us to smile? The verbal instruction is received by the higher thinking centers in the brain, including the auditory cortex and language centers. From there it is relayed to the motor cortex, which specializes in producing voluntary movements. Despite the apparent simplicity, smiling involves the careful orchestration of dozens of tiny muscles in the appropriate sequence. As far as the motor cortex (which doesn't specialize in smiling) is concerned, this is as complex a feat as playing Rachmaninoff with no piano lessons, and consequently the effort fails completely. Your smile is forced. You look like an idiot[105] with a frozen expression.

When a person suffers a stroke in the right motor cortex-the specialized brain region that orchestrates voluntary muscle movements on the left side of the body—problems appear on the left. Many stroke victims are paralyzed on the right or the left side of their bodies, depending on what side the brain injury occurred. Yet, when these patients yawn, they stretch out both arms spontaneously. Much to their amazement, their paralyzed limb comes to life. It does so because there is a different brain loop involved in yawning, and that brain loop also controls arm movement—this loop is closely linked to the respiratory centers in the brain stem. These movements are emotional (smiling) and reflexive (yawning), respectively. They are not voluntary muscle movements.[106]

Voluntary movement is controlled by different brain circuits. The primary motor cortex is located just in front of the central fissure that separates the frontal and parietal

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[105] V.S. Ramachandran, *Phantoms in the Brain*

[106] Ibid.

lobes, and the supplementary motor area and the premotor cortex are adjacent to the primary motor cortex.[107] The primary somatosensory cortex is just behind the central fissure, running alongside the primary motor cortex. There are rich connections between all these regions, especially between the primary motor cortex and the primary somatosensory cortex.[108]

The body is normally mapped to the primary motor cortex and the somatosensory cortex, for movement and sensation respectively. Each part of the body is represented disproportionately to its size, according to the number of neurons (sensitivity) in each part. The more sensitive parts have a greater cortical area devoted to them. If we follow the strip of the somatosensory cortex from the top of the brain down, starting on the inside fold of the hemisphere, we find, first the genitals, then above it the feet and legs; now as you turn onto the outside of the hemisphere, we find the trunk, neck, shoulder, arm, followed by the hand, with the fingers receiving a disproportionate area, especially the thumb; next follows the head from the top down, meaning forehead first and ending with the lips (again, disproportionately represented) and chin.; and last the thorax and voice box (the pharynx). These disproportionate representations are due to the fact that the lips and fingers, for example, are much more sensitive to touch and are capable of very fine discrimination.

For the most part the map is orderly though upside down: The neurons controlling the foot are represented at the top and the outstretched arm is at the bottom. Upon closer examination, the map is not entirely continuous. The face is not near the neck, where it should be, but is below the hand, and the genitals instead of being between the thighs, are located next to the foot.[109] When you touch any part of the body, you can measure the firing of respective cells in the cortex. Conversely, if you stimulate electrically any part of the motor cortex (which is mapped similarly), the corresponding body part will move. A simple action, like reach out with a right arm and pick up the cup, is translated from a simple command, "pick up the cup," into a coordinated sequence of myriad instructions to the multiplicity of muscles necessary to accomplish the task. It is a top-down process. A simple command goes down a hierarchy, ultimately activating many individual muscles in the right sequence.

To understand how the brain manages and controls movement, it is helpful to look at some abnormal conditions.

The persistence of sensation in limbs long after amputation is a well-known phenomenon called phantom limbs. When you lose an arm, the arm stops sending signals to the arm area in the cortex, and in the absence of signals, the synaptic connections between neighboring areas, in this case the face area, become active and invade the area that used to be the hand. Because of this invasion, when certain areas

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[107] Carl Sagan, *Dragons of Eden*

[108] Jean Talairach and Pierre Tournoux, *Co-Planar Stereotaxic Atlas of the Human Brain.*

[109] Carl Sagan, *Dragons of Eden.*

of the face are stimulated, the corresponding missing arm area fires away, creating the sensation that the arm is there. In this way, an equivalent map of the missing arm is transposed onto the cheek. If the loss of the arm is slightly above the elbow, the upper arm area will also invade the missing arm area. In this case there will be a new map of the missing arm transposed on the face and also on the upper arm near the shoulder.

Generally, neurons in adjacent areas are interconnected. Connections between the adjoining hand and face maps ordinarily are inactive, but in the absence of signals from the hand, the hand map "looks" for other inputs. In so doing the inputs that were "silent" from neighboring areas become the new inputs fooling the hand area to believe that the hand is still there.

When you decide to move your hand (the real hand), the chain of events leading to its movement originates in the frontal lobes—especially in the motor cortex. It has been determined that the motor cortex is concerned mainly with simple movements like wiggling your finger or smacking your lips. The supplementary motor area is in charge of more complex skills, such as waving good-by. It oversees passing the specific instructions of the required movement in the proper sequence to the motor cortex. Lastly these signals will travel down the spinal chord to the appropriate muscles on the opposite side of the body, making the specific movements take place as the muscles contract.

Every time a signal is sent from the supplementary motor area to the motor cortex, it is relayed to the muscles and they move. Identical copies of these signals are sent to the cerebellum and the parietal lobes. Once these signals are sent to the muscle, a feedback loop is set in motion. As the muscles execute the movement, signals from the spindles and joints are sent back to the brain, informing the parietal lobes and the cerebellum that, "yes," the movement is being properly executed. There is also another channel of feedback communication in the spinal cord; the motor (down) pathway connects with the upcoming sensory pathway. As a result, the loop is closed quicker as confirmation is sent back to the brain that the motor command is on its way before it reaches the muscle and the motion is executed. These two structures help to compare your intention with the actual performance and modify the motor commands as needed. Thus intentions are transformed into smoothly coordinated movements.[110]

These connections in the spinal cord remain intact after the loss or amputation of the phantom limb. This short feedback pathway between motor instructions and sensory signals of the missing limb combine with the mixture of the sensory information from the face and upper arm, through remapping (creating the phantom hand), and converge with the confirmation of the motor command to the missing hand, which produces a vibrant, dynamic image of the phantom hand—an image that is updated as the phantom hand moves. This creates the very realistic illusion of movement in the phantom limb.

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[110] William H. Calvin, *The Cerebral Symphony.*

In the case of a paralyzed limb, the signal to move is sent, but vision confirms that there is no motion. As the signal is sent out again, many times, the visual feedback confirms no movement—and a learned paralysis is stamped into the brain's circuitry.[111] If the limb is later amputated, sometimes the paralysis is passed on to the phantom!

When patients report having a paralyzed left phantom limb, using mirrors and the right limb to fool the brain into "seeing" the phantom limb, some patients have learned to un-paralyze their phantoms. By seeing the phantom limb (seeing the mirror-image of the real limb), and then, willfully moving both simultaneously, movement is restored to the paralyzed phantom. In some cases, the phantom limb disappeared in part or completely. This suggests that when the parietal lobe is presented with conflicting signals—visual feedback relaying movement while the muscles are saying the arm is not there—the mind resolves the paradox with a form of denial—there really is no arm there.

Pain is one of the least understood of all sensory experiences. Pain in a phantom limb is a source of great frustration to anyone who experiences it. Occasionally, one complaint is that the phantom hand curls up into a tight, white-knuckled fist, fingers digging painfully into the palm. With the use of the mirror box, the patient positions his or her remaining hand to superimpose its reflection over the phantom hand, and after making a fist with the normal hand, tries to unclench both hands simultaneously. About half the patients report, some even on the first try, that the phantom hand opened, lessening the pain immediately.

Consider what happens in your brain when a motor command is sent from the premotor and motor cortex to make a fist. Once the hand is clenched, feedback signals from the muscles and joints in your hand are sent back through the spinal chord to the brain saying, "Slow down, enough. More pressure and it could hurt." This proprioceptive feedback applies brakes, automatically with great speed and precision. When the limb is missing, the feedback isn't possible and the brain continues to send the signal, Clench More. Motor output is amplified even further (to a level that far exceeds anything you or I would ever experience) and the "sense of effort" may itself be experienced as pain. The mirror works by providing visual feedback, taking the place of the proprioreceptive feedback, and the hand can be unclenched.

But why the sensation of nails digging into the palm? There is a memory link in the brain, between the motor command to clench and the unmistakable sensation of "nails digging", so you can readily summon the image in your mind. Yet, even though you can imagine vividly the nails digging, you don't actually feel the sensation and say, "Ouch, that hurts." The reason is that the real skin on the real palm is reporting that there is no pain. In the case of a phantom hand, there are no signals countermanding to forbid those stored pain memories.

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[111] V.S. Ramachandran, *Phantoms of the Brain.*

It seems extraordinary that you could use a visual illusion to eliminate pain, but bear in mind that pain is an illusion—constructed in your brain like any other sensory experience. Using one illusion to erase another shouldn't be so surprising after all.

There is a deeper message here: Your own body is a phantom, one that your brain has temporarily constructed purely for convenience. This phantom allows the brain to control the movements of the body.[112]

What can be more real than our own bodies? However, we have seen how the brain has carefully created a map of our body to control it in order to navigate through the environment and to communicate our internal state to others. The body we experience is essentially a phantom that feels and corresponds to our experience of ourselves in contact with the environment. It is a continuously updated illusion that relies on the most recent memories of our body and its changes in relation to its parts as well as in relation to the world around us. The brain constructs one continuous, seamless map, integrating the visual map of the outside world with the internal, proprioceptor map of our bodies. Our bodies are in reality memories of perceptions of various parts integrated into a whole.

A memory of each complicated movement has to be stored and activated at the appropriate time. Many movements can't be figured out in advance and therefore need many hours of practice to be able to repeat consistently. Practice, after all, makes perfect. The computations involved in coordinating all the muscles of the body to achieve a particular movement, like running, for example, cannot be done in real time. To place one foot in front of the other, to coordinate the arms and head and torso to maintain balance—these sequences have to be memorized, and with small variations from the cerebellum can be managed in real time.

When you imagine moving your fingers, the supplementary motor area becomes active, just as if you moved your fingers. It is remembering the movement, so to speak. When you do move your fingers, the motor cortex becomes active also. The supplementary cortex is thus receiving signals through various parts: from the thalamus and the somatosensory area of where the body parts are; from the parietal lobes it receives signals of the body image and spatial orientation and from the prefrontal connections impulses that help get sequences of movements in the right order for the motor cortex to execute.

The supplementary motor area has as many connections to the spinal cord as the primary motor cortex. The connections between the supplementary motor and the primary motor cortex are just as rich. It also has extensive connections to and from the parietal lobe (information about body image and spatial matters) and to the ventral thalamus (and through there to the basal ganglia, another major component of the movement-control system) that the motor cortex doesn't have. The basal ganglia, with these circuits and loops, integrates the extrapersonal with the intrapersonal space creating one single seamless space.

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[112] Ibid.

It is worthwhile to see in detail some of the connections of the different layers of the cortex as this will help to understand how the echoes from the cortex aid the thalamus in reflecting (relaying) signals. The cortex is made of up of six thin layers, named from the outside-in, layers I through VI. Echoes from the cortex aid the thalamus to coordinate where to send the incoming sensory signals (the thalamus matches incoming signals with echoes) and reflects (relays) the signals accordingly to the cortex for further processing.

The echo signals from the somatosensory, supplementary motor, the parietal (which in turn is also receiving signals from the visual system) and the anterior prefrontal lobe are continuously streaming to the thalamus waiting for signals from the body indicating a change (no signal, means no change), and as soon as a proprioceptive signal is received and matched (by the thalamus) to an echo, it is relayed (reflected) to the appropriate cortex area. This last receiving area in turn activates all the relevant areas in the cortex by sending handshake signals, and in this fashion coordinates everything necessary to send the signals to the motor cortex in the right sequence for whatever movements are needed in a seemingly effortless manner.

There are two types of thalamic nuclei, depending on the afferent fibers they receive from ascending pathways or descending from the cortex. First order nuclei receive their primary afferent signals from the ascending pathways and corticothalamic afferents from layer VI, which also send signals to the thalamic reticular nucleus. The second, higher order nuclei receive most of their primary afferents from the pyramidal cells in cortical layer V. However, these corticothalamic projections don't branch out to the thalamic reticular nucleus.[113]

First order nuclei of the thalamus match echoes to incoming sensory signals and reflect the signals to layer IV, the input layer of the appropriate sensory areas. Excitation in layer IV causes neurons in the same column in layer II and III to become excited, which activate neurons in layers V and VI. The cortex processes signals in a hierarchical manner by sending forward and feed-back handshakes (information). Converging forward handshakes from lower areas arrive in layer IV, which activate layers II and III in the same area, which project axons to the higher-level areas. Thus information flows up the hierarchy. But information also flows down the hierarchy through feedback handshakes. Layer VI neurons project to layer I in the area hierarchically below. In layer I the axons spread horizontally, thus information flowing down hierarchically from one column has the potential to activate many columns in areas below it. Neurons from layers II, III and V have dendrites in layer I, so they can become active by the feedback running through layer I. Neurons in layers II, III and V can excite neurons in layer VI, which in turn project to layer I of the next level below.

Synapses close to the cell body have a stronger influence on whether a neuron will generate a pulse compared to synapses further away. However, the cast majority

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[113] Guillery RW. *Anatomical evidence concerning role of the thalamus in corticortical communication: a brief review.* J Anat 1995 Dec;187(Pt 3):583-92.

of synapses are not close to the body. As a general rule, forward handshakes (neural impulses) moving up the cortical hierarchy are transferred via synapses close to the cell body, which increase the certainty that they will pass from area to area. Also as a general rule, feedback handshakes, flowing down the hierarchy, do so via synapses far from the cell body. Cells in layers II, III and V send dendrites into layer I and form many synapses there.[114] But layer I is a mass of synapses receiving input from the higher cortical areas and input from reflected signals from the thalamus originated by layer V echoes. These signals propagate over a large amount of columns in a local area. So even though these synapses are far from layer II, III and V neurons, the multiple inputs amplify the effect. We also have to keep in mind that in some cases the feedback projections vastly outnumber the forward projections, which indicate that feedback is just as important or more. In this way the forward and feedback handshakes have more or less the same certainty of being sent up and down respectively.

The neurons in layer V project to the second order nuclei, which in turn project back to layer I. Thus the echoes coming from layer V assist in directing the reflecting of signals to layer I. When the sensory signals are reflected to layer IV, handshakes are immediately sent through layers II and III to the next level above. When the signals are reflected to layer I, it reinforces the signals from layer VI from the next higher level. There is a delayed feedback here that Jeff Hawkins proposes is similar to auto-associative memories that learn sequences.

In order to free the brain for other activities, the brain excels at automatizing sequences of movements by storing them in memory, particularly if they are part of habits or routines. The sequences of movements are activated through echoes and handshakes, which are an integral part of the memory of a movement; for example, walking or the action of hammering a nail, or getting dressed. To change how you do these activities requires attention.

The primary motor cortex differs from that of sensory areas in the other lobes. Whereas the sensory areas have a thick layer IV (mostly input from the thalamus) and a thin layer V (where descending projections originate to the brain stem, spinal cord and striatum, and the thalamus), the primary motor has a thin layer IV and a thick layer V. The thalamic terminations, in the motor areas, have a wider laminar distribution than sensory areas.[115]

Some movements are closely linked to emotions and consequently are orchestrated by the limbic system, which controls emotions. It is important, from a social view, that emotions be communicated to others, and this is accomplished by gestures and body postures. Facial expressions are universal across cultures and some are even universal across species. Think of aggression and the baring of teeth. Perhaps a small variation of the baring teeth evolved into a smile, as a signal that we are friend and not foe.

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[114] Jeff Hawkins, *On Intelligence*.

[115] John H. Martin, *Neuroanatomy*.

Most of us find it relatively easy to read different expressions, of disgust, shame or fear, for example. There is a natural, close relationship between our emotions that produces characteristic facial expressions and body language across many cultures: anger, sadness, joy, disgust and surprise.

The memories of our body movements and face gestures or body postures generated by reflexes, feelings or by voluntary action are a part and parcel of our everyday existence. As such, we need to consider communication with hand gestures, body postures and facial expressions as an integral and basic form of language that the brain uses to communicate our internal state to others. As a good (and highly evolved) example of this we have pantomime and dance.

The interconnectedness of the motor system with the senses, of the motor system to memories, of the senses to memories, of the motor system with the expression of emotions should be a fundamental part of any brain theory.

Vision

Vision is one of the most studied senses, at least in neurological terms. Vision is complicated enough to be thought of as three separate senses—motion, color, and luminance (contrast).[116] From any point of view, seeing is a complicated process. The visual system consists of one very large primary system, a secondary system, and a number of minor systems. They all receive input from some of the million or so ganglion cells (neurons) at the back of the eye. The primary system connects to the visual cortex in the occipital lobe, via a part of the thalamus called the Lateral Geniculate Nucleus (LGN). The secondary system projects to the superior colliculus at the top of the midbrain. Each eye sends signals to both sides of the brain, so that both the left and the right visual cortex are receiving information from both eyes.

Any particular ganglion cell will respond vigorously to a small spot of light turned on (or off) in one particular part of the visual field. In total darkness, a ganglion cell fires at a low, irregular rate, called its background rate. A spot of light on the retina will generally excite a group of ganglion cells, though not all to the same degree.

There are two types of ganglion cells, and both are divided into two classes of ganglion cells; the M cells and the P cells. The M cells send signals faster to the brain. They respond well to small differences in light intensity and so handle low contrast well. The P cells are more numerous and their responses are more proportional to their input than the M cells. They are more interested in fine detail, higher contrast, and especially color. The neurons of the retina are not just transmitting raw information; they have started the job of processing information and are doing so in more ways than one.

Both the M and P cells send their axons to the LGN in the thalamus, where it is relayed to the visual cortex. The retina also projects to the superior colliculus. P cells do

[116] Jeff Hawkins, *On Intelligence*.

not do so, but some M cells do. The lack of P-cell input means the superior colliculus is colorblind. Each eye projects to both sides of the brain, but it does so in such a way that the left side of the brain receives input relating only to the right half of the visual field, meaning the right half of the left eye, and the right half of the right eye. So everything we see to the right of our center of gaze goes to the left LGN, on its way to the visual cortex and also to the left superior colliculus. The two halves of the brain exchange information through several tracts of nerve fibers of which the largest is the corpus callosum.[117]

The colliculus is a three-layered structure. The neurons in the upper region are selective for movement and are very interested in small stimuli and their response to the change of light is often very transient. These are all factors that command attention; they signal, "Look out, there is something there."

The LGN is a relay; the principal cells receive input directly from the retina and send their axons directly to the visual area of the cortex. There are no other neurons in between, hence the name relay. The M and P inputs are largely kept apart, as are the inputs from the two eyes.

So far we have only talked about the principal excitatory cells. Inhibitory neurons use neurotransmitters that make the firing of other neurons less probable, and therefore they need to receive many more signals to fire. The inhibitory neurons fall into two main classes, those in the LGN proper and those in a thin sheet of cells called the reticulus nucleus of the thalamus. This thin sheet of cells surrounds much of the thalamus. Its neurons are all inhibitory. They receive excitation from most of the axons passing to and from the cortex and they interact with each other. Their output is mapped onto the underlying neurons of the thalamus immediately beneath them.

If the thalamus can be described as a gateway to the cortex, then the reticular nucleus would be the guardian of the gateway. Here is where the main matching of stimulus to echoes is achieved and the orchestration of where and how to send the impulses begin. This is when awareness starts; this is where attention is controlled.

Visual awareness is the result of the brain's attempt to make sense of the information coming into the eyes and to express it in a compact and well-organized manner. This information is likely to be needed in several distinct places; the higher planning levels for use of spatial relations and the motor system, to allow us to navigate through the world. Visual attention, when engaged, also sends the signals to the hippocampal system (involved in the temporary storage or coding of episodic memory).

Vision seems to be a hierarchical process, where at each higher step, more information is interpreted according to the output of the lower steps, which are also affected by feedback signals. This view is supported by the general responses of the neurons in the different areas. As we ascend the hierarchy two rough rules apply, (1) the sizes of the receptive fields increase, so that for the highest areas they often cover the whole visual hemifield and even part of the other half of the visual field

[117] Francis Crick, *The Astonishing Hypothesis.*

(connected via the corpus callosum); and (2) the features to which neurons respond become more complex.

Neurons in different areas respond to different characteristics; in one area, some neurons respond to certain contours, while in another area they respond in a less simple way to patterns of movement. Neurons in another adjacent area, fire in response to movements that correspond to approaching objects, in other areas to receding objects. In another area, neurons respond to perceived color, rather than a particular wavelength.

Moving to higher areas we find neurons that respond to the front view of a face. Other neurons respond best to a face seen in profile. In one area, neurons are mainly interested in where an object is in relation to the head or the body, and much less in what it is. The inferotemporal regions are more concerned with what it is.

The general pattern is that each area receives several inputs from lower areas. These lower areas have already extracted more complex features than the rather simple ones to which the primary visual area responds. It then operates on this combination of inputs to produce more complex features, which are then passed on to higher levels. At the same time, the information flows in somewhat separate but interacting streams.

The whole system does not operate like a one-shot, static picture. It operates by many transient, dynamic interactions, conducted at a fairly fast rate, producing a continuous dynamic representation of the world. Eventually, memories of objects are constructed and stored by categories that generally have shared characteristics, i.e., memories for animals, man-made objects, faces, plants, tools, and so on.

Eye movements are also part of the visual system. The eyes make quick movements, called saccades, about three times a second. The eye moves and stops. The stops are called fixations. For instance, each V1 (in the occipital cortex) neuron might fire vigorously when a line or an edge slanted thirty degrees enters the receptive field. The slanted edge has little meaning; it could be the side of a palm tree, or the side of the letter M, or a hill. With each new fixation the neurons receptive field comes to rest on a new and entirely different portion of visual space. On some fixations the cell will fire strongly, on others it will fire weakly or not at all. Thus in each saccade, many cells in V1 are likely to change their activity.[118] Higher up the hierarchy, other cells will fire and remain active as long as the palm tree, the letter M or a hill are in the visual field.

When viewing a face, the sequence of patterns determined by saccades is not fixed. Sometimes the fixations will follow an "eye-eye-nose-mouth" order, at other times the order might be "mouth-eye-nose-eye." The components of a face are a sequence. They are statistically related and tend to occur together in time, although the order might vary. If you perceive "face" while fixating on "nose," the likely next saccade will fixate on "eye" or "mouth."[119] The cortex certainly would not expect "pen" or "car" to appear in the next saccade.

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[118] Jeff Hawkins, *On Intelligence.*

[119] Ibid.

In general there exist two visual systems; one that creates a spatial representation of the environment; and, another, which interprets what it is that we actually see.

The spatial map is only concerned in how close or how far objects are, on how even or uneven the ground is, how high or low obstacles are. This map will be used to control the body as it moves through the world, as well as aiding the neck and head movements to focus our senses on particular details of the external environment.

The interpretation of what is "out" there is accomplished through a complex system of memories. Essentially we "remember" what we see. In lower (hierarchical) visual areas, neurons that respond to contours, or lines, or colors, or movement, pass on this information upwards, where more complex representations can be made. Thus, a slanted line and skin color, plus a particular contour or context will be interpreted as nose. Therefore, if an eye is near, a face is automatically identified, and higher up, our mother's face will effortlessly be identified.

Seeing is a hierarchical processing of signals. It is worthwhile to see how neural impulses are sent up and down the cortical hierarchy. This is very similar to what was presented in the previous section. Different senses follow the same cellular arrangements. I have adapted, with the addition of echoes, handshakes and reflected signals, the following from Jeff Hawkins, *On Intelligence*. First let's see how forward handshakes move up the hierarchy. The initial reflecting of sensory signals from the thalamus arrives at layer IV, the main input layer. Consequently, converging inputs from lower regions always arrive at layer IV of higher areas. In passing they also form a connection with layer VI. Layer IV neurons then send projections to layers II and III within their column. When a column projects information up, sends forward handshakes, many layer II and III neurons connect to layer IV of the next higher area.

Signals flowing down the hierarchy, the feedback handshakes, take a less direct path. Layer VI cells are the downward-projecting output cells from a cortical column and project to layer I in the regions hierarchically below. Here in layer I, the axons spread over long distances in the lower cortical region. Thus signals flowing down the hierarchy from one column has the potential to activate many columns in the areas below it. There are very few cells in layer I, but neurons in layers II, III, and V have dendrites in layer I, so these cells can be excited by the feedback [handshakes] distributed all across layer I. The axons coming from layer II and III neurons form synapses in layer V as they leave the cortex and are believed to excite cells in layers V and VI. So we can say that as information flows down the hierarchy, it has a less direct route. It can branch in many directions via the spread in layer I. Feedback information starts in a layer VI neuron in the higher regions; it spreads across layer I in the lower region. Some neurons in layers II, III and V in the lower area are excited, and some of these excite layer VI cells, which project to layer I in regions hierarchically below, and so on.[120]

[120] Jeff Hawkins, *On Intelligence*.

Specific sensory signals activate related memories through handshakes. Related memories include expectations of what will happen next. As related memories are activated some neurons in layers V and VI send echoes to the thalamus to help orchestrate the distribution of sensory signals. In the case of vision it takes several levels of processing up the hierarchy to get to these related memories, lets call them expectations or predictions. Motion, boundaries, and so on, create an expectation of what will appear in the visual field next. The echo that matches best with what actually occurs next will determine, moment by moment, which way to propagate the visual signal. In this way the various possible predictions guide what we see, and what we see trims the number of possible predictions. The feedback handshakes going to layer I assist in interpreting the small changes that a particular object goes through as it moves through our visual field. Even though an object's perspective, distance from us, or its movement or rotation changes, we still recognize the object and keep track of the specific details and changes through time.

The visual inputs, through handshakes, activate related areas in the auditory and tactile memories, and in this way create a deeper understanding of what we see. The handshakes activate areas that are related to expected events, which depend on the physical movement of the body and the changes in orientation of the head or eye movements. In this elegant way, the visual field is adjusted to remain constant, instead of the ground passing below us or the sky passing above us as we move forward, or bobbing up and down as we walk.

The feedback handshakes from these other areas, tactile or auditory, as they flow back, can be interpreted as prediction signals because they are sending information related to what is expected (i.e., how the ground will feel as our feet touch it when we walk, or the sound of our footsteps.) The way the cortex achieves these predictions is analogous to how the motor cortex generates movement. Information flows through the hierarchy from high to the lower areas. Sensory signals simultaneously flow in anywhere and everywhere in the hierarchy—and then flow back down all areas of the hierarchy, leading to predictions or motor behavior. Although the motor cortex has some special attributes, it is correct to see it as just a part of one large hierarchical memory-prediction system. It's almost like another sense. Seeing, hearing, touching and acting are profoundly intertwined.[121] And, as we will see later, memories are intensely sensitive to emotional states. Consequently, there is an emotional component to what our senses interpret and what our responses are.

It has been known that particularly large layer V neurons within the motor cortex (M1) make direct contact with muscles and motor regions of the spinal cord. These neurons literally make the muscles contract and relax. If you speak, type, or perform any sophisticated behavior, these neurons are firing on and off in a highly coordinated way. Recently, researchers have discovered that large layer V neurons may play a role in

[121] Ibid.

behavior in other parts of the cortex. Large layer V neurons in the visual cortex project to part of the brain that moves de eyes (area 8). So the visual areas of the cortex, such as V2 and V4, not only process visual input, but they help determine the movement of the eyes themselves, and therefore what reaches the visual cortex. These large layer V cells are found throughout the cortex, suggesting a more widespread role in all kinds of movement.[122] More importantly, the axons of these large layer V neurons split in two. One branch goes to the thalamus sending the echoes of anticipated movements and serves to find matches with the proprioceptive signals, adjusting and orchestrating the overall motor output. However, these echoes produce the reflecting of signals to layer I, activating broad areas of columns. The echoes from layer VI make the reflected signals go to layer IV of a single column. When both signals coincide, the anticipation of expected signals can be narrowed to a single solution.

Ambiguous inputs need to be resolved by ultimately activating a single column but generally several columns are activated reflecting the ambiguity. Therefore a column with strong inputs should inhibit the activity in other columns. These interneurons only affect the area surrounding a column, but many columns in an area can be active. In essence, when a signal arrives from the thalamus or from a layer below, it activates cells in layer IV. Layer IV neurons activate layer II and III neurons. Layer II and III neurons send forward handshakes to a higher-level area, as well as activating layer V and VI neurons within the same column. The whole column becomes active when receiving a reflected sensory signal or a signal from a level below. Layer II, III and V have many synapses in layer I. If these synapses are active when layer II, III and V are firing, the synapses are strengthened. When these synapses become strong enough, they can make cells in layers II, III and V fire even when layer IV cells haven't fired. Thus through handshakes in layer I, neurons learn to anticipate even before receiving signals from layer IV. When a column becomes active via layer I, it is expecting to receive a signal from layer IV from an area lower in the hierarchy. It is anticipating, it is predicting. The layer VI neuron also becomes active simultaneously and sends a signal to layer I of a lower area, reinforcing the input signal (if it arrives) into layer IV of the lower area. Only the column where the signal arrived becomes reinforced and inhibits its neighbors standing out more clearly.

Under certain conditions, half the input to layer I is driven by activity in layer V. This represents what was happening moments before. When the order of these patterns occurs consistently over time, the columns will store the sequence of patterns, and fire one after another in proper sequence. The other half of the input to layer I comes from layer VI neurons in the hierarchically higher area. This information is more stable. It represents the "name" of the sequence. With respect to sound, if your columns are phonemes, then it is the spoken word you are hearing. If the columns are active with spoken words, then the impulses from above are the speech you are reciting. Thus

[122] Ibid.

the information in layer I represents the name of the sequence (through feedback handshakes) and the last item of the sequence (through forward handshakes). In this way, a particular column can be shared among many different sequences without getting confused. Columns "learn" to fire in the right context and in the correct order.

However, neurons receive and send input to many surrounding columns. Ninety percent of all synapses are from cells outside the column, and most of the synapses are not in layer I. Neurons in layers II, III and V have thousands of synapses in layer I, but also thousands of synapses in their own layers. Usually, activity in nearby columns has a strong correlation. For example if a line is moving or changing orientation in your visual field, it will activate successive columns. More often, the information needed to predict a column's activity is more widespread, thus the role of layer I synapses.

In the lower levels of the cortical hierarchy, each column's activity changes with each new sensory input. But as you go up the hierarchy, columns remain active prior to the arrival of new inputs from the lower areas. The activity of layers II and III passes on to a higher level through projections to layer IV. The activity of these cells is the input to a higher level. But that's a problem. In order for the hierarchy to work, a constant pattern during learned sequences must be relayed; the name of the sequence has to be relayed. Before learning a sequence, only the details can be relayed. The cortex needs some way to keep the input in the next region constant during learned or memorized sequences. We need to turn off the handshakes of the layer II and III neurons when a column predicts its activity, or, alternately, to make these cells active when the column can't predict its activity. The echoes from layer V fit the latter alternative. Echoes are set to match learned stimuli, otherwise there is only a direct relay of signals to the higher areas of the cortex. The echoes from layer V (which are representing learned stimuli) cause the thalamus to reflect the signal to layer I of the next higher cortical area. This activates neurons in layers II, III and V, which in turn make layer VI neurons fire back to layer I of the lower area reinforcing the original reflected signals to layer IV. In this way, memorized stimuli keep layers II and III sending forward handshakes to the next level and become stable.

Before learning, columns can only become active via layer IV inputs. After learning, columns can become active through memory.[123]

The classical Hebbian algorithm using auto-associative memories can learn spatial patterns and sequences of patterns. The main problem is that the memories can't handle variation well. Jeff Hawkins proposed that the cortex has gotten around this problem by stacking auto-associative memories in a hierarchy and partly by using a sophisticated columnar architecture. I have added the use of echo and reflect signals through the thalamus to make the model of the cortex more powerful.

Paradoxically, the way we see is by remembering what we have seen before and the way we remember is the same as how we see. The cortex uses interconnected memory systems to solve many problems.

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[123] Ibid.

It is worthwhile to see in added detail how vision works through memories. Let's suppose a slanted line is detected at the first or second level in the visual cortical hierarchy. This could be interpreted as part of a nose, the side of the letter "W" or the side of a sand dune. Layer VI sends an echo to the thalamus, which reflects the signal back to layer IV. Layer V sends and echo to the thalamus, which reflects the signal to layer I of the next higher area.

At this point, the slanted line could be any of three things (could be a lot more), and consequently layers II and III send a forward handshake to the next higher level. In this case, three different areas, the "nose" area, the "W" area and the "sand dune" area. Simultaneously, other aspects of the scene, let's say the "contour" area determines to forward the signal to "nose" area, the "sand dune" area, the "cliff" area, but not the "W" area. This will strengthen the "nose" and "sand dune" area. If simultaneously we add the signal from the "color" area, lets say the color of skin, this is also forwarded to the "nose" area, and perhaps the "cliff" area. The echo of layer V is reflected to a higher level, in this case, the slanted line, or the color of skin or the contour is reflected to the nose area. The input of the reflected signal to layer I of the "nose" area, in addition to the forward handshake to layer IV, establish that as long as any of these inputs is present, nose is a better alternative. The feedback handshake from layer VI to layer I of the areas below strengthens the signal even more. The result is that the slanted line, the contour and the color now stand in for "nose."

At this level, the "nose" interpretation is passed on to a higher area, the "face" area, sending a forward handshake from layers II and III to layer IV. The echo from layer V, again will have the thalamus reflect the signal to layer I. Simultaneously, other facial features, lets say "eyes" or "mouth" (if a face is present) will converge on layer IV of the "face" area, and their respective V echoes will reflect the original signal to layer I. In just three or four levels of the hierarchy, we have gone from slanted lines, contours, color, and so on to "face." The interesting thing is that as long as the "face," "nose," and/or "slanted line," "contour" and "skin color are active, the slanted line, contour or color stand in for "face." The original sensory input now is interpreted as a more complex object. One more level up would get us to "animal faces" or "human faces" and then on to my "wife's face."

The general pattern is that each area receives several inputs from lower areas. These lower areas have already extracted more complex features than the rather simple ones to which V1 responds. It then operates on this combination of inputs to produce more complex features, which are then passed on to higher levels. Simultaneously, the information flows in somewhat separate but interacting streams.

At certain levels in the hierarchy, some cells become active and stay active when entire objects appear anywhere in the visual field. In the IT area, a cell might fire robustly whenever a face is visible. As we move from the retina to the IT area, neurons change from quickly changing their rates of firing depending on if they are spatially specific or tiny-feature recognition, to constantly firing, spatially nonspecific, object recognition groups of neurons.

Information also flows downward, from higher areas to lower areas. There are bundles of axons that go from IT to lower areas like V4, V2 and V1. Moreover, there are as many if not more feedback connections in visual cortex as there are feedforward connections.[124] The feedback handshake signals alter the feedforward signals as they indicate what an object is and also anticipate what will happen to the object in the visual field, as we move through space, or even as saccades change the focus of the eye.

Axons from layers II and III generally form synapses in layer V as they leave the cortex. Likewise, axons projecting to layer IV from lower areas of the cortex make a synapse in layer VI. So here we find an intersection where the forward and feedback handshakes converge forming a specific prediction out of the many possible. The other columns representing other potential predictions don't meet these criteria, and therefore don't fire. Neurons in layer VI will fire when this happens, and this will be interpreted as seeing or about to see something specific. The job of neurons in layer VI is to announce to lower areas that a specific representation of the world, whether true, imagined, or expected is happening.

Feedback is needed for the cortex to make predictions. Prediction requires a comparison between what is happening and what is expected to happen. What is happening flows upward and what is expected to happen flows downwards. Where the two sets intersect this is what we perceive. Combining partial prediction with partial sensory input resolves ambiguity by filling in missing information and deciding between alternate possibilities.

Neurons in layer VI, in addition to sending their output to layer I of lower cortical areas, can also send their output into layer IV of their own column. When they do, the predictions become the input. In the case of vision, this is how we visualize in our "mind's eye." In the auditory regions, we "hear" voices as we think.[125]

The whole system does not operate like a one-shot, static picture. It operates by many transient, dynamic interactions, conducted at a fairly fast rate, producing a continuous dynamic representation of the world. [126]

We have to keep in mind (a figure of speech) that there is no such thing as a direct perception. We don't have a "mother" or "father" sensor. The brain is in a dark cavity in our skulls with nothing other than neural impulses streaming in through our input sensory signals. The motor cortex generates motor commands inversely as the sensory areas interpret the sensory signals, but memories of events are generated similarly to motor commands. On the sensory side a wide variety of inputs becomes a stable cell assembly that represents some abstract concept. On the motor side a stable cell assembly representing an abstract motion (hitting a tennis ball or signing

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[124] Jeff Hawkins, *On Intelligence*.

[125] Ibid.

[126] Francis Crick, *The Astonishing Hypothesis*.

your name) is carried out using many muscles and respecting a wide variety of other constraints. This symmetry should not be surprising if the cortex runs a single algorithm through out.[127]

What can be more real than what we see out there? However, we have seen in a very simplified way how the brain effortlessly (seemingly) constructs an image of our environment and interprets it, not only spatially but also contextually. What we see feels real, because it is in accordance with our past experience. It seems to be an exact match of our past memories. The brain creates another great illusion using the light that impinges on our eyes.

Memory

In my discussion of movement and vision, the concept of memories becomes vital. The different memory systems are a central part to understanding the brain's functions and malfunctions. These memory systems are distributed throughout various areas of the cortex and are sometimes shared for diverse senses.

Memories with shared characteristics help to interpret more of the information coming into the brain from the senses or the body; for example, memory banks of faces, of plants, of tools, of animals, and so on. The cortex in Homo sapiens is mostly an expanded memory system that allows more memories to be stored by categories, by context and by emotions. The memory systems that are used by the different senses to extract the most information from the world around us are sometimes shared, as this information needs to be integrated into a coherent whole. Our increased memory systems have been (evolutionary speaking) co-opted for further tasks.

The same visual memories that help us interpret what is in our visual field allow us to "see" in our "mind's eye" and to imagine as well as manipulate objects in "space" and "turn" them in our "heads".

Hearing memories used by the brain to interpret the information from our ears allow us to make sense of speech sounds, building from sounds into words, and in turn give words a syntactical and grammatical meaning, and eventually we "think" (using these voices) in terms of a voice in our "head". Actually several voices or ideas are continuously going on inside my and everyone else's "head".

Most of the ideas presented in this section are taken from Daniel Schacter's excellent book, Searching for Memory.

Our memories feel uniquely ours, quite distinct from everybody else's. We feel this way because our memories are rooted in the ongoing series of episodes and incidents that constitute our daily lives. Our subjective sense of remembering the past is such a familiar and frequent part of our inner lives that we may fail to see any need to examine it. As we think back, we may feel as though we are focusing on images, sounds and

[127] Jeff Hawkins, *On Intelligence*.

emotions that are slumbering somewhere in our memory. As plausible as this seems, it is fundamentally misleading. Our experience of remembering an event does, naturally, partly depend on information about the event that has been stored in our brains. However, there are other contributors to the subjective sense of remembering, and to appreciate memory's fragile power we need to understand them.[128]

Every time you start to drive your car, you are calling on old knowledge and skills acquired earlier, but you do not feel you are reliving your past. These uses of the past call on two of the brain's major memory systems: semantic memory, which contains conceptual and factual knowledge, and procedural memory, which allows us to learn skills an acquire habits. But there is something special about the subjective experience of explicitly remembering past incidents that separates it from other uses of memory (storing and retrieving information). In order to be "experienced" as memory, the information must be recalled in the context of a particular time and place and with some emotional reference to oneself as a participant in the episode. Remembering, for the rememberer, is a mental time travel, a sort of reliving of something that happened in the past.

Jeff Hawkins conceived of a thought experiment. He calls it the "altered door." Every one of us goes through our front door when we arrive home. We reach out, turn the knob, walk in and shut it behind us. It is all a habit, like driving a car. Suppose that someone, while you were away, changes something about the front door. It could be anything, change the latch, the knob, the placement of the knob, the weight of the door, or simply make the hinges squeaky or paint it a different color. This time, when you attempt to open the door, you will quickly detect that something is different, either that the location of the knob is different, or the color is changed, or if it feels heavier when you push with the wrong amount of force.

