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## Depression. An Overview with a focus on ECT

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Depression is an illness and like any illness it has an impact on biological, psychological and social functioning. Individuals may experience job difficulties, interpersonal difficulties and a variety of physical symptoms that may mimic other conditions. It is common for the patient to attribute the experience of depression to a life event, for example, the failure to get promoted or marital stress. It is human to make meaning of our experiences. It is important for clinicians to remember that the meaning we make does not always have etiologic implications.

Years ago when I was a medical intern, I had the task of telling a man that he had Hodgkin's disease, a potentially fatal cancer. Nothing I could say could convince him that he did not "catch" Hodgkin's disease because he went out for a walk on a cold rainy night in Venice, during a recent European vacation. A few weeks after his damp walk he developed chills, fever and headaches - the first symptoms of Hodgkin's - that were enough like a common cold that he was convinced that his failure to carry an umbrella caused the disease.

My Hodgkin's patient's explanation served two purposes. It explained something medical science could not explain, "why him", and it gave him some sense of control over an otherwise inexplicable life-threatening event. Patients suffering from depression and their friends and families engage in a similar process of trying to explain the illness. Social factors such as the depressed individual's recent relocation to a new city leaving them socially isolated or

psychological factors like the failure to get a much- desired raise are often offered as explanations of the cause of illness. Like my Hodgkin's patient's experience of cold and coryza that felt like an ordinary flu, patients with depression often experience it as the outcome of immediate life events.

Social and psychological events are closer to everyday human experience than neurotransmitter dysfunction in the central nervous system and are readily identified as causative agents. It was an intimate part of my patient with Hodgkin's disease experience to wander out on a cold damp night and wake up with symptoms of a cold. His explanation of his cancer gave him a sense of control and understanding that medical science could not. From his vantage point his explanation of his illness had more power than any I could offer.

When the depressed patient offers physical symptoms as their initial complaint their condition can be confused with a neurological disorder if memory problems and headaches figure prominently in their presentation or cancer if weight loss and fatigue is prominent. In fact, depression has replaced syphilis as the great imitator of other conditions. This is in part due to the myriad of physical symptoms that can plague the depressed individual but also to the stigma attached to having a psychiatric disorder in our culture.

This stigma plays an active role in the course of depression and is in and of itself a major public health issue. Individuals are reluctant to describe psychological symptoms such as inexplicable sadness or uncontrollable crying to their physicians. Likewise, their physicians are often reluctant to ask about such symptoms. The hesitancy by both the patient and the physician to speak directly to psychological suffering is related to a prejudice that depression is not a real illness like pneumonia.

There is no laboratory test for depression to lend confirmation to a clinical diagnosis. Yet

depression is a highly treatable disorder with 70 to 90 percent of depressed patients recovering. It is also a very common disorder. A recent national survey showed that during any given year more than 10% of all Americans (almost 26 million people) suffer from major depression and related mood disorders. Unfortunately because of the stigma patients are reluctant to tell their doctors that they feel sad because they fear it is a sign of weakness and doctors hesitate to ask. This leads only one in three depressed patients to seek treatment and only half of those who do seek treatment to be correctly diagnosed.

The parallel to syphilis extends to diagnosis and treatment. Even after effective treatment for syphilis was available patients did not readily offer information about their sexual histories. Physicians were also reluctant to probe. Even after a laboratory test was developed for the disease the diagnosis was often missed because physicians were reluctant to order the test out of fear of embarrassing their patients. It was only after it became a required screening test in order to obtain a marriage license and a routine test upon hospital admission was the disease more commonly diagnosed. Clearly prejudice and stigma interfere in the treatment of illness.

Depression also shares with syphilis a significant morbidity and mortality rate if untreated or inadequately treated. The human cost of untreated depression is striking. Lost productivity at work, sick days and suicide are all sequelae of depression. There are more than 36,000 suicides annually in the United States, making suicide one of the five leading causes of death in adults and one of the three leading causes of death in adolescents. Fifty percent of these deaths are attributed to untreated depression in one of its forms.

Because of the stigmatization of mental illness people are dying. It is an obligation of health care professionals to speak out against prejudice and work to overcome stigma through education. One of the difficulties in understanding the illness is the poor choice of the word

"depression". This reflects an unfortunate tendency of psychiatry to borrow ordinary words from common usage and use them in uncommon ways. We speak of feeling "depressed" when it rains on Saturday spoiling a weekend outing or our favorite team loses a ball game. We extrapolate from this to the experience of our ill patients. Yet it remains a poor description of an illness that affects appetite, sleep, energy, sex drive, concentration, memory and not consistently mood.

William Styron, in a remarkable memoir of his experience of depression, writes eloquently of this linguistic error.

*"As one who has suffered from the malady in extremis yet returned to tell the tale, I would lobby for a truly arresting designation. Brainstorm, for instance, has unfortunately been preempted to describe, somewhat jocularly, intellectual inspiration. But something along these lines is needed. Told that someone's mood disorder has evolved into a storm - a veritable howling tempest in the brain, which is indeed what a clinical depression resembles like nothing else - even the uninformed layman might display sympathy rather than the standard reaction that depression evokes, something akin to "So what?" or "You'll pull out of it" or "We all have bad days." The phrase "nervous breakdown" seems to be on its way out, certainly deservedly so, owing to its insinuation of a vague spinelessness, but we still seem destined to be saddled with depression until a better, sturdier name is created."*

I read this passage from Styron and remember the time I ran a psychiatric section in a general hospital that was located in an attached wing of the main medical/surgical section. One day in the Doctors' Lounge a colleague pointed over to the psychiatric unit and said, "What's the matter with those people over there? They go in the hospital because they're depressed. I get depressed and I play golf to feel better. Someone ought to smack some sense into them."

Perhaps if I had told him that they were in the hospital because their brains were on fire

he would have understood better.

## THE NATURE OF DEPRESSION

### *The Subjective Experience*

Like most illnesses, the depressed patient's experience of their condition varies widely. This should not be surprising given that depression does not discriminate between its victims and effects all age groups, sexes and socio-economic levels. Individuals experience themselves as physically ill, losing their minds or more subtly in the chronically depressed patient - as not ill at all - but merely suffering from existential angst that affects their outlook on life.

Depression can have widely different onsets and courses in individuals - thus affecting the subjective experience of the illness. Typically depression occurs acutely with symptoms unfolding over a several week period and without treatment resolves in about six months. It may occur in the context of significant psychological or physiological stress or occur when there are no apparent stressors. When stressors exist the individual and his or her immediate family is likely to attribute the illness to a failure to cope with the stress. Depression is more likely to be

experienced as a medical condition when its onset is sudden and there is no apparent environmental cause.

