ENDOCRINE AND ELECTROLYTE CONSIDERATIONS RECOVERY FROM BRAIN DAMAGE

A THESIS

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INTRODUCTION

Determinations of intellectual and mental functioning represent one of the most significant health-care concerns. Psychiatrists divide organic mental syndrome (OMS) into acute and chronic forms. Designation of the acute form suggests reversibility of the syndrome, whereas the chronic OMS recover as their nutritional, metabolic and/or structural lesions improve. OMS is not of unitary etiology and may result from a wide variety of insults. Factors contributing to OMS include thyroid disease, hepatic or renal failure, drug toxicity, alcohol abuse and nutritional deficiencies of such entities as folate, thiamine and vitamin B_{12}. Neurological diagnosis and intervention techniques are becoming increasingly complicated by the fantastic accumulation of knowledge and methodology as applied to medicine. Too often diagnosis is literally taken to be categorization. Once classified the patient enters a realm constrained by the limits established by the pathological label. This approach does not expose the patient to a variety of systematic considerations interacting simultaneous within one intellect to deduce interventions conducive to potentiation of the quality of that patient's life.

The symptoms of a patient with hypothyroidism are some

of the easiest to diagnose and correctly identify (Hamburger, 1978; Larsen, 1980). Complaints of being tired, cold and lethargic usually indicate a metabolic dysfunction, especially when these symptoms persist. The strategic role of the hypothalamus in modulating the metabolic functions and more specifically the effects of the hypothalamus on the thyroid gland and its profound regulatory function on the metabolic rate must be evaluated. Effects of the hormone thyroxin stimulate the oxidation of fuel molecules in most of the cells of the body. The intracellular target of the hormone seems to be the mitochondrion. It is proposed that lack of thyroxin due to hypothalamus deficiency elicit the same behavioral effects as a patient exhibits after brain damage (Whybrow & Ferrell, 1974), and thus may be incorrectly diagnosed as OMS. Hyponatremia may elicit the same behavioral effects and as with hypothyroidism is easily diagnosed by clinical-chemical screens, has straightforward clinical patterns and is readily reversible with appropriate intervention (Tucci, 1981).

Psychological distress and depression can also elicit the same overt symptoms as an endocrine imbalance (Abramson, Seligman & Teasdale, 1976; Nelson & Barlow, 1981). Both the physiological and psychological symptoms must be

addressed to rule out depression as the major cause of the lethargic condition (Kilstrom & Nasby, 1981). Depression and hypothyroidism or hyponatremia, however, are not mutually exclusive. Any endocrine disturbance that elicits an abnormal, chronic fatigue state directly contributes to depression (Böll, 1978; Cobb, 1960).

Hypersomnia, lethargy and social isolation as secondary problems following an insult to the brain, are often not addressed because these clinical signs do not fit within the framework of the diagnosed pathology. Specifically, in a case of diagnosed spinal meningitis, integration of secondary complaints were not incorporated into the treatment regimen. These complaints included: (1) Reports of hypersomnia by the patient or his family (2) several neurological reports stating thalamic and hypothalamic damage secondary to the intial insult (3) lack of motivation by the patient in spite of repeated demonstration of his capabilities. In this case, consults only addressed the original brain damage and the secondary problems were not evaluated. Secondary complications can be the most debilitating factors and can prevent recovery of the patient.

PROBLEM STATEMENT

The purpose of this study is:

1. To identify the presenting problems of the meningitis which were addressed and treated by attending physicians,
2. To identify secondary areas which sustained damage due to the primary insult of meningitis,
3. To demonstrate that the hypersomnia and other abnormal behaviors of the patient resulted from a secondary dysfunction rather than from residual effects of meningitis.

LITERATURE REVIEW

A review of the literature in the area of endocrine imbalance produced two general areas of focus: hypothyroidism and hyponatremia.

In the absence of thyroxin, the basal metabolic rate may fall to 50% of its normal value (Griffiths, 1981). Tiredness and lethargy are due to defficient secretions of of pituitary hormone thyrotropin (Griffiths, 1981; McGilvery, 1979). Mental dysfunctions are observed in patients with hyper or hypothyroidism who have not sustained an insult to the brain (Whybrow & Ferrell, 1974). The mental dysfunctions fall essentially under two headings; impairment of cognitive functions and disturbance of affect. The hypothyroid groups appeared to be more grossly disturbed than the hyperthyroid group. Somatic concern, anxiety, emotional withdrawal, conceptual disorganization, depressive mood and blunted affect were noted. Motor retardation reached a significance of $p<0.05$. The hypothyroid group fell within the range of that considered by Reitan to be evidence of brain damage as reflected by objective testing. Lidz (1949) and Kleinschmit and Waxenberg (1956) noted that a profound depressive state frequently preceeded the clinical onset of thyrotoxicosis. Dahlstrom and Prange (1960) stated that

hypothyroid patients reported depression; and the MMPI profile were very similar to those of individuals diagnosed as psychotically depressed.

The intricate interrelationship between the function of the hypothalmus and pituitary and behavior must be addressed in order to determine whether the patient's initial brain injury caused the behavioral deficits or if a secondary hypothalamic deficit is responsible for the deficit.

The most frequent cause of injury to the brain is "stroke", or cerebrovascular accident (CVA) followed by open head or closed head traumatic injury. Another frequent cause of injury to the brain is infection by bacterial or viral organisms. Meningitis, or inflammation of the meninges of the brain, is the response of the brain to infection. Severe or prolonged meningitis can leave the individual with pronounced and long-lasting defects in cerebral function (Brookshire, 1973).

The major emphasis of the present research will be to study meningitis and the hypothalamic damage that was secondary to the original inflammation of the meninges of the brain.

Lipton (1974) presents an eloquent description of the role of the hypothalamus.

> It is by now well established that significant signals from the external or internal environment are transmitted neuronally to the central nervous system (CNS). Here they may elicit not only motor responses but also, in man, thoughts and feelings without motions (emotions). In addition, through appropriate neuronal projections downward, they elicit autonomic and neuroendocrine responses. The latter are mediated through the hypothalmus which functions as a transducer of the electrical energy of the neuronal signal to chemical signals in the form of polypeptides synthesized in the hypothalamus and transmitted via the hypothalamic hypophyseal portal vessels to the pituitary and perhaps elsewhere. The pituitary releases its hormones into the general circulation from which they are picked up by target endocrine organs, which are in turn activated to synthesize and release their hormones. These affect many cells throughout the body, but also feed back to the central nervous system, altering its threshold for response to stimuli and also regulating the capacity of the hypothalamus and pituitary to produce their hormones. There is thus an exquisitely controlled set of systems designed to modulate appropriate behavior and homeostasis.

Whybrow and Ferrell (1974) conducted a study to define more precisely the part played by the thyroid hormones in the maintenance of normal behavior; they attempted to correct such deficiencies in a prospective study.

All persons between the ages of 16 and 65 who were admitted to the study had a suspected thyroid disturbance. Those individuals with a history of previous disturbance unassociated with diagnosed thyroid illness, epilepsy, mental deficiency, arteriosclerosis or other cardiovascular diseases, plus individuals with fewer than eight years of formal education, were excluded from the study. A total of

17 persons were evaluated, ten with confirmed hyperthyroidism (mean protein-bound iodine, PBI, 14, 6 µg/100 cc) and seven with confirmed hypothyroidism (mean PBI 1.85 µg/100 cc).

After identification, a careful evaluation of the individual's mental status was undertaken using: A subjective test of mood (The Clyde Mood Scale), a clinical interview, and tests of cognitive functions (the Porteus Maze Test and the Trailmaking Test of Reitan). The patients also completed the MMPI and the Brief Psychiatric Rating Scale. The results of the investigation demonstrated a disturbance of mental function in both the hyper and hypothyroid groups.

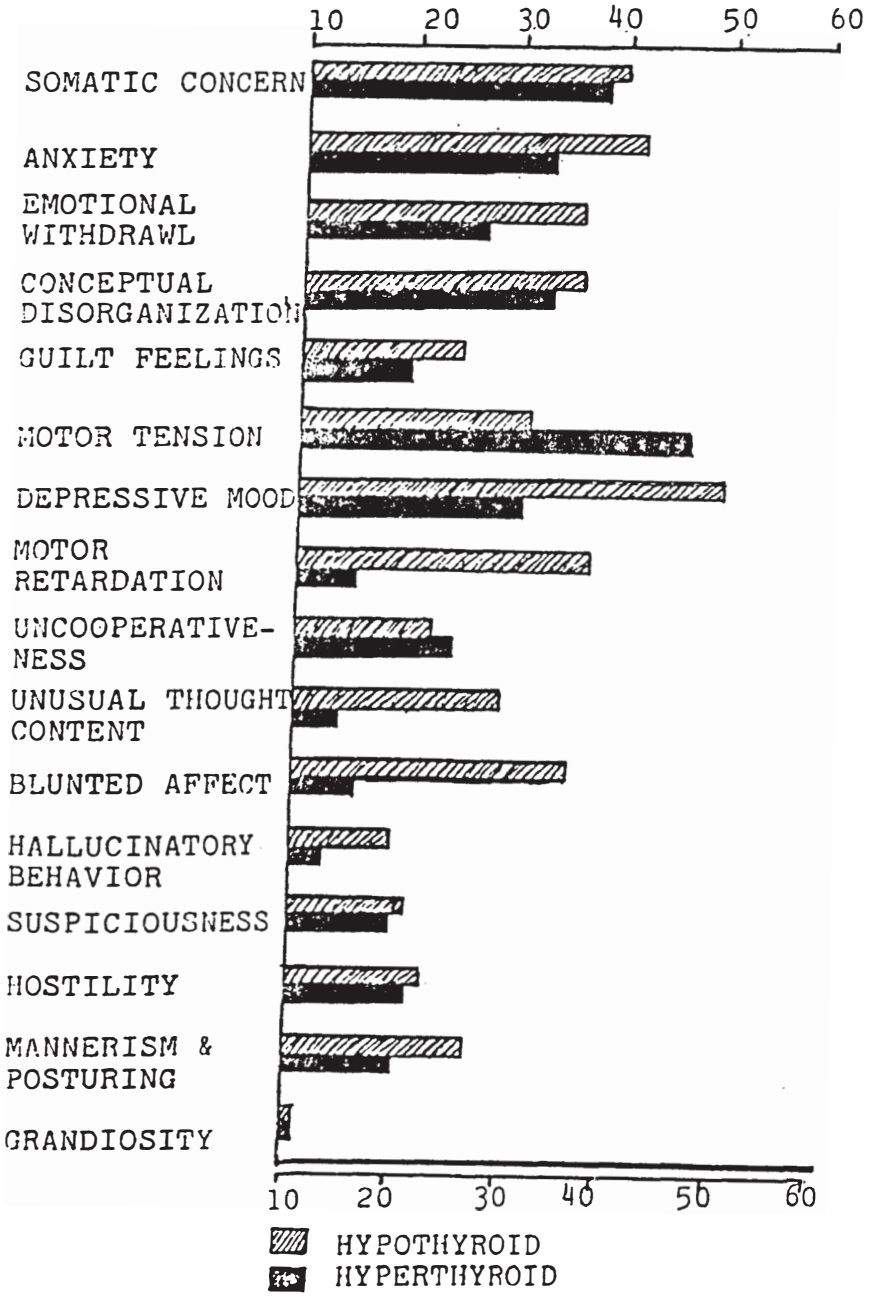
The mental dysfunction observed fell essentially under two headings. One was an impairment of cognitive function, the other a disturbance of affect. According to the researchers, in both instances the hypothyroid group seemed to be more grossly disturbed than the hyperthyroid group, but in both the impairment was considerable (See Figure 1).

In the hypothyroid group, the impairment of cognitive functions included a noticed deterioration of recent memory and difficulty in concentration. This profound disturbance was reflected in objective testing. The hypothyroid group fell within the range of that considered by Reitan to be evidence of brain damage.

The predominant disturbances of affect, in the hypothy-

Figure 1

Endocrine Dysfunctions-Thyroid Considerations Comparison of Hypothyroidism and Hyperthyroidism on the Brief Psychiatric Rating Scale



Mean Ratings of Brief Psychiatric Rating Scale

roid group, were those of marked depression, prefound melancholia, death wishes and thoughts of suicide. One patient was concerned that she was "losing her mind". An EEG, on this patient, showed bilateral slow waves consistent with a diffuse organic impairment. Another patient in the hypothyroid group showed signs of pseudobulbar emotional instability and cried uncontrollably although the discussions had no obvious depressive connotations.

The most intriguing findings of this study were the striking differences in the predominant mood of the two groups. No severe depression was noted in the hyperthyroid group inspite of a gross disturbance of body function in several of the individuals. This is consistent with the findings of Artunkal and Togrol (1964), who also used the MMPI in an evaluation of 20 thyrotoxic women prior to treatment and after treatment.

Lidz (1949) and Kleinschmidt and Waxenberg (1956) have noted that a profound depressive state frequently preceeds the clinical onset of thyrotoxicosis. This is consistent with the present study. In the hypothyroid group, all patients reported depression; and the MMPI profile of the group was very similar to those individuals diagnosed as psychotically depressed (Dahlstrom and Prange, 1960). Reports of depression, both of mood and physical status, were

the leading presenting symptoms in hypothyroidism. This suggests that the present study is not on isolated finding (Whybrow, et al., 1969).

The major impact of this study was all of the hypothyroid individuals had common complaints, euphoria, physical fatigue, mental confusion and depression. These are the same symptoms a patient or his family will report after brain damage. The emphasis on the previous study was dealing with individuals who had not sustained brain injury, however, the symptoms presented were the same.

The biological insights presented by Whybrow and Ferrell (1974), correlating several areas of metabolic disturbances that were common to thyroid disorders and affective illness, provide a basis for understanding a protective action of the thyroid hormone. T_4 index levels of circulating hormone correlated negatively with mood; that is the higher the levels of hormone, the less sadness in the hyperthyroid group. After euthyroidism had been established, the testing procedure was repeated and return to normal functioning was noted in most cases. The hypothyroid group, however, demonstrated some residual cognitive impairment.

Joel I. Hamburger (1978) describes hypothyroidism as a complex clinical syndrome resulting from a deficiency of thyroid hormone, and emphasizes that thyroid hormones

influence all organ systems with manifestations of hypothyroidism being multiple and diverse. The pathophysiologic mechanisms that directly impacts on this particular research is an inadequate mass of functioning thyroid tissue, resulting from inflammatory reactivity. Subacute thyroiditis (SAT) progresses from the acute phase, in the average patient, to complete recovery. Only a small proportion have permanent impairment of function. As the thyroiditis progresses from the acute phase to recovery, there is often a period during which there is impaired capacity for hormonal synthesis in conjunction with depleted hormone stores that resulted from the discharge of preformed hormones during the acute inflammatory phase. This period is normally brief and may easily pass unrecognized by the physician unless the patient happens to be re-evaluated at the appropriate time.

Rall (1972) described diverse etiologies of hypothyroidism to include: hypothyroidism secondary to failure of thyroid-stimulating hormone (TSH) secretion. Hamburger (1978) included the TSH and sub-categorized: A. Secondary (Pituitary) hypothyroidism; B. Tertiary (Hypothalamic) hypothyroidism. Rall (1972, p.302) stated, "it has recently been appreciated that hypothyroidism ranges from athyreosis with maxedema to fairly mild degrees of hypothyroidism.

Perhaps the most difficult phase of the diagnosis is to consider its possibility." Hypothyroidism secondary to failure of TSH secretion is elicited by any destructive lesion of the anterior pituitary. Usually these lesions suppress the secretion of other pituitary hormones and the hypothyroidism is only a part of the complete picture. Apparently only a few cases of isolated TSH deficiency have been reported. A deficiency of thyrotropin-releasing hormone (TRH) from the hypothalamus could elicit the same picture in the presence of a normal anterior pituitary.

When hypothyroidism is mild it may be overlooked quite easily and even relatively severe deficiency may be difficult to recognize (Hamburger, 1978). Examination of the following systems are suggested:

A. Cutaneous system- Facial puffiness and dry course skin.

B. Voice changes - Voice becomes increasingly husky.

C. Nervous system - The more the patient depends upon mental acuity in his daily activities, the more likely he is to complain of forgetfulness and loss of intellectual facility. As hypothyroidism is prolonged and becomes increasingly severe, a progression from somnolence to confusion, semistupor, and finally coma may be observed. The impairment of cerebral functions may result from cerebral edema as part of the consequences of inappropriate antidiuretic hormone secretions (emphasis added).

D. Muscular changes - Cramping is common.

E. Gastrointestinal system - Constipation.

Treatment of hypothyroidism is tenuous and during the course of treatment manic may be observed. A patient treated with thyroxine developed manic symptomatology following normalization of her thyroid status. The progressive appearance of the manic symptomology developed over a 7-day period. This observation is consistent with observations that peak psysiologic activity of thyroxine occurs 7-10 days after administration (Goodman, 1970). Manic symptoms were controlled by neuroleptics with symptoms abating within 2 weeks allowing for withdrawal of psychotropic medication. The research, however, did not indicate any follow up return visit for evaluation as to the patient's affective disorder and possible reoccurrence.

The hormones of the hypothalamus and the neurotransmitters as regulators of endocrine function are well documented (Guillemin & Burgus, 1972; Frohman, 1978). There is a delicate balance between neuropharmacological, neurotransmitter, and endocrinological function. Frohman (1978) found that many well-known pharmacologic agents, used in non-neurological diseases have their main effects on neurotransmitter secretion and have begun to be used in the treatment of endocrine disorders. Conversely many of the

pharmacologic agents used for other diseases can inhibit neurotransmitters, the precursor for the neurotransmitter or the enzyme that can be rate-limiting enzyme in neurotransmitter synthesis.

The drugs cited in the research can block neurotransmission at the pre or post synaptic receptor site. In the attempts to manipulate neurotransmitter levels in the CNS, the drugs could produce secondary changes that could overcome the effects they are intended to produce. An example such as monoamine oxidase inhibitor, which delays normal inactivation of norepinephrine, causes an increase in concentration. By this action, the drug alters the equilibrium of the system and secondarily causes a decrease in norepinephrine synthesis via the feedback loop system. Chronic administration of a receptor blocker may also result in an increase in the receptor number, leading to a tolerance to drug effects and a state of supersensitivity to the neurotransmitter upon drug withdrawal. The same alteration of equilibrium by drugs, i.e., amphetamines, elicits a stimulating effect to release newly synthesized dopamine and norepinephrine by nerve terminals. Depending on the particular drug's mode of action, the result is a net increase or decrease in the secretion of a releasing or inhibitory factor by the hypothalamic cell.

"Hypothyroidism is a common endocrine disorder, readily diagnosed and easily treated" (Larsen 1980, p.47). However, the subtle abnormalities that do not appear to be related to thyroid deficiency are often overlooked or attributed to another casual factors. In the case of a cerebral insult from external trauma (i.e., car accident) or internal trauma (i.e., disease state or cardiovascular accident) the subtle or often quite obvious endocrine disorder has been attributed to the primary insult to the brain. A patient's physiological and behavioral condition, following brain trauma, can mask the endocrine disorder. Larsen (1980) suggested the physician must exercise extreme care when diagnosing elderly persons; hypothyroidism may demonstrate the overt symptoms of senility or cerebrovascular disease and be masked.

Many of the clinical features of thyroid deficiency in its milder or more nonspecific form include: fatigue, weakness, cold intolerance, constipation, and dry skin and hair. These symptoms also are found in individuals who do not have a thyroid deficiency. Neurological symptoms include: memory loss, sommolence, cerebellar ataxia, paresthesias, hearing loss, and muscle cramps. Again, the symptoms in the older individual are not always an indication of a thyroid deficiency. Paradoxically the symptoms that are associated with the elderly individual and a patient after

a brain insult that are considered normal for their age or post traumatic condition can be the symptoms of an endocrine disorder.

One of the most ironic assumptions, by the lay person, is hypothyroidism is commonly thought to be associated with marked weight gain. However, the weight gain is usually limited to 5-10 lbs., which is fluid retained in the myxedematous connective tissue. Another paradox associated with the elderly individual or a patient after a brain insult was the slowing down of physical capacities. After compounding the other symptoms with arthroligias (joint stiffness), clinical evaluation of hypothyroidism in an older person, may never take place.

Larsen (1980) suggests the best clinical index of hypothyroidism is the degree to which the deep tendon reflexes are delayed in their relaxation phase; this applies to the patient without brain insult. This symptom alone is an indication for appropriate clinical biochemical testing. But, what if the individual has both the primary insult to the brain and secondary to the insult has hypothyroidism; is the secondary symptomology considered as the natural sequence of effects of the primary insult or diagnosed and treated as as seperate entity?

Disorders of fluid and electrolyte balance occur with

great frequency in clinical medicine. Abnormalities may have a profound effect on the cellular functions and the clinical manifestations are widely varied. Many patients with fluid and electrolyte disorders demonstrate neuropsychiatric signs and symptoms. These manifestations can vary from a subtle change in personality to overt psychiatric dysfunction. The symptoms can start with a generalized apathy and lethargic condition and progress in extreme cases to coma and death. Or start with lassitude and general weakness and progress to a quadriplegia and respiratory paralysis in extreme cases (Tucci, 1981). These clinical disorders are readily recognized by abnormalities in serum or plasma electrolyte values.

Hyponatremia, (serum sodium levels less than 136 meq/1), can occur as a result of sodium deficits and/or an excess of body water, and in some cases can be related to potassium losses. Disorders of sodium and water metabolism are among the most common clinical abnormalities in electrolyte balance. Normal handling of water and its excretion are dependent upon renal function and solute excretion and on the secretion of antidiuretic hormone (ADH), cortisol and thyroxine. ADH is secreted in response to osmotic and non-osmotic stimuli. Osmoreceptors located in the anterior hypothalamus are highly sensitive to small changes in

osmolality. ADH may also be released in response to other nonosmotic stimuli, such as pain, various emotional states and pharmacologic agents. Alcohol and water, on the other hand, can elicit ADH release. Thyroid hormones also play a critical role in water and sodium metabolism. Hypothyroidism and the hypometabolic state may be associated with depression in glomerular filtration and renal plasma flow, this leads to a diminished or delayed excretion of a water load, retnetion of sodium, and a dilutional hyponatremia.

Patients with "inappropriate ADH syndrome" or SIADH, present the clinical picture of water intoxication and hyponatremia (Mendelssohon & Rothschild, 1977; Tucci, 1981). Renal perfusion is increased and urinary sodium excretion is excessive (Leaf, et al., 1953; Jaenike & Waterhouse, 1961). Urinary concentrations and osmobality are increased in the face of plasma hypoosmolality. A wide variety of clinical disorders and drugs are associated with this syndrome. Pertinent to the present research, the one that impacts on the patient involved would be spinal meningitis.