There is only one way to interpret your reaction to the altered door: your brain makes low-level sensory predictions about what it expects to see, hear, feel at every moment, and it does so in parallel. All areas of the cortex are simultaneously trying to predict what their next experience will be. Visual areas make predictions about edges, shapes, objects, locations and motions. Auditory areas make predictions about tones, direction to source, and patterns of sound. Somatosensory areas make predictions about touch, texture, contour, and temperature.[129]

The sensory signal (sight or sound or touch) of something familiar (our door, our house) will be matched by the thalamus to the echo of the memory of that something. This initiates a cascade of handshakes to activate everything relevant to the stimulus, which include the memories of what is expected to happen next.

Prediction means that neurons become active in advance of actually receiving sensory input. When they receive the handshakes activated by the initial stimulus, they

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[128] Daniel L. Schacter, *Searching For Memory*.

[129] Jeff Hawkins, *On Intelligence*.

activate an echo, which will make it easier to match with the incoming expected signal. The various possible future scenarios that have been encountered (that are stored in memory) are all activated. When one of them is matched, this in turn will activate the next possible scenarios, eliminating the possibility of the other previous ones.

As you approach the door, your cortex is forming a slew of predictions based on past experience. As you reach out, it will predict what you will feel on your fingers, when you will feel the door, and at what angle your joints will be when you actually touch the door. It will predict how much resistance the door offers when you push it open. When all predictions are met, you are not even aware what your cortex did. But if your expectations about the door are violated, the error will cause you to take notice. Correct predictions result in understanding. Incorrect predictions result in confusion and prompt you to pay attention.[130]

When you remember something, do you see yourself in the scene? Or do you see the scene through your eyes, as if you were looking outward, so that you yourself are not an object in the scene? These two modes of remembering are referred to as field and observer memories, respectively.

People experience more field memories when focusing on feelings and experience more observer memories when focusing on objective circumstances. This means that an important part of your recollective experience—whether or not you see yourself as a participant in a remembered event—is, to a large extent, constructed or invented at the time of recall. The way you remember an event depends on your purposes and goals at the time you recall it. This observation suggests that the emotional intensity of a memory is determined, in part, by the way in which you go about remembering the episode. And the emotions that you attribute to the past may sometimes arise from the way in which you set out to retrieve a memory in the present.

In this context, there is a memory of the feeling of the emotion, which in itself turns on a complete set of memories, relating to that particular emotion. This is coordinated by the caudate nucleus. It detects specific signals from the amygdala and engages the related thoughts and experiences indirectly by signaling to the thalamus and the cortex.

There are two subjective experiences, referred to as "remembering" and "knowing" the past. Several studies have shown that recall of visual information about a physical setting or context of an event is crucial to having a "remember" experience. Why does retrieving visual images tend to make us feel that we are remembering a real event?[131] Part of the reason is that some of the same brain regions are involved in several activities: visual imagery, visual perception and remembering. If we rely on these areas to perceive the external world, it should not be surprising that when we use them to create visual images, they might feel like a residue of actual past events.

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130 Ibid.

131 Daniel L. Schacter, *Searching For Memory*.

This has an important implication: creating visual images may lead us to believe that we are remembering an event even when the incident never happened.

Though it is clearly important, visual reexperiencing is probably not the sole basis of the subjective sense of remembering. We are also likely to feel we are remembering something from the past when we can recall associations and ideas and feelings that occurred to us during the initial episode.

Scientists agree that the brain does not operate like a video camera or a copying machine. Then what aspects of reality do remain in memory once an episode has concluded? What we believe about ourselves is determined by what we remember about our pasts.

Studying patterns of spared and impaired functions that are the result of specific brain damage can be used to infer the structure of the brain. We infer the functional and neural separability of a circuit assumed as necessary for the performance of a task if it can be damaged independently of other processes. This logic can be extended to differences in performance for types of stimuli.[132] For example, some neurons selectively fire when recognizing faces and may be used to infer the existence of special circuits to process faces. However, this observation does not unequivocally support the inference of a distinct circuit for processing faces. It could be that faces differ from other objects not in terms of a processing circuit, but in the levels of processing complexity.[133] There are, however, many areas that appear to have distinct processing mechanisms. For example, the dissociation of processing words versus objects indicates the existence of two distinct circuits, one related to hearing and the other, to the visual.

The general idea that memories are built from fragments of experience can help understand key aspects of the rememberer's recollective experience, as well as memory distortions and effects of implicit memory.

Research has shown that short-term memories last for only seconds. These temporary records on which attention is working is called working memory. There are momentary short-lasting handshakes established between different areas of the cortex to keep attention focused on these bits of information to allow us to act upon them.

Long-term memories depend on a different network of brain structures than working memory. People with damage to the inner part of the temporal lobes in the center of the brain have no difficulty retaining a string of digits for several seconds, yet have great difficulty forming and remembering enduring memories. Other people with damage to a specific part of the parietal lobe on the cortical surface can form long-term memories, but cannot hold and repeat a string of digits. They lack a specific part of working memory, known as the phonological loop that most of us rely on when we need to hold a small amount of linguistic information for several seconds.[134]

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[132] Ibid

[133] Ibid.

[134] Ibid.

To establish a durable memory, incoming information must be encoded much more thoroughly or deeply by associating it meaningfully with knowledge that already exists in memory.[135] In other words, extensive handshakes must be established between many of the characteristics of an object or event. Attention to shared characteristics with previous memories will allow a quicker and more extensive series of handshakes to be established, as well as easily tying into the previous existing sets of handshakes. These will be activated by reflecting echoes that are similar. It is a highly efficient system to add memories and tie them into the old ones.

What we already know shapes what we select and encode; things that are meaningful to us spontaneously elicit the kind of elaborations that promote later recall. Our memory systems are built so we are likely to remember what is most important to us.[136] Think of having gone to the last family reunion where one hundred relatives showed up. You can probably name the one hundred people from memory because of all the deep associations to each one of your relatives: cousins, brothers, spouses, and their children, not to mention that you know them all well. However if you went to a reunion with a hundred strangers and you were introduced to all of them, you most probably would have a difficult time naming even a few of the people that you met with any accuracy.

Encoding of novel events involves the hippocampus. Research has shown that brain-injured patients with damage to the hippocampus can produce a severe loss of memory for recent experiences.

The hippocampus is one of several anatomically related structures that play an important role in explicit remembering.[137]

In general, the cortex interprets stimuli by using memories, and matching echoes to stimuli. As the stimulus is passed up the hierarchical areas of the cortex, the handshakes activate expected patterns, which in turn generate new echoes, which when matched, confirm the expectation by reflecting the signal to the area that generated the echo. However, if the lower levels cannot interpret something, the stimulus rises up the hierarchy until an area "knows" how to interpret the stimulus. When events (in other words, patterns) occur that aren't anticipated, the signals are relayed up the hierarchy until an area can make sense of them. If lower regions fail to predict expected patterns, this is considered an error and they pass the signal higher up. This is repeated until an area anticipates a pattern.[138]

Ultimately, the hippocampus is activated in the presence of novel stimuli. The hippocampus lies at the highest level of the cortical hierarchy. The hippocampus stores the new memory, and slowly transfers it to the cortex for later recall.

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[135] Ibid.

[136] Ibid.

[137] Ibid.

[138] Jeff Hawkins, *On Intelligence.*

The hippocampal response to a novel event is activated when stimuli and echoes from the cortex don't match. The hippocampus then alerts the thalamus, which automatically signal the frontal lobes (regions 9 and 10) and activates area 40—we recognize this process as paying attention. Attention will produce a series of handshakes to many other regions in the cortex to find similarities with the present stimulus. In the absence of a match (then it must be novel), the hippocampus is activated to search for other similarities or alternate contexts, sending its own impulses hoping to find more echoes from the frontal lobe activation and start the encoding process. Once attention is on the stimulus, another network may come on line, involving the left frontal inferior lobe, which in turn makes a wealth of semantic associations (area 40 and 39) and knowledge available if needed. In this way the necessary associations between past experiences and the novel stimulus help encode in a manner that will increase the probability of being able to recall the new stimulus. As a consequence, novel events are much easier to remember than specific events that have been encountered many times repeatedly.

There is a concept called an engram, defined as transient or enduring changes in our brains that result from encoding an experience.[139] The brain records an event by strengthening the connections (establishing a handshake) between groups of neurons that participate in encoding the experience. Handshakes will quickly activate all the relevant connections. In the future, when one of the echo signals of any of these differing regions is matched by a new stimulus, the whole network (engram) will be activated. A typical incident in our lives consists of numerous sights, sounds, actions, smells and words. Different areas of the brain analyze these varied aspects of an event. As a result, neurons in the different regions become more strongly connected to one another. This new pattern of connections constitutes the brain's record of the event: the engram.

As you read these words, there are thousands, maybe millions of engrams in some form in your brain. These patterns of connections have the potential to enter awareness, to contribute to explicit remembering when the echo signal finds a match and activates the engram; but as long as the echoes aren't matched, these engrams lie dormant, waiting for the right match to be activated by the right stimulus. Only a fraction of the original event need be present in order to trigger recall of the entire episode.

The brain ultimately engages in an act of "construction" during the retrieval process. Posterior regions of the cortex region that are concerned with perceptual analysis hold on to fragments of sensory experience—bits and pieces of sights and sounds. Various other regions of the cortex contain tags, in the form of handshakes, that bind sensory fragments to one another and to preexisting knowledge, thereby constituting complex records of past encodings. A memory is triggered when an echo and its tags simultaneously link sensory fragments that were part of an episode.

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[139] Daniel L. Schacter, *Searching For Memory*.

This retrieved memory is a temporary orchestration of activity in several distinct regions—the voices, the images, the sensations, and the feelings—a reconstruction with many contributors.

An increased blood flow in the frontal lobes (particularly the right lobe) during explicit retrieval reflects the mental effort involved in searching memory[140]. This search for echoes and tags that could be a close match to the required retrieval request is slow and methodical.

There are different memory systems using various tactics to encode and retrieve. The active effort of remembering something specific is known as strategic retrieval. When we remember something through associated characteristics it is called associative retrieval.

If the strategic retrieval system is impaired and the automatic retrieval process is intact, it should be possible to remember reasonably well in the presence of the right matching of stimulus to an echo.

For the rememberer, the engram (the stored fragments of an episode) and the memory (the subjective experience of recollecting the past event) are not the same thing. The stored fragments contribute to the conscious experience of remembering, but they are only a part of it. Another important part is the stimulus and the match with the echoes, which is generally referred to as the cue. The cue activates the engram, which produces a new, emergent entity—the recollective experience—that differs from its constituents.

When we encode an experience, connections between active neurons become stronger, and this pattern of brain activity becomes the engram. Later, as we try to remember the experience, a retrieval cue (stimulus matched to an echo) will induce another pattern of activity in the brain. If this pattern is similar enough to a previously encoded pattern, remembering will occur.[141] When we remember, we complete a pattern with the best match available in memory.

Memories are an emergent property of the cue and the engram. How do we convert the fragmentary remains of experience into an autobiographical narrative that endures over time and constitutes the stories of our lives?

Daniel Schacter mentions three kinds of autobiographical knowledge arranged hierarchically. At the highest level we find lifetime periods: lengthy segments of our lives that are measured in years or decades, say living in Carmel, or when the kids were little. In the middle of the hierarchy we find general events: extended, composite episodes that are measured in days, weeks or months such as playing soccer in college, vacationing at the Grand Canyon, or the first professional job you had. The bottom of the hierarchy would cover event-specific knowledge: individual episodes that are measured in seconds, minutes or hours, such as the moment you first saw the Grand

[140] Ibid.

[141] Ibid.

Canyon, the guy that knocked your teammate unconscious, or when you forgot your homework.

Lifetime periods help us to find general-event knowledge and event-specific knowledge; they provide the skeletal structure of our autobiographical memories. This leads to the prospect that there is no single representation or engram stored in memory that has a one-to-one relationship with the mental experience of recollecting one's past. Instead, such experiences are always constructed by combining bits of information from each of the three levels of autobiographical knowledge. Just as memories for individual events resemble jigsaw puzzles that are assembled from many pieces, so do the stories of our lives.

What we experience as an autobiographical memory is constructed from knowledge of lifetime periods, general events, and specific episodes. These in turn are put together by images, sounds, thoughts and feelings.

Moreover, there seem to be three different long-term memory systems: episodic memory, which is for recollecting specific incidents from our pasts; semantic memory, part of the vast network of associations and concepts that underlies the general knowledge of the world; and procedural memory, which allows us to learn skills and know how to do things.

What we see and what we hear, what we think and what we feel are linked together to form new explicit memories by this neural system. Thus they are also essential for episodic memory as well as contributing to the formation of new semantic memories. They are highly selective of novel events.

The brain systems that support episodic and semantic memories allow us to recognize objects in the world, to travel in time, and to construct our life stories in a seemingly effortless manner.

When people are influenced by a past experience without any awareness that they are remembering, this is defined as implicit memory.

The hippocampus with its specialized GABA neurons (inhibitory) that are electrically coupled by gap junctions can activate many interconnected cells at once. In this way signals can be sent simultaneously to many different regions to find a match (or a close match) and activate a small part of a memory. Quickly the when, the how, with whom, at what time, in what period of my life and so on is remembered, not necessarily with complete accuracy. It is a reconstruction process that can be affected by newer experiences, as well as by similar memories.

A novel and shocking event might activate a special brain mechanism. The event is so important that the memory might be stored as if a picture has been taken. This "freezes" whatever happens at the moment we learn of the shocking event. It is as if a flashbulb went off. Appropriately, this phenomenon is known as flashbulb memory. Although flashbulb memories are not one hundred per cent accurate, a high level of confidence in a memory is a hallmark of flashbulb memory.

Memories of episodes that are highly emotional are different from ordinary memories. Might it be that traumatic emotional memories are unusually accurate and

might depend on special brain mechanisms? Stress hormones act on the brain and create a state of heightened alertness and reinforce the circuitry involved in memory formation.

Traumatic memories are generally more accurate than other memories, but sometimes are subject to distortion. These distortions are attributable to perceptual errors that occur at the time of the event, caused by the stress of the shocking episode. A related kind of emotional filtering seems to occur with combat "flashbacks" of war veterans, which are often so intense that the veterans report they feel as though they are reliving an actual experience. Flashbacks sometimes contain elements of real, imagined, and/or feared events.

Suicidal, depressed patients remember the general emotional gist of past experiences, but do not recollect as many specific details as nondepressed people do. Patients' depressed moods focus attention on the general negative themes in everyday incidents that fit their previous negative experience. People suffering from depression learn and remember negative events better than positive ones.

Thinking in different modalities basically is an exadaptation of our hearing and visual sensory systems. The memory systems that are used by these sensory systems have been greatly expanded (compared to other mammals) and are also interconnected through semantic memory. Thinking, in terms of voices in our head, is an expansion of the use of hearing and the associated memory systems. Similarly, thinking, in visual images, is an expanded use of the cortex areas used to process vision. Thinking and communicating can also be done using body language and motion.

What can be more real than our memories? Ultimately, we are only what we can remember of our lives. If we have many happy memories, we will say that we have had a happy life. If, on the contrary, we have many sad memories, we will feel that our lives have been sad. Under certain conditions, as we will see, hardly any memories will be accessible.

However, as we have seen, what we remember is a reconstruction of past episodes using combinations of memories, not necessarily an exact recall in every detail. But we do, in general, retain the gist of what our lives are. We do retain a general notion of who we are, but in a sense these memories are a great illusion created in our brains that allows us to stay alive.

Awareness and Attention

Arousal is a minimum state of activity in the brain that permits the use of the senses for gathering information from the environment. It involves a preliminary detection system and is mostly concerned with spatial orientation. Awareness is primarily involved with where objects are in relation to the body.

There are at least three awareness system: a somatosensory awareness that maps the relative positions of all parts of the body with respect to one another, as well as processing information from the tactile and temperature signals sent by all receptors

in the skin; a hearing awareness; and a visual awareness. These three awareness systems are closely coordinated by the thalamus. The thalamus refers temporally in a sequential order the stimuli it receives. These three systems working in unison allow for a seemingly effortless body orientation through space. Gustatory and olfactory awareness systems may also be involved.

Automatically, awareness carefully controls eye, neck and head (primarily) movements to center the senses on relevant stimuli as needed as the body moves through space. The awareness systems are genetically pre-wired and are carefully coordinated by the superior colliculus at the top of the mid brain. The superior colliculus receives inputs from the eye, ear and body. These inputs are roughly mapped. The basal ganglia, through the reticular formation, the midbrain and the cerebellum, coordinate body movements automatically to help direct the senses. The neurons in the upper level of the colliculus are very selective for movement, small stimuli in the field of vision and momentary changes of light. All of these are factors that command attention, and the thalamus is automatically activated to do so in a bottom-up fashion.

Attention evolved through natural selection to focus on important stimuli conveyed by the senses to the brain. When an appropriate stimulus, determined by the thalamus through matching echoes and incoming sensory signals, is detected, or when a certain threshold of change is detected, the thalamus, through the attentional systems, activates all relevant memories to that particular stimulus; in this way the brain interprets better and quicker what is the nature of the stimulus. Attention ignores most stimuli to deal more effectively with one object or situation. Attention deals with things serially, first with one object, then another. This natural process helps focus on ever-changing internal or external stimuli concerned mostly with appetites, emotions, thoughts (internal) and the changing environment (external).

Each area of the cortex attempts to interpret the signals reaching it as part of known sequences of patterns, or memories. If they can, they pass on a stable pattern—the name of the sequence—to the next higher area. If an unexpected pattern arrives, then this is what is passed up to the next higher area. The higher area might be able to interpret the pattern as part of it's own sequence. If so, it will feedback to the lower area and produce a stable output. Simultaneously, it will also send a stable pattern up to the next higher area. The higher an unfamiliar pattern needs to go to be interpreted, the more areas of the cortex get involved in resolving the unexpected input. Finally when a region thinks it can understand the unexpected event, it generates a new prediction. This prediction propagates down the hierarchy as far as it can go. If the new prediction is incorrect, an error will be detected.[142] Two things happen at this moment: a) an echo is sent to the thalamus, and b) the unknown stimulus will be sent up the hierarchy until some region can interpret it as part of a known sequence or memory. The echo that was sent to the thalamus automatically triggers the attentional systems. This would be

[142] Jeff Hawkins, *On Intelligence*.

a bottom-up initiative. When this happens, the attentional systems focus the senses on the unknown and thus activate a whole new set of forward handshakes.

In a world that is known and predictable, most of the up-and-down flow of patterns happens rapidly and occurs in the lower areas of the cortex and everything is quickly resolved. Confusion occurs when the cortex can't find any memory that matches the stimulus. Thus the stimulus races all the way up the hierarchy. Until finally, a high level prediction is made. If it is correct, the prediction travels down the hierarchy all the way to the bottom. In less than a second all areas are given a sequence that fits the stimulus.[143]

When the highest areas in the cortex can't understand the stimulus, the brain interprets it as novel, a never encountered stimulus, and as a result it is passed on to the highest area of the brain, the hippocampus. The hippocampus then initiates the construction of the new memory.

Attention is necessary for encoding a stimulus for later recall. The handshake (associative) signals that it uses to help interpret what the object is, in what context, where in space and time, will be used to represent the event, and later can be available for recall. The handshakes can include referred information to help keep events in order.

When attention is focused on extrapersonal space, it constructs the present. Attention is equivalent to remembering the now. Our previous experiences, the knowledge we have stored, of objects, people, faces, movements, actions and so on, will determine how we construct the present.

Semantic memory is continuously integrated to our senses to interpret the incoming signals. Attention filters out unattended events or objects, and, conversely, an attended event is reacted to more rapidly, at a lower threshold and more accurately. Attention helps encode the present and remembering retrieves the past. Remembering is paying attention to the past. The present has the added quality of being accompanied by sensory information that is perceived in almost real time. The past lacks this quality, and attention detects the difference.

The thalamus automatically and continuously shifts attention by genetically encoded routines. When the signals from our senses change, the thalamus relays this change and attention shifts to focus on the change. The thalamus achieves this by sending signals to the frontal lobes, which have rich connections to the three other lobes, the hypothalamus, the areas that control eye movement and speech, and to the tegmentum, which coordinates neck movements to orient the senses. The eyes shift ever so often, generally attracted towards moving objects; the hearing shifts from one sound to another, and then back, mostly checking if the sound is still the same. No change means nothing new.

Also, after a certain time interval, and there is evidence that this response might be genetically set also: when nothing is new, attention shifts to something else. Attention

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143 Ibid.

shifts probably entail a three-step process: disengage, move, engage. Attention focuses the senses on the new stimulus; otherwise, attention, when not activated by the thalamus, will be downgraded to awareness. Awareness is continuously monitoring the spatial and temporal aspects of our environment, as the thalamus is searching for signals, ready to activate attention to help deal with the unexpected. Under certain conditions attention can appear to be absent, but sometimes it simply has shifted inwards: attention is focusing on internal handshakes from the entire cortex; attention is focused on thinking or on the body itself; or attention can be in "remembering" mode.

The three attentional systems are activated through three sets, each made up of three pathways from the thalamus. When these areas (the visual associative, the auditory integration, and the somatosensory associative) become activated, we feel that we are paying attention.

Through interconnections between them, all attentional systems are activated simultaneously: the auditory, the visual and body attention. In this way, there is attention on the environment visually and auditorily with special attention to where the body is in relation to the extrapersonal space. Any one of these attentional subsystems could be activated alone for specific tasks, for example, listening to music or watching a tennis match. When one of these systems is not functioning properly, we might not be aware of it, as we can't pay attention to it.

Depending on the nature of the stimulus, as well as the sensory type of input, the thalamus will use a different pathway to relay the stimulus to each of these areas. The thalamus can also speed up a response when needed by getting ready to relay the response signals to the body.

Attention also refers events sequentially in time in such a way that the episode, when it becomes part of our immediate past memory, will be perceived as being in concurrence with all our past experiences. The episode will be remembered in the right sequence, as indexed by the thalamus. Even though most everything happened outside consciousness, we still feel that we were not only witnesses to the event, but were active participants and decision makers during the event. We might even remember the event in slow motion, as the adrenalin rushing though our system temporarily sped up everything, including all our cognitive and perceptive functions, as well as our motor responses.

The use of the attentional system for what we call remembering is a constructive process. The past is reconstructed from fragments of memories of the event. The reconstruction of a past episode will not only depend on how the different parts of the episode, the where, with whom, when, the how, and so on were encoded, but also on the specific cue that was used to elicit the memory. The difference between remembering and the present is a distinct feeling that the memory is a past part of our lives—we know we are not actually seeing or hearing or feeling the past event, even though we can clearly re-enact or communicate what made up the past event.

The brain uses speech to communicate what it is thinking to other brains. When it is not communicating with others, the brain uses an internal speech to think, except

that the mouth, tongue and vocal chords are not being activated. It is a process similar to imagining moving a finger, but not really moving it. The brain imagines speaking, but doesn't speak. The brain uses auditory areas to think, crafting, in effect, an internal "voice". When attention is focused internally on these areas it is concentrating on what it is "saying," that is, thinking.

In the same way that the brain uses the memory systems of our hearing sense to interpret speech, to speak and to think (verbally), the brain uses the memories of the visual system to imagine or visualize, and to manipulate and turn objects in our "mind's eye". The brain can think visually, even though it can only communicate this form of visual thinking by translating it to speech, which can produce distortions from the original thought.

Areas in the inferior temporal lobes, lying adjacent and below the auditory integration area and immediately to the front of the visual and auditory integration system, suggest an architecture for thinking, where coordination between these areas and the frontal lobes tap into the incredible stream of memories that is always present as echoes and handshakes throughout the cortex. Thinking is a process that literally happens all over the cortex, tapping into a multiplicity of memory systems and coordinated between the frontal and temporal lobes.

When different emotions are deployed, the brain detects these subtle changes in the body, manifesting a particular feeling. What feels like thinking is a continuous stream of memories, and can happen in several modalities simultaneously. What we call thinking is the sounds or visions or body gestures that are endlessly firing away, stored in our memory systems. What we call ideas and imagination is this gushing flow of pulsating neurons that represent combinations of past experiences, reinterpreted auditorily or visually.

Thinking purposefully requires attention. Attention is the mechanism that can bring into focus a desired way of thinking to reach a formal solution or potential course of future action. Attention is a very quick and rich way to access many semantic memories, episodic memories and past experiences and make them available to the thinking process. In this way, attention can activate any one or all of our thinking modes.

Thinking logically requires communicating with one's self in a way similar to how we communicate with others, that is, in a manner that is comprehensible. Comprehension makes sense of stimuli and ideas. While attention can only be paid to one idea at a time, thinking is a sequential set of ideas flowing in a way that can be comprehended. Coordinating the flow of ideas in such a fashion is a learned process where only some associations are acceptable and others are not. It is a complicated phenomenon, best illustrated with speech; and speech is best learnt as a child. Once past a certain age, if one was not exposed to spoken language, this ability is lost.

The modality in which we think, see, speak or use body language depends on what attentional subsystem is being excited. Of course we can think in parallel in all of these modes simultaneously. Different emotions produce subtle changes in the body and brain and affect the modes of thinking by making certain memories more accessible

and others less so. In this way, emotions push thinking in certain directions that have proven successful in the past. In this way emotions affect cognition. It is the richer gamut of memories that humans (compared to other mammals) can store combined with a stronger attentional system that produces what we call a greater intelligence. This intelligence is measured not in what we can think, but in the problems that we can solve. What we can think compared to other animals, is probably just a question of degrees. Moving up the evolutionary ladder, intelligence is just a question of shades of gray.

When attention is focused internally on any one or all of these three attentional centers, we have access to the rich internal space that we call ourselves—our memories, our thoughts, our visions, our aspirations, our appetites, our bodies and our feelings. We cannot underestimate the importance of emotions in generating a sense of self. Thinking happens in the brain, independent of the body, and that is why it feels like it is separate from the body, even though the brain is an integral part of the body. The brain creates a model of the body, but not of itself.

Two distinct processes can shift attention. One is emotional; it switches the focus of attention to the emotional competent stimulus (ECS) if it is considered more important. The other is a voluntary control, which also plays a role in deciding if the emotional competent stimulus is or not more important. The prefrontal lobes have rich connections to the thalamus and can signal it to shift attention. Also, the frontal lobes have been associated with executive functions that are involved in weighing alternative possible courses of action as well as implementing them. This is where decision making takes place, allowing you to choose from different courses of action according to what is happening in the present—what you remember and know about the situation and what can possibly happen in the future according to past experience of different actions.

In complex tasks involving multiple kinds of mental activities, executive functions plan the sequence of mental steps and schedule the various activities, switching attention as needed. Voluntary control, like attention, can only do one thing at a time. It can initiate a multiplicity of movements or a sequence of activities, but these, once initiated, become automatic. Voluntary control is goal oriented. The executive system can be overloaded if it has to work on unrelated goals at the same time, especially if the goals conflict with one another.

The serial nature of attention, essentially dealing with one stimulus at a time while ignoring most others, and then discarding it in order to deal with a newer or different stimulus, is quite reminiscent of consciousness, where we can be aware of only one (or very few) stimulus at a time. Similarly, recalling past events or remembering our lives, is a serial process. We can only remember clearly one event at a time. The similarities between the process of attention, remembering and consciousness jump out: they are serial and exclusionary of other stimuli. All three are variations of one single neural mechanism used in different ways: focusing the senses combined with their respective sensory memory systems, i.e., different uses of attention. Attention constructs the present using incoming sensory data with the aid of sensory memory

systems, remembering reconstructs the past using stored memories, i.e., attention focused internally on past memories. Consciousness is the perception of either of these two with an additional, emotional component, the sense of self, which we will review later.

Thinking or imagining is a further use of the attentional systems using the sensory memory systems in a slightly different form. From this point of view, there is no neural correlate of consciousness. Consciousness is a complex process that "happens" over many different cortical areas and is carefully orchestrated by the thalamus with frontal lobe intervention as well as all the cortical attentional (called by others associative areas) areas. And ultimately, the thalamo-cortical signaling will be regulated, in part, by the output of the basal ganglia.

There is hardly anyone in the world that would dispute that most animals are capable of "paying" attention. After all, we can be certain that most of them react to "important" opportunities or threats because they focus their sensory systems on such stimuli. Whether most animals spend any time thinking, philosophizing, or pondering about what their sensory systems focus on is outside the scope of this study. But I feel confident, that by definition, they are paying attention (their sensory systems are focusing on a stimulus) and therefore, if their brains perceives this, they are conscious to that degree. Many animals, perhaps more than most humans (which in a great majority of cases choose not to or can't pay attention to some stimuli), are more conscious of many environmental stimuli. Animals pay more attention to detail; humans dismiss more information as being irrelevant, and thus pay less attention than, at least, some other mammals. In this way, we could argue that most other animals are more conscious than humankind because they shift attention to specific environmental stimuli easier than humans. However, we cannot disregard the emotional component of consciousness arising from a sense of self. And here is where the problem of quantitatively ascertaining the degrees of reflective consciousness, lies.

No one would argue that fish don't pay attention; when you throw them a morsel to eat, they immediately turn towards it and bite it. However, it would be difficult to ascertain if they have any thoughts or feelings regarding the "morsel," or whether it is simply an automatic, genetically-set reaction to bite the morsel.

The reflective nature of consciousness is probably proportionate to the variety and size of the various memory systems that have evolved. Consciousness is an exadaptation of the sensory memory systems that help interpret what is out there—consciousness comes in gradations, basically by interpreting better the external stimuli (perceptual consciousness), and eventually, with enough memory, the internal milieu—the thoughts and memories—of the brain (reflective consciousness). Consciousness is an emergent property of the attentional systems combined with increased memory capacity and types of memory, of which emotions add to the memories that can be activated and integrated to the sense of self.

Understanding the normal functioning of awareness and attention is basic to begin to comprehend mood disorders.

Were You at Our Tennis Match?

Consider the case of a tennis doubles match. Your opponent serves to you at a modest one hundred miles per hour, and while the ball is approaching, you can decide to go cross-court, down the line, or lob. You also have to decide if you will do this with a forehand or a backhand shot; and once you decide that you are going cross-court with a backhand, you also need to decide if it will be an under spin or top spin shot. And then, in the corner of your eye, you detect a slight motion, perhaps just a hint of the beginning of a motion, of the net man moving towards the center of the court indicating his intention to poach. In that split second, you change your mind, and change your body motion, primarily the orientation of your shoulders, and you execute a shot down the line to take advantage of your opponent's movement.

Experiments on the subjective evaluation of the interval of time that represent the present indicate that this "present" is about three seconds long: all else is mere reminiscence or anticipation.

How finely can we divide this little three-second present? The shortest perceivable time division—called the fusion threshold—is between two and thirty milliseconds, depending on the sensory modality. Two sounds seem to fuse into one if they are separated by less than two to five milliseconds. Two successive touches merge if they occur within ten to twenty milliseconds of one another, while flashes of light blur together if they are separated by less than 20 to 30 milliseconds. Humans consider two events as "presently" perceived, if their temporal separation is between three milliseconds and three seconds.

However, if a sensation on the skin lasts less than 500 milliseconds, an enormously long time compared to the 10-20 milliseconds of transit time required for the nerve signal to travel to the cortex, the stimulation is not consciously perceived. This does not mean that a skin shock has to be at least 0.5 second long in order to be felt, but only that the handshake signals produced by skin shock at the cortex must last at least 0.5 seconds before the skin shock can become part of the conscious experience. If we had to wait 0.5 seconds before experiencing what we touched, our tactile sense would be useless for all but the slowest of physical activities. Typical tactile reaction times are on the order of 100 milliseconds—the time it takes to perceive a touch and push a button.[144] How can we reconcile the observation that 0.5 second of neural activity is needed to build up a conscious touch sensation with the fact that we can feel a touch and take action five times faster than the time these perceptions are required to become conscious?

Axonal spikes travel down a neuron at speeds of 1.5 to 90 millimeters per millisecond, depending on the type of neuron and whether the axon is myelinated or not. In 20 milliseconds this is equivalent to the spike moving between 3 and 180

[144] Nick Herbert, *Elemental Mind.*

centimeters (roughly 1 inch and six feet respectively). Messages between different areas of the cortex travel at speeds of 1.5 to 5 millimeters per millisecond. Messages traveling down the spine move at speeds between 20 and 90 millimeters per millisecond. A neural message takes about the same time to go from one half of the brain to the other as it does to travel all the way down to the leg

What seems to be going on is this: the tactile signal reaches the brain in about 10-20 milliseconds and is not consciously perceived. But this arrival time is unconsciously noted in some way—tagged sequentially by the thalamus. If the cortical activity continues for the minimum time (about 0.5 seconds) to produce a conscious sensation, then the touch is registered as conscious. However, the touch is not experienced 0.5 seconds later: it is instead "referred" to the previous time indexed by the initial pulse arrival at the thalamus. It is as if the initial tactile pulse sets a "marker" in time, and this "marker" is redeemed if future cortical events produce enough sustained neural activity to promote the shock signal into consciousness.[145]

Back to our tennis game: my opponent serves at 100 miles per hour, which is the equivalent of 146.5 feet per second. The court is 72 feet long, so, roughly, I have about 0.5 seconds to respond between the time he hits his serve and the time the ball reaches me. My vision could potentially process about 16 "snapshots" (of 30 milliseconds each) to calculate where the ball is going to be when the time comes for me to hit it (one snapshot about every 4 feet). I don't see a strobe light effect of a ball every four feet; instead, my brain fills in the details and I see the ball in a perfectly continuous trajectory. In that half second I have to decide everything we mentioned earlier.

If I change my mind at the last split second and decide to go down the line, as my opponent tries to poach, this decision has to be made (about 100 milliseconds maximum as the ball hits the court and bounces towards me) before I make contact with the ball. Both my opponent and I report that we waited until the last possible moment, he to move and me to change my shot. Part or all of this decision process was done unconsciously, "referred" back to the sequential times when the sensations arrived in the cortex.

I, however, vividly feel that I consciously experienced all this; that I was a direct participant in this change of decision. I am aware that I changed my mind (and my motions) as a consequence of the movement I detected (about three or four visual "snapshots" of 20-30 milliseconds each) about 100 milliseconds before hitting the ball.

Consider also that when my opponent serves to me, the sound of the ball as it is hit by his racquet (traveling at the speed of sound of about 800 feet per second) takes roughly 90 milliseconds to reach my ear; about the same time that I used to change my mind. I, however, experience the sound as being in exact synchronization with what I am seeing and not as the sound happening when the ball is almost one quarter of the way towards me. I was aware or conscious that the sound was produced simultaneously

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145 Ibid.

with the visual image of the racquet touching the ball. Again, I am "referring" in time the information relayed by the senses so that I experience reality as it actually is, as my experience dictates. Also, this reality is experienced as being "out there", not inside my head.

In experiments with subjects presented with pictures that have a good or bad connotation, it has been determined it takes about 120 milliseconds to activate the regions in the ventromedial prefrontal region that react to unpleasant or pleasant stimuli.[146] Can it be that consciousness would need about the same time or more to be activated? However, there are numerous movements and reactions that need to be coordinated in 10-20 millisecond time responses.

There is a difference between a programmed, deterministic mechanical response and the mental process we call consciousness. Consciousness is more than perceiving and knowing; it is knowing that you know. The brain feels conscious when it thinks. The motion to produce a top-spin forehand can be a memorized (practiced) motion, and the cross-court or down-the-line shot might be a small variation (changing your feet and shoulders) of this movement, and all these movements might be outside consciousness, but we know what the correct motion feels like, and we know that we voluntarily changed our shot at the last split second.

We couldn't have been conscious (thinking) in the real sequential times (or at all) of all events (the sounds and images) that our senses relayed to us. We would not have had time either to coordinate all the signals our brain relayed to our muscles to achieve the desired shot, much less, when at the last split second we changed our minds. Because of the phenomena called "referred" in sequential time, everything was presented to our consciousness in the correct temporal sequences, and sight and sound were synchronized to match our experience. We couldn't have been conscious in real time; however it feels like we were there, and didn't miss a thing! The brain is a great illusionist.

Emotions

In this section on emotions, I borrow ideas from Antonio Damasio's book *The Feeling of What Happens*, and Joseph LeDoux's book *The Emotional Brain*. However, there are a few changes and departures from their ideas.

From our own experience, we know that emotions can be most varied. Some responses are easily apparent; think of the muscles in the face adopting configurations that are typical of joy, sorrow or anger, or of the skin blanching as a reaction to bad news or flushing in a situation of embarrassment. Consider the body postures that signify joy, defiance, sadness, or discouragement; or the sweaty and clammy hands of apprehension; the racing heart associated with pride or the slowing, near stillness of the heart in terror.

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[146] Joseph LeDoux, *The Emotional Brain*.

Emotions produce physical modifications by triggering myriad chemical changes; release hormones into the bloodstream and produce a series of alterations in the organs, body and brain. When the brain experiences emotions, the hypothalamus, basal forebrain and brain stem release neurotransmitters, such as monoamines, norepinephrine, serotonin and dopamine, and by doing so, the brain temporarily changes the working of many neural circuits. As a consequence of the increase or decrease of these neurotransmitters we have a sense of thoughts speeding up or slowing down, as well as a sensation of pleasure or unpleasantness.

The changes are controlled by two different routes: the bloodstream, where chemical molecules act on receptors in the cells that constitute body tissues; and, neural pathways where the electrochemical signals are sent to other neurons, muscular fibers or organs (such as the adrenal gland) which in turn can release chemicals of their own into the bloodstream. The result of this coordinated chemical and neural change is a change in the state of the organism. This includes changes in the body and the brain itself. The release of neurotransmitters alters the mode of processing of numerous brain circuits, activates related memories, triggers certain specific behaviors (e.g., bonding, playing or crying), and modifies the signaling of body states to the brain.

Pain and pleasure are two different regulatory modes. Pain causes organisms to close themselves in, freezing and withdrawing from their surroundings. Pleasure, on the other hand, is associated with behaviors such as seeking and approaching. Pleasure causes organisms to open themselves up and out towards the environment, approaching it, exploring it, thus increasing both their opportunity for survival and their vulnerability.

Different emotions are produced by different brain systems. However, the brain induces emotions from a remarkably small number of brain sites, most of them subcortical. The main subcortical structures are in the brain stem region, hypothalamus and basal forebrain.

Emotions are the result of largely unpredictable combinations of several concurrent regulatory processes engaged as reactions to whatever internal metabolic adjustments are needed; and coordinated with whatever external situations are being handled by other emotions, appetites, or intellectual calculations. This ever-changing result of this cauldron of interactions is our “state of being;” good, bad, or in-between. When asked, “How we feel,” we consult this “state of being,” and answer accordingly.

Each emotion helps the brain monitor the state of the body and indicates a slightly different state. The perception of an emotion is what we call a feeling. This feeling will be specific to each emotion. We speak of the thrill of surprise, the tingling sensation that goes down the back when something extraordinary happens; twinges of guilt; throbs of passion; pangs of sadness; gnawing grief. Our language reflects a changing physiology as feelings shift. These subtle changes in physiology also affect the brain. Memories of past experiences and feelings are intimately woven and

trigger each other. Particular tastes and smells, not surprisingly, have strong links to memories and feelings.[147]

There have been many attempts at classifying emotions in various categories. Most of these have been manifestly inadequate. Antonio Damasio[148] finds it helpful to classify the emotions in three tiers: background emotions, primary emotions, and social emotions.

Background emotions are very subtle, but remarkably important. These can include slight malaise or excitement, edginess or tranquility, energy or enthusiasm. They reflect very small changes in the internal state of the body, and are so subtle that they are only felt when attention is focused on them. Background emotions are the consequence of deploying certain combinations of simple regulatory reactions (e.g., basic homeostatic processes, pain and pleasure behaviors, and appetites). Background emotions can be distinguished from moods, which refer to the sustaining of a given emotion over long periods of times, lasting hours or days.

Primary (or basic) emotions are easier to define because the most common emotions are in this group. The frequent list includes fear, anger, disgust, surprise, sadness and happiness. There are good reasons for this centrality. These emotions are universal across cultures and in non-human species as well. The circumstances that cause the emotions and patterns of behavior that define them are also quite consistent across cultures and species.