Despite its name, a depressed mood is a variable symptom of depressive disorders. Many patients who become acutely ill feel that something is awry with their bodies. They have headaches, feel fatigued but sleep poorly and lose their desire to eat. An individual may have difficulty focusing or completing a task leading to poor work performance or an inability to work depending on one's profession. The degree of physical concerns runs a continuum from complaints about specific symptoms such as, headaches, to delusional concerns, such as a belief that one's body is rotting, or riddled with cancer. In recent times the practitioner often has to manage the conviction that the patient has AIDS that no amount of reassurance or negative HIV testing can alleviate.

Gastrointestinal complaints are quite common. These range from sensations of cramping, an aching pain to diarrhea and increased flatulence. It has been estimated that more than half the individuals diagnosed as having irritable bowel syndrome suffer from depression. Consequently, it is common in some communities with a high concentration of medical specialists, for the depressed patient to have upper and lower GI series before presenting to a psychiatrist for an evaluation.

This category of subjective experience that emphasizes primarily physical concerns often leads to misdiagnosis and mismanagement of the illness. In my clinical practice patients who experience a variety of physical symptoms are likely to come to my consulting room only after they have had extensive medical work-ups for medical diseases ranging from brain tumors to an occult malignancy. While many patients find it reassuring to have a normal battery of tests, the act of simply ordering the test can deepen the conviction of serious disease for some patients.

This is particularly problematic in the patient for whom somatic symptoms have taken on delusional proportions.

In order to assure a therapeutic outcome, any approach to evaluating the depressed patient must weigh the relative need for a complete medical work-up with the effort to avoid worsening the patient's potentially delusional concerns. The depressed patient often experiences themselves as losing control of their mind and their body and extensive medical evaluations add to the concern. In addition, the patient may be worried about the effect of medication on their mind and body and consequently be reluctant to accept antidepressant treatment.

*Mary*

*Mary B. was a 24-year-old single female graduate student who had just begun a new academic year when she began to have trouble sleeping and lost her appetite. Over a three-week period she lost ten pounds and found it increasingly hard to focus on her work. She became preoccupied with a brief relationship with a fellow student three years previously during which time she thought she could remember at least one episode of unprotected sex. She went to the University Health Service and requested HIV testing. She was seen briefly by an intern who told her she probably had a viral syndrome but agreed to do the HIV testing. Although the test results were negative, she was convinced that the physician would not have ordered it unless he too believed she had AIDS. She ruminated about his casual use of the term "viral syndrome". AIDS is a virus; he probably just didn't want to frighten me by saying AIDS. But why else would he say "virus"? She returned to the University Health Services and demanded that they tell her the truth about her HIV testing. Alarmed by her agitation an emergency psychiatric consultation was requested. Seeing a psychiatrist further frightened the patient and several hours went into*

*convincing her to start medication for depression.*

The difficulty that treaters face with evaluating patients like Mary is that they present with significant physical symptoms - weight loss, diminished concentration and at times pain e.g., severe headaches. In approaching the depressed patient it is imperative that depression be understood and presented as a serious illness, not a diagnosis of exclusion. Depression is the most common problem in all patients who see their family doctor or other primary care practitioner and it is the diagnosis most often missed. Since depression can be both an early sign as well as a result of physical illness the problem of diagnosis may seem complex. Our current nosology offers some solution to the problem primary care clinicians face in deciding when to look for cancer, multiple sclerosis or thyroid disease. DSM-III separated primary and secondary dysthymia - "secondary" designating a relationship to a pre-existing psychiatric disorder or medical illness. This distinction has been eliminated in DSM-IV because of its lack of usefulness. In practical terms, when depression is present it should be diagnosed and treated and the patient educated as to its nature. Too often the search for co-existing medical problems takes precedent over the treatment of the clearly present depression - adding to the patients suffering and concern.

Depression is a physical illness. In fact depressed patients suffer more than most patients with chronic physical illnesses. Among serious illnesses, only arthritis causes patients more chronic pain and only advanced coronary artery disease causes sufferers to spend more days in bed. Unless depression is recognized and diagnosed early it is difficult to address seriously.

A patient I have treated for more than a decade now gets depressed in much the same way as Mary. One of the first symptoms of her depression is the morbid conviction that she has AIDS. Every bump and freckle that she has had for years is painfully experienced as absolute

proof that she is dying from AIDS. After much experience the patient now will call and say "I know I'm getting depressed again, I think I have AIDS". This level of understanding and knowledge about her illness is what we must strive for in all our patients because it leads to better disease management. This can only be achieved by correct early diagnosis and patient education.

A final category of depression that has profound effects on the individual's experience of himself or herself is the category of illness that is chronic and in what is somewhat of a misnomer often referred to as milder forms of mood disturbance. Dysthymia and its companion disorder, cyclothymia, are pervasive, chronic and at times subtle disturbances of mood that can effect one's core identity. Because these fluctuations in mood are milder than full-blown episodes of depression or mania they are likely to be attributed to the individual's personality. In fact, both conditions are commonly confused with personality disorders. The humorlessness, anhedonia, gloominess and judgmental attitude can be mistaken for personality traits by both the clinician and the patient. Avoidant, passive-aggressive, dependent and borderline personality disorders all have characteristics in common with these mood disorders. Subjectively, the individual with a chronic mood disturbance that is discontinuous from their environment often experiences their symptoms as part of their identity.

In fact this area of overlap between the personality disorders and mood disorders offers sobering lessons in psychiatric treatment. Many of these patients were treated for decades as having personality disorders with the mood disorder largely unaddressed. One of the unheralded side-effects or perhaps benefits of the SSRIs is that their relatively low risk/benefit ratio led to the treatment of patients who in the past would not have been considered "sick enough" for pharmacotherapy. Some of the remarkable personality changes attributed to prozac when it first

became available was no more than its exerting an antidepressant effect in individuals who previous to its availability would not have been considered depressed enough to receive treatment.

Prior to the availability of the SSRIs pharmacotherapy was complicated by the both the relative risk of side effects of the medications and their relatively low LD-50 (the amount of medication consumed that would kill 50% of the individuals consuming it). Medications were reserved for individuals with severe symptomatology and other individuals; the overwhelming majority of patients with dysthymic disorder for example, were treated with psychotherapy. When prozac became available with its relatively benign side effects and almost nonexistent risk of overdose it began to be used in the population of patients with less severe symptoms. Clinicians were surprised at the difference it made in this group. Effects on personality that were initially attributed to prozac were actually no more than the disappearance of the depression and the amelioration of its effects on the individual.