Hyponatremia in patients with primary adreno-cortical insufficiencies is associated with volume contractions attributed to movements of sodium and water out of the extracellular space, redistributed to bone and cartilage, and imappropriate urinary and gastrointestinal losses of sodium as a result of diminished aldosterone secretions

(Fuisz, 1963; Tucci & Lauler, 1972; Berl & Schrier, 1978).

The mental status of patients (64) was evaluated with serum levels of Na+ of 128 meq/l or less and it was found that 39 patients exhibited neurological manifestations. Fourteen patients had acute hyponatremia of less than 12 hours duration with a mean level of 112 meq/l. and all were symptomatic. In this group of 14 patients all had depression of sensorium, four had grand mal seizures, and five were comatose. Half of these patients died. Arieff, et al., 1976). The neuropsychiatric symptoms appear to be related to brain cell swelling which results from movement of water from the extra to the intracellular compartment. The cases described by Arieff, et al., (1976) were the extreme examples of what low sodium (Na+) can do physiologically and to the mental status of patients. This research was included to emphasize the importance of the electrolyte balance to the functioning of the brain and body.

Gold and Robertson's (1981) research emphasizes the role of the hypothalamic peptide hormones, arginine vasopressin more commonly referred to as ADH and its effects on the central nervous system (CNS) functions. The role of ADH on the renal system has already been described. The implications of ADH and the study of disturbances in human behavior have not been as well documented.

The mutual interactions between ADH and CNS functions are thought to influence several specific behavioral and physiological processes. ADH has been established to have significant role in memory consolidation (de Weid, 1976, 1977). According to Gold and Robertson (1981) ADH's role in cognition is one of the most important factors in psychiatric disturbances or following brain damage.

ADH is synthesized in the hypothalamus. There the hormone's pathway is to posterior lobe of the pituitary where it is stored and subsequently released to modulate renal water excretion. A second ADH pathway leads to the third ventricle and into the cerebrospinal fluid (CSF). This second pathway has a major impact on physiological and behavioral processes. Zimmerman and Robinson (1976) postulated that the CSF pathway mediates ADH's role in facilitating memory consolidation in experimental animals. de Wied, (1976) demonstrated that injections of ADH antisera in the rat will induce an almost complete deficit of passive avoidance behavior, while administration of ADH will reverse experimentally induced amnesia of avoidance behavior. The third pathway described ADH secretion into the hypophyseal portal blood, which affects biological rhythms.

Raichel and Grubb (1978) conducted a study to support their hypothesis that, ADH, when centrally released, exerts

a specific regulatory effect on brain water content by modulating the permeability of the brain vascular bed, especially blood brain barrier capillaries. They administered ADH intrathecally and demonstrated significant increase in brain water permeability. This change was not demonstrated in cerebral blood flow. This research strongly suggested that ADH, either circulating in the CSF or transported axonally and terminating on brain capillaries, plays a specific role in regulating the brain's extracellular and intracellular fluid. This role is seperate from its role in regulating systematic fluid and electrolyte balance.

The tonicity of body water is ordinarily maintained within a relatively narrow range and boundaries are well defined by the setpoint of the thirst osmoreptor and by the threshold of ADH secretions. Between these limits body tonicity is regulated even more precisely by small adjustments in free-water excretion brought about by changes in ADH secretions relating to changes in extracellular and intracellular tonicity. Any pathological or pharmacological interference with the synthesis, storage, release, or action of any neurotransmitters or neuromodulators could result in alterations in normal setpoint for the threshold and/or sensitivity of ADH secretions. This altered threshold could be expected to have predictable consequences on plasma

osmolality and overall fluid and electrolyte homeostasis.

Recent experimental evidence indicates that ADH can influence several processes that are significant in the symptom complex of affective illness. These symptom complexes include alteration in memory, pain sensitivity, the synchronization of biological rhythms, the quality of rapid eye movement (REM) sleep, and the regulation of fluid and electrolyte balance (Gold, et al., 1979). ADH is functionally linked to monoamine neurotransmitter systems and is also altered by pharmacological agents which affect mood.

A systematic study of ADH functions in patients with affective illnesses (bipolar)showed lower levels of ADH in the cerebrospinal fluid of bipolar depressed patients compared to the control group. This was consistent with previous experiments (Gold, et al., 1978) that central ADH's function is augmented in mania and relatively diminished in depression. The drugs used in this research were lithium and carbamezapine. Carbamezapine augmented the effects of ADH; and lithium appeared to function as an ADH receptor antagonist. The speculative postulation was that this research represented a some what different concept of psychotropic drug action, in which drug efficacy is related to the stabilizing of the functional activity of a central peptide.

de Weid, et al., (1965, 1975) conducted studies

which show imaired memory functions in experimental animals with either hereditary deficiencies of ADH or artificial disruption of ADH functions. In either case, memory function was restored by the administration of ADH or its analogs. The link between central ADH functions and endogenously depressed patients that have long been described as having difficulty in retrieval of previously adaptive behaviors and in the retention of newly learned material (Stromgren, 1977). These memory deficits in depressed patients are consistent with the hypothesis of decreased ADH functions.

It has been demonstrated in both animals and humans that the administration of ADH modulates the noradrenergic systems in dorsal, septal, parafascicular, hippocampal, and lateral thalamic nuclei-regions which have been identified as important components involved in memory consolidation (Ramaekers, et al., 1977). ADH's sustained modulation of specific sets of target neurons which regulate a complex behavioral function such as memory consolidation is consistent with current theories of central peptide action (Barker, 1977).

Rapid eye movement (REM) sleep appears to be the stage of sleep which is particularly important to the consolidation of long-term memory. This is the stage of sleep which

is often disturbed in affective illness (Kupfer, 1977; Vogel, 1977). Drugs which facilitate memory consolidation elicit changes in REM sleep similar to those induced by ADH (Long and Loizzo, 1973).

Most of the research on ADH's central action has been based predominantly on animal data. However, clinical reports of the efficacy of ADH analog in the treatment of amnesia and memory loss secondary to senile dementia are promising (Oliveros, et al., 1978). Also, carbamezapine, a potentiator of the ADH receptor, has been shown to improve symptoms relating to alertness and mental functioning in a group of epileptic patients (Dodrill and Troupin, 1977).

Therapy to assure the recovery of all the patient's functions, after brain injury, has sorely neglected the intellectual and emotional aspects of the patient. The emphasis has been placed on physical therapy, speech therapy, occupational therapy.

When the patient is dismissed from the hospital, he or she has been helped physically, and often follow-up therapy is continued on an outpatient basis. This type of therapy and it's positive effects cannot be overemphasized as to the quality of help it provides. However, little effort or study has been done to improve the intellectual and emotional deficits that affect the brain.

Exceptions are the prolonged and extensive work of Teuber (1975) that presents an excellent picture of the natural history of recovery over a period of up to 30 years. There is very little emphasis placed on the relationship between physical difficulties and cognitive and emotional adjustment in daily living after brain damage. Bond (1975) suggests studies of this relationship, "need to be wildly promulgated since rehabilitation service err most frequently in concentrating heavily on physical disabilities with very little attention paid to intellectual and emotional handicaps." The attitude of most hospitals and physicians appears to be an erroneous assumption that physical handicaps may be improved by therapy, and that psychological handicaps may not.

Most patients leave the hospital able to talk, walk and are physically greatly improved, but unless the intellectual and emotional aspects of brain damage are addressed, the patient cannot function as he or she did prior to the injury. It is not uncommon to have the patient demonstrate fantastic physical improvement in the hospital and regress physically after returning to the home or work situation.

Physicians normally, are concerned with the physiological progress and when follow up trips are made to the office, the emphasis is on physical progress, not intellectual

progress. The standard answer to a question pertaining to mental or emotional problems, reported by the family or patient, "is that time will take care of the problem or that is just the way he or she will be due to the effects of the injury." Difficulties of a moderate or even severe nature are frequently seen in patients whose physical appearance, social conversation, and even IQ scores suggest normality. Such invisible deficits can be highly disruptive to the patient and family alike. The patients failure to adjust to the daily living pattern are often attributed to emotional and motivational problems because subtle cognitive deficits are not detected in the non-demanding environment of the hospital and early convalescent period.

Boll (1981) stresses the importance of neuropsychology in diagnosis. Head injuries represent the type of disorder ideally suited to benefit from a neuropsychological diagnosis and program for cognitive intervention. The importance of a diagnosis, prior to the patients attempt to return to normal activities, cannot be over emphasized.

Consider a patient with outward appearances of no cognitive or minimal deficit who returns to work or school too early and without benefit of a neuropsychological assessment of intellectual impairment. The cognitively demanding work in the abilities in which the patient is most deficit has

consequences that can compound the problem. The consequences include predictable failure with concurrent and increasing anxiety, decreased self-concept, and depression. According to Boll (1981) this conditions the patient's fear of reattempting tasks once recovery has taken place. This elicits family stress created by misunderstood behavioral alterations.

Fear of failure by the patient is the natural result of failure to educate the patient and family as to what deficits the patient has and how to handle behavioral problems when they arise. Once the damage to the brain is healed, the cognitive and emotional liabilities are more detrimental to the patient and family than the brain damage itself. The secondary gains, as a lateral effect of the symptoms or illness, are those which are felt to be beneficial to the patient. This can be a conscious or unconscous effect. According to Appelbaum (1977) in The Anatomy of Change, the attention a patient received due to an illness is self-perpetuating as long as the behavior is reinforced. In essence, as long as a patient can get by with doing nothing and the family or institution make no demands and lets the maladaptive and self-defeating behavior be rewarded, the patient has no reason to change. The type of brain damage referred to is that when the patient is not "self motivated" to change.

For every classic example of a highly motivated person who overcomes insurmountable physical handicaps to return to achieve impossible feats, there are thousands of patients who give up and are dependent on family or society, i.e., institutions all their life. These are the patients who desperately need neuropsychological help but so not know where or who to contact for assistance. The family may care but may not know cognitive retraining is possible and know less about how it works.

The problem that presents a delemma to many patients and families is terminology. What is cognition and what is cognitive-behavioral assessment and intervention? It is not a return to strictly introspective, subjective mentalism of self-reports as to what a patient is thinking (Mahoney, 1977; Meichenbaum, 1977). The perception of mental events is included with the process building on data gathered in as scientific manner as possible. Kendall and Hallon (1979) stress the goals as increasing the breadth and power of explanatory concepts and therapeutics without sacrificing methodological rigor. Cognition behavior is the study of mental events and the effects on behavior with emphasis on early assessment and intervention following brain damage regardless of the genesis of injury.

The logical approach is to prevent maladaptive behavior

and emotional repercussions. Once behavior/emotional patterns are formed, they are much harder to break and may be the key variables that inhibit rehabilitation.

Learned helplessness and the casual attributions patients make for uncontrollable events they experience influence the chronicity of the helplessness deficits as well as self-esteem. This controllability leads to chronic, generalized helplessness deficits. Bulman and Wortman (1977) found the more that victims of an accident (severe spinal cord injury) blamed themselves for the accident, the better they coped. The more they blamed another person or situation, the more poorly they coped with their deficit. The relationships between self-blame and superior coping is surprising. According to Wortman and Dintzer (1978), self-blame may be adaptive if there are concrete responses that people can make to alter their situation. These findings are consistent with other research by Heckhausen (1977) on assessing therapeutically relevant motivation. That is, the decision to become committed to a goal appears to be determined by the expectancy that the goal is attainable.

Maladaptive depression and learned helplessness are classic consequences of responses that are maintained by social attention (positive consequent stiluli). The persons providing that attention must be included in the cognitive,

behavioral intervention package. It has been demonstrated that by controlling extrinsic variables, treatment success is enhanced but it has not been demonstrated that controlling this variable is requisite in all cases (Haynes, 1979; O'Leary, 1972). Functional analysis, that is identification of all the variables of which a specific target response is a function, is crucial to successful intervention. Nelson and Barlow (1981) assumed that if the social attention that maintained the given depressed helpless response was not altered, then the problematic response could not change. Past learning history, attributional patterns, specific strengths and weaknesses, and all medical background must be assessed to design an individual regime for remedial therapy and rehabilitation.

Nelson and Barlow (1981) also suggested looking at personality disorders; that the patient may have had a life-long history of reinforcement for performing a particular "role" -- the martyr role or the sick role. Conversely there may have been roles that had been sublimated for years that, due to brain damage, could be played with an excellent excuse for assuming a different "role." It is possible the pre-morbid role may have been forced on the person and the resentment may not surface until after a traumatic brain injury.

In assessing consequent stimuli (environmental variables), both the positive consequences that are maintaining the problematic response and the negative consequences that make them problematic must be addressed. In terms of Mowrer's neurotic paradox (1950), abnormal behaviors may be both "self-perpetuating: (i.e., have positive consequences) and "self-defeating" (i.e., have negative consequences). By using the gradient of reinforcement, since the short-term positive consequences (daily experiences) may be more powerful than long-term negative consequences (future, unknowns and fears), a function of intervention should be to educate the patient and family to the long-term negative consequences.

A complete understanding by patient and family that after brain damage, forgetfulness, confusion, poor understanding, poor judgement and self-defeating and even hurtful behavior are due to the injury and the associated cognitive loss; this is a painful experience for all involved. However an honest evaluation as to the nature of altered behavior and emotional responses is normally received with relief by the patient and family. Boll, et al., (1974) feel this produces a willingness within the family to change the family environment and adjust to minimize stress to the patient. Stress has been shown to make the impaired person

behave even less adequately; this contributes more to the feeling of failure and many patients simply give up trying to do anything rather than face the stress that failure evokes. This in time reinforces the learned helplessness.

No evaluation of brain damage and the subsequent adjustment failures (without neuropsychological assessments and rehabilitation therapy) is complete unless one includes the classic work of Kurt Goldstein. Goldstein spent his long and productive life studying symptoms and behavior patterns not as isolated events but as reactions that are embedded in and are expressions of the total organism. According to Goldstein (1948), a symptom is not simply a manifestation of changes in a specific function or structure of the organism; it is also to be considered as a form of adjustment made by the sick or defective person.

Beginning with his observations on soldiers who had received head wounds during the First World War, Goldstein has conducted numerous investigations on the effects of injury to the brain. These studies have been brought together and summarized in his book After-effects of Brain Injuries in War (1942). This book is based on the observations of nearly two thousand patients, some of whom were seen more or less continuously for ten years, and it gives an account of the neurological and psychological symptoms of patients

suffering from brain damage, methods of testing psychological functions, and the treatment of this type of patient.

In Goldstein's book he stresses the patients as good hospital patients who readily adjust to routine activities; by devoting full attention to this type of task, the patients were able to avoid the unusual and unexpected situations. Goldstein found that this behavior was a symptom resulting from their deficit. In other words, their routine was an expression of the struggle of the changed personality to cope with the defect by avoiding situations that can no longer be mastered or adjusted to. Much of the behavior of brain-injured persons is compensatory in character and permits them to come to terms with the world in the best possible way under the given circumstances of their deficit.

Goldstein also emphasized a patient will make an effort to learn and expand his potential, in the environment, as much as possible if there is some incentive for doing so. Without a motivational desire to change, the patients prefered routine tasks.

> The careful analysis of the meaning of a symptom also requires an extensive study of all aspects of the person's functioning. It is not enough ordinarily to give a patient a battery of tests that yield a set of numerical scores of pluses and minuses. The examiner must be alert to qualitative features of the patients' performance as well and pay heed to even the slightest clue that may shed

> light upon behavior. He or she must not depend entirely upon diagnostic tests useful as these may be, for understanding the patient. The examiner must also observe patients in their daily life under more or less natural conditions, for the patient's success and failure in meeting the common problems of daily existance are the ultimate tests of their abilities. Goldstein favors the intensive study of a single case over a fairly long period of time.

Seligman (1975) noted striking parallels between learned helplessness, induced in laboratory experiments, and the phenomenon of reactive depression, so-called because the state is presumably brought on by some emotionally upsetting event such as loss of a job, death of a loved one, or failure in some valued activity. This study is consistent with the blunting of affect and depressed cognitive state following brain injury.

Depressed individuals are typically slowed down in their speech and body movements; they feel unable to act or to make decisions; they appear to have "given up" and suffer from what one writer (Beck, 1967) describes as paralysis of will. When patients are asked to perform some task, those who are depressed usually describe their performance as much worse than it actually was. According to Seligam (1975), "it is not a generalized pessimism, but pessimism specific to the effects on one's own skilled actions (p.122)."

The brain--behavior relationships have been the subject

of interest and inquiry since the first recorded Edwin Smith Surgical Payprus circa 2500 B.C. with descriptions of injuries to the brain and subsequent behavior effects on other parts of the body. However, it is only recently that systematic research and investigation of brain-behavior relationships have been utilized to provide a framework for assessment of neuropsychological disorders and procedures that can be utilized in rehabilitation therapy by the clinicians (Walsh, 1978).

Boll (1981) in his description of the "brain as the organ of the mind" suggested that "art and science of understanding human behavior has developed almost to the exclusion of consideration of the brain as a relevent factor (p.47)." The discussions, among psychologists, without references to the brain's organization, Boll stated, "underscores our extreme tardiness in establishing what is a recent and rapidly spreading requirement for study in this area as part of professional preparation (Golden & Kuperman), 1980)." This emphasis, on behavior, by psychologists to the exclusion of the understanding of the functions of the brain and conversely the emphasis on the brain by the neurosurgeons with scant attention devoted to the behavior elicited by the brain, appears to be an era which must end if patients are to derive full benefit of both fields.

Boll (1981) indicated that in the past that the normal brain was of little interest to the clinician. "When damaged, however, it appears to spring magically to life, demanding attention or at least identification (Boll, 1981, p.47)." The psychologists label, the physicians label, but the implications of this damage and the impinging on people's lives has in the past been tossed back and forth between the professionals. Neither the psychologists nor the physiologists placed the effects of damage to this organ in its' proper context. The human brain damaged or normal has finally earned attention with the advent of the highly integrated science of neuropsychology that covers both neurological or physiological aspects of behavior as well as psychological. The cognitive approach to therapy after brain damage promises a rapprochement between the clinic and the laboratory. There appears to be an irresistible trend towards a cognitive-behavioral hybrid that holds that maladaptive behaviors are elicited by maladaptive cognitions, and that behavioral change can be produced by means of a cognitive change (Kihlstrom & Nasby, 1981). Just as there is no neuropathological unity across brain damage, so there is no unity in its behavioral effects. Lashley stated: "It should be a fundamental principle of neural interpretation of psychological functions that the nervous activities are

as complex as psychological activities which they constitute (Cobb, 1960, p. xx)." The psychologists's corollary to this dictum suggests, "our methods for assessing behavioral change should be as complex as the brain which subserves the behaviors and behavioral changes (Boll, 1978, p.602)."

A single case study will be presented to emphasize the psychological and emotional deficits of a brain damaged patient (W.M.) who did not receive early neuropsychological diagnosis and rehabilitation. The conditioned helplessness and fear of failure had been reinforced by the patient's family and friends due to lack of understanding of the detrimental effects of treating him like a child. The patient's wife was the only one who did not contribute to his learned helplessness.

The major problem at the start of the intervention was lack of motivation. Three years had elapsed since the original brain damage due to spinal meningitis; the patient was not therapy ready due to conditioned maladaptive responses.

One concept that must be used with W.M. and all older patients is the levels and hierarchical laws of cognitive processing and learning by comparing the child and the adult. Examples were used from Luria's, The Working Brain (1973) to explain how much less critical the primary input cortical areas are to the adult in learning or relearning after brain

damage has a fantastic impact on the older patient once it has been completely explained and understood. What this concept holds for the adult will be the security that they do have something to fall back on and more advantages to start retraining with because they had more prior to the injury that they had already learned and had become part of their repertoire. This should help counterbalance the feeling of being too old to learn and the controversial subject of neuronal plasticity of the brain with the child's brain considered more plastic compared to an adult brain.

Stressing that each action requires the combined workings of many units in the brain should help dispel the notion that even if task-specific functions are impaired, this does not preclude all sub-cortical units of the functional system are impaired.

The condition of this patient should elucidate that an earlier neuropsychological assessment and rehabilitation program would have prevented the emotional and cognitive problems that have prevented the patient from trying to help himself. The behavioral "mind set" and fear of failure could have been circumvented if this area had been addressed at the time W.M. was in the hospital. Concurrent with physical therapy, speech therapy and occupational therapy, cognitive retraining should have been started. As indicated

in Appendix A, all therapy is recommended while the patient is still in the hospital as soon as the physician releases the patient for rehabilitation. At no time during the past three years was a recommendation made by any of the physicians to have a neuropsychological assessment and testing evaluations made. The reasons W.M. was evaluated by Dr. M. was at this researcher's insistance. The evaluation was done but no specific treatment modality was instigated and no progress was made. Trips to Dr. M. were discontinued after six months. His final diagnosis was fear of failure, which had been observed from the beginning, compounded by the progressive learned helplessness. The recommended program, by Dr. M., was a structured day. W.M. had no understanding of what was meant by a structured day at home.

He is now aware of what structure in the home environment means. It involves having a reason to get out of bed and an on-going project to start reinforcing a feeling of self-worth and accomplishments. He must be motivated prior to any attempt to do structured rehabilitation.

On an informal basis, pre-testing has been done in the home to point out what tasks he can accomplish with no effort. Some areas of the brain that had been previously diagnosed as damaged now appear to be functioning adequately. The right hemisphere requisite for certain math skills

appears intact. He is able to dance without loss of balance or rhythm. He plays bridge (more slowly than prior to the insult) which indicates the prefrontal lobe used in formation of plans and programs of actions has not been affected to any degree. No receptive or expressive aphasia can be detected.

As indicated in Appendix A, the parietal lobes sustained severe injury, but at this time a sensory evaluation of W.M. shows no loss of afferent recognition by all modalities using the usual testing for lack of perception of sensation. This check, on an informal basis, included both sides of the body. The visual problem still persists but is not as bad as W.M. claims. The bilateral damage to the third cranial nerve will be further assessed to determined the best compensatory method to employ during therapy.

The left parietal lobe damage appears secondary to the left frontal involvement that inhibits fine motor control in the execution of specific tasks. The gross motor control shows only minimal loss.