The social emotions include sympathy, embarrassment, shame, guilt, pride, jealousy, envy, gratitude, admiration, indignation, and contempt. A whole retinue of regulatory reactions along with elements present in primary emotions can be identified as subcomponents of social emotions in varied combinations. The nested incorporation of components from lower tiers is apparent. Think of how the social emotion "contempt" borrows facial expressions of "disgust," a primary emotion that evolved in association with the automatic and beneficial rejection of potentially toxic food.

In some instances, emotional responses are innate; in others they may require help from an appropriate exposure to the environment.

Emotions guide organisms' behaviors by activating or suppressing certain brain circuits and produce automatic reactions. Organisms can achieve a positive result without feeling an emotion or even deciding to produce the reactions that lead to the positive result. These automatic emotions create conditions in the human organism that, once mapped in the nervous system, can be represented as pleasurable or painful and eventually known as feelings. We humans, conscious of the relation between certain objectives and certain emotions, can willfully strive to control our emotions, to some extent.

As we develop as humans, most objects that surround us can trigger some form of emotion or another, weak or strong, good or bad, and can do so whether we are

147 Antonio Damasio, *The Feeling of What Happens*.

148 Ibid.

conscious of the emotion or not. Some of these triggers are set genetically by evolution, but some are associated in our brains with emotionally competent objects by virtue of our experience.[149]

An emotionally competent stimulus is one that produces automatically an emotional reaction. The process starts with an appraisal-evaluation phase, initiated by the detection of an emotionally competent stimulus (the ECS).

In neural terms, images related to the emotionally competent object must be represented in one or more of the brain's sensory memory systems, such as the visual or auditory regions. This image is made available to emotion-triggering sites in the form of an echo signal with a tag.

The thalamus, as it continuously receives the echoing signals from the cortex, tries to match the incoming sensory signal to any one echo that might match an ECS. If a match is found, it will activate a number of emotion-execution sites elsewhere in the brain. These sites cause the immediate changes that occur in the body and the brain regions that support the emotion-feeling process. The process can reverberate and amplify itself, or shrivel and close down.

In the language of neuroanatomy and neurophysiology, this process begins when neural signals of a certain configuration that are holding patterns corresponding to the threatening object originate in the visual cortices; these match the echoes and handshakes that help signal that the object is threatening; they are relayed in parallel along several pathways to several brain structures. When a match is found (by the thalamus between stimulus and echo), the amygdala will be activated and send signals to other brain regions, thus producing the cascade of events that will become an emotion, in this case, fear.

The amygdala is an important interface between visual and auditory ECS and the triggering of emotions, in particular, but not exclusively, fear and anger. When the amygdala is damaged, fear and anger are not possible. Activation of the amygdala shows a linear relationship with decreasing intensity of happiness and increasing intensity of fear.

Emotionally competent stimuli are detected very quickly, ahead of selective attention. When parts of the visual cortex are damaged in a way that cause a blind field of vision (e.g., certain categories of objects can't be seen), an emotionally competent object (e.g., a happy face) nevertheless breaks through the blindness barrier and is indeed detected. The triggering emotional machinery captures these stimuli as they bypass the normal channels—channels that might have led to cognitive appraisal but simply could not do so because of the blindness. This is accomplished by setting up an echo signal with a tag that, when matched in the future, will automatically trigger an emotional response even before the stimulus is processed cognitively.

The ventromedial frontal area and especially the orbital cortex are tuned to detecting the emotional significance of more complex stimuli, for example objects and

[149] Ibid.

situations, natural and learned. Social emotions are triggered by complex and specific patterns of handshakes that when presented together are recognized as emotional competent stimuli through genetic and learned responses from our parents in our childhood. Once these conditions are met, the emotional competent stimulus will trigger a signal by the ventromedial frontal area to the thalamus, and the appropriate chemical and neural responses will be set in motion to produce the suitable emotion with all the body and brain changes that this implies.

Facial expressions, vocalizations, body postures, and specific patterns of behavior (e.g., running, freezing, parenting) are thus enacted. The body chemistries as well as organs, such as heart and lungs, assist. Emotion is all about transition and commotion, sometimes real body upheaval. In a parallel set of commands the brain structures that support image-production and attention change as well. Some areas of the cortex become less active while others become especially active.[150]

Emotions help to activate certain memories to initiate adequate behaviors in response to particular stimuli. They manage this in two ways: (1) they intrude on the attentional systems and (2) they initiate certain behaviors (motivations) like fleeing or moving towards the stimulus. Motivation is used here as a term that refers to neural activity that guides us to a goal, outcomes that are desirable and for which we will exert effort, or the opposite, one that we dread and will exert effort to prevent, escape from, or avoid.

The system used to defend against danger is different from the system used for procreation. The feelings that result from the emotions generated, fear and sexual pleasure, do not have a common origin. There is no such thing as the "emotion generator" and there is no single brain system dedicated to create all emotions. Perhaps, then, we need to focus on classes of emotions. Brain regions have functions because they are part of integrated systems. Mental functions involve many different regions working together; each function requires a unique set of interconnected regions, forming its own system. Many regions might be used by different systems.

The information from the senses, as we have seen, passes through the thalamic relays on its way to its specialized cortical areas. These thalamic regions are specialized in matching the echoes of the cortex with the inputs from the senses and then reflecting those signals; ear signals are sent to the auditory cortex, skin sensations are routed to the somatosensory cortex, and the eye signals are relayed to the visual cortex.[151]

When appropriate (a signal matches a certain echo), the thalamus signals the amygdala. The amygdala will also receive signals form other areas of the cortex. In this way the various signals fine-tune the amygdala's responses.

The signals that reach the ear are transmitted to the auditory brainstem nucleus (cochlear nucleus), and then cross to the opposite side to the inferior colliculus of

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[150] Ibid.

[151] Joseph LeDoux, *The Emotional Brain.*

the midbrain. Axons then travel to the auditory thalamic relay nucleus, the medial geniculate. From there the signal is relayed to the amygdala if the thalamus determines it to be a danger signal (because of the match between stimulus and the echoes with tags arriving from the cortex). The signal is also reflected (relayed) to the auditory cortex (because of the echo), which in turn is made up of several regions and sub regions.

The amygdala—through the central nucleus—connects to the brain stem areas that are involved in the control of the heart rate and other autonomic nervous systems. The stimulation of the central nucleus elicits freezing responses. Lesions to the central nucleus interfere with every measure of conditioned fear: freezing behavior, autonomic responses, suppression of pain, stress hormone release, and reflex potentiation. Because of the matching to a danger echo, the auditory stimulus also travels from the thalamus to the lateral nucleus of the amygdala. The lateral nucleus seems to receive the conditioned response signals; the central nucleus, together with the hypothalamus controls the activation of the response control systems.

Neurons that project from the thalamus to the primary auditory cortex are narrowly tuned—they are very particular about what they will respond to. Cells that project to the amygdala respond to a much wider range of stimuli and are said to be broadly tuned. When two similar sounds are used in a conditioning response, the thalamus will send the amygdala essentially the same information, regardless of which stimulus it is processing (the echoes are a close enough match), but after the cortex processes the different stimuli, it will send the amygdala different signals. The amygdala receives the danger signal at the same time that the auditory cortex does. The auditory cortex interprets the signal for any emotional content and then signals the amygdala.

Although the thalamic system cannot make fine distinctions, it has an important advantage over the cortical input pathway to the amygdala: time. It takes about twelve milliseconds for an acoustic signal to reach the amygdala through the thalamic pathway and twice as long through the cortical pathway. The thalamic pathway can't tell the amygdala what is there, but can provide a fast signal that warns that something dangerous may be there; it is quick and dirty.[152]

Imagine walking through the forest. A crackling sound occurs; it goes straight to the amygdala through the thalamic pathway. The sound also goes from the thalamus to the auditory cortex, which recognizes the sound to be a dry twig that snapped under the weight of your boot or a rattlesnake shaking its tail. By the time the prefrontal cortex processes the difference, the amygdala already has started a fear response to the potential rattlesnake. The information from the thalamus is unfiltered and biased to provoking a reaction. The prefrontal cortex's job is to prevent the inappropriate response rather than to produce the appropriate one.

Alternatively, suppose there is a slender, curved shape on the path; the curvature and slenderness reach the amygdala through the thalamus that received it from the

[152] Ibid.

eye. If it is a snake, the amygdala is already ahead of the game. The fight-flight response is automatically activated. If it weren't a snake, the prefrontal cortex would then send a signal to the amygdala to stop the response, in essence confirming a false alarm. The cost of treating a stick as a snake is less, in the long run, than the cost of treating a snake as a stick.[153]

It has now been established that the amygdala and the prefrontal cortex are reciprocally related: in order for the amygdala to respond to fear reactions, the prefrontal region has to be shut down. Pathological fear may occur when the amygdala is unchecked by the prefrontal cortex. Clearly, decision making in emotional situations is impaired when there is damage to the medial and ventral prefrontal cortex. Possible therapies might involve methods to increase activity in the prefrontal areas so that the amygdala is less free to express fear.

The medial prefrontal cortex may serve as an interface between cognitive and emotional systems, allowing cognitive information to regulate emotional responses. In return, emotional processing in the amygdala might influence decision-making (by the types of handshakes sent out) and other cognitive functions (how the signals are interpreted) of the prefrontal cortex.

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This is a good example of how the brain retrieves a memory, remembers, according to, first, the emotion and second, by context. The emotion is fear, the context is freefall:

I was up on an eighteen-foot ladder, almost two stories high, installing a water tank for a solar heater. I needed to drill a few screws into the wall to fix a metal support for the tank. As I exerted pressure on the drill, my feet exerted pressure on the ladder, which stood on a mossy patio. As a result, the ladder's feet started to slip. Immediately, adrenaline, as part of the fear response, started rushing through my body. My thoughts speeded up, and the fear was immediately placed into the context of falling. As the ladder slipped, seemingly in slow motion, I quickly concluded that I could not afford to stay on the falling ladder, as surely I would break both legs when they got tangled in the ladder's rungs. Once that decision was made, I jumped off the ladder, to face a slightly different situation—freefall from seventeen feet up. I thought about what kind of fractures I would sustain as I hit the cement floor. But as I fell, in mid air, I clearly remembered when I was eight years old and practiced rolling onto our lawn as I jumped many times from a twelve-foot wall. My course of action crystallized as I concentrated on rolling the instant that I touched the ground to brake my fall. When I made contact with the ground, I rolled, feet to knees, right arm to shoulder, tucked my head under, curled, on my back, then buttocks, feet forward . . . and got up without a scratch.

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153 Ibid.

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The amygdala by way of its connections with the hippocampus and other regions of the explicit memory system strengthens the relation between emotions and the event. Later, an emotion will more easily activate the related memories. On the other hand, the connections from the hippocampus, perhaps, help recreate the emotion that is related to a memory.[154] The hippocampus places the emotion into a context by activating the related objects that made up similar events.

Drives, Needs and Beliefs

Drives are defined as internal urges to maintain homeostatic balance. These are qualitatively different than emotions. Yet, both emotions and drives promote responses or actions of some form or another.

When levels of various substances in the blood go beyond specific limits, various neuropeptides are automatically released, and are part of the mechanisms that activate particular neural circuits. For example, it has long been known that angiotensin, a hormone that is also a peptide, mediates thirst. Chemically speaking, angiotensin translates as an altered state that makes humans and animals want to drink water.[155]

When an interaction with the environment is necessary to restore homeostasis a drive is triggered to produce the appropriate response. The organism then responds in a series of automatic, genetically set behaviors. Drives are triggered by internal changes, emotions are triggered by external circumstances, but both are means to guide behavior.

Emotions are more about avoiding threats and taking advantage of opportunities in the environment. Behaviors generated emotionally have greater range and flexibility because of the added memory systems activated by each emotion. However, the activation of behaviors through needs is similar to emotional responses, even if the triggering mechanisms are slightly different. Because of this, it is worthwhile to see in detail some of the neural circuits involved in generating behavioral responses to both internal and external stimuli.

Some of these genetic-set behaviors or needs have been suggested by various authors such as; affiliation (to draw near and cooperate with an allied other), aggression, autonomy (to break free or shake off restraint), defendance (to defend against assault), harmavoidance, fleeing, eating, drinking, and suckling to name a few. In humans, of course, many more needs have been proposed, such as abasement, achievement, counteraction, deference, dominance, nurturance, play, shame avoidance and understanding among others.

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154 Ibid.

155 Candace Pert, *Molecules of Emotion,* 1997.

As an example of a genetically set response, Thompson's gazelles, known as tommies, which are prayed upon by many carnivores, present a complex behavior when fleeing. When a tommy sees something suspicious it becomes obviously alert; its head is held high, the ears pointed forward, muscles tense, its eyes locked on whatever attracted its attention. The tommy then stomps the ground with a foreleg, and emits a soft snort known as a "quiff." Quiffs seem to occur when something has alerted the tommy, and they cause others to assume the alert posture and look in the same direction. When a serious attack occurs the tummies flee at a gallop of speeds of 45-60 km per hour, which is faster than any of their predators can run except for a very short distance in the initial charge. Tommies also run in other ways: they stot, jumping a meter or so in the air, holding their legs relatively stiff. Stotting usually occurs at the start of the chase, but only when the pursuer is not to close, and at the end when the predator has given up. But when closely pressed the tommies always gallop. The type of gait also varies according to the type of predator; stotting is common when tommies are chased by hyenas or wild dogs, but is employed rarely with lions, cheetahs, and leopards, which achieve higher speeds in the initial charge. When the whole herd is being chased, most or all of them bounce around in an irregular fashion, which seems to confuse many predators and hinder their concentration on a single gazelle, so more often than not, they all escape.[156]

In this case, the tommies must be able to identify the organisms around them. First, separating predators from other herbivores, and then, separating hyenas or dogs from felines, which can attain much higher speeds in the initial charge; cheetahs being able to develop speeds of up to 110 km per hour. Different fleeing strategies are used under different conditions, depending on the predator and the distance between them. This requires memory systems that can identify, if not individuals, at least categories.

A range of compensatory responses is engaged when water depletion of either the intra- or extracellular compartment occurs. These responses (e.g., vasopressin secretion, stimulation of the renin-angiotensin-aldosterone system, sympathetic activation, and reduced renal solute and water excretion) have the effect of minimizing changes in body fluid volume and composition. However, such mechanisms, although of undoubted benefit to the animal, do not restore body fluids to the original state. For this to occur, water losses must be replenished. Therefore, thirst, which provides the drive or urge to drink, is an important component of the coordinated sequence of physiological responses that maintain the volume and composition of body fluids. Finding water and drinking is the needed behavioral response.

Recent studies using positron emission tomography in human volunteers identified several brain regions that became activated during an intravenous infusion of hypertonic saline that produced a strong thirst sensation in these subjects. In particular, the anterior and posterior parts of the cingulate cortex were activated, and on satiation

[156] Donald Griffin, *Animal Minds*.

of the thirst, these areas rapidly declined in activity.[157] These cingulate regions (which have been implicated in other goal-directed behaviors) probably play a role in the modulation or definition of urges.

The entorhinal area in the guinea pig projects directly to nonhippocampal regions: anterior cingulate cortex area 24 and the infralimbic cortex-area 25), retrosplenial cortex (area 29), cingulate cortex (areas 23), prelimbic cortex (area 32), perirhinal cortex (areas 35, 36), prepyriform cortex (area 51B), and insular cortex (areas 13-16). The entorhinal area also projects to subcortical targets: distinct terminations were observed in the lateral thalamic nucleus, the striatum, and the accumbens nucleus. The cells giving rise to the projections to the hippocampus were found to lie in layers II and III, those projecting to the nonhippocampal cortical regions originate in layer IV, and those projecting to the striatum and the accumbens to lie in layers V and VI. Many of the efferent projections to the cerebral cortical regions are associated with reciprocal projections from these regions to the superficial layers (I-III) of the entorhinal cortex. The entorhinal efferent projections generally terminate ipsilaterally. A weak termination is, however, present at the contralateral side. The efferent projections of the entorhinal area represent a route for important caudally directed, nonfornical hippocampal output.[158]

From a hierarchical/cytoarchitectural point of view, the nonhippocampal regions, primarily the cingulate cortex, are receiving inputs similar to the entorhinal cortex (from layer IV), whereas the hippocampus is one level higher, receiving input from the processing layers (II and III). The striatum and the nucleus accumbens are one level lower (layers V and VI) in the hierarchy, and through the reciprocal connections are feeding back to layer I, probably reinforcing or modulating the output of the hippocampus.

The periaqueductal gray receives afferents from the basal nucleus of the amygdala and the (GABAergic and cholinergic) medial septal nucleus, as well as efferents from the reticular formation. The periaqueductal gray projects to the serotonin-containing neurons of the raphe nuclei. The midbrain, through widespread diffuse projections, can activate large cortical areas. This cytoarchitecture suggests urges are initiated in the midbrain, and signals from other brain structures dampen, strengthen or modulate the urges.

Many areas in the midbrain receive numerous afferents from many cortical, subcortical and ascending pathways from the body, and are instrumental in regulating many of the main neurotransmitter systems: cholinergic, dopaminergic, noradrenergic and serotonergic. There are various circular (circular in the sense that if you move around the circuit, eventually you will get back to the starting point) circuits in the brain. It is helpful to think of nerve impulses being sent in a clockwise or counterclockwise direction, and

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[157] Michael J. McKinley and Alan Kim Johnson, *The Physiological Regulation of Thirst and Fluid Intake.* News in Physiological Services, Vol. 19, No. 1, 1-6, February 2004.

[158] Sorensen K.E., *Projections of the entorhinal area to the striatum, nucleus accumbens, and the cerebral cortex in the guinea pig.* J Comp Neurol., 1985 Aug 15;238(3):308-22.

not only from a higher to a lower level in the hierarchy. However, the hippocampus is at the highest level, hierarchically, in this circular pathway, whether the signals are moving clockwise or counterclockwise. In neurological terms, there exist efferent projections from the hippocampus in either direction. This system is primarily dopaminergic.

The substantia nigra and the ventral tegmental area project dopaminergically to the striatum and the frontal lobes, as well as the amygdala, the lateral septum and the nucleus accumbens.[159] Dopamine strongly influences brain circuits engaged in organizing behavior and planning movements. There are five types of dopamine receptors, which complicate matters: some are excitatory and some inhibitory.[160] We have to look closely at different inputs into the nucleus accumbens when we consider drives, needs and beliefs. Essentially, urges are initiated by various body (chemical) signals to the midbrain. The ventral tegmental area sends signals to the nucleus accumbens. Feedback circuits from the basal ganglia, the prefrontal cortex, and the entorhinal cortex regulate these. The signals to the nucleus accumbens are further modified by inputs from the hippocampus and the amygdala. In the following paragraphs we can see in detail how these circuits work.

The orbital frontal cortex, adaptively modulates lower structures, inhibits drives and regulates arousal. Alexander Luria showed that disturbances of the orbital frontal regions elicit changes in affective process, such as, lack of self-control, emotional outburst, generalized disinhibition, and disorganization of personality. This was the rationality behind lobotomies. Patients that underwent a lobotomy lacked a depth of personality and presented a lack of ability to follow "instinctual" drives or to maintain a fully affective "consciousness" of self. It was also observed that these patients showed impairment of internalization and symbolic elaboration.[161]

The nucleus accumbens is 95% composed of medium spiny GABAergic (inhibitory) projection neurons, which are part of the main output of this nucleus. Most of the rest of the interneurons are cholinergic (excitatory). The output neurons of the nucleus accumbens send axon projections to the ventral pallidum, which are GABAergic. Two serial GABAergic projections become excitatory (i.e, (-) (-)=+). The ventral pallidum, in turn, projects to the thalamus, which projects to the prefrontal cortex (glutamate and aspartate-excitatory impulses).[162] Major inputs to the nucleus accumbens include the prefrontal cortex, amygdala, hippocampus, and dopaminergic neurons located in the ventral tegmental area in the midbrain, which connect via the mesolimbic pathway. Thus the nucleus accumbens is often described as one part of a cortico-striato-thalamo-cortical loop.

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159 J. Thomas Curtis, Zuoxin Wang, *Ventral tegmental area involvement in pair bonding in male prairie voles*. 2005.

160 John H. Martin, *Neuroanatomy*.

161 A.N. Shore, *Affect Regulation and the Repair of the Self*. 2003.

162 John H. Martin, *Neuroanatomy*.

Stimulation of the nucleus accumbens evokes a potent inhibition in neurons of the ventral tegmental area. GABA is likely to act as a transmitter in this descending inhibitory system.[163] Whenever the ventral tegmental area sends dopaminergic impulses to the nucleus accumbens, it automatically acknowledges by sending a strong inhibitory (GABA) feedback signal. The dopaminergic signals to the prefrontal cortex are excitatory (D1) in nature, and the feedback signals to the ventral tegmental are glutamatergic to the GABA interneurons, also producing inhibitory action. The prefrontal acetylcholine signals to the nucleus accumbens are also excitatory (nicotinic receptors), which also would send GABAergic inhibitory signals to the ventral tegmental area. Thus every time the ventral tegmental area releases dopamine, strong automatic feedback signals quickly dampen this activity.

This convergence of signals in the nucleus accumbens defines the threshold for action, as well as the type of response. These neural connections suggest that the nucleus accumbens is instrumental in initiating actions. The midbrain sends a signal to the nucleus accumbens, which is modified by other inputs to determine the nature of the action: the amygdala sends an emotional component (a preference or aversion for the space), the limbic areas provide an emotional memory of the space, and the hippocampus sends a contextual and spatial component that regulates the prefrontal cortex inputs. The prefrontal cortex evaluates the stimulus and signals the nucleus accumbens accordingly. The activity in the hippocampus determines when and if signals from the prefrontal cortex have an effect on nucleus accumbens groups of neurons.

The signals of the midbrain in turn are also modified by signals from the prefrontal cortex as well as the output nuclei of the basal ganglia, thus closing several feedback loops. The increased memory systems provided by the cortex, in this case the prefrontal cortex, allow for, not only an increased set of responses, but greater flexibility of each response. More importantly, evaluation of the stimulus by the prefrontal cortex can change automatic responses completely, but only within the context provided by the hippocampus.

The thalamus reflects (relays) the excitatory signals (glutamate and aspartate) from the basal ganglia outputs. The areas in the cortex where these signals were relayed will determine how this need is perceived, and consequently will define the type of need. Different cortical areas will interpret specific needs.

The orbital frontal cortex and the anterior cingulate gyrus are wired directly into the gut control centers of the brain, which serve to cause a strong visceral sensation that something is wrong,[164] and reinforces the urge to begin some action. Given the extensive inputs to the orbito-frontal cortex, as well as its extensive projections to structures related to emotional areas and subcortical drive centers in the hypothalamus and arousal and

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[163] P. Wolf, H.R. Olpe, D. Avrith and H.L. Haas, *GABAergic inhibition of neurons in the ventral tegmental area.* Cellular and Molecular Life Sciences. Vol 34., No. 1, January 1978.

[164] Jeffrey Schwartz, *The Mind and the Brain.*

reward centers in the midbrain, it is one of the cortical areas receiving information from almost the entire brain and influencing the output of many of its circuits.

Echoes become established (in the cortex) in memory to represent past experiences, and an integral part of the echo is the associated emotion. Memories with an associated emotion become a representation of past reality. An idea or an imagined event, even though rooted in past experience, lacks this emotional association. This lack of emotional association will be rightly perceived by the brain as separate from reality or the past, and will be interpreted simply as an idea or imagined event. When the thalamus matches echoes associated with an emotion, it triggers the associated emotion by sending signals to the amygdala. The amygdala serves as a memory of emotions through its reciprocal cortical connections and as such modulates the associated emotion of the particular echo.

However, when a concept becomes bonded to an emotional state, or when semantic memories become associated with an emotion, they become a belief and are perceived as fact. In this way, beliefs are also represented by echoes, which are processed by the brain as true past experiences, precisely, because they have an associated emotion. Most beliefs are established early in life and serve as shortcuts to produce particular behaviors by triggering particular emotions. The beliefs take the place of experience; beliefs are an elegant engineering solution to trigger an emotion and guide responses without the benefit of experience; on the negative side, beliefs have less behavioral response flexibility. Beliefs are an evolutionary development to pass on experience through emotional transfer. However, there is only a short window of opportunity (species specific) to pass experience as belief through emotional transfers to the next generation.

Beliefs can be seen as an evolutionary transition to guide responses somewhere between drives and secondary emotions. Beliefs guide behavioral responses inflexibly, as do primary emotions such as fear or anger. Beliefs are dogmatic, but have an emotional component to them. The emotional component is a form of altered brain chemistry, equivalent to an emotional state, which activates the belief (or single memory) that guides the response. With a limited memory system, a single memory or belief can be stored and activated by the associated emotion. Later on, evolutionarily speaking, with larger memory systems (larger cortex), the emotion activates all related memories, instead of a single one. These memories then serve to guide behavior in a flexible manner, but within the boundaries of experience.

When ingesting cannabinoids, THC or opiates, they quickly concentrate in the ventral tegmental area, the nucleus accumbens, the hippocampus, the cerebellum, and in small cortical areas widely distributed throughout the frontal and parietal lobes and the cingulate cortex. The action of THC is most notable in the nucleus accumbens, as there is an increase of neural impulses. Cocaine accumulates, similarly in the ventral tegmental areas, the nucleus accumbens and the caudate nucleus. [165]

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[165] *The Brain and the Actions of Cocaine, Opiates and Marijuana,* National Institute on Drug Abuse.

Cocaine and alcohol change the accumbens' activity as dopamine release is increased and serotonin is inhibited. Serotonin has to do with satiety. The action of dopamine in this circuit reinforces behaviors or desires by sending dopaminic projections to the prefrontal cortex (via the mediodorsal nucleus of the thalamus). When urges or desires are satisfied, more dopamine is released, producing a positive emotional feeling. This is why many call it, the reward circuit. The first dopaminergic input into the nucleus accumbens comes from the ventral tegmental area, which produces an undefined urge. As a result of this, the activity in the caudate nucleus is perceived by the brain as the urge. The exact nature of the urge is modulated by other inputs. The hippocampus' projections to the nucleus accumbens have to do with guiding behavior contextually in response to needs or desires produced by internal stimuli (in rats, preferring certain places to satisfy the need, the context is preferred locations that satisfy certain urges), whereas the amygdala's projections (because of their emotional component) create or reinforce beliefs that guide responses. The altered chemistry of the emotion is what activates the one and only memory, the belief. The belief—a concept attached to an emotional component—serves to guide the response. The prefrontal cortex evaluates the brain's activity in relation to how positive or negative a situation is and also affects the dopaminergic signals to the nucleus accumbens through its projections to this area.

Ultimately, it is the caudate nucleus that initiates actions and responses with its TAN neurons firing simultaneously in particular patterns, which are precisely determined by the combinations of these three inputs into the nucleus accumbens. The output of the caudate nucleus, through the so called direct and indirect path, to the thalamus projects to the prefrontal motor regions, which initiate the necessary movements. The convergent, triple dopaminergic signals to the nucleus accumbens along with an increasing or decreasing serotonergic input determine the caudate nucleus' output. This is the archipraxi switch (from the Greek archizo=begin and praxi=action). This switch initiates not only action, but determines the type of action as well. In general, a motor action is enacted, but also, according to certain emotions, the associated beliefs or memories are triggered, which in turn serve to further guide the behavioral response. Needless to say, all this motor action needs to be embedded within the integrated external-internal space created by the basal ganglia.

On the evolutionary scale, the archipraxi switch goes from automatic, genetically triggered behaviors in response to set stimuli, to learned responses set in memory as beliefs (some call this instincts), to more flexible responses triggered by more variable stimuli in proportion to an increasing cortex that accommodates larger memory systems. For this to happen, a spatial element (provided by vision), a body-control element (provided by the proprioceptive signals), a spatial-contextual signal (provided by the hippocampus) and an evaluation, or emotional signal (of whether it is "good" or "bad," or positive or negative, provided by the amygdala) have to converge to produce the adequate set of responses.

It now becomes obvious that the greater the memory systems in the cortex (i.e., a greater cortex), the greater the types of behaviors that can be enacted as well as a

greater flexibility of each behavior. Different components of a memory—its context or its associated emotion—modify the interpretation of the drive, and as a consequence produce slightly different behavioral response based on particular needs, beliefs, or previous experiences. Signals from the hippocampus modulate the response according to context or circumstance, and the signals from the amygdala activate a belief. The two combined are perceived as an unbearable urge to act in a particular fashion. The signals from the frontal lobes amplify or dampen the urge. Paradoxically, it is possible that the brain, under certain conditions, can construct a belief contrary to a need, i.e., the urge to have sex with the belief that sex is wrong.

The Regulation of Emotions

Emotions are a direct expression of bio-regulation. Some of the structures or regions in the brain identified as emotion-triggering sites are the amygdala, the hypothalamus, the thalamus and in the cortex a part of the frontal lobe known as the ventromedial prefrontal cortex and the supplementary motor area, and the cingulate among others.[166]

The amygdala is an important interface between visual and auditory ECS and the triggering of emotions, in particular, but not exclusively, fear and anger. When the amygdala is damaged, fear and anger are not possible. Activation of the amygdala shows a linear relationship with decreasing intensity of happiness and increasing intensity of fear.

Emotionally competent stimuli are detected very quickly, ahead of selective attention. This is accomplished by setting up an echo signal with a tag that, when a stimulus is matched by the thalamus, will automatically trigger an emotional response even before the stimulus is processed cognitively.

Social emotions are triggered by complex and specific patterns of handshakes from the sensory and other cortex areas that, when presented together, are recognized by the frontal lobes as emotional competent stimuli (ECS) through genetic and learned responses in our childhood. When the echo signals of the orbital and prefrontal cortex representing the complicated patterns of the ECS is matched by the thalamus and the caudate nucleus, the appropriate social emotion is automatically triggered.

Social emotions differ slightly from primary emotions. In primary emotions, the thalamus detects an exact match between incoming sensory signals and the echoes from the cortex representing the primary ECS and the emotion is triggered automatically. In social emotions, the ventromedial and orbital frontal areas detect complicated patterns of handshakes representing the social ECS. Depending on the handshakes' qualities, echoes are sent to the limbic system and the thalamus and automatically signals are sent to initiate the appropriate chemical and neural responses without further matching with sensory signals, which trigger the proper social emotion.

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[166] Ibid.

The brain attempts to maintain at all times a one-to-one internal emotional landscape with respect to the external conditions in order to produce the best response. This depends on the brain creating and maintaining a dynamic model of the external environment and generating, moment by moment, a new internal emotional representation in response.

Neurons in the ventromedial prefrontal region respond rapidly and differently to the pleasant or unpleasant emotional content of pictures. Unpleasant emotions activate the right side more.[167] This suggests a certain asymmetry in which the right side is more involved with negative emotions than the left. More likely, more activity on the right side, relative to the left, is perceived as a negative emotion.

The hippocampus is connected to the amygdala and provides it with information about the context of the present emotion. The amygdala sends signals to the hypothalamus, midbrain, pons and medulla, and thus helps control the emotional reactions according to the context. The amygdala also projects to the periaqueductal gray matter and, in this way the extent of an emotional reaction (gestures and motor actions) can be carefully adjusted depending on the specifics of a particular situation.

The emotion-execution sites include the hypothalamus, the basal forebrain and some nuclei in the brain stem tegmentum. The hypothalamus is the master executor of many chemical responses that are part of our emotions. Directly or via the pituitary gland it releases into the bloodstream chemical molecules that alter the internal milieu, the function of viscera, and the function of the nervous system. Oxytocin and vasopressin, both peptides, are examples of molecules released under the control of the hypothalamic nucleus with the help of the posterior pituitary gland.[168]

Echoes become established to represent past experiences, and an integral part of the echo is the associated emotion (we will come back to this point in the chapters dealing with emotions). Memories with an associative emotion become a representation of past reality. An idea or an imagined event, even though rooted in past experience, lacks this emotional association. This lack of emotional association will be rightly perceived by the brain as separate from reality, and will be interpreted simply as an idea or imagined event. When the thalamus matches echoes associated with an emotion, it triggers the associated emotion by sending signals to the amygdala. The amygdala serves as a memory of emotions and as such triggers the associated emotion of the particular echo. However, when a concept becomes bonded to an emotional state, it becomes a belief. In this way, beliefs are also represented by echoes, which are processed by the brain as true past experience, precisely, because they have an associated emotion. Most beliefs are established early in life and serve as shortcuts to produce particular behaviors by triggering particular emotions. The beliefs serve to take the place of experience; they are quick and dirty shortcuts to trigger an emotion and guide responses.

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[167] Jeffrey M. Schwartz, *The Mind and the Brain.*

[168] Ibid.

The nucleus accumbens is 95% composed of medium spiny GABAergic (inhibitory) projection neurons, which are part of the main output of this nucleus. Most of the rest of the interneurons are cholinergic (excitatory). The output neurons of the nucleus accumbens send axon projections to the ventral pallidum (VP), which are GABAergic. Two serial GABAergic projections become excitatory. The VP, in turn, projects to the mediodorsal (MD) nucleus of the thalamus, which projects to the prefrontal cortex (glutamate and aspartate-excitatory).[169] Major inputs to the nucleus accumbens include the prefrontal cortex, amygdala, hippocampus, and dopaminergic neurons located in the ventral tegmental area (VTA) in the midbrain, which connect via the mesolimbic pathway. Thus the nucleus accumbens is often described as one part of a cortico-striato-thalamo-cortical loop.

The nucleus accumbens is regarded as the emotion-motor interface, in view of its limbic afferent and somatomotor and autonomic efferent connections. Within the accumbens, there appear to be specific areas in which limbic afferent fibres, derived from the hippocampus and the basolateral amygdala, overlap. These afferent inputs have been suggested to converge monosynaptically on cells within the accumbens and are hypothesized to play a role in paradigms such as conditioned place preference (in rats).[170]

Cocaine and alcohol change the accumbens' activity as dopamine release is increased and serotonin is inhibited. Serotonin has to with satiety. The action of dopamine in this circuit reinforces behaviors or desires by sending dopaminic projections to the prefrontal cortex (via de MD). When urges or desires are satisfied, more dopamine is released, producing a positive emotional feeling. This is why it is called, by many, the reward circuit. The hippocampus' projections to the nucleus accumbens have to do with creating (or defining) needs or desires (in rats, preferring certain places), whereas the amygdala's projections (because of their emotional component) create or reinforce beliefs that guide responses.

This is a good example of how different components of a memory—its context or its associated emotion—modify the interpretation of the memory, and as a consequence produce a different behavioral response based on a need or on a belief. Signals from the hippocampus produce a need (or aversion, depending on whether the context is positive or negative), and the signals from the amygdala produce a belief. The two combined become an unbearable urge to act. However, it is possible that the brain, under certain conditions, can construct a belief contrary to a need or vice-versa, i.e., the urge to have sex with the belief that sex is wrong.

Many emotional behaviors (like attachment and nurturing) depend on the timely availability of these hormones within the brain structures that command the execution

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169 John H. Martin, *Neuroanatomy.*

170 French, SJ, Totterdell, S, *Individual nucleus accumbens-projection neurons receive both basolateral amygdala and ventral subicular afferents in rats.* Neuroscience. 2003;119(1):19-13.

of these behaviors. It is crucial to have the right responses at the right times. Attempting to have sex with a predator could be lethal under the best of conditions.

Likewise, the local availability of molecules, which modulate neural activity, like dopamine and serotonin, causes certain behaviors to occur. The sort of behaviors experienced as rewarding or pleasurable appear to depend on the release of dopamine from one particular area (the ventrotegmental area in the brain stem) and its availability in yet another area (the nucleus accumbens in the basal forebrain).

In short, various brain nuclei that control the movement of the face, tongue, pharynx and larynx are the ultimate executors of many behaviors, simple and complex, that define emotions, from gestures and body postures, to courting or fleeing, to laughing or crying and fighting. It makes sense that the controlled movements of vocalizations (screaming or shouting), or facial gestures (laughing or smiling), or body postures (leaning forwards or slumping) are intimately linked to communicating the different emotional responses. Facial expressions, vocalizations, body postures and specific patterns of behavior (e.g., running, freezing, parenting and so on) are thus enacted. Emotion is all about transition and commotion, sometimes real body upheaval. In a parallel set of commands the brain structures that support image-production and attention change as well. Depending on the chemistry of each emotion, some areas of the cortex become less active while others become particularly active.

Emotions elicit particular memories to initiate certain behaviors in response to specific stimuli. They manage this in three ways: (1) the chemistry of the emotional state activates all memories related to the emotion, as this chemistry is specific to the memories; (2) they intrude on the attentional systems; and (3) they initiate certain behaviors (motivations or modes of thinking) like fleeing or approaching the stimulus. Motivation is used here as a term that refers to neural activity that guides us to a goal, outcomes that are desirable and for which we will exert effort, or to the opposite, an outcome that we dread and will exert effort to prevent, escape from or avoid.

Goals direct action. They can be a specific stimulus (food) or an abstract belief or idea (it is worth dying defending freedom). Some of our beliefs are obtained early in childhood by conditioning (families should or should not stay together) or by observational learning (we learn to parent as young children and we learn to hunt by playing games) or even by force of imagination (God is on my side).

Not all motivations are induced by emotions, but emotions are powerful motivators to action. Motivations can be modified by past experience. The purpose of these beliefs is to push the brain into a state that more likely will produce an instrumental response.

In the presence of an emotionally arousing stimulus, the brain is placed in a motive state, which leads to coordinated information processing within and across areas, and results in invigoration and guidance of behavior towards positive goals and away from aversive ones. The motor regions are strongly activated and movement is initiated. Behavior can be potentially invigorated by anything that activates certain cells and

causes them to release dopamine. Novel stimuli are a prime example of invigorating stimuli. But invigoration alone is not enough; behavior also needs to be guided. The amygdala, when it detects a positive outcome increases the dopamine release and reinforces the signal.

Once an emotional habit is well learned, the brain system involved in expressing it becomes simpler. Once learned, the procedure might be transferred to the cortex and stored as an echo. The echo, then, can trigger that emotion. With an expanded cortex, more habits can be stored and enacted. This is similar to explicit learning: initially, both the hippocampus and cortex are involved, but once the hippocampus has slowly "taught" the cortex the memory, the memory persists without the aid of the hippocampus. The hippocampus may well be involved in the guidance of behavior on the basis of spatial and other kinds of relational cues in the environment. In order to find good things and avoid bad ones, you need to know where you are, where you need to go, and how to get there from here, as well as activating all the memories that might be useful to guide you on the way. The hippocampus contextualizes emotions, and if the context is appropriate, reinforces the emotion; conversely if the context is inappropriate, the hippocampus defuses the emotion.

Goal directed behavior is best thought of in functional terms. Normally we try to escape or avoid harmful stimuli, but in some cases, we have to actively engage something dangerous in order to achieve protection. Hunting is a good example. Sometimes, fighting is a better alternative to fleeing or the only alternative. Decision-making compresses trial and error learning experiences into an instantaneous mental evaluation about what the consequence of a particular action will be for a given situation. It requires integrating information from various sources: perceptual information, relevant facts and experiences, feedback from the emotional systems and the physiological consequences of the emotional arousal, expectations of results of various possible actions and so on.

We have to be careful not to fall into the illusion that all executive functions happen in the prefrontal cortex. For example, recent studies point to contributions from the parietal regions to the decision making process of what eye movements to control. The executive functions are a process that happens in many areas working in unison, particularly engaging the attentional systems.

The thalamus, according to signals (produced by the echoes and handshakes corresponding to matches with external or internal stimuli) from the orbital cortex, will generate a degree of calmness or anxiety indicative of how positive or negative the event is. The activity of the amygdala is regulated through circuits that link the thalamus to the amygdala and project back to the prefrontal and orbitofrontal cortical areas, reflecting the degree of anxiety or calmness. The levels of dopamine, in part, will then determine the degree of pleasure or desirability.

The brain communicates its internal emotional state to other brains using specific universally recognizable facial movements, gestures, body postures and the prosody of speech. In social animals this is of extreme importance.

The emotional displays reflect in very subtle ways the emotional states of the brain; and an emotional transfer is achieved when properly read by the recipient. And more interestingly, producing the exact facial expression of an emotion generates the actual emotion. This has big therapeutic potential.