This overlap between the symptoms of mood disorders and the symptoms of personality disorders still has enormous clinical implications in the treatment of the refractory patient. There is still a tendency amongst psychiatrist to revert to a personality diagnosis in patients who are medication resistant. This will be discussed in more detail in the section on ECT.

## THE ECONOMICS OF DEPRESSION

At least 11 million people in the United States (6% of the adult population) have an episode of depression each year making depression a major public health issue. Despite the fact that it is the most common problem of patients consulting primary care practitioners the correct diagnosis is often not made but instead the concomitant physical symptoms are evaluated and treated. Consequently it is dismaying but not surprising that of the annual \$44 billion cost of the disorder only 28% is the direct cost of treating the disorder.

Depressed patients often present primary care physicians with somatic complaints such as lethargy, anorexia, headaches, stomachaches and insomnia and tend to be high utilizers of medical services. Depressed patients use healthcare services three times more often than nondepressed patients. The depressed patients' medical costs, even after controlling for severity of general medical co-morbidity, are twice as high as costs for nondepressed patients. They also make seven times more visits to emergency room than patients without depression. Not surprisingly the annual nonpsychiatric healthcare costs of those patients with depression (\$4,246) is almost twice as high as individuals without depression (\$2,371).

Depression has a significant economic impact on the workforce as well. Absenteeism and lower productivity is estimated to account for 55% of the annual cost (\$24 billion). This amounts to \$3000 a year per depressed employee compared to \$180 a year for nondepressed employees.

18,000 depression-associated suicides account for 17% of the cost. This is a startling figure when compared with the annual expenditures on antidepressants, which account for only 3% of the total cost or \$1.2 billion.

In America the third-party reimbursement system often encourages the under diagnosis and under treatment of depression. Many managed-care companies separate the management of psychiatric services by "carving out" the mental health benefit and contracting with a specialized company to manage the care. Because these companies are usually paid a set amount of money to provide psychiatric treatment the fewer patients they treat the more money they're likely to make. The problem with this is that it incentivizes them to encourage underutilization of psychiatric services. To compound the problem primary care doctors are often not paid for treating psychiatric illness, which discourages the diagnoses of depression and encourages diagnoses such as headache or fatigue syndromes. As a consequence, looking for new cases of depression that have not been treated, often called "case finding", is economically discouraged. Recently there has been a movement to demonstrate the economically added value of case finding in psychiatry.

## ELECTROCONVULSIVE THERAPY

*A young healthy man who has offended the head psychiatric nurse is wheeled into a surgical suite restrained to a large hospital cot. While he is fully conscious and screaming, electrodes are applied to his head and a doctor administers an electric current. The man screams and writhes on the cot as the current passes through his brain. His body rises against the restraints and he has a full-blown grand mal seizure.*

This remarkably powerful scene from *One Flew Over the Cuckoo's Nest* has had an enduring effect on attitudes about ECT. It is striking how many individuals, both professionals and nonprofessionals alike, have formed their image of ECT around this scene from a

Hollywood movie. No matter that this depiction of ECT is the equivalent of portraying open-heart surgery as something that is done to patients to punish them and typically is done without anesthesia while they are awake. If such a scene were aired in a horror movie it would not likely result in a clamoring to ban coronary bypass surgery. Yet *One Flew Over the Cuckoo's Nest* in many ways had just that result for patients suffering from depression. For more than a decade, from the early seventies into the early eighties, ECT became a rarely used treatment. From the perspective of a cultural critic - ECT in this movie metaphorically represented the coercive nondemocratic use of power and clearly had as its reference point the U.S. military during the Vietnam War. Unfortunately this scene has endured for many people, including healthcare professionals, as a realistic representation of ECT treatment.

Of course, one could argue that ECT fell into ill repute because it was over-utilized and utilized in clinical situations where it wasn't indicated. Continuing the parallel with cardiac surgery, we know that over a ten year period from the early seventies to the early eighties, approximately 50,000 bypass procedures were performed a year in this country. We know now that 50% of those procedures had no therapeutic effect and were unnecessary. Given that bypass surgery has about a 1% mortality rate (compared to 1 in 10,000 for ECT), these statistics mean that 2500 people died over a ten-year period from surgery that had no benefit. Why wasn't there a clamor to ban cardiac bypass surgery?

While the comparison is dramatic, the attitude about ECT is informative. Jack Nicholson's portrayal aired at a time that psychiatry was in the beginning stages of a paradigm shift. The existing paradigm in the early seventies viewed psychiatric illness as the result of moral backsliding, or moral weakness, psychological factors related to the effect of childrearing patterns or in the political climate of the times - an effort of the prevailing social powers to label

its dissidents. This paradigm made it easy to accept the portrayal of ECT as a potent political weapon used to curb nonconforming behavior. The current paradigm, that views mood disorders as the result of genetic, physiologic and to a lesser extent, psychological variables has resulted in ECT being viewed appropriately as no more than an effective treatment in the clinicians' armamentarium in the treatment of depression.

Numerous studies have shown that ECT is the most effective treatment for depression with a response rate of about 85 to 90% compared to 55 to 60% for medications. ECT is also effective for mania but is rarely used for this purpose because of the relatively high response of manic patients to medications.

The risks of ECT, like all treatments, have to be weighed against the benefits. ECT is the fastest acting antidepressant treatment with patients starting to have symptom relief after one or two treatments. Typically a series of treatments is needed - averaging about six over a two-week treatment. Sometimes patients recover in as few as three treatments or need as many as fifteen.

ECT is the induction of a grand mal seizure through the application of an electric current in a patient who is both asleep and transiently paralyzed. Because a seizure is necessary for a therapeutic effect - the high profile side effects of ECT - confusion and memory disturbance often occur. Seizures typically cause a retrograde amnesia where individuals lose their last several hours of memory. There may also be transient disorientation and confusion. A recent study that tested memory function before ECT and two months after the treatment found that patients', memory functioning had improved.

Until very recently ECT had been used primarily as a treatment of last resort when all other interventions failed. Because of its effectiveness and rapidity of action this has been changing. A recent editorial in the New England Journal of Medicine suggested that it be

offered to patients early in their course of depression as an appropriate treatment option. When the effect of ECT (quick response rate, high efficacy rate) is weighed against medication (slower response rate, lower efficacy) some patients may opt for ECT after several medication trials rather than suffering with months of illness.