Diller (unpublished) indicated the need to develop ingenious and unconventional approaches to assist in the treatment and long range management of patients after brain damage. Whatever works for a specific individual in a remediation process has value; this may be task and patient

specific but should be noted for future use. The single case study has the advantage of demonstrating the application of many different techniques to help the wide constellation of individual differences each patient brings to therapy. The single case study also permits the assessment of skill in "real life" settings rather than in the controlled clinical or hospital environment. The goals, which are determined by the patient, can be more readily qualified and the progress can be evaluated. This type of remediation is more amendable to change as indicated by task specific progress, the patients mood, the fatigue factor on a particular day and all the extrinsic variables that may mask or confound progress in group treatment. Group therapy may be indicated after the initial problem of lack of self-esteem has been resolved.

Diller (unpublished: 1981) emphasized some critical issues to be addressed for the decade ahead. Emotional problems was one of the critical issues and involved the impact on both patient and family; this has been described previously as the major problem in this case.

Becher (1980) stated: very little is known about the way disabled people spend their time. This also is an ignored area as presented earlier. Emphasis must be placed on how little help W.M. received after six months of out-

patient consultation on a monthly basis with Dr. M. The structured day had absolutely no meaning or impact; consequently, W.M. simply agreed to everything suggested and did absolutely nothing when at home. These disturbing facts cannot be ignored by anyone interested in intervention and rehabilitation.

The correlation between family involvement and ultimate disposition of patient is evidence for the importance of the interpersonal environment in predicting the outcome of the patient's return to the social mainstream. The interpersonal environment probably overlaps the intrapersonal environment, in this case. The "no correlation" statistic between family involvement and functional recovery may well be the result of the lack of family involvement at the rehabilitative level in the institutional setting -- a situation that many researchers deem it necessary to change (Belcher, et al., 1978; Lehmann, et al., 1975; Brundy, et al., 1979). Psychological tests and educational level both have impact on the rehabilitative outcome, but they will not be considered in this paper because psychological tests are the one predictor that correlates positively with both conditions and because the premorbid educational level cannot be changed. The lack of correlation between financial resources as a predictor and other conditions is surprising and

is not consistent with the results of other studies (Belcher, et al., 1978; Brundy, et al., 1979).

In discussing environments, it is important to bear in mind that whether or not environment is hostile rests to a great extent on how the individual perceives it and what his previous experiences have been. The most critical environment may well be the hospital/rehabilitation facility in terms of how much dependency on other individuals its rehabilitation program involves. An atmosphere in which a patient receives the very best personal care and attention can seem hostile when the patient has probably cared for himself, and others as well, for the better part of his lifetime. Optimal involvement of the patient in his own recovery, therefore, may be critical to the final disposition of that patient.

Belcher, et al., (1978) cite two studies conducted on stroke patients. In the first study, two matched groups were used. One group received full rehabilitation training for self help activities (dressing, feeding, etc.) while the other received special nursing care and attention. At discharge, no differences between the groups were found; however, comparison of a program that emphasized early mobilization and stroke prevention instruction with one using common rehabilitation procedures revealed that 83

percent more of the first group were discharged as totally independent in self care. This may indicate that the speed with which the patient can exert an independent impact on his own recovery affects the outcome.

Biofeedback is another recent technique that allows the patient to participate in his own recovery at an early stage (Golden, 1978). During this process electrodes are placed on the disturbed muscle and impulses are transmitted to an electromyograph which provides a sensitive measure of any electrical activity (contraction, relaxation). Feedback is immediately accessible to the patient through an audio or visual signal. This technique allows the patient to work almost exclusively with the machine only, and encourages the patient to devise strategies by which he can control his muscle activity. The patient manipulates the machine (or perhaps, the machine manipulates the patient), and the role of the therapist becomes secondary.

Brundy (1979) conducted a study in which 70 hemiparetics monitored their muscle activity through biofeedback techniques. Results showed a positive correlation between the use of biofeedback and the regaining of purposeful movements regardless of the severity of the patients condition. Other factors that were not significant relative to improvement were medical history, etiology, hemianopsia,

and expressive aphasia. Researchers conclude that the future for the hemiparetic should be one where he helps himself through the use of biofeedback techniques. The future role of the physician and other therapists, they feel, should be one of guiding, advising, and integrating family members into the rehabilitation process. In such a situation the patient cannot help but feel that he is very much more the master of his own recovery than he would be in a traditional rehabilitative setting.

Marmo (1974), in the Journal of Occupational Therapy, suggests that retraining of self care skills be postponed until the patient shows significant improvement in sensory, perceptive and cognitive skills. Then, just as the child learns to feed and dress himself when the relevent processes mature, so can the recovering adult begin to perform these tasks when the relevent skills have sufficiently improved. This would eliminate the therapist mediated "trial and error" re-acquisition of skills that were fundamental to the patient in his premorbid state.

Thus, rehabilitative intervention and the manner of instituting such a program can affect outcome. Further, a possible predictor of ultimate outcome may be the degree of dependency developed during the rehabilitative process.

HYPOTHESIS

It is proposed that the primary injury to the brain-spinal meningitis was emphasized to the exclusion of the secondary thalamic-hypothalamic damage. The secondary problem will be accepted as the presenting problem and the one that elicited the more severe, long lasting and debilitating effects.

METHODS AND PROCEDURES

A single case study will be presented. Male age fifty-six years at time of insult, race-caucasian, right-handed.

All medical records will be examined from September, 1978 - January, 1982. A complete electrolyte profile will be included, and a complete neuropsychological evaluation and tests will be administered. The significance of the results will be evaluated and recommendations for rehabilitation and further research will be determined.

History of Patient:

This diagnosis was prior to the final diagnosis of bacterial meningitis in 1978:

(1) Patient moves his right side better than he moves the left side, and does respond to painful stimuli

(2) Semicomatose

(3) Urinalysis was normal

(4) Electrolytes showed sodium of 131, potassium was 3.3, chloride of 84 and bicarb of 32.

(5) CAT scan negative

On 9-30-78, Mr. M. had been in Intensive Care Unit for a period of almost two weeks. Surgery was performed to correct a duodenal ulcer -- not related to the present illness. The examination showed that the patient appeared to

understand directions and commands, but was deficient in expressive speech.

During the patients three months in the hospital in 1978 (September-December), the discharge report included the following results:

(1) Marked ptosis and bilateral third nerve palsy in the left hemisphere

(2) Neurological complications of hemiparesis

(3) Essentially normal arteriograms in the carotid and vertebral distribution.

(4) C A T scan was unremarkable except for some mild atrophy

(5) SMA 12 stayed fairly normal throughout the hospital stay

(6) Blood gases, electrolytes remained fairly normal throughout the hospital stay

(7) Hemoglobins were normal except during the time of bleeding and this was corrected by blood transfusion

(8) His second spinal tap showed 238 white cells, 52% lymphs, 42% segs, 90% protein, positive gamma globulin and 60% sugar. The initial fluid revealed 8400 white cells, 85% segs, and 234 protein, 5% lymphocytes and 40% sugar.

Impressions after recovery from surgery in September, 1978 and recovery from acute stage of meningitis:

(1) The patient was awake and alert, but quite lethargic and listless

(2) Responses are slow

(3) Orientation intact

(4) Oriented as to time, place and person, however, age, and current date were not known

(5) Examination of the extremities shows that this patient is currently ambulatory with some minimal standby assistance. Deep tendon reflexes are 1/4+ and equal bilaterally. Extensors of the foot are intact as well as flexors and he is able to do dorsiflex and plantar reflex with excellent strength and coordination

Physical therapy and Occupational therapy were instituted as well as remediation in activities of daily living, perceptual processing and physical endurance.

An E.E.G. was performed 9-17-78. Diagnosis: Dysrhythmia Grade 11. Impression: Abnormal E.E.G., indicating left cerebral dysfunction primarily in the parietal temporal area in the presence of some bilateral cerebral dysfunction.

Cranial computerized automated tomography revealed:

1. Clouding of the left frontal and left maxillary

sinuses, consistent with inflammation. Questionable cysts of polyps in the left frontal sinus.

2. Questionable bony deformity overlying the frontal sinuses, which may be correlate with history of trauma.

Cranial computerized automated tomography on May 9, 1979 was normal with no evidence of atrophy or structural changes.

December 28, 1978, Mr. M. was evaluated in a neurology clinic by one of the attending physicians envolved in this case, the opinion at that time was:

(1) Gradual and dramatic improvement post insult.

(2) Intermittent double vision, some blurring of vision, some unsteadiness in his balance.

(3) Personality change -- belligerent, emotionally unstable.

(4) Lack of psychomotor coordination.

(5) Oriented to time, place and person (Ox3).

(6) General knowledge and information is good.

(7) Evidence of ptosis in the left eye with some weakness of adduction of the left eye and elevation-some elevation paresis of the right eye.

(8) Pupils are equal and reactive and fundi look normal.

(9) Neck is supple.

(10) Bilateral hyperactive reflexes with bilateral mild quadriparesis (left greater than right) and increased generalized reflexes.

(11) Minimal features of pseudobulbar palsy. Inderal-60 milligrams was prescribed for hyperactivity and emotional instability.

January 25, 1980, Mr. M. was reevaluated in a neurological clinic -- the opinion at that time was:

(1) slow mental functions.

(2) Significant thalamic and hypothalamic cerebral dysfunction secondary to meningitis.

(3) Memory problems which appeared to be in remission, but were inconsistent in nature.

(4) Severe ataxia.

(5) Hypersomnia for the last three or four months.

At this time he was switched from Tagament because it may be having some effect on his mental status and the observation was made that, "I have seen a number of patients who have lethergene confusion from Tagament." This was to be discontinued for a short time and another antiacid (not specified) used. Inderal 60 mgs. a day was continued, and Ritalin 5 milligrams twice daily was prescribed to maintain reticular activating system activity.

February 4, 1980, Mr. M. was under the care of Dr. -- for approximately a year. The report included:

(1) Mild bilateral third nerve palsy

(2) Emotional incontinence, crying out, involuntary control of his emotions

(3) Difficulty with his balance

(4) Excessive sleepiness -- helped somewhat by Ritalin but has not helped his personality problems

(5) Patient is totally disabled

Recommendation: Disability compensation (see Appendix A for complete evaluation 2-4-80).

"He is totally disabled. He is unable to manage and take care of his own affairs. He needs almost constant supervision by his wife. If she were not there to take care of him he would sleep a good part of the day and would probably not eat."

February 18, 1980, Mr. M. was evaluated by a psychiatrist. The evaluation procedures included: Clinical Interview, Wechsler Adult Intelligence Scale, Wechsler Memory Scale, Wide Range Achievement Test, Word Naming Tests, Finger Tapping Tests, Television Name Writing Test, Trailmaking Tests A & B, Sensory-Perceptual Examination, MMPI, Test of Attention and Interpersonal Style, Cornell Medical Index, and SCL-90R. The results are in Appendix A.

After the initial visit to Dr. M., the psychiatrist, the research for this paper was instigated. Included is the 1980 evaluation by Dr. M. and observations by self and the work that impacts on this study.

Onset of spinal meningitis and encephalitis hit W.M. at a most vulnerable time, due to excessive stress about his youngest son who had cancer. During the time W.M. was hospitalized for spinal meningitis, encephalitis, and in a coma, his son died. When W.M. returned to reality enough, to be told of this tragic event, he regressed and for many months could not accept the trauma. W.M.'s depression, over the death of his son, did not diminish with time and was compounded by pseudobulbar neurological involvement evidenced by periods of uncontrolled crying. His disease left him with fine motor nerve damage and double vision due to both the right and left hemisphere of the brain sustaining damage as well as the spinal cord.

The most prevailing symptoms, after his return home, were apathy, depression and lack of motivation. The attending physician said that time would produce internal healing to a certain degree. Prior to the visit to Dr. __, not one of the many physicians suggested a psychological evaluation. The entire emphasis had been on the physical aspect of W.M.'s illness. After the last visit to the hospital, a

C.A.T. scan showed no change in the brain. Mrs. M. was informed this was the way W.M. would always be and no suggestion of a program for mental rehabilitation was made.

Behavioral inconsistencies include defects in episodic memory or disorientation as to events occurring in place and time. At various times W.M. relates a story about a visit to San Antonio which had occured many years ago but related as though he had just returned from the trip. This event occurs only in stressful situations as in office visits to physicians.

W.M. spent two weeks in ___ Hospital for psychological testing and evaluation. The M.M.P.I. form R, Test of Attentional and Interpersonal Styles, S.C.L. -90-R and Cornell Medical Index were administered verbally by myself due to W.M.'s inability to read small print and motor control dysfunction which caused him to start filling in the blanks and keep on writing until the entire test was covered with squiggle marks. There was another problem in W.M.'s lack of motivation to finish the requirement task. The tests were administered in my home when W.M. was out for a week-end pass from the hospital.

W.M. showed marked improvement in his attention span, and he worked with the test in my home for a period of three hours with no loss of ability to comprehend the question

asked. It was also observed, by myself, he had a recall on previous questions which had been asked thirty minutes to one day before; even though the question was repeated with a slightly different phraseology.

When W.M. returned to the hospital after the week-end, occupational therapy and physical therapy were given in the hospital. W.M. was able to start and complete the task of making a ceramic flower container. He showed definite interest in his feeling of accomplishment which was the first and only positive response exhibited in a year or longer.

Dr. __ did not suggest a particular treatment for behavior modification. This area was to be designed utilizing whatever skills W.M. had enjoyed in the past and working toward retraining eye-hand co-ordination. At the present, I plan on working with W.M. on his ceramics as he resents his wife asking him to do anything. As she is the caretaker figure, the resentment appears normal as W.M. is quite dependent on her.

In office evaluation by Dr. __, after W.M.'s two weeks in the hospital:

1. Fine sense of humor.
2. No medication indicated - (Dr. __ took W.M. off of Inderal and Riddlin during hospital admission and questioned the use of Inderal by previous physician).

3. Pain experienced, by loss of son Mike, is organic in nature.
4. I.Q. testing rated W.M. 101 at present.
5. M.M.P.I. picked up on depression.
6. Guilt feelings about Mike's death must be talked out.
7. Structured routine at home must be started.
8. Reading comprehension, W.M. can't do well, at present, due to brain damage; his reading level is 4th grade.
9. Math scoring is low.
10. Recent memory is impaired, will be forgetful.
11. Depression is the primary problem; will re-test for intensity in six months or a year.
12. Memory will fluctuate - this is organic.
13. Eyes, suspect the damage is permanent.
14. Main impairment is recent due to no input

Recommended Modality:

1. Structure - The Key. Seven days a week.
2. Relay messages to other son Buddy who worked for W.M., that W.M. will continue to cry out but will de-intensify.
3. Three days a week at work suggested for more input.

4. Office visits are scheduled each two weeks then once a month, at a later date, when W.M. shows improvement.
5. Just ask or tell W.M. what to do and that is it; do not let him get by with child-like behavior.

Two weeks after W.M. was dismissed from the hospital, Dr. __ recommended a highly structured schedule for W.M. Dr. __ indicated the reason W.M. regressed back in time, with the story about the trip to San Antonio, was to fill in the gaps due to no input since his illness. It is imperative that he have a reason for getting out of bed. This will be the major thrust surrounding my participation with this case.

The first approach will be repeated explanations, to W.M., of his illness, the exact nature of why he is depressed, feels helpless and the entire background of his feelings and actions. This is designed to make him fully aware of where he was, why and the recommended help to overcome the depression. Each talk session, when feasible, will be followed with a working session on the project of W.M.'s choise.

Session #1

Our discussion continued until our project was fully

understood by W.M. We poured two ceramic pots. The only assistance required, by W.M., was telling him when the mould was full and lifting the mould to pour the excess slip out. Mrs. M. and I did fine trimming of wet clay.

Session #2

Our discussion was the same as the first one but with more emphasis on the difference between normal grief and neurotic or psychotic grief. W.M.'s responses about the day before were jokes about psychotic states, and he tries to emulate psychotic behavior. W.M. was unable to stimulate hallucinations when he tried to as a joke. He also could not maintain delusions in a joking manner for any length of time. He tried to manifest psychotic symptoms mentioned the evening before and was unable to recall almost all symptoms and characteristics of each psychotic category.

Comment: I am using comparisons between normal, neurotic and psychotic states to convince W.M. what Dr. __ has told him is true.

Session #3

I stressed, to W.M., the need for him at the shop by his son, Buddy; not for physical help but for moral support and guidance by his father.

Week #3

A self evaluation, by W.M., after he was unable to fit

into the list of D.S.M. categories was quite positive. Question: How does that make you feel? Answer: Normal! W.M.'s signature is almost back to normal at this time.

Week #4

An observation by Mrs. M.:

When W.M. is very angry, he can do things the same way he did before his illness i.e., he can swing his coat around his shoulders and slip his arms into the sleeves without any difficulty. He then walked home from his parents house, which is three long blocks away, without shuffling his feet or stumbling. The trip took five minutes and W.M. did this in 30^{o} weather.

The tile was placed in the fountain by Mrs. M. and I. W.M. spread the tile adhesive on for us and handed the tiles to us when he saw we were ready. W.M. worked, with encouragement, for several hours but when he passed a point of exhaustion both physical and mental this task proved counterproductive. The following day he was regressed and too tired to care about the project.

W.M.'s self esteem is still quite low. This symptom often overrides the depression. His low self esteem, in certain areas, existed prior to the illness.

Problem: W.M.'s parents, sisters and brothers do not believe in a psychological approach to help W.M. They

still persist in treating him like a baby and appear to be reinforcing his low self worth. They would wait on him hand and foot if Mrs. M. did not intervene. The family, normally, get together at least once a week at his parent's home. His sister worked at the shop and this does not help W.M. to follow through with Dr. __ suggested program.

Week #5

The tile was finished by Mrs. M. and my husband due to W.M.'s falling and hurting his side. W.M. had no interest in the project prior to his fall.

Week #6

W.M. was on muscle relaxers, due to the fall, and had no attention span on any subjects. No progress was made this week.

Week #7

I will try a new approach; W.M. enjoys math but scored low on the battery of tests in this area so I will try teaching Algebra to him as a mental stimuli. He should be able to work the problems without assistance. I plan to have him do homework assignments. Then we can go over them together which hopefully will add more mental activity.

Week #8

W.M.'s eyes have improved from 20-85 to 20-25 with glasses. Dr. __ said W.M.'s depression was still quite

severe which is the reason he still won't get up and voluntarily do anything unless someone works with him. If the depression has not improved by the next visit, Dr. __ will prescribe antidepression medication. Dr. __ did not feel W.M.'s attitude was abnormal. I feel time and more patience on my part will eventually help W.M. back to a stable condition.

This approach did not produce the desired results due to W.M.'s physical and mental depression, lack of motivation plus the fact that he was asleep most of the day.

We still continued to visit and discuss current events but no progress was demonstrated in the time between 1980 through December, 1981. At this time, I was working on my Master's Degree at Texas Woman's University and the pieces of the puzzle began to fit together. The evidence was pointing to an electrolyte and endocrine disorder as the major etiology.

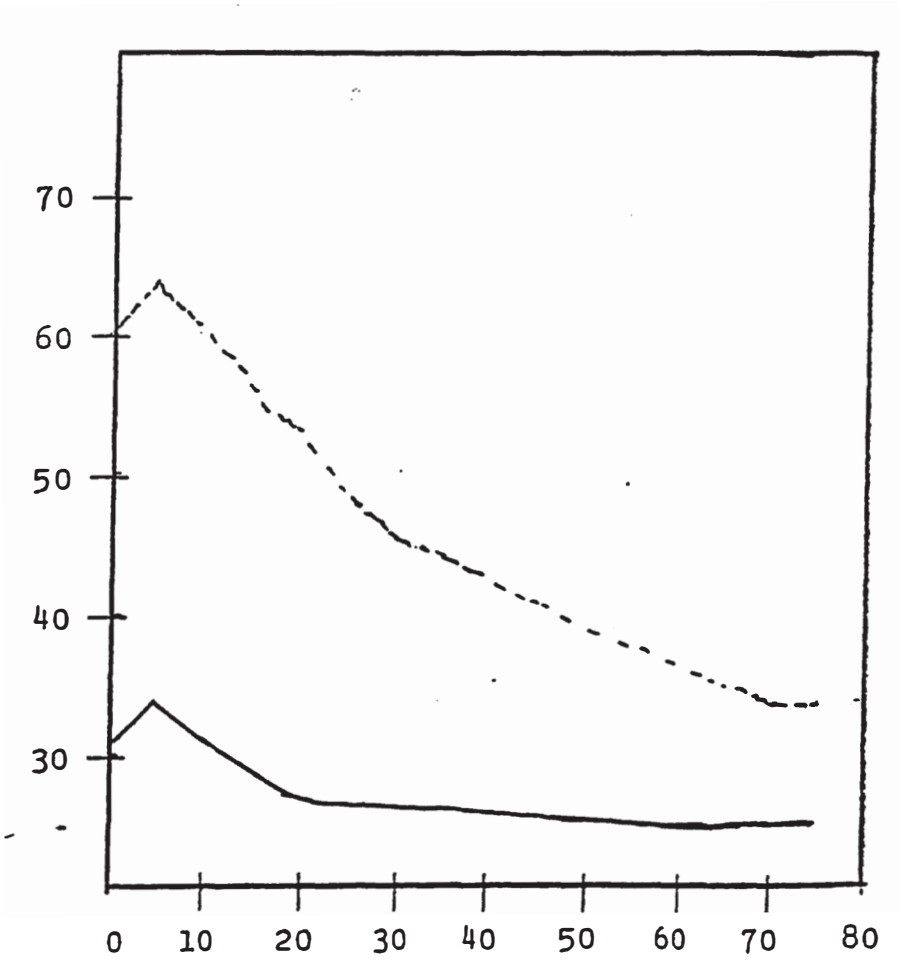
The past medical resords of the patient in this case were reviewed to determine if any physician had conducted an electrolyte profile and also to determine if blood tests had been done to address the problems with hypersomnia (See Appendix A. January 25, 1980). The sleep cycle disturbance was described as secondary to his hypothalamus disturbance and his cerebral dysfunction from his meningitis. The

patient, if not awakened, would sleep twenty-hours. This condition had persisted from the time he left the hospital in December, 1978. The patient also complained of being cold at all times. The strategic role of the hypothalamus in regulating the metabolic functions and in this case the thyroid gland specifically had not been evaluated. The patient was exhibiting all symptoms of hypothyroidism; in adults, it is marked by decreased metabolic rate, tiredness and lethargy that are due to deficient secretions of the pituitary hormone thyrotropin. Thyroxin, due to stimulation of thyrotropin, is secreted from the thyroid gland. The secretion of the thyroid gland has a profound effect on the metabolic rate. The hormone thyroxine stimulates the oxidation of food molecules in most of the cells in the body. That is, it increases the rate of fuel and oxygen consumption and of ATP and heat production. The intracellular target of the hormone seems to be the mitochondria. In the absence of thyroxine, the basal metabolic rate may fall to 50% of its normal value (Griffiths, 1981). See Figure 2.