Interestingly, recreating an emotional facial expression generates the emotion. Raising the inner eyebrows, and raising the cheeks, and lowering of the corner of the lips, will be sufficient to create marked changes in the autonomic system. This generates sadness and anguish. If you lower the brows, and raise the upper eyelid, and narrow the eyelids, and press the lips together, you generate anger. The heartbeat will go up ten to twelve beats and the hands will get hot. By simply learning how to create the facial expressions that correspond to stressful emotions as anger, sadness and fear, will produce the corresponding physiological changes.[171]

The orbital prefrontal cortex is positioned as a convergence zone where the cortex and the subcortex meet. It is the only cortical structure with direct connections to the hypothalamus, the amygdala, and the reticular formation in the brain stem that regulates arousal.[172] Other areas of the cortex, through extensive handshakes to the orbital prefrontal regions activate neurons that process facial gestures and prosody. These handshake signals can also activate clusters capable of appraising changes in the social environment, especially social interactions.

Neurons in the parietal and frontal lobes, called mirror neurons, that fire when you move your hand as well as when you just watch someone else move their hand, also have counterparts that not only fire when you stick your tongue out or purse your lips, but also when you see someone else do it, even though you have never seen your own lips or tongue.[173] The mirror neurons guide imaginary replication of other's expressions, and through the association of emotions to motor commands linked to emotional expressions, trigger subtle emotional reactions.

Emotions do not only elicit specific, related memories and behaviors, but also, through the external expression of these emotions, communicate these emotional changes to others. What is known in psychotherapy as transference and countertransference is a continuous process of emotional information transfer between individuals. Species-specific, emotional, expressive displays in the sender activate the attentional systems of the receiver, thus becoming emotionally competent stimulus. As such, the expressive displays automatically trigger the same emotion in the receiver. When the brain of the receiver perceives the emotion, the meaning of the emotional display is felt directly. The attentional systems hover, shifting from the external sensory stimuli (the expressive displays) to the internal emerging bodily sensations (the emotions generated as an automatic response). Thus the emotional communication achieves a somatic transference.

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171 Malcolm Gladwell, *Blink*.

172 Allan Schore, *Affect Regulation*.

173 V. S. Ramachandran, *A Brief Tour of Human Consciousness*.

When emotionally competent visual and auditory stimuli emanating from a face are detected (by matching facial and prosodic echoes), handshakes are sent to the orbitofrontal cortex. These handshakes are interpreted as particular ECSs by the orbitofrontal cortex. Due to its unique connections, concurrent changes in the emotional or body state can be enacted as a response to the external emotional information. This reciprocal process reflects the changes in internal states. This transfer of emotional information begins in early development between child and mother, and continues throughout life in social interactions.

The emotional information transfer between two or more people is achieved in a variety of ways. The transference of emotional information is based on recognizing the affective expressive qualities using various sensory signals (visual, auditory, tactile, kinesthetic and olfactory). Facial expressions are automatically mimicked. When we do so, our sequence of facial movements can trigger the same emotion that generated the facial expression. The emotional transfer is achieved by generating internally the same emotion, generally at a less intense level. We "recognize" emotions from visually presented facial expressions by reproducing internally the emotion that would generate the same facial expression. This non-verbal process allows us to feel what others are feeling.

A gaze between one individual and another also acts as a strong channel for transmission of emotional states. It has been observed that the pupil of the eye acts as a nonverbal communication device.[174]

Under normal conditions every transference of emotional information elicits a countertransference that confirms the emotional communication. These reactions are very fast, occurring in microseconds, reflecting the somatic emotional changes. Transference of emotional positive changes generates positive changes in the receiver, just as transference of negative emotional changes produce negative changes in the receiver. These transferences are very valuable in a social environment to: a) quickly spread information about how good or bad an event is; b) to ease coordination of actions among a group as a response to an outside threat or opportunity; and c) to facilitate adaptive behaviors such as attachment, bonding, fleeing or attacking. These are a bodily-based perception of meaning. The feelings are a viscero-sensation that serves to evaluate if an event is "good" or "bad" in proportion to the feeling generated. Thus sensory inputs alter the internal environment to heighten or dull the perception of the external world and elicit a behavioral response.

Not surprisingly, since the memories to identify faces and prosody are on the right side, recent studies have shown that the right hemisphere is faster than the left hemisphere in processing emotional content from facial expressions and spoken language.

The right hemisphere is, more so than the left, deeply connected into not only the limbic system but also the sympathetic and parasympathetic branches of the autonomic nervous system that controls the somatic expressions of all emotional states. Because the

[174] Hess, E.H., *The role of pupil size in communication*.

hypothalamo-pituitary-adrenocortical axis and the sympathetic-adrenomedullary axis are both under the main control of the right cortex,[175] the right side is responsible to a greater degree to mediate adaptive functions by generating the appropriate emotions. Basic emotions—excitement, fear, elation, rage, disgust, and shame—produce differentiable autonomic activity. The intensity of the feelings, positive or negative, elicited by the emotional reactions, produces non-verbal evaluations of events; we "know in our guts" if it is good or bad. The automatic and fleeting emotional expressions produce an emotional reaction in the receiver. This resonating process is interactively regulated, amplified, and can be held in short term memory long enough to be felt and recognized. At this point the right-brain "gut" knowledge becomes available to the verbal (thinking) left hemisphere for further processing.

The ability to communicate to others and, read in others, these subtle emotional states, varies from individual to individual (and from species to species).

After the brain's perception of the emotion, the non-verbal emotional experience in the right hemisphere must be transferred to the left hemisphere for linguistic expression of the feeling. The bodily sensations produced by the emotion are perceived as feelings and eventually the feeling can be verbally articulated. This allows for a linkage of the non-verbal implicit and verbal explicit memories through emotions.

When a stimulus generates an emotion, the emotion gives rise to a bodily felt sensation, which is the implicit knowledge. It is knowing without words. The explicit knowledge emerges at another level. It is formed from pre-conceptual, implicit, and incomplete information through the interaction of feelings and symbols (words or thoughts). The explicit knowledge is not a previously hidden one that now becomes clear, but one that is formed by putting feelings into words. This process, the verbalization of emotions, is not always possible.

There are several emotional regulation modes: one is an interactive regulation via two or more emotionally interacting individuals, the emotional information transfer; another, is an interactive regulation through the external changing conditions that trigger changes in the internal emotional state; and lastly, is a self-regulatory process, based on feedback and automatic tendencies to reach a neutral state of calmness.

The Isorropic Circuit

This elegant system, comprised of a few small structures buried deep in the middle of the brain (primarily, the habenula and the medial and lateral septal nuclei, including the diagonal band of Broca), receives its major inputs from the thalamus and the hippocampus and is continuously adjusting the internal emotional state to the external, ever-changing environment, attempting to produce, at all times, the adequate emotional response. This system, through the signals it sends to the reticular formation,

[175] Allan Schore, *Affect Regulation.*

is an emotional arousal and, balancing and tuning system. It summarizes the activity of the entire brain, taking into account emotions, cognitive signals translated into contexts and meanings, and accordingly, fine-tunes the signals of all neurotransmitter systems in the brain stem.

This arousal system, in a slow gradation of excitability, from less to more, associated with negative and positive emotions, using asymmetries in the brain, detects and generates very fine emotional distinctions covering a whole range: from happy, changing slightly to blissful, playful, grateful, safe, relaxed, undecided, uneasy, frustrated, alert, tense, cautious, anxious, afraid, all the way to panicky; or conversely, starting from lonely, changing to bored, humble, bashful, ashamed, envious, determined, optimistic, joyous, all the way to loved.

The primary function of this circuit is to try to balance activity in both hemispheres, continuously attempting to restore neutral calmness, reflecting its evolutionary path when lateralization was less prevalent in more primitive brains. This habenula-septal circuit is important enough to deserve it own name, the isorropic circuit (from the Greek, isorropia=balance). It is worthwhile to see in detail how the isorropic circuit works.

I recommend reviewing the details of this important circuit. However, the reader may go to next chapter, The Self, where we present how this circuit is related to the generation of the self.

The convergence of signals from the cortex and the output of the basal ganglia (where the integration of external with internal space is accomplished by the basal ganglia loops through a continuous stream of echoes that activate implicit memories and trigger emotions automatically) to the thalamus as well as the extensive reciprocal connections of the cortex to the hippocampus are the circuits where a variety of cortical signals can be unified and the attentional systems controlled. This unification and control of attention permits the activation of explicit and episodic memories aided by the very subtle activation of the needed emotions.

If we add the connections to the hippocampus, the thalamus and the basal ganglia to the isorropic circuit, we can identify the center where all cortical activities are streamlined into a single unified perception. This perception has several components: a spatial/somatosensory (a body/external space integration); a sensory (from all senses); echoes from all cortical areas (implicit memories) as well as an emotional component, which in turn has the potential to activate handshakes (explicit memories); and, an associated context (activating attention to focus on associative areas—more handshakes). When the attentional systems—the somatosensory, the visual and the auditory—focus on one salient stimulus to extract the most possible information, and produce a certain feeling, this feeling is as close as possible to a technical definition of consciousness. Consciousness is what it feels like to be here.

Emotions can be labeled positive or negative. In general, situations and events conducive to positive emotions should be promoted; those leading to negative emotions, avoided. Such emotions should not be confused with motivations of approaching or withdrawing; both easily found behaviors within the context of experiencing a positive

or negative emotion. Approaching or withdrawing behaviors depend on previous knowledge and/or a particular context.

The right hemisphere computes on a moment-by-moment basis the emotional salience of external stimuli. This is equivalent to sending out handshakes with a "valence tag," in which perceptions are given a negative or positive affective value reflecting degrees of pleasure-unpleasure.

Positive emotions are associated with a very slight increase of neural activity in the whole brain, and activation of the left anterior cingulate cortex, relative to the right. Conversely, negative emotions are linked to a slight decrease in neural activity overall and activation of the right anterior cingulate cortex, relative to the left.

Emotions can produce asymmetrical activation, where, for example, anger produces more left frontal activation and fear more right frontal activation.[176] In this case we need to think of anger as a positive emotion and fear as a negative one.

There are circuits associated with positive and negative valenced emotions. Using the output of these circuits, the orbitofrontal circuits alter the brain's basic rates of signaling: the positive states speeding up the rate of signaling and the negative states reducing the overall rates. The job of the isorropic circuit is to restore balance within certain ranges as soon as feasible.

As we have mentioned, the output of the TAN neurons in the caudate nucleus is directed to the basal ganglia, whose output in turn is mostly directed to the thalamus.[177] The caudate nucleus is the interface between emotions and memories. The thalami are connected contralaterally (in about 70 per cent of people, in women more than men, and in women more strongly than in men) through the massa intermedia.[178] Could there be some relation between the 30% that do not have a massa intermedia, making it harder to balance activity on both hemispheres, and a tendency to suffer unipolar or bipolar disorder?

The output of the isorropic circuit (through a series of circuits that connect the cortex to the hippocampus and the thalamus, other projections that connect both hemispheres of the brain, and feedback loops to the hippocampus, involving inhibitory and excitatory neurotransmitters) to the reticular formation, which controls release of all major neurotransmitters, can alter the functioning of the whole brain.

In a nutshell, the habenulas receive major information from the activity of the thalami, reflecting total cortical activity from each hemisphere, including information from the amygdala, mirroring the emotional state. As a minor input, the habenulas receive, through the medial septal nucleus, contextual information from the hippocampus. The habenula on each side connect to each other through the habenular commissure.

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[176] Jan Wacker, Marcus Heldmann, and Gerhard Stemmler, *Separating Emotion and Motivational Direction in Fear and Anger: Effects on Frontal Asymmetry.*

[177] Jeffrey M. Schwartz, *The Mind and the Brain.*

[178] John H Martin, *Neuroanatomy.*

Thus the habenula has efferents from various structures involving different neurotransmitters: acetylcholine, serotonin and dopamine from the septal nucleus and primarily GABA (which can be affected by dopamine) from the thalamus' stria medullaris. The hippocampal and the habenular commissures, more so than the massa intermedia that joins the thalami, assist in maintaining a balance between the activity of the two hemispheres by speeding up or slowing down one hemisphere with respect to the other.

This architecture suggests a strong emotional modulatory role for these circuits: when activity in the brain picks up (positive emotions), the GABAergic interneurons of the isorropic circuit slow it down; when activity slows down (negative emotions), the activity of GABAergic interneurons slows down, and brain activity increases. However, the increase or decrease of brain activity modulated by the habenula is not a linear function because GABAergic interneurons also slow down or speed up cholinergic activity inversely. Simultaneously, the cholinergic activity increases or decreases the inhibitory action of GABAergic projection neurons, but because the cholinergic excitation is through slow muscarinic receptors, there is a time lag. This time lag, allows the influence of dopamine and serotonin to change the functioning of the brain. However, at the level of serotonin or dopamine, any attempt to pharmacologically influence the functioning of this circuit is very slow, sometimes even up to two weeks.

Indirectly, through the thalamus and the hippocampus, when positive emotions are present the left habenula receives greater excitatory signals than the right habenula—the activity in the left anterior cingulate is greater compared to the right and activity of the cortex as a whole is increased—excitatory (more+) from the whole cortex and excitatory (more+) from the left anterior cingulate through the thalamus. Let's call it (more+)(more+) excitation. Whereas the effect on the right habenula under similar conditions is excitatory (more+) from the whole cortex but less from the right anterior cingulate (less-). Let's call this (more+)(less-) excitation. This will establish a net excitatory effect from the left to the right habenula, attempting to restore a balance in both hemispheres by making the signals from both habenulas (left and right) to the reticular formation more similar. This net excitatory effect from left to right through the habenular commissure is perceived by the brain as pleasant.

Adding to the excitatory (or inhibitory) effect is the action of the massa intermedia (when existent) and of the hippocampal commissure, stimulating (or inhibiting) each hippocampus to match each other's activity and through their many glutamate and cholinergic cortical connections increase or decrease each hemisphere's activity as well.

Similarly, when negative emotions are present the left habenula receives less excitatory signals than the right habenula—the activity in the right anterior cingulate is greater compared to the left and the activity of the cortex as a whole is decreased—less excitatory (less-) from the whole cortex and less excitatory (less-) from the left anterior cingulate. The excitation is (less-)(less-). Whereas the effect on the right habenula is less from the whole cortex (less-) but greater from the right anterior cingulate (more+); this produces (less-)(more+) excitation. This is not necessarily the same as

(more+) (less-), because the degree of excitation from the whole cortex and the anterior cingulate are not equivalent. In this case, a net excitatory effect is produced from the right to the left habenula. This net excitatory effect from right to left is perceived by the brain as unpleasant.

The contralateral communication between the medial septal nuclei and the habenulas also helps regulate the output of each habenula to the interpeduncular nucleus and the reticular formation. Thus, GABAergic, cholinergic, dopaminergic and serotonergic systems excite a cholinergic output to the reticular formation. The actions of these circuits continuously attempt to establish a balance between the activities of each hemisphere. When the activity of both hemispheres is equal, a preferred state of neutral calmness is achieved.

The isorropic circuit's main activity is to return the whole brain to a desired neutral state, speeding up or slowing down the activity of each hemisphere or the entire cortex as needed. Dictated by the cytoarchitecture, the isorropic circuit can re-establish a neutral calmness at a faster rate when the emotion is positive than when it is negative.

Primarily, the isorropic circuit achieves five actions: 1) determines the emotional state through the indirect connections from the amygdala; 2) determines the context of the emotion through the connections from the hippocampus; and 3) measures and compares the neural activity in each hemisphere to determine if a positive or negative emotion is present by comparing the activity of the left and right habenulas aided by the signals from the thalami, the amygdalae and the hippocampi. According to these first three actions the isorropic circuit then: 4) modulates the activity in each hemisphere by dampening or quickening activity as needed; and 5) increases or decreases overall activity of the entire cortex. The overall net result of the actions of the isorropic circuit is to naturally drift, at varying rates, towards a neutral state.

When there is relatively more activity in the right cingulate cortex, the emotion is determined to be undesirable (negative, feels unpleasant, and proportionally, everything is done to lessen or rid the organism of it). When the reverse is true, the emotion is determined to be desirable (positive, feels pleasant, and proportionally, everything should be done to increase the outcome).

Slowly, depending on the changing external environment and internal conditions, which produce various responses and behaviors, the isorropic circuit will manage to restore calmness. This is one way of saying that positive and negative emotions are a transitory condition.

The output of the isorropic circuit depends on a comparison of the entire cortical echoes. The echoes, in turn, depend on the emotional state as well as all the feedback handshakes generated by the emotion and the context of the emotion. These continuous feedback circuits summarize the totality of brain activity in a compact way. They fine-tune the emotional landscape of the brain by regulating the reticular formation activities, including regulation of arousal, motor control and vegetative functions.

Thus, the outputs of the isorropic circuit (projections from the habenula, through the habenulointerpeduncular tract, to the interpeduncular nucleus in the reticular formation), affect most all the principal neurotransmitter systems:

a) Acetylcholine through the basal nucleus of Meynert (to the cortex) and the septal nuclei (to the hippocampal formation). Acetylcholine augments excitability of cortical neurons, especially in the association areas.
b) Dopamine through the substantia nigra pars compacta and ventral tegmental area, prepare the brain for action. This is an activation-excitatory circuit.
c) Serotonin through the Raphe nuclei. The actions of this system are diverse because there are many types of serotonin receptors.
d) Noradrenaline through the locus ceruleus with projections to the thalamus, amygdala and hippocampal formations as well as medullary projections. This system plays an important role in reacting to stress, and particularly the activation of fear and anger.

Through the various projections mentioned, the isorropic circuit affects and regulates the output of other loops. The amygdala projects to the brain stem areas that are involved in the control of the heart rate and other autonomic nervous systems that will regulate the body's responses as emotions change.

The reticular formation is the core of the brain stem. Neurons of this region regulate the neuronal excitability of the entire cortex.

The right hemisphere evaluates emotions more than the left hemisphere. The right brain contains circuits involved in regulating intense emotional-homeostatic processes and modulates negative primary emotions such as fear and disgust and negative social emotions such as shame, as well as positive emotions such as excitement and joy.

When the brain perceives emotions it creates the illusion of feelings. These feelings serve to guide our responses to the ever-changing environment. The ability to adapt depends on the regulatory process to return smoothly to a neutral state of calmness.

The Self

The cholinergic (acetylcholine) projections of the isorropic circuit act through muscarinic receptors. The muscarinic receptors are slow-acting, consequently their effects occur with a time lag. This time lag, in chaos-theory terms, allows the isorropic circuit to wander about a strange attractor, which under normal conditions represents the point of neutral calmness. Try to visualize a small, variable circular trajectory moving about a point, but never settling on the point.

Under most conditions, this strange attractor (mathematically represented by a point) is the state towards where the isorropic circuit's actions tend to modulate the cortex's activities. Let's call this attractor the isorropic attractor. The subtle wandering of the isorropic attractor around neutral calm permits the isorropic circuit to generate and regulate a wide spectrum of emotions. Potentially, this wandering permits the generation of myriad slightly differing emotions as a response to a vast array of changing internal and external stimuli.

When the isorropic attractor is close to neutral calmness a wider spectrum of potential emotional responses is available, positive and negative, and by activating memories associated with the emotions, allows for activation of a greater pool of memories. The speed at which small emotional changes can be generated also allows the search for the greatest potential number of contexts. Thus the brain achieves a state that can generate the maximum variety of behaviors in the quickest time possible as response to the ever-changing environment. This places the organism in a state of maximum adaptability. When the brain detects this healthy state of maximum adaptability it is felt as a sense of self.

Some philosophers have called this state "the center of the self." Others have called it the "true essence." Franz Kafka, the Austrian philosopher and poet, recommended, perhaps as a means to reach neutral calmness, "You need not leave your room. Remain sitting at your table and listen. You need not even listen, simply wait. You need not even wait, just learn to become quiet, and still, and solitary. The world will freely offer itself to you to be unmasked. It has no choice; it will roll in ecstasy at your feet."[179]

Under certain conditions the isorropic attractor locks into a state different than neutral calmness, in which case the brain produces a mood or a long lasting emotional state.

The wider the spectrum of emotional responses that can be triggered, the more intense the sense of self is felt. Conversely, the narrower the spectrum, the less intense the sense of self is perceived. This translates into a nonverbal knowledge that many memories can be quickly activated because the brain can flutter, ever so slightly from emotion to emotion, changing from moment to moment the chemical milieu of the body and brain. Normally, the brain is changing, second by second, the emotional internal landscape in subtle ways that are almost imperceptible.

Again, in chaos theory terms, when the isorropic circuit's attractor is wandering on the positive emotional side, the brain perceives this as a high degree of self-esteem. If it is wandering on the negative emotional side, the degree of self-esteem is low.

When the isorropic circuit is close to neutral calmness, autobiographical memories can be more easily activated because of the continuous wandering of the isorropic attractor; as a result, most all emotions associated (through handshakes) with all the memories can be quickly triggered.

When the isorropic attractor is far from neutral calm, short term, working and explicit memories can be affected, primarily because thalamic control of the attentional systems can be disrupted.

In contrast, semantic, source and procedural memory, because of a low or inexistent relation to emotions can still be activated even when the isorropic attractor is far from neutral calmness. The hippocampus can detect a particular context, independent of an emotion, and send the appropriate signals and activate these types of memories because they are independent of the emotional state.

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[179] Deepak Chopra, *The Seven Spiritual Laws of Success*. 1994.

If environmental or internal stimuli push the isorropic attractor outside the chemical ranges that permit homeostatic balance, the sense of self becomes distorted; the spectrum of emotions that can be potentially generated, thus the memories that can be activated, are greatly diminished and the behavioral responses limited. The limited choice of behaviors, imposed by stressful conditions, under a wide array of circumstances, can still generate a correct response. But the longer the stressful conditions prevail, the smaller the number of responses that can be activated and the less the probability that the correct response will be generated.

In extreme cases, when the isorropic attractor is pushed far enough from the range of homeostatic balance, the brain will no longer be able to perceive a sense of self; the organism can no longer adapt.

The sense of self is an indirect measure of the organism's capacity to adapt.

There are other aspects to the sense of self superimposed on the activity of the isorropic circuit. As we saw in detail at the tennis match, there is a constructive process of the present. Even when there is a referral in time of sensory signals and backward masking, and these signals are processed faster than they can reach awareness, this constructive process creates a sense of continuity of our experience with an accompanying feeling of past (using different memory systems to remember events) and present (using attentional systems to create it).

When our attentional systems activate biographical memories, in spite of the diversity of sensory signals, memories, beliefs and thoughts, a coherent sense of self emerges. In this way the brain constructs continuity and a unity of the self.

But still, there is more to the self; there exists a sense of embodiment, a feeling that we are bonded to our bodies. The somatosensory attentional system, using all pain, pressure, temperature and proprioceptive signals creates this illusion and firmly roots us in our bodies. And beyond this, the self is empowered with a sense of agency or free will, even if it is generated outside awareness, which allows us to feel we can direct our bodies and our actions.

These different aspects of the self are embedded in an ever-changing emotional milieu, which as we have seen activates relevant memories. With the use of mirror neurons, through emotional expressions and emotional transfers, the brain perceives feelings as well as feelings in others. Ultimately, the activity of the attentional systems permits us to reflect, of becoming aware of our self with all its subtleties. The attentional systems, somatosensory, visual and auditory (perhaps olfactory and gustatory as well), along with their memory systems, are the circuits that generate the sense of self and our perception of consciousness.

The self has many sides to it and as a result any of these different aspects can be individually disturbed. However, given the predominant emotional nature of the self, which activates memories associated to the emotion according to a context, even in the extreme case of split-brain patients whose two hemispheres have been surgically disconnected, the patients don't exhibit split personalities or a double subjectivity.

Awareness orients the individual in the environment. Attention lengthens and strengthens the neural signals which are responding to the sensory focus of attention. At this level we can define perceptual consciousness as the perception and cognition of the body and the environment. At a higher level, emotions activate their related memories simply by the chemistry of the emotion, which is an integral part of the memories, thus intensifying the neural signals that represent the memories. Emotions make many more memories accessible. At this level, we have the intensification of neural signals by the attentional system and the added intensification of neural signals related to the emotion. Superimposed on all these neural processes, the sense of self, the activity of the isorropic circuit, is perceived as reflective consciousness. Reflective consciousness could thus be defined as the activity of awareness, combined with the activity of the attentional systems, joined to emotional responses embedded in the activity of the isorropic circuit. In other words, reflective consciousness is another aspect of the sense of self.

The normal functioning of the isorropic circuit and disturbances of the self will determine personality traits. The personality traits will determine the patterns of response to various stimuli. The interaction of the isorropic circuit with the basal ganglia circuits is genetic, whereas the interaction of the cortex and the hippocampus with the basal ganglia is stored as memories and is mostly learned. In a gradation from normal to maladaptive personality traits, nature and nurture will interact to produce individual responses.

The American Psychiatric Association recognizes marked disturbances of personality traits that affect cognition and affectivity. Part of the diagnostic criteria for Personality Disorders is when these patterns cause significant distress or impairment in social or other important areas. These enduring patterns of cognition and affectivity are inflexible, stable and of long duration.[180]

If we lessen the gravity, or reduce the number of symptoms of the diagnostic criteria for the recognized personality disorders, each personality disorder could be similar to someone we know. Consequently, basic personality traits could be deduced from this list in a gradient: trust/distrust, attach/detach from relationships, cognitive or perceptual distortions, sympathy/empathy, cautious/impulsive, affective distortions of self or others, submissive/dominating and orderliness/disorderliness.

Fear & Panic Attacks

Interestingly, the thalamo-amygdala and cortico-amygdala pathways converge in the lateral nucleus of the amygdala. Once the signal is received there, it can be relayed through the internal amygdala pathways to the central nucleus, which in turn releases the full repertoire of defensive reactions. The amygdala sends impulses

[180] Diagnostic and Statistical Manual of Mental Disorders. DSM-IV-TR.

to the hypothalamus to release a hormone called corticotrophin releasing factor, or CRF, which signals the pituitary and adrenal glands to flood the bloodstream with epinephrine (adrenaline), norepinephrine and cortisol. These stress hormones shut down nonemergency systems, such as digestion and immunity, and direct the body's resources to fighting or fleeing. Perspiration increases to regulate body temperature, breathing quickens to take in more oxygen, the heart beats faster, the liver releases sugar into the blood for added energy, blood pressure rises. All the benefits produced by this response to a passing threat can, over a longer term, wear the body down. Constant stress can lead to impaired memory, a weakened immune system, high blood pressure and stomach ulcers.[181]

Under normal conditions, fear is a short-term response to an immediate threat signaled by the amygdala as stress hormones are released by the paraventricular hypothalamus. Anxiety is a long-term response compared to fear. Anxiety is an emotion triggered by unpredictable threats from stimuli that indicate something is not right.[182]

We can think of a fear circuit in a simplistic way: a danger stimulus goes from senses to thalamus. If a match is made between a dangerous stimulus and a dangerous echo, the signal is sent automatically to the amygdala. Simultaneously, the signal is relayed to the sensory cortex. The amygdala triggers the fear response. The sensory cortex processes the stimulus, determines what it is and passes it to the prefrontal cortex, which in turn, will determine if the stimuli is or isn't a threat. If the prefrontal cortex determines it is not a threat, it will signal the amygdala to dampen and stop the fear response. If it determines it is a threat, it will quantify how big of a threat and signal the amygdala to reinforce or dampen the fear accordingly.

The hippocampus will be alerted when a danger stimulus has been matched and will quickly assess the context of the stimulus; in the right context, the hippocampus will signal the amygdala and reinforce the fight-flight response accordingly. If the stimulus is in a wrong context, the hippocampus will signal the amygdala and the fear response will be dampened. The hippocampus signals can override the signals from the sensory cortex as they enter the amygdala farther down the fear circuit. In the absence of a signal from prefrontal cortex and the hippocampus, the amygdala locks into a positive feedback loop and the fear response automatically escalates slowly until, in less than ten minutes, it can become a panic attack.

People suffering from panic attacks and phobias have lost the ability to turn the fear response off. Panic attacks can happen within the context of any Anxiety Disorder as well as any Mood Disorder. The sense that something is wrong and beyond control might be enough of an emotional competent stimulus to trigger an attack.

A panic attack has a sudden onset and builds to a peak rapidly and is often accompanied by a sense of imminent danger or impending doom and a strong urge

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[181] Robert M. Zapolsky, *Why Zebras Don't Get Ulcers.*

[182] Joseph LeDoux, *The Emotional Brain.*

to escape. The anxiety that accompanies a panic attack can be differentiated from generalized anxiety by its discrete nature and its typically greater severity.

In the case of panic attacks, the thalamus incorrectly detects a danger signal, confusing a signal with the wrong echo (one that is not reflecting the ECS) and it triggers the fear mechanism. The signal is then reflected to the cortex. A real threat would be reflected to the right area of the cortex, and in turn the cortex would signal the amygdala and reinforce the original thalamic signal. In normal conditions, if the processing of the signal were determined to be no threat, the cortex would signal the amygdala accordingly and quickly dampen the fear. In a panic attack, the signal was reflected to an area that does not detect threats (because of the confusion between signal and echo) and consequently this area cannot signal the amygdala either way. The hippocampus isn't alerted either and therefore it doesn't detect a threat, because there is none or moreover, the context doesn't indicate a threat. Under these conditions, the amygdala continues with the fear response and it quickly escalates. When the Panic Attack is triggered for no reason and is completely unexpected it is termed uncued.

When the thalamus detects a signal and matches it to a danger echo, it initiates the fear response and reflects the signal to the cortex; and when the cortex evaluates the danger and does not signal the amygdala that it was a false alarm (it's a stick not a snake), the amygdala continues escalating the fear signal. This is what is known as a situationally bound (cued) Panic Attack.

Finally, there can be a situation in which the thalamus matches a stimulus to a dangerous echo, initiates the fear response, reflects the signal to the appropriate area in the cortex, the appropriate signal is sent to the hippocampus to evaluate the context, but the hippocampus fails to evaluate that the signal is in the wrong context, and sends a danger signal, instead of a false alarm signal, to the amygdala. This signal (the stick bites, watch out) overrides the sensory signal (it's a stick not a snake; sorry, false alarm). This is known as situationally predisposed Panic Attack. The attack is similar to situationally bound Panic Attacks but is not invariably associated with the cue. Sometimes they happen and sometimes they don't, or the panic attack occurs sometime later as the hippocampus belatedly sends a danger signal.

It is clear that there are small differences in each of these different types of panic attacks, and each one will require a different therapeutic approach.

The essential feature of Panic Disorder is the presence of recurring, unexpected panic attacks followed by at least one month of concern over having another panic attack or worry about the possible implications or consequences. There can also be a significant behavioral change related to the attacks.

Distinct from fear and anger, which are clarifying emotions, anxiety, an error-detecting scheme, often is confusing. Anxiety can be resolved by figuring out what is wrong and correcting it, or simply by realizing that there is nothing wrong and dampening the anxiety.

In short the amygdala receives low level (rough) sensory signals from the thalamus, higher level information from sensory-specific cortex, and still higher level (sensory

independent) information about the general situation from the hippocampus. Through such connections, the amygdala is able to process the emotional significance of individual stimuli as well as complex situations. The amygdala is involved in the appraisal of emotional meaning. It is where trigger stimuli do their triggering!

It is easy to see how a malfunction of any of these pathways might lead to emotional disorders. If in some individuals thalamic pathways are dominant or become uncoupled from cortical pathways, these people might form emotional memories on the basis of stimulus events that do not coincide with their ongoing conscious perceptions. Because thalamic pathways to the amygdala exit the sensory system before conscious perceptions are created at the cortical level, which only represent features and fragments of stimuli, these will not necessarily coincide with the perceptions occurring in the cortex. Such people would have very poor insight into their emotions. Similarly, if the hippocampal system were uncoupled from the thalamic and cortical projections to the amygdala, you might have people who express inappropriate emotions to the immediate context, including the social context.[183]

These ideas could indicate an approach to some possible therapies, through the investigation of which pathways are weak or nonexistent, and find therapies that could strengthen or create new ones.

More Emotions

To understand mood disorders, one must have a rudimentary understanding of emotions. Each emotion is a particular chemical state of the brain. Emotions exert powerful influences on cognitive processing by activating related memories. Attention, perception, memory, decision making, thinking—all are modified by emotions. When an emotion is detected it is perceived as a feeling and the feeling intrudes on the attentional systems. Emotions in the wrong context can spell disaster.

Emotions intrude on attention when a second stimulus is emotionally significant (another echo is matched by the thalamus) and the thalamus overrides the attentional systems by sending a signal to shift attention to this new stimulus. In other words, when the amygdala receives a signal signifying an ECS, attention shifts to even an unattended event.

Laughter

Each emotion has its own circuits and loops. Different studies of the brain, suggest, for example, the existence of a laughter circuit. When a region in the supplementary motor cortex, close to the area in the frontal lobes that receives input from the brain's emotional centers, is stimulated, patients laugh spontaneously. In cases where a person

[183] Ibid.

literally dies of laughter (she could not stop laughing until, exhausted, she died), subsequent autopsies have revealed damage to portions of the limbic system: the hypothalamus, mammilary bodies and the cingulate girus, all of which are involved in emotions. Given the well-known role of the limbic system in the fear circuit, it is not surprising that it is also involved in the aborting of a reaction in response to a false alarm—laughter. The feeling of merriment that accompanies laughter is a further indication that the limbic system is activated.

The main purpose of laughter could be to allow the individual to alert others in the social group (usually kin) that a detected anomaly is trivial, nothing to worry about. The laughing person in effect announces her discovery that there has been a false alarm; that the rest of the group need not waste precious energy and resources responding to a spurious threat. This would also explain why laughter is contagious, for the value of the signal would be amplified as it spread through the social group.

Normal laughter is triggered by a specific, usually mirthful stimulus accompanied by mood elevation and is a coordinated motor function involving facial and respiratory muscles.[184]

Laughter could also be part of a bonding process, not only socially but at a deep emotional level, starting in childhood between parents and children and continuing throughout life.

OCD & Changing the Brain

A discussion of Obsessive Compulsive Disorder and possible therapies helps to illustrate how complex emotional behaviors can arise.

As Dr. Jeffrey Schwartz explains in his book, *The Mind and the Brain*, one of the most striking aspects of Obsessive Compulsive Disorder (OCD) urges is that they seem apart from, and at odds with, one's intrinsic sense of self. They seem to arise from a part of the mind that is not you, as if an impostor were inside your mind. OCD can manifest itself as obsessions about order or symmetry, as expressed in an irresistible need to line up the silverware or hoarding. Paradoxically, giving in to the urge to wash or check or count or sort, which is done in the vain hope of making the dreadful feeling recede, backfires. An OCD compulsion does not dissipate like a scratched itch. Instead, giving in to the urge exacerbates the sense that something is wrong.

OCD patients dread the arrival of the obsessive thought and are ashamed and embarrassed by the compulsive behavior. They carry out behaviors they are desperate to escape, either because they hope that doing so will prevent some imagined horror or because resisting the impulse leaves the mind unbearably ridden with anxiety and tortured by insistent, intrusive urges. Since the obsessions can't be silenced, the compulsions can't be resisted.

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[184] V.S. Ramachandran, *Phantoms in the Brain*

Intense and persistent firing in the orbital frontal cortex causes an intense visceral sensation that something is wrong, and that action of some kind—such as counting, or checking if appliances are on—is needed to make things right. The reason for the visceral sense of dread that OCD patients suffer is that the orbital frontal cortex and the anterior cingulate gyrus are wired directly into the gut control centers of the brain.

After signals from the sensory areas have been processed and are considered by the orbital frontal cortex through certain handshakes to be warnings about potential threats, a signal will be sent to the thalamus to initiate the sequence to release stress hormones, which are felt as anxiety. The difference between anxiety and fear is that fear is triggered before the signal is processed by the sensory areas in the cortex; anxiety is triggered after the signals are processed in the cortex. Anxiety deals with potential threats; fear with immediate danger.

Another overactive region in OCD patients was the striatum. The Striatum is composed of two major information receiving structures: the caudate nucleus and the putamen, which nestle beside each other deep in the core of the brain just in front of the ears. The putamen acts as a major relay and switching station for motor activity as well as integrating visual information with the body and the caudate nucleus acts as a switching for thought and emotion.

Cells in the caudate known as tonically active neurons (TANs) tend to be found between striosomes and matrisomes; small clusters where information from emotion processing parts of the brain, and clumps of axon terminals where information from the thinking, reasoning, cortex, reach the caudate, respectively.

By virtue of their position TANs can integrate emotion and thought. They are the neurons responsible for activating related thought to specific emotions and vice versa. They fire in characteristic patterns when the brain senses something with a positive or negative emotional meaning. TAN's respond dramatically to visual or auditory stimuli that are linked through behavioral conditioning, to reward or punishment.

It seems that distinct environmental cues, associated with different emotional meanings, elicit different behavioral and cognitive responses as TANs shift the output flow in the striatum. This is how thoughts associated to emotions are turned on. When a particular emotion is felt, the tan neurons look for matching echoes; when found, they close the connection and stimulate the relevant thoughts. Conversely, when thoughts are active, they intrude by sending stronger echoes to the striatum and in turn make the tan neurons lock and send the appropriate signals to initiate an emotional response.

The striatum receives input from the entire cortex, with the caudate receiving specifically strong input from the prefrontal areas. Prefrontal inputs include those from the orbital frontal cortex and anterior cingulate error-detection circuitry. It is now often called the "OCD loop". When this loop is working properly, the result is a finely tuned mechanism that can precisely modulate the orbital frontal cortex and anterior cingulate by adjusting the degree to which the thalamus drives these areas. When the

modulation is faulty, the error detector circuit can be over-activated and thus locked into a pattern of repetitive firing. This triggers an overpowering feeling that something is wrong, accompanied by compulsive attempts to somehow make it right.

As a result the direct pathway seems to be stuck in the "on" position. This is what Jeffrey Schwartz calls Brain Lock: the brain can't move to the next thought and its related behavior. The thalamus continuously receives a "something is wrong" signal.

Located behind and above the orbital cortex, the anterior cingulate gyrus also has connections to the vital brain centers that control the gut and heart. The anterior cingulate gyrus seems to amplify the gut-level feeling of anxiety.

With this, a picture of the brain abnormalities underlying OCD emerges. The malfunctions center on the circuitry within the orbital frontal cortex (the "error alarm" circuit) and the basal ganglia, which acts as a switching station. In OCD this error circuit is inappropriately and chronically activated, probably because a malfunction in the gating function of the TANs in the caudate nucleus allows the prefrontal cortex to be stimulated continuously. The result is a persistent feeling (which engenders thoughts) that something is wrong or excessively risky.

Any successful therapy for OCD would need to enhance the gating function of the caudate so that the worry circuit can quiet down and allow a patient to resist OCD urges. The first step is to become aware that the urge is a manifestation of a mental disorder by directing attention to the urge and becoming convinced that how one responds to the urge can change the brain's circuitry. This process is called Relabeling. When done regularly, Relabeling stops the unpleasant feelings of OCD from being unpleasant; understanding their true nature gives a feeling of control. By Relabeling their thoughts as manifestations of a medical disorder, patients willfully make a cognitive shift away from self-identification with the experience into the stream of consciousness.

The second step is to make the patients conscious that there is a neuroanatomical basis of their symptoms, an overactive region in their brains, which is causing the OCD. This is called Reattributing. Having Relabeled an intrusive thought or insistent urge as a symptom of OCD, the patient then attributes it to aberrant messages generated by a brain disease and thus fortifies the awareness that it is not his true self.

Relabeling clarifies what is happening and Reattributing affirms why it's happening. The accentuation of Relabeling and Reattributing tends to amplify attention and allows the patient to separate himself from the intrusive experience. The essence of paying attention during a bout of OCD is to recognize obsessive thoughts as soon as they arise and Refocus attention onto some adaptive behavior. Directed focusing of attention becomes the key action during treatment. The goal of this step is not to obliterate or banish the thought, but rather to initiate an adaptive behavior unrelated to the disturbing feeling even when the feeling is very much present.