#### *JUDGE P*

*Judge P is a 58 year old Superior Court judge who began to experience difficulty eating, difficulty sleeping, a sense of inner turmoil, dysphoric mood and the persistent belief that an affair that he had twelve years previously would be made public and his career would be ruined. His psychiatrist began him on fluoxetine and haldoperidal and he experienced little response. Augmentation was tried with desipramine then lithium, again with no response. After six weeks of persistent symptoms he was hospitalized because of suicidal ideation. Despite both persecutory and somatic delusions he recognized that he was suffering an illness and requested ECT treatment because it was the best chance of returning to the bench quickly. He underwent a series of six treatments that resulted in a complete amelioration of his symptoms and he was back in court in less than three weeks.*

Clearly the choice of ECT has to be weighed against the patient's illness, their physical health and their treatment history. But this choice should be made based on rational facts not Hollywood movies. ECT is both a simple and yet a complex treatment for depression that has been evolving over the last 65 years. In order to fully appreciate the use of such an aggressive treatment for depression the clinician must appreciate depression for what it is - an illness that can be life threatening, and as debilitating and impairing to quality of life as any form of cancer.

ECT has its origins in the work of Ugo Cerletti an Italian neurologist who was studying epilepsy in Rome in the 1930s. Working to develop an experimental model of epilepsy Cerletti

used chemicals and then electrical current to induce seizures. Through experimental work with animals he found that the passing of an alternating current of 125 volts through the head for a fraction of a second caused epileptic forms of attack which were not life threatening. Prior to using electrical current to induce seizures chemical means such as camphor had been used. The results from chemically induced seizures were too unpredictable and difficult to control to be clinically useful.

Building on the foundation of using electrical energy to induce seizures in animals it was noted that patients who were epileptic and depressed experienced an amelioration of their depression following an epileptic attack. Cerletti postulated that inducing a seizure in a depressed patient who was not epileptic would have therapeutic results. In 1937 he was asked to consult on the case of a man found in the railway station in a near-catatonic stupor. Modifying the device he used in animals Cerletti treated the patient with a series of electrically induced seizures over the next two weeks and described a complete resolution of his symptoms. This treatment represented the beginning of the use of electrically induced therapeutic seizures in psychiatry.

Over the next three decades ECT was used widely as a treatment for severe psychiatric disorders. It was used with mixed success to treat disparate conditions including schizophrenia, bipolar mania and depression, delusional depression, unipolar depression and obsessive-compulsive disorder. The enthusiastic use of ECT was in part due to its perceived efficacy in a number of dire clinical settings but also due to the lack of any effective pharmacological agents until the 1950s.

Although ECT was widely used little advances were made in the technique until the early 1960s with the introduction of modern anesthesia. The use of anesthesia, in particular,

neuromuscular blocking agents, eliminated many of the potential severe physical side effects from the treatment. In addition, a major psychological problem with the treatment - inducing a seizure in a still conscious patient - was also eliminated.

The next major development in ECT was the introduction in the 1980s of pulse wave machines. Until then ECT apparatus administered energy in the form of a sine wave (see figure 1). The pulse wave apparatus allows for significantly better control of the energy wave and consequently allows for titration of seizure threshold. The widespread use of the pulse wave machine led to the growth of research into the relationship of the seizure threshold and clinical response. This will be discussed further on in this chapter.

The third procedural modification to advance ECT has been the use of concurrent EEG monitoring. Prior to the use of EEG monitoring in the late 1980s practitioners relied on the observable seizure as a measure of treatment success or failure. It was subsequently documented that the correlation of total seizure duration was consistently underestimated by using the observable motor seizure and not an EEG measurement. Because of the observation of the lack of correlation in addition to the increasing interest in seizure morphology as a determinate of seizure efficacy it has become standard clinical practice to monitor at least one channel of EEG reading during the treatment.

The fourth major change in ECT treatment is still unfolding. There is increasing evidence that the placement of the electrodes and the energy level used to induce the seizure effect both therapeutic outcome and the potential for cognitive side effects. Three different lead placements are used in clinical practice along with careful titration of seizure threshold and stimulus intensity. The clinical significance of these lead placements will be discussed below in the section on *The Procedure*.

### *Patient Criteria*

When and for whom is ECT an appropriate treatment? Unfortunately the answers to these questions can vary widely based on the preferences of the individual psychiatrist, the treatment center and at times the availability of the treatment. There are four general criteria that inform the decision to administer ECT. The first and most obvious is the patient diagnosis. ECT has demonstrable efficacy in the treatment of depression in all its major forms. This includes major depression, bipolar depression and major depression with psychotic features. Depending on the study ECT's efficacy ranges from 60-90%. These figures are more dramatic when considered in the context of the literature because ECT is rarely used until the patient has tried at least several antidepressants so a bias toward a treatment resistant population is built into many of the studies. The data for major depression with psychotic features is both dramatic and intriguing. In this patient group the response rate is approximately 90%. This figure is so superior to the response rate to medication or even the response rate to ECT of major depression without psychotic features that it has led some investigators to suggest that psychotic depression may be a separate biological entity. There is little data for using ECT to treat Dysthymic Disorder largely because it is not perceived as a serious enough illness to consider this form of treatment. Experience using ECT to treat anxiety disorders has been disappointing. Trials of ECT with intractable OCD have shown no efficacy in the absence of co-morbid major depression. It is also ineffective for the treatment of chronic schizophrenia although some studies have show improvement in positive symptoms when used conjunctively with clozapine.

ECT has also been shown to be effective in the treatment of bipolar, mania, although it is rarely used for this condition currently because the response rate to medication is so high. Bipolar patients can at times run the risk of induction of mania when a depressive episode is

being treated although not with the frequency that this occurs with antidepressant therapy.

*Will was a 28-year-old graduate student with a family history of bipolar disorder who presented with profound melancholia, anergia, anorexia and suicidal ideation. After three antidepressant trials on an outpatient basis he was hospitalized following a suicide attempt by drug overdose. He underwent a series of 6 bilateral ECT treatments leading to a resolution of his depressive symptoms. Three days after discharge he returned to the inpatient unit and began yelling at the nurses' station that his graduate dissertation had been stolen from him and demanded its return. On examination he claimed total memory loss for his thesis and insisted that the ECT had erased it from his mind. On further interview he revealed racing thoughts, flight of ideas, pressured speech and persecutory delusions. It was concluded that he was in a manic state and he was readmitted to the hospital and required several more ECT treatments to resolve the mania. Interestingly, his memory of his thesis work returned when his thoughts became more organized and his mood returned to a euthymic state.*

The next two important criteria in patient selection are linked – the current clinical status of the patient and the history of previous treatment and response to treatment. An initial course of ECT is rarely the first treatment modality except in a psychiatric emergency. Psychiatric emergencies that necessitate immediate initiation of ECT are circumscribed. They are confined to malignant suicidality to the extent that the delay of a pharmacological response could be life-threatening and likewise dense catatonia where the nutritional status of the individual is such that a delay for pharmacological response could be equally life-threatening. Even in these situations emergency medication intervention is almost always utilized before ECT is initiated.