In order to get an uncontaminated physical including and electrolyte profile and blood analysis, the patient's wife agreed to take him off of all medication for ten days. The masking effect of medication on a patients true profile has been well documented (Frohman, 1978). Ritalin was the

Figure 2

Comparison of Abnormal and Normal Metabolic Rate and Changes over Time (Male Population)



medication the patient had been using. As Ritalin is one of the only drugs that can act as a stimulant or a depressant on the C.N.S., it was necessary to see what the patients behavior would be without his medication. He had been off of Ritalin for a week in the past three years so no problem was anticipated. His prescribed dose was two tablets, 10mg. each, per day but he was rarely awake before 3:00 p.m. to 5:00 p.m., this prohibited his taking the second pill.

Five days after the patient had been off of Ritalin we were having one of our Algebra therapy sessions that had been instigated to improve visual motor control and test the patients long-term memory as well as other sub-skills; at this session I noticed lack of arousal and attention. At first, my thought was the five days without Ritalin had contributed to his disorganized state. The patient started to work a problem, complained of being dizzy, put his head down and blacked out for about one minute. He had taken one Elavil earlier in the day and had slept in the chair until I arrived. The name Elavil indicated to the patient's wife this was an "upper," this was the reason he received the medication to keep him awake. The combination of no Ritalin and a powerful anti-depressant elicited the blackout. A registered nurse, who was the neighbor, checked him

and noted low blood pressure but no other serious problems.

The appointment with the family physician, a specialists in internal medicine, confirmed the hypothesis. The patient's low blood pressure was 74/50 at the time of the office visit. The blood test was done and finally a true profile of his condition was reported one week later (See Appendix A., 1981). The symptoms of a hypoactive thyroid which had been the observation for two years were finally not masked by the various drugs the patient had been on for three years (See Appendix A for summary of drugs administered). The tests also showed low sodium profile. The antiacids he had been on for years contained aluminum and magnisium. This combination forms unabsorbable complexes with dietary phosphate (McGilvery, 1979). Attention to the phosphate and potassium (K) balance after a pathological disturbance of the metabolism must be a consideration.

Requisite to the resting cell membrane potential are the sodium-potassium balance for any neuronal activity, and in this particular case the functionality of the brain was severely impaired.

The effects of hypothyroid compounded by low sodium and bound potassium kept the patient asleep, and when he was awake he was at a non-functional level physically and mentally due to lack of cortical arousal.

The medical records did not show that an electrolyte profile or thyroid test had been done since the patient was released from the hospital in 1978; this information was also confirmed by the patient's wife.

Hypothyroidism or hyponatremia alone could have elicited the behavioral responses that are the emphasis of the present research. If this had been the only symptomology, and no major insult to the brain had occured, the problem would have been addressed and resolved. The initial spinal meningitis was the only problem that was acknowledged that received specific treatment. The secondary and far more debilitating problems were not considered.

Significant hypothalamic damage was reported from 1978 to 1981 in many different physician's reports. Also in each report there was a notation that the patient's wife reported, "he sleeps all the time and never gets up voluntarily." The depression was reported repeatedly, and the MMPI picked up on depression. All of the consults are included in Appendix A.

The primary etiology was addressed but the secondary diagnosis was not treated. This patient has been in a somnambulistic state since September, 1978. In December, 1981, he had a complete, unmasked electrolyte profile, and the results are in Appendix A. His blood pressure was up

from December, 1981-74/50 to 110/70 in April 1982. An anti-diuretic and supplimentary thyroid hormone have this gentleman awake and functioning. The behavioral and emotional aspects of prolonged thyroid and ADH deficits will require an extensive rehabilitation program after a complete neuropsychological evaluation has been done. See Table 2 for Physical Evaluation.

Table 2

PHYSICAL EVALUATIONS 1978-1979

1.	Bi-lateral 3rd nerve palsy	1980
2.	Partial and mild increased reflexes and toe signs in lower extremities	1978
3.	Significant thalamic and hypothalamic cerebral dysfunctions	1978
4.	Left hemiparesis	1978
5.	Severe ataxia	1978
6.	Hypersomnia	1978
7.	Droopy left eyelid	1978
8.	Double vision - some blurriness of vision	1978
9.	Plosis in left eye - weakness of adduction of the left eye and elevation	1978
10.	Minimal features of pseudobular palsy	1978
11.	E.E.G. Abnormal left cerebral dysfunction	1978
12.	C A T - Normal (1979 compared to 1978)	1978 & 1979
13.	Oriented to time and place - O.K.	1979

See Appendix A for detailed descriptions.

RESULTS AND DISCUSSION

Neurobiological Evaluation

A neurobiological evaluation of the patient in April, 1982 using a complete battery of tests showed the following results. On the Weschler Adult Intelligence Scale-Verbal I.Q. 97, Performance I.Q. 100, Full Scale I.Q. 97. This falls within the normal range of intelligence. Comparison of this score and the 1980 score are presented in Figure 3, and Table 2. Of considerable interest that impacts on the patient's crystalized or pre-morbid I.Q. was on the Vocabulary section he refused to answer words that pertained to his previous profession as a mechanic. He also refused to answer or respond to words with an emotional connotation. Compared to the 1980 evaluation he had dropped from a Verbal I.Q. Score of 102, but had increased from a Performance I.Q. Score of 88 in 1980 to a Score of 100 in 1982. In 1980, he was able to recall six digits forward and four digits backwards which is within normal range, in 1982 he was able to recall seven digits forward and six digits backwards. Results are presented in Table 2. Digits measures attention, perseveration, image persistance, distractability and short term memory. Digits backwards are also an indicator of spatial memory, auditory memory and this subtest

is among the most sensitive tests of brain injury, being somewhat more sensitive to left brain injury than to right.

Figure 3

Test Scores Achieved on the Weschler Adult Intelligence Scale Before and After Administration of Hormones Thyroid and Antidiuretic

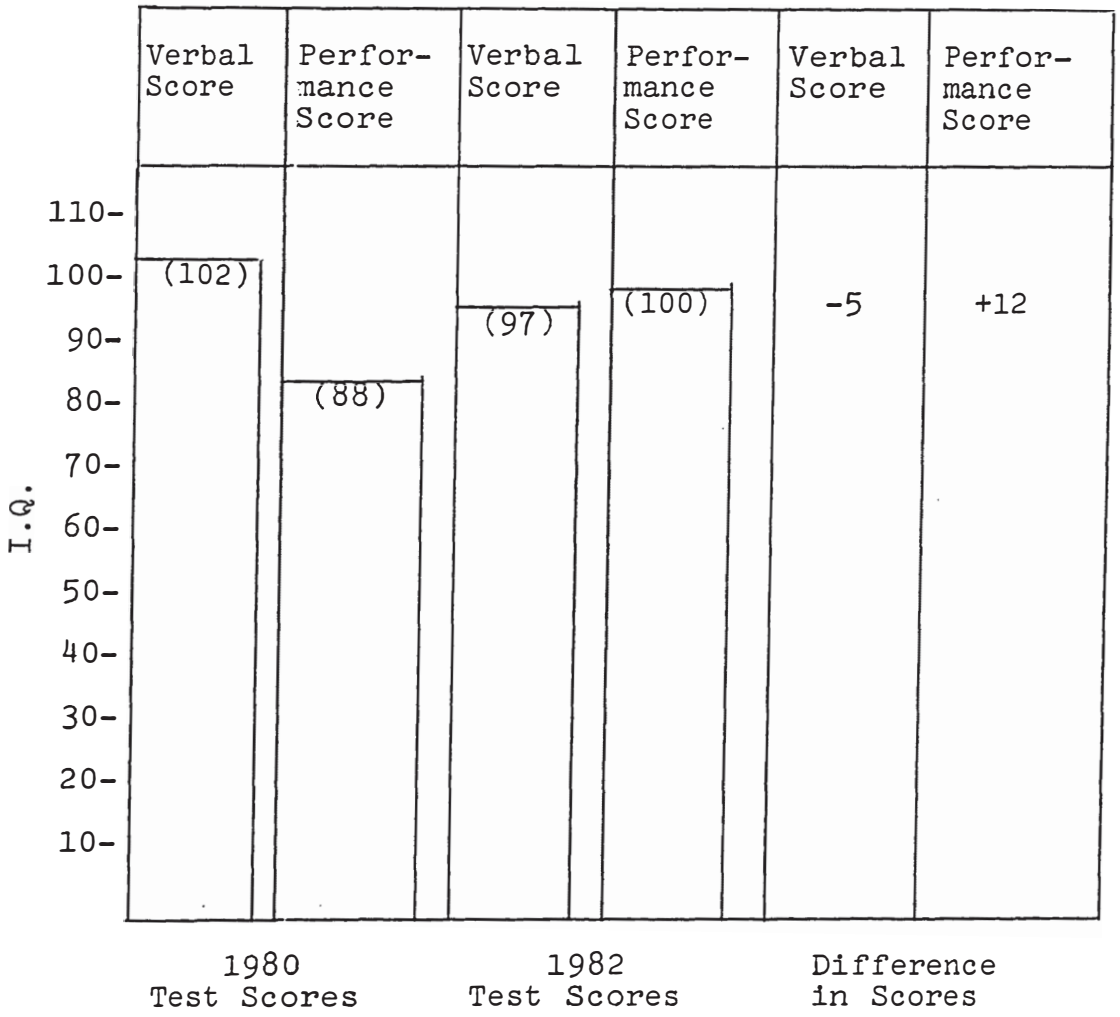


Table 2

Summary of Test Scores on the Wechler Adult Intelligence Scale: Comparison from 1980 to 1982 Scores

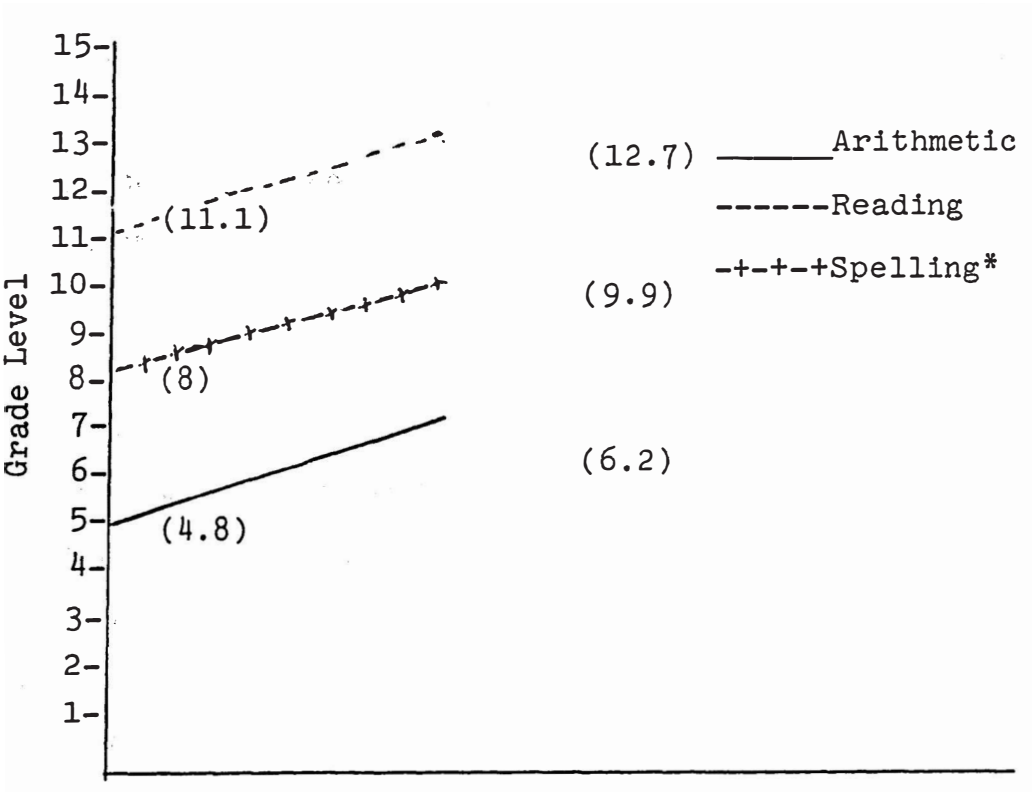
Verbal Tests Performance Tests	Raw Score	Scaled Score	Verbal Tests Performance Tests	Raw Score	Scaled Score
Information	*	*	Information	21	11
Digit Span Forward	6		Digit Span Forward	7	
Digit Span Backward	4		Digit Span Backward	6	9 total
Vocabulary			Vocabulary	42	9
Arithmetic			Arithmetic	10	9
Comprehension			Comprehension	22	11
Similarities			Similarities	7	6
Verbal Score			Verbal Score		54
Performance Tests			Performance Tests		
Picture Completion			Picture Completion	15	13
Picture Arrangement			Picture Arrangement	4	2
Block Design			Block Design	21	9
Object Assembly			Object Assembly	32	10
Digit Symbol			Digit Symbol	30	5
Performance Score			Performance Score		39
Sum of Scaled Scores		I.Q.	Sum of Scaled Scores		I.Q.
VERBAL		102	VERBAL	54	97
PERFORMANCE		88	PERFORMANCE	39	100
FULL SCALE		102	FULL SCALE	93	97

*The Raw Scores and Scaled Scores were not in the previous evaluation. Only Digits Forward and Digits Backward.

Similarities Subtest was one of the patients lowest scores. It is sensitive to brain injury, especially in the left temporal and parietal areas; these were the areas most effected according to the medical reports. This is a test of verbal abilities and the patients ability to find a common category for two objects, however, it does not correlate highly with verbal I.Q. The Arithmetic Subtest, tests the patient's ability to do arithmetic problems without a paper or pencil. Immediate memory, attention, spatial memory are required in order to process from auditory input to expressive language output. Errors may not represent a real deficit in arithmetic skills therefore the Wide Range Achievement Test was used to determine whether a deficit in arithmetic skills existed. The results are presented in Figure 4. The only other area that showed a depressed score was digit symbol. The patient understood the verbal instructions but persisted in doing all of one symbol/number instead of doing them in the correct sequential line form. Repeatedly he was stopped and instructed to do one right after the next but he insisted on doing all the symbols for #1 first before he would start on #2 etc. This test is sensitive to visual retention, psychomotor skills and eye hand coordination. It is an indicator of lack of synthesis in the parietal, temporal, and occipital lobes.

Figure 4

Test Scores on The Wide Range Achievement Tests
Before and After Administration of Hormones
Thyroid and Antidiuretic



*The Spelling test was done verbally in 1980 the evaluator said a written test would have earned the patient a much lower score

Block design, picture completion and object assembly were within the normal range. The block design requires visual-spatial skills; a depressed score on this is a good indicator of constructional apraxia. Object assembly requires speed and psychomotor coordination. Picture completion is excellent for testing recognition and discrimination; this subtest also demonstrates the person's social I.Q. Grooved Peg Board Test was administered to check psychomotor coordination the results obtained were: Right (dominant hand) two minutes-forty seconds with one drop; Left hand-one minute-fifty five seconds. Tactile, visual motor integration and fine visual/motor skills were depressed on the right hand. Porteus Tests-Vineland Revision Mazes elicited the following results: Portus Test XIV-time fifty seconds, two errors (out of line), Portus Test Adult I: two minutes-twenty one seconds, restarted one time, six errors. Planning and visual accuracy are involved in this test, as well as spatial orientation. There is a doubt that this is a valid test of patient's ability to orient in the real environment, W.M. appears to have spatial orientation when traveling in a car, can correctly give directions to a familiar location and has no problem in left/right commands. Trail Making Parts A & B were administered: Trails A, time thirty nine seconds which is within the normal range. Left

frontal lobe used in sequencing and scanning appears to be intact. Trail Making B showed the following results: Time, one minute and nine seconds, with three errors. This part of the Halstead-Reitan is sensitive to visual scanning, ability to shift mind-set, and is considered to be an indicator of organisity on both parts A & B.

Wide Range Achievement Test: Reading-Grade Rating 12.7-, Percentile-97. Spelling-Grade Rating 9.9-Percentile -81. Arithmetic-Grade Rating 6.2-Percentile-63. Handwriting on spelling test average or above average. See Appendix C for examples of handwriting before and after instigation of thyroid hormones and antidiuretic. This patient's language skills fall in the superior range. His pronunciation on old and new words, phrases and complete paragraphs is clear and concise. This may not always be apparent to strangers as he has become somewhat concious about his relationship with persons who did not know him prior to his illness.

W.M.'s scores on the criteria sub-tests showed the following results:

1) Recognition and Discrimination-only one error out of a possible twenty.
2) Spatial Orientation-no errors out of a possible twenty.

3) Proverbs Test-(40 question long form) within normal limits.

4) Word Fluency-average range spelled out verbally, time sixty seconds for each three trials.

5) Finger Tapping, on ten seconds, three trials per hand, well within the normal range. The same results were obtained on the sixty seconds, three trials per hand; both hands showed the same mean score with a slight decline on the left hand on the last two trials. This was the side that was affected after meningitis in 1978.

6) Line placement of letters of the alphabet in the correct sequence with and without cue showed all trials were in correct spatial orientation. The same results were obtained with slanted line rotation from a 180^{o} line base.

7) Berry Visual Motor Integration-no distortion in twenty four copying tasks.

8) Subtracted 3 from 20 and kept subtracting from each new number all the way down - no errors and extreme speed.

9) Detailed Recognition 1940-1970's, 22 correct out of a possible 30.

Peabody Picture Vocabulary Test - I.Q. 117 (only 6

missed out of a possible 175 responses. Auditory Comprehension-Excellent no receptive aphasia shown. Patient followed all commands and instructions with no hesitation except when he was too fatigued from the testing session. The tests included: touching objects, body parts, left/right discrimination, (including the command to touch your right elbow with your right hand), the comment was, "are you kidding that is impossible." Skin writing tests were performed with no errors using both numbers and letters of the alphabet. Between the finger, and two point discrimination tests were administered with no errors. Sensory tests performed on all body parts using light touch elicited the correct responses. Raven Progressive Matrices: I.Q. 120-90 Percentile. This is considered a test of spatial reasoning.

Culture Fair, Scale 2, Form B-Time ran out on each of the four tests, however, only one (1) error on Tests 1,2,3, and no error on Test 4. MMPI: Paranoid trend shown is possible due to treatment received and inability to get appropriate relief. This is consistent with an elevated score in depression. Resentfulness, hostility and anger are also indicated. Sensitivity is also noted. This person is having difficulty expressing anger towards others, and this anger is therefore being focused inward with self-blame and

covert blame directed towards others. Schitzoprenia scale is within normal limits. This is a marked improvement from 1980 when W.M. had clinically significant elevation on the schitzoprenia scale due to mental confusion and lack of orientation.

See Appendix C for handwriting sample, algebra comparison from December, 1981 to April, 1982 and other tests.

Hyponatremia

The results of the low sodium and its impact on the functionality of all body processes was diagnosed December 8, 1981. The sodium in mEq. per liter was 130. The normal range in the blood in adults should be 139-146 mEq. per liter. Chloride was also low. The profile showed chloride as 95 mEq. per liter. The normal range in the blood in adults should be 99-108 mEq. per liter. The results are summarized in Figure 5 and Figure 6. The potassium was 4.5 mEq. per liter which falls within the normal range of 4.1 - 5.6 mEq. per liter. Florinef 1.0 mg 1 tablet in the morning was prescribed at that time. The patient was to continue his Ritalin and Tagamet. This was the first time an electrolyte profile had been done since Mr. M's release from the hospital in December, 1978. It is impossible to determine at what time in the past he developed hyponatremia. The patient's decline in functions including memory could have started at any point and have been masked.

The post-traumatic shock that is the natural effect of such a severe and prolonged illness has no set time frame for determining how long the complete healing processes will take. One physician indicated this healing would not be complete for two years. The gradual decline in Mr. M's mental processes was considered as a result of his illness,

Figure 5

Electrolyte Profile-Sodium-Changes form 1978 Through 1982. Values Expressed as mEq. per liter. December 1981 to April 1982--Emphasis.