Refocusing requires significant willpower. Even when the patient has Relabeled and Reattributed the obsession and compulsions, the anxiety and dread can still feel frighteningly real. Refocusing, therefore, has to center on a pleasant, familiar "good habit" kind of behavior. In essence the patient must substitute a "good" circuit for a "bad"

one. In other words he must change the firing pattern of the TANs and slowly change a negative situation for a positive one.[185] Through repetition other competing positive echoes will dampen the effect of the negative echoes. The diversion can be anything, but a physical activity is especially effective, like shooting baskets or gardening. The most difficult part of treatment, Refocusing attention away from the intrusive thought rather than waiting passively for the feeling to go away requires will and courage.

Setting a finite length of time to resist giving in to an urge helps patients. The fifteen minutes should not be a passive waiting period; it must be an adaptive activity intended to activate a new brain loop. Refocusing alleviates the overwhelming sense of being "stuck in gear". This is where Relabeling and Reattributing come in handy: they both help the patient keep a clear mind about who they are and what the disease process is. This mental clarity has tremendous therapeutic value, for it keeps the Refocusing process moving forward. It also reinforces the insight that active will is separable from passive brain processes. Attention is extremely important to help encode the newer, positive experiences and slowly overwhelm the negative ones.

The next step in therapy is to exploit the brain's tendency to pick up on repetitive behaviors and make them automatic—that is, to form new habits. Patients change their focus from "I have to wash again" to "I'm going to the garden". If done regularly, the urge to wash will produce a habitual association: the impulse to go work in the garden. Ideally, the anxiety felt as an urge to wash will be replaced by a new habit: an anxiety-free desire to go work in the garden.

The last step is Revaluing. Revaluing is a deep form of Relabeling. Relabeling is a superficial encoding which leads to no diminution or improved ability to cope. Revaluing means quickly recognizing the disturbing thoughts as senseless, as false, as errant brain signals not even worth the gray matter they rode in on, let alone worth acting on.

Done regularly, Refocusing strengthens a new automatic loop and weakens the old, pathological one—training the brain, in effect, to replace old bad habits programmed into the caudate nucleus and basal ganglia with healthy new ones. When the focus of attention shifts, so do patterns of brain activity. With regular use of frontal cortex, changes occur in the gating function of the caudate, and mental function improves. Relabeling and Refocusing attention begin to be automatic. In this way frontal cortex thought process begin to be wired directly to the caudate.[186]

Delirium, Dementia and Amnestic and other Cognitive Disorders

A Brief explanation of Delirium, Dementia and Amnestic and other Cognitive Disorders: Knowledge of these Disorders serves as a building block to understand Depression, Manic Depression and Schizophrenia and eventually suicide.

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185 Jeffrey Schwartz, *The Mind and the Brain.*

186 Ibid.

Following the definitions in the Manual, the DSM-IV, the predominant disturbance with Delirium, Dementia and Amnestic and Other Cognitive Disorders, is a deficit in cognition.

The essential feature of Delirium is a reduced clarity of the awareness of the environment. The ability to focus, shift and sustain attentions is impaired.[187] This suggests problems in the thalamus's ability to engage, sustain or shift one or all attentional subsystems (the visual, hearing and somatosensory). The thalamus's shifting rhythms become too fast or too slow and uncoordinated. As a result the attentional systems become grossly unsynchronized. The thalamus's ability to tag sequentially the order of stimuli is also impaired. This is manifested in changes in cognition (that might include memory impairment, disorientation, or language disturbance) or development of a perceptual disturbance.

Memory impairment is noticeable mostly in recent memory. The ability to form new memories is greatly diminished when the hearing and visual attentional subsystems are not functioning properly. The person is easily distracted (attention shifting at wrong time) by any irrelevant stimulus. Disorientation is generally manifested in time or place (not knowing whether it is day or night or where one is), which is indicative of a loss of the sequential tagging quality (in time) of attention. In mild delirium, disorientation to time might be the first symptom to appear.

Different manifestations will be present, depending on which attentional system is disrupted. When the hearing attentional subsystem is affected, speech or language disturbances may be evident as dysarthria (impairment to articulate), dysnomia (inability to name objects), dysgraphia (impairment to write), or even aphasia. As the hearing attentional system is shifted improperly, speech can be rambling and irrelevant, or pressured and incoherent, with unpredictable switching from subject to subject. Perceptual impairment might be present as misinterpretations as when a knock on the door is confused with a gunshot. Misperceptions range from simple and uniform to highly complex, and can include other sensory modalities such as gustatory, olfactory, visual and tactile. When attention shifts incorrectly, it tends to confuse the handshakes to the relevant memories and activates the wrong memories.

When improper shifting impairs the visual attentional subsystem, perceptual disturbances may include illusions (confusing the folds in the bedclothes as animate objects) and hallucinations (seeing something that isn't there).[188] Illusions arise as a result of the visual attention shifting rapidly in an improper sequence and confusing some shapes or borders with wrong categories of memories. Hallucinations are produced when the visual attention cannot differentiate between reality and imagination.

The handshakes and tags that are normally used by attention to jump start relative memories quickly are disrupted and incorrect groups are activated in turn. As a result, the individual might have delusional convictions of the reality of his hallucinations, but exhibits emotional and behavioral responses consistent with their content.

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[187] *DSM-IV-TR*

[188] Ibid.

The shifting of the attentional subsystems tends to fluctuate during the course of the day and the level of disturbance varies accordingly. The person can seem normal in the morning and change later in the day or night.[189]

When the sensorimotor attentional system is disrupted, disturbed psychomotor behavior ensues; these might include groping or picking at bedclothes, attempting to get out of bed when it is unsafe, and sudden movements. On the other hand, the person might have decreased psychomotor activity manifested as sluggishness and lethargy and even stupor. Psychomotor activity often shifts from one extreme to the other. In general, when one attentional system is perturbed by quicker or slower shifting, all attentional systems are perturbed in the same way. While hyperactive, the individual is more likely to have hallucinations, delusions, and agitation; whereas the individual in the hypoactive state is less likely to show these symptoms.

All of these disturbances can bring on emotional disturbances also. Some are natural, like anxiety, fear, anger and depression. Other emotions are just the result of the caudate nucleus matching thoughts to feelings: apathy, irritability and euphoria, and rapid shifting of moods.

If fear is marked, the person might attack those that are perceived to be threatening, or sustain heavy injuries trying to escape from a falsely perceived threat. These emotions might bring on accompanying physical responses like calling out, screaming, muttering or moaning.

The essential feature of dementia is multiple cognitive deficits (including memory impairment), and at least one of the following: aphasia (deterioration of language function), apraxia (impairment of motor activities), agnosia (failure to recognize objects), or a diminution of executive functions. In dementia one or several of the attentional subsystems is impaired. When this happens there is a diminution in the storage and retrieval of certain types of memories.

If the hearing attentional system is affected, difficulty in accessing certain language related memories will be manifested as difficulty in producing the names of individuals or objects. The speech might become vague with long circumlocutory phrases. Comprehension of spoken or written language might be compromised. In extreme cases the individual might be mute or have a speech pattern characterized by echolalia (echoing what is heard) or palilalia (repeating sounds or words over and over).[190]

If the somatosensory attentional system is affected, this will be manifested as reduced ability to execute motor activities despite intact motor abilities, sensory function and comprehension of the task required. The affected individuals might be impaired in their ability to pantomime the use of objects (like combing hair) or to execute a known motor act (like waving good-by). The deficit can be expressed

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189 Ibid.

190 Ibid

in specific motor actions like cooking, dressing or drawing. Also, even though they might exhibit normal tactile sensations, they are unable to identify objects placed in their hands by touch alone.

In turn if the visual attentional system is impaired, individuals will exhibit agnosia; in spite of having normal vision they lose the ability to recognize categories of objects like chairs or pencils. In extreme cases they can't recognize members of their families or even their own reflection in the mirror. They might also be spatially disoriented and have difficulty with spatial tasks.[191]

Executive functions can also be impaired as one or more of the attentional subsystems can't be focused. Since the attentional systems are required to access various kinds of information, many planning functions are disrupted. Executive functions include the ability to think abstractly and to plan, initiate, sequence, monitor, and stop complex behavior. The individual having difficulty with novel tasks may manifest impairment of abstract thinking. Poor judgment and poor insight are also common. The affected individuals may make unrealistic assessments of their abilities; they may underestimate the risks involved in their activities; occasionally they may become violent; suicidal behavior may occur, particularly in the early stages as a result of distortion or loss of sense of self.[192]

Things can become more complicated, Delirium might be superimposed on Dementia: because the thalamus is unable to synchronize the attentional systems and simultaneously the attentional subsystems are impaired.

The main feature of Amnestic Disorders is the inability to learn new things, or make new memories, or recall previous events. The Amnestic Disorders are characterized by multiple cognitive deficits (primarily memory impairment). The ability to learn and recall new information is always affected, whereas remembering previously learned information occurs more variably. When the amnestic disorder is severe, the patient might lack insight into his or her memory deficits. Most cognitive functions remain unimpaired. With profound amnesia, occasionally there is disorientation to time and space, but rarely to self.[193]

In manifestations of memory loss, exactly what kind of past events or types of information can't be remembered? What kind of new information can't be encoded or learned? As we have seen, there are many different memory subsystems; when one or several of these are affected, different kinds or types of memory are inaccessible even if the relevant attentional subsystem is functioning properly. Depending on where the cortex has been damaged or where the flow of the loop has been interrupted, the specific deficit or deficits will be manifested. Essentially the correct handshakes that are associated with other memories are disrupted,

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191 Ibid.

192 Ibid.

193 Ibid.

and this causes the memory to be inaccessible. Sometimes, accidentally, a different association handshake might be triggered and the memory will suddenly be available for recall.

To give an idea of the wide range of problems that have been observed through studying different kinds of lesions, here is some of the different types of memory proposed by some researchers:[194] short-term memory, working memory, intermediate and a long-term memory, explicit and implicit memory, flashbulb memory, source memory, episodic memory, semantic and procedural memory, and autobiographical memory, subdivided into three categories: a lifetime memory (e.g., I lived in Arizona), a general event memory (I vacationed in Arizona), and event specific memory (I visited the Grand Canyon).

The brain uses different strategies to encode and retrieve memories. Each memory category might use some or all of these strategies. To encode, we have deep, shallow and associative strategies. Perhaps there are more. By a repeat/rehearse mechanism we can strengthen these memories. By a leading/distortion mechanism we can change these memories. Retrieval uses various mechanisms: associative, strategic, implicit and explicit, priming, and cues. Some retrieval strategies can even be state dependent, that is one can access a memory when one is in the same state as when the event happened, i.e., under the influence of a drug or alcohol. And again, perhaps there are more.[195]

Studies of damage to certain regions of the brain, lead us to suspect that the hippocampus is involved in explicit memories. The frontal lobes have been identified with activity in strategic retrieval, the purposeful search for memories. The temporal lobes have to do with memories for specific skills, like speaking, reading and writing, recognizing faces and remembering names. Also certain regions of the temporal lobes have to do with memories of categories of objects, like furniture, living or inanimate, tools (which in turn seems to be connected to the motor cortex, which is used to manipulate the tools.), etc.

Impairment of any one or several of these memory systems can produce a wide range of conditions ranging from hardly noticeable to completely incapacitating behavior. Worse, inability to remember certain things or events might bring on, over time, a different psychiatric disorder.

Amnestic Disorders can be Transient (hours or days, up to a month) or Chronic (more than a month).[196]

There are many documented cases where one day, all of a sudden, for unknown reasons, a patient recovers much if not all of his or her lost memories.[197]

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194 Daniel L. Schacter, *Searching for Memory*.

195 Ibid.

196 *DSM-IV-TR*

197 Daniel L Schacter, *Searching For Memory*.

A New Approach to Depression and Manic-Depression

So far, in the discussion of the brain we mostly looked at circuits. We took for granted they worked properly, although loops could lock and be unable to disengage. In the case of mania and depression many circuits are involved. Essentially, mania triggers many positive emotions concurrently and depression triggers many negative emotions simultaneously. Exactly which combination of emotions, and which memories are related to these emotions, determines how differently, from individual to individual these mood disorders express themselves. In my opinion Mania and Depression are both a Thought Disorder more so than a Mood Disorder; the speed of the thoughts triggering the emotions. Manic-depressive patients may have subtle deficits in short— and long-term memory, attention, and executive functions.

In mania and hypomania, we need to explore at the level of neurotransmitters. If there is a very minor increase of glutamate, a small rate of excitation of (projection) neurons ensues, and everything will speed up slightly in the cortex. This probably happens because of a genetic predisposition, as relatives of manic-depressives are more likely to also be manic-depressive, unipolar depressive or schizophrenic. Glutamate is not only a major excitatory neurotransmitter but also an essential metabolite; it is also a building block of GABA, an inhibitory neurotransmitter.

The endocrine system, to complicate matters more, might also be involved. Many aspects of the endocrine system are regulated by the hypothalamus: regulation of sleep, appetite, and sexual drive. In addition, the secretion of hormones by the hypothalamus depends on levels of hormones secreted by the pituitary, thyroid, adrenal and sex glands (testes and ovaries). This complex feedback system could be affected at any one of several levels.

In manic-depressive patients, and even more so in unipolar depressive cases, abnormalities from all three parts of the endocrine system have been observed. In patients with rapid cycling, low thyroid functioning has been detected.

Ultimately, stress has been linked in many cases to the onset of unipolar depression and manic-depression. The stress response also fits nicely with the hypothalamic-pituitary-adrenal axis. This would also be compatible with a genetic predisposition. I believe that stress is a normal reaction to prolonged depression, but stress can be responsible for the onset of depression.

In mania and hypomania, the isorropic circuit locks into a positive state. The isorropic attractor settles away from neutral calm as the action of the inhibitory (GABA) is not enough to slow down the activity of the entire cortex.

When neurons signal each other faster, thoughts and speech become accelerated, accompanied by increased physical activity, decreased need for sleep, increased sexual activity and a possible enhancement of the senses. Prior to the availability of medication, the increased activity could lead to exhaustion and even death. Studies show normal individuals change their thoughts every five to six seconds, compared to less than two seconds for manic patients. The number of syllables spoken in a minute by a manic

patient is between 180 and 200; a normal person's 120 to 155. This acceleration will interfere with attention as the individual will be more easily distracted and have greater difficulty concentrating.

The changed speed and patterns of thought excite the associated emotions by signaling the thalamus and tricking it, triggering a feeling of joy or even euphoria, sometimes with inappropriate spontaneous laughter. Self-esteem grows disproportionately with decreased inhibitions and a greater sense of importance. As a result of the increased self esteem, risky and bizarre behavior is common; grandiose delusions or paranoid delusions are also quite possible; increases in use of alcohol and drugs are frequent. The increased speed of thoughts activates associated positive emotions and activates the pleasure centers. It is also known that some agents that increase dopamine (e.g., L-dopa, bromocriptine, cocaine) may induce mania or mania-like behaviors and feelings; the good feelings induce a faster mode of thinking. Also, it is known that some agents that block dopamine (e.g., haloperidol) may decrease mania.

As a result of the increased self-esteem and speed of thought, all these changes in activities seem perfectly logical to the person in a manic phase. Sensuality is pervasive and the desire to seduce and be seduced irresistible. The manic-depressive's erratic behavior may seed social violence or sometimes self-destructive behavior. In a few cases, as a result of their actions, they might provoke others to try to kill them, or they may even try to kill themselves.

The symptoms of mania (or depression) mix with each individual's personality and thoughts to create a unique medley that differs from person to person and even from day to day. Since each person has a different stream of memories, these memories will bring on their associated emotions. In the manic phase, irritable and quickly shifting moods are common; shifting rhythms of thinking trigger different emotions. Just as emotions activate associated memories and ways of thinking, speeded-up thinking and certain memories will elicit a sense of elevated mood. The euphoric state in turn will feed back and activate positive thoughts and make bad memories inaccessible. The result: an inflated sense of self, leading to grandiose plans and extremely dangerous behavior to self and others

When manic symptoms are somewhat lesser, the condition is called hypomania. The symptoms last less than four days; delusions and hallucinations are not present; and, in contrast to mania, hypomania is not severe enough to cause marked impairment in functioning. In some individuals the change in functioning may even take the form of a marked increase in efficiency, accomplishments, or creativity.

This period of excitation can last for as little as one day or as long as several weeks. Varying from person to person, the excess excitation will eventually produce a slight deficit of other neurotransmitters as their normal replacement rate can't keep up with the speeded up consumption rate. Levels of serotonin, norepinephrine, dopamine and acetylcholine eventually drop slightly below normal. When this happens, a change in the speed of thinking is the end result. Now, the cortex's signaling rates slow to rates

less than normal. As the neurons decrease their firing, glutamate is restored to a normal level and made available for GABA synthesis. This brings GABA levels back to their normal balance with glutamate and momentarily also slows down the brain. The slower thinking will be associated with negative emotions, and activate them as the brain is locked into a slower mode. Some of these possible emotions are sadness, emptiness, fear, anxiety, diminished pleasure, worthlessness, and excessive or inappropriate guilt. There can be persistent anger and increased irritability, even an exaggerated sense of frustration as negative moods shift quickly.

In terms of the isorropic circuit, the increased levels of GABA (inhibitory) push the activity of the circuit away from neutral calm, into the negative side. The isorropic attractor swings to the opposing side of its manic point.

This slowed-down brain produces physical symptoms as well: facial expressions and demeanor associated with sadness; decrease or increase in appetite, psychomotor agitation (inability to sit still, pacing, hand-wringing, rubbing or pulling of the skin, clothing or other objects) or retardation (slowed speech and body movements; increased pauses before answering; speech that is decreased in volume, inflection, amount or variety of content and even muteness), insomnia or hypersomnia and fatigue or loss of energy. A significant reduction in sexual desire is present occasionally. The slower brain also exhibits diminished ability to think, to remember (mostly the positive), and to concentrate.

However, there is one area (the ventromedial prefrontal cortex) with marked hyperactivity during depression. This area of the brain is responsible for planning and detecting possible future threats as well as participating in other higher mental functions. This area is heavily involved in executive functions and voluntary actions. The hyperactivity results when the brain sends out handshakes to try to determine what is wrong, and, unable to find an answer, locks into this state. This explains the general feeling of anxiety that is commonly reported.

All the present negative emotions mixed up and coupled with the inability to remember and to think produce a feeling of indescribable blackness, of unfathomable emptiness, of interior doom, which adds to a greatly diminished sense of self. In severe depression, this in turn will produce a feeling that is worse than terrible grief. As a result of these feelings of worthlessness and guilt, associated thoughts are triggered that may include negative evaluations of one's worth or guilty preoccupations over minor past failings.

The survival impulses of the organism are diminished in proportion to the loss of self. This greatly diminished or even lost sense of self, in some cases, will produce recurrent thoughts of death, recurring suicidal ideation without a specific plan, or worse, a suicide attempt or a specific plan for committing suicide. These thoughts might range from a belief that others are better off if the person were dead to actually carrying out a specific plan to commit suicide. The frequency, intensity and lethality of these thoughts vary tremendously from person to person, from day to day. Motivations for suicide may include a desire to give up in the face of perceived insurmountable

obstacles or an intense wish to end an excruciatingly painful emotional state that is perceived as having no end.

Again, the thoughts and memories of each person will be uniquely linked to individually varying negative emotions; as a consequence, each person will express a depressed state differently. Our emotions lie at the center of the experience that most of us take for granted, the presence of a well defined, predictable and unique subjective entity we call the "self". When our emotions become disordered, our sense of self comes into doubt; we have great difficulty perceiving the difference between health and illness.

Depression might be mild, moderate or severe. In cases of mild and moderate depression, the person might seem to be operating normally, but this appearance is only through great effort.

In contrast to the depressive phase of bipolar disorder, the onset of unipolar depression is produced independently of a manic episode. The difference is that the balance between GABA and glutamate has gone in the opposite direction. There is a slight decrease in glutamate excitatory action. This decrease will eventually lower the levels of norepinephrine, serotonin and dopamine, but probably not acetylcholine. This might explain the difference in psychomotor agitation (unipolar) and psychomotor retardation (bipolar). It also explains why antidepressants might produce mania in bipolar patients, since a slight increase in activity by any of the neurotransmitters (serotonin, norepinephrine and dopamine) might increase the activity of glutamate, which is present in almost normal amounts. In the case of unipolar depression, glutamate might be at lower than normal levels and a slight indirect increase of it by an antidepressant will help restore its normal level.

The difference between the depressive phase of manic depression and unipolar depression is a small but subtle one. In the former case, depression was brought by a slight lowering of the normal levels of various neurotransmitters through the excessive action of glutamate. In other words the brain crashed. In the latter case, depression (unipolar) was brought by a lowering of levels of some neurotransmitters because of a slight decrease of glutamate excitation; a slowing down of the brain. A small, but important difference: originally, levels of neurotransmitters, primarily glutamate and GABA, will be slightly different. As a consequence, not surprisingly, the pharmacology of these two illnesses should be different. The neurochemical levels and similarities between the two, manic-depression and unipolar depression, in some individuals might be almost indistinguishable at a given time, but for the most part there are going to be important differences between these two. Given the fact that there are many receptors for each neurotransmitter, it should not be surprising that from person to person, the pharmacology would vary accordingly. Much more needs to be done in this important area.

Under normal conditions emotional transference is routinely achieved. Positive affects facilitate the interactive generation of higher and more enduring levels of positive emotions. Particularly in a depressed state, negative affects generate higher and

more enduring negative emotional levels. This in turn leads most people to avoid being around depressed persons. This exacerbates the depression as the person participates less, or not at all, in positive emotional transferences.

Depression might be mild, moderate or severe. In cases of mild and moderate depression, the person might seem to be operating normally, but this appearance is only through great effort. The effort to diminish the negative emotional transfer, particularly with close, loved ones in order to spare the internal generation of awful feelings in them, leads to isolation. The isolation leads to a worsening of depression.

Great effort should be expended to lift the depression. The continued depression, inexorably, almost always, slowly leads to intolerable conditions.

When people suffer a depressed mood for most of the day more days than not, it is called Dysthymic Disorder. Dysthymic Disorder is to Unipolar Depressive Disorder like hypomania is to mania—a less severe form. If mania or hypomania occurs, then the diagnosis would be for Manic Episode or Hypomanic Episode, not Dysthymic Disorder. The diagnosis for Cyclothymic Disorder is used if the manic and depressed moods alternate more than four times in one year.

Dysthymic disorder is probably brought on by the lowering of just one or two of the neurotransmitters associated with depression: serotonin, norepinephrine and dopamine.

Schizophrenia

Schizophrenia and Schizophreniform Disorder are much the same, except for the duration of the disorder. In Schizophrenia the symptoms should be present for at least six months with at least one month of active symptoms. In Schizophreniform Disorder, the disturbance lasts less than a month.

The essential feature of these two disorders is a mixture of two or more characteristic signs and symptoms, both positive and negative. The positive symptoms are the following: delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior. The negative symptoms are: affective flattening (no emotional response), alogia (poverty of speech) and avolition (inability to initiate and persist in goal-directed activities).

I think that the imbalance of excitatory and inhibitory neurotransmitters in this group of disorders is slightly different than it is in Depression and Manic Depression. I believe there exists a complex interrelated balance between acetylcholine and dopamine (cholinergic-dopaminic). The picture that emerges here is a little more complicated than the glutamate-GABA relationship of bipolar and unipolar depression.

The main evidence suggests that in Schizophrenia and Psychotic Disorders acetylcholine release rates are less than normal. This, in general, reduces dopamine levels almost in a direct proportion.

Acetylcholine works on two different types of excitatory receptors called nicotinic and muscarinic. The nicotinic receptors are of fast onset, and of short duration; the

muscarinic receptors are relatively slow in comparison. Dopamine, on the other hand, can act in an excitatory or inhibitory mode, depending on the dopaminic receptors of the neurons. There are five different types of receptors, known as D1, D2, D3, D4 and D5. The most common and predominant are D1 and D2. D1 is the most abundant and is excitatory in nature. D2, the second most abundant, is inhibitory. D3 and D4 receptors are also inhibitory. There is also, to make things more intertwined, some evidence that in some circuits, as cholinergic activity is suppressed, there is an increase in dopaminergic activity. On the other hand, complicating matters, primarily through the inhibitory properties (D2, D3 and D4), a lessening of dopamine in some circuits increases acetylcholine release, and here is where the crux lies.

It has been recently reported (April 24, 2000, UMHSmedia@umich.edu) that the cholinergic system tends to suppress positive symptoms that are exacerbated by an increase in dopaminergic activity, and that the corresponding increase in cholinergic activity then leads to an intensification of negative symptoms. In a series of experiments when acetylcholine was blocked in muscarinic receptors researchers found significantly increased positive symptoms and lowered negative symptoms.

These studies do not contradict each other; they are, I suspect, looking at different aspects of the dopamine-acetylcholine balances.

To begin to understand Schizophrenia, it is helpful to start with Delusional Disorder. When the hippocampus is slightly impaired, instead of doing its normal job of associating sensory information to a context, its electrically fused neurons and circuits can get locked into a certain configuration and keep putting the new information into one single particular context. When it does this, it starts to categorize more and more echoes as being related to the same context, even when originally they had been encoded in different contexts. It has been shown that blocking D2 receptors in the ventral hippocampus impairs memory. Impairment of muscarinic receptors in the brain produces cognitive dysfunctions as well. Also, more dopamine (D2—inhibitory) to the hippocampus leads to more acetylcholine release. This would suggest that lowering dopamine (D2—inhibitory) to the hippocampus produces less handshakes (acetylcholine related) to locate relevant memories and construct contexts; it locks into the particular mode in which it is functioning and doesn't switch as needed. The result is the presence of delusions caused by the wrong belief or context being associated to the incoming sensory information.

In the case of Delusional Disorder, the delusions are non-bizarre, meaning that they could be conceivable in real life (e.g., being followed, poisoned, infected, loved at a distance, etc). Apart from the direct impact of the delusions, psychosocial functioning is more or less normal, and behavior, as long as it is consistent with the delusion, is not odd or bizarre. There can be mood episodes of brief duration compared to the delusions, but the particular train of thoughts brings these on during this imbalance in the brain.

This slight decrease in acetylcholine has an impact on the thalamus as well. The thalamus is receiving cholinergic inputs from the mid brain, but acetylcholine by itself

cannot activate or shut down the neurons of the thalamus. Acetylcholine makes the system more sensitive to sensory input. Lack of acetylcholine slows down the thalamus and this has a direct consequence on the attentional subsystems. In Delusional Disorder the temporal dissociation of the attentional systems produces mild visual or auditory hallucinations. On the other hand, tactile and olfactory hallucinations might be prominent, especially if they are related to the delusion (e.g., infested with insects with delusions of infestation or the perception that one emits a foul odor from a body orifice associated with delusions of reference).

When the hippocampus locks into a context, the sensory information is filtered through this context and is deformed accordingly. The delusions produced by this effect fall mostly into a few categories: Erotomanic (another person is in love with one), Grandiose (having some great or unrecognized talent), Jealous (lover is unfaithful), Persecutory (being conspired against, followed, poisoned), Somatic (problems with bodily functions or odors from orifices), Mixed (various delusions together or alternating) and, of course, a category for unspecified (all others).

Patients with Delusional Disorder may develop irritable or Dysphoric mood as a reaction to their delusional beliefs. This slightly slowing down of signaling in the brain can account for why a Major Depressive Episode occurs more frequently in individuals with Delusional Disorder than in the general population.

If we look at Schizophrenia as a worsening of Delusional Disorder, and we continue to decrease ever so slightly cholinergic activity the symptoms increase. Both the positive and negative symptoms can be explained in this manner.

When the hippocampus slows its release of acetylcholine because of receiving less dopamine (D2-inhibition) beyond the levels in Delusional Disorder, Schizophrenia sets in. In this case the delusions are more extreme and can become bizarre, as the context of processing sensory information is more distorted. Bizarreness, of course, can be hard to judge. Delusions are deemed bizarre if they are clearly implausible and do not derive from ordinary life experiences. The content of the delusions, as with Delusional Disorder, can be of several themes (persecutory, referential, somatic, religious or grandiose). An example of a nonbizarre delusion is the belief that one is under surveillance. Examples of bizarre delusion are a person's belief that a stranger has removed his internal organs or a stranger controls his thoughts (thought insertion by aliens; and, or his thoughts have been taken away from him (thought withdrawal by some outside force). The misinterpretation of perceptions or experiences worsens.

When the thalamus is slowed down enough, the attentional systems become disorganized and uncoordinated. And as we have seen, this produces hallucinations.

Hallucinations may occur in any sensory modality, but auditory hallucinations are by far the most common. They are experienced as voices, familiar or unfamiliar, that are perceived as distinct from the hearer's thoughts. Two or more voices conversing with one another or maintaining a running commentary on the person's thoughts or behaviors is particularly characteristic.

Disorganized thinking ("formal thought disorder") is considered by some to be the most important feature in Schizophrenia. Because of the difficulty in developing an objective definition of thought disorder and how to measure it, in a clinical setting inferences about thought are based primarily on the individual's speech disorders.

It has been established that blocking of the muscarinic receptors exacerbates symptoms of schizophrenia. By inference a lessening of cholinergic activity will allow dissociations of different memory systems, even very closely related subsystems that might be used for thinking and/or speech. Manifestation of this might vary: the individual "slips off track" from one topic to another (derailment or loose associations); answers to questions might be obliquely related or completely unrelated (tangentiality); and, rarely, speech may be so severely disorganized that it is nearly incomprehensible and resembles receptive aphasia in its linguistic disorganization (incoherence and word salad).

Once we understand the cause of the positive symptoms it becomes easier to understand the negative symptoms. Primarily for the same reasons (decrease of cholinergic activity) but affecting the executive function areas in the prefrontal cortex, disorganized behavior might present itself in a variety of ways, ranging from childlike silliness to unpredictable agitation. There can be problems in goal-directed behavior, or the person might appear disheveled, or dressed in an unusual manner (wearing many coats or scarves on a hot day); there might be clearly inappropriate behavior (masturbating in public) or unpredictable and untriggered agitation (shouting or swearing).

This disorganization blocks activation of any associated emotions. The systems that so beautifully activate related emotions to certain thoughts are confused by the disorganized nature of the thoughts, and this is manifested as affective flattening. No emotions mean no emotive responses, and can clearly be seen by the person's face appearing immobile and unresponsive, with poor eye contact and reduced body language. Even though the person might smile occasionally, his or her range of emotional expressiveness is clearly diminished most of the time.

If disorganized speech is related to disorganized thought, alogia might be simply another side of the same coin, manifested by brief, laconic, empty replies. The person with alogia appears to have a diminution of thoughts reflected in decreased fluency and productivity of speech.

Again, if in the prefrontal cortex executive functions are slowed down enough because of a lessening of cholinergic-dopaminic excitation, these areas will not be able to coordinate the information from the different memory systems to plan future actions. This will obviously be manifested as avolition. The individual might sit for long periods of time, show little interest in work or social activities and be unable to initiate or persist in goal-directed activities.

It is of utmost importance that when I say reduced cholinergic activity, we keep in mind the complex web of interactions between the dopamine and the acetylcholine systems. Keep in mind that in some circuits, an increase in dopamine (through

increased inhibition D2, D3 and D4) decreases acetylcholine activity. Also, in other circuits, decreased acetylcholine activity shows increases of dopamine activity, but overall, a reduction of acetylcholine activity means a reduced production of dopamine. The very specifics of each circuit are being researched as we speak and should lead to better and more specific medications.

Suicide

Why do people commit suicide? Like most wars, several elements have to converge to produce a situation conducive to an act so contrary to life and the survival drive. Even though I am enumerating them in a particular order, the first condition is the only one that is truly necessary, although not the only one, for suicide to occur.

First, and probably foremost, a person must experience a loss of the sense of self. This happens when the activity of the isorropic circuit is pushed beyond the ranges of homeostatic balance towards the negative side, beyond the point of depression. This condition is often, but not always, produced during depression and the depressed phase of manic-depression, occasionally in schizophrenia and schizophreniform disorder, and eventually when personality disorders are present. With slowed or disorganized thinking, negative emotions are triggered and brought to the surface. The isorropic attractor is pushed to the negative side in proportion to the intensity of the negative emotions. At the same time, because of the effect of the negative emotions on the brain, certain modes of thinking, primarily all the memories associated with positive emotions, are partially inaccessible. Eventually, the range of choices becomes limited to only one or perhaps two.

The inaccessibility of these memories produces a distortion of the sense of self that generates a condition that skilled writers cannot begin to describe adequately. There are no words for it and the best one can hope for is a good image: I could not find love in myself, a skittering black darkness, had a berth in a pounding hell, a veritable howling tempest, downwards into hell's loneliest black depths. I feel that by combining several descriptions, a better sense of this condition can be conveyed, for example: the hidden, shadowy terror of devouring misery crashed down on me with a clammy chill.

Because I believe it is so important to understand this mental state, I have given it its own special name, idiozimia (from the Greek, idios=self and zimia=loss). And, I have named a partial reduction or diminution of a sense of self, archidiozimia (from the Greek archi=beginning).

Idiozimia produces a constriction of cognition, affect and intellect. As the sense of self is distorted, insight and understanding diminish. It can be perceived, when depressed, as a menagerie of simultaneous negative feelings, which produce in the individual who suffers from it, a torment greater than the sum of each of its parts. The combination of negative feelings is individual to each person, and this makes each individual's experience different. Just as each person has unique memories, so each person will experience depression uniquely. Each individual's thoughts and

experiences are unique, and when they are activated by the negative emotions, will be expressed in special ways.

Given the distorted nature of thinking and memories during idiozimia or archidiozimia, symptoms of the loss of self can be an increased sense of burdensomeness and a loss of belongingness (obviously both related to low self-esteem), and increased pessimism. I repeat, these should be viewed as symptoms of idiozimia, and not as causes of suicide. Idiozimia is what leads to suicide, not its symptoms. This is a subtle but important difference.

A history of a diminution or loss of the sense of self must be the most important predictive symptom of a potential suicide as this is a necessary precondition for suicide to become possible. But archidiozimia or idiozimia is not a sufficient condition either; other elements must be present. Suicidal thoughts might be a manifestation that archidiozimia or a complete loss of self are present but are not in himself or herself proof of anything. Suicidal thoughts could have been caused by a previous idiozimia even if at the present moment the self is intact. The problem, however, is that archidiozimia or idiozimia can present itself abruptly and unpredictably. And the loss of self cannot be judged by outward appearances either. Even individuals who present chronic suicidal behavior are at imminent risk for only limited periods, consistent with the idea that some researchers have proposed (Litmann, 1990.) called the suicide zone.[198] Within the framework I propose, suicide is only possible during the mental state of idiozimia, where the individual becomes disconnected from his or her self. In this sense, suicide is state dependent and suicide intent varies over time fluctuating with stress and other underlying mental disorders.

This is the reason why so many patients with failed attempts express or acknowledge little insight into their suicidality. They suffered a disconnect from themselves during this time. It is probable that only when they return to a state of idiozimia will they be able to remember exactly what happened and what they felt, perhaps, to the exclusion of everything else. If such is the case, it will seem to them, that the time or events between the two suicide events have not transpired. With this in mind it begins to make sense of why people with previous suicide attempts are at the greatest risk. When they fall again into a state of idiozimia, only the memories of previous idiozimias are available; certainly a highly disorienting experience. Also, it is possible that people who have experienced idiozimia without killing themselves, don't remember what happened and can truly say that they have never had any suicidal thoughts or intentions.

Idiozimia, especially accompanied by depression (with its multiple negative emotional states) produces unendurable psychological pain. The urge to stop, or at least move away from the intolerable pain and the unacceptable anguish becomes a primary drive.

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[198] R., Litman, *Suicide What do they have in mind? In D. Jacobs & H. Brown (Eds.), Suicide: Understanding and responding* (pp. 528-546). 1990.

When idiozimia is the dominant mental state it becomes clear that skills such as problem solving, emotion regulation, distress tolerance or anger management become almost nil. Similarly, maladaptive personality traits are a product of the distortion of the self influencing both self-image and interpersonal relationships.

An important affective aspect of idiozimia, because memories of loved ones are nonexistent, is the perception of an eternal feeling of aloneness. It is a state that seems always to have been and that will never end. Some have described it as an experience of utter isolation, accompanied by a sense of unreality, with a quality of emptiness, horror, devastation and denigration.[199] The idiozimic person does not take others into consideration, for the simple reason that most memories of others are inaccessible.

Idiozimia can also occur during other conditions, but generally is associated with extreme emotional states. For example, a loss of self can be experienced during extreme danger, as transpires occasionally during war, when a soldier attacks the enemy with disregard for his own safety. Idiozimia also ensues during extreme anger, when attack, or assault or death of others can be the result, especially in acts of self-defense, or defense of loved ones. The term legal insanity could be defined better using this concept. This would address the issue of at what point does an individual lose contact with him or herself and stops being responsible for his or her actions.

Idiozimia, in a similar fashion to the fight-flight response, is accompanied by a lack of sensitivity to physical pain as well as no fear of pain. Both of these conditions are conducive to possible self-harm, or at minimum, conducive to disregard potential injurious consequences of one's actions.

A second element is suicidal thoughts. The state of archidiozimia allows suicidal thoughts to emerge which otherwise would be unthinkable. The possibility of inflicting damage to oneself becomes less illogical. There is a huge gap between suicidal thoughts and acting on them, but once the thoughts are part of a possible solution, the gap becomes smaller. The continued intrusion of these suicidal thoughts slowly makes them seem more possible, more likely. A plan might emerge; this is a real danger sign. The tools or methods for suicide might be perfected or put into place; a very serious sign. At this point, if idiozimia is present, it might just be a matter of time.

Third element: the slow vanishing of inner strength. The intense suffering that accompanies depression and some combination of symptoms in schizophrenia and schizophreniform disorder can be so acute that ending one's life becomes a real alternative. The effort to stay alive in the face of extreme torment is emotionally exhausting and seems insurmountable. The struggle to continue a semblance of normalcy uses up all possible energy; only in the mildest or very moderate cases can this outward appearance be achieved to begin with. Hardly any energy remains to do anything else. The desire for calm and peace might be overwhelming but the effort

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199 R.W. Maris, *Assessment and Prediction of Suicide*. 1992.

necessary to achieve this seems elusive and impossible. The strength to continue living erodes slowly until death seems a welcome act.

Fourth element: hopelessness. This one was mentioned repeatedly before. The suffering becomes so great that the situation starts to feel hopeless. In tandem with the hopeless feeling comes the feeling of being helpless. Relief from the pain seems beyond one's grasp. That the suffering might eventually subside does not seem possible. Slowly, death becomes a longed-for relief. The renewed negative feelings, after having enjoyed a respite, can spiral out of control. Disappointment and frustration add up to all other negative feelings. Perspective on the nature of the pain is lost, magnifying its intensity like a long felt toothache pushing the individual slowly to a form of insanity. The seeming hopelessness of the situation needs to be obsessively ended. A quick solution becomes imperative. The wrong solution, if continued existence is considered a desired goal, is quickly equated with death. However, if ending the hopelessness-helplessness of relieving the suffering is considered a necessary goal, death becomes a logical act.

Fifth element: damage to the hippocampus. Because of prolonged and intense stress, secretions of glucocorticoids damage the hippocampus. The hippocampus will store and retrieve the memories of all related objects to the event, in this way creating a context. When the hippocampus is damaged, the emotional context will be perceived wrongly. The negative emotions, put into the wrong context, will exacerbate the effect of the first four elements.

It is important to consider the effects of the dopaminergic afferents to the nucleus accumbens. The presence of dopamine on the nucleus accumbens from the afferents from the ventral tegmental area in the brain stem reinforce desires by sending dopaminic signals to the prefrontal cortex (via de thalamus). The hippocampus' projections to the nucleus accumbens promote or define needs (or aversions) by providing a spatial or social context, whereas the amygdala's projections (because of their emotional component) create or reinforce beliefs that guide responses.

Sixth Element: the aftoktonic switch. Within the context of the last four elements, or because of their repetitiveness in past experience, the echoes streaming from the cortex reinforce and increase the chance that the thalamus will match incoming signals with external sensory signals and erroneously, but automatically, trigger the respective emotions linked to hopelessness and vanishing strength and activate the related suicidal thoughts. Once this happens, the amygdala will signal the nucleus accumbens and amplify these, and turn the suicidal thoughts into a belief. Simultaneously, the hippocampus signals the nucleus accumbens and increases the urge signaled from the ventral tegmental area. With a stressed, damaged hippocampus, the hippocampus and amygdala signal, respectively, an urge to end life within the context of hopelessness, and a belief that death is welcome. This binary combination becomes an unbearable urge to commit suicide by whatever means available. This is what I call the aftoktonic switch. Suicide is not a random, purposeless act. To the sufferer, under these conditions, it becomes the only available solution to his or her problems. At best, the need to stop

consciousness to end unendurable pain is irresistible, which leads to a suicidal act. When idiozimia is present and the aftoktonic switch is triggered, only one overriding, imperative, single-minded urge to end it all is inevitable, as all other alternatives become inaccessible.