Because these situations are relatively rare the decision to implement ECT is usually based on the longitudinal history of the illness including drug trials, the patient's current clinical status and the patient's preference. A general clinical rule of thumb is a trial of at least three appropriate medications for an adequate time period at an adequate dose. Like all clinical rules individual patient characteristics require flexibility. While previous medication trials are appropriate there are often times that more than four trials occur before ECT is thought of as an option. Likewise, patient preference may play a role in the decision for earlier use of ECT. At times adequate medication trials are not possible.

*Mary is a 54-year-old white female with severe Crohn's Disease and Irritable Bowel Syndrome. Because of her GI disease she was unable to tolerate and adequate trial of fluoxetine, paroxetine, sertraline, amitriptyline, nefazadone, or mirtazapine. All trials at sub therapeutic doses resulted in severe GI symptoms. Mary experienced chronic dysphoria, insomnia and intense psychic pain. After consultation a trial of supra-threshold right unilateral ECT was initiated. The patient responded to an acute series of 7 treatments with a marked improvement of her symptoms. This was followed by a tapering dose of ECT out to monthly intervals. She was then treated monthly for one year while she remained symptom free.*

The fourth criterion for patient selection is physical health. While there are no absolute contraindications to ECT there are many health factors that complicate the risks of ECT. The aspects of ECT to be considered during patient selection are the patient's ability to tolerate general anesthesia and their ability to withstand the physiologic effects of the treatment. The risks of the anesthesia are relatively minor and rarely at issue. There is always a risk of an untoward reaction to the anesthetic agents – the incidence is about 1 in 100,000. The risk is increased in patients with significant pulmonary disease or upper airway disease that might

complicate airway management during the procedure. Significant obesity is the most common factor to complicate airway management and may necessitate prophylactically intubating the patient during treatment. Severe asthma can also lead to the development of problems with the airway during treatment and needs to be evaluated carefully before treatment. Patients on bronchodilators are commonly prescribed them just prior to initiating the treatment procedure.

Two physiologic factors about the treatment itself are germane to the patient's medical status. The induction of a grand mal seizure during ECT leads to a transient rise in CNS pressure. Any condition in which raising the CNS pressure is contraindicated needs to be carefully evaluated. These tend to be space occupying lesions, recent CNS events e.g., bleeds, aneurysms and venous malformations. The treatment also results in significant hemodynamic changes that can lead to brief asystole, hypertension, hypotension, tachycardia, bradycardia and both atrial and ventricular arrhythmias. While these are typically mild and self-contained the individual's premorbid cardiac status needs to be considered carefully.

We now do a form of ECT referred to as cardiac-modified ECT in which patient is pretreated with glycopyrolate or atropine to prevent the vagally mediated bradycardia and labetalol to prevent hypertension. While there is always the potential for cardiovascular complications pretreating the patient greatly mediates the risk. The patient with significant cardiac disease may need a change in induction agent. Etomidate which has the least cardiac effects is often used in these cases.

There remain no absolute contraindications to ECT. In the presence of significant medical risk it is important to weigh the risk of treatment versus the prolongation of the depressive disorder.

About fifteen years ago I consulted on the care of *John*, a 48 year old white male who developed a depression characterized by anergia, apathy, worthlessness, dysphoria and a near-psychotic conviction that he was dying and could no longer care for himself. John had cerebral palsy with significant motor impairment but was able to walk with arm and leg braces and ran his own successful business. He developed a series of significant side effects to medication trials. Fluoxetine caused anorexia; desipramine resulted in SIADH; perphenazine resulted in NMS; and lithium resulted in severe tremors. After 6 months he remained wheelchair confined because of the anergia and apathy. He also had lost 20 pounds over the same period. Because of his near-delusional depression I recommended ECT. A routine MRI was ordered because of his treatment resistance and his cerebral palsy. The MRI revealed a moderate sized aneurysm. Neurosurgical consultation estimated the risk of rupture at 1% per year. The neurosurgeon estimated the risk of rupture at 2% per ECT treatment. Because of the patient's history of significant side effects from medication and his not insignificant risk of a CNS bleed I recommended against the ECT and the patient was treated with continued pharmacological trials. It took 5 more months for his depressive symptoms to abate and for him to return to his premorbid psychological baseline. During that time he lost the ability to walk and was wheelchair confined. Because of severe muscle atrophy he was unable to be rehabilitated and more than a decade later is confined to a nursing home. He remains a permanent reminder to me that the assessment of risk is not only the assessment of the risk of the treatment but must include an assessment of the risk of not treating.

The decision to administer ECT is similar to any medical procedure – the possible benefits must be weighed against the possible risks to the patient. However, unlike the risks of most surgical conditions where the risk of the identified illness is usually clear, the risk of the

depressive illness to the patient can vary widely from individual to individual and the assessment of risk must be done for each patient. In the dramatic case of John the risk was the rupture of an aneurysm, severe cognitive damage and possibly death versus the loss of his lower extremities. Fortunately not all patients must make such a devil's bargain.

### *The Procedure*

For the most part the preparation for ECT is the same as any surgical procedure. The patient is cleared medically. Medical clearance consists of a physical examination, a recent EKG and laboratory testing. It is standard practice to obtain a complete battery usually consisting of a CBC, electrolytes, BUN, creatinine, liver function tests, a TSH and Beta HCG when indicated. An EEG and MRI of the brain are only ordered when clinically indicated. The patient is instructed to not eat or drink anything after midnight prior to the treatment. This ensures an empty stomach at the time of treatment and reduces the risk of aspiration pneumonia. The time required to be NPO is actually eight hours and centers that treat into the early afternoon will instruct their patients accordingly. The patient should be also instructed not to chew gum because it increases gastric secretions and may also increase the risk for aspiration. Patients are also instructed not to smoke prior to the treatment because of its effect on the airway, blood pressure and pulse.

In our center we ask the patients to wear loose clothes to the treatment. Some treatment centers have the patient change a into hospital gown prior to the treatment. The patient is asked to void immediately before entering the treatment room. Voiding in addition to being NPO for eight hours prior to the treatment almost entirely eliminates incontinence during the treatment.