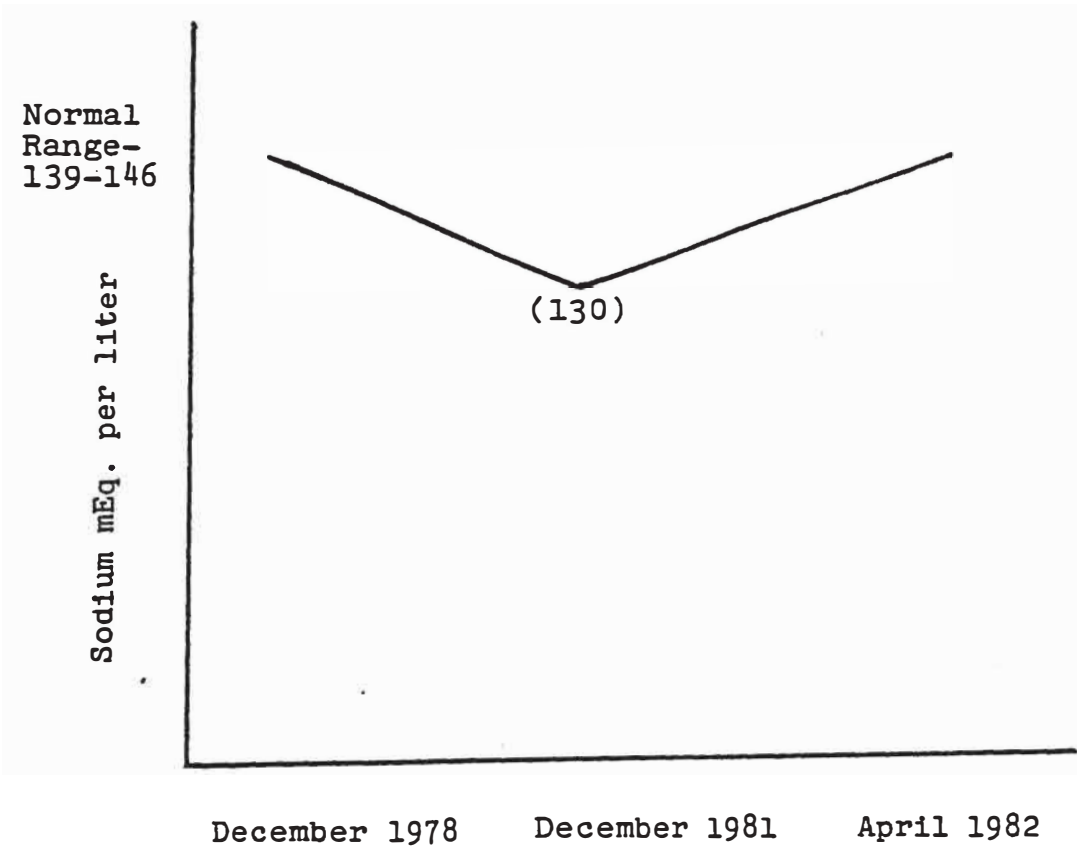
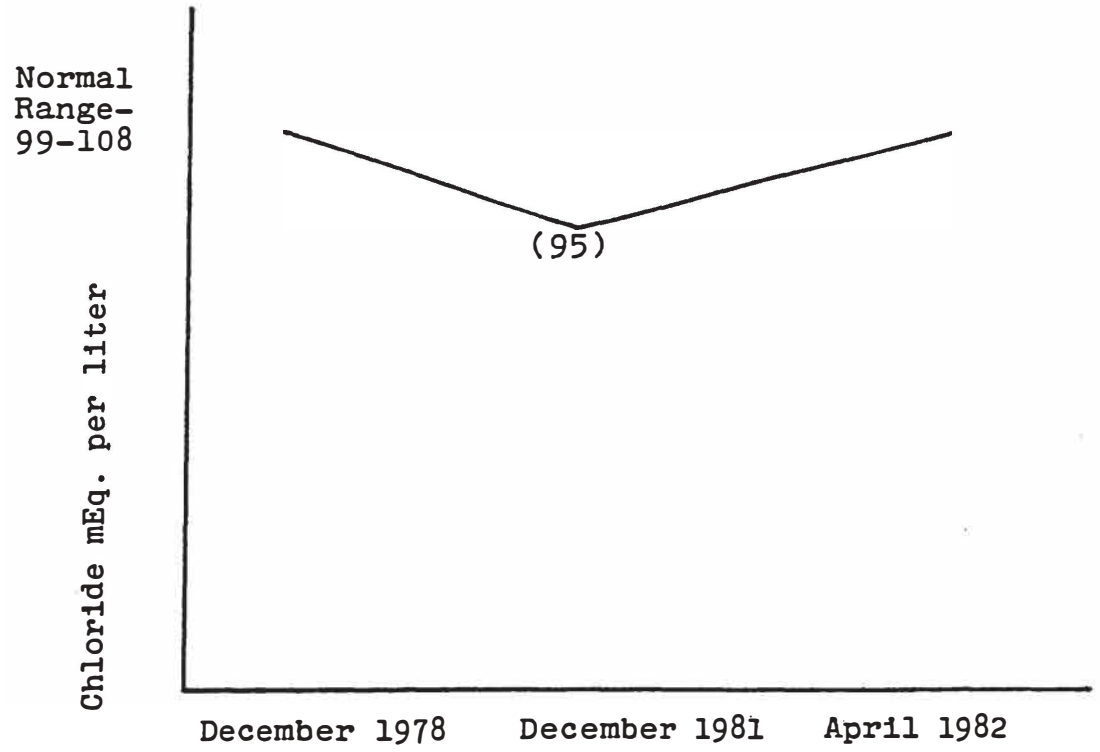


Figure 6

Electrolyte Profile-Chloride-Changes from 1978 Through 1982. Values Expressed as mEq. per liter. December 1981 to April 1982--Emphasis.



depression over his sons death while he was in the hospital himself, and his inability to work at his previous profession. His clumsiness when he tried to do daily living tasks further upset and depressed him. The family at this point were treating him like a helpless baby; many close friends treated him like he was deaf and retarded.

Hypothyroidism

The same evaluation of the electrolyte imbalance on December 8, 1981 also showed he had a T-3 of 40.5. The normal range is 44-59. He was placed on one grain of Proloid daily at that time. His blood pressure was extremely low -- 74/50. His blood pressure about April 8, 1982 had gone up to 110/70. His urine was essentially normal with a Ph of 5.0. Blood count at this time was entirely normal with 14.7 grams of hemoglobin and 6,600 leukocytes with a normal differential and adequate platelets.

The mental dysfunctions shown by Mr. M. are found in patients with hypothyroidism alone. The mental dysfunctions fall essentially under two headings; impairment of cognitive functions and disturbance of affect. All the symptoms Mr. M. has been demonstrating since his illness are the same as those in a hypothyroid population prior to medication. The results of low thyroid hormones are shown in Figure 7, Figure 8, and Figure 9.

Figure 7

Changes in Blood Pressure After Administration
Of Hormones-December 1981 to April 1982
Thyroid and Antidiuretic

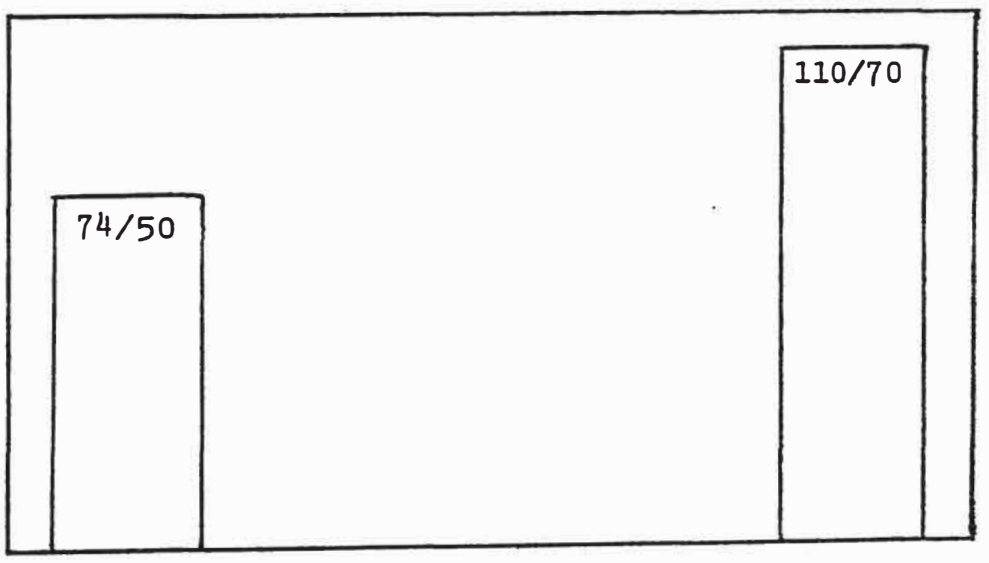


Figure 8

Thyroid Values (T-3) From 1978, 1981 to 1982
Changes Shown Over Time (1978 Hospital-
1981 and 1982 Outpatient) Normal
Range Shown for Males

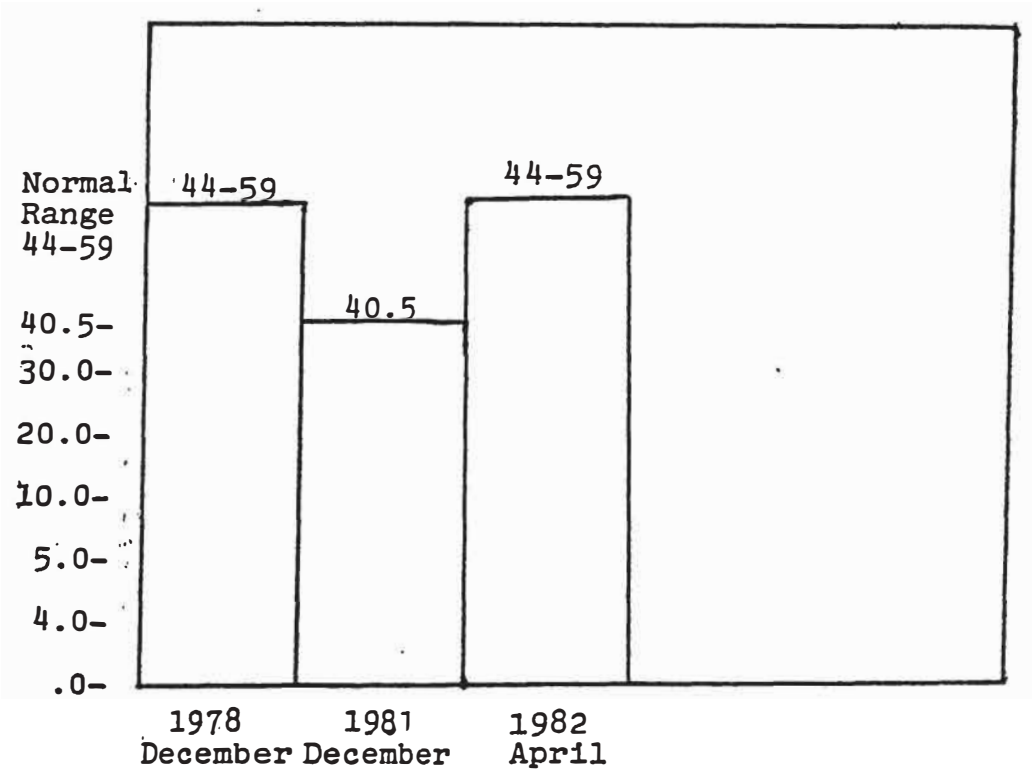
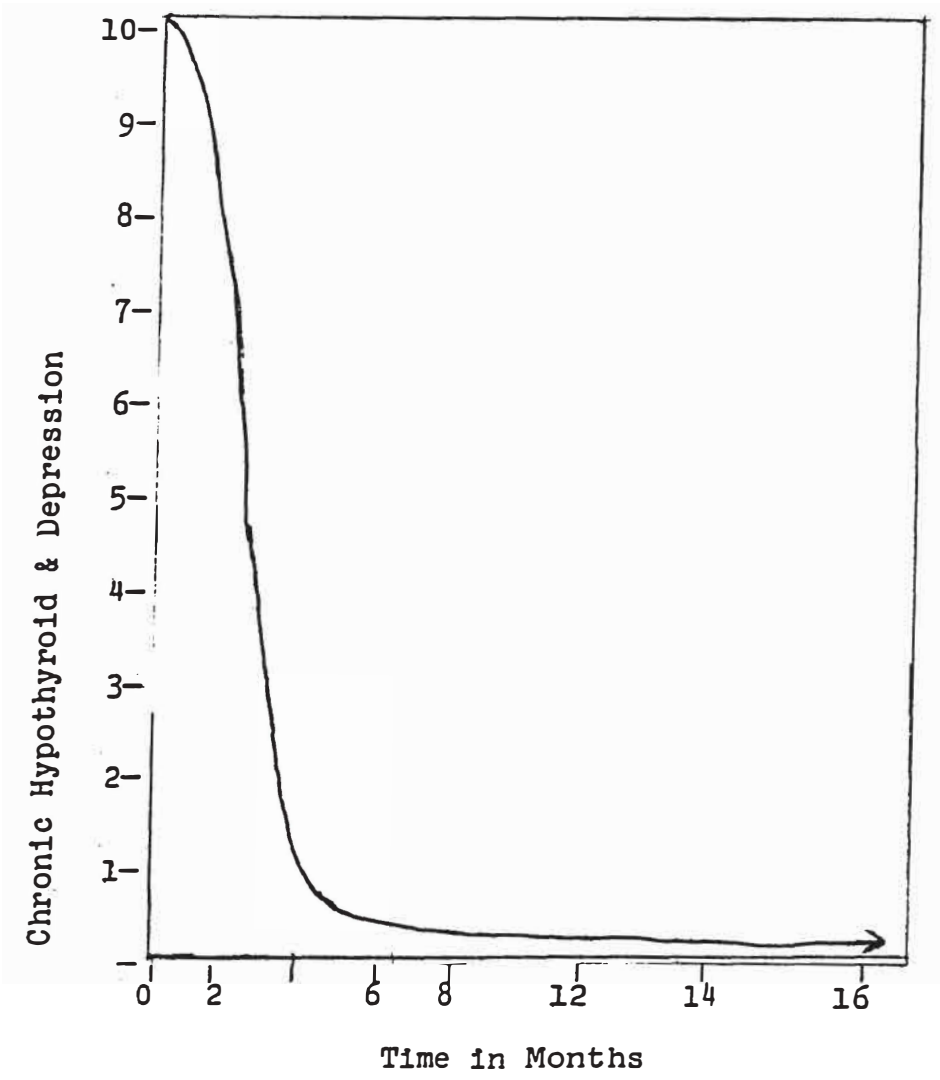


Figure 9

Changes over Time and Depression
Endocrine (Thyroid) Dysfunctions



There is no way of determining at what point he developed hypothyroidism. The hypersomnia was noted from 1978 through 1981 in the medical records. Hypersomnia, memory problems, motor retardation and depression appeared too often to be ignored. The medical reports did not suggest the secondary envolvement might be the step limiting factor in this patient's recovery.

The evaluation in December, 1981 speaks for itself as to the patient's most debilitating effects of the 1978 bacterial meningitis. The thalamic-hypothalamic pituitary axis and the damage to this critical area are the primary etiology in this patient's case.

DISCUSSION

The masking effect of the initial lable of spinal meningitis has now been resolved. The electrolyte and endocrine dysfunctions have now been evaluated and proper treatment of the secondary considerations, hypersomnia due to hypothyroidism and hyponatremia, have been resolved. Thyroid hormones and antidiuretic hormones have elicited the desired physiological and cognitive results. The test results both physical and mental confirm the hypothesis.

Compared to Mr. M's 1980 evaluation, on the tests in 1982 he demonstrated a marked improvement in all areas. No difficulty was observed in solving pencil and paper arithmetic problems. The poor vision and difficulty in sustaining his attention-concentration that was present in 1980 has improved significantly. Mr. M. in 1980, did not know what year it was and thought he had experienced a stroke. He also thought his children were still living at home and ten years younger than their actual age at that time. At times he talked about trips that were taken ten to twenty years prior to various cities. He refered to the trips as last week. Mr. M. now knows what year it is and the retrograde amnesia has abated for events with the exception of the time in 1978 when he was in the hospital with meningitis.

He has a brief but sketchy memory of a few of his therapists. No confabulation has been demonstrated as he previously had done to fill in the blank spots in the past.

He repeatedly demonstrated eye hand coordination and psychomotor skills when playing the computer pong-games. No effort was made by several people who played against him to let him win. In all instances, he beat this researcher by a sizeable score. He has obviously learned compensatory tactics for the double vision due to damage to the third cranial nerve. His depth perception and eye hand coordination was also evaluated using an indoor golf putting device. He experienced no difficulty in balance or in correctly driving the ball into the opening. He beat this researcher, again, by a sizeable score.

Three weeks after he had been placed on thyroid hormone and an antidiuretic he showed fantastic improvement in his ability to do algebra problems. One of the sessions prior to the medication, he had taken three hours to do the problems. After medication, similar problems only took him thirty minutes with no lack of confidence, the work was not messy and the problems were more difficult than the previous problems that took three hours. Samples are in Appendix C.

He is completely able to do minor repairs on cars or

correctly and in detail describe each step on a complicated repair. He plays bridge with all the planning, sequencing and verification of each move that envolved an intact frontal lobe in order to execute these processes.

The right hemisphere requisite for rhythm in dancing, singing and non-verbal skills is intact. He has not lost his previous expertise in dancing with perfect rhythm. His balance is also intact while dancing.

One final factor that further demonstrates the hormone deficiencies have been successful in increasing cortical arousal is W.M.'s sex drive is up for the first time since September, 1978. The thalamic-hypothalamus pituitary axis also modulates the sex hormones.

CONCLUSION

Four years of following this patient's progress and close observation of what he could not do prior to the diagnosis and administration of thyroid hormone and anti-diuretic hormones leaves no doubt in my mind that the secondary damage to the hypothalamus pituitary axis was what inhibited the patient's rehabilitation.

The fact that he is now cognizant that three and one half years have passed without his being aware of what has been going on, plus control of his life has been out of his hands, has had severe ramifications. In spite of repeated explanations of why he could not function or remember normally with an endocrine and electrolyte imbalance, and his complete understanding of his problems, the emotional and psysiological deficits that were a result of prolonged hypothyroidism and hyponatremia, have convinced Mr. M. he cannot try to resume a normal life. He has no motivation to try.

The learned helplessness, fear of failure and dependency fostered rehabilitation are the direct result of not addressing the secondary and far more debilitating damage to the hypothalamic-pituitary axis. It is not known at the present time what the effects of prolonged hyponatremia

have on memory or the effects of prolonged hypothyroidism on prognosis for recovery.

IMPLICATIONS FOR FUTURE RESEARCH

Results from this study suggest that the deleterious effects of hypothyroidism and hyponatremia on patient recovery after brain damage need to be widely promulgated. The effects of low A D H on memory processes indicate the masking effect of cerebral trauma might prevent adequately addressing secondary damage to the brain. Clinical signs of hypothyroidism may be masked by the primary etiologies such as meningitis. The masking effect of various drugs can further confound evaluation of the major and more debilitating problem of the patient.

Education of the patient's family following brain damage to a family member should receive more emphasis. Education could include what to do in order to best help the patient reattain independence in self-care; this could be instigated while the patient is still in the hospital. The patient's family should have an understanding of what dependency fostering rehabilitation leads to and the impact on his or her recovery. Low self esteem, fear of failure, and learned helplessness are quite often the end results of lack of information by the caretaker or patient's family.

Results indicate that an inappropriate follow up of the patient's mental status and physical status resulted

in a prolonged delay on his recovery. It is proposed that whenever a patient exhibits a memory deficit over a prolonged time period that a complete electrolyte profile be done. The patient should be off all medication prior to an evaluation. Progress of the patient's mental status should begin in the hospital with careful notations made on follow up visits. Should there be an abnormal decline at any point secondary complicating factors should be evaluated as a seperate etiology from the primary brain damage. Seperate consideration of the presenting problem could be the step that permits the full recovery of the patient back to a normal life.

The last consideration is the cost factor. Once a patient has been declared disabled, all taxpayers are affected indirectly. Disability benefits are continued for life. Would it not be more cost effective to return as many patients as possible to their previous occupation than to have to pay for their support?

SUMMARY

CASE REPORT

ENDOCRINE AND ELECTROLYTE CONSIDERATIONS
RECOVERY FROM BRAIN DAMAGE

Alice Aline A. Clark

A single case is presented in which, following traumatic injury involving brain damage to the right and left hemispheres, the subject's condition deteriorated after his release from the hospital. The subject had spinal meningitis in September, 1978 and was released in December 1978. The subject was male, caucasian, right handed, and 56 years of age at the time of insult. His condition was stable at the time of release. No recommendations other than physical therapy were indicated at the time of release. The subject had hypersomnia from the residual effects of the trauma that lasted until December 1981. This secondary problem (hypersomnia) and indications of hypothyroidism had not been evaluated. An electrolyte profile was indicated and a complete blood analysis to determine the etiology of the presenting symptoms. Neurobiological and neuropsychological evaluations were done. The result of the electrolyte profile showed an abnormally low sodium count (130-normal range for males 139-146); the chloride count (95-normal range for males: 99-108). The thyroid value, (T-3) was 40.5 (normal range for males - 44-59). Blood pressure in December was 74/50. The subject was placed on thyroid hormones and an antidiuretic at this time. The results in April 1982 showed; blood pressure 110/70, chloride values within normal range, and sodium within normal range. Neurobiological tests showed the subject's I.Q. on the Weschler Adult Intelligence scale to fall within the normal range. (Verbal I.Q. 970, Performance I.Q., 100, full scale I.Q. 97. All of the criteria subtests were within normal range. The MMPI was within normal range. It was concluded that secondary thalamic-hypothalamic damage was the problem that did not receive consideration and prevented this subject's rehabilitation.

APPENDICES

APPENDIX A

LLOYD J. GREGORY, JR., M. D.
ALLEN C. BULLOCK, JR., M. D.
AMERICAN GENERAL BLDG.
2727 ALLEN PARKWAY
HOUSTON, TEXAS 77019

INTERNAL MEDICINE AND DISEASES OF THE CHEST

April 21, 1982

Walter Zetusky, M.D.
Texas Woman's University
1100 M.D. AndersonBlvd.
Houston, Texas 77030

Re: F. W M

Dear Dr. Zetusky:

Mr. Walter M is a post-Meningitic who was found to have a T-3 of 40.5 on December 8, 1981. He was placed on one grain of Proloid daily at that time and is continued on same. His blood pressure about April 8, 1982 was 110/70. His serum sodium and chloride were low on electrolyte profile in December 1981. Etiology of such is undetermined. His urine was essentially normal with a Ph of 5.0. Blood count at this time was entirely normal with 14.7 grams of hemoglobin and 6,600 leukocytes with a normal differential and adequate platelets. I hope this answers your question. The man is also on Tagamet and Ritalin.

Lloyd J. Gregory, Jr. M.D.

ml

4-27-82

Re. F.W. M
Phone conversation

74/50 Blood pressure---Dec. 81
Sodium 130
Potassium---4.5
Chloride---95

A D H---Florinef 1.0 mg 1 tablet
in the morning

MO - 118 5/75

NEUROPSYCHOLOGICAL CONS A: C:

NAME: W F. M AGE: 57 SEX: Male OCCUPATION: Retired servic station operator. REFERRED BY: , M.D.
CONSULTANT: , Ph. D.

REASON FOR REFERRAL: Neuropsychological evaluation was requested to determine the extent of deterioration in this patient's cognitive functioning secondary to a histor of encephalitis in 1978.

EVALUATION PROCEDURES: Clinical Interview, Wechsler Adult Intelligence Scale, Wechsl Memory Scale, Wide Range Achievement Test, Word Naming Tests, Finger Tapping Tests, Television Name Writing Test, Trailmaking Tests A & B, Sensory-Perceptual Examinatio MMPI, Test of Attentional and Interpersonal Style, Cornell Medical Index, and SCL-90-R.