There are many pointless deaths, but suicide is never a needless act. More often than not, during the act of suicide, it is perceived as the best and only possible response to alleviate the excruciating situation.

Increased glutamatergic action in the raphe nuclei increases serotonergic activity. The nucleus accumbens, through its serotonergic afferents, will respond automatically by lowering the threshold for impulsivity/aggressivity, making it more likely that these behaviors will be enacted. If this is coupled with a low 5-HT serotonergic activity related to low harm avoidance, then this neatly explains the well-known facts that improvements in depressive symptoms correspond to rising levels of serotonergic activity and that suicidal behavior increases with an improvement in depressive symptoms. Under certain conditions, this would also explain the increased risk of suicide with use of SSRI's, especially in the beginning of treatment of people under eighteen years of age.[200]

Even though the absolute risk is low, SSRI antidepressants were associated with nearly a fivefold risk for suicide for people over 66 during the first months of treatment, even after adjusting for depression and psychiatric care, and were often more violent in nature.[201]

Seventh element: this is so important that I have given it a special name, the phobothymic switch (from Greek, phobos=fear and thymos=wrath or anger). This element might be independent of the second, third, fourth and fifth elements just described. It is common that depression or schizophrenia is accompanied by anxiety or panic attacks. Depression and schizophrenia are states that clearly indicate that something is wrong, and the fear and anxiety associated with these states could be normal, healthy responses to a dangerous mood disorder. In order for the amygdala to build up a fear response the prefrontal region must be shut down, otherwise the prefrontal lobes signal the amygdala and fear slowly dissipates when danger is not present. However, when the amygdala is completely unchecked by the prefrontal lobes, the fear escalates and a panic attack ensues. An unbearable inner terror builds up.

The first step of the fight-flight response is freezing, a strategy that helps fool predators into thinking that you are dead, or makes it harder for them to detect you. At this moment the phobothymic switch can go either way, fear or aggression is equally possible. Freezing also provides a small amount of time to evaluate the threat and determine to flee, attack, or simply return to a resting state: false alarm. This state feels strangely both like anger and fear, a sort of physical prickling sensation down the spine.

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200 Roy-Byrne Peter, *SSRI's and Suicide Risk: A Concern for Adults, Too?* March 9,2005. Journal Watch Psychiatry.

201 Thrash Barry, *SSRI's Linked to Short-Term Suicide in the Elderly.* 2006. HealthDay News.

When the anxiety attack or the fear escalates into a full-blown panic attack, there is a moment when suddenly the phobothymic switch is activated: the flight mechanism switches to a fight response. The uncontrolled fear turns into uncontrolled aggression. In this aggressive mode, the perceived threat, in this case an irrational panic, which is translated to a feeling of imminent death, is immediately confronted with an anger attack. A confrontation with the biggest known fear, imminent death, becomes not only logical, but seems the only solution, the only way to win, the only way to survive. When the tiger has chased us into a dead-end canyon and there is nowhere to flee, we inevitably turn to face it and fight it to the death. There is no other choice. In a normal, healthy situation, at some point the threat is evaluated (not rationally, there is no time) by the cortex and determined that escape is impossible, or conversely, that the threat can be removed by quick action.

Uncontrolled aggression is channeled against the source of the threat (the panic attack), which is being generated by one's own brain. Suddenly, extinguishing the root of this threat becomes absolutely imperative. An attack against one's self, suicide, is the focus of the aggressive behavior. It is an experience of relentless scorn against one's self. It is easy to try to see this as a form of self-hate, but aggression is more in line with this behavior.

At some point the amygdala receives a signal from the pre frontal cortex, and automatically the response changes from flight to fight mode; from fear to anger; from a panic attack to an anger attack. It is two sides of the same coin. The phobothymic switch is an elegant engineering solution to automatically and quickly change the strategy for survival; at some point, the organism is better off fighting than fleeing.

The question then becomes when, not if, the panic turns into a fight mode. In this particular instance, when the individual is also experiencing idiozimia, or even archidiozimia, this switch into an aggressive mode becomes lethal. Each individual, according to his or her experience, will have a different point at which he or she will go from a flight mode to a full fight response. This point is probably impossible to determine in advance. Being young and male probably increases the probability that this will happen. I suspect that the stronger, mentally and physically, an individual is, the lower the threshold for the panic attack to switch to aggression.

The phobothymic switch described is probably the reason why we lose so many young with no forewarning signs. They, themselves are not even aware that this could happen. The first time they experience this is their last. If they survive this first experience, because of some distraction or interruption, they will then move into the category of potentially suicidal. Most of us don't know how we are going to react to a huge threat we have never encountered before, until we are faced with it. Similarly, the phobothymic switch will flip over to attack mode during a panic attack without warning. When this happens, the greatest possible type of aggression, the suicide, lashing out and killing the source of danger, killing oneself, the cause of the panic attack, is a response that the victim hadn't even contemplated before.

Out of the despairing darkness, a calm collected anger overcomes the individual as he embarks on facing the greatest fear, death itself. He or she fights his last battle and wins. But by winning can never rise again to fight.

There are two separate, but related conditions that lead to a suicide attempt: one through the aftoktonic switch, i.e., the combination of a strong urge with a strong belief to end one's life; the other, through the phobothymic switch, where uncontrolled aggression is directed to eliminate the source of a perceived threat (i.e., the source being the self that created the panic attack). Firsters and most completers experienced the fifth and/or sixth element alongside idiozimia, i.e., their brains triggered the aftoktonic and/or phobothymic switch during a loss of the sense of self, whereas most attempters only experienced a combination (or all) of the first four elements described. When both conditions are experienced simultaneously alongside a state of idiozimia, or even archidiozimia, suicide is truly the only possible response.

In consideration of the aftoktonic and phobothymic switch, any knowledge of methods to die or competence regarding suicide, will dramatically increase the lethality of a suicide attempt. It is in this sense that previous suicidal ideation, or plans to execute a suicide can have any meaning regarding suicide risk. It is in this sense that familiarity and availability of guns would lead to use a gun to commit suicide when either of these switches is triggered. Similarly, knowledge of bungy-chord jumping could lead to the use of a rope to hang oneself. It is not necessarily a thought-out choice, simply the only choice available in memory that can be used with lethal intent.

Once idiozimia is experienced, it can be experienced again, perhaps at lower thresholds. This is why previous suicide attempts are one of the most important markers for suicide risk. If you have been there once, you can be there again. The only memories available in this state would be the ones relating to these previous (idiozimic) states of mind, creating the sensation that life is a continuous idiozimic state, a completely empty and disorienting experience or even worse, the only memories available would be all previous suicide attempts; life would seem a long, continuous attempt to kill oneself. Not from a rational, judicious sense of failed attempts, but of one continuous, necessary, overwhelming belief that death is imperative, that death is the only course of action. When the aftoktonic or phobothymic switch has been triggered previously, similarly, the threshold would be lowered to newly trigger these switches in the future. This explains why as episodes of suicidality increase, their relation to external triggers decreases, and their severity and lethality increases.

In some instances, mostly in the case of women, the choice of method for suicide is benevolent enough that the chances for surviving the attempt are greatly increased. Women in general choose less violent physical means to end their lives, increasing the number of suicide attempts in comparison to young men who in turn have a higher suicide rate than women.

Suicide Autopsy

In the preface to her book *My Son . . . My Son*, Iris Bolton, believing that self-destruction is unexplainable, laid out the dilemma that she and all parents who lose a child to suicide face:

> *I don't know why.*
> *I'll never know why.*
> *I don't have to know why.*
> *I don't like it.*
> *What I have to do is make a choice*
> *About my living.*

There are, broadly speaking, two approaches to the study of psychological phenomena. One is called longitudinal and one cross-sectional. Longitudinal studies are concerned with information over time—objective behavioral items over a lifetime, such as previous suicide attempts, parents' divorce, setbacks in early life, and so on. In the case of mood disorders and suicide these are traditionally accepted as prodromal clues or premonitory signs. The cross-sectional approach is concerned with the "now," or perhaps as long ago as a month. It asks what is going on (now)? How much do you hurt now? Its approach is more clinical, introspective and analytical.

In many suicide cases, particularly when there has been little or even no warning, only an "after the fact" analysis can be done in order to attempt to explain or understand why it happened. This process was termed a psychological autopsy by E. Shneidman. An attempt is made to reconstruct a person's life, their thoughts, feelings, and behaviors during their last weeks, through interviews with friends, family, and physicians, sifting through relevant writings, and so on, in order to attempt to explain the circumstances leading to their self-destruction. In this sense, a psychological autopsy is a cross-sectional study. Still, in any specific case, given the limitations on current knowledge, it is understood generally that no one can deliver an authoritative conclusion about why a suicide occurred.

I wrote a book about my son's suicide to explain his (and hopefully all) suicide(s). In my book *A Thousand Moments of Solitude,* I presented autobiographical material—my son's and mine—interwoven with an explanation of the specialized vocabulary that psychiatrists use and the symptoms used to define psychiatric illnesses, along with a social critique about how society is unprepared to deal with suicide, but more importantly, my personal brain theory (to put suicide into a scientific framework),

presented here, as well as fiction (which allowed me to explore suicide under differing conditions) to come to understand suicide in general. In certain ways, the book is a suicide autopsy. I wrote it for many reasons. Chief among them, I thought many parents of suicide victims would be interested in an explanation of their child's death. However, the few parents of suicide victims I have met personally, even after receiving a free copy of my book, did not read it. My impression is they have made peace with the fact, one way or another, and the pain of revisiting such a traumatic event is not worth reading about a "new" explanation. I was hoping they would give me valuable feedback, but they have made a decision, in Iris Bolton's words, about their living.

The original purpose of the psychological autopsy was to aide the coroner of Los Angeles County in making decisions about the mode of death in death certificates (whether death was natural, accidental, a suicide or a homicide). In the context of this book, and for many, a suicide autopsy can be the only way to attempt to explain how and why a suicide happened. Shneidman, in his book, *Autopsy of a Suicidal Mind* presents such an exercise in a specific case. He looks at an extensive suicide note (written over the weekend) of a 33 year-old suicide victim, whom he calls Arthur, and interviews the mother, father, brother, sister, ex-wife, ex-girlfriend, best friend, psychotherapist and psychiatrist. Arthur studied medicine, unsatisfied with his life he became a lawyer, and ultimately killed himself with an overdose on a Sunday night after having breakfast with his father and spending the day with his best friend. Shneidman sent all this material to eight prominent suicide experts and asked them to perform a psychological autopsy and render their opinions of why suicide happened.

The conclusions reached in a psychological autopsy will, inevitably, reflect the beliefs or premises of the ones performing it. I want to emphasize that I have great respect and admiration for all these experts. They have labored more than anyone to attempt to bring help, solace and improve the lives of countless sufferers of mood disorders who are suicidal. I present this material, not to criticize anyone's conclusions, but to show how difficult the subject of suicide is, and how far ranging the ideas and theories relating to it are, even among the topmost suicide scholars.

Without going into the details of Arthur's suicide, the responses of each different expert show the difficulties in explaining suicide without a fully coherent theory of the brain.

After Arthur's death, his psychotherapist, who treated him as a child, as an adolescent, and as an adult, concluded that the suicide was the result of a biological, physiological vulnerability, compounded by the difficulties of his early family life.

Arthur's psychiatrist, who treated him while he was in medical school, knew from the beginning that he would do himself in. In the suicide autopsy interview he stated clearly, "Well, [Arthur] he comes in and tells me that the night before—he is very articulate—he was sitting in a chair, he wrapped cellophane around his face multiple times to suffocate himself, and the burning in his lungs got so bad that he ripped it off because the pain was so terrible; and that what he was planning for the night that I was seeing him was to repeat the cellophane, but he would duct-tape his arms to the

chair so that when the pain happened he wouldn't be able to extricate himself. I didn't think he would kill himself then, but I knew he would kill himself. I knew it 100%."

When the psychiatrist was asked, referring to Arthur's depression, *"He knew that he had come out of it?"*

The psychiatrist responded, "Its clear that he would have said: 'Of course I would come out of it, but I don't want to be there again, and if I have to be there again, I would rather die.' An he was clear on it." In answering another question the psychiatrist added, "He was petrified of his disorder and returning to it, and that it meant a model or scheme of his life invaded his thinking at all times."

The psychiatrist believes that some cases are malignant and nothing will ultimately save these patients. "This guy had it bad," referring to Arthur's suicide tendencies, "he died from it, and we do the best we can; and I am telling you this was the clearest case I have ever seen. He was persuaded that if it comes back he was going to kill himself." The psychiatrist finished the interview with the following words, "And I really felt, all we were doing was buying time. I think my role, and I believe this in my heart, is to fight as hard as I can against the patient's wish to be dead, and that's what I'm supposed to do."

Morton Silverman, who among the many titles and positions he has held related to suicide, is the editor of the scientific journal of the American Association of Suicidology. Silverman, who's report is presented first, focuses on the suicide note and on the fact that Arthur wanted to have the last word, which allows the decedent [Arthur] to explain, to bring closure (or not), to assuage guilt, to dictate next steps, to control, to absolve, or to blame. He then analyses some contradictions in the suicide note.

Silverman looks at the typical background that might be associated with suicide: Arthur was bullied in school, had a learning disability, poor school performance (until high school); clear cognitive, physical and psychosocial developmental delays; severe behavioral tantrums until age 12; selective eating; parental divorce at age 10; rigid maternal nurturing; at least one suicide attempt; psychiatric inpatient hospitalization; early psychopathology with psychotherapy from ages 7-15; mother, grandmother and brother with depressive episodes, divorced, etc. Silverman, however, accepts that all this is not enough to explain his suicide.

Further on, Silverman looks at many subtle aspects of Arthur's life through the eyes of those who knew him. I summarize, for brevity, what jumped out at me:

This man [Arthur] seems to have been on a mission to find identity, peace of mind, emotional stability, a sense of predictability, and security.

Arthur became a doctor and then finished law school. He was quite an accomplished man. For some reason, Silverman surmises, Arthur was unable to store for an emotional "rainy day" all the loving, caring and support that were directed at him . . . He died after a weekend of struggling with his emptiness, fears of failure, and sense of abandonment.

Silverman presents a psychiatric perspective, at the end of which he admits, "I would not convey that I could take away his pain or his susceptibility to repetitive

depressive episodes nor that I could relieve him of his pervasive negative views . . . His psychiatrist in medical school described him as having a chronic psychiatric malignancy. With his [Arthur's] permission, I would have tried to engage his girlfriend in couples therapy and his family in family therapy. Group therapy might have offered him a chance to see that others suffer as well and struggle to stay alive. Another attempt at medications was indicated. Electroconvulsive therapy might have been of great help and provided relief.

"Yet, had he not been protected from a premature death? He was sustained for many years by a dedicated psychotherapist and a perceptive psychiatrist, who offered him hope and provided caring and respect. He lived for thirteen years beyond his first serious suicide attempt."

Silverman finishes with a final note, where in part he writes, "What I find most tragic about this case is that here we have a very bright young man, well educated and well trained in two demanding professions, who had available to him multiple resources, as well as much knowledge about treatment modalities and alternatives. That in the end, he chose to reject options available to him (such as inpatient or outpatient electroconvulsive therapy, psychiatric treatment, and medical consultations for alternative chemotherapy) is a tragedy. This is the case of a young man searching for authenticity, identity, and individuation. Every time he entered situations that allowed him a taste of these things, they weren't sustainable—either due to circumstances or to his inability to adjust to them.

"Arthur knew the pain he endured daily and feared the pain that was coming. He was too 'smart' for his own good. And therein lay the ultimate tragedy, the ultimate enigma, and the ultimate paradox."

Robert E. Litman's (who among his many degrees, titles and positions, at one time was president of the American Association of Suicidology) psychological autopsy is presented second. He begins with a brief review of how he and his colleagues at the Los Angeles Suicide Prevention Center, conceptualized suicide as a crisis, and their treatment model was a crisis intervention. They thought of the presuicidal state as one of transient perturbation following trauma or serious loss. They emphasized helping people get through these time-limited stress periods so that natural healing would occur.

Litman admits that the suicide mortality rate of people that called for help is about 1 per cent after two years. Psychological autopsies revealed that, when these (now deceased) patients called, they had been chronically psychiatrically ill and chronically suicidal. He goes on to state that, "Their suicidal behaviors did not represent a crises as much as they represented repetitive behaviors. The most effective intervention was to help these people find a stable and continuing treatment resource and encourage them to stick with it."

The line that best illustrates how Litman sees Arthur's pain is, "There was a profound narcissistic grandiosity in his way of thinking. When Arthur said to his estranged girlfriend that she must come back to him to love him or he would kill himself, he

illustrated the mind-set of the multitalented suicidal person: 'My way or no way.'" In other words, a lack of absolute control over the environment, and an inability to accept this is what leads to Arthur's suicide.

Jerome Motto, also at one time, president of the American Association of Suicidology, is third. Motto reviews quickly "The Event," and then "The Problem," which essentially is how to understand the source of that motivation, the seemingly ever-present experience or anticipated recurrence of an excruciatingly painful emotional state—graphically described [by Arthur] to his sister "as if someone was hammering nails into him every second into every part of his body" and to his girlfriend "like lying on a bed of needles." Such a complaint by a suffering person is so unusual that it presents a unique challenge to find a plausible explanation for it, a challenge made no easier by the fact that the experience apparently dated from very early childhood.

Motto presents a brief etiological perspective, in which essentially he suggests that Arthur might have suffered autistic disorder. This would explain delayed speech, aggressiveness and temper tantrums. An oversensitivity and exaggerated reaction to sensory stimuli—touch, sound, light, odors—is also described, as are abnormalities in eating, such as limiting diet to a few foods.

Clinicians have focused on the hypersensitivity to stimuli to explain why autistic individuals may try to relate to the world about them but repeatedly withdraw from it again. This reaction is attributed to the world being too stimulating, and, because they are excruciatingly sensitive to stimuli, they are forced to retreat. This extreme sensitivity has been termed *sensory integration disorder*. One aspect of the sensitivity to touch has been expressed thus: "Imagine yourself in clothes so irritating that they seemed lined with metal scraping brushes." One [Motto] wonders if this sensitivity is related to some of Arthur's descriptions of his pain.

Motto concludes the etiological perspective, "However labeled, it is clear that Arthur's lifelong pathology was paired with some remarkable strengths. His intelligence, and insight were clearly demonstrated, and, in the course of his valiant struggle to overcome his disorder, others described him as tough, articulate, compassionate, funny, sweet, big-hearted, hardworking, loving, nurturing, a really good man, and a mensch. This may be considered ample evidence of a life well lived, however short and painful."

In Motto's section of "A Clinical Perspective," he states, "A basic concept in treating suicidal persons is that a suicidal act can be expected if the person's level of psychic pain—or anticipated pain—exceeds the person's threshold of pain tolerance. Thus the pain level and the pain tolerance thresholds require ongoing monitoring, as both are prone to fluctuation." He goes on, as he mentions emotional fatigue as a critical precipitant of suicide, "If not relieved, a stress that was long coped with in the past can generate increasing fatigue, which gradually reduces the pain tolerance level till it is exceeded by the pain level; thus a suicide occurs with no visible warning."

Another element in Arthur's situation, Motto continues, is the possibility that his autistic disorder prevented the introjection of meaningful relationships that provide an essential stabilizing influence on emotional life.

I summarize Motto's conclusions as, "there is no way to know whether Arthur's suicide might have been preventable." It might have been delayed as many who knew him suggested.

The fourth consultation presented is by Norman Farberow, who has also been president of the American Association of Suicidology, but has spent most of his life as a director of the Los Angeles Suicide Prevention Center.

Farberow begins by answering the question "Why did Arthur kill himself?" According to Farberow, Arthur's suicide was the result of a confluence of psychological factors with neurobiological and physiological factors. The most prominent of the psychological factors was a self-image in which he saw himself as inadequate, incapable, weak, and inferior. He explores this concept in some detail.

Farberow reminds us that Arthur had made a number of suicide attempts in the past, some of them quite serious, with the degree of ambivalence in them varying considerably. More recently, it seems that the level of his ambivalence had dropped and his attempts had become more lethal. At least one was close to fatal.

He then reviews Arthur's family role in his suicide. Of note is the following reference to Arthur's father's (who is a physician) understanding of his son's suicide, "He is somewhat simplistic in attributing suicide to four basic factors: chemical or something inherent; diet, because Arthur ate a limited variety of foods; physical, because Arthur was weak and scrawny as a child; and treatment, because his therapists did not do enough to keep him alive. On the other hand, he was aware of the degree to which Arthur felt he was a failure and suffered the constant fear of being exposed as a sham. He (Arthur's father) reverses the process by which suicide occurred by attributing the feelings of loneliness, inconsequentiality, pessimism, and fear of failure to Arthur's depression instead of recognizing that it was those feelings that were the basis of his depression."

Farberow goes on to imply that the suicide might be a result of a neurological or physiological disability that led Arthur to believe that he was weak, underdeveloped, uncoordinated and was an unredeemable failure. The only road to escape such a failure was to kill himself. Farberow recounts how members of Arthur's family may have not expressed it, but believe that Arthur's suicide was inevitable. His father, sister, and brother believe it was "chemical"; his girlfriend and physician believe it was a "psychiatric malignancy." His psychotherapist feels that a biological explanation was most important and that biological factors were exacerbated by the difficulties he experienced in life. His best friend is the only one who believes that perhaps more positive experiences or relationships might have helped.

Farberow goes on to emphasize that, "One is not born with a poor self-image; one is taught that, in the same way that good self-image is taught. The capacity for self-love, adequate self-image, and self-confidence is imparted to the individual by the major figures in his rearing." He then adds revealingly, "One may be born with neurophysiological defects, but assuming there is adequate intellect, they can be overcome, so that suicide is not the only and inevitable resolution," implying strongly that low self-esteem is at the root of Arthur's suicide.

Farberow ends by stating that Arthur could have been saved. But it would have required a therapist who firmly believed he was worth saving, that restructuring Arthur's self-image was possible, and that Arthur was worth the investment of the effort, the time and the energy, along with medication to help him over the inevitable rough spots.

John Maltsberger does the fifth psychological autopsy. He is a past president of the American Association of Suicidology, and at the Harvard Medical School, is associate clinical professor of psychiatry.

Maltsberger feels metaphorically that Arthur was somehow doomed from the start, that he was "star-crossed," or under a malign and inexorable influence. He was pursued across his life by unremitting mental anguish. There was no escaping it, until in desperation he killed himself. More recently psychoanalysts discerned the operation of murderous introjects taking possession of the minds of intractably suicidal persons. The up-to-date among us today attribute such evil mental dooms to biology.

Disorder brain biology has its part to play in suicide, but the more immediate, proximate cause of the suicide we have at hand was ineluctable, unendurable anguish. When no succor comes, the desperate sufferer turns to death for the only possible relief from "psychache." Maltsberger, later goes on: Something was clearly wrong with the way his brain worked, and we know that children with this kind of social difficulty are particularly vulnerable to major mental illness. Maltsberger accepts that the causes of these child abnormalities are obscure. He conjectures about the possibility that brain-injurious effects of viral infections during pregnancy or anoxia at the time of birth might have played a role. He goes on to point out that many children are born irritable and high-strung, but, given ameliorative circumstances, can in the course of development learn to moderate their moods and quiet themselves down.

Maltsberger continues, "In adolescence, Arthur's tantrums and rages slowly appeared to have turned around, no longer taking the outer world, but the patient himself, as their target. As a small boy, he raged against his mother because she didn't offer him perfectly what he wanted. As a man, he scorned himself because he was not perfect."

The tragedy of the story is plain: Arthur went through life looking for something, anything, from outside himself, to help moderate his intolerable feelings—but to be acceptable it had to be perfect.

Maltsberger indicates that the pharmacopoeia available for depression had by no means been exhausted. Neither is it clear if the doses were correct for adequate periods. If he could not tolerate lithium salts, other mood-stabilizing drugs *might* have helped. Rounding off some of the options, Maltsberger offers, "Electroconvulsive treatment might have made a difference, although it would not have been a permanent solution. There are those who would advise a neurosurgical approach, though I have known patients who believed suicide a better option. (I have met some clinicians who think that, too.)[202]

[202] Author's note: Maltsberger has met clinicians that agree with that.

Maltsberger concludes, "My experience with patients like Arthur makes me mistrust split therapies, with one person in charge of drug prescription and another in charge of psychotherapy." However, Maltsberger didn't see any evidence that in this case the split treatment resulted in Arthur's suicide.

The sixth consultation is done by Ronald Maris. He is a sociologist and one of the outstanding scholars of suicidology and for years he has been the director of the Suicide Center at the University of South Carolina.

Maris believes that a series of factors converged in this case. Maris points out that Arthur raged at the world, at life itself. Not only did he fight with his mother, he fought his older brother, his father, his pal, his teachers and schoolmates. He admits that the rage might be biologically based, but eventually, rage tends to turn back on oneself.

Added to his depression, Maris explains "suicidal careers," where Arthur's fragile coping crumbled with repeated depressive episodes, repeated psychic pain, repeated interpersonal failures, and repeated suicide ideation and suicide attempts. His suicide risk gradually summated until a pain and suicide threshold was non-dramatically (if not this day, then some other) breached. Maris also mentions low serotonergic levels found in animal studies including impulsivity, disinhibition, sleep difficulties, pain-proneness, conduct disorders, mood volatility, poor peer relationships, and suicidal behavior (it sounds like Arthur to Maris, I guess).

Additional factors were Arthur's high expectations of himself and others, the "nothing is good enough" attitude and his inability to accept love.

The fact that Arthur wrote a lengthy suicide note, did not get drunk or use a gun, among other features, suggests to Maris, that Arthur's suicide was different or unique in ways that hinted at interventions. He briefly discusses ethical issues such as how long and how forcibly can you keep someone alive against his own wishes. Maris concludes that perhaps if Arthur could have bought some time, the edge would have come off his exquisite psychic pain. But, perhaps not.

The seventh psychological consultation is by David Rudd, currently president of the American Association of Suicidology. He has written more than seventy publications on cognitive therapy and on treating suicidal behavior.

Rudd begins by reminding us of the tragic loss of the preciousness of life. He clearly notices Arthur's persistent suffering. He identifies a new psychache, the one manifested by the pain of the suicide survivors in their interviews.

Rudd presents a special perspective, one founded in cognitive theory but bolstered by Murray's notion of psychological needs and Shneidman's construct and theory of psychache. Cognitive theory advances the idea of modes. A mode refers to suborganizations within the personality that incorporate the cognitive, affective, behavioral, and motivational systems of the personality. These component parts of the mode are interactive and interdependent. In this context, there is a suicide-specific mode, which incorporates the individual's suicidal beliefs dominated by hopelessness. In short, the suicidal belief system is a means of verbalizing the individual's

psychache. Within the suicidal belief system, identifiable psychological needs have been frustrated.

It is perhaps easier to understand suicide, Rudd continues, from a linear time perspective, moving from a triggering event (internal or external) to activation of the belief system, concomitant emotional and physiological response, and behavior that facilitates (or impedes) the patient's suicidality. Aside from hopelessness, Arthur's suicidal belief system has four central themes: (1) unlovability, (2) helplessness, (3) poor distress tolerance, or psychache, and (4) perceived burdensomeness.

Summarizing Rudd's words: From a cognitive standpoint Arthur came to believe that his suffering was intolerable and unremitting and consequently he was a burden to others. Arthur was clearly hypersensitive. It is obvious that Arthur's parents identified a problem and sought out care and treatment since an early age. It appears that Arthur himself reached the same conclusions as his parents, that he was deficient in some way: unlovable (in spite of evidence to the contrary), and a sense of helplessness (persistent dysphoria and anhedonia). Arthur seems to latch on to a biological explanation, acknowledging little control or influence on his depression.

Rudd questions Arthur's understanding of his suicide belief system and the cyclical nature of his suicidal behavior. Did Arthur recognize that there were identifiable triggers, many of which he could influence and target in therapy?

Rudd concludes that Arthur's suicide could have been averted, provided he had received competent and appropriate care. Suicide is about loss. Cognitive therapy for suicidality is about loss of perspective and understanding. Rudd muses at the end, "that, as with every suicide, hindsight is twenty-twenty."

Avery Weisman, at the age of 90 years, does the eighth consultation. He is currently retired, but in Shneidman's mind, he is the dean of American Suicidology.

According to those who should know, Weisman begins, Arthur talked about his inevitable suicide from childhood on. This does not imply that he was depressed from childhood on, even though he was in psychotherapy for tantrums and so forth at an early age. Later he was socially isolated, but then at some point he became very popular, with many friends.

A few paragraphs later, Weisman continues, "Although Arthur was convinced about his grim fate and made numerous suicide attempts, the record of near misses is peculiar." This in reference to Arthur's knowledge as a doctor and his botched first suicide attempt on that final weekend.

I try to summarize some of Weisman's ideas about suicide in general, and in Arthur's case in particular: The immediate precipitants of suicide are almost as inaccessible as the private thoughts and feelings that push a reluctant person over the edge. Why anyone kills himself or herself at a specific time is better not asked; it is tricky, enigmatic, and futile. In his clinical practice with patients of all sorts, the "why" question should never get an answer and should be understood not as a real question, but as an entreaty, appealing to a higher source for justification, such as, "Why me?" and "Why oh why?"

Weisman goes on and admits, "I like to believe that with collective experience, we can distinguish objective from subjective information, at least most of the time. Psychological facts are, after all, not so much facts as low-level theories."

Weisman moves to explain his ideas on suicide evaluation. Assessing a suicide, before or after, involves two major considerations: risk factors and rescue factors. Numbers place Arthur in a large group of potential suicides, even if he sits alone reading a newspaper in a dingy hotel lobby. However, a past history of suicide attempts, along with admission of depression, might be taken most seriously as a potential precipitant, especially if he talks about being better off dead. The continuum of risk factors ranges from impersonal correlates to private intrapersonal dejection and suffering. Timing is only an approximation.

Weisman asks, "Must every suicide be preceded by depression? Or, given the relative success of antidepressants, do clinicians presume that there is some degree of depression behind every attempt? I yield to those with up-to-date skill in medicating depression and potential suicide. I beseech them to retain a regard for those of us born earlier who had little to offer besides a strong intention to preserve life."

Weisman wonders about Arthur, "Arthur's risk factors scarcely need repetition. His rescue factors include his significant others, Arthur's competence and undoubted success, and two skilled psychiatrists, one of whom was available for years and the other for the crucial period before death.

"One possible rescue factor was Arthur's unwillingness to take a lethal dose of whatever he ingested; he seems to have "failed" to die several times. This was an escape condition that he didn't take advantage of in his final attempt. He certainly knew the toxic amount, given his professional experience. Another rescue factor was Arthur's abundant qualities that, just possibly, he could have used to reinforce the courage to cope and find meaning in his existence.

"Although I am sure that Arthur's psychiatrists were competent and conscientious, Arthur was asked about hospitalization and ECT, only to refuse. Although he had attempted suicide by tape and asphyxiation just the night before, further avenues were discussed with him as if he was a rational colleague."

Weisman concludes, "With a word or two about the courage to cope with vulnerability in therapy. Quite apart from the antidepressants, which would help to a degree, learning how to cope effectively and to grapple with misfortune can be accomplished, even allowing for qualified skepticism. It is one thing to hope, but hope needs help and should be worked for. Despair needs help, too, with every means possible, when dealing with the enigma of suicide."

Shneidman's book ends with a moving personal letter to Arthur's mother. First, Shneidman hopes that the autopsy has produced some answers. He then expresses eloquently his deep sorrow for Arthur's loss.

In Shneidman's own words, these are the most salient points relating to Arthur's potential salvation and his ultimate demise, "I don't see what I could have done that I don't ordinarily (or even extraordinarily) do. I don't see what I might have done that

your son wouldn't have trumped. His pain and narcissism were more potent than my benign expertise, even at its most active. I cannot honestly say that I would have saved him, but I can swear to you that I would have tried my best to keep him alive. And perhaps I would have been more draconian than those who treated him. But that is hindsight. In the end, Arthur marshaled and focused all his considerable strengths to his deathful purpose. His assets were the tools of his undoing. Suicide is winning the hand but being unable to walk away from the table—and plunging the entire room into unexpected and pervasive sorrow.

"I am not sure that even Frieda Fromm-Reichman or Marguerite Sechahaye—legendary therapists of difficult patients—working together could have saved Arthur. But I want to reserve the delusion that he would not die under my care. I must take this position if I am going to bring anyone ashore.

"What I think is crucial here is the conscious focus of the therapist on the psychache, on the reduction and mollification of that pain, combined with the necessary redefinition and reconceptualization of that pain as somehow bearable after all.

"As a therapist I am aware of his towering narcissism, his view that his suffering is somehow unique, that he is special among men—a kind of malignant grandiosity that asserts that no one has ever had it as bad as he has. This almost delusional greatness-of-*my*-pain seems to be present in many suicidal people.

"Each person who commits suicide must take some responsibility for his own death. We are entitled to be critical of Arthur for his self-centered and shortsighted act. In a psychological autopsy we must speak candidly about the deceased. If our accounts were limited to "nothing-but-good," then we would be giving a eulogy.

"Part of our discomfort in this case has to do with our puzzlement over how he could be so thoughtless. The answer, I believe, lies in the constriction, the concentration, the tunneling of vision, the pathological narrowing and focus of the Self that is a usual part of the suicidal state. Boris Pasternak, the famous author, writing of the suicide of several young Russian poets, put it this way:

"A man who decides to commit suicide puts a full stop to his being, he turns his back on his past, he declares himself bankrupt and his memories to be unreal. They can no longer help or save him, he has put himself beyond their reach. The continuity of his inner life is broken, and his personality is at an end. And perhaps what finally makes him kill himself is not the firmness of his resolve but the unbearable quality of this anguish which is empty because life stopped and he can no longer feel it."[203]

Shneidman, close to the end of his letter hopes if he had had a chance that, "Together, he and I would redefine and fine-tune our understanding that, in actual practice, "unbearable" and "intolerable" really mean barely bearable and somehow tolerable, and that these terms can be incorporated into a dour pattern for life-long survival. Hopefully, he could come to do what Emperor Hirohito ordered his

[203] Boris Pasternak, *I remember: Sketches for an autobiography.* 1959.

overwhelmed people to do at the end of World War II: to suffer the unsufferable and to endure the unendurable—and to live.

"In the end we see there is no simple understanding of any one suicide. We end up, unhappily, thinking about it and puzzling over it for the rest of our lives as to who and what played this role or that role in the tragic ending and whether Arthur was star-crossed from early on."

Shneidman finishes the letter to Arthur's mom, "With every best personal wish for your health, your welfare, and your indomitable spirit."

In Shneidman's fine book we see clearly a wide range of theories, approaches and opinions regarding Arthur's suicide. If I peform a psychological autopsy, using my brain theory, what would it show? Would it make any more sense to the reader than the autopsies presented by the experts? I'll let the reader answer that for himself or herself.

First, and foremost, it is probable that Arthur suffered a congenital slowing of the brain due to decreased glutamate activity; or in simple terms, he was born depressed. I quote from the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR): Depression in children and adolescents might share the core diagnostic symptoms of depression in adults, but there are data that suggest that the prominence in symptoms may change with age. Certain symptoms such as somatic complaints, irritability and social withdrawal are particularly common in children. In prepubertal children, Major Depressive Episodes occur more frequently in conjunction with other mental disorders, especially Disruptive Behavior Disorders, Attention-Deficit Disorders, and Anxiety Disorders than in isolation. In adolescents, Major Depressive Episodes are frequently associated with Disruptive Behavior Disorders, Attention-Deficit Disorders, Anxiety Disorders, Substance-Related Disorders, and Eating Disorders.

It requires a highly trained psychiatrist to diagnose correctly depression in a small child or even an adolescent. It is practically impossible for "normal" parents to relate tantrums in a child or irritability in an adolescent with depression. Arthur's psychotherapist determined that the anger was due to his parents' divorce.

Arthur's irritability as a child, as a manifestation of depression, would explain the basis for his strained relationship with his mother. Irritability, as part of depression, also could elucidate his tantrums and his rage directed at others. Depression also explains nicely his social withdrawal, especially as a younger boy. His eating particularities, according to the Manual, can also be associated with depression.

When Arthur, as a child, externalized the most visible part of his depression, his irritability or his anger, then it would be perfectly natural that others, especially older children, would react with anger as a response to anger. This is a natural part of the emotional transference process. This is why he was continuously bullied as a child, especially by his older brother. The bullying is part of a childish process to restrain the anger of a fellow child.

In accord with my brain theory, it follows that when an individual is depressed, only depressed and negative memories can be activated. Thus, the "low" self-esteem seen

by most (and Arthur himself) as a part of Arthur's life, is just a natural consequence of the depression. His suicide attempt at fifteen was indicative of how critical his state of mind was at this age, and serves to illustrate how a young mind might interpret what is happening to them as normal, because they have never (or hardly ever) experienced anything different; or simply, in that negative emotional state they can't remember the good things. A depressed person is always looking for causes, trying to rationalize why they feel bad, and inevitably they do find (or invent) an explanation. Once a belief is established that some event, situation or relationship (or lack of) is the cause of the bad feelings, every effort will be expended to correct this and avoid, or get rid of, the negative emotions. Thus, Arthur "needed" to get married and his mood would improve, and later "needed" to get divorced for the same reason. He thought that becoming a doctor would solve his depression and when it didn't, he sought a new career, and so he became a lawyer. Arthur sought relief from his depression through his relationship with his girlfriend. And when it didn't provide any relief, the relationship became the cause of the depression, and "needed" to be terminated. An so Arthur goes from explanation to explanation trying to relieve his misery, when the real explanation is that he is suffering a disease: depression. And depression leads eventually to idiozimia, or a loss of self, the condition necessary, but not sufficient, for suicide to occur.

What could have been done to save Arthur? We need better therapies and medications. Only fifty per cent of depressed patients respond to antidepressants the first time, and there are 20 per cent of cases where medications do not work at all. It is possible that, through trial and error, an existing medication could've been found that would work in Arthur's case. But perhaps not. The rate of success of present therapies alone is even lower than antidepressants. But if the right medication could be found, the right therapy could help keep another bout of depression at bay.

More importantly, Arthur, in his final months, isolated himself further, primarily from his mother, sister and brother. The lack of emotional transference and countertransference that these relationships had provided contributed to his downward spiral and exacerbated his condition. Love and friendship must be maintained at all costs to have a chance of success. We must keep in mind that the more severe the depression, the greater the distortion of the self. Thus Arthur, shortly before the end of his life, became archidiozimic (if not idiozimic) due to the lack of accessibility to positive memories. The archidiozimia is accompanied by tremendous emotional pain for which there is no words, only metaphors at best. This would explain his "it feels like pins all over my body." The repeated bouts of depression led to Arthur's helplessness and hopelessness with accompanying sense of loss of will and strength to keep on. Under these conditions, suicidal ideation becomes a logical, rational solution and suicide is a question of time. Fear of another round of depression adds to the anxiety. Interestingly, Arthur does not seem to have reached a full idiozimic state in the days before his demise, as he was able to write a long, coherent, suicide note. However, his depressed state had been so pervasive in his life, that paradoxically, he was able to tap

into important relationships even in his depressed state. His frame of reference to depression dominated most of his life, and allowed him to function somewhat, even close to the end: he talked on the phone to his ex-girlfriend, and he saw his father and best friend on the day he died. But, perhaps his suicide happened only until he became idiozimic, on that fateful Sunday night, when he felt he had exhausted all his other alternatives.