After entering the treatment room the patient removes his shoes and lies down in a hospital gurney. The patient is connected to an EKG monitor and a pulse oximeter. An intravenous line is placed for administering anesthetic agents. If the patient has a history of GERD he is pretreated with bicarbonate in order to reduce the potential for gastric reflux and aspiration. In our center the patient is then brought into a treatment area where attachment to the ECT apparatus occurs. The patient is attached to a one or two channel EEG. Electrode placement is determined. Actual placement of the electrodes is delayed until the patient is asleep. The patient is attached to an EKG monitor, a blood pressure monitor and a pulse oximeter in the treatment room.

The anesthesiologist premedicates the patient with glycopyrolate or atropine and blood pressure medication if indicated. Some practitioners regard the use of glycopyrolate or atropine as elective reserving their use for patients with heavy secretions or significant asystole and/or bradycardia during the treatment. Because of the multiple stimulations necessary to induce a seizure during the first treatment and consequently the multiple risks for asystole we always premedicate the patient with glycopyrolate for the first treatment. Under normal circumstances glycopyrolate does not cross the blood-brain barrier and it was once believed that there was less potential for increasing post-ECT anti-cholinergic induced confusion when using compared to atropine, which readily crosses the blood-brain barrier. However it has been demonstrated that ECT alters the permeability of the blood-brain barrier to large molecules allowing glycopyrolate to penetrate freely and consequently losing its advantage. The choice of agents is largely made by the preference of the anesthesiologist with some preferring glycopyrolate because of its superior effect on reducing salivary secretions and others preferring atropine for its increased ability to reduce asystole.

There are two side effects of these agents: prolonged dry mouth and urinary retention. In our treatment experience complaints of dry mouth are quite common and can endure for hours following the treatment. We have seen only two cases of urinary retention in 10,000 treatments. Both cases were elderly men with benign prostatic hypertrophy. Unfortunately, the elderly patients are at greatest risk for asystole and bradycardia and require pretreatment.

A blood pressure cuff is placed around the patient's right ankle but not inflated. The anesthesiologist then induces the patient most commonly using methohexital. Other common induction agents include etomidate, propofol, and ketamine. Once the patient is asleep the electrodes are placed and the ankle cuff is inflated to 100 points over the systolic pressure. Succinylcholine is then administered to induce paralysis. It is important for the patient to be fully asleep prior to administering succinylcholine to avoid the sensation of paralysis in a conscious patient. On rare occasion we have had a patient describe this experience with surprisingly accurate recall – surprisingly since it occurs just before the induction of a seizure.

After the succinylcholine is given there is a period of less than a minute before full paralysis is reached. Usually small fasciculations are visible and end at the point of paralysis. A bite block is then placed to reduce the risk of the patient biting his lip or tongue since the local effect of the electrical stimulation contracts the masseter muscles, which results in a clenching of the jaw.

A typical seizure duration is under 60 seconds. Infrequently emergence delirium occurs and the patient may become agitated after the treatment. This is treated with 1 or 2 mg of midazolam iv immediately postictally in subsequent treatments. Once the patient begins to breathe spontaneously he is taken to the recovery area. Complete recovery and readiness for discharge varies but usually ranges from 30 to 60 minutes.

### *Problems on the Day of Treatment*

It is not unusual for patients to feel disoriented and confused for a number of hours following a treatment. Many patients also complain of lethargy. Commonly patients will want to return home and lie down for a few hours. They should be encouraged to rest. Some patients also experience headaches. An over-the-counter analgesic such as aspirin or ibuprofen is usually sufficient to treat this. If patients complain of headaches following a treatment we will prophylactically treat them with iv ketorolac or fentanyl immediately prior to the next treatment. We have found that this dramatically reduces the incident of headache.

Following the index treatment many patients complain of diffuse muscle aches and pains. Typically the discomfort is most severe in the face, chest and shoulders. This pain is from the succinylcholine administered during treatment. When the succinylcholine wears off there is a rebound contraction of muscle fibers that is dysynchronous and can result in muscle pain. Many patients compare this to the soreness one feels on exercising for the first time in many months. Again the discomfort can be relieved with aspirin or ibuprofen. When this pain occurs some anesthesiologists will administer low dose mivacurium or a small initial dose of succinylcholine (sometimes referred to as a “self-taming” dose) in an effort to minimize the fasciculations and subsequent pain. This is controversial and many believe it to be ineffective. Fortunately in our experience the pain is transient and always worse after the first treatment. For the most part, patients appear to accommodate to this side effect and it is rarely an issue after the second or third treatment.

Some patients will experience nausea and vomiting following a treatment. This almost always occurs in the recovery area and is a reaction to the anesthetic agent or rarely the pain medication. 4 mg of ondansetron intravenously is almost always sufficient to relieve these

symptoms. If the symptoms are severe and recur with each treatment consideration should be given to changing the anesthetic agent to propofol – the one agent with an antiemetic effect.

On rare occasion, approximately one in one hundred in our experience, patients are incontinent during the treatment. For some patients even voiding minutes before the treatment does not seem to prevent the incontinence. These patients are asked to change into hospital pajamas and an adult diaper prior to the treatment.

A rare patient will develop prolonged postictal delirium and remain confused and profoundly disoriented for days following a treatment. The management of the delirium is similar to the management of any demented patient. They must be watched and kept safe. If the family is unable to provide supervision then the patient may need to be hospitalized until the delirium clears – usually 24 hours or less.

#### *Technical Aspects of ECT*

The practice of ECT requires little manual technique but much cognitive proficiency. The three areas that require choice and judgment are electrode placement, dose of electrical charge and the frequency of treatments. All three have both therapeutic and adverse effects. The choice of electrode placement affects treatment length, potential side effects and outcome sufficiently to warrant a pretreatment discussion of options with the patient and/or their family.

Bilateral electrode placement (BL) refers to placing the electrodes on the temporal fossa on each side of the head. This placement at seizure threshold is arguably the most clearly efficacious of the treatment choices. It is also the method that yields the fastest response. Unfortunately it is also associated with the highest degree of cognitive side effects. The most common alternative placement is right unilateral (RUL) which involves placing one electrode just to the right of the vertex of the head and the other on the right temporal fossa. This results in

the electrical current being concentrated away from the language areas on the left side of the brain providing a theoretical mechanism to explain the lessening of the memory impairment that is typically seen with bilateral ECT. The efficacy of RUL is dependent of the extent which the electrical stimulus exceeds a given patient's seizure threshold. If the energy dose is at seizure threshold RUL is not effective. Stimulation with settings 2 to 5 times the patient's seizure threshold is effective with stimulation at 6 to 12-fold seizure threshold having energy comparable to BL ECT. The cognitive advantages of RUL ECT decrease as the stimulation increases over seizure threshold. Overall, however, the cognitive advantages of RUL over BL are retained even at high stimulus settings.