BEHAVIORAL OBSERVATIONS AND SIGNIFICANT INTERVIEW DATA: During the examination the patient was pleasant and cooperative, but easily confused and occasionally disoriented During the interview his train of thought would wander if external direction was not provided. He did not appear to know the age of his oldest son or daughter and at one point he was unable to remember what year this was. His affect was appropriate, but depressed. In particular, when talking about his youngest son, who died at age 21, reportedly due to cancer, he exploded into tears, perseverating in this response whenever the focus of discussion led him to an association with his son's death. Mr. M stated that he and his wife decided he should come to Hospital for evaluation this February because it was clear he was unable to function normally mentally. Specifically, he stated that he is no longer able to coordinate his "hands and head". He has also observed a significant decrease in is learning ability and great difficulty with his memory. Mr. M is a service station operator and mechanic by profession, but for the past year he has been unable to work. He has additionally noted that his interest in his business has diminished and that he has in the past several months been sleeping more than usual, with little interest in getting up. Although he couldn't remember the exact date, he felt that he had had a stroke during the past year, which was diagnosed after his wife was unable to wake him one morning and he was taken to the Hospital for evaluation. However, he did not recall being told that there were any changes in his physical condition or noting that there was any major change in his mental abilities at that time.

The patient was born in Louisiana and moved with his family to Houston in 1929. His father originally was employed with the railroad and then subsequently with a chemical company where he became a vice-president in the Traffic Department. He has two sisters, one of whom is divorced and works for him in his service station and garage in which he has a half interest with a partner. The patient reports an uneventful, happy childhood. Both parents are living in Houston and described as each being a "great person". Mr. M completed high school and attended one semester of college at Texas A&M University, dropping out because of failing grades.

CONT

M.D.

M , W F. ds

0 79 77 71 3

, M.D.

D&T 2-18/19-80

History, Physical Examination, Consultations, Progress Notes, Discharge Summary

CONs 2

He states this was not upsetting to him because he never had a strong interest in academic subjects. Following this, he worked for a Houston shipbuilding company, working on a scaffold, before becoming involved in the automobile service and repair business. He served in the Army in 1942 for ten months, doing construction work in San Antonio until he was discharged medically for a bad knee and bad back. Prior to his most recent difficulties the patient reports being generally healthy and able to work.

NEUROPSYCHOLOGICAL TEST FINDINGS: The Neuropsychological evaluation shows that the patient is currently functioning within the normal range of intelligence with a Verbal IQ Score of 102, Performance IQ Score of 88 and Full Scale IQ Score of 96. His immediate memory and learning ability is significantly reduced in relation to his level of intellectual functioning as he obtained a Memory Quotient of 79 on the Wechsler Memory Scale. His new learning ability for both verbal and non-verbal material is significantly impaired with even more severe deficits observed in his immediate and 30 minute delayed recall for complex semantic material (a brief story passage) and geometric designs. He also shows a marked deficit in his flexibility in organizing different cognitive functions as evidenced on the Trailmaking Tests, which require visual search and sequencing of numbers or number/letter alternations in ascending order. Mr. M also evidenced a moderate deficit in his verbal concept formation and a construction dyspraxia. His bilateral finger dexterity is moderately impaired, whereas he shows only a mild impairment in his grapho-motor coordination on the Television Name Writing Test with both hands.

Despite these negative findings, however, there is substantial preservation of higher cortical functions as evidenced in the patient's normal verbal IQ score. Indeed, his vocabulary was above average and his general fund of information and comprehension of social conventions and common sense judgment was average. He was able to recall six digits forward and four digits backward, which is within the normal range. His abstract verbal reasoning and his ability to perform mental arithmetic, while slightly below average, is within normal limits for an individual of his age. While he commite some errors on the aphasia screening test, these appeared to be due to his tendency to become confused rather than any real deficit in his expressive or receptive language abilities. Mr. M exhibited considerable difficulty in solving paper and pencil arithmetic problems, but this was in part due to his poor vision and difficulties in sustaining his attention-concentration. Whereas he earned a grade equivalent score of 4.6 on the Arithmetic test, he performed at the 11.1 grade level on the Reading (Word Recognition) Test. His Spelling skills are judged to be at approximately eighth grade level, based on his spelling the words out loud rather than writing them down (which would have earned him a much lower score). The Sensory-Perceptual examination was generally unremarkable except for some suggestion of suppression in auditory perception in both ears.

Assessment of the patient's current emotional functioning shows that he is experiencing clinically significant levels of emotional distress, principly in the areas of obsessive compulsive indecision and worry, depression, irritability and phobic anxiety, and emotional confusion. His MMPI Profile was within normal limits with the exception of the clinically significant elevation on the schizophrenia scale, which is seen as a reflection of his mental and emotional - M.D.

CONT

M , W. F. ds

0 79 77 71 3

, M.D.

History, Physical Examination, Consultations, Progress Notes, Discharge Summary

MO - 116 5/7

CONS 3

Date & Time

confusion secondary to his organic brain dysfunction. Relative to his other profil scales, there is an elevation in his depression scale, which is also reflective of the impact of the changes in his mental capabilities. On the test of Attentional an Interpersonal Style, Mr. M describes himself as being easily distracted and confused if he has to attend to more than a few external events or thoughts or feelings at any one point in time. He further describes himself as having great dif ficulty in situations where a broad attentional focus is necessary for accurate and adaptive information processing. Presently he also appears to have great difficult in making decisions and is prone to indecisive, obsessive, compulsive vacillation when faced with competing choices. Interpersonally he is seen as being rather sub- assertive and quite unexpressive of his ideas. Affectively he is much more comfort expressing his positive rather than negative feelings.

SUMMARY AND RECOMMENDATIONS: While the Neuropsychological test findings provide clear evidence of impairments in cognitive functioning consistent with the impressic of organic brain dysfunction, Mr. M does show at present relatively good pres vation of his verbal intelligence. These capabilities, however, are probably not readily apparent to the outside observer because of the patient's tendency to become confused and disoriented and because of his marked deficits in his verbal and non- verbal immediate and delayed memory for complex novel material. Although it thus may be anticipated he will have difficulties in dealing with new and complex situati he may be more capable than is readily apparent in dealing with simple, familiar problems and situations. Although use of mnemonic aids (eg. writing things down), may be of only very limited utility in compensating for his memory defects where new learing is concerned, use of schedules involving timely repetition of well learn habit patterns may facilitate maintenance of autonomous functioning on the part of the patient. Establishment of familiar daily routines may also be therapeutic in reducing the patient's dependence on significant others and his clinically significa levels of depression and emotional confusion. It can be expected, however, that when faced with new situations or circumstances requiring more than a limited amount of attention, that Mr. M may become "overloaded" and unable to respond appropria ly. Effective decision-making on his part in such situations cannot be expected and assistance from significant others at such times would be appropriate.

Thank you for referring this interesting patient.

M.D.

M , Wa F. ds

0 79 77 71 3

, M.D.

History, Physical Examination, Consultations, Progress Notes, Discharge Summary

February 4, 1980

Texas Rehabilitation Commission
Division of Disability Determination
1 Chevy Chase 7700 Chevy Chase DRive
Austin, Texas 78752

Re: W . M

Dear Sir:

I have been taking care of Mr. M now for approximately a year. Back in February 1979 he had a short history of headache, fever and had a severe episode of bacterial meningitis which resulted in him being unconscious and having seizures and subsequently gastrointestinal bleeding. He was in hospital for a couple of months at Memorial Southwest and eventually discharged and since that time he has been left with severe bilateral third nerve palsy, mild confusion and memory disturbance, difficulty with his balance and marked difficulty with judgement. He has had emotional incontinence and rage reactions.

I have been taking care of him now for a year and have seen him on a number of occasions, the most recent was four or five days ago and at that time he still had evidence of mild bilateral third nerve palsy with emotional incontinence, crying out, involuntary control of his emotions and some difficulty with his balance.

He has been on a program of Ritalin because of excessive sleepiness and this has helped somewhat but this has not helped his personality problem.

As far as I'm concerned at this time the patient is totally disabled. He is unable to manage and take care of his own affairs. He needs almost constant supervision by his wife. If she were not there to take care of him he would sleep a good part of the day and would probably not even eat. We will send you copies of our letters and evaluation of this on this patient but I feel strongly that he has a very severe residual encephalopathy and cranial nerve dysfunction secondary to his bacterial meningitis. I hope this is the information you require for a disability decision.

January 25, 1980

Re: F. W. M

I saw Mr. M. in my neurology clinic on January 24, 1980. This gentleman appears to be having increasing difficulty. He is having more sleeping problems at night. He has been having periods of rages and outburst and his wife has been having quite a time with him. His neurological examination is unchanged. He still shows evidence of very slow mental function with bilateral third nerve palsy. He has partial and mild increased reflexes and toe signs in the lower extremities. He also has emotional incontenance. I repeated his cat scan about three weeks ago and it does not show any further change in the ventricular size or any other abnormalities, and there is no evidence that he has any further progression of his neurological disease. It is my feeling that he has probably had significant thalamic and hypothalamic cerebral dysfunction secondary to his meningitis, and I think we are seeing a significant residual. I am also a little bit concerned that perhaps the Tagamet may be having some effect on his mental status and I have seen a number of patients who have had lethergene confusion from Tagamet, and I wonder if it would be best to discontinue it for a short time and put him back on antiacids and see how he does. I have also started him on Inderal 60 mgs. a day which may be of some benefit for violent outbursts and irrational behavior. If this is not of benefit, then we will consider the possibility of using antipsychotic medication and some other medications in that field. I will be calling shortly about him in regard to his Tagamet medication.

Notes on F.W. M

Mr. M... is a gentleman who we saw at least two months ago or three months ago with a diagnosis of bacterial meningitis which gradually resolved. He left with numerous deficits such as a left third nerve palsy, a left hemiparesis and some memory problems with confusion which has slowly been resolving. He has also had severe ataxia.

In the last three or four months he has had some problems with hypersomnia where he has increasing sleeping problems. He apparently sleeps most of the day unless his wife arouses him and he is awake at night. He apparently has had this sleep cycle disturbance ever since he had his meningitis.

Neurologically he still has evidence of a left hemiparesis, a droopy left eye lid, partial third nerve palsy and some memory problems.

I think that this patient has a sleep cycle disturbance likely secondary to disturbance in his hypothalmus and his cerebral dysfunction from his meningitis. I thought perhaps a small dose of Ritalin may help him during the day - try to keep him a little bit more awake. I have given him a prescription for Ritalin to take 5 milligrams in the morning and in the afternoon and see how he does. I discussed this with his wife and she will let me know how he does in the next three or four days.

For F.W.M [illegible]

Address _____ Date 1/18/80

℞

LABEL

Ritilan 5mg.
#100
one twice daily

[illegible] _____ M. D.

Non-Rept. _____ Rept. _____ Times Rept. PRN _____

DEPT. of ELECTROENCEPHALOGRAPHY

M.D
M.D.
M. D.

Referring Physician: M.D.

EEG# 53773

E.E.G. Diagnosis: Dysrhythmia Grade II,

REPORT:

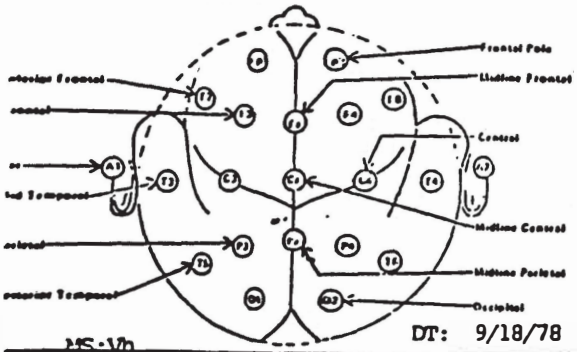
ALPHA: 9-10 c.p.s. activity up to 60 microvolts.

BETA: A small amount of 15-20 c.p.s. activity up to 20 microvolts.

OTHER: There is a moderate amount of 2.5 to 3 Hz per second activity up to 70 microvolts that appears primarily in the left parietal temporal area. At times a small amount of this activity is present also on the right side.

HYPERVENTILATION & PHOTIC STIMULATION: Not performed.

IMPRESSION: Abnormal EEG, indicating left cerebral dysfunction primarily in the parietal temporal area in the presence of some bialteral cerebral dysfunction.



MS-Vh DT: 9/18/78

M.D.

Name of Patient	Age	Date:	Room No.	Patient Number
M F.	56	9/17/79	ICU-13	11811737

70-83-0830 Rev. 8-72

DEPARTMENT OF RADIOLOGY

M. , F. M:57 DOB:3-1-22 #73222311 OP
79-1036 5-9-79 Dr.

COMPUTED TOMOGRAPHY (HEAD): Scans were made before and during intravenous infusion of 300ccs of 30% Renografin. The ventricles and subarachnoid structures are of normal size and shape and in normal position. No masses, abnormal densities, abnormal areas of contrast enhancement or other abnormalities are seen. There is no significant change in the CT appearance of the brain as compared to our previous exam of Sept. 25, 1978. No low density areas are visualized as suggested on Sept. 16, 1978.

OPINION: Normal computed tomography scans of head.

sjc

, M.D.

DEPARTMENT OF RADIOLOGY

M , F W (56) ER — ICU 13
C78-18847 Dr. 9-16-78

SKULL: Only very limited films could be obtained. no definite fracture is seen in the calvarium; there is questionably some deformity of the cortex overlying the frontal sinuses. The sella appears normal. No definite intracranial calcifications are seen.

SINUSES: Very limited films show considerably clouding of theleft maxillary sinus, also some clouding of the lft frontal sinus, questionably with some polypoid soft tissue masses in the left frontal sinuses.

CHEST: AP bedside film shows both lungs expanded. Some prominent markingsm are present in the left base, seen through the heart shadow, and there could be minimal infiltrate in this region. Otherwise, the lung fields and pleural spaces appear clear. The heart is small. There is no mediastinal displacement. A nasogastric tube passes through the esophagus into the stomach. Electrodes are on the chest.

DEPARTMENT OF RADIOLOGY

OPINION: 1. Clouding of the left frontal and left maxillary sinuses, consistant with inflammation. Questionable cysts or palyps in the left frontal sinus.
2. Questionable bony deformity overlying the frontal sinuses, which should be correlated with any history of cranum.
3. Possible infiltrate in the left lung base. Progress films suggested.

, M.D.

jrk
9-18-78, 2:15pm

December 28, 1978

Re: W. F. M

I saw Mr. M. in my neurology clinic on December 26, 1978. This gentleman has continued to do well since his meningitis problem three months ago. Currently he has has intermittent double vision, some blurriness of vision and some unsteadiness in his balance. He has also had a change of personality according to his wife in that he has been very, at times, belligerent, screams at her and has been pretty clumsy at times using his right arm when he eats. He has otherwise, however, continued to improve and has done remarkably well. He has not had any blackouts. He is oriented to time, place and person. General knowledge and information is good. He has evidence of ptosis in the left eye with some weakness of adduction of the left eye and elevation. There is also some elevation paresis of the right eye. Pupils are equal and reactive and fundi look normal. His neck is supple. His reflexes are hyperactive bilaterally. He has actually a bilateral mild quadriparesis left greater than right and increased generalized reflexes. He has some emotional incontinence and some has some minimal features of pseudobulbar palsy.

I think overall Mr. M. has made a gradual and dramatic improvement and I have discussed at great length with him and his wife about some of the problems and complications that he has had since his meningitis and that we hope he will continue to show improvement.

If his double vision improves further I think that he may benefit from a mild opthalmological procedure where they could actually raise the left lid so that he could use the left eye for vision. I told him in the meantime to intermittently use a patch to patch either eye separately so he doesn't suppress any vision for any more than twenty four hours.

I have given him a small dose of Inderal - 60 milligrams a day to take for some of his belligerency and hyperactivity which has sometimes been of benefit in organic brain disorders. He was on Mellaril in hospital but

Page 2 Re: W. F. M

did not have any response to this.

I plan to communicate with him again in the next two weeks.

Best ~~wishes for the~~ new year,

DATE OF ADMISSION:

DATE OF DISCHARGE:

This is a fifty-six year old gentleman who three weeks prior to admission developed pain in his sinuses and forehead with coughing and sneezing and developed increase in headaches. He had a little low grade fever at that time and was admitted to and under the care of a physician and apparantly did have a little stiff neck at this time, but no other abnormalities. He was treated with some mild antibiotics and decongestants and sent home. He became rapidly ill and he was admitted to the hospital in the decreasing, lethargic and essentially comatose state a number of days after discharge from the hospital. He has a history of chronic sinus infection which had been repaired a number of years ago in the frontal region, but this had not caused any problem. He had not been exposed to anybody who had been particularly sick. At the time of admission the patient was comatose. He moved his right side with better than the left, but left hemiparesis was noted. He had evidence of severe stiffness of his neck. Pupil was smaller on the left than the right, but reactive to light. His corneal reflexes were symmetrical. He had generalized increased reflexes, much greater on the left than on the right and bilateral toe signs. He had no hemorrhages, no organomegaly or lymphadenopathy. The general medical examination was normal. Patient initially was felt toeither have a brain abscess or possible meningitis and his CAT scan was done which did not show any mass effect and was normal. Skull x-ray showed a large frontal sinus clotting and maxillary sinus clotting. Spinal fluid exam was then done, the fluid was clotting and 8 cc. was taken out. It was clear that this patient had a meningitis and he was initially started on 20 million units of penicillin. He was also seen at that time in consultation by Dr. S who agreed with his treatment at this time, but also felt that Chloromycetin, 1 gr. every four hours be given along with the penicillin to cover gram negative organisms. The patient began to make some improvement in the hospital. He developed more left hemiparesis as his treatment involved in the Intensive Care Unit. He had mild deviation of the right eye with midpositionnonreactive pupils and mild ptosis. It was felt that he was likely developing bilateral third nerve involvement from basilar infiltration and chronic and severe meningitis infection. Also possibility of venous thrombosis or cortical inflammation had to be considered. Because of his hemiparesis and confusional state and severe neurological problems underwent an angiography to rule out a possible epidural abscess and apparently this turned out to be completely normal. His EEG showed diffuse slowing with more accentuation on the left. The patient approximately ten days after hospitalization developed severe massive GI bleeding and was treated symptomatically with ice irrigation, and saline irrigations of the stomach along with NG tube insertion. He was subsequently seen by Dr. R the gastroenterologist who examined him and felt that he had a large gastric ulcer. He subsequently continued to bleed further and on 10/4/78, the patient underwent surgery for chronic duodenal ulcer, exploratory laparotomy with vagotomy and pyloroplasty under Dr. L 's care and assistant. The patient began to improve neurologically. His left arm still was weak but he started to move it. He became more alert and appeared to comprehend and began to speak very well. He still had evidence of bilateral third nerve involvement and left hemiparesis. Repeat CSF approximately one month after hospitalization

Continued on Page Two —

_____ M.D.

ATTENDING PHYSICIAN: , M. D.

DISCHARGE SUMMARY

Room:

was essentially normal. The patient developed some mild temperature on and off during time of his ICU care which was not a major problem. He was subsequently transferred to the Rehabilitation Unit and began to show marked improvement and at the time of discharge was able to walk with assistance. He will have marked ptosis and bilateral third nerve palsy in the left hemiparesis.

The patient's lab studies in the hospital were very complicated and one has to see the chart for this. He had evidence as mentioned of clouding of the left frontal and maxillary sinuses. His arteriograms were essentially normal in the carotid and vertebral distribution. CAT scan was unremarkable except for some mild atrophy. The patient's SMA 12 stayed fairly normal throughout the hospital stay. Blood gases, electrolytes and SMA 12 remained fairly normal. Hemoglobins were normal except during the time of bleeding and this was corrected by blood transfusion. The patient's initial spinal fluid examination did not grow any organisms, either fungi or bacteria. Numerous blood cultures that were done were negative. His second spinal tap showed 238 white cells, 52% lymphs, 42% segs, 90% protein, positive gamma globulin and 60% sugar. The initial fluid revealed 8400 white cells, 85% segs, and 234 protein, 5% lymphocytes and 40 sugar.

The patient was finally discharged on no major medication and will be followed by myself, Dr. S and Dr. L as an outpatient.

FINAL DIAGNOSIS: BACTERIAL MENENGITIS with neurological complications of hemiparesis and bilateral third nerve palsy.

RD:ws:TCDC
D: 2-18-79
T: 2-19-79

ATTENDING PHYSICIAN: _____ M.D.
M. D.

DISCHARGE SUMMARY

M. , W .
#
RM #

DATE OF CONSULTATION: 9-16-78

CONSULTING PHYSICIAN:

CONSULTATION NOTE: This is a very interesting 56 year old white male whom I am asked to see because of meningitis. The best that we can tell, the patient is semi-comatose at this time, but the patient, about three weeks ago began to develop severe frontal and maxillary type headaches suggestive of sinus problems. He was then recently admitted to , Hospital about one week ago with fever and headaches and some mild stiffness of the neck. He was treated with some intramuscular Ampicillin for three or four days and apparently his neck pain got somewhat better, and apparently the fever however persisted. The patient was then dismissed, probably on no antibiotics at that time. The records are not available from that admission however. The patient then yesterday, the day prior to admission, went to a chiropractor because his neck pain began to recur. His fever and headaches persisted at that time. He came home from the chiropractor and went down to lie down and later that evening could not be aroused by his family and was brought to the Emergency Room. He was found to have 104 fever and stiff neck and seemed to be moving his left side poorly and the possibility of a subdural empyema or brain abscess was entertained. Stat skull films were done which showed clouding of the right maxillary and right frontal sinuses with some fluid! An emergency CT scan was done about one o'clock this morning which did not reveal any space occupying lesions and no obvious subdural empyema. At that point a lumbar puncture was then performed which revealed cloudy spinal fluid with about 8000 cells, 90% of which were polys and the rest of which were lymphs. He had glucose of the spinal fluid of 40 with simultaneous blood sugar of 146-and protein which was 234 mgm%. Gram stain which I just looked at reveals numerous polys, few lymphocytes, occasional red cells but no definite organism was seen.

On physical examination at this time this is a semicomatose white male having labored respirations, lots of upper respiratory secretions. The examination revealed the tympanic membranes to be clear. The left pupils was pin point and the right pupil was about 1-2 mm larger and both reacted poorly to light. The fundi could not be visualized and there was some questionable deviation of the left eye. The oral pharynx could not be adequately seen. There is an oral air-place. The neck was markedly stiff.
LUNGS: bilateral upper respiratory- sounds as well as some rhonchi. No definite rales heard.
CARDIAC: difficult to hear because of the multiple air-way sounds. There were no obvious blowing murmurs.
ABDOMEN: soft without organomegaly, masses or tenderness.
EXTREMITIES: unremarkable
NEUROLOGICAL: as per Dr. , but it does appear that the patient moves his right side better than he moves the left side, and does respond to painful stimuli, but that is it.