A Few Words on Therapy

Medical developments have produced superb methods for correct diagnosis of physical disease. Whenever a patient presents diagnostic problems, the history, physical examination, laboratory procedures, tests and environmental agents are all routinely searched through. As a result some degree of mastery of differential diagnosis leads to a range of appropriate responses to restore the patient's health. Training in psychiatry hardly ever equips the physician so well. The training is abbreviated because the lack of a unified brain or mental theory makes most problems mysterious and without quantitative measures comparable to those of general medicine. Not surprisingly, the clinician confronted with many obscure mental problems which can be life threatening, with no firm guidelines between normal and abnormal, reaches out for new wonder drugs hoping that psychiatric and physical diagnoses can be treated similarly. This is why psychiatric problems, which probably require, more systems, completeness, and attention compared to some other physical ailments, receive quick fixes, shallow reassurances, and trial and error groping.

A good psychological examination needs to observe the patient in all respects—his or her speech, emotional expression (or lack of), movement, dress and all other outer signs. It should also require learning of the inner world of the person, which under the best of circumstances is hard to achieve. The normal every-day experience of emotional transference and countertransference processes should lead the clinician to a self-examination, which will serve to give clues of the patient's true state of mind, or at least to the reactions to the clinician. Family and close friends should be consulted and, minimally, should be cautioned about the chances of suicide (in most cases low, but a one in five, or even a one in twenty warrants this warning and education) and the nature of unpredictable suicide.

Research has demonstrated clearly that the absence of a parent through death, divorce, illness, or a parent with a time-demanding profession contributes to many forms of emotional disorder, especially the anger, depression, and the low self-esteem that accompany teenage suicide. In order to understand the growing epidemic of suicide among the young, the role of physically and emotionally absent parents must be considered.[204]

But, as always, the fact remains that most children coming from a one-parent family or broken homes, or who have divorced parents don't commit suicide. And

[204] Armand Nicholi, Jr., M.D., *The Harvard Guide to Psychiatry.*

there are, sadly, individuals that have had every possible advantage during childhood: adequate healthcare, good looks, loving parents and family, friends, emotional and financial security, a college education, and so on, who suddenly embark on the awful path of suicide.

True in many cases, but particularly in adolescents, what is termed an Adjustment Disorder, defined by the Manual (DSM-IV) as the development of clinically significant emotional or behavioral symptoms in response to an identifiable stressor—which in my opinion, could denote a perfectly normal reaction to terrible events—is confused with the cause of depression, anxiety or other disturbances such as truancy, vandalism, or reckless driving. On the other hand this diagnosis has often been used as a "wastepaper basket," that includes all adolescent problems not understood by the examiner. Many of us can detect behavior changes from normal to abnormal, even though the boundaries can be quite fuzzy. Most of us know when behavior becomes "weird." In general, my advice is to use common sense and err on the side of caution when dealing with problematic youths. Seeking psychiatric help should never be viewed as stigmatizing.

Caution must be used when considering a therapy for a particular psychiatric problem. Not all therapies are successful for everything, and not all people respond equally to the same therapy. Therapy is a process that attempts to change the way a person thinks, feels, and/or acts.

For the most part, more and more, psychiatrists tend to try to solve the problem with medications; a fifteen-minute consultation, diagnosis, prescription, and further visits to tweak the medication. On the other hand, psychologists can't prescribe medications, and concentrate on therapies based on talking and addressing body imbalances, flows, thoughts, energies or feelings. Curiously, for the most part, psychiatrists will only concern themselves with the patient's subjective view of "are you feeling better" in order to ascertain whether to increase or decrease a dosage or add or subtract a particular medication, or address a side effect. And the majority of psychologists will not concern themselves with the medications, much less dosages, that their patients are taking. They only concern themselves with the measurable results, even if these are subjective, to determine how efficacious is their therapy. Of course, one is billed for every visit, whether the prescribed therapy is working or not.

Psychiatrists and psychologists should understand that therapy and medication are part of *complementary tools available to treat mental illness.* Yet, there seems to be a split in psychiatry. How is it possible that medications and therapies are so often not considered in unison? In the words of William Normand, a practicing psychoanalyst, "Psychiatry has gone from being brainless to being mindless." The answer, perhaps, is economic. First, to receive a degree in psychiatry requires many more years of studies than a degree in psychology. Psychologists charge by the hour, psychiatrists by consultation, generally fifteen minutes, and in general, their income is proportional to the years of study.

Psychiatrists originally did a lot of talking and therapy; it was the only game in town, as the medication arsenal was almost nil. However, as more and more medications

became available, they realized how much more money could be made by prescribing medications and were happy to relinquish the talking to the psychologists. This is not to say that they are cynical and don't believe they are helping many, which they are. The psychologists, in turn, were happy to get all this business previously dominated by the psychiatrists. And, since both know that what the other is doing only works partially in many cases, if at all, they don't concern themselves with it. Considered as proof by many of them, that either medication or therapy is not working, is the fact that the patient is trying to supplement one or the other. Fortunately, there are some practitioners who will actively encourage both, medication and therapy. But to increase the chances and the rate of success, it is crucial to use both together. Part of therapy should be an education on the chemistry of the mental disorder and on what the medication is expected to do and of how it works.

When I had a problem with a cervical disk, the disk was removed surgically and the vertebrae were fused with a titanium plaque and screws. I was in the hospital about twenty-two hours. The bill was close to $42,000.00. My neurologist charged close to $8,000.00 for the one hour and twenty minute operation. I saw him a few minutes later that day. It is evident that he is a busy man, and that his time, financially speaking, is much better used in the operating room than talking to patients. At approximately $6,000.00 per hour, he is not going to waste time talking to patients or holding their hands. Let someone else do that. Many psychiatrists, likewise, let the psychologists do the talking and hand holding.

Responsible clinicians or even hospital staff members or concerned friend or family, commonly make a "contract" with a patient to avoid self-destructive behavior or to call if an impulse to commit suicide appears. The usefulness of such a "contract" has been studied very little. It is Dr. Kay Jamison's and my experience that when the time comes to commit suicide most people won't, or can't call. However, as part of any suicide-prevention-management program, it can't hurt. A suicide-prevention "contract" is best utilized as part of a comprehensive evaluation and treatment plan or in conjunction with a formal assessment of suicide risk. It is of the utmost importance to remember that in certain cases risk will never be zero and that agreement to a contract does not abolish the risk. A suicide-prevention contract cannot be the foundation or the beginning and end of suicide risk management.

New theories and therapies come into vogue all the time, and then drop into oblivion. The history of psychiatry is strewn with many ideas and concepts that have gone by the wayside. Concepts once held to be truths are discounted later as myths or superstitions. The more durable theories—neuropsychiatry, psychoanalysis and behaviorism—differ theoretically as well as in experimental and clinical approaches. Despite their differences, they share one basic assumption: hidden forces over which he or she has no control victimize the emotionally disturbed. These three schools maintain that the source of the patient's disturbance lies beyond his awareness; they gloss over his conscious conceptions and his specific thoughts and fantasies.

Emotions are triggered to produce a certain type of behavior. The caudate nucleus is responsible for activating all memories and modes of thinking associated with each emotion as well as activating emotions when certain modes of thinking are detected in order to produce a quick response. The caudate nucleus is a two way street. For example, when I think of Mitch, my son, different emotions are triggered depending on how I think of him; sadness, joy, nostalgia, yearning.

Each emotion in turn has its own particular circuits in the brain. The pathology arises when emotions are triggered for no apparent reason at all, or once triggered are not defused appropriately. Emotional disturbances may be present when these circuits are activated for wrong reasons or lock on and can't be turned off as needed. The wrong emotions can be triggered when thinking slows down or speeds up incorrectly for one reason or another. Other serious problems arise when the attentional systems are perturbed, either because they are not working synchronously, or stop functioning normally. In these latter cases, emotions are put into the wrong context, producing improper responses.

Extreme emotions can trigger a cascade of events in the brain that lead to archidiozimia and eventually to idiozimia, necessary conditions for suicide to occur.

Any therapy, to be successful, has to address these points. Most therapies fail to understand the loss of the sense of self, or confuse it with low self-esteem. Most therapies touch on these points tangentially or indirectly and their success will be proportionate to how they touch on these points. Hopefully with the model presented here, scientifically established risk prediction of high accuracy and precision will be developed, along with therapies with greater demonstrable positive results.

Using the ideas presented here, an obvious part of any successful psychotherapy are activities that can help speed up the brain to alleviate depression: efforts to avoid negative thoughts and exercises to have a better appraisal of self, can lead to having positive thoughts more often (this is the main element of cognitive therapy); improvement of interpersonal relations, especially because of the emotional transference and countertransference process can help immensely to improve mood; physical activities, such as sports or dancing, should be pursued. Any methods that decrease the chance of idiozimia should be pursued, such as helping the patient reconnect with his past, especially the positive aspects. Increasing the belief that suicide is wrong should reduce the risk of suicide. However, beliefs are probably established more strongly during a short window in childhood or early adolescence. This could explain why some countries with high religiosity have lower suicide rates.

Searching by trial and error for the correct medication or dosage, because we still don't fully understand how antidepressants work, should be pursued until one that works is found.

To give an example: lithium is generally part of the treatment to stabilize mood swings in manic depressive illness. The use of anticonvulsant drugs to treat manic episodes goes back to the 70's. Anticonvulsants may be the treatment of choice for patients with rapid cycles or a prior history of lithium failure or intolerance. Rapidly cycling moods

present are an increased suicide risk factor. However, whether the anticonvulsants are preferable for patients with mixed states remains to be seen.[205] On the other hand, it is also important to be circumspect about replacing lithium, which has a demonstrated capacity to decrease suicide rates in high-risk populations, with anticonvulsants, which do not. Given the importance of this clinical problem, it is surprising that so little research has examined combined lithium-anticonvulsant therapy.[206]

One group of patients admitted to a psychiatric hospital after lethal suicide attempts showed a suicide mortality rate of 22%, with most suicides occurring within the first year of follow-up. Experienced clinicians must assume that their work with high risk patients is therapeutically effective. They must trust that their efforts (especially if continued after hospital discharge over many months or years) prevent suicide, and that these efforts improve the quality of their patients' lives. At the time of writing their therapeutic effectiveness has not been refuted empirically, but it has not been thoroughly established either. In some properly selected patients that seek clinical attention, there is, however, reason to believe that treatment does protect against suicide. Yet, the suicide rate in the population at large has not been reduced at all.[207]

Coming from the nineteenth-century doctrines of physicalism, traditional neuropsychiatry tries to find biological explanations, such as chemical or neurological abnormalities, and applies drugs and other physical measures to relieve the emotional disorder. Neuropsychiatry is interested in a person's thoughts and feelings primarily as a diagnostic tool. Abnormal ideation and feeling states are regarded as manifestations of an underlying physical process or as a clue to a disturbance in neurochemistry. The neuropsychiatrist with his confidence in biological causes will administer drugs or use physical treatments like electro-shock therapy.

Psychoanalysis, which also has it's underpinning in the nineteenth century, attributes the person's neurosis to unconscious psychological factors: the unconscious elements are sealed off by psychological barriers that can only be penetrated by psychoanalytical interpretations. It attempts to get to the cause of a maladaptive condition, often by making unconscious (repressed) memories conscious. This is a long, arduous, slow process. Psychoanalysis might be good at explaining things, but it is not efficient at changing them.

Unconscious memories are really only inaccessible memories. Inaccessible, because an emotion is an integral part of the memory, and therefore, the memory is only accessible when the emotion is present.

Psychoanalysis regards conscious thoughts as a disguised representation of unconscious conflicts that are presumably causing the problem. The patient's explanations are considered as rationalizations and as coping defense mechanisms. As a consequence, the person's ideas, his reasoning and judgements, his common

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205 F.K. Goodwin and K.R. Jamison, *Manic Depressive Illness.* 1990.

206 Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervnetion.*

207 R.W. Maris, *Assessment and Prediction of Suicide.*

sense solutions are not taken at face value; they are treated as clues to concealed components of the mind. Psychoanalysis attempts to cure the neurosis by uncovering hidden (repressed) ideas and wishes and by translating the conscious thoughts and fantasies into their presumed symbolic meanings.

Psychoanalysis is less accepted today than in the past. Sometimes it requires four or five sessions a week for up to six years. There are other related therapies called Psychoanalytic psychotherapy, in which the therapy is less frequent on a weekly basis, but can still last up to three years. And more importantly, psychoanalysis has been proven to be quite ineffective to correct most emotional disorders.

As we saw previously, there are no unconscious memories in the Freudian sense. Memories are only accessible when the related emotional state exists. Visit a friend four or five times a week for years and tell him about your problems, and the results might be comparable to psychoanalysis.

Behavior therapy, whose roots can be traced even farther back to the eighteenth century, regards the emotional disturbance in terms of involuntary reflexes based on accidental conditionings that occurred previously in life. According to behavioral theory, the person cannot modify these conditioned reflexes simply by knowing about them and trying to will them away, so he requires the application of "counter conditioning" by a good behavior therapist. Practitioners of this therapy have downgraded thinking because in their zeal to be an exact science like physics, they reject data and concepts derived from man's reflections on his conscious experience. Only directly observed behavior is used in forming explanations. The thoughts, feelings and ideas that are only accessible to the person experiencing them *are not* considered valid data!

The behavior therapist, with his faith in the deterministic role of the environment, attempts to cure the neurosis through rewards and punishments, exposing the patient by degrees to situations or objects that are causing the disturbance. Behavior therapy is based on the principle of counter conditioning, which states that a person can overcome maladaptive anxiety elicited by a situation or an object by approaching the feared situation gradually and in a psychophysiological state that inhibits anxiety.

For anxiety and psychotic disorders, there are a number of different group therapies that use different approaches. Although, their success rate is low, they mainly help to reduce the severity of the symptoms. The sessions seem to work best for helping to restore health when facing extreme normal reactions, like grieving or overcoming stressful situations. There is Supportive, Analytically Oriented, Transactional Group Therapy and Behavioral Group Therapy. Sessions vary from once to three times a week for a few months to up to three years.[208]

However, groups are helpful in skill building and can provide the patient an interpersonal outlet to complement individual therapy. Preferentially, these therapies should use the same theoretical framework as the individual therapy to avoid confusion.

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[208] Harold I. Kaplan & Benjamin J. Saddock, *Synopsis of Psychiatry.*

It is recommended that the patient not participate in any more than one group activity while involved in individual treatment. It can become overwhelming in many ways—emotional, financial and practical.[209]

Cognitive therapy has its roots thousands of years ago, perhaps to the time of the Stoics, who considered that a man's conceptions (or misconceptions) of events rather than the events themselves are the key to his emotional disturbances. There is a supposition that the person's consciousness is a key to understanding and solving his psychological disturbance within the scope of his own awareness. Cognitive therapy takes as a starting point the notion that dysfunctional mental states (beliefs, attitudes, ideas) contribute significantly to psychopathology, and that the pathological conditions can be altered by helping the patient to identify and correct the beliefs. Cognitive therapy suggests that the person's problems are derived from distortions of reality based on erroneous premises and assumptions. It assumes that these incorrect conceptions originated in defective learning during the person's cognitive development.

Modern cognitive therapy arose from research on depressed patients, whose thoughts and images were found to be negatively biased in a systematic and habitual way, reflecting a negative view of the self, the world and the future. In cognitive therapy the hopelessness and pessimistic view of the future alongside low self-esteem are considered the precursors of suicidal ideation and intent. We have seen how negative emotions automatically trigger negative memories and vice-versa. This is not a question of faulty thinking, it is simply how the brain works. This is the challenge for therapies: understanding how the brain works and using the natural mechanisms to return to neutral calmness; using the interrelatedness between memories and emotions; understanding the mechanisms whereby an unlinking, when necessary, between memories and emotions is necessary. Therapeutic goals should include the separating of certain memories from their respective emotional states, or conversely through the use of positive memories trigger positive emotions to counteract the effect of mood disorders.

In general, present therapies, regardless of their origin, follow a simple treatment: the therapist helps the patient unravel his distortions in thinking and to learn alternative, more realistic ways to formulate his experiences. Cognitive therapy works well for dysthymia and nonendogenous depression. It is a short-term structured therapy that uses active collaboration between the patient and the therapist to achieve therapeutic goals. It places the patient in the role of scientist and uses his already available tools and experiences to approach problems that seem insoluble to him.

Cognitive therapy was revolutionary, in that it shifted the focus of therapy to the patient's life outside sessions, as opposed to the therapeutic transference, to the present, as opposed to the patient's early childhood, and to the contents of consciousness, as opposed to the unconscious.[210]

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[209] David Rudd, Thomas Joiner & Hasan Rajab, *Treating Suicidal Behavior.*

[210] Robert Leahy, Editor. *Contemporary Cognitive Therapy.* 2004.

The cognitive approach includes four processes: (1) eliciting automatic thoughts, (2) testing automatic thoughts, (3) identifying maladaptive underlying assumptions, and (4) testing the validity of maladaptive assumptions. Automatic thoughts are cognitions between external events and the person's emotional reaction. The therapist then teaches the patient how to see the validity of his or her thoughts. The goal is to reject inaccurate or exaggerated automatic thought with careful consideration. As the patient repeatedly does this, patterns become apparent, representing rules or maladaptive general assumptions that guide the patient's life. Ultimately the validity of the maladaptive assumptions must be tested and corrected when necessary.

Vulnerability is conceptualized as a trait rather than as the kind of state that characterizes depression. Vulnerability remains constant, even as episodes of depression emerge and then disappear. Vulnerability is also viewed as endogenous to the person (contrary to risk), as well as typically being viewed as *dormant* unless it is activated by stressful life events. In this view, stress is the principal activator of schemas (which are conceptualized as stored bodies of knowledge that interact with incoming information to influence selective attention and memory search) or cognitive structures. As a consequence dysfunctional schemas give rise to negative cognitions and congruent processing only when activated.[211]

Cognitive therapy has been applied mainly to depression (with or without suicidal ideation); it has also been used with other conditions, such as panic attacks, obsessive-compulsive disorders, paranoid disorders, and somatoform disorders.[212]

Here is an example of how cognitive therapy works, think of a depressed patient who thinks, "I never do anything right," is asked questions to recall prior and current accomplishments. If the depression is mild enough they will be able to do so, and in the process, help balance depressive conclusions about self, the world, and future (the so called negative cognitive triad) and temporarily improve mood.

Cognitive therapy makes ample use of the fact that people don't make decisions in a rational and dispassionate manner, and that their decisions are affected by past experience and underlying assumptions, heuristics, biases and emotional arousal.

Aaron Beck proposed that depression and anxiety are on a continuum with normal emotional experiences, and that all emotional experiences are linked to cognitions. Cognitive therapy, as outlined by Beck, requires cognitive specificity, in which each emotion is associated with particular cognitive themes. Depression is paired with cognitive themes of pessimism, self criticism and hopelessness. Anxiety is accompanied by themes of threat, danger, and vulnerability. Anger is marked by themes of violation and hurt, along with perceptions of others as malevolent. Each affective disorder is related to its own "disorder-specific cognitive profile."[213]

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[211] Ibid.

[212] Aaron T. Beck, *Cognitive Therapy*

[213] Robert Leahy, Editor. *Contemporary Cognitive Therapy*. 2004.

In my brain theory, there is a slight but subtle difference: emotions are an integral part of memories and depression is a confluence of many negative emotions triggered simultaneously. Therefore, each individual will present slightly differing, individual memories according to which negative emotions are concurrently triggered. Pessimism, self criticism and hopelessness are just a good generalization.

Similarly, cognitive models have been developed for suicide, stress and anger, and these have been successfully used for interpersonal relationships.

A whole body of studies provides remarkably consistent evidence for the model of looming vulnerability. According to this model, some individuals develop a distinct danger schema, the looming cognitive style (LCS), which produces unique cognitive risk for anxiety states and disorders by inducing individuals to formulate mental representations or expectations that portray threats as rapidly intensifying and rising in risk. Consequently the individuals who develop the LCS are likely to have difficulty habituating to potential threats, to demonstrate increased vigilance and anxiety, to perceive a time of urgency and imperative need for action, and to overutilize cognitive and behavioral avoidance strategies. The model of looming vulnerability postulates that Generalized Anxiety Disorder (GAD) and the tendency to worry and avoid unpleasant emotions are based on the LCS.

Second, since the LCS involves the generation of animated fear-inducing mental scenarios, images, and expectations of being overtaken by rapidly escalating risks and dangers, it may lead to higher levels of anxiety.[214]

Here, the model fails to recognize that memories that trigger anxiety, in a vicious circle, trigger other memories that are related to anxiety. When the anxiety is not defused because the threat or danger is perceived as real (a problem of cognition), the anxiety escalates.

The growing use of cognitive therapies with anxiety disorders, and a shift to coping-oriented rather than mastery-oriented therapeutic approaches has created a package of cognitive-behavioral therapy techniques that typically involve three primary elements. First, relaxation methods as generalizable skills whenever anxiety or worry might be noticed. Second, cognitive therapy to deal with the frequent detection/interpretation of threat to help generate alternative ways of perceiving and believing. And third, helping to build a habit of strengthening the interrelatedness of these two first strategies to cope better in response to daily stressors and stress. It is noteworthy that some studies indicate that only about one-half of the clients so treated reach good functioning at follow-up assessments. One of the effects of this approach is that the clients discover that most of the things that they worry about do not happen, and that they can cope quite well with those that do happen. All this contributes to a decline in their general tendency to perceive threat and worry.

Individuals intolerant of uncertainty, typical of GAD, find ambiguity stressful and upsetting, and have a hard time functioning in uncertain situations. They believe that

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214 Ibid.

uncertainty is negative and should be avoided, and that being uncertain is unfair. Because of this, many patients with GAD report that "even if everything is going well now, I worry that things will change." There seem to be connections between intolerance of uncertainty and GAD. Standard cognitive techniques do not necessarily decrease worry and anxiety, but it did help patients arrive at a more realistic estimate of the probability of a feared outcome. Behavior therapy seeks to alter maladaptive conditions (basically, bad habits) through the learning principles of behaviorist psychology, like extinction (aversion therapy to eliminate a habit) and positive reinforcement. Another technique is relaxation training to try to produce opposite physiological effects to those of anxiety: a slow heart rate, increased peripheral blood flow, deep slow breathing and the like. These therapies work best for specific phobias, obsessions and compulsions, and certain sexual disorders.

GAD does not lend itself well to exposure models and techniques of early behavioral therapy, given its lack of identifiable and circumscribed anxiety elicitors, and consequential absence of clear avoidance behaviors. GAD was often treated with general anxiety reduction techniques that did not specifically target its main feature—excessive and uncontrollable worry. Although these treatments were effective to some extent, residual worry and anxiety as well as high relapse rates were common. Because of this, many set out to identify specific cognitive and behavioral processes involved in GAD. Intolerance of uncertainty and worry in GAD have a relationship with a patient's tendency to focus on potential negative outcomes, exaggerating such outcomes and overestimating the probability of such occurrences. However, cognitive-behavioral therapies did increase the tolerance of uncertainty and consequently reduced the amount of time worrying.

Cognitive-behavioral therapies help patients recognize, accept and develop strategies for dealing with uncertainty. It teaches patients to distinguish between two types of worries—those about current problems and those about potential problems—and to apply a different strategy for each. About 50% of patients with GAD who receive cognitive-behavioral therapy achieve high end-state functioning or recovery.

Cognitive-behavioral therapy in GAD and Panic Disorder appears to be superior to most therapies, and at least equivalent to other behavior therapies. In the long run, many patients with comorbid disorders should get treatment targeting those other problems.

It is important to keep in mind that many studies that mention "cognitive-behavioral" therapies include interventions when cognitive restructuring, situational exposure, imaginal exposure, systematic desensitization, relaxation training and/or anxiety management training were utilized.[215]

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[215] Richard Heimberg, Cynthia Turk and Douglas Mennin, Editors. *Generalized Anxiety Disorder,* 2004.

Typically, the best results are achieved when medication and cognitive behavioral therapy are applied concurrently. However, given the evidence for the relatively less successful outcome of this combination in the treatment for panic disorder, such combination treatment strategies should be approached cautiously. Ultimately, benzodiazepines are no longer considered the pharmacological treatment of choice for GAD, with an increasing attention to SSRI's. Cognitive-behavioral therapy outperforms behavioral therapy alone. With respect to GAD, only about 32% of those receiving cognitive-behavioral therapy and 16% of those receiving behavioral therapy met the criteria for high end-state functioning.

A. Beck suggested that the concept of "fear" refers to a cognitive process that centers on the "primary appraisal" of threat. In his model the cognitive phenomena associated with anxiety occur at multiple levels. Threat ideation, in the form of thoughts or imagery, is caused by an interaction between basic cognitive processes (memory, attention, interpretation) and a person's underlying belief structures or cognitive structures concerned with personal vulnerability to threat. The interaction between this basic processes and structures produces biases in selective attention (or avoidance), memory, and exaggerated interpretation of threat stimuli.[216]

In many cases depression is a consequence of GAD. Social phobia and other anxiety disorders are also common comorbid conditions and clinical experience has suggested that avoidant, dependent and obsessive-compulsive personality disorders are commonly comorbid with GAD as well. When GAD is comorbid with major depression accompanied by hopelessness and suicidal ideation, these latter symptoms should be the first focus of treatment.[217] Needless to say, much work still needs to be done in this area.

Still, one of the therapies with the best record for curing depression is cognitive-behavioral therapy. This type of therapy combines elements of both behavioral and cognitive approaches. This is a form of psychodynamic therapy based on emotional and mental responses to external events, in the present and in childhood with tightly focused objectives. This approach tries to force the mind to think in certain ways, and by doing so, change one's reality. The therapist traces the sequence of events that have led the patient to their present difficulties. The patient then learns why certain events are depressing and tries to free himself of inappropriate responses. The patient is taught to neutralize his "automatic thoughts." Feelings, from this point of view, are not direct responses to the world: what happens in the world affects cognition, and cognition in turn affects feelings. If the patient can alter the cognition, then he can alter the respective moods.[218]

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[216] Robert Leahy, *Contemporary Cognitive Therapy.* 2004.

[217] Richard Heimberg, Cynthia Turk and Douglas Mennin, Editors. *Generalized Anxiety Disorder,* 2004.

[218] Andrew Solomon, *The Noonday Demon.*

Effective cognitive-behavioral therapies have been developed and used with relative efficacy for Panic disorder with agoraphobia, social phobia, specific phobia, and even obsessive-compulsive disorder.[219] Even though some aspects of cognitive and behavioral therapy are irreconcilable, used together, they are more effective than either alone. This shows that there is some truth to each.

Cognitive therapy applied to bipolar disorder, in comparison to a control group, had significantly fewer relapses (27% vs. 57%), significantly fewer days in hospitals, significant longer time to first manic relapse, higher levels of functioning, and better work performance. However, the most interesting finding was that the intervention did not have a significant impact on depression.[220]

Cognitive therapy fails to understand that emotions activate specific memories when they assume that faulty thinking is at the root of triggering the wrong emotion. However, they are correct in assuming that changing a mode of thinking can change the underlying emotions.

Cognitive therapy has proven to have limited success in the case of borderline personality disorder. According to the Manual (DSM-IV), rigidity is a hallmark of personality disorders and patients' distorted thoughts and self-defeating behaviors are extremely resistant to modification. When challenged, these patients rigidly, reflexively, and sometimes aggressively cling to what they already believe to be true about themselves and the world. In a revised model of cognitive therapy for personality disorders, personality is defined as "specific patterns of social, motivational and cognitive-affective processes." Personality includes behaviors, thought processes, emotional responses and motivational needs.

A mode is an integrated network of cognitive, affective, emotional, and behavioral components. In treatment, a patient learns to utilize the conscious control system to deactivate modes. When trigger events are reinterpreted in a manner inconsistent with a mode, the mode can be deactivated. Furthermore, modes can be modified. Treatment centers on correcting "black or white" beliefs.

In cognitive therapy, in relation to personality disorders, five main modes have been identified: abandoned child, angry and impulsive child, punitive parent, detached protector and healthy adult. When a patient with borderline personality disorder is suicidal or parasuicidal, it is important for the therapist to recognize which mode is experiencing the urge. This is very revealing, as it shows a very simplified view of suicide. Is the urge coming from the Punitive Parent mode? Here we see the assumption that suicide is a form of punishment for being bad. Or is the urge coming from the Abandoned Child mode? In which case, the assumption seems to be that suicide is a response to unbearable loneliness. Is the urge coming from the Detached Protector,

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[219] Richard Heimberg, Cynthia Turk and Douglas Mennin, Editors. *Generalized Anxiety Disorder.* 2004.

[220] Robert Leahy, Editor. *Contemporary Cognitive Therapy.* 2004.

in an effort to distract from emotional pain through physical pain, or to pierce the numbness and feel something? Or is it coming from the Angry or Impulsive Child? In which case suicide is seen as a desire to get revenge or hurt another person. Following these ideas, cognitive therapy assumes that the patient has a different reason for attempting suicide, depending on which mode is active.

Even when the patient is not suicidal, cognitive therapy assumes one of the modes is active, should be identified, and the therapeutic strategy, using cognitive and behavioral work, is adjusted accordingly. The therapist-patient relationship is also adjusted to fit the mode.

Traditionally, cognitive therapy evolved to incorporate an emphasis on core beliefs and underlying assumptions earlier in the course of treatment, plus greater attention to childhood antecedents and the therapeutic relationship, to go along with the emphasis on current life problems. This was referred to as the three-legged stool.

Presently, there is a tendency to streamline talking therapies to focus on practical improvements, rather than searching, dwelling and beating to death childhood traumas. Obviously, the more we understand how the brain works, the more we can improve therapies.

Relapse for depression in the first two years after cognitive therapy with medications was 15 per cent, compared to 21 per cent with cognitive therapy alone, 32 per cent with drug continuation alone and as high as 50 per cent after medication with no continuation.

Well-being therapy, an extension of cognitive therapy that incorporates attention to positive activities and self-perceptions, has been shown to reduce the risk for recurrence when added to continuation medication.[221]

Independent of the approach, a strong therapeutic relationship and alliance with the patient is essential. The quality of this relationship has been found to be predictive of response across a number of different treatments. For the clinician to respond appropriately requires patience, persistence and comfort with dealing with hostility or interpersonal aggression in the form of emotional transference. In dealing with suicide, cognitive-behavioral therapy makes three fundamental assumptions:

1) The patient is manifesting serious psychopathology. This translates, as the patient has considerable interpersonal difficulties, and will include dealing with the clinician. At a minimum, it is expected that the clinician will treat the patient better than everyone else, and being closer to the patient will bear the brunt of the patient's anger and hostility.
2) The patient is seen at his or her worst, that is, when he or she is acutely suicidal.
3) The hallmark of effective crisis intervention is not only symptom resolution but also skill building and limited personality development.

[221] Ibid.

As we have seen, the state of idiozimia can appear unpredictably and its duration might be minutes or hours. The reappearance of idiozimia might or might not occur within months. The clinician, except right after a "failed" suicide attempt or immediately before a suicide attempt, can seldom expect to see the patient in this state.

After a determination of the severity of dysfunction evidenced by the patient a decision on treatment will be made. If the patient is at high risk, he or she will remain hospitalized. However, if the risk is determined to be low enough, the patient will be treated as an outpatient. Depending on the risk, special consideration will be made, including setting up a suicide watch. At present, a high-risk will only be determined by very serious or obvious psychopathology or past history.

While considering risk many factors will be checked. Other self-destructive behavior might be present, such as self-mutilation, substance abuse, aggressiveness and sexual acting out. These behaviors might need to be addressed independently. If personality disorders and mood disorders or schizophrenia are present simultaneously, the complexity of the diagnostic picture is increased proportionately. The clinician should consider how all this impairs the patient. What symptoms, deficient skills and maladaptive personality traits are present?

Generally, an effort at differentiating between suicidal ideators, single attempters and multiple attempters will be performed. Without including concepts like the isorropic circuit, idiozimia, the phobothymic switch and the aftoktonic switch, it will be impossible to scientifically predict when or how ideators become attempters, or how or when single attempters will become multiple attempters.

Suicidality is viewed as a general construct with three discernible manifestations of psychopathology: 1) Symptoms(i.e., depression, anxiety, hopelessness, panic, suicidal ideation, guilt, anger, etc.); 2) Skill deficits (i.e., problem solving, emotion regulation, distress tolerance, interpersonal skills and anger management); and 3) Maladaptive personality traits influencing both self-image and interpersonal relationships with friends and family.[222] In reality, idiozimia (loss of self) caused by a mental disorder is at the center of suicidality, all the rest is manifestations of this mental state. Idiozimia, or even archidiozimia, will be manifested as skill deficits and maladaptive behaviors, due to the constriction of memories. The greater the distortion of the self, the lesser the capacity to adapt, or to respond correctly, because there will be less memories available.

Symptom resolution obviously will aid the patient in feeling better, and skill building and personality development will aid substantially in avoiding a relapse. At present, this is about as good as it gets. With much luck, this is enough. But in many cases, tragically, it is not.

We have seen under which conditions the isorropic circuit's activity is pushed far from neutral calm producing idiozimia. It now becomes clear that skill deficits and

[222] David Rudd, Thomas Joiner & Hasan Rajab, *Treating Suicidal Behavior.*

maladaptive traits are a direct consequence of this mental state. This knowledge should give the clinician more tools to restore mental health.

Another therapy with similar rates of success in treating depression is known as interpersonal therapy (IPT). Gerald Klerman and his wife, Myrna Weissman, formulated this approach. IPT focuses on the immediate reality of current day-to-day life. It fixes things in the present. It tries to teach the patient how to make the most of whatever he is; it does not attempt to make the patient a deeper person. It is a short-term therapy with boundaries and limits. It assumes that stressors trigger depression, and that these can be cleaned up through well-advised interaction with others.

Treatment is done in two stages. In the first, the patient is taught to understand his depression as an external affliction and is informed of the prevalence of the disorder. His symptoms are sorted out and named. He assumes the role of the sick one and identifies a process of getting better. The patient catalogs his present relationships, and with the therapist, defines what he gets from each one, and what he wants from each one. Second, the therapist works with the patient to figure out what the best strategies are to elicit what is needed in the patient's life. Problems are sorted into four categories: grief; differences in roles with different relationships (what you give in relation to what you expect); states of stressful transition in personal and professional life; and isolation. The therapist and the patient establish a few attainable goals and decide how long to work toward them.[223] Through interactions with others, hopefully, emotional changes will be achieved. ITP indirectly uses the positive emotional attachments to produce positive thoughts and behaviors.

There have been some similarities noted between individuals who attempt suicide and individuals diagnosed with borderline personality disorder. Suicide attempt rates of 69 to 75 percent have been reported. Indeed, the presence of suicidal acts is one of the criteria for the disorder. Dialectical behavior therapy was developed specifically for the chronically suicidal patient, that is, one who is unremittingly high in suicidal ideation., frequently threatens or talks about suicide or has difficulty articulating reasons for staying alive.

Dialectical behavior therapy was developed from a combined motivational and capability model of suicidal behavior. The idea is twofold: (1) suicidal individuals lack important interpersonal, self regulation (including emotional regulation) and distress tolerance skills and capabilities, and (2) personal and environmental factors inhibit the use of behavioral skills the individual does have, interfere with the development of new skills and capacities, and often reinforce inappropriate and suicidal behavior. The emphasis on capability management is similar to the focus on behavioral skills training, stress management, and relapse prevention among various behavioral therapies. It is compatible with supportive psychodynamic psychotherapies. The emphasis on changing motivational factors is similar to behavioral treatments emphasizing exposure (for example, desensitization, flooding, cue exposure) and to

[223] Ibid.

models based on principles of operant conditioning (that is, reinforcement, extinction, and aversive contingencies). It is also compatible with cognitive therapies and expressive psychodynamic therapies.

In practice, focusing on patient change, either by motivation or by enhancing skills, is experienced as irrelevant by chronically suicidal individuals and precipitates withdrawal, noncompliance, and early dropout from treatment. Also, skills-training to the extent necessary is extremely difficult if not impossible within the context of a therapy oriented to reducing the motivation to die. And, of course, new behavioral coping skills are extremely hard to remember and apply in a state of crisis, and applying them to suicidal situations is extraordinarily difficult.[224]

As long as there is no understanding of the role of idiozimia in suicide, most therapies will only be able to beat around the bush, sometimes helping indirectly by affecting some side issues, which accidentally diminish idiozimia and therefore lessen the possibility of suicide. Obviously, an individual that experiences archidiozimia is unlikely to ameliorate or tolerate the emotional, interpersonal, and behavioral stresses in their lives. He or she will also suffer cognitive restrictions, and impairment of problem solving due to lack of access to specific (mostly positive) memories. From this point of view, solving or reducing any of these situations will lessen the archidiozimic state and help restore some balance, thereby increasing the individual's coping skills.

Other therapies derive from the idea of energy systems. Eden wrote that there are eight major systems: the aura, the chakras, the meridians, the Celtic wave, the basic grid, the five rhythms, the triple warmer, and the strange flows.

The energy that surrounds all living things is the aura. Bioenergy is said to enter the body through seven energy centers called chakras. Six are located along a line paralleling the spinal column, and the seventh is said to extend out of the top of the head. The chakras connect with the meridians and other energy levels. The meridian system is said to involve fourteen energy pathways: two central vessels that run vertically on the center of the front and back of the body, plus twelve primary meridians that exist bilaterally. Each meridian is said to have two channels of energy. One flows close to the skin and is presumably the one accessible to an acupuncturist and an energy psychotherapist. The other, flowing deeper inside the torso, passes through the organ with which it is associated. Along each of the meridians are the acupuncture points, some 365 in the twelve primary meridians alone. Most of these energy systems were developed in China and India thousands of years ago.

The main principles of energy psychology are the existence of a subtle energy system in all living things. This is manifested by energy flows and when the flows are blocked, disorders result; disturbing thoughts and emotions triggered by traumas affect the meridians. Each meridian is related to an organ, which in turn is related to a set of

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[224] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

emotions. By treating the correct points in a meridian, the flow will be reestablished and the accompanying emotional disturbance will clear.[225]

These energy therapies are emerging as the therapies of choice, partly because of the low rate of success of the other therapies, partly as a choice of last resort, and partly due to their success rate.

In the 1960's the chiropractor George Goodheart, building on the knowledge of manual muscle testing, developed applied kinesiology, and this was later mixed with meridian theory to promote health. In the 70's psychiatrist John Diamond, trained in applied kinesiology, discovered that the different meridians and their associated organs were related to different emotions.

Psychologist Roger Callahan, building on this work developed what came to be known as Callahan Techniques-thought field therapy (TFT). Later, in the 1990's these evolved into what is now known as Evolving Thought Field Therapy. Once the organ involved with the negative state is found, the practitioner treats the meridian rather than the organ itself. Using muscle testing he determines which points need to be tapped and in what order. He added a procedure called eye roll at the end of successful treatment.

Several variations on this have been developed. Gary Craig and Adrienne Fowlie suspected that all these complicated sequences were not necessary. In 2000, John Diepold developed an alternative to tapping. Acupuncturist Tapas Fleming developed another technique popular with energy therapists in 1993. This involves the patient and therapist touching simultaneously meridian points, a chakra and the occipital region of the head.

All these rapidly evolving therapies are known as energy psychology or energy therapy.

There are several methods and versions of energy treatments. According to some of these methods, some problems have several aspects, and not until the presenting one is cleared is an underlying one apparent and accessible to treatment. The patient must be thinking of the problem for it to be treatable. There is the assumption that treatment can be blocked by a number of factors, including neurological disorganization, psychological reversal, and energy toxin systems. Therapy is designed to address all this. Energy psychologists and acupuncturists believe that the meridian, chakra, and aura systems underlie affect, and are affected by cognitive, emotional, sensory, and chemical events. Energy practitioners believe they are intervening at the most fundamental level.

The rate of success of these energy therapies increases when combined with eye movement desensitization and reprocessing (EMDR) psychotherapy.

Francine Shapiro developed EMDR in the late 1990's. Though applied to most disorders, EMDR is recommended particularly for treatment of traumatic memories

[225] John G. Hartung & Michael D. Galvin, *Energy Psychology and EMDR*.

and the mental, emotional, behavioral, and interpersonal problems associated with traumatic experiences.