Unlike RUL ECT BL appears to require an energy setting just high enough over the seizure threshold to induce a seizure. Further increases in stimulus intensity only seem to increase cognitive side effects. It has been suggested and incorporated into the practice guidelines issued by the American Psychiatric Association (2001) that a subset of particularly treatment resistant patients might require ultra-high dose BL ECT in order to respond. The evidence for this is thin and it is not a common practice.

It should not be surprising that RUL ECT requires more treatment sessions than BL ECT. The only accurate way to calculate seizure threshold is at the time of the first treatment. Because the seizure occurs at threshold the first RUL ECT treatment is essentially wasted. The second treatment is done at a setting many-fold above the threshold and is essentially the first effective treatment. Consequently RUL ECT reduces cognitive effects but increases the patient's exposure to anesthesia. Albeit this exposure is a very low risk compared to the incidence and risk of cognitive side effects there are often times when a fast response is necessary (a

hospitalized suicidal patient) or anesthesia risk is a concern (a cardiac patient) when BL ECT appears to be the treatment of choice.

A third electrode placement that is seeing increasing use is bifrontal (BF). In BF ECT the electrodes are placed on either side of the forehead 5 centimeters above the lateral canthus of the eye. In BF ECT the electrical current is concentrated away from mesial temporal lobe structures involved in memory formation. Preliminary reports indicate that this placement might preserve the efficacy of BL ECT while reducing the potential for cognitive side effects. More data is necessary to determine if this is accurate.

Another technical issue with ECT concerns the frequency of treatment. The convention in the United States is to do the acute treatments thrice weekly – usually Mondays, Wednesdays and Fridays. In England and Europe it is common to treat patients twice weekly. Both frequencies yield therapeutic results with equivalent benefit with the twice-weekly interval producing less cognitive impairment but a slower response.

The last technical issue is the relationship of the seizure to clinical response. Until relatively recently the practitioner's focus was on seizure duration. Some believed that the more seizure seconds the greater the likelihood of response. It was standard practice to require seizure duration of at least 25 seconds and to restimulate the patient if the duration fell short. We know from data with RUL ECT dosed at seizure threshold that even lengthy seizures yielded no significant efficacy. EEG studies have shown that a generalized seizure is necessary for a clinical response and that the involvement of deep brain structures is also more predictive of clinical response. In actual clinical practice what is observed is postictal suppression immediately following the seizure. This refers to a marked reduction in observable EEG activity following the seizure. The degree of postictal suppression is directly correlated with neuronal

involvement so that the greater the number of neurons involved in the seizure the greater the postictal suppression. As a consequence it has become standard practice to monitor at least one channel of seizure activity during a treatment and measure postictal suppression. Good postictal suppression with even short seizure duration of 10 to 15 seconds is usually judged as adequate. Because of this, methods to lengthen the seizure such as caffeine infusion prior to treatment are rarely used.

The goal of ECT is to induce a controlled well-generalized seizure followed by postictal suppression in an anesthetized patient. The factors that effect achieving this goal are the age of the patient, the energy limitations of the ECT apparatus, the number of previous treatments, electrode placement, oxygenation, medication and choice of induction agent. If a seizure does not occur all of these factors need to be evaluated.

An individual patient's seizure threshold varies widely and increases with age. The older the patient the higher the seizure threshold is likely to be and more energy required to induce a seizure. The amount of energy delivered in a stimulus is inversely proportional to resistance. The substance between the electrodes – skin, bone, air and the distance between the electrodes, affects resistance. Consequently bilateral lead placement requires the highest energy levels to induce a seizure because the electrodes are placed the furthest apart. ECT also raises the seizure threshold so more energy will be required to induce a seizure over the treatment course. Occasionally we have treated an elderly patient with bilateral ECT and have had to switch to unilateral by the end of the course of treatment in order to induce a seizure.

Another strategy would be to use caffeine pretreatment to lower the seizure threshold – usually 1 to 1.5 grams intravenously five minutes prior to the treatment. (An important caveat about the use of caffeine in the US relates to its labeling. The US manufacture includes the filler

material in the labeling dose for caffeine often leading to using half the ordered amount. It is important to inspect the label before administering caffeine to avoid this error.) Anesthetic agents also affect the threshold with methylhexital having little effect and propofol having a pronounced anti-seizure effect. As mentioned previously ketamine actually lowers the seizure threshold and can be a very useful induction agent in patients where inducing a seizure is difficult.

The choice of anesthetic agents is often influenced by the treatment experience. Methylhexital is the standard agent because of its short half-life and minimal effect on the seizure threshold. If patients experience nausea or vomiting they may be switched to propofol because of its anti-emetic effect. There is also a lower incidence of emergence delirium in patients on propofol so it may also be used when emergence is a problem. Propofol is problematic however in patients with short seizure durations since it has significant anticonvulsant properties and has been shown to profoundly shorten seizure durations. This shortening of seizure duration has not been shown to have an effect on the efficacy of the treatment. Ketamine is used in patients with short seizure durations because it lowers seizure threshold and lengthens seizure duration. Because it is an NMDA antagonist it may also have some utility in minimizing cognitive side effects. Etomidate may slightly lower seizure threshold and has the least cardiac effects and is useful in patients with heart disease.

Lower oxygenation levels also raise seizure threshold so it is important to monitor and maintain oxygen saturation at 100% prior to stimulation. This usually involves simply ventilating the patient well after the anesthetic agent is given prior to administering the treatment.

Concomitant medication can also affect seizure threshold. Many patients are being treated with anti-epileptic agents for mood stabilization. These obviously should be discontinued. Every effort should be made to keep patients benzodiazepine-free. Often low dose chlorpromazine can help with anxiety and has the added benefit of lowering the seizure threshold.

In summary the procedure of administering ECT requires no manual skill but significant cognitive knowledge. It requires thoughtfulness both about the treatment itself and in one's approach to the patient and the family.

### The Family's Experience

The family of the depressed patient, like the family of any individual suffering with a serious illness, can be the greatest source of support and solace and actively aid in the recovery process. Unfortunately, family members of the depressed individual struggle with a concept of illness that is unique to depression. Unlike illnesses that exist in the body in ways that give the appearance of being markedly discontinuous from the patient's emotional environment, such as rheumatoid arthritis or coronary artery disease, depression gives the impression of being related to if not caused by the patient's emotional milieu. Although it can be argued that rheumatoid arthritis or coronary artery disease are profoundly influenced by psychological factors they do not seem to exist on a continuum with these factors. The difference is both obvious and subtle. Joint swelling, a cardinal symptom of rheumatoid arthritis, is experienced as a clear physical

symptom of an illness. Depressed mood, a cardinal symptom of serious depression, is experienced as an emotional event with probable emotional causes. It is unnatural for us to think of depressed mood as a physical symptom.

Since the depressed individual does not give the impression of being physically ill and a cardinal symptom of their illness is their mood, there is an almost irresistible tendency on the part of their loved ones' to try and alleviate their mood. The family wants the depressed member to "snap out of it" or "cheer up" and will go to great lengths to achieve this end. Whereas the coronary patient's family won't let him mow the lawn or shovel snow, and the arthritic's family encourages them to avoid stressing their joints, the depressed patient's family will actively pressure them to change their mood.

Families will throw parties, rent movies, and plan vacations in what is usually a vain effort to get the patient to snap out of it. This effort, although entirely well meaning, often puts an inordinate burden on the patient to try and "act happy." Asking someone with depression to cheer up is the emotional equivalent of asking a pneumonia sufferer not to cough or a cardiac patient to run a mile. The mere cajoling is perceived as a rebuke and a criticism, not an effort to help. As a consequence of the burden of family pressure many patients actually feel relief at hospitalization because no one is encouraging them to control their mood and act cheerful. In fact, for many depression sufferers, hospitalization is the first real external recognition that they are ill. Many depression sufferers will date their recovery to their admission day to a psychiatric unit because it marks the first clear public acknowledgement that they suffer from an illness.

Another even more potentially devastating aspect of the family's experience is that of anger. At times it can be impossible not to be angry with the depressed patient. An individual with a depressed mood that is not environmentally congruent can give the impression of being

angry and critical of those around them. When active efforts are made to evoke a smile or a cheerful response and instead produce a blank stare the family member feels defeated and rejected. Often the family will complain that nothing they do seems good enough - that their best efforts produce no results and seem to have no effect. If the family has been uneducated about depression, particularly in first-time sufferers who sometimes go untreated for months, considerable effort needs to be made to help them become allies in the recovery process. Often the clinician starts with two strikes down because the family has already formulated its understanding of the illness and has assigned considerable blame and responsibility to the sufferer. It is crucial for the treating clinician to understand and respect the family's understanding of the illness, to educate the family about depression and enlist them as allies in the recovery process.

It is painful to live with an individual in a depressed state. It is a natural tendency to want to help a loved one feel better and the family needs help understanding that their intuitive inclination to place demands on the patient to change their mood is no more effective and as destructive as pressuring the pneumonia sufferer not to cough. The family may need to be actively assisted in this viewpoint because it is often so hard for them to view depression as an illness. They must be helped to understand that depression is an actual diagnosis with clear effective treatment options. While psychosocial stressors may have played a large role in the development of depression they may not be immediately relevant in the treatment and/or the recovery process.

Again an apt analogy may be coronary artery disease. Cardiologists are frequently faced with the families of acute MI patients in the CCU waiting room who regale them with a litany of Type-A behavior. "Doctor, he's the kind of guy who honks his horn when the light turns green.

He can't stand to wait in line at the movies. He does three tasks at once." While all these behaviors are risk factors for acute MI the cardiologist and family know that the first thing that needs to be done is to treat the physiologic aspects of acute myocardial infarction - not modify Type A behavior. In the same manner, the family of the depressed patient must be helped to understand that the depression needs to be treated first not longstanding issues related to one's personal history.

The way the family and the patient understand depression cannot only affect the recovery process but also impact on their lives for years to come. If there is a consensus that some external factor caused the depression there is an overwhelming tendency to eliminate the external factor. I have repeatedly seen families and patients assign etiologic significance to school or work and decide that quitting the situation would eliminate the depression. A significant part of helping the family and patient recover is educating them to **not** make significant life decisions until recovery is well underway.

### *Vincent R.*

Vincent R. is a 36 year old white married man with two daughters ages 2 and 4. He had no psychiatric history until his wife brought him to the emergency room because he had stopped eating the day before because of the growing belief that his food was poisoned. The patient had started a job as a ski instructor and manager of the ski shop at a nearby resort 6 weeks earlier. The job was a major career advance for the patient and he had been under some pressure to get the shop ready for the ski season. A month prior to admission, he began to sleep poorly, feel less energetic and weak during the day. His wife described him as "dragging his body up the stairs" when he came home from work. He looked "exhausted."

Initially Mrs. R greeted concerns about the level of her husband's depression with skepticism. She felt that his symptoms clearly developed after he started his new job, that he looked progressively more tired as the weeks went on and complained that customers were trying to harm him. When she learned that he had stopped eating because he believed that the hospital food was poisoned she reacted by blaming the poor quality of hospital food and began bringing him meals from home.

Much time and education was required for Mrs. R. to understand that her husband was suffering from an illness called "psychotic depression" that was largely independent of external circumstances. With the aid of counseling she began to understand her own worries about his illness and her fears about the future. She stopped urging Mr. R. to resign from his job - a move she hoped would relieve his symptoms - "If he didn't think he had to go back to work he'd feel better." She was able to then participate in Mr. R.'s decision to undergo a series of ECT treatments. Not surprisingly, because he suffered from a delusional depression, Mr. R. completely remitted after 6 treatments. He was able to return to work two weeks later.

Because the cardinal symptoms of depression often involve emotion states the family is posed with the problem of understand that emotional states can occur independent of internal or external environmental factors. We are accustomed to experiencing our feelings as reactions to thoughts, circumstances and often to those around us. We are happy because we are with our loved ones. When we are with our loved ones and feel sad it is easy to think that the sadness is because something they said or didn't say. The family of the depressed patient must understand that the depression is not a reaction to them but rather a state that arises because of a physiologic problem that requires treatment.

When depressed patients are considered for ECT both they and their families are often in a desperate state. They require more support than they might need earlier in the treatment course because ECT is often considered after a number of treatment failures. After failures of medication it is hard to be optimistic about ECT. The families, too, are subjected to negative images of ECT in our culture and may get negative reactions from other family members who do not approve of the patient's treatment. Fear can be a major obstacle to the appropriate use of ECT. It is the responsibility of the mental health practitioner to be sufficiently educated about ECT to allay their own fears and therefore be capable of allaying the fears of the patient and the family.

se and sine wave comparison. Energy = area under curve