Lab values - his urinalysis was normal. Electrolytes showed sodium of 131, potassium

M.D.
Attending Physician

M.D.
Consulting Physician

CONSULTATION

M , M.
Room: ICU-#13
#118

PACIENTE

was 3.3, chloride of 84 and bicarb of 32. His arterial blood gases revealed PO2 of 47, PCO2 of 34, pH of 7.56.

IMPRESSION & DISCUSSION: this is a very ill gentleman with probable meningitis, secondary to sinus focus. In adults, even with acute sinusitis, one has to of course consider pneumococcus, but one also still has to consider hemophillus influenzae, even though this is a rare cause of meningitis in adults. The patient was given 5 grams of Penicillin stat just prior to my seeing him by Dr. [illegible], but I think that one should add additional Chloramphenicol to cover hemophillus and anerobes and leave the Penicillin to cover the pneumococcus pending definitive cultures. Since the patient has a partially treated meningitis, since he had received prior Ampicillin, we may have difficulty knowing the true pathogen in which case I think that CTH should be sent should the cultures remain negative. Also, want to just leave in the back of our minds, the possibility of a subdural empyema despite the negative CT scan for if there is a very thin collection, this still can be missed, even with the CT scan, especially without contrast studies which could not be done on this patient because of his clinical conditions.

Thank you very much for allowing me to see this interesting patient. I will follow him with your permission.

RS/jo #C* TCDC
D:9-16-78
T:9-17-78

_____ M.D.
M.D.
Consulting Physician

CONSULTATION

M [illegible], W [illegible]
ICU #13
#118_____

DATE OF CONSULTATION: 9-30-78

CONSULTING PHYSICIAN:

CONCLUSION: Upper GI bleed, most likely secondary to duodenal ulcer.
We need to rule out the presence of an erosive esophagitis or erosive gastritis, gastric ulcer and other causes of upper GI bleeding.

RECOMMENDATIONS: 1) upper GI endoscopy. I will schedule this and to it this afternoon.

COMMENT: This 56 year old male has been admitted to the Intensive Care Unit at the Hospital because of meningitis. He has been in the Intensive Care Unit for a period of almost two weeks and he has been noted to have evidence of upper GI bleeding in the form of frank blood in the N-G tube. On review of his records, it is noted that he had an acute drop in his hemoglobin and hematocrit on 9-22-78, and that he has had the N-G tube in position for almost 10 days. During the last two or three hours the N-G tube was disconnected, and he developed upper GI bleeding again on 9-29-78. In the last 24 hours he has required almost 10 units of blood in the form of transfusions.

On talking to the patient's wife, she says that the patient has been a heavy coffee drinker, drinking several cups of coffee, 10 to 15 at least daily, but consuming very little or no aspirin or aspirin-containing compounds at all. He is not known to have definite evidence of ulcer disease, and he has not apparently had any upper GI series performed or medical hospitalizations relating to peptic ulcer disease in the past.

The details about his present illness, not related to his upper GI bleeding, has been well documented in the chart, and I shall not discuss them here.

On examination the patient appears to understand and comprehend directions and commands, but is unable to express himself clearly. He does not seem to be in any acute distress at the present time. The nasogastric tube is in place, and the returns are clear at the moment and he does not show evidence of fresh bleeding. He does not have stigmata of chronic liver disease, and examination of the abdomen in particular does not reveal any hepatosplenomegaly. Bowel sounds are within normal limits. The rectal examination reveals melanotic stool but no evidence of fresh blood in the rectum.

I think this person deserves an emergency endoscopy to assess the nature of the upper bleeding and I shall perform this this afternoon. Thank you very much for asking me to see this patient in consultation. I will follow this patient closely with you in the hospital if you wish.

AFR/jo #C* TCDC
D:10-2-78

M.D.
M.D.

CONSULTATION

M. [illegible], M.
#11811787
ICU #13

T.C. 1978 MEDICAL RECORDS

PAGE TWO

OPERATIVE REPORT (CONTINUED)

then irrigation of this area was performed with normal saline. Attention was turned to the reconstruction of the pyloroplasty. Two stay sutures of # 3-0 CH silk were used as traction sutures. A suture of # 3-0 CH chromic was used in running fashion using the Connell type of suture. This inverted the edges of the duodenum well. Then interrupted sutures of # 3-0 CH silk were used to further invert the sutures. These were placed in interrupted fashion. After completion of this row the entire layer of chromic had been turned inward. A good opening was left in the created pyloroplasty. Following this the right upper quadrant was further irrigated with normal saline solution and then the procedure was completed by replacing the abdominal contents into their respective positions and closing the abdomen. The closure was performed with # 0 chromic general closure on the peritoneum and # 0 Prolene on the fascia layer in running fashion with interrupted sutures of # 0 silk. The skin was then closed with staples. The patient left the operating room in fair condition and was transferred back to the Intensive Care Unit to be maintained on the MA-1 ventilator.

JL/dah TCDC °C
DD: 10/2/78
DT: 10/2/78

_____ M.D.

, M.D.

OPERATIVE REPORT	M , M.

ATTENDING PHYSICIAN: , M.D.

OPERATION: Esophagogastroduodenoscopy.

SURGEON: , M.D.

PROCEDURE: With the patient in the left lateral position Cetacaine throat spray was applied to the posterior pharynx. The Olympus D3 fiberoptic instrument was easily introduced through the pharynx into the esophagus without any difficulty. Upon entering the esophagus there was a small hiatus hernia of doubtful significance. At the distal end of the esophagus, extending into the fundal area there was a patch of erosive esophagitis and gastritis but this was not actively bleeding at the present time. Upon entering the stomach there was no evidence of any fresh ulceration of bleeding. The pylorus appeared irregular and indicative of chronic peptic ulcer disease. Upon entering the duodenum, the duodenal bulb, significant finding was a large chronic duodenal ulcer with no evidence of fresh bleeding at the present time. The duodenum beyond appeared within normal limits and this ulcer seemed to be a shallow ulcer but it was occupying more than half the area in the bulb. The scope was then withdrawn and the above findings were confirmed. The patient tolerated the procedure well and this procedure was terminated.

IMPRESSION: Chronic duodenal ulceration. Large duodenal ulcer which is not actively bleeding now. Evidence of chronic peptic ulcer disease. Mild erosive esophagitis.

RECOMMENDATIONS: It is perhaps better to leave the nasogastric tube out for the present time being because of the presence of mild erosive esophagitis which could be another source of bleeding. Antacids and Tagamet as ordered but I would increase the Tagamet to 300 mg. every six hours instead of eight hours and I would give the antacids every two hours for now. Prohibitive, 30 mg. at night will also help in the presence of chronic peptic ulcer disease. Observe the hemoglobin and hematocrit frequently. A surgical consult would be in order at the present time. The problem has been discussed with Dr. and the family and I will follow this patient.

APR/dah TCDC 9E
DD: 9/20/78
DT: 10/3/78

M.D.

., M.D.

OPERATIVE REPORT

M : , M
Hospital # 11811787

TISSUE REPORT

DATE October 4, 1978

#11811787 ICU-13

PATIENT NAME M[illegible] (56) Dr. CASE NO. 78-12227

SURGEON: Dr.

Specimen Submitted: Section vagus nerve

Operative Procedure: Exp. lap., vagotomy

Operative Diagnosis: G.I. bleeding.

GROSS: The specimen is submitted as a section of vagus nerve. It consists of two irregular portions of soft tissue partly covered by lobules of fat. They are approximately 12 x 7 x 3 and 15 x 9 x 2 mm. Also submitted in the same container is an additional portion of fibro-adipose tissue that measures about 4 mm. in largest diameter. All submitted in two blocks. (LEM:jr 10-4-78)

MICROSCOPIC: Sections of the portions of tissue submitted reveal loose fibrous connective tissue with small blood vessels and numerous bundles of nerve fibers. Also noted are areas of recent hemorrhage.

DIAGNOSIS: Nerves (Vagus), resected portions.

EXAMINED BY_____ M.D.

LEM:ee 10-5-78

DOCTORS COPY

DATE OF CONSULTATION:

HISTORY: Mr. M is a 56 year old married white male who has had a very stormy present illness. It began in early September with nonspecific symptoms of headache and cephalgia and he was admitted under the care of Dr. at the Hospital in acute semicomatose state of undetermined etiology. He had a known history of chronic sinusitis with a history of recent meningitis and gastritis and esophagitis and/or duodenitis type of symptoms previously. He was referred to Dr. at this hospital whose impression was that the patient probably had a meningitis and he was begun on penicillin. In addition, consultation with the infectious disease service, Dr. , was obtained and his impression also was a probable meningitis and added chloramphenicol to his penicillin therapy. He was then switched to p.o. amoxicillin suspension plus gentamicin (100 mg., IV) and then was tapered down to strictly the amoxicillin suspension and this how has also been discontinued. His only other medications are vitamin supplementations. He does take Tagamet (300 mg., with meals). During this hospitalization, he developed an acute gastrointestinal bleed and was seen by Dr. in surgical consultation and ultimately underwent an exploratory laparotomy on 10-4-78 with lysis of adhesions, exploration of stomach and duodenal area, as well as vagotomy and pyloroplasty. He tolerated it fairly well. His diagnosis was made prior to surgery by Dr. by esophagogastroduodenoscopy. The patient has been followed both by Dr. and by Dr.

The patient has presently been referred to the rehabilitation service for consultation as regards rehabilitation potential. He is now on the rehabilitation unit and has begun his physical therapy and occupational therapy.

On physical examination, the patient is awake and alert but is quite lethagic and listless. His repponses are slow. Complete orientation seems to be intact but this is difficult to assess completely. He does know that it is 1978 and he knows that he is in the hospital and he is able to given Dr. 's name as his physician. He, however, did not give me his age and did not know the month or date. Some of this may well be due to confusion from a prolonged hospital course and is not at all unexpected in a patient who has undergone the rather exhaustive acute medical and surgical management that this patient has undergone. Head and neck examination is unremarkable. He does have a mild early systolic bruit in the left carotid. The chest is unremarkable with lung fields that are clear and equal bilaterally. The abdomen shows a well healed surgical scar. The abdomen is not tender and bowel sounds are present and normally active. Examination of the extremities shows that this patient is currently ambulatory with some minimal to standby assistance. He has good strength, equal bilaterally. Grip strength is perfect. Deep tendon reflexes are 1+/4+ and equal bilaterally. Extensors of the foot are intact as well as flexors and he is able to dorsiflex and plantar flex with excellent strength and coordination.

--cont'd. on next page--

M.D.

, M. D.

CONSULTATION

M
11911787
Room: 712

T C 1978

MEDICAL RECORDS

page two

IMPRESSION:

1. Meningitis, status post treatment.
2. Generalized weakness and debility following severe late stage meningitis as well as abdominal exploration and surgery.

PLAN: Theppatient will be begun on rehabilitation care. Physical therapy will begin work with standups, general conditioning, transfers, and progressive ambulation andoooccupational therapy will work with his activities and daily living and endurance program and perceptual activities.

_____ M.D.

, M. D.

CONSULTATION--cont'd.

M , M.
11911787
Room 712

MEDICAL RECORDS

APPENDIX B

CONCENT FORM FOR ELECTROLYTE AND
ENDOCRINE STUDY

We are studying electrolyte and endocrine dysfunctions and their effects after brain damage. Tests and evaluations will be made to determine some of the ways this effects the patient's recovery if there is an endocrine or electrolyte dysfunction. We request the patient to have an electrolyte and thyroid evaluation done by the physician of his choice. We also would like to do a personal evaluation of the patient's physical and mental ststus after the physical examination by his physician.

The confidentiality of all materials will be maintained. We expect the findings of this study to be of benefit to professionals engaged in rehabilitation of patients after brain damage.

We will be happy to answer any questions you have. If you wish to drop out of the study before it is completed, you are free to do so.

I understand these procedures and agree to participate.

_____Signature

_____ Evaluator

APPENDIX. C

✓ equipment
✓ Majority
X instute
✓ Literature
✓ Severence
✓ museum
✓ Precious
✓ Illogical
✓ decision
✓ Quantity
✓ apitive
✓ Necessity
✓ Opportunity
✓ Anxiety

Conscience

X Cons
X Physican
✓ Courteous
✓ position
X Lucity
X exaggate
X Knowledge
X Locoquen
X Medival
X Ofimenall
X Ress
X sovrenty
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X Irrestible
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✓ Guarantee

perseverance

X Preedent

(14)

(14)

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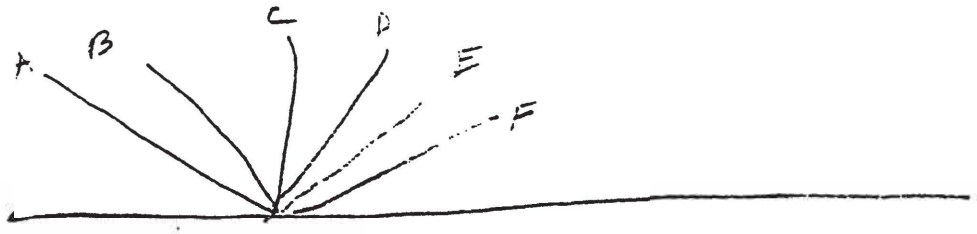
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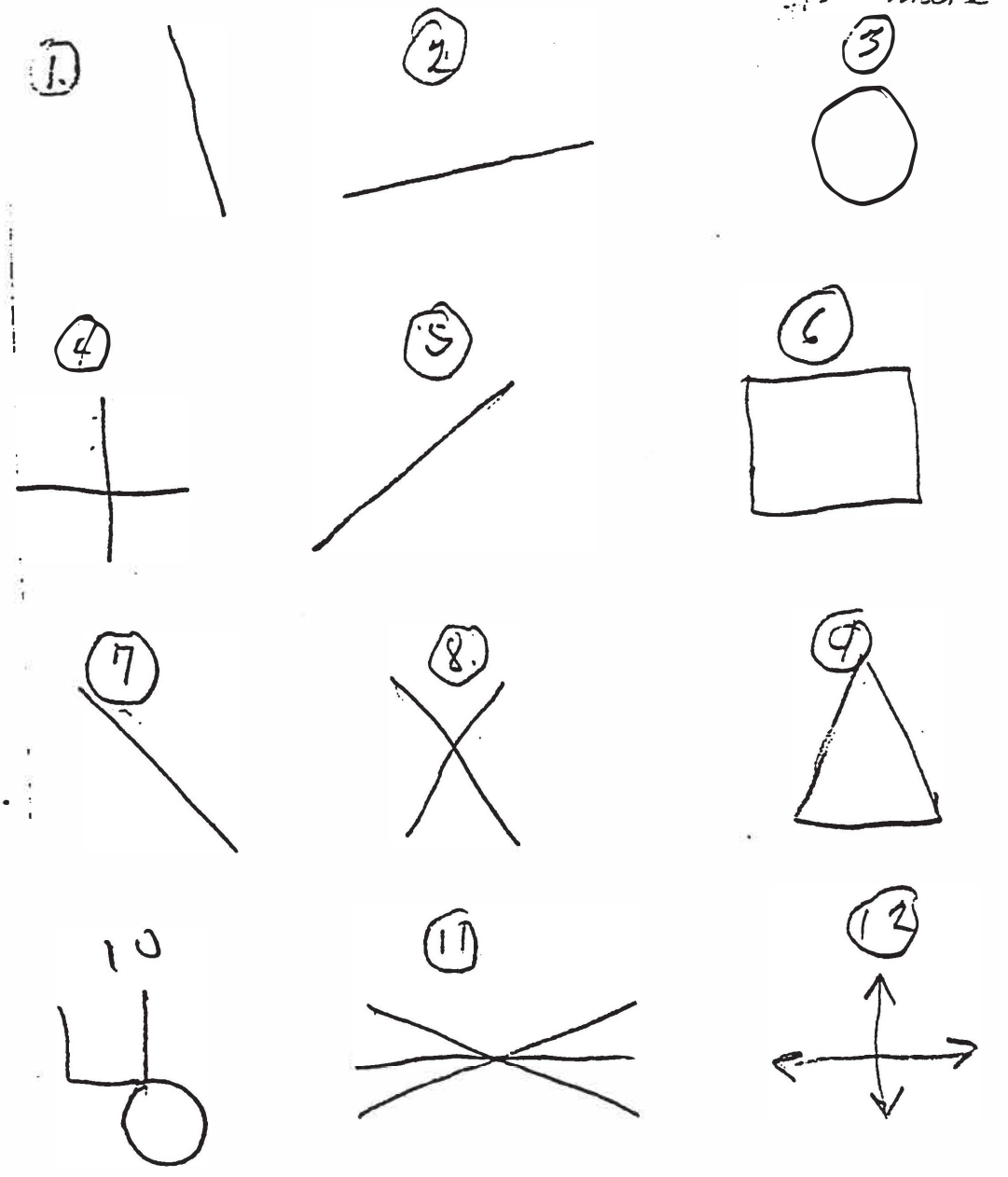
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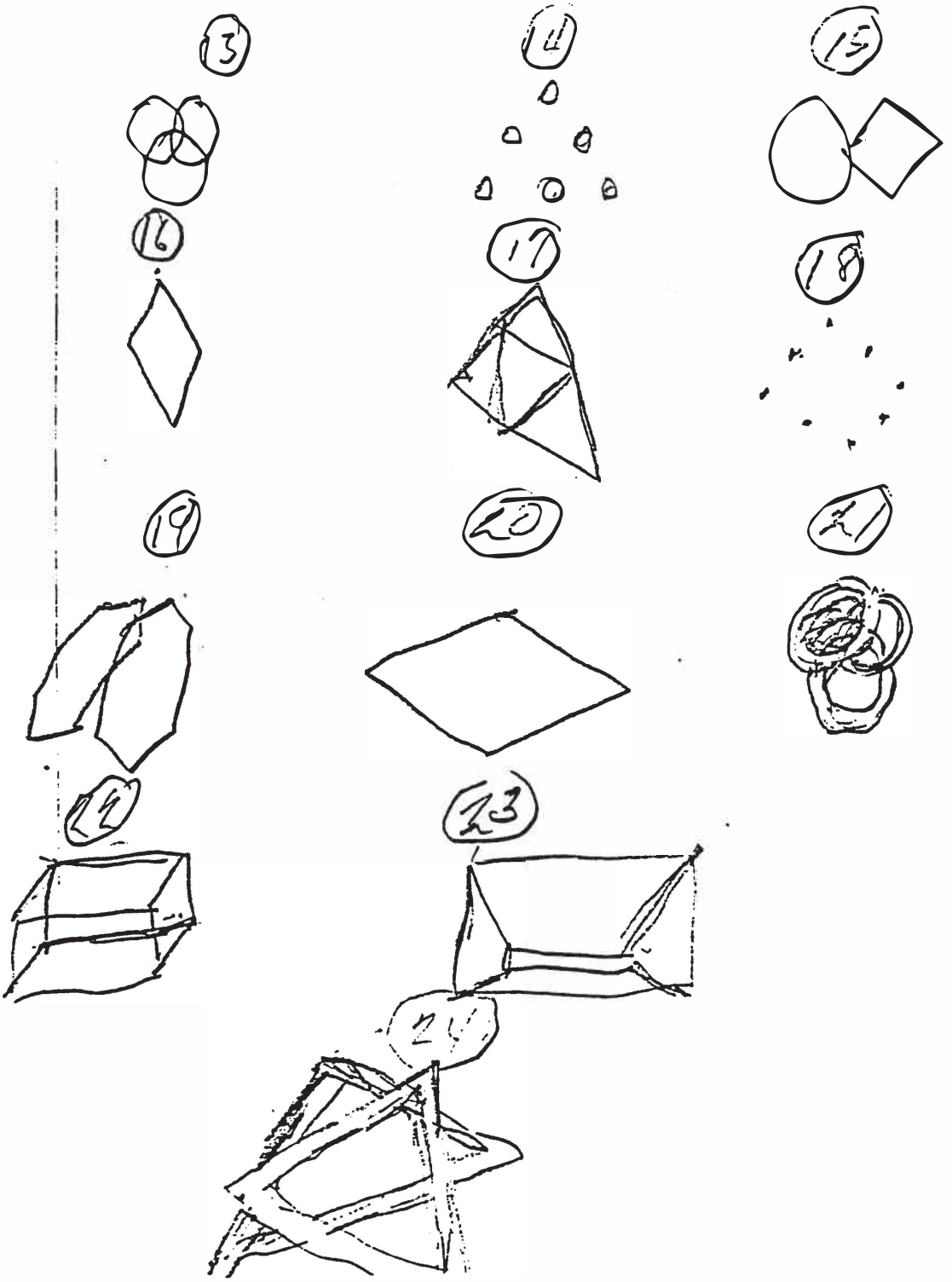
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13
24

INSTRUCTIONS

(SC)

Look at the two rows of letters below.

A M G E W I N D T E Y K Z C I R O C K W Q E H O W L O Z N P E

B E L T O T H U L L V A Y F S M I P L A N F O U R Y U N T R I

Notice that a circle has been drawn around groups of four consecutive letters that spell out a common English word. Only four-letter words are marked. The same letters are used in only one word at a time.

Below are more rows of letters on which to practice. Draw a circle around each set of four adjacent letters which spell out a common English word. Go ahead. Do not wait for any signal. Read along the rows from the left side of the page to the right side.

K I Y O U R G A C S P I H A N D V Z Q J U X D I E D O L R O O M

V O R S Z R E A D U O F T E B E E N A Q P A U N T A Q S E R T L

X J A K C O W B N O S E L I Q X E S T F A S T W D O M T R I Z E

E L B I E B E S T B T R E X D O W N G U T N E L A S T W I T C I

When the signal is given (not yet), turn the page and draw a circle around each set of four adjacent letters in a row that spells out a common English word. Go from left to right in each row. Work fast. Mark only four-letter words.