In 1987 Shapiro noticed that as she thought about something disturbing, her eyes spontaneously made rapid movements from lower left to upper right, which seemed to cause the thought to lose its disturbing quality. Most people, however, need help in keeping their eyes moving to achieve the same effect. Shapiro developed methods by moving her hands and asking her patients to follow with their eyes. Subsequently other forms of bilateral, alternating stimulation in addition to the eye movements have been used effectively, but eye movement has been retained in the name of the method. Shapiro noticed that most of the disturbing thoughts her clients reported were related to anxiety, so she used the word desensitization to describe the anxiety reducing effects of the eye movement. Shapiro added strategies to resolve symptoms further and taught her clients coping skills.

These are the following principles of EMDR: (1) People can heal themselves. Each person has a natural ability to process disturbing life events to the point where these events become simple memories; (2) the self healing system can become blocked or "stalled"; (3) blocked processing affects individuals in past, present and future time. The unresolved past and present symptoms, then, interfere with future functioning. EMDR follows a three-pronged treatment approach, which involves healing the past, removing the present symptoms, and addressing possible future manifestations of the issue. And (4) EMDR reactivates the self-healing system with eye movements and other bilateral alternating stimulations.

The practitioner focuses on helping the patient: (a) pay attention to the past trauma as they notice the present resources available; (b) reactivate the self healing system; (c) with bilateral stimulation maintain and accelerate the self healing system as the patient revisits the trauma long enough to reprocess it adaptively; (d) allow the adaptive and accelerated processing to proceed in a positive, curative direction assisted by the self healing principle; (e) insure self healing remains active after EMDR and is available to manage future traumatic events. As patients revisit the traumatic past, they often report re-experiencing an event in its original form, with similar intensity, emotions, thoughts and memories. These "abreactions" surprise patients because of the unexpected intensity of the memory as well as other associated memories (called childhood folders). Abreactions are neither required nor evident in all EMDR sessions, but their appearance is sufficiently frequent that they compromise another principle of EMDR. This is why, practitioners claim, only professionals should be trained in this method.[226]

EMDR shares with psychoanalytic tradition the view that traumatic memories have not yet been incorporated into more reality-based perceptions and that treatment

[226] Ibid.

must be directed at updating earlier events, often from childhood. Behavioral terms are used frequently, but with a different connotation. The EMDR assumption that past trauma needs to be re-experienced is at odds with the traditional behavioral focus on the present. The desensitization that occurs in EMDR refers to the reduction of negative emotions through confronting and reprocessing of traumatic memories. Shapiro emphasizes the cognitive aspects of the EMDR model. He implies that changes in thinking are more likely to precede and cause, rather than accompany or result from, changes in other aspects of functioning, such as emotional, sensory, imaginal or behavioral. In this sense EMDR is a cognitive therapy.

There is overlap between EMDR and other psychotherapies in terms of informed consent, history taking, use of homework, and follow-up. Rapport between therapist and patients is also seen as essential, though EMDR therapists are more likely to attribute treatment benefit to the EMDR method than to the therapist or the healing power of the therapeutic relationship.[227]

When good therapists integrate EMDR with their particular form of therapy, they increase their rate of success as well as shortening the duration of treatment. Andrew Solomon, after experiencing EMDR said, "I always came out of my EMDR therapist's office reeling (in a good way); and the things I learned have stayed with me and enriched my conscious mind. It is a powerful process."[228]

There exist strong relations between emotions and gestures, body postures, facial expressions, and eye movements. For example, smiling will generally produce a small increase in the sense of well-being. The EMDR therapy makes a patient focus on the past traumatic events in the context of the present. Although not explicitly part of EMDR theory, some of the eye movements coincidentally access the emotional connections to the traumatic event. Other eye movements are unrelated to the emotion, and because of this, as the patient concentrates on the traumatic memories, these become separate from the negative emotions that they triggered. It is not a question of self-healing, but a question of triggering, through eye movements, a different assortment of emotions. In turn these emotions can be dealt with easier than the original negative emotion that was being triggered. When it works, slowly, in a step-by-step fashion, the traumatic memory and associated mode of thinking is distanced more and more from the negative feelings.

Hopefully future therapeutic methods will take into consideration the deep relationships between emotions, context and memories. Hopefully the concepts of archidiozimia, idiozimia and the aftoktonic and phobothymic switches will lead to a better understanding of suicide and can also contribute to improved therapies.

At the very minimum, the unexamined life can seldom be brought back to health without some close examination. A profound examination is almost always

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[227] Ibid.

[228] Andrew Solomon, *The Noonday Demon*.

revealing. Intimate friendships and relationships help maintain health through these examinations. Therapists base their practices in listening closely and attentively while the patient gets in touch with their true motivations, so that they can begin to understand why they act and feel in certain ways. Many times naming something is a way of subduing it, and knowing the source of the problem can be useful in solving it. At least the therapies might teach some tricks to cope a little better and improve the quality of life slightly. In some cases, isolation and depression are a vicious circle, and a therapist can help connect the patient with his friends and relatives and mitigate the severity of the situation.

The combination of therapy with medications has some beneficial interactions. In some cases medications improve concentration or reduce painful feelings and thus facilitate therapy. Medications can decrease distorted or irrational thinking, and improve the effects of therapy. The therapy will help with compliance through better understanding of the illness. Therapy can help with withdrawal from medications. And last, therapy produces biological changes in the brain that help alleviate biochemical abnormalities.

On the other hand, we must keep in mind that there can be negative interactions. Medications can interfere with learning and memory. Medications cause dependency. These can reduce the effectiveness of therapy. Medications can relieve symptoms and lessen motivation to continue with therapy. In cases of physical illness (as opposed to mental disorders), therapy can cause stress and worsen things.

According to correctional Suicidology expert Joseph Rowan, the most important element in suicide prevention behind bars is the presence of human interaction (I would add that this should be the case in all suicide prevention programs). Vulnerable, potentially suicidal inmates (or anyone who is suicidal) need close observation and access to meaningful support networks and, obviously, isolation and loneliness exacerbate life stress. Rowan has noted that, in fact, the mere placement of inmates in cells with other inmates significantly reduces the risk of suicide, simply through the social presence of and interaction with others.[229]

It has been shown that therapy, at best, is nearly as successful as medications for pulling people out of depression. On the other hand, therapy has a protective effect on recurrence, and is a good strategy for preventing a next episode. Less than half of depressives experience significant improvement with just medication; less than half experienced significant improvement with cognitive behavioral analysis (one of the most successful therapies for depression); yet more than 80 percent experienced significant improvement after being treated with both.[230]

In some cases, because clearly there are some biological roots, especially in manic depression, there is a tendency to minimize the importance of psychotherapy and due

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[229] R. W. Maris, *Assessment and Prediction of Suicide.*

[230] Andrew Solomon, *The Noonday Demon.*

to the cost, it is seen in some circles as a luxury rather than a necessity. There is quite a bit of evidence showing that psychotherapies in combination with mood-stabilizing medications is more effective than medications alone. In one study comparing patients receiving lithium alone with patients that received lithium and behavioral therapy, 70 percent of the lithium alone group relapsed compared with 13 percent of the therapy and lithium group.[231]

Most therapies are only as good as the therapist. The relationship between patient and therapist can be more important than therapeutic method. In times of crises, when all else fails, it is the therapeutic relation ship itself that may keep the patient in this life. A deep, intimate relationship can probably help a lot by constructive conversations. In an important study done in 1979, research demonstrated that any form of therapy could be effective when both the patient and therapist acted in good faith, the patient believed that the therapist was good, respected and liked the therapist, and last that the therapist had an ability to form understanding relationships. The experimenters chose English professors with this quality of human understanding and found that, on average, the English professors were able to help their "patients" as much as professional therapists.[232] A deep, intimate relationship, because of emotional transference, can be just as healing, and is probably the most important element in therapy regardless of the therapeutic approach. It is important to keep in mind that treating a highly suicidal patient is a serious emotional drain on any clinician. Clinicians should be limited to treating one or maybe two cases simultaneously at the risk of becoming too detached and therefore ineffectual.

Robert Litman, at one time president of the American Association of Suicidology, wrote, " . . . there is a strict limit to how many suicidal patients any therapist can treat at one time. It is important that the personal life of the therapist is in order, because this kind of treatment is a marathon, and it can be exhausting. If the exhaustion starts to show, the treatment ends . . . An important problem in suicide prevention is that there are many chronically suicidal persons in the world and limited resources to treat them with psychotherapy. That is why we turn so hopefully to psychopharmacology for help."[233]

Countertransference in therapy, especially with borderline personality disorder patients, is often intense, especially when dealing with suicidality. Therapist fantasies of being a competent and capable rescuer mix with powerful feelings of frustration, helplessness, anger, disappointment, devaluation, and hopelessness. Ultimately it can lead to rejection of the patient. Countertransference, if unaddressed, can increase the patient's suicide risk. The feelings produced by countertransference may be intolerable to the therapist and may be expressed in potentially devastating ways for the patient if

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[231] Douglas Jacobs, *The Harvard Medical School Guide to Suicide Assessment and Intervention.*

[232] Andrew Solomon, *The Noonday Demon.*

[233] Edwin S. Shneidman. *Autopsy of a Suicidal Mind.* 2004.

they are not acknowledged and addressed. In the hospital countertransference issues are often intensified because several staff are involved. Countertransferential responses can be reinforced by group and staff dynamics.[234]

K.R. Jamison, in a note of appreciation in her book *Night Falls Fast* writes, "The debt I owe my psychiatrist is beyond description. I remember sitting in his office a hundred times during those grim months and each time thinking, What on earth can he say that will make me feel better or keep me alive? Well, there never was anything he could say, that's the funny thing. It was all the stupid, desperately optimistic, condescending things he didn't say that kept me alive; all the compassion and warmth I felt from him that could not have been said; all the intelligence, competence, and time he put into it; and his granite belief that mine was a life worth living. He was terribly direct, which was terribly important, and he was willing to admit the limits of his understanding and treatments and when he was wrong. Most difficult to put into words, but in many ways, the essence of everything: he taught me that the road from suicide to life is cold and colder and colder still, but—with steely effort, the grace of God, and an inevitable break in the weather—that I could make it."

Treating suicidality requires considerable time and energy, frequently more than therapists can expend and definitely more than provided by insurance carriers. Hopefully with many of the concepts presented here, more streamlined and more successful methods might be introduced.

Finding the right therapist can be daunting. Andrew Solomon recounts his search for a new therapist after terminating with psychoanalysis. "I tried eleven therapists in six weeks. Some of the therapists seemed wise. Some of them were outlandish. One woman had covered all her furniture with Saran Wrap to protect it from her yapping dogs. I left when one of the dogs peed on my shoe. One man gave me the wrong address for his office, and one told me that that I had no real problems and should lighten up a bit. There was the woman who told me she didn't believe in emotion, and the man who seemed to believe in nothing else. There was the cognitivist, the Freudian who bit his nails, the Jungian, and the autodidact. One man kept interrupting me to tell me that I was *just* like him. Several seemed simply to not get it when I tried to explain to them who I was." Solomon ends with this quote from Steven Hyman, "We try to do studies of drugs versus therapy. Have we done studies on bright therapists versus incompetent ones? We are really Lewis and Clark in this area."

Diet and exercise improve or lessen mood disorders. When the body becomes physically active, endorphins are produced, and endorphins make you fell better; good when you are normal, and less bad when you feel terrible. By pushing the body, the brain will follow and become more active, and thus the negative feelings are lessened. Diet cannot cause a depression to remit, but it can help to raise certain levels of neurotransmitters or hormones. Eating well can help reduce recurrences. For

[234] Douglas Jacobs, *The Harvard medical School Guide to Suicide Assessment and Intervention.*

example, sugar and carbohydrates appear to raise the absorption of tryptophan in the brain, which in turn raises serotonin levels. As another example, dopamine synthesis relies on B vitamins, especially B12.

The evidence for beneficial mood effect from omega-3 fatty acids is the strongest of all, the theory being that food rich in B vitamins raises the level of omega-3 fatty acids.

Fluctuating levels of blood sugar, with highs and lows throughout the day, can produce depressive symptoms as part of a syndrome of adrenal exhaustion.[235]

Under certain conditions, hypnosis might help, and massages can improve moods slightly. Optimism and even belief can be helpful. Anything that improves the symptoms of depression can be the beginning of the long spiral upwards in the return to normalcy.

Not all competing theories can be right, but some therapists maintain that apparently contradictory models of change may all have merit in different contexts, and that any aspect of human functioning can at one time be a cause, at another an effect. This is the main argument for why sometimes in one case a particular therapy works when others didn't, or why a particular therapy works in some cases but not all.

A brief word on psychopharmacology is in order. In general, mood-altering drugs take one or two weeks to kick in. The phase of recovery can last a long time. Many psychiatrists feel that this is a dangerous time, because during the worst of a depression, the patients generally don't have enough energy to feed themselves, much less kill themselves, but in this emerging period they have enough energy to carry out a suicide.

Andrew Solomon, deep in depression, recounts, "I was also aware that if I didn't allow myself the relief of considering suicide, I would soon explode from within and commit suicide. I felt the fatal tentacles of this despair wrapping themselves around my arms and legs. Soon they would hold the fingers I would need to take the right pills or pull the trigger, and when I died, they would be the only motion left. I knew that the voice of reason was the voice of reason, but I also knew that by reason I would deny all the poison within me, and I felt already some strange despairing ecstasy at the thought of the end."[236]

Many people don't like depending on the drugs, or stop taking them to avoid side effects in an effort to feel normal again. Any good psychopharmacologist can confirm that going off drugs should be done gradually and under supervision. And some people have to take medication all their life.

The medications available to treat mood disorders are addressing some neurotransmitter deficiencies, and producing their benefits in a roundabout way. In some cases they work better than in others, and in some cases they don't work at all. About eighty per cent of patients are responsive to these drugs. However, only about fifty

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[235] Ibid.

[236] Andrew Solomon, *The Noonday Demon.*

per cent respond positively to the first drug they are given. Good psychopharmacologists obtain the best results using an individually tailored cocktail of medications and managing carefully the side effects.

Andrew Solomon candidly reports, "I have had some sexual side effects—a slightly decreased libido and the universal problem of a much-delayed orgasm. A few years ago, I added Wellbutrin to my regimen; it seemed to get my libido running again, though things have never come up to old standards. My psychopharmacologist has also given me Viagra, just in case I get that side effect, and has since added dexamphetamine, which is supposed to increase sexual drive. I think it does but it also makes me twitchy. My body seems to go through shifts beyond my ability to discern, and what works splendidly one night may be tricky the next. Zyprexa is sedating and I mostly sleep too much, about ten hours a night, but I have Xanax around for the occasional night when I am assaulted by sensation and cannot get my eyes closed."[237]

Mental disorders, even when mild, and without suicidal ideation are tough. There is no way around that. Everything possible must be brought to bear to reduce the suffering when these illnesses strike.

[237] Ibid.

Improving the Odds Against Suicide

Proper risk assessment of suicide is the area where we need to improve the most, especially when it comes to potential firsters. As we have seen, the necessary mental state that leads to suicide is archidiozimia, the distortion of the self, which eventually leads to idiozimia, the loss of self, the one and necessary condition for suicide to occur.

Mood disorders, such as unipolar and bipolar depression, schizophrenia, and personality disorders are predisposing risks that can (but not necessarily) lead to idiozimia. Under these conditions, potentiating risks, such as a devastating loss should be viewed carefully as these can aggravate an existing mental state. Care should be taken to evaluate what is a potentiating risk; a divorce might improve someone's lifestyle, not necessarily be a bad thing; a strong family support might be negative if the individual feels he is a burden to society. Many people who have committed suicide recently suffered a major loss, such as the loss of a loved one, a separation from a lover, the loss of job or home or health, or loss of freedom. However, it is simplistic to explain suicide as being caused by such losses. These losses are perhaps more than the drop that filled the bucket, but are not the cause of suicide, just one more event in the long chain that leads to suicide.

Over the past thirty years youth (ages 15-24) suicide has become the second leading killer of this group in the United States. Few efforts to prevent suicide have been done at a national level, and most of the programs done have been at the state or local level.

Predicting suicide is quite different than preventing suicide. Too often there are very few clear links between the assessments conducted and the interventions carried out. With present knowledge many youths who are at risk for suicide cannot be effectively identified and many who could be correctly identified as at risk are unlikely to seek out help.

The classic public health model of disease prevention has three distinct levels of prevention: primary intervention focuses on reduction of the incidence of new cases in the overall population, rather than reduction of the effects of the existing disorder; secondary intervention focuses on identifying specific individuals who are showing early but clear signs of the focal disorder(s), and the preventive goal here is the reduction of the intensity, severity and duration of the disorder (this does not include prevention of its onset or reduction of incidence in the population); and tertiary prevention, which refers to interventions with specific individuals who have an existing (often serious or severe) disorder. Prevention in the tertiary level is used not in the sense of prevention of the disorder, but instead, prevention of the residual and radiating effects that the persons' serious disorder may have on their lives and the lives of those around them.

Tertiary prevention, at best, is treatment, while secondary prevention becomes early intervention and primary intervention alone assumes the mantle of prevention.[238]

Primary intervention deals with healthy individuals, in general more than 99.9 percent of the population. To change suicide at this level would require intervention by almost all, even urban planners, educators, recreational workers, housing professionals, architects, parents—to mention a few—as well as institutional changes at the level of congress, schools and the medical profession. This would be the only way to modify a broad set of conditions that may predispose to or protect the population against suicide and associated dysfunctions. At this point, we cannot change sets of conditions that haven't been clearly identified as leading to mental disorders, let alone suicide. In many cases, even when we can reduce the availability of certain means to suicide, it is impractical. For example, to mention one point of view, making the Golden Gate Bridge inaccessible to pedestrians, does not preclude people from jumping from other high places.

In 1994, the Committee on Prevention of Mental Disorders proposed a new classification for the study of prevention of mental disorders. These approaches represent a new paradigm in public mental health policy; namely, a move toward truly effective primary prevention strategies and treatment and health status maintenance. An important distinction made by the Committee's report concerns a scheme of classification based on three types of intervention: *universal* preventative, *selective* preventative and *indicated* preventative interventions.

Universal interventions may be directed at an entire population thought to be in danger for developing mental disorders. For example, public education campaign to inform the public about the dangers of substance abuse or early warning signs of depression. Selective measures refer to those targeted at a subgroup with a shared risk factor, even if they are not showing any symptomatology. For example, targeting children of parents with manic depressive illness or who have been victims of physical or sexual abuse. Indicated measures refer to those employed with people who already have early signs and symptoms of a mental disorder, such as anxiety, sleep disturbance, early patterns of eating disorders, "flashbacks," agitation and so on. All three types of measures represent subtypes of primary prevention. This more elaborate approach is justified due to the expanded pharmacopoeia and advances in neuroscience, biology, and genetic research into the etiology of mental disorders, which hopefully will permit targeting of individuals at high risk for suicide.[239]

But, studies show that for a wide range of developmental outcomes, efforts to identify unique and/or specific etiological agents are neither appropriate nor supported by the data.[240] Indeed, it would be difficult not to relate everything negative

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238 R.W. Maris, *Assessment and Prediction of Suicide.*

239 Douglas Jacobs, *The Harvard Medical School Guide To Suicide Assessment and Intervention.*

240 A.J. Sameroff and B.H. Fiese. *Conceptual issues in prevention.* In D. Schaffer, I. Phillips, N.B. Enzer, M.M. Silverman and V. Anthony (Eds.), *Prevention of Mental Disorders, alcohol and other drug use in children and adolescents.* 1989.

to suicide, from the stresses and strains of everyday life to severe psychopathology and trauma, including many life experiences, adaptive demands or psychological hardships. A tough life is not predictive of anything. Basically, life is tough, and in most cases a tough life does not lead to suicide. On the other hand, since life is tough, it would be almost impossible to find a suicide case that hadn't experienced a tough life.

Even though we know that severe, prolonged unmanageable stress, major life transitions and the lack of control over the environment are but a few of the conditions associated with increased risk for a variety of mental and physical disorders, we cannot predict with any accuracy which individuals will succumb to mental disorders. And, by contrast, strong social support, solid family relations, well-developed coping skills and self-sufficiency reduces the likelihood of mental disorders. Still, there are some that fall prey to mental disease in spite of enjoying all these advantages.

Basically, given the current knowledge of suicide and the methods available to predict and assess suicide, primary intervention becomes impractical and too costly because of the many false positives. The suicide rate of the general population being somewhere between 2-10 per 100,000 people makes it practically impossible to pin point such few individuals consistently.

Most assumptions dealing with suicide postulate that there are genetic predisposing vulnerabilities which interact with environmental stresses (the classical diatheses). Therefore, the modification of the environment is the only thing to be done because obviously, the genetic factors can't be changed. The question then becomes at which environmental level does the threshold of vulnerability kick in. The intervention would be, when the risk condition is amenable to change, to target and change that condition. Alternately, if we could identify the individuals that have a genetic risk, the goal of intervention would be to shift the individual's threshold along the diatheses-stress to reduce the risk.

Most of the few school-based intervention programs to reduce suicide have been based on some of these assumptions. When the program is person-centered, it targets risk behavior modification, information provision, behavioral or academic skill enhancement, social competence enhancement and motivational change and affective coping.

Other programs did not target at-risk students, instead efforts to engage others—parents, students, teachers, and administrators—in identifying at-risk students. One-third to one-half of adolescent suicides communicate in some way their intention to confidants. The logic being that if adolescents are trained to help identify suicidal peers (who in turn are more likely to confide in other teens rather than adults) many will be identified. However, the data regarding peer identification suggest that few peers subsequently confide in adults, and even that male students sometimes respond to distressed peers in unhelpful ways.[241]

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[241] Douglas Jacobs, *The Harvard medical School Guide To Suicide Assessment and Intervention.*

Programs based on the assumption that there is some specific deficit or competence in the person that requires bolstering focus on facilitating coping with life events and transitions, peer interaction enhancement, and setting related competence enhancement. These have been shown to be useful in environments where the lack of family support, or the nonexistence of family are the norm. Obviously, a solid family environment can make a huge difference in all aspects of children's lives, not only pertaining to suicide.

Young people in schools that serve low-level socioeconomic communities may be at risk simply because they come to school with lower levels of academic preparation than are necessary. In many such schools, the majority of students have lower-than-adaptive aspirations and expectations of the future—conditions that either relate directly to heightened levels of substance abuse and other risk behaviors associated with youth suicide, or predispose the students to other disorders, such as depression. Any improvement in these areas should be done for its own sake, and if in the process suicide rates are lowered, so much the better.

Understanding there is a lack of family support and following the same logic, other programs seek either to provide necessary resources or conditions that facilitate positive development and coping, and/or to modify or remove conditions that are developmentally damaging and hazardous. These programs lead to modifications of the schools' social climate, teacher expectancy messages, increasing ecological congruence and resource enhancement and opportunities, to mention but a few.

All these programs bring benefits, even though not necessarily relating to suicide prevention. For this to work, attempts to identify specific youths at risk of suicide has to be performed. Focusing on the vulnerabilities and symptoms, such as suicidal ideation, previous suicide attempts and major changes in habits or affect, is the best that can be done presently. Although some of these conditions may show us potential suicide risk candidates, they tell us little about what to do or change. Furthermore, they require the often ineffectual and inefficient as well as resource-expensive strategy of individual screenings.

As far as reducing suicide rates, many of the school-based efforts yielded quite disappointing results, and often brought new problems with them while they resolved others. Some of these prevention efforts have been shown to reduce rates of serious socioemotional disorder in children and youths and to be cost-effective. However, any reduction of suicide rates has yet to be adequately documented.[242]

The lack of understanding of the true nature of suicide continues to hamper efforts to reduce suicide rates. And here is another example of this: the effectiveness of suicide prevention centers and the Samaritans in preventing suicide is in dispute. Few studies that have found a preventive effect have shown a statistically significant one.[243] People on the verge of committing suicide simply don't start calling prevention

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[242] R. W. Maris, *Assessment and Prediction of Suicide.*

[243] Douglas Jacobs, *The Harvard medical School Guide To Suicide Assessment and Intervention.*

centers, it is highly unlikely that in that state of mind they would even remember the phone number or that such centers exist.

Eliminating suicide completely is an ideal, if unattainable, goal. However, if we consider that more than 90 percent of suicides involve some form of mental illness or personality disorder (and substance abuse, which might be a form of self-medication), it becomes obvious that identifying and treating mental disorders is the appropriate level of intervention to reduce suicide. Hopefully, effective intervention at this level would greatly reduce the need for tertiary prevention, and would greatly complement the tertiary intervention when needed. What should be obvious is that there are high levels of association between severe mental pathological conditions and suicide and these should be used as predictors with a caveat: most people with mental disorders do not complete suicide (except borderline personality disorder). Still, even though there is minimal risk of suicide, sufferers of mental disorders should be helped as much as those suffering from any other illness.

The ability to screen for early symptoms for mental illness and the increasing frequency with which care providers could treat, uncomplicated mental disorders such as anxiety and depression could provide a broad suicide prevention intervention program

Any effective intervention programs at the youth level, of course, would involve education on mental health, which would diminish (or hopefully eliminate) the stigma of mental disorders, and thereby, increase the number of young people affected who would seek help when affected by such disorders. Also, educating the young will, over the years, reduce among the population as a whole the stigma attached to anyone who suffers such diseases. Information about suicide and associated symptomatology, means of early identification and referral should be disseminated in detail at the high-school and college level. This doesn't mean in-depth courses on mental health. A 4 year program, perhaps once a year or semester a 2-3 hour lecture with a question and answer session is enough for juniors and seniors in high-school and freshmen and sophomores in college.

Once young people seek help or are identified as probable mental disorders cases, one of the first steps in assessing suicidal risk should be to look into past family history regarding psychiatric disorders, particularly ones related to suicidal behavior, and past suicide attempts, as these can influence a patient's thinking about suicide. The practicing therapist or psychiatrist should use collateral sources such as previous medical records, and consult with family and friends. This will give objective facts that can be compared to a patient's self-reports. Even if there is no family history of high-risk behaviors and events or other dysfunction in the family, if a mood disorder or a personality disorder has been diagnosed, a high suicide risk should be assigned to the case, and the patient and his family advised so. Emphasis should be made on educating all in contact with the patient on suicide symptoms, as well as informing the patient and family about the probabilities (in most cases) against suicide actually happening. It can not be stressed enough: education of the patient and his family is

of the utmost importance. Education of the therapist *should* be secondary, but in many cases is also necessary, as they are not aware, tragically in many cases, of how high (a 1 in 5 or higher) chance of suicide is present with some mental disorders.

Second, it is important to assess the patient's current state of mind, focusing on present mood disorders, past self-destructive behavior and previous suicide attempts. Prolonged or recurrent mood disorders are much more risky in terms of causing idiozimia, but past suicide attempts are indicative of having experienced archidiozimia or idiozimia in the past. Loneliness and hopelessness are symptoms that can indicate potential suicide risks, but not necessarily the cause of suicide attempts. Agitation, lack of concentration, lack of personal care and sleep disorders should all be warning signs to be taken seriously.

Men complete suicide at three times a greater rate than women, but women make more attempts than men. This is probably due to the fact that in men the threshold of the phobothymic switch is probably much lower and therefore men use more lethal means than women. In the U.S. men tend to use firearms and hanging, while women use poisons and drug overdoses.

There is a naïve tendency to think that most suicides among the young are related to interpersonal problems, whereas suicides among older people are often related to chronic illness and long-term financial losses. But one fact is true, the elderly use more lethal means to commit suicide (and are much more successful in completing their attempts).

Suicidal people are more overwhelmed by their daily lives, or perceived stress. The fact that they are archidiozimic or idiozimic makes them less adaptable because of greatly reduced, accessible memories; thus, they are less able to cope with stress and they experience more negative emotions. Suicidal people report distinct differences in life stress.

Not surprisingly, people with low levels of social support and with limited or nonexistent social ties are at much greater risk for suicide.

There is increasing recognition that mental disorders co-occur and/or evolve from one type to another. With co-occurring mental disorders, especially when adding anxiety and panic attacks, the risk of suicide is greatly increased.

In the appendix I offer an empirical (and I want to emphasize empirical) equation to determine the risk of suicide. It should be kept in mind that this is somewhat similar to predicting the weather. Conditions change rapidly and so assessments of suicide risk should be updated accordingly. It is extremely important to note that the numbers (or percentages) I am suggesting are, at best, a good guesstimate of mine. *Much more study should be made to determine what these numbers should be.* Hopefully these numbers reflect the complex interactions that lead to suicide. The effects of medication and therapy should be studied more closely and obviously subtracted. In essence, what we are attempting to do is determine the risk of becoming idiozimic, the one necessary condition for suicide to be carried out.

However, between 70 and 80 percent of suicides involve a diagnosed depression, and comorbidity of anxiety or panic attacks increase the risk of suicide significantly,

even though some researchers claim that panic attacks by themselves are not clearly associated with suicide. When personality disorders are related to increased rates of suicide, generally the personality disorder coexists with other high-risk disorders such as substance abuse or mood disorders.

The day before my son Mitch died, I was reading an article in Time magazine related to Freudian ideas. Four days after his passing, I stumbled on the open Time article, and the word aggression jumped out at me from the page. Immediately, in some intuitive manner, I understood that aggression was deeply connected with fear and panic attacks—that the fight-flight response has two sides. My intimate knowledge of my son allowed me to quickly understand a part of why he committed suicide. Mitch was a special case. If I wanted to help others, I needed to explain, not only the specific case of my son, but also all suicide cases. Eventually I was able to put it all into a scientific framework. This can only serve as a guide for future research, for better therapies and pharmacologies. Hopefully, soon, we will make advances in this direction.

Independent of everything, the truth is the system is poorly equipped to deal with most suicide cases. So what can be done now?

First, suicidal people fail to understand the true nature of their affliction. They underestimate the powerful forces of their depression and overestimate what they can achieve mentally and willfully. Partly for these reasons, and perhaps because of a certain shyness or even embarrassment, they do not share with others much of what goes on under the surface.

Second, suicidal people and their loved ones are almost completely unaware of how dangerous and how quickly the situation can deteriorate. Ignorance leads to a false sense of security. A strong support system should be in place involving family and friends.

Third, there is generally no one at high schools, less so in colleges, that are familiar with mental illness in general and suicide in particular, especially when this is a problem that has been on the rise among the young for the last few decades. At colleges there are medical services that might include several psychiatrists, but at present the diagnosis for suicide is not very good, because of the lack of understanding.

Fourth, the privacy laws concerning a doctor's diagnosis do not (unless special forms are signed) allow doctors to communicate with friends or relatives. Therefore, the necessary education of family to help achieve a successful resolution becomes less probable. If the patient denies the seriousness of his or her condition, as often happens, and she stops either her medication or the visits to the doctor or both, the family is not alerted of the lack of follow up that is so necessary. The privacy laws are such that physicians need to go out of their way to release information. In practice, they are too busy to do so. The privacy laws need to be changed so that family and friends—not everybody—can easily be alerted and warned about particular symptoms that can be lethal or which could be potentially indicative of suicidal behaviors in order to help prevent more unnecessary deaths.

In an interview with a psychiatrist of a patient who had committed suicide and knew he was suicidal, reveals that after a meeting with the family of the deceased, the mom

asked him, "Why didn't anyone tell us that he was suffering this bad?" The psychiatrist responded, "The sad fact is, he was an adult, and we don't call moms and don't tell moms about their adult children."[244]

Fifth, the failure of the medical profession is partly due to certain arrogance "that we are experts and know how things are supposed to be." I am not implying that such attitude pertains to all doctors. But this attitude, also present in most branches of human knowledge, is widespread in some parts of the medical community. Doctors spend little time explaining why they diagnose something, or what the diagnosis means, even less on how they decided on a particular treatment, and definitely, say nothing about what they don't know. I am not implying that there is malice. In many cases it is expedient, quick and easy. Doctors are just following a standard routine or procedure, get paid for it, and move on to the next patient.

Many times, perhaps, doctors should say, "I don't know," or "I don't understand." There should be some humility in the face of uncertainty and ignorance.

In many cases the message that gets through to the patient is, "The doctor is not listening to me." Or they yield to the professional in the white coat, "The doctor knows best." Or, in many cases there is an attitude that, "They can't help me," or "There is very little they can do," which paradoxically is true in many cases.

Sixth, the system does not educate the public. Doctors fail to educate patients and their relatives on the possible outcomes, particularly in the case of depression, manic depression and schizophrenia. It should be imperative that they alert their patients and family about dangerous possibilities, even though these might be considered low (by the doctors), like a one in five or one in six. At the very least, educate their patients in what they know and what they don't know. Educate their patients on dangerous signs, or if ignorant of this, at least alert the patients about the limitations of their knowledge.

Schools and Universities should have some programs to educate their students and faculty on the basics of mental disorders and suicide among the young. Even if only a few students or faculty are aware of some of the signs, help could be provided quicker in some cases.

We need to eliminate the stigma, the shame or embarrassment we feel about mental illness, and see it for what it is—a disease like any other. This requires extensive education of all. In particular, the people in contact with our young in our schools should be made much more aware of suicide, its signs, its prevention and cure. Psychiatrists and psychologists are continuously coming into contact with people suffering from mood disorders, and yet they are not alerting friends and families to the terrible dangers of these illnesses that in the extreme culminate in self-destruction. Short, informative lectures on mental disorders and their true nature, in the latter years in high school and early years in college will go a long way in educating our young.

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[244] Edwin Shneidman, *Autopsy of a Suicidal Mind*. 2004.

Generally this is not done, because of a bad cost-effective analysis. Statistically, in a high school of 500 hundred students (there are about ten thousand high schools in the country), a suicide might occur every two or three years, maybe. The point is that depression and other mental problems are more pervasive, and the stigma attached to them is what needs to be eliminated. The sooner a mental disorder is treated, the more likely a positive, life-time result can be achieved. The suffering caused by mental disorders should be treated aggressively, as soon as possible

Seventh, concerning certain mental disorders, there are severe limitations in what can be accomplished with medication or therapy today. Most therapies and medications are only scratching the periphery of the problem. For example, in depression, medications are concentrating on serotonin enhancing properties, instead of addressing the glutamate and GABA imbalance I propose. Low levels of serotonin, norepinephrine or dopamine are a result of this imbalance, and when medications correct levels of one of these neurotransmitters, in a few cases it helps, but in many it doesn't. In manic depression psychiatrists are mostly trying to control mood swing with lithium or other medications, again not paying attention to the glutamate and GABA relationship. Lithium contributes to slowing down the effects of sodium in producing neural pulses and indirectly regulates the excitatory nature of glutamate. In the case of schizophrenia, the use of psychotic drugs, mostly dealing with dopamine, is partially on track, but psychiatrists are disregarding the fine balance between dopamine and acetylcholine that I propose.

We need improved therapies. Therapies are only partially successful, and sometimes only in a small percentage of cases, because they are not addressing the real connections between memory, thinking and emotions. Emotions have the purpose of eliciting certain modes of thinking and activating related memories. Conversely, certain types of thinking or memories can elicit related emotions, but this two-way system is stronger in the former case. Therapies, in a similar manner as medications, for certain mental disorders fail to address this fundamental issue except in a roundabout way. When therapies achieve a partial improvement or even enjoy a success, it is only in very specific cases where the peripheral issues indirectly alleviate the problem. However, at the very minimum, therapy helps to make some sense of the confusion, can return some sense of control, and gives hope.

Eighth, we need to change the laws concerning insurance of medical benefits with respect to mental disorders so they are properly covered and included in all health policies. This requires education of politicians. It is imperative that financial reasons should not get in the way of treating mental sickness.

There is a tendency in mental health policy, because of new drugs, to reduce hospitalization, decrease use of involuntary commitment and these organizational changes presage greater reliance on patient knowledge and self-determination, and constraints on effective (insured) demand for services, which would make it necessary for mental health providers to anticipate the need for bearing greater risk with patients who are suicidal. All this is unlikely, unless mandated by law.

Ninth, limits on lethal weapons. In the U.S. sixty-five percent of all teen suicides are committed with guns. Every time a gun is used to prevent a crime it is used sixty-seven times as often in an accidental or suicidal death. In many cases, if the means to suicide are not immediately available, the short window of impulsivity that leads to an act of suicide passes and for the future the person might be forewarned that they can become suicidal and seek help.

Over a period of five years, in part, because most of the time my son Mitch was fine, all these things conspired against him. Many small mistakes (or ignorance) stacked on top of one another, lead to a deadly end. Anyone of these small mistakes, alone, would not be fatal. Paradoxically, if Mitch's condition had been worse, any one of these factors might have been changed, and affected some of the others, and his death avoided.

Our first step is changing or improving the situation in each and every one of the issues mentioned. This is the basic goal. But, keep in mind that any change that can be affected in any of these areas will decrease the chances of suicide.

The second, and more difficult step is to use the scientific framework provided here to guide us in creating better therapies and more selective pharmacological solutions. The direct interrelation between emotions and memories should be explored better. The deep connections and relations that exist between facial expression, gestures and body postures and emotions and vice-versa should be brought to bear on mental problems. Dancing, singing, even acting should be explored as therapeutic tools. Physical contact, such as massages or caresses, or even, perhaps, sexual activity, should be explored as part of the arsenal to correct mood disorders. Physical activity and exercise should be essential elements in restoring health. Therapies should include emotional transference and countertransference, not only between patient and therapist, but between patient and loved ones, because they are a daily occurrence; social interactions and intimate relations should be promoted at all costs. I am confident that a few are already incorporating some of these ideas into psychotherapies as I write. It is essential to understand why some of these things work, because then, advances can be made much faster. And it is long over due.

Appendix

L=(SMD+SPD+R+O+SI+G+PA)

L=Lethality
SMD=Severeness of Mood Disorder (ranging from 0.0 to 0.1 from none to extreme; if two concurrent mood disorders are diagnosed, they should be added, i.e., schizophrenia and depression could add to 0.2)
SPD=Severeness of Personality Disorder (0.0 to 0.1 from none to extreme)
R=Recurrence or length of suffering (0.1 per year or per episode, whatever is less)
O=Other aggravating situations, such as panic attacks or anxiety attacks, anger, hopelessness, guilt or anhedonia (from 0.0 to 0.1 for each)
SI=Suicidal Ideation. SI=Plan+Means+Knowledge (each element from 0.0 to 0.1 From none to good, with a total of 0.3 if all are "good")
G=Gender (male=0.1, female=0.03)
PA=Previous attempts (0.05 for one 0.1 for 2 or more)

For example, a male (G=0.1) person with a moderate depression (SMD=0.05), that has suffered this condition for five years (R=0.5) with severe panic attacks (O=0.1), with no plan to commit suicide (plan=0.0), but with the means (owns a gun or has access to a rope, means=0.1) and the knowledge (knows how to use the gun or the rope, knowledge=0.1, then SI=0.2), lethality (L) would be L=0.95. Lethality could be greater than one. Even though probabilistically it doesn't make sense, it means that if a suicide attempt is performed, the success (if such a thing can be called success) of the attempt is that much more certain, or in the case or risk assessment, increases the chances of a completed suicide.

R= L(MD+PD+AA+PA+A+LC+SD+PA+DC+SA-MT)

L=Lethality
R=Risk in percent
MD=Mood Disorder (Unipolar depression 20%, bipolar depression 40%, schizophrenia 15%)
PD=Personality disorder (Borderline personality disorder 10%, other personality disorders 2-5%)
AA=Anxiety attacks or severe anxiety (10%)
PA=Panic attacks (10%)

A=Severe agitation (10%)
LC=Lack of concentration (10%)
SD=Sleep disorders (10%)
PA=Previous suicide attempts (0.05 for one or 0.1 for 2 or more attempts)
DC=Drastic changes (0.0 to 0.1 for each such as neglect of appearance, loss of favorite activities)
SA=Substance abuse
MT=medication and/or therapy (0.1 for each, but if receiving both, 0.2 for each, as there is an obvious additive effect)

For example, a person with unipolar depression (20%), severe panic attacks (10%), severe agitation (10%), lack of concentration (10%), sleep disorders (10%) and no previous suicide attempts (0%), with a lethality of 0.95 would be considered a suicide risk as R=0.57%, or a little bit less than 2 out of 3 chance of a completed suicide. This hypothetical case is a very high risk. However, if you change unipolar to bipolar, then R=0.76, or a 3 in 4 chance of a completed suicide. In the first case mentioned, if receiving present medications and therapy, then the risk of suicide would be lessened to R=0.19, or less than 1 in 5.

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