SC-Form 1

Draw a circle around each four-letter word in the rows below

Date 7-1-82

Subject WM

Examiner AC

L K Y S A N D T E H P E E K L H R G X Z P U I L R O A M I G R D S Q H A C K J T J O R S P V

L Y D N F J M S I C K R A W H U N P L I Y R A S H W Z O K F L I T J H U B Q H A S H K C Z U

K U Y H G K L M B G F Y J W O R E H U G T K N G F D S O M E U H T G F D L M B G Y U I J F X

O P K J H N B G R E A D T G H I K E N H P E R K J H Y G T R J H G T R J H G F R V Y B J I U

F U S E T H J N H G R F Y U H G F D C S A Q M O R E F G U H U J H T R F I J K H F V E I N J

U P O L J U H Y T R F G H D W Q A P K H B V T U S E N T J U H S A M E T H O O K H T G X E K

K I N Y H E D F G U M E R E G W I N K I H T D Z P O K N G R Q A X V S T O P L O U Y G C D Q

Q D G S L A M J H R E A L O J H T F R E D J S Z V N I J K J R E W Z N P U H M O S T Y X A P

P L I N E K Y F C M W Q P Z G X J T G M A K R Y I J G R D M A K E A S I A J O P L G Z X W Y

K U H T R G H V D E N E W S J K I G T E D N J M O N K U Y T R F W D E T R I M L K J N E G Y

I J H Y T C E D S R V B N M L K I R O N D H E A P J G U F W E Q S D B J U Y H Y T F U Z X P

J I U Y C O L T S E E M U F G D S H J K L G R A Y I H T R D W X C V B J K G F T R E O U R S

P M O R E H Y T Q S C F T Y J M O K N H T R F C U D O S E H V F C U R H J N V F M U S S O Z

K I J H G V C D W H U I F O O L K J H H T E V O O R O S Y X C C J U P A V E H G U C D W R H

K O P T F X W D V R U S H J N G B T F D S E O L P K J H V W A L K O H C D W G N K U Y T D C

H V D E W G R E W T P J B C D Q A Z F Y M I L L O H C W D F G J B I R P L A N H T D B G I F

I H V F E W D V H U O K N N O S E T F X P K N Z W Y S K J H G I Z M O L D H V E G K L K U G

R G N J Y T F X D W D X O T I L T N H E A T O J B V C F V E A L O B J R F E G S H O W Z G W

U G N R F S U U H S I D E X F Y J L M G R T E A R I J H B G F C D S X W F B J I O K N B V Z

Z A D G J L W A I T H O O B Z G D A L U N B S L A Y I H V D W Q D G H N K P I G V L A N D G

N H T D Z W I L L X J B G G J H V E F U H S A F E M I N E J Y G F C D W F B H U I P L J B V

J L O S T H T R D V B H T Y D S Q Z J O P L J F E L T H V W E F D V H U I J B F G H Y T R R

12-1-81

exercise #28 page #60

(1) $1/3x - 3/4x + 5/12x = 1 - 11/6x$

~~4 + 4x + 10x~~

(15) $\frac{3h}{1} - h = \frac{3}{2} = \frac{4}{5}$

$\frac{-3h}{-10}$

(13.) $5x - .55 = .33 - 3x$

$8x = .88$

$x = .11$

(14) $.23 - x = .95 - 3x$

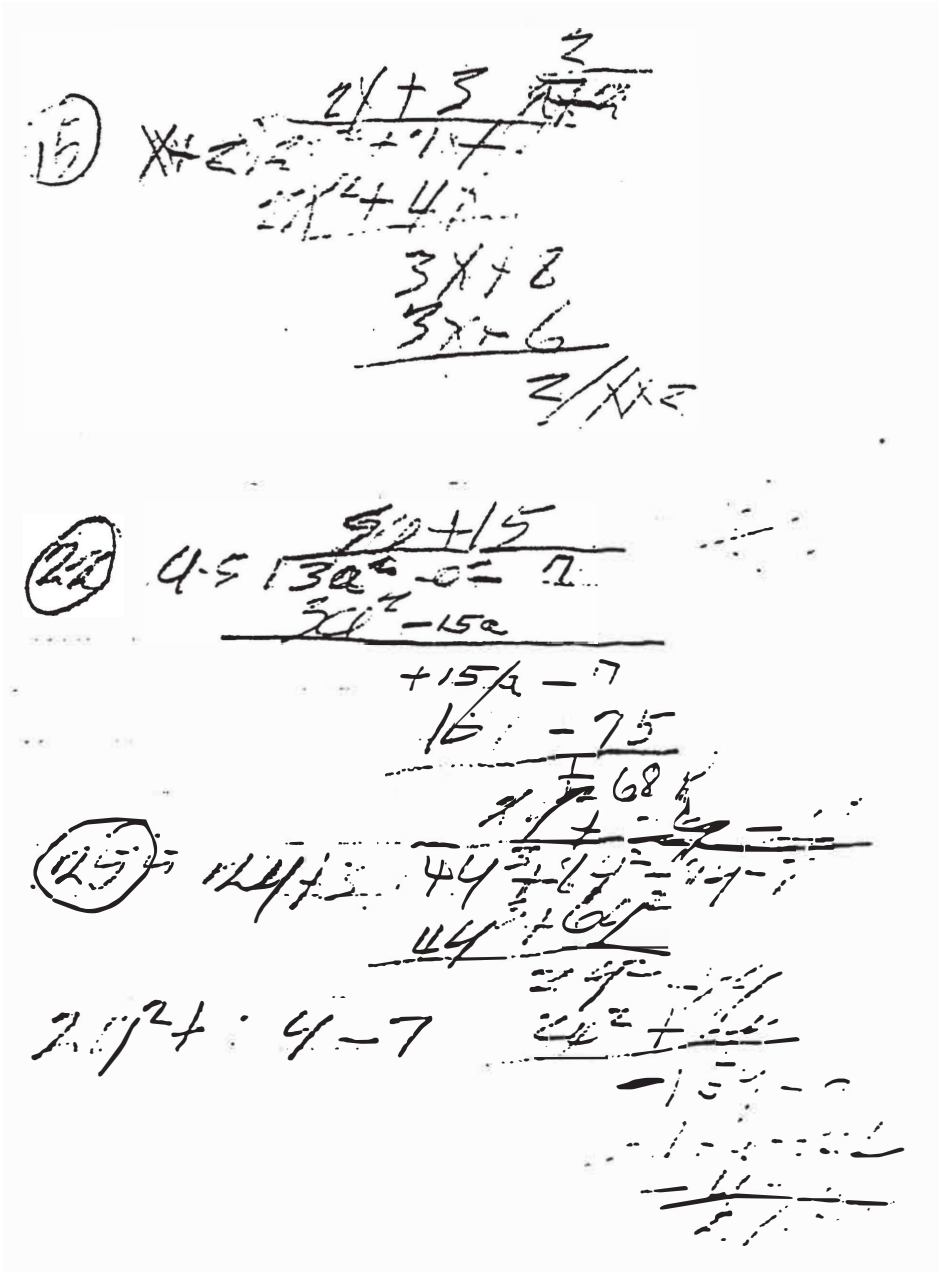
$-2x = .72$

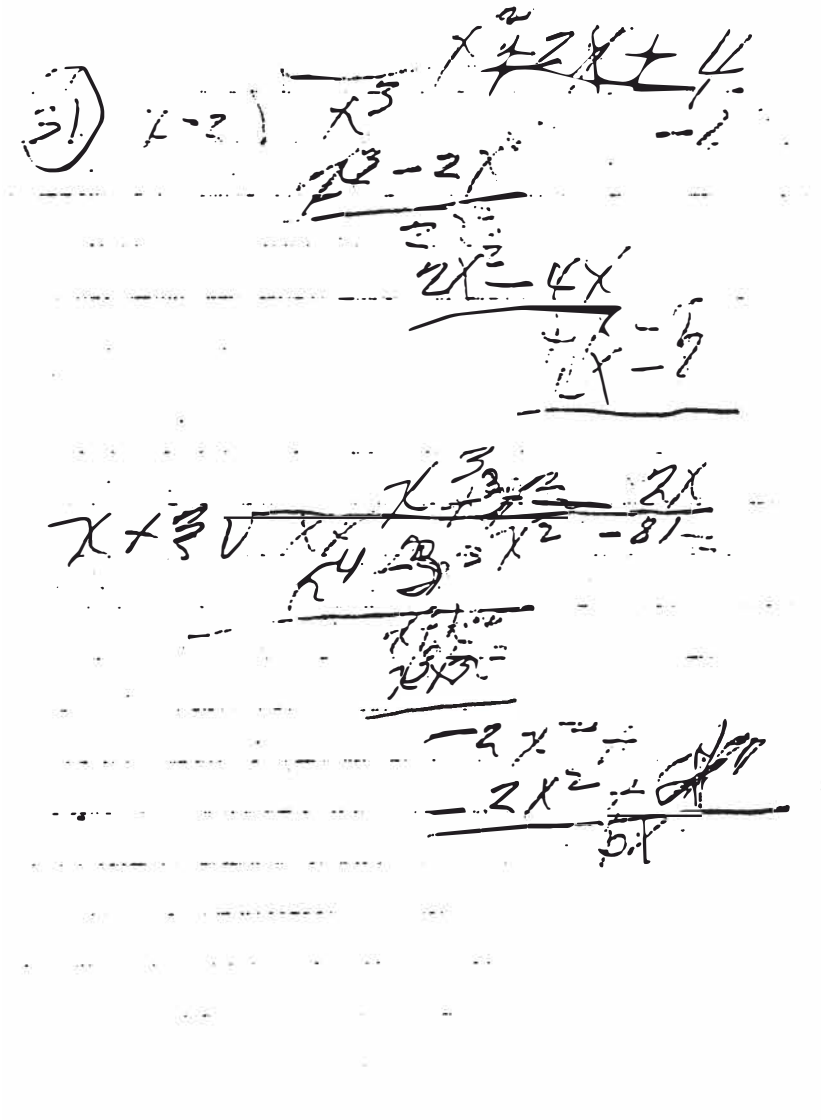
$x = -.36$

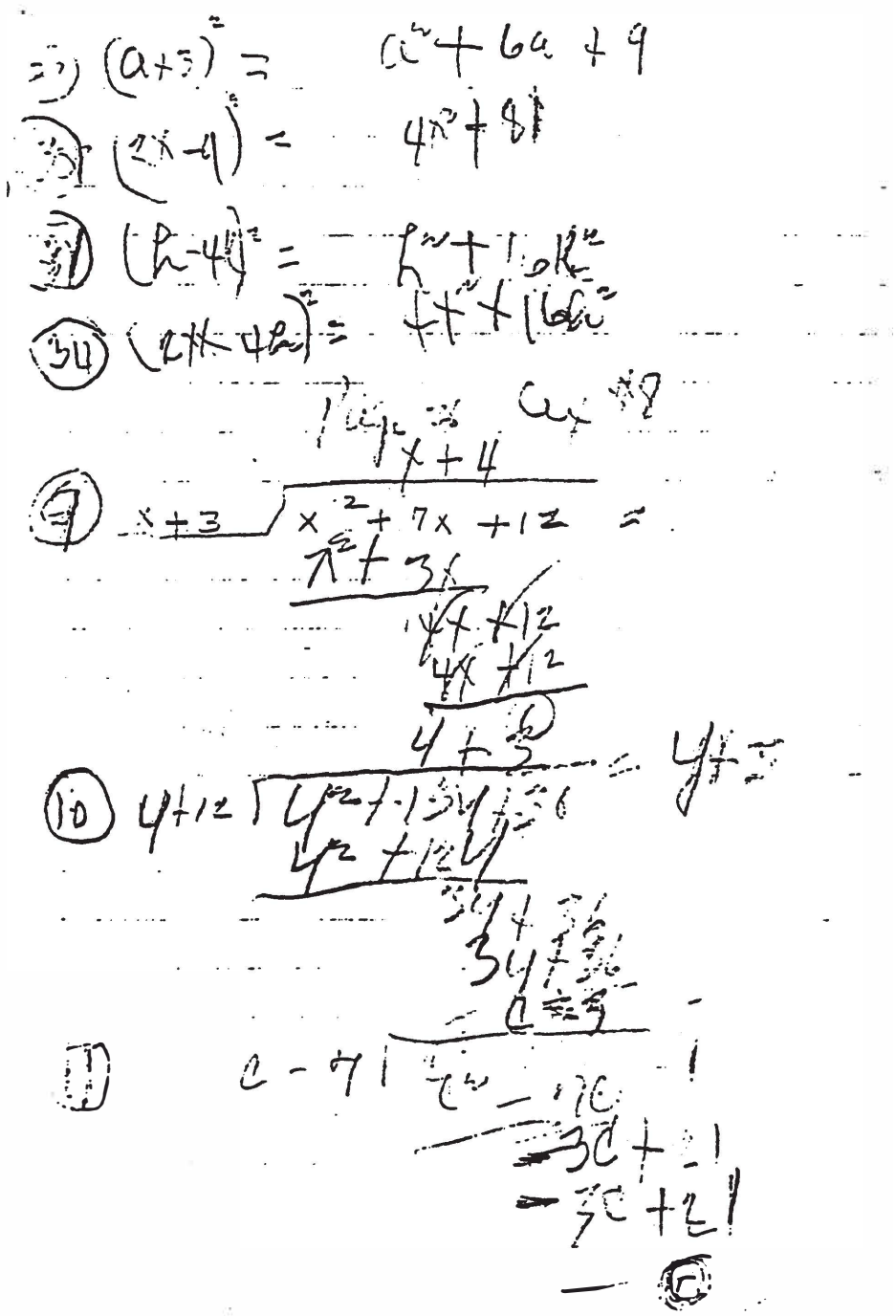
(16.) $\frac{2x}{15} - \frac{x}{3} = \frac{x}{5} - \frac{2}{5}$

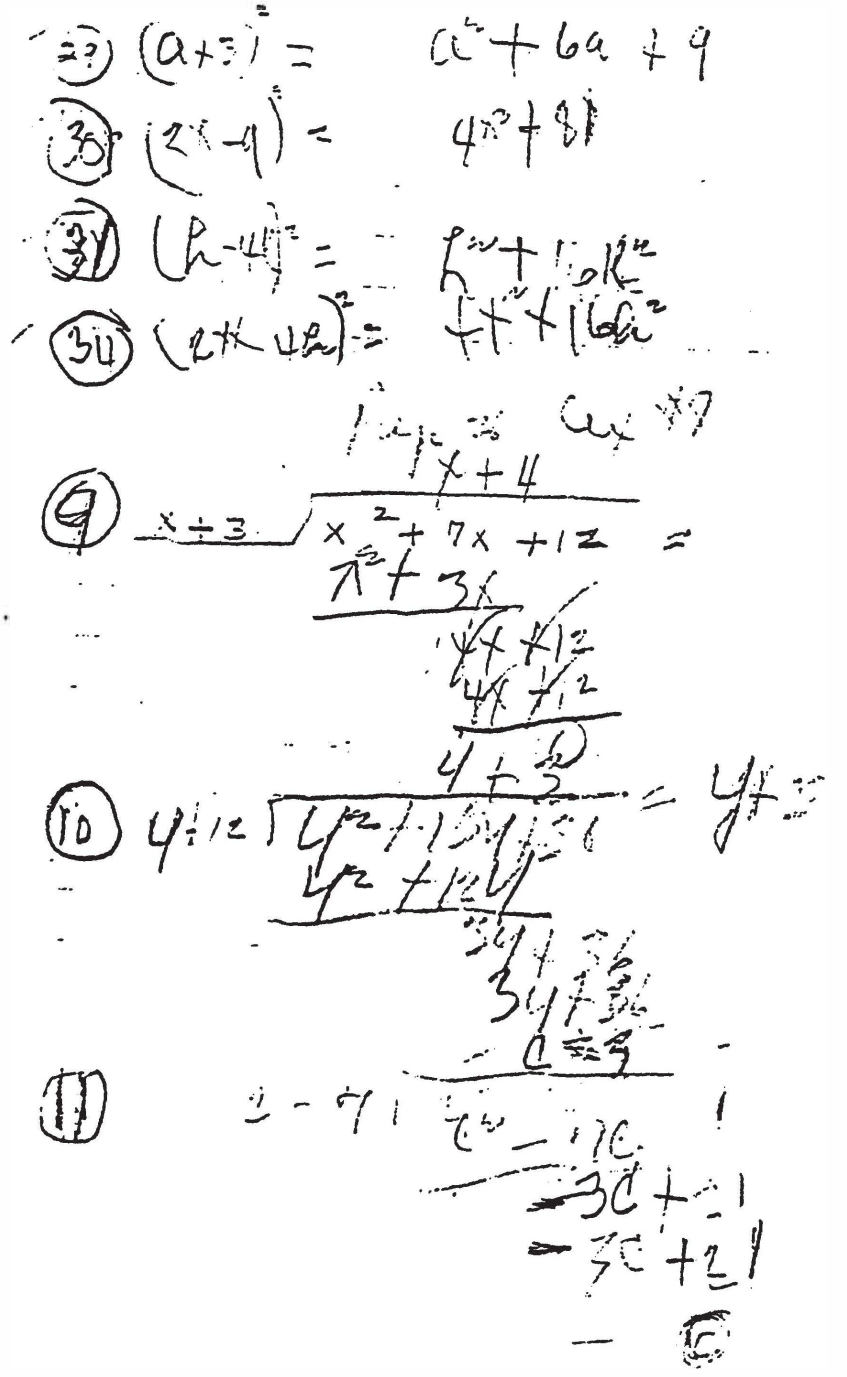
$\frac{2x - 5x}{15}$ $\frac{3x \; 18}{15}$

$\frac{-6x}{15}$






(a+3)² = a² + 6a + 9
(2x-4)² = 4x² + 8
(h-4)² = h² + 10h
(2k-4h)² = 4k² + 16h²
x + 4
x + 3 | x² + 7x + 12 =
x² + 3x
4x + 12
4x + 12
0
4 + 3 = y + 3
4 + 12 | y² + 13y + 36
y² + 12y
y + 36
y + 36
0
c - 7 | c² - 10c + 21
- 7c + 21
0


(29) (a+3)² = a² + 6a + 9
(30) (2x-4)² = 4x² + 8x
(31) (k-4)² = k² + 16k²
(34) (2x+4a)² = 4x² + 16a²
(9) x+3 | x² + 7x + 12 = x+4
x² + 3x
4x + 12
4x + 12
0
(10) 4+2 | 4² + 12 = 4+2
4² + 12
0
(11) 2 - 7 | 2² - 7
-3c + 21
-3c + 21
0

Ex-7, pg. 25 (12:20 PM)

$2x(-3x-5x^3) = \boxed{6x^2-10x^4}$

$a(a^2-a^3) = \boxed{a^3-a^4}$

$(x-5)(2x+7) = \boxed{2x^2-3x-35}$

-35, $+7x$, $-10x$

$(x+5)(2x+7) = \boxed{2x^2+17x+35}$

$+7x$

$(3y-2)(2-5y) =$

$(3y-2)(-5y+2) = -15y^2+16y-4$

$+10y$, $+6y$

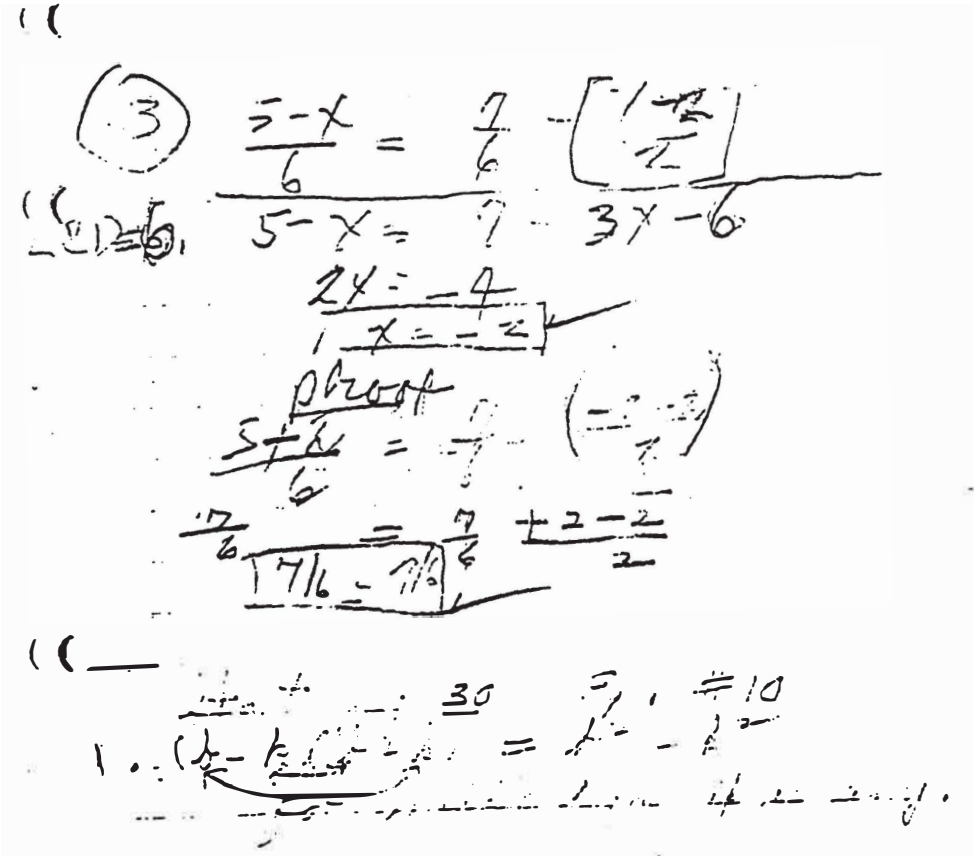
$\boxed{15y^2-16y+4}$

$(x+y)(x-y) = x^2-y^2$

$(x-5)(2x+7) = 2x^2-10x+7x-35$

$\boxed{2x^2-3x-35}$

$x-3(7-x) = x-3(x+7) = \boxed{x^2+11x-21=0}$



$$\left[\frac{z-7}{2}\right] = \left[\frac{1-z}{10}\right]$$

$$5z - 35 = 1 - z$$

$$6z = 36$$

$$z = 6$$

29

$$.17x - .35z = .08 - .09x$$

$$.26x = .370$$

$$x = .085$$

31

$$\left[\frac{4-3x}{3}\right] - \frac{21}{12} = \left[\frac{5x-3}{4}\right]$$

$$16 - 8x = 21 - 15x + 9$$

$$7x = 14$$

$$x = 2$$

$$\frac{4-4}{3} = \frac{21}{12} - \left[\frac{10-3}{4}\right]$$

$$\frac{0}{3} = \frac{21}{12} - \frac{7}{4}$$

$$\frac{0}{0} = 21 - 21$$

$$0 = 0$$

— Exercise 25 Page 60

(time 30 min)

① $x-5 = 5x+7$
$-4x = 10$
$4x = -10$
$x = -\frac{10}{4} = -\frac{5}{2}$

② $5-3y = 2-4y$
$y = -3$

③ $4z+5 = 8-2z$
$6z = 3$
$z = \frac{1}{2}$

④ $2(4+x) = 8+3x$
$8+2x = 8+3x$
$-x = 0$
$x = 0$

⑤ $6x+15 = 5x+2$
$2(x+3) = 2(x+5)$
$4x = 5$
$x = 1\frac{1}{4}x$

